

1 **SARS-CoV-2 ORF1ab^{A1061S} potentiate autoreactive T cell responses**
2 **via epitope mimicry: an explanation to hepatitis of unknown cause**

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12

13 Abstract

14 The World Health Organization have recently announced outbreak news of acute,
 15 severe hepatitis of unknown cause in children under a Covid-19 pandemic. Whether it
 16 is associated with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)
 17 infection is still under debating. Here, we performed genomic sequence alignment
 18 analysis of the genome of SARS-Cov-2 (Wuhan-hu-1) to the human genome
 19 reference. Sequence analysis revealed that the SARS-CoV-2 ORF1ab¹⁰⁵⁶⁻¹¹⁷³
 20 presented high identities with the human protein PAPR14⁵³⁻¹⁷⁶(3Q6Z_A). After
 21 searching the fully sequenced SARS-CoV-2 genomes deposited in GISAID
 22 (<https://www.gisaid.org/>), we detected 170 SARS-CoV-2 variants with mutation in
 23 ORF1ab¹⁰⁶¹, where alanine (A) was substituted by serine (S). This alteration made a
 24 7-amino acid peptide (VVVNASN) in ORF1ab¹⁰⁵⁶⁻¹⁰⁶² identical to its counterpart in
 25 PARP14⁵³⁻⁵⁹(3Q6Z_A). HLA prediction suggested that the peptides with high
 26 identities in PARP14 and ORF1ab could be presented by a same globally prevalent
 27 HLA-A*11:01 molecule. And in consistent with the first reported case of hepatitis of
 28 unknown, SARS-CoV-2 ORF1ab^{VVVNASN} variants were mostly identified as Delta
 29 lineages in UK by the late 2021, with an overall frequency of 0.00161%. Thus, our
 30 preliminary results raised a possibility that infection by SARS-CoV-2
 31 ORF1ab^{VVVNASN} variant might elicit an autoimmune T cell response via epitope
 32 mimicry and is associated with the outbreak of unknown hepatitis. We anticipated that
 33 these findings will alert the human societies to pay more attention to rare mutations
 34 beyond the spike proteins.

35 Key words

36 SARS-CoV-2, Hepatitis of unknown cause, Epitope mimicry, ORF1ab, A1061S
 37 mutation

38

39 Main text

40 The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) have caused a
41 global pandemic in the last two years and are still evolving nowadays. Although we
42 have made great progresses in understanding their virology and key steps of life cycle
43 [1], long-term and systematic impacts of these virus on human are still missing,
44 especially of that on the systemic immune status. Beside SARS-CoV-2, the World
45 Health Organization have announced outbreak news of acute, severe hepatitis of
46 unknown origin in children on 23, April 2022 [2]. And according to the records by
47 European Centre for Disease Prevention and Control, the total number of cases
48 reported worldwide were approximately 450 by 11, May 2022[3]. All of the patients
49 were found in children under 16 years old, and it was estimated that more than 10% of
50 the children have required liver transplantation [2].

51 The etiology of this disease is current unknown. However, some of these children
52 have been identified with ongoing or recent SARS-CoV-2 infections in Israel and the
53 USA [2]. Although links between long-lasting effects from the SARS-CoV-2
54 infection with hepatitis of unknown origin were currently without experimental
55 examination, a case report highlighted a possible association between SARS-CoV-2
56 infection and subsequent development of T cell participated autoimmune liver
57 disease[4]. In supporting, SARS-CoV-2 superantigens have been hypothesized to
58 involve in excessive T cell activation and possibly the pathogenesis of hepatitis of
59 unknown cause[5].

60 T cell responses are critically important to eliminate virus infection and also for
61 SARS-CoV-2[6]. The elimination of virus finally resulted in a persistent T cell-pools
62 that processing diverse T cell receptor (TCR) repertoires to virus related antigens,
63 thus leaving a protection for the secondary infections by the same virus. Typically,
64 these T cells were kept in check and did not attack human tissues. However, in some
65 cases, virus infection led to immune disorders and caused autoimmune diseases,
66 which were related to the disturbed peripheral tolerance[7, 8]. In the cases of SARS-
67 CoV-2 infections, severe immune perturbations have been noted[9], thus rising
68 possibilities that impaired peripheral T cell tolerance would occur.

69 Based on that knowledge, we hypothesized that virus mutations might generate new

70 antigens that mimicked self-peptides and elicited an autoimmune response in
71 appropriate inflammatory microenvironments, and thus associated with the emerging
72 of hepatitis of unknown cause.

73 **PSI-blast identified a motif with high identity between human PARP14 and** 74 **SARS-CoV-2 ORF1ab**

75 To test the hypothesis above, we made a whole-genome alignment of SARS-CoV-2
76 (wh-hu-1)[10] to the human reference genome with PSI-blast in default parameters.
77 Among all the SARS-CoV-2 proteins aligned, only a hit in ORF1ab (ORF1ab¹⁰⁵⁶⁻¹¹⁷³)
78 was found, which presented high identity with a sequence in human PARP14
79 (PARP14⁵³⁻¹⁷⁶, sequence ID: 3Q6Z_A) (**Fig. 1A**). The most identical peptide was
80 located in a motif with 7 amino acids residuals, in which only a differential amino
81 acid was found in ORF1ab¹⁰⁶¹ comparing to PARP14⁵⁸ (**Fig. 1A**). In addition,
82 PAPR14⁶²⁻⁷⁵(sequence ID: 3Q6Z_A) also shared high identity with ORF1ab¹⁰⁶⁵⁻
83 ¹⁰⁷⁸(**Fig. 1A**).

84 **Rare mutation increased identity between ORF1ab¹⁰⁵⁶⁻¹⁰⁶² and PARP14⁵³⁻⁵⁹**

85 Since the release of the first sequence of SARS-CoV-2 (wuhan-hu-1) in December
86 2019, the virus has undergone numerous mutations. The accumulation of mutations in
87 the genome of SARS-CoV-2 will affect functional properties and may alter
88 infectivity, disease severity or interactions with host immunity. It was reasonable to
89 speculate that there might be some SARS-CoV-2 variants with mutations occurring in
90 ORF1ab¹⁰⁵⁶⁻¹⁰⁶², thus resulting in an increased identity to human PARP14⁵³⁻⁵⁹
91 (3Q6Z_A). To test that hypothesis, we downloaded the proteins of SARS-CoV-2
92 variants deposited on GISAID (<https://www.gisaid.org/>). Sequence analysis was focus
93 on the amino acid sequence from 1055 to 1077 on ORF1a protein. Among all the
94 SARS-CoV-2 protein sequences (10,541,935 in total, by May 10th, 2022), 170
95 possessed an alanine (A) to serine (S) substitution on the site 1061 of ORF1ab (**Fig.**
96 **1B and 1C**). Noteworthy, this alteration made the core amino acid sequences
97 identical to that of human PARP14⁵³⁻⁵⁹ (3Q6Z_A). Further analysis revealed that
98 SARS-CoV-2 ORF1ab^{VVVNASN} variants has been recorded in 15 countries across 5
99 continents (**Fig. 1B and 1C**). The variants varied by countries with an overall
100 frequency of 0.00161% worldwide (**Fig. 1C**). In consistent with the firstly reported

101 cases of hepatitis of unknown, SARS-CoV-2 ORF1ab^{VVNASN} variants was mostly
102 detected in the UK (135) and the USA (18) (**Fig. 1D**).

103 In addition to ORF1ab^{A1061S} substitution, several other mutations that would
104 potentially increase the sequence identity were also identified. For example,
105 ORF1ab^{G1073A} have been emerging at a relative high frequency (**Fig. 2A, table 1**).
106 This mutation led a 11-amino acid motif in ORF1ab (LKHGGGVAAAL) to be more
107 similar in chemical properties, comparing to human PARP14 (LKHYGGLAAAL)
108 (**Fig. 2A**). The 170 variants bearing A1061S mutation in ORF1ab were also annotated
109 to the Variants of Concern (VOC) and most of mutational variants are found within
110 SARS-CoV-2 Delta lineage (**Fig. 2B**). Although the Omicron variants were causing
111 most of the infections globally, an Israel research group has warned that Delta
112 variants were still undergoing circulating in parallel to Omicron variants and might
113 maintain its circulation in future[11].

114 **HLA prediction suggested potential overlaps of HLA in binding human and** 115 **virus peptides**

116 T cells recognized peptides presented by specific array of HLA molecules. Thus,
117 HLA overlapping was a pre-determinant for cross T cell reactivities induced by
118 epitope mimicry. We used an online HLA binding prediction tool
119 (<http://tools.iedb.org/mhcii/>) to predict peptide binding abilities by a default array of
120 MHC-I molecules, allowing to evaluate the binding potential and usage overlaps of
121 HLA molecules by high similar peptides from human PARP14 and SARS-CoV2
122 ORF1ab. We selected a total of 23 amino acid surrounding the identical 7-amino acid
123 of ORF1ab^{A1061S} and PARP14 as inputs respectively. As expected, we found that the
124 peptide VVNASNELK in human PARP14 and VVNASNVYK in ORF1ab^{A1061S}
125 could be presented by a same HLA-A*11:01 molecule with comparable high-affinity
126 (**Fig. 2C**). Importantly, OFR1ab^{A1061S} mutation showed increased binding ability of
127 this peptide to HLA-A*11:01 molecule, as comparing to its wuhan-hu-1 counterpart
128 (**Fig. 2C**). These findings suggested both the self- and virus- peptides with high
129 sequence identity could be presented by a same HLA molecule, supporting that
130 specific T-cell clones restricted to an HLA-A*11:01 molecules might be cross-
131 activated by virus peptides mimicry and led to autoimmune responses . Indeed, HLA-
132 A*11:01 was one of the most prevalent HLAs across the world [8], suggesting virus

133 antigen presentation by HLA-A*11:01 were generally applicable to the worldwide.

134 **Discussion**

135 Deregulated T cell responses are common triggers of various autoimmune diseases.
136 Epitope mimicry of host proteins by pathogen is a common inducer in susceptible
137 individuals to induce biased immune response versus tolerance, leading to tissue
138 damage[12]. Here we found a mutation in SARS-CoV-2 ORF1ab may lead to
139 increased identity between pathogen peptides with human proteins, providing a
140 computational evidence for understanding the leading cause of SARS-CoV-2
141 associated autoimmune diseases, and also provided a new thought on the outbreaks of
142 children hepatitis of unknown cause under a background of SARS-CoV-2 pandemic.

143 The outbreak of hepatitis of unknown cause was currently restricted to children under
144 16 years old. It should be noted that in children, their thymus output was kept in stack,
145 T cell repertoires were continuously making, and peripheral tolerance was under
146 establishing [13]. The disturbance of systematic and local immune microenvironment
147 by SARS-CoV2 infection was likely to affect the establishment of normal T cell
148 tolerances, thus possibly explained the preference of children to this type of hepatitis.
149 Based on that, we anticipated that HLA genotyping may facilitate to uncovering the
150 real cause of hepatitis.

151 According to the fully sequenced genomes deposited in GISAID, the frequency of
152 SARS-CoV-2 ORF1ab^{VVVNASN} variants was around 0.0000161. Among 517,648,631
153 confirmed SARS-CoV-2 cases by 15, May 2022 (<https://covid19.who.int/>), it was
154 roughly estimated that 8334 people, irrespective of children or adults, might be
155 subjected to risks of autoimmune T cell responses and possibly the hepatitis of
156 unknown cause. However, other unknown factors may also participate to exacerbate
157 disease morbidity and severity, and indeed we have noted some mutations will
158 increase the sequence identity between ORF1ab¹⁰⁶⁵⁻¹⁰⁷⁸ and PAPR14⁶²⁻⁷⁵(3Q6Z_A).
159 Most of the variants bearing A1061S substitution were in Delta lineage, and it should
160 be noteworthy that Delta variants were still circulating in parallel with Omicron
161 variants[11]. Thus, mutations in these sites should call for our great concerns.
162 However, our results were still preliminary and we only aimed to discussing a
163 possible association of SARS-CoV-2 infection with children acute hepatitis of

164 unknown cause. Further experimental validation of this hypothesis presented here was
165 urgently needed to figure out the nature of hepatitis of unknown cause.

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169 Both Yu Wang and Yuexing Liu contributed to the conceptional design and data
170 processing.

Conflict of interest:

172 The authors declared no conflict of interest.

Fundings and ethics:

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209

210

211 **Figure legends:**

212 **Figure 1. SARS-CoV-2 ORF1ab^{A1061S} substitution increased sequence identity**
 213 **with human PARP14 and variant's epidemiology.**

214 (A) PSI blast showing the alignment of SARS-CoV-2 ORF1ab¹⁰⁵⁶⁻¹¹⁷³ with
 215 PARP14⁵³⁻¹⁶⁸(3Q6Z_A). (B) Global distribution of the fully sequenced SARS-CoV-2
 216 genomes possessing A1601S mutation in SARS-CoV-2 ORF1ab VVVNAAN motif.
 217 (Basal layer map was created with BioRender.com)(C) Counts (and proportions) of
 218 ORF1ab^{VVVNASN} variants across 15 countries. (D) Monthly trends in accumulated
 219 number of ORF1ab^{VVVNASN} variants across 15 countries.

220 **Figure 2. Sequence analysis of variants bearing ORF1ab^{A1061S} substitution.**

221 (A) Mutation preferences and rates in SARS-CoV-2 ORF1ab¹⁰⁵⁵⁻¹⁰⁷⁹ as compared to
 222 wuhan-hu-1. (B) Pie plot showing phylogenetic assignment of the SARS-CoV-2
 223 variant (ORF1ab^{VVVNASN}) with pangolin[14]. (C) Predicted binding ability by HLA-
 224 A*11:01 to human PARP14 and SARS-CoV-2 ORF1ab generated peptide.

Figure 1. SARS-CoV-2 ORF1ab^{A1061S} substitution increased sequence similarities with human PARP14 and variant's epidemiology.

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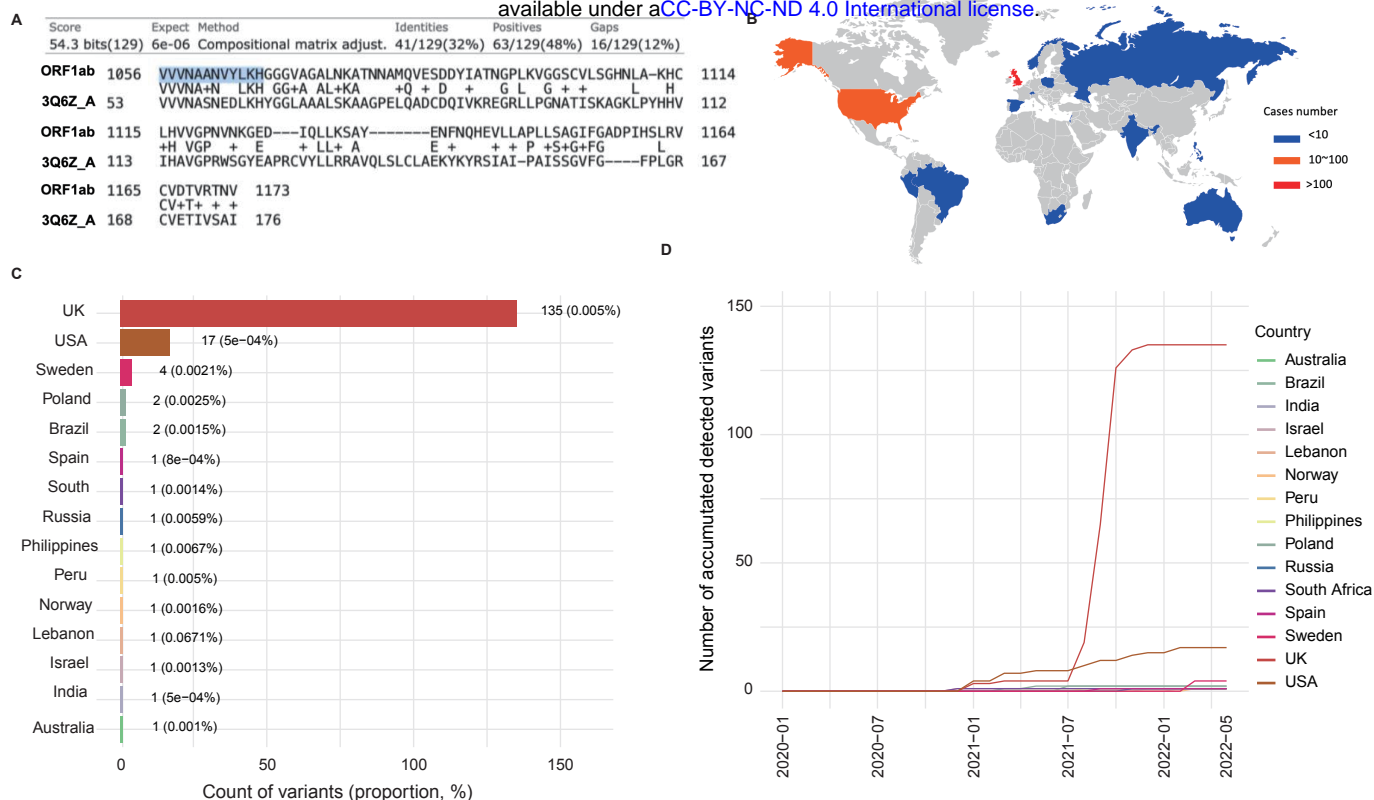


Figure 2. Sequence analysis of variants bearing OFR1ab^{A1061S} substitution.

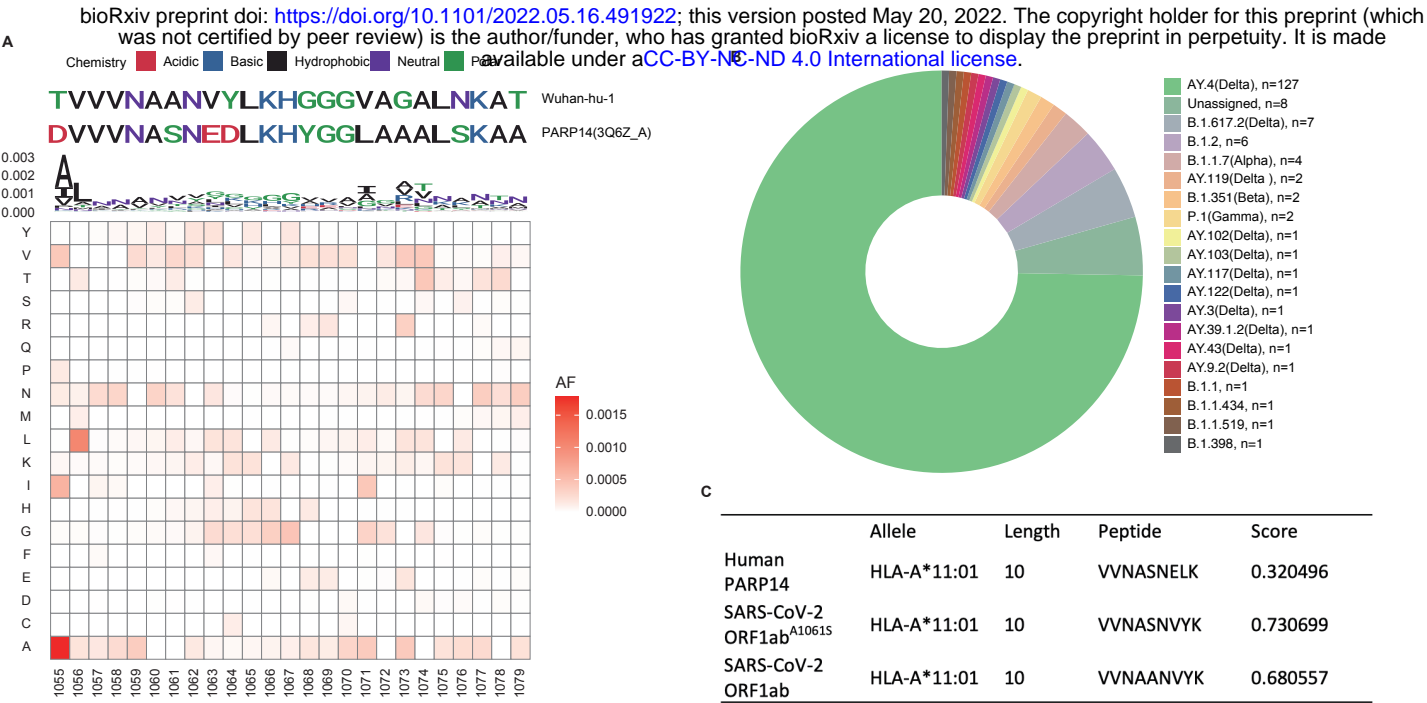


Table1. Mutation frequency in SARS-CoV-2 ORF1ab¹⁰⁵⁵⁻¹⁰⁷⁹ as compared to wuhan_hu_1
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position	composite	allele_frequency	position	composite	allele_frequency	position	composite	allele_frequency
1055	T	0.9968422	1063	Q	1.25E-06	1071	Y	2.69E-06
1055	A	0.0017836	1063	S	9.60E-07	1071	S	2.50E-06
1055	I	6.00E-04	1063	C	7.68E-07	1071	P	1.15E-06
1055	V	4.14E-04	1063	M	5.76E-07	1071	C	9.60E-08
1055	P	1.35E-04	1063	P	3.84E-07	1072	A	0.9993339
1055	N	1.26E-04	1063	R	9.60E-08	1072	G	1.94E-04
1055	K	4.59E-05	1064	Y	0.9989918	1072	V	1.60E-04
1055	G	2.19E-05	1064	G	2.14E-04	1072	N	1.20E-04
1055	Y	1.39E-05	1064	K	1.91E-04	1072	K	6.73E-05
1055	L	1.06E-05	1064	L	1.80E-04	1072	L	4.22E-05
1055	H	2.78E-06	1064	V	1.52E-04	1072	T	3.10E-05
1055	S	1.15E-06	1064	C	1.09E-04	1072	E	1.58E-05
1055	M	1.06E-06	1064	H	7.22E-05	1072	H	1.01E-05
1055	R	8.64E-07	1064	A	4.22E-05	1072	S	7.68E-06
1055	D	3.84E-07	1064	N	2.35E-05	1072	Y	6.05E-06
1055	Q	1.92E-07	1064	I	1.21E-05	1072	D	3.07E-06
1055	E	9.60E-08	1064	D	2.98E-06	1072	Q	3.07E-06
1056	V	0.9983827	1064	M	2.69E-06	1072	I	2.69E-06
1056	L	0.0010483	1064	S	2.40E-06	1072	M	2.30E-06
1056	A	1.82E-04	1064	T	1.15E-06	1072	P	1.06E-06
1056	T	1.32E-04	1064	E	9.60E-07	1072	C	9.60E-08
1056	M	1.05E-04	1064	Q	5.76E-07	1073	G	0.9982889
1056	N	8.86E-05	1064	F	3.84E-07	1073	A	3.97E-04
1056	K	2.53E-05	1064	P	9.60E-08	1073	V	3.79E-04
1056	G	1.90E-05	1065	L	0.9991379	1073	R	3.39E-04
1056	Y	8.16E-06	1065	G	2.15E-04	1073	L	1.80E-04
1056	H	2.78E-06	1065	K	1.91E-04	1073	E	1.62E-04
1056	I	1.44E-06	1065	H	1.80E-04	1073	K	1.06E-04
1056	P	1.34E-06	1065	Y	1.30E-04	1073	N	6.81E-05
1056	E	1.15E-06	1065	A	5.36E-05	1073	T	4.62E-05
1056	Q	9.60E-07	1065	V	4.85E-05	1073	M	7.78E-06
1056	D	7.68E-07	1065	N	1.18E-05	1073	I	6.05E-06
1056	S	1.92E-07	1065	F	7.01E-06	1073	S	5.57E-06
1057	V	0.9994063	1065	T	6.34E-06	1073	H	5.38E-06
1057	N	2.45E-04	1065	M	5.95E-06	1073	D	2.88E-06
1057	A	1.52E-04	1065	D	4.80E-06	1073	Q	2.40E-06
1057	I	6.82E-05	1065	I	2.88E-06	1073	P	1.73E-06
1057	F	3.20E-05	1065	Q	2.69E-06	1073	Y	1.25E-06
1057	K	2.76E-05	1065	P	1.92E-06	1073	C	8.64E-07
1057	Y	1.79E-05	1065	E	1.34E-06	1074	A	0.9984284
1057	G	1.67E-05	1065	S	7.68E-07	1074	T	4.19E-04
1057	L	1.57E-05	1065	C	9.60E-08	1074	V	4.11E-04
1057	H	7.68E-06	1066	K	0.9990457	1074	N	2.43E-04
1057	D	3.65E-06	1066	G	3.42E-04	1074	L	1.80E-04
1057	Q	2.59E-06	1066	H	1.78E-04	1074	G	1.56E-04
1057	P	2.21E-06	1066	L	1.33E-04	1074	K	5.30E-05
1057	T	1.44E-06	1066	A	7.36E-05	1074	S	4.79E-05
1057	S	8.64E-07	1066	V	6.61E-05	1074	D	3.95E-05
1057	E	4.80E-07	1066	R	5.33E-05	1074	Q	7.68E-06
1057	M	2.88E-07	1066	E	4.19E-05	1074	M	4.13E-06
1058	V	0.9992974	1066	N	3.49E-05	1074	E	2.88E-06
1058	N	2.93E-04	1066	T	1.23E-05	1074	Y	2.21E-06
1058	A	2.39E-04	1066	Q	6.15E-06	1074	P	1.44E-06
1058	Y	4.56E-05	1066	I	5.28E-06	1074	C	1.15E-06

1058	I	3.90E-05	1066	D	2.50E-06	1074	I	9.60E-07
1058	L	0.809105	1066	S	1.92E-06	1074	L	0.991155
1058	K	2.22E-05	1066	M	1.06E-06	1075	N	2.88E-04
1058	G	1.38E-05	1066	P	9.60E-07	1075	K	1.91E-04
1058	P	9.22E-06	1067	H	0.9989511	1075	A	1.90E-04
1058	H	4.03E-06	1067	G	4.45E-04	1075	T	1.11E-04
1058	T	3.74E-06	1067	Y	1.53E-04	1075	V	2.33E-05
1058	D	1.25E-06	1067	K	1.37E-04	1075	I	1.99E-05
1058	E	1.15E-06	1067	V	1.23E-04	1075	G	1.69E-05
1058	M	1.15E-06	1067	A	9.54E-05	1075	D	1.44E-05
1058	Q	2.88E-07	1067	Q	3.48E-05	1075	M	8.74E-06
1058	F	1.92E-07	1067	N	3.27E-05	1075	S	6.05E-06
1058	S	1.92E-07	1067	L	1.06E-05	1075	Q	4.13E-06
1059	N	0.9991649	1067	E	3.36E-06	1075	F	3.55E-06
1059	A	3.72E-04	1067	T	3.17E-06	1075	P	3.26E-06
1059	V	2.44E-04	1067	R	2.98E-06	1075	E	2.40E-06
1059	Y	6.04E-05	1067	D	2.11E-06	1075	C	9.60E-07
1059	L	4.78E-05	1067	M	2.11E-06	1075	Y	8.64E-07
1059	K	2.83E-05	1067	P	1.54E-06	1075	H	2.88E-07
1059	T	2.24E-05	1067	S	1.06E-06	1076	N	0.9992106
1059	G	1.95E-05	1067	I	8.64E-07	1076	A	2.37E-04
1059	S	1.50E-05	1067	C	5.76E-07	1076	K	1.81E-04
1059	P	1.01E-05	1068	G	0.9991909	1076	L	1.30E-04
1059	H	8.83E-06	1068	V	2.00E-04	1076	S	8.46E-05
1059	E	2.78E-06	1068	A	1.65E-04	1076	T	4.67E-05
1059	D	1.82E-06	1068	H	1.29E-04	1076	V	2.83E-05
1059	Q	1.15E-06	1068	E	1.25E-04	1076	D	2.56E-05
1059	M	8.64E-07	1068	R	1.04E-04	1076	M	1.76E-05
1059	I	5.76E-07	1068	N	2.52E-05	1076	G	1.12E-05
1059	C	9.60E-08	1068	L	2.01E-05	1076	Q	8.74E-06
1060	A	0.9992057	1068	D	1.10E-05	1076	E	7.87E-06
1060	N	3.11E-04	1068	Y	9.89E-06	1076	Y	5.38E-06
1060	V	1.39E-04	1068	K	5.76E-06	1076	C	2.59E-06
1060	Y	1.08E-04	1068	T	4.90E-06	1076	H	1.15E-06
1060	L	6.06E-05	1068	S	2.88E-06	1076	I	9.60E-07
1060	K	4.70E-05	1068	Q	2.21E-06	1076	P	6.72E-07
1060	T	3.67E-05	1068	I	1.54E-06	1077	K	0.9989977
1060	G	3.14E-05	1068	P	1.15E-06	1077	N	3.68E-04
1060	S	2.14E-05	1068	M	7.68E-07	1077	A	2.51E-04
1060	H	1.76E-05	1068	C	2.88E-07	1077	T	1.86E-04
1060	D	1.08E-05	1069	G	0.9992562	1077	M	5.09E-05
1060	P	4.42E-06	1069	V	2.04E-04	1077	R	3.22E-05
1060	E	3.07E-06	1069	R	1.57E-04	1077	E	2.16E-05
1060	M	1.73E-06	1069	E	1.25E-04	1077	V	1.95E-05
1060	Q	8.64E-07	1069	A	1.16E-04	1077	G	1.88E-05
1060	I	4.80E-07	1069	L	5.56E-05	1077	Q	1.76E-05
1060	C	9.60E-08	1069	N	2.47E-05	1077	Y	1.06E-05
1061	A	0.999099	1069	K	1.15E-05	1077	S	8.93E-06
1061	V	2.67E-04	1069	Y	1.10E-05	1077	I	5.76E-06
1061	N	1.92E-04	1069	I	1.02E-05	1077	D	5.19E-06
1061	T	1.18E-04	1069	T	8.83E-06	1077	P	2.88E-06
1061	L	1.10E-04	1069	S	6.34E-06	1077	L	2.50E-06
1061	K	6.26E-05	1069	M	5.28E-06	1077	C	2.88E-07
1061	H	4.57E-05	1069	D	3.36E-06	1077	H	9.60E-08
1061	Y	4.02E-05	1069	P	2.59E-06	1078	A	0.9990958
1061	G	3.40E-05	1069	Q	8.64E-07	1078	T	2.55E-04
1061	S	1.71E-05	1069	H	6.72E-07	1078	N	2.23E-04
1061	D	4.13E-06						

1061	I	3.36E-06
1061	Q	1.82E-06
1061	M	7.68E-07
1061	P	4.80E-07
1061	C	9.60E-08
1062	N	0.9990169
1062	V	2.17E-04
1062	Y	1.80E-04
1062	A	1.45E-04
1062	S	1.25E-04
1062	K	1.08E-04
1062	G	7.45E-05
1062	H	6.12E-05
1062	L	4.96E-05
1062	T	1.19E-05
1062	D	7.11E-06
1062	E	1.06E-06
1062	M	1.06E-06
1062	Q	9.60E-07
1062	I	4.80E-07
1062	P	9.60E-08
1063	V	0.9988616
1063	G	2.42E-04
1063	Y	1.90E-04
1063	L	1.82E-04
1063	N	1.56E-04
1063	I	1.05E-04
1063	H	1.05E-04
1063	A	5.28E-05
1063	F	4.79E-05
1063	K	4.74E-05
1063	T	3.17E-06
1063	E	1.92E-06
1063	D	1.44E-06

1069	C	9.60E-08
1070	A	2.98E-04
1070	V	1.95E-04
1070	L	7.35E-05
1070	N	5.66E-05
1070	C	5.19E-05
1070	S	4.82E-05
1070	D	4.03E-05
1070	K	2.84E-05
1070	M	9.12E-06
1070	T	6.63E-06
1070	Q	5.47E-06
1070	Y	4.80E-06
1070	H	2.40E-06
1070	E	2.11E-06
1070	I	1.34E-06
1070	P	5.76E-07
1070	R	3.84E-07
1070	F	9.60E-08
1071	V	0.9985597
1071	I	4.22E-04
1071	A	3.91E-04
1071	G	3.14E-04
1071	L	1.10E-04
1071	N	7.37E-05
1071	K	6.11E-05
1071	F	1.39E-05
1071	E	1.03E-05
1071	T	9.60E-06
1071	Q	9.22E-06
1071	H	9.12E-06
1071	D	7.20E-06
1071	M	2.88E-06

1078	K	1.29E-04
1078	V	8.90E-05
1078	M	5.98E-05
1078	Q	4.56E-05
1078	S	2.14E-05
1078	D	1.86E-05
1078	P	1.56E-05
1078	G	1.39E-05
1078	I	9.12E-06
1078	L	8.83E-06
1078	E	8.74E-06
1078	Y	2.98E-06
1078	H	1.34E-06
1078	C	1.15E-06
1079	T	0.9991392
1079	N	3.63E-04
1079	A	1.99E-04
1079	M	1.05E-04
1079	Q	5.98E-05
1079	V	5.00E-05
1079	E	1.76E-05
1079	L	1.56E-05
1079	S	1.38E-05
1079	D	1.18E-05
1079	G	1.03E-05
1079	I	4.90E-06
1079	K	4.90E-06
1079	Y	2.30E-06
1079	P	1.44E-06
1079	C	1.15E-06
1079	H	2.88E-07