UNIVERSIDADE D
COIMBRA

Isa Morais Penas

The Role of The Immune System in Polyglutamine Diseases:

immunogenicity prediction of ataxin-3 and its cleavage fragments

Dissertação no âmbito do Mestrado em Biotecnologia Farmacêutica, orientada pela Doutora Magda Matos Santana e pelo Professor Doutor Luís Pereira de Almeida e apresentada à Faculdade de Farmácia da Universidade de Coimbra

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A todos vós, muito obrigada.

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ABBREVIATIONS

AD	Alzheimer Disease
ALS	Amyotrophic Lateral Sclerosis
APC	Antigen-Presenting Cell
β2M	β-2-Microglubulin
BCR	B-Cell Receptor
CNS	Central Nervous System
DAMP	Damage Associated Molecular Pattern
DRPLA	Dentatorubral-Pallidoluysian Atrophy
GHRH	Growth Hormone Releasing Hormone
EPO	Erythropoietin
HD	Huntington's Disease
HLA	Human Leukocyte Antigen
HTT	Huntingtin
IEDB	Immune Epitope Database
Ig	Immunoglobulin
IL	Interleukins
IFN-β	Interferon-β
MHC	Major Histocompatibility Complex
MJD	Machado-Joseph Disease
NCBI	National Center for Biotechnology Information
NK	Natural Killer
PAMP	Pathogen Associated Molecular Pattern
PD	Parkinson Disease
Ply	Pneumolysin
PolyQ	Polyglutamine
PRRs	Pattern Recognition Receptors
SBMA	Spinobulbar Muscular Atrophy
SCA	Spinocerebellar Ataxia
TCR	T-Cell Receptor
Tc	T-Cytotoxic
Th	T-Helper
Treg	T-Regulatory
TNF	Tumor Necrosis Factor

UIM Ubiquitin Interaction Motif

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ABSTRACT

Machado Joseph Disease (MJD), also known as Spinocerebellar Ataxia Type 3 (SCA3), belongs to a group of nine inherited neurodegenerative disorders caused by expansions of CAG repeats encoding polyglutamine (PolyQ) tracts, named PolyQ diseases. MJD age of onset varies from 4 to 75 years of age and the pathogenesis of the disease results in clinical symptoms, mainly progressive ataxia which is usually the first reported symptom. There is no treatment able to stop or delay disease progression.

The proteolytic cleavage of the mutant ATXN3 protein results in the generation of PolyQ containing fragments that has been suggested as having a greater toxicity than the intact full-length protein. Proteolytic cleavage of proteins in PolyQ diseases has been proposed as a key event in the molecular pathogenesis, this is called the toxic fragment hypothesis

Recent studies shown that adaptive immune system influence the progression of several neurodegenerative diseases, activating inflammatory pathways and contribute to neurodegeneration. However, evidence regarding the role of adaptive immune system in PolyQ diseases is limited.

Thus, the main aim of this study was to perform an *in silico* analysis of the immunogenicity of wild-type and mutant ATXN3, and its derived cleavage fragments, to further perform *in vitro*/*in vivo* studies for understanding the role of adaptive immune system in MJD.

We carried out this study with the help of three bioinformatics tools, TepiTool to predict the ATXN3-MHC molecules binding affinity, CD4 T Cell Immunogenicity Prediction tool to identify possible immunogenic peptides and iProt-Sub to find potential cleavage sites of ATXN3.

Although ATXN3 does not appear to be high immunogenic, we were able to identify potential cleavage sites that leads to the formation of 23 fragments that could be immunogenic and cause the toxicity of the fragments. In conclusion, we were able to identify potential ATXN3 peptides for performing further in vitro studies to better understand the role of the immune system in MJD.

Keywords: MJD, ATXN3, immunogenicity, T-Cell, toxic fragments

RESUMO

A doença de Machado Joseph (MJD), também conhecida como ataxia espinocerebelar tipo 3 (SCA3), pertence a um grupo de nove doenças neurodegenerativas hereditárias, causadas pela expansão de repetições CAG que codificam tratos de poliglutaminas (PolyQ), denominadas doenças de PolyQ. O aparecimento da MJD pode variar de 4 a 75 anos e a patogénese da doença resulta em sintomas clínicos, sendo caracterizada principalmente por ataxia progressiva, que geralmente é o primeiro sintoma relatado. Atualmente, não existe tratamento capaz de curar ou retardar a progressão da doença.

A clivagem proteolítica da proteína PolyQ mutante, resulta na formação de fragmentos que contêm as PolyQ e que geram mais toxicidade do que na proteína intacta de comprimento total. Esta clivagem de proteínas nas doenças PolyQ foi proposta como um evento chave na patogénese molecular, o que originou a hipótese dos fragmentos tóxicos.

Estudos recentes mostram que o sistema imunitário adaptativo influencia a progressão de várias doenças neurodegenerativas, ativando vias inflamatórias e contribuindo para a neurodegeneração. No entanto, as evidências sobre o papel do sistema imunitário em doenças PolyQ são limitadas.

Assim, o objetivo principal deste estudo foi realizar uma análise *in silico* da imunogenicidade da ATXN3 *wild-type* e mutante, e os fragmentos derivados da sua clivagem, para realizar estudos *in vitro*/*in vivo* de forma a compreender o papel do sistema imunitário adaptativo na MJD.

Este estudo foi realizado com o auxílio de três ferramentas bioinformáticas, a TepiTool para prever a afinidade de ligação entre a ATXN3 e as moléculas MHC. A ferramenta de previsão de imunogenicidade de células T CD4 (*CD4 T Cell Imunogenicity Prediction Tool*) para identificar péptidos que pudessem ser imunogénicos. E a iProt-Sub para identificar proteases e encontrar potenciais locais de clivagem da ATXN3.

Embora a ATXN3 não pareça ser altamente imunogénica, fomos capazes de identificar potenciais locais de clivagem que levam à formação de 23 fragmentos que podem ser imunogénicos e causar a toxicidade dos fragmentos. Concluindo, fomos capazes de identificar potenciais péptidos derivados da ATXN3, para a realização de estudos *in vitro* de forma a conseguir compreender qual o papel do sistema imunitário na MJD.

Palavras-chave: MJD, ATXN3, imunogenicidade, células T, fragmentos tóxicos

CHAPTER 1
INTRODUCTION

1.1. Polyglutamine Diseases

Polyglutamine (PolyQ) expansion diseases are dominant late-onset genetic disorders, except Spinobulbar Muscular Atrophy (SBMA) which is inherited in an X chromosome-linked recessive manner, characterized by an unstable trinucleotide CAG (cytosine–adenine–guanine) expansion in the codifying region of their respective associated genes (Fan *et al.*, 2014; Matos, Macedo-Ribeiro and Carvalho, 2011). Currently, there are nine known inherited neurodegenerative disorders caused by expansions of CAG repeats encoding PolyQ tracts. These include Huntington's Disease (HD), SBMA, Dentatorubral-Pallidoluysian Atrophy (DRPLA) and six autosomal dominant forms of Spinocerebellar Ataxias (SCAs) (SCA1, 2, 3, 6, 7, and 17) (Fan *et al.*, 2014; Matos, Macedo-Ribeiro and Carvalho, 2011).

Although PolyQ disorders share several important clinical and pathological characteristics, the proteins associated with each different disorder share no homology being structurally and functionally unrelated. The only common genetic feature is the pathogenic repeat expansion (Lieberman, Shakkottai and Albin, 2019).

There is evidence that shows a negative correlation between PolyQ tract expansion and the age of onset of the diseases, which means that a greater number of CAG repeats results in an earlier development. This fact shows the significance that the expanded PolyQ sequence has on the mechanisms leading to the disease(Fan *et al.*, 2014; Lieberman, Shakkottai and Albin, 2019; Matos, Macedo-Ribeiro and Carvalho, 2011). Furthermore, Burright *et al.* (1995) and Ikeda *et al.* (1996) in their studies have demonstrated that expression of the expanded PolyQ stretch alone, results in progressive degeneration of neurons and motor perturbation suggesting that the expanded PolyQ tract is sufficient to cause typical phenotypes of the PolyQ diseases (Burright *et al.*, 1995; Ikeda *et al.*, 1996; Takeuchi and Nagai, 2017).

Although the common mechanism between PolyQ diseases is the protein aggregation (see section 1.2.), the exact pathogenic mechanism for each disease is not fully understood. Nevertheless, data suggests that the disruption of the native protein functions correlated with the toxic gain of function conveyed by the expanded PolyQ stretch leads to the disruption of several common cellular processes contributing to pathology (Buijsen *et al.*, 2019).

Clinically all these diseases are characterized by a selective neuronal loss followed by physical and psychological complications. The features vary among the different diseases and the affected genes are unrelated. (Table 1 - Biologic features of PolyQ expansion diseases) (Lieberman, Shakkottai and Albin, 2019; Matos, Macedo-Ribeiro and Carvalho, 2011).

DISEASE	GENE	PROTEIN	CAG REPEATS	MAIN SITE OF PATHOGENESIS (See Figure 1)	CLINICAL FEATURES
HD	HTT	Huntingtin	9-36 37-160	Striatum, globus pallidus, substantia nigra	Chorea, dystonia, dementia
SBMA	AR	Androgen Receptor	9-36 38-62	Medulla oblongata, skeletal muscle	Weakness, bulbar symptom, fasciculations, tremors, gynecomastia
DRPLA	ATN1	Atrophin-1	9-36 38-62	White matter, globus pallidus, subthalamic nucleus, dentate nucleus	Myoclonus epilepsy, ataxia, chorea, dementia
SCA 1	ATXN1	Ataxin-1	6-44 39-82	Brainstem, spinal cord, purkinje cells, dentate nucleus	Ataxia, bulbar symptom, spasticity, polyneuropathy, cognitive impairment
SCA 2	ATXN2	Ataxin-2	13-31 32-500	Cerebellum, purkinje cells, pons, thalamus, substantia nigra	Ataxia, polyneuropathy, parkinsonism
SCA 3	ATXN3	Ataxin-3	10-44 60-87	Striatum, substantia nigra, cranial nerve motor nuclei	Ataxia, spasticity, polyneuropathy, diplopia, dystonia
SCA 6	CACNA1A	α1A-Volatage-Dependent Calcium Channel Subunit	4-18 19-33	Purkinje cells, dentate nucleus	Ataxia, dysarthria, down-beating nystagmus
SCA 7	ATXN7	Ataxin-7	4-35 37-306	Purkinje cells, pons, dentate nucleus, retina	Ataxia, retinal degeneration, ophthalmoplegia
SCA 17	TBP	TATA Box-Binding Protein	25-44 47-63	Cerebral cortex, striatum, purkinje cells	Ataxia, dementia, psychosis, seizures, extrapyramidal signs

Table 1 – Biologic features of PolyQ expansion diseases.

Adapted from Matos, Macedo-Ribeiro and Carvalho, 2011; Shao and Diamond, 2007; Stoyas and La Spada, 2018.

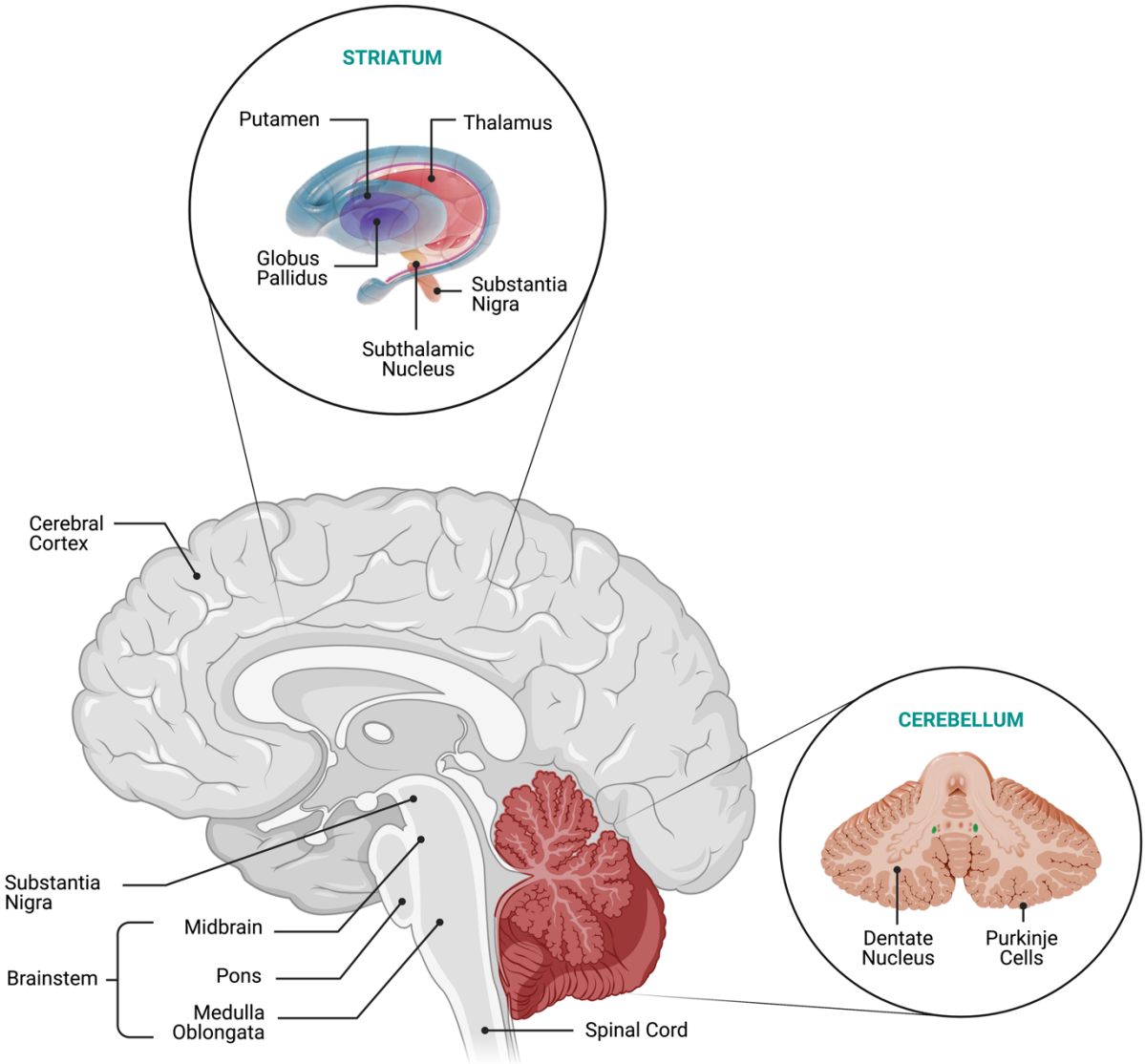

Figure 1 – Main sites of brain affected areas by PolyQ diseases.

Adapted from Moore, Agur and Dalley, 2013.

1.2. Protein Aggregation and Cleavage in PolyQ Diseases

One common characteristic of all PolyQ expansion diseases is the formation of protein aggregation or inclusion bodies. PolyQ expanded proteins are misfolded into multiple species of monomers, which may then form oligomers and incorporate into amorphous insoluble aggregates to form the pathogenic inclusions and cause toxicity (Matos, Macedo-Ribeiro and Carvalho, 2011; Stoyas and La Spada, 2018).

Having this in consideration, proteolytic cleavage of the mutant PolyQ protein results in the generation of PolyQ containing fragments that result in a greater toxicity than the intact full-length protein (Buijsen *et al.*, 2019).

Aggregates present in PolyQ diseases are present in the nucleus and cytoplasm, and contain parts of the respective disease proteins, ubiquitin, and several important homeostatic proteins (Havel, Li and Li, 2009).

The protein aggregation leads to transcriptional dysregulation, RNA toxicity, dysregulation of the ubiquitin proteasome system, and autophagy (Buijsen *et al.*, 2019).

The discovery of PolyQ aggregates triggered a persistent debate over the pathogenic significance of inclusion pathology in PolyQ disorders. One hypothesis suggests that the inclusions have pathogenic effects leading to the physical disruption of normal cellular functions and are important mediators of neuronal dysfunction and death. An alternative hypothesis supports the idea that PolyQ inclusions are nonpathogenic and exert a protective cellular mechanism moved against the toxicity of the misfolded expanded protein oligomers. There are evidence suggesting that both of this hypothesis could be correct and PolyQ inclusions exhibit both pathogenic and protective qualities, might be protective in early disease phases but pathogenic in later phases (Lieberman, Shakkottai and Albin, 2019).

A protease is an enzyme that catalyzes proteolysis and play essential roles in apoptosis. Proteolytic cleavage of proteins in PolyQ diseases has been proposed as a key event in the molecular pathogenesis, this is called the toxic fragment hypothesis. The base of this hypothesis is that the proteolytic cleavage of the mutant proteins liberates toxic fragments containing the expanded PolyQ. The accumulation of these toxic protein fragments may lead to the activation of additional proteolytic proteases and the eventual demise of the cell and neurodegeneration (Figure 2) (Weber *et al.*, 2017; Wellington *et al.*, 2000; Wellington and Hayden, 2000).

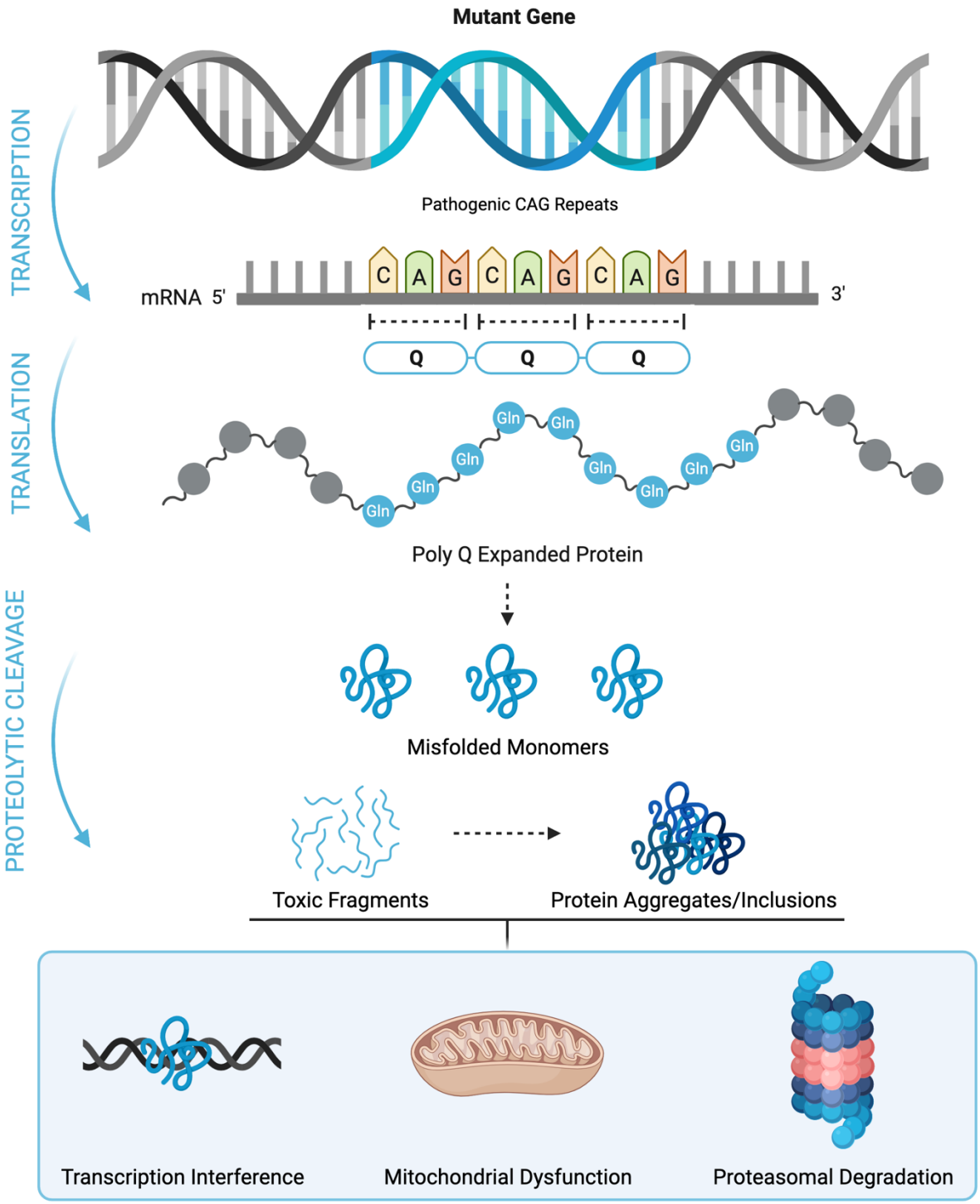

Figure 2 – Pathogenesis of PolyQ diseases.

The CAG repeated expansion tract in genomic DNA is transcribed into mRNA and translated into a protein containing an elongated glutamine (Q) tract. This PolyQ disease protein then misfolds due to the extended glutamine tract. Misfolded PolyQ-containing proteins can interfere with transcription, be cleaved by proteases to form toxic fragments or they may form oligomers that then form inclusions. Thus, it can disturb nuclear transcription, proteasomal degradation, and mitochondrial function. Adapted from Matos, Macedo-Ribeiro and Carvalho, 2011; Shao and Diamond, 2007.

The presence of proteolytically derived fragments of mutant proteins was reported for all PolyQ diseases and the toxic fragment hypothesis is supported by evidence from several studies. Caspases and calpains, proteases of the cysteine family, have been detected in numerous models of disease, and inhibition of their activity suppresses disease protein proteolysis and subsequent pathogenesis *in vitro* and *in vivo* (Berke *et al.*, 2004; Gafni and Ellerby, 2002; Goldberg *et al.*, 1996; Haacke *et al.*, 2006; Hubener *et al.*, 2013; Simões *et al.*, 2014; Weber *et al.*, 2014; Wellington *et al.*, 2000).

Goldberg *et al.* (1996) found that the presence of an expanded PolyQ tract increased the rate of cleavage of huntingtin by apopain-like proteases, therefore linking the HD mutation with a key pro-apoptotic cysteine protease.

In 1998, Wellington *et al.* (1998) demonstrated that huntingtin, atrophin-1, androgen receptor and ataxin-3 are cleaved by caspases and that the inhibition of cleavage by caspases reduces toxicity. Later, in 2000 they have shown that calpains can also cleave huntingtin and produce smaller fragments than those derived from caspase cleavage and therefore more toxic (Wellington *et al.*, 1998, 2000).

Berk *et al.* (2004) presented evidence that ataxin-3 is a substrate for caspase-mediated proteolysis in tissue culture and that the proteolysis of ataxin-3 increased formation of insoluble aggregates and that this aggregation can be suppressed by treatment with caspase inhibitors.

Haacke *et al.* (2006) has shown, *in vitro*, that calpain inhibition is sufficient to suppresses protein aggregation of expanded ataxin-3. The research of Hübener *et al.* (2012), *in vitro* and *in vivo*, indicates that ataxin-3 is cleaved by calpains, and that increased proteolytic cleavage of ataxin-3 results in a more severe and faster progressing neurological phenotype. To support this fact Simões *et al.* (2014) also demonstrated that calpain inhibition reduces ataxin-3 cleavage alleviating neuropathology and motor impairments in mouse model. Recently, Simões *et al.* (2021) also found, in a mouse model, that a fragment cleaved by calpain was the major responsible for the striatal degeneration.

These studies provide further support that the generation protease-mediated fragments of PolyQ-containing proteins contributes actively to disease progression. Although the only proteases mentioned were caspases and calpains, it is important to understand how proteins containing PolyQ tracts may be proteolytically cleaved, and which are the proteases involved.

1.3. Machado-Joseph Disease/SCA3

Machado-Joseph Disease (MJD, also known as SCA3, is a rare form of ataxia, however it is the most common SCA worldwide, with an overall average prevalence of 1 to 5 per 100 000

habitants, even though there are marked geographic differences in prevalence (Buijsen *et al.*, 2019; Scott *et al.*, 2020). It was first described as an autosomal dominant cerebellar ataxia in Portuguese immigrants in the United States; the family was native from Azores, descendants of William Machado and the disease was named after this family (Nakano, Dawson and Spence, 1972). Later, Rosenberg *et al.* (1976) described Joseph disease as a new genetic entity, also in a family with Azorean ancestry. The patients had similar symptoms, suggesting that they comprised a single genetic entity with variable phenotypes (Rosenberg *et al.*, 1976). Throughout the years more cases of the disease were described, and a consensus was made to name it Machado-Joseph Disease, since Coutinho and Andrade (1978) proposed that both diseases were variation of the same clinical disorder.

1.3.1. ATXN3 Gene and Protein

The MJD *locus* was mapped to chromosome 14 (between 14q24.3 and 14q32.1) in 1993 by Takiyama *et al.* (1993). In the next year, Kawaguchi *et al.* (1994) identified the gene MJD1/ATXN3 and mapped it to chromosome 14q32.1, this was supported in 2001 by Ichikawa *et al.* (2001) studies. This gene encodes for ataxin-3 (ATXN3) and comprises 11 exons, with the CAG repeat residing at the C-terminus, in exon 10. The trinucleotide repeats are quite unstable usually ranging from 12 to 44 triplets in healthy individuals and from 60 to 87 in MJD patients (Bettencourt and Lima, 2011; Ichikawa *et al.*, 2001; Kawaguchi *et al.*, 1994). Four different species of ATXN3 transcripts were reported by Ichikawa *et al.* (2001). These different mRNA species seem to result from differential splicing of exons 10 and 11 and alternative polyadenylation of exon 11 of ATXN3 gene.

ATXN3 protein was originally reported by Kawaguchi *et al.* (1994) to be composed of 339 amino acid residues plus the variable number of glutamine repeats with an estimated molecular weight of 40-42 kDa for normal individuals. The longest splice variant of ATXN3 possesses 376 amino acids (Albrecht *et al.*, 2004; Kawaguchi *et al.*, 1994). ATXN3 protein is ubiquitously expressed in neuronal and non-neuronal tissues, existing both in the cytoplasm and the nucleus of various cell types. However, Paulson *et al.* (1997) demonstrated that in neurons ATXN3 is predominantly a cytoplasmic protein. This protein belongs to the family of cysteine proteases and structurally it is composed of a highly conserved N-terminal Josephin domain (between amino acid 1 and 170) with de-ubiquitination activity. ATXN3 also contains a flexible C-terminal tail with 2 or 3 Ubiquitin Interaction Motifs (UIMs) that bind to polyubiquitylated proteins, and the polymorphic PolyQ tract (Figure 3) (Albrecht *et al.*, 2004; Tzvetkov and Breuer, 2007).

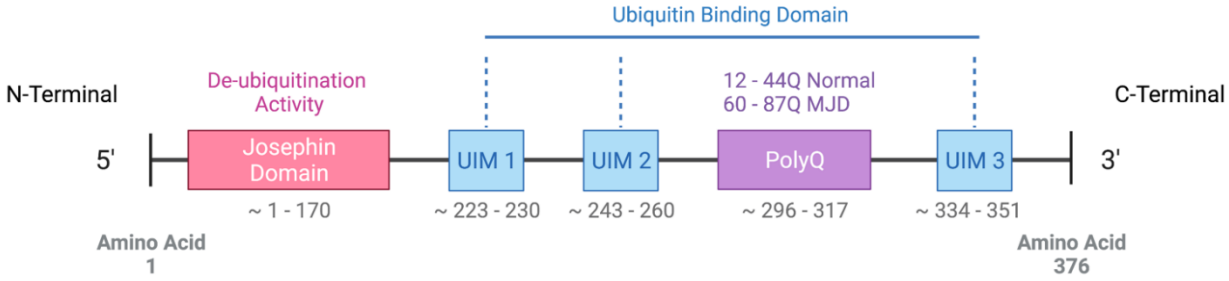

Figure 3 – Protein architecture of ATXN3.

ATXN3 consists of a de-ubiquitinating N-terminal Josephin domain (amino acid ~ 1-170), a flexible C-terminal tail containing 2 or 3 UIMs (amino acid ~ 223-230, ~ 243-260, ~ 334-351) and the PolyQ region (amino acid ~ 296-317). Adapted from (Albrecht *et al.*, 2004).

The physiologic role of ATXN3 is still unclear, however, it seems to take part in several cellular pathways. Some experimental evidence indicates that the normal function of ATXN3 involves transcriptional regulation, cytoskeletal organization, and DNA damage repair (Gao *et al.*, 2015; Li *et al.*, 2002; Nijman *et al.*, 2005; Rodrigues *et al.*, 2010). Additionally, Doss-Pepe *et al.* (2003) proposed that ATXN3 is an multiubiquitin chain recognition subunit, in the proteasome, that receives ubiquitinated substrates, and so play a role in the recognition of proteolytic substrates by the proteasome. Furthermore, it has been established that ATXN3 can be activated directly by ubiquitination, these facts suggest that ATXN3 normally participates in protein quality control pathways in the cell (Nóbrega *et al.*, 2018).

1.3.2. Mechanisms of Disease

Although in the past decade there has been extensive research into the MJD disease, the molecular mechanisms implicated in the pathogenesis of MJD are not yet fully understood. However, the PolyQ expansion seems to be the key factor to the disease. This expansion leads to conformational changes, conferring toxic properties to ATXN3 and cause the aggregation of the mutated protein (as shown in Figure 2 for PolyQ diseases), it also causes its nuclear accumulation in specific neuronal regions that are undergoing degeneration (Paulson *et al.*, 1997).

The mutant ATXN3 inclusions in MJD-affected brain regions are considered a hallmark of the disease (Paulson *et al.*, 1997). *In vivo* evidence acquired by Bichelmeier *et al.* (2007) suggest that aggregation is required to the establishment of the disease-associated dysfunction and degeneration. Moreover, the length of the PolyQ repeat is associated with increased levels of

aggregation. Nevertheless, there is no clear correlation between the presence of aggregates and neurodegeneration (Bichelmeier *et al.*, 2007).

The proteolytic cleavage of ATXN3 mutant protein was suggested to generate toxic fragments, as already referred for all PolyQ disorders, leading to cell dysfunction, and causing susceptibility to form aggregates (Haacke *et al.*, 2006; Simões *et al.*, 2014). There is also evidence of involvement of proteases, particularly calpains and caspases, in ATXN3 cleavage. This suggests an important role of proteolytic enzymes in disease pathogenesis by cleavage-induced toxicity and perhaps subsequent initiation of an aggregation cascade (Berke *et al.*, 2004; Wellington *et al.*, 1998). The presence of insoluble ubiquitinated neuronal inclusions containing the mutant protein was proposed to originate an impairment in the axonal transport as well as in the nuclear function (Evers, Toonen and Roon-Mom, 2013).

Taking this in account ATXN3 is clearly involved in several mechanisms implicated in MJD pathogenesis, and there are several pathways from which mutant ATXN3 could originate toxicity (Figure 4) (Bichelmeier *et al.*, 2007; Evers, Toonen and Roon-Mom, 2013). These pathways comprise the proteolytic cleavage of mutant ATXN3, impairment of axonal transport, dysregulation of calcium homeostasis, dysfunction of mitochondria, dysregulation of transcription, ubiquitin-proteasome system dysfunction, and altered protein-protein interactions (reviewed in Nóbrega *et al.* 2018).

Although, the PolyQ expansion seems to be the key factor to the disease, recently *in vivo* models of another PolyQ disease (HD), demonstrated that untranslated transcripts with expanded CAG repeats lead to cell degeneration and dysfunction. This suggests that the PolyQ expanded tract can lead to the formation of an elongated CAG repeat mRNA, which can by itself cause toxicity (Evers, Toonen and Roon-Mom 2013; Martí, 2016). This may be an important fact to enhance the mechanisms behind MJD and other PolyQ diseases.

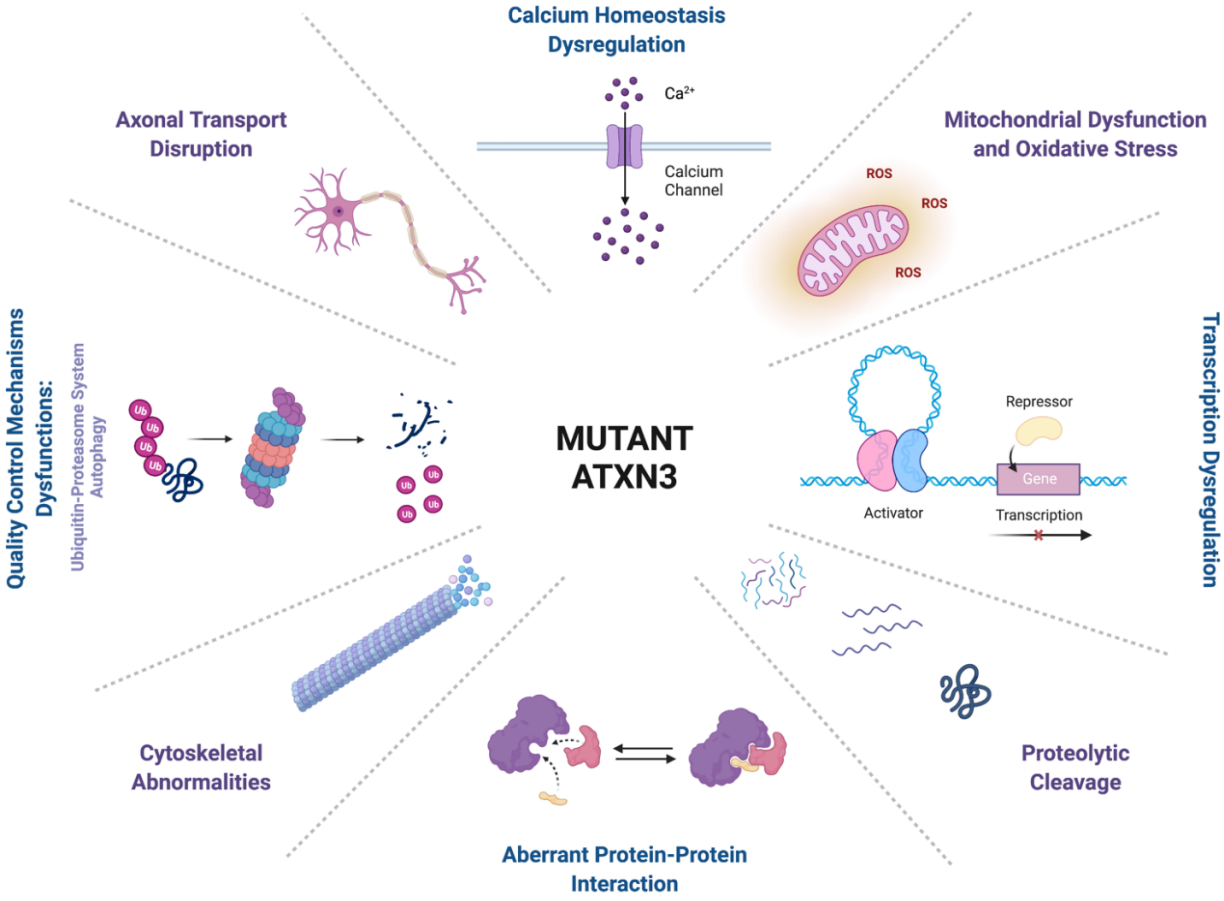

Figure 4 – Mechanisms implicated in the pathogenesis of MJD.

The PolyQ repeat in mutant ATXN3 triggers conformational changes, leading to a misfolded protein that confers toxicity. The proteolytic cleavage originates fragments that are likely to aggregate and produce ubiquitinated inclusions, this may compromise protein interaction and quality control mechanisms such as autophagy and ubiquitin-proteasome system. It also can cause cytoskeletal abnormalities leading to the impairment in the axonal transport. Other consequences that can also be important to the pathogenesis of MJD are transcriptional dysregulation, mitochondrial dysfunction, and calcium homeostasis dysregulation. Adapted from Gatchel and Zoghbi, 2005; Nóbrega *et al.*, 2018.

1.3.3. Clinical Presentation and Neuropathology

MJD is highly pleomorphic, since it presents high variability at the age of onset, neurological signs, and degree of incapacity presented by different patients (Bettencourt and Lima, 2011).

Age of onset varies from 4 to 75 years of age, despite usually starts at 40 years, the mean survival time is 21 years (ranging from 7 to 29 years) after onset of disease (Bettencourt and Lima, 2011; Nakano, Dawson and Spence, 1972; Nóbrega *et al.*, 2018).

MJD pathogenesis results in clinical symptoms, it is mainly characterized by progressive ataxia which is usually the first reported symptom. However, corticospinal, and autonomic nervous system dysfunctions and neuropathy, and other general neuromuscular complications are also observed. These include dystonia, dysarthria, spasticity, amyotrophy, rigidity, fasciculations and other visual and motor disorders (Bettencourt and Lima, 2011; Matos, Macedo-Ribeiro and Carvalho, 2011; Rosenberg *et al.*, 1976).

The phenotype variability, that is, the diversity of clinical manifestations and the progressive nature of MJD make its clinical classification difficult and nowadays of limited clinical value to neurologists. Based on the age of disease onset and neurological signs Coutinho and Andrade (1978) systematized the disease phenotypes into three main clinical types. Some authors consider a type four, which is a rare presentation with parkinsonian features, and a type five was also proposed (Bettencourt and Lima, 2011; Coutinho and Andrade, 1978).

The progressive motor impairment in MJD results of neuropathological alterations, involving neuronal loss in several brain regions. Similarly, to the diversity of clinical symptoms the neurodegeneration also affects wide brain regions, such as cerebellum, basal ganglia, brainstem and spinal cord (Koeppen, 2018; Paulson *et al.*, 1997; Takiyama *et al.*, 1993).

The aggregates containing mutant ATXN3 tend to accumulate as NIIs (Neuronal Intranuclear Inclusions), these are particularly found in brainstem neurons, substantia nigra, thalamus, and in a lesser extent, in striatum. Apart from the mutant protein, NIIs also contain other proteins, including members of the cell quality control system, which may be mobilized against ATXN3 misfolding and aggregation (Paulson *et al.*, 1997; Schmidt *et al.*, 2002).

Autopsies of MJD patients revealed a reduced size of the cerebellum, possibly due to the shrinkage of the dentate nucleus. Consequently, the main ataxia-causing deficit resides in failure of glutamatergic output to thalamus, derived from the dentate nucleus failure. The autopsies also revealed lesion of the substantia nigra, neuronal loss in basal ganglia, and motor and non-motor nuclei of the brain stem and spinal cord (Koeppen, 2018).

Given ATXN3 ubiquitous expression, cellular expression of the disease gene is not itself sufficient to explain selective neuronal degeneration, suggesting that other cell-specific factors are involved in the restricted neuropathology observed in MJD (Paulson *et al.*, 1997). Breuer *et al.* (2010) suggest that nuclear aggregation may eventually escape the cytoplasmic quality control, resulting in aggregation in the nuclear compartment.

There have been episodic reports of reactive gliosis, increased expression of cytokines and proinflammatory chemokines, which are compatible with mutant ATXN3 induced changes in brain inflammatory mediators (Evert *et al.*, 2001, 2003; Huizinga *et al.*, 2009). Gonçalves *et al.* (2013) research revealed reactive gliosis and the loss of synaptic markers as precocious events in MJD. In addition, they propose a time-course of neuropathological features, whereas synaptotoxicity and astrogliosis are precocious modifications followed by microgliosis and neuronal dysfunction, appearance of NIIs and evident neuronal damage (Gonçalves *et al.*, 2013).

There is no cure for MJD, and the available treatments only relieve symptomatology and do not block disease progression. The understanding of pathogenic mechanisms that leads to disease and the pathways involved, are considerable factors to develop an adequate therapeutic.

1.4. Immune System

The immune system is a complex network of cells, chemicals and processes that function to protect against disease or other potentially damaging foreign bodies. Typically, the immune system is divided in two parallel and complementing activities, – innate and adaptive immunity - determined by the speed and specificity of the reaction. The innate immune system is the first line of defense, it is antigen-independent (nonspecific) toward a particular kind of pathogen. The adaptive immune system is antigen-dependent and antigen-specific (Marshall *et al.*, 2018; Porcelli, 2017; Storey and Jordan, 2008).

1.4.1. Innate Immune System

The innate immune system acts as the first line of host defense against pathogens and comprises four types of defensive barriers: anatomic (skin and mucous membrane), physiologic (temperature, pH, and chemical mediators), endocytic and phagocytic, and inflammatory. It has some constitutive mechanisms which means that they are continuously expressed and are not significantly modulated by the presence or absence of infection. Although not antigen-specific, the innate system is able to distinguish foreign molecules. The inducible mechanisms of innate immunity involve increased production of mediators and upregulation of effector functions that eliminate microorganisms (Marshall *et al.*, 2018; Porcelli, 2017; Storey and Jordan, 2008).

In innate immunity the Pattern Recognition Receptors (PRRs) allow a limited range of immune cells to detect and rapidly respond to pathogens that share common structures. The

Toll-Like Receptors (TLRs) are a type of PRR that are expressed on the membranes of leukocytes including dendritic cells, macrophages, Natural Killer (NK) cells and cells of the adaptive immunity, T and B cells, and non-immune cells (epithelial and endothelial cells) (Marshall *et al.*, 2018; Porcelli, 2017).

The targets of innate immune recognition are known as Pathogen Associated Molecular Patterns (PAMPs), since most pathogens contain PAMPs, this strategy allows the generation of at least partial immunity against most infections. Furthermore, the innate immune system can respond to host-derived molecules released by cells undergoing necrotic death, these molecules are generally named Damage Associated Molecular Patterns (DAMPs) (Marshall *et al.*, 2018; Porcelli, 2017).

The innate immune responses are much more rapid than adaptive because the expression of PRRs is not clonal and there is a rapid recruitment of immune cells to sites of infection and inflammation through the production of cytokines and chemokines. Rather than undergoing proliferation and expansion, as in the case of adaptive immune responses, when PRRs are engaged by recognition of their associated PAMPs or DAMPs, effector cells bearing the PRRs are triggered to perform their immune effector functions immediately (Marshall *et al.*, 2018; Porcelli, 2017; Storey and Jordan, 2008).

For initiating cell recruitment, the release of some vital inflammatory cytokines by macrophages is crucial: Tumor Necrosis Factor-Alpha (TNF-α) and Interleukins (ILs). Dysregulated production of these cytokines is regularly associated with autoimmune diseases, making them important therapeutic targets (Marshall *et al.*, 2018).

Although phagocytosis and cytokine/chemokine production are the two key effector functions of macrophages in the innate immune response, these cells also have a role in adaptive responses as Antigen-Presenting Cells (APCs) and as targets of the effector components of the cellular and humoral adaptive responses, being activated by T cell-derived cytokines and antibodies (Marshall *et al.*, 2018; Porcelli, 2017; Storey and Jordan, 2008).

The complement system is a biochemical cascade of events to identify and opsonize pathogens, with amplification stages, the activation of a single molecule leads to the generation of thousands of molecules. In the presence of a foreign substance the complement can be activated by three pathways: the classical by antigen-antibody reactions, the alternative by polysaccharides from yeasts, and gram-negative bacteria. This complement action of the innate immune response provides clearance against foreign bodies or substances, and it can also activate the adaptive immune response (Marshall *et al.*, 2018; Porcelli, 2017; Storey and Jordan, 2008).

Innate immune response comprises several cells including phagocytes (macrophages and neutrophils), NK cells, dendritic cells, mast cells, basophils, eosinophils, and innate lymphoid cells. Besides effector cells, innate immunity comprises soluble factors that can bind foreign molecules and help in their elimination (Marshall *et al.*, 2018).

1.4.2. Adaptive Immune System

Adaptive immunity is supported by the actions of innate immune response. When innate immunity is ineffective adaptive immunity is critical and acts. The adaptive immunity is a highly specific system, in contrast to those of the innate immune system, interact with the environmental agent in highly discriminative way. It comprises the generation of pathogen-specific immunologic effector pathways to recognize and respond to an individual antigen. Adaptive immunity has immunologic memory, the cells expressing a certain receptor after being exposed to a pathogen can persist in the host, providing the capacity to eliminate a specific pathogen in case of re-exposure. So adaptive immunity display specificity, heterogeneity, and memory (Bonilla and Oettgen, 2010; Marshall *et al.*, 2018; Storey and Jordan, 2008).

The adaptive immune system is composed of relatively small numbers of cells with specificity to recognize an individual immunogen, the responding cells must proliferate, forming a cell clone and differentiate into effector cells (Storey e Jordan, 2008).

The two major components of the adaptive immune system are humoral immunity and cell-mediated immunity. These responses are articulated by lymphocytes, more specifically by B- and T-cells (Bonilla and Oettgen, 2010; Marshall *et al.*, 2018; Sanchez-Trincado, Gomez-Perosanz and Reche, 2017).

An epitope, also known as antigenic determinant, is the region of an antigen that bind to an antibody, thereby eliciting either cellular or humoral immune response. Epitopes are usually non-self-proteins, in the case of autoimmune diseases sequences derived from host that can be recognized are also designated epitopes (Sanchez-Trincado, Gomez-Perosanz and Reche, 2017).

Cell-mediated immunity is a process carried out by T cells without the participation of antibody. T cells mature in the thymus and are activated to proliferate through the action of APCs, which includes dendritic cells, macrophages, and B cells. The receptor that is present on T cells surface, known as T Cell Receptor (TCR) enables the recognition of antigens when they are displayed on the surface of APCs bound to Major Histocompatibility Complex (MHC) molecules. The MHC or, in humans, the Human Leukocyte Antigen (HLA) is a gene complex, located on the short arm of chromosome 6, that encodes the MHC/HLA proteins in humans.

These are cell surface proteins essential for the acquired immune system to recognize foreign molecules, which determines histocompatibility. There are hundreds of allelic variants of HLAs, each of these variants has a distinct binding pattern. The peptides that bind to the class I or II molecules are different, and different HLAs have characteristic amino acid residue patterns in the binding peptide sequence. HLAs are divided in two main classes, class I and class II (Figure 5), and its main function is to present antigen to T cells (Bonilla and Oettgen, 2010; Choo, 2007; Marshall *et al.*, 2018; Sanchez-Trincado, Gomez-Perosanz and Reche, 2017; Storey and Jordan, 2008).

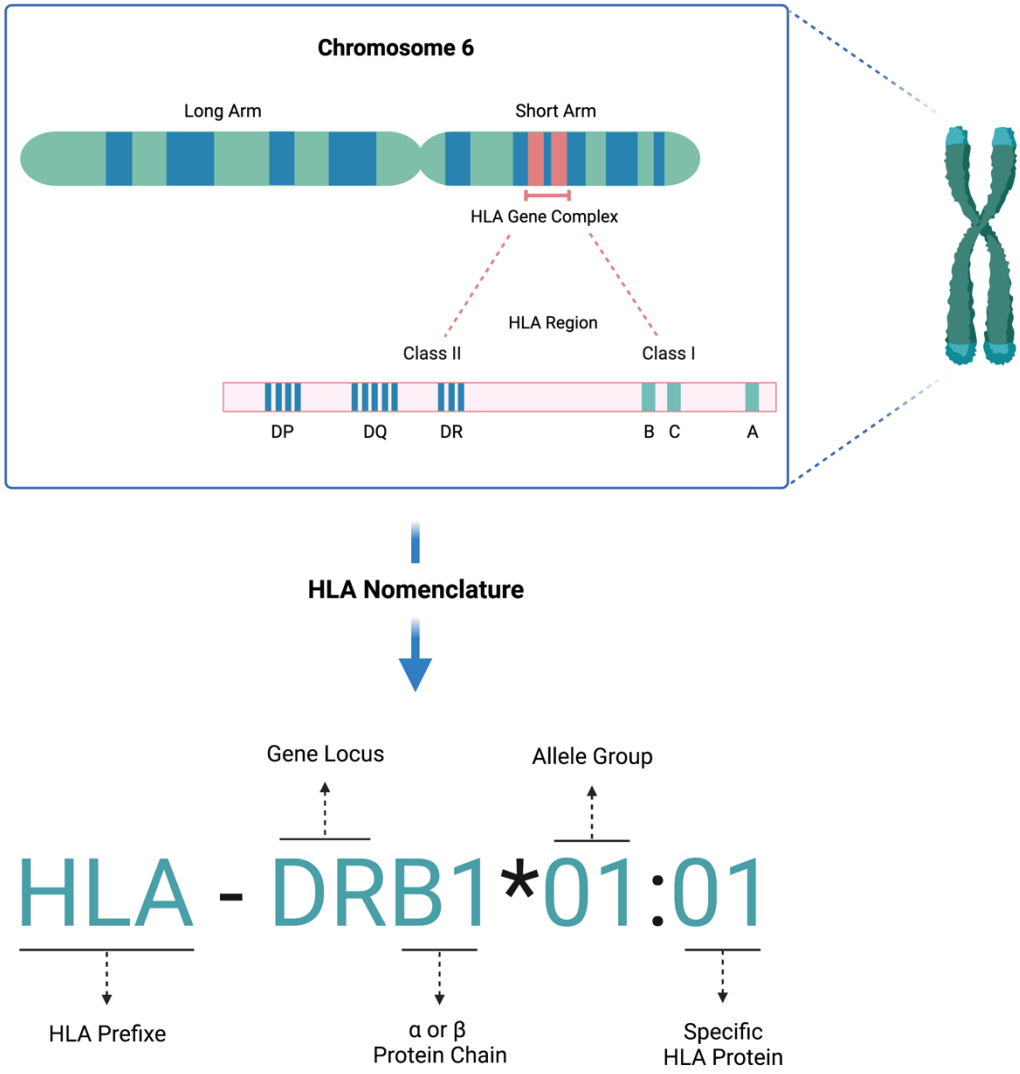

Figure 5 – HLA Complex and nomenclature.

HLA gene complex is localized on the short arm of chromosome 6, and it is divided into three regions. The class III region does not encode HLA molecules, it contains mainly genes for complement components and TNF. In this figure are only demonstrated the regions of class I and II. The class I region, comprises HLA-A, HLA-B and HLA-C genes that encode class I molecules. Class II has three regions, DP, DQ and DR, these regions consist of a series of sub-regions containing A and B genes, encoding α and β chains. The class II regions could have one or more A and B genes (Choo, 2007).

The expression of MHC-I and MHC-II is regulated by cytokines. T cell epitopes are presented by MHC-I and MHC-II, which means T cells recognize foreign antigens in the form of short peptides that have been processed and displayed on the cell surface bound to MHC-I or MHC-II molecules. MHC-I molecules are recognized by CD8 T cells and MHC-II molecules are recognized by CD4 T cells. Subsequently, there are CD8 and CD4 T cell epitopes. Following T CD8 epitope recognition T cells become T-cytotoxic (Tc), and CD4 T cells become T-helper (Th) or T-regulatory (Treg) (Figure 6) (Okeke and Uzonna, 2019; Sanchez-Trincado, Gomez-Perosanz and Reche, 2017).

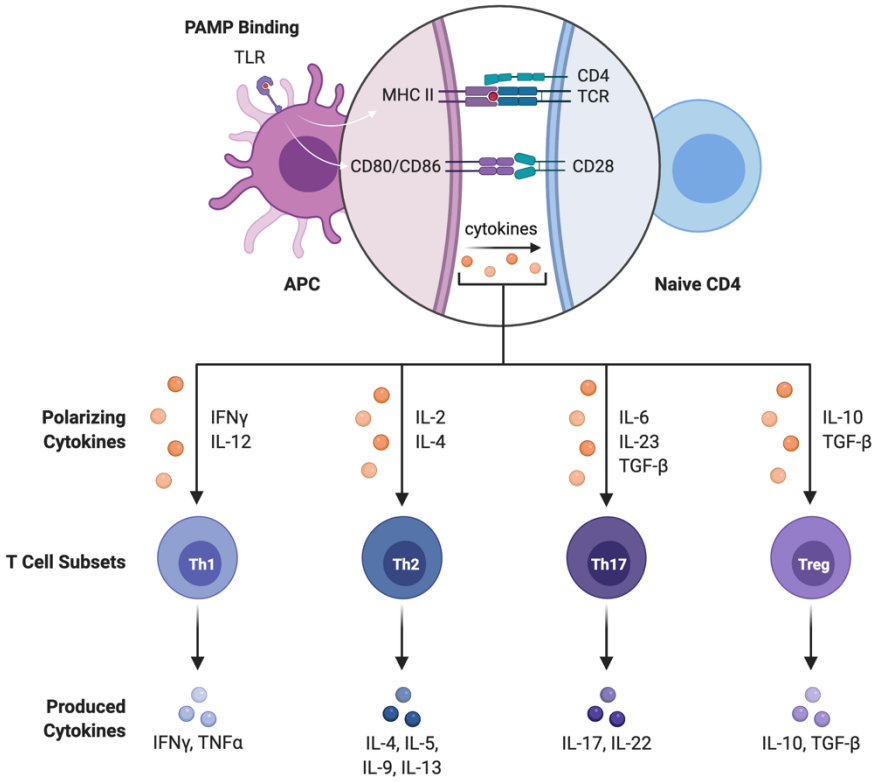

Figure 6 – CD4 T Cells Differentiation.

Class II cells recognize exogenously derived antigens, this expression is mainly restricted to APCs such as B-cells, macrophages, and dendritic cells, and activated T lymphocytes. Exogenous proteins are endocytosed, and the class II molecule-peptide complex is transported to the cell surface and recognized by the TCR of CD4+ cell. According to the type of polarizing cytokines, T cells will differentiate into Th or Treg, and will also produce characteristic cytokines (Choo, 2007; Okeke and Uzonna, 2019; Sanchez-Trincado, Gomez-Perosanz and Reche, 2017).

Th and Treg have roles in delayed hypersensitivity, antibody production, inflammation, and immunosuppression (or regulation). Tc releases vesicles containing perforin, which

punctures the target cell, and granzyme, which induces apoptosis, into the vicinity of infected cells, destroying them (Figure 7) (Okeke and Uzonna, 2019; Sanchez-Trincado, Gomez-Perosanz and Reche, 2017).

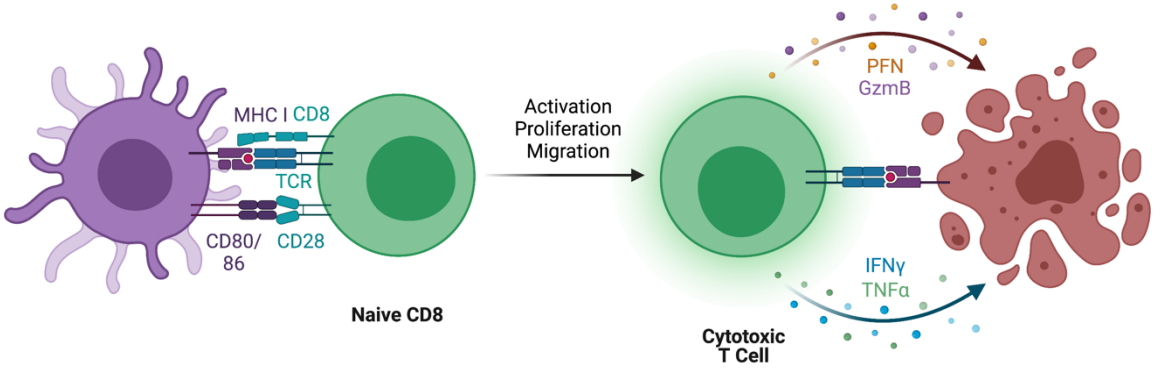

Figure 7 – CD8 T Cells Differentiation.

Class I T cells recognize endogenous antigens, the MHC-I peptide complex on the cell surface is recognized by the TCR of CD8 lymphocytes. Since CD8 T cell is activated, proliferates, and migrates to the infected local, producing granzyme and perforin to induce cell apoptosis, and cytokines (Choo, 2007).

T lymphocytes play an important role in both cell-mediated and humoral immune response of the adaptive immune system as they promote inflammation by cytokine production, helping B lymphocytes and regulating immunosuppressive responses (Okeke and Uzonna, 2019; Sanchez-Trincado, Gomez-Perosanz and Reche, 2017).

The humoral immunity is a process carried out by antibodies (immunoglobulin) produced by and secreted from B-cells and specifically binds extracellular antigen. B cells become activated and proliferate by antigen via B-Cell Receptors (BCR) and/or by PAMPs via their TLRs. Under signals of T cells and other cells, such as dendritic cells, B lymphocytes arise in the bone marrow to produce antigen specificity (antibodies). After developing in their primary lymphoid organs, they migrate to secondary lymphoid organs (lymph nodes and spleen) to capture circulating antigens from lymph and blood, often under signals provided by the influence of innate immune system and B cells. Regulated by an array of adhesion molecules and chemokine receptors, lymphocytes can then travel to many sites of the body to exert effector functions (Bonilla and Oettgen, 2010; Marshall *et al.*, 2018; Sanchez-Trincado, Gomez-Perosanz and Reche, 2017; Storey and Jordan, 2008).

A B cell epitope is the antigen portion binding to the immunoglobulin (Ig) or antibody. There are five major classes of Igs: IgM, IgG, IgA, IgD, and IgE, each differing in physical, chemical, and biologic properties. This Igs function as antibodies and have the property with the antigen (immunogen) that triggered their production. They have two extremely important

functions during the immune response: the antigen-recognition that confers its exquisite specificity to react with different molecular structures (the epitopes) and an effector function, by the interaction with other effector cells, phagocytic cells, and mediator molecules (for example, complement system) (Bonilla and Oettgen, 2010; Marshall *et al.*, 2018; Sanchez-Trincado, Gomez-Perosanz and Reche, 2017; Storey and Jordan, 2008).

1.4.3. Immune System in Neurodegenerative Diseases

Microglia and astrocytes are the principal components of the innate immune system in the Central Nervous System (CNS). Under physiological conditions, microglia is responsible to respond to neuronal damage and remove the damaged cells by phagocytosis. The cerebrospinal fluid-blood barrier, blood-brain barrier, and blood-spinal cord barrier along with the expression of immune regulatory molecules act to limit immune activation and immune-mediated damage in the CNS (Amor and Woodroofe, 2014; Rodrigues *et al.*, 2014).

In recent years the role of the immune system in neurodegenerative diseases such as Amyotrophic Lateral Sclerosis (ALS), Alzheimer's Disease (AD) and Parkinson Disease (PD) has become a major focus in scientific community. Since is known that both, nervous and immune systems change with age, resulting in a loss of regulation of immune responses in the healthy brain may be a contributing factor to neurodegeneration (Amor and Woodroofe, 2014; Ciccocioppo *et al.*, 2020).

There are several studies that link, in theory, neurodegeneration with the immune activation, assuming that innate and adaptive immune system are crucially implicated in neurodegenerative diseases (Baruch *et al.*, 2015; Golde and Miller, 2009; Schwartz and Deczkowska, 2016). The theory is that protein aggregates lead to activation of the innate immune system and posterior chronic inflammation and activation of the adaptive immune system leading to neurodegeneration (Ciccocioppo *et al.*, 2020).

Salminem *et al.* (2009) in AD and McGeer and McGeer (2002) in ALS, support the hypothesis that extracellular and intracellular protein aggregates could act like PAMPs leading to chronic innate immune activation by PRRs. Innate and adaptive immunity in neurodegenerative diseases was also described by Amor *et al.* (2010), innate immune responses can be triggered in different ways to remove dying and apoptosing neurons, infectious agents or altered proteins (aggregates). This can lead to chronic microglia activation and may lead to neuronal damage. It can also lead to the recruitment of cells of the adaptive immune system into the CNS, more precisely CD8 T cells, this cytotoxic T cells directly target neurons which express MHC-I molecules and contribute to neuronal damage or destruction (Amor *et al.*, 2010). This role of CD8 T cells in neurodegenerative diseases has been described

by Howe *et al.* (2007) in multiple sclerosis, by Medana *et al.* (2001) in neuritis and by Bauer, Bien and Lassmann (2002) in Rasmussen syndrome (rare disorder of the CNS characterized by chronic progressive inflammation).

As described in 1.4.2, the CD4 T cells can be subtyped into Th and Treg. Th cells were described by Huang *et al.* (2009) to provide both neuroprotection and neuronal loss since some produce pro-inflammatory cytokines conducing to neuroinflammation as well as indirectly up-regulate microglia-mediated neuroprotection. Normally Treg display the ability to attenuate neuroinflammation by suppression of effector T cells activation during chronic inflammation (Ciccocioppo *et al.*, 2020). In AD transgenic animal models, Dansokho *et al.* (2016) and Schwartz and Deczkowska (2016) proposed that the adaptive immune system is impaired and that Treg stimulation modulates brain inflammation in this disease. In PD for instance, Reynolds *et al.* (2009) have proposed the attenuation of microglial activation by Treg in early disease stages and with the progression of the disease regulatory mechanisms fail, inducing microglial apoptosis, favoring inflammation. These studies exhibit the important role of Treg in neuroinflammation (Dansokho *et al.*, 2016; Reynolds *et al.*, 2009; Schwartz and Deczkowska, 2016).

Regarding PolyQ diseases most of the information concerning immune response refers to HD, and the immune activation is found both at the CNS and in a peripheral level (blood) (Olejniczak, Urbanek and Krzyzosiak, 2015).

One of the similarities in PolyQ diseases is neuroinflammation characterized by the presence of microglial cells, which are a typical marker for innate immune activation in the CNS suggesting the involvement of inflammation in pathogenesis of the diseases. Once activated, microglia can modulate the inflammatory response and contribute to eliminate a primary source of inflammation. In contrast, it can also enhance inflammation, promoting neurotoxicity. This may influence the disease outcome as microglial heterogeneity is present in neurodegenerative diseases (Olejniczak, Urbanek and Krzyzosiak, 2015; Rodrigues *et al.*, 2014).

Shin *et al.* (2005) findings suggest that mutant huntingtin (HTT) may affect various functions of glial cells, mainly in astrocytes, including their production of chemokines and neurotrophic factors thus reducing the neuroprotective function of these cell, which can contribute to neuronal dysfunction in mice. Microglia activation in HD was also observed in mouse models by Simmons *et al.* (2007) and the activation was shown to increase over the duration of the disease. Also, Pavese *et al.* (2006) findings provided evidence to correlate the level of microglial activation with HD severity. Crotti *et al.* (2014) using mice expressing mutant HTT, showed a role of mutant HTT in disrupting the regulation of microglia and that

microglia activation in HD is sufficient to promote neuronal degeneration. Björkqvist *et al.* (2008) have also found that in a peripheral level, patients with HD and mouse models, exhibited increased levels of IL-6, IL-8, and TNF-α, which are proinflammatory cytokines, even before clinical onset of HD proposing a mechanism of early innate immune activation in the disease. Besides, Träger *et al.* (2014) by silencing the expression of mutant HTT the production of the proinflammatory cytokines was ceased, suggesting that the mutant HTT was causing the immune cell activation. Furthermore, they also found evidence in the association between CAG repeat length and the level of TNF- α produced in HD (Träger *et al.*, 2014).

Kwan *et al.* (2012) isolated blood monocytes from HD patients to study migration defects, with these they have shown that the migration of monocytes and macrophages was severely impaired. Based on these data, they proposed a lack in cytokine and chemokine signaling mechanisms in HD, which may lead to a chronic increase in proinflammatory cytokines and chemokines in the CNS and consequently microglia activation enhancing inflammation.

The immune response involves a balanced interaction between the innate and adaptive immune system, in HD the first line of inflammation undoubtedly involves the innate immune system, but adaptive immune responses are also altered (Ellrichmann *et al.*, 2013). In Björkqvist *et al.* (2008) study IL-4 levels were significantly increased in plasma samples from HD patients. IL-4 is an anti-inflammatory cytokine involved in Th cell activation, which actions result in diminished neuroinflammation. The increase of IL-4 was mainly seen in late stages of the disease and may implicate an adaptive response to chronic immune activation.

Complement system has also been described in HD, Singhrao S. K. *et al.* (1999) reported the expression of several complement components and regulators of the classical pathway of complement activation in HD brain samples with severe atrophy. They propose that possibly the long polyglutamine expression render mutant huntingtin a strong activator of the complement system.

The role of immune system in other PolyQ diseases is less explored. However, for MJD, Evert *et al.* (2001, 2003 and 2006), confirmed the involvement of inflammatory processes in pathogenesis of MJD using cell lines and brain tissues. They identified genes involved in immune response, that supported the hypothesis that the pathogenic effect of mutant ATXN3 induced an inflammatory cascade enhancing the transcription of cytokines.

A study conducted by Mendonça et al. (2019) also demonstrated the implication of the immune system in MJD. Since, with the use of ibuprofen in mouse models, they observed a reduction of several inflammation markers, including TNF-α and IL1β.

The studies described above have shown several immune response markers that have been observed in the blood and CNS of patients and mouse models, which confirms the involvement of the immune response in PolyQ diseases, mainly in HD. Thus, it is important to better understand the pathway and the role of the immune system in the pathomechanism of all PolyQ diseases.

1.5. *In Silico* Immunogenicity Analysis

The first steps in immunoinformatic were in the 1980s, developed by Hopp and Woods (1983). Nowadays, *in silico* tools to identify the location of both B- and T-cell epitopes and to assess the potential for immunogenicity have been developed. These tools provide an alternative or an auxiliary to more complex *in vitro* or *in vivo* immunogenicity assays. With the aid of epitope predictive software and databases, it is possible to make an informed decision about the likelihood that a protein will provoke an immune response. According to their receptor epitopes can be B or T cell epitopes, these epitopes can be of different chemical nature. However, most of them are proteins and those are the subjects for epitope prediction methods (Sanchez-Trincado, Gomez-Perosanz and Reche, 2017; Yang and Yu, 2009).

There are essentially mainly four approaches to predict epitopes: sequence-based methods, structure-based methods, hybrid methods and consensus methods (Table 2), where the last one is the most accurate (Yang and Yu, 2009).

	PREDICTION APPROACHES		
	Basis	**How it Works**	**Limitations**
Sequence-based	Sequence dictates the structure, and the identical structure leads to identical function	Sequence–sequence comparison methods, used to find sequence homologues and assign amino acid scores by multiple alignment of query protein sequence, with template sequences in a database	Proteins with high sequence identity may possess differential functions and large datasets are required
Structure-based	3D protein structure to screen potential binders	Structural similarity between query protein and template proteins are used to predict epitopes of interest	High computational cost and development complexity, lower predictive performance than sequence-based
Hybrid	Sequential + structural analysis	Predictive methods taking advantage of both sequential and structural information	Possibility of false positives because different antibodies have overlapping binding sites
Consensus	Consensus from multiple methods	Combine strengths of various methods. Usually uses three methods and a prediction result is calculated with the results of all methods.	Computational limitations, compromised prediction speed, different output formats and the difficulty of integrating prediction results into the final consensus result

Table 2 – Epitope prediction approaches.

Adapted from Yang and Yu, 2009.

1.5.1. T-Cell Epitope Prediction Methods

Presentation of epitopes by both MHC-I and MHC-II contributes to the initiation of an immune response, since binding of HLA molecules to peptide epitopes, is essential for the activation of antigen-specific T cells. The T-cell epitope prediction goal is to identify the shortest peptides within an antigen that can stimulate either CD4 or CD8 T-cells (Greenbaum *et al.*, 2011; Jawa *et al.*, 2013; Sanchez-Trincado, Gomez-Perosanz and Reche, 2017).

T-cell epitope immunogenicity rely on three basic steps: antigen processing, peptide binding to MHC molecules, and recognition of MHC/peptide complex by a cognate TCR. The second, MHC-peptide binding, is the most selective one and it is the main basis to anticipate T-cell epitopes (Sanchez-Trincado, Gomez-Perosanz and Reche, 2017).

MHC-I and MHC-II molecules have similar structures, however there are differences between them that must be considered for peptide-binding prediction. MHC-I molecules can only bind short peptides with 8-11 amino acids. On the other hand, MHC-II binding grove is open at both ends and peptides can vary widely in length, typically 9-22 or 12-20 residues, although only a core of 9 residues (peptide-binding core) bind to MHC-II. Therefore, prediction methods for peptide-MHC-II binding often target to identify these peptide-binding cores. Taking this in consideration peptide-binding prediction to MHC-II molecules is less accurate than that of MHC-I molecules (Sanchez-Trincado, Gomez-Perosanz and Reche, 2017; Yang and Yu, 2009).

Physicochemical properties of epitopes are an important point to keep in mind to create a useful epitope prediction tool. There are an enormous number of physicochemical properties associated with epitopes, such as exposed surface, flexibility, hydrophilicity, charge, number of proline residues, accessibility, and the proximity of the segment towards the C- or N-terminal of the protein. To simplify computation, simpler quantitative descriptors of amino acid properties are used. The techniques include, binding motifs, quantitative matrices, virtual matrices, machine learning algorithms, evolutionary algorithms, linear programming, and others, are used to identify the binding peptide (Yang and Yu, 2009). Data driven methods for peptide-MHC binding prediction are based on peptide sequences that are known to bind to MHC molecules, these sequences are available in specialized epitope databases, for example Immune Epitope Database (IEDB) (Sanchez-Trincado, Gomez-Perosanz and Reche, 2017).

The strength of binding (affinity) of a peptide to an MHC molecule is an important factor that determinates potential immunogenicity, however, these indirect methods cannot give highly reliable prediction, as some MHC binders do not elicit immune response. Though, prediction accuracy is improving and there are numerous methods to predict peptide-MHC

binding. Using sequence-based methods we can have a prediction for peptide-MHC binding based on peptide sequences, available in specialized epitope databases, that are known to bind to MHC molecules. Undeniably, computational tools that predict peptides-MHC binding affinity can save valuable time and resources needed for the epitope identification process (Sanchez-Trincado, Gomez-Perosanz and Reche, 2017; Yang and Yu, 2009).

1.5.2. T-Cell Epitope Prediction Platforms

There are several *in silico* tools of T cell epitope mapping, by utilizing modern computational techniques these are able to quickly screen large datasets. Each software has a different principle of design, and therefore variations in the predictions are expected. Also, these methods depend on the data selection for model building, training, validation, epitope types and the user ability to correctly use the predictors tools. The MHC binding predictive software's are expanding rapidly and some of these tools are free ware and can be found and used online, other are companies' properties and the desired MHC prediction have to be purchased (Yang and Yu, 2009).

A list of T-cell epitopes related prediction servers and resources can be consulted in Table 3.

	Type	Developers/ Institution	Description	Website
EpiMatrix	Commercial	Groot and Martin EpiVax, Inc.	MHC I and II conserved and epitopes	www.epivax.com
Rankpep	Public	Reche Harvard Medical School	Prediction of MHC I and II binding peptides	http://bio.dfci.harvard.edu/RANKPEP/
IEDB	Public	Vita *et al.*	Mixed collection of tools of assorted derivation	www.iedb.com
Epibase	Commercial	Lasters and Stas Algonomics NV/Lonza, Inc.	T-cell epitope screening	www.lonza.com
TCED/ iTope	Commercial	Baker and Carr Antitope, Ltd.	Screen antibodies and proteins for potential immunogenicity	www.antitope.co.uk/
NetCTL	Public	Larsen *et al.* DTU Health Tech	Predicts CTL epitopes in proteins	http://www.cbs.dtu.dk/services/NetCTL
NetMHC	Public	Andreatta and Nielsen DTU Health Tech	Peptide-MHC I binding prediction	http://www.cbs.dtu.dk/services/NetMHC
ProPred	Public	Singh and Raghava Bioinformatics Center	Peptide-MHC II binding prediction	www.imtech.res.in/raghava/propred/

Table 3 – T-cell epitopes related prediction servers and resources.

Adapted from Jawa *et al., 2013;* Yang and Yu, 2009.

1.5.2.1. TepiTool

IEDB provides a collection of tools for the prediction and analysis of immune epitopes. It has several categories of tools, the ones of interest for this work are T-cell epitope prediction tools. The T-cell tools includes MHC-I and MHC-II binding predictions, as well as immunogenicity predictions. (Vita *et al.*, 2018).

The TepiTool (http://tools.iedb.org/tepitool/) is a computational free tool that is part of the IEDB, from a given set of amino acid sequences it provides prediction of T cell epitope candidates based on peptide binding to MHC-I and MHC-II molecules. It provides some of the top MHC binding prediction algorithms for hundreds of alleles, from various species. This tool has a step-by-step instruction with recommendations, helpful for prediction of the best T cell epitope candidates (Paul *et al.*, 2016).

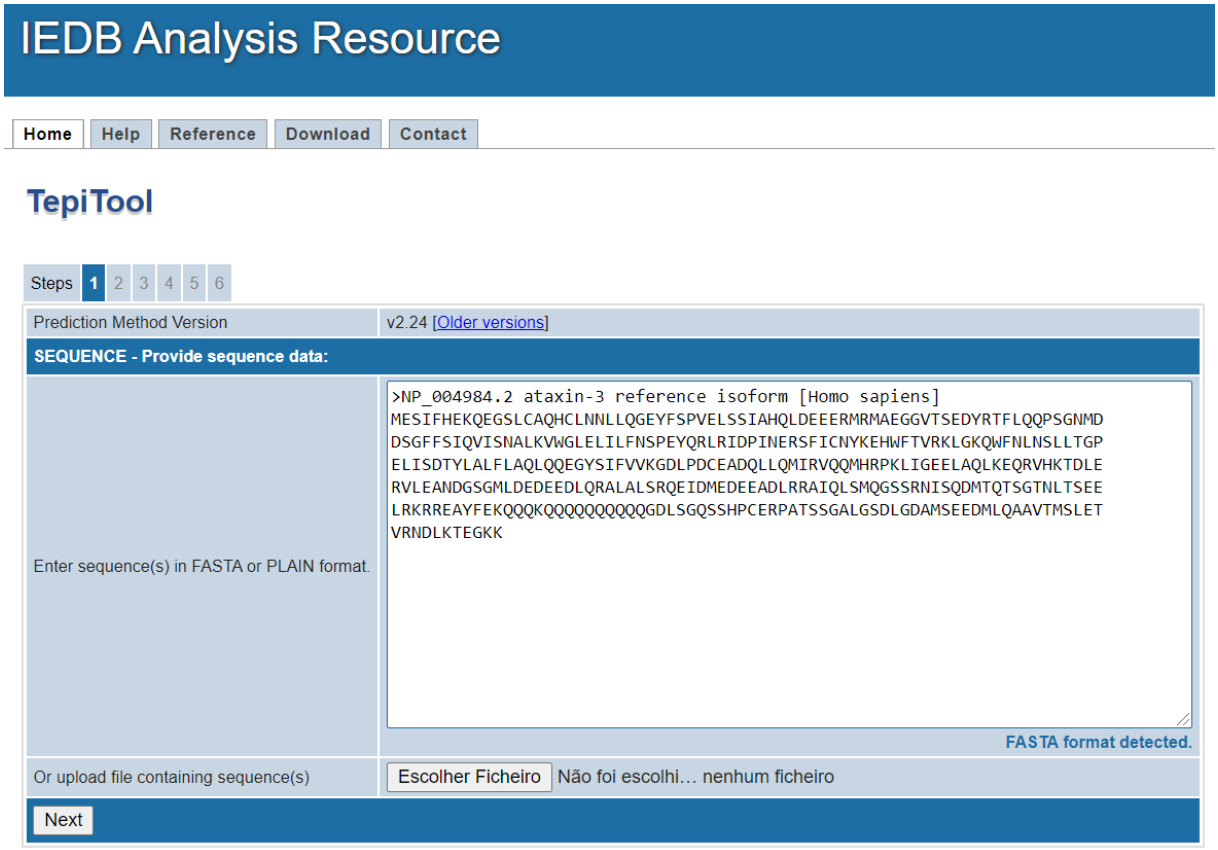

Figure 8 – Example image of the TepiTool tool.

Acquired at http://tools.iedb.org/tepitool/ (Paul *et al.*, 2016).

1.5.2.2. CD4 T Cell Immunogenicity Prediction

In IEDB, we can also find the "CD4 T Cell Immunogenicity Prediction" tool, this is a free tool that can be found online (http://tools.iedb.org/CD4episcore), which purpose is to predict the relative ability of a peptide-MHC complex to elicit an immune response.

Using human immunogenicity data associated with HLA class II binding predictions, Paul *et al.* (2018) identified sets of epitopes with high immunological activity and optimized a strategy to predict the top epitopes recognized by human populations.

The T cell immunogenicity can be predicted using 7-allele method, immunogenicity method and combined method. The combined method predicts the final score that combines the predictions from 7-allele method and immunogenicity method. (Paul *et al.*, 2015; Dhanda *et. al.*, 2018).

The description of the use of the tool as well as the steps to be performed are described in the methods, section 3.1.2.

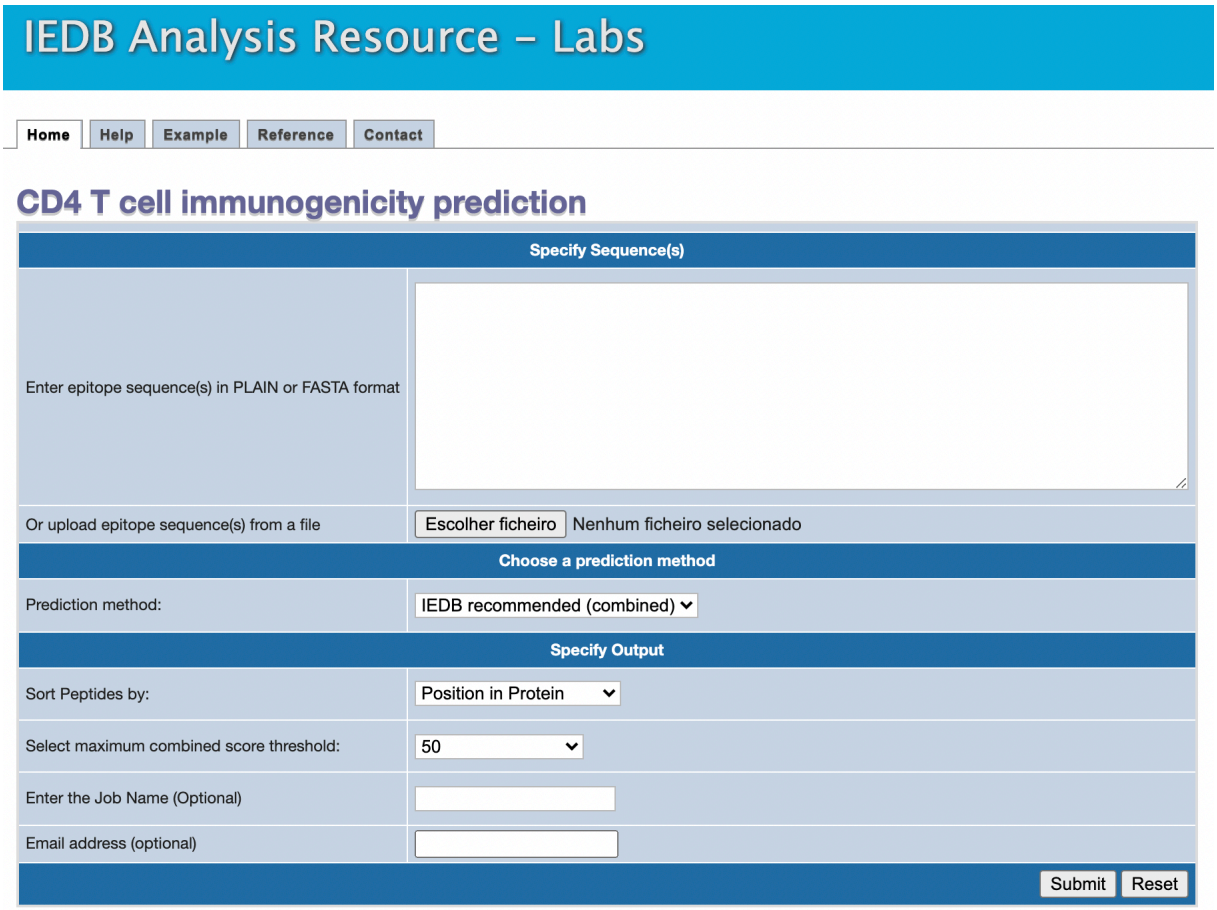

Figure 9 – Example image of the CD4 T Cell Immunogenicity Prediction tool.

Acquired at http://tools.iedb.org/CD4episcore/ (Dhanda *et al.*, 2018).

CHAPTER 2

OBJECTIVES

The immune system is known to play an important role in neurodegenerative diseases, including in PolyQ diseases. Despite the involvement of the innate immune system in disease pathology has been established over the last years, evidence regarding the role of adaptive immune system in PolyQ diseases is limited. Nevertheless, recent studies shown that adaptive immune system influence the progression of several neurodegenerative diseases, activating inflammatory pathways and contribute to neurodegeneration.

In this context, the main aim of this study was to perform an *in silico* analysis of the immunogenicity of wild-type and mutant ATXN3, and its derived cleavage fragments, to further perform *in vitro* studies for understanding the role of adaptive immune system in MJD.

The specific aims were the following:

1) to perform an *in silico* analysis to predict T cell epitopes with affinity to MCH-I and MCH-II molecules of wild-type and mutant ATXN3;
2) To perform an *in silico* analysis to predict the CD4 T Cell Immunogenicity of wild-type and mutant ATXN3, to identify putative peptides that might be immunogenic;
3) To identify the cleavage sites and fragments derived from ATXN3 proteolytic cleavage that could generate toxic fragments;
4) To identify proteolytic fragments with potential to be highly immunogenic;
5) To select the most promising ATXN3 fragments for *in vitro* testing of neuronal toxicity and immune system activation.

CHAPTER 3
MATERIALS AND METHODS

3.1. Evaluation of Immunogenicity

3.1.1. T-Cell MHC-I and MHC-II Epitope Binding Prediction

To evaluate the peptide-MHC I and MHC II binding the TepiTool tool (http://tools.iedb.org/tepitool/) was used. For analysis, 6 steps to insert input parameters were required. All fields, except sequences and alleles, were filled with default recommended settings for prediction and selection of optimum peptides (Paul *et al.*, 2016).

Predictions were made both for MHC-I and MHC-II binding prediction, for the full-length ATXN3 and mutant ATXN3. The same prediction was also made for human proteins with well-known immunogenicity, such as Interferon-β (IFN-β), Growth Hormone Releasing Hormone (GHRH), Erythropoietin (EPO), albumin and β-2-Microglobulin (β2M). Proteins from infectious agents described as immunogenic, were also predicted with TepiTool for MHC-I and MHC-II binding prediction, such as Pneumococcal Surface Protein A (PspA), BlpZ (fusion protein) and Pneumolysin (Ply) (Van de Garde *et al.*,2019).

The first step in the prediction process is to provide the protein sequence that should be scanned for MHC binding peptides. In this step, the full-length reference isoform of ATXN3 sequence was inserted in FASTA format. This step was performed with the sequence of all proteins described above; the sequence used for each protein can be found in Figure 1 of the supplementary data.

After the first step with the protein intake sequence, all the further steps were likewise performed for all the proteins analyzed.

Step 2 aimed to select the host specie and MHC allele class. The specie selected was the human, for both predictions, MHC-I and MHC-II.

The next step corresponds to the selection of the alleles for the binding prediction. In step 3, for MHC-I molecules, there are 5 options available. The option selected was the panel of 27 most frequent alleles, since contains a list with the 27 HLA most frequent alleles in the global population. For the prediction of MHC-II in the allele selection, there are 3 options available, the option selected was the one that predict for a panel of 26 most frequent alleles, which contains human MHC-II alleles from DP, DQ and DR loci. The options for both MHC classes were chosen to be the most representative.

Then, in step 4, it was necessary to select the peptides to be included in prediction. The parameters selected will allow for the definition of large sets of peptides, or limited sets based on more stringent criteria. Since MHC-I molecules can only bind short peptides with 8-11 amino acids, those were the peptide lengths to be considered in prediction and for this reason

the option selected was the default setting for moderate number of peptides. As already explained in 1.4.1., MHC-II molecules have an open binding groove and peptides can vary widely in length, however, the interaction between a peptide and an MHC-II molecule is provided only by a peptide core of about 9 amino acids length (9mer). Therefore, normally peptides of 15 residues are used for MHC-II binding predictions. The selected option was the 15mer peptides overlapping by 10 amino acid residues. With this setting the software provided the minimum number of peptides with all possible 9mer binding cores that have at least one flanking residue on both sides. Thus, the possibility of selecting redundant peptides with the same binding core can be avoid. In this step, the option of handling duplicate peptides only has influence when the input has more than one sequence, so it is not a feature to be considered in this case.

After the allele selection, it is required to select the preferred methods for binding prediction. In step 5, it is possible to choose from a list different MHC-I and MHC-II binding prediction methods. The selected method for both MHC-I and MHC-II prediction was the IEDB recommended, since this selection uses the best possible method for a given MHC molecule, based on availability of predictors and previously observed performance. For both MHC-I and MHC-II binding predictions, for a given allele, the IEDB recommended method represents a consensus method, considering 3 different methods for each type of MHC class. Then, the expected predictive performances are based on large scale evaluations of the performance of the MHC classes binding predictions.

The second field that needs to be selected in step 5 provides options of different approaches for selection of predicted peptides. The method selected in this field was based on predicted percentile rank, since it is the most accurate of the available options. In this method a lower percentile rank indicates higher affinity. The percentile rank is generated by comparing the peptides predicted binding affinity against a large set of peptides from a database. Percentile scores provide a uniform scale allowing comparisons across different predictors. Since it has been chosen a consensus method, the median percentile rank of the 3 methods involved is used to generate the consensus percentile rank. In this parameter, it is possible to provide the cutoff percentile rank for determining binders. For MHC-I molecules the default value is 1.0, meaning all peptides with percentile rank ≤ 1.0 will be selected as predicted peptides. Meanwhile, the recommended cutoff for human MHC-II alleles is 10.0, so this is the default value provided and all peptides with a percentile rank ≤ 10.0 will be selected as predicted peptides.

The last step is to review all the selections, enter job details, and submit data. In this step all the inputs are submitted to generate the output results.

The output results acquired with the TepiTool were transposed to tables that can be found in the supplementary data (Table I to XVIII).

3.1.2. CD4 T-Cell Immunogenicity Prediction

The CD4 T Cell Immunogenicity Prediction tool is a free approach to predict HLA class II immunogenicity at the population level, regardless of specific HLA haplotype, since it has a training neural network with well-characterized sets of immunogenic epitopes dominant in general human populations (Dhanda *et al*., 2018).

This prediction was made for full length ATXN3, mutant ATXN3 and for predicted ATXN3 cleavage fragments, as described in 3.1.3.

The first input parameter required by CD4 T-cell immunogenicity prediction tool is the peptide sequence. Then it is possible to choose the prediction method; the options available are the 7-allele method, the immunogenicity method and the IEDB recommended, being this last one the combination of the first two methods. The 7-Allele method is an optimized method for prediction of HLA class II responses, it predicts immunogenicity based on the median percentile predicted binding of seven alleles which are representative of the binding motifs most commonly recognized in the general human population. The immunogenicity method has a training neural network with well-characterized sets of immunogenic epitopes dominant in general human populations. The method selected was the IEDB recommended, since it has a better performance, once it combines immunogenicity and HLA binding predictions (Dhanda *et al*., 2018).

After the method selection it is necessary to specify the output selection, i.e. the selection of the score threshold. The scores range from 0 to 100, but if the score is higher than 50 the probability of a peptide immunogenicity is lower than non-immunogenicity. If the score is above 50 the probability of an immune response is higher than non-response. To see how the ATXN3 behaves the threshold selected for full-length and mutant protein was 100 (Dhanda *et al*., 2018).

3.1.3. Peptides Identification

The iProt-Sub is a bioinformatics tool for *in silico* prediction of protease-specific substrates and their cleavage sites. This way it is possible to predict the specific substrates that each protease targets. It covers four major proteases families: cysteine, metallo, serine and aspartic, and 38 different proteases. iProt-Sub uses a two-step feature selection procedure that integrates heterogeneous sequence and structural features to further remove redundant and

irrelevant features to improve the cleavage site prediction accuracy. A web server of iProt-Sub is available online at http://iProt-Sub.erc.monash.edu/. iProt-Sub was chosen because it is a recent method developed by Song *et al.* (2019) with the aim to construct, optimize, and validate protease-specific models to achieve a better performance than other available tools. Their experimental evaluations indicate that the proposed method outperforms previously developed methods. Besides, most of the tools there is a need to select the protease of interest to make the prediction, while iProt-Sub for a given substrate sequence will identify which, if any, of its 38 proteases will cleave that substrate. This feature was of particular interest since we can evaluate what other proteases besides caspases may cleave PolyQ subtracts (Song *et al.*, 2019).

To predict the cleavage sites of full-length ATXN3 using iProt-Sub tool, it was only necessary to use the protein reference sequence as an input. This information was acquired at the NCBI (National Center for Biotechnology Information) website (https://www.ncbi.nlm.nih.gov/). The reference sequence used can be consulted in the supplementary data (Figure I).

CHAPTER 4
RESULTS

4.1. T-Cell MHC-I and MHC-II Epitope Binding Prediction

4.1.1. Wild-Type ATXN3

To investigate whether ATXN3 is a highly immunogenic protein, i.e., can induce an immune response, an *in silico* analysis of ATXN3 affinity for MHC-I and MHC-II molecules was performed using the TepiTool.

TepiTool is a computational free tool that provides the prediction of peptide-MHC I and II binding and generates possible T cell epitope candidates. The results are given in a percentile rank score, corresponding a lower percentile rank to higher affinity.

The ATXN3 isoform used for this analysis is composed of 361 amino acids (Figure 10). In total, TepiTool identified 456 peptides within the ATXN3 sequence with predicted MHC-I binding with a percentile rank inferior to 1.00, (supplementary data Table I), the recommended value for the prediction of for human MHC-I alleles. The Table 1 of supplementary data shows all the peptides with MHC-I affinity, the position in the protein where peptide starts and ends, the HLA class I allele for which the peptide in question may have affinity and the corresponding percentile rank.

```
>NP_004984.2 ataxin-3 reference isoform [Homo sapiens]

MESIFHEKQEGSLCAQHCLNNLLQGEYFSPVELSSIAHQLDEEERMRMAEGGVTSEDYRTFLQQPSGNMD
DSGFFSIQVISNALKVWGLELILFNSPEYQRLRIDPINERSFICNYKEHWFTVRKLGKQWFNLNSLLTGP
ELISDTYLALFLAQLQQEGYSIFVVKGDLPDCEADQLLQMIRVQQMHRPKLIGEELAQLKEQRVHKTDLE
RVLEANDGSGMLDEDEEDLQRALALSRQEIDMEDEEADLRRAIQLSMQGSSRNISQDMTQTSGTNLTSEE
LRKRREAYFEKQQQKQQQQQQQQQQGDLSGQSSHPCERPATSSGALGSDLGDAMSEEDMLQAAVTMSLET
VRNDLKTEGKK
```

Figure 10 – Sequence of full-length ATXN3 reference isoform in FASTA format.

Acquired at https://www.ncbi.nlm.nih.gov/protein/NP_004984.2.

Similarly, full-length ATXN3-MHC-II binding was predicted using the same tool. TepiTool originated a total of 140 results of peptide binding affinity for MHC-II with a percentile rank inferior 10.0, that is the recommended cutoff for human MHC-II alleles (supplementary data Table II).

To better understand the biological significance of these results, the prediction with TepiTool was made with 5 proteins with known immunogenicity and 3 proteins that were

already studied with another bioinformatic tool to compare the results. These proteins include IFN-β, EPO, β2M, albumin, GHRH, PspA, BlpZ and Ply.

The binding prediction for MHC-I and MHC-II of the 6 proteins with well-known immunogenicity (IFN-β, EPO, β2M, albumin, GHRH) can be found in supplementary data Tables III to XII. For the results of PspA, BlpZ and Ply consult table XIII to XVIII of supplementary data.

The table below (Table 4) it is a summary of results obtained from proteins-MHC-I binding prediction. It has all proteins with well-known immunogenicity, the proteins described in literature as immunogenic and the full length ATXN3. The summary of the results of MHC-II binding prediction can be found in Table 5.

Protein	Amino Acids Number	Nº of Predicted Peptides Binding to MHC-I	MHC I Sore
BlpZ	77	160	2,08
IFN-β	187	377	2,02
Ply	471	900	1,91
EPO	193	357	1,85
Albumin	609	1012	1,66
β2M	119	192	1,61
GHRH	108	147	1,36
ATXN3	**361**	**454**	**1,26**
PspA	744	843	1,13

Table 4 – Summary table with all the predicted proteins with the TepiTool, for MHC-I binding prediction.

In these tables, and for each protein, the total number of amino acids contained in the protein, the number of peptides that binds to MHC and a MCH score, calculated by the ratio between the number of predicted peptides binding to MCH and the total number of amino acids of the protein, is presented.

Protein	Amino Acids Number	Nº of Predicted Peptides Binding to MHC-II	MHC II Sore
BlpZ	77	99	1,29
EPO	193	115	0,60
GHRH	108	62	0,57
IFN-β	187	89	0,48
ATXN3	**361**	**140**	**0,39**
β2M	119	46	0,39
Albumin	609	203	0,33
Ply	471	153	0,32
PspA	744	204	0,27

Table 5 – Summary table with all the predicted proteins with the TepiTool, for MHC-II binding prediction.

The scores of MHC-I and MHC-II binding prediction were summed to calculate a score of immunogenicity. With this score it was elaborated a ranking of protein binding affinity to MHC molecules (Table 6 and Figure 11).

Protein	MHC Molecules Score
BlpZ	3,36
IFN-β	2,49
EPO	2,45
Ply	2,24
β2M	2,00
Albumin	2,00
GHRH	1,94
ATXN3	1,65
PspA	1,40

Table 6 – Score results of the proteins-MHC binding prediction.

The Figure 11 compares the calculated score of the proteins with known immunogenicity and ATXN3, obtained through the prediction with TepiTool, where the proteins with known immunogenicity and ATXN3 are found in with relation to the ranking created by Van de Garde *et al.* (2019). ATXN3 is one of the less immunogenic proteins in the ranking of total immunogenicity and MHC-I prediction, however that does not mean it is not immunogenic. In MHC-II prediction ranking ATXN3 is in a higher position, occupying the middle place in the ranking.

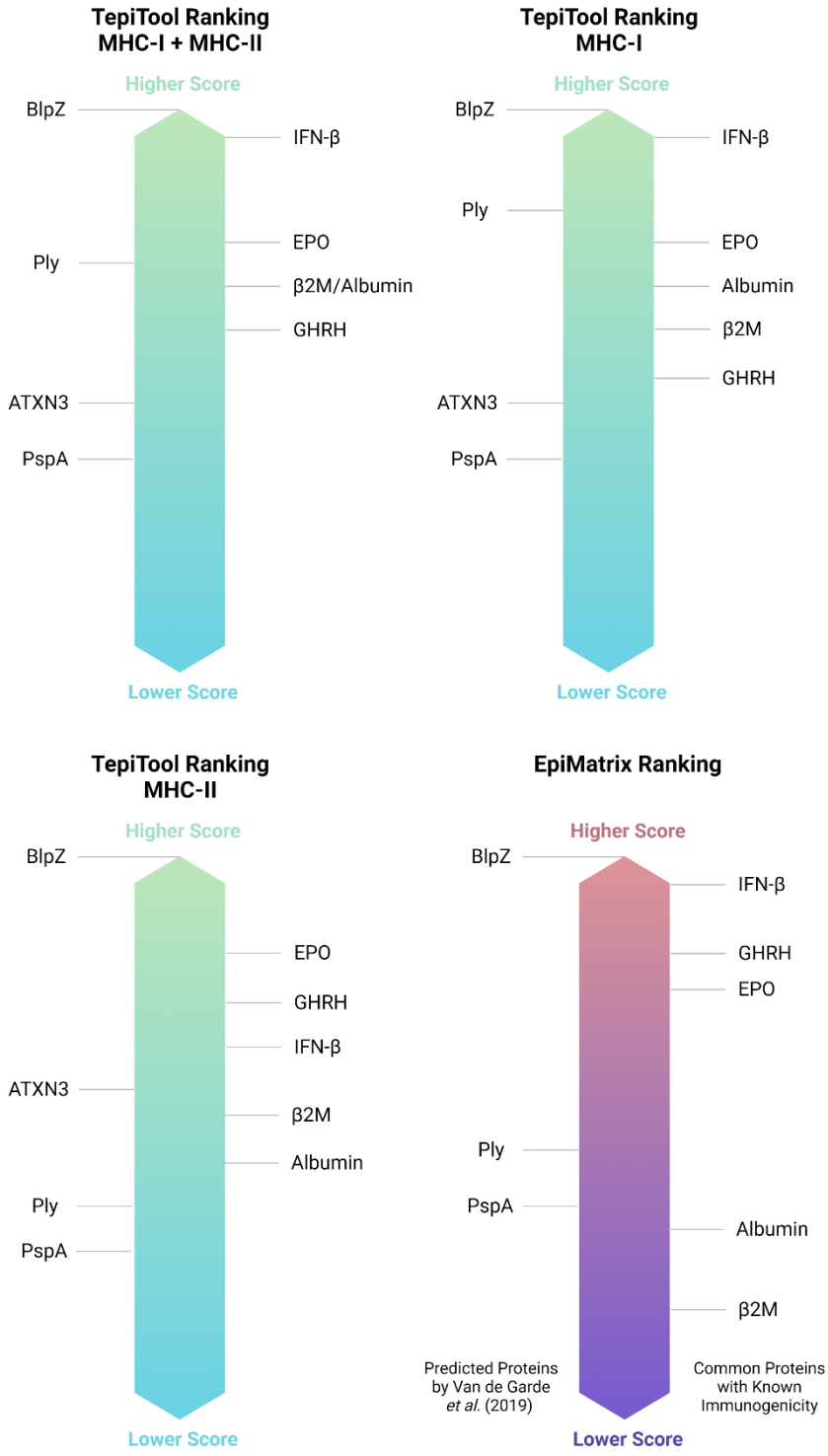

Figure 11 – Ranking of proteins-MHC binding prediction.

4.1.2. Mutant ATXN3

The TepiTool was also used to predict mutant ATXN3-MHC bindings. For the prediction of mutant ATXN3 to the reference isoform sequence of ATXN3 were added several glutamines to make a total of 80 repetitions, to mimic the mutant protein (Figure 12).

Figure 12 – Sequence of mutant ATXN3.

However, as it was already expected, the results obtained with mutant ATXN3 did not differ from those obtained with full-length ATXN3 (supplementary data Table I and II), since that the polyglutamine site is not a peptide affinity site and the results obtained with TepiTool are based on databases of peptide binding to MHC molecules. Indeed, the peptides that make up the sequence besides the polyglutamine repeating chain are the same.

4.2. CD4 T-Cell Immunogenicity Prediction

4.2.1. Wild-Type ATXN3

To predict the CD4 T cell immunogenicity of ATXN3 (see Figure 10 for sequence), we used the CD4 T cell Immunogenicity Prediction tool of IEDB.

After analysis, a total of 71 outputs were obtained for the immunogenicity prediction of full-length ATXN3, when using a threshold of 100. Data regarding the immunogenic peptide sequence (amino acids and the start and end position of the peptide in the protein), the peptide core, the combined score (between the immunogenicity score and the 7-allele method) and the individual results of the 7 representative alleles can be found in supplementary data Table XIX.

Table 7 contains the most relevant peptides (threshold < 50), the four peptides in this table are the ones that are more likely to be immunogenic than non-immunogenic. Besides the predicted peptides, it also shows the peptide core, and the position of the peptide in the protein, the score of the 7-allele method, the immunogenicity method, and the combined score.

Peptide	Start	End	Combined Score	Immunogenicity Score	Peptide Core	7-Allele Score
DSGFFSIQVISNALK	71	85	38.87016	86.3754	IQVISNALK	7.2
SIQVISNALKVWGLE	76	90	39.54152	79.3538	IQVISNALK	13.0
SPEYQRLRIDPINER	96	110	49.76712	97.4178	YQRLRIDPI	18.0
QLLQMIRVQQMHRPK	176	190	47.46268	94.6567	IRVQQMHRP	16.0

Table 7 – CD4 immunogenicity prediction of full-length wild-type and mutant ATXN3 with a threshold of 50.

4.2.2. Mutant ATXN3

The sequence of mutant ATXN3 shown in Figure 12 was used as input to calculate the CD4 T-cell immunogenicity prediction. The total peptides obtained as output with this tool, with a threshold of 100, was 2815, in which the first 72 peptides were the same obtained for full-length ATXN3, as expected. From the peptide 72 to the last result obtained, there was 2744 results of peptides only composed by glutamines, all with a combined score of 99.9836, in which the only difference in these results was the start and end position of the peptide in the protein (data not shown). The first 71 results can be consulted in the supplementary data Table XIX, which corresponds to the results of full-length ATXN3.

Being the first 71 results acquired for mutant ATXN3 equal to the full-length ATXN3, the results obtained with a threshold of 50 are also the same (Table 7).

4.3. ATXN3 Cleavage Peptides Immunogenicity Prediction

4.3.1. ATXN3 Cleavage Peptides Identification

ATXN3 can be cleaved by proteases and the proteolytic cleavage of mutant proteins in PolyQ diseases leads to the formation of fragments, which are believed to cause toxicity (toxic fragments hypothesis described in section 1.2). We hypothesize that toxicity of the fragments could be due to its immunogenicity. To further understand this matter, we first identify these possible toxic fragments by using an *in silico* approach through the iProt-Sub tool.

The iProt-Sub tool is a tool which predict proteases cleavage sites within a protein. The prediction is made for four major proteases families.

Table 8 correspond to the results obtained with iProt-Sub tool of predicted proteases cleavage sites. The table is separated by protease family and contains the protease and position where the protein can be cleaved, the cut segment, the size of the N- and C- fragments and the probability score of the cleavage occurring.

* Peptide described in literature (Wellington *et al.*, 1998; Berke, *et al.*, 2004; Weber *et al.*, 2017; Simões *et al.* 2021)

CYSTEINE FAMILY

PROTEASE	POSITION	SEGMENT	N-FRAGMENT	C-FRAGMENT	SCORE
Cathepsin L	290	AYFE|KQQQ	35.35 kDa	8.82 kDa	1.00
Calpain-1	319	CERP|ATSS	38.95 kDa	5.21 kDa	1.00
	210	TDLE|RVLE	25.73 kDa	18.44 kDa	0.95
	48	RMRM|AEGG	5.81 kDa	38.35 kDa	0.95
Calpain-2	137*	**NSLL**|TGPE	16.90 kDa	27.26 kDa	1.00
	327	GALG|SDLG	39.83 kDa	4.34 kDa	0.99
	312	SGQS|SHPC	38.14 kDa	6.02 kDa	0.95
	200	AQLK|EQRV	24.49 kDa	19.67 kDa	0.94
	360	LHYS|SFPL	43.72 kDa	0.44 kDa	0.93
Caspase-3	171*	**DLPD**|CEAD	20.97 kDa	23.19 kDa	1.00
	228*	**DEED**|LQRA	27.93 kDa	16.23 kDa	1.00
	145*	**LISD**|TYLA	17.83 kDa	26.33 kDa	0.96
	217*	**EAND**|GSGM	26.52 kDa	17.64 kDa	0.96
	248	EEAD|LRRA	30.25 kDa	13.92 kDa	0.96
Caspase-7	41	HQLD|EEER	4.85 kDa	39.31 kDa	1.00
	248	EEAD|LRRA	30.25 kDa	13.92 kDa	0.96
	244*	**DMED|EEAD**	29.80 kDa	14.36 kDa	0.95
	171*	**DLPD**|CEAD	20.97 kDa	23.19 kDa	0.95
	228*	**DEED**|LQRA	27.93 kDa	16.23 kDa	0.95
Caspase-6	241	QEID|MEDE	29.43 kDa	14.74 kDa	1.00
	248	EEAD|LRRA	30.25 kDa	13.92 kDa	0.97
	228*	**DEED**|LQRA	27.93 kDa	16.23 kDa	0.96
	168	VKGD|LPDC	20.65 kDa	23.51 kDa	0.95
	175	CEAD|QLLQ	21.39 kDa	22.77 kDa	0.94
	105	LRID|PINE	12.83 kDa	31.34 kDa	0.94
	217*	**EAND**|GSGM	26.52 kDa	17.64 kDa	0.94
	225*	**LDED**|EEDL	27.56 kDa	16.60 kDa	0.94
	171*	**DLPD**|CEAD	20.97 kDa	23.19 kDa	0.94
	307	QQGD|LSGQ	37.56 kDa	6.61 kDa	0.94
	329*	**LGSD**|LGKA	40.03 kDa	4.13 kDa	0.93
	70	GNMD|DSGF	8.55 kDa	35.62 kDa	0.93
Caspase-8	171*	**DLPD**|CEAD	20.97 kDa	23.19 kDa	1.00
	241	QEID|MEDE	29.43 kDa	14.74 kDa	0.94

METALLO FAMILY					
PROTEASE	POSITION	SEGMENT	N-FRAGMENT	C-FRAGMENT	SCORE
Matrix Metallopeptidase-1	32	SPVE|LSSI	3.89 kDa	40.27 kDa	1.00
	201	QLKE|QRVH	24.62 kDa	19.54 kDa	0.94
Matrix Metallopeptidase-8	263	SSRN|ISQD	32.06 kDa	12.10 kDa	1.00
	280	TSEE|LRKR	34.00 kDa	10.16 kDa	1.00
	275	SGTN|LTSE	33.44 kDa	10.72 kDa	0.98
	57	TSED|YRTF	6.89 kDa	37.27 kDa	0.97
Matrix Metallopeptidase-2	307	QQGD|LSGQ	37.56 kDa	6.61 kDa	1.00
	32	SPVE|LSSI	3.89 kDa	40.27 kDa	0.95
	329*	**LGSD**|LGKA	40.03 kDa	4.13 kDa	0.95
Matrix Metallopeptidase-9	141*	TGPE|**LISD**	17.41 kDa	26.76 kDa	1.00
Matrix Metallopeptidase-3	198	ELAQ|LKEQ	24.25 kDa	19.91 kDa	1.00
	279	LTSE|ELRK	33.87 kDa	10.29 kDa	0.95
Matrix Metallopeptidase-7	198	ELAQ|LKEQ	24.25 kDa	19.91 kDa	1.00
	329*	**LGSD**|LGKA	40.03 kDa	4.13 kDa	0.95
	32	SPVE|LSSI	3.89 kDa	40.27 kDa	0.93
Matrix Metallopeptidase-12	32	SPVE|LSSI	3.89 kDa	40.27 kDa	1.00
Matrix Metallopeptidase-13	101	EYQR|LRID	12.33 kDa	31.83 kDa	1.00
	57	TSED|YRTF	6.89 kDa	37.27 kDa	0.96
	263	SSRN|ISQD	32.06 kDa	12.10 kDa	0.94
Membrane-type Matrix Metallopeptidase-12	182	QMIR|VQQM	22.28 kDa	21.89 kDa	1.00
ADAMTS4 Peptidase	173	PDCE|ADQL	21.21 kDa	22.96 kDa	1.00
	8	FHEK|QEGS	1.00 kDa	43.17 kDa	0.98
	280	TSEE|LRKR	34.00 kDa	10.16 kDa	0.94
	329*	**LGSD**|LGKA	40.03 kDa	4.13 kDa	0.94
	57	TSED|YRTF	6.89 kDa	37.27 kDa	0.94
	248	EEAD|LRRA	30.25 kDa	13.92 kDa	0.93
	203	KEQR|VHKT	24.90 kDa	19.26 kDa	0.93
Neprilysin	101	EYQR|LRID	12.33 kDa	31.83 kDa	1.00
	208	HKTD|LERV	25.48 kDa	18.68 kDa	0.99
Insulysin	260	MQGS|SRNI	31.70 kDa	12.46 kDa	1.00
	311	LSGQ|SSHP	38.06 kDa	6.10 kDa	0.97
	310	DLSG|QSSH	37.93 kDa	6.23 kDa	0.97
	45	EEER|MRMA	5.40 kDa	38.77 kDa	0.96
	282	EELR|KRRE	34.27 kDa	9.89 kDa	0.96

SERINE FAMILY					
PROTEASE	POSITION	SEGMENT	N-FRAGMENT	C-FRAGMENT	SCORE
Granzyme B	329*	**LGSD**|LGKA	40.03 kDa	4.13 kDa	1.00
	241	QEID|MEDE	29.43 kDa	14.74 kDa	1.00
	267	ISQD|MTQT	32.50 kDa	11.66 kDa	0.99
	171*	**DLPD**|CEAD	20.97 kDa	23.19 kDa	0.98
	223	GMLD|EDEE	27.32 kDa	16.85 kDa	0.97
	175	CEAD|QLLQ	21.39 kDa	22.77 kDa	0.96
	225*	**LDED**|EEDL	27.56 kDa	16.60 kDa	0.94
	307	QQGD|LSGQ	37.56 kDa	6.61 kDa	0.93
Kallikrein-related Peptidase 5	124	FTVR|KLGK	15.25 kDa	28.92 kDa	1.00
Elastase-2	68	PSGN|MDDS	8.30 kDa	35.86 kDa	1.00
	271	MTQT|SGTN	32.96 kDa	11.20 kDa	1.00
Granzyme A	124	FTVR|KLGK	15.25 kDa	28.92 kDa	1.00
	190	HRPK|LIGE	23.28 kDa	20.88 kDa	0.96
	211	DLER|VLEA	25.88 kDa	18.28 kDa	0.95
	237	ALSR|QEID	28.94 kDa	15.22 kDa	0.94
Plasmin	250	ADLR|RAIQ	30.52 kDa	13.65 kDa	1.00
	103	QRLR|IDPI	12.60 kDa	31.56 kDa	0.96
	284	LRKR|REAY	34.55 kDa	9.61 kDa	0.95
	182	QMIR|VQQM	22.28 kDa	21.89 kDa	0.95
	45	EEER|MRMA	5.40 kDa	38.77 kDa	0.95
	200	AQLK|EQRV	24.49 kDa	19.67 kDa	0.93
Kallikrein-related Peptidase 4	285	RKRR|EAYF	34.71 kDa	9.45 kDa	1.00
Furin	285	RKRR|EAYF	34.71 kDa	9.45 kDa	1.00

ASPARTIC FAMILY					
PROTEASE	POSITION	SEGMENT	N-FRAGMENT	C-FRAGMENT	SCORE
Cathepsin D	91	GLEL|ILFN	11.08 kDa	33.08 kDa	1.00
	74	DSGF|FSIQ	9.07 kDa	35.09 kDa	1.00
	102	YQRL|RIDP	12.44 kDa	31.72 kDa	0.96
	120	KEHW|FTVR	14.74 kDa	29.42 kDa	0.95
	195	IGEE|LAQL	23.94 kDa	20.23 kDa	0.94
	209	KTDL|ERVL	25.60 kDa	18.57 kDa	0.93

Table 8 – Results acquired with the iProt-Tool of the predicted cleavage sites of ATXN3.

For the cysteine family, seven proteases including, cathepsin L, calpain-1 and -2, caspase-3, -6, -7 and -8 are predicted to cleave ATXN3. Sixteen fragments were expected to have high probability of being obtained by cleavage, since were obtained eight cleavage sites with a score of 1.00. Among the 33 results of cysteine family proteases cleavage site prediction, 13 of this cleavage sites were already described in literature (Wellington *et al*., 1997; Berke *et al*., 2004; Simões *et al.,* 2021; Weber *et al*.,2017).

For the metallo family 34 results of cleavage sites were obtained related to the predicted cleavage by 12 different proteases, being 10 results of high cleavage score. Nevertheless, only four of these were described in literature. The Serine family had 3 cleavage sites that coincided with described cleavage sites. For this protease family a total of 23 cleavage sites were obtained, generated for 7 proteases, and with 9 results with a score of 1.00, meaning high probability of cleavage. Aspartic family prediction shows the cleavage sites that are cleaved by Cathepsin D, 2 out of the 6 results acquired had a score of 1.00 and none of these cleavage sites has been described.

4.4. ATXN3 Cleavage Peptides Selection for *in vitro* Studies

4.4.1. Fragments Described in Literature

Despite the value of the *in silico* tools, experimental validation of the immunogenicity is critical, as the platforms have several limitations (Yang and Yu, 2009; Dhanda *et al*., 2019)

To restrict the number of peptides for *in vitro* validation, we search in the literature for already described cleavage peptides. The studies of Wellington *et al.* (1998) and Berke *et al*. (2004) identified nine potential caspase cleavage sites, and a recent study conducted by Simões *et al.* (2021) identified three calpain cleavage sites, one in full length wild-type ATXN3 (at amino acid position 194) and two in mutant ATXN3 (at position 253 and 273). Weber *et al.* (2017) also highlighted two calpain cleavage sites at amino acid 208 and 256.

To confirm the high potential of *in vitro* formation of the toxic fragments, the described peptides were sought into the iProt-Sub results. From the nine caspase cleavage sites described six were found in the obtained results. Also, with this analysis we found more proteases, besides caspases that could possibly cleave the peptides in the cleavage sites described in literature. The exact amino acid positions described for calpain cleavage sites were not found in the iProt-Sub results. However, Simões *et al.* (2021) identified fragments with a molecular mass of approximately 26 and 34kDa, with this data four segments cleaved by calpains in the iProt-Sub prediction were found to have a similar molecular mass. The cross of information

between iPro-Sub results and the fragments described in literature can be consulted in Table 9. The table contains the peptide segments at the cleavage site and the cleavage site; the size of N- and C- fragments, that may be helpful to elucidate possible toxicity of the fragments formed by the cleavage; the iProt-Sub score of the cleavage prediction; the column referent to proteases expresses the proteases acquired in iProt-Sub that could cleave the peptides described in literature; and the references, if available.

ATXN3 Cleavage Sites						
SEGMENT	**POSITION**	**N-FRAG**	**C-FRAG**	**SCORE**	**PROTEASE**	**REFERENCE**
NSLL	137	16.90 kDa	27.26 kDa	1.00	Calpain-2	Simões *et al.* (2021)
LISD	145	17.83 kDa	26.33 kDa	1.00	Caspase-3	Berke *et al.* (2004) and Wellington *et al.* (1998)
				0.96	Caspase-8	
DLPD	171	20.97 kDa	23.19 kDa	1.00	Granzime B	
				1.00	Caspase-7	
				0.98	Caspase-6	Berke *et al.* (2004) and Wellington *et al.* (1998)
				0.95	Metallopeptidase-9	
				0.94	Caspase-3	Berke *et al.* (2004) and Wellington *et al.* (1998)
AQLK	200	24.49 kDa	19.67 kDa	0.94	Calpain-2	Simões *et al.* (2021)
				0,93	Plasmin	
TDLE	210	25.73 kDa	18.44 kDa	0.95	Calpain-1	Simões *et al.* (2021)
EAND	217	26.52 kDa	17.64 kDa	0.96	Caspase-3	Berke *et al.* (2004)
				0.94	Caspase-6	Berke *et al.* (2004)
LDED	225	27.56 kDa	16.60 kDa	0.94	Caspase-6	Berke *et al.* (2004) and Wellington *et al.* (1998)
					Granzime B	
DEED	228	27.93 kDa	16.23 kDa	1.00	Caspase-3	Berke *et al.* (2004) and Wellington *et al.* (1998)
				0.96	Caspase-6	Berke *et al.* (2004) and Wellington *et al.* (1998)
				0.95	Caspase-7	
SGQS	312	38.14 kDa	6.02 kDa	0.95	Calpain-2	Simões *et al.* (2021)
LGSD	329	40.03 kDa	4.13 kDa	1.00	Granzime B	
				0.95	Metallopeptidase-2	
				0.95	Metallopeptidase-7	
				0.94	ADAMTS4 Peptidase	
				0.93	Caspase-6	Berke *et al.* (2004)

Table 9 – Prediction of protease substrates and their cleavage sites for ATXN3.

Caspase-3 and -6 are the proteases that are known to cleave ATXN3. In Table 9 we can see that iProt-Sub showed that various proteases appear to possible cleave in the same described cleavage sites that these proteases, since four other proteases besides caspase-3 and -6 have scored to the same cleavage sites. For calpains cleavage sites, only plasmin appeared to cleave at one of caspase-2 cleavage site. DLPD and LGSD, at position 171 and 329, respectively, are the ones that seem to be cleaved by a higher number of proteases.

It is important to test, *in vitro,* the peptides with higher affinity for MHC molecules and higher immunogenicity score. From the analysis made in 4.1.2 and 4.2.2 we identified three peptide cores with potential to be highly immunogenic (Table 10, 11 and 12).

Peptide Core:	IQVISNALK	IRVQQMHRP	YQRLRIDPI
Combined Score:	≈ 39	≈ 48	≈ 49
Immunogenicity Score:	≈ 82	≈ 95	≈ 97
7-Allele Score:	≈ 10	≈ 17	≈ 18
Start Position:	77	181	99
End Position:	85	189	107

Table 10 – Summary table of the peptide cores acquired with the CD4 Immunogenicity Prediction tool, with the average values of prediction.

After selecting the most promising cleavage sites that may form the peptide fragments that occur *in vivo* (Table 9), we searched the peptides core with higher immunogenicity results (Table 10) in the fragments obtained through the described cleavage sites (Figure II to XII) of supplementary data). With this information we located in which of the fragments were the peptide cores (Table 11). The table contains the segments described in literature, the fragment in which the PolyQ repeats will be after the cleavage, the amino acid position of the cleavage site and the number of peptide cores in N- and C-fragment after the cleavage. The fragments containing one or more peptide cores should be the ones to consider studying *in vitro*. That is, the N- and C- fragment of the described peptide segments between cleavage site 137 and 171, and the N-fragment of the segments between 200 and 329.

	Site of PolyQ Repeats	**Cleavage Site (Amino Acid Position)**	**Number of Peptide Cores in N-Fragment**	**Number of Peptide Cores in C-Fragment**
NSLL	C-Fragment	137	2	1
LISD		145		
DLPD		171		
AQLK		200	3	0
TDLE		210		
EAND		217		
LDED		225		
DEED		228		
SGQS	N- Fragment	312		
LGSD		329		

Table 11 – Identification of peptide cores in the described fragments of ATXN3.

4.4.2. Identification of New Fragments

In addition to the fragments identified in the literature, and based on the results acquired with the iProt-Sub, we were also able to identify proteases (and corresponding cleavage sites) with a high probability to cleave ATXN3 and generate new fragments that could be toxic. To elaborate the Table 12, first we selected the ATXN3 cleavage sites that are not described in literature with the score of 1.00, which means those with higher ability to be a target. Since the last peptide core found (Table 10) ends ate position 189, all the cleavage sites after this position will form a fragment containing the three peptide cores at the N-fragment. So, we selected the cleavage sites that originate fragments that contain the three peptide cores that could be immunogenic (supplementary data Figure XIII to XXI).

We found ten cleavage sites that could originate ten new fragments containing the immunogenic peptide cores. These fragments would be interesting to study *in vitro*.

Cleavage Score	1.00
Number of Peptide Cores in N-Fragment	3

SEGMENT	POSITION	N-FRAGMENT	C-FRAGMENT	PROTEASE
ELAQ|LKEQ	198	24.25 kDa	19.91 kDa	Matrix Metallopeptidase-3 Matrix Metallopeptidase-7
QEID|MEDE	241	29.43 kDa	14.74 kDa	Caspase-6 Granzyme B
ADLR|RAIQ	250	30.52 kDa	13.65 kDa	Plasmin
MQGS|SRNI	260	31.70 kDa	12.46 kDa	Insulysin
SSRN|ISQD	263	32.06 kDa	12.10 kDa	Matrix Metallopeptidase-8
MTQT|SGTN	271	32.96 kDa	11.20 kDa	Elastase-2
TSEE|LRKR	280	34.00 kDa	10.16 kDa	Matrix Metallopeptidase-8
RKRR|EAYF	285	34.71 kDa	9.45 kDa	Kallikrein-related Peptidase 4 Furin
AYFE|KQQQ	290	35.35 kDa	8.82 kDa	Cathepsin L
QQGD|LSGQ	307	37.56 kDa	6.61 kDa	Matrix Metallopeptidase-2

Table 12 – Identification of potentially immunogenic new fragments.

CHAPTER 5
DISCUSSION

The role of adaptive immune system is poorly understood in PolyQ disorders, including in MJD. In this study, we used two *in silico* tools to predict the immunogenicity of ATXN3 protein: the TepiTool to predict protein-MHC-I and II binding, and the CD4 T Cell Immunogenicity Prediction tool that combines the evaluation of immunogenicity and the binding affinity to MHC molecules. Moreover, the iProt-Sub tool was used to predict possible cleavage sites (and derived fragments) that may be associated with proteolytic cleavage of ATXN3, and finally identify putative toxic ATXN3 fragments for in vitro studies, based on their immunogenicity potential and probability of being formed in physiological conditions.

For the prediction of immunogenicity of ATXN3, first it was evaluated the peptide binding affinity to MHC-I and MHC-II molecules of the full-length ATXN3 with the TepiTool. From the data obtained with TepiTool, wild-type ATXN3 does not appear to be high immunogenic. To better understand the relevance of the data obtained we used the protein sequence of common proteins with known-immunogenicity, including IFN-β, EPO, GHRH, albumin and β2M, and PspA, BlpZ and Ply sequences (Van de Garde et al., 2019) to predict peptide binding affinity to MHC-I and MHC-II molecules of these proteins, with the same bioinformatic tool.

IFN-β is a cytokine that is capable to stimulate the immune response, the other proteins EPO, GHRH, albumin and β2M, are endogenous proteins that were described by Van de Garde *et al.* (2019) and were used as a comparison.

PspA, is a surface-exposed protein virulence factor of *Streptococcus pneumoniae, that* has been suggested as a potential pneumococcal vaccine antigen (Kye *et al.*, 2019). Baril *et al.* (2006) evaluated the capacity of PspA to stimulate CD4 T-cells and demonstrated that PspA is efficient at eliciting, both cellular and antibody responses. Van de Garde *et al.* (2019) went further and evaluated the most immunogenic regions of previously described peptides as immunogenic targets for CD4 T-cells, first *in silico* (with EpiMatrix) and then confirmed their predictions *in vitro*. The PspA predicted peptide was associated with a polyfunctional cytokine response and was among the proteins with the highest stimulation index of T-cell proliferation *in vivo*. Van de Garde *et al.* (2019) study also have shown other potentially immunogenic proteins, among them, BlpZ had the best score of potential binding to T cell epitopes *in silico* and also showed one of the highest stimulation indices *in vivo*. Ply showed the highest proliferative response *in vivo*. In this study Van de Garde *et al.* (2019) composed a ranking of immunogenicity, comparing the proteins they studied with proteins with well-known immunogenicity.

Similarly, to the study of Van de Garde *et al.* (2019), with the results acquired with TepiTool, we developed a ranking of proteins affinity to MHC-I, MHC-II and for the sum of both (Figure 10). In MHC-II ranking, the predicted proteins are in similar places to the Van de

Garde *et al.* (2019) ranking, validating this as the most relevant ranking to compare ATXN3 results.

When evaluating the ATXN3 in MHC-II ranking position with the prediction made with TepiTool, ATXN3 is in the middle close to the most common proteins. Regarding the other two rankings, MHC-I and MHC-I+MHC-II, ATXN3 appears to be less immunogenic, whereas BlpZ showed the most affinity to MHC molecules, in both MHC-I and MHC-II binding predictions and PspA had the lowest affinity to MHC molecules. However, the MHC-II ranking seems to be the most relevant, since it is more similar to the Van de Garde *et al.* (2019) study which was validated *in vitro*.

Although that in general ATXN3 does not seem high immunogenic, it was always above PspA in the predictive rankings. Since, a PspA epitope was found to have a high stimulation index of T-cell proliferation *in vivo* it is important to identify not only the peptides with high affinity for MHC molecules but the ones who can actually provide an immune response.

A higher affinity to MHC molecules may in fact induce T cell responses, however it is important to understand whether the predicted peptides with affinity for MHC, that is, possible T-cell epitopes, can or not produce an immune response. A protein can have a higher score of affinity to MHC but that does not mean that the peptides that bind to that MHC molecule can actually be capable of producing an immune response. Although HLA binding is necessary it is not sufficient by itself to produce a T cell immune response (Paul *et al.*, 2015; Dhanda *et al.*, 2018). Thus, *in silico* prediction of the ability of peptides to bind to MHC molecules will not identify the exact location of T-cell epitopes, neither identify the exact peptides capable of produce an immune response, since several factors affect this response and immunogenicity can be influenced by antigen processing (by the APC) and the size of TCR that is capable of recognize the peptide/epitope-MHC complex.

To predict mutant ATXN3 immunogenicity, it was used a sequence to mimic mutant ATXN3 for understanding whether this protein could be more immunogenic than wild-type. However, the results obtained with mutant ATXN3 did not differ from those obtained with full-length since the PolyQ site is not a peptide affinity site. In this case, tool's limitations need to be considered. As the TepiTool results are acquired based on databases of peptide binding to MHC-I and MHC-II molecules the only difference between both sequences is the PolyQ repeats. Logically, the other peptides that compose the sequence the prediction will be the same. Although the PolyQ region does not seem to have affinity to MHC molecules neither to be immunogenic, that does not mean it is not, since it could bind to other proteins or RNA in cell and that could confer immunogenicity (Fiszer and Krzyzosiak, 2013).

Since a higher score of peptides affinity to MHC molecules could be relevant but it does not mean that the peptide is more or less immunogenic, we used The CD4 Immunogenicity Prediction tool to predict the immunogenicity of full-length and mutant ATXN3. This tool is an improved version of the free available MHC-II immunogenicity prediction tools, since identify sequence motifs to distinguishing immunogenic peptides recognized by CD4 T cells from non-recognized peptides, independent of the restricting HLA class II allele (Dhanda *et al.,* 2018).

Once more, the only difference between full-length ATXN3 and mutant ATXN3 are the PolyQ repeats so the results of the peptides immunogenicity prediction were the same, except for the peptides composed of glutamines. Although the peptides that contain the PolyQ repeats do not have a high immunogenicity score, they can somehow influence the immunogenicity of the remaining protein. Since, when we add more "Q" to the sequence used in the prediction, the more results are obtained by the prediction tool.

Although ATXN3 seems to have poor immunogenicity, CD4 Immunogenicity Prediction Tool identified three peptide cores that could be immunogenic. As it is described that in MJD (and other PolyQ diseases) toxicity might be related with the formation of toxic ATXN3 fragments instead of full-length protein (Weber *et al.*, 2017; Wellington *et al.*, 2000; Wellington and Hayden, 2000), we hypothesize that immunogenicity could be derived from highly immunogenic ATXN3 fragments.

After the ATXN3 immunogenicity analyses, and to explore this hypothesis, we first identify possible fragments that could be formed endogenously, using the iProt-Sub tool to predict ATXN3 cleavage sites by proteases. Since we acquired several results of predicted cleavage sites and to short the data obtained, we searched the literature to see if there were cleavage sites already confirmed.

Thus, based on studies conducted by Wellington *et al.* (1998), Berke *et al.* (2004), Weber *et al.* (2017) and Simões *et al.* (2021) we went to search the cleavage sites they described in the results acquired with iProt-Sub. Wellington *et al.* (1998) and Berke *et al.* (2004) found evidence that the cleavage of proteins by caspases will produce toxic fragments for the cells. Wellington *et al.* (1998) have tried to answer several questions, since Goldberg *et al.* (1996) observed that HTT is a substrate for proteolytic cleavage by caspase-3. Wellington and his group questioned if other caspases could cleave HTT and if caspases could cleave other proteins that causes PolyQ diseases. In this way, their studies provide the amino acid position and peptide segments of potential cleavage sites of caspases for four proteins causing PolyQ diseases (ATXN3, HTT, AR and ATN1). Berke *et al.* (2004) followed Wellington *et al.* (1998) studies and highlight some potential caspase cleavage sites in ATXN3.

Weber *et al.* (2017) and Simões *et al.* (2021) researched for calpain cleavage sites. Weber *et al.* (2017) confirmed ATXN3 is a substrate for calpain cleavage in patient-derived cell lines and post-mortem brain tissue, and then found ATXN3 is primarily cleaved at two sites.

Simões *et al.* (2014, 2021) investigated the potential role of calpain inhibition and predicted 3 calpain cleavage sites based when fragments size. They found that the ATXN3 cleavage by calpain at two sites originates a fragment with ≈34kDa and a ≈26kDa fragment, that are required for protein aggregation. They also have shown that the C-terminal ≈26 kDa fragment is the main contributor to striatal degeneration.

The cleavage sites described in literature were used to cross-reference with the information acquired from the iProt-Sub results. We obtained several cleavage sites that were already described; however, we also acquired several results with a high probability of occurring that are not described. This proteases with a high probability of cleaving ATXN3, could also form new toxic fragments.

In short, with this research we identified ten cleavage sites that coincided with those described by Wellington *et al.* (1998), Berke *et al.* (2004), Weber *et al.* (2017) and Simões *et al.* (2021). We also found that besides the described caspases and calpains, there were other protease to cleave the protein at the same cleavage sites, this could mean there is a higher probability to form the fragments described as toxic in physiological or pathological conditions.

As already referred, the *in silico* analysis have several limitations and therefore it is important to perform *in vitro* (and afterwards *in vivo*) validation.

Since we hypothesized that the toxic fragments could be related to their immunogenicity, we aimed to identify the best candidates to study *in vitro,* that is, the fragments that appear to be toxic when cleaved. Using the results acquired with CD4 T Cell Immunogenicity Prediction tool we searched for the peptide cores that have the probability to induce an immune response in the fragments generated through the ten described cleavage sites. Considering that immunogenicity is what confers the toxic potential to the fragments, the ones to consider will be the ones with the greatest number of peptide cores. Of the twenty fragments formed by the described cleavage sites, seven of them have the three immunogenic peptides core in the N-fragment. This could mean that these are the most immunogenic fragments, and therefore the most toxic. However, Simões *et al.* (2021) identified a C-terminal ≈26 kDa fragment to be the main responsible for striatal degeneration, the fragment of our study that seems to have these characteristics only have one of the predicted peptide cores in the C-fragment.

Consequently, it would be important to consider all the fragments obtained with the described cleavage sites, that contain one or more peptide cores and not only the ones with

the most peptide cores, since some could be more immunogenic than others and that could influence the immunogenicity of the whole fragment. Since we identified 13 fragments with at least one peptide core (Table 11), these are the ones that we must consider studying *in vitro*.

Since we found several putative ATXN3 cleavage sites that are not yet described in literature but had high predictive scores, we selected the most promising cleavage sites that could form new toxic fragments. Once the peptide cores had similar immunogenicity results and we do not know which of these are in fact the most immunogenic, we selected the fragments that, when cleaved, contains the three peptide cores, and for this reason will be considered more immunogenic (Table 12). From this analyze we found ten new fragments containing the immunogenic peptide cores. It would be of great interest to use these fragments to study *in vitro* for evaluation of toxicity and ability to induce an immune response.

Important to notice that this study had some limitations, since most platforms of immunogenicity and binding predictions are paid and not free available. Furthermore, despite the efforts and advances of T cell epitope prediction tools, the extensive polymorphism and high heterogeneity of HLA molecules in general population represents an obstacle to epitope identification approaches, principally in HLA class II molecules (Paul *et al.*, 2015; Dhanda *et al.*, 2018). *In silico* methods constantly over-predict the number of T-cell epitopes present in a protein sequence, and the factors that influence immune response, are also a limitation for bioinformatics tools. However, these tools are a useful aid to identifying peptide sequences that have the potential to bind to MHC molecules to help to limit the *in vivo* and *in vitro* analysis time spent and costs.

Overall, with the aid of three bioinformatic tools, we identified 20 possible cleavage sites of ATXN3 that were already validated in the literature. From these, 13 fragments were predicted to contain immunogenic peptide cores. Moreover, from the analyses of the cleavage sites prediction, we found more proteases with high possibility to cleave ATXN3 which from here we found 10 fragments that could also contain immunogenic peptide cores and cause putative fragments that could induce toxicity due immune activation. In total, there are 23 fragments of interest, that would be important to study *in vitro* to understand if there is indeed immunogenicity conferred by the toxic fragments.

In conclusion, and despite the limitations, we were able to identify potential ATXN3 peptides for performing further *in vitro* studies to better understand the role of the immune system in MJD.

CHAPTER 7
REFERENCES

REFERENCES

AGRAWAL, Neeraj J. *et al.* - Aggregation in Protein-Based Biotherapeutics: Computational Studies and Tools to Identify Aggregation-Prone Regions. Journal of Pharmaceutical Sciences. ISSN 00223549. 100:12 (2011) 5081–5095. doi: 10.1002/jps.22705.

AHMED, Raija K. S.; MAEURER, Markus J. - T-Cell Epitope Mapping. In SCHUTKOWSKI, MIKE; REINEKE, ULRICH (Eds.) - Epitope Mapping Protocols Methods in Molecular Biology. Totowa, NJ: Humana Press, 2009 [Consulted 8 May 2021]. Available in Internet: http://link.springer.com/10.1007/978-1-59745-450-6_31>. ISBN 978-1-934115-17-6v. 524. p. 427–438.

ALBRECHT, Mario *et al.* - Structural and functional analysis of ataxin-2 and ataxin-3: Analysis of ataxins 2 and 3. European Journal of Biochemistry. ISSN 00142956, 14321033. 271:15 (2004) 3155–3170. doi: 10.1111/j.1432-1033.2004.04245.x.

AMOR, Sandra *et al.* - Inflammation in neurodegenerative diseases. Immunology. ISSN 00192805, 13652567. 129:2 (2010) 154–169. doi: 10.1111/j.1365-2567.2009.03225.x.

AMOR, Sandra; WOODROOFE, M. Nicola - Innate and adaptive immune responses in neurodegeneration and repair. Immunology. ISSN 00192805. 141:3 (2014) 287–291. doi: 10.1111/imm.12134.

ANDREATTA, Massimo; NIELSON, Morten - Gapped sequence alignment using artificial neural networks: application to the MHC class I system. Bioinformatics. 32:4 (2016). 511-517. doi:10.1093/bioinformatics/btv639.

BARIL, L. *et al.* - Pneumococcal surface protein A (PspA) is effective at eliciting T cell-mediated responses during invasive pneumococcal disease in adults. Clinical and Experimental Immunology. ISSN 0009-9104, 1365-2249. 145:2 (2006) 277–286. doi: 10.1111/j.1365-2249.2006.03148.x.

BARUCH, Kuti *et al.* - Breaking immune tolerance by targeting Foxp3+ regulatory T cells mitigates Alzheimer's disease pathology. Nature Communications. ISSN 2041-1723. 6:1 (2015) 7967. doi: 10.1038/ncomms8967.

BAUER, Jan; BIEN, Christian G.; LASSMANN, Hans - Rasmussen's encephalitis: a role for autoimmune cytotoxic T lymphocytes. Current Opinion in Neurology. 15:2 (2002). 197-200. doi:10.1097/00019052-200204000-00012

BERKE, Sarah J. Shoesmith *et al.* - Caspase-mediated proteolysis of the polyglutamine disease protein ataxin-3. Journal of Neurochemistry. ISSN 0022-3042, 1471-4159. 89:4 (2004) 908–918. doi: 10.1111/j.1471-4159.2004.02369.x.

BETTENCOURT, Conceição; LIMA, Manuela - Machado-Joseph Disease: from first

descriptions to new perspectives. Orphanet Journal of Rare Diseases. ISSN 1750-1172. 6:1 (2011) 35. doi: 10.1186/1750-1172-6-35.

BICHELMEIER, U. *et al.* - Nuclear Localization of Ataxin-3 Is Required for the Manifestation of Symptoms in SCA3: *In Vivo* Evidence. Journal of Neuroscience. ISSN 0270-6474, 1529-2401. 27:28 (2007) 7418–7428. doi: 10.1523/JNEUROSCI.4540-06.2007.

BJÖRKQVIST, Maria *et al.* - A novel pathogenic pathway of immune activation detectable before clinical onset in Huntington's disease. Journal of Experimental Medicine. ISSN 1540-9538, 0022-1007. 205:8 (2008) 1869–1877. doi: 10.1084/jem.20080178.

BONILLA, Francisco A.; OETTGEN, Hans C. - Adaptive immunity. Journal of Allergy and Clinical Immunology. ISSN 00916749. 125:2 (2010) S33–S40. doi: 10.1016/j.jaci.2009.09.017.

BRADFORD, Jennifer W.; LI, Shihua; LI, Xiao-Jiang - Polyglutamine toxicity in non-neuronal cells. Cell Research. ISSN 1001-0602, 1748-7838. 20:4 (2010) 400–407. doi: 10.1038/cr.2010.32.

BREUER, Peter *et al.* - Nuclear Aggregation of Polyglutamine-expanded Ataxin-3. Journal of Biological Chemistry. ISSN 00219258. 285:9 (2010) 6532–6537. doi: 10.1074/jbc.M109.036335.

BRYSON, Christine J.; JONES, Tim D.; BAKER, Matthew P. - Prediction of Immunogenicity of Therapeutic Proteins: Validity of Computational Tools. BioDrugs. ISSN 1173-8804. 24:1 (2010) 1–8. doi: 10.2165/11318560-000000000-00000.

BUIJSEN, Ronald A. M. *et al.* - Genetics, Mechanisms, and Therapeutic Progress in Polyglutamine Spinocerebellar Ataxias. Neurotherapeutics. ISSN 1933-7213, 1878-7479. 16:2 (2019) 263–286. doi: 10.1007/s13311-018-00696-y.

BURRIGHT, Eric N. *et al.* - SCA1 transgenic mice: A model for neurodegeneration caused by an expanded CAG trinucleotide repeat. Cell. ISSN 00928674. 82:6 (1995) 937–948. doi: 10.1016/0092-8674(95)90273-2.

CHOO, Sung Yoon - The HLA System: Genetics, Immunology, Clinical Testing, and Clinical Implications. Yonsei Medical Journal. ISSN 0513-5796. 48:1 (2007) 11. doi: 10.3349/ymj.2007.48.1.11.

CICCOCIOPPO, Fausta *et al.* - Neurodegenerative diseases as proteinopathies-driven immune disorders. Neural Regeneration Research. ISSN 1673-5374. 15:5 (2020) 850. doi: 10.4103/1673-5374.268971.

COUTINHO, P.; ANDRADE, C. - Autosomal dominant system degeneration in Portuguese families of the Azores Islands: A new genetic disorder involving cerebellar, pyramidal, extrapyramidal and spinal cord motor functions. Neurology. ISSN 0028-3878, 1526-632X. 28:7 (1978) 703–703. doi: 10.1212/WNL.28.7.703.

CROTTI, Andrea *et al.* - Mutant Huntingtin promotes autonomous microglia activation via myeloid lineage-determining factors. Nature Neuroscience. ISSN 1097-6256, 1546-1726. 17:4 (2014) 513–521. doi: 10.1038/nn.3668.

DANSOKHO, Cira *et al.* - Regulatory T cells delay disease progression in Alzheimer-like pathology. Brain. ISSN 0006-8950, 1460-2156. 139:4 (2016) 1237–1251. doi: 10.1093/brain/awv408.

DEGROOT, A.; MCMURRY, J.; MOISE, L. - Prediction of immunogenicity: in silico paradigms, ex vivo and *in vivo* correlates. Current Opinion in Pharmacology. ISSN 14714892. 8:5 (2008) 620–626. doi: 10.1016/j.coph.2008.08.002.

DHANDA, Sandeep Kumar *et al.* - Predicting HLA CD4 Immunogenicity in Human Populations. Frontiers in Immunology. ISSN 1664-3224. 9 (2018) 1369. doi: 10.3389/fimmu.2018.01369.

DOSS-PEPE, Ellen W. *et al.* - Ataxin-3 Interactions with Rad23 and Valosin-Containing Protein and Its Associations with Ubiquitin Chains and the Proteasome Are Consistent with a Role in Ubiquitin-Mediated Proteolysis. Molecular and Cellular Biology. ISSN 0270-7306, 1098-5549. 23:18 (2003) 6469–6483. doi: 10.1128/MCB.23.18.6469-6483.2003.

ELLRICHMANN, Gisa *et al.* - The Role of the Immune System in Huntington's Disease. Clinical and Developmental Immunology. ISSN 1740-2522, 1740-2530. (2013) 1–11. doi: 10.1155/2013/541259.

EVERS, Melvin M.; TOONEN, Lodewijk J. A.; ROON-MOM, Willeke M. C. VAN - Ataxin-3 Protein and RNA Toxicity in Spinocerebellar Ataxia Type 3: Current Insights and Emerging Therapeutic Strategies. Molecular Neurobiology. ISSN 0893-7648, 1559-1182. (2013). doi: 10.1007/s12035-013-8596-2.

EVERT, Bernd O. *et al.* - Inflammatory Genes Are Upregulated in Expanded Ataxin-3-Expressing Cell Lines and Spinocerebellar Ataxia Type 3 Brains. The Journal of Neuroscience. ISSN 0270-6474, 1529-2401. 21:15 (2001) 5389–5396. doi: 10.1523/JNEUROSCI.21-15-05389.2001.

EVERT, Bernd O. *et al.* - Gene Expression Profiling in Ataxin-3 Expressing Cell Lines Reveals Distinct Effects of Normal and Mutant Ataxin-3. Journal of Neuropathology & Experimental Neurology. ISSN 0022-3069, 1554-6578. 62:10 (2003) 1006–1018. doi: 10.1093/jnen/62.10.1006.

FAN, Hueng Chuen *et al.* - Polyglutamine (PolyQ) diseases: Genetics to treatments. Cell Transplantation. ISSN 09636897. 23:4–5 (2014) 441–458. doi: 10.3727/096368914X678454.

FAN, Hueng-Chuen *et al.* - Polyglutamine (PolyQ) Diseases: Genetics to Treatments. Cell Transplantation. ISSN 0963-6897, 1555-3892. 23:4–5 (2014) 441–458. doi:

10.3727/096368914X678454.

FISZER, Agnieszka; KRZYZOSIAK, Wlodzimierz J. - RNA toxicity in polyglutamine disorders: concepts, models, and progress of research. Journal of Molecular Medicine. ISSN 0946-2716, 1432-1440. 91:6 (2013) 683–691. doi: 10.1007/s00109-013-1016-2.

GAFNI, Juliette; ELLERBY, Lisa M. - Calpain Activation in Huntington's Disease. The Journal of Neuroscience. ISSN 0270-6474, 1529-2401. 22:12 (2002) 4842–4849. doi: 10.1523/JNEUROSCI.22-12-04842.2002.

GAO, Rui *et al.* - Inactivation of PNKP by Mutant ATXN3 Triggers Apoptosis by Activating the DNA Damage-Response Pathway in SCA3. PLOS Genetics. ISSN 1553-7404. 11:1 (2015) e1004834. doi: 10.1371/journal.pgen.1004834.

GARDE, Martijn D. B. VAN DE *et al.* - Prediction and Validation of Immunogenic Domains of Pneumococcal Proteins Recognized by Human CD4 $^{+}$ T Cells. Infection and Immunity. ISSN 0019-9567, 1098-5522. 87:6 (2019). doi: 10.1128/IAI.00098-19.

GATCHEL, Jennifer R.; ZOGHBI, Huda Y. - Diseases of Unstable Repeat Expansion: Mechanisms and Common Principles. Nature Reviews Genetics. ISSN 1471-0056, 1471-0064. 6:10 (2005) 743–755. doi: 10.1038/nrg1691.

GOLDBERG, Y. P. *et al.* - Cleavage of huntingtin by apopain, a proapoptotic cysteine protease, is modulated by the polyglutamine tract. Nature Genetics. ISSN 1061-4036, 1546-1718. 13:4 (1996) 442–449. doi: 10.1038/ng0896-442.

GOLDE, Todd E.; MILLER, Victor M. - Proteinopathy-induced neuronal senescence: a hypothesis for brain failure in Alzheimer's and other neurodegenerative diseases. Alzheimer's Research & Therapy. ISSN 1758-9193. 1:2 (2009) 5. doi: 10.1186/alzrt5.

GONÇALVES, Nélio *et al.* - Caffeine and adenosine A $_{2A}$ receptor inactivation decrease striatal neuropathology in a lentiviral-based model of Machado-Joseph disease: Caffeine Alleviates MJD. Annals of Neurology. ISSN 03645134. 73:5 (2013) 655–666. doi: 10.1002/ana.23866.

GOTI, D. - A Mutant Ataxin-3 Putative-Cleavage Fragment in Brains of Machado-Joseph Disease Patients and Transgenic Mice Is Cytotoxic above a Critical Concentration. Journal of Neuroscience. ISSN 0270-6474, 1529-2401. 24:45 (2004) 10266–10279. doi: 10.1523/JNEUROSCI.2734-04.2004.

GREENBAUM, Jason *et al.* - Functional classification of class II human leukocyte antigen (HLA) molecules reveals seven different supertypes and a surprising degree of repertoire sharing across supertypes. Immunogenetics. ISSN 0093-7711, 1432-1211. 63:6 (2011) 325–335. doi: 10.1007/s00251-011-0513-0.

HAACKE, Annette *et al.* - Proteolytic cleavage of polyglutamine-expanded ataxin-3 is

critical for aggregation and sequestration of non-expanded ataxin-3. Human Molecular Genetics. ISSN 1460-2083, 0964-6906. 15:4 (2006) 555–568. doi: 10.1093/hmg/ddi472.

HAVEL, Lauren S.; LI, Shihua; LI, Xiao-Jiang - Nuclear accumulation of polyglutamine disease proteins and neuropathology. Molecular Brain. ISSN 1756-6606. 2:1 (2009) 21. doi: 10.1186/1756-6606-2-21.

HOPP, T. P.; WOOD, K. R. - A computer program for predicting protein antigenic determinants. Molecular Immunology. 20:4 (1983). 483-489. doi:10.1016/0161-5890(83)90029-9.

HOWE, Charles L. *et al.* - CD8+ T cells directed against a viral peptide contribute to loss of motor function by disrupting axonal transport in a viral model of fulminant demyelination. Journal of Neuroimmunology. ISSN 01655728. 188:1–2 (2007) 13–21. doi: 10.1016/j.jneuroim.2007.04.005.

HUANG, Xiuyan *et al.* - CD 4+ T cells in the pathobiology of neurodegenerative disorders. Journal of Neuroimmunology. ISSN 01655728. 211:1–2 (2009) 3–15. doi: 10.1016/j.jneuroim.2009.04.006.

HUBENER, J. *et al.* - Calpain-mediated ataxin-3 cleavage in the molecular pathogenesis of spinocerebellar ataxia type 3 (SCA3). Human Molecular Genetics. ISSN 0964-6906, 1460-2083. 22:3 (2013) 508–518. doi: 10.1093/hmg/dds449.

HUIZINGA, R. *et al.* - T-cell responses to neurofilament light protein are part of the normal immune repertoire. International Immunology. ISSN 0953-8178, 1460-2377. 21:4 (2009) 433–441. doi: 10.1093/intimm/dxp011.

ICHIKAWA, Y. *et al.* - The genomic structure and expression of MJD, the Machado-Joseph disease gene. Journal of Human Genetics. ISSN 1434-5161, 1435-232X. 46:7 (2001) 413–422. doi: 10.1007/s100380170060.

IKEDA, H. *et al.* - Expanded polyglutamine in the Machado-Joseph disease protein induces cell death in vitro and *in vivo*. 13:2 (1996) 196–202. doi: 10.1038/ng0696-196.

JARDIM, A. *et al.* - Immunoprotective Leishmania major synthetic T cell epitopes. Journal of Experimental Medicine. ISSN 0022-1007, 1540-9538. 172:2 (1990) 645–648. doi: 10.1084/jem.172.2.645.

JAWA, Vibha *et al.* - T-cell dependent immunogenicity of protein therapeutics: Preclinical assessment and mitigation. Clinical Immunology. ISSN 15216616. 149:3 (2013) 534–555. doi: 10.1016/j.clim.2013.09.006.

KAWAGUCHI, Yoshiya *et al.* - CAG expansions in a novel gene for Machado-Joseph disease at chromosome 14q32.1. Nature Genetics. ISSN 1061-4036, 1546-1718. 8:3 (1994) 221–228. doi: 10.1038/ng1194-221.

KIM, Seung Hyun *et al.* - Immune inflammatory modulation as a potential therapeutic strategy of stem cell therapy for ALS and neurodegenerative diseases. BMB Reports. ISSN 1976-670X. 51:11 (2018) 545–546. doi: 10.5483/BMBRep.2018.51.11.255.

KOEPPEN, Arnulf H. - The Neuropathology of Spinocerebellar Ataxia Type 3/Machado-Joseph Disease. In NÓBREGA, CLÉVIO; PEREIRA DE ALMEIDA, LUÍS (Eds.) - Polyglutamine Disorders Advances in Experimental Medicine and Biology. Cham: Springer International Publishing, 2018 [Consulted 6 November 2020]. Available in Internet: http://link.springer.com/10.1007/978-3-319-71779-1_11>. ISBN 978-3-319-71778-4v. 1049. p. 233–241.

KUMAR, Sandeep *et al.* - Autoimmune Responses to Soluble Aggregates of Amyloidogenic Proteins Involved in Neurodegenerative Diseases: Overlapping Aggregation Prone and Autoimmunogenic regions. Scientific Reports. ISSN 2045-2322. 6:1 (2016) 22258. doi: 10.1038/srep22258.

KWAN, Wanda *et al.* - Mutant huntingtin impairs immune cell migration in Huntington disease. Journal of Clinical Investigation. ISSN 0021-9738. 122:12 (2012) 4737–4747. doi: 10.1172/JCI64484.

KYE, Yoon-Chul *et al.* - Intranasal immunization with pneumococcal surface protein A in the presence of nanoparticle forming polysorbitol transporter adjuvant induces protective immunity against the Streptococcus pneumoniae infection. Acta Biomaterialia. ISSN 17427061. 90:2019 (2019) 362–372. doi: 10.1016/j.actbio.2019.03.049.

LARSEN, Mette V. *et al.* - Large-scale validation of methods for cytotoxic T-lymphocyte epitope prediction. BMC Bioinformatics. 8:424 (2007). doi:10.1186/1471-2105-8-424.

LI, Fusheng *et al.* - Ataxin-3 Is a Histone-binding Protein with Two Independent Transcriptional Corepressor Activities. Journal of Biological Chemistry. ISSN 00219258. 277:47 (2002) 45004–45012. doi: 10.1074/jbc.M205259200.

LIANG, Zhanfeng *et al.* - Impact of aging immune system on neurodegeneration and potential immunotherapies. Progress in Neurobiology. ISSN 03010082. 157:2017 (2017) 2–28. doi: 10.1016/j.pneurobio.2017.07.006.

LIEBERMAN, Andrew P.; SHAKKOTTAI, Vikram G.; ALBIN, Roger L. - Polyglutamine Repeats in Neurodegenerative Diseases. Annual Review of Pathology: Mechanisms of Disease. ISSN 1553-4006, 1553-4014. 14:1 (2019) 1–27. doi: 10.1146/annurev-pathmechdis-012418-012857.

MARSHALL, Jean S. *et al.* - An introduction to immunology and immunopathology. Allergy, Asthma & Clinical Immunology. ISSN 1710-1492. 14:S2 (2018) 49. doi: 10.1186/s13223-018-0278-1.

MARTÍ, Eulália - RNA toxicity induced by expanded CAG repeats in Huntington's disease: RNA toxicity. Brain Pathology. ISSN 10156305. 26:6 (2016) 779–786. doi: 10.1111/bpa.12427.

MATOS, Carlos A.; MACEDO-RIBEIRO, Sandra DE; CARVALHO, Ana Luísa - Polyglutamine diseases: The special case of ataxin-3 and Machado-Joseph disease. Progress in Neurobiology. ISSN 03010082. 95:1 (2011) 26–48. doi: 10.1016/j.pneurobio.2011.06.007.

MATOS, Carlos A.; MACEDO-RIBEIRO, Sandra DE; CARVALHO, Ana Luísa - Polyglutamine diseases: The special case of ataxin-3 and Machado–Joseph disease. Progress in Neurobiology. ISSN 03010082. 95:1 (2011) 26–48. doi: 10.1016/j.pneurobio.2011.06.007.

MCGEER, P. L.; MCGEER, E. G. - Inflammatory processes in amyotrophic lateral sclerosis. Muscle & Nerve. ISSN 0148-639X, 1097-4598. 26:4 (2002) 459–470. doi: 10.1002/mus.10191.

MEDANA, Isabelle *et al.* - Transection of Major Histocompatibility Complex Class I-Induced Neurites by Cytotoxic T Lymphocytes. The American Journal of Pathology. ISSN 00029440. 159:3 (2001) 809–815. doi: 10.1016/S0002-9440(10)61755-5.

MOORE, Keith; AGUR, Anne; DALLEY, Arthur F. - Clinically oriented anatomy. 7. ed. Philadelphia: Lippincott Williams and Wilkins, 2013. ISBN 978-1-4511-8745-8.

MÜLLER, I. *et al.* - Gamma interferon response in secondary Leishmania major infection: role of CD8+ T cells. Infection and Immunity. ISSN 0019-9567, 1098-5522. 61:9 (1993) 3730–3738. doi: 10.1128/iai.61.9.3730-3738.1993.

NAKANO, K. K.; DAWSON, D. M.; SPENCE, A. - Machado disease: A hereditary ataxia in Portuguese emigrants to Massachusetts. Neurology. ISSN 0028-3878, 1526-632X. 22:1 (1972) 49–49. doi: 10.1212/WNL.22.1.49.

NIJMAN, Sebastian M. B. *et al.* - A Genomic and Functional Inventory of Deubiquitinating Enzymes. Cell. ISSN 00928674. 123:5 (2005) 773–786. doi: 10.1016/j.cell.2005.11.007.

NÓBREGA, Clévio *et al.* - Molecular Mechanisms and Cellular Pathways Implicated in Machado-Joseph Disease Pathogenesis. In NÓBREGA, CLÉVIO; PEREIRA DE ALMEIDA, LUÍS (Eds.) - Polyglutamine Disorders Advances in Experimental Medicine and Biology. Cham: Springer International Publishing, 2018. [Consulted 6 November 2020]. Available in Internet: http://link.springer.com/10.1007/978-3-319-71779-1_18>. ISBN 978-3-319-71778-4v. 1049. p. 349–367.

OKEKE, Emeka B.; UZONNA, Jude E. - The Pivotal Role of Regulatory T Cells in the Regulation of Innate Immune Cells. Frontiers in Immunology. ISSN 1664-3224. 10:2019 (2019) 680. doi: 10.3389/fimmu.2019.00680.

OLEJNICZAK, Marta; URBANEK, Martyna O.; KRZYZOSIAK, Wlodzimierz J. - The Role of the Immune System in Triplet Repeat Expansion Diseases. Mediators of Inflammation. ISSN 0962-9351, 1466-1861. (2015) 1–11. doi: 10.1155/2015/873860.

PAUL, Sinu *et al.* - Development and validation of a broad scheme for prediction of HLA class II restricted T cell epitopes. Journal of Immunological Methods. ISSN 00221759. 422 (2015) 28–34. doi: 10.1016/j.jim.2015.03.022.

PAUL, Sinu *et al.* - TepiTool: A Pipeline for Computational Prediction of T Cell Epitope Candidates. Current Protocols in Immunology. ISSN 1934-3671, 1934-368X. 114:1 (2016). doi: 10.1002/cpim.12.

PAULSON, Henry L. *et al.* - Intranuclear Inclusions of Expanded Polyglutamine Protein in Spinocerebellar Ataxia Type 3. Neuron. ISSN 08966273. 19:2 (1997) 333–344. doi: 10.1016/S0896-6273(00)80943-5.

PAULSON, Henry L. *et al.* - Machado-Joseph disease gene product is a cytoplasmic protein widely expressed in brain. Annals of Neurology. ISSN 0364-5134, 1531-8249. 41:4 (1997) 453–462. doi: 10.1002/ana.410410408.

PAVESE, N. *et al.* - Microglial activation correlates with severity in Huntington disease: A clinical and PET study. Neurology. ISSN 0028-3878, 1526-632X. 66:11 (2006) 1638–1643. doi: 10.1212/01.wnl.0000222734.56412.17.

PORCELLI, Steven A. - Innate Immunity. In Kelley and Firestein's Textbook of Rheumatology. [S.l.]: Elsevier, 2017 [Consulted 11 December 2021]. Available in Internet: https://linkinghub.elsevier.com/retrieve/pii/B9780323316965000176>. ISBN 978-0-323-31696-5. p. 274–287.

REYNOLDS, Ashley D. *et al.* - Proteomic Studies of Nitrated Alpha-Synuclein Microglia Regulation by CD4+CD25+ T Cells. Journal of Proteome Research. ISSN 1535-3893, 1535-3907. 8:7 (2009) 3497–3511. doi: 10.1021/pr9001614.

REZVAN, H.; REES, R.; ALI, S. A. - Immunogenicity of MHC Class I Peptides Derived from Leishmania mexicana Gp63 in HLA-A2.1 Transgenic (HHDII) and BALB/C Mouse Models. Iranian journal of parasitology. 7:4 (2012) 27-40.

RODRIGUES, Ana-João *et al.* - Absence of ataxin-3 leads to cytoskeletal disorganization and increased cell death. Biochimica et Biophysica Acta (BBA) - Molecular Cell Research. ISSN 01674889. 1803:10 (2010) 1154–1163. doi: 10.1016/j.bbamcr.2010.07.004.

RODRIGUES, Maria C. O. *et al.* - The innate and adaptive immunological aspects in neurodegenerative diseases. Journal of Neuroimmunology. ISSN 01655728. 269:1–2 (2014) 1–8. doi: 10.1016/j.jneuroim.2013.09.020.

ROSENBERG, R. N. *et al.* - Autosomal dominant striatonigral degeneration: A clinical, pathologic, and biochemical study of a new genetic disorder. Neurology. ISSN 0028-3878, 1526-632X. 26:8 (1976) 703–703. doi: 10.1212/WNL.26.8.703.

SALMINEN, Antero *et al.* - Inflammation in Alzheimer's disease: Amyloid-β oligomers

trigger innate immunity defence via pattern recognition receptors. Progress in Neurobiology. ISSN 03010082. 87:3 (2009) 181–194. doi: 10.1016/j.pneurobio.2009.01.001.

SANCHEZ-TRINCADO, Jose L.; GOMEZ-PEROSANZ, Marta; RECHE, Pedro A. - Fundamentals and Methods for T- and B-Cell Epitope Prediction. Journal of Immunology Research. ISSN 2314-8861, 2314-7156. (2017) 1–14. doi: 10.1155/2017/2680160.

SCHMIDT, Thorsten *et al.* - Protein surveillance machinery in brains with spinocerebellar ataxia type 3: Redistribution and differential recruitment of 26S proteasome subunits and chaperones to neuronal intranuclear inclusions: Proteasome and Chaperones in SCA3. Annals of Neurology. ISSN 03645134. 51:3 (2002) 302–310. doi: 10.1002/ana.10101.

SCHWARTZ, Michal; DECZKOWSKA, Aleksandra - Neurological Disease as a Failure of Brain–Immune Crosstalk: The Multiple Faces of Neuroinflammation. Trends in Immunology. ISSN 14714906. 37:10 (2016) 668–679. doi: 10.1016/j.it.2016.08.001.

SCOTT, Stephanie Suzanne De Oliveira *et al.* - Natural history and epidemiology of the spinocerebellar ataxias: Insights from the first description to nowadays. Journal of the Neurological Sciences. ISSN 0022510X. 417 (2020) 117082. doi: 10.1016/j.jns.2020.117082.

SHAO, J.; DIAMOND, M. I. - Polyglutamine diseases: emerging concepts in pathogenesis and therapy. Human Molecular Genetics. ISSN 0964-6906, 1460-2083. 16:R2 (2007) R115–R123. doi: 10.1093/hmg/ddm213.

SHIN, Ji-Yeon *et al.* - Expression of mutant huntingtin in glial cells contributes to neuronal excitotoxicity. Journal of Cell Biology. ISSN 1540-8140, 0021-9525. 171:6 (2005) 1001–1012. doi: 10.1083/jcb.200508072.

SIMMONS, Danielle A. *et al.* - Ferritin accumulation in dystrophic microglia is an early event in the development of Huntington's disease. Glia. ISSN 08941491, 10981136. 55:10 (2007) 1074–1084. doi: 10.1002/glia.20526.

SIMÕES, Ana Teresa *et al.* - Calpain inhibition reduces ataxin-3 cleavage alleviating neuropathology and motor impairments in mouse models of Machado–Joseph disease. Human Molecular Genetics. ISSN 0964-6906, 1460-2083. 23:18 (2014) 4932–4944. doi: 10.1093/hmg/ddu209.

SIMÕES, Ana Teresa *et al.* - Identification of the calpain-generated toxic fragment of ataxin-3 protein provides new avenues for therapy of Machado–Joseph disease Spinocerebellar ataxia type 3. Neuropathology and Applied Neurobiology. ISSN 0305-1846, 1365-2990. (2021) 12748. doi: 10.1111/nan.12748.

SINGHRAO, S. K. *et al.* - Increased Complement Biosynthesis by Microglia and Complement Activation on Neurons in Huntington's Disease. Experimental Neurology. ISSN 00144886. 159:2 (1999) 362–376. doi: 10.1006/exnr.1999.7170.

SONG, Jiangning *et al.* - iProt-Sub: a comprehensive package for accurately mapping and predicting protease-specific substrates and cleavage sites. Briefings in Bioinformatics. ISSN 1477-4054. 20:2 (2019) 638–658. doi: 10.1093/bib/bby028.

STOREY, Mel; JORDAN, Sue - An overview of the immune system. Nursing Standard. ISSN 0029-6570, 2047-9018. 23:15 (2008) 47–56. doi: 10.7748/ns2008.12.23.15.47.c6738.

STOYAS, Colleen A.; LA SPADA, Albert R. - The CAG–polyglutamine repeat diseases: a clinical, molecular, genetic, and pathophysiologic nosology. In Handbook of Clinical Neurology. [S.l.]: Elsevier, 2018 [Consulted 6 November 2020]. Available in Internet: https://linkinghub.elsevier.com/retrieve/pii/B9780444632333000117>. ISBN 978-0-444-63233-3v. 147. p. 143–170.

TAKEUCHI, Toshihide; NAGAI, Yoshitaka - Protein Misfolding and Aggregation as a Therapeutic Target for Polyglutamine Diseases. Brain Sciences. ISSN 2076-3425. 7:12 (2017) 128. doi: 10.3390/brainsci7100128.

TAKIYAMA, Y. *et al.* - The gene for Machado–Joseph disease maps to human chromosome 14q. Nature Genetics. ISSN 1061-4036, 1546-1718. 4:3 (1993) 300–304. doi: 10.1038/ng0793-300.

TARLAC, V.; STOREY, E. - Role of proteolysis in polyglutamine disorders. Journal of Neuroscience Research. ISSN 0360-4012, 1097-4547. 74:3 (2003) 406–416. doi: 10.1002/jnr.10746.

THAMEEM DHEEN, S.; KAUR, Charanjit; LING, Eng-Ang - Microglial Activation and its Implications in the Brain Diseases. Current Medicinal Chemistry. ISSN 09298673. 14:11 (2007) 1189–1197. doi: 10.2174/092986707780597961.

TODI, Sokol V. *et al.* - Ubiquitination directly enhances activity of the deubiquitinating enzyme ataxin-3. The EMBO Journal. ISSN 0261-4189, 1460-2075. 28:4 (2009) 372–382. doi: 10.1038/emboj.2008.289.

TRÄGER, Ulrike *et al.* - HTT-lowering reverses Huntington's disease immune dysfunction caused by NFκB pathway dysregulation. Brain. ISSN 1460-2156, 0006-8950. 137:3 (2014) 819–833. doi: 10.1093/brain/awt355.

TZVETKOV, Nikolay; BREUER, Peter - Josephin domain-containing proteins from a variety of species are active de-ubiquitination enzymes. Biological Chemistry. ISSN 14374315, 14316730. 388:9 (2007) 973–978. doi: 10.1515/BC.2007.107.

VITA, R. *et al.* - The Immune Epitope Database (IEDB): 2018 update. Nucleic Acids Research. 47(D1) (2019). D339-D343. doi: 10.1093/nar/gky1006.

WEBER, Jonasz J. *et al.* - A combinatorial approach to identify calpain cleavage sites in the Machado-Joseph disease protein ataxin-3. Brain. ISSN 0006-8950, 1460-2156. 140:5 (2017)

1280–1299. doi: 10.1093/brain/awx039.

WEBER, Jonasz Jeremiasz *et al.* - From Pathways to Targets: Understanding the Mechanisms behind Polyglutamine Disease. BioMed Research International. ISSN 2314-6133, 2314-6141. (2014) 1–22. doi: 10.1155/2014/701758.

WELLINGTON, Cheryl L. *et al.* - Caspase Cleavage of Gene Products Associated with Triplet Expansion Disorders Generates Truncated Fragments Containing the Polyglutamine Tract. Journal of Biological Chemistry. ISSN 00219258. 273:15 (1998) 9158–9167. doi: 10.1074/jbc.273.15.9158.

WELLINGTON, Cheryl L. *et al.* - Inhibiting Caspase Cleavage of Huntingtin Reduces Toxicity and Aggregate Formation in Neuronal and Nonneuronal Cells. Journal of Biological Chemistry. ISSN 00219258. 275:26 (2000) 19831–19838. doi: 10.1074/jbc.M001475200.

WELLINGTON, Cheryl L.; HAYDEN, Michael R. - Caspases and neurodegeneration: on the cutting edge of new therapeutic approaches: Caspases and neurodegeneration. Clinical Genetics. ISSN 00099163. 57:1 (2000) 1–10. doi: 10.1034/j.1399-0004.2000.570101.x.

WRAITH, David C.; NICHOLSON, Lindsay B. - The adaptive immune system in diseases of the central nervous system. Journal of Clinical Investigation. ISSN 0021-9738. 122:4 (2012) 1172–1179. doi: 10.1172/JCI58648.

YANG, Xingdong; YU, Xinglong - An introduction to epitope prediction methods and software: Epitope prediction methods and software. Reviews in Medical Virology. ISSN 10529276. 19:2 (2009) 77–96. doi: 10.1002/rmv.602.

YUSHCHENKO, Tetyana; DEUERLING, Elke; HAUSER, Karin - Insights into the Aggregation Mechanism of PolyQ Proteins with Different Glutamine Repeat Lengths. Biophysical Journal. ISSN 00063495. 114:8 (2018) 1847–1857. doi: 10.1016/j.bpj.2018.02.037.

CHAPTER 8
SUPPLEMENTARY DATA

Figure I – Sequence of proteins in FASTA format.

Acquired at https://www.ncbi.nlm.nih.gov/.

```
>NP_004984.2 ataxin-3 reference isoform [Homo sapiens]

MESIFHEKQEGSLCAQHCLNNLLQGEYFSPVELSSIAHQLDEEERMRMAEGGVTSEDYRTFLQQPSGNMDDSGFFSIQVISNALKVWGLELILF
NSPEYQRLRIDPINERSFICNYKEHWFTVRKLGKQWFNLNSLLTGPELISDTYLALFLAQLQQEGYSIFVVKGDLPDCEADQLLQMIRVQQMHR
PKLIGEELAQLKEQRVHKTDLERVLEANDGSGMLDEDEEDLQRALALSRQEIDMEDEEADLRRAIQLSMQGSSRNISQDMTQTSGTNLTSEELR
KRREAYFEKQQQKQQQQQQQQQQGDLSGQSSHPCERPATSSGALGSDLGDAMSEEDMLQAAVTMSLETVRNDLKTEGKK
```

```
>AAC41702.1 interferon-beta [Homo sapiens]

FQKEDAAVTIYEMLQNIFAIFRQDSSSTGWNETIVENLLANVYHQRNHLKTVLEEKLEKEDFTRGKRMSSLHLKRYYGRILHYLKAKEDSHCAWT
IVRVEILRNFYVINRLTGYLRN
```

```
>CAA26095.1 erythropoietin [Homo sapiens]

TKVNFYAWKRMEVGQQAVEVWQGLALLSEAVLRGQALLVNSSQPWEPLQLHVDKAVSGLRSLTTLLRALGAQKEAISPPDAASAAPLRTITADTF
RKLFRVYSNFLRGKLKLYTGEACRTGDR
```

```
>AAH98109.1 Growth hormone releasing hormone [Homo sapiens]

MPLWVFFFVILTLSNSSHCSPPPPLTLRMRRYADAIFTNSYRKVLGQLSARKLLQDIMSRQQGESNQERGARARLGRQVDSMWAEQKQMELESILV
ALLQKHSRNSQG
```

```
>AAA98797.1 albumin [Homo sapiens]

MKWVTFISLLFLFSSAYSRGVFRRDAHKSEVAHRFKDLGEENFKALVLIAFAQYLQQCPFEDHVKLVNEVTEFAKTCVADESAENCDKSLHTLFGD
KLCTVATLRETYGEMADCCAKQEPERNECFLQHKDDNPNLPRLVRPEVDVMCTAFHDNEETFLKKYLYEIARRHPYFYAPELLFFAKRYKAAFTEC
CQAADKAACLLPKLDELRDEGKASSAKQRLKCASLQKFGERAFKAWAVARLSQRFPKAEFAEVSKLVTDLTKVHTECCHGDLLECADDRADLAKYI
CENQDSISSKLKECCEKPLLEKSHCIAEVENDEMPADLPSLAADFVESKDVCKNYAEAKDVFLGMFLYEYARRHPDYSVVLLLRLAKTYETTLEKC
CAAADPHECYAKVFDEFKPLVEEPQNLIKQNCELFEQLGEYKFQNALLVRYTKKVPQVSTPTLVEVSRNLGKVGSKCCKHPEAKRMPCAEDYLSVV
LNQLCVLHEKTPVSDRVTKCCTESLVNRRPCFSALEVDETYVPKEFNAETFTFHADICTLSEKERQIKKQTALVELVKHKPKATKEQLKAVMDDFA
AFVEKCCKADDKETCFAEEGKKLVAASQAALGL
```

```
>AAA51811.1 beta-2-microglobulin [Homo sapiens]

MSRSVALAVLALLSLSGLEGIQRTPKIQVYSRHPAENGKSNFLNCYVSGFHQSDIEVDLLKNGERIEKVEHSDLSFSKDWSFYLLYYTEFTPTEKDEY
ACRVNHVTLSQPKIVKWDRDM
```

```
>AAK74703.1 BlpZ protein, fusion [Streptococcus pneumoniae TIGR4]

MYKHLFFLDSKTLDRLTPYILVLASDTIAFNVFVLTFVSAVVFNFLNSMLALMAIFIGAGYVVGFWLLILNENQRAN
```

```
>AAK75991.1 pneumolysin [Streptococcus pneumoniae TIGR4]

MANKAVNDFILAMNYDKKKLLTHQGESIENRFIKEGNQLPDEFVVIERKKRSLSTNTSDISVTATNDSRLYPGALLVVDETLLENNPTLLAVDRA
PMTYSIDLPGLASSDSFLQVEDPSNSSVRGAVNDLLAKWHQDYGQVNNVPARMQYEKITAHSMEQLKVKFGSDFEKTGNSLDIDFNSVHSGEKQI
QIVNFKQIYYTVSVDAVKNPGDVFQDTVTVEDLKQRGISAERPLVYISSVAYGRQVYLKLETTSKSDEVEAAFEALIKGVKVAPQTEWKQILDNT
EVKAVILGGDPSSGARVVTGKVDMVEDLIQEGSRFTADHPGLPISYTTSFLRDNVVATFQNSTDYVETKVTAYRNGDLLLDHSGAYVAQYYITWN
ELSYDHQGKEVLTPKAWDRNGQDLTAHFTTSIPLKGNVRNLSVKIRECTGLAWEWWRTVYEKTDLPLVRKRTISIWGTTLYPQVEDKVEND
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>AAK74303.1 pneumococcal surface protein A [Streptococcus pneumoniae TIGR4]

MNKKKMILTSLASVAILGAGFVTSQPTFVRAEESPQVVEKSSLEKKYEEAKAKADTAKKDYETAKKKAEDAQKKYEDDQKRTEEKARKEAEASQKL
NDVALVVQNAYKEYREVQNQRSKYKSDAEYQKKLTEVDSKIEKARKEQQDLQNKFNEVRAVVVPEPNALAETKKKAEEAKAEEKVAKRKYDYATLK
VALAKKEVEAKELEIEKLQYEISTLEQEVATAQHQVDNLKKLLAGADPDDGTEVIEAKLKKGEAELNAKQAELAKKQTELEKLLDSLDPEGKTQDE
LDKEAEEAELDKKADELQNKVADLEKEISNLEILLGGADPEDDTAALQNKLAAKKAELAKKQTELEKLLDSLDPEGKTQDELDKEAEEAELDKKAD
ELQNKVADLEKEISNLEILLGGADSEDDTAALQNKLATKKAELEKTQKELDAALNELGPDGDEEETPAPAPQPEQPAPAPKPEQPAPAPKPEQPAP
APKPEQPAPAPKPEQPAPAPKPEQPAKPEKPAEEPTQPEKPATPKTGWKQENGMWYFYNTDGSMAIGWLQNNGSWYYLNANGAMATGWVKDGDTWY
YLEASGAMKASQWFKVSDKWYYVNSNGAMATGWLQYNGSWYYLNANGDMATGWLQYNGSWYYLNANGDMATGWAKVNGSWYYLNANGAMATGWAKV
NGSWYYLNANGSMATGWVKDGDTWYYLEASGAMKASQWFKVSDKWYYVNGLGALAVNTTVDGYKVNANGEWV
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Table 1 – Results of the full-length and mutant ATXN3-MHC-I binding prediction. Acquired with TepiTool (http://tools.iedb.org/tepitool/).

ATXN-3 MHC-I				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILLE RANK	ALLELE
182	190	RVQQMHRPK	0.01	HLA-A*30:01
155	163	LQQEGYSIF	0.01	HLA-B*15:01
188	196	RPKLIGEEL	0.01	HLA-B*07:02
318	326	RPATSSGAL	0.01	HLA-B*07:02
25	33	GEYFSPVEL	0.01	HLA-B*40:01
287	295	AYFEKQQQK	0.03	HLA-A*30:01
245	253	EEADLRRAI	0.03	HLA-B*44:02
248	255	DLRRAIQL	0.04	HLA-B*08:01
32	40	ELSSIAHQL	0.04	HLA-A*68:02
101	110	RLRIDPINER	0.04	HLA-A*31:01
245	253	EEADLRRAI	0.04	HLA-B*44:03
178	186	LQMIRVQQM	0.05	HLA-B*08:01
86	94	VWGLELILF	0.05	HLA-A*23:01
190	199	KLIGEELAQL	0.05	HLA-A*02:01
110	120	RSFICNYKEHW	0.05	HLA-B*57:01
121	130	FTVRKLGKQW	0.05	HLA-B*57:01
115	124	NYKEHWFTVR	0.06	HLA-A*33:01
86	94	VWGLELILF	0.06	HLA-A*24:02
24	33	QGEYFSPVEL	0.06	HLA-B*40:01
53	61	VTSEDYRTF	0.06	HLA-B*58:01
85	93	KVWGLELIL	0.07	HLA-A*32:01
85	94	KVWGLELILF	0.07	HLA-A*32:01
93	101	LFNSPEYQR	0.07	HLA-A*33:01
49	58	AEGGVTSEDY	0.07	HLA-B*44:03
31	40	VELSSIAHQL	0.07	HLA-B*40:01
198	206	QLKEQRVHK	0.08	HLA-A*30:01
228	235	DLQRALAL	0.08	HLA-B*08:01
195	203	ELAQLKEQR	0.08	HLA-A*33:01
137	147	LTGPELISDTY	0.08	HLA-A*01:01
139	147	GPELISDTY	0.08	HLA-B*35:01
110	120	RSFICNYKEHW	0.08	HLA-B*58:01
53	61	VTSEDYRTF	0.08	HLA-B*57:01
29	36	SPVELSSI	0.09	HLA-B*51:01
49	58	AEGGVTSEDY	0.09	HLA-B*44:02
190	199	KLIGEELAQL	0.09	HLA-A*02:03
195	203	ELAQLKEQR	0.09	HLA-A*68:01
122	130	TVRKLGKQW	0.09	HLA-B*57:01
128	136	KQWFNLNSL	0.10	HLA-A*32:01
147	155	YLALFLAQL	0.10	HLA-A*02:03
190	199	KLIGEELAQL	0.10	HLA-A*02:06
93	101	LFNSPEYQR	0.10	HLA-A*31:01
128	136	KQWFNLNSL	0.11	HLA-A*02:06
147	155	YLALFLAQL	0.11	HLA-A*02:01
178	186	LQMIRVQQM	0.11	HLA-B*15:01
79	87	VISNALKVW	0.11	HLA-B*57:01
201	209	EQRVHKTDL	0.12	HLA-B*08:01
78	86	QVISNALKV	0.12	HLA-A*68:02
182	190	RVQQMHRPK	0.12	HLA-A*03:01
79	87	VISNALKVW	0.12	HLA-B*58:01
188	196	RPKLIGEEL	0.13	HLA-B*08:01
115	123	NYKEHWFTV	0.13	HLA-A*24:02
96	104	SPEYQRLRI	0.13	HLA-B*07:02
187	196	HRPKLIGEEL	0.13	HLA-B*07:02
203	211	RVHKTDLER	0.13	HLA-A*31:01
121	130	FTVRKLGKQW	0.13	HLA-B*58:01
80	87	ISNALKVW	0.13	HLA-B*58:01
78	87	QVISNALKVW	0.13	HLA-B*57:01
103	112	RIDPINERSF	0.14	HLA-A*32:01

285	295	REAYFEKQQQK	0.14	HLA-A*30:01
108	116	NERSFICNY	0.14	HLA-B*44:03
279	288	EELRKRREAY	0.15	HLA-B*44:02
340	348	LQAAVTMSL	0.15	HLA-A*02:06
92	101	ILFNSPEYQR	0.15	HLA-A*31:01
135	143	SLLTGPELI	0.15	HLA-A*02:01
25	33	GEYFSPVEL	0.15	HLA-B*44:03
286	295	EAYFEKQQQK	0.15	HLA-A*68:01
80	87	ISNALKVW	0.15	HLA-B*57:01
115	123	NYKEHWFTV	0.16	HLA-A*23:01
158	165	EGYSIFVV	0.16	HLA-B*51:01
173	181	EADQLLQMI	0.16	HLA-B*51:01
143	151	ISDTYLALF	0.16	HLA-A*01:01
340	348	LQAAVTMSL	0.16	HLA-B*15:01
53	61	VTSEDYRTF	0.17	HLA-A*32:01
180	188	MIRVQQMHR	0.17	HLA-A*33:01
198	206	QLKEQRVHK	0.17	HLA-A*03:01
90	99	ELILFNSPEY	0.18	HLA-A*26:01
105	113	DPINERSFI	0.18	HLA-B*51:01
245	255	EEADLRRAIQL	0.18	HLA-B*44:02
25	33	GEYFSPVEL	0.18	HLA-B*44:02
48	58	MAEGGVTSEDY	0.18	HLA-A*01:01
138	147	TGPELISDTY	0.18	HLA-B*35:01
344	352	VTMSLETVR	0.18	HLA-A*31:01
78	87	QVISNALKVW	0.18	HLA-B*58:01
79	87	VISNALKVW	0.19	HLA-A*32:01
71	79	DSGFFSIQV	0.19	HLA-A*68:02
108	116	NERSFICNY	0.19	HLA-B*44:02
91	99	LILFNSPEY	0.19	HLA-B*35:01
191	199	LIGEELAQL	0.20	HLA-A*02:06
245	255	EEADLRRAIQL	0.20	HLA-B*44:03
190	200	KLIGEELAQLK	0.20	HLA-A*03:01
85	93	KVWGLELIL	0.21	HLA-A*02:06
287	295	AYFEKQQQK	0.21	HLA-A*31:01
101	110	RLRIDPINER	0.21	HLA-A*03:01
179	186	QMIRVQQM	0.22	HLA-B*08:01
75	83	FSIQVISNA	0.22	HLA-A*68:02
279	288	EELRKRREAY	0.22	HLA-B*44:03
182	190	RVQQMHRPK	0.22	HLA-A*11:01
151	160	FLAQLQQEGY	0.22	HLA-B*15:01
344	352	VTMSLETVR	0.22	HLA-A*68:01
147	155	YLALFLAQL	0.23	HLA-A*02:06
172	180	CEADQLLQM	0.23	HLA-B*44:03
92	102	ILFNSPEYQRL	0.23	HLA-A*02:01
227	235	EDLQRALAL	0.24	HLA-B*08:01
145	153	DTYLALFLA	0.24	HLA-A*68:02
48	58	MAEGGVTSEDY	0.24	HLA-B*35:01
153	163	AQLQQEGYSIF	0.24	HLA-B*15:01
154	163	QLQQEGYSIF	0.24	HLA-B*15:01
180	188	MIRVQQMHR	0.24	HLA-A*31:01
122	130	TVRKLGKQW	0.25	HLA-A*32:01
228	237	DLQRALALSR	0.25	HLA-A*33:01
78	86	QVISNALKV	0.25	HLA-A*02:06
172	180	CEADQLLQM	0.25	HLA-B*40:01
125	133	KLGKQWFNL	0.26	HLA-A*32:01
139	147	GPELISDTY	0.26	HLA-B*53:01
283	291	KRREAYFEK	0.26	HLA-A*30:01
197	206	AQLKEQRVHK	0.26	HLA-A*30:01
225	233	DEEDLQRAL	0.26	HLA-B*40:01
122	130	TVRKLGKQW	0.26	HLA-B*58:01
191	199	LIGEELAQL	0.27	HLA-A*02:01
197	206	AQLKEQRVHK	0.27	HLA-A*03:01
143	151	ISDTYLALF	0.27	HLA-B*58:01
112	120	FICNYKEHW	0.27	HLA-B*58:01
31	40	VELSSIAHQL	0.28	HLA-B*44:02
225	233	DEEDLQRAL	0.28	HLA-B*44:03

343	351	AVTMSLETV	0.28	HLA-A*02:06
245	253	EEADLRRAI	0.28	HLA-B*40:01
213	222	LEANDGSGML	0.28	HLA-B*40:01
92	99	ILFNSPEY	0.28	HLA-B*15:01
278	288	SEELRKRREAY	0.29	HLA-B*44:02
317	326	ERPATSSGAL	0.29	HLA-B*07:02
23	33	LQGEYFSPVEL	0.29	HLA-B*40:01
112	120	FICNYKEHW	0.29	HLA-B*57:01
146	155	TYLALFLAQL	0.3	HLA-A*23:01
224	233	EDEEDLQRAL	0.3	HLA-B*44:02
172	180	CEADQLLQM	0.3	HLA-B*44:02
225	233	DEEDLQRAL	0.3	HLA-B*44:02
72	80	SGFFSIQVI	0.3	HLA-B*51:01
152	160	LAQLQQEGY	0.3	HLA-B*35:01
224	233	EDEEDLQRAL	0.3	HLA-B*44:03
31	40	VELSSIAHQL	0.3	HLA-B*44:03
182	190	RVQQMHRPK	0.3	HLA-A*31:01
280	288	ELRKRREAY	0.31	HLA-B*08:01
151	160	FLAQLQQEGY	0.31	HLA-A*01:01
92	101	ILFNSPEYQR	0.31	HLA-A*33:01
245	255	EEADLRRAIQL	0.31	HLA-B*40:01
273	283	GTNLTSEELRK	0.31	HLA-A*11:01
184	191	QQMHRPKL	0.32	HLA-B*08:01
91	99	LILFNSPEY	0.32	HLA-A*30:02
318	327	RPATSSGALG	0.32	HLA-B*07:02
32	40	ELSSIAHQL	0.32	HLA-A*02:03
135	143	SLLTGPELI	0.32	HLA-A*02:03
188	197	RPKLIGEELA	0.32	HLA-B*07:02
117	126	KEHWFTVRKL	0.32	HLA-B*40:01
78	87	QVISNALKVW	0.33	HLA-A*32:01
99	107	YQRLRIDPI	0.33	HLA-B*08:01
141	149	ELISDTYLA	0.33	HLA-A*68:02
85	93	KVWGLELIL	0.33	HLA-A*02:01
214	222	EANDGSGML	0.34	HLA-A*68:02
344	352	VTMSLETVR	0.34	HLA-A*33:01
137	147	LTGPELISDTY	0.34	HLA-B*35:01
280	288	ELRKRREAY	0.35	HLA-A*26:01
32	40	ELSSIAHQL	0.35	HLA-A*26:01
129	137	QWFNLNSLL	0.35	HLA-A*23:01
183	191	VQQMHRPKL	0.35	HLA-B*08:01
128	136	KQWFNLNSL	0.35	HLA-A*02:01
135	143	SLLTGPELI	0.35	HLA-A*02:06
276	284	LTSEELRKR	0.35	HLA-A*68:01
169	178	LPDCEADQLL	0.36	HLA-B*53:01
53	61	VTSEDYRTF	0.36	HLA-A*23:01
32	40	ELSSIAHQL	0.36	HLA-B*08:01
81	89	SNALKVWGL	0.36	HLA-B*08:01
128	136	KQWFNLNSL	0.36	HLA-B*15:01
85	94	KVWGLELILF	0.36	HLA-B*57:01
158	166	EGYSIFVVK	0.36	HLA-A*68:01
110	120	RSFICNYKEHW	0.37	HLA-A*32:01
152	160	LAQLQQEGY	0.37	HLA-A*01:01
23	31	LQGEYFSPV	0.37	HLA-A*02:06
340	348	LQAAVTMSL	0.38	HLA-A*32:01
285	295	REAYFEKQQQK	0.38	HLA-A*31:01
85	94	KVWGLELILF	0.39	HLA-A*23:01
53	61	VTSEDYRTF	0.39	HLA-A*24:02
32	40	ELSSIAHQL	0.39	HLA-A*02:01
85	94	KVWGLELILF	0.4	HLA-A*24:02
112	120	FICNYKEHW	0.41	HLA-B*53:01
111	120	SFICNYKEHW	0.41	HLA-A*23:01
78	87	QVISNALKVW	0.41	HLA-B*53:01
138	147	TGPELISDTY	0.41	HLA-B*53:01
214	222	EANDGSGML	0.42	HLA-A*26:01
232	240	ALALSRQEI	0.42	HLA-A*02:03
280	289	ELRKRREAYF	0.43	HLA-B*08:01

92	99	ILFNSPEY	0.43	HLA-A*30:02
233	240	LALSRQEI	0.43	HLA-B*51:01
25	34	GEYFSPVELS	0.43	HLA-B*40:01
85	94	KVWGLELILF	0.43	HLA-B*58:01
76	85	SIQVISNALK	0.43	HLA-A*11:01
229	237	LQRALALSR	0.43	HLA-A*31:01
201	211	EQRVHKTDLER	0.43	HLA-A*68:01
53	61	VTSEDYRTF	0.44	HLA-A*26:01
26	36	EYFSPVELSSI	0.44	HLA-A*23:01
146	155	TYLALFLAQL	0.44	HLA-A*24:02
82	89	NALKVWGL	0.44	HLA-B*08:01
24	33	QGEYFSPVEL	0.44	HLA-B*44:03
93	103	LFNSPEYQRLR	0.44	HLA-A*33:01
95	103	NSPEYQRLR	0.44	HLA-A*33:01
213	221	LEANDGSGM	0.44	HLA-B*40:01
129	137	QWFNLNSLL	0.45	HLA-A*24:02
29	38	SPVELSSIAH	0.45	HLA-B*35:01
71	79	DSGFFSIQV	0.45	HLA-B*51:01
186	196	MHRPKLIGEEL	0.45	HLA-B*07:02
139	148	GPELISDTYL	0.45	HLA-B*40:01
77	87	IQVISNALKVW	0.45	HLA-B*57:01
79	87	VISNALKVW	0.46	HLA-B*53:01
55	62	SEDYRTFL	0.46	HLA-B*40:01
75	83	FSIQVISNA	0.46	HLA-A*02:06
26	33	EYFSPVEL	0.47	HLA-B*40:01
226	235	EEDLQRALAL	0.47	HLA-B*40:01
224	233	EDEEDLQRAL	0.47	HLA-B*40:01
340	348	LQAAVTMSL	0.47	HLA-A*02:03
286	295	EAYFEKQQQK	0.48	HLA-A*33:01
26	36	EYFSPVELSSI	0.49	HLA-A*24:02
157	165	QEGYSIFVV	0.49	HLA-B*40:01
246	253	EADLRRAI	0.49	HLA-B*51:01
19	27	LNNLLQGEY	0.49	HLA-A*30:02
103	112	RIDPINERSF	0.49	HLA-B*58:01
141	151	ELISDTYLALF	0.5	HLA-A*26:01
173	181	EADQLLQMI	0.5	HLA-B*53:01
24	33	QGEYFSPVEL	0.5	HLA-B*44:02
111	120	SFICNYKEHW	0.5	HLA-A*24:02
278	288	SEELRKRREAY	0.5	HLA-B*44:03
145	152	DTYLALFL	0.5	HLA-B*51:01
38	46	HQLDEEERM	0.5	HLA-A*02:06
188	196	RPKLIGEEL	0.51	HLA-B*35:01
83	91	ALKVWGLEL	0.51	HLA-A*02:03
221	229	MLDEDEEDL	0.51	HLA-A*02:01
91	99	LILFNSPEY	0.51	HLA-B*15:01
53	61	VTSEDYRTF	0.52	HLA-B*53:01
83	91	ALKVWGLEL	0.52	HLA-B*08:01
101	110	RLRIDPINER	0.52	HLA-A*30:01
191	199	LIGEELAQL	0.52	HLA-A*02:03
143	151	ISDTYLALF	0.52	HLA-B*57:01
95	103	NSPEYQRLR	0.52	HLA-A*68:01
137	147	LTGPELISDTY	0.53	HLA-B*53:01
203	211	RVHKTDLER	0.53	HLA-A*30:01
203	211	RVHKTDLER	0.53	HLA-A*11:01
78	86	QVISNALKV	0.54	HLA-A*26:01
279	289	EELRKRREAYF	0.54	HLA-B*44:02
197	206	AQLKEQRVHK	0.54	HLA-A*11:01
275	284	NLTSEELRKR	0.54	HLA-A*68:01
226	235	EEDLQRALAL	0.55	HLA-B*44:02
92	102	ILFNSPEYQRL	0.55	HLA-A*02:03
280	288	ELRKRREAY	0.55	HLA-B*15:01
243	253	EDEEADLRRAI	0.56	HLA-B*44:02
29	37	SPVELSSIA	0.56	HLA-B*35:01
200	209	KEQRVHKTDL	0.56	HLA-B*40:01
100	110	QRLRIDPINER	0.56	HLA-A*31:01
198	206	QLKEQRVHK	0.56	HLA-A*31:01

318	326	RPATSSGAL	0.57	HLA-B*35:01
96	104	SPEYQRLRI	0.57	HLA-B*51:01
125	133	KLGKQWFNL	0.57	HLA-A*02:01
142	150	LISDTYLAL	0.57	HLA-A*02:06
93	103	LFNSPEYQRLR	0.57	HLA-A*31:01
188	196	RPKLIGEEL	0.58	HLA-B*53:01
26	33	EYFSPVEL	0.58	HLA-A*23:01
6	13	HEKQEGSL	0.58	HLA-B*40:01
197	205	AQLKEQRVH	0.58	HLA-B*15:01
203	211	RVHKTDLER	0.58	HLA-A*03:01
138	147	TGPELISDTY	0.59	HLA-A*01:01
226	233	EEDLQRAL	0.59	HLA-B*40:01
336	346	EEDMLQAAVTM	0.59	HLA-B*40:01
103	113	RIDPINERSFI	0.59	HLA-B*07:02
344	352	VTMSLETVR	0.59	HLA-A*11:01
101	110	RLRIDPINER	0.59	HLA-A*11:01
343	352	AVTMSLETVR	0.59	HLA-A*31:01
26	33	EYFSPVEL	0.6	HLA-A*24:02
336	346	EEDMLQAAVTM	0.6	HLA-B*44:03
338	346	DMLQAAVTM	0.6	HLA-B*35:01
151	160	FLAQLQQEGY	0.61	HLA-A*26:01
155	163	LQQEGYSIF	0.61	HLA-A*23:01
76	84	SIQVISNAL	0.61	HLA-B*08:01
141	150	ELISDTYLAL	0.61	HLA-A*68:02
29	36	SPVELSSI	0.61	HLA-B*07:02
29	37	SPVELSSIA	0.61	HLA-B*07:02
340	348	LQAAVTMSL	0.61	HLA-A*02:01
190	200	KLIGEELAQLK	0.61	HLA-A*02:01
139	147	GPELISDTY	0.62	HLA-A*01:01
171	180	DCEADQLLQM	0.62	HLA-B*44:03
226	235	EEDLQRALAL	0.62	HLA-B*44:03
4	13	IFHEKQEGSL	0.62	HLA-B*08:01
156	164	QQEGYSIFV	0.62	HLA-A*02:06
336	344	EEDMLQAAV	0.63	HLA-B*40:01
273	283	GTNLTSEELRK	0.63	HLA-A*03:01
226	233	EEDLQRAL	0.64	HLA-B*44:02
155	163	LQQEGYSIF	0.64	HLA-A*24:02
91	101	LILFNSPEYQR	0.64	HLA-A*33:01
115	124	NYKEHWFTVR	0.64	HLA-A*31:01
112	120	FICNYKEHW	0.65	HLA-A*32:01
226	233	EEDLQRAL	0.65	HLA-B*44:03
246	253	EADLRRAI	0.65	HLA-B*08:01
128	136	KQWFNLNSL	0.65	HLA-B*40:01
287	295	AYFEKQQQK	0.65	HLA-A*33:01
137	147	LTGPELISDTY	0.65	HLA-B*58:01
137	147	LTGPELISDTY	0.65	HLA-B*57:01
91	101	LILFNSPEYQR	0.66	HLA-A*31:01
342	352	AAVTMSLETVR	0.66	HLA-A*31:01
83	91	ALKVWGLEL	0.67	HLA-A*32:01
122	130	TVRKLGKQW	0.67	HLA-B*53:01
279	289	EELRKRREAYF	0.67	HLA-B*44:03
243	253	EDEEADLRRAI	0.67	HLA-B*44:03
187	196	HRPKLIGEEL	0.67	HLA-B*08:01
105	112	DPINERSF	0.67	HLA-B*35:01
335	344	SEEDMLQAAV	0.67	HLA-B*40:01
53	61	VTSEDYRTF	0.67	HLA-B*15:01
153	160	AQLQQEGY	0.67	HLA-B*15:01
92	101	ILFNSPEYQR	0.67	HLA-A*03:01
333	342	AMSEEDMLQA	0.68	HLA-A*02:03
61	69	FLQQPSGNM	0.68	HLA-A*02:03
32	40	ELSSIAHQL	0.68	HLA-A*02:06
120	130	WFTVRKLGKQW	0.68	HLA-B*57:01
194	203	EELAQLKEQR	0.68	HLA-A*68:01
117	126	KEHWFTVRKL	0.69	HLA-B*44:02
117	126	KEHWFTVRKL	0.69	HLA-B*44:03
211	221	RVLEANDGSGM	0.69	HLA-B*40:01

155	164	LQQEGYSIFV	0.69	HLA-A*02:06
52	61	GVTSEDYRTF	0.69	HLA-B*57:01
246	255	EADLRRAIQL	0.7	HLA-B*08:01
77	87	IQVISNALKVW	0.7	HLA-B*58:01
178	186	LQMIRVQQM	0.7	HLA-A*02:06
76	85	SIQVISNALK	0.7	HLA-A*03:01
103	112	RIDPINERSF	0.7	HLA-B*57:01
336	346	EEDMLQAAVTM	0.71	HLA-B*44:02
169	177	LPDCEADQL	0.71	HLA-B*53:01
151	160	FLAQLQQEGY	0.71	HLA-A*30:02
220	229	GMLDEDEEDL	0.71	HLA-A*02:01
316	326	CERPATSSGAL	0.72	HLA-B*07:02
332	340	DAMSEEDML	0.72	HLA-B*51:01
115	123	NYKEHWFTV	0.73	HLA-B*08:01
196	204	LAQLKEQRV	0.73	HLA-B*51:01
78	86	QVISNALKV	0.73	HLA-A*02:03
147	155	YLALFLAQL	0.74	HLA-B*08:01
268	276	MTQTSGTNL	0.74	HLA-A*68:02
71	80	DSGFFSIQVI	0.74	HLA-B*51:01
343	351	AVTMSLETV	0.74	HLA-A*02:03
336	344	EEDMLQAAV	0.75	HLA-B*44:02
338	346	DMLQAAVTM	0.75	HLA-B*08:01
194	203	EELAQLKEQR	0.75	HLA-A*33:01
179	188	QMIRVQQMHR	0.75	HLA-A*31:01
51	61	GGVTSEDYRTF	0.75	HLA-B*57:01
105	112	DPINERSF	0.76	HLA-B*53:01
314	324	HPCERPATSSG	0.76	HLA-B*07:02
31	40	VELSSIAHQL	0.76	HLA-A*02:01
31	40	VELSSIAHQL	0.76	HLA-A*02:03
92	102	ILFNSPEYQRL	0.76	HLA-A*02:06
244	253	DEEADLRRAI	0.77	HLA-B*44:02
151	160	FLAQLQQEGY	0.77	HLA-B*35:01
171	180	DCEADQLLQM	0.78	HLA-B*44:02
128	137	KQWFNLNSLL	0.78	HLA-A*32:01
223	233	DEDEEDLQRAL	0.78	HLA-B*40:01
184	192	QQMHRPKLI	0.78	HLA-B*08:01
200	209	KEQRVHKTDL	0.79	HLA-B*44:02
341	348	QAAVTMSL	0.79	HLA-B*51:01
23	31	LQGEYFSPV	0.79	HLA-A*02:03
190	200	KLIGEELAQLK	0.79	HLA-A*11:01
343	352	AVTMSLETVR	0.79	HLA-A*68:01
281	289	LRKRREAYF	0.8	HLA-B*08:01
336	344	EEDMLQAAV	0.81	HLA-B*44:03
148	155	LALFLAQL	0.81	HLA-B*51:01
152	160	LAQLQQEGY	0.81	HLA-A*30:02
52	61	GVTSEDYRTF	0.81	HLA-B*58:01
142	150	LISDTYLAL	0.81	HLA-A*02:01
142	150	LISDTYLAL	0.81	HLA-A*02:03
173	182	EADQLLQMIR	0.81	HLA-A*68:01
169	177	LPDCEADQL	0.82	HLA-B*35:01
173	180	EADQLLQM	0.82	HLA-B*51:01
72	79	SGFFSIQV	0.82	HLA-B*51:01
287	295	AYFEKQQQK	0.82	HLA-A*11:01
310	318	GQSSHPCER	0.82	HLA-A*31:01
141	150	ELISDTYLAL	0.83	HLA-A*26:01
53	61	VTSEDYRTF	0.83	HLA-B*35:01
158	166	EGYSIFVVK	0.83	HLA-A*33:01
256	264	SMQGSSRNI	0.84	HLA-A*32:01
64	74	QPSGNMDDSGF	0.84	HLA-B*53:01
91	99	LILFNSPEY	0.84	HLA-A*01:01
247	255	ADLRRAIQL	0.84	HLA-B*08:01
76	84	SIQVISNAL	0.84	HLA-B*07:02
287	295	AYFEKQQQK	0.84	HLA-A*30:02
339	348	MLQAAVTMSL	0.84	HLA-A*02:03
115	125	NYKEHWFTVRK	0.85	HLA-A*33:01
59	67	RTFLQQPSG	0.85	HLA-A*30:01

187	196	HRPKLIGEEL	0.86	HLA-B*53:01
213	221	LEANDGSGM	0.86	HLA-B*44:03
177	186	LLQMIRVQQM	0.86	HLA-B*08:01
173	181	EADQLLQMI	0.86	HLA-A*68:02
189	199	PKLIGEELAQL	0.86	HLA-A*02:01
276	284	LTSEELRKR	0.87	HLA-A*33:01
276	284	LTSEELRKR	0.87	HLA-A*31:01
111	120	SFICNYKEHW	0.87	HLA-B*57:01
335	343	SEEDMLQAA	0.88	HLA-B*44:02
91	99	LILFNSPEY	0.88	HLA-A*26:01
19	27	LNNLLQGEY	0.88	HLA-A*01:01
117	125	KEHWFTVRK	0.88	HLA-A*30:01
287	295	AYFEKQQQK	0.88	HLA-A*03:01
122	130	TVRKLGKQW	0.89	HLA-A*26:01
332	340	DAMSEEDML	0.89	HLA-B*53:01
340	348	LQAAVTMSL	0.89	HLA-B*40:01
104	113	IDPINERSFI	0.89	HLA-B*51:01
333	342	AMSEEDMLQA	0.89	HLA-A*02:01
343	352	AVTMSLETVR	0.89	HLA-A*11:01
92	101	ILFNSPEYQR	0.89	HLA-A*68:01
27	36	YFSPVELSSI	0.9	HLA-A*24:02
256	264	SMQGSSRNI	0.9	HLA-A*02:03
198	206	QLKEQRVHK	0.9	HLA-A*11:01
180	188	MIRVQQMHR	0.9	HLA-A*68:01
178	186	LQMIRVQQM	0.91	HLA-A*32:01
138	148	TGPELISDTYL	0.91	HLA-B*40:01
280	287	ELRKRREA	0.91	HLA-B*08:01
111	120	SFICNYKEHW	0.91	HLA-B*58:01
59	69	RTFLQQPSGNM	0.92	HLA-A*32:01
335	343	SEEDMLQAA	0.92	HLA-B*44:03
137	147	LTGPELISDTY	0.93	HLA-A*30:02
78	86	QVISNALKV	0.93	HLA-A*02:01
103	112	RIDPINERSF	0.93	HLA-B*15:01
61	69	FLQQPSGNM	0.93	HLA-B*15:01
244	253	DEEADLRRAI	0.94	HLA-B*44:03
277	285	TSEELRKRR	0.94	HLA-A*33:01
102	110	LRIDPINER	0.94	HLA-A*33:01
101	110	RLRIDPINER	0.94	HLA-A*33:01
128	137	KQWFNLNSLL	0.94	HLA-A*02:06
274	284	TNLTSEELRKR	0.94	HLA-A*68:01
223	233	DEDEEDLQRAL	0.95	HLA-B*44:02
27	36	YFSPVELSSI	0.95	HLA-A*23:01
334	342	MSEEDMLQA	0.95	HLA-A*01:01
38	46	HQLDEEERM	0.95	HLA-B*40:01
335	343	SEEDMLQAA	0.95	HLA-B*40:01
196	204	LAQLKEQRV	0.95	HLA-B*08:01
82	89	NALKVWGL	0.95	HLA-B*51:01
28	36	FSPVELSSI	0.95	HLA-B*51:01
173	180	EADQLLQM	0.96	HLA-B*35:01
85	93	KVWGLELIL	0.96	HLA-B*58:01
347	356	SLETVRNDLK	0.96	HLA-A*03:01
101	111	RLRIDPINERS	0.96	HLA-A*31:01
121	131	FTVRKLGKQWF	0.96	HLA-B*57:01
92	99	ILFNSPEY	0.97	HLA-B*35:01
169	178	LPDCEADQLL	0.97	HLA-B*35:01
214	222	EANDGSGML	0.97	HLA-B*35:01
103	112	RIDPINERSF	0.97	HLA-A*30:02
179	188	QMIRVQQMHR	0.97	HLA-A*33:01
343	351	AVTMSLETV	0.97	HLA-A*02:01
200	209	KEQRVHKTDL	0.98	HLA-B*44:03
223	233	DEDEEDLQRAL	0.98	HLA-B*44:03
93	102	LFNSPEYQRL	0.98	HLA-A*24:02
343	351	AVTMSLETV	0.98	HLA-A*68:02
154	162	QLQQEGYSI	0.98	HLA-A*02:01
213	221	LEANDGSGM	0.99	HLA-B*44:02
182	191	RVQQMHRPKL	0.99	HLA-A*30:01

Table II – Results of the full-length and mutant ATXN3-MHC-II binding prediction. Acquired with TepiTool (http://tools.iedb.org/tepitool/).

ATXN-3 MHC-II				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
175	189	DQLLQMIRVQQMHRP	0.01	HLA-DRB4*01:01
141	155	ELISDTYLALFLAQL	0.11	HLA-DPA1*02:01/DPB1*01:01
99	113	YQRLRIDPINERSFI	0.27	HLA-DRB1*03:01
325	339	ALGSDLGDAMSEEDM	0.61	HLA-DQA1*04:01/DQB1*04:02
71	85	DSGFFSIQVISNALK	0.78	HLA-DRB1*04:01
233	247	LALSRQEIDMEDEEA	0.89	HLA-DQA1*05:01/DQB1*02:01
141	155	ELISDTYLALFLAQL	0.90	HLA-DPA1*03:01/DPB1*04:02
78	92	QVISNALKVWGLELI	0.90	HLA-DQA1*01:02/DQB1*06:02
146	160	TYLALFLAQLQQEGY	0.94	HLA-DQA1*04:01/DQB1*04:02
146	160	TYLALFLAQLQQEGY	1.20	HLA-DRB4*01:01
136	150	LLTGPELISDTYLAL	1.30	HLA-DPA1*02:01/DPB1*01:01
146	160	TYLALFLAQLQQEGY	1.50	HLA-DQA1*03:01/DQB1*03:02
83	97	ALKVWGLELILFNSP	1.80	HLA-DQA1*01:01/DQB1*05:01
115	129	NYKEHWFTVRKLGKQ	1.80	HLA-DRB1*11:01
187	201	HRPKLIGEELAQLKE	1.90	HLA-DPA1*03:01/DPB1*04:02
141	155	ELISDTYLALFLAQL	2.00	HLA-DPA1*01:03/DPB1*02:01
83	97	ALKVWGLELILFNSP	2.00	HLA-DPA1*02:01/DPB1*01:01
175	189	DQLLQMIRVQQMHRP	2.00	HLA-DQA1*01:02/DQB1*06:02
30	44	PVELSSIAHQLDEEE	2.00	HLA-DQA1*05:01/DQB1*02:01
17	31	HCLNNLLQGEYFSPV	2.10	HLA-DPA1*01:03/DPB1*02:01
71	85	DSGFFSIQVISNALK	2.10	HLA-DRB1*04:05
136	150	LLTGPELISDTYLAL	2.20	HLA-DPA1*01:03/DPB1*02:01
89	103	LELILFNSPEYQRLR	2.30	HLA-DRB1*13:02
146	160	TYLALFLAQLQQEGY	2.40	HLA-DPA1*02:01/DPB1*01:01
30	44	PVELSSIAHQLDEEE	2.50	HLA-DQA1*04:01/DQB1*04:02
136	150	LLTGPELISDTYLAL	2.60	HLA-DPA1*03:01/DPB1*04:02
78	92	QVISNALKVWGLELI	2.60	HLA-DPA1*03:01/DPB1*04:02
71	85	DSGFFSIQVISNALK	2.60	HLA-DRB5*01:01
141	155	ELISDTYLALFLAQL	2.70	HLA-DPA1*01/DPB1*04:01
136	150	LLTGPELISDTYLAL	2.80	HLA-DPA1*01/DPB1*04:01
66	80	SGNMDDSGFFSIQVI	2.90	HLA-DQA1*01:01/DQB1*05:01
226	240	EEDLQRALALSRQEI	2.90	HLA-DRB5*01:01
341	355	QAAVTMSLETVRNDL	3.00	HLA-DRB1*03:01
129	143	QWFNLNSLLTGPELI	3.00	HLA-DRB1*04:05
89	103	LELILFNSPEYQRLR	3.10	HLA-DRB3*02:02
187	201	HRPKLIGEELAQLKE	3.20	HLA-DPA1*02:01/DPB1*01:01
83	97	ALKVWGLELILFNSP	3.20	HLA-DPA1*03:01/DPB1*04:02
89	103	LELILFNSPEYQRLR	3.20	HLA-DRB1*12:01
246	260	EADLRRAIQLSMQGS	3.30	HLA-DRB4*01:01
136	150	LLTGPELISDTYLAL	3.40	HLA-DRB3*01:01
83	97	ALKVWGLELILFNSP	3.50	HLA-DPA1*01/DPB1*04:01
89	103	LELILFNSPEYQRLR	3.50	HLA-DPA1*02:01/DPB1*01:01
17	31	HCLNNLLQGEYFSPV	3.50	HLA-DPA1*03:01/DPB1*04:02
146	160	TYLALFLAQLQQEGY	3.50	HLA-DRB1*04:05
141	155	ELISDTYLALFLAQL	3.60	HLA-DRB1*01:01
336	350	EEDMLQAAVTMSLET	3.70	HLA-DQA1*01:02/DQB1*06:02
83	97	ALKVWGLELILFNSP	3.80	HLA-DPA1*02:01/DPB1*05:01
146	160	TYLALFLAQLQQEGY	3.80	HLA-DPA1*03:01/DPB1*04:02
78	92	QVISNALKVWGLELI	3.80	HLA-DQA1*01:01/DQB1*05:01
175	189	DQLLQMIRVQQMHRP	3.80	HLA-DRB1*08:02
325	339	ALGSDLGDAMSEEDM	4.10	HLA-DQA1*03:01/DQB1*03:02
99	113	YQRLRIDPINERSFI	4.10	HLA-DRB3*01:01
336	350	EEDMLQAAVTMSLET	4.20	HLA-DQA1*04:01/DQB1*04:02
336	350	EEDMLQAAVTMSLET	4.40	HLA-DRB1*07:01
129	143	QWFNLNSLLTGPELI	4.50	HLA-DRB3*02:02
146	160	TYLALFLAQLQQEGY	4.60	HLA-DRB1*12:01
89	103	LELILFNSPEYQRLR	4.60	HLA-DRB1*15:01
22	36	LLQGEYFSPVELSSI	4.80	HLA-DPA1*02:01/DPB1*01:01

180	194	MIRVQQMHRPKLIGE	4.90	HLA-DRB4*01:01
124	138	RKLGKQWFNLNSLLT	5.20	HLA-DQA1*01:01/DQB1*05:01
22	36	LLQGEYFSPVELSSI	5.30	HLA-DPA1*01:03/DPB1*02:01
44	58	ERMRMAEGGVTSEDY	5.30	HLA-DQA1*05:01/DQB1*03:01
89	103	LELILFNSPEYQRLR	5.40	HLA-DPA1*01:03/DPB1*02:01
141	155	ELISDTYLALFLAQL	5.40	HLA-DPA1*02:01/DPB1*05:01
71	85	DSGFFSIQVISNALK	5.40	HLA-DRB3*02:02
22	36	LLQGEYFSPVELSSI	5.50	HLA-DPA1*03:01/DPB1*04:02
115	129	NYKEHWFTVRKLGKQ	5.50	HLA-DRB5*01:01
71	85	DSGFFSIQVISNALK	5.60	HLA-DRB4*01:01
124	138	RKLGKQWFNLNSLLT	5.80	HLA-DPA1*02:01/DPB1*01:01
141	155	ELISDTYLALFLAQL	5.90	HLA-DQA1*01:02/DQB1*06:02
214	228	EANDGSGMLDEDEED	5.90	HLA-DQA1*04:01/DQB1*04:02
226	240	EEDLQRALALSRQEI	5.90	HLA-DRB1*03:01
89	103	LELILFNSPEYQRLR	6.00	HLA-DPA1*01/DPB1*04:01
124	138	RKLGKQWFNLNSLLT	6.10	HLA-DRB1*04:05
146	160	TYLALFLAQLQQEGY	6.20	HLA-DQA1*01:01/DQB1*05:01
214	228	EANDGSGMLDEDEED	6.20	HLA-DQA1*05:01/DQB1*02:01
71	85	DSGFFSIQVISNALK	6.20	HLA-DRB1*08:02
56	70	EDYRTFLQQPSGNMD	6.30	HLA-DRB1*04:05
50	64	EGGVTSEDYRTFLQQ	6.40	HLA-DPA1*01:03/DPB1*02:01
44	58	ERMRMAEGGVTSEDY	6.40	HLA-DQA1*04:01/DQB1*04:02
238	252	QEIDMEDEEADLRRA	6.60	HLA-DQA1*05:01/DQB1*02:01
56	70	EDYRTFLQQPSGNMD	6.70	HLA-DRB1*04:01
83	97	ALKVWGLELILFNSP	6.70	HLA-DRB4*01:01
146	160	TYLALFLAQLQQEGY	6.80	HLA-DRB1*01:01
124	138	RKLGKQWFNLNSLLT	6.80	HLA-DRB3*02:02
292	306	QQQKQQQQQQQQQQG	6.80	HLA-DRB4*01:01
193	207	GEELAQLKEQRVHKT	6.90	HLA-DRB4*01:01
71	85	DSGFFSIQVISNALK	7.00	HLA-DPA1*03:01/DPB1*04:02
214	228	EANDGSGMLDEDEED	7.00	HLA-DQA1*03:01/DQB1*03:02
141	155	ELISDTYLALFLAQL	7.00	HLA-DQA1*04:01/DQB1*04:02
175	189	DQLLQMIRVQQMHRP	7.00	HLA-DRB1*04:05
146	160	TYLALFLAQLQQEGY	7.10	HLA-DPA1*01:03/DPB1*02:01
71	85	DSGFFSIQVISNALK	7.20	HLA-DRB1*07:01
169	183	LPDCEADQLLQMIRV	7.30	HLA-DPA1*03:01/DPB1*04:02
251	265	RAIQLSMQGSSRNIS	7.30	HLA-DRB1*03:01
35	49	SIAHQLDEEERMRMA	7.30	HLA-DRB1*03:01
71	85	DSGFFSIQVISNALK	7.30	HLA-DRB1*15:01
17	31	HCLNNLLQGEYFSPV	7.50	HLA-DPA1*02:01/DPB1*01:01
44	58	ERMRMAEGGVTSEDY	7.50	HLA-DQA1*03:01/DQB1*03:02
30	44	PVELSSIAHQLDEEE	7.60	HLA-DQA1*03:01/DQB1*03:02
259	273	GSSRNISQDMTQTSG	7.60	HLA-DRB1*04:01
175	189	DQLLQMIRVQQMHRP	7.60	HLA-DRB1*12:01
71	85	DSGFFSIQVISNALK	7.70	HLA-DPA1*02:01/DPB1*01:01
71	85	DSGFFSIQVISNALK	7.95	HLA-DRB1*12:01
22	36	LLQGEYFSPVELSSI	8.00	HLA-DQA1*01:01/DQB1*05:01
124	138	RKLGKQWFNLNSLLT	8.00	HLA-DRB1*04:01
282	296	RKRREAYFEKQQQKQ	8.01	HLA-DPA1*01/DPB1*04:01
233	247	LALSRQEIDMEDEEA	8.10	HLA-DQA1*01:01/DQB1*05:01
267	281	DMTQTSGTNLTSEEL	8.10	HLA-DQA1*03:01/DQB1*03:02
129	143	QWFNLNSLLTGPELI	8.10	HLA-DRB1*04:01
83	97	ALKVWGLELILFNSP	8.20	HLA-DPA1*01:03/DPB1*02:01
141	155	ELISDTYLALFLAQL	8.20	HLA-DQA1*03:01/DQB1*03:02
233	247	LALSRQEIDMEDEEA	8.20	HLA-DQA1*03:01/DQB1*03:02
141	155	ELISDTYLALFLAQL	8.40	HLA-DQA1*01:01/DQB1*05:01
56	70	EDYRTFLQQPSGNMD	8.40	HLA-DRB1*01:01
259	273	GSSRNISQDMTQTSG	8.60	HLA-DRB1*03:01
89	103	LELILFNSPEYQRLR	8.70	HLA-DPA1*02:01/DPB1*05:01
141	155	ELISDTYLALFLAQL	8.70	HLA-DQA1*05:01/DQB1*02:01
35	49	SIAHQLDEEERMRMA	8.70	HLA-DQA1*05:01/DQB1*02:01
146	160	TYLALFLAQLQQEGY	8.70	HLA-DQA1*05:01/DQB1*02:01
71	85	DSGFFSIQVISNALK	8.70	HLA-DRB1*01:01
175	189	DQLLQMIRVQQMHRP	8.70	HLA-DRB1*11:01
17	31	HCLNNLLQGEYFSPV	8.70	HLA-DRB1*12:01
115	129	NYKEHWFTVRKLGKQ	8.80	HLA-DPA1*02:01/DPB1*05:01

146	160	TYLALFLAQLQQEGY	8.80	HLA-DQA1*01:02/DQB1*06:02
78	92	QVISNALKVWGLELI	8.90	HLA-DPA1*01:03/DPB1*02:01
110	124	RSFICNYKEHWFTVR	8.90	HLA-DPA1*01:03/DPB1*02:01
346	360	MSLETVRNDLKTEGK	8.90	HLA-DRB1*03:01
336	350	EEDMLQAAVTMSLET	9.00	HLA-DRB1*04:05
78	92	QVISNALKVWGLELI	9.01	HLA-DPA1*01/DPB1*04:01
124	138	RKLGKQWFNLNSLLT	9.20	HLA-DRB1*11:01
129	143	QWFNLNSLLTGPELI	9.40	HLA-DPA1*01:03/DPB1*02:01
330	344	LGDAMSEEDMLQAAV	9.40	HLA-DQA1*04:01/DQB1*04:02
83	97	ALKVWGLELILFNSP	9.40	HLA-DRB1*12:01
89	103	LELILFNSPEYQRLR	9.50	HLA-DQA1*01:01/DQB1*05:01
175	189	DQLLQMIRVQQMHRP	9.50	HLA-DRB1*04:01
30	44	PVELSSIAHQLDEEE	9.70	HLA-DRB1*04:05
251	265	RAIQLSMQGSSRNIS	9.70	HLA-DRB3*02:02
110	124	RSFICNYKEHWFTVR	9.80	HLA-DPA1*02:01/DPB1*05:01
94	108	FNSPEYQRLRIDPIN	9.90	HLA-DRB3*01:01

Table III – Results of the IFN-β-MHC-I binding prediction.

Acquired with TepiTool (http://tools.iedb.org/tepitool/).

IFN-β MHC-I				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
71	81	FQKEDAAVTIY	0.01	HLA-B*15:01
137	145	RMSSLHLKR	0.01	HLA-A*31:01
146	154	YYGRILHYL	0.01	HLA-A*23:01
145	153	RYYGRILHY	0.01	HLA-A*30:02
146	154	YYGRILHYL	0.01	HLA-A*24:02
176	184	YVINRLTGY	0.01	HLA-A*26:01
63	71	EEIKQLQQF	0.01	HLA-B*44:02
63	71	EEIKQLQQF	0.01	HLA-B*44:03
144	153	KRYYGRILHY	0.03	HLA-A*30:02
43	51	WQLNGRLEY	0.03	HLA-B*15:01
121	129	TVLEEKLEK	0.03	HLA-A*11:01
16	24	STTALSMSY	0.04	HLA-A*30:02
105	113	VENLLANVY	0.04	HLA-B*44:02
149	157	RILHYLKAK	0.04	HLA-A*03:01
105	113	VENLLANVY	0.04	HLA-B*44:03
165	173	TIVRVEILR	0.04	HLA-A*68:01
145	154	RYYGRILHYL	0.05	HLA-A*23:01
145	153	RYYGRILHY	0.05	HLA-A*23:01
80	88	IYEMLQNIF	0.05	HLA-A*24:02
155	164	KAKEDSHCAW	0.05	HLA-B*57:01
145	155	RYYGRILHYLK	0.06	HLA-A*30:01
145	153	RYYGRILHY	0.06	HLA-A*30:01
137	146	RMSSLHLKRY	0.06	HLA-A*30:02
16	24	STTALSMSY	0.06	HLA-A*26:01
176	184	YVINRLTGY	0.06	HLA-B*15:01
73	81	KEDAAVTIY	0.06	HLA-B*44:03
16	24	STTALSMSY	0.06	HLA-A*01:01
155	164	KAKEDSHCAW	0.06	HLA-B*58:01
61	71	IPEEIKQLQQF	0.07	HLA-B*53:01
175	184	FYVINRLTGY	0.07	HLA-A*26:01
145	153	RYYGRILHY	0.07	HLA-A*24:02
145	154	RYYGRILHYL	0.07	HLA-A*24:02
75	83	DAAVTIYEM	0.07	HLA-B*35:01
168	176	RVEILRNFY	0.08	HLA-A*30:02
80	88	IYEMLQNIF	0.08	HLA-A*23:01
73	81	KEDAAVTIY	0.08	HLA-B*44:02
176	184	YVINRLTGY	0.09	HLA-A*30:02
139	147	SSLHLKRYY	0.09	HLA-A*30:02
79	87	TIYEMLQNI	0.09	HLA-A*02:06

120	129	KTVLEEKLEK	0.09	HLA-A*11:01
145	153	RYYGRILHY	0.1	HLA-A*32:01
149	157	RILHYLKAK	0.1	HLA-A*30:01
112	120	VYHQRNHLK	0.1	HLA-A*30:01
75	83	DAAVTIYEM	0.1	HLA-B*51:01
79	87	TIYEMLQNI	0.1	HLA-A*02:03
137	147	RMSSLHLKRYY	0.11	HLA-A*30:02
108	116	LLANVYHQR	0.11	HLA-A*31:01
108	116	LLANVYHQR	0.12	HLA-A*33:01
101	109	NETIVENLL	0.12	HLA-B*40:01
42	51	LWQLNGRLEY	0.13	HLA-B*15:01
79	87	TIYEMLQNI	0.14	HLA-A*68:02
124	132	EEKLEKEDF	0.14	HLA-B*44:02
40	48	KLLWQLNGR	0.14	HLA-A*31:01
145	155	RYYGRILHYLK	0.14	HLA-A*31:01
120	129	KTVLEEKLEK	0.14	HLA-A*03:01
137	145	RMSSLHLKR	0.14	HLA-A*03:01
43	51	WQLNGRLEY	0.15	HLA-A*30:02
22	32	MSYNLLGFLQR	0.15	HLA-A*31:01
92	100	RQDSSSTGW	0.16	HLA-A*32:01
138	146	MSSLHLKRY	0.16	HLA-A*30:02
23	32	SYNLLGFLQR	0.18	HLA-A*33:01
138	146	MSSLHLKRY	0.18	HLA-A*01:01
168	176	RVEILRNFY	0.18	HLA-A*01:01
15	24	FSTTALSMSY	0.18	HLA-A*01:01
16	24	STTALSMSY	0.18	HLA-B*35:01
174	184	NFYVINRLTGY	0.19	HLA-A*26:01
124	132	EEKLEKEDF	0.19	HLA-B*44:03
133	141	TRGKRMSSL	0.2	HLA-B*08:01
176	186	YVINRLTGYLR	0.2	HLA-A*33:01
16	24	STTALSMSY	0.2	HLA-A*11:01
75	83	DAAVTIYEM	0.21	HLA-A*26:01
102	112	ETIVENLLANV	0.21	HLA-A*68:02
79	87	TIYEMLQNI	0.21	HLA-A*02:01
61	68	IPEEIKQL	0.22	HLA-B*51:01
176	184	YVINRLTGY	0.22	HLA-B*35:01
136	145	KRMSSLHLKR	0.22	HLA-A*31:01
139	147	SSLHLKRYY	0.23	HLA-B*57:01
118	126	HLKTVLEEK	0.24	HLA-A*30:01
62	71	PEEIKQLQQF	0.24	HLA-B*44:03
7	15	LQIALLLCF	0.24	HLA-B*15:01
118	126	HLKTVLEEK	0.24	HLA-A*03:01
114	122	HQRNHLKTV	0.25	HLA-B*08:01
107	116	NLLANVYHQR	0.25	HLA-A*33:01
102	110	ETIVENLLA	0.25	HLA-A*68:02
63	72	EEIKQLQQFQ	0.25	HLA-B*44:03
60	68	DIPEEIKQL	0.26	HLA-A*26:01
75	83	DAAVTIYEM	0.26	HLA-B*53:01
134	144	RGKRMSSLHLK	0.26	HLA-A*30:01
106	116	ENLLANVYHQR	0.26	HLA-A*33:01
23	32	SYNLLGFLQR	0.26	HLA-A*31:01
136	146	KRMSSLHLKRY	0.27	HLA-A*30:02
42	51	LWQLNGRLEY	0.27	HLA-A*30:02
111	119	NVYHQRNHL	0.27	HLA-A*68:02
72	81	QKEDAAVTIY	0.27	HLA-B*44:03
157	164	KEDSHCAW	0.28	HLA-B*44:02
165	173	TIVRVEILR	0.28	HLA-A*33:01
41	51	LLWQLNGRLEY	0.28	HLA-B*15:01
17	24	TTALSMSY	0.29	HLA-A*01:01
137	146	RMSSLHLKRY	0.29	HLA-B*15:01
121	129	TVLEEKLEK	0.29	HLA-A*03:01
137	146	RMSSLHLKRY	0.3	HLA-A*32:01
83	91	MLQNIFAIF	0.3	HLA-A*32:01
16	24	STTALSMSY	0.3	HLA-B*15:01
149	157	RILHYLKAK	0.3	HLA-A*11:01
84	92	LQNIFAIFR	0.3	HLA-A*31:01

62	71	PEEIKQLQQF	0.31	HLA-B*44:02
136	144	KRMSSLHLK	0.31	HLA-A*30:01
72	81	QKEDAAVTIY	0.31	HLA-B*44:02
83	91	MLQNIFAIF	0.31	HLA-B*15:01
33	43	SSNCQCQKLLW	0.31	HLA-B*57:01
15	24	FSTTALSMSY	0.32	HLA-A*26:01
104	113	IVENLLANVY	0.32	HLA-A*01:01
145	153	RYYGRILHY	0.32	HLA-A*31:01
103	113	TIVENLLANVY	0.33	HLA-A*26:01
111	119	NVYHQRNHL	0.33	HLA-B*08:01
61	71	IPEEIKQLQQF	0.33	HLA-B*35:01
92	100	RQDSSSTGW	0.33	HLA-B*58:01
111	120	NVYHQRNHLK	0.33	HLA-A*68:01
24	32	YNLLGFLQR	0.34	HLA-A*33:01
92	100	RQDSSSTGW	0.35	HLA-B*44:02
63	72	EEIKQLQQFQ	0.35	HLA-B*44:02
145	154	RYYGRILHYL	0.35	HLA-A*30:02
159	168	DSHCAWTIVR	0.36	HLA-A*33:01
73	83	KEDAAVTIYEM	0.36	HLA-B*40:01
139	147	SSLHLKRYY	0.36	HLA-B*58:01
16	24	STTALSMSY	0.37	HLA-A*32:01
103	113	TIVENLLANVY	0.37	HLA-B*44:03
157	164	KEDSHCAW	0.37	HLA-B*44:03
21	29	SMSYNLLGF	0.38	HLA-A*32:01
103	113	TIVENLLANVY	0.38	HLA-B*44:02
75	83	DAAVTIYEM	0.38	HLA-A*68:02
143	153	LKRYYGRILHY	0.38	HLA-A*30:02
138	146	MSSLHLKRY	0.38	HLA-B*57:01
137	147	RMSSLHLKRYY	0.39	HLA-A*32:01
92	100	RQDSSSTGW	0.39	HLA-B*44:03
144	153	KRYYGRILHY	0.4	HLA-A*23:01
132	141	FTRGKRMSSL	0.4	HLA-B*08:01
60	68	DIPEEIKQL	0.4	HLA-A*68:02
73	81	KEDAAVTIY	0.4	HLA-A*30:02
63	71	EEIKQLQQF	0.4	HLA-B*40:01
73	81	KEDAAVTIY	0.4	HLA-B*40:01
16	24	STTALSMSY	0.4	HLA-B*58:01
33	43	SSNCQCQKLLW	0.4	HLA-B*58:01
63	71	EEIKQLQQF	0.41	HLA-A*26:01
83	92	MLQNIFAIFR	0.41	HLA-A*33:01
41	51	LLWQLNGRLEY	0.41	HLA-A*30:02
176	186	YVINRLTGYLR	0.41	HLA-A*68:01
79	87	TIYEMLQNI	0.42	HLA-A*32:01
116	123	RNHLKTVL	0.42	HLA-B*08:01
145	153	RYYGRILHY	0.42	HLA-B*15:01
83	91	MLQNIFAIF	0.43	HLA-A*23:01
71	81	FQKEDAAVTIY	0.43	HLA-A*01:01
108	116	LLANVYHQR	0.43	HLA-A*68:01
61	71	IPEEIKQLQQF	0.44	HLA-B*44:03
111	120	NVYHQRNHLK	0.44	HLA-A*11:01
21	29	SMSYNLLGF	0.44	HLA-B*15:01
101	109	NETIVENLL	0.45	HLA-B*44:03
73	80	KEDAAVTI	0.45	HLA-B*40:01
161	169	HCAWTIVRV	0.46	HLA-A*68:02
159	166	DSHCAWTI	0.46	HLA-B*51:01
20	29	LSMSYNLLGF	0.46	HLA-B*58:01
111	120	NVYHQRNHLK	0.46	HLA-A*03:01
139	149	SSLHLKRYYGR	0.46	HLA-A*31:01
157	166	KEDSHCAWTI	0.47	HLA-B*40:01
32	40	RSSNCQCQK	0.47	HLA-A*11:01
165	173	TIVRVEILR	0.47	HLA-A*31:01
121	129	TVLEEKLEK	0.47	HLA-A*68:01
61	71	IPEEIKQLQQF	0.48	HLA-B*44:02
134	141	RGKRMSSL	0.48	HLA-B*08:01
138	146	MSSLHLKRY	0.48	HLA-B*58:01
145	153	RYYGRILHY	0.49	HLA-A*26:01

139	147	SSLHLKRYY	0.49	HLA-A*32:01
129	138	KEDFTRGKRM	0.49	HLA-B*44:02
82	92	EMLQNIFAIFR	0.49	HLA-A*33:01
145	154	RYYGRILHYL	0.49	HLA-A*30:01
137	145	RMSSLHLKR	0.49	HLA-A*30:01
20	29	LSMSYNLLGF	0.49	HLA-B*57:01
73	81	KEDAAVTIY	0.5	HLA-A*01:01
104	113	IVENLLANVY	0.5	HLA-B*44:03
114	122	HQRNHLKTV	0.5	HLA-A*02:03
137	147	RMSSLHLKRYY	0.5	HLA-B*57:01
17	24	TTALSMSY	0.51	HLA-A*26:01
139	147	SSLHLKRYY	0.51	HLA-A*01:01
176	186	YVINRLTGYLR	0.51	HLA-A*31:01
138	146	MSSLHLKRY	0.52	HLA-A*26:01
81	88	YEMLQNIF	0.52	HLA-B*40:01
81	90	YEMLQNIFAI	0.52	HLA-B*40:01
79	87	TIYEMLQNI	0.52	HLA-B*51:01
104	112	IVENLLANV	0.52	HLA-A*02:06
137	145	RMSSLHLKR	0.52	HLA-A*11:01
79	88	TIYEMLQNIF	0.53	HLA-A*24:02
169	176	VEILRNFY	0.53	HLA-B*44:03
114	122	HQRNHLKTV	0.53	HLA-A*30:01
40	48	KLLWQLNGR	0.53	HLA-A*03:01
137	144	RMSSLHLK	0.53	HLA-A*03:01
138	147	MSSLHLKRYY	0.53	HLA-B*57:01
22	32	MSYNLLGFLQR	0.53	HLA-A*68:01
104	113	IVENLLANVY	0.54	HLA-B*44:02
64	71	EIKQLQQF	0.54	HLA-B*44:03
99	109	GWNETIVENLL	0.54	HLA-B*40:01
145	153	RYYGRILHY	0.54	HLA-A*03:01
83	92	MLQNIFAIFR	0.54	HLA-A*31:01
144	153	KRYYGRILHY	0.55	HLA-A*24:02
154	164	LKAKEDSHCAW	0.55	HLA-B*57:01
155	164	KAKEDSHCAW	0.56	HLA-B*53:01
22	32	MSYNLLGFLQR	0.56	HLA-A*33:01
176	185	YVINRLTGYL	0.57	HLA-A*26:01
50	59	EYCLKDRRNF	0.57	HLA-A*23:01
144	154	KRYYGRILHYL	0.57	HLA-A*23:01
101	109	NETIVENLL	0.57	HLA-B*44:02
176	184	YVINRLTGY	0.57	HLA-A*01:01
81	88	YEMLQNIF	0.57	HLA-B*44:03
101	108	NETIVENL	0.57	HLA-B*40:01
146	153	YYGRILHY	0.57	HLA-A*30:02
121	129	TVLEEKLEK	0.57	HLA-A*30:01
120	129	KTVLEEKLEK	0.57	HLA-A*30:01
106	116	ENLLANVYHQR	0.57	HLA-A*68:01
81	88	YEMLQNIF	0.58	HLA-B*44:02
50	59	EYCLKDRRNF	0.58	HLA-A*24:02
129	138	KEDFTRGKRM	0.58	HLA-B*44:03
61	68	IPEEIKQL	0.58	HLA-B*07:02
103	112	TIVENLLANV	0.58	HLA-A*02:06
111	119	NVYHQRNHL	0.59	HLA-A*26:01
126	136	KLEKEDFTRGK	0.59	HLA-A*03:01
112	120	VYHQRNHLK	0.59	HLA-A*31:01
64	71	EIKQLQQF	0.6	HLA-B*44:02
155	164	KAKEDSHCAW	0.61	HLA-A*32:01
83	91	MLQNIFAIF	0.61	HLA-A*24:02
147	154	YGRILHYL	0.61	HLA-B*08:01
17	26	TTALSMSYNL	0.61	HLA-A*68:02
138	147	MSSLHLKRYY	0.62	HLA-A*01:01
144	153	KRYYGRILHY	0.62	HLA-A*30:01
41	49	LLWQLNGRL	0.62	HLA-A*02:01
138	147	MSSLHLKRYY	0.63	HLA-A*30:02
104	113	IVENLLANVY	0.63	HLA-A*30:02
140	149	SLHLKRYYGR	0.63	HLA-A*31:01
149	157	RILHYLKAK	0.63	HLA-A*31:01

169	176	VEILRNFY	0.64	HLA-B*44:02
167	176	VRVEILRNFY	0.64	HLA-A*01:01
43	51	WQLNGRLEY	0.64	HLA-B*35:01
169	177	VEILRNFYV	0.64	HLA-B*40:01
137	145	RMSSLHLKR	0.64	HLA-A*33:01
177	186	VINRLTGYLR	0.64	HLA-A*31:01
144	154	KRYYGRILHYL	0.65	HLA-A*24:02
112	119	VYHQRNHL	0.65	HLA-B*08:01
129	138	KEDFTRGKRM	0.65	HLA-B*40:01
147	155	YGRILHYLK	0.65	HLA-A*30:01
16	24	STTALSMSY	0.65	HLA-B*57:01
64	73	EIKQLQQFQK	0.65	HLA-A*68:01
18	26	TALSMSYNL	0.66	HLA-B*51:01
32	40	RSSNCQCQK	0.66	HLA-A*03:01
123	132	LEEKLEKEDF	0.67	HLA-B*44:02
137	146	RMSSLHLKRY	0.67	HLA-A*03:01
176	184	YVINRLTGY	0.68	HLA-A*32:01
118	126	HLKTVLEEK	0.68	HLA-A*11:01
159	168	DSHCAWTIVR	0.68	HLA-A*68:01
103	112	TIVENLLANV	0.69	HLA-A*68:02
17	24	TTALSMSY	0.69	HLA-A*30:02
92	100	RQDSSSTGW	0.69	HLA-B*57:01
163	171	AWTIVRVEI	0.7	HLA-A*24:02
44	51	QLNGRLEY	0.7	HLA-B*15:01
178	186	INRLTGYLR	0.71	HLA-A*33:01
78	88	VTIYEMLQNIF	0.71	HLA-B*57:01
164	173	WTIVRVEILR	0.71	HLA-A*68:01
93	100	QDSSSTGW	0.72	HLA-B*44:02
78	88	VTIYEMLQNIF	0.72	HLA-A*24:02
146	153	YYGRILHY	0.72	HLA-A*24:02
167	176	VRVEILRNFY	0.72	HLA-A*30:02
15	24	FSTTALSMSY	0.72	HLA-A*30:02
137	147	RMSSLHLKRYY	0.72	HLA-B*15:01
175	184	FYVINRLTGY	0.73	HLA-A*30:02
90	100	IFRQDSSSTGW	0.73	HLA-B*58:01
22	32	MSYNLLGFLQR	0.73	HLA-A*11:01
116	126	RNHLKTVLEEK	0.73	HLA-A*03:01
118	126	HLKTVLEEK	0.73	HLA-A*31:01
16	24	STTALSMSY	0.73	HLA-A*68:01
71	81	FQKEDAAVTIY	0.74	HLA-A*30:02
154	164	LKAKEDSHCAW	0.74	HLA-B*58:01
176	185	YVINRLTGYL	0.74	HLA-A*02:06
84	91	LQNIFAIF	0.74	HLA-B*15:01
123	132	LEEKLEKEDF	0.75	HLA-B*44:03
52	59	CLKDRRNF	0.75	HLA-B*08:01
44	51	QLNGRLEY	0.75	HLA-A*30:02
156	164	AKEDSHCAW	0.76	HLA-B*44:02
79	88	TIYEMLQNIF	0.76	HLA-A*23:01
164	172	WTIVRVEIL	0.76	HLA-B*08:01
114	123	HQRNHLKTVL	0.76	HLA-B*15:01
16	24	STTALSMSY	0.76	HLA-A*03:01
146	153	YYGRILHY	0.77	HLA-A*23:01
80	87	IYEMLQNI	0.77	HLA-A*24:02
100	109	WNETIVENLL	0.77	HLA-B*40:01
22	30	MSYNLLGFL	0.77	HLA-A*68:02
112	120	VYHQRNHLK	0.77	HLA-A*33:01
35	43	NCQCQKLLW	0.77	HLA-B*58:01
105	113	VENLLANVY	0.78	HLA-B*40:01
81	91	YEMLQNIFAIF	0.79	HLA-B*44:02
64	71	EIKQLQQF	0.79	HLA-A*26:01
132	141	FTRGKRMSSL	0.79	HLA-B*07:02
1	9	MTNKCLLQI	0.79	HLA-B*58:01
171	180	ILRNFYVINR	0.79	HLA-A*31:01
145	155	RYYGRILHYLK	0.8	HLA-A*30:02
112	119	VYHQRNHL	0.81	HLA-A*24:02
146	154	YYGRILHYL	0.81	HLA-B*08:01

34	43	SNCQCQKLLW	0.81	HLA-B*58:01
156	164	AKEDSHCAW	0.82	HLA-B*44:03
43	51	WQLNGRLEY	0.82	HLA-A*01:01
17	24	TTALSMSY	0.82	HLA-B*35:01
168	176	RVEILRNFY	0.83	HLA-A*32:01
81	91	YEMLQNIFAIF	0.83	HLA-B*44:03
32	40	RSSNCQCQK	0.83	HLA-A*30:01
92	100	RQDSSSTGW	0.84	HLA-B*53:01
63	71	EEIKQLQQF	0.84	HLA-B*53:01
1	9	MTNKCLLQI	0.84	HLA-A*68:02
19	27	ALSMSYNLL	0.84	HLA-A*02:03
92	100	RQDSSSTGW	0.84	HLA-B*15:01
168	176	RVEILRNFY	0.84	HLA-B*15:01
34	43	SNCQCQKLLW	0.84	HLA-B*57:01
130	138	EDFTRGKRM	0.85	HLA-B*44:02
80	87	IYEMLQNI	0.85	HLA-A*23:01
103	112	TIVENLLANV	0.85	HLA-A*02:03
105	113	VENLLANVY	0.85	HLA-B*15:01
178	186	INRLTGYLR	0.85	HLA-A*31:01
144	153	KRYYGRILHY	0.86	HLA-A*32:01
137	146	RMSSLHLKRY	0.86	HLA-A*01:01
79	88	TIYEMLQNIF	0.87	HLA-A*26:01
61	68	IPEEIKQL	0.87	HLA-B*08:01
108	116	LLANVYHQR	0.87	HLA-A*03:01
107	116	NLLANVYHQR	0.87	HLA-A*68:01
163	171	AWTIVRVEI	0.88	HLA-A*23:01
105	113	VENLLANVY	0.88	HLA-A*30:02
60	68	DIPEEIKQL	0.88	HLA-B*51:01
170	180	EILRNFYVINR	0.88	HLA-A*33:01
140	149	SLHLKRYYGR	0.88	HLA-A*33:01
137	144	RMSSLHLK	0.88	HLA-A*30:01
165	173	TIVRVEILR	0.88	HLA-A*11:01
175	184	FYVINRLTGY	0.88	HLA-B*15:01
15	24	FSTTALSMSY	0.89	HLA-B*35:01
157	166	KEDSHCAWTI	0.9	HLA-B*44:02
167	175	VRVEILRNF	0.9	HLA-A*23:01
114	123	HQRNHLKTVL	0.9	HLA-B*08:01
176	185	YVINRLTGYL	0.9	HLA-A*02:03
1	9	MTNKCLLQI	0.9	HLA-B*57:01
118	126	HLKTVLEEK	0.9	HLA-A*68:01
1	9	MTNKCLLQI	0.91	HLA-A*32:01
64	71	EIKQLQQF	0.91	HLA-B*08:01
7	15	LQIALLLCF	0.92	HLA-A*32:01
168	175	RVEILRNF	0.92	HLA-A*32:01
61	68	IPEEIKQL	0.92	HLA-B*53:01
78	88	VTIYEMLQNIF	0.92	HLA-A*23:01
72	81	QKEDAAVTIY	0.92	HLA-A*01:01
114	122	HQRNHLKTV	0.92	HLA-B*15:01
176	184	YVINRLTGY	0.92	HLA-A*68:01
63	73	EEIKQLQQFQK	0.93	HLA-B*44:03
141	149	LHLKRYYGR	0.93	HLA-A*33:01
145	153	RYYGRILHY	0.93	HLA-B*58:01
176	184	YVINRLTGY	0.94	HLA-A*11:01
155	165	KAKEDSHCAWT	0.94	HLA-B*57:01
145	154	RYYGRILHYL	0.95	HLA-A*32:01
111	119	NVYHQRNHL	0.95	HLA-B*07:02
145	153	RYYGRILHY	0.95	HLA-A*33:01
44	52	QLNGRLEYC	0.95	HLA-A*02:03
90	100	IFRQDSSSTGW	0.95	HLA-B*57:01
49	59	LEYCLKDRRNF	0.96	HLA-B*44:02
81	91	YEMLQNIFAIF	0.96	HLA-B*40:01
160	168	SHCAWTIVR	0.97	HLA-A*33:01
138	146	MSSLHLKRY	0.97	HLA-B*15:01
35	43	NCQCQKLLW	0.97	HLA-B*57:01
166	175	IVRVEILRNF	0.97	HLA-B*57:01
102	109	ETIVENLL	0.98	HLA-A*68:02

147	154	YGRILHYL	0.99	HLA-A*24:02
115	123	QRNHLKTVL	0.99	HLA-B*08:01
22	29	MSYNLLGF	0.99	HLA-B*58:01
130	138	EDFTRGKRM	1.0	HLA-B*44:03
21	29	SMSYNLLGF	1.0	HLA-A*24:02
92	100	RQDSSSTGW	1.0	HLA-A*30:02
84	92	LQNIFAIFR	1.0	HLA-A*33:01
140	147	SLHLKRYY	1.0	HLA-B*15:01

Table IV - Results of the IFN-β-MHC-II binding prediction.

Acquired with TepiTool (http://tools.iedb.org/tepitool/).

IFN-β MHC-II				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
87	101	IFAIFRQDSSSTGWN	0.04	HLA-DRB1*04:01
164	178	WTIVRVEILRNFYVI	0.12	HLA-DRB1*15:01
164	178	WTIVRVEILRNFYVI	0.36	HLA-DPA1*03:01/DPB1*04:02
103	117	TIVENLLANVYHQRN	0.56	HLA-DRB1*12:01
142	156	HLKRYYGRILHYLKA	0.77	HLA-DRB5*01:01
169	183	VEILRNFYVINRLTG	0.87	HLA-DRB1*15:01
142	156	HLKRYYGRILHYLKA	0.88	HLA-DRB1*15:01
98	112	TGWNETIVENLLANV	0.97	HLA-DRB1*13:02
69	83	QQFQKEDAAVTIYEM	1.20	HLA-DQA1*04:01/DQB1*04:02
159	173	DSHCAWTIVRVEILR	1.30	HLA-DPA1*03:01/DPB1*04:02
79	93	TIYEMLQNIFAIFRQ	1.30	HLA-DRB5*01:01
142	156	HLKRYYGRILHYLKA	1.50	HLA-DPA1*02:01/DPB1*05:01
169	183	VEILRNFYVINRLTG	1.60	HLA-DRB1*11:01
19	33	ALSMSYNLLGFLQRS	1.70	HLA-DPA1*01/DPB1*04:01
79	93	TIYEMLQNIFAIFRQ	2.36	HLA-DRB1*12:01
74	88	EDAAVTIYEMLQNIF	2.40	HLA-DQA1*03:01/DQB1*03:02
159	173	DSHCAWTIVRVEILR	2.40	HLA-DRB4*01:01
79	93	TIYEMLQNIFAIFRQ	2.50	HLA-DRB1*01:01
142	156	HLKRYYGRILHYLKA	2.70	HLA-DPA1*02:01/DPB1*01:01
164	178	WTIVRVEILRNFYVI	3.20	HLA-DPA1*02:01/DPB1*01:01
4	18	KCLLQIALLLCFSTT	3.20	HLA-DPA1*03:01/DPB1*04:02
19	33	ALSMSYNLLGFLQRS	3.30	HLA-DPA1*01:03/DPB1*02:01
79	93	TIYEMLQNIFAIFRQ	3.30	HLA-DRB1*04:01
169	183	VEILRNFYVINRLTG	3.30	HLA-DRB3*02:02
19	33	ALSMSYNLLGFLQRS	3.40	HLA-DPA1*02:01/DPB1*05:01
74	88	EDAAVTIYEMLQNIF	3.50	HLA-DQA1*04:01/DQB1*04:02
159	173	DSHCAWTIVRVEILR	3.60	HLA-DQA1*01:01/DQB1*05:01
169	183	VEILRNFYVINRLTG	3.80	HLA-DRB1*13:02
147	161	YGRILHYLKAKEDSH	4.10	HLA-DPA1*02:01/DPB1*05:01
103	117	TIVENLLANVYHQRN	4.20	HLA-DRB3*02:02
103	117	TIVENLLANVYHQRN	4.40	HLA-DRB1*13:02
47	61	GRLEYCLKDRRNFDI	4.60	HLA-DRB1*03:01
147	161	YGRILHYLKAKEDSH	4.70	HLA-DRB5*01:01
164	178	WTIVRVEILRNFYVI	4.80	HLA-DRB1*04:05
169	183	VEILRNFYVINRLTG	4.90	HLA-DQA1*01:01/DQB1*05:01
13	27	LCFSTTALSMSYNLL	5.00	HLA-DQA1*01:02/DQB1*06:02
69	83	QQFQKEDAAVTIYEM	5.00	HLA-DQA1*03:01/DQB1*03:02
164	178	WTIVRVEILRNFYVI	5.10	HLA-DPA1*02:01/DPB1*05:01
164	178	WTIVRVEILRNFYVI	5.20	HLA-DRB4*01:01
13	27	LCFSTTALSMSYNLL	5.30	HLA-DRB1*04:01
13	27	LCFSTTALSMSYNLL	5.50	HLA-DPA1*01/DPB1*04:01
38	52	CQKLLWQLNGRLEYC	5.55	HLA-DRB1*12:01
79	93	TIYEMLQNIFAIFRQ	5.70	HLA-DPA1*02:01/DPB1*05:01
38	52	CQKLLWQLNGRLEYC	6.10	HLA-DQA1*01:01/DQB1*05:01
116	130	RNHLKTVLEEKLEKE	6.20	HLA-DPA1*02:01/DPB1*01:01
74	88	EDAAVTIYEMLQNIF	6.20	HLA-DPA1*03:01/DPB1*04:02
63	77	EEIKQLQQFQKEDAA	6.20	HLA-DRB5*01:01
111	125	NVYHQRNHLKTVLEE	6.30	HLA-DQA1*04:01/DQB1*04:02

79	93	TIYEMLQNIFAIFRQ	6.30	HLA-DRB1*04:05
87	101	IFAIFRQDSSSTGWN	6.30	HLA-DRB3*02:02
19	33	ALSMSYNLLGFLQRS	6.40	HLA-DPA1*02:01/DPB1*01:01
98	112	TGWNETIVENLLANV	6.70	HLA-DPA1*03:01/DPB1*04:02
4	18	KCLLQIALLLCFSTT	6.70	HLA-DQA1*01:02/DQB1*06:02
147	161	YGRILHYLKAKEDSH	6.90	HLA-DRB1*04:05
164	178	WTIVRVEILRNFYVI	7.00	HLA-DRB1*07:01
134	148	RGKRMSSLHLKRYYG	7.10	HLA-DRB1*11:01
4	18	KCLLQIALLLCFSTT	7.35	HLA-DPA1*01/DPB1*04:01
63	77	EEIKQLQQFQKEDAA	7.40	HLA-DRB4*01:01
164	178	WTIVRVEILRNFYVI	7.60	HLA-DRB1*08:02
4	18	KCLLQIALLLCFSTT	7.60	HLA-DRB1*15:01
169	183	VEILRNFYVINRLTG	7.70	HLA-DPA1*02:01/DPB1*05:01
79	93	TIYEMLQNIFAIFRQ	7.70	HLA-DRB3*02:02
98	112	TGWNETIVENLLANV	7.80	HLA-DQA1*05:01/DQB1*02:01
69	83	QQFQKEDAAVTIYEM	7.90	HLA-DQA1*01:02/DQB1*06:02
69	83	QQFQKEDAAVTIYEM	7.90	HLA-DRB1*13:02
13	27	LCFSTTALSMSYNLL	8.00	HLA-DPA1*02:01/DPB1*01:01
19	33	ALSMSYNLLGFLQRS	8.10	HLA-DPA1*03:01/DPB1*04:02
24	38	YNLLGFLQRSSNCQC	8.10	HLA-DRB1*04:01
79	93	TIYEMLQNIFAIFRQ	8.10	HLA-DRB1*11:01
4	18	KCLLQIALLLCFSTT	8.20	HLA-DPA1*02:01/DPB1*05:01
52	66	CLKDRRNFDIPEEIK	8.20	HLA-DQA1*01:01/DQB1*05:01
164	178	WTIVRVEILRNFYVI	8.32	HLA-DRB1*12:01
74	88	EDAAVTIYEMLQNIF	8.40	HLA-DPA1*02:01/DPB1*01:01
164	178	WTIVRVEILRNFYVI	8.40	HLA-DRB1*13:02
164	178	WTIVRVEILRNFYVI	8.40	HLA-DRB3*01:01
164	178	WTIVRVEILRNFYVI	8.60	HLA-DRB1*11:01
38	52	CQKLLWQLNGRLEYC	8.70	HLA-DRB1*15:01
4	18	KCLLQIALLLCFSTT	9.00	HLA-DRB4*01:01
116	130	RNHLKTVLEEKLEKE	9.20	HLA-DPA1*02:01/DPB1*05:01
164	178	WTIVRVEILRNFYVI	9.30	HLA-DPA1*01:03/DPB1*02:01
13	27	LCFSTTALSMSYNLL	9.30	HLA-DPA1*03:01/DPB1*04:02
142	156	HLKRYYGRILHYLKA	9.40	HLA-DPA1*01/DPB1*04:01
74	88	EDAAVTIYEMLQNIF	9.60	HLA-DPA1*02:01/DPB1*05:01
74	88	EDAAVTIYEMLQNIF	9.70	HLA-DQA1*05:01/DQB1*02:01
69	83	QQFQKEDAAVTIYEM	9.70	HLA-DRB3*01:01
38	52	CQKLLWQLNGRLEYC	9.70	HLA-DRB3*02:02
4	18	KCLLQIALLLCFSTT	9.90	HLA-DPA1*01:03/DPB1*02:01
79	93	TIYEMLQNIFAIFRQ	9.90	HLA-DQA1*01:01/DQB1*05:01
24	38	YNLLGFLQRSSNCQC	9.90	HLA-DRB1*11:01

Table V – Results of the EPO-MHC-I binding prediction.

Acquired with TepiTool (http://tools.iedb.org/tepitool/).

EPO MHC-I				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
167	175	KLFRVYSNF	0.01	HLA-A*32:01
170	179	RVYSNFLRGK	0.01	HLA-A*03:01
163	171	DTFRKLFRV	0.02	HLA-A*68:02
72	80	KVNFYAWKR	0.02	HLA-A*31:01
66	76	ITVPDTKVNFY	0.02	HLA-A*01:01
170	179	RVYSNFLRGK	0.03	HLA-A*30:01
175	182	FLRGKLKL	0.03	HLA-B*08:01
99	107	EAVLRGQAL	0.03	HLA-B*08:01
67	76	TVPDTKVNFY	0.04	HLA-A*01:01
15	23	SLLSLPLGL	0.04	HLA-A*02:01
68	76	VPDTKVNFY	0.04	HLA-B*35:01
150	158	DAASAAPLR	0.04	HLA-A*68:01
68	76	VPDTKVNFY	0.05	HLA-B*53:01
67	76	TVPDTKVNFY	0.05	HLA-A*26:01
170	179	RVYSNFLRGK	0.05	HLA-A*11:01

88	96	VEVWQGLAL	0.05	HLA-B*40:01
163	170	DTFRKLFR	0.06	HLA-A*33:01
89	97	EVWQGLALL	0.06	HLA-A*68:02
158	166	RTITADTFR	0.06	HLA-A*31:01
68	78	VPDTKVNFYAW	0.07	HLA-B*53:01
158	167	RTITADTFRK	0.07	HLA-A*11:01
19	26	LPLGLPVL	0.08	HLA-B*51:01
82	90	EVGQQAVEV	0.09	HLA-A*68:02
98	107	SEAVLRGQAL	0.09	HLA-B*40:01
67	75	TVPDTKVNF	0.1	HLA-A*26:01
153	160	SAAPLRTI	0.1	HLA-B*51:01
81	91	MEVGQQAVEVW	0.1	HLA-B*44:02
68	76	VPDTKVNFY	0.11	HLA-A*01:01
15	23	SLLSLPLGL	0.11	HLA-A*02:06
81	91	MEVGQQAVEVW	0.11	HLA-B*44:03
89	97	EVWQGLALL	0.12	HLA-A*26:01
150	158	DAASAAPLR	0.12	HLA-A*33:01
83	91	VGQQAVEVW	0.12	HLA-B*58:01
128	136	GLRSLTTLL	0.14	HLA-A*02:03
175	183	FLRGKLKLY	0.14	HLA-B*15:01
31	41	RLICDSRVLQR	0.14	HLA-A*31:01
66	75	ITVPDTKVNF	0.14	HLA-B*57:01
70	80	DTKVNFYAWKR	0.15	HLA-A*33:01
113	122	QPWEPLQLHV	0.15	HLA-B*51:01
33	42	ICDSRVLQRY	0.15	HLA-A*01:01
15	23	SLLSLPLGL	0.15	HLA-A*02:03
87	96	AVEVWQGLAL	0.15	HLA-B*40:01
158	167	RTITADTFRK	0.15	HLA-A*03:01
163	172	DTFRKLFRVY	0.16	HLA-A*26:01
113	120	QPWEPLQL	0.16	HLA-B*51:01
160	169	ITADTFRKLF	0.17	HLA-B*57:01
86	94	QAVEVWQGL	0.18	HLA-A*68:02
88	97	VEVWQGLALL	0.18	HLA-B*40:01
66	75	ITVPDTKVNF	0.18	HLA-B*58:01
147	157	SPPDAASAAPL	0.19	HLA-B*07:02
181	189	KLYTGEACR	0.19	HLA-A*31:01
98	108	SEAVLRGQALL	0.2	HLA-B*40:01
83	91	VGQQAVEVW	0.2	HLA-B*57:01
160	168	ITADTFRKL	0.21	HLA-A*68:02
144	152	EAISPPDAA	0.21	HLA-A*68:02
4	11	HECPAWLW	0.21	HLA-B*44:03
135	143	LLRALGAQK	0.21	HLA-A*03:01
105	115	QALLVNSSQPW	0.21	HLA-B*57:01
167	175	KLFRVYSNF	0.22	HLA-B*15:01
4	11	HECPAWLW	0.23	HLA-B*44:02
167	175	KLFRVYSNF	0.24	HLA-A*23:01
76	83	YAWKRMEV	0.24	HLA-B*51:01
93	101	GLALLSEAV	0.24	HLA-A*02:03
113	120	QPWEPLQL	0.24	HLA-B*07:02
4	12	HECPAWLWL	0.24	HLA-B*40:01
18	26	SLPLGLPVL	0.25	HLA-A*02:01
159	167	TITADTFRK	0.25	HLA-A*11:01
2	11	GVHECPAWLW	0.25	HLA-B*57:01
38	46	VLQRYLLEA	0.26	HLA-A*02:01
22	32	GLPVLGAPPRL	0.26	HLA-A*02:01
158	166	RTITADTFR	0.26	HLA-A*11:01
167	177	KLFRVYSNFLR	0.26	HLA-A*31:01
170	177	RVYSNFLR	0.26	HLA-A*31:01
66	76	ITVPDTKVNFY	0.27	HLA-A*30:02
101	109	VLRGQALLV	0.28	HLA-A*02:03
105	115	QALLVNSSQPW	0.28	HLA-B*58:01
107	115	LLVNSSQPW	0.29	HLA-A*32:01
98	107	SEAVLRGQAL	0.29	HLA-B*44:02
113	121	QPWEPLQLH	0.29	HLA-B*35:01
131	139	SLTTLLRAL	0.29	HLA-A*02:03
18	26	SLPLGLPVL	0.29	HLA-A*02:03

18	26	SLPLGLPVL	0.29	HLA-A*02:06
2	11	GVHECPAWLW	0.29	HLA-B*58:01
160	170	ITADTFRKLFR	0.29	HLA-A*31:01
15	23	SLLSLPLGL	0.3	HLA-A*32:01
152	160	ASAAPLRTI	0.3	HLA-A*32:01
61	68	SLNENITV	0.3	HLA-A*02:03
70	78	DTKVNFYAW	0.31	HLA-B*53:01
76	83	YAWKRMEV	0.31	HLA-B*08:01
67	76	TVPDTKVNFY	0.31	HLA-A*30:02
67	76	TVPDTKVNFY	0.31	HLA-B*35:01
1	11	MGVHECPAWLW	0.31	HLA-B*57:01
67	75	TVPDTKVNF	0.32	HLA-A*24:02
158	167	RTITADTFRK	0.32	HLA-A*30:01
93	101	GLALLSEAV	0.32	HLA-A*02:01
1	9	MGVHECPAW	0.32	HLA-B*58:01
37	47	RVLQRYLLEAK	0.32	HLA-A*03:01
134	143	TLLRALGAQK	0.32	HLA-A*03:01
68	75	VPDTKVNF	0.33	HLA-B*53:01
98	107	SEAVLRGQAL	0.33	HLA-B*44:03
66	76	ITVPDTKVNFY	0.33	HLA-B*57:01
67	75	TVPDTKVNF	0.34	HLA-A*23:01
72	80	KVNFYAWKR	0.34	HLA-A*33:01
44	52	LEAKEAENI	0.34	HLA-B*40:01
70	78	DTKVNFYAW	0.34	HLA-B*57:01
164	172	TFRKLFRVY	0.35	HLA-A*30:02
23	32	LPVLGAPPRL	0.35	HLA-B*51:01
15	25	SLLSLPLGLPV	0.35	HLA-A*02:01
2	11	GVHECPAWLW	0.36	HLA-A*32:01
155	165	APLRTITADTF	0.36	HLA-B*07:02
38	46	VLQRYLLEA	0.36	HLA-A*02:03
72	80	KVNFYAWKR	0.36	HLA-A*11:01
160	169	ITADTFRKLF	0.36	HLA-B*58:01
31	41	RLICDSRVLQR	0.36	HLA-A*03:01
175	183	FLRGKLKLY	0.37	HLA-A*26:01
67	76	TVPDTKVNFY	0.37	HLA-B*53:01
167	175	KLFRVYSNF	0.37	HLA-A*24:02
37	47	RVLQRYLLEAK	0.37	HLA-A*30:01
19	26	LPLGLPVL	0.37	HLA-B*07:02
158	166	RTITADTFR	0.37	HLA-A*68:01
166	175	RKLFRVYSNF	0.38	HLA-A*32:01
161	169	TADTFRKLF	0.38	HLA-B*53:01
68	78	VPDTKVNFYAW	0.38	HLA-B*57:01
159	168	TITADTFRKL	0.39	HLA-A*68:02
150	160	DAASAAPLRTI	0.39	HLA-B*51:01
135	143	LLRALGAQK	0.4	HLA-A*30:01
45	52	EAKEAENI	0.4	HLA-B*51:01
1	11	MGVHECPAWLW	0.4	HLA-B*58:01
66	76	ITVPDTKVNFY	0.4	HLA-B*58:01
23	32	LPVLGAPPRL	0.41	HLA-B*53:01
83	91	VGQQAVEVW	0.41	HLA-B*53:01
171	180	VYSNFLRGKL	0.41	HLA-A*24:02
86	96	QAVEVWQGLAL	0.41	HLA-B*40:01
181	189	KLYTGEACR	0.41	HLA-A*03:01
1	9	MGVHECPAW	0.41	HLA-B*57:01
106	115	ALLVNSSQPW	0.42	HLA-A*32:01
82	91	EVGQQAVEVW	0.42	HLA-B*53:01
23	33	LPVLGAPPRLI	0.42	HLA-B*51:01
150	157	DAASAAPL	0.42	HLA-B*51:01
107	115	LLVNSSQPW	0.42	HLA-B*58:01
170	180	RVYSNFLRGKL	0.43	HLA-A*32:01
23	32	LPVLGAPPRL	0.43	HLA-B*07:02
160	170	ITADTFRKLFR	0.43	HLA-A*68:01
100	108	AVLRGQALL	0.44	HLA-A*32:01
160	168	ITADTFRKL	0.44	HLA-A*32:01
95	103	ALLSEAVLR	0.44	HLA-A*31:01
1	9	MGVHECPAW	0.45	HLA-B*53:01

124	132	KAVSGLRSL	0.46	HLA-A*32:01
171	180	VYSNFLRGKL	0.46	HLA-A*23:01
121	129	HVDKAVSGL	0.46	HLA-A*02:06
37	44	RVLQRYLL	0.47	HLA-B*08:01
144	154	EAISPPDAASA	0.47	HLA-A*68:02
124	132	KAVSGLRSL	0.47	HLA-B*07:02
68	75	VPDTKVNF	0.47	HLA-B*07:02
42	52	YLLEAKEAENI	0.47	HLA-A*02:01
169	179	FRVYSNFLRGK	0.47	HLA-A*03:01
174	182	NFLRGKLKL	0.48	HLA-A*23:01
170	178	RVYSNFLRG	0.48	HLA-A*30:01
113	120	QPWEPLQL	0.49	HLA-B*53:01
163	171	DTFRKLFRV	0.49	HLA-B*51:01
121	129	HVDKAVSGL	0.5	HLA-A*68:02
170	179	RVYSNFLRGK	0.5	HLA-A*31:01
160	168	ITADTFRKL	0.5	HLA-B*57:01
105	115	QALLVNSSQPW	0.51	HLA-B*53:01
88	96	VEVWQGLAL	0.51	HLA-B*44:03
4	13	HECPAWLWLL	0.51	HLA-B*40:01
175	183	FLRGKLKLY	0.51	HLA-A*30:02
66	76	ITVPDTKVNFY	0.52	HLA-B*53:01
152	160	ASAAPLRTI	0.52	HLA-B*58:01
112	120	SQPWEPLQL	0.52	HLA-A*02:06
70	78	DTKVNFYAW	0.52	HLA-B*58:01
66	76	ITVPDTKVNFY	0.53	HLA-A*26:01
113	120	QPWEPLQL	0.53	HLA-B*35:01
152	160	ASAAPLRTI	0.53	HLA-A*30:01
147	155	SPPDAASAA	0.53	HLA-B*07:02
171	179	VYSNFLRGK	0.53	HLA-A*30:01
133	143	TTLLRALGAQK	0.53	HLA-A*11:01
170	180	RVYSNFLRGKL	0.53	HLA-A*03:01
163	171	DTFRKLFRV	0.54	HLA-A*26:01
174	182	NFLRGKLKL	0.54	HLA-A*24:02
66	76	ITVPDTKVNFY	0.54	HLA-B*35:01
168	177	LFRVYSNFLR	0.54	HLA-A*33:01
160	168	ITADTFRKL	0.54	HLA-A*02:06
19	26	LPLGLPVL	0.55	HLA-B*35:01
160	168	ITADTFRKL	0.55	HLA-B*58:01
108	115	LVNSSQPW	0.55	HLA-B*58:01
155	165	APLRTITADTF	0.56	HLA-B*53:01
99	107	EAVLRGQAL	0.56	HLA-B*35:01
179	187	KLKLYTGEA	0.56	HLA-A*02:03
131	139	SLTTLLRAL	0.56	HLA-A*02:01
130	138	RSLTTLLRA	0.57	HLA-A*30:01
159	167	TITADTFRK	0.57	HLA-A*68:01
121	130	HVDKAVSGLR	0.57	HLA-A*68:01
160	168	ITADTFRKL	0.58	HLA-A*26:01
107	115	LLVNSSQPW	0.58	HLA-B*53:01
38	46	VLQRYLLEA	0.58	HLA-A*02:06
72	80	KVNFYAWKR	0.58	HLA-A*03:01
70	78	DTKVNFYAW	0.59	HLA-A*26:01
113	122	QPWEPLQLHV	0.59	HLA-B*53:01
34	42	CDSRVLQRY	0.59	HLA-A*01:01
68	75	VPDTKVNF	0.59	HLA-B*08:01
124	132	KAVSGLRSL	0.59	HLA-B*58:01
75	83	FYAWKRMEV	0.6	HLA-A*24:02
100	107	AVLRGQAL	0.6	HLA-B*08:01
68	75	VPDTKVNF	0.6	HLA-B*35:01
95	103	ALLSEAVLR	0.6	HLA-A*11:01
95	103	ALLSEAVLR	0.6	HLA-A*03:01
19	26	LPLGLPVL	0.61	HLA-B*53:01
75	83	FYAWKRMEV	0.61	HLA-B*08:01
160	170	ITADTFRKLFR	0.61	HLA-A*33:01
99	107	EAVLRGQAL	0.61	HLA-B*51:01
89	97	EVWQGLALL	0.61	HLA-A*02:06
99	107	EAVLRGQAL	0.62	HLA-A*26:01

154	162	AAPLRTITA	0.62	HLA-B*08:01
61	68	SLNENITV	0.63	HLA-A*02:01
152	160	ASAAPLRTI	0.63	HLA-B*57:01
113	121	QPWEPLQLH	0.64	HLA-B*53:01
94	101	LALLSEAV	0.64	HLA-B*51:01
11	19	WLLLSLLSL	0.64	HLA-A*02:01
158	165	RTITADTF	0.64	HLA-B*58:01
81	91	MEVGQQAVEVW	0.64	HLA-B*58:01
158	168	RTITADTFRKL	0.64	HLA-B*57:01
6	14	CPAWLWLLL	0.65	HLA-B*53:01
87	96	AVEVWQGLAL	0.65	HLA-B*44:03
4	12	HECPAWLWL	0.66	HLA-B*44:03
161	169	TADTFRKLF	0.66	HLA-B*35:01
39	47	LQRYLLEAK	0.66	HLA-A*30:01
25	33	VLGAPPRLI	0.66	HLA-A*02:03
128	137	GLRSLTTLLR	0.66	HLA-A*03:01
17	26	LSLPLGLPVL	0.66	HLA-B*57:01
124	132	KAVSGLRSL	0.66	HLA-B*57:01
107	115	LLVNSSQPW	0.66	HLA-B*57:01
64	72	ENITVPDTK	0.66	HLA-A*68:01
88	96	VEVWQGLAL	0.67	HLA-B*44:02
8	16	AWLWLLLSL	0.67	HLA-A*23:01
167	175	KLFRVYSNF	0.67	HLA-A*30:02
32	41	LICDSRVLQR	0.67	HLA-A*33:01
110	118	NSSQPWEPL	0.68	HLA-A*68:02
161	168	TADTFRKL	0.68	HLA-B*51:01
161	169	TADTFRKLF	0.69	HLA-A*01:01
68	76	VPDTKVNFY	0.69	HLA-A*30:02
163	171	DTFRKLFRV	0.7	HLA-B*08:01
128	136	GLRSLTTLL	0.7	HLA-A*02:01
93	101	GLALLSEAV	0.7	HLA-A*02:06
57	66	AEHCSLNENI	0.71	HLA-B*44:02
121	130	HVDKAVSGLR	0.71	HLA-A*33:01
124	132	KAVSGLRSL	0.71	HLA-A*30:01
98	108	SEAVLRGQALL	0.72	HLA-B*44:02
38	47	VLQRYLLEAK	0.72	HLA-A*03:01
87	96	AVEVWQGLAL	0.73	HLA-B*44:02
168	176	LFRVYSNFL	0.73	HLA-B*08:01
99	108	EAVLRGQALL	0.73	HLA-B*08:01
173	180	SNFLRGKL	0.73	HLA-B*08:01
17	26	LSLPLGLPVL	0.73	HLA-B*58:01
161	169	TADTFRKLF	0.73	HLA-B*58:01
37	47	RVLQRYLLEAK	0.73	HLA-A*11:01
81	91	MEVGQQAVEVW	0.73	HLA-B*57:01
158	168	RTITADTFRKL	0.74	HLA-A*32:01
163	172	DTFRKLFRVY	0.74	HLA-A*01:01
57	66	AEHCSLNENI	0.74	HLA-B*40:01
67	75	TVPDTKVNF	0.74	HLA-A*68:02
157	166	LRTITADTFR	0.74	HLA-A*68:01
86	94	QAVEVWQGL	0.75	HLA-A*26:01
167	176	KLFRVYSNFL	0.75	HLA-A*32:01
112	120	SQPWEPLQL	0.75	HLA-A*24:02
66	75	ITVPDTKVNF	0.75	HLA-A*24:02
168	177	LFRVYSNFLR	0.75	HLA-A*31:01
72	80	KVNFYAWKR	0.75	HLA-A*68:01
65	73	NITVPDTKV	0.76	HLA-A*68:02
128	137	GLRSLTTLLR	0.76	HLA-A*31:01
174	182	NFLRGKLKL	0.77	HLA-B*08:01
108	115	LVNSSQPW	0.77	HLA-B*57:01
66	75	ITVPDTKVNF	0.78	HLA-A*23:01
75	83	FYAWKRMEV	0.78	HLA-A*23:01
67	77	TVPDTKVNFYA	0.78	HLA-A*68:02
67	77	TVPDTKVNFYA	0.79	HLA-A*01:01
153	162	SAAPLRTITA	0.79	HLA-A*68:02
33	42	ICDSRVLQRY	0.79	HLA-A*30:02
173	181	SNFLRGKLK	0.79	HLA-A*30:01

44	52	LEAKEAENI	0.8	HLA-B*44:02
4	14	HECPAWLWLLL	0.8	HLA-B*40:01
81	90	MEVGQQAVEV	0.8	HLA-B*40:01
68	76	VPDTKVNFY	0.8	HLA-B*07:02
68	75	VPDTKVNF	0.8	HLA-B*51:01
4	12	HECPAWLWL	0.81	HLA-B*44:02
174	183	NFLRGKLKLY	0.82	HLA-A*30:02
82	91	EVGQQAVEVW	0.82	HLA-B*58:01
133	143	TTLLRALGAQK	0.82	HLA-A*30:01
159	167	TITADTFRK	0.82	HLA-A*03:01
98	108	SEAVLRGQALL	0.83	HLA-B*44:03
99	107	EAVLRGQAL	0.83	HLA-B*07:02
155	162	APLRTITA	0.84	HLA-B*07:02
99	107	EAVLRGQAL	0.84	HLA-A*68:02
133	143	TTLLRALGAQK	0.84	HLA-A*03:01
167	175	KLFRVYSNF	0.84	HLA-B*57:01
57	66	AEHCSLNENI	0.85	HLA-B*44:03
44	52	LEAKEAENI	0.85	HLA-B*44:03
112	120	SQPWEPLQL	0.85	HLA-B*40:01
111	120	SSQPWEPLQL	0.85	HLA-B*58:01
160	170	ITADTFRKLFR	0.85	HLA-A*11:01
108	115	LVNSSQPW	0.86	HLA-B*53:01
112	120	SQPWEPLQL	0.86	HLA-A*23:01
99	108	EAVLRGQALL	0.86	HLA-A*68:02
144	152	EAISPPDAA	0.87	HLA-B*35:01
112	121	SQPWEPLQLH	0.87	HLA-B*15:01
170	177	RVYSNFLR	0.87	HLA-A*03:01
71	78	TKVNFYAW	0.87	HLA-B*57:01
130	139	RSLTTLLRAL	0.87	HLA-B*57:01
23	32	LPVLGAPPRL	0.88	HLA-B*35:01
14	23	LSLLSLPLGL	0.88	HLA-A*02:01
121	130	HVDKAVSGLR	0.88	HLA-A*31:01
118	126	LQLHVDKAV	0.89	HLA-A*02:06
124	132	KAVSGLRSL	0.89	HLA-A*02:06
32	41	LICDSRVLQR	0.89	HLA-A*31:01
67	75	TVPDTKVNF	0.9	HLA-A*32:01
68	76	VPDTKVNFY	0.9	HLA-B*08:01
113	122	QPWEPLQLHV	0.9	HLA-B*07:02
106	115	ALLVNSSQPW	0.9	HLA-B*58:01
113	120	QPWEPLQL	0.91	HLA-B*08:01
35	43	DSRVLQRYL	0.91	HLA-B*08:01
112	120	SQPWEPLQL	0.91	HLA-B*15:01
34	42	CDSRVLQRY	0.92	HLA-B*44:03
131	139	SLTTLLRAL	0.92	HLA-B*08:01
100	108	AVLRGQALL	0.92	HLA-A*02:06
160	169	ITADTFRKLF	0.93	HLA-A*32:01
127	135	SGLRSLTTL	0.93	HLA-B*08:01
173	183	SNFLRGKLKLY	0.93	HLA-A*30:02
17	25	LSLPLGLPV	0.93	HLA-B*51:01
68	78	VPDTKVNFYAW	0.93	HLA-B*58:01
22	32	GLPVLGAPPRL	0.93	HLA-A*02:06
179	189	KLKLYTGEACR	0.93	HLA-A*31:01
94	102	LALLSEAVL	0.94	HLA-B*51:01
34	42	CDSRVLQRY	0.95	HLA-B*44:02
160	169	ITADTFRKLF	0.95	HLA-A*26:01
98	107	SEAVLRGQAL	0.95	HLA-B*08:01
71	80	TKVNFYAWKR	0.95	HLA-A*33:01
131	139	SLTTLLRAL	0.95	HLA-A*02:06
114	122	PWEPLQLHV	0.96	HLA-A*24:02
67	75	TVPDTKVNF	0.96	HLA-B*35:01
167	175	KLFRVYSNF	0.96	HLA-B*58:01
37	45	RVLQRYLLE	0.96	HLA-A*30:01
171	179	VYSNFLRGK	0.96	HLA-A*31:01
121	129	HVDKAVSGL	0.97	HLA-A*26:01
35	43	DSRVLQRYL	0.97	HLA-B*51:01
96	106	LLSEAVLRGQA	0.97	HLA-A*02:01

66	75	ITVPDTKVNF	0.98	HLA-A*32:01
8	16	AWLWLLLSL	0.98	HLA-A*24:02
160	168	ITADTFRKL	0.98	HLA-B*51:01
66	75	ITVPDTKVNF	0.99	HLA-A*26:01
25	33	VLGAPPRLI	0.99	HLA-A*32:01
152	160	ASAAPLRTI	0.99	HLA-A*68:02
170	177	RVYSNFLR	0.99	HLA-A*11:01
158	166	RTITADTFR	0.99	HLA-A*03:01
86	94	QAVEVWQGL	1.0	HLA-B*35:01
64	73	ENITVPDTKV	1.0	HLA-A*68:02
167	177	KLFRVYSNFLR	1.0	HLA-A*03:01
67	76	TVPDTKVNFY	1.0	HLA-B*15:01

Table VI – Results of the EPO-MHC-II binding prediction.
Acquired with TepiTool (http://tools.iedb.org/tepitool/).

Eritropoietina MHC-II				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
166	180	RKLFRVYSNFLRGKL	0.12	HLA-DRB1*15:01
11	25	WLLLSLLSLPLGLPV	0.24	HLA-DRB1*01:01
5	19	ECPAWLWLLLSLLSL	0.25	HLA-DPA1*03:01/DPB1*04:02
29	43	PPRLICDSRVLQRYL	0.62	HLA-DRB1*03:01
5	19	ECPAWLWLLLSLLSL	0.66	HLA-DQA1*01:01/DQB1*05:01
166	180	RKLFRVYSNFLRGKL	0.67	HLA-DPA1*02:01/DPB1*05:01
129	143	LRSLTTLLRALGAQK	0.91	HLA-DRB1*11:01
124	138	KAVSGLRSLTTLLRA	0.95	HLA-DPA1*02:01/DPB1*05:01
11	25	WLLLSLLSLPLGLPV	0.99	HLA-DPA1*03:01/DPB1*04:02
129	143	LRSLTTLLRALGAQK	1.10	HLA-DPA1*02:01/DPB1*05:01
74	88	NFYAWKRMEVGQQAV	1.10	HLA-DRB1*04:05
74	88	NFYAWKRMEVGQQAV	1.10	HLA-DRB1*08:02
104	118	GQALLVNSSQPWEPL	1.10	HLA-DRB3*02:02
129	143	LRSLTTLLRALGAQK	1.20	HLA-DRB1*08:02
158	172	RTITADTFRKLFRVY	1.40	HLA-DPA1*01/DPB1*04:01
99	113	EAVLRGQALLVNSSQ	1.40	HLA-DRB1*13:02
124	138	KAVSGLRSLTTLLRA	1.40	HLA-DRB5*01:01
166	180	RKLFRVYSNFLRGKL	1.50	HLA-DRB5*01:01
5	19	ECPAWLWLLLSLLSL	1.60	HLA-DRB1*11:01
129	143	LRSLTTLLRALGAQK	1.80	HLA-DRB5*01:01
5	19	ECPAWLWLLLSLLSL	2.00	HLA-DPA1*01:03/DPB1*02:01
124	138	KAVSGLRSLTTLLRA	2.00	HLA-DRB1*04:01
124	138	KAVSGLRSLTTLLRA	2.00	HLA-DRB1*11:01
158	172	RTITADTFRKLFRVY	2.30	HLA-DPA1*01:03/DPB1*02:01
129	143	LRSLTTLLRALGAQK	2.30	HLA-DQA1*01:02/DQB1*06:02
166	180	RKLFRVYSNFLRGKL	2.55	HLA-DPA1*01/DPB1*04:01
166	180	RKLFRVYSNFLRGKL	2.60	HLA-DPA1*02:01/DPB1*01:01
5	19	ECPAWLWLLLSLLSL	2.70	HLA-DPA1*01/DPB1*04:01
166	180	RKLFRVYSNFLRGKL	2.90	HLA-DPA1*01:03/DPB1*02:01
11	25	WLLLSLLSLPLGLPV	3.00	HLA-DRB1*11:01
158	172	RTITADTFRKLFRVY	3.10	HLA-DPA1*02:01/DPB1*05:01
104	118	GQALLVNSSQPWEPL	3.10	HLA-DRB1*13:02
11	25	WLLLSLLSLPLGLPV	3.20	HLA-DRB1*07:01
158	172	RTITADTFRKLFRVY	3.30	HLA-DPA1*03:01/DPB1*04:02
124	138	KAVSGLRSLTTLLRA	3.40	HLA-DRB1*01:01
129	143	LRSLTTLLRALGAQK	3.40	HLA-DRB1*04:01
89	103	EVWQGLALLSEAVLR	3.50	HLA-DQA1*03:01/DQB1*03:02
124	138	KAVSGLRSLTTLLRA	3.60	HLA-DRB1*04:05
11	25	WLLLSLLSLPLGLPV	3.60	HLA-DRB1*15:01
166	180	RKLFRVYSNFLRGKL	3.70	HLA-DRB1*08:02
11	25	WLLLSLLSLPLGLPV	4.10	HLA-DPA1*02:01/DPB1*01:01
5	19	ECPAWLWLLLSLLSL	4.20	HLA-DPA1*02:01/DPB1*05:01
89	103	EVWQGLALLSEAVLR	4.50	HLA-DQA1*04:01/DQB1*04:02
5	19	ECPAWLWLLLSLLSL	4.70	HLA-DRB1*04:01

166	180	RKLFRVYSNFLRGKL	4.80	HLA-DRB3*02:02
178	192	GKLKLYTGEACRTGD	5.10	HLA-DRB1*15:01
99	113	EAVLRGQALLVNSSQ	5.20	HLA-DRB1*01:01
94	108	LALLSEAVLRGQALL	5.30	HLA-DRB4*01:01
11	25	WLLLSLLSLPLGLPV	5.40	HLA-DPA1*01/DPB1*04:01
116	130	EPLQLHVDKAVSGLR	5.40	HLA-DRB1*03:01
134	148	TLLRALGAQKEAISP	5.40	HLA-DRB1*04:05
11	25	WLLLSLLSLPLGLPV	5.50	HLA-DRB1*12:01
124	138	KAVSGLRSLTTLLRA	5.50	HLA-DRB1*15:01
166	180	RKLFRVYSNFLRGKL	5.60	HLA-DRB1*13:02
20	34	PLGLPVLGAPPRLIC	5.70	HLA-DQA1*05:01/DQB1*03:01
69	83	PDTKVNFYAWKRMEV	5.90	HLA-DPA1*01:03/DPB1*02:01
5	19	ECPAWLWLLLSLLSL	5.90	HLA-DRB1*01:01
124	138	KAVSGLRSLTTLLRA	6.00	HLA-DPA1*01/DPB1*04:01
171	185	VYSNFLRGKLKLYTG	6.00	HLA-DPA1*02:01/DPB1*05:01
166	180	RKLFRVYSNFLRGKL	6.00	HLA-DRB1*01:01
5	19	ECPAWLWLLLSLLSL	6.10	HLA-DPA1*02:01/DPB1*01:01
11	25	WLLLSLLSLPLGLPV	6.20	HLA-DPA1*01:03/DPB1*02:01
89	103	EVWQGLALLSEAVLR	6.20	HLA-DPA1*02:01/DPB1*01:01
158	172	RTITADTFRKLFRVY	6.20	HLA-DRB1*03:01
11	25	WLLLSLLSLPLGLPV	6.30	HLA-DQA1*01:01/DQB1*05:01
74	88	NFYAWKRMEVGQQAV	6.30	HLA-DRB1*04:01
134	148	TLLRALGAQKEAISP	6.40	HLA-DRB5*01:01
34	48	CDSRVLQRYLLEAKE	6.50	HLA-DQA1*01:01/DQB1*05:01
171	185	VYSNFLRGKLKLYTG	6.50	HLA-DRB1*11:01
11	25	WLLLSLLSLPLGLPV	6.50	HLA-DRB5*01:01
74	88	NFYAWKRMEVGQQAV	6.60	HLA-DRB1*11:01
129	143	LRSLTTLLRALGAQK	6.70	HLA-DRB1*01:01
11	25	WLLLSLLSLPLGLPV	6.90	HLA-DRB4*01:01
89	103	EVWQGLALLSEAVLR	7.00	HLA-DRB1*01:01
129	143	LRSLTTLLRALGAQK	7.00	HLA-DRB1*04:05
116	130	EPLQLHVDKAVSGLR	7.00	HLA-DRB3*02:02
79	93	KRMEVGQQAVEVWQG	7.10	HLA-DQA1*01:02/DQB1*06:02
134	148	TLLRALGAQKEAISP	7.10	HLA-DRB1*01:01
5	19	ECPAWLWLLLSLLSL	7.10	HLA-DRB1*15:01
129	143	LRSLTTLLRALGAQK	7.15	HLA-DRB1*12:01
166	180	RKLFRVYSNFLRGKL	7.20	HLA-DRB1*04:05
34	48	CDSRVLQRYLLEAKE	7.30	HLA-DPA1*01:03/DPB1*02:01
89	103	EVWQGLALLSEAVLR	7.40	HLA-DQA1*05:01/DQB1*02:01
79	93	KRMEVGQQAVEVWQG	7.40	HLA-DQA1*05:01/DQB1*02:01
129	143	LRSLTTLLRALGAQK	7.60	HLA-DPA1*03:01/DPB1*04:02
166	180	RKLFRVYSNFLRGKL	7.60	HLA-DPA1*03:01/DPB1*04:02
158	172	RTITADTFRKLFRVY	7.70	HLA-DPA1*02:01/DPB1*01:01
166	180	RKLFRVYSNFLRGKL	7.70	HLA-DRB1*11:01
99	113	EAVLRGQALLVNSSQ	7.80	HLA-DQA1*01:02/DQB1*06:02
124	138	KAVSGLRSLTTLLRA	7.80	HLA-DQA1*01:02/DQB1*06:02
124	138	KAVSGLRSLTTLLRA	7.90	HLA-DPA1*03:01/DPB1*04:02
151	165	AASAAPLRTITADTF	8.00	HLA-DQA1*05:01/DQB1*03:01
145	159	AISPPDAASAAPLRT	8.00	HLA-DQA1*05:01/DQB1*03:01
89	103	EVWQGLALLSEAVLR	8.10	HLA-DRB1*04:05
89	103	EVWQGLALLSEAVLR	8.15	HLA-DPA1*01/DPB1*04:01
5	19	ECPAWLWLLLSLLSL	8.20	HLA-DRB1*04:05
166	180	RKLFRVYSNFLRGKL	8.20	HLA-DRB1*07:01
158	172	RTITADTFRKLFRVY	8.20	HLA-DRB3*01:01
94	108	LALLSEAVLRGQALL	8.40	HLA-DRB1*01:01
84	98	GQQAVEVWQGLALLS	8.40	HLA-DRB1*15:01
84	98	GQQAVEVWQGLALLS	8.60	HLA-DPA1*03:01/DPB1*04:02
5	19	ECPAWLWLLLSLLSL	8.70	HLA-DRB5*01:01
89	103	EVWQGLALLSEAVLR	8.90	HLA-DQA1*01:02/DQB1*06:02
94	108	LALLSEAVLRGQALL	9.00	HLA-DPA1*02:01/DPB1*05:01
171	185	VYSNFLRGKLKLYTG	9.05	HLA-DRB1*12:01
34	48	CDSRVLQRYLLEAKE	9.20	HLA-DPA1*02:01/DPB1*01:01
89	103	EVWQGLALLSEAVLR	9.20	HLA-DPA1*03:01/DPB1*04:02
129	143	LRSLTTLLRALGAQK	9.30	HLA-DRB1*15:01
145	159	AISPPDAASAAPLRT	9.60	HLA-DQA1*04:01/DQB1*04:02
89	103	EVWQGLALLSEAVLR	9.80	HLA-DQA1*05:01/DQB1*03:01

69	83	PDTKVNFYAWKRMEV	9.80	HLA-DRB1*11:01
57	71	AEHCSLNENITVPDT	9.80	HLA-DRB1*13:02
99	113	EAVLRGQALLVNSSQ	9.80	HLA-DRB4*01:01
20	34	PLGLPVLGAPPRLIC	9.90	HLA-DRB1*13:02
11	25	WLLLSLLSLPLGLPV	9.90	HLA-DRB1*13:02

Table VII - Results of the GHRH-MHC-I binding prediction.
Acquired with TepiTool (http://tools.iedb.org/tepitool/).

GHRH MHC-I				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
43	51	KVLGQLSAR	0.01	HLA-A*31:01
35	43	AIFTNSYRK	0.01	HLA-A*11:01
32	41	YADAIFTNSY	0.02	HLA-A*01:01
35	43	AIFTNSYRK	0.03	HLA-A*03:01
93	101	SILVALLQK	0.04	HLA-A*11:01
43	52	KVLGQLSARK	0.04	HLA-A*03:01
20	27	SPPPPLTL	0.04	HLA-B*07:02
34	42	DAIFTNSYR	0.04	HLA-A*68:01
34	42	DAIFTNSYR	0.05	HLA-A*33:01
93	101	SILVALLQK	0.05	HLA-A*03:01
31	41	RYADAIFTNSY	0.06	HLA-A*30:02
20	27	SPPPPLTL	0.06	HLA-B*51:01
32	41	YADAIFTNSY	0.08	HLA-B*35:01
87	96	KQMELESILV	0.09	HLA-A*02:06
89	98	MELESILVAL	0.09	HLA-B*40:01
52	60	KLLQDIMSR	0.1	HLA-A*31:01
87	95	KQMELESIL	0.11	HLA-A*02:06
83	91	WAEQKQMEL	0.13	HLA-B*08:01
91	99	LESILVALL	0.13	HLA-B*40:01
42	51	RKVLGQLSAR	0.14	HLA-A*31:01
75	83	LGRQVDSMW	0.14	HLA-B*57:01
34	41	DAIFTNSY	0.15	HLA-B*35:01
20	29	SPPPPLTLRM	0.15	HLA-B*07:02
52	60	KLLQDIMSR	0.15	HLA-A*03:01
35	43	AIFTNSYRK	0.19	HLA-A*30:01
87	95	KQMELESIL	0.19	HLA-B*15:01
43	52	KVLGQLSARK	0.2	HLA-A*11:01
89	99	MELESILVALL	0.2	HLA-B*40:01
75	83	LGRQVDSMW	0.2	HLA-B*58:01
31	41	RYADAIFTNSY	0.22	HLA-A*23:01
20	28	SPPPPLTLR	0.22	HLA-A*33:01
74	83	RLGRQVDSMW	0.23	HLA-A*32:01
31	41	RYADAIFTNSY	0.23	HLA-A*24:02
91	98	LESILVAL	0.23	HLA-B*40:01
84	91	AEQKQMEL	0.23	HLA-B*40:01
43	51	KVLGQLSAR	0.23	HLA-A*03:01
5	13	VFFFVILTL	0.25	HLA-A*23:01
87	96	KQMELESILV	0.26	HLA-A*02:01
43	51	KVLGQLSAR	0.26	HLA-A*11:01
74	83	RLGRQVDSMW	0.27	HLA-B*57:01
44	52	VLGQLSARK	0.28	HLA-A*03:01
40	48	SYRKVLGQL	0.29	HLA-A*23:01
34	43	DAIFTNSYRK	0.3	HLA-A*68:01
1	8	MPLWVFFF	0.32	HLA-B*53:01
40	48	SYRKVLGQL	0.32	HLA-A*24:02
31	41	RYADAIFTNSY	0.32	HLA-A*01:01
93	101	SILVALLQK	0.32	HLA-A*30:01
67	75	QERGARARL	0.32	HLA-B*40:01
20	27	SPPPPLTL	0.34	HLA-B*08:01
33	41	ADAIFTNSY	0.34	HLA-B*44:02
87	95	KQMELESIL	0.35	HLA-A*32:01

96	104	VALLQKHSR	0.35	HLA-A*33:01
77	87	RQVDSMWAEQK	0.35	HLA-A*11:01
92	101	ESILVALLQK	0.35	HLA-A*68:01
20	28	SPPPPLTLR	0.35	HLA-A*68:01
33	41	ADAIFTNSY	0.37	HLA-B*44:03
34	41	DAIFTNSY	0.4	HLA-A*26:01
87	95	KQMELESIL	0.41	HLA-A*02:01
20	27	SPPPPLTL	0.42	HLA-B*53:01
43	52	KVLGQLSARK	0.42	HLA-A*30:01
64	72	ESNQERGAR	0.42	HLA-A*33:01
32	41	YADAIFTNSY	0.43	HLA-B*53:01
5	13	VFFFVILTL	0.43	HLA-A*24:02
67	75	QERGARARL	0.43	HLA-B*44:02
47	54	QLSARKLL	0.43	HLA-B*08:01
74	83	RLGRQVDSMW	0.43	HLA-B*58:01
69	77	RGARARLGR	0.43	HLA-A*31:01
96	104	VALLQKHSR	0.43	HLA-A*31:01
81	91	SMWAEQKQMEL	0.44	HLA-A*02:01
52	60	KLLQDIMSR	0.45	HLA-A*11:01
1	9	MPLWVFFFV	0.46	HLA-B*51:01
32	41	YADAIFTNSY	0.47	HLA-A*26:01
87	95	KQMELESIL	0.47	HLA-B*40:01
67	75	QERGARARL	0.48	HLA-B*44:03
20	29	SPPPPLTLRM	0.5	HLA-B*51:01
81	89	SMWAEQKQM	0.5	HLA-A*02:01
64	72	ESNQERGAR	0.51	HLA-A*68:01
20	29	SPPPPLTLRM	0.52	HLA-B*53:01
33	43	ADAIFTNSYRK	0.52	HLA-A*11:01
51	60	RKLLQDIMSR	0.53	HLA-A*03:01
84	91	AEQKQMEL	0.54	HLA-B*44:02
19	27	CSPPPPLTL	0.54	HLA-A*24:02
33	41	ADAIFTNSY	0.55	HLA-A*01:01
89	97	MELESILVA	0.55	HLA-B*40:01
81	89	SMWAEQKQM	0.55	HLA-B*15:01
32	41	YADAIFTNSY	0.56	HLA-A*30:02
77	87	RQVDSMWAEQK	0.56	HLA-A*03:01
32	42	YADAIFTNSYR	0.57	HLA-A*01:01
19	27	CSPPPPLTL	0.58	HLA-A*23:01
43	51	KVLGQLSAR	0.58	HLA-A*33:01
20	29	SPPPPLTLRM	0.6	HLA-B*35:01
90	98	ELESILVAL	0.6	HLA-A*68:02
20	27	SPPPPLTL	0.61	HLA-B*35:01
72	82	RARLGRQVDSM	0.61	HLA-B*07:02
33	43	ADAIFTNSYRK	0.61	HLA-A*03:01
19	27	CSPPPPLTL	0.64	HLA-B*58:01
32	42	YADAIFTNSYR	0.64	HLA-A*68:01
19	27	CSPPPPLTL	0.65	HLA-B*51:01
40	48	SYRKVLGQL	0.65	HLA-A*30:01
28	37	RMRRYADAIF	0.65	HLA-B*15:01
37	45	FTNSYRKVL	0.66	HLA-B*08:01
35	43	AIFTNSYRK	0.66	HLA-A*68:01
81	89	SMWAEQKQM	0.67	HLA-A*32:01
84	91	AEQKQMEL	0.67	HLA-B*44:03
89	98	MELESILVAL	0.67	HLA-B*44:03
1	8	MPLWVFFF	0.67	HLA-B*35:01
33	42	ADAIFTNSYR	0.67	HLA-A*68:01
75	83	LGRQVDSMW	0.68	HLA-B*53:01
21	29	PPPPLTLRM	0.68	HLA-B*53:01
91	99	LESILVALL	0.68	HLA-B*44:03
42	51	RKVLGQLSAR	0.68	HLA-A*03:01
18	28	HCSPPPPLTLR	0.69	HLA-A*33:01
43	51	KVLGQLSAR	0.69	HLA-A*30:01
51	60	RKLLQDIMSR	0.69	HLA-A*31:01
90	98	ELESILVAL	0.7	HLA-B*08:01
91	99	LESILVALL	0.73	HLA-B*44:02
42	52	RKVLGQLSARK	0.74	HLA-A*03:01

73	83	ARLGRQVDSMW	0.74	HLA-B*57:01
89	98	MELESILVAL	0.75	HLA-B*44:02
21	29	PPPPLTLRM	0.75	HLA-B*51:01
17	27	SHCSPPPPLTL	0.77	HLA-A*24:02
19	28	CSPPPPLTLR	0.77	HLA-A*68:01
87	96	KQMELESILV	0.79	HLA-A*02:03
60	69	RQQGESNQER	0.8	HLA-A*31:01
38	45	TNSYRKVL	0.81	HLA-B*08:01
18	27	HCSPPPPLTL	0.81	HLA-B*58:01
1	8	MPLWVFFF	0.82	HLA-B*51:01
95	104	LVALLQKHSR	0.84	HLA-A*33:01
33	41	ADAIFTNSY	0.85	HLA-A*30:02
87	97	KQMELESILVA	0.85	HLA-A*02:06
31	41	RYADAIFTNSY	0.86	HLA-A*32:01
17	27	SHCSPPPPLTL	0.87	HLA-A*23:01
78	87	QVDSMWAEQK	0.87	HLA-A*11:01
95	104	LVALLQKHSR	0.88	HLA-A*68:01
89	99	MELESILVALL	0.9	HLA-B*44:03
18	28	HCSPPPPLTLR	0.9	HLA-A*68:01
20	28	SPPPPLTLR	0.92	HLA-A*26:01
43	53	KVLGQLSARKL	0.92	HLA-A*32:01
28	36	RMRRYADAI	0.92	HLA-A*32:01
46	54	GQLSARKLL	0.92	HLA-B*15:01
44	52	VLGQLSARK	0.93	HLA-A*11:01
43	51	KVLGQLSAR	0.94	HLA-A*68:01
84	94	AEQKQMELESI	0.96	HLA-B*40:01
84	94	AEQKQMELESI	0.98	HLA-B*44:02
83	91	WAEQKQMEL	0.98	HLA-B*35:01
18	28	HCSPPPPLTLR	0.98	HLA-A*31:01
92	99	ESILVALL	0.99	HLA-B*51:01

Table VIII – Results of the GHRH-MHC-II binding prediction.
Acquired with TepiTool (http://tools.iedb.org/tepitool/).

GHRH MHC-II				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
1	15	MPLWVFFFVILTLSN	0.05	HLA-DQA1*01:01/DQB1*05:01
1	15	MPLWVFFFVILTLSN	0.14	HLA-DPA1*01/DPB1*04:01
1	15	MPLWVFFFVILTLSN	0.24	HLA-DPA1*03:01/DPB1*04:02
1	15	MPLWVFFFVILTLSN	0.31	HLA-DPA1*02:01/DPB1*05:01
39	53	NSYRKVLGQLSARKL	0.38	HLA-DRB1*01:01
1	15	MPLWVFFFVILTLSN	0.38	HLA-DRB1*04:05
1	15	MPLWVFFFVILTLSN	0.43	HLA-DPA1*02:01/DPB1*01:01
1	15	MPLWVFFFVILTLSN	0.54	HLA-DPA1*01:03/DPB1*02:01
6	20	FFFVILTLSNSSHCS	0.67	HLA-DRB1*04:01
6	20	FFFVILTLSNSSHCS	0.77	HLA-DRB1*04:05
39	53	NSYRKVLGQLSARKL	1.20	HLA-DRB5*01:01
27	41	LRMRRYADAIFTNSY	1.50	HLA-DRB1*08:02
39	53	NSYRKVLGQLSARKL	1.90	HLA-DRB1*04:05
6	20	FFFVILTLSNSSHCS	2.10	HLA-DRB1*08:02
27	41	LRMRRYADAIFTNSY	2.60	HLA-DPA1*02:01/DPB1*05:01
39	53	NSYRKVLGQLSARKL	2.60	HLA-DRB1*08:02
1	15	MPLWVFFFVILTLSN	2.80	HLA-DQA1*05:01/DQB1*02:01
90	104	ELESILVALLQKHSR	3.00	HLA-DRB1*11:01
33	47	ADAIFTNSYRKVLGQ	3.30	HLA-DRB1*13:02
85	99	EQKQMELESILVALL	3.50	HLA-DPA1*02:01/DPB1*01:01
1	15	MPLWVFFFVILTLSN	3.50	HLA-DRB1*04:01
39	53	NSYRKVLGQLSARKL	3.50	HLA-DRB1*11:01
6	20	FFFVILTLSNSSHCS	3.60	HLA-DRB3*02:02
6	20	FFFVILTLSNSSHCS	3.80	HLA-DPA1*02:01/DPB1*01:01
1	15	MPLWVFFFVILTLSN	3.90	HLA-DRB1*11:01
39	53	NSYRKVLGQLSARKL	4.00	HLA-DRB1*04:01

90	104	ELESILVALLQKHSR	4.30	HLA-DRB1*12:01
6	20	FFFVILTLSNSSHCS	4.40	HLA-DPA1*03:01/DPB1*04:02
33	47	ADAIFTNSYRKVLGQ	4.50	HLA-DRB3*02:02
44	58	VLGQLSARKLLQDIM	4.60	HLA-DRB5*01:01
85	99	EQKQMELESILVALL	4.80	HLA-DQA1*01:02/DQB1*06:02
22	36	PPPLTLRMRRYADAI	5.00	HLA-DRB1*11:01
85	99	EQKQMELESILVALL	5.10	HLA-DPA1*03:01/DPB1*04:02
33	47	ADAIFTNSYRKVLGQ	5.10	HLA-DRB1*11:01
1	15	MPLWVFFFVILTLSN	5.10	HLA-DRB1*15:01
85	99	EQKQMELESILVALL	5.40	HLA-DRB4*01:01
6	20	FFFVILTLSNSSHCS	6.10	HLA-DRB1*11:01
90	104	ELESILVALLQKHSR	6.30	HLA-DPA1*03:01/DPB1*04:02
27	41	LRMRRYADAIFTNSY	6.30	HLA-DRB1*15:01
90	104	ELESILVALLQKHSR	6.30	HLA-DRB4*01:01
33	47	ADAIFTNSYRKVLGQ	6.60	HLA-DPA1*01:03/DPB1*02:01
39	53	NSYRKVLGQLSARKL	6.75	HLA-DRB1*12:01
39	53	NSYRKVLGQLSARKL	7.10	HLA-DRB1*07:01
39	53	NSYRKVLGQLSARKL	7.10	HLA-DRB1*09:01
50	64	ARKLLQDIMSRQQGE	7.20	HLA-DRB1*03:01
39	53	NSYRKVLGQLSARKL	7.70	HLA-DRB4*01:01
85	99	EQKQMELESILVALL	7.80	HLA-DQA1*05:01/DQB1*02:01
1	15	MPLWVFFFVILTLSN	7.80	HLA-DRB1*08:02
39	53	NSYRKVLGQLSARKL	7.80	HLA-DRB1*15:01
6	20	FFFVILTLSNSSHCS	8.00	HLA-DRB1*07:01
50	64	ARKLLQDIMSRQQGE	8.00	HLA-DRB4*01:01
44	58	VLGQLSARKLLQDIM	8.50	HLA-DRB4*01:01
90	104	ELESILVALLQKHSR	8.80	HLA-DPA1*02:01/DPB1*01:01
6	20	FFFVILTLSNSSHCS	8.80	HLA-DRB1*15:01
39	53	NSYRKVLGQLSARKL	8.80	HLA-DRB3*02:02
6	20	FFFVILTLSNSSHCS	9.05	HLA-DPA1*01/DPB1*04:01
44	58	VLGQLSARKLLQDIM	9.20	HLA-DRB1*09:01
44	58	VLGQLSARKLLQDIM	9.30	HLA-DRB1*01:01
6	20	FFFVILTLSNSSHCS	9.50	HLA-DPA1*02:01/DPB1*05:01
90	104	ELESILVALLQKHSR	9.50	HLA-DQA1*01:02/DQB1*06:02
6	20	FFFVILTLSNSSHCS	9.80	HLA-DRB1*01:01
6	20	FFFVILTLSNSSHCS	9.95	HLA-DRB1*12:01

Table IX – Results of the Albumin-MHC-I binding prediction.

Acquired with TepiTool (http://tools.iedb.org/tepitool/).

Albumin MHC-I				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
364	372	DYSVVLLLR	0.01	HLA-A*33:01
172	181	YFYAPELLFF	0.01	HLA-A*23:01
375	383	KTYETTLEK	0.01	HLA-A*30:01
154	162	NEETFLKKY	0.01	HLA-B*44:02
172	180	YFYAPELLF	0.01	HLA-A*23:01
227	235	LQKFGERAF	0.01	HLA-B*15:01
172	181	YFYAPELLFF	0.01	HLA-A*24:02
427	435	FQNALLVRY	0.01	HLA-B*15:01
417	425	ELFEQLGEY	0.01	HLA-A*26:01
257	265	KLVTDLTKV	0.01	HLA-A*02:03
244	252	SQRFPKAEF	0.01	HLA-B*15:01
374	383	AKTYETTLEK	0.01	HLA-A*11:01
173	181	FYAPELLFF	0.01	HLA-A*23:01
513	521	SALEVDETY	0.01	HLA-B*35:01
154	162	NEETFLKKY	0.01	HLA-B*44:03
173	181	FYAPELLFF	0.01	HLA-A*24:02
473	481	AEDYLSVVL	0.01	HLA-B*40:01
375	383	KTYETTLEK	0.01	HLA-A*11:01
375	383	KTYETTLEK	0.01	HLA-A*03:01
250	258	AEFAEVSKL	0.01	HLA-B*40:01

423	431	GEYKFQNAL	0.01	HLA-B*40:01
221	229	RLKCASLQK	0.02	HLA-A*30:01
247	255	FPKAEFAEV	0.02	HLA-B*51:01
170	178	HPYFYAPEL	0.02	HLA-B*51:01
172	180	YFYAPELLF	0.02	HLA-A*24:02
65	73	KLVNEVTEF	0.02	HLA-B*15:01
15	23	SAYSRGVFR	0.02	HLA-A*31:01
393	401	CYAKVFDEF	0.02	HLA-A*24:02
374	383	AKTYETTLEK	0.02	HLA-A*03:01
257	265	KLVTDLTKV	0.02	HLA-A*02:01
439	447	VPQVSTPTL	0.02	HLA-B*07:02
560	568	KPKATKEQL	0.02	HLA-B*07:02
15	23	SAYSRGVFR	0.03	HLA-A*33:01
439	447	VPQVSTPTL	0.03	HLA-B*51:01
546	553	QIKKQTAL	0.03	HLA-B*08:01
156	164	ETFLKKYLY	0.03	HLA-A*26:01
5	13	TFISLLFLF	0.03	HLA-A*23:01
393	401	CYAKVFDEF	0.03	HLA-A*23:01
250	258	AEFAEVSKL	0.03	HLA-B*44:02
545	553	RQIKKQTAL	0.03	HLA-B*15:01
476	484	YLSVVLNQL	0.03	HLA-A*02:01
221	229	RLKCASLQK	0.03	HLA-A*03:01
257	265	KLVTDLTKV	0.03	HLA-A*02:06
250	258	AEFAEVSKL	0.03	HLA-B*44:03
362	370	HPDYSVVLL	0.04	HLA-B*53:01
345	353	EAKDVFLGM	0.04	HLA-A*26:01
518	526	DETYVPKEF	0.04	HLA-B*44:02
234	242	AFKAWAVAR	0.04	HLA-A*31:01
476	484	YLSVVLNQL	0.04	HLA-A*02:03
170	178	HPYFYAPEL	0.04	HLA-B*35:01
518	526	DETYVPKEF	0.04	HLA-B*44:03
136	144	LPRLVRPEV	0.04	HLA-B*07:02
427	435	FQNALLVRY	0.05	HLA-A*30:02
170	180	HPYFYAPELLF	0.05	HLA-B*53:01
58	66	CPFEDHVKL	0.05	HLA-B*53:01
348	356	DVFLGMFLY	0.05	HLA-A*26:01
58	66	CPFEDHVKL	0.05	HLA-B*51:01
155	164	EETFLKKYLY	0.05	HLA-B*44:02
92	101	TLFGDKLCTV	0.05	HLA-A*02:03
171	181	PYFYAPELLFF	0.05	HLA-A*23:01
171	180	PYFYAPELLF	0.05	HLA-A*23:01
52	60	AQYLQQCPF	0.05	HLA-B*15:01
158	166	FLKKYLYEI	0.05	HLA-A*02:03
571	579	VMDDFAAFV	0.05	HLA-A*02:01
170	178	HPYFYAPEL	0.05	HLA-B*07:02
15	23	SAYSRGVFR	0.05	HLA-A*68:01
239	247	AVARLSQRF	0.06	HLA-A*32:01
396	404	KVFDEFKPL	0.06	HLA-A*32:01
65	73	KLVNEVTEF	0.06	HLA-A*32:01
100	108	TVATLRETY	0.06	HLA-A*26:01
570	578	AVMDDFAAF	0.06	HLA-A*26:01
322	329	MPADLPSL	0.06	HLA-B*51:01
173	180	FYAPELLF	0.06	HLA-A*24:02
171	180	PYFYAPELLF	0.06	HLA-A*24:02
171	181	PYFYAPELLFF	0.06	HLA-A*24:02
525	533	EFNAETFTF	0.06	HLA-A*24:02
233	242	RAFKAWAVAR	0.06	HLA-A*31:01
570	578	AVMDDFAAF	0.06	HLA-B*15:01
155	164	EETFLKKYLY	0.06	HLA-B*44:03
362	370	HPDYSVVLL	0.06	HLA-B*35:01
423	432	GEYKFQNALL	0.06	HLA-B*40:01
170	178	HPYFYAPEL	0.07	HLA-B*53:01
439	447	VPQVSTPTL	0.07	HLA-B*53:01
181	189	FAKRYKAAF	0.07	HLA-B*08:01
525	533	EFNAETFTF	0.07	HLA-A*23:01
171	179	PYFYAPELL	0.07	HLA-A*23:01

150	158	AFHDNEETF	0.07	HLA-A*24:02
342	350	NYAEAKDVF	0.07	HLA-A*24:02
171	179	PYFYAPELL	0.07	HLA-A*24:02
161	169	KYLYEIARR	0.07	HLA-A*31:01
14	23	SSAYSRGVFR	0.07	HLA-A*31:01
396	405	KVFDEFKPLV	0.07	HLA-A*02:06
438	447	KVPQVSTPTL	0.07	HLA-B*07:02
386	394	AAADPHECY	0.07	HLA-B*35:01
58	66	CPFEDHVKL	0.07	HLA-B*35:01
253	262	AEVSKLVTDL	0.07	HLA-B*40:01
335	343	ESKDVCKNY	0.08	HLA-A*26:01
426	435	KFQNALLVRY	0.08	HLA-A*30:02
11	19	FLFSSAYSR	0.08	HLA-A*33:01
545	553	RQIKKQTAL	0.08	HLA-B*08:01
150	158	AFHDNEETF	0.08	HLA-A*23:01
173	180	FYAPELLF	0.08	HLA-A*23:01
155	163	EETFLKKYL	0.08	HLA-B*44:02
164	172	YEIARRHPY	0.08	HLA-B*44:02
15	24	SAYSRGVFRR	0.08	HLA-A*31:01
277	287	CADDRADLAKY	0.08	HLA-A*01:01
226	235	SLQKFGERAF	0.08	HLA-B*15:01
512	521	FSALEVDETY	0.08	HLA-B*35:01
362	370	HPDYSVVLL	0.08	HLA-B*07:02
155	163	EETFLKKYL	0.08	HLA-B*44:03
83	93	AENCDKSLHTL	0.08	HLA-B*40:01
165	173	EIARRHPYF	0.09	HLA-A*26:01
100	108	TVATLRETY	0.09	HLA-A*30:02
234	242	AFKAWAVAR	0.09	HLA-A*33:01
424	434	EYKFQNALLVR	0.09	HLA-A*33:01
529	537	ETFTFHADI	0.09	HLA-A*68:02
512	521	FSALEVDETY	0.09	HLA-A*01:01
5	13	TFISLLFLF	0.09	HLA-A*24:02
476	484	YLSVVLNQL	0.09	HLA-A*02:06
100	108	TVATLRETY	0.09	HLA-B*15:01
92	101	TLFGDKLCTV	0.09	HLA-A*02:01
334	343	VESKDVCKNY	0.09	HLA-B*44:03
513	521	SALEVDETY	0.1	HLA-B*53:01
389	397	DPHECYAKV	0.1	HLA-B*51:01
434	442	RYTKKVPQV	0.1	HLA-A*24:02
396	404	KVFDEFKPL	0.1	HLA-A*02:06
570	579	AVMDDFAAFV	0.1	HLA-A*02:06
16	24	AYSRGVFRR	0.1	HLA-A*31:01
100	108	TVATLRETY	0.1	HLA-B*35:01
426	434	KFQNALLVR	0.1	HLA-A*31:01
439	447	VPQVSTPTL	0.1	HLA-B*35:01
164	172	YEIARRHPY	0.1	HLA-B*44:03
249	258	KAEFAEVSKL	0.1	HLA-B*40:01
570	578	AVMDDFAAF	0.11	HLA-A*32:01
215	223	ASSAKQRLK	0.11	HLA-A*30:01
522	531	VPKEFNAETF	0.11	HLA-B*53:01
166	174	IARRHPYFY	0.11	HLA-A*30:02
386	394	AAADPHECY	0.11	HLA-A*30:02
232	242	ERAFKAWAVAR	0.11	HLA-A*33:01
362	370	HPDYSVVLL	0.11	HLA-B*51:01
254	262	EVSKLVTDL	0.11	HLA-A*68:02
446	454	TLVEVSRNL	0.11	HLA-A*02:03
334	343	VESKDVCKNY	0.11	HLA-B*44:02
322	329	MPADLPSL	0.11	HLA-B*35:01
225	233	ASLQKFGER	0.11	HLA-A*31:01
559	568	HKPKATKEQL	0.11	HLA-B*07:02
158	166	FLKKYLYEI	0.11	HLA-A*02:01
552	560	ALVELVKHK	0.11	HLA-A*03:01
430	438	ALLVRYTKK	0.11	HLA-A*03:01
39	48	GEENFKALVL	0.11	HLA-B*40:01
40	48	EENFKALVL	0.11	HLA-B*40:01
373	381	LAKTYETTL	0.12	HLA-B*08:01

2	11	KWVTFISLLF	0.12	HLA-A*23:01
441	450	QVSTPTLVEV	0.12	HLA-A*68:02
386	394	AAADPHECY	0.12	HLA-A*01:01
40	48	EENFKALVL	0.12	HLA-B*44:02
571	579	VMDDFAAFV	0.12	HLA-A*02:06
47	55	VLIAFAQYL	0.12	HLA-A*02:01
126	136	FLQHKDDNPNL	0.12	HLA-A*02:01
178	186	LLFFAKRYK	0.12	HLA-A*03:01
15	24	SAYSRGVFRR	0.12	HLA-A*68:01
434	442	RYTKKVPQV	0.13	HLA-A*23:01
133	140	NPNLPRLV	0.13	HLA-B*51:01
136	144	LPRLVRPEV	0.13	HLA-B*51:01
392	401	ECYAKVFDEF	0.13	HLA-A*24:02
236	246	KAWAVARLSQR	0.13	HLA-A*31:01
97	105	KLCTVATLR	0.13	HLA-A*31:01
15	23	SAYSRGVFR	0.13	HLA-A*11:01
423	431	GEYKFQNAL	0.13	HLA-B*44:03
158	166	FLKKYLYEI	0.14	HLA-B*08:01
342	350	NYAEAKDVF	0.14	HLA-A*23:01
519	528	ETYVPKEFNA	0.14	HLA-A*68:02
449	457	EVSRNLGKV	0.14	HLA-A*68:02
549	557	KQTALVELV	0.14	HLA-A*02:06
386	394	AAADPHECY	0.14	HLA-B*15:01
40	48	EENFKALVL	0.14	HLA-B*44:03
317	326	VENDEMPADL	0.14	HLA-B*40:01
345	354	EAKDVFLGMF	0.15	HLA-A*26:01
362	371	HPDYSVVLLL	0.15	HLA-B*53:01
15	24	SAYSRGVFRR	0.15	HLA-A*33:01
353	361	MFLYEYARR	0.15	HLA-A*33:01
442	450	VSTPTLVEV	0.15	HLA-A*02:06
558	568	KHKPKATKEQL	0.15	HLA-B*07:02
437	447	KKVPQVSTPTL	0.15	HLA-B*07:02
322	329	MPADLPSL	0.15	HLA-B*07:02
524	533	KEFNAETFTF	0.15	HLA-B*44:03
201	209	CLLPKLDEL	0.15	HLA-A*02:01
155	162	EETFLKKY	0.15	HLA-B*44:03
443	452	STPTLVEVSR	0.15	HLA-A*68:01
374	383	AKTYETTLEK	0.16	HLA-A*30:01
490	499	KTPVSDRVTK	0.16	HLA-A*30:01
165	173	EIARRHPYF	0.16	HLA-B*08:01
170	178	HPYFYAPEL	0.16	HLA-B*08:01
156	164	ETFLKKYLY	0.16	HLA-A*01:01
524	533	KEFNAETFTF	0.16	HLA-A*24:02
256	265	SKLVTDLTKV	0.16	HLA-A*02:03
570	578	AVMDDFAAF	0.16	HLA-B*35:01
490	499	KTPVSDRVTK	0.16	HLA-A*11:01
247	255	FPKAEFAEV	0.17	HLA-B*08:01
25	34	DAHKSEVAHR	0.17	HLA-A*33:01
419	427	FEQLGEYKF	0.17	HLA-B*44:02
423	431	GEYKFQNAL	0.17	HLA-B*44:02
341	350	KNYAEAKDVF	0.17	HLA-A*24:02
545	553	RQIKKQTAL	0.17	HLA-A*02:06
170	180	HPYFYAPELLF	0.17	HLA-B*35:01
419	427	FEQLGEYKF	0.17	HLA-B*44:03
9	17	LLFLFSSAY	0.17	HLA-B*15:01
219	229	KQRLKCASLQK	0.18	HLA-A*30:01
524	533	KEFNAETFTF	0.18	HLA-A*23:01
391	401	HECYAKVFDEF	0.18	HLA-A*24:02
387	394	AADPHECY	0.18	HLA-A*01:01
539	547	TLSEKERQI	0.18	HLA-A*02:03
401	411	FKPLVEEPQNL	0.18	HLA-A*02:01
524	533	KEFNAETFTF	0.18	HLA-B*40:01
375	384	KTYETTLEKC	0.18	HLA-A*03:01
149	158	TAFHDNEETF	0.19	HLA-B*53:01
322	329	MPADLPSL	0.19	HLA-B*53:01
392	401	ECYAKVFDEF	0.19	HLA-A*23:01

345	353	EAKDVFLGM	0.19	HLA-A*68:02
2	11	KWVTFISLLF	0.19	HLA-A*24:02
524	533	KEFNAETFTF	0.19	HLA-B*44:02
545	553	RQIKKQTAL	0.2	HLA-A*32:01
504	512	SLVNRRPCF	0.2	HLA-B*08:01
219	227	KQRLKCASL	0.2	HLA-B*08:01
170	180	HPYFYAPELLF	0.2	HLA-A*23:01
439	448	VPQVSTPTLV	0.2	HLA-B*51:01
344	354	AEAKDVFLGMF	0.2	HLA-B*44:02
158	166	FLKKYLYEI	0.2	HLA-A*02:06
417	425	ELFEQLGEY	0.2	HLA-B*35:01
250	259	AEFAEVSKLV	0.2	HLA-B*44:03
421	431	QLGEYKFQNAL	0.2	HLA-B*40:01
550	558	QTALVELVK	0.2	HLA-A*11:01
419	427	FEQLGEYKF	0.2	HLA-B*40:01
344	351	AEAKDVFL	0.2	HLA-B*40:01
177	185	ELLFFAKRY	0.21	HLA-A*26:01
165	174	EIARRHPYFY	0.21	HLA-A*26:01
34	43	RFKDLGEENF	0.21	HLA-A*23:01
333	343	FVESKDVCKNY	0.21	HLA-A*01:01
155	162	EETFLKKY	0.21	HLA-B*44:02
502	512	TESLVNRRPCF	0.21	HLA-B*44:02
250	259	AEFAEVSKLV	0.21	HLA-B*44:02
344	354	AEAKDVFLGMF	0.21	HLA-B*44:03
446	454	TLVEVSRNL	0.21	HLA-A*02:01
396	405	KVFDEFKPLV	0.21	HLA-A*02:01
239	247	AVARLSQRF	0.21	HLA-B*15:01
417	425	ELFEQLGEY	0.21	HLA-B*15:01
20	28	GVFRRDAHK	0.21	HLA-A*03:01
439	447	VPQVSTPTL	0.22	HLA-B*08:01
14	23	SSAYSRGVFR	0.22	HLA-A*33:01
34	43	RFKDLGEENF	0.22	HLA-A*24:02
83	93	AENCDKSLHTL	0.22	HLA-B*44:02
516	526	EVDETYVPKEF	0.22	HLA-B*44:02
522	531	VPKEFNAETF	0.22	HLA-B*35:01
242	250	RLSQRFPKA	0.22	HLA-A*02:03
247	255	FPKAEFAEV	0.22	HLA-B*07:02
362	369	HPDYSVVL	0.22	HLA-B*07:02
322	330	MPADLPSLA	0.22	HLA-B*35:01
344	353	AEAKDVFLGM	0.22	HLA-B*44:03
516	526	EVDETYVPKEF	0.22	HLA-B*44:03
373	383	LAKTYETTLEK	0.22	HLA-A*11:01
386	396	AAADPHECYAK	0.22	HLA-A*11:01
215	223	ASSAKQRLK	0.22	HLA-A*11:01
426	435	KFQNALLVRY	0.22	HLA-B*15:01
391	401	HECYAKVFDEF	0.23	HLA-A*23:01
2	10	KWVTFISLL	0.23	HLA-A*23:01
20	28	GVFRRDAHK	0.23	HLA-A*30:01
563	571	ATKEQLKAV	0.23	HLA-A*30:01
174	181	YAPELLFF	0.23	HLA-A*24:02
47	55	VLIAFAQYL	0.23	HLA-A*02:03
201	209	CLLPKLDEL	0.23	HLA-A*02:06
154	164	NEETFLKKYLY	0.23	HLA-B*44:03
402	411	KPLVEEPQNL	0.23	HLA-A*02:01
344	353	AEAKDVFLGM	0.23	HLA-B*40:01
225	235	ASLQKFGERAF	0.23	HLA-B*15:01
375	383	KTYETTLEK	0.23	HLA-A*31:01
14	23	SSAYSRGVFR	0.23	HLA-A*68:01
155	164	EETFLKKYLY	0.24	HLA-A*26:01
431	439	LLVRYTKKV	0.24	HLA-A*02:03
152	162	HDNEETFLKKY	0.24	HLA-B*44:03
369	377	LLLRLAKTY	0.24	HLA-B*15:01
226	236	SLQKFGERAFK	0.24	HLA-A*03:01
329	337	LAADFVESK	0.24	HLA-A*68:01
341	350	KNYAEAKDVF	0.25	HLA-A*23:01
174	181	YAPELLFF	0.25	HLA-A*23:01

30	38	EVAHRFKDL	0.25	HLA-B*08:01
156	163	ETFLKKYL	0.25	HLA-B*08:01
560	568	KPKATKEQL	0.25	HLA-B*08:01
15	23	SAYSRGVFR	0.25	HLA-A*30:01
2	10	KWVTFISLL	0.25	HLA-A*24:02
344	353	AEAKDVFLGM	0.25	HLA-B*44:02
118	126	QEPERNECF	0.25	HLA-B*44:02
149	158	TAFHDNEETF	0.25	HLA-B*35:01
473	481	AEDYLSVVL	0.25	HLA-B*44:03
571	579	VMDDFAAFV	0.25	HLA-A*02:03
502	512	TESLVNRRPCF	0.25	HLA-B*44:03
153	162	DNEETFLKKY	0.25	HLA-B*44:03
92	101	TLFGDKLCTV	0.25	HLA-A*02:06
47	55	VLIAFAQYL	0.25	HLA-A*02:06
543	553	KERQIKKQTAL	0.25	HLA-B*40:01
221	230	RLKCASLQKF	0.25	HLA-B*15:01
552	560	ALVELVKHK	0.25	HLA-A*11:01
516	524	EVDETYVPK	0.25	HLA-A*68:01
11	19	FLFSSAYSR	0.25	HLA-A*68:01
169	179	RHPYFYAPELL	0.26	HLA-A*23:01
473	481	AEDYLSVVL	0.26	HLA-B*44:02
238	246	WAVARLSQR	0.26	HLA-A*33:01
69	78	EVTEFAKTCV	0.26	HLA-A*68:02
422	431	LGEYKFQNAL	0.26	HLA-B*40:01
373	383	LAKTYETTLEK	0.26	HLA-A*03:01
11	19	FLFSSAYSR	0.26	HLA-A*31:01
156	164	ETFLKKYLY	0.26	HLA-A*68:01
169	179	RHPYFYAPELL	0.27	HLA-A*24:02
10	19	LFLFSSAYSR	0.27	HLA-A*33:01
362	369	HPDYSVVL	0.27	HLA-B*35:01
169	178	RHPYFYAPEL	0.27	HLA-B*07:02
201	209	CLLPKLDEL	0.27	HLA-A*02:03
126	136	FLQHKDDNPNL	0.27	HLA-A*02:06
170	179	HPYFYAPELL	0.28	HLA-A*23:01
149	158	TAFHDNEETF	0.28	HLA-A*24:02
556	565	LVKHKPKATK	0.28	HLA-A*30:01
417	427	ELFEQLGEYKF	0.28	HLA-B*44:02
513	521	SALEVDETY	0.28	HLA-A*30:02
154	164	NEETFLKKYLY	0.28	HLA-B*44:02
152	162	HDNEETFLKKY	0.28	HLA-B*44:02
442	450	VSTPTLVEV	0.28	HLA-A*68:02
396	404	KVFDEFKPL	0.28	HLA-A*02:03
563	571	ATKEQLKAV	0.28	HLA-A*02:03
522	531	VPKEFNAETF	0.28	HLA-B*07:02
490	499	KTPVSDRVTK	0.28	HLA-A*03:01
513	521	SALEVDETY	0.28	HLA-B*58:01
166	174	IARRHPYFY	0.28	HLA-B*57:01
386	394	AAADPHECY	0.29	HLA-A*26:01
569	578	KAVMDDFAAF	0.29	HLA-A*32:01
173	182	FYAPELLFFA	0.29	HLA-A*23:01
217	225	SAKQRLKCA	0.29	HLA-B*08:01
217	227	SAKQRLKCASL	0.29	HLA-B*08:01
170	179	HPYFYAPELL	0.29	HLA-A*24:02
394	401	YAKVFDEF	0.29	HLA-A*24:02
367	375	VVLLLRLAK	0.29	HLA-A*30:01
513	521	SALEVDETY	0.29	HLA-A*01:01
417	425	ELFEQLGEY	0.29	HLA-A*30:02
58	67	CPFEDHVKLV	0.29	HLA-B*51:01
362	369	HPDYSVVL	0.29	HLA-B*51:01
362	371	HPDYSVVLLL	0.29	HLA-B*51:01
396	405	KVFDEFKPLV	0.29	HLA-A*02:03
11	21	FLFSSAYSRGV	0.29	HLA-A*02:03
464	472	HPEAKRMPC	0.29	HLA-B*07:02
328	337	SLAADFVESK	0.29	HLA-A*11:01
215	223	ASSAKQRLK	0.29	HLA-A*03:01
149	158	TAFHDNEETF	0.3	HLA-A*23:01

57	66	QCPFEDHVKL	0.3	HLA-B*53:01
4	13	VTFISLLFLF	0.3	HLA-A*23:01
28	36	KSEVAHRFK	0.3	HLA-A*30:01
34	44	RFKDLGEENFK	0.3	HLA-A*30:01
417	427	ELFEQLGEYKF	0.3	HLA-B*44:03
402	411	KPLVEEPQNL	0.3	HLA-A*02:03
300	308	KECCEKPLL	0.3	HLA-B*40:01
524	531	KEFNAETF	0.3	HLA-B*40:01
20	28	GVFRRDAHK	0.3	HLA-A*11:01
19	28	RGVFRRDAHK	0.3	HLA-A*03:01
170	179	HPYFYAPELL	0.31	HLA-B*53:01
173	182	FYAPELLFFA	0.31	HLA-A*24:02
434	442	RYTKKVPQV	0.31	HLA-A*30:01
166	174	IARRHPYFY	0.31	HLA-A*30:01
156	164	ETFLKKYLY	0.31	HLA-A*30:02
66	74	LVNEVTEFA	0.31	HLA-A*68:02
322	330	MPADLPSLA	0.31	HLA-B*51:01
470	479	MPCAEDYLSV	0.31	HLA-B*51:01
545	553	RQIKKQTAL	0.31	HLA-B*40:01
367	375	VVLLLRLAK	0.31	HLA-A*11:01
513	521	SALEVDETY	0.32	HLA-A*26:01
231	238	GERAFKAW	0.32	HLA-B*44:02
83	93	AENCDKSLHTL	0.32	HLA-B*44:03
118	126	QEPERNECF	0.32	HLA-B*44:03
126	136	FLQHKDDNPNL	0.32	HLA-A*02:03
58	66	CPFEDHVKL	0.32	HLA-B*07:02
348	358	DVFLGMFLYEY	0.33	HLA-A*26:01
136	144	LPRLVRPEV	0.33	HLA-B*08:01
180	188	FFAKRYKAA	0.33	HLA-B*08:01
452	460	RNLGKVGSK	0.33	HLA-A*30:01
164	174	YEIARRHPYFY	0.33	HLA-B*44:03
247	255	FPKAEFAEV	0.33	HLA-B*35:01
570	579	AVMDDFAAFV	0.33	HLA-A*02:03
570	579	AVMDDFAAFV	0.33	HLA-A*02:01
39	47	GEENFKALV	0.33	HLA-B*40:01
219	229	KQRLKCASLQK	0.33	HLA-A*03:01
417	426	ELFEQLGEYK	0.33	HLA-A*68:01
172	180	YFYAPELLF	0.34	HLA-A*32:01
194	202	QAADKAACL	0.34	HLA-B*08:01
424	432	EYKFQNALL	0.34	HLA-A*24:02
153	162	DNEETFLKKY	0.34	HLA-B*44:02
16	24	AYSRGVFRR	0.34	HLA-A*33:01
231	238	GERAFKAW	0.34	HLA-B*44:03
170	179	HPYFYAPELL	0.34	HLA-B*51:01
256	265	SKLVTDLTKV	0.34	HLA-A*02:01
446	454	TLVEVSRNL	0.34	HLA-A*02:06
570	578	AVMDDFAAF	0.34	HLA-A*02:06
375	384	KTYETTLEKC	0.34	HLA-A*11:01
172	182	YFYAPELLFFA	0.35	HLA-A*23:01
475	484	DYLSVVLNQL	0.35	HLA-A*23:01
170	180	HPYFYAPELLF	0.35	HLA-A*24:02
517	526	VDETYVPKEF	0.35	HLA-B*44:02
100	108	TVATLRETY	0.35	HLA-A*01:01
255	264	VSKLVTDLTK	0.35	HLA-A*30:01
13	23	FSSAYSRGVFR	0.35	HLA-A*33:01
517	526	VDETYVPKEF	0.35	HLA-B*44:03
28	35	KSEVAHRF	0.35	HLA-B*58:01
367	375	VVLLLRLAK	0.35	HLA-A*03:01
65	75	KLVNEVTEFAK	0.35	HLA-A*03:01
375	383	KTYETTLEK	0.35	HLA-A*68:01
25	34	DAHKSEVAHR	0.35	HLA-A*68:01
550	558	QTALVELVK	0.35	HLA-A*68:01
239	247	AVARLSQRF	0.36	HLA-A*26:01
221	230	RLKCASLQKF	0.36	HLA-A*32:01
544	553	ERQIKKQTAL	0.36	HLA-B*08:01
253	262	AEVSKLVTDL	0.36	HLA-B*44:02

164	174	YEIARRHPYFY	0.36	HLA-B*44:02
322	331	MPADLPSLAA	0.36	HLA-B*35:01
252	259	FAEVSKLV	0.36	HLA-B*51:01
402	411	KPLVEEPQNL	0.36	HLA-B*07:02
361	370	RHPDYSVVLL	0.36	HLA-B*07:02
246	255	RFPKAEFAEV	0.36	HLA-B*07:02
200	209	ACLLPKLDEL	0.36	HLA-A*02:01
14	23	SSAYSRGVFR	0.36	HLA-A*11:01
239	249	AVARLSQRFPK	0.36	HLA-A*11:01
513	521	SALEVDETY	0.36	HLA-B*15:01
15	23	SAYSRGVFR	0.36	HLA-A*03:01
394	401	YAKVFDEF	0.37	HLA-A*23:01
172	182	YFYAPELLFFA	0.37	HLA-A*24:02
362	370	HPDYSVVLL	0.37	HLA-B*08:01
345	353	EAKDVFLGM	0.37	HLA-B*35:01
401	411	FKPLVEEPQNL	0.37	HLA-A*02:06
569	578	KAVMDDFAAF	0.37	HLA-B*15:01
328	337	SLAADFVESK	0.37	HLA-A*68:01
254	262	EVSKLVTDL	0.38	HLA-A*26:01
512	521	FSALEVDETY	0.38	HLA-B*53:01
475	484	DYLSVVLNQL	0.38	HLA-A*24:02
253	262	AEVSKLVTDL	0.38	HLA-B*44:03
326	334	LPSLAADFV	0.38	HLA-B*51:01
170	179	HPYFYAPELL	0.38	HLA-B*07:02
267	275	TECCHGDLL	0.38	HLA-B*40:01
556	565	LVKHKPKATK	0.38	HLA-A*03:01
328	337	SLAADFVESK	0.38	HLA-A*03:01
417	427	ELFEQLGEYKF	0.39	HLA-A*26:01
524	531	KEFNAETF	0.39	HLA-B*44:03
348	356	DVFLGMFLY	0.39	HLA-B*35:01
250	259	AEFAEVSKLV	0.39	HLA-B*40:01
239	247	AVARLSQRF	0.39	HLA-B*58:01
15	24	SAYSRGVFRR	0.39	HLA-A*11:01
242	252	RLSQRFPKAEF	0.4	HLA-A*32:01
56	66	QQCPFEDHVKL	0.4	HLA-B*53:01
163	173	LYEIARRHPYF	0.4	HLA-A*24:02
214	223	KASSAKQRLK	0.4	HLA-A*30:01
362	371	HPDYSVVLLL	0.4	HLA-B*07:02
11	21	FLFSSAYSRGV	0.4	HLA-A*02:01
396	404	KVFDEFKPL	0.4	HLA-A*02:01
174	182	YAPELLFFA	0.4	HLA-A*02:06
243	252	LSQRFPKAEF	0.4	HLA-B*15:01
521	531	YVPKEFNAETF	0.41	HLA-A*24:02
410	418	NLIKQNCEL	0.41	HLA-B*08:01
178	186	LLFFAKRYK	0.41	HLA-A*30:01
19	28	RGVFRRDAHK	0.41	HLA-A*30:01
509	517	RPCFSALEV	0.41	HLA-B*07:02
39	46	GEENFKAL	0.41	HLA-B*40:01
320	329	DEMPADLPSL	0.41	HLA-B*40:01
242	252	RLSQRFPKAEF	0.41	HLA-B*15:01
244	252	SQRFPKAEF	0.42	HLA-A*32:01
164	173	YEIARRHPYF	0.42	HLA-B*44:02
570	579	AVMDDFAAFV	0.42	HLA-A*68:02
369	377	LLLRLAKTY	0.42	HLA-A*30:02
230	238	FGERAFKAW	0.42	HLA-B*58:01
329	337	LAADFVESK	0.42	HLA-A*11:01
418	427	LFEQLGEYKF	0.43	HLA-A*23:01
424	432	EYKFQNALL	0.43	HLA-A*23:01
342	351	NYAEAKDVFL	0.44	HLA-A*24:02
417	425	ELFEQLGEY	0.44	HLA-A*01:01
427	435	FQNALLVRY	0.44	HLA-A*01:01
320	329	DEMPADLPSL	0.44	HLA-B*44:03
46	54	LVLIAFAQY	0.44	HLA-A*30:02
291	299	NQDSISSKL	0.44	HLA-A*02:06
162	172	YLYEIARRHPY	0.44	HLA-B*15:01
8	17	SLLFLFSSAY	0.44	HLA-B*15:01

66	75	LVNEVTEFAK	0.44	HLA-A*11:01
139	147	LVRPEVDVM	0.44	HLA-B*15:01
352	360	GMFLYEYAR	0.44	HLA-A*31:01
254	264	EVSKLVTDLTK	0.44	HLA-A*68:01
247	255	FPKAEFAEV	0.45	HLA-B*53:01
430	438	ALLVRYTKK	0.45	HLA-A*30:01
29	38	SEVAHRFKDL	0.45	HLA-B*40:01
401	411	FKPLVEEPQNL	0.45	HLA-A*02:03
155	163	EETFLKKYL	0.45	HLA-B*40:01
166	174	IARRHPYFY	0.45	HLA-B*58:01
512	521	FSALEVDETY	0.45	HLA-B*58:01
2	11	KWVTFISLLF	0.46	HLA-A*32:01
375	383	KTYETTLEK	0.46	HLA-A*32:01
386	394	AAADPHECY	0.46	HLA-B*53:01
418	427	LFEQLGEYKF	0.46	HLA-A*24:02
201	209	CLLPKLDEL	0.46	HLA-B*08:01
356	365	YEYARRHPDY	0.46	HLA-B*44:03
9	17	LLFLFSSAY	0.46	HLA-B*35:01
347	356	KDVFLGMFLY	0.46	HLA-A*30:02
322	331	MPADLPSLAA	0.46	HLA-B*07:02
221	229	RLKCASLQK	0.46	HLA-A*11:01
13	23	FSSAYSRGVFR	0.46	HLA-A*31:01
4	13	VTFISLLFLF	0.46	HLA-B*57:01
416	425	CELFEQLGEY	0.47	HLA-A*26:01
322	330	MPADLPSLA	0.47	HLA-B*53:01
356	365	YEYARRHPDY	0.47	HLA-B*44:02
476	484	YLSVVLNQL	0.47	HLA-B*08:01
385	394	CAAADPHECY	0.47	HLA-A*01:01
441	450	QVSTPTLVEV	0.47	HLA-A*02:06
47	55	VLIAFAQYL	0.48	HLA-A*32:01
100	108	TVATLRETY	0.48	HLA-A*32:01
251	259	EFAEVSKLV	0.48	HLA-A*68:02
291	299	NQDSISSKL	0.48	HLA-B*40:01
411	419	LIKQNCELF	0.48	HLA-B*15:01
589	597	ETCFAEEGK	0.48	HLA-A*68:01
63	73	HVKLVNEVTEF	0.49	HLA-A*26:01
163	173	LYEIARRHPYF	0.49	HLA-A*23:01
524	531	KEFNAETF	0.49	HLA-B*44:02
57	66	QCPFEDHVKL	0.49	HLA-B*35:01
139	147	LVRPEVDVM	0.49	HLA-B*35:01
65	74	KLVNEVTEFA	0.49	HLA-A*02:03
425	435	YKFQNALLVRY	0.49	HLA-B*15:01
362	369	HPDYSVVL	0.5	HLA-B*53:01
278	287	ADDRADLAKY	0.5	HLA-A*01:01
164	173	YEIARRHPYF	0.5	HLA-B*44:03
442	450	VSTPTLVEV	0.5	HLA-B*51:01
549	558	KQTALVELVK	0.5	HLA-A*11:01
117	126	KQEPERNECF	0.5	HLA-B*15:01
519	526	ETYVPKEF	0.51	HLA-A*26:01
322	329	MPADLPSL	0.51	HLA-B*08:01
345	353	EAKDVFLGM	0.51	HLA-B*51:01
373	381	LAKTYETTL	0.51	HLA-B*51:01
57	66	QCPFEDHVKL	0.51	HLA-B*07:02
545	553	RQIKKQTAL	0.51	HLA-A*02:03
584	592	KADDKETCF	0.51	HLA-B*58:01
516	524	EVDETYVPK	0.51	HLA-A*11:01
238	246	WAVARLSQR	0.51	HLA-A*68:01
589	598	ETCFAEEGKK	0.51	HLA-A*68:01
100	108	TVATLRETY	0.52	HLA-B*53:01
25	35	DAHKSEVAHRF	0.52	HLA-B*53:01
50	60	AFAQYLQQCPF	0.52	HLA-A*23:01
556	564	LVKHKPKAT	0.52	HLA-B*08:01
242	250	RLSQRFPKA	0.52	HLA-A*30:01
396	404	KVFDEFKPL	0.52	HLA-A*30:01
168	178	RRHPYFYAPEL	0.52	HLA-B*07:02
521	531	YVPKEFNAETF	0.53	HLA-A*23:01

237	247	AWAVARLSQRF	0.53	HLA-A*24:02
469	479	RMPCAEDYLSV	0.53	HLA-A*02:01
97	105	KLCTVATLR	0.53	HLA-A*03:01
88	97	KSLHTLFGDK	0.53	HLA-A*03:01
427	435	FQNALLVRY	0.54	HLA-A*26:01
133	141	NPNLPRLVR	0.54	HLA-A*33:01
242	250	RLSQRFPKA	0.54	HLA-A*02:01
256	265	SKLVTDLTKV	0.54	HLA-A*02:06
88	97	KSLHTLFGDK	0.54	HLA-A*11:01
512	521	FSALEVDETY	0.55	HLA-A*26:01
320	329	DEMPADLPSL	0.55	HLA-B*44:02
286	295	KYICENQDSI	0.55	HLA-A*24:02
584	592	KADDKETCF	0.55	HLA-A*01:01
249	258	KAEFAEVSKL	0.55	HLA-B*44:03
525	533	EFNAETFTF	0.55	HLA-B*35:01
156	164	ETFLKKYLY	0.55	HLA-B*35:01
184	192	RYKAAFTEC	0.55	HLA-A*30:01
592	600	FAEEGKKLV	0.55	HLA-B*51:01
64	73	VKLVNEVTEF	0.55	HLA-B*15:01
239	249	AVARLSQRFPK	0.55	HLA-A*03:01
28	35	KSEVAHRF	0.55	HLA-B*57:01
525	533	EFNAETFTF	0.56	HLA-A*26:01
369	377	LLLRLAKTY	0.56	HLA-A*32:01
172	180	YFYAPELLF	0.56	HLA-B*53:01
523	533	PKEFNAETFTF	0.56	HLA-A*24:02
155	164	EETFLKKYLY	0.56	HLA-A*01:01
362	369	HPDYSVVL	0.56	HLA-B*08:01
174	182	YAPELLFFA	0.56	HLA-A*68:02
233	241	RAFKAWAVA	0.56	HLA-A*30:01
170	180	HPYFYAPELLF	0.56	HLA-B*51:01
239	247	AVARLSQRF	0.56	HLA-B*57:01
290	298	ENQDSISSK	0.56	HLA-A*68:01
525	533	EFNAETFTF	0.57	HLA-B*53:01
570	578	AVMDDFAAF	0.57	HLA-A*23:01
25	33	DAHKSEVAH	0.57	HLA-B*35:01
170	179	HPYFYAPELL	0.57	HLA-B*35:01
156	164	ETFLKKYLY	0.57	HLA-A*33:01
199	209	AACLLPKLDEL	0.57	HLA-A*02:01
563	571	ATKEQLKAV	0.57	HLA-A*02:06
321	329	EMPADLPSL	0.57	HLA-A*02:06
549	558	KQTALVELVK	0.57	HLA-A*03:01
34	44	RFKDLGEENFK	0.57	HLA-A*31:01
501	509	CTESLVNRR	0.57	HLA-A*68:01
429	437	NALLVRYTK	0.57	HLA-A*68:01
417	426	ELFEQLGEYK	0.58	HLA-A*26:01
427	435	FQNALLVRY	0.58	HLA-A*32:01
423	432	GEYKFQNALL	0.58	HLA-B*44:03
416	425	CELFEQLGEY	0.58	HLA-B*44:03
172	180	YFYAPELLF	0.58	HLA-B*35:01
362	371	HPDYSVVLLL	0.58	HLA-B*35:01
427	435	FQNALLVRY	0.58	HLA-B*35:01
446	454	TLVEVSRNL	0.58	HLA-A*68:02
233	242	RAFKAWAVAR	0.58	HLA-A*33:01
96	105	DKLCTVATLR	0.58	HLA-A*33:01
360	370	RRHPDYSVVLL	0.58	HLA-B*07:02
57	66	QCPFEDHVKL	0.58	HLA-B*51:01
171	178	PYFYAPEL	0.58	HLA-B*51:01
469	479	RMPCAEDYLSV	0.58	HLA-A*02:03
230	238	FGERAFKAW	0.58	HLA-B*57:01
181	189	FAKRYKAAF	0.59	HLA-B*35:01
135	144	NLPRLVRPEV	0.59	HLA-B*07:02
65	73	KLVNEVTEF	0.59	HLA-A*02:06
374	384	AKTYETTLEKC	0.59	HLA-A*11:01
160	169	KKYLYEIARR	0.59	HLA-A*31:01
599	607	LVAASQAAL	0.6	HLA-A*68:02
172	180	YFYAPELLF	0.6	HLA-A*30:02

9	17	LLFLFSSAY	0.6	HLA-A*30:02
425	435	YKFQNALLVRY	0.6	HLA-A*30:02
514	524	ALEVDETYVPK	0.6	HLA-A*11:01
63	73	HVKLVNEVTEF	0.6	HLA-B*15:01
166	174	IARRHPYFY	0.6	HLA-B*15:01
156	164	ETFLKKYLY	0.61	HLA-A*32:01
173	181	FYAPELLFF	0.61	HLA-B*53:01
539	547	TLSEKERQI	0.61	HLA-B*08:01
507	515	NRRPCFSAL	0.61	HLA-B*08:01
40	48	EENFKALVL	0.61	HLA-B*08:01
315	322	AEVENDEM	0.61	HLA-B*40:01
321	329	EMPADLPSL	0.61	HLA-A*68:02
170	180	HPYFYAPELLF	0.61	HLA-B*07:02
410	418	NLIKQNCEL	0.61	HLA-A*02:01
430	438	ALLVRYTKK	0.61	HLA-A*11:01
100	108	TVATLRETY	0.61	HLA-A*68:01
415	425	NCELFEQLGEY	0.62	HLA-A*26:01
65	73	KLVNEVTEF	0.62	HLA-A*23:01
152	162	HDNEETFLKKY	0.62	HLA-A*01:01
15	22	SAYSRGVF	0.62	HLA-B*08:01
435	442	YTKKVPQV	0.62	HLA-B*08:01
161	169	KYLYEIARR	0.62	HLA-A*33:01
549	557	KQTALVELV	0.62	HLA-A*02:01
214	222	KASSAKQRL	0.62	HLA-B*58:01
569	578	KAVMDDFAAF	0.62	HLA-B*58:01
404	413	LVEEPQNLIK	0.62	HLA-A*11:01
14	24	SSAYSRGVFRR	0.62	HLA-A*31:01
243	252	LSQRFPKAEF	0.62	HLA-B*57:01
66	75	LVNEVTEFAK	0.62	HLA-A*68:01
30	38	EVAHRFKDL	0.63	HLA-A*26:01
438	447	KVPQVSTPTL	0.63	HLA-A*32:01
237	247	AWAVARLSQRF	0.63	HLA-A*23:01
40	49	EENFKALVLI	0.63	HLA-B*44:02
166	174	IARRHPYFY	0.63	HLA-B*35:01
240	249	VARLSQRFPK	0.63	HLA-A*30:01
162	170	YLYEIARRH	0.63	HLA-A*03:01
52	60	AQYLQQCPF	0.64	HLA-A*32:01
470	479	MPCAEDYLSV	0.64	HLA-B*53:01
560	569	KPKATKEQLK	0.64	HLA-B*07:02
450	460	VSRNLGKVGSK	0.64	HLA-A*30:01
386	394	AAADPHECY	0.64	HLA-B*58:01
402	411	KPLVEEPQNL	0.64	HLA-A*02:06
178	186	LLFFAKRYK	0.64	HLA-A*11:01
47	54	VLIAFAQY	0.64	HLA-B*15:01
249	258	KAEFAEVSKL	0.65	HLA-B*44:02
423	432	GEYKFQNALL	0.65	HLA-B*44:02
153	162	DNEETFLKKY	0.65	HLA-A*01:01
166	173	IARRHPYF	0.65	HLA-B*08:01
429	437	NALLVRYTK	0.65	HLA-A*33:01
397	405	VFDEFKPLV	0.65	HLA-A*02:01
65	74	KLVNEVTEFA	0.65	HLA-A*02:01
413	422	KQNCELFEQL	0.65	HLA-A*02:06
513	521	SALEVDETY	0.65	HLA-B*57:01
391	401	HECYAKVFDEF	0.66	HLA-B*44:02
230	238	FGERAFKAW	0.66	HLA-B*53:01
418	427	LFEQLGEYKF	0.66	HLA-B*44:02
570	578	AVMDDFAAF	0.66	HLA-B*53:01
523	533	PKEFNAETFTF	0.66	HLA-A*23:01
166	174	IARRHPYFY	0.66	HLA-A*01:01
41	49	ENFKALVLI	0.66	HLA-A*68:02
444	452	TPTLVEVSR	0.66	HLA-A*33:01
365	373	YSVVLLLRL	0.66	HLA-B*58:01
56	66	QQCPFEDHVKL	0.66	HLA-A*02:06
242	250	RLSQRFPKA	0.66	HLA-A*02:06
138	147	RLVRPEVDVM	0.66	HLA-B*15:01
539	549	TLSEKERQIKK	0.66	HLA-A*03:01

442	452	VSTPTLVEVSR	0.66	HLA-A*31:01
10	19	LFLFSSAYSR	0.66	HLA-A*31:01
391	401	HECYAKVFDEF	0.67	HLA-B*44:03
27	35	HKSEVAHRF	0.67	HLA-B*44:03
99	108	CTVATLRETY	0.67	HLA-A*01:01
394	401	YAKVFDEF	0.67	HLA-B*35:01
106	114	ETYGEMADC	0.67	HLA-A*68:02
322	330	MPADLPSLA	0.67	HLA-B*07:02
567	575	QLKAVMDDF	0.67	HLA-B*15:01
446	456	TLVEVSRNLGK	0.67	HLA-A*03:01
374	383	AKTYETTLEK	0.67	HLA-A*68:01
173	181	FYAPELLFF	0.68	HLA-A*26:01
516	526	EVDETYVPKEF	0.68	HLA-A*26:01
172	180	YFYAPELLF	0.68	HLA-A*26:01
326	333	LPSLAADF	0.68	HLA-B*53:01
418	427	LFEQLGEYKF	0.68	HLA-B*44:03
143	151	EVDVMCTAF	0.68	HLA-B*35:01
41	48	ENFKALVL	0.68	HLA-B*08:01
85	93	NCDKSLHTL	0.68	HLA-B*08:01
345	353	EAKDVFLGM	0.68	HLA-B*08:01
83	90	AENCDKSL	0.68	HLA-B*40:01
557	565	VKHKPKATK	0.68	HLA-A*30:01
539	547	TLSEKERQI	0.68	HLA-A*02:01
486	493	VLHEKTPV	0.68	HLA-A*02:03
149	158	TAFHDNEETF	0.68	HLA-B*58:01
370	377	LLRLAKTY	0.68	HLA-B*15:01
164	172	YEIARRHPY	0.68	HLA-B*15:01
504	512	SLVNRRPCF	0.68	HLA-B*15:01
46	54	LVLIAFAQY	0.68	HLA-B*15:01
46	54	LVLIAFAQY	0.69	HLA-A*26:01
372	381	RLAKTYETTL	0.69	HLA-A*32:01
348	356	DVFLGMFLY	0.69	HLA-A*01:01
486	493	VLHEKTPV	0.69	HLA-B*08:01
565	572	KEQLKAVM	0.69	HLA-B*40:01
468	476	KRMPCAEDY	0.69	HLA-A*30:02
443	452	STPTLVEVSR	0.69	HLA-A*33:01
387	396	AADPHECYAK	0.69	HLA-A*11:01
348	356	DVFLGMFLY	0.69	HLA-A*68:01
13	23	FSSAYSRGVFR	0.69	HLA-A*68:01
53	60	QYLQQCPF	0.7	HLA-A*23:01
335	343	ESKDVCKNY	0.7	HLA-A*01:01
289	299	CENQDSISSKL	0.7	HLA-B*40:01
423	433	GEYKFQNALLV	0.7	HLA-B*40:01
522	531	VPKEFNAETF	0.7	HLA-B*51:01
4	11	VTFISLLF	0.7	HLA-B*58:01
89	97	SLHTLFGDK	0.7	HLA-A*03:01
99	108	CTVATLRETY	0.71	HLA-A*26:01
27	35	HKSEVAHRF	0.71	HLA-B*44:02
154	163	NEETFLKKYL	0.71	HLA-B*44:02
40	49	EENFKALVLI	0.71	HLA-B*44:03
29	38	SEVAHRFKDL	0.71	HLA-B*44:03
563	571	ATKEQLKAV	0.71	HLA-A*68:02
599	607	LVAASQAAL	0.71	HLA-B*07:02
56	66	QQCPFEDHVKL	0.71	HLA-B*07:02
514	522	ALEVDETYV	0.71	HLA-A*02:01
598	606	KLVAASQAA	0.71	HLA-A*02:03
442	450	VSTPTLVEV	0.71	HLA-B*58:01
449	457	EVSRNLGKV	0.72	HLA-A*26:01
166	174	IARRHPYFY	0.72	HLA-A*32:01
29	38	SEVAHRFKDL	0.72	HLA-B*44:02
364	371	DYSVVLLL	0.72	HLA-A*24:02
4	13	VTFISLLFLF	0.72	HLA-A*24:02
50	60	AFAQYLQQCPF	0.72	HLA-A*24:02
340	350	CKNYAEAKDVF	0.72	HLA-A*24:02
53	60	QYLQQCPF	0.72	HLA-A*24:02
350	358	FLGMFLYEY	0.72	HLA-A*01:01

164	172	YEIARRHPY	0.72	HLA-B*35:01
546	554	QIKKQTALV	0.72	HLA-B*08:01
133	140	NPNLPRLV	0.72	HLA-B*07:02
444	454	TPTLVEVSRNL	0.72	HLA-B*07:02
233	240	RAFKAWAV	0.72	HLA-B*51:01
59	66	PFEDHVKL	0.72	HLA-B*51:01
4	13	VTFISLLFLF	0.72	HLA-B*58:01
397	405	VFDEFKPLV	0.72	HLA-A*02:06
550	558	QTALVELVK	0.72	HLA-A*03:01
14	23	SSAYSRGVFR	0.72	HLA-A*03:01
416	425	CELFEQLGEY	0.73	HLA-B*44:02
396	405	KVFDEFKPLV	0.73	HLA-A*32:01
570	578	AVMDDFAAF	0.73	HLA-A*24:02
13	21	FSSAYSRGV	0.73	HLA-A*68:02
546	554	QIKKQTALV	0.73	HLA-A*02:03
226	236	SLQKFGERAFK	0.73	HLA-A*11:01
103	111	TLRETYGEM	0.73	HLA-B*15:01
239	247	AVARLSQRF	0.74	HLA-A*23:01
244	252	SQRFPKAEF	0.74	HLA-B*07:02
244	252	SQRFPKAEF	0.75	HLA-B*44:02
347	356	KDVFLGMFLY	0.75	HLA-A*32:01
531	540	FTFHADICTL	0.75	HLA-A*68:02
349	358	VFLGMFLYEY	0.75	HLA-A*30:02
244	252	SQRFPKAEF	0.75	HLA-A*30:02
214	223	KASSAKQRLK	0.75	HLA-A*03:01
443	452	STPTLVEVSR	0.75	HLA-A*31:01
83	91	AENCDKSLH	0.76	HLA-B*44:02
143	151	EVDVMCTAF	0.76	HLA-A*26:01
389	398	DPHECYAKVF	0.76	HLA-B*53:01
342	351	NYAEAKDVFL	0.76	HLA-A*23:01
424	431	EYKFQNAL	0.76	HLA-B*08:01
30	38	EVAHRFKDL	0.76	HLA-A*68:02
106	116	ETYGEMADCCA	0.76	HLA-A*68:02
348	356	DVFLGMFLY	0.76	HLA-A*30:02
366	375	SVVLLLRLAK	0.76	HLA-A*11:01
66	75	LVNEVTEFAK	0.76	HLA-A*03:01
88	98	KSLHTLFGDKL	0.76	HLA-B*57:01
156	164	ETFLKKYLY	0.76	HLA-B*57:01
541	549	SEKERQIKK	0.77	HLA-B*44:02
172	179	YFYAPELL	0.77	HLA-A*23:01
65	73	KLVNEVTEF	0.77	HLA-A*24:02
421	431	QLGEYKFQNAL	0.77	HLA-B*44:03
239	247	AVARLSQRF	0.77	HLA-A*24:02
514	521	ALEVDETY	0.77	HLA-A*01:01
385	394	CAAADPHECY	0.77	HLA-B*35:01
56	66	QQCPFEDHVKL	0.77	HLA-B*35:01
442	450	VSTPTLVEV	0.77	HLA-A*02:01
255	265	VSKLVTDLTKV	0.77	HLA-A*02:01
54	64	YLQQCPFEDHV	0.77	HLA-A*02:01
569	579	KAVMDDFAAFV	0.77	HLA-A*02:01
200	209	ACLLPKLDEL	0.77	HLA-A*02:06
66	74	LVNEVTEFA	0.77	HLA-A*02:06
11	19	FLFSSAYSR	0.77	HLA-A*03:01
242	249	RLSQRFPK	0.77	HLA-A*03:01
504	512	SLVNRRPCF	0.78	HLA-A*32:01
154	163	NEETFLKKYL	0.78	HLA-B*44:03
424	431	EYKFQNAL	0.78	HLA-B*40:01
118	126	QEPERNECF	0.78	HLA-B*40:01
472	481	CAEDYLSVVL	0.78	HLA-B*40:01
181	188	FAKRYKAA	0.78	HLA-B*08:01
239	247	AVARLSQRF	0.78	HLA-A*30:02
65	73	KLVNEVTEF	0.78	HLA-B*58:01
35	45	FKDLGEENFKA	0.78	HLA-A*02:01
158	167	FLKKYLYEIA	0.78	HLA-A*02:03
200	209	ACLLPKLDEL	0.78	HLA-A*02:03
345	353	EAKDVFLGM	0.79	HLA-B*53:01

402	411	KPLVEEPQNL	0.79	HLA-B*53:01
559	568	HKPKATKEQL	0.79	HLA-B*08:01
396	405	KVFDEFKPLV	0.79	HLA-A*68:02
509	517	RPCFSALEV	0.79	HLA-B*51:01
212	221	EGKASSAKQR	0.79	HLA-A*33:01
219	227	KQRLKCASL	0.79	HLA-B*15:01
223	233	KCASLQKFGER	0.79	HLA-A*31:01
65	73	KLVNEVTEF	0.8	HLA-A*26:01
389	398	DPHECYAKVF	0.8	HLA-B*35:01
522	530	VPKEFNAET	0.8	HLA-B*07:02
132	141	DNPNLPRLVR	0.8	HLA-A*33:01
362	372	HPDYSVVLLLR	0.8	HLA-A*33:01
225	235	ASLQKFGERAF	0.8	HLA-B*57:01
65	73	KLVNEVTEF	0.81	HLA-A*30:02
162	172	YLYEIARRHPY	0.81	HLA-A*30:02
352	360	GMFLYEYAR	0.81	HLA-A*33:01
177	184	ELLFFAKR	0.81	HLA-A*33:01
321	329	EMPADLPSL	0.81	HLA-A*02:01
353	361	MFLYEYARR	0.81	HLA-A*31:01
237	246	AWAVARLSQR	0.81	HLA-A*31:01
569	578	KAVMDDFAAF	0.82	HLA-A*26:01
172	179	YFYAPELL	0.82	HLA-A*24:02
275	284	LECADDRADL	0.82	HLA-B*40:01
584	592	KADDKETCF	0.82	HLA-B*35:01
220	227	QRLKCASL	0.82	HLA-B*08:01
136	146	LPRLVRPEVDV	0.82	HLA-B*07:02
214	222	KASSAKQRL	0.82	HLA-B*07:02
174	181	YAPELLFF	0.82	HLA-B*51:01
425	434	YKFQNALLVR	0.82	HLA-A*33:01
65	73	KLVNEVTEF	0.82	HLA-A*02:01
4	11	VTFISLLF	0.82	HLA-B*57:01
162	172	YLYEIARRHPY	0.83	HLA-A*26:01
286	295	KYICENQDSI	0.83	HLA-A*23:01
229	238	KFGERAFKAW	0.83	HLA-A*23:01
179	189	LFFAKRYKAAF	0.83	HLA-A*23:01
229	238	KFGERAFKAW	0.83	HLA-A*24:02
56	66	QQCPFEDHVKL	0.83	HLA-B*51:01
100	108	TVATLRETY	0.83	HLA-B*58:01
552	560	ALVELVKHK	0.83	HLA-A*30:01
234	242	AFKAWAVAR	0.83	HLA-A*30:01
45	54	ALVLIAFAQY	0.83	HLA-B*15:01
172	180	YFYAPELLF	0.83	HLA-B*15:01
39	47	GEENFKALV	0.84	HLA-B*44:02
39	47	GEENFKALV	0.84	HLA-B*44:03
391	398	HECYAKVF	0.84	HLA-B*44:03
397	405	VFDEFKPLV	0.84	HLA-A*24:02
232	240	ERAFKAWAV	0.84	HLA-B*08:01
439	448	VPQVSTPTLV	0.84	HLA-B*07:02
570	578	AVMDDFAAF	0.84	HLA-A*30:02
8	16	SLLFLFSSA	0.84	HLA-A*02:03
565	575	KEQLKAVMDDF	0.85	HLA-B*44:02
421	431	QLGEYKFQNAL	0.85	HLA-B*44:02
26	35	AHKSEVAHRF	0.85	HLA-A*23:01
545	553	RQIKKQTAL	0.85	HLA-A*30:01
434	443	RYTKKVPQVS	0.85	HLA-A*30:01
249	257	KAEFAEVSK	0.85	HLA-A*11:01
162	172	YLYEIARRHPY	0.86	HLA-B*44:02
464	472	HPEAKRMPC	0.86	HLA-B*08:01
194	202	QAADKAACL	0.86	HLA-A*68:02
322	330	MPADLPSLA	0.86	HLA-A*68:02
305	314	KPLLEKSHCI	0.86	HLA-B*07:02
233	242	RAFKAWAVAR	0.86	HLA-A*30:01
545	553	RQIKKQTAL	0.86	HLA-A*02:01
255	265	VSKLVTDLTKV	0.86	HLA-A*02:03
28	36	KSEVAHRFK	0.86	HLA-A*11:01
181	189	FAKRYKAAF	0.86	HLA-B*15:01

386	394	AAADPHECY	0.87	HLA-B*44:02
165	174	EIARRHPYFY	0.87	HLA-A*01:01
156	166	ETFLKKYLYEI	0.87	HLA-A*68:02
536	545	DICTLSEKER	0.87	HLA-A*33:01
486	494	VLHEKTPVS	0.87	HLA-A*02:03
571	581	VMDDFAAFVEK	0.87	HLA-A*03:01
375	385	KTYETTLEKCC	0.87	HLA-A*03:01
325	333	DLPSLAADF	0.88	HLA-A*26:01
174	181	YAPELLFF	0.88	HLA-B*53:01
244	252	SQRFPKAEF	0.88	HLA-B*44:03
593	601	AEEGKKLVA	0.88	HLA-B*40:01
473	482	AEDYLSVVLN	0.88	HLA-B*40:01
164	172	YEIARRHPY	0.88	HLA-B*40:01
69	77	EVTEFAKTC	0.88	HLA-A*68:02
4	12	VTFISLLFL	0.88	HLA-B*58:01
244	252	SQRFPKAEF	0.88	HLA-A*30:01
426	434	KFQNALLVR	0.88	HLA-A*30:01
431	439	LLVRYTKKV	0.88	HLA-A*02:01
321	329	EMPADLPSL	0.88	HLA-A*02:03
550	560	QTALVELVKHK	0.88	HLA-A*11:01
255	264	VSKLVTDLTK	0.88	HLA-A*03:01
161	170	KYLYEIARRH	0.88	HLA-A*03:01
4	13	VTFISLLFLF	0.89	HLA-A*32:01
83	91	AENCDKSLH	0.89	HLA-B*44:03
46	54	LVLIAFAQY	0.89	HLA-B*35:01
326	333	LPSLAADF	0.89	HLA-B*35:01
25	35	DAHKSEVAHRF	0.89	HLA-B*35:01
545	553	RQIKKQTAL	0.89	HLA-B*07:02
45	54	ALVLIAFAQY	0.89	HLA-A*30:02
8	17	SLLFLFSSAY	0.89	HLA-A*30:02
366	375	SVVLLLRLAK	0.89	HLA-A*03:01
569	578	KAVMDDFAAF	0.89	HLA-B*57:01
321	329	EMPADLPSL	0.9	HLA-A*26:01
165	172	EIARRHPY	0.9	HLA-A*26:01
4	12	VTFISLLFL	0.9	HLA-A*32:01
394	401	YAKVFDEF	0.9	HLA-B*53:01
411	418	LIKQNCEL	0.9	HLA-B*08:01
470	480	MPCAEDYLSVV	0.9	HLA-B*51:01
363	372	PDYSVVLLLR	0.9	HLA-A*33:01
441	450	QVSTPTLVEV	0.9	HLA-A*02:03
220	229	QRLKCASLQK	0.9	HLA-A*03:01
4	12	VTFISLLFL	0.9	HLA-B*57:01
502	510	TESLVNRRP	0.91	HLA-B*44:02
156	164	ETFLKKYLY	0.91	HLA-B*53:01
364	371	DYSVVLLL	0.91	HLA-A*23:01
173	183	FYAPELLFFAK	0.91	HLA-A*23:01
148	158	CTAFHDNEETF	0.91	HLA-A*24:02
532	540	TFHADICTL	0.91	HLA-A*24:02
571	579	VMDDFAAFV	0.91	HLA-A*01:01
344	354	AEAKDVFLGMF	0.91	HLA-B*40:01
150	158	AFHDNEETF	0.91	HLA-B*35:01
242	250	RLSQRFPKA	0.91	HLA-B*08:01
545	553	RQIKKQTAL	0.91	HLA-A*30:02
374	384	AKTYETTLEKC	0.91	HLA-A*03:01
326	334	LPSLAADFV	0.92	HLA-B*53:01
344	351	AEAKDVFL	0.92	HLA-B*44:03
502	510	TESLVNRRP	0.92	HLA-B*44:03
141	151	RPEVDVMCTAF	0.92	HLA-B*07:02
464	473	HPEAKRMPCA	0.92	HLA-B*07:02
345	355	EAKDVFLGMFL	0.92	HLA-A*68:02
480	490	VLNQLCVLHEK	0.92	HLA-A*03:01
221	229	RLKCASLQK	0.92	HLA-A*31:01
12	22	LFSSAYSRGVF	0.93	HLA-A*24:02
528	537	AETFTFHADI	0.93	HLA-B*40:01
173	181	FYAPELLFF	0.93	HLA-B*35:01
343	351	YAEAKDVFL	0.93	HLA-B*08:01

501	509	CTESLVNRR	0.93	HLA-A*33:01
372	381	RLAKTYETTL	0.93	HLA-A*02:03
365	373	YSVVLLLRL	0.93	HLA-B*57:01
512	521	FSALEVDETY	0.93	HLA-B*57:01
164	172	YEIARRHPY	0.94	HLA-A*26:01
317	326	VENDEMPADL	0.94	HLA-B*44:03
396	404	KVFDEFKPL	0.94	HLA-A*68:02
172	180	YFYAPELLF	0.94	HLA-B*58:01
469	479	RMPCAEDYLSV	0.94	HLA-A*02:06
391	398	HECYAKVF	0.95	HLA-B*44:02
446	454	TLVEVSRNL	0.95	HLA-A*32:01
158	166	FLKKYLYEI	0.95	HLA-A*32:01
279	287	DDRADLAKY	0.95	HLA-B*44:03
162	172	YLYEIARRHPY	0.95	HLA-B*44:03
386	394	AAADPHECY	0.95	HLA-B*44:03
364	373	DYSVVLLLRL	0.95	HLA-A*24:02
26	35	AHKSEVAHRF	0.95	HLA-A*24:02
103	111	TLRETYGEM	0.95	HLA-B*08:01
173	181	FYAPELLFF	0.95	HLA-B*58:01
548	558	KKQTALVELVK	0.95	HLA-A*11:01
443	452	STPTLVEVSR	0.95	HLA-A*11:01
15	24	SAYSRGVFRR	0.95	HLA-A*03:01
512	521	FSALEVDETY	0.95	HLA-B*15:01
593	601	AEEGKKLVA	0.96	HLA-B*44:02
541	549	SEKERQIKK	0.96	HLA-B*44:03
358	367	YARRHPDYSV	0.96	HLA-B*08:01
440	447	PQVSTPTL	0.96	HLA-B*07:02
334	343	VESKDVCKNY	0.96	HLA-A*30:02
350	358	FLGMFLYEY	0.96	HLA-A*30:02
243	252	LSQRFPKAEF	0.96	HLA-B*58:01
442	450	VSTPTLVEV	0.96	HLA-A*02:03
375	384	KTYETTLEKC	0.96	HLA-A*30:01
444	452	TPTLVEVSR	0.96	HLA-A*68:01
317	326	VENDEMPADL	0.97	HLA-B*44:02
39	49	GEENFKALVLI	0.97	HLA-B*40:01
141	151	RPEVDVMCTAF	0.97	HLA-B*35:01
335	343	ESKDVCKNY	0.97	HLA-A*30:02
41	49	ENFKALVLI	0.97	HLA-B*51:01
426	434	KFQNALLVR	0.97	HLA-A*33:01
549	557	KQTALVELV	0.97	HLA-A*02:03
447	456	LVEVSRNLGK	0.97	HLA-A*03:01
178	186	LLFFAKRYK	0.97	HLA-A*31:01
394	402	YAKVFDEFK	0.97	HLA-A*68:01
344	351	AEAKDVFL	0.98	HLA-B*44:02
83	90	AENCDKSL	0.98	HLA-B*44:02
141	151	RPEVDVMCTAF	0.98	HLA-B*53:01
397	405	VFDEFKPLV	0.98	HLA-A*23:01
173	183	FYAPELLFFAK	0.98	HLA-A*24:02
361	369	RHPDYSVVL	0.98	HLA-A*24:02
251	258	EFAEVSKL	0.98	HLA-B*40:01
546	554	QIKKQTALV	0.98	HLA-A*68:02
435	442	YTKKVPQV	0.98	HLA-B*51:01
60	67	FEDHVKLV	0.99	HLA-B*40:01
198	206	KAACLLPKL	0.99	HLA-B*58:01
198	206	KAACLLPKL	0.99	HLA-A*02:06
28	36	KSEVAHRFK	0.99	HLA-A*03:01
539	548	TLSEKERQIK	0.99	HLA-A*03:01
14	22	SSAYSRGVF	0.99	HLA-B*15:01
333	343	FVESKDVCKNY	1.0	HLA-A*26:01
176	185	PELLFFAKRY	1.0	HLA-B*44:03
180	189	FFAKRYKAAF	1.0	HLA-A*24:02
387	395	AADPHECYA	1.0	HLA-A*01:01
511	521	CFSALEVDETY	1.0	HLA-A*01:01
394	401	YAKVFDEF	1.0	HLA-B*51:01
195	205	AADKAACLLPK	1.0	HLA-A*11:01
354	362	FLYEYARRH	1.0	HLA-A*03:01

396	404	KVFDEFKPL	1.0	HLA-B*15:01
229	238	KFGERAFKAW	1.0	HLA-B*57:01

Table X – Results of the Albumin-MHC-II binding prediction.
Acquired with TepiTool (http://tools.iedb.org/tepitool/).

Albumin MHC-II				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
232	246	ERAFKAWAVARLSQR	0.11	HLA-DRB1*09:01
1	15	MKWVTFISLLFLFSS	0.18	HLA-DPA1*01/DPB1*04:01
507	521	NRRPCFSALEVDETY	0.29	HLA-DQA1*05:01/DQB1*02:01
11	25	FLFSSAYSRGVFRRD	0.29	HLA-DRB5*01:01
6	20	FISLLFLFSSAYSRG	0.32	HLA-DRB5*01:01
1	15	MKWVTFISLLFLFSS	0.37	HLA-DPA1*01:03/DPB1*02:01
359	373	ARRHPDYSVVLLLRL	0.39	HLA-DPA1*03:01/DPB1*04:02
364	378	DYSVVLLLRLAKTYE	0.39	HLA-DRB1*11:01
364	378	DYSVVLLLRLAKTYE	0.41	HLA-DRB1*12:01
1	15	MKWVTFISLLFLFSS	0.42	HLA-DPA1*03:01/DPB1*04:02
396	410	KVFDEFKPLVEEPQN	0.63	HLA-DQA1*04:01/DQB1*04:02
170	184	HPYFYAPELLFFAKR	0.70	HLA-DPA1*02:01/DPB1*05:01
1	15	MKWVTFISLLFLFSS	0.84	HLA-DQA1*05:01/DQB1*02:01
170	184	HPYFYAPELLFFAKR	0.87	HLA-DPA1*02:01/DPB1*01:01
175	189	APELLFFAKRYKAAF	0.98	HLA-DPA1*02:01/DPB1*05:01
346	360	AKDVFLGMFLYEYAR	1.05	HLA-DPA1*01/DPB1*04:01
473	487	AEDYLSVVLNQLCVL	1.20	HLA-DPA1*03:01/DPB1*04:02
322	336	MPADLPSLAADFVES	1.20	HLA-DQA1*04:01/DQB1*04:02
6	20	FISLLFLFSSAYSRG	1.30	HLA-DPA1*02:01/DPB1*05:01
1	15	MKWVTFISLLFLFSS	1.30	HLA-DPA1*02:01/DPB1*05:01
364	378	DYSVVLLLRLAKTYE	1.30	HLA-DPA1*03:01/DPB1*04:02
569	583	KAVMDDFAAFVEKCC	1.40	HLA-DQA1*04:01/DQB1*04:02
43	57	FKALVLIAFAQYLQQ	1.50	HLA-DPA1*01/DPB1*04:01
346	360	AKDVFLGMFLYEYAR	1.50	HLA-DPA1*01:03/DPB1*02:01
346	360	AKDVFLGMFLYEYAR	1.50	HLA-DPA1*02:01/DPB1*01:01
309	323	EKSHCIAEVENDEMP	1.50	HLA-DQA1*05:01/DQB1*02:01
1	15	MKWVTFISLLFLFSS	1.50	HLA-DRB1*15:01
543	557	KERQIKKQTALVELV	1.50	HLA-DRB4*01:01
43	57	FKALVLIAFAQYLQQ	1.60	HLA-DPA1*01:03/DPB1*02:01
595	609	EGKKLVAASQAALGL	1.60	HLA-DRB1*09:01
1	15	MKWVTFISLLFLFSS	1.70	HLA-DRB1*04:05
569	583	KAVMDDFAAFVEKCC	1.70	HLA-DRB3*01:01
421	435	QLGEYKFQNALLVRY	1.70	HLA-DRB3*02:02
43	57	FKALVLIAFAQYLQQ	1.80	HLA-DPA1*02:01/DPB1*01:01
569	583	KAVMDDFAAFVEKCC	1.80	HLA-DQA1*03:01/DQB1*03:02
369	383	LLLRLAKTYETTLEK	1.90	HLA-DPA1*02:01/DPB1*05:01
553	567	LVELVKHKPKATKEQ	1.90	HLA-DRB1*08:02
157	171	TFLKKYLYEIARRHP	1.90	HLA-DRB5*01:01
6	20	FISLLFLFSSAYSRG	2.00	HLA-DPA1*01:03/DPB1*02:01
473	487	AEDYLSVVLNQLCVL	2.10	HLA-DPA1*02:01/DPB1*01:01
421	435	QLGEYKFQNALLVRY	2.10	HLA-DRB1*04:01
1	15	MKWVTFISLLFLFSS	2.10	HLA-DRB1*07:01
152	166	HDNEETFLKKYLYEI	2.20	HLA-DPA1*01:03/DPB1*02:01
6	20	FISLLFLFSSAYSRG	2.20	HLA-DRB1*15:01
346	360	AKDVFLGMFLYEYAR	2.30	HLA-DQA1*01:01/DQB1*05:01
170	184	HPYFYAPELLFFAKR	2.30	HLA-DQA1*05:01/DQB1*02:01
421	435	QLGEYKFQNALLVRY	2.30	HLA-DRB1*01:01
11	25	FLFSSAYSRGVFRRD	2.30	HLA-DRB1*07:01
431	445	LLVRYTKKVPQVSTP	2.30	HLA-DRB1*08:02
369	383	LLLRLAKTYETTLEK	2.35	HLA-DRB1*12:01
175	189	APELLFFAKRYKAAF	2.40	HLA-DRB1*11:01
162	176	YLYEIARRHPYFYAP	2.40	HLA-DRB1*11:01
175	189	APELLFFAKRYKAAF	2.40	HLA-DRB1*15:01
170	184	HPYFYAPELLFFAKR	2.50	HLA-DPA1*01:03/DPB1*02:01

421	435	QLGEYKFQNALLVRY	2.50	HLA-DPA1*03:01/DPB1*04:02
6	20	FISLLFLFSSAYSRG	2.70	HLA-DRB1*04:05
43	57	FKALVLIAFAQYLQQ	2.80	HLA-DPA1*02:01/DPB1*05:01
512	526	FSALEVDETYVPKEF	2.80	HLA-DQA1*05:01/DQB1*02:01
255	269	VSKLVTDLTKVHTEC	2.80	HLA-DRB1*03:01
564	578	TKEQLKAVMDDFAAF	3.00	HLA-DRB3*01:01
351	365	LGMFLYEYARRHPDY	3.00	HLA-DRB5*01:01
359	373	ARRHPDYSVVLLLRL	3.10	HLA-DPA1*02:01/DPB1*01:01
421	435	QLGEYKFQNALLVRY	3.10	HLA-DPA1*02:01/DPB1*05:01
1	15	MKWVTFISLLFLFSS	3.30	HLA-DRB1*11:01
6	20	FISLLFLFSSAYSRG	3.35	HLA-DPA1*01/DPB1*04:01
17	31	YSRGVFRRDAHKSEV	3.40	HLA-DRB3*01:01
391	405	HECYAKVFDEFKPLV	3.60	HLA-DPA1*02:01/DPB1*05:01
170	184	HPYFYAPELLFFAKR	3.60	HLA-DPA1*03:01/DPB1*04:02
595	609	EGKKLVAASQAALGL	3.60	HLA-DQA1*01:02/DQB1*06:02
6	20	FISLLFLFSSAYSRG	3.60	HLA-DRB1*04:01
157	171	TFLKKYLYEIARRHP	3.60	HLA-DRB1*11:01
43	57	FKALVLIAFAQYLQQ	3.69	HLA-DRB1*12:01
1	15	MKWVTFISLLFLFSS	3.70	HLA-DPA1*02:01/DPB1*01:01
421	435	QLGEYKFQNALLVRY	3.70	HLA-DRB1*13:02
62	76	DHVKLVNEVTEFAKT	3.80	HLA-DRB1*04:01
247	261	FPKAEFAEVSKLVTD	3.90	HLA-DRB1*11:01
421	435	QLGEYKFQNALLVRY	4.00	HLA-DPA1*02:01/DPB1*01:01
157	171	TFLKKYLYEIARRHP	4.20	HLA-DPA1*02:01/DPB1*05:01
43	57	FKALVLIAFAQYLQQ	4.20	HLA-DRB1*15:01
43	57	FKALVLIAFAQYLQQ	4.50	HLA-DQA1*05:01/DQB1*02:01
431	445	LLVRYTKKVPQVSTP	4.50	HLA-DRB1*11:01
43	57	FKALVLIAFAQYLQQ	4.60	HLA-DRB4*01:01
226	240	SLQKFGERAFKAWAV	4.80	HLA-DPA1*02:01/DPB1*05:01
175	189	APELLFFAKRYKAAF	4.80	HLA-DRB1*08:02
364	378	DYSVVLLLRLAKTYE	4.80	HLA-DRB1*15:01
564	578	TKEQLKAVMDDFAAF	4.90	HLA-DQA1*05:01/DQB1*02:01
1	15	MKWVTFISLLFLFSS	4.90	HLA-DRB1*01:01
530	544	TFTFHADICTLSEKE	4.90	HLA-DRB3*01:01
232	246	ERAFKAWAVARLSQR	4.90	HLA-DRB3*02:02
421	435	QLGEYKFQNALLVRY	5.00	HLA-DRB5*01:01
175	189	APELLFFAKRYKAAF	5.10	HLA-DPA1*01:03/DPB1*02:01
507	521	NRRPCFSALEVDETY	5.10	HLA-DQA1*04:01/DQB1*04:02
351	365	LGMFLYEYARRHPDY	5.10	HLA-DRB1*11:01
553	567	LVELVKHKPKATKEQ	5.20	HLA-DRB5*01:01
232	246	ERAFKAWAVARLSQR	5.30	HLA-DPA1*02:01/DPB1*01:01
396	410	KVFDEFKPLVEEPQN	5.30	HLA-DQA1*03:01/DQB1*03:02
548	562	KKQTALVELVKHKPK	5.30	HLA-DRB1*11:01
478	492	SVVLNQLCVLHEKTP	5.40	HLA-DRB1*04:05
548	562	KKQTALVELVKHKPK	5.40	HLA-DRB5*01:01
364	378	DYSVVLLLRLAKTYE	5.50	HLA-DRB5*01:01
6	20	FISLLFLFSSAYSRG	5.59	HLA-DRB1*12:01
346	360	AKDVFLGMFLYEYAR	5.60	HLA-DPA1*03:01/DPB1*04:02
43	57	FKALVLIAFAQYLQQ	5.60	HLA-DQA1*04:01/DQB1*04:02
346	360	AKDVFLGMFLYEYAR	5.60	HLA-DQA1*05:01/DQB1*02:01
359	373	ARRHPDYSVVLLLRL	5.65	HLA-DPA1*01/DPB1*04:01
232	246	ERAFKAWAVARLSQR	5.70	HLA-DPA1*03:01/DPB1*04:02
421	435	QLGEYKFQNALLVRY	5.70	HLA-DRB3*01:01
391	405	HECYAKVFDEFKPLV	5.80	HLA-DPA1*02:01/DPB1*01:01
11	25	FLFSSAYSRGVFRRD	5.80	HLA-DRB1*01:01
6	20	FISLLFLFSSAYSRG	5.80	HLA-DRB1*11:01
180	194	FFAKRYKAAFTECCQ	5.90	HLA-DPA1*02:01/DPB1*05:01
6	20	FISLLFLFSSAYSRG	5.90	HLA-DRB1*01:01
232	246	ERAFKAWAVARLSQR	5.90	HLA-DRB1*08:02
43	57	FKALVLIAFAQYLQQ	6.00	HLA-DQA1*03:01/DQB1*03:02
6	20	FISLLFLFSSAYSRG	6.00	HLA-DRB1*07:01
162	176	YLYEIARRHPYFYAP	6.00	HLA-DRB5*01:01
426	440	KFQNALLVRYTKKVP	6.10	HLA-DQA1*01:02/DQB1*06:02
226	240	SLQKFGERAFKAWAV	6.10	HLA-DRB1*15:01
48	62	LIAFAQYLQQCPFED	6.20	HLA-DPA1*02:01/DPB1*01:01
87	101	DKSLHTLFGDKLCTV	6.20	HLA-DPA1*03:01/DPB1*04:02

43	57	FKALVLIAFAQYLQQ	6.20	HLA-DPA1*03:01/DPB1*04:02
6	20	FISLLFLFSSAYSRG	6.20	HLA-DRB3*02:02
157	171	TFLKKYLYEIARRHP	6.30	HLA-DPA1*03:01/DPB1*04:02
530	544	TFTFHADICTLSEKE	6.30	HLA-DRB1*04:01
391	405	HECYAKVFDEFKPLV	6.40	HLA-DPA1*01:03/DPB1*02:01
351	365	LGMFLYEYARRHPDY	6.40	HLA-DPA1*02:01/DPB1*05:01
232	246	ERAFKAWAVARLSQR	6.40	HLA-DQA1*01:01/DQB1*05:01
473	487	AEDYLSVVLNQLCVL	6.40	HLA-DRB1*04:05
512	526	FSALEVDETYVPKEF	6.40	HLA-DRB3*01:01
322	336	MPADLPSLAADFVES	6.50	HLA-DQA1*03:01/DQB1*03:02
43	57	FKALVLIAFAQYLQQ	6.50	HLA-DRB1*01:01
6	20	FISLLFLFSSAYSRG	6.60	HLA-DQA1*01:01/DQB1*05:01
1	15	MKWVTFISLLFLFSS	6.60	HLA-DQA1*01:01/DQB1*05:01
564	578	TKEQLKAVMDDFAAF	6.60	HLA-DQA1*04:01/DQB1*04:02
421	435	QLGEYKFQNALLVRY	6.60	HLA-DRB1*15:01
530	544	TFTFHADICTLSEKE	6.80	HLA-DQA1*03:01/DQB1*03:02
595	609	EGKKLVAASQAALGL	6.80	HLA-DRB3*02:02
421	435	QLGEYKFQNALLVRY	7.00	HLA-DPA1*01:03/DPB1*02:01
364	378	DYSVVLLLRLAKTYE	7.10	HLA-DPA1*02:01/DPB1*01:01
595	609	EGKKLVAASQAALGL	7.10	HLA-DRB1*01:01
48	62	LIAFAQYLQQCPFED	7.20	HLA-DPA1*01:03/DPB1*02:01
421	435	QLGEYKFQNALLVRY	7.25	HLA-DPA1*01/DPB1*04:01
351	365	LGMFLYEYARRHPDY	7.30	HLA-DPA1*01:03/DPB1*02:01
152	166	HDNEETFLKKYLYEI	7.30	HLA-DPA1*02:01/DPB1*01:01
157	171	TFLKKYLYEIARRHP	7.30	HLA-DPA1*02:01/DPB1*01:01
170	184	HPYFYAPELLFFAKR	7.40	HLA-DPA1*01/DPB1*04:01
473	487	AEDYLSVVLNQLCVL	7.40	HLA-DPA1*01:03/DPB1*02:01
247	261	FPKAEFAEVSKLVTD	7.40	HLA-DPA1*02:01/DPB1*01:01
1	15	MKWVTFISLLFLFSS	7.40	HLA-DQA1*01:02/DQB1*06:02
70	84	VTEFAKTCVADESAE	7.40	HLA-DQA1*03:01/DQB1*03:02
322	336	MPADLPSLAADFVES	7.40	HLA-DQA1*05:01/DQB1*02:01
170	184	HPYFYAPELLFFAKR	7.40	HLA-DRB3*01:01
346	360	AKDVFLGMFLYEYAR	7.50	HLA-DPA1*02:01/DPB1*05:01
232	246	ERAFKAWAVARLSQR	7.50	HLA-DRB1*07:01
275	289	LECADDRADLAKYIC	7.50	HLA-DRB3*01:01
232	246	ERAFKAWAVARLSQR	7.70	HLA-DRB1*01:01
152	166	HDNEETFLKKYLYEI	7.80	HLA-DPA1*03:01/DPB1*04:02
445	459	PTLVEVSRNLGKVGS	7.80	HLA-DRB1*11:01
11	25	FLFSSAYSRGVFRRD	7.80	HLA-DRB3*02:02
473	487	AEDYLSVVLNQLCVL	7.85	HLA-DPA1*01/DPB1*04:01
364	378	DYSVVLLLRLAKTYE	7.90	HLA-DRB1*08:02
87	101	DKSLHTLFGDKLCTV	8.00	HLA-DPA1*01:03/DPB1*02:01
219	233	KQRLKCASLQKFGER	8.00	HLA-DRB4*01:01
523	537	PKEFNAETFTFHADI	8.10	HLA-DPA1*01:03/DPB1*02:01
1	15	MKWVTFISLLFLFSS	8.10	HLA-DRB1*04:01
157	171	TFLKKYLYEIARRHP	8.20	HLA-DPA1*01:03/DPB1*02:01
369	383	LLLRLAKTYETTLEK	8.20	HLA-DRB1*04:05
70	84	VTEFAKTCVADESAE	8.20	HLA-DRB1*04:05
1	15	MKWVTFISLLFLFSS	8.20	HLA-DRB4*01:01
87	101	DKSLHTLFGDKLCTV	8.30	HLA-DPA1*02:01/DPB1*01:01
162	176	YLYEIARRHPYFYAP	8.35	HLA-DRB1*12:01
6	20	FISLLFLFSSAYSRG	8.40	HLA-DPA1*03:01/DPB1*04:02
11	25	FLFSSAYSRGVFRRD	8.40	HLA-DRB1*04:01
421	435	QLGEYKFQNALLVRY	8.40	HLA-DRB1*11:01
175	189	APELLFFAKRYKAAF	8.40	HLA-DRB1*12:01
564	578	TKEQLKAVMDDFAAF	8.40	HLA-DRB4*01:01
396	410	KVFDEFKPLVEEPQN	8.50	HLA-DPA1*02:01/DPB1*01:01
369	383	LLLRLAKTYETTLEK	8.50	HLA-DPA1*02:01/DPB1*01:01
314	328	IAEVENDEMPADLPS	8.70	HLA-DQA1*05:01/DQB1*02:01
255	269	VSKLVTDLTKVHTEC	8.70	HLA-DRB3*01:01
364	378	DYSVVLLLRLAKTYE	8.80	HLA-DPA1*01/DPB1*04:01
595	609	EGKKLVAASQAALGL	8.90	HLA-DRB1*08:02
364	378	DYSVVLLLRLAKTYE	9.00	HLA-DPA1*02:01/DPB1*05:01
232	246	ERAFKAWAVARLSQR	9.10	HLA-DPA1*02:01/DPB1*05:01
507	521	NRRPCFSALEVDETY	9.10	HLA-DQA1*03:01/DQB1*03:02
553	567	LVELVKHKPKATKEQ	9.10	HLA-DRB1*11:01

483	497	QLCVLHEKTPVSDRV	9.20	HLA-DRB1*04:01
11	25	FLFSSAYSRGVFRRD	9.20	HLA-DRB1*09:01
146	160	VMCTAFHDNEETFLK	9.20	HLA-DRB3*01:01
157	171	TFLKKYLYEIARRHP	9.30	HLA-DRB1*04:01
180	194	FFAKRYKAAFTECCQ	9.30	HLA-DRB1*15:01
351	365	LGMFLYEYARRHPDY	9.30	HLA-DRB1*15:01
247	261	FPKAEFAEVSKLVTD	9.40	HLA-DQA1*04:01/DQB1*04:02
595	609	EGKKLVAASQAALGL	9.40	HLA-DQA1*05:01/DQB1*03:01
11	25	FLFSSAYSRGVFRRD	9.50	HLA-DPA1*02:01/DPB1*05:01
92	106	TLFGDKLCTVATLRE	9.50	HLA-DRB1*03:01
523	537	PKEFNAETFTFHADI	9.60	HLA-DPA1*02:01/DPB1*05:01
281	295	RADLAKYICENQDSI	9.60	HLA-DRB3*01:01
35	49	FKDLGEENFKALVLI	9.70	HLA-DPA1*01:03/DPB1*02:01
152	166	HDNEETFLKKYLYEI	9.70	HLA-DPA1*02:01/DPB1*05:01
70	84	VTEFAKTCVADESAE	9.80	HLA-DQA1*04:01/DQB1*04:02
48	62	LIAFAQYLQQCPFED	9.80	HLA-DQA1*05:01/DQB1*02:01
232	246	ERAFKAWAVARLSQR	9.80	HLA-DRB1*15:01

Table XI – Results of the β2M-MHC-I binding prediction.
Acquired with TepiTool (http://tools.iedb.org/tepitool/).

β2M MHC-I				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
21	30	IQRTPKIQVY	0.01	HLA-B*15:01
105	115	VTLSQPKIVKW	0.01	HLA-B*57:01
107	115	LSQPKIVKW	0.01	HLA-B*57:01
107	115	LSQPKIVKW	0.01	HLA-B*58:01
82	90	FYLLYYTEF	0.03	HLA-A*23:01
106	115	TLSQPKIVKW	0.03	HLA-B*57:01
105	115	VTLSQPKIVKW	0.03	HLA-B*58:01
101	111	RVNHVTLSQPK	0.04	HLA-A*03:01
82	90	FYLLYYTEF	0.04	HLA-A*24:02
74	82	LSFSKDWSF	0.04	HLA-B*57:01
106	114	TLSQPKIVK	0.05	HLA-A*03:01
35	43	AENGKSNFL	0.05	HLA-B*40:01
35	43	AENGKSNFL	0.05	HLA-B*44:02
88	98	TEFTPTEKDEY	0.05	HLA-B*44:03
74	82	LSFSKDWSF	0.05	HLA-B*58:01
107	115	LSQPKIVKW	0.06	HLA-A*32:01
106	115	TLSQPKIVKW	0.06	HLA-B*58:01
23	32	RTPKIQVYSR	0.07	HLA-A*31:01
33	42	HPAENGKSNF	0.07	HLA-B*35:01
74	82	LSFSKDWSF	0.08	HLA-A*32:01
88	98	TEFTPTEKDEY	0.08	HLA-B*44:02
35	43	AENGKSNFL	0.08	HLA-B*44:03
33	43	HPAENGKSNFL	0.09	HLA-B*07:02
90	98	FTPTEKDEY	0.1	HLA-A*01:01
106	114	TLSQPKIVK	0.1	HLA-A*11:01
105	114	VTLSQPKIVK	0.1	HLA-A*11:01
71	80	HSDLSFSKDW	0.1	HLA-B*57:01
68	78	KVEHSDLSFSK	0.11	HLA-A*11:01
33	42	HPAENGKSNF	0.11	HLA-B*53:01
71	80	HSDLSFSKDW	0.11	HLA-B*58:01
33	42	HPAENGKSNF	0.12	HLA-B*07:02
72	80	SDLSFSKDW	0.14	HLA-B*44:02
105	114	VTLSQPKIVK	0.15	HLA-A*03:01
21	30	IQRTPKIQVY	0.15	HLA-A*30:02
101	111	RVNHVTLSQPK	0.16	HLA-A*11:01
106	115	TLSQPKIVKW	0.16	HLA-A*32:01
107	115	LSQPKIVKW	0.16	HLA-B*53:01
75	83	SFSKDWSFY	0.17	HLA-A*30:02
81	90	SFYLLYYTEF	0.18	HLA-A*23:01

35	42	AENGKSNF	0.18	HLA-B*44:02
68	78	KVEHSDLSFSK	0.19	HLA-A*03:01
101	111	RVNHVTLSQPK	0.19	HLA-A*30:01
1	9	MSRSVALAV	0.2	HLA-A*30:01
24	32	TPKIQVYSR	0.2	HLA-A*33:01
19	27	EGIQRTPKI	0.2	HLA-B*51:01
81	90	SFYLLYYTEF	0.21	HLA-A*24:02
72	80	SDLSFSKDW	0.21	HLA-B*44:03
107	116	LSQPKIVKWD	0.21	HLA-B*57:01
108	115	SQPKIVKW	0.22	HLA-B*57:01
74	83	LSFSKDWSFY	0.23	HLA-A*01:01
76	86	FSKDWSFYLLY	0.24	HLA-A*01:01
74	83	LSFSKDWSFY	0.24	HLA-A*30:02
72	82	SDLSFSKDWSF	0.24	HLA-B*57:01
108	115	SQPKIVKW	0.24	HLA-B*58:01
21	29	IQRTPKIQV	0.25	HLA-A*30:01
68	76	KVEHSDLSF	0.25	HLA-A*32:01
105	115	VTLSQPKIVKW	0.25	HLA-A*32:01
35	42	AENGKSNF	0.25	HLA-B*44:03
23	30	RTPKIQVY	0.27	HLA-A*30:02
22	30	QRTPKIQVY	0.27	HLA-A*30:02
76	84	FSKDWSFYL	0.29	HLA-A*68:02
106	114	TLSQPKIVK	0.3	HLA-A*30:01
74	82	LSFSKDWSF	0.3	HLA-B*53:01
72	82	SDLSFSKDWSF	0.3	HLA-B*58:01
90	98	FTPTEKDEY	0.32	HLA-A*26:01
105	114	VTLSQPKIVK	0.32	HLA-A*30:01
78	86	KDWSFYLLY	0.32	HLA-A*30:02
91	98	TPTEKDEY	0.34	HLA-B*35:01
76	84	FSKDWSFYL	0.35	HLA-A*02:06
51	59	HQSDIEVDL	0.35	HLA-A*02:06
19	27	EGIQRTPKI	0.35	HLA-B*08:01
74	82	LSFSKDWSF	0.35	HLA-B*35:01
107	116	LSQPKIVKWD	0.38	HLA-B*58:01
87	95	YTEFTPTEK	0.39	HLA-A*01:01
74	83	LSFSKDWSFY	0.39	HLA-B*57:01
87	95	YTEFTPTEK	0.4	HLA-A*11:01
66	74	IEKVEHSDL	0.4	HLA-B*40:01
74	82	LSFSKDWSF	0.41	HLA-A*23:01
72	82	SDLSFSKDWSF	0.42	HLA-A*32:01
20	30	GIQRTPKIQVY	0.42	HLA-B*15:01
30	39	YSRHPAENGK	0.43	HLA-A*30:01
104	114	HVTLSQPKIVK	0.44	HLA-A*03:01
4	12	SVALAVLAL	0.44	HLA-A*68:02
74	83	LSFSKDWSFY	0.48	HLA-B*58:01
52	60	QSDIEVDLL	0.5	HLA-A*01:01
33	43	HPAENGKSNFL	0.5	HLA-B*40:01
87	95	YTEFTPTEK	0.52	HLA-A*68:01
33	43	HPAENGKSNFL	0.52	HLA-B*53:01
89	98	EFTPTEKDEY	0.54	HLA-A*01:01
101	109	RVNHVTLSQ	0.54	HLA-A*30:01
78	86	KDWSFYLLY	0.54	HLA-B*44:03
33	43	HPAENGKSNFL	0.55	HLA-B*35:01
104	114	HVTLSQPKIVK	0.56	HLA-A*11:01
20	30	GIQRTPKIQVY	0.56	HLA-A*30:02
88	98	TEFTPTEKDEY	0.57	HLA-A*01:01
52	61	QSDIEVDLLK	0.57	HLA-A*01:01
77	86	SKDWSFYLLY	0.57	HLA-A*30:02
107	117	LSQPKIVKWDR	0.59	HLA-A*31:01
5	13	VALAVLALL	0.59	HLA-B*51:01
51	60	HQSDIEVDLL	0.6	HLA-A*02:06
76	86	FSKDWSFYLLY	0.6	HLA-A*30:02
5	12	VALAVLAL	0.61	HLA-B*51:01
59	68	LLKNGERIEK	0.62	HLA-A*03:01
52	61	QSDIEVDLLK	0.62	HLA-A*11:01
75	82	SFSKDWSF	0.62	HLA-A*24:02

4	12	SVALAVLAL	0.62	HLA-A*26:01
78	86	KDWSFYLLY	0.62	HLA-B*44:02
7	15	LAVLALLSL	0.62	HLA-B*51:01
17	26	GLEGIQRTPK	0.63	HLA-A*03:01
23	32	RTPKIQVYSR	0.63	HLA-A*11:01
74	82	LSFSKDWSF	0.63	HLA-B*15:01
51	59	HQSDIEVDL	0.63	HLA-B*40:01
68	76	KVEHSDLSF	0.63	HLA-B*58:01
11	21	ALLSLSGLEGI	0.64	HLA-A*02:01
74	82	LSFSKDWSF	0.65	HLA-A*24:02
4	13	SVALAVLALL	0.65	HLA-A*68:02
1	9	MSRSVALAV	0.65	HLA-B*51:01
23	30	RTPKIQVY	0.66	HLA-B*15:01
90	98	FTPTEKDEY	0.66	HLA-B*35:01
69	76	VEHSDLSF	0.66	HLA-B*44:03
106	116	TLSQPKIVKWD	0.66	HLA-B*57:01
4	12	SVALAVLAL	0.67	HLA-A*32:01
98	105	YACRVNHV	0.68	HLA-B*51:01
4	12	SVALAVLAL	0.69	HLA-A*02:06
99	107	ACRVNHVTL	0.69	HLA-B*08:01
59	69	LLKNGERIEKV	0.7	HLA-A*02:03
75	82	SFSKDWSF	0.7	HLA-A*23:01
23	30	RTPKIQVY	0.7	HLA-B*57:01
56	65	EVDLLKNGER	0.72	HLA-A*68:01
69	76	VEHSDLSF	0.72	HLA-B*44:02
108	115	SQPKIVKW	0.73	HLA-B*44:02
107	117	LSQPKIVKWDR	0.74	HLA-B*57:01
14	24	SLSGLEGIQRT	0.75	HLA-A*02:01
56	65	EVDLLKNGER	0.75	HLA-A*33:01
109	117	QPKIVKWDR	0.75	HLA-A*33:01
68	76	KVEHSDLSF	0.75	HLA-B*15:01
23	30	RTPKIQVY	0.75	HLA-B*58:01
107	115	LSQPKIVKW	0.76	HLA-A*23:01
89	98	EFTPTEKDEY	0.76	HLA-A*26:01
4	12	SVALAVLAL	0.76	HLA-B*07:02
22	30	QRTPKIQVY	0.77	HLA-A*26:01
38	46	GKSNFLNCY	0.77	HLA-A*30:02
33	43	HPAENGKSNFL	0.77	HLA-B*08:01
21	31	IQRTPKIQVYS	0.77	HLA-B*15:01
73	82	DLSFSKDWSF	0.77	HLA-B*53:01
22	30	QRTPKIQVY	0.79	HLA-B*44:02
106	115	TLSQPKIVKW	0.79	HLA-B*53:01
97	107	EYACRVNHVTL	0.81	HLA-A*24:02
76	84	FSKDWSFYL	0.81	HLA-A*32:01
69	76	VEHSDLSF	0.81	HLA-B*40:01
108	115	SQPKIVKW	0.81	HLA-B*44:03
30	39	YSRHPAENGK	0.82	HLA-A*03:01
101	109	RVNHVTLSQ	0.82	HLA-A*03:01
78	86	KDWSFYLLY	0.82	HLA-A*32:01
76	84	FSKDWSFYL	0.82	HLA-B*58:01
78	86	KDWSFYLLY	0.83	HLA-A*01:01
76	83	FSKDWSFY	0.83	HLA-A*01:01
76	84	FSKDWSFYL	0.83	HLA-A*02:01
108	115	SQPKIVKW	0.84	HLA-A*32:01
23	32	RTPKIQVYSR	0.84	HLA-A*68:01
83	90	YLLYYTEF	0.85	HLA-A*23:01
22	30	QRTPKIQVY	0.85	HLA-B*44:03
76	84	FSKDWSFYL	0.85	HLA-B*57:01
39	46	KSNFLNCY	0.86	HLA-A*30:02
78	87	KDWSFYLLYY	0.86	HLA-A*30:02
108	115	SQPKIVKW	0.87	HLA-A*23:01
104	114	HVTLSQPKIVK	0.87	HLA-A*30:01
1	9	MSRSVALAV	0.88	HLA-A*68:02
13	23	LSLSGLEGIQR	0.89	HLA-A*31:01
106	114	TLSQPKIVK	0.89	HLA-A*68:01
104	112	HVTLSQPKI	0.9	HLA-A*68:02

68	76	KVEHSDLSF	0.91	HLA-A*01:01
108	115	SQPKIVKW	0.91	HLA-A*24:02
107	115	LSQPKIVKW	0.91	HLA-B*44:02
5	13	VALAVLALL	0.91	HLA-B*58:01
107	115	LSQPKIVKW	0.92	HLA-A*24:02
35	42	AENGKSNF	0.92	HLA-B*40:01
76	86	FSKDWSFYLLY	0.92	HLA-B*57:01
75	84	SFSKDWSFYL	0.94	HLA-A*24:02
83	90	YLLYYTEF	0.95	HLA-A*24:02
66	76	IEKVEHSDLSF	0.95	HLA-B*44:02
75	85	SFSKDWSFYLL	0.96	HLA-A*24:02
74	84	LSFSKDWSFYL	0.96	HLA-B*57:01
83	91	YLLYYTEFT	0.97	HLA-A*02:01
76	86	FSKDWSFYLLY	0.97	HLA-A*26:01
14	23	SLSGLEGIQR	0.97	HLA-A*31:01
45	55	CYVSGFHQSDI	0.99	HLA-A*24:02
73	82	DLSFSKDWSF	0.99	HLA-A*26:01
99	107	ACRVNHVTL	0.99	HLA-B*07:02
71	80	HSDLSFSKDW	1.0	HLA-A*01:01
75	85	SFSKDWSFYLL	1.0	HLA-A*23:01
75	84	SFSKDWSFYL	1.0	HLA-A*23:01

Table XII – Results of the β2M-MHC-II binding prediction.

Acquired with TepiTool (http://tools.iedb.org/tepitool/).

β2M MHC-II				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
73	87	DLSFSKDWSFYLLYY	0.04	HLA-DRB3*01:01
79	93	DWSFYLLYYTEFTPT	0.60	HLA-DPA1*01/DPB1*04:01
79	93	DWSFYLLYYTEFTPT	0.82	HLA-DPA1*01:03/DPB1*02:01
73	87	DLSFSKDWSFYLLYY	1.40	HLA-DPA1*02:01/DPB1*01:01
79	93	DWSFYLLYYTEFTPT	1.50	HLA-DPA1*02:01/DPB1*01:01
1	15	MSRSVALAVLALLSL	1.50	HLA-DPA1*03:01/DPB1*04:02
1	15	MSRSVALAVLALLSL	1.70	HLA-DPA1*02:01/DPB1*01:01
79	93	DWSFYLLYYTEFTPT	1.70	HLA-DQA1*01:01/DQB1*05:01
73	87	DLSFSKDWSFYLLYY	2.80	HLA-DQA1*01:01/DQB1*05:01
25	39	PKIQVYSRHPAENGK	3.10	HLA-DRB1*15:01
1	15	MSRSVALAVLALLSL	3.30	HLA-DQA1*05:01/DQB1*02:01
1	15	MSRSVALAVLALLSL	3.50	HLA-DQA1*01:02/DQB1*06:02
73	87	DLSFSKDWSFYLLYY	3.60	HLA-DPA1*02:01/DPB1*05:01
79	93	DWSFYLLYYTEFTPT	3.60	HLA-DPA1*03:01/DPB1*04:02
7	21	LAVLALLSLSGLEGI	3.60	HLA-DPA1*03:01/DPB1*04:02
99	113	ACRVNHVTLSQPKIV	3.60	HLA-DRB1*09:01
73	87	DLSFSKDWSFYLLYY	4.10	HLA-DPA1*01:03/DPB1*02:01
73	87	DLSFSKDWSFYLLYY	4.30	HLA-DPA1*01/DPB1*04:01
1	15	MSRSVALAVLALLSL	4.40	HLA-DRB1*01:01
1	15	MSRSVALAVLALLSL	4.45	HLA-DPA1*01/DPB1*04:01
79	93	DWSFYLLYYTEFTPT	4.60	HLA-DRB1*15:01
79	93	DWSFYLLYYTEFTPT	4.70	HLA-DQA1*05:01/DQB1*02:01
79	93	DWSFYLLYYTEFTPT	4.80	HLA-DPA1*02:01/DPB1*05:01
99	113	ACRVNHVTLSQPKIV	4.80	HLA-DRB3*02:02
94	108	EKDEYACRVNHVTLS	4.90	HLA-DRB3*02:02
46	60	YVSGFHQSDIEVDLL	5.00	HLA-DQA1*05:01/DQB1*02:01
84	98	LLYYTEFTPTEKDEY	5.50	HLA-DPA1*02:01/DPB1*01:01
99	113	ACRVNHVTLSQPKIV	5.70	HLA-DRB1*07:01
1	15	MSRSVALAVLALLSL	5.90	HLA-DQA1*05:01/DQB1*03:01
1	15	MSRSVALAVLALLSL	6.10	HLA-DRB1*07:01
1	15	MSRSVALAVLALLSL	6.30	HLA-DQA1*03:01/DQB1*03:02
7	21	LAVLALLSLSGLEGI	6.40	HLA-DPA1*02:01/DPB1*01:01
1	15	MSRSVALAVLALLSL	6.40	HLA-DQA1*04:01/DQB1*04:02
99	113	ACRVNHVTLSQPKIV	6.50	HLA-DRB1*13:02
73	87	DLSFSKDWSFYLLYY	6.50	HLA-DRB1*15:01

84	98	LLYYTEFTPTEKDEY	6.60	HLA-DPA1*01:03/DPB1*02:01
1	15	MSRSVALAVLALLSL	6.70	HLA-DPA1*02:01/DPB1*05:01
84	98	LLYYTEFTPTEKDEY	6.90	HLA-DPA1*01/DPB1*04:01
79	93	DWSFYLLYYTEFTPT	7.00	HLA-DRB1*04:05
73	87	DLSFSKDWSFYLLYY	7.30	HLA-DRB1*07:01
25	39	PKIQVYSRHPAENGK	8.00	HLA-DRB1*08:02
7	21	LAVLALLSLSGLEGI	8.10	HLA-DRB1*15:01
46	60	YVSGFHQSDIEVDLL	9.20	HLA-DQA1*01:01/DQB1*05:01
84	98	LLYYTEFTPTEKDEY	9.20	HLA-DRB1*04:01
7	21	LAVLALLSLSGLEGI	9.45	HLA-DRB1*12:01
79	93	DWSFYLLYYTEFTPT	9.50	HLA-DRB1*04:01

Table XIII – Results of the BlpZ-MHC-I binding prediction.
Acquired with TepiTool (http://tools.iedb.org/tepitool/).

BlpZ MHC-I				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
11	19	KTLDRLTPY	0.01	HLA-A*30:02
26	34	DTIAFNVFV	0.01	HLA-A*68:02
15	23	RLTPYILVL	0.02	HLA-A*32:01
11	19	KTLDRLTPY	0.04	HLA-A*32:01
22	30	VLASDTIAF	0.04	HLA-B*15:01
15	23	RLTPYILVL	0.04	HLA-A*02:01
11	19	KTLDRLTPY	0.05	HLA-A*30:01
53	61	MAIFIGAGY	0.06	HLA-A*26:01
53	61	MAIFIGAGY	0.06	HLA-B*35:01
29	37	AFNVFVLTF	0.07	HLA-A*23:01
33	41	FVLTFVSAV	0.08	HLA-A*02:06
15	23	RLTPYILVL	0.09	HLA-A*02:03
15	23	RLTPYILVL	0.1	HLA-A*02:06
11	19	KTLDRLTPY	0.1	HLA-A*11:01
60	68	GYVVGFWLL	0.11	HLA-A*23:01
12	20	TLDRLTPYI	0.11	HLA-A*02:01
29	37	AFNVFVLTF	0.12	HLA-A*24:02
6	15	FFLDSKTLDR	0.13	HLA-A*33:01
60	68	GYVVGFWLL	0.16	HLA-A*24:02
33	41	FVLTFVSAV	0.17	HLA-A*02:03
11	19	KTLDRLTPY	0.17	HLA-B*15:01
33	41	FVLTFVSAV	0.18	HLA-A*02:01
7	16	FLDSKTLDRL	0.18	HLA-A*02:01
41	49	VVFNFLNSM	0.19	HLA-A*32:01
55	65	IFIGAGYVVGF	0.2	HLA-A*23:01
28	35	IAFNVFVL	0.2	HLA-B*51:01
37	45	FVSAVVFNF	0.21	HLA-A*26:01
11	19	KTLDRLTPY	0.21	HLA-A*03:01
9	19	DSKTLDRLTPY	0.23	HLA-A*26:01
36	45	TFVSAVVFNF	0.23	HLA-A*23:01
11	19	KTLDRLTPY	0.24	HLA-A*26:01
12	20	TLDRLTPYI	0.24	HLA-A*02:06
45	53	FLNSMLALM	0.25	HLA-A*02:03
45	53	FLNSMLALM	0.25	HLA-A*02:01
37	45	FVSAVVFNF	0.26	HLA-A*32:01
11	19	KTLDRLTPY	0.27	HLA-B*57:01
41	49	VVFNFLNSM	0.28	HLA-A*26:01
11	19	KTLDRLTPY	0.28	HLA-A*01:01
35	43	LTFVSAVVF	0.28	HLA-B*58:01
25	34	SDTIAFNVFV	0.29	HLA-A*68:02
22	30	VLASDTIAF	0.3	HLA-A*32:01
35	43	LTFVSAVVF	0.3	HLA-B*57:01
12	19	TLDRLTPY	0.31	HLA-A*01:01
58	66	GAGYVVGFW	0.31	HLA-B*58:01
10	19	SKTLDRLTPY	0.32	HLA-A*26:01

26	35	DTIAFNVFVL	0.32	HLA-A*68:02
33	41	FVLTFVSAV	0.32	HLA-A*68:02
37	45	FVSAVVFNF	0.33	HLA-B*53:01
22	30	VLASDTIAF	0.33	HLA-B*35:01
53	61	MAIFIGAGY	0.35	HLA-A*30:02
11	19	KTLDRLTPY	0.35	HLA-B*58:01
36	45	TFVSAVVFNF	0.36	HLA-A*24:02
55	65	IFIGAGYVVGF	0.37	HLA-A*24:02
22	32	VLASDTIAFNV	0.39	HLA-A*02:01
28	37	IAFNVFVLTF	0.39	HLA-B*57:01
58	66	GAGYVVGFW	0.39	HLA-B*57:01
10	19	SKTLDRLTPY	0.4	HLA-A*30:02
51	59	ALMAIFIGA	0.41	HLA-A*02:01
53	61	MAIFIGAGY	0.41	HLA-B*15:01
51	59	ALMAIFIGA	0.42	HLA-A*02:03
28	37	IAFNVFVLTF	0.42	HLA-B*58:01
57	66	IGAGYVVGFW	0.42	HLA-B*57:01
35	43	LTFVSAVVF	0.43	HLA-A*32:01
35	43	LTFVSAVVF	0.43	HLA-B*15:01
23	30	LASDTIAF	0.44	HLA-B*35:01
37	45	FVSAVVFNF	0.45	HLA-A*23:01
7	16	FLDSKTLDRL	0.45	HLA-A*02:03
41	49	VVFNFLNSM	0.46	HLA-A*02:06
28	37	IAFNVFVLTF	0.48	HLA-A*23:01
37	45	FVSAVVFNF	0.48	HLA-B*35:01
53	62	MAIFIGAGYV	0.48	HLA-A*68:02
51	59	ALMAIFIGA	0.48	HLA-A*02:06
57	66	IGAGYVVGFW	0.48	HLA-B*58:01
26	34	DTIAFNVFV	0.52	HLA-A*26:01
53	61	MAIFIGAGY	0.52	HLA-B*53:01
10	19	SKTLDRLTPY	0.52	HLA-A*01:01
48	56	SMLALMAIF	0.53	HLA-A*32:01
59	68	AGYVVGFWLL	0.54	HLA-A*23:01
5	13	LFFLDSKTL	0.54	HLA-A*23:01
37	45	FVSAVVFNF	0.54	HLA-B*58:01
1	8	MYKHLFFL	0.55	HLA-A*24:02
54	62	AIFIGAGYV	0.55	HLA-A*02:03
21	30	LVLASDTIAF	0.55	HLA-B*15:01
35	43	LTFVSAVVF	0.56	HLA-B*35:01
1	8	MYKHLFFL	0.57	HLA-A*23:01
11	20	KTLDRLTPYI	0.57	HLA-A*02:06
11	21	KTLDRLTPYIL	0.58	HLA-B*57:01
16	24	LTPYILVLA	0.59	HLA-A*68:02
38	45	VSAVVFNF	0.59	HLA-B*58:01
29	37	AFNVFVLTF	0.61	HLA-A*32:01
24	32	ASDTIAFNV	0.61	HLA-A*01:01
12	20	TLDRLTPYI	0.61	HLA-A*02:03
28	37	IAFNVFVLTF	0.62	HLA-B*53:01
47	55	NSMLALMAI	0.63	HLA-B*51:01
11	20	KTLDRLTPYI	0.65	HLA-A*32:01
61	69	YVVGFWLLI	0.65	HLA-A*68:02
22	32	VLASDTIAFNV	0.65	HLA-A*02:03
17	24	TPYILVLA	0.66	HLA-B*51:01
45	53	FLNSMLALM	0.66	HLA-A*02:06
48	56	SMLALMAIF	0.66	HLA-B*15:01
24	33	ASDTIAFNVF	0.67	HLA-A*01:01
27	35	TIAFNVFVL	0.67	HLA-A*68:02
26	34	DTIAFNVFV	0.68	HLA-B*51:01
7	16	FLDSKTLDRL	0.69	HLA-A*02:06
11	20	KTLDRLTPYI	0.69	HLA-B*57:01
24	32	ASDTIAFNV	0.7	HLA-A*02:06
11	21	KTLDRLTPYIL	0.71	HLA-A*32:01
5	13	LFFLDSKTL	0.71	HLA-A*24:02
41	49	VVFNFLNSM	0.72	HLA-A*68:02
34	42	VLTFVSAVV	0.72	HLA-A*02:03
59	68	AGYVVGFWLL	0.73	HLA-A*24:02

15	23	RLTPYILVL	0.73	HLA-B*08:01
15	24	RLTPYILVLA	0.74	HLA-A*02:01
15	23	RLTPYILVL	0.74	HLA-B*15:01
11	19	KTLDRLTPY	0.74	HLA-A*31:01
55	63	IFIGAGYVV	0.76	HLA-A*23:01
38	46	VSAVVFNFL	0.76	HLA-B*58:01
42	50	VFNFLNSML	0.77	HLA-A*24:02
6	15	FFLDSKTLDR	0.77	HLA-A*31:01
11	21	KTLDRLTPYIL	0.78	HLA-B*58:01
37	46	FVSAVVFNFL	0.79	HLA-A*68:02
22	30	VLASDTIAF	0.8	HLA-A*23:01
50	57	LALMAIFI	0.8	HLA-B*51:01
15	23	RLTPYILVL	0.81	HLA-A*23:01
22	30	VLASDTIAF	0.82	HLA-A*24:02
37	45	FVSAVVFNF	0.82	HLA-A*24:02
23	32	LASDTIAFNV	0.82	HLA-A*68:02
15	24	RLTPYILVLA	0.82	HLA-A*02:03
45	52	FLNSMLAL	0.83	HLA-B*08:01
39	46	SAVVFNFL	0.83	HLA-B*51:01
35	43	LTFVSAVVF	0.85	HLA-B*53:01
11	20	KTLDRLTPYI	0.86	HLA-B*58:01
38	45	VSAVVFNF	0.86	HLA-B*57:01
28	37	IAFNVFVLTF	0.87	HLA-A*32:01
41	49	VVFNFLNSM	0.87	HLA-B*35:01
24	33	ASDTIAFNVF	0.87	HLA-B*58:01
20	30	ILVLASDTIAF	0.87	HLA-B*15:01
37	45	FVSAVVFNF	0.87	HLA-B*57:01
28	37	IAFNVFVLTF	0.88	HLA-A*24:02
37	45	FVSAVVFNF	0.88	HLA-A*68:02
61	69	YVVGFWLLI	0.88	HLA-A*02:06
34	43	VLTFVSAVVF	0.88	HLA-B*57:01
42	50	VFNFLNSML	0.89	HLA-A*23:01
38	46	VSAVVFNFL	0.89	HLA-A*68:02
53	61	MAIFIGAGY	0.9	HLA-A*68:01
30	38	FNVFVLTFV	0.91	HLA-A*68:02
55	63	IFIGAGYVV	0.92	HLA-A*24:02
15	23	RLTPYILVL	0.92	HLA-A*24:02
38	46	VSAVVFNFL	0.92	HLA-B*57:01
26	33	DTIAFNVF	0.94	HLA-A*26:01
53	61	MAIFIGAGY	0.94	HLA-A*01:01
47	55	NSMLALMAI	0.94	HLA-A*68:02
6	13	FFLDSKTL	0.95	HLA-B*08:01
5	15	LFFLDSKTLDR	0.96	HLA-A*33:01
12	21	TLDRLTPYIL	0.96	HLA-A*02:01
41	49	VVFNFLNSM	0.96	HLA-B*15:01
10	19	SKTLDRLTPY	0.97	HLA-A*11:01
52	61	LMAIFIGAGY	0.98	HLA-B*15:01
35	45	LTFVSAVVFNF	0.98	HLA-B*57:01
48	56	SMLALMAIF	1.0	HLA-A*23:01

Table XIV – Results of the BlpZ-MHC-II binding prediction.

Acquired with TepiTool (http://tools.iedb.org/tepitool/).

BlpZ MHC-II				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
56	70	FIGAGYVVGFWLLIL	0.36	HLA-DPA1*01/DPB1*04:01
26	40	DTIAFNVFVLTFVSA	0.76	HLA-DPA1*01/DPB1*04:01
32	46	VFVLTFVSAVVFNFL	1.15	HLA-DPA1*01/DPB1*04:01
38	52	VSAVVFNFLNSMLAL	2.10	HLA-DPA1*01/DPB1*04:01
61	75	YVVGFWLLILNENQR	4.10	HLA-DPA1*01/DPB1*04:01
50	64	LALMAIFIGAGYVVG	8.51	HLA-DPA1*01/DPB1*04:01
1	15	MYKHLFFLDSKTLDR	8.95	HLA-DPA1*01/DPB1*04:01

56	70	FIGAGYVVGFWLLIL	0.60	HLA-DPA1*01:03/DPB1*02:01
26	40	DTIAFNVFVLTFVSA	1.70	HLA-DPA1*01:03/DPB1*02:01
32	46	VFVLTFVSAVVFNFL	2.30	HLA-DPA1*01:03/DPB1*02:01
38	52	VSAVVFNFLNSMLAL	3.40	HLA-DPA1*01:03/DPB1*02:01
1	15	MYKHLFFLDSKTLDR	5.80	HLA-DPA1*01:03/DPB1*02:01
61	75	YVVGFWLLILNENQR	6.60	HLA-DPA1*01:03/DPB1*02:01
43	57	FNFLNSMLALMAIFI	8.70	HLA-DPA1*01:03/DPB1*02:01
61	75	YVVGFWLLILNENQR	1.10	HLA-DPA1*02:01/DPB1*01:01
26	40	DTIAFNVFVLTFVSA	1.80	HLA-DPA1*02:01/DPB1*01:01
56	70	FIGAGYVVGFWLLIL	2.70	HLA-DPA1*02:01/DPB1*01:01
32	46	VFVLTFVSAVVFNFL	4.40	HLA-DPA1*02:01/DPB1*01:01
38	52	VSAVVFNFLNSMLAL	4.80	HLA-DPA1*02:01/DPB1*01:01
21	35	LVLASDTIAFNVFVL	7.40	HLA-DPA1*02:01/DPB1*01:01
1	15	MYKHLFFLDSKTLDR	8.40	HLA-DPA1*02:01/DPB1*01:01
38	52	VSAVVFNFLNSMLAL	1.60	HLA-DPA1*02:01/DPB1*05:01
61	75	YVVGFWLLILNENQR	3.80	HLA-DPA1*02:01/DPB1*05:01
1	15	MYKHLFFLDSKTLDR	4.10	HLA-DPA1*02:01/DPB1*05:01
11	25	KTLDRLTPYILVLAS	7.70	HLA-DPA1*02:01/DPB1*05:01
32	46	VFVLTFVSAVVFNFL	8.50	HLA-DPA1*02:01/DPB1*05:01
26	40	DTIAFNVFVLTFVSA	9.00	HLA-DPA1*02:01/DPB1*05:01
56	70	FIGAGYVVGFWLLIL	0.31	HLA-DPA1*03:01/DPB1*04:02
61	75	YVVGFWLLILNENQR	1.20	HLA-DPA1*03:01/DPB1*04:02
32	46	VFVLTFVSAVVFNFL	2.80	HLA-DPA1*03:01/DPB1*04:02
43	57	FNFLNSMLALMAIFI	3.00	HLA-DPA1*03:01/DPB1*04:02
26	40	DTIAFNVFVLTFVSA	3.30	HLA-DPA1*03:01/DPB1*04:02
38	52	VSAVVFNFLNSMLAL	4.40	HLA-DPA1*03:01/DPB1*04:02
11	25	KTLDRLTPYILVLAS	5.20	HLA-DPA1*03:01/DPB1*04:02
1	15	MYKHLFFLDSKTLDR	9.20	HLA-DPA1*03:01/DPB1*04:02
56	70	FIGAGYVVGFWLLIL	0.04	HLA-DQA1*01:01/DQB1*05:01
61	75	YVVGFWLLILNENQR	0.15	HLA-DQA1*01:01/DQB1*05:01
1	15	MYKHLFFLDSKTLDR	1.60	HLA-DQA1*01:01/DQB1*05:01
38	52	VSAVVFNFLNSMLAL	2.50	HLA-DQA1*01:01/DQB1*05:01
43	57	FNFLNSMLALMAIFI	1.80	HLA-DQA1*01:02/DQB1*06:02
50	64	LALMAIFIGAGYVVG	4.50	HLA-DQA1*01:02/DQB1*06:02
61	75	YVVGFWLLILNENQR	3.10	HLA-DQA1*05:01/DQB1*02:01
16	30	LTPYILVLASDTIAF	5.90	HLA-DQA1*05:01/DQB1*02:01
32	46	VFVLTFVSAVVFNFL	6.00	HLA-DQA1*05:01/DQB1*02:01
50	64	LALMAIFIGAGYVVG	1.70	HLA-DQA1*05:01/DQB1*03:01
56	70	FIGAGYVVGFWLLIL	7.30	HLA-DQA1*05:01/DQB1*03:01
43	57	FNFLNSMLALMAIFI	0.24	HLA-DRB1*01:01
38	52	VSAVVFNFLNSMLAL	0.24	HLA-DRB1*01:01
11	25	KTLDRLTPYILVLAS	4.70	HLA-DRB1*01:01
16	30	LTPYILVLASDTIAF	5.10	HLA-DRB1*01:01
32	46	VFVLTFVSAVVFNFL	6.50	HLA-DRB1*01:01
1	15	MYKHLFFLDSKTLDR	1.40	HLA-DRB1*03:01
16	30	LTPYILVLASDTIAF	8.60	HLA-DRB1*03:01
21	35	LVLASDTIAFNVFVL	8.60	HLA-DRB1*03:01
6	20	FFLDSKTLDRLTPYI	9.00	HLA-DRB1*03:01
43	57	FNFLNSMLALMAIFI	1.10	HLA-DRB1*04:01
38	52	VSAVVFNFLNSMLAL	1.10	HLA-DRB1*04:01
32	46	VFVLTFVSAVVFNFL	2.00	HLA-DRB1*04:01
61	75	YVVGFWLLILNENQR	2.10	HLA-DRB1*04:01
21	35	LVLASDTIAFNVFVL	3.40	HLA-DRB1*04:01
16	30	LTPYILVLASDTIAF	5.90	HLA-DRB1*04:01
1	15	MYKHLFFLDSKTLDR	7.00	HLA-DRB1*04:01
43	57	FNFLNSMLALMAIFI	1.70	HLA-DRB1*04:05
61	75	YVVGFWLLILNENQR	1.80	HLA-DRB1*04:05
38	52	VSAVVFNFLNSMLAL	2.00	HLA-DRB1*04:05
32	46	VFVLTFVSAVVFNFL	5.90	HLA-DRB1*04:05
16	30	LTPYILVLASDTIAF	6.00	HLA-DRB1*04:05
1	15	MYKHLFFLDSKTLDR	6.60	HLA-DRB1*04:05
32	46	VFVLTFVSAVVFNFL	2.00	HLA-DRB1*07:01
11	25	KTLDRLTPYILVLAS	6.10	HLA-DRB1*07:01
43	57	FNFLNSMLALMAIFI	8.70	HLA-DRB1*07:01
43	57	FNFLNSMLALMAIFI	0.23	HLA-DRB1*09:01
26	40	DTIAFNVFVLTFVSA	2.90	HLA-DRB1*09:01

32	46	VFVLTFVSAVVFNFL	4.40	HLA-DRB1*09:01
38	52	VSAVVFNFLNSMLAL	2.10	HLA-DRB1*11:01
43	57	FNFLNSMLALMAIFI	4.10	HLA-DRB1*11:01
43	57	FNFLNSMLALMAIFI	1.31	HLA-DRB1*12:01
38	52	VSAVVFNFLNSMLAL	4.95	HLA-DRB1*12:01
50	64	LALMAIFIGAGYVVG	7.05	HLA-DRB1*12:01
32	46	VFVLTFVSAVVFNFL	8.30	HLA-DRB1*12:01
11	25	KTLDRLTPYILVLAS	9.25	HLA-DRB1*12:01
38	52	VSAVVFNFLNSMLAL	4.30	HLA-DRB1*13:02
16	30	LTPYILVLASDTIAF	9.10	HLA-DRB1*13:02
38	52	VSAVVFNFLNSMLAL	2.00	HLA-DRB1*15:01
16	30	LTPYILVLASDTIAF	4.20	HLA-DRB1*15:01
11	25	KTLDRLTPYILVLAS	5.10	HLA-DRB1*15:01
32	46	VFVLTFVSAVVFNFL	5.80	HLA-DRB1*15:01
56	70	FIGAGYVVGFWLLIL	6.30	HLA-DRB1*15:01
1	15	MYKHLFFLDSKTLDR	7.30	HLA-DRB1*15:01
43	57	FNFLNSMLALMAIFI	8.40	HLA-DRB1*15:01
50	64	LALMAIFIGAGYVVG	9.20	HLA-DRB1*15:01
61	75	YVVGFWLLILNENQR	9.60	HLA-DRB1*15:01
16	30	LTPYILVLASDTIAF	7.00	HLA-DRB3*01:01
1	15	MYKHLFFLDSKTLDR	8.10	HLA-DRB3*01:01
32	46	VFVLTFVSAVVFNFL	8.90	HLA-DRB3*01:01
38	52	VSAVVFNFLNSMLAL	3.00	HLA-DRB3*02:02
43	57	FNFLNSMLALMAIFI	4.60	HLA-DRB3*02:02
16	30	LTPYILVLASDTIAF	2.40	HLA-DRB4*01:01
32	46	VFVLTFVSAVVFNFL	7.50	HLA-DRB5*01:01

Table XV – Results of the Ply-MHC-I binding prediction.

Acquired with TepiTool (http://tools.iedb.org/tepitool/).

Ply MHC-I				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
126	134	AVNDLLAKW	0.01	HLA-A*32:01
212	220	DVFQDTVTV	0.01	HLA-A*68:02
56	64	NTSDISVTA	0.01	HLA-A*68:02
327	335	LPISYTTSF	0.01	HLA-B*53:01
69	77	RLYPGALLV	0.01	HLA-A*02:03
91	99	AVDRAPMTY	0.01	HLA-A*01:01
389	397	KEVLTPKAW	0.01	HLA-B*44:02
389	397	KEVLTPKAW	0.01	HLA-B*44:03
327	335	LPISYTTSF	0.01	HLA-B*35:01
451	461	RTISIWGTTLY	0.02	HLA-A*30:02
447	455	LVRKRTISI	0.02	HLA-B*08:01
442	450	KTDLPLVRK	0.02	HLA-A*11:01
81	89	TLLENNPTL	0.02	HLA-A*02:01
69	77	RLYPGALLV	0.02	HLA-A*02:01
91	99	AVDRAPMTY	0.03	HLA-A*30:02
228	236	ISAERPLVY	0.03	HLA-A*30:02
192	200	IVNFKQIYY	0.03	HLA-A*30:02
342	350	ATFQNSTDY	0.03	HLA-A*30:02
349	359	DYVETKVTAYR	0.03	HLA-A*33:01
363	371	LLLDHSGAY	0.03	HLA-B*15:01
234	242	LVYISSVAY	0.03	HLA-B*15:01
271	279	KVAPQTEWK	0.03	HLA-A*11:01
387	397	QGKEVLTPKAW	0.03	HLA-B*44:02
388	397	GKEVLTPKAW	0.03	HLA-B*44:02
350	358	YVETKVTAY	0.03	HLA-A*01:01
388	397	GKEVLTPKAW	0.03	HLA-B*44:03
69	77	RLYPGALLV	0.03	HLA-A*02:06
321	331	TADHPGLPISY	0.03	HLA-A*01:01
196	204	KQIYYTVSV	0.03	HLA-A*02:06
126	134	AVNDLLAKW	0.03	HLA-B*58:01

371	379	YVAQYYITW	0.04	HLA-A*32:01
69	77	RLYPGALLV	0.04	HLA-A*32:01
453	461	ISIWGTTLY	0.04	HLA-A*30:02
39	46	LPDEFVVI	0.04	HLA-B*51:01
206	214	AVKNPGDVF	0.04	HLA-B*15:01
442	450	KTDLPLVRK	0.04	HLA-A*03:01
90	99	LAVDRAPMTY	0.04	HLA-A*01:01
228	236	ISAERPLVY	0.04	HLA-A*01:01
387	397	QGKEVLTPKAW	0.04	HLA-B*44:03
81	89	TLLENNPTL	0.04	HLA-A*02:06
126	134	AVNDLLAKW	0.04	HLA-B*57:01
445	453	LPLVRKRTI	0.05	HLA-B*08:01
222	230	DLKQRGISA	0.05	HLA-B*08:01
327	335	LPISYTTSF	0.05	HLA-B*51:01
69	78	RLYPGALLVV	0.05	HLA-A*02:03
148	156	MQYEKITAH	0.05	HLA-B*15:01
224	232	KQRGISAER	0.05	HLA-A*31:01
442	451	KTDLPLVRKR	0.05	HLA-A*31:01
371	379	YVAQYYITW	0.06	HLA-B*53:01
239	247	SVAYGRQVY	0.06	HLA-A*30:02
367	375	HSGAYVAQY	0.06	HLA-A*30:02
405	412	TAHFTTSI	0.06	HLA-B*51:01
374	382	QYYITWNEL	0.06	HLA-A*24:02
68	77	SRLYPGALLV	0.06	HLA-A*02:03
335	343	FLRDNVVAT	0.06	HLA-A*02:03
239	247	SVAYGRQVY	0.06	HLA-B*15:01
271	279	KVAPQTEWK	0.06	HLA-A*03:01
234	242	LVYISSVAY	0.06	HLA-B*35:01
324	331	HPGLPISY	0.06	HLA-B*35:01
188	196	KQIQIVNFK	0.07	HLA-A*30:01
239	247	SVAYGRQVY	0.07	HLA-A*26:01
71	78	YPGALLVV	0.07	HLA-B*51:01
445	453	LPLVRKRTI	0.07	HLA-B*51:01
251	259	ETTSKSDEV	0.07	HLA-A*68:02
329	337	ISYTTSFLR	0.07	HLA-A*31:01
89	99	LLAVDRAPMTY	0.07	HLA-A*01:01
350	358	YVETKVTAY	0.07	HLA-B*35:01
95	103	APMTYSIDL	0.07	HLA-B*07:02
371	379	YVAQYYITW	0.07	HLA-B*58:01
124	134	RGAVNDLLAKW	0.07	HLA-B*57:01
350	358	YVETKVTAY	0.08	HLA-A*26:01
240	248	VAYGRQVYL	0.08	HLA-B*08:01
45	53	VIERKKRSL	0.08	HLA-B*08:01
374	382	QYYITWNEL	0.08	HLA-A*23:01
404	412	LTAHFTTSI	0.08	HLA-A*68:02
459	468	TLYPQVEDKV	0.08	HLA-A*02:03
62	71	VTATNDSRLY	0.08	HLA-A*01:01
188	196	KQIQIVNFK	0.08	HLA-A*11:01
327	335	LPISYTTSF	0.08	HLA-B*07:02
271	278	KVAPQTEW	0.08	HLA-B*58:01
236	244	YISSVAYGR	0.08	HLA-A*68:01
162	171	KVKFGSDFEK	0.09	HLA-A*30:01
5	13	AVNDFILAM	0.09	HLA-A*26:01
370	379	AYVAQYYITW	0.09	HLA-A*23:01
55	64	TNTSDISVTA	0.09	HLA-A*68:02
105	115	GLASSDSFLQV	0.09	HLA-A*02:03
175	183	SLDIDFNSV	0.09	HLA-A*02:01
228	236	ISAERPLVY	0.09	HLA-B*15:01
458	467	TTLYPQVEDK	0.09	HLA-A*11:01
69	78	RLYPGALLVV	0.09	HLA-A*02:01
105	115	GLASSDSFLQV	0.09	HLA-A*02:01
26	34	ESIENRFIK	0.09	HLA-A*68:01
124	134	RGAVNDLLAKW	0.1	HLA-A*32:01
368	376	SGAYVAQYY	0.1	HLA-A*30:02
140	150	QVNNVPARMQY	0.1	HLA-A*30:02
352	359	ETKVTAYR	0.1	HLA-A*33:01

332	340	TTSFLRDNV	0.1	HLA-A*68:02
265	273	ALIKGVKVA	0.1	HLA-A*02:03
426	435	RECTGLAWEW	0.1	HLA-B*44:02
5	13	AVNDFILAM	0.1	HLA-A*02:06
162	171	KVKFGSDFEK	0.1	HLA-A*11:01
68	77	SRLYPGALLV	0.1	HLA-A*02:01
426	435	RECTGLAWEW	0.1	HLA-B*44:03
162	171	KVKFGSDFEK	0.1	HLA-A*03:01
125	134	GAVNDLLAKW	0.1	HLA-B*58:01
156	164	HSMEQLKVK	0.11	HLA-A*30:01
234	242	LVYISSVAY	0.11	HLA-A*30:02
196	204	KQIYYTVSV	0.11	HLA-A*02:03
370	379	AYVAQYYITW	0.11	HLA-A*24:02
279	287	KQILDNTEV	0.11	HLA-A*02:06
232	240	RPLVYISSV	0.11	HLA-B*07:02
424	433	KIRECTGLAW	0.12	HLA-A*32:01
232	240	RPLVYISSV	0.12	HLA-B*51:01
212	220	DVFQDTVTV	0.12	HLA-B*51:01
81	89	TLLENNPTL	0.12	HLA-A*02:03
175	183	SLDIDFNSV	0.12	HLA-A*02:06
376	384	YITWNELSY	0.12	HLA-B*35:01
364	372	LLDHSGAYV	0.12	HLA-A*02:01
342	350	ATFQNSTDY	0.12	HLA-B*15:01
125	134	GAVNDLLAKW	0.12	HLA-B*57:01
312	320	DLIQEGSRF	0.13	HLA-A*26:01
387	395	QGKEVLTPK	0.13	HLA-A*30:01
236	244	YISSVAYGR	0.13	HLA-A*33:01
240	248	VAYGRQVYL	0.13	HLA-B*51:01
264	272	EALIKGVKV	0.13	HLA-B*51:01
107	115	ASSDSFLQV	0.13	HLA-A*02:06
1	9	MANKAVNDF	0.13	HLA-B*35:01
90	99	LAVDRAPMTY	0.13	HLA-B*35:01
190	199	IQIVNFKQIY	0.13	HLA-B*15:01
196	204	KQIYYTVSV	0.13	HLA-A*02:01
335	344	FLRDNVVATF	0.13	HLA-B*15:01
188	196	KQIQIVNFK	0.13	HLA-A*03:01
150	158	YEKITAHSM	0.13	HLA-B*40:01
228	236	ISAERPLVY	0.13	HLA-B*58:01
124	134	RGAVNDLLAKW	0.13	HLA-B*58:01
216	224	DTVTVEDLK	0.13	HLA-A*68:01
371	379	YVAQYYITW	0.13	HLA-B*57:01
451	460	RTISIWGTTL	0.14	HLA-A*32:01
271	279	KVAPQTEWK	0.14	HLA-A*30:01
222	232	DLKQRGISAER	0.14	HLA-A*33:01
295	303	DPSSGARVV	0.14	HLA-B*51:01
453	461	ISIWGTTLY	0.14	HLA-A*01:01
342	350	ATFQNSTDY	0.14	HLA-A*01:01
228	236	ISAERPLVY	0.14	HLA-B*35:01
273	282	APQTEWKQIL	0.14	HLA-B*07:02
350	358	YVETKVTAY	0.14	HLA-B*15:01
140	148	QVNNVPARM	0.15	HLA-A*26:01
452	461	TISIWGTTLY	0.15	HLA-A*26:01
126	134	AVNDLLAKW	0.15	HLA-B*53:01
390	399	EVLTPKAWDR	0.15	HLA-A*33:01
367	375	HSGAYVAQY	0.15	HLA-A*01:01
115	123	VEDPSNSSV	0.15	HLA-B*40:01
200	208	YTVSVDAVK	0.15	HLA-A*68:01
369	379	GAYVAQYYITW	0.15	HLA-B*57:01
224	232	KQRGISAER	0.16	HLA-A*30:01
5	15	AVNDFILAMNY	0.16	HLA-A*30:02
81	90	TLLENNPTLL	0.16	HLA-A*02:01
459	467	TLYPQVEDK	0.16	HLA-A*03:01
218	226	VTVEDLKQR	0.16	HLA-A*68:01
268	278	KGVKVAPQTEW	0.16	HLA-B*57:01
271	278	KVAPQTEW	0.16	HLA-B*57:01
5	13	AVNDFILAM	0.17	HLA-A*32:01

235	244	VYISSVAYGR	0.17	HLA-A*33:01
363	371	LLLDHSGAY	0.17	HLA-A*30:02
403	412	DLTAHFTTSI	0.17	HLA-A*68:02
186	195	GEKQIQIVNF	0.17	HLA-B*44:02
69	78	RLYPGALLVV	0.17	HLA-A*02:06
156	165	HSMEQLKVKF	0.17	HLA-B*57:01
342	350	ATFQNSTDY	0.18	HLA-A*26:01
212	220	DVFQDTVTV	0.18	HLA-A*26:01
427	435	ECTGLAWEW	0.18	HLA-B*53:01
226	236	RGISAERPLVY	0.18	HLA-A*30:02
192	200	IVNFKQIYY	0.18	HLA-A*01:01
150	158	YEKITAHSM	0.18	HLA-B*44:02
367	376	HSGAYVAQYY	0.18	HLA-A*01:01
438	448	TVYEKTDLPLV	0.18	HLA-A*02:06
63	71	TATNDSRLY	0.18	HLA-B*35:01
363	372	LLLDHSGAYV	0.18	HLA-A*02:01
459	468	TLYPQVEDKV	0.18	HLA-A*02:01
255	263	KSDEVEAAF	0.18	HLA-B*58:01
217	226	TVTVEDLKQR	0.18	HLA-A*68:01
140	150	QVNNVPARMQY	0.19	HLA-A*26:01
191	199	QIVNFKQIY	0.19	HLA-A*26:01
234	242	LVYISSVAY	0.19	HLA-A*26:01
334	344	SFLRDNVVATF	0.19	HLA-A*23:01
186	195	GEKQIQIVNF	0.19	HLA-B*44:03
261	271	AAFEALIKGVK	0.19	HLA-A*11:01
156	164	HSMEQLKVK	0.19	HLA-A*11:01
268	278	KGVKVAPQTEW	0.19	HLA-B*58:01
13	21	MNYDKKKLL	0.2	HLA-B*08:01
31	39	RFIKEGNQL	0.2	HLA-A*23:01
137	147	DYGQVNNVPAR	0.2	HLA-A*33:01
445	453	LPLVRKRTI	0.2	HLA-B*07:02
327	336	LPISYTTSFL	0.2	HLA-B*07:02
148	158	MQYEKITAHSM	0.2	HLA-B*15:01
285	292	TEVKAVIL	0.2	HLA-B*40:01
451	461	RTISIWGTTLY	0.2	HLA-B*57:01
271	278	KVAPQTEW	0.21	HLA-A*32:01
350	358	YVETKVTAY	0.21	HLA-A*30:02
31	39	RFIKEGNQL	0.21	HLA-A*24:02
431	439	LAWEWWRTV	0.21	HLA-B*51:01
410	418	TSIPLKGNV	0.21	HLA-A*68:02
363	372	LLLDHSGAYV	0.21	HLA-A*02:03
150	158	YEKITAHSM	0.21	HLA-B*44:03
74	82	ALLVVDETL	0.21	HLA-A*02:01
329	337	ISYTTSFLR	0.21	HLA-A*11:01
188	195	KQIQIVNF	0.21	HLA-B*15:01
453	461	ISIWGTTLY	0.21	HLA-B*58:01
61	69	SVTATNDSR	0.21	HLA-A*68:01
196	204	KQIYYTVSV	0.22	HLA-A*32:01
62	71	VTATNDSRLY	0.22	HLA-A*30:02
364	371	LLDHSGAY	0.22	HLA-A*01:01
54	62	STNTSDISV	0.22	HLA-A*68:02
105	115	GLASSDSFLQV	0.22	HLA-A*02:06
459	467	TLYPQVEDK	0.22	HLA-A*11:01
453	461	ISIWGTTLY	0.22	HLA-B*15:01
240	249	VAYGRQVYLK	0.22	HLA-A*03:01
451	461	RTISIWGTTLY	0.22	HLA-B*58:01
350	359	YVETKVTAYR	0.22	HLA-A*68:01
216	226	DTVTVEDLKQR	0.22	HLA-A*68:01
269	278	GVKVAPQTEW	0.22	HLA-B*57:01
192	200	IVNFKQIYY	0.23	HLA-A*26:01
103	112	LPGLASSDSF	0.23	HLA-B*53:01
434	442	EWWRTVYEK	0.23	HLA-A*33:01
452	461	TISIWGTTLY	0.23	HLA-A*30:02
451	461	RTISIWGTTLY	0.23	HLA-A*01:01
258	266	EVEAAFEAL	0.23	HLA-A*68:02
34	43	KEGNQLPDEF	0.23	HLA-B*44:02

232	242	RPLVYISSVAY	0.23	HLA-B*35:01
239	247	SVAYGRQVY	0.23	HLA-B*35:01
191	199	QIVNFKQIY	0.23	HLA-B*15:01
369	379	GAYVAQYYITW	0.23	HLA-B*58:01
451	461	RTISIWGTTLY	0.24	HLA-A*32:01
42	51	EFVVIERKKR	0.24	HLA-A*33:01
367	376	HSGAYVAQYY	0.24	HLA-A*30:02
320	329	FTADHPGLPI	0.24	HLA-A*68:02
192	200	IVNFKQIYY	0.24	HLA-B*15:01
156	165	HSMEQLKVKF	0.24	HLA-B*58:01
324	331	HPGLPISY	0.25	HLA-B*53:01
242	250	YGRQVYLKL	0.25	HLA-B*08:01
327	335	LPISYTTSF	0.25	HLA-B*08:01
442	450	KTDLPLVRK	0.25	HLA-A*30:01
227	236	GISAERPLVY	0.25	HLA-A*30:02
334	344	SFLRDNVVATF	0.25	HLA-A*24:02
103	112	LPGLASSDSF	0.25	HLA-B*35:01
453	461	ISIWGTTLY	0.25	HLA-B*35:01
310	320	VEDLIQEGSRF	0.25	HLA-B*44:03
412	421	IPLKGNVRNL	0.25	HLA-B*07:02
297	306	SSGARVVTGK	0.25	HLA-A*11:01
240	249	VAYGRQVYLK	0.25	HLA-A*11:01
245	255	QVYLKLETTSK	0.25	HLA-A*03:01
1	9	MANKAVNDF	0.26	HLA-B*53:01
70	78	LYPGALLVV	0.26	HLA-A*24:02
98	106	TYSIDLPGL	0.26	HLA-A*24:02
158	165	MEQLKVKF	0.26	HLA-B*44:02
259	266	VEAAFEAL	0.26	HLA-B*40:01
130	138	LLAKWHQDY	0.26	HLA-B*15:01
91	99	AVDRAPMTY	0.26	HLA-B*15:01
349	358	DYVETKVTAY	0.27	HLA-A*26:01
126	134	AVNDLLAKW	0.27	HLA-A*26:01
371	379	YVAQYYITW	0.27	HLA-A*23:01
264	272	EALIKGVKV	0.27	HLA-B*08:01
238	247	SSVAYGRQVY	0.27	HLA-A*30:02
191	199	QIVNFKQIY	0.27	HLA-A*30:02
321	331	TADHPGLPISY	0.27	HLA-B*35:01
364	372	LLDHSGAYV	0.27	HLA-A*02:06
37	45	NQLPDEFVV	0.27	HLA-A*02:06
147	156	RMQYEKITAH	0.27	HLA-B*15:01
152	162	KITAHSMEQLK	0.27	HLA-A*03:01
235	244	VYISSVAYGR	0.27	HLA-A*31:01
424	433	KIRECTGLAW	0.27	HLA-B*57:01
228	236	ISAERPLVY	0.27	HLA-B*57:01
390	399	EVLTPKAWDR	0.27	HLA-A*68:01
346	354	NSTDYVETK	0.27	HLA-A*68:01
376	384	YITWNELSY	0.28	HLA-A*26:01
69	77	RLYPGALLV	0.28	HLA-A*30:01
452	461	TISIWGTTLY	0.28	HLA-A*01:01
130	138	LLAKWHQDY	0.28	HLA-A*30:02
255	263	KSDEVEAAF	0.28	HLA-A*01:01
216	226	DTVTVEDLKQR	0.28	HLA-A*33:01
34	43	KEGNQLPDEF	0.28	HLA-B*44:03
83	90	LENNPTLL	0.28	HLA-B*40:01
239	249	SVAYGRQVYLK	0.28	HLA-A*11:01
352	359	ETKVTAYR	0.28	HLA-A*68:01
453	461	ISIWGTTLY	0.29	HLA-A*26:01
192	200	IVNFKQIYY	0.29	HLA-A*32:01
92	99	VDRAPMTY	0.29	HLA-A*01:01
107	115	ASSDSFLQV	0.29	HLA-A*68:02
227	236	GISAERPLVY	0.29	HLA-B*15:01
192	200	IVNFKQIYY	0.29	HLA-A*03:01
147	156	RMQYEKITAH	0.29	HLA-A*03:01
32	39	FIKEGNQL	0.3	HLA-B*08:01
357	365	AYRNGDLLL	0.3	HLA-A*24:02
240	249	VAYGRQVYLK	0.3	HLA-A*30:01

347	355	STDYVETKV	0.3	HLA-A*01:01
310	320	VEDLIQEGSRF	0.3	HLA-B*44:02
227	236	GISAERPLVY	0.3	HLA-A*01:01
417	426	NVRNLSVKIR	0.3	HLA-A*33:01
175	183	SLDIDFNSV	0.3	HLA-A*02:03
158	165	MEQLKVKF	0.3	HLA-B*44:03
68	77	SRLYPGALLV	0.3	HLA-A*02:06
261	270	AAFEALIKGV	0.3	HLA-A*02:06
169	176	FEKTGNSL	0.3	HLA-B*40:01
192	200	IVNFKQIYY	0.3	HLA-A*11:01
447	456	LVRKRTISIW	0.3	HLA-B*57:01
410	419	TSIPLKGNVR	0.3	HLA-A*68:01
329	337	ISYTTSFLR	0.3	HLA-A*68:01
67	75	DSRLYPGAL	0.31	HLA-B*08:01
376	384	YITWNELSY	0.31	HLA-A*01:01
63	71	TATNDSRLY	0.31	HLA-A*01:01
232	242	RPLVYISSVAY	0.31	HLA-A*30:02
347	355	STDYVETKV	0.31	HLA-A*68:02
329	337	ISYTTSFLR	0.31	HLA-A*33:01
461	468	YPQVEDKV	0.31	HLA-B*51:01
122	130	SVRGAVNDL	0.31	HLA-B*07:02
38	46	QLPDEFVVI	0.31	HLA-A*02:06
269	278	GVKVAPQTEW	0.31	HLA-B*58:01
357	365	AYRNGDLLL	0.32	HLA-A*23:01
14	21	NYDKKKLL	0.32	HLA-B*08:01
64	71	ATNDSRLY	0.32	HLA-A*01:01
153	161	ITAHSMEQL	0.32	HLA-A*68:02
97	106	MTYSIDLPGL	0.32	HLA-A*68:02
440	448	YEKTDLPLV	0.32	HLA-B*40:01
326	335	GLPISYTTSF	0.32	HLA-B*15:01
426	435	RECTGLAWEW	0.32	HLA-B*57:01
367	375	HSGAYVAQY	0.33	HLA-A*26:01
13	20	MNYDKKKL	0.33	HLA-B*08:01
375	382	YYITWNEL	0.33	HLA-A*24:02
426	433	RECTGLAW	0.33	HLA-B*44:02
409	418	TTSIPLKGNV	0.33	HLA-A*68:02
54	64	STNTSDISVTA	0.33	HLA-A*68:02
64	71	ATNDSRLY	0.33	HLA-A*30:02
438	448	TVYEKTDLPLV	0.33	HLA-A*68:02
141	150	VNNVPARMQY	0.33	HLA-A*30:02
91	99	AVDRAPMTY	0.33	HLA-B*35:01
335	343	FLRDNVVAT	0.33	HLA-A*02:01
451	461	RTISIWGTTLY	0.33	HLA-B*15:01
232	242	RPLVYISSVAY	0.33	HLA-B*15:01
125	133	GAVNDLLAK	0.33	HLA-A*11:01
426	435	RECTGLAWEW	0.33	HLA-B*58:01
84	92	ENNPTLLAV	0.34	HLA-A*68:02
426	433	RECTGLAW	0.34	HLA-B*44:03
363	371	LLLDHSGAY	0.34	HLA-B*35:01
5	13	AVNDFILAM	0.34	HLA-A*02:03
298	306	SGARVVTGK	0.34	HLA-A*11:01
458	467	TTLYPQVEDK	0.34	HLA-A*68:01
269	278	GVKVAPQTEW	0.35	HLA-A*32:01
375	382	YYITWNEL	0.35	HLA-A*23:01
98	106	TYSIDLPGL	0.35	HLA-A*23:01
444	451	DLPLVRKR	0.35	HLA-A*33:01
341	350	VATFQNSTDY	0.35	HLA-A*30:02
94	103	RAPMTYSIDL	0.35	HLA-B*07:02
364	372	LLDHSGAYV	0.35	HLA-A*02:03
273	281	APQTEWKQI	0.35	HLA-B*07:02
454	464	SIWGTTLYPQV	0.35	HLA-A*02:01
259	267	VEAAFEALI	0.35	HLA-B*40:01
153	162	ITAHSMEQLK	0.35	HLA-A*11:01
458	467	TTLYPQVEDK	0.35	HLA-A*03:01
247	255	YLKLETTSK	0.35	HLA-A*03:01
453	461	ISIWGTTLY	0.35	HLA-B*57:01

5	15	AVNDFILAMNY	0.36	HLA-A*26:01
240	248	VAYGRQVYL	0.36	HLA-A*32:01
70	78	LYPGALLVV	0.36	HLA-A*23:01
460	468	LYPQVEDKV	0.36	HLA-A*24:02
220	228	VEDLKQRGI	0.36	HLA-B*44:02
90	99	LAVDRAPMTY	0.36	HLA-A*30:02
61	71	SVTATNDSRLY	0.36	HLA-A*30:02
38	46	QLPDEFVVI	0.36	HLA-A*02:01
342	350	ATFQNSTDY	0.36	HLA-A*11:01
427	435	ECTGLAWEW	0.36	HLA-B*58:01
142	150	NNVPARMQY	0.37	HLA-A*26:01
404	412	LTAHFTTSI	0.37	HLA-A*32:01
362	371	DLLLDHSGAY	0.37	HLA-A*26:01
150	158	YEKITAHSM	0.37	HLA-B*08:01
340	350	VVATFQNSTDY	0.37	HLA-A*30:02
350	359	YVETKVTAYR	0.37	HLA-A*33:01
390	397	EVLTPKAW	0.37	HLA-B*44:03
5	13	AVNDFILAM	0.37	HLA-B*35:01
326	335	GLPISYTTSF	0.37	HLA-B*07:02
238	247	SSVAYGRQVY	0.37	HLA-B*15:01
188	196	KQIQIVNFK	0.37	HLA-A*31:01
260	268	EAAFEALIK	0.37	HLA-A*68:01
157	165	SMEQLKVKF	0.38	HLA-A*32:01
91	99	AVDRAPMTY	0.38	HLA-A*32:01
370	379	AYVAQYYITW	0.38	HLA-A*32:01
239	248	SVAYGRQVYL	0.38	HLA-A*68:02
80	89	ETLLENNPTL	0.38	HLA-A*68:02
447	455	LVRKRTISI	0.38	HLA-A*30:01
174	183	NSLDIDFNSV	0.38	HLA-A*68:02
260	267	EAAFEALI	0.38	HLA-B*51:01
5	13	AVNDFILAM	0.38	HLA-B*15:01
371	379	YVAQYYITW	0.39	HLA-A*26:01
390	397	EVLTPKAW	0.39	HLA-B*44:02
322	331	ADHPGLPISY	0.39	HLA-B*44:02
348	358	TDYVETKVTAY	0.39	HLA-A*01:01
56	65	NTSDISVTAT	0.39	HLA-A*68:02
151	161	EKITAHSMEQL	0.39	HLA-A*68:02
191	200	QIVNFKQIYY	0.39	HLA-A*30:02
63	71	TATNDSRLY	0.39	HLA-A*30:02
447	455	LVRKRTISI	0.39	HLA-B*07:02
5	13	AVNDFILAM	0.39	HLA-A*02:01
281	289	ILDNTEVKA	0.39	HLA-A*02:01
384	392	YDHQGKEVL	0.39	HLA-B*40:01
240	248	VAYGRQVYL	0.39	HLA-A*02:06
372	379	VAQYYITW	0.39	HLA-B*58:01
191	200	QIVNFKQIYY	0.4	HLA-A*26:01
25	33	GESIENRFI	0.4	HLA-B*44:02
438	447	TVYEKTDLPL	0.4	HLA-A*68:02
322	331	ADHPGLPISY	0.4	HLA-B*44:03
342	351	ATFQNSTDYV	0.4	HLA-A*68:02
386	395	HQGKEVLTPK	0.4	HLA-A*30:01
376	384	YITWNELSY	0.4	HLA-A*30:02
281	290	ILDNTEVKAV	0.4	HLA-A*02:01
155	164	AHSMEQLKVK	0.4	HLA-A*11:01
90	99	LAVDRAPMTY	0.4	HLA-B*15:01
367	376	HSGAYVAQYY	0.41	HLA-A*26:01
228	236	ISAERPLVY	0.41	HLA-A*26:01
228	236	ISAERPLVY	0.41	HLA-A*32:01
375	384	YYITWNELSY	0.41	HLA-A*23:01
241	250	AYGRQVYLKL	0.41	HLA-A*24:02
46	53	IERKKRSL	0.41	HLA-B*08:01
326	335	GLPISYTTSF	0.41	HLA-B*35:01
445	455	LPLVRKRTISI	0.41	HLA-B*07:02
23	32	HQGESIENRF	0.41	HLA-B*15:01
91	99	AVDRAPMTY	0.42	HLA-A*26:01
69	78	RLYPGALLVV	0.42	HLA-A*32:01

229	236	SAERPLVY	0.42	HLA-A*01:01
140	150	QVNNVPARMQY	0.42	HLA-A*01:01
351	358	VETKVTAY	0.42	HLA-B*44:03
258	267	EVEAAFEALI	0.42	HLA-A*68:02
375	384	YYITWNELSY	0.42	HLA-A*30:02
190	200	IQIVNFKQIYY	0.42	HLA-A*30:02
25	33	GESIENRFI	0.42	HLA-B*40:01
442	451	KTDLPLVRKR	0.42	HLA-A*11:01
91	99	AVDRAPMTY	0.42	HLA-A*11:01
280	288	QILDNTEVK	0.42	HLA-A*11:01
226	236	RGISAERPLVY	0.43	HLA-A*01:01
327	336	LPISYTTSFL	0.43	HLA-B*51:01
105	113	GLASSDSFL	0.43	HLA-A*02:03
38	46	QLPDEFVVI	0.43	HLA-A*02:03
459	468	TLYPQVEDKV	0.43	HLA-A*02:06
245	255	QVYLKLETTSK	0.43	HLA-A*11:01
236	244	YISSVAYGR	0.43	HLA-A*31:01
241	250	AYGRQVYLKL	0.44	HLA-A*23:01
389	398	KEVLTPKAWD	0.44	HLA-B*44:02
363	371	LLLDHSGAY	0.44	HLA-A*01:01
438	448	TVYEKTDLPLV	0.44	HLA-A*02:01
140	150	QVNNVPARMQY	0.44	HLA-B*15:01
371	379	YVAQYYITW	0.45	HLA-A*24:02
438	445	TVYEKTDL	0.45	HLA-B*08:01
25	33	GESIENRFI	0.45	HLA-B*44:03
240	248	VAYGRQVYL	0.45	HLA-A*68:02
440	447	YEKTDLPL	0.45	HLA-B*40:01
451	461	RTISIWGTTLY	0.45	HLA-A*11:01
361	371	GDLLLDHSGAY	0.45	HLA-B*15:01
311	320	EDLIQEGSRF	0.46	HLA-A*26:01
255	263	KSDEVEAAF	0.46	HLA-A*32:01
327	336	LPISYTTSFL	0.46	HLA-B*53:01
126	134	AVNDLLAKW	0.46	HLA-B*44:02
368	376	SGAYVAQYY	0.46	HLA-A*01:01
239	247	SVAYGRQVY	0.46	HLA-A*01:01
349	358	DYVETKVTAY	0.46	HLA-A*01:01
389	398	KEVLTPKAWD	0.46	HLA-B*44:03
5	13	AVNDFILAM	0.46	HLA-A*68:02
209	218	NPGDVFQDTV	0.46	HLA-B*51:01
220	228	VEDLKQRGI	0.46	HLA-B*40:01
186	195	GEKQIQIVNF	0.46	HLA-B*40:01
367	375	HSGAYVAQY	0.46	HLA-B*58:01
211	220	GDVFQDTVTV	0.46	HLA-A*02:06
297	306	SSGARVVTGK	0.46	HLA-A*03:01
194	202	NFKQIYYTV	0.47	HLA-B*08:01
232	240	RPLVYISSV	0.47	HLA-B*08:01
238	247	SSVAYGRQVY	0.47	HLA-A*01:01
25	32	GESIENRF	0.47	HLA-B*44:03
234	242	LVYISSVAY	0.48	HLA-A*32:01
351	358	VETKVTAY	0.48	HLA-B*44:02
179	188	DFNSVHSGEK	0.48	HLA-A*33:01
457	467	GTTLYPQVEDK	0.48	HLA-A*11:01
363	371	LLLDHSGAY	0.49	HLA-A*26:01
81	90	TLLENNPTLL	0.49	HLA-A*02:06
156	164	HSMEQLKVK	0.49	HLA-A*03:01
329	337	ISYTTSFLR	0.49	HLA-A*03:01
409	419	TTSIPLKGNVR	0.49	HLA-A*68:01
90	99	LAVDRAPMTY	0.5	HLA-A*26:01
81	89	TLLENNPTL	0.5	HLA-A*32:01
205	214	DAVKNPGDVF	0.5	HLA-B*53:01
341	350	VATFQNSTDY	0.5	HLA-A*01:01
44	53	VVIERKKRSL	0.5	HLA-B*08:01
407	415	HFTTSIPLK	0.5	HLA-A*30:01
148	156	MQYEKITAH	0.5	HLA-A*30:02
105	113	GLASSDSFL	0.5	HLA-A*02:01
190	200	IQIVNFKQIYY	0.5	HLA-B*15:01

157	165	SMEQLKVKF	0.5	HLA-B*15:01
255	263	KSDEVEAAF	0.5	HLA-B*57:01
63	71	TATNDSRLY	0.51	HLA-B*53:01
130	138	LLAKWHQDY	0.51	HLA-A*01:01
367	375	HSGAYVAQY	0.51	HLA-B*35:01
69	77	RLYPGALLV	0.51	HLA-A*30:02
107	115	ASSDSFLQV	0.51	HLA-A*02:03
82	90	LLENNPTLL	0.51	HLA-A*02:01
454	464	SIWGTTLYPQV	0.51	HLA-A*02:06
376	384	YITWNELSY	0.51	HLA-B*15:01
69	77	RLYPGALLV	0.51	HLA-A*03:01
168	176	DFEKTGNSL	0.52	HLA-B*08:01
368	376	SGAYVAQYY	0.52	HLA-B*35:01
192	200	IVNFKQIYY	0.52	HLA-B*35:01
327	336	LPISYTTSFL	0.52	HLA-B*35:01
140	148	QVNNVPARM	0.52	HLA-A*68:02
67	77	DSRLYPGALLV	0.52	HLA-A*68:02
265	273	ALIKGVKVA	0.52	HLA-A*02:01
148	158	MQYEKITAHSM	0.52	HLA-A*02:06
74	82	ALLVVDETL	0.52	HLA-A*02:06
347	355	STDYVETKV	0.52	HLA-A*02:06
146	156	ARMQYEKITAH	0.52	HLA-B*15:01
370	379	AYVAQYYITW	0.52	HLA-B*57:01
327	335	LPISYTTSF	0.53	HLA-A*26:01
238	247	SSVAYGRQVY	0.53	HLA-A*26:01
125	134	GAVNDLLAKW	0.53	HLA-A*32:01
91	100	AVDRAPMTYS	0.53	HLA-A*01:01
205	214	DAVKNPGDVF	0.53	HLA-B*35:01
424	432	KIRECTGLA	0.53	HLA-A*30:01
322	331	ADHPGLPISY	0.53	HLA-A*30:02
5	13	AVNDFILAM	0.53	HLA-A*30:02
135	144	HQDYGQVNNV	0.53	HLA-A*02:03
438	448	TVYEKTDLPLV	0.53	HLA-A*02:03
1	9	MANKAVNDF	0.53	HLA-B*58:01
226	236	RGISAERPLVY	0.53	HLA-B*58:01
372	379	VAQYYITW	0.53	HLA-B*57:01
63	71	TATNDSRLY	0.54	HLA-A*26:01
321	331	TADHPGLPISY	0.54	HLA-A*26:01
375	384	YYITWNELSY	0.54	HLA-A*24:02
220	228	VEDLKQRGI	0.54	HLA-B*44:03
148	158	MQYEKITAHSM	0.54	HLA-B*08:01
264	272	EALIKGVKV	0.54	HLA-A*68:02
454	461	SIWGTTLY	0.54	HLA-A*30:02
90	99	LAVDRAPMTY	0.54	HLA-B*58:01
91	99	AVDRAPMTY	0.54	HLA-B*58:01
89	99	LLAVDRAPMTY	0.54	HLA-B*15:01
246	255	VYLKLETTSK	0.54	HLA-A*03:01
35	43	EGNQLPDEF	0.55	HLA-B*53:01
326	335	GLPISYTTSF	0.55	HLA-B*53:01
149	158	QYEKITAHSM	0.55	HLA-B*44:02
342	350	ATFQNSTDY	0.55	HLA-B*35:01
240	247	VAYGRQVY	0.55	HLA-B*35:01
298	306	SGARVVTGK	0.55	HLA-A*30:01
424	433	KIRECTGLAW	0.55	HLA-B*58:01
265	273	ALIKGVKVA	0.55	HLA-A*02:06
451	460	RTISIWGTTL	0.55	HLA-B*57:01
451	461	RTISIWGTTLY	0.56	HLA-A*26:01
206	214	AVKNPGDVF	0.56	HLA-A*26:01
228	236	ISAERPLVY	0.56	HLA-B*53:01
194	202	NFKQIYYTV	0.56	HLA-A*23:01
194	202	NFKQIYYTV	0.56	HLA-A*24:02
81	89	TLLENNPTL	0.56	HLA-B*08:01
99	107	YSIDLPGLA	0.56	HLA-A*68:02
295	302	DPSSGARV	0.56	HLA-B*51:01
432	440	AWEWWRTVY	0.56	HLA-A*30:02
279	288	KQILDNTEVK	0.56	HLA-A*11:01

391	399	VLTPKAWDR	0.56	HLA-A*31:01
205	214	DAVKNPGDVF	0.57	HLA-A*26:01
90	99	LAVDRAPMTY	0.57	HLA-B*53:01
460	468	LYPQVEDKV	0.57	HLA-A*23:01
25	32	GESIENRF	0.57	HLA-B*44:02
6	15	VNDFILAMNY	0.57	HLA-A*01:01
61	71	SVTATNDSRLY	0.57	HLA-A*01:01
191	199	QIVNFKQIY	0.57	HLA-B*35:01
374	384	QYYITWNELSY	0.57	HLA-A*30:02
321	331	TADHPGLPISY	0.57	HLA-A*30:02
190	199	IQIVNFKQIY	0.57	HLA-A*30:02
363	372	LLLDHSGAYV	0.57	HLA-A*02:06
153	162	ITAHSMEQLK	0.57	HLA-A*03:01
387	397	QGKEVLTPKAW	0.57	HLA-B*57:01
154	162	TAHSMEQLK	0.57	HLA-A*68:01
451	460	RTISIWGTTL	0.58	HLA-B*58:01
135	144	HQDYGQVNNV	0.58	HLA-A*02:06
367	375	HSGAYVAQY	0.58	HLA-B*15:01
427	435	ECTGLAWEW	0.58	HLA-B*57:01
114	124	QVEDPSNSSVR	0.58	HLA-A*68:01
157	165	SMEQLKVKF	0.59	HLA-A*23:01
5	15	AVNDFILAMNY	0.59	HLA-A*01:01
167	176	SDFEKTGNSL	0.59	HLA-B*40:01
34	43	KEGNQLPDEF	0.59	HLA-B*40:01
84	94	ENNPTLLAVDR	0.59	HLA-A*33:01
391	399	VLTPKAWDR	0.59	HLA-A*33:01
270	278	VKVAPQTEW	0.59	HLA-B*58:01
81	91	TLLENNPTLLA	0.59	HLA-A*02:01
214	223	FQDTVTVEDL	0.59	HLA-A*02:06
261	271	AAFEALIKGVK	0.59	HLA-A*03:01
206	214	AVKNPGDVF	0.6	HLA-A*32:01
453	461	ISIWGTTLY	0.6	HLA-A*32:01
4	13	KAVNDFILAM	0.6	HLA-A*32:01
335	343	FLRDNVVAT	0.6	HLA-A*02:06
270	279	VKVAPQTEWK	0.6	HLA-A*11:01
156	164	HSMEQLKVK	0.6	HLA-A*68:01
336	344	LRDNVVATF	0.61	HLA-A*23:01
157	165	SMEQLKVKF	0.61	HLA-A*24:02
114	123	QVEDPSNSSV	0.61	HLA-B*40:01
302	310	VVTGKVDMV	0.61	HLA-A*02:06
404	412	LTAHFTTSI	0.61	HLA-A*02:06
451	461	RTISIWGTTLY	0.61	HLA-A*03:01
322	331	ADHPGLPISY	0.62	HLA-A*01:01
445	455	LPLVRKRTISI	0.62	HLA-B*08:01
333	341	TSFLRDNVV	0.62	HLA-B*51:01
232	242	RPLVYISSVAY	0.63	HLA-A*32:01
126	134	AVNDLLAKW	0.63	HLA-B*44:03
420	428	NLSVKIREC	0.63	HLA-B*08:01
228	236	ISAERPLVY	0.63	HLA-A*11:01
5	15	AVNDFILAMNY	0.63	HLA-A*11:01
234	242	LVYISSVAY	0.63	HLA-A*03:01
218	226	VTVEDLKQR	0.63	HLA-A*31:01
156	165	HSMEQLKVKF	0.64	HLA-A*32:01
377	384	ITWNELSY	0.64	HLA-A*01:01
283	291	DNTEVKAVI	0.64	HLA-B*51:01
142	150	NNVPARMQY	0.64	HLA-A*30:02
428	435	CTGLAWEW	0.64	HLA-B*58:01
240	247	VAYGRQVY	0.64	HLA-B*15:01
390	397	EVLTPKAW	0.65	HLA-B*53:01
115	123	VEDPSNSSV	0.65	HLA-B*44:02
439	447	VYEKTDLPL	0.65	HLA-A*24:02
257	266	DEVEAAFEAL	0.65	HLA-B*40:01
205	213	DAVKNPGDV	0.65	HLA-B*51:01
239	249	SVAYGRQVYLK	0.65	HLA-A*03:01
239	247	SVAYGRQVY	0.66	HLA-A*32:01
372	379	VAQYYITW	0.66	HLA-B*53:01

383	392	SYDHQGKEVL	0.66	HLA-A*24:02
114	123	QVEDPSNSSV	0.66	HLA-A*68:02
261	270	AAFEALIKGV	0.66	HLA-A*68:02
127	134	VNDLLAKW	0.66	HLA-B*58:01
265	272	ALIKGVKV	0.66	HLA-A*02:03
298	306	SGARVVTGK	0.66	HLA-A*03:01
410	419	TSIPLKGNVR	0.66	HLA-A*31:01
149	158	QYEKITAHSM	0.67	HLA-B*44:03
254	263	SKSDEVEAAF	0.67	HLA-A*01:01
95	103	APMTYSIDL	0.67	HLA-B*35:01
42	50	EFVVIERKK	0.67	HLA-A*33:01
122	130	SVRGAVNDL	0.67	HLA-A*30:01
370	379	AYVAQYYITW	0.67	HLA-B*58:01
199	208	YYTVSVDAVK	0.67	HLA-A*68:01
69	78	RLYPGALLVV	0.68	HLA-A*24:02
7	15	NDFILAMNY	0.68	HLA-B*44:03
255	263	KSDEVEAAF	0.68	HLA-B*35:01
346	355	NSTDYVETKV	0.68	HLA-A*68:02
89	99	LLAVDRAPMTY	0.68	HLA-A*30:02
279	287	KQILDNTEV	0.68	HLA-A*02:01
452	461	TISIWGTTLY	0.68	HLA-B*15:01
442	451	KTDLPLVRKR	0.68	HLA-A*03:01
143	150	NVPARMQY	0.69	HLA-A*26:01
410	419	TSIPLKGNVR	0.69	HLA-A*33:01
153	161	ITAHSMEQL	0.69	HLA-B*58:01
148	156	MQYEKITAH	0.69	HLA-A*03:01
226	236	RGISAERPLVY	0.69	HLA-B*57:01
309	319	MVEDLIQEGSR	0.69	HLA-A*68:01
188	195	KQIQIVNF	0.7	HLA-A*32:01
426	436	RECTGLAWEWW	0.7	HLA-B*44:02
328	335	PISYTTSF	0.7	HLA-B*53:01
328	335	PISYTTSF	0.7	HLA-B*35:01
458	468	TTLYPQVEDKV	0.7	HLA-A*68:02
43	51	FVVIERKKR	0.7	HLA-A*33:01
196	204	KQIYYTVSV	0.7	HLA-B*15:01
5	15	AVNDFILAMNY	0.7	HLA-B*15:01
417	426	NVRNLSVKIR	0.7	HLA-A*68:01
239	248	SVAYGRQVYL	0.71	HLA-A*32:01
442	450	KTDLPLVRK	0.71	HLA-A*01:01
302	310	VVTGKVDMV	0.71	HLA-A*68:02
192	200	IVNFKQIYY	0.71	HLA-A*30:01
269	279	GVKVAPQTEWK	0.71	HLA-A*30:01
240	248	VAYGRQVYL	0.71	HLA-A*30:01
211	220	GDVFQDTVTV	0.71	HLA-A*02:03
367	375	HSGAYVAQY	0.71	HLA-B*57:01
115	123	VEDPSNSSV	0.72	HLA-B*44:03
340	350	VVATFQNSTDY	0.72	HLA-A*01:01
377	384	ITWNELSY	0.72	HLA-A*30:02
25	34	GESIENRFIK	0.72	HLA-A*11:01
149	158	QYEKITAHSM	0.73	HLA-A*24:02
47	55	ERKKRSLST	0.73	HLA-B*08:01
341	350	VATFQNSTDY	0.73	HLA-B*35:01
448	455	VRKRTISI	0.73	HLA-B*08:01
321	329	TADHPGLPI	0.73	HLA-B*51:01
369	377	GAYVAQYYI	0.73	HLA-B*51:01
39	46	LPDEFVVI	0.74	HLA-B*53:01
125	134	GAVNDLLAKW	0.74	HLA-B*53:01
422	431	SVKIRECTGL	0.74	HLA-B*08:01
233	240	PLVYISSV	0.74	HLA-B*51:01
240	248	VAYGRQVYL	0.74	HLA-B*58:01
200	208	YTVSVDAVK	0.74	HLA-A*11:01
126	133	AVNDLLAK	0.74	HLA-A*11:01
428	435	CTGLAWEW	0.74	HLA-B*57:01
350	358	YVETKVTAY	0.75	HLA-B*53:01
373	382	AQYYITWNEL	0.75	HLA-A*24:02
229	236	SAERPLVY	0.75	HLA-B*35:01

408	418	FTTSIPLKGNV	0.75	HLA-A*68:02
237	247	ISSVAYGRQVY	0.75	HLA-A*30:02
240	248	VAYGRQVYL	0.75	HLA-A*02:03
279	288	KQILDNTEVK	0.75	HLA-A*03:01
69	76	RLYPGALL	0.76	HLA-A*32:01
373	382	AQYYITWNEL	0.76	HLA-A*23:01
259	267	VEAAFEALI	0.76	HLA-B*44:03
25	32	GESIENRF	0.76	HLA-B*40:01
284	292	NTEVKAVIL	0.76	HLA-B*08:01
211	220	GDVFQDTVTV	0.76	HLA-A*68:02
153	161	ITAHSMEQL	0.76	HLA-A*02:06
113	123	LQVEDPSNSSV	0.76	HLA-A*02:06
226	236	RGISAERPLVY	0.76	HLA-B*15:01
91	99	AVDRAPMTY	0.76	HLA-A*03:01
156	165	HSMEQLKVKF	0.77	HLA-A*26:01
400	408	NGQDLTAHF	0.77	HLA-B*53:01
160	169	QLKVKFGSDF	0.77	HLA-B*08:01
35	43	EGNQLPDEF	0.77	HLA-B*35:01
95	103	APMTYSIDL	0.77	HLA-B*08:01
103	112	LPGLASSDSF	0.77	HLA-B*07:02
342	350	ATFQNSTDY	0.77	HLA-B*58:01
139	148	GQVNNVPARM	0.77	HLA-B*15:01
232	242	RPLVYISSVAY	0.78	HLA-B*07:02
229	237	SAERPLVYI	0.78	HLA-B*51:01
81	90	TLLENNPTLL	0.78	HLA-A*02:03
279	287	KQILDNTEV	0.78	HLA-A*02:03
430	439	GLAWEWWRTV	0.78	HLA-A*02:03
154	162	TAHSMEQLK	0.78	HLA-A*11:01
127	134	VNDLLAKW	0.78	HLA-B*57:01
234	242	LVYISSVAY	0.79	HLA-A*11:01
148	156	MQYEKITAH	0.8	HLA-A*26:01
321	331	TADHPGLPISY	0.8	HLA-B*53:01
389	397	KEVLTPKAW	0.8	HLA-B*40:01
240	248	VAYGRQVYL	0.8	HLA-B*07:02
94	101	RAPMTYSI	0.8	HLA-B*51:01
147	156	RMQYEKITAH	0.8	HLA-A*30:02
52	62	SLSTNTSDISV	0.8	HLA-A*02:03
82	92	LLENNPTLLAV	0.8	HLA-A*02:03
281	290	ILDNTEVKAV	0.8	HLA-A*02:03
124	133	RGAVNDLLAK	0.8	HLA-A*11:01
91	99	AVDRAPMTY	0.81	HLA-B*53:01
355	365	VTAYRNGDLLL	0.81	HLA-A*24:02
191	200	QIVNFKQIYY	0.81	HLA-A*01:01
84	92	ENNPTLLAV	0.81	HLA-B*51:01
240	248	VAYGRQVYL	0.81	HLA-A*02:01
190	198	IQIVNFKQI	0.81	HLA-A*02:06
271	279	KVAPQTEWK	0.81	HLA-A*68:01
259	267	VEAAFEALI	0.82	HLA-B*44:02
342	350	ATFQNSTDY	0.82	HLA-A*32:01
440	448	YEKTDLPLV	0.82	HLA-B*44:03
426	436	RECTGLAWEWW	0.82	HLA-B*44:03
336	344	LRDNVVATF	0.82	HLA-A*24:02
431	440	LAWEWWRTVY	0.82	HLA-B*35:01
260	270	EAAFEALIKGV	0.82	HLA-A*68:02
356	364	TAYRNGDLL	0.82	HLA-B*51:01
368	376	SGAYVAQYY	0.82	HLA-B*15:01
228	236	ISAERPLVY	0.82	HLA-A*03:01
23	31	HQGESIENR	0.82	HLA-A*31:01
440	448	YEKTDLPLV	0.83	HLA-B*44:02
270	278	VKVAPQTEW	0.83	HLA-B*44:03
230	237	AERPLVYI	0.83	HLA-B*40:01
247	255	YLKLETTSK	0.83	HLA-A*30:01
310	320	VEDLIQEGSRF	0.83	HLA-B*15:01
340	350	VVATFQNSTDY	0.83	HLA-B*15:01
241	249	AYGRQVYLK	0.83	HLA-A*31:01
240	249	VAYGRQVYLK	0.83	HLA-A*68:01

234	242	LVYISSVAY	0.84	HLA-A*01:01
79	89	DETLLENNPTL	0.84	HLA-B*40:01
142	150	NNVPARMQY	0.84	HLA-B*35:01
445	455	LPLVRKRTISI	0.84	HLA-B*51:01
107	115	ASSDSFLQV	0.84	HLA-A*30:01
211	220	GDVFQDTVTV	0.84	HLA-A*02:01
107	115	ASSDSFLQV	0.84	HLA-A*02:01
212	220	DVFQDTVTV	0.84	HLA-A*02:06
273	281	APQTEWKQI	0.85	HLA-B*51:01
52	62	SLSTNTSDISV	0.85	HLA-A*02:01
56	64	NTSDISVTA	0.85	HLA-A*02:06
441	450	EKTDLPLVRK	0.85	HLA-A*11:01
425	435	IRECTGLAWEW	0.85	HLA-B*57:01
323	331	DHPGLPISY	0.86	HLA-A*26:01
158	165	MEQLKVKF	0.86	HLA-B*40:01
323	331	DHPGLPISY	0.86	HLA-B*35:01
312	320	DLIQEGSRF	0.86	HLA-B*35:01
402	412	QDLTAHFTTSI	0.86	HLA-A*68:02
228	237	ISAERPLVYI	0.86	HLA-A*68:02
417	425	NVRNLSVKI	0.86	HLA-A*68:02
246	255	VYLKLETTSK	0.86	HLA-A*30:01
150	158	YEKITAHSM	0.86	HLA-B*15:01
350	359	YVETKVTAYR	0.86	HLA-A*31:01
239	249	SVAYGRQVYLK	0.86	HLA-A*68:01
62	71	VTATNDSRLY	0.87	HLA-A*26:01
335	344	FLRDNVVATF	0.87	HLA-A*26:01
412	421	IPLKGNVRNL	0.87	HLA-B*53:01
439	447	VYEKTDLPL	0.87	HLA-A*23:01
355	365	VTAYRNGDLLL	0.87	HLA-A*23:01
356	365	TAYRNGDLLL	0.87	HLA-A*24:02
370	377	AYVAQYYI	0.87	HLA-A*24:02
67	77	DSRLYPGALLV	0.87	HLA-B*51:01
369	376	GAYVAQYY	0.87	HLA-A*30:02
204	214	VDAVKNPGDVF	0.87	HLA-B*15:01
62	71	VTATNDSRLY	0.87	HLA-B*57:01
7	15	NDFILAMNY	0.88	HLA-B*44:02
294	303	GDPSSGARVV	0.88	HLA-B*07:02
156	163	HSMEQLKV	0.88	HLA-B*51:01
62	71	VTATNDSRLY	0.88	HLA-B*58:01
63	71	TATNDSRLY	0.88	HLA-B*58:01
346	354	NSTDYVETK	0.88	HLA-A*11:01
269	279	GVKVAPQTEWK	0.88	HLA-A*11:01
312	320	DLIQEGSRF	0.88	HLA-B*15:01
155	164	AHSMEQLKVK	0.88	HLA-A*03:01
255	263	KSDEVEAAF	0.89	HLA-B*53:01
232	241	RPLVYISSVA	0.89	HLA-B*07:02
240	247	VAYGRQVY	0.89	HLA-A*30:02
412	421	IPLKGNVRNL	0.89	HLA-B*51:01
218	226	VTVEDLKQR	0.89	HLA-A*33:01
64	72	ATNDSRLYP	0.89	HLA-A*30:01
26	34	ESIENRFIK	0.89	HLA-A*11:01
386	395	HQGKEVLTPK	0.89	HLA-A*03:01
428	436	CTGLAWEWW	0.89	HLA-B*57:01
43	51	FVVIERKKR	0.89	HLA-A*68:01
143	152	NVPARMQYEK	0.89	HLA-A*68:01
441	450	EKTDLPLVRK	0.89	HLA-A*68:01
270	278	VKVAPQTEW	0.9	HLA-B*44:02
271	278	KVAPQTEW	0.9	HLA-B*53:01
150	158	YEKITAHSM	0.9	HLA-B*35:01
43	53	FVVIERKKRSL	0.9	HLA-B*08:01
45	53	VIERKKRSL	0.9	HLA-B*07:02
171	180	KTGNSLDIDF	0.9	HLA-B*58:01
404	412	LTAHFTTSI	0.9	HLA-A*02:03
82	90	LLENNPTLL	0.9	HLA-A*02:03
341	350	VATFQNSTDY	0.9	HLA-B*15:01
271	279	KVAPQTEWK	0.9	HLA-A*31:01

38	48	QLPDEFVVIER	0.9	HLA-A*68:01
364	372	LLDHSGAYV	0.91	HLA-A*01:01
97	107	MTYSIDLPGLA	0.91	HLA-A*68:02
331	340	YTTSFLRDNV	0.91	HLA-A*68:02
235	242	VYISSVAY	0.91	HLA-A*30:02
367	376	HSGAYVAQYY	0.91	HLA-B*58:01
82	92	LLENNPTLLAV	0.91	HLA-A*02:01
37	45	NQLPDEFVV	0.91	HLA-A*02:01
59	69	DISVTATNDSR	0.91	HLA-A*68:01
319	327	RFTADHPGL	0.92	HLA-A*24:02
206	214	AVKNPGDVF	0.92	HLA-B*07:02
411	419	SIPLKGNVR	0.92	HLA-A*33:01
192	200	IVNFKQIYY	0.92	HLA-B*58:01
239	248	SVAYGRQVYL	0.92	HLA-A*02:03
232	242	RPLVYISSVAY	0.92	HLA-A*03:01
90	99	LAVDRAPMTY	0.92	HLA-B*57:01
230	237	AERPLVYI	0.93	HLA-B*44:02
310	320	VEDLIQEGSRF	0.93	HLA-B*40:01
192	199	IVNFKQIY	0.93	HLA-A*30:02
430	439	GLAWEWWRTV	0.93	HLA-A*02:01
152	162	KITAHSMEQLK	0.93	HLA-A*11:01
195	204	FKQIYYTVSV	0.93	HLA-A*02:06
240	248	VAYGRQVYL	0.93	HLA-B*57:01
453	461	ISIWGTTLY	0.94	HLA-B*53:01
447	456	LVRKRTISIW	0.94	HLA-A*32:01
234	242	LVYISSVAY	0.94	HLA-B*53:01
193	200	VNFKQIYY	0.94	HLA-A*30:02
5	13	AVNDFILAM	0.94	HLA-A*30:01
69	76	RLYPGALL	0.94	HLA-A*02:01
335	344	FLRDNVVATF	0.94	HLA-A*02:03
431	439	LAWEWWRTV	0.94	HLA-A*02:06
230	237	AERPLVYI	0.95	HLA-B*44:03
126	134	AVNDLLAKW	0.95	HLA-A*23:01
383	392	SYDHQGKEVL	0.95	HLA-A*23:01
142	150	NNVPARMQY	0.95	HLA-A*01:01
69	76	RLYPGALL	0.95	HLA-A*02:03
387	395	QGKEVLTPK	0.95	HLA-A*11:01
430	440	GLAWEWWRTVY	0.95	HLA-B*15:01
447	455	LVRKRTISI	0.96	HLA-A*32:01
199	207	YYTVSVDAV	0.96	HLA-A*24:02
428	436	CTGLAWEWW	0.96	HLA-B*58:01
139	147	GQVNNVPAR	0.96	HLA-A*31:01
148	158	MQYEKITAHSM	0.97	HLA-A*32:01
38	46	QLPDEFVVI	0.97	HLA-A*24:02
184	192	HSGEKQIQI	0.97	HLA-B*58:01
82	89	LLENNPTL	0.97	HLA-A*02:01
75	83	LLVVDETLL	0.97	HLA-A*02:01
218	226	VTVEDLKQR	0.97	HLA-A*11:01
453	461	ISIWGTTLY	0.97	HLA-A*11:01
1	9	MANKAVNDF	0.97	HLA-B*57:01
376	384	YITWNELSY	0.98	HLA-B*53:01
372	382	VAQYYITWNEL	0.98	HLA-A*23:01
263	272	FEALIKGVKV	0.98	HLA-B*40:01
212	220	DVFQDTVTV	0.98	HLA-B*35:01
415	423	KGNVRNLSV	0.98	HLA-B*08:01
221	230	EDLKQRGISA	0.98	HLA-B*08:01
332	341	TTSFLRDNVV	0.98	HLA-A*68:02
440	450	YEKTDLPLVRK	0.98	HLA-A*11:01
5	13	AVNDFILAM	0.98	HLA-A*11:01
224	232	KQRGISAER	0.98	HLA-A*03:01
381	389	ELSYDHQGK	0.98	HLA-A*68:01
234	244	LVYISSVAYGR	0.98	HLA-A*68:01
258	266	EVEAAFEAL	0.99	HLA-A*26:01
61	71	SVTATNDSRLY	0.99	HLA-A*26:01
372	382	VAQYYITWNEL	0.99	HLA-A*24:02
321	329	TADHPGLPI	0.99	HLA-A*01:01

39	47	LPDEFVVIE	0.99	HLA-B*35:01
26	33	ESIENRFI	0.99	HLA-B*51:01
330	337	SYTTSFLR	0.99	HLA-A*33:01
280	288	QILDNTEVK	0.99	HLA-A*68:01
21	31	LTHQGESIENR	0.99	HLA-A*68:01
149	158	QYEKITAHSM	1.0	HLA-A*23:01
108	115	SSDSFLQV	1.0	HLA-A*01:01
371	379	YVAQYYITW	1.0	HLA-B*35:01
324	332	HPGLPISYT	1.0	HLA-B*07:02
404	412	LTAHFTTSI	1.0	HLA-B*51:01
155	164	AHSMEQLKVK	1.0	HLA-A*30:01
148	156	MQYEKITAH	1.0	HLA-A*02:06

Table XVI – Results of the Ply-MHC-II binding prediction.
Acquired with TepiTool (http://tools.iedb.org/tepitool/).

Ply MHC-II				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
327	341	LPISYTTSFLRDNVV	2.00	HLA-DPA1*01/DPB1*04:01
453	467	ISIWGTTLYPQVEDK	2.55	HLA-DPA1*01/DPB1*04:01
102	116	DLPGLASSDSFLQVE	3.00	HLA-DPA1*01/DPB1*04:01
107	121	ASSDSFLQVEDPSNS	6.25	HLA-DPA1*01/DPB1*04:01
368	382	SGAYVAQYYITWNEL	8.70	HLA-DPA1*01/DPB1*04:01
429	443	TGLAWEWWRTVYEKT	2.30	HLA-DPA1*01:03/DPB1*02:01
368	382	SGAYVAQYYITWNEL	2.90	HLA-DPA1*01:03/DPB1*02:01
327	341	LPISYTTSFLRDNVV	4.80	HLA-DPA1*01:03/DPB1*02:01
102	116	DLPGLASSDSFLQVE	5.50	HLA-DPA1*01:03/DPB1*02:01
190	204	IQIVNFKQIYYTVSV	7.40	HLA-DPA1*01:03/DPB1*02:01
237	251	ISSVAYGRQVYLKLE	5.80	HLA-DPA1*02:01/DPB1*01:01
190	204	IQIVNFKQIYYTVSV	6.90	HLA-DPA1*02:01/DPB1*01:01
72	86	PGALLVVDETLLENN	7.00	HLA-DPA1*02:01/DPB1*01:01
373	387	AQYYITWNELSYDHQ	7.70	HLA-DPA1*02:01/DPB1*01:01
327	341	LPISYTTSFLRDNVV	7.70	HLA-DPA1*02:01/DPB1*01:01
185	199	SGEKQIQIVNFKQIY	8.10	HLA-DPA1*02:01/DPB1*01:01
327	341	LPISYTTSFLRDNVV	1.70	HLA-DPA1*02:01/DPB1*05:01
242	256	YGRQVYLKLETTSKS	5.00	HLA-DPA1*02:01/DPB1*05:01
1	15	MANKAVNDFILAMNY	6.70	HLA-DPA1*02:01/DPB1*05:01
257	271	DEVEAAFEALIKGVK	7.60	HLA-DPA1*02:01/DPB1*05:01
72	86	PGALLVVDETLLENN	6.90	HLA-DPA1*03:01/DPB1*04:02
327	341	LPISYTTSFLRDNVV	7.50	HLA-DPA1*03:01/DPB1*04:02
185	199	SGEKQIQIVNFKQIY	8.40	HLA-DPA1*03:01/DPB1*04:02
190	204	IQIVNFKQIYYTVSV	8.50	HLA-DPA1*03:01/DPB1*04:02
404	418	LTAHFTTSIPLKGNV	9.40	HLA-DPA1*03:01/DPB1*04:02
373	387	AQYYITWNELSYDHQ	3.30	HLA-DQA1*01:01/DQB1*05:01
1	15	MANKAVNDFILAMNY	5.50	HLA-DQA1*01:01/DQB1*05:01
429	443	TGLAWEWWRTVYEKT	7.50	HLA-DQA1*01:01/DQB1*05:01
128	142	NDLLAKWHQDYGQVN	8.50	HLA-DQA1*01:01/DQB1*05:01
368	382	SGAYVAQYYITWNEL	9.30	HLA-DQA1*01:01/DQB1*05:01
294	308	GDPSSGARVVTGKVD	1.40	HLA-DQA1*01:02/DQB1*06:02
263	277	FEALIKGVKVAPQTE	3.90	HLA-DQA1*01:02/DQB1*06:02
363	377	LLLDHSGAYVAQYYI	8.60	HLA-DQA1*01:02/DQB1*06:02
232	246	RPLVYISSVAYGRQV	8.90	HLA-DQA1*01:02/DQB1*06:02
81	95	TLLENNPTLLAVDRA	9.20	HLA-DQA1*01:02/DQB1*06:02
252	266	TTSKSDEVEAAFEAL	1.30	HLA-DQA1*03:01/DQB1*03:02
107	121	ASSDSFLQVEDPSNS	3.50	HLA-DQA1*03:01/DQB1*03:02
102	116	DLPGLASSDSFLQVE	5.20	HLA-DQA1*03:01/DQB1*03:02
209	223	NPGDVFQDTVTVEDL	8.10	HLA-DQA1*03:01/DQB1*03:02
453	467	ISIWGTTLYPQVEDK	8.20	HLA-DQA1*03:01/DQB1*03:02
257	271	DEVEAAFEALIKGVK	8.80	HLA-DQA1*03:01/DQB1*03:02

67	81	DSRLYPGALLVVDET	4.50	HLA-DQA1*04:01/DQB1*04:02
252	266	TTSKSDEVEAAFEAL	4.80	HLA-DQA1*04:01/DQB1*04:02
363	377	LLLDHSGAYVAQYYI	8.20	HLA-DQA1*04:01/DQB1*04:02
102	116	DLPGLASSDSFLQVE	9.00	HLA-DQA1*04:01/DQB1*04:02
453	467	ISIWGTTLYPQVEDK	9.50	HLA-DQA1*04:01/DQB1*04:02
196	210	KQIYYTVSVDAVKNP	2.70	HLA-DQA1*05:01/DQB1*02:01
373	387	AQYYITWNELSYDHQ	8.10	HLA-DQA1*05:01/DQB1*02:01
72	86	PGALLVVDETLLENN	8.30	HLA-DQA1*05:01/DQB1*02:01
303	317	VTGKVDMVEDLIQEG	8.60	HLA-DQA1*05:01/DQB1*02:01
67	81	DSRLYPGALLVVDET	9.10	HLA-DQA1*05:01/DQB1*02:01
294	308	GDPSSGARVVTGKVD	1.90	HLA-DQA1*05:01/DQB1*03:01
287	301	VKAVILGGDPSSGAR	3.00	HLA-DQA1*05:01/DQB1*03:01
363	377	LLLDHSGAYVAQYYI	8.10	HLA-DQA1*05:01/DQB1*03:01
147	161	RMQYEKITAHSMEQL	5.10	HLA-DRB1*01:01
232	246	RPLVYISSVAYGRQV	7.80	HLA-DRB1*01:01
242	256	YGRQVYLKLETTSKS	9.10	HLA-DRB1*01:01
404	418	LTAHFTTSIPLKGNV	9.80	HLA-DRB1*01:01
72	86	PGALLVVDETLLENN	0.79	HLA-DRB1*03:01
8	22	DFILAMNYDKKKLLT	1.30	HLA-DRB1*03:01
41	55	DEFVVIERKKRSLST	1.40	HLA-DRB1*03:01
224	238	KQRGISAERPLVYIS	1.70	HLA-DRB1*03:01
358	372	YRNGDLLLDHSGAYV	2.80	HLA-DRB1*03:01
363	377	LLLDHSGAYVAQYYI	3.40	HLA-DRB1*03:01
88	102	TLLAVDRAPMTYSID	3.80	HLA-DRB1*03:01
13	27	MNYDKKKLLTHQGES	6.10	HLA-DRB1*03:01
332	346	TTSFLRDNVVATFQN	7.10	HLA-DRB1*03:01
209	223	NPGDVFQDTVTVEDL	7.70	HLA-DRB1*03:01
437	451	RTVYEKTDLPLVRKR	9.40	HLA-DRB1*03:01
242	256	YGRQVYLKLETTSKS	0.41	HLA-DRB1*04:01
332	346	TTSFLRDNVVATFQN	1.40	HLA-DRB1*04:01
72	86	PGALLVVDETLLENN	3.60	HLA-DRB1*04:01
232	246	RPLVYISSVAYGRQV	3.60	HLA-DRB1*04:01
373	387	AQYYITWNELSYDHQ	3.80	HLA-DRB1*04:01
135	149	HQDYGQVNNVPARMQ	4.30	HLA-DRB1*04:01
247	261	YLKLETTSKSDEVEA	4.70	HLA-DRB1*04:01
107	121	ASSDSFLQVEDPSNS	7.00	HLA-DRB1*04:01
29	43	ENRFIKEGNQLPDEF	7.40	HLA-DRB1*04:01
88	102	TLLAVDRAPMTYSID	8.00	HLA-DRB1*04:01
287	301	VKAVILGGDPSSGAR	8.70	HLA-DRB1*04:01
196	210	KQIYYTVSVDAVKNP	8.90	HLA-DRB1*04:01
72	86	PGALLVVDETLLENN	2.30	HLA-DRB1*04:05
232	246	RPLVYISSVAYGRQV	2.70	HLA-DRB1*04:05
274	288	PQTEWKQILDNTEVK	4.20	HLA-DRB1*04:05
242	256	YGRQVYLKLETTSKS	5.40	HLA-DRB1*04:05
263	277	FEALIKGVKVAPQTE	7.30	HLA-DRB1*04:05
107	121	ASSDSFLQVEDPSNS	7.50	HLA-DRB1*04:05
373	387	AQYYITWNELSYDHQ	9.00	HLA-DRB1*04:05
338	352	DNVVATFQNSTDYVE	9.00	HLA-DRB1*04:05
166	180	GSDFEKTGNSLDIDF	9.80	HLA-DRB1*04:05
404	418	LTAHFTTSIPLKGNV	2.80	HLA-DRB1*07:01
232	246	RPLVYISSVAYGRQV	5.70	HLA-DRB1*07:01
327	341	LPISYTTSFLRDNVV	6.00	HLA-DRB1*07:01
442	456	KTDLPLVRKRTISIW	7.10	HLA-DRB1*07:01
398	412	DRNGQDLTAHFTTSI	9.00	HLA-DRB1*07:01
147	161	RMQYEKITAHSMEQL	9.10	HLA-DRB1*07:01
263	277	FEALIKGVKVAPQTE	0.55	HLA-DRB1*08:02
41	55	DEFVVIERKKRSLST	3.50	HLA-DRB1*08:02
88	102	TLLAVDRAPMTYSID	4.10	HLA-DRB1*08:02
442	456	KTDLPLVRKRTISIW	4.90	HLA-DRB1*08:02
232	246	RPLVYISSVAYGRQV	4.90	HLA-DRB1*08:02
190	204	IQIVNFKQIYYTVSV	6.20	HLA-DRB1*08:02
190	204	IQIVNFKQIYYTVSV	6.60	HLA-DRB1*09:01
94	108	RAPMTYSIDLPGLAS	7.10	HLA-DRB1*09:01
404	418	LTAHFTTSIPLKGNV	8.00	HLA-DRB1*09:01
41	55	DEFVVIERKKRSLST	0.34	HLA-DRB1*11:01
442	456	KTDLPLVRKRTISIW	4.30	HLA-DRB1*11:01

412	426	IPLKGNVRNLSVKIR	6.80	HLA-DRB1*11:01
257	271	DEVEAAFEALIKGVK	6.90	HLA-DRB1*11:01
263	277	FEALIKGVKVAPQTE	7.00	HLA-DRB1*11:01
232	246	RPLVYISSVAYGRQV	1.75	HLA-DRB1*12:01
190	204	IQIVNFKQIYYTVSV	3.29	HLA-DRB1*12:01
81	95	TLLENNPTLLAVDRA	1.60	HLA-DRB1*13:02
412	426	IPLKGNVRNLSVKIR	1.80	HLA-DRB1*13:02
135	149	HQDYGQVNNVPARMQ	4.50	HLA-DRB1*13:02
332	346	TTSFLRDNVVATFQN	5.30	HLA-DRB1*13:02
442	456	KTDLPLVRKRTISIW	6.10	HLA-DRB1*13:02
417	431	NVRNLSVKIRECTGL	6.90	HLA-DRB1*13:02
358	372	YRNGDLLLDHSGAYV	8.20	HLA-DRB1*13:02
232	246	RPLVYISSVAYGRQV	2.50	HLA-DRB1*15:01
190	204	IQIVNFKQIYYTVSV	2.80	HLA-DRB1*15:01
352	366	ETKVTAYRNGDLLLD	9.90	HLA-DRB1*15:01
358	372	YRNGDLLLDHSGAYV	1.10	HLA-DRB3*01:01
332	346	TTSFLRDNVVATFQN	1.50	HLA-DRB3*01:01
94	108	RAPMTYSIDLPGLAS	2.10	HLA-DRB3*01:01
88	102	TLLAVDRAPMTYSID	3.50	HLA-DRB3*01:01
8	22	DFILAMNYDKKKLLT	5.50	HLA-DRB3*01:01
317	331	GSRFTADHPGLPISY	5.60	HLA-DRB3*01:01
1	15	MANKAVNDFILAMNY	7.70	HLA-DRB3*01:01
72	86	PGALLVVDETLLENN	8.90	HLA-DRB3*01:01
332	346	TTSFLRDNVVATFQN	1.10	HLA-DRB3*02:02
81	95	TLLENNPTLLAVDRA	1.50	HLA-DRB3*02:02
412	426	IPLKGNVRNLSVKIR	1.70	HLA-DRB3*02:02
88	102	TLLAVDRAPMTYSID	2.80	HLA-DRB3*02:02
232	246	RPLVYISSVAYGRQV	3.00	HLA-DRB3*02:02
50	64	KRSLSTNTSDISVTA	4.00	HLA-DRB3*02:02
8	22	DFILAMNYDKKKLLT	5.30	HLA-DRB3*02:02
196	210	KQIYYTVSVDAVKNP	5.70	HLA-DRB3*02:02
135	149	HQDYGQVNNVPARMQ	6.50	HLA-DRB3*02:02
404	418	LTAHFTTSIPLKGNV	6.60	HLA-DRB3*02:02
373	387	AQYYITWNELSYDHQ	9.50	HLA-DRB3*02:02
8	22	DFILAMNYDKKKLLT	6.30	HLA-DRB4*01:01
442	456	KTDLPLVRKRTISIW	6.50	HLA-DRB4*01:01
185	199	SGEKQIQIVNFKQIY	8.50	HLA-DRB4*01:01
232	246	RPLVYISSVAYGRQV	0.05	HLA-DRB5*01:01
41	55	DEFVVIERKKRSLST	0.16	HLA-DRB5*01:01
177	191	DIDFNSVHSGEKQIQ	1.30	HLA-DRB5*01:01
8	22	DFILAMNYDKKKLLT	1.60	HLA-DRB5*01:01
257	271	DEVEAAFEALIKGVK	3.90	HLA-DRB5*01:01
242	256	YGRQVYLKLETTSKS	4.20	HLA-DRB5*01:01
327	341	LPISYTTSFLRDNVV	6.20	HLA-DRB5*01:01
147	161	RMQYEKITAHSMEQL	9.50	HLA-DRB5*01:01
404	418	LTAHFTTSIPLKGNV	9.90	HLA-DRB5*01:01

Table XVII – Results of the PspA-MHC-I binding prediction.

Acquired with TepiTool (http://tools.iedb.org/tepitool/).

PspA MHC-I				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
421	429	ATKKAELEK	0.01	HLA-A*30:01
610	618	LQYNGSWYY	0.01	HLA-A*30:02
604	612	AMATGWLQY	0.01	HLA-A*30:02
264	272	ELAKKQTEL	0.01	HLA-B*08:01
109	117	EYREVQNQR	0.01	HLA-A*33:01
177	186	AEEKVAKRKY	0.01	HLA-B*44:02
203	212	KELEIEKLQY	0.01	HLA-B*44:03
189	197	ATLKVALAK	0.01	HLA-A*11:01
177	186	AEEKVAKRKY	0.01	HLA-B*44:03
245	253	EVIEAKLKK	0.01	HLA-A*68:01

314	322	KEISNLEIL	0.01	HLA-B*40:01
220	228	EVATAQHQV	0.01	HLA-A*68:02
88	96	KEAEASQKL	0.01	HLA-B*40:01
51	59	KAKADTAKK	0.02	HLA-A*30:01
610	618	LQYNGSWYY	0.02	HLA-B*15:01
189	197	ATLKVALAK	0.02	HLA-A*03:01
87	96	RKEAEASQKL	0.02	HLA-B*40:01
257	265	ELNAKQAEL	0.03	HLA-B*08:01
112	120	EVQNQRSKY	0.03	HLA-A*26:01
88	96	KEAEASQKL	0.03	HLA-B*44:02
604	612	AMATGWLQY	0.03	HLA-B*15:01
111	120	REVQNQRSKY	0.03	HLA-B*44:02
95	103	KLNDVALVV	0.03	HLA-A*02:03
142	151	KEQQDLQNKF	0.03	HLA-B*44:02
203	212	KELEIEKLQY	0.03	HLA-B*44:02
178	186	EEKVAKRKY	0.03	HLA-B*44:02
415	423	ALQNKLATK	0.03	HLA-A*03:01
142	151	KEQQDLQNKF	0.03	HLA-B*44:03
95	103	KLNDVALVV	0.03	HLA-A*02:01
432	441	KELDAALNEL	0.03	HLA-B*40:01
340	348	AAKKAELAK	0.04	HLA-A*30:01
549	557	LQNNGSWYY	0.04	HLA-A*30:02
530	538	QENGMWYFY	0.04	HLA-B*44:02
425	434	AELEKTQKEL	0.04	HLA-B*44:02
331	339	DTAALQNKL	0.04	HLA-A*68:02
611	619	QYNGSWYYL	0.04	HLA-A*24:02
178	186	EEKVAKRKY	0.04	HLA-B*44:03
99	107	VALVVQNAY	0.04	HLA-B*35:01
530	538	QENGMWYFY	0.04	HLA-B*44:03
111	120	REVQNQRSKY	0.04	HLA-B*44:03
88	96	KEAEASQKL	0.04	HLA-B*44:03
34	43	SPQVVEKSSL	0.04	HLA-B*07:02
651	659	KVNGSWYYL	0.05	HLA-A*32:01
583	591	AMKASQWFK	0.05	HLA-A*30:01
105	113	NAYKEYREV	0.05	HLA-B*51:01
38	47	VEKSSLEKKY	0.05	HLA-B*44:02
611	619	QYNGSWYYL	0.05	HLA-A*23:01
568	577	WVKDGDTWYY	0.05	HLA-A*01:01
334	342	ALQNKLAAK	0.05	HLA-A*03:01
38	47	VEKSSLEKKY	0.05	HLA-B*44:03
425	434	AELEKTQKEL	0.05	HLA-B*44:03
86	96	ARKEAEASQKL	0.05	HLA-B*40:01
586	596	ASQWFKVSDKW	0.05	HLA-B*57:01
189	197	ATLKVALAK	0.06	HLA-A*30:01
727	735	AVNTTVDGY	0.06	HLA-A*30:02
98	107	DVALVVQNAY	0.06	HLA-A*26:01
1	8	MNKKKMIL	0.06	HLA-B*08:01
204	212	ELEIEKLQY	0.06	HLA-A*01:01
583	591	AMKASQWFK	0.06	HLA-A*03:01
189	198	ATLKVALAKK	0.06	HLA-A*11:01
591	599	KVSDKWYYV	0.06	HLA-A*02:06
256	265	AELNAKQAEL	0.06	HLA-B*40:01
22	30	VTSQPTFVR	0.06	HLA-A*68:01
425	434	AELEKTQKEL	0.06	HLA-B*40:01
520	528	KPATPKTGW	0.07	HLA-B*53:01
102	110	VVQNAYKEY	0.07	HLA-A*30:02
188	195	YATLKVAL	0.07	HLA-B*08:01
213	221	EISTLEQEV	0.07	HLA-A*68:02
683	691	GSMATGWVK	0.07	HLA-A*11:01
586	596	ASQWFKVSDKW	0.07	HLA-B*58:01
265	272	LAKKQTEL	0.08	HLA-B*08:01
22	30	VTSQPTFVR	0.08	HLA-A*31:01
95	103	KLNDVALVV	0.08	HLA-A*02:06
421	429	ATKKAELEK	0.08	HLA-A*11:01
222	232	ATAQHQVDNLK	0.08	HLA-A*11:01
549	557	LQNNGSWYY	0.08	HLA-B*15:01

117	126	RSKYKSDAEY	0.09	HLA-A*30:02
588	596	QWFKVSDKW	0.09	HLA-A*23:01
86	96	ARKEAEASQKL	0.09	HLA-B*44:02
664	672	AMATGWAKV	0.09	HLA-A*02:03
21	29	FVTSQPTFV	0.09	HLA-A*02:06
28	37	FVRAEESPQV	0.09	HLA-A*02:06
591	599	KVSDKWYYV	0.09	HLA-A*02:01
296	305	AELDKKADEL	0.09	HLA-B*40:01
314	323	KEISNLEILL	0.09	HLA-B*40:01
646	656	ATGWAKVNGSW	0.09	HLA-B*57:01
525	535	KTGWKQENGMW	0.09	HLA-B*57:01
624	632	DMATGWLQY	0.1	HLA-A*26:01
146	154	DLQNKFNEV	0.1	HLA-B*08:01
180	188	KVAKRKYDY	0.1	HLA-A*30:02
315	323	EISNLEILL	0.1	HLA-A*68:02
37	47	VVEKSSLEKKY	0.1	HLA-A*01:01
68	75	AEDAQKKY	0.1	HLA-B*44:03
86	96	ARKEAEASQKL	0.1	HLA-B*44:03
568	576	WVKDGDTWY	0.11	HLA-A*26:01
113	121	VQNQRSKYK	0.11	HLA-A*30:01
608	618	GWLQYNGSWYY	0.11	HLA-A*30:02
529	538	KQENGMWYFY	0.11	HLA-A*30:02
20	28	GFVTSQPTF	0.11	HLA-A*23:01
98	106	DVALVVQNA	0.11	HLA-A*68:02
218	228	EQEVATAQHQV	0.11	HLA-A*68:02
209	217	KLQYEISTL	0.11	HLA-A*02:03
68	75	AEDAQKKY	0.11	HLA-B*44:02
6	14	MILTSLASV	0.11	HLA-A*02:06
108	117	KEYREVQNQR	0.11	HLA-A*31:01
189	198	ATLKVALAKK	0.11	HLA-A*03:01
565	575	ATGWVKDGDTW	0.11	HLA-B*58:01
568	577	WVKDGDTWYY	0.12	HLA-A*26:01
588	596	QWFKVSDKW	0.12	HLA-A*24:02
226	234	HQVDNLKKL	0.12	HLA-A*02:06
243	253	GTEVIEAKLKK	0.12	HLA-A*11:01
415	424	ALQNKLATKK	0.12	HLA-A*03:01
201	209	EAKELEIEK	0.12	HLA-A*68:01
21	29	FVTSQPTFV	0.13	HLA-A*68:02
21	29	FVTSQPTFV	0.13	HLA-A*02:03
591	599	KVSDKWYYV	0.13	HLA-A*02:03
103	111	VQNAYKEYR	0.13	HLA-A*31:01
209	217	KLQYEISTL	0.13	HLA-A*02:01
663	671	GAMATGWAK	0.13	HLA-A*11:01
244	253	TEVIEAKLKK	0.13	HLA-A*11:01
270	279	TELEKLLDSL	0.13	HLA-B*40:01
263	272	AELAKKQTEL	0.13	HLA-B*40:01
651	659	KVNGSWYYL	0.14	HLA-A*30:01
304	312	ELQNKVADL	0.14	HLA-B*08:01
67	75	KAEDAQKKY	0.14	HLA-A*01:01
194	202	ALAKKEVEA	0.14	HLA-A*02:03
250	258	KLKKGEAEL	0.14	HLA-A*02:03
93	101	SQKLNDVAL	0.14	HLA-B*15:01
129	139	KLTEVDSKIEK	0.14	HLA-A*03:01
580	589	ASGAMKASQW	0.14	HLA-B*57:01
209	217	KLQYEISTL	0.15	HLA-A*32:01
20	28	GFVTSQPTF	0.15	HLA-A*24:02
338	346	KLAAKKAEL	0.15	HLA-A*02:03
203	210	KELEIEKL	0.15	HLA-B*40:01
591	599	KVSDKWYYV	0.16	HLA-A*32:01
610	618	LQYNGSWYY	0.16	HLA-A*32:01
107	117	YKEYREVQNQR	0.16	HLA-A*33:01
111	120	REVQNQRSKY	0.16	HLA-A*30:02
37	45	VVEKSSLEK	0.16	HLA-A*11:01
22	30	VTSQPTFVR	0.16	HLA-A*11:01
21	30	FVTSQPTFVR	0.16	HLA-A*68:01
565	575	ATGWVKDGDTW	0.16	HLA-B*57:01

220	228	EVATAQHQV	0.17	HLA-A*26:01
620	629	NANGDMATGW	0.17	HLA-B*53:01
419	427	KLATKKAEL	0.17	HLA-B*08:01
651	658	KVNGSWYY	0.17	HLA-A*30:02
603	612	GAMATGWLQY	0.17	HLA-A*30:02
256	265	AELNAKQAEL	0.17	HLA-B*44:02
129	137	KLTEVDSKI	0.17	HLA-A*02:01
338	348	KLAAKKAELAK	0.17	HLA-A*03:01
421	429	ATKKAELEK	0.17	HLA-A*03:01
606	616	ATGWLQYNGSW	0.17	HLA-B*57:01
529	537	KQENGMWYF	0.18	HLA-A*32:01
604	612	AMATGWLQY	0.18	HLA-A*32:01
519	528	EKPATPKTGW	0.18	HLA-B*53:01
142	151	KEQQDLQNKF	0.18	HLA-B*15:01
190	198	TLKVALAKK	0.18	HLA-A*03:01
646	656	ATGWAKVNGSW	0.18	HLA-B*58:01
591	599	KVSDKWYYV	0.19	HLA-A*30:01
67	75	KAEDAQKKY	0.19	HLA-A*30:02
129	137	KLTEVDSKI	0.19	HLA-A*02:03
419	427	KLATKKAEL	0.19	HLA-A*02:03
256	265	AELNAKQAEL	0.19	HLA-B*44:03
291	298	KEAEEAEL	0.19	HLA-B*40:01
583	591	AMKASQWFK	0.19	HLA-A*31:01
198	205	KEVEAKEL	0.19	HLA-B*40:01
331	339	DTAALQNKL	0.2	HLA-A*26:01
415	423	ALQNKLATK	0.2	HLA-A*30:01
36	43	QVVEKSSL	0.2	HLA-B*08:01
461	470	APAPKPEQPA	0.2	HLA-B*07:02
463	472	APKPEQPAPA	0.2	HLA-B*07:02
583	591	AMKASQWFK	0.2	HLA-A*11:01
419	429	KLATKKAELEK	0.2	HLA-A*03:01
604	612	AMATGWLQY	0.2	HLA-A*03:01
600	609	NSNGAMATGW	0.2	HLA-B*58:01
580	589	ASGAMKASQW	0.2	HLA-B*58:01
600	609	NSNGAMATGW	0.2	HLA-B*57:01
22	30	VTSQPTFVR	0.21	HLA-A*33:01
301	309	KADELQNKV	0.21	HLA-A*02:06
6	14	MILTSLASV	0.21	HLA-A*02:03
520	528	KPATPKTGW	0.21	HLA-B*07:02
430	438	TQKELDAAL	0.21	HLA-B*15:01
36	45	QVVEKSSLEK	0.21	HLA-A*11:01
727	736	AVNTTVDGYK	0.21	HLA-A*11:01
95	103	KLNDVALVV	0.22	HLA-A*32:01
338	346	KLAAKKAEL	0.22	HLA-B*08:01
146	155	DLQNKFNEVR	0.22	HLA-A*33:01
143	151	EQQDLQNKF	0.22	HLA-B*44:02
263	272	AELAKKQTEL	0.22	HLA-B*44:02
28	37	FVRAEESPQV	0.22	HLA-A*02:01
608	618	GWLQYNGSWYY	0.22	HLA-B*15:01
562	570	GAMATGWVK	0.22	HLA-A*11:01
727	736	AVNTTVDGYK	0.22	HLA-A*03:01
334	343	ALQNKLAAKK	0.22	HLA-A*03:01
132	141	EVDSKIEKAR	0.22	HLA-A*68:01
579	589	EASGAMKASQW	0.23	HLA-B*53:01
680	689	NANGSMATGW	0.23	HLA-B*53:01
99	107	VALVVQNAY	0.23	HLA-A*30:02
87	96	RKEAEASQKL	0.23	HLA-B*44:02
28	37	FVRAEESPQV	0.23	HLA-A*02:03
87	96	RKEAEASQKL	0.23	HLA-B*44:03
143	151	EQQDLQNKF	0.23	HLA-B*44:03
21	29	FVTSQPTFV	0.23	HLA-A*02:01
414	423	AALQNKLATK	0.23	HLA-A*03:01
727	735	AVNTTVDGY	0.24	HLA-A*26:01
559	568	NANGAMATGW	0.24	HLA-B*53:01
339	346	LAAKKAEL	0.24	HLA-B*08:01
547	557	GWLQNNGSWYY	0.24	HLA-A*30:02

650	658	AKVNGSWYY	0.24	HLA-A*30:02
604	612	AMATGWLQY	0.24	HLA-A*01:01
6	14	MILTSLASV	0.24	HLA-A*02:01
198	207	KEVEAKELEI	0.24	HLA-B*40:01
415	423	ALQNKLATK	0.24	HLA-A*11:01
13	21	SVAILGAGF	0.25	HLA-A*26:01
190	198	TLKVALAKK	0.25	HLA-A*30:01
102	110	VVQNAYKEY	0.25	HLA-B*15:01
525	535	KTGWKQENGMW	0.25	HLA-B*58:01
622	632	NGDMATGWLQY	0.26	HLA-A*01:01
609	618	WLQYNGSWYY	0.26	HLA-A*30:02
157	165	VVVPEPNAL	0.26	HLA-A*02:06
568	576	WVKDGDTWY	0.26	HLA-B*15:01
582	591	GAMKASQWFK	0.26	HLA-A*11:01
222	232	ATAQHQVDNLK	0.26	HLA-A*03:01
98	108	DVALVVQNAYK	0.26	HLA-A*68:01
591	598	KVSDKWYY	0.27	HLA-A*30:02
296	305	AELDKKADEL	0.27	HLA-B*44:02
591	599	KVSDKWYYV	0.27	HLA-A*68:02
268	276	KQTELEKLL	0.27	HLA-A*02:06
409	416	SEDDTAAL	0.27	HLA-B*40:01
113	120	VQNQRSKY	0.27	HLA-B*15:01
117	126	RSKYKSDAEY	0.27	HLA-B*15:01
581	589	SGAMKASQW	0.27	HLA-B*58:01
119	128	KYKSDAEYQK	0.28	HLA-A*30:01
727	735	AVNTTVDGY	0.28	HLA-A*01:01
5	14	KMILTSLASV	0.28	HLA-A*02:03
568	577	WVKDGDTWYY	0.28	HLA-B*15:01
245	253	EVIEAKLKK	0.28	HLA-A*11:01
587	596	SQWFKVSDKW	0.29	HLA-A*32:01
19	28	AGFVTSQPTF	0.29	HLA-A*23:01
340	349	AAKKAELAKK	0.29	HLA-A*30:01
549	557	LQNNGSWYY	0.29	HLA-A*01:01
730	739	TTVDGYKVNA	0.29	HLA-A*68:02
624	632	DMATGWLQY	0.29	HLA-A*01:01
623	632	GDMATGWLQY	0.29	HLA-A*30:02
28	37	FVRAEESPQV	0.29	HLA-A*68:02
108	117	KEYREVQNQR	0.29	HLA-A*33:01
164	172	ALAETKKKA	0.29	HLA-A*02:03
30	40	RAEESPQVVEK	0.29	HLA-A*11:01
419	429	KLATKKAELEK	0.3	HLA-A*30:01
683	691	GSMATGWVK	0.3	HLA-A*30:01
189	198	ATLKVALAKK	0.3	HLA-A*30:01
504	512	QPAKPEKPA	0.3	HLA-B*07:02
530	537	QENGMWYF	0.3	HLA-B*44:03
610	617	LQYNGSWY	0.3	HLA-B*15:01
100	108	ALVVQNAYK	0.3	HLA-A*03:01
274	284	KLLDSLDPEGK	0.3	HLA-A*03:01
185	193	KYDYATLKV	0.31	HLA-A*24:02
518	528	PEKPATPKTGW	0.31	HLA-B*44:02
203	212	KELEIEKLQY	0.31	HLA-A*30:02
651	659	KVNGSWYYL	0.31	HLA-A*02:06
103	110	VQNAYKEY	0.31	HLA-B*15:01
718	726	YYVNGLGAL	0.32	HLA-A*24:02
548	557	WLQNNGSWYY	0.32	HLA-A*01:01
219	228	QEVATAQHQV	0.32	HLA-A*68:02
205	212	LEIEKLQY	0.32	HLA-B*44:03
230	238	NLKKLLAGA	0.32	HLA-A*02:03
727	735	AVNTTVDGY	0.32	HLA-B*15:01
102	111	VVQNAYKEYR	0.32	HLA-A*31:01
125	133	EYQKKLTEV	0.33	HLA-B*08:01
297	305	ELDKKADEL	0.33	HLA-B*08:01
19	28	AGFVTSQPTF	0.33	HLA-A*24:02
125	133	EYQKKLTEV	0.33	HLA-A*24:02
530	537	QENGMWYF	0.33	HLA-B*44:02
85	95	KARKEAEASQK	0.33	HLA-A*30:01

330	339	DDTAALQNKL	0.33	HLA-A*68:02
624	632	DMATGWLQY	0.33	HLA-A*30:02
263	272	AELAKKQTEL	0.33	HLA-B*44:03
98	107	DVALVVQNAY	0.33	HLA-B*35:01
452	461	APAPQPEQPA	0.33	HLA-B*07:02
664	672	AMATGWAKV	0.33	HLA-A*02:01
620	629	NANGDMATGW	0.33	HLA-B*58:01
606	616	ATGWLQYNGSW	0.33	HLA-B*58:01
176	184	KAEEKVAKR	0.33	HLA-A*31:01
244	253	TEVIEAKLKK	0.33	HLA-A*68:01
180	188	KVAKRKYDY	0.34	HLA-A*32:01
1	11	MNKKKMILTSL	0.34	HLA-B*08:01
93	101	SQKLNDVAL	0.34	HLA-B*08:01
329	339	EDDTAALQNKL	0.34	HLA-A*68:02
158	166	VVPEPNALA	0.34	HLA-A*68:02
174	184	EAKAEEKVAKR	0.34	HLA-A*33:01
314	322	KEISNLEIL	0.34	HLA-B*44:03
296	305	AELDKKADEL	0.34	HLA-B*44:03
463	471	APKPEQPAP	0.34	HLA-B*07:02
566	575	TGWVKDGDTW	0.34	HLA-B*58:01
680	689	NANGSMATGW	0.34	HLA-B*58:01
581	589	SGAMKASQW	0.34	HLA-B*57:01
529	537	KQENGMWYF	0.35	HLA-A*23:01
587	596	SQWFKVSDKW	0.35	HLA-A*23:01
53	61	KADTAKKDY	0.35	HLA-A*01:01
51	61	KAKADTAKKDY	0.35	HLA-A*30:02
194	202	ALAKKEVEA	0.35	HLA-A*02:01
244	251	TEVIEAKL	0.35	HLA-B*40:01
121	129	KSDAEYQKK	0.35	HLA-A*11:01
141	151	RKEQQDLQNKF	0.35	HLA-B*15:01
582	589	GAMKASQW	0.35	HLA-B*58:01
333	342	AALQNKLAAK	0.35	HLA-A*03:01
99	107	VALVVQNAY	0.36	HLA-B*53:01
729	737	NTTVDGYKV	0.36	HLA-A*68:02
624	632	DMATGWLQY	0.36	HLA-B*35:01
338	346	KLAAKKAEL	0.36	HLA-A*02:01
37	45	VVEKSSLEK	0.36	HLA-A*03:01
647	656	TGWAKVNGSW	0.36	HLA-B*57:01
718	726	YYVNGLGAL	0.37	HLA-A*23:01
178	188	EEKVAKRKYDY	0.37	HLA-B*44:02
569	577	VKDGDTWYY	0.37	HLA-A*30:02
118	126	SKYKSDAEY	0.37	HLA-A*30:02
129	137	KLTEVDSKI	0.37	HLA-A*02:06
559	568	NANGAMATGW	0.37	HLA-B*58:01
609	618	WLQYNGSWYY	0.37	HLA-B*15:01
203	212	KELEIEKLQY	0.38	HLA-A*01:01
518	528	PEKPATPKTGW	0.38	HLA-B*44:03
327	335	DPEDDTAAL	0.38	HLA-B*35:01
194	203	ALAKKEVEAK	0.38	HLA-A*03:01
546	555	IGWLQNNGSW	0.38	HLA-B*57:01
174	183	EAKAEEKVAK	0.38	HLA-A*68:01
143	151	EQQDLQNKF	0.39	HLA-A*26:01
314	322	KEISNLEIL	0.39	HLA-B*44:02
132	140	EVDSKIEKA	0.39	HLA-A*68:02
557	565	YLNANGAMA	0.39	HLA-A*02:03
312	322	LEKEISNLEIL	0.39	HLA-B*40:01
130	139	LTEVDSKIEK	0.39	HLA-A*11:01
420	427	LATKKAEL	0.4	HLA-B*08:01
142	151	KEQQDLQNKF	0.4	HLA-B*40:01
520	528	KPATPKTGW	0.4	HLA-B*58:01
681	691	ANGSMATGWVK	0.4	HLA-A*11:01
334	342	ALQNKLAAK	0.4	HLA-A*11:01
586	595	ASQWFKVSDK	0.4	HLA-A*11:01
143	151	EQQDLQNKF	0.4	HLA-B*15:01
683	691	GSMATGWVK	0.4	HLA-A*03:01
244	253	TEVIEAKLKK	0.4	HLA-A*03:01

109	117	EYREVQNQR	0.4	HLA-A*31:01
680	689	NANGSMATGW	0.4	HLA-B*57:01
547	555	GWLQNNGSW	0.41	HLA-A*23:01
187	195	DYATLKVAL	0.41	HLA-A*24:02
205	212	LEIEKLQY	0.41	HLA-B*44:02
126	133	YQKKLTEV	0.41	HLA-B*08:01
610	618	LQYNGSWYY	0.41	HLA-A*01:01
567	577	GWVKDGDTWYY	0.41	HLA-A*01:01
568	576	WVKDGDTWY	0.41	HLA-B*35:01
117	126	RSKYKSDAEY	0.41	HLA-B*58:01
36	45	QVVEKSSLEK	0.41	HLA-A*03:01
559	568	NANGAMATGW	0.41	HLA-B*57:01
569	577	VKDGDTWYY	0.42	HLA-A*01:01
202	212	AKELEIEKLQY	0.42	HLA-A*01:01
432	441	KELDAALNEL	0.42	HLA-B*44:03
264	272	ELAKKQTEL	0.42	HLA-A*02:03
588	596	QWFKVSDKW	0.42	HLA-B*58:01
603	612	GAMATGWLQY	0.42	HLA-B*15:01
243	253	GTEVIEAKLKK	0.42	HLA-A*03:01
185	193	KYDYATLKV	0.43	HLA-A*23:01
587	596	SQWFKVSDKW	0.43	HLA-A*24:02
529	537	KQENGMWYF	0.43	HLA-A*24:02
547	555	GWLQNNGSW	0.43	HLA-A*24:02
605	612	MATGWLQY	0.43	HLA-B*35:01
132	141	EVDSKIEKAR	0.43	HLA-A*33:01
226	234	HQVDNLKKL	0.43	HLA-A*02:03
539	548	NTDGSMAIGW	0.43	HLA-B*58:01
587	595	SQWFKVSDK	0.43	HLA-A*03:01
677	685	YYLNANGSM	0.44	HLA-A*24:02
187	195	DYATLKVAL	0.44	HLA-B*08:01
548	556	WLQNNGSWY	0.44	HLA-A*01:01
338	348	KLAAKKAELAK	0.44	HLA-A*30:01
184	192	RKYDYATLK	0.44	HLA-A*30:01
334	342	ALQNKLAAK	0.44	HLA-A*30:01
529	537	KQENGMWYF	0.44	HLA-A*30:02
678	686	YLNANGSMA	0.44	HLA-A*02:03
587	596	SQWFKVSDKW	0.44	HLA-B*58:01
118	126	SKYKSDAEY	0.44	HLA-B*15:01
529	537	KQENGMWYF	0.44	HLA-B*15:01
51	59	KAKADTAKK	0.44	HLA-A*03:01
576	584	YYLEASGAM	0.45	HLA-A*24:02
66	75	KKAEDAQKKY	0.45	HLA-A*01:01
609	618	WLQYNGSWYY	0.45	HLA-A*01:01
123	130	DAEYQKKL	0.45	HLA-B*51:01
239	247	DPDDGTEVI	0.45	HLA-B*51:01
221	228	VATAQHQV	0.45	HLA-B*51:01
124	133	AEYQKKLTEV	0.45	HLA-B*40:01
250	258	KLKKGEAEL	0.45	HLA-A*02:01
546	555	IGWLQNNGSW	0.45	HLA-B*58:01
250	258	KLKKGEAEL	0.45	HLA-B*15:01
338	346	KLAAKKAEL	0.46	HLA-A*32:01
588	596	QWFKVSDKW	0.46	HLA-A*32:01
219	228	QEVATAQHQV	0.46	HLA-B*44:02
548	557	WLQNNGSWYY	0.46	HLA-A*30:02
212	221	YEISTLEQEV	0.46	HLA-B*40:01
200	210	VEAKELEIEKL	0.46	HLA-B*40:01
419	427	KLATKKAEL	0.46	HLA-A*02:01
209	217	KLQYEISTL	0.46	HLA-A*02:06
67	75	KAEDAQKKY	0.46	HLA-B*58:01
579	589	EASGAMKASQW	0.46	HLA-B*58:01
37	46	VVEKSSLEKK	0.46	HLA-A*11:01
414	423	AALQNKLATK	0.46	HLA-A*11:01
526	535	TGWKQENGMW	0.46	HLA-B*57:01
178	188	EEKVAKRKYDY	0.47	HLA-B*44:03
173	181	EEAKAEEKV	0.47	HLA-B*44:03
454	463	APQPEQPAPA	0.47	HLA-B*07:02

308	316	KVADLEKEI	0.47	HLA-A*02:06
100	107	ALVVQNAY	0.47	HLA-B*15:01
204	212	ELEIEKLQY	0.48	HLA-A*26:01
576	584	YYLEASGAM	0.48	HLA-A*23:01
610	619	LQYNGSWYYL	0.48	HLA-A*24:02
219	228	QEVATAQHQV	0.48	HLA-B*44:03
21	30	FVTSQPTFVR	0.48	HLA-A*33:01
314	321	KEISNLEI	0.48	HLA-B*40:01
587	595	SQWFKVSDK	0.48	HLA-A*11:01
13	21	SVAILGAGF	0.48	HLA-B*15:01
99	107	VALVVQNAY	0.48	HLA-B*15:01
677	685	YYLNANGSM	0.49	HLA-A*23:01
432	441	KELDAALNEL	0.49	HLA-B*44:02
568	577	WVKDGDTWYY	0.49	HLA-A*30:02
99	107	VALVVQNAY	0.49	HLA-B*58:01
647	656	TGWAKVNGSW	0.49	HLA-B*58:01
526	535	TGWKQENGMW	0.49	HLA-B*58:01
188	197	YATLKVALAK	0.49	HLA-A*11:01
604	612	AMATGWLQY	0.49	HLA-A*11:01
226	234	HQVDNLKKL	0.49	HLA-B*15:01
113	121	VQNQRSKYK	0.49	HLA-A*03:01
55	65	DTAKKDYETAK	0.49	HLA-A*68:01
308	316	KVADLEKEI	0.5	HLA-A*32:01
125	133	EYQKKLTEV	0.5	HLA-A*23:01
88	97	KEAEASQKLN	0.5	HLA-B*40:01
664	672	AMATGWAKV	0.5	HLA-A*02:06
620	629	NANGDMATGW	0.5	HLA-B*57:01
102	110	VVQNAYKEY	0.51	HLA-A*26:01
239	247	DPDDGTEVI	0.51	HLA-B*53:01
540	548	TDGSMAIGW	0.51	HLA-B*44:02
529	538	KQENGMWYFY	0.51	HLA-B*44:03
248	256	EAKLKKGEA	0.51	HLA-B*08:01
663	671	GAMATGWAK	0.51	HLA-A*30:01
100	108	ALVVQNAYK	0.51	HLA-A*11:01
429	438	KTQKELDAAL	0.51	HLA-B*15:01
567	575	GWVKDGDTW	0.52	HLA-A*23:01
122	130	SDAEYQKKL	0.52	HLA-B*44:02
173	181	EEAKAEEKV	0.52	HLA-B*44:02
124	133	AEYQKKLTEV	0.52	HLA-B*44:02
610	618	LQYNGSWYY	0.52	HLA-B*35:01
313	322	EKEISNLEIL	0.52	HLA-B*40:01
66	75	KKAEDAQKKY	0.52	HLA-A*30:02
587	596	SQWFKVSDKW	0.52	HLA-B*57:01
648	656	GWAKVNGSW	0.53	HLA-A*23:01
187	195	DYATLKVAL	0.53	HLA-A*23:01
112	120	EVQNQRSKY	0.53	HLA-A*01:01
250	258	KLKKGEAEL	0.53	HLA-B*08:01
248	258	EAKLKKGEAEL	0.53	HLA-B*08:01
105	113	NAYKEYREV	0.53	HLA-A*68:02
207	217	IEKLQYEISTL	0.53	HLA-B*40:01
582	589	GAMKASQW	0.53	HLA-B*57:01
566	575	TGWVKDGDTW	0.53	HLA-B*57:01
604	612	AMATGWLQY	0.54	HLA-A*26:01
32	40	EESPQVVEK	0.54	HLA-B*44:02
529	538	KQENGMWYFY	0.54	HLA-B*44:02
176	186	KAEEKVAKRKY	0.54	HLA-B*44:02
414	423	AALQNKLATK	0.54	HLA-A*30:01
610	619	LQYNGSWYYL	0.54	HLA-A*02:06
37	46	VVEKSSLEKK	0.54	HLA-A*03:01
250	258	KLKKGEAEL	0.55	HLA-A*32:01
648	656	GWAKVNGSW	0.55	HLA-A*24:02
264	272	ELAKKQTEL	0.55	HLA-A*68:02
30	40	RAEESPQVVEK	0.55	HLA-A*03:01
416	424	LQNKLATKK	0.55	HLA-A*03:01
539	548	NTDGSMAIGW	0.55	HLA-B*57:01
579	589	EASGAMKASQW	0.55	HLA-B*57:01

567	575	GWVKDGDTW	0.56	HLA-A*24:02
272	279	LEKLLDSL	0.56	HLA-B*40:01
37	47	VVEKSSLEKKY	0.56	HLA-A*30:02
736	743	KVNANGEW	0.56	HLA-B*58:01
608	616	GWLQYNGSW	0.57	HLA-A*23:01
198	207	KEVEAKELEI	0.57	HLA-B*44:02
117	126	RSKYKSDAEY	0.57	HLA-A*01:01
32	40	EESPQVVEK	0.57	HLA-B*44:03
562	570	GAMATGWVK	0.57	HLA-A*30:01
582	590	GAMKASQWF	0.57	HLA-B*58:01
588	596	QWFKVSDKW	0.57	HLA-B*57:01
36	45	QVVEKSSLEK	0.57	HLA-A*68:01
226	234	HQVDNLKKL	0.58	HLA-B*40:01
312	319	LEKEISNL	0.58	HLA-B*40:01
109	118	EYREVQNQRS	0.58	HLA-A*33:01
53	61	KADTAKKDY	0.58	HLA-A*30:02
463	473	APKPEQPAPAP	0.58	HLA-B*07:02
326	335	ADPEDDTAAL	0.58	HLA-B*07:02
10	19	SLASVAILGA	0.58	HLA-A*02:03
416	424	LQNKLATKK	0.58	HLA-A*11:01
566	576	TGWVKDGDTWY	0.59	HLA-A*26:01
549	557	LQNNGSWYY	0.59	HLA-A*32:01
294	302	EEAELDKKA	0.59	HLA-B*44:02
609	617	WLQYNGSWY	0.59	HLA-A*01:01
603	612	GAMATGWLQY	0.59	HLA-A*01:01
314	323	KEISNLEILL	0.59	HLA-B*44:03
461	469	APAPKPEQP	0.59	HLA-B*07:02
30	40	RAEESPQVVEK	0.59	HLA-A*30:01
340	348	AAKKAELAK	0.59	HLA-A*11:01
623	632	GDMATGWLQY	0.59	HLA-B*15:01
199	209	EVEAKELEIEK	0.59	HLA-A*68:01
586	596	ASQWFKVSDKW	0.6	HLA-A*23:01
124	133	AEYQKKLTEV	0.6	HLA-B*44:03
294	302	EEAELDKKA	0.6	HLA-B*44:03
198	207	KEVEAKELEI	0.6	HLA-B*44:03
49	57	EAKAKADTA	0.6	HLA-B*08:01
3	11	KKKMILTSL	0.6	HLA-B*08:01
649	657	WAKVNGSWY	0.6	HLA-B*35:01
87	97	RKEAEASQKLN	0.6	HLA-B*40:01
301	309	KADELQNKV	0.6	HLA-A*02:01
129	139	KLTEVDSKIEK	0.6	HLA-A*11:01
51	59	KAKADTAKK	0.6	HLA-A*11:01
582	591	GAMKASQWFK	0.6	HLA-A*03:01
13	21	SVAILGAGF	0.61	HLA-A*32:01
610	619	LQYNGSWYYL	0.61	HLA-A*23:01
219	228	QEVATAQHQV	0.61	HLA-B*40:01
93	101	SQKLNDVAL	0.61	HLA-B*40:01
530	538	QENGMWYFY	0.61	HLA-A*30:02
527	537	GWKQENGMWYF	0.62	HLA-A*23:01
588	596	QWFKVSDKW	0.62	HLA-B*53:01
568	576	WVKDGDTWY	0.62	HLA-A*01:01
529	538	KQENGMWYFY	0.62	HLA-A*01:01
430	438	TQKELDAAL	0.62	HLA-B*40:01
157	165	VVVPEPNAL	0.62	HLA-A*68:02
611	618	QYNGSWYY	0.62	HLA-A*30:02
724	732	GALAVNTTV	0.62	HLA-A*02:06
661	671	ANGAMATGWAK	0.62	HLA-A*11:01
725	735	ALAVNTTVDGY	0.62	HLA-B*15:01
663	671	GAMATGWAK	0.62	HLA-A*03:01
107	117	YKEYREVQNQR	0.62	HLA-A*31:01
119	128	KYKSDAEYQK	0.62	HLA-A*31:01
245	253	EVIEAKLKK	0.63	HLA-A*26:01
419	427	KLATKKAEL	0.63	HLA-A*32:01
646	656	ATGWAKVNGSW	0.63	HLA-A*32:01
270	279	TELEKLLDSL	0.63	HLA-B*44:02
177	185	AEEKVAKRK	0.63	HLA-B*44:02

549	556	LQNNGSWY	0.63	HLA-B*15:01
547	557	GWLQNNGSWYY	0.63	HLA-B*15:01
117	126	RSKYKSDAEY	0.63	HLA-B*57:01
314	323	KEISNLEILL	0.64	HLA-B*44:02
143	151	EQQDLQNKF	0.64	HLA-A*23:01
608	616	GWLQYNGSW	0.64	HLA-A*24:02
270	279	TELEKLLDSL	0.64	HLA-B*44:03
112	120	EVQNQRSKY	0.64	HLA-A*30:02
113	120	VQNQRSKY	0.64	HLA-A*30:02
39	47	EKSSLEKKY	0.65	HLA-B*44:02
39	47	EKSSLEKKY	0.65	HLA-B*44:03
167	175	ETKKKAEEA	0.65	HLA-B*08:01
590	599	FKVSDKWYYV	0.65	HLA-A*68:02
108	117	KEYREVQNQR	0.65	HLA-A*30:01
433	441	ELDAALNEL	0.65	HLA-A*02:01
333	342	AALQNKLAAK	0.65	HLA-A*11:01
210	217	LQYEISTL	0.65	HLA-B*15:01
567	576	GWVKDGDTWY	0.65	HLA-B*15:01
130	139	LTEVDSKIEK	0.65	HLA-A*68:01
512	520	AEEPTQPEK	0.66	HLA-B*44:02
587	596	SQWFKVSDKW	0.66	HLA-B*44:02
198	205	KEVEAKEL	0.66	HLA-B*44:03
587	596	SQWFKVSDKW	0.66	HLA-B*44:03
430	438	TQKELDAAL	0.66	HLA-B*08:01
239	247	DPDDGTEVI	0.66	HLA-B*35:01
205	214	LEIEKLQYEI	0.66	HLA-B*40:01
206	214	EIEKLQYEI	0.66	HLA-A*68:02
23	31	TSQPTFVRA	0.66	HLA-A*68:02
432	441	KELDAALNEL	0.66	HLA-A*02:01
22	30	VTSQPTFVR	0.66	HLA-A*03:01
520	528	KPATPKTGW	0.66	HLA-B*57:01
176	186	KAEEKVAKRKY	0.67	HLA-B*44:03
203	210	KELEIEKL	0.67	HLA-B*44:03
540	548	TDGSMAIGW	0.67	HLA-B*44:03
122	130	SDAEYQKKL	0.67	HLA-B*44:03
568	578	WVKDGDTWYYL	0.67	HLA-A*01:01
65	75	KKKAEDAQKKY	0.67	HLA-A*01:01
157	165	VVVPEPNAL	0.67	HLA-B*35:01
173	181	EEAKAEEKV	0.67	HLA-B*40:01
499	507	APKPEQPAK	0.67	HLA-B*07:02
577	586	YLEASGAMKA	0.67	HLA-A*02:03
415	424	ALQNKLATKK	0.67	HLA-A*11:01
36	46	QVVEKSSLEKK	0.67	HLA-A*11:01
264	272	ELAKKQTEL	0.68	HLA-A*26:01
177	187	AEEKVAKRKYD	0.68	HLA-B*44:02
521	528	PATPKTGW	0.68	HLA-B*53:01
527	537	GWKQENGMWYF	0.68	HLA-A*24:02
100	110	ALVVQNAYKEY	0.68	HLA-A*30:02
648	657	GWAKVNGSWY	0.68	HLA-A*30:02
95	102	KLNDVALV	0.68	HLA-A*02:03
111	120	REVQNQRSKY	0.68	HLA-B*15:01
198	205	KEVEAKEL	0.69	HLA-B*44:02
139	147	KARKEQQDL	0.69	HLA-B*07:02
651	659	KVNGSWYYL	0.69	HLA-A*30:02
725	735	ALAVNTTVDGY	0.69	HLA-A*30:02
245	253	EVIEAKLKK	0.69	HLA-A*33:01
610	618	LQYNGSWYY	0.69	HLA-A*11:01
727	735	AVNTTVDGY	0.69	HLA-A*11:01
188	197	YATLKVALAK	0.69	HLA-A*03:01
129	137	KLTEVDSKI	0.7	HLA-A*32:01
581	589	SGAMKASQW	0.7	HLA-B*53:01
649	658	WAKVNGSWYY	0.7	HLA-B*35:01
91	99	EASQKLNDV	0.7	HLA-A*68:02
6	14	MILTSLASV	0.7	HLA-A*68:02
433	441	ELDAALNEL	0.7	HLA-A*02:06
167	176	ETKKKAEEAK	0.7	HLA-A*68:01

203	213	KELEIEKLQYE	0.71	HLA-B*44:03
549	557	LQNNGSWYY	0.71	HLA-B*35:01
167	175	ETKKKAEEA	0.71	HLA-A*68:02
651	659	KVNGSWYYL	0.71	HLA-A*02:01
113	121	VQNQRSKYK	0.71	HLA-A*11:01
224	232	AQHQVDNLK	0.71	HLA-A*11:01
157	165	VVVPEPNAL	0.72	HLA-A*32:01
185	195	KYDYATLKVAL	0.72	HLA-A*24:02
623	632	GDMATGWLQY	0.72	HLA-A*01:01
568	576	WVKDGDTWY	0.72	HLA-A*30:02
416	424	LQNKLATKK	0.72	HLA-A*30:01
308	316	KVADLEKEI	0.72	HLA-A*02:03
190	198	TLKVALAKK	0.72	HLA-A*11:01
112	121	EVQNQRSKYK	0.72	HLA-A*68:01
49	58	EAKAKADTAK	0.72	HLA-A*68:01
335	343	LQNKLAAKK	0.73	HLA-A*03:01
610	618	LQYNGSWYY	0.74	HLA-A*26:01
143	151	EQQDLQNKF	0.74	HLA-A*24:02
465	473	KPEQPAPAP	0.74	HLA-B*07:02
586	595	ASQWFKVSDK	0.74	HLA-A*30:01
587	595	SQWFKVSDK	0.74	HLA-A*30:01
150	158	KFNEVRAVV	0.74	HLA-A*30:01
37	46	VVEKSSLEKK	0.74	HLA-A*30:01
99	107	VALVVQNAY	0.74	HLA-B*57:01
202	212	AKELEIEKLQY	0.75	HLA-B*44:03
586	596	ASQWFKVSDKW	0.75	HLA-A*24:02
67	75	KAEDAQKKY	0.75	HLA-B*35:01
188	195	YATLKVAL	0.75	HLA-B*51:01
119	126	KYKSDAEY	0.75	HLA-A*30:02
119	129	KYKSDAEYQKK	0.75	HLA-A*30:01
430	438	TQKELDAAL	0.75	HLA-A*02:06
340	348	AAKKAELAK	0.75	HLA-A*03:01
568	575	WVKDGDTW	0.76	HLA-B*53:01
176	186	KAEEKVAKRKY	0.76	HLA-A*01:01
719	727	YVNGLGALA	0.76	HLA-A*68:02
5	14	KMILTSLASV	0.76	HLA-A*02:01
610	618	LQYNGSWYY	0.76	HLA-A*03:01
147	155	LQNKFNEVR	0.76	HLA-A*31:01
111	119	REVQNQRSK	0.77	HLA-B*44:02
90	99	AEASQKLNDV	0.77	HLA-B*44:02
512	520	AEEPTQPEK	0.77	HLA-B*44:03
177	185	AEEKVAKRK	0.77	HLA-B*44:03
52	61	AKADTAKKDY	0.77	HLA-A*01:01
588	598	QWFKVSDKWYY	0.77	HLA-A*30:02
722	732	GLGALAVNTTV	0.77	HLA-A*02:01
93	102	SQKLNDVALV	0.77	HLA-A*02:06
189	197	ATLKVALAK	0.77	HLA-A*31:01
607	616	TGWLQYNGSW	0.77	HLA-B*57:01
18	28	GAGFVTSQPTF	0.78	HLA-A*23:01
268	276	KQTELEKLL	0.78	HLA-B*40:01
644	652	DMATGWAKV	0.78	HLA-A*68:02
157	165	VVVPEPNAL	0.78	HLA-B*07:02
648	658	GWAKVNGSWYY	0.78	HLA-A*30:02
109	119	EYREVQNQRSK	0.78	HLA-A*33:01
257	265	ELNAKQAEL	0.78	HLA-A*02:03
224	233	AQHQVDNLKK	0.78	HLA-A*11:01
516	525	TQPEKPATPK	0.78	HLA-A*11:01
520	528	KPATPKTGW	0.79	HLA-B*44:02
203	210	KELEIEKL	0.79	HLA-B*44:02
157	165	VVVPEPNAL	0.79	HLA-A*26:01
102	110	VVQNAYKEY	0.79	HLA-A*01:01
110	120	YREVQNQRSKY	0.79	HLA-A*30:02
194	202	ALAKKEVEA	0.79	HLA-A*02:06
20	30	GFVTSQPTFVR	0.79	HLA-A*31:01
242	250	DGTEVIEAK	0.79	HLA-A*68:01
101	110	LVVQNAYKEY	0.8	HLA-A*26:01

315	323	EISNLEILL	0.8	HLA-A*26:01
556	564	YYLNANGAM	0.8	HLA-A*24:02
143	151	EQQDLQNKF	0.8	HLA-B*40:01
418	427	NKLATKKAEL	0.8	HLA-B*08:01
582	591	GAMKASQWFK	0.8	HLA-A*30:01
548	557	WLQNNGSWYY	0.8	HLA-B*15:01
177	187	AEEKVAKRKYD	0.81	HLA-B*44:03
263	272	AELAKKQTEL	0.81	HLA-A*02:03
624	632	DMATGWLQY	0.81	HLA-B*15:01
224	233	AQHQVDNLKK	0.81	HLA-A*03:01
415	423	ALQNKLATK	0.81	HLA-A*31:01
131	139	TEVDSKIEK	0.82	HLA-B*44:02
546	555	IGWLQNNGSW	0.82	HLA-A*23:01
131	139	TEVDSKIEK	0.82	HLA-B*44:03
546	555	IGWLQNNGSW	0.82	HLA-A*24:02
31	38	AEESPQVV	0.82	HLA-B*40:01
603	612	GAMATGWLQY	0.82	HLA-B*35:01
220	229	EVATAQHQVD	0.82	HLA-A*68:02
463	470	APKPEQPA	0.82	HLA-B*07:02
14	22	VAILGAGFV	0.82	HLA-B*51:01
176	186	KAEEKVAKRKY	0.82	HLA-A*30:02
588	597	QWFKVSDKWY	0.82	HLA-A*30:02
610	617	LQYNGSWY	0.82	HLA-A*30:02
224	232	AQHQVDNLK	0.82	HLA-A*30:01
158	166	VVPEPNALA	0.82	HLA-A*02:06
335	343	LQNKLAAKK	0.82	HLA-A*11:01
425	433	AELEKTQKE	0.83	HLA-B*44:02
303	312	DELQNKVADL	0.83	HLA-B*40:01
312	321	LEKEISNLEI	0.83	HLA-B*40:01
200	207	VEAKELEI	0.83	HLA-B*40:01
203	212	KELEIEKLQY	0.83	HLA-B*40:01
89	96	EAEASQKL	0.83	HLA-B*51:01
174	181	EAKAEEKV	0.83	HLA-B*51:01
567	576	GWVKDGDTWY	0.83	HLA-A*30:02
20	30	GFVTSQPTFVR	0.83	HLA-A*11:01
186	195	YDYATLKVAL	0.84	HLA-A*24:02
8	16	LTSLASVAI	0.84	HLA-A*68:02
201	210	EAKELEIEKL	0.84	HLA-A*68:02
274	284	KLLDSLDPEGK	0.84	HLA-A*11:01
209	217	KLQYEISTL	0.84	HLA-B*15:01
180	188	KVAKRKYDY	0.84	HLA-B*15:01
586	595	ASQWFKVSDK	0.84	HLA-A*03:01
174	184	EAKAEEKVAKR	0.84	HLA-A*68:01
586	596	ASQWFKVSDKW	0.85	HLA-A*32:01
600	609	NSNGAMATGW	0.85	HLA-B*53:01
537	546	FYNTDGSMAI	0.85	HLA-A*24:02
204	212	ELEIEKLQY	0.85	HLA-B*35:01
520	528	KPATPKTGW	0.85	HLA-B*35:01
454	462	APQPEQPAP	0.85	HLA-B*07:02
222	232	ATAQHQVDNLK	0.85	HLA-A*30:01
562	570	GAMATGWVK	0.85	HLA-A*03:01
580	590	ASGAMKASQWF	0.85	HLA-B*57:01
120	129	YKSDAEYQKK	0.86	HLA-A*01:01
99	107	VALVVQNAY	0.86	HLA-A*01:01
530	538	QENGMWYFY	0.86	HLA-A*01:01
590	598	FKVSDKWYY	0.86	HLA-A*30:02
645	652	MATGWAKV	0.86	HLA-B*51:01
726	735	LAVNTTVDGY	0.86	HLA-A*30:02
103	110	VQNAYKEY	0.86	HLA-A*30:02
121	129	KSDAEYQKK	0.86	HLA-A*03:01
568	577	WVKDGDTWYY	0.87	HLA-B*35:01
105	113	NAYKEYREV	0.87	HLA-B*08:01
257	265	ELNAKQAEL	0.87	HLA-A*68:02
91	99	EASQKLNDV	0.87	HLA-B*51:01
220	228	EVATAQHQV	0.87	HLA-A*02:06
41	51	SSLEKKYEEAK	0.87	HLA-A*11:01

51	61	KAKADTAKKDY	0.87	HLA-B*15:01
189	197	ATLKVALAK	0.87	HLA-A*68:01
36	46	QVVEKSSLEKK	0.87	HLA-A*68:01
566	575	TGWVKDGDTW	0.88	HLA-B*53:01
111	119	REVQNQRSK	0.88	HLA-B*44:03
649	658	WAKVNGSWYY	0.88	HLA-A*01:01
157	165	VVVPEPNAL	0.88	HLA-A*02:03
157	165	VVVPEPNAL	0.88	HLA-B*15:01
143	151	EQQDLQNKF	0.89	HLA-B*53:01
726	735	LAVNTTVDGY	0.89	HLA-A*01:01
432	442	KELDAALNELG	0.89	HLA-B*40:01
92	101	ASQKLNDVAL	0.89	HLA-B*15:01
425	433	AELEKTQKE	0.9	HLA-B*44:03
610	618	LQYNGSWYY	0.9	HLA-B*44:03
219	227	QEVATAQHQ	0.9	HLA-B*44:03
426	434	ELEKTQKEL	0.9	HLA-B*08:01
725	732	ALAVNTTV	0.9	HLA-A*02:03
560	570	ANGAMATGWVK	0.9	HLA-A*11:01
293	302	AEEAELDKKA	0.91	HLA-B*44:02
556	564	YYLNANGAM	0.91	HLA-A*23:01
122	130	SDAEYQKKL	0.91	HLA-B*40:01
450	458	TPAPAPQPE	0.91	HLA-B*35:01
116	126	QRSKYKSDAEY	0.91	HLA-A*30:02
649	658	WAKVNGSWYY	0.91	HLA-A*30:02
301	309	KADELQNKV	0.91	HLA-B*51:01
103	111	VQNAYKEYR	0.91	HLA-A*33:01
249	258	AKLKKGEAEL	0.91	HLA-A*02:03
245	253	EVIEAKLKK	0.91	HLA-A*03:01
581	589	SGAMKASQW	0.92	HLA-A*32:01
313	322	EKEISNLEIL	0.92	HLA-B*44:03
124	132	AEYQKKLTE	0.92	HLA-B*44:03
18	28	GAGFVTSQPTF	0.92	HLA-A*24:02
204	212	ELEIEKLQY	0.92	HLA-A*30:02
187	197	DYATLKVALAK	0.92	HLA-A*33:01
102	111	VVQNAYKEYR	0.92	HLA-A*33:01
192	200	KVALAKKEV	0.92	HLA-A*30:01
570	577	KDGDTWYY	0.93	HLA-A*01:01
172	181	AEEAKAEEKV	0.93	HLA-B*40:01
181	191	VAKRKYDYATL	0.93	HLA-B*08:01
101	110	LVVQNAYKEY	0.93	HLA-A*30:02
111	121	REVQNQRSKYK	0.93	HLA-A*30:01
610	619	LQYNGSWYYL	0.93	HLA-A*02:01
414	424	AALQNKLATKK	0.93	HLA-A*11:01
421	429	ATKKAELEK	0.93	HLA-A*31:01
736	743	KVNANGEW	0.93	HLA-B*57:01
226	234	HQVDNLKKL	0.94	HLA-B*44:02
179	188	EKVAKRKYDY	0.94	HLA-A*26:01
142	151	KEQQDLQNKF	0.94	HLA-A*32:01
226	234	HQVDNLKKL	0.94	HLA-B*44:03
301	309	KADELQNKV	0.94	HLA-A*01:01
651	659	KVNGSWYYL	0.94	HLA-B*58:01
335	343	LQNKLAAKK	0.94	HLA-A*30:01
333	342	AALQNKLAAK	0.94	HLA-A*30:01
5	14	KMILTSLASV	0.94	HLA-A*02:06
413	423	TAALQNKLATK	0.94	HLA-A*03:01
548	556	WLQNNGSWY	0.94	HLA-B*15:01
124	132	AEYQKKLTE	0.95	HLA-B*44:02
254	263	GEAELNAKQA	0.95	HLA-B*44:02
449	457	ETPAPAPQP	0.95	HLA-A*68:02
331	340	DTAALQNKLA	0.95	HLA-A*68:02
610	619	LQYNGSWYYL	0.95	HLA-A*30:02
527	536	GWKQENGMWY	0.95	HLA-A*30:02
95	102	KLNDVALV	0.95	HLA-A*02:01
226	234	HQVDNLKKL	0.95	HLA-A*02:01
587	597	SQWFKVSDKWY	0.95	HLA-B*15:01
202	212	AKELEIEKLQY	0.96	HLA-B*44:02

141	151	RKEQQDLQNKF	0.96	HLA-B*44:02
608	618	GWLQYNGSWYY	0.96	HLA-A*32:01
110	120	YREVQNQRSKY	0.96	HLA-A*01:01
89	96	EAEASQKL	0.96	HLA-B*40:01
604	612	AMATGWLQY	0.96	HLA-B*35:01
63	71	TAKKKAEDA	0.96	HLA-B*08:01
461	471	APAPKPEQPAP	0.96	HLA-B*07:02
591	599	KVSDKWYYV	0.96	HLA-A*30:02
581	591	SGAMKASQWFK	0.96	HLA-A*03:01
36	46	QVVEKSSLEKK	0.96	HLA-A*03:01
67	75	KAEDAQKKY	0.96	HLA-B*57:01
651	659	KVNGSWYYL	0.97	HLA-A*24:02
726	735	LAVNTTVDGY	0.97	HLA-B*35:01
93	103	SQKLNDVALVV	0.97	HLA-A*02:01
203	213	KELEIEKLQYE	0.98	HLA-B*44:02
649	656	WAKVNGSW	0.98	HLA-B*53:01
254	263	GEAELNAKQA	0.98	HLA-B*44:03
293	302	AEEAELDKKA	0.98	HLA-B*44:03
427	434	LEKTQKEL	0.98	HLA-B*40:01
97	107	NDVALVVQNAY	0.98	HLA-B*35:01
587	597	SQWFKVSDKWY	0.98	HLA-A*30:02
98	106	DVALVVQNA	0.99	HLA-A*26:01
203	211	KELEIEKLQ	0.99	HLA-B*44:03
734	743	GYKVNANGEW	0.99	HLA-A*24:02
431	441	QKELDAALNEL	0.99	HLA-B*40:01
143	151	EQQDLQNKF	0.99	HLA-B*35:01
157	166	VVVPEPNALA	0.99	HLA-A*68:02
602	612	NGAMATGWLQY	0.99	HLA-A*30:02
65	75	KKKAEDAQKKY	0.99	HLA-A*30:02
331	339	DTAALQNKL	0.99	HLA-B*51:01
580	590	ASGAMKASQWF	0.99	HLA-B*58:01
93	103	SQKLNDVALVV	0.99	HLA-A*02:03
529	537	KQENGMWYF	0.99	HLA-A*02:06
651	659	KVNGSWYYL	1.0	HLA-A*23:01
102	110	VVQNAYKEY	1.0	HLA-B*35:01
181	189	VAKRKYDYA	1.0	HLA-B*08:01
452	460	APAPQPEQP	1.0	HLA-B*07:02
30	38	RAEESPQVV	1.0	HLA-B*51:01
566	576	TGWVKDGDTWY	1.0	HLA-A*30:02
146	154	DLQNKFNEV	1.0	HLA-A*02:03
590	599	FKVSDKWYYV	1.0	HLA-A*02:06

Table XVIII – Results of the PspA-MHC-II binding prediction.
Acquired with TepiTool (http://tools.iedb.org/tepitool/).

PspA MHC-II				
PEPTIDE START	PEPTIDE END	PEPTIDE	PERCENTILE RANK	ALLELE
14	28	VAILGAGFVTSQPTF	5.95	HLA-DPA1*01/DPB1*04:01
580	594	ASGAMKASQWFKVSD	9.05	HLA-DPA1*01/DPB1*04:01
712	726	KVSDKWYYVNGLGAL	6.00	HLA-DPA1*01:03/DPB1*02:01
606	620	ATGWLQYNGSWYYLN	9.30	HLA-DPA1*01:03/DPB1*02:01
182	196	AKRKYDYATLKVALA	9.40	HLA-DPA1*01:03/DPB1*02:01
3	17	KKKMILTSLASVAIL	9.90	HLA-DPA1*01:03/DPB1*02:01
207	221	IEKLQYEISTLEQEV	3.80	HLA-DPA1*02:01/DPB1*01:01
3	17	KKKMILTSLASVAIL	5.90	HLA-DPA1*02:01/DPB1*01:01
182	196	AKRKYDYATLKVALA	6.50	HLA-DPA1*02:01/DPB1*01:01
310	324	ADLEKEISNLEILLG	8.80	HLA-DPA1*02:01/DPB1*01:01
188	202	YATLKVALAKKEVEA	4.30	HLA-DPA1*02:01/DPB1*05:01
182	196	AKRKYDYATLKVALA	4.70	HLA-DPA1*02:01/DPB1*05:01
3	17	KKKMILTSLASVAIL	5.50	HLA-DPA1*02:01/DPB1*05:01
585	599	KASQWFKVSDKWYYV	5.80	HLA-DPA1*02:01/DPB1*05:01
263	277	AELAKKQTELEKLLD	7.10	HLA-DPA1*02:01/DPB1*05:01

201	215	EAKELEIEKLQYEIS	8.90	HLA-DPA1*02:01/DPB1*05:01
580	594	ASGAMKASQWFKVSD	9.60	HLA-DPA1*02:01/DPB1*05:01
3	17	KKKMILTSLASVAIL	2.10	HLA-DPA1*03:01/DPB1*04:02
310	324	ADLEKEISNLEILLG	6.70	HLA-DPA1*03:01/DPB1*04:02
207	221	IEKLQYEISTLEQEV	8.60	HLA-DPA1*03:01/DPB1*04:02
570	584	KDGDTWYYLEASGAM	4.40	HLA-DQA1*01:01/DQB1*05:01
268	282	KQTELEKLLDSLDPE	6.10	HLA-DQA1*01:01/DQB1*05:01
611	625	QYNGSWYYLNANGDM	7.10	HLA-DQA1*01:01/DQB1*05:01
530	544	QENGMWYFYNTDGSM	9.80	HLA-DQA1*01:01/DQB1*05:01
8	22	LTSLASVAILGAGFV	0.85	HLA-DQA1*01:02/DQB1*06:02
575	589	WYYLEASGAMKASQW	2.40	HLA-DQA1*01:02/DQB1*06:02
94	108	QKLNDVALVVQNAYK	3.20	HLA-DQA1*01:02/DQB1*06:02
656	670	WYYLNANGAMATGWA	3.30	HLA-DQA1*01:02/DQB1*06:02
3	17	KKKMILTSLASVAIL	4.20	HLA-DQA1*01:02/DQB1*06:02
556	570	YYLNANGAMATGWVK	5.60	HLA-DQA1*01:02/DQB1*06:02
310	324	ADLEKEISNLEILLG	6.30	HLA-DQA1*01:02/DQB1*06:02
147	161	LQNKFNEVRAVVVPE	6.70	HLA-DQA1*01:02/DQB1*06:02
597	611	YYVNSNGAMATGWLQ	7.20	HLA-DQA1*01:02/DQB1*06:02
315	329	EISNLEILLGGADPE	7.50	HLA-DQA1*01:02/DQB1*06:02
396	410	EISNLEILLGGADSE	7.50	HLA-DQA1*01:02/DQB1*06:02
717	731	WYYVNGLGALAVNTT	7.60	HLA-DQA1*01:02/DQB1*06:02
677	691	YYLNANGSMATGWVK	9.30	HLA-DQA1*01:02/DQB1*06:02
207	221	IEKLQYEISTLEQEV	2.00	HLA-DQA1*03:01/DQB1*03:02
147	161	LQNKFNEVRAVVVPE	2.30	HLA-DQA1*03:01/DQB1*03:02
444	458	DGDEEETPAPAPQPE	5.00	HLA-DQA1*03:01/DQB1*03:02
153	167	EVRAVVVPEPNALAE	5.00	HLA-DQA1*03:01/DQB1*03:02
570	584	KDGDTWYYLEASGAM	5.50	HLA-DQA1*03:01/DQB1*03:02
212	226	YEISTLEQEVATAQH	6.40	HLA-DQA1*03:01/DQB1*03:02
237	251	GADPDDGTEVIEAKL	6.50	HLA-DQA1*03:01/DQB1*03:02
315	329	EISNLEILLGGADPE	7.20	HLA-DQA1*03:01/DQB1*03:02
430	444	TQKELDAALNELGPD	9.40	HLA-DQA1*03:01/DQB1*03:02
430	444	TQKELDAALNELGPD	3.30	HLA-DQA1*04:01/DQB1*04:02
207	221	IEKLQYEISTLEQEV	3.90	HLA-DQA1*04:01/DQB1*04:02
285	299	TQDELDKEAEEAELD	4.90	HLA-DQA1*04:01/DQB1*04:02
147	161	LQNKFNEVRAVVVPE	5.10	HLA-DQA1*04:01/DQB1*04:02
252	266	KKGEAELNAKQAELA	7.20	HLA-DQA1*04:01/DQB1*04:02
237	251	GADPDDGTEVIEAKL	8.70	HLA-DQA1*04:01/DQB1*04:02
401	415	EILLGGADSEDDTAA	9.40	HLA-DQA1*04:01/DQB1*04:02
207	221	IEKLQYEISTLEQEV	3.90	HLA-DQA1*05:01/DQB1*02:01
717	731	WYYVNGLGALAVNTT	1.80	HLA-DQA1*05:01/DQB1*03:01
14	28	VAILGAGFVTSQPTF	3.50	HLA-DQA1*05:01/DQB1*03:01
8	22	LTSLASVAILGAGFV	3.80	HLA-DQA1*05:01/DQB1*03:01
656	670	WYYLNANGAMATGWA	4.00	HLA-DQA1*05:01/DQB1*03:01
575	589	WYYLEASGAMKASQW	4.30	HLA-DQA1*05:01/DQB1*03:01
556	570	YYLNANGAMATGWVK	4.80	HLA-DQA1*05:01/DQB1*03:01
661	675	ANGAMATGWAKVNGS	4.90	HLA-DQA1*05:01/DQB1*03:01
642	656	NGDMATGWAKVNGSW	6.00	HLA-DQA1*05:01/DQB1*03:01
597	611	YYVNSNGAMATGWLQ	8.40	HLA-DQA1*05:01/DQB1*03:01
3	17	KKKMILTSLASVAIL	8.50	HLA-DQA1*05:01/DQB1*03:01
677	691	YYLNANGSMATGWVK	8.80	HLA-DQA1*05:01/DQB1*03:01
717	731	WYYVNGLGALAVNTT	0.13	HLA-DRB1*01:01
3	17	KKKMILTSLASVAIL	0.56	HLA-DRB1*01:01
551	565	NNGSWYYLNANGAMA	1.80	HLA-DRB1*01:01
182	196	AKRKYDYATLKVALA	2.00	HLA-DRB1*01:01
672	686	VNGSWYYLNANGSMA	2.20	HLA-DRB1*01:01
656	670	WYYLNANGAMATGWA	2.50	HLA-DRB1*01:01
651	665	KVNGSWYYLNANGAM	2.90	HLA-DRB1*01:01
8	22	LTSLASVAILGAGFV	2.90	HLA-DRB1*01:01
19	33	AGFVTSQPTFVRAEE	3.30	HLA-DRB1*01:01
188	202	YATLKVALAKKEVEA	4.00	HLA-DRB1*01:01
147	161	LQNKFNEVRAVVVPE	5.00	HLA-DRB1*01:01
556	570	YYLNANGAMATGWVK	5.80	HLA-DRB1*01:01
575	589	WYYLEASGAMKASQW	6.00	HLA-DRB1*01:01
570	584	KDGDTWYYLEASGAM	6.10	HLA-DRB1*01:01
592	606	VSDKWYYVNSNGAMA	7.80	HLA-DRB1*01:01
677	691	YYLNANGSMATGWVK	8.40	HLA-DRB1*01:01

712	726	KVSDKWYYVNGLGAL	8.60	HLA-DRB1*01:01
3	17	KKKMILTSLASVAIL	6.40	HLA-DRB1*03:01
153	167	EVRAVVVPEPNALAE	1.10	HLA-DRB1*04:01
672	686	VNGSWYYLNANGSMA	2.00	HLA-DRB1*04:01
535	549	WYFYNTDGSMAIGWL	2.20	HLA-DRB1*04:01
551	565	NNGSWYYLNANGAMA	3.10	HLA-DRB1*04:01
592	606	VSDKWYYVNSNGAMA	3.90	HLA-DRB1*04:01
651	665	KVNGSWYYLNANGAM	4.60	HLA-DRB1*04:01
656	670	WYYLNANGAMATGWA	4.60	HLA-DRB1*04:01
182	196	AKRKYDYATLKVALA	4.80	HLA-DRB1*04:01
545	559	AIGWLQNNGSWYYLN	5.60	HLA-DRB1*04:01
540	554	TDGSMAIGWLQNNGS	6.10	HLA-DRB1*04:01
570	584	KDGDTWYYLEASGAM	6.50	HLA-DRB1*04:01
575	589	WYYLEASGAMKASQW	6.50	HLA-DRB1*04:01
25	39	QPTFVRAEESPQVVE	7.90	HLA-DRB1*04:01
556	570	YYLNANGAMATGWVK	9.50	HLA-DRB1*04:01
677	691	YYLNANGSMATGWVK	9.50	HLA-DRB1*04:01
611	625	QYNGSWYYLNANGDM	3.40	HLA-DRB1*04:05
268	282	KQTELEKLLDSLDPE	4.20	HLA-DRB1*04:05
672	686	VNGSWYYLNANGSMA	4.20	HLA-DRB1*04:05
551	565	NNGSWYYLNANGAMA	4.60	HLA-DRB1*04:05
106	120	AYKEYREVQNQRSKY	5.40	HLA-DRB1*04:05
19	33	AGFVTSQPTFVRAEE	7.20	HLA-DRB1*04:05
651	665	KVNGSWYYLNANGAM	7.20	HLA-DRB1*04:05
592	606	VSDKWYYVNSNGAMA	7.40	HLA-DRB1*04:05
182	196	AKRKYDYATLKVALA	8.80	HLA-DRB1*04:05
570	584	KDGDTWYYLEASGAM	9.30	HLA-DRB1*04:05
147	161	LQNKFNEVRAVVVPE	1.20	HLA-DRB1*07:01
3	17	KKKMILTSLASVAIL	1.50	HLA-DRB1*07:01
19	33	AGFVTSQPTFVRAEE	1.80	HLA-DRB1*07:01
8	22	LTSLASVAILGAGFV	4.50	HLA-DRB1*07:01
153	167	EVRAVVVPEPNALAE	6.40	HLA-DRB1*08:02
19	33	AGFVTSQPTFVRAEE	0.22	HLA-DRB1*09:01
717	731	WYYVNGLGALAVNTT	0.30	HLA-DRB1*09:01
551	565	NNGSWYYLNANGAMA	0.64	HLA-DRB1*09:01
592	606	VSDKWYYVNSNGAMA	0.65	HLA-DRB1*09:01
575	589	WYYLEASGAMKASQW	1.10	HLA-DRB1*09:01
3	17	KKKMILTSLASVAIL	1.20	HLA-DRB1*09:01
651	665	KVNGSWYYLNANGAM	1.60	HLA-DRB1*09:01
656	670	WYYLNANGAMATGWA	1.70	HLA-DRB1*09:01
672	686	VNGSWYYLNANGSMA	1.90	HLA-DRB1*09:01
712	726	KVSDKWYYVNGLGAL	2.20	HLA-DRB1*09:01
580	594	ASGAMKASQWFKVSD	2.50	HLA-DRB1*09:01
597	611	YYVNSNGAMATGWLQ	3.10	HLA-DRB1*09:01
182	196	AKRKYDYATLKVALA	3.90	HLA-DRB1*09:01
556	570	YYLNANGAMATGWVK	4.90	HLA-DRB1*09:01
25	39	QPTFVRAEESPQVVE	7.40	HLA-DRB1*09:01
677	691	YYLNANGSMATGWVK	8.90	HLA-DRB1*09:01
606	620	ATGWLQYNGSWYYLN	9.60	HLA-DRB1*09:01
666	680	ATGWAKVNGSWYYLN	9.70	HLA-DRB1*09:01
182	196	AKRKYDYATLKVALA	5.70	HLA-DRB1*11:01
188	202	YATLKVALAKKEVEA	5.90	HLA-DRB1*11:01
672	686	VNGSWYYLNANGSMA	6.90	HLA-DRB1*11:01
225	239	QHQVDNLKKLLAGAD	8.10	HLA-DRB1*11:01
3	17	KKKMILTSLASVAIL	1.20	HLA-DRB1*12:01
545	559	AIGWLQNNGSWYYLN	7.55	HLA-DRB1*12:01
717	731	WYYVNGLGALAVNTT	7.90	HLA-DRB1*12:01
585	599	KASQWFKVSDKWYYV	9.75	HLA-DRB1*12:01
99	113	VALVVQNAYKEYREV	9.80	HLA-DRB1*12:01
545	559	AIGWLQNNGSWYYLN	4.70	HLA-DRB1*13:02
672	686	VNGSWYYLNANGSMA	6.00	HLA-DRB1*13:02
551	565	NNGSWYYLNANGAMA	6.70	HLA-DRB1*13:02
592	606	VSDKWYYVNSNGAMA	6.70	HLA-DRB1*13:02
3	17	KKKMILTSLASVAIL	7.40	HLA-DRB1*13:02
147	161	LQNKFNEVRAVVVPE	7.50	HLA-DRB1*13:02
89	103	EAEASQKLNDVALVV	7.70	HLA-DRB1*13:02

656	670	WYYLNANGAMATGWA	8.50	HLA-DRB1*13:02
19	33	AGFVTSQPTFVRAEE	9.20	HLA-DRB1*13:02
606	620	ATGWLQYNGSWYYLN	9.70	HLA-DRB1*13:02
556	570	YYLNANGAMATGWVK	9.90	HLA-DRB1*13:02
3	17	KKKMILTSLASVAIL	5.00	HLA-DRB1*15:01
606	620	ATGWLQYNGSWYYLN	5.70	HLA-DRB1*15:01
535	549	WYFYNTDGSMAIGWL	0.35	HLA-DRB3*01:01
551	565	NNGSWYYLNANGAMA	1.50	HLA-DRB3*01:01
651	665	KVNGSWYYLNANGAM	2.10	HLA-DRB3*01:01
565	579	ATGWVKDGDTWYYLE	2.80	HLA-DRB3*01:01
606	620	ATGWLQYNGSWYYLN	3.80	HLA-DRB3*01:01
545	559	AIGWLQNNGSWYYLN	4.40	HLA-DRB3*01:01
656	670	WYYLNANGAMATGWA	4.70	HLA-DRB3*01:01
114	128	QNQRSKYKSDAEYQK	4.80	HLA-DRB3*01:01
585	599	KASQWFKVSDKWYYV	5.40	HLA-DRB3*01:01
25	39	QPTFVRAEESPQVVE	5.60	HLA-DRB3*01:01
89	103	EAEASQKLNDVALVV	6.80	HLA-DRB3*01:01
19	33	AGFVTSQPTFVRAEE	7.30	HLA-DRB3*01:01
592	606	VSDKWYYVNSNGAMA	7.60	HLA-DRB3*01:01
730	744	TTVDGYKVNANGEWV	8.00	HLA-DRB3*01:01
712	726	KVSDKWYYVNGLGAL	8.70	HLA-DRB3*01:01
570	584	KDGDTWYYLEASGAM	8.90	HLA-DRB3*01:01
672	686	VNGSWYYLNANGSMA	9.30	HLA-DRB3*01:01
525	539	KTGWKQENGMWYFYN	9.60	HLA-DRB3*01:01
656	670	WYYLNANGAMATGWA	0.09	HLA-DRB3*02:02
592	606	VSDKWYYVNSNGAMA	0.11	HLA-DRB3*02:02
551	565	NNGSWYYLNANGAMA	0.12	HLA-DRB3*02:02
672	686	VNGSWYYLNANGSMA	0.21	HLA-DRB3*02:02
556	570	YYLNANGAMATGWVK	0.42	HLA-DRB3*02:02
677	691	YYLNANGSMATGWVK	0.52	HLA-DRB3*02:02
597	611	YYVNSNGAMATGWLQ	0.75	HLA-DRB3*02:02
651	665	KVNGSWYYLNANGAM	0.80	HLA-DRB3*02:02
545	559	AIGWLQNNGSWYYLN	1.40	HLA-DRB3*02:02
636	650	WYYLNANGDMATGWA	1.40	HLA-DRB3*02:02
616	630	WYYLNANGDMATGWL	1.40	HLA-DRB3*02:02
19	33	AGFVTSQPTFVRAEE	1.60	HLA-DRB3*02:02
535	549	WYFYNTDGSMAIGWL	1.80	HLA-DRB3*02:02
3	17	KKKMILTSLASVAIL	2.10	HLA-DRB3*02:02
717	731	WYYVNGLGALAVNTT	3.20	HLA-DRB3*02:02
723	737	LGALAVNTTVDGYKV	3.30	HLA-DRB3*02:02
611	625	QYNGSWYYLNANGDM	3.90	HLA-DRB3*02:02
575	589	WYYLEASGAMKASQW	4.00	HLA-DRB3*02:02
712	726	KVSDKWYYVNGLGAL	4.70	HLA-DRB3*02:02
606	620	ATGWLQYNGSWYYLN	5.10	HLA-DRB3*02:02
730	744	TTVDGYKVNANGEWV	6.40	HLA-DRB3*02:02
147	161	LQNKFNEVRAVVVPE	6.50	HLA-DRB3*02:02
530	544	QENGMWYFYNTDGSM	8.90	HLA-DRB3*02:02
201	215	EAKELEIEKLQYEIS	1.50	HLA-DRB4*01:01
3	17	KKKMILTSLASVAIL	7.00	HLA-DRB4*01:01
207	221	IEKLQYEISTLEQEV	9.20	HLA-DRB4*01:01
332	346	TAALQNKLAAKKAEL	1.20	HLA-DRB5*01:01
413	427	TAALQNKLATKKAEL	2.50	HLA-DRB5*01:01
19	33	AGFVTSQPTFVRAEE	3.10	HLA-DRB5*01:01
575	589	WYYLEASGAMKASQW	3.20	HLA-DRB5*01:01
188	202	YATLKVALAKKEVEA	3.90	HLA-DRB5*01:01
99	113	VALVVQNAYKEYREV	9.00	HLA-DRB5*01:01
672	686	VNGSWYYLNANGSMA	9.60	HLA-DRB5*01:01

Table XIX – Results of the full-length ATXN3-CD4 T cell immunogenicity prediction.

Acquired with CD4 T Cell Immunogenicity Prediction tool (http://tools.iedb.org/CD4episcore/).

Peptide	Start	End	Combined Score	Immunogenicity Score	Peptide Core	Median Percentile Rank (7-Allele)	HLA-DRB1:03:01	HLA-DRB1:07:01	HLA-DRB1:15:01	HLA-DRB3:01:01	HLA-DRB3:02:02	HLA-DRB4:01:01	HLA-DRB5:01:01
DSGFFSIQVISNALK	71	85	38.87016	86.3754	IQVISNALK	7.2	59.0	7.2	7.3	65.0	5.4	5.6	2.6
SIQVISNALKVWGLE	76	90	39.54152	79.3538	IQVISNALK	13.0	35.0	11.0	13.0	52.0	3.5	27.0	6.3
QLLQMIRVQQMHRPK	176	190	47.46268	94.6567	IRVQQMHRP	16.0	16.0	48.0	19.0	48.0	15.0	0.01	11.0
SPEYQRLRIDPINER	96	110	49.76712	97.4178	YQRLRIDPI	18.0	1.3	37.0	46.0	4.4	18.0	5.8	48.0
RAIQLSMQGSSRNIS	251	265	53.61248	92.0312	MQGSSRNIS	28.0	7.3	38.0	28.0	56.0	9.7	12.0	34.0
VWGLELILFNSPEYQ	86	100	53.86208	88.1552	LILFNSPEY	31.0	36.0	63.0	4.6	31.0	14.0	14.0	38.0
ELISDTYLALFLAQL	141	155	54.03252	99.0813	LISDTYLAL	24.0	26.0	35.0	11.0	11.0	24.0	20.0	41.0
LILFNSPEYQRLRID	91	105	54.56228	91.4057	LILFNSPEY	30.0	19.0	39.0	9.0	45.0	6.3	45.0	30.0
YKEHWFTVRKLGKQW	116	130	55.24904	72.1226	EHWFTVRKL	44.0	56.0	30.0	57.0	76.0	44.0	42.0	5.5
QAAVTMSLETVRNDL	341	355	55.67868	95.6967	TMSLETVRN	29.0	3.0	22.0	77.0	29.0	56.0	25.0	40.0
EADLRRAIQLSMQGS	246	260	56.78076	98.4519	LRRAIQLSM	29.0	14.0	26.0	34.0	58.0	37.0	3.3	29.0
EEDMLQAAVTMSLET	336	350	57.84208	98.1052	DMLQAAVTM	31.0	16.0	4.4	52.0	38.0	22.0	31.0	38.0
EEDLQRALALSRQEI	226	240	58.47856	99.6964	LQRALALSR	31.0	5.9	31.0	41.0	77.0	32.0	16.0	2.9
LGKQWFNLNSLLTGP	126	140	58.64328	95.6082	QWFNLNSLL	34.0	62.0	32.0	23.0	48.0	2.2	48.0	34.0
EDYRTFLQQPSGNMD	56	70	58.7432	97.358	FLQQPSGNM	33.0	60.0	33.0	32.0	41.0	22.0	36.0	19.0
IRVQQMHRPKLIGEE	181	195	59.40232	96.0058	IRVQQMHRP	35.0	21.0	35.0	41.0	77.0	37.0	9.6	35.0
MHRPKLIGEELAQLK	186	200	59.84604	97.1151	IGEELAQLK	35.0	35.0	84.0	61.0	35.0	71.0	14.0	33.0
EYFSPVELSSIAHQL	26	40	60.0408	97.602	EYFSPVELS	35.0	33.0	17.0	57.0	50.0	35.0	30.0	46.0
FLAQLQQEGYSIFVV	151	165	64.07992	98.6998	LAQLQQEGY	41.0	56.0	41.0	34.0	19.0	52.0	20.0	62.0
FNLNSLLTGPELISD	131	145	64.55744	98.3936	LNSLLTGPE	42.0	42.0	36.0	37.0	65.0	32.0	72.0	55.0
FTVRKLGKQWFNLNS	121	135	64.80004	88.5001	VRKLGKQWF	49.0	66.0	83.0	42.0	75.0	49.0	17.0	38.0
RLRIDPINERSFICN	101	115	64.97864	96.4466	LRIDPINER	44.0	0.96	63.0	53.0	13.0	24.0	44.0	60.0
LLTGPELISDTYLAL	136	150	65.62932	99.5733	ELISDTYLA	43.0	28.0	43.0	51.0	3.4	36.0	64.0	75.0
TYLALFLAQLQQEGY	146	160	66.38524	98.4631	LAQLQQEGY	45.0	55.0	49.0	41.0	48.0	45.0	1.2	24.0
MSLETVRNDLKTEGK	346	360	66.6532	90.133	VRNDLKTEG	51.0	8.9	74.0	68.0	40.0	71.0	45.0	51.0
LIGEELAQLKEQRVH	191	205	67.31096	97.7774	LAQLKEQRV	47.0	46.0	85.0	47.0	77.0	80.0	13.0	27.0
SIFVVKGDLPDCEAD	161	175	68.02464	96.5616	SIFVVKGDL	49.0	16.0	70.0	48.0	49.0	68.0	20.0	74.0
LTSEELRKRREAYFE	276	290	68.24496	98.6124	EELRKRREA	48.0	39.0	69.0	48.0	43.0	89.0	60.0	45.0
SFICNYKEHWFTVRK	111	125	68.35352	95.8838	NYKEHWFTV	50.0	68.0	50.0	32.0	23.0	20.0	76.0	56.0
SRNISQDMTQTSGTN	261	275	68.90768	98.7692	QDMTQTSGT	49.0	8.6	90.0	87.0	42.0	49.0	30.0	91.0
DEEERMRMAEGGVTS	41	55	68.95256	97.3814	MRMAEGGVT	50.0	50.0	76.0	51.0	49.0	48.0	56.0	37.0
SNALKVWGLELILFN	81	95	69.09328	99.2332	ALKVWGLEL	49.0	54.0	49.0	24.0	49.0	63.0	9.2	46.0
PINERSFICNYKEHW	106	120	70.00868	95.5217	ERSFICNYK	53.0	63.0	78.0	53.0	23.0	27.0	85.0	42.0
DCEADQLLQMIRVQQ	171	185	71.36436	97.4109	LLQMIRVQQ	54.0	34.0	75.0	42.0	54.0	61.0	11.0	61.0
QQEGYSIFVVKGDLP	156	170	72.23416	92.0854	GYSIFVVKG	59.0	92.0	36.0	49.0	59.0	70.0	20.0	72.0
EQRVHKTDLERVLEA	201	215	74.2186	98.5465	HKTDLERVL	58.0	38.0	58.0	80.0	49.0	70.0	38.0	73.0
LAQLKEQRVHKTDLE	196	210	76.81408	96.0352	LKEQRVHKT	64.0	71.0	56.0	70.0	77.0	64.0	20.0	48.0
NLLQGEYFSPVELSS	21	35	77.469	97.6725	LLQGEYFSP	64.0	70.0	59.0	55.0	51.0	64.0	70.0	79.0
SGNMDDSGFFSIQVI	66	80	77.67872	99.6968	NMDDSGFFS	63.0	63.0	53.0	56.0	16.0	85.0	67.0	95.0

LRKRREAYFEKQQQK	281	295	77.95728	98.8932	RKRREAYFE	64.0	80.0	90.0	60.0	64.0	86.0	51.0	62.0
FLQQPSGNMDDSGFF	61	75	78.04992	99.1248	FLQQPSGNM	64.0	56.0	64.0	71.0	21.0	77.0	89.0	41.0
QHCLNNLLQGEYFSP	16	30	79.31896	97.7974	NNLLQGEYF	67.0	67.0	90.0	43.0	79.0	47.0	44.0	74.0
MESIFHEKQEGSLCA	1	15	81.313	98.2825	IFHEKQEGS	70.0	66.0	77.0	83.0	59.0	85.0	65.0	70.0
SLETVRNDLKTEGKK	347	361	81.35952	90.8988	LETVRNDLK	75.0	9.0	85.0	85.0	77.0	75.0	67.0	55.0
VELSSIAHQLDEEER	31	45	82.4358	99.5895	ELSSIAHQL	71.0	65.0	45.0	83.0	71.0	77.0	16.0	81.0
SMQGSSRNISQDMTQ	256	270	82.8788	96.197	MQGSSRNIS	74.0	46.0	60.0	88.0	81.0	52.0	74.0	93.0
GSLCAQHCLNNLLQG	11	25	83.33272	98.8318	AQHCLNNLL	73.0	80.0	56.0	74.0	76.0	50.0	44.0	73.0
MLDEDEEDLQRALAL	221	235	83.62616	99.5654	EEDLQRALA	73.0	36.0	66.0	91.0	73.0	96.0	73.0	83.0
DMEDEEADLRRAIQL	241	255	84.68772	99.2193	EEADLRRAI	75.0	35.0	69.0	93.0	44.0	92.0	75.0	87.0
GDAMSEEDMLQAAVT	331	345	84.72864	99.3216	SEEDMLQAA	75.0	56.0	93.0	75.0	68.0	95.0	67.0	90.0
RALALSRQEIDMEDE	231	245	85.76188	98.9047	ALALSRQEI	77.0	28.0	80.0	93.0	77.0	73.0	11.0	91.0
TSGTNLTSEELRKRR	271	285	85.91744	99.2936	LTSEELRKR	77.0	25.0	77.0	80.0	85.0	81.0	70.0	25.0
MRMAEGGVTSEDYRT	46	60	87.2316	99.579	MRMAEGGVT	79.0	71.0	90.0	76.0	79.0	94.0	90.0	68.0
QDMTQTSGTNLTSEE	266	280	87.39392	98.4848	MTQTSGTNL	80.0	91.0	19.0	78.0	93.0	78.0	80.0	90.0
KTDLERVLEANDGSG	206	220	88.48604	93.7151	RVLEANDGS	85.0	75.0	92.0	88.0	79.0	89.0	60.0	85.0
TSSGALGSDLGDAMS	321	335	89.5774	97.9435	LGSDLGDAM	84.0	22.0	90.0	81.0	15.0	89.0	84.0	91.0
GGVTSEDYRTFLQQP	51	65	90.05104	99.1276	GVTSEDYRT	84.0	26.0	90.0	84.0	25.0	88.0	85.0	83.0
RVLEANDGSGMLDED	211	225	90.8088	98.022	RVLEANDGS	86.0	63.0	87.0	86.0	16.0	23.0	90.0	88.0
GDLSGQSSHPCERPA	306	320	91.1494	98.8735	LSGQSSHPC	86.0	86.0	82.0	68.0	97.0	92.0	87.0	73.0
SRQEIDMEDEEADLR	236	250	92.48092	99.2023	MEDEEADLR	88.0	37.0	99.0	97.0	47.0	99.0	87.0	88.0
IAHQLDEEERMRMAE	36	50	92.82056	98.5514	HQLDEEERM	89.0	7.3	93.0	94.0	35.0	89.0	85.0	90.0
EAYFEKQQQKQQQQQ	286	300	95.03336	99.5834	AYFEKQQQK	92.0	92.0	98.0	95.0	95.0	91.0	14.0	64.0
CERPATSSGALGSDL	316	330	96.06512	99.1628	PATSSGALG	94.0	96.0	57.0	97.0	94.0	94.0	88.0	88.0
LGSDLGDAMSEEDML	326	340	96.21964	98.0491	LGSDLGDAM	95.0	31.0	95.0	98.0	45.0	98.0	87.0	98.0
HEKQEGSLCAQHCLN	6	20	96.24224	99.6056	HEKQEGSLC	94.0	97.0	88.0	94.0	94.0	95.0	67.0	95.0
KGDLPDCEADQLLQM	166	180	96.37816	99.9454	CEADQLLQM	94.0	52.0	97.0	96.0	33.0	94.0	76.0	99.0
QSSHPCERPATSSGA	311	325	97.2724	99.181	HPCERPATS	96.0	97.0	90.0	99.0	96.0	98.0	95.0	95.0
QQQQQGDLSGQSSHP	301	315	97.30308	99.2577	GDLSGQSSH	96.0	99.0	96.0	96.0	77.0	98.0	52.0	98.0
NDGSGMLDEDEEDLQ	216	230	98.85692	98.6423	GMLDEDEED	99.0	39.0	99.0	99.0	45.0	100.0	87.0	99.0
QQQQQQQQQQGDLSG	296	310	99.39088	99.9772	QQQQQQGDL	99.0	100.0	99.0	100.0	97.0	100.0	16.0	99.0
KQQQKQQQQQQQQQQ	291	305	99.97928	99.9482	KQQQKQQQQ	100.0	100.0	99.0	100.0	97.0	100.0	8.8	100.0

Table XX – Results of the "QQQQQQQQQQQQQQ" peptide of the mutant ATXN3-CD4 T cell immunogenicity prediction. Acquired with CD4 T Cell Immunogenicity Prediction tool (http://tools.iedb.org/CD4episcore/).

Peptide	Start	End	Combined Score	Immunogenicity Score	Peptide Core	Median Percentile Rank (7-Allele)	HLA-DRB1:03:01	HLA-DRB1:07:01	HLA-DRB1:15:01	HLA-DRB3:01:01	HLA-DRB3:02:02	HLA-DRB4:01:01	HLA-DRB5:01:01
QQQQQQQQQQQQQQ	X	X	99.99836	99.9959	QQQQQQQQQ	100.0	100.0	99.0	100.0	97.0	100.0	17.0	100.0

Figure II – Identification of immunogenic peptide cores in the fragment after the NSLL segment of ATXN3 sequence.

Figure III – Identification of immunogenic peptide cores in the fragment after the LISD segment of ATXN3 sequence.

Figure IV – Identification of immunogenic peptide cores in the fragment after the DLPD segment of ATXN3 sequence.

ATXN3 SEQUENCE SEGMENT (CUT AFTER DLPD SEGMENT)
N-FRAGMENT

MESIFHEKQEGSLCAQHCLNNLLQGEYFSPVELSSIAHQLDEEERMRMAEGGVTSEDYRTFLQQPSGNMDD
SGFFSIQVISNALKVWGLELILFNSPEYQRLRIDPINERSFICNYKEHWFTVRKLGKQWFNLNSLLTG
PELISDTYLALFLAQLQQEGYSIFVVKGDLPD

ATXN3 SEQUENCE SEGMENT (CUT AFTER DLPD SEGMENT)
C-FRAGMENT

CEADQLLQMIRVQQMHRPKLIGEELAQLKEQRVHKTDLERVLEANDGSGMLDEDEEDLQRALALSRQEID
MEDEEADLRRAIQLSMQGSSRNISQDMTQTSGTNLTSEELRKRREAYFEKQQQKQQQQQQQQQGDLSGQS
SHPCERPATSSGALGSDLGDAMSEEDMLQAAVTMSLETVRNDLKTEGKK

ATXN3 SEQUENCE SEGMENT (CUT AFTER DLPD SEGMENT)
C-FRAGMENT WITH POLYQ CHAIN REPEAT

CEADQLLQMIRVQQMHRPKLIGEELAQLKEQRVHKTDLERVLEANDGSGMLDEDEEDLQRALALSRQEID
MEDEEADLRRAIQLSMQGSSRNISQDMTQTSGTNLTSEELRKRREAYFEKQQQKQQQQQQQQQQQQQQQQ
QQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQGDLSGQ
SSHPCERPATSSGALGSDLGDAMSEEDMLQAAVTMSLETVRNDLKTEGKK

PolyQ Chain Repeat

Figure V – Identification of immunogenic peptide cores in the fragment after the AQLK segment of ATXN3 sequence.

ATXN3 SEQUENCE SEGMENT (CUT AFTER AQLK SEGMENT)
N-FRAGMENT

MESIFHEKQEGSLCAQHCLNNLLQGEYFSPVELSSIAHQLDEEERMRMAEGGVTSEDYRTFLQQPSGNMD
DSGFFSIQVISNALKVWGLELILFNSPEYQRLRIDPINERSFICNYKEHWFTVRKLGKQWFNLNSLL
TGPELISDTYLALFLAQLQQEGYSIFVVKGDLPDCEADQLLQMIRVQQMHRPKLIGEELAQLK

ATXN3 SEQUENCE SEGMENT (CUT AFTER AQLK SEGMENT)
C-FRAGMENT

EQRVHKTDLERVLEANDGSGMLDEDEEDLQRALALSRQEIDMEDEEADLRRAIQLSMQGSSRNISQDMTQ
TSGTNLTSEELRKRREAYFEKQQQKQQQQQQQQQQGDLSGQSSHPCERPATSSGALGSDLGDAMSEEDML
QAAVTMSLETVRNDLKTEGKK

ATXN3 SEQUENCE SEGMENT (CUT AFTER AQLK SEGMENT)
C-FRAGMENT WITH POLYQ CHAIN REPEATS

EQRVHKTDLERVLEANDGSGMLDEDEEDLQRALALSRQEIDMEDEEADLRRAIQLSMQGSSRNISQDMTQ
TSGTNLTSEELRKRREAYFEKQQQKQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQ
QQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQGDLSGQSSHPCERPATSSGALGSDLGDAMSEEDML
QAAVTMSLETVRNDLKTEGKK

PolyQ Chain Repeat

Figure VI – Identification of immunogenic peptide cores in the fragment after the TDLE segment of ATXN3 sequence.

Figure VII – Identification of immunogenic peptide cores in the fragment after the EAND segment of ATXN3 sequence.

Figure VIII – Identification of immunogenic peptide cores in the fragment after the LDED segment of ATXN3 sequence.

ATXN3 SEQUENCE SEGMENT (CUT AFTER LDED SEGMENT)
N-FRAGMENT

MESIFHEKQEGSLCAQHCLNNLLQGEYFSPVELSSIAHQLDEEERMRMAEGGVTSEDYRTFLQQPSGNMD
DSGFFSIQVISNALKVWGLELILFNSPEYQRLRIDPINERSFICNYKEHWFTVRKLGKQWFNLNSLL
TGPELISDTYLALFLAQLQQEGYSIFVVKGDLPDCEADQLLQMIRVQQMHRPKLIGEELAQLKEQRVHK
TDLERVLEANDGSGMLDED

ATXN3 SEQUENCE SEGMENT (CUT AFTER LDED SEGMENT)
C-FRAGMENT

EEDLQRALALSRQEIDMEDEEADLRRAIQLSMQGSSRNISQDMTQTSGTNLTSEELRKRREAYFEKQQQK
QQQQQQQQQQGDLSGQSSHPCERPATSSGALGSDLGDAMSEEDMLQAAVTMSLETVRNDLKTEGKK

ATXN3 SEQUENCE SEGMENT (CUT AFTER LDED SEGMENT)
C-FRAGMENT WITH POLYQ CHAIN REPEATS

EEDLQRALALSRQEIDMEDEEADLRRAIQLSMQGSSRNISQDMTQTSGTNLTSEELRKRREAYFEKQQQK
QQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQ
QQQQQQQQQQGDLSGQSSHPCERPATSSGALGSDLGDAMSEEDMLQAAVTMSLETVRNDLKTEGKK

PolyQ Chain Repeat

Figure IX – Identification of immunogenic peptide cores in the fragment after the DEED segment of ATXN3 sequence.

ATXN3 SEQUENCE SEGMENT (CUT AFTER DEED SEGMENT)
N-FRAGMENT

MESIFHEKQEGSLCAQHCLNNLLQGEYFSPVELSSIAHQLDEEERMRMAEGGVTSEDYRTFLQQPSGNMD
DSGFFSIQVISNALKVWGLELILFNSPEYQRLRIDPINERSFICNYKEHWFTVRKLGKQWFNLNSLL
TGPELISDTYLALFLAQLQQEGYSIFVVKGDLPDCEADQLLQMIRVQQMHRPKLIGEELAQLKEQRVHK
TDLERVLEANDGSGMLDEDEED

ATXN3 SEQUENCE SEGMENT (CUT AFTER DEED SEGMENT)
C-FRAGMENT

LQRALALSRQEIDMEDEEADLRRAIQLSMQGSSRNISQDMTQTSGTNLTSEELRKRREAYFEKQQQKQQ
QQQQQQQQGDLSGQSSHPCERPATSSGALGSDLGDAMSEEDMLQAAVTMSLETVRNDLKTEGKK

ATXN3 SEQUENCE SEGMENT (CUT AFTER DEED SEGMENT)
C-FRAGMENT WITH POLYQ CHAIN REPEATS

LQRALALSRQEIDMEDEEADLRRAIQLSMQGSSRNISQDMTQTSGTNLTSEELRKRREAYFEKQQQKQQ
QQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQ
QQQQQQQQQGDLSGQSSHPCERPATSSGALGSDLGDAMSEEDMLQAAVTMSLETVRNDLKTEGKK

PolyQ Chain Repeat

Figure X – Identification of immunogenic peptide cores in the fragment after the SGQS segment of ATXN3 sequence.

ATXN3 SEQUENCE SEGMENT (CUT AFTER SGQS SEGMENT)
N-FRAGMENT

MESIFHEKQEGSLCAQHCLNNLLQGEYFSPVELSSIAHQLDEEERMRMAEGGVTSEDYRTFLQQPSGNMD
DSGFFSIQVISNALKVWGLELILFNSPEYQRLRIDPINERSFICNYKEHWFTVRKLGKQWFNLNSLL
TGPELISDTYLALFLAQLQQEGYSIFVVKGDLPDCEADQLLQMIRVQQMHRPKLIGEELAQLKEQRVHK
TDLERVLEANDGSGMLDEDEEDLQRALALSRQEIDMEDEEADLRRAIQLSMQGSSRNISQDMTQTSGTNL
TSEELRKRREAYFEKQQQKQQQQQQQQQQGDLSGQS

ATXN3 SEQUENCE SEGMENT (CUT AFTER SGQS SEGMENT)
N-FRAGMENT WITH POLYQ CHAIN REPEAT

MESIFHEKQEGSLCAQHCLNNLLQGEYFSPVELSSIAHQLDEEERMRMAEGGVTSEDYRTFLQQPSGNMD
DSGFFSIQVISNALKVWGLELILFNSPEYQRLRIDPINERSFICNYKEHWFTVRKLGKQWFNLNSLL
TGPELISDTYLALFLAQLQQEGYSIFVVKGDLPDCEADQLLQMIRVQQMHRPKLIGEELAQLKEQRVHK
TDLERVLEANDGSGMLDEDEEDLQRALALSRQEIDMEDEEADLRRAIQLSMQGSSRNISQDMTQTSGTNL
TSEELRKRREAYFEKQQQKQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQ
QQQQQQQQQQQQQQQQQQQQQQQQQQQQQGDLSGQS

PolyQ Chain Repeat

ATXN3 SEQUENCE SEGMENT (CUT AFTER SGQS SEGMENT)
C-FRAGMENT

SHPCERPATSSGALGSDLGDAMSEEDMLQAAVTMSLETVRNDLKTEGKK

Figure XI – Identification of immunogenic peptide cores in the fragment after the LGSD segment of ATXN3 sequence.

ATXN3 SEQUENCE SEGMENT (CUT AFTER LGSD SEGMENT)
N-FRAGMENT

MESIFHEKQEGSLCAQHCLNNLLQGEYFSPVELSSIAHQLDEEERMRMAEGGVTSEDYRTFLQQPSGNMD
DSGFFSIQVISNALKVWGLELILFNSPEYQRLRIDPINERSFICNYKEHWFTVRKLGKQWFNLNSLL
TGPELISDTYLALFLAQLQQEGYSIFVVKGDLPDCEADQLLQMIRVQQMHRPKLIGEELAQLKEQRVHK
TDLERVLEANDGSGMLDEDEEDLQRALALSRQEIDMEDEEADLRRAIQLSMQGSSRNISQDMTQTSGTNL
TSEELRKRREAYFEKQQQKQQQQQQQQQQGDLSGQSSHPCERPATSSGALGSD

ATXN3 SEQUENCE SEGMENT (CUT AFTER LGSD SEGMENT)
N-FRAGMENT WITH POLYQ CHAIN REPEAT

MESIFHEKQEGSLCAQHCLNNLLQGEYFSPVELSSIAHQLDEEERMRMAEGGVTSEDYRTFLQQPSGNMD
DSGFFSIQVISNALKVWGLELILFNSPEYQRLRIDPINERSFICNYKEHWFTVRKLGKQWFNLNSLL
TGPELISDTYLALFLAQLQQEGYSIFVVKGDLPDCEADQLLQMIRVQQMHRPKLIGEELAQLKEQRVHK
TDLERVLEANDGSGMLDEDEEDLQRALALSRQEIDMEDEEADLRRAIQLSMQGSSRNISQDMTQTSGTNL
TSEELRKRREAYFEKQQQKQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQQ
QQQQQQQQQQQQQQQQQQQQQQQQQQQQQGDLSGQSSHPCERPATSSGALGSD

PolyQ Chain Repeat

ATXN3 SEQUENCE SEGMENT (CUT AFTER LGSD SEGMENT)
C-FRAGMENT

LGDAMSEEDMLQAAVTMSLETVRNDLKTEGKK

Figure XII – Identification of immunogenic peptide cores in the fragment after the ELAQ segment of ATXN3 sequence.

Figure XIII – Identification of immunogenic peptide cores in the fragment after the QEID segment of ATXN3 sequence.

Figure XIV – Identification of immunogenic peptide cores in the fragment after the ADLR segment of ATXN3 sequence.

Figure XV – Identification of immunogenic peptide cores in the fragment after the MQGS segment of ATXN3 sequence.

Figure XVI – Identification of immunogenic peptide cores in the fragment after the SSRN segment of ATXN3 sequence.

Figure XVII – Identification of immunogenic peptide cores in the fragment after the MTQT segment of ATXN3 sequence.

Figure XVIII – Identification of immunogenic peptide cores in the fragment after the TSEE segment of ATXN3 sequence.

Figure XIX – Identification of immunogenic peptide cores in the fragment after the RKRR segment of ATXN3 sequence.

Figure XX – Identification of immunogenic peptide cores in the fragment after the AYFE segment of ATXN3 sequence.

Figure XXI – Identification of immunogenic peptide cores in the fragment after the QQGD segment of ATXN3 sequence.