



Research Article

Open Access

Predicted high affinity binding of prion PRP^C protein to Human Leukocyte Antigen (HLA)

Apostolos P. Georgopoulos^{1,2,3*}, Lisa M. James^{1,2,4}, Matthew Sanders^{1,2,3}

¹The HLA and Chronic Diseases Research Groups, Brain Sciences Center, Department of Veterans Affairs Health Care System, Minneapolis, Minnesota, USA.

²Department of Neuroscience, University of Minnesota Medical School, Minneapolis, Minnesota, USA.

³Institute for Health Informatics, University of Minnesota Medical School, Minneapolis, Minnesota, USA.

⁴Department of Psychiatry, University of Minnesota Medical School, Minneapolis, Minnesota, USA.

Article Info

Article Notes

Received: February 5th, 2026

Accepted: March 20th, 2026

*Correspondence:

*Dr. Apostolos P. Georgopoulos. Brain Sciences Center (11B), Minneapolis VAHCS, One Veterans Drive, Minneapolis, MN 55417, USA; Email: omega@umn.edu.

© 2026 Georgopoulos AP et al. This article is distributed under the terms of the Creative Commons Attribution 4.0 International License.

Keywords

Prion PRP^C protein

Human Leukocyte Antigen (HLA)

Autoimmunity

Binding affinity

ABSTRACT

Misfolding of the cellular prion protein (PRP^C) is associated with fatal neurodegenerative prion diseases for which no treatments are currently available. Although the immune system is generally non-responsive (tolerant) to self-proteins such as PRP^C, evidence of anti-prion antibodies suggests escape from self-tolerance in some individuals and supports the potential for the human immune system to be leveraged against prion disease. Human leukocyte antigen (HLA) plays a central role in rejecting endogenous non-self proteins (e.g. cancer neoantigens) by activating CD8+ cytolytic T cells via the Class I system (HLA-I) and CD4+ helper T cells via the Class II (HLA-II) system. Here we investigated the predicted binding affinity of 334 HLA molecules with all possible linear 9-mer (for HLA-I) and 15-, 18- and 22-mer (for HLA-II) PRP^C peptides to identify peptide-HLA (pHLA) complexes with strong predicted binding ($IC_{50} < 50$ nM). We found that 12.4% of all prion peptides tested showed strong binding affinity to HLA molecules and that 20.2% of HLA alleles were able to bind strongly with PRP^C peptides. These findings suggest that carriers of certain HLA alleles that are capable of binding strongly to PRP^C peptides may have enhanced protection against prion disease, through reduction in the overall amount of PRP^C available for conversion to the misfolded, infectious scrapie isoform (PRP^{Sc}) of PRP^C and, potentially, by destroying it. These findings have implications for other disorders including common neurodegenerative diseases characterized by protein misfolding (e.g. α -synuclein, huntingtin, amyloid, tau, etc.).

Introduction

Prion disease is an infectious, fatal neurodegenerative disease with global prevalence and no known treatment¹⁻⁸. It is due to the posttranslational misfolding of the cellular prion protein (PRP^C), a common, naturally occurring protein. PrP^C is conserved across species and is found in numerous tissues throughout the brain and periphery⁹, including red blood cells¹⁰, platelets¹¹ and CD8+/CD4+ T lymphocytes¹². While PrP^C has been implicated in several physiological functions of the nervous and immune systems, the specific roles of PrP^C are unclear and possibly redundant with other proteins as evidenced by contradictory and/or null findings from PrP^C knock-out studies^{9,14}. What is clear is that misfolding of PrP^C due to genetic mutations in the human major prion protein (PRNP) gene or various conditions within cells is associated with fatal neurodegenerative disorders resulting from accumulation and propagation of infectious prion protein (PrP^{Sc}) in the brain^{1,15}. At this time, there are no treatments available for prion diseases although several strategies are actively being investigated including vaccines and other immunotherapies^{16,17}.

Relative to other infectious agents such as viruses and bacteria, prions pose unique challenges in terms of natural immunity, immunotherapy, and vaccine development. The human immune system is well-equipped to monitor for, and mount a response against, non-self antigens, be them endogenous (e.g. cancer neoantigens) or exogenous (e.g. viral proteins), while sparing self-antigens (tolerance), processes for which Human Leukocyte Antigen (HLA) is critical. However, despite conformational differences, the amino acid sequence of both PrP^C and PrP^{Sc} are identical; thus, activation of immune system responses targeting infectious agents is hampered by immune tolerance to self-proteins (PrP^C in this case), a challenge not only for natural immunity but also for development of prophylactic or treatment approaches for prion diseases^{16,17}. Nonetheless, antibodies against PrP^C have been documented in humans without signs of prion pathology, indicating escape from self-tolerance and innocuous anti-PrP^C autoimmunity in some cases^{18,19}. Moreover, several neuroprotective PrP^C-binding antibody fragments have been identified from human antibody repertoires suggesting that anti-PrP^C antibodies exist in the population, contrary to expectations related to self-tolerance¹⁸. Overcoming self-tolerance via antigen selection and optimization may be a promising and plausible avenue for developing therapeutic strategies against prion diseases¹⁶. Since PrP^{Sc} requires PrP^C for propagation, reduction of PrP^C via antibodies is an appealing strategy¹⁸.

HLA molecules are cell-surface glycoproteins that work in concert to eliminate non-self proteins, including, e.g. proteins of pathogens (e.g. viral/bacterial) or neoantigens of cancer²⁰. HLA molecules belong to two major classes, Class I (HLA-I) and Class II (HLA-II). Both HLA-I and HLA-II molecules are cell surface glycoproteins that present protein peptides to T cells. With respect to HLA Class I system, HLA-I molecules (encoded by the classical A, B, C genes) are expressed in all nucleated cells and produce molecules that bind with high affinity short peptides (mostly 9-mer) generated by the degradation of mostly endogenous proteins in the proteasome. The stable peptide-HLA-I complex (pHLA-I) moves to the cell surface where it is presented to circulating CD8+ T cells. CD8+ T cells that recognize the specific pHLA complex are activated and destroy cells that contain the non-self protein via various mechanisms, hence their direct cytotoxicity. With respect to HLA Class II system, HLA-II molecules (encoded by the classical DPB1, DQB1 and DRB1 genes) are expressed in specialized antigen presenting cells (e.g. macrophages, dendritic cells) and bind with high affinity longer peptides (mostly 15-mer) generated by the degradation of mostly exogenous proteins in the endo-lysosome compartment. The stable pHLA-II complex moves to the cell surface where it is presented to circulating CD4+ T cells which engage the B cells for production of antibodies against the offending protein but also enhance the activation of CD8+ T cells and

also possess cytotoxic properties themselves. The cross-presentation pathway allows for processing of endogenous and exogenous antigens by both HLA-I and HLA-II systems. The HLA region is the most polymorphic in the human genome²¹; consequently, there is tremendous variability in HLA composition across individuals. The HLA composition of each individual determines the repertoire of antigens that can bind with sufficient affinity to promote an immune response²²⁻²⁵. Although the large HLA polymorphism almost guarantees survival at the population level, each individual carries only 12 HLA alleles, 2 per classical genes of HLA-I and HLA-II, which means that the success of the individual in dealing with/eliminating infectious and non-self antigens will be restricted, depending on the individual's HLA genetic makeup.

Rejecting non-self proteins presupposes that those can be distinguished from self proteins. The mechanisms by which self proteins are recognized as such and are not attacked by the immune system ("immune tolerance") are fairly complex and incompletely understood²⁶. It is widely believed that escape from immune tolerance is a major contributing factor to autoimmune disorders^{27,28}, with a recent estimated prevalence of 4.6% in the United States²⁹. The HLA immunogenetic makeup is the main genetic factor underlying escape from immune tolerance³⁰. Although it is commonly assumed that escape from immune tolerance, i.e. attacking self-proteins, is detrimental to health, this need not be universally true, since the health outcome would depend on the self protein being attacked. Naturally, we assume that all naturally occurring proteins in the body are "good", and that is correct assuming the protein in question stays in its original configuration. The case in point is PRP^C: in its natural form it is useful and innocuous but when (for ill-understood reasons) it misfolds, it transforms into the infectious and deadly PRP^{Sc}. In this case, escape from immune tolerance against PRP^C could be beneficial, as it would reduce the number of PRP^C available for converting to PRP^{Sc}. Interestingly, although PRP^C is widely expressed in many tissues, its absence in knockout mice lacking the PRNP gene has not been associated with serious health issues⁹. Escape of PRP^C from immune tolerance has been indicated by the reported existence of anti-PrP^C antibodies¹⁸, for which the HLA-II system would be involved. To our knowledge, there has been no systematic evaluation of HLA-related escape of PRP^C from immune tolerance. Here, we assessed *in silico* the predicted binding affinity of 334 HLA molecules (142 HLA-I and 192 HLA-II) with PrP^C peptides to search for and identify those capable of strong pHLA binding. Such molecules would underlie the hypothetical escape of PRP^C from immune tolerance, leading to reduction of PRP^C numbers directly (via CD8+ T cell activation, enhanced by CD4+ T cell activation) and/or indirectly (via CD4+ T cell activation of B cells for the production of anti-PRP^C antibodies).

Materials and Methods

Human prion protein (PRP^C)

The amino acid (AA) sequence of the human major prion protein (PRP gene) was retrieved from the Uniprot database (<https://www.uniprot.org/>) on September 10, 2025 and is given in Table 1.

HLA alleles

We investigated 142 HLA-I and 192 HLA-II common alleles³¹ shown in Tables S2 and S3, respectively.

In silico determination of Predicted Binding Affinities PRP^C

Predicted binding affinities were obtained for antigen peptides using the Immune Epitope Database (IEDB) NetMHCpan (ver. 4.1) tool^{32,33}; accessed on September 12, 2025. More specifically, we used the sliding window approach³⁴⁻³⁶ to test exhaustively all possible linear 9-mer peptides for HLA-I predictions and 15-, 18- 22-mer peptides for HLA-II predictions. The method is illustrated in Fig. 1 for 9-mer and 15-mer peptides of PRP^C. For each pair of peptide-HLA molecule tested, this tool gave, as an output, the IC₅₀ of the predicted binding affinity; *the smaller the IC₅₀, the stronger the binding affinity*. An IC₅₀ value of < 50 nM (nanomolar) was regarded strong and 50 nM < IC₅₀ < 500 nM values were regarded moderate³⁷. Given a protein of N amino acid length and a peptide length of k

AA, there are N-k+1 binding affinity predictions returned by the prediction tool. The numbers of 9-, 15-, 18-, and 22-mer peptides tested are given in Table 2.

Statistical analyses

The IBM-SPSS statistical package (version 30.0.0.0 172) was used for implementing statistical analyses. Standard statistical methods were used; all correlations are Pearson. All P-values reported are 2-sided, $\alpha = 0.05$.

Results

Predicted binding affinities

We investigated 142 HLA-I alleles (41 HLA-A, 29 HLA-B, 72 HLA-C) (Table S1) and 192 HLA-II alleles (41 HLA-DPB1, 35 HLA-DQB1, 116 HLA-DRB1) (Table S2). The numbers of peptide-HLA allele complexes (pHLA) tested are shown in Table 2, together with the numbers and percentages of predicted strong and moderate binding affinities of 9-, 15-, 18-, and 22-mer peptides to HLA molecules. As expected, overall strong affinities were observed less frequently than moderate ones (0.369% vs. 4.305%). Details of pHLA complexes binding with high affinity are given in Tables S3-S6.

Peptides

Of the total 952 PRP^C peptides tested (Table 2), 116 (12.18%) distinct peptides showed strong predicted

Table 1. Amino acid sequence of PRP^C protein.

UniProt ID: P04156	Human major prion protein PRP ^C (PRNP gene)	253 AA
MANLGCWMLVLFVATWSDLGLCKKRPKPGGWNTGGSRYPGQGSPGGNRYPPQGGGGWGQPHGGGGWGQPHGGGGWGQPHGGGGWGQPHGGGGWGQGGGTDSQWNKPSKPKTNMKHMAGAAAAGAVVGGGLGGYMLGSAMSRPIIFGSDYEDRYRENMHRYPNQVYRPMDEYSNQNNFVHDCVNITIKQHTVTTTTKGENFTETDVKMMMERVVEQMCITQYERESQAYYQRGSSMVLFSPPVILLISFLIPLIVG		

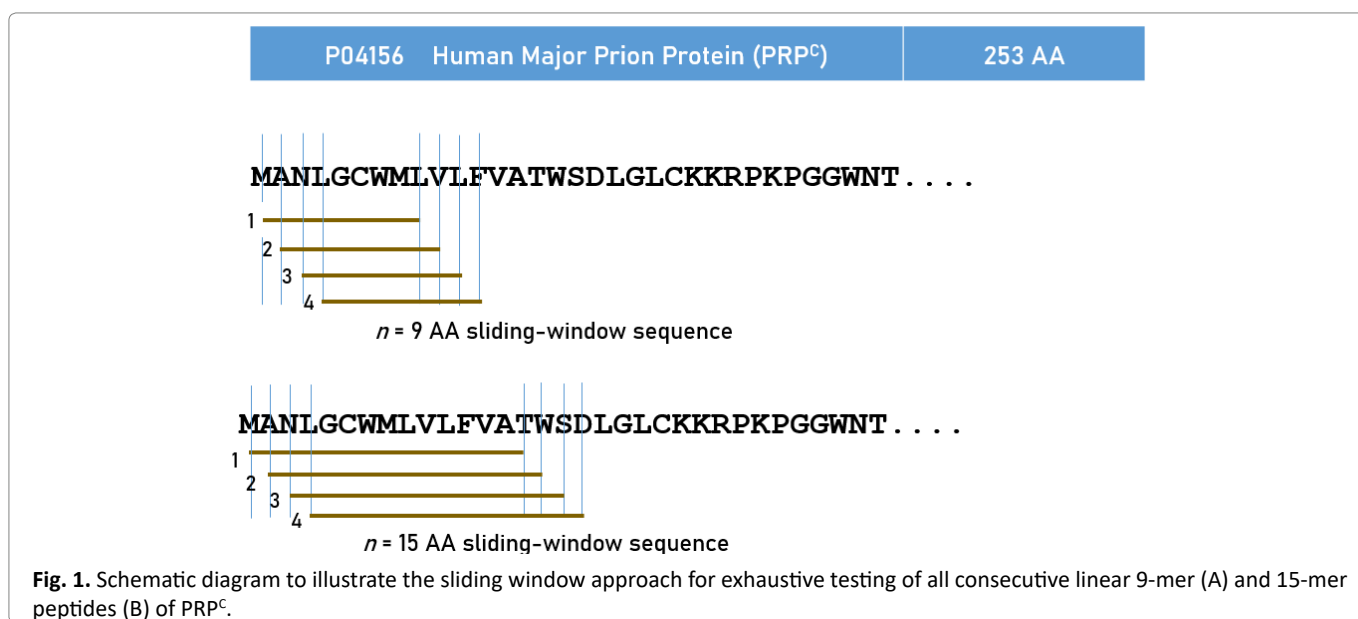


Table 2. Counts (and percentages) of pHLA tested and their predicted binding affinities.

HLA	N Tested		Total	N (%) Predicted binding affinity	
	Peptides	Alleles		Strong $IC_{50} < 50$ nM	Moderate 50 nM $\leq IC_{50} < 500$ nM
HLA-I (9-mer)	245	142	34,790	73 (0.210%)	295 (0.848%)
HLA-II (15-mer)	239	192	45,888	315 (0.686)	2966 (6.464)
HLA-II (18-mer)	236	192	45,312	26 (0.057)	895 (1.975)
HLA-II (22-mer)	232	192	44,544	215 (0.483)	3186 (7.152)
Total	952		170,534	629 (0.369)	7342 (4.305)

Table 3. The 116 unique PRP^c peptides binding with high affinity ($IC_{50} < 50$ nM) to HLA-I and HLA-II molecules.

#	9-mer	#	15-mer	#	18-mer	#	22-mer
1	MVLFSSPPV	1	AAGAVVGGGLGGYMLG	1	GSSMVLFSPPVILLISF	1	AAGAVVGGGLGGYMLGSAMSRPI
2	LLISFLIFL	2	AGAVVGGGLGGYMLGS	2	KPKTNMKHMAGAAAAGAV	2	AGAVVGGGLGGYMLGSAMSRPII
3	LISFLIFLI	3	AVVGGGLGGYMLGSAM	3	KTNMKHMAGAAAAGAVVG	3	AVVGGGLGGYMLGSAMSRPIIHF
4	VLFSPPVI	4	AYYQRGSSMVLFSPP	4	PKTNMKHMAGAAAAGAVV	4	AYYQRGSSMVLFSPPVILLIS
5	YYQRGSSMV	5	ESQAYYQRGSSMVLFS	5	QRGSSMVLFSPPVILLI	5	DEYSNQNNFVHDCVNITIKQHT
6	AYYQRGSSM	6	GAVVGGGLGGYMLGSA	6	RGSSMVLFSPPVILLIS	6	ERESQAYYQRGSSMVLFSPPV
7	AVVGGGLGGY	7	GGLGGYMLGSAMSRP	7	SKPKTNMKHMAGAAAAGA	7	ESQAYYQRGSSMVLFSPPVIL
8	YYRENMHRY	8	GGYMLGSAMSRPIIH	8	SSMVLFSPPVILLISFL	8	EYSNQNNFVHDCVNITIKQHTV
9	MSRPIIHF	9	GLGGYMLGSAMSRPI	9	YQRGSSMVLFSPPVILL	9	GAVVGGGLGGYMLGSAMSRPIIH
10	KTNMKHMAG	10	GSSMVLFSPPVILL	10	YYQRGSSMVLFSPPVIL	10	GGLGGYMLGSAMSRPIIHFSGD
11	HSQWNKPSK	11	GYMLGSAMSRPIIHF			11	GLGGYMLGSAMSRPIIHFSGDY
12	RYRENMHR	12	KPKTNMKHMAGAAAA			12	GSSMVLFSPPVILLISFLIFL
13	RYPNQVYYR	13	KTNMKHMAGAAAAGA			13	KPKTNMKHMAGAAAAGAVVGG
14	QMCITQYER	14	LGGYMLGSAMSRPII			14	KPSKPKTNMKHMAGAAAAGAVV
15	KPSKPKTNM	15	MKHMAGAAAAGAVVG			15	KTNMKHMAGAAAAGAVVGG
16	YQRGSSMVL	16	MLGSAMSRPIIHFSG			16	LGGYMLGSAMSRPIIHFSGDYE
17	AMSRPIIHF	17	MVLFSSPPVILLISF			17	MDEYSNQNNFVHDCVNITIKQH
18	MHRYPNQVY	18	NMKHMAGAAAAGAVV			18	NKPSKPKTNMKHMAGAAAAGAV
19	YERESQAYY	19	NNFVHDCVNITIKQH			19	NMKHMAGAAAAGAVVGG
20	HRYPNQVYY	20	NQNNFVHDCVNITIK			20	PKTNMKHMAGAAAAGAVVGG
21	MKHMAGAAA	21	PKTNMKHMAGAAAAG			21	PMDEYSNQNNFVHDCVNITIKQ
22	ILLISFLIF	22	QAYYQRGSSMVLFS			22	PSKPKTNMKHMAGAAAAGAVV
23	FSSPPVILL	23	QNNFVHDCVNITIKQ			23	QAYYQRGSSMVLFSPPVILLI
24	DEYSNQNNF	24	QRGSSMVLFSPPVI			24	QRGSSMVLFSPPVILLISFLI
25	VYYRPMDEY	25	RESQAYYQRGSSMVL			25	QWNKPSKPKTNMKHMAGAAAAG
26	LFVATWSDL	26	RGSSMVLFSPPVIL			26	QYERESQAYYQRGSSMVLFSPP
27	LFSSPPVIL	27	SKPKTNMKHMAGAAA			27	RESQAYYQRGSSMVLFSPPVI
		28	SMVLFSPPVILLIS			28	RGSSMVLFSPPVILLISFLIF
		29	SNQNNFVHDCVNITI			29	SKPKTNMKHMAGAAAAGAVVGG
		30	SQAYYQRGSSMVLFS			30	SNQNNFVHDCVNITIKQHTVTT
		31	SSMVLFSPPVILLI			31	SQAYYQRGSSMVLFSPPVILL
		32	TNMKHMAGAAAAGAV			32	SQWNKPSKPKTNMKHMAGAAA
		33	VGGGLGGYMLGSAMSR			33	SSMVLFSPPVILLISFLIFLI
		34	VLFSPPVILLISFL			34	TNMKHMAGAAAAGAVVGG
		35	YMLGSAMSRPIIHF			35	TQYERESQAYYQRGSSMVLFS
		36	YQRGSSMVLFSPPV			36	VGGGLGGYMLGSAMSRPIIHF
		37	YYQRGSSMVLFSPP			37	VGGGLGGYMLGSAMSRPIIHF
						38	WNKPSKPKTNMKHMAGAAAAGA
						39	YERESQAYYQRGSSMVLFSPP
						40	YQRGSSMVLFSPPVILLISFL
						41	YSNQNNFVHDCVNITIKQHTVT
						42	YYQRGSSMVLFSPPVILLISF

binding affinity ($IC_{50} < 50$ nM) to HLA molecules and are shown in Table 3, amounting to a total of 629 peptides

(given that they bound to more than one HLA molecule). The location of these peptides identified to have strong

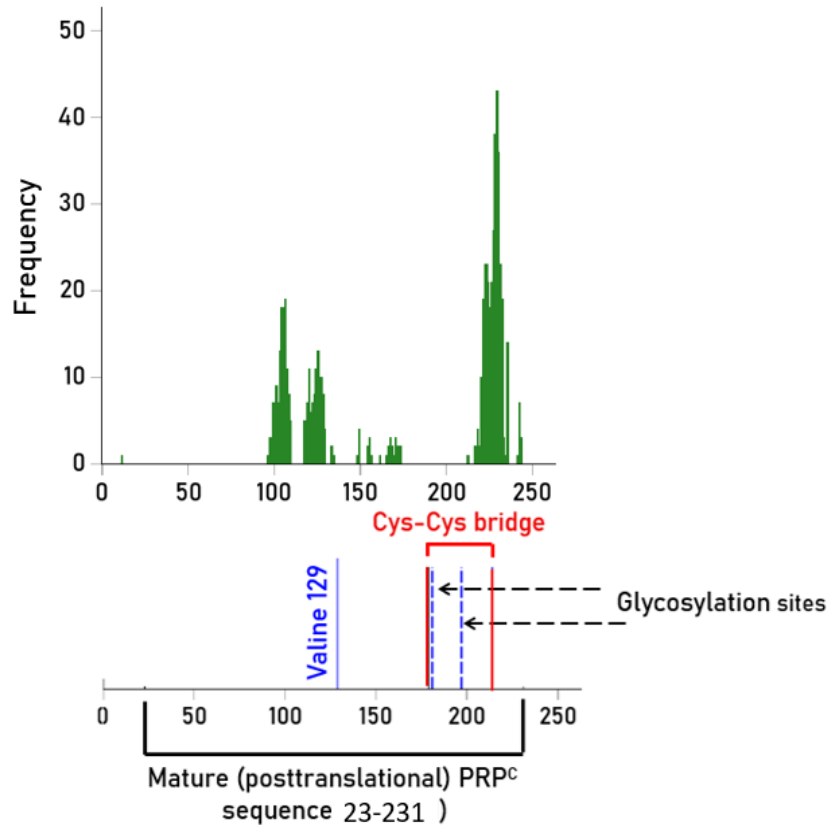


Fig. 2. Bar graph shows the locations of the start of peptide sequences with predicted high binding affinity to all HLA molecules along the PRP^C. N = 629 peptide locations from Tables S3-S6.

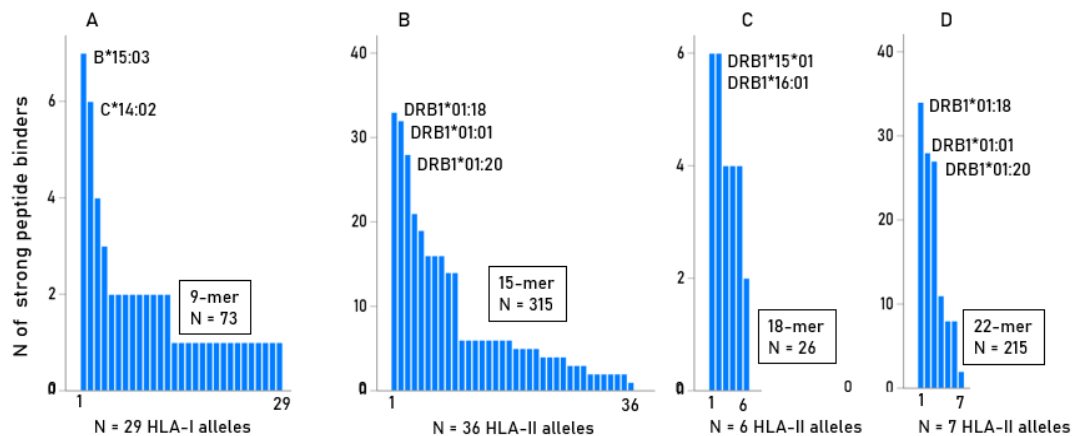


Fig. 3. The counts of strongly binding peptides are plotted against alleles in a decreasing order. A, 9-mer peptides, HLA-I alleles; B, 15-mer peptides, HLA-II alleles; C, 18-mer peptides, HLA-II alleles; D, 22-mer peptides, HLA-II alleles. The bars in the X-axis indicate alleles in Table 2 plotted in the same order.

predicted bindings to any of the HLA tested (Tables S3-S6) is shown in Fig. 2. We found that 271/629 (43.1%) peptides were located within the mature (posttranslational) PRP^C (residues 23-231), arranged in 3 clusters. Overall, as can be seen more precisely in Tables S3-S6, several peptides contained the protective residue 129 (valine), glycosylation residues 181 and 197, and stabilizing Cys-Cys bridge residues 179 and 214. Antibodies against those

peptides could potentially destabilize PRP^C making it prone to misfolding, an issue we discuss further in the Discussion section below.

Alleles

With respect to HLA-I, 29 (20.4%) alleles of the 142 tested, showed strong binding to at least one of the 245 peptides (9-mer) tested (range 1-7; Table 2; Fig.3A)

and they were spread across all 3 HLA-I genes (A, B, C). In contrast, all strongly binding HLA-II molecules were confined to the DRB1 gene. More specifically, of the 192 alleles tested, (a) 36 (18.7%) showed strong binding to at least one of the 239 15-mer peptides tested (range 1-33; Table 2, Fig. 3B), (b) 6 (3.1%) showed strong binding to at least one of the 236 18-mer peptides tested (range 2-6; Table 2, Fig. 3C), and (c) 7 (3.6%) showed strong binding to at least one of the 239 22-mer peptides tested (range 2-34; Table 2, Fig. 3D). These results show that 15-mer peptides were the most effective HLA-II binders regarding both the total number of strong binders (N = 315) and spread among 36 alleles, whereas 18-mer peptides were the least effective (N = 26 strong binders among 6 alleles). Interestingly, 22-mer peptides were also effective binders (N = 119) but were spread only across 7 alleles. Since PRP^C is a natural, host protein, the presence of strong binding in all peptide lengths tested (9, 15, 18, 22-mer), although at different proportions, indicate evasion of tolerance during thymic selection.

Overlap with other human proteins

We tested for possible overlap of the 116 peptides above to other human proteins by comparing them against the human proteome dataset version 24.1, provided by The Human Protein Atlas [The Human Protein Atlas. Accessed on October 22, 2025. https://www.proteinatlas.org/about/download#protein_atlas_data], comprising a total of 83607 human proteins.] They occurred only in 2 variants of the canonical PRP^C human protein (UNIPROT accession number P04156; 253 AA), namely truncated P04156 human prion proteins with accession numbers A2A2V1 (249 AA) and X6RKS3 (217 AA) with assumed similar function. Hence, involvement of HLA-II (15-mer, CD4+, antibody production) is restricted to PRP^C proteins only.

Discussion

Misfolding of PRP^C to infectious PRP^{Sc} results in fatal neurodegeneration due to the accumulation of PrP^{Sc} and lack of available treatment. It has been recognized that reduction of PrP^C via antibodies is an appealing strategy as it would reduce the number of PrP^C molecules available to convert to PrP^{Sc}^{17,18}. Although self-tolerance to PrP^C may limit antibody production, recent reports suggest that, for some individuals, natural immune system responses overcome self-tolerance as evidenced by detection of PrP^C autoantibodies in the general population in the absence of any disease-specific association^{18,19}. HLA is instrumental in the production of antibodies (via the CD4+ T cells of the HLA-II system) and for attacking and eliminating non-self proteins by direct destruction (via the CD8+ T cells of the HLA-I system, aided by the CD4+ helper T cells). With respect to HLA and autoimmunity, escape from immune

tolerance can involve either or both of the HLA (Class I and II) systems. Here we evaluated both systems. The overall percentage of predicted high affinity binding was below 1% for all tests (Table 2), in keeping with similar HLA-I estimates for the whole human proteome³⁷. Strongly binding HLA molecules were observed in 41/142 (28.9%) HLA-I alleles tested and occurred in all 3 genes (A, B, C; Table 4). In contrast, there were 36/192 (18.7%) strong HLA-II binders, all from the DRB1 gene. Since strong HLA binding would result in destruction of PRP^C, our findings suggest that carriers of certain HLA alleles that bind strongly to PrP^C may have enhanced protection against prion disease, reflecting “good” autoimmunity in the sense that strong HLA-peptide binding affinity may reduce the number of potential PRP^C misfoldings by reducing the number of available PRP^C molecules available. In contrast, individuals lacking HLA molecules capable of strong PrP^C peptide binding may be at greater risk of developing prion disease due to reduced ability to mount an immune response aimed at PrP^C elimination. It is noteworthy that PRP^C is cleaved in the proteasome³⁸ and, therefore, its 9-mer peptides can be presented to HLA-I alleles. Proteasome activity is inhibited by PRP^{Sc}³⁸, contributing to PRP^{Sc} accumulation in the cell. In addition, PRP^C and PRP^{Sc} are degraded in the lysosome³⁹, hence providing longer peptides (15-mer) for presentation to HLA-II molecules. Therefore, both HLA-I and HLA-II classes would contribute to limiting the number of PRP^C molecules available for misfolding. In addition, HLA-II molecules could be directly involved in lowering the PRP^{Sc} numbers via (a) reduction of PRP^C supply directly (via CD8+ T cell activation, enhanced by CD4+ T cell activation) and/or indirectly (via CD4+ T cell activation of B cells for the production of anti-PRP^C antibodies), and (b) destruction of PRP^{Sc} via CD4+ activation.

The current findings extend beyond natural immunity and point to specific peptides that may be useful for vaccine development (Table 3). Identification of immunogenic PrP^C peptides is an active line of research in pursuit of vaccines for prion diseases¹⁶. It is worth pointing out that immunogenicity of PrP^C peptides depends on HLA binding. Given the extreme heterogeneity of HLA²¹ and the effect of single amino acid differences on binding affinity²³, the immunogenicity of a given PrP^C peptide is specific to a given HLA molecule. Each individual possesses a limited repertoire of HLA alleles that code for cell-surface HLA molecules. Here, we identified specific PrP^C peptide sequences that are predicted to bind strongly to a given a specific HLA molecule and could be considered for vaccine development. That being said, the translational potential of these findings rests on experimental validation including in vitro validation of binding assays, antigen presentation, and engagement of CD8+/CD4+ T cells and B-cells as well as in vivo validation in animal models and human epidemiological data. Such validation studies are

Table 4. Counts (N) of peptides with strong binding affinity ($IC_{50} < 50$ nM) to the listed HLA-I and HLA-II alleles (ranked from high to low counts).

9-mer	15-mer	18-mer	22-mer				
Allele	N strong	Allele	N strong	Allele	N strong	Allele	N strong
B*15:03	7	DRB1*01:18	33	DRB1*15:01	6	DRB1*01:18	34
C*14:02	6	DRB1*01:01	32	DRB1*15:06	6	DRB1*01:01	28
A*02:35	4	DRB1*01:20	28	DRB1*01:01	4	DRB1*01:20	27
A*30:01	3	DRB1*01:29	21	DRB1*01:18	4	DRB1*01:24	11
A*31:01	3	DRB1*01:24	19	DRB1*01:20	4	DRB1*01:02	8
C*07:02	3	DRB1*01:11	16	DRB1*15:02	2	DRB1*01:29	8
A*02:01	2	DRB1*07:01	16	Total	26	DRB1*16:02	2
A*02:02	2	DRB1*09:01	16			Total	118
A*02:06	2	DRB1*01:02	14				
A*02:30	2	DRB1*10:01	14				
A*02:63	2	DRB1*11:14	6				
A*02:77	2	DRB1*13:02	6				
A*24:03	2	DRB1*13:23	6				
B*15:01	2	DRB1*13:97	6				
B*18:01	2	DRB1*15:01	6				
C*03:02	2	DRB1*15:06	6				
C*03:03	2	DRB1*15:07	6				
C*03:04	2	DRB1*16:02	6				
A*02:05	1	DRB1*15:02	5				
A*26:01	1	DRB1*15:03	5				
A*26:08	1	DRB1*15:15	5				
A*29:01	1	DRB1*15:37	5				
A*29:02	1	DRB1*04:01	4				
A*30:02	1	DRB1*04:72	4				
A*68:02	1	DRB1*13:96	4				
B*07:02	1	DRB1*14:32	4				
B*07:05	1	DRB1*04:10	3				
B*15:17	1	DRB1*16:05	3				
B*15:18	1	DRB1*16:09	3				
B*39:02	1	DRB1*04:04	2				
C*07:01	1	DRB1*11:02	2				
C*12:03	1	DRB1*11:65	2				
C*15:02	1	DRB1*13:01	2				
C*15:04	1	DRB1*14:01	2				
C*15:05	1	DRB1*14:54	2				
C*15:06	1	DRB1*16:01	1				
C*15:09	1	Total	315				
C*16:01	1						
C*16:02	1						
C*16:04	1						
C*17:01	1						
Total	73						

particularly important in light of potential model error of *in silico* predictions, the possibility of destabilizing effects of antibody binding to glycosylation sites^{40,41} and cysteine bridge residues^{42,43}, and evidence of potential neurotoxicity resulting from the interaction of antibodies with specific domains of PrP^{C44}.

Finally, the findings here may hold relevance for other neurodegenerative conditions. Prion-like misfolding

and aggregation of proteins including amyloid- β , tau, α -synuclein, and superoxide dismutase 1 have been implicated in the pathophysiology associated with Alzheimer's disease, tauopathies, Parkinson's disease, and amyotrophic lateral sclerosis, respectively⁴⁵⁻⁴⁷. HLA has also been implicated in risk/protection associated with various neurodegenerative diseases^{48,49}. Based on the current findings, it is possible that documented HLA-related protection is partially related to HLA-mediated

binding and elimination of misfolded proteins before they accumulate and lead to disease, a hypothesis that remains to be investigated.

Author Contributions

A.P.G. conceived the study and retrieved the PRP^c AA sequence; M.S. performed HLA predicted affinity and peptide-protein determinations; A.P.G. performed data analysis; L.M.J. and A.P.G. wrote the paper. All authors edited and approved the paper.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: Partial funding for this study was provided by the University of Minnesota (the American Legion Brain Sciences Chair and the Kunin Chair in Women's Healthy Brain Aging) and the U.S. Department of Veterans Affairs. The sponsors had no role in the current study design, analysis or interpretation, or in the writing of this paper. The contents do not represent the views of the U.S. Department of Veterans Affairs or the United States Government.

Ethical Approval

This article does not contain any studies with human participants performed by any of the authors.

Data Availability

All data used were retrieved from freely accessible websites and, as such, are publicly and freely available [ref. [33]: <http://tools.iedb.org/mhci/>].

References

1. Bellini P, Ruggiero F, Benedetti A, et al. Human prion disease: pathogenesis, diagnosis and public health. *Viruses* 2026; 18(2): 216. <https://doi.org/10.3390/v18020216>
2. Udayakumar S, Girigoswami A, Girigoswami K. A review on current theories and potential therapies for prion diseases. *Mol Biol Rep.* 2025; 52: 674. doi: 10.1007/s11033-025-10754-2.
3. Abdul H, Konold T, Spiropoulos J, Lewis PA. New frontiers in animal prion diseases. *Annu Rev Anim Biosci.* 2026; 14: 219-339. doi: 10.1146/annurev-animal-111523-102335.
4. Caughey B, Artikis E, Shoup D, et al. Prions and protein aggregates as pathogens, self-propagating structures, biomarkers, and therapeutic targets. *Microbiol Mol Biol Rev.* 2025; 89(4): e00007-25. doi: 10.1128/mmb.00007-25.
5. Casey C, Sleator RD. Prions: structure, function, evolution, and disease. *Arch Microbiol.* 2025; 207(1):1. doi: 10.1007/s00203-024-04200-3.
6. Holman AP, Kurouski D. Prion diseases: Lessons from historical outbreaks and potential emerging ones. *Protein Sci.* 2025; 34(6): e70175. doi: 10.1002/pro.70175.
7. Shimamura MI, Satoh K. Challenges and revisions in diagnostic criteria: Advancing early detection of prion diseases. *Int J Mol Sci.* 2025; 26(5): 2037. doi: 10.3390/ijms26052037.
8. Gao LP, Tian TT, Xiao K, et al. Updated global epidemiology atlas of human prion diseases. *Front Public Health.* 2024; 12: 1411489. doi: 10.3389/fpubh.2024.1411489.
9. Linden R, Martins VR, Prado MA, et al. Physiology of the prion protein. *Physiol Rev.* 2008; 88: 673-728. doi: 10.1152/physrev.00007.2007
10. Vostal JG, Holada K, Simak J. Expression of cellular prion protein on blood cells: potential functions in cell physiology and pathophysiology of transmissible spongiform encephalopathy diseases. *Transfus Med Rev.* 2001; 15(4): 268-81. doi: 10.1053/tmrv.2001.26957.
11. Holada K, Glierova H, Simak J, Vostal JG. Expression of cellular prion protein on platelets from patients with gray platelet or Hermansky-Pudlak syndrome and the protein's association with alpha-granules. *Haematologica.* 2006; 91(8): 1126-9.
12. Starke R, Harrison P, Mackie I, et al. The expression of prion protein (PrP(C)) in the megakaryocyte lineage. *J Thromb Haemost.* 2005; 3(6): 1266-73. doi: 10.1111/j.1538-7836.2005.01343.x.
13. Li R, Liu D, Zanusso G, et al. The expression and potential function of cellular prion protein in human lymphocytes. *Cell Immunol.* 2001; 207: 49-58. doi: 10.1006/cimm.2000.1751.
14. Wulf MA, Senatore A, Aguzzi A. The biological function of the cellular prion protein: an update. *BMC Biol.* 2017; 15: 34. doi: 10.1186/s12915-017-0375-5
15. Prusiner SB. Prions. *Proc Natl Acad Sci U S A.* 1998; 95: 13363-83. doi: 10.1073/pnas.95.23.13363
16. Napper S, Schatzl HM. Vaccines for prion diseases: a realistic goal?. *Cell Tissue Res.* 2023; 392: 367-392. <https://doi.org/10.1007/s00441-023-03749-7>
17. Liu F, Lu W, Liu L. New implications for prion diseases therapy and prophylaxis. *Front Mol Neurosci.* 2024; 17: 1324702. doi: 10.3389/fnmol.2024.1324702
18. Senatore A, Frontzek K, Emmenegger M, et al. Protective anti-prion antibodies in human immunoglobulin repertoires. *EMBO Mol Med.* 2020; 12: e12739. doi: 10.15252/emmm.202012739
19. Frontzek K, Carta M, Losa M, et al. Autoantibodies against the prion protein in individuals with PRNP mutations. *Neurology.* 2020; 95: e2028-e2037. doi: 10.1212/WNL.0000000000009183
20. Georgopoulos AP, James LM. Solid tumor rejection using personalized incompatible human leukocyte antigen (HLA) and blood group ABH antigens. *Explor Med.* 2025; 6: 10.37349/emed.2025.1001375. doi: 10.37349/emed.2025.1001375.
21. Trowsdale J, Knight JC. Major histocompatibility complex genomics and human disease. *Annu Rev Genomics Hum Genet.* 2013; 14: 301-23. doi: 10.1146/annurev-genom-091212-153455
22. Hov JR, Kosmoliaptis V, Traherne JA, et al. Electrostatic modifications of the human leukocyte antigen-DR P9 peptide-binding pocket and susceptibility to primary sclerosing cholangitis. *Hepatology* 2011; 53: 1967-76. doi: 10.1002/hep.24299
23. Davenport MP, Quinn CL, Chic RM, et al. Naturally processed peptides from two disease-resistance-associated HLA-DR13 alleles show related sequence motifs and the effects of the dimorphism at position 86 of the HLA-DR beta chain. *Proc Natl Acad Sci U S A.* 1995; 92: 6567-71. doi: 10.1073/pnas.92.14.6567
24. van Deutekom HW, Kesmir C. Zooming into the binding groove of HLA molecules: which positions and which substitutions change peptide binding most? *Immunogenetics.* 2015; 67: 425-36. doi: 10.1007/s00251-015-0849-y

25. Paul S, Weiskopf D, Angelo MA, et al. HLA class I alleles are associated with peptide-binding repertoires of different size, affinity, and immunogenicity. *J Immunol.* 2013; 191: 5831-9. doi: 10.4049/jimmunol.1302101
26. Han L, Wu T, Zhang Q, Qi A, Zhou X. Immune tolerance regulation is critical to immune homeostasis. *J Immunol Res.* 2025; 2025: 5006201. doi: 10.1155/jimr/5006201.
27. Mackay IR. Science, medicine, and the future: Tolerance and autoimmunity. *BMJ.* 2000; 321(7253): 93-6. doi: 10.1136/bmj.321.7253.93.
28. Kamradt T, Mitchison NA. Tolerance and autoimmunity. *N Engl J Med.* 2001; 344(9): 655-64. doi: 10.1056/NEJM200103013440907.
29. Abend AH, He I, Bahroos N, et al. Estimation of prevalence of autoimmune diseases in the United States using electronic health record data. *J Clin Invest.* 2024; 135(4): e178722. doi: 10.1172/JCI178722.
30. Sartoris S, Del Pozzo G. Exploring the HLA complex in autoimmunity: From the risk haplotypes to the modulation of expression. *Clin Immunol.* 2024; 265: 110266. doi: 10.1016/j.clim.2024.110266.
31. Hurley CK, Kempenich J, Wadsworth K, et al. Common, intermediate and well-documented HLA alleles in world populations: CIWD version 3.0.0. *HLA.* 2020; 95: 516-31. doi: 10.1111/tan.13811
32. Reynisson B, Alvarez B, Paul S, Peters B, Nielsen M. NetMHCpan-4.1 and NetMHCIpan-4.0: improved predictions of MHC antigen presentation by concurrent motif deconvolution and integration of MS MHC eluted ligand data. *Nucleic Acids Res.* 2020; 48: W449-W454. doi: 10.1093/nar/gkaa379
33. IEDB Analysis Resource. Accessed on October 18, 2025. <http://tools.iedb.org/mhci/>
34. Charonis S, James LM, Georgopoulos AP. In silico assessment of binding affinities of three dementia-protective Human Leukocyte Antigen (HLA) alleles to nine human herpes virus antigens. *Curr Res Transl Med.* 2020; 68: 211-216. doi: 10.1016/j.retram.2020.06.002
35. Charonis S, Tsilibary EP, Georgopoulos A. SARS-CoV-2 virus and Human Leukocyte Antigen (HLA) Class II: investigation in silico of binding affinities for COVID-19 protection and vaccine development. *J Immunol Sci.* 2020; 4: 12-23. doi: 10.29245/2578-3009/2020/4.1198
36. Charonis SA, Tsilibary EP, Georgopoulos AP. In silico investigation of binding affinities between human leukocyte antigen class I molecules and SARS-CoV-2 virus spike and ORF1ab proteins. *Explor Immunol.* 2021; 1: 16-26. doi: 10.37349/ei.2021.00003
37. Istrail S, Florea L, Halldorsson BV, et al. Comparative immunopeptidomics of humans and their pathogens. *Proc Natl Acad Sci USA.* 2004; 101: 13268-72. doi: 10.1073/pnas.0404740101.
38. Deriziotis P, Tabrizi SJ. Prions and the proteasome. *Biochim Biophys Acta.* 2008; 1782(12): 713-22. doi: 10.1016/j.bbdis.2008.06.011.
39. Goold R, McKinnon C, Tabrizi SJ. Prion degradation pathways: Potential for therapeutic intervention. *Mol Cell Neurosci.* 2015; 66(Pt A): 12-20. doi: 10.1016/j.mcn.2014.12.009.
40. Yi CW, Wang LQ, Huang JJ, Pan K, Chen J, Liang Y. Glycosylation significantly inhibits the aggregation of human prion protein and decreases its cytotoxicity. *Sci Rep.* 2018; 8(1): 12603. doi: 10.1038/s41598-018-30770-6.
41. Schilling KM, Jorwal P, Ubilla-Rodriguez NC, et al. N-glycosylation is a potent regulator of prion protein neurotoxicity. *J Biol Chem.* 2023; 299(9): 105101. doi: 10.1016/j.jbc.2023.105101.
42. Maiti NR, Surewicz WK. The role of disulfide bridge in the folding and stability of the recombinant human prion protein. *J Biol Chem.* 2001; 276(4): 2427-31. doi: 10.1074/jbc.M007862200.
43. Mossuto MF. Disulfide bonding in neurodegenerative misfolding diseases. *Int J Cell Biol.* 2013; 2013: 318319. doi: 10.1155/2013/318319.
44. Sonati T, Reimann R, Falsig J, et al. The toxicity of antiprion antibodies is mediated by the flexible tail of the prion protein. *Nature.* 2013; 501: 102-106. <https://doi.org/10.1038/nature12402>
45. Marciniuk K, Taschuk R, Napper S. Evidence for prion-like mechanisms in several neurodegenerative diseases: potential implications for immunotherapy. *J Immunol Res.* 2013; 2013: 473706. doi: 10.1155/2013/473706
46. Shrivastava NA, Aperia A, Melki R, Triller A. Physico-pathologic mechanisms involved in neurodegeneration: Misfolded protein-plasma membrane interactions. *Neuron.* 2017; 95: 33-50. doi: 10.1016/j.neuron.2017.05.026
47. Sarnataro D. Attempt to untangle the prion-like misfolding mechanism for neurodegenerative diseases. *Int J Mol Sci.* 2018; 19: 3081. doi: 10.3390/ijms19103081
48. James LM, Georgopoulos AP. Immunogenetic epidemiology of dementia and Parkinson's Disease in 14 Continental European Countries: shared Human Leukocyte Antigen (HLA) profiles. *J Immunol Sci.* 2021; 5(2): 16-26. doi: 10.29245/2578-3009/2021/2.1209.
49. James LM, Georgopoulos AP. Immunogenetic Epidemiology of Motor Neuron Diseases in 14 Continental Western European Countries. *J Immunological Sci.* (2021); 5(3): 22-28

Supplementary Tables

Table S1. List of the 142 HLA-I alleles used.

Index	Allele		Allele
1	HLA-A*01:01	41	HLA-A*80:01
2	HLA-A*02:01	42	HLA-B*07:02
3	HLA-A*02:02	43	HLA-B*07:04
4	HLA-A*02:05	44	HLA-B*07:05
5	HLA-A*02:06	45	HLA-B*08:01
6	HLA-A*02:17	46	HLA-B*13:02
7	HLA-A*02:30	47	HLA-B*14:01
8	HLA-A*02:35	48	HLA-B*14:02
9	HLA-A*02:63	49	HLA-B*14:03
10	HLA-A*02:77	50	HLA-B*15:01
11	HLA-A*03:01	51	HLA-B*15:03
12	HLA-A*03:02	52	HLA-B*15:07
13	HLA-A*03:81	53	HLA-B*15:09
14	HLA-A*11:01	54	HLA-B*15:10
15	HLA-A*11:02	55	HLA-B*15:16
16	HLA-A*23:01	56	HLA-B*15:17
17	HLA-A*24:02	57	HLA-B*15:18
18	HLA-A*24:03	58	HLA-B*15:24
19	HLA-A*25:01	59	HLA-B*15:35
20	HLA-A*26:01	60	HLA-B*18:01
21	HLA-A*26:08	61	HLA-B*18:09
22	HLA-A*26:12	62	HLA-B*27:02
23	HLA-A*29:01	63	HLA-B*27:04
24	HLA-A*29:02	64	HLA-B*27:05
25	HLA-A*30:01	65	HLA-B*27:07
26	HLA-A*30:02	66	HLA-B*27:08
27	HLA-A*30:04	67	HLA-B*27:10
28	HLA-A*31:01	68	HLA-B*35:01
29	HLA-A*32:01	69	HLA-B*35:02
30	HLA-A*33:01	70	HLA-B*35:03
31	HLA-A*33:03	71	HLA-B*35:08
32	HLA-A*34:01	72	HLA-B*35:17
33	HLA-A*34:02	73	HLA-B*37:01
34	HLA-A*36:01	74	HLA-B*38:01
35	HLA-A*66:01	75	HLA-B*39:01
36	HLA-A*68:01	76	HLA-B*39:02
37	HLA-A*68:02	77	HLA-B*39:05
38	HLA-A*68:37	78	HLA-B*39:06
39	HLA-A*74:01	79	HLA-B*39:24
40	HLA-A*74:03	80	HLA-B*40:01
		81	HLA-B*40:02

82	HLA-B*41:01
83	HLA-B*41:02
84	HLA-B*42:02
85	HLA-B*44:02
86	HLA-B*44:03
87	HLA-B*44:04
88	HLA-B*44:05
89	HLA-B*44:07
90	HLA-B*44:27
91	HLA-B*45:01
92	HLA-B*47:01
93	HLA-B*48:01
94	HLA-B*48:07
95	HLA-B*49:01
96	HLA-B*50:01
97	HLA-B*50:02
98	HLA-B*51:01
99	HLA-B*51:02
100	HLA-B*51:07
101	HLA-B*51:09
102	HLA-B*52:01
103	HLA-B*53:01
104	HLA-B*54:01
105	HLA-B*55:01
106	HLA-B*56:01
107	HLA-B*57:01
108	HLA-B*57:02
109	HLA-B*57:03
110	HLA-B*58:01
111	HLA-B*58:02
112	HLA-B*59:01
113	HLA-B*81:01
114	HLA-C*01:02
115	HLA-C*02:02
116	HLA-C*02:10
117	HLA-C*03:02
118	HLA-C*03:03
119	HLA-C*03:04
120	HLA-C*04:01
121	HLA-C*05:01
122	HLA-C*06:02
123	HLA-C*07:01
124	HLA-C*07:02

125	HLA-C*07:04
126	HLA-C*07:19
127	HLA-C*08:01
128	HLA-C*08:02
129	HLA-C*08:03
130	HLA-C*12:02
131	HLA-C*12:03
132	HLA-C*14:02
133	HLA-C*15:02
134	HLA-C*15:04
135	HLA-C*15:05
136	HLA-C*15:06
137	HLA-C*15:09
138	HLA-C*16:01
139	HLA-C*16:02
140	HLA-C*16:04
141	HLA-C*17:01
142	HLA-C*18:01

Table S2. List of the 192 HLA-II alleles used.

Index	Allele
1	DPB1*01:01
2	DPB1*02:01
3	DPB1*02:02
4	DPB1*03:01
5	DPB1*04:01
6	DPB1*04:02
7	DPB1*05:01
8	DPB1*06:01
9	DPB1*09:01
10	DPB1*10:01
11	DPB1*104:01
12	DPB1*105:01
13	DPB1*11:01
14	DPB1*124:01
15	DPB1*126:01
16	DPB1*13:01
17	DPB1*14:01
18	DPB1*15:01
19	DPB1*16:01
20	DPB1*17:01
21	DPB1*19:01
22	DPB1*20:01

23	DPB1*23:01	66	DQB1*06:07
24	DPB1*26:01	67	DQB1*06:08
25	DPB1*28:01	68	DQB1*06:09
26	DPB1*30:01	69	DQB1*06:11
27	DPB1*33:01	70	DQB1*06:14
28	DPB1*34:01	71	DQB1*06:15
29	DPB1*35:01	72	DQB1*06:18
30	DPB1*39:01	73	DQB1*06:19
31	DPB1*40:01	74	DQB1*06:22
32	DPB1*41:01	75	DQB1*06:27
33	DPB1*46:01	76	DQB1*06:32
34	DPB1*47:01	77	DRB1*01:01
35	DPB1*49:01	78	DRB1*01:02
36	DPB1*55:01	79	DRB1*01:03
37	DPB1*71:01	80	DRB1*01:11
38	DPB1*72:01	81	DRB1*01:18
39	DPB1*81:01	82	DRB1*01:20
40	DPB1*85:01	83	DRB1*01:24
41	DPB1*91:01	84	DRB1*01:29
42	DQB1*02:02	85	DRB1*03:01
43	DQB1*03:01	86	DRB1*03:02
44	DQB1*03:02	87	DRB1*03:04
45	DQB1*03:03	88	DRB1*03:05
46	DQB1*03:04	89	DRB1*03:11
47	DQB1*03:05	90	DRB1*03:13
48	DQB1*03:10	91	DRB1*03:15
49	DQB1*03:14	92	DRB1*03:41
50	DQB1*03:17	93	DRB1*04:01
51	DQB1*03:19	94	DRB1*04:02
52	DQB1*03:23	95	DRB1*04:03
53	DQB1*03:25	96	DRB1*04:04
54	DQB1*04:01	97	DRB1*04:05
55	DQB1*04:02	98	DRB1*04:06
56	DQB1*04:03	99	DRB1*04:07
57	DQB1*05:01	100	DRB1*04:08
58	DQB1*05:02	101	DRB1*04:10
59	DQB1*05:03	102	DRB1*04:11
60	DQB1*05:06	103	DRB1*04:17
61	DQB1*05:11	104	DRB1*04:44
62	DQB1*06:01	105	DRB1*04:53
63	DQB1*06:02	106	DRB1*04:56
64	DQB1*06:03	107	DRB1*04:72
65	DQB1*06:04	108	DRB1*07:01

109	DRB1*08:01
110	DRB1*08:02
111	DRB1*08:03
112	DRB1*08:04
113	DRB1*08:24
114	DRB1*08:30
115	DRB1*08:36
116	DRB1*09:01
117	DRB1*09:02
118	DRB1*10:01
119	DRB1*11:01
120	DRB1*11:02
121	DRB1*11:03
122	DRB1*11:04
123	DRB1*11:06
124	DRB1*11:07
125	DRB1*11:08
126	DRB1*11:10
127	DRB1*11:11
128	DRB1*11:12
129	DRB1*11:13
130	DRB1*11:14
131	DRB1*11:19
132	DRB1*11:27
133	DRB1*11:28
134	DRB1*11:29
135	DRB1*11:37
136	DRB1*11:42
137	DRB1*11:46
138	DRB1*11:49
139	DRB1*11:54
140	DRB1*11:58
141	DRB1*11:62
142	DRB1*11:65
143	DRB1*11:74
144	DRB1*11:84
145	DRB1*12:01
146	DRB1*12:02
147	DRB1*12:03
148	DRB1*12:16
149	DRB1*13:01
150	DRB1*13:02
151	DRB1*13:03

152	DRB1*13:05
153	DRB1*13:07
154	DRB1*13:11
155	DRB1*13:12
156	DRB1*13:14
157	DRB1*13:21
158	DRB1*13:23
159	DRB1*13:26
160	DRB1*13:33
161	DRB1*13:50
162	DRB1*13:61
163	DRB1*13:66
164	DRB1*13:96
165	DRB1*13:97
166	DRB1*14:01
167	DRB1*14:02
168	DRB1*14:03
169	DRB1*14:04
170	DRB1*14:05
171	DRB1*14:06
172	DRB1*14:07
173	DRB1*14:12
174	DRB1*14:23
175	DRB1*14:27
176	DRB1*14:32
177	DRB1*14:38
178	DRB1*14:44
179	DRB1*14:54
180	DRB1*14:68
181	DRB1*15:01
182	DRB1*15:02
183	DRB1*15:03
184	DRB1*15:06
185	DRB1*15:07
186	DRB1*15:15
187	DRB1*15:37
188	DRB1*16:01
189	DRB1*16:02
190	DRB1*16:04
191	DRB1*16:05
192	DRB1*16:09

Table S3. Peptides (9-mer) of PRP^c binding with high affinity (IC₅₀ < 50 nM) to HLA-I molecules.

Index	Allele	Start	End	Peptide	min(IC ₅₀)
1	A*02:01	232	240	MVLFSSPPV	19.28
2	A*02:01	242	250	LLISFLIFL	21.74
3	A*02:02	242	250	LLISFLIFL	21.62
4	A*02:02	243	251	LISFLIFLI	22.31
5	A*02:05	232	240	MVLFSSPPV	36.84
6	A*02:06	232	240	MVLFSSPPV	6.41
7	A*02:06	242	250	LLISFLIFL	45.43
8	A*02:30	232	240	MVLFSSPPV	19.28
9	A*02:30	242	250	LLISFLIFL	21.74
10	A*02:35	232	240	MVLFSSPPV	11.92
11	A*02:35	233	241	VLFSPPVI	28.52
12	A*02:35	242	250	LLISFLIFL	33.02
13	A*02:35	243	251	LISFLIFLI	42.66
14	A*02:63	242	250	LLISFLIFL	21.62
15	A*02:63	243	251	LISFLIFLI	22.31
16	A*02:77	232	240	MVLFSSPPV	19.28
17	A*02:77	242	250	LLISFLIFL	21.74
18	A*24:03	225	233	YYQRGSSMV	17.43
19	A*24:03	224	232	AYYQRGSSM	32.21
20	A*26:01	120	128	AVVGGLGGY	18.35
21	A*26:08	120	128	AVVGGLGGY	22.27
22	A*29:01	149	157	YYRENMHRY	30.34
23	A*29:02	149	157	YYRENMHRY	30.34
24	A*30:01	134	142	MSRPIIHF	20.52
25	A*30:01	106	114	KTNMKHMAG	24.40
26	A*30:01	96	104	HSQWNKPSK	33.00
27	A*30:02	120	128	AVVGGLGGY	22.99
28	A*31:01	148	156	RYYRENMHR	9.50
29	A*31:01	156	164	RYPNQVYYR	11.00
30	A*31:01	212	220	QMCITQYER	28.98
31	A*68:02	232	240	MVLFSSPPV	10.70
32	B*07:02	101	109	KPSKPKTNM	24.54
33	B*07:05	101	109	KPSKPKTNM	14.38
34	B*15:01	226	234	YQRGSSMVL	15.83
35	B*15:01	133	141	AMSRPIIHF	35.33
36	B*15:03	226	234	YQRGSSMVL	3.40
37	B*15:03	154	162	MHRYPNQVY	7.32
38	B*15:03	133	141	AMSRPIIHF	17.45
39	B*15:03	218	226	YERESQAYY	18.85
40	B*15:03	155	163	HRYPNQVYY	32.51
41	B*15:03	109	117	MKHMAGAAA	38.59
42	B*15:03	241	249	ILLISFLIF	47.91
43	B*15:17	235	243	FSSPPVILL	16.04
44	B*15:18	154	162	MHRYPNQVY	31.57
45	B*18:01	167	175	DEYSNQNNF	26.50
46	B*18:01	218	226	YERESQAYY	34.56
47	B*39:02	226	234	YQRGSSMVL	17.58
48	C*03:02	235	243	FSSPPVILL	19.16
49	C*03:02	226	234	YQRGSSMVL	35.20
50	C*03:03	235	243	FSSPPVILL	7.82
51	C*03:03	226	234	YQRGSSMVL	37.08
52	C*03:04	235	243	FSSPPVILL	7.82
53	C*03:04	226	234	YQRGSSMVL	37.08
54	C*07:01	155	163	HRYPNQVYY	32.07
55	C*07:02	149	157	YYRENMHRY	37.07
56	C*07:02	155	163	HRYPNQVYY	38.83
57	C*07:02	224	232	AYYQRGSSM	46.16
58	C*12:03	235	243	FSSPPVILL	12.15
59	C*14:02	224	232	AYYQRGSSM	2.49
60	C*14:02	149	157	YYRENMHRY	6.21
61	C*14:02	225	233	YYQRGSSMV	11.39
62	C*14:02	161	169	VYYRPMDEY	23.88
63	C*14:02	11	19	LFVATWSDL	29.66
64	C*14:02	234	242	LFSSPPVIL	33.27
65	C*15:02	235	243	FSSPPVILL	17.92
66	C*15:04	235	243	FSSPPVILL	23.04
67	C*15:05	235	243	FSSPPVILL	24.99
68	C*15:06	235	243	FSSPPVILL	11.97
69	C*15:09	235	243	FSSPPVILL	23.04
70	C*16:01	235	243	FSSPPVILL	11.54
71	C*16:02	235	243	FSSPPVILL	13.36
72	C*16:04	235	243	FSSPPVILL	16.02
73	C*17:01	235	243	FSSPPVILL	11.12

Table S4. Peptides (15-mer) of PRP^c binding with high affinity (IC₅₀ < 50 nM) to HLA-II molecules.

Index	Allele	Start	End	Peptide	IC ₅₀	53	DRB1*01:11	230	244	SSMVLFSPPVILLI	28.76
1	DRB1*01:01	106	120	KTNMKHMAGAAAAGA	6.93	54	DRB1*01:11	229	243	GSSMVLFSPPVILL	29.84
2	DRB1*01:01	223	237	QAYYQRGSSMVLFS	7.40	55	DRB1*01:11	231	245	SMVLFSPPVILLIS	30.40
3	DRB1*01:01	105	119	PKTNMKHMAGAAAAG	7.65	56	DRB1*01:11	104	118	KPKTNMKHMAGAAAA	32.10
4	DRB1*01:01	104	118	KPKTNMKHMAGAAAA	7.99	57	DRB1*01:11	107	121	TNMKHMAGAAAAGAV	35.42
5	DRB1*01:01	222	236	SQAYYQRGSSMVLFS	8.36	58	DRB1*01:11	127	141	GYMLGSAMSRPIHF	38.36
6	DRB1*01:01	107	121	TNMKHMAGAAAAGAV	8.50	59	DRB1*01:11	232	246	MVLFSPPVILLISF	44.43
7	DRB1*01:01	108	122	NMKHMAGAAAAGAVV	10.72	60	DRB1*01:11	125	139	LGGYMLGSAMSRPII	44.56
8	DRB1*01:01	224	238	AYYQRGSSMVLFS	11.13	61	DRB1*01:11	126	140	GGYMLGSAMSRPIIH	45.39
9	DRB1*01:01	221	235	ESQAYYQRGSSMVL	11.35	62	DRB1*01:11	228	242	RGSSMVLFSPPVIL	46.12
10	DRB1*01:01	127	141	GYMLGSAMSRPIHF	12.29	63	DRB1*01:18	106	120	KTNMKHMAGAAAAGA	7.93
11	DRB1*01:01	126	140	GGYMLGSAMSRPIIH	13.74	64	DRB1*01:18	223	237	QAYYQRGSSMVLFS	8.43
12	DRB1*01:01	125	139	LGGYMLGSAMSRPII	14.53	65	DRB1*01:18	105	119	PKTNMKHMAGAAAAG	9.26
13	DRB1*01:01	230	244	SSMVLFSPPVILLI	15.75	66	DRB1*01:18	222	236	SQAYYQRGSSMVLFS	9.36
14	DRB1*01:01	231	245	SMVLFSPPVILLIS	16.06	67	DRB1*01:18	104	118	KPKTNMKHMAGAAAA	10.16
15	DRB1*01:01	229	243	GSSMVLFSPPVILL	16.19	68	DRB1*01:18	107	121	TNMKHMAGAAAAGAV	10.37
16	DRB1*01:01	124	138	GLGGYMLGSAMSRPI	18.61	69	DRB1*01:18	221	235	ESQAYYQRGSSMVL	12.36
17	DRB1*01:01	128	142	YMLGSAMSRPIHFG	19.17	70	DRB1*01:18	127	141	GYMLGSAMSRPIHF	12.43
18	DRB1*01:01	103	117	SKPKTNMKHMAGAAA	21.12	71	DRB1*01:18	224	238	AYYQRGSSMVLFS	12.48
19	DRB1*01:01	119	133	GAVVGGLGGYMLGSA	22.16	72	DRB1*01:18	229	243	GSSMVLFSPPVILL	12.61
20	DRB1*01:01	225	239	YYQRGSSMVLFS	22.22	73	DRB1*01:18	230	244	SSMVLFSPPVILLI	13.03
21	DRB1*01:01	228	242	RGSSMVLFSPPVIL	22.52	74	DRB1*01:18	126	140	GGYMLGSAMSRPIIH	13.32
22	DRB1*01:01	232	246	MVLFSPPVILLISF	23.21	75	DRB1*01:18	108	122	NMKHMAGAAAAGAVV	13.58
23	DRB1*01:01	109	123	MKHMAGAAAAGAVVG	24.72	76	DRB1*01:18	125	139	LGGYMLGSAMSRPII	13.94
24	DRB1*01:01	220	234	RESQAYYQRGSSMVL	25.35	77	DRB1*01:18	231	245	SMVLFSPPVILLIS	14.78
25	DRB1*01:01	120	134	AVVGGLGGYMLGSAM	28.96	78	DRB1*01:18	228	242	RGSSMVLFSPPVIL	16.69
26	DRB1*01:01	123	137	GGLGGYMLGSAMSRP	28.97	79	DRB1*01:18	124	138	GLGGYMLGSAMSRPI	18.88
27	DRB1*01:01	118	132	AGAVVGGLGGYMLGS	30.05	80	DRB1*01:18	128	142	YMLGSAMSRPIHFG	19.71
28	DRB1*01:01	122	136	VGGLGGYMLGSAMSR	36.63	81	DRB1*01:18	232	246	MVLFSPPVILLISF	22.06
29	DRB1*01:01	227	241	QRGSSMVLFSPPVI	36.82	82	DRB1*01:18	119	133	GAVVGGLGGYMLGSA	23.48
30	DRB1*01:01	233	247	VLFSPPVILLISFL	41.55	83	DRB1*01:18	220	234	RESQAYYQRGSSMVL	25.66
31	DRB1*01:01	129	143	MLGSAMSRPIHFGS	43.97	84	DRB1*01:18	225	239	YYQRGSSMVLFS	25.68
32	DRB1*01:01	117	131	AAGAVVGGLGGYMLG	46.16	85	DRB1*01:18	227	241	QRGSSMVLFSPPVI	26.17
33	DRB1*01:02	106	120	KTNMKHMAGAAAAGA	21.53	86	DRB1*01:18	103	117	SKPKTNMKHMAGAAA	26.67
34	DRB1*01:02	127	141	GYMLGSAMSRPIHF	23.12	87	DRB1*01:18	120	134	AVVGGLGGYMLGSAM	28.47
35	DRB1*01:02	105	119	PKTNMKHMAGAAAAG	25.47	88	DRB1*01:18	118	132	AGAVVGGLGGYMLGS	30.65
36	DRB1*01:02	104	118	KPKTNMKHMAGAAAA	27.77	89	DRB1*01:18	123	137	GGLGGYMLGSAMSRP	32.26
37	DRB1*01:02	230	244	SSMVLFSPPVILLI	28.07	90	DRB1*01:18	109	123	MKHMAGAAAAGAVVG	33.06
38	DRB1*01:02	107	121	TNMKHMAGAAAAGAV	28.45	91	DRB1*01:18	233	247	VLFSPPVILLISFL	39.27
39	DRB1*01:02	229	243	GSSMVLFSPPVILL	30.49	92	DRB1*01:18	122	136	VGGLGGYMLGSAMSR	42.11
40	DRB1*01:02	231	245	SMVLFSPPVILLIS	30.75	93	DRB1*01:18	117	131	AAGAVVGGLGGYMLG	44.28
41	DRB1*01:02	128	142	YMLGSAMSRPIHFG	31.72	94	DRB1*01:18	226	240	YQRGSSMVLFSPPV	46.28
42	DRB1*01:02	126	140	GGYMLGSAMSRPIIH	32.46	95	DRB1*01:18	129	143	MLGSAMSRPIHFGS	48.50
43	DRB1*01:02	108	122	NMKHMAGAAAAGAVV	37.20	96	DRB1*01:20	106	120	KTNMKHMAGAAAAGA	8.31
44	DRB1*01:02	125	139	LGGYMLGSAMSRPII	40.18	97	DRB1*01:20	105	119	PKTNMKHMAGAAAAG	9.36
45	DRB1*01:02	228	242	RGSSMVLFSPPVIL	47.95	98	DRB1*01:20	104	118	KPKTNMKHMAGAAAA	9.75
46	DRB1*01:02	119	133	GAVVGGLGGYMLGSA	48.50	99	DRB1*01:20	107	121	TNMKHMAGAAAAGAV	10.18
47	DRB1*01:11	223	237	QAYYQRGSSMVLFS	18.16	100	DRB1*01:20	229	243	GSSMVLFSPPVILL	11.23
48	DRB1*01:11	222	236	SQAYYQRGSSMVLFS	20.57	101	DRB1*01:20	127	141	GYMLGSAMSRPIHF	11.49
49	DRB1*01:11	106	120	KTNMKHMAGAAAAGA	21.23	102	DRB1*01:20	230	244	SSMVLFSPPVILLI	11.87
50	DRB1*01:11	224	238	AYYQRGSSMVLFS	27.11	103	DRB1*01:20	108	122	NMKHMAGAAAAGAVV	12.12
51	DRB1*01:11	105	119	PKTNMKHMAGAAAAG	27.74	104	DRB1*01:20	231	245	SMVLFSPPVILLIS	13.29
52	DRB1*01:11	221	235	ESQAYYQRGSSMVL	27.98	105	DRB1*01:20	228	242	RGSSMVLFSPPVIL	14.56

106	DRB1*01:20	126	140	GGYMLGSAMSRPIIH	14.73	160	DRB1*01:29	228	242	RGSSMVLFSPPVIL	37.89
107	DRB1*01:20	119	133	GAVVGLGGYMLGSA	14.77	161	DRB1*01:29	124	138	GLGGYMLGSAMSRPI	43.18
108	DRB1*01:20	125	139	LGGYMLGSAMSRPII	16.65	162	DRB1*01:29	225	239	YYQRGSSMVLFSPP	49.43
109	DRB1*01:20	128	142	YMLGSAMSRPIIHFG	16.75	163	DRB1*01:29	109	123	MKHMAGAAAAGAVVG	49.99
110	DRB1*01:20	118	132	AGAVVGLGGYMLGS	18.85	164	DRB1*04:01	172	186	QNNFVHDCVNITIKQ	22.64
111	DRB1*01:20	120	134	AVVGLGGYMLGSAM	19.21	165	DRB1*04:01	171	185	NQNNFVHDCVNITIK	23.88
112	DRB1*01:20	232	246	MVLFSPPVILLISF	22.84	166	DRB1*04:01	173	187	NNFVHDCVNITIKQH	30.82
113	DRB1*01:20	227	241	QRGSSMVLFSPPVI	23.38	167	DRB1*04:01	170	184	SNQNNFVHDCVNITI	37.41
114	DRB1*01:20	103	117	SKPKTNMKHMAGAAA	24.78	168	DRB1*04:04	229	243	GSSMVLFSPPVILL	36.22
115	DRB1*01:20	124	138	GLGGYMLGSAMSRPI	25.19	169	DRB1*04:04	228	242	RGSSMVLFSPPVIL	42.31
116	DRB1*01:20	117	131	AAGAVVGLGGYMLG	27.24	170	DRB1*04:10	229	243	GSSMVLFSPPVILL	39.61
117	DRB1*01:20	109	123	MKHMAGAAAAGAVVG	30.35	171	DRB1*04:10	228	242	RGSSMVLFSPPVIL	44.46
118	DRB1*01:20	223	237	QAYYQRGSSMVLFS	30.93	172	DRB1*04:10	230	244	SSMVLFSPPVILLI	49.89
119	DRB1*01:20	222	236	SQAYYQRGSSMVLFS	31.98	173	DRB1*04:72	172	186	QNNFVHDCVNITIKQ	25.13
120	DRB1*01:20	129	143	MLGSAMSRPIIHFGS	36.80	174	DRB1*04:72	171	185	NQNNFVHDCVNITIK	26.85
121	DRB1*01:20	121	135	VVGLGGYMLGSAMS	37.26	175	DRB1*04:72	173	187	NNFVHDCVNITIKQH	34.26
122	DRB1*01:20	221	235	ESQAYYQRGSSMVLFS	40.23	176	DRB1*04:72	170	184	SNQNNFVHDCVNITI	42.02
123	DRB1*01:20	123	137	GGLGGYMLGSAMSRP	43.03	177	DRB1*07:01	127	141	GYMLGSAMSRPIIHF	14.05
124	DRB1*01:24	106	120	KTNMKHMAGAAAAGA	14.71	178	DRB1*07:01	230	244	SSMVLFSPPVILLI	16.37
125	DRB1*01:24	223	237	QAYYQRGSSMVLFS	16.19	179	DRB1*07:01	126	140	GGYMLGSAMSRPIIH	16.61
126	DRB1*01:24	222	236	SQAYYQRGSSMVLFS	17.64	180	DRB1*07:01	229	243	GSSMVLFSPPVILL	16.64
127	DRB1*01:24	105	119	PKTNMKHMAGAAAAG	17.83	181	DRB1*07:01	125	139	LGGYMLGSAMSRPII	17.75
128	DRB1*01:24	104	118	KPKTNMKHMAGAAAA	19.83	182	DRB1*07:01	231	245	SMVLFSPPVILLIS	17.78
129	DRB1*01:24	107	121	TNMKHMAGAAAAGAV	21.38	183	DRB1*07:01	222	236	SQAYYQRGSSMVLFS	20.35
130	DRB1*01:24	230	244	SSMVLFSPPVILLI	22.94	184	DRB1*07:01	228	242	RGSSMVLFSPPVIL	20.68
131	DRB1*01:24	224	238	AYYQRGSSMVLFS	23.12	185	DRB1*07:01	128	142	YMLGSAMSRPIIHFG	20.86
132	DRB1*01:24	221	235	ESQAYYQRGSSMVLFS	23.48	186	DRB1*07:01	223	237	QAYYQRGSSMVLFS	21.18
133	DRB1*01:24	229	243	GSSMVLFSPPVILL	24.22	187	DRB1*07:01	221	235	ESQAYYQRGSSMVLFS	21.81
134	DRB1*01:24	231	245	SMVLFSPPVILLIS	24.73	188	DRB1*07:01	232	246	MVLFSPPVILLISF	26.93
135	DRB1*01:24	127	141	GYMLGSAMSRPIIHF	27.32	189	DRB1*07:01	124	138	GLGGYMLGSAMSRPI	28.93
136	DRB1*01:24	108	122	NMKHMAGAAAAGAVV	32.50	190	DRB1*07:01	220	234	RESQAYYQRGSSMVL	29.31
137	DRB1*01:24	125	139	LGGYMLGSAMSRPII	36.15	191	DRB1*07:01	224	238	AYYQRGSSMVLFS	33.65
138	DRB1*01:24	228	242	RGSSMVLFSPPVIL	36.69	192	DRB1*07:01	227	241	QRGSSMVLFSPPVI	48.94
139	DRB1*01:24	126	140	GGYMLGSAMSRPIIH	37.21	193	DRB1*09:01	127	141	GYMLGSAMSRPIIHF	14.17
140	DRB1*01:24	128	142	YMLGSAMSRPIIHFG	39.57	194	DRB1*09:01	126	140	GGYMLGSAMSRPIIH	15.03
141	DRB1*01:24	232	246	MVLFSPPVILLISF	40.00	195	DRB1*09:01	125	139	LGGYMLGSAMSRPII	15.74
142	DRB1*01:24	225	239	YYQRGSSMVLFSPP	46.98	196	DRB1*09:01	223	237	QAYYQRGSSMVLFS	19.76
143	DRB1*01:29	106	120	KTNMKHMAGAAAAGA	13.48	197	DRB1*09:01	222	236	SQAYYQRGSSMVLFS	20.12
144	DRB1*01:29	223	237	QAYYQRGSSMVLFS	16.08	198	DRB1*09:01	128	142	YMLGSAMSRPIIHFG	20.37
145	DRB1*01:29	105	119	PKTNMKHMAGAAAAG	16.80	199	DRB1*09:01	221	235	ESQAYYQRGSSMVLFS	22.50
146	DRB1*01:29	222	236	SQAYYQRGSSMVLFS	18.18	200	DRB1*09:01	124	138	GLGGYMLGSAMSRPI	23.44
147	DRB1*01:29	104	118	KPKTNMKHMAGAAAA	19.55	201	DRB1*09:01	229	243	GSSMVLFSPPVILL	27.57
148	DRB1*01:29	107	121	TNMKHMAGAAAAGAV	19.66	202	DRB1*09:01	230	244	SSMVLFSPPVILLI	27.99
149	DRB1*01:29	127	141	GYMLGSAMSRPIIHF	23.35	203	DRB1*09:01	220	234	RESQAYYQRGSSMVL	29.39
150	DRB1*01:29	224	238	AYYQRGSSMVLFS	23.60	204	DRB1*09:01	224	238	AYYQRGSSMVLFS	30.37
151	DRB1*01:29	221	235	ESQAYYQRGSSMVLFS	24.51	205	DRB1*09:01	231	245	SMVLFSPPVILLIS	31.07
152	DRB1*01:29	108	122	NMKHMAGAAAAGAVV	24.56	206	DRB1*09:01	228	242	RGSSMVLFSPPVIL	32.28
153	DRB1*01:29	230	244	SSMVLFSPPVILLI	24.91	207	DRB1*09:01	232	246	MVLFSPPVILLISF	44.53
154	DRB1*01:29	231	245	SMVLFSPPVILLIS	25.70	208	DRB1*09:01	129	143	MLGSAMSRPIIHFGS	47.82
155	DRB1*01:29	229	243	GSSMVLFSPPVILL	25.90	209	DRB1*10:01	106	120	KTNMKHMAGAAAAGA	30.07
156	DRB1*01:29	126	140	GGYMLGSAMSRPIIH	27.73	210	DRB1*10:01	104	118	KPKTNMKHMAGAAAA	31.48
157	DRB1*01:29	125	139	LGGYMLGSAMSRPII	30.43	211	DRB1*10:01	125	139	LGGYMLGSAMSRPII	32.36
158	DRB1*01:29	128	142	YMLGSAMSRPIIHFG	35.49	212	DRB1*10:01	108	122	NMKHMAGAAAAGAVV	33.99
159	DRB1*01:29	232	246	MVLFSPPVILLISF	37.79	213	DRB1*10:01	124	138	GLGGYMLGSAMSRPI	34.28

214	DRB1*10:01	107	121	TNMKHMAGAAAAGAV	34.34	268	DRB1*15:01	227	241	QRGSSMVLFSPPVI	11.59
215	DRB1*10:01	105	119	PKTNMKHMAGAAAAG	35.27	269	DRB1*15:01	231	245	SMVLFSPPVILLIS	16.16
216	DRB1*10:01	223	237	QAYYQRGSSMVLFS	37.37	270	DRB1*15:01	226	240	YQRGSSMVLFSPPV	26.85
217	DRB1*10:01	230	244	SSMVLFSPPVILLI	37.51	271	DRB1*15:02	229	243	GSSMVLFSPPVILL	15.69
218	DRB1*10:01	231	245	SMVLFSPPVILLIS	39.19	272	DRB1*15:02	230	244	SSMVLFSPPVILLI	18.32
219	DRB1*10:01	229	243	GSSMVLFSPPVILL	40.56	273	DRB1*15:02	228	242	RGSSMVLFSPPVIL	19.33
220	DRB1*10:01	222	236	SQAYYQRGSSMVLFS	42.71	274	DRB1*15:02	227	241	QRGSSMVLFSPPVI	27.71
221	DRB1*10:01	126	140	GGYMLGSAMSRPIIH	43.38	275	DRB1*15:02	231	245	SMVLFSPPVILLIS	31.93
222	DRB1*10:01	127	141	GYMLGSAMSRPIHF	48.02	276	DRB1*15:03	229	243	GSSMVLFSPPVILL	17.57
223	DRB1*11:02	229	243	GSSMVLFSPPVILL	40.89	277	DRB1*15:03	230	244	SSMVLFSPPVILLI	19.61
224	DRB1*11:02	230	244	SSMVLFSPPVILLI	47.89	278	DRB1*15:03	228	242	RGSSMVLFSPPVIL	21.19
225	DRB1*11:14	230	244	SSMVLFSPPVILLI	16.54	279	DRB1*15:03	227	241	QRGSSMVLFSPPVI	28.49
226	DRB1*11:14	229	243	GSSMVLFSPPVILL	17.72	280	DRB1*15:03	231	245	SMVLFSPPVILLIS	29.05
227	DRB1*11:14	231	245	SMVLFSPPVILLIS	20.32	281	DRB1*15:06	229	243	GSSMVLFSPPVILL	7.85
228	DRB1*11:14	228	242	RGSSMVLFSPPVIL	22.37	282	DRB1*15:06	228	242	RGSSMVLFSPPVIL	8.91
229	DRB1*11:14	232	246	MVLFSPPVILLISF	36.97	283	DRB1*15:06	230	244	SSMVLFSPPVILLI	9.27
230	DRB1*11:14	227	241	QRGSSMVLFSPPVI	45.24	284	DRB1*15:06	227	241	QRGSSMVLFSPPVI	11.59
231	DRB1*11:65	229	243	GSSMVLFSPPVILL	40.89	285	DRB1*15:06	231	245	SMVLFSPPVILLIS	16.16
232	DRB1*11:65	230	244	SSMVLFSPPVILLI	47.89	286	DRB1*15:06	226	240	YQRGSSMVLFSPPV	26.85
233	DRB1*13:01	229	243	GSSMVLFSPPVILL	40.89	287	DRB1*15:07	229	243	GSSMVLFSPPVILL	11.44
234	DRB1*13:01	230	244	SSMVLFSPPVILLI	47.89	288	DRB1*15:07	228	242	RGSSMVLFSPPVIL	13.25
235	DRB1*13:02	230	244	SSMVLFSPPVILLI	16.54	289	DRB1*15:07	230	244	SSMVLFSPPVILLI	13.77
236	DRB1*13:02	229	243	GSSMVLFSPPVILL	17.72	290	DRB1*15:07	227	241	QRGSSMVLFSPPVI	18.01
237	DRB1*13:02	231	245	SMVLFSPPVILLIS	20.32	291	DRB1*15:07	231	245	SMVLFSPPVILLIS	23.33
238	DRB1*13:02	228	242	RGSSMVLFSPPVIL	22.37	292	DRB1*15:07	226	240	YQRGSSMVLFSPPV	42.03
239	DRB1*13:02	232	246	MVLFSPPVILLISF	36.97	293	DRB1*15:15	229	243	GSSMVLFSPPVILL	19.30
240	DRB1*13:02	227	241	QRGSSMVLFSPPVI	45.24	294	DRB1*15:15	228	242	RGSSMVLFSPPVIL	22.90
241	DRB1*13:23	230	244	SSMVLFSPPVILLI	16.54	295	DRB1*15:15	230	244	SSMVLFSPPVILLI	23.51
242	DRB1*13:23	229	243	GSSMVLFSPPVILL	17.72	296	DRB1*15:15	227	241	QRGSSMVLFSPPVI	32.69
243	DRB1*13:23	231	245	SMVLFSPPVILLIS	20.32	297	DRB1*15:15	231	245	SMVLFSPPVILLIS	39.44
244	DRB1*13:23	228	242	RGSSMVLFSPPVIL	22.37	298	DRB1*15:37	229	243	GSSMVLFSPPVILL	14.70
245	DRB1*13:23	232	246	MVLFSPPVILLISF	36.97	299	DRB1*15:37	228	242	RGSSMVLFSPPVIL	17.96
246	DRB1*13:23	227	241	QRGSSMVLFSPPVI	45.24	300	DRB1*15:37	230	244	SSMVLFSPPVILLI	18.02
247	DRB1*13:96	230	244	SSMVLFSPPVILLI	27.77	301	DRB1*15:37	227	241	QRGSSMVLFSPPVI	26.69
248	DRB1*13:96	229	243	GSSMVLFSPPVILL	31.89	302	DRB1*15:37	231	245	SMVLFSPPVILLIS	33.36
249	DRB1*13:96	231	245	SMVLFSPPVILLIS	32.08	303	DRB1*16:01	229	243	GSSMVLFSPPVILL	44.40
250	DRB1*13:96	228	242	RGSSMVLFSPPVIL	45.95	304	DRB1*16:02	229	243	GSSMVLFSPPVILL	24.00
251	DRB1*13:97	230	244	SSMVLFSPPVILLI	16.54	305	DRB1*16:02	228	242	RGSSMVLFSPPVIL	28.05
252	DRB1*13:97	229	243	GSSMVLFSPPVILL	17.72	306	DRB1*16:02	230	244	SSMVLFSPPVILLI	31.26
253	DRB1*13:97	231	245	SMVLFSPPVILLIS	20.32	307	DRB1*16:02	227	241	QRGSSMVLFSPPVI	40.22
254	DRB1*13:97	228	242	RGSSMVLFSPPVIL	22.37	308	DRB1*16:02	223	237	QAYYQRGSSMVLFS	42.04
255	DRB1*13:97	232	246	MVLFSPPVILLISF	36.97	309	DRB1*16:02	222	236	SQAYYQRGSSMVLFS	44.81
256	DRB1*13:97	227	241	QRGSSMVLFSPPVI	45.24	310	DRB1*16:05	229	243	GSSMVLFSPPVILL	30.97
257	DRB1*14:01	230	244	SSMVLFSPPVILLI	46.08	311	DRB1*16:05	230	244	SSMVLFSPPVILLI	36.81
258	DRB1*14:01	229	243	GSSMVLFSPPVILL	46.16	312	DRB1*16:05	228	242	RGSSMVLFSPPVIL	41.62
259	DRB1*14:32	229	243	GSSMVLFSPPVILL	31.39	313	DRB1*16:09	229	243	GSSMVLFSPPVILL	37.42
260	DRB1*14:32	230	244	SSMVLFSPPVILLI	31.90	314	DRB1*16:09	230	244	SSMVLFSPPVILLI	45.49
261	DRB1*14:32	231	245	SMVLFSPPVILLIS	38.51	315	DRB1*16:09	228	242	RGSSMVLFSPPVIL	49.59
262	DRB1*14:32	228	242	RGSSMVLFSPPVIL	38.61						
263	DRB1*14:54	230	244	SSMVLFSPPVILLI	46.08						
264	DRB1*14:54	229	243	GSSMVLFSPPVILL	46.16						
265	DRB1*15:01	229	243	GSSMVLFSPPVILL	7.85						
266	DRB1*15:01	228	242	RGSSMVLFSPPVIL	8.91						
267	DRB1*15:01	230	244	SSMVLFSPPVILLI	9.27						

Table S5. Peptides (18-mer) of PRP^c binding with high affinity (IC₅₀ < 50 nM) to HLA-II molecules.

Index	Allele	Start	End	Peptide	min(IC ₅₀)
1	DRB1*01:01	104	121	KPKTNMKHMAGAAAAGAV	40.57
2	DRB1*01:01	106	123	KTNMKHMAGAAAAGAVVG	45.40
3	DRB1*01:01	105	122	PKTNMKHMAGAAAAGAVV	41.81
4	DRB1*01:01	103	120	SKPKTNMKHMAGAAAAGA	41.32
5	DRB1*01:18	104	121	KPKTNMKHMAGAAAAGAV	44.95
6	DRB1*01:18	106	123	KTNMKHMAGAAAAGAVVG	49.85
7	DRB1*01:18	105	122	PKTNMKHMAGAAAAGAVV	45.73
8	DRB1*01:18	103	120	SKPKTNMKHMAGAAAAGA	46.74
9	DRB1*01:20	104	121	KPKTNMKHMAGAAAAGAV	43.48
10	DRB1*01:20	106	123	KTNMKHMAGAAAAGAVVG	45.77
11	DRB1*01:20	105	122	PKTNMKHMAGAAAAGAVV	43.13
12	DRB1*01:20	103	120	SKPKTNMKHMAGAAAAGA	45.02
13	DRB1*15:01	229	246	GSSMVLFSPPVILLISF	32.76
14	DRB1*15:01	227	244	QRGSSMVLFSPPVILLI	30.91
15	DRB1*15:01	228	245	RGSSMVLFSPPVILLIS	31.08
16	DRB1*15:01	230	247	SSMVLFSPPVILLISFL	45.29
17	DRB1*15:01	226	243	YQRGSSMVLFSPPVILL	31.89
18	DRB1*15:01	225	242	YYQRGSSMVLFSPPVIL	45.47
19	DRB1*15:02	227	244	QRGSSMVLFSPPVILLI	49.16
20	DRB1*15:02	228	245	RGSSMVLFSPPVILLIS	49.60
21	DRB1*15:06	229	246	GSSMVLFSPPVILLISF	32.76
22	DRB1*15:06	227	244	QRGSSMVLFSPPVILLI	30.91
23	DRB1*15:06	228	245	RGSSMVLFSPPVILLIS	31.08
24	DRB1*15:06	230	247	SSMVLFSPPVILLISFL	45.29
25	DRB1*15:06	226	243	YQRGSSMVLFSPPVILL	31.89
26	DRB1*15:06	225	242	YYQRGSSMVLFSPPVIL	45.47

Table S6. Peptides (22-mer) of PRP^c binding with high affinity (IC₅₀ < 50 nM) to HLA-II molecules.

Index	Allele	Start	End	Peptide	min(IC ₅₀)
1	DRB1*01:01	120	141	AVVGGGLGGYMLGSAMSRPIIHF	41.34
2	DRB1*01:01	224	245	AYYQRGSSMVLFSPPVILLIS	44.62
3	DRB1*01:01	219	240	ERESQAYYQRGSSMVLFSPPV	33.36
4	DRB1*01:01	221	242	ESQAYYQRGSSMVLFSPPVIL	31.14
5	DRB1*01:01	119	140	GAVVGGGLGGYMLGSAMSRPIIH	48.17
6	DRB1*01:01	123	144	GGLGGYMLGSAMSRPIIHFGSD	41.93
7	DRB1*01:01	124	145	GLGGYMLGSAMSRPIIHFGSDY	44.44
8	DRB1*01:01	104	125	KPKTNMKHMAGAAAAGAVVGGL	11.43
9	DRB1*01:01	101	122	KPSKPKTNMKHMAGAAAAGAVV	11.19
10	DRB1*01:01	106	127	KTNMKHMAGAAAAGAVVGGLGG	13.15
11	DRB1*01:01	125	146	LGGYMLGSAMSRPIIHFGSDYE	49.95
12	DRB1*01:01	100	121	NKPSKPKTNMKHMAGAAAAGAV	11.51
13	DRB1*01:01	108	129	NMKHMAGAAAAGAVVGGLGGYM	47.22
14	DRB1*01:01	105	126	PKTNMKHMAGAAAAGAVVGGLG	11.99
15	DRB1*01:01	102	123	PSKPKTNMKHMAGAAAAGAVVG	11.07
16	DRB1*01:01	223	244	QAYYQRGSSMVLFSPPVILLI	33.97
17	DRB1*01:01	98	119	QWNKPSKPKTNMKHMAGAAAAG	16.89
18	DRB1*01:01	217	238	QYERESQAYYQRGSSMVLFSPP	34.65
19	DRB1*01:01	220	241	RESQAYYQRGSSMVLFSPPVI	31.87
20	DRB1*01:01	103	124	SKPKTNMKHMAGAAAAGAVVGG	11.16
21	DRB1*01:01	222	243	SQAYYQRGSSMVLFSPPVILL	31.05
22	DRB1*01:01	97	118	SQWNKPSKPKTNMKHMAGAAA	24.66
23	DRB1*01:01	107	128	TNMKHMAGAAAAGAVVGGLGGY	23.58
24	DRB1*01:01	216	237	TQYERESQAYYQRGSSMVLFS	36.19

25	DRB1*01:01	122	143	VGGLGGYMLGSAMSRPIIHFGS	40.95
26	DRB1*01:01	121	142	VVGGLGGYMLGSAMSRPIIHFG	40.91
27	DRB1*01:01	99	120	WNKPSKPNTNMKHMAGAAAAGA	12.11
28	DRB1*01:01	218	239	YERESQAYYQRGSSMVLFSPP	34.31
29	DRB1*01:02	104	125	KPKTNMKHMAGAAAAGAVVGGL	41.26
30	DRB1*01:02	101	122	KPSKPNTNMKHMAGAAAAGAVV	40.60
31	DRB1*01:02	106	127	KTNMKHMAGAAAAGAVVGGLGG	48.16
32	DRB1*01:02	100	121	NKPSKPNTNMKHMAGAAAAGAV	44.16
33	DRB1*01:02	105	126	PKTNMKHMAGAAAAGAVVGGLG	43.60
34	DRB1*01:02	102	123	PSKPNTNMKHMAGAAAAGAVVG	40.00
35	DRB1*01:02	103	124	SKPNTNMKHMAGAAAAGAVVGG	40.19
36	DRB1*01:02	99	120	WNKPSKPNTNMKHMAGAAAAGA	47.85
37	DRB1*01:18	117	138	AAGAVVGGLGGYMLGSAMSRPI	47.54
38	DRB1*01:18	118	139	AGAVVGGLGGYMLGSAMSRPII	41.12
39	DRB1*01:18	120	141	AVVGGLGGYMLGSAMSRPIIHF	36.04
40	DRB1*01:18	224	245	AYYQRGSSMVLFSPPVILLIS	30.74
41	DRB1*01:18	219	240	ERESQAYYQRGSSMVLFSPPV	32.42
42	DRB1*01:18	221	242	ESQAYYQRGSSMVLFSPPVIL	27.00
43	DRB1*01:18	119	140	GAVVGGLGGYMLGSAMSRPIIH	38.44
44	DRB1*01:18	123	144	GGLGGYMLGSAMSRPIIHFGSD	38.96
45	DRB1*01:18	124	145	GLGGYMLGSAMSRPIIHFGSDY	41.66
46	DRB1*01:18	229	250	GSSMVLFSPPVILLISFLIFL	41.31
47	DRB1*01:18	104	125	KPKTNMKHMAGAAAAGAVVGGL	14.24
48	DRB1*01:18	101	122	KPSKPNTNMKHMAGAAAAGAVV	13.96
49	DRB1*01:18	106	127	KTNMKHMAGAAAAGAVVGGLGG	16.27
50	DRB1*01:18	125	146	LGGYMLGSAMSRPIIHFGSDYE	47.23
51	DRB1*01:18	100	121	NKPSKPNTNMKHMAGAAAAGAV	14.52
52	DRB1*01:18	105	126	PKTNMKHMAGAAAAGAVVGGLG	14.91
53	DRB1*01:18	102	123	PSKPNTNMKHMAGAAAAGAVVG	13.83
54	DRB1*01:18	223	244	QAYYQRGSSMVLFSPPVILLI	26.13
55	DRB1*01:18	227	248	QRGSSMVLFSPPVILLISFLI	36.11
56	DRB1*01:18	98	119	QWNKPSKPNTNMKHMAGAAAAG	22.26
57	DRB1*01:18	217	238	QYERESQAYYQRGSSMVLFSPP	35.05
58	DRB1*01:18	220	241	RESQAYYQRGSSMVLFSPPVI	29.27
59	DRB1*01:18	228	249	RGSSMVLFSPPVILLISFLIF	37.85
60	DRB1*01:18	103	124	SKPNTNMKHMAGAAAAGAVVGG	13.92
61	DRB1*01:18	222	243	SQAYYQRGSSMVLFSPPVILL	25.74
62	DRB1*01:18	97	118	SQWNKPSKPNTNMKHMAGAAA	30.80
63	DRB1*01:18	107	128	TNMKHMAGAAAAGAVVGGLGGY	29.25
64	DRB1*01:18	216	237	TQYERESQAYYQRGSSMVLFS	36.58
65	DRB1*01:18	122	143	VGGLGGYMLGSAMSRPIIHFGS	37.89
66	DRB1*01:18	121	142	VVGGLGGYMLGSAMSRPIIHFG	37.31
67	DRB1*01:18	99	120	WNKPSKPNTNMKHMAGAAAAGA	15.41
68	DRB1*01:18	218	239	YERESQAYYQRGSSMVLFSPP	34.67
69	DRB1*01:18	226	247	YQRGSSMVLFSPPVILLISFL	34.87
70	DRB1*01:18	225	246	YYQRGSSMVLFSPPVILLISF	32.86
71	DRB1*01:20	117	138	AAGAVVGGLGGYMLGSAMSRPI	47.77
72	DRB1*01:20	118	139	AGAVVGGLGGYMLGSAMSRPII	45.70
73	DRB1*01:20	120	141	AVVGGLGGYMLGSAMSRPIIHF	38.50
74	DRB1*01:20	224	245	AYYQRGSSMVLFSPPVILLIS	41.77
75	DRB1*01:20	119	140	GAVVGGLGGYMLGSAMSRPIIH	42.92
76	DRB1*01:20	123	144	GGLGGYMLGSAMSRPIIHFGSD	41.96
77	DRB1*01:20	124	145	GLGGYMLGSAMSRPIIHFGSDY	44.18
78	DRB1*01:20	104	125	KPKTNMKHMAGAAAAGAVVGGL	14.27
79	DRB1*01:20	101	122	KPSKPNTNMKHMAGAAAAGAVV	14.23

80	DRB1*01:20	106	127	KTNMKHMAGAAAAGAVVGLGG	15.99
81	DRB1*01:20	125	146	LGGYMLGSAMSRPIIHFGSDYE	48.81
82	DRB1*01:20	100	121	NKPSKPKTNMKHMAGAAAAGAV	14.73
83	DRB1*01:20	105	126	PKTNMKHMAGAAAAGAVVGLG	14.82
84	DRB1*01:20	102	123	PSKPKTNMKHMAGAAAAGAVVG	13.99
85	DRB1*01:20	223	244	QAYYQRGSSMVLFSPPVILLI	43.10
86	DRB1*01:20	227	248	QRGSSMVLFSPPVILLISFLI	44.83
87	DRB1*01:20	98	119	QWNKPSKPKTNMKHMAGAAAAG	21.97
88	DRB1*01:20	228	249	RGSSMVLFSPPVILLISFLIF	47.52
89	DRB1*01:20	103	124	SKPKTNMKHMAGAAAAGAVVGG	14.02
90	DRB1*01:20	222	243	SQAYYQRGSSMVLFSPPVILL	45.27
91	DRB1*01:20	97	118	SQWNKPSKPKTNMKHMAGAAAA	30.52
92	DRB1*01:20	107	128	TNMKHMAGAAAAGAVVGLGGY	27.16
93	DRB1*01:20	122	143	VGGLGGYMLGSAMSRPIIHFGS	40.90
94	DRB1*01:20	121	142	VVGGLGGYMLGSAMSRPIIHFG	39.96
95	DRB1*01:20	99	120	WNKPSKPKTNMKHMAGAAAAGA	15.77
96	DRB1*01:20	226	247	YQRGSSMVLFSPPVILLISFL	43.10
97	DRB1*01:20	225	246	YYQRGSSMVLFSPPVILLISF	41.87
98	DRB1*01:24	221	242	ESQAYYQRGSSMVLFSPPVIL	48.20
99	DRB1*01:24	104	125	KPKTNMKHMAGAAAAGAVVGLL	31.39
100	DRB1*01:24	101	122	KPSKPKTNMKHMAGAAAAGAVV	29.98
101	DRB1*01:24	106	127	KTNMKHMAGAAAAGAVVGLGG	37.76
102	DRB1*01:24	100	121	NKPSKPKTNMKHMAGAAAAGAV	31.40
103	DRB1*01:24	105	126	PKTNMKHMAGAAAAGAVVGLG	33.54
104	DRB1*01:24	102	123	PSKPKTNMKHMAGAAAAGAVVG	29.82
105	DRB1*01:24	223	244	QAYYQRGSSMVLFSPPVILLI	48.34
106	DRB1*01:24	103	124	SKPKTNMKHMAGAAAAGAVVGG	30.27
107	DRB1*01:24	222	243	SQAYYQRGSSMVLFSPPVILL	46.37
108	DRB1*01:24	99	120	WNKPSKPKTNMKHMAGAAAAGA	33.68
109	DRB1*01:29	104	125	KPKTNMKHMAGAAAAGAVVGLL	32.22
110	DRB1*01:29	101	122	KPSKPKTNMKHMAGAAAAGAVV	30.89
111	DRB1*01:29	106	127	KTNMKHMAGAAAAGAVVGLGG	38.28
112	DRB1*01:29	100	121	NKPSKPKTNMKHMAGAAAAGAV	33.05
113	DRB1*01:29	105	126	PKTNMKHMAGAAAAGAVVGLG	34.45
114	DRB1*01:29	102	123	PSKPKTNMKHMAGAAAAGAVVG	30.52
115	DRB1*01:29	103	124	SKPKTNMKHMAGAAAAGAVVGG	30.97
116	DRB1*01:29	99	120	WNKPSKPKTNMKHMAGAAAAGA	36.40
117	DRB1*04:01	167	188	DEYSNQNNFVHDCVNITIKQHT	47.78
118	DRB1*04:01	168	189	EYSNQNNFVHDCVNITIKQHTV	48.64
119	DRB1*04:01	166	187	MDEYSNQNNFVHDCVNITIKQH	48.26
120	DRB1*04:72	167	188	DEYSNQNNFVHDCVNITIKQHT	45.08
121	DRB1*04:72	168	189	EYSNQNNFVHDCVNITIKQHTV	45.25
122	DRB1*04:72	166	187	MDEYSNQNNFVHDCVNITIKQH	45.31
123	DRB1*04:72	165	186	PMDEYSNQNNFVHDCVNITIKQ	47.27
124	DRB1*04:72	170	191	SNQNNFVHDCVNITIKQHTVTT	49.79
125	DRB1*04:72	169	190	YSNQNNFVHDCVNITIKQHTVT	46.82
126	DRB1*07:01	120	141	AVVGLGGYMLGSAMSRPIIHF	43.75
127	DRB1*07:01	224	245	AYYQRGSSMVLFSPPVILLIS	43.35
128	DRB1*07:01	221	242	ESQAYYQRGSSMVLFSPPVIL	45.20
129	DRB1*07:01	123	144	GGLGGYMLGSAMSRPIIHFGSD	48.94
130	DRB1*07:01	223	244	QAYYQRGSSMVLFSPPVILLI	41.42
131	DRB1*07:01	222	243	SQAYYQRGSSMVLFSPPVILL	42.15
132	DRB1*07:01	122	143	VGGLGGYMLGSAMSRPIIHFGS	46.44
133	DRB1*07:01	121	142	VVGGLGGYMLGSAMSRPIIHFG	44.77
134	DRB1*07:01	225	246	YYQRGSSMVLFSPPVILLISF	46.96
135	DRB1*09:01	120	141	AVVGLGGYMLGSAMSRPIIHF	39.69

136	DRB1*09:01	123	144	GGLGGYMLGSAMSRPIIHFGSD	43.50
137	DRB1*09:01	124	145	GLGGYMLGSAMSRPIIHFGSDY	46.63
138	DRB1*09:01	122	143	VGGLGGYMLGSAMSRPIIHFGS	41.48
139	DRB1*09:01	121	142	VVGGLGGYMLGSAMSRPIIHFG	40.27
140	DRB1*10:01	104	125	KPKTNMKHMAGAAAAGAVVGGL	35.74
141	DRB1*10:01	101	122	KPSKPKTNMKHMAGAAAAGAVV	36.27
142	DRB1*10:01	106	127	KTNMKHMAGAAAAGAVVGGLGG	39.84
143	DRB1*10:01	100	121	NKPSKPKTNMKHMAGAAAAGAV	39.49
144	DRB1*10:01	105	126	PKTNMKHMAGAAAAGAVVGGLG	37.21
145	DRB1*10:01	102	123	PSKPKTNMKHMAGAAAAGAVVG	35.16
146	DRB1*10:01	103	124	SKPKTNMKHMAGAAAAGAVVGG	35.11
147	DRB1*10:01	99	120	WKNPSKPKTNMKHMAGAAAAGA	41.60
148	DRB1*15:01	224	245	AYYQRGSSMVLFSPPVILLIS	14.84
149	DRB1*15:01	221	242	ESQAYYQRGSSMVLFSPPVIL	18.97
150	DRB1*15:01	229	250	GSSMVLFSPPVILLISFLIFL	19.47
151	DRB1*15:01	223	244	QAYYQRGSSMVLFSPPVILLI	15.12
152	DRB1*15:01	227	248	QRGSSMVLFSPPVILLISFLI	15.70
153	DRB1*15:01	220	241	RESQAYYQRGSSMVLFSPPVI	28.96
154	DRB1*15:01	228	249	RGSSMVLFSPPVILLISFLIF	16.90
155	DRB1*15:01	222	243	SQAYYQRGSSMVLFSPPVILL	15.82
156	DRB1*15:01	230	251	SSMVLFSPPVILLISFLIFLI	34.93
157	DRB1*15:01	226	247	YQRGSSMVLFSPPVILLISFL	15.10
158	DRB1*15:01	225	246	YYQRGSSMVLFSPPVILLISF	14.84
159	DRB1*15:02	224	245	AYYQRGSSMVLFSPPVILLIS	27.59
160	DRB1*15:02	221	242	ESQAYYQRGSSMVLFSPPVIL	35.12
161	DRB1*15:02	229	250	GSSMVLFSPPVILLISFLIFL	36.74
162	DRB1*15:02	223	244	QAYYQRGSSMVLFSPPVILLI	27.35
163	DRB1*15:02	227	248	QRGSSMVLFSPPVILLISFLI	29.79
164	DRB1*15:02	228	249	RGSSMVLFSPPVILLISFLIF	32.11
165	DRB1*15:02	222	243	SQAYYQRGSSMVLFSPPVILL	28.26
166	DRB1*15:02	226	247	YQRGSSMVLFSPPVILLISFL	28.60
167	DRB1*15:02	225	246	YYQRGSSMVLFSPPVILLISF	27.92
168	DRB1*15:03	224	245	AYYQRGSSMVLFSPPVILLIS	37.74
169	DRB1*15:03	221	242	ESQAYYQRGSSMVLFSPPVIL	38.39
170	DRB1*15:03	229	250	GSSMVLFSPPVILLISFLIFL	45.63
171	DRB1*15:03	223	244	QAYYQRGSSMVLFSPPVILLI	37.90
172	DRB1*15:03	227	248	QRGSSMVLFSPPVILLISFLI	39.50
173	DRB1*15:03	220	241	RESQAYYQRGSSMVLFSPPVI	47.93
174	DRB1*15:03	228	249	RGSSMVLFSPPVILLISFLIF	41.74
175	DRB1*15:03	222	243	SQAYYQRGSSMVLFSPPVILL	38.19
176	DRB1*15:03	226	247	YQRGSSMVLFSPPVILLISFL	38.60
177	DRB1*15:03	225	246	YYQRGSSMVLFSPPVILLISF	38.05
178	DRB1*15:06	224	245	AYYQRGSSMVLFSPPVILLIS	14.84
179	DRB1*15:06	221	242	ESQAYYQRGSSMVLFSPPVIL	18.97
180	DRB1*15:06	229	250	GSSMVLFSPPVILLISFLIFL	19.47
181	DRB1*15:06	223	244	QAYYQRGSSMVLFSPPVILLI	15.12
182	DRB1*15:06	227	248	QRGSSMVLFSPPVILLISFLI	15.70
183	DRB1*15:06	220	241	RESQAYYQRGSSMVLFSPPVI	28.96
184	DRB1*15:06	228	249	RGSSMVLFSPPVILLISFLIF	16.90
185	DRB1*15:06	222	243	SQAYYQRGSSMVLFSPPVILL	15.82
186	DRB1*15:06	230	251	SSMVLFSPPVILLISFLIFLI	34.93
187	DRB1*15:06	226	247	YQRGSSMVLFSPPVILLISFL	15.10
188	DRB1*15:06	225	246	YYQRGSSMVLFSPPVILLISF	14.84
189	DRB1*15:07	224	245	AYYQRGSSMVLFSPPVILLIS	23.80
190	DRB1*15:07	221	242	ESQAYYQRGSSMVLFSPPVIL	29.09
191	DRB1*15:07	229	250	GSSMVLFSPPVILLISFLIFL	31.69

192	DRB1*15:07	223	244	QAYYQRGSSMVLFSPPVILLI	24.10
193	DRB1*15:07	227	248	QRGSSMVLFSPPVILLISFLI	25.66
194	DRB1*15:07	220	241	RESQAYYQRGSSMVLFSPPVI	42.37
195	DRB1*15:07	228	249	RGSSMVLFSPPVILLISFLIF	27.71
196	DRB1*15:07	222	243	SQAYYQRGSSMVLFSPPVILL	25.16
197	DRB1*15:07	226	247	YQRGSSMVLFSPPVILLISFL	24.57
198	DRB1*15:07	225	246	YYQRGSSMVLFSPPVILLISF	24.06
199	DRB1*15:15	224	245	AYYQRGSSMVLFSPPVILLIS	45.18
200	DRB1*15:15	221	242	ESQAYYQRGSSMVLFSPPVIL	44.17
201	DRB1*15:15	223	244	QAYYQRGSSMVLFSPPVILLI	42.55
202	DRB1*15:15	222	243	SQAYYQRGSSMVLFSPPVILL	41.70
203	DRB1*15:15	226	247	YQRGSSMVLFSPPVILLISFL	48.40
204	DRB1*15:15	225	246	YYQRGSSMVLFSPPVILLISF	46.89
205	DRB1*15:37	224	245	AYYQRGSSMVLFSPPVILLIS	27.30
206	DRB1*15:37	221	242	ESQAYYQRGSSMVLFSPPVIL	38.22
207	DRB1*15:37	229	250	GSSMVLFSPPVILLISFLIFL	38.22
208	DRB1*15:37	223	244	QAYYQRGSSMVLFSPPVILLI	27.71
209	DRB1*15:37	227	248	QRGSSMVLFSPPVILLISFLI	29.97
210	DRB1*15:37	228	249	RGSSMVLFSPPVILLISFLIF	32.76
211	DRB1*15:37	222	243	SQAYYQRGSSMVLFSPPVILL	29.46
212	DRB1*15:37	226	247	YQRGSSMVLFSPPVILLISFL	28.48
213	DRB1*15:37	225	246	YYQRGSSMVLFSPPVILLISF	27.75
214	DRB1*16:02	223	244	QAYYQRGSSMVLFSPPVILLI	47.36
215	DRB1*16:02	222	243	SQAYYQRGSSMVLFSPPVILL	46.94