Identification of a simple and novel cut-point based CSF and MRI signature for predicting Alzheimer's disease progression that reinforces the 2018 NIA-AA research framework

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Abstract

The 2018 NIA-AA research framework proposes a classification system with beta-Amyloid deposition, pathologic Tau, and neurodegeneration (ATN) for the diagnosis and staging of Alzheimer's Disease (AD). Data from the ADNI (AD neuroimaging initiative) database can be utilized to identify diagnostic signatures for predicting AD progression, and to determine the utility of this NIA-AA research framework. Profiles of 320 peptides from baseline cerebrospinal

fluid (CSF) samples of 287 normal, mild cognitive impairment (MCI) and AD subjects followed over a 3-10 year period were measured via multiple reaction monitoring (MRM) mass spectrometry. CSF A β ₄₂, total-Tau (tTau), phosphorylated-Tau (pTau-181) and hippocampal volume were also measured. From these candidate markers, optimal diagnostic signatures with decision thresholds to separate AD and normal subjects were first identified via unbiased regression and tree-based algorithms. The best performing signature determined via cross-validation was then tested in an independent group of MCI subjects to predict future progression. This multivariate analysis yielded a simple diagnostic signature comprising CSF pTau-181 to A β ₄₂ ratio, MRI hippocampal volume and a novel PTPRN peptide, with a decision threshold on each marker. When applied to a separate MCI group at baseline, subjects meeting this signature criteria experience 4.3-fold faster progression to AD compared to a 2.2-fold faster progression using only conventional markers. This novel 4-marker signature represents an advance over the current diagnostics based on widely used marker, and is much easier to use in practice than recently published complex signatures. In addition, this signature reinforces the ATN construct from the 2018 NIA-AA research framework.

Introduction

Tools to provide an early diagnosis and prediction of progression to Alzheimer Disease (AD) are of critical importance. Early diagnosis allows caregivers to plan for additional needs which will decrease the overall financial burden of the illness [1, 2]. In addition, early diagnosis may help identify common comorbidities such as depression or undernutrition [3, 4], and may spur lifestyle interventions to mitigate some of the cognitive impairments associated with aging [5]. Finally, identifying individuals who are more likely to progress will help enrich clinical trial populations with subjects with more rapid progression, potentially shortening trial duration.

Early pathological changes of AD are seen years before the clinical diagnosis of AD. Most studies have shown that individuals with mild cognitive impairment (MCI) carry AD pathological burden and have a substantial risk (~10-15% per year) of development of dementia [6, 7]. Thus, as new therapeutics are developed that target AD-related pathology, MCI may

represent a state during which early intervention may change the trajectory of patient outcomes. However, therapeutics targeting A β will likely carry potential risks of significant side-effects, as documented in clinical trials [8-10], thus limiting their use to those with a high risk of subsequent cognitive decline. Therefore, what is needed is an approach to accurately identify MCI patients with the highest risk of conversion to AD.

Multiple potential biomarkers have been identified to aid in the prediction of conversion of MCI to AD. For example, cognitive and behavioral biomarkers have been proposed to identify individuals at high-risk for conversion [11-13]. In addition, biomarkers based on brain imaging or measurements in bodily fluids have been identified (for recent reviews see [14, 15]). The latter groups of biomarkers have recently been organized into a generalizable research framework. This framework, labeled AT(N), describes three classes of biomarkers: 1) “A” or aggregated amyloid-based (e.g., cerebrospinal fluid (CSF) A β ₄₂ levels, amyloid positron emission tomography (PET)), 2) “T” or aggregated tau-based (e.g., CSF phosphorylated tau [pTau-181], tau PET) and 3) “N” or neuronal degeneration-based (e.g., volumetric magnetic resonance imaging (MRI), fluorodeoxyglucose (FDG) PET, CSF total tau (tTau)) [16, 17]. Furthermore, Jack et al, 2018, advocate extending this to the ATX(N) framework, where X can include additional markers from the multiarray-omics platforms. This research framework is intended to form a common approach by which investigators can communicate about and classify novel biomarkers, thereby allowing their more rapid integration into current research.

CSF-based biomarkers have been of interest since they represent an assessment of biochemical changes in the central nervous system. The most commonly-observed changes in the CSF of AD subjects have been a reduction of A β ₄₂ and increase in pTau-181 [18, 19]. We recently identified a 16-analyte CSF signature which showed higher sensitivity and specificity than any combination of A β ₄₂, tTau and pTau-181 for the diagnosis of AD vs. controls, and, when applied to an independent dataset of MCI subjects, outperformed traditional biomarkers in prediction of conversion to AD [20]. Unfortunately, a complicated 16-analyte signature is not practical for clinical purposes. Multi-analyte signatures require quality-control measures for each analyte and do not provide an intuitive understanding of how changes in the biomarker impact the disease. Therefore, here, using data from only the AD and age-matched Normal (NL) subjects from the

ADNI (AD neuroimaging initiative) database, we identify optimal diagnostic signatures with decision thresholds on a few markers that separate AD and NL subjects via unbiased regression and tree-based algorithms [21, 22]. The best performing signatures from the NL and AD subjects were then tested in an independent group of MCI subjects at baseline to determine their ability to predict the future progression. By developing a simple, yet powerful, signature for prediction of MCI to AD, this work confirms and extends the AT(N) framework and provides a potential new tool for clinicians to use to advise decision making and for researchers to enrich clinical trials with MCI subjects with a higher likelihood of conversion.

Methods

Data used for this research were mostly identical to that used in Llano et al (2017), except that the progression data on MCI now extends to another two years, and we include data from the conventional biomarkers (CSF amyloid/tau and MRI HV) in the analysis. For the sake of completeness, we repeat some of the key information pertaining to these data in this paper. The ADNI database (adni.loni.usc.edu) utilized in this research was launched in 2003 as a public-private partnership, led by Principal Investigator Michael W. Weiner, MD. The primary goal of ADNI has been to test whether serial MRI, PET, other biological markers, and clinical and neuropsychological assessments can be combined to measure the progression of MCI and early AD. For up-to-date information, see www.adni-info.org. This study was conducted across multiple clinical sites and was approved by the Institutional Review Boards of all of the participating institutions. Informed written consent was obtained from all participants at each site. Data used for the analyses presented here were accessed on February 24, 2018. Although the ADNI database continues to be updated on an ongoing basis, most newly added biomarker data are from later time points (i.e., beyond 1 year), in contrast to the baseline data used in this study.

Patient Population

This research was focused on only those subjects in the ADNI database for whom data from both the conventional markers (CSF amyloid/tau and MRI HV) and novel markers (320 peptides from the multiple reaction monitoring (MRM) proteomics panel) were available at baseline. This included 287 subjects with AD, MCI and NL from the ADNI study that received clinical, neuropsychological, and biomarker assessments which were repeated every six months for a

period of 3 to 10 years. NL individuals were free of memory complaints or depression and had a Mini-Mental State Examination (MMSE) score above 25 and a Clinical Dementia Rating (CDR) score of 0. MCI individuals could have MMSE scores of 23 to 30 and required a CDR of 0.5 and an informant-verified memory complaint substantiated by abnormal education-adjusted scores on the Wechsler Memory Scale Revised—Logical Memory II. AD patients could have MMSE scores of 20 to 27 and a CDR of 0.5 or 1.0.

Imaging:

All participants received 1.5 Tesla (T) structural MRI at baseline and at every six months for the next several years. In addition, approximately 25% also received 3.0 T MRI. Cognitive assessments and neuroimaging procedures were carried out within two weeks of each other. In this research, we utilized only the baseline HV data measured via MRI and computed using the FreeSurfer software at the University of California in San Francisco. Details regarding this software can be found in the “UCSF FreeSurfer Methods” PDF document under “MR Image Analysis” in the ADNI section of <https://ida.loni.usc.edu/> as well as in [23-25].

CSF Samples:

CSF levels of A β ₄₂, tTau, and pTau-181 were determined using Innogenetics’ INNO-BIA AlzBio3 immunoassay on a Luminex xMAP platform (see [19] for details). These CSF samples were also processed in the Caprion Proteomics platform that uses mass spectrometry to evaluate the ability of a panel of peptides to discriminate between disease states and predict disease progression. The CSF multiplex MRM panel was developed by Caprion Proteomics in collaboration with the ADNI Biomarker Consortium Project Team. A total of 320 peptides generated from tryptic digests of 143 proteins were used in this study (see Supplemental Table 1 and supplemental table in [20] for list of peptides and proteins).

Details regarding the technology, quality control and validation of the MRM platform can be found in the Use of Targeted Mass Spectrometry Proteomic Strategies to Identify CSF-Based Biomarkers in Alzheimer’s Disease Data Primer (found under Biomarkers Consortium CSF Proteomics MRM Data Primer at ida.loni.usc.edu). In brief, as described in the data primer and in [26], plasma proteins were depleted from CSF samples using a Multiple Affinity Removal

System (MARS-14) column, and digested with trypsin (1:25 protease:protein ratio). The samples were then lyophilized, desalted and analyzed by LC/MRM-MS analysis on a QTRAP 5500 LC-MS/MS system at Caprion Proteomics. MRM experiments were performed on triple quadrupole (Q) mass spectrometers. The first (Q1) and third (Q3) mass analyzer were used to isolate a peptide ion and a corresponding fragment ion. The fragment ions were generated in Q2 by collision induced dissociation. All peptide levels are presented as normalized and log2-transformed intensities as we and others have done previously [20, 26], which is identical to the manner in which they were provided in the quality-controlled dataset.

Statistical Methods:

Optimal combinatorial signatures with simple decision thresholds on each marker were first identified from the AD and NL subjects. This was performed in an unbiased, data driven manner via regression and tree-based computational algorithms called Patient Rule Induction Method [21] and Sequential BATting [22]. Prior to application of these algorithms on the 320-peptide MRM panel, promising peptide candidates were identified using the same algorithm utilized in [20], using the logistic regression model with Lasso penalty [27] and a bootstrap procedure [28] to improve the stability of the lasso parameter estimate.

The predictive performance of the optimal signature from each algorithm for differentiating the AD and NL subjects was then evaluated via 10 iterations of five-fold cross-validation. In this procedure, the original data were divided into five random subsets (folds), each fold was left out one at a time, and the remaining four folds were used to derive a signature, which was then used to predict the disease state of each subject in the left-out fold. This process was carried out for each left-out fold one at a time and the predictions of all the five left-out folds were aggregated. For better stability and robustness, this cross-validation procedure was repeated 10 times and the median of each these performance measures was calculated. All steps of the model building and signature derivation process were fully embedded within this cross-validation to further reduce any possible bias [29].

The optimal signature from the best performing algorithm determined via the above cross-validation procedure (i.e., the signature that best differentiated AD and NL subjects) was then

tested on a separate independent group of 135 MCI subjects at baseline, to predict their future progression to AD. MCI subjects predicted to be AD-like (“Signature Positive” at baseline) were considered as future converters to AD, and those predicted to be NL-like (“Signature Negative” at baseline) were considered as non-converters. These baseline predictions were then compared to the follow-up clinical data. Performance metrics such as the PPV, NPV and overall accuracy were calculated by comparing the predictions to the known progression status of the MCI subjects to AD over the next 36 months. Comparisons of the performance metrics between different signatures were carried out via exact McNemar’s test.

The performance of this signature was then evaluated in terms of its ability to differentiate the future time to progression from MCI to AD of these baseline signature-positive and signature-negative MCI subjects via Kaplan-Meier analysis. For this evaluation, the progression of MCI subjects to AD over the entire future time course until the last follow-up visit was taken into consideration.

This analysis procedure was carried out separately for the following subsets of markers, along with APOE genetic status, age, gender and education (4 markers):

- MRI brain HV: 5 total markers (the 4 markers above + HV)
- CSF A β ₄₂, tTau, pTau-181, ratios of tTau to A β ₄₂ & pTau-181 to A β ₄₂ (AT): 9 total markers
- AT + HV: 10 total markers, and
- AT + HV + 320 peptides from the CSF MRM panel: 330 total markers

This evaluation of the AD versus NL peptide signature on the future progression of a separate group of MCI subjects to AD not only served as an independent verification of the utility of the signature, but also put it to a greater test to see whether it is robust enough to address a different and more important question related to the prediction of future progression of the MCI subjects to AD. The analysis procedure described here is summarized in Figure 1.

All analyses related to predictive modeling and signature derivation were carried out using R (<http://www.R-project.org>), version 3.4.1, with the publicly available package, SubgrpID [22].

The time to progression analysis of the derived signatures and related assessments were carried out using JMP®, version 13.2.

Results:

Disease-state Demographics:

Table 1A summarizes the key demographics of the 66 AD, 135 MCI and 86 NL subjects, and Table 1B provides a breakdown of the 135 MCI subjects in terms of their future progression. The subjects were balanced across groups in terms of age and education (both $p > 0.05$). There were significantly more males (59.1%) than females (40.9%) in the study, though similar numbers of male and female MCI subjects converted to AD over a three-year period (44% vs. 54.6%, $p = 0.248$, Chi-squared test). As shown previously [30], the presence of at least one copy of the APO-E4 allele was a risk factor for AD (71.2% AD, 52.6% MCI and 24.4% NL, $p < 0.0001$, Chi-squared test). In addition, this allele also tracked with MCI to AD progression over a 36-month period (37.5% of non-E4 vs. 56.3% of E4 progressed to AD, $p = 0.029$, Chi-squared test).

Disease state classification:

The distribution of the conventional biomarkers for NL, MCI and AD subjects are shown in Figures 2A-D. While the means significantly differ across groups ($p < 0.0001$), the considerable overlap of expression levels greatly limits the diagnostic utility of any of these markers on their own.

Multivariate analysis of the various markers using data-driven computational algorithms described above yielded optimal signatures for differentiating the disease states and prediction of disease progression. These signatures are summarized in Table 2. Interestingly, the signature derived from the conventional and novel markers took a very simple form based on only a few markers, with representations from both the conventional markers and the novel MRM panel, along with a cut-point on each of them; it took the form of $HV < 7.65 \text{ cm}^3$, ratio of pTau to $A\beta_{42} > 0.09$ and a PTPRN peptide (sequence SELEAQTGLQILQTGVGQR, referred to here as PTPRN.SELE) < 10.22 intensity units. Figures 2E-F show the significant decline of PTPRN.SELE in AD relative to both NL ($p = 0.002$) and MCI ($p = 0.004$), and a trend towards a decline in baseline MCI subjects that progress to AD in the future ($p = 0.065$).

Prediction of MCI to AD progression:

For disease state classification, the signatures derived from all data scenarios have similar levels of overall accuracy, with no discernable advantage of adding novel markers from the MRM panel to the conventional markers. However, for the prediction of 36-month progression in the independent group of 135 MCI subjects at baseline, the signature derived from the collection of both conventional and novel markers significantly outperforms the signatures based on the conventional markers ($p=0.0002$), with the NPV increasing from 70.2% to 77.6% ($p=0.0032$) and the PPV increasing slightly from 60.2% to 61.6% ($p=0.0107$). Thus, the addition of a novel PTPRN peptide from the MRM panel to the conventional AD markers substantially improves the prediction of 36-month disease progression in MCI subjects at baseline.

Based on the available 3-10 year follow-up clinical data available on these subjects, the performance of the optimal signatures from all the scenarios was further assessed on this independent group of baseline MCI subjects with respect to their future time to progression. Table 3 includes a summary of the 25th percentile, median, and 75th percentile time to progression of the signature negative and signature positive subjects, and the overall Hazard Ratio with 95% confidence bands. Based on these results, the optimal combination of conventional markers showed a Hazard Ratio of 2.2 suggesting that the MCI subjects meeting the criteria of this signature experience 2.2-fold faster progression to AD. However, the MCI subjects that meet the signature criterion from the scenario that includes the PTPRN peptide experience 4.3-fold faster progression to AD, as shown in Figure 3. To determine if the impact of PTPRN was likely isolated to the particular peptide fragment (PPRN.SELE, the other two PTPRN peptides (AEAPALFSR, referred to as PTPRN.AEAP and LAAVLAGYGVLELR, referred to as PTPRN.LAAV) in the MRM panel were also assessed. The pairwise correlations between these three peptides are all over 87% (data not shown).

Discussion

Summary:

We examined the ability of a simple optimized multivariate signature comprising conventional biomarkers combined with an array of novel CSF peptides from the ADNI database to both

classify AD disease state and to predict MCI to AD conversion. We observed that both conventional AD biomarkers (HV and CSF pTau/A β ₄₂ ratio) and conventional biomarkers combined with an array of novel CSF peptides performed similarly in terms of classifying disease state (AD vs. NL). However, when these optimized signatures were applied to an independent group of MCI subjects, the signature combining conventional markers with a novel peptide analyte derived from PTPRN substantially outperformed the conventional biomarkers in predicting MCI to AD conversion by nearly twofold. In addition, the combined signature contains only four elements: HV, CSF A β ₄₂, tTau and the PTPRN.SELE peptide, thus making it simple enough to be tractable for clinical and research purposes. These data may also open new lines of investigation regarding the role of PTPRN in AD as well as confirming and extending the proposed AT(N) framework for AD biomarkers.

PTPRN and AD:

PTPRN is expressed widely in neurons throughout the mouse and human brain, including areas associated with AD neurodegeneration such as hippocampus and neocortex [31, 32]. It is a membrane-spanning protein phosphatase with cytoplasmic and luminal components and is found in the membranes of secretory granules. The gene for PTPRN is also highly expressed in pancreatic islet cells, and antibodies against this protein are found in type 1 diabetes, hence its alternative name islet-antigen 2 [33]. Deficiency in PTPRN is associated with glucose intolerance in animal models [34] as well as impaired learning [35]. Given the associations between diabetes, insulin resistance and AD [36-38], it is possible that the PTPRN/AD association seen in the current study points to a new and specific role for metabolic dysregulation in the pathophysiology of AD to complement other metabolic hypotheses of AD [39, 40].

Several previous studies have identified PTPRN as a potential marker of AD. For example, downregulation of the expression of the PTPRN gene has been observed in the hippocampus of sporadic AD subjects [41] as well as the posterior cingulate area of early-onset AD and presenilin-1 mutation-related dementia [42]. In addition, when incorporated into a three-gene classifier, PTPRN expression levels have been found to discriminate between patients with AD pathology and no symptoms, and those with only AD pathology [43]. Finally, in a preliminary study of genetic interactions with CSF pTau levels for predicting MCI to AD conversion,

PTPRN levels showed differences with respect to CSF pTau levels in MCI to AD converters compared to non-converters [44].

Implications of the prediction of MCI-AD conversion:

Over the years, several groups have examined the ability of multi-modal combination biomarkers (i.e., combinations of imaging, cognitive, body fluid and other markers) to predict the conversion of MCI to AD. Ideally, utilizing an approach such as the AT(N) framework, a combination biomarker should merge several orthogonal measurements reflecting different underlying biological processes. Larger combinations of biomarkers have the potential to increase the predictive power of the combination biomarker. The multiplicity of biomarkers is limited by clinical reality such that it is often impractical and costly to obtain multiple studies in individual patients. Therefore, a challenge in developing combination biomarkers is to develop combinations that provide high predictive MCI to AD accuracy and are clinically feasible.

Here, we have identified a 4-marker signature that combines volumetric MRI and CSF testing, both feasible clinical tests, that outperforms standard biomarkers in the prediction of MCI to AD. Although other studies have found that combinations of volumetric MRI and CSF measures can predict MCI to AD conversion [45-50], a unique aspect of the current biomarker signature is that it was initially developed using disease state markers from one population of subjects, and then validated on an independent group of individuals with MCI, increasing its generalizability. In addition, because the 4-marker signature is in the form of simple decision cut-points, it can readily be applied for clinical trial patient enrollment and in clinical practice for physicians without the need for complex calculations. It will be beneficial in the future to evaluate the performance of this signature in databases containing other neurodegenerative diseases to determine the specificity of these markers against related illnesses. In addition, further evaluation and validation of PTPRN as a diagnostic and progression marker for patients with early signs of cognitive impairment, in conjunction with the core beta-amyloid and tau markers, in line with the ATN construct proposed in the 2018 NIA-AA consensus paper may provide additional insights about AD pathology.

Figure legends:

Figure 1: Statistical analysis flow-scheme

Figure 2: Distribution of four conventional markers of AD (A: CSF A β ₄₂, B: CSF pTau-181, C: CSF tTau, D: MRI HV) are shown for the NL, MCI and AD subjects at baseline. Among the MCI subjects, those that progressed to AD over 36 months are shown in red and rest are shown in blue. The bottom and top ends of the box represent the first and third quartiles respectively, with the line inside the box representing the median. Lines extending out of the ends of the box indicate the range of the data, minus the outliers. The points outside the lines are the low and high outliers. E) Distribution of PTPRN.SELE peptide (in normalized log₂ transformed intensity units) is shown across the NL, MCI and AD groups at baseline, and F) for the baseline MCI subjects that either progressed to AD or remained stable over the next 36 months.

Figure 3: Time to progression profiles of the signature positive versus signature negative MCI subjects with the shaded 95% confidence bands are shown here via Kaplan-Meier analysis. The effect of signature based on only the conventional markers (HV and AT) is illustrated in Figure 4A and the signature with both the conventional markers and the novel PTPRN.SELE peptide from the MRM panel is shown in Figure 3B. Patients meeting the signature criterion that includes this PTPRN peptide experience 4.3-fold faster progression to AD (hazard ratio = 4.4), relative to the 2.2-fold faster progression without this peptide.

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Table 1: Disease-state demographics

		AD	MCI	NL
		(n=66)	(n=135)	(n=86)
Gender (n)	M	37	91	44
	F	29	44	42
Apo-E (n)	E4	47	71	21
	Non-E4	19	6	65
Age (years, Mean +/- SD)		75.1 +/- 7.5	74.8 +/- 7.4	75.8 +/- 5.6
Education (years, Mean +/- SD)		15.1 +/- 3	16 +/- 3	15.6 +/- 3
MMSE (Mean +/- SD)		23.5 +/- 1.9	26.9 +/- 1.7	29.1 +/- 1

		MCI to AD converters	Stable MCI
		(n=64)	(n=71)
Gender (n)	M	40	51
	F	24	20
Apo-E (n)	E4	40	31
	Non-E4	24	40
Age (years, Mean +/- SD)		74.9 +/- 7.6	74.7 +/- 7.2
Education (years, Mean +/- SD)		15.6 +/- 3.0	16.4 +/- 2.9
MMSE (Mean +/- SD)		26.4 +/- 1.7	27.4 +/- 1.6

Table 2: Performance of optimal signatures

Data type	Diagnostic Criteria for Signature positive	AD versus Normal Diagnosis (internal cross-validation)			36m MCI Progression to AD (independent validation)		
		PPV	NPV	Accuracy	PPV (MCI to AD)	NPV (Stable MCI)	Accuracy
AT	tTau / Ab1-42 > 0.59	71.6%	80.5%	76.5%	58.1%	66.1%	61.7%
HV	HV < 6.41 and ApoE4 +	92.7%	74.8%	79.6%	61.2%	60.5%	60.7%
AT + HV	HV < 7.0, pTau > 18.1, and tTau / Ab1-42 > 0.36	73.4%	78.4%	76.3%	60.2%	70.2%	64.4%
AT + HV + MRM	HV < 7.65, pTau / Ab1-42 > 0.09, and PTPRN.SELE < 10.22	75%	79.6%	77.6%	61.6%	77.6%	67.4%

Table 3: Time to progression (T2P) of MCI subjects to AD using optimal signatures

Data type	Diagnostic Criteria for Signature positive	Signature Negative		Signature Positive		Hazard Ratio (95% C.I.)
		N	T2P (months) Q1, Q2, Q3	N	T2P (months) Q1, Q2, Q3	
AT	$t\text{Tau} / \text{Ab1-42} > 0.59$	59	23.4, 71.6, 108	76	13.6, 25.7, 72.0	1.9 (1.2, 3.1)
HV	$\text{HV} < 6.41$ and $\text{ApoE4} +$	86	18.6, 48.2, 108	49	13.1, 31.5, 60.0	2.0 (1.3, 3.2)
AT + HV	$\text{HV} < 7.0$, $p\text{Tau} > 18.1$, and $t\text{Tau} / \text{Ab1-42} > 0.36$	57	24.4, 71.6, 108	78	12.6, 25.7, 72.0	2.2 (1.4, 3.6)
AT + HV + MRM	$\text{HV} < 7.65$, $p\text{Tau} / \text{Ab1-42} > 0.09$, and $\text{PTPRN.SELE} < 10.22$	49	48.0, 96.5, 120	86	12.7, 24.1, 54.9	4.3 (2.5, 7.7)

Figure 1

Figure 2

Figure 3

Supplemental Table 1. List of Peptides. All peptides, proteins and UniProt accession numbers from the peptides measured in this study.

Peptide	Protein	Uniprot accession number
A1AT_AVLTIDEK	Alpha-1-antitrypsin	P01009
A1AT_LSGTYDLK	Alpha-1-antitrypsin	P01009
A1AT_SVLGQLGITK	Alpha-1-antitrypsin	P01009
A1BG_NGVAQEPVHLDSPAIK	Alpha-1B-glycoprotein	P04217
A1BG_SGLSTGWTQLSK	Alpha-1B-glycoprotein	P04217
A2GL_DLLLPQDLR	Leucine-rich alpha-2-glycoprotein	P02750
A2GL_VAAGAFQGLR	Leucine-rich alpha-2-glycoprotein	P02750
A4_LVFFAEDVGSNK	Amyloid beta A4 protein	P05067
A4_THPHFVIPYR	Amyloid beta A4 protein	P05067
A4_WYFDVTEGK	Amyloid beta A4 protein	P05067
AACT_ADLSGITGAR	Alpha-1-antichymotrypsin	P01011
AACT_EIGELYLPK	Alpha-1-antichymotrypsin	P01011
AACT_NLAVSQVVHK	Alpha-1-antichymotrypsin	P01011
AATC_IVASTLSNPelfEWTGNVK	Aspartate aminotransferase, cytoplasmic	P17174
AATC_LALGDDSPALK	Aspartate aminotransferase, cytoplasmic	P17174
AATC_NLDYVATSIHEAVTK	Aspartate aminotransferase, cytoplasmic	P17174
AATM_FVTVQTISGTGALR	Aspartate aminotransferase, mitochondrial	P00505
AFAM_DADPDFFAK	Afamin	P43652
AFAM_FLVNLVK	Afamin	P43652
AFAM_LPNNVLQEK	Afamin	P43652
ALDOA_ALQASALK	Fructose-bisphosphate aldolase A	P04075
ALDOA_QLLLTADDR	Fructose-bisphosphate aldolase A	P04075
AMBP_AFIQLWAFDAVK	Protein AMBP	P02760
AMBP_ETLLQDFR	Protein AMBP	P02760
AMBP_FLYHK	Protein AMBP	P02760
AMD_IPVDEEAFVIDFKPR	Peptidyl-glycine alpha-amidating monooxygenase	P19021
AMD_IVQFSPSGK	Peptidyl-glycine alpha-amidating monooxygenase	P19021
AMD_NGQWTLIGR	Peptidyl-glycine alpha-amidating monooxygenase	P19021
APLP2_HYQHVLAVDPEK	Amyloid-like protein 2	P05067
APLP2_WYFDLSK	Amyloid-like protein 2	P05067
APOB_IAELSATAQEIIK	Apolipoprotein B-100	P04114

APOB_SVSLPSLDPASAK	Apolipoprotein B-100	P04114
APOB_TGISPLALIK	Apolipoprotein B-100	P04114
APOD_VLNQELR	Apolipoprotein D	P05090
APOE_AATVGSAGQPLQER	Apolipoprotein E	P02649
APOE_CLAVYQAGAR	Apolipoprotein E	P02649
APOE_LAVYQAGAR	Apolipoprotein E	P02649
APOE_LGADMEDVR	Apolipoprotein E	P02649
APOE_LGPLVEQGR	Apolipoprotein E	P02649
B2MG_VEHSDFSFSK	Beta-2-microglobulin	P61769
B2MG_VNHNLSQPK	Beta-2-microglobulin	P61769
B3GN1_EPGEFALLR	N-acetyllactosaminide beta-1,3-N-acetylglucosaminyltransferase	Q9NY97
B3GN1_TALASGGVLDASGDYR	N-acetyllactosaminide beta-1,3-N-acetylglucosaminyltransferase	Q9NY97
B3GN1_YEAAVPDPR	N-acetyllactosaminide beta-1,3-N-acetylglucosaminyltransferase	Q9NY97
BACE1_SIVDSGTTNLR	Beta-secretase 1	P56817
BASP1_ETPAATEAPSSTPK	Brain acid soluble protein 1	P80723
BTD_LSSGLVTAALYGR	Biotinidase	P43251
BTD_SHLIIAQVAK	Biotinidase	P43251
C1QB_LEQGENVFLQATDK	Complement C1q subcomponent subunit B	P02746
C1QB_VPGLYYFTYHASSR	Complement C1q subcomponent subunit B	P02746
CA2D1_FVVTGGITR	Voltage-dependent calcium channel subunit alpha-2/delta-1	P54289
CA2D1_IKPVFIEDANFGR	Voltage-dependent calcium channel subunit alpha-2/delta-1	P54289
CA2D1_TASGVNQLVDIYEK	Voltage-dependent calcium channel subunit alpha-2/delta-1	P54289
CAD13_DIQGSLQDIFK	Cadherin-13	P55290
CAD13_INENTGSVSVTR	Cadherin-13	P55290
CAD13_YEVSSPYFK	Cadherin-13	P55290
CADM3_EGSVPPLK	Cell adhesion molecule 3	Q8N126
CADM3_GNPVPQQYLWEK	Cell adhesion molecule 3	Q8N126
CADM3_SLVTVLGIPQKPIITGYK	Cell adhesion molecule 3	Q8N126
CAH1_VLDALQAIK	Carbonic anhydrase 1	P00915
CAH1_YSSLAEAAASK	Carbonic anhydrase 1	P00915
CATA_LFAYPDTHR	Catalase	P04040
CATD_LVDQNIFSFLSR	Cathepsin D	P07339
CATD_VSTLPAITLK	Cathepsin D	P07339
CATD_YSQAVPAVTEGPIPEVLK	Cathepsin D	P07339
CATL1_VFQEPLFYEAPR	Cathepsin L1	P07711

CCKN_AHLGALLAR	Cholecystokinin	P06307
CCKN_NLQNLDPSHR	Cholecystokinin	P06307
CD14_AFPALTSLDSDNPGLGER	Monocyte differentiation antigen CD14	P08571
CD14_FPAIQNLALR	Monocyte differentiation antigen CD14	P08571
CD14_SWLAELQQWLKPGLK	Monocyte differentiation antigen CD14	P08571
CD59_AGLQVYNK	CD59 glycoprotein	P13987
CERU_IYHSHIDAPK	Ceruloplasmin	P00450
CERU_NNEGTYYSNPYNPQSR	Ceruloplasmin	P00450
CFAB_DAQYAPGYDK	Complement factor B	P00751
CFAB_VSEADSSNADWVTK	Complement factor B	P00751
CFAB_YGLVTYATYPK	Complement factor B	P00751
CH3L1_ILGQQVPYATK	Chitinase-3-like protein 1	P36222
CH3L1_SFTLASSETGVGAPISGPGIPGR	Chitinase-3-like protein 1	P36222
CH3L1_VTIDSSYDIK	Chitinase-3-like protein 1	P36222
CLUS_IDSLENDR	Clusterin	P10909
CLUS_SGSGLVGR	Clusterin	P10909
CLUS_VTTVASHTSDSDVPSGVTEVVV K	Clusterin	P10909
CMGA_EDSLEAGLPLQVR	Chromogranin-A	P10645
CMGA_SEALAVDGAGKPGAEEAQDPE GK	Chromogranin-A	P10645
CMGA_SGEATDGARPPALPEPMQESK	Chromogranin-A	P10645
CMGA_SGELEQEEER	Chromogranin-A	P10645
CMGA_YPGPQAEGLDSEGLSGLVDR	Chromogranin-A	P10645
CNDP1_ALEQDLPVNIK	Beta-Ala-His dipeptidase	Q96KN2
CNDP1_VFQYIDLHQDEFVQTLK	Beta-Ala-His dipeptidase	Q96KN2
CNDP1_WNYIEGTK	Beta-Ala-His dipeptidase	Q96KN2
CNTN1_DGEYVVEVR	Contactin-1	Q12860
CNTN1_TTKPYPADIVVQFK	Contactin-1	Q12860
CNTN2_IIVQAQPEWLK	Contactin-2	Q02246
CNTN2_TTGPGGDGIPAETHIVR	Contactin-2	Q02246
CNTN2_VIASNILGTGEPSPSSK	Contactin-2	Q02246
CNTP2_HELQHPIAR	Contactin-associated protein-like 2	Q9UHC6
CNTP2_VDNAPDQQNSHPDLAQEEIR	Contactin-associated protein-like 2	Q9UHC6
CNTP2_YSSSDWVTQYR	Contactin-associated protein-like 2	Q9UHC6
CO2_DFHINLFR	Complement C2	P06681
CO2_HAIILLTDGK	Complement C2	P06681

CO2_SSGWQTPGATR	Complement C2	P06681
CO3_IHWESASLLR	Complement C3	P01024
CO3_LSINTHPSQKPLSITVR	Complement C3	P01024
CO3_TELRPGETLNVNLLR	Complement C3	P01024
CO3_TGLQEVEVK	Complement C3	P01024
CO3_VPVAVQGEDTVQSLTQGDGVAK	Complement C3	P01024
CO4A_DHAVDLIQK	Complement C4-A	P0C0L4
CO4A_GSFEFPVGDAVSK	Complement C4-A	P0C0L4
CO4A_LGQYASPTAK	Complement C4-A	P0C0L4
CO4A_NVNFQK	Complement C4-A	P0C0L4
CO4A_VLSLAQEQVGGSPK	Complement C4-A	P0C0L4
CO4A_VTASDPLDTLGSEALSPGGVA SLLR	Complement C4-A	P0C0L4
CO5_DINYVNPVIK	Complement C5	P01031
CO5_TLLPVSKPEIR	Complement C5	P01031
CO5_VFQFLEK	Complement C5	P01031
CO6_ALNHLPLEYNSALYSR	Complement component C6	P13671
CO6_SEYGAALAWK	Complement component C6	P13671
CO8B_IPGIFELGISSQSDR	Complement component C8 beta chain	P07358
CO8B_SDLEVAHYK	Complement component C8 beta chain	P07358
CO8B_YEFILK	Complement component C8 beta chain	P07358
COCH_GVISNSGGPVR	Cochlin	O43405
CRP_ESDTSYVSLK	C-reactive protein	P02741
CSTN1_GNLAGLTR	Calsyntenin-1	O94985
CSTN1_IHGQNVFPDAVVVDK	Calsyntenin-1	O94985
CSTN1_IPDGVVSVSPK	Calsyntenin-1	O94985
CSTN3_ATGEGLIR	Calsyntenin-3	Q9BQT9
CSTN3_ESLLDITSLQQR	Calsyntenin-3	Q9BQT9
CUTA_TQSSLVPALTDVFR	Protein CutA	O60888
CYTC_ALDFAVGEYNK	Cystatin-C	P01034
DAG1_GVHYISVSATR	Dystroglycan	Q14118
DAG1_LVPVVNNR	Dystroglycan	Q14118
DAG1_VTIPTDLIASSGDIK	Dystroglycan	Q14118
DIAC_ATYIQNYR	Di-N-acetylchitinase	Q01459
ENOG_GNPTVEVDLYTAK	Gamma-enolase	P09104
ENOG_LGAEVYHTLK	Gamma-enolase	P09104
ENPP2_SYPEILTLK	Ectonucleotide pyrophosphatase/phosphodiesterase family member 2	Q13822

ENPP2_WWGGQPLWITATK	Ectonucleotide pyrophosphatase/phosphodiesterase family member 2	Q13822
EXTL2_VIVVWNNIGEK	Exostosin-like 2	Q9UBQ6
FABPH_SIVTLDGGK	Fatty acid-binding protein, heart	P05413
FABPH_SLGVGFATR	Fatty acid-binding protein, heart	P05413
FAM3C_GINVALANGK	Protein FAM3C	Q92520
FAM3C_SALDTAAR	Protein FAM3C	Q92520
FAM3C_SPFEQHIK	Protein FAM3C	Q92520
FAM3C_TGEVLDTK	Protein FAM3C	Q92520
FBLN1_AITPPHPASQANIIFDITEGNLR	Fibulin-1	P23142
FBLN1_IIEVEEEQEDPYLNDR	Fibulin-1	P23142
FBLN1_TGYFDFGISR	Fibulin-1	P23142
FBLN3_IPSNPSHR	EGF-containing fibulin-like extracellular matrix protein 1	Q12805
FBLN3_LTIIVGPFSF	EGF-containing fibulin-like extracellular matrix protein 1	Q12805
FBLN3_SGNENGEFYLR	EGF-containing fibulin-like extracellular matrix protein 1	Q12805
FETUA_AHYDLR	Alpha-2-HS-glycoprotein	P02765
FETUA_FSVVYAK	Alpha-2-HS-glycoprotein	P02765
FETUA_HTLNQIDEVK	Alpha-2-HS-glycoprotein	P02765
FMOD_YLPFVPSR	Fibromodulin	Q06828
GFAP_ALAAELNQLR	Glial fibrillary acidic protein	P14136
GOLM1_DQLVIPDGQEEEQEAAGEGR	Golgi membrane protein 1	Q8NBJ4
GOLM1_QQLQALSEPQPR	Golgi membrane protein 1	Q8NBJ4
GRIA4_EYPGSETPPK	Glutamate receptor 4	P48058
GRIA4_LQNILEQIVSVGK	Glutamate receptor 4	P48058
GRIA4_NTDQEYTAFR	Glutamate receptor 4	P48058
HBA_FLASVSTVLTSK	Hemoglobin subunit alpha	P69905
HBA_TYFPHFDLSHGSAQVK	Hemoglobin subunit alpha	P69905
HBA_VGAHAGEYGAEALER	Hemoglobin subunit alpha	P69905
HBB_EFTPPVQAAYQK	Hemoglobin subunit beta	P68871
HBB_SAVTALWGK	Hemoglobin subunit beta	P68871
HBB_VNVDEVGGGEALGR	Hemoglobin subunit beta	P68871
HEMO_NFPSPVDAAFR	Hemopexin	P02790
HEMO_QGHNSVFLIK	Hemopexin	P02790
HEMO_SGAQATWTELPWPHEK	Hemopexin	P02790
I18BP_LWEGSTSR	Interleukin-18-binding protein	O95998
IBP2_HGLYNLK	Insulin-like growth factor-binding protein 2	P18065
IBP2_LIQGAPTIR	Insulin-like growth factor-binding protein 2	P18065

IGSF8_DTQFSYAVFK	Immunoglobulin superfamily member 8	Q969P0
IGSF8_LQGDVVLK	Immunoglobulin superfamily member 8	Q969P0
IGSF8_VVAGEVQVQR	Immunoglobulin superfamily member 8	Q969P0
ITIH1_EVAFDLEIPK	Inter-alpha-trypsin inhibitor heavy chain H1	P19827
ITIH1_QYYEGSEIVVAGR	Inter-alpha-trypsin inhibitor heavy chain H1	P19827
ITIH5_SYLEITPSR	Inter-alpha-trypsin inhibitor heavy chain H5	Q86UX2
KAIN_FYYLIASETPGK	Kallistatin	P29622
KAIN_LGFTDLFSK	Kallistatin	P29622
KAIN_VGSALFLSHNLK	Kallistatin	P29622
KAIN_WADLSGITK	Kallistatin	P29622
KLK10_ALQLPYR	Kallikrein-10	O43240
KLK11_LPHTLR	Kallikrein-11	Q9UBX7
KLK6_ESSQEQQSVVR	Kallikrein-6	Q92876
KLK6_LSELIQPLPLER	Kallikrein-6	Q92876
KLK6_YTNWIKQ	Kallikrein-6	Q92876
KNG1_DIPTNSPELEETLTHITK	Kininogen-1	P01042
KNG1_QVVAGLNFR	Kininogen-1	P01042
KNG1_TVGSDTFYFSK	Kininogen-1	P01042
KPYM_LDIDSPITAR	Pyruvate kinase isozymes M1/M2	P14618
L1CAM_AQLLVGSPGPVPR	Neural cell adhesion molecule L1	P32004
L1CAM_LVLSDLHLLTQSQVR	Neural cell adhesion molecule L1	P32004
L1CAM_WRPVDLAQVK	Neural cell adhesion molecule L1	P32004
LAMB2_AQGIAQGAIR	Laminin subunit beta-2	Q61292
LPHN1_LVVSQNLNPYTLR	Latrophilin-1	O94910
LPHN1_SGETVINTANYHDTSPYR	Latrophilin-1	O94910
LRC4B_HLEILQLSK	Leucine-rich repeat-containing protein 4B	Q9NT99
LRC4B_LTTVPTQAFEYLSK	Leucine-rich repeat-containing protein 4B	Q9NT99
LTBP2_EQDAPVAGLQPVER	Latent-transforming growth factor beta-binding protein 2	Q14767
MIME_ESAYLYAR	Mimecan	P20774
MIME_ETVIIPNEK	Mimecan	P20774
MIME_LEGNPVILGK	Mimecan	P20774
MOG_VVHLYR	Myelin-oligodendrocyte glycoprotein	Q16653
MUC18_EVTVPVFYPTK	Cell surface glycoprotein MUC18	P43121
MUC18_GATLALTQVTPQDER	Cell surface glycoprotein MUC18	P43121

NBL1_LALFPDK	Neuroblastoma suppressor of tumorigenicity 1	P41271
NCAM1_AGEQDATIHLK	Neural cell adhesion molecule 1	P13591
NCAM1_GLGEISAASEFK	Neural cell adhesion molecule 1	P13591
NCAM2_ASGSPEPAISWFR	Neural cell adhesion molecule 2	O15394
NCAM2_IELSQTAK	Neural cell adhesion molecule 2	O15394
NCAN_APVLELEK	Neurocan core protein	O14594
NCAN_LSSAIIAAPR	Neurocan core protein	O14594
NEGR1_SSIFAGGDK	Neuronal growth regulator 1	Q7Z3B1
NEGR1_VVNFAPTIQEI	Neuronal growth regulator 1	Q7Z3B1
NEGR1_WSVDPR	Neuronal growth regulator 1	Q7Z3B1
NELL2_AFLFQDTPR	Protein kinase C-binding protein NELL2	Q99435
NELL2_FTGSSWIK	Protein kinase C-binding protein NELL2	Q99435
NELL2_SALAYVDGK	Protein kinase C-binding protein NELL2	Q99435
NEO1_DVVASLVSTR	Neogenin	Q92859
NEUS_ALGITEIFIK	Neuroserpin	Q99574
NEUS_QEVPLATLEPLVK	Neuroserpin	Q99574
NGF_SAPAAIAAR	Beta-nerve growth factor	P01138
NICA_ALADVATVLGR	Nicastrin	Q92542
NICA_APDVTTLP	Nicastrin	Q92542
NPTX1_FQLTFPLR	Neuronal pentraxin-1	Q15818
NPTX1_LENLEQYSR	Neuronal pentraxin-1	Q15818
NPTX2_LESLEHQLR	Neuronal pentraxin-2	P47972
NPTX2_TESTLNALLQR	Neuronal pentraxin-2	P47972
NPTXR_ELDVLQGR	Neuronal pentraxin receptor	O95502
NPTXR_LVEAFGGATK	Neuronal pentraxin receptor	O95502
NRCAM_SLPSEASEQYLTK	Neuronal cell adhesion molecule	Q92823
NRCAM_VFNTPEGVPSAPSLK	Neuronal cell adhesion molecule	Q92823
NRCAM_YIVSGTPTFVPYLIK	Neuronal cell adhesion molecule	Q92823
NRX1A_DLFIDGQSK	Neurexin-1	Q9ULB1
NRX1A_ITTQITAGAR	Neurexin-1	Q9ULB1
NRX1A_SDLYIGGVAK	Neurexin-1	Q9ULB1
NRX2A_AIVADPVTFK	Neurexin-2	Q9P2S2
NRX2A_LGERPPALLGSQGLR	Neurexin-2	Q9P2S2
NRX2A_LSALTSTVK	Neurexin-2	Q9P2S2
NRX3A_IYGEVVK	Neurexin-3	Q9Y4C0
NRX3A_SDLFQFK	Neurexin-3	Q9Y4C0
OSTP_AIPVAQDLNAPSDWDSR	Osteopontin	P10451
PCSK1_ALAHLLEAER	ProSAAS	Q9UHG2

PCSK1_GEAAGAVQELAR	ProSAAS	Q9UHG2
PCSK1_NSDPALGLDDDPDAPAAQLAR	ProSAAS	Q9UHG2
PDYN_FLPSISTK	Proenkephalin-B	P01213
PDYN_LSGSFLK	Proenkephalin-B	P01213
PDYN_SVGEGPYSELAK	Proenkephalin-B	P01213
PEDF_DTDGTGALLFIGK	Pigment epithelium-derived factor	P36955
PEDF_SSFVAPLEK	Pigment epithelium-derived factor	P36955
PEDF_TVQAVLTVPK	Pigment epithelium-derived factor	P36955
PGRP2_AGLLRPDYALLGHR	N-acetylmuramoyl-L-alanine amidase	Q96PD5
PGRP2_TFTLLDPK	N-acetylmuramoyl-L-alanine amidase	Q96PD5
PIMT_VQLVVGDR	Protein-L-isoaspartate(D-aspartate) O-methyltransferase	P22061
PLDX1_LYGPSEPHSR	Plexin domain-containing protein 1	Q8IUU5
PLMN_EAQLPVIENK	Plasminogen	P00747
PLMN_HSIFTPETNPR	Plasminogen	P00747
PLMN_LSSPAVITDK	Plasminogen	P00747
PPN_VHQSPDGTLLIYNLR	Papilin	O95428
PRDX1_DISLSYK	Peroxiredoxin-1	Q06830
PRDX1_LVQAFQFTDK	Peroxiredoxin-1	Q06830
PRDX2_GLFIDGK	Peroxiredoxin-2	P32119
PRDX2_IGKPAPDFK	Peroxiredoxin-2	P32119
PRDX3_HLSVNDLPVGR	Thioredoxin-dependent peroxide reductase, mitochondrial	P30048
PRDX6_LIALSIDSVEDHLAWSK	Peroxiredoxin-6	P30041
PRDX6_LSIYPATTGR	Peroxiredoxin-6	P30041
PTGDS_AQGFTEDTIVFLPQTDK	Prostaglandin-H2 D-isomerase	P41222
PTGDS_WFSAGLASNSSWLR	Prostaglandin-H2 D-isomerase	P41222
PTPRN_AEAPALFSR	Receptor-type tyrosine-protein phosphatase-like N	Q16849
PTPRN_LAAVLGYGVLELR	Receptor-type tyrosine-protein phosphatase-like N	Q16849
PTPRN_SELEAQTGLQILQTGVGQR	Receptor-type tyrosine-protein phosphatase-like N	Q16849
PVRL1_ITQVTWQK	Poliovirus receptor-related protein 1	G3IGH2
SCG1_GEAGAPGEEDIQGPTK	Secretogranin-1	P05060
SCG1_HLEEPGETQNAFLNER	Secretogranin-1	P05060
SCG1_NYLNNGEAGAPGK	Secretogranin-1	P05060

SCG1_SSQGGSLPSEEK	Secretogranin-1	P05060
SCG2_ALEYIENLR	Secretogranin-2	P13521
SCG2_IILEALR	Secretogranin-2	P13521
SCG2_VLEYLNQEK	Secretogranin-2	P13521
SCG3_ELSEARPLNEQIAEAEDK	Secretogranin-3	Q8WXD2
SCG3_FQDDPDGLHQLDGTPLTAEDIV HK	Secretogranin-3	Q8WXD2
SCG3_LNVEDVDSTK	Secretogranin-3	Q8WXD2
SE6L1_ETGTPIWTSR	Seizure 6-like protein	Q9BYH1
SE6L1_SPTNTISVYFR	Seizure 6-like protein	Q9BYH1
SIAE_ELSNTAAYQSVR	Sialate O-acetyltransferase	Q9HAT2
SLIK1_SLPVDVFAGVSLSK	SLIT and NTRK-like protein 1	Q96PX8
SODC_GDGPVQGIINFEQK	Superoxide dismutase [Cu-Zn]	P00441
SODC_HVGDLGNVTADK	Superoxide dismutase [Cu-Zn]	P00441
SODC_TLVVHEK	Superoxide dismutase [Cu-Zn]	P00441
SODE_AGLAASLAGPHSIVGR	Extracellular superoxide dismutase [Cu-Zn]	P08294
SODE_AVVVHAGEDDLGR	Extracellular superoxide dismutase [Cu-Zn]	P08294
SODE_VTGVLFR	Extracellular superoxide dismutase [Cu-Zn]	P08294
SORC1_TIAVYEEFR	VPS10 domain-containing receptor SorCS1	Q8WY21
SPON1_VTLAAPPYFR	Spondin-1	Q9HCB6
SPRL1_HIQETEWQSQEGK	SPARC-like protein 1	Q14515
SPRL1_HSASDDYFIPSQAFLEAER	SPARC-like protein 1	Q14515
SPRL1_VLTHSELAPLR	SPARC-like protein 1	Q14515
TGFB1_LLAPSDSPEWLSFDVTGVVR	Transforming growth factor beta- 1	P01137
THRB_ETAASLLQAGYK	Prothrombin	P00734
THRB_YGFYTHVFR	Prothrombin	P00734
TIMP1_GFQALGDAADIR	Metalloproteinase inhibitor 1	P01033
TIMP1_SEEFLIAGK	Metalloproteinase inhibitor 1	P01033
TNR21_ASNLIGTYR	Tumor necrosis factor receptor superfamily member 21	O75509
TRFM_ADTDGGLIFR	Melanotransferrin	P08582
TTHY_TSESGELHGLTTEEFVEGIYK	Transthyretin	P02766
TTHY_VEIDTK	Transthyretin	P02766
UBB_ESTLHLVLR	Polyubiquitin-B	P0CG47
UBB_TITLVEPSDTIENVK	Polyubiquitin-B	P0CG47
UBB_TLSDYNIQK	Polyubiquitin-B	P0CG47
VASN_NLHDLVDSDNQLER	Vasorin	Q6EMK4
VASN_SLTLGIEPVSPSLR	Vasorin	Q6EMK4

VASN_YLQGSSVQLR	Vasorin	Q6EMK4
VGf_AYQGVAAPFPK	Neurosecretory protein VGf	O15240
VGf_NSEPQDEGELFQGVDPK	Neurosecretory protein VGf	O15240
VGf_THLGEALAPLSK	Neurosecretory protein VGf	O15240
VTDB_EFSHLGK	Vitamin D-binding protein	P02774
VTDB_HLSLLTTLSNR	Vitamin D-binding protein	P02774
VTDB_VPTADLEDVLPLAEDITNILSK	Vitamin D-binding protein	P02774