Modulation Of Endoplasmic Reticulum Aminopeptidase 2 (ERAP2)-Mediated Immune Response

Reference To Sequence Listing

This application includes a Sequence Listing filed electronically as a text file named 18923805102SEQ, created on July 23, 2021, with a size of 1,243 kilobytes. The Sequence Listing is incorporated herein by reference.

Field

The present disclosure is directed, in part, to methods of treating subjects having an immune disorder by administering a therapeutically effective amount of an Endoplasmic Reticulum Aminopeptidase 2 (ERAP2) inhibitor to the subject, and optionally an Endoplasmic Reticulum Aminopeptidase 1 (ERAP1) agonist or inhibitor and/or an HLA-Aw19 inhibitor, and also provides methods of identifying subjects having an increased risk for developing an MHC-I-opathy.

Background

The cellular immune response in humans relies at least partly on the presentation of small peptides that are 8 to 10 amino acids long, which are bound proteins of the major histocompatibility complex (MHC) (i.e., class I MHC molecules). These small peptides are derived from the proteolytic degradation of proteins (foreign antigens and self-antigens). One source of these antigens come from infected or malignantly transformed cells that express particular protein molecules that, upon degradation, yield distinct antigenic peptides that are presented on the cell surface complexed with MHC class I molecules (MHCI). Cytotoxic T cells can recognize these complexes of MHC molecules with degraded protein antigens and induce apoptotic cell death. Aberrant generation of antigenic peptides can lead to immune system evasion or to autoimmune reactions.

Although most antigenic peptides are initially produced by the proteasome, many of them are larger than the final antigenic epitope and contain one or more additional amino acids at their N-termini. These antigenic peptide precursors are transported into the endoplasmic reticulum (ER), where they are further degraded by at least two different aminopeptidases, ERAP1 and ERAP2, to generate the mature antigenic peptides for complexing

with MHC class I molecules. Thus, the activity of ERAP1 and ERAP2 can directly affect the presentation of antigenic peptides complexed with particular MHC molecules in a beneficial or adverse manner, thus altering the immune response. Accordingly, there continues to be a need for identifying subjects that have particular MHC-I-opathies related to ERAP2 activity and treatment of the same.

Birdshot Chorioretinopathy (BSCR) is a rare autoimmune uveitis predominately affecting individuals over the age of 50 of European descent and treated with immunomodulatory therapies. The disease presents with vitritis and gradual decline in vision due to choroidal and retinal inflammatory lesions and atrophy. T cells have been identified in the retinal and choroidal tissues as well as the vitreous of affected BSCR eyes.

Summary

The present disclosure provides methods of treating a subject having an immune disorder, the methods comprising administering to the subject a therapeutically effective amount of an ERAP2 inhibitor. Optionally, an ERAP1 agonist or inhibitor and/or an HLA-Aw19 inhibitor is also administered to the subject. The immune disorder can be an MHC-I-opathy or an MHC-II-opathy. The MHC-I-opathy can be BSCR, Ankylosing Spondylitis (AS), Behçet's disease, psoriasis, Juvenile Idiopathic Arthritis (JIA), inflammatory bowel disease (IBD), or Crohn's disease (CD).

Disclosed herein is the use of an Endoplasmic Reticulum Aminopeptidase 2 (ERAP2) inhibitor in the manufacture of a medicament for treating a subject having an immune disorder.

The present disclosure also provides methods of treating a subject having an MHC-I-opathy, the methods comprising: performing or having performed an assay on a biological sample from the subject to determine whether the subject comprises: i) an MHC-I-opathy-related HLA genotype; and ii) a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein; and administering to the subject a therapeutically effective amount of an ERAP2 inhibitor, wherein the subject comprises both an MHC-I-opathy-related HLA genotype and a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein; wherein the presence of both the MHC-I-opathy-related HLA genotype and the functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein indicates that the subject is a candidate for treating the MHC-I-opathy by inhibiting ERAP2. The assay performed or having been performed on the biological sample from the subject can

further determine whether the subject comprises a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein; and the methods can further comprise administering to the subject a therapeutically effective amount of an ERAP1 agonist or inhibitor, wherein the subject comprises an MHC-I-opathy-related HLA genotype and does or does not comprise a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein; wherein the presence of an MHC-I-opathy-related HLA genotype and the absence of a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein indicates that the subject is a candidate for treating the MHC-I-opathy by activating ERAP1; wherein the presence of an MHC-I-opathy-related HLA genotype and the presence of a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein indicates that the subject is a candidate for treating the MHC-I-opathy by inhibiting ERAP1.

Disclosed herein is the use of an ERAP2 inhibitor in the manufacture of a medicament for treating a subject having an MHC-I-opathy, wherein an assay performed or having been performed on a biological sample from the subject determines whether the subject comprises: i) an MHC-I-opathy-related HLA genotype; and ii) a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein; wherein the medicament is to be administered to the subject, wherein the subject comprises both an MHC-I-opathy-related HLA genotype and a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein; and wherein the presence of both the MHC-I-opathy-related HLA genotype and the functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein indicates that the subject is a candidate for treating the MHC-I-opathy by inhibiting ERAP2.

The present disclosure also provides methods of identifying a subject having an increased risk for developing an MHC-I-opathy, the methods comprising: performing or having performed an assay on a biological sample from the subject to determine whether the subject comprises: i) an MHC-I-opathy-related HLA genotype; and ii) a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein; wherein: when the subject has both the MHC-I-opathy-related HLA genotype and the functional ERAP2 protein or the nucleic acid molecule encoding the functional ERAP2 protein, then the subject has an increased risk of developing the MHC-I-opathy; when the subject lacks the MHC-I-opathy-related HLA genotype, or lacks the functional ERAP2 protein or the nucleic acid molecule encoding the functional ERAP2 protein, or lacks both, then the subject has a decreased risk of developing the MHC-I-opathy; and when the subject comprises two copies of the MHC-I-opathy-related HLA

genotype, then the subject has an increased risk of developing the MHC-I-opathy compared to comprising a single copy of the MHC-I-opathy-related HLA genotype. The assay performed or having been performed on the biological sample from the subject can further determine whether the subject comprises a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein. When the subject has the MHC-I-opathy-related HLA genotype and lacks the functional ERAP1 protein or the nucleic acid molecule encoding the functional ERAP1 protein, then the subject has an increased risk of developing the MHC-I-opathy; and when the subject lacks the MHC-I-opathy-related HLA genotype, or has the functional ERAP1 protein or the nucleic acid molecule encoding the functional ERAP1 protein, or both, then the subject has a decreased risk of developing the MHC-I-opathy.

Disclosed herein is a method of identifying a subject having an increased risk for developing an MHC-I-opathy, the method comprising: performing or having performed an assay on a biological sample from the subject to determine whether the subject comprises: i) an MHC-I-opathy-related HLA genotype; and ii) a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein; wherein: when the subject has both the MHC-I-opathy-related HLA genotype and the functional ERAP2 protein or the nucleic acid molecule encoding the functional ERAP2 protein, then the subject has an increased risk of developing the MHC-I-opathy; when the subject lacks the MHC-I-opathy-related HLA genotype, or lacks the functional ERAP2 protein or the nucleic acid molecule encoding the functional ERAP2 protein, or lacks both, then the subject has a decreased risk of developing the MHC-I-opathy; and when the subject comprises two copies of the MHC-I-opathy-related HLA genotype, then the subject has an increased risk of developing the MHC-I-opathy compared to comprising a single copy of the MHC-I-opathy-related HLA genotype.

Brief Description Of The Drawings

The patent or application file contains at least one drawing executed in color. Copies of this patent or patent application publication with color drawing(s) will be provided by the Office upon request and payment of the necessary fee.

Figure 1 shows a table of HLA-A 2nd allele frequencies in the French cohort compared to UKB and GHS EUR A29 carriers. Alleles belonging to the Aw19 broad antigen group that increase risk are A29, A30, A31 and A33 (red) and A32 exhibits protection (green). A Fisher's exact test combining all Aw19 risk alleles presents the strongest enrichment in all comparisons.

Only alleles that have three or more case carriers are presented. Table is sorted by p-values when comparing case frequencies against A29 controls in UKB.

Figure 2 shows Aw19 enrichment in Birdshot cases. Odds-ratio for BSCR, comparing frequencies of 14 HLA-A alleles that are present in three or more cases (>1%, x-axis) in 286 UParis cases compared with 108 UParis controls (Blue), GHS control cohort #1 (n= 4,014, dark green), GHS control cohort #2 (n= 2,829, bright green) and UKB controls (n= 38,543, yellow). Aw19 alleles show the highest ORs (red box) that replicates with large A29 control cohorts, with the exception of A32 that is depleted in cases (green box). * p<0.01

Figure 3 shows a table of HLA-A 2nd allele frequencies in the French cohort compared to UKB and GHS EUR A29 carriers. Alleles belonging to the Aw19 broad antigen group that increase risk are A29, A30, A31 and A33 (red) and A32 exhibits protection (green). A logistic regression test with covariates included for sex and six principal components, calculated based on genetic array data for each analytic set. Results are presented for all HLA-A alleles that have three or more case carriers. Alleles are sorted as in Figure 1. *Three principal components.

Figure 4 shows a table of haplotype analysis of ERAP1. Haplotype analysis examining the association of all eight ERAP1 haplotypes with the case-control status, showing that Hap1 and Hap2 are strongly associated with protection from BSCR.

Figure 5 shows a table of top SNPs in ERAP1 and ERAP2 regions. Variants in ERAP1 and ERAP2 are genome-wide significant when analyzed together with previous results (125 cases and 670 controls (Kuiper 2014). Rs10044354 is the top association in the ERAP1-ERAP2 locus in the previous GWAS of Dutch and Spanish cohorts, while rs27432 is the top association in the region in the current French cohort. The LD between the two loci is also presented. *The reference A-allele is the minor allele, risk is the G-allele.

Figure 6 shows ERAP2 splice region variant is protective for BSCR. The common ERAP2 splice region variant rs2248374 that disrupts ERAP2 expression is protective in the current BSCR cohort and the previous Spanish and Dutch cohorts.

Figure 7 shows the combined risk of ERAP1, ERAP2 and two copies of Aw19. Utilizing 286 Birdshot cases and 4,014 controls from GHS cohort #1 to calculate additive risk while combining risk factors in ERAP1, ERAP2 and Aw19. Panel A) An additive genotype model of ERAP2 risk signal tagged by rs10044354 and single (A29/-) or double (A29/Aw19) Aw19 copies relative to lowest risk combination of rs10044354-CC and one copy of Aw19 allele (A29). Panel B) An additive genotype model of ERAP1 risk signal tagged by rs27432 and single (A29/-) or

double (A29/Aw19) Aw19 copies relative to lowest risk combination of rs27432 -AA and one copy of Aw19 allele (A29). Panel C) An additive genotype model of ERAP1 risk signal tagged by rs27432 and ERAP2 signal tagged by rs10044354 relative to lowest risk combination of rs27432-AA and rs10044354-CC. Panel D) An additive genotype model of ERAP1 and ERAP2 risk signals and single (A29/-) or double (A29/Aw19) Aw19 copies relative to lowest risk combination. The genotypes are combined as following: 0 = ERAP1 and ERAP2 homozygous for protective allele. 1/[01],[01]/1 = either homozygous protective or heterozygous genotypes of both ERAP1 and ERAP2. 2/.,./2 = homozygous risk allele of either ERAP1 or ERAP2. 2/2 = homozygous risk allele of both ERAP1 and ERAP2.

Figure 8 shows a table of the combined risk of ERAP2 and Aw19. Utilizing 286 Birdshot cases and 4,014 controls from GHS cohort #1 to calculate additive risk while combining risk factors in ERAP2 and Aw19. An additive genotype model of ERAP2 risk signal tagged by rs10044354 and single (A29/-) or double (A29/Aw19) Aw19 copies relative to lowest risk combination of rs10044354-CC and one copy of Aw19 allele (A29).

Figure 9 shows a table of the combined risk of ERAP1 and Aw19. Utilizing 286 Birdshot cases and 4,014 controls from GHS cohort #1 to calculate additive risk while combining risk factors in ERAP1 and Aw19. An additive genotype model of ERAP1 risk signal tagged by rs27432 and single (A29/-) or double (A29/Aw19) Aw19 copies relative to lowest risk combination of rs27432-AA and one copy of Aw19 allele (A29).

Figure 10 shows a table of the combined risk of ERAP1 and ERAP2. Utilizing 286 Birdshot cases and 4,014 controls from GHS cohort #1 to calculate additive risk while combining risk factors in ERAP1 and ERAP2. An additive genotype model of ERAP1 risk signal tagged by rs27432 and ERAP2 signal tagged by rs10044354 relative to lowest risk combination of rs27432-AA and rs10044354-CC.

Figure 11 shows a table of the combined risk of ERAP1, ERAP2 and Aw19. Utilizing 286 Birdshot cases and 4,014 controls from GHS cohort #1 to calculate additive risk while combining risk factors in ERAP1, ERAP2 and Aw19. An additive genotype model of ERAP1 and ERAP2 risk signals and single (A29/-) or double (A29/Aw19) Aw19 copies relative to lowest risk combination. The genotypes are combined as following: 0 = ERAP1 and ERAP2 homozygous for protective allele. 1/[01],[01]/1 = either homozygous protective or heterozygous genotypes of both ERAP1 and ERAP2. 2/.,./2 = homozygous risk allele of either ERAP1 or ERAP2. 2/2 = homozygous risk allele of both ERAP1 and ERAP2.

Figure 12 shows differences between risk Aw19 alleles and A32. Panel A) Sequence differences between risk Aw19 alleles (red) and protective A32 allele (green). A32 exhibits F at position 9 as is the reference A:01:01 allele, while risk alleles are either T or S at that position. The Bw4 epitope sequence is apparent at positions 79-83 or A32 only.

Description Of Embodiments

Various terms relating to aspects of the present disclosure are used throughout the specification and claims. Such terms are to be given their ordinary meaning in the art, unless otherwise indicated. Other specifically defined terms are to be construed in a manner consistent with the definitions provided herein.

Unless otherwise expressly stated, it is in no way intended that any method or aspect set forth herein be construed as requiring that its steps be performed in a specific order. Accordingly, where a method claim does not specifically state in the claims or descriptions that the steps are to be limited to a specific order, it is in no way intended that an order be inferred, in any respect. This holds for any possible non-expressed basis for interpretation, including matters of logic with respect to arrangement of steps or operational flow, plain meaning derived from grammatical organization or punctuation, or the number or type of aspects described in the specification.

As used herein, the singular forms “a,” “an” and “the” include plural referents unless the context clearly dictates otherwise.

As used herein, the term “about” means that the recited numerical value is approximate and small variations would not significantly affect the practice of the disclosed embodiments. Where a numerical value is used, unless indicated otherwise by the context, the term “about” means the numerical value can vary by ±10% and remain within the scope of the disclosed embodiments.

As used herein, the term “comprising” may be replaced with “consisting” or “consisting essentially of” in particular embodiments as desired.

As used herein, the terms “nucleic acid”, “nucleic acid molecule”, “nucleic acid sequence”, “polynucleotide”, or “oligonucleotide” can comprise a polymeric form of nucleotides of any length, can comprise DNA and/or RNA, and can be single-stranded, double-stranded, or multiple stranded. One strand of a nucleic acid also refers to its complement.

As used herein, the term “subject” includes any animal, including mammals. Mammals include, but are not limited to, farm animals (such as, for example, horse, cow, pig), companion animals (such as, for example, dog, cat), laboratory animals (such as, for example, mouse, rat, rabbits), and non-human primates (such as, for example, apes and monkeys). In some embodiments, the subject is a human. In some embodiments, the subject is a patient under the care of a physician.

The present disclosure provides methods of treating a subject having an immune disorder, the methods comprising administering to the subject an ERAP2 inhibitor. In some embodiments, the immune disorder is an MHC-I-opathy. In some embodiments, the immune disorder is an MHC-II-opathy. In some embodiments, the MHC-I-opathy is Birdshot Chorioretinopathy (BSCR), Ankylosing Spondylitis (AS), Behçet’s disease, psoriasis, Juvenile Idiopathic Arthritis (JIA), inflammatory bowel disease (IBD), or Crohn’s disease (CD). In some embodiments, the MHC-I-opathy is BSCR. In some embodiments, the MHC-I-opathy is AS. In some embodiments, the MHC-I-opathy is Behçet’s disease. In some embodiments, the MHC-I-opathy is psoriasis. In some embodiments, the MHC-I-opathy is JIA. In some embodiments, the MHC-I-opathy is IBD. In some embodiments, the MHC-I-opathy is CD.

In some embodiments, the MHC-I-opathy is BSCR. In some embodiments, the method further comprises detecting the presence or absence of an HLA-Aw19 allele in a biological sample obtained from the subject. In some embodiments, the subject is HLA-Aw19$^+$. In some embodiments, the subject is or is suspected of being HLA-A*29$^+$, HLA-A*30$^+$, HLA-A*31$^+$, or HLA-A*33$^+$, or any combination thereof. In some embodiments, the method further comprises determining whether the subject has one or two copies of an HLA-Aw19 allele. In some embodiments, the subject has a single copy of HLA-Aw19. In some embodiments, the subject has two copies of HLA-Aw19. In some embodiments, the subject is HLA-A*29$^+$/HLA-A*30$^+$. In some embodiments, the subject is HLA-A*29$^+$/HLA-A*31$^+$. In some embodiments, the subject is HLA-A*29$^+$/HLA-A*33$^+$.

In some embodiments, the subject having BSCR is not HLA-A*29$^+$.

In some embodiments, the subject having BSCR has a copy of at least any two of HLA-A*29, HLA-A*30, HLA-A*31, or HLA-A*33. In some embodiments, the subject having BSCR has a copy of at least any three of HLA-A*29, HLA-A*30, HLA-A*31, or HLA-A*33. In some embodiments, the subject having BSCR has a copy of all of HLA-A*29, HLA-A*30, HLA-A*31, or HLA-A*33.

In some embodiments, the subject having BSCR has one copy of each HLA-A*29 and HLA-A*30. In some embodiments, the subject having BSCR has one copy of each HLA-A*29 and HLA-A*31. In some embodiments, the subject having BSCR has one copy of each HLA-A*29 and HLA-A*33. In some embodiments, the subject having BSCR has one copy of each HLA-A*30 and HLA-A*31. In some embodiments, the subject having BSCR has one copy of each HLA-A*30 and HLA-A*33. In some embodiments, the subject having BSCR has one copy of each HLA-A*31 and HLA-A*33.

In some embodiments, the subject having BSCR has one copy of HLA-A*29 and two copies of HLA-A*30. In some embodiments, the subject having BSCR has one copy of HLA-A*29 and two copies of HLA-A*31. In some embodiments, the subject having BSCR has one copy of HLA-A*29 and two copies of HLA-A*33. In some embodiments, the subject having BSCR has one copy of HLA-A*30 and two copies of HLA-A*31. In some embodiments, the subject having BSCR has one copy of HLA-A*30 and two copies HLA-A*33. In some embodiments, the subject having BSCR has one copy of HLA-A*31 and two copies of HLA-A*33.

In some embodiments, the subject having BSCR has two copies of HLA-A*29 and one copy of HLA-A*30. In some embodiments, the subject having BSCR has two copies of HLA-A*29 and one copy of HLA-A*31. In some embodiments, the subject having BSCR has two copies of HLA-A*29 and one copy of HLA-A*33. In some embodiments, the subject having BSCR has two copies of HLA-A*30 and one copy of HLA-A*31. In some embodiments, the subject having BSCR has two copies of HLA-A*30 and one copy of HLA-A*33. In some embodiments, the subject having BSCR has two copies of HLA-A*31 and one copy of HLA-A*33.

In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*29 and two copies of HLA-A*30. In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*29 and two copies of HLA-A*31. In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*29 and two copies of HLA-A*33. In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*30 and two copies of HLA-A*31. In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*30 and two copies of HLA-A*33. In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*31 and two copies of HLA-A*33.

In some embodiments, the method further comprises administering to the subject an HLA-Aw19 inhibitor. In some embodiments, the HLA-Aw19 inhibitor is an antibody. In some

embodiments, the antibody is an anti-HLA-A*29 antibody. In some embodiments, the HLA-Aw19 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA (shRNA) that hybridizes to an HLA-Aw19. In some embodiments, the HLA-Aw19 is HLA-A*29.

In some embodiments, the MHC-I-opathy is AS. In some embodiments, the method further comprises detecting the presence or absence of HLA-B*27 or HLA-B*40 in a biological sample obtained from the subject. In some embodiments, the subject is or is suspected of being HLA-B*27$^+$. In some embodiments, the subject is or is suspected of being HLA-B*40$^+$. In some embodiments, the method further comprises determining whether the subject has one or two copies of HLA-B*27 or HLA-B*40. In some embodiments, the subject has a single copy of HLA-B*27 or HLA-B*40. In some embodiments, the subject has two copies of HLA-B*27 or HLA-B*40. In some embodiments, the method further comprises administering to the subject an HLA-B*27 inhibitor or an HLA-B*40 inhibitor. In some embodiments, the HLA-B*27 inhibitor or HLA-B*40 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-B*27 antibody or an anti-HLA-B*40 antibody. In some embodiments, the HLA-B*27 inhibitor or HLA-B*40 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-B*27 or an HLA-B*40.

In some embodiments, the MHC-I-opathy is Behçet's disease. In some embodiments, the method further comprises detecting the presence or absence of HLA-B*51 in a biological sample obtained from the subject. In some embodiments, the subject is or is suspected of being HLA-B*51$^+$. In some embodiments, the method further comprises determining whether the subject has one or two copies of HLA-B*51. In some embodiments, the subject has a single copy of HLA-B*51. In some embodiments, the subject has two copies of HLA-B*51. In some embodiments, the method further comprises administering to the subject an HLA-B*51 inhibitor. In some embodiments, the HLA-B*51 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-B*51 antibody. In some embodiments, the HLA-B*51 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-B*51.

In some embodiments, the MHC-I-opathy is psoriasis. In some embodiments, the method further comprises detecting the presence or absence of HLA-C*06 in a biological sample obtained from the subject. In some embodiments, the subject is or is suspected of being HLA-C*06$^+$. In some embodiments, the method further comprises determining whether the subject has one or two copies of HLA-C*06. In some embodiments, the subject has a single copy of HLA-C*06. In some embodiments, the subject has two copies of HLA-C*06. In some embodiments, the method further comprises administering to the subject an HLA-C*06 inhibitor. In some embodiments, the HLA-C*06 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-C*06 antibody. In some embodiments, the HLA-C*06 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-C*06.

In some embodiments, the MHC-I-opathy is JIA. In some embodiments, the method further comprises detecting the presence or absence of HLA-B*27 and/or DRB1 in a biological sample obtained from the subject. In some embodiments, the subject is or is suspected of being HLA-B*27$^+$ and/or DRB1$^+$. In some embodiments, the method further comprises determining whether the subject has one or two copies of HLA-B*27 and/or DRB1. In some embodiments, the subject has a single copy of HLA-B*27 and/or DRB1. In some embodiments, the subject has two copies of HLA-B*27 and/or DRB1. In some embodiments, the method further comprises administering to the subject an HLA-B*27 inhibitor and/or a DRB1 inhibitor. In some embodiments, the HLA-B*27 inhibitor and/or DRB1 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-B*27 antibody or an anti-DRB1 antibody. In some embodiments, the HLA-B*27 inhibitor and/or DRB1 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-B*27 and/or an DRB1.

In some embodiments, the MHC-I-opathy is IBD or CD. In some embodiments, the method further comprises detecting the presence or absence of HLA-C*07 in a biological sample obtained from the subject. In some embodiments, the subject is or is suspected of being HLA-C*07$^+$. In some embodiments, the method further comprises determining whether the subject has one or two copies of HLA-C*07. In some embodiments, the subject has a single copy of HLA-C*07. In some embodiments, the subject has two copies of HLA-C*07. In some

embodiments, the method further comprises administering to the subject an HLA-C*07 inhibitor. In some embodiments, the HLA-C*07 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-C*07 antibody. In some embodiments, the HLA-C*07 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-C*07.

In some embodiments, the ERAP2 inhibitor comprises a small molecule degrader, a proteoloysis-targeting chimera, an immunomodulatory drug, or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to ERAP2 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule that hybridizes to ERAP2 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an siRNA that hybridizes to ERAP2 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an shRNA that hybridizes to ERAP2 mRNA. In some embodiments, the ERAP2 inhibitor comprises an anti-ERAP2 antibody. In some embodiments, the ERAP2 inhibitor comprises a pseudopeptide. In some embodiments, the pseudopeptide is a phosphinic pseudopeptide. In some embodiments, the phosphinic pseudopeptide is DG002 or DG013 (see, for example, Zervoudi et al., Proc. Natl. Acad. Sci. USA, 2013, 110, 19890-19895). In some embodiments, the phosphinic pseudopeptide is DG002. In some embodiments, the phosphinic pseudopeptide is DG013. In some embodiments, the ERAP2 inhibitor comprises a small molecule. In some embodiments, the ERAP2 inhibitor is compound 4, compound 15, compound 16, compound 5, or analogues of compound 5, which are drug-like carboxylic acids and bioisosters screened for enhanced selectivity for ERAP2 over ERAP1 (see, Medve et al., European Journal of Medicinal Chemistry, 2021, 211, 113053). In some embodiments, the ERAP2 inhibitor is a phosphonic or phosphinic acid compound with higher affinity for ERAP2 than ERAP1 (see, Weglarz-Tomczak et al., Bioorg. Med. Chem. Lett., 2016, 26, 4122-4126). Additional ERAP2 inhibitors are described in, for example, Georgiadis et al., Curr. Medic. Chem., 2019, 26, 2715-2729.

In any of the embodiments described herein, the methods can further comprise administering to the subject an ERAP1 agonist or inhibitor, depending upon the MHC-I-opathy. For AS and psoriasis, an ERAP1 inhibitor can be administered. For the remaining MHC-I-opathies, an ERAP1 agonist can be administered.

In some embodiments, the ERAP1 agonist comprises an oligonucleotide. In some embodiments, the oligonucleotide is ODN1826. In some embodiments, the ERAP1 agonist comprises a peptide. In some embodiments, the ERAP1 agonist comprises a lipopeptide. In some embodiments, the lipopeptide is Pam3CSK4 or FSL-1. In some embodiments, the lipopeptide is Pam3CSK4. In some embodiments, the lipopeptide is FSL-1. In some embodiments, the ERAP1 agonist comprises a small molecule. In some embodiments, the ERAP1 agonist can comprise an ERAP1-specific transcriptional activator, an ERAP1 protein stabilizer, an agonist of ERAP1 enzymatic activity, or an activator of ERAP1 secretion. In some embodiments, the ERAP1 agonist can comprise an ERAP1-specific transcriptional activator. In some embodiments, the ERAP1 agonist can comprise an ERAP1 protein stabilizer. In some embodiments, the ERAP1 agonist can comprise an agonist of ERAP1 enzymatic activity. In some embodiments, the ERAP1 agonist can comprise an activator of ERAP1 secretion. Additional examples of ERAP1 agonists are described in, for example, Goto et al., J. Immunol., 2014, 192, 4443-4452.

In some embodiments, the ERAP1 inhibitor comprises a small molecule degrader, a proteoloysis-targeting chimera, an immunomodulatory drug, or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to ERAP1 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule that hybridizes to ERAP1 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an siRNA that hybridizes to ERAP1 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an shRNA that hybridizes to ERAP1 mRNA. In some embodiments, the ERAP1 inhibitor comprises an anti-ERAP1 antibody. In some embodiments, the ERAP1 inhibitor is DG002 and DG013 (see, Zervoudi et al., Proc. Nat'l Acad. Sci. USA, 2013, 110, 19890-19895). In some embodiments, the ERAP1 inhibitor is a phosphinic dipeptide or tripeptide analog (see, Weglarz-Tomczak et al., Bioorg. Med. Che, Lett., 2016, 26, 4122-4126). In some embodiments, the ERAP1 inhibitor is (N-(N-(2-(1H-indol-3-yl)ethyl)carbamimidoyl) -2,5-difluorobenzenesulfonamide), (1-(1-(4-acetylpiperazine-1-carbonyl)cyclohexyl) -3-(p-tolyl)urea), or (4-methoxy-3-(N-(2-(piperidin-1-yl)-5-(trifluoromethyl)phenyl) sulfamoyl)benzoic acid (see, Maben et al., J. Med. Chem., 2020, 63, 103-121). In some embodiments, the ERAP1 inhibitor is (4aR,5S,6R,8S,8aR)-5-(2-(Furan-3-yl)ethyl)-8-hydroxy-5,6,8a-trimethyl-3,4,4a,5,6,7,8,8a-octahydronaphthalene-1-

carboxylic acid (see, Liddle et al., J. Med. Chem., 2020, 63, 3348-3358). In some embodiments, the ERAP1 inhibitor is DG013A or a phosphinic tripeptide or dipeptide or an aminophosphonic derivative, or 3,4-diaminobenzoic (DABA) derivative, or a derivative of thimerosal, (see, Georgiadis et al., Cur. Med. Chem., 2019, 26, 2715-2729). In some embodiments, the ERAP1 inhibitor is a benzofuran or 7-Benzofuran amide variation (see, Deddouche-Grass et al., ACS Med. Chem. Lett., 2021, 12, 1137-1142).

In any of the embodiments described herein, any of the inhibitors or other agents described herein can form a component of an antibody-drug-conjugate (ADC). For example, an ERAP1 inhibitor or an ERAP2 inhibitor can be conjugated to an antibody, or antigen-binding fragment thereof. The inhibitor can comprise a small molecule degrader, a proteoloysis-targeting chimera, an immunomodulatory drug, or an inhibitory nucleic acid molecule.

The present disclosure also provides methods of treating a subject having an MHC-I-opathy. In some embodiments, the method comprises performing or having performed an assay on a biological sample from the subject to determine whether the subject comprises: i) an MHC-I-opathy-related HLA genotype; and ii) a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein. In some embodiments, the method comprises administering to the subject a therapeutically effective amount of an ERAP2 inhibitor, wherein the subject comprises both an MHC-I-opathy-related HLA genotype and a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein. The presence of both the MHC-I-opathy-related HLA genotype and the functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein indicates that the subject is a candidate for treating the MHC-I-opathy by inhibiting ERAP2.

In some embodiments, the MHC-I-opathy is BSCR and the MHC-I-opathy-related HLA genotype comprises an HLA-Aw19 allele. In some embodiments, the HLA-Aw19 allele is an HLA-A*29 allele, an HLA-A*30 allele, an HLA-A*31 allele, or an HLA-A*33 allele, or any combination thereof. In some embodiments, the subject has a single copy of the HLA-Aw19 allele. In some embodiments, the HLA-Aw19 allele is an HLA-A*29 allele. In some embodiments, the HLA-Aw19 allele is an HLA-A*30 allele. In some embodiments, the HLA-Aw19 allele is an HLA-A*31 allele. In some embodiments, the HLA-Aw19 allele is an HLA-A*33 allele. In some embodiments, the subject has two copies of the HLA-Aw19 allele. In some embodiments, the subject is or is suspected of being HLA-A*29^{+}/HLA-A*30^{+}. In some embodiments, the subject is or is suspected

of being HLA-A*29$^+$/HLA-A*31$^+$. In some embodiments, the subject is or is suspected of being HLA-A*29$^+$/HLA-A*33$^+$.

In some embodiments, the subject having BSCR is not HLA-A*29$^+$.

In some embodiments, the subject having BSCR has a copy of at least any two of HLA-A*29, HLA-A*30, HLA-A*31, or HLA-A*33. In some embodiments, the subject having BSCR has a copy of at least any three of HLA-A*29, HLA-A*30, HLA-A*31, or HLA-A*33. In some embodiments, the subject having BSCR has a copy of all of HLA-A*29, HLA-A*30, HLA-A*31, or HLA-A*33.

In some embodiments, the subject having BSCR has one copy of each HLA-A*29 and HLA-A*30. In some embodiments, the subject having BSCR has one copy of each HLA-A*29 and HLA-A*31. In some embodiments, the subject having BSCR has one copy of each HLA-A*29 and HLA-A*33. In some embodiments, the subject having BSCR has one copy of each HLA-A*30 and HLA-A*31. In some embodiments, the subject having BSCR has one copy of each HLA-A*30 and HLA-A*33. In some embodiments, the subject having BSCR has one copy of each HLA-A*31 and HLA-A*33.

In some embodiments, the subject having BSCR has one copy of HLA-A*29 and two copies of HLA-A*30. In some embodiments, the subject having BSCR has one copy of HLA-A*29 and two copies of HLA-A*31. In some embodiments, the subject having BSCR has one copy of HLA-A*29 and two copies of HLA-A*33. In some embodiments, the subject having BSCR has one copy of HLA-A*30 and two copies of HLA-A*31. In some embodiments, the subject having BSCR has one copy of HLA-A*30 and two copies HLA-A*33. In some embodiments, the subject having BSCR has one copy of HLA-A*31 and two copies of HLA-A*33.

In some embodiments, the subject having BSCR has two copies of HLA-A*29 and one copy of HLA-A*30. In some embodiments, the subject having BSCR has two copies of HLA-A*29 and one copy of HLA-A*31. In some embodiments, the subject having BSCR has two copies of HLA-A*29 and one copy of HLA-A*33. In some embodiments, the subject having BSCR has two copies of HLA-A*30 and one copy of HLA-A*31. In some embodiments, the subject having BSCR has two copies of HLA-A*30 and one copy of HLA-A*33. In some embodiments, the subject having BSCR has two copies of HLA-A*31 and one copy of HLA-A*33.

In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*29 and two copies of HLA-A*30. In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*29 and two copies of HLA-A*31. In

some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*29 and two copies of HLA-A*33. In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*30 and two copies of HLA-A*31. In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*30 and two copies of HLA-A*33. In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*31 and two copies of HLA-A*33.

In some embodiments, the method further comprises administering to the subject an HLA-Aw19 inhibitor. In some embodiments, the HLA-Aw19 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-A*29 antibody. In some embodiments, the HLA-Aw19 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-Aw19. In some embodiments, the HLA-Aw19 is HLA-A*29.

In some embodiments, the MHC-I-opathy is AS and the MHC-I-opathy-related HLA genotype comprises an HLA-B*27 allele or an HLA-B*40 allele. In some embodiments, the subject has a single copy of HLA-B*27 or HLA-B*40. In some embodiments, the subject has two copies of HLA-B*27 or HLA-B*40. In some embodiments, the method further comprises administering to the subject an HLA-B*27 inhibitor or an HLA-B*40 inhibitor. In some embodiments, the HLA-B*27 inhibitor or HLA-B*40 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-B*27 antibody or an anti-HLA-B*40 antibody. In some embodiments, the HLA-B*27 inhibitor or HLA-B*40 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-B*27 or an HLA-B*40.

In some embodiments, the MHC-I-opathy is Behçet's disease and the MHC-I-opathy-related HLA genotype comprises an HLA-B*51 allele. In some embodiments, the subject has a single copy of HLA-B*51. In some embodiments, the subject has two copies of HLA-B*51. In some embodiments, the method further comprises administering to the subject an HLA-B*51 inhibitor. In some embodiments, the HLA-B*51 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-B*51 antibody. In some embodiments, the HLA-B*51 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some

embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-B*51.

In some embodiments, the MHC-I-opathy is psoriasis and the MHC-I-opathy-related HLA genotype comprises an HLA-C*06 allele. In some embodiments, the subject has a single copy of HLA-C*06. In some embodiments, the subject has two copies of HLA-C*06. In some embodiments, the method further comprises administering to the subject an HLA-C*06 inhibitor. In some embodiments, the HLA-C*06 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-C*06 antibody. In some embodiments, the HLA-C*06 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-C*06.

In some embodiments, the MHC-I-opathy is JIA and the MHC-I-opathy-related HLA genotype comprises an HLA-B*27 and/or DRB1. In some embodiments, the subject has a single copy of HLA-B*27 and/or DRB1. In some embodiments, the subject has two copies of HLA-B*27 and/or. In some embodiments, the method further comprises administering to the subject an HLA-B*27 inhibitor and/or a DRB1 inhibitor. In some embodiments, the HLA-B*27 inhibitor and/or DRB1 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-B*27 antibody and/or a DRB1 antibody. In some embodiments, the HLA-B*27 inhibitor and/or DRB1 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-B*27 or DRB1.

In some embodiments, the MHC-I-opathy is IBD or CD and the MHC-I-opathy-related HLA genotype comprises an HLA-C*07 allele. In some embodiments, the subject has a single copy of HLA-C*07. In some embodiments, the subject has two copies of HLA-C*07. In some embodiments, the method further comprising administering to the subject an HLA-C*07 inhibitor. In some embodiments, the HLA-C*07 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-C*07 antibody. In some embodiments, the HLA-C*07 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-C*07.

In any of the embodiments described herein, the nucleic acid molecule comprises genomic DNA, mRNA, or cDNA obtained from mRNA. In some embodiments, the nucleic acid

molecule comprises genomic DNA. In some embodiments, the nucleic acid molecule comprises mRNA. In some embodiments, the nucleic acid molecule comprises cDNA obtained from mRNA.

In any of the embodiments described herein, the ERAP2 inhibitor comprises a small molecule degrader, a proteoloysis-targeting chimera, an immunomodulatory drug, or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to ERAP2 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule that hybridizes to ERAP2 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an siRNA that hybridizes to ERAP2 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an shRNA that hybridizes to ERAP2 mRNA. In some embodiments, the ERAP2 inhibitor comprises an anti-ERAP2 antibody. In some embodiments, the ERAP2 inhibitor comprises a pseudopeptide. In some embodiments, the pseudopeptide is a phosphinic pseudopeptide. In some embodiments, the phosphinic pseudopeptide is DG002 or DG013. In some embodiments, the ERAP2 inhibitor comprises a small molecule.

In any of the embodiments described herein, the assay performed or having been performed on the biological sample from the subject can further determine whether the subject comprises a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein. In some embodiments, the method further comprises administering to the subject a therapeutically effective amount of an ERAP1 agonist or inhibitor (depending upon the MHC-I-opathy), wherein the subject comprises an MHC-I-opathy-related HLA genotype and does or does not comprise a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein. The presence of an MHC-I-opathy-related HLA genotype and the absence of a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein indicates that the subject is a candidate for treating the MHC-I-opathy by activating ERAP1. The presence of an MHC-I-opathy-related HLA genotype and the presence of a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein indicates that the subject is a candidate for treating the MHC-I-opathy by inhibiting ERAP1.

In any of the embodiments described herein, the ERAP1 agonist comprises an oligonucleotide. In some embodiments, the oligonucleotide is ODN1826. In some embodiments, the ERAP1 agonist comprises a peptide. In some embodiments, the ERAP1 agonist comprises a lipopeptide. In some embodiments, the lipopeptide is Pam3CSK4 or FSL-1. In some embodiments, the lipopeptide is Pam3CSK4. In some embodiments, the lipopeptide is

FSL-1. In some embodiments, the ERAP1 agonist comprises a small molecule. In some embodiments, the ERAP1 agonist can comprise an ERAP1-specific transcriptional activator, an ERAP1 protein stabilizer, an agonist of ERAP1 enzymatic activity, or an activator of ERAP1 secretion. In some embodiments, the ERAP1 agonist can comprise an ERAP1-specific transcriptional activator. In some embodiments, the ERAP1 agonist can comprise an ERAP1 protein stabilizer. In some embodiments, the ERAP1 agonist can comprise an agonist of ERAP1 enzymatic activity. In some embodiments, the ERAP1 agonist can comprise an activator of ERAP1 secretion. Additional examples of ERAP1 agonists are described in, for example, Goto et al., J. Immunol., 2014, 192, 4443-4452.

In any of the embodiments described herein, the ERAP1 inhibitor comprises a small molecule degrader, a proteoloysis-targeting chimera, an immunomodulatory drug, or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to ERAP1 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule that hybridizes to ERAP1 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an siRNA that hybridizes to ERAP1 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an shRNA that hybridizes to ERAP1 mRNA. In some embodiments, the ERAP1 inhibitor comprises an anti-ERAP1 antibody.

HLA-class-I antibodies can be generated by numerous methodologies with different degrees of antigen/allele specificity attained and are reported to be used for *in vitro* assays. HLA-B*27 antibodies can be generated by numerous methodologies. In addition, three commercially available antibodies for HLA-B27 flow cytometric screening include the monoclonal mouse anti-human ABC-m3, FD705, and GS145.2 which have been shown to each have differing levels of cross-reactivity to other HLA-B antigens/alleles (Levering et al., Cytometry B Clin. Cytom., 2003, 54, 28-38). HLA-B*51 antibodies can also be generated by numerous methodologies. For example, antibodies to a broader HLA-Bw4 epitope can be obtained from clone REA274 (e.g., HLA-B members: B5, B5102, B5103, B13, B17, B37, B38, B44, B47, B49, B51, B52, B53, B57, B58, B59, B63, and B77; HLA-A members: A9, A23, A24, A25, and A*32). In addition, antibodies to HLA-B*51/B*52/B*35 can be obtained from clone HDG8D9 (Drabbels et al., Blood, 2011, 118, e149-55). HLA-C*06 antibodies can also be generated by numerous methodologies. For example, pan HLA-C antibodies can be obtained from clone DT-9 (which also recognizes HLA-E) (Braud et al., Curr. Biol., 1998, 8, 1-10). Broad anti-HLA-C

antibodies can be obtained from clone L31 (which also recognizes some HLA-B alleles) (Setini et al., Hum. Immunol., 1996, 46, 69-81). HLA-Cw6 scFv can also be generated which has weak binding to HLA-Cw2,4,5 (Marget et al., Mol. Immunol., 2005, 42, 643-649).

In some embodiments, the assay for determining whether the subject comprises an MHC-I-opathy-related and/or MHC-II-opathy-related HLA genotype and a functional ERAP2 protein and/or ERAP1 protein, or a nucleic acid molecule encoding a functional ERAP2 protein and/or ERAP1 protein, is a genotyping assay or sequencing assay. In some embodiments, the nucleic acid molecule encoding a functional ERAP2 protein and/or ERAP1 protein comprises genomic DNA, mRNA, or cDNA obtained from mRNA. By comparing the nucleotide or protein sequence of the ERAP2 protein and/or ERAP1 protein in the sample from a subject to the wild type sequence for ERAP2 protein and/or ERAP1 protein or nucleic acid molecule, or to published sequences of variant ERAP2 proteins and/or ERAP1 proteins or nucleic acid molecules having reduced or no activity, a determination can be made whether the subject comprises a functional ERAP2 protein and/or ERAP1 protein, or a nucleic acid molecule encoding a functional ERAP2 protein and/or ERAP1 protein. In addition, although an individual ERAP2 protein and/or ERAP1 protein may have biological activity, the overall function of the ERAP2 protein and/or ERAP1 protein may not be functional due to reduced levels of expression. Thus, as used herein, an ERAP2 protein and/or ERAP1 protein can be determined not to be functional because the ERAP2 protein and/or ERAP1 protein lacks or had reduced biological activity or because the expression level is reduced.

Determining whether a subject has an MHC-I-opathy-related and/or MHC-II-opathy-related HLA genotype and/or a functional ERAP2 protein and/or ERAP1 protein, or a nucleic acid molecule encoding a functional ERAP2 protein and/or ERAP1 protein, in a biological sample from a subject can be carried out by any of the methods described herein. In some embodiments, these methods can be carried out *in vitro*. In some embodiments, these methods can be carried out *in situ*. In some embodiments, these methods can be carried out *in vivo*. In any of these embodiments, the nucleic acid molecule can be present within a biological sample obtained from the subject.

The biological sample can be derived from any cell, tissue, or biological fluid from the subject. The biological sample may comprise any clinically relevant tissue, such as a bone marrow sample, a tumor biopsy, a fine needle aspirate, or a sample of bodily fluid, such as blood, gingival crevicular fluid, plasma, serum, lymph, ascitic fluid, cystic fluid, or urine. In some

cases, the sample comprises a buccal swab. The biological sample used in the methods disclosed herein can vary based on the assay format, nature of the detection method, and the tissues, cells, or extracts that are used as the sample. A biological sample can be processed differently depending on the assay being employed. For example, when detecting any particular nucleic acid molecule, preliminary processing designed to isolate or enrich the biological sample for the particular nucleic acid molecule can be employed. A variety of techniques may be used for this purpose. Various methods to detect the presence or level of an mRNA molecule or the presence of a particular genomic DNA locus can be used.

In some embodiments, the biological sample comprises a cell or cell lysate. Such methods can further comprise, for example, obtaining a biological sample from the subject comprising genomic nucleic acid molecules or mRNA molecules, and if mRNA, optionally reverse transcribing the mRNA into cDNA. In some embodiments, the method is an *in vitro* method. In some embodiments, the assay comprises RNA sequencing (RNA-Seq). In some embodiments, the assays also comprise reverse transcribing mRNA into cDNA, such as by the reverse transcriptase polymerase chain reaction (RT-PCR).

Illustrative examples of nucleic acid sequencing techniques include, but are not limited to, chain terminator (Sanger) sequencing and dye terminator sequencing. Other methods involve nucleic acid hybridization methods other than sequencing, including using labeled primers or probes directed against purified DNA, amplified DNA, and fixed cell preparations (fluorescence in situ hybridization (FISH)). In some methods, a target nucleic acid molecule may be amplified prior to or simultaneous with detection. Illustrative examples of nucleic acid amplification techniques include, but are not limited to, polymerase chain reaction (PCR), ligase chain reaction (LCR), strand displacement amplification (SDA), and nucleic acid sequence based amplification (NASBA). Other methods include, but are not limited to, ligase chain reaction, strand displacement amplification, and thermophilic SDA (tSDA).

Administration of any of the therapeutic agents described herein (including the ERAP2 inhibitor, the ERAP1 agonist or inhibitor, and/or the HLA inhibitor) can be in a therapeutically effective amount to be determined by a health care professional. Administration of any of the therapeutic agents can be repeated, for example, after one day, two days, three days, five days, one week, two weeks, three weeks, one month, five weeks, six weeks, seven weeks, eight weeks, two months, or three months. The repeated administration can be at the same dose or at a different dose. The administration can be repeated once, twice, three times, four times,

five times, six times, seven times, eight times, nine times, ten times, or more. For example, according to certain dosage regimens a subject can receive therapy for a prolonged period of time such as, for example, 6 months, 1 year, or more.

Administration of any of the therapeutic agents can occur by any suitable route including, but not limited to, parenteral, intravenous, oral, subcutaneous, intra-arterial, intracranial, intrathecal, intraperitoneal, topical, intranasal, intra-articular, intravitreal, intracameral, subretinal, suprachoroidal, or intramuscular.

Pharmaceutical compositions for administration are desirably sterile and substantially isotonic and manufactured under GMP conditions. Pharmaceutical compositions can be provided in unit dosage form (i.e., the dosage for a single administration). Pharmaceutical compositions can be formulated using one or more physiologically and pharmaceutically acceptable carriers, diluents, excipients or auxiliaries. The formulation depends on the route of administration chosen. The term “pharmaceutically acceptable” means that the carrier, diluent, excipient, or auxiliary is compatible with the other ingredients of the formulation and not substantially deleterious to the recipient thereof.

The terms “treat”, “treating”, and “treatment” and “prevent”, “preventing”, and “prevention” as used herein, refer to eliciting the desired biological response, such as a therapeutic and prophylactic effect, respectively. In some embodiments, a therapeutic effect comprises one or more of a decrease/reduction in an MHC-I-opathy and/or MHC-II-opathy, a decrease/reduction in the severity of an MHC-I-opathy and/or MHC-II-opathy (such as, for example, a reduction or inhibition of development of an MHC-I-opathy and/or MHC-II-opathy), a decrease/reduction in symptoms and MHC-I-opathy-related effects and/or MHC-II-opathy-related effects, delaying the onset of symptoms and MHC-I-opathy-related effects and/or MHC-II-opathy-related effects, reducing the severity of symptoms of MHC-I-opathy-related effects and/or MHC-II-opathy-related effects, reducing the severity of an acute episode, reducing the number of symptoms and MHC-I-opathy-related effects and/or MHC-II-opathy-related effects, reducing the latency of symptoms and MHC-I-opathy-related effects and/or MHC-II-opathy-related effects, an amelioration of symptoms and MHC-I-opathy-related effects and/or MHC-II-opathy-related effects, reducing secondary symptoms, reducing secondary infections, preventing relapse to an MHC-I-opathy and/or MHC-II-opathy, decreasing the number or frequency of relapse episodes, increasing latency between symptomatic episodes, increasing time to sustained progression, speeding recovery, or increasing efficacy of or decreasing

resistance to alternative therapeutics, and/or an increased survival time of the subject, following administration of the agent or composition comprising the agent. A prophylactic effect may comprise a complete or partial avoidance/inhibition or a delay of an MHC-I-opathy and/or MHC-II-opathy development/progression (such as, for example, a complete or partial avoidance/inhibition or a delay), and an increased survival time of the affected subject, following administration of a therapeutic protocol. Treatment of an MHC-I-opathy and/or MHC-II-opathy encompasses the treatment of subjects already diagnosed as having any form of the MHC-I-opathy and/or MHC-II-opathy at any clinical stage or manifestation, the delay of the onset or evolution or aggravation or deterioration of the symptoms or signs of an MHC-I-opathy and/or MHC-II-opathy, and/or preventing and/or reducing the severity of an MHC-I-opathy and/or MHC-II-opathy.

In some embodiments, the antisense nucleic acid molecules targeted to ERAP2 comprise or consist of the nucleotide sequences shown in Table 1.

Table 1

Sequence	SEQ ID NO:
AUCUGCCUGUUUGAGUUGGG	1
UAUCUGCCUGUUUGAGUUGG	2
UUAUCUGCCUGUUUGAGUUG	3
CUUUAUCUGCCUGUUUGAGU	4
UGAUCUCUCUUUAUCUGCCU	5
UUGAUCUCUCUUUAUCUGCC	6
UCAUGCUCAUCUCUUUGAUC	7
CUCAUGCUCAUCUCUUUGAU	8
GGACUGUAUCUCAUGCUCAU	9
GGGACUGUAUCUCAUGCUCA	10
CGGGCAGCUUCUGUGAUCUU	11
UUUAAUCAAACACCUCCCGG	12
AUUUAAUCAAACACCUCCCG	13
CUCCAGUUAUGUCACAUGGG	14
GGCUCCAGUUAUGUCACAUG	15
CUGGCUCCAGUUAUGUCACA	16
AGUUCUUCAUGGCACUGCAC	17

UAGUUCUUCAUGGCACUGCA	18
AGACAAGUUAAUAUCCAGGC	19
GCAGAAGAAUGGAACAUGAA	20
GUGUGAAUUAACCAUUGCAG	21
CUGUGUGAAUUAACCAUUGC	22
AGCAGUAAAAUCCUCUGUGA	23
AAGCAGUAAAAUCCUCUGUG	24
GGCUGUUAAGCAGUAAAAUC	25
GAUGGCUGUUAAGCAGUAAA	26
AAGAUGGCUGUUAAGCAGUA	27
CAAGAUGGCUGUUAAGCAGU	28
CACUGAGAACUGAGAACAAA	29
AUGGCACUGAGAACUGAGAA	30
GAUGGCACUGAGAACUGAGA	31
UAGAUGGCACUGAGAACUGA	32
CUAGAUGGCACUGAGAACUG	33
GAUAACUAGAUGGCACUGAG	34
UGAUAACUAGAUGGCACUGA	35
GUGAUAACUAGAUGGCACUG	36
GAAGUGAUAACUAGAUGGCA	37
UGAAGUGAUAACUAGAUGGC	38
GUGAAGUGAUAACUAGAUGG	39
CUCAGUGAAGUGAUAACUAG	40
CCUCAGUGAAGUGAUAACUA	41
UCCUCAGUGAAGUGAUAACU	42
GAUCCUCAGUGAAGUGAUAA	43
UUAGUGGCUACUGGGAAAGC	44
AUUAGUGGCUACUGGGAAAG	45
CAUUAGUGGCUACUGGGAAA	46
CCAUUAGUGGCUACUGGGAA	47

In some embodiments, the antisense nucleic acid molecules targeted to ERAP2 comprise or consist of the nucleotide sequences shown in Table 2.

Table 2

Sequence	SEQ ID NO:
AUCUUAAAUCAUGCUGCUGC	48
CUCCAGUUAUGUCACAUGGG	49
GGCUCCAGUUAUGUCACAUG	50
CUGGCUCCAGUUAUGUCACA	51
AGUUCUUCAUGGCACUGCAC	52
UAGUUCUUCAUGGCACUGCA	53
AGACAAGUUAAUAUCCAGGC	54
GCAGAAGAAUGGAACAUGAA	55
GUGUGAAUUAACCAUUGCAG	56
CUGUGUGAAUUAACCAUUGC	57
AGCAGUAAAAUCCUCUGUGA	58
AAGCAGUAAAAUCCUCUGUG	59
GGCUGUUAAGCAGUAAAAUC	60
GAUGGCUGUUAAGCAGUAAA	61
AAGAUGGCUGUUAAGCAGUA	62
CAAGAUGGCUGUUAAGCAGU	63
CACUGAGAACUGAGAACAAA	64
AUGGCACUGAGAACUGAGAA	65
GAUGGCACUGAGAACUGAGA	66
UAGAUGGCACUGAGAACUGA	67
CUAGAUGGCACUGAGAACUG	68
GAUAACUAGAUGGCACUGAG	69
UGAUAACUAGAUGGCACUGA	70
GUGAUAACUAGAUGGCACUG	71
GAAGUGAUAACUAGAUGGCA	72

UGAAGUGAUAACUAGAUGGC	73
GUGAAGUGAUAACUAGAUGG	74
CUCAGUGAAGUGAUAACUAG	75
CCUCAGUGAAGUGAUAACUA	76
UCCUCAGUGAAGUGAUAACU	77
GAUCCUCAGUGAAGUGAUAA	78
UUAGUGGCUACUGGGAAAGC	79
AUUAGUGGCUACUGGGAAAG	80
CAUUAGUGGCUACUGGGAAA	81
CCAUUAGUGGCUACUGGGAA	82
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GGAUUACAGGCGUGAGCCAC	637
GGGAUUACAGGCGUGAGCCA	638
UGGGAUUACAGGCGUGAGCC	639
CUGGGAUUACAGGCGUGAGC	640
GCUGGGAUUACAGGCGUGAG	641
UGCUGGGAUUACAGGCGUGA	642
GUGCUGGGAUUACAGGCGUG	643
AGUGCUGGGAUUACAGGCGU	644
AAGUGCUGGGAUUACAGGCG	645
AAAGUGCUGGGAUUACAGGC	646
CAAAGUGCUGGGAUUACAGG	647
CCAAAGUGCUGGGAUUACAG	648
UCUCAGCCUCCCAAAGUGCU	649
UUCUCAGCCUCCCAAAGUGC	650
CUUCUCAGCCUCCCAAAGUG	651
CCUUCUCAGCCUCCCAAAGU	652

CCCUUCUCAGCCUCCCAAAG	653
AUCUCCUGACCUCGUGAUCC	654
CAUCUCCUGACCUCGUGAUC	655
CCAUCUCCUGACCUCGUGAU	656
UCCAUCUCCUGACCUCGUGA	657
CUCCAUCUCCUGACCUCGUG	658
UCUCCAUCUCCUGACCUCGU	659
CUCACCGUGUUAGCCAGGAU	660
UCUCACCGUGUUAGCCAGGA	661
GUCUCACCGUGUUAGCCAGG	662
GGUCUCACCGUGUUAGCCAG	663
GGGUCUCACCGUGUUAGCCA	664
UACAGGCACCUGCCACCAUG	665
CUACAGGCACCUGCCACCAU	666
ACUACAGGCACCUGCCACCA	667
AGCUGGGACUACAGGCACCU	668
UAGCUGGGACUACAGGCACC	669
CUGCCGAGUAGCUGGGACUA	670
CUCACUGCAAGCUCCACCUC	671
GCUCACUGCAAGCUCCACCU	672
UCGGCUCACUGCAAGCUCCA	673
UCUCGGCUCACUGCAAGCUC	674
AUCUCGGCUCACUGCAAGCU	675
AAUCUCGGCUCACUGCAAGC	676
CAAUCUCGGCUCACUGCAAG	677
GCAAUCUCGGCUCACUGCAA	678
GUGCAAUCUCGGCUCACUGC	679
GGUGCAAUCUCGGCUCACUG	680
UGGUGCAAUCUCGGCUCACU	681

GUGGUGCAAUCUCGGCUCAC	682
AGUGGUGCAAUCUCGGCUCA	683
CAGUGGUGCAAUCUCGGCUC	684
GCAGUGGUGCAAUCUCGGCU	685
AGGCUGGAAUGCAGUGGUGC	686
CAGGCUGGAAUGCAGUGGUG	687
CCAGGCUGGAAUGCAGUGGU	688
ACCCAGGCUGGAAUGCAGUG	689
CACCCAGGCUGGAAUGCAGU	690
UCACCCAGGCUGGAAUGCAG	691
GUCACCCAGGCUGGAAUGCA	692
AGUCACCCAGGCUGGAAUGC	693
CAGUCACCCAGGCUGGAAUG	694
UCAGUCACCCAGGCUGGAAU	695
CUCAGUCACCCAGGCUGGAA	696
GACAGAGUCUCGCUCAGUCA	697
GUCAUGUCUGAUGUACCUUU	698
AGUCAUGUCUGAUGUACCUU	699
CAGUCAUGUCUGAUGUACCU	700
GCAGUCAUGUCUGAUGUACC	701
GGCAGUCAUGUCUGAUGUAC	702
AGGCAGUCAUGUCUGAUGUA	703
CAGGCAGUCAUGUCUGAUGU	704
GCAGGCAGUCAUGUCUGAUG	705
UGCAGGCAGUCAUGUCUGAU	706
CUGUUUGACUUCAUGCAGGC	707
UACCCUGUUUGACUUCAUGC	708
GUUACCCUGUUUGACUUCAU	709
GGUGUUACCCUGUUUGACUU	710

UGGUGUUACCCUGUUUGACU	711
CUGGUGUUACCCUGUUUGAC	712
ACUGGUGUUACCCUGUUUGA	713
CACUGGUGUUACCCUGUUUG	714
UCACUGGUGUUACCCUGUUU	715
UUCACUGGUGUUACCCUGUU	716
CUUCACUGGUGUUACCCUGU	717
AGCUUCACUGGUGUUACCCU	718
UUGAGCUUCACUGGUGUUAC	719
CUUGAGCUUCACUGGUGUUA	720
ACUUGAGCUUCACUGGUGUU	721
GACUUGAGCUUCACUGGUGU	722
CUCUUGACUUGAGCUUCACU	723
UCCACAGCUCUUGACUUGAG	724
AUCCACAGCUCUUGACUUGA	725
UAUCCACAGCUCUUGACUUG	726
GACAAAAUAUCCACAGCUCU	727
AGACAAAAUAUCCACAGCUC	728
GACUUCUUGUUGGUUAGACA	729
UGAGACUUCUUGUUGGUUAG	730
CUUGAGACUUCUUGUUGGUU	731
UCUUGAGACUUCUUGUUGGU	732
UUCUUGAGACUUCUUGUUGG	733
GCCUGAUUUCUUGAGACUUC	734
UGCCUGAUUUCUUGAGACUU	735
CAUCUUUUGGCUGGAUACGA	736
GCAUCUUUUGGCUGGAUACG	737
CCACCACUCCAUCAUCAUGC	738
CCCACCACUCCAUCAUCAUG	739

GAUACUGAACUGGCCUUCCC	740
UAGGAUACUGAACUGGCCUU	741
AUAGGAUACUGAACUGGCCU	742
AAUAGGAUACUGAACUGGCC	743
AAAUAGGAUACUGAACUGGC	744
GGAGAAAGUCACUUGAGGCU	745
UGGAGAAAGUCACUUGAGGC	746
AAUGGAGAAAGUCACUUGAG	747
AGCAAUGGAGAAAGUCACUU	748
UAGCGUGAAGCAAUGGAGAA	749
AUAGCGUGAAGCAAUGGAGA	750
CAUAGCGUGAAGCAAUGGAG	751
GCAUAGCGUGAAGCAAUGGA	752
GGCAUAGCGUGAAGCAAUGG	753
UGGCAUAGCGUGAAGCAAUG	754
GUGGCAUAGCGUGAAGCAAU	755
AGUGGCAUAGCGUGAAGCAA	756
UAGUGGCAUAGCGUGAAGCA	757
AUAGUGGCAUAGCGUGAAGC	758
AAUAGUGGCAUAGCGUGAAG	759
AAAUAGUGGCAUAGCGUGAA	760
AAAAUAGUGGCAUAGCGUGA	761
GCAAAAUAGUGGCAUAGCGU	762
AGCAAAAUAGUGGCAUAGCG	763
AAGCAAAAUAGUGGCAUAGC	764
AACUGGUUGGGCAAACUAUA	765
AAACUGGUUGGGCAAACUAU	766
AAAACUGGUUGGGCAAACUA	767
UAAAACUGGUUGGGCAAACU	768

GUAAAACUGGUUGGGCAAAC	769
CGUAAAACUGGUUGGGCAAA	770
GACGUAAAACUGGUUGGGCA	771
GGACGUAAAACUGGUUGGGC	772
UGGACGUAAAACUGGUUGGG	773
UUGGACGUAAAACUGGUUGG	774
CUUGGACGUAAAACUGGUUG	775
CCUUGGACGUAAAACUGGUU	776
UCCUUGGACGUAAAACUGGU	777
UUCCUUGGACGUAAAACUGG	778
UUUCCUUGGACGUAAAACUG	779
GGCUAAUUUUCCUUGGACGU	780
UGGCUAAUUUUCCUUGGACG	781
GCAUUGGCUAAUUUUCCUUG	782
UUCCUGAUCCUUGCCUUUCA	783
UCUGGUUUCCUGAUCCUUGC	784
UCUCUGGUUUCCUGAUCCUU	785
GUCUCUGGUUUCCUGAUCCU	786
AGUCUCUGGUUUCCUGAUCC	787
AAGUCUCUGGUUUCCUGAUC	788
CCUGACACCUAGUUUCUAAU	789
AACCCUGACACCUAGUUUCU	790
AUAAACCCUGACACCUAGUU	791
GAUAAACCCUGACACCUAGU	792
UGAUAAACCCUGACACCUAG	793
CUUGAUAAACCCUGACACCU	794
UCUUGAUAAACCCUGACACC	795
UUCUUGAUAAACCCUGACAC	796
CUUCUUGAUAAACCCUGACA	797

CCUUCUUGAUAAACCCUGAC	798
GCCUUCUUGAUAAACCCUGA	799
GGCCUUCUUGAUAAACCCUG	800
UGGCCUUCUUGAUAAACCCU	801
UUCCUGGCCUUCUUGAUAAA	802
CAGAGACCAUUCAUUUGGAA	803
CCAGAGACCAUUCAUUUGGA	804
ACCAGAGACCAUUCAUUUGG	805
UGACCAGAGACCAUUCAUUU	806
UUGACCAGAGACCAUUCAUU	807
UUUGACCAGAGACCAUUCAU	808
AUUUGACCAGAGACCAUUCA	809
CAUUUGACCAGAGACCAUUC	810
AUUCAUUUGACCAGAGACCA	811
UUGUAUCUCUGUGAGGGCAG	812
GUAAGGCUUAAACCAAAUGG	813
CAGAUUCGACUUCAUUUGGA	814
GGCAGAUUCGACUUCAUUUG	815
GGGCAGAUUCGACUUCAUUU	816
AGGGCAGAUUCGACUUCAUU	817
GAGGGCAGAUUCGACUUCAU	818
UGAGGGCAGAUUCGACUUCA	819
GUGAGGGCAGAUUCGACUUC	820
UGUGAGGGCAGAUUCGACUU	821
CUGUGAGGGCAGAUUCGACU	822
CUUGUGUCUCUGUGAGGGCA	823
UUCUUGUGUCUCUGUGAGGG	824
AUUUCAGAGAGCAAGAAGCG	825
CAUUUCAGAGAGCAAGAAGC	826

GGCAUUUCAGAGAGCAAGAA	827
CAUUUAGCAGGGCAUUUCAG	828
GCAUUUAGCAGGGCAUUUCA	829
AGCAUUUAGCAGGGCAUUUC	830
AGAAGCAUUUAGCAGGGCAU	831
GAGAAGCAUUUAGCAGGGCA	832
CUCUUGACCCAACAUCCCAA	833
CCUCUUGACCCAACAUCCCA	834
UGGAUUGACUAACACUUUCC	835
GUGGAUUGACUAACACUUUC	836
CUGUGGCAUCAAAGAAAGGU	837
UCUGACUGUGGCAUCAAAGA	838
UCUCUGACUGUGGCAUCAAA	839
GGCCACCCAAACUGUAUUCU	840
ACCACCAAGUCAAAGUUAGA	841
CACCACCAAGUCAAAGUUAG	842
GUCCACCACCAAGUCAAAGU	843
GGUCCACCACCAAGUCAAAG	844
AGGAAGGUCCACCACCAAGU	845
AAGGAAGGUCCACCACCAAG	846
CAAGGAAGGUCCACCACCAA	847
CCAAGGAAGGUCCACCACCA	848
ACCAAGGAAGGUCCACCACC	849
AACCAAGGAAGGUCCACCAC	850
AAACCAAGGAAGGUCCACCA	851
GUUGAUGGUACAGUUCUACU	852
UGUUGAUGGUACAGUUCUAC	853
CAUUGUUGAUGGUACAGUUC	854
GGAAACAUUGUUGAUGGUAC	855

GUAUUGGCACAGUAAUUCUC	856
CAGUAUUGGCACAGUAAUUC	857
UCAAAACAGUAUUGGCACAG	858

In some embodiments, the siRNA molecules targeted to ERAP2 comprise or consist of the nucleotide sequences (sense and antisense strands) shown in Table 3.

Table 3

Sense Sequence	**SEQ ID NO:**	**Antisense Sequence**	**SEQ ID NO:**
AAAGAUCACAGAAGCUGCC	859	GGCAGCUUCUGUGAUCUUU	860
AAGAUCACAGAAGCUGCCC	861	GGGCAGCUUCUGUGAUCUU	862
AGAUCACAGAAGCUGCCCG	863	CGGGCAGCUUCUGUGAUCU	864
CCGGGAGGUGUUUGAUUAA	865	UUAAUCAAACACCUCCCGG	866
CGGGAGGUGUUUGAUUAAA	867	UUUAAUCAAACACCUCCCG	868
GGGAGGUGUUUGAUUAAAU	869	AUUUAAUCAAACACCUCCC	870
GGAGGUGUUUGAUUAAAUU	871	AAUUUAAUCAAACACCUCC	872
GAGGUGUUUGAUUAAAUUC	873	GAAUUUAAUCAAACACCUC	874
AUGUGACAUAACUGGAGCC	875	GGCUCCAGUUAUGUCACAU	876
UGUGACAUAACUGGAGCCA	877	UGGCUCCAGUUAUGUCACA	878
UGGAGCCAGUGCAGUGCCA	879	UGGCACUGCACUGGCUCCA	880
GCCAGUGCAGUGCCAUGAA	881	UUCAUGGCACUGCACUGGC	882
CCAGUGCAGUGCCAUGAAG	883	CUUCAUGGCACUGCACUGG	884
CAGUGCAGUGCCAUGAAGA	885	UCUUCAUGGCACUGCACUG	886
AGUGCAGUGCCAUGAAGAA	887	UUCUUCAUGGCACUGCACU	888
AGAUUAGCCUGGAUAUUAA	889	UUAAUAUCCAGGCUAAUCU	890
GAUUAGCCUGGAUAUUAAC	891	GUUAAUAUCCAGGCUAAUC	892
AUUAGCCUGGAUAUUAACU	893	AGUUAAUAUCCAGGCUAAU	894
UUAGCCUGGAUAUUAACUU	895	AAGUUAAUAUCCAGGCUAA	896
UAGCCUGGAUAUUAACUUG	897	CAAGUUAAUAUCCAGGCUA	898
GCCUGGAUAUUAACUUGUC	899	GACAAGUUAAUAUCCAGGC	900
CCUGGAUAUUAACUUGUCU	901	AGACAAGUUAAUAUCCAGG	902
CUGGAUAUUAACUUGUCUU	903	AAGACAAGUUAAUAUCCAG	904

UGGAUAUUAACUUGUCUUC	905	GAAGACAAGUUAAUAUCCA	906
GGAUAUUAACUUGUCUUCU	907	AGAAGACAAGUUAAUAUCC	908
AUUAACUUGUCUUCUAGAG	909	CUCUAGAAGACAAGUUAAU	910
AGAAUAGAUUUCAUGUUCC	911	GGAACAUGAAAUCUAUUCU	912
GAAUAGAUUUCAUGUUCCA	913	UGGAACAUGAAAUCUAUUC	914
UAGAUUUCAUGUUCCAUUC	915	GAAUGGAACAUGAAAUCUA	916
GAUUUCAUGUUCCAUUCUU	917	AAGAAUGGAACAUGAAAUC	918
AUUUCAUGUUCCAUUCUUC	919	GAAGAAUGGAACAUGAAAU	920
UUUCAUGUUCCAUUCUUCU	921	AGAAGAAUGGAACAUGAAA	922
UUCAUGUUCCAUUCUUCUG	923	CAGAAGAAUGGAACAUGAA	924
UCAUGUUCCAUUCUUCUGC	925	GCAGAAGAAUGGAACAUGA	926
CAUGUUCCAUUCUUCUGCA	927	UGCAGAAGAAUGGAACAUG	928
UCUUCUGCAAUGGUUAAUU	929	AAUUAACCAUUGCAGAAGA	930
UUCUGCAAUGGUUAAUUCA	931	UGAAUUAACCAUUGCAGAA	932
CAUUCACAGAGGAUUUUAC	933	GUAAAAUCCUCUGUGAAUG	934
AUUCACAGAGGAUUUUACU	935	AGUAAAAUCCUCUGUGAAU	936
ACAGAGGAUUUUACUGCUU	937	AAGCAGUAAAAUCCUCUGU	938
CAGAGGAUUUUACUGCUUA	939	UAAGCAGUAAAAUCCUCUG	940
AGAGGAUUUUACUGCUUAA	941	UUAAGCAGUAAAAUCCUCU	942
GAGGAUUUUACUGCUUAAC	943	GUUAAGCAGUAAAAUCCUC	944
AUAUGCAUUUGUUCUCAGU	945	ACUGAGAACAAAUGCAUAU	946
AUGCAUUUGUUCUCAGUUC	947	GAACUGAGAACAAAUGCAU	948
UUUGUUCUCAGUUCUCAGU	949	ACUGAGAACUGAGAACAAA	950
UUGUUCUCAGUUCUCAGUG	951	CACUGAGAACUGAGAACAA	952
GUUCUCAGUUCUCAGUGCC	953	GGCACUGAGAACUGAGAAC	954
UUCUCAGUUCUCAGUGCCA	955	UGGCACUGAGAACUGAGAA	956
CAGUUCUCAGUGCCAUCUA	957	UAGAUGGCACUGAGAACUG	958
AGUUCUCAGUGCCAUCUAG	959	CUAGAUGGCACUGAGAACU	960
UCAGUGCCAUCUAGUUAUC	961	GAUAACUAGAUGGCACUGA	962
CAGUGCCAUCUAGUUAUCA	963	UGAUAACUAGAUGGCACUG	964
AGUGCCAUCUAGUUAUCAC	965	GUGAUAACUAGAUGGCACU	966

GUGCCAUCUAGUUAUCACU	967	AGUGAUAACUAGAUGGCAC	968
UGCCAUCUAGUUAUCACUU	969	AAGUGAUAACUAGAUGGCA	970
GCCAUCUAGUUAUCACUUC	971	GAAGUGAUAACUAGAUGGC	972
CCAUCUAGUUAUCACUUCA	973	UGAAGUGAUAACUAGAUGG	974
CAUCUAGUUAUCACUUCAC	975	GUGAAGUGAUAACUAGAUG	976
AUCUAGUUAUCACUUCACU	977	AGUGAAGUGAUAACUAGAU	978
UCUAGUUAUCACUUCACUG	979	CAGUGAAGUGAUAACUAGA	980
CUAGUUAUCACUUCACUGA	981	UCAGUGAAGUGAUAACUAG	982
UAGUUAUCACUUCACUGAG	983	CUCAGUGAAGUGAUAACUA	984
AGUUAUCACUUCACUGAGG	985	CCUCAGUGAAGUGAUAACU	986
GUUAUCACUUCACUGAGGA	987	UCCUCAGUGAAGUGAUAAC	988
UUAUCACUUCACUGAGGAU	989	AUCCUCAGUGAAGUGAUAA	990
UAUCACUUCACUGAGGAUC	991	GAUCCUCAGUGAAGUGAUA	992
AUCACUUCACUGAGGAUCC	993	GGAUCCUCAGUGAAGUGAU	994
UCACUUCACUGAGGAUCCU	995	AGGAUCCUCAGUGAAGUGA	996
GCUUUCCCAGUAGCCACUA	997	UAGUGGCUACUGGGAAAGC	998
CUUUCCCAGUAGCCACUAA	999	UUAGUGGCUACUGGGAAAG	1000
UUUCCCAGUAGCCACUAAU	1001	AUUAGUGGCUACUGGGAAA	1002
UUCCCAGUAGCCACUAAUG	1003	CAUUAGUGGCUACUGGGAA	1004
UCCCAGUAGCCACUAAUGG	1005	CCAUUAGUGGCUACUGGGA	1006
CUUGGCAGGAGCUAAGGCU	1007	AGCCUUAGCUCCUGCCAAG	1008

In some embodiments, the siRNA molecules targeted to ERAP2 comprise or consist of the nucleotide sequences (sense and antisense strands) shown in Table 4.

Table 4

Sense Sequence	**SEQ ID NO:**	**Antisense Sequence**	**SEQ ID NO:**
UCAAAUCUGCAGCAGCAUG	1009	CAUGCUGCUGCAGAUUUGA	1010
CAAAUCUGCAGCAGCAUGA	1011	UCAUGCUGCUGCAGAUUUG	1012
AAAUCUGCAGCAGCAUGAU	1013	AUCAUGCUGCUGCAGAUUU	1014
AUGUGACAUAACUGGAGCC	1015	GGCUCCAGUUAUGUCACAU	1016
UGUGACAUAACUGGAGCCA	1017	UGGCUCCAGUUAUGUCACA	1018

UGGAGCCAGUGCAGUGCCA	1019	UGGCACUGCACUGGCUCCA	1020
GCCAGUGCAGUGCCAUGAA	1021	UUCAUGGCACUGCACUGGC	1022
CCAGUGCAGUGCCAUGAAG	1023	CUUCAUGGCACUGCACUGG	1024
CAGUGCAGUGCCAUGAAGA	1025	UCUUCAUGGCACUGCACUG	1026
AGUGCAGUGCCAUGAAGAA	1027	UUCUUCAUGGCACUGCACU	1028
AGAUUAGCCUGGAUAUUAA	1029	UUAAUAUCCAGGCUAAUCU	1030
GAUUAGCCUGGAUAUUAAC	1031	GUUAAUAUCCAGGCUAAUC	1032
AUUAGCCUGGAUAUUAACU	1033	AGUUAAUAUCCAGGCUAAU	1034
UUAGCCUGGAUAUUAACUU	1035	AAGUUAAUAUCCAGGCUAA	1036
UAGCCUGGAUAUUAACUUG	1037	CAAGUUAAUAUCCAGGCUA	1038
GCCUGGAUAUUAACUUGUC	1039	GACAAGUUAAUAUCCAGGC	1040
CCUGGAUAUUAACUUGUCU	1041	AGACAAGUUAAUAUCCAGG	1042
CUGGAUAUUAACUUGUCUU	1043	AAGACAAGUUAAUAUCCAG	1044
UGGAUAUUAACUUGUCUUC	1045	GAAGACAAGUUAAUAUCCA	1046
GGAUAUUAACUUGUCUUCU	1047	AGAAGACAAGUUAAUAUCC	1048
AUUAACUUGUCUUCUAGAG	1049	CUCUAGAAGACAAGUUAAU	1050
AGAAUAGAUUUCAUGUUCC	1051	GGAACAUGAAAUCUAUUCU	1052
GAAUAGAUUUCAUGUUCCA	1053	UGGAACAUGAAAUCUAUUC	1054
UAGAUUUCAUGUUCCAUUC	1055	GAAUGGAACAUGAAAUCUA	1056
GAUUUCAUGUUCCAUUCUU	1057	AAGAAUGGAACAUGAAAUC	1058
AUUUCAUGUUCCAUUCUUC	1059	GAAGAAUGGAACAUGAAAU	1060
UUUCAUGUUCCAUUCUUCU	1061	AGAAGAAUGGAACAUGAAA	1062
UUCAUGUUCCAUUCUUCUG	1063	CAGAAGAAUGGAACAUGAA	1064
UCAUGUUCCAUUCUUCUGC	1065	GCAGAAGAAUGGAACAUGA	1066
CAUGUUCCAUUCUUCUGCA	1067	UGCAGAAGAAUGGAACAUG	1068
UCUUCUGCAAUGGUUAAUU	1069	AAUUAACCAUUGCAGAAGA	1070
UUCUGCAAUGGUUAAUUCA	1071	UGAAUUAACCAUUGCAGAA	1072
CAUUCACAGAGGAUUUUAC	1073	GUAAAAUCCUCUGUGAAUG	1074
AUUCACAGAGGAUUUUACU	1075	AGUAAAAUCCUCUGUGAAU	1076
ACAGAGGAUUUUACUGCUU	1077	AAGCAGUAAAAUCCUCUGU	1078
CAGAGGAUUUUACUGCUUA	1079	UAAGCAGUAAAAUCCUCUG	1080

AGAGGAUUUUACUGCUUAA	1081	UUAAGCAGUAAAAUCCUCU	1082
GAGGAUUUUACUGCUUAAC	1083	GUUAAGCAGUAAAAUCCUC	1084
AUAUGCAUUUGUUCUCAGU	1085	ACUGAGAACAAAUGCAUAU	1086
AUGCAUUUGUUCUCAGUUC	1087	GAACUGAGAACAAAUGCAU	1088
UUUGUUCUCAGUUCUCAGU	1089	ACUGAGAACUGAGAACAAA	1090
UUGUUCUCAGUUCUCAGUG	1091	CACUGAGAACUGAGAACAA	1092
GUUCUCAGUUCUCAGUGCC	1093	GGCACUGAGAACUGAGAAC	1094
UUCUCAGUUCUCAGUGCCA	1095	UGGCACUGAGAACUGAGAA	1096
CAGUUCUCAGUGCCAUCUA	1097	UAGAUGGCACUGAGAACUG	1098
AGUUCUCAGUGCCAUCUAG	1099	CUAGAUGGCACUGAGAACU	1100
UCAGUGCCAUCUAGUUAUC	1101	GAUAACUAGAUGGCACUGA	1102
CAGUGCCAUCUAGUUAUCA	1103	UGAUAACUAGAUGGCACUG	1104
AGUGCCAUCUAGUUAUCAC	1105	GUGAUAACUAGAUGGCACU	1106
GUGCCAUCUAGUUAUCACU	1107	AGUGAUAACUAGAUGGCAC	1108
UGCCAUCUAGUUAUCACUU	1109	AAGUGAUAACUAGAUGGCA	1110
GCCAUCUAGUUAUCACUUC	1111	GAAGUGAUAACUAGAUGGC	1112
CCAUCUAGUUAUCACUUCA	1113	UGAAGUGAUAACUAGAUGG	1114
CAUCUAGUUAUCACUUCAC	1115	GUGAAGUGAUAACUAGAUG	1116
AUCUAGUUAUCACUUCACU	1117	AGUGAAGUGAUAACUAGAU	1118
UCUAGUUAUCACUUCACUG	1119	CAGUGAAGUGAUAACUAGA	1120
CUAGUUAUCACUUCACUGA	1121	UCAGUGAAGUGAUAACUAG	1122
UAGUUAUCACUUCACUGAG	1123	CUCAGUGAAGUGAUAACUA	1124
AGUUAUCACUUCACUGAGG	1125	CCUCAGUGAAGUGAUAACU	1126
GUUAUCACUUCACUGAGGA	1127	UCCUCAGUGAAGUGAUAAC	1128
UUAUCACUUCACUGAGGAU	1129	AUCCUCAGUGAAGUGAUAA	1130
UAUCACUUCACUGAGGAUC	1131	GAUCCUCAGUGAAGUGAUA	1132
AUCACUUCACUGAGGAUCC	1133	GGAUCCUCAGUGAAGUGAU	1134
UCACUUCACUGAGGAUCCU	1135	AGGAUCCUCAGUGAAGUGA	1136
GCUUUCCCAGUAGCCACUA	1137	UAGUGGCUACUGGGAAAGC	1138
CUUUCCCAGUAGCCACUAA	1139	UUAGUGGCUACUGGGAAAG	1140
UUUCCCAGUAGCCACUAAU	1141	AUUAGUGGCUACUGGGAAA	1142

UUCCCAGUAGCCACUAAUG	1143	CAUUAGUGGCUACUGGGAA	1144
UCCCAGUAGCCACUAAUGG	1145	CCAUUAGUGGCUACUGGGA	1146
CUUGGCAGGAGCUAAGGCU	1147	AGCCUUAGCUCCUGCCAAG	1148
CCAGUGUGGUCAUUCCUCU	1149	AGAGGAAUGACCACACUGG	1150
GUCAUUCCUCUCCAUUAUG	1151	CAUAAUGGAGAGGAAUGAC	1152
CAUUCCUCUCCAUUAUGAC	1153	GUCAUAAUGGAGAGGAAUG	1154
AUUCCUCUCCAUUAUGACC	1155	GGUCAUAAUGGAGAGGAAU	1156
CUCUCCAUUAUGACCUCUU	1157	AAGAGGUCAUAAUGGAGAG	1158
UCUCCAUUAUGACCUCUUU	1159	AAAGAGGUCAUAAUGGAGA	1160
CUCCAUUAUGACCUCUUUG	1161	CAAAGAGGUCAUAAUGGAG	1162
UCCAUUAUGACCUCUUUGU	1163	ACAAAGAGGUCAUAAUGGA	1164
CCAUUAUGACCUCUUUGUC	1165	GACAAAGAGGUCAUAAUGG	1166
CAUUAUGACCUCUUUGUCC	1167	GGACAAAGAGGUCAUAAUG	1168
CCCAAUCUCACCUCUCUGG	1169	CCAGAGAGGUGAGAUUGGG	1170
UCUCACCUCUCUGGACUUU	1171	AAAGUCCAGAGAGGUGAGA	1172
CUCACCUCUCUGGACUUUG	1173	CAAAGUCCAGAGAGGUGAG	1174
UCACCUCUCUGGACUUUGU	1175	ACAAAGUCCAGAGAGGUGA	1176
UCUCUGGACUUUGUUGCAU	1177	AUGCAACAAAGUCCAGAGA	1178
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CUGGACUUUGUUGCAUCUG	1181	CAGAUGCAACAAAGUCCAG	1182
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AUUAUUUAGCCUCAAGUGA	2299	UCACUUGAGGCUAAAUAAU	2300
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AAGUGACUUUCUCCAUUGC	2303	GCAAUGGAGAAAGUCACUU	2304
AGUGACUUUCUCCAUUGCU	2305	AGCAAUGGAGAAAGUCACU	2306
GUGACUUUCUCCAUUGCUU	2307	AAGCAAUGGAGAAAGUCAC	2308
UUUCUCCAUUGCUUCACGC	2309	GCGUGAAGCAAUGGAGAAA	2310
UUCUCCAUUGCUUCACGCU	2311	AGCGUGAAGCAAUGGAGAA	2312
UCUCCAUUGCUUCACGCUA	2313	UAGCGUGAAGCAAUGGAGA	2314
CUCCAUUGCUUCACGCUAU	2315	AUAGCGUGAAGCAAUGGAG	2316
UCCAUUGCUUCACGCUAUG	2317	CAUAGCGUGAAGCAAUGGA	2318
CCAUUGCUUCACGCUAUGC	2319	GCAUAGCGUGAAGCAAUGG	2320

CAUUGCUUCACGCUAUGCC	2321	GGCAUAGCGUGAAGCAAUG	2322
AUUGCUUCACGCUAUGCCA	2323	UGGCAUAGCGUGAAGCAAU	2324
UUGCUUCACGCUAUGCCAC	2325	GUGGCAUAGCGUGAAGCAA	2326
UGCUUCACGCUAUGCCACU	2327	AGUGGCAUAGCGUGAAGCA	2328
GCUUCACGCUAUGCCACUA	2329	UAGUGGCAUAGCGUGAAGC	2330
CUUCACGCUAUGCCACUAU	2331	AUAGUGGCAUAGCGUGAAG	2332
UUCACGCUAUGCCACUAUU	2333	AAUAGUGGCAUAGCGUGAA	2334
UCACGCUAUGCCACUAUUU	2335	AAAUAGUGGCAUAGCGUGA	2336
CACGCUAUGCCACUAUUUU	2337	AAAAUAGUGGCAUAGCGUG	2338
CGCUAUGCCACUAUUUUGC	2339	GCAAAAUAGUGGCAUAGCG	2340
GCUAUGCCACUAUUUUGCU	2341	AGCAAAAUAGUGGCAUAGC	2342
CUAUGCCACUAUUUUGCUU	2343	AAGCAAAAUAGUGGCAUAG	2344
UAUGCCACUAUUUUGCUUC	2345	GAAGCAAAAUAGUGGCAUA	2346
AUGCCACUAUUUUGCUUCU	2347	AGAAGCAAAAUAGUGGCAU	2348
UGCCACUAUUUUGCUUCUU	2349	AAGAAGCAAAAUAGUGGCA	2350
AACCUUGCUUAGUAUUCUA	2351	UAGAAUACUAAGCAAGGUU	2352
CCUUGCUUAGUAUUCUAUA	2353	UAUAGAAUACUAAGCAAGG	2354
AGUAUUCUAUAGUUUGCCC	2355	GGGCAAACUAUAGAAUACU	2356
GUAUUCUAUAGUUUGCCCA	2357	UGGGCAAACUAUAGAAUAC	2358
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AUUCUAUAGUUUGCCCAAC	2361	GUUGGGCAAACUAUAGAAU	2362
UAUAGUUUGCCCAACCAGU	2363	ACUGGUUGGGCAAACUAUA	2364
AUAGUUUGCCCAACCAGUU	2365	AACUGGUUGGGCAAACUAU	2366
UAGUUUGCCCAACCAGUUU	2367	AAACUGGUUGGGCAAACUA	2368
AGUUUGCCCAACCAGUUUU	2369	AAAACUGGUUGGGCAAACU	2370
GUUUGCCCAACCAGUUUUA	2371	UAAAACUGGUUGGGCAAAC	2372
UUUGCCCAACCAGUUUUAC	2373	GUAAAACUGGUUGGGCAAA	2374
UUGCCCAACCAGUUUUACG	2375	CGUAAAACUGGUUGGGCAA	2376
UGCCCAACCAGUUUUACGU	2377	ACGUAAAACUGGUUGGGCA	2378
GCCCAACCAGUUUUACGUC	2379	GACGUAAAACUGGUUGGGC	2380
CCCAACCAGUUUUACGUCC	2381	GGACGUAAAACUGGUUGGG	2382

CCAACCAGUUUUACGUCCA	2383	UGGACGUAAAACUGGUUGG	2384
CAACCAGUUUUACGUCCAA	2385	UUGGACGUAAAACUGGUUG	2386
AACCAGUUUUACGUCCAAG	2387	CUUGGACGUAAAACUGGUU	2388
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ACUAGGUGUCAGGGUUUAU	2393	AUAAACCCUGACACCUAGU	2394
CUAGGUGUCAGGGUUUAUC	2395	GAUAAACCCUGACACCUAG	2396
UAGGUGUCAGGGUUUAUCA	2397	UGAUAAACCCUGACACCUA	2398
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CAGGAAGGCCUUUUGGGUU	2419	AACCCAAAAGGCCUUCCUG	2420
AGGAAGGCCUUUUGGGUUA	2421	UAACCCAAAAGGCCUUCCU	2422
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GAAGGCCUUUUGGGUUAAG	2425	CUUAACCCAAAAGGCCUUC	2426
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AGGCCUUUUGGGUUAAGCC	2429	GGCUUAACCCAAAAGGCCU	2430
GGCCUUUUGGGUUAAGCCU	2431	AGGCUUAACCCAAAAGGCC	2432
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UCCAAAUGAAUGGUCUCUG	2435	CAGAGACCAUUCAUUUGGA	2436
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CAAAUGAAUGGUCUCUGGU	2439	ACCAGAGACCAUUCAUUUG	2440
AAAUGAAUGGUCUCUGGUC	2441	GACCAGAGACCAUUCAUUU	2442
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UACCAUUUGGUUUAAGCCU	2483	AGGCUUAAACCAAAUGGUA	2484
ACCAUUUGGUUUAAGCCUU	2485	AAGGCUUAAACCAAAUGGU	2486
CCAUUUGGUUUAAGCCUUA	2487	UAAGGCUUAAACCAAAUGG	2488
CAUUUGGUUUAAGCCUUAC	2489	GUAAGGCUUAAACCAAAUG	2490
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UAGAUGCAGCACCAUAUUU	2533	AAAUAUGGUGCUGCAUCUA	2534
AGAUGCAGCACCAUAUUUU	2535	AAAAUAUGGUGCUGCAUCU	2536
GAUGCAGCACCAUAUUUUA	2537	UAAAAUAUGGUGCUGCAUC	2538
AUGCAGCACCAUAUUUUAU	2539	AUAAAAUAUGGUGCUGCAU	2540
AGCACCAUAUUUUAUAACC	2541	GGUUAUAAAAUAUGGUGCU	2542
GCACCAUAUUUUAUAACCC	2543	GGGUUAUAAAAUAUGGUGC	2544
ACCAUAUUUUAUAACCCAG	2545	CUGGGUUAUAAAAUAUGGU	2546
CCAUAUUUUAUAACCCAGC	2547	GCUGGGUUAUAAAAUAUGG	2548
CAUAUUUUAUAACCCAGCU	2549	AGCUGGGUUAUAAAAUAUG	2550
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CCAGCUUUAGCAUUUCUUC	2555	GAAGAAAUGCUAAAGCUGG	2556
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GCGCUUCUUGCUCUCUGAA	2559	UUCAGAGAGCAAGAAGCGC	2560
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AAUGCCCUGCUAAAUGCUU	2563	AAGCAUUUAGCAGGGCAUU	2564
AUGCCCUGCUAAAUGCUUC	2565	GAAGCAUUUAGCAGGGCAU	2566
UGCCCUGCUAAAUGCUUCU	2567	AGAAGCAUUUAGCAGGGCA	2568

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UGAAUAAGGUAGUUUGGAA	2573	UUCCAAACUACCUUAUUCA	2574
GAAUAAGGUAGUUUGGAAU	2575	AUUCCAAACUACCUUAUUC	2576
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AUCACUCUACAUACAGAUA	2581	UAUCUGUAUGUAGAGUGAU	2582
UCACUCUACAUACAGAUAG	2583	CUAUCUGUAUGUAGAGUGA	2584
CACUCUACAUACAGAUAGU	2585	ACUAUCUGUAUGUAGAGUG	2586
ACUCUACAUACAGAUAGUA	2587	UACUAUCUGUAUGUAGAGU	2588
CUCUACAUACAGAUAGUAA	2589	UUACUAUCUGUAUGUAGAG	2590
UGUAAAUGACUGAUGUUUG	2591	CAAACAUCAGUCAUUUACA	2592
GUAAAUGACUGAUGUUUGC	2593	GCAAACAUCAGUCAUUUAC	2594
UAAAUGACUGAUGUUUGCA	2595	UGCAAACAUCAGUCAUUUA	2596
AAAUGACUGAUGUUUGCAU	2597	AUGCAAACAUCAGUCAUUU	2598
GAUGUUUGCAUUAUUAAGG	2599	CCUUAAUAAUGCAAACAUC	2600
ACUUGGGAUGUUGGGUCAA	2601	UUGACCCAACAUCCCAAGU	2602
UUGGGAUGUUGGGUCAAGA	2603	UCUUGACCCAACAUCCCAA	2604
UGGGAUGUUGGGUCAAGAG	2605	CUCUUGACCCAACAUCCCA	2606
GGGAUGUUGGGUCAAGAGG	2607	CCUCUUGACCCAACAUCCC	2608
AAAGUGUUAGUCAAUCCAC	2609	GUGGAUUGACUAACACUUU	2610
GAAGGUCAAUUAUAAUUCC	2611	GGAAUUAUAAUUGACCUUC	2612
CCAUAUACCUUUCUUUGAU	2613	AUCAAAGAAAGGUAUAUGG	2614
CAUAUACCUUUCUUUGAUG	2615	CAUCAAAGAAAGGUAUAUG	2616
AUAUACCUUUCUUUGAUGC	2617	GCAUCAAAGAAAGGUAUAU	2618
UAUACCUUUCUUUGAUGCC	2619	GGCAUCAAAGAAAGGUAUA	2620
ACCUUUCUUUGAUGCCACA	2621	UGUGGCAUCAAAGAAAGGU	2622
AGAAUACAGUUUGGGUGGC	2623	GCCACCCAAACUGUAUUCU	2624
GAAUACAGUUUGGGUGGCC	2625	GGCCACCCAAACUGUAUUC	2626
AAUACAGUUUGGGUGGCCA	2627	UGGCCACCCAAACUGUAUU	2628
AUACAGUUUGGGUGGCCAU	2629	AUGGCCACCCAAACUGUAU	2630

UACAGUUUGGGUGGCCAUG	2631	CAUGGCCACCCAAACUGUA	2632
ACAGUUUGGGUGGCCAUGG	2633	CCAUGGCCACCCAAACUGU	2634
CAGUUUGGGUGGCCAUGGA	2635	UCCAUGGCCACCCAAACUG	2636
AGUUUGGGUGGCCAUGGAU	2637	AUCCAUGGCCACCCAAACU	2638
GUUUGGGUGGCCAUGGAUG	2639	CAUCCAUGGCCACCCAAAC	2640
UUUGGGUGGCCAUGGAUGU	2641	ACAUCCAUGGCCACCCAAA	2642
UCAGAUCAUUUCAUGGAAU	2643	AUUCCAUGAAAUGAUCUGA	2644
CAGAUCAUUUCAUGGAAUC	2645	GAUUCCAUGAAAUGAUCUG	2646
GAUCAUUUCAUGGAAUCUU	2647	AAGAUUCCAUGAAAUGAUC	2648
AAGUAUCUUUGACUCUAAC	2649	GUUAGAGUCAAAGAUACUU	2650
AGUAUCUUUGACUCUAACU	2651	AGUUAGAGUCAAAGAUACU	2652
GUAUCUUUGACUCUAACUU	2653	AAGUUAGAGUCAAAGAUAC	2654
AUCUUUGACUCUAACUUUG	2655	CAAAGUUAGAGUCAAAGAU	2656
UCUUUGACUCUAACUUUGA	2657	UCAAAGUUAGAGUCAAAGA	2658
CUUUGACUCUAACUUUGAC	2659	GUCAAAGUUAGAGUCAAAG	2660
UUUGACUCUAACUUUGACU	2661	AGUCAAAGUUAGAGUCAAA	2662
UUGACUCUAACUUUGACUU	2663	AAGUCAAAGUUAGAGUCAA	2664
CUCUAACUUUGACUUGGUG	2665	CACCAAGUCAAAGUUAGAG	2666
UCUAACUUUGACUUGGUGG	2667	CCACCAAGUCAAAGUUAGA	2668
CUAACUUUGACUUGGUGGU	2669	ACCACCAAGUCAAAGUUAG	2670
UAACUUUGACUUGGUGGUG	2671	CACCACCAAGUCAAAGUUA	2672
ACUUUGACUUGGUGGUGGA	2673	UCCACCACCAAGUCAAAGU	2674
CUUUGACUUGGUGGUGGAC	2675	GUCCACCACCAAGUCAAAG	2676
UUUGACUUGGUGGUGGACC	2677	GGUCCACCACCAAGUCAAA	2678
UUGACUUGGUGGUGGACCU	2679	AGGUCCACCACCAAGUCAA	2680
UGACUUGGUGGUGGACCUU	2681	AAGGUCCACCACCAAGUCA	2682
UUGGUGGUGGACCUUCCUU	2683	AAGGAAGGUCCACCACCAA	2684
UGGUGGUGGACCUUCCUUG	2685	CAAGGAAGGUCCACCACCA	2686
GGUGGUGGACCUUCCUUGG	2687	CCAAGGAAGGUCCACCACC	2688
GUGGUGGACCUUCCUUGGU	2689	ACCAAGGAAGGUCCACCAC	2690
UGGUGGACCUUCCUUGGUU	2691	AACCAAGGAAGGUCCACCA	2692

GGUGGACCUUCCUUGGUUU	2693	AAACCAAGGAAGGUCCACC	2694
GUGGACCUUCCUUGGUUUU	2695	AAAACCAAGGAAGGUCCAC	2696
CAUGUAUUUUAGCAUAAGG	2697	CCUUAUGCUAAAAUACAUG	2698
AAAUGUGUAUGAGUUUCAG	2699	CUGAAACUCAUACACAUUU	2700
AAUGUGUAUGAGUUUCAGU	2701	ACUGAAACUCAUACACAUU	2702
AUGUGUAUGAGUUUCAGUA	2703	UACUGAAACUCAUACACAU	2704
GUGUAUGAGUUUCAGUAGA	2705	UCUACUGAAACUCAUACAC	2706
UGUAUGAGUUUCAGUAGAA	2707	UUCUACUGAAACUCAUACA	2708
UUCCAUAAAUAUGCAGAGU	2709	ACUCUGCAUAUUUAUGGAA	2710
CCAUAAAUAUGCAGAGUUC	2711	GAACUCUGCAUAUUUAUGG	2712
UGAAUGCAUUUGCAAUUUC	2713	GAAAUUGCAAAUGCAUUCA	2714
GAAUGCAUUUGCAAUUUCU	2715	AGAAAUUGCAAAUGCAUUC	2716
AUGCAUUUGCAAUUUCUAG	2717	CUAGAAAUUGCAAAUGCAU	2718
UGCAUUUGCAAUUUCUAGG	2719	CCUAGAAAUUGCAAAUGCA	2720
GCAUUUGCAAUUUCUAGGA	2721	UCCUAGAAAUUGCAAAUGC	2722
CAUUUGCAAUUUCUAGGAU	2723	AUCCUAGAAAUUGCAAAUG	2724
UUUGCAAUUUCUAGGAUUC	2725	GAAUCCUAGAAAUUGCAAA	2726
UGCAAUUUCUAGGAUUCUA	2727	UAGAAUCCUAGAAAUUGCA	2728
GCAAUUUCUAGGAUUCUAA	2729	UUAGAAUCCUAGAAAUUGC	2730
UCUAAAGAAUUGAGUACAG	2731	CUGUACUCAAUUCUUUAGA	2732
CUAAAGAAUUGAGUACAGA	2733	UCUGUACUCAAUUCUUUAG	2734
AGAAUUACUGUGCCAAUAC	2735	GUAUUGGCACAGUAAUUCU	2736
GAAUUACUGUGCCAAUACU	2737	AGUAUUGGCACAGUAAUUC	2738
AAUUACUGUGCCAAUACUG	2739	CAGUAUUGGCACAGUAAUU	2740
CUGUGCCAAUACUGUUUUG	2741	CAAAACAGUAUUGGCACAG	2742
UGUGCCAAUACUGUUUUGA	2743	UCAAAACAGUAUUGGCACA	2744
GUGCCAAUACUGUUUUGAU	2745	AUCAAAACAGUAUUGGCAC	2746
GCCAAUACUGUUUUGAUAA	2747	UUAUCAAAACAGUAUUGGC	2748

In some embodiments, the antisense nucleic acid molecules targeted to ERAP1 comprise or consist of the nucleotide sequences shown in Table 5.

Table 5

Sequence	SEQ ID NO:
CGCCUUUCUGAACGCGGAUC	2749
ACGCCUUUCUGAACGCGGAU	2750
CACGCCUUUCUGAACGCGGA	2751
UGCACGCCUUUCUGAACGCG	2752
GAUCAGGCGUAGGAAGUGCA	2753
GGAUCAGGCGUAGGAAGUGC	2754
GGGAUCAGGCGUAGGAAGUG	2755
GAAGCUGCGAGGUUGCGAUG	2756
GGAAGCUGCGAGGUUGCGAU	2757
GGGAAUUGGUAAAUGAGCGC	2758
AGGGAAUUGGUAAAUGAGCG	2759
GAAGGGAAUUGGUAAAUGAG	2760
GGAAGGGAAUUGGUAAAUGA	2761
CCAGGAAGGGAAUUGGUAAA	2762
CCCAGGAAGGGAAUUGGUAA	2763
AAGCCGCAACUCCCAGGAAG	2764
GAAGCCGCAACUCCCAGGAA	2765
AAAGGGUAAACGGGAGUGGG	2766
GAAAGGGUAAACGGGAGUGG	2767
GGAAAGGGUAAACGGGAGUG	2768
GAAAGUGAAAGUGGAGCCCG	2769
GGACCGAAAGUGAAAGUGGA	2770
AGGACCGAAAGUGAAAGUGG	2771
CAGGACCGAAAGUGAAAGUG	2772
CCAGGACCGAAAGUGAAAGU	2773
UCUUGCUCUACCUACCUGGG	2774
UUCUUGCUCUACCUACCUGG	2775

CUUCUUGCUCUACCUACCUG	2776
CACCAUCUUCUUGCUCUACC	2777
ACACCAUCUUCUUGCUCUAC	2778
AACACCAUCUUCUUGCUCUA	2779
AAACACCAUCUUCUUGCUCU	2780
GUUAAGAGAGCCAACAGUGA	2781
AGUUAAGAGAGCCAACAGUG	2782
ACACAGUUAAGAGAGCCAAC	2783
UGGACACAGUUAAGAGAGCC	2784
AGUGGACACAGUUAAGAGAG	2785
UGAAGGAGUGGACACAGUUA	2786
AUGAAGGAGUGGACACAGUU	2787
ACCAUGAAGGAGUGGACACA	2788
ACACCAUGAAGGAGUGGACA	2789
GACACCAUGAAGGAGUGGAC	2790
UGACACCAUGAAGGAGUGGA	2791
CUGACACCAUGAAGGAGUGG	2792
CUCUGACACCAUGAAGGAGU	2793
GCUCUGACACCAUGAAGGAG	2794
UGCUCUGACACCAUGAAGGA	2795
GUGCUCUGACACCAUGAAGG	2796
GGAGAUGCUUCAGUGCUCUG	2797
UGGAGAUGCUUCAGUGCUCU	2798
GUUUUGGAGAUGCUUCAGUG	2799
UACGUUUUGGAGAUGCUUCA	2800
CUACGUUUUGGAGAUGCUUC	2801
ACUACGUUUUGGAGAUGCUU	2802
CACUACGUUUUGGAGAUGCU	2803
AUCACUACGUUUUGGAGAUG	2804

CAUCACUACGUUUUGGAGAU	2805
CCAUCACUACGUUUUGGAGA	2806
CCCAUCACUACGUUUUGGAG	2807
UCCCAUCACUACGUUUUGGA	2808
GUCCCAUCACUACGUUUUGG	2809
UGUCCCAUCACUACGUUUUG	2810
GUGUCCCAUCACUACGUUUU	2811
GGUGUCCCAUCACUACGUUU	2812
UGGUGUCCCAUCACUACGUU	2813
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UAUUCCAAGGAAAUGGUGUC	2815
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GAGAUCAUAAUGAACUGGGA	2817
AGAGAUCAUAAUGAACUGGG	2818
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GGUCAGCGUGGUAAGGUUUG	2820
AGGUCAGCGUGGUAAGGUUU	2821
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GAAGGUCAGCGUGGUAAGGU	2823
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GUGAUUUCUACUUUCGUGGU	2830
ACUGGCUGUGAUUUCUACUU	2831
GACUGGCUGUGAUUUCUACU	2832
CUGACUGGCUGUGAUUUCUA	2833

UGCAGGAUGAUGGUGCUGGU	2834
AUGCAGGAUGAUGGUGCUGG	2835
CUAUGCAGGAUGAUGGUGCU	2836
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GUGACUAUGCAGGAUGAUGG	2838
GGUGACUAUGCAGGAUGAUG	2839
UGGUGACUAUGCAGGAUGAU	2840
GUGGUGACUAUGCAGGAUGA	2841
GGUGGUGACUAUGCAGGAUG	2842
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UCACCUGUGCAAUACCAAAU	3366
AUCACCUGUGCAAUACCAAA	3367
AAUCACCUGUGCAAUACCAA	3368
ACUAAUCACCUGUGCAAUAC	3369
GACUAAUCACCUGUGCAAUA	3370
UGACUAAUCACCUGUGCAAU	3371
UUGACUAAUCACCUGUGCAA	3372
CCUUGACUAAUCACCUGUGC	3373
UCCUUGACUAAUCACCUGUG	3374
UUCCUUGACUAAUCACCUGU	3375
CUUCCUUGACUAAUCACCUG	3376
ACUUCCUUGACUAAUCACCU	3377
GACUUCCUUGACUAAUCACC	3378
AGACUUCCUUGACUAAUCAC	3379
CAGACUUCCUUGACUAAUCA	3380
AAGGCUGUGGGACCAAAGCU	3381
GCAAGGCUGUGGGACCAAAG	3382
GGCAAGGCUGUGGGACCAAA	3383
UGCUGUGAGGCAAGGCUGUG	3384

CAUGCUGUGAGGCAAGGCUG	3385
ACAUGCUGUGAGGCAAGGCU	3386
UUUACAUGCUGUGAGGCAAG	3387
AUUUACAUGCUGUGAGGCAA	3388
GAAGAACCUCAGCAUCAAUA	3389
AGAAGAACCUCAGCAUCAAU	3390
UAGAAGAACCUCAGCAUCAA	3391
CAGUAGAAGAACCUCAGCAU	3392
CUAGCAGUAGAAGAACCUCA	3393
ACUAGCAGUAGAAGAACCUC	3394
GUCACUUUCAUACUAGCAGU	3395
CCCAAUUCACACCAGUAAAA	3396
UCCCAAUUCACACCAGUAAA	3397
CUUCCCAAUUCACACCAGUA	3398
CGUCAUGGAAUAGCAUUGUU	3399
ACGUCAUGGAAUAGCAUUGU	3400
CAAACGUCAUGGAAUAGCAU	3401
ACAAACGUCAUGGAAUAGCA	3402
UACAAACGUCAUGGAAUAGC	3403
CACAUAGUCAAGGACCUUAA	3404
ACACAUAGUCAAGGACCUUA	3405
UACACAUAGUCAAGGACCUU	3406
GUACACAUAGUCAAGGACCU	3407
CGAGAUGGCAGAGAUUAACU	3408
GCGAGAUGGCAGAGAUUAAC	3409
AUUUAGCGAGAUGGCAGAGA	3410
GAUUUAGCGAGAUGGCAGAG	3411
UGAUUUAGCGAGAUGGCAGA	3412
AUGAUUUAGCGAGAUGGCAG	3413

GAUGAUUUAGCGAGAUGGCA	3414
UGAUGAUUUAGCGAGAUGGC	3415
CUGAUGAUUUAGCGAGAUGG	3416
ACUGAUGAUUUAGCGAGAUG	3417
GACUGAUGAUUUAGCGAGAU	3418
AGACUGAUGAUUUAGCGAGA	3419
GAUCAGGCACCUUCUAUUUC	3420
AGAUCAGGCACCUUCUAUUU	3421
UUAGGAAGAUCAGGCACCUU	3422
AUUAGGAAGAUCAGGCACCU	3423
UAUUAGGAAGAUCAGGCACC	3424
GCAGCACAUCUUAUUUGGGA	3425
AGCAGCACAUCUUAUUUGGG	3426
AAGCAGCACAUCUUAUUUGG	3427
CUCGGUAAGCAGCACAUCUU	3428
CCUCGGUAAGCAGCACAUCU	3429
ACCUCGGUAAGCAGCACAUC	3430
UACCUCGGUAAGCAGCACAU	3431
AUACCUCGGUAAGCAGCACA	3432
GAUACCUCGGUAAGCAGCAC	3433
UGAUACCUCGGUAAGCAGCA	3434
GUGAUACCUCGGUAAGCAGC	3435
CGUGAUACCUCGGUAAGCAG	3436
CCGUGAUACCUCGGUAAGCA	3437
CCCGUGAUACCUCGGUAAGC	3438
UCAACGACCCAAGCUGGAGC	3439
UUCAACGACCCAAGCUGGAG	3440
CUUCAACGACCCAAGCUGGA	3441
CCCAGCUUCAACGACCCAAG	3442

UCUCUGAAGUGGUUUCCCAA	3443
AUCUCUGAAGUGGUUUCCCA	3444
CAUCUCUGAAGUGGUUUCCC	3445
GAUGGCAUUGUGCUCAUAAU	3446
UGAUGGCAUUGUGCUCAUAA	3447
GUGAUGGCAUUGUGCUCAUA	3448
GUUAAGUGAUGGCAUUGUGC	3449
AGUUAAGUGAUGGCAUUGUG	3450
GAGUCUAUUUGGUCAAGAGA	3451
UGAGUCUAUUUGGUCAAGAG	3452
CCAUAACUCACUCACUAGAA	3453
AGCACCAUAACUCACUCACU	3454
CCAGCACCAUAACUCACUCA	3455
GCCAGCACCAUAACUCACUC	3456
AGCCAGCACCAUAACUCACU	3457
UAGCCAGCACCAUAACUCAC	3458
AUAGCCAGCACCAUAACUCA	3459
UAUAGCCAGCACCAUAACUC	3460
AAUAUAGCCAGCACCAUAAC	3461
CAAAAUAUAGCCAGCACCAU	3462
GCAAAAUAUAGCCAGCACCA	3463
GCAACAUUUGAGAGGGCAGU	3464
AGCAACAUUUGAGAGGGCAG	3465
UAGCAACAUUUGAGAGGGCA	3466
CUAGCAACAUUUGAGAGGGC	3467
CCUAGCAACAUUUGAGAGGG	3468
UCCUAGCAACAUUUGAGAGG	3469
UUCCUAGCAACAUUUGAGAG	3470
AAAUUCUGGCAAGCAAGGCC	3471

UCAAAUUCUGGCAAGCAAGG	3472
AGUCAAAUUCUGGCAAGCAA	3473
AAGUCAAAUUCUGGCAAGCA	3474
AAAGUCAAAUUCUGGCAAGC	3475
CUGAAACCUGACACCGUAUA	3476
CCUGAAACCUGACACCGUAU	3477
CCCUGAAACCUGACACCGUA	3478
ACCCUGAAACCUGACACCGU	3479
CACCAAAGAGUUCUGGACUU	3480
UCACCAAAGAGUUCUGGACU	3481
GUCACCAAAGAGUUCUGGAC	3482
AGUCACCAAAGAGUUCUGGA	3483
AAAACGGCUUUACAGGGUAU	3484
GACCAUAGGUUGUUUCAUAC	3485
CAGACCAUAGGUUGUUUCAU	3486
CCAGACCAUAGGUUGUUUCA	3487
UCCAGACCAUAGGUUGUUUC	3488
UUCCAGACCAUAGGUUGUUU	3489
UUUCCAGACCAUAGGUUGUU	3490
AUUUCCAGACCAUAGGUUGU	3491
AAUUUCCAGACCAUAGGUUG	3492
CACAUAUAGCUUUGCGAUAC	3493
GCACAUAUAGCUUUGCGAUA	3494
UGCACAUAUAGCUUUGCGAU	3495
AUGCACAUAUAGCUUUGCGA	3496
UAUGCACAUAUAGCUUUGCG	3497
GCAAACUCAAAGCAUAGAUC	3498

In some embodiments, the siRNA molecules targeted to ERAP1 comprise or consist of the nucleotide sequences (sense and antisense strands) shown in Table 6.

Table 6

Sense Sequence	**SEQ ID NO:**	**Antisense Sequence**	**SEQ ID NO:**
AAAGGCGUGCACUUCCUAC	3499	GUAGGAAGUGCACGCCUUU	3500
AAGGCGUGCACUUCCUACG	3501	CGUAGGAAGUGCACGCCUU	3502
CGUGCACUUCCUACGCCUG	3503	CAGGCGUAGGAAGUGCACG	3504
GUGCACUUCCUACGCCUGA	3505	UCAGGCGUAGGAAGUGCAC	3506
UGCACUUCCUACGCCUGAU	3507	AUCAGGCGUAGGAAGUGCA	3508
UCGCAACCUCGCAGCUUCC	3509	GGAAGCUGCGAGGUUGCGA	3510
CGGCGUGCAGCGCUCAUUU	3511	AAAUGAGCGCUGCACGCCG	3512
GGCGUGCAGCGCUCAUUUA	3513	UAAAUGAGCGCUGCACGCC	3514
GCGUGCAGCGCUCAUUUAC	3515	GUAAAUGAGCGCUGCACGC	3516
CGUGCAGCGCUCAUUUACC	3517	GGUAAAUGAGCGCUGCACG	3518
GUGCAGCGCUCAUUUACCA	3519	UGGUAAAUGAGCGCUGCAC	3520
UGCAGCGCUCAUUUACCAA	3521	UUGGUAAAUGAGCGCUGCA	3522
GCAGCGCUCAUUUACCAAU	3523	AUUGGUAAAUGAGCGCUGC	3524
CAGCGCUCAUUUACCAAUU	3525	AAUUGGUAAAUGAGCGCUG	3526
GCUCAUUUACCAAUUCCCU	3527	AGGGAAUUGGUAAAUGAGC	3528
CUCAUUUACCAAUUCCCUU	3529	AAGGGAAUUGGUAAAUGAG	3530
UCAUUUACCAAUUCCCUUC	3531	GAAGGGAAUUGGUAAAUGA	3532
CAUUUACCAAUUCCCUUCC	3533	GGAAGGGAAUUGGUAAAUG	3534
AUUUACCAAUUCCCUUCCU	3535	AGGAAGGGAAUUGGUAAAU	3536
CUUCCUGGGAGUUGCGGCU	3537	AGCCGCAACUCCCAGGAAG	3538
UUCCUGGGAGUUGCGGCUU	3539	AAGCCGCAACUCCCAGGAA	3540
UCCUGGGAGUUGCGGCUUC	3541	GAAGCCGCAACUCCCAGGA	3542
CCCACUCCCGUUUACCCUU	3543	AAGGGUAAACGGGAGUGGG	3544
CCACUCCCGUUUACCCUUU	3545	AAAGGGUAAACGGGAGUGG	3546
GCUCCACUUUCACUUUCGG	3547	CCGAAAGUGAAAGUGGAGC	3548
CUCCACUUUCACUUUCGGU	3549	ACCGAAAGUGAAAGUGGAG	3550
ACUUUCACUUUCGGUCCUG	3551	CAGGACCGAAAGUGAAAGU	3552

CCCAGGUAGGUAGAGCAAG	3553	CUUGCUCUACCUACCUGGG	3554
CCAGGUAGGUAGAGCAAGA	3555	UCUUGCUCUACCUACCUGG	3556
CAGGUAGGUAGAGCAAGAA	3557	UUCUUGCUCUACCUACCUG	3558
AGGUAGGUAGAGCAAGAAG	3559	CUUCUUGCUCUACCUACCU	3560
GGUAGAGCAAGAAGAUGGU	3561	ACCAUCUUCUUGCUCUACC	3562
GUAGAGCAAGAAGAUGGUG	3563	CACCAUCUUCUUGCUCUAC	3564
UAGAGCAAGAAGAUGGUGU	3565	ACACCAUCUUCUUGCUCUA	3566
AGAGCAAGAAGAUGGUGUU	3567	AACACCAUCUUCUUGCUCU	3568
GAGCAAGAAGAUGGUGUUU	3569	AAACACCAUCUUCUUGCUC	3570
GAAGAUGGUGUUUCUGCCC	3571	GGGCAGAAACACCAUCUUC	3572
UCACUGUUGGCUCUCUUAA	3573	UUAAGAGAGCCAACAGUGA	3574
CACUGUUGGCUCUCUUAAC	3575	GUUAAGAGAGCCAACAGUG	3576
ACUGUUGGCUCUCUUAACU	3577	AGUUAAGAGAGCCAACAGU	3578
GGCUCUCUUAACUGUGUCC	3579	GGACACAGUUAAGAGAGCC	3580
GCUCUCUUAACUGUGUCCA	3581	UGGACACAGUUAAGAGAGC	3582
UAACUGUGUCCACUCCUUC	3583	GAAGGAGUGGACACAGUUA	3584
AACUGUGUCCACUCCUUCA	3585	UGAAGGAGUGGACACAGUU	3586
ACUGUGUCCACUCCUUCAU	3587	AUGAAGGAGUGGACACAGU	3588
UGUGUCCACUCCUUCAUGG	3589	CCAUGAAGGAGUGGACACA	3590
GUGUCCACUCCUUCAUGGU	3591	ACCAUGAAGGAGUGGACAC	3592
UGUCCACUCCUUCAUGGUG	3593	CACCAUGAAGGAGUGGACA	3594
GUCCACUCCUUCAUGGUGU	3595	ACACCAUGAAGGAGUGGAC	3596
UCCACUCCUUCAUGGUGUC	3597	GACACCAUGAAGGAGUGGA	3598
CCACUCCUUCAUGGUGUCA	3599	UGACACCAUGAAGGAGUGG	3600
CACUCCUUCAUGGUGUCAG	3601	CUGACACCAUGAAGGAGUG	3602
ACUCCUUCAUGGUGUCAGA	3603	UCUGACACCAUGAAGGAGU	3604
AGCACUGAAGCAUCUCCAA	3605	UUGGAGAUGCUUCAGUGCU	3606
GCACUGAAGCAUCUCCAAA	3607	UUUGGAGAUGCUUCAGUGC	3608
CACUGAAGCAUCUCCAAAA	3609	UUUUGGAGAUGCUUCAGUG	3610
AACGUAGUGAUGGGACACC	3611	GGUGUCCCAUCACUACGUU	3612
ACGUAGUGAUGGGACACCA	3613	UGGUGUCCCAUCACUACGU	3614

GGACACCAUUUCCUUGGAA	3615	UUCCAAGGAAAUGGUGUCC	3616
GACACCAUUUCCUUGGAAU	3617	AUUCCAAGGAAAUGGUGUC	3618
ACACCAUUUCCUUGGAAUA	3619	UAUUCCAAGGAAAUGGUGU	3620
CACCAUUUCCUUGGAAUAA	3621	UUAUUCCAAGGAAAUGGUG	3622
CCAUUUCCUUGGAAUAAAA	3623	UUUUAUUCCAAGGAAAUGG	3624
UUCCUUGGAAUAAAAUACG	3625	CGUAUUUUAUUCCAAGGAA	3626
UCCUUGGAAUAAAAUACGA	3627	UCGUAUUUUAUUCCAAGGA	3628
CCUUGGAAUAAAAUACGAC	3629	GUCGUAUUUUAUUCCAAGG	3630
UGGAAUAAAAUACGACUUC	3631	GAAGUCGUAUUUUAUUCCA	3632
GAAUAAAAUACGACUUCCU	3633	AGGAAGUCGUAUUUUAUUC	3634
GUCAUCCCAGUUCAUUAUG	3635	CAUAAUGAACUGGGAUGAC	3636
CCCAGUUCAUUAUGAUCUC	3637	GAGAUCAUAAUGAACUGGG	3638
CCAGUUCAUUAUGAUCUCU	3639	AGAGAUCAUAAUGAACUGG	3640
CAGUUCAUUAUGAUCUCUU	3641	AAGAGAUCAUAAUGAACUG	3642
UACCACGCUGACCUUCUGG	3643	CCAGAAGGUCAGCGUGGUA	3644
ACCACGCUGACCUUCUGGG	3645	CCCAGAAGGUCAGCGUGGU	3646
AAUCACAGCCAGUCAGCCC	3647	GGGCUGACUGGCUGUGAUU	3648
AUCACAGCCAGUCAGCCCA	3649	UGGGCUGACUGGCUGUGAU	3650
CACCAGCACCAUCAUCCUG	3651	CAGGAUGAUGGUGCUGGUG	3652
ACCAGCACCAUCAUCCUGC	3653	GCAGGAUGAUGGUGCUGGU	3654
CCAGCACCAUCAUCCUGCA	3655	UGCAGGAUGAUGGUGCUGG	3656
CAGCACCAUCAUCCUGCAU	3657	AUGCAGGAUGAUGGUGCUG	3658
AGCACCAUCAUCCUGCAUA	3659	UAUGCAGGAUGAUGGUGCU	3660
GCACCAUCAUCCUGCAUAG	3661	CUAUGCAGGAUGAUGGUGC	3662
CACCAUCAUCCUGCAUAGU	3663	ACUAUGCAGGAUGAUGGUG	3664
ACCAUCAUCCUGCAUAGUC	3665	GACUAUGCAGGAUGAUGGU	3666
CCAUCAUCCUGCAUAGUCA	3667	UGACUAUGCAGGAUGAUGG	3668
CAUCCUGCAUAGUCACCAC	3669	GUGGUGACUAUGCAGGAUG	3670
UAGUCACCACCUGCAGAUA	3671	UAUCUGCAGGUGGUGACUA	3672
AGUCACCACCUGCAGAUAU	3673	AUAUCUGCAGGUGGUGACU	3674
ACCACCUGCAGAUAUCUAG	3675	CUAGAUAUCUGCAGGUGGU	3676

CCACCUGCAGAUAUCUAGG	3677	CCUAGAUAUCUGCAGGUGG	3678
CACCUGCAGAUAUCUAGGG	3679	CCCUAGAUAUCUGCAGGUG	3680
ACCUGCAGAUAUCUAGGGC	3681	GCCCUAGAUAUCUGCAGGU	3682
CCUGCAGAUAUCUAGGGCC	3683	GGCCCUAGAUAUCUGCAGG	3684
CAAAUUGCACUGCUGGCUC	3685	GAGCCAGCAGUGCAAUUUG	3686
UCCUUGUCGGGCUCCCGUA	3687	UACGGGAGCCCGACAAGGA	3688
CUUGUCGGGCUCCCGUACA	3689	UGUACGGGAGCCCGACAAG	3690
CACAGUUGUCAUUCACUAU	3691	AUAGUGAAUGACAACUGUG	3692
ACAGUUGUCAUUCACUAUG	3693	CAUAGUGAAUGACAACUGU	3694
CAGUUGUCAUUCACUAUGC	3695	GCAUAGUGAAUGACAACUG	3696
AGUUGUCAUUCACUAUGCU	3697	AGCAUAGUGAAUGACAACU	3698
GUUGUCAUUCACUAUGCUG	3699	CAGCAUAGUGAAUGACAAC	3700
CAUUCACUAUGCUGGCAAU	3701	AUUGCCAGCAUAGUGAAUG	3702
AUUCACUAUGCUGGCAAUC	3703	GAUUGCCAGCAUAGUGAAU	3704
UUCACUAUGCUGGCAAUCU	3705	AGAUUGCCAGCAUAGUGAA	3706
UCACUAUGCUGGCAAUCUU	3707	AAGAUUGCCAGCAUAGUGA	3708
CACUAUGCUGGCAAUCUUU	3709	AAAGAUUGCCAGCAUAGUG	3710
ACUAUGCUGGCAAUCUUUC	3711	GAAAGAUUGCCAGCAUAGU	3712
AGACUUUCCACGGAUUUUA	3713	UAAAAUCCGUGGAAAGUCU	3714
GACUUUCCACGGAUUUUAC	3715	GUAAAAUCCGUGGAAAGUC	3716
ACUUUCCACGGAUUUUACA	3717	UGUAAAAUCCGUGGAAAGU	3718
AACUGAGGAUACUAGCAUC	3719	GAUGCUAGUAUCCUCAGUU	3720
ACUGAGGAUACUAGCAUCA	3721	UGAUGCUAGUAUCCUCAGU	3722
CUGAGGAUACUAGCAUCAA	3723	UUGAUGCUAGUAUCCUCAG	3724
UGAGGAUACUAGCAUCAAC	3725	GUUGAUGCUAGUAUCCUCA	3726
UGCAGCUAGAAUGGCCUUU	3727	AAAGGCCAUUCUAGCUGCA	3728
AAUGGCCUUUCCCUGCUUU	3729	AAAGCAGGGAAAGGCCAUU	3730
GCCUUUCCCUGCUUUGAUG	3731	CAUCAAAGCAGGGAAAGGC	3732
CUUUCCCUGCUUUGAUGAA	3733	UUCAUCAAAGCAGGGAAAG	3734
ACCUGCCUUCAAAGCAAGU	3735	ACUUGCUUUGAAGGCAGGU	3736
CUGCCUUCAAAGCAAGUUU	3737	AAACUUGCUUUGAAGGCAG	3738

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GCCUUCAAAGCAAGUUUCU	3741	AGAAACUUGCUUUGAAGGC	3742
CAAAGCAAGUUUCUCAAUC	3743	GAUUGAGAAACUUGCUUUG	3744
AAGCAAGUUUCUCAAUCAA	3745	UUGAUUGAGAAACUUGCUU	3746
AGCAAGUUUCUCAAUCAAA	3747	UUUGAUUGAGAAACUUGCU	3748
GCAAGUUUCUCAAUCAAAA	3749	UUUUGAUUGAGAAACUUGC	3750
GAGCCAAGGCACCUAGCCA	3751	UGGCUAGGUGCCUUGGCUC	3752
GGCACCUAGCCAUCUCCAA	3753	UUGGAGAUGGCUAGGUGCC	3754
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CACCUAGCCAUCUCCAAUA	3757	UAUUGGAGAUGGCUAGGUG	3758
ACCUAGCCAUCUCCAAUAU	3759	AUAUUGGAGAUGGCUAGGU	3760
CCUAGCCAUCUCCAAUAUG	3761	CAUAUUGGAGAUGGCUAGG	3762
CUAGCCAUCUCCAAUAUGC	3763	GCAUAUUGGAGAUGGCUAG	3764
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AAAUCUGUGACUGUUGCUG	3769	CAGCAACAGUCACAGAUUU	3770
AAUCUGUGACUGUUGCUGA	3771	UCAGCAACAGUCACAGAUU	3772
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UGACUGUUGCUGAAGGACU	3777	AGUCCUUCAGCAACAGUCA	3778
ACUGUUGCUGAAGGACUCA	3779	UGAGUCCUUCAGCAACAGU	3780
CUGUUGCUGAAGGACUCAU	3781	AUGAGUCCUUCAGCAACAG	3782
UGUUGCUGAAGGACUCAUA	3783	UAUGAGUCCUUCAGCAACA	3784
GUUGCUGAAGGACUCAUAG	3785	CUAUGAGUCCUUCAGCAAC	3786
UUGCUGAAGGACUCAUAGA	3787	UCUAUGAGUCCUUCAGCAA	3788
UGCUGAAGGACUCAUAGAA	3789	UUCUAUGAGUCCUUCAGCA	3790
GCUGAAGGACUCAUAGAAG	3791	CUUCUAUGAGUCCUUCAGC	3792
CUGAAGGACUCAUAGAAGA	3793	UCUUCUAUGAGUCCUUCAG	3794
UGAAGGACUCAUAGAAGAC	3795	GUCUUCUAUGAGUCCUUCA	3796
AAGGACUCAUAGAAGACCA	3797	UGGUCUUCUAUGAGUCCUU	3798
AGGACUCAUAGAAGACCAU	3799	AUGGUCUUCUAUGAGUCCU	3800

GGACUCAUAGAAGACCAUU	3801	AAUGGUCUUCUAUGAGUCC	3802
GACUCAUAGAAGACCAUUU	3803	AAAUGGUCUUCUAUGAGUC	3804
ACUCAUAGAAGACCAUUUU	3805	AAAAUGGUCUUCUAUGAGU	3806
CUCAUAGAAGACCAUUUUG	3807	CAAAAUGGUCUUCUAUGAG	3808
UCAUAGAAGACCAUUUUGA	3809	UCAAAAUGGUCUUCUAUGA	3810
CAUAGAAGACCAUUUUGAU	3811	AUCAAAAUGGUCUUCUAUG	3812
AUAGAAGACCAUUUUGAUG	3813	CAUCAAAAUGGUCUUCUAU	3814
UAGAAGACCAUUUUGAUGU	3815	ACAUCAAAAUGGUCUUCUA	3816
AGAAGACCAUUUUGAUGUC	3817	GACAUCAAAAUGGUCUUCU	3818
UGAUGUCACUGUGAAGAUG	3819	CAUCUUCACAGUGACAUCA	3820
GAUGUCACUGUGAAGAUGA	3821	UCAUCUUCACAGUGACAUC	3822
AUGUCACUGUGAAGAUGAG	3823	CUCAUCUUCACAGUGACAU	3824
UGUGAAGAUGAGCACCUAU	3825	AUAGGUGCUCAUCUUCACA	3826
GUGAAGAUGAGCACCUAUC	3827	GAUAGGUGCUCAUCUUCAC	3828
ACCUAUCUGGUGGCCUUCA	3829	UGAAGGCCACCAGAUAGGU	3830
CCUAUCUGGUGGCCUUCAU	3831	AUGAAGGCCACCAGAUAGG	3832
CUAUCUGGUGGCCUUCAUC	3833	GAUGAAGGCCACCAGAUAG	3834
UAUCUGGUGGCCUUCAUCA	3835	UGAUGAAGGCCACCAGAUA	3836
AUCUGGUGGCCUUCAUCAU	3837	AUGAUGAAGGCCACCAGAU	3838
UCUGGUGGCCUUCAUCAUU	3839	AAUGAUGAAGGCCACCAGA	3840
CUGGUGGCCUUCAUCAUUU	3841	AAAUGAUGAAGGCCACCAG	3842
UGGUGGCCUUCAUCAUUUC	3843	GAAAUGAUGAAGGCCACCA	3844
GGCCUUCAUCAUUUCAGAU	3845	AUCUGAAAUGAUGAAGGCC	3846
GCCUUCAUCAUUUCAGAUU	3847	AAUCUGAAAUGAUGAAGGC	3848
CCUUCAUCAUUUCAGAUUU	3849	AAAUCUGAAAUGAUGAAGG	3850
AGAUUUUGAGUCUGUCAGC	3851	GCUGACAGACUCAAAAUCU	3852
UUUGAGUCUGUCAGCAAGA	3853	UCUUGCUGACAGACUCAAA	3854
UUGAGUCUGUCAGCAAGAU	3855	AUCUUGCUGACAGACUCAA	3856
UGAGUCUGUCAGCAAGAUA	3857	UAUCUUGCUGACAGACUCA	3858
GAGUCUGUCAGCAAGAUAA	3859	UUAUCUUGCUGACAGACUC	3860
AGUCUGUCAGCAAGAUAAC	3861	GUUAUCUUGCUGACAGACU	3862

GUCUGUCAGCAAGAUAACC	3863	GGUUAUCUUGCUGACAGAC	3864
UCUGUCAGCAAGAUAACCA	3865	UGGUUAUCUUGCUGACAGA	3866
UGUCAGCAAGAUAACCAAG	3867	CUUGGUUAUCUUGCUGACA	3868
GUCAGCAAGAUAACCAAGA	3869	UCUUGGUUAUCUUGCUGAC	3870
UCAGCAAGAUAACCAAGAG	3871	CUCUUGGUUAUCUUGCUGA	3872
CAGCAAGAUAACCAAGAGU	3873	ACUCUUGGUUAUCUUGCUG	3874
AGCAAGAUAACCAAGAGUG	3875	CACUCUUGGUUAUCUUGCU	3876
GCAAGAUAACCAAGAGUGG	3877	CCACUCUUGGUUAUCUUGC	3878
CAAGAUAACCAAGAGUGGA	3879	UCCACUCUUGGUUAUCUUG	3880
AAGAUAACCAAGAGUGGAG	3881	CUCCACUCUUGGUUAUCUU	3882
UAACCAAGAGUGGAGUCAA	3883	UUGACUCCACUCUUGGUUA	3884
AACCAAGAGUGGAGUCAAG	3885	CUUGACUCCACUCUUGGUU	3886
ACCAAGAGUGGAGUCAAGG	3887	CCUUGACUCCACUCUUGGU	3888
CCAAGAGUGGAGUCAAGGU	3889	ACCUUGACUCCACUCUUGG	3890
CAAGAGUGGAGUCAAGGUU	3891	AACCUUGACUCCACUCUUG	3892
AAGAGUGGAGUCAAGGUUU	3893	AAACCUUGACUCCACUCUU	3894
AGAGUGGAGUCAAGGUUUC	3895	GAAACCUUGACUCCACUCU	3896
GAGUGGAGUCAAGGUUUCU	3897	AGAAACCUUGACUCCACUC	3898
AGUGGAGUCAAGGUUUCUG	3899	CAGAAACCUUGACUCCACU	3900
GUGGAGUCAAGGUUUCUGU	3901	ACAGAAACCUUGACUCCAC	3902
UGGAGUCAAGGUUUCUGUU	3903	AACAGAAACCUUGACUCCA	3904
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UUCUGCCUAUUUUCAUUUG	5013	CAAAUGAAAAUAGGCAGAA	5014
UCUGCCUAUUUUCAUUUGC	5015	GCAAAUGAAAAUAGGCAGA	5016
GCCUAUUUUCAUUUGCUUU	5017	AAAGCAAAUGAAAAUAGGC	5018
AUUUUCUUUCUAGUUGUGG	5019	CCACAACUAGAAAGAAAAU	5020
UUUUCUUUCUAGUUGUGGC	5021	GCCACAACUAGAAAGAAAA	5022
UUCUUUCUAGUUGUGGCUG	5023	CAGCCACAACUAGAAAGAA	5024
UCUCCCAAAUAAGAUGUGC	5025	GCACAUCUUAUUUGGGAGA	5026
CUCCCAAAUAAGAUGUGCU	5027	AGCACAUCUUAUUUGGGAG	5028
AAGAUGUGCUGCUUACCGA	5029	UCGGUAAGCAGCACAUCUU	5030
AGAUGUGCUGCUUACCGAG	5031	CUCGGUAAGCAGCACAUCU	5032
GAUGUGCUGCUUACCGAGG	5033	CCUCGGUAAGCAGCACAUC	5034
AUGUGCUGCUUACCGAGGU	5035	ACCUCGGUAAGCAGCACAU	5036
UGUGCUGCUUACCGAGGUA	5037	UACCUCGGUAAGCAGCACA	5038
CUGCUUACCGAGGUAUCAC	5039	GUGAUACCUCGGUAAGCAG	5040

UGCUUACCGAGGUAUCACG	5041	CGUGAUACCUCGGUAAGCA	5042
GCUUACCGAGGUAUCACGG	5043	CCGUGAUACCUCGGUAAGC	5044
CUUACCGAGGUAUCACGGG	5045	CCCGUGAUACCUCGGUAAG	5046
GGCUCCAGCUUGGGUCGUU	5047	AACGACCCAAGCUGGAGCC	5048
GCUCCAGCUUGGGUCGUUG	5049	CAACGACCCAAGCUGGAGC	5050
CUCCAGCUUGGGUCGUUGA	5051	UCAACGACCCAAGCUGGAG	5052
UCCAGCUUGGGUCGUUGAA	5053	UUCAACGACCCAAGCUGGA	5054
CCAGCUUGGGUCGUUGAAG	5055	CUUCAACGACCCAAGCUGG	5056
CUUGGGUCGUUGAAGCUGG	5057	CCAGCUUCAACGACCCAAG	5058
UUGGGUCGUUGAAGCUGGG	5059	CCCAGCUUCAACGACCCAA	5060
CAGCAAGUUUAGCAUCUUC	5061	GAAGAUGCUAAACUUGCUG	5062
CAAUGCCAUCACUUAACUA	5063	UAGUUAAGUGAUGGCAUUG	5064
AUGCCAUCACUUAACUAUA	5065	UAUAGUUAAGUGAUGGCAU	5066
UGCCAUCACUUAACUAUAA	5067	UUAUAGUUAAGUGAUGGCA	5068
GCCAUCACUUAACUAUAAC	5069	GUUAUAGUUAAGUGAUGGC	5070
CCAUCACUUAACUAUAACU	5071	AGUUAUAGUUAAGUGAUGG	5072
CAUCACUUAACUAUAACUC	5073	GAGUUAUAGUUAAGUGAUG	5074
UCUCUUGACCAAAUAGACU	5075	AGUCUAUUUGGUCAAGAGA	5076
CUCUUGACCAAAUAGACUC	5077	GAGUCUAUUUGGUCAAGAG	5078
UCUUGACCAAAUAGACUCA	5079	UGAGUCUAUUUGGUCAAGA	5080
CUUGACCAAAUAGACUCAU	5081	AUGAGUCUAUUUGGUCAAG	5082
UUGACCAAAUAGACUCAUA	5083	UAUGAGUCUAUUUGGUCAA	5084
GACCAAAUAGACUCAUAAU	5085	AUUAUGAGUCUAUUUGGUC	5086
UUUCCUGUUGUACAUUUAG	5087	CUAAAUGUACAACAGGAAA	5088
GGUUAUUCAAAGUUUUCAG	5089	CUGAAAACUUUGAAUAACC	5090
UAUUCAAAGUUUUCAGCUC	5091	GAGCUGAAAACUUUGAAUA	5092
AUUCAAAGUUUUCAGCUCU	5093	AGAGCUGAAAACUUUGAAU	5094
UCAGCUCUUUUAAAAUUGC	5095	GCAAUUUUAAAAGAGCUGA	5096
CAGCUCUUUUAAAAUUGCU	5097	AGCAAUUUUAAAAGAGCUG	5098
AGCUCUUUUAAAAUUGCUC	5099	GAGCAAUUUUAAAAGAGCU	5100
GCUCUUUUAAAAUUGCUCU	5101	AGAGCAAUUUUAAAAGAGC	5102

AAUUGCUCUGAAUAAGUUC	5103	GAACUUAUUCAGAGCAAUU	5104
AUUGCUCUGAAUAAGUUCU	5105	AGAACUUAUUCAGAGCAAU	5106
UUGCUCUGAAUAAGUUCUA	5107	UAGAACUUAUUCAGAGCAA	5108
UGCUCUGAAUAAGUUCUAG	5109	CUAGAACUUAUUCAGAGCA	5110
CUGAAUAAGUUCUAGUGAG	5111	CUCACUAGAACUUAUUCAG	5112
AUAAGUUCUAGUGAGUGAG	5113	CUCACUCACUAGAACUUAU	5114
UUCUAGUGAGUGAGUUAUG	5115	CAUAACUCACUCACUAGAA	5116
UCUAGUGAGUGAGUUAUGG	5117	CCAUAACUCACUCACUAGA	5118
UGAGUUAUGGUGCUGGCUA	5119	UAGCCAGCACCAUAACUCA	5120
GAGUUAUGGUGCUGGCUAU	5121	AUAGCCAGCACCAUAACUC	5122
AGUUAUGGUGCUGGCUAUA	5123	UAUAGCCAGCACCAUAACU	5124
GUUAUGGUGCUGGCUAUAU	5125	AUAUAGCCAGCACCAUAAC	5126
UUAUGGUGCUGGCUAUAUU	5127	AAUAUAGCCAGCACCAUAA	5128
UAUGGUGCUGGCUAUAUUU	5129	AAAUAUAGCCAGCACCAUA	5130
AUGGUGCUGGCUAUAUUUU	5131	AAAAUAUAGCCAGCACCAU	5132
UGGUGCUGGCUAUAUUUUG	5133	CAAAAUAUAGCCAGCACCA	5134
GGUGCUGGCUAUAUUUUGC	5135	GCAAAAUAUAGCCAGCACC	5136
UGCCCUCUCAAAUGUUGCU	5137	AGCAACAUUUGAGAGGGCA	5138
GCCCUCUCAAAUGUUGCUA	5139	UAGCAACAUUUGAGAGGGC	5140
CCCUCUCAAAUGUUGCUAG	5141	CUAGCAACAUUUGAGAGGG	5142
CCUCUCAAAUGUUGCUAGG	5143	CCUAGCAACAUUUGAGAGG	5144
CUCUCAAAUGUUGCUAGGA	5145	UCCUAGCAACAUUUGAGAG	5146
AAAUGUUGCUAGGAAUUCA	5147	UGAAUUCCUAGCAACAUUU	5148
AAUGUUGCUAGGAAUUCAU	5149	AUGAAUUCCUAGCAACAUU	5150
AUGUUGCUAGGAAUUCAUA	5151	UAUGAAUUCCUAGCAACAU	5152
UGUUGCUAGGAAUUCAUAC	5153	GUAUGAAUUCCUAGCAACA	5154
GUUGCUAGGAAUUCAUACU	5155	AGUAUGAAUUCCUAGCAAC	5156
UUGCUAGGAAUUCAUACUG	5157	CAGUAUGAAUUCCUAGCAA	5158
UGCUAGGAAUUCAUACUGC	5159	GCAGUAUGAAUUCCUAGCA	5160
AAAGCAAUGAAUAAGCAUG	5161	CAUGCUUAUUCAUUGCUUU	5162
AAGCAAUGAAUAAGCAUGC	5163	GCAUGCUUAUUCAUUGCUU	5164

AGCAAUGAAUAAGCAUGCC	5165	GGCAUGCUUAUUCAUUGCU	5166
GCAAUGAAUAAGCAUGCCU	5167	AGGCAUGCUUAUUCAUUGC	5168
AAUGAAUAAGCAUGCCUGU	5169	ACAGGCAUGCUUAUUCAUU	5170
AUGAAUAAGCAUGCCUGUU	5171	AACAGGCAUGCUUAUUCAU	5172
UGAAUAAGCAUGCCUGUUU	5173	AAACAGGCAUGCUUAUUCA	5174
GAAUAAGCAUGCCUGUUUU	5175	AAAACAGGCAUGCUUAUUC	5176
AUAAGCAUGCCUGUUUUCC	5177	GGAAAACAGGCAUGCUUAU	5178
AUGCCUGUUUUCCCAUGGC	5179	GCCAUGGGAAAACAGGCAU	5180
UGCCUGUUUUCCCAUGGCC	5181	GGCCAUGGGAAAACAGGCA	5182
GCCUGUUUUCCCAUGGCCU	5183	AGGCCAUGGGAAAACAGGC	5184
CCUGUUUUCCCAUGGCCUU	5185	AAGGCCAUGGGAAAACAGG	5186
CUGUUUUCCCAUGGCCUUG	5187	CAAGGCCAUGGGAAAACAG	5188
UUCCCAUGGCCUUGCUUGC	5189	GCAAGCAAGGCCAUGGGAA	5190
GGCCUUGCUUGCCAGAAUU	5191	AAUUCUGGCAAGCAAGGCC	5192
UUGCUUGCCAGAAUUUGAC	5193	GUCAAAUUCUGGCAAGCAA	5194
UGCUUGCCAGAAUUUGACU	5195	AGUCAAAUUCUGGCAAGCA	5196
GCUUGCCAGAAUUUGACUU	5197	AAGUCAAAUUCUGGCAAGC	5198
CUUGCCAGAAUUUGACUUU	5199	AAAGUCAAAUUCUGGCAAG	5200
UUGCCAGAAUUUGACUUUU	5201	AAAAGUCAAAUUCUGGCAA	5202
GAUAUACUACUAUUGCUUG	5203	CAAGCAAUAGUAGUAUAUC	5204
AUAUUGUGGUAUACGGUGU	5205	ACACCGUAUACCACAAUAU	5206
UAUUGUGGUAUACGGUGUC	5207	GACACCGUAUACCACAAUA	5208
AUUGUGGUAUACGGUGUCA	5209	UGACACCGUAUACCACAAU	5210
UUGUGGUAUACGGUGUCAG	5211	CUGACACCGUAUACCACAA	5212
UGUGGUAUACGGUGUCAGG	5213	CCUGACACCGUAUACCACA	5214
GUGGUAUACGGUGUCAGGU	5215	ACCUGACACCGUAUACCAC	5216
UAUACGGUGUCAGGUUUCA	5217	UGAAACCUGACACCGUAUA	5218
AUACGGUGUCAGGUUUCAG	5219	CUGAAACCUGACACCGUAU	5220
UACGGUGUCAGGUUUCAGG	5221	CCUGAAACCUGACACCGUA	5222
UGUCAGGUUUCAGGGUUUU	5223	AAAACCCUGAAACCUGACA	5224
UUCAUUUCAAAUACUCCCU	5225	AGGGAGUAUUUGAAAUGAA	5226

CAUUUCAAAUACUCCCUUU	5227	AAAGGGAGUAUUUGAAAUG	5228
UAGGUAUAACUGUUGAUGA	5229	UCAUCAACAGUUAUACCUA	5230
AGGUAUAACUGUUGAUGAA	5231	UUCAUCAACAGUUAUACCU	5232
UUAUUGUCCAUUUAUACCC	5233	GGGUAUAAAUGGACAAUAA	5234
UAUUGUCCAUUUAUACCCU	5235	AGGGUAUAAAUGGACAAUA	5236
AUUGUCCAUUUAUACCCUG	5237	CAGGGUAUAAAUGGACAAU	5238
UUGUCCAUUUAUACCCUGU	5239	ACAGGGUAUAAAUGGACAA	5240
UGUCCAUUUAUACCCUGUA	5241	UACAGGGUAUAAAUGGACA	5242
GUCCAUUUAUACCCUGUAA	5243	UUACAGGGUAUAAAUGGAC	5244
UAUACCCUGUAAAGCCGUU	5245	AACGGCUUUACAGGGUAUA	5246
AUACCCUGUAAAGCCGUUU	5247	AAACGGCUUUACAGGGUAU	5248
UACCCUGUAAAGCCGUUUU	5249	AAAACGGCUUUACAGGGUA	5250
AGCCGUUUUAGAAUGUAAU	5251	AUUACAUUCUAAAACGGCU	5252
GCCGUUUUAGAAUGUAAUA	5253	UAUUACAUUCUAAAACGGC	5254
AGGUAAUCCAAAAUGUACU	5255	AGUACAUUUUGGAUUACCU	5256
GGUAAUCCAAAAUGUACUA	5257	UAGUACAUUUUGGAUUACC	5258
ACAUUUGUAUUGUCUCUUC	5259	GAAGAGACAAUACAAAUGU	5260
CAUUUGUAUUGUCUCUUCU	5261	AGAAGAGACAAUACAAAUG	5262
UUGUAUCGCAAAGCUAUAU	5263	AUAUAGCUUUGCGAUACAA	5264
UGUAUCGCAAAGCUAUAUG	5265	CAUAUAGCUUUGCGAUACA	5266
GUAUCGCAAAGCUAUAUGU	5267	ACAUAUAGCUUUGCGAUAC	5268
UAUCGCAAAGCUAUAUGUG	5269	CACAUAUAGCUUUGCGAUA	5270
AUCGCAAAGCUAUAUGUGC	5271	GCACAUAUAGCUUUGCGAU	5272
UCGCAAAGCUAUAUGUGCA	5273	UGCACAUAUAGCUUUGCGA	5274
UGAUCUAUGCUUUGAGUUU	5275	AAACUCAAAGCAUAGAUCA	5276
GAUCUAUGCUUUGAGUUUG	5277	CAAACUCAAAGCAUAGAUC	5278
AUCUAUGCUUUGAGUUUGC	5279	GCAAACUCAAAGCAUAGAU	5280
UCUAUGCUUUGAGUUUGCU	5281	AGCAAACUCAAAGCAUAGA	5282
CUAUGCUUUGAGUUUGCUU	5283	AAGCAAACUCAAAGCAUAG	5284
UAUGCUUUGAGUUUGCUUU	5285	AAAGCAAACUCAAAGCAUA	5286
AUGCUUUGAGUUUGCUUUC	5287	GAAAGCAAACUCAAAGCAU	5288

AUCUUACCUAAAAGUACUG	5289	CAGUACUUUUAGGUAAGAU	5290
CUUACCUAAAAGUACUGAA	5291	UUCAGUACUUUUAGGUAAG	5292

In some embodiments, the antisense nucleic acid molecules targeted to HLA-A comprise or consist of the nucleotide sequences shown in Table 7.

Table 7

Sequence	**SEQ ID NO:**
GUCACUGCUUGCAGCCUGAG	5293
UGUCACUGCUUGCAGCCUGA	5294
CUGUCACUGCUUGCAGCCUG	5295
ACUGUCACUGCUUGCAGCCU	5296
CACUGUCACUGCUUGCAGCC	5297
GCACUGUCACUGCUUGCAGC	5298
GGCACUGUCACUGCUUGCAG	5299
GGGCACUGUCACUGCUUGCA	5300
UGGGCACUGUCACUGCUUGC	5301
UCUCACACUUUACAAGCUGU	5302
GUCUCACACUUUACAAGCUG	5303
UGUCUCACACUUUACAAGCU	5304
CUGUCUCACACUUUACAAGC	5305
GCUGUCUCACACUUUACAAG	5306
AGCUGUCUCACACUUUACAA	5307

In some embodiments, the siRNA molecules targeted to HLA-A comprise or consist of the nucleotide sequences (sense and antisense strands) shown in Table 8.

Table 8

Sense Sequence	**SEQ ID NO:**	**Antisense Sequence**	**SEQ ID NO:**
GGCUGCAAGCAGUGACAGU	5308	ACUGUCACUGCUUGCAGCC	5309
GCUGCAAGCAGUGACAGUG	5310	CACUGUCACUGCUUGCAGC	5311
CUGCAAGCAGUGACAGUGC	5312	GCACUGUCACUGCUUGCAG	5313
UGCAAGCAGUGACAGUGCC	5314	GGCACUGUCACUGCUUGCA	5315

GCAAGCAGUGACAGUGCCC	5316	GGGCACUGUCACUGCUUGC	5317
CAAGCAGUGACAGUGCCCA	5318	UGGGCACUGUCACUGCUUG	5319
CUCACAGCUUGUAAAGUGU	5320	ACACUUUACAAGCUGUGAG	5321

In some embodiments, the antisense nucleic acid molecules targeted to HLA-B comprise or consist of the nucleotide sequences shown in Table 9.

Table 9

Sequence	**SEQ ID NO:**
UGCACGCAGCCUGAGAGUAG	5322
CUGCACGCAGCCUGAGAGUA	5323

In some embodiments, the siRNA molecules targeted to HLA-B comprise or consist of the nucleotide sequences (sense and antisense strands) shown in Table 10.

Table 10

Sense Sequence	**SEQ ID NO:**	**Antisense Sequence**	**SEQ ID NO:**
CUACUCUCAGGCUGCGUGC	5324	GCACGCAGCCUGAGAGUAG	5325
UACUCUCAGGCUGCGUGCA	5326	UGCACGCAGCCUGAGAGUA	5327

In some embodiments, the antisense nucleic acid molecules targeted to HLA-C comprise or consist of the nucleotide sequences shown in Table 11.

Table 11

Sequence	**SEQ ID NO:**
GCUGUCUCAGGCUUUACAAG	5328
AGCUGUCUCAGGCUUUACAA	5329

In some embodiments, the siRNA molecules targeted to HLA-C comprise or consist of the nucleotide sequences (sense and antisense strands) shown in Table 12.

Table 12

Sense Sequence	SEQ ID NO:	Antisense Sequence	SEQ ID NO:
UGCUCUCAGGCUGCGUGCA	5330	UGCACGCAGCCUGAGAGCA	5331

The inhibitory nucleic acid molecules disclosed herein can comprise RNA, DNA, or both RNA and DNA. The inhibitory nucleic acid molecules can also be linked or fused to a heterologous nucleic acid sequence, such as in a vector, or a heterologous label. For example, the inhibitory nucleic acid molecules disclosed herein can be within a vector or as an exogenous donor sequence comprising the inhibitory nucleic acid molecule and a heterologous nucleic acid sequence. The inhibitory nucleic acid molecules can also be linked or fused to a heterologous label. The label can be directly detectable (such as, for example, fluorophore) or indirectly detectable (such as, for example, hapten, enzyme, or fluorophore quencher). Such labels can be detectable by spectroscopic, photochemical, biochemical, immunochemical, or chemical means. Such labels include, for example, radiolabels, pigments, dyes, chromogens, spin labels, and fluorescent labels. The label can also be, for example, a chemiluminescent substance; a metal-containing substance; or an enzyme, where there occurs an enzyme-dependent secondary generation of signal. The term “label” can also refer to a “tag” or hapten that can bind selectively to a conjugated molecule such that the conjugated molecule, when added subsequently along with a substrate, is used to generate a detectable signal. For example, biotin can be used as a tag along with an avidin or streptavidin conjugate of horseradish peroxidate (HRP) to bind to the tag, and examined using a calorimetric substrate (such as, for example, tetramethylbenzidine (TMB)) or a fluorogenic substrate to detect the presence of HRP. Exemplary labels that can be used as tags to facilitate purification include, but are not limited to, myc, HA, FLAG or 3XFLAG, 6XHis or polyhistidine, glutathione-S-transferase (GST), maltose binding protein, an epitope tag, or the Fc portion of immunoglobulin. Numerous labels include, for example, particles, fluorophores, haptens, enzymes and their calorimetric, fluorogenic and chemiluminescent substrates and other labels.

The disclosed inhibitory nucleic acid molecules can comprise, for example, nucleotides or non-natural or modified nucleotides, such as nucleotide analogs or

nucleotide substitutes. Such nucleotides include a nucleotide that contains a modified base, sugar, or phosphate group, or that incorporates a non-natural moiety in its structure. Examples of non-natural nucleotides include, but are not limited to, dideoxynucleotides, biotinylated, aminated, deaminated, alkylated, benzylated, and fluorophor-labeled nucleotides.

The inhibitory nucleic acid molecules disclosed herein can also comprise one or more nucleotide analogs or substitutions. A nucleotide analog is a nucleotide which contains a modification to either the base, sugar, or phosphate moieties. Modifications to the base moiety include, but are not limited to, natural and synthetic modifications of A, C, G, and T/U, as well as different purine or pyrimidine bases such as, for example, pseudouridine, uracil-5-yl, hypoxanthin-9-yl (I), and 2-aminoadenin-9-yl. Modified bases include, but are not limited to, 5-methylcytosine (5-me-C), 5-hydroxymethyl cytosine, xanthine, hypoxanthine, 2-aminoadenine, 6-methyl and other alkyl derivatives of adenine and guanine, 2-propyl and other alkyl derivatives of adenine and guanine, 2-thiouracil, 2-thiothymine and 2-thiocytosine, 5-halouracil and cytosine, 5-propynyl uracil and cytosine, 6-azo uracil, cytosine and thymine, 5-uracil (pseudouracil), 4-thiouracil, 8-halo, 8-amino, 8-thiol, 8-thioalkyl, 8-hydroxyl and other 8-substituted adenines and guanines, 5-halo (such as, for example, 5-bromo), 5-trifluoromethyl and other 5-substituted uracils and cytosines, 7-methylguanine, 7-methyladenine, 8-azaguanine, 8-azaadenine, 7-deazaguanine, 7-deazaadenine, 3-deazaguanine, and 3-deazaadenine.

Nucleotide analogs can also include modifications of the sugar moiety. Modifications to the sugar moiety include, but are not limited to, natural modifications of the ribose and deoxy ribose as well as synthetic modifications. Sugar modifications include, but are not limited to, the following modifications at the 2' position: OH; F; O-, S-, or N-alkyl; O-, S-, or N-alkenyl; O-, S- or N-alkynyl; or O-alkyl-O-alkyl, wherein the alkyl, alkenyl, and alkynyl may be substituted or unsubstituted C_{1-10}alkyl or C_{2-10}alkenyl, and C_{2-10}alkynyl. Exemplary 2' sugar modifications also include, but are not limited to, $-O[(CH_2)_nO]_mCH_3$, $-O(CH_2)_nOCH_3$, $-O(CH_2)_nNH_2$, $-O(CH_2)_nCH_3$, $-O(CH_2)_n$-ONH_2, and $-O(CH_2)_nON[(CH_2)_nCH_3)]_2$, where n and m, independently, are from 1 to about 10. Other modifications at the 2' position include, but are not limited to, C_{1-10}alkyl, substituted lower alkyl, alkaryl, aralkyl,

O-alkaryl or O-aralkyl, SH, SCH_3, OCN, Cl, Br, CN, CF_3, OCF_3, $SOCH_3$, SO_2CH_3, ONO_2, NO_2, N_3, NH_2, heterocycloalkyl, heterocycloalkaryl, aminoalkylamino, polyalkylamino, substituted silyl, an RNA cleaving group, a reporter group, an intercalator, a group for improving the pharmacokinetic properties of an oligonucleotide, or a group for improving the pharmacodynamic properties of an oligonucleotide, and other substituents having similar properties. Similar modifications may also be made at other positions on the sugar, particularly the 3' position of the sugar on the 3' terminal nucleotide or in 2'-5' linked oligonucleotides and the 5' position of 5' terminal nucleotide. Modified sugars can also include those that contain modifications at the bridging ring oxygen, such as CH_2 and S. Nucleotide sugar analogs can also have sugar mimetics, such as cyclobutyl moieties in place of the pentofuranosyl sugar.

Nucleotide analogs can also be modified at the phosphate moiety. Modified phosphate moieties include, but are not limited to, those that can be modified so that the linkage between two nucleotides contains a phosphorothioate, chiral phosphorothioate, phosphorodithioate, phosphotriester, aminoalkylphosphotriester, methyl and other alkyl phosphonates including 3'-alkylene phosphonate and chiral phosphonates, phosphinates, phosphoramidates including 3'-amino phosphoramidate and aminoalkylphosphoramidates, thionophosphoramidates, thionoalkylphosphonates, thionoalkylphosphotriesters, and boranophosphates. These phosphate or modified phosphate linkage between two nucleotides can be through a 3'-5' linkage or a 2'-5' linkage, and the linkage can contain inverted polarity such as 3'-5' to 5'-3' or 2'-5' to 5'-2'. Various salts, mixed salts, and free acid forms are also included. Nucleotide substitutes also include peptide nucleic acids (PNAs).

In some embodiments, the antisense nucleic acid molecules are gapmers, whereby the first one to seven nucleotides at the 5' and 3' ends each have 2'-methoxyethyl (2'-MOE) modifications. In some embodiments, the first five nucleotides at the 5' and 3' ends each have 2'-MOE modifications. In some embodiments, the first one to seven nucleotides at the 5' and 3' ends are RNA nucleotides. In some embodiments, the first five nucleotides at the 5' and 3' ends are RNA nucleotides. In some embodiments, each of the backbone linkages between the nucleotides is a phosphorothioate linkage.

In some embodiments, the siRNA molecules have termini modifications. In some embodiments, the 5’ end of the antisense strand is phosphorylated. In some embodiments, 5’-phosphate analogs that cannot be hydrolyzed, such as 5’-(E)-vinyl-phosphonate are used.

In some embodiments, the siRNA molecules have backbone modifications. In some embodiments, the modified phosphodiester groups that link consecutive ribose nucleosides have been shown to enhance the stability and *in vivo* bioavailability of siRNAs The non-ester groups (-OH, =O) of the phosphodiester linkage can be replaced with sulfur, boron, or acetate to give phosphorothioate, boranophosphate, and phosphonoacetate linkages. In addition, substituting the phosphodiester group with a phosphotriester can facilitate cellular uptake of siRNAs and retention on serum components by eliminating their negative charge. In some embodiments, the siRNA molecules have sugar modifications. In some embodiments, the sugars are deprotonated (reaction catalyzed by exo- and endonucleases) whereby the 2’-hydroxyl can act as a nucleophile and attack the adjacent phosphorous in the phosphodiester bond. Such alternatives include 2’-O-methyl, 2’-O-methoxyethyl, and 2’-fluoro modifications.

In some embodiments, the siRNA molecules have base modifications. In some embodiments, the bases can be substituted with modified bases such as pseudouridine, 5’-methylcytidine, N6-methyladenosine, inosine, and N7-methylguanosine.

In some embodiments, the siRNA molecules are conjugated to lipids. Lipids can be conjugated to the 5’ or 3’ termini of siRNA to improve their *in vivo* bioavailability by allowing them to associate with serum lipoproteins. Representative lipids include, but are not limited to, cholesterol and vitamin E, and fatty acids, such as palmitate and tocopherol.

In some embodiments, a representative siRNA has the following formula:
Sense: mN*mN*/i2FN/mN/i2FN/mN/i2FN/mN/i2FN/mN/i2FN/mN/i2FN/mN/i2FN/mN/
i2FN/*mN*/32FN/
Antisense: /52FN/*/i2FN/*mN/i2FN/mN/i2FN/mN/i2FN/mN/i2FN/mN/i2FN/mN/i2FN/mN/
i2FN/mN/i2FN/mN*N*N

wherein: “N” is the base; “2F” is a 2’-F modification; “m” is a 2’-O-methyl modification, “I” is an internal base; and “*” is a phosphorothioate backbone linkage.

The present disclosure also provides vectors comprising any one or more of the inhibitory nucleic acid molecules disclosed herein. In some embodiments, the vectors comprise any one or more of the inhibitory nucleic acid molecules disclosed herein and a heterologous nucleic acid. The vectors can be viral or nonviral vectors capable of transporting a nucleic acid molecule. In some embodiments, the vector is a plasmid or cosmid (such as, for example, a circular double-stranded DNA into which additional DNA segments can be ligated). In some embodiments, the vector is a viral vector, wherein additional DNA segments can be ligated into the viral genome. Expression vectors include, but are not limited to, plasmids, cosmids, retroviruses, adenoviruses, adeno-associated viruses (AAV), plant viruses such as cauliflower mosaic virus and tobacco mosaic virus, yeast artificial chromosomes (YACs), Epstein-Barr (EBV)-derived episomes, and other expression vectors known in the art.

The present disclosure also provides compositions comprising any one or more of the inhibitory nucleic acid molecules disclosed herein. In some embodiments, the composition is a pharmaceutical composition. In some embodiments, the compositions comprise a carrier and/or excipient. Examples of carriers include, but are not limited to, poly(lactic acid) (PLA) microspheres, poly(D,L-lactic-coglycolic-acid) (PLGA) microspheres, liposomes, micelles, inverse micelles, lipid cochleates, and lipid microtubules. A carrier may comprise a buffered salt solution such as PBS, HBSS, etc.

The present disclosure also provides methods of identifying a subject having an increased risk for developing an MHC-I-opathy and/or an MHC-II-opathy. The methods comprise performing or having performed an assay on a biological sample from the subject to determine whether the subject comprises: i) an MHC-I-opathy-related HLA genotype and/or an MHC-II-opathy-related HLA genotype; and ii) a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein. When the subject has both the MHC-I-opathy-related HLA genotype and/or an MHC-II-opathy-related HLA genotype and the functional ERAP2 protein or the nucleic acid molecule encoding the functional ERAP2 protein, then the subject has an increased risk of developing the MHC-I-opathy and/or an MHC-II-opathy. When the subject lacks the MHC-I-opathy-related HLA genotype and/or an MHC-II-opathy-related HLA genotype, or lacks the functional ERAP2 protein or the nucleic

acid molecule encoding the functional ERAP2 protein, or lacks both, then the subject has a decreased risk of developing the MHC-I-opathy and/or an MHC-II-opathy. In some embodiments, the method further comprises determining whether the subject has a single copy of the MHC-I-opathy-related HLA genotype and/or an MHC-II-opathy-related HLA genotype or two copies of the MHC-I-opathy-related HLA genotype and/or an MHC-II-opathy-related HLA genotype. When the subject comprises two copies of the MHC-I-opathy-related HLA genotype and/or an MHC-II-opathy-related HLA genotype, then the subject has an increased risk of developing the MHC-I-opathy and/or an MHC-II-opathy compared to comprising a single copy of the MHC-I-opathy-related HLA genotype and/or an MHC-II-opathy-related HLA genotype.

In some embodiments, the MHC-I-opathy is BSCR. In some embodiments, the subject is HLA-Aw19$^+$. In some embodiments, the subject is or is suspected of being HLA-A*29$^+$, HLA-A*30$^+$, HLA-A*31$^+$, or HLA-A*33$^+$, or any combination thereof. In some embodiments, the subject has a single copy of HLA-Aw19. In some embodiments, the subject has two copies of HLA-Aw19. In some embodiments, the subject is HLA-A*29$^+$/HLA-A*30$^+$. In some embodiments, the subject is HLA-A*29$^+$/HLA-A*31$^+$. In some embodiments, the subject is HLA-A*29$^+$/HLA-A*33$^+$.

In some embodiments, the subject having BSCR is not HLA-A*29$^+$.

In some embodiments, the subject having BSCR has a copy of at least any two of HLA-A*29, HLA-A*30, HLA-A*31, or HLA-A*33. In some embodiments, the subject having BSCR has a copy of at least any three of HLA-A*29, HLA-A*30, HLA-A*31, or HLA-A*33. In some embodiments, the subject having BSCR has a copy of all of HLA-A*29, HLA-A*30, HLA-A*31, or HLA-A*33.

In some embodiments, the subject having BSCR has one copy of each HLA-A*29 and HLA-A*30. In some embodiments, the subject having BSCR has one copy of each HLA-A*29 and HLA-A*31. In some embodiments, the subject having BSCR has one copy of each HLA-A*29 and HLA-A*33. In some embodiments, the subject having BSCR has one copy of each HLA-A*30 and HLA-A*31. In some embodiments, the subject having BSCR has one copy of each HLA-A*30 and HLA-A*33. In some embodiments, the subject having BSCR has one copy of each HLA-A*31 and HLA-A*33.

In some embodiments, the subject having BSCR has one copy of HLA-A*29 and two copies of HLA-A*30. In some embodiments, the subject having BSCR has one copy of HLA-A*29 and two copies of HLA-A*31. In some embodiments, the subject having BSCR has one copy of HLA-A*29 and two copies of HLA-A*33. In some embodiments, the subject having BSCR has one copy of HLA-A*30 and two copies of HLA-A*31. In some embodiments, the subject having BSCR has one copy of HLA-A*30 and two copies HLA-A*33. In some embodiments, the subject having BSCR has one copy of HLA-A*31 and two copies of HLA-A*33.

In some embodiments, the subject having BSCR has two copies of HLA-A*29 and one copy of HLA-A*30. In some embodiments, the subject having BSCR has two copies of HLA-A*29 and one copy of HLA-A*31. In some embodiments, the subject having BSCR has two copies of HLA-A*29 and one copy of HLA-A*33. In some embodiments, the subject having BSCR has two copies of HLA-A*30 and one copy of HLA-A*31. In some embodiments, the subject having BSCR has two copies of HLA-A*30 and one copy of HLA-A*33. In some embodiments, the subject having BSCR has two copies of HLA-A*31 and one copy of HLA-A*33.

In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*29 and two copies of HLA-A*30. In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*29 and two copies of HLA-A*31. In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*29 and two copies of HLA-A*33. In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*30 and two copies of HLA-A*31. In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*30 and two copies of HLA-A*33. In some embodiments, the subject having BSCR or suspected of having BSCR has two copies of HLA-A*31 and two copies of HLA-A*33.

In some embodiments, the method further comprises administering to the subject an HLA-Aw19 inhibitor. In some embodiments, the HLA-Aw19 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-A*29 antibody. In some embodiments, the HLA-Aw19 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid

molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA (shRNA) that hybridizes to an HLA-Aw19. In some embodiments, the HLA-Aw19 is HLA-A*29.

In some embodiments, the MHC-I-opathy is AS. In some embodiments, the subject is or is suspected of being HLA-B*27$^+$ or HLA-B*40$^+$. In some embodiments, the subject has a single copy of HLA-B*27 or HLA-B*40. In some embodiments, the subject has two copies of HLA-B*27 or HLA-B*40. In some embodiments, the method further comprises administering to the subject an HLA-B*27 inhibitor or an HLA-B*40 inhibitor. In some embodiments, the HLA-B*27 inhibitor or HLA-B*40 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-B*27 antibody or an anti-HLA-B*40 antibody. In some embodiments, the HLA-B*27 inhibitor or HLA-B*40 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-B*27 or HLA-B*40.

In some embodiments, the MHC-I-opathy is Behçet's disease. In some embodiments, the subject is or is suspected of being HLA-B*51$^+$. In some embodiments, the subject has a single copy of HLA-B*51. In some embodiments, the subject has two copies of HLA-B*51. In some embodiments, the method further comprises administering to the subject an HLA-B*51 inhibitor. In some embodiments, the HLA-B*51 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-B*51 antibody. In some embodiments, the HLA-B*51 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-B*51.

In some embodiments, the MHC-I-opathy is psoriasis. In some embodiments, the subject is or is suspected of being HLA-C*06$^+$. In some embodiments, the subject has a single copy of HLA-C*06. In some embodiments, the subject has two copies of HLA-C*06. In some embodiments, the method further comprises administering to the subject an HLA-C*06 inhibitor. In some embodiments, the HLA-C*06 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-C*06 antibody. In some embodiments, the HLA-C*06 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.

In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-C*06.

In some embodiments, the MHC-I-opathy is JIA. In some embodiments, the subject is or is suspected of being HLA-B*27$^+$ and/or DRB1$^+$. In some embodiments, the subject has a single copy of HLA-B*27 and/or DRB1. In some embodiments, the subject has two copies of HLA-B*27 and/or DRB1. In some embodiments, the method further comprises administering to the subject an HLA-B*27 inhibitor and/or a DRB1 inhibitor. In some embodiments, the HLA-B*27 inhibitor and/or DRB1 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-B*27 antibody or an anti-DRB1 antibody. In some embodiments, the HLA-B*27 inhibitor and/or DRB1 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-B*27 and/or an DRB1.

In some embodiments, the MHC-I-opathy is IBD or CD. In some embodiments, the subject is or is suspected of being HLA-C*07$^+$. In some embodiments, the subject has a single copy of HLA-C*07. In some embodiments, the subject has two copies of HLA-C*07. In some embodiments, the method further comprises administering to the subject an HLA-C*07 inhibitor. In some embodiments, the HLA-C*07 inhibitor is an antibody. In some embodiments, the antibody is an anti-HLA-C*07 antibody. In some embodiments, the HLA-C*07 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to an HLA-C*07.

In any of the embodiments described herein, the methods can further comprise administering to the subject having an increased risk of developing the MHC-I-opathy-related HLA genotype and/or an MHC-II-opathy an ERAP2 inhibitor. In some embodiments, the ERAP2 inhibitor comprises a small molecule degrader, a proteoloysis-targeting chimera, an immunomodulatory drug, or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to ERAP2 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule that hybridizes to ERAP2 mRNA. In some

embodiments, the inhibitory nucleic acid molecule is an siRNA that hybridizes to ERAP2 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an shRNA that hybridizes to ERAP2 mRNA. In some embodiments, the ERAP2 inhibitor comprises an anti-ERAP2 antibody. In some embodiments, the ERAP2 inhibitor comprises a pseudopeptide. In some embodiments, the pseudopeptide is a phosphinic pseudopeptide. In some embodiments, the phosphinic pseudopeptide is DG002 or DG013. In some embodiments, the phosphinic pseudopeptide is DG002. In some embodiments, the phosphinic pseudopeptide is DG013. In some embodiments, the ERAP2 inhibitor comprises a small molecule.

In any of the embodiments described herein, the assay performed or having been performed on the biological sample from the subject can further determine whether the subject comprises a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein. When the subject has the MHC-I-opathy-related HLA genotype and/or MHC-II-opathy-related HLA genotype and lacks the functional ERAP1 protein or the nucleic acid molecule encoding the functional ERAP1 protein, then the subject has an increased risk of developing the MHC-I-opathy (for MHC-I-opathies except AS and psoriasis) and/or MHC-II-opathy. When the subject has the MHC-I-opathy-related HLA genotype and/or MHC-II-opathy-related HLA genotype and has a functional ERAP1 protein or a nucleic acid molecule encoding the functional ERAP1 protein, then the subject has an increased risk of developing the MHC-I-opathy (for AS and psoriasis). When the subject lacks the MHC-I-opathy-related HLA genotype and/or MHC-II-opathy-related HLA genotype, or has the functional ERAP1 protein or the nucleic acid molecule encoding the functional ERAP1 protein, or both, then the subject has a decreased risk of developing the MHC-I-opathy and/or MHC-II-opathy.

In any of the embodiments described herein, the methods can further comprise administering to the subject an ERAP1 agonist or inhibitor, depending upon the MHC-I-opathy. For AS and psoriasis, an ERAP1 inhibitor can be administered. For the remaining MHC-I-opathies, an ERAP1 agonist can be administered.

In some embodiments, the ERAP1 agonist comprises an oligonucleotide. In some embodiments, the oligonucleotide is ODN1826. In some embodiments, the ERAP1 agonist

comprises a peptide. In some embodiments, the ERAP1 agonist comprises a lipopeptide. In some embodiments, the lipopeptide is Pam3CSK4 or FSL-1. In some embodiments, the lipopeptide is Pam3CSK4. In some embodiments, the lipopeptide is FSL-1. In some embodiments, the ERAP1 agonist comprises a small molecule. In some embodiments, the ERAP1 agonist can comprise an ERAP1-specific transcriptional activator, an ERAP1 protein stabilizer, an agonist of ERAP1 enzymatic activity, or an activator of ERAP1 secretion. In some embodiments, the ERAP1 agonist can comprise an ERAP1-specific transcriptional activator. In some embodiments, the ERAP1 agonist can comprise an ERAP1 protein stabilizer. In some embodiments, the ERAP1 agonist can comprise an agonist of ERAP1 enzymatic activity. In some embodiments, the ERAP1 agonist can comprise an activator of ERAP1 secretion. Additional examples of ERAP1 agonists are described in, for example, Goto et al., J. Immunol., 2014, 192, 4443-4452.

In some embodiments, the ERAP1 inhibitor comprises a small molecule degrader, a proteoloysis-targeting chimera, an immunomodulatory drug, or an inhibitory nucleic acid molecule. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, an siRNA, or an shRNA that hybridizes to ERAP1 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an antisense nucleic acid molecule that hybridizes to ERAP1 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an siRNA that hybridizes to ERAP1 mRNA. In some embodiments, the inhibitory nucleic acid molecule is an shRNA that hybridizes to ERAP1 mRNA. In some embodiments, the ERAP1 inhibitor comprises an anti-ERAP1 antibody.

In some embodiments, the assay for determining whether the subject comprises an MHC-I-opathy-related and/or MHC-II-opathy-related HLA genotype and a functional ERAP2 protein and/or ERAP1 protein, or a nucleic acid molecule encoding a functional ERAP2 protein and/or ERAP1 protein, is a genotyping assay or sequencing assay. In some embodiments, the nucleic acid molecule encoding a functional ERAP2 protein and/or ERAP1 protein comprises genomic DNA, mRNA, or cDNA obtained from mRNA. By comparing the nucleotide or protein sequence of the ERAP2 protein and/or ERAP1 protein in the sample from a subject to the wild type sequence for ERAP2 protein and/or ERAP1 protein or nucleic acid molecule, or to published sequences of variant ERAP2 proteins and/or ERAP1 proteins

or nucleic acid molecules having reduced or no activity, a determination can be made whether the subject comprises a functional ERAP2 protein and/or ERAP1 protein, or a nucleic acid molecule encoding a functional ERAP2 protein and/or ERAP1 protein. In addition, although an individual ERAP2 protein and/or ERAP1 protein may have biological activity, the overall function of the ERAP2 protein and/or ERAP1 protein may not be functional due to reduced levels of expression. Thus, as used herein, an ERAP2 protein and/or ERAP1 protein can be determined not to be functional because the ERAP2 protein and/or ERAP1 protein lacks or had reduced biological activity or because the expression level is reduced.

Determining whether a subject has an MHC-I-opathy-related and/or MHC-II-opathy-related HLA genotype and/or a functional ERAP2 protein and/or ERAP1 protein, or a nucleic acid molecule encoding a functional ERAP2 protein and/or ERAP1 protein, in a biological sample from a subject can be carried out by any of the methods described herein. In some embodiments, these methods can be carried out *in vitro*. In some embodiments, these methods can be carried out *in situ*. In some embodiments, these methods can be carried out *in vivo*. In any of these embodiments, the nucleic acid molecule can be present within a biological sample obtained from the subject.

The biological sample can be derived from any cell, tissue, or biological fluid from the subject. The biological sample may comprise any clinically relevant tissue, such as a bone marrow sample, a tumor biopsy, a fine needle aspirate, or a sample of bodily fluid, such as blood, gingival crevicular fluid, plasma, serum, lymph, ascitic fluid, cystic fluid, or urine. In some cases, the sample comprises a buccal swab. The biological sample used in the methods disclosed herein can vary based on the assay format, nature of the detection method, and the tissues, cells, or extracts that are used as the sample. A biological sample can be processed differently depending on the assay being employed. For example, when detecting any particular nucleic acid molecule, preliminary processing designed to isolate or enrich the biological sample for the particular nucleic acid molecule can be employed. A variety of techniques may be used for this purpose. Various methods to detect the presence or level of an mRNA molecule or the presence of a particular genomic DNA locus can be used.

In some embodiments, the biological sample comprises a cell or cell lysate. Such methods can further comprise, for example, obtaining a biological sample from the subject comprising genomic nucleic acid molecules or mRNA molecules, and if mRNA, optionally reverse transcribing the mRNA into cDNA. In some embodiments, the method is an *in vitro* method. In some embodiments, the assay comprises RNA sequencing (RNA-Seq). In some embodiments, the assays also comprise reverse transcribing mRNA into cDNA, such as by the reverse transcriptase polymerase chain reaction (RT-PCR).

Detecting the presence or absence of any particular HLA allele can be carried out by numerous techniques. Detection of HLA-A alleles on a 2-digit and 4-digit resolution can be carried out. For example, an assay that targets the HLA region in high resolution (all class-I and class-II genes) can be used. In some embodiments, the assay amplifies the full HLA gene (in this case HLA-A) from the 5'UTR to the 3'UTR and provides genetic variants across the full amplicon (the DNA that is the product of this amplification of the gene). A method can then be used to call the HLA-A alleles with high accuracy (e.g., PHLAT2; Bai et al., Methods Mol. Biol., 2018, 1802, 193-201). The output of PHLAT2 provides the HLA-A 4-digits allele data for each sample, which can be used for the analysis that identified other Aw19 alleles as enriched in Birdshot cases. In addition, commercial sources of HLA typing are available.

Detecting the presence or absence a functional ERAP2 protein and/or ERAP1 protein, or a nucleic acid molecule encoding a functional ERAP2 protein and/or ERAP1 protein, can be carried out by numerous techniques. For example, detection of presence or absence of ERAP2 protein and the relevant nucleotide sequence can be carried out as described in Andres et al., PLoS Genetics, 2010, 6, 1-13. For example, a subject having an ERAP2 intronic variant designated rs2248374-A has a functional ERAP2 protein, or a nucleic acid molecule encoding a functional ERAP2 protein, and has an increased risk of developing the MHC-I-opathy. A subject having an ERAP2 variant designated rs10044354, HapA has a functional ERAP2 protein, or a nucleic acid molecule encoding a functional ERAP2 protein, and has an increased risk of developing the MHC-I-opathy. In addition, a subject having an ERAP1 intronic variant designated rs27432-G does not have a functional ERAP1 protein, or a nucleic acid molecule encoding a functional ERAP1 protein, and has an increased risk of

developing the MHC-I-opathy. A subject having an ERAP1 variant designated rs2287987, Hap 10 does not have a functional ERAP1 protein, or a nucleic acid molecule encoding a functional ERAP1 protein, and has an increased risk of developing the MHC-I-opathy. A subject having an ERAP2 splice variant designated rs2248374-G does not have a functional ERAP2 protein, or a nucleic acid molecule encoding a functional ERAP2 protein, and has a decreased risk of developing the MHC-I-opathy.

In order that the subject matter disclosed herein may be more efficiently understood, examples are provided below. It should be understood that these examples are for illustrative purposes only and are not to be construed as limiting the claimed subject matter in any manner. Throughout these examples, molecular cloning reactions, and other standard recombinant DNA techniques, were carried out according to methods described in Maniatis et al., Molecular Cloning - A Laboratory Manual, 2nd ed., Cold Spring Harbor Press (1989), using commercially available reagents, except where otherwise noted.

Examples

Example 1: Methods

Study subjects and samples

The genomic DNA samples from 286 patients with BSCR and 108 unrelated healthy local French volunteers that exhibited HLA tissue typing common in the French population were included in this study. The patients were recruited at Hôpital Cochin, Paris, France. All patients met the criteria for diagnosis of BSCR as defined both by an international consensus conference held in 2002 and by the Standardization of Uveitis Nomenclature (SUN) Working Group. In brief, all patients had a posterior bilateral uveitis with multifocal cream-colored or yellow-orange, oval or round choroidal lesions (“birdshot spots”). Although the presence of the HLA-A*29 allele was not a requirement for the diagnosis of BSCR according to the international criteria, all patients included in the current study carried the HLA-A*29 allele. The control DNA samples were collected from volunteer donors recruited by the hematopoietic stem cell donor center of Rheims for France Greffe de Moelle Registry, and local control healthy individuals of the Registry. The DNA samples were isolated from peripheral blood samples using a standard salting out method or QIAamp Blood Kit (Qiagen,

Chatsworth, CA, USA). Quality and quantity of DNA was determined by UV spectrophotometry and the concentration was adjusted to 100 ng/ml. Signed informed consent documentation was obtained from all participants, and all research adhered to the tenets set forth in the Declaration of Helsinki. All study-related data acquisitions were approved by the Paris Cochin institutional review board.

Genetic data

A comprehensive approach was taken to both sequence the exomes and genotype all samples, to allow for identification of common and rare variants filtered based on high quality calls. DNA from participants was genotyped on the Illumina Global Screening Array (GSA) and imputed to the HRC reference panel. Prior to imputation, the variants that had a MAF >= 0.1%, missingness < 1% and HWE p-value > 10^{-15} were retained. Imputation using the HRC reference panel yielded 8,385,561 variants with imputation INFO>0.3 and MAF>0.5%.

Exome sequencing was performed to a mean depth of 31X, followed by variant calling and quality control as reported previously (Van Hout et al., Nature, 2020, 586, 749-756), resulting in 238,942 variants. When integrated, this produced an overall dataset with 8,459,907 variants: 65.5% common (MAF > 5%), 34.5% low-frequency (0.5% < MAF < 5%) and 0.01% rare (MAF < 0.5%).

HLA genotyping

HLA Class I genes (HLA-A, -B, and -C) were amplified in a multiplex PCR reaction with primers encompassing the full genomic loci for each target. The resulting amplicons were enzymatically fragmented to an average size of 250 base pairs and prepared for Illumina sequencing (New England Biolabs, Ipswich, MA). The libraries were sequenced on the Illumina HiSeq 2500 platform on a rapid run flow cell using paired-end 125 base pair reads with dual 10 base pair indexes. Upon completion of sequencing, raw data from each Illumina HiSeq run was gathered in local buffer storage and uploaded to the DNAnexus platform (Reid et al., BMC Bioinformatics, 2014, 15, 30) for automated analysis. The FASTQ-formatted reads were converted from the BCL files and assigned to samples identified by specific barcodes using the bcl2fastq conversion software (Illumina Inc., San Diego, CA). All the reads in sample-specific FASTQ files were subject to HLA typing analysis using an

updated version of PHLAT program (Bai et al., BMC Genomics, 2014, 15, 325) with the reference sequences consisting of GRCh38 genomic sequences and HLA type reference sequences in the IPD-IMGT/HLA database v3.30.0 (Robinson et al., Hum. Immunol., 2016, 77, 233-237).

In addition, HLA allele imputation was performed following SNP2HLA (Jia et al., PLoS One, 2013, 8, e64683) with the T1DGC HLA allele reference panel (Rich et al., Ann. N.Y. Acad. Sci., 2006, 1079, 1-8). HRC-imputed genotypes in the extended Major Histocompatibility Complex (MHC) region (chr6:25-35Mb) were filtered for high INFO score (>0.9) and certainty (maximum GP>0.8 for all genotyped), in order to increase overlap with the T1DGC reference panel, were re-phased along with chromosome 6 array genotypes using SHAPEIT4 (Delaneau et al., Nat. Commun., 2019, 10, 5436), and were imputed using Minimac4 (Das et al., Nat. Genet., 2016, 48, 1284-1287). HLA allele imputation quality was assessed by examining INFO score vs MAF, and imputed vs reference panel MAF.

Genetic association analyses

Association analyses in each study were performed using the genome-wide Firth logistic regression test implemented in SAIGE (Mbatchou et al., bioRxiv, 2020, 2020.2006.2019.162354, doi:10.1101/2020.06.19.162354; and Zhou et al., Nat. Genet., 2018, 50, 1335-1341). In this implementation, Firth's approach is applied when the p-value from standard logistic regression score test is below 0.05. The directly genotyped variants with a minor allele frequency (MAF) >1%, <10% missingness, Hardy-Weinberg equilibrium test *P*-value>10^{-15} and linkage-disequilibrium (LD) pruning (1000 variant windows, 100 variant sliding windows and r^2<0.1) were included for GRM for SAIGE. The association model included as covariates sex and the first 10 ancestry-informative principal components (PCs) derived from the GRM dataset. Haplotype analyses were performed using PLINK 1.0 (Purcell et al., Am. J. Hum. Genet., 2007, 81, 559-575) *--chap* and *--hap-assoc* and *--hap-logistic*, and in *R*. High haplotype imputation and phasing quality was indicated by PLINK *--hap-phase* maximum likelihood haplotype genotypes' posterior probabilities all equal to one.

HLA-A allele association analyses

Association of HLA-A alleles was performed as follows: for each sample, both HLA-A alleles were typed as described above. Following HLA allele typing, related samples were

removed. For the remaining cohort of 282 cases and 106 controls, one HLA-A allele that is not A*29 (the "second" allele) was obtained next. Samples carrying two copies of A*29, were considered having A*29 as the second allele. The cohort was then subjected to a Fisher's exact test, which tested the association of each allele that was identified in three or more BSCR cases, with the case-control status. To answer the question of whether the A19 allele group is also associated with the case-control status, the samples were combined, and tested together in two different ways: carrying all Aw19 alleles (A*29, A*30, A*31, A*32 and A*33). Since A*32 is biologically different than the other Aw19 alleles in its peptide binding domain, a group that is made of samples carrying all Aw19 alleles excluding A*32 was also constructed and tested. The final odds-ratios and p-values are presented in the table in Figure 1.

Example 2: HLA-Aw19 Broad Antigen Serotype Alleles and BSCR Risk

The HLA-A29-controlled cohort allowed for examination of the HLA region while controlling for the strong association of HLA-A29 with BSCR, and therefore to detect possible additional association signals in the HLA region.

First, it was asked whether rare variants on the HLA-A29 background were enriched in BSCR cases. No significant enrichments of rare single or aggregated variants were identified either within or outside the MHC region.

Second, the question was whether other HLA-A alleles in addition to the HLA-A29 allele increased BSCR risk. An assay to type HLA-A alleles in this cohort (see Methods) was constructed, and tested the second HLA-A allele (other than the known first HLA-A29) was tested for association with BSCR. Additional HLA-A alleles were found to be associated with BSCR, and those with the largest effects belonged to the same HLA-Aw19 broad antigen serotype group: HLA-A29:02, A30:02, A31:01 and A33:01 (Figure 1). As a group, HLA-Aw19 alleles were significantly enriched in the second allele of BSCR patients (OR=4.44, p=2.2e-03, Figure 2, blue bars). This result suggests, for example, that individuals carrying two copies of HLA-A29 would be at a greater risk of developing BSCR compared to those carrying one copy. It also suggests that other Aw19 allele may play a role in BSCR co-susceptibility or pathogenesis in concert with A29. The sole exception within the HLA-Aw19 serotype group

is HLA-A32, which has been reported not to share the defining Aw19 binding domain (McKenzie et al., Genes Immun., 1999, 1, 120-129); HLA-A32 appears to be depleted in BSCR cases and thus protective against BSCR (OR=0.28, p=0.1).

The above results presented two issues due to the small numbers of controls in UParis (n=108): 1) The frequency of alleles might not represent the frequency of HLA-A alleles in general EUR population. 2) While the high ORs replicate in several HLA-Aw19 alleles, the numbers are not sufficient to support significant associations. To tackle these concerns, the frequency of HLA-A alleles in three other large European (EUR) ancestry control populations, two cohorts from the Geisinger Health System (GHS cohort #1, n=77,198 and GHS cohort #2, n=59,072) and the UK Biobank (UKB, n=463,315) were examined. In all three datasets, the EUR samples carrying at least one HLA-A29 allele were selected, matching the BSCR cohort: 4,014 A29 carriers from GHS cohort #1 (5.2% of all EUR subjects), 2,829 A29 carriers from GHS cohort #2 (4.8% of all EUR), and 38,543 A29 carriers from UKB (8.3% of all EUR). The frequencies of the second HLA-A alleles in these cohorts were compared to those observed in the BSCR cohort (Figure 2, Figure 1). The results support the enrichment of four of the five HLA-Aw19 alleles in BSCR cases, with highest increased risk for HLA-A30:02 (GHS cohort #1 OR=4.31, GHS cohort #2 OR=6.6, UKB OR= 4.6) and HLA-A33 (GHS cohort #1 OR=3.4, GHS cohort #2 OR=2.8, UKB OR= 4.9). When combining samples carrying the four co-susceptibility alleles A29, A30, A31 and A33, was found a highly significant enrichment in BSCR cases was found a when compared with the larger control cohorts (GHS cohort #1 p-val= 1.29E-06, GHS cohort #2 p-val= 1.07E-06, UKB p-val= 9.62E-07, Figure 1 top row). This analysis excludes A32 because of its biological difference in the sequence of the peptide binding domain as previously reported. The additional analyses with all Aw19 alleles including A32 showed that the enrichment in cases is reduced when it is included (Figure 1 bottom row).

In order to test whether these associations are affected by measurable confounders, logistic regression tests were conducted to evaluate the effects of the second HLA-A allele in HLA-A29 carriers, in UParis BSCR cases compared with each control cohort, with covariates included for sex and principal components, calculated based on genetic

array data for each analytic set (Figure 3). The results were consistent with increased risk for the HLA-Aw19 co-susceptibility alleles, A29, A30, A31, and A33.

Example 3: HLA-A32 Exhibits Protection from BSCR in an HLA-A29 Positive Cohort

HLA-A32 is underrepresented in BSCR cases (3/286, ~1%) versus A29 carrier controls (4/108, 3.7%), corresponding to a nominally significant protection from risk (OR=0.28, p=0.1; Figure 1). When compared with the larger control cohorts, the trend protection is maintained with both UKB controls (3.4%, OR=0.3, p=0.02) and GHS controls (cohort #1: 3.8%, OR=0.27, p=0.01; cohort #2: 3.7%, OR=0.27, p=0.02). While nominally significant, this result does not pass the threshold of multiple test correction (p=3.57e-03) and will need to be further validated with additional case cohorts.

Example 4: ERAP1 and ERAP2 are Independently Associated with BSCR

All variants and gene burdens were tested for association with case-control status, while controlling for sex and ten principal components, using a generalized linear mixed model (SAIGE). Due to the fact that both cases and controls were A29 allele carriers, the expected strong HLA-A signal was at least partially controlled, as evidenced by the strongest HLA p-value = 8.98E-07, compared with p=6.6e-74 with 125 cases in the previous BSCR report (Kuiper et al., Hum. Mol. Genet., 2014, 23, 6081-6087). Overall, no locus passed the genome wide significance threshold (p< 5e-8). Other than the remnant signal at HLA-A, only the *ERAP1/ERAP2-LNPEP* locus on chromosome 5 showed an association with disease at p<1e-6 (Figure 2).

The top association within the *ERAP1/ERAP2-LNPEP* locus is the *ERAP1* intronic variant rs27432 (OR (95% CI)= 2.58 (1.78 – 3.76), p= 6.6e-7), a strong eQTL associated with decreased ERAP1 expression (Kuiper et al., Hum. Mol. Genet., 2018, 27, 4333-4343; and Paladini et al., Sci. Rep., 2018, 8, 10398), which also tags the risk-increasing common ERAP1 haplotype. A haplotype analysis was further performed to assess *ERAP1* haplotype associations with BSCR status in the present data. The results were consistent with three levels of risk differentiated by nonsynonymous ERAP1 variant haplotypes corresponding to Kuiper et al. Haps 1+2 (OR=0.41, Cases AF=0.17, controls AF=0.35, p=6.7e-06), Hap10

(OR=1.78, Cases AF=0.28, controls AF=0.17, p=8.0e-03), and haplotypes 3-8 (OR=1.32, Cases AF=0.55, controls AF=0.48, p=0.11) (Figure 4).

The previously reported top association for BSCR at this locus tags a common variant near ERAP2/LNPEP, rs10044354. This reported risk allele is in a strong linkage disequilibrium (D'=0.99, R^2=0.76), with a strong eQTL increasing *ERAP2* expression. The results show a nominal association of rs10044354 with increased risk for Birdshot (OR (95% CI) = 1.55 (1.13 - 2.11), p= 5.8e-3). Furthermore, no significant evidence was found for an interaction of rs10044354 with rs27432-rs2287987 haplotypes (conditional haplotype test p=0.46).

Next, a meta-analysis of the results with the published results from Kuiper et al. was carried out, which yielded genome-wide significant associations for both ERAP1 (rs27432, OR (95% CI)=2.46 (1.85-3.26), p=4.07e-10) and ERAP2 (rs10044354, OR (95% CI)=1.95 (1.55-2.44), p=6.2e-09) loci with BSCR (Figure 1). Both previous and current studies showed consistent directionality for both variants, which, separated by over 201,222 bp, show low linkage disequilibrium (LD) in the present cohort (R2=0.18, D'=0.79).

The expression of *ERAP2* has been previously reported to be disrupted by a common splice region variant (rs2248374, AF=0.53) that causes mis-splicing of intron 10 and eventual transcript degradation via nonsense-mediated decay (Andres et al., PLoS Genet., 2010, 6, e1001157; and Coulombe-Huntington et al., PLoS Genet., 2009, 5, e1000766), and which is in high LD with rs10044354 (R2=0.8, D'=1). Thus, about 25% of the population of most ancestries (including European, AF=0.53; African, AF=0.57 and South Asian, AF=0.58) is estimated to be lacking an active ERAP2 protein. Both datasets were examined for rs2248374 associations and found that it is protective for BSCR with nominal significance in both datasets (Figure 5). Furthermore, ERAP2-rs2248374 that disrupts ERAP2 expression is protective (OR 0.56; 95% CI [0.45-0.70]; p=2.39e-07; Figure 6). In summary, higher expression of ERAP2 protein increases risk for BSCR and a lower expression is protective.

Example 5: Cumulative Effect of HLA-Aw19 Alleles and *ERAP1/ERAP2* Haplotypes on BSCR Risk

The potential interactions between the *ERAP1* and *ERAP2* association signals and between HLA-Aw19 and *ERAP1/ERAP2* signals was examined by calculating the cumulative effects of HLA-Aw19, ERAP1 and ERAP2 genotypes on BSCR risk using the 286 cases and the 4,014 A29 carriers from the GHS cohort #1. First, an analysis of ERAP2-rs10044354 risk haplotype, the top non-MHC signal in Kuiper et al. was performed, stratified by single (A29/-) versus double (A29/AW19) Aw19 background, which yielded a trend of increased risk with additional ERAP2-rs10044354-T variant alleles, particularly on the double A29/AW19 background (Figure 7, Panel A). The combination of rs10044354-TT and two copies of Aw19 with 12 cases and 34 controls was found to have the highest risk (OR=9.9 [4.4-21.2], p=1.66e-07, Figure 8).

A similar analysis of the ERAP1-rs27432 risk haplotype, the top non-MHC association, stratified by single (A29/-) versus double (A29/AW19) Aw19 background, yielded the same trend of increased risk with additional ERAP1-rs27432-G variant alleles, particularly on the double A29/AW19 background (OR=6.2 [2.7-15.51], p=1.54e-06, Figure 7, Panel B and Figure 9).

The combined effects of the ERAP1 risk haplotype tagged by rs27432, and the ERAP2 risk haplotype tagged by rs10044354 were calculated next (Figure 7, Panel C). It was found that the highest risk is conferred by the combination of ERAP1-rs27432-GG and ERAP2-rs10044354-TT (OR=3.6 [1.62-9.45], p=4.03e-04, Figure 10), and as mentioned above, these data are consistent with additive effects of the variants/haplotypes.

Next, all risk haplotypes were combined to a single risk analysis. Due to the small number of cases, the genotypes of intermediate genotypes were combined into four main groups: 1) homozygous to the protective alleles in both ERAP1 and ERAP2, homozygous in one and 2) heterozygous in the other, 3) homozygous risk allele in either ERAP1 or ERAP2, and 4) homozygous risk allele in both ERAP1 or ERAP2 (Figure 7, Panel D and Figure 11). A gradual increase in risk with the addition of each risk allele was observed, with the highest risk presented when carrying homozygous risk alleles in both ERAP1 and ERAP2, on top of two copies of A19 alleles (OR=13.53 [3.79-54.77], p=1.17e-05). These results suggest that both ERAP1 and/or ERAP2 confer greater BSCR risk, which is further increased in the double Aw19 background.

Example 6: Absolute BSCR Risk

The calculation of the absolute risk of BSCR when considering all risk alleles is presented in Figure 7, Panel D. Since the prevalence of BSCR in general population is estimated at 0.2-1.7:100,000 (Minos et al., Orphanet J. Rare Dis., 2016, 11, 61), 1:100,000 was used as an approximation. The absolute risk was further calculated when carrying one A29 carrier based on the frequency of A29 in UKB EUR population of 8%, and reach an absolute risk of 1:29,000 (Figure 11). It was observed that the absolute risk climbs with each risk genotype presented in Figure 7, Panel D, reaching the most prominent risk at 1:2,160 for cases that carry homozygous risk alleles for both ERAP1 and ERAP2, and two copies of Aw19 alleles. Exhibiting a significant increase in absolute risk of disease when carrying all three risk haplotypes.

Example 7: Discussion

The sequencing of a new large BSCR patient cohort and HLA-A*29 controls has confirmed the importance of the ERAP1 and ERAP2 polymorphisms in increasing risk for developing BSCR. ERAP1 and ERAP2 reside back-to-back on chromosome five in opposite orientation and share the regulatory regions, which upregulate one and downregulate the other, and vice versa. The association of both ERAP1 and ERAP2 haplotypes is consistent with a mechanism in which coordinated decreased ERAP1 and increased ERAP2 expression contribute to disease risk. Several studies have reported that the ERAP1 and ERAP2 haplotypes affect their expression as well as the resulting peptidome (Kuiper et al., Hum. Mol. Genet., 2018, 27, 4333-4343; Paladini et al., Sci. Rep., 2018, 8, 10398; and Sanz-Bravo et al., Mol. Cell Proteomics, 2018, 17, 1564-1577).

The present study found that several other HLA-Aw19 family alleles (HLA- A29, A30, A31, A33) contribute additional risk as the second HLA-A allele, in addition to HLA-A29 risk allele. HLA-Aw19 family alleles have a similar antigen-binding sequence and therefore would bind similar peptide motifs. Hence, the enrichment of Aw19 alleles in cases supports the inferred mechanism underlying activation of the immune response in BSCR: having two copies of these alleles may increase the cell-surface presentation of specific types of

peptides in BSCR cases compared to HLA-A29 positive controls. Furthermore, it was found that the HLA-A32 allele within the Aw19 family is potentially protective.

These results indicate that a decreased expression of ERAP1 and an increased expression of ERAP2 confer stronger risk for BSCR than each one separately. Furthermore, this effect is increased in the presence of two copies of HLA-Aw19. The combined and additive effect of risk factors associated with peptide processing and presentation is suggestive of a peptide presentation threshold hypothesis as a driving mechanism for the immune response underlying development of BSCR disease. Results from this and other studies suggest that increased ERAP2 along with decreased ERAP1 expression in BSCR cases would lead to higher availability of ERAP2-processed peptides for presentation onto HLA class I proteins. Additional HLA-Aw19 alleles, with similar peptide-binding properties, would increase presentation of similar peptides. Therefore, both the production of a unique peptide pool by dominant ERAP2 activity and the increased expression of HLA-Aw19 risk allele proteins presenting these peptides may increase the likelihood that a putative ocular autoantigen would be processed and presented above a certain threshold to activate an immune response. On the other hand, having lower expression of ERAP2 (and higher expression of ERAP1), along with a single HLA-A*29 allele, lowers the ocular antigenic peptide presentation below the threshold and thus reduces the risk of generating the immunological response leading to BSCR in HLA-A*29 healthy control carriers. This further highlights the importance of the shaping and generation of the available peptide pool by ERAPs to be presented by specific HLA class I proteins in promoting the generation of an immune response or, in the case of autoimmune disease, an aberrant response to a self-antigen.

ERAP1 and ERAP2 polymorphisms and risk haplotypes have also been reported in other HLA class I-associated autoimmune diseases (Babaie et al., Mol. Immunol., 2020, 121, 7-19; and Yao et al., Hum. Immunol., 2019, 80, 325-334). Polymorphisms in ERAP1 increase risk for Ankylosing Spondylitis in HLA-B*27 carriers, for psoriasis vulgaris in HLA-C*06 carriers, and for Behçet's disease in HLA-B*51 carriers, further supporting the combinatorial impact of peptide trimming and HLA class I allele in initiating autoimmune responses (Evans et al., Nat. Genet., 2011, 43, 761-767; Wisniewski et al., Hum. Immunol., 2018, 79, 109-116;

Nat. Genet., 2010, 42, 985-990; and Takeuchi et al., Ann. Rheum. Dis., 2016, 75, 2208-2211). Ankylosing Spondylitis and Behçet's disease-associated ERAP1 variants have also been experimentally shown to shape the resulting HLA-B*27 and HLA-B*51 peptidome, respectively (Sanz-Bravo et al., Mol. Cell Proteomics, 2018, 17, 1308-1323; and Guasp et al., J. Biol. Chem., 2017, 292, 9680-9689). Therefore, it is possible that the combination of risk ERAP1/ERAP2 haplotypes and specific risk HLA class I alleles can predispose an individual to develop an HLA class I associated disease in a similar fashion as it is hypothesized for BSCR. This implies that the peptide threshold hypothesis may have broader implications as a disease mechanism in HLA class I associated immunological diseases.

HLA-A32 is the only HLA-Aw19 member that is found at lower rates in BSCR patients compared to controls, suggesting that it could be protective. The HLA-Aw19 serotype was initially identified by antibody binding to related family members; however, this identifies the HLA-A proteins based on structure outside of the peptide-binding groove. Serofamilies have since been re-analyzed by overall and peptide binding region sequences (McKenzie et al., Genes Immun. 1999, 1, 120-129). Comparison of the sequences in the peptide binding region reveals that HLA-A32 is more distantly related than the other Aw19 alleles which are identified as novel risk factors in this present study: HLA-A29, A30, A31, A33. When examining the differences in sequence between these Aw19 alleles, two main differences are evident: at position 9, which is part of the peptide binding domain, and a stretch of amino-acids at positions 79-83 that is only found in HLA-A32 and not the other Aw19 alleles (Figure 12). Theoretically, the peptide pool bound by HLA-A32 would differ from the remaining members of the Aw19 family and would not activate the same subset of responding CD8 T cells. This adds further evidence supporting the hypothesis of the threshold requirement of an increased concentration of the driving autoantigenic peptide pool presented on high-risk HLA-A proteins as a driving component for development of BSCR uveitis.

In summary, the combinatorial impact of ERAP1/2 shaping the immunopeptidome along with differential peptide selection by the key residues in HLA-A29 and HLA-Aw19 family members supports the immunological hypothesis of a peptide pool that is generated by this combination and available for immune cell recognition and activation initiating an

inflammatory cascade. Avenues to reduce the expression and recognition of ERAP2-processed and HLA-Aw19-presented peptides in the eye may be beneficial against BSCR disease and/or progression.

Various modifications of the described subject matter, in addition to those described herein, will be apparent to those skilled in the art from the foregoing description. Such modifications are also intended to fall within the scope of the appended claims. Each reference (including, but not limited to, journal articles, U.S. and non-U.S. patents, patent application publications, international patent application publications, gene bank accession numbers, and the like) cited in the present application is incorporated herein by reference in its entirety.

What Is Claimed Is:

~~1. A method of treating a subject having an immune disorder, the method comprising administering to the subject a therapeutically effective amount of an Endoplasmic Reticulum Aminopeptidase 2 (ERAP2) inhibitor.~~

~~2. The method according to claim 1, wherein the immune disorder is an MHC-I-opathy.~~

~~3. The method according to claim 2, wherein the MHC-I-opathy is Birdshot Chorioretinopathy (BSCR).~~

~~4. The method according to claim 3, the method further comprising detecting the presence or absence of an HLA-Aw19 allele in a biological sample obtained from the subject.~~

~~5. The method according to claim 4, the method further comprising determining whether the subject has one or two copies of an HLA-Aw19 allele.~~

~~6. The method according to any one of claims 3 to 5, wherein the subject is HLA-Aw19$^+$.~~

~~7. The method according to claim 6, wherein the subject is HLA-A*29$^+$, HLA-A*30$^+$, HLA-A*31$^+$, or HLA-A*33$^+$, or any combination thereof.~~

~~8. The method according to claim 6 or claim 7, wherein the subject has two copies of HLA-Aw19.~~

~~9. The method according to claim 8, wherein the subject is HLA-A*29$^+$/HLA-A*30$^+$.~~

~~10. The method according to claim 8, wherein the subject is HLA-A*29$^+$/HLA-A*31$^+$.~~

~~11. The method according to claim 8, wherein the subject is HLA-A*29$^+$/HLA-A*33$^+$.~~

~~12. The method according to any one of claims 3 to 11, the method further comprising administering to the subject an HLA-Aw19 inhibitor.~~

~~13. The method according to claim 12, wherein the HLA-Aw19 inhibitor is an antibody.~~

~~14. The method according to claim 13, wherein the antibody is an anti-HLA-A*29 antibody.~~

~~15. The method according to claim 12, wherein the HLA-Aw19 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~16. The method according to claim 15, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-Aw19.~~

~~17. The method according to claim 16, wherein the HLA-Aw19 is HLA-A*29.~~

~~18. The method according to claim 2, wherein the MHC-I-opathy is Ankylosing Spondylitis (AS).~~

~~19. The method according to claim 18, the method further comprising detecting the presence or absence of HLA-B*27 or HLA-B*40 in a biological sample obtained from the subject.~~

~~20. The method according to claim 19, the method further comprising determining whether the subject has one or two copies of HLA-B*27 or HLA-B*40.~~

~~21. The method according to any one of claims 18 to 20, wherein the subject is HLA-B*27$^+$ or HLA-B*40$^+$.~~

~~22. The method according to claim 21, wherein the subject has two copies of HLA-B*27 or HLA-B*40.~~

~~23. The method according to any one of claims 18 to 22, the method further comprising administering to the subject an HLA-B*27 inhibitor or an HLA-B*40 inhibitor.~~

~~24. The method according to claim 23, wherein the HLA-B*27 inhibitor or HLA-B*40 inhibitor is an antibody.~~

~~25. The method according to claim 24, wherein the antibody is an anti-HLA-B*27 antibody or an anti-HLA-B*40 antibody.~~

~~26. The method according to claim 23, wherein the HLA-B*27 inhibitor or HLA-B*40 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~27. The method according to claim 26, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-B*27 or an HLA-B*40.~~

~~28. The method according to claim 2, wherein the MHC-I-opathy is Behçet's disease.~~

~~29. The method according to claim 28, the method further comprising detecting the presence or absence of HLA-B*51 in a biological sample obtained from the subject.~~

~~30. The method according to claim 29, the method further comprising determining whether the subject has one or two copies of HLA-B*51.~~

~~31. The method according to any one of claims 28 to 30, wherein the subject is HLA-B*51+.~~

~~32. The method according to claim 31, wherein the subject has two copies of HLA-B*51.~~

~~33. The method according to any one of claims 28 to 32, the method further comprising administering to the subject an HLA-B*51 inhibitor.~~

~~34. The method according to claim 33, wherein the HLA-B*51 inhibitor is an antibody.~~

~~35. The method according to claim 34, wherein the antibody is an anti-HLA-B*51 antibody.~~

~~36. The method according to claim 33, wherein the HLA-B*51 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~37. The method according to claim 36, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-B*51.~~

~~38. The method according to claim 2, wherein the MHC-I opathy is psoriasis.~~

~~39. The method according to claim 38, the method further comprising detecting the presence or absence of HLA-C*06 in a biological sample obtained from the subject.~~

~~40. The method according to claim 39, the method further comprising determining whether the subject has one or two copies of HLA-C*06.~~

~~41. The method according to any one of claims 38 to 40, wherein the subject is HLA-C*06+.~~

~~42. The method according to claim 41, wherein the subject has two copies of HLA-C*06.~~

~~43. The method according to any one of claims 38 to 42, the method further comprising administering to the subject an HLA-C*06 inhibitor.~~

~~44. The method according to claim 43, wherein the HLA-C*06 inhibitor is an antibody.~~

~~45. The method according to claim 44, wherein the antibody is an anti-HLA-C*06 antibody.~~

~~46. The method according to claim 43, wherein the HLA-C*06 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~47. The method according to claim 46, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-C*06.~~

~~48. The method according to claim 2, wherein the MHC-I-opathy is Juvenile Idiopathic Arthritis (JIA).~~

~~49. The method according to claim 48, the method further comprising detecting the presence or absence of HLA-B*27 in a biological sample obtained from the subject.~~

~~50. The method according to claim 49, the method further comprising determining whether the subject has one or two copies of HLA-B*27.~~

~~51. The method according to any one of claims 48 to 50, wherein the subject is HLA-B*27$^+$.~~

~~52. The method according to claim 51, wherein the subject has two copies of HLA-B*27.~~

~~53. The method according to any one of claims 48 to 52, the method further comprising administering to the subject an HLA-B*27 inhibitor.~~

~~54. The method according to claim 53, wherein the HLA-B*27 inhibitor is an antibody.~~

~~55. The method according to claim 54, wherein the antibody is an anti-HLA-B*27 antibody.~~

~~56. The method according to claim 53, wherein the HLA-B*27 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~57. The method according to claim 56, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-B*27.~~

~~58. The method according to claim 2, wherein the MHC-I-opathy is inflammatory bowel disease (IBD) or Crohn's disease (CD).~~

~~59. The method according to claim 58, the method further comprising detecting the presence or absence of HLA-C*07 in a biological sample obtained from the subject.~~

~~60. The method according to claim 59, the method further comprising determining whether the subject has one or two copies of HLA-C*07.~~

~~61. The method according to any one of claims 58 to 60, wherein the subject is HLA-C*07+.~~

~~62. The method according to claim 61, wherein the subject has two copies of HLA-C*07.~~

~~63. The method according to any one of claims 58 to 62, the method further comprising administering to the subject an HLA-C*07 inhibitor.~~

~~64. The method according to claim 63, wherein the HLA-C*07 inhibitor is an antibody.~~

~~65. The method according to claim 64, wherein the antibody is an anti-HLA-C*07 antibody.~~

~~66. The method according to claim 63, wherein the HLA-C*07 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~67. The method according to claim 66, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-C*07.~~

~~68. The method according to any one of claims 1 to 67, wherein the ERAP2 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~69. The method according to claim 68, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to ERAP2 mRNA.~~

~~70. The method according to any one of claims 1 to 67, wherein the ERAP2 inhibitor comprises an anti-ERAP2 antibody.~~

~~71. The method according to any one of claims 1 to 67, wherein the ERAP2 inhibitor comprises a pseudopeptide.~~

~~72. The method according to claim 71, wherein the pseudopeptide is a phosphinic pseudopeptide.~~

~~73. The method according to claim 72, wherein the phosphinic pseudopeptide is DG002 or DG013.~~

74. The method according to any one of claims 1 to 67, wherein the ERAP2 inhibitor comprises a small molecule.

75. The method according to any one of claims 1 to 74, further comprising administering to the subject a therapeutically effective amount of an Endoplasmic Reticulum Aminopeptidase 1 (ERAP1) agonist or inhibitor.

76. The method according to claim 75, wherein the ERAP1 agonist comprises an oligonucleotide.

77. The method according to claim 76, wherein the oligonucleotide is ODN1826.

78. The method according to claim 75, wherein the ERAP1 agonist comprises a lipopeptide.

79. The method according to claim 78, wherein the lipopeptide is Pam3CSK4 or FSL-1.

80. The method according to claim 75, wherein the ERAP1 agonist comprises a small molecule.

81. The method according to claim 75, wherein the ERAP1 inhibitor comprises an inhibitory nucleic acid molecule.

82. The method according to claim 75, wherein the ERAP1 inhibitor comprises a small molecule.

83. A method of treating a subject having an MHC-I-opathy, the method comprising:

performing or having performed an assay on a biological sample from the subject to determine whether the subject comprises:

i) an MHC-I-opathy-related HLA genotype; and

ii) a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein; and

administering to the subject a therapeutically effective amount of an ERAP2 inhibitor, wherein the subject comprises both an MHC-I-opathy-related HLA genotype and a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein;

wherein the presence of both the MHC-I-opathy-related HLA genotype and the functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein indicates that the subject is a candidate for treating the MHC-I-opathy by inhibiting ERAP2.

~~84. The method according to claim 83, wherein the MHC-I-opathy is Birdshot Chorioretinopathy (BSCR) and the MHC-I-opathy-related HLA genotype comprises an HLA-Aw19 allele.~~

~~85. The method according to claim 84, wherein HLA-Aw19 allele is an HLA-A*29 allele, an HLA-A*30 allele, an HLA-A*31 allele, or an HLA-A*33 allele, or any combination thereof.~~

~~86. The method according to claim 84 or claim 85, wherein the subject has two copies of the HLA-Aw19 allele.~~

~~87. The method according to claim 86, wherein the subject is HLA-A*29$^+$/HLA-A*30$^+$.~~

~~88. The method according to claim 86, wherein the subject is HLA-A*29$^+$/HLA-A*31$^+$.~~

~~89. The method according to claim 86, wherein the subject is HLA-A*29$^+$/HLA-A*33$^+$.~~

~~90. The method according to any one of claims 84 to 89, the method further comprising administering to the subject an HLA-Aw19 inhibitor.~~

~~91. The method according to claim 90, wherein the HLA-Aw19 inhibitor is an antibody.~~

~~92. The method according to claim 91, wherein the antibody is an anti-HLA-A*29 antibody.~~

~~93. The method according to claim 90, wherein the HLA-Aw19 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~94. The method according to claim 93, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-Aw19.~~

~~95. The method according to claim 94, wherein the HLA-Aw19 is HLA-A*29.~~

~~96. The method according to claim 83, wherein the MHC-I-opathy is Ankylosing Spondylitis (AS) and the MHC-I-opathy-related HLA genotype comprises an HLA-B*27 allele or an HLA-B*40 allele.~~

~~97. The method according to claim 96, wherein the subject has two copies of HLA-B*27 or HLA-B*40.~~

~~98. The method according to claim 96 or claim 97, the method further comprising administering to the subject an HLA-B*27 inhibitor or an HLA-B*40 inhibitor.~~

~~99. The method according to claim 96, wherein the HLA-B*27 inhibitor or HLA-B*40 inhibitor is an antibody.~~

~~100. The method according to claim 99, wherein the antibody is an anti-HLA-B*27 antibody or an anti-HLA-B*40 antibody.~~

~~101. The method according to claim 98, wherein the HLA-B*27 inhibitor or HLA-B*40 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~102. The method according to claim 101, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-B*27 or an HLA-B*40.~~

~~103. The method according to claim 83, wherein the MHC I opathy is Behçet's disease and the MHC I opathy-related HLA genotype comprises an HLA-B*51 allele.~~

~~104. The method according to claim 103, wherein the subject has two copies of HLA-B*51.~~

~~105. The method according to claim 103 or claim 104, the method further comprising administering to the subject an HLA-B*51 inhibitor.~~

~~106. The method according to claim 105, wherein the HLA-B*51 inhibitor is an antibody.~~

~~107. The method according to claim 106, wherein the antibody is an anti-HLA-B*51 antibody.~~

~~108. The method according to claim 105, wherein the HLA-B*51 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~109. The method according to claim 108, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-B*51.~~

~~110. The method according to claim 83, wherein the MHC I opathy is psoriasis and the MHC I opathy-related HLA genotype comprises an HLA-C*06 allele.~~

~~111. The method according to claim 110, wherein the subject has two copies of HLA-C*06.~~

~~112. The method according to claim 110 or claim 111, the method further comprising administering to the subject an HLA-C*06 inhibitor.~~

~~113. The method according to claim 112, wherein the HLA-C*06 inhibitor is an antibody.~~

~~114. The method according to claim 113, wherein the antibody is an anti-HLA-C*06 antibody.~~

~~115. The method according to claim 112, wherein the HLA-C*06 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~116. The method according to claim 115, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-C*06.~~

~~117. The method according to claim 83, wherein the MHC-I-opathy is Juvenile Idiopathic Arthritis (JIA) and the MHC-I-opathy-related HLA genotype comprises an HLA-B*27.~~

~~118. The method according to claim 117, wherein the subject has two copies of HLA-B*27.~~

~~119. The method according to claim 117 or claim 118, the method further comprising administering to the subject an HLA-B*27 inhibitor.~~

~~120. The method according to claim 119, wherein the HLA-B*27 inhibitor is an antibody.~~

~~121. The method according to claim 120, wherein the antibody is an anti-HLA-B*27 antibody.~~

~~122. The method according to claim 119, wherein the HLA-B*27 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~123. The method according to claim 122, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-B*27.~~

~~124. The method according to claim 83, wherein the MHC-I-opathy is inflammatory bowel disease (IBD) or Crohn's disease (CD) and the MHC-I-opathy-related HLA genotype comprises an HLA-C*07 allele.~~

~~125. The method according to claim 124, wherein the subject has two copies of HLA-C*07.~~

~~126. The method according to claim 124 or claim 125, the method further comprising administering to the subject an HLA-C*07 inhibitor.~~

~~127. The method according to claim 126, wherein the HLA-C*07 inhibitor is an antibody.~~

~~128. The method according to claim 127, wherein the antibody is an anti-HLA-C*07 antibody.~~

~~129. The method according to claim 126, wherein the HLA-C*07 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~130. The method according to claim 129, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-C*07.~~

~~131. The method according to any one of claims 83 to 130, wherein the nucleic acid molecule comprises genomic DNA, mRNA, or cDNA obtained from mRNA.~~

~~132. The method according to any one of claims 83 to 131, wherein the ERAP2 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~133. The method according to claim 132, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to ERAP2 mRNA.~~

~~134. The method according to any one of claims 83 to 131, wherein the ERAP2 inhibitor comprises an anti-ERAP2 antibody.~~

~~135. The method according to any one of claims 83 to 131, wherein the ERAP2 inhibitor comprises a pseudopeptide.~~

~~136. The method according to claim 135, wherein the pseudopeptide is a phosphinic pseudopeptide.~~

~~137. The method according to claim 136, wherein the phosphinic pseudopeptide is DG002 or DG013.~~

~~138. The method according to any one of claims 83 to 131, wherein the ERAP2 inhibitor comprises a small molecule.~~

~~139. The method according to any one of claims 83 to 138, wherein:~~

~~the assay performed or having been performed on the biological sample from the subject further determines whether the subject comprises a functional Endoplasmic Reticulum Aminopeptidase 1 (ERAP1) protein or a nucleic acid molecule encoding a functional ERAP1 protein; and~~

~~the method further comprises administering to the subject a therapeutically effective amount of an ERAP1 agonist, wherein the subject comprises an MHC-I-opathy-~~

related HLA genotype and does not comprise a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein;

wherein the presence of an MHC-I-opathy-related HLA genotype and the absence of a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein indicates that the subject is a candidate for treating the MHC-I-opathy by activating ERAP1.

140. The method according to claim 139, wherein the ERAP1 agonist comprises a comprises an oligonucleotide.

141. The method according to claim 140, wherein the oligonucleotide is ODN1826.

142. The method according to claim 139, wherein the ERAP1 agonist comprises a lipopeptide.

143. The method according to claim 142, wherein the lipopeptide is Pam3CSK4 or FSL-1.

144. The method according to claim 139, wherein the ERAP1 agonist comprises a small molecule.

145. The method according to any one of claims 83 to 138, wherein:

the assay performed or having been performed on the biological sample from the subject further determines whether the subject comprises a functional Endoplasmic Reticulum Aminopeptidase 1 (ERAP1) protein or a nucleic acid molecule encoding a functional ERAP1 protein; and

the method further comprises administering to the subject a therapeutically effective amount of an ERAP1 inhibitor, wherein the subject comprises an MHC-I-opathy-related HLA genotype and comprises a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein;

wherein the presence of an MHC-I-opathy-related HLA genotype and the presence of a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein indicates that the subject is a candidate for treating the MHC-I-opathy by inhibiting ERAP1.

146. The method according to claim 145, wherein the ERAP1 inhibitor comprises an oligonucleotide.

~~147. The method according to claim 145, wherein the ERAP1 inhibitor comprises a small molecule.~~

~~148. A method of identifying a subject having an increased risk for developing an MHC-I-opathy, the method comprising:~~

~~performing or having performed an assay on a biological sample from the subject to determine whether the subject comprises:~~

~~i) an MHC-I-opathy-related HLA genotype; and~~

~~ii) a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein;~~

~~wherein:~~

~~when the subject has both the MHC-I-opathy-related HLA genotype and the functional ERAP2 protein or the nucleic acid molecule encoding the functional ERAP2 protein, then the subject has an increased risk of developing the MHC-I-opathy;~~

~~when the subject lacks the MHC-I-opathy-related HLA genotype, or lacks the functional ERAP2 protein or the nucleic acid molecule encoding the functional ERAP2 protein, or lacks both, then the subject has a decreased risk of developing the MHC-I-opathy; and~~

~~when the subject comprises two copies of the MHC-I-opathy-related HLA genotype, then the subject has an increased risk of developing the MHC-I-opathy compared to comprising a single copy of the MHC-I-opathy-related HLA genotype.~~

~~149. The method according to claim 148, wherein the MHC-I-opathy is Birdshot Chorioretinopathy (BSCR) and the MHC-I-opathy-related HLA genotype comprises an HLA-Aw19 allele.~~

~~150. The method according to claim 149, wherein HLA-Aw19 allele is an HLA-A*29 allele, an HLA-A*30 allele, an HLA-A*31 allele, or an HLA-A*33 allele, or any combination thereof.~~

~~151. The method according to claim 149 or claim 150, wherein the subject has two copies of the HLA-Aw19 allele.~~

~~152. The method according to claim 151, wherein the subject is HLA-A*29$^+$/HLA-A*30$^+$.~~

~~153. The method according to claim 151, wherein the subject is HLA-A*29$^+$/HLA-A*31$^+$.~~

~~154. The method according to claim 151, wherein the subject is HLA-A*29$^+$/HLA-A*33$^+$.~~

~~155. The method according to any one of claims 149 to 154, the method further comprising administering an HLA-Aw19 inhibitor to the subject having an increased risk of developing the MHC-I-opathy.~~

~~156. The method according to claim 155, wherein the HLA-Aw19 inhibitor is an antibody.~~

~~157. The method according to claim 156, wherein the antibody is an anti-HLA-A*29 antibody.~~

~~158. The method according to claim 155, wherein the HLA-Aw19 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~159. The method according to claim 158, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-Aw19.~~

~~160. The method according to claim 159, wherein the HLA-Aw19 is HLA-A*29.~~

~~161. The method according to claim 148, wherein the MHC-I-opathy is Ankylosing Spondylitis (AS) and the MHC-I-opathy-related HLA genotype comprises an HLA-B*27 allele or an HLA-B*40 allele.~~

~~162. The method according to claim 161, wherein the subject has two copies of HLA-B*27 or HLA-B*40.~~

~~163. The method according to claim 161 or claim 162, the method further comprising administering an HLA-B*27 inhibitor or an HLA-B*40 inhibitor to the subject having an increased risk of developing the MHC-I-opathy.~~

~~164. The method according to claim 163, wherein the HLA-B*27 inhibitor or HLA-B*40 inhibitor is an antibody.~~

~~165. The method according to claim 164, wherein the antibody is an anti-HLA-B*27 antibody or an anti-HLA-B*40 antibody.~~

~~166. The method according to claim 163, wherein the HLA-B*27 inhibitor or HLA-B*40 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~167. The method according to claim 166, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-B*27 or an HLA-B*40.~~

~~168. The method according to claim 148, wherein the MHC-I-opathy is Behçet's disease and the MHC-I-opathy-related HLA genotype comprises an HLA-B*51 allele.~~

~~169. The method according to claim 168, wherein the subject has two copies of HLA-B*51.~~

~~170. The method according to claim 168 or claim 169, the method further comprising administering an HLA-B*51 inhibitor to the subject having an increased risk of developing the MHC-I-opathy.~~

~~171. The method according to claim 170, wherein the HLA-B*51 inhibitor is an antibody.~~

~~172. The method according to claim 170, wherein the antibody is an anti-HLA-B*51 antibody.~~

~~173. The method according to claim 170, wherein the HLA-B*51 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~174. The method according to claim 173, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-B*51.~~

~~175. The method according to claim 148, wherein the MHC-I-opathy is psoriasis and the MHC-I-opathy-related HLA genotype comprises an HLA-C*06 allele.~~

~~176. The method according to claim 175, wherein the subject has two copies of HLA-C*06.~~

~~177. The method according to claim 175 or claim 176, the method further comprising administering an HLA-C*06 inhibitor to the subject having an increased risk of developing the MHC-I-opathy.~~

~~178. The method according to claim 177, wherein the HLA-C*06 inhibitor is an antibody.~~

~~179. The method according to claim 178, wherein the antibody is an anti-HLA-C*06 antibody.~~

~~180. The method according to claim 177, wherein the HLA-C*06 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~181. The method according to claim 180, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-C*06.~~

~~182. The method according to claim 148, wherein the MHC-I-opathy is Juvenile Idiopathic Arthritis (JIA) and the MHC-I-opathy-related HLA genotype comprises an HLA-B*27.~~

~~183. The method according to claim 182, wherein the subject has two copies of HLA-B*27.~~

~~184. The method according to claim 182 or claim 183, the method further comprising administering an HLA-B*27 inhibitor to the subject having an increased risk of developing the MHC-I-opathy.~~

~~185. The method according to claim 184, wherein the HLA-B*27 inhibitor is an antibody.~~

~~186. The method according to claim 185, wherein the antibody is an anti-HLA-B*27 antibody.~~

~~187. The method according to claim 184, wherein the HLA-B*27 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~188. The method according to claim 187, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-B*27.~~

~~189. The method according to claim 148, wherein the MHC-I-opathy is inflammatory bowel disease (IBD) or Crohn's disease (CD) and the MHC-I-opathy-related HLA genotype comprises an HLA-C*07 allele.~~

~~190. The method according to claim 189, wherein the subject has two copies of HLA-C*07.~~

~~191. The method according to claim 189 or claim 190, the method further comprising administering an HLA-C*07 inhibitor to the subject having an increased risk of developing the MHC-I-opathy.~~

~~192. The method according to claim 191, wherein the HLA-C*07 inhibitor is an antibody.~~

~~193. The method according to claim 192, wherein the antibody is an anti-HLA-C*07 antibody.~~

~~194. The method according to claim 191, wherein the HLA-C*07 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~195. The method according to claim 194, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to an HLA-C*07.~~

~~196. The method according to any one of claims 148 to 195, wherein the nucleic acid molecule comprises genomic DNA, mRNA, or cDNA obtained from mRNA.~~

~~197. The method according to any one of claims 148 to 196, the method further comprising administering an ERAP2 inhibitor to the subject having an increased risk of developing the MHC I opathy.~~

~~198. The method according to claim 197, wherein the ERAP2 inhibitor comprises a small molecule degrader or an inhibitory nucleic acid molecule.~~

~~199. The method according to claim 198, wherein the inhibitory nucleic acid molecule is an antisense nucleic acid molecule, a small interfering RNA (siRNA), or a short hairpin RNA that hybridizes to ERAP2 mRNA.~~

~~200. The method according to claim 197, wherein the ERAP2 inhibitor comprises an anti-ERAP2 antibody.~~

~~201. The method according to claim 197, wherein the ERAP2 inhibitor comprises a pseudopeptide.~~

~~202. The method according to claim 201, wherein the pseudopeptide is a phosphinic pseudopeptide.~~

~~203. The method according to claim 202, wherein the phosphinic pseudopeptide is DG002 or DG013.~~

~~204. The method according to claim 197, wherein the ERAP2 inhibitor comprises a small molecule.~~

~~205. The method according to any one of claims 148 to 204, wherein:~~

~~the assay performed or having been performed on the biological sample from the subject further determines whether the subject comprises a functional Endoplasmic Reticulum Aminopeptidase 1 (ERAP1) protein or a nucleic acid molecule encoding a functional ERAP1 protein; and~~

~~when the subject has the MHC-I-opathy-related HLA genotype and lacks the functional ERAP1 protein or the nucleic acid molecule encoding the functional ERAP1 protein, then the subject has an increased risk of developing the MHC-I-opathy; and~~

~~when the subject lacks the MHC-I-opathy-related HLA genotype, or has the functional ERAP1 protein or the nucleic acid molecule encoding the functional ERAP1 protein, or both, then the subject has a decreased risk of developing the MHC-I-opathy.~~

~~206. The method according to claim 205, the method further comprising administering an ERAP1 agonist to the subject having an increased risk of developing the MHC-I-opathy.~~

~~207. The method according to claim 206, wherein the ERAP1 agonist comprises a comprises an oligonucleotide.~~

~~208. The method according to claim 207, wherein the oligonucleotide is ODN1826.~~

~~209. The method according to claim 206, wherein the ERAP1 agonist comprises a lipopeptide.~~

~~210. The method according to claim 209, wherein the lipopeptide is Pam3CSK4 or FSL-1.~~

~~211. The method according to claim 206, wherein the ERAP1 agonist comprises a small molecule.~~

~~212. The method according to any one of claims 148 to 204, wherein:~~

~~the assay performed or having been performed on the biological sample from the subject further determines whether the subject comprises a functional Endoplasmic Reticulum Aminopeptidase 1 (ERAP1) protein or a nucleic acid molecule encoding a functional ERAP1 protein; and~~

~~when the subject has the MHC-I-opathy-related HLA genotype and has the functional ERAP1 protein or the nucleic acid molecule encoding the functional ERAP1 protein, then the subject has an increased risk of developing the MHC-I-opathy; and~~

~~when the subject lacks the MHC-I-opathy-related HLA genotype, or does not comprise the functional ERAP1 protein or the nucleic acid molecule encoding the functional ERAP1 protein, or both, then the subject has a decreased risk of developing the MHC-I-opathy.~~

~~213. The method according to claim 212, the method further comprising administering an ERAP1 inhibitor to the subject having an increased risk of developing the MHC-I-opathy.~~

~~214. The method according to claim 213, wherein the ERAP1 inhibitor comprises an inhibitory nucleic acid molecule.~~

~~215. The method according to claim 213, wherein the ERAP1 inhibitor comprises a small molecule.~~

1. Use of an Endoplasmic Reticulum Aminopeptidase 2 (ERAP2) inhibitor in the manufacture of a medicament for treating a subject having an immune disorder.

2. The use according to claim 1, wherein the immune disorder is an MHC-I-opathy.

3. The use according to claim 2, wherein the MHC-I-opathy is Birdshot Chorioretinopathy (BSCR).

4. The use according to claim 3, wherein the presence or absence of an HLA-Aw19 allele has been further detected in a biological sample obtained from the subject.

5. The use according to claim 2, wherein the MHC-I-opathy is Ankylosing Spondylitis (AS).

6. The use according to claim 5, wherein the presence or absence of HLA-B*27 or HLA-B*40 has been further detected in a biological sample obtained from the subject.

7. The use according to claim 2, wherein the MHC-I-opathy is Behçet's disease.

8. The use according to claim 7, wherein the presence or absence of HLA-B*51 has been further detected in a biological sample obtained from the subject.

9. The use according to claim 2, wherein the MHC-I-opathy is psoriasis.

10. The use according to claim 9, the method further comprising detecting the presence or absence of HLA-C*06 in a biological sample obtained from the subject.

11. The use according to claim 2, wherein the MHC-I-opathy is Juvenile Idiopathic Arthritis (JIA).

12. The use according to claim 11, wherein the presence or absence of HLA-B*27 has been further detected in a biological sample obtained from the subject.

13. The use according to claim 2, wherein the MHC-I-opathy is inflammatory bowel disease (IBD) or Crohn's disease (CD).

14. The use according to claim 13, wherein the presence or absence of HLA-C*07 has been further detected in a biological sample obtained from the subject.

15. Use of an ERAP2 inhibitor in the manufacture of a medicament for treating a subject having an MHC-I-opathy, wherein an assay performed or having been performed on a biological sample from the subject determines whether the subject comprises:

i) an MHC-I-opathy-related HLA genotype; and

ii) a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein;

wherein the medicament is to be administered to the subject, wherein the subject comprises both an MHC-I-opathy-related HLA genotype and a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein; and

wherein the presence of both the MHC-I-opathy-related HLA genotype and the functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein indicates that the subject is a candidate for treating the MHC-I-opathy by inhibiting ERAP2.

16. The use according to claim 15, wherein the MHC-I-opathy is Birdshot Chorioretinopathy (BSCR) and the MHC-I-opathy-related HLA genotype comprises an HLA-Aw19 allele.

17. The use according to claim 15, wherein the MHC-I-opathy is Ankylosing Spondylitis (AS) and the MHC-I-opathy-related HLA genotype comprises an HLA-B*27 allele or an HLA-B*40 allele.

18. The use according to claim 15, wherein the MHC-I-opathy is Behçet's disease and the MHC-I-opathy-related HLA genotype comprises an HLA-B*51 allele.

19. The use according to claim 15, wherein the MHC-I-opathy is psoriasis and the MHC-I-opathy-related HLA genotype comprises an HLA-C*06 allele.

20. The use according to claim 15, wherein the MHC-I-opathy is Juvenile Idiopathic Arthritis (JIA) and the MHC-I-opathy-related HLA genotype comprises an HLA-B*27.

21. The use according to claim 15, wherein the MHC-I-opathy is inflammatory bowel disease (IBD) or Crohn's disease (CD) and the MHC-I-opathy-related HLA genotype comprises an HLA-C*07 allele.

22. The use according to claim 15, wherein:

the assay performed or having been performed on the biological sample from the subject further determines whether the subject comprises a functional Endoplasmic Reticulum Aminopeptidase 1 (ERAP1) protein or a nucleic acid molecule encoding a functional ERAP1 protein; and

wherein a therapeutically effective amount of an ERAP1 agonist is to be further administered to the subject, wherein the subject comprises an MHC-I-opathy-related HLA genotype and does not comprise a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein;

wherein the presence of an MHC-I-opathy-related HLA genotype and the absence of a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein indicates that the subject is a candidate for treating the MHC-I-opathy by activating ERAP1.

23. The use according to claim 15, wherein:

the assay performed or having been performed on the biological sample from the subject further determines whether the subject comprises a functional Endoplasmic Reticulum Aminopeptidase 1 (ERAP1) protein or a nucleic acid molecule encoding a functional ERAP1 protein; and

wherein a therapeutically effective amount of an ERAP1 inhibitor is to be further administered to the subject, wherein the subject comprises an MHC-I-opathy-related HLA genotype and comprises a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein;

wherein the presence of an MHC-I-opathy-related HLA genotype and the presence of a functional ERAP1 protein or a nucleic acid molecule encoding a functional ERAP1 protein indicates that the subject is a candidate for treating the MHC-I-opathy by inhibiting ERAP1.

24. A method of identifying a subject having an increased risk for developing an MHC-I-opathy, the method comprising:

performing or having performed an assay on a biological sample from the subject to determine whether the subject comprises:

i) an MHC-I-opathy-related HLA genotype; and

ii) a functional ERAP2 protein or a nucleic acid molecule encoding a functional ERAP2 protein;

wherein:

when the subject has both the MHC-I-opathy-related HLA genotype and the functional ERAP2 protein or the nucleic acid molecule encoding the functional ERAP2 protein, then the subject has an increased risk of developing the MHC-I-opathy;

when the subject lacks the MHC-I-opathy-related HLA genotype, or lacks the functional ERAP2 protein or the nucleic acid molecule encoding the functional ERAP2 protein, or lacks both, then the subject has a decreased risk of developing the MHC-I-opathy; and

when the subject comprises two copies of the MHC-I-opathy-related HLA genotype, then the subject has an increased risk of developing the MHC-I-opathy compared to comprising a single copy of the MHC-I-opathy-related HLA genotype.

25. The method according to claim 24, wherein the MHC-I-opathy is Birdshot Chorioretinopathy (BSCR) and the MHC-I-opathy-related HLA genotype comprises an HLA-Aw19 allele.

26. The method according to claim 24, wherein the MHC-I-opathy is Ankylosing Spondylitis (AS) and the MHC-I-opathy-related HLA genotype comprises an HLA-B*27 allele or an HLA-B*40 allele.

27. The method according to claim 24, wherein the MHC-I-opathy is Behçet's disease and the MHC-I-opathy-related HLA genotype comprises an HLA-B*51 allele.

28. The method according to claim 24, wherein the MHC-I-opathy is psoriasis and the MHC-I-opathy-related HLA genotype comprises an HLA-C*06 allele.

29. The method according to claim 24, wherein the MHC-I-opathy is Juvenile Idiopathic Arthritis (JIA) and the MHC-I-opathy-related HLA genotype comprises an HLA-B*27.

30. The method according to claim 24, wherein the MHC-I-opathy is inflammatory bowel disease (IBD) or Crohn's disease (CD) and the MHC-I-opathy-related HLA genotype comprises an HLA-C*07 allele.

31. The method according to claim 24, wherein:

the assay performed or having been performed on the biological sample from the subject further determines whether the subject comprises a functional Endoplasmic Reticulum Aminopeptidase 1 (ERAP1) protein or a nucleic acid molecule encoding a functional ERAP1 protein; and

when the subject has the MHC-I-opathy-related HLA genotype and lacks the functional ERAP1 protein or the nucleic acid molecule encoding the functional ERAP1 protein, then the subject has an increased risk of developing the MHC-I-opathy; and

when the subject lacks the MHC-I-opathy-related HLA genotype, or has the functional ERAP1 protein or the nucleic acid molecule encoding the functional ERAP1 protein, or both, then the subject has a decreased risk of developing the MHC-I-opathy.

32. The method according to claim 24, wherein:

the assay performed or having been performed on the biological sample from the subject further determines whether the subject comprises a functional Endoplasmic Reticulum Aminopeptidase 1 (ERAP1) protein or a nucleic acid molecule encoding a functional ERAP1 protein; and

when the subject has the MHC-I-opathy-related HLA genotype and has the functional ERAP1 protein or the nucleic acid molecule encoding the functional ERAP1 protein, then the subject has an increased risk of developing the MHC-I-opathy; and

when the subject lacks the MHC-I-opathy-related HLA genotype, or does not comprise the functional ERAP1 protein or the nucleic acid molecule encoding the functional ERAP1 protein, or both, then the subject has a decreased risk of developing the MHC-I-opathy.