

Are Covid19 mRNA Injections the Cause of Turbo Cancers due to Prion Behavior of P53 Tumor Suppressor?

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ABSTRACT.

After an exploration of the state of the art concerning the possible interactions between gene P53, zinc fingers, spike protein of viruses and sarscov2 Covid19 injections, and Prions, we strengthen this thesis by demonstrating, through the theory of the Master Code of Biology, why P53 hot spots are so mutagenic and why these regions have a high potential for Prion-like behavior.

INTRODUCTION:

We demonstrated in [1] (Perez, Moret Chalmin, Montagnier, 2023), then, in [2] (Perez, 2024), how the Prion spike region of the sarscov2 virus and mRNA injections had generated dozens of fatal cases of a new form of CreutzfeldtJakob.

Scientists have also observed the statistical link between vaccines and dementia or Alzheimer's [3] (Roh JH et al, 2024).

See also psychiatric diseases following covid jab in korea in [4] (Kim H. J. et al, 2024).

Could a Prion-like behavior of the P51 tumor suppressor also contribute to the observed emergence of hyper-rapid cancers following Covid injections?

Are there links between mRNA Covid19 jabs and Cancer?

In [5] (Khatri JK et al, 2024), authors shows a plausible association of mRNA vaccine and lymphomatous processes, especially AITL. With having TFH cell signature of AITL and the role of mRNA vaccine to induce robust TFH response and germinal center proliferation.

One of the main anti-cancer protective genes is P53.

The link between p53 and its hypothetical Prion function is contested in [6] (Billant et al, 2021).

On the contrary, we have gathered below various elements which would reinforce this hypothesis of a possible Prion function of p53. We will also study the role of zinc in p53 and the possible effect of the Sarscov2 Spike protein and its mRNA injections.

Mortality and Covid jabs:

In 2021 and 2022, in all countries that have imposed MRNA Covid vaccines, we observe an increase in mortality, including for young age groups. In their article, [7] (Mostert et al, 2024) entitled "Excess mortality across countries in the Western World since the COVID-19 pandemic: 'Our World in Data' estimates of January 2020 to December 2022", the authors demonstrate in particular "The total number of excess deaths in 47 countries of the Western World was 3,098,456 from 1 January 2020 until 31 December 2022. Excess mortality was documented in 41 countries (87%) in 2020, 42 countries (89 %) in 2021 and 43 countries (91%) in 2022."

In trialsite June 2024, Dr Mathias Hoben (<https://trialsitenews.com/p/trialsitenews/investigation-into-death-rates-among-alberta-assisted-living-and-nursing-home-residents-late-2021-death-surge-occurs-despite-pervasive-vaccination-6bbf0891>) analyses mortality in Alberta, Canada, during the first 2 years of the COVID-19 pandemic, relative to the 3 years before. The results were just published in the *Journal of the American Medical Directors*. [8] (Hogan D. et al, 2024). Excess Deaths in Assisted Living and Nursing Homes during the COVID-19 Pandemic in Alberta, CanaThis strong analysis mix the Covid virus period (2020) and the mandatory vaccine period (221)"Turbo Cancers":

"Turbo Cancers":

The main side effects of these fake vaccines, sometimes fatal, are most often of a cardiological and neurological nature [1] (Perez et al. 2023) but we also observe many cancers with abnormally rapid emergence and then fatal outcome, also some named them "Turbocancer".

P53 the Tumor Suppressor gene:

P53 [9] (Olivier M, Hollstein M, Hainaut P, 2010), °is involved by mutations in about one cancer case by two.

As such, p53 has been described as "the guardian of the genome" because of its role in conserving stability by preventing genome mutation.

Covid19 jabs, SV40 promoter and P53:

SV40 promoters are there in billions or copies per MRNA Covid19 injection dose. My friend Dr Kevin McCairn in Japan says that because SV40 was discovered in Covid mRNA vaccines, "SV40 role in oncogenesis still needs more study and with most cancers you need multiple hits.

Low white blood cells, spike interaction with P53 and perhaps SV40 is a combo we have to take seriously given cancer rise Seen."

See also in (<https://www.floridahealth.gov/newsroom/2024/01/20240103-halt-use-covid19-mrna-vaccines.pr.html>) Florida State Surgeon General Calls for Halt in the Use of COVID-19 mRNA Vaccines Contact: Communications Office NewsMedia@flhealth.gov, 850-245-4111 Tallahassee, Fla.— On December 6, 2023, State Surgeon General Dr. Joseph A. Ladapo sent a letter to the United States Food and Drug Administration (FDA) Commissioner Dr. Robert M. Califf and Center for Disease Control and Prevention (CDC) Director Dr. Mandy Cohen regarding questions pertaining to the safety assessments and the discovery of billions of DNA fragments per dose of the Pfizer and Moderna COVID-19 mRNA vaccines. The Surgeon General outlined concerns regarding nucleic acid contaminants in the approved Pfizer and Moderna COVID-19 mRNA vaccines, particularly in the presence of lipid nanoparticle complexes, and Simian Virus 40 (SV40) promoter/enhancer DNA. Lipid nanoparticles are an efficient vehicle for delivery of the mRNA in the COVID-19 vaccines into human cells and may therefore be an equally efficient vehicle for delivering contaminant DNA into human cells. The presence of SV40 promoter/ DNA may also pose a unique and heightened risk of DNA integration into human cells.

P53 and Zinc fingers:

P53 is characterized by the presence of a zinc atom, see Figure 1.

The large percentage of transcription factors require zinc to bind DNA. p53 is unique among zinc-dependent transcription factors. The conformation of p53 is unusually malleable: p53 binds zinc extremely tightly when folded, but is intrinsically unstable in the absence of zinc. Whether the wild-type protein folds in the cell is largely determined by the concentration of available zinc atoms. Consequently, zinc dysregulation in the cell as well as a large percentage of tumorigenic p53 mutations can cause p53 to lose zinc, misfold, and forfeit its tumor suppressing activity. See particularly [10] (Ha J. H., 2022).

Sarscov2 and zinc:

In [11] (Rheingold S Z, 2023) "Zinc Supplementation Associated With a Decrease in Mortality in COVID-19 Patients: A Meta-analysis", the author shows how a zinc supplementation increase spike effect impact then mortality. "We must recall that this sentence remain also in the case of spike from MRNA Jabs. See also [Metallomics](#). 2022; 14(7): mfac047. Published online 2022 Jun 29. doi: [10.1093/mtomcs/mfac047](https://doi.org/10.1093/mtomcs/mfac047) PMID: PMC9314716 PMID: [35767875](https://pubmed.ncbi.nlm.nih.gov/35767875/).

The zinc proteome of SARS-CoV-2:

[Claudia Andreini](#), et al shows by Structural biology studies on severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) revealing that zinc(II) is the most common metal ion that binds sarscov2 proteins. Because SARS-CoV-2 spike protein enhances ACE2 activity. SARS-CoV-2 use ACE2 as its entry receptor, in doi: [10.1016/j.peptides.2020.170477](https://doi.org/10.1016/j.peptides.2020.170477)

Y Pollack et al show interactions between ACE2 and zinc, particularly they conclude that even though ACE2 is a zinc metalloproteinase, more zinc does not necessarily mean

higher enzyme activity. Details in [12] (Pollack Y, 2021).

Spike and p53:

Various authors show that SARS-CoV-2 Spike Protein Disrupts p53 Tumor Suppressor Pathway and light on the intricate interplay between the SARS-CoV-2 spike protein and the tumor suppressor p53, a key guardian of genomic integrity.

An interesting study we came across refers to Mdm2, which apparently plays a role in restraining and regulating p53 keeping its activities under control [13] (Lee, J, 2010).

Why this seems important is the paper by Professor El-Deiry, one of the leading expert of p53 functionality, who found that the Spike protein appears to impact Mdm2 function [14] (Shengliang Zhang et al, 2024a) and in [15] (Shengliang Zhang et al, 2024b) Zhang S. shows that SARS-CoV-2 spike protein mediates host cell infection and cell-cell fusion that causes stabilization of tumor suppressor p53 protein. In-silico analysis previously suggested that SARS-CoV-2 spike interacts with p53 directly.

Finally, this reported state of the art demonstrates strong links and interactions between the 4 topics:

- Spike from sarscov2 virus and mRNA vaccines.

- Zinc amount available in the host organism.

- P53 Tumor Suppressor Gene function.

- Prion function.

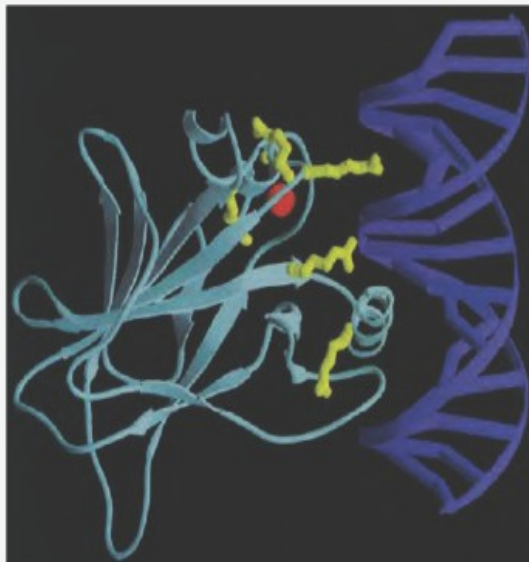


Figure 2: picture: p53 interacting with DNA. Method: X-ray diffraction. [Credits: See Cho Y et al. for details!]

Figure 1 – TP53 interaction with DNA molecule

METHODS

PLAAC Prion potential analysis.

For the assessment of prion-like characteristics in the p53 proteins, we will employ the Prion-Like Amino Acid Composition (PLAAC) tool, accessible at <https://plaac.wi.mit.edu/> [16] (Lancaster A. K., 2014). This analysis will help determine if the unintended proteins possess prion-like properties, which could have significant implications.

In Figure 2 we show the colors sorted potential prion function of the 20 amino acids.

<= high. Low =>
NQYGM SPAHT FRVID LKCWE

N and Q are the higher potential Prion amino acids. Red ones are the lower potential Prion function amino acids.

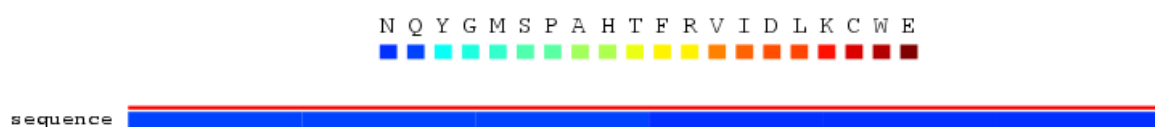


Figure 2. Hierarchy of the potential Prion function amino acids.

MASTER CODE ANALYSIS

The Master Code is a consequence of the basic research discovery "Formula for Life" related principally in the book CODEX BIOGENESIS [17] (Pérez JC, 2009), and in the main article "six fractal codes of Life" prefaced by my friend RIP Nobelprizewinner pr Luc Montagnier [18] (Perez, 2021).

The discovery of a simple numerical formula for the projection of all the atomic mass of m0-sustaining CONHSP bioatoms leads to the emergence of a set of Nested CODES unifying all the biological, genetic and genomic components by unifying them from bioatoms up to 'to whole genomes. In particular, we demonstrate the existence of a digital meta-code common to the three languages of biology that are RNA, DNA and amino acid sequences. Through this meta-code, genomic and proteomic images appear almost analogous and correlated. We called this the MASTER CODE of Biology.

Finally 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.

Figures 3, 4 and 5 illustrate the Genomics/Proteomics Master Code unification.

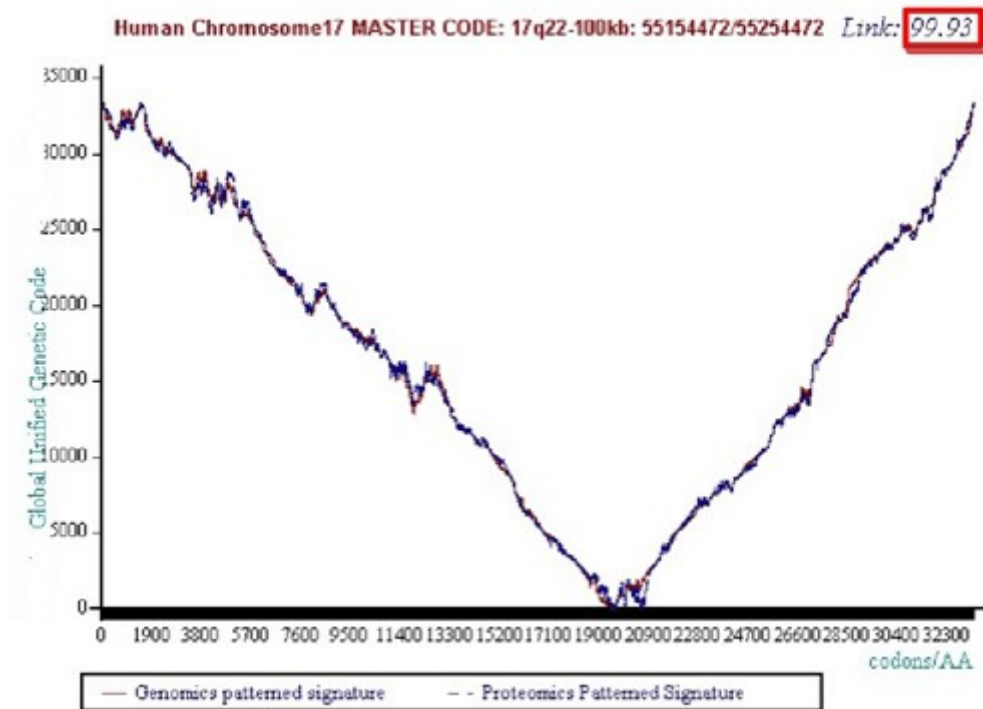


Figure3 shows 99.93% Master Code unification in the case of 100000 bases pairs in human chromosome 17.

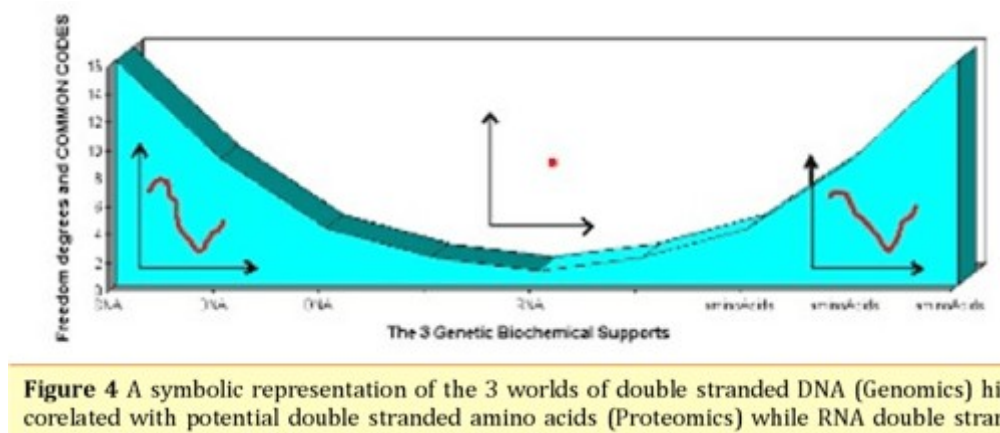


Figure4 shows Theoretical equivalence at double stranded level between Genomics and proteomics images and the neutral effect of RNA world (like a Zero of Biology).

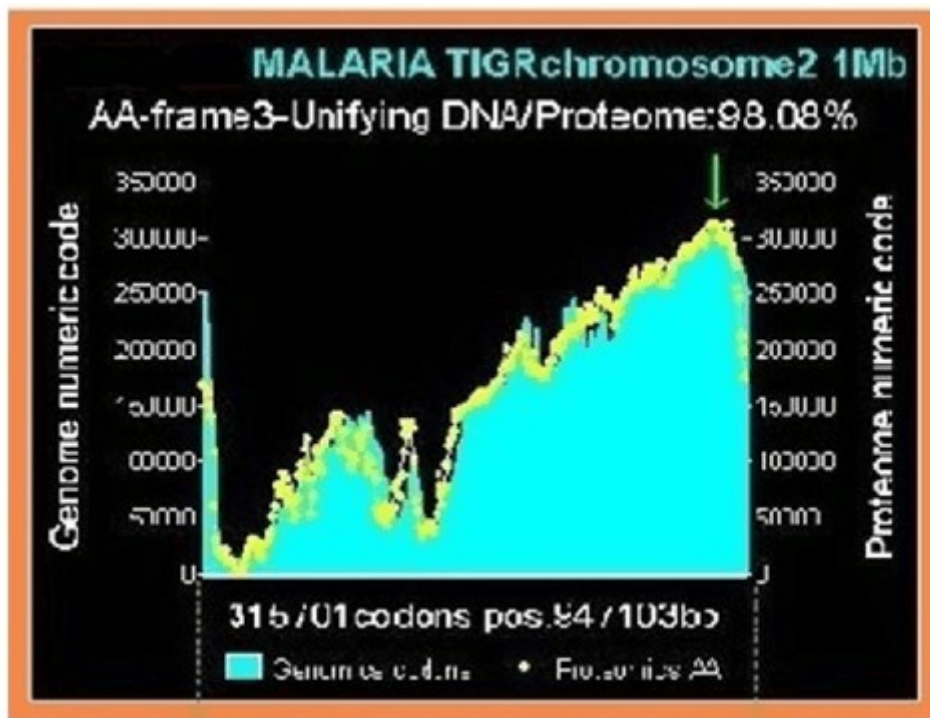


Figure 6 illustration of the high correlation coupling between genomics and proteomics

Figure5 shows the Master Code for whole Malaria chromosome.

RESULTS and DISCUSSION:

Could a Prion-like behavior of the P51 tumor suppressor also contribute to the observed emergence of hyper-rapid cancers following Covid injections?

PREDICTING WITH THE "MASTER CODE" FULL CARTOGRAPHY OF P53 MUTAGENES HOTSPOTS:

In this article published in 2018, [19] (Perez Jc, 2018), we summarize: Mutations in the TP53 gene are encountered in about one in every two cases of cancer. The locations and frequencies of the mutations are well known and listed. It is therefore on these mutations of TP53 that we validate here a theoretical method of prediction of the mutagenic regions of TP53. This method uses the Master Code of Biology, revealing a coupling and unification between the Genomics and Proteomics codes for any DNA sequence analyzed. The "score" of these couplings highlights the functional regions of genes, proteins, chromosomes and genomes. Of the 393 codons of TP53, and for the 61 possible values of these codons authorized by the genetic code (i.e. 393x61 genes simulated), we prioritize the corresponding Master Code scores. Codons with scores close to 1 correspond to conserved regions whereas codons with scores close to 61 reveal highly mutagenic regions. Our method is then validated and correlated with the real mutations observed experimentally on hundreds of cases.

Let us return to the strong potential of this method, here applied to P53, but universal, because it can be generalized to the prediction of mutagenic regions of any protein.

Let's think about this analogy.

There will inevitably be a distortion between these 2 messages. What the Master Code measures is the quality of this translation measured not at the local level of each word but on the global scale of the message in its entirety.

Gen Lights"- A Method for Mapping Regions of Mutability of a Gene. Our Master Code basic research output could predict the Mutability level of each codon value and position relating to its effect at the whole structure level of the Genomics/Proteomics Unification data. Then, we could associate with each codon position a "Mutability Coefficient" varying from 1 to 61: i. 1 if the codon is the best by 61 possible values (without STOP codons values). ii. 61 if any codon change increases the global Genomics/ Proteomics coupling ratio of the whole gene. We note that low coefficient region correspond with optimal and "CONSERVED" regions. Contrarily, high coefficient regions correspond with high MUTABILITY experimentally observed regions. Then to summarize, in the case of gene = p53 long of 393 amino acids, For each codon position « i » from i = 1 to 393, DO: build 61 pseudo sequences where gene (i) = each possible coding codon Compute scores = 61 Master Code methods (gene (i)) Compute score (real codon i) = score of the real codon i in the hierarchy of 61 scores (i). Final result is an array of 393 scores with values between 1 and 61. Low values codon scores near 1 reveals conserved optimal codons related to a good Master Code coupling ratio. Contrarily, high values codon scores near 61 reveals bad Master Code coupling ratio, then probably a high potential mutagen codon location (Figures 6, 7, 8 and 9).

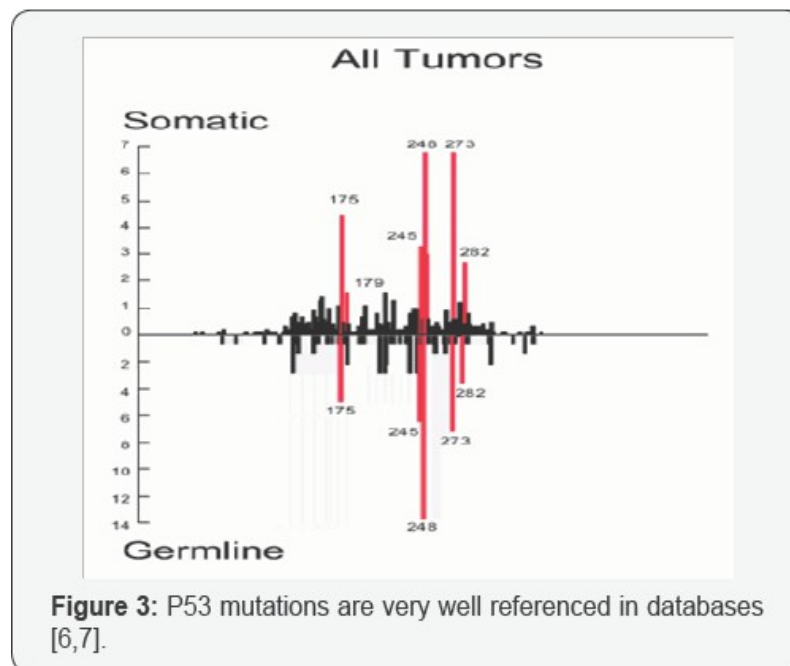


Figure6 In red the main TP53 Hotspots

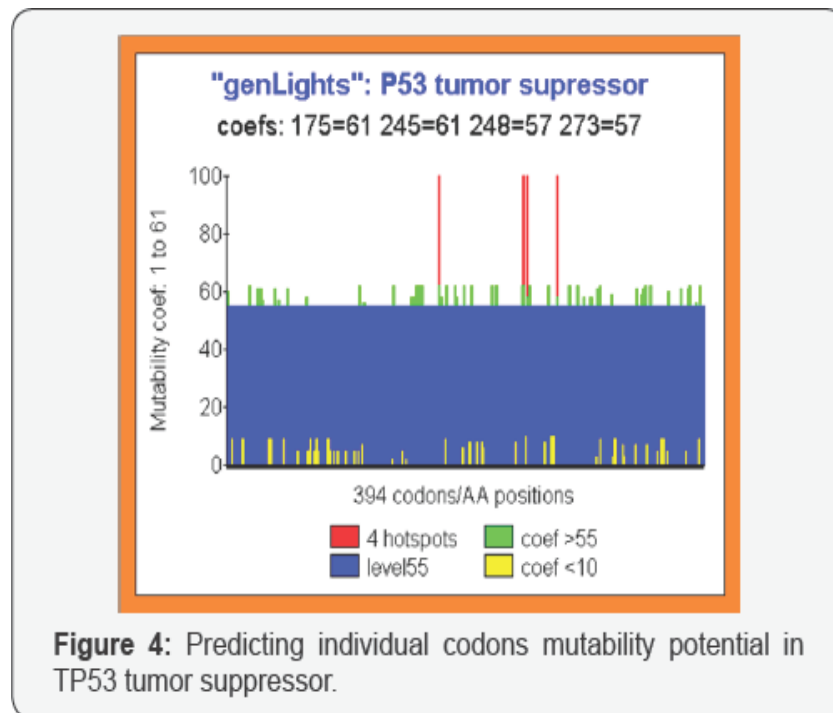


Figure7 Evidence of mutagene codons (green) are correlated with hotspots (red)

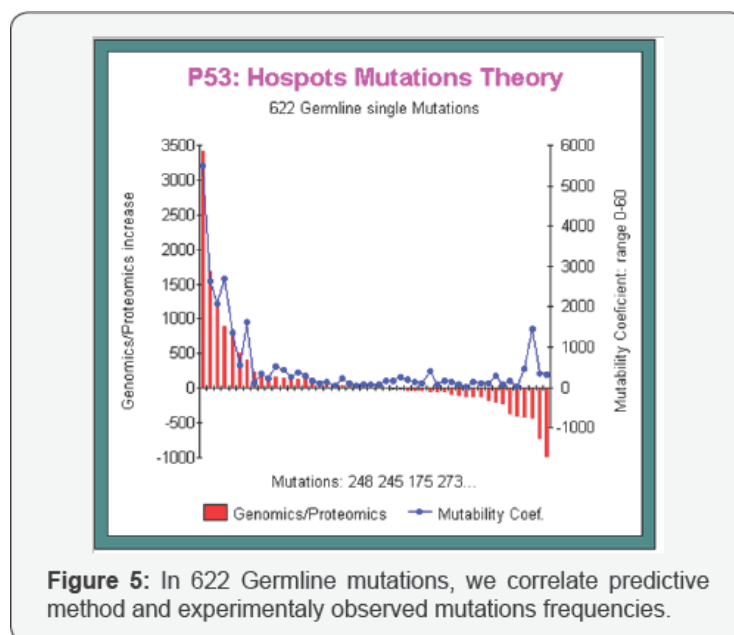


Figure8 622 germline mutations

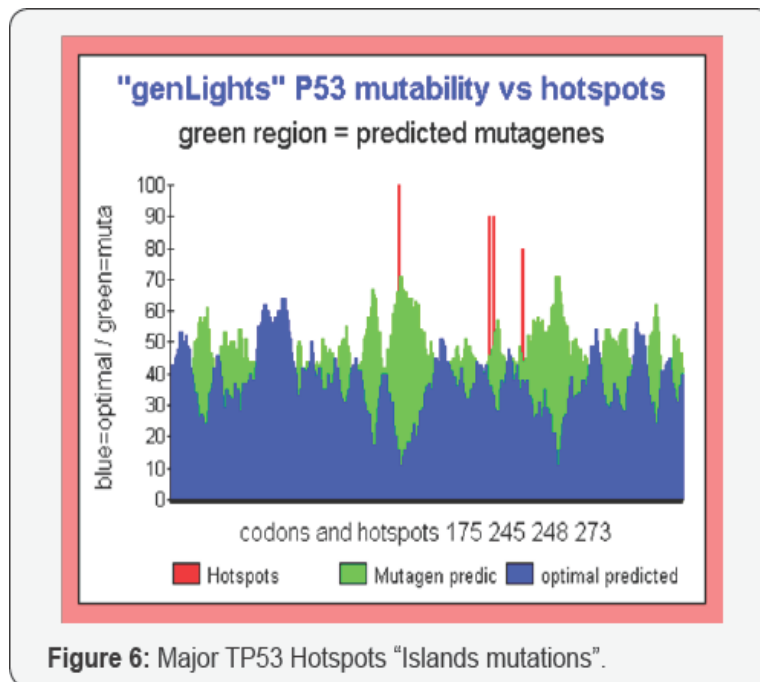


Figure9 evidence of mutagene areas (green)

The main mutations have been found to be highly frequent at the four mutational "hotspots" at codons 175, 245, 248 and 273. (Figure6)

Then, what about the Prediction of these Mutations by our theoretical predictive method? In the Figure 7, we show a typical output: All codons positions are affected by a Mutability Coefficient in the range of 1 to 61: 1 signify that this real codon value is the optimal one possible. 61 signify that any mutation on this position increases the global Genomics/Proteomics organization of the gene. This kind of codons has then a high Mutability power. In the figure, we show (red) the four Hotspots codons positions 174 245 248 273. Green bars illustrate high mutability codons (coef >55). Contrarily yellow bars illustrate high conserved and optimal codons.

Then, the prediction mutability score coefficient of the 4 Hotspots is Perfect:

- i. Hotspot 175: coef=61 (the higher mutability possible level).
- ii. Hotspot 245: coef=61 (the higher mutability possible level).
- iii. Hotspot 248: coef=57 (very high mutability coef ie range 1-61).
- iv. Hotspot 273: coef=57 (very high mutability coef ie ange 1-61) (Figure 8).

In this other simulation run (Figure 9), we analyse 622 GERMLINE single mutations. The correlation between Experience and Prediction is Perfect: Horizontally, we represent all mutations sorted by decrease frequency values: on the left, high frequency mutations (codons 248 245 175 273...). On the right, rare mutations points. Simultaneously, the red bars represent the mutation effect on Genomics/Proteomics: on the left, this ratio increases, then on the right, this ratio decreases! In blue, we plot the evolution of the mutability coef: high for frequent mutations, low or flat for others. Now, we have a good proof of the Prediction power of our Master Code based theoretical predictive mutagenesis method improved on the best example on Mutability: P53, the « *King Cancers gene* » (Figure 9).

These results are remarkable. For what? What does a score of 61 mean here for the 2 HOTSPOTS 175 and 245? This means that the worldwide reference gene has the worst possible master code while any other mutation on these codons would always produce a better master code. These 2 amino acids are therefore potentially very mutagenic.

And what happens to the 3 restarted codons (62 63 64)? They would correspond to the 3 STOP codons. They would therefore produce a truncated P53 protein and therefore non-functional. For the other 2 HOTSPOTS with a score of 57, codons 248 and 273, this means that they are also very strongly mutagenic, a score of 57 meaning that 56 mutations produce a better master code while only the remaining 4 produce a worse master code.

COULD P53 RUN LIKE A PRION?

The question we will ask ourselves here will be: "could the P53 hot spots, whose extreme mutability we have just predicted, behave like Prions during certain of these mutations?"

The one letter amino acids sequence of P53 is as follows: TP53 Tumor Suppressor gene.

https://www.ncbi.nlm.nih.gov/protein/NP_001394193.1?report=fasta cellular tumor antigen p53 isoform a [Homo sapiens] NCBI Reference Sequence: NP_001394193.1

[GenPept Identical Proteins Graphics](#) >NP_001394193.1 cellular tumor antigen p53 isoform a [Homo sapiens]

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MEEPQSDPSVEPPLSQETFSDLWKLLPENNVLSPLPSQAMDDLMLSPDDIEQWFTEDPG
PDEAPRMPEAAAPPVAPAPAAPTPAAPAPAPSWPLSSSVPSQKTYQGSYGFRGLGFLHSGT
AKSVTCTYSPALNKMFCQLAKTCPVQLWVDSTPPPGTRVRAMAIYKQSQHMTEVVRRCP
HHERCSDSDGLAPPQHILIRVEGNLRVEYLDDRNTFRHSVVVPYEPPEVGSDCTTIHYN
MCNSSCMGGMNRRPILTIITLEDSSGNLLGRNSFEVRVCACPGRDRRTEENLRKKGEP
HHELPPGSTKRALPNNTSSSPQPKKKPLDGEYFTLQIRGRERFEMFRELNEALELKDAQA
GKEPGGSRAHSSHLSKSKKGQSTSRRHKLMFKTEGPDS
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1 meepqsdpsv epplsqetfs dlwkllpenn vlsplpsqam ddlmlspddi eqwftedpgp
61 deaprmpeaa ppvapapaap tpaapapaps wplsssvpsq ktyqgsygfr lgflhsgtak
121 svtctyspal nkmfcqlakt cpvqlwvdst pppgtrvram aiykqsqhmt evvrrcphhe
181 rcsdsdglap pqhlirvegn lrveylddrn tfrhsvvpy eppevgdct tihynymcns
241 scmnggmnrp iltitleds sgnllgrnsf evrvcacpgr drrteenlr kkgephhelp
301 pgstkralpn ntssspqpkk kpldgeyftl qirgrerfem frelnealel kdaqagkepg
```

361 gsrahsshlk skkgqstsrh kklmfktegp dsd //

We consider now the 20 amino acids Prion function hierarchy classification. Prion hierarchy

+. -

NQYGMSPAHTFRVIDLKCWE

See also colors codes in Figure 10.

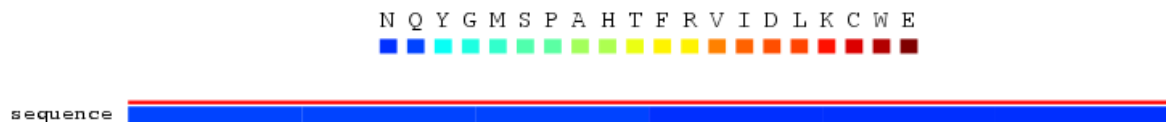


Figure 10 Hierarchy of Prion potential for the 20 amino acids.

Hot blue green colors in the left (NQ etc...) are Prion facility.

Cold red colors in the right (WE) are not Prion facility.

Prion hierarchy

+. -

NQYGMSPAHTFRVIDLKCWE

In Figure11 we analyse the PLAAC Prion function of the whole 393 amino acids of TP53 wild type.

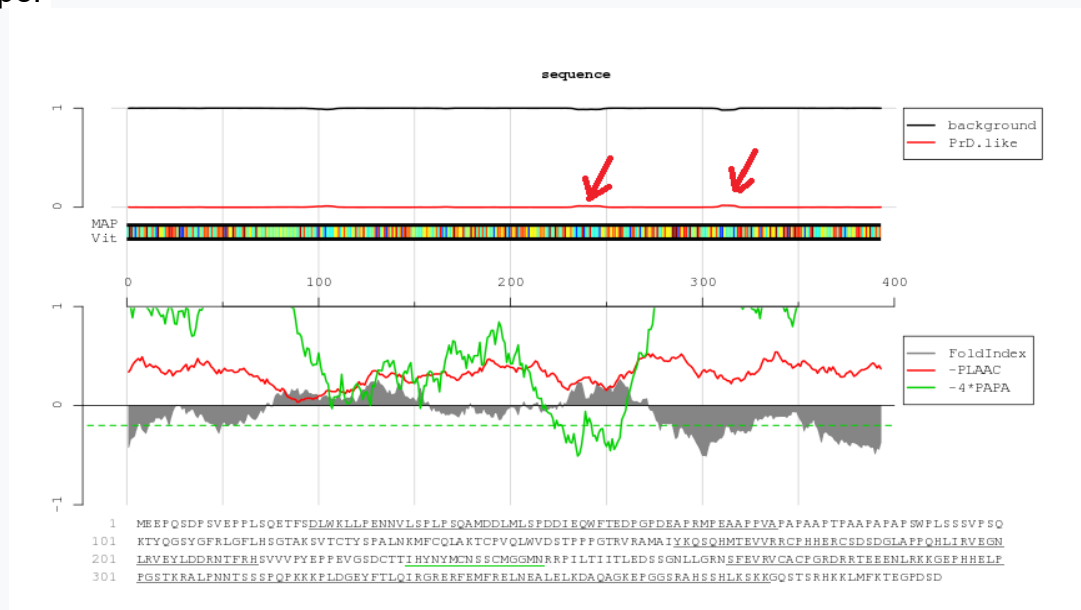


Figure11 shows particularly an hot potentially Prion facility in 230-250 area with high level of blue green.

This region contains the 2 major HOTSPOTS codons 245 and 248. hat significates that mutations in these regions could swap from wild type P53 to a potential change in a possible Prion status. To explore this scenario we analyse now a set of real world mutations involved in leukemia patients. In [20] (Hou HA, 2015) Hou, HA., Chou, WC., Kuo, YY. et al. TP53 mutations in de novo acute myeloid leukemia patients: longitudinal follow-ups show the mutation is stable during disease evolution. Blood Cancer Journal 5, e331 (2015). <https://doi.org/10.1038/bcj.2015.59> <https://www.nature.com/articles/bcj201559> Hou et al analysed a total of 36 different TP53 mutations identified in 35 patients (Figure12).

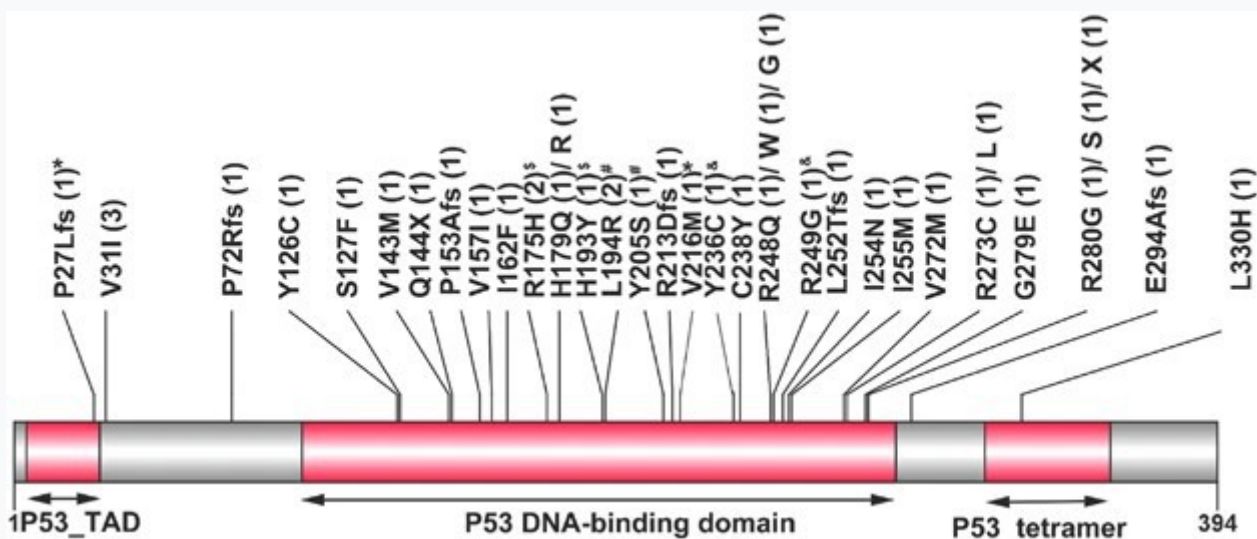


Figure 12 mutations in the P53 DNA binding domain

We analyse now the case of P53 DNA binding domain.

Our goal was to find if these mutations are neutral at Prion facility level or contrary if there increase the Prion risk.

For this purpose we compute for each individual mutation the increase of decrease number of locations within the 20 amino acids Prion scale as follows.

Prion scale hierarchy

+ -
NQYGMSPAHTFRVIDLKCWE

Y126C. -15
 S127F. - 5
 V143M. +8
 P153A. - 1
 V157I. - 1
 I162F. +3
 R175H. +3. 2 times
 H179Q. +7
 H179R. - 3
 H193Y. +6
 L194R. +4. 2 times
 Y205S. - 3
 R213D. - 3
 V216M. +8
 Y236C. - 15
 C238Y. +15
 R248Q. +10
 R248W. - 7
 T248G. +6
 R249G. +8
 L252T. +6
 I254N. +13
 I255M. +9
 V272M. +8
 R273C. - 6
 R273L. - 4
 G279E. - 16

12times- total - 79
 17times+. Total +121

Finally, mutations decrease Prion potential in 12 cases but increase Prion potential in 17 cases. If we cumulated now the distances within the Prion scale hierarchy the result is very interesting: The cumulated Prion increase distances (121) is 53% greater than the Prion decrease distances (79). Then globally real world mutations observed INCREASE the capability of a TP53 prion function in the case of mutations.

CONCLUSIONS:

We have just, by 2 radically independent methods, on the one hand, predicted the mutagenic regions of TP53, and, on the other hand, predicted the regions likely to evolve towards Prion behavior in the event of mutations.

Now let's compare in figure 9 (master code) the very green and therefore mutagenic regions. In addition to the HOTSPOTS regions we see a maximum site around codons 300.

Let's now compare these same regions 300 in Figure 11 (PLAAC prions).

We then discover that this region is predominantly blue green, therefore susceptible to Prion function.

We therefore have here a convergence, via 2 independent methods, of the potential of TP54 to take prion conformations.

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Ronald Reece is a former Intelligence Analyst who served as a contract with the Iraq Survey Group (ISG) between 2004 until 2005. During the past 4 years he has become a Subject Matter Expert on the use of Early Treatment options for RNA viruses, relying upon the suppressed research of Dr. Ralph Baric in 2010 using Zinc + Zinc Ionophores to inhibit the replication of RNA viruses. This research culminated in the creation of the Zelenko Protocol (Zinc + HCQ) for Early treatment of the SARS-2 virus in March, 2020.

Mr. Reece also serves as the Director of Technical Analysis, Special Projects, for the Near East Center for Strategic Engagement (NEC-SE), an independent think-tank with the goal of providing accurate, and timely analysis to political and military policy makers to enhance strategic cooperation around the globe.

Mr. Reece is also the co-author with Sami Mackenzie of PROTOCOL-Z, currently in the final pre-publication stage, that details the story of the Zelenko Protocol, and the role Dr. Ralph Baric played in its creation as an Early treatment for SARS-2. It also argues its likely effectiveness against all RNA viruses. It reveals the critical role that Zinc plays in fortifying our immunological response to not only RNA viruses, but also cancer, and many other maladies.

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