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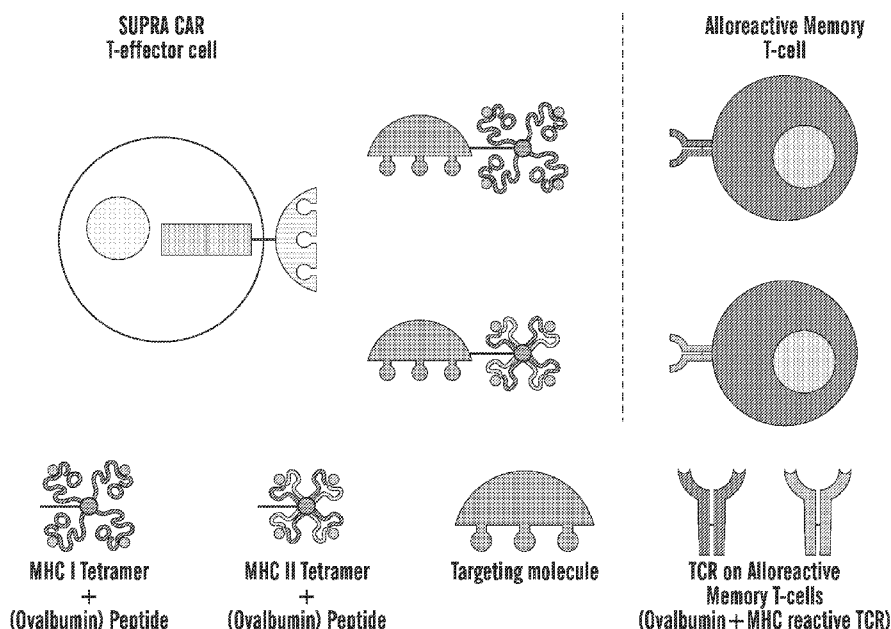
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(54) Title: CAL-T CONSTRUCTS AND USES THEREOF

**FIG. 10**

(57) **Abstract:** The technology described herein is directed to compositions comprising components of multi-component CALs or CARs, e.g., a TCR recognition domain; and one or both of: (a) an intracellular signaling domain; and (b) a first-type protein interaction domain. Further provided herein are methods for treating or preventing an autoimmune disease, a transplant rejection, or graft versus host disease.

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- *as to the applicant's entitlement to claim the priority of the earlier application (Rule 4.17(iii))*

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CAL-T CONSTRUCTS AND USES THEREOF

CROSS-REFERENCE TO RELATED APPLICATIONS

[0001] This application claims benefit under 35 U.S.C. § 119(e) of U.S. Provisional Application No. 62/916,924 filed October 18, 2019, the contents of which are incorporated herein by reference in their entirety.

SEQUENCE LISTING

[0002] The instant application contains a Sequence Listing which has been submitted electronically in ASCII format and is hereby incorporated by reference in its entirety. Said ASCII copy, created on October 16, 2020, is named 701586-096030WOPT_SL.txt and is 540,018 bytes in size.

TECHNICAL FIELD

[0003] The technology described herein relates to chimeric antigen ligand (CAL) technology, e.g., for treating of autoimmune and/or T-cell mediated conditions.

BACKGROUND

[0004] The major cause of autoimmunity and transplant rejection, e.g., following an organ transplant, is autoreactive and alloreactive T cells attacking the patient's or donor's organ(s), tissue, and/or cells. Current treatments for preventing autoimmunity and transplant rejection involve stringent immunosuppression, which can lead to severe, unwanted side effects including extreme susceptibility to infection, malignant and benign neoplasms, multiple organ and systems failure. In order to prevent autoimmunity and transplant rejection without inducing unwanted side effects, it would be advantageous to delete autoreactive and alloreactive T cells in a patient receiving an organ transplant without needing to additionally administer immunosuppressant medications, or to reduce the doses of the immunosuppressant regime delivered concurrently. This type of therapy, however, has not been successfully demonstrated.

[0005] T cells recognize their target cells by using receptors on their cell surface which are called T Cell Receptors (TCRs). TCRs have a recognition portion and a signaling portion. When the recognition portion binds to the natural complexes formed in the body in the presence of a diseased cell, the signaling portion is activated, which leads to the T cell engaging in killing activity or recruiting other immune cells to destroy the diseased cell. The natural complexes are also known as peptide-major histocompatibility complexes (pMHC complexes). CAR-T cell therapy is known and seeks to help T cells recognize autoreactive and alloreactive T cells. This is accomplished by genetically altering a T cell so that it expresses a chimeric antigen receptor (CAR). The CAR is an altered TCR, in which the natural recognition portion is removed and replaced with a synthetic

recognition portion that is designed to more effectively recognize the autoreactive and alloreactive T cells by very specifically detecting the presence of a molecule unique to the autoreactive and alloreactive T cells. These CAR-T cells are then given to a patient. Inside the patient, their synthetic CAR molecules will bind to the autoreactive and alloreactive T cells and in the act of that binding, activate the T cell, resulting in the patient's own immune system attacking the diseased T-cells.

SUMMARY

[0006] Described herein are methods and compositions relating to chimeric antigen ligands (CAL). The CALs described herein comprise a TCR recognition domain and a biomolecular interaction domain. The TCR recognition permits the CAL to bind to autoreactive and/or alloreactive T cells in an antigen specific manner. The biomolecular interaction domain permits an immune killer cell (e.g., a NK cell, a T cell, or a dendritic cell) to bind to the CAL, thereby promoting killing of the autoreactive and/or alloreactive T cell by the immune killer cell.

[0007] The TCR recognition domain of a CAL binds specifically to a TCR, e.g., a TCR expressed on the surface of an autoreactive and/or alloreactive T cell. Exemplary but non-limiting TCR recognition domains include peptide-MHC complexes, e.g., in monomeric, oligomeric, or multimeric form. The TCR recognition domain can comprise natural or synthetic sequences. Specific examples of peptide-MHC complexes that include autoreactive or alloreactive antigens are provided elsewhere herein.

[0008] The biomolecular interaction domain of the CAL permits specific binding of the CAL with a second biomolecule, e.g., a receptor on the immune killer cell. In some embodiments, the biomolecular interaction domain of the CAL is recognized by an endogenous receptor on the immune killer cell. In some embodiments, the biomolecular interaction domain of the CAL is recognized by an engineered receptor on the immune killer cell. Exemplary but non-limiting biomolecular interaction domains include FITC (which can be recognized by a FITC CAR-T cell system), a leucine zipper domain, a zinc finger domain, PSD95-Dlg1-zo-1 (PDZ) domains, a streptavidin domain and a streptavidin binding protein (SBP) domain, a gibberellin insensitive (GIA) and/or a gibberellin insensitive dwarf1 (GID1), a PYL domain and an ABI domain, a chemically-induced pair of interaction domains as described elsewhere herein, a Snap-tag, a Halo tag, a T14-3-3-cdeltaC and/or a C-Terminal peptide of PMA2 (CT52), a (R)-Phycoerythrin (R-PE/ PE) and/or a R-PE/PE binding protein a Fab domain, and/or an anti-CD3 domain.

[0009] As noted, the CALs described herein can be used in combination with CAR-T systems. Exemplary but non-limiting CAR-T systems suitable for use with the methods and

compositions described herein include SUPRA CAR and SPLIT CAR. CAR-T systems are discussed in more detail elsewhere herein and are known in the art.

[0010] In one aspect of any of the embodiments, described herein is a composition, comprising: a) a TCR recognition domain; and one or both of: b) an intracellular signaling domain; and c) a biomolecular interaction domain (e.g., a first-type biomolecular interaction domain).

[0011] In one aspect of any of the embodiments, described herein is a composition comprising: a first polypeptide comprising at least a portion of a TCR recognition domain and a first-type biomolecular interaction domain; and a signaling polypeptide comprising a second-type biomolecular interaction domain and an intracellular signaling domain; wherein the first-type and second-type biomolecular interaction domains bind specifically to each other. In one aspect of any of the embodiments, described herein is a composition comprising: a first polypeptide comprising at least a portion of a TCR recognition domain and a first-type biomolecular interaction domain; and a recognition polypeptide comprising a second recognition domain and a third-type biomolecular interaction domain; wherein the first-type and third-type biomolecular interaction domains bind specifically to each other. In one aspect of any of the embodiments, described herein is a composition comprising: a first polypeptide comprising at least a portion of a TCR recognition domain and a first-type biomolecular interaction domain; and a signaling polypeptide comprising a second-type biomolecular interaction domain and an intracellular signaling domain; and a recognition polypeptide comprising a second recognition domain and a third-type biomolecular interaction domain; wherein the second-type and third-type biomolecular interaction domains compete for binding to the first-type biomolecular interaction domain. In some embodiments of any of the aspects, the third-type biomolecular interaction domain and first-type biomolecular interaction domain have a higher affinity for each other than the second-type biomolecular interaction domain and first-type biomolecular interaction domain.

[0012] In one aspect of any of the embodiments, described herein is a composition comprising: a first polypeptide comprising at least a portion of a TCR recognition domain and a first-type biomolecular interaction domain; a signaling polypeptide comprising a second-type biomolecular interaction domain, a fourth-type biomolecular interaction domain, and an intracellular signaling domain; and a recognition polypeptide comprising a second recognition domain and a fifth-type biomolecular interaction domain; wherein the first-type biomolecular interaction domain and the second-type biomolecular interaction domain bind specifically to each other; and wherein the fourth-type biomolecular interaction domain and the fifth-type biomolecular interaction domain bind specifically to each other. In some embodiments of any of the aspects, the fourth-type biomolecular interaction

domain and fifth-type biomolecular interaction domain have a weaker affinity than the second-type biomolecular interaction domain and first-type protein interaction domain. In some embodiments of any of the aspects, the first polypeptide further comprises a sixth-type biomolecular interaction domain and the recognition polypeptide further comprises a seventh-type biomolecular interaction domain which bind specifically to each other.

[0013] In some embodiments of any of the aspects, the first polypeptide comprises the entire TCR recognition domain. In some embodiments of any of the aspects, the TCR recognition domain comprises at least two separate polypeptide sequences, the first polypeptide comprises at least one of the separate polypeptide sequences of the TCR recognition domain, and the first polypeptide is bound to or complexed with a second or further polypeptide sequences of the TCR recognition domain to form a TCR recognition domain. In some embodiments of any of the aspects, the TCR recognition domain comprises a non-polypeptide component.

[0014] In some embodiments of any of the aspects, the second recognition domain is specific for a target that is not recognized by the TCR recognition domain. In some embodiments of any of the aspects, the second recognition domain is specific for a target that is found on a healthy and/or non-target cell and not on a diseased and/or target cell.

[0015] In some embodiments of any of the aspects, the TCR recognition domain comprises a MHC (Major Histocompatibility Complex); a MHC-peptide complex; featureless peptide MHC; or a MHC-peptide fusion. In some embodiments of any of the aspects, the peptide is a human or non-human peptide. In some embodiments of any of the aspects, the peptide is a Minor Histocompatibility Antigen (MiHA). In some embodiments of any of the aspects, the MHC is a monomer, dimer, trimer, tetramer, pentamer, dextramer or other oligomer form. In some embodiments of any of the aspects, the MHC-peptide complex is a monomer, dimer, trimer, tetramer, pentamer, dextramer or other oligomer form. In some embodiments of any of the aspects, the MHC-peptide fusion is a monomer, dimer, trimer, tetramer, pentamer, dextramer or other oligomer form. In some embodiments of any of the aspects, the MHC is a dimer, trimer, tetramer, pentamer, dextramer or other oligomer form. In some embodiments of any of the aspects, the MHC-peptide complex is a dimer, trimer, tetramer, pentamer, dextramer or other oligomer form. In some embodiments of any of the aspects, the MHC-peptide fusion is a dimer, trimer, tetramer, pentamer, dextramer or other oligomer form. In some embodiments of any of the aspects, the MHC is a MHC class I or a MHC class II.

[0016] In some embodiments of any of the aspects, the TCR recognition domain comprises a CD1 domain or a CD1 domain-ligand complex or fusion. In some embodiments of any of the aspects, the CD1 is CD1d.

[0017] In some embodiments of any of the aspects, the biomolecular interaction domains are found on an extracellular portion of the respective polypeptides. In some embodiments of any of the aspects,

- a. wherein the biomolecular interaction domain(s) is a leucine zipper, or any binding pair of biomolecular interaction domains are collectively a pair of leucine zippers;
- b. wherein the biomolecular interaction domain(s) is a BZip (RR) and/or a AZip (EE), or any binding pair of biomolecular interaction domains are collectively a BZip (RR) and a AZip (EE);
- c. wherein the biomolecular interaction domain(s) is a PSD95-Dlg1-zo-1 (PDZ) domain;
- d. wherein the biomolecular interaction domain(s) is a streptavidin and/or a streptavidin binding biomolecular (SBP) or any binding pair of biomolecular interaction domains are collectively a streptavidin and a streptavidin binding biomolecular (SBP);
- e. wherein the biomolecular interaction domain(s) is a FKBP-binding domain of mTOR (FRB) and/or a FK506 binding biomolecular (FKBP) or any binding pair of biomolecular interaction domains are collectively a FKBP-binding domain of mTOR (FRB) and a FK506 binding biomolecular (FKBP);
- f. wherein the biomolecular interaction domain(s) is a cyclophilin-Fas fusion biomolecular (CyP-Fas) and/or a FK506 binding biomolecular (FKBP) or any binding pair of biomolecular interaction domains are collectively a cyclophilin-Fas fusion biomolecular (CyP-Fas) and a FK506 binding biomolecular (FKBP);
- g. wherein the biomolecular interaction domain(s) is a calcineurin A (CNA) and/or a FK506 binding biomolecular (FKBP) or any binding pair of biomolecular interaction domains are collectively a calcineurin A (CNA) and a FK506 binding biomolecular (FKBP);
- h. wherein the biomolecular interaction domain(s) is a gibberellin insensitive (GIA) and/or a gibberellin insensitive dwarf1 (GID1) or any binding pair of biomolecular interaction domains are collectively a gibberellin insensitive (GIA) and a gibberellin insensitive dwarf1 (GID1);
- i. wherein the biomolecular interaction domain(s) is a Snap-tag and/or a Halo tag, or any binding pair of biomolecular interaction domains are collectively a Snap-tag and a Halo tag;
- j. wherein the biomolecular interaction domain(s) is a T14-3-3-cdeltaC and/or a C-Terminal peptides of PMA2 (CT52), or any binding pair of biomolecular interaction domains are collectively a T14-3-3-cdeltaC and a C-Terminal peptides of PMA2 (CT52);

- k. wherein the biomolecular interaction domain(s) is a PYL and/or a ABI, or any binding pair of biomolecular interaction domains are collectively a PYL and a ABI;
- l. wherein the biomolecular interaction domain(s) is a nucleotide tag and/or a zinc finger domain, or any binding pair of biomolecular interaction domains are collectively a nucleotide tag and a zinc finger domain;
- m. wherein the biomolecular interaction domain(s) is a nucleotide tag, or any binding pair of biomolecular interaction domains are collectively a pair of nucleotide tags;
- n. wherein the biomolecular interaction domain(s) is a Fluorescein isothiocyanate (FITC) and/or a FITC binding biomolecular or any binding pair of protein interaction domains are collectively a FITC and a FITC binding protein; and/or
- o. wherein the protein interaction domain(s) is a (R)-Phycoerythrin (R-PE/ PE) and/or a R-PE/PE binding protein or any binding pair of protein interaction domains are collectively a (R)-Phycoerythrin (R-PE/ PE) and a R-PE/PE binding protein.

In some embodiments of any of the aspects, the nucleotide tag is a DNA tag or dsDNA tag.

[0018] In some embodiments of any of the aspects, the intracellular signaling domain comprises or is a signaling domain from one or more proteins selected from the group consisting of: TCR ζ , FcR γ , FcR β , CD3 γ , CD35, CD3 ζ , CD3C, CD22, CD79a, CD79b, CD66d, CARD11, CD2, CD7, CD27, CD28, CD30, CD40, CD54 (ICAM), CD83, CD134 (OX40), CD137 (4-1BB), CD150 (SLAMF1), CD152 (CTLA4), CD223 (LAG3), CD270 (HVEM), CD273 (PD-L2), CD274 (PD- L1), CD278 (ICOS), DAP10, LAT, KD2C SLP76, TRIM, and ZAP70.

[0019] In one aspect of any of the embodiments, described herein is a cell comprising and/or expressing a composition described herein. In one aspect of any of the embodiments, described herein is a composition comprising a first polypeptide of any of the preceding claims and a cell expressing or comprising the signaling polypeptide of any of the preceding claims.

[0020] In some embodiments of any of the aspects, the TCR recognition domain comprises a MHC allogeneic, autologous, or xenogeneic to the cell. In some embodiments of any of the aspects, the TCR recognition domain comprises a synthetic MHC. In some embodiments of any of the aspects, the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is allogeneic, autologous, or xenogeneic to the cell. In some embodiments of any of the aspects, the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is synthetic.

[0021] In some embodiments of any of the aspects, the cell is a NK cell, dendritic cell, regulatory T cell, or effector T cell. In some embodiments of any of the aspects, the cell is engineered to express one of more of the polypeptide(s) of the composition. In some

embodiments of any of the aspects, the cell is engineered to express the signaling polypeptide of the composition. In some embodiments of any of the aspects, the cell is further engineered to knockout or knockdown the native MHC I/II. In some embodiments of any of the aspects, the cell is further engineered to knockdown the native MHC I/II expressed on the cell surface.

[0022] In one aspect of any of the embodiments, described herein is a composition, comprising a TCR recognition domain and an intracellular signaling domain but not comprising a biomolecular interaction domain (e.g., a first-type biomolecular interaction domain). In one aspect of any of the embodiments, described herein is a composition, comprising a TCR recognition domain and a biomolecular interaction domain (e.g., a first-type biomolecular interaction domain) and but not comprising an intracellular signaling domain.

[0023] In one aspect of any of the embodiments, described herein is a method of treating or preventing an autoimmune disease or condition; T cell mediated inflammation or immune response; malignant T cell condition; transplant rejection; or GvHD in a subject in need thereof, the method comprising administering to the subject a composition and/or cell of any of the preceding claims. In some embodiments of any of the aspects, the TCR recognition domain comprises a MHC allogeneic to the subject.

[0024] In some embodiments of any of the aspects, the TCR recognition domain comprises a MHC autologous to the transplant cells. In some embodiments of any of the aspects, the TCR recognition domain comprises a MHC xenogeneic to the transplant cells. In some embodiments of any of the aspects, the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is allogeneic to the subject. In some embodiments of any of the aspects, the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is autologous to the transplant cells. In some embodiments of any of the aspects, the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is autologous to the transplant cells. In some embodiments of any of the aspects, the transplant is any human or non-human cell, tissue, or organ. In some embodiments of any of the aspects, the transplant is an allogeneic hematopoietic stem cell or solid organ transplantation.

[0025] In some embodiments of any of the aspects, the malignant T cell condition is T cell acute lymphoblastic leukemia or T cell lymphoblastic lymphoma.

[0026] In some embodiments of any of the aspects, the autoimmune disease is type 1 diabetes, vitiligo, multiple sclerosis, alopecia, celiac disease, pemphigus, rheumatoid arthritis, or scleroderma. In some embodiments of any of the aspects, the autoimmune disease is thyroiditis, type 1 diabetes mellitus, Hashimoto's thyroiditis, Graves' disease, celiac disease, multiple sclerosis, Guillain-Barre syndrome, Addison's disease, and

Raynaud's phenomenon, Goodpasture's disease, arthritis (rheumatoid arthritis such as acute arthritis, chronic rheumatoid arthritis, gout or gouty arthritis, acute gouty arthritis, acute immunological arthritis, chronic inflammatory arthritis, degenerative arthritis, type II collagen-induced arthritis, infectious arthritis, Lyme arthritis (e.g., post treatment Lyme disease syndrome), proliferative arthritis, psoriatic arthritis, Still's disease, vertebral arthritis, and juvenile-onset rheumatoid arthritis, arthritis chronica progrediente, arthritis deformans, polyarthritis chronica primaria, reactive arthritis, and ankylosing spondylitis), palindromic arthritis, inflammatory hyperproliferative skin diseases, psoriasis such as plaque psoriasis, guttate psoriasis, pustular psoriasis, and psoriasis of the nails, atopy including atopic diseases such as hay fever and Job's syndrome, dermatitis including contact dermatitis, chronic contact dermatitis, exfoliative dermatitis, allergic dermatitis, allergic contact dermatitis, dermatitis herpetiformis, nummular dermatitis, seborrheic dermatitis, non-specific dermatitis, primary irritant contact dermatitis, and atopic dermatitis, x-linked hyper IgM syndrome, allergic intraocular inflammatory diseases, urticaria such as chronic allergic urticaria and chronic idiopathic urticaria, including chronic autoimmune urticaria, myositis, polymyositis/dermatomyositis, juvenile dermatomyositis, toxic epidermal necrolysis, scleroderma (including systemic scleroderma), sclerosis such as systemic sclerosis, multiple sclerosis (MS) such as spino-optical MS, primary progressive MS (PPMS), and relapsing remitting MS (RRMS), progressive systemic sclerosis, atherosclerosis, arteriosclerosis, sclerosis disseminata, ataxic sclerosis, neuromyelitis optica (NMO), inflammatory bowel disease (IBD) (for example, Crohn's disease, autoimmune-mediated gastrointestinal diseases, colitis such as ulcerative colitis, colitis ulcerosa, microscopic colitis, collagenous colitis, colitis polyposa, necrotizing enterocolitis, and transmural colitis, and autoimmune inflammatory bowel disease), bowel inflammation, pyoderma gangrenosum, erythema nodosum, primary sclerosing cholangitis, respiratory distress syndrome, including adult or acute respiratory distress syndrome (ARDS), meningitis, inflammation of all or part of the uvea, iritis, choroiditis, an autoimmune hematological disorder, rheumatoid spondylitis, rheumatoid synovitis, hereditary angioedema, cranial nerve damage as in meningitis, herpes gestationis, pemphigoid gestationis, pruritis scroti, autoimmune premature ovarian failure, sudden hearing loss due to an autoimmune condition, IgE-mediated diseases such as anaphylaxis and allergic and atopic rhinitis, encephalitis such as Rasmussen's encephalitis and limbic and/or brainstem encephalitis, uveitis, such as anterior uveitis, acute anterior uveitis, granulomatous uveitis, nongranulomatous uveitis, phacoantigenic uveitis, posterior uveitis, or autoimmune uveitis, glomerulonephritis (GN) with and without nephrotic syndrome such as chronic or acute glomerulonephritis such as primary GN, immune-mediated GN, membranous GN (membranous nephropathy), idiopathic membranous GN or

idiopathic membranous nephropathy, membrano- or membranous proliferative GN (MPGN), including Type I and Type II, and rapidly progressive GN, proliferative nephritis, autoimmune polyglandular endocrine failure, balanitis including balanitis circumscripta plasmacellularis, balanoposthitis, erythema annulare centrifugum, erythema dyschromicum perstans, erythema multiform, granuloma annulare, lichen nitidus, lichen sclerosus et atrophicus, lichen simplex chronicus, lichen spinulosus, lichen planus, lamellar ichthyosis, epidermolytic hyperkeratosis, premalignant keratosis, pyoderma gangrenosum, allergic conditions and responses, allergic reaction, eczema including allergic or atopic eczema, asteatotic eczema, dyshidrotic eczema, and vesicular palmoplantar eczema, asthma such as asthma bronchiale, bronchial asthma, and auto-immune asthma, conditions involving infiltration of T cells and chronic inflammatory responses, immune reactions against foreign antigens such as fetal A-B-O blood groups during pregnancy, chronic pulmonary inflammatory disease, autoimmune myocarditis, leukocyte adhesion deficiency, lupus, including lupus nephritis, lupus cerebritis, pediatric lupus, non-renal lupus, extra-renal lupus, discoid lupus and discoid lupus erythematosus, alopecia lupus, systemic lupus erythematosus (SLE) such as cutaneous SLE or subacute cutaneous SLE, neonatal lupus syndrome (NLE), and lupus erythematosus disseminatus, juvenile onset (Type I) diabetes mellitus, including pediatric insulin-dependent diabetes mellitus (IDDM), adult onset diabetes mellitus (Type II diabetes), autoimmune diabetes, idiopathic diabetes insipidus, diabetic retinopathy, diabetic nephropathy, diabetic large-artery disorder, immune responses associated with acute and delayed hypersensitivity mediated by cytokines and T-lymphocytes, sarcoidosis, granulomatosis including lymphomatoid granulomatosis, Wegener's granulomatosis, agranulocytosis, vasculitides, including vasculitis, large-vessel vasculitis (including polymyalgia rheumatica and giant-cell (Takayasu's) arteritis), medium-vessel vasculitis (including Kawasaki's disease and polyarteritis nodosa/periarteritis nodosa), microscopic polyarteritis, immunovascularitis, CNS vasculitis, cutaneous vasculitis, hypersensitivity vasculitis, necrotizing vasculitis such as systemic necrotizing vasculitis, and ANCA-associated vasculitis, such as Churg-Strauss vasculitis or syndrome (CSS) and ANCA-associated small-vessel vasculitis, temporal arteritis, autoimmune aplastic anemia, Coombs positive anemia, Diamond Blackfan anemia, hemolytic anemia or immune hemolytic anemia including autoimmune hemolytic anemia (AIHA), pernicious anemia (anemia perniciosa), Addison's disease, pure red cell anemia or aplasia (PRCA), Factor VIII deficiency, hemophilia A, autoimmune neutropenia, pancytopenia, leukopenia, diseases involving leukocyte diapedesis, CNS inflammatory disorders, multiple organ injury syndrome such as those secondary to septicemia, trauma or hemorrhage, antigen-antibody complex-mediated diseases, anti-glomerular basement membrane disease, anti-phospholipid

antibody syndrome, allergic neuritis, Behcet's disease/syndrome, Castleman's syndrome, Goodpasture's syndrome, Reynaud's syndrome, Sjogren's syndrome, Stevens-Johnson syndrome, pemphigoid such as pemphigoid bullous and skin pemphigoid, pemphigus (including pemphigus vulgaris, pemphigus foliaceus, pemphigus mucus-membrane pemphigoid, and pemphigus erythematosus), autoimmune polyendocrinopathies, Reiter's disease or syndrome, an immune complex disorder such as immune complex nephritis, antibody-mediated nephritis, polyneuropathies, chronic neuropathy such as IgM polyneuropathies or IgM-mediated neuropathy, and autoimmune or immune-mediated thrombocytopenia such as idiopathic thrombocytopenic purpura (ITP) including chronic or acute ITP, scleritis such as idiopathic cerato-scleritis, episcleritis, autoimmune disease of the testis and ovary including autoimmune orchitis and oophoritis, primary hypothyroidism, hypoparathyroidism, autoimmune endocrine diseases including thyroiditis such as autoimmune thyroiditis, Hashimoto's disease, chronic thyroiditis (Hashimoto's thyroiditis), or subacute thyroiditis, idiopathic hypothyroidism, Grave's disease, polyglandular syndromes such as autoimmune polyglandular syndromes (or polyglandular endocrinopathy syndromes), paraneoplastic syndromes, including neurologic paraneoplastic syndromes such as Lambert-Eaton myasthenic syndrome or Eaton-Lambert syndrome, stiff-man or stiff-person syndrome, encephalomyelitis such as allergic encephalomyelitis or encephalomyelitis allergica and experimental allergic encephalomyelitis (EAE), myasthenia gravis such as thymoma-associated myasthenia gravis, cerebellar degeneration, neuromyotonia, opsoclonus or opsoclonus myoclonus syndrome (OMS), and sensory neuropathy, multifocal motor neuropathy, Sheehan's syndrome, autoimmune hepatitis, lupoid hepatitis, giant-cell hepatitis, autoimmune chronic active hepatitis, lymphoid interstitial pneumonitis (LIP), bronchiolitis obliterans (non-transplant) vs NSIP, Guillain-Barre syndrome, Berger's disease (IgA nephropathy), idiopathic IgA nephropathy, linear IgA dermatosis, acute febrile neutrophilic dermatosis, subcorneal pustular dermatosis, transient acantholytic dermatosis, cirrhosis such as primary biliary cirrhosis and pneumonocirrhosis, autoimmune enteropathy syndrome, Celiac or Coeliac disease, celiac sprue (gluten enteropathy), refractory sprue, idiopathic sprue, cryoglobulinemia, amyotrophic lateral sclerosis (ALS; Lou Gehrig's disease), coronary artery disease, autoimmune ear disease such as autoimmune inner ear disease (AIED), autoimmune hearing loss, polychondritis such as refractory or relapsed or relapsing polychondritis, pulmonary alveolar proteinosis, Cogan's syndrome/nonsyphilitic interstitial keratitis, Bell's palsy, Sweet's disease/syndrome, rosacea autoimmune, zoster-associated pain, amyloidosis, a non-cancerous lymphocytosis, a primary lymphocytosis, which includes monoclonal B cell lymphocytosis (e.g., benign monoclonal gammopathy and monoclonal gammopathy of

undetermined significance, MGUS), peripheral neuropathy, paraneoplastic syndrome, channelopathies including channelopathies of the CNS, autism, inflammatory myopathy, focal or segmental or focal segmental glomerulosclerosis (FSGS), endocrine ophthalmopathy, uveoretinitis, chorioretinitis, autoimmune hepatological disorder, fibromyalgia, multiple endocrine failure, Schmidt's syndrome, adrenalitis, gastric atrophy, presenile dementia, demyelinating diseases such as autoimmune demyelinating diseases and chronic inflammatory demyelinating polyneuropathy, Dressler's syndrome, alopecia areata, alopecia totalis, CREST syndrome (calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia), male and female autoimmune infertility, e.g., due to anti-spermatozoan antibodies, mixed connective tissue disease, Chagas' disease, rheumatic fever, recurrent abortion, farmer's lung, erythema multiforme, post-cardiotomy syndrome, Cushing's syndrome, bird-fancier's lung, allergic granulomatous angiitis, benign lymphocytic angiitis, Alport's syndrome, alveolitis such as allergic alveolitis and fibrosing alveolitis, interstitial lung disease, transfusion reaction, Sampter's syndrome, Caplan's syndrome, endocarditis, endomyocardial fibrosis, diffuse interstitial pulmonary fibrosis, interstitial lung fibrosis, pulmonary fibrosis, idiopathic pulmonary fibrosis, cystic fibrosis, endophthalmitis, erythema elevatum et diutinum, erythroblastosis fetalis, eosinophilic facitis, Shulman's syndrome, Felty's syndrome, cyclitis such as chronic cyclitis, heterochronic cyclitis, iridocyclitis (acute or chronic), or Fuch's cyclitis, Henoch-Schonlein purpura, SCID, sepsis, endotoxemia, post-vaccination syndromes, Evan's syndrome, autoimmune gonadal failure, Sydenham's chorea, post-streptococcal nephritis, thromboangitis obliterans, thyrotoxicosis, tabes dorsalis, chorioiditis, giant-cell polymyalgia, chronic hypersensitivity pneumonitis, keratoconjunctivitis sicca, idiopathic nephritic syndrome, minimal change nephropathy, benign familial and ischemia-reperfusion injury, transplant organ reperfusion, retinal autoimmunity, aphthae, aphthous stomatitis, arteriosclerotic disorders, aspermiogenesis, autoimmune hemolysis, Boeck's disease, enteritis allergica, erythema nodosum leprosum, idiopathic facial paralysis, chronic fatigue syndrome, febris rheumatica, Hamman-Rich's disease, sensorineural hearing loss, ileitis regionalis, leucopenia, transverse myelitis, primary idiopathic myxedema, ophthalmia sympathica, polyradiculitis acuta, pyoderma gangrenosum, acquired splenic atrophy, vitiligo, toxic-shock syndrome, conditions involving infiltration of T cells, leukocyte-adhesion deficiency, immune responses associated with acute and delayed hypersensitivity mediated by cytokines and T-lymphocytes, diseases involving leukocyte diapedesis, multiple organ injury syndrome, antigen-antibody complex-mediated diseases, antiglomerular basement membrane disease, allergic neuritis, autoimmune polyendocrinopathies, oophoritis, primary myxedema, autoimmune atrophic gastritis, rheumatic diseases, mixed connective tissue

disease, nephrotic syndrome, insulinitis, polyendocrine failure, autoimmune polyglandular syndrome type I, adult-onset idiopathic hypoparathyroidism (AOIH), myocarditis, nephrotic syndrome, primary sclerosing cholangitis, acute or chronic sinusitis, ethmoid, frontal, maxillary, or sphenoid sinusitis, an eosinophil-related disorder such as eosinophilia, pulmonary infiltration eosinophilia, eosinophilia-myalgia syndrome, Löffler's syndrome, chronic eosinophilic pneumonia, tropical pulmonary eosinophilia, granulomas containing eosinophils, seronegative spondyloarthritides, polyendocrine autoimmune disease, sclerosing cholangitis, sclera, episclera, Bruton's syndrome, transient hypogammaglobulinemia of infancy, Wiskott-Aldrich syndrome, ataxia telangiectasia syndrome, angiectasis, autoimmune disorders associated with collagen disease, rheumatism, allergic hypersensitivity disorders, glomerulonephritides, reperfusion injury, ischemic reperfusion disorder, lymphomatous tracheobronchitis, inflammatory dermatoses, dermatoses with acute inflammatory components, and autoimmune uveoretinitis (AUR).

[0027] In some embodiments of any of the aspects, the T cell mediated immune response is an anti-drug specific response to a biologic, cell therapy, and/or gene therapy. In some embodiments of any of the aspects, the biologic, cell-therapy, or gene therapy is an adeno-associated virus (AAV) gene therapy, a genome editing agent, or enzyme replacement therapy.

[0028] In some embodiments of any of the aspects, the disease is type 1 diabetes and the TCR recognition domain comprises sequences with at least 80% or at least 95% sequence identity to: one or more of SEQ ID NOs: 8-17; HLA-A*0201 and at least one of SEQ ID NOs: 2013-2016 and 2031-2033; or HLA-A*02:01 and at least one of SEQ ID NOs: 20128-2129. In some embodiments of any of the aspects, the disease is vitiligo and the TCR recognition domain comprises sequences with at least 80% or at least 95% sequence identity to: SEQ ID NO: 18, 19, and one of 20-22; or comprises HLA-A*0201 and SEQ ID NO: 2018; or HLA-A*0301 and SEQ ID NO: 2019; or comprises HLA-A*2402 and SEQ ID NO: 2020; or HLA-A*0101 and SEQ ID NO: 2021. In some embodiments of any of the aspects, the method is a method of treating and/or preventing GvHD and the TCR recognition domain comprises sequences with at least 80% or at least 95% sequence identity to: HLA-A*0101 and at least one of SEQ ID NOs: 2034-2037; or HLA-B*0702 and SEQ ID NO: 2038; or HLA-B*0801 and SEQ ID NO: 2039. In some embodiments of any of the aspects, the disease is type 1 diabetes and the TCR recognition domain comprises one or more of SEQ ID NOs: 8-17; comprises HLA-A*0201 and at least one of SEQ ID NOs: 2013-2016 and 2031-2033; or comprises HLA-A*02:01 and at least one of SEQ ID NOs: 20128-2129. In some embodiments of any of the aspects, the disease is vitiligo and the TCR recognition domain comprises SEQ ID NO: 18, 19, and one of 20-22; or comprises HLA-

A*0201 and SEQ ID NO: 2018; or comprises HLA-A*0301 and SEQ ID NO: 2019; or comprises HLA-A*2402 and SEQ ID NO: 2020; or comprises HLA-A*0101 and SEQ ID NO: 2021. In some embodiments of any of the aspects, the method is a method of treating and/or preventing GvHD and the TCR recognition domain comprises HLA-A*0101 and at least one of SEQ ID NOS: 2034-2037; or comprises HLA-B*0702 and SEQ ID NO: 2038; or comprises HLA-B*0801 and SEQ ID NO: 2039.

BRIEF DESCRIPTION OF THE DRAWINGS

[0029] Fig. 1 demonstrates that pMHC tetramer + FITC (adaptor) binds to the target cells (OTi) in a dose dependent fashion or manner. I-H2Kb: MHC class I tetramer. I-Ab- : Control tetramer. Figure discloses SEQ ID NOS 2752-2753, 2750 and 2754, respectively, in order of appearance.

[0030] Fig. 2 depicts expression of activation marker CD69 on OTi cells at different time points (24, 48, 72 hrs.). Binding of Jurkat+ pMHC to OTi cells does not change Jurkat live count (right). Figure discloses SEQ ID NOS 2750, 2750 and 2754, respectively, in order of appearance.

[0031] Fig. 3 depicts the expression of CD69 on Jurkat CAR, e.g., CAL T cells in different time points (24, 48, 72 hrs.). Figure discloses SEQ ID NOS 2750 and 2755, respectively, in order of appearance.

[0032] Fig. 4 depicts the cytotoxicity of Primary human CD8 CAR, , e.g., CAL T cells with different concentrations of tetramers (left). Minimal activation of target cells (CD69 on CAR, e.g., CAL T cells and target cells) after coculturing with OTi cells (figure 2left). I-Ab: Ctrl/ H2Kb: MHC-I. Figure discloses SEQ ID NOS 2750, 2754 and 2750, respectively, in order of appearance.

[0033] Fig. 5 demonstrates that the cytotoxicity of human CD8 CAR, e.g., CAL T is highly specific and was not seen with ctrl tetramer (left). No cytotoxicity was seen on killer CAR, e.g., CAL T cells or bystander CD4 T Cells after co-culturing with pMHC and splenocytes. Figure discloses SEQ ID NOS 2754, 2750, 2754, 2750, 2754 and 2750, respectively, in order of appearance.

[0034] Fig. 6A depicts a schematic of SUPRA CAR design applied to provide Universal CAL. This design separates the cell targeting molecule module from the killer cell. Fig. 6B depicts that SUPRA CARs can be designed with CD3ζ domain uncoupled from the costimulatory domains to provide Universal CAL as described herein.

[0035] Figs. 7A-7D depict key features of the SUPRA CAR systems that can be applied to Universal CAL. Fig. 7A demonstrates that zipCAR activation is tunable through modulation of zipFv concentration, zipper affinity, scFv affinity, and zipCAR expression level in human primary CD4 T cells, as demonstrated by IFN-γ production. Fig. 7B demonstrates that SUPRA CAR system, as applied to Universal CAL, can perform combination antigen detection to form AND gate logic in CD4 T cells. Fig. 7C demonstrates that xenograft animal tumor model shows tumor eradication (as demonstrated by luciferase photon flux given by the tumor cells) by the SUPRA CAR T cells. Fig. 7D demonstrates that SUPRA CARs as applied to Universal CAL can be used to control different cell

types, such as CD4 and CD8 T cells, against two different antigens. CD69 expression (a T cell activation marker) is quantified with flow cytometry for CD4 and CD8 T cells. (From Cho, Collins, and Wong, Cell. 2018)

[0036] Fig. 8 depicts the timeline of double Hu-PBMC-HSCT-skin graft mouse model generation.

[0037] Fig. 9 depicts a summary of double hu-PBMC-HSCT-skin graft mouse model generation.

[0038] Fig. 10 depicts key features of pMHC multimer + CAR, e.g., CAL T cell system.

[0039] Fig. 11 depicts a table of experimental design. Figure discloses SEQ ID NOS 2750, 2756 and 2754, respectively, in order of appearance.

[0040] Fig. 12 demonstrates verification of FITC-conjugated tetramer mediated activation. Figure discloses SEQ ID NOS 2750 and 2751, respectively, in order of appearance.

[0041] Fig. 13 depicts a time course of FITC-conjugated tetramer mediated activation. Figure discloses SEQ ID NOS 2750, 2751, 2750, 2751, 2750, 2751, 2750 and 2751, respectively, in order of appearance.

[0042] Fig. 14 depicts a graph of Jurkat cell counts. Figure discloses SEQ ID NOS 2750 and 2754, respectively, in order of appearance.

[0043] Fig. 15 depicts tetramer staining. Figure discloses SEQ ID NOS 2750, 2754, 2751, 2756, 2754, 2750 and 2754, from left to right and top to bottom.

[0044] Fig. 16 depicts heatmaps of indicated staining levels. Figure discloses SEQ ID NOS 2751, 2757, 2754, 2750, 2754, 2751, 2754, 2757, 2750, 2754, 2751, 2754, 2757, 2750 and 2754, respectively, in order of appearance.

[0045] Fig. 17 depicts a table of experimental design.

[0046] Figs. 18-20 depict graphs of cytotoxicity levels. Fig. 18 discloses SEQ ID NOS 2754, 2750, 2754 and 2750 from left to right. Fig. 19 discloses SEQ ID NOS 2754 and 2750, respectively, in order of appearance. Fig. 20 discloses SEQ ID NOS 2750, 2754, 2750 and 2754 from left to right.

[0047] Fig. 21 depicts the levels of CD69 on OTi CD8 T cells. Figure discloses SEQ ID NOS 2750, 2754, 2750 and 2754 from left to right.

[0048] Fig. 22 depicts schematics of two embodiments of the technology described herein.

[0049] Fig. 23 depicts a schematic of the FU-CAL embodiments of the technology described herein.

[0050] Fig. 24 depicts a schematic of the CAL-BITE embodiments of the technology described herein. The left panel depicts blinatumomab, which is described in more detail in Weiner et al. The Molecular Basis of Cancer 2015 683-694.e3.

[0051] Fig. 25 depicts a schematic of the CAL technology disarming autoreactive T cells.

[0052] Figs. 26A-26B depict schematics of T cells design. MHC can be mouse or human. The MiHA can be ovalbumin (against OTi or OTii) or disparate antigens between donor and recipient.

[0053] Fig. 27 depicts a graph demonstrating that pMHC tetramer + FITC (adaptor) binds to the target cells (OTi) in a dose dependent fashion, while OTii specific tetramer does not.

[0054] Fig. 28A depicts expression of CD69 on OTi cells at different time points (24, 48, 72 hrs.). Fig. 28B is a graph demonstrating that binding of Jurkat+ pMHC to OTi cells does not change Jurkat live count.

[0055] Fig. 29 depicts a graph demonstrating that cytotoxicity of pMHC-CAR against 1E6 T cell clone exhibits a dose dependent association.

[0056] Fig. 30 depicts a graph demonstrating that CAR Jurkat T cells activation (as measured by CD69 surface marker) after exposure to target cells (OTi or OTii TCR expressing T cells) and the corresponding tetramer (OTi or OTii specific) (n=4).

[0057] Fig. 31 depicts graphs demonstrating that cytotoxicity of human CD8 CAR T is highly specific and was not seen with ctrl tetramer. No cytotoxicity was seen on killer CAR T cells or bystander CD4 T Cells after co-culturing with pMHC and splenocytes (n=4).

[0058] Fig. 32 depicts a graph demonstrating cytotoxicity of Primary CD8 CAR T cells against OTi TCR T cells with different concentration of tetramer.

DETAILED DESCRIPTION

[0059] Aspects of the invention described herein relate to chimeric antigen ligands. As used herein, “chimeric antigen ligand” or “CAL” refers to an artificially constructed molecule comprising a TCR recognition domain (e.g. an polypeptide comprising at least one MHC sequence as described herein) and at least one biomolecular interaction domain. The TCR recognition domain is selected to bind to specific populations of T cells that it is desirable to target and/or destroy, e.g., for therapeutic purposes in T-cell mediated diseases. In some embodiments, the population of targeted T cells is a population of polyclonal pathogenic T cells. The biomolecular interaction domain is selected to bind to a second cell, e.g., a NK cell, thereby colocalizing the targeted T cell and the second cell and promoting or increasing the inhibition and/or destruction of the targeted T cell. In various embodiments, the CAL is selected to have high affinity or avidity for the TCR, e.g., the TCR variable domain.

[0060] The CALs can be used herein with endogenous cells, e.g., in some embodiments, no engineered cells are administered to the subject. In other embodiments, the CALs can be used with engineered cells, e.g., engineered NK cells. In such cases, the engineered cells can comprise one or more CARs, e.g., a CAR comprising an extracellular domain with a biomolecular interaction domain that specifically binds with the biomolecular interaction domain of the CAL.

[0061] Accordingly, described herein are chimeric antigen receptors (CARs) in which the recognition and signaling portions of the CAR are separate polypeptides. The two separate polypeptides that make up a complete CAR are able to interact and form the complete CAR by way of

protein interaction domains. This permits flexible, modular CAR-T therapy which is capable of complex logic computation, providing a more precise and effective approach to immunotherapy.

[0062] In one aspect of any of the embodiments is a CAL and/or chimeric antigen receptor (CAR) having multiple components, and/or a cell or composition comprising a multi-component CAL and/or CAR. Multi-component CALs/CARs are also referred to herein variously as SMART CAL/CAR or SUPRA.

[0063] As used herein, traditional “chimeric antigen receptor” or “CAR” refers to an artificially constructed hybrid polypeptide comprising an antigen-binding domain (e.g. an antigen-binding portion of an antibody (e.g. a scFv)) linked to a cell signaling and/or cell activation domain. In some embodiments the cell-signaling domain can be a T-cell signaling domain. In some embodiments, the cell activation domain can be a T-cell activation domain. CARs have the ability to redirect the specificity and reactivity of T cells and other immune cells (e.g., NK cells) toward a selected target in a non-MHC-restricted manner, exploiting the antigen-binding properties of monoclonal antibodies.

[0064] The non-MHC-restricted antigen recognition gives T-cells expressing CARs the ability to recognize an antigen independent of antigen processing, thus bypassing a major mechanism of tumor escape. Moreover, when expressed in T-cells, CARs advantageously do not dimerize with endogenous T-cell receptor (TCR) alpha and beta chains. Most commonly, the CAR's extracellular binding domain is composed of a single chain variable fragment (scFv) derived from fusing the variable heavy and light regions of a murine or humanized monoclonal antibody. Alternatively, scFvs may be used that are derived from Fabs (instead of from an antibody, e.g., obtained from Fab libraries), in various embodiments, this scFv is fused to a transmembrane domain and then to an intracellular signaling domain. "First- generation" CARs include those that solely provide CD3zeta signals upon antigen binding, "Second- generation" CARs include those that provide both costimulation (e.g. CD28 or CD 137) and activation (CD3Q). "Third-generation" CARs include those that provide multiple costimulation (e.g. CD28 and CD 137) and activation (CO3Q). In various embodiments, the CAR is selected to have high affinity or avidity for the antigen. Further discussion of CARs can be found, e.g., in Maus et al. Blood 2014 123:2624-35; Reardon et al. Neuro-Oncology 2014 16:1441-1458; Hoyos et al. Haematologica 2012 97:1622; Byrd et al. J Clin Oncol 2014 32:3039-47; Maher et al. Cancer Res 2009 69:4559-4562; and Tamada et al. Clin Cancer Res 2012 18:6436-6445; each of which is incorporated by reference herein in its entirety.

[0065] As used herein, “multi-component CAL” refers to a CAL comprising at least two separate polypeptides, neither of which polypeptides is capable of both ligand recognition and signaling activation on its own. As used herein, “multi-component CAR” refers to a CAR comprising at least two separate polypeptides, neither of which polypeptides is capable of both ligand recognition and signaling activation on its own. In some embodiments, the at least two separate polypeptides each comprise a protein interaction domain that permits interaction, e.g., binding of the separate

polypeptides. In some embodiments, one of the at least two separate polypeptides is a transmembrane polypeptide having an intracellular T cell receptor (TCR) signaling domain and a second of the at least two separate polypeptides is an extracellular polypeptide having a ligand-binding domain. In some embodiments, a multi-component CAL and/or CAR can comprise two, three, four, five, six, seven, eight, nine, ten or more separate polypeptides.

[0066] Various aspects provided herein provide a composition comprising multiple components of a multi-component CAL and/or CAR.

[0067] In one aspect of the embodiments is a composition, e.g., a single molecule, comprising a TCR recognition domain; and one or both of: (a) an intracellular signaling domain; and (b) a first-type protein interaction domain. In one aspect of the embodiments is a composition, e.g., a single molecule comprising a TCR recognition domain; and a first-type biomolecular (e.g., protein) interaction domain. Further provided herein is a multi-component CAL and/or CAR comprising a TCR recognition domain; and one or both of: (a) an intracellular signaling domain; and (b) a first-type protein interaction domain. In some embodiments, the composition, e.g, single molecule, comprising a TCR recognition domain; and a first-type biomolecular (e.g., protein) interaction domain does not comprise an antibody, antibody domain, or antibody reagent.

[0068] Another aspect of the embodiments is a composition comprising (a) a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain; and (b) a signaling polypeptide comprising a second-type protein interaction domain and an intracellular signaling domain; wherein the first-type and second-type protein interaction domains bind specifically to each other. Further provided herein is a multi-component CAL and/or CAR comprising a) a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain; and (b) a signaling polypeptide comprising a second-type protein interaction domain and an intracellular signaling domain; wherein the first-type and second-type protein interaction domains bind specifically to each other.

[0069] Another aspect of the embodiments is a composition comprising (a) a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain; and (b) a recognition polypeptide comprising a second recognition domain and a third-type protein interaction domain; wherein the first-type and third-type protein interaction domains bind specifically to each other. Further provided herein is a multi-component CAL and/or CAR comprising (a) a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain; and (b) a recognition polypeptide comprising a second recognition domain and a third-type protein interaction domain; wherein the first-type and third-type protein interaction domains bind specifically to each other

[0070] Another aspect of the embodiments is a composition comprising (a) a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain; and (b) a signaling

polypeptide comprising a second-type protein interaction domain and an intracellular signaling domain; and (c) a recognition polypeptide comprising a second recognition domain and a third-type protein interaction domain; wherein the second-type and third-type protein interaction domains compete for binding to the first-type protein interaction domain. Further provided herein is a multi-component CAL and/or CAR comprising (a) a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain; and (b) a signaling polypeptide comprising a second-type protein interaction domain and an intracellular signaling domain; and (c) a recognition polypeptide comprising a second recognition domain and a third-type protein interaction domain; wherein the second-type and third-type protein interaction domains compete for binding to the first-type protein interaction domain.

[0071] In various embodiments, the third-type protein interaction domain and first-type protein interaction domain have a higher affinity for each other than the second-type protein interaction domain and first-type protein interaction domain. Affinity can be measured by one skilled in the art using standard methods, for example, by measuring its equilibrium dissociation constant (K_d).

[0072] Another aspect of the embodiments is a composition comprising (a) a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain; (b) a signaling polypeptide comprising a second-type protein interaction domain, a fourth-type protein interaction domain, and an intracellular signaling domain; and (c) a recognition polypeptide comprising a second recognition domain and a fifth-type protein interaction domain; wherein the first-type protein interaction domain and the second-type protein interaction domain bind specifically to each other; and wherein the fourth-type protein interaction domain and the fifth-type protein interaction domain bind specifically to each other. Further provided herein is a multi-component CAL and/or CAR comprising (a) a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain; (b) a signaling polypeptide comprising a second-type protein interaction domain, a fourth-type protein interaction domain, and an intracellular signaling domain; and (c) a recognition polypeptide comprising a second recognition domain and a fifth-type protein interaction domain; wherein the first-type protein interaction domain and the second-type protein interaction domain bind specifically to each other; and wherein the fourth-type protein interaction domain and the fifth-type protein interaction domain bind specifically to each other.

[0073] In various embodiments, the fourth-type protein interaction domain and fifth-type protein interaction domain have a weaker affinity than the second-type protein interaction domain and first-type protein interaction domain. Affinity can be measured as described above.

[0074] In various embodiments, the first polypeptide further comprises a sixth-type protein interaction domain and the recognition polypeptide further comprises a seventh-type protein interaction domain which bind specifically to each other.

[0075] In some embodiments, the first polypeptide comprises the entire TCR recognition domain. In some embodiments, the TCR recognition domain comprises at least two separate polypeptide sequences, the first polypeptide comprises at least one of the separate polypeptide sequences of the TCR recognition domain, and the first polypeptide is bound to or complexed with a second or further polypeptide sequences of the TCR recognition domain to form a TCR recognition domain.

[0076] In some embodiments, a composition described herein can comprise multiple copies or instances of a TCR recognition domain(s), e.g. the TCR recognition domain can be a multimer, or oligomer. In some embodiments, a composition described herein can comprise multiple copies or instances of a first polypeptide as described herein.

[0077] In various embodiments, the second recognition domain is specific for a target that is not recognized by the TCR recognition domain. In one embodiment, the second recognition domain is specific for a target that is found on a healthy and/or non-target cell and not on a diseased and/or target cell.

[0078] As used herein, “TCR recognition domain” refers to a domain or portion of a polypeptide that can target or bind specifically to a TCR, e.g., a TCR expressed on the surface of T cell. In some embodiments, the TCR recognition domain can be a TCR variable region (TCR-VR) recognition domain, i.e. is can target or bind specifically to the variable region of a TCR, e.g., a TCR expressed on the surface of a T cell. The TCR recognition domain sequence can be autologous, allogeneic, or xenogeneic to a given subject. In some embodiments, the TCR recognition domain sequence is a wild-type protein or sequence. In some embodiments, the TCR recognition domain sequence is a naturally-occurring variant, e.g., an allele of a wild-type protein or sequence. In some embodiments, the TCR recognition domain sequence is modified relative to a wild-type protein, e.g. chemically modified. In some embodiments, the TCR recognition domain sequence is a derivative and/or variant of a wild-type sequence. In some embodiments, the TCR recognition domain sequence can be a human, or non-human sequence.

[0079] A TCR recognition domain can comprise a MHC polypeptide, a MHC polypeptide sequence, and/or comprise a portion of a MHC sequence. The MHC and/or MHC sequence can be autologous, allogeneic, or xenogeneic to a given subject. In some embodiments, the MHC and/or MHC sequence is a wild-type protein or sequence. In some embodiments, the MHC and/or MHC sequence is a naturally-occurring variant, e.g., an allele of MHC. In some embodiments, the MHC and/or MHC sequence is modified relative to a wild-type protein, e.g. chemically modified. In some embodiments, the MHC and/or MHC sequence is a derivative and/or variant of a wild-type MHC sequence. In some embodiments, the MHC and/or MHC sequence can be or comprise a human, or non-human sequence.

[0080] In one embodiment, the TCR recognition domain comprises a MHC (Major Histocompatibility Complex), a MHC-peptide complex, or a MHC-peptide fusion. In some embodiments, the TCR recognition domain can comprise a featureless peptide MHC, or a MHC without peptides, or any other molecule that can target or bind specifically to the variable region of the TCR.

[0081] The MHC, which is also referred to as the human leukocyte antigen (HLA), is comprised of a set of genes that code for cell surface proteins essential for the acquired. e.g., adaptive immune system to recognize foreign molecules in vertebrates, which in turn determines histocompatibility. The MHC gene family is divided into three subgroups: MHC class I, MHC class II, and MHC class III. Class I MHC molecules have the $\beta 2$ microglobulin subunit which can only be recognised by CD8 co-receptors. Class II MHC molecules have $\beta 1$ and $\beta 2$ subunits and can be recognized by CD4 co-receptors. In this way MHC molecules chaperone, which type of lymphocytes bind to the given antigen with high affinity, since different lymphocytes express different T-Cell Receptor (TCR) co-receptors. Components of the MHC are known in the art and can be readily identified by a skilled person. The MHC is further described in, e.g., Janeway CA Jr, Travers P, Walport M, et al, Immunobiology: The Immune System in Health and Disease, 5th edn (New York: Garland Science, 2001); Vigneron N, Stroobant V, Chapiro J, Ooms A, Degiovanni G, Morel S, et al. (April 2004). "An antigenic peptide produced by peptide splicing in the proteasome". Science. 304 (5670): 587–90; and K. Murphy, "Antigen recognition by T cells," in Janeway's Immunobiology, 8th, Ed., Garland Science, 2012, pp. 138-153; which are incorporated herein by reference in their entireties. A complete MHC class I complex comprises one MHC class I heavy chain, one peptide ligand sequence, and a beta 2 microglobulin. In some embodiments, a TCR recognition domain comprises one MHC class I heavy chain, one peptide ligand sequence, and a beta 2 microglobulin. A complete MHC class II complex comprises an MHC class II alpha chain, MHC class II beta chain, and one peptide ligand sequence. In some embodiments, a TCR recognition domain comprises an MHC class II alpha chain, MHC class II beta chain, and one peptide ligand sequence. These three components can either be assembled together as separate sequences (e.g., by intramolecular binding of multiple peptide molecules) or can be expressed as fusion proteins with intervening linker sequences (e.g., see Schmittnaegel et al., 2016; which is incorporated by reference herein in its entirety).

[0082] The MHC (Major Histocompatibility Complex), MHC-peptide complex, MHC-peptide fusion, featureless peptide MHC, can be selected on the basis of the disease or condition to be treated/prevented. Specific MHCs, peptides, and/or antigens that are associated with the diseases described herein are known in the art and an appropriate MHC, peptide, and/or antigen can be selected by one of ordinary skill in the art. For example, databases of suitable MHC, peptide, and/or antigen sequences are available on the world wide web at iedb.org; immunespace.org; immgen.org; import.org; peptideatlast.org/repository/; uniprot.org; ncbi.nlm.nih.gov/protein/;

immunedata.org/index.php; immuneprofiting.org/hipc/; allergenonline.org/databasebrowse.shtml; and itntrialshare.org. Further examples are also provided in Smatti et al. 2019 Viruses 11:762; Beretta-Piccoli et al. 2019 J Autoimmu 94:1-6; and Cusick et al. 2012 Clinical Reviews in Allergy and Immunology; each of which is incorporated by reference herein in its entirety. The specific examples of TCR recognition domains provides herein are exemplary and non-limiting. One of skill in the art can identify relevant auto-antigenic pMHCs, allogeneic peptide MHCs, and autogenic peptide MHCs in addition to those described herein, e.g., from the art and/or from donor cells. Such identification is within the skill of the ordinary practitioner.

[0083] As an illustrative and non-limiting example, for compositions and methods relating to type 1 diabetes, the TCR recognition domain can comprise one or more of SEQ ID NOs: 8-17. In some embodiments, the TCR recognition domain can comprise SEQ ID NO: 8, 9, and one of 10-17. In some embodiments, the TCR recognition domain can comprise sequences with at least 80%, at least 85%, at least 90%, at least 95%, at least 98% or greater sequence identity to SEQ ID NO: 8, 9, and one of 10-17. In some embodiments, the TCR recognition domain can comprise sequences with at least 95% sequence identity to SEQ ID NO: 8, 9, and one of 10-17, and which retain the wild-type activity of SEQ ID NOs: 8, 9, and one of 10-17.

[0084] SEQ ID NO: 8 HLA-A*0201 (MHC class I heavy chain allele, wildtype, human)
 MAVMAPRTLVLALLSGALALTQTWAGSHSMRYFFTSVSRPGRGEPRIAVGYVDDTQFVRFDSDAASQR
 MEPRAPWIEQEGPEYWDGETRQVKAHSQTHRVDLGLRQYQSEAGSHTVQRMYGCDVGSQDWRFLRG
 YHQYAYDGKDYIALKEDLRSWTAADMAAQTTKHKWEAAHVAEQLRAYLEGTCVEWLRRLRYLENGKETLQ
 RTDAPKTHMTHHAVSDHEATLRCWALSFPYPAEITLTWQRDGEDQTQDTELVTETRPAGDGTQKWAQAVV
 VPSGQEQRYTCHVQHEGLPKPLTLRWEPSQPTIPIVGIAGLVLFQAVITGAVVAAMWRRKSSDRK
 GGSYSQAASSDSAQGSQSDVSLTACKV

[0085] SEQ ID NO: 9 Beta-2 microglobulin (wildtype, human)
 MSRSVALAVLALLSLSGLEGIQRTPKIQVYSRHPAENGKSNFLNCYVSGFHQSDIEVDLLKNGERIEK
 VEHSDFSFSKDWSEFYLLYYTEFTPTTEKDEYACRVNHVTLSQPKIVKWDRDM

[0086] SEQ ID NO: 10 Preproinsulin₁₅₋₂₄ (wildtype, human)
 ALWGPDPAAA

[0087] SEQ ID NO: 11 Preproinsulin₁₅₋₂₄ altered peptide ligand #1 (synthetic, Cole et al., 2016)
 AQWGPDPAAA

[0088] SEQ ID NO: 12 Preproinsulin₁₅₋₂₄ altered peptide ligand #2 (synthetic, Cole et al., 2016)
 RQWGPDPAAV

[0089] SEQ ID NO: 13 Preproinsulin₁₅₋₂₄ altered peptide ligand #3 (wildtype, *Clostridium asparagiforme*, Cole et al., 2016)

RQFGPDWIVA

[0090] SEQ ID NO: 14 Preproinsulin₁₅₋₂₄ altered peptide ligand #4 (synthetic, Cole et al., 2016)

YQFGPDFPIA

[0091] SEQ ID NO: 15 Preproinsulin₁₅₋₂₄ altered peptide ligand #5 (synthetic, Cole et al., 2016)

RQFGPDFPTI

[0092] SEQ ID NO: 16 Preproinsulin₁₅₋₂₄ altered peptide ligand #6 (synthetic, Cole et al., 2016)

YLGGPDFPTI

[0093] SEQ ID NO: 17 Preproinsulin₁₅₋₂₄ altered peptide ligand #7 (wildtype, *Bacteroides fragilis*, Cole et al., 2016)

MVWGPDLYV

[0094] As a further illustrative example, for compositions and methods relating vitiligo, the TCR recognition domain can comprise one or more of SEQ ID NOs: 18-22. In some embodiments, the TCR recognition domain can comprise SEQ ID NO: 18, 19, and one of 20-22. In some embodiments, the TCR recognition domain can comprise sequences with at least 80%, at least 85%, at least 90%, at least 95%, at least 98% or greater sequence identity to SEQ ID NO: 18, 19, and one of 20-22. In some embodiments, the TCR recognition domain can comprise sequences with at least 95% sequence identity to SEQ ID NO: 18, 19, and one of 20-22, and which retain the wild-type activity of SEQ ID NOs: 18, 19, and one of 20-22.

[0095] SEQ ID NO: 18 HLA-A*0201 (MHC class I heavy chain allele, wildtype, human, same as previous HLA-A2)

MAVMAPRTLVLALLSGALALTQTWAGSHSMRYFFTSVSRPGRGEPFIAVGYVDDTQFVRFSDAASQR
MEPRAPWIEQEGPEYWDGETRKVKASHSQTHRVDLGLTGRGYNQSEAGSHTVQRMYGCDVGSDWRFLRG
YHQYAYDGKDYIALKEDLRSWTAADMAAQTTKHKWEAAHVAEQLRAYLEGTCVEWLRRLRYLENGKETLQ
RTDAPKTHMTHHAVSDHEATLRCWALSFPYPAEITLTWQRDGEDQTQDTELVEVTRPAGDGTQKWAADV
VPSGQEQRYTCHVQHEGLPKPLTLRWEPSQPTIPIVGIIAGLVLFGLAVITGAVVAAMWRRKSSDRK
GGYSQAASSDSAQGSVDVSLTACKV

[0096] SEQ ID NO: 19 Beta-2 microglobulin (wildtype, human, same as previous Beta-2 microglobulin)

MSRSVALAVLALLSLSGLEGIQRTPKIQVYSRHPAENGKSNFLNCYVSGFHQSDIEVDLLKNGERIEK
VEHSDLSFSKDWSFYLLYYTEFTPTTEKDEYACRVNHVTLSQPKIVKWDRDM

[0097] SEQ ID NO: 20 MART-1₂₆₋₃₅ (wildtype, human)

ELAGIGILTV

[0098] SEQ ID NO: 21 Tyrosinase₃₆₈₋₃₇₆ (wildtype, human)

YMDGTMSQV

[0099] SEQ ID NO: 22 gp100₂₀₉₋₂₁₇ (wildtype, human)

ITDQVPFSV

[00100] As further illustrative and non-limiting examples, the following pairs of MHC and antigens provided in Table 5 are known in the art. In some embodiments, the TCR recognition domain can comprise one or more of the following indicated MHC / peptide pairs, e.g., the TCR recognition domain can comprise one of the indicated MHC alleles and the indicated corresponding peptide. In some embodiments, the TCR recognition domain can comprise sequences with at least 80%, at least 85%, at least 90%, at least 95%, at least 98% or greater sequence identity to one of the following indicated MHC / peptide pairs, e.g., one of the indicated MHC alleles and the indicated corresponding peptide. In some embodiments, the TCR recognition domain can comprise sequences with at least 95% sequence identity to one of the following indicated MHC / peptide pairs, e.g., one of the indicated MHC alleles and the indicated corresponding peptide, wherein those sequences retain the wild-type activity of the MHC allele and the corresponding peptide.

[00101] Table 5: Each line of the following Table provides a MHC allele (whose sequence is available in publically-accessible databases, e.g., NCBI), and an antigen sequence. The antigen source and/or the relevant disease are also indicated on some lines. In some examples provided in Table 5, the peptides are antigen mimics and their origin is indicated.

HLA-A*0201	VMNILLQYVV (SEQ ID NO: 2013)	GAD65	Diabetes
HLA-A*0201	VMNILLQYVV (SEQ ID NO: 2013)	GAD65	Diabetes
HLA-A*0201	YAYDGKDYIA (SEQ ID NO: 2014)	HLA-A2	Diabetes
HLA-A*0201	MVWESGCTV (SEQ ID NO: 2015)	IA-2	Diabetes
HLA-A*0201	VIVMLTPLV (SEQ ID NO: 2016)	IA-2	Diabetes
HLA-A*0201	YTCPLCRAPV (SEQ ID NO: 2017)	SAA	Autoimmune
HLA-A*0201	YMDGTMSQV (SEQ ID NO: 2018)	Tyrosinase	Vitiligo
HLA-A*0301	YMVPFIPLYR (SEQ ID NO: 2019)	Tyrosinase	Vitiligo
HLA-A*2402	AFLPWHRLF (SEQ ID NO: 2020)	Tyrosinase	Vitiligo
HLA-A*0101	SSDYVIPIGTY (SEQ ID NO: 2021)	Tyrosinase	Vitiligo
HLA-A*02:01	VLHDDLLEA (SEQ ID NO: 2022)	HA-1 137-145	Minor Histocompatibility Antigen
HLA-A*02:01	RTLDKVLEV (SEQ ID NO: 2023)	miHAg HA-8	Minor Histocompatibility Antigen
HLA-A*02:01	FIDSYICQV (SEQ ID NO: 2024)	miHAg H-Y (human SMCY) 311-319	Minor Histocompatibility Antigen
DRB1*04:01	GAGSLQPLALEGSLQKRG A (SEQ ID NO: 2025)	Proinsulin 73-90	
DRB1*04:01	IAFTSEHSHFSLK A (SEQ ID NO: 2026)	GAD65 274-286	
DRB1*15:01	DENPVVHFFKNIVTPRTPP (SEQ ID NO: 2027)	Myelin basic protein	83-101
DRB1*04:01	GIVEQCCTSICSLYQ A (SEQ ID NO: 2028)	Proinsulin 90-104	
DRB1*04:01	NFIRMVISNPAAT A (SEQ ID NO: 2029)	GAD65 555-567	
DRB1*04:01	DVMNILLQYVVKSFDRSTKV (SEQ ID NO: 2030)	GAD65113-132	
HLA-A*0201	ALWGPDPAAA (SEQ ID NO: 2031)	Insulin	Diabetes

HLA-A*0201	HLVEALYLV (SEQ ID NO: 2032)	Insulin	Diabetes
HLA-A*0201	KLQVFLIVL (SEQ ID NO: 2033)	IAPP	Diabetes
HLA-A*0101	IVDCLTEMY (SEQ ID NO: 2034)	USP9Y	Graft vs Host
HLA-A*0201	VLHDDLLEA (SEQ ID NO: 2035)	HA-1	Graft vs Host
HLA-A*0201	RTLDKVLEV (SEQ ID NO: 2036)	HA-8	Graft vs Host
HLA-A*0201	FIDSYICQV (SEQ ID NO: 2037)	H-Y	Graft vs Host
HLA-B*0702	SPSVDKARAE (SEQ ID NO: 2038)	SMCY	Graft vs Host
HLA-B*0801	LPHNHTDL (SEQ ID NO: 2039)	TPR-protein	Graft vs Host
Antigen Mimicry			
HLA-A*0201	FLDKGTYTL (SEQ ID NO: 2040)	BALF4	EBV
HLA-A*0201	GLCTLVAML (SEQ ID NO: 2041)	BMLF1	EBV
HLA-A*0201	TLDYKPLSV (SEQ ID NO: 2042)	BMRF1	EBV
HLA-A*0201	YVLDHLIVV (SEQ ID NO: 2043)	BRLF1	EBV
HLA-A*0201	LLDFVRFMGV (SEQ ID NO: 2044)	EBNA 3B	EBV
HLA-A*0201	YLLEMLWRL (SEQ ID NO: 2045)	LMP-1	EBV
HLA-A*0201	YLQQNWWTL (SEQ ID NO: 2046)	LMP-1	EBV
HLA-A*0201	CLGGLTMMV (SEQ ID NO: 2047)	LMP-2A	EBV
HLA-A*0201	FLYALALLL (SEQ ID NO: 2048)	LMP-2A	EBV
HLA-A*0301	RLRAEAQVK (SEQ ID NO: 2049)	EMNA 3A	EBV
HLA-A*1101	AVFDRKSDAK (SEQ ID NO: 2050)	EBNA 3B	EBV
HLA-A*1101	IVTDFSVIK (SEQ ID NO: 2051)	EBNA 3B	EBV
HLA-A*2402	DYCNVLNKEF (SEQ ID NO: 2052)	BRLF1	EBV
HLA-A*2402	TYGPVFMCL (SEQ ID NO: 2053)	LMP-2	EBV
HLA-A*2902	IACPIVMRY (SEQ ID NO: 2054)	BRLF1	EBV
HLA-A*6801	IVTDFSVIK (SEQ ID NO: 2051)	EBNA 3B	EBV
HLA-B*0702	RPQGGSRPEFVKL (SEQ ID NO: 2055)	BMRF1	EBV
HLA-B*0702	RPPIFIRRL (SEQ ID NO: 2056)	EBNA 3A	EBV
HLA-B*0702	QPRAPIRPI (SEQ ID NO: 2057)	EBNA 6	EBV
HLA-B*0801	RAKFKQLL (SEQ ID NO: 2058)	BZLF1	EBV
HLA-B*0801	FLRGRAYGL (SEQ ID NO: 2059)	EBNA 3A	EBV
HLA-B*3501	EPLPQGQLTAY (SEQ ID NO: 2060)	BZLF1	EBV

HLA-B*3501	EPLSQSQITAY (SEQ ID NO: 2061)	BZLF1	EBV
HLA-B*3501	HPVAEADYFEY (SEQ ID NO: 2062)	EBNA 1	EBV
HLA-B*3501	HPVGDADYFEY (SEQ ID NO: 2063)	EBNA 1	EBV
HLA-B*3501	HPVGEADYFEY (SEQ ID NO: 2064)	EBNA 1	EBV
HLA-B*3501	HPVGQADYFEY (SEQ ID NO: 2065)	EBNA 1	EBV
HLA-B*3501	YPLHEQHGM (SEQ ID NO: 2066)	EBNA 3A	EBV
HLA-A*0101	VTEHDTLLY (SEQ ID NO: 2067)	UL44	CMV
HLA-A*0201	VLEETSVML (SEQ ID NO: 2068)	IE-1	CMV
HLA-A*0201	NLVPMTATV (SEQ ID NO: 2069)	pp65	CMV
HLA-A*0301	KLGGALQAK (SEQ ID NO: 2070)	IE-1	CMV
HLA-A*2301	QYDPVAALF (SEQ ID NO: 2071)	pp65	CMV
HLA-A*2402	AYAQQIFKI (SEQ ID NO: 2072)	IE-1	CMV
HLA-A*2402	QYDPVAALF (SEQ ID NO: 2071)	pp65	CMV
HLA-A*2402	VYALPLKML (SEQ ID NO: 2073)	pp65	CMV
HLA-B*0702	RPHERNGFTVL (SEQ ID NO: 2074)	pp65	CMV
HLA-B*0702	TPRVTGGGAM (SEQ ID NO: 2075)	pp65	CMV
HLA-B*0801	ELKRKMIYM (SEQ ID NO: 2076)	IE-1	CMV
HLA-B*0801	ELNRKMIYM (SEQ ID NO: 2077)	IE-1	CMV
HLA-B*0801	ELRRKMMYM (SEQ ID NO: 2078)	IE-1	CMV
HLA-B*0801	QIKVRVDMV (SEQ ID NO: 2079)	IE-1	CMV
HLA-B*3501	IPSINVHHY (SEQ ID NO: 2080)	pp65	CMV
HLA-B*3501	LPLNVGLPIIGVM (SEQ ID NO: 2081)	UL138	CMV
A*01:01	SADNNNSEY (SEQ ID NO: 2082)	AAV VP1 492-500	
A*01:01	TDLGQNLLY (SEQ ID NO: 2083)	Adenovirus 5 Hexon 886-894	
A*01:01	EADPTGHSY (SEQ ID NO: 2084)	MAGE-A1 161-169	
A*01:01	EVDPIGHLY (SEQ ID NO: 2085)	MAGE-A3 168-176	
A*01:01	KSDICTDEY (SEQ ID NO: 2086)	Tyrosinase 243-251 (244S)	
A*01:01	KCDICTDEY (SEQ ID NO: 2087)	Tyrosinase 243-251	
A*01:01	QSLEIISRY (SEQ ID NO: 2088)	Mcl-1 177-185	
A*01:01	YVDFREYEEY (SEQ ID NO: 2089)	FLT3 ITD	
A*01:01	TLDTLTAFY (SEQ ID NO: 2090)	Mesothelin 429-437	
A*01:01	LTDDRLLFTCY (SEQ ID NO: 2091)	PLEKHM2	
A*01:01	DSDPDSFQDY (SEQ ID NO: 2092)	Tyr A1a 454-463	
A*01:01	EADPIGHLY (SEQ ID NO: 2093)	MAGEA3	
A*01:01	EVDPASNTY (SEQ ID NO: 2094)	MAGE-A4 169-177	
A*01:01	HSTNGVTRIY (SEQ ID NO: 2095)	PSMA	
A*01:01	ILDTAGREEY (SEQ ID NO: 2096)	N-ras 55-64	
A*01:01	LVDVMPWLQY (SEQ ID NO: 2097)	Cytochrome P450 240-249	
A*01:01	RSDSGQQARY (SEQ ID NO: 2098)	AIM-2	
A*01:01	VTEPGTAQY (SEQ ID NO: 2099)	Minor antigen HA-3T (Lbc oncogene 451-459)	
A*01:01	VYDFFVWLHY (SEQ ID NO: 2100)	TRP-2 181-190	
A*01:01	YSEHPTFTSQY (SEQ ID NO: 2101)	HCMV pp65 363-373	
A*01:01	VTEHDTLLY (SEQ ID NO: 2102)	HCMV pp50 245-253	
A*01:01	FTSDYYQLY (SEQ ID NO: 2103)	SARS-CoV-2 ORF3a 207-215 (confirmed epitope)	
A*01:01	TTDPSFLGRY (SEQ ID NO: 2104)	SARS-CoV-2 Replicase polyprotein 1ab 1637-1646	
A*01:01	PTDNYITTY (SEQ ID NO: 2105)	SARS-CoV-2 Replicase polyprotein 1ab 1621-1629	
A*01:01	LLDTASALY (SEQ ID NO: 2106)	HBV core 30-38	

A*01:01 ATDALMTGY (SEQ ID NO: 2107) HCV NS3 1435-1443
 A*01:01 ATDALMTGF (SEQ ID NO: 2108) HCV NS3 1436-1444
 A*01:01 CTELKLSY (SEQ ID NO: 2109) Influenza A (PR8) NP 44-52
 A*01:01 VSDGGPNLY (SEQ ID NO: 2110) Influenza A PB1 591-599
 A*01:01 IVDCLTEMY (SEQ ID NO: 2111) DRRFY (1521-1529))
 A*02:01 ALCNTDSPL (SEQ ID NO: 2112) iLR1
 A*02:01 ALKDVEERV (SEQ ID NO: 2113) MAGE-C2 336-344
 A*02:01 LLAARAIVAI (SEQ ID NO: 2114) iLR1 59-68
 A*02:01 RLWQELSDI (SEQ ID NO: 2115) circadian clock protein PASD1 691-700
 A*02:01 LLFGLALIEV (SEQ ID NO: 2116) MAGE-C2 191-200
 A*02:01 FLDPRPLTV (SEQ ID NO: 2117) CYP190
 A*02:01 STLCQVEPV (SEQ ID NO: 2118) MPP11
 A*02:01 VLQMKEEDV (SEQ ID NO: 2119) iLR1
 A*02:01 AIQDLCLAV (SEQ ID NO: 2120) NPM1
 A*02:01 QLLIKAVNL (SEQ ID NO: 2121) MPP11
 A*02:01 AIQDLCVAV (SEQ ID NO: 2122) NPM1
 A*02:01 ALTPVVVTL (SEQ ID NO: 2123) cyclin-dependent kinase 4 170-178
 A*02:01 KLQVFLIVL (SEQ ID NO: 2124) T1D Diabetes human prepro islet amyloid polypeptide pplAPP 513
 A*02:01 VMNILLQYV (SEQ ID NO: 2125) GAD65 114-123
 A*02:01 SLSRFSWGA (SEQ ID NO: 2126) Myelin basic protein 110-118
 A*02:01 HLVEALYLV (SEQ ID NO: 2127) Insulin B chain 10-18
 A*02:01 LNIDLLWSV (SEQ ID NO: 2128) T1D Diabetes IGRP 228-236
 A*02:01 VLFGLGFAI (SEQ ID NO: 2129) T1D Diabetes IGRP 265-273
 A*02:01 ALWGPDPAIA (SEQ ID NO: 2130) Proinsulin precursor 15-24
 A*02:01 MVWESGCTV (SEQ ID NO: 2131) IA-2 797-805
 A*02:01 YTCPLCRAPV (SEQ ID NO: 2132) SSA 55-56 55-64
 A*02:01 VIVMLTPLV (SEQ ID NO: 2133) IA-2 805-813
 A*02:01 AITEVECFL (SEQ ID NO: 2134) VP1 44-52
 A*02:01 FLHCIVFNV (SEQ ID NO: 2135) large T antigen 410-418
 A*02:01 LLMWEAVTV (SEQ ID NO: 2136) VP1 108-116
 A*02:01 CLLPKMDSV (SEQ ID NO: 2137) large T antigen 398-406
 A*02:01 FLWGPRLV (SEQ ID NO: 2138) MAGEA3 271-279
 A*02:01 IMDQVPFSV (SEQ ID NO: 2139) gp100 (pmel17) 209-217
 A*02:01 YLEPGPVTV (SEQ ID NO: 2140) gp100 (pmel) 280-288 (288V)
 A*02:01 YLSGADLNL (SEQ ID NO: 2141) Carcinoembryonic antigen (CEA)-derived peptide CAP1-6D
 A*02:01 SLLMWITQC (SEQ ID NO: 2142) NY-ESO-1 157-165 (9C)
 A*02:01 KTWGQYWQV (SEQ ID NO: 2143) gp100 (pmel17) 154-162
 A*02:01 YLEPGPVTA (SEQ ID NO: 2144) gp100
 A*02:01 YMDGTMSQV (SEQ ID NO: 2145) Tyrosinase 369-377 (371D)
 A*02:01 YLSGANLNL (SEQ ID NO: 2146) Carcinogenic Embryonic Antigen (CEA) 571-579
 A*02:01 ELAGIGILTV (SEQ ID NO: 2147) MelanA / MART 26-35
 A*02:01 ILAKFLHWL (SEQ ID NO: 2148) Telomerase 540-548
 A*02:01 ALQPGTALL (SEQ ID NO: 2149) Prostate Stem Cell Antigen (PSCA) 14-22
 A*02:01 VISNDVCAQV (SEQ ID NO: 2150) Prostate Specific Antigen-1 (PSA-1) 154-163
 A*02:01 RLVDDEFLLV (SEQ ID NO: 2151) Telomerase Reverse Transcriptase 865-873
 A*02:01 GVLVGVALI (SEQ ID NO: 2152) Carcinogenic Embryonic Antigen (CEA) 694-702
 A*02:01 VLYRYGSFSV (SEQ ID NO: 2153) gp100 (pmel17) 476-485
 A*02:01 PLFQVPEPV (SEQ ID NO: 2154) Alpha-fetoprotein isoform 1 137-145
 A*02:01 FMNKFYIEI (SEQ ID NO: 2155) Human alfa fetoprotein 158-166

A*02:01 GLSPNLNRFL (SEQ ID NO: 2156) Alpha-fetoprotein isoform 2 167-176
 A*02:01 KVLEYVIKV (SEQ ID NO: 2157) MAGEA1 278-286
 A*02:01 LLGRNSFEV (SEQ ID NO: 2158) p53 264-272
 A*02:01 LLLTLVLTV (SEQ ID NO: 2159) MUC-1 12-20
 A*02:01 ILHNGAYSL (SEQ ID NO: 2160) HER-2/neu 435-443
 A*02:01 RLLQETELV (SEQ ID NO: 2161) HER-2/neu 689-697
 A*02:01 KIFGSLAFL (SEQ ID NO: 2162) HER-2/neu 369-377
 A*02:01 LLLLDVAPL (SEQ ID NO: 2163) HSP1A 459-467
 A*02:01 LLDVAPLSL (SEQ ID NO: 2164) HSP1A 461-469
 A*02:01 HLYQGCQVV (SEQ ID NO: 2165) Receptor tyrosine-protein kinase erbB-2 48-56
 A*02:01 HLSTAFARV (SEQ ID NO: 2166) G250 (renal cell carcinoma) 217-225
 A*02:01 VLQELNVTV (SEQ ID NO: 2167) Leukocyte Proteinase-3 (Wegener's autoantigen)
 169-177
 A*02:01 KVAELVHFL (SEQ ID NO: 2168) MAGEA3 112-120
 A*02:01 VLAGVGFFI (SEQ ID NO: 2169) EPHA2 550-558
 A*02:01 FLYTLLREV (SEQ ID NO: 2170) STEAP 86-94
 A*02:01 ILLWQPIPV (SEQ ID NO: 2171) Prostatic Acid Phosphatase-3 (PAP-3) 135-143
 A*02:01 RLQEERTCKV (SEQ ID NO: 2172) BIR
 A*02:01 QLCPICRAPV (SEQ ID NO: 2173) Livin/ML-IAP280 175-184
 A*02:01 VLGEAWRDQV (SEQ ID NO: 2174) TRAP 45-54
 A*02:01 LLLTVLTVV (SEQ ID NO: 2175) Tumor Mucin Antigen 13-21
 A*02:01 GLYDGM EHL (SEQ ID NO: 2176) MAGEA-10 254-262
 A*02:01 SLLMWITQV (SEQ ID NO: 2177) NY-ESO-1 157-165
 A*02:01 LMLGEFLKL (SEQ ID NO: 2178) Survivin 96-104
 A*02:01 YLFFYRKS (SEQ ID NO: 2179) mTERT 572-580
 A*02:01 ELTLGEFLKL (SEQ ID NO: 2180) survivin 95-104
 A*02:01 FLTPKKLQCV (SEQ ID NO: 2181) Prostate Specific Antigen-1 (PSA-1) 141-150
 A*02:01 KLQCVDLHV (SEQ ID NO: 2182) Prostate Specific Antigen 146-154
 A*02:01 TLAPATEPA (SEQ ID NO: 2183) Mucin 79-87
 A*02:01 YLQVNSLQTV (SEQ ID NO: 2184) Telomerase Reverse Transcriptase (hTRT) 988-997
 A*02:01 SLGEQQYSV (SEQ ID NO: 2185) WT1 187-195
 A*02:01 SLEENIVIL (SEQ ID NO: 2186) RHAMM 275-283
 A*02:01 YMNGTMSQV (SEQ ID NO: 2187) Tyrosinase 368-376
 A*02:01 ILSLELMKL (SEQ ID NO: 2188) Receptor for hyaluronic acid-mediated motility
 (RHAMM) 165-173
 A*02:01 PLFDFSWLSL (SEQ ID NO: 2189) Bcl-2 208-217
 A*02:01 LLGATCMFV (SEQ ID NO: 2190) CyclinD 101-109
 A*02:01 ALYVDSLFFL (SEQ ID NO: 2191) PRAME PRA 300-309
 A*02:01 GLMEEMSAL (SEQ ID NO: 2192) Human Mena protein (overexpressed in breast
 cancer)
 A*02:01 TMNGSKSPV (SEQ ID NO: 2193) hMena 502-510
 A*02:01 GVYDGREHTV (SEQ ID NO: 2194) MAGE-A4 230-239
 A*02:01 YLNDHLEPWI (SEQ ID NO: 2195) Bcl-X 173-182
 A*02:01 ALDVYNGLL (SEQ ID NO: 2196) Prostatic acid phosphatase precursor (PAP) 299-307
 A*02:01 ALFDIESKV (SEQ ID NO: 2197) PSM P2 (prostate)
 A*02:01 SLAMLDLLHV (SEQ ID NO: 2198) Mutant anaplastic lymphoma kinase 1220-1229
 A*02:01 YLNTVQPTCV (SEQ ID NO: 2199) EGF-R 1138-1147
 A*02:01 KLFGTSGQKT (SEQ ID NO: 2200) EGF-R-479 350-359
 A*02:01 RMPEAAPPV (SEQ ID NO: 2201) p53 65-73
 A*02:01 PLTSIISAV (SEQ ID NO: 2202) Receptor tyrosine-protein kinase erbB-2 728-736
 A*02:01 VLAGGFFLL (SEQ ID NO: 2203) PSMA 27-38

A*02:01 LLHETDSAV (SEQ ID NO: 2204) PSMA/PSM-P1 4-12
 A*02:01 VMAGVGSPYV (SEQ ID NO: 2205) Receptor tyrosine-protein kinase erbB-2
 819-828
 A*02:01 VLPLTVAEV (SEQ ID NO: 2206) Mesothelin 530-538
 A*02:01 SLLFLLFSL (SEQ ID NO: 2207) Mesothelin 20-28
 A*02:01 QLFEELQEL (SEQ ID NO: 2208) Heme oxygenase-1 212-220
 A*02:01 VLDGLDVLL (SEQ ID NO: 2209) PRAME 100-108
 A*02:01 RLASFYDWPL (SEQ ID NO: 2210) BIR7 90-99
 A*02:01 LIAHNQVRQV (SEQ ID NO: 2211) HER-2/neu (85-94)
 A*02:01 ILHDGAYSL (SEQ ID NO: 2212) HER-2 434-443
 A*02:01 FVGEEFTDV (SEQ ID NO: 2213) GPC3 144-152 (overexpressed in hepatocellular carcinoma)
 A*02:01 LLLIWFRPV (SEQ ID NO: 2214) BKV Ltag 579-587
 A*02:01 KLQDASAEV (SEQ ID NO: 2215) HM1.24-aa 126-134
 A*02:01 SLYSFPEPEA (SEQ ID NO: 2216) PRAME
 A*02:01 SLLQHLIGL (SEQ ID NO: 2217) PRAME 425-433
 A*02:01 VIFDFLHCI (SEQ ID NO: 2218) BKV Ltag 406-414
 A*02:01 VLDFAPPGA (SEQ ID NO: 2219) WT1
 A*02:01 TLPGYPPHV (SEQ ID NO: 2220) PAX-5 311-319
 A*02:01 YMEHNNVYTV (SEQ ID NO: 2221) Fibromodulin 250-259
 A*02:01 YLQHNEIQEV (SEQ ID NO: 2222) Fibromodulin 206-215
 A*02:01 SLVDVMPWL (SEQ ID NO: 2223) Cytochrome p450 1B1 239-248
 A*02:01 RLMNDMTAV (SEQ ID NO: 2224) HSP105 128-136
 A*02:01 RLARLALVL (SEQ ID NO: 2225) Trophoblast glycoprotein 17-25
 A*02:01 FLTGNQLAV (SEQ ID NO: 2226) 5T4 97-105
 A*02:01 LLLAGLFSL (SEQ ID NO: 2227) Fibromodulin 7-15
 A*02:01 FLGYLILGV (SEQ ID NO: 2228) Prostatic Acid Phosphatase-3 (PAP-3)
 A*02:01 SLFLGILSV (SEQ ID NO: 2229) CD20 188-196 (B cell malignancies)
 A*02:01 AVLPLLELV (SEQ ID NO: 2230) MCL-1 139-147
 A*02:01 SLSEKTVLL (SEQ ID NO: 2231) CD59 glycoprotein precursor 106-114
 A*02:01 YMCSFLFNL (SEQ ID NO: 2232) Ewing Tumor EZH2 666-674
 A*02:01 YLISGDSPV (SEQ ID NO: 2233) CD33 65-73 (1Y2L)
 A*02:01 KASEKIFYV (SEQ ID NO: 2234) SSX2 41-49
 A*02:01 FLAKLNNTV (SEQ ID NO: 2235) HCA587 317-325
 A*02:01 GLAPPQHLIRV (SEQ ID NO: 2236) p53 187-197
 A*02:01 VIMPCSWWV (SEQ ID NO: 2237) Chondromodulin-I 319-327
 A*02:01 KVVEFLAML (SEQ ID NO: 2238) MAGE-C1 1083-1091
 A*02:01 LTLGEFLKL (SEQ ID NO: 2239) Survivin-3A 96-104
 A*02:01 ALPFGFILV (SEQ ID NO: 2240) IL13R 345-353
 A*02:01 TLADFDPRV (SEQ ID NO: 2241) EphA2
 A*02:01 ALMEQQHYV (SEQ ID NO: 2242) ITGB8 662-670
 A*02:01 CLTSTVQLV (SEQ ID NO: 2243) HER-2/neu 789-797
 A*02:01 GLLGASVLGL (SEQ ID NO: 2244) Telomerase Reverse Transcriptase (hTERT) 674-683
 A*02:01 QLLDGFMITL (SEQ ID NO: 2245) PASD1 39-48
 A*02:01 YLVGNVCIL (SEQ ID NO: 2246) PASD1 168-176
 A*02:01 ALLTSRLRFI (SEQ ID NO: 2247) Telomerase Reverse Transcriptase (hTERT) 615-624
 A*02:01 RLSSCPVA (SEQ ID NO: 2248) TGF beta receptor type-2 131-139
 A*02:01 FLYDDNQRV (SEQ ID NO: 2249) Topoisomerase II-alpha-b 828-836
 A*02:01 YLIELIDRV (SEQ ID NO: 2250) TACE 250-258
 A*02:01 FLAEDALNTV (SEQ ID NO: 2251) Epithelial Discoidin Domain Receptor 1 (EDDR1)
 867-876

A*02:01 GLMKYIGEV (SEQ ID NO: 2252) TRPM8 187-195
 A*02:01 AILALLPAL (SEQ ID NO: 2253) Prostate Stem Cell Antigen (PSCA) 105-133
 A*02:01 GLQHWVPEL (SEQ ID NO: 2254) BA46 (Lactadherin) 97-106
 A*02:01 GVRGRVEEI (SEQ ID NO: 2255) BCR-ABL
 A*02:01 ITDQVPFSV (SEQ ID NO: 2256) gp100 (pmel) 209-217
 A*02:01 KLCPVQLWV (SEQ ID NO: 2257) p53 139-147
 A*02:01 KVAEELVHFL (SEQ ID NO: 2258) MAGEA3 112-120 (alternative version)
 A*02:01 SLPPPGTRV (SEQ ID NO: 2259) p53 149-157
 A*02:01 YLGSYGFRLL (SEQ ID NO: 2260) p53 103-111
 A*02:01 YLQLVFGIEV (SEQ ID NO: 2261) MAGEA2 157-166
 A*02:01 TLQDIVYKL (SEQ ID NO: 2262) BMI1 74-82
 A*02:01 YAILDLPVSV (SEQ ID NO: 2263) L-dopachrome tautomerase 488-496
 A*02:01 AMVGAVLTA (SEQ ID NO: 2264) Tyrosinase 482-190
 A*02:01 ATVGIMIGV (SEQ ID NO: 2265) CEACAM5 687-695
 A*02:01 YVDPVITSI (SEQ ID NO: 2266) Hepatocyte growth factor receptor 673-681
 A*02:01 GVLLWEIFSL (SEQ ID NO: 2267) VEGFR1 28-37
 A*02:01 LMAQEALAFLL (SEQ ID NO: 2268) CAMEL 2-11
 A*02:01 RVA(PHOSPHO-S)PTSGV (SEQ ID NO: 2269) Insulin receptor substrate-2 1097-1105
 A*02:01 RVASPTSGV (SEQ ID NO: 2270) IRS-2 1097-1105
 A*02:01 ALNVYNGLL (SEQ ID NO: 2271) ACPP 299-307
 A*02:01 ALSPVPPVV (SEQ ID NO: 2272) Bcl-2 85-93
 A*02:01 ALVCYGPGL (SEQ ID NO: 2273) FAP alpha 463-471
 A*02:01 ALWPWLLMAT (SEQ ID NO: 2274) RNF43 11-20
 A*02:01 ALYLMELTM (SEQ ID NO: 2275) CB9L2
 A*02:01 CLPSPSTPV (SEQ ID NO: 2276) BMI1 271-279
 A*02:01 ELSDSLGPV (SEQ ID NO: 2277) PASD1 695-703
 A*02:01 FLFLRNFSLL (SEQ ID NO: 2278) TARP(V28L)27-35
 A*02:01 FLPSPLFFLL (SEQ ID NO: 2279) TARP(P5L) 5-13
 A*02:01 GLFKCGIAV (SEQ ID NO: 2280) FAP 639-647
 A*02:01 GLIQLVEGV (SEQ ID NO: 2281) TRAG-3 4-12
 A*02:01 ILGVLTSLV (SEQ ID NO: 2282) DLK1 309-317
 A*02:01 LLVPTCVFLV (SEQ ID NO: 2283) 691-700
 A*02:01 MLAVFLPIV (SEQ ID NO: 2284) STEAP 292-300 (293L)
 A*02:01 NLFETPVEA (SEQ ID NO: 2285) 194-202
 A*02:01 QLGEQCWTV (SEQ ID NO: 2286) PSCA 44-51 (51A)
 A*02:01 RLAEQYAYI (SEQ ID NO: 2287) SART3 309-317
 A*02:01 SIDWFMVTV (SEQ ID NO: 2288) p31-39
 A*02:01 SILLRDAGLV (SEQ ID NO: 2289) TRAG-3 57-66
 A*02:01 SLFEPPLPPG (SEQ ID NO: 2290) PSMA 85-93
 A*02:01 SQADALKYV (SEQ ID NO: 2291) EZH2 729-737
 A*02:01 WLSLKTLLSL (SEQ ID NO: 2292) Bcl-2 214-223
 A*02:01 YLNRHLHTWI (SEQ ID NO: 2293) BCL-2 180-189
 A*02:01 YLQWIEFSI (SEQ ID NO: 2294) Prominin1 744-752
 A*02:01 YLYQWLGA PV (SEQ ID NO: 2295) Osteocalcin 51-60
 A*02:01 KLMSNSTDL (SEQ ID NO: 2296) HSP105 234-243
 A*02:01 RLQGISPKI (SEQ ID NO: 2297) SSX2 103-111
 A*02:01 AILALLPALL (SEQ ID NO: 2298) PSCA
 A*02:01 ALIHHNTHL (SEQ ID NO: 2299) HER2 466-474
 A*02:01 CMHLLLEAV (SEQ ID NO: 2300) MG50 624-632
 A*02:01 FLIIWQNTM (SEQ ID NO: 2301) FSP26
 A*02:01 FLPWHRLFLL (SEQ ID NO: 2302) Tyrosinase 207-216

A*02:01 FVWLHYYSV (SEQ ID NO: 2303) TRP2 185-193(L)
 A*02:01 GLFGDIYLA (SEQ ID NO: 2304) CSNK1A1 26-34
 A*02:01 GLFGDIYLA (SEQ ID NO: 2305) CSNK1A1 26-35
 A*02:01 ILLRDAGLV (SEQ ID NO: 2306) TRAG-3L 58-66
 A*02:01 ILLVVVLGV (SEQ ID NO: 2307) Receptor tyrosine-protein kinase erbB-2 707-715
 A*02:01 ILNAMIAMI (SEQ ID NO: 2308) HAUS3 154-162
 A*02:01 KASEYLQLV (SEQ ID NO: 2309) MAGEA2 153-161
 A*02:01 KIWEELSVL (SEQ ID NO: 2310) MAGEA3 220-228
 A*02:01 KLIDRTE(S)L (SEQ ID NO: 2311) LSP1 325-333
 A*02:01 KLTGDENFTI (SEQ ID NO: 2312) Tyrosinase precursor 224-233
 A*02:01 LLCYSCKAQV (SEQ ID NO: 2313) PSCA 17-26
 A*02:01 LLEAVPAV (SEQ ID NO: 2314) MG50 69-77
 A*02:01 LLNQLQVNL (SEQ ID NO: 2315) Mucin2 467-475
 A*02:01 LLRDAGLVKM (SEQ ID NO: 2316) TRAP 59-68
 A*02:01 LLRRYNVAKV (SEQ ID NO: 2317) SOX11 266-275
 A*02:01 LLSHGAVIEV (SEQ ID NO: 2318) Ankyrin NYBR1 158-167
 A*02:01 LVFGIELMEV (SEQ ID NO: 2319) MAGEA3 160-169
 A*02:01 LVFGIEVVEV (SEQ ID NO: 2320) MAGEA12 160-169
 A*02:01 MLWGWREHV (SEQ ID NO: 2321) Mucin2 645-653
 A*02:01 PLQPEQLQV (SEQ ID NO: 2322) Receptor tyrosine-protein kinase erbB-2 437-445
 A*02:01 QLMAFNHLI (SEQ ID NO: 2323) PAX3/FKHR 135-143
 A*02:01 QLMPYGCLL (SEQ ID NO: 2324) Receptor tyrosine-protein kinase erbB-2 845-853
 A*02:01 RLGPITLMCL (SEQ ID NO: 2325) MG50 1244-1252
 A*02:01 RLTRFLSRV (SEQ ID NO: 2326) CyclinD 228-236
 A*02:01 RTF(S)PTYGL (SEQ ID NO: 2327) Desmuslin 426-434
 A*02:01 SILLRDAGL (SEQ ID NO: 2328) TRAP 57-65
 A*02:01 SLADEAEVYL (SEQ ID NO: 2329) GAS7 Neoepitope
 A*02:01 SLDDYNHLV (SEQ ID NO: 2330) L-dopachrome tautomerase 288-296
 A*02:01 SLYKFSPFPL (SEQ ID NO: 2331) O-linked N-acetylglucosamine transferase FSP06
 A*02:01 SMTR(S)PPRV (SEQ ID NO: 2332) SFRS2B 241-249
 A*02:01 TLEEITGYL (SEQ ID NO: 2333) Receptor tyrosine-protein kinase erbB-2 448-456
 A*02:01 TLHCDCEIL (SEQ ID NO: 2334) MG50 210-218
 A*02:01 VLEPPGARDV (SEQ ID NO: 2335) BIR 7 230-239
 A*02:01 VLLALLMAGL (SEQ ID NO: 2336) Prostate stem cell antigen 4-13
 A*02:01 VLSVNVDPV (SEQ ID NO: 2337) MG50 625-633
 A*02:01 VLVKSPNHV (SEQ ID NO: 2338) Receptor tyrosine-protein kinase erbB-4 890-898
 A*02:01 VMIG(S)PKKV (SEQ ID NO: 2339) Tensin3 1558-1566
 A*02:01 VVLGVVFGI (SEQ ID NO: 2340) Receptor tyrosine-protein kinase erbB-2 743-751
 A*02:01 WLPKILGEV (SEQ ID NO: 2341) MG50 1051-1059
 A*02:01 WLQYFPNPV (SEQ ID NO: 2342) Cytochrome P450 246-254
 A*02:01 YLLDLSTNHL (SEQ ID NO: 2343) Fibromodulin 7-15
 A*02:01 YLWWVNNQSL (SEQ ID NO: 2344) CEA 176-185
 A*02:01 ALGGHPLLGV (SEQ ID NO: 2345) Dickkopf-related protein 1 20-29
 A*02:01 ALLAGLVSL (SEQ ID NO: 2346) FGFR4 676-684
 A*02:01 ALLTYMIAHI (SEQ ID NO: 2347) Thymidylate synthase 231-240
 A*02:01 ALMDKSLHV (SEQ ID NO: 2348) MART-1 56-64
 A*02:01 ALPPPLMLL (SEQ ID NO: 2349) Heparanase 8-16
 A*02:01 ALSVMGVYV (SEQ ID NO: 2350) MAGEA9 223-231
 A*02:01 ALVEFEDVL (SEQ ID NO: 2351) hnRNP L 140-148
 A*02:01 ALWPWLLMA (SEQ ID NO: 2352) RNF43 11-19

A*02:01 184-193	AMLGHTTMEV (SEQ ID NO: 2353)	Melanocyte-specific secreted glycoprotein
A*02:01	AVIGALLAV (SEQ ID NO: 2354)	Melanocyte-specific secreted glycoprotein 20-28
A*02:01	CLYGNVEKV (SEQ ID NO: 2355)	hnRNP L 404-412
A*02:01	DLIFGLNAL (SEQ ID NO: 2356)	Heparanase 185-193
A*02:01	ELFQDLSQL (SEQ ID NO: 2357)	ETV5 54-53
A*02:01	FAWERVERGL (SEQ ID NO: 2358)	Cyclin-dependent kinase inhibitor 1 97-105
A*02:01	FIASNGVKLV (SEQ ID NO: 2359)	ACTN4 118-127 (K5N)
A*02:01	FLALIICNA (SEQ ID NO: 2360)	Tubulin beta 4 283-291
A*02:01	FLDEFMEGV (SEQ ID NO: 2361)	Malic enzyme 224-232
A*02:01	RMFPNAPYL (SEQ ID NO: 2362)	WT-1 126-134 (Wilms tumor)
A*02:01	RLNMFTPYI (SEQ ID NO: 2363)	Chlamydia trachomatis MOMP 258-266
A*02:01	NMFTPYIGV (SEQ ID NO: 2364)	MOMP precursor 283-291
A*02:01	NLVPMVATV (SEQ ID NO: 2365)	HCMV pp65 495-504
A*02:01	VLEETSVML (SEQ ID NO: 2366)	HCMV IE1 316-324 (UL123)
A*02:01	VLAELVKQI (SEQ ID NO: 2367)	HCMV IE1 81-89
A*02:01	MLNIPSINV (SEQ ID NO: 2368)	pp65 120-128
A*02:01	LLLDRLNQL (SEQ ID NO: 2369)	SARS-CoV Nucleocapsid protein 223-231 (conserved in SARS-CoV-2)
A*02:01	FIAGLIAIV (SEQ ID NO: 2370)	SARS-CoV-2 Spike glycoprotein 1220-1228 (confirmed epitope)
A*02:01	ALNTLVKQL (SEQ ID NO: 2371)	SARS-CoV Spike glycoprotein precursor 940-948 (conserved in SARS-CoV-2)
A*02:01	LITGRLOSL (SEQ ID NO: 2372)	SARS-CoV-2 Spike glycoprotein 996-1004 (confirmed epitope)
A*02:01	NLNESLIDL (SEQ ID NO: 2373)	SARS-CoV Spike glycoprotein precursor 1174-1182 (conserved in SARS-Cov-2)
A*02:01	VLNDILSRL (SEQ ID NO: 2374)	SARS-CoV Spike glycoprotein precursor 958-966 (conserved in SARS-Cov-2)
A*02:01	YLQPRTFLL (SEQ ID NO: 2375)	SARS-Cov-2 Spike glycoprotein 269-277 (confirmed epitope)
A*02:01	LLYDANYFL (SEQ ID NO: 2376)	SARS-CoV-2 ORF3a 139-147 (confirmed epitope)
A*02:01	RLQSLQTYV (SEQ ID NO: 2377)	SARS-CoV-2 Spike glycoprotein 1000-1008 (confirmed subdominant epitope)
A*02:01	KLWAQCVQL (SEQ ID NO: 2378)	SARS-CoV-2 ORF1ab 3886-3894 (confirmed epitope)
A*02:01	TLYAVATTI (SEQ ID NO: 2379)	Dengue NS4b 40-48
A*02:01	KLAEIFKL (SEQ ID NO: 2380)	Dengue NS5 563-571
A*02:01	ILIRTGLLVI (SEQ ID NO: 2381)	Dengue NS2b 97-106
A*02:01	AIKRGLRTL (SEQ ID NO: 2382)	Dengue NS3 112-120
A*02:01	LLLGLMILL (SEQ ID NO: 2383)	Dengue NS4a 56-64
A*02:01	VLLLVTHYA (SEQ ID NO: 2384)	Dengue NS4b 111-119
A*02:01	GLCTLVAML (SEQ ID NO: 2385)	EBV BMLF-1 259-267
A*02:01	CLGGLTMMV (SEQ ID NO: 2386)	EBV LMP-2 426-434
A*02:01	YLLEMLWRL (SEQ ID NO: 2387)	EBV LMP-1 125-133
A*02:01	YLQQNWWTL (SEQ ID NO: 2388)	EBV LMP1 159-167
A*02:01	YVLDHLIVV (SEQ ID NO: 2389)	EBV BRLF1 109-117
A*02:01	FLYALALLL (SEQ ID NO: 2390)	EBV LMP-2 356-364
A*02:01	TLDYKPLSV (SEQ ID NO: 2391)	EBV BMRF1 208-216
A*02:01	LLDFVRFMGV (SEQ ID NO: 2392)	EBV EBNA-3C 284-293
A*02:01	FLDKGTYYL (SEQ ID NO: 2393)	EBV BALF-4 276-284
A*02:01	FLPSDFFPSV (SEQ ID NO: 2394)	HBV core antigen 18-27

A*02:01 FLLTRILTI (SEQ ID NO: 2395) HBV envelope 183-191
 A*02:01 GLSPTVWLSV (SEQ ID NO: 2396) HBV surface antigen 185-194
 A*02:01 WLSLLVPFV (SEQ ID NO: 2397) HBV surface antigen 172-181
 A*02:01 FLLSLGIHL (SEQ ID NO: 2398) HBV polymerase 573-581
 A*02:01 FLPSDFFPSI (SEQ ID NO: 2399) HBV core 18-27 (subtype ADR4)
 A*02:01 VLHKRTLGL (SEQ ID NO: 2400) HBV X 92-100
 A*02:01 GLSRYVARL (SEQ ID NO: 2401) HBV Pol 455-463
 A*02:01 YMDDVVLGA (SEQ ID NO: 2402) HBV Polymerase 548-556
 A*02:01 KLHLYSHPI (SEQ ID NO: 2403) HBV Pol 502-510
 A*02:01 ELMTLATWV (SEQ ID NO: 2404) HBV core protein 64-72
 A*02:01 DLMGYIPAV (SEQ ID NO: 2405) HCV core 132-140
 A*02:01 CINGVCWTV (SEQ ID NO: 2406) HCV NS3 1073-1081
 A*02:01 YLLPRRGPR (SEQ ID NO: 2407) HCV core 35-44
 A*02:01 VLSDFKTWL (SEQ ID NO: 2408) HCV NS5a 1987-1995
 A*02:01 ALYDVVTKL (SEQ ID NO: 2409) HCV NS5b 2594-2602
 A*02:01 KLVALGINAV (SEQ ID NO: 2410) HCV NS3 1406-1415
 A*02:01 LLFNILGGWV (SEQ ID NO: 2411) HCV NS4b 1807-1816
 A*02:01 KLSGLGINAV (SEQ ID NO: 2412) HCV NS3 1406-1415
 A*02:01 DLMGYIPLV (SEQ ID NO: 2413) HCV core 132-140
 A*02:01 CVNGVCWTV (SEQ ID NO: 2414) HCV NS3 1073-1081
 A*02:01 GLQDCTMLV (SEQ ID NO: 2415) HCV NS5B 2727-2735
 A*02:01 SLYNTVATL (SEQ ID NO: 2416) HIV-1 gag p17 76-84
 A*02:01 ILKEPVHGV (SEQ ID NO: 2417) HIV-1 RT 476-484
 A*02:01 TLNAWVKV (SEQ ID NO: 2418) HIV-1 gag p24 19-27
 A*02:01 KLTPLCVTL (SEQ ID NO: 2419) HIV-1 env gp120 90-98
 A*02:01 GLADQLIHL (SEQ ID NO: 2420) HIV-1 vif 101-109
 A*02:01 LTFGWCFKL (SEQ ID NO: 2421) HIV-1 nef 137-145
 A*02:01 FLGKIWPS (SEQ ID NO: 2422) Gag 433-440
 A*02:01 ALVEMGHHA (SEQ ID NO: 2423) HIV Vpu 66-74
 A*02:01 RTLNAWVKV (SEQ ID NO: 2424) HIV gag 150-158
 A*02:01 NVWATHACV (SEQ ID NO: 2425) HIV env gp 67-7
 A*02:01 SLLNATAIAV (SEQ ID NO: 2426) HIV env 816-825
 A*02:01 SLFNTVATL (SEQ ID NO: 2427) HIV gag 77-85
 A*02:01 SLVKHHMYI (SEQ ID NO: 2428) HIV vif 23-31
 A*02:01 VIYHYVDDL (SEQ ID NO: 2429) HIV pol
 A*02:01 YMLDLQPETT (SEQ ID NO: 2430) HPV 16 E7 11-20
 A*02:01 KLPQLCTEL (SEQ ID NO: 2431) HPV 16 E6 18-26
 A*02:01 YMLDLQPET (SEQ ID NO: 2432) HPV 16 E7 11-19
 A*02:01 MLDLQPETT (SEQ ID NO: 2433) HPV 16 E7 12-20
 A*02:01 VLMIKALEL (SEQ ID NO: 2434) Non muscle Myosin-9 741-749
 A*02:01 QLFNHTMFI (SEQ ID NO: 2435) Non-muscle Myosin 478-486
 A*02:01 QMARLAWEA (SEQ ID NO: 2436) 1116-1124
 A*02:01 LLFGYPVYV (SEQ ID NO: 2437) Human T-cell lymphotropic virus-1 (HTLV-1) tax 11-19
 A*02:01 AVLDGLLSL (SEQ ID NO: 2438) HTLV bZIP factor 42-50
 A*02:01 GLLSLEEEL (SEQ ID NO: 2439) bZIP factor 26-34
 A*02:01 GILGFVFTL (SEQ ID NO: 2440) Influenza A MP 58-66
 A*02:01 ILGFVFTLTV (SEQ ID NO: 2441) Influenza A MP 59-68
 A*02:01 KLGEFYNQMM (SEQ ID NO: 2442) Flu BNP 85-94 (Influenza B)
 A*02:01 SITEVECFL (SEQ ID NO: 2443) VP1 36-44
 A*02:01 ILMWEAVTL (SEQ ID NO: 2444) VP1 100-108

A*02:01	ALPHIIDEV (SEQ ID NO: 2445)	LCMV envelope gp 10-18
A*02:01	YLVSIHL (SEQ ID NO: 2446)	LCMV envelope gp 447-455
A*02:01	SLNQTVHSL (SEQ ID NO: 2447)	NP 69-77
A*02:01	YLNKIQNSL (SEQ ID NO: 2448)	Plasmodium falciparum CSP 334-342
A*02:01	FIDSYICQV (SEQ ID NO: 2449)	miHAg H-Y (human SMCY) 311-319
A*02:01	YIGEVLSV (SEQ ID NO: 2450)	HA-2
A*02:01	VLHDDLLEA (SEQ ID NO: 2451)	Minor Histocompatibility Antigen HA-1 137-145
A*02:01	RTLDKVLEV (SEQ ID NO: 2452)	miHAg HA-8
A*02:01	NEGATIVE (SEQ ID NO: 2453)	Negative Control
A*02:01	TMFPHIIVDV (SEQ ID NO: 2454)	Norovirus VP1 139-148
A*02:01	LLDVPTAAV (SEQ ID NO: 2455)	Interferon gamma inducible protein (GILT) 30 27-35
A*02:01	RILGAVAKV (SEQ ID NO: 2456)	Vinculin 822-830
A*02:01	LMWYELSKI (SEQ ID NO: 2457)	KSHVF-8 gB.492-500
A*02:01	ILEDIVLTL (SEQ ID NO: 2458)	Streptococcus pyogenes Cas9 615-623
A*02:01	KMLKEMGEV (SEQ ID NO: 2459)	RSV NP 137-145
A*02:01	KLIANNTRV (SEQ ID NO: 2460)	Mycobacterium bovis antigen 85-A 200-208
A*02:01	GLPVEYLQV (SEQ ID NO: 2461)	Mycobacterium bovis antigen 85-A 6-14
A*02:01	GILTVSVAV (SEQ ID NO: 2462)	16 kDa
A*02:01	AMASTEGRV (SEQ ID NO: 2463)	ESAT-6
A*02:01	VLTDGNPPEV (SEQ ID NO: 2464)	19 kDa
A*02:01	KVDDTFYYV (SEQ ID NO: 2465)	Vaccinia virus Host range protein 2 74-82
A*02:01	ILDDNLYKV (SEQ ID NO: 2466)	Vaccinia virus Copenhagen Protein G5 18-26
A*02:01	ALWALPHAA (SEQ ID NO: 2467)	IE62 593-601
A*02:01	RLDDDGNFQL (SEQ ID NO: 2468)	West Nile Virus NY-99 polyprotein precursor (1452-1461)
A*02:01	ATWAENIQV (SEQ ID NO: 2469)	West Nile virus NY-99 polyprotein precursor 3390-3398
A*02:01	YTMDGEYRL (SEQ ID NO: 2470)	West Nile virus NY-99 polyprotein precursor 2023-2031
A*02:01	SVGGVFTSV (SEQ ID NO: 2471)	WNV envelope gp 430-438
A*02:01	SLFGQRIEV (SEQ ID NO: 2472)	WNV nonstructural protein 4B 15-23
A*02:01	LLWNGPMAV (SEQ ID NO: 2473)	NS4B 214-222
A*03:01	KQSSKALQR (SEQ ID NO: 2474)	bcr-abl 210 kD fusion protein 21-29
A*03:01	ALLAVGATK (SEQ ID NO: 2475)	gp100 (pmel17) 17-25
A*03:01	ATGFKQSSK (SEQ ID NO: 2476)	bcr-abl 210 kD fusion protein 259-269
A*03:01	RISTFKNWPK (SEQ ID NO: 2477)	Survivin-3A 18-27 (27K)
A*03:01	RLGLQVRKNK (SEQ ID NO: 2478)	RhoC 176-185 (177L)
A*03:01	RLLFFAPTR (SEQ ID NO: 2479)	Mcl-1 95-103
A*03:01	QVLKKIAQK (SEQ ID NO: 2480)	HMOX1 145-153
A*03:01	RIAAWMATY (SEQ ID NO: 2481)	165-173
A*03:01	KLGGALQAK (SEQ ID NO: 2482)	HCMV IE1 184-192
A*03:01	KTFPPTPEK (SEQ ID NO: 2483)	SARS-CoV-2 Nucleocapsid protein 362-370 (confirmed epitope)
A*03:01	ELERAADV (SEQ ID NO: 2484)	Dengue NS2b 52-60
A*03:01	RVSTVQQLTK (SEQ ID NO: 2485)	Dengue C 22-31
A*03:01	RIEPSWADV (SEQ ID NO: 2486)	Dengue NS3 64-74
A*03:01	RVIDPRRCMK (SEQ ID NO: 2487)	Dengue NS3 422-431
A*03:01	KITAEWLWK (SEQ ID NO: 2488)	Dengue NS5 375-383
A*03:01	RLRAEAQVK (SEQ ID NO: 2489)	EBV EBNA 3A 603-611
A*03:01	RVRAYTYSK (SEQ ID NO: 2490)	EBV BRLF1
A*03:01	RVCEKMALY (SEQ ID NO: 2491)	HCV NS5B 2588-2596

A*03:01 QVPLRPMTYK (SEQ ID NO: 2492) HIV-1 nef 73-82
 A*03:01 RLRPGGKKK (SEQ ID NO: 2493) HIV-1 gag p17 19-27
 A*03:01 AIFQSSMTK (SEQ ID NO: 2494) HIV pol 325-333
 A*03:01 KLCLRFLSK (SEQ ID NO: 2495) HPV 33 E6 64-72
 A*03:01 ILRGSAVHK (SEQ ID NO: 2496) Influenza A (PR8) NP 265-274
 A*11:01 KTFPPTPEPK (SEQ ID NO: 2483) SARS-CoV-2 Nucleocapsid protein 362-370
 (confirmed epitope)
 A*11:01 GTSGSPIINR (SEQ ID NO: 2497) Dengue NS3 serotype 3&4 133-142
 A*11:01 GTSGSPIIDK (SEQ ID NO: 2498) Dengue NS3 133-142
 A*11:01 GTSGSPIVNR (SEQ ID NO: 2499) NS3 serotype 1 133-142
 A*11:01 GTSGSPIVDR (SEQ ID NO: 2500) Dengue NS3 serotype 2 133-142
 A*11:01 GTSGSPIADK (SEQ ID NO: 2501) Dengue NS3 133-142
 A*11:01 RVSTVQQLTk (SEQ ID NO: 2485) Dengue C 22-31
 A*11:01 RIEPSWADVk (SEQ ID NO: 2486) Dengue NS3 64-74
 A*11:01 RVIDPRRCMK (SEQ ID NO: 2487) Dengue NS3 422-431
 A*11:01 KITAEWLWK (SEQ ID NO: 2488) Dengue NS5 375-383
 A*11:01 IVTDFSVIK (SEQ ID NO: 2502) EBV EBNA-4 416-424
 A*11:01 SSCSSCPLSK (SEQ ID NO: 2503) EBV LMP-2 340-349
 A*11:01 ATIGTAMYK (SEQ ID NO: 2504) EBV BRLF1 134-142
 A*11:01 AVFDRKSDAK (SEQ ID NO: 2505) EBNA3B 399-408
 A*11:01 YVNVNMGLK (SEQ ID NO: 2506) HBV core antigen 88-96
 A*11:01 YVNTNMGLK (SEQ ID NO: 2507) HBV core 88-96
 A*11:01 STLPETTVVRR (SEQ ID NO: 2508) HBV core 141-151
 A*11:01 AVDLSHFLK (SEQ ID NO: 2509) HIV nef 84-92
 A*11:01 ACQGVGGPGHK (SEQ ID NO: 2510) HIV gag p24
 A*11:01 NTLEQTVKK (SEQ ID NO: 2511) HPV 33 E6 86-94
 A*11:01 SIIPSGPLK (SEQ ID NO: 2512) Influenza A MP 13-21
 A*11:01 RMVLASTTAK (SEQ ID NO: 2513) Influenza A MP1 178-187
 A*11:01 KSMREEYRK (SEQ ID NO: 2514) Influenza A MP2 70-78
 A*24:02 TYFSLNNKF (SEQ ID NO: 2515) Adenovirus 5 Hexon 37-45
 A*24:02 TYACFVSNL (SEQ ID NO: 2516) Carcinogenic Embryonic Antigen (CEA) 652-660
 A*24:02 AFLPWHLRF (SEQ ID NO: 2517) Tyrosinase 188-196
 A*24:02 IMPKAGLLI (SEQ ID NO: 2518) MAGE-A3
 A*24:02 VYFFLPDHL (SEQ ID NO: 2519) gp100-intron 4 (170-178)
 A*24:02 EYLQLVFGI (SEQ ID NO: 2520) MAGEA2 156-164
 A*24:02 TYLPTNASL (SEQ ID NO: 2521) HER-2/neu 63-71
 A*24:02 VYGFVRACL (SEQ ID NO: 2522) Telomerase reverse transcriptase (hTERT) 461-469
 A*24:02 TFPDLESEF (SEQ ID NO: 2523) MAGEA3 97-105
 A*24:02 DYLLQYVLQI (SEQ ID NO: 2524) MiHA ACC1 15-23
 A*24:02 RYCNLEGPII (SEQ ID NO: 2525) Lymphocyte antigen 6 complex locus K (LY6K) 177-186
 A*24:02 AYACNTSTL (SEQ ID NO: 2526) Survivin 80-88
 A*24:02 CYASGWGSI (SEQ ID NO: 2527) Prostate Specific Antigen-1 153-161
 A*24:02 DYLLNEWGSRF (SEQ ID NO: 2528) CDH3 807-816
 A*24:02 EYCPGGNLF (SEQ ID NO: 2529) MELK 87-95 (93N)
 A*24:02 EYYELFVNI (SEQ ID NO: 2530) DEP DC1 294-302
 A*24:02 GYCTQIGIF (SEQ ID NO: 2531) HENMT1 221-229
 A*24:02 IYTWIEDHF (SEQ ID NO: 2532) FOXM1 262-270
 A*24:02 NYQPVWLCL (SEQ ID NO: 2533) RNF43 721-729 (722Y)
 A*24:02 RYNAQCQETI (SEQ ID NO: 2534) Midkine 110-119
 A*24:02 EYRALQLHL (SEQ ID NO: 2535) CA9 219-227

A*24:02 SYRNEIAYL (SEQ ID NO: 2536) TTK protein kinase 551-559
 A*24:02 VYLRVRPLL (SEQ ID NO: 2537) KIF20A 67-75
 A*24:02 VYYNWQYLL (SEQ ID NO: 2538) IL13r 146-154
 A*24:02 VYALPLKML (SEQ ID NO: 2539) HCMV pp65 113-121
 A*24:02 QYDPVAALF (SEQ ID NO: 2540) HCMV pp65 341-349
 A*24:02 AYAQKIFKI (SEQ ID NO: 2541) CMV IE-1 248-256
 A*24:02 QYSDRRWCF (SEQ ID NO: 2542) Dengue NS3 557-565 (Singapore/S275/1990)
 A*24:02 TYGPVFMCL (SEQ ID NO: 2543) EBV LMP-2 419-427
 A*24:02 PYLFWLAAI (SEQ ID NO: 2544) EBV LMP2 131-139
 A*24:02 TYGPVFMSL (SEQ ID NO: 2545) EBV LMP2 419-427
 A*24:02 EYLVSGFVW (SEQ ID NO: 2546) HBV core 117-125
 A*24:02 KYTSFPWLL (SEQ ID NO: 2547) HBV polymerase 756-764
 A*24:02 FFPSIRDLL (SEQ ID NO: 2548) HBV core protein 23-31
 A*24:02 AYSQQTRGL (SEQ ID NO: 2549) HCV NS3 1031-1039
 A*24:02 RYPLTFGWY (SEQ ID NO: 2550) HIV-1 Nef 134-143
 A*24:02 RYLKDQQLL (SEQ ID NO: 2551) HIV-1 gag gp41 67-75
 A*24:02 RYLRDQQLL (SEQ ID NO: 2552) HIV env
 A*24:02 RYPLTFGWCF (SEQ ID NO: 2553) HIV nef 143-152
 A*24:02 RYPLTFGW (SEQ ID NO: 2554) HIV nef
 A*24:02 VYDFAFRDL (SEQ ID NO: 2555) HPV16 E6
 A*24:02 SFHSLHLLF (SEQ ID NO: 2556) HTLV Tax 301-309
 A*29:02 KEKYIDQEEL (SEQ ID NO: 2557) HSP90 alpha 280-288 (Pathologic Conditions)
 A*29:02 LYNTVATLY (SEQ ID NO: 2558) HIV gag 79-86
 A*29:02 SFDPIPIHY (SEQ ID NO: 2559) HIV env 216-224
 A*29:02 SFNCRGEFFY (SEQ ID NO: 2560) HIV env 382-391
 A*68:01 TVSGNILTIR (SEQ ID NO: 2561) NY-ESO-1 127-136
 B*07:02 VPQYGYLTL (SEQ ID NO: 2562) AAV2 372-380
 B*07:02 KPYSGTAYNSL (SEQ ID NO: 2563) Adenovirus Hexon 114-124
 B*07:02 KPYSGTAYNAL (SEQ ID NO: 2564) Adenovirus Hexon 114-124
 B*07:02 LPLMRKAYL (SEQ ID NO: 2565) LT antigen 27-35
 B*07:02 LPWHRLFLL (SEQ ID NO: 2566) Tyrosinase 208-216
 B*07:02 EPR(PHOSPHO-S)PSHSM (SEQ ID NO: 2567) Insulin receptor substrate 2
 B*07:02 TPNQRQNVC (SEQ ID NO: 2568) P2X5
 B*07:02 APRGVRMAV (SEQ ID NO: 2569) LAGE-1 46-54
 B*07:02 LPVSPRLQL (SEQ ID NO: 2570) CEACAM 185-193
 B*07:02 TPRVTGGGAM (SEQ ID NO: 2571) HCMV pp65 417-426
 B*07:02 RPHERNGFTVL (SEQ ID NO: 2572) HCMV pp65 265-275
 B*07:02 SPRWYFYLL (SEQ ID NO: 2573) SARS-CoV-2 Nucleocapsid protein 105-113
 (confirmed epitope)
 B*07:02 APTRVVAAEM (SEQ ID NO: 2574) Dengue NS3 serotype 2 222-231
 B*07:02 RPPIFIRRL (SEQ ID NO: 2575) EBV EBNA-3A 247-255
 B*07:02 RPQGGSRPEFVKL (SEQ ID NO: 2576) EBV BMRF1 116-128
 B*07:02 QPRAPIRPI (SEQ ID NO: 2577) EBV EBNA-3C 881-889
 B*07:02 LPSDFFPSV (SEQ ID NO: 2578) HBV core 19-27
 B*07:02 GPRLGVRAT (SEQ ID NO: 2579) HCV core 41-49
 B*07:02 DPRRRSRNL (SEQ ID NO: 2580) HCV core 111-119
 B*07:02 IPRRIRQGL (SEQ ID NO: 2581) HIV-1 env gp120 848-856
 B*07:02 TPGPGVRYPL (SEQ ID NO: 2582) HIV-1 nef 128-137
 B*07:02 GPGHKARVL (SEQ ID NO: 2583) HIV gag p24 223-231
 B*07:02 KPTLKEYVL (SEQ ID NO: 2584) HPV 33 E7 5-13
 B*07:02 LPVSCPEDL (SEQ ID NO: 2585) bZIP factor 10-18

B*07:02 QPEWFRNVL (SEQ ID NO: 2586) Influenza A PB1 329-337
 B*07:02 SPIVPSFDM (SEQ ID NO: 2587) Influenza A NP 473-481
 B*07:02 SPSVDKARAE (SEQ ID NO: 2588) MiHAg SMCY 1041-1051
 B*08:01 GFKQSSKAL (SEQ ID NO: 2589) bcr-abl 210 kD fusion protein 19-27
 B*08:01 ELRRKMMYM (SEQ ID NO: 2590) IE1 199-207
 B*08:01 ELKRKMIYM (SEQ ID NO: 2591) CMV IE-1 199-207 (R/L Position 3, M/I Position 7)
 B*08:01 RIKQKGIL (SEQ ID NO: 2592) Dengue NS3 25-32
 B*08:01 LEKTKKDL (SEQ ID NO: 2593) Dengue NS4a 6-13
 B*08:01 FLRGRAYGL (SEQ ID NO: 2594) EBV EBNA-3A 193-201
 B*08:01 RAKFKQLL (SEQ ID NO: 2595) EBV BZLF-1 190-197
 B*08:01 QAKWRLQTL (SEQ ID NO: 2596) EBV EBNA3A 158-166
 B*08:01 GLKILQLL (SEQ ID NO: 2597) HBV external core Ag
 B*08:01 HSKKKCDEL (SEQ ID NO: 2598) HCV NS3 1395-1403
 B*08:01 FLKEKGGL (SEQ ID NO: 2599) HIV-1 nef 90-97
 B*08:01 GEIYKRWII (SEQ ID NO: 2600) HIV-1 gag p24 261-269
 B*08:01 EIYKRWII (SEQ ID NO: 2601) HIV p24 gag 128-135
 B*08:01 YLKDQQLL (SEQ ID NO: 2602) Env 586-593
 B*15:01 CLIPTAMAF (SEQ ID NO: 2603) Dengue C 107-115
 B*15:01 RLRPGGKKKY (SEQ ID NO: 2604) HIV-1 p17 20-29
 B*27:05 GRFGLATEK (SEQ ID NO: 2605) BRAF 594-601 (600E)
 B*27:05 GRFGLATVK (SEQ ID NO: 2606) BRAF 594-601 (600V)
 B*27:05 RMFPNAPYL (SEQ ID NO: 2362) WT-1 126-134 (Wilms tumor)
 B*27:05 ARKLLLDNL (SEQ ID NO: 2607) PqqC-like protein 70-78
 B*27:05 NRAKQVIKL (SEQ ID NO: 2608) Probable ATP-dependent Clp protease ATP-binding subunit 7-15
 B*27:05 QRNAPRITF (SEQ ID NO: 2609) SARS-CoV-2 Nucleocapsid protein 9-17 (confirmed epitope)
 B*27:05 ARMILMTHF (SEQ ID NO: 2610) HCV NS5B 2841-2849
 B*27:05 KRWIILGLNK (SEQ ID NO: 2611) HIV-1 gag p24 265-274
 B*27:05 KRWIIMGLNK (SEQ ID NO: 2612) HIV-1 Gag p24 263-272
 B*27:05 GRAFVTIGK (SEQ ID NO: 2613) HIV-1 gp100 103-111
 B*27:05 SRYWAIRTR (SEQ ID NO: 2614) Influenza A NP 383-391
 B*35:01 IPYLDGTFY (SEQ ID NO: 2615) Adenovirus Hexon
 B*35:01 MPFATPMEA (SEQ ID NO: 2616) NY-ESO-1 94-102
 B*35:01 IPSINVHHY (SEQ ID NO: 2617) HCMV pp65 123-131
 B*35:01 TPEGIITL (SEQ ID NO: 2618) Dengue NS3 500-508
 B*35:01 YPLHEQHGM (SEQ ID NO: 2619) EBV EBNA-3A 458-466
 B*35:01 EPLPQGQLTAY (SEQ ID NO: 2620) EBV BZLF-1 54-64
 B*35:01 HPVAEADYFEY (SEQ ID NO: 2621) EBV EBNA-1 407-417(4A)
 B*35:01 HPNIEEVAL (SEQ ID NO: 2622) HCV NS3 1359-1367
 B*35:01 CPNSSIVY (SEQ ID NO: 2623) HCV E1 207-214
 B*35:01 NPDIVIQY (SEQ ID NO: 2624) HIV-1 RT 330-338
 B*35:01 NPDIVIQY (SEQ ID NO: 2625) HIV-1 HIV-1 RT 328-336
 B*35:01 VPLDEDFRKY (SEQ ID NO: 2626) HIV-1 HIV-1 RT 273-282
 B*40:01 MEVTPSGTWL (SEQ ID NO: 2627) SARS-CoV-2 Nucleocapsid protein 322-330 (confirmed epitope)
 B*40:01 GEARKTFVEL (SEQ ID NO: 2628) Dengue NS3 528-537
 B*40:01 IEDPPFNSL (SEQ ID NO: 2629) EBV LMP2 200-208
 B*40:01 REISVPAEIL (SEQ ID NO: 2630) HCV NS5a 2266-2275
 B*40:01 KEKGGLEGL (SEQ ID NO: 2631) HIV-1 Nef 92-100
 C*06:02 TRATKMQVI (SEQ ID NO: 2632) pp65 211-219

C*06:02 QIKVRVDM (SEQ ID NO: 2633) IE1 88-95
 C*06:02 TRRFLPQIL (SEQ ID NO: 2634) NS3 205-213
 C*07:02 CRVLCCYVL (SEQ ID NO: 2635) IE1 309-317
 E*01:01 VMAPRTLIL (SEQ ID NO: 2636) HLA-C leader sequence peptide
 E*01:01 VMAPRTLVL (SEQ ID NO: 2637) HLA-A leader sequence peptide
 A*01:01 SADNNNSEY (SEQ ID NO: 2082) AAV VP1 492-500
 A*01:01 TDLGQNLLY (SEQ ID NO: 2083) Adenovirus 5 Hexon 886-894
 A*01:01 EADPTGHSY (SEQ ID NO: 2084) MAGE-A1 161-169
 A*01:01 EVDPIGHLY (SEQ ID NO: 2085) MAGE-A3 168-176
 A*01:01 KSDICTDEY (SEQ ID NO: 2086) Tyrosinase 243-251 (244S)
 A*01:01 KCDICTDEY (SEQ ID NO: 2087) Tyrosinase 243-251
 A*01:01 QSLEIISRY (SEQ ID NO: 2088) Mcl-1 177-185
 A*01:01 YVDFREYEYY (SEQ ID NO: 2089) FLT3 ITD
 A*01:01 TLDTLTAFY (SEQ ID NO: 2090) Mesothelin 429-437
 A*01:01 LTDDRLFTCY (SEQ ID NO: 2091) PLEKHM2
 A*01:01 DSDPDSFQDY (SEQ ID NO: 2092) Tyr A1a 454-463
 A*01:01 EADPIGHLY (SEQ ID NO: 2093) MAGEA3
 A*01:01 EVDPASNTY (SEQ ID NO: 2094) MAGE-A4 169-177
 A*01:01 HSTNGVTRIIY (SEQ ID NO: 2095) PSMA
 A*01:01 ILDTAGREEY (SEQ ID NO: 2096) N-ras 55-64
 A*01:01 LVDVMPWLQY (SEQ ID NO: 2097) Cytochrome P450 240-249
 A*01:01 RSDSGQQARY (SEQ ID NO: 2098) AIM-2
 A*01:01 VTEPGTAQY (SEQ ID NO: 2099) Minor antigen HA-3T (Lbc oncogene 451-459)
 A*01:01 VYDFFVWLHY (SEQ ID NO: 2100) TRP-2 181-190
 A*01:01 YSEHPTFTSQY (SEQ ID NO: 2101) HCMV pp65 363-373
 A*01:01 VTEHDTLLY (SEQ ID NO: 2102) HCMV pp50 245-253
 A*01:01 FTSDYYQLY (SEQ ID NO: 2103) SARS-CoV-2 ORF3a 207-215 (confirmed epitope)
 A*01:01 TTDPSTFLGRY (SEQ ID NO: 2638) SARS-CoV-2 Replicase polyprotein 1ab 1637-1646
 (confirmed epitope)
 A*01:01 PTDNYITTY (SEQ ID NO: 2639) SARS-CoV-2 Replicase polyprotein 1ab 1621-1629
 (confirmed epitope)
 A*01:01 LLDTASALY (SEQ ID NO: 2106) HBV core 30-38
 A*01:01 ATDALMTGY (SEQ ID NO: 2107) HCV NS3 1435-1443
 A*01:01 ATDALMTGF (SEQ ID NO: 2108) HCV NS3 1436-1444
 A*01:01 CTELKLSY (SEQ ID NO: 2109) Influenza A (PR8) NP 44-52
 A*01:01 VSDGGPNLY (SEQ ID NO: 2110) Influenza A PB1 591-599
 A*01:01 IVDCLTEMY (SEQ ID NO: 2111) DRRFY (1521-1529))
 A*0201 ALCNTDSPL (SEQ ID NO: 2112) iLR1
 A*0201 ALKDVEERV (SEQ ID NO: 2113) MAGE-C2 336-344
 A*0201 LLAARAIVAI (SEQ ID NO: 2114) iLR1 59-68
 A*0201 RLWQELSDI (SEQ ID NO: 2115) circadian clock protein PASD1 691-700
 A*0201 LLFGLALIEV (SEQ ID NO: 2116) MAGE-C2 191-200
 A*0201 FLDPRPLTV (SEQ ID NO: 2117) CYP190
 A*0201 STLCQVEPV (SEQ ID NO: 2118) MPP11
 A*0201 VLQMKEEDV (SEQ ID NO: 2119) iLR1
 A*0201 AIQDLCLAV (SEQ ID NO: 2120) NPM1
 A*0201 QLLIKAVNL (SEQ ID NO: 2121) MPP11
 A*0201 AIQDLCAVAV (SEQ ID NO: 2122) NPM1
 A*0201 ALTPVVVTL (SEQ ID NO: 2123) cyclin-dependent kinase 4 170-178
 A*02:01 KLQVFLIVL (SEQ ID NO: 2640) T1D Diabetes human prepro islet amyloid
 polypeptide pplAPP 5-13

A*02:01 VMNILLQYV (SEQ ID NO: 2125) GAD65 114-123
 A*02:01 SLSRFSWGA (SEQ ID NO: 2126) Myelin basic protein 110-118
 A*02:01 HLVEALYLV (SEQ ID NO: 2127) Insulin B chain 10-18
 A*02:01 LNIDLLWSV (SEQ ID NO: 2128) T1D Diabetes IGRP 228-236
 A*02:01 VLFGLGFAI (SEQ ID NO: 2129) T1D Diabetes IGRP 265-273
 A*02:01 ALWGPDPAAA (SEQ ID NO: 2130) Proinsulin precursor 15-24
 A*02:01 MVWESGCTV (SEQ ID NO: 2131) IA-2 797-805
 A*02:01 YTCPLCRAPV (SEQ ID NO: 2132) SSA SS-56 55-64
 A*02:01 VIVMLTPLV (SEQ ID NO: 2133) IA-2 805-813
 A*02:01 AITEVECFL (SEQ ID NO: 2134) VP1 44-52
 A*02:01 FLHCIVFNV (SEQ ID NO: 2135) large T antigen 410-418
 A*02:01 LLMWEAVTV (SEQ ID NO: 2136) VP1 108-116
 A*02:01 CLLPKMDSV (SEQ ID NO: 2137) large T antigen 398-406
 A*02:01 FLWGPRALV (SEQ ID NO: 2138) MAGEA3 271-279
 A*02:01 IMDQVPFSV (SEQ ID NO: 2139) gp100 (pmel17) 209-217
 A*02:01 YLEPGPVTV (SEQ ID NO: 2140) gp100 (pmel) 280-288 (288V)
 A*02:01 YLSGADLNL (SEQ ID NO: 2141) Carcinoembryonic antigen (CEA)-derived peptide
 CAP1-6D
 A*02:01 SLLMWITQC (SEQ ID NO: 2142) NY-ESO-1 157-165 (9C)
 A*02:01 KTWGQYWQV (SEQ ID NO: 2143) gp100 (pmel17) 154-162
 A*02:01 YLEPGPVTA (SEQ ID NO: 2144) gp100
 A*02:01 YMDGTMSQV (SEQ ID NO: 2145) Tyrosinase 369-377 (371D)
 A*02:01 YLSGANLNL (SEQ ID NO: 2146) Carcinogenic Embryonic Antigen (CEA) 571-579
 A*02:01 ELAGIGILTV (SEQ ID NO: 2147) MelanA / MART 26-35
 A*02:01 ILAKFLHWL (SEQ ID NO: 2148) Telomerase 540-548
 A*02:01 ALQPGTALL (SEQ ID NO: 2149) Prostate Stem Cell Antigen (PSCA) 14-22
 A*02:01 VISNDVCAQV (SEQ ID NO: 2150) Prostate Specific Antigen-1 (PSA-1) 154-163
 A*02:01 RLVDDFLLV (SEQ ID NO: 2151) Telomerase Reverse Transcriptase 865-873
 A*02:01 GVLVGVALI (SEQ ID NO: 2152) Carcinogenic Embryonic Antigen (CEA) 694-702
 A*02:01 VLYRYGSFSV (SEQ ID NO: 2153) gp100 (pmel17) 476-485
 A*02:01 PLFQVPEPV (SEQ ID NO: 2154) Alpha-fetoprotein isoform 1 137-145
 A*02:01 FMNKFYIEI (SEQ ID NO: 2155) Human alfa fetoprotein 158-166
 A*02:01 GLSPNLNRFL (SEQ ID NO: 2156) Alpha-fetoprotein isoform 2 167-176
 A*02:01 KVLEYVIKV (SEQ ID NO: 2157) MAGEA1 278-286
 A*02:01 LLGRNSFEV (SEQ ID NO: 2158) p53 264-272
 A*02:01 LLLTLVLTV (SEQ ID NO: 2159) MUC-1 12-20
 A*02:01 ILHNGAYSL (SEQ ID NO: 2160) HER-2/neu 435-443
 A*02:01 RLLQETELV (SEQ ID NO: 2161) HER-2/neu 689-697
 A*02:01 KIFGSLAFL (SEQ ID NO: 2162) HER-2/neu 369-377
 A*02:01 LLLLDVAPL (SEQ ID NO: 2163) HSP1A 459-467
 A*02:01 LLDVAPLSL (SEQ ID NO: 2164) HSP1A 461-469
 A*02:01 HLYQGCQVV (SEQ ID NO: 2165) Receptor tyrosine-protein kinase erbB-2 48-56
 A*02:01 HLSTAFARV (SEQ ID NO: 2166) G250 (renal cell carcinoma) 217-225
 A*02:01 VLQELNVTV (SEQ ID NO: 2167) Leukocyte Proteinase-3 (Wegener's autoantigen)
 169-177
 A*02:01 KVAELVHFL (SEQ ID NO: 2168) MAGEA3 112-120
 A*02:01 VLAGVGFFI (SEQ ID NO: 2169) EPHA2 550-558
 A*02:01 FLYTLLREV (SEQ ID NO: 2170) STEAP 86-94
 A*02:01 ILLWQPIPV (SEQ ID NO: 2171) Prostatic Acid Phosphatase-3 (PAP-3) 135-143
 A*02:01 RLQEERTCKV (SEQ ID NO: 2172) BIR
 A*02:01 QLCPICRAPV (SEQ ID NO: 2173) Livin/ML-IAP280 175-184

A*02:01 VLGEAWRDQV (SEQ ID NO: 2174) TRAP 45-54
 A*02:01 LLLTVLTVV (SEQ ID NO: 2175) Tumor Mucin Antigen 13-21
 A*02:01 GLYDGMEHL (SEQ ID NO: 2176) MAGEA-10 254-262
 A*02:01 SLLMWITQV (SEQ ID NO: 2177) NY-ESO-1 157-165
 A*02:01 LMLGEFLKL (SEQ ID NO: 2178) Survivin 96-104
 A*02:01 YLFFYRKSV (SEQ ID NO: 2179) mTERT 572-580
 A*02:01 ELTLGEFLKL (SEQ ID NO: 2180) survivin 95-104
 A*02:01 FLTPKKLQCV (SEQ ID NO: 2181) Prostate Specific Antigen-1 (PSA-1) 141-150
 A*02:01 KLQCVLDHV (SEQ ID NO: 2182) Prostate Specific Antigen 146-154
 A*02:01 TLAPATEPA (SEQ ID NO: 2183) Mucin 79-87
 A*02:01 YLQVNSLQTV (SEQ ID NO: 2184) Telomerase Reverse Transcriptase (hTERT) 988-997
 A*02:01 SLGEQQYSV (SEQ ID NO: 2185) WT1 187-195
 A*02:01 SLEENIVIL (SEQ ID NO: 2186) RHAMM 275-283
 A*02:01 YMNGTMSQV (SEQ ID NO: 2187) Tyrosinase 368-376
 A*02:01 ILSLELMKL (SEQ ID NO: 2188) Receptor for hyaluronic acid-mediated
 motility (RHAMM) 165-173
 A*02:01 PLDFSWLSL (SEQ ID NO: 2189) Bcl-2 208-217
 A*02:01 LLGATCMFV (SEQ ID NO: 2190) CyclinD 101-109
 A*02:01 ALYVDSLFFL (SEQ ID NO: 2191) PRAME PRA 300-309
 A*02:01 GLMEEMSAL (SEQ ID NO: 2192) Human Mena protein (overexpressed in breast
 cancer)
 A*02:01 TMNGSKSPV (SEQ ID NO: 2193) hMena 502-510
 A*02:01 GUYDGREHTV (SEQ ID NO: 2194) MAGE-A4 230-239
 A*02:01 YLNDHLEPWI (SEQ ID NO: 2195) Bcl-X 173-182
 A*02:01 ALDVYNGLL (SEQ ID NO: 2196) Prostatic acid phosphatase precursor (PAP) 299-307
 A*02:01 ALFDIESKV (SEQ ID NO: 2197) PSM P2 (prostate)
 A*02:01 SLAMLDLLHV (SEQ ID NO: 2198) Mutant anaplastic lymphoma kinase 1220-1229
 A*02:01 YLNTVQPTCV (SEQ ID NO: 2199) EGF-R 1138-1147
 A*02:01 KLFGTSGQKT (SEQ ID NO: 2200) EGF-R-479 350-359
 A*02:01 RMPEAAPPV (SEQ ID NO: 2201) p53 65-73
 A*02:01 PLTSIISAV (SEQ ID NO: 2202) Receptor tyrosine-protein kinase erbB-2 728-736
 A*02:01 VLAGGFFLL (SEQ ID NO: 2203) PSMA 27-38
 A*02:01 LLHETDSAV (SEQ ID NO: 2204) PSMA/PSM-P1 4-12
 A*02:01 VMAGVGSPYV (SEQ ID NO: 2205) Receptor tyrosine-protein kinase erbB-2
 819-828
 A*02:01 VLPLTVAEV (SEQ ID NO: 2206) Mesothelin 530-538
 A*02:01 SLLFLLFSL (SEQ ID NO: 2207) Mesothelin 20-28
 A*02:01 QLFEELQEL (SEQ ID NO: 2208) Heme oxygenase-1 212-220
 A*02:01 VLDGLDVLL (SEQ ID NO: 2209) PRAME 100-108
 A*02:01 RLASFYDWPL (SEQ ID NO: 2210) BIR7 90-99
 A*02:01 LIAHNQVRQV (SEQ ID NO: 2211) HER-2/neu (85-94)
 A*02:01 ILHDGAYSL (SEQ ID NO: 2212) HER-2 434-443
 A*02:01 FVGEFFTDV (SEQ ID NO: 2213) GPC3 144-152 (overexpressed in hepatocellular
 carcinoma)
 A*02:01 LLLIWFRPV (SEQ ID NO: 2214) BKV Ltag 579-587
 A*02:01 KLQDASAEV (SEQ ID NO: 2215) HM1.24-aa 126-134
 A*02:01 SLYSFPEPEA (SEQ ID NO: 2216) PRAME
 A*02:01 SLLQHILGL (SEQ ID NO: 2217) PRAME 425-433
 A*02:01 VIFDFLHCI (SEQ ID NO: 2218) BKV Ltag 406-414
 A*02:01 VLDFAPPGA (SEQ ID NO: 2219) WT1
 A*02:01 TLPGYPPHV (SEQ ID NO: 2220) PAX-5 311-319

A*02:01	YMEHNNVYTV (SEQ ID NO: 2221)	Fibromodulin 250-259
A*02:01	YLQHNEIQEV (SEQ ID NO: 2222)	Fibromodulin 206-215
A*02:01	SLVDVMPWL (SEQ ID NO: 2223)	Cytochrome p450 1B1 239-248
A*02:01	RLMNDMTAV (SEQ ID NO: 2224)	HSP105 128-136
A*02:01	RLARLALVL (SEQ ID NO: 2225)	Trophoblast glycoprotein 17-25
A*02:01	FLTGNQLAV (SEQ ID NO: 2226)	5T4 97-105
A*02:01	LLLAGLFSL (SEQ ID NO: 2227)	Fibromodulin 7-15
A*02:01	FLGYLILGV (SEQ ID NO: 2228)	Prostatic Acid Phosphatase-3 (PAP-3)
A*02:01	SLFLGILSV (SEQ ID NO: 2229)	CD20 188-196 (B cell malignancies)
A*02:01	AVLPLLELV (SEQ ID NO: 2230)	MCL-1 139-147
A*02:01	SLSEKTVLL (SEQ ID NO: 2231)	CD59 glycoprotein precursor 106-114
A*02:01	YMCSFLFNL (SEQ ID NO: 2232)	Ewing Tumor EZH2 666-674
A*02:01	YLISGDSVP (SEQ ID NO: 2233)	CD33 65-73 (1Y2L)
A*02:01	KASEKIFYV (SEQ ID NO: 2234)	SSX2 41-49
A*02:01	FLAKLNNTV (SEQ ID NO: 2235)	HCA587 317-325
A*02:01	GLAPPQHLIRV (SEQ ID NO: 2236)	p53 187-197
A*02:01	VIMPCSWWV (SEQ ID NO: 2237)	Chondromodulin-I 319-327
A*02:01	KVVEFLAML (SEQ ID NO: 2238)	MAGE-C1 1083-1091
A*02:01	LTLGEFLKL (SEQ ID NO: 2239)	Survivin-3A 96-104
A*02:01	ALPFGFILV (SEQ ID NO: 2240)	IL13R 345-353
A*02:01	TLADFDPRV (SEQ ID NO: 2241)	EphA2
A*02:01	ALMEQQHYV (SEQ ID NO: 2242)	ITGB8 662-670
A*02:01	CLTSTVQLV (SEQ ID NO: 2243)	HER-2/neu 789-797
A*02:01	GLLGASVLGL (SEQ ID NO: 2244)	Telomerase Reverse Transcriptase (hTERT) 674-683
A*02:01	QLLDGFMITL (SEQ ID NO: 2245)	PASD1 39-48
A*02:01	YLVGNVCIL (SEQ ID NO: 2246)	PASD1 168-176
A*02:01	ALLTSRLRFI (SEQ ID NO: 2247)	Telomerase Reverse Transcriptase (hTERT) 615-624
A*02:01	RLSSCPVA (SEQ ID NO: 2248)	TGF beta receptor type-2 131-139
A*02:01	FLYDDNQRV (SEQ ID NO: 2249)	Topoisomerase II-alpha-b 828-836
A*02:01	YLIELIDRV (SEQ ID NO: 2250)	TACE 250-258
A*02:01	FLAEDALNTV (SEQ ID NO: 2251)	Epithelial Discoidin Domain Receptor 1 (EDDR1) 867-876
A*02:01	GLMKYIGEV (SEQ ID NO: 2252)	TRPM8 187-195
A*02:01	AILALLPAL (SEQ ID NO: 2253)	Prostate Stem Cell Antigen (PSCA) 105-133
A*02:01	GLQHWVPEL (SEQ ID NO: 2254)	BA46 (Lactadherin) 97-106
A*02:01	GVRGRVEEI (SEQ ID NO: 2255)	BCR-ABL
A*02:01	ITDQVPFSV (SEQ ID NO: 2256)	gp100 (pmel) 209-217
A*02:01	KLCPVQLWV (SEQ ID NO: 2257)	p53 139-147
A*02:01	KVAEELVHFL (SEQ ID NO: 2258)	MAGEA3 112-120 (alternative version)
A*02:01	SLPPPGRV (SEQ ID NO: 2259)	p53 149-157
A*02:01	YLGSYGFRL (SEQ ID NO: 2260)	p53 103-111
A*02:01	YLQLVFGIEV (SEQ ID NO: 2261)	MAGEA2 157-166
A*02:01	TLQDIVYKL (SEQ ID NO: 2262)	BMI1 74-82
A*02:01	YAILDPVSV (SEQ ID NO: 2263)	L-dopachrome tautomerase 488-496
A*02:01	AMVGAVLTA (SEQ ID NO: 2264)	Tyrosinase 482-190
A*02:01	ATVGIMIGV (SEQ ID NO: 2265)	CEACAM5 687-695
A*02:01	YVDPVITSI (SEQ ID NO: 2266)	Hepatocyte growth factor receptor 673-681
A*02:01	GVLLWEIFSL (SEQ ID NO: 2267)	VEGFR1 28-37
A*02:01	LMAQEALAFI (SEQ ID NO: 2268)	CAMEL 2-11
A*02:01	RVA(PHOSPHO-S)PTSGV (SEQ ID NO: 2269)	Insulin receptor substrate-2 1097-1105

A*02:01 RVASPTSGV (SEQ ID NO: 2270) IRS-2 1097-1105
 A*02:01 ALNVYNGLL (SEQ ID NO: 2271) ACPP 299-307
 A*02:01 ALSPVPPVV (SEQ ID NO: 2272) Bcl-2 85-93
 A*02:01 ALVCYGPPI (SEQ ID NO: 2273) FAP alpha 463-471
 A*02:01 ALWPWLLMAT (SEQ ID NO: 2274) RNF43 11-20
 A*02:01 ALYLMELTM (SEQ ID NO: 2275) CB9L2
 A*02:01 CLPSPSTPV (SEQ ID NO: 2276) BMI1 271-279
 A*02:01 ELSDSLGPV (SEQ ID NO: 2277) PASD1 695-703
 A*02:01 FLFLRNFSL (SEQ ID NO: 2278) TARP(V28L)27-35
 A*02:01 FLPSPLFFFL (SEQ ID NO: 2279) TARP(P5L) 5-13
 A*02:01 GLFKCGIAV (SEQ ID NO: 2280) FAP 639-647
 A*02:01 GLIQLVEGV (SEQ ID NO: 2281) TRAG-3 4-12
 A*02:01 ILGVLTSLV (SEQ ID NO: 2282) DLK1 309-317
 A*02:01 LLVPTCVFLV (SEQ ID NO: 2283) 691-700
 A*02:01 MLAVFLPIV (SEQ ID NO: 2284) STEAP 292-300 (293L)
 A*02:01 NLFETPVEA (SEQ ID NO: 2285) 194-202
 A*02:01 QLGEQCWTV (SEQ ID NO: 2286) PSCA 44-51 (51A)
 A*02:01 RLAEQAYI (SEQ ID NO: 2287) SART3 309-317
 A*02:01 SIDWFMVTV (SEQ ID NO: 2288) p31-39
 A*02:01 SILLRDAGLV (SEQ ID NO: 2289) TRAG-3 57-66
 A*02:01 SLFEP PPPG (SEQ ID NO: 2290) PSMA 85-93
 A*02:01 SQADALKYV (SEQ ID NO: 2291) EZH2 729-737
 A*02:01 WLSLKTLLSL (SEQ ID NO: 2292) Bcl-2 214-223
 A*02:01 YLNRHLHTWI (SEQ ID NO: 2293) BCL-2 180-189
 A*02:01 YLQWIEFSI (SEQ ID NO: 2294) Prominin1 744-752
 A*02:01 YLYQWL GAPV (SEQ ID NO: 2295) Osteocalcin 51-60
 A*02:01 KLMSSNSTDL (SEQ ID NO: 2296) HSP105 234-243
 A*02:01 RLQGISPKI (SEQ ID NO: 2297) SSX2 103-111
 A*02:01 AILALLPALL (SEQ ID NO: 2298) PSCA
 A*02:01 ALIHNNHNL (SEQ ID NO: 2299) HER2 466-474
 A*02:01 CMHLLLEAV (SEQ ID NO: 2300) MG50 624-632
 A*02:01 FLIIWQNTM (SEQ ID NO: 2301) FSP26
 A*02:01 FLPWHLRLL (SEQ ID NO: 2302) Tyrosinase 207-216
 A*02:01 FVWLHYYSV (SEQ ID NO: 2303) TRP2 185-193(L)
 A*02:01 GLFGDIYLA (SEQ ID NO: 2304) CSNK1A1 26-34
 A*02:01 GLFGDIYLAI (SEQ ID NO: 2305) CSNK1A1 26-35
 A*02:01 ILLRDAGLV (SEQ ID NO: 2306) TRAG-3L 58-66
 A*02:01 ILLVVVLGV (SEQ ID NO: 2307) Receptor tyrosine-protein kinase erbB-2 707-715
 A*02:01 ILNAMI AKI (SEQ ID NO: 2308) HAUS3 154-162
 A*02:01 KASEYLQLV (SEQ ID NO: 2309) MAGEA2 153-161
 A*02:01 KIWEELSVL (SEQ ID NO: 2310) MAGEA3 220-228
 A*02:01 KLIDRTE(S)L (SEQ ID NO: 2311) LSP1 325-333
 A*02:01 KLTGDENFTI (SEQ ID NO: 2312) Tyrosinase precursor 224-233
 A*02:01 LLCYSCKAQV (SEQ ID NO: 2313) PSCA 17-26
 A*02:01 LLLEAVPAV (SEQ ID NO: 2314) MG50 69-77
 A*02:01 LLNQLQVNL (SEQ ID NO: 2315) Mucin2 467-475
 A*02:01 LLRDAGLVKM (SEQ ID NO: 2316) TRAP 59-68
 A*02:01 LLRRYNVAKV (SEQ ID NO: 2317) SOX11 266-275
 A*02:01 LLSHGAVIEV (SEQ ID NO: 2318) Ankyrin NYBR1 158-167
 A*02:01 LVFGIELMEV (SEQ ID NO: 2319) MAGEA3 160-169
 A*02:01 LVFGIEVVEV (SEQ ID NO: 2320) MAGEA12 160-169

A*02:01 MLWGWREHV (SEQ ID NO: 2321) Mucin2 645-653
 A*02:01 PLQPEQLQV (SEQ ID NO: 2322) Receptor tyrosine-protein kinase erbB-2 437-445
 A*02:01 QLMAFNHLI (SEQ ID NO: 2323) PAX3/FKHR 135-143
 A*02:01 QLMPYGCLL (SEQ ID NO: 2324) Receptor tyrosine-protein kinase erbB-2 845-853
 A*02:01 RLGPMLMCL (SEQ ID NO: 2325) MG50 1244-1252
 A*02:01 RLTRFLSRV (SEQ ID NO: 2326) CyclinD 228-236
 A*02:01 RTF(S)PTYGL (SEQ ID NO: 2327) Desmuslin 426-434
 A*02:01 SILLRDAGL (SEQ ID NO: 2328) TRAP 57-65
 A*02:01 SLADEAEVYL (SEQ ID NO: 2329) GAS7 Neoepitope
 A*02:01 SLDDYNHLV (SEQ ID NO: 2330) L-dopachrome tautomerase 288-296
 A*02:01 SLYKFSPFPL (SEQ ID NO: 2331) O-linked N-acetylglucosamine transferase FSP06
 A*02:01 SMTR(S)PPRV (SEQ ID NO: 2332) SFRS2B 241-249
 A*02:01 TLEEITGYL (SEQ ID NO: 2333) Receptor tyrosine-protein kinase erbB-2 448-456
 A*02:01 TLHCDCEIL (SEQ ID NO: 2334) MG50 210-218
 A*02:01 VLEPPGARDV (SEQ ID NO: 2335) BIR 7 230-239
 A*02:01 VLLALLMAGL (SEQ ID NO: 2336) Prostate stem cell antigen 4-13
 A*02:01 VLSVNVDPV (SEQ ID NO: 2337) MG50 625-633
 A*02:01 VLVKSPNHV (SEQ ID NO: 2338) Receptor tyrosine-protein kinase erbB-4 890-898
 A*02:01 VMIG(S)PKKV (SEQ ID NO: 2339) Tensin3 1558-1566
 A*02:01 VVLGVVFGI (SEQ ID NO: 2340) Receptor tyrosine-protein kinase erbB-2 743-751
 A*02:01 WLPKILGEV (SEQ ID NO: 2341) MG50 1051-1059
 A*02:01 WLQYFPNPV (SEQ ID NO: 2342) Cytochrome P450 246-254
 A*02:01 YLLDLSTNHL (SEQ ID NO: 2343) Fibromodulin 7-15
 A*02:01 YLWWVNNQSL (SEQ ID NO: 2344) CEA 176-185
 A*02:01 ALGGHPLLGV (SEQ ID NO: 2345) Dickkopf-related protein 1 20-29
 A*02:01 ALLAGLVSL (SEQ ID NO: 2346) FGFR4 676-684
 A*02:01 ALLTYMIAHI (SEQ ID NO: 2347) Thymidylate synthase 231-240
 A*02:01 ALMDKSLHV (SEQ ID NO: 2348) MART-1 56-64
 A*02:01 ALPPPLMLL (SEQ ID NO: 2349) Heparanase 8-16
 A*02:01 ALSVMGVYV (SEQ ID NO: 2350) MAGEA9 223-231
 A*02:01 ALVEFEDVL (SEQ ID NO: 2351) hnRNP L 140-148
 A*02:01 ALWPWLLMA (SEQ ID NO: 2352) RNF43 11-19
 A*02:01 AMLGTHTMEV (SEQ ID NO: 2353) Melanocyte-specific secreted glycoprotein
 184-193
 A*02:01 AVIGALLAV (SEQ ID NO: 2354) Melanocyte-specific secreted glycoprotein 20-28
 A*02:01 CLYGNVEKV (SEQ ID NO: 2355) hnRNP L 404-412
 A*02:01 DLIFGLNAL (SEQ ID NO: 2356) Heparanase 185-193
 A*02:01 ELFQDLSQL (SEQ ID NO: 2357) ETV5 54-53
 A*02:01 FAWERVRL (SEQ ID NO: 2358) Cyclin-dependent kinase inhibitor 1 97-105
 A*02:01 FIASNGVKLV (SEQ ID NO: 2359) ACTN4 118-127 (K5N)
 A*02:01 FLALIICNA (SEQ ID NO: 2360) Tubulin beta 4 283-291
 A*02:01 FLDEFMEGV (SEQ ID NO: 2361) Malic enzyme 224-232
 A*02:01 RMFPNAPYL (SEQ ID NO: 2362) WT-1 126-134 (Wilms tumor)
 A*02:01 RLNMFTPYI (SEQ ID NO: 2363) Chlamydia trachomatis MOMP 258-266
 A*02:01 NMFTPYIGV (SEQ ID NO: 2364) MOMP precursor 283-291
 A*02:01 NLVPMVATV (SEQ ID NO: 2365) HCMV pp65 495-504
 A*02:01 VLEETSVML (SEQ ID NO: 2366) HCMV IE1 316-324 (UL123)
 A*02:01 VLAELVKQI (SEQ ID NO: 2367) HCMV IE1 81-89
 A*02:01 MLNIPSINV (SEQ ID NO: 2368) pp65 120-128
 A*02:01 LLLDRLNQL (SEQ ID NO: 2369) SARS-CoV Nucleocapsid protein 223-231 (conserved
 in SARS-CoV-2)

A*02:01	FIAGLIAIV (SEQ ID NO: 2370)	SARS-CoV-2 Spike glycoprotein 1220-1228 (confirmed epitope)
A*02:01	ALNTLVKQL (SEQ ID NO: 2371)	SARS-CoV Spike glycoprotein precursor 940-948 (conserved in SARS-CoV-2)
A*02:01	LITGRLQSL (SEQ ID NO: 2372)	SARS-CoV-2 Spike glycoprotein 996-1004 (confirmed epitope)
A*02:01	NLNESLIDL (SEQ ID NO: 2373)	SARS-CoV Spike glycoprotein precursor 1174-1182 (conserved in SARS-Cov-2)
A*02:01	VLNDILSRL (SEQ ID NO: 2374)	SARS-CoV Spike glycoprotein precursor 958-966 (conserved in SARS-Cov-2)
A*02:01	YLQPRTFLL (SEQ ID NO: 2375)	SARS-Cov-2 Spike glycoprotein 269-277 (confirmed epitope)
A*02:01	LLYDANYFL (SEQ ID NO: 2376)	SARS-CoV-2 ORF3a 139-147 (confirmed epitope)
A*02:01	RLQSLQTYV (SEQ ID NO: 2377)	SARS-CoV-2 Spike glycoprotein 1000-1008 (confirmed subdominant epitope)
A*02:01	KLWAQCVQL (SEQ ID NO: 2378)	SARS-CoV-2 ORF1ab 3886-3894 (confirmed epitope)
A*02:01	TLYAVATTI (SEQ ID NO: 2379)	Dengue NS4b 40-48
A*02:01	KLAEIFKL (SEQ ID NO: 2380)	Dengue NS5 563-571
A*02:01	ILIRTGLLVI (SEQ ID NO: 2381)	Dengue NS2b 97-106
A*02:01	AIKRGLRTL (SEQ ID NO: 2382)	Dengue NS3 112-120
A*02:01	LLLGLMILL (SEQ ID NO: 2383)	Dengue NS4a 56-64
A*02:01	VLLLVTHTYA (SEQ ID NO: 2384)	Dengue NS4b 111-119
A*02:01	GLCTLVAML (SEQ ID NO: 2385)	EBV BMLF-1 259-267
A*02:01	CLGGLTMMV (SEQ ID NO: 2386)	EBV LMP-2 426-434
A*02:01	YLLEMLWRL (SEQ ID NO: 2387)	EBV LMP-1 125-133
A*02:01	YLQQNWWTL (SEQ ID NO: 2388)	EBV LMP1 159-167
A*02:01	YVLDHLIVV (SEQ ID NO: 2389)	EBV BRLF1 109-117
A*02:01	FLYALALLL (SEQ ID NO: 2390)	EBV LMP-2 356-364
A*02:01	TLDYKPLSV (SEQ ID NO: 2391)	EBV BMRF1 208-216
A*02:01	LLDFVRFMGV (SEQ ID NO: 2392)	EBV EBNA-3C 284-293
A*02:01	FLDKGTYTL (SEQ ID NO: 2393)	EBV BALF-4 276-284
A*02:01	FLPSDFFPSV (SEQ ID NO: 2394)	HBV core antigen 18-27
A*02:01	FLLTRILTI (SEQ ID NO: 2395)	HBV envelope 183-191
A*02:01	GLSPTVWLSV (SEQ ID NO: 2396)	HBV surface antigen 185-194
A*02:01	WLSLLVPFV (SEQ ID NO: 2397)	HBV surface antigen 172-181
A*02:01	FLLSLGIHL (SEQ ID NO: 2398)	HBV polymerase 573-581
A*02:01	FLPSDFFPSI (SEQ ID NO: 2399)	HBV core 18-27 (subtype ADR4)
A*02:01	VLHKRTLGL (SEQ ID NO: 2400)	HBV X 92-100
A*02:01	GLSRYVARL (SEQ ID NO: 2401)	HBV Pol 455-463
A*02:01	YMDDVVLGA (SEQ ID NO: 2402)	HBV Polymerase 548-556
A*02:01	KLHLYSHPI (SEQ ID NO: 2403)	HBV Pol 502-510
A*02:01	ELMTLATWV (SEQ ID NO: 2404)	HBV core protein 64-72
A*02:01	DLMGYIPAV (SEQ ID NO: 2405)	HCV core 132-140
A*02:01	CINGVCWTV (SEQ ID NO: 2406)	HCV NS3 1073-1081
A*02:01	YLLPRRGPR (SEQ ID NO: 2407)	HCV core 35-44
A*02:01	VLSDFKTWL (SEQ ID NO: 2408)	HCV NS5a 1987-1995
A*02:01	ALYDVVTKL (SEQ ID NO: 2409)	HCV NS5b 2594-2602
A*02:01	KLVALGINAV (SEQ ID NO: 2410)	HCV NS3 1406-1415
A*02:01	LLFNILGGWV (SEQ ID NO: 2411)	HCV NS4b 1807-1816
A*02:01	KLSGLGINAV (SEQ ID NO: 2412)	HCV NS3 1406-1415
A*02:01	DLMGYIPLV (SEQ ID NO: 2413)	HCV core 132-140

A*02:01 CVNGVCWTV (SEQ ID NO: 2414) HCV NS3 1073-1081
 A*02:01 GLQDCTMLV (SEQ ID NO: 2415) HCV NS5B 2727-2735
 A*02:01 SLYNTVATL (SEQ ID NO: 2416) HIV-1 gag p17 76-84
 A*02:01 ILKEPVHGV (SEQ ID NO: 2417) HIV-1 RT 476-484
 A*02:01 TLNAWVKV (SEQ ID NO: 2418) HIV-1 gag p24 19-27
 A*02:01 KLTPLCVTL (SEQ ID NO: 2419) HIV-1 env gp120 90-98
 A*02:01 GLADQLIHL (SEQ ID NO: 2420) HIV-1 vif 101-109
 A*02:01 LTFGWCFKL (SEQ ID NO: 2421) HIV-1 nef 137-145
 A*02:01 FLGKIWPS (SEQ ID NO: 2422) Gag 433-440
 A*02:01 ALVEMGHHA (SEQ ID NO: 2423) HIV Vpu 66-74
 A*02:01 RTLNAWVKV (SEQ ID NO: 2424) HIV gag 150-158
 A*02:01 NVWATHACV (SEQ ID NO: 2425) HIV env gp 67-7
 A*02:01 SLLNATAIAV (SEQ ID NO: 2426) HIV env 816-825
 A*02:01 SLFNTVATL (SEQ ID NO: 2427) HIV gag 77-85
 A*02:01 SLVKHHMYI (SEQ ID NO: 2428) HIV vif 23-31
 A*02:01 VIYHYVDDL (SEQ ID NO: 2429) HIV pol
 A*02:01 YMLDLQPETT (SEQ ID NO: 2430) HPV 16 E7 11-20
 A*02:01 KLPQLCTEL (SEQ ID NO: 2431) HPV 16 E6 18-26
 A*02:01 YMLDLQPET (SEQ ID NO: 2432) HPV 16 E7 11-19
 A*02:01 MLDLQPETT (SEQ ID NO: 2433) HPV 16 E7 12-20
 A*02:01 VLMIKALEL (SEQ ID NO: 2434) Non muscle Myosin-9 741-749
 A*02:01 QLFNHTMFI (SEQ ID NO: 2435) Non-muscle Myosin 478-486
 A*02:01 QMARLAWEA (SEQ ID NO: 2436) 1116-1124
 A*02:01 LLFGYPVYV (SEQ ID NO: 2437) Human T-cell lymphotropic virus-1 (HTLV-1) tax 11-19
 A*02:01 AVLDGLLSL (SEQ ID NO: 2438) HTLV bZIP factor 42-50
 A*02:01 GLLSLEEL (SEQ ID NO: 2439) bZIP factor 26-34
 A*02:01 GILGFVFTL (SEQ ID NO: 2440) Influenza A MP 58-66
 A*02:01 ILGFVFTLTV (SEQ ID NO: 2441) Influenza A MP 59-68
 A*02:01 KLGEFYNQMM (SEQ ID NO: 2442) Flu BNP 85-94 (Influenza B)
 A*02:01 SITEVECFL (SEQ ID NO: 2443) VP1 36-44
 A*02:01 ILMWEAVTL (SEQ ID NO: 2444) VP1 100-108
 A*02:01 ALPHIIDDEV (SEQ ID NO: 2445) LCMV envelope gp 10-18
 A*02:01 YLVSIFLHL (SEQ ID NO: 2446) LCMV envelope gp 447-455
 A*02:01 SLNQTVHSL (SEQ ID NO: 2447) NP 69-77
 A*02:01 YLNKIQNSL (SEQ ID NO: 2448) Plasmodium falciparum CSP 334-342
 A*02:01 FIDSYICQV (SEQ ID NO: 2449) miHAg H-Y (human SMCY) 311-319
 A*02:01 YIGEVLVSV (SEQ ID NO: 2450) HA-2
 A*02:01 VLHDDLLEA (SEQ ID NO: 2451) Minor Histocompatibility Antigen HA-1 137-145
 A*02:01 RTLDKVLEV (SEQ ID NO: 2452) miHAg HA-8
 A*02:01 NEGATIVE (SEQ ID NO: 2453) Negative Control
 A*02:01 TMFPHIIVDV (SEQ ID NO: 2454) Norovirus VP1 139-148
 A*02:01 LLDVPTAAV (SEQ ID NO: 2455) Interferon gamma inducible protein (GILT) 30 27-35
 A*02:01 RILGAVAKV (SEQ ID NO: 2456) Vinculin 822-830
 A*02:01 LMWYELSKI (SEQ ID NO: 2457) KSHVF-8 gB.492-500
 A*02:01 ILEDIVLTL (SEQ ID NO: 2458) Streptococcus pyogenes Cas9 615-623
 A*02:01 KMLKEMGEV (SEQ ID NO: 2459) RSV NP 137-145
 A*02:01 KLIANNTRV (SEQ ID NO: 2460) Mycobacterium bovis antigen 85-A 200-208
 A*02:01 GLPVEYLQV (SEQ ID NO: 2461) Mycobacterium bovis antigen 85-A 6-14
 A*02:01 GILTVSVAV (SEQ ID NO: 2462) 16 kDa
 A*02:01 AMASTEGNV (SEQ ID NO: 2463) ESAT-6

A*02:01 VLTDGNPPEV (SEQ ID NO: 2464) 19 kDa
 A*02:01 KVDDTFYYV (SEQ ID NO: 2465) Vaccinia virus Host range protein 2 74-82
 A*02:01 ILDDNLYKV (SEQ ID NO: 2466) Vaccinia virus Copenhagen Protein G5 18-26
 A*02:01 ALWALPHAA (SEQ ID NO: 2467) IE62 593-601
 A*02:01 RLDDDGNFQL (SEQ ID NO: 2468) West Nile Virus NY-99 polyprotein precursor
 (1452-1461)
 A*02:01 ATWAENIQV (SEQ ID NO: 2469) West Nile virus NY-99 polyprotein precursor 3390-
 3398
 A*02:01 YTMDGEYRL (SEQ ID NO: 2470) West Nile virus NY-99 polyprotein precursor 2023-
 2031
 A*02:01 SVGGVFTSV (SEQ ID NO: 2471) WNV envelope gp 430-438
 A*02:01 SLFGQRIEV (SEQ ID NO: 2472) WNV nonstructural protein 4B 15-23
 A*02:01 LLWNGPMAV (SEQ ID NO: 2473) NS4B 214-222
 A*03:01 KQSSKALQR (SEQ ID NO: 2474) bcr-abl 210 kD fusion protein 21-29
 A*03:01 ALLAVGATK (SEQ ID NO: 2475) gp100 (pmel17) 17-25
 A*03:01 ATGFKQSSK (SEQ ID NO: 2476) bcr-abl 210 kD fusion protein 259-269
 A*03:01 RISTFKNWPK (SEQ ID NO: 2477) Survivin-3A 18-27 (27K)
 A*03:01 RLGLQVRKNK (SEQ ID NO: 2478) RhoC 176-185 (177L)
 A*03:01 RLLFFAPTR (SEQ ID NO: 2479) Mcl-1 95-103
 A*03:01 QVLKKIAQK (SEQ ID NO: 2480) HMOX1 145-153
 A*03:01 RIAAWMATY (SEQ ID NO: 2481) 165-173
 A*03:01 KLGGALQAK (SEQ ID NO: 2482) HCMV IE1 184-192
 A*03:01 KTFPPTPEK (SEQ ID NO: 2483) SARS-CoV-2 Nucleocapsid protein 362-370
 (confirmed epitope)
 A*03:01 ELERAADV K (SEQ ID NO: 2484) Dengue NS2b 52-60
 A*03:01 RVSTVQQLTK (SEQ ID NO: 2485) Dengue C 22-31
 A*03:01 RIEPSWADV K (SEQ ID NO: 2486) Dengue NS3 64-74
 A*03:01 RVIDPRRCMK (SEQ ID NO: 2487) Dengue NS3 422-431
 A*03:01 KITAEWLWK (SEQ ID NO: 2488) Dengue NS5 375-383
 A*03:01 RLRAEAQVK (SEQ ID NO: 2489) EBV EBNA 3A 603-611
 A*03:01 RVRAYTYSK (SEQ ID NO: 2490) EBV BRLF1
 A*03:01 RVCEKMALY (SEQ ID NO: 2491) HCV NS5B 2588-2596
 A*03:01 QVPLRPMTYK (SEQ ID NO: 2492) HIV-1 nef 73-82
 A*03:01 RLRPGGKKK (SEQ ID NO: 2493) HIV-1 gag p17 19-27
 A*03:01 AIFQSSMTK (SEQ ID NO: 2494) HIV pol 325-333
 A*03:01 KLCLRFLSK (SEQ ID NO: 2495) HPV 33 E6 64-72
 A*03:01 ILRGSAVHK (SEQ ID NO: 2496) Influenza A (PR8) NP 265-274
 A*11:01 KTFPPTPEK (SEQ ID NO: 2483) SARS-CoV-2 Nucleocapsid protein 362-370
 (confirmed epitope)
 A*11:01 GTSGSPIINR (SEQ ID NO: 2497) Dengue NS3 serotype 3&4 133-142
 A*11:01 GTSGSPIIDK (SEQ ID NO: 2498) Dengue NS3 133-142
 A*11:01 GTSGSPIVNR (SEQ ID NO: 2499) NS3 serotype 1 133-142
 A*11:01 GTSGSPIVDR (SEQ ID NO: 2500) Dengue NS3 serotype 2 133-142
 A*11:01 GTSGSPIADK (SEQ ID NO: 2501) Dengue NS3 133-142
 A*11:01 RVSTVQQLTK (SEQ ID NO: 2485) Dengue C 22-31
 A*11:01 RIEPSWADV K (SEQ ID NO: 2486) Dengue NS3 64-74
 A*11:01 RVIDPRRCMK (SEQ ID NO: 2487) Dengue NS3 422-431
 A*11:01 KITAEWLWK (SEQ ID NO: 2488) Dengue NS5 375-383
 A*11:01 IVTDFSVIK (SEQ ID NO: 2502) EBV EBNA-4 416-424
 A*11:01 SSCSSCPLSK (SEQ ID NO: 2503) EBV LMP-2 340-349
 A*11:01 ATIGTAMYK (SEQ ID NO: 2504) EBV BRLF1 134-142

A*11:01 AVFDRKSDAK (SEQ ID NO: 2505) EBNA3B 399-408
 A*11:01 YVNVNMGLK (SEQ ID NO: 2506) HBV core antigen 88-96
 A*11:01 YVNTNMGLK (SEQ ID NO: 2507) HBV core 88-96
 A*11:01 STLPETTVVRR (SEQ ID NO: 2508) HBV core 141-151
 A*11:01 AVDLSHFLK (SEQ ID NO: 2509) HIV nef 84-92
 A*11:01 ACQGVGGPGHK (SEQ ID NO: 2510) HIV gag p24
 A*11:01 NTLEQTVKK (SEQ ID NO: 2511) HPV 33 E6 86-94
 A*11:01 SIIPSGPLK (SEQ ID NO: 2512) Influenza A MP 13-21
 A*11:01 RMVLASTTAK (SEQ ID NO: 2513) Influenza A MP1 178-187
 A*11:01 KSMREEYRK (SEQ ID NO: 2514) Influenza A MP2 70-78
 A*24:02 TYFSLNNKF (SEQ ID NO: 2515) Adenovirus 5 Hexon 37-45
 A*24:02 TYACFVSNL (SEQ ID NO: 2516) Carcinogenic Embryonic Antigen (CEA) 652-660
 A*24:02 AFLPWHRLF (SEQ ID NO: 2517) Tyrosinase 188-196
 A*24:02 IMPKAGLLI (SEQ ID NO: 2518) MAGE-A3
 A*24:02 VYFFLPDHL (SEQ ID NO: 2519) gp100-intron 4 (170-178)
 A*24:02 EYLQLVFGI (SEQ ID NO: 2520) MAGEA2 156-164
 A*24:02 TYLPTNASL (SEQ ID NO: 2521) HER-2/neu 63-71
 A*24:02 VYGFVRACL (SEQ ID NO: 2522) Telomerase reverse transcriptase (hTERT) 461-469
 A*24:02 TFPDLESEF (SEQ ID NO: 2523) MAGEA3 97-105
 A*24:02 DYLLQYVLQI (SEQ ID NO: 2524) MiHA ACC1 15-23
 A*24:02 RYCNLEGPI (SEQ ID NO: 2525) Lymphocyte antigen 6 complex locus K (LY6K) 177-186
 A*24:02 AYACNTSTL (SEQ ID NO: 2526) Survivin 80-88
 A*24:02 CYASGWGSI (SEQ ID NO: 2527) Prostate Specific Antigen-1 153-161
 A*24:02 DYLNWGSRF (SEQ ID NO: 2528) CDH3 807-816
 A*24:02 EYCPGGNLF (SEQ ID NO: 2529) MELK 87-95 (93N)
 A*24:02 EYYELFVNI (SEQ ID NO: 2530) DEP DC1 294-302
 A*24:02 GYCTQIGIF (SEQ ID NO: 2531) HENMT1 221-229
 A*24:02 IYTWIEDHF (SEQ ID NO: 2532) FOXM1 262-270
 A*24:02 NYQPVWLCL (SEQ ID NO: 2533) RNF43 721-729 (722Y)
 A*24:02 RYNAQCQETI (SEQ ID NO: 2534) Midkine 110-119
 A*24:02 EYRALQLHL (SEQ ID NO: 2535) CA9 219-227
 A*24:02 SYRNEIAYL (SEQ ID NO: 2536) TTK protein kinase 551-559
 A*24:02 VYLRVRPLL (SEQ ID NO: 2537) KIF20A 67-75
 A*24:02 VYYNWQYLL (SEQ ID NO: 2538) IL13r 146-154
 A*24:02 VYALPLKML (SEQ ID NO: 2539) HCMV pp65 113-121
 A*24:02 QYDPVAALF (SEQ ID NO: 2540) HCMV pp65 341-349
 A*24:02 AYAQKIFKI (SEQ ID NO: 2541) CMV IE-1 248-256
 A*24:02 QYSDRRWCF (SEQ ID NO: 2542) Dengue NS3 557-565 (Singapore/S275/1990)
 A*24:02 TYGPVFMCL (SEQ ID NO: 2543) EBV LMP-2 419-427
 A*24:02 PYLFWLAAI (SEQ ID NO: 2544) EBV LMP2 131-139
 A*24:02 TYGPVFMSL (SEQ ID NO: 2545) EBV LMP2 419-427
 A*24:02 EYLVSFVW (SEQ ID NO: 2546) HBV core 117-125
 A*24:02 KYTSFPWLL (SEQ ID NO: 2547) HBV polymerase 756-764
 A*24:02 FFPSIRDLL (SEQ ID NO: 2548) HBV core protein 23-31
 A*24:02 AYSQQTRGL (SEQ ID NO: 2549) HCV NS3 1031-1039
 A*24:02 RYPLTFGWY (SEQ ID NO: 2550) HIV-1 Nef 134-143
 A*24:02 RYLKDQQLL (SEQ ID NO: 2551) HIV-1 gag gp41 67-75
 A*24:02 RYLRDQQLL (SEQ ID NO: 2552) HIV env
 A*24:02 RYPLTFGWCF (SEQ ID NO: 2553) HIV nef 143-152
 A*24:02 RYPLTFGW (SEQ ID NO: 2554) HIV nef

A*24:02 VYDFAFRDL (SEQ ID NO: 2555) HPV16 E6
 A*24:02 SFHSLHLLF (SEQ ID NO: 2556) HTLV Tax 301-309
 A*29:02 KEKYIDQEEL (SEQ ID NO: 2557) HSP90 alpha 280-288 (Pathologic Conditions)
 A*29:02 LYNTVATLY (SEQ ID NO: 2558) HIV gag 79-86
 A*29:02 SFDPIPIHY (SEQ ID NO: 2559) HIV env 216-224
 A*29:02 SFNCRGEFFY (SEQ ID NO: 2560) HIV env 382-391
 A*68:01 TVSGNLTIR (SEQ ID NO: 2561) NY-ESO-1 127-136
 B*07:02 VPQYGYLTL (SEQ ID NO: 2562) AAV2 372-380
 B*07:02 KPYSGTAYNSL (SEQ ID NO: 2563) Adenovirus Hexon 114-124
 B*07:02 KPYSGTAYNAL (SEQ ID NO: 2564) Adenovirus Hexon 114-124
 B*07:02 LPLMRKAYL (SEQ ID NO: 2565) LT antigen 27-35
 B*07:02 LPWHRLFLL (SEQ ID NO: 2566) Tyrosinase 208-216
 B*07:02 EPR(PHOSPHO-S)PSHSM (SEQ ID NO: 2567) Insulin receptor substrate 2
 B*07:02 TPNQRQNVC (SEQ ID NO: 2568) P2X5
 B*07:02 APRGVMAV (SEQ ID NO: 2569) LAGE-1 46-54
 B*07:02 LPVSPRLQL (SEQ ID NO: 2570) CEACAM 185-193
 B*07:02 TPRVTGGGAM (SEQ ID NO: 2571) HCMV pp65 417-426
 B*07:02 RPHERNGFTVL (SEQ ID NO: 2572) HCMV pp65 265-275
 B*07:02 SPRWYFYLL (SEQ ID NO: 2573) SARS-CoV-2 Nucleocapsid protein 105-113
 (confirmed epitope)
 B*07:02 APTRVVAEM (SEQ ID NO: 2574) Dengue NS3 serotype 2 222-231
 B*07:02 RPPIFIRRL (SEQ ID NO: 2575) EBV EBNA-3A 247-255
 B*07:02 RPQGGSRPEFVKL (SEQ ID NO: 2576) EBV BMRF1 116-128
 B*07:02 QPRAPIRPI (SEQ ID NO: 2577) EBV EBNA-3C 881-889
 B*07:02 LPSDFFPSV (SEQ ID NO: 2578) HBV core 19-27
 B*07:02 GPRLGVRAT (SEQ ID NO: 2579) HCV core 41-49
 B*07:02 DPRRRSRNL (SEQ ID NO: 2580) HCV core 111-119
 B*07:02 IPRRIRQGL (SEQ ID NO: 2581) HIV-1 env gp120 848-856
 B*07:02 TPGPGVRYPL (SEQ ID NO: 2582) HIV-1 nef 128-137
 B*07:02 GPGHKARVL (SEQ ID NO: 2583) HIV gag p24 223-231
 B*07:02 KPTLKEYVL (SEQ ID NO: 2584) HPV 33 E7 5-13
 B*07:02 LPVSCPEDL (SEQ ID NO: 2585) bZIP factor 10-18
 B*07:02 QPEWFRNVL (SEQ ID NO: 2586) Influenza A PB1 329-337
 B*07:02 SPIVPSFDM (SEQ ID NO: 2587) Influenza A NP 473-481
 B*07:02 SPSVDKARAEL (SEQ ID NO: 2588) MiHA Ag SMCY 1041-1051
 B*08:01 GFKQSSKAL (SEQ ID NO: 2589) bcr-abl 210 kD fusion protein 19-27
 B*08:01 ELRRKMMYM (SEQ ID NO: 2590) IE1 199-207
 B*08:01 ELKRKMIYM (SEQ ID NO: 2591) CMV IE-1 199-207 (R/L Position 3, M/I Position 7)
 B*08:01 RIKQKGIL (SEQ ID NO: 2592) Dengue NS3 25-32
 B*08:01 LEKTKKDL (SEQ ID NO: 2593) Dengue NS4a 6-13
 B*08:01 FLRGRAYGL (SEQ ID NO: 2594) EBV EBNA-3A 193-201
 B*08:01 RAKFKQLL (SEQ ID NO: 2595) EBV BZLF-1 190-197
 B*08:01 QAKWRLQTL (SEQ ID NO: 2596) EBV EBNA3A 158-166
 B*08:01 GLKILQLL (SEQ ID NO: 2597) HBV external core Ag
 B*08:01 HSKKKCDEL (SEQ ID NO: 2598) HCV NS3 1395-1403
 B*08:01 FLKEKGGL (SEQ ID NO: 2599) HIV-1 nef 90-97
 B*08:01 GEIYKRWII (SEQ ID NO: 2600) HIV-1 gag p24 261-269
 B*08:01 EIIYKRWII (SEQ ID NO: 2601) HIV p24 gag 128-135
 B*08:01 YLKDQQLL (SEQ ID NO: 2602) Env 586-593
 B*15:01 CLIPTAMAF (SEQ ID NO: 2603) Dengue C 107-115
 B*15:01 RLRPGGKKKY (SEQ ID NO: 2604) HIV-1 p17 20-29

B*27:05	GRFGLATEK (SEQ ID NO: 2605)	BRAF 594-601 (600E)
B*27:05	GRFGLATVK (SEQ ID NO: 2606)	BRAF 594-601 (600V)
B*27:05	RMFPNAPYL (SEQ ID NO: 2362)	WT-1 126-134 (Wilms tumor)
B*27:05	ARKLLLDNL (SEQ ID NO: 2607)	PqqC-like protein 70-78
B*27:05	NRAKQVIKL (SEQ ID NO: 2608)	Probable ATP-dependent Clp protease ATP-binding subunit 7-15
B*27:05	QRNAPRITF (SEQ ID NO: 2609)	SARS-CoV-2 Nucleocapsid protein 9-17 (confirmed epitope)
B*27:05	ARMILMTHF (SEQ ID NO: 2610)	HCV NS5B 2841-2849
B*27:05	KRWIILGLNK (SEQ ID NO: 2611)	HIV-1 gag p24 265-274
B*27:05	KRWIIMGLNK (SEQ ID NO: 2612)	HIV-1 Gag p24 263-272
B*27:05	GRAFVTIGK (SEQ ID NO: 2613)	HIV-1 gp100 103-111
B*27:05	SRYWAIRTR (SEQ ID NO: 2614)	Influenza A NP 383-391
B*35:01	IPYLDGTFY (SEQ ID NO: 2615)	Adenovirus Hexon
B*35:01	MPFATPMEA (SEQ ID NO: 2616)	NY-ESO-1 94-102
B*35:01	IPSINVHHY (SEQ ID NO: 2617)	HCMV pp65 123-131
B*35:01	TPEGIPTL (SEQ ID NO: 2618)	Dengue NS3 500-508
B*35:01	YPLHEQHGM (SEQ ID NO: 2619)	EBV EBNA-3A 458-466
B*35:01	EPLPQGQLTAY (SEQ ID NO: 2620)	EBV BZLF-1 54-64
B*35:01	HPVAEADYFEY (SEQ ID NO: 2621)	EBV EBNA-1 407-417(4A)
B*35:01	HPNIEEVAL (SEQ ID NO: 2622)	HCV NS3 1359-1367
B*35:01	CPNSSIVY (SEQ ID NO: 2623)	HCV E1 207-214
B*35:01	NPDIVYQY (SEQ ID NO: 2624)	HIV-1 RT 330-338
B*35:01	NPDIVYQY (SEQ ID NO: 2625)	HIV-1 HIV-1 RT 328-336
B*35:01	VPLDEDFRKY (SEQ ID NO: 2626)	HIV-1 HIV-1 RT 273-282
B*40:01	MEVTPSGTWL (SEQ ID NO: 2627)	SARS-CoV-2 Nucleocapsid protein 322-330 (confirmed epitope)
B*40:01	GEARKTFVEL (SEQ ID NO: 2628)	Dengue NS3 528-537
B*40:01	IEDPPFNSL (SEQ ID NO: 2629)	EBV LMP2 200-208
B*40:01	REISVPAEIL (SEQ ID NO: 2630)	HCV NS5a 2266-2275
B*40:01	KEKGGLEGL (SEQ ID NO: 2631)	HIV-1 Nef 92-100
C*06:02	TRATKMQVI (SEQ ID NO: 2632)	pp65 211-219
C*06:02	QIKVRVDM (SEQ ID NO: 2633)	IE1 88-95
C*06:02	TRRFLPQIL (SEQ ID NO: 2634)	NS3 205-213
C*07:02	CRVLCCYVL (SEQ ID NO: 2635)	IE1 309-317
E*01:01	VMAPRTLIL (SEQ ID NO: 2636)	HLA-C leader sequence peptide
E*01:01	VMAPRTLVL (SEQ ID NO: 2637)	HLA-A leader sequence peptide
DRB1*01:01	DSVTPMILKAQKGGNL (SEQ ID NO: 2641)	Dog dander Can f 1 33-48
DRB1*01:01	KCIEWEKAQHGA (SEQ ID NO: 2642)	Mugwort pollen Art v 1 25-36
DRB1*01:01	LPVVLENARILKNCVDAK (SEQ ID NO: 2643)	Cat dander Fel d 1 53-70
DRB1*01:01	LRQMRTVTPIRMQGG (SEQ ID NO: 2644)	House dust mite Der p1 96-110
DRB1*01:01	VAAAPQVKYAVFEAALTAKI (SEQ ID NO: 2645)	Timothy grass Phl p 5 p26
DRB1*01:01	YESYKFIPALEAAVK (SEQ ID NO: 2646)	Grass pollen allergen Phl p 5 196-210
DRB1*01:01	ETLLRAVESYLLAHS (SEQ ID NO: 2647)	Birch pollen allergen Bet v 1 142-156
DRB1*01:01	ACYEFLWGPRLVETS (SEQ ID NO: 2648)	MAGE-A3 267-282
DRB1*01:01	LKEFTVSGNILTIRL (SEQ ID NO: 2649)	NY-ESO-1 123-137
DRB1*01:01	LLEFYLAMPFATPME (SEQ ID NO: 2650)	NY-ESO-1 87-101
DRB1*01:01	KVPIKWMALLESILRRRF (SEQ ID NO: 2651)	HER2 883-899
DRB1*01:01	LPLKMLNIPSINVH (SEQ ID NO: 2652)	CMV pp65 116-129

DRB1*01:01	GAALQIPFAMQMAYRF (SEQ ID NO: 2653)	SARS-CoV S protein (873-888)
(conserved in SARS-CoV-2)		
DRB1*01:01	MAYRFNGIGVTQNVLY (SEQ ID NO: 2654)	SARS-CoV S protein (884-899)
(conserved in SARS-CoV-2)		
DRB1*01:01	QLIRAAEIRASANLAATK (SEQ ID NO: 2655)	SARS-CoV S protein (993-1010)
(conserved in SARS-CoV-2)		
DRB1*01:01	TVFYNIIPMPL (SEQ ID NO: 2656)	EBV EBNA2 280-290
DRB1*01:01	TSLYNLRRGTALA (SEQ ID NO: 2657)	EBV EBNA1 515-527
DRB1*01:01	QAGFLLRILTIPQS (SEQ ID NO: 2658)	HBV envelope 179-194
DRB1*01:01	PPAYRPPNAPILSTL (SEQ ID NO: 2659)	HBV core 158-172
DRB1*01:01	LCWGELMTLATWVGVN (SEQ ID NO: 2660)	HBV core 60-75
DRB1*01:01	TLLFNILGGWVAA (SEQ ID NO: 2661)	HCV polyprotein 1806?1818, NS4b
DRB1*01:01	DYVDRFYKTLRAE (SEQ ID NO: 2662)	HIV-1 gag 295-307
DRB1*01:01	KRWIILGLNKIVRMYSPTSI (SEQ ID NO: 2663)	HIV-1 gag 263-282
DRB1*01:01	FRDYVDRFYKTLRAEQASQE (SEQ ID NO: 2664)	HIV-1 gag 293-312
DRB1*01:01	SGPLKAEIAQRLEDV (SEQ ID NO: 2665)	Influenza A MP 17-31
DRB1*01:01	PKYVKQNTLKLAT (SEQ ID NO: 2666)	Influenza A HA 307-319
DRB1*01:01	EYLNKIQNSLSTEWSPCSVT (SEQ ID NO: 2667)	CSP 326-345
DRB1*01:01	PVSKMRMATPLLMQA (SEQ ID NO: 2668)	CLIP 87-101
DRB1*01:01	QEIMMQHTYPISA (SEQ ID NO: 2669)	TT 257-269
DRB1*01:01	GEEYLILSARDVLAV (SEQ ID NO: 2670)	Mtb 10 kDa chaperonin GroES
DRB1*03:01	HTYTIDWTKDAVTWS (SEQ ID NO: 2671)	Aspergillus fumigatus Crf1/p41 171-185
DRB1*03:01	VYYLTRDPTTPLARAA (SEQ ID NO: 2672)	HCV polyprotein 2800-2815, NS5b
DRB1*03:01	PIVQLQGDSNCLKCFR (SEQ ID NO: 2673)	HPV E2 285-300
DRB1*03:01	MEAIAKRLDACQDQLELYE (SEQ ID NO: 2674)	HPV E2 1-20
DRB1*03:01	RQIFGDYKTTIC (SEQ ID NO: 2675)	PLP 98-109
DRB1*03:01	PVSKMRMATPLLMQA (SEQ ID NO: 2668)	CLIP 87-101
DRB1*03:01	MAKTIAYDEEARR (SEQ ID NO: 2676)	Mtb groEL 1-13
DRB1*03:01	KTIAYDEEARR (SEQ ID NO: 2677)	Mtb groEL 3-13
DRB1*04:01	APYHFDLSGHAFG (SEQ ID NO: 2678)	Rye grass Lol p1 124-136
DRB1*04:01	ELEKYQQLNSERGVPN (SEQ ID NO: 2679)	Cow dander Bos d 2 143-158
DRB1*04:01	ETLLRAVESYLLAHS (SEQ ID NO: 2680)	Birch pollen allergen Bet v 1 141-155
DRB1*04:01	VAAAPQVKYAVFEAALTKAI (SEQ ID NO: 2645)	Timothy grass Phl p 5 p26
DRB1*04:01	FHTYTIDWTKDAVTW (SEQ ID NO: 2681)	Aspergillus fumigatus Crf1/p41 170-184
DRB1*04:01	NFIRMVISNPAAT (SEQ ID NO: 2682)	GAD65 555-567
DRB1*04:01	IAFTSEHSHFSK (SEQ ID NO: 2683)	GAD65 274-286
DRB1*04:01	GAGSLQPLALEGSLQKRG (SEQ ID NO: 2684)	Proinsulin 73-90
DRB1*04:01	GIVEQCCTSICSLYQ (SEQ ID NO: 2685)	Proinsulin 90-104
DRB1*04:01	ATEG[cit]V[cit]VNSAYQDK (SEQ ID NO: 2686)	Cit aggregan 89-103
DRB1*04:01	ATIKAEFV[cit]AETPYM (SEQ ID NO: 2687)	Cit CILP 297?311
DRB1*04:01	DVMNILLQYVVKSFDRSTKV (SEQ ID NO: 2030)	GAD65 113-132
DRB1*04:01	GKLYGI[cit]DV[cit]STRDR (SEQ ID NO: 2688)	Cit CILP 982?996
DRB1*04:01	GVYAT[cit]SSAV[cit]L[cit]SSVPGVR (SEQ ID NO: 2689)	Cit vimentin aa 59-78
DRB1*04:01	IFDS[cit]GNPTVEVDLF (SEQ ID NO: 2690)	Cit alpha-enolase 11-25
DRB1*04:01	KGMAALPRLIAFTSEHSHFS (SEQ ID NO: 2691)	GAD65 265?284
DRB1*04:01	QDFTN[cit]INKLKNS (SEQ ID NO: 2692)	Cit fibrinogen-alpha 79-91
DRB1*04:01	WNRQLYPEWTEAQRDL (SEQ ID NO: 2693)	gp100 44-59
DRB1*04:01	ISPNSVFSQWRVVCDSLEDYD (SEQ ID NO: 2694)	Typr1 277-297
DRB1*04:01	ESEFQAALSRKVAKL (SEQ ID NO: 2695)	MAGE-A6 102-116
DRB1*04:01	LTQYFVQENYLEYRQVPG (SEQ ID NO: 2696)	MAGE-A6 246-263
DRB1*04:01	YACFVSNLATGRNNS (SEQ ID NO: 2697)	CEA 653-667

DRB1*04:01 NYTLRVDCTPLMYSL (SEQ ID NO: 2698) PSMA 459-473
 DRB1*04:01 IYRRRLMKQDFSVPQLPHS (SEQ ID NO: 2699) gp100 615-633
 DRB1*04:01 ALHIYMDGTMSQVQGSA (SEQ ID NO: 2700) tyrosinase 365-381
 DRB1*04:01 RNGYRALMDKSLHVGTCALTRR (SEQ ID NO: 2701) MART-1 51-73
 DRB1*04:01 LKEFTVSGNILTIRL (SEQ ID NO: 2649) NY-ESO-1 123-137
 DRB1*04:01 LLEFYLAMPFATPME (SEQ ID NO: 2650) NY-ESO-1 87-101
 DRB1*04:01 KVPIKWMALLESILRRRF (SEQ ID NO: 2651) HER2 883-899
 DRB1*04:01 QALNTLVKQLSSNFGAI (SEQ ID NO: 2702) SARS-CoV S protein (939-955)(conserved in SARS-CoV-2)
 DRB1*04:01 QLIRAAEIRASANLAATK (SEQ ID NO: 2655) SARS-CoV S protein (993-1010)
 (conserved in SARS-CoV-2)
 DRB1*04:01 PYYVVDLSVRGM (SEQ ID NO: 2703) EBV BHRF1 122-133
 DRB1*04:01 AEGLRALLARSHVER (SEQ ID NO: 2704) EBV EBNA1 482-496
 DRB1*04:01 GQTYHLIVDTDSLGNPSLSV (SEQ ID NO: 2705) EBV EBNA2 11-30
 DRB1*04:01 LSLFLPSDFPFSVRDL (SEQ ID NO: 2706) HBV core 45-59
 DRB1*04:01 GYKVLVLNPSVAATL (SEQ ID NO: 2707) HCV polyprotein 1248-1262, NS3
 DRB1*04:01 SGIQYLALSTLPGNPAIASL (SEQ ID NO: 2708) HCV polyprotein 1770-1790, NS4b
 DRB1*04:01 AFSPEVIPMFSALESGATPQ (SEQ ID NO: 2709) HIV-1 gag 163-182
 DRB1*04:01 FWRGENGRKTRIAYERMCNILKGK (SEQ ID NO: 2710) Influenza A NP 206-229
 DRB1*04:01 GFVFTLTVPSE (SEQ ID NO: 2711) Influenza A MP 61-72
 DRB1*04:01 PKYVKQNTLKLAT (SEQ ID NO: 2666) Influenza A HA 307-319
 DRB1*04:01 EYLNKIQNSLSTEWSPCSVT (SEQ ID NO: 2667) CSP 326-345
 DRB1*04:01 RTELLKDAIGEGK (SEQ ID NO: 2712) MOG 97-109
 DRB1*04:01 TWTCQSIAPFSKTSASIGS (SEQ ID NO: 2713) PLP 180-199
 DRB1*04:01 PVSKMRMATPLLMQA (SEQ ID NO: 2668) CLIP 87-101
 DRB1*04:01 SSPKPWIYATSNLAS (SEQ ID NO: 2714) Rituximab Light chain 41-55
 DRB1*04:02 PKYVKQNTLKLAT (SEQ ID NO: 2666) Influenza A HA 307-319
 DRB1*04:02 PVSKMRMATPLLMQA (SEQ ID NO: 2668) CLIP 87-101
 DRB1*04:05 LWVNNQSLPVSP (SEQ ID NO: 2715) CEA 321-333
 DRB1*04:05 PKYVKQNTLKLAT (SEQ ID NO: 2666) Influenza A HA 307-319
 DRB1*04:05 PVSKMRMATPLLMQA (SEQ ID NO: 2668) CLIP 87-101
 DRB1*07:01 DRVNFKYSFSVIE (SEQ ID NO: 2716) Alder pollen Aln g 1 76-88
 DRB1*07:01 VAAAPQVKYAVFEAALTKAI (SEQ ID NO: 2645) Timothy grass Phl p 5 p26
 DRB1*07:01 VGLLKAVESYLLA (SEQ ID NO: 2717) Alder pollen Aln g 1 142-154
 DRB1*07:01 YYSNVTATRLSSTNS (SEQ ID NO: 2718) Pertussis toxin subunit 2 129-144
 DRB1*07:01 EPDVYYTSAFVFPTK (SEQ ID NO: 2719) CMV pp65 177-191
 DRB1*07:01 PDDYSNTHSTRYVT (SEQ ID NO: 2720) CMV gB 215-229
 DRB1*07:01 VPGLYSPCRAFFNKEELL (SEQ ID NO: 2721) EBV MCP 1264-1281
 DRB1*07:01 PGPLRESIVCYFMVFLQTHI (SEQ ID NO: 2722) EBV EBNA1 551-570
 DRB1*07:01 VYGGSKTSLYNLRRGTALAI (SEQ ID NO: 2723) EBV EBNA1 509-528
 DRB1*07:01 PRSPTVFYNIPPMPLPPSQL (SEQ ID NO: 2724) EBV EBNA2 276-295
 DRB1*07:01 AYCLWMMMLISQAEAALELIT (SEQ ID NO: 2725) HCV NS2 732-753
 DRB1*07:01 WPLLLLLLALPQRAYAQ (SEQ ID NO: 2726) HCV NS2 47-63
 DRB1*07:01 SLTITSLRRHNWITSCS (SEQ ID NO: 2727) HCV NS5a 1957-1975
 DRB1*07:01 TTVRLRAYMNTPLPVC (SEQ ID NO: 2728) HCV NS3 1535-1551
 DRB1*07:01 FRDYVDRFYKTLRAEQASQE (SEQ ID NO: 2664) HIV-1 gag 293-312
 DRB1*07:01 PKYVKQNTLKLAT (SEQ ID NO: 2666) Influenza A HA 307-319
 DRB1*07:01 EYLNKIQNSLSTEWSPCSVT (SEQ ID NO: 2667) CSP 326-345
 DRB1*07:01 PVSKMRMATPLLMQA (SEQ ID NO: 2668) CLIP 87-101
 DRB1*07:01 WVVKQTPGRGLEWIGA (SEQ ID NO: 2729) Rituximab Heavy chain 36-50
 DRB1*07:01 SSPKPWIYATSNLAS (SEQ ID NO: 2714) Rituximab Light chain 41-55

DRB1*07:01	EWVAEIRSKSINSAT (SEQ ID NO: 2730)	Infliximab Heavy chain 46-60
DRB1*09:01	VAAAPQVKYAVFEAALTKEI (SEQ ID NO: 2645)	Timothy grass Phl p 5 p26
DRB1*09:01	LLEFYLAMPFATPME (SEQ ID NO: 2650)	NY-ESO-1 87-101
DRB1*09:01	PVSKMRMATPLLMQA (SEQ ID NO: 2668)	CLIP 87-101
DRB1*09:01	MTEQQWNFAGIEAAA (SEQ ID NO: 2731)	Mtb ESAT6 1-15
DRB1*11:01	LEAAFNAEFNEIRRVLLEEN (SEQ ID NO: 2732)	Peanut Ara h 1 321-340
DRB1*11:01	TSRNNPFYFSPRRFSTRYGN (SEQ ID NO: 2733)	Peanut Ara h 1 169-188
DRB1*11:01	VVNKGTGNLELVAVRKEQQQ (SEQ ID NO: 2734)	Peanut Ara h 1 457-476
DRB1*11:01	VAAAPQVKYAVFEAALTKEI (SEQ ID NO: 2645)	Timothy grass Phl p 5 p26
DRB1*11:01	TSYVKVLHMHMVKISG (SEQ ID NO: 2735)	MAGE-A3 281-295
DRB1*11:01	ENIQRFLPNPAGVQLEDPEF (SEQ ID NO: 2736)	Factor VIII 589?608
DRB1*11:01	FRDYVDRFYKTLRAEQASQE (SEQ ID NO: 2664)	HIV-1 gag 293-312
DRB1*11:01	PKYVKQNTLKLAT (SEQ ID NO: 2666)	Influenza A HA 307-319
DRB1*11:01	PVSKMRMATPLLMQA (SEQ ID NO: 2668)	CLIP 87-101
DRB1*11:01	VSIDKFRIFCKALNPK (SEQ ID NO: 2737)	TT 1084-1099
DRB1*11:01	FNNFTVSFWLRVPKVSASHLE (SEQ ID NO: 2738)	TT 947-967
DRB1*11:01	KFIKRYTPNNEIDSF (SEQ ID NO: 2739)	TT 1174-1189
DRB1*11:01	QYIKANSKFIGITEL (SEQ ID NO: 2740)	TT 830-844
DRB1*11:01	WVKQTPGRGLEWIGA (SEQ ID NO: 2729)	Rituximab Heavy chain 36-50
DRB1*15:01	VAAAPQVKYAVFEAALTKEI (SEQ ID NO: 2645)	Timothy grass Phl p 5 p26
DRB1*15:01	VGLLKAVESYLLA (SEQ ID NO: 2717)	Alder pollen Aln g 1 142-154
DRB1*15:01	AVNIVGYSNAQGVVDY (SEQ ID NO: 2741)	House dust mite Der p1 251-265
DRB1*15:01	ETLLRAVESYLLAHS (SEQ ID NO: 2647)	Birch pollen allergen Bet v 1 142-156
DRB1*15:01	LRQMRTVTPIRMQGG (SEQ ID NO: 2644)	House dust mite Der p1 96-110
DRB1*15:01	DENPVVHFFKNIVTPRTPP (SEQ ID NO: 2027)	Myelin basic protein 83-101
DRB1*15:01	MSIYVYALPLKMLNI (SEQ ID NO: 2742)	CMV pp65 109-123
DRB1*15:01	NFPYLVAYQATVCARA (SEQ ID NO: 2743)	HCV polyprotein 1582?1597, NS3
DRB1*15:01	GINAVAYYRGLDVS (SEQ ID NO: 2744)	HCV polyprotein 1411?1425, NS3
DRB1*15:01	TTVRLRAYMNTPLPVC (SEQ ID NO: 2728)	HCV NS3 1535-1551
DRB1*15:01	PVSKMRMATPLLMQA (SEQ ID NO: 2668)	CLIP 87-101
DRB1*15:01	SSPKPWIYATSNLAS (SEQ ID NO: 2714)	Rituximab Light chain 41-55
DRB1*15:01	EWVAEIRSKSINSAT (SEQ ID NO: 2730)	Infliximab Heavy chain 46-60
DRB1*15:01	MHVSVFMAYPEMLAA (SEQ ID NO: 2745)	Mtb PE family protein
DRB1*15:01	MSQIMYNYPAMMAHA (SEQ ID NO: 2746)	Mtb ESAT-6 like protein

[00102] As further illustrative and non-limiting examples, the following antigens provided in Table 6 are known in the art to be relevant to celiac disease. In some embodiments, the TCR recognition domain can comprise one or more of the peptides provided in Table 6. In some embodiments, the TCR recognition domain can comprise a sequence with at least 80%, at least 85%, at least 90%, at least 95%, at least 98% or greater sequence identity to one of the peptides of Table 6. In some embodiments, the TCR recognition domain can comprise a sequences with at least 95% sequence identity to one of peptides of Table 6, wherein that sequence retains the wild-type activity of the peptide of Table 6.

[00103] Table 6. The sequences are, in the order presented, SEQ ID NOs:1000-2012.

ID	Type	Description	Toxicity Form	HLADQ Refs	SeqLen	Sequence
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1	alpha-gliadin	alpha-gliadin CT-1 (p1-p22 of B 3142)	Toxic	Native	Unknown	41	
22	VPVPQLQPQNPSQQQPQEQVPL						
2	alpha-gliadin	alpha-gliadin peptide CT-2 (p23-p53 of B 3142)	Toxic	Native	Unknown		
41	31	VQQQQFPGQQQPFPQPYPQPQPFPSQQPY					
3	alpha-gliadin	alpha-gliadin p14 (p1-p19)	Immunogenic	Native	DQ2	34	19
		VRVPVPQLQPQNPSQQQPQ					
4	alpha-gliadin	alpha-gliadin p15 (p11-p28)	Immunogenic	Native	DQ2	34	18
		QNPSQQQPQEQVPLVQQQ					
5	alpha-gliadin	alpha-gliadin p209	Immunogenic	Native	DQ2	39,34	20
		FPGQQQPFPQPYPQPQPF					
7	alpha-gliadin	alpha-gliadin (p44-p55)	Immunogenic, Toxic	Native	HLA-DR	10	12
		PQPQPFPSQQPY					
8	alpha-gliadin	Epitope DQ2-alpha-I/II/III	Immunogenic	Native	DQ2	62	20
		YLQLQFPQPQLPYPQPQLP					
9	alpha-gliadin	alpha2-gliadin 1420 (p56-p70)	Immunogenic	Native	DQ2, DQ8		17
	15	YLQLQFPQPQLPYP					
10	alpha-gliadin	alpha2-gliadin 33-mer (p57-p89)	Immunogenic	Native	DQ2, DQ8		
(DQ2/8)	2,25	33 LQLQFPQPQLPYPQPQLPYPQPQLPYPQPQPF					
11	alpha-gliadin	Deamidated alpha2-gliadin 33-mer (p57-p89)	Immunogenic	Deamidated			
DQ2.5	61	33 LQLQFPQPQLPYPQPQLPYPQPQLPYPQPQPF					
12	alpha-gliadin	alpha-2 gliadin G8 (p56-p75)	Toxic, Immunogenic	Native	DQ2		
51,63	20	LQLQFPQPQLPYPQPQLPY					
13	alpha-gliadin	alpha-2 gliadin G9 (p56-p75; E65)	Immunogenic	Deamidated			
DQ2	51	20 LQLQFPQPQLPYPQPQLPY					
14	alpha-gliadin	alpha-9 gliadin G5 (p56-p68)	Immunogenic	Native	DQ2	51	13
		LQLQFPQPQLPY					
15	alpha-gliadin	alpha-9 gliadin G5 (p56-p68; E65)	Immunogenic	Deamidated			
DQ2	51	13 LQLQFPQPQLPY					
16	alpha-gliadin	Wheat peptide W02	Immunogenic	Native	DQ2	62,86	16
		QLQFPQPQLPYPQPQ					
17	alpha-gliadin	Glia-alpha9 (p57-p71)	Immunogenic	Native	DQ2	9	15
		QLQFPQPQLPYPQP					
18	alpha-gliadin	Glia-alpha9 (p57-p71; T69 and H70)	Immunogenic	Native	DQ2	9	
	15	QLQFPQPQLPYTHP					
19	alpha-gliadin	Glia-alpha9 (p57-p71; R59)	Immunogenic	Native	DQ2	9	15
		QLRFPQPQLPYPQP					
20	alpha-gliadin	Glia-alpha9 (p57-p71; H63 and H70)	Immunogenic	Native	DQ2	9	
	15	QLQFPQPQLPYPHP					
21	alpha-gliadin	Glia-alpha9 (p57-p71; A63)	Immunogenic	Native	DQ2	9	15
		QLQFPQQAQLPYPQP					
22	alpha-gliadin	Glia-alpha9 (p57-p70)	Immunogenic	Native	DQ2	8	14
		QLQFPQPQLPYPQ					
23	alpha-gliadin	Glia-alpha9 (p57-p70; E65)	Immunogenic	Deamidated	DQ2	8	
	14	QLQFPQPQLPYPQ					
24	alpha-gliadin	alpha-9 gliadin (p57-p68); alpha2/alpha9 gliadin	Immunogenic	Native			
DQ2	14,23,2,84	12 QLQFPQPQLPY					
25	alpha-gliadin	alpha-9 gliadin (p57-p68; E65); alpha-I	Immunogenic	Deamidated			
DQ2	43,14,8,84	12 QLQFPQPQLPY					
26	alpha-gliadin	alpha-9 gliadin epitope homolog (p57-p68; E63 (considered native form of synthetic))	Immunogenic	Native	DQ2	8	12
		QLQFPPEPQLPY					

27	alpha-gliadin	alpha-9 gliadin epitope homolog (p57-p68; E63 and E65 (tTG-treated form))	Immunogenic	Deamidated	DQ2	8	12	QLQPFPEPELPY	
28	alpha-gliadin	alpha-9 gliadin epitope homolog (p57-p68; Q64 (considered native form of synthetic))	Immunogenic	Native	DQ2	8	12	QLQPFPPQQLPY	
29	alpha-gliadin	alpha-9 gliadin epitope homolog (p57-p68; Q64 and E63 (tTG-treated form))	Immunogenic	Deamidated	DQ2	8	12	QLQPFPEQQLPY	
30	alpha-gliadin	alpha-9 gliadin epitope homolog (p57-p68; Q64 and E65 (tTG-treated form))	Immunogenic	Deamidated	DQ2	8	12	QLQPFPPQQLPY	
31	alpha-gliadin	alpha-9 gliadin epitope homolog (p57-p68; Q64, E63 and E65 (tTG-treated form))	Immunogenic	Deamidated	DQ2	8	12	QLQPFPEQQLPY	
32	alpha-gliadin	DQ2-Glia-alpha1 epitope (p58-p72)	Immunogenic	Native	DQ2	59	15	LQPFPPQLPYPPQPQ	
33	alpha-gliadin	DQ2-Glia-alpha1 epitope (p58-p72; E65)	Immunogenic	Deamidated	DQ2	59	15	LQPFPPQPELPYPPQPQ	
34	alpha-gliadin	DQ2-Glia-alpha1 epitope (p58-p72; S64)	Immunogenic	Native	DQ2	59	15	LQPFPPQSQLPYPPQPQ	
35	alpha-gliadin	DQ2-Glia-alpha1 epitope (p58-p72; S64 and E65)	Immunogenic	Deamidated	DQ2	59	15	LQPFPPQSELPYPPQPQ	
36	alpha-gliadin	DQ2-alpha-I epitope (p58-p68)	Immunogenic	Native	DQ2	47	11	LQPFPPQQLPY	
37	alpha-gliadin	DQ2-alpha-I epitope (p58-p68; E65 considered Deamidated form)	Immunogenic	Deamidated	DQ2	47	11	LQPFPPQPELPY	
38	alpha-gliadin	Glia-alpha2 (p60-p76)	Immunogenic	Native	DQ2	8	17	QPFPPQQLPYPPQQLPY	
39	alpha-gliadin	Glia-alpha2 (p60-p76; E66)	Immunogenic	Deamidated	DQ2	8	17	QPFPPQPELPYPPQQLPY	
40	alpha-gliadin	Glia-alpha2 (p60-p76; E73)	Immunogenic	Deamidated	DQ2	8	17	QPFPPQQLPYPPQPELPY	
41	alpha-gliadin	Glia-alpha2 (p60-p76; E66 and E73)	Immunogenic	Deamidated	DQ2	8	17	QPFPPQPELPYPPQPELPY	
42	alpha-gliadin	Glia-alpha2 (p60-p69)	Immunogenic	Native	DQ2	45	10	QPFPPQQLPY	
43	alpha-gliadin	Glia-alpha2 (p60-p69; E66)	Immunogenic	Deamidated	DQ2	45	10	QPFPPQPELPY	
44	alpha-gliadin	alpha2-gliadin 1421 (p61-p75)	Immunogenic	Native	DQ2, DQ8	17	15	PPFPQQLPYPPQQLP	
45	alpha-gliadin	Glia-alpha2 (p61-p71)	Immunogenic	Native	DQ2	59	11	PPFPQQLPYPPQ	
46	alpha-gliadin	Glia-alpha2 (p61-p71; E66)	Immunogenic	Deamidated	DQ2	59	11	PPFPQPELPYPPQ	
47	alpha-gliadin	Glia-alpha2 (p61-p71; T70 and H71)	Immunogenic	Native	DQ2	59	11	PPFPQQLPYTH	
48	alpha-gliadin	Glia-alpha2 (p61-p71; T70, H71 and E66)	Immunogenic	Deamidated	DQ2	59	11	PPFPQPELPYTH	
49	alpha-gliadin	Glia-alpha2 (p61-p71; H64)	Immunogenic	Native	DQ2	59	11	PPFHPQLPYPPQ	
50	alpha-gliadin	Glia-alpha2 (p61-p71; H64 and E66)	Immunogenic	Deamidated	DQ2	59	11	PPFHPPELPYPPQ	
53	alpha-9 gliadin	DQ2.5_glia_alpha-la	Immunogenic	Native	DQ2.5	14,90,17,23	9	PPFPQQLPY	

54	alpha-9 gliadin	DQ2.5_glia_alpha-1a	Immunogenic	Deamidated	DQ2.5	14,90,17,23	9
		PFPQPELPY					
55	alpha-gliadin	alpha-2 gliadin 1206 (p62-p75)	Immunogenic	Native	DQ2	14,1,2	14
		PQPQLPYQPQLPY					
56	alpha-gliadin	alpha-II/alpha-III epitope (p62-p75; E72)	Immunogenic	Deamidated			
	DQ2 14	14 PQPQLPYQPPELPY					
57	alpha-gliadin	alpha-2 gliadin (p62-p75; E65)	Immunogenic	Deamidated	DQ2		
	14,25 14	PQPELPYPQPQLPY					
58	alpha-gliadin	alpha-2 gliadin (p62-p75; E65 and E72)	Immunogenic	Deamidated			
	DQ2 14	14 PQPELPYPQPPELPY					
59	alpha-gliadin	G4-9A gliadin (p62-p75; E65 and A70)	Immunogenic	Deamidated			
	DQ2 51	14 PQPELPYPAPQLPY					
60	alpha-gliadin	G4-11A gliadin (p62-p75; E65 and A72)	Immunogenic	Deamidated			
	DQ2 51	14 PQPELPYPQPALPY					
61	alpha-gliadin	G4-12A gliadin (p62-p75; E65 and A73)	Immunogenic	Deamidated			
	DQ2 51	14 PQPELPYPQPQAPY					
62	alpha-gliadin	G4-13A gliadin (p62-p75; E65 and A74)	Immunogenic	Deamidated			
	DQ2 51	14 PQPELPYPQPQLAY					
63	alpha-gliadin	G4-14A gliadin (p62-p75; E65 and A70)	Immunogenic	Deamidated			
	DQ2 51	14 PQPELPYPQPQLPA					
64	alpha-gliadin	alpha-2 gliadin (p62-p73)	Immunogenic	Native	DQ2	25,47	12
		PQPQLPYQPQL					
65	alpha-gliadin	alpha-2 gliadin (p62-p73; E65)	Immunogenic	Deamidated	DQ2		
	25,47 12	PQPELPYPQPQL					
66	alpha-gliadin	alpha-II (p62-p72)	Immunogenic	Native	DQ2	43	11
		PQPQLPYQPQ					
67	alpha-gliadin	alpha-II (p62-p72; E65 and E72)	Immunogenic	Deamidated	DQ2		43
	11	PQPELPYPQPE					
68	alpha-2 gliadin	CAUTION 100% match to one fungal protein and many wheat proteins with multiple epitopes	DQ2.5_glia_alpha2	Immunogenic	Native	DQ2.5	14,17,23 9
		PQPQLPYPQ					
69	alpha-2 gliadin	DQ2.5_glia alpha2	Immunogenic	Deamidated	DQ2.5	14,17,23	9
		PQPELPYPQ					
70	alpha-gliadin	alpha2-gliadin 1423 (p71-p85)	Immunogenic	Native	DQ2, DQ8		17
	15	QPQLPYQPQLPYPQ					
71	alpha-gliadin	a-gliadin (p62-p84)	Immunogenic	Native	DQ2	62	20
		PQLPYQPQLPYQPQLPY					
72	alpha-gliadin	alpha-III-gliadin (p62-p79)	Immunogenic	Native	DQ2	25	12
		PQLPYQPQLPY					
73	alpha-gliadin	alpha-III-gliadin (p62-p79; E76)	Immunogenic	Deamidated	DQ2		25
	12	PQLPYQPPELPY					
74	alpha-gliadin	alpha-I epitope	Immunogenic	Native	DQ2	25	12
		LQLPFPQPQLPY					
75	alpha-gliadin	alpha-I epitope	Deamidated form	Immunogenic	Deamidated		
	DQ2 25	12 LQLPFPQPPELPY					
76	alpha-gliadin	Glia-alpha2 25-mer (p64-p89)	Immunogenic	Native	DQ2	57	25
		PQLPQLFPQYPQPQLPYQPQPF					
77	alpha-gliadin	Glia-alpha2 25-mer (p64-p89; E65)	Immunogenic	Deamidated			
	DQ2 57	25 PELPQLFPQYPQPQLPYQPQPF					
78	alpha-gliadin	Glia-alpha2 25-mer (p64-p89; E79)	Immunogenic	Deamidated			
	DQ2 57	25 PQLPQLFPQYPQPPELPYPQPQPF					

79	alpha-gliadin	Glia-alpha2 25-mer (p64–p89; E65 and E79)	Immunogenic	Deamidated				
	DQ2 57	25 PELPQFLQPQYPQPELPYPQPQPF						
80	alpha-gliadin	alpha2-gliadin 1422 (p66-p80)	Immunogenic	Native	DQ2	17	15	
		QLPYPQPQLPYPQPQ						
81	alpha-gliadin	alpha-III epitope (p66-p78)	Immunogenic	Native	DQ2	47	13	
		QLPYPQPQLPYPQ						
82	alpha-gliadin	alpha-III epitope (p66-p78; E72)	Immunogenic	Deamidated	DQ2	47		
	13	QLPYPQPELPYPQ						
83	alpha-gliadin	Wheat peptide W01	Immunogenic	Native	DQ2	62	12	
		LPYPQPQLPYPQ						
84	alpha-3 gliadin	DQ2.5_glia_alpha 1b	Immunogenic	Native	DQ2.5	23	9	
		PYPQPQLPY						
85	alpha-3 gliadin	DQ2.5_glia_alpha 1b	Immunogenic	Deamidated	DQ2.5	23	9	
		PYPQPELPY						
86	alpha-gliadin	Glia-alpha2 18-mer (p71– p89)	Immunogenic	Native	DQ2	82	18	
		QPQYPQPQLPYPQPQPF						
87	alpha-gliadin	Glia-alpha2 18-mer (p71– p89; E79)	Immunogenic	Deamidated				
	DQ2 82,57	18 QPQYPQPELPYPQPQPF						
88	alpha-gliadin	Wheat peptide W18	Immunogenic	Native	DQ2	62,82	20	
		PQLPYPQPQLPYPQPQPF						
89	alpha-gliadin	Wheat peptide W18	Immunogenic	Deamidated	DQ2	62	20	
		PQLPYPQPELPYPQPQPF						
90	alpha-gliadin	Wheat peptide W18	Immunogenic	Native	DQ2	62	16	
		YPQPQLPYPQPQPF						
91	alpha-gliadin	Wheat peptide W18	Immunogenic	Deamidated	DQ2	62	16	
		YPQPELPYPQPQPF						
92	alpha-gliadin	alpha2-gliadin 1424 (p76-p90)	Immunogenic	Native	DQ2	17	15	
		YPQPQLPYPQPQPF						
93	alpha-20 gliadin	DQ2.5_glia_alpha 3	Immunogenic	Native	DQ2.5	90,8	9	
		FRPQQYPYQ						
94	alpha-20 gliadin	DQ2.5_glia_alpha 3	Immunogenic	Deamidated	DQ2.5	90,8	9	
		FRPEQYPYQ						
95	alpha-gliadin	Gliadin (p198-p222)	Immunogenic	Native	DQ8 (DQ2/8, DQ1/8)	3		
	25	QQPQQQYPSGQGSFQPSQQNPQAAQ						
96	alpha-gliadin	alpha-2 gliadin (p219-p242) AJ133612	Immunogenic	Native	DQ8	6		
	24	QQPQQQYPSGQGSFQPSQQNPQAAQ						
97	alpha-gliadin	alpha-2 gliadin (p219-p242; E229 and E237) AJ133612	Immunogenic					
	Deamidated	DQ8 6 24 QQPQQQYPSGEGSFQPSQENPQAAQ						
98	alpha-gliadin	alpha-2 gliadin (p219-p242; E229) AJ133612	Immunogenic	Deamidated				
	DQ8 6	24 QQPQQQYPSGEGSFQPSQQNPQAAQ						
99	alpha-gliadin	alpha-2 gliadin (p219-p242; E237) AJ133612	Immunogenic	Deamidated				
	DQ8 6	24 QQPQQQYPSGQGSFQPSQENPQAAQ						
100	alpha-gliadin	alpha-gliadin (p220-p239) P18573	Immunogenic	Native	DQ8	54		
	20	QPQQQYPSGQGSFQPSQQNP						
101	alpha-gliadin	Gda09 (p202–p219) P18573	Immunogenic	Native	DQ8 (DQ2/8, DQ1/8)	3,4	18	
		QQYPSGQGSFQPSQQNPQ						
102	alpha-gliadin	Gda09 (p203–p220) P18573 (alpha-gliadin (alpha-I))	Immunogenic					
	Native DQ8	5 18 QYPSGQGSFQPSQQNPQA						
103	alpha-gliadin	Gda09 (p203–p220; E216) P18573 (alpha-gliadin (alpha-I))						
	Immunogenic	Deamidated DQ8 5 18 QYPSGEGSFQPSQENPQA						

104	alpha-gliadin	alpha2-gliadin 1447 (p226-p240) 15 YPSGQGSFQPSQQNP	Immunogenic	Native	DQ8	17
105	alpha-gliadin	Gliadin (p205-p222) PSGQGSFQPSQQNPQAQG	Immunogenic	Native	DQ8 (DQ2/8)	3 18
106	alpha-gliadin (DQ2/8)	Gliadin (p205-p216) ; alpha2-gliadin 46,3 12 PSGQGSFQPSQQ	Immunogenic	Native	DQ8	
107	alpha-gliadin (DQ2/8)	Gliadin (p205-p215) ; alpha2-gliadin 46,3 11 PSGQGSFQPSQ	Immunogenic	Native	DQ8	
108	alpha-gliadin 46,4 12	Gda09 (p206-p217) P18573 SGQGSFQPSQQN	Immunogenic	Native	DQ8 (DQ2/8)	
109	alpha-gliadin DQ8 (DQ2/8)	Gda09 (p206-p217; E215) P18573 46 12 SGQGSFQPSEQN	Immunogenic	Deamidated		
110	alpha-gliadin DQ8 (DQ2/8)	Gda09 (p206-p217; E208) P18573 46,83,4 12 SGEGSFQPSQQN	Immunogenic	Deamidated		
111	alpha-gliadin DQ8 (DQ2/8)	Gda09 (p206-p217; E216) P18573 46,4 12 SGQGSFQPSQEN	Immunogenic	Deamidated		
112	alpha-gliadin DQ8 (DQ2/8)	Gda09 (p206-p217; E208 and E216) P18573 3,4 12 SGEGSFQPSQEN	Immunogenic	Deamidated		
113	alpha-gliadin	alpha2-gliadin (p228-p236) 9 GQGSFQPSQ	Immunogenic	Native	DQ8 (DQ2/8)	6
115	alpha-2 gliadin	CAUTION 100% identity match to Dicot plant protein and many wheat family				
	DQ8_glia_alpha 1					
	DQ8.5_glia_alpha 1	Immunogenic	Native	DQ8 (DQ2/8)	6,90,3 9	QGSFQPSQQ
116	alpha-2 gliadin	DQ8_glia_alpha 1				
	DQ8.5_glia_alpha 1	Immunogenic	Deamidated	DQ8 (DQ2/8)	90,17,83	9
		EGSFQPSQQ				
117	alpha-2 gliadin	DQ8_glia_alpha 1				
	DQ8.5_glia_alpha 1	Immunogenic	Deamidated	DQ8 (DQ2/8)	90,17	9
		QGSFQPSQE				
118	alpha-2 gliadin	DQ8_glia_alpha 1				
	DQ8.5_glia_alpha 1	Immunogenic	Deamidated	DQ8 (DQ2/8)	90,17	9 EGSFQPSQE
119	alpha-gliadin	alpha2-gliadin 1448 (p231-p245) 15 GSFQPSQQNPQAQGS	Immunogenic	Native	DQ8	17
120	alpha-gliadin	alpha2-gliadin 1450 (p241-p255) 15 QAQGSVQPQQLPQFE	Immunogenic	Native	DQ8 and	
	weak DQ2	17				
121	alpha-gliadin	Wheat peptide W02 MQLQFPQPQLPYQPQLPY	Immunogenic	Native	DQ2	62 20
122	alpha-gliadin	Wheat peptide W02 MQLQFPQPQLPYQPQLPY	Immunogenic	Deamidated	DQ2	62 20
123	alpha-gliadin	Wheat peptide W02 QLQFPQPQLPYQPQ	Immunogenic	Deamidated	DQ2	62 16
124	alpha-gliadin	Wheat peptide W02 ELQFPQPQLPYQPQ	Immunogenic	Deamidated	DQ2	62 16
125	alpha-gliadin	Wheat peptide W02 QPFPQPQLPYQPQ	Immunogenic	Native	DQ2	62 12
126	alpha-gliadin	Wheat peptide W02 QPFPQPQLPYQPQ	Immunogenic	Deamidated	DQ2	62 12
127	gamma-gliadin	Wheat peptide W01 PQFPQPQLPYQPQLPYQP	Immunogenic	Native	DQ2	62 20
128	gamma-gliadin	Wheat peptide W01 PQFPQPQLPYQPQLPYQP	Immunogenic	Deamidated	DQ2	62 20

129	gamma-gliadin	Wheat peptide W01	Immunogenic	Deamidated	DQ2	62	12
		LPYPQPELPYPQ					
130	alpha-gliadin	Wheat peptide W34	Immunogenic	Native	DQ2	62	20
		VAHAIIIMHQQQQQQQEQQQ					
131	alpha-gliadin	Wheat peptide W34	Immunogenic	Native	DQ2	62	16
		VAHAIIIMHQQQQQQQE					
132	alpha-gliadin	Analog of alpha-gliadin (p31-p49; A31)	Toxic	Native	DQ2	50	19
		AGQQQPFPPQQYPQPQPF					
133	alpha-gliadin	Analog of alpha-gliadin (p31-p49; A36)	Toxic	Native	DQ2	50	19
		LGQQQAFPPQQYPQPQPF					
134	alpha-gliadin	alpha20-gliadin (p91-p106)	Immunogenic	Native	DQ2	8	16
		PQPFRRPQQYPQPQPQ					
135	alpha-gliadin	alpha20-gliadin (p93-106; E97)	Immunogenic	Deamidated	DQ2	8	
		16 PQPFRRPEQQYPQPQPQ					
136	alpha-gliadin	Glia-alpha20-gliadin (p93-p106)	Immunogenic	Native	DQ2	19	14
		PFRPQQYPQPQPQ					
137	alpha-gliadin	Glia-alpha20-gliadin (p93-p106; E97)	Immunogenic	Deamidated	DQ2	19	14
		PFRPEQQYPQPQPQ					
138	alpha-gliadin	Glia-alpha20-gliadin (p96-106) minimal epitope	Immunogenic	Native	DQ2	19	11
		PQQYPQPQPQ					
139	alpha-gliadin	Glia-alpha20-gliadin (p96-106; E97) minimal epitope, synthetic	Immunogenic	Deamidated	DQ2	19	11
		PEQQYPQPQPQ					
140	alpha-gliadin	alpha-gliadin(p123-p132)	Immunogenic	Native	DQ8	64,30	10
		QLIPCMDVVL					
141	alpha-gliadin	alpha-gliadin (p206-p217)	Toxic	Native	DQ2 (A1 B8 DR3 DQ2 and A		
		24 B8 DR3 13 DQ2)					
142	alpha-gliadin	Peptide XT (1-55)	Toxic	Native	Unknown	29	55
		VRVPVPLQLPQNPSQQQPQEQLVPLVQQQQFLGQQQPFPPQQYPQPQPFPSQQPY					
143	alpha-gliadin	Peptide XT (p1-p30)	Toxic	Native	Unknown	29	30
		VRVPVPLQLPQNPSQQQPQEQLVPLVQQQQF					
144	alpha-gliadin	alpha-gliadin B 3142 (p3-p55)	Immunogenic, Toxic	Native	Unknown		
		40 53 VPVPQLQPNPSQQQPQEQLVPLVQQQQFGGQQQPFPPQQYPQPQPFPSQQPY					
146	alpha-gliadin	alpha-gliadin p19 (p21-p40)	Immunogenic	Native	DQ2	34	20
		QVPLVQQQQFLGQQQPFPPQ					
147	alpha-gliadin	alpha-gliadin p134	Immunogenic	Native	DQ2 (alpha1*0501,		
		39 19 QFLGQQQPFPPQQYPQPQ					
148	alpha-gliadin	alpha-gliadin p135	Immunogenic	Native	DQ2 (alpha1*0501,		
		39 18 FLGQQQPFPPQQYPQPQ					
149	alpha-gliadin	Peptide XT (p31-p55)	Immunogenic, Toxic	Native	Unknown		
		10,29 25 LGQQQPFPPQQYPQPQPFPSQQPY					
150	alpha-gliadin	alpha-gliadin (p31-p49)	Immunogenic, Toxic	Native	DQ2 (alpha1*0501,		
		39,11,49,34 19 LGQQQPFPPQQYPQPQPF					
151	alpha-gliadin	alpha-gliadin p126	Immunogenic	Native	DQ2 (alpha1*0501,		
		10,39,13,15,66,79 17 LGQQQPFPPQQYPQPQ					
152	alpha-gliadin	alpha-gliadin (p31-p43)	Immunogenic, Toxic	Native	HLA-DR		
		10,13,15,72,79 13 LGQQQPFPPQQPY					
153	alpha-gliadin	alpha-gliadin CAB76960 (p253-p272)	Immunogenic	Native	DQ8		54
		20 AMCNVYIPPYCAMAPFGIFG					
154	alpha-gliadin	alpha-gliadin (proline-rich domain)	Immunogenic	Native	Unknown		
		42 16 CPQPFPSQQPYLQLQG					

155	alpha-gliadin	alpha-gliadin (p5-p22) (proline-rich domain)	Immunogenic	Native				
	Unknown	42 18 CPQLQPQNPSQQQPQEQG						
156	alpha-gliadin	alpha-gliadin (p51-p70) Toxic	Native	DQ2	37	20		
		SQQPYLQLQFPQPQLPYSQ						
157	alpha-gliadin	Wheat peptide W08	Immunogenic	Native	DQ2	62	20	
		LQLQFPQPQLPYSQPQPF						
158	alpha-gliadin	Wheat peptide W08	Immunogenic	Deamidated	DQ2	62	20	
		LQLQFPQPQLPYSQPQPF						
159	alpha-gliadin	Glia-alpha9 (p57-p71; S69)	Immunogenic	Native	DQ2	9	15	
		QLQFPQPQLPYSQ						
160	alpha-gliadin	DQ2-Glia-alpha1 epitope (p58-p72; S69)	Immunogenic	Native				
	DQ2 59	15 LQFPQPQLPYSQPQ						
161	alpha-gliadin	DQ2-Glia-alpha1 epitope (p58-p72; S69 and E65)	Immunogenic					
	Deamidated	DQ2 59 15 LQFPQPQLPYSQPQ						
162	alpha-gliadin	DQ2-Glia-alpha1 epitope (p58-p72; S64 and S69)	Immunogenic					
	Native DQ2	59 15 LQFPQSQLPYSQPQ						
163	alpha-gliadin	DQ2-Glia-alpha1 epitope (p58-p72; S64, S69 and E65)	Immunogenic					
	Deamidated	DQ2 59 15 LQFPQSELPYSQPQ						
164	alpha-gliadin	Wheat peptide W08	Immunogenic	Native	DQ2	62	12	
		QFPQPQLPYSQ						
165	alpha-gliadin	Wheat peptide W08	Immunogenic	Deamidated	DQ2	62	12	
		QFPQPQLPYSQ						
166	alpha-gliadin	Glia-alpha	Immunogenic	Native	DQ2	59	11	
		PFPQPQLPYSQ						
167	alpha-gliadin	Glia-alpha in Deamidated form	Immunogenic	Deamidated	DQ2	59		
	11	PFPQPQLPYSQ						
168	alpha-gliadin	alpha-gliadin (p202-p220)	Toxic	Native	DQ2	11	19	
		QQYPLGQGSFRPSQNPQA						
169	alpha-gliadin	alpha-gliadin CAB76961 (p251-p270)	Immunogenic	Native	DQ8	54		
	20	VYIPPYCTIAPFGIFTNYR						
170	alpha-gliadin	Wheat peptide W13	Immunogenic	Native	DQ2	62	20	
		LQLQFPQPQLPYLQPQPF						
171	alpha-gliadin	Wheat peptide W13	Immunogenic	Deamidated	DQ2	62	20	
		LQLQFPQPQLPYLQPQPF						
172	alpha-gliadin	Glia-alpha9 (p57-p71; L69)	Immunogenic	Native	DQ2	9	15	
		QLQFPQPQLPYLQ						
173	alpha-gliadin	Wheat peptide W13	Immunogenic	Native	DQ2	62	12	
		QFPQPQLPYLQ						
174	alpha-gliadin	Wheat peptide W13	Immunogenic	Deamidated	DQ2	62	12	
		QFPQPQLPYLQ						
175	alpha-gliadin	alpha-gliadin 4037	Immunogenic	Native	Unknown	60	17	
		PPYCTIVPFGIFTNYR						
176	alpha-gliadin	alpha-gliadin	Immunogenic	Native	DQ2	62	20	
		LQLQFPQPQLPYYPQPQPF						
177	alpha-gliadin	alpha-Glia (p57-p73)	Immunogenic	Native	DQ2	57	17	
		QLQFPQPQLPYYPQPQ						
178	alpha-gliadin	alpha-Glia (p57-p73; E65)	Immunogenic	Deamidated	DQ2	57		
	17	QLQFPQPQLPYYPQPQ						
179	alpha-gliadin	alpha-Glia (p57-p73; T65 and S73)	Immunogenic	Native	DQ2	57		
	17	QLQFPQPQLPYYPQPS						

180	alpha-gliadin	alpha-gliadin (p57-p73; S73)	Immunogenic	Native	DQ2	28	17
		QLQFPQPQLPYQPQS					
181	alpha-gliadin	alpha-gliadin (p57-p73; S73 and E65)	Immunogenic	Deamidated			
	DQ2	28 17 QLQFPQPQLPYQPQS					
182	alpha-gliadin	Wheat peptide W09	Immunogenic	Native	DQ2	62	20
		LQFPQPQPFLPYQPQ					
183	alpha-gliadin	alpha-Glia AG11 (p78 –p95)	Immunogenic	Native	DQ2	57	17
		PQPQPFLPYQPQS					
184	alpha-gliadin	alpha-Glia AG11 (p78 –p95; E86)	Immunogenic	Deamidated			
	DQ2	57 17 PQPQPFLPYQPQS					
185	alpha-gliadin	Wheat peptide W09	Immunogenic	Native	DQ2	62	14
		QPQPFLPYQPQ					
186	alpha-gliadin	Wheat peptide W09	Immunogenic	Deamidated	DQ2	62	14
		EPQPFLPYQPQ					
187	alpha-gliadin	Wheat peptide W09	Immunogenic	Native	DQ2	62	12
		PQPFLPYQPQ					
188	alpha-gliadin	alpha-gliadin p211	Immunogenic	Native	DQ2	34	20
		FPGQQQFPQPQPQPF					
189	alpha-gliadin	alpha-Glia AG12 (p82–p98)	Immunogenic	Native	DQ2	57	17
		PQPQPFPQLPYQPQS					
190	alpha-gliadin	alpha-Glia AG12 (p82–p98; E90)	Immunogenic	Deamidated	DQ2	57	
	17	PQPQPFPQLPYQPQS					
191	omega-gliadin	Gliadin AAG17702 (p80-p99)	Immunogenic	Native	DQ8	54	20
		PFTQPQQPTPIQPQQPFPQ					
192	omega-gliadin	Wheat peptide W27	Immunogenic	Native	DQ2	62	11
		PFTQPQQPTPI					
193	omega-gliadin	Gliadin AAG17702 (p88-p107)	Immunogenic	Native	DQ8	54	20
		TPIQPQQPFPQPQQPQPF					
194	omega-gliadin	Wheat peptide W25	Immunogenic	Native	DQ2	62	11
		TPIQPQQPFPQ					
195	omega-gliadin	Wheat peptide W30; Rye peptide R28	Immunogenic	Native	DQ2	62	
	12	PQQPFPQPQPQ					
197	omega-gliadin	omega-gliadin	Immunogenic	Native	DQ2	62	20
		PQQPQPQPFPQPQPFPW					
198	omega-gliadin	Epitope DQ2-omega-I/II	Immunogenic	Native	DQ2	62	20
		PQQPQPFPQPQPFPWQPQ					
199	omega-gliadin	p4-p18 omega-gliadin of AAG17702 (p81-p102)	Immunogenic	Native			
	DQ2	62 15 PQQPQPFPQPQPQPF					
200	omega-gliadin	p5-p19 omega-gliadin of AAG17702 (p81-p102)	Immunogenic	Native			
	DQ2	62 15 QQPQPFPQPQPQPF					
201	omega-gliadin	DQ2-omega-1 omega-Glia (p102–p118)	Immunogenic	Native	DQ2	57	
	17	QPQPFPQPQPFPWQP					
202	omega-gliadin	DQ2-omega-1 omega-Glia (p102–p118; E104)	Immunogenic	Deamidated			
	DQ2	57 17 QPEQPFPQPQPFPWQP					
203	omega-gliadin	Wheat peptide W03, W19, B01	Immunogenic	Deamidated	DQ2		
	62,86	17 QPEQPFPQPQPFPWQP					
204	omega-gliadin	omega-Glia 17mer	Immunogenic	Deamidated	DQ2.5	61	17
		QPQPFPQPQPFPWQP					
205	omega-gliadin	omega-gliadin AAG17702 substituted by Lysine (p89-p102; E95 K89)	Immunogenic	Native	DQ2	62	14
		KPFPQPEQPFPWQP					

206	omega-gliadin	omega-gliadin AAG17702 substituted by Lysine (p89-p102; E95 K90)							
	Immunogenic	Native	DQ2	62	14	QKFPQPEQFPWQP			
207	omega-gliadin	omega-gliadin AAG17702 substituted by Lysine (p89-p102; E95 K91)							
	Immunogenic	Native	DQ2	62	14	QPKPQPEQFPWQP			
208	omega-gliadin	omega-gliadin AAG17702 substituted by Lysine (p89-p102; E95 K99)							
	Immunogenic	Native	DQ2	62	14	QFPQPEQPFKWQP			
209	omega-gliadin	omega-gliadin AAG17702 substituted by Lysine (p89-p102; E95 K100)							
	Immunogenic	Native	DQ2	62	14	QFPQPEQPFKQP			
210	omega-gliadin	omega-gliadin AAG17702 substituted by Lysine (p89-p102; E95 K101)							
	Immunogenic	Native	DQ2	62	14	QFPQPEQFPWKP			
211	omega-gliadin	omega-gliadin AAG17702 substituted by Lysine (p89-p102; E95 K102)							
	Immunogenic	Native	DQ2	62	14	QFPQPEQFPWQK			
212	omega-gliadin	p6-p20 omega-gliadin of AAG17702 (p81-p102)	Immunogenic	Native					
	DQ2	62	15	QPQQPFPQPQFPW					
213	omega-gliadin	p7-p21 omega-gliadin of AAG17702 (p81-p102)	Immunogenic	Native					
	DQ2	62	15	PQQPFPQPQFPWQ					
214	omega-gliadin	p8-p22 omega-gliadin of AAG17702 (p81-p102), Wheat peptide W3							
	Immunogenic	Native	DQ2	62	15	QQPFPQPQFPWQP			
215	omega-gliadin	Wheat peptide W03	Immunogenic	Deamidated	DQ2	62	15		
		EQPFPQPEQFPWQP							
216	omega-gliadin	Wheat peptide W03, W19, Barley peptide B01	Immunogenic	Native					
	DQ2	62	20	QFPQPQFPWQPQFPQ					
217	omega-gliadin	Wheat peptide W03, W19, Barley peptide B01	Immunogenic	Deamidated					
	DQ2	62	20	QFPQPEQFPWQPQFPQ					
218	omega-gliadin	Wheat peptide W03, Barley peptide B01	Immunogenic	Native					
	DQ2	62	12	QFPQPQFPW					
219	omega-gliadin	Wheat peptide W03, Barley peptide B01	Immunogenic	Deamidated					
	DQ2	62	12	QFPQPEQFPW					
220	omega-II gliadin	DQ2.5_glia_omega 2	Immunogenic	Deamidated	DQ2.5	88,90,62			
		9 PQPEQFPW							
221	omega-II gliadin	DQ2.5_glia_omega 2	Immunogenic	Native	DQ2	88,90,62	9		
		PQPQFPW							
222	omega-gliadin	Wheat peptide W19, Barley peptide B19	Immunogenic	Native					
	DQ2	62	12	FPWQPQFPQ					
223	omega-gliadin	Wheat peptide W19	Immunogenic	Deamidated	DQ2	62	12		
		FPWQPEQFPQ							
224	omega-gliadin	Wheat peptide W30	Immunogenic	Native	DQ2	62	20		
		PLQPQPFPQPQFPQ							
225	omega-gliadin	omega-gliadin	Immunogenic	Native	DQ2	62	20		
		FPQPQPFPQPQLPFPQS							
226	omega-gliadin	Wheat peptide W06	Immunogenic	Native	DQ2	62	20		
		QQQPFPQPQLPFPQSEQ							
227	omega-gliadin	Wheat peptide W06	Immunogenic	Native	DQ2	62	12		
		QFPQPQLPFPQ							
228	omega-gliadin	Gliadin AAG17702 p173-p192	Immunogenic	Native	DQ8	54	20		
		QQPFPQPQFPQPQPIP							
229	omega-gliadin	Wheat peptide W32, Barley peptide B25, Rye peptide R26							
	Immunogenic	Native	DQ2	62	12	FPQPQPQFPQ			
230	omega-gliadin	Wheat peptide W04	Immunogenic	Native	DQ2	62	20		
		PQPQPFPQPQPIVQPQ							

231	omega-gliadin	Wheat peptide W04	Immunogenic	Native	DQ2	62	11	
	PFPQPQQPIPV							
232	omega-gliadin	Gliadin AAG17702 p186-p205	Immunogenic	Native	DQ8	54	20	
	QPQQPIPVQPQQSFPQQSQQ							
233	omega-gliadin	Wheat peptide W20	Immunogenic	Native	DQ2	62	20	
	FPELQQPIPQQPQQPFPLQP							
234	omega-gliadin	Wheat peptide W20	Immunogenic	Native	DQ2	62	12	
	PIPQQPQQPFPL							
235	omega-gliadin	Gliadin AAG17702 p225-p244	Immunogenic	Native	DQ8	54	20	
	PQQPQQPFPLQPQQPFPPQQP							
236	omega-gliadin	Wheat peptide W26, Barley peptide B20	Immunogenic	Native	DQ2	62	12	
	PFPLQPQQPFPPQ							
237	omega-gliadin	Gliadin AAG17702 p239-p258	Immunogenic	Native	DQ8	54	20	
	PFPQQPQQPFPPQQPQQSFPQ							
246	omega5-gliadin/LMW glutenin	Glu-5 peptide epitope in native form	Immunogenic	Native	DQ2	21	12	
	QQQQIPQQPQQF							
247	omega5-gliadin/LMW glutenin	Glu-5 peptide epitope in native form	Immunogenic	Native	DQ2	21	12	
	QQQQLPQQPQQF							
248	omega5-gliadin/LMW glutenin	Glu-5 peptide epitope in Deamidated form	Immunogenic	Deamidated	DQ2	21	12	
	QEQQIPEQPQQF							
249	omega5-gliadin/LMW glutenin	Glu-5 peptide epitope in Deamidated form	Immunogenic	Deamidated	DQ2	21	12	
	QEQQLPEQPQQF							
252	omega5-gliadin/LMW glutenin	CAUTION 100% matches with 4 fungal proteins but multiple epitopes on Triticum Glu-5 minimal epitope in native form	Immunogenic	Native	DQ2	19	9	
	QIPQQPQQF							
253	omega5-gliadin/LMW glutenin	Glu-5 minimal epitope in Deamidated form	Immunogenic	Deamidated	DQ2	19	9	
	QIPEQPQQF							
254	omega5-gliadin/LMW glutenin	CAUTION 100% match with fungal and parasite proteins, less with Glu-5 minimal epitope in native form	Immunogenic	Native	DQ2	19	9	
	QLPQQPQQF							
255	omega5-gliadin/LMW glutenin	Glu-5 minimal epitope in Deamidated form	Immunogenic	Deamidated	DQ2	19	9	
	QLPEQPQQF							
256	omega5-gliadin/LMW glutenin	Glu-5 minimal epitope in Deamidated form	Immunogenic	Deamidated	DQ2 (DQ2.2 and DQ2.5)	44	9	
	EIPEQPQQF							
257	omega5-gliadin/LMW glutenin	Glu-5 minimal epitope in Deamidated form	Immunogenic	Deamidated	DQ2 (DQ2.2 and DQ2.5)	44	9	
	ELPEQPQQF							
258	gamma-gliadin or LMW glutenin	Glu-5	Immunogenic	Native	DQ2	19	21	
	QQISQPQIPQQQQIPQQPQQF							
259	gamma-gliadin or LMW glutenin	Glu-5 in Deamidated form	Immunogenic	Deamidated	DQ2	19	21	
	QEISQPQIPQQQQIPQQPQQF							
260	gamma-gliadin or LMW glutenin	Glu-5 in Deamidated form	Immunogenic	Deamidated	DQ2	19	21	
	QQISQPEIPQQQQIPQQPQQF							
261	gamma-gliadin or LMW glutenin	Glu-5 in Deamidated form	Immunogenic	Deamidated	DQ2	19	21	
	QQISQPQIPQEQQIPQQPQQF							
262	gamma-gliadin or LMW glutenin	Glu-5 in Deamidated form	Immunogenic	Deamidated	DQ2	19	21	
	QQISQPQIPQQQEIPQQPQQF							
263	gamma-gliadin or LMW glutenin	Glu-5 in Deamidated form	Immunogenic	Deamidated	DQ2	19	21	
	QEISQPEIPQQQQIPQQPQQF							
264	gamma-gliadin or LMW glutenin	Glu-5 in Deamidated form	Immunogenic	Deamidated	DQ2	19	21	
	QEISQPQIPQEQQIPQQPQQF							

265	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPQIPQQQEIPQQPQQF		
266	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPEIPQEQQIPQQPQQF		
267	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPEIPQQQEIPQQPQQF		
268	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPQIPQEQQEIPQQPQQF		
269	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPEIPQEQQIPQQPQQF		
270	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPEIPQQQEIPQQPQQF		
271	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPQIPQEQQEIPQQPQQF		
272	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPEIPQEQQEIPQQPQQF		
273	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPEIPQEQQEIPQQPQQF		
274	gamma-gliadin or LMW gluteninGlu-5 Immunogenic Native	DQ2 19 21	
	QQISQPQLPQQQIPQQPQQF		
275	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPQLPQQQIPQQPQQF		
276	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPELPQQQIPQQPQQF		
277	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPQLPQEQQIPQQPQQF		
278	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPQLPQQQEIPQQPQQF		
279	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPELPQQQIPQQPQQF		
280	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPQLPQEQQIPQQPQQF		
281	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPQLPQQQEIPQQPQQF		
282	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPELPQEQQIPQQPQQF		
283	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPELPQQQEIPQQPQQF		
284	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPQLPQEQQEIPQQPQQF		
285	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPELPQEQQIPQQPQQF		
286	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPELPQQQEIPQQPQQF		
287	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPQLPQEQQEIPQQPQQF		
288	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPELPQEQQEIPQQPQQF		
289	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPELPQEQQEIPQQPQQF		

290	gamma-gliadin or LMW gluteninGlu-5	Immunogenic	Native	DQ2	19	21
	QQISQPQIPQQQQLPQQPQQF					
291	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QEISQPQIPQQQQLPQQPQQF					
292	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QQISQPEIPQQQQLPQQPQQF					
293	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QQISQPQIPQEEQQLPQQPQQF					
294	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QQISQPQIPQQQQLPQQPQQF					
295	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QEISQPEIPQQQQLPQQPQQF					
296	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QEISQPQIPQEEQQLPQQPQQF					
297	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QEISQPQIPQQQQLPQQPQQF					
298	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QQISQPEIPQEEQQLPQQPQQF					
299	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QQISQPEIPQQQQLPQQPQQF					
300	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QQISQPQIPQEEQQLPQQPQQF					
301	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QEISQPEIPQEEQQLPQQPQQF					
302	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QEISQPEIPQQQQLPQQPQQF					
303	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QEISQPQIPQEEQQLPQQPQQF					
304	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QQISQPEIPQEEQQLPQQPQQF					
305	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QEISQPEIPQEEQQLPQQPQQF					
306	gamma-gliadin or LMW gluteninGlu-5	Immunogenic	Native	DQ2	19	21
	QQLSQPQIPQQQQIPQQPQQF					
307	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QELSQPQIPQQQQIPQQPQQF					
308	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QQLSQPEIPQQQQIPQQPQQF					
309	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QQLSQPQIPQEEQQIPQQPQQF					
310	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QQLSQPQIPQQQEIPQQPQQF					
311	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QELSQPEIPQQQQIPQQPQQF					
312	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QELSQPQIPQEEQQIPQQPQQF					
313	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QELSQPQIPQQQEIPQQPQQF					
314	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic				
	DQ2 19 21 QQLSQPEIPQEEQQIPQQPQQF					

315	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPEIPQQQEIPQQPQQF		
316	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPQIPQEQEIPQQPQQF		
317	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPEIPQEQQIPQQPQQF		
318	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPEIPQQQEIPQQPQQF		
319	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPQIPQEQEIPQQPQQF		
320	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPEIPQEQEIPQQPQQF		
321	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPEIPQEQEIPQQPQQF		
322	gamma-gliadin or LMW gluteninGlu-5 Immunogenic Native	DQ2 19 21	
	QQLSQPQLPQQQIPQQPQQF		
323	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPQLPQQQIPQQPQQF		
324	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPELPQQQIPQQPQQF		
325	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPQLPQEQQIPQQPQQF		
326	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPQLPQQQEIPQQPQQF		
327	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPELPQQQIPQQPQQF		
328	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPQLPQEQQIPQQPQQF		
329	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPQLPQQQEIPQQPQQF		
330	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPELPQEQQIPQQPQQF		
331	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPELPQQQEIPQQPQQF		
332	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPQLPQEQEIPQQPQQF		
333	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPELPQEQQIPQQPQQF		
334	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPELPQQQEIPQQPQQF		
335	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPQLPQEQEIPQQPQQF		
336	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPELPQEQEIPQQPQQF		
337	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPELPQEQEIPQQPQQF		
338	gamma-gliadin or LMW gluteninGlu-5 Immunogenic Native	DQ2 19 21	
	QQLSQPQIPQQQLPQQPQQF		
339	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPQIPQQQLPQQPQQF		

340	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPEIPQQQLPQQPQQF		
341	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPQIPQEQQLPQQPQQF		
342	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPQIPQQQELPQQPQQF		
343	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPEIPQQQLPQQPQQF		
344	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPQIPQEQQLPQQPQQF		
345	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPQIPQQQELPQQPQQF		
346	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPEIPQEQQLPQQPQQF		
347	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPEIPQQQELPQQPQQF		
348	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPQIPQEQELPQQPQQF		
349	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPEIPQEQQLPQQPQQF		
350	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPEIPQQQELPQQPQQF		
351	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPQIPQEQELPQQPQQF		
352	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPEIPQEQELPQQPQQF		
353	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPEIPQEQELPQQPQQF		
354	gamma-gliadin or LMW gluteninGlu-5 Immunogenic Native	DQ2 19 21	
	QQISQPQLPQQQLPQQPQQF		
355	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPQLPQQQLPQQPQQF		
356	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPELPQQQLPQQPQQF		
357	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPQLPQEQQLPQQPQQF		
358	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPQLPQQQELPQQPQQF		
359	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPELPQQQLPQQPQQF		
360	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPQLPQEQQLPQQPQQF		
361	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPQLPQQQELPQQPQQF		
362	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPELPQEQQLPQQPQQF		
363	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPELPQQQELPQQPQQF		
364	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPQLPQEQELPQQPQQF		

365	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPELPQEQQLPQQPQQF		
366	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPELPQQQQLPQQPQQF		
367	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPQLPQEQQLPQQPQQF		
368	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQISQPELPQEQQLPQQPQQF		
369	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QEISQPELPQEQQLPQQPQQF		
370	gamma-gliadin or LMW gluteninGlu-5 Immunogenic Native	DQ2 19 21	
	QQLSQPQLPQQQLPQQPQQF		
371	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPQLPQQQLPQQPQQF		
372	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPELPQQQLPQQPQQF		
373	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPQLPQEQQLPQQPQQF		
374	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPQLPQQQLPQQPQQF		
375	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPELPQQQLPQQPQQF		
376	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPQLPQEQQLPQQPQQF		
377	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPQLPQQQLPQQPQQF		
378	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPELPQEQQLPQQPQQF		
379	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPELPQQQLPQQPQQF		
380	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPQLPQEQQLPQQPQQF		
381	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPELPQEQQLPQQPQQF		
382	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPELPQQQLPQQPQQF		
383	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPQLPQEQQLPQQPQQF		
384	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QQLSQPELPQEQQLPQQPQQF		
385	gamma-gliadin or LMW gluteninGlu-5 in Deamidated form	Immunogenic	Deamidated
	DQ2 19 21 QELSQPELPQEQQLPQQPQQF		
386	gamma-gliadin gamma-gliadin P08079 Immunogenic Native	DQ8 54 20	
	QQFLQPQQPFPQQPQQPYPQ		
387	gamma-gliadin gamma-gliadin P08079 Immunogenic Native	DQ8 54 17	
	QQFLQPQQPFPQQPQQP		
388	gamma-gliadin gamma-5 gliadin (p59-p84) Immunogenic	Native DQ2 48 26	
	FLQPQQPFPQQPQQPYPQQPQQPFPQ		
389	gamma-gliadin gamma-5 gliadin (p59-p84; E63) Immunogenic	Deamidated DQ2 48	
	26 FLQPEQFPQQPQQPYPQQPQQPFPQ		

390	gamma-gliadin	gamma-5 gliadin (p59-p84; E68)	Immunogenic	Deamidated	DQ2	48
	26	FLQPQQPFPEQPQQPYPQQPQQPFPQ				
391	gamma-gliadin	gamma-5 gliadin (p59-p84; E71)	Immunogenic	Deamidated	DQ2	48
	26	FLQPQQPFPPQQPEQPYPQQPQQPFPQ				
392	gamma-gliadin	gamma-5 gliadin (p59-p84; E76)	Immunogenic	Deamidated	DQ2	48
	26	FLQPQQPFPPQQPQQPYPEQPQQPFPQ				
393	gamma-gliadin	gamma-5 gliadin (p59-p84; E79)	Immunogenic	Deamidated	DQ2	48
	26	FLQPQQPFPPQQPQQPYPQQPEQPFPQ				
394	gamma-gliadin	gamma-5 gliadin (p59-p84; E63 and E68)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPEQPFPEQPQQPYPQQPQQPFPQ				
395	gamma-gliadin	gamma-5 gliadin (p59-p84; E63 and E71)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPEQPFPPQQPEQPYPQQPQQPFPQ				
396	gamma-gliadin	gamma-5 gliadin (p59-p84; E63 and E76)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPEQPFPPQQPQQPYPEQPQQPFPQ				
397	gamma-gliadin	gamma-5 gliadin (p59-p84; E63 and E79)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPEQPFPPQQPQQPYPQQPEQPFPQ				
398	gamma-gliadin	gamma-5 gliadin (p59-p84; E68 and E71)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPQQPFPEQPEQPYPQQPQQPFPQ				
399	gamma-gliadin	gamma-5 gliadin (p59-p84; E68 and E76)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPQQPFPEQPQQPYPEQPQQPFPQ				
400	gamma-gliadin	gamma-5 gliadin (p59-p84; E68 and E79)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPQQPFPEQPQQPYPQQPEQPFPQ				
401	gamma-gliadin	gamma-5 gliadin (p59-p84; E71 and E76)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPQQPFPPQQPEQPYPQQPQQPFPQ				
402	gamma-gliadin	gamma-5 gliadin (p59-p84; E71 and E79)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPQQPFPPQQPEQPYPQQPEQPFPQ				
403	gamma-gliadin	gamma-5 gliadin (p59-p84; E76 and E79)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPQQPFPPQQPQQPYPEQPEQPFPQ				
404	gamma-gliadin	gamma-5 gliadin (p59-p84; E63, E68 and E71)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPEQPFPEQPEQPYPQQPQQPFPQ				
405	gamma-gliadin	gamma-5 gliadin (p59-p84; E63, E68 and E76)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPEQPFPEQPQQPYPEQPQQPFPQ				
406	gamma-gliadin	gamma-5 gliadin (p59-p84; E63, E68 and E79)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPEQPFPEQPQQPYPQQPEQPFPQ				
407	gamma-gliadin	gamma-5 gliadin (p59-p84; E68, E71 and E76)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPQQPFPEQPEQPYPQQPQQPFPQ				
408	gamma-gliadin	gamma-5 gliadin (p59-p84; E68, E71 and E79)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPQQPFPEQPEQPYPQQPEQPFPQ				
409	gamma-gliadin	gamma-5 gliadin (p59-p84; E71, E76 and E79)	Immunogenic	Deamidated		
	DQ2 48	26 FLQPQQPFPPQQPEQPYPQQPEQPFPQ				
410	gamma-gliadin	gamma-5 gliadin (p59-p84; E63, E68, E71, E76 and E79)	Immunogenic			
	Deamidated	DQ2 48 26 FLQPEQPFPEQPEQPYPQQPEQPFPQ				
411	gamma-gliadin	gamma-5 gliadin (p60-p79) ;	Immunogenic	Native	DQ2	57,23,48,2 20
	gamma-3 and gamma-5 peptide 1317	LQPQQPFPPQQPQQPYPQQPQ				
412	gamma-gliadin	DQ2-gamma-V gamma-Glia (p78 –p97; E81)	Immunogenic	Deamidated		
	DQ2 57,23,2 20	LQPEQPFPPQQPQQPYPQQPQ				
413	gamma-gliadin	DQ2-gamma-V gamma-Glia (p78 –p97; E86)	Immunogenic	Deamidated		
	DQ2 57,23,2 20	LQPQQPFPEQPQQPYPQQPQ				
414	gamma-gliadin	DQ2-gamma-V gamma-Glia (p78 –p97; E89)	Immunogenic	Deamidated		
	DQ2 57,23,2 20	LQPQQPFPPQQPEQPYPQQPQ				

415	gamma-gliadin	DQ2-gamma-V	gamma-Glia (p78 –p97; E94)	Immunogenic	Deamidated			
	DQ2	57,23,2	20	LQPQQPFPPQQPQQPYPEQPQ				
416	gamma-gliadin	DQ2-gamma-V	gamma-Glia (p78 –p97; E81 and E86)	Immunogenic				
	Deamidated	DQ2	57,23,2	20	LQPEQPFPEQPQQPYPPQQPQ			
417	gamma-gliadin	DQ2-gamma-V	gamma-Glia (p78 –p97; E81 and E89)	Immunogenic				
	Deamidated	DQ2	57,23,2	20	LQPEQPFPPQQPEQPYPQQPQ			
418	gamma-gliadin	DQ2-gamma-V	gamma-Glia (p78 –p97; E81 and E94)	Immunogenic				
	Deamidated	DQ2	57,23,2	20	LQPEQPFPPQQPQQPYPEQPQ			
419	gamma-gliadin	DQ2-gamma-V	gamma-Glia (p78 –p97; E86 and E89)	Immunogenic				
	Deamidated	DQ2	57,23,2	20	LQPQQPFPEQPEQPYPQQPQ			
420	gamma-gliadin	DQ2-gamma-V	gamma-Glia (p78 –p97; E86 and E64)	Immunogenic				
	Deamidated	DQ2	57,23,2	20	LQPQQPFPEQPQQPYPEQPQ			
421	gamma-gliadin	DQ2-gamma-V	gamma-Glia (p78 –p97; E89 and E94)	Immunogenic				
	Deamidated	DQ2	57,23,2	20	LQPQQPFPPQQPEQPYPPEQPQ			
422	gamma-gliadin	DQ2-gamma-V	gamma-Glia (p78 –p97; E81, E86 and E89)					
	Immunogenic	Deamidated	DQ2	57,23,2	20	LQPEQPFPEQPEQPYPQQPQ		
423	gamma-gliadin	DQ2-gamma-V	gamma-Glia (p78 –p97; E81, E86 and E94)					
	Immunogenic	Deamidated	DQ2	57,23,2	20	LQPEQPFPEQPQQPYPEQPQ		
424	gamma-gliadin	DQ2-gamma-V	gamma-Glia (p78 –p97; E86, E89 and E94)					
	Immunogenic	Deamidated	DQ2	57,23,2	20	LQPQQPFPEQPEQPYPPEQPQ		
425	gamma-gliadin	DQ2-gamma-V	gamma-Glia (p78 –p97; E81, E86, E89 and E94)					
	Immunogenic	Deamidated	DQ2	57,23,2	20	LQPEQPFPEQPEQPYPPEQPQ		
426	gamma-gliadin	gamma3/gamma4	Immunogenic	Native	DQ2	25	17	
	PQQPFPPQQPQQPYPPQQP							
427	gamma-gliadin	gamma5 (p62-p74)	Immunogenic	Native	DQ2	25	13	
	PQQPFPPQQPQQPY							
428	gamma-gliadin	gamma5 (p62-p74; E68)	Immunogenic	Deamidated	DQ2	25	13	
	PQQPFPEQPQQPY							
429	gamma-gliadin	gamma5 (p62-p74; E63 and E68)		Immunogenic	Deamidated			
	DQ2	25	13	PEQPFPEQPQQPY				
430	gamma-gliadin	gamma5 (p62-p74; E68 and E71)		Immunogenic	Deamidated			
	DQ2	25	13	PQQPFPEQPEQPYP				
431	gamma-gliadin	gamma5 (p62-p74; E63, E68 and E71)		Immunogenic	Deamidated			
	DQ2	25	13	PEQPFPEQPEQPYP				
432	gamma-gliadin	gamma-5 gliadin (p62-p72)	Immunogenic	Native	DQ2	47,48	11	
	PQQPFPPQQPQQ							
433	gamma-gliadin	gamma5-gliadin (p62-p72; E68)	Immunogenic	Deamidated	DQ2	47		
	11	PQQPFPEQPQQ						
434	gamma-gliadin	gamma5 (p62-p72; E68, E63 and E71)	Immunogenic	Deamidated				
	DQ2	25	11	PEQPFPEQPEQ				
435	gamma-gliadin	gamma23mer	Immunogenic	Native	DQ2.5	61	23	
	QQPFPPQQPQQPYPPQQPQQPFPPQ							
436	gamma-gliadin	gamma23mer (in considered Deamidated form)	Immunogenic	Deamidated				
	DQ2.5	61	23	EQPFPEQPEQPYPPEQPEQPFPQP				
437	gamma-gliadin	gamma5-gliadin (p60-p79)	Immunogenic	Native	DQ2	21	14	
	QQPFPPQQPQQPYPPQ							
438	gamma-5 gliadin	DQ2.5_glia_gamma 5	Immunogenic	Native	DQ2.5	25,90,23		
	9	QQPFPPQQPQ						
439	gamma-5 gliadin	DQ2.5_glia_gamma 5	Immunogenic	Deamidated	DQ2.5			
	25,90,23	9	QQPFPEQPQ					

440	gamma-gliadin	CAUTION 100% match to Archaea protein lower to others and to gamma5 (p63-p71; E63, E68 and E71)	Immunogenic	Deamidated	DQ2	25	9	EQPFPEQPE
441	gamma-gliadin	DQ2-gamma-III gamma-Glia (p83-p97)	Immunogenic	Native	DQ2	57,48,23,2	15	PFQQPQQPYPPQQPQ
442	gamma-gliadin	DQ2-gamma-III gamma-Glia (p83-p97; E86)	Immunogenic	Deamidated	DQ2	57,48,23,2	15	PFPEQPQQPYPPQQPQ
443	gamma-gliadin	DQ2-gamma-III gamma-Glia (p83-p97; E89)	Immunogenic	Deamidated	DQ2	57,48,23,2	15	PFQQPPEQPYPPQQPQ
444	gamma-gliadin	DQ2-gamma-III gamma-Glia (p83-p97; E86 and E89)	Immunogenic	Deamidated	DQ2	57,48,23,2	15	PFPEQPPEQPYPPQQPQ
445	gamma-gliadin	Wheat peptide W23	Immunogenic	Native	DQ2	62	12	PFQQPQQPYPPQ
446	gamma-gliadin	gamma-gliadin (p66-p80) AJ416339	Immunogenic	Native	DQ8, DQ2	17	15	FPQQPQQPYPPQQPQ
447	gamma-gliadin	gamma-gliadin (p66-p80; E68) AJ416339	Immunogenic	Deamidated	DQ8	17,83	15	FPEQPQQPYPPQQPQ
448	gamma-gliadin	gamma-gliadin (p66-p80; E71) AJ416339	Immunogenic	Deamidated	DQ2	17	15	FPQQPEQPYPPQQPQ
449	gamma-gliadin	gamma-gliadin (p66-p80; E76) AJ416339	Immunogenic	Deamidated	DQ8, DQ2	17	15	FPQQPQQPYPEQPQQ
450	gamma-gliadin	gamma-gliadin (p66-p80; E71 and 76) AJ416339	Immunogenic	Deamidated	DQ8	17	15	FPEQPQQPYPEQPQQ
451	gamma-gliadin	gamma5 (p66-p78)	Immunogenic	Native	DQ2	23	13	FPQQPQQPYPPQQP
452	gamma-gliadin	gamma5 (p66-p78; E68 and E71)	Immunogenic	Deamidated	DQ2	23	13	FPEQPPEQPYPPQQP
453	gamma-gliadin	gamma5 (p66-p78; E68 and E72)	Immunogenic	Deamidated	DQ2	23	13	FPEQPPEQPYPPQQP
454	gamma-gliadin	gamma5 (p66-p77)	Immunogenic	Native	DQ2	25	12	FPQQPQQPYPPQQ
455	gamma-gliadin	gamma5 (p66-p77; E71)	Immunogenic	Deamidated	DQ2	25	12	FPQQPEQPYPPQQ
456	gamma-gliadin	gamma5 (p66-p77; E68, E71 and E76)	Immunogenic	Deamidated	DQ2	25	12	FPEQPPEQPYPEQ
457	gamma-gliadin	gamma5 (p67-p77; E68, E71 and E76)	Immunogenic	Deamidated	DQ2	25	11	PEQPPEQPYPEQ
458	gamma-1 and gamma 5 gliadin	CAUTION 100% matches to many fungal and parasite proteins as well as DQ2.5_glia_gamma 3						
	DQ8_glia_gamma 1b	Immunogenic	Native	DQ2.5/DQ8	17,23,78	9		QQPQQPYPPQ
459	gamma-5 gliadin	DQ2.5_glia_gamma 3						
	DQ8_glia_gamma1b	Immunogenic	Deamidated	DQ2.5/DQ8	25,17,78	9		EQPEQPYPE
460	gamma-1 gliadin	DQ2.5_glia_gamma 3						
	DQ8_glia_gamma 1b	Immunogenic	Deamidated	DQ2.5/DQ8	25,17,78	9		EQPQQPYPE
461	gamma-1 gliadin	DQ2.5_glia_gamma 3						
	DQ8_glia_gamma 1b	Immunogenic	Deamidated	DQ2.5/DQ8	17,78	9		EQPQQPFPE
462	gamma-III gliadin	CAUTION 100% identity matches to fungal and lower to other fungal proteins as DQ2.5_glia_gamma 3						

DQ8_glia_gamma 1b	Immunogenic	Deamidated	DQ2.5/DQ8	17,78	9	QQPEQPYPQ
463	gamma-gliadin	Predicted gamma-gliadin peptide	Immunogenic	Native	DQ8	
(DQ2/8)	22	14	QQYPQQPQQPFPQ			
464	gamma-gliadin	CAUTION many 100% identity matches to legume and bacterial proteins and also gamma-Vib gliadin	Immunogenic	Native	DQ2	25 9
465	gamma-gliadin	CAUTION 100% match to bacterial protein and lower to others and gamma-Vib gliadin in Deamidated form	Immunogenic	Deamidated	DQ2	25 9
						EQYPQQPQ
466	gamma-gliadin	CAUTION 100% identity to bacterial protein and lower to others and gamma-Vib gliadin in Deamidated form	Immunogenic	Deamidated	DQ2	25 9
						QQPYPEQPQ
467	gamma-gliadin	gamma-Vib gliadin in Deamidated form	Immunogenic	Deamidated	DQ2	25 9
						EQPYPEQPQ
468	gamma-gliadin	CAUTION many 100% matches to fish, fungi and other non-wheat family proteins also a few to Secalin Glia-gamma2	Immunogenic	Native	DQ2 (DQ2.2 and DQ2.5)	44 9
						PYPQQPQQP
469	gamma-gliadin	Glia-gamma2 in Deamidated form	Immunogenic	Deamidated	DQ2 (DQ2.2 and DQ2.5)	44,83 9
						PYPEQPQQP
470	gamma-gliadin	Glia-gamma2 in Deamidated form	Immunogenic	Deamidated	DQ2 (DQ2.2 and DQ2.5)	44 9
						PYPQQPEQP
471	gamma-gliadin	CAUTION 100% match to bacterial protein and lizard protein lower to grapes and wheat Glia-gamma2 in Deamidated form	Immunogenic	Deamidated	DQ2 (DQ2.2 and DQ2.5)	44 9
						PYPEQEQP
472	gamma-gliadin	CAUTION 100% 4 matches to Candida 2 to bacteria and many to secalins and wheat gamma2-gliadin	Immunogenic	Native	DQ2.5/DQ8	90,17,8 9
						QQPQQPFPQ
473	gamma-2 gliadin	CAUTION 100% match to fungal protein and lower to Candida and to Gliadin epitope: gamma-I	Immunogenic	Deamidated	DQ2.5/DQ8	90,17,8 9
						EQPQQPFPQ
474	gamma-2 gliadin	Gliadin epitope: gamma-VII	Immunogenic	Deamidated	DQ2.5/DQ8	90,17,8 9
						QQPEQPFPQ
475	gamma-2 gliadin	CAUTION 100% match to a bacterial protein / lower to triticum gammaVII-gliadin in Deamidated form	Immunogenic	Deamidated	DQ2.5/DQ8	25,90,17 9
						EQPEQPFPQ
476	gamma-gliadin	gamma-Glia (p105-p118)	Immunogenic	Native	DQ2	57 14
						PQQQLPQQPAQL
477	gamma-gliadin	gamma-Glia (p105-p118; E113)	Immunogenic	Deamidated	DQ2	57 14
						PQQQLQPEQPAQL
478	gamma1-gliadinWheat peptide W37		Immunogenic	Native	DQ2	62 20
						ATANMQVDPGQVQWPQQP
479	gamma1-gliadinWheat peptide W37		Immunogenic	Native	DQ2	62 12
						QVDPGQVQWPQ
480	gamma1-gliadinalgamma-gliadin 1370 (p1-p30) ; gamma-gliadin M2 M36999 (p11-p30) homologous to DQ2-alpha-I		Immunogenic	Native	DQ2, DQ8	17,23 20
						WPQQQFPQPQQPFCQQPQR
481	gamma1-gliadinalgamma-gliadin 1371 (p21-p40)		Immunogenic	Native	DQ2	17 20
						QQPFCQQPQRTPQPHQTFH
482	gamma1-gliadinalgamma-gliadin 1372 (p31-p50)		Immunogenic	Native	DQ2	17 20
						TIPQPHQTFHHQPQQTFPQP
483	gamma1-gliadinalgamma-gliadin 1372 (p41-p60)		Immunogenic	Native	DQ2, DQ8	17 20
						HQPQQTFPQPQQTYPHQPPQ

484	gamma1-gliadine	gamma-gliadin 1372 (p51-p70)	Immunogenic	Native	DQ2	17	20
		QQTYPHQPPQQFPQTQQPQQ					
485	gamma1-gliadine	gamma-gliadin 1375 (p61-p80) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Native	DQ2, DQ8	25,17,23 20
		QFPQTQQPQQPFPQPQQTFP					
486	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E64) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPETQQPQQPFPQPQQTFP					
487	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E66) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPQTEQPQQPFPQPQQTFP					
488	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E69) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPQTQQPEQPFPQPQQTFP					
489	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E76) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPQTQQPQQPFPQPEQTFP					
490	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E64 and E66) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPETEQPQQPFPQPQQTFP					
491	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E64 and E69) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPETQQPEQPFPQPQQTFP					
492	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E64 and E76) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPETQQPQQPFPQPEQTFP					
493	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E66 and E69) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPQTEQPQQPFPQPQQTFP					
494	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E66 and E76) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPQTEQPQQPFPQPEQTFP					
495	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E69 and E76) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPQTQQPEQPFPQPEQTFP					
496	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E64, E66 and E69) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPETEQPQQPFPQPQQTFP					
497	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E64, E66 and E76) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPETEQPQQPFPQPEQTFP					
498	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E64, E69 and E76) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPETQQPEQPFPQPEQTFP					
499	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E66, E69 and E76) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPQTEQPQQPFPQPEQTFP					
500	gamma1-gliadine	gamma-gliadin 1375 (p61-p80; E64, E66, E69 and E76) ; gamma-gliadin M7 M36999 (p61-p80)	homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25 20
		QFPETEQPQQPFPQPEQTFP					

501	gamma1-gliadinWheat peptide W28, W33 PQQPFPQQQTFPQQPQLPF	Immunogenic	Native	DQ2	62	20
502	gamma1-gliadinalgamma-gliadin 1376 (p71-p90); gamma-gliadin M8 M36999 (71-80) homologous to DQ2-alpha-I and DQ2-gamma-IV PFPQPQQQTFPQQPQLPFPQQ	Immunogenic	Native	DQ2, DQ8	17,23	20
503	gamma1-gliadinWheat peptide W33 PFPQPQQQTFPQ	Immunogenic	Native	DQ2	62	11
504	gamma1-gliadinWheat peptide W28 PQQTFPQQPQLP	Immunogenic	Native	DQ2	62	12
505	gamma1-gliadinWheat peptide W10 SQQPQQQFSQPQQQFPQPQQ	Immunogenic	Native	DQ2	62	20
506	gamma1-gliadinDQ2-y-IV y-Glia (p117-p132) QQFSQPQQQFPQPQQ	Immunogenic	Native	DQ2	57,23	15
507	gamma1-gliadinDQ2-y-IV y-Glia (p117-p132; E123) DQ2 57,23 15 QQFSQPEQQFPQPQQ	Immunogenic	Deamidated			
508	gamma1-gliadinDQ2-y-IV y-Glia (p117-p132; E125) DQ2 57,23 15 QQFSQPQQEFQPQPQQ	Immunogenic	Deamidated			
509	gamma1-gliadinDQ2-y-IV y-Glia (p117-p132; E123 and E125) DQ2 57,23 15 QQFSQPEQEFQPQPQQ	Immunogenic	Deamidated			
510	gamma1-gliadinWheat peptide W10 QQFSQPQQQFPQ	Immunogenic	Native	DQ2	62	12
511	gamma1-gliadinalgamma-IV (p101-p113) QFSQPQQQFPQPQ	Immunogenic	Native	DQ2	23	13
512	gamma1-gliadinalgamma-gliadin (gamma5 p102-p113) 12 FSQPQQQFPQPQ	Immunogenic	Native	DQ2	23	
513	gamma1-gliadinalgamma5 (p102-p113; E106) 12 FSQPEQQFPQPQ	Immunogenic	Deamidated	DQ2	23	
514	gamma1-gliadinalgamma5 (p102-p113; E108) 12 FSQPQQEFQPQPQ	Immunogenic	Deamidated	DQ2	23	
515	gamma1-gliadinalgamma5 (p102-p113; E106 and E108) DQ2 25,23 12 FSQPEQEFQPQPQ	Immunogenic	Deamidated			
516	gamma1-gliadinalgamma5 (p102-p111) FSQPQQQFPQ	Immunogenic	Native	DQ2	25	10
517	gamma1-gliadinalgamma5 (p102-p111; E106) 10 FSQPEQQFPQ	Immunogenic	Deamidated	DQ2	25	
518	gamma1-gliadinalgamma5 (p102-p111; E108) 10 FSQPQQEFQPQ	Immunogenic	Deamidated	DQ2	25	
519	gamma1-gliadinalgamma5 (p102-p111; E106 and E108) DQ2 25 10 FSQPEQEFQPQ	Immunogenic	Deamidated			
520	gamma1-gliadinalgamma-IV (p103-p114) SQPQQQFPQPQQ	Immunogenic	Native	DQ2	23	12
521	gamma-5 gliadin glia-gamma 4a	CAUTION 100% match to fungal protein and many wheat proteins Immunogenic	Native	DQ2.5	90,25,23	9
522	gamma-5 gliadin 9 SQPEQEFQPQ	glia-gamma 4a Immunogenic	Deamidated	DQ2.5	90,25,23	
523	gamma1-gliadinalgamma-gliadin of GDB2_WHEAT (SwissProt P08453 GI:121101) (p140-p150) Immunogenic	Native	DQ2	24	11	QQPQQSFQPQQ
524	gamma1-gliadinalgamma-gliadin of GDB2_WHEAT (SwissProt P08453 GI170738) (p141-p150) Immunogenic	Native	DQ2	24	10	QPQQSFQPQQ
525	gamma1-gliadinalgamma-gliadin of GDB2_WHEAT (SwissProt P08453 GI170738) (p141-p150; E148)	Immunogenic	Deamidated	DQ2	24	10
						QPQQSFPEQQ

526	gamma-gliadin	Wheat peptide W07	Immunogenic	Native	DQ2	62	20
	WPQQQPFPPQPPFCQQPQQ						
527	gamma-gliadin	Wheat peptide W07	Immunogenic	Deamidated	DQ2	62	20
	WPQQQPFPPQPEQPFCCQQPQQ						
529	gamma-gliadin	Wheat peptide W07	Immunogenic	Deamidated	DQ2	62	12
	QPFPPQPEQPFCCQ						
530	gamma-gliadin	Predicted gamma-gliadin	Immunogenic	Native	DQ8 (DQ2/8)	22	
	14	QFPQTQQPQQPFPQ					
531	gamma-gliadin	gamma-gliadin M36999 (p63-p76; E66)	Immunogenic	Synthesised as			
	Deamidated	DQ8 17 14	PQTEQPQQPFPQPQ				
532	gamma-gliadin	gamma-gliadin M36999 (p63-p76; E69)	Immunogenic	Synthesised as			
	Deamidated	DQ2 17 14	PQTQQPEQPFPQPQ				
533	gamma-gliadin	gamma-gliadin M36999 (p63-p76; E66 and E69)	Immunogenic	Synthesised			
	as Deamidated	DQ2, DQ8 17 14	PQTEQPPEQPFPQPQ				
534	gamma-gliadin	gamma-gliadin 1375 (p61-p80) ; gamma-gliadin M7 M36999 (61-80)	Immunogenic	Native	DQ2	25	11
	homologous to DQ2-gamma-III						
535	gamma-gliadin	gamma-gliadin 1375 (p61-p80; E62 and E65), ; gamma-gliadin M7 M36999 (61-80) homologous to DQ2-gamma-III	Immunogenic	Deamidated	DQ2	25	11
	TEQPEQPFPQP						
536	gamma-gliadin	gamma-gliadin 1377 (p81-p100)	Immunogenic	Native	DQ2, DQ8	17	
	20	QQPQLPFPQQPQQPFPQPQQ					
537	gamma-gliadin	gamma-gliadin (p84-p97)	Immunogenic	Native	DQ8 (DQ2/8)	22	
	14	QLPFPQQPQQPFPQ					
538	gamma-gliadin	Glia-gamma2 (p89-p102)	Immunogenic	Native	DQ2	20	10
	PFPQQPQQPF						
539	gamma-gliadin	Glia-gamma2 (p89-p102; E92)	Immunogenic	Deamidated	DQ2	20	
	10	PFPEQPQQPF					
540	gamma-gliadin	Glia-gamma2 (p89-p102; E94)	Immunogenic	Deamidated	DQ2	20	
	10	PFPQQPEQPF					
541	gamma-gliadin	Glia-gamma2 (p89-p102; E92 and E94)	Immunogenic	Deamidated			
	DQ2	20 10	PFPEQPQQPF				
542	gamma-gliadin	CAUTION 100% match to a fungal protein and many Secalins gamma-Gliadin (p90-p102)	Immunogenic	Native	DQ2	27	9
	FPQQPQQPF						
543	gamma-gliadin	gamma-Gliadin (p90-p102; E92)	Immunogenic	Deamidated	DQ2	27	
	9	FPEQPQQPF					
544	gamma-gliadin	gamma-Gliadin (p90-p102; E96)	Immunogenic	Deamidated	DQ2	27	
	9	FPQQPQEPPF					
545	gamma-gliadin	gamma-Gliadin (p90-p102; E92 and E96)	Immunogenic	Deamidated			
	DQ2	27 9	FPEQPQEPPF				
546	gamma-gliadin	gamma-gliadin 1378 (p91-p110), ; gamma-gliadin M10 M36999 (91-110)	Immunogenic	Native	DQ2, DQ8	17,23	20
	homologous to DQ2-alpha-I						
	PQQPFPQQPQQPFPQSQQ						
547	gamma-gliadin	Wheat peptide W36	Immunogenic	Native	DQ2	62	20
	QQPAQYEVIRSLVRLTPNM						
548	gamma-gliadin	Wheat peptide W36	Immunogenic	Native	DQ2	62	16
	QYEVIRSLVRLTPNM						
549	gamma-gliadin	Wheat peptide W36	Immunogenic	Deamidated	DQ2	62	15
	EYEVIRSLVRLTPN						
550	gamma-gliadin	Wheat peptide W36	Immunogenic	Native	DQ2	62	15
	QYQVIRSLVRLTPN						

551	gamma-gliadin	gamma-gliadin AAK84778 (p74-p93)	Immunogenic	Native	DQ8	54
		20 QQQFIQPQQPFPQQPQQTYP				
552	gamma-gliadin	Wheat peptide W14	Immunogenic	Native	DQ2	62 12
		QQFIQPQQPFPQ				
553	gamma-gliadin	Predicted gamma-gliadin	Immunogenic	Native	DQ8 (DQ2/8)	22
		14 PFPQTQQPQQPFPQ				
554	gamma-gliadin	gamma-gliadin 1379 (p101-p120)	Immunogenic	Native	DQ2, DQ8	
		17 20 PQQPFPQSQQPQQPFPQPQQ				
555	gamma-gliadin	Predicted gamma-gliadin	Immunogenic	Native	DQ8 (DQ2/8)	22
		14 PFPQSQQPQQPFPQ				
556	gamma-gliadin	Wheat peptide W16	Immunogenic	Native	DQ2	62 20
		SQQPQQPFPQPQQQFPQPQQ				
557	gamma-gliadin	gamma-gliadin 1380 (p111-p130) ; gamma-gliadin M12 M36999 (111-130)	Immunogenic	Native	DQ2	25,17,23 20
		homologous to DQ2-gamma-IV				
		PQQPFPQPQQQFPQPQQPQQ				
558	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E112) ; gamma-gliadin M12 M36999 (111-130)	Immunogenic	Deamidated	DQ2	25 20
		homologous to DQ2-gamma-IV				
		PEQFPFPQPQQQFPQPQQPQQ				
559	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E119) ; gamma-gliadin M12 M36999 (111-130)	Immunogenic	Deamidated	DQ2	25 20
		homologous to DQ2-gamma-IV				
		PQQPFPQPEQQFPQPQQPQQ				
560	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E121) ; gamma-gliadin M12 M36999 (111-130)	Immunogenic	Deamidated	DQ2	25 20
		homologous to DQ2-gamma-IV				
		PQQPFPQPQQEFQPQPQQPQQ				
561	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E126) ; gamma-gliadin M12 M36999 (111-130)	Immunogenic	Deamidated	DQ2	25 20
		homologous to DQ2-gamma-IV				
		PQQPFPQPQQQFPQPEQPQQ				
562	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E112 and E119) ; gamma-gliadin M12 M36999 (111-130)	Immunogenic	Deamidated	DQ2	25
		homologous to DQ2-gamma-IV				
		20 PEQFPFPQPEQQFPQPQQPQQ				
563	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E112 and E121) ; gamma-gliadin M12 M36999 (111-130)	Immunogenic	Deamidated	DQ2	25
		homologous to DQ2-gamma-IV				
		20 PEQFPFPQPQQEFQPQPQQPQQ				
564	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E112 and E126) ; gamma-gliadin M12 M36999 (111-130)	Immunogenic	Deamidated	DQ2	25
		homologous to DQ2-gamma-IV				
		20 PEQFPFPQPQQQFPQPEQPQQ				
565	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E119 and E121) ; gamma-gliadin M12 M36999 (111-130)	Immunogenic	Deamidated	DQ2	25
		homologous to DQ2-gamma-IV				
		20 PQQPFPQPEQEFQPQPQQPQQ				
566	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E119 and E126) ; gamma-gliadin M12 M36999 (111-130)	Immunogenic	Deamidated	DQ2	25
		homologous to DQ2-gamma-IV				
		20 PQQPFPQPEQQFPQPEQPQQ				
567	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E121 and E126) ; gamma-gliadin M12 M36999 (111-130)	Immunogenic	Deamidated	DQ2	25
		homologous to DQ2-gamma-IV				
		20 PQQPFPQPQQEFQPQPEQPQQ				
568	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E112, E119 and E121) ; gamma-gliadin M12 M36999 (111-130)	Immunogenic	Deamidated	DQ2	25
		homologous to DQ2-gamma-IV				
		20 PEQFPFPQPEQEFQPQPQQPQQ				
569	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E112, E119 and E126) ; gamma-gliadin M12 M36999 (111-130)	Immunogenic	Deamidated	DQ2	25
		homologous to DQ2-gamma-IV				
		20 PEQFPFPQPEQQFPQPEQPQQ				

570	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E112, E121 and E126) ; gamma-gliadin M12 M36999 (111-130) homologous to DQ2-gamma-IV	Immunogenic	Deamidated	DQ2	25	20	PEQPFPPQPEQFPQPEQPQQ
571	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E119, E121 and E126) ; gamma-gliadin M12 M36999 (111-130) homologous to DQ2-gamma-IV	Immunogenic	Deamidated	DQ2	25	20	PQQPFPPQPEQFPQPEQPQQ
572	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E112, E119, E121 and E126) ; gamma-gliadin M12 M36999 (111-130) homologous to DQ2-gamma-IV	Immunogenic	Deamidated	DQ2	25	20	PEQPFPPQPEQFPQPEQPQQ
573	gamma-gliadin	gamma-gliadin 1380 (p111-p130) ; gamma-gliadin M12 M36999 (111-130) homologous to DQ2-gamma-IV ; W16	Immunogenic	Native	DQ2	62,25	12	FPQPQQQFPQPQ
574	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E115) ; gamma-gliadin M12 M36999 (111-130) homologous to DQ2-gamma-IV	Immunogenic	Deamidated	DQ2	25	12	FPQPEQQFPQPQ
575	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E117) ; gamma-gliadin M12 M36999 (111-130) homologous to DQ2-gamma-IV	Immunogenic	Deamidated	DQ2	25	12	FPQPQQEFPPQPQ
576	gamma-gliadin	gamma-gliadin 1380 (p111-p130; E115 and E117) ; gamma-gliadin M12 M36999 (111-130) homologous to DQ2-gamma-IV	Immunogenic	Deamidated	DQ2	25	12	FPQPEQEFPPQPQ
577	gamma-gliadin	CAUTION 100% matches to 3 fungal and metazoan proteins and wheat glia-gamma 4b	Immunogenic	Native	DQ2.5	90,25	9	PQPQQQFPQ
578	gamma-gliadin	glia-gamma 4b	Immunogenic	Deamidated	DQ2.5	90,25	9	PQPEQQFPQ
579	gamma-gliadin	glia gamma 4b	Immunogenic	Deamidated	DQ2.5	90,25	9	PQPQQEFPPQ
580	gamma-gliadin	glia-gamma 4b	Immunogenic	Deamidated	DQ2.5	90,25	9	PQPEQEFPPQ
581	gamma-gliadin	Wheat peptide W16	Immunogenic	Deamidated	DQ2	62	16	GQQPFPPQPEQFPQPG
582	gamma-gliadin	Wheat peptide W16	Immunogenic	Deamidated	DQ2	62	13	QPFPPQPEQFPQP
583	gamma-1 gliadin	glia-alpha 1, glia-gamma 1	Immunogenic	Native	DQ2.5/DQ8			8,74,76,24 9 PQQSFPQQQ
584	gamma-gliadin	glia-alpha1, glia-gamma1	Immunogenic	Deamidated	DQ2.5/DQ8			8,74,76,24 9 PQQSFPQQE
585	gamma-1 gliadin	glia-alpha 1, glia-gamma 1	Immunogenic	Deamidated	DQ2.5/DQ8	8,76,24	9	PQQSFPQQE
586	gamma-1 gliadin	glia-alpha1, glia-gamma1	Immunogenic	Deamidated	DQ2.5, DQ8	8,17,76,24	9	PQQSFPQQQ
587	gamma-gliadin	Glia-gamma30-gliadin (p222-p236)	Immunogenic	Native	DQ2	19	15	VQGQGIIQPQQPAQL
588	gamma-gliadin	Glia-gamma30-gliadin (p222-236; E225)	Immunogenic	Deamidated	DQ2	19	15	VQGEIIQPQQPAQL
589	gamma-gliadin	Glia-gamma30-gliadin (p222-236; E231)	Immunogenic	Deamidated	DQ2	19	15	VQGQGIIQPEQPAQL
590	gamma-gliadin	Glia-gamma30-gliadin (p222-236; E225 and E231)	Immunogenic	Deamidated	DQ2	19	15	VQGEIIQPEQPAQL
591	gamma-gliadin	DQ2-y -II y-Glia (p222-p236)	Immunogenic	Native	DQ2	57	15	GQGIIQPQQPAQLIR

592	gamma-gliadin	DQ2-y -II y-Glia (p222-p236; E229)	Immunogenic	Deamidated				
	DQ2	57 15	GQGIIQPEQPAQLIR					
593	gamma-gliadin	gamma5-gliadin (p227-p237) ; gamma-II epitope	Immunogenic					
	Native DQ2	25 11	GIIQPQQPAQL					
594	gamma-gliadin	gamma5-gliadin (p227-237; E232)	Immunogenic	Deamidated				
	DQ2	25 11	GIIQPEQPAQL					
595	gamma-gliadin	gamma5-gliadin (p228-237)	Immunogenic	Native	DQ2	47,25	10	
		IIQPQQPAQL						
596	gamma-gliadin	gamma5-gliadin (p228-237; E232)	Immunogenic	Deamidated				
	DQ2	47,25 10	IIQPEQPAQL					
597	gamma-gliadin	gamma-2 peptide 1306; Glia-gamma30-gliadin (p227-p235) minimal epitope	Immunogenic					
	Native DQ2	19,23,2 9	IIQPQQPAQ					
598	gamma-gliadin	gamma-2 peptide 1306; Glia-gamma30-gliadin (p228-p235; E232) minimal epitope	Immunogenic	Deamidated	DQ2	19 9		
599	gamma-5 gliadin	glia-gamma 2	Immunogenic	Native	DQ2.5	90,8,25	9	
		IQPQQPAQL						
600	gamma-5 gliadin	glia-gamma 2	Immunogenic	Deamidated	DQ2.5	90,8,25	9	
		IQPEQPAQL						
601	gamma-gliadin	gamma-gliadin 1381 (p121-p140) ; gamma-gliadin M13 M36999 (121-140) identical to DQ2-gamma-I	Immunogenic	Native	DQ2, DQ8	17,23	20	
		QFPQQPQQSFPQQQPAI						
602	gamma-gliadin	DQ2-gamma-I gamma-Glia (p139-p153)	Immunogenic	Native				
	DQ2	57 15	PQQPQQSFPQQQPA					
603	gamma-gliadin	DQ2-gamma-I gamma-Glia (p139-p153; E147)	Immunogenic	Deamidated				
	DQ2	57 15	PQQPQQSFPEQQQPA					
604	gamma-gliadin	DQ2-gamma-I gamma-Glia (p139-p153; E150)	Immunogenic	Deamidated				
	DQ2	57 15	PQQPQQSFPQQEQPA					
605	gamma-gliadin	DQ2-gamma-I gamma-Glia (p139-p153; E147 and E150)	Immunogenic	Deamidated	DQ2	57 15		
606	gamma-gliadin	gamma-gliadin 1382 (p131-p150)	Immunogenic	Native	DQ8	17		
		20 SFPQQQPAIQSFLQQQMNP						
607	gamma-gliadin	gamma-gliadin 1383 (p141-p160)	Immunogenic	Native	DQ8	17		
		20 QSFLQQQMNPCKNFLQQCN						
608	gamma-gliadin	gamma-gliadin 1388 (p201-p220)	Immunogenic	Native	DQ2	17		
		20 IHSVHSHIMQQEQQGVPI						
609	gamma-gliadin	gamma-gliadin M23 M36999 (221-240) homologous to DQ2-gamma-II	Immunogenic	Native	DQ2	17,23	20	
610	gamma-gliadin	gamma-gliadin 1391 (p231-p250) ; gamma-gliadin M24 M36999 (231-250) identical to DQ2-gamma-II	Immunogenic	Native	DQ2	17,23	20	
		LGIIQPQQPAQLEGIRSLVL						
611	gamma-gliadin	Gluten peptide #19	Immunogenic	Native	DQ2 (DQ2.5)	61	18	
		PHQPQQQVPQPQQPQQPF						
612	gamma-gliadin	Predicted gamma-gliadin	Immunogenic	Native	DQ8 (DQ2/8)			
		22,8 14	QQPFPQQPQQPFPQ					
613	gamma-gliadin	Glia-gamma2 in Deamidated form	Immunogenic	Deamidated				
	DQ2	8 14	QQPFPEQPEQPFQ					
614	gamma-gliadin	Glia-gamma2 in Deamidated form	Immunogenic	Deamidated				
	DQ2	8 14	QQPFPQQPEQPFQ					
615	gamma-gliadin	Glia-gamma2 in Deamidated form	Immunogenic	Deamidated				
	DQ2	8 14	QQPFPEQPPQPFQ					

616	gamma-gliadin	gamma-gliadin P08453 (p94-p113)	Immunogenic	Deamidated				
	DQ8	54 20 QTQQPQQPFPPQQPQQPFQT						
617	gamma-gliadin	Predicted gamma-gliadin	Immunogenic	Native	DQ8 (DQ2/8)	22		
	14	PPQLQQPQQPFPPQ						
618	gamma-gliadin	gamma-I gliadin 1206	Immunogenic	Native	DQ2	2	21	
		YQQLPQPQQPQQSFPPQQQRPF						
619	gamma-gliadin	gamma-type gliadin of GDB2_WHEAT (SwissProt P08453) (p134-p153)	Immunogenic	Deamidated	DQ2	24	20	QQLPQPQQPQQSFPPQQQRPF
620	gamma-gliadin	Glia-gamma1 epitope	Immunogenic	Native	DQ2	9	17	QPQQPQQSFPPQQQRPF
621	gamma-gliadin	Glia-gamma1(p138-p153)	Immunogenic	Native	DQ2	19	16	QPQQPQQSFPPQQQRPF
622	gamma-gliadin	Glia-gamma1 (p139-p153)	Immunogenic	Native	DQ2	8	15	PQQPQQSFPPQQQRPF
623	gamma-gliadin	Glia-gamma1 (p139-p153; E148)	Immunogenic	Deamidated	DQ2	8	15	PQQPQQSFPEQQQRPF
624	gamma-gliadin	Glia-gamma1 (p139-p153; E140 and E148)	Immunogenic	Deamidated	DQ2	8	15	PEQPQQSFPEQQQRPF
625	gamma-gliadin	Glia-gamma1 (p139-p153; E148 and E150)	Immunogenic	Deamidated	DQ2	8	15	PQQPQQSFPEQERPF
626	gamma-gliadin	Glia-gamma1 (p139-p153; E140, E148 and E150)	Immunogenic	Deamidated	DQ2	8	15	PEQPQQSFPEQERPF
627	gamma-gliadin	gamma-I, gamma-Gliadin (p139-p152)	Immunogenic	Native	DQ2			43,25 14 PQQPQQSFPPQQRP
628	gamma-gliadin	gamma-Gliadin (p139-p152; E140, E148 and E150) E residues in the gliadin peptides are introduced to mimic the deamidation mediated by tissue transglutaminase.	Immunogenic	Deamidated	DQ2	43,25	14	PEQPQQSFPEQERP
629	gamma-gliadin	gamma-gliadin (p139-p152; E148)	Immunogenic	Deamidated	DQ2	55	14	PQQPQQSFPEQQRP
630	gamma-gliadin	gamma-gliadin (p139-p152; E140 and E148)	Immunogenic	Deamidated	DQ2 (DQ2.2 and DQ2.5)	25	14	PEQPQQSFPEQQRP
631	gamma-gliadin	P-3 gamma-gliadin (p139-p152; K139, E140, E148 and E150)	Immunogenic	Synthesised as Deamidated	DQ2	56	14	KEQPQQSFPEQERP
632	gamma-gliadin	P-2 gamma-gliadin (p139-p152; K140, E148 and E150)	Immunogenic	Synthesised as Deamidated	DQ2	56	14	PKQPQQSFPEQERP
633	gamma-gliadin	P-1 gamma-gliadin (p139-p152; K141, E140, E148 and E150)	Immunogenic	Synthesised as Deamidated	DQ2	56	14	PEKPQQSFPEQERP
634	gamma-gliadin	P1 y-gliadin(p139-p152; K142, E140, E148 and E150)	Immunogenic	Synthesised as Deamidated	DQ2	56	14	PEQKQQSFPEQERP
635	gamma-gliadin	P2 y-gliadin (p139-p152; K143, E140, E148 and E150)	Immunogenic	Synthesised as Deamidated	DQ2	56	14	PEQPKQSFPEQERP
636	gamma-gliadin	P4 y-gliadin (p139-p152; K144, E140, E148 and E150)	Immunogenic	Synthesised as Deamidated	DQ2	56	14	PEQPQQKFPEQERP
637	gamma-gliadin	P9 gamma-gliadin (p139-p152; E140, E148 and K150)	Immunogenic	Synthesised as Deamidated	DQ2	56	14	PEQPQQSFPEQKRP
638	gamma-gliadin	P10 gamma-gliadin (p139-p152; K151, E140, E148 and E150)	Immunogenic	Synthesised as Deamidated	DQ2	56	14	PEQPQQSFPEQKRP
639	gamma-gliadin	P11 y-gliadin (p139-p152; K152, E140, E148 and E150)	Immunogenic	Synthesised as Deamidated	DQ2	56	14	PEQPQQSFPEQERK
640	gamma-gliadin	gamma-I epitope in native form	Immunogenic	Native	DQ2	47	12	QPQQSFPPQQRP

641	gamma-gliadin	Deamidated form of gamma-I epitope	Immunogenic	Deamidated				
	DQ2	47 12	QPQQSFPEQQRP					
642	gamma-gliadin	Glia-alpha20	Immunogenic	Native	DQ2	9	16	
			QQSFPQQRPFIQPSL					
643	gamma-gliadin	gamma-gliadin AAK84772 (p130-p149)	Immunogenic	Native	DQ8		54	
	20	PQPQQPQLPFPQQPQQPFPQ						
644	gamma-gliadin	Predicted gamma-gliadin	Immunogenic	Native	DQ8 (DQ2/8)		22	
	14	PFPQQPQQPQQPFPQ						
645	gamma-gliadin	gamma-gliadin AAK84776 (p102-p121)	Immunogenic	Native	DQ8		54	
	20	QQPLPQQPQQPQQPFPQSQQP						
646	gamma-gliadin	gamma-gliadin AAK84772 (p121-p140)	Immunogenic	Native	DQ8		54	
	20	QPQQPQQPFPQQQQPLIQPY						
647	gamma-gliadin	Wheat peptide W35	Immunogenic	Native	DQ2	62	20	
		PQQPFPQQPQQQFPQPQQPQ						
648	gamma-gliadin	Wheat peptide W35	Immunogenic	Native	DQ2	62	12	
		PFPQQPQQQFPQ						
649	gamma-gliadin	Wheat peptide W31	Immunogenic	Native	DQ2	62	20	
		QFPFQLQQPQQPLQPQQPQ						
650	gamma-gliadin	Wheat peptide W31	Immunogenic	Native	DQ2	62	12	
		QFPFQLQQPQQP						
651	LMW glutenin	Wheat peptide W15 LMW	Immunogenic	Native	DQ2	62	20	
		SHIPGLERPWQQQPLPPQQT						
652	LMW glutenin	Wheat peptide W15 LMW	Immunogenic	Native	DQ2	62	15	
		QGLERPWQQQPLPPQ						
653	LMW glutenin	Wheat peptide W15 LMW	Immunogenic	Deamidated	DQ2		62	
	15	EGLERPWQEQLPPQ						
654	LMW glutenin	Wheat peptide W15 LMW	Immunogenic	Native	DQ2	62	12	
		LERPWQQQPLPP						
655	LMW glutenin	Wheat peptide W11	Immunogenic	Deamidated	DQ2	62	16	
		GQQAFPQPEQTFPHQG						
656	LMW glutenin	Wheat peptide W11	Immunogenic	Native	DQ2	62	15	
		QQAFPQPQQTFPHQP						
657	LMW glutenin	Wheat peptide W11	Immunogenic	Deamidated	DQ2	62	15	
		EQAFPQPEQTFPHQP						
658	LMW glutenin	Wheat peptide W11	Immunogenic	Native	DQ2	62	20	
		QAFPQPQQTFPHQPQQQFPQ						
659	LMW glutenin	Wheat peptide W11	Immunogenic	Native	DQ2	62	12	
		QAFPQPQQTFPH						
660	LMW glutenin	Wheat peptide W11	Immunogenic	Deamidated	DQ2	62	12	
		QAFPQPEQTFPH						
661	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60)	Immunogenic	Native				
	DQ2	19 15	QQPPFSQQQQQLPQ					
662	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E52)	Immunogenic	Deamidated				
	DQ2	19 15	QQPPFSEQQQQLPQ					
663	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E53)	Immunogenic	Deamidated				
	DQ2	19 15	QQPPFSQEQQQQLPQ					
664	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E55)	Immunogenic	Deamidated				
	DQ2	19 15	QQPPFSQQQEQQLPQ					
665	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E56)	Immunogenic	Deamidated				
	DQ2	19 15	QQPPFSQQQQEPLPQ					

666	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E52 and 53)	Immunogenic	Deamidated	DQ2	19	15	QQPPFSEEQQLPQ
667	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E52 and 55)	Immunogenic	Deamidated	DQ2	19	15	QQPPFSEQQQLPQ
668	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E52 and 56)	Immunogenic	Deamidated	DQ2	19	15	QQPPFSEQQQLPQ
669	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E53 and 55)	Immunogenic	Deamidated	DQ2	19	15	QQPPFSQEQQLPQ
670	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E53 and 56)	Immunogenic	Deamidated	DQ2	19	15	QQPPFSQEQQLPQ
671	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E55 and 56)	Immunogenic	Deamidated	DQ2	19	15	QQPPFSQQQEEPLPQ
672	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E52, 53 and 55)	Immunogenic	Deamidated	DQ2	19	15	QQPPFSEEQQLPQ
673	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E52, 53 and 56)	Immunogenic	Deamidated	DQ2	19	15	QQPPFSEQQQLPQ
674	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E53, 55 and 56)	Immunogenic	Deamidated	DQ2	19	15	QQPPFSQEQEEPLPQ
675	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E52, 55 and 56)	Immunogenic	Deamidated	DQ2	19	15	QQPPFSEQQEEPLPQ
676	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p46-p60; E52, 53, 55 and 56)	Immunogenic	Deamidated	DQ2	19	15	QQPPFSEQQEEPLPQ
693	gamma-gliadin or LMW glutenin	CAUTION 100% matches to 5 microbial proteins and to wheat proteins	Glutenin-Glt-17 (p50-p58)	Immunogenic	Native	DQ2	27	9
								FSQQQQQL
694	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p50-p58; E52)	Immunogenic	Deamidated	DQ2	27	9	FSEQQQL
695	gamma-gliadin or LMW glutenin	CAUTION 100% matches to two Pinus proteins / not wheat	Glutenin-Glt-17 (p50-p58; E53)	Immunogenic	Deamidated	DQ2	27	9
								FSQEQQL
696	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p50-p58; E55)	Immunogenic	Deamidated	DQ2	27	9	FSQQQQL
697	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p50-p58; E52 and E53)	Immunogenic	Deamidated	DQ2	27	9	FSEQQQL
698	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p50-p58; E52 and E55)	Immunogenic	Deamidated	DQ2	27	9	FSEQQQL
699	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p50-p58; E53 and E55)	Immunogenic	Deamidated	DQ2	27	9	FSQEQQL
700	gamma-gliadin or LMW glutenin	Glutenin-Glt-17 (p50-p58; E52, E53 and E55)	Immunogenic	Deamidated	DQ2	27	9	FSEQQQL
701	gamma-gliadin or LMW glutenin	Glutenin-17 epitope homolog	Immunogenic	Native	DQ2	8,19	15	QQPPFSQQQPVLPQ
702	gamma-gliadin or LMW glutenin	Glutenin-17 epitope homolog in Deamidated form	Immunogenic	Deamidated	DQ2	8	15	QQPPFSEQQQPVLPQ
703	gamma-gliadin or LMW glutenin	Glutenin-17 epitope homolog in Deamidated form	Immunogenic	Deamidated	DQ2	8	15	QQPPFSQQEQPVLPQ
704	gamma-gliadin or LMW glutenin	Glutenin-17 epitope homolog in Deamidated form	Immunogenic	Deamidated	DQ2	8	15	QQPPFSEQQPVLPQ
705	gamma-gliadin or LMW glutenin	LMW T cell epitope	Immunogenic	Deamidated	DQ2	62	15	EQPPFSEQQPVLPQ

706	Glut-L1 CAUTION 100% match to a fungal protien Hebeloma sp. & many wheat Glt-17 (Var1)	Immunogenic	Native	DQ2.2	44,90	9	PFSQQQQPV	
707	glut-L1 Glt-17 (Var1)	Immunogenic	Deamidated	DQ2.2	44,90	9	PFSEQQQP	
708	glut-L1 Glt-17 (Var1)	Immunogenic	Deamidated	DQ2.2	44,90	9	PFSQQEQPV	
709	glut-L1 Glt-17 (Var1)	Immunogenic	Deamidated	DQ2.2	44,90	9	PFSEQQEQPV	
710	gamma-gliadin or LMW gluteninWheat peptide W12	Immunogenic	Native	DQ2		62	20 CKVFLQQQCSPVAMPQRLAR	
711	gamma-gliadin or LMW gluteninWheat peptide W12	Immunogenic	Native	DQ2		62	16 LQQQCSPVAMPQRLAR	
712	gamma-gliadin or LMW-glutenin	Wheat peptide W05	Immunogenic	Native			DQ2 62 20 PQQQPFPQPQQPFSQQPQQ	
713	gamma-gliadin or LMW-glutenin	Wheat peptide W05	Immunogenic	Deamidated			DQ2 62 20 PQQQPFPQPQEFPFSQQPQQ	
714	gamma-gliadin or LMW-glutenin	Wheat peptide W05	Immunogenic	Native			DQ2 62 12 QPFPQPQQPFSQ	
715	gamma-gliadin or LMW-glutenin	Wheat peptide W05	Immunogenic	Deamidated			DQ2 62 12 QPFPQPQEFPFSQ	
716	gamma-gliadin or LMW-glutenin	Wheat peptide W17	Immunogenic	Native			DQ2 62 20 QPFPQPQQPQLPFPQQPQQ	
717	gamma-gliadin or LMW-glutenin	Wheat peptide W17	Immunogenic	Native			DQ2 62 15 QPFPQPQQPQLPFPQ	
718	gamma-gliadin or LMW-glutenin	Wheat peptide W17	Immunogenic	Deamidated			DQ2 62 15 EPFPQPEQPELPFPQ	
719	gamma-gliadin or LMW-glutenin	Wheat peptide W17	Immunogenic	Native			DQ2 62 12 QPFPQPQQPQLP	
720	LMW glutenin GLT/GLIA homologue peptide 12	Immunogenic	Native	DQ2		19	15 QQPPFSQQQQPPFSQ	
721	LMW glutenin LMW glutenin-glt-156 (p40-p59)	Immunogenic	Native	DQ2		19	20 QQQQPPFSQQQQSPFSQQQQ	
722	LMW glutenin LMW glutenin-glt-156 (p40-p59; E48)	Immunogenic	Deamidated				DQ2 19 20 QQQQPPFSEQQQSPFSQQQQ	
723	LMW glutenin LMW glutenin-glt-156 (p40-p59; E51)	Immunogenic	Deamidated				DQ2 19 20 QQQQPPFSQQQESPFSQQQQ	
724	LMW glutenin LMW glutenin-glt-156 (p40-p59; E48 and E51)	Immunogenic	Deamidated				DQ2 19 20 QQQQPPFSEQQESPFSQQQQ	
725	LMW glutenin Homolog of Deamidated Glt-156 minimal epitope (p40-p59)	Immunogenic	Deamidated	DQ2	20	15	QQQQPPFSEEQESPY	
726	LMW glutenin Homolog of Deamidated Glt-156 minimal epitope (p40-p59)	Immunogenic	Deamidated	DQ2	20	15	QQQQPPFSEEQESPL	
727	LMW glutenin Deamidated Glt-156 minimal epitope (p40-p59)	Immunogenic	Deamidated	DQ2	20	15	QQQQPPFSEEQESPF	
728	LMW glutenin Glt-156 minimal epitope (p41-p55)	Immunogenic	Deamidated	DQ2	20	15	QQQQPPFSEEQESPF	
729	LMW glutenin Glt-156 minimal epitope in considered native form	Immunogenic		DQ2	20	15	QQPPFSQQQQSPFSQ	
730	LMW glutenin Glt-156 minimal epitope in considered Deamidated form	Immunogenic	Deamidated	DQ2	20	15	QQPPFSEEQESPF	
731	LMW glutenin GLT/GLIA homologue peptide 4	Immunogenic	Native	DQ2		19	12 QQPPFSQQQQSP	
732	LMW glutenin Glt-156 minimal epitope	Immunogenic	Deamidated	DQ2		20	15 QPPFSEEQESPF	

733	LMW glutenin	LMW glutenin-glt-156 (p40-p59)	Immunogenic	Native	DQ2	16
	14	QPPFSQQQQSPFSQ				
734	LMW glutenin	Glt-156 minimal epitope in considered native form	Immunogenic			
	Native DQ2	20 15 PPFSQQQQSPFSQQQ				
735	LMW glutenin	Glt-156 minimal epitope in considered Deamidated form				
	Immunogenic	Deamidated DQ2 20 15 PPFSEEQESPFSQQQ				
736	LMW glutenin	Glt-156 minimal epitope in considered native form	Immunogenic			
	Native DQ2	20 15 PFSQQQQSPFSQQQ				
737	LMW glutenin	Glt-156 minimal epitope in considered Deamidated form				
	Immunogenic	Deamidated DQ2 20 15 PFSEEQESPFSQQQ				
738	LMW glutenin	LMW glutenin-glt-156 (p45-p54) minimal epitope	Immunogenic			
	Native DQ2	19,20 10 PFSQQQQSPF				
739	LMW glutenin	LMW glutenin-glt-156 (p45-p54; E48) minimal epitope	Immunogenic			
	Deamidated	DQ2 19,20 10 PFSEQQQSPF				
740	LMW glutenin	LMW glutenin-glt-156 (p45-p54; E49) minimal epitope	Immunogenic			
	Deamidated	DQ2 20 10 PFSQEQQSPF				
741	LMW glutenin	LMW glutenin-glt-156 (p45-p54; E51) minimal epitope	Immunogenic			
	Deamidated	DQ2 19,20 10 PFSQQQESPF				
742	LMW glutenin	LMW glutenin-glt-156 (p45-p54; E48 and E51) minimal epitope				
	Immunogenic	Deamidated DQ2 19,20 10 PFSEQQESPF				
743	LMW glutenin	LMW glutenin-glt-156 (p45-p54; E48 and E49) minimal epitope				
	Immunogenic	Deamidated DQ2 20 10 PFSEQQQSPF				
745	LMW glutenin	LMW glutenin-glt-156 (p45-p54; E49 and E49) minimal epitope				
	Immunogenic	Deamidated DQ2 20 10 PFSQEQESPF				
746	LMW glutenin	LMW glutenin-glt-156 (p45-p54; E48, E49 and E51) minimal epitope				
	Immunogenic	Deamidated DQ2 20 10 PFSEEQESPF				
747	glut-L2	LMW glutenin-glt-156 (p46-p54)	Immunogenic	Native	DQ2.5	19,90,27
	9	FSQQQQSPF				
748	glut-L2	LMW glutenin-glt-156 (p46-p54; E48)	Immunogenic	Deamidated	DQ2.5	
	19,90,27	9 FSEQQQSPF				
749	glut-L2	LMW glutenin-glt-156 (p46-p54; E51)	Immunogenic	Deamidated	DQ2.5	
	19,90,27	9 FSQQQESPF				
750	glut-L2	LMW glutenin-glt-156 (p46-p54; E48 and E51)	Immunogenic	Deamidated		
	DQ2.5 19,90,27	9 FSEQQESPF				
751	LMW glutenin	GLT/GLIA homologue peptide 13	Immunogenic	Native	DQ2	19
	15	QPPFSQQQQPQFSQ				
752	LMW glutenin	Gluten peptide #25	Immunogenic	Native	DQ2 (DQ2.5)	61 19
		SHQQQPFPPQQPYPQQPYPS				
753	gamma-gliadin	14-mer-2 gamma-Glia (p173-p186)	Immunogenic	Native	DQ2	57
	14	PQQPFPSQQQQPLI				
754	gamma-gliadin	14-mer-2 gamma-Glia (p173-p186) in Deamidated form	Immunogenic			
	Deamidated	DQ2 57 14 PQQPFPSQQEQPLI				
755	LMW glutenin	GLT/GLIA homologue peptide 17	Immunogenic	Native	DQ2	19
	15	QPPFSQQQQPILPQ				
756	LMW glutenin	Glt-156 homolog	Immunogenic	Native	DQ2	21 14
		QPPFSQQQQPILPQ				
757	LMW glutenin	Glt-156 homolog in Deamidated form	Immunogenic	Deamidated		
	DQ2 21 14	QPPFSEQEQPILPQ				
762	LMW glutenin	GLT/GLIA homologue peptide 16	Immunogenic	Native	DQ2	19
	15	QPPFSQQQQPILL				

763	LMW glutenin	Glt-156 homolog	Immunogenic	Native	DQ2	21	14	
		QPPFSQQQQPILL						
764	LMW glutenin	Glt-156 homolog in Deamidated form	Immunogenic	Deamidated				
	DQ2	21 14						
		QPPFSEEQEQPILL						
765	HMW glutenin	Wheat peptide W21 HMW	Immunogenic	Native	DQ2	62	20	
		QGQQGYYPISPQQSGGQQP						
766	HMW glutenin	Wheat peptide W21 HMW	Immunogenic	Native	DQ2	62	16	
		QGQQGYYPISPQQSGQ						
767	HMW glutenin	Wheat peptide W21 HMW	Immunogenic	Deamidated		DQ2	62	
	16	EGQQGYYPISPQQSGQ						
768	HMW glutenin	Naturally occurring glutenins p722-p736 (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15	GQAGYYPTSPQQSGQ
769	HMW glutenin	Naturally occurring glutenins p722-p736 (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15	GQQGYPTSPQQPGQ
770	HMW glutenin	Naturally occurring glutenins p722-p736 (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15	GQQGYYPISPQQSGQ
771	HMW glutenin	Wheat peptide W29	Immunogenic	Native	DQ2	62	20	
		GQQQSGYYPTSPQQSGQEAT						
772	HMW glutenin	Wheat peptide W29	Immunogenic	Native	DQ2	62	16	
		GQQQSGYYPTSPQQSG						
773	HMW glutenin	Wheat peptide W24 HMW	Immunogenic	Native	DQ2	62	20	
		PGQQQSGYYPTSPQQSGQKQ						
774	HMW glutenin	Wheat peptide W24 HMW	Immunogenic	Native	DQ2	62	16	
		PGQQQSGYYPTSPQQS						
775	HMW glutenin	Naturally occurring glutenins (p722-p736) (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15	GQSGYYPTSPQQSGQ
776	HMW glutenin	Naturally occurring glutenins (p722-p736) (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15	GQQGYYPISPQQQLGQ
777	HMW glutenin	Naturally occurring glutenins (p722-p736) (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15	GQLGYPTSPQQSGQ
778	HMW glutenin	Wheat peptide W22 HMW	Immunogenic	Native	DQ2	62	20	
		LQPGQQPGYYPTSPQQIGQ						
779	HMW glutenin	Wheat peptide W22 HMW	Immunogenic	Native	DQ2	62	16	
		QQQPGYYPTSPQQIGQ						
780	HMW glutenin	Naturally occurring glutenins (p722-p736) (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15	GQPGYYPTSPQQIGQ
781	HMW-Glutenin	Naturally occurring glutenins (p722-p736) (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15	GQPGYYPTSPQQPGQ
782	HMW-Glutenin	Naturally occurring glutenins (p722-p736) (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15	GQPGYYPTSPQQSGQ
783	HMW-Glutenin	Naturally occurring glutenins p722-p736 (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15	GQQGYPTSLQQPGQ
784	HMW-Glutenin	HMW glutenin-glt04 (p707-p742)	Immunogenic	Native	DQ8 (DQ2/8)	7	36	SGQQQRPGQWLQPGQQGYYPTSPQQSGGQQQLGQ
785	HMW-Glutenin	HMW glutenin-glt04 (p719-p736)	Immunogenic	Native	DQ8 (DQ2/8)	7	18	PGQQQQGYPTSPQQSGQ
786	HMW-Glutenin	glt04 (p722-p736)	Immunogenic	Native	DQ8 (DQ2/8)	7	15	GQQGYPTSPQQSGQ
787	HMW-Glutenin	glt04 (p722-p735)	Immunogenic	Native	DQ8 (DQ2/8)	7	14	GQQGYPTSPQQSG

788	HMW-Glutenin glt04 (p722-p734)	Immunogenic	Native	DQ8 (DQ2/8)	7	13
	GQQGYPTSPQQS					
789	HMW-Glutenin glt04 (p723-p736)	Immunogenic	Native	DQ8 (DQ2/8)	7	14
	QGGYPTSPQQSGQ					
790	HMW-Glutenin glt04 (p723-p735)	Immunogenic	Native	DQ8 (DQ2/8)	7	13
	QGGYPTSPQQSG					
791	HMW-Glutenin glt04 (p723-p735; E724)	Immunogenic	Deamidated	DQ8 (DQ2/8)		
	7,83 13 QEGYPTSPQQSG					
792	HMW-Glutenin glt04 (p723-p734)	Immunogenic	Native	DQ8 (DQ2/8)	52	12
	QGGYPTSPQQS					
793	HMW-Glutenin glt04 (p724-p735)	Immunogenic	Native	DQ8 (DQ2/8)	7	12
	QGGYPTSPQQSG					
794	glut-H1 HMW glutenin (p724-p734)	Immunogenic	Native	DQ8, DQ8.5	7,76,27	11
	QGGYPTSPQQS					
797	HMW-Glutenin glt04 (p725-p735)	Immunogenic	Native	DQ8 (DQ8.5)	7,83	11
	GGYPTSPQQSG					
798	HMW-Glutenin Naturally occurring glutenins (p722-p736) (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15
	GQQGYYPISPPQPGQ					
799	HMW-Glutenin Naturally occurring glutenins (p722-p736) (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15
	GQQGYPTSPQQSPQ					
800	HMW-Glutenin Naturally occurring glutenins (p722-p736) (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15
	GQQGYPTSPQQLGQ					
801	HMW-Glutenin Naturally occurring glutenins (p722-p736) (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15
	GQQGYPTSPQHPPGQ					
802	HMW-Glutenin Naturally occurring glutenins (p722-p736) (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15
	GQPGYPTSPQLSGQ					
803	HMW-Glutenin Naturally occurring glutenins (p722-p736) (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15
	GQHGYPTSPQLSGQ					
804	HMW-Glutenin Naturally occurring glutenins (p722-p736) (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15
	GQQGYPTSPQQPPQ					
805	HMW-Glutenin Naturally occurring glutenins (p722-p736) (homolog of glt04)	Immunogenic	Native	DQ8 (DQ2/8)	7	15
	GQQGYPTSVQQPGQ					
806	Hordein Barley peptide B04, B17	Immunogenic	Native	DQ2	62	20
	QPQQPQFPQQPVPQQPY					
807	Hordein Barley peptide B17	Immunogenic	Native	DQ2	62	16
	PQQPQFPQQPVPQQP					
808	Hordein Barley peptide B04	Immunogenic	Native	DQ2	62	12
	PQQPVPQQPQPY					
809	Hordein Barley peptide B05, B08 in native form	Immunogenic	Native	DQ2	62	
	20 PQQFPQQIPQQPQYPQQP					
810	Hordein Barley peptide B05, B08 in Deamidated form	Immunogenic	Deamidated			
	DQ2 62 20 PQQFPQQPIEQPQYPQQP					
811	Hordein Barley peptide B05	Immunogenic	Native	DQ2	62	16
	PQQFPQQIPQQPQPY					
812	Hordein Barley peptide B05	Immunogenic	Deamidated	DQ2	62	16
	PQQFPQQPIEQPQPY					
813	Hordein Barley peptide B06	Immunogenic	Native	DQ2	62	20
	QQPQPFSSQQIPQQPQYPQ					
814	Hordein Barley peptide B06	Immunogenic	Deamidated	DQ2	62	20
	QQPQPFSSQQPIEQPQYPQ					

815	Hordein	Barley peptide B06	Immunogenic	Native	DQ2	62	12	
	SQQPIPPQQPY							
816	Hordein	Barley peptide B06	Immunogenic	Deamidated	DQ2	62	12	
	SQQPIPEQQPY							
817	Hordein	Barley peptide B18	Immunogenic	Native	DQ2	62	20	
	QPQFPQQPIPLQPHQPYTQ							
818	Hordein	Barley peptide B18	Immunogenic	Native	DQ2	62	12	
	QPQFPQQPIPL							
819	Hordein	Barley peptide B13	Immunogenic	Native	DQ2	62	20	
	PQPYPQQPFPQQPPFCQQ							
820	Hordein	Barley peptide B13	Immunogenic	Native	DQ2	62	16	
	PQPYPQQPFPQQPP							
821	Hordein	Barley peptide B09, B12, B30	Immunogenic	Native	DQ2	62	20	
	QQPFPQQPFPQQPYPQQP							
822	Hordein	Barley peptide B09	Immunogenic	Native	DQ2	62	16	
	QQPFPQQPFPQQPYP							
823	Hordein	Barley peptide B30	Immunogenic	Native	DQ2	62	11	
	QQPFPQQPFPQ							
824	Hordein	Barley peptide B12	Immunogenic	Native	DQ2	62	16	
	PQQPFPQQPYPQQP							
825	Hordein	Barley peptide B11	Immunogenic	Native	DQ2	62	20	
	QPQYPQQPYPQQPFPQ							
826	Hordein	Barley peptide B11	Immunogenic	Native	DQ2	62	12	
	QPQYPQQPYP							
827	gamma-gliadin or LMW gluteninGlu-21 in considered native form					Immunogenic		
	Native	DQ2	19	21	QPQFPQQSEQSQPFPQPF			
828	Hordein	Barley peptide B03	Immunogenic	Native	DQ2	62	20	
	QPQQPFPQQPIPYQQP							
829	Hordein	Barley peptide B03	Immunogenic	Deamidated	DQ2	62	16	
	GQQPFPQPEQPIPYQG							
830	Hordein	Barley peptide B03	Immunogenic	Native	DQ2	62	12	
	QFPFPQQPIPY							
831	Hordein	Barley peptide B03	Immunogenic	Deamidated	DQ2	62	12	
	QFPFPQPEQPIPY							
832	Hordein	Barley peptide B02	Immunogenic	Native	DQ2	62	20	
	WQPQQPFPQQPFPPLQPPQ							
833	Hordein	Barley peptide B02	Immunogenic	Deamidated	DQ2	62	20	
	WQPQQPFPQPEQFPPLQPPQ							
834	Hordein	Barley peptide B02	Immunogenic	Deamidated	DQ2	62	16	
	GQQPFPQPEQFPPLQG							
835	Hordein	Barley peptide B02	Immunogenic	Native	DQ2	62	12	
	QFPFPQQPFPPL							
836	Hordein	Barley peptide B02	Immunogenic	Deamidated	DQ2	62	12	
	QFPFPQPEQFPPL							
837	Hordein	Barley peptide B19	Immunogenic	Native	DQ2	62	20	
	LPRPQQPFPWQPQQPFPQPQ							
838	Hordein	Barley peptide B26	Immunogenic	Native	DQ2	62	20	
	QPQQPFPPLQPPFPWQPQQ							
839	Hordein	Barley peptide B26	Immunogenic	Native	DQ2	62	12	
	PFPLQPPFPW							

840	Hordein	Barley peptide B29	Immunogenic	Native	DQ2	62	20	
	QPQQPFSFSQQPQQPFPLQP							
841	Hordein	Barley peptide B29	Immunogenic	Deamidated	DQ2	62	16	
	GFSFSQQPEQPFPLQG							
842	Hordein	Barley peptide B14	Immunogenic	Native	DQ2	62	20	
	FQQPQQSYPVQPQQPFPPQPQ							
843	Hordein	Barley peptide B14	Immunogenic	Deamidated	DQ2	62	16	
	GQSYPVQPEQPFPPQPG							
844	Hordein	Barley peptide B14	Immunogenic	Native	DQ2	62	12	
	SYPVQPQQPFPPQ							
845	Hordein	Barley peptide B14	Immunogenic	Deamidated	DQ2	62	12	
	SYPVQPEQPFPPQ							
846	Hordein	Barley peptide B29	Immunogenic	Native	DQ2	62	12	
	SFSQQPQQPFPL							
847	Hordein	Barley peptide B29	Immunogenic	Deamidated	DQ2	62	12	
	SFSQQPEQPFPL							
848	omega-gliadin	Wheat peptide W19	Immunogenic	Deamidated	DQ2	62	16	
	GQPFPPWQPEQPFPPQPG							
849	Hordein	Barley peptide B15	Immunogenic	Native	DQ2	62	20	
	YPQQPQPFPQQPIPQQPQPY							
850	Hordein	Barley peptide B15	Immunogenic	Native	DQ2	62	12	
	QPQPFPQQPIPQ							
851	hor-3	Hor-I	Immunogenic	Native	DQ2.5	88,62	9	PIPQQPQPY
852	hor-3	Hor-I	Immunogenic	Deamidated	DQ2.5	88,62	9	PIPEQPQPY
853	Hordein	Barley peptide B16	Immunogenic	Native	DQ2	62	20	
	QQQPFPQQPIPQQPQYPQQ							
854	Hordein	Barley peptide B16	Immunogenic	Native	DQ2	62	11	
	QQPFPQQPIPQ							
855	Hordein	Barley peptide B08	Immunogenic	Deamidated	DQ2	62	16	
	PQQPIPQQPQYPQQP							
856	Hordein	Barley peptide B08	Immunogenic	Deamidated	DQ2	62	16	
	PQQPIPEQPQYPQQP							
857	Hordein	Barley peptide B08	Immunogenic	Native	DQ2	62,86	16	
	QPQQPIPQQPQYPQQ							
858	Hordein	Barley peptide B08	Immunogenic	Deamidated	DQ2	62,86	16	
	EPEQPIPEQPQYPQQ							
859	Hordein	Hordein core epitope in native form		Immunogenic	Native	DQ2	27	
	9	FPPQQPFPQ						
860	Hordein	Hordein core epitope in demainated form		Immunogenic	Deamidated			
	DQ2	27	9	FPPEQPFPQ				
861	Hordein	Barley peptide B21, B25		Immunogenic	Native	DQ2	62	20
	PFPQQPQQPFPQQPFRQQ							
862	Hordein	Barley peptide B21	Immunogenic	Deamidated	DQ2	62	20	
	PFPQQPQQPFPQPEQPFRRQ							
863	Hordein	alpha9-Hordein in native form		Immunogenic	Native	DQ2	8	14
	PQQPFPQPQQPFRQ							
864	Hordein	alpha9-Hordein in Deamidated form		Immunogenic	Deamidated			
	DQ2	8	14	PQQPFPQPEQPFRRQ				
865	Hordein	Barley peptide B21	Immunogenic	Native	DQ2	62	13	
	QQPFPQPQQPFRQ							

866	Hordein	Barley peptide B21	Immunogenic	Deamidated	DQ2	62	13
	QQPFQPEQPFQRQ						
867	hor-1	Hordein/Secalin	Immunogenic	Native	DQ2.5	88,90,8,27,62	9
868	hor-1	Hordein/Secalin	Immunogenic	Deamidated	DQ2.5	88,90,8,27,62	9
	PFPQPEQPF						
869	Hordein	Barley peptide B22	Immunogenic	Native	DQ2	62	20
	PQQPFQPPQPFQQTIPQQP						
870	Hordein	Barley peptide B22	Immunogenic	Native	DQ2	62	12
	QQPFQPPQPFQ						
871	Hordein	Barley peptide B27	Immunogenic	Native	DQ2	62	20
	TFPPSQPNPLQPPQPFPLQ						
872	Hordein	Barley peptide B27	Immunogenic	Native	DQ2	62	13
	PNPLQPPQPFPLQ						
873	Hordein	Barley peptide B23, B24	Immunogenic	Native	DQ2	62	20
	NPLQPPQPFPLQPPQPPQPF						
874	Hordein	Barley peptide B23	Immunogenic	Native	DQ2	62	16
	NPLQPPQPFPLQPPQ						
875	Hordein	Barley peptide B24	Immunogenic	Native	DQ2	62	16
	PLQPPQPFPLQPPQ						
876	Hordein	Barley peptide B10	Immunogenic	Native	DQ2	62	20
	PQQPPQPFQPPQPFQSWQPQ						
877	Hordein	Barley peptide B10	Immunogenic	Deamidated	DQ2	62	20
	PQQPPQPFQPEQPFQSWQPQ						
878	Hordein	Barley peptide B10	Immunogenic	Native	DQ2	62	12
	QPFQPPQPFQSW						
879	Hordein	Barley peptide B10	Immunogenic	Deamidated	DQ2	62	12
	QPFQPEQPFQSW						
880	Hordein	Barley peptide B28	Immunogenic	Native	DQ2	62	20
	PQQTIPQPPQPFPLQPPQ						
881	Hordein	Barley peptide B28	Immunogenic	Native	DQ2	62	12
	TIPQPPQPFPL						
882	Hordein	Barley peptide B20	Immunogenic	Native	DQ2	62	20
	QQPFPLQPPQPFQPPQPFQ						
883	gamma-gliadin	Wheat peptide W26	Immunogenic	Deamidated	DQ2	62	16
	GQPFPLQPEQPFQPG						
884	gamma-gliadin	Wheat peptide W26	Immunogenic	Deamidated	DQ2	62	12
	PFPLQPEQPFQ						
885	gamma-hordein	Barley peptide B07	Immunogenic	Native	DQ2	62	20
	QSQQQFPQPPQPFQPPQPPQ						
886	gamma-hordein	alpha2-Hordein in native form	Immunogenic	Native	DQ2	8	14
	QQFPQPPQPFQPPQ						
887	gamma-hordein	alpha2-Hordein in Deamidated form	Immunogenic	Deamidated	DQ2	8	14
	QEFQPPQPPQPPQPPQ						
888	gamma-hordein	alpha2-Hordein in Deamidated form	Immunogenic	Deamidated	DQ2	8	14
	QQFPQPEQPFQPPQ						
889	gamma-hordein	alpha2-Hordein in Deamidated form	Immunogenic	Deamidated	DQ2	8	14
	QEFQPEQPFQPPQ						
890	gamma-hordein	Barley peptide B07	Immunogenic	Native	DQ2	62	12
	QQFPQPPQPFQ						
891	hor-2	Hordein/Secalin	Immunogenic	Native	DQ2.5	90,8,27	9
	PQPQQPFQ						

892	hor-2 Hordein/Secalin	Immunogenic	Deamidated	DQ2.5	90,8,27	9	
	PQPEQPFQ						
893	Secalin Rye peptide R05	Immunogenic	Deamidated	DQ2	62	16	
	GQPAPIQPEQPFQ						
894	Secalin Rye peptide R05, R26	Immunogenic	Native	DQ2	62	20	
	PAPIQPQQPFQPFQ						
895	Secalin Rye peptide R05	Immunogenic	Deamidated	DQ2	62	12	
	PAPIQPEQPFQ						
896	Secalin Rye peptide R05	Immunogenic	Native	DQ2	62	12	
	PAPIQPQQPFQ						
897	Secalin Rye peptide R12	Immunogenic	Native	DQ2	62	20	
	FPQQPQQPFQPFQ						
898	Secalin Rye peptide R12	Immunogenic	Deamidated	DQ2	62	16	
	GQQPFQPEQELPLQ						
899	Secalin Rye peptide R12	Immunogenic	Native	DQ2	62	12	
	QPFQPFQELPL						
900	Secalin Rye peptide R12	Immunogenic	Deamidated	DQ2	62	12	
	QPFQPEQELPL						
901	Secalin Rye peptide R29	Immunogenic	Native	DQ2	62	20	
	PTPIQPQQPFQPFQ						
902	Secalin Rye peptide R29	Immunogenic	Native	DQ2	62	12	
	PFQPFQPFQ						
903	Secalin gamma1-Secalin in native form	Immunogenic	Native	DQ2	90,27	9	
	PQQSFQ						
904	Secalin gamma1-Secalin in Deamidated form	Immunogenic	Deamidated	DQ2	90,27	9	
	PQQSFQ						
905	Secalin Rye peptide R10	Immunogenic	Native	DQ2	62	20	
	FPLQPQQPFQPEQIISQ						
906	Secalin Rye peptide R10	Immunogenic	Native	DQ2	62	12	
	PFQPEQIISQ						
907	Secalin Rye peptide R25	Immunogenic	Native	DQ2	62	20	
	FPQQPEQIISQPFPLQ						
908	Secalin Rye peptide R25	Immunogenic	Native	DQ2	62	15	
	PEQIISQPFPL						
909	Secalin Rye peptide R22	Immunogenic	Native	DQ2	62	20	
	PQQLFPLQPFPQPFQ						
910	Secalin Rye peptide R22	Immunogenic	Native	DQ2	62	11	
	LFPLQPFPQ						
911	Secalin alpha9-Secalin in native form	Immunogenic	Native	DQ2	8	14	
	PQQPFQPFQ						
912	Secalin alpha9-Secalin in Deamidated form	Immunogenic	Deamidated	DQ2	14	8	
	PEQPFQPFQ						
913	Secalin alpha9-Secalin in Deamidated form	Immunogenic	Deamidated	DQ2	14	8	
	PQQPFQPEQPFQ						
914	Secalin alpha9-Secalin in Deamidated form	Immunogenic	Deamidated	DQ2	14	8	
	PEQPFQPEQPFQ						
915	Secalin alpha2-Secalin in native form	Immunogenic	Native	DQ2	8	14	
	QPFQPFQSFQ						
916	Secalin alpha2-Secalin in Deamidated form	Immunogenic	Deamidated	DQ2	14	8	
	QPFQPEQPFQSFQ						

917	gamma-secalin Rye peptide R21 NMQVGPSGQVEWPQQQLPQ	Immunogenic	Native	DQ2	62	20
918	gamma-secalin Rye peptide R21 GMQVGPSGEVEWPQQG	Immunogenic	Native	DQ2	62	16
919	gamma-secalin Rye peptide R21 QVGPSGQVEWPQ	Immunogenic	Native	DQ2	62	12
920	gamma-secalin Rye peptide R21 QVGPSGEVEWPQ	Immunogenic	Native	DQ2	62	12
921	gamma-secalin Rye peptide R13, R28 SPQPQQPYPPQPPQPPQ	Immunogenic	Native	DQ2	62	20
922	gamma-secalin Rye peptide R13 GQPEQPYPEQPPQPPQ	Immunogenic	Deamidated	DQ2	62	16
923	gamma-secalin Rye peptide R13 PQQPYPPQPPQ	Immunogenic	Native	DQ2	62	12
924	gamma-secalin Rye peptide R13 PEQPYPEQPPQ	Immunogenic	Deamidated	DQ2	62	12
925	gamma-secalin Rye peptide R23 PQTQQPQQPPQPPQPPQLF	Immunogenic	Native	DQ2	62	20
926	gamma-secalin Rye peptide R23 PQTQQPQQPPQ	Immunogenic	Native	DQ2	62	12
927	gamma-secalin Rye peptide R27 PQEPQQLFPQSQQPQQPPQ	Immunogenic	Native	DQ2	62	20
928	gamma-secalin Rye peptide R27 PQSQQPQQPPQ	Immunogenic	Native	DQ2	62	12
929	gamma-secalin Rye peptide R17 QTQQSIPQQPPQPPQPPF	Immunogenic	Native	DQ2	62	20
930	gamma-secalin Rye peptide R17 QSIPQQPPQPPQ	Immunogenic	Native	DQ2	62	12
931	gamma-secalin Rye peptide R02 SIPQQPPQPPQPPQPPQSQ	Immunogenic	Native	DQ2	62	20
932	gamma-secalin Rye peptide R02 SIPQQPPQPPQPEQPPQSQ	Immunogenic	Deamidated	DQ2	62	20
933	gamma-secalin Rye peptide R02 GQQPPQPEQPPQSG	Immunogenic	Deamidated	DQ2	62	16
934	gamma-secalin Rye peptide R02 QPPQPEQPPQS	Immunogenic	Deamidated	DQ2	62	13
935	gamma-secalin Rye peptide R02 QPPQPPQPPQ	Immunogenic	Native	DQ2	62	12
936	gamma-secalin Rye peptide R02 QPPQPEQPPQ	Immunogenic	Deamidated	DQ2	62	12
937	omega-Secalin Rye peptide R07 QYSPYQPQQPPQPPQPTPI	Immunogenic	Native	DQ2	62	20
938	omega-Secalin Rye peptide R07 GQYSPYQPEQPPQPG	Immunogenic	Deamidated	DQ2	62	16
939	omega-Secalin Rye peptide R07 YSPYQPQQPPQ	Immunogenic	Native	DQ2	62	12
940	omega-Secalin Rye peptide R07 YSPYQPEQPPQ	Immunogenic	Deamidated	DQ2	62	12
941	omega-Secalin Rye peptide R03 GQQPPQPEQPTPIQG	Immunogenic	Deamidated	DQ2	62	16

942	omega-Secalin Rye peptide R03, R04	Immunogenic	Native	DQ2	62	20	
	QPFQPQQPTPIQPQQPFPQ						
943	omega-Secalin Rye peptide R03	Immunogenic	Deamidated	DQ2	62	12	
	QPFQPQEQTPI						
944	omega-Secalin Rye peptide R03	Immunogenic	Native	DQ2	62	12	
	QPFQPQQPTPI						
945	omega-Secalin Rye peptide R04	Immunogenic	Deamidated	DQ2	62	16	
	GQPTPIQPEQPFPQQG						
946	omega-Secalin Rye peptide R04	Immunogenic	Native	DQ2	62	12	
	PTPIQPQQPFPQ						
947	omega-Secalin Rye peptide R04	Immunogenic	Deamidated	DQ2	62	12	
	PTPIQPEQPFPQ						
948	omega-Secalin Rye peptide R01, R09	Immunogenic	Native	DQ2	62	20	
	QQLPLQPQQPFPQPQQPIQ						
949	omega-Secalin Rye peptide R09	Immunogenic	Native	DQ2	62	12	
	QLPLQPQQPFPQ						
950	omega-Secalin Rye peptide R01	Immunogenic	Native	DQ2	62	12	
	QPFQPQQPIQ						
951	omega-Secalin Sec-gamma1	Immunogenic	Native	DQ2	8	14	
	PQQPQQSFQPPQR						
952	omega-Secalin Sec-gamma1 in Deamidated form	Immunogenic	Deamidated	DQ2	8	14	
	PEQPQQSFQPPQR						
953	omega-Secalin Sec-gamma1 in Deamidated form	Immunogenic	Deamidated	DQ2	8	14	
	PQQPEQSFQPPQR						
954	omega-Secalin Sec-gamma1 in Deamidated form	Immunogenic	Deamidated	DQ2	8	14	
	PQQPQQSFPEQPQR						
955	omega-Secalin Sec-gamma1 in Deamidated form	Immunogenic	Deamidated	DQ2	8	14	
	PEQPEQSFQPPQR						
956	omega-Secalin Sec-gamma1 in Deamidated form	Immunogenic	Deamidated	DQ2	8	14	
	PEQPQQSFPEQPQR						
957	omega-Secalin Sec-gamma1 in Deamidated form	Immunogenic	Deamidated	DQ2	8	14	
	PQQPEQSFPEQPQR						
958	omega-Secalin Sec-gamma1 in Deamidated form	Immunogenic	Deamidated	DQ2	8	14	
	PEQPEQSFPEQPQR						
959	omega-Secalin Rye peptide R20	Immunogenic	Native	DQ2	62	20	
	EQIISQQPFLQPQQPFSQ						
960	omega-Secalin Rye peptide R20	Immunogenic	Native	DQ2	62	12	
	PFPLQPQQPFSQ						
961	omega-Secalin Rye peptide R6	Immunogenic	Deamidated	DQ2	62	16	
	GQPQQPFPEQPEQIIG						
962	omega-Secalin Rye peptide R06, R11, R16	Immunogenic	Native	DQ2	62	20	
	PQQPFPQQPEQIIPQQPQQP						
963	omega-Secalin Rye peptide R6	Immunogenic	Native	DQ2	62	12	
	PQQPFPQQPEQI						
964	omega-Secalin Rye peptide R6	Immunogenic	Deamidated	DQ2	62	12	
	PQQPFPEQPEQI						
965	omega-Secalin Rye peptide R11	Immunogenic	Native	DQ2	62	16	
	QQPFPQQPEQIIPQQP						
966	omega-Secalin Rye peptide R11	Immunogenic	Deamidated	DQ2	62	16	
	EQPFPEQPEQIIPQQP						

967	omega-Secalin Rye peptide R11	Immunogenic	Deamidated	DQ2	62	16
	GQFPQQPEQIIPQQG					
968	omega-Secalin Rye peptide R11	Immunogenic	Deamidated	DQ2	62	12
	PFPQQPEQIIPQ					
969	omega-Secalin Rye peptide R16	Immunogenic	Native	DQ2	62	12
	PEQIIPQQPQQP					
970	omega-Secalin Rye peptide R08	Immunogenic	Native	DQ2	62	20
	SQQPQRPPQQPFPQQPQQIIP					
971	omega-Secalin Rye peptide R08	Immunogenic	Native	DQ2	62	13
	RPQQPFPQQPQQI					
972	omega-Secalin Rye peptide R15	Immunogenic	Native	DQ2	62	20
	QPQQIIPQQPQQPFLQPQQ					
973	omega-Secalin Rye peptide R15	Immunogenic	Native	DQ2	62	12
	IIPQQPQQPFL					
974	omega-Secalin Rye peptide R14, R19	Immunogenic	Native	DQ2	62	20
	QQPQQPFLQPQQPVPQQPQ					
975	omega-Secalin Rye peptide R14	Immunogenic	Native	DQ2	62	16
	QQPQQPFLQPQQPVP					
976	omega-Secalin Rye peptide R19	Immunogenic	Native	DQ2	62	16
	QPFPLQPQQPVPQQPQ					
977	omega-Secalin Rye peptide R18	Immunogenic	Native	DQ2	62	20
	QQPFLQPQQPFSQPQQPFL					
978	omega-Secalin Rye peptide R18	Immunogenic	Native	DQ2	62	11
	FLLQPQQPFSQ					
979	omega-Secalin Rye peptide R24	Immunogenic	Native	DQ2	62	20
	SPQQPQLPFPQQPQPFVVVV					
980	omega-Secalin Rye peptide R24	Immunogenic	Deamidated	DQ2	62	20
	SPQQPQLPFPQQPQPFVVVV					
981	omega-Secalin Rye peptide R24	Immunogenic	Native	DQ2	62	12
	LPFPQQPQPFVV					
982	omega-Secalin Rye peptide R24	Immunogenic	Deamidated	DQ2	62	12
	LPFPQQPQPFVV					
983	gamma-avenin Av-alpha9B in native form	Immunogenic	Native	DQ2	8	14
	QYQPYPEQQPQPFVQ					
984	gamma-avenin Av-alpha9B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14 QYQPYPEQQPQPFVQ					
985	gamma-avenin Homolog of oat avenin-derived T cell-stimulatory peptide in Deamidated form	Immunogenic	Deamidated	DQ2	62	15
	EYQPYPEQQPILQQ					
986	gamma-avenin Homolog of oat avenin-derived T cell-stimulatory peptide in native form	Immunogenic	Native	DQ2	62	15
	QYQPYPPQQQPILQQ					
987	ave-1b gliadin alpha avenin-9	Immunogenic	Native	DQ2.5	90,18,8 9	PYPEQQQPF
988	ave-1b gliadin alpha avenin-9	Immunogenic	Deamidated	DQ2.5	90,18,8 9	PYPEQQQPF
	PYPEQQQPF					
989	Avenin T cell recognized Avenin epitope HPLC fraction 9	Immunogenic	Native	DQ2	18	
	31 TTTVQYDPSEYQPYPEQQEPFVQQPQPFVQ					
990	Avenin T cell recognized Avenin epitope HPLC fraction 4	Immunogenic	Native	DQ2	18	
	22 TTTVQYDPSEYQPYPEQQEPF					
991	Avenin T cell recognized Avenin epitope HPLC fraction 9	Immunogenic	Native	DQ2	18	
	28 TTTVQYNPSEYQPYPEQQEPFVQQQPF					
992	Avenin T cell recognized Avenin epitope HPLC fraction 9	Immunogenic	Native	DQ2	18	
	27 TTVQYNPSEYQPYPEQQEPFVQQQPF					

993	Avenin	T cell recognized Avenin epitope HPLC fraction 9	Immunogenic	Native	DQ2	18	
	25	VQYNPSEQYQPYPEQQEPFVQQQPF					
994	Avenin	T cell recognized Avenin epitope HPLC fraction 3	Immunogenic	Native	DQ2	18	
	22	TTTVQYNPSEQYQPYPEQQEPF					
995	Avenin	T cell recognized Avenin epitope HPLC fraction 8	Immunogenic	Native	DQ2	18	
	29	TTTVQYDPSEQYQPYPEQQEPFVQQQPF					
996	Avenin	T cell recognized Avenin epitope HPLC fraction 8	Immunogenic	Native	DQ2	18	
	30	TTTVQYNPSEQYQPYPEQQEPFVQQQPFV					
997	Avenin	T cell recognized Avenin epitope HPLC fraction 9	Immunogenic	Native	DQ2	18	
	29	PSEQYQPYPEQQEPFVQQQPFVQQQPF					
998	Avenin	Avenin 1490 in native form	Immunogenic	Native	DQ2	18	19
		SEQYQPYPEQQEPFVQQQ					
999	Avenin	Avenin 1490 in Deamidated form	Immunogenic	Deamidated	DQ2	18	
	19	SEQYQPYPEQEEPFFVQQQ					
1000	Avenin	Av-alpha9A in native form	Immunogenic	Native	DQ2	8	14
		QYQPYPEQQEPFVQ					
1001	Avenin	Av-alpha9A in Deamidated form	Immunogenic	Deamidated	DQ2	8	14
		QYQPYPEQEEPFFVQ					
1002	Avenin	Avenin 1505	Immunogenic	Native	DQ2	18	12 YQPYPEQQEPFV
1003	Avenin	Avenin 1504 (deamidated form of Avenin 1505)	Immunogenic	Deamidated	DQ2	18	12 YQPYPEQEEPFFV
1004	ave-1	gliadin alpha avenin-9	Immunogenic	Native	DQ2.5	90,18,8	9 PYPEQQEPF
1005	ave-1	gliadin alpha avenin-9	Immunogenic	Deamidated	DQ2.5	90,18,8	9 PYPEQEEPFF
1006	Avenin	Av-gamma2B	Immunogenic	Native	DQ2	8	14
		QQPFVQQQPFVQQ					
1007	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	EQPFVQQQPFVQQ					
1008	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	QQPFVEQQQPFVQQ					
1009	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	EQPFVEQQQPFVQQ					
1010	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	QQPFVQEQQPFVQQ					
1011	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	EQPFVQEQQPFVQQ					
1012	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	QQPFVEEQQPFVQQ					
1013	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	EQPFVEEQQPFVQQ					
1014	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	QQPFVQQEQPFVQQ					
1015	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	EQPFVQQEQPFVQQ					
1016	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	QQPFVEEQQPFVQQ					
1017	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	EQPFVEEQQPFVQQ					
1018	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	QQPFVQEEQPFVQQ					

1019	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	EQPFVQEEQPFVQQ					
1020	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	QQPFVEEEQPFVQQ					
1021	Avenin	Av-gamma2B in Deamidated form	Immunogenic	Deamidated	DQ2	8	
	14	EQPFVEEEQPFVQQ					
1022	Avenin	Avenin core epitope in native form	Immunogenic	Native	DQ2	27	9
		FVQQQQQPF					
1023	Avenin	Avenin core epitope in Deamidated form	Immunogenic	Deamidated	DQ2	27	9
		FVQQQEQPF					
1024	gamma-gliadin or LMW glutenin	Glu-21 minimal epitope in considered native form	Immunogenic	Native	DQ2	19	12
		QSEQSQQPFQPFQ					
1025	glia-gamma 3, glia-gamma 1b	gamma gliadin	Immunogenic	Deamidated	DQ2.5 or DQ8	90,17,23	9
		EQPQQPYYPQ					
1026	glia-gamma 3, glia-gamma 1b	gamma gliadin	Immunogenic	Deamidated	DQ2.5 or DQ8	90,17,23	9
		QQPQQPYPE					
1027	glia-gamma 3, glia-gamma 1b	gamma gliadin	Immunogenic	Deamidated	DQ2.5 or DQ8	90,78,23	9
		EQPEQPYYPQ					
1028	glia-gamma 3, glia-gamma 1b	CAUTION, 100% match to Candida protein / gamma gliadin	Immunogenic	Deamidated	DQ2.5 or DQ8	90,78,23	9
		QQPEQPYPE					
1029	glia-gamma 3	gamma 5 gliadin	Immunogenic	Deamidated	DQ2.5	90,78,23	9
		SQPEQQFPQ					
1030	glia-gamma 3	gamma 5 gliadin	Immunogenic	Deamidated	DQ2.5	90,78,23	9
		SQPQQEFPQ					
1031	glia-gamma 1b, glia-gamma 4c	gammaVII-gliadin	Immunogenic	Deamidated	DQ2.5	90,17,25	9
		QQPQQPFPE					
1032	glia-gamma 4c, glia-gamma 1b	gammaVII-gliadin	Immunogenic	Deamidated	DQ2.5	90,17,25,8	9
		QQPEQPFPE					
1033	glia-gamma 4c, glia-gamma 1b	gammaVII-gliadin	Immunogenic	Deamidated	DQ2.5	90,17,25	9
		EQPEQPFPE					
1034	glia-gamma 4c	gamma gliadin	Immunogenic	Native	DQ2.5	90,62,78,81	9
		PQPQQPFCQ					
1035	glia-gamma 4d	gamma gliadin	Immunogenic	Deamidated	DQ2.5	90,62,78,81	9
		PQPEQPFCE					
1036	glia-gamma 4d	gamma gliadin	Immunogenic	Deamidated	DQ2.5	90,62,78,81	9
		PQPQQPFCE					
1037	glia-gamma 4d	gamma gliadin	Immunogenic	Deamidated	DQ2.5	90,62,78,81	9
		PQPEQPFCE					
1038	glia-gamma 4e	gliadin omega 1	Immunogenic	Native	DQ2.5	90,86	9
		PQPQQPFSQ					
1039	glia-gamma 4e	gliadin omega 1	Immunogenic	Deamidated	DQ2.5	90,86	9
		PQPEQPFSE					
1040	glia-omega 3	gliadin omega 3	Immunogenic	Native	DQ2.5	86,90	9
		PFPQPQQPI					
1041	glia-omega 3	gliadin omega 3	Immunogenic	Deamidated	DQ2.5	86,90	9
		PFPQPQQPI					
1042	glia-omega 4	CAUTION 100% match to 2 Prunus sp. peptides / gliadin omega 4	Immunogenic	Native	DQ2.5	86,90	9
		PQPQQPIPV					
1043	glia-omega 4	gliadin omega 4	Immunogenic	Deamidated	DQ2.5	86,90	9
		PQPEQPIPV					
1044	glia-omega 5	gliadin omega 5	Immunogenic	Native	DQ2.5	86,90	9
		LQPQQPFPQ					

1045	glia-omega 5 LQPEQPFPPQ	gliadin omega 5	Immunogenic	Deamidated	DQ2.5	86,90	9
1046	Avenin Q QQPFMQQQQPFMQP	avenin-gliadin like	Immunogenic	Native	DQ2 or DQ8	87	14
1047	Avenin Q-5 QQPFVQQQQQPFVQ	avenin-gliadin like	Immunogenic	Native	DQ2 or DQ8	88	14
1048	glia-gamma 1 PEQSFPQQQ	gamma gliadin 1	Immunogenic	Deamidated	DQ2.5	90,74	9
1049	glia-gamma 1 PEQSFPQQE	gamma gliadin	Immunogenic	Deamidated	DQ2.5	90,74	9
1050	ave-1 06 QYQPYPEQQQPILQQQ	Avenin gliadin like	Immunogenic	Native	DQ2.5	88	16
1051	ave-1 06 QYQPYPEQQQPILQQQ	avenin-gliadin like	Immunogenic	Deamidated	DQ2.5	88	16
1052	ave-1 04 QQYQYPQQQPFMQPL	avenin-gliadin like	Immunogenic	Native	DQ2.5	88	16
1053	ave-1 04 EQYQPYPEQQQPFMQPL	avenin-gliadin like	Immunogenic	Deamidated	DQ2.5	88	16

CD1D multimer and ligand:

[00104] In one embodiment, the peptide is a human, non-human, or synthetic/engineered peptide. For example, the peptide is a Minor Histocompatibility Antigen (MiHA).

[00105] Exemplary MiHAs are known in the art, e.g., in Spierings et al. Tissue Antigens 2014 84:347-360; which is incorporated by reference herein. MiHAs are typically utilized in embodiments relating to transplantation, where it refers to epitopes that are created because of protein sequence variation in polymorphic proteins.

[00106] Residues of the antigenic peptide that engages the MHC binding groove can loosely be divided into two types: anchor residues, which engage with the MHC molecule and confer stability to the MHC-peptide complex (for example, see residues P2, P3, P5, P6, P7, and P9 in Bowness et al. 1999 Expert Reviews in Molecular Medicine 16:1-10; which is incorporated by reference herein in its entirety), and interfacial residues, which are solvent-exposed and can engage with the cognate T-cell receptor (see, e.g., residues P1, P4, and P8 in Bowness). A featureless peptide is a peptide in which anchor residues are preserved, while interfacial residues of the peptide are mutated to alanine or glycine residues to prevent TCR binding. A featureless peptide-MHC is therefore a MHC peptide complex in which the presented peptide is a featureless peptide. Featureless peptide MHCs are typically used in embodiments relating to transplantation tolerance. In patients receiving MHC-mismatched solid organ or hematopoietic stem cell transplants, use of a CAL T cell presenting the relevant donor-mismatched featureless peptide-MHC CAL can permit selective depletion of recipient alloreactive T cells targeted towards this mismatched donor HLA allele.

[00107] The MHC can be a monomer, dimer, trimer, tetramer, pentamer, dextramer or other oligomer form. In embodiments where more than one MHC unit is present, e.g., the MHC is oligomeric, the units can be provided in series, e.g., in a chain, or provided arrayed in one or more

dimensions around a central point or linker, provided conjugated/bound in any geometry to a scaffold molecule, or any combination of the foregoing. Naturally occurring examples of TCR recognition domain structures in the art include MHC dimers (Lebowitz et al., 1999 Cellular Immunology 192:175-184), tetramers (Altman et al., 1996 Science 274:94-96), pentamers (proimmune.com/introduction-to-pentamers/), octamers (Guillame et al., 2003 JBC 278:4500-4509), dextramers (Batard et al., 2006 Journal of Immunological Methods 310:136-148), dodecamers (Huang et al., 2016 PNAS 113:E1890-7), lipid vesicles (Mallet-Designé et al., 2003 The Journal of Immunology 170:123-131), and quantum dots (Chattopadhyay et al., 2006 Nature Medicine 12:972-7). Each of the foregoing references is incorporated by reference herein in its entirety.

[00108] In some embodiments, the TCR recognition domain can comprise a CD1 domain (e.g., a CD1d domain), e.g., a sequence comprising an extracellular domain of CD1, (e.g., CD1). As used herein, “cluster of differentiation 1 family member d” or “CD1d” refers to a cell surface protein that displays lipid antigens to T cells. The sequences of several CD1d isoforms, and the structure of CD1d are known in the art, see, e.g., the 3 isoforms provided in the NCBI database for CD1d (Gene ID 912), and Bagchi et al., 2018 and Oleinika et al., Nature Communications 2018 9:684; which are incorporated by reference herein in their entireties. For example, isoform 1 of CD1d is SEQ ID NO: 5 (NCBI Ref Seq NP_001757.1), isoform 2 of CD1d is SEQ ID NO: 6 (NCBI Ref Seq NP_001306074.1), and isoform 3 of CD1d is SEQ ID NO: 7 (NCBI Ref Seq NP_001358690.1). In some embodiments, the CD1d domain comprises a sequence with at least 80%, at least 85%, at least 90%, at least 95%, at least 98% or greater sequence identity to one of SEQ ID NOs: 5-7. In some embodiments, the CD1d domain comprises a sequence with at least 80%, at least 85%, at least 90%, at least 95%, at least 98% or greater sequence identity to the extracellular domain of one of SEQ ID NOs: 5-7 (e.g., amino acids 20-301 of SEQ ID NO: 5). In some embodiments, the CD1d domain comprises a sequence with at least at least 95% sequence identity to one of SEQ ID NOs: 5-7 and retains the lipid binding activity of the wild-type reference sequence. In some embodiments, the CD1d domain comprises a sequence with at least 95% sequence identity to the extracellular domain of one of SEQ ID NOs: 5-7 (e.g., amino acids 20-301 of SEQ ID NO: 5) and retains the lipid binding activity of the wild-type reference sequence.

[00109] In some embodiments, the CD1 domain further comprises a ligand, e.g., a non-peptide ligand. Exemplary, non-limiting ligands of CD1 domains include but are not limited to:

Protein (Allele) Ligand

human CD1a	C-glycoside GSL-1 OCH
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human CD1b	C-glycoside GSL-1 OCH
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human CD1c	C-glycoside GSL-1 OCH
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human CD1d	OCH
human CD1d	PBS-57

[00110] In some embodiments, a TCR recognition domain can comprise sequences or molecules in addition to the, e.g., MHC, pMHC, of CD1 domains and sequences. For example, it can further comprise other polypeptide and/or non-polypeptide components that enable multimerization.

Exemplary components that permit multimerization can include biotin (non-polypeptide) and/or streptavidin polypeptide that is used to permit tetramerization.

[00111] In various embodiments, protein interaction domains are found on an extracellular portion of the respective polypeptides.

[00112] As used herein, “recognition polypeptide” refers to an extracellular polypeptide having a ligand-binding domain. In some embodiments, the ligand-binding domain can be an antibody reagent. In some embodiments, the recognition polypeptide can further comprise a protein interaction domain.

[00113] As used herein, “signaling polypeptide” refers to a transmembrane polypeptide having an intracellular signaling domain, e.g., a T cell receptor (TCR) signaling domain. In some embodiments, the signaling polypeptide can further comprise a protein interaction domain. In some embodiments, the signaling polypeptide can further comprise an extracellular protein interaction domain.

[00114] As used herein, “biomolecular interaction domain” refers to a domain that permits specific binding of two separate molecules to each other. The molecules can be or can comprise polypeptides. In some embodiments, one or both of the molecules or biomolecular interaction domains can be a non-peptide. When a pair of biomolecular interaction domains is provided herein, they permit two or more molecules to bind specifically, e.g. one of the biomolecular interaction domains can bind specifically to the second biomolecular interaction domain. In some embodiments, specific binding can occur when two separate biomolecular interaction domains, e.g., of a pair, are present. In some embodiments, specific binding can occur when three or more separate biomolecular interaction domains are present. It is noted that protein interaction domains are a type of biomolecular interaction domains and where one is specified herein, the other may always be substituted.

[00115] As used herein, when a molecule is referred to as a protein or polypeptide, it comprises a protein, peptide, or polypeptide sequence, but may comprise additional motifs, modifications, or domains of a non-proteinaceous nature. A number of exemplary biomolecular interaction domains, as well as pairs of protein interaction domains are provided elsewhere herein. In some embodiments, the biomolecular interaction domains comprise, consist, or consist essentially of proteins or

polypeptides. In some embodiments, the biomolecular interaction domains comprise, consist, or consist essentially of non-proteinaceous molecules. In some embodiments, one of a pair of biomolecular interaction domains can comprise, consist, or consist essentially of proteins or polypeptides and the second of the pair of biomolecular interactions domains can comprise, consist, or consist essentially of a non-proteinaceous molecule (e.g., FITC and anti-FITC). Exemplary protein interaction domains are known in the art and can be used in embodiments of the aspects described herein.

[00116] As used herein, “protein interaction domain” refers to a domain that permits specific binding of two separate polypeptides to each other. A number of exemplary protein interaction domains, as well as pairs of protein interaction domains are provided elsewhere herein. In some embodiments, the protein interaction domains of the polypeptides of a multi-component CAL and/or CAR can bind specifically, e.g. one of the protein interaction domains can bind specifically to a second protein interaction domain of the multi-component CAL and/or CAR. In some embodiments, specific binding can occur when two separate protein interaction domains are present. In some embodiments, specific binding can occur when three or more separate protein interaction domains are present. Exemplary protein interaction domains are known in the art and can be used in embodiments of the aspects described herein.

[00117] In some embodiments of any of the aspects described herein, the protein interaction domains can be leucine zipper domains. Leucine zipper domains are a type of protein-protein interaction domain commonly found in transcription factors characterized by leucine residues evenly spaced through a α -helix. Leucine zippers may form heterodimers or homodimers. A number of leucine zipper domains, as well as their ability to bind each other, are known in the art and discussed further, e.g., in Reinke et al. JACS 2010 132:6025-31 and Thompson et al. ACS Synth Biol 2012 1:118-129; each of which is incorporated by reference herein in its entirety. In some embodiments, one leucine zipper domain is BZip (RR) and the second leucine zipper domain is AZip (EE). In some embodiments, the sequence of a BZip (RR) leucine zipper domain is MDPDLEIRAAFLRQRNTALRTEVAELEQEVQRLNEVSQYETRYGPLGGGK (SEQ ID NO: 3). In some embodiments, the sequence of a AZip (EE) leucine zipper domain is MDPDLEIEAAFLERENTALETRVAELRQRVQRLNRVSQYRTRYGPLGGGK (SEQ ID NO: 4). Further exemplary leucine zipper domains are described in Reinke et al. JACS 2010 132:6025-31; which is incorporated by reference herein in its entirety. For example, suitable leucine zipper domains can include SYNZIP 1 to SYNZIP 48, and BATF, FOS, ATF4, ATF3, BACH1, JUND, NFE2L3, and HEPTAD. Binding affinities of various combinations of these domains are described, e.g., at Fig. 1 of Reinke et al. In some embodiments, a suitable pair of leucine zipper domains has a dissociation constant (Kd) of 1000 nM or less. In some embodiments, a suitable pair of leucine zipper domains has a dissociation constant (Kd) of 100 nM or less. In some embodiments, a suitable pair of

leucine zipper domains has a dissociation constant (K_d) of 10 nM or less. In some embodiments, a suitable pair of leucine zipper domains has a dissociation constant (K_d) of 1 nM or less.

[00118] Further exemplary pairs of protein interaction domains can include a) PSD95-Dlg1-zo-1 (PDZ) domains; b) a streptavidin domain and a streptavidin binding protein (SBP) domain; and c) a PYL domain and an ABI domain.

[00119] In some embodiments of any of the aspects described herein, the protein interaction domains can be chemically-induced protein interaction domains, e.g., domains that will only bind specifically in the presence of a third molecule, e.g., a small molecule or drug. Exemplary pairs of chemically-induced protein interaction domains can include: FKBP-binding domain of mTOR (FRB) and FK506 binding protein (FKBP) (binding of which is activated by tacrolimus, everolimus, or a rapalog); cyclophilin-Fas fusion protein (CyP-Fas) and FK506 binding protein (FKBP) (binding of which is activated by FKCsA); calcineurin A (CNA) and FK506 binding protein (FKBP) (binding of which is activated by FK506); gibberellin insensitive (GIA) and gibberellin insensitive dwarf1 (GID1) (binding of which is activated by gibberellin); Snap-tag and Halo tag (binding of which is activated by HaXS); and T14-3-3-cdeltaC and C-Terminal peptides of PMA2 (CT52) (binding of which is activated by fusicoccin). Further description of chemically-induced protein interaction domains can be found in the art, e.g., Miyamoto et al. *Nat Chem Biol.* 2012 Mar 25; 8(5): 465–470 and Belshaw et al. *PNAS* 1996 93:4604-4607; each of which is incorporated herein by reference in its entirety.

[00120] In some embodiments of any of the aspects described herein, the protein interaction domains can comprise at least one nucleotide tag and at least one zinc finger domain. Zinc finger domains are characterized by the coordination of a zinc ion in order to stabilize their tertiary structure. The particular folds that appear in zinc fingers can vary. In some embodiments, a zinc finger domain can be a nucleotide-binding zinc finger domain. In some embodiments, a zinc finger domain can be a DNA-binding zinc finger domain. In some embodiments, the protein interaction domain of the recognition polypeptide is a nucleotide tag and the extracellular protein interaction domain of the signaling polypeptide is a zinc finger domain. In some embodiments, a nucleotide tag can be a DNA tag. In some embodiments, a nucleotide tag can be a dsDNA tag comprising the entire recognition sequence for the zinc finger domain being used. Exemplary zinc finger domains and their cognate nucleotide tags are described in the art, e.g. Mali et al. *Nature Methods* 2013 10:403-406; which is incorporated by reference herein in its entirety. In some embodiments, a zinc finger domain can be sZF15 as described in Mali et al. *Nature Methods* 2013 10:403-406.

[00121] In one embodiment, the protein interaction domain(s) is a BZip (RR) and/or a AZip (EE), or any binding pair of protein interaction domains are collectively a BZip (RR) and a AZip (EE).

[00122] In some embodiments of any of the aspects described herein, the protein interaction domains can comprise a pair of substantially complementary nucleotide tags, e.g., fully complementary or complementary enough to hybridize specifically. The degree of complementarity

necessary may vary depending on the total length of the tags and G/C content of the complementary portions. One of skill in the art can readily determine the relevant affinity necessary for tags of a given size and G/C content. In some embodiments, a nucleotide tag can be a DNA tag.

[00123] In some embodiments, a nucleotide tag can be a DNA tag. In some embodiments, a nucleotide tag can be a dsDNA tag.

[00124] In one embodiment, the protein interaction domain(s) is a gibberellin insensitive (GIA) and/or a gibberellin insensitive dwarf1 (GID1) or any binding pair of protein interaction domains are collectively a gibberellin insensitive (GIA) and a gibberellin insensitive dwarf1 (GID1).

[00125] In one embodiment, the protein interaction domain(s) is a Snap-tag and/or a Halo tag, or any binding pair of protein interaction domains are collectively a Snap-tag and a Halo tag.

[00126] In one embodiment, the protein interaction domain(s) is a T14-3-3-cdeltaC and/or a C-Terminal peptides of PMA2 (CT52), or any binding pair of protein interaction domains are collectively a T14-3-3-cdeltaC and a C-Terminal peptides of PMA2 (CT52).

[00127] In one embodiment, the protein interaction domain(s) is a PYL and/or a ABI, or any binding pair of protein interaction domains are collectively a PYL and a ABI.

[00128] In one embodiment, the biomolecular interaction domain(s) is a Fluorescein isothiocyanate (FITC) and/or a FITC binding protein or any binding pair of biomolecular interaction domains are collectively a FITC and a FITC binding protein.

[00129] In one embodiment, the biomolecular interaction domain(s) is a (R)-Phycoerythrin (R-PE/PE) and/or a R-PE/PE binding protein or any binding pair of biomolecular interaction domains are collectively a (R)-Phycoerythrin (R-PE/PE) and/or a R-PE/PE binding protein.

[00130] In some embodiments, the biomolecular domain on the molecule comprising a TCR recognition domain and biomolecular interaction domain (i.e. a CAL) binds specifically to a native cell surface molecule on a NK cell, dendritic cell, or T cell. In such embodiments, a subject can be treated as described herein without administration of an engineered cell. In some embodiments, the biomolecular domain on the molecule comprising a TCR recognition domain and biomolecular interaction domain (i.e. a CAL) binds specifically to a native cell surface molecule on a NK cell. In some embodiments, the biomolecular domain on the molecule comprising a TCR recognition domain and biomolecular interaction domain (i.e. a CAL) binds specifically to a native cell surface molecule on dendritic cell. Exemplary native cell surface molecules for such embodiments include, but are not limited to CD3. Other suitable cell surface molecules are the CD cell surface proteins. CDs are known in the art and one of ordinary skill can readily select one expressed by the desired cell type(s). For example, see the HCDM database at hcdm.org and the lists available on the world wide web at chemeurope.com/en/encyclopedia/List_of_human_clusters_of_differentiation.html; and docs.abcam.com/pdf/immunology/Guide-to-human-CD-antigens.pdf. Accordingly, exemplary biomolecular interaction domains for such embodiments include but are not limited to an anti-CD3

antibody reagent, or a Fab domain. One of skill in the art is aware of other cell surface molecules found, or found exclusively, on the cell surface of the relevant cell type(s), as well as multiple reagents that can bind (e.g., bind specifically) to such cell surface molecules.

[00131] In aspects with a single recognition polypeptide and a single signaling polypeptide that are able to bind specifically without a third polypeptide, the multiple-component CALs or CARs described herein will activate in the presence of the target ligand, thereby inducing T cell activity in the vicinity of the target ligand. Further described herein are multiple-component CALs or CARs capable of logic computation, for example, multiple-component CALs or CARs that serve as AND, OR, or NOT logic gates.

[00132] In some aspects, described herein are compositions that comprise components of a multi-component CAL and/or CAR that permits AND gate logic. In these aspects, activation of the multi-component CAL and/or CAR happens only in the presence of two target ligands; recognition of a single target ligand is not sufficient for activation. Such multi-component CALs or CARs can permit greater specificity and reduce off-target effects. Any single ligand that is a good marker for a target cell or tissue may occur elsewhere in a subject, resulting in off-target effects. However, requiring the recognition of two separate marker ligands reduces the odds of off-target activity.

[00133] In one embodiment, a nucleotide tag can be a DNA tag or dsDNA tag.

[00134] Further embodiments of AND logic gate multi-component CALs and CARs are described herein.

[00135] In some embodiments of any of the aspects described herein, the compositions comprise components of a multi-component CAL and/or CAR that are NOT logic gate. For example, recognition of a second target ligand by a second recognition polypeptide can prevent interaction (e.g. specific binding) of the signaling polypeptide and first recognition polypeptide. Such embodiments can permit suppression of T cell activity in inappropriate and/or off-target tissues. For example, the second target ligand can be a marker of a tissue that is particularly sensitive to T cell activity, is a known area of off-target activity, and/or shares markers with the desired target tissue. In some embodiments, in a NOT gate multi-component CAL and/or CAR, the second target ligand is not a ligand found in the target tissue and/or cells, e.g., in or on a disease T cell. In some embodiments, the second target ligand of a NOT logic gate multi-component CAL and/or CAR is found on a healthy and/or non-target cell and not on a diseased and/or target cell. Various 2- and 3-dimensional configurations of such pairs of nucleotide pairs are known in the art.

[00136] In some embodiments, the target ligand recognized by the second recognition polypeptide is found on a healthy and/or non-target cell and not on a diseased and/or target cell. In some embodiments, the protein interaction domain of the second recognition polypeptide and the protein interaction domain of the first recognition polypeptide have a greater affinity than the protein interaction domain of the signaling polypeptide and the protein interaction domain of the first

recognition polypeptide. In some embodiments, the protein interaction domain of the second recognition polypeptide and the protein interaction domain of the signaling polypeptide have a greater affinity than the protein interaction domain of the signaling polypeptide and the protein interaction domain of the first recognition polypeptide. Relative binding affinities can be determined experimentally, e.g., by binding affinity assays known in the art and relative binding affinities are known for a number of combinations of protein interaction domains described herein, see, e.g. Reinke et al. JACS 2010 132:6025-31; which is incorporated by reference herein in its entirety. In some embodiments, the binding affinity of the recognition polypeptide protein interaction domains can be at least 2x greater than the binding affinity of the first recognition polypeptide protein interaction domain and the signaling polypeptide interaction domain. In some embodiments, the binding affinity of the recognition polypeptide protein interaction domains can be at least 5x greater than the binding affinity of the first recognition polypeptide protein interaction domain and the signaling polypeptide interaction domain. In some embodiments, the binding affinity of the recognition polypeptide protein interaction domains can be at least 10x greater than the binding affinity of the first recognition polypeptide protein interaction domain and the signaling polypeptide interaction domain.

[00137] As used herein, “target ligand” refers to a molecule in or on a cell which can be bound by a ligand-binding domain. Non-limiting examples of such molecules can include polypeptides, lipids, saccharides, and the like. In some embodiments, the target ligand can be an extracellular molecule. In some embodiments, the target ligand can be a cell surface molecule.

[00138] In some embodiments, e.g., those relating to a multi-component CAL and/or CAR with a single recognition polypeptide or an AND gate multi-component CAL and/or CAR, the target ligand (e.g. the first and/or second target ligand) can be a ligand expressed in a target tissue. In some embodiments, the target ligand can be expressed constitutively in the target tissue and/or cell. In some embodiments, the target ligand can be expressed exclusively in the target tissue and/or cell. In some embodiments, the target ligand can be expressed at a higher level in the target tissue and/or cell than in other tissues and/or cells. As recognition of a target ligand in embodiments relating to a multi-component CAL and/or CAR with a single recognition polypeptide or an AND gate multi-component CAL and/or CAR can result in T cell activation (e.g. cell killing activity of the cell comprising the target ligand), the target ligand can be selected to target T cell activity in a desirable and/or therapeutic way, e.g., by targeting a disease cell. In some embodiments, a target ligand is a ligand found in/on a diseased and/or target cell. In some embodiments, the target ligand specifically bound by a recognition polypeptide that can specifically bind with a signaling polypeptide or is a portion of an AND gate multi-component CAL and/or CAR is a ligand found in/on a diseased and/or target cell. In some embodiments, a target ligand specifically bound by a recognition polypeptide that can specifically bind with a signaling polypeptide or is a portion of an AND gate multi-component CAL and/or CAR is a ligand found on a diseased and/or target cell and not on a healthy and/or non-target

cell. In some embodiments, the diseased cell is an autoreactive or alloreactive T cell. In some embodiments, the target ligand specifically bound by a recognition polypeptide that can specifically bind with a signaling polypeptide or is a portion of an AND gate multi-component CAL and/or CAR is found on the surface of a disease cell. In some embodiments, a recognition polypeptide that can specifically bind with a signaling polypeptide or is a portion of an AND gate multi-component CAL and/or CAR specifically binds to a target ligand on the surface of a disease cell, e.g. as compared to binding to normal cells.

[00139] In some embodiments, a composition and/or cell described herein can further comprise a second multi-component CAL and/or CAR according to any of the aspects and embodiment described herein for the first multi-component CAL and/or CAR. By way of non-limiting example, a second CAL and/or CAR can be designed to bind specifically to (and, e.g., be activated by or inhibited by) different target ligands than those to which the first multi-component CAL and/or CAR specifically binds (and, e.g. is activated by or inhibited by). This can provide increased specificity, reduced off-target effects, and/or reduced effective dosages for the methods described herein. In some embodiments, the recognition domain of second multi-component CAL and/or CAR bind specifically to different target ligands than those bound by the recognition domain of the first multi-component CAL and/or CAR. In some embodiments, the antibody reagents of second multi-component CAL and/or CAR bind specifically to different target ligands than those bound by the antibody reagents of the first multi-component CAL and/or CAR.

[00140] In some embodiments, the second multi-component CAL and/or CAR can comprise an inhibitory intracellular signaling domain, e.g., T cell receptor (TCR) signaling domain, e.g., one that inhibits engineered cell, e.g., T cell, activity. In such embodiments, the second multi-component can therefore be designed to operate in opposition to the first multi-component CAL and/or CAR, e.g. permitting inhibition of T cell activation while the first multi-component CAL and/or CAR permits activation of T cell activity. Inhibitory intracellular TCR signaling domains are known in the art and can include, by way of non-limiting example, PD1; CTLA4; BTLA; KIR; LAG-3; TIM-3; A2aR; LAIR-1; and TGIT. In one embodiment, non-active mimetics of an activating TCR can be used.

[00141] In some embodiments, described herein is a composition comprising a cell, a CAL, and a CAR (e.g., with the CAR comprising either an inhibitory domain or costimulation/activation domain).

[00142] In one embodiment, the TCR recognition domain comprises a MHC allogeneic to the cell. In one embodiment, the TCR recognition domain comprises a peptide allogeneic to the cell. In one embodiment, the TCR recognition domain comprises a MHC allogeneic to the target cell. In one embodiment, the TCR recognition domain comprises a peptide allogeneic to the target cell. In one embodiment, the TCR recognition domain comprises a non-self peptide relative to the target cell.

[00143] In some embodiments, the target ligand specifically bound by a recognition polypeptide that can specifically bind with the signaling polypeptide of the second multi-component CAL and/or

CAR comprising an inhibitory intracellular signaling domain is a ligand found on a healthy and/or non-target cell. In some embodiments, the target ligand specifically bound by a recognition polypeptide that can specifically bind with the signaling polypeptide of the second multi-component CAL and/or CAR comprising an inhibitory intracellular signaling domain is a ligand found on a healthy and/or non-target cell and not on a diseased and/or target cell. In some embodiments, the second multi-component CAL and/or CAR comprising an inhibitory intracellular signaling domain can be an OR logic gate according to any of the embodiments described herein and the second target ligand can be a ligand found in/on, or specific to, diseased cells.

[00144] In some embodiments of any of the aspects, a ligand-binding domain can comprise or consist essentially of an antibody reagent. In some embodiments, the antibody reagent can be an immunoglobulin molecule, a monoclonal antibody, a chimeric antibody, a CDR-grafted antibody, a human antibody, a humanized antibody, a Fab, a Fab', a F(ab')₂, a Fv, a disulfide linked Fv, a scFv, a single domain antibody, a diabody, a multispecific antibody, a dual specific antibody, an anti-idiotypic antibody, and/or a bispecific antibody.

[00145] In some embodiments, the intracellular signaling domain can be a T-cell activation domain. In some embodiments, the intracellular signaling domain is a signaling domain from a protein selected from the group consisting of: TCR ζ , FcR γ , FcR β , CD3 γ , CD3 δ , CD3 ϵ , CD3 ζ , CD22, CD79a, CD79b, CD66d, CARD11, CD2, CD7, CD27, CD28, CD30, CD40, CD54 (ICAM), CD83, CD134 (OX40), CD137 (4-1BB), CD150 (SLAMF1), CD152 (CTLA4), CD223 (LAG3), CD270 (HVEM), CD273 (PD-L2), CD274 (PD-L1), CD278 (ICOS), DAP10, LAT, NKD2C SLP76, TRIM, and ZAP70. In some embodiments, the signaling domain can be a paralog or ortholog of any of the foregoing.

Multi-Component CAR/CAL Cells

[00146] Presented herein are cells that express the compositions or multi-component CARs and/or CALs presented herein. A cell can be any cell, for example, any mammalian cell, e.g., a human cell. In one embodiment, the cell is a dendritic cell, regulatory T cell, or effector T cell. In one embodiment the cell is a dendritic cell (CAL DC), a T cell (e.g., effector, regulatory, etc.) (CAL-T); regulatory T cell, effector T cell, natural killer cell (CAL NK), or any other myeloid cell.

[00147] In one aspect, described herein is an engineered cell expressing and/or comprising one or more multi-component CARs/CALs, or a composition comprising the same as described herein, e.g., at least one signaling polypeptide and at least one recognition polypeptide. In some embodiments, the cell is a natural killer (NK) cell, dendritic cell, regulatory T cell, effector T cell. Such cells expressing and/or comprising both a signaling polypeptide and at least one recognition polypeptide of a multi-component CAR/CAL are referred to herein as “complete multi-component CAR/CAL” cells. In some embodiments, a complete multi-component CAR/CAL cell expresses both a signaling

polypeptide (e.g., a CAR) and at least one recognition polypeptide (e.g. a CAL or adaptor as described elsewhere herein) of a multi-component CAL and/or CAR. In some embodiments, a complete multi-component CAL and/or CAR cell comprises nucleic acid sequences encoding both a signaling polypeptide and at least one recognition polypeptide of a multi-component CAL and/or CAR. In some embodiments, a signaling polypeptide is present on the membrane of a cell. In some embodiments, the one or more recognition polypeptides are present in the extracellular space, e.g., the recognition polypeptide(s) can be expressed and secreted by the cell or the cell can be contacted by recognition polypeptides provided from another source (e.g. produced synthetically or by another cell and optionally, purified or processed before the contacting step).

[00148] In any of the aspects described herein, e.g., those relating to either a complete or partial multi-component CAL and/or CAR cell, the recognition and/or signaling polypeptide can be under the control of an inducible and/or repressible promoter. Such promoters allow the expression of the polypeptide to be increased or decreased as desired and are in contrast to constitutive promoters. The term "constitutively active promoter" refers to a promoter of a gene which is expressed at all times within a given cell. Exemplary promoters for use in mammalian cells include cytomegalovirus (CMV), Elongation Factor 1a (EF1a), and the like. The term "inducible promoter" refers to a promoter of a gene which can be expressed in response to a given signal, for example addition or reduction of an agent. Non-limiting examples of an inducible promoter are promoters that are regulated in a specific tissue type, a promoter regulated by a steroid hormone, by a polypeptide hormone (e.g., by means of a signal transduction pathway), or by a heterologous polypeptide (e.g., the tetracycline-inducible systems, "Tet-On" and "Tet-Off"; see, e.g., Clontech Inc., CA, Gossen and Bujard, *Proc. Natl. Acad. Sci. USA* 89:5547, 1992, and Paillard, *Human Gene Therapy* 9:983, 1989; each of which are incorporated by reference herein in its entirety). In some embodiments, expression of the polypeptide can be precisely regulated, for example, by using an inducible regulatory sequence that is sensitive to certain physiological regulators, e.g., circulating glucose levels, or hormones (Docherty *et al.*, 1994, *FASEB J.* 8:20-24). Such inducible expression systems, suitable for the control of expression in cells or in mammals include, for example, regulation by ecdysone, by estrogen, progesterone, tetracycline, chemical inducers of dimerization, and isopropyl-beta-D1 - thiogalactopyranoside (IPTG). A person skilled in the art would be able to choose the appropriate regulatory/promoter sequence based on the intended use of the polypeptide.

[00149] In some embodiments, the expression of one or more of the recognition or signaling polypeptides can be constitutive. In some embodiments, the expression of one or more of the recognition or signaling polypeptides can be transient. Transient expression can be achieved by, e.g., use of transient and/or inducible expression promoters or by use of transient vectors, e.g. those that do not incorporate into the genome and/or persist in the target cell. By way of non-limiting example, derivatives of viruses such as the bovine papillomavirus (BPV-1), or Epstein-Barr virus (pHEBo,

pREP-derived and p205) can be used for transient expression of nucleic acids in eukaryotic cells. For other suitable expression systems as well as general recombinant procedures, see *Molecular Cloning A Laboratory Manual*, 2nd Ed., ed. by Sambrook, Fritsch and Maniatis (Cold Spring Harbor Laboratory Press: 1989) Chapters 16 and 17; which is incorporated by reference herein in its entirety. In some embodiments, the signaling polypeptide of a multi-component CAL and/or CAR can be constitutively expressed and the recognition polypeptide can be transiently expressed. In some embodiments, the recognition polypeptide of a multi-component CAL and/or CAR can be constitutively expressed and the signaling polypeptide can be transiently expressed. In some embodiments, the recognition polypeptide of a multi-component CAL can be constitutively expressed and the signaling polypeptide can be provided exogenously. In some embodiments, the recognition polypeptide of a multi-component CAL can be transiently expressed and the signaling polypeptide can be provided exogenously. In some embodiments, the signaling polypeptide of a multi-component CAL and/or CAR can be constitutively expressed and the recognition polypeptide can be provided exogenously. In some embodiments, the signaling polypeptide of a multi-component CAL and/or CAR can be transiently expressed and the recognition polypeptide can be provided exogenously.

[00150] The CARs and CALs described herein can be produced according to any method known in the art, e.g., recombinant expression or peptide synthesis. Exemplary methods can include, the NIH Tetramer Core Facility's MHC expression protocols (available on the world wide web at tetramer.yerkes.emory.edu/support/protocols#1); ProImmune's pentamer protocols (available on the world wide web at proimmune.com/protocols-2/); Immudex's dextramer protocols (available on the word wide web at immudex.com/resources/protocols/) and the CAR T cell production protocols provided in the "Primary Human T cells Isolation and Culture" and "Lentiviral Transduction of Human T cells" sub-sections in "Method Details" section of Cho et al., 2018 Cell 173:1426-1438; each of which is incorporated by reference herein in its entirety.

[00151] In one aspect, described herein is a method of killing a target cell, the method comprising contacting the cell with a complete multi-component CAR cell, CAR, and/or CAL according to any of the embodiments described herein. In some embodiments, the target cell can be a diseased cell, e.g., an autoreactive or alloreactive T cell. In one aspect, described herein is a method of treating or preventing a disease, e.g., an autoimmune diseases or conditions; T cell mediated inflammation or immune response; transplant rejection, or GvHD, comprising administering a complete multi-component CAR cell, CAR, and/or CAL according to any of the embodiments described herein. In one aspect, described herein is a method of treating or preventing autoimmune diseases or conditions; T cell mediated inflammation or immune response; transplant rejection; or GvHD, comprising administering a complete multi-component CAR cell, CAR, and/or CAL according to any of the embodiments described herein.

[00152] Another aspect provided herein is a method of preventing and/or treating a malignant T cell condition in a subject in need thereof, the method comprising administering to the subject any of the compositions and/or cells described herein. In one aspect, described herein is a method of treating or preventing a malignant T cell condition in a subject, comprising administering a complete multi-component CAR/CAL, CAR and/or CAL as according to any of the embodiments described herein. In one aspect, described herein is a method of treating or preventing a malignant T cell condition in a subject, comprising administering a complete multi-component CAR cell, CAR, and/or CAL according to any of the embodiments described herein

[00153] In some embodiments, the complete multi-component CAL and/or CAR cell can be autologous or allogeneic to the subject. In some embodiments, the complete multi-component CAL and/or CAR cell can be derived and/or descended from a cell obtained from the subject or a third party and has been modified *ex vivo* to comprise the at least one multi-component CAL and/or CAR, e.g., genetically engineered to comprise nucleic acid sequences encoding both a signaling polypeptide and at least one recognition polypeptide of a multi-component CAL and/or CAR. In some embodiments, the method can further comprise the steps of obtaining a cell from a subject (e.g. a dendritic cell, regulatory T cell, or effector T cell), altering the cell to comprise nucleic acid sequences encoding both a signaling polypeptide and at least one recognition polypeptide of a multi-component CAL and/or CAR, and then administering the cell to the subject.

[00154] In one embodiment, the engineered cell is further modified to lack or have reduced expression of the native MHCII, e.g., as measured on the cell surface. Methods for engineering a cell to reduce or eliminate the native MHCII are known in the art and include, e.g., expression of RNA interference (such as short hairpin RNA, small interfering RNA, double stranded RNA, etc.), expression of an inhibitory oligonucleotide, nuclease-based inhibition (such as CRISPR, TALEN, Meganuclease, etc.), and expression of a KDEL-motif (SEQ ID NO: 2749) containing binding protein capable of restricting MHCII to the endoplasmic reticulum. One skilled in the art can assess whether knockdown of native MHCII is achieved by assessing mRNA or protein levels of MHCII via, e.g., PCR-based assays or western blotting, respectively.

[00155] In one embodiment, the cell is further engineered to knockout the native MHCII. Methods for engineering a cell to knockout the native MHCII are known in the art and include, e.g., expression of RNA interference (such as short hairpin RNA, small interfering RNA, double stranded RNA, etc.), expression of an inhibitory oligonucleotide, nuclease-based inhibition (such as CRISPR, TALEN, Meganuclease, etc.). One skilled in the art can assess whether knockout of native MHCII is achieved by assessing mRNA or protein levels of MHCII via, e.g., PCR-based assays or western blotting, respectively.

[00156] In one aspect, described herein is an engineered cell expressing and/or comprising one or more of the compositions according to any of the embodiments described herein. In one aspect,

described herein is an engineered cell expressing and/or comprising one or more multi-component CAL and/or CAR signaling polypeptides according to any of the embodiments described herein. In some embodiments, the cell is a dendritic cell, regulatory T cell, or effector T cell. In some embodiments, the cell is a T cell. Such cells expressing and/or comprising a multi-component CAL and/or CAR signaling polypeptide are referred to herein as “partial multi-component CAL” cells or “partial multi-component CAR” cells. In some embodiments, the partial multi-component CAL and/or CAR cell does not express, e.g., does not comprise a nucleic acid sequence encoding, a multi-component CAL and/or CAR recognition polypeptide. In some embodiments, a partial multi-component CAL and/or CAR cell comprises a nucleic acid sequence encoding at least one multi-component CAL and/or CAR signaling polypeptide. In some embodiments, the multi-component CAL and/or CAR signaling polypeptide is present on the membrane of the cell, e.g., is expressed as a transmembrane protein at detectable levels. In some embodiments, the signaling polypeptide further comprises a secondary protein interaction domain that specifically binds with the protein interaction domain of the second recognition polypeptide, e.g., the signaling polypeptide is part of an AND gate multi-component CAL and/or CAR as described elsewhere herein. In some embodiments, the cell can further comprise a second multi-component CAL and/or CAR signaling polypeptide, e.g., a signaling polypeptide that is part of a second multi-component CAL and/or CAR according to any of the embodiments described herein.

[00157] In one aspect, described herein is a method of killing a target cell, the method comprising contacting the target cell with a partial multi-component CAL and/or CAR cell according to any of the embodiments described herein and contacting the target cell with at least one recognition polypeptide of the multi-component CAL and/or CAR. In some embodiments, the target cell can be a diseased cell, e.g., an autoreactive or alloreactive T cell. In some embodiments, the target cell can be a diseased cell, e.g., a cancer cell (e.g., a T cell- or T cell precursor cell- derived neoplasm, hereafter referred to as “T cell neoplasms”). In some embodiments, the method can further comprise the steps of obtaining a cell from a subject (e.g. a NK cell, a dendritic cell, regulatory T cell, or effector T cell), altering the cell to comprise a nucleic acid sequence encoding a signaling polypeptide of a multi-component CAL and/or CAR, and then administering the cell to the subject.

[00158] In some embodiments of any of the methods described herein, a pair of protein interaction domains of a multi-component CAL and/or CAR can comprise chemically induced binding domains and the method can further comprise administering a compound that induces binding of the domains. In some embodiments, when one protein interaction domain is FKBP-binding domain of mTOR (FRB) and a second protein interaction domain is FK506 binding protein (FKBP), the method further comprises administering tacrolimus, a rapalog, or everolimus. In some embodiments, when one protein interaction domain is cyclophilin-Fas fusion protein (CyP-Fas) and a second protein interaction domain is FK506 binding protein (FKBP), the method further comprises administering

FKCsA. In some embodiments, when one protein interaction domain is calcineurin A (CNA) and a second protein interaction domain is FK506 binding protein (FKBP), the method further comprises administering FK506. In some embodiments, when one protein interaction domain is gibberellin insensitive (GIA) and a second protein interaction domain is gibberellin insensitive dwarf1 (GID1), the method further comprises administering gibberellin. In some embodiments, when one protein interaction domain is Snap-tag and a second protein interaction domain is Halo tag, the method further comprises administering HaXS. In some embodiments, when one protein interaction domain is T14-3-3-cdeltaC and a second protein interaction domain is C-Terminal peptides of PMA2 (CT52), the method further comprises administering fusicoccin.

[00159] In some embodiments of any of the aspects described herein, a recognition and/or signaling polypeptide of a multi-component CAL and/or CAR can be engineered. In some embodiments of any of the aspects described herein, a recognition and/or signaling polypeptide of a multi-component CAL and/or CAR can be transgenic. In some embodiments of any of the aspects described herein, a recognition and/or signaling polypeptide of a multi-component CAL and/or CAR can be recombinant. In some embodiments of any of the aspects described herein, a recognition and/or signaling polypeptide of a multi-component CAL and/or CAR can be heterologous to a cell. In some embodiments of any of the aspects described herein, a recognition and/or signaling polypeptide of a multi-component CAL and/or CAR can be heterologous to a T cell. In some embodiments of any of the aspects described herein, a recognition and/or signaling polypeptide of a multi-component CAL and/or CAR can be heterologous to a human T cell. In some embodiments of any of the aspects described herein, a recognition and/or signaling polypeptide of a multi-component CAL and/or CAR can be exogenous to a cell. In some embodiments of any of the aspects described herein, a recognition and/or signaling polypeptide of a multi-component CAL and/or CAR can be exogenous to a T cell. In some embodiments of any of the aspects described herein, a recognition and/or signaling polypeptide of a multi-component CAL and/or CAR can be exogenous to a human T cell.

[00160] It is specifically contemplated herein that each of the individual embodiments described herein can be combined, e.g., in a single cell. By way of non-limiting example, a single cell could comprise a first complete multi-component CAL and/or CAR and a second partial multi-component CAL and/or CAR, wherein each multi-component CAL and/or CAR can be according to any of the embodiments described herein.

[00161] In some embodiments, the methods described herein relate to CAL-immune cell therapies. In some embodiments, the methods described herein relate to CAR-immune cell therapies such as CAR-T therapy. Standard CAR-T and related therapies relate to adoptive cell transfer of immune cells (e.g. T cells) expressing a CAR that binds specifically to a targeted cell type (e.g. disease cells, e.g., autoreactive or alloreactive T cells) to treat a subject, e.g., for an autoimmune

diseases or conditions; T cell mediated inflammation or immune response; transplant rejection; or GvHD.

[00162] In some embodiments, the cells administered as part of the therapy can be autologous to the subject. In some embodiments, the cells administered as part of the therapy are not autologous to the subject. In some embodiments, the cells are engineered and/or genetically modified to express a multi-component CAL and/or CAR, or portion thereof as described herein. Further discussion of CAR-T therapies can be found, e.g., in Maus et al. *Blood* 2014 123:2624-35; Reardon et al. *Neuro-Oncology* 2014 16:1441-1458; Hoyos et al. *Haematologica* 2012 97:1622; Byrd et al. *J Clin Oncol* 2014 32:3039-47; Maher et al. *Cancer Res* 2009 69:4559-4562; and Tamada et al. *Clin Cancer Res* 2012 18:6436-6445; each of which is incorporated by reference herein in its entirety.

[00163] In some embodiments, the technology described herein relates to a syringe or catheter, including an organ-specific catheter (e.g., renal catheter, biliary catheter, cardiac catheter, etc.), comprising a therapeutically effective amount of a composition described herein.

Methods of treatment

[00164] In some embodiments, the methods described herein relate to the treatment or prevention of transplant rejection in a subject having a cell, tissue, or organ transplant with one or more compositions, CALs, CARs, or cells as described herein. In another embodiment, the methods described herein relate to the treatment or prevention of GvHD in a subject having a cell, tissue, or organ transplant with one or more compositions, CALs, CARs, or cells as described herein.

[00165] As used herein, “GvHD” refers to a disease characterized by the active process of donor cells attacking the recipient’s own cells. GvHD can develop soon after a transplant, e.g., within weeks or months (acute GvHD), or can occur much later after the transplant, e.g., at least 3-6 months later (chronic GvHD). Symptoms of acute GvHD include, but are not limited to, skin rash or blisters, abdominal pain or discomfort, diarrhea, jaundice, and edema. Symptoms of chronic GvHD include, but are not limited to, changes to skin or nail texture, hair loss or thinning, muscle pain or weakness, blurred vision, mouth sores, shortness of breath, persistent cough, abdominal pain or discomfort, and diarrhea.

[00166] A subject can be identified as having or be at risk of having GvHD by a skilled clinician. Diagnostic tests useful in identifying a subject having GvHD are known in the art and will vary based on the type of transplant the subject has received. The diagnosis of GvHD is made by, for example, physical examination for the signs and symptoms for GvHD known in the art, serologic testing for dysfunction of the liver, gall bladder, kidney, and hematopoietic cells, histologic analysis of biopsies obtained from affected organs, and radiologic imaging of affected organs. In one embodiment, the method further comprises administering at least a second therapeutic. In one embodiment, the composition, CARs, CALs, or cells described herein are administered in combination with Abatacept

(Orencia®) or Belatacept (Nulojix®). Abatacept and Belatacept, developed by Bristol-Meyers Squibb, are fusion proteins composed of the Fc region of the immunoglobulin IgG1 fused to the extracellular domain of CTLA-4. Abatacept is currently approved by the FDA for treatment of rheumatoid arthritis. Belatacept, which only differs from Abatacept by two amino acids, is an immunosuppressant intended to prevent rejection following a kidney transplant.

[00167] In one embodiment, the transplant is vascularized composite allograft (VCA). In one embodiment, the transplant is any human or non-human cell, tissue, or organ. In another embodiment, the transplant is any type of transplants procedures, e.g., any heart transplant, any lung transplant, any liver transplant, any pancreas transplant, any cornea transplant, any trachea transplant, any kidney transplant, any skin transplant, any pancreatic islet cell transplant, any allograft (e.g., a transplantation of allogeneic tissue), any xenograft (e.g., a transplantation of xenogeneic tissue), or any autograft (e.g., a transplantation of tissue). A skilled practitioner will be able to perform a transplant or identify a subject having had a transplant using standard procedural protocols.

[00168] In some embodiments, the methods described herein relate to the treatment or prevention of an autoimmune diseases or conditions, or hypersensitivity reaction I-IV, or immune reaction against foreign therapeutic proteins/molecules, or T cell mediated inflammation or immune response; with compositions, CARs, CALs, or cells as described herein. Subjects having an autoimmune disease can be identified by a physician using current methods of diagnosing an autoimmune disease. Symptoms and/or complications of an autoimmune disease which characterize these conditions and aid in diagnosis are well known in the art and include but are not limited to, fatigue, achy muscles, swelling and redness, low-grade fever, numbness or tingling of the hands or feet, hair loss, and/or skin rash. Tests that may aid in a diagnosis of, e.g. autoimmune disease include, but are not limited to, blood counts, and an antinuclear antibody test (ANA). A family history of autoimmune disease, or having risk factors for autoimmune disease (e.g. gender, age, ethnicity, and exposure to environmental agents, such as procainamide, hydralazine, mercury, gold, or silver) can also aid in determining if a subject is likely to have autoimmune disease or in making a diagnosis of autoimmune disease.

[00169] As used herein, the term "autoimmune disease", "autoimmune condition", or "autoimmune disease or disorder" herein is a disease or disorder arising from and directed against an individual's own tissues or a co-segregate or manifestation thereof or resulting condition therefrom.

[00170] Auto-immune related diseases and disorders arise from an overactive and/or abnormal immune response of the body against substances (autoantigens) and tissues normally present in the body, otherwise known as self or autologous substance. This dysregulated inflammatory reaction causes an exaggerated response by macrophages, granulocytes, lymphocytes, and/or T-lymphocytes leading to abnormal tissue damage and cell death. Subsequent loss of function is associated with inflammatory tissue damage.

[00171] Autoantigens, as used herein, are endogenous proteins or fragments thereof that are involved in or elicit this pathogenic immune response. Autoantigen can be any substance, or a portion thereof normally found within a mammal that, in an autoimmune disease, becomes the primary (or a primary, or secondary) target of attack by the immune system. The term also includes antigenic substances that induce conditions having the characteristics of an autoimmune disease when administered to mammals. Additionally, the term includes peptidic subclasses consisting essentially of immunodominant epitopes or immunodominant epitope regions of autoantigens. Immunodominant epitopes or regions in induced autoimmune conditions are fragments of an autoantigen that can be used instead of the entire autoantigen to induce the disease. In humans afflicted with an autoimmune disease, immunodominant epitopes or regions are fragments of antigens specific to the tissue or organ under autoimmune attack and recognized by a substantial percentage (e.g. a majority though not necessarily an absolute majority) of autoimmune attack T-cells.

[00172] Autoantigens that are known to be associated with autoimmune disease include myelin proteins with demyelinating diseases, e.g. multiple sclerosis and experimental autoimmune myelitis; collagens and rheumatoid arthritis; insulin, proinsulin, glutamic acid decarboxylase 65 (GAD65); and islet cell antigen (ICA512; ICA12) for insulin dependent diabetes.

[00173] A common feature in a number of autoimmune related diseases and inflammatory conditions is the involvement of pro-inflammatory CD4⁺ T cells. These T cells are responsible for the release of inflammatory, Th1 type cytokines. Cytokines characterized as Th1 type include interleukin 2 (IL-2), γ -interferon, TNF α and IL-12. In some embodiments, cytokines characterized as Th1 type include interleukin 2 (IL-2), interferon γ , and TNF α . Such pro-inflammatory cytokines act to stimulate the immune response, in many cases resulting in the destruction of autologous tissue. Cytokines associated with suppression of T cell response are the Th2 type, and include IL-10, IL-4 and TGF- β . It has been found that Th1 and Th2 type T cells may use the identical antigen receptor in response to an immunogen; in the former producing a stimulatory response and, in the latter, a suppressive response.

[00174] T cell mediated inflammation, or a T cell mediated immune response is inflammation and/or an immune response in which T cells and/or T cell activity contributes to or originates the inflammation/immune response.

[00175] As used herein, "inflammation" refers to the complex biological response to harmful stimuli, such as pathogens, damaged cells, or irritants. Inflammation is a protective attempt by the organism to remove the injurious stimuli as well as initiate the healing process for the tissue. Accordingly, the term "inflammation" includes any cellular process that leads to the production of pro-inflammatory cytokines, inflammation mediators and/or the related downstream cellular events resulting from the actions of the cytokines thus produced, for example, fever, fluid accumulation, swelling, abscess formation, and cell death. Inflammation can include both acute responses (i.e.,

responses in which the inflammatory processes are active) and chronic responses (i.e., responses marked by slow progression and formation of new connective tissue). Acute and chronic inflammation may be distinguished by the cell types involved. Acute inflammation often involves polymorphonuclear neutrophils; whereas chronic inflammation is normally characterized by a lymphohistiocytic and/or granulomatous response. An inflammatory condition is any disease state characterized by inflammatory tissues (for example, infiltrates of leukocytes such as lymphocytes, neutrophils, macrophages, eosinophils, mast cells, basophils and dendritic cells) or inflammatory processes which provoke or contribute to the abnormal clinical and histological characteristics of the disease state.

[00176] As used herein, an "immune response" refers to a response by a cell of the immune system, such as a B cell, T cell (CD4 or CD8), regulatory T cell, antigen-presenting cell, dendritic cell, monocyte, macrophage, NKT cell, NK cell, basophil, eosinophil, or neutrophil, to a stimulus (e.g., to an a disease, an antigen, or healthy cells, e.g., in the case of autoimmunity). In some embodiments of the aspects described herein, an immune response is a T cell response, such as a CD4+ response or a CD8+ response. Such responses by these cells can include, for example, cytotoxicity, proliferation, cytokine or chemokine production, trafficking, or phagocytosis, and can be dependent on the nature of the immune cell undergoing the response. Stimulation of an immune response refers to an induction or increase of the immune response. Suppression of an immune response refers to an elimination or decrease of the immune response.

[00177] A "cell-mediated immune response" is one mediated by T-cells and/or other white blood cells. A "cell-mediated immune response" is elicited by the presentation of antigenic epitopes in association with Class I or Class II molecules of the major histocompatibility complex (MHC), CD1 or other non-classical MHC-like molecules. This activates antigen-specific CD4+ T helper cells or CD8+ cytotoxic lymphocyte cells ("CTLs"). CTLs have specificity for peptide antigens that are presented in association with proteins encoded by classical or non-classical MHCs and expressed on the surfaces of cells. CTLs help induce and promote the intracellular destruction of intracellular microbes, or the lysis of cells infected with such microbes. Another aspect of cellular immunity involves an antigen-specific response by helper T-cells. Helper T-cells act to help stimulate the function, and focus the activity of, nonspecific effector cells against cells displaying peptide or other antigens in association with classical or non-classical MHC molecules on their surface. A "cell-mediated immune response" also refers to the production of cytokines, chemokines and other such molecules produced by activated T-cells and/or other white blood cells, including those derived from CD4+ and CD8+ T-cells. The stimulation of a cell-mediated immunological response may be determined by a number of assays, such as by lymphoproliferation (lymphocyte activation) assays, CTL cytotoxic cell assays, by assaying for T-lymphocytes specific for the antigen in a sensitized subject, or by measurement of cytokine production by T cells in response to re-stimulation with

antigen. Such assays are well known in the art. See, e.g., Erickson et al. (1993) *J. Immunol.* 151:4189-4199; and Doe et al. (1994) *Eur. J. Immunol.* 24:2369-2376.

[00178]

[00179] In some embodiments, the T cell mediated immune response is a response to a drug administered to the subject. It is contemplated herein that the present technology can be utilized for depletion of anti-drug specific T cells to prevent immune responses against administered biologics, cell therapies, and/or gene therapies. For example, the methods and compositions described herein can be used to prevent or treat anti-AAV and anti-transgene immune responses for administered adeno-associated virus (AAV) gene therapies, preventing or treating immune responses to genome editing agents such as CRISPR/Cas9, Transcription activator-like effector nucleases (TALENs), or Zinc Finger Nucleases (ZFNs), and preventing or treating immune responses to enzyme replacement therapies such as recombinant human acid α -glucosidase (Pompe disease), α -L-iduronidase (Mucopolysaccharidosis I), and α -galactosidase (Fabry disease).

[00180] In one embodiment of any one of the methods described, the autoimmune disorder is selected from the group consisting of thyroiditis, type 1 diabetes mellitus, Hashimoto's thyroiditis, Graves' disease, celiac disease, multiple sclerosis, Guillain-Barre syndrome, Addison's disease, and Raynaud's phenomenon, Goodpasture's disease, arthritis (rheumatoid arthritis such as acute arthritis, chronic rheumatoid arthritis, gout or gouty arthritis, acute gouty arthritis, acute immunological arthritis, chronic inflammatory arthritis, degenerative arthritis, type II collagen-induced arthritis, infectious arthritis, Lyme arthritis (e.g., post treatment Lyme disease syndrome), proliferative arthritis, psoriatic arthritis, Still's disease, vertebral arthritis, and juvenile-onset rheumatoid arthritis, arthritis chronica progrediente, arthritis deformans, polyarthritis chronica primaria, reactive arthritis, and ankylosing spondylitis), palindromic arthritis, inflammatory hyperproliferative skin diseases, psoriasis such as plaque psoriasis, guttate psoriasis, pustular psoriasis, and psoriasis of the nails, atopy including atopic diseases such as hay fever and Job's syndrome, dermatitis including contact dermatitis, chronic contact dermatitis, exfoliative dermatitis, allergic dermatitis, allergic contact dermatitis, dermatitis herpetiformis, nummular dermatitis, seborrheic dermatitis, non-specific dermatitis, primary irritant contact dermatitis, and atopic dermatitis, x-linked hyper IgM syndrome, allergic intraocular inflammatory diseases, urticaria such as chronic allergic urticaria and chronic idiopathic urticaria, including chronic autoimmune urticaria, myositis, polymyositis/dermatomyositis, juvenile dermatomyositis, toxic epidermal necrolysis, scleroderma (including systemic scleroderma), sclerosis such as systemic sclerosis, multiple sclerosis (MS) such as spino-optical MS, primary progressive MS (PPMS), and relapsing remitting MS (RRMS), progressive systemic sclerosis, atherosclerosis, arteriosclerosis, sclerosis disseminata, ataxic sclerosis, neuromyelitis optica (NMO), inflammatory bowel disease (IBD) (for example, Crohn's disease, autoimmune-mediated gastrointestinal diseases, colitis such as ulcerative colitis, colitis ulcerosa, microscopic colitis,

collagenous colitis, colitis polyposa, necrotizing enterocolitis, and transmural colitis, and autoimmune inflammatory bowel disease), bowel inflammation, pyoderma gangrenosum, erythema nodosum, primary sclerosing cholangitis, respiratory distress syndrome, including adult or acute respiratory distress syndrome (ARDS), meningitis, inflammation of all or part of the uvea, iritis, choroiditis, an autoimmune hematological disorder, rheumatoid spondylitis, rheumatoid synovitis, hereditary angioedema, cranial nerve damage as in meningitis, herpes gestationis, pemphigoid gestationis, pruritis scroti, autoimmune premature ovarian failure, sudden hearing loss due to an autoimmune condition, IgE-mediated diseases such as anaphylaxis and allergic and atopic rhinitis, encephalitis such as Rasmussen's encephalitis and limbic and/or brainstem encephalitis, uveitis, such as anterior uveitis, acute anterior uveitis, granulomatous uveitis, nongranulomatous uveitis, phacoantigenic uveitis, posterior uveitis, or autoimmune uveitis, glomerulonephritis (GN) with and without nephrotic syndrome such as chronic or acute glomerulonephritis such as primary GN, immune-mediated GN, membranous GN (membranous nephropathy), idiopathic membranous GN or idiopathic membranous nephropathy, membrano- or membranous proliferative GN (MPGN), including Type I and Type II, and rapidly progressive GN, proliferative nephritis, autoimmune polyglandular endocrine failure, balanitis including balanitis circumscripta plasmacellularis, balanoposthitis, erythema annulare centrifugum, erythema dyschromicum perstans, erythema multiform, granuloma annulare, lichen nitidus, lichen sclerosus et atrophicus, lichen simplex chronicus, lichen spinulosus, lichen planus, lamellar ichthyosis, epidermolytic hyperkeratosis, premalignant keratosis, pyoderma gangrenosum, allergic conditions and responses, allergic reaction, eczema including allergic or atopic eczema, asteatotic eczema, dyshidrotic eczema, and vesicular palmoplantar eczema, asthma such as asthma bronchiale, bronchial asthma, and auto-immune asthma, conditions involving infiltration of T cells and chronic inflammatory responses, immune reactions against foreign antigens such as fetal A-B-O blood groups during pregnancy, chronic pulmonary inflammatory disease, autoimmune myocarditis, leukocyte adhesion deficiency, lupus, including lupus nephritis, lupus cerebritis, pediatric lupus, non-renal lupus, extra-renal lupus, discoid lupus and discoid lupus erythematosus, alopecia lupus, systemic lupus erythematosus (SLE) such as cutaneous SLE or subacute cutaneous SLE, neonatal lupus syndrome (NLE), and lupus erythematosus disseminatus, juvenile onset (Type I) diabetes mellitus, including pediatric insulin-dependent diabetes mellitus (IDDM), adult onset diabetes mellitus (Type II diabetes), autoimmune diabetes, idiopathic diabetes insipidus, diabetic retinopathy, diabetic nephropathy, diabetic large-artery disorder, immune responses associated with acute and delayed hypersensitivity mediated by cytokines and T-lymphocytes, sarcoidosis, granulomatosis including lymphomatoid granulomatosis, Wegener's granulomatosis, agranulocytosis, vasculitides, including vasculitis, large-vessel vasculitis (including polymyalgia rheumatica and giant-cell (Takayasu's) arteritis), medium-vessel vasculitis (including Kawasaki's disease and polyarteritis nodosa/periarteritis nodosa), microscopic polyarteritis, immunovascularitis, CNS vasculitis, cutaneous vasculitis,

hypersensitivity vasculitis, necrotizing vasculitis such as systemic necrotizing vasculitis, and ANCA-associated vasculitis, such as Churg-Strauss vasculitis or syndrome (CSS) and ANCA-associated small-vessel vasculitis, temporal arteritis, autoimmune aplastic anemia, Coombs positive anemia, Diamond Blackfan anemia, hemolytic anemia or immune hemolytic anemia including autoimmune hemolytic anemia (AIHA), pernicious anemia (anemia perniciosa), Addison's disease, pure red cell anemia or aplasia (PRCA), Factor VIII deficiency, hemophilia A, autoimmune neutropenia, pancytopenia, leukopenia, diseases involving leukocyte diapedesis, CNS inflammatory disorders, multiple organ injury syndrome such as those secondary to septicemia, trauma or hemorrhage, antigen-antibody complex-mediated diseases, anti-glomerular basement membrane disease, anti-phospholipid antibody syndrome, allergic neuritis, Behcet's disease/syndrome, Castleman's syndrome, Goodpasture's syndrome, Reynaud's syndrome, Sjogren's syndrome, Stevens-Johnson syndrome, pemphigoid such as pemphigoid bullous and skin pemphigoid, pemphigus (including pemphigus vulgaris, pemphigus foliaceus, pemphigus mucus-membrane pemphigoid, and pemphigus erythematosus), autoimmune polyendocrinopathies, Reiter's disease or syndrome, an immune complex disorder such as immune complex nephritis, antibody-mediated nephritis, polyneuropathies, chronic neuropathy such as IgM polyneuropathies or IgM-mediated neuropathy, and autoimmune or immune-mediated thrombocytopenia such as idiopathic thrombocytopenic purpura (ITP) including chronic or acute ITP, scleritis such as idiopathic cerato-scleritis, episcleritis, autoimmune disease of the testis and ovary including autoimmune orchitis and oophoritis, primary hypothyroidism, hypoparathyroidism, autoimmune endocrine diseases including thyroiditis such as autoimmune thyroiditis, Hashimoto's disease, chronic thyroiditis (Hashimoto's thyroiditis), or subacute thyroiditis, idiopathic hypothyroidism, Grave's disease, polyglandular syndromes such as autoimmune polyglandular syndromes (or polyglandular endocrinopathy syndromes), paraneoplastic syndromes, including neurologic paraneoplastic syndromes such as Lambert-Eaton myasthenic syndrome or Eaton-Lambert syndrome, stiff-man or stiff-person syndrome, encephalomyelitis such as allergic encephalomyelitis or encephalomyelitis allergica and experimental allergic encephalomyelitis (EAE), myasthenia gravis such as thymoma-associated myasthenia gravis, cerebellar degeneration, neuromyotonia, opsoclonus or opsoclonus myoclonus syndrome (OMS), and sensory neuropathy, multifocal motor neuropathy, Sheehan's syndrome, autoimmune hepatitis, lupoid hepatitis, giant-cell hepatitis, autoimmune chronic active hepatitis, lymphoid interstitial pneumonitis (LIP), bronchiolitis obliterans (non-transplant) vs NSIP, Guillain-Barre syndrome, Berger's disease (IgA nephropathy), idiopathic IgA nephropathy, linear IgA dermatosis, acute febrile neutrophilic dermatosis, subcorneal pustular dermatosis, transient acantholytic dermatosis, cirrhosis such as primary biliary cirrhosis and pneumonocirrhosis, autoimmune enteropathy syndrome, Celiac or Coeliac disease, celiac sprue (gluten enteropathy), refractory sprue, idiopathic sprue, cryoglobulinemia, amyotrophic lateral sclerosis (ALS; Lou Gehrig's disease), coronary artery disease, autoimmune ear disease such as

autoimmune inner ear disease (AIED), autoimmune hearing loss, polychondritis such as refractory or relapsed or relapsing polychondritis, pulmonary alveolar proteinosis, Cogan's syndrome/nonsyphilitic interstitial keratitis, Bell's palsy, Sweet's disease/syndrome, rosacea autoimmune, zoster-associated pain, amyloidosis, a non-cancerous lymphocytosis, a primary lymphocytosis, which includes monoclonal B cell lymphocytosis (e.g., benign monoclonal gammopathy and monoclonal gammopathy of undetermined significance, MGUS), peripheral neuropathy, paraneoplastic syndrome, channelopathies including channelopathies of the CNS, autism, inflammatory myopathy, focal or segmental or focal segmental glomerulosclerosis (FSGS), endocrine ophthalmopathy, uveoretinitis, chorioretinitis, autoimmune hepatological disorder, fibromyalgia, multiple endocrine failure, Schmidt's syndrome, adrenalitis, gastric atrophy, presenile dementia, demyelinating diseases such as autoimmune demyelinating diseases and chronic inflammatory demyelinating polyneuropathy, Dressler's syndrome, alopecia areata, alopecia totalis, CREST syndrome (calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia), male and female autoimmune infertility, e.g., due to anti-spermatozoan antibodies, mixed connective tissue disease, Chagas' disease, rheumatic fever, recurrent abortion, farmer's lung, erythema multiforme, post-cardiotomy syndrome, Cushing's syndrome, bird-fancier's lung, allergic granulomatous angiitis, benign lymphocytic angitis, Alport's syndrome, alveolitis such as allergic alveolitis and fibrosing alveolitis, interstitial lung disease, transfusion reaction, Sampter's syndrome, Caplan's syndrome, endocarditis, endomyocardial fibrosis, diffuse interstitial pulmonary fibrosis, interstitial lung fibrosis, pulmonary fibrosis, idiopathic pulmonary fibrosis, cystic fibrosis, endophthalmitis, erythema elevatum et diutinum, erythroblastosis fetalis, eosinophilic fasciitis, Shulman's syndrome, Felty's syndrome, cyclitis such as chronic cyclitis, heterochronic cyclitis, iridocyclitis (acute or chronic), or Fuch's cyclitis, Henoch-Schonlein purpura, SCID, sepsis, endotoxemia, post-vaccination syndromes, Evan's syndrome, autoimmune gonadal failure, Sydenham's chorea, post-streptococcal nephritis, thromboangitis obliterans, thyrotoxicosis, tabes dorsalis, chorioiditis, giant-cell polymyalgia, chronic hypersensitivity pneumonitis, keratoconjunctivitis sicca, idiopathic nephritic syndrome, minimal change nephropathy, benign familial and ischemia-reperfusion injury, transplant organ reperfusion, retinal autoimmunity, aphthae, aphthous stomatitis, arteriosclerotic disorders, aspermiogenesis, autoimmune hemolysis, Boeck's disease, enteritis allergica, erythema nodosum leprosum, idiopathic facial paralysis, chronic fatigue syndrome, febris rheumatica, Hamman-Rich's disease, sensorineural hearing loss, ileitis regionalis, leucopenia, transverse myelitis, primary idiopathic myxedema, ophthalmia sympathica, polyradiculitis acuta, pyoderma gangrenosum, acquired splenic atrophy, vitiligo, toxic-shock syndrome, conditions involving infiltration of T cells, leukocyte-adhesion deficiency, immune responses associated with acute and delayed hypersensitivity mediated by cytokines and T-lymphocytes, diseases involving leukocyte diapedesis, multiple organ injury syndrome, antigen-antibody complex-mediated diseases, antiglomerular basement membrane disease,

allergic neuritis, autoimmune polyendocrinopathies, oophoritis, primary myxedema, autoimmune atrophic gastritis, rheumatic diseases, mixed connective tissue disease, nephrotic syndrome, insulinitis, polyendocrine failure, autoimmune polyglandular syndrome type I, adult-onset idiopathic hypoparathyroidism (AOIH), myocarditis, nephrotic syndrome, primary sclerosing cholangitis, acute or chronic sinusitis, ethmoid, frontal, maxillary, or sphenoid sinusitis, an eosinophil-related disorder such as eosinophilia, pulmonary infiltration eosinophilia, eosinophilia-myalgia syndrome, Löffler's syndrome, chronic eosinophilic pneumonia, tropical pulmonary eosinophilia, granulomas containing eosinophils, seronegative spondyloarthritides, polyendocrine autoimmune disease, sclerosing cholangitis, sclera, episclera, Bruton's syndrome, transient hypogammaglobulinemia of infancy, Wiskott-Aldrich syndrome, ataxia telangiectasia syndrome, angiectasis, autoimmune disorders associated with collagen disease, rheumatism, allergic hypersensitivity disorders, glomerulonephritides, reperfusion injury, ischemic re-perfusion disorder, lymphomatous tracheobronchitis, inflammatory dermatoses, dermatoses with acute inflammatory components, and autoimmune uveoretinitis (AUR). In some embodiments, the autoimmune disease or condition or T cell mediated inflammation can be neurodegeneration, e.g., Alzheimer's or Parkinson disease.

[00181] In some embodiments of any of the aspects, the autoimmune disease or condition, or T cell mediated inflammation can be type 1 diabetes, rheumatoid arthritis, multiple sclerosis, pemphigus, alopecia, lupus, vitiligo, or chronic fatigue syndrome.

[00182] As used herein, "malignant T cell condition" refers to a condition in which T cells display one or more of uncontrolled growth (i.e., division beyond normal limits), invasion (i.e., intrusion on and destruction of adjacent tissues), and metastasis (i.e., spread to other locations in the body via lymph or blood). Non-limiting examples of malignant T cell conditions include T cell cancers, lymphoma, leukemia, T cell acute lymphoblastic leukemia, and T cell lymphoblastic lymphoma.

[00183] The compositions and methods described herein can be administered to a subject to treat or prevent an autoimmune diseases or conditions; T cell mediated inflammation or immune response; or transplant rejection. In some embodiments, the methods described herein comprise administering an effective amount of compositions, CALs, CARs, or cells described herein to a subject in order to alleviate a symptom of an autoimmune diseases or conditions; T cell mediated inflammation or immune response; or transplant rejection. As used herein, "alleviating a symptom" is ameliorating any condition or symptom associated with the disease or condition. As compared with an equivalent untreated control, such reduction is by at least 5%, 10%, 20%, 40%, 50%, 60%, 80%, 90%, 95%, 99% or more as measured by any standard technique. A variety of means for administering the compositions described herein to subjects are known to those of skill in the art. Such methods can include, but are not limited to oral, parenteral, intravenous, intramuscular, subcutaneous, transdermal, airway (aerosol), pulmonary, cutaneous, topical, injection, or intratumoral administration. Administration can be local or systemic.

[00184] The administration of the compositions contemplated herein may be carried out in any convenient manner, including by aerosol inhalation, injection, ingestion, transfusion, implantation or transplantation. In a preferred embodiment, compositions are administered parenterally. The phrases “parenteral administration” and “administered parenterally” as used herein refers to modes of administration other than enteral and topical administration, usually by injection, and includes, without limitation, intravascular, intravenous, intramuscular, intraarterial, intrathecal, intracapsular, intraorbital, intratumoral, intracardiac, intradermal, intraperitoneal, transtracheal, subcutaneous, subcuticular, intraarticular, subcapsular, subarachnoid, intraspinal and intrasternal injection and infusion. In one embodiment, the compositions contemplated herein are administered to a subject by direct injection into a tumor, lymph node, or site of infection.

[00185] It can generally be stated that a pharmaceutical composition comprising the cells, e.g., T cells or CAL cells or CAR cells, described herein may be administered at a dosage of 10^2 to 10^{10} cells/kg body weight, preferably 10^5 to 10^6 cells/kg body weight, including all integer values within those ranges. The number of cells will depend upon the ultimate use for which the composition is intended as will the type of cells included therein. For uses provided herein, the cells are generally in a volume of a liter or less, can be 500 mLs or less, even 250 mLs or 100 mLs or less. Hence the density of the desired cells is typically greater than 10^6 cells/ml and generally is greater than 10^7 cells/ml, generally 10^8 cells/ml or greater. The clinically relevant number of immune cells can be apportioned into multiple infusions that cumulatively equal or exceed 10^5 , 10^6 , 10^7 , 10^8 , 10^9 , 10^{10} , 10^{11} , or 10^{12} cells. In some aspects of the present invention, particularly since all the infused cells will be redirected to a particular or specific target antigen, lower numbers of cells, in the range of 10^6 /kilogram (10^6 - 10^{11} per patient) may be administered. CAL and/or CAR expressing cell compositions may be administered multiple times at dosages within these ranges. The cells may be allogeneic, syngeneic, xenogeneic, or autologous to the patient undergoing therapy. If desired, the treatment may also include administration of mitogens (e.g., PHA) or lymphokines, cytokines, and/or chemokines (e.g., IFN- γ , IL-2, IL-12, TNF-alpha, IL-18, and TNF-beta, GM-CSF, IL-4, IL-13, Flt3-L, RANTES, MIP1 α , etc.) as described herein to enhance induction of the immune response.

[00186] In some embodiments, the dosage can be from about 1×10^5 cells to about 1×10^8 cells per kg of body weight. In some embodiments, the dosage can be from about 1×10^6 cells to about 1×10^7 cells per kg of body weight. In some embodiments, the dosage can be about 1×10^6 cells per kg of body weight. In some embodiments, one dose of cells can be administered. In some embodiments, the dose of cells can be repeated, e.g., once, twice, or more. In some embodiments, the dose of cells can be administered on, e.g., a daily, weekly, or monthly basis.

[00187] The dosage ranges for the agent, e.g., a CAL, CAR, cell, or composition described herein depend upon the potency, and encompass amounts large enough to produce the desired effect e.g., prevention of transplant rejection, reduction in inflammation, etc. The dosage should not be so large

as to cause unacceptable adverse side effects. Generally, the dosage will vary with the age, condition, and sex of the patient and can be determined by one of skill in the art. The dosage can also be adjusted by the individual physician in the event of any complication. In some embodiments, the dosage ranges from 0.001 mg/kg body weight to 0.5 mg/kg body weight. In some embodiments, the dose range is from 5 µg/kg body weight to 100 µg/kg body weight. Alternatively, the dose range can be titrated to maintain serum levels between 1 µg/mL and 1000 µg/mL. For systemic administration, subjects can be administered a therapeutic amount, such as, e.g., 0.1 mg/kg, 0.5 mg/kg, 1.0 mg/kg, 2.0 mg/kg, 2.5 mg/kg, 5 mg/kg, 10 mg/kg, 15 mg/kg, 20 mg/kg, 25 mg/kg, 30 mg/kg, 40 mg/kg, 50 mg/kg, or more.

[00188] Administration of the doses recited above can be repeated. In some embodiments, the doses are given once a day, or multiple times a day, for example but not limited to three times a day. In some embodiments, the doses recited above are administered daily for several weeks or months. The duration of treatment depends upon the subject's clinical progress and responsiveness to therapy.

[00189] In some embodiments, the dose can be from about 2 mg/kg to about 15 mg/kg. In some embodiments, the dose can be about 2 mg/kg. In some embodiments, the dose can be about 4 mg/kg. In some embodiments, the dose can be about 5 mg/kg. In some embodiments, the dose can be about 6 mg/kg. In some embodiments, the dose can be about 8 mg/kg. In some embodiments, the dose can be about 10 mg/kg. In some embodiments, the dose can be about 15 mg/kg. In some embodiments, the dose can be from about 100 mg/m² to about 700 mg/m². In some embodiments, the dose can be about 250 mg/m². In some embodiments, the dose can be about 375 mg/m². In some embodiments, the dose can be about 400 mg/m². In some embodiments, the dose can be about 500 mg/m².

[00190] In some embodiments, the dose can be administered intravenously. In some embodiments, the intravenous administration can be an infusion occurring over a period of from about 10 minutes to about 3 hours. In some embodiments, the intravenous administration can be an infusion occurring over a period of from about 30 minutes to about 90 minutes.

[00191] In some embodiments the dose can be administered about weekly. In some embodiments, the dose can be administered weekly. In some embodiments, the dose can be administered weekly for from about 12 weeks to about 18 weeks. In some embodiments the dose can be administered about every 2 weeks. In some embodiments the dose can be administered about every 3 weeks. In some embodiments, the dose can be from about 2 mg/kg to about 15 mg/kg administered about every 2 weeks. In some embodiments, the dose can be from about 2 mg/kg to about 15 mg/kg administered about every 3 weeks. In some embodiments, the dose can be from about 2 mg/kg to about 15 mg/kg administered intravenously about every 2 weeks. In some embodiments, the dose can be from about 2 mg/kg to about 15 mg/kg administered intravenously about every 3 weeks. In some embodiments, the dose can be from about 200 mg/m² to about 400 mg/m² administered intravenously about every week. In some embodiments, the dose can be from about 200 mg/m² to about 400 mg/m² administered intravenously about every 2 weeks. In some embodiments, the dose can be from about

200 mg/m² to about 400 mg/m² administered intravenously about every 3 weeks. In some embodiments, a total of from about 2 to about 10 doses are administered. In some embodiments, a total of 4 doses are administered. In some embodiments, a total of 5 doses are administered. In some embodiments, a total of 6 doses are administered. In some embodiments, a total of 7 doses are administered. In some embodiments, a total of 8 doses are administered. In some embodiments, the administration occurs for a total of from about 4 weeks to about 12 weeks. In some embodiments, the administration occurs for a total of about 6 weeks. In some embodiments, the administration occurs for a total of about 8 weeks. In some embodiments, the administration occurs for a total of about 12 weeks. In some embodiments, the initial dose can be from about 1.5 to about 2.5 fold greater than subsequent doses.

[00192] In some embodiments, the dose can be from about 1 mg to about 2000 mg. In some embodiments, the dose can be about 3 mg. In some embodiments, the dose can be about 10 mg. In some embodiments, the dose can be about 30 mg. In some embodiments, the dose can be about 1000 mg. In some embodiments, the dose can be about 2000 mg. In some embodiments, the dose can be about 3 mg given by intravenous infusion daily. In some embodiments, the dose can be about 10 mg given by intravenous infusion daily. In some embodiments, the dose can be about 30 mg given by intravenous infusion three times per week.

[00193] A therapeutically effective amount is an amount of an agent that is sufficient to produce a statistically significant, measurable change in, or prevent the occurrence of an autoimmune disease or condition; T cell mediated inflammation or immune response; transplant rejection; or GvHD. Such effective amounts can be gauged in clinical trials as well as animal studies.

[00194] An agent can be administered intravenously by injection or by gradual infusion over time. Given an appropriate formulation for a given route, for example, agents useful in the methods and compositions described herein can be administered intravenously, intranasally, by inhalation, intraperitoneally, intramuscularly, subcutaneously, intracavity, and can be delivered by peristaltic means, if desired, or by other means known by those skilled in the art. It is preferred that the compounds used herein are administered orally, intravenously or intramuscularly. Local administration, e.g., directly to the site of an organ or tissue transplant is also specifically contemplated.

[00195] Therapeutic compositions containing at least one agent can be conventionally administered in a unit dose, for example. The term “unit dose” when used in reference to a therapeutic composition refers to physically discrete units suitable as unitary dosage for the subject, each unit containing a predetermined quantity of active material calculated to produce the desired therapeutic effect in association with the required physiologically acceptable diluent, i.e., carrier, or vehicle.

[00196] The compositions are administered in a manner compatible with the dosage formulation, and in a therapeutically effective amount. The quantity to be administered and timing depends on the

subject to be treated, capacity of the subject's system to utilize the active ingredient, and degree of therapeutic effect desired.

[00197] In embodiments where the subject is administered a partial multi-component CAL and/or CAR cell and a recognition polypeptide, the partial multi-component CAL and/or CAR cell and a recognition polypeptide can be administered together or separately. In embodiments where the subject is separately administered a partial multi-component CAL and/or CAR cell and a recognition polypeptide each of the compositions can be administered, separately, according to any of the dosages and administration routes/routines described herein.

[00198] Precise amounts of active ingredient required to be administered depend on the judgment of the practitioner and are particular to each individual. However, suitable dosage ranges for systemic application are disclosed herein and depend on the route of administration. Suitable regimes for administration are also variable but are typified by an initial administration followed by repeated doses at one or more hour intervals by a subsequent injection or other administration. Alternatively, continuous intravenous infusion sufficient to maintain concentrations in the blood in the ranges specified for in vivo therapies are contemplated.

[00199] In some embodiments, the methods further comprise administering a composition, CAL, or CAR, or cell described herein along with one or more additional autoimmune, GvHD, or transplant rejection agents, biologics, drugs, or treatments as part of a combinatorial therapy. Exemplary treatments for transplant rejection or GvHD include but are not limited to, Immunosuppressive drugs, e.g., Cyclosporine (Neoral, Sandimmune, Gengraf, and Restasis), Tacrolimus (Prograf, Protopic, Astagraf XL, and Envarsus XR), Methotrexate (Trexall, Rasuvo, Rheumatrex, and Otrexup (PF)), Sirolimus (Rapamune), Mycophenolic acid (Myfortic and CellCept), Rituximab (Rituxan), etanercept (Enbrel), pentostatin (Nipent), ruxolitinib (Jakafi); Chemotherapies, e.g., Methotrexate (Trexall, Rasuvo, Rheumatrex, and Otrexup (PF)), antithymocyte globulin (Atgam, Thymoglobulin); Steroids, e.g., Prednisone (Deltasone, Rayos, and Prednisone Intensol), Methylprednisolone (Medrol, Solu-Medrol, and Depo-Medrol), budesonide (Entocort EC, Uceris); Antifungal, e.g., Posaconazole (Noxafil); Antiviral drugs, e.g., Acyclovir (Zovirax and Sitavig), Valacyclovir (Valtrex); and Antibiotics, e.g., Sulfamethoxazole / Trimethoprim (Bactrim, Sulfatrim, and Bactrim DS); Protease inhibitors, e.g. alpha1-proteinase inhibitor (Zemaira); extracorporeal photopheresis; monoclonal antibodies (daclizumab (Zinbryta), basiliximab (Simulect)), Brentuximab vedotin (Adcetris), Alemtuzumab (Campath, Lemtrada), Tocilizumab (Actemra); infusion of mesenchymal stromal cells.

[00200] Exemplary treatments for autoimmune disease include but are not limited to, Insulin, e.g., Insulin glulisine (Apidra and Apidra SoloStar), Insulin detemir (Levemir and Levemir FlexTouch), Insulin aspart (NovoLog, Novolog Flexpen, and Novolog PenFill), Insulin lispro (Humalog and Humalog KwikPen), Insulin, Insulin glargine (Lantus, Lantus Solostar, and Toujeo SoloStar); Dietary supplement, e.g., glucose tablets; and Hormones, e.g., Glucagon (GlucaGen and Glucagon Emergency

Kit (human)), antidiabetic agents (Metformin (D-Care DM2, Fortamet, Glucophage, Glucophage XR, Glumetza, Riomet), glucagon-like peptide-1 (GLP-1) receptor agonist (liraglutide (Saxenda; Victoza) or semaglutide (Ozempic) or sodium-glucose co-transporter 2 (SGLT2) inhibitor: empagliflozin (Jardiance), canagliflozin (Invokana); sulfonylureas: glipizide (GlipiZIDE XL, Glucotrol, Glucotrol XL); Meglitinide Analogs: repaglinide (Prandin); Thiazolidinedione: pioglitazone (Actos); dipeptidyl peptidase-4 (DPP-4) inhibitors: Sitagliptin (Januvia), Saxagliptin (Onglyza), Linagliptin (Tradjenta), Alogliptin (Nesina)

[00201] The efficacy of a given treatment, e.g., for an autoimmune diseases or conditions; T cell mediated inflammation or immune response; transplant rejection; or GvHD, can be determined by the skilled clinician. However, a treatment is considered “effective treatment,” as the term is used herein, if any one or all of the signs or symptoms of are altered in a beneficial manner or other clinically accepted symptoms are improved, or even ameliorated, e.g., by at least 10% following treatment with an agent as described herein. Efficacy can also be measured by a failure of an individual to worsen as assessed by hospitalization or need for medical interventions (i.e., progression of the disease is halted). Methods of measuring these indicators are known to those of skill in the art and/or described herein.

[00202] An effective amount for the treatment of a disease means that amount which, when administered to a mammal in need thereof, is sufficient to result in effective treatment as that term is defined herein, for that disease. Efficacy of an agent can be determined by assessing physical indicators of, for example autoimmune disease (e.g., result of an ANA), T cell mediated inflammation or immune response, malignant T cell condition, transplant rejection (e.g., high fever, tenderness at transplant site, etc.), or GvHD (e.g., redness, pain, or other symptoms at transplant site). Effective amounts, toxicity, and therapeutic efficacy can be determined by standard pharmaceutical procedures in cell cultures or experimental animals, e.g., for determining the LD50 (the dose lethal to 50% of the population) and the ED50 (the dose therapeutically effective in 50% of the population). The dosage can vary depending upon the dosage form employed and the route of administration utilized. The dose ratio between toxic and therapeutic effects is the therapeutic index and can be expressed as the ratio LD50/ED50. Compositions and methods that exhibit large therapeutic indices are preferred. A therapeutically effective dose can be estimated initially from cell culture assays. Also, a dose can be formulated in animal models to achieve a circulating plasma concentration range that includes the IC50 (i.e., the concentration of the active ingredient(s), which achieves a half-maximal inhibition of symptoms) as determined in cell culture, or in an appropriate animal model. Levels in plasma can be measured, for example, by immunoassay, various DNA detection technologies, or high performance liquid chromatography. The effects of any particular dosage can be monitored by a suitable bioassay, e.g., assay to assess reaction following transplant, level of inflammation, ANA measurement, among

others. The dosage can be determined by a physician and adjusted, as necessary, to suit observed effects of the treatment.

[00203] Efficacy can also be measured by a failure of an individual to worsen as assessed by hospitalization, or need for medical interventions (i.e., progression of the disease is halted). Methods of measuring these indicators are known to those of skill in the art and/or are described herein. Treatment includes any treatment of a disease in an individual or an animal (some non-limiting examples include a human or an animal) and includes: (1) inhibiting the disease, e.g., preventing a worsening of symptoms (e.g. pain or inflammation); or (2) relieving the severity of the disease, e.g., causing regression of symptoms. An effective amount for the treatment of a disease means that amount which, when administered to a subject in need thereof, is sufficient to result in effective treatment as that term is defined herein, for that disease. Efficacy of an agent can be determined by assessing physical indicators of a condition or desired response, (e.g. a reduction of inflammation, etc.). It is well within the ability of one skilled in the art to monitor efficacy of administration and/or treatment by measuring any one of such parameters, or any combination of parameters. Efficacy can be assessed in animal models of a condition described herein, for example treatment of autoimmune disease, transplant rejection or GVHD. When using an experimental animal model, efficacy of treatment is evidenced when a statistically significant change in a marker is observed, e.g. inflammation.

[00204] In some embodiments, the technology described herein relates to a pharmaceutical composition comprising a CAL and/or CAR, or a multi-component CAL and/or CAR (or portion thereof, or cell comprising a CAL and/or CAR, or a multi-component CAL and/or CAR) as described herein, and optionally a pharmaceutically acceptable carrier. In some embodiments, the active ingredients of the pharmaceutical composition comprise a CAL and/or CAR or a multi-component CAL and/or CAR (or portion thereof, or cell comprising a CAL and/or CAR or a multi-component CAL and/or CAR) as described herein. In some embodiments, the active ingredients of the pharmaceutical composition consist essentially of a CAL and/or CAR or a multi-component CAL and/or CAR (or portion thereof, or cell comprising a CAL and/or CAR or a multi-component CAL and/or CAR) as described herein. In some embodiments, the active ingredients of the pharmaceutical composition consist of a CAL and/or CAR or a multi-component CAL and/or CAR (or portion thereof, or cell comprising a multi-component CAL and/or CAR) as described herein.

[00205] Pharmaceutically acceptable carriers and diluents include saline, aqueous buffer solutions, solvents and/or dispersion media. The use of such carriers and diluents is well known in the art. Some non-limiting examples of materials which can serve as pharmaceutically-acceptable carriers include: (1) sugars, such as lactose, glucose and sucrose; (2) starches, such as corn starch and potato starch; (3) cellulose, and its derivatives, such as sodium carboxymethyl cellulose, methylcellulose, ethyl cellulose, microcrystalline cellulose and cellulose acetate; (4) powdered tragacanth; (5) malt; (6)

gelatin; (7) lubricating agents, such as magnesium stearate, sodium lauryl sulfate and talc; (8) excipients, such as cocoa butter and suppository waxes; (9) oils, such as peanut oil, cottonseed oil, safflower oil, sesame oil, olive oil, corn oil and soybean oil; (10) glycols, such as propylene glycol; (11) polyols, such as glycerin, sorbitol, mannitol and polyethylene glycol (PEG); (12) esters, such as ethyl oleate and ethyl laurate; (13) agar; (14) buffering agents, such as magnesium hydroxide and aluminum hydroxide; (15) alginic acid; (16) pyrogen-free water; (17) isotonic saline; (18) Ringer's solution; (19) ethyl alcohol; (20) pH buffered solutions; (21) polyesters, polycarbonates and/or polyanhydrides; (22) bulking agents, such as polypeptides and amino acids (23) serum component, such as serum albumin, HDL and LDL; (22) C2-C12 alcohols, such as ethanol; and (23) other non-toxic compatible substances employed in pharmaceutical formulations. Wetting agents, coloring agents, release agents, coating agents, sweetening agents, flavoring agents, perfuming agents, preservative and antioxidants can also be present in the formulation. The terms such as "excipient", "carrier", "pharmaceutically acceptable carrier" or the like are used interchangeably herein. In some embodiments, the carrier inhibits the degradation of the active agent as described herein.

[00206] In some embodiments, the pharmaceutical composition comprising a multi-component CAL and/or CAR (or portion thereof, or cell comprising a multi-component CAL and/or CAR) as described herein can be a parenteral dose form. Since administration of parenteral dosage forms typically bypasses the patient's natural defenses against contaminants, parenteral dosage forms are preferably sterile or capable of being sterilized prior to administration to a patient. Examples of parenteral dosage forms include, but are not limited to, solutions ready for injection, dry products ready to be dissolved or suspended in a pharmaceutically acceptable vehicle for injection, suspensions ready for injection, and emulsions. In addition, controlled-release parenteral dosage forms can be prepared for administration of a patient, including, but not limited to, DUROS®-type dosage forms and dose-dumping.

[00207] Suitable vehicles that can be used to provide parenteral dosage forms of a multi-component CAL and/or CAR (or portion thereof, or cell comprising a multi-component CAL and/or CAR) as disclosed within are well known to those skilled in the art. Examples include, without limitation: sterile water; water for injection USP; saline solution; glucose solution; aqueous vehicles such as but not limited to, sodium chloride injection, Ringer's injection, dextrose Injection, dextrose and sodium chloride injection, and lactated Ringer's injection; water-miscible vehicles such as, but not limited to, ethyl alcohol, polyethylene glycol, and propylene glycol; and non-aqueous vehicles such as, but not limited to, corn oil, cottonseed oil, peanut oil, sesame oil, ethyl oleate, isopropyl myristate, and benzyl benzoate. Compounds that alter or modify the solubility of a pharmaceutically acceptable salt of an active ingredient can also be incorporated into the parenteral dosage forms of the disclosure, including conventional and controlled-release parenteral dosage forms.

[00208] Pharmaceutical compositions can also be formulated to be suitable for oral administration, for example as discrete dosage forms, such as, but not limited to, tablets (including without limitation scored or coated tablets), pills, caplets, capsules, chewable tablets, powder packets, cachets, troches, wafers, aerosol sprays, or liquids, such as but not limited to, syrups, elixirs, solutions or suspensions in an aqueous liquid, a non-aqueous liquid, an oil-in-water emulsion, or a water-in-oil emulsion. Such compositions contain a predetermined amount of the pharmaceutically acceptable salt of the disclosed compounds, and may be prepared by methods of pharmacy well known to those skilled in the art. See generally, Remington: The Science and Practice of Pharmacy, 21st Ed., Lippincott, Williams, and Wilkins, Philadelphia PA. (2005).

[00209] Conventional dosage forms generally provide rapid or immediate drug release from the formulation. Depending on the pharmacology and pharmacokinetics of the drug, use of conventional dosage forms can lead to wide fluctuations in the concentrations of the drug in a patient's blood and other tissues. These fluctuations can impact a number of parameters, such as dose frequency, onset of action, duration of efficacy, maintenance of therapeutic blood levels, toxicity, side effects, and the like. Advantageously, controlled-release formulations can be used to control a drug's onset of action, duration of action, plasma levels within the therapeutic window, and peak blood levels. In particular, controlled- or extended-release dosage forms or formulations can be used to ensure that the maximum effectiveness of a drug is achieved while minimizing potential adverse effects and safety concerns, which can occur both from under-dosing a drug (i.e., going below the minimum therapeutic levels) as well as exceeding the toxicity level for the drug. In some embodiments, the composition can be administered in a sustained release formulation.

[00210] Controlled-release pharmaceutical products have a common goal of improving drug therapy over that achieved by their non-controlled release counterparts. Ideally, the use of an optimally designed controlled-release preparation in medical treatment is characterized by a minimum of drug substance being employed to cure or control the condition in a minimum amount of time. Advantages of controlled-release formulations include: 1) extended activity of the drug; 2) reduced dosage frequency; 3) increased patient compliance; 4) usage of less total drug; 5) reduction in local or systemic side effects; 6) minimization of drug accumulation; 7) reduction in blood level fluctuations; 8) improvement in efficacy of treatment; 9) reduction of potentiation or loss of drug activity; and 10) improvement in speed of control of diseases or conditions. Kim, Cherng-ju, Controlled Release Dosage Form Design, 2 (Technomic Publishing, Lancaster, Pa.: 2000).

[00211] Most controlled-release formulations are designed to initially release an amount of drug (active ingredient) that promptly produces the desired therapeutic effect, and gradually and continually release other amounts of drug to maintain this level of therapeutic or prophylactic effect over an extended period of time. In order to maintain this constant level of drug in the body, the drug must be released from the dosage form at a rate that will replace the amount of drug being

metabolized and excreted from the body. Controlled-release of an active ingredient can be stimulated by various conditions including, but not limited to, pH, ionic strength, osmotic pressure, temperature, enzymes, water, and other physiological conditions or compounds.

[00212] A variety of known controlled- or extended-release dosage forms, formulations, and devices can be adapted for use with the salts and compositions of the disclosure. Examples include, but are not limited to, those described in U.S. Pat. Nos.: 3,845,770; 3,916,899; 3,536,809; 3,598,123; 4,008,719; 5,674,533; 5,059,595; 5,591,767; 5,120,548; 5,073,543; 5,639,476; 5,354,556; 5,733,566; and 6,365,185 B1; each of which is incorporated herein by reference. These dosage forms can be used to provide slow or controlled-release of one or more active ingredients using, for example, hydroxypropylmethyl cellulose, other polymer matrices, gels, permeable membranes, osmotic systems (such as OROS® (Alza Corporation, Mountain View, Calif. USA)), or a combination thereof to provide the desired release profile in varying proportions.

[00213] Described herein is a therapy called “CAL-cell therapy”, which seeks to help immune killer cells recognize autoreactive and alloreactive T cells. This is accomplished by genetically altering an immune cell so that it expresses a chimeric antigen ligand (CAL). The CAL is an altered ligand, in which the natural recognition portion is removed and replaced with a synthetic recognition portion, (including all synthetic or natural peptide MHC complexes) that is designed to more effectively recognize the autoreactive and alloreactive T cells by very specifically detecting the presence of a T cell receptor unique to the autoreactive and alloreactive T cells. These CAL immune cells are then given to a patient. Inside the patient, their synthetic CAL ligand molecules will bind to the autoreactive and alloreactive T cells and in the act of that binding, activate the killer immune cells, resulting in the engineered CAL-immune cells attacking the pathologic autoreactive and alloreactive T-cells to eliminate or reduce allo- or autoreactive immune response. This has particular applications for autoimmune conditions, cell -, tissue -, organ- transplants, and Graft vs. Host Disease (GvHD).

[00214] This invention is based, in part, on the finding that an engineered polypeptide presented herein can recognize and bind to the specific T cell receptor on the disease-causing T cells, deleting said T cells, e.g., eliminating or reducing autoimmune diseases or conditions; T cell mediated inflammation or immune response; and cell, tissue, organ transplants and Graft vs. Host Disease (GvHD). In some embodiments, the engineered polypeptide is composed of a peptide-major histocompatibility complex (pMHC) (e.g. as a monomer, oligomer, or multimer) as the recognition site for the TCR of an allogeneic or an autoreactive T cell. In some embodiments, the pMHC is one of the complexes described herein, e.g., see Tables 5 and 6.

[00215] In some embodiments, the engineered polypeptide is composed of a pMHC conjugated to a FITC, PE, or other biomolecular interaction domain described herein. The engineered polypeptide

can also be considered as an adaptor molecule (referred to herein as Chimeric Antigen Ligand (CAL)) composed of a TCR recognition domain (e.g., peptide-HLA monomers, oligomers, or multimers) fused to a biomolecular interaction domain. The CAL technology described herein can target T cell clones in an antigen specific manner and is not dependent on any specific CAR construct to exhibit killing effect.

[00216] In some embodiments, the engineered polypeptide is a CAR composed of a peptide-HLA (e.g., as a monomer, oligomer, or multimer) as the recognition site fused to signaling domains from T cell receptors. Further provided herein is a split version of this CAR system, in which the CAR is composed of two pieces. The first piece is a universal CAR (also referred to herein as a Uni CAL) with T cell signaling domains as the intracellular portion and a biomolecular interaction domain as the extracellular domain, e.g., that is specific to each disease state. The second piece is an adaptor molecule (referred to herein as Chimeric Antigen Ligand (CAL)) composed of a TCR recognition domain (e.g., peptide-HLA monomers, oligomers, or multimers) fused to the cognate biomolecular interaction domain.

[00217] In some embodiments, the engineered polypeptide is a CAR composed of a peptide-HLA (e.g., as a monomer, oligomer, or multimer) as the recognition site fused to signaling domains from T cell receptors. Further provided herein is a split version of this CAR system, in which the CAR is composed of two pieces. The first piece is a universal CAR (also referred to herein as a Uni CAL) with T cell signaling domains as the intracellular portion and a biomolecular interaction domain as the extracellular domain, e.g., that is specific to each disease state. The second piece is an adaptor molecule (referred to herein as Chimeric Antigen Ligand (CAL)) composed of a TCR recognition domain (e.g., peptide-HLA monomers, oligomers, or multimers) fused to the cognate biomolecular interaction domain. It is noted that protein interaction domains are a type of biomolecular interaction domains and where one is specified herein, the other may always be substituted. The CAL technology described herein can target T cells clones in an antigen specific manner and is not dependent on any specific CAR construct to exhibit killing effect.

[00218] Accordingly, one aspect presented herein provides a composition comprising (a) chimeric antigen ligand or a TCR recognition domain; and one or both of (a) an intracellular signaling domain; and (b) a first-type protein interaction domain.

[00219] Another aspect provided herein provides a composition comprising (a) a first polypeptide comprising a chimeric antigen ligand or TCR recognition domain and a first-type protein interaction domain; and (b) a signaling polypeptide comprising a second-type protein interaction domain and an intracellular signaling domain; wherein the first-type and second-type protein interaction domains bind specifically to each other.

[00220] Another aspect provided herein provides a composition comprising (a) a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain; and (b) a

recognition polypeptide comprising a second recognition domain and a third-type protein interaction domain; wherein the first-type and third-type protein interaction domains bind specifically to each other.

[00221] Another aspect provided herein provides a composition comprising (a) a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain; and (b) a signaling polypeptide comprising a second-type protein interaction domain and an intracellular signaling domain; and (c) a recognition polypeptide comprising a second recognition domain and a third-type protein interaction domain; wherein the second-type and third-type protein interaction domains compete for binding to the first-type protein interaction domain.

[00222] In one embodiment of any aspect, the third-type protein interaction domain and first-type protein interaction domain have a higher affinity for each other than the second-type protein interaction domain and first-type protein interaction domain.

[00223] Another aspect provided herein provides a composition comprising (a) a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain; (b) a signaling polypeptide comprising a second-type protein interaction domain, a fourth-type protein interaction domain, and an intracellular signaling domain; and (c) a recognition polypeptide comprising a second recognition domain and a fifth-type protein interaction domain; wherein the first-type protein interaction domain and the second-type protein interaction domain bind specifically to each other; and wherein the fourth-type protein interaction domain and the fifth-type protein interaction domain bind specifically to each other.

[00224] In one embodiment of any aspect, the fourth-type protein interaction domain and fifth-type protein interaction domain have a weaker affinity than the second-type protein interaction domain and first-type protein interaction domain.

[00225] In one embodiment of any aspect, the first polypeptide further comprises a sixth-type protein interaction domain and the recognition polypeptide further comprises a seventh-type protein interaction domain which bind specifically to each other.

[00226] In one embodiment of any aspect, the second recognition domain is specific for a target that is not recognized by the TCR recognition domain.

[00227] In one embodiment of any aspect, the second recognition domain is specific for a target that is found on a healthy and/or non-target cell and not on a diseased and/or target cell.

[00228] TCR recognition domains may not comprise a single polypeptide, but rather comprise two or more polypeptides and may additionally even comprise non-polypeptides. For example, a single MHC class II tetramer TCR recognition domain can comprise 4 biotin small molecules and 16 polypeptides (4 peptides, 4 MHC class II alpha chains, 4 MHC class II beta chains, and 4 streptavidin proteins). Other types of TCR recognition domains may additionally comprise other non-polypeptide molecules (e.g., MHC dextramers contain a polysaccharide backbone to which the MHCs are

anchored to). In some embodiments, a composition described herein can comprise multiple copies or instances of a TCR recognition domain(s), e.g. the TCR recognition domain can be a multimer, or oligomer. In some embodiments, a composition described herein can comprise multiple copies or instances of a first polypeptide as described herein. In some embodiments, the first polypeptide comprises the entire TCR recognition domain. In some embodiments, the TCR recognition domain comprises at least two separate polypeptide sequences, the first polypeptide comprises at least one of the separate polypeptide sequences of the TCR recognition domain, and the first polypeptide is bound to or complexed with a second or further polypeptide sequences of the TCR recognition domain to form a TCR recognition domain.

[00229] In one embodiment of any aspect, the TCR recognition domain comprises a MHC (Major Histocompatibility Complex); a MHC-peptide complex; or a MHC-peptide fusion.

[00230] In one embodiment of any aspect, the peptide is a human, non-human, or synthetic/engineered peptide. The peptides can further comprise non-proteinaceous motifs, modifications, or domains, e.g., they can comprise glycosylation and/or lipids. In one embodiment of any aspect, the peptide is a Minor Histocompatibility Antigen (MiHA).

[00231] In one embodiment of any aspect, the MHC is a monomer, dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.

[00232] In one embodiment of any aspect, the protein interaction domains are found on an extracellular portion of the respective polypeptides.

[00233] In one embodiment of any aspect, (a) the protein interaction domain(s) is a leucine zipper, or any binding pair of protein interaction domains are collectively a pair of leucine zippers; (b) the protein interaction domain(s) is a BZip (RR) and/or a AZip (EE), or any binding pair of protein interaction domains are collectively a BZip (RR) and a AZip (EE); (c) the protein interaction domain(s) is a PSD95-Dlg1-zo-1 (PDZ) domain; (d) the protein interaction domain(s) is a streptavidin and/or a streptavidin binding protein (SBP) or any binding pair of protein interaction domains are collectively a streptavidin and a streptavidin binding protein (SBP); (e) the protein interaction domain(s) is a FKBP-binding domain of mTOR (FRB) and/or a FK506 binding protein (FKBP) or any binding pair of protein interaction domains are collectively a FKBP-binding domain of mTOR (FRB) and a FK506 binding protein (FKBP); (f) the protein interaction domain(s) is a cyclophilin-Fas fusion protein (CyP-Fas) and/or a FK506 binding protein (FKBP) or any binding pair of protein interaction domains are collectively a cyclophilin-Fas fusion protein (CyP-Fas) and a FK506 binding protein (FKBP); (g) the protein interaction domain(s) is a calcineurin A (CNA) and/or a FK506 binding protein (FKBP) or any binding pair of protein interaction domains are collectively a calcineurin A (CNA) and a FK506 binding protein (FKBP); (h) the protein interaction domain(s) is a gibberellin insensitive (GIA) and/or a gibberellin insensitive dwarf1 (GID1) or any binding pair of protein interaction domains are collectively a gibberellin insensitive (GIA) and a gibberellin

insensitive dwarf1 (GID1); (i) the protein interaction domain(s) is a Snap-tag and/or a Halo tag, or any binding pair of protein interaction domains are collectively a Snap-tag and a Halo tag; (j) the protein interaction domain(s) is a T14-3-3-cdeltaC and/or a C-Terminal peptides of PMA2 (CT52), or any binding pair of protein interaction domains are collectively a T14-3-3-cdeltaC and a C-Terminal peptides of PMA2 (CT52); (k) the protein interaction domain(s) is a PYL and/or a ABI, or any binding pair of protein interaction domains are collectively a PYL and a ABI; (l) the protein interaction domain(s) is a nucleotide tag and/or a zinc finger domain, or any binding pair of protein interaction domains are collectively a nucleotide tag and a zinc finger domain; (m) wherein the biomolecular interaction domain(s) is a nucleotide tag, or any binding pair of biomolecular interaction domains are collectively a pair of nucleotide tags; (n) the protein interaction domain(s) is a Fluorescein isothiocyanate (FITC) and/or a FITC binding protein or any binding pair of protein interaction domains are collectively a FITC and a FITC binding protein; and/or (o) the protein interaction domain(s) is a (R)-Phycoerythrin (R-PE/ PE) and/or a R-PE/PE binding protein or any binding pair of protein interaction domains are collectively a FITC and a FITC binding protein.

[00234] In one embodiment of any aspect, the nucleotide tag is a DNA tag or dsDNA tag.

[00235] In one embodiment of any aspect, the intracellular signaling domain is a signaling domain from a protein selected from the group consisting of: TCR ζ , FcR γ , FcR β , CD3 γ , CD35, CD3 ζ , CD3C, CD22, CD79a, CD79b, CD66d, CARD11, CD2, CD7, CD27, CD28, CD30, CD40, CD54 (ICAM), CD83, CD134 (OX40), CD137 (4-1BB), CD150 (SLAMF1), CD152 (CTLA4), CD223 (LAG3), CD270 (HVEM), CD273 (PD-L2), CD274 (PD- L1), CD278 (ICOS), DAP10, LAT, KD2C SLP76, TRIM, and ZAP70.

[00236] In one embodiments of any aspect, a cell comprising and/or expressing a comprising a composition comprising a TCR recognition domain and an intracellular signaling domain further comprises a TCR signaling-responsive promoter operatively linked to a payload transgene. Such embodiments permit transgene payload expression specifically to and/or in the vicinity of a targeted T cell. Suitable promoters and transgene are known in the art, e.g., those promoters and transgenes used in “TRUCK CAR” technology. An exemplary promoter is a NFAT-sensitive promoter. Exemplary transgene payloads can include checkpoint inhibitors (e.g., CTLA-4, [Ipilimumab, Tremelimumab] or PD-1 [Nivolumab, Pembrolizumab, Pidilizumab]) or proinflammatory cytokines (e.g., IL-2, IL-12, etc). In our case this will be used for in-situ targeting of a patient’s anticancer T-cells to very specifically and locally deliver activating agents like one or more checkpoint inhibitors (CTLA-4, [Ipilimumab, Tremelimumab] or PD-1 [Nivolumab, Pembrolizumab, Pidilizumab]) and/or proinflammatory cytokines, (e.g. IL-2, IL-12, etc.) that can push them to expansion and effector phenotype. With this targeted delivery, it is expected to see more efficacy without the systemic side effects caused by checkpoint inhibitors and cytokines administered systemically. Further, combination therapy with multiple different agents is possible with the described technolog as there is minimal

systemic side effect due to paracrine delivery. Further description of suitable promoters, payloads, as well as how to make and use TRUCK technology is described, e.g., Peterson et al. *Front. Oncol.* 2019 9:69; Chmielewski et al. *Advances in Cell and Gene Therapy* 2020 3:e84; Chmielewski et al. *Expert opinion Biol Ther* 2015 15:1145-54; and Chmielewski et al. *Immunol Rev* 2014 257:83-90; each of which is incorporated by reference herein in its entirety. In some embodiments, the cell can be allogeneic, e.g., and engineered once and given as pulse therapy. In some embodiments, the cell can be a T cell or any other cell type described herein, e.g., a NK cell. Exemplary, non-limiting proinflammatory cytokines include IFNs, IFN- γ , TNF α , TGF- β , IL-1 β , IL-6, IL-4, IL-10, IL-13, IL-2, IL-12, IL-15, and IL-27.

[00237] A promoter can be said to drive expression or drive transcription of the nucleic acid sequence that it regulates. The phrases “operably linked,” “operatively positioned,” “operatively linked,” “under control,” and “under transcriptional control” indicate that a promoter is in a correct functional location and/or orientation in relation to a nucleic acid sequence it regulates to control transcriptional initiation and/or expression of that sequence.

[00238] In some embodiments, a composition comprising a TCR recognition domain and an biomolecular interaction domain is a soluble molecule and/or soluble complex.

[00239] Another aspect provided herein is a cell comprising and/or expressing the composition of any of the compositions described herein.

[00240] In one embodiment of any aspect, the TCR recognition domain comprises a MHC allogeneic to the cell comprising and/or expressing the composition. In one embodiment of any aspect, the TCR recognition domain comprises a MHC allogeneic to the cell that the TCR originated from.

[00241] In one embodiment of any aspect, the TCR recognition domain comprises a peptide allogeneic to the cell comprising and/or expressing the composition. In one embodiment of any aspect, the TCR recognition domain comprises a peptide allogeneic to the cell that the TCR originated from.

[00242] In one embodiment of any aspect, the cell is a dendritic cell (CAL DC), a T cell (e.g., effector, regulatory, etc.) (CAL-T); regulatory T cell, effector T cell, natural killer cell (CAL NK), or any other myeloid cell. In one embodiment of any aspect, the cell is engineered to express the polypeptide(s) of the composition. In one embodiment of any aspect, the cell is engineered to express the signaling polypeptide of the composition. In one embodiment of any aspect, the cell is further engineered to knockout the native MHC I/II. In one embodiment of any aspect, the cell is further engineered to lack cell surface expression of native MHC I/II.

[00243] Another aspect provided herein is a chimeric antigen receptor (CAR) comprising (a) an anti-CD127 and/or anti-CD45RO recognition domain; and (b) an intracellular signaling domain.

[00244] Another aspect provided herein is a composition comprising a first polypeptide comprising: (a) an anti-CD127 and/or anti-CD45RO recognition domain; (b) a first-type protein interaction domain; and a second polypeptide comprising (a) a second-type protein interaction domain; and (b) an intracellular signaling domain; wherein the first-type protein interaction domain and the second-type protein interaction domain bind specifically to each other.

[00245] Another aspect provided herein is a composition comprising a first polypeptide comprising (a) an anti-CD127 recognition domain; (b) a first-type protein interaction domain; a second polypeptide comprising (a) an anti-CD45RO recognition domain; (b) a fifth-type protein interaction domain; and a third polypeptide comprising (a) a second-type and a fourth-type protein interaction domain; and (b) an intracellular signaling domain; wherein the first-type protein interaction domain and the second-type protein interaction domain bind specifically to each other; and wherein the fourth-type protein interaction domain and the fifth-type protein interaction domain bind specifically to each other.

[00246] Another aspect provided herein is a cell comprising any of the CARs described herein, or any of the compositions described herein.

[00247] Another aspect provided herein is a method of preventing and/or treating an autoimmune diseases or conditions or T cell mediated inflammation or immune response; or treating or preventing transplant rejection or GvHD in a subject in need thereof, the method comprising administering to the subject any of the compositions and/or cells described herein. Another aspect provided herein is a method of preventing and/or treating a malignant T cell condition in a subject in need thereof, the method comprising administering to the subject any of the compositions and/or cells described herein.

[00248] In various embodiments of any aspect, the TCR recognition domain comprises a MHC allogeneic to the subject, a MHC autologous to the transplant cells, a peptide allogeneic to the subject, or a peptide autologous to the transplant cells.

[00249] In one embodiment of any aspect, the transplant is vascularized composite allotransplantation (VCA).

[00250] In one embodiment of any aspect, the autoimmune disease is type 1 diabetes, multiple sclerosis, rheumatoid arthritis, or scleroderma.

[00251] One aspect of the embodiments provided herein is a CAR T cell that targets a CD127+/CD45RO+ T cell. In one embodiment, the CD127+/CD45RO+ T cell is a CD127+/CD45RO+ memory T cell. In one embodiment, the CD127+/CD45RO+ T cell is an alloreactive CD127+/CD45RO+ T cell.

[00252] Accordingly, one aspect herein provides a CAR comprising (a) an anti-CD127 and/or anti-CD45RO recognition domain; and (b) intracellular signaling domain. Further provided herein is a composition comprising (a) an anti-CD127 and/or anti-CD45RO recognition domain; and (b) intracellular signaling domain.

[00253] Another aspect of the provides a composition comprising a first polypeptide comprising: (a) an anti-CD127 and/or anti-CD45RO recognition domain; (b) a first-type protein interaction domain; and a second polypeptide comprising: (c) a second-type protein interaction domain; and (d) an intracellular signaling domain; wherein the first-type protein interaction domain and the second-type protein interaction domain bind specifically to each other.

[00254] Another aspect of the provides a composition comprising a first polypeptide comprising: (a) an anti-CD127 recognition domain; (b) a first-type protein interaction domain; a second polypeptide comprising: (c) an anti-CD45RO recognition domain; (d) a fifth-type protein interaction domain; and a third polypeptide comprising: (e) a second-type and a fourth-type protein interaction domain; and (f) an intracellular signaling domain; wherein the first-type protein interaction domain and the second-type protein interaction domain bind specifically to each other; and wherein the fourth-type protein interaction domain and the fifth-type protein interaction domain bind specifically to each other.

[00255] In one embodiment, the anti-CD127 recognition domain recognizes and binds to the sequence of CD127 on a CD127+ cell, e.g., a CD127+/CD45RO+ T cell. As used herein, “CD127”, also referred to as Interleukin 7 receptor a (IL7Ra), ILRA, IL7RA, CDW127, or IL-7R-alpha, is a cell surface receptor that has been shown to have a role in V(D)J recombination during lymphocyte development. Defects in CD127 have been associated with severe combined immunodeficiency (SCID). Sequences for CD127 are known for a number of species, e.g., human CD127 (NCBI Gene ID: 3575), mRNA (NCBI Ref Seq: NM_002185.5), and polypeptide (NCBI Ref Seq: NP_002176.2). CD127 refers to all naturally occurring variants or isoforms of CD127. In one embodiment, the CD127 polypeptide sequence is presented in SEQ ID NO: 1. In some embodiments of any of the aspects, the CD127 polypeptide can be an ortholog, variant, and/or allele of SEQ ID NO: 1.

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1  MTILGTTFGM VFSLQVVSF ESGYAQNGDL EDAELDDYSF SCYSQLEVNG
SQHSLTCAFE
61  DPDVNITNLE FEICGALVEV KCLNFRKLQE IYFIETKKFL LIGKSNICVK
VGEKSLTCKK
121 IDLTTIVKPE APFDLSVVYR EGANDFVVTF NTSHLQKKYV KVLMDHVAIR
QEKDENKWITH
181 VNLSSTKLTL LQRKLQPAAM YEIKVRSIPD HYFKGFWSEW SPSYYFRTPE
INNSSGEMDP
241 ILLTISILSF FSVALLVILA CVLWKKRIKP IVWPSLPDHK KTLLEHLCKKP
RKNLNVSFNP
301 ESFLDCQIHR VDDIQARDEV EGFLQDTFPQ QLEESEKQRL GGDVQSPNCP
SEDEVITPES

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361 FGRDSSLTCL AGNVSACDAP ILSSSRSLDC RESGKNPHV YQDLLLSLGT
TNSTLPPPF

421 LQSGILTLPN VAQGQPILTS LGSNQEEAYV TMSSFYQNO (SEQ ID NO: 1)

[00256] In one embodiment, the anti-CD45RO recognition domain recognizes and binds to the sequence of CD45RO on a CD45RO+ cell, e.g., a CD127+/CD45RO+ T cell. As used herein, “CD45RO”, also referred to as PTPRC, LCA, LY5, B220, CD45, L-CA, T200, CD45R, and GP180, is to a cell surface signaling molecule that been shown to be an essential regulator of T- and B-cell antigen receptor signaling. Sequences for CD45RO are known for a number of species, e.g., human CD45RO (NCBI Gene ID: 5788), mRNA (NCBI Ref Seq: NM_001267798.2), and polypeptide (NCBI Ref Seq: NP_001254727.1). CD45RO refers to all naturally occurring variants or isoforms of CD45RO. In one embodiment, the CD45RO polypeptide sequence is presented in SEQ ID NO: 2. In some embodiments of any of the aspects, the CD45RO polypeptide can be an ortholog, variant, and/or allele of SEQ ID NO: 2.

1 MTMYLWLKLL AFGFAFLDTE VFVTGQSPTP SPTGHLQAE QGSQSKSPNL
KSREADSSAF
61 SWWPKAREPL TNHWSKSKSP KAEELGV (SEQ ID NO: 2)

[00257] For convenience, the meaning of some terms and phrases used in the specification, examples, and appended claims, are provided below. Unless stated otherwise, or implicit from context, the following terms and phrases include the meanings provided below. The definitions are provided to aid in describing particular embodiments, and are not intended to limit the claimed invention, because the scope of the invention is limited only by the claims. Unless otherwise defined, all technical and scientific terms used herein have the same meaning as commonly understood by one of ordinary skill in the art to which this invention belongs. If there is an apparent discrepancy between the usage of a term in the art and its definition provided herein, the definition provided within the specification shall prevail.

[00258] For convenience, certain terms employed herein, in the specification, examples and appended claims are collected here.

[00259] The terms “decrease”, “reduced”, “reduction”, or “inhibit” are all used herein to mean a decrease by a statistically significant amount. In some embodiments, “reduce,” “reduction” or “decrease” or “inhibit” typically means a decrease by at least 10% as compared to a reference level (e.g. the absence of a given treatment) and can include, for example, a decrease by at least about 10%, at least about 20%, at least about 25%, at least about 30%, at least about 35%, at least about 40%, at least about 45%, at least about 50%, at least about 55%, at least about 60%, at least about 65%, at least about 70%, at least about 75%, at least about 80%, at least about 85%, at least about 90%, at least about 95%, at least about 98%, at least about 99%, or more. As used herein, “reduction” or

“inhibition” does not encompass a complete inhibition or reduction as compared to a reference level. “Complete inhibition” is a 100% inhibition as compared to a reference level. A decrease can be preferably down to a level accepted as within the range of normal for an individual without a given disorder.

[00260] The terms “increased”, “increase”, “enhance”, or “activate” are all used herein to mean an increase by a statically significant amount. In some embodiments, the terms “increased”, “increase”, “enhance”, or “activate” can mean an increase of at least 10% as compared to a reference level, for example an increase of at least about 20%, or at least about 30%, or at least about 40%, or at least about 50%, or at least about 60%, or at least about 70%, or at least about 80%, or at least about 90% or up to and including a 100% increase or any increase between 10-100% as compared to a reference level, or at least about a 2-fold, or at least about a 3-fold, or at least about a 4-fold, or at least about a 5-fold or at least about a 10-fold increase, or any increase between 2-fold and 10-fold or greater as compared to a reference level. In the context of a marker or symptom, a “increase” is a statistically significant increase in such level.

[00261] As used herein, a “subject” means a human or animal. Usually the animal is a vertebrate such as a swine, primate, rodent, domestic animal or game animal. Primates include chimpanzees, cynomolgus monkeys, spider monkeys, and macaques, e.g., Rhesus. Rodents include mice, rats, woodchucks, ferrets, rabbits and hamsters. Domestic and game animals include cows, horses, pigs, deer, bison, buffalo, feline species, e.g., domestic cat, canine species, e.g., dog, fox, wolf, avian species, e.g., chicken, emu, ostrich, and fish, e.g., trout, catfish and salmon. In some embodiments, the subject is a mammal, e.g., a primate, e.g., a human. The terms, “individual,” “patient” and “subject” are used interchangeably herein.

[00262] Preferably, the subject is a mammal. The mammal can be a human, non-human swine, primate, mouse, rat, dog, cat, horse, or cow, but is not limited to these examples. Mammals other than humans can be advantageously used as subjects that represent animal models of autoimmune diseases or conditions; T cell mediated inflammation or immune response; malignant T cell condition; transplant rejection; or GvHD. A subject can be male or female.

[00263] A subject can be one who has been previously diagnosed with or identified as suffering from or having a condition in need of treatment (e.g. an autoimmune disease or condition; T cell mediated inflammation or immune response; malignant T cell condition; transplant rejection; or GvHD) or one or more complications related to such a condition, and optionally, have already undergone treatment for an autoimmune disease or condition; T cell mediated inflammation or immune response; malignant T cell condition; transplant rejection; or GvHD or the one or more complications related to an autoimmune disease, transplant rejection, or GvHD. Alternatively, a subject can also be one who has not been previously diagnosed as having an autoimmune disease or condition; T cell mediated inflammation or immune response; malignant T cell condition; transplant

rejection; or GvHD or one or more complications related to an autoimmune disease or condition; T cell mediated inflammation or immune response; malignant T cell condition; transplant rejection; or GvHD. For example, a subject can be one who exhibits one or more risk factors for an autoimmune disease or condition; T cell mediated inflammation or immune response; malignant T cell condition; transplant rejection; or GvHD or one or more complications related to an autoimmune disease or condition; T cell mediated inflammation or immune response; malignant T cell condition; transplant rejection; or GvHD or a subject who does not exhibit risk factors.

[00264] A “subject in need” of treatment for a particular condition can be a subject having that condition, diagnosed as having that condition, or at risk of developing that condition, e.g., an autoimmune disease or condition; T cell mediated inflammation or immune response; malignant T cell condition; transplant rejection; or GvHD.

[00265] In some embodiments, a nucleic acid encoding a CAL, CAR, a multi-component CAL and/or CAR or portion thereof as described herein is comprised by a vector. In some of the aspects described herein, a nucleic acid sequence encoding a multi-component CAL and/or CAR, or portion thereof as described herein, or any module thereof, is operably linked to a vector. The term “vector”, as used herein, refers to a nucleic acid construct designed for delivery to a host cell or for transfer between different host cells. As used herein, a vector can be viral or non-viral. The term “vector” encompasses any genetic element that is capable of replication when associated with the proper control elements and that can transfer gene sequences to cells. A vector can include, but is not limited to, a cloning vector, an expression vector, a plasmid, phage, transposon, cosmid, chromosome, virus, virion, etc.

[00266] As used herein, the term “expression vector” refers to a vector that directs expression of an RNA or polypeptide from sequences linked to transcriptional regulatory sequences on the vector. The sequences expressed will often, but not necessarily, be heterologous to the cell. An expression vector may comprise additional elements, for example, the expression vector may have two replication systems, thus allowing it to be maintained in two organisms, for example in human cells for expression and in a prokaryotic host for cloning and amplification. The term “expression” refers to the cellular processes involved in producing RNA and proteins and as appropriate, secreting proteins, including where applicable, but not limited to, for example, transcription, transcript processing, translation and protein folding, modification and processing. “Expression products” include RNA transcribed from a gene, and polypeptides obtained by translation of mRNA transcribed from a gene. The term “gene” means the nucleic acid sequence that is transcribed (DNA) to RNA in vitro or in vivo when operably linked to appropriate regulatory sequences. The gene may or may not include regions preceding and following the coding region, e.g. 5' untranslated (5'UTR) or “leader” sequences and 3' UTR or “trailer” sequences, as well as intervening sequences (introns) between individual coding segments (exons).

[00267] As used herein, the term “viral vector” refers to a nucleic acid vector construct that includes at least one element of viral origin and has the capacity to be packaged into a viral vector particle. The viral vector can contain the nucleic acid encoding a CAL and/or CAR described herein, e.g., a multi-component CAL and/or CAR, or portion thereof as described herein in place of non-essential viral genes. The vector and/or particle may be utilized for the purpose of transferring any nucleic acids into cells either in vitro or in vivo. Numerous forms of viral vectors are known in the art.

[00268] By “recombinant vector” is meant a vector that includes a heterologous nucleic acid sequence, or “transgene” that is capable of expression in vivo or in the transduced cells. It should be understood that the vectors described herein can, in some embodiments, be combined with other suitable compositions and therapies. In some embodiments, the vector is episomal. The use of a suitable episomal vector provides a means of maintaining the nucleotide of interest in the subject in high copy number extra chromosomal DNA thereby eliminating potential effects of chromosomal integration.

[00269] As used herein, the term “nucleic acid” or “nucleic acid sequence” refers to any molecule, preferably a polymeric molecule, incorporating units of ribonucleic acid, deoxyribonucleic acid or an analog thereof. The nucleic acid can be either single-stranded or double-stranded. A single-stranded nucleic acid can be one nucleic acid strand of a denatured double-stranded DNA. Alternatively, it can be a single-stranded nucleic acid not derived from any double-stranded DNA. In one aspect, the nucleic acid can be DNA. In another aspect, the nucleic acid can be RNA, e.g., single-stranded or double-stranded RNA. Suitable nucleic acid molecules are DNA, including genomic DNA or cDNA. Other suitable nucleic acid molecules are RNA, including mRNA.

[00270] As used herein, the terms “protein” and “polypeptide” are used interchangeably herein to designate a series of amino acid residues, connected to each other by peptide bonds between the alpha-amino and carboxy groups of adjacent residues. The terms “protein”, and “polypeptide” refer to a polymer of amino acids, including modified amino acids (e.g., phosphorylated, glycosylated, glycosylated, etc.) and amino acid analogs, regardless of its size or function. “Protein” and “polypeptide” are often used in reference to relatively large polypeptides, whereas the term “peptide” is often used in reference to small polypeptides, but usage of these terms in the art overlaps. The terms “protein” and “polypeptide” are used interchangeably herein when referring to a gene product and fragments thereof. Thus, exemplary polypeptides or proteins include gene products, naturally occurring proteins, homologs, orthologs, paralogs, fragments and other equivalents, variants, fragments, and analogs of the foregoing.

[00271] As used herein an “antibody” refers to IgG, IgM, IgA, IgD or IgE molecules or antigen-specific antibody fragments thereof (including, but not limited to, a Fab, F(ab')₂, Fv, disulphide linked Fv, scFv, single domain antibody, closed conformation multispecific antibody, disulphide-linked scfv, diabody), whether derived from any species that naturally produces an antibody, or created by

recombinant DNA technology; whether isolated from serum, B-cells, hybridomas, transfectomas, yeast or bacteria.

[00272] As described herein, an "antigen" is a molecule that is bound by a binding site on an antibody agent. Typically, antigens are bound by antibody ligands and are capable of raising an antibody response in vivo. An antigen can be a polypeptide, protein, nucleic acid or other molecule or portion thereof. The term "antigenic determinant" refers to an epitope on the antigen recognized by an antigen-binding molecule, and more particularly, by the antigen-binding site of said molecule.

[00273] As used herein, the term "antibody reagent" refers to a polypeptide that includes at least one immunoglobulin variable domain or immunoglobulin variable domain sequence and which specifically binds a given antigen. An antibody reagent can comprise an antibody or a polypeptide comprising an antigen-binding domain of an antibody. In some embodiments, an antibody reagent can comprise a monoclonal antibody or a polypeptide comprising an antigen-binding domain of a monoclonal antibody. For example, an antibody can include a heavy (H) chain variable region (abbreviated herein as VH), and a light (L) chain variable region (abbreviated herein as VL). In another example, an antibody includes two heavy (H) chain variable regions and two light (L) chain variable regions. The term "antibody reagent" encompasses antigen-binding fragments of antibodies (e.g., single chain antibodies, Fab and sFab fragments, F(ab')₂, Fd fragments, Fv fragments, scFv, and domain antibodies (dAb) fragments (see, e.g. de Wildt et al., Eur J. Immunol. 1996; 26(3):629-39; which is incorporated by reference herein in its entirety)) as well as complete antibodies. An antibody can have the structural features of IgA, IgG, IgE, IgD, IgM (as well as subtypes and combinations thereof). Antibodies can be from any source, including mouse, rabbit, pig, rat, and primate (human and non-human primate) and primatized antibodies. Antibodies also include midibodies, humanized antibodies, chimeric antibodies, and the like.

[00274] The VH and VL regions can be further subdivided into regions of hypervariability, termed "complementarity determining regions" ("CDR"), interspersed with regions that are more conserved, termed "framework regions" ("FR"). The extent of the framework region and CDRs has been precisely defined (see, Kabat, E. A., et al. (1991) Sequences of Proteins of Immunological Interest, Fifth Edition, U.S. Department of Health and Human Services, NIH Publication No. 91-3242, and Chothia, C. et al. (1987) J. Mol. Biol. 196:901-917; which are incorporated by reference herein in their entireties). Each VH and VL is typically composed of three CDRs and four FRs, arranged from amino-terminus to carboxy-terminus in the following order: FR1, CDR1, FR2, CDR2, FR3, CDR3, FR4.

[00275] The terms "antigen-binding fragment" or "antigen-binding domain", which are used interchangeably herein are used to refer to one or more fragments of a full length antibody that retain the ability to specifically bind to a target of interest. Examples of binding fragments encompassed within the term "antigen-binding fragment" of a full length antibody include (i) a Fab fragment, a

monovalent fragment consisting of the VL, VH, CL and CH1 domains; (ii) a F(ab')₂ fragment, a bivalent fragment including two Fab fragments linked by a disulfide bridge at the hinge region; (iii) an Fd fragment consisting of the VH and CH1 domains; (iv) an Fv fragment consisting of the VL and VH domains of a single arm of an antibody, (v) a dAb fragment (Ward et al., (1989) Nature 341:544-546; which is incorporated by reference herein in its entirety), which consists of a VH or VL domain; and (vi) an isolated complementarity determining region (CDR) that retains specific antigen-binding functionality.

[00276] As used herein, the term “specific binding” refers to a chemical interaction between two molecules, compounds, cells and/or particles wherein the first entity binds to the second, target entity with greater specificity and affinity than it binds to a third entity which is a non-target. In some embodiments, specific binding can refer to an affinity of the first entity for the second target entity which is at least 10 times, at least 50 times, at least 100 times, at least 500 times, at least 1000 times or greater than the affinity for the third nontarget entity. A reagent specific for a given target is one that exhibits specific binding for that target under the conditions of the assay being utilized. In some embodiments, binding described herein can be preferential binding, e.g., binding between two molecules, compounds, cells and/or particles wherein the first entity binds to the second, target entity with at least 2 times greater specificity and affinity than it binds to a third entity which is a non-target.

[00277] Additionally, and as described herein, a recombinant humanized antibody can be further optimized to decrease potential immunogenicity, while maintaining functional activity, for therapy in humans. In this regard, functional activity means a polypeptide capable of displaying one or more known functional activities associated with a recombinant antibody or antibody reagent thereof as described herein. Such functional activities include, e.g. the ability to bind to a target.

[00278] As used herein, the terms “treat,” “treatment,” “treating,” or “amelioration” refer to therapeutic treatments, wherein the object is to reverse, alleviate, ameliorate, inhibit, slow down or stop the progression or severity of a condition associated with a disease or disorder, e.g. autoimmune disease or condition; T cell mediated inflammation or immune response; malignant T cell condition; transplant rejection; or GvHD. The term “treating” includes reducing or alleviating at least one adverse effect or symptom of a condition, disease or disorder associated with a, e.g. autoimmune disease or condition; T cell mediated inflammation or immune response; malignant T cell condition; transplant rejection; or GvHD. Treatment is generally “effective” if one or more symptoms or clinical markers are reduced. Alternatively, treatment is “effective” if the progression of a disease is reduced or halted. That is, “treatment” includes not just the improvement of symptoms or markers, but also a cessation of, or at least slowing of, progress or worsening of symptoms compared to what would be expected in the absence of treatment. Beneficial or desired clinical results include, but are not limited to, alleviation of one or more symptom(s), diminishment of extent of disease, stabilized (i.e., not worsening) state of disease, delay or slowing of disease progression, amelioration or palliation of the

disease state, remission (whether partial or total), and/or decreased mortality, whether detectable or undetectable. The term "treatment" of a disease also includes providing relief from the symptoms or side-effects of the disease (including palliative treatment).

[00279] As used herein, the term "pharmaceutical composition" refers to the active agent in combination with a pharmaceutically acceptable carrier e.g. a carrier commonly used in the pharmaceutical industry. The phrase "pharmaceutically acceptable" is employed herein to refer to those compounds, materials, compositions, and/or dosage forms which are, within the scope of sound medical judgment, suitable for use in contact with the tissues of human beings and animals without excessive toxicity, irritation, allergic response, or other problem or complication, commensurate with a reasonable benefit/risk ratio.

[00280] As used herein, the term "administering," refers to the placement of an agent, e.g., a CAL, CAR, composition, or cell as disclosed herein into a subject by a method or route which results in at least partial delivery of the agent at a desired site. Pharmaceutical compositions comprising the compounds disclosed herein can be administered by any appropriate route which results in an effective treatment in the subject.

[00281] Immune checkpoint inhibitors inhibit one or more immune checkpoint proteins. The immune system has multiple inhibitory pathways that are critical for maintaining self-tolerance and modulating immune responses. For example, in T-cells, the amplitude and quality of response is initiated through antigen recognition by the T-cell receptor and is regulated by immune checkpoint proteins that balance co-stimulatory and inhibitory signals. In some embodiments of any of the aspects, a subject or patient is treated with at least one inhibitor of an immune checkpoint protein. As used herein, "immune checkpoint protein" refers to a protein which, when active, exhibits an inhibitory effect on immune activity, e.g., T cell activity. Exemplary immune checkpoint proteins can include PD-1 (e.g., NCBI Gene ID: 5133); PD-L1 (e.g., NCBI Gene ID: 29126); PD-L2 (e.g., NCBI Gene ID: 80380); TIM-3 (e.g., NCBI Gene ID: 84868); CTLA4 (e.g., NCBI Gene ID: 1493); TIGIT (e.g., NCBI Gene ID: 201633); KIR (e.g., NCBI Gene ID: 3811); LAG3 (e.g., NCBI Gene ID: 3902); DD1- α (e.g., NCBI Gene ID: 64115); A2AR (e.g., NCBI Gene ID: 135); B7-H3 (e.g., NCBI Gene ID: 80381); B7-H4 (e.g., NCBI Gene ID: 79679); BTLA (e.g., NCBI Gene ID: 151888); IDO (e.g., NCBI Gene ID: 3620); TDO (e.g., NCBI Gene ID: 6999); HVEM (e.g., NCBI Gene ID: 8764); GAL9 (e.g., NCBI Gene ID: 3965); 2B4 (belongs to the CD2 family of molecules and is expressed on all NK, $\gamma\delta$, and memory CD8+ ($\alpha\beta$) T cells) (e.g., NCBI Gene ID: 51744); CD160 (also referred to as BY55) (e.g., NCBI Gene ID: 11126); and various B-7 family ligands. B7 family ligands include, but are not limited to, B7- 1, B7-2, B7-DC, B7-H1, B7-H2, B7-H3, B7-H4, B7-H5, B7-H6 and B7-H7.

[00282] Non-limiting examples of immune checkpoint inhibitors (with checkpoint targets and manufacturers noted in parentheses) can include: MGA271 (B7-H3: MacroGenics); ipilimumab (CTLA-4; Bristol Meyers Squibb); pembrolizumab (PD-1; Merck); nivolumab (PD-1; Bristol Meyers

Squibb); atezolizumab (PD-L1; Genentech); galiximab (B7.1; Biogen); IMP321 (LAG3; Immunetp); BMS-986016 (LAG3; Bristol Meyers Squibb); SMB-663513 (CD137; Bristol-Meyers Squibb); PF-05082566 (CD137; Pfizer); IPH2101 (KIR; Innate Pharma); KW-0761 (CCR4; Kyowa Kirin); CDX-1127 (CD27; CellDex); MEDI-6769 (Ox40; MedImmune); CP-870,893 (CD40; Genentech); tremelimumab (CTLA-4; Medimmune); pidilizumab (PD-1; Medivation); MPDL3280A (PD-L1; Roche); MEDI4736 (PD-L1; AstraZeneca); MSB0010718C (PD-L1; EMD Serono); AUNP12 (PD-1; Aurigene); avelumab (PD-L1; Merck); durvalumab (PD-L1; Medimmune); IMP321, a soluble Ig fusion protein (Brignone et al., 2007, J. Immunol. 179:4202-4211); the anti-B7-H3 antibody MGA271 (Loo et al., 2012, Clin. Cancer Res. July 15 (18) 3834); TIM3 (T-cell immunoglobulin domain and mucin domain 3) inhibitors (Fourcade et al., 2010, J. Exp. Med. 207:2175-86 and Sakuishi et al., 2010, J. Exp. Med. 207:2187-94); anti-CTLA-4 antibodies described in US Patent Nos: 5,811,097; 5,811,097; 5,855,887; 6,051,227; 6,207,157; 6,682,736; 6,984,720; and 7,605,238; tremelimumab, (ticilimumab, CP-675,206); ipilimumab (also known as 10D1, MDX-D010); PD-1 and PD-L1 blockers described in US Patent Nos. 7,488,802; 7,943,743; 8,008,449; 8,168,757; 8,217,149, and PCT Published Patent Application Nos: WO03042402, WO2008156712, WO2010089411, WO2010036959, WO2011066342, WO2011159877, WO2011082400, and WO2011161699; nivolumab (MDX 1106, BMS 936558, ONO 4538); lambrolizumab (MK-3475 or SCH 900475); CT-011; AMP-224; and BMS-936559 (MDX- 1105-01). The foregoing references are incorporated by reference herein in their entireties.

[00283] The term “statistically significant” or “significantly” refers to statistical significance, e.g., a rejection of the null hypothesis with a p-value of less than 0.05 and generally means a two standard deviation (2SD) or greater difference.

[00284] Other than in the operating examples, or where otherwise indicated, all numbers expressing quantities of ingredients or reaction conditions used herein should be understood as modified in all instances by the term “about.” The term “about” when used in connection with percentages can mean $\pm 1\%$.

[00285] As used herein the term “comprising” or “comprises” is used in reference to compositions, methods, and respective component(s) thereof, that are essential to the method or composition, yet open to the inclusion of unspecified elements, whether essential or not.

[00286] The term “consisting of” refers to compositions, methods, and respective components thereof as described herein, which are exclusive of any element not recited in that description of the embodiment.

[00287] As used herein the term “consisting essentially of” refers to those elements required for a given embodiment. The term permits the presence of elements that do not materially affect the basic and novel or functional characteristic(s) of that embodiment.

[00288] The singular terms "a," "an," and "the" include plural referents unless context clearly indicates otherwise. Similarly, the word "or" is intended to include "and" unless the context clearly indicates otherwise. Although methods and materials similar or equivalent to those described herein can be used in the practice or testing of this disclosure, suitable methods and materials are described below. The abbreviation, "e.g." is derived from the Latin *exempli gratia* and is used herein to indicate a non-limiting example. Thus, the abbreviation "e.g." is synonymous with the term "for example."

[00289] Unless otherwise defined herein, scientific and technical terms used in connection with the present application shall have the meanings that are commonly understood by those of ordinary skill in the art to which this disclosure belongs. It should be understood that this invention is not limited to the particular methodology, protocols, and reagents, etc., described herein and as such can vary. The terminology used herein is for the purpose of describing particular embodiments only and is not intended to limit the scope of the present invention, which is defined solely by the claims.

Definitions of common terms in immunology and molecular biology can be found in The Merck Manual of Diagnosis and Therapy, 19th Edition, published by Merck Sharp & Dohme Corp., 2011 (ISBN 978-0-911910-19-3); Robert S. Porter et al. (eds.), The Encyclopedia of Molecular Cell Biology and Molecular Medicine, published by Blackwell Science Ltd., 1999-2012 (ISBN 9783527600908); and Robert A. Meyers (ed.), Molecular Biology and Biotechnology: a Comprehensive Desk Reference, published by VCH Publishers, Inc., 1995 (ISBN 1-56081-569-8); Immunology by Werner Luttmann, published by Elsevier, 2006; Janeway's Immunobiology, Kenneth Murphy, Allan Mowat, Casey Weaver (eds.), Taylor & Francis Limited, 2014 (ISBN 0815345305, 9780815345305); Lewin's Genes XI, published by Jones & Bartlett Publishers, 2014 (ISBN-1449659055); Michael Richard Green and Joseph Sambrook, Molecular Cloning: A Laboratory Manual, 4th ed., Cold Spring Harbor Laboratory Press, Cold Spring Harbor, N.Y., USA (2012) (ISBN 1936113414); Davis et al., Basic Methods in Molecular Biology, Elsevier Science Publishing, Inc., New York, USA (2012) (ISBN 044460149X); Laboratory Methods in Enzymology: DNA, Jon Lorsch (ed.) Elsevier, 2013 (ISBN 0124199542); Current Protocols in Molecular Biology (CPMB), Frederick M. Ausubel (ed.), John Wiley and Sons, 2014 (ISBN 047150338X, 9780471503385), Current Protocols in Protein Science (CPPS), John E. Coligan (ed.), John Wiley and Sons, Inc., 2005; and Current Protocols in Immunology (CPI) (John E. Coligan, ADA M Kruisbeek, David H Margulies, Ethan M Shevach, Warren Strobe, (eds.) John Wiley and Sons, Inc., 2003 (ISBN 0471142735, 9780471142737), the contents of which are all incorporated by reference herein in their entireties.

[00290] Other terms are defined herein within the description of the various aspects of the invention.

[00291] All patents and other publications; including literature references, issued patents, published patent applications, and co-pending patent applications; cited throughout this application are expressly incorporated herein by reference for the purpose of describing and disclosing, for

example, the methodologies described in such publications that might be used in connection with the technology described herein. These publications are provided solely for their disclosure prior to the filing date of the present application. Nothing in this regard should be construed as an admission that the inventors are not entitled to antedate such disclosure by virtue of prior invention or for any other reason. All statements as to the date or representation as to the contents of these documents is based on the information available to the applicants and does not constitute any admission as to the correctness of the dates or contents of these documents.

[00292] The description of embodiments of the disclosure is not intended to be exhaustive or to limit the disclosure to the precise form disclosed. While specific embodiments of, and examples for, the disclosure are described herein for illustrative purposes, various equivalent modifications are possible within the scope of the disclosure, as those skilled in the relevant art will recognize. For example, while method steps or functions are presented in a given order, alternative embodiments may perform functions in a different order, or functions may be performed substantially concurrently. The teachings of the disclosure provided herein can be applied to other procedures or methods as appropriate. The various embodiments described herein can be combined to provide further embodiments. Aspects of the disclosure can be modified, if necessary, to employ the compositions, functions and concepts of the above references and application to provide yet further embodiments of the disclosure. Moreover, due to biological functional equivalency considerations, some changes can be made in protein structure without affecting the biological or chemical action in kind or amount. These and other changes can be made to the disclosure in light of the detailed description. All such modifications are intended to be included within the scope of the appended claims.

[00293] Specific elements of any of the foregoing embodiments can be combined or substituted for elements in other embodiments. Furthermore, while advantages associated with certain embodiments of the disclosure have been described in the context of these embodiments, other embodiments may also exhibit such advantages, and not all embodiments need necessarily exhibit such advantages to fall within the scope of the disclosure.

[00294] All patents and other publications identified in the specification and examples are expressly incorporated herein by reference for all purposes. These publications are provided solely for their disclosure prior to the filing date of the present application. Nothing in this regard should be construed as an admission that the inventors are not entitled to antedate such disclosure by virtue of prior invention or for any other reason. All statements as to the date or representation as to the contents of these documents is based on the information available to the applicants and does not constitute any admission as to the correctness of the dates or contents of these documents.

[00295] Although preferred embodiments have been depicted and described in detail herein, it will be apparent to those skilled in the relevant art that various modifications, additions, substitutions, and the like can be made without departing from the spirit of the invention and these are therefore

considered to be within the scope of the invention as defined in the claims which follow. Further, to the extent not already indicated, it will be understood by those of ordinary skill in the art that any one of the various embodiments herein described and illustrated can be further modified to incorporate features shown in any of the other embodiments disclosed herein.

[00296] Some embodiments of the technology described herein can be defined according to any of the following numbered paragraphs:

1. A composition, comprising:
 - a. a TCR recognition domain; and
 - one or both of:
 - b. an intracellular signaling domain; and
 - c. a first-type protein interaction domain.
2. A composition comprising:
 - a. a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain; and
 - b. a signaling polypeptide comprising a second-type protein interaction domain and an intracellular signaling domain;

wherein the first-type and second-type protein interaction domains bind specifically to each other.

3. A composition comprising:
 - a. a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain; and
 - b. a recognition polypeptide comprising a second recognition domain and a third-type protein interaction domain;

wherein the first-type and third-type protein interaction domains bind specifically to each other.

4. A composition comprising:
 - a. a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain; and
 - b. a signaling polypeptide comprising a second-type protein interaction domain and an intracellular signaling domain; and
 - c. a recognition polypeptide comprising a second recognition domain and a third-type protein interaction domain;

wherein the second-type and third-type protein interaction domains compete for binding to the first-type protein interaction domain.

5. The composition of any of paragraphs 3-4, wherein the third-type protein interaction domain and first-type protein interaction domain have a higher affinity for each other than the second-type protein interaction domain and first-type protein interaction domain.
6. A composition comprising:
 - a. a first polypeptide comprising a TCR recognition domain and a first-type protein interaction domain;
 - b. a signaling polypeptide comprising a second-type protein interaction domain, a fourth-type protein interaction domain, and an intracellular signaling domain; and
 - c. a recognition polypeptide comprising a second recognition domain and a fifth-type protein interaction domain;

wherein the first-type protein interaction domain and the second-type protein interaction domain bind specifically to each other; and

wherein the fourth-type protein interaction domain and the fifth-type protein interaction domain bind specifically to each other.
7. The composition of paragraph 6, wherein the fourth-type protein interaction domain and fifth-type protein interaction domain have a weaker affinity than the second-type protein interaction domain and first-type protein interaction domain.
8. The composition of any of paragraphs 6-7, wherein the first polypeptide further comprises a sixth-type protein interaction domain and the recognition polypeptide further comprises a seventh-type protein interaction domain which bind specifically to each other.
9. The composition of any of paragraphs 3-8, wherein the second recognition domain is specific for a target that is not recognized by the TCR recognition domain.
10. The composition of any of paragraphs 3-9, wherein the second recognition domain is specific for a target that is found on a healthy and/or non-target cell and not on a diseased and/or target cell.
11. The composition of any of the preceding paragraphs, wherein the TCR recognition domain comprises a MHC (Major Histocompatibility Complex); a MHC-peptide complex; or a MHC-peptide fusion.
12. The composition of paragraph 11, wherein the peptide is a human or non-human peptide.
13. The composition of any of paragraphs 11-12, wherein the peptide is a Minor Histocompatibility Antigen (MiHA).
14. The composition of any of paragraphs 11-13, wherein the MHC is a monomer, dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.
15. The composition of any of the preceding paragraphs, wherein the protein interaction domains are found on an extracellular portion of the respective polypeptides.
16. The composition of any of the preceding paragraphs,

- a. wherein the protein interaction domain(s) is a leucine zipper, or any binding pair of protein interaction domains are collectively a pair of leucine zippers;
 - b. wherein the protein interaction domain(s) is a BZip (RR) and/or a AZip (EE), or any binding pair of protein interaction domains are collectively a BZip (RR) and a AZip (EE);
 - c. wherein the protein interaction domain(s) is a PSD95-Dlg1-zo-1 (PDZ) domain;
 - d. wherein the protein interaction domain(s) is a streptavidin and/or a streptavidin binding protein (SBP) or any binding pair of protein interaction domains are collectively a streptavidin and a streptavidin binding protein (SBP);
 - e. wherein the protein interaction domain(s) is a FKBP-binding domain of mTOR (FRB) and/or a FK506 binding protein (FKBP) or any binding pair of protein interaction domains are collectively a FKBP-binding domain of mTOR (FRB) and a FK506 binding protein (FKBP);
 - f. wherein the protein interaction domain(s) is a cyclophilin-Fas fusion protein (CyP-Fas) and/or a FK506 binding protein (FKBP) or any binding pair of protein interaction domains are collectively a cyclophilin-Fas fusion protein (CyP-Fas) and a FK506 binding protein (FKBP);
 - g. wherein the protein interaction domain(s) is a calcineurinA (CNA) and/or a FK506 binding protein (FKBP) or any binding pair of protein interaction domains are collectively a calcineurinA (CNA) and a FK506 binding protein (FKBP);
 - h. wherein the protein interaction domain(s) is a gibberellin insensitive (GIA) and/or a gibberellin insensitive dwarf1 (GID1) or any binding pair of protein interaction domains are collectively a gibberellin insensitive (GIA) and a gibberellin insensitive dwarf1 (GID1);
 - i. wherein the protein interaction domain(s) is a Snap-tag and/or a Halo tag, or any binding pair of protein interaction domains are collectively a Snap-tag and a Halo tag;
 - j. wherein the protein interaction domain(s) is a T14-3-3-cdeltaC and/or a C-Terminal peptides of PMA2 (CT52), or any binding pair of protein interaction domains are collectively a T14-3-3-cdeltaC and a C-Terminal peptides of PMA2 (CT52);
 - k. wherein the protein interaction domain(s) is a PYL and/or a ABI, or any binding pair of protein interaction domains are collectively a PYL and a ABI; and/or
 - l. wherein the protein interaction domain(s) is a nucleotide tag and/or a zinc finger domain, or any binding pair of protein interaction domains are collectively a nucleotide tag and a zinc finger domain.
17. The composition of paragraph 16, wherein the nucleotide tag is a DNA tag or dsDNA tag.

18. The composition of any of the preceding paragraphs, wherein the intracellular signaling domain is a signaling domain from a protein selected from the group consisting of:

TCRC; FcRy; FcRp; CD3y; CD35; CD3s; CD3C; CD22; CD79a; CD79b; CD66d; CARD11; CD2; CD7; CD27; CD28; CD30; CD40; CD54 (ICAM); CD83; CD134 (OX40); CD137 (4-1BB); CD150 (SLAMF1); CD152 (CTLA4); CD223 (LAG3); CD270 (HVEM); CD273 (PD-L2); CD274 (PD- L1); CD278 (ICOS); DAP10; LAT; KD2C SLP76; TRIM; ZAP70; and 41BB.

19. A cell comprising and/or expressing the composition of any of the preceding paragraphs.
20. The cell of paragraph 19, wherein the TCR recognition domain comprises a MHC allogenic to the cell.
21. The cell of paragraph 19, wherein the TCR recognition domain comprises a peptide allogenic to the cell.
22. The cell of any of paragraphs 19-21, wherein the cell is a dendritic cell, regulatory T cell, or effector T cell.
23. The cell of any of paragraphs 19-22, wherein the cell is engineered to express the polypeptide(s) of the composition.
24. The cell of any of paragraphs 19-22, wherein the cell is engineered to express the signaling polypeptide of the composition.
25. The cell of any of paragraphs 19-24, wherein the cell is further engineered to knockout the native MHCI/II.
26. A chimeric antigen receptor (CAR) comprising:
- an anti-CD127 and/or anti-CD45RO recognition domain;
 - an intracellular signaling domain.
27. A composition comprising:
- a first polypeptide comprising:
 - an anti-CD127 and/or anti-CD45RO recognition domain;
 - a first-type protein interaction domain; and
 - a second polypeptide comprising:
 - a second-type protein interaction domain; and
 - an intracellular signaling domain;
- wherein the first-type protein interaction domain and the second-type protein interaction domain bind specifically to each other.
28. A composition comprising:
- a first polypeptide comprising:
 - an anti-CD127 recognition domain;
 - a first-type protein interaction domain;

a second polypeptide comprising:

- c. an anti-CD45RO recognition domain;
- d. a fifth-type protein interaction domain; and

a third polypeptide comprising:

- e. a second-type and a fourth-type protein interaction domain; and
- f. an intracellular signaling domain;

wherein the first-type protein interaction domain and the second-type protein interaction domain bind specifically to each other; and

wherein the fourth-type protein interaction domain and the fifth-type protein interaction domain bind specifically to each other.

29. A cell comprising the CAR or composition of any of paragraphs 26-28.
30. A method of treating an autoimmune disease or treating or preventing transplant rejection or GVHD in a subject in need thereof, the method comprising administering to the subject a composition and/or cell of any of the preceding paragraphs.
31. The method of paragraph 30, wherein the TCR recognition domain comprises a MHC allogenic to the subject.
32. The method of paragraph 30, wherein the TCR recognition domain comprises a MHC autologous to the transplant cells.
33. The method of paragraph 30, wherein the TCR recognition domain comprises a peptide allogenic to the subject.
34. The method of paragraph 30, wherein the TCR recognition domain comprises a peptide autologous to the transplant cells.
35. The method of any of paragraphs 32 or 34, wherein the transplant is vascularized composite allotransplantation (VCA).
36. The method of any of paragraphs 35, wherein the autoimmune disease is type 1 diabetes, multiple sclerosis, rheumatoid arthritis, or scleroderma.

[00297] Some embodiments of the technology described herein can be defined according to any of the following numbered paragraphs:

1. A composition, comprising:
 - a. a TCR recognition domain; and
 - one or both of:
 - b. an intracellular signaling domain; and
 - c. a first-type biomolecular interaction domain.
2. A composition comprising:
 - a. a first polypeptide comprising at least a portion of a TCR recognition domain and a first-type biomolecular interaction domain; and

- b. a signaling polypeptide comprising a second-type biomolecular interaction domain and an intracellular signaling domain;

wherein the first-type and second-type biomolecular interaction domains bind specifically to each other.

3. A composition comprising:

- a. a first polypeptide comprising at least a portion of a TCR recognition domain and a first-type biomolecular interaction domain; and
- b. a recognition polypeptide comprising a second recognition domain and a third-type biomolecular interaction domain;

wherein the first-type and third-type biomolecular interaction domains bind specifically to each other.

4. A composition comprising:

- a. a first polypeptide comprising at least a portion of a TCR recognition domain and a first-type biomolecular interaction domain; and
- b. a signaling polypeptide comprising a second-type biomolecular interaction domain and an intracellular signaling domain; and
- c. a recognition polypeptide comprising a second recognition domain and a third-type biomolecular interaction domain;

wherein the second-type and third-type biomolecular interaction domains compete for binding to the first-type biomolecular interaction domain.

5. The composition of any of paragraphs 3-4, wherein the third-type biomolecular interaction domain and first-type biomolecular interaction domain have a higher affinity for each other than the second-type biomolecular interaction domain and first-type biomolecular interaction domain.

6. A composition comprising:

- a. a first polypeptide comprising at least a portion of a TCR recognition domain and a first-type biomolecular interaction domain;
- b. a signaling polypeptide comprising a second-type biomolecular interaction domain, a fourth-type biomolecular interaction domain, and an intracellular signaling domain; and
- c. a recognition polypeptide comprising a second recognition domain and a fifth-type biomolecular interaction domain;

wherein the first-type biomolecular interaction domain and the second-type biomolecular interaction domain bind specifically to each other; and

wherein the fourth-type biomolecular interaction domain and the fifth-type biomolecular interaction domain bind specifically to each other.

7. The composition of paragraph 6, wherein the fourth-type biomolecular interaction domain and fifth-type biomolecular interaction domain have a weaker affinity than the second-type biomolecular interaction domain and first-type protein interaction domain.
8. The composition of any of paragraphs 6-7, wherein the first polypeptide further comprises a sixth-type biomolecular interaction domain and the recognition polypeptide further comprises a seventh-type biomolecular interaction domain which bind specifically to each other.
9. The composition of any of paragraphs 2-8, wherein the first polypeptide comprises the entire TCR recognition domain.
10. The composition of any of paragraphs 2-8, wherein the TCR recognition domain comprises at least two separate polypeptide sequences, the first polypeptide comprises at least one of the separate polypeptide sequences of the TCR recognition domain, and the first polypeptide is bound to or complexed with a second or further polypeptide sequences of the TCR recognition domain to form a TCR recognition domain.
11. The composition of any of paragraphs 1-10, wherein the TCR recognition domain comprises a non-polypeptide component.
12. The composition of any of paragraphs 3-11, wherein the second recognition domain is specific for a target that is not recognized by the TCR recognition domain.
13. The composition of any of paragraphs 3-12, wherein the second recognition domain is specific for a target that is found on a healthy and/or non-target cell and not on a diseased and/or target cell.
14. The composition of any of the preceding paragraphs, wherein the TCR recognition domain comprises a MHC (Major Histocompatibility Complex); a MHC-peptide complex; featureless peptide MHC; or a MHC-peptide fusion.
15. The composition of paragraph 14, wherein the peptide is a human or non-human peptide.
16. The composition of any of paragraphs 14-15, wherein the peptide is a Minor Histocompatibility Antigen (MiHA).
17. The composition of any of paragraphs 14-16, wherein the MHC is a monomer, dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.
18. The composition of any of paragraphs 14-17, wherein the MHC-peptide complex is a monomer, dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.
19. The composition of any of paragraphs 14-17, wherein the MHC-peptide fusion is a monomer, dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.
20. The composition of any of paragraphs 14-17, wherein the MHC is a dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.
21. The composition of any of paragraphs 14-17, wherein the MHC-peptide complex is a dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.

22. The composition of any of paragraphs 14-17, wherein the MHC-peptide fusion is a dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.
23. The composition of any of paragraphs 14-22, wherein the MHC is a MHC class I or a MHC class II.
24. The composition of any of paragraphs 1-13, wherein the TCR recognition domain comprises a CD1 domain or a CD1 domain-ligand complex or fusion.
25. The composition of paragraph 24, wherein the CD1 is CD1d.
26. The composition of any of the preceding paragraphs, wherein the biomolecular interaction domains are found on an extracellular portion of the respective polypeptides.
27. The composition of any of the preceding paragraphs,
 - a. wherein the biomolecular interaction domain(s) is a leucine zipper, or any binding pair of biomolecular interaction domains are collectively a pair of leucine zippers;
 - b. wherein the biomolecular interaction domain(s) is a BZip (RR) and/or a AZip (EE), or any binding pair of biomolecular interaction domains are collectively a BZip (RR) and a AZip (EE);
 - c. wherein the biomolecular interaction domain(s) is a PSD95-Dlg1-zo-1 (PDZ) domain;
 - d. wherein the biomolecular interaction domain(s) is a streptavidin and/or a streptavidin binding biomolecular (SBP) or any binding pair of biomolecular interaction domains are collectively a streptavidin and a streptavidin binding biomolecular (SBP);
 - e. wherein the biomolecular interaction domain(s) is a FKBP-binding domain of mTOR (FRB) and/or a FK506 binding biomolecular (FKBP) or any binding pair of biomolecular interaction domains are collectively a FKBP-binding domain of mTOR (FRB) and a FK506 binding biomolecular (FKBP);
 - f. wherein the biomolecular interaction domain(s) is a cyclophilin-Fas fusion biomolecular (CyP-Fas) and/or a FK506 binding biomolecular (FKBP) or any binding pair of biomolecular interaction domains are collectively a cyclophilin-Fas fusion biomolecular (CyP-Fas) and a FK506 binding biomolecular (FKBP);
 - g. wherein the biomolecular interaction domain(s) is a calcineurin A (CNA) and/or a FK506 binding biomolecular (FKBP) or any binding pair of biomolecular interaction domains are collectively a calcineurin A (CNA) and a FK506 binding biomolecular (FKBP);
 - h. wherein the biomolecular interaction domain(s) is a gibberellin insensitive (GIA) and/or a gibberellin insensitive dwarf1 (GID1) or any binding pair of biomolecular interaction domains are collectively a gibberellin insensitive (GIA) and a gibberellin insensitive dwarf1 (GID1);

- i. wherein the biomolecular interaction domain(s) is a Snap-tag and/or a Halo tag, or any binding pair of biomolecular interaction domains are collectively a Snap-tag and a Halo tag;
 - j. wherein the biomolecular interaction domain(s) is a T14-3-3-cdeltaC and/or a C-Terminal peptides of PMA2 (CT52), or any binding pair of biomolecular interaction domains are collectively a T14-3-3-cdeltaC and a C-Terminal peptides of PMA2 (CT52);
 - k. wherein the biomolecular interaction domain(s) is a PYL and/or a ABI, or any binding pair of biomolecular interaction domains are collectively a PYL and a ABI;
 - l. wherein the biomolecular interaction domain(s) is a nucleotide tag and/or a zinc finger domain, or any binding pair of biomolecular interaction domains are collectively a nucleotide tag and a zinc finger domain;
 - m. wherein the biomolecular interaction domain(s) is a nucleotide tag, or any binding pair of biomolecular interaction domains are collectively a pair of nucleotide tags;
 - n. wherein the biomolecular interaction domain(s) is a Fluorescein isothiocyanate (FITC) and/or a FITC binding biomolecular or any binding pair of protein interaction domains are collectively a FITC and a FITC binding protein; and/or
 - o. wherein the protein interaction domain(s) is a (R)-Phycoerythrin (R-PE/ PE) and/or a R-PE/PE binding protein or any binding pair of protein interaction domains are collectively a (R)-Phycoerythrin (R-PE/ PE) and a R-PE/PE binding protein.
28. The composition of paragraph 27, wherein the nucleotide tag is a DNA tag or dsDNA tag.
29. The composition of any of the preceding paragraphs, wherein the intracellular signaling domain comprises or is a signaling domain from one or more proteins selected from the group consisting of:
- TCR ζ , FcR γ , FcR β , CD3 γ ; CD35; CD3 ζ ; CD3C; CD22; CD79a; CD79b; CD66d; CARD11; CD2; CD7; CD27; CD28; CD30; CD40; CD54 (ICAM); CD83; CD134 (OX40); CD137 (4-1BB); CD150 (SLAMF1); CD152 (CTLA4); CD223 (LAG3); CD270 (HVEM); CD273 (PD-L2); CD274 (PD- L1); CD278 (ICOS); DAP10; LAT; KD2C SLP76; TRIM; and ZAP70.
30. A cell comprising and/or expressing the composition of any of the preceding paragraphs.
31. A composition comprising a first polypeptide of any of the preceding paragraphs and a cell expressing or comprising the signaling polypeptide of any of the preceding paragraphs.
32. The cell or composition of any of paragraphs 30-31, wherein the TCR recognition domain comprises a MHC allogeneic, autologous, or xenogeneic to the cell.
33. The cell or composition of any of paragraphs 30-32, wherein the TCR recognition domain comprises a synthetic MHC.

34. The cell or composition of any of paragraphs 30-33, wherein the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is allogeneic, autologous, or xenogeneic to the cell.
35. The cell or composition of any of paragraphs 30-34, wherein the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is synthetic.
36. The cell or composition of any of paragraphs 30-35, wherein the cell is a NK cell, dendritic cell, regulatory T cell, or effector T cell.
37. The cell or composition of any of paragraphs 30-36, wherein the cell is engineered to express one of more of the polypeptide(s) of the composition.
38. The cell or composition of any of paragraphs 30-37, wherein the cell is engineered to express the signaling polypeptide of the composition.
39. The cell or composition of any of paragraphs 30-38, wherein the cell is further engineered to knockout or knockdown the native MHC I/II.
40. The cell or composition of any of paragraphs 30-39, wherein the cell is further engineered to knockdown the native MHC I/II expressed on the cell surface.
41. A chimeric antigen receptor (CAR) comprising:
 - a. an anti-CD127 and/or anti-CD45RO recognition domain;
 - b. an intracellular signaling domain.
42. A composition comprising:
 - a first polypeptide comprising:
 - a. an anti-CD127 and/or anti-CD45RO recognition domain;
 - b. a first-type protein interaction domain; and
 - a second polypeptide comprising:
 - c. a second-type protein interaction domain; and
 - d. an intracellular signaling domain;

wherein the first-type protein interaction domain and the second-type protein interaction domain bind specifically to each other.
43. A composition comprising:
 - a first polypeptide comprising:
 - a. an anti-CD127 recognition domain;
 - b. a first-type protein interaction domain;
 - a second polypeptide comprising:
 - c. an anti-CD45RO recognition domain;
 - d. a fifth-type protein interaction domain; and
 - a third polypeptide comprising:
 - e. a second-type and a fourth-type protein interaction domain; and

- f. an intracellular signaling domain;
wherein the first-type protein interaction domain and the second-type protein interaction domain bind specifically to each other; and
wherein the fourth-type protein interaction domain and the fifth-type protein interaction domain bind specifically to each other.
44. A cell comprising the CAR or composition of any of paragraphs 41-43.
45. A method of treating or preventing an autoimmune disease or condition; T cell mediated inflammation or immune response; malignant T cell condition; transplant rejection; or GvHD in a subject in need thereof, the method comprising administering to the subject a composition and/or cell of any of the preceding paragraphs.
46. The method of paragraph 45, wherein the TCR recognition domain comprises a MHC allogeneic to the subject.
47. The method of paragraph 45, wherein the TCR recognition domain comprises a MHC autologous to the transplant cells.
48. The method of paragraph 45, wherein the TCR recognition domain comprises a MHC xenogeneic to the transplant cells.
49. The method of paragraph 45, wherein the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is allogeneic to the subject.
50. The method of paragraph 45, wherein the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is autologous to the transplant cells.
51. The method of paragraph 45, wherein the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is autologous to the transplant cells.
52. The method of any of paragraphs 45-51, wherein the MHC and/or the peptide is synthetic.
53. The method of any of paragraphs 45-52, wherein the transplant is any human or non-human cell, tissue, or organ.
54. The method of any of paragraphs 45-53, wherein the transplant is an allogeneic hematopoietic stem cell or solid organ transplantation.
55. The method of any of paragraphs 45-52, wherein the malignant T cell condition is T cell acute lymphoblastic leukemia or T cell lymphoblastic lymphoma.
56. The method of any of paragraphs 45-52, wherein the autoimmune disease is type 1 diabetes, vitiligo, multiple sclerosis, alopecia, celiac disease, pemphigus, rheumatoid arthritis, or scleroderma.
57. The method of any of paragraphs 45-52, wherein the autoimmune disease is thyroiditis, type 1 diabetes mellitus, Hashimoto's thyroiditis, Graves' disease, celiac disease, multiple sclerosis, Guillain-Barre syndrome, Addison's disease, and Raynaud's phenomenon, Goodpasture's disease, arthritis (rheumatoid arthritis such as acute arthritis, chronic rheumatoid arthritis,

gout or gouty arthritis, acute gouty arthritis, acute immunological arthritis, chronic inflammatory arthritis, degenerative arthritis, type II collagen-induced arthritis, infectious arthritis, Lyme arthritis (e.g., post treatment Lyme disease syndrome), proliferative arthritis, psoriatic arthritis, Still's disease, vertebral arthritis, and juvenile-onset rheumatoid arthritis, arthritis chronica progrediente, arthritis deformans, polyarthritis chronica primaria, reactive arthritis, and ankylosing spondylitis), palindromic arthritis, inflammatory hyperproliferative skin diseases, psoriasis such as plaque psoriasis, guttate psoriasis, pustular psoriasis, and psoriasis of the nails, atopy including atopic diseases such as hay fever and Job's syndrome, dermatitis including contact dermatitis, chronic contact dermatitis, exfoliative dermatitis, allergic dermatitis, allergic contact dermatitis, dermatitis herpetiformis, nummular dermatitis, seborrheic dermatitis, non-specific dermatitis, primary irritant contact dermatitis, and atopic dermatitis, x-linked hyper IgM syndrome, allergic intraocular inflammatory diseases, urticaria such as chronic allergic urticaria and chronic idiopathic urticaria, including chronic autoimmune urticaria, myositis, polymyositis/dermatomyositis, juvenile dermatomyositis, toxic epidermal necrolysis, scleroderma (including systemic scleroderma), sclerosis such as systemic sclerosis, multiple sclerosis (MS) such as spino-optical MS, primary progressive MS (PPMS), and relapsing remitting MS (RRMS), progressive systemic sclerosis, atherosclerosis, arteriosclerosis, sclerosis disseminata, ataxic sclerosis, neuromyelitis optica (NMO), inflammatory bowel disease (IBD) (for example, Crohn's disease, autoimmune-mediated gastrointestinal diseases, colitis such as ulcerative colitis, colitis ulcerosa, microscopic colitis, collagenous colitis, colitis polyposa, necrotizing enterocolitis, and transmural colitis, and autoimmune inflammatory bowel disease), bowel inflammation, pyoderma gangrenosum, erythema nodosum, primary sclerosing cholangitis, respiratory distress syndrome, including adult or acute respiratory distress syndrome (ARDS), meningitis, inflammation of all or part of the uvea, iritis, choroiditis, an autoimmune hematological disorder, rheumatoid spondylitis, rheumatoid synovitis, hereditary angioedema, cranial nerve damage as in meningitis, herpes gestationis, pemphigoid gestationis, pruritis scroti, autoimmune premature ovarian failure, sudden hearing loss due to an autoimmune condition, IgE-mediated diseases such as anaphylaxis and allergic and atopic rhinitis, encephalitis such as Rasmussen's encephalitis and limbic and/or brainstem encephalitis, uveitis, such as anterior uveitis, acute anterior uveitis, granulomatous uveitis, nongranulomatous uveitis, phacoantigenic uveitis, posterior uveitis, or autoimmune uveitis, glomerulonephritis (GN) with and without nephrotic syndrome such as chronic or acute glomerulonephritis such as primary GN, immune-mediated GN, membranous GN (membranous nephropathy), idiopathic membranous GN or idiopathic membranous nephropathy, membrano- or membranous proliferative GN (MPGN), including Type I and Type II, and rapidly progressive GN, proliferative nephritis, autoimmune polyglandular

endocrine failure, balanitis including balanitis circumscripta plasmacellularis, balanoposthitis, erythema annulare centrifugum, erythema dyschromicum perstans, erythema multiform, granuloma annulare, lichen nitidus, lichen sclerosus et atrophicus, lichen simplex chronicus, lichen spinulosus, lichen planus, lamellar ichthyosis, epidermolytic hyperkeratosis, premalignant keratosis, pyoderma gangrenosum, allergic conditions and responses, allergic reaction, eczema including allergic or atopic eczema, asteatotic eczema, dyshidrotic eczema, and vesicular palmoplantar eczema, asthma such as asthma bronchiale, bronchial asthma, and auto-immune asthma, conditions involving infiltration of T cells and chronic inflammatory responses, immune reactions against foreign antigens such as fetal A-B-O blood groups during pregnancy, chronic pulmonary inflammatory disease, autoimmune myocarditis, leukocyte adhesion deficiency, lupus, including lupus nephritis, lupus cerebritis, pediatric lupus, non-renal lupus, extra-renal lupus, discoid lupus and discoid lupus erythematosus, alopecia lupus, systemic lupus erythematosus (SLE) such as cutaneous SLE or subacute cutaneous SLE, neonatal lupus syndrome (NLE), and lupus erythematosus disseminatus, juvenile onset (Type I) diabetes mellitus, including pediatric insulin-dependent diabetes mellitus (IDDM), adult onset diabetes mellitus (Type II diabetes), autoimmune diabetes, idiopathic diabetes insipidus, diabetic retinopathy, diabetic nephropathy, diabetic large-artery disorder, immune responses associated with acute and delayed hypersensitivity mediated by cytokines and T-lymphocytes, sarcoidosis, granulomatosis including lymphomatoid granulomatosis, Wegener's granulomatosis, agranulocytosis, vasculitides, including vasculitis, large-vessel vasculitis (including polymyalgia rheumatica and giant-cell (Takayasu's) arteritis), medium-vessel vasculitis (including Kawasaki's disease and polyarteritis nodosa/periarthritis nodosa), microscopic polyarteritis, immunovascularitis, CNS vasculitis, cutaneous vasculitis, hypersensitivity vasculitis, necrotizing vasculitis such as systemic necrotizing vasculitis, and ANCA-associated vasculitis, such as Churg-Strauss vasculitis or syndrome (CSS) and ANCA-associated small-vessel vasculitis, temporal arteritis, autoimmune aplastic anemia, Coombs positive anemia, Diamond Blackfan anemia, hemolytic anemia or immune hemolytic anemia including autoimmune hemolytic anemia (AIHA), pernicious anemia (anemia perniosa), Addison's disease, pure red cell anemia or aplasia (PRCA), Factor VIII deficiency, hemophilia A, autoimmune neutropenia, pancytopenia, leukopenia, diseases involving leukocyte diapedesis, CNS inflammatory disorders, multiple organ injury syndrome such as those secondary to septicemia, trauma or hemorrhage, antigen-antibody complex-mediated diseases, anti-glomerular basement membrane disease, anti-phospholipid antibody syndrome, allergic neuritis, Behcet's disease/syndrome, Castleman's syndrome, Goodpasture's syndrome, Reynaud's syndrome, Sjogren's syndrome, Stevens-Johnson syndrome, pemphigoid such as pemphigoid bullous and skin pemphigoid, pemphigus

(including pemphigus vulgaris, pemphigus foliaceus, pemphigus mucus-membrane pemphigoid, and pemphigus erythematosus), autoimmune polyendocrinopathies, Reiter's disease or syndrome, an immune complex disorder such as immune complex nephritis, antibody-mediated nephritis, polyneuropathies, chronic neuropathy such as IgM polyneuropathies or IgM-mediated neuropathy, and autoimmune or immune-mediated thrombocytopenia such as idiopathic thrombocytopenic purpura (ITP) including chronic or acute ITP, scleritis such as idiopathic cerato-scleritis, episcleritis, autoimmune disease of the testis and ovary including autoimmune orchitis and oophoritis, primary hypothyroidism, hypoparathyroidism, autoimmune endocrine diseases including thyroiditis such as autoimmune thyroiditis, Hashimoto's disease, chronic thyroiditis (Hashimoto's thyroiditis), or subacute thyroiditis, idiopathic hypothyroidism, Grave's disease, polyglandular syndromes such as autoimmune polyglandular syndromes (or polyglandular endocrinopathy syndromes), paraneoplastic syndromes, including neurologic paraneoplastic syndromes such as Lambert-Eaton myasthenic syndrome or Eaton-Lambert syndrome, stiff-man or stiff-person syndrome, encephalomyelitis such as allergic encephalomyelitis or encephalomyelitis allergica and experimental allergic encephalomyelitis (EAE), myasthenia gravis such as thymoma-associated myasthenia gravis, cerebellar degeneration, neuromyotonia, opsoclonus or opsoclonus myoclonus syndrome (OMS), and sensory neuropathy, multifocal motor neuropathy, Sheehan's syndrome, autoimmune hepatitis, lupoid hepatitis, giant-cell hepatitis, autoimmune chronic active hepatitis, lymphoid interstitial pneumonitis (LIP), bronchiolitis obliterans (non-transplant) vs NSIP, Guillain-Barre syndrome, Berger's disease (IgA nephropathy), idiopathic IgA nephropathy, linear IgA dermatosis, acute febrile neutrophilic dermatosis, subcorneal pustular dermatosis, transient acantholytic dermatosis, cirrhosis such as primary biliary cirrhosis and pneumonocirrhosis, autoimmune enteropathy syndrome, Celiac or Coeliac disease, celiac sprue (gluten enteropathy), refractory sprue, idiopathic sprue, cryoglobulinemia, amyotrophic lateral sclerosis (ALS; Lou Gehrig's disease), coronary artery disease, autoimmune ear disease such as autoimmune inner ear disease (AIED), autoimmune hearing loss, polychondritis such as refractory or relapsed or relapsing polychondritis, pulmonary alveolar proteinosis, Cogan's syndrome/nonsyphilitic interstitial keratitis, Bell's palsy, Sweet's disease/syndrome, rosacea autoimmune, zoster-associated pain, amyloidosis, a non-cancerous lymphocytosis, a primary lymphocytosis, which includes monoclonal B cell lymphocytosis (e.g., benign monoclonal gammopathy and monoclonal gammopathy of undetermined significance, MGUS), peripheral neuropathy, paraneoplastic syndrome, channelopathies including channelopathies of the CNS, autism, inflammatory myopathy, focal or segmental or focal segmental glomerulosclerosis (FSGS), endocrine ophthalmopathy, uveoretinitis, chorioretinitis, autoimmune hepatological disorder, fibromyalgia, multiple

endocrine failure, Schmidt's syndrome, adrenalitis, gastric atrophy, presenile dementia, demyelinating diseases such as autoimmune demyelinating diseases and chronic inflammatory demyelinating polyneuropathy, Dressler's syndrome, alopecia areata, alopecia totalis, CREST syndrome (calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia), male and female autoimmune infertility, e.g., due to anti-spermatozoan antibodies, mixed connective tissue disease, Chagas' disease, rheumatic fever, recurrent abortion, farmer's lung, erythema multiforme, post-cardiotomy syndrome, Cushing's syndrome, bird-fancier's lung, allergic granulomatous angiitis, benign lymphocytic angiitis, Alport's syndrome, alveolitis such as allergic alveolitis and fibrosing alveolitis, interstitial lung disease, transfusion reaction, Sampter's syndrome, Caplan's syndrome, endocarditis, endomyocardial fibrosis, diffuse interstitial pulmonary fibrosis, interstitial lung fibrosis, pulmonary fibrosis, idiopathic pulmonary fibrosis, cystic fibrosis, endophthalmitis, erythema elevatum et diutinum, erythroblastosis fetalis, eosinophilic fasciitis, Shulman's syndrome, Felty's syndrome, cyclitis such as chronic cyclitis, heterochronic cyclitis, iridocyclitis (acute or chronic), or Fuch's cyclitis, Henoch-Schonlein purpura, SCID, sepsis, endotoxemia, post-vaccination syndromes, Evan's syndrome, autoimmune gonadal failure, Sydenham's chorea, post-streptococcal nephritis, thromboangitis obliterans, thyrotoxicosis, tabes dorsalis, chorioiditis, giant-cell polymyalgia, chronic hypersensitivity pneumonitis, keratoconjunctivitis sicca, idiopathic nephritic syndrome, minimal change nephropathy, benign familial and ischemia-reperfusion injury, transplant organ reperfusion, retinal autoimmunity, aphthae, aphthous stomatitis, arteriosclerotic disorders, aspermiogenesis, autoimmune hemolysis, Boeck's disease, enteritis allergica, erythema nodosum leprosum, idiopathic facial paralysis, chronic fatigue syndrome, febris rheumatica, Hamman-Rich's disease, sensorineural hearing loss, ileitis regionalis, leucopenia, transverse myelitis, primary idiopathic myxedema, ophthalmia sympathica, polyradiculitis acuta, pyoderma gangrenosum, acquired splenic atrophy, vitiligo, toxic-shock syndrome, conditions involving infiltration of T cells, leukocyte-adhesion deficiency, immune responses associated with acute and delayed hypersensitivity mediated by cytokines and T-lymphocytes, diseases involving leukocyte diapedesis, multiple organ injury syndrome, antigen-antibody complex-mediated diseases, antglomerular basement membrane disease, allergic neuritis, autoimmune polyendocrinopathies, oophoritis, primary myxedema, autoimmune atrophic gastritis, rheumatic diseases, mixed connective tissue disease, nephrotic syndrome, insulinitis, polyendocrine failure, autoimmune polyglandular syndrome type I, adult-onset idiopathic hypoparathyroidism (AOIH), myocarditis, nephrotic syndrome, primary sclerosing cholangitis, acute or chronic sinusitis, ethmoid, frontal, maxillary, or sphenoid sinusitis, an eosinophil-related disorder such as eosinophilia, pulmonary infiltration eosinophilia,

eosinophilia-myalgia syndrome, Löffler's syndrome, chronic eosinophilic pneumonia, tropical pulmonary eosinophilia, granulomas containing eosinophils, seronegative spondyloarthritides, polyendocrine autoimmune disease, sclerosing cholangitis, sclera, episclera, Bruton's syndrome, transient hypogammaglobulinemia of infancy, Wiskott-Aldrich syndrome, ataxia telangiectasia syndrome, angiectasis, autoimmune disorders associated with collagen disease, rheumatism, allergic hypersensitivity disorders, glomerulonephritides, reperfusion injury, ischemic re-perfusion disorder, lymphomatous tracheobronchitis, inflammatory dermatoses, dermatoses with acute inflammatory components, and autoimmune uveoretinitis (AUR).

58. The method of any of paragraphs 45-52, wherein the T cell mediated immune response is an anti-drug specific response to a biologic, cell therapy, and/or gene therapy.
59. The method of paragraph 58, wherein the biologic, cell-therapy, or gene therapy is an adeno-associated virus (AAV) gene therapy, a genome editing agent, or enzyme replacement therapy.
60. The method of any of paragraphs 45-52, wherein the disease is type 1 diabetes and the TCR recognition domain comprises sequences with at least 80% or at least 95% sequence identity to: one or more of SEQ ID NOs: 8-17; HLA-A*0201 and at least one of SEQ ID NOs: 2013-2016 and 2031-2033; or HLA-A*02:01 and at least one of SEQ ID NOs: 20128-2129.
61. The method of any of paragraphs 45-52, wherein the disease is vitiligo and the TCR recognition domain comprises sequences with at least 80% or at least 95% sequence identity to: SEQ ID NO: 18, 19, and one of 20-22; or comprises HLA-A*0201 and SEQ ID NO: 2018; or HLA-A*0301 and SEQ ID NO: 2019; or comprises HLA-A*2402 and SEQ ID NO: 2020; or HLA-A*0101 and SEQ ID NO: 2021.
62. The method of any of paragraphs 45-52, wherein the method is a method of treating and/or preventing GvHD and the TCR recognition domain comprises sequences with at least 80% or at least 95% sequence identity to: HLA-A*0101 and at least one of SEQ ID NOs: 2034-2037; or HLA-B*0702 and SEQ ID NO: 2038; or HLA-B*0801 and SEQ ID NO: 2039.
63. The method of any of paragraphs 45-52, wherein the disease is type 1 diabetes and the TCR recognition domain comprises one or more of SEQ ID NOs: 8-17; comprises HLA-A*0201 and at least one of SEQ ID NOs: 2013-2016 and 2031-2033; or comprises HLA-A*02:01 and at least one of SEQ ID NOs: 20128-2129.
64. The method of any of paragraphs 45-52, wherein the disease is vitiligo and the TCR recognition domain comprises SEQ ID NO: 18, 19, and one of 20-22; or comprises HLA-A*0201 and SEQ ID NO: 2018; or comprises HLA-A*0301 and SEQ ID NO: 2019; or comprises HLA-A*2402 and SEQ ID NO: 2020; or comprises HLA-A*0101 and SEQ ID NO: 2021.
65. The method of any of paragraphs 45-52, wherein the method is a method of treating and/or preventing GvHD and the TCR recognition domain comprises HLA-A*0101 and at least one of SEQ ID NOs:

2034-2037; or comprises HLA-B*0702 and SEQ ID NO: 2038; or comprises HLA-B*0801 and SEQ ID NO: 2039.

[00298] Some embodiments of the technology described herein can be defined according to any of the following numbered paragraphs:

1. A composition, comprising:
 - a. a TCR recognition domain; and
 - one or both of:
 - b. an intracellular signaling domain; and
 - c. a biomolecular interaction domain.
2. The composition of paragraph 1, comprising a TCR recognition domain and a biomolecular interaction domain.
3. The composition of paragraph 1, comprising a TCR recognition domain and an intracellular signaling domain.
4. The composition of paragraph 1, wherein the biomolecular interaction domain of c) is a first-type biomolecular interaction domain and the composition further comprises a signaling polypeptide comprising a second-type biomolecular interaction domain and an intracellular signaling domain;

wherein the first-type and second-type biomolecular interaction domains bind specifically to each other.
5. The composition of paragraph 1, wherein the TCR recognition domain comprises a MHC (Major Histocompatibility Complex); a MHC-peptide complex; a featureless peptide MHC; or a MHC-peptide fusion.
6. The composition of paragraph 1, wherein the TCR recognition domain comprises a CD1 domain or a CD1 domain-ligand complex or fusion.
7. The composition of paragraph 6, wherein the CD1 is CD1d.
8. The composition of paragraph 1, wherein the TCR recognition domain is a monomer, dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.
9. The composition of paragraph 1,
 - a. wherein the biomolecular interaction domain(s) is a leucine zipper, or any binding pair of biomolecular interaction domains are collectively a pair of leucine zippers;
 - b. wherein the biomolecular interaction domain(s) is a BZip (RR) and/or a AZip (EE), or any binding pair of biomolecular interaction domains are collectively a BZip (RR) and a AZip (EE);
 - c. wherein the biomolecular interaction domain(s) is a PSD95-Dlg1-zo-1 (PDZ) domain;

- d. wherein the biomolecular interaction domain(s) is a streptavidin and/or a streptavidin binding biomolecular (SBP) or any binding pair of biomolecular interaction domains are collectively a streptavidin and a streptavidin binding biomolecular (SBP);
- e. wherein the biomolecular interaction domain(s) is a FKBP-binding domain of mTOR (FRB) and/or a FK506 binding biomolecular (FKBP) or any binding pair of biomolecular interaction domains are collectively a FKBP-binding domain of mTOR (FRB) and a FK506 binding biomolecular (FKBP);
- f. wherein the biomolecular interaction domain(s) is a cyclophilin-Fas fusion biomolecular (CyP-Fas) and/or a FK506 binding biomolecular (FKBP) or any binding pair of biomolecular interaction domains are collectively a cyclophilin-Fas fusion biomolecular (CyP-Fas) and a FK506 binding biomolecular (FKBP);
- g. wherein the biomolecular interaction domain(s) is a calcineurin A (CNA) and/or a FK506 binding biomolecular (FKBP) or any binding pair of biomolecular interaction domains are collectively a calcineurin A (CNA) and a FK506 binding biomolecular (FKBP);
- h. wherein the biomolecular interaction domain(s) is a gibberellin insensitive (GIA) and/or a gibberellin insensitive dwarf1 (GID1) or any binding pair of biomolecular interaction domains are collectively a gibberellin insensitive (GIA) and a gibberellin insensitive dwarf1 (GID1);
- i. wherein the biomolecular interaction domain(s) is a Snap-tag and/or a Halo tag, or any binding pair of biomolecular interaction domains are collectively a Snap-tag and a Halo tag;
- j. wherein the biomolecular interaction domain(s) is a T14-3-3-cdeltaC and/or a C-Terminal peptides of PMA2 (CT52), or any binding pair of biomolecular interaction domains are collectively a T14-3-3-cdeltaC and a C-Terminal peptides of PMA2 (CT52);
- k. wherein the biomolecular interaction domain(s) is a PYL and/or a ABI, or any binding pair of biomolecular interaction domains are collectively a PYL and a ABI;
- l. wherein the biomolecular interaction domain(s) is a nucleotide tag and/or a zinc finger domain, or any binding pair of biomolecular interaction domains are collectively a nucleotide tag and a zinc finger domain;
- m. wherein the biomolecular interaction domain(s) is a nucleotide tag, or any binding pair of biomolecular interaction domains are collectively a pair of nucleotide tags;
- n. wherein the biomolecular interaction domain(s) is a Fluorescein isothiocyanate (FITC) and/or a FITC binding biomolecular or any binding pair of protein interaction domains are collectively a FITC and a FITC binding protein; and/or

- o. wherein the protein interaction domain(s) is a (R)-Phycoerythrin (R-PE/ PE) and/or a R-PE/PE binding protein or any binding pair of protein interaction domains are collectively a (R)-Phycoerythrin (R-PE/ PE) and a R-PE/PE binding protein.
- 10. The composition of paragraph 9, wherein the nucleotide tag is a DNA tag or dsDNA tag.
- 11. The composition of paragraph 2, further comprising a cell expressing or comprising the signaling polypeptide.
- 12. The composition of paragraph 11, wherein the TCR recognition domain is allogeneic, autologous, or xenogenic to the cell.
- 13. The composition of paragraph 11, wherein the TCR recognition domain is synthetic.
- 14. The composition of paragraph 11, wherein the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is allogeneic, autologous, or xenogenic to the cell.
- 15. The composition of paragraph 11, wherein the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is synthetic.
- 16. The composition of paragraph 11, wherein the cell is a NK cell, dendritic cell, regulatory T cell, or effector T cell.
- 17. The composition of paragraph 11, wherein the cell is further engineered to knockdown the native MHC I/II expressed on the cell surface.
- 18. A method of treating or preventing an autoimmune disease or condition; T cell mediated inflammation or immune response; malignant T cell condition; transplant rejection; or GvHD in a subject in need thereof, the method comprising administering to the subject a composition of paragraph 1.
- 19. The method of paragraph 18, wherein the transplant is an allogeneic hematopoietic stem cell or solid organ transplantation.
- 20. The method of paragraph 18, wherein the malignant T cell condition is T cell acute lymphoblastic leukemia or T cell lymphoblastic lymphoma.
- 21. The method of paragraph 18, wherein the autoimmune disease is type 1 diabetes, vitiligo, multiple sclerosis, alopecia, celiac disease, pemphigus, rheumatoid arthritis, or scleroderma.
- 22. The method of paragraph 18, wherein the T cell mediated immune response is an anti-drug specific response to a biologic, cell therapy, and/or gene therapy.
- 23. The method of paragraph 22, wherein the biologic, cell-therapy, or gene therapy is an adeno-associated virus (AAV) gene therapy, a genome editing agent, or enzyme replacement therapy.
- 24. The method of paragraph 18, wherein the disease is type 1 diabetes and the TCR recognition domain comprises sequences with at least 95% sequence identity to: one or more of SEQ ID NOs: 8-17; HLA-A*0201 and at least one of SEQ ID NOs: 2013-2016 and 2031-2033; or HLA-A*02:01 and at least one of SEQ ID NOs: 20128-2129.

25. The method of paragraph 18, wherein the disease is vitiligo and the TCR recognition domain comprises sequences with at least 95% sequence identity to: SEQ ID NO: 18, 19, and one of 20-22; or HLA-A*0201 and SEQ ID NO: 2018; or HLA-A*0301 and SEQ ID NO: 2019; or comprises HLA-A*2402 and SEQ ID NO: 2020; or HLA-A*0101 and SEQ ID NO: 2021.
26. The method of paragraph 18, wherein the method is a method of treating and/or preventing GvHD and the TCR recognition domain comprises sequences with at least 95% sequence identity to: HLA-A*0101 and at least one of SEQ ID NOs: 2034-2037; or HLA-B*0702 and SEQ ID NO: 2038; or HLA-B*0801 and SEQ ID NO: 2039.

EXAMPLES

Example 1

[00299] Auto- and allo-reactive T cells attacking patient's or donor's cells or organs is the major cause of autoimmunity and transplant rejection. Current treatments involve stringent immunosuppressant therapy, which can lead to severe side effects. Specifically, depletion of auto and alloreactive T cells prevents autoimmunity and transplant rejection without immunosuppressant modalities. T cells engineered with a Chimeric antigen ligand (CAL) can redirect their specificity toward the pathologic T cells. T cells engineered with a Chimeric antigen receptor (CAR) can redirect their specificity and have already been approved to treat some types of B cell malignancies. Currently, engineered regulatory T cell, which can inhibit immune reactions in an antigen-dependent manner, is under investigation to expand the application of CAR T cells therapies such as autoimmune disease and transplant rejection.

[00300] To specifically deplete disease causing T cells, described herein is a CAL and/or CAR that can recognize and bind to the specific T cell receptor on the disease causing T cells. This CAL and/or CAR is a composed of a peptide-HLA (e.g., a monomer or multimer or oligomer thereof) as the recognition domain fused to signaling domains from T cell receptors. A split version of the system can also be generated where the system is composed of two pieces. One piece is an universal CAR with T cell signaling domains as the intracellular portion and a biomolecular interaction domain as the extracellular domain. The second piece is an adaptor molecule, e.g, a CAL composed of a peptide-HLA oligomers (or monomer or multimer) fused to the cognate biomolecular interaction domain.

[00301] Most CAR T cells therapies for autoimmune disease and transplant rejection uses regulatory T cells and are designed to target the organ to provide organ-specific, e.g., local immunosuppression. In contrast, the present, e.g., CAL, design is targeting the source immune cells inside that patient that are causing the disease. Accordingly, provided herein is a cell-based therapy for autoimmune diseases and transplant rejection.

Example 2

[00302] Dendritic cells (DCs) are the interconnection between innate and adaptive immune system and are able to induce protective immune responses following stimulation by a variety of stimuli. Based on their phenotype and function, DCs can be divided into conventional DCs and plasmacytoid DCs (pDCs). Many studies have shown that DCs are essential mediators of pro-inflammatory or anti-inflammatory (tolerogenic) responses. The subsets of DCs that suppress immune responses are generally known as tolerogenic DCs because of their functions in inducing T cell apoptosis, anergy and regulatory T cells (Tregs).

[00303] A tolerogenic state in DCs (tol-DC) can be induced using several pharmacological agents, such as cyclosporine A, rapamycin, dexamethasone, vitamin A, vitamin D or other cytokines and growth factors. Recently, the insertion of exogenous DNA to enhance tol-DC function has been investigated as a therapeutic possibility for treating autoimmune diseases. 'Killer' DCs, obtained by transfection with DNA encoding FasL or TNF-related apoptosis-inducing ligand, efficiently induce T-cell apoptosis and prevent the rejection of heart grafts in animal models.

[00304] However, the effect of these DCs are not specific. Overexpression of inhibitory molecules, including IL-10, TGF- β , CTLA-4 and SOCS1, enable tol-DCs to more efficiently induce Tregs which might cause systemic immune suppression.

[00305] Alloreactive immune cells can be suppressed in a specific manner using the methods described herein, e.g., by using a universal UniCAR DC system that presents the donors' pMHC tetra / dextramers (e.g., the CAL) and binds to a genetically engineered DC (K/O MHC I / II but presents donor peptide on the tetra / dextramer), . In some embodiments, the pMHC can be provided as a monomer, oligomer, or multimer.

[00306] Any form of the recipient pMHC (e.g., monomer / oligomer / tetramer / dextramer)+ targeting module (e.g, the CAL) can be used with CAR-DC for the suppression of allo-reactive T-cells in the recipients. A library of identified different alloantigens + correlated or associated with MHC monomers/ oligomers / tetramers / dextramers (e.g. HLA-A2+insulin peptide) and attached to the targeting molecules of the CAL and/or CAR system can be produced. Such a library can be commercially available to be combined with DCs for targeting and destroying the auto reactive T-cells.

[00307] Cells can be produced that are genetically modified with the specific gene deletion of some genes (IL-12 and NF- κ B, MHC I and MHC II [The targeting module will provide these cells with the donor's MHCs]) and insertion of some other genes (IL-10, TGF- β , CTLA-4 and SOCS1). These cells can be commercially ready to use and could be combined with the specific donor, pMHC and targeting module chosen from the aforementioned library.

Example 3

[00308] Alloreactive memory T cells have been shown to have the key role in the activation of the recipient immune system and rejection of the allograft. They are known to be the main barrier for tolerance induction through mixed chimerism and other strategies. These cells are very resilient and not responsive to the preconditioning protocols and they recover from irradiation and T cell depletion rapidly. By using CAR-T-effector (Conventional, Supra, Universal CAL or CAR) and attaching any form of the donors' pMHC (monomer, oligomer, tetramer or dextramer) (e.g., a CAL) to the system, the resulting compositions of the invention can be used to decrease or eliminate the need for improved immunosuppressant therapy in the context of transplantation. The engraftment of hematopoietic stem cells can achieve durable mixed chimerism with minimal or no need for toxic preconditioning protocols.

[00309] If donor pMHC (monomer/oligomer/tetramer/dextramer; e.g., a CAL) is used in combination with CAR T effector for the abrogation of the allo/xeno reactive T-cells in the recipient, the need for immunosuppressants would be decreased or eliminated. A library of different Donor MHC monomers/oligomers/tetramers attached to the targeting molecules (e.g., the CALs) of the SUPRA / UNI / universal CAL and/or CAR system can be produced to be commercially available. A shelf ready library of the most prevalent MHC (+ immunologic wildtype or synthetic peptide molecules that could be used to be combined with the MHC) monomers/oligomers/tetramers could be commercially produced and offered to be used with this system.

Example 4

[00310] The involvement of the adaptive immune system in auto-immune diseases has been extensively characterized. T cells are critical contributors to autoimmune diseases. Conventional T (Teff or T helper) cell subsets that play a role in B cell activation and differentiation produce various inflammatory cytokines and destroy target cells with direct cytotoxicity. CAR-T cells have been used to destroy autoimmune B and T cells in a fashion similar to the way in which CD19CAR T cells target and destroy leukemia cells. Targeting autoreactive memory T and B-cells has shown some results. As these methods are nonspecific, they induce some extent of generalized immune suppression and they are not completely effective. The correlation of particular peptide+MHC molecules with certain autoimmune conditions have been expansively studied. By using the available human/animal model pMHC monomer/oligomer/tetra/dextramers and combining them with the SUPRA / UNI / universal CAL and/or CAR technology, we are able to target the autoreactive immune cells in a highly specific manner.

[00311] If any form of auto-antigen on pMHC (monomer/oligomer/tetramer/dextramer) (e.g., the CAL) is used in combination with CAR T effector / Treg to abrogate autoreactive T-cells in the recipient, the need for immunomodulatory drugs would be decreased or eliminated. A library of

identified autoantigens and correlated MHC monomer/oligomer/tetra/dextramers (e.g. HLA-A2+insulin peptide) attached to the targeting molecules of the SUPRA / UNI / universal CAL and/or CAR system can be produced to be commercially available to be combined with ideal SUPRA or universal CAR T-reg / T effector for targeting and destroying the autoreactive T-cells.

Example 5

[00312] Although new advances have increased survival after allogeneic hematopoietic stem cell transplantation (HCT), chronic graft-versus-host disease (GvHD) is still the leading cause of late morbidity and mortality after transplant. Current treatment choices are limited in efficacy specifically in steroid-refractory disease, and there is no robust data to help with management decisions.

[00313] Adoptive T cell therapy (ACT) refers to the therapeutic use of T cells. T cells genetically engineered to express chimeric antigen receptors (CAR) constitute the most clinically advanced form of ACT approved to date for the treatment of CD19-positive leukemias and lymphomas and have produced remarkable results in the clinic. The technology described herein permits the opportunity to target diseased cells with specific antigens or receptors very accurately. In the context of GvHD, universal UNICAR T cells can be designed to find the recipient reactive T cell in the donor T cell populations. If the pMHC of the recipients is fused to a Target Module that binds to the UniCAR (e.g. to form a CAL), this system can recognize the TCR repertoires in the donor T/B cell population that can bind to those MHCs. If the recipients' pMHCs are fused to a target module that binds to the universal CAR, this system can recognize and bind to the T cell repertoires in the donor T cell population that can bind to those MHCs.

[00314] If the recipient pMHC (monomer / oligomer / tetramer / dextramer) (e.g., a CAL) is used in combination with CAR T effector / Treg for the abrogation of the recipient reactive T-cells in the donor HSC, these reactive T-cells will be depleted and GvHD would not happen. A library of identified different antigens + correlated human / animal models MHC tetramers / dextramers (e.g. HLA-A2+ peptide) attached to the targeting molecules of the SUPRA / UNI CAR system can be produced to be commercially being available to be combined with ideal SUPRA CAR T-reg / T effector for targeting and destroying the reactive T-cells against the recipients. A library of wild type and/or synthetic MHC monomers/oligomers (e.g. HLA-A2+ peptide) attached to the targeting molecules of the universal CAL system can be generated to be mixed with universal CAL T-reg / T effector for targeting and killing the reactive T-cells against the recipients within the donor T cell population.

Example 6

[00315] Although new advances have increased survival after allogeneic hematopoietic stem cell transplantation (HCT), chronic graft-versus-host disease (GvHD) is still the leading cause of late

morbidity and mortality after transplant. Current treatment choices are limited in efficacy specifically in steroid-refractory disease, and there is no robust data to help with management decisions.

[00316] Adoptive T cell therapy (ACT) refers to the therapeutic use of T cells. T cells genetically engineered to express chimeric antigen receptors (CAR) constitute the most clinically advanced form of ACT approved to date for the treatment of CD19-positive leukemias and lymphomas and produced remarkable results in clinical. This technology provides the opportunity to target the cells with specific antigens or receptors very accurately. In the context of GvHD, UNICAR T cells can be designed to find the recipient reactive T cell in the donor T cell populations. If the pMHC of the recipients be fused to a Target Module that bind to the UniCAR, this system can recognize the TCR repertoires in the donor T/B cell population that can bind to those MHCs.

[00317] If the recipient pMHC (tetramer / dextramer) is used in combination with CAR T effector / Treg for the abrogation of the recipient reactive T-cells in the donor HSC, these cells will be depleted and GvHD would not happen. A library of identified different antigens + correlated human / animal models MHC tetramers / dextramers (e.g. HLA-A2+ peptide) attached to the targeting molecules of the SUPRA / UNI CAR system can be produced to be commercially being available to be combined with ideal SUPRA CAR T-reg / T effector for targeting and destroying the reactive T-cells against the recipients.

Example 7 - Engineered Lymphocytes for Prevention of Pediatrics Vascularized Composite Allograft Rejection Engineered

[00318] Described herein is the development of a clinically applicable tolerance-inducing regimen for VCA transplantation through the establishment of stable mixed chimerism, augmented by the state-of-the-art CAL and/or CAR T cell adoptive immunotherapy and synthetic biology.

[00319] Mixed chimerism in animal models and human allograft recipients have only shown to be transient, suggesting that tolerance here relies on the peripheral inactivation of donor-specific T cell. The difficulty in achieving durable mixed chimerism and long-term graft acceptance is the presence of high levels of alloreactive memory T cells that are known to hinder tolerance induction in sensitized rodents, NHPs, and human. The present approach to achieving stable mixed chimerism is to utilize advanced engineering techniques to generate CAL and/or CAR T cell therapeutics to specifically delete the recipient's alloreactive memory immune cells that are reactive against the donor bone marrow and tissue cells to help with engraftment of HSCT and stable mixed chimerism. Selective depletion of memory T lymphocytes with CAL and/or CAR-T cell therapy can help to achieve durable mixed chimerism and tolerance which eventually leads to an immunosuppressant free regimen. Currently this challenge is addressed by administering several immunosuppressant combinations in combination with intense whole-body irradiation which result in depletion of all the immune cells as a consequence of nonspecific targeting. This shotgun approach imposes a severe

immune compromised state to the recipients which subsequently brings myriads of consequences including opportunistic infections and malignancies. The technology herein can delete specifically memory T cells (Aim 1) or only alloreactive memory T cells (Aim 2):

[00320] Aim 1: It has been shown that CD127+/CD45RO+ memory T cells have a significant role as central, effector and stem cell memory T cells and are known as the most potent constituents of the alloreactive T cell repertoire. These cells have been shown to be the major contributor in chronic rejection of the allograft. To address the challenge of memory T cells a CAL and/or CAR T-effector cell can be generated with scfv against two general markers of memory T cells (CD127+ and CD45RO+). This will be followed by the induction of mixed chimerism protocol in a double humanized mouse model. The results from this aim will show depletion of memory T-cells increases the efficacy of mixed chimerism protocol by improving the engraftment of hematopoietic stem cells hence helping to eliminate or decrease the need for immunosuppressant in mixed chimerism induction protocols.

[00321] Aim 2: Due to the diversity of antigen-specific T cells in the context of transplantation, a CAL and/or CAR system is provided that has the flexibility to locate and attack different alloreactive T cells simultaneously. CAL and/or CAR T-eff cells with donor pMHC can target alloreactive T-cells that have TCR against the donor MHCs. We will start with single and double specific antigen-MHC systems. Then, we will utilize a peptide library that is generated from the donor's allograft peptidome to be loaded on commercially-available, exchangeable peptide-MHC multimers. The combination of these pMHCs and CAL and/or CAR T cells will be used to target and destroy alloreactive T cells of the recipients. This development provides a flexible CAL and/or CAR design that can target antigen specific alloreactive T cells.

[00322] Described herein is the investigation of a novel adaptive immune cell therapy strategy, making use of the CAL and/or CAR T-cell technology, for VCA tolerance induction.

Example 8 - Engineered Lymphocytes for Prevention of Pediatrics Vascularized Composite Allograft Rejection

[00323] Significance: There are nearly 2 million people living with limb loss in the US, where over 185,000 amputations occur each year, most of which are sustained by victims of burn injuries, traffic accidents, and medical conditions¹. 112600 children with amputations were treated in US emergency departments alone from 1990 to 2002. Children are frequently the victims of severe burns with limb loss² and they will suffer from its consequences for the rest of their lives. Over the last decade, vascularized composite allotransplantation (VCA), the transplantation of limbs and face from a deceased donor, has become a good alternative for the reconstruction of devastating injuries of these specialized tissues. VCA represents a unique new treatment option for severe soft tissue defects following burn injury to achieve both psychosocial and functional rehabilitation³. The world's first

pediatric bilateral hand transplant was successfully performed between an unrelated donor-recipient pair in 2016⁴. The shortcoming is that despite the use of potent immunosuppressive drugs, acute rejection of “foreign” VCA occurs in up to 90% of patients⁵. Increased doses of immunosuppression, with numerous life-threatening complications, is the current approach to prevent loss of the VCA⁵.

[00324] During the course of evolution, our immune system has gained the ability to recognize self from nonself cells and attack the “foreigners” by cells such as T Lymphocytes. The Human Leukocyte Antigen (HLA) system (Major Histocompatibility Complex [MHC] in human) comprises cell surface molecules specialized to present antigenic peptides to the T-cell⁶⁷. These peptides are known as Minor Histocompatibility Antigens (MiHA). MiHA sequences can differ among individuals and many of these differences can be recognized by T cells, hence causing the initiation of the rejection process. T-cells are trained to recognize self from non-self MHC / peptides. Each person can present a combination of 12 different MHC class I / II on the surface of his/her cells. (HLA) typing is used to match recipients and donors MHCs for transplants. A close match between a donor’s and a recipient’s HLA markers is essential for a successful transplant outcome. A potential donor must match a minimum of 6-8 HLA markers which makes finding an ideal donor very difficult. Even after finding the semi ideal donor these patients will be on immunosuppressants for the rest of their life⁸. While these drugs are generally effective, the sequelae of such chronic immunosuppression are well known, and most recipients continue to develop myriad side effects and complications, including opportunistic infections, multiple organ dysfunction, and malignancies.

[00325] Transplantation tolerance that allows for the elimination of immunosuppressive drugs, has been the “holy grail” for transplantation medicine since its beginnings⁹. Developing a reproducible, safe tolerance induction protocol would expand VCA use in burn patients as well as in congenital anomalies as demonstrated by the successful transplantation of the arm and hand⁶ and the lower extremity¹⁰. The extension into other organ transplants is also a clear direction to broaden the significance. The overall goal of this research is to enable the clinically applicable tolerance-inducing regimen for VCA transplantation through the establishment of stable mixed chimerism, augmented by the state-of-the-art adoptive immunotherapy and synthetic biology.

[00326] Tolerance can be induced through the development of a mixed chimerism protocol for VCA transplants in partially mismatched subjects^{8,11}. Mixed chimerism is the use of a donor hematopoietic stem cell transplant (HSCT) along with a VCA to induce a hybrid immune system in the recipient that recognizes the donor VCA as “self” and therefore does not reject it. Mixed chimerism in nonhuman primates (NHPs) and human allograft recipients has only shown to be transient, suggesting that tolerance here relies on the peripheral inactivation of donor-specific T cell. The difficulty in achieving durable mixed chimerism and long-term graft acceptance is the presence of high levels of alloreactive memory T cells that are known to hinder tolerance induction in both sensitized rodents^{12, 13} and NHPs^{14,15}. The present approach to achieving stable mixed chimerism is to

utilize advanced engineering techniques to generate T cell therapeutics to specifically delete the recipient's alloreactive memory immune cells that are reactive against the donor bone marrow and tissue cells.

[00327] Cellular Immunotherapy

[00328] Recently, T cells that are genetically engineered to express chimeric antigen receptor (CAR) produced remarkable results for the treatment of CD19-positive leukemias and lymphomas, leading to complete remissions in pediatric patients^{16,17} and evidencing that immune cells can be targeted and eliminated by CAR T cell technology. Thus far, CAR T therapeutics have led to two FDA-approved therapies—the two medication were licensed by Novartis and Gilead in the value 9 and 12 billion dollars¹⁸. First-generation CARs contained only an extracellular antigen-binding domain, a transmembrane domain, and the signaling domain of CD3z. In later generation CARs, intracellular costimulatory domains, derived from either CD28 / 4-1BB were added to enhance proliferation, persistence, and activity¹⁹. Recently, to expand the capability of CAR T cells, we introduced a split, universal, and “programmable” (SUPRA) CAR system that simultaneously encompasses multiple critical features²⁰. This system has the ability to switch targets without reengineering the T cells, finely tune T cell activation strength, and sense and respond to multiple antigens. These features make the split CAR-T technology uniquely suitable for targeting alloreactive T-cells in recipients and enhancing transplantation tolerance.

[00329] Mixed Chimerism and Tolerance induction in VCA

[00330] Large animal models of Mixed Chimerism: Our laboratory achieved transient mixed chimerism in two-haplotype full-mismatch MGH miniature swine after musculocutaneous VCA and nonmyeloablative recipient conditioning (total body irradiation (TBI) and transient T-cell depletion) concomitant to bone marrow transplantation (BMT)²¹. While the immunomodulatory effect of BMT was demonstrated through *in vitro* unresponsiveness to the donor, the chimerism levels fell rapidly following cessation of immunosuppression with resulting VCA rejection and loss. This was the first experimental VCA study to demonstrate that transient chimerism alone is neither sufficient for achieving transplant tolerance nor prolonging VCA survival.

[00331] Recently, in order to progress towards clinical translation, the previous Mixed Chimerism Induction Protocols (MCIP) in miniature swine were modified to remove T-cell depletion completely in exchange for a higher dose, TBI and TI commencing two days prior to surgery. Following osteomyocutaneous VCA, a peri-operative course of co-stimulatory blockade was given (on POD0, 2, 4 and 6) in addition to a 30-day regimen of tacrolimus (target levels: 10-15 ng/mL) before gradual taper to discontinuation on POD 45²². With this MCIP, we were indeed able to achieve stable mixed chimerism (n=2) following donor BMT without the development of split tolerance in MHC class I mismatched/class II matched recipients and have successfully withdrawn all immunosuppression for >100 days. Building on these results, the next iteration towards clinical translation of these studies is

to decrease the toxicity of current protocol to be able to extend it for achieving stable mixed chimerism across full MHC-mismatch barriers - which can utilize immunomodulatory strategies such as CAL and/or CAR T cell therapy for depletion of alloreactive memory T cells that may eventually result in elimination or decrease in the immunosuppressive regimen for VCA transplantation.

[00332] Small animal models of Mixed Chimerism: The principles that underlie tolerance induction through mixed chimerism induction protocol are the same for all species. Briefly, mice can be conditioned with intraperitoneal mAb injections including, anti-CD8 and anti-CD40L, anti-CD154, costim. blockade (on day 0 with respect to the BMT). This will be combined with, TBI to be given 6 h before injection of 2.5×10^5 bone marrow cells (BMC). The compositions and methods described herein can be used for improving mixed chimerism induction protocol in mice models.

[00333] Described herein is a CAL and/or CAR designed to target and destroy alloreactive memory T cells thereby improving the engraftment of hematopoietic stem cells and achieving durable mixed chimerism with minimal or no need for toxic preconditioning protocols. To test this multi-pronged approach, we will first evaluate two different strategies of targeting alloreactive memory T cells. The first approach is to design a CAL and/or CAR system that will specifically attack memory T cells through surface markers that uniquely identify this cell population regardless of their TCR specificity. This approach will spare the other immune cells, and, thus retain a healthy immune system.

[00334] A second approach is to design CAL and/or CARs that specifically bind to alloreactive TCRs on the recipient's T cells. To do so, instead of using a single-chain variable fragments (scFvs) that is in all classical CAR T cells, we will use peptide-MHC multimers as the ligand recognition domain on the CAL and/or CAR. However, due to a large number of unique alloreactive TCR, many different CAL and/or CAR/pMHCs are needed. This poses a challenge in designing and introducing these CAL and/or CARs into T cells. To circumvent this challenge, we propose to use a two-component split CAL and/or CAR design composed of universal CAL and/or CAR and an adaptor molecule that bridges the CAL and/or CAR T cells to alloreactive T cells. The universal CAL and/or CAR comprises a scFv targeting FITC fused to TCR signaling domains. The adaptor molecules consist of pMHC tetramer fused to a FITC. The addition of pMHC tetramer will bind to the alloreactive TCR and recruit the universal CAL and/or CAR T cell to kill alloreactive T cells. This design allows us to be able to target many variants of TCR with only one CAL and/or CAR. Next, the donor MHCs will be loaded with donor tissue specific peptides that will be generated by utilizing previously published protocols. We can first design CAL and/or CARs to target mouse MHC in a humanized / skin graft mouse model.

[00335] Data- Targeting Alloreactive T-cells with peptide-MHC tetramers
The following have been addressed by *in vitro* data:

1. Whether targeting a memory T cell by using CAR T + pMHC multimer is obtainable. In this embodiment, the anti-FITC CAR expressed by the Jurkat cells is a CAR and the pMHC multimer is a CAL.

A. Would the Tetramer efficiently bind to the CAL and/or CAR and target T cells?

B. Would the Jurkat CAL and/or CAR T cells get activated by attaching to the adaptor + Target

C. Would the Target OT I cells get activated and try to kill the Killer Target T cells?

2. Whether killing a memory T cell with primary CD8 T cell + pMHC multimer would be feasible?

[00336] 1. Peptide-MHCs Tetramers activate Jurkat Cells but not the Target Cells

[00337] We used anti-FITC CAR Jurkat cells. These cells are an immortalized line of T lymphocyte cells that are used to study activation, signaling, and expression of various chemokine receptors of T cells. Jurkat cells cannot act as a killer cell but will respond to and get activated by the ligands in the same way as actual T cells. Fig. 1 shows tetramers attaching to and staining OTi target T cells in a dose dependent fashion.

[00338] The anti-FITC CAR Jurkat cells used in these experiments comprise the following nucleotide sequence and express the following polypeptide sequence.

[00339] SEQ ID NO: 2747. DNA sequence of anti-FITC CAR. Nucleotides 1-9 = Kozak sequence. Nucleotides 10-78 = CD8a leading peptide. Nucleotides 79-831 = anti-FITC ScFv. Nucleotides 832-861 = myc. Nucleotides 874-1008 = CD8 hinge. Nucleotides 1009-1211 = CD28. Nucleotides 1212-1338 = 4-1BB. Nucleotides 1339-1677 = CD3 ζ . Nucleotides 1678-2388 mCHERRY.

gccgccaccATGGCCTTACCAGTGACCGCCTTGCTCCTGCCGCTGGCCTTGCTGCTCCACGCC
GCCAGGCCGGCTGCAcaagttcagctcgttgaatccggcggaacctgttcaaccgggggggtccctccgccttagttgtccgcgat
ctggttttacgttttgatcattttccatgtcatgggtgcggcaggctcccggggggggactcgaatgggttgcgggtcttagcgcccgatcaagctc
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ccaatccgaggacgaggccgactactactgtgtgcttgggacgactctctcagagtttttggggaccgggtacaaaactgactgttcttgggG
AACAAAACTCATCTCAGAAGAAGATCTGAATGGGGCCGCAACCACGACGCCAGCGCCG
CGACCACCAACACCGGCGCCCAACATCGCGTTGCAGCCCCTGTCCCTGCGCCCAGAGGC
GTGCCGGCCAGCGGCGGGGGGCGCAGTGCACACGAGGGGGCTGGACTTCGCCTGTGATT
TTTGGGTGCTGGTGGTGGTGGTGGAGTCCTGGCTTGCTATAGCTTGCTAGTAACAGTGG
CCTTTATTATTTTCTGGGTGAGGAGTAAGAGGAGCAGGCTCCTGCACAGTGACTACATGA
ACATGACTCCCCGCCGCCCGGGCCCAACCGCAAGCATTACCAGCCCTATGCCCCACCAC

GCGACTTCGCAGCCTATCGCTCCAAACGGGGCAGAAAGAACTCCTGTATATATTCAAA
 CAACCATTTATGAGACCAGTACAACTACTCAAGAGGAAGATGGCTGTAGCTGCCGATT
 TCCAGAAGAAGAAGAAGGAGGATGTGAACTGAGAGTGAAGTTCAGCAGGAGCGCAGAC
 GCCCCCGCGTACAAGCAGGGCCAGAACCAGCTCTATAACGAGCTCAATCTAGGACGAAG
 AGAGGAGTACGATGTTTTGGACAAGAGACGTGGCCGGGACCCTGAGATGGGGGGAAAG
 CCGAGAAGGAAGAACCCTCAGGAAGGCCTGTACAATGAACTGCAGAAAGATAAGATGG
 CGGAGGCCTACAGTGAGATTGGGATGAAAGGCGAGCGCCGGAGGGGCAAGGGGCACGA
 TGGCCTTTACCAGGGTCTCAGTACAGCCACCAAGGACACCTACGACGCCCTTCACATGCA
 GGCCCTGCCCCCTCGCGGCatgGTGAGCAAGGGCGAGGAGGATAACATGGCCATCATCAA
 GGAGTTCATGCGCTTCAAGGTGCACATGGAGGGCTCCGTGAACGGCCACGAGTTCGAGA
 TCGAGGGCGAGGGCGAGGGCCGCCCTACGAGGGCACCCAGACCGCCAAGCTGAAGGT
 GACCAAGGGTGGCCCCCTGCCCTTCGCCTGGGACATCCTGTCCCCCTCAGTTCATGTACGG
 CTCCAAGGCCTACGTGAAGCACCCCGCCGACATCCCCGACTACTTGAAGCTGTCCTTCCC
 CGAGGGCTTCAAGTGGGAGCGCGTGATGAACTTCGAGGACGGCGGCGTGGTGACCGTGA
 CCCAGGACTCCTCCCTGCAGGACGGCGAGTTCATCTACAAGGTGAAGCTGCGCGGCACC
 AACTTCCCCTCCGACGGCCCCGTAATGCAGAAGAAGACCATGGGCTGGGAGGCCTCCTC
 CGAGCGGATGTACCCCGAGGACGGCGCCCTGAAGGGCGAGATCAAGCAGAGGCTGAAG
 CTGAAGGACGGCGGCCACTACGACGCTGAGGTCAAGACCACCTACAAGGCCAAGAAGCC
 CGTGCAGCTGCCC GGCGCCTACAACGTCAACATCAAGTTGGACATCACCTCCCACAACG
 AGGACTACACCATCGTGGAACAGTACGAACGCGCCGAGGGCCGCCACTCCACCGGCGGC
 ATGGACGAGCTGTACAAGtaa

[00340] SEQ ID NO: 2748. Peptide sequence of anti-FITC CAR. Residues 1-21 = CD8a leading peptide. Residues 22-274 = anti-FITC ScFv. Residues 275-284 = myc. Residues 289-333 = CD8 hinge. Residues 334-401 = CD28. Residues 402-443 = 4-1BB. Residues 444-556 = CD3ζ. Residues 557-792 mCHERRY.

MALPVTALLLPLALLLHAARPAAQVQLVESGGNLVQPGSLRLSCAASGFTFGSFSMSWVR
 QAPGGGLEWVAGLSARSSLTHYADSVKGRFTISRDNKNSVYLQMNSLRVEDTAVYYCAR
 RSYDSSGYAGHFYSYMDVWGQGTLLTVSGGGGSGGGGSSVLTQPSSVSAAPGQKV
 TISCSGSTSNIGNNYVSWYQQHPGKAPKLMIYDVSKRPSGVPDRFSGSKSGNSASLDISGLQS
 EDEADYYCAAWDDSLSEFLFGTGTKLTVLGEQKLISEEDLNGAATTTTPAPRPPTPAPTIALQP
 LSLRPEACRPAAGGAVHTRGLDFACDFWVLVVGVLACYSLLVTVAFIIFWVRSKRSRLH
 SDYMNMTPRRPGPTRKHYQPYAPPRDFAAYRSKRGRKKLLYIFKQPFMRPVQTTQEEDGCS
 CRFEEEEGGCELRVKFSRSADAPAYKQGQNQLYNELNLGRREEYDVLDRRGRDPPEMGGK
 PRRKNPQEGLYNELQKDKMAEAYSEIGMKGERRRGKGHGGLYQGLSTATKDTYDALHMQ
 ALPPRGMVSKGEEDNMAIIKEFMRFKVHMEGSVNGHEFEIEGEGEGRPYEGTQTAKLKVTK
 GGPLPFAWDILSPQFMYGSKAYVKHPADIPDYLKLSFPEGFKWERVMNFEDGGVVTVTQDS

SLQDGEFIYKVKLGRGTNFPDGPVMQKKTMGWEASSERMYPEDGALKGEIKQRLKLKDGGH
YDAEVKTTYKAKKPVQLPGAYNVNIKLDITSHNEDYTIVEQYERAEGRHSTGGMDELYK

[00341] Next, we showed that binding of adaptors to the OTi target T cells would not activate them. To evaluate that, we looked at CD69 expression which is a general activation marker for T cells. Interestingly, although adaptor binds to and stains target T cells (Fig. 1) it did not cause activation of OTi target T cells (Fig. 2).

[00342] B. Subsequently, we studied the activation of Jurkat CAR T cells after binding to the adaptors. As shown in Fig. 3, binding of adaptor to the Jurkat T cells causes high expression of CD69 at all measured time points.

[00343] This activating binding of Jurkat cells - pMHC is in contrary to the binding of OTi (target) - adaptor (Fig. 2). The best explanation for this interesting finding may lie behind the fact that FITC / anti-FITC binding between Jurkat - pMHC is much stronger than OTI - pMHC as the first one is a high affinity antigen-antibody binding and the second one is a weaker TCR-pMHC binding. This difference in the strength of binding between CAR - adaptor and Target - adaptor makes this novel approach an ideal setting for the purpose of targeting memory T cells.

[00344] C. We also looked at the Jurkat cells count after 24 hrs. as an indicator of their condition and whether this activation would cause any cell death indicated by a decrease in their numbers (Fig. 2).

[00345] 2. Primary CD8 anti-FITC CAR T cells can specifically target and kill alloreactive OTi cells.

[00346] The previous set of experiments with Jurkat +adaptor +OTi target cells confirmed the feasibility of targeting alloreactive T cells by adaptor molecules and showed activation of CAR T cells against the target cells. Next, we wanted to study whether primary CD8 CAR T killer cells + adaptor molecules can be used not only to identify but also to kill the OTi T cells. As shown in the previous set of experiments the binding between pMHC and target cell was not causing activation of target T cells in contrary to the adaptor (FITC+ pMHC, e.g., the CAL) - CAR T cells binding. Hence, we used human primary CD8 T cells to see whether the strength of CART +adaptor (e.g., CAL) +target is enough to initiate and accomplish the killing process. Different concentrations of adaptor+CD8 CAR T were co-cultured with OTi T cell to see the activation and cytotoxicity of each T cell type. As shown in Figs. 4 and 5, CD8 CAR T cells cytotoxicity on OTi cells is dependent on the adaptor (e.g., CAL) concentration and was over to 50% with the maximum dose that we used. Additionally, this cytotoxicity is highly specific. (Fig. 5) Human CD8 anti-FITC CAR T cells were

used here. Similar validations can be conducted with mouse anti-FITC CARs in both *in vivo* and *in vitro* experiments.

[00347] Described herein is a novel method to use pMHC multimers for specifically targeting and destroying allo / autoreactive T cells responsible for graft rejection and autoimmune diseases. Our approach is the first demonstration of utilizing pMHC multimer (e.g. CAL) in combination with CAR T cell technology for specific targeting of alloreactive T cell population. We believe this approach is the most specific strategy for eliminating the destructive role of alloreactive T cells in transplantation. The potential application of this method extends far beyond elimination of immunosuppressants in transplantation rejection and GVHD treatment and could be applicable to many autoimmune conditions including type 1 diabetes, multiple sclerosis, rheumatoid arthritis, scleroderma, and myriad disorders that originate from auto reactive T cells dysfunction.

[00348] Provided herein is a novel approach for targeting the memory repertoire with anti CD45RO / CD127 with CAL and/or CAR T cell therapy. Although this strategy might not be as specific as using pMHC multimers, it still conveys a significant novelty. By depleting the memory T cells population there is much less of a need for induction protocols.

[00349] Also contemplated herein are individualized peptide libraries based on the differences between the recipient and the donor proteome and immune-peptidome. Hundreds to thousands of MHC-associated peptides can now be identified in a single measurement using optimal biological model systems. As mentioned earlier, studies involving MHC identical grafts indicate that minor histocompatibility antigens may also mediate rejection. Human Immuno-Peptidome Project (HIPP) is an international project that was created to accelerate research toward robust and comprehensive analysis of immunopeptidome. HIPP has published the technical guidelines that represent the information required to sufficiently conduct and interpret all of the immunopeptidomics experiments. These nonhomogeneous peptides can potentially initiate rejection through activating alloreactive T cells.

[00350] By utilizing these peptides in combination with exchangeable MHC tetramer systems, we can employ these peptides to identify and target donor reactive T cells. The default peptide on exchangeable peptide MHC systems can be removed by UV light exposure and replaced by any desired peptide. The donor multimers MHCs can be generated using commercially available services, unloaded with UV and then loaded with donor tissue specific peptides that will be generated by utilizing previously published protocols. By utilizing these peptides in combination with exchangeable MHC tetramer systems, we can employ these peptides to identify and target donor reactive T cells.

[00351] As described herein, selective depletion of memory T cells with CAR-T cell therapy can help in achieving durable mixed chimerism and tolerance which lead to the elimination or decrease in immunosuppressive regimens. Currently this challenge is addressed by administering intense whole-

body irradiation with several immunosuppressant combinations for targeting all the immune cell populations. This shotgun approach results in severe immune compromised state in the recipient which subsequently brings myriads of side effects and complications, including opportunistic infections, reno-vascular dysfunction, and malignancies.

[00352] Memory T cells (CD127+/CD45RO+) are known to be the main contributor in rejecting allografts 2425. Hence, their depletion is expected to help engraftment of HSC and increase the survival of allografts. To investigate this hypothesis, we will generate CAL and/or CAR T-effector cell with scfv against a general marker of memory T cells (CD127+and CD45RO+). This will be followed by induction of mixed chimerism protocol in a humanized mice model. The engraftment and stability of mixed chimerism will be followed by the presence of donor lymphoid and myeloid cells in the recipient blood stream until the study endpoints.

[00353] Activity 1.1 Generation and evaluation of AND logic CAL and/or CAR T cells to only target cells co-expressing both CD127+ and CD45RO+. Since no single surface marker can uniquely specify memory T cells, we will need to target a combination of CD markers to ensure specificity. We chose CD127 and CD45RO because they have proven to uniquely identify memory T cells 2627. To accomplish this combinatorial targeting, we will leverage an advance CAR system called SUPRA. This (SUPRA) CAR is a two-component receptor system comprises a universal receptor (zipCAR) expressed on T cells and a targeting scFv adaptor (zipFv). The zipCAR universal receptor is generated from the fusion of intracellular signaling domains (Figure 6) and a leucine zipper as the extracellular domain. The zipFv adaptor molecule is generated from the fusion of a cognate leucine zipper and an scFv. The scFv on the zipFv binds to the antigen, while the leucine zipper binds to and activates the zipCAL and/or zipCAR on the T cells. For transplantation immunotherapy, these features can mitigate over-activation and enhance specificity.

[00354] The SUPRA CAL and/or CAR system can also be designed to perform AND combinatorial logic of antigen recognition. Orthogonal leucine zipper pairs can be used to generate CAL and/or CARs with split signaling domains (e.g., CD3z, CD28, 4-1BB), thus enabling independent and simultaneous control of these pathways (Fig 6B). T cells need both CD3z and costimulatory signaling simultaneously to be fully activated therefore, each CAL and/or CAR can be readily paired with scFvs that target different antigens, thereby enabling two antigen combinatorial and logical antigen sensing.

[00355] Using the SUPRA system in human primary T cells, we have demonstrated that the activity of SUPRA CARs can be finely regulated via multiple mechanisms to limit over-activation (Fig. 7). SUPRA CAR T cells can also logically respond to multiple antigens for improved target specificity. Furthermore, orthogonal SUPRA CARs can be used to inducibly regulate multiple signaling pathways or T cell subtypes to increase the breadth of immune responses that can be

achieved. A summary of the SUPRA CAL and/or CAR T cell features used for this project can be found in Figs. 6A-6B, 7A-7D.

[00356] zipCAR Receptor Construct Design: As described herein, AND logic CAL and/or CAR T are generated by fusing different leucine zippers to the hinge region of the human CD8a chain and transmembrane and cytoplasmic regions of the human CD28, or CD3z signaling endo-domains. All CAL and/or CARs can contain a myc tag to verify surface expression. Besides, these primary T cells will also be fused to mCherry after CD3z chain to visualize expression.

[00357] zipFv Construct Design: The zipFv molecules contain an scFv against CD127 fused to an SYN2 (to stimulate the CD3z SYN1 zipCAR), and an scFv against CD45RO fused to a JUN zipper (to stimulate the costimulatory FOS zipCAR). The scfvs sequences are available and can be constructed rapidly through commercial DNA synthesis²⁸.

[00358] zipCAR transduction: Human PBMC are purified using separation kits. are introduced into primary human T cells via retroviral transduction. Expression of zipCARs is quantified via myc and V5-tag immunostaining and flow cytometry. As controls, a zipCAR that contains both CD3z and CD28 (i.e. SYN1-CD28-CD3z) can be used. This zipCAR cannot perform logic computation. A CD45RO-SYN2 or CD127-SYN2 zipFv can be added to ensure that zipCARs can kill CD45RO+ or CD127+ cells. These controls will also serve as reference for specificity.

[00359] Evaluate the activity and efficacy of anti-CD127+/CD45RO+CAR T cells in vitro

[00360] Co-culturing PBMCs with CD127+ /CD45RO+ cells with SUPRA CAR T cells to investigate the specificity of targeting

[00361] This experiment examines whether this system specifically kills CD127+ and CD45RO+ double positive T cells amongst a large population of PBMCs. PBMCs are obtained either from known VCA recipients or Mass General Blood Bank Center after being approved by IRB. First, human CD45RO+ and CD127+ are stained to identify the initial percentage of memory cells in the whole population (CD45RO /CD127 double positive vs double neg). Next, PBMCs are co-cultured with zipCAR expressing CD8+ T cells with zipFvs (anti CD127 and anti CD45RO) with the same condition. At the study time points (24, 48, 72hrs) significant changes in the ratio of CD45RO/CD127 +/+ to -/- will be considered as an indicator of whether SUPRA CARs were efficient in targeting specific T cells rather than non-specific cytotoxic effects. Additionally, engineered T cells and zipFvs will be cocultured with the memory T cells at 3 different zipFvs concentrations (5, 25, 50ng/well) to determine the correlation between cell killing and zipFv concentration. T cell activation (CD69 expression, IL-2 and IFN-g in the media) and cytotoxicity against memory T cells (no. of remaining live cells) will also be measured. The proliferation of T cells will be measured by cell counting. All conditions will be tested at least in triplicate.

[00362] Table 1 – outline of experiments

1	PBMC - Ctrl
2	PBMC + SUPRA CAR T
3	PBMC + SUPRA CAR T + scFv against CD45RO
4	PBMC + SUPRA CAR T + scFv against CD127
5	PBMC + SUPRA CAR T + scFvs CD127 + CD45RO

[00363] To evaluate and characterize the effect of memory T cells on the HSCT engraftment.

[00364] Generation of a humanized mouse model with the VCA-recipients PBMCs

[00365] To examine the effect of CD45RO / CD127 targeting with CAR T cells on the graft take and mixed chimerism sustainability, a humanized mouse model is generated by utilizing the PMBCs of VCA-recipients (Figs. 8A, 8B). The humanized PBMC model has the fastest engraftment rate using adult PBMCs and enables studies that require a strong memory T cell function. VCA recipients' PBMCs hold an alloreactive population of memory T cells (CD45RO+/ CD127+) and makes this a reliable model for studying their depletion effects on the rejection of the graft. The number of mice used per group in this study is chosen based on the recommendations from previous studies ²⁹. In brief, irradiated NSG (MHC I/II K/O) (3 Gy) mice will be injected with 5×10^5 PBMC cells and human peripheral reconstitution is measured by flow cytometry every other week ³⁰.

[00366] Testing the effects of memory T cells depletion on mixed chimerism establishment cells in a skin graft humanized mouse model : The NSG (MHC I/II K/O) mouse model that was generated by using the VCA-recipient PBMCs are used as the foundation for induction of mixed chimerism. The presence of memory T cells in the human PBMCs of this model imitates the challenges for induction of mixed chimerism in human subjects. NSG Hu-PBMCs are treated with / without (ctrl) anti CD45RO/CD127 CAR T to deplete the memory compartment of their T cells population (Figs. 8A, 8B).

[00367] Fetal CD34+ HSC (and skin tissue) can be obtained from Advanced Bioscience Resources. Fetal HSCs are utilized to evaluate the generation and establishment of multi-lineage engraftment by using previously published Mixed Chimerism Induction Protocols (MCIP) in mice ²³. In short, male mice are treated with anti CD154, anti-CD40, CTLA-4 and receive 3 Gy TBI. Six hours later they receive 2×10^5 CD34+ HSC bone marrow cells. Engraftment is determined by measuring the presence of donor myeloid and lymphoid cells at different time points. Mixed chimerism is defined as at least 5% of leukocytes in each of the lineages being donor derived. Less than 5% or loss of multi-lineage status is considered as failure of chimerism.

[00368] Next, the aforementioned humanized mouse model (VCA-Recipient PBMCs-CD34 HSC) is utilized as a skin graft model to test the effect of depleting CD45RO / CD127 cells on allograft survival. Fetal skin graft is obtained from Advanced Bioscience Resources from the same CD34+

HSC donor. At week 10 post PBMC injection, skin grafts from the HSC donor and another allogeneic source are used to test whether nonspecific depletion of memory T cell repertoire affects the skin grafts survival (Fig. 8, 9). Mice are followed until graft rejection or POD 100.

[00369] Skin grafting will be conducted using previously published protocols³¹. In short, 2 pieces of tail skin from the donor are transplanted on the back of one recipient. Following dressing removal (POD7), mice and their skin grafts are evaluated daily. Graft failure is determined by the physical appearance of the graft; including color, texture, and contour. Histology is also performed to provide supportive data regarding the clinical signs of graft rejection or engraftment. Grafts with more than 50% shrinkage in size or change in color and flexibility (dark and firm grafts) are considered as failed. Skin graft are followed for 100 days.

[00370] Table 2 – outline of experiments

#	N	Recipient (NSG-Hu-VCA-Rec.-PBMCs)	Mixed Chimerism	Skin Grafts
1	8	Control - No treatment	(MCIP)	+
2	8	Control - zipFv (anti CD45RO / CD127)	PBMCs + HSCT + (MCIP)	+
3	8	Control - SUPRA CAR T	PBMCs + HSCT + (MCIP)	+
4	8	SUPRA CAR T + zipFv (anti CD45RO / CD127)	PBMCs + HSCT + (MCIP)	+
5	8	SUPRA CAR T + zipFv (anti CD45RO / CD127)	No PBMCs + HSCT, No MCIP	+
6	8	SUPRA CAR T + zipFv (anti CD45RO / CD127)	No MCIP	+
7	8	SUPRA CAR T + zipFv (anti CD45RO / CD127)	1/3 of regular dose MCIP	+

[00371] Off-target toxicity occurs when T cells are not able to distinguish normal cells from target cells. In this case CD45RO is the marker that is mainly expressed on the T cells but CD127 is a more common surface marker and can be found on B and dendritic cells. Even though the “Combinatorial AND Logic” CAR system that is described herein has shown to be highly specific, there is a chance for off-target effects in this system. As an alternative approach, all of the CD45RO+ population can be targeted, which consists of not only central memory T cells but also targets effector T cells as well. This will cause more T cells to be targeted nonspecifically but simultaneously might decrease the chance of immune reaction against the graft.

[00372] Some of the CAR T effector cells will express memory phenotype such as CD45RO /CD127 after remaining activated for a period of time. This means that CAR T cells might attack each other after changing phenotype. Long activation of CAR T cells within the patient's body has always been a matter of concern. This hypothetical situation can be considered as a safety step as CAR T cell will clear out themselves after completing their task which is destroying memory T cells. On the other

hand, if turns out to be true, this self-eliminary phenomenon might increase the number of CAR T cells that are needed to effectively treat the patients.

[00373] Benchmark for success: Blood drawn from the vain tail (40ul) is used every other week for the assessment of peripheral blood chimerism. Monoclonal anti-donor HLA antibody is used for monitoring of chimerism in the various leukocyte lineages, including CD3+/CD8+, CD3+/CD4+, CD3+/CD8-/CD4- (naive T cells), T-Regs, naive and mature B cells (CD19+/CD78- and CD19-/CD78+). Animals are considered chimeric when at least 5% of WBCs in all of these lineages are donor derived. Additionally, the systemic immune status of humanized mice is assessed pre-HSC transplantation and at week 4 and week 8 post-transplant by carboxyfluorescein diacetate succinimidyl ester (CFSE) mixed lymphocyte reaction (MLR) proliferation assays. MLR results are compared before PMBC-HSC transplantation and after study end point.

[00374] **Determine the efficacy of pMHC-CAR T-effector cells on specific depletion of recipient alloreactive T cells and establishment of Mixed Chimerism.**

[00375] Described herein is a flexible CAL and/or CAR design that can target any alloantigen specific T cells. Due to the diversity of alloantigen-specific T cells in the context of transplantation, the CAL and/or CAR system that has the flexibility to locate and attack different alloreactive T cells simultaneously. CAL and/or CAR T-eff. cells with (donor) pMHC can target alloreactive T-cells.

[00376] *The effect of CAL and/or CAR T cell therapy on targeting a double antigen-MHC system (OTi and OTii)*

[00377] In short, pMHC multimer attached to adaptor molecule (e.g., the CAL) + CAR T effector cell is used in the single and double specific antigen-MHC systems. The antigen-specific CAL and/or CAR system can be tested in vitro and in the OTi & ii transgenic mouse model. This model is genetically engineered such that all T cells express receptors that are specific for recognizing chicken ovalbumin peptides (257-264 (OTi) and 329-337 (OTii) in the context of H2Kb (OTi) and I-A b (OTii)). The T cells of these mice models are engineered to express TCRs that specifically recognize pMHC class I / II + Ovalbumin peptides. More importantly, this highly controlled model can be leveraged establish correlation between T cell activities and CAL and/or CAR T cell experimental parameters, such coculture conditions, pMHC affinity, pMHC multimer concentration.

[00378] Design, build, and test anti-FITC CAR/pMHC (e.g., the CAL) system in vitro against antigen specific T cells.

[00379] As mentioned above, older generations of CAR T cells needed to be genetically redesigned to be able to recognize new or multiple targets. To overcome this challenge, mouse split universal CD8 CAR is generated as described herein. For generating mouse FITC-CAR similar steps that were described previously for human anti-FITC CAR can be utilized ²⁰. To evaluate the efficacy of C67BL/6 mouse anti-FITC CAR T cells efficacy, the experiments that are conducted and

mentioned in the section above with human CAR T cells (Fig. 1-5) are repeated with mouse CAR T cells.

[00380] Table 3 – outline of experiments

Group	N	Recipient (B6-wild type + OTI /ii)
1	6	Control – No CAR/ pMHC
2	6	Control + FITC pMHC multimer
3	6	Control + CAR T (no multimer)
4	6	CAR T + FITC pMHC multimer)

[00381] Evaluate the efficacy of CAR T-eff. + FITC-pMHC tetramers (e.g. a CAL) for targeting alloreactive T-cells in the OTi and OTii transgenic mice.

[00382] Ovalbumin-pMHC-CAR T-cells effective in recognizing and destroying OTi and OTii T cells a mixture of the OTi and OTii T-cells are transferred to the wild type mice (B6) recipients. The read out of this experiment is the cell count of OTi & ii before and after CAL and/or CAR T cell therapy. The results show the effect of the CAR T-eff cells with the Ovalbumin peptide + MHC tetramer (e.g., the CAL) is successful in targeting the specific clone of OTi & ii cells.

[00383] Establish the potency of multiple peptide / multiple MHC (e.g., CAL) - CAR T cell therapy for targeting alloreactive T cell repertoire on mixed chimerism and skin graft survival.

[00384] The most significant barrier against graft take is full mismatch across MHCs of donors and recipients³². This challenge is addressed herein by using MHC (e.g., CAL) + CAR T cells to target alloreactive T cells. These MHC multimers are commercially available and can be readily conjugated with FITC labeling to be recognized by the anti FITC CAR T cells. Alternatively, there are many other available options for split universal CAL and/or CAR technology including leucine zippers which provide the option of tuning the affinity of the binding between adaptor (e.g., CAL) and CAR. On the other hand, Minor Histocompatibility Antigens (MiHA) are short immunogenic peptides originating from digested intra / extracellular proteins presented by Major Histocompatibility Complex (human leukocyte antigen). Disparities in minor histocompatibility antigens between individuals who are even MHC matched can induce an immune response after transplantation. These differences have shown to impede tolerance induction through mixed chimerism³³.

[00385] Generating a donor-recipient disparities peptide library for evaluating the efficacy of targeting alloreactive T cells in a humanized mouse model.

[00386] The role of the default peptides on these exchangeable-peptide MHC systems can be switched with the desired peptides (from the donor) using UV light^{34,35}. The large collection of peptides associated with human leukocyte antigens (HLA) is referred to as the human

immunopeptidome. The desired peptides for this study will be generated by the previously established protocols for immune-peptidome library generation^{36,37,38}.

[00387] Generating tissue-specific immune peptide library from the donor and recipient.

[00388] Utilizing previously established protocols we can extract and identify the immunopeptidome of the donor and the recipient. We then identify the peptide sequences that are distinctively different between the donor and the recipient using software tools (below). These disparities in the donors' and the recipients' immune-peptide sequences have the potential to mediate immune rejection. The sequences of the peptides from the donor-recipient disparity peptide library are generated in practical scale by commercially available services³⁹ and loaded on peptide-exchangeable MHC multimers (e.g., a CAL) (QuickSwitch MBL international or Biolegend FlexTetramer) to ultimately be bound to CAR T cells and used for targeting alloreactive T cells in the aforementioned in vitro and in vivo models (Fig. 8, 9). In brief, donor and recipient MHCs associated peptides are isolated independently by immunoaffinity purification using the anti-HLA monoclonal antibodies³⁶. Eluted peptides are identified by different LC-MS/MS systems in DIA (Data Independent A) mode. Mass Spectrometry output files are converted, searched, and statistically validated using software tools (NETMHC, SysMHC). The identified peptides are then clustered (by GibbsCluster v.1) and annotated by length and predicted MHC binding affinity (NetMHC v.4). The final list of high-confidence donor / recipient MHC-associated peptides is compared between the recipient and the donor and the disparate sequences used for building high-quality donor-recipient disparity peptide library, which is employed as a source for generating peptides to be combined with MBL QuickSwitch (or Bio-legend FlexT) MHC tetramer system.

[00389] Table 4 – outline of experiments

Group	N	Recipient (NSG-Hu-VCA-Rec.-PBMCs)	Mixed Chimerism	Skin Grafts
1	8	Control - No treatment	(MCIP)	+
2	8	SUPRA CAR T + FITC / (peptide MHC multimer)	PBMCs + HSCT + (MCIP)	+
3	8	Control - SUPRA CAR T	PBMCs + HSCT + (MCIP)	+
4	8	SUPRA CAR T + FITC / (peptide MHC multimer)	PBMCs + HSCT + (MCIP)	+
5	8	SUPRA CAR T + FITC / (peptide MHC multimer)	No PBMCs + HSCT, No MCIP	+
6	8	SUPRA CAR T + FITC / (peptide MHC multimer)	No MCIP	+
7	8	SUPRA CAR T + FITC / (peptide MHC multimer)	¼ of regular dose MCIP	+

[00390] Evaluating the efficacy of targeting alloreactive T cells in the multiple antigen / MHC setting on mixed chimerism establishment in a skin graft humanized mouse model.

[00391] In the actual context of allotransplantation, both MHC mismatch and MiHA will be a potential source of immune response and rejection. This experiment addresses the question of whether combining the donor-recipient disparate peptide sequences with exchangeable-peptide MHC system (e.g., a CAL) can be used in combination with split CAR T cells to target alloreactive T cells.

[00392] Prior to initiating this experiment, human fetal HSC + skin graft from the same donor is obtained from Advanced Bioscience Resources (CA, US). HLA typing of the donor cells is done and used for generating donor (FITC+ exchangeable-peptide) MHC multimer. Peptides that are used with these multimer are generated based on the results of the work described above herein. Briefly, the exchangeable-peptide MHC (e.g., the CAL) is exposed to UV for the dissociation of their default UV-labile peptide. The disparity peptide library provides the desired peptides to replace the default peptide of this system. Using the same double humanized PBMC / HSCT mouse model the efficacy of this donor/recipient disparity peptide library + MHC is evaluated for specific targeting of alloreactive memory T cells and skin allograft survival. As shown in Figs. 8A-8B, Hu-PBMC treated with exchanged peptide MHC (e.g., CAL) + CAR T. Subsequently, HSCT with fetal HSC is performed and 6 weeks later skin from the same HSC donor is grafted on the back of the mice. Presence of mixed chimerism and graft survival is evaluated using the criteria that was mentioned earlier herein.

[00393] The immunopeptidome library is novel and has not previously been adapted for the context of transplantation. As an alternative to immunopeptidome mapping and disparity library generation, we can apply whole-peptidome (donor-recipient) disparity library, a well-known method.

[00394] Specificity and killing efficiency of CAL and/or CAR T cells, and chimerism levels can be studied in vitro and in vivo. Peripheral mechanisms of rejection or regulation can be assessed with serial mixed lymphocyte reaction (MLR) assays, at experimental endpoint. For our in vivo studies, our standard clinical definition of tolerance in rodents, which will serve as a benchmark, is rejection and immunosuppression-free survival for 100 days post-HSCT will serve as a benchmark. Skin grafts are assessed as mentioned earlier, based on the color, texture and size of shrinkage.

[00395] Impact: Described herein is a novel adaptive immune cell therapy strategy, making use of the CAL and/or CAR T-cell technology, for VCA tolerance induction. Musculoskeletal extremity injuries, including traumatic amputations are frequently seen in the pediatric burn and trauma population. These patients often struggle with severe functional and psychosocial challenges and decreased quality and quantity of life. In turn, the bottleneck to wide application of VCA, with its obvious benefits, would be lifted and effect a paradigm shift in the current management of devastating pediatric burns, which often result in severe disfigurement and amputations. Additionally, successful results enable VCA to be performed for trauma or congenital defects.

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Example 9

[00397] Jurkat + pMHC – OTi

[00398] Described herein is the investigation of whether killer CAR T cells activation by binding to the adaptor (pMHC, e.g. a CAL)) + Target. The experimental design is depicted in Fig. 11.

[00399] FITC-conjugated tetramer mediated activation was verified (Fig. 12) and followed in a time course (Fig. 13). Anti-FITC Jurkat vs OT-I experiments were conducted using 100k cells anti-FITC CAR Jurkat, 100k cells splenocytes from OT-I mouse, and 5 ug/mL to 78 ng/mL FITC-conjugated tetramer (positive, H2Kb-SIINFELK (SEQ ID NO: 2750); negative, I-Ab-AAHAEINEA (SEQ ID NO: 2751)). Total Jurkat cell counts at 24 hours (Fig. 14) and tetramer staining levels (Fig. 15) were also determined. The levels of tetramer and CD69 were also determined (Fig. 16).

[00400] This experiment demonstrated that Jurkat cells get activated in a dose dependent fashion up to 1 ug/ml of tetramer and OTi binding and staining is dose dependent. No cytotoxicity effects were seen on the killer cells and no prominent activation was seen on the target cells.

[00401] Primary T cells – OTi

[00402] Experimental design is shown in Fig. 17. Human CD8 pMHC-CAR T killing is highly specific and MHC-CAR T cell does not kill bystander CD4 T cells (Fig. 18). No cytotoxicity effect was seen on human CD8 pMHC- CAR T (Fig. 19). Cytotoxicity against CAR T cells was long-lived (Fig. 20) and levels of CD69 on OTi CD8 T cells was followed (Fig. 21).

[00403] This experiment demonstrated activation of target cells with no significant cytotoxicity on the killer cells. It is possible that Killer Cells activation might be suboptimal under these conditions and use of primary T cells could include fresh, syngeneic cells for improved activation.

Example 10

[00404] 1E6 T cell clone is a well-established CD8⁺ specific for the major β -cell autoantigen, preproinsulin (PPI). The 1E6 clone mediates β -cell-specific killing via recognition of a highly distinctive HLA A*0201-presented signal peptide epitope (PPI15-24) that exhibits glucose-dependent presentation on the surface of human β -cells (9). The inventors generated and tested a split pMHC-CAR that targets 1E6 T-cells through A*0201+peptides. Effective killing of 1E6 T-cells was observed (Fig. 29)

Example 11

[00405] CALs comprising ovalbumin MiHA (against OTi or OTii) were constructed (Fig. 26). pMHC tetramer + FITC (adaptor) binds to the target cells (OTi) in a dose dependent fashion, while OTii specific tetramer does not (Fig. 27). Binding of Jurkat+ pMHC to OTi cells does not change Jurkat live count (Fig. 28B and 30). However, human CD8 CAR T were cytotoxic with high specificity (Fig. 31 and 32). Cytotoxicity was not seen with ctrl tetramer. No cytotoxicity was seen on killer CAR T cells or bystander CD4 T Cells after co-culturing with pMHC and splenocytes.

What is claimed herein is:

1. A composition, comprising:
 - a. a TCR recognition domain; and
 - one or both of:
 - b. an intracellular signaling domain; and
 - c. a first-type biomolecular interaction domain.
2. A composition comprising:
 - a. a first polypeptide comprising at least a portion of a TCR recognition domain and a first-type biomolecular interaction domain; and
 - b. a signaling polypeptide comprising a second-type biomolecular interaction domain and an intracellular signaling domain;wherein the first-type and second-type biomolecular interaction domains bind specifically to each other.
3. A composition comprising:
 - a. a first polypeptide comprising at least a portion of a TCR recognition domain and a first-type biomolecular interaction domain; and
 - b. a recognition polypeptide comprising a second recognition domain and a third-type biomolecular interaction domain;wherein the first-type and third-type biomolecular interaction domains bind specifically to each other.
4. A composition comprising:
 - a. a first polypeptide comprising at least a portion of a TCR recognition domain and a first-type biomolecular interaction domain; and
 - b. a signaling polypeptide comprising a second-type biomolecular interaction domain and an intracellular signaling domain; and
 - c. a recognition polypeptide comprising a second recognition domain and a third-type biomolecular interaction domain;wherein the second-type and third-type biomolecular interaction domains compete for binding to the first-type biomolecular interaction domain.
5. The composition of any of claims 3-4, wherein the third-type biomolecular interaction domain and first-type biomolecular interaction domain have a higher affinity for each other than the second-type biomolecular interaction domain and first-type biomolecular interaction domain.
6. A composition comprising:
 - a. a first polypeptide comprising at least a portion of a TCR recognition domain and a first-type biomolecular interaction domain;

- b. a signaling polypeptide comprising a second-type biomolecular interaction domain, a fourth-type biomolecular interaction domain, and an intracellular signaling domain; and
 - c. a recognition polypeptide comprising a second recognition domain and a fifth-type biomolecular interaction domain;
- wherein the first-type biomolecular interaction domain and the second-type biomolecular interaction domain bind specifically to each other; and
- wherein the fourth-type biomolecular interaction domain and the fifth-type biomolecular interaction domain bind specifically to each other.
7. The composition of claim 6, wherein the fourth-type biomolecular interaction domain and fifth-type biomolecular interaction domain have a weaker affinity than the second-type biomolecular interaction domain and first-type protein interaction domain.
 8. The composition of any of claims 6-7, wherein the first polypeptide further comprises a sixth-type biomolecular interaction domain and the recognition polypeptide further comprises a seventh-type biomolecular interaction domain which bind specifically to each other.
 9. The composition of any of claims 2-8, wherein the first polypeptide comprises the entire TCR recognition domain.
 10. The composition of any of claims 2-8, wherein the TCR recognition domain comprises at least two separate polypeptide sequences, the first polypeptide comprises at least one of the separate polypeptide sequences of the TCR recognition domain, and the first polypeptide is bound to or complexed with a second or further polypeptide sequences of the TCR recognition domain to form a TCR recognition domain.
 11. The composition of any of claims 1-10, wherein the TCR recognition domain comprises a non-polypeptide component.
 12. The composition of any of claims 3-11, wherein the second recognition domain is specific for a target that is not recognized by the TCR recognition domain.
 13. The composition of any of claims 3-12, wherein the second recognition domain is specific for a target that is found on a healthy and/or non-target cell and not on a diseased and/or target cell.
 14. The composition of any of the preceding claims, wherein the TCR recognition domain comprises a MHC (Major Histocompatibility Complex); a MHC-peptide complex; featureless peptide MHC; or a MHC-peptide fusion.
 15. The composition of claim 14, wherein the peptide is a human or non-human peptide.
 16. The composition of any of claims 14-15, wherein the peptide is a Minor Histocompatibility Antigen (MiHA).

17. The composition of any of claims 14-16, wherein the MHC is a monomer, dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.
18. The composition of any of claims 14-17, wherein the MHC-peptide complex is a monomer, dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.
19. The composition of any of claims 14-17, wherein the MHC-peptide fusion is a monomer, dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.
20. The composition of any of claims 14-17, wherein the MHC is a dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.
21. The composition of any of claims 14-17, wherein the MHC-peptide complex is a dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.
22. The composition of any of claims 14-17, wherein the MHC-peptide fusion is a dimer, trimer, tetramer, pentamer, dextramer or other oligomer form.
23. The composition of any of claims 14-22, wherein the MHC is a MHC class I or a MHC class II.
24. The composition of any of claims 1-13, wherein the TCR recognition domain comprises a CD1 domain or a CD1 domain-ligand complex or fusion.
25. The composition of claim 24, wherein the CD1 is CD1d.
26. The composition of any of the preceding claims, wherein the biomolecular interaction domains are found on an extracellular portion of the respective polypeptides.
27. The composition of any of the preceding claims,
 - a. wherein the biomolecular interaction domain(s) is a leucine zipper, or any binding pair of biomolecular interaction domains are collectively a pair of leucine zippers;
 - b. wherein the biomolecular interaction domain(s) is a BZip (RR) and/or a AZip (EE), or any binding pair of biomolecular interaction domains are collectively a BZip (RR) and a AZip (EE);
 - c. wherein the biomolecular interaction domain(s) is a PSD95-Dlg1-zo-1 (PDZ) domain;
 - d. wherein the biomolecular interaction domain(s) is a streptavidin and/or a streptavidin binding biomolecular (SBP) or any binding pair of biomolecular interaction domains are collectively a streptavidin and a streptavidin binding biomolecular (SBP);
 - e. wherein the biomolecular interaction domain(s) is a FKBP-binding domain of mTOR (FRB) and/or a FK506 binding biomolecular (FKBP) or any binding pair of biomolecular interaction domains are collectively a FKBP-binding domain of mTOR (FRB) and a FK506 binding biomolecular (FKBP);
 - f. wherein the biomolecular interaction domain(s) is a cyclophilin-Fas fusion biomolecular (CyP-Fas) and/or a FK506 binding biomolecular (FKBP) or any

- binding pair of biomolecular interaction domains are collectively a cyclophilin-Fas fusion biomolecular (CyP-Fas) and a FK506 binding biomolecular (FKBP);
- g. wherein the biomolecular interaction domain(s) is a calcineurin A (CNA) and/or a FK506 binding biomolecular (FKBP) or any binding pair of biomolecular interaction domains are collectively a calcineurin A (CNA) and a FK506 binding biomolecular (FKBP);
 - h. wherein the biomolecular interaction domain(s) is a gibberellin insensitive (GIA) and/or a gibberellin insensitive dwarf1 (GID1) or any binding pair of biomolecular interaction domains are collectively a gibberellin insensitive (GIA) and a gibberellin insensitive dwarf1 (GID1);
 - i. wherein the biomolecular interaction domain(s) is a Snap-tag and/or a Halo tag, or any binding pair of biomolecular interaction domains are collectively a Snap-tag and a Halo tag;
 - j. wherein the biomolecular interaction domain(s) is a T14-3-3-cdeltaC and/or a C-Terminal peptides of PMA2 (CT52), or any binding pair of biomolecular interaction domains are collectively a T14-3-3-cdeltaC and a C-Terminal peptides of PMA2 (CT52);
 - k. wherein the biomolecular interaction domain(s) is a PYL and/or a ABI, or any binding pair of biomolecular interaction domains are collectively a PYL and a ABI;
 - l. wherein the biomolecular interaction domain(s) is a nucleotide tag and/or a zinc finger domain, or any binding pair of biomolecular interaction domains are collectively a nucleotide tag and a zinc finger domain;
 - m. wherein the biomolecular interaction domain(s) is a nucleotide tag, or any binding pair of biomolecular interaction domains are collectively a pair of nucleotide tags;
 - n. wherein the biomolecular interaction domain(s) is a Fluorescein isothiocyanate (FITC) and/or a FITC binding biomolecular or any binding pair of protein interaction domains are collectively a FITC and a FITC binding protein; and/or
 - o. wherein the protein interaction domain(s) is a (R)-Phycoerythrin (R-PE/ PE) and/or a R-PE/PE binding protein or any binding pair of protein interaction domains are collectively a (R)-Phycoerythrin (R-PE/ PE) and a R-PE/PE binding protein.
28. The composition of claim 27, wherein the nucleotide tag is a DNA tag or dsDNA tag.
29. The composition of any of the preceding claims, wherein the intracellular signaling domain comprises or is a signaling domain from one or more proteins selected from the group consisting of:

TCR ζ , FcR γ , FcR β , CD3 γ ; CD35; CD3 ζ ; CD3C; CD22; CD79a; CD79b; CD66d; CARD11; CD2; CD7; CD27; CD28; CD30; CD40; CD54 (ICAM); CD83; CD134

(OX40); CD137 (4-1BB); CD150 (SLAMF1); CD152 (CTLA4); CD223 (LAG3); CD270 (HVEM); CD273 (PD-L2); CD274 (PD-L1); CD278 (ICOS); DAP10; LAT; KD2C SLP76; TRIM; and ZAP70.

30. A cell comprising and/or expressing the composition of any of the preceding claims.
31. A composition comprising a first polypeptide of any of the preceding claims and a cell expressing or comprising the signaling polypeptide of any of the preceding claims.
32. The cell or composition of any of claims 30-31, wherein the TCR recognition domain comprises a MHC allogeneic, autologous, or xenogeneic to the cell.
33. The cell or composition of any of claims 30-32, wherein the TCR recognition domain comprises a synthetic MHC.
34. The cell or composition of any of claims 30-33, wherein the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is allogeneic, autologous, or xenogeneic to the cell.
35. The cell or composition of any of claims 30-34, wherein the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is synthetic.
36. The cell or composition of any of claims 30-35, wherein the cell is a NK cell, dendritic cell, regulatory T cell, or effector T cell.
37. The cell or composition of any of claims 30-36, wherein the cell is engineered to express one or more of the polypeptide(s) of the composition.
38. The cell or composition of any of claims 30-37, wherein the cell is engineered to express the signaling polypeptide of the composition.
39. The cell or composition of any of claims 30-38, wherein the cell is further engineered to knockout or knockdown the native MHCI/II.
40. The cell or composition of any of claims 30-39, wherein the cell is further engineered to knockdown the native MHCI/II expressed on the cell surface.
41. A chimeric antigen receptor (CAR) comprising:
 - a. an anti-CD127 and/or anti-CD45RO recognition domain;
 - b. an intracellular signaling domain.
42. A composition comprising:
 - a first polypeptide comprising:
 - a. an anti-CD127 and/or anti-CD45RO recognition domain;
 - b. a first-type protein interaction domain; and
 - a second polypeptide comprising:
 - c. a second-type protein interaction domain; and
 - d. an intracellular signaling domain;

wherein the first-type protein interaction domain and the second-type protein interaction domain bind specifically to each other.

43. A composition comprising:

a first polypeptide comprising:

- a. an anti-CD127 recognition domain;
- b. a first-type protein interaction domain;

a second polypeptide comprising:

- c. an anti-CD45RO recognition domain;
- d. a fifth-type protein interaction domain; and

a third polypeptide comprising:

- e. a second-type and a fourth-type protein interaction domain; and
- f. an intracellular signaling domain;

wherein the first-type protein interaction domain and the second-type protein interaction domain bind specifically to each other; and

wherein the fourth-type protein interaction domain and the fifth-type protein interaction domain bind specifically to each other.

44. A cell comprising the CAR or composition of any of claims 41-43.

45. A method of treating or preventing an autoimmune disease or condition; T cell mediated inflammation or immune response; malignant T cell condition; transplant rejection; or GvHD in a subject in need thereof, the method comprising administering to the subject a composition and/or cell of any of the preceding claims.

46. The method of claim 45, wherein the TCR recognition domain comprises a MHC allogeneic to the subject.

47. The method of claim 45, wherein the TCR recognition domain comprises a MHC autologous to the transplant cells.

48. The method of claim 45, wherein the TCR recognition domain comprises a MHC xenogeneic to the transplant cells.

49. The method of claim 45, wherein the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is allogeneic to the subject.

50. The method of claim 45, wherein the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is autologous to the transplant cells.

51. The method of claim 45, wherein the TCR recognition domain comprises a MHC and a peptide, wherein the peptide is autologous to the transplant cells.

52. The method of any of claims 45-51, wherein the MHC and/or the peptide is synthetic.

53. The method of any of claims 45-52, wherein the transplant is any human or non-human cell, tissue, or organ.
54. The method of any of claims 45-53, wherein the transplant is an allogeneic hematopoietic stem cell or solid organ transplantation.
55. The method of any of claims 45-52, wherein the malignant T cell condition is T cell acute lymphoblastic leukemia or T cell lymphoblastic lymphoma.
56. The method of any of claims 45-52, wherein the autoimmune disease is type 1 diabetes, vitiligo, multiple sclerosis, alopecia, celiac disease, pemphigus, rheumatoid arthritis, or scleroderma.
57. The method of any of claims 45-52, wherein the autoimmune disease is thyroiditis, type 1 diabetes mellitus, Hashimoto's thyroiditis, Graves' disease, celiac disease, multiple sclerosis, Guillain-Barre syndrome, Addison's disease, and Raynaud's phenomenon, Goodpasture's disease, arthritis (rheumatoid arthritis such as acute arthritis, chronic rheumatoid arthritis, gout or gouty arthritis, acute gouty arthritis, acute immunological arthritis, chronic inflammatory arthritis, degenerative arthritis, type II collagen-induced arthritis, infectious arthritis, Lyme arthritis (e.g., post treatment Lyme disease syndrome), proliferative arthritis, psoriatic arthritis, Still's disease, vertebral arthritis, and juvenile-onset rheumatoid arthritis, arthritis chronica progrediente, arthritis deformans, polyarthritis chronica primaria, reactive arthritis, and ankylosing spondylitis), palindromic arthritis, inflammatory hyperproliferative skin diseases, psoriasis such as plaque psoriasis, guttate psoriasis, pustular psoriasis, and psoriasis of the nails, atopy including atopic diseases such as hay fever and Job's syndrome, dermatitis including contact dermatitis, chronic contact dermatitis, exfoliative dermatitis, allergic dermatitis, allergic contact dermatitis, dermatitis herpetiformis, nummular dermatitis, seborrheic dermatitis, non-specific dermatitis, primary irritant contact dermatitis, and atopic dermatitis, x-linked hyper IgM syndrome, allergic intraocular inflammatory diseases, urticaria such as chronic allergic urticaria and chronic idiopathic urticaria, including chronic autoimmune urticaria, myositis, polymyositis/dermatomyositis, juvenile dermatomyositis, toxic epidermal necrolysis, scleroderma (including systemic scleroderma), sclerosis such as systemic sclerosis, multiple sclerosis (MS) such as spino-optical MS, primary progressive MS (PPMS), and relapsing remitting MS (RRMS), progressive systemic sclerosis, atherosclerosis, arteriosclerosis, sclerosis disseminata, ataxic sclerosis, neuromyelitis optica (NMO), inflammatory bowel disease (IBD) (for example, Crohn's disease, autoimmune-mediated gastrointestinal diseases, colitis such as ulcerative colitis, colitis ulcerosa, microscopic colitis, collagenous colitis, colitis polyposa, necrotizing enterocolitis, and transmural colitis, and autoimmune inflammatory bowel disease), bowel inflammation, pyoderma gangrenosum, erythema nodosum, primary sclerosing cholangitis, respiratory distress syndrome, including

adult or acute respiratory distress syndrome (ARDS), meningitis, inflammation of all or part of the uvea, iritis, choroiditis, an autoimmune hematological disorder, rheumatoid spondylitis, rheumatoid synovitis, hereditary angioedema, cranial nerve damage as in meningitis, herpes gestationis, pemphigoid gestationis, pruritis scroti, autoimmune premature ovarian failure, sudden hearing loss due to an autoimmune condition, IgE-mediated diseases such as anaphylaxis and allergic and atopic rhinitis, encephalitis such as Rasmussen's encephalitis and limbic and/or brainstem encephalitis, uveitis, such as anterior uveitis, acute anterior uveitis, granulomatous uveitis, nongranulomatous uveitis, phacoantigenic uveitis, posterior uveitis, or autoimmune uveitis, glomerulonephritis (GN) with and without nephrotic syndrome such as chronic or acute glomerulonephritis such as primary GN, immune-mediated GN, membranous GN (membranous nephropathy), idiopathic membranous GN or idiopathic membranous nephropathy, membrano- or membranous proliferative GN (MPGN), including Type I and Type II, and rapidly progressive GN, proliferative nephritis, autoimmune polyglandular endocrine failure, balanitis including balanitis circumscripta plasmacellularis, balanoposthitis, erythema annulare centrifugum, erythema dyschromicum perstans, erythema multiform, granuloma annulare, lichen nitidus, lichen sclerosus et atrophicus, lichen simplex chronicus, lichen spinulosus, lichen planus, lamellar ichthyosis, epidermolytic hyperkeratosis, premalignant keratosis, pyoderma gangrenosum, allergic conditions and responses, allergic reaction, eczema including allergic or atopic eczema, asteatotic eczema, dyshidrotic eczema, and vesicular palmoplantar eczema, asthma such as asthma bronchiale, bronchial asthma, and auto-immune asthma, conditions involving infiltration of T cells and chronic inflammatory responses, immune reactions against foreign antigens such as fetal A-B-O blood groups during pregnancy, chronic pulmonary inflammatory disease, autoimmune myocarditis, leukocyte adhesion deficiency, lupus, including lupus nephritis, lupus cerebritis, pediatric lupus, non-renal lupus, extra-renal lupus, discoid lupus and discoid lupus erythematosus, alopecia lupus, systemic lupus erythematosus (SLE) such as cutaneous SLE or subacute cutaneous SLE, neonatal lupus syndrome (NLE), and lupus erythematosus disseminatus, juvenile onset (Type I) diabetes mellitus, including pediatric insulin-dependent diabetes mellitus (IDDM), adult onset diabetes mellitus (Type II diabetes), autoimmune diabetes, idiopathic diabetes insipidus, diabetic retinopathy, diabetic nephropathy, diabetic large-artery disorder, immune responses associated with acute and delayed hypersensitivity mediated by cytokines and T-lymphocytes, sarcoidosis, granulomatosis including lymphomatoid granulomatosis, Wegener's granulomatosis, agranulocytosis, vasculitides, including vasculitis, large-vessel vasculitis (including polymyalgia rheumatica and giant-cell (Takayasu's) arteritis), medium-vessel vasculitis (including Kawasaki's disease and polyarteritis nodosa/periarthritis nodosa), microscopic polyarteritis, immunovascularitis, CNS vasculitis,

cutaneous vasculitis, hypersensitivity vasculitis, necrotizing vasculitis such as systemic necrotizing vasculitis, and ANCA-associated vasculitis, such as Churg-Strauss vasculitis or syndrome (CSS) and ANCA-associated small-vessel vasculitis, temporal arteritis, autoimmune aplastic anemia, Coombs positive anemia, Diamond Blackfan anemia, hemolytic anemia or immune hemolytic anemia including autoimmune hemolytic anemia (AIHA), pernicious anemia (anemia perniciosa), Addison's disease, pure red cell anemia or aplasia (PRCA), Factor VIII deficiency, hemophilia A, autoimmune neutropenia, pancytopenia, leukopenia, diseases involving leukocyte diapedesis, CNS inflammatory disorders, multiple organ injury syndrome such as those secondary to septicemia, trauma or hemorrhage, antigen-antibody complex-mediated diseases, anti-glomerular basement membrane disease, anti-phospholipid antibody syndrome, allergic neuritis, Behcet's disease/syndrome, Castleman's syndrome, Goodpasture's syndrome, Reynaud's syndrome, Sjogren's syndrome, Stevens-Johnson syndrome, pemphigoid such as pemphigoid bullous and skin pemphigoid, pemphigus (including pemphigus vulgaris, pemphigus foliaceus, pemphigus mucus-membrane pemphigoid, and pemphigus erythematosus), autoimmune polyendocrinopathies, Reiter's disease or syndrome, an immune complex disorder such as immune complex nephritis, antibody-mediated nephritis, polyneuropathies, chronic neuropathy such as IgM polyneuropathies or IgM-mediated neuropathy, and autoimmune or immune-mediated thrombocytopenia such as idiopathic thrombocytopenic purpura (ITP) including chronic or acute ITP, scleritis such as idiopathic cerato-scleritis, episcleritis, autoimmune disease of the testis and ovary including autoimmune orchitis and oophoritis, primary hypothyroidism, hypoparathyroidism, autoimmune endocrine diseases including thyroiditis such as autoimmune thyroiditis, Hashimoto's disease, chronic thyroiditis (Hashimoto's thyroiditis), or subacute thyroiditis, idiopathic hypothyroidism, Grave's disease, polyglandular syndromes such as autoimmune polyglandular syndromes (or polyglandular endocrinopathy syndromes), paraneoplastic syndromes, including neurologic paraneoplastic syndromes such as Lambert-Eaton myasthenic syndrome or Eaton-Lambert syndrome, stiff-man or stiff-person syndrome, encephalomyelitis such as allergic encephalomyelitis or encephalomyelitis allergica and experimental allergic encephalomyelitis (EAE), myasthenia gravis such as thymoma-associated myasthenia gravis, cerebellar degeneration, neuromyotonia, opsoclonus or opsoclonus myoclonus syndrome (OMS), and sensory neuropathy, multifocal motor neuropathy, Sheehan's syndrome, autoimmune hepatitis, lupoid hepatitis, giant-cell hepatitis, autoimmune chronic active hepatitis, lymphoid interstitial pneumonitis (LIP), bronchiolitis obliterans (non-transplant) vs NSIP, Guillain-Barre syndrome, Berger's disease (IgA nephropathy), idiopathic IgA nephropathy, linear IgA dermatosis, acute febrile neutrophilic dermatosis, subcorneal pustular dermatosis, transient acantholytic dermatosis, cirrhosis such

as primary biliary cirrhosis and pneumonocirrhosis, autoimmune enteropathy syndrome, Celiac or Coeliac disease, celiac sprue (gluten enteropathy), refractory sprue, idiopathic sprue, cryoglobulinemia, amyotrophic lateral sclerosis (ALS; Lou Gehrig's disease), coronary artery disease, autoimmune ear disease such as autoimmune inner ear disease (AIED), autoimmune hearing loss, polychondritis such as refractory or relapsed or relapsing polychondritis, pulmonary alveolar proteinosis, Cogan's syndrome/nonsyphilitic interstitial keratitis, Bell's palsy, Sweet's disease/syndrome, rosacea autoimmune, zoster-associated pain, amyloidosis, a non-cancerous lymphocytosis, a primary lymphocytosis, which includes monoclonal B cell lymphocytosis (e.g., benign monoclonal gammopathy and monoclonal gammopathy of undetermined significance, MGUS), peripheral neuropathy, paraneoplastic syndrome, channelopathies including channelopathies of the CNS, autism, inflammatory myopathy, focal or segmental or focal segmental glomerulosclerosis (FSGS), endocrine ophthalmopathy, uveoretinitis, chorioretinitis, autoimmune hepatological disorder, fibromyalgia, multiple endocrine failure, Schmidt's syndrome, adrenalitis, gastric atrophy, presenile dementia, demyelinating diseases such as autoimmune demyelinating diseases and chronic inflammatory demyelinating polyneuropathy, Dressler's syndrome, alopecia areata, alopecia totalis, CREST syndrome (calcinosis, Raynaud's phenomenon, esophageal dysmotility, sclerodactyly, and telangiectasia), male and female autoimmune infertility, e.g., due to anti-spermatozoan antibodies, mixed connective tissue disease, Chagas' disease, rheumatic fever, recurrent abortion, farmer's lung, erythema multiforme, post-cardiotomy syndrome, Cushing's syndrome, bird-fancier's lung, allergic granulomatous angiitis, benign lymphocytic angiitis, Alport's syndrome, alveolitis such as allergic alveolitis and fibrosing alveolitis, interstitial lung disease, transfusion reaction, Sampter's syndrome, Caplan's syndrome, endocarditis, endomyocardial fibrosis, diffuse interstitial pulmonary fibrosis, interstitial lung fibrosis, pulmonary fibrosis, idiopathic pulmonary fibrosis, cystic fibrosis, endophthalmitis, erythema elevatum et diutinum, erythroblastosis fetalis, eosinophilic fasciitis, Shulman's syndrome, Felty's syndrome, cyclitis such as chronic cyclitis, heterochronic cyclitis, iridocyclitis (acute or chronic), or Fuch's cyclitis, Henoch-Schonlein purpura, SCID, sepsis, endotoxemia, post-vaccination syndromes, Evan's syndrome, autoimmune gonadal failure, Sydenham's chorea, post-streptococcal nephritis, thromboangitis obliterans, thyrotoxicosis, tabes dorsalis, chorioiditis, giant-cell polymyalgia, chronic hypersensitivity pneumonitis, keratoconjunctivitis sicca, idiopathic nephritic syndrome, minimal change nephropathy, benign familial and ischemia-reperfusion injury, transplant organ reperfusion, retinal autoimmunity, aphthae, aphthous stomatitis, arteriosclerotic disorders, aspermiogenesis, autoimmune hemolysis, Boeck's disease, enteritis allergica, erythema nodosum leprosum, idiopathic facial paralysis, chronic fatigue syndrome, febris rheumatica, Hamman-Rich's

disease, sensorineural hearing loss, ileitis regionalis, leucopenia, transverse myelitis, primary idiopathic myxedema, ophthalmia sympathica, polyradiculitis acuta, pyoderma gangrenosum, acquired splenic atrophy, vitiligo, toxic-shock syndrome, conditions involving infiltration of T cells, leukocyte-adhesion deficiency, immune responses associated with acute and delayed hypersensitivity mediated by cytokines and T-lymphocytes, diseases involving leukocyte diapedesis, multiple organ injury syndrome, antigen-antibody complex-mediated diseases, antglomerular basement membrane disease, allergic neuritis, autoimmune polyendocrinopathies, oophoritis, primary myxedema, autoimmune atrophic gastritis, rheumatic diseases, mixed connective tissue disease, nephrotic syndrome, insulinitis, polyendocrine failure, autoimmune polyglandular syndrome type I, adult-onset idiopathic hypoparathyroidism (AOIH), myocarditis, nephrotic syndrome, primary sclerosing cholangitis, acute or chronic sinusitis, ethmoid, frontal, maxillary, or sphenoid sinusitis, an eosinophil-related disorder such as eosinophilia, pulmonary infiltration eosinophilia, eosinophilia-myalgia syndrome, Löffler's syndrome, chronic eosinophilic pneumonia, tropical pulmonary eosinophilia, granulomas containing eosinophils, seronegative spondyloarthritides, polyendocrine autoimmune disease, sclerosing cholangitis, sclera, episclera, Bruton's syndrome, transient hypogammaglobulinemia of infancy, Wiskott-Aldrich syndrome, ataxia telangiectasia syndrome, angiectasis, autoimmune disorders associated with collagen disease, rheumatism, allergic hypersensitivity disorders, glomerulonephritides, reperfusion injury, ischemic re-perfusion disorder, lymphomatous tracheobronchitis, inflammatory dermatoses, dermatoses with acute inflammatory components, and autoimmune uveoretinitis (AUR).

58. The method of any of claims 45-52, wherein the T cell mediated immune response is an anti-drug specific response to a biologic, cell therapy, and/or gene therapy.
59. The method of claim 58, wherein the biologic, cell-therapy, or gene therapy is an adeno-associated virus (AAV) gene therapy, a genome editing agent, or enzyme replacement therapy.
60. The method of any of claims 45-52, wherein the disease is type 1 diabetes and the TCR recognition domain comprises sequences with at least 80% or at least 95% sequence identity to: one or more of SEQ ID NOs: 8-17; HLA-A*0201 and at least one of SEQ ID NOs: 2013-2016 and 2031-2033; or HLA-A*02:01 and at least one of SEQ ID NOs: 20128-2129.
61. The method of any of claims 45-52, wherein the disease is vitiligo and the TCR recognition domain comprises sequences with at least 80% or at least 95% sequence identity to: SEQ ID NO: 18, 19, and one of 20-22; or comprises HLA-A*0201 and SEQ ID NO: 2018; or HLA-A*0301 and SEQ ID NO: 2019; or comprises HLA-A*2402 and SEQ ID NO: 2020; or HLA-A*0101 and SEQ ID NO: 2021.
62. The method of any of claims 45-52, wherein the method is a method of treating and/or preventing GvHD and the TCR recognition domain comprises sequences with at least 80% or at least 95%

sequence identity to: HLA-A*0101 and at least one of SEQ ID NOs: 2034-2037; or HLA-B*0702 and SEQ ID NO: 2038; or HLA-B*0801 and SEQ ID NO: 2039.

63. The method of any of claims 45-52, wherein the disease is type 1 diabetes and the TCR recognition domain comprises one or more of SEQ ID NOs: 8-17; comprises HLA-A*0201 and at least one of SEQ ID NOs: 2013-2016 and 2031-2033; or comprises HLA-A*02:01 and at least one of SEQ ID NOs: 20128-2129.
64. The method of any of claims 45-52, wherein the disease is vitiligo and the TCR recognition domain comprises SEQ ID NO: 18, 19, and one of 20-22; or comprises HLA-A*0201 and SEQ ID NO: 2018; or comprises HLA-A*0301 and SEQ ID NO: 2019; or comprises HLA-A*2402 and SEQ ID NO: 2020; or comprises HLA-A*0101 and SEQ ID NO: 2021.
65. The method of any of claims 45-52, wherein the method is a method of treating and/or preventing GvHD and the TCR recognition domain comprises HLA-A*0101 and at least one of SEQ ID NOs: 2034-2037; or comprises HLA-B*0702 and SEQ ID NO: 2038; or comprises HLA-B*0801 and SEQ ID NO: 2039.

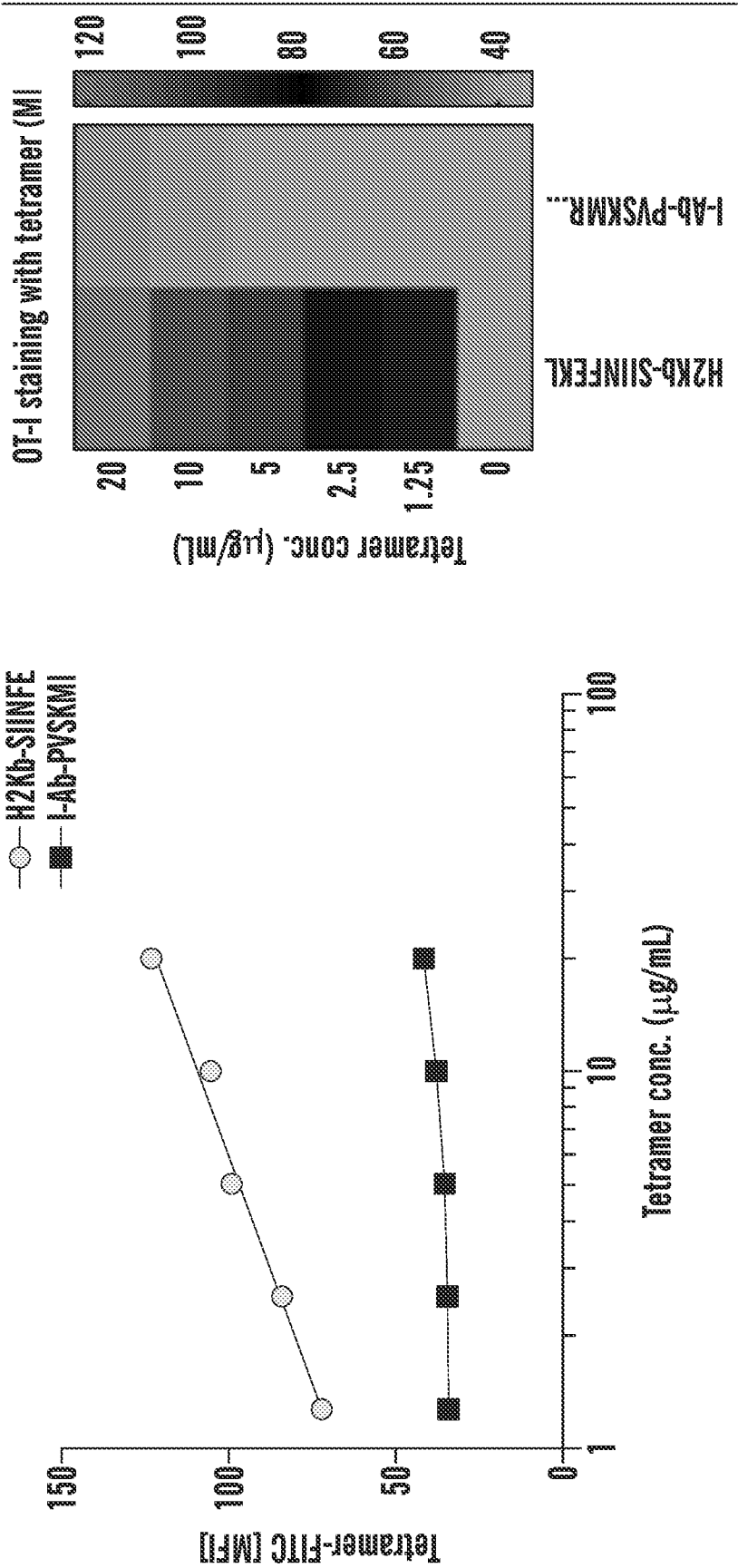


FIG. 1

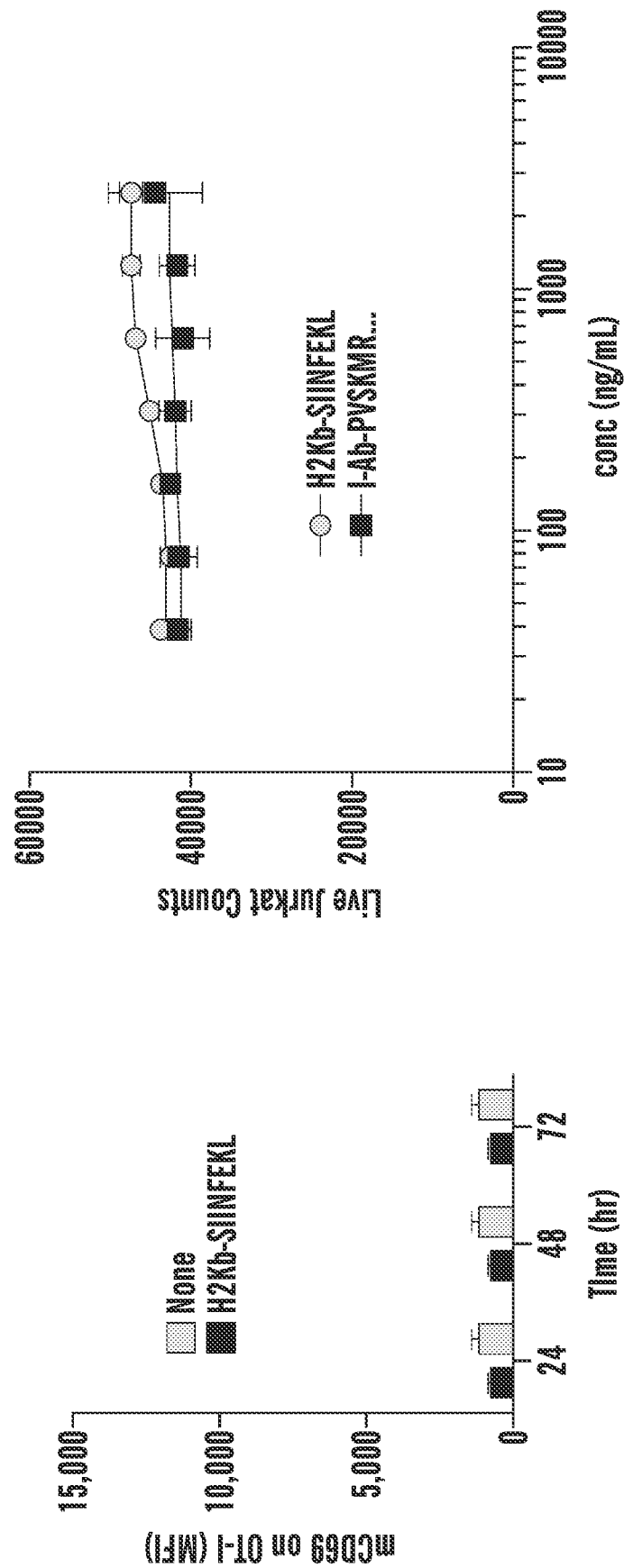
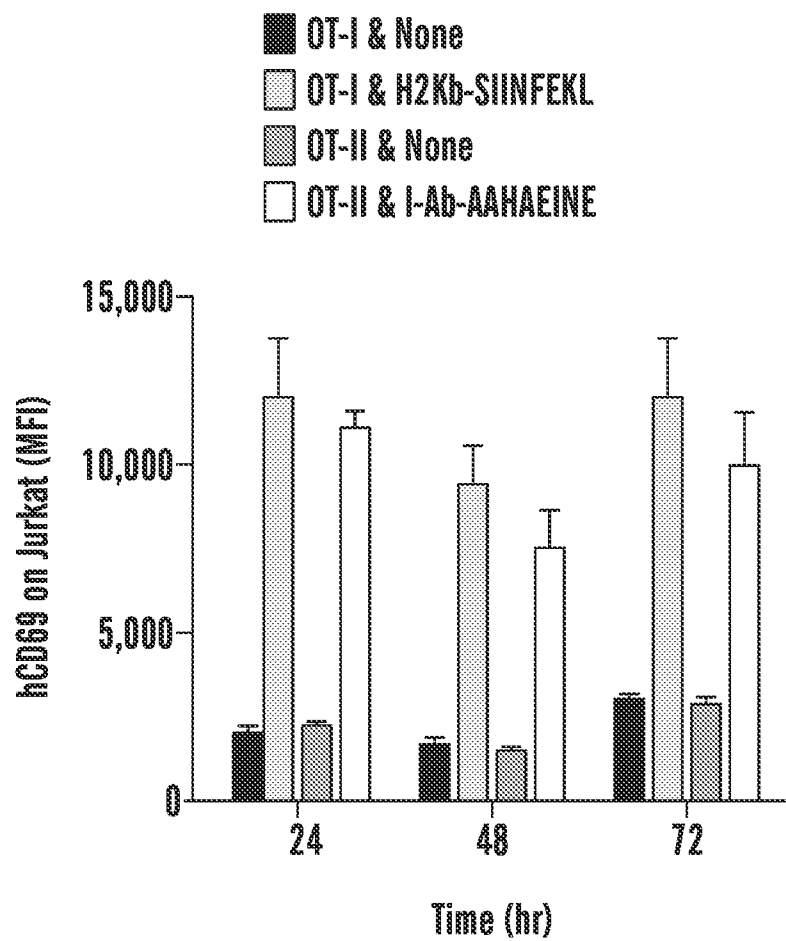
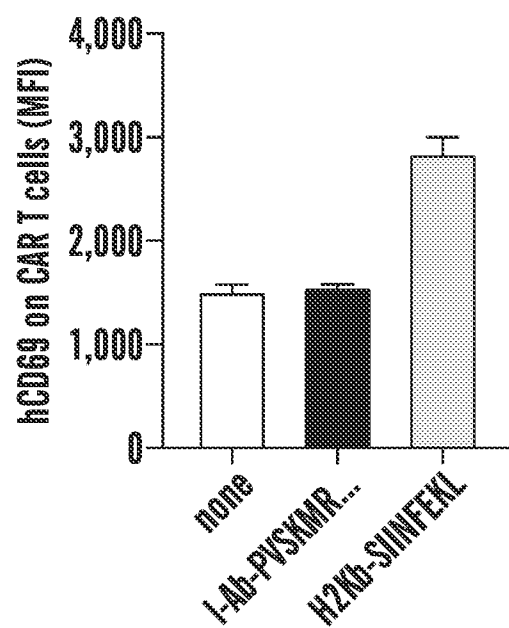
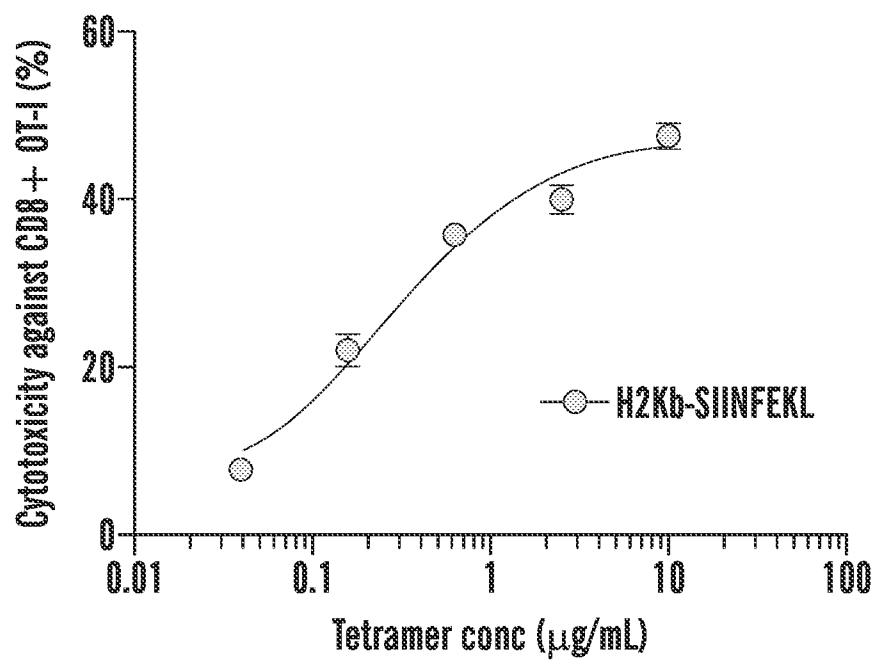


FIG. 2

**FIG. 3**

**FIG. 4**

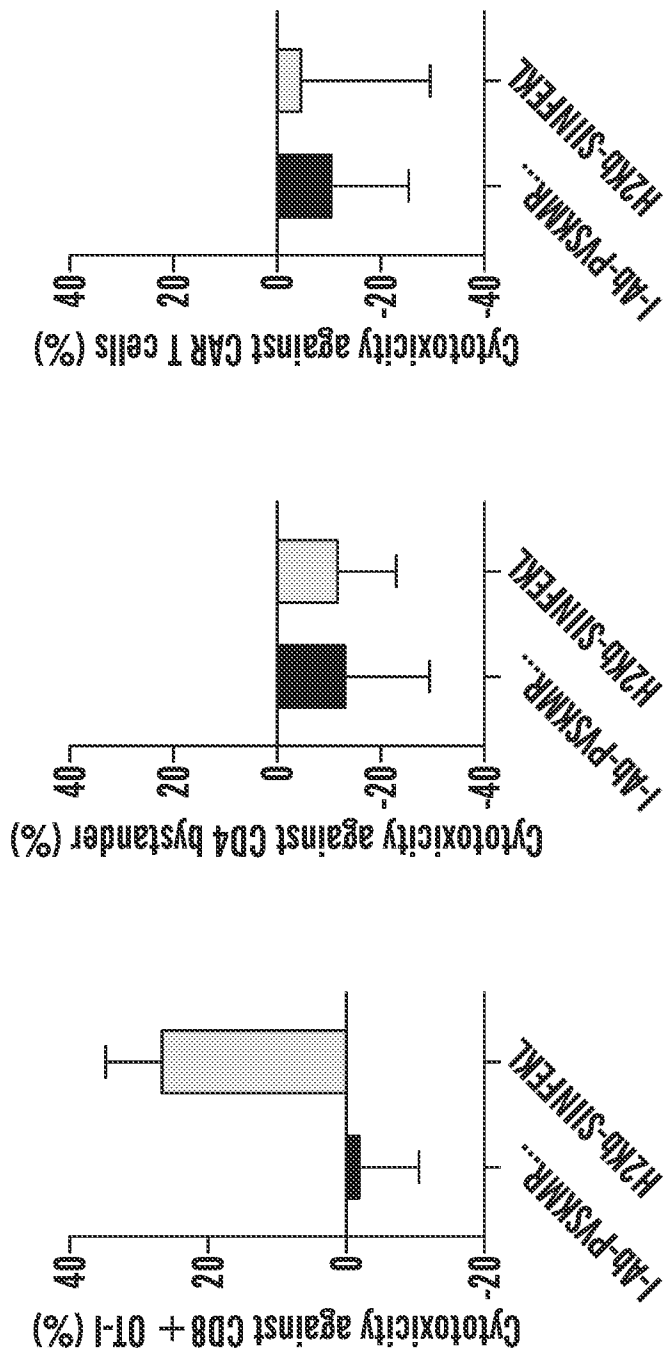


FIG. 5

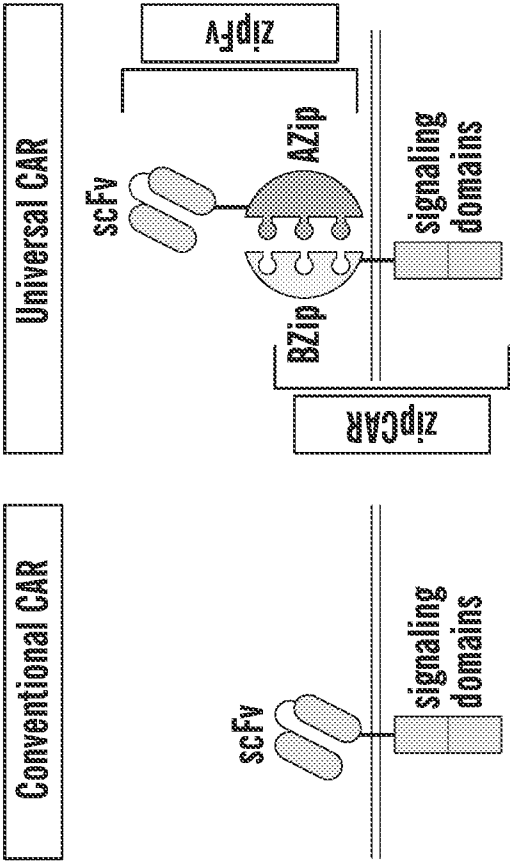


FIG. 6A

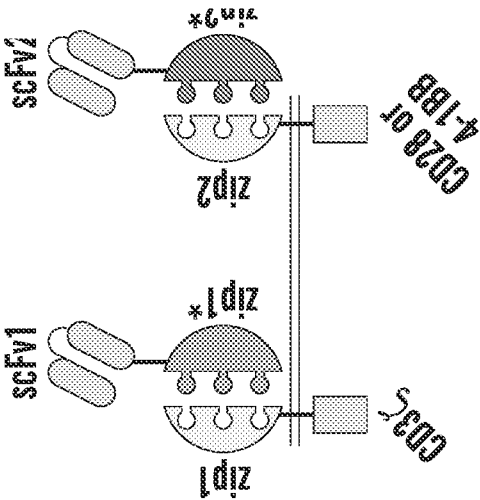


FIG. 6B

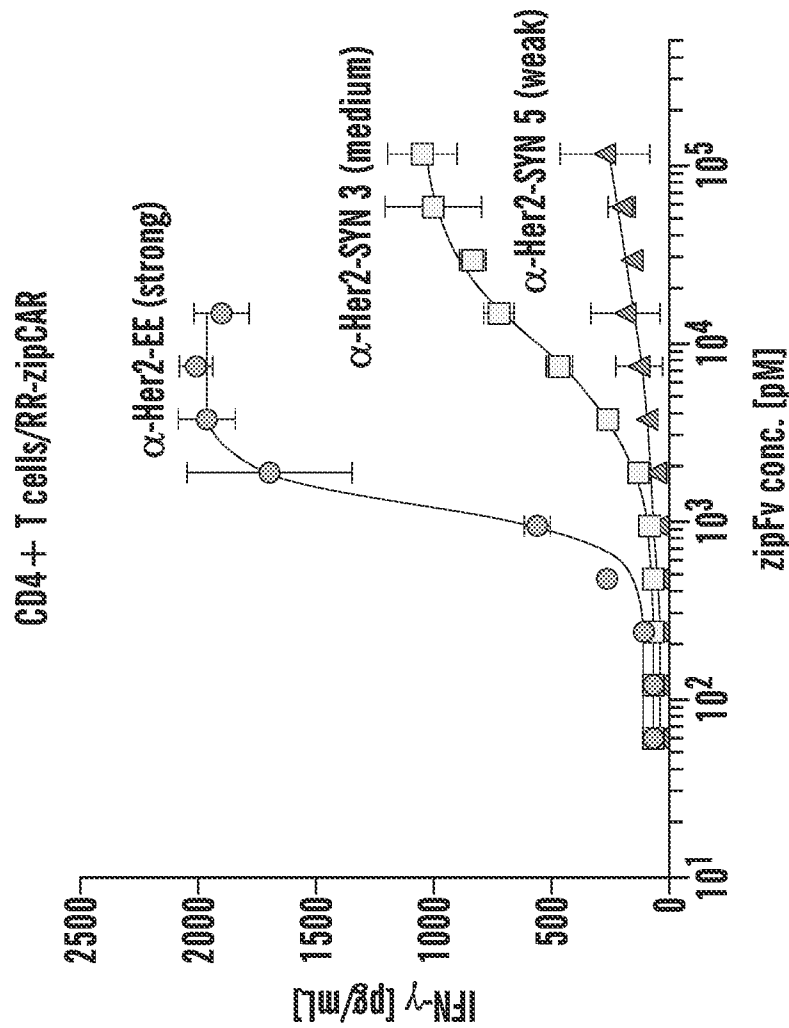
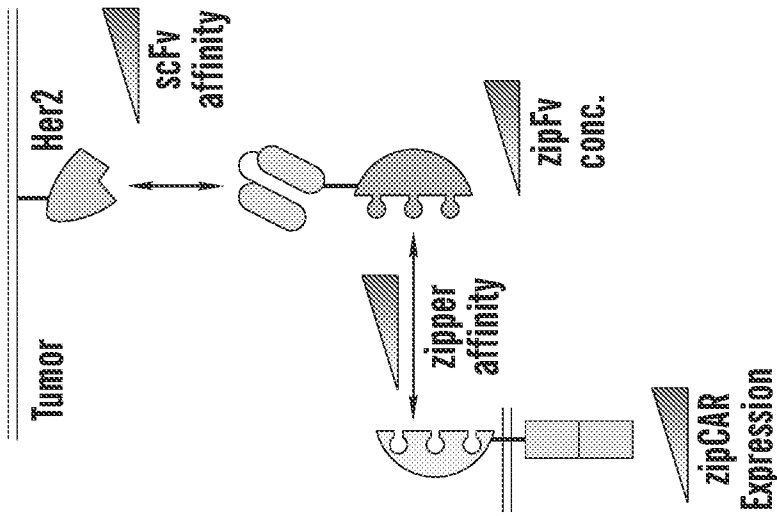


FIG. 7A



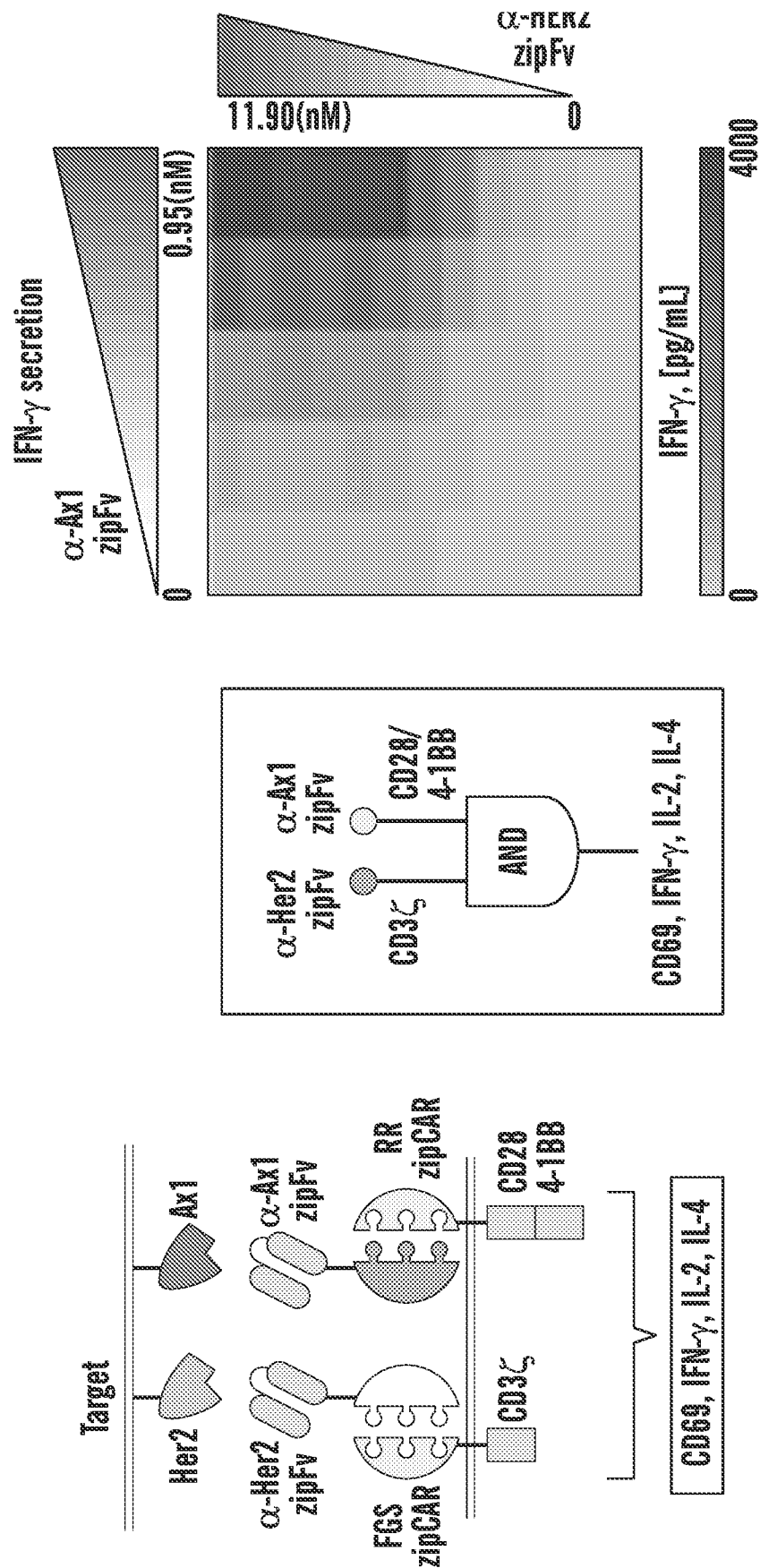


FIG. 7B

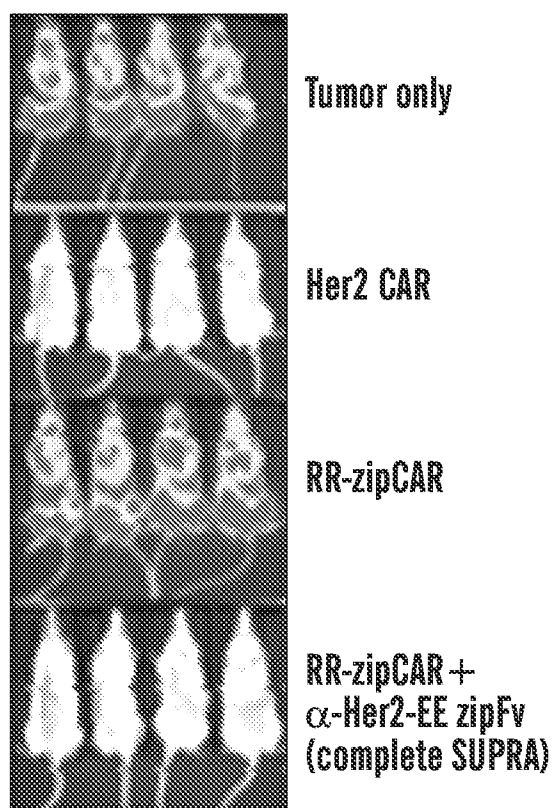


FIG. 7C

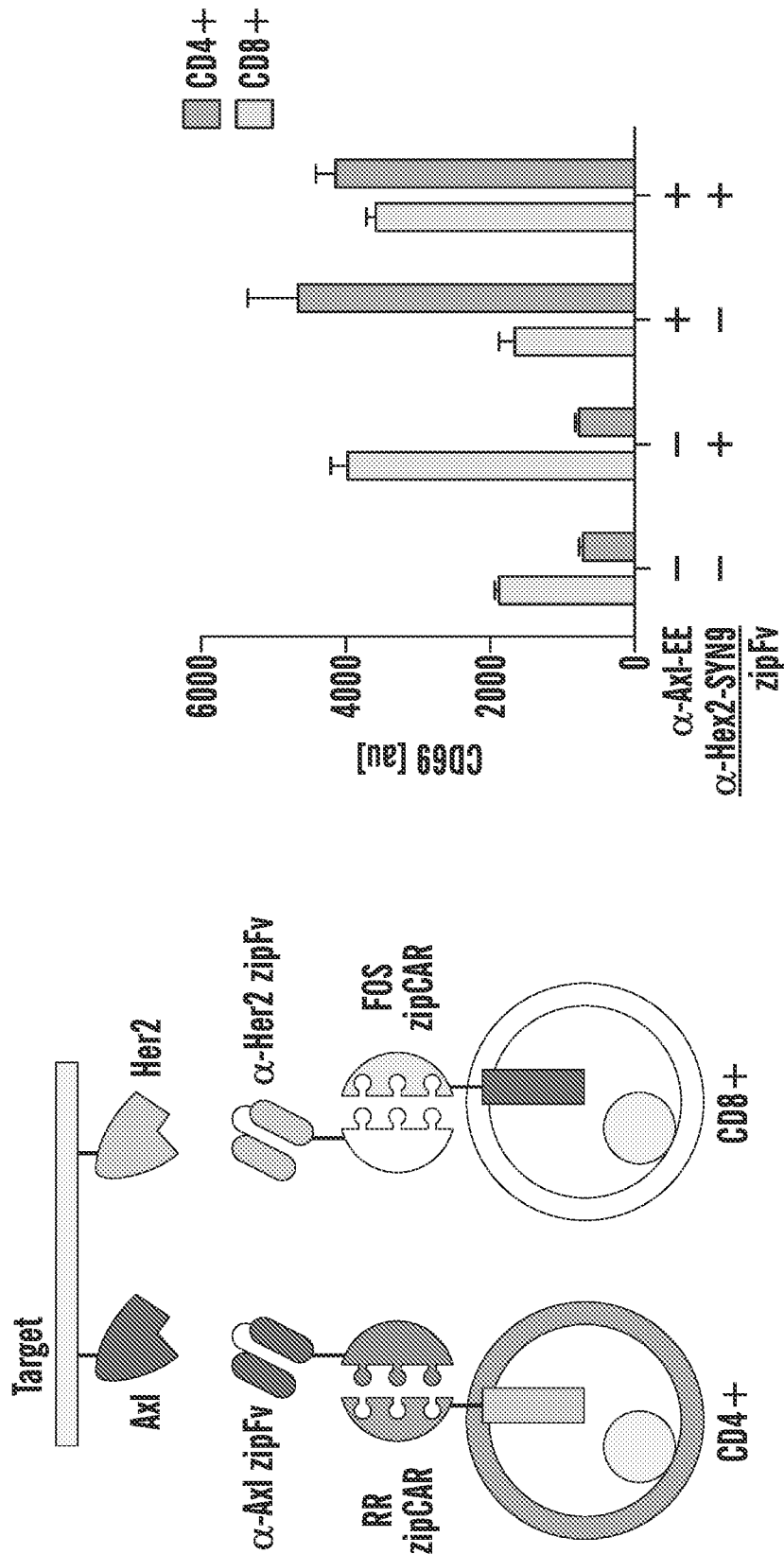


FIG. 7D

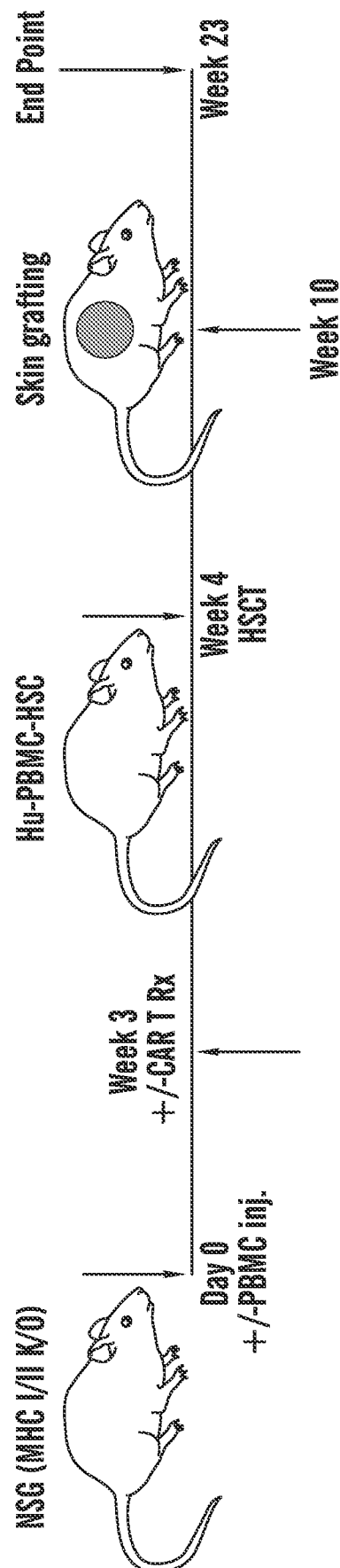
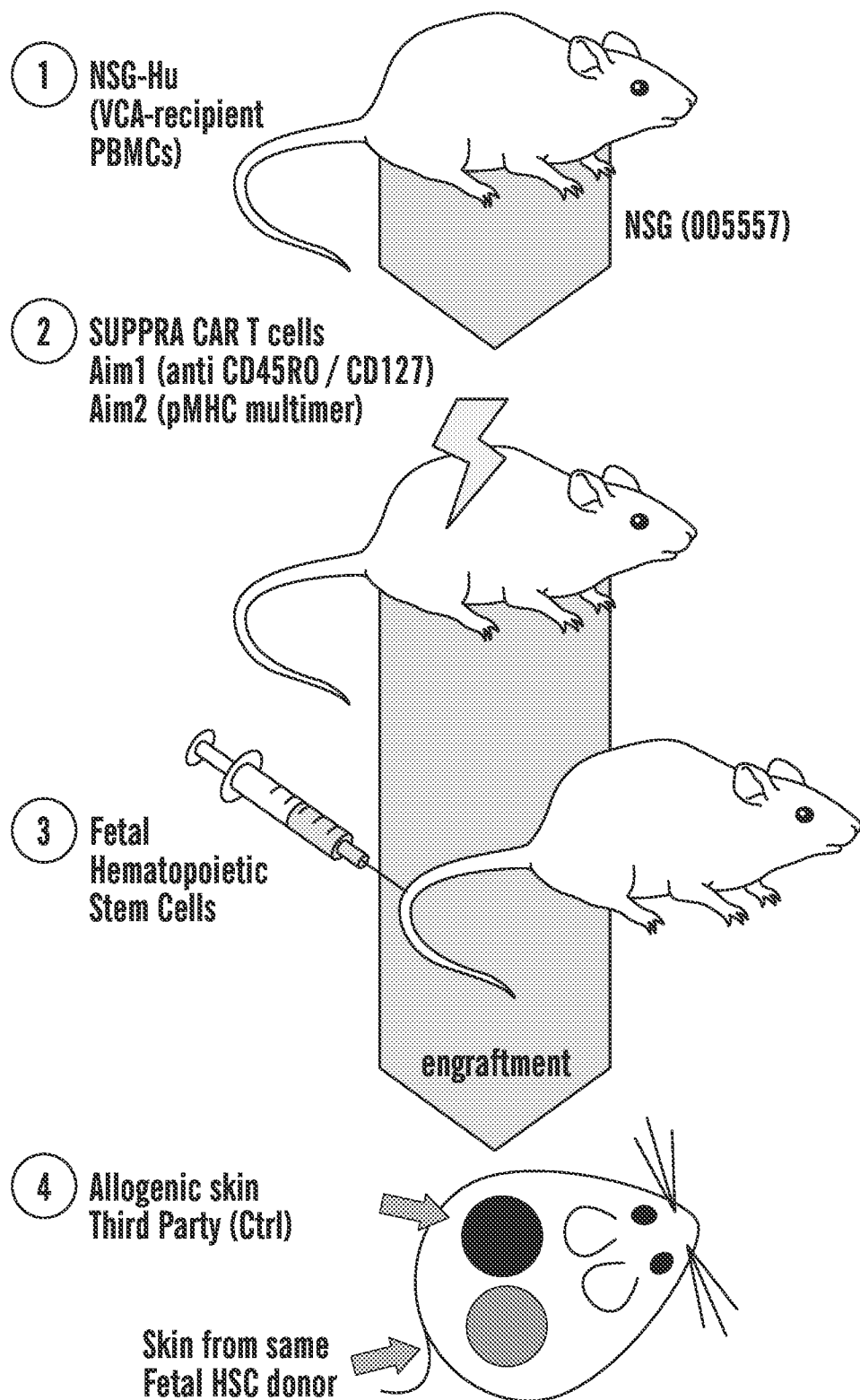


FIG. 8

**FIG. 9**

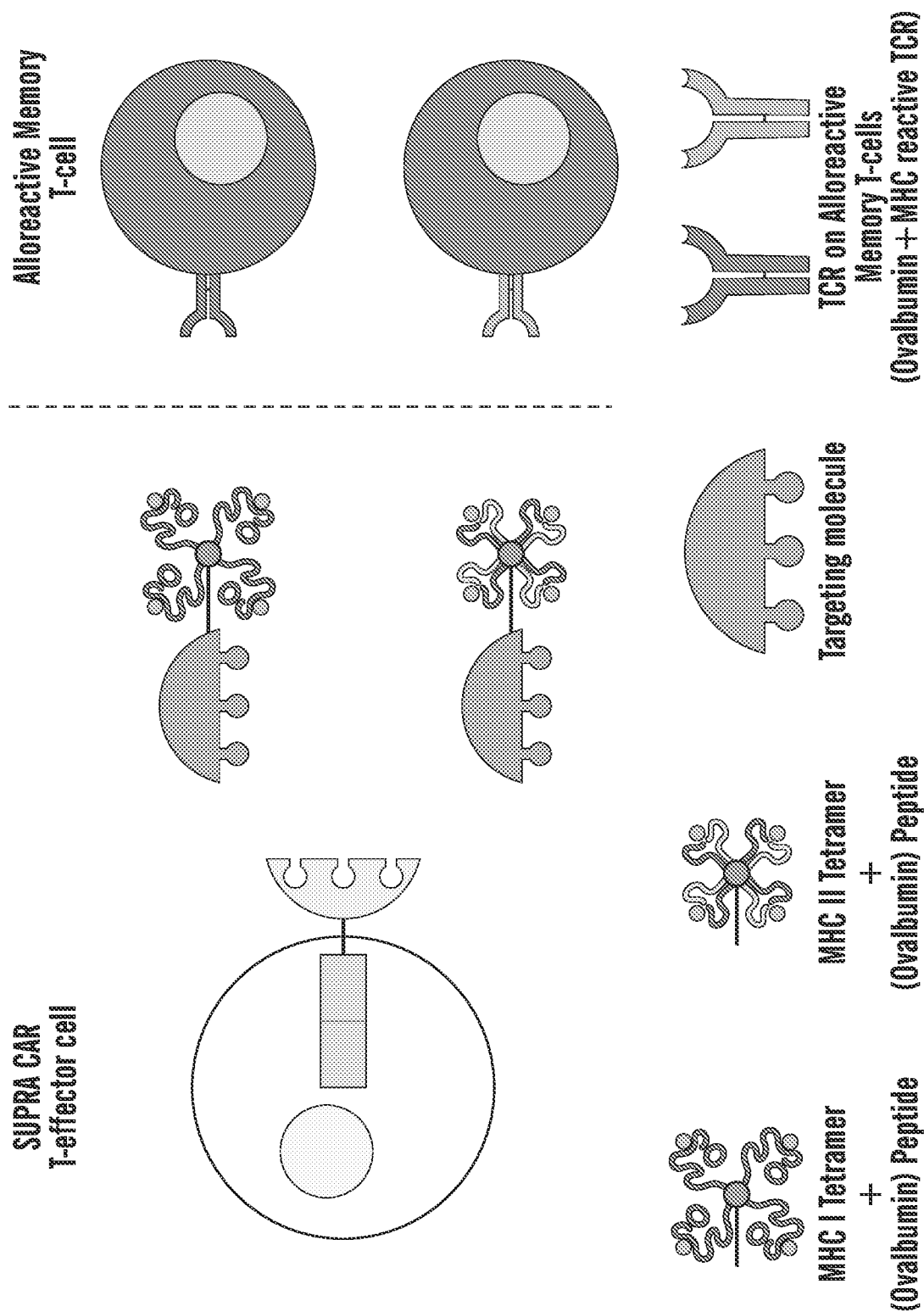


FIG. 10

OTi		OTii									
1	2	3	4	5	6	7	8	9	10	11	12
50 (A)	50 (A)	50 (A)	50 (C)	50 (C)	50 (C)	50 (B)	50 (B)	50 (B)	50 (C)	50 (C)	50 (C)
25 (A)	25 (A)	25 (A)	25 (C)	25 (C)	25 (C)	25 (B)	25 (B)	25 (B)	25 (C)	25 (C)	25 (C)
12.5	12.5	12.5	12.5	12.5	12.5	12.5	12.5	12.5	12.5	12.5	12.5
6.25	6.25	6.25	6.25	6.25	6.25	6.25	6.25	6.25	6.25	6.25	6.25
3.1	3.1	3.1	3.1	3.1	3.1	3.1	3.1	3.1	3.1	3.1	3.1
1.5	1.5	1.5	1.5	1.5	1.5	1.5	1.5	1.5	1.5	1.5	1.5
0.73	0.73	0.73	0.73	0.73	0.73	0.73	0.73	0.73	0.73	0.73	0.73
0	0	0	0	0	0	0	0	0	0	0	0

A: H2Kb-SINFEKL (for OT-I) anti-mouse CD8-PerCP-Cy5.5 (for OT-I).
B: I-Ab-HAINEA (for OT-II) anti-mouse CD107a-APC - 4 hr
C: I-Ab-PVSKMR... (negative control) anti-human CD69a-APC - 24 hr
anti-mouse CD69-APC

FIG. 11

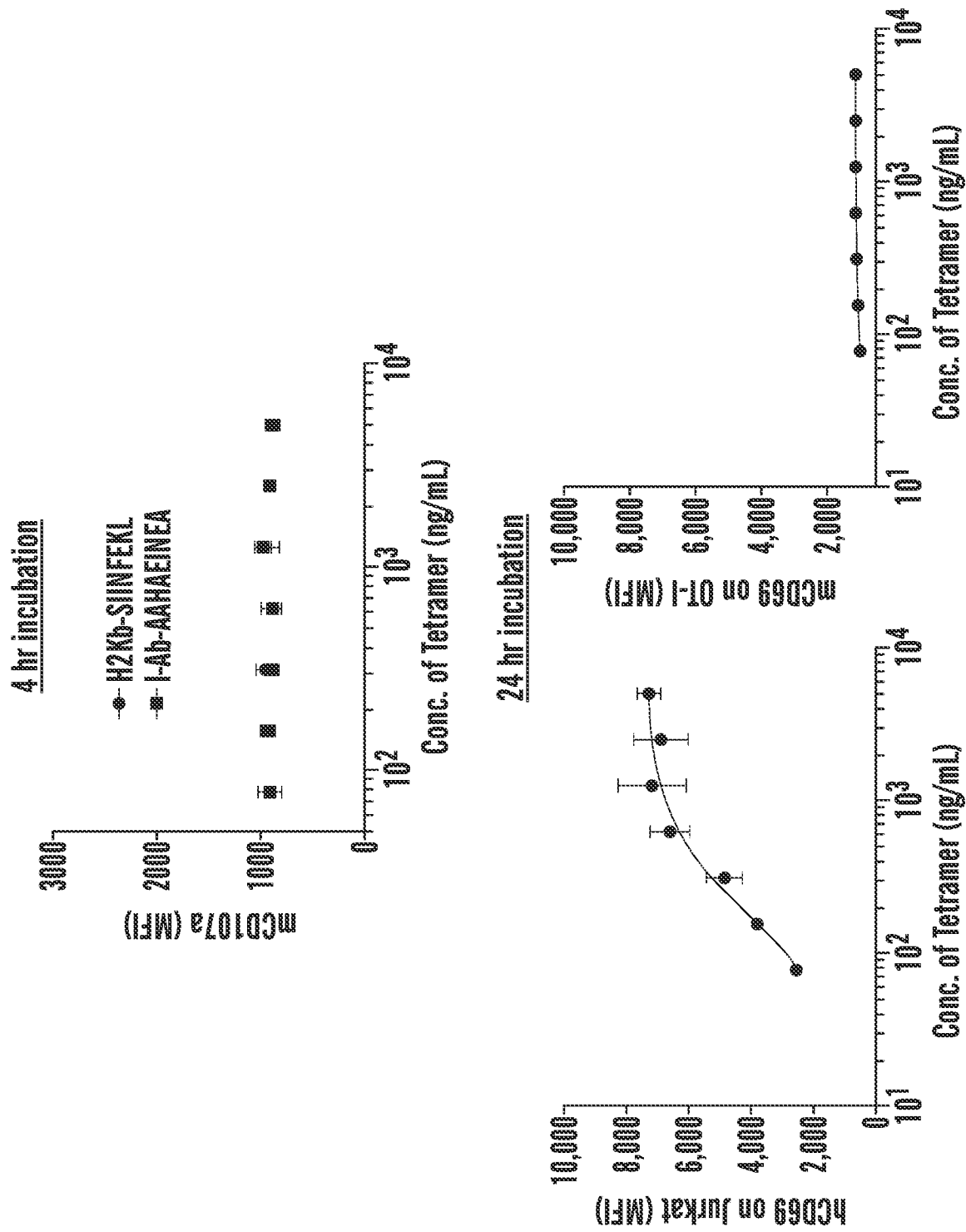


FIG. 12

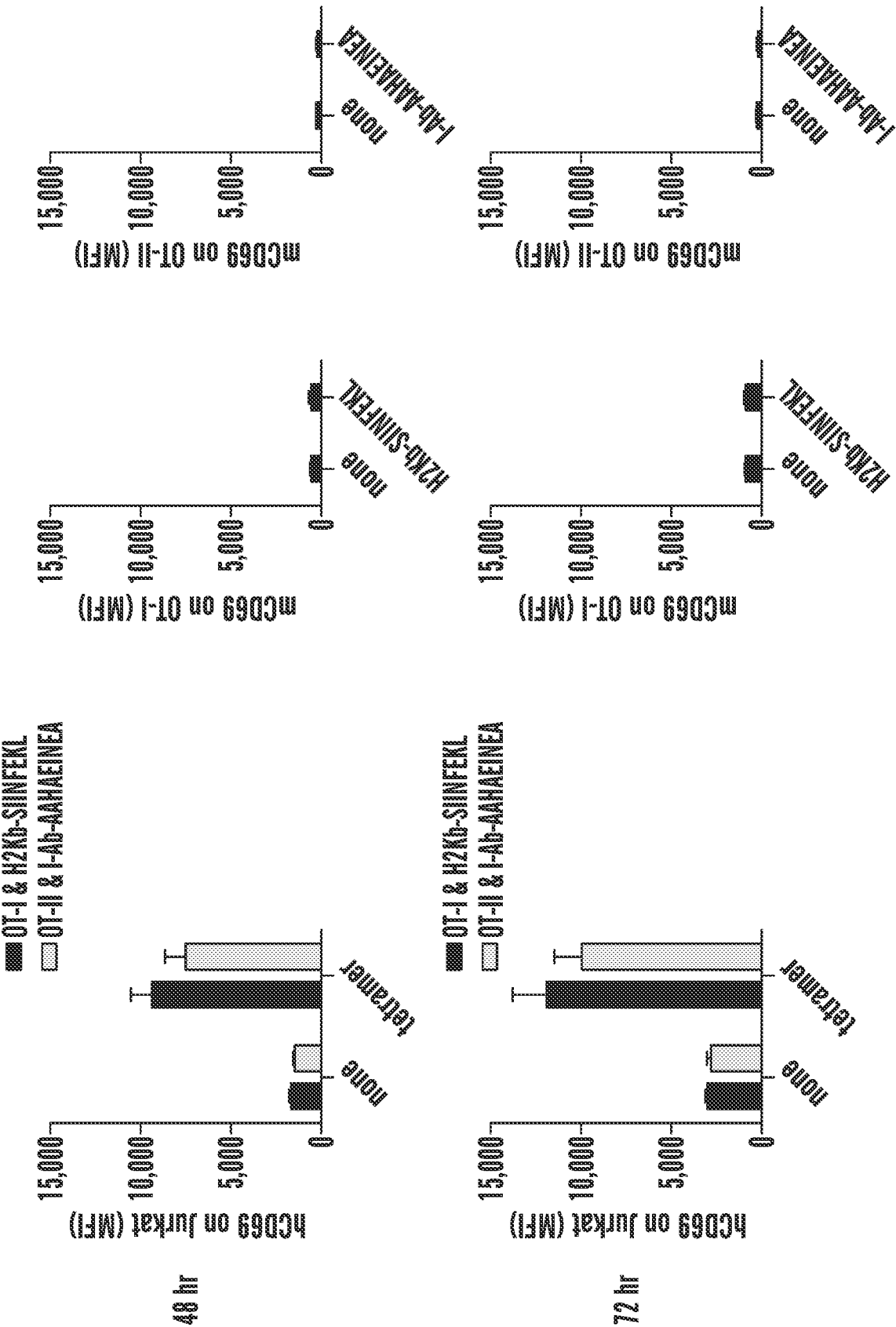
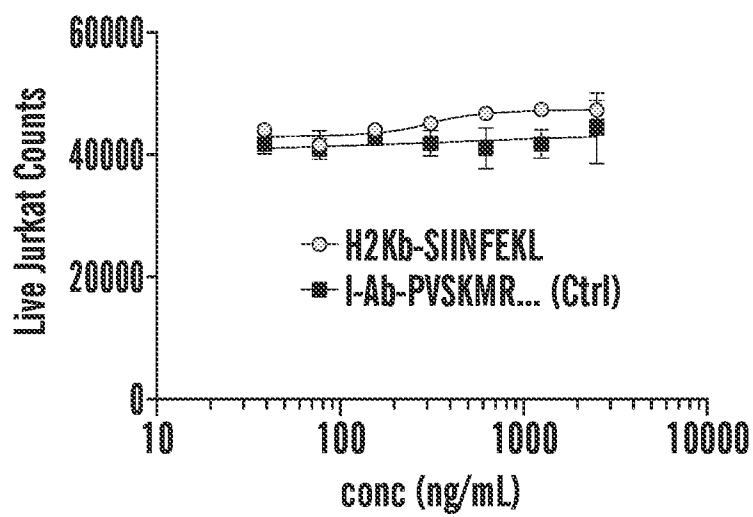
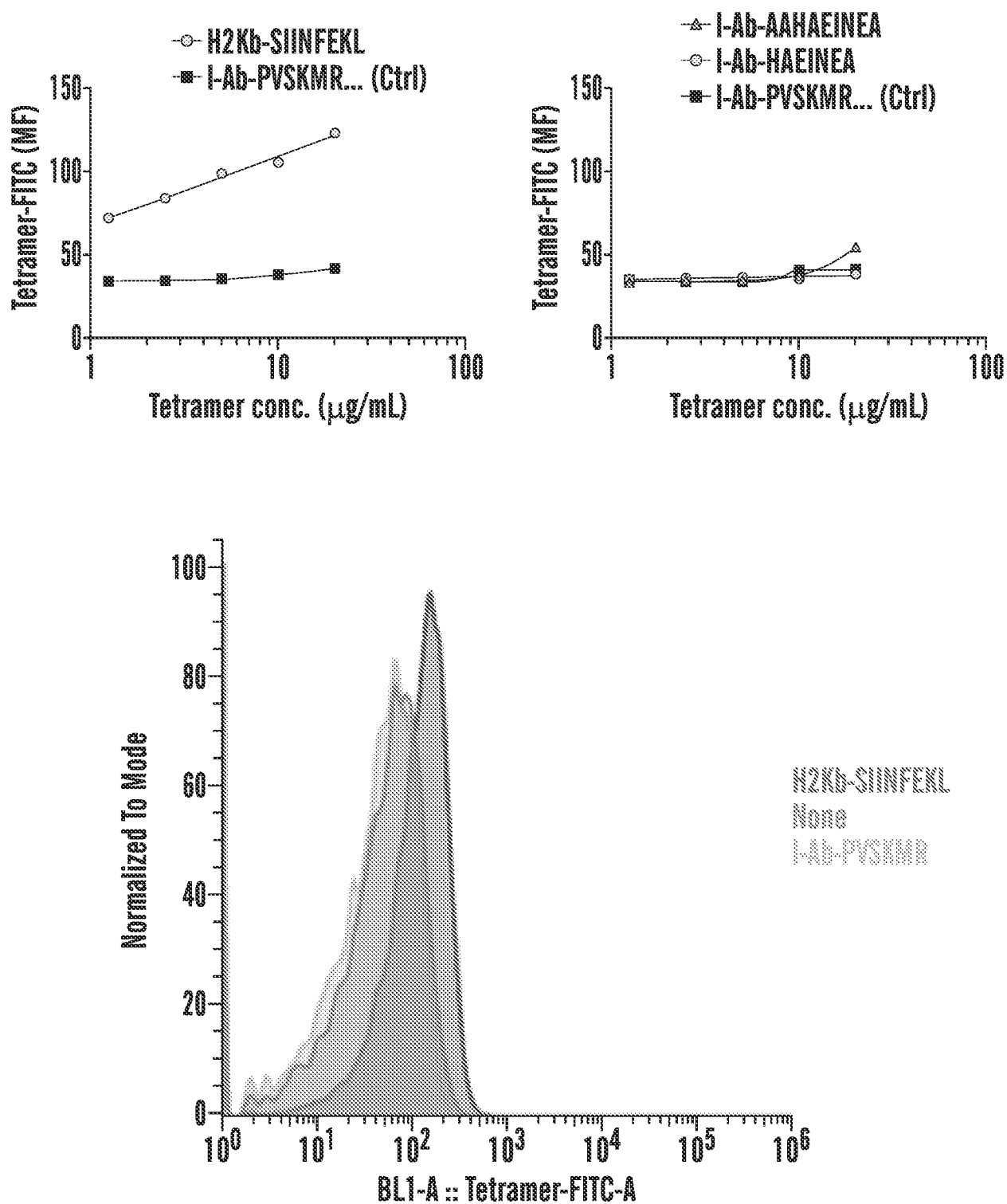


FIG. 13

**FIG. 14**

**FIG. 15**

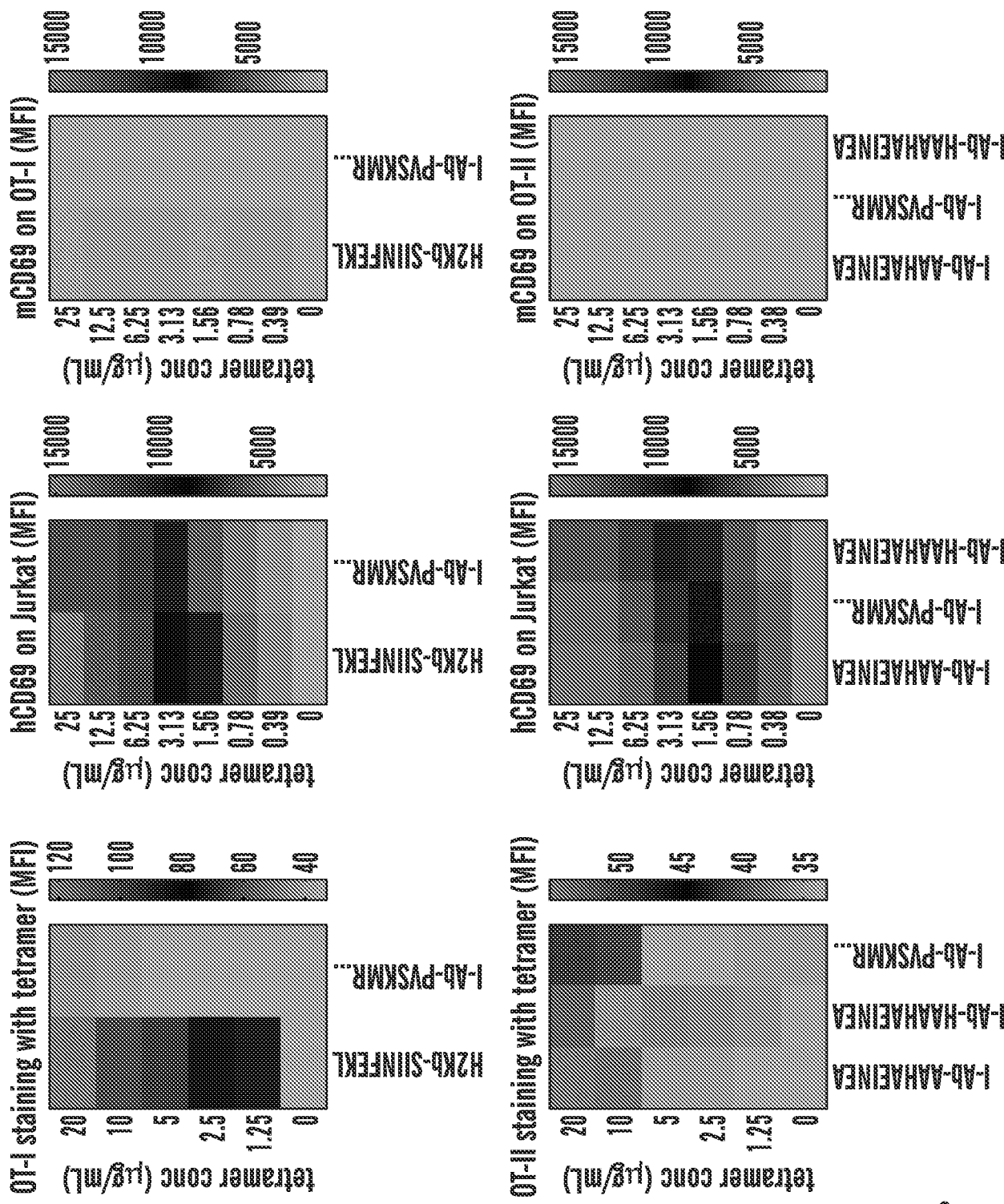


FIG. 16

OTi					OTii							
1	2	3	4	5	6	7	8	9	10	11	12	
200 (A)	200 (A)	200 (A)	200 (C)	200 (C)	200 (C)	1000 (B)	1000 (B)	1000 (B)	1000 (C)	1000 (C)	1000 (C)	
50 (A)	50 (A)	50 (A)	50 (C)	50 (C)	50 (C)	250 (B)	250 (B)	250 (B)	250 (C)	250 (C)	250 (C)	
12.5	12.5	12.5	12.5	12.5	12.5	62.5	62.5	62.5	62.5	62.5	62.5	
3.1	3.1	3.1	3.1	3.1	3.1	15.6	15.6	15.6	15.6	15.6	15.6	
0.73	0.73	0.73	0.73	0.73	0.73	3.9	3.9	3.9	3.9	3.9	3.9	
0	0	0	0	0	0	0	0	0	0	0	0	
200 (A)	200 (A)	200 (A)	200 (C)	200 (C)	200 (C)	1000 (B)	1000 (B)	1000 (B)	1000 (C)	1000 (C)	1000 (C)	
CD8-FITC	CD8-FITC	CD8-FITC	CD4-FITC	CD4-FITC	CD4-FITC	CD8-FITC	CD8-FITC	CD8-FITC	CD4-FITC	CD4-FITC	CD4-FITC	

FIG. 17

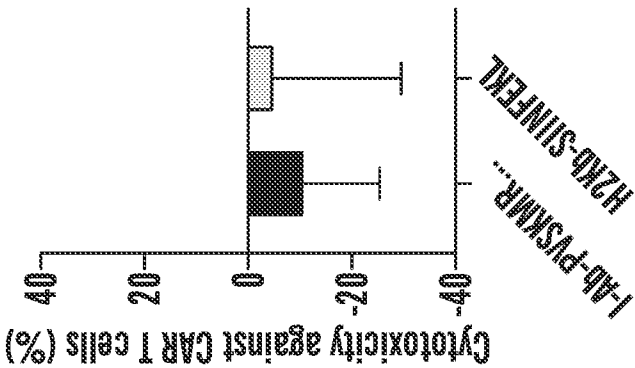


FIG. 19

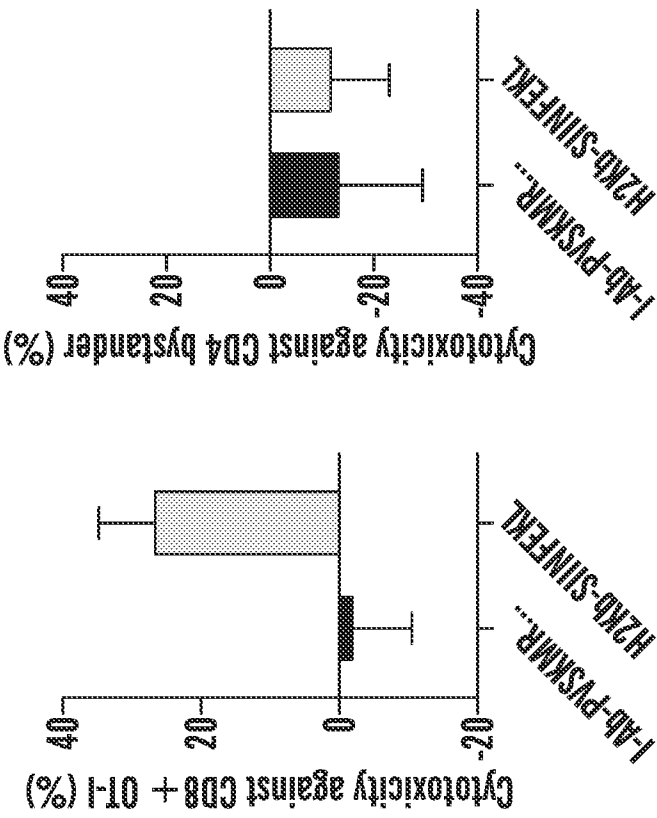


FIG. 18

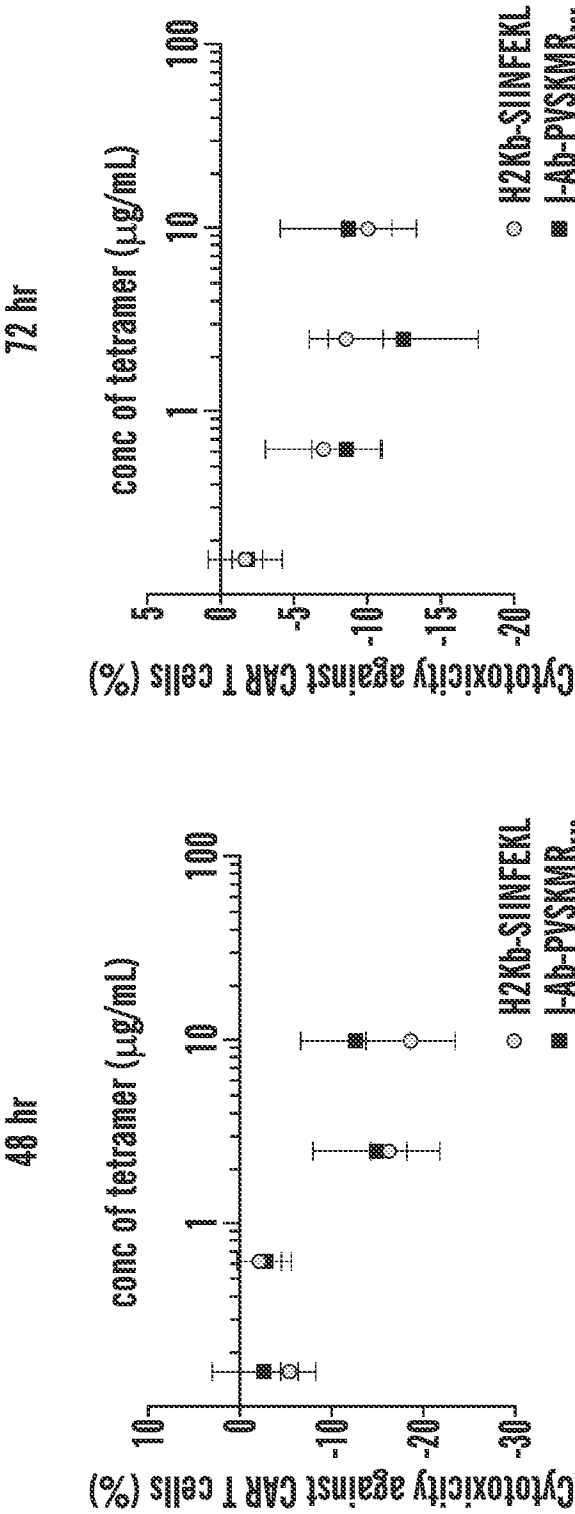


FIG. 20

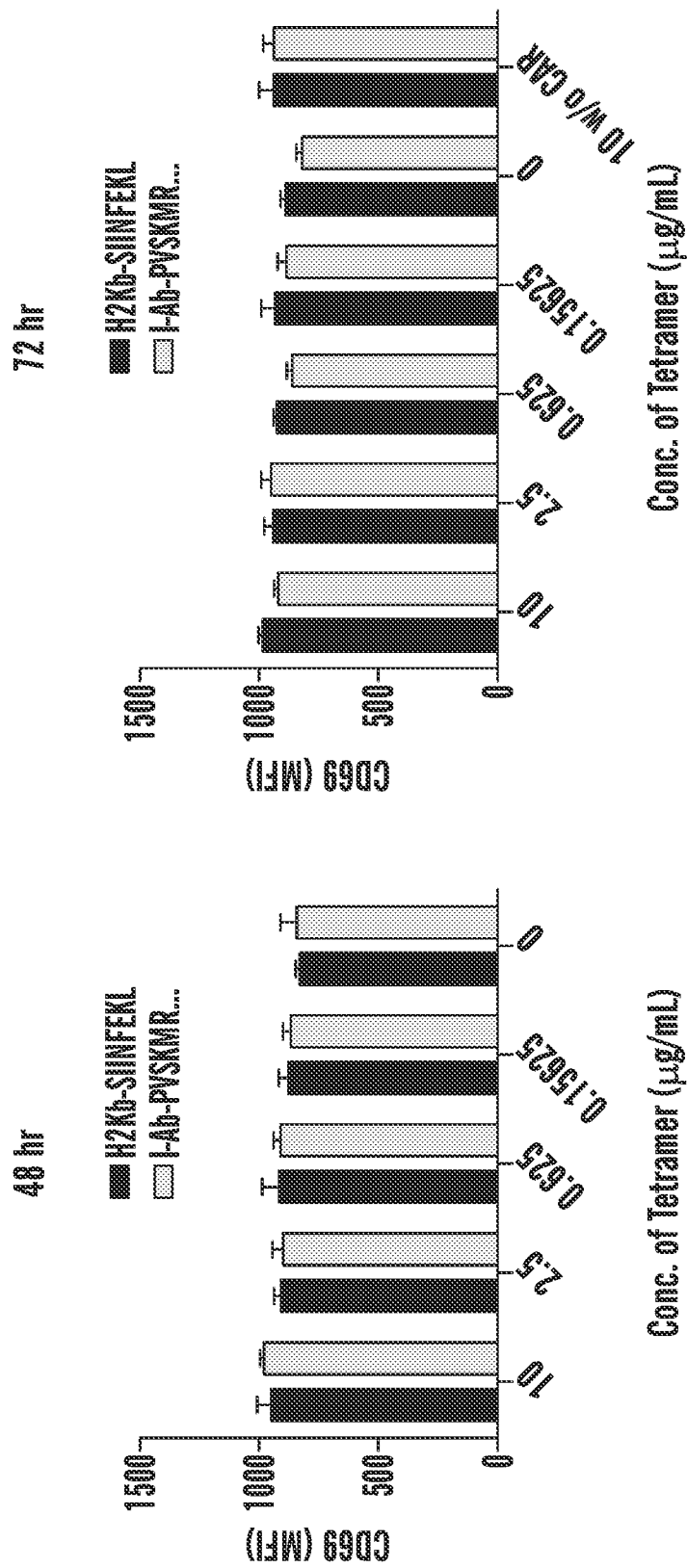
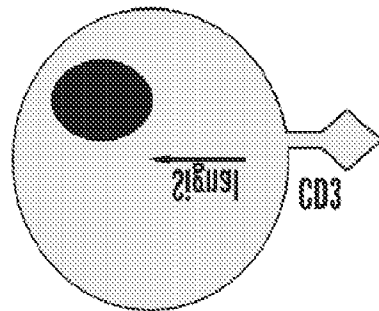


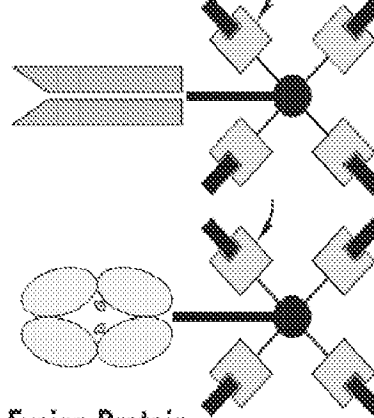
FIG. 21

(No CAR T cell Approach)

Patients' Effector Cells

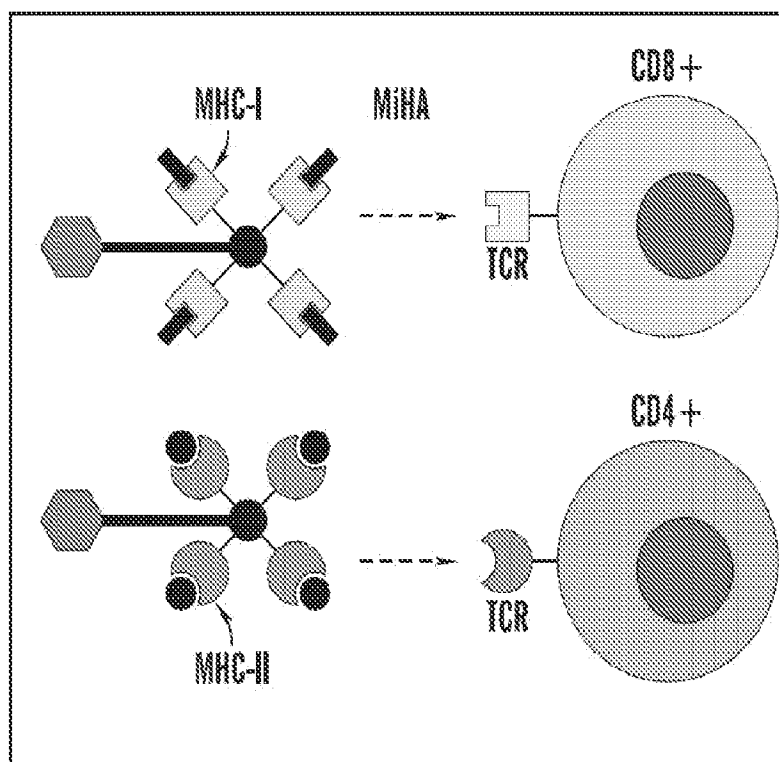


AntiCD3



Fusion Protein
(Ab Constant region)

pMHC-CAL Technology



T cells

FIG. 22

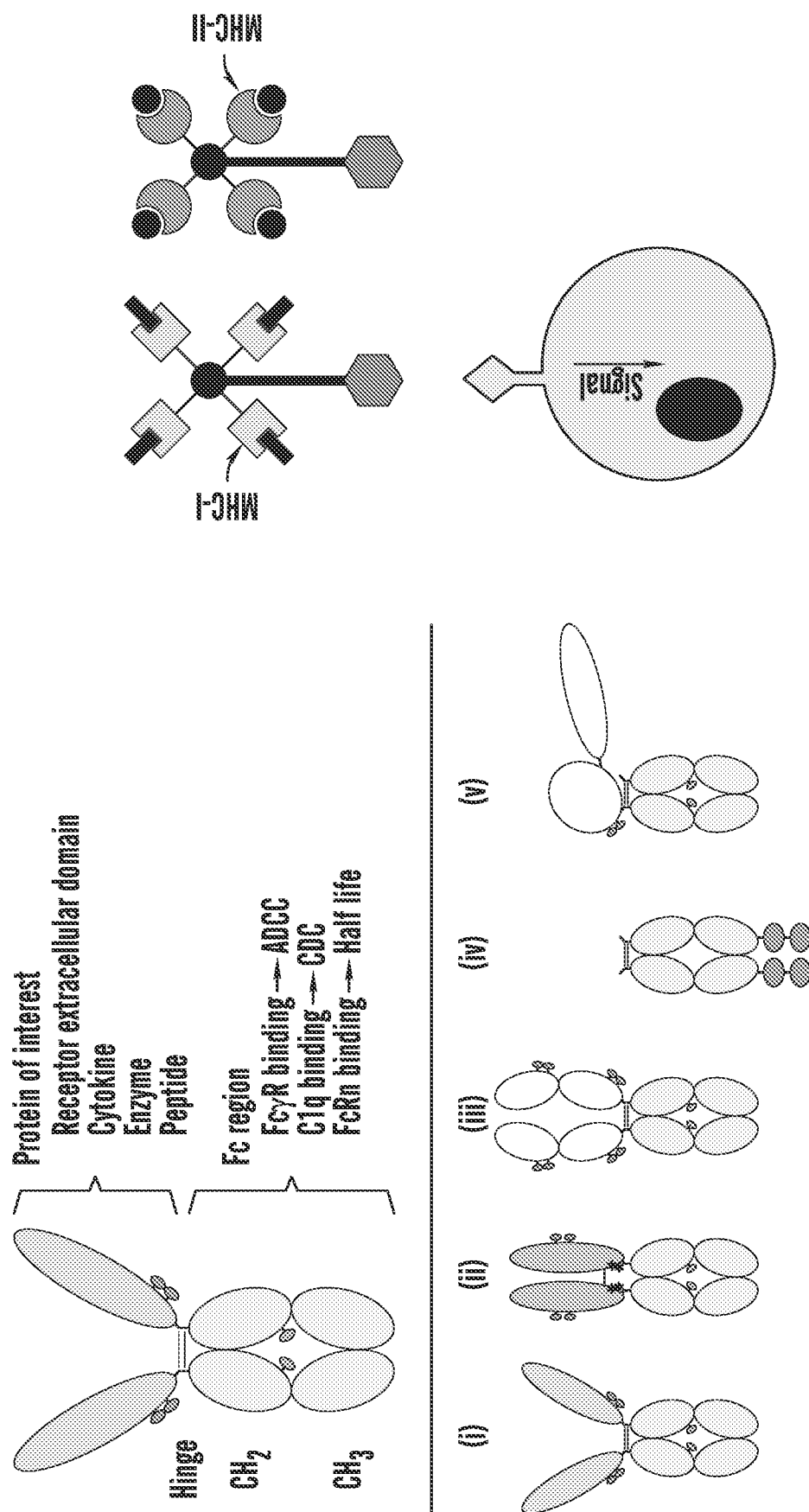


FIG. 23

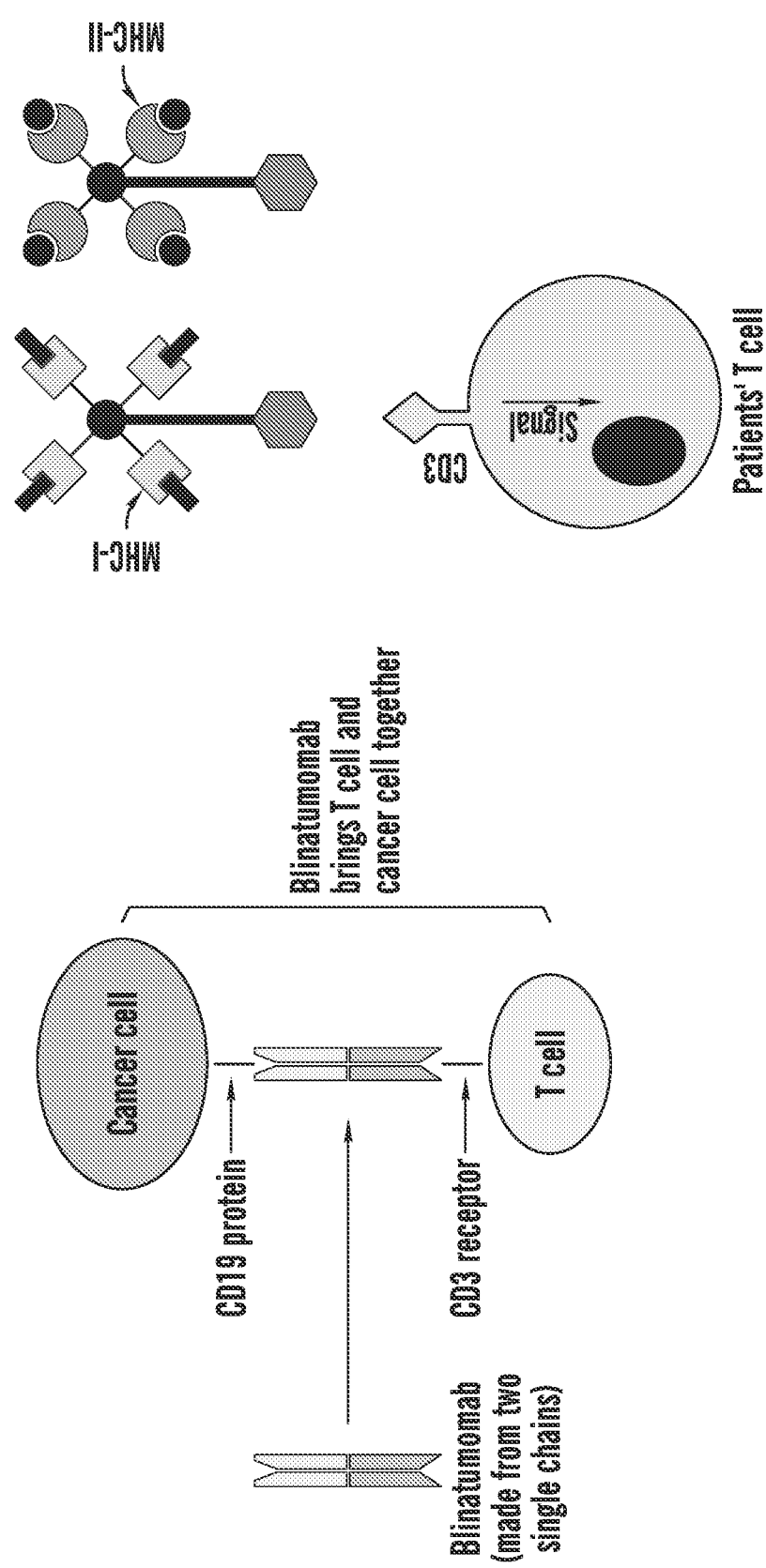


FIG. 24

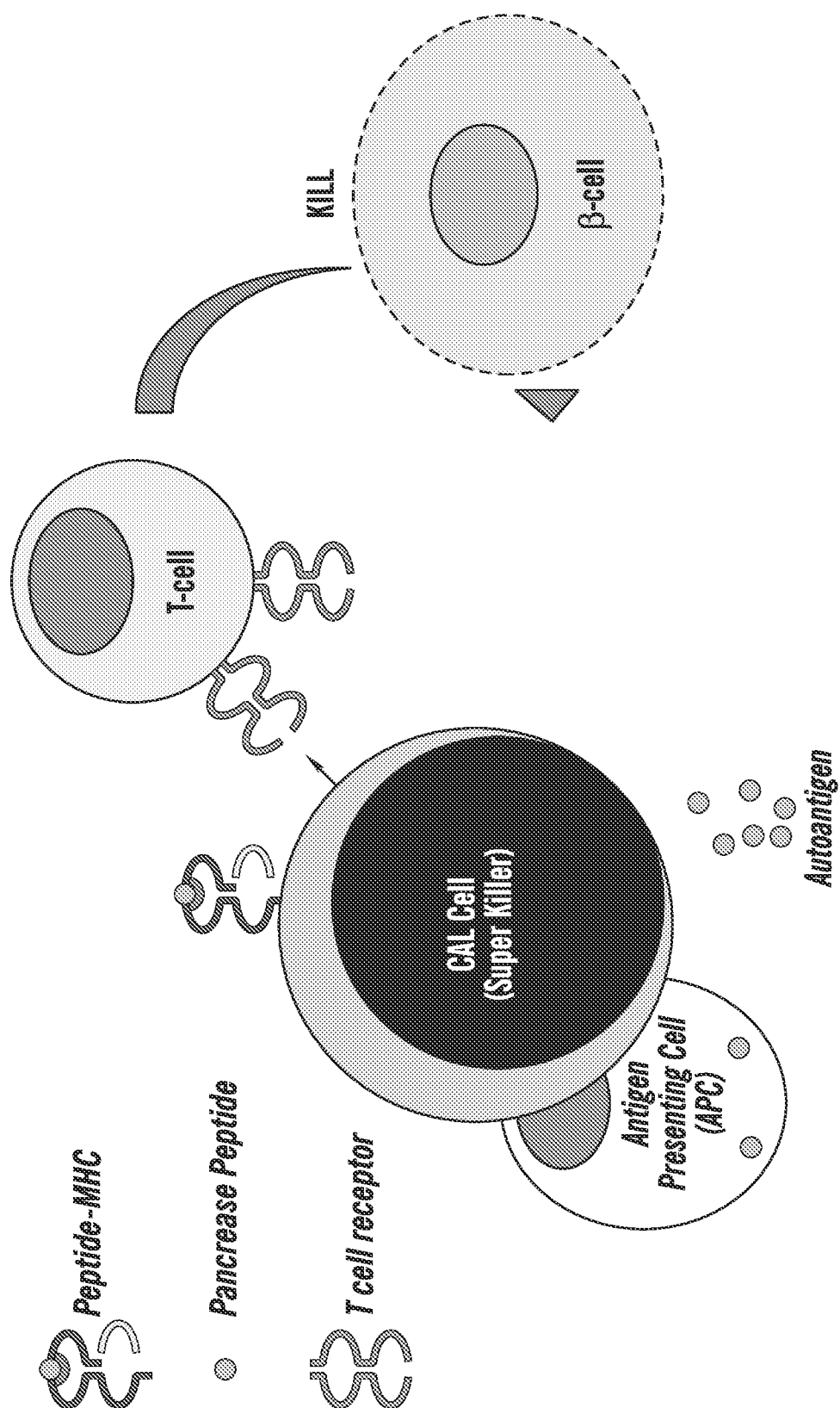
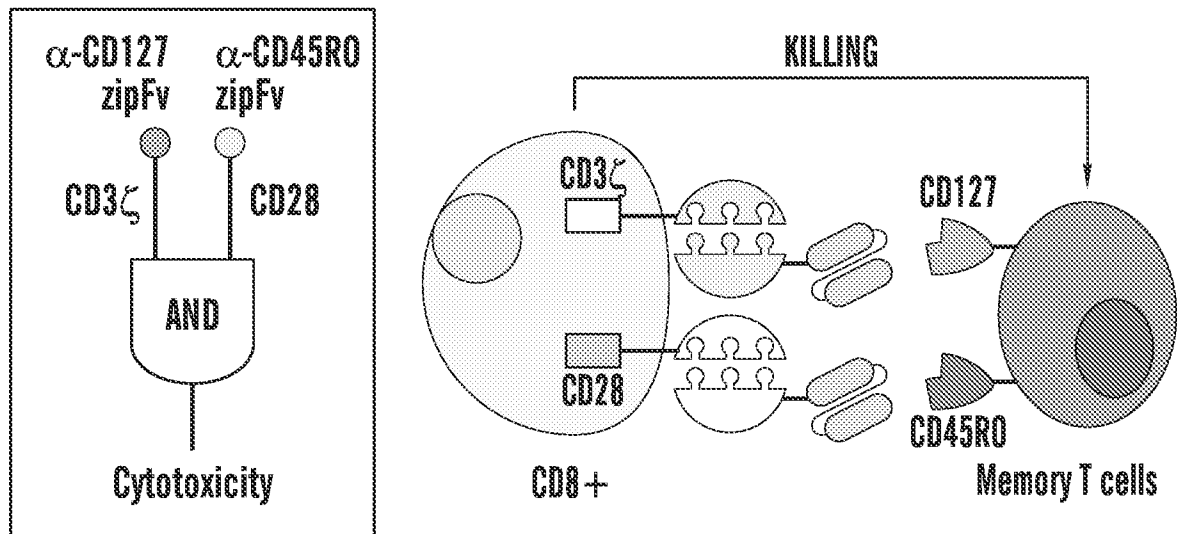
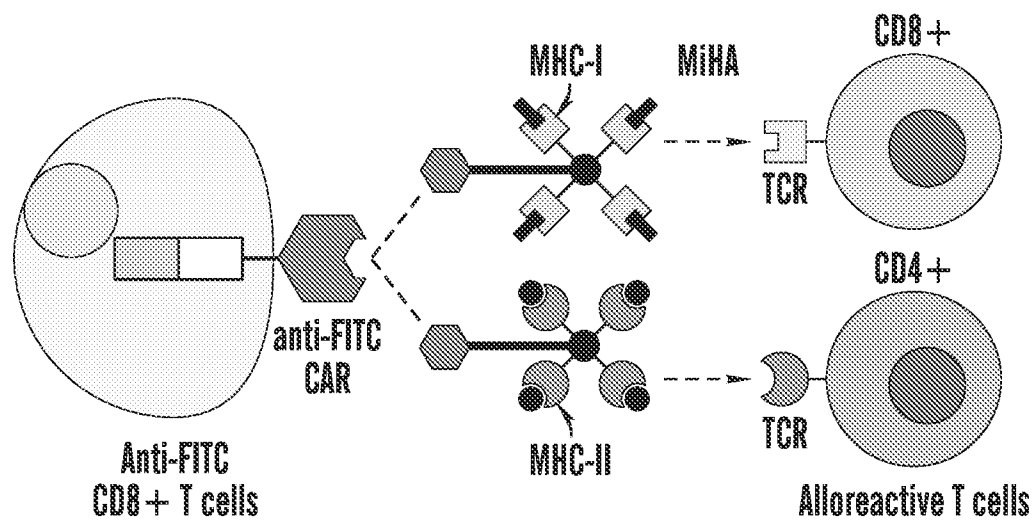
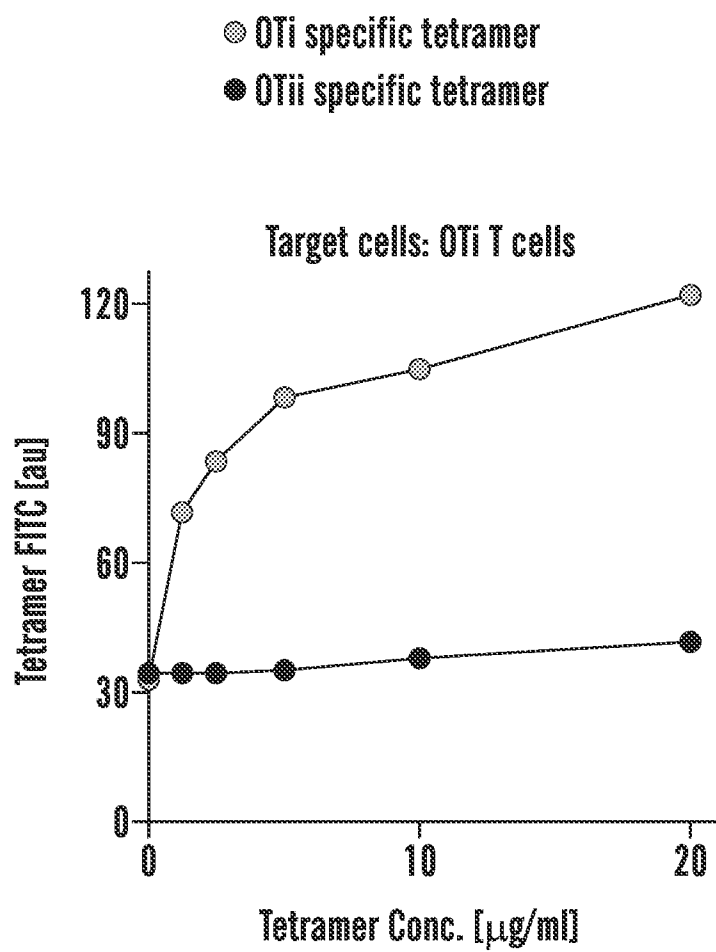


FIG. 25

**FIG. 26A****FIG. 26B**

**FIG. 27**

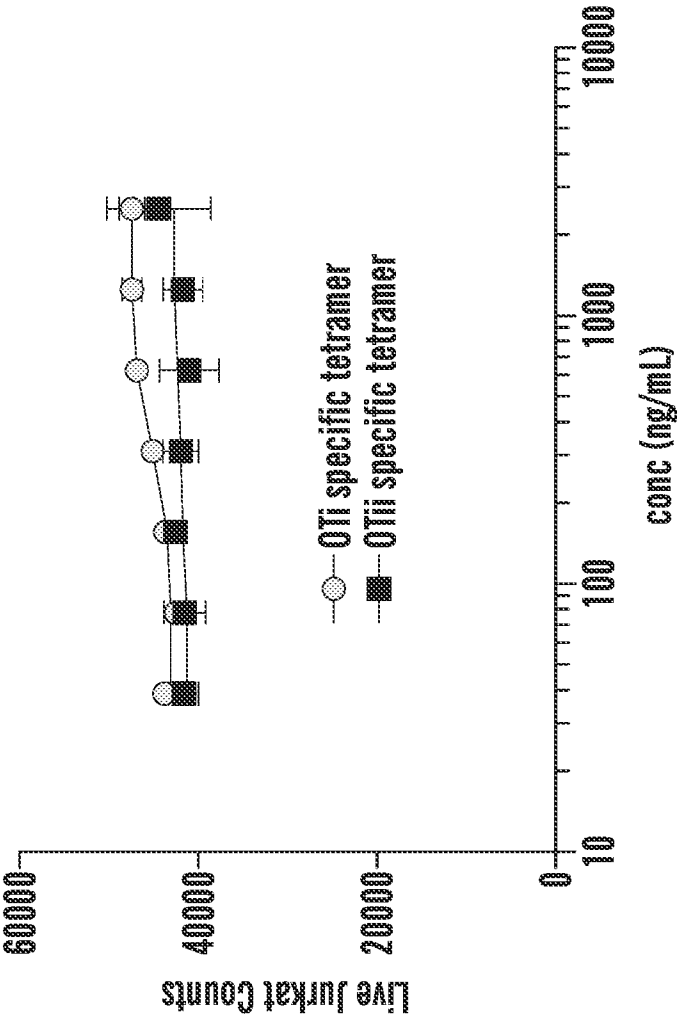


FIG. 28B

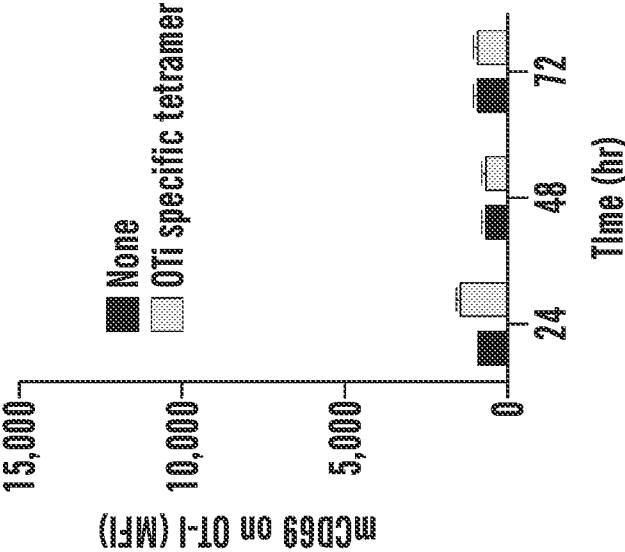
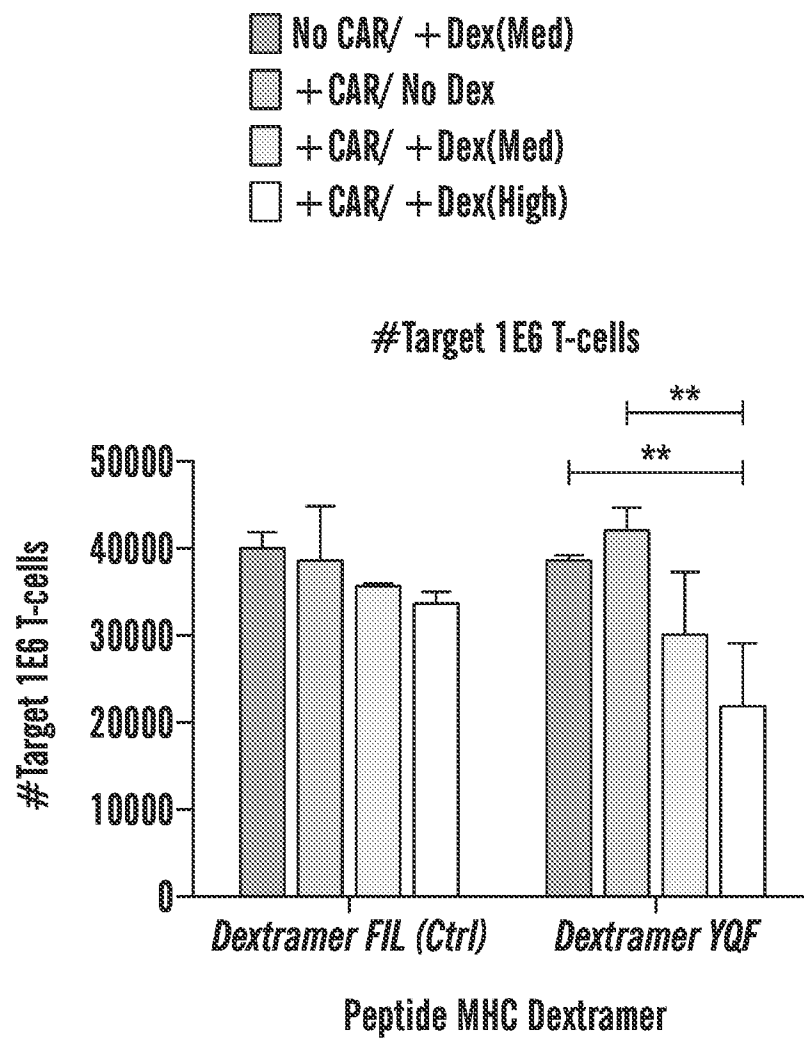
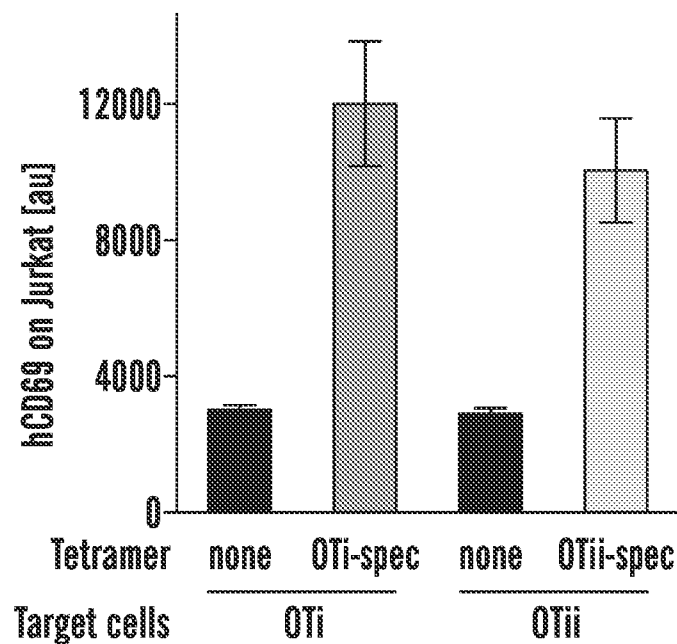
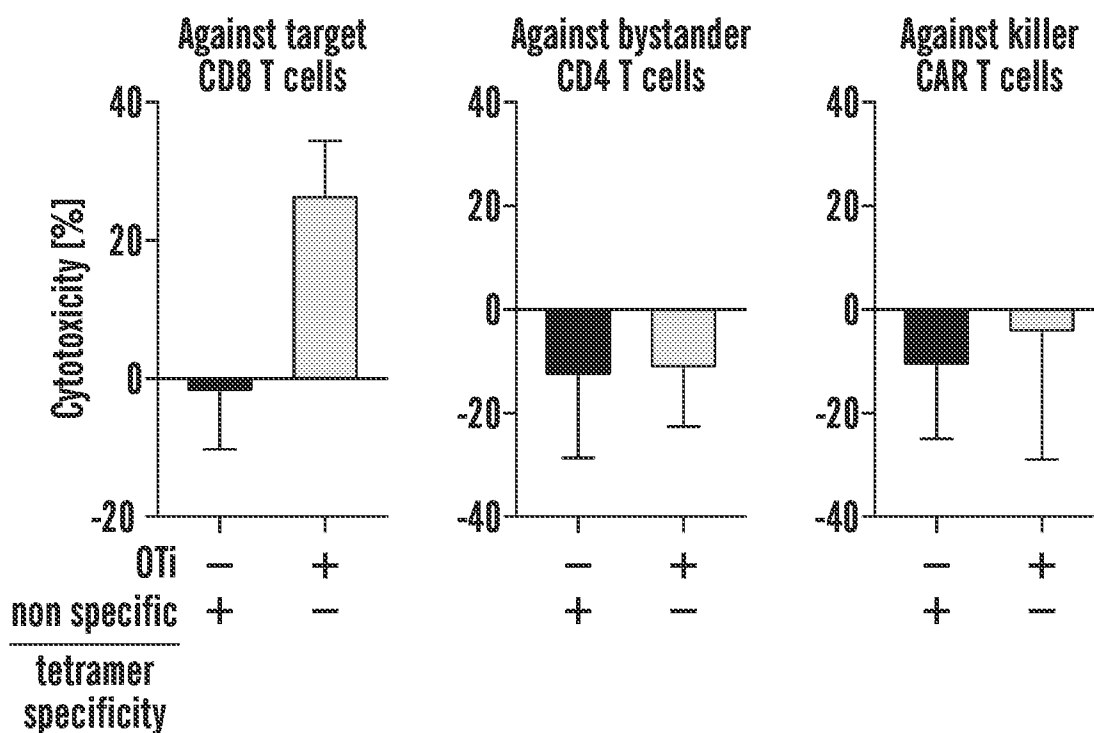
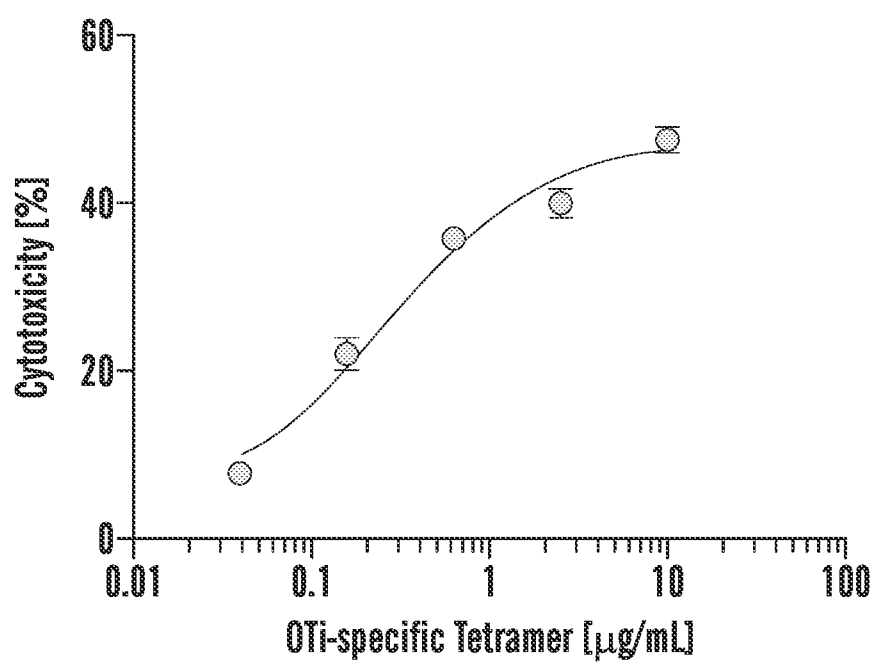


FIG. 28A

**FIG. 29**

**FIG. 30****FIG. 31**

***FIG. 32***

INTERNATIONAL SEARCH REPORT

International application No.

PCT/US 20/55980

A. CLASSIFICATION OF SUBJECT MATTER

IPC - C07K 16/28, A61K 38/17 (2020.01)

CPC - C07K 16/2863, A61K 38/177, C07K 2317/622, C07K 2319/02, C07K 2319/70, C07K 2319/73

According to International Patent Classification (IPC) or to both national classification and IPC

B. FIELDS SEARCHED

Minimum documentation searched (classification system followed by classification symbols)

See Search History document

Documentation searched other than minimum documentation to the extent that such documents are included in the fields searched

See Search History document

Electronic data base consulted during the international search (name of data base and, where practicable, search terms used)

See Search History document

C. DOCUMENTS CONSIDERED TO BE RELEVANT

Category*	Citation of document, with indication, where appropriate, of the relevant passages	Relevant to claim No.
X — Y	WO 2018/204717 A1 (HARPOON THERAPEUTICS, INC.) 08 November 2018 (08.11.2018); para [0015], [0023], [0048], [0051], [0054], [0137]; Figure 11	1-2 — 3-8, 42, 44/42
X — Y	WO 2019/099440 A1 (ARCELLX, INC.) 23 May 2019 (23.05.2019); abstract; para [0178], [0268], [0589], [0656]	41, 44/41 — 42-43, 44/(42-43)
Y	US 2018/0346541 A1 (TRUSTEES OF BOSTON UNIVERSITY) 06 December 2018 (06.12.2018); abstract; para [0012], [0015], [0026], [0215]	3-8, 43, 44/43

☐ Further documents are listed in the continuation of Box C.☐ See patent family annex.

* Special categories of cited documents:

"A" document defining the general state of the art which is not considered to be of particular relevance

"D" document cited by the applicant in the international application

"E" earlier application or patent but published on or after the international filing date

"L" document which may throw doubts on priority claim(s) or which is cited to establish the publication date of another citation or other special reason (as specified)

"O" document referring to an oral disclosure, use, exhibition or other means

"P" document published prior to the international filing date but later than the priority date claimed

"T" later document published after the international filing date or priority date and not in conflict with the application but cited to understand the principle or theory underlying the invention

"X" document of particular relevance; the claimed invention cannot be considered novel or cannot be considered to involve an inventive step when the document is taken alone

"Y" document of particular relevance; the claimed invention cannot be considered to involve an inventive step when the document is combined with one or more other such documents, such combination being obvious to a person skilled in the art

"&" document member of the same patent family

Date of the actual completion of the international search

19 December 2020

Date of mailing of the international search report

21 JAN 2021

Name and mailing address of the ISA/US

Mail Stop PCT, Attn: ISA/US, Commissioner for Patents
P.O. Box 1450, Alexandria, Virginia 22313-1450

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INTERNATIONAL SEARCH REPORT

International application No.

PCT/US 20/55980

Box No. II Observations where certain claims were found unsearchable (Continuation of item 2 of first sheet)

This international search report has not been established in respect of certain claims under Article 17(2)(a) for the following reasons:

1. ☐ Claims Nos.:
because they relate to subject matter not required to be searched by this Authority, namely:

2. ☐ Claims Nos.:
because they relate to parts of the international application that do not comply with the prescribed requirements to such an extent that no meaningful international search can be carried out, specifically:

3. ☒ Claims Nos.: 9-40, 45-65
because they are dependent claims and are not drafted in accordance with the second and third sentences of Rule 6.4(a).

Box No. III Observations where unity of invention is lacking (Continuation of item 3 of first sheet)

This International Searching Authority found multiple inventions in this international application, as follows:

1. ☐ As all required additional search fees were timely paid by the applicant, this international search report covers all searchable claims.
2. ☐ As all searchable claims could be searched without effort justifying additional fees, this Authority did not invite payment of additional fees.
3. ☐ As only some of the required additional search fees were timely paid by the applicant, this international search report covers only those claims for which fees were paid, specifically claims Nos.:

4. ☐ No required additional search fees were timely paid by the applicant. Consequently, this international search report is restricted to the invention first mentioned in the claims; it is covered by claims Nos.:

Remark on Protest

- ☐ The additional search fees were accompanied by the applicant's protest and, where applicable, the payment of a protest fee.
- ☐ The additional search fees were accompanied by the applicant's protest but the applicable protest fee was not paid within the time limit specified in the invitation.
- ☐ No protest accompanied the payment of additional search fees.