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# *Emergence of a New Creutzfeldt-Jakob Disease: 26 Cases of the Human Version of Mad-Cow Disease, Days After a COVID-19 Injection*

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*Luc Montagnier, MD, and Nobel Laureate, esteemed colleague and friend, passed from this world on February 8, 2022 not long after the completion of the preliminary draft of this work which his co-authors have carried forward to this updated report with some additional cases and new information. Perhaps this may be the most important work of Luc's lifetime expressing his incredible genius and spirit. While hospitalized, he continued to attach the greatest importance to the publication of this article. He is honored by the Luc Montagnier Foundation Quai Gustave-Ador 62 1207, Geneva, Switzerland.*

## ABSTRACT

Creutzfeldt-Jakob Disease, the formerly rare but universally fatal prion disease in humans, normally progresses over several decades before it leads to death. In the Appendix to this paper, we highlight the presence of a prion region in the spike protein of the original SARS-CoV-2, and in all the "vaccine" variants built from the Wuhan virus. The prion region in the spike of SARS-CoV-2 has a density of mutations eight times greater than that of the rest of the spike, and, yet, strangely that entire prion region disappears completely in the Omicron variant. In the main body of our text, we present 26 cases of Creuzfeldt-Jacob Disease, all diagnosed in 2021 with the first symptoms appearing within an average of 11.38 days after a Pfizer, Moderna, or AstraZeneca COVID-19 injection. Because the causal progression, the etiopathogenesis, of these atypical and new cases of human prion disease — cases of what is apparently a totally new form of rapidly developing Creuzfeldt-Jacob Disease — we focus on the chronology of the symptomatic development. We consider it from an anamnestic point of view — one in which we compare the typical development of pre-COVID cases of Creuzfeldt-Jacob Disease to the extremely accelerated development of similar symptoms in the 26 cases under examination. By such an approach, we hope to work out the etiopathogenesis critical to understanding this new and much more rapidly developing human prion disease. By recalling the sequential pathway of that the formerly subacute and slowly developing disease followed in the past, and by comparing it with this new, extremely acute, rapidly developing prion disease — one following closely after one or more of the COVID-19 injections — we believe it is correct to infer that the injections caused the disease in these 26 cases. If so, they have probably also caused a many other cases that have gone undiagnosed because of their rapid progression to death. By late 2021, 20 had died within 4.76 months of the offending injection. Of those, 8 died suddenly within 2.5 months confirming the rapid progression of this accelerated form of Creuzfeldt-Jacob Disease. By June 2022, 5 more patients had died, and at the time of this current writing, only 1 remains still alive.

**Keywords:** *Creutzfeldt-Jacob Disease, onset of CJD, prion protein, SARS-CoV-2 variants, spike protein, COVID-19 mRNA vaccines, neuropsychiatric disease, evolution of the COVID virus*

## INTRODUCTION

Prions are self-templating protein aggregates that stably perpetuate distinct biological states. On the occasion of the Nobel Prize in Physiology or Medicine 1997, the committee gave a good definition of the prion research breakthrough spearheaded by Stanley B. Prusiner:

> Creutzfeldt-Jakob disease and related illnesses affecting people and animals involve the degeneration of brain cells. In 1982 Stanley Prusiner was able to isolate a suspected infectious agent, a protein that he called a prion. He identified the gene behind the prion protein, but determined that it is also present in healthy people and animals. Stanley Prusiner showed that the prion molecules are folded in a different way than the normal proteins and that the folding of the prion can be transferred to normal proteins. This is the basis for the illness.

Prions are proteins that can switch from non-aggregated states to self-templating highly ordered aggregates. This property allows them to confer stable changes in biological states, and in doing so, to cause fatal disease in animals and humans.

### A PRION REGION IN COVID-19 SARS-COV-2 AND IN THE "VACCINES"

It has been suggested (Seneff & Nigh, 2021; Classen, 2021b; Tetz & Tetz, 2022), that there is a prion region in all spike proteins produced by the SARS-CoV-2 virus. The presence of the prion region in the SARS-CoV-2 spike embedded in the COVID-19 injectables was formally demonstrated by Tetz and Tetz (2022) as summed up in Figure 1. And, in Perez, Lounnas, and Montagnier (2021), we showed that all SARS-CoV-2 Wuhan strain variants, and all of the COVID-19 vaccines have this prion region, although it disappears totally in the Omicron variant (for the details of that disappearance, see the Appendix to this paper).

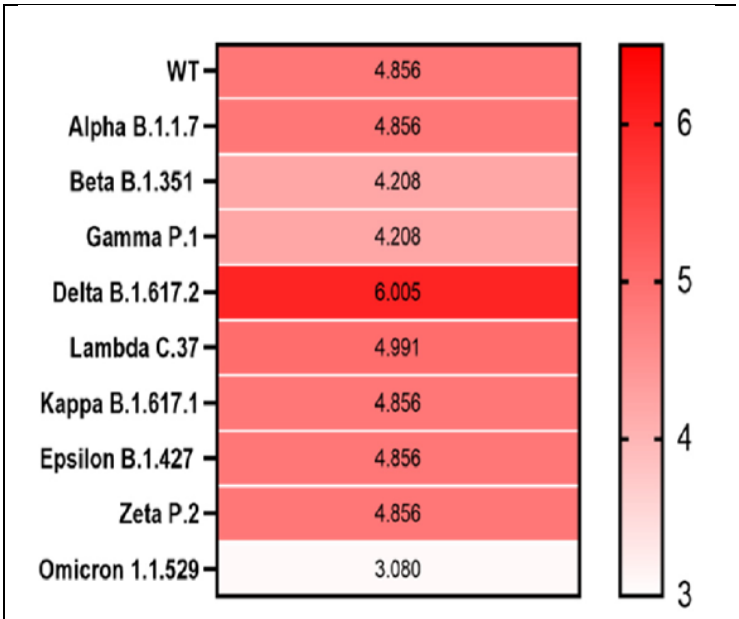


Figure 1. This figure is actually Figure 3 from Tetz and Tetz (copyright 2022, their Figure 3). Heatmap showing PrD within the S protein in SARS-CoV-2 variants. The correlation between the LLR scores of the identified PrDs in the S protein across different SARS-CoV-2 variants is presented. Mean LLC scores of S protein are denoted using a color scale, ranging from white (minimum) to saturated red (maximum). Higher LLC scores indicate a greater likelihood that the analyzed protein is a prion.

Stephanie Seneff, PhD, who works in the Computer Science and Artificial Intelligence Laboratory at the Massachusetts Institute of Technology (MIT), along with her colleague Greg Nigh from Naturopathic Oncology in Portland, Oregon, identified a "GxxxG signature motif" within the coding sequence for the mRNA portion of the injections that they say increases the risk that misfolding will occur, creating toxic oligomers, that are the basis of prion disease. They call this the "glycine zipper motif" (2021). It is characterized by a pattern of two glycine residues spaced by three intervening amino acids, represented as GxxxG. Particularly, the bovine prion linked to Mad Cow Disease also has a spectacular sequence of ten GxxxGs in a row. Similarly, the

SARS-CoV-2 spike transmembrane protein contains five GxxxG motifs in its sequence. As a result, from the genetic viewpoint, it is plausible and probable that such a sequence can behave as a prion.

### The Spike Proteins Demonstrate Prion Behavior

Even before the analysis more fully reported here was undertaken, it had been demonstrated clinically that the prion region of the SARS-CoV-2 injectables can produce prion behavior in their spike proteins (Young et al., 2020; Idrees & Kumar, 2021; Kuvandık et al., 2021). Here we expand on that record with all of the 26 cases that we began to analyze a few weeks before an additional 50 cases of rapidly developing Creutzfeldt-Jacob Disease appeared in France very soon after the injection of a first or second dose of the Pfizer or Moderna injectables. Bearing in mind from the outset that it usually takes decades for prion disease to manifest itself, the question we address here is: *why and how can this same fatal disease quickly manifest itself following these injections?* We suggest that it is necessary to suppose, and to carefully test the hypothesis, that we are probably dealing here with a new form of Creutzfeldt-Jacob Disease as more or less predicted by Herrmann et al. (2015), Seneff and Nigh (2021), Classen (2021a), by Seneff, Nigh, et al. (2022), and by Kyriakopoulos, et al. (2022).

To our knowledge, the first report of a link between COVID-19 "vaccination" and the new and rapidly developing Creutzfeldt-Jakob Disease came from Kuvandik, et al. (2021). Their report concerned an 82 year-old Turkish patient who received an injection of the Chinese Sinovac vaccine (CoronaVac, Sinovac Life Sciences, Beijing, China). Even the more traditional, better known, slow developing form of the disease is quite rare. Only 28 cases of Creuzfeldt-Jacob Disease were diagnosed in France between 1992 and 2019. One of those cases was a research technician who died in 2019 and who was believed to have contracted the deadly disease in 2010 in a laboratory where prions were under study (Santé Publique France, 2021). Later, after that fatality, in the summer of 2021, a second technician at a French public research laboratory where prions were under study also died. Because of those two deaths, and a lawsuit that followed the first technician's death, all research in France on prions was frozen (Société, Toulouse, Haute-Garonne, 2022).

### A New Creutzfeldt-Jakob Disease After an Injection of a COVID-19 "Vaccine"?

Our focus, in the main text of this paper, is on explaining the 26 cases detailed in Table 1 who displayed what seemed to be an almost spontaneous emergence of a new, rapidly developing form of Creutzfeldt-Jakob Disease. For these 26 cases, the symptoms appeared within a couple of weeks to a month after the affected person received a COVID-19 "vaccine". We provide the details on the progression of disease for those 26 cases in Table 1.[1] Our cases were mainly from France (23 of them), but additional cases soon appeared elsewhere in Europe and in the USA. All of them seemed to arise after an injection of a Pfizer, Moderna, or AstraZeneca "vaccine". In what follows here, in the main text, we analyze the fully documented evolution and timing of the symptoms of these 26 cases and update our results from our prior preprint version of this paper.[2] We also summarize in Figures 1-3 some of the surprising differences

[1] Although 26 seems small number when compared against the 12.7 billion doses of COVID-19 vaccine administered to 5.4 billion recipients , it nevertheless gets us into the reliable statistical ballpark for generalization to whole populations on the basis of the long-standing central limit theorem (Le Cam, 1986; Tate, 1965).

[2] Some of the results discussed here were previously presented on our behalf by Claire Moret-Chalmin (2022) at the 16th World Congress on Controversies in Neurology (CONy) in London, March 24-27, 2022.

between this new form of rapidly developing Creutzfeldt-Jakob Disease and the much slower prion disease previously known by similar symptoms but developing not in weeks or progressing to a fatality in about one year, but developing over about a decade and lasting one or more additional decades.

## The Princeps Case Doyer

A central case, with typical progression of the new rapidly developing disease — one distinguished from the other 26 cases only because of her husband's courageous advocacy on her behalf and for others impacted by the same new disease — is the person we refer to as "Princeps Case D".
She was a French woman, at the age of 72, who showed the first clinical signs just 14 days after her second shot of SARS-CoV-2 "vaccine". She experienced paresthesia in the dorsal part of her left foot, vertigo, reported the "foggy brain" symptom, fatigue, depression, and she showed left hyperalgesic sciatica. A vestibular MRI revealed what looked like ancient, very old, white matter infarct lesions such as those observed in those with advanced Alzheimer's disease. After she was hospitalized in CHR de Beauvais for 5 days, her blood stopped flowing normally, and it was difficult to impossible to acquire a typical syringe puncture to draw a sample of her blood into a test-tube. When she returned home, new clinical signs appeared: gait disturbances, hyperesthesia of her right leg. She also reported nocturnal burning pain with urination. She had violent myoclonus spasms and likely epileptic seizures. Rapid neurological decline was observed.

The Neurological Department of the American Hospital in Paris concluded that Mauricette Doyer had Creutzfeldt-Jacob Disease: this diagnosis was confirmed with a lumbar puncture, by critical biomarkers including protein 14-3-3, also by EEG, diffusion-weighted magnetic resonance imaging (dfMRI) and Fluid-Attenuated Inversion Recovery (FLAIR) by a Positron Emission Tomography (PET) scan, all of which were positive and together have very high sensitivity and specificity enabling a reliable and valid diagnosis of Creutzfeldt-Jacob Disease. At week 10 the patient was akinetic, mute, bedridden, and had hypersomnia with a typical akinetic mutism state of Creutzfeldt-Jacob Disease. From that time forward, she was hospitalized at home with anxiety attacks, agitation, myoclonus, requiring parenteral (intravenous) nutrition, suffering with intermittent respiratory distress, and being treated with Midazolam for palliative care. Our observations indicate to us that the prolonged survival period for individuals with this rapidly developing prion disease is likely due to applying ameliorative management procedures as explained by Iwasaki et al. (2015). In the widely publicized case of M. Doyer — who was our very important patient, Princeps Doyer — that sort of procedure was implemented by her family and was continued after she was no longer able to move or speak. To explain why she was so important to our work, and the discovery and analysis of the other 26 cases discussed in detail below in this paper, it is useful for us to refer to a published statement by her husband. On October 31, 2021, Marc Doyer wrote:

> I decided to support and join the action of the association VERITY France whose families have already gathered. We are committed to bringing the truth to light, we have in common the desire to bring together a large gathering in order to create a civic force aimed at illuminating the darkness in which our families are plunged. For my part, it's my wife Mauricette [our Princeps Doyer], whom I lose a little more every day, half of myself, through a horrible illness (Creutzfeldt-Jakob). I am also continuing my quest for testimonials with the help of the COVID-19 France Association, which will provide everyone with an independent national platform for reporting cases of side effects. Like all the families concerned, our life will never be the same again, however my determination for truth will accompany me forcefully until my last breath [see his original statement in French at https://www.verity-france.org/marc-doyer-rejoint-verity-france/].

The progression of the disease in Princeps Doyer is summed up as Case 4 in Table 1, and also in Figure 2. She received her offending second dose of the Pfizer product on May 5, 2021 and developed symptoms by

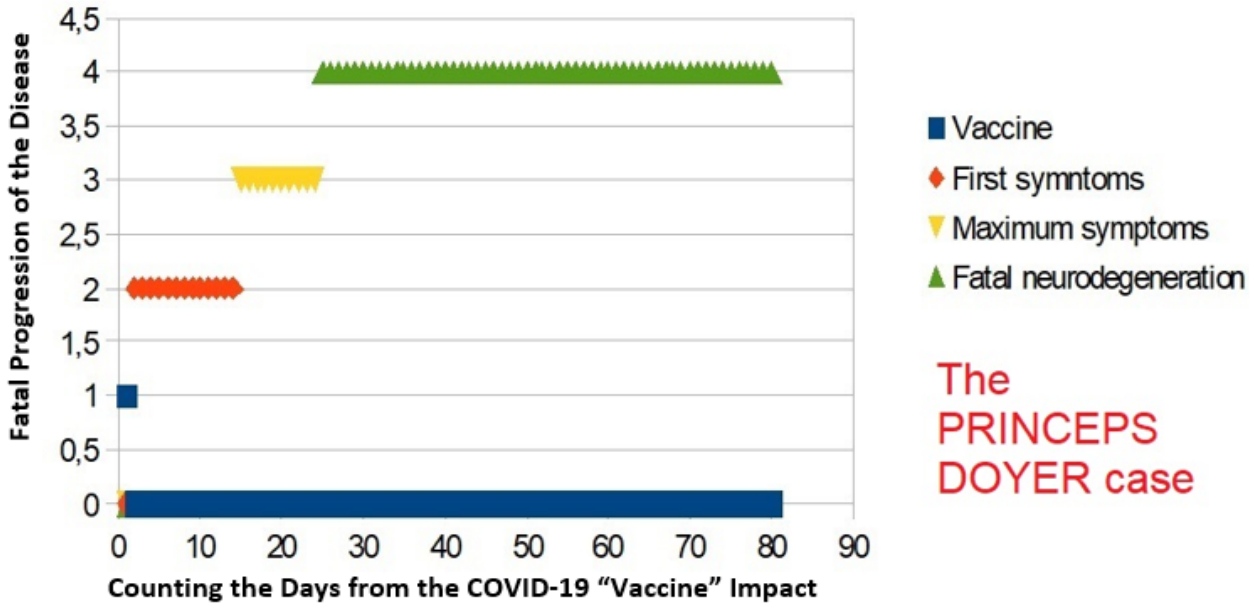


Figure 2. Of the 26 cases of the new, rapidly developing Creutzfeldt-Jacob Disease discussed here, the leading one identified and singled out by her husband, Marc Doyer, and designated on that account as "the Princeps Doyer Case", was Mauricette Doyer. She was Marc Doyer's wife. He subsequently joined Verity France, the COVID-19 France Association. At the age of 72 the symptoms of the new form of Creutzfeldt-Jacob Disease that we are discussing in this paper first appeared only 14 days after a Pfizer injection for COVID-19.

May 19, just 14 days later. By July 5th she was experiencing severe symptoms just 61 days into the disease which was firmly diagnosed on that date. Her fatal neurodegeneration progressed to her death on May 3, 2022 within almost exactly one year from the date of the suspected causal injection of the Pfizer vaccine.

Figure 3 shows the results of brain scans for Princeps Doyer. Procedures included MRI, PET, and EEG (D M), Brain MRI (Diffusion Weighted Imaging), Fluid-Attenuated Inversion Recovery (FLAIR) and (T2). The results showed abnormalities in the parietal lobes predominantly on the left side, and of the cingulate gyrus. The FDG-PET showed hypometabolism of the right hemisphere predominantly in the right frontal and parietal lobes. The EEG (lower right of Figure 3) showed a tell-tale 6 Hz background activity and 6 seconds of 1 Hz triphasic periodic spikes in the right hemisphere. Key patterns in the blue rectangle of the EEG represent typical evidence to justify the diagnosis of Creutzfeldt-Jakob Disease.

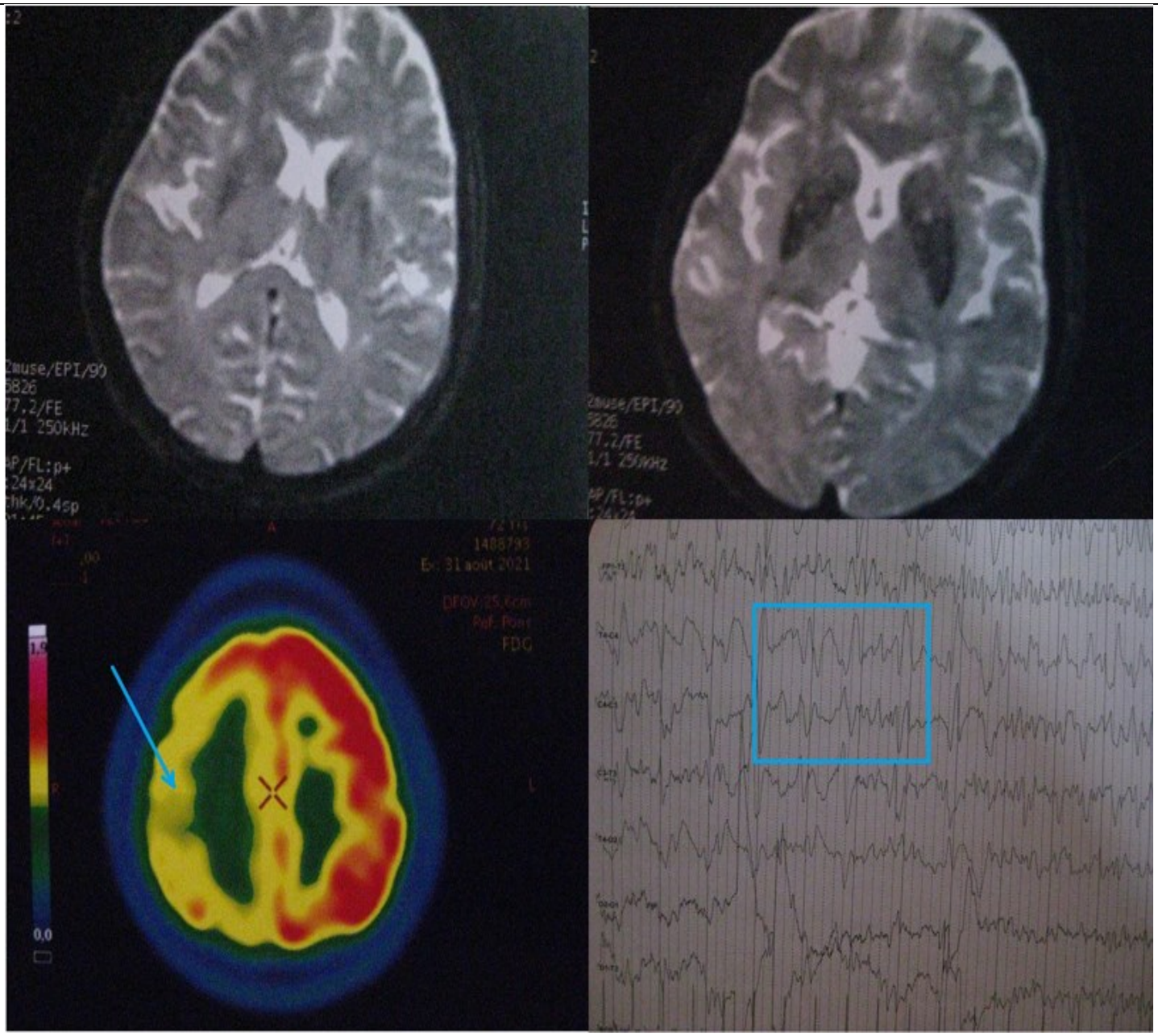


Figure 3. Brain scans of the Princeps Doyer: MRI, PET and EEG (D M), Brain MRI (Diffusion Weighted Imaging) and (Fluid-Attenuated Inversion Recovery, FLAIR) and (T2) showing abnormalities of the parietal lobes predominantly on the left side and of the cingulate gyrus. The FDG-PET shows hypometabolism of the right hemisphere predominantly in the right frontal and parietal lobes. The EEG (lower right) shows 6 Hz background activity and 6 seconds of 1 Hz triphasic periodic spikes in the right hemisphere. The blue rectangle in the EEG is a typical proof of Creutzfeldt-Jakob Disease with, in her case, 6 seconds of 1 Hertz triphasic periodic spikes.

**Table 1**
**Progression of the Rapidly Developing New Form of Creutzfeldt-Jacob Disease in the 26 Cases Under Study**

| Case Number | Days to 1st | Days til Death | Country | Age | Sex | Dose & Source | Date Received | First Symptoms | CJD Diagnosis | Severe Symptoms | Deceased |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 10 | 76 | France, Montpellier CHU | 72 | M | Pfizer 2nd | 20-Apr-21 | 30-Apr-21 | 20-May-21 | 20-May-21 | 6-Jul-21 |
| 2 | 7 | 78 | France, Bordeaux Pellegrin CHU | 52 | M | Pfizer 2nd | 28-May-21 | 5-Jun-21 | 28-Jul-21 | 28-Jul-21 | 16-Sep-21 |
| 3 | 1 | 78 | France. Rothschild Foundation | 48 | F | Pfizer 2nd | 25-Aug-21 | 26-Aug-21 | 08-Oct-21 | 9-Oct-21 | 13-Nov-21 |
| 4 | 14 | 365 | France, American Hospital (Princeps Doyer) | 72 | F | Pfizer 2nd | 5-May-21 | 19-May-21 | 5-Jul-21 | 5-Jul-21 | ‡ 3-May-22 |
| 5 | 10 | 56 | France, Tours CHU | 73 | M | Pfizer 2nd | 30-Apr-21 | 10-May-21 | 07-Jun-21 | 7-Jun-21 | 23-Jun-21 |
| 6 | 8 | 68 | France, Nantes CHU | 75 | M | Pfizer 2nd | 18-Mar-21 | 26-Mar-21 | 18-Apr-21 | 8-Apr-21 | 26-May-21 |
| 7 | 15 | 113 | France, Lille CHU (KJ16) | 60 | M | Pfizer 3rd | 31-Aug-21 | 15-Sep-21 | 25-Nov-21 | 15-Oct-21 | 23-Dec-21 |
| 8 | 15 | 78 | Israel, Jerusalem | 62 | M | Pfizer 2nd | 22-May-21 | 7-Jun-21 | 19-Jun-21 | 19-Jun-21 | 10-Aug-21 |
| 9 | 1 | 187 | France, Chambery Hospital (KJ17) | 50 | F | Pfizer 1st | 10-Jun-21 | 6/11/2021 | 06-Dec-21 | 1-Sep-21 | 17-Dec-21 |
| 10 | 1 | 66 | Belgium, Charleroi CHU | 69 | M | Pfizer 1st | 8-Apr-21 | 9-Apr-21 | 12-May-21 | 12-May-21 | 14-Jun-21 |
| 11 | 15 | 202 | Switzerland, Lugano | 67 | F | Moderna 2nd | 22-May-21 | 7-Jun-21 | 01-Dec-21 | 18-Jun-21 | 14-Dec-21 |
| 12 | 15 | 72 | France, Amiens CHU | 70 | F | Pfizer 3rd | 18-Nov-21 | 3-Dec-21 | 11-Jan-22 | 2-Jan-22 | 1-Feb-22 |
| 13 | 30 | 115 | France, Cherbourg CHU | 77 | F | AstraZeneca 2nd | 30-Jul-21 | 31-Aug-21 | 01-Oct-21 | 1-Oct-21 | 25-Nov-21 |

| | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 14 | 5 | 232 | France, Ivory Center Francilien | 62 | M | Pfizer 1st | 6-Jul-21 | 11-Jul-21 | 10-Dec-21 | still living | ‡ 28-Feb-22 |
| 15 | 15 | 395 | France, Salpêtrière Hospital | 72 | F | Pfizer 1st | 7-Jun-21 | 22-Jun-21 | 20-Aug-21 | 11-Nov-21 | ‡ 12-Feb-22 (palliative care; alive Aug-22) |
| 16 | 15 | 210 | France, Cahors (KJ10) | 72 | M | Pfizer 2nd | 31-May-21 | 15-Jun-21 | 08-Oct-21 | 8-Oct-21 | 30-Dec-21 |
| 17 | 15 | 335 | France, Toulouse CHU (Patient 1 4 22) | 38 | F | Pfizer 2nd | 20-Jul-21; 11-Dec-21 Delta | 10-Jan-22 (after end COVID) | 08-Mar-22 | 25-Mar | ‡ before 22-Jun-22 |
| 18 | 15 | 370 | France, Strasbourg CHU (Patient 2 4 22) | 68 | F | Pfizer 2nd | 15-May-21 | 30-Jun-21 | 01-Dec-21 | 1-Aug-21 | ‡ before 22-Jun-22 |
| 19 | 17 | 235 | France, Clermont Ferrand (Patient 4 4 22) | 75 | M | Pfizer 2nd | 17-Apr-21 | 4-May-21 | 5-Dec-21 | 15-Sep-21 | 15-Dec-21 |
| 20 | 7 | 365 | France, Caen CHU (Patient 12 4 22) | 64 | F | Pfizer 2nd; Moderna 3rd | 21-Jun-21; 27-Dec-21 | 28-Jun-21 | 21-Aug-21 | 21-Aug-21 | ‡ before 22-Jun-22 |
| 21 | 17 | 210 | France, Chateauroux CHU (Patient 15 4 22) | 64 | F | AstraZeneca 2nd | 28-May-21 | 15-Jun-21 | 7-Dec-21 | 20-Nov-21 | 28-Dec-21 |
| 22 | 30 | 186 | Bordeaux, R. P. | 75 | M | AstraZeneka | 11-Jun-21 | 11-Jul-21 | 16-Dec-21 | 11-Nov-21 | 17-Dec-21 |
| 23 | 2 | 276 | Chateauroux Saint Antoine | F | 78 | Pfizer 2nd | 1-Mar-21 | 3-Mar-21 | 15-Nov-21 | 1-Jul-21 | 8-Dec-21 |
| 24 | 11 | 87 | USA (Cheryl C., reported by Redshaw, 2022) | F | 64 | Pfizer 2nd | 25-Apr-21 | 6-May-21 | 12-Jul-21 | 19-Jun-21 | 22-Jul-21 |
| 25 | 1 | 142 | USA (Carol B., Redshaw, 2022) | F | 70 | Moderna 2nd | 17-Mar-21 | 18-Mar-21 | 15-Jul-21 | 15-Jul-21 | 2-Aug-21 |
| 26 | 4 | 150 | USA (Jennifer D. S., Redshaw, 2022) | F | 60 | Pfizer 2nd | 21-Sep-21 | 25-Sep-21 | 23-Jan-22 | 24-Dec-21 | 21-Feb-22 |

‡ The **bolded** characters represent the 6 patients who were still alive when we first drafted this table in February 2022. We updated their status in August 2022. By that time 5 of the survivors had died. Only one remained alive in August 2022. In our shared professional judgment, we believe this last patient benefited from the antioxidant protocol prescribed by our late colleague, Luc Montagnier.

## MOVING ON TO THE DIAGNOSIS OF ALL 26 CASES IN TABLE 1

In a landmark study by Lemstra et al. (2000), illustrating a robust method for diagnosing Creutzfeldt–Jakob Disease, the authors examined 112 patients for protein 14-3-3. The sensitivity and specificity for Creutzfeldt-Jacob Disease — when used in the highly typical semiological setting and exploration of their cases — were 97% for sensitivity and 87% for specificity. The combination of increased T-tau levels and increased T-tau to P-tau ratios in patients with Creutzfeldt-Jacob Disease also has high specificity in routine clinical applications. That is to say, clinicians and researchers can be quite certain that the prion disease has been correctly diagnosed in the patients studied. The recently developed RT-QuIC test allows for highly sensitive and specific detection of Creutzfeldt-Jacob Disease in human cerebrospinal fluid and is also a key diagnostic tool in spite of the fact that it may overlook 11% to 23% of Creutzfeldt-Jacob Disease cases (Orrú et al., 2015; Green, 2019; Rhoads et al., 2020). Collectively applied, with due diligence, combined with the other diagnostic procedures we used for our 26 cases, such proven methods constitute a kind of gold standard for diagnosing and authenticating cases of Creutzfeldt-Jacob Disease. Those cases are described with important details about the onset and progression of the disease in Table 1 with follow up analyses in Figures 4 and 5 characterizing respectively the onset and progression to death.

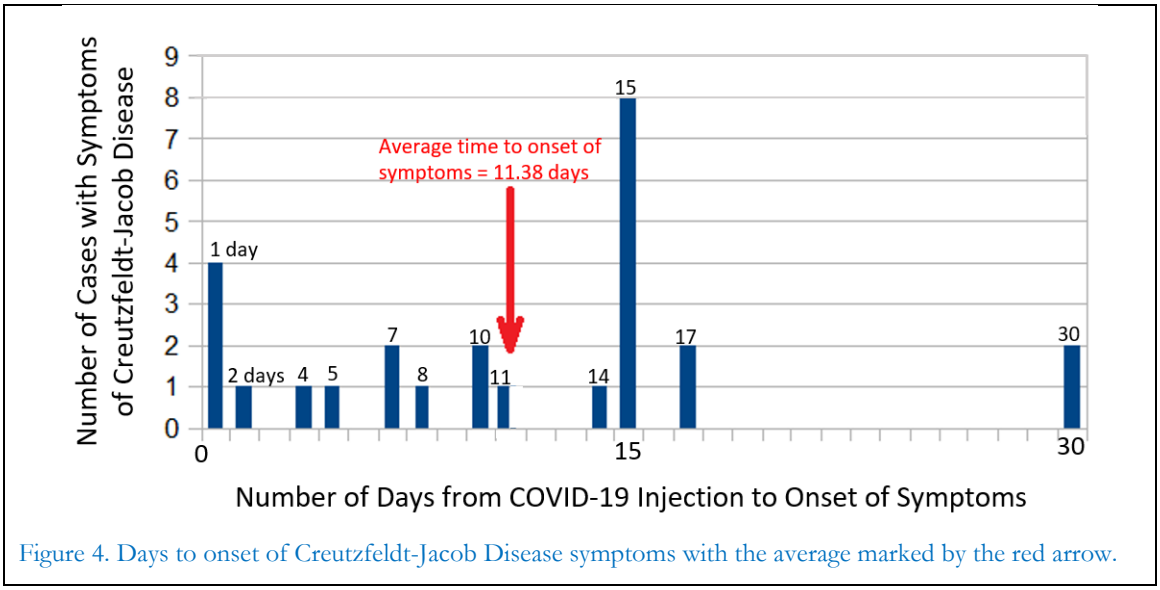


Figure 4. Days to onset of Creutzfeldt-Jacob Disease symptoms with the average marked by the red arrow.

In Figure 4, we summarize the dramatic rapidity of the onset of symptoms in this new variety of deadly prion disease, and in Figure 5, we clinch the case for the rapid progression of this new form of prion disease to its fatal conclusion — a variety that was apparently caused in these 26 cases by one or more COVID-19 "vaccine" injections.

Following the release of our data, in an earlier preprinted draft of this article, some additional cases were reported to us from the USA. In fact, according to the Vaccine Adverse Event Reporting System (VAERS), between December 14, 2020 and April 1, 2022, 20 cases of Creutzfeldt-Jacob Disease attributed to COVID vaccines were reported and 19 deaths were recorded. The majority of those VAERS cases occurred in the 65 to 75 age range and involved, as in the 26 cases we have focused on in this study, an

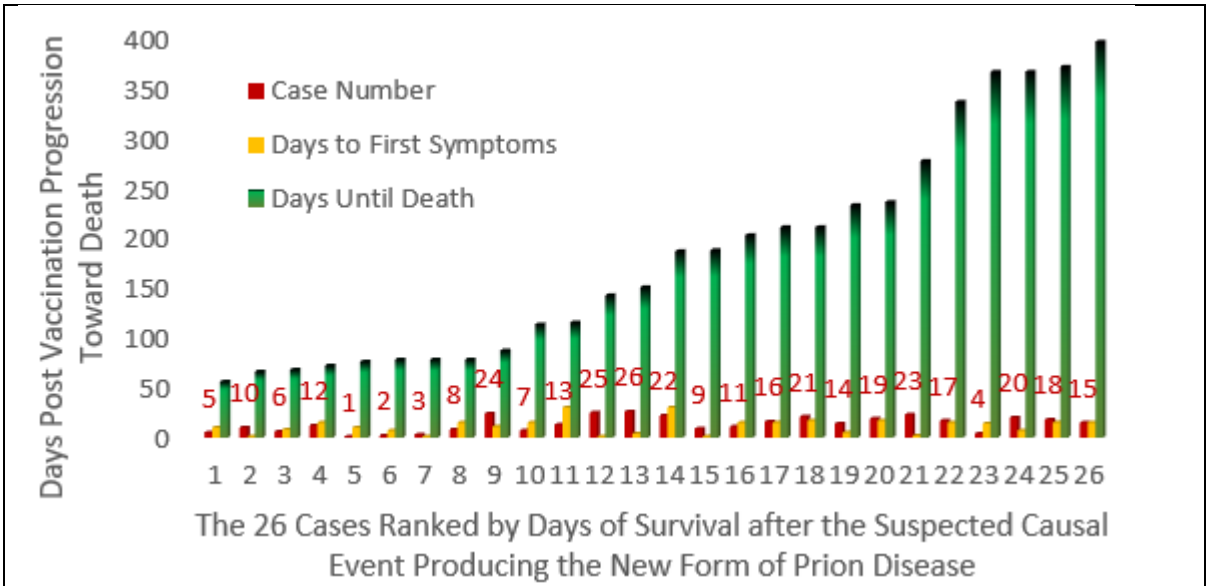


Figure 5. A summary of progression toward death for the 26 cases of the new form of Creutzfeldt-Jacob Disease believed to be caused by COVID-19 "vaccine" (updated to or last check on the survivors in August 2022).

atypical, sudden onset of symptoms within a couple of weeks or at most a month after a COVID-19 injection, as was observed in all of the cases in our data sample. Upon examination of the progression of disease in the 26 cases we have closely studied and reported on here, we noted that cases following the mRNA injections manufactured by Pfizer and Moderna, tended to have symptom onset within about two weeks whereas recipients of the AstraZeneca viral-vectored DNA delivery system required as long as 30 days to begin showing symptoms of the disease.

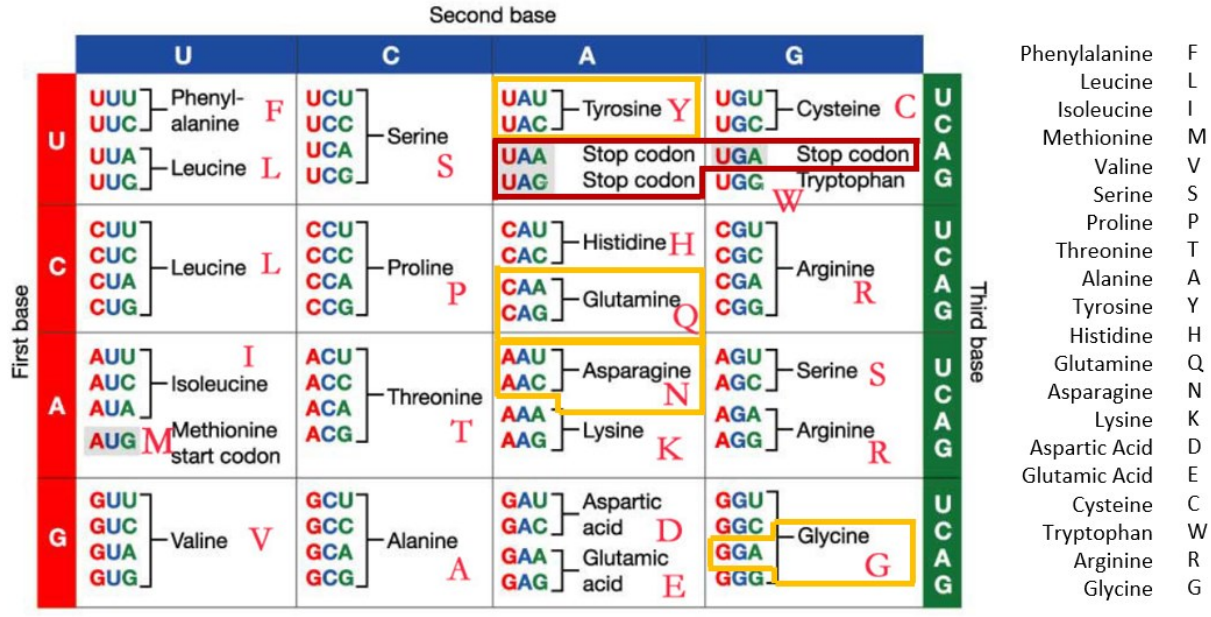


Figure 6. The Universal Genetic Code T C A G in its two-dimensional table and the relative locations of the NQYG prion facilitator amino acids relating to stop codon locations. Note that the 4 amino acid prion function facilitators N Q Y G are close to the 3 stop codons (accounting for 20% of the amino acids).

To summarize, of the 26 cases analyzed, the first symptoms of Creutzfeldt-Jacob Disease appeared on average 11.38 days after the injection of the COVID-19 "vaccine". Of these 26 cases, 20 had died at the time of the first draft of this article when 6 of the infected individuals were still alive. The 20 deaths occurred only 4.76 months after the injection. Among them, 8 died within 2.5 months. All this confirms the radically different nature of this new form of Creutzfeldt-Jacob Disease, whereas the symptoms of the classic form require several decades. Figure 5 is updated to August 2022 when only one patient was still surviving.

#### A POSSIBLE PATH TOWARDS UNDERSTANDING THE PRION EFFECTS

To establish the empirical proof, as indirect as it may be, it is useful to begin this part of our argument by considering some facts from the well-known table of the Universal Genetic Code as highlighted in Figure 6. The idea about how to proceed began with two observations about the Universal Genetic Code. For one, in building a protein from mRNA codons, there is a trap to avoid: not to accidentally land on one of the 3 stop codons, and for the other, if we are interested in the codons for the amino acids N, Q, Y, and G — the 20% of amino acids involved in the prion function — it seems likely that these amino acids could, by their biophysical nature, account for weak links in the otherwise stable DNA helix. From this came the idea that the two-dimensional table of the genetic code could provide the topology within which the 3 stop codons represent a kind of "hole" in the vicinity of which the slightest mutation of a nucleotide could pose a problem. Then, it occurred to us to locate the 4 amino acids N Q Y G vis-à-vis the "well" (the sink hole) formed by the 3 stop codons as shown in Table 2.

**Table 2**

**Amino Acid Mutations Occurring Near Stop Codons in the Universal Genetic Code Table**

| **N Q Y G the four amino acids increasing Prion function** | | | | |
|---|---|---|---|---|
| **Stop** | **N** | **Q** | **Y** | **G** |
| UAA | **A**A**U** | **C**AA | UA**U** | |
| UAG | **A**A**C** | **C**AG | UA**C** | |
| Stop | | | | |
| UGA | | | | **G**GA |
| **Number of mutations by codon** | 2 | 1 | 1 | 1 |

Our intuition was of a geometric nature: in the 2D topology of the genetic code table (Figure 6), there are 3 stop codons that sign the end code of a protein. The 4 most pro-prion amino acids being Y Q N G, we wondered if they might created a kind of "degeneracy" close or very close to the "proteomic black hole" that constitute the 3 stop codons? For this we will focus on the restrictive case where the mutation of a single nucleotide on Y Q N G would switch to a Stop codon. Our answer is "yes", it could be happening. Table 5 shows that amino acids N Q Y G are "topologically" close to the stop codons in the two-dimensional table; in 5 of 7 cases of stop <==> N Q Y G mutations, a single mutated base would suffice. This provides the case for the 3 prion amino acids Q Y and G.[3] Therefore, we concluded that this thesis necessitated further exploration in the interest of better understanding how prions work. That more detailed exploration, however, we have placed in the Appendix to this paper.

[3] Upon reading this paper, along with 11 other reviewers, Stephanie Seneff, PhD., commented that "Y, Q and G are all present in the YQAGS sequence that links to the C-terminal domain of the prion protein through molecular mimicry".

## Discussion

A key point of our article is the conclusive demonstration, if not the discovery, of a prion region in the spike protein of the Wuhan strain, but not in Omicron. Moreover, because the COVID-19 injectables include the mRNA coding sequence of the Wuhan spike protein, our work extends to the offending prion portion in all of those injectables. Our earlier preprint was referred to and quoted by Seneff, Kyriakopoulos, et al., (2022) as follows:

> Montagnier and his team identified a segment of the spike protein that they thought had characteristic prion-like features. Within that segment is a piece that has five amino acids, YQAGS [then quoting our work, she wrote:] "The human prion protein has the same piece … Except for the middle one (YQRGS), the other four amino acids are all identical with this piece near the C-terminal end of the human prion protein. So, it's really perfect. It's a place where, if you get antibodies to that, it's basically a death sentence". [Also see her comment in footnote 3.]

Our research has been limited by major obstacles beyond our control. For one, the officially acknowledged "side effects" attributed to the COVID-19 injections are known to be fewer in number and less severe that the ones occurring in reality. The only official published study, one that was sponsored but later completely ignored by the federal authorities in the USA (Lazarus et al., 2010), found that "fewer than 1% of vaccine adverse events are reported" to the official regulators. Therefore, it is impossible to say how many cases of the new form of prion disease are actually occurring especially in the countries with very high vaccination rates such as Israel, Australia, the USA, the United Kingdom, and in Europe.

Moreover, because the official basis today for diagnosing Creutzfeldt-Jacob Disease, or any prion disease condition, is still an autopsy, we note that the number of autopsies for suspected cases of Creutzfeldt-Jacob Disease remains marginal at best and completely missing for the vast majority of cases. In Europe, the best services actually performing any autopsies or other diagnostic laboratory procedures have all been prevented since July 27, 2021 (Société, Toulouse, Haute-Garonne, December 9, 2022; LADEPECHE.fr, March 17, 2022) from analyzing any tissue samples with even suspected prion disease.

All the qualified French agencies — these include INRAE, ANSES, CNRS, INSERM, and CEA — "froze" all research dealing with prion disease from July 27, 2021 because of the death of a 33-year-old technician working at the National Veterinary School of Toulouse. She was reported to have contracted what had to be the rapidly developing form of the disease that we are reporting on here (Moore, August 2021; Société, Toulouse, Haute-Garonne, December 9, 2022). However, she was supposedly infected from her laboratory work and not from a COVID-19 injectable. None of the mainstream publications pointed out whether or not the deceased young woman had received a COVID-19 injection prior to her prion symptoms and death.

With the foregoing limitations in mind, it is highly probable that the real number of cases of the new form of Creutzfeldt-Jacob Disease is greater than any European agency (or any other authority) has reported.

## Conclusion

In summary, of the 26 cases analyzed in the present study, the first symptoms of Creutzfeldt-Jacob Disease appeared on the average within 11.38 days after the injection of a COVID-19 "vaccine". Twenty deaths occurred only 4.76 months after such an injection and 8 of them died suddenly within 2.5 months. For the 6 patients who did not die before the appearance of our preprint article (February 2022), in a review of their status in August 2022 (see updated Figure 5), it was discovered that 5 of them had died and only one

was still living. We must note that, like several other patients, this last patient was treated according to an antioxidant protocol prescribed by Luc Montagnier, MD, who himself died February 8, 2022.

The clinical facts reported in this article confirm an unnatural, injection related, new form of Creutzfeldt-Jacob Disease. The stereotypic post-vaccine symptoms appear almost immediately, or very soon after a jab, usually the second one, suggesting that the first injection is a potentiating event, all of which are followed by lightening-fast development of the disease progressing to its final conclusion in death. The advance warnings from Luc Montagnier widely referred to in the media — for just two examples leading to many references on the internet, in his speech before the Luxembourg Parliament on January 12, 2021 (World Freedom Alliance, 2021) and in an interview on May 18, 2021 (published by the RAIR Foundation USA, 2021) — were based on his far-sighted genetic studies of pathogenic prion regions in the proteins of different species. Those intensive genetic studies led to the hypothesis of the now demonstrated Creutzfeldt-Jacob Disease "side effects" of the COVID-19 "vaccines". He predicted the causation of this new neurodegenerative form of Creutzfeldt-Jacob Disease on the basis of his study of the insertion of the pathogenic prion in the SARS-CoV-2 spike protein. Together with his team, he monitored all the research leading from the genetic laboratory work to the clinical effects of this new form of Creutzfeldt-Jacob Disease.

## Acknowledgments

Primarily we thank Marc Doyer, founding President of the Creutzfeldt-Jacob Disease Association of France. In addition to diagnosing and referring us to the Creutzfeldt-Jacob Disease of his own dear wife, Mauricette, the first of the princeps Doyer, within a few months had established his Association known as VERITY Creutzfeldt-Jacob Disease, and had gone on with admirable energy and tenacity in the face of his personal adversity to collect at first more than 40 cases of Creutzfeldt-Jacob Disease including 16 of the cases during the life of Luc Montagnier and 2 months later 50 more, of whom 23 confirmed cases are reported on here. We also thank Professor Richard M. Fleming, PhD, MD, JD, also a physicist-nuclear-cardiologist-attorney, https://www.amazon.fr/COVID-19-Bioweapon-Scientific-Forensic-investigation/dp/1510770194, who in 2020 had already suggested a link between the spike protein and prion diseases. Our thanks also extend to Amos D. Korczyn, who served as President of CONy for the 13th World Congress on Controversies in Neurology, who is also Professor Emeritus in Neurology, Department of Neurology Tel Aviv University, https://cony.comtecmed.com/korzcyn/ and who encouraged us to draft this article. Finally, we thank Stephanie Seneff, PhD, of the MIT Computer Science and Artificial Intelligence Laboratory, https://worldcouncilforhealth.org/multimedia/stephanie-seneff-covid-vaccines-disease/. It was she who reported the princeps Mauricette Doyer as a worldwide reference case of the possible link between COVID-19 vaccines and Creutzfeldt-Jacob Disease.

# *Appendix: Complementary Methods Applied to Prion Regions*

In what follows, we use two complementary methods of prion analysis: first, is the PLAAC software (Lancaster et al., 2014; illustrated in Figure 7) which makes it possible to detect, from an amino acid sequence, regions likely to develop a prion function; second, is the "Master Code of DNA" making it possible to test and possibly confirm the hypothesis of a prion function by highlighting certain structures or patterns in the signature curves of the Master Code unifying the genomic and proteomic interactions of the prion sequences on which we focus attention.

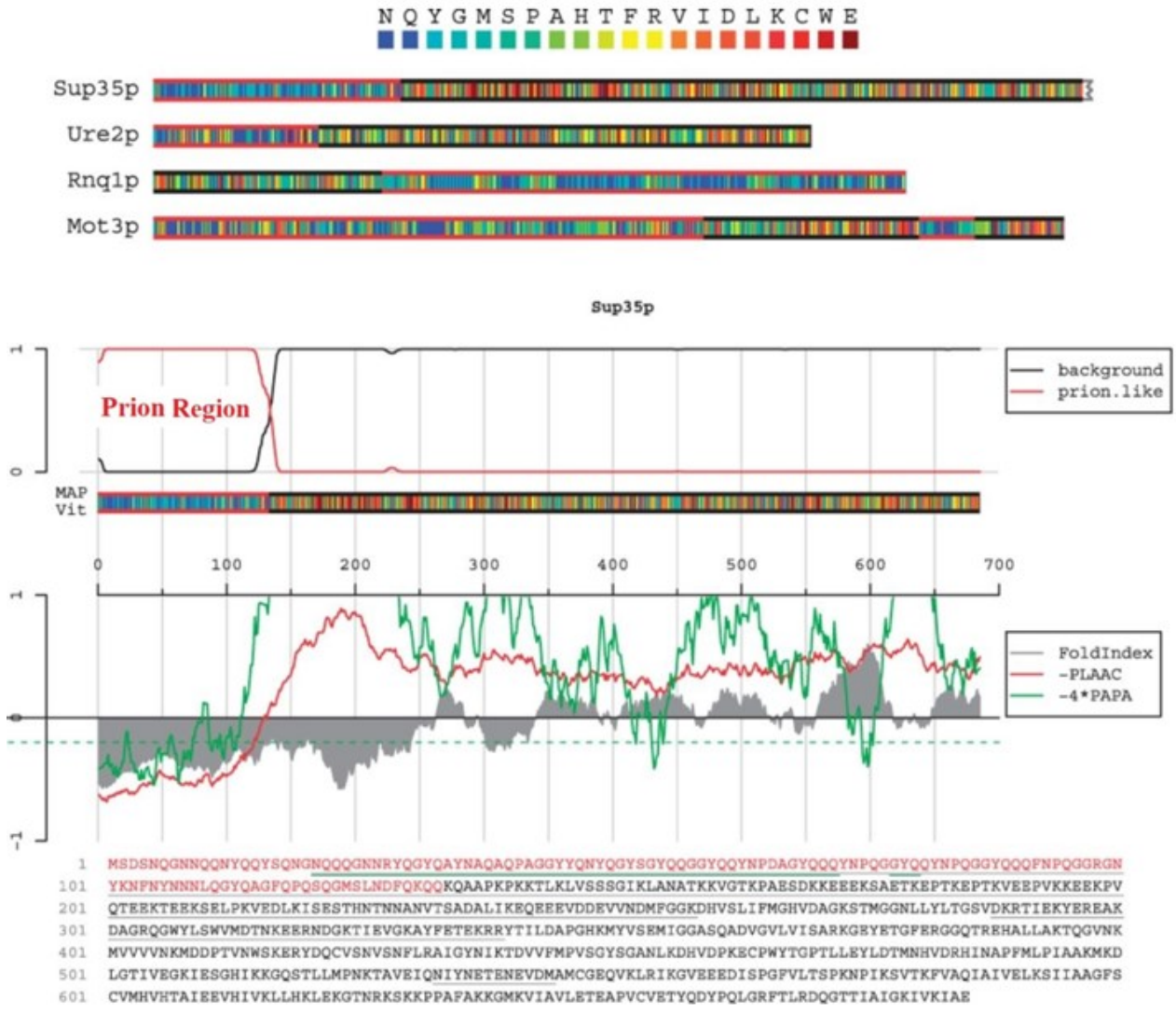


Figure 7. Illustrating the PLAAC software application with the yeast prion Sup35p. Top: four known yeast prion proteins with each amino acid color-coded by its enrichment log-likelihood ratio in PrLDs (styled after the Sequence Enrichment Visualization Tool; http://jura.wi.mit.edu/cgi-bin/bio/draw_enrichment.pl), with HMM parse indicated by outer bars. Bottom: detailed visualization of the yeast Sup35 protein, including several prion-prediction scores.

# METHODS

### PLAAC ANALYSIS

We illustrate the PLAAC method here using the Sup35 prion from a yeast (Figure 7) — in particular, Saccharomyces cerevisiae S288C translation termination factor GTPase eRF3 (SUP35), partial mRNA (NCBI reference sequence: NM_001180479.3 https://www.ncbi.nlm.nih.gov/nuccore/398365952). At the top of the figure, Sup35p is compared in the brightly colored ribbons extending horizontally across the page, with Ure2p, Rnq1p, and Mot3p, respectively. Immediately beneath those ribbons, in Figure 7, in the three wide rows across the page in the middle, we give a more detailed PLAAC analysis focusing on Sup35p. The PLAAC software detects a prion region in the first 120 amino acids of the SUP35 protein. This is confirmed by the red curve just under the Sup35p label in black letters. Details follow in the fluctuating green, red, and gray squiggly lines just above the amino acid sequences labeled, 1, 101, 201, 301, 401, and 501 at the bottom of the figure.

**Table 3**
**PLAAC Conventions Explained Briefly**

**LEGEND PLAAC results**

**==> The two curves at the top for Sup35p** are complementary resulting from Markov chain processes (Markov, 1971)

**Background BLACK —**

**Prion Disease-like RED —**

**==> The three curves below (in the middle of the Sup35p portion)**

**FOLD INDEX GRAY** — (entropy like indicator) low (negative) if possible prion function

**PLAAC RED** — low (negative) if possible prion function

**PAPA GREEN** — [Pleiotropic Architecture Pathway Analysis] second complementary method, high if state transitions

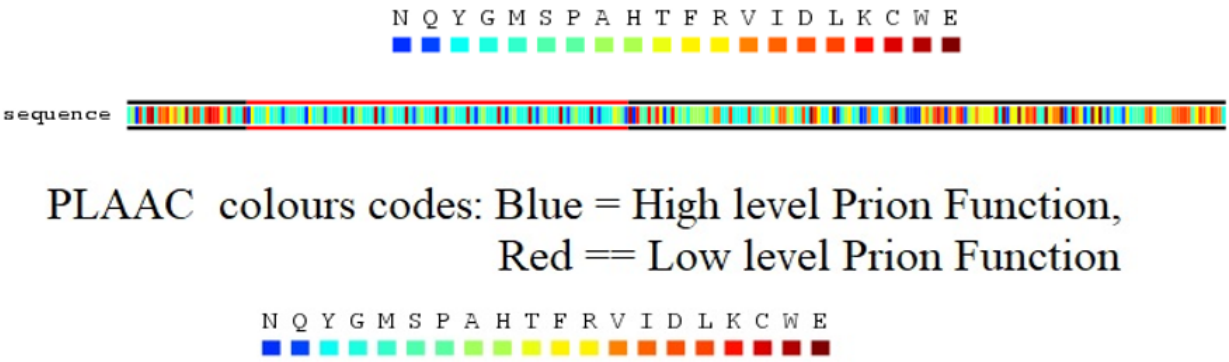


Figure 8. PLAAC colour conventions explained.

### MASTER CODE ANALYSIS

The "Master Code" method allows, from the only atomic masses common to DNA, RNA, and amino acids, for numerical values to highlight a kind of Meta-Code which unifies the 3 codes of DNA, RNA, and amino acid sequences. Particularly, the Master Code curves measure the level of coupling or correlation that unifies the genomic (DNA) and proteomic (amino acid) expressions for any sequence, whether the sequence in DNA codes for a protein or not. In Perez (2009, 2015, 2017a, 2017b), we analyzed all types of prions in beginning from the early 2000s including those for Mad Cow Disease and ranging across those prions in plants, yeast, humans, cattle, sheep, etc. In doing so, back then, we had already highlighted a kind of "signature" — a waveform of an invariant shape — which would be common to all prions: the typical signature of the Master Code took the characteristic form of a "W" (or by symmetry, if inverted, an "M"). We extended our analysis to the amyloid formations in Alzheimer's disease (Perez & Montagnier, 2021).

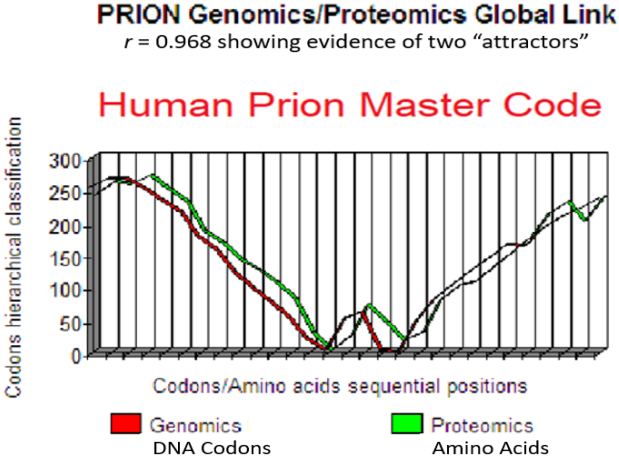


Figure 9. "W" structure appeared as a kind of INVARIANT, COMMON to all prions. Here, we illustrate the case of the human PRNP prion.

By applying the PLAAC software, on the one hand (Figure 7), in conjunction with the "Master Code", on the other, we are able to detect and then confirm the possible, even probable presence, of a prion function in Sup35p. In Figure 8, we show a probable prion function using PLAAC, and then, as illustrated in Figure 9, with the "Master Code", we are able to confirm the "W" structure, or its symmetrical shape "M", for the regions brought forward by PLAAC. Following up, we observed that these prion regions from PLAAC are always confirmed by "continuously diminishing" the "Master Code" curves. The demonstration of the invariant "W" or "M" signature wave-form is also characteristic, for instance, of the human PRNP prion as shown in the following section, especially in `Figure 10`.

## RESULTS and DISCUSSION

In this section, we first present various studies of prions: Creutzfeldt-Jacob Disease in humans, Mad Cow Disease in cattle, and the equivalent in sheep. In the second step, we prove the disappearance of the possible prion function from the last Omicron variant, one supposedly evolved naturally from SARS-CoV-2, while the prion function is not only highlighted in the Wuhan parent strain, but also in *all* of its other variants, and in *all* the "injectable" products (see Shimon Yanowitz as cited by David Hughes, 2022, in this journal) of Pfizer, Moderna, and so forth. Then, in a third step, we look for possible prion functions in 25 spike proteins all supposedly evolving, or derived by "vaccine" engineers, from the Wuhan initial strain — right on out to its last Omicron worldwide variant where the prion function strangely disappears.

## PRIONS IN HUMANS (PRNP), SHEEP (TDP-43), AND CATTLE (MAD COW DISEASE)

In this section, taking them in the following order, we deal with the known prions that have been studied closely in humans, sheep, and cattle.

### *The Human PRNP Prion*

In considering the human PRNP prion, we begin with the translation of its complete DNA coding sequence (cds) as recorded by the National Center for Biotechnology Information (NCBI) in the GenBank database at this URL: https://www.ncbi.nlm.nih.gov/nuccore/AF085477.2 into the amino acid sequence of the "Homo sapiens prion protein precursor (PRNP) gene":

MANLGCWMLVLFVATWSDLGLCKKRPKPGGWNTGGSRYPGQGSPGGNRYPPQGGGGWGQP
HGGGWGQPHGGGWGQPHGGGWGQPHGGGWGQPHGGGWGQPHGGGWGQPHGGGWG
QPHGGGWGQGGGTHSQWNKPSKPKTNMKHMAGAAAAGAVVGGLGGYMLGSAMSRPIIHFG
SDYEDRYYRENMHRYPNQVYYRPMDEYSNQNNFVHDCVNITIKQHTVTTTTKGENFTETDVK
MMERVVEQMCITQYERESQAYYQRGSSMVLFSSPPVILLISFLIFLIVG

Applying the PLAAC software at http://plaac.wi.mit.edu we get the result reported in Figure 11. Then, combining that result with the "Master Code" as shown in Figure 12, we confirm the wave-form of the signature in each instance.

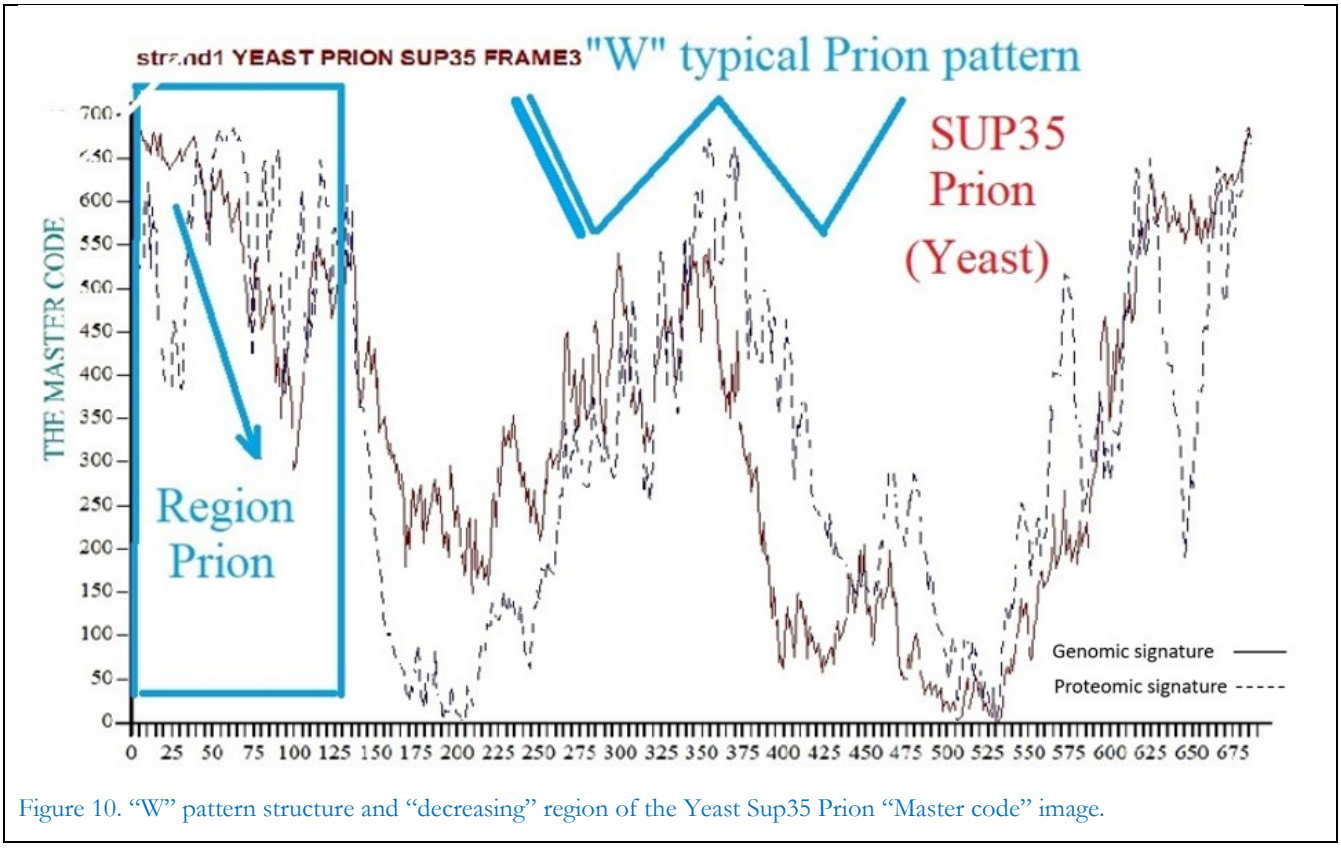


Figure 10. "W" pattern structure and "decreasing" region of the Yeast Sup35 Prion "Master code" image.

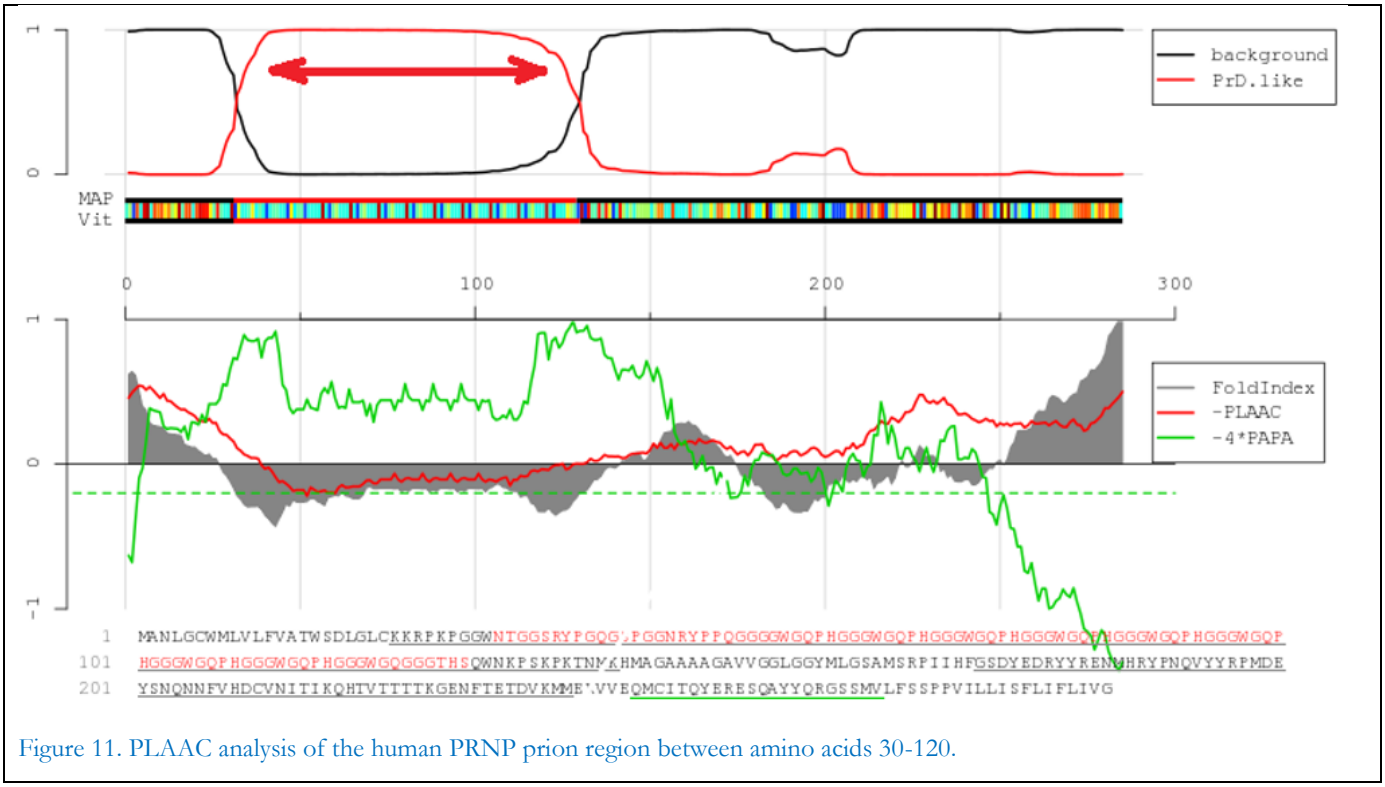


Figure 11. PLAAC analysis of the human PRNP prion region between amino acids 30-120.

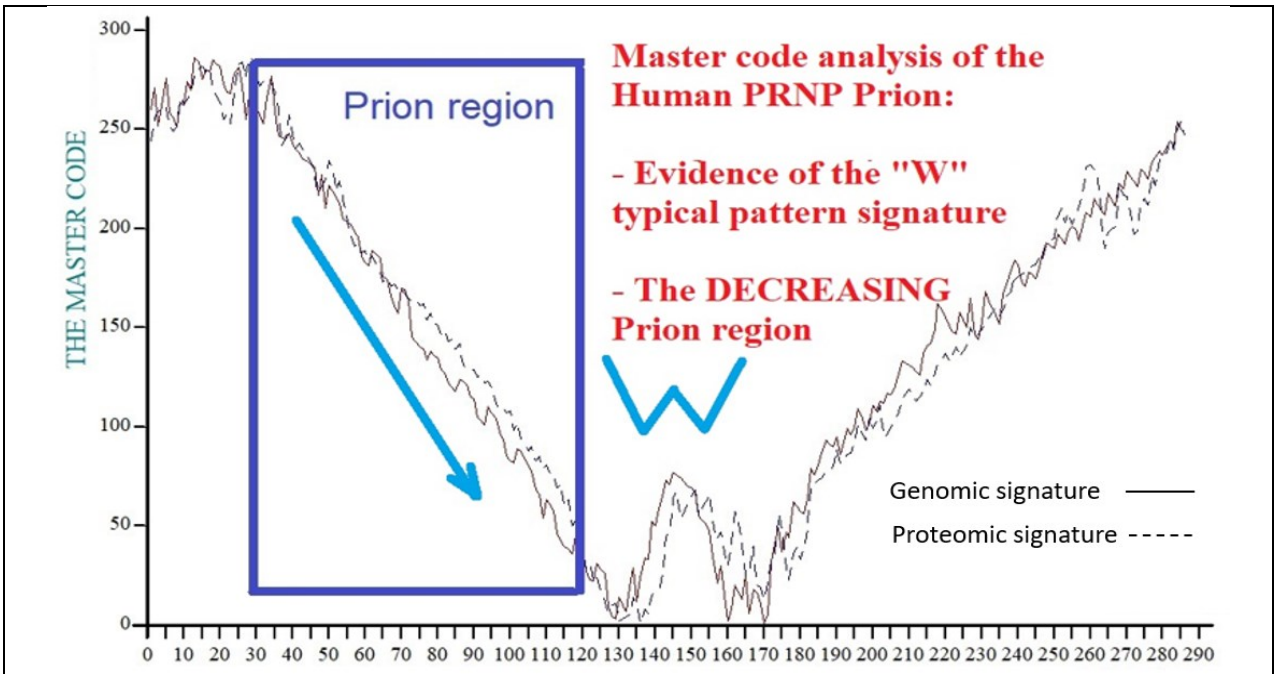


Figure 12. Confirmation of the human PRNP prion region by the "Master Code".

### *The Ovis Prion (Sheep) Prion*

In Figures 13 and 14, we apply the same systems of analysis to the sheep prion — Ovis aries prion protein (PRNP) — as already applied to human PRNP.

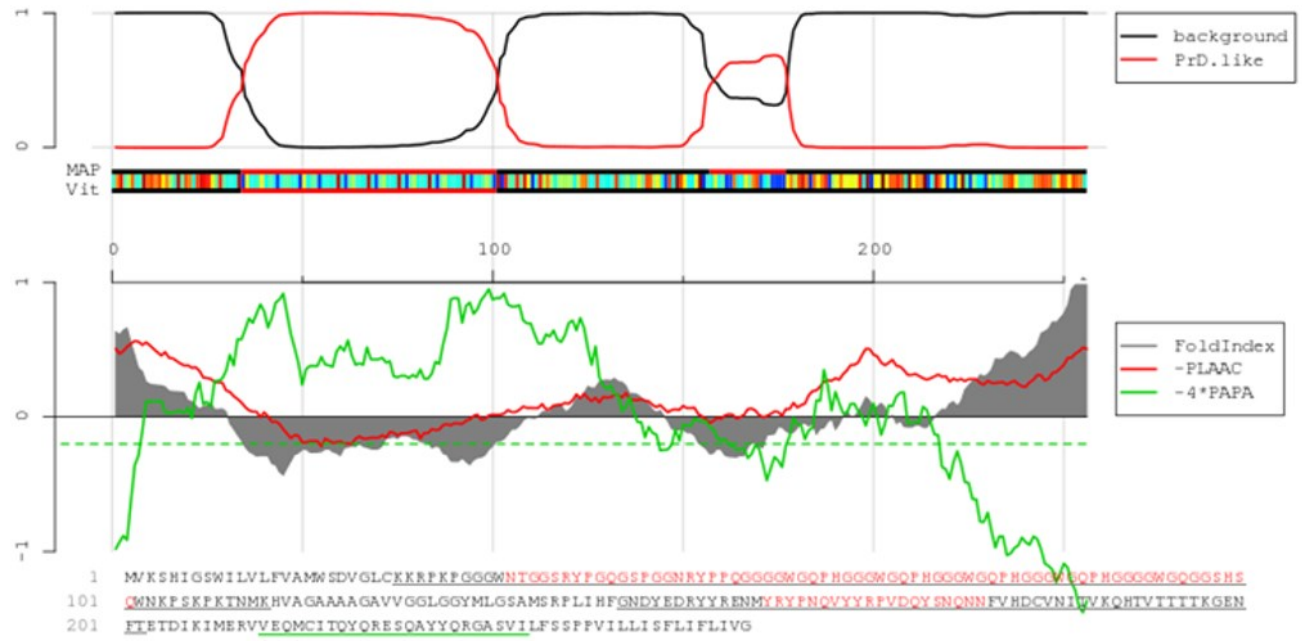


Figure 13. PLAAC analysis of the Ovis sheep PRNP prion showing evidence of that prion in the region between amino acids 40-90 and perhaps 160-180.

What follows here is the translation of the DNA coding sequence in the sheep as recorded by NCBI in the GenBank at https://www.ncbi.nlm.nih.gov/protein/NP_001009481.1?report=fasta for the major precursor of Ovis aries prion protein (PRNP), mRNA, reference sequence NM_001009481.1 (with its multiple synonyms):

CDS 161..931

/gene="PRNP"

/gene_synonym="prion; Prp; PRPC; SIP"

/note="major prion protein; prion protein (p27-30)

(Creutzfeldt-Jakob disease, Gerstmann-Strausler-Scheinker

syndrome, fatal familial insomnia)"

/codon_start=1

/product="major prion protein precursor"

/protein_id="NP_001009481.1"

/db_xref="GeneID:493887"

/translation="MVKSHIGSWILVLFVAMWSDVGLCKKRPKPGGGWNTGGSRYPGQ
GSPGGNRYPPQGGGGWGQPHGGGWGQPHGGGWGQPHGGGWGQPHGGGGWGQGGSHS
QW

NKPSKPKTNMKHVAGAAAAGAVVGGLGGYMLGSAMSRPLIHFGNDYEDRYYRENMYRY
PNQVYYRPVDQYSNQNNFVHDCVNITVKQHTVTTTTKGENFTETDIKIMERVVEQMCI
TQYQRESQAYYQRGASVILFSSPPVILLISFLIFLIVG"

Also note the results of GenPept Identical Proteins Graphics:

>NP_001009481.1 major prion protein precursor [Ovis aries]

MVKSHIGSWILVLFVAMWSDVGLCKKRPKPGGGWNTGGSRYPGQGSPGGNRYPPQGGGGWG
QPHGGGWGQPHGGGWGQPHGGGWGQPHGGGGWGQGGSHSQWNKPSKPKTNMKHVAG
AAAAGAVVGGLGGYMLGSAMSRPLIHFGNDYEDRYYRENMYRYPNQVYYRPVDQYSNQNNF
VHDCVNITVKQHTVTTTTKGENFTETDIKIMERVVEQMCITQYQRESQAYYQRGASVILFSSPP
VILLISFLIFLIVG

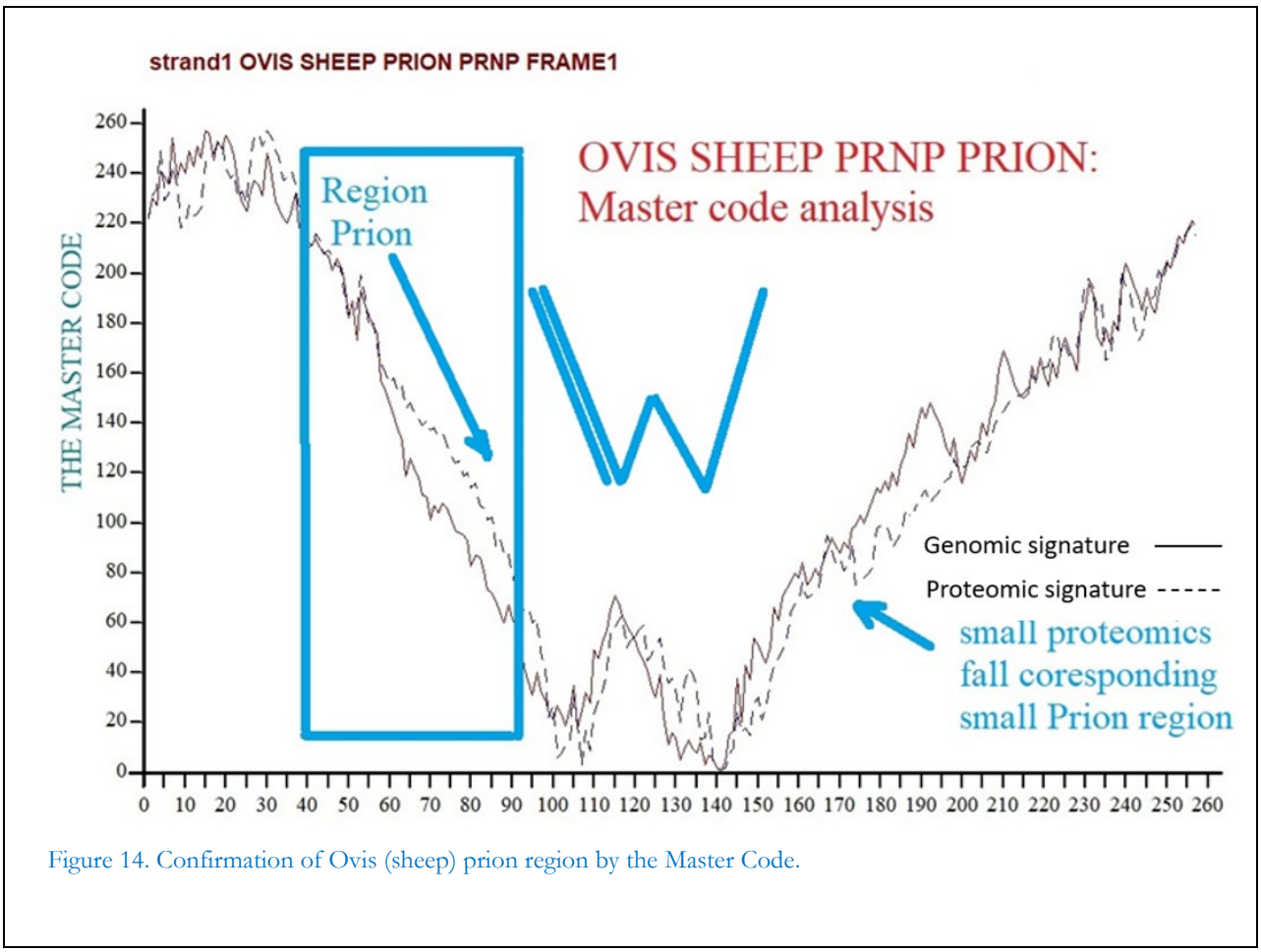


Figure 14. Confirmation of Ovis (sheep) prion region by the Master Code.


https://doi.org/10.56098/ijvtpr.v3i1.66

Then, applying the PLAAC software at http://plaac.wi.mit.edu combined with the "Master Code" as in the instance of the human prion, we get the results shown in Figure 13 and Figure 14 for the sheep PRNP.

### *The Bos Taurus (cow) prion*

Next, we move on to the Bos Taurus (cow) prion at https://www.ncbi.nlm.nih.gov/nuccore/AB457178.1 as analysed in Figures 15 and 16. At the GenBank for reference sequence AB457178.1 we find the following:

gene 1..1352

/gene="prn"

CDS 11..805

/gene="prn"

/note="alternative splicing: see also Acc# AB457179.1"

/codon_start=1

/product="prion protein"

/protein_id="BBD75290.1"

/translation="MVKSHIGSWILVLFVAMWSDVGLCKKRPKPGGGWNTGGSRYPGQ
GSPGGNRYPPQGGGGWGQPHGGGWGQPHGGGWGQPHGGGWGQPHGGGWGQPHGGGG
WGQGGTHGQWNKPSKPKTNMKHVAGAAAAGAVVGGLGGYMLGSAMSRPLIHFGSDYEDRY
YRENMHRYPNQVYYRPVDQYSNQNNFVHDCVNITVKEHTVTTTTKGENFTETD

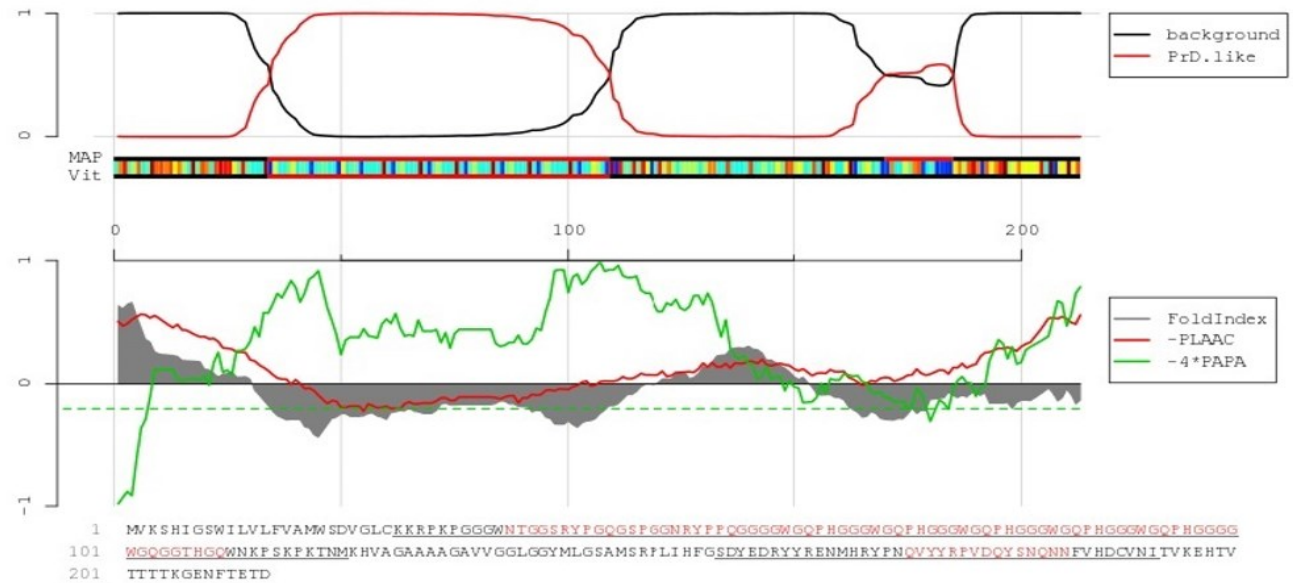


Figure 15. PLAAC analysis of the Bos Taurus, Mad Cow Disease, showing evidence of a prion region between amino acids 40-90 and perhaps 170-180.

With PLAAC from http://plaac.wi.mit.edu, we get the results shown in Figures 15 as confirmed in Figure 16 with the "Master Code".

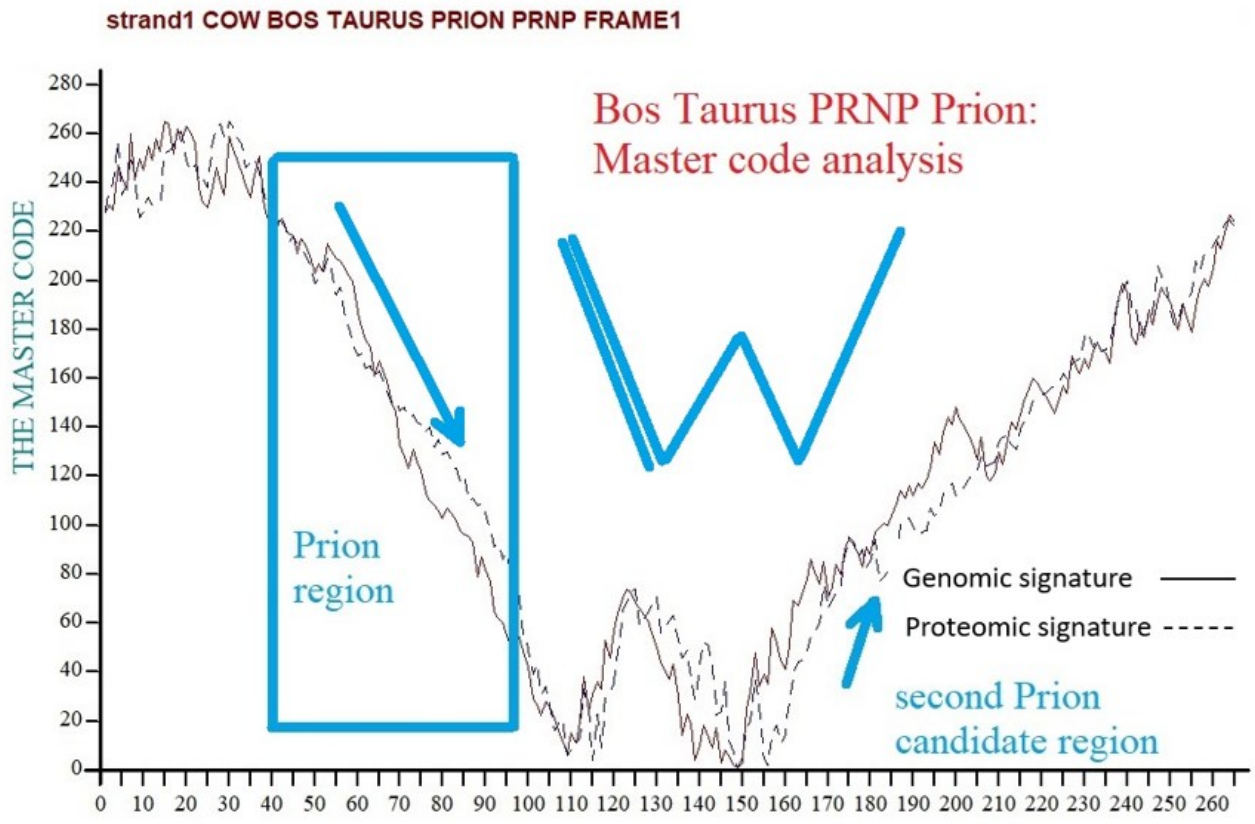


Figure 16. Confirmation of Bos Taurus (Mad Cow Disease) prion region by the Master Code.

**ANOTHER PRION RISK: TDP-43 PRIONS**

Classen (2021a) suggested that the targeted spike protein could interact potentially converting intracellular RNA binding protein TAR and DNA binding protein TDP-43 fusing them in Sarcoma (FUS) into their pathologic prion conformations. Here we analyse TDP-43 prion properties noted previously by Nonaka et al. (2013) and by McAlary et al. (2019). Beginning at the NCBI website URL: https://www.ncbi.nlm.nih.gov/gene?term=(tdp43[gene])%20AND%20(Homo%20sapiens[orgn])%20AND%20alive[prop]%20NOT%20newentry[gene]&sort=weight we find the following for TARDBP TAR DNA binding protein [ Homo sapiens (human) ] Gene ID: 23435, at https://www.ncbi.nlm.nih.gov/nuccore/NM_007375.4 which we analyze as in prior instances as shown respectively in Figures 17 and 18. The NCBI reference sequence NM_007375.4 yields the following:

CDS 103..1347

/gene="TARDBP"

/gene_synonym="ALS10; TDP-43"

/note="TAR DNA-binding protein-43"

/codon_start=1

/product="TAR DNA-binding protein 43"

/protein_id="NP_031401.1"

/db_xref="CCDS:CCDS122.1"

/db_xref="GeneID:23435"

/db_xref="HGNC:HGNC:11571"

/db_xref="MIM:605078"

/translation="MSEYIRVTEDENDEPIEIPSEDDGTVLLSTVTAQFPGACGLRYR
NPVSQCMRGVRLVEGILHAPDAGWGNLVYVVNYPKDNKRKMDETDASSAVKVKRAVQK
TSDLIVLGLPWKTTEQDLKEYFSTFGEVLMVQVKKDLKTGHSKGFGFVRFTEYETQVK
VMSQRHMIDGRWCDCKLPNSKQSQDEPLRSRKVFVGRCTEDMTEDELREFFSQYGDVM
DVFIPKPFRAFAFVTFADDQIAQSLCGEDLIIKGISVHISNAEPKHNSNRQLERSGRF
GGNPGGFGNQGGFGNSRGGGAGLGNNQGSNMGGGMNFGAFSINPAMMAAAQAALQSSW
GMMGMLASQQNQSGPSGNNQNQGNMQREPNQAFGSGNNSYSGSNSGAAIGWGSASNAG
SGSGFNGGFGSSMDSKSSGWGM

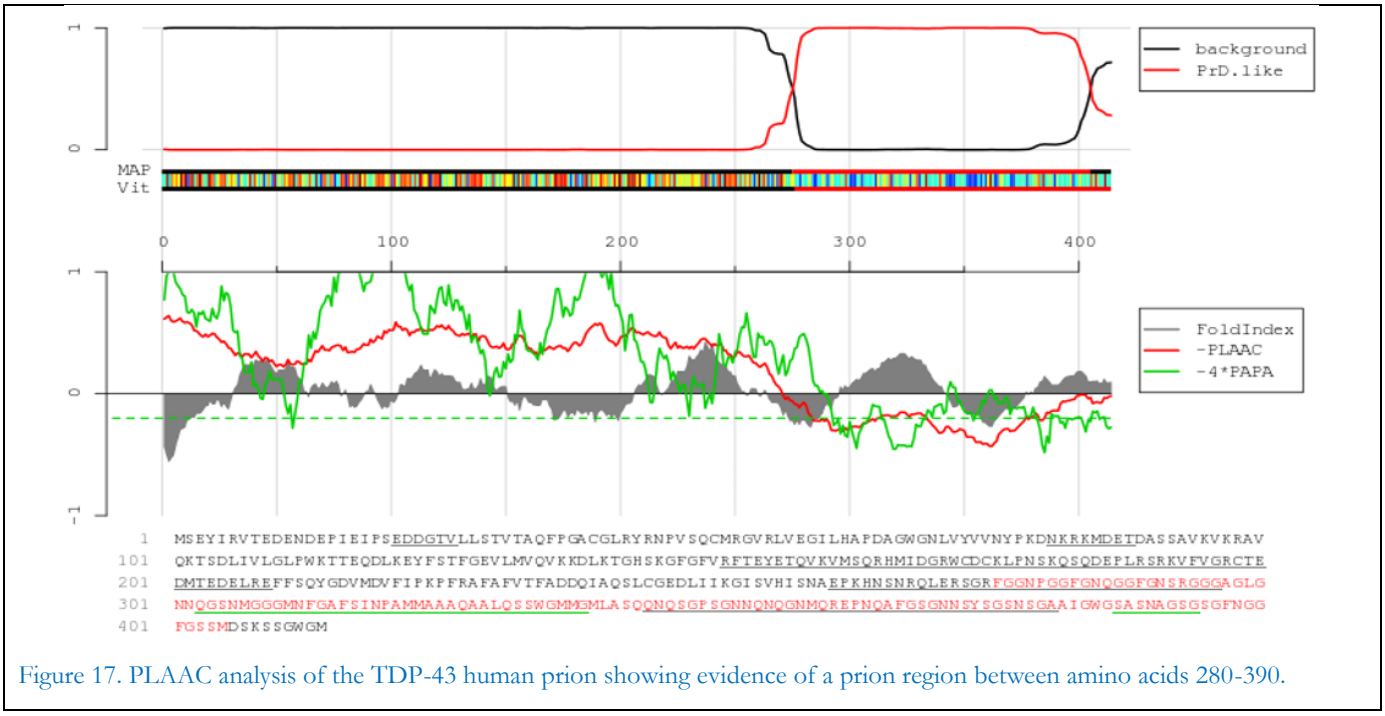


Figure 17. PLAAC analysis of the TDP-43 human prion showing evidence of a prion region between amino acids 280-390.

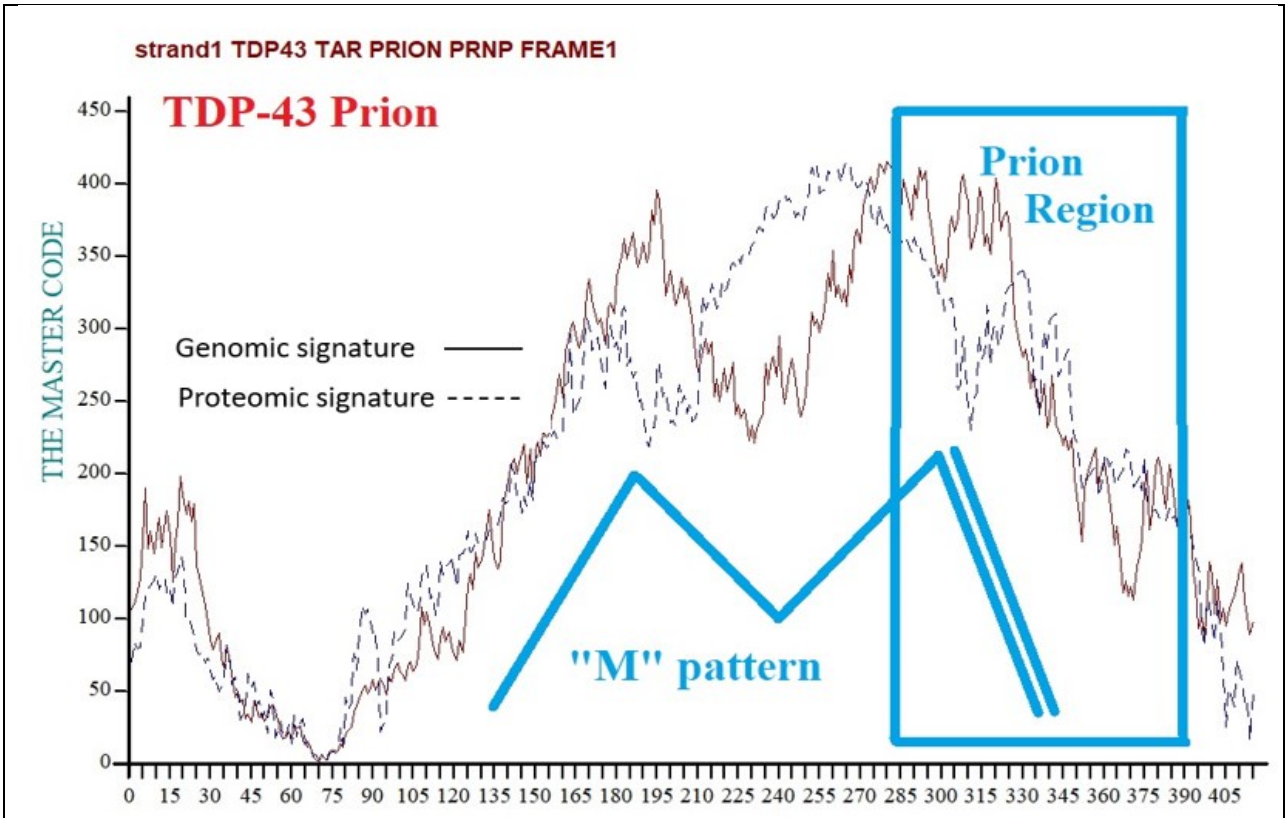


Figure 18. Confirmation of human TDP-43 prion region by the Master Code.

**PRION FUNCTION DISAPPEARS IN OMICRON**

Whereas all the SARS-CoV-2 variants derived either by natural mutation or by "vaccine" engineering from the Wuhan spike origin show a prion region, the Omicron variant, upon analysis does not. Figure 19 shows the prion region in the SARS-CoV-2 Wuhan virus analyzed with PLAAC software from http://plaac.wi.mit.edu. Here is the PLAAC zoom on the 38 amino acid sequence (473-510) shows that the "window prion" of the Wuhan spike is present:

SKVGGNYNYLYRLFRKSNLKPFERDISTEIYQAGSTPCNGVEGFNCYFPLQSYGFQPTNGVGYQ
PYRVVVLSFELLHAPATVCGPKKSTNLVKNKCVN

That presence is confirmed in Figure 20 using the maser code.

However, when we do the ZOOM on the 38 amino acids (473-510) looking for the WINDOW PRION in the Omicron spike with PLAAC software (http://plaac.wi.mit.edu) as shown in Figure 21, it is gone.

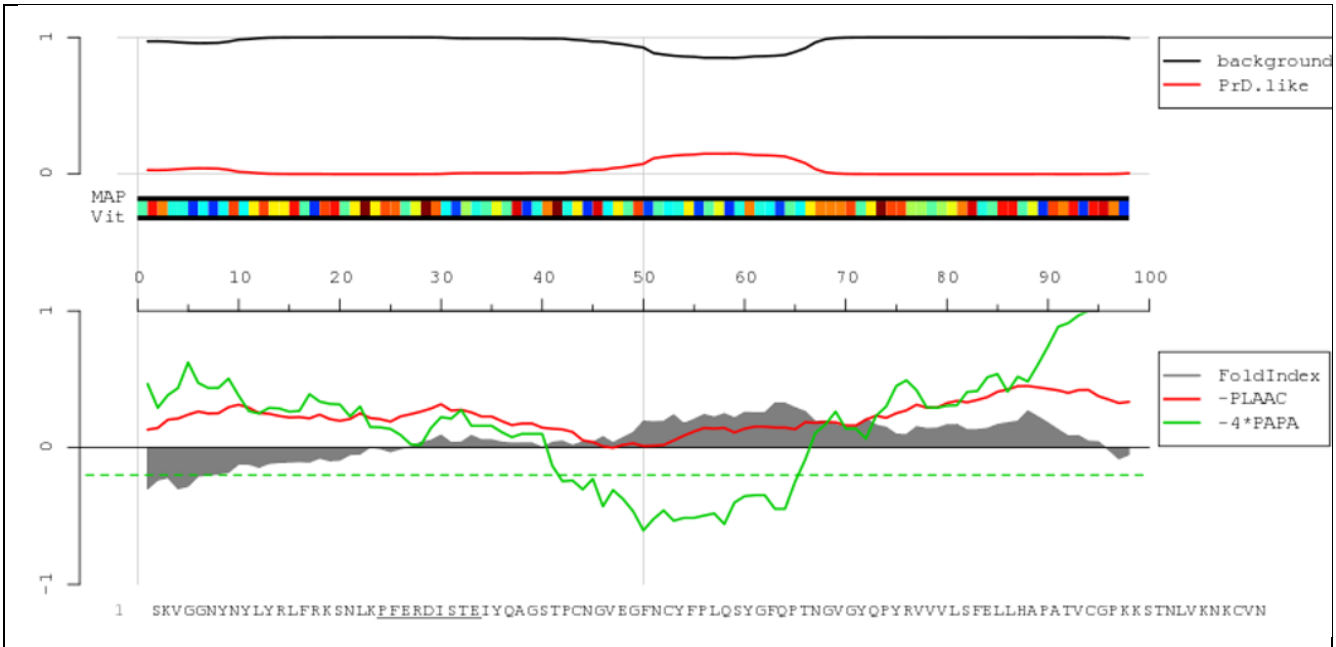


Figure 19. PLAAC evidence of a prion region overlapping the Wuhan SARS-CoV-2 spike prion consisting of part of its sequence. We also examined and considered the 100 amino acids flanking this prion region on both sides of what is shown.

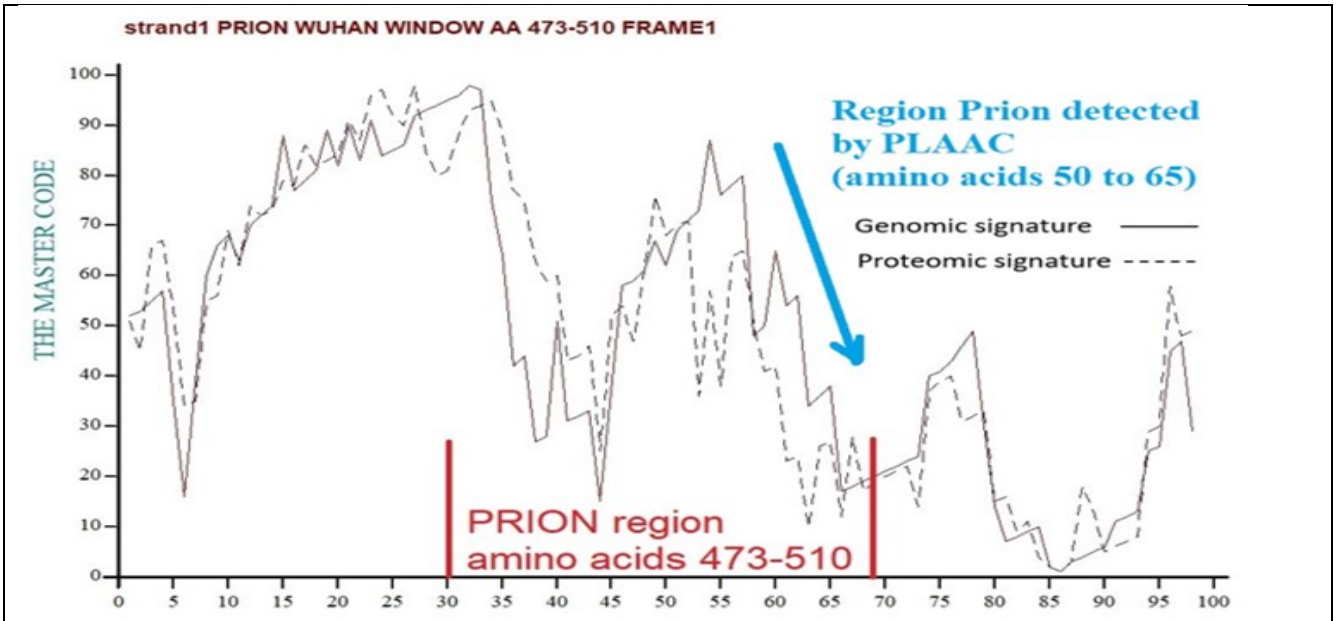


Figure 20. Master code confirmation of a prion region in the 100 amino acids overlapping the Wuhan prion region. All of the spike proteins from the SARS-CoV-2 Wuhan variant, with the singular exception of Omicron as we will show later in this paper, have the prion area designated here that we believe is causally associated with the new form of rapidly developing and quickly progressing fatal Creutzfeldt-Jacob Disease.

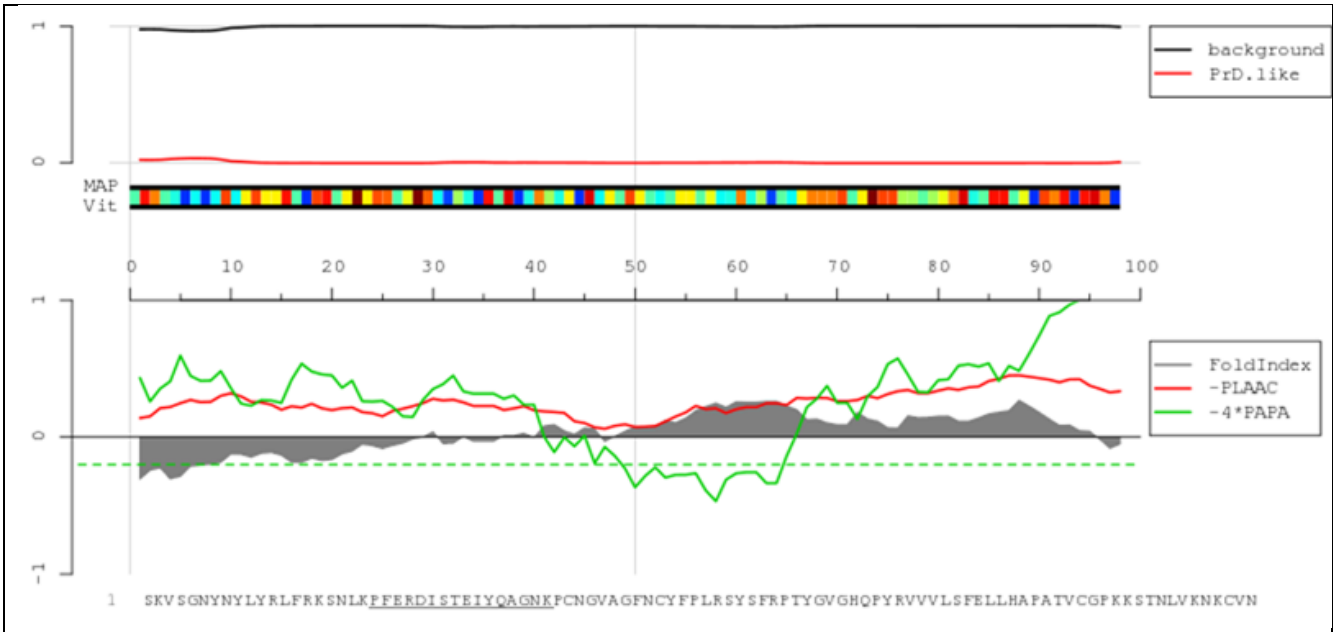


Figure 21. PLAAC evidence in the 100 amino acid "window" in the SoCal (southern California) variant of Omicron (the first USA Omicron case reported in the literature) that the prion region disappears totally. The question is, how could it be completely erased? Is this the work of the innate, adaptive, and complement human immune defense systems built into our own DNA (Santiago, 2022b)? Or what else can it be? These questions constitute an important part of the remaining mystery to be solved.

Comparing the spikes of the Wuhan and Omicron viruses in Figures 19, 20 and 21, it seems we should analyze closely the incidence of the 8 amino acid mutations located in the prion region (473, 474, and so on up to 510). These are the amino acids which differentiate the Wuhan parent strain and the latest Omicron variant. The 8 mutations in question are the following from https://covariants.org/variants/21K.Omicron:

1. **S:**S477N
2. **S:**T478K
3. **S:**E484A
4. **S:**Q493R
5. **S:**G496S
6. **S:**Q498R
7. **S:**N501Y
8. **S:**Y505H

The 38 amino acids of the Omicron prion spike are specified in the 114 bases of DNA as follows:

TATCAGGCCGGTAACAAACCTTGTAATGGTGTTGCAGGTTTTAATTGTTACTTTCCTTTACGA
TCATATAGTTTCCGACCCACTTATGGTGTTGGTCACCAACCATACAGAGTA

And, here they are marked in their positions between those numbered 473 to 510:

**473** **510**

**YQAGNKPCNGVAGFNCYFPLRSYSFRPTYGVGHQPYRV**

XX X X X X X X

1 2 3 4 5 6 7 8

PLAAC analysis of the same 38 amino acid sequence demonstrates the *total* disappearance of the prion function although the presence of these 38 amino acids is conserved in the Omicron spike protein as seen in Figure 22.

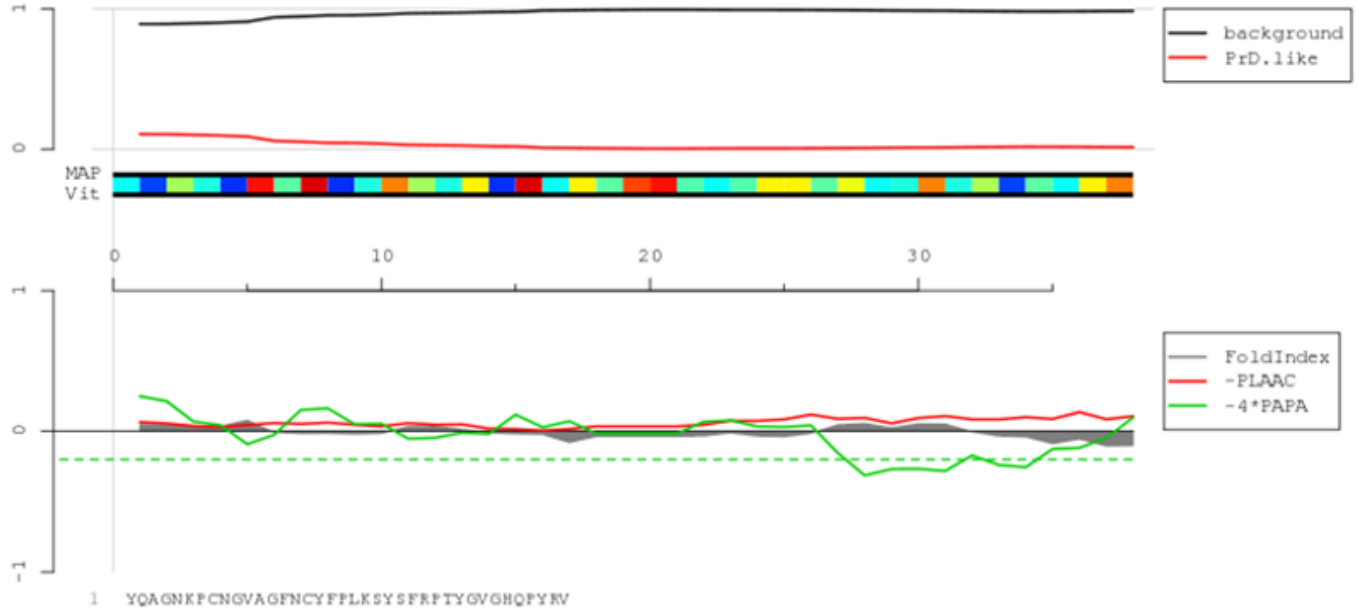


Figure 22. Here we are zooming in on the prion region, or, rather where it is expected to be in the Omicron variant, but as the reader can see, that prion function has disappeared completely. It has vanished from the SoCal Omicron variant. It is no longer present in the sequence of 38 amino acids spelled out immediately above this caption.

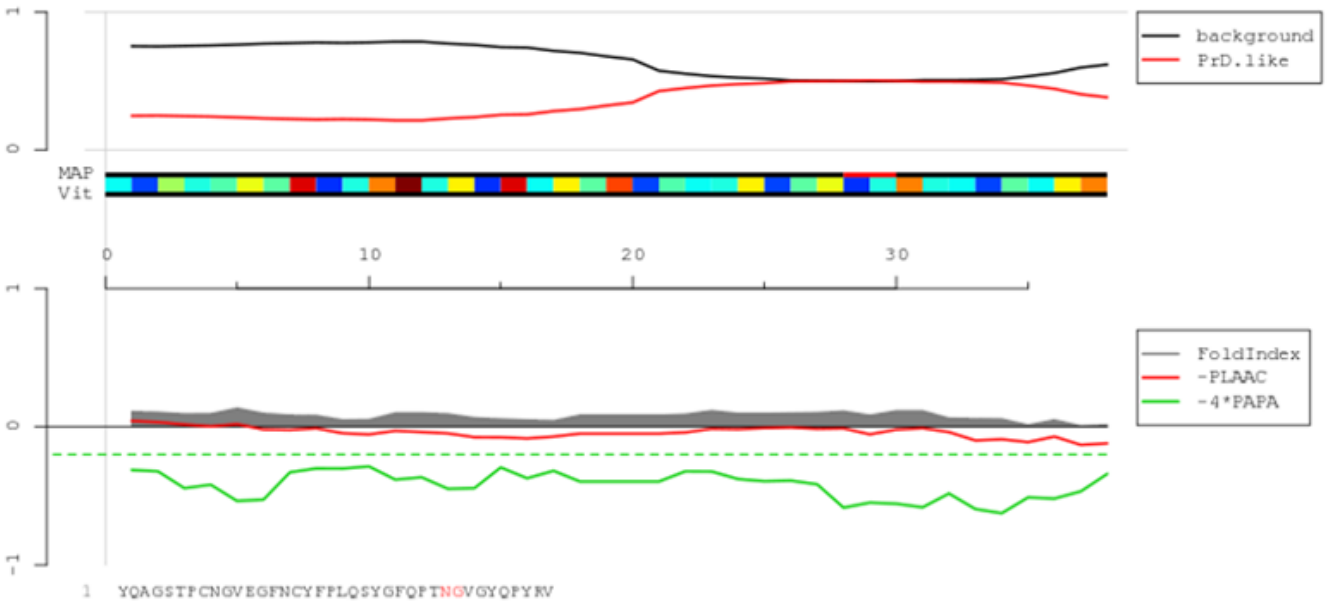


Figure 23. The PLAAC software shows the prion function plainly present in the Wuhan initial sequence, though in Figure 16 we found it was entirely absent from the SoCal variant known as Omicron.

Next, we perform the same analysis on the Wuhan parent strain keeping in mind that all the COVID-19 "vaccines" — the same ones that have been injected into 5.4 billion human beings (Pharmaceutical Technology, 2022) — have been constructed from that same Wuhan spike sequence. In Figure 23, contrary to what is missing from the Omicron variant, the potential function of the prion is well revealed by the PLAAC software. But it seems reasonable to ask what is the "PLAAC distance" between the 2 respective results for the Omicron variant compared against the original Wuhan spike. What follows immediately is a characterization of the Wuhan prion spike consisting of nucleotides prion region of 114 bases. They are followed in Figure 24 by the contrast between the two spikes.

ZOOMPRIONWUHAN <== SPIKREF[1416 on 114]

ZOOMPRIONWUHAN

TATCAGGCCGGTAGCACACCTTGTAATGGTGTTGAAGGTTTTAATTGTTACTTTCCTTTACAA
TCATATGGTTTCCAACCCACTAATGGTGTTGGTTACCAACCATACAGAGTA


https://doi.org/10.56098/ijvtpr.v3i1.66

- \+   -   +   +   +   +   +

WUHAN YQAG ST PCNGV E GFNCYFPL Q SY G F Q PT N GVG Y QPYRV

OMICRON YQAG **NK** PCNGV **A** GFNCYFPL **R** SY **S** F **R** PT **Y** GVG **H** QPYRV

\+ -   +   -   -   -   -   -

Distances - 7 10 2 10 2 6 ==> - 37

Distances + 5 12 ==> + 17

Difference reinforcing Prion function : - 20

Figure 24. Prion nature classification hierarchy between the 20 amino acids.

Given the analysis in Figure 24, we can conclude by asserting that the 8 amino acid mutations, accounting for 21% of the small region in question *actually* account for the *total disappearance* of the prion function from the SoCal Omicron variant.

Two questions then arise and demand attention:

*1/ Was this Prion region "natural" (as some claimed; notably Fauci's collaborators as documented by Kennedy, 2021) or was it chimerical (a man-made bioweapon of some sort intended to do harm) along the lines of Fleming, 2021, as well as articles published previously in this journal by Oller, 2021, Hughes 2022, Santiago, 2022a, and by Kyrie and Broudy, 2022a, 2022b) when the Wuhan virus emerged?*

*2/ Was the suppression of the prion function natural following the "humanization" of the virus or was it also engineered? If the latter, to what end? To cover up the prior engineering of the original Wuhan virus? Or, is the human immune defense complex capable of such precise demolition of the prion region in the spike? If the latter, why did it not apply to the Wuhan variants prior to Omicron?*

*The foregoing questions remain "open" and are yet to be answered definitively.*

## POSSIBLE PRION FUNCTIONS IN 25 SPIKE PROTEINS FROM SARS-COV2 STRAINS, VARIANTS, OR "VACCINES" REPRESENTATIVE OF THE EVOLUTION OF THE SARS-COV2 VIRUS PANDEMIC.

We studied the spike sequences of 25 SARS-CoV-2 genomes. In these spikes we searched for possible regions likely to have the functionality of a prion. For this we use the PLAAC bioinformatics software (Lancaster et al., 2014) and the "Master Code". In Figure 25 we recall the 8 amino acid mutations differentiating the prion regions from the spikes of Wuhan SARS-CoV-2 and Omicron.

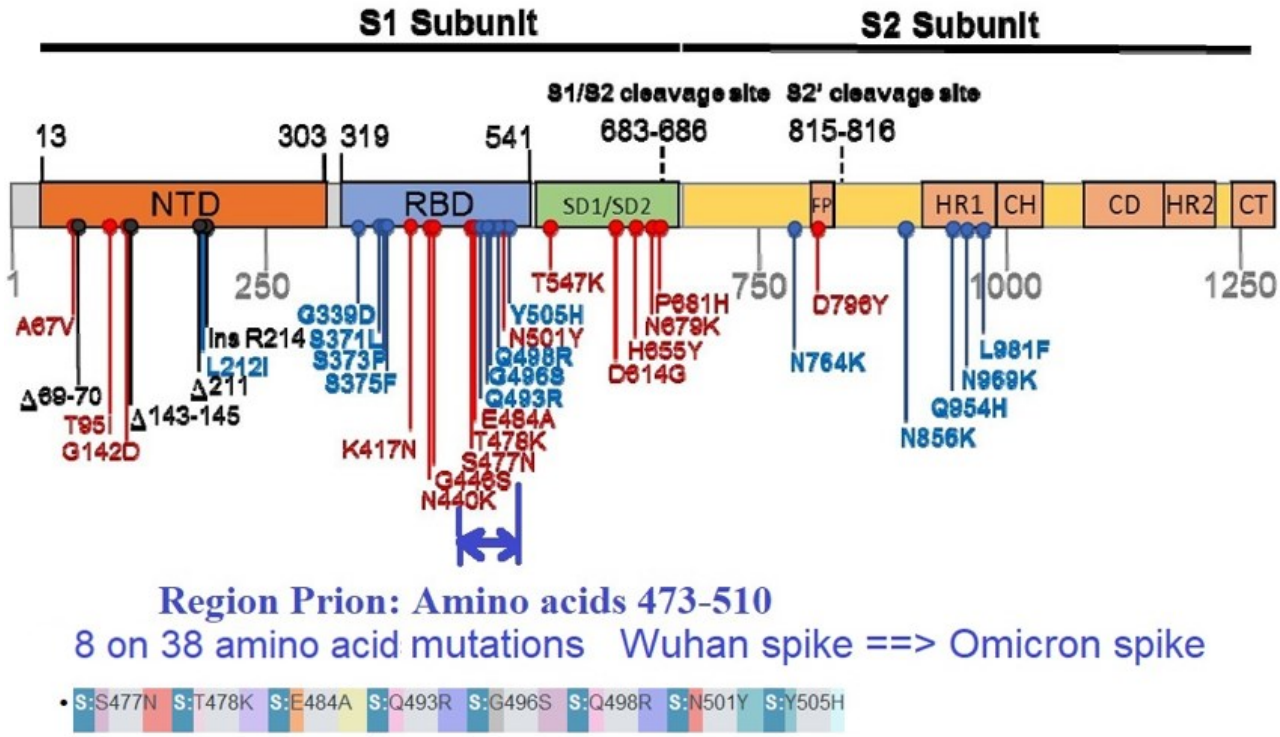


Figure 25. The 8 amino acid mutations differentiating the prion regions from the Wuhan SARS-CoV-2 and Omicron spikes.

Figure 25 shows the Genomic/Proteomic image of the Master Code relating to the region of 100 amino acids flanking the small prion region of 38 amino acids.

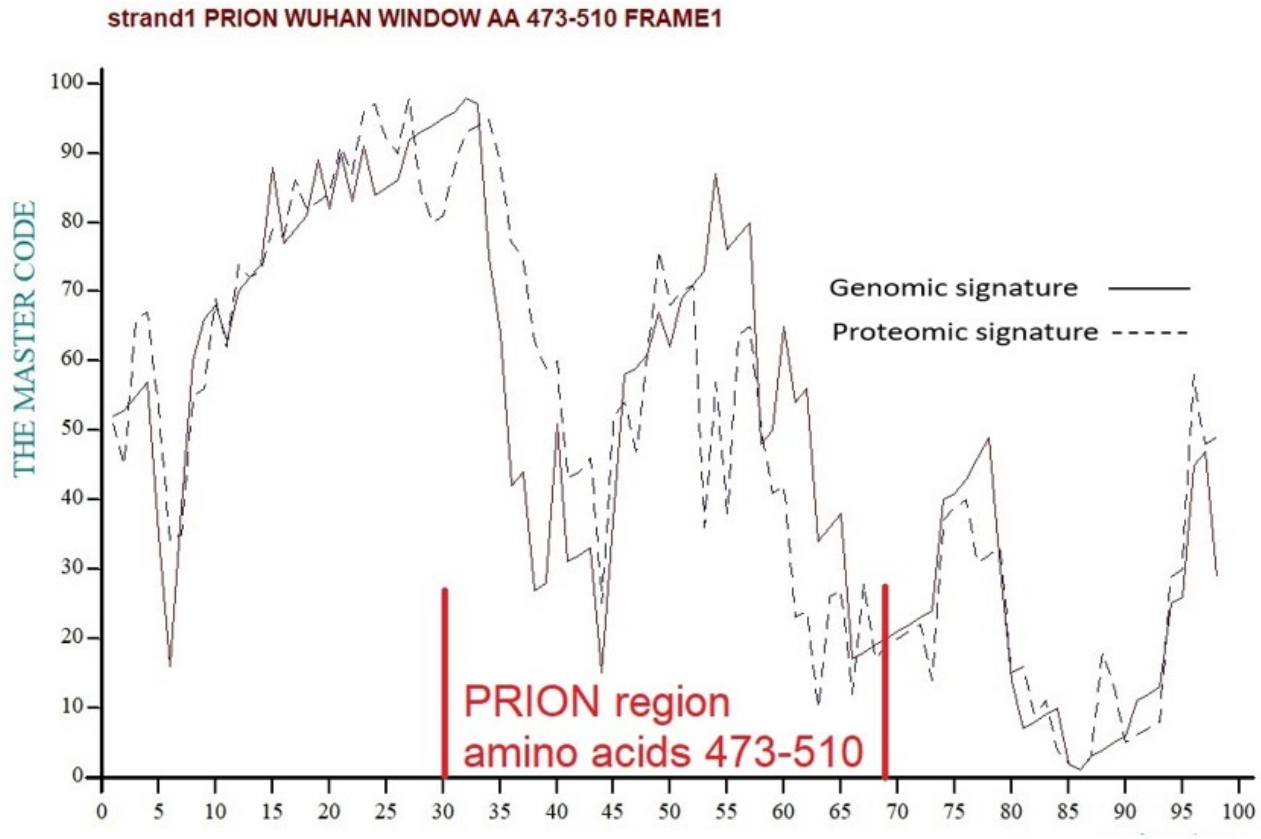


Figure 26. Genomic/proteomic image of the Master Code relating to the region of 100 amino acids flanking the small prion region of 38 amino acids. All spike protein sequences from the initial SARS-CoV-2 (Wuhan virus) and all of its variants (that have been studied or reported) including the Pfizer and Moderna injectables have this prion area which we suggest is the potential cause of the new rapidly developing Creutzfeldt-Jacob Disease.

### *Analysing the Representative Strains of the 10 Main SARS-CoV-2 Variants*

Figures 23 through 26 demonstrate with both the PLAAC software and the Master Code method the presence of the prion region centered on (or very near) amino acid 500 of the spike as seen in Figure 21. We see that this prion is not only present in the Delta variant of SARS-CoV-2 (Figure 23) but also in the Pfizer and Moderna "vaccine" spikes (Figures 24-26). Logically, it must be in *all* the COVID-19 "vaccines" because they were built from the spike of the Wuhan SARS-CoV-2 virus.

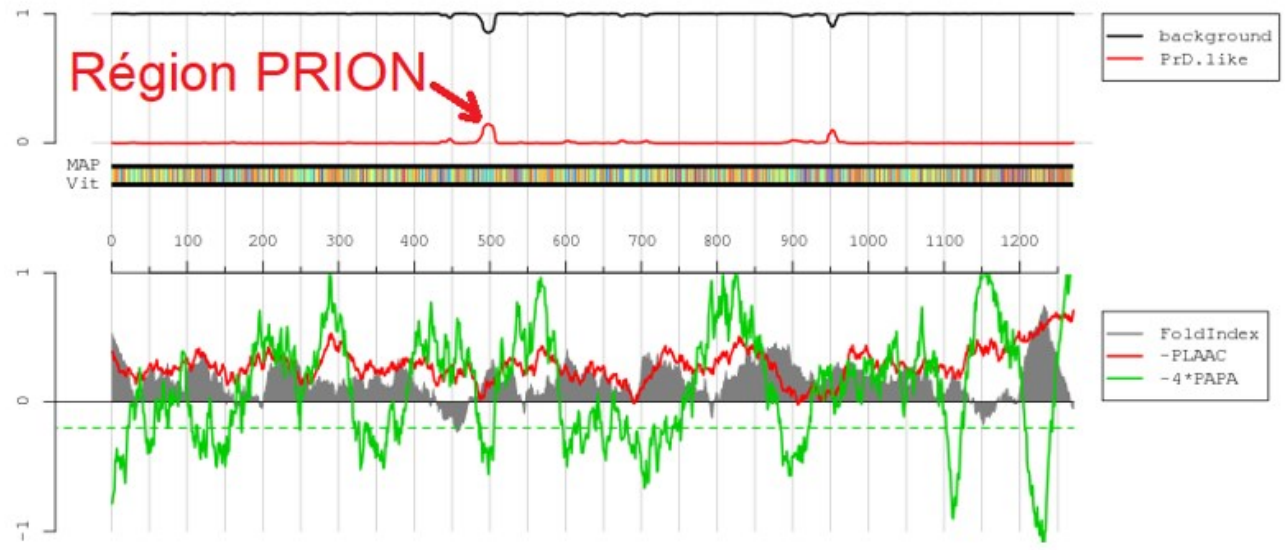


Figure 27. PLAAC software demonstrates the presence of the Prion region around amino acids 500 of the spike of Moderna.

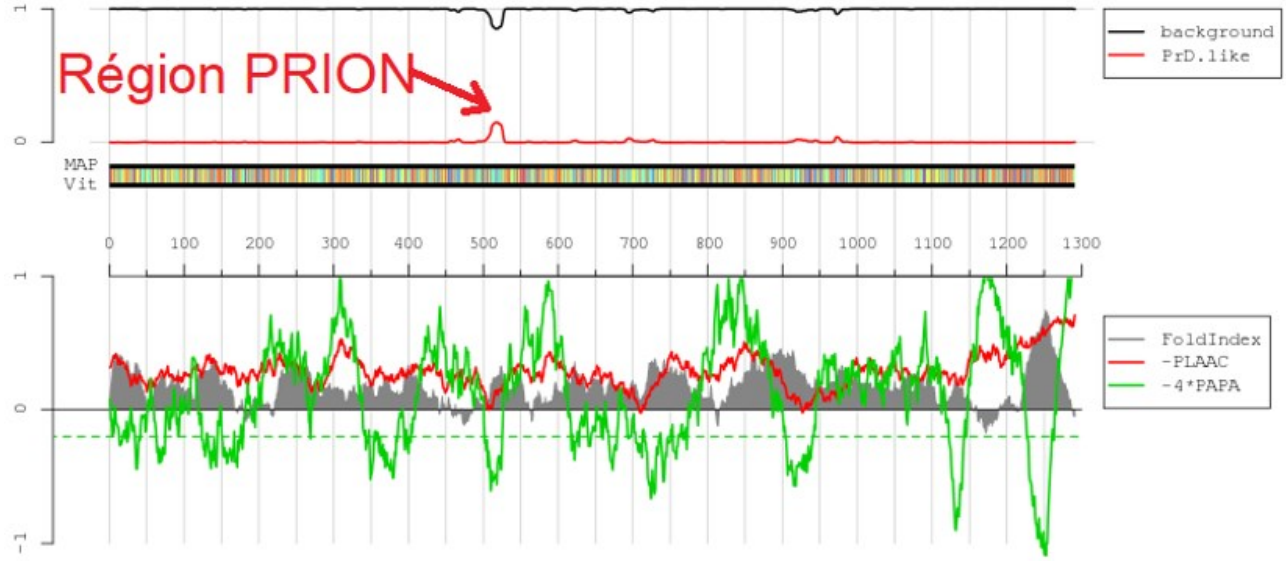


Figure 28. PLAAC software demonstrates the presence of the prion region around amino acid 500 of the spike of the Pfizer COVID-19 "vaccine".


https://doi.org/10.56098/ijvtpr.v3i1.66

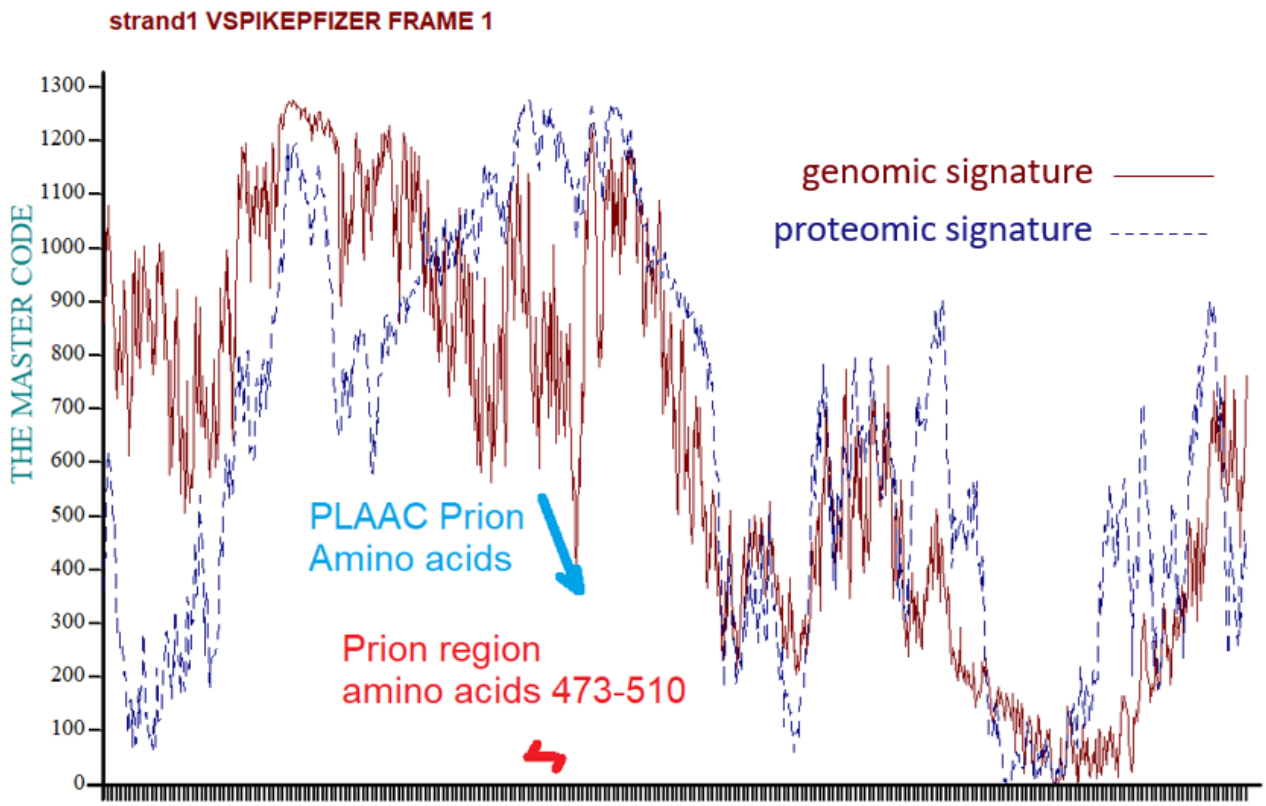


Figure 29. The Master Code method provides a global analysis of the roughness, the fractal texture (as defined by the mathematician Mandelbrot, of both genomic and proteomic signatures of the spike prion region.

As demonstrated in Perez (2021), it can be seen that, compared to the Wuhan spike prion region of Figure 29, the prion region of the Pfizer vaccine, especially in its genomic signature, has chaotic Master Code curves with *fractal roughness* (Mandelbrot, 1975, 1982, 2010; also Pellionisz, 2008, 2012).[4] This *roughness* results from the "G" base doping of this sequence as discussed previously in this journal by Seneff and colleagues (Seneff & Nigh, 2021; Seneff, Kyriakopoulos, et al., 2022; Seneff, Nigh, et al., 2022), the purpose of which seems to be to increase the stability of the mRNA without changing the amino acid sequence. As Perez (2021) has noted, the *fractal* character of the genomic signature, depends on the vagueness allowed (the "wobble" as it has been called by some; for example, Mauro & Chappell, 2014) in the translation linking of codons to particular amino acids.

A PLAAC analysis of the Moderna "vaccine" spike, the amino acid sequence of which follows immediately here, again reveals the prion region also found in Pfizer, Delta, and the Wuhan spikes:

MFVFLVLLPLVSSQCVNLTTRTQLPPAYTNSFTRGVYYPDKVFRSSVLHSTQDLFLPFFSNVTWFH
AIHVSGTNGTKRFDNPVLPFNDGVYFASTEKSNIIRGWIFGTTLDSKTQSLLIVNNATNVVIKVC

[4] **Editor's Note:** The references to Pellionisz were added by the Editor with thanks to Andras J. Pellionisz who holds PhD degrees in computing, biology, and physics. We are also grateful to him for reviewing this work prior to its appearnce here in the *IJVTPR*.

EFQFCNDPFLGVYYHKNNKSWMESEFRVYSSANNCTFEYVSQPFLMDLEGKQGNFKNLREFV
FKNIDGYFKIYSKHTPINLVRDLPQGFSALEPLVDLPIGINITRFQTLLALHRSYLTPGDSSSGWTA
GAAAYYVGYLQPRTFLLKYNENGTITDAVDCALDPLSETKCTLKSFTVEKGIYQTSNFRVQPTES
IVRFPNITNLCPFGEVFNATRFASVYAWNRKRISNCVADYSVLYNSASFSTFKCYGVSPTKLNDLC
FTNVYADSFVIRGDEVRQIAPGQTGKIADYNYKLPDDFTGCVIAWNSNNLDSKVGGNYNYLYR
LFRKSNLKPFERDISTEIYQAGSTPCNGVEGFNCYFPLQSYGFQPTNGVGYQPYRVVVLSFELLH
APATVCGPKKSTNLVKNKCVNFNFNGLTGTGVLTESNKKFLPFQQFGRDIADTTDAVRDPQTL
EILDITPCSFGGVSVITPGTNTSNQVAVLYQDVNCTEVPVAIHADQLTPTWRVYSTGSNVFQTRA
GCLIGAEHVNNSYECDIPIGAGICASYQTQTNSPRRARSVASQSIIAYTMSLGAENSVAYSNNSIAI
PTNFTISVTTEILPVSMTKTSVDCTMYICGDSTECSNLLLQYGSFCTQLNRALTGIAVEQDKNTQ
EVFAQVKQIYKTPPIKDFGGFNFSQILPDPSKPSKRSFIEDLLFNKVTLADAGFIKQYGDCLGDIA
ARDLICAQKFNGLTVLPPLLTDEMIAQYTSALLAGTITSGWTFGAGAALQIPFAMQMAYRFNGIG
VTQNVLYENQKLIANQFNSAIGKIQDSLSSTASALGKLQDVVNQNAQALNTLVKQLSSNFGAIS
SVLNDILSRLDPPEAEVQIDRLITGRLQSLQTYVTQQLIRAAEIRASANLAATKMSECVLGQSKRV
DFCGKGYHLMSFPQSAPHGVVFLHVTYVPAQEKNFTTAPAICHDGKAHFPREGVFVSNGTHW
FVTQRNFYEPQIITTDNTFVSGNCDVVIGIVNNTVYDPLQPELDSFKEELDKYFKNHTSPDVDL
GDISGINASVVNIQKEIDRLNEVAKNLNESLIDLQELGKYEQYIKWPWYIWLGFIAGLIAIVMVT
IMLCCMTSCCSCLKGCCSCGSCCKFDEDDSEPVLKGVKLHYT

Using the PLAAC software, Figure 30 shows the spike prion of the Wuhan SARS-CoV-2 present exactly as expected near the 500th amino acid in the Moderna spike.

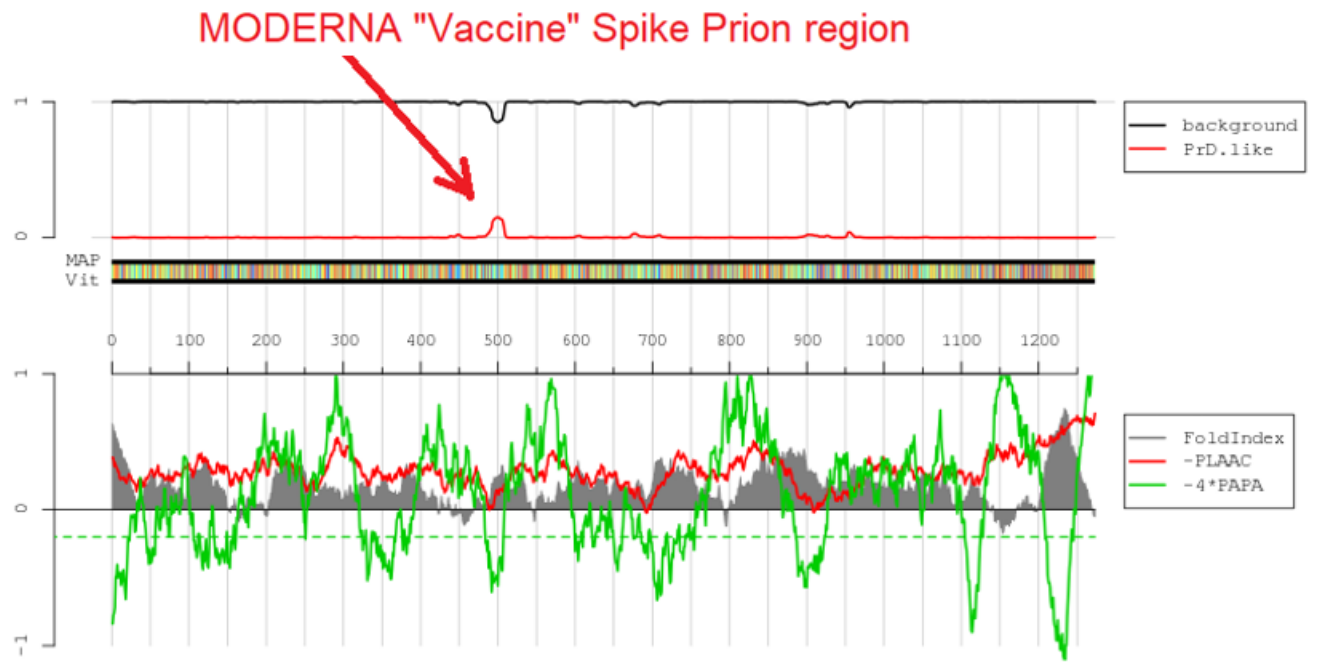


Figure 30. PLAAC software demonstrates the presence of the prion region around amino acid 500 in the Moderna spike protein just as was found also for Pfizer as shown in Figure 22.

We note in Table 4 that the prion region, however, does not exist in the Bat RaTG13 which was sometimes touted as the source of the Wuhan virus (for instance, see Cohen, 2020, and Swarajya Staff, 2020). Curiously, however, it is present in ScovZC45 and ScovZXC21, though it is located within the first 50 amino acids of their spikes and not in the area of the 500th amino acid.

**Table 4**
**Presence of the Prion Region in *All* historical SARS-CoV-2 Spikes Except Bat RaTG13**

| **Identification of main SARS-CoV2, variants and vaccines** | **PRION region amino acids 473-510** | **Notes** |
|---|---|---|
| SARS-CoV2 Wuhan | YES | |
| ALPHA (UK) | YES | |
| BETA (South Africa) | YES | |
| GAMMA (Brazil) | YES | |
| DELTA (India) | YES | |
| **mRNA vaccins Pfizer** | **YES** | |
| **mRNA vaccins Moderna** | **YES** | |
| batRaTG13 | NO | Prion region totally absent |
| ScovZC45 | YES (shifted) | In the 50 first amino acids |
| ScovZXC21 | YES (shifted) | In the 50 first amino acids |

## ANALYSING THE FIRST 7 CASES OF OMICRON IN THE WORLD

In this section we turn to study of the very first cases of patients with Omicron, in South Africa, Europe, the USA, and Canada. In ALL of these cases, the prion region has disappeared (Table 5).

**Table 5**
**The First Seven Omicron Cases with No Prion Region in *Any* of Them**

| **Ref** | **Identification of first Omicron worldwide patient strains** | **Prion region** |
|---|---|---|
| SOSA1 | **One of the 3 first cases in South Africa** | none |
| SOSA2 | **One of the 3 first cases in South Africa** | none |
| SOSA3 | **One of the 3 first cases in South Africa** | none |
| SOBEL | **First case in Belgium** | none |
| SOCAN | **First case in Canada** | none |
| SOMIN | **Second case in USA and first case in Minesota** | none |
| SUK | **First case in UK** | none |
| **Results** | | **None** |

### ANALYSING 8 USA OMICRON PATIENTS RANDOMLY SELECTED FROM GENBANK

In this subsection, we study the cases of eight patients affected by Omicron and coming from different states in the USA. In *ALL* 8 of them, again, the prion region has disappeared.

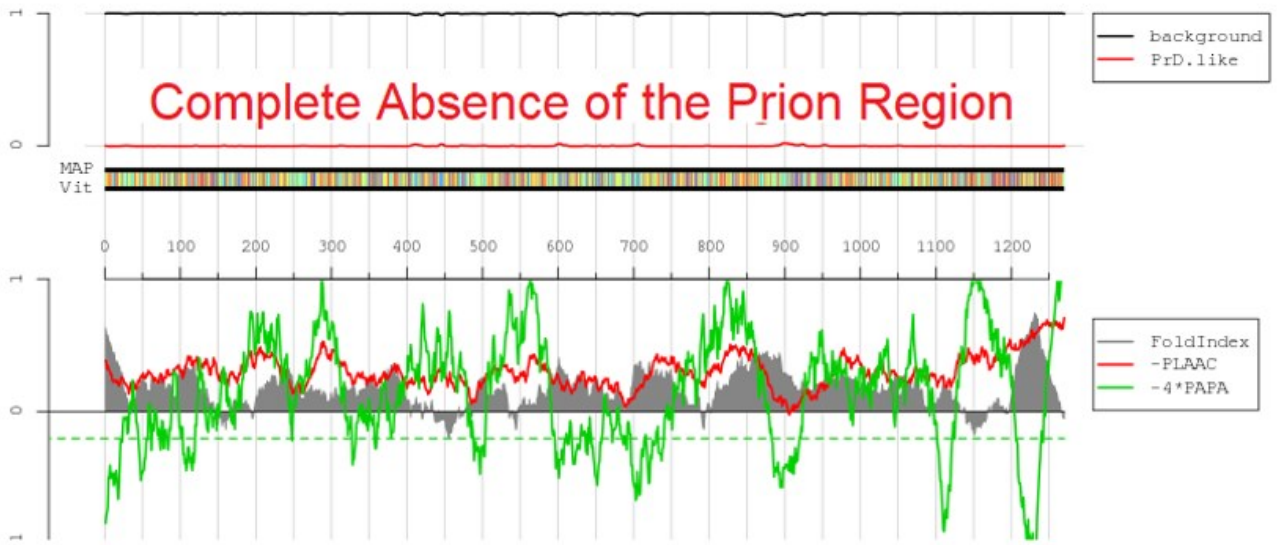


Figure 31. Here is the PLAAC analysis of the last USA Omicron patient, sequence ID OM084409.1 dated December 12, 2021, a Texas resident, showing that the prion region has totally disappeared from this strain of the virus. For that to happen, the mutations required must be 8 times greater than in the entire rest of the spike protein.

**Table 6**
**PLAAC Analysis of Seven Omicron Cases from Various US States Showing the Prion Region Has Totally Disappeared from *All* of Them**

| Ref | Identification Omicron USA patient strain | Prion region |
|---|---|---|
| SUSA1 | Sequence ID: **OM084744.1** **USA/KY** | none |
| SUSA2 | Sequence ID: **OM084702.1** **USA/KY** | none |
| SUSA3 | Sequence ID: **OM084601.1** **USA/TN** | none |
| SUSA4 | Sequence ID: **OM084601.1** **USA/TN** | none |
| SUSA5 | Sequence ID: **OM084538.1** **USA/KY** | none |
| SUSA6 | Sequence ID: **OM084529.1** **USA/IN** | none |
| SUSA7 | Sequence ID: **OM084430.1** **USA/OH** | none |
| SUSA8 | Sequence ID: **OM084409.1** **USA/TX** | none |
| **Results** | | **None** |

**Table 7**
**Prion region in various SARS-CoV2 Variants and Vaccines**

| **Identification of main SARS-CoV-2 variants** | **PRION region amino acids 473-510 detected by PLAAC** | **PRION region amino acids 473-510 not detected by PLAAC** |
|---|---|---|
| SARS-CoV-2 Wuhan (D614G) | YES | |
| ALPHA (UK) | YES | |
| BETA (South Africa) | YES | |
| GAMMA (Brazil) | YES | |
| DELTA (India) | YES | |
| **OMICRON (South Africa)** | | **YES** |
| **Identification of SARS-CoV-2** | **PRION region** | **PRION region** |
| **mRNA vaccine Pfizer** | **YES** | |
| **mRNA vaccine Moderna** | **YES** | |
| **Astra Zeneca vaccine** | **YES** | |
| **Janssen vaccine** | **YES** | |

### MEANING OF THE W OR M STRUCTURES OF THE PRION MASTER CODE IMAGES

We observed that all the prions had Master Code image patterns in the shape of either a "W" or an "M". Also, the prion regions detected by PLAAC corresponded to descending parts of these images. Several years ago, we had the idea of imagining a kind of hypothetical gene which would be formed by the sequence of the 64 codons of the universal genetic code. What then would have been the genomic/proteomic signature in the Master Code? It would be the one shown in Figure 7>>. Curiously, we note that it too has an "M" shape.

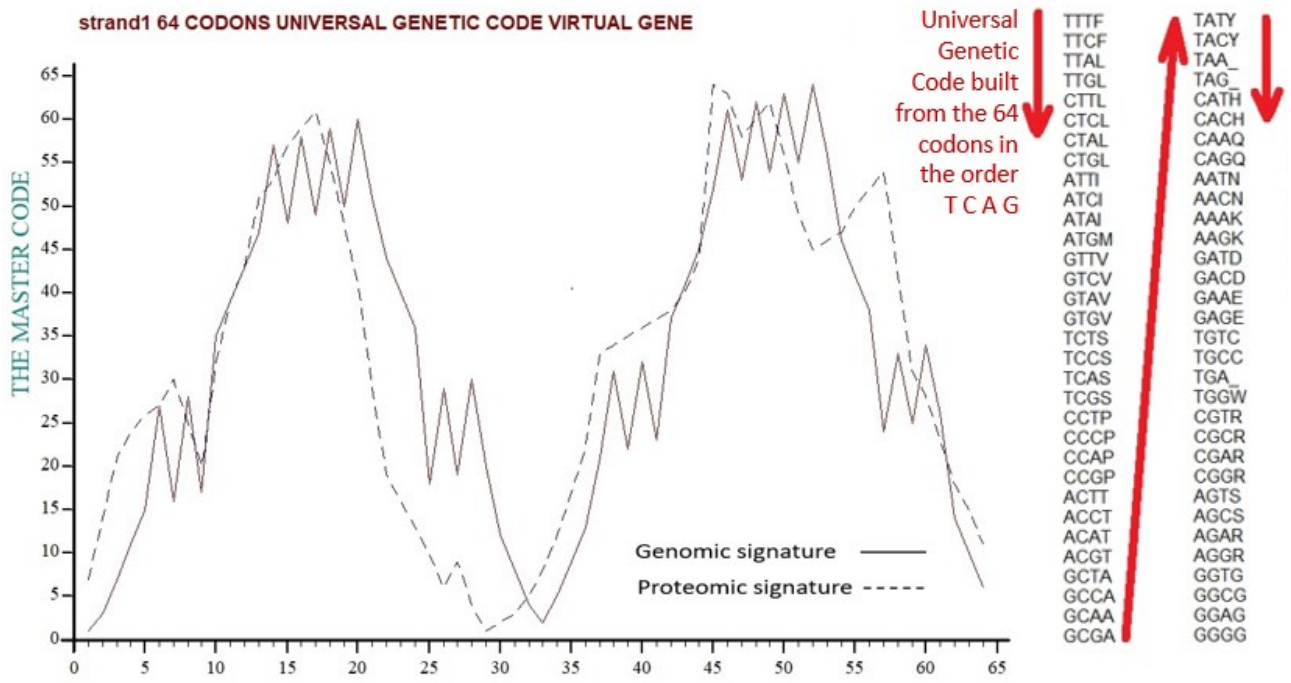


Figure 32. The "M" shape running throughout the Master Code of a synthetic gene built from the Universal Genetic Code of 64 codons in the order T C A G.

In the Table of the Genetic Code (at the viewer's right in Figure 5), the codons are classified according to the regular order TCAG. We also observe (at the left side of Figure 5) that it is the second base of the codon triplets that dictates the meta structure of the Master Code image following the TCAG meta-order. Consequently, the descending regions of both "M" patterns are rich in C and G bases. Therefore, the prion regions detected by PLAAC are ones in which the increasing CG richness in the double strand of DNA, produces its regular "descending" shape. Finally, let us note that the mRNA vaccines of Pfizer and Moderna were doped with CG bases without modifying the corresponding amino acids (using the vagueness allowed by the Genetic Code). So, although their prion region remains identical to that of the initial Wuhan Spike strain at the amino acid level, one can think that this CG base doping could amplify the prion effect of vaccines if some energy dynamic, for example, from electromagnetic sources as recently discussed by various researchers examining the COVID-19 injectables (Sarlangue, et al. 2021), were introduced during the translation of mRNA into amino acids.

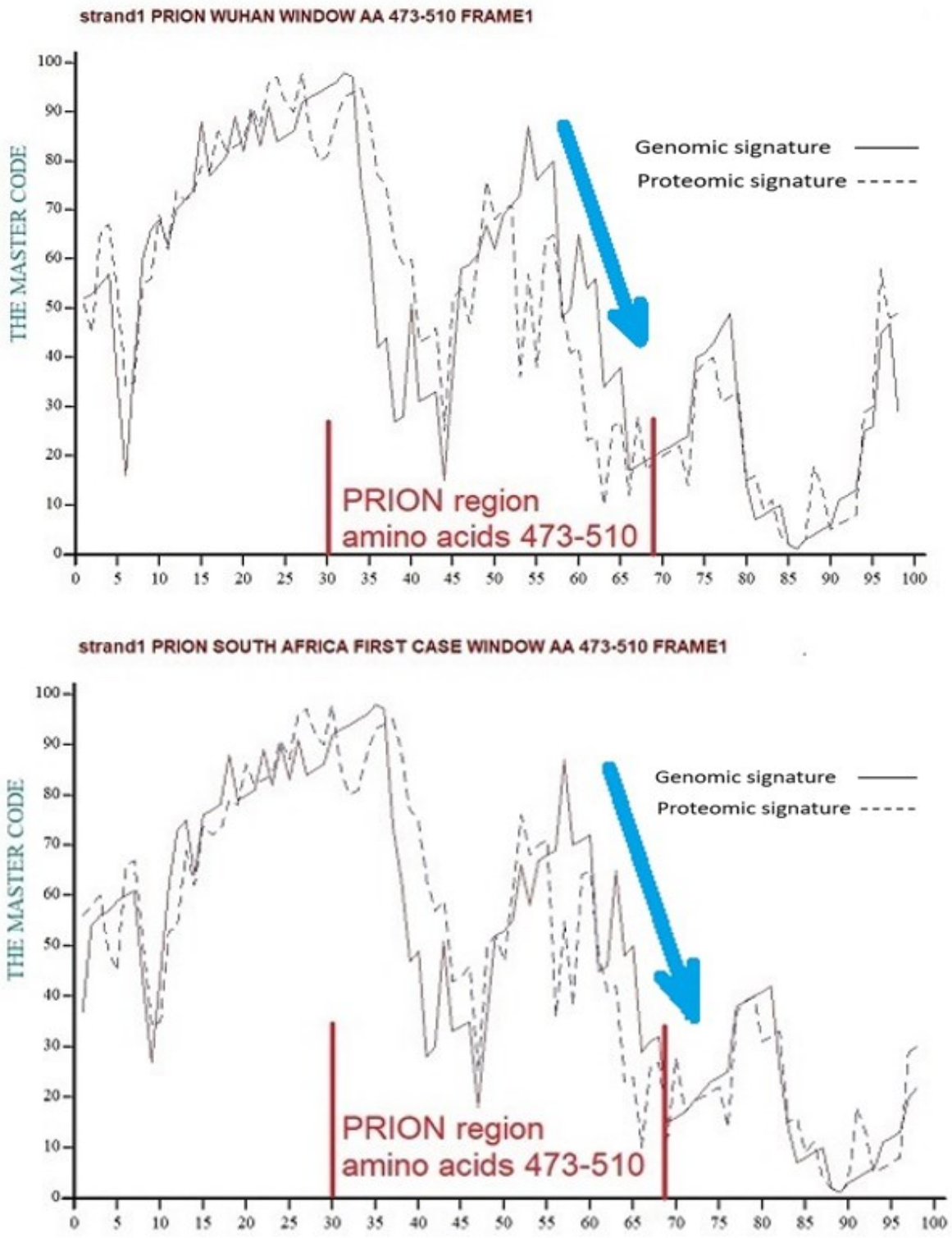


Figure 33. Comparing Master Code pattern genomic/proteomic signatures between spike prion regions in the Wuhan SARS-CoV-2 and Omicron viruses and finding no major differences.


https://doi.org/10.56098/ijvtpr.v3i1.66

In Figure 33, although the Master Code images of the respective prion regions of SARS-CoV-2 Wuhan and Omicron appear very similar, we note however that the transition of this region from Wuhan to Omicron results from the 8 amino acid mutations of this prion region leading to an improvement of more than 2% of the genomic/proteomic coupling, from 88.45% ==> 90.63%. We interpret this to be a better adaptation of the Omicron virus to its human host. It is interesting to discuss the relevance and consistency of this prion region by highlighting it in the spikes of all pre-Omicron variants as well as in the spikes of all COVID-19 vaccines. The weak point in such a suggestive qualitative comparison is that the results remain qualitative. The comparison would be more conclusive if there were a quantitative basis to assess all of the relevant cases. Given that the PLAAC amplitude of the prion region of SARS-CoV-2 remains low compared to the human prion PRNP, if such a contrast existed for the Wuhan and Omicron cases, it would constitute a kind of empirical proof by inhibition or negation: indeed, we demonstrate how and by which mutations this prion region could disappear... and, indeed, how it disappeared from *all* the Omicron variants analyzed in this paper. In doing so, our empirical proof, becomes very strong: it is analogous to using the shadow of an object to prove the existence of light. Sad to say, the actual cases of the rapidly developing new form of Creutzfeldt-Jakob Disease appearing in some people soon after the injection of a COVID-19 vaccine — cases that we have presentd in the main body of this paper — seems to show that the hypothetical causal prion function that we believe we have detected does indeed exist.

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*Chers amis de la Fondation, Madame, Monsieur,*

Voilà déjà presque un an que le Professeur Luc Montagnier nous a quittés.

La Fondation Luc Montagnier, les amis, les collaborateurs du Professeur, ont décidé de lui rendre l'hommage dû à l'un des grands scientifiques de tous les temps, Prix Nobel de médecine et de physiologie pour la découverte du plus grand fléau de notre époque.

Cet hommage prendra la forme d'une conférence animée par différents intervenants issus du monde scientifique, médical et artistique qui se tiendra le Vendredi 17 février 2023 de 17h à 20h à Paris dans l'amphithéâtre du 32 l'avenue Hoche.

Cette conférence mettra en avant d'une part la poursuite des travaux du Professeur Montagnier, mais aussi les dernières découvertes et des sujets d'actualité. Elle nous permettra de lui rendre un hommage plus large, aussi poignant et émouvant que lors de ses obsèques. Ce sera l'occasion de rappeler tout ce qu'il a apporté à la communauté scientifique et médicale.

Nous espérons que vous serez des nôtres pour saluer avec nous la mémoire d'un homme qui a dédié sa vie à la recherche avec toujours un temps d'avance sur son temps.

Nous comptons sur votre présence et vous remercions par avance.

Toute l'équipe de la fondation



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