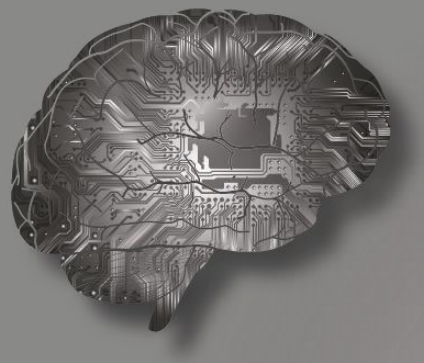

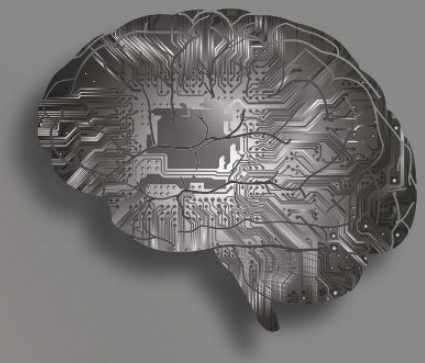

The SARS-CoV-2 Spike Protein: An Engineered PRION Like Incapacitating Agent

Kevin W. McCairn Ph.D.

Vejon Health 2023

Post Vax/Long COVID Congress - The Silent Disaster

20+ Years Investigating Cortico-Basalganglia Neurodegenerative & Neuropsychiatric Disease

ResearchGate

Kevin W McCairn Edit

Ph.D · Principal Investigator at Korea Brain Research Institute

Japan | *Website*

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577.9	9,897	1,139	35
Research Interest Score	Reads	Citations	Recommendations
+1.1 last week	+34 last week	+4 last week	---

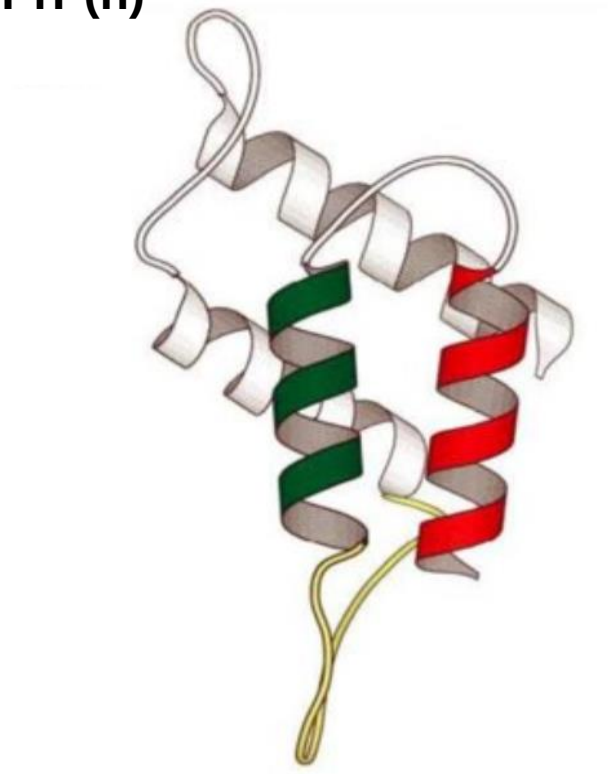
What Are Amyloids & PRIONS?

Type	Protein/Peptide
Amyloidogenic	
Aβ	Amyloid beta - associated with Alzheimer's disease
α-synuclein	Associated with Parkinson's disease
Serum amyloid A	Associated with systemic amyloidosis
Tau	Associated with Alzheimer's disease and other tauopathies, such as frontotemporal dementia
Prion-Like	
PrP- Scrapie Form	Prion protein - associated with Creutzfeldt-Jakob disease, kuru, fatal familial insomnia, and variant Creutzfeldt-Jakob disease

What Are Amyloids & PRIONS?

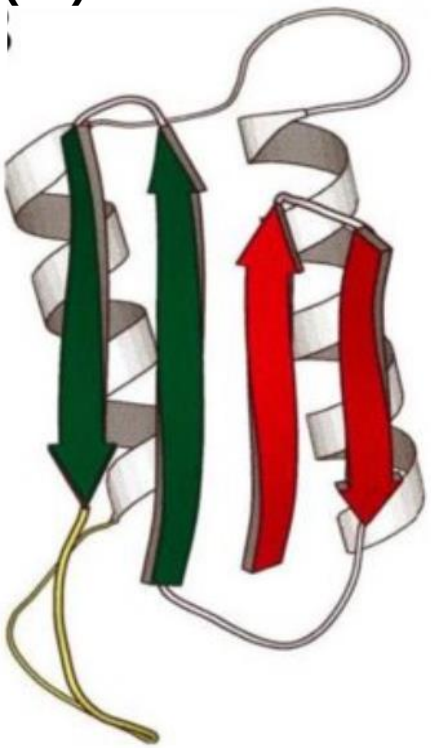
Tissue: PRION
(proteinaceous infective particle)

PrP(n)



Alpha Helical

PrP(sc)



Beta-Pleated Sheet

PRIONS & Amyloids Weaponization

Prions as Bioweapons?

Much Ado About Nothing; or Apt Concerns Over Tiny Proteins used in Biowarfare

Add bookmark

Tags: CBRN CBRNE National Security

By: James Giordano, Jennifer Snow, Joseph DeFranco

09/13/2019

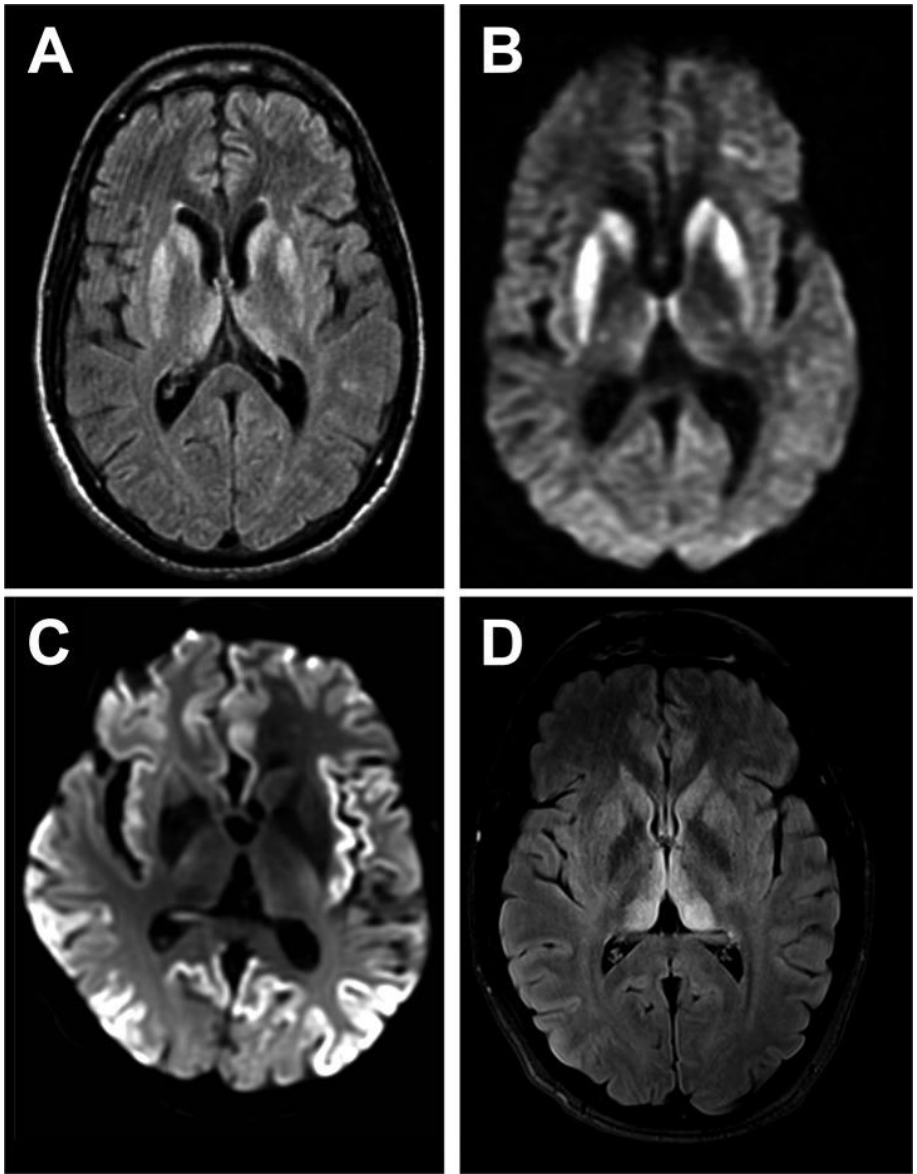
Although instances of biowarfare and bioterrorism have been rare in the 21st century, somewhat inconspicuous, but rapidly advancing bioscience and technology that are capable of being weaponized are becoming ever more difficult to surveille, regulate, and govern. One such emerging threat is

Kuru Kuru A Transmissible PRION Disorder

Canonical PRION Disorders

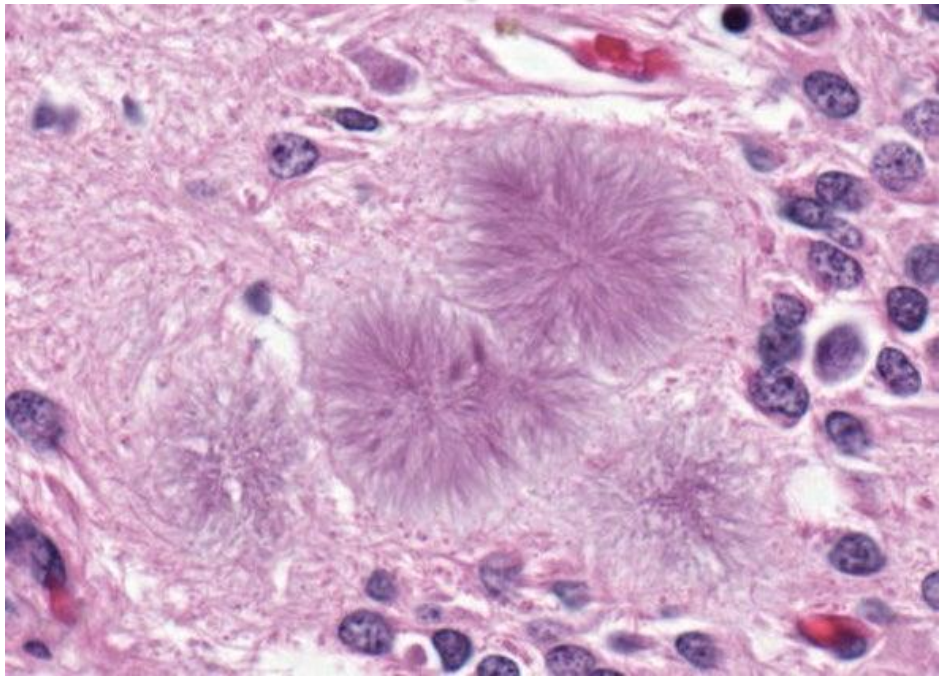
Scientific Situational Awareness: Is There A Problem/Threat?

Canonical PRION Disorders



← MRI Abnormalities

Tissue: Amyloid Plaques



https://phil.cdc.gov/Details.aspx?pid=10130

Scientific Situational Awareness: Is There A Problem/Threat?

Non-Canonical PRION Disorders

Science

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HOME > SCIENCE > VOL. 349, NO. 6248 > ALZHEIMER'S AND PARKINSON'S DISEASES: THE PRION CONCEPT IN RELATION TO ASSEMBLED AB, TAU, AND A-...

REVIEW

Alzheimer's and Parkinson's diseases: The prion concept in relation to assembled Aβ, tau, and α-synuclein

MICHEL GOEDERT

SCIENCE · 7 Aug 2015 · Vol 349,Issue 6248 · DOI: 10.1126/science.1255555

Converging paradigms in neurodegeneration

Parkinson's disease and Alzheimer's disease are progressive neurodegenerative diseases with increasing prevalence in our aging populations. Recent evidence suggests that some of the molecular mechanisms involved in the pathology of these diseases have similarities to those observed in infectious prion diseases such as bovine spongiform encephalopathy (mad cow disease). Goedert reviews how the spread of a variety of pathological protein aggregates is involved in neurodegenerative disease.

Science, this issue p. 10.1126/science.1255555

Scientific Situational Awareness: Is There A Problem/Threat?

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Alzheimer's and Parkinson's diseases: The prion concept in relation to assembled Aβ, tau, a

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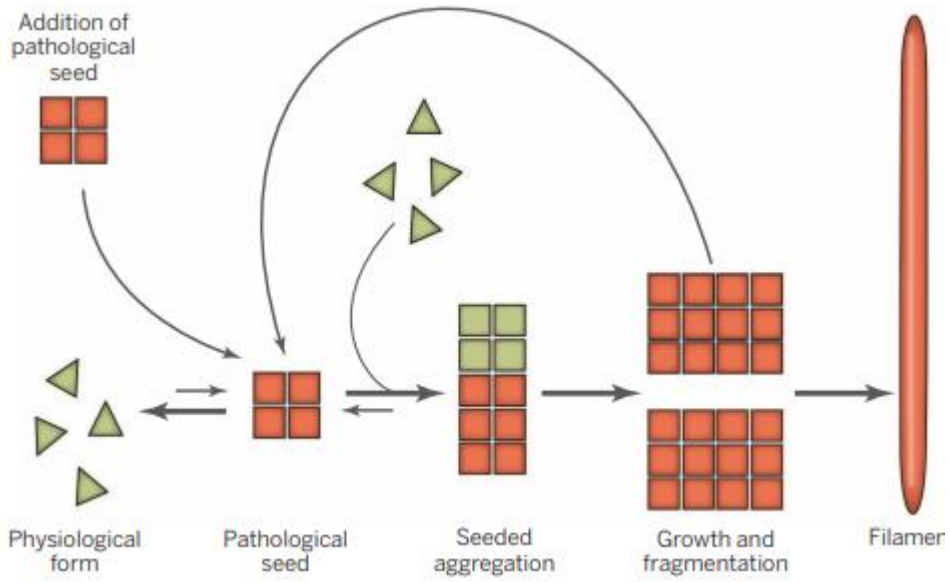
SCIENCE · 7 Aug 2015 · Vol 349,Issue 6248 · DOI: 10.1126/science.1255555

1,718 484



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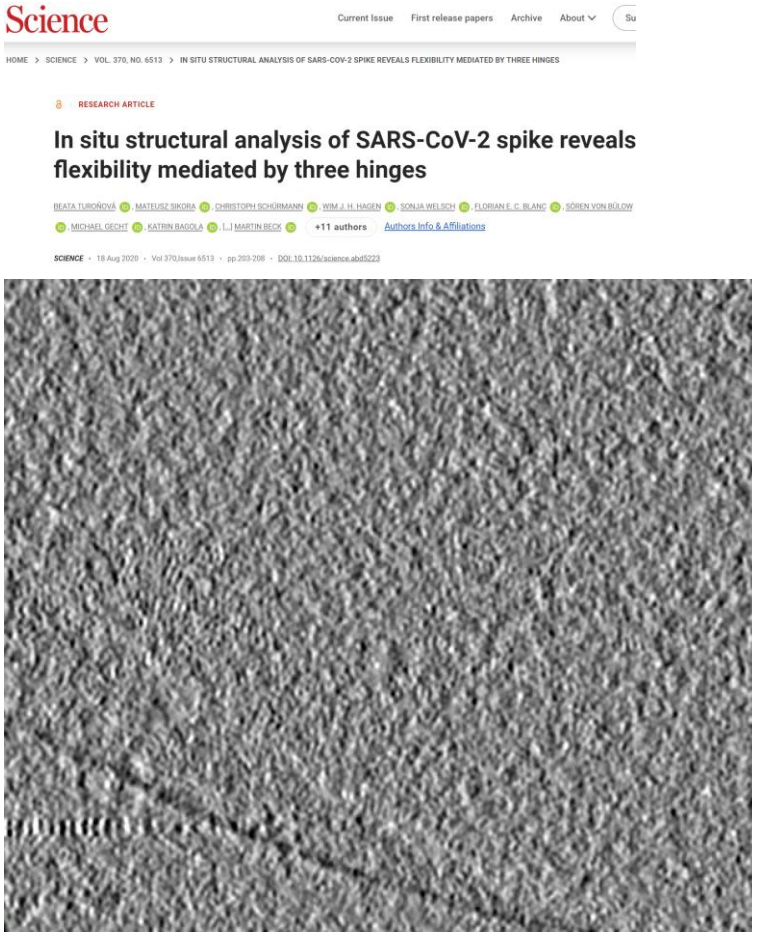
Science, this issue p. 10.1126/science.1255555

The SARS-CoV-2 Virus A Neurotropic GoF Chimera

SARS-CoV-2 Has Amyloidogenic & PRION Like Properties



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HOME > SCIENCE > VOL. 370, NO. 6513 > IN SITU STRUCTURAL ANALYSIS OF SARS-COV-2 SPIKE REVEALS FLEXIBILITY MEDIATED BY THREE HINGES

RESEARCH ARTICLE

In situ structural analysis of SARS-CoV-2 spike reveals flexibility mediated by three hinges

BEATA TURONOVÁ, MATEUSZ SIKORA, CHRISTOPH SCHÜRMANN, WIM J. H. HAGEN, SONJA WELSCH, FLORIAN E. C. BLANC, SÖREN VON BÜLOW, MICHAEL GECHT, KATRIN BAGOLA, [...] MARTIN BECK +11 authors Authors Info & Affiliations

SCIENCE • 18 Aug 2020 • Vol 370,Issue 6513 • pp.203-208 • DOI: 10.1126/science.abd5223

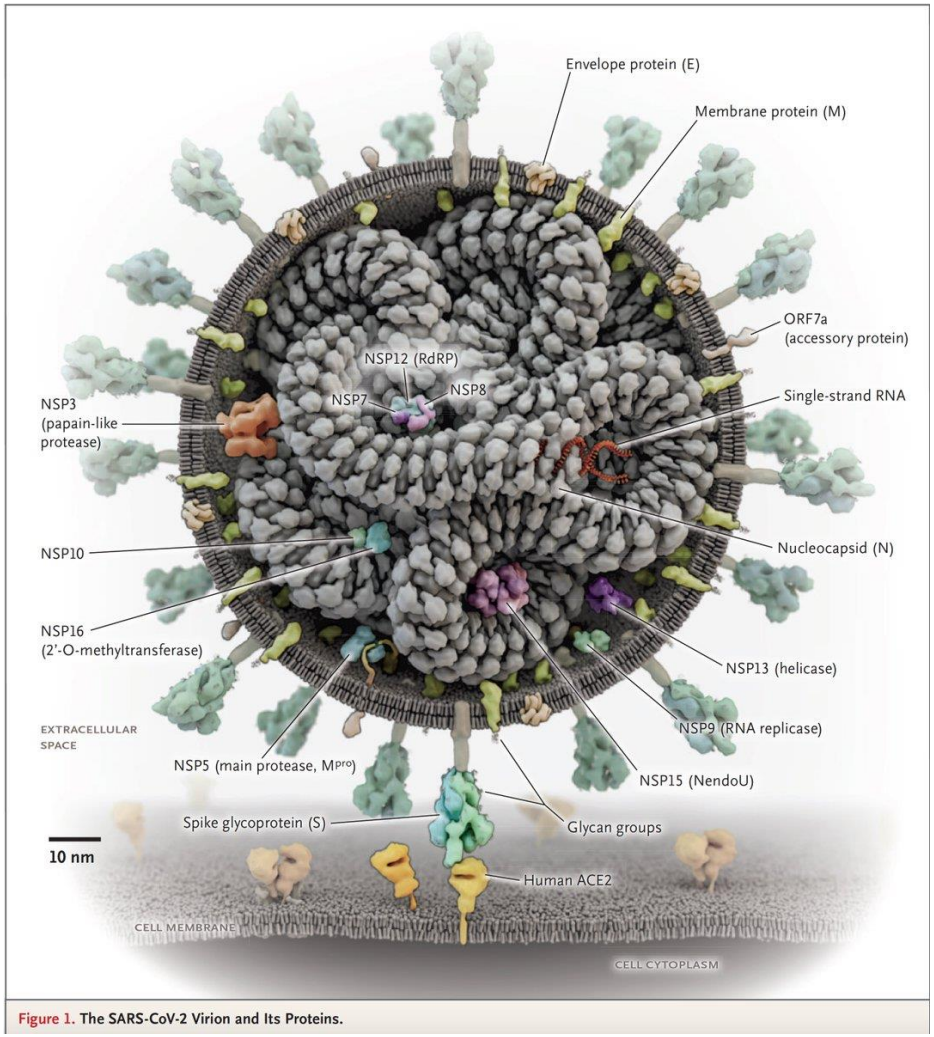

Figure 1. The SARS-CoV-2 Virion and Its Proteins.

The SARS-CoV-2 Virus A Neurotropic GoF Chimera

nature communications

Article

https://doi.org/10.1038/s41467-023-40228-7

Neuroinvasion and anosmia are independent phenomena upon infection with SARS-CoV-2 and its variants

Received: 17 October 2022

Accepted: 11 July 2023

Published online: 26 July 2023

Check for updates

Guilherme Dias de Melo[1], Victoire Perraud[1,14], Flavio Alvarez[2,3,14], Alba Vieites-Prado[4,14], Seonhee Kim[1], Lauriane Kergoat[1], Anthony Coleon[1], Bettina Salome Trüeb[5], Magali Tichit[6], Aurèle Piazza[7], Agnès Thierry[7], David Hardy[6], Nicolas Wolff[2], Sandie Munier[8], Romain Koszul[7], Etienne Simon-Lorière[9], Volker Thiel[10], Marc Lecuit[11,12], Pierre-Marie Lledo[13], Nicolas Renier[4], Florence Larrous[1,15] & Hervé Bourhy[1,15]

Anosmia was identified as a hallmark of COVID-19 early in the pandemic, however, with the emergence of variants of concern, the clinical profile induced by SARS-CoV-2 infection has changed, with anosmia being less frequent. Here, we assessed the clinical, olfactory and neuroinflammatory conditions of golden hamsters infected with the original Wuhan SARS-CoV-2 strain, its isogenic ORF7-deletion mutant and three variants: Gamma, Delta, and Omicron/BA.1. We show that infected animals develop a variant-dependent clinical disease including anosmia, and that the ORF7 of SARS-CoV-2 contributes to the induction of olfactory dysfunction. Conversely, all SARS-CoV-2 variants are neuroinvasive, regardless of the clinical presentation they induce. Taken together, this confirms that neuroinvasion and anosmia are independent phenomena upon SARS-CoV-2 infection. Using newly generated nanoluciferase-expressing SARS-CoV-2, we validate the olfactory pathway as a major entry point into the brain in vivo and demonstrate in vitro that SARS-CoV-2 travels retrogradely and anterogradely along axons in microfluidic neuron-epithelial networks.

SARS-CoV-2 Spike Protein Is Highly Amyloidogenic

Gain of Function With PRION Disorders

JACS
JOURNAL OF THE AMERICAN CHEMICAL SOCIETY

Amyloidogenesis of SARS-CoV-2 Spike Protein

Sofie Nyström* and Per Hammarström*

Cite this: *J. Am. Chem. Soc.* 2022, 144, 20, 8945−8950
Publication Date: May 17, 2022
https://doi.org/10.1021/jacs.2c03925

SARS-CoV-2 Spike Protein Is Highly Amyloidogenic

Gain of Function With PRION Disorders

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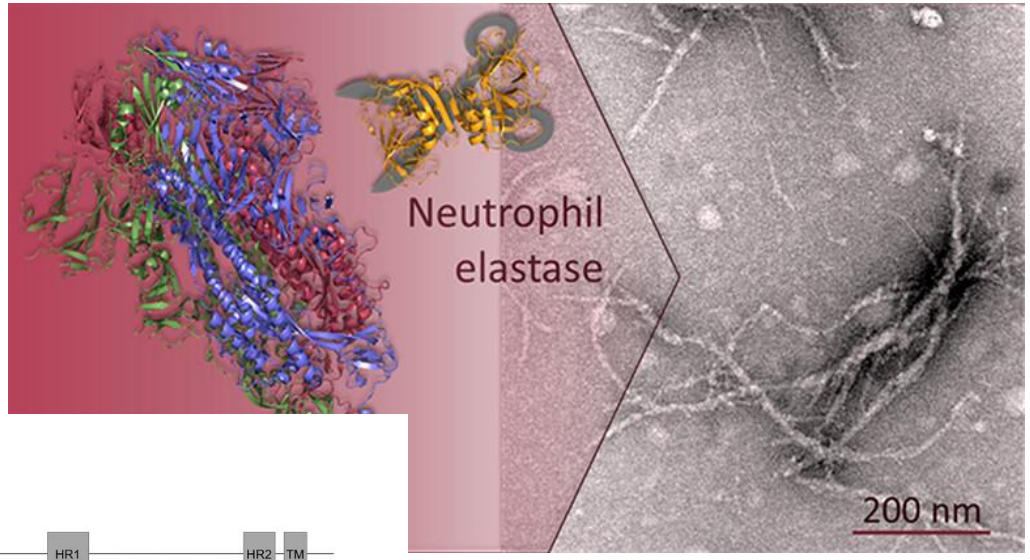
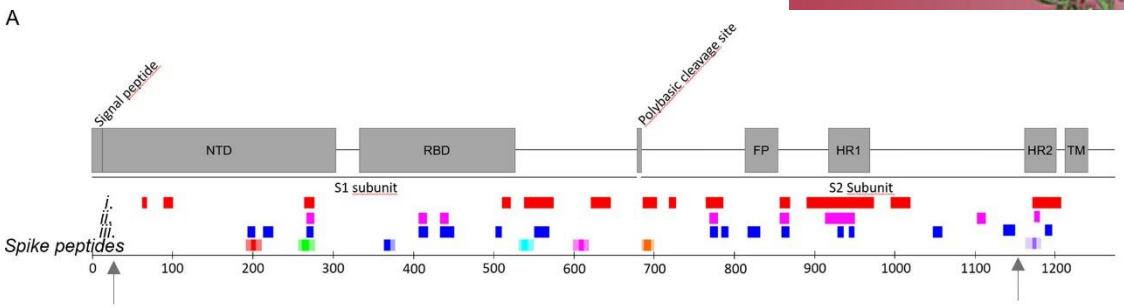
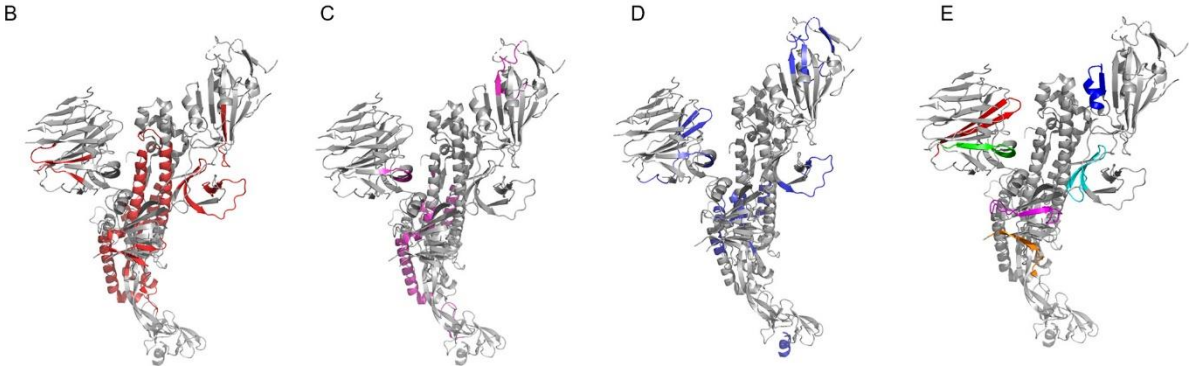
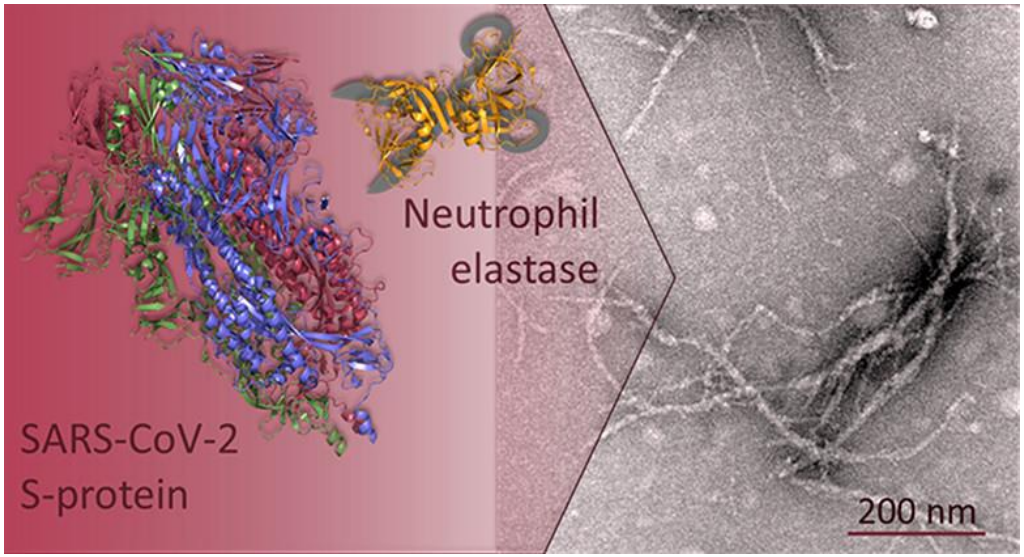






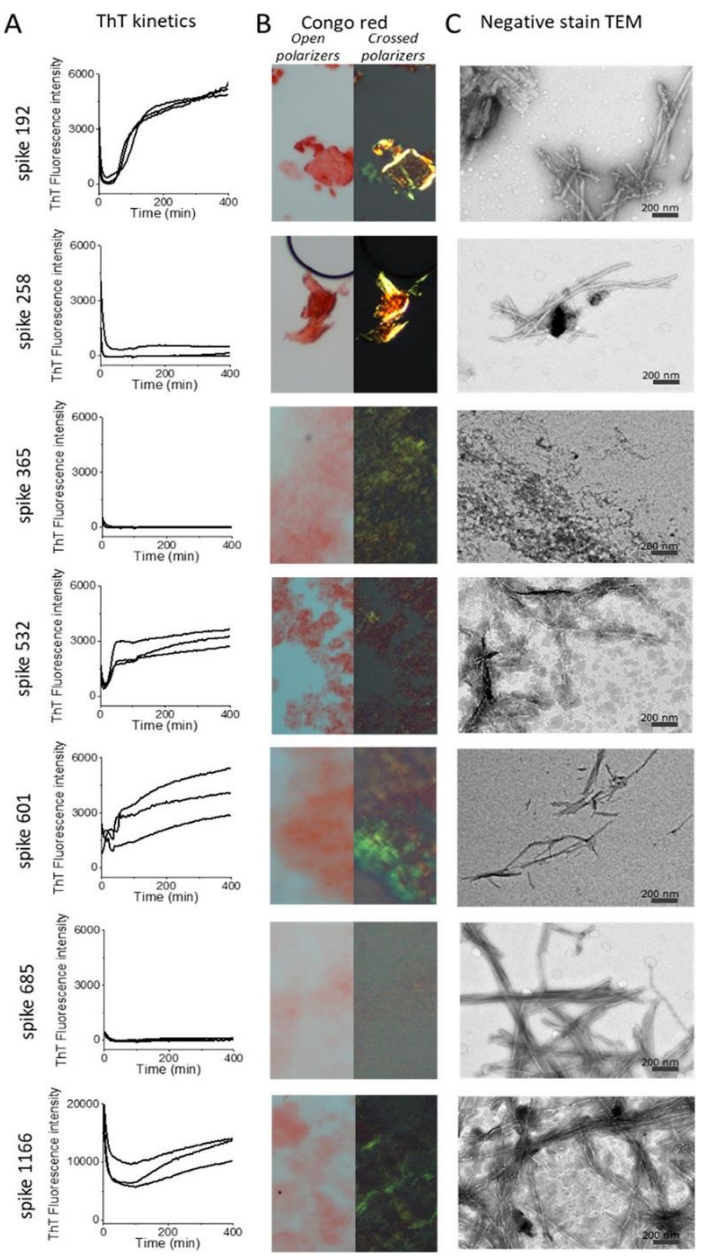
SARS-CoV-2 Spike Protein Is Highly Amyloidogenic




```
MERS          EDEILEWFGITQTAQGVHLFSSRYVD----LYGG--------------N-MFQFATLPVYD
SARS          SLDVSEKSG---NFKHLREFVFKNKDGFLYVYKGYQPIDVVRDLPSGFNTLKPIFKLPLGI
SARSCOV2      LMDLEGKQG---NFKNLREFVFKNIDGYFKIYSKHTPINLVRDLPQGFSALEPLVDLPIGI
                ::    *   . : :: *  :  *    :*                . :  :  **:
                        176                                              233

OC43          TYDVNAT------YLYFHFYQEGGTFYAYFTDTG------FVTKF-----LFNVYLGM
229E          NGTNTSH------SVCNGCVGHSENVFAVESGGY------IPSNF-----AFNNWFLL
NL63          NGRIVNY------TVCDDCNGYTDNIFSVQQDGR------IPNGF-----SFNNWFLL
HKU1          FTYNVSTD------WLYFFYQERGTFYAYYADSG------MPTTF-----LFSLYLGT
SARSCOV2      LMDLEGKQGNFKNLREFVFKNIDGYFKIYSKHTPINLVRDLPQGFSALEPLVDLPIGI
                                  .                :   *      ..  :
                        176                                      233
```

Figure S8 A) WALTZ prediction of amyloidogenic sequences in Spike proteins from several corona viruses known to infect humans **B)** Sequence alignments of spike192 and flanking amino acids from the three corona viruses causing severe disease. MERS S-protein does not contain the amyloidogenic sequence, SARS S-protein contains a similar amyloidogenic sequence as SARS-CoV-2 S-protein in this part of the amino acid sequence. SARS-CoV-2 S-protein segment 194-213, highlighted in red was a peptide predicted for elastase cleavage.



SARS-CoV-2 Spike Protein & Amyloidogenic Microclots

BIOSCIENCE REPORTS

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Volume 41, Issue 8
August 2021

RESEARCH ARTICLE | AUGUST 20 2021

SARS-CoV-2 spike protein S1 induces fibrin(ogen) resistant to fibrinolysis: implications for microclot formation in COVID-19

Lize M. Grobbelaar; Chantelle Venter; Mare Vlok; Malebogo Ngoepe; Gert Jacobus Laubscher; Petrus Johannes Lourens; Janami Steenkamp; Douglas B. Kell; Etheresia Pretorius

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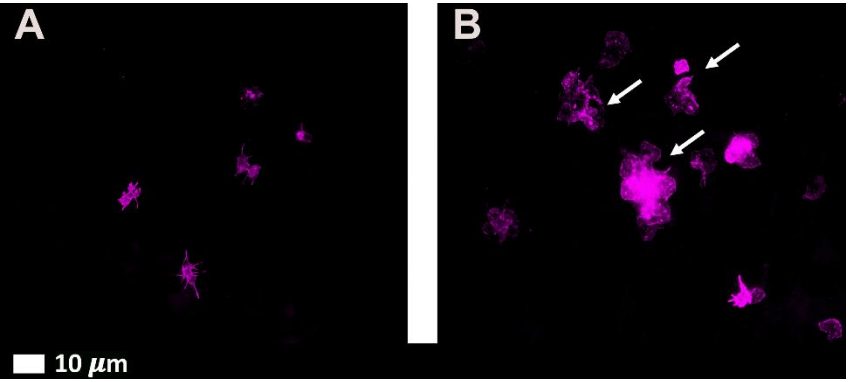
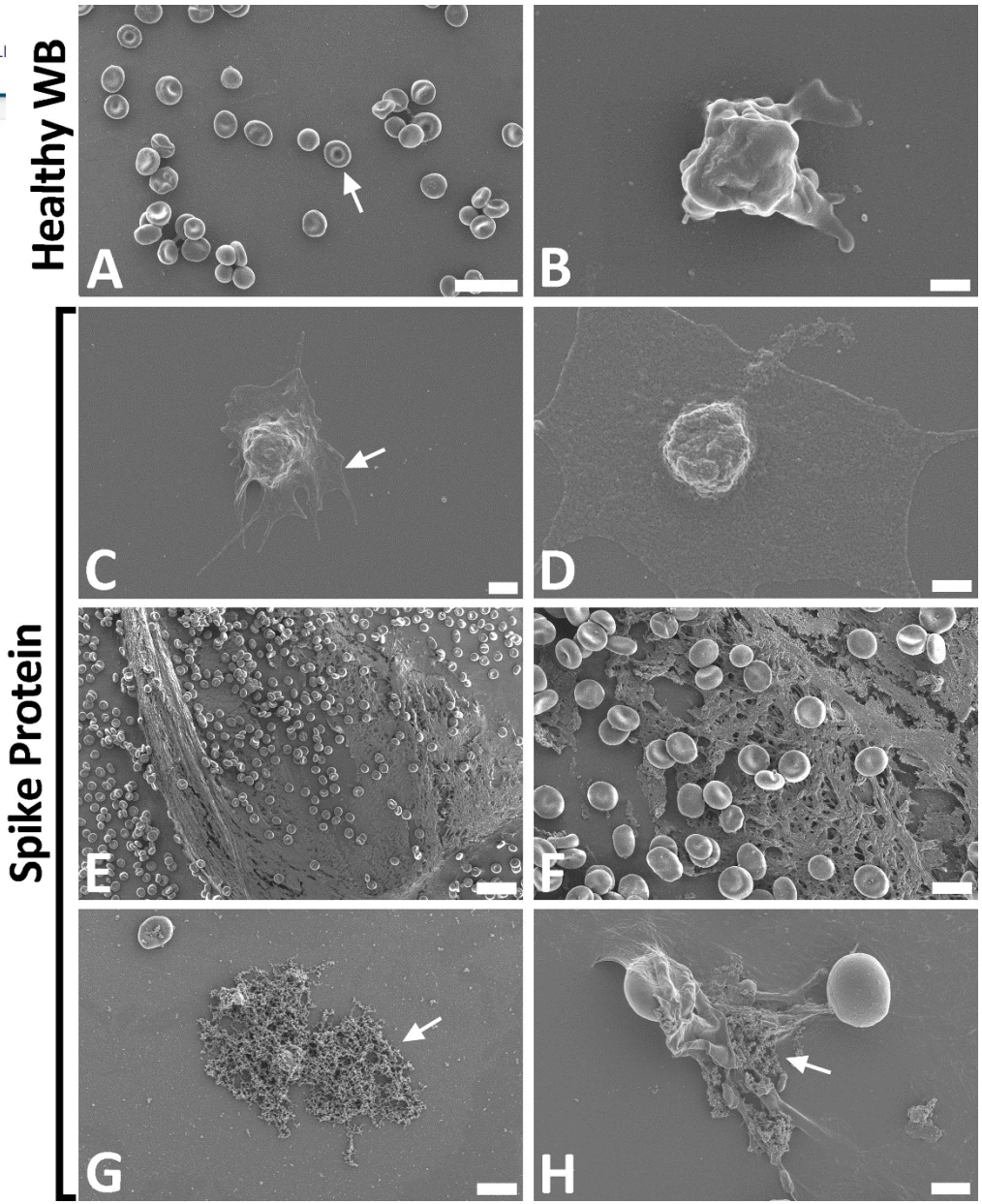
Biosci Rep (2021) 41 (8): BSR20210611.
https://doi.org/10.1042/BSR20210611 Article history





SARS-CoV-2 Contains HIV Like Epitope Homologies (GP120)

Gain of Function With PRION Disorders

Dr Ah Kahn Syed

Pseudonym. @arkmedic on twitter. MD PhD.

Subscribed

Arkmedic's blog
By Dr Ah Kahn Syed

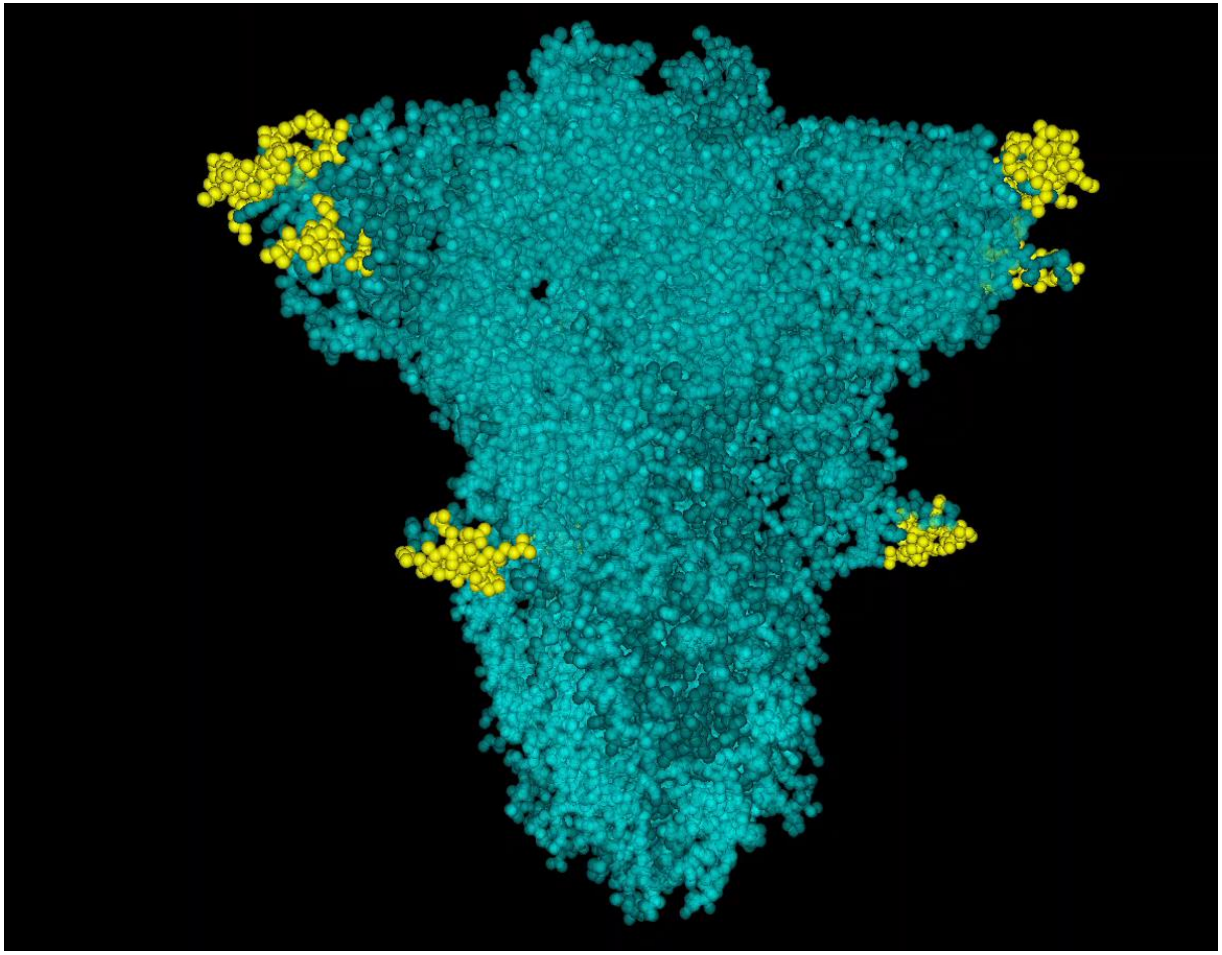
Stuff you didn't know about medicine

Absolute proof: The Gp-120 sequences prove beyond all doubt that "COVID-19" was man-made

The "missing link" was there in Pradhan's paper all along, we just needed to ask the right question: "where are the genome sequences for the Gp-120 inserts"

Dr Ah Kahn Syed
Apr 10

125 90



Cold Spring Harbor Laboratory

bioRχiv
THE PREPRINT SERVER FOR BIOLOGY

bioRxiv posts many COVID19-related papers. A reminder: they have not been formally peer-reviewed and should not guide health-related behavior or be reported in the press as conclusive.

New Results

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Uncanny similarity of unique inserts in the 2019-nCoV spike protein to HIV-1 gp120 and Gag

Prashant Pradhan, Ashutosh Kumar Pandey, Akhilesh Mishra, Parul Gupta, Praveen Kumar Tripathi, Manoj Balakrishnan Menon, James Gomes, Perumal Vivekanandan, Bishwajit Kundu

doi: https://doi.org/10.1101/2020.01.30.927871

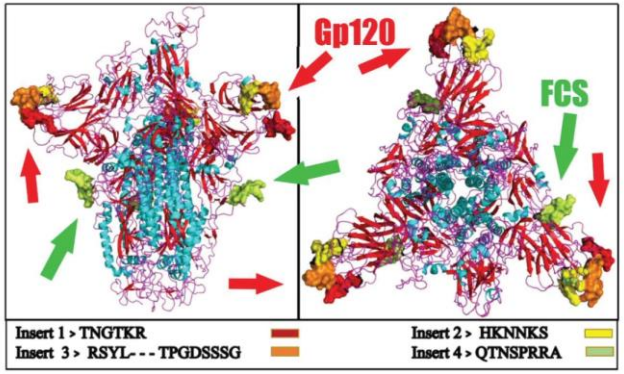
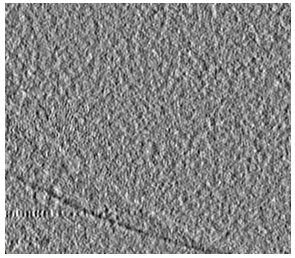


Figure 3. Modelled homo-trimer spike glycoprotein of 2019-nCoV virus. The inserts from HIV envelop protein are shown with colored beads, present at the binding site of the protein.



Science

In situ structural analysis of SARS-CoV-2 spike reveals flexibility mediated by three hinges

SARS-CoV-2 Contains HIV Like Epitope Homologies (GP120)

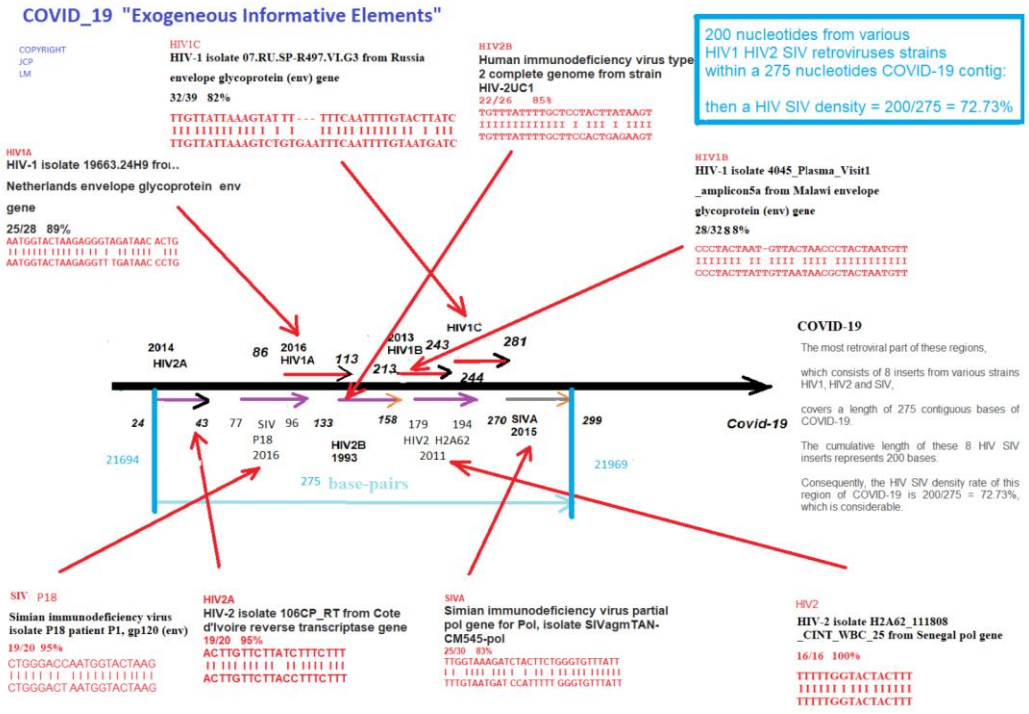
Gain of Function With PRION Disorders

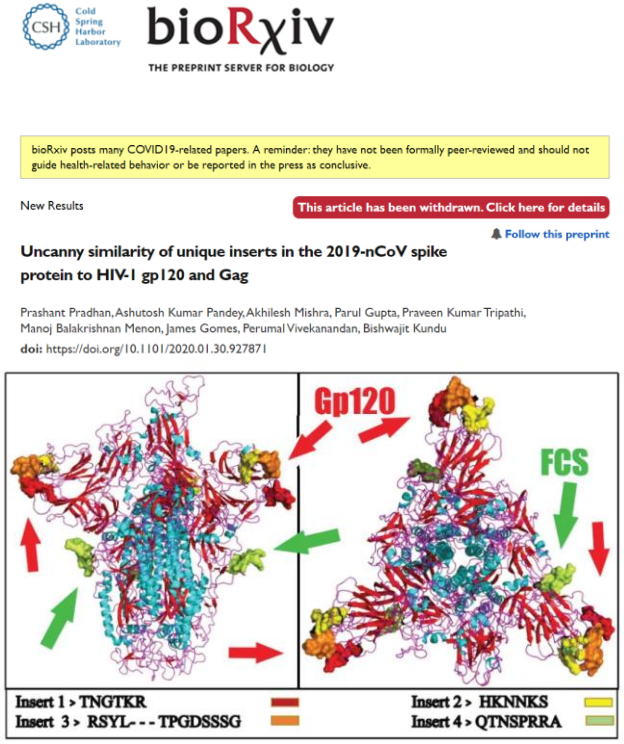
COVID-19, SARS and Bats Coronaviruses Genomes Unexpected Exogenous RNA Sequences

Jean Claude Perez, Luc Montagnier

Jean-Claude Perez, PhD Maths § Computer Science Bordeaux University, RETIRED interdisciplinary researcher (IBM Emeritus, IBM European Research Center on Artificial Intelligence Montpellier), Bordeaux metropole, France

Luc Montagnier , Paris, France





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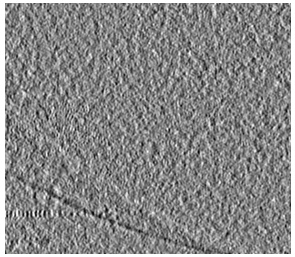

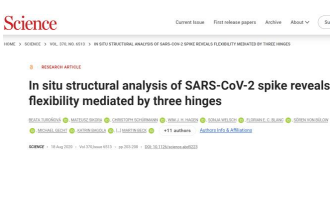

HIV GP120 Is Neuroinflammatory & Amyloidogenic

Arch. Pharm. Res. (2021) 44:689–701
https://doi.org/10.1007/s12272-021-01340-8

Online ISSN 1976-3786
Print ISSN 0253-6269

Archives of Pharmacal Research
www.springer.com/12272

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RESEARCH ARTICLE

Amyloidogenic, neuroinflammatory and memory dysfunction effects of HIV-1 gp120

Young-Jung Lee[3] · In Jun Yeo[1] · Dong Young Choi[2] · Jaesuk Yun[1] · Dong Ju Son[1] · Sang-Bae Han[1] · Jin Tae Hong[1]

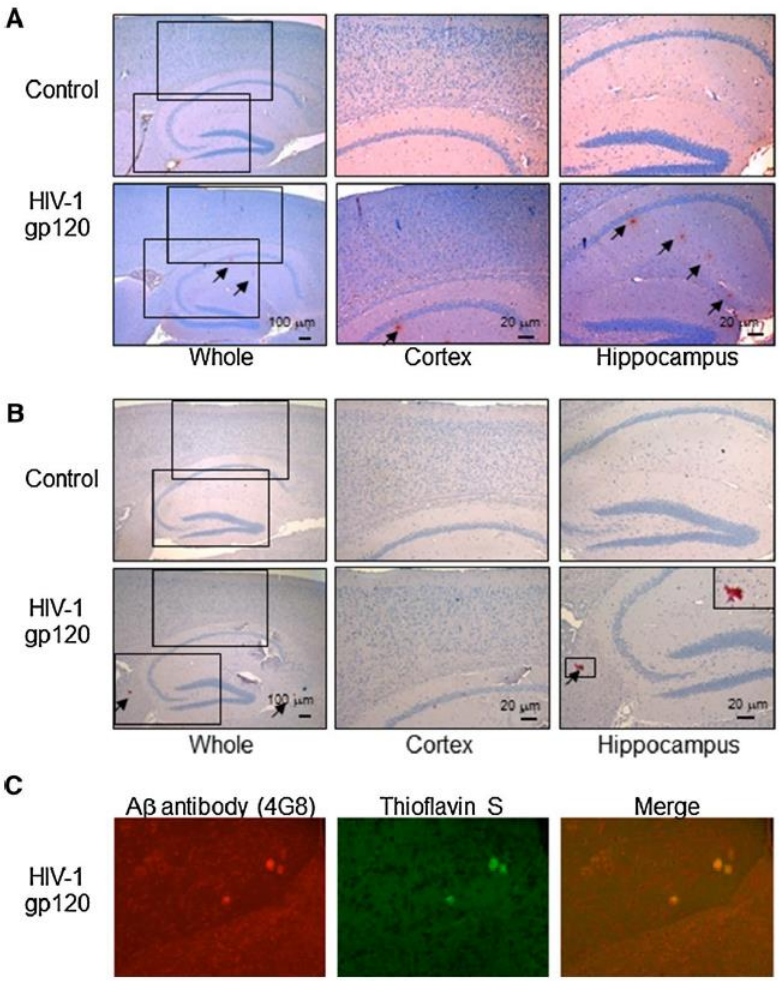
Received: 1 April 2021 / Accepted: 30 June 2021 / Published online: 23 July 2021

Abstract Human immunodeficiency virus 1 (HIV-1) infection can cause several HIV-associated neurocognitive disorders a variety of neurological impairments characterized by the loss of cortical and subcortical neurons and decreased cognitive and motor function. HIV-1 gp120, the major envelope glycoprotein on viral particles, acts as a binding protein for viral entry and is known to be an agent of neuronal cell death. To determine the mechanism of HIV-1 gp120-induced memory dysfunction, we performed mouse intracerebroventricular (i.c.v.) infusion with HIV-1 gp120 protein (300 ng per mouse) and investigated memory impairment and amyloidogenesis. Infusion of the HIV-1 gp120 protein induced memory dysfunction, which was evaluated using passive avoidance and water maze tests. Infusion of HIV-1 gp120 induced neuroinflammation, such as the release of iNOS and COX-2 and the activation of astrocytes and microglia and increased the mRNA and protein levels of IL-6, ICAM-1, M-CSF, TIM, and IL-2. In particular, we found that the infusion of HIV-1 gp120 induced the accumulation of amyloid plaques and signs of elevated amyloidogenesis, such as increased expression of amyloid precursor protein and BACE1 and increased β-secretase activity. Therefore, these studies suggest that HIV-1 gp120 may induce memory impairment through Aβ accumulation and neuroinflammation.

Keywords HIV-1 · gp120 · Amyloid beta · Neuroinflammation

Introduction

Alzheimer's disease (AD) is the most common cause of dementia, accounting for 50–75% of all cases (Ferri et al. 2009; Lee et al. 2010) and the most common neurological complication. AD is defined by progressive synaptic impairment, excessive formation and accumulation of amyloid-beta (Aβ), and neuroinflammation (Blennow et al. 2006; Lee et al. 2010). Although the underlying mechanism of AD development remains unclear, experimental data have



mRNA Gene Transfection Causes Amyloidogenic Cascades

CASE REPORT

SN

Subcutaneous Uptake on [18F]Florbetaben PET/CT: a Case Report of Possible Amyloid-Beta Immune-Reactivity After COVID-19 Vaccination

Riccardo Laudicella[1,2,3] · Irene Andrea Burger[3,4] · Francesco Panasiti[1,2] · Costanza Longo[1,2] · Salvatore Scalisi[1] · Fabio Minutoli[2] · Sergio Baldari[2] · Luigi Maria Edoardo Grimaldi[5] · Pierpaolo Alongi[1]

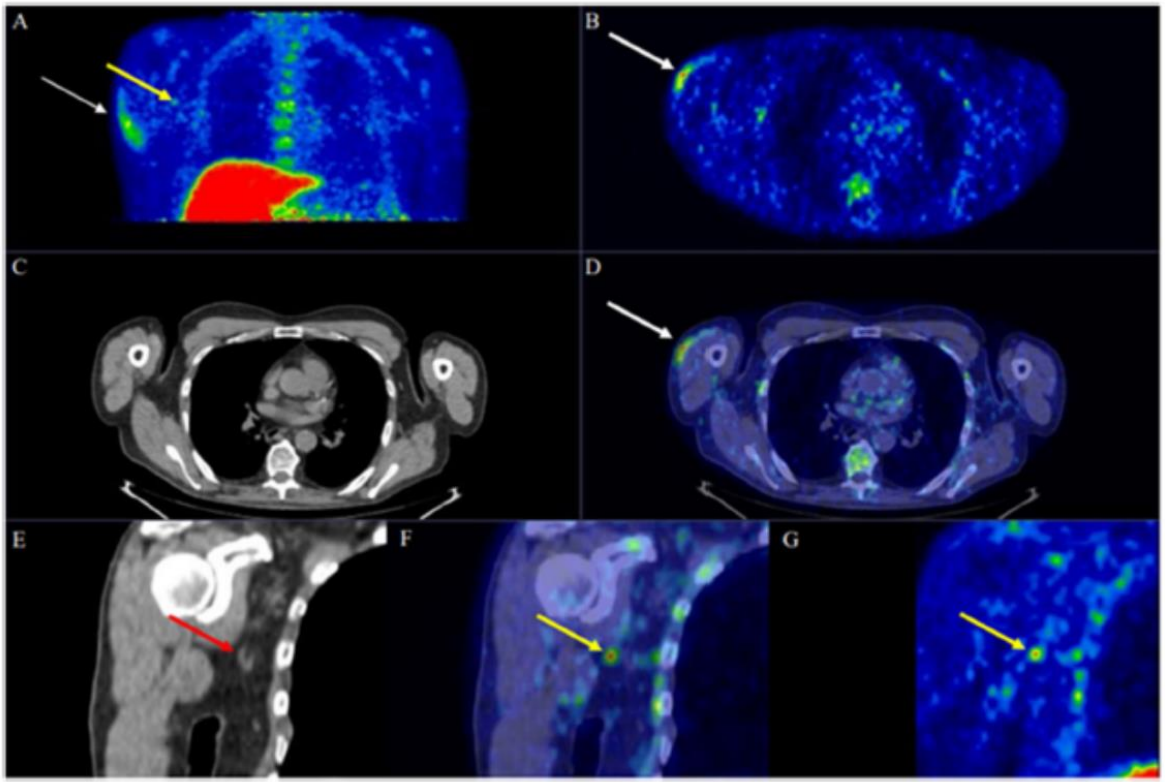
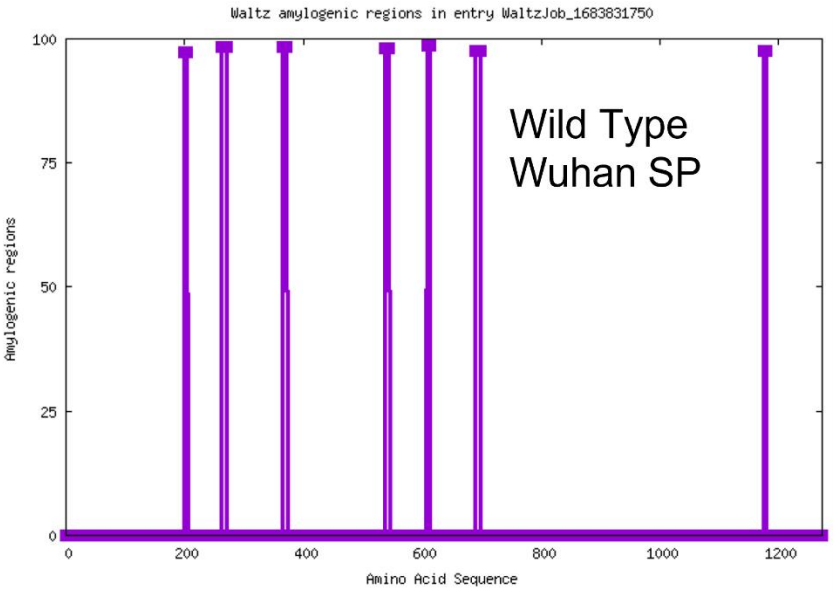
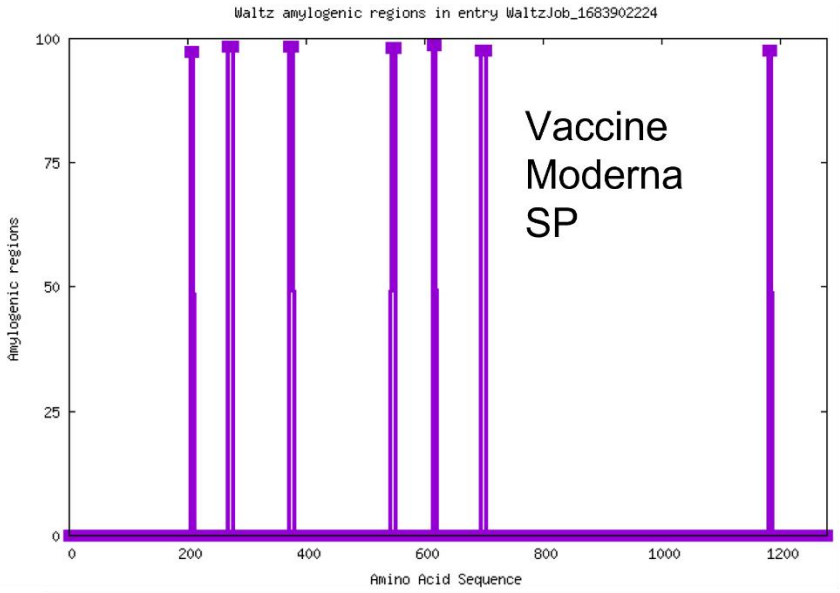
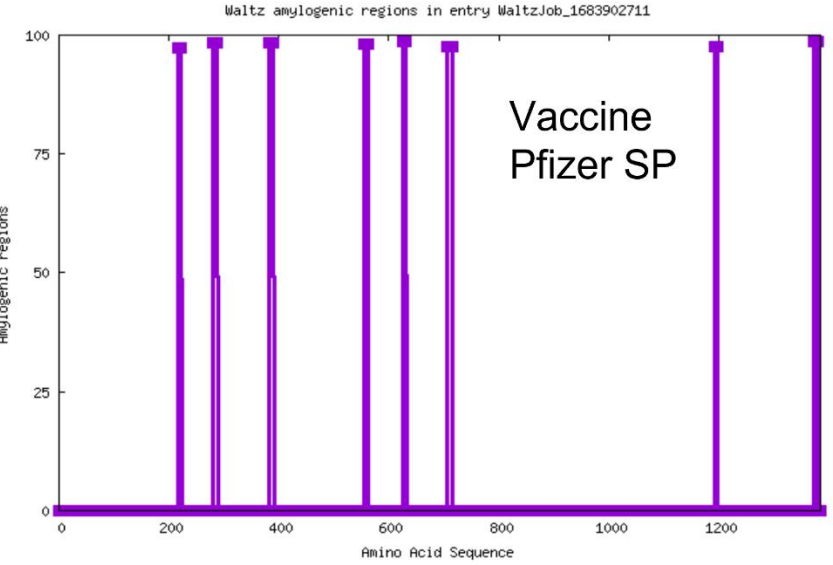


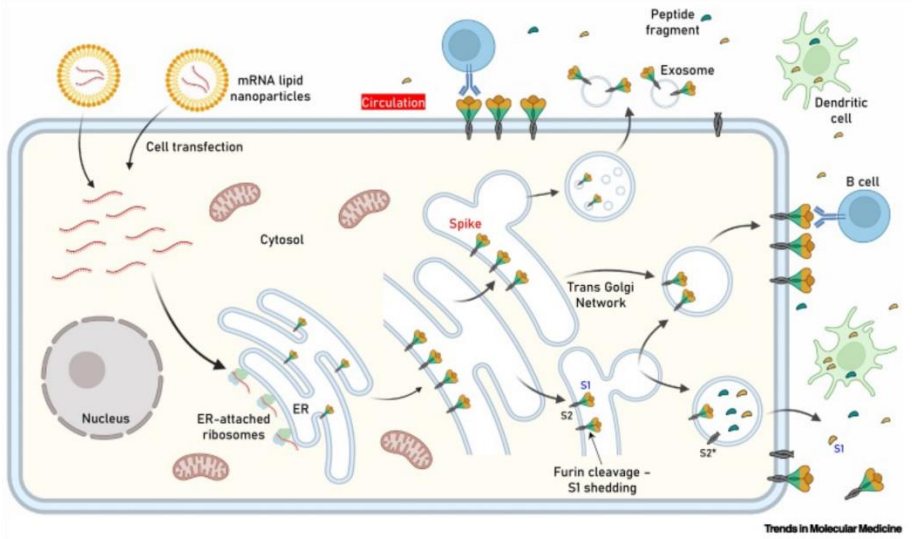
Fig. 1 [^{18}F]Florbetaben PET/CT: MIP (**A**), PET (axial-**B**, coronal-**G**), CT (axial-**C**, coronal-**E**), PET/CT (axial-**D**, coronal-**F**) images demonstrated ill-defined uptake in the right arm's subcutaneous tissues (SUVmax 5.6; **white-arrows**) and next to a possible right-axillar lymph node (SUVmax 4.75; **yellow-arrows**) evident on low-dose CT scan without breathing control (**red arrows**). Reprinted with permission from Nuclear Medicine Unit, Fondazione Istituto G. Giglio, Cefalù (Palermo), Italy.

mRNA Gene Transfection Causes Amyloidogenic Cascades









Trougakos et al. 2022

mRNA Gene Transfection Correlated With CJD In France

Gain of Function With PRION Disorders

Towards the emergence of a new form of the neurodegenerative Creutzfeldt-Jakob disease: Twenty six cases of CJD declared a few days after a COVID-19 "vaccine" Jab

Jean Claude Perez, PhD Maths§Computer Science Bordeaux University ; Retired (IBM European Research center on Artificial Intelligence Montpellier France) ; Bordeaux metropole France; https://orcid.org/0000-0001-6446-2042 France
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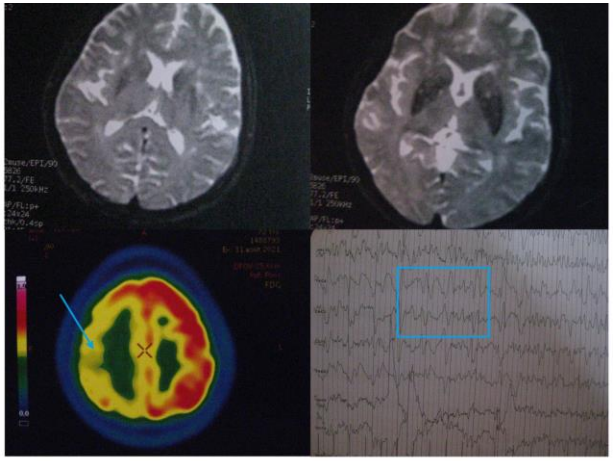
Luc Montagnier R.I.P MD. Virologist, Fondation Luc Montagnier Quai Gustave-Ador 62 1207 Genève, Switzerland



Case 4 (M.D): MRI, PET and EEG proofs

Figure 31 – The case of M.D.: MRI, PET and EEG (D. M) -Brain MRI (Diffusion Weighted Imaging) and (Fluid-Attenuated Inversion Recovery : FLAIR) and (T2) : abnormalities of

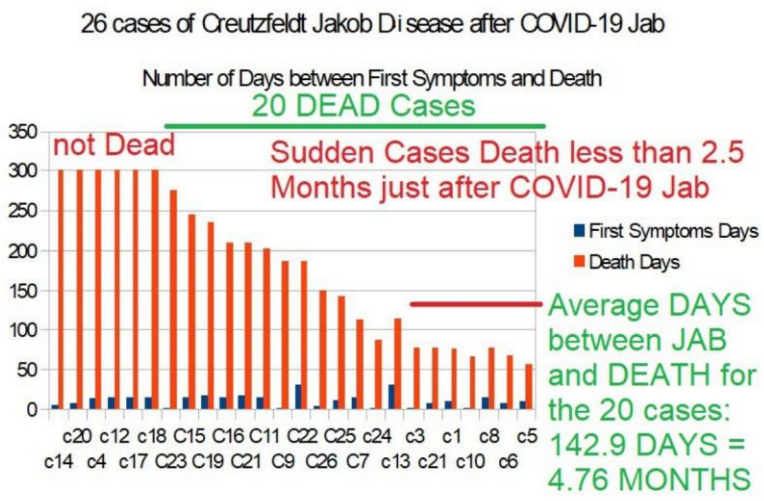


Figure 34 – The distribution of numbers of days between SARS-CoV2 Jab and first CJD Symptoms.

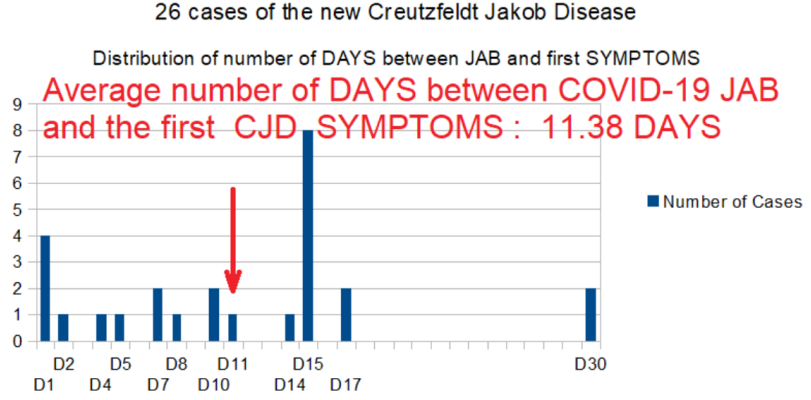
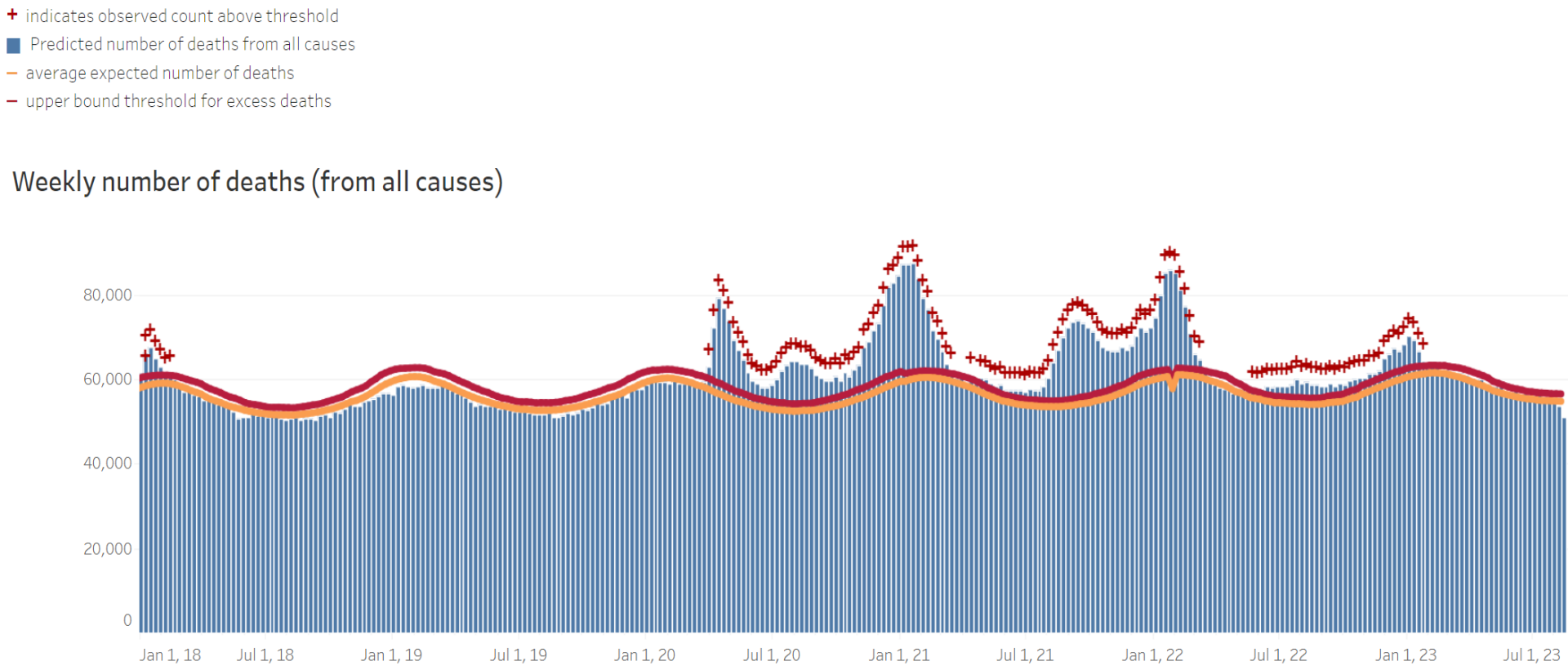


Figure 33– The distribution of numbers of days between SARS-CoV2 Jab and first CJD Symptoms.

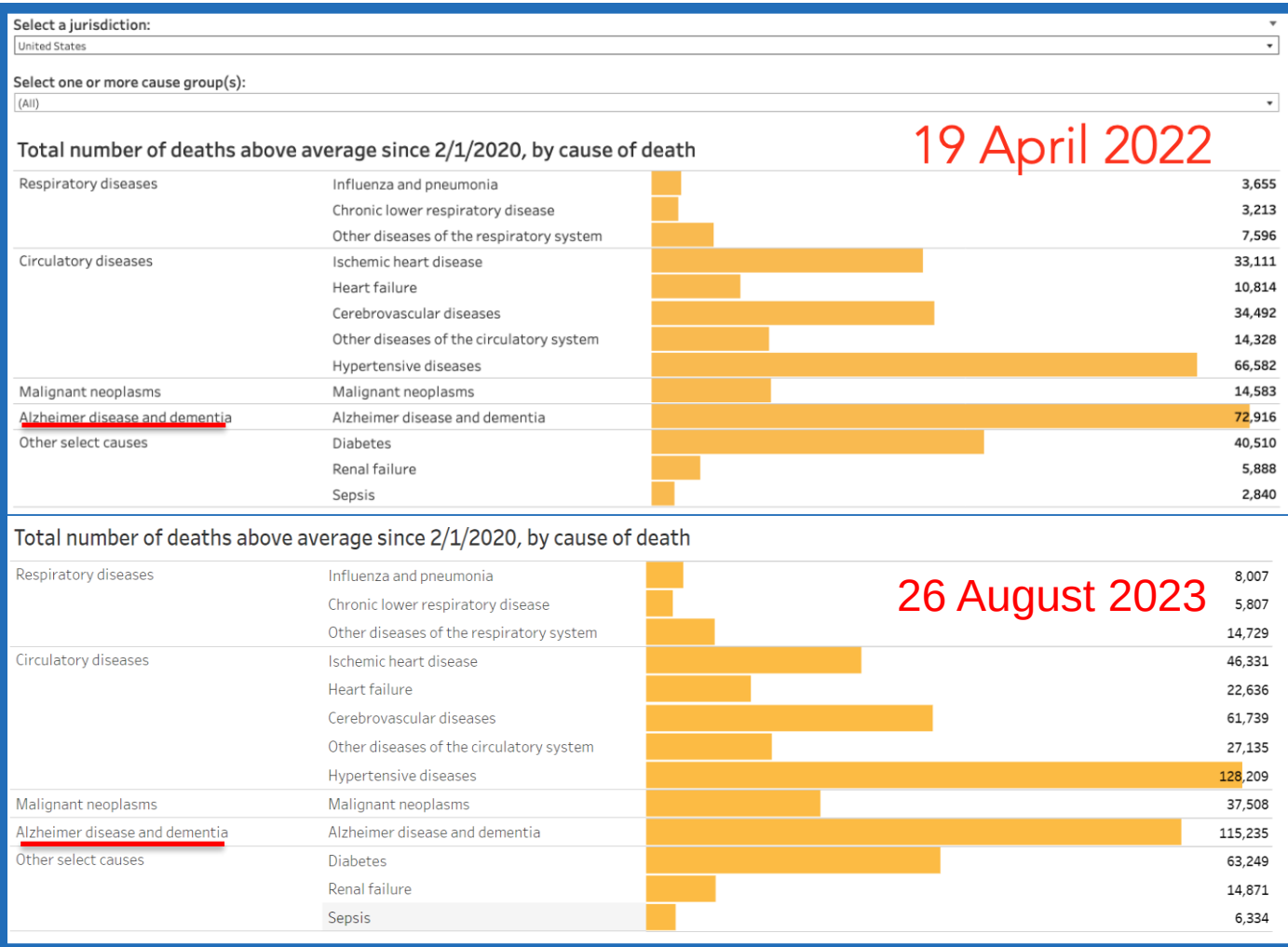
Sustained All Cause Mortality (Non-COVID) Has Defined Pandemic

https://www.cdc.gov/nchs/nvss/vsrr/covid19/excess_deaths.htm



Sustained All Cause Mortality (Non-COVID) Has Defined Pandemic

In Absolute Numbers the People Tend to be over 45-years of Age, Caucasian & Dying from InflammoThrom-botic & Prion Diseases.



https://www.cdc.gov/nchs/nvss/vsrr/covid19/excess_deaths.htm

Sustained All Cause Mortality (Non-COVID) Has Defined Pandemic

% Excess Deaths (Non-COVID-19) by Age Group, Sex & Month (that week ended in)
England
Weeks Ending 27Mar20 - 30Jun23
Source Data:- Office for Health Improvement and Disparities
Graphic:- @OutsideAllan

		0-24		25-49		50-64		65-74		75-84		85+		Total
Year	Mont	Females	Males	Females	Males	Females	Males	Females	Males	Females	Males	Females	Males	
2020	3	-10.0%	-23.0%	-2.8%	2.1%	0.2%	-3.7%	-8.4%	-2.0%	-5.5%	-0.6%	-6.3%	-1.8%	-3.8%
2020	4	-5.8%	-20.5%	7.5%	0.2%	12.4%	16.1%	13.4%	18.2%	25.4%	25.9%	40.6%	36.1%	26.9%
2020	5	-15.8%	-12.0%	-1.6%	-2.3%	3.7%	4.9%	-1.8%	-4.2%	-0.5%	-1.8%	10.9%	0.2%	1.9%
2020	6	-23.9%	-21.7%	-8.0%	-7.3%	-6.0%	-2.4%	-3.3%	-8.0%	-8.6%	-10.6%	-11.3%	-13.2%	-9.4%
2020	7	-16.6%	-9.0%	0.7%	0.3%	-6.5%	2.6%	-3.1%	-4.8%	-7.1%	-7.4%	-9.2%	-13.9%	-7.2%
2020	8	1.4%	-1.3%	1.8%	5.9%	2.4%	4.1%	-5.2%	-2.5%	-0.7%	-1.2%	-2.7%	-5.4%	-1.8%
2020	9	-1.4%	-15.4%	5.6%	6.0%	1.6%	5.8%	0.2%	2.4%	0.4%	-0.1%	-2.2%	-1.9%	0.1%
2020	10	-24.0%	-11.4%	2.3%	7.4%	4.1%	4.0%	0.9%	-3.0%	-2.4%	-4.2%	-3.8%	-4.6%	-2.3%
2020	11	-11.3%	-6.1%	3.6%	5.1%	1.9%	5.9%	-7.5%	-2.9%	-8.1%	-7.0%	-7.3%	-10.7%	-5.8%
2020	12	-5.2%	-8.2%	5.5%	6.0%	-5.8%	3.1%	-10.2%	-10.9%	-14.9%	-16.1%	-14.9%	-17.7%	-12.6%
2021	1	-3.1%	-8.1%	0.3%	-11.0%	-10.4%	-6.3%	-17.0%	-16.4%	-19.9%	-23.1%	-22.8%	-26.7%	-20.1%
2021	2	2.0%	4.6%	2.9%	-1.2%	-8.2%	-3.7%	-12.6%	-15.1%	-18.2%	-17.4%	-19.3%	-24.9%	-16.7%
2021	3	16.4%	-1.2%	-4.0%	5.4%	-7.7%	-3.4%	-13.3%	-14.2%	-18.2%	-20.6%	-22.3%	-25.2%	-17.8%
2021	4	-12.5%	-13.8%	2.4%	-5.3%	-5.9%	0.6%	-9.0%	-11.4%	-13.5%	-14.3%	-16.3%	-19.3%	-13.1%
2021	5	-9.0%	7.9%	2.1%	1.2%	-5.6%	3.1%	-10.1%	-7.5%	-8.8%	-7.5%	-10.0%	-10.4%	-7.7%
2021	6	9.3%	3.5%	-2.4%	5.2%	-0.9%	6.6%	-4.6%	-5.0%	-5.1%	-3.3%	-5.6%	-7.2%	-3.9%
2021	7	-7.5%	0.7%	2.4%	3.8%	7.0%	7.2%	0.2%	2.2%	0.7%	1.7%	-0.3%	-2.0%	1.1%
2021	8	-9.0%	-5.6%	-0.5%	2.9%	5.8%	6.8%	5.3%	1.2%	2.7%	0.0%	3.9%	1.2%	2.6%
2021	9	-4.2%	-6.7%	6.2%	5.9%	8.3%	4.8%	7.7%	6.5%	6.0%	4.5%	4.1%	1.2%	4.6%
2021	10	1.0%	5.9%	1.0%	11.8%	9.9%	10.3%	3.5%	3.3%	0.1%	0.9%	1.2%	-0.1%	2.4%
2021	11	10.1%	9.9%	1.0%	3.4%	2.5%	9.0%	6.4%	6.5%	4.5%	0.6%	5.7%	0.1%	3.9%
2021	12	2.2%	5.3%	-4.2%	3.1%	6.8%	6.9%	1.9%	2.0%	0.8%	-3.8%	0.4%	-3.7%	0.0%
2022	1	-10.4%	-9.8%	-2.6%	-9.7%	-8.4%	-4.2%	-14.3%	-12.3%	-14.7%	-17.4%	-20.7%	-23.4%	-16.8%
2022	2	7.5%	16.2%	-2.7%	-4.6%	-3.8%	3.6%	-8.1%	-5.0%	-11.5%	-13.8%	-16.0%	-18.6%	-11.7%
2022	3	10.3%	8.7%	-6.1%	4.4%	-8.3%	3.4%	-7.5%	-6.3%	-9.2%	-12.0%	-14.7%	-15.2%	-10.3%
2022	4	-28.2%	-1.4%	-5.6%	-5.0%	-2.0%	-3.8%	-8.9%	-7.3%	-8.9%	-10.9%	-9.5%	-12.1%	-9.0%
2022	5	-12.3%	6.4%	4.9%	-5.0%	2.5%	8.9%	-0.1%	1.6%	1.7%	2.4%	3.2%	1.4%	2.3%
2022	6	17.9%	16.5%	14.8%	8.1%	18.4%	17.0%	8.9%	9.2%	10.4%	7.5%	8.8%	9.3%	10.0%
2022	7	12.2%	5.1%	6.0%	8.8%	4.6%	11.0%	4.7%	5.5%	7.1%	2.8%	8.3%	6.9%	6.5%
2022	8	10.5%	7.8%	-4.9%	10.2%	17.1%	12.5%	0.2%	7.2%	4.5%	5.6%	10.8%	4.1%	7.1%
2022	9	-0.6%	10.4%	9.5%	8.1%	13.7%	16.9%	7.9%	5.2%	5.8%	6.7%	7.6%	7.5%	7.8%
2022	10	3.3%	5.0%	16.7%	9.2%	11.8%	10.1%	7.5%	6.3%	6.3%	6.4%	9.0%	7.4%	7.9%
2022	11	11.7%	6.7%	7.9%	11.9%	10.5%	13.2%	9.9%	6.8%	2.5%	1.8%	6.6%	1.9%	5.5%
2022	12	26.3%	6.1%	6.3%	4.1%	13.4%	12.4%	6.4%	3.7%	9.6%	-0.5%	7.5%	3.6%	5.9%
2023	1	-4.3%	1.2%	18.4%	3.9%	13.1%	16.2%	7.6%	7.8%	10.4%	2.2%	10.2%	-0.2%	7.1%
2023	2	17.2%	16.7%	7.2%	11.0%	2.7%	11.4%	5.1%	2.7%	0.9%	-3.2%	-2.1%	-7.4%	-0.4%
2023	3	9.1%	13.2%	8.0%	9.1%	3.8%	9.6%	0.6%	-1.6%	-5.8%	-4.6%	-4.5%	-6.8%	-2.7%
2023	4	3.4%	14.5%	4.6%	3.4%	5.3%	11.6%	1.3%	1.5%	-0.1%	-2.1%	-1.9%	-2.0%	0.2%
2023	5	-2.1%	30.0%	16.5%	16.2%	8.1%	16.9%	2.0%	8.5%	5.8%	6.8%	8.0%	3.2%	7.4%
2023	6	20.9%	11.0%	4.3%	10.2%	10.8%	16.4%	2.9%	5.5%	3.9%	4.4%	2.8%	4.4%	5.2%

Scale						
< -10%	< -5%	< 0%	> 0%	> 5%	>10%	>15%

Scientific Situational Awareness: Is There A Problem/Threat?

Gain of Function PRION Disorders & Long COVID

Cellular and Molecular Neurobiology (2023) 43:2621–2626
https://doi.org/10.1007/s10571-023-01342-8

REVIEW PAPER

Potential Prion Involvement in Long COVID-19 Neuropathology, Including Behavior

George B. Stefano[1] · Pascal Büttiker[1] · Simon Weissenberger[2] · Martin Anders[1] · Jiri Raboch[1] · Radek Ptacek[1] · Richard M. Kream[1]

Received: 3 March 2023 / Accepted: 22 March 2023 / Published online: 28 March 2023

Abstract
Prion' is a term used to describe a protein infectious particle responsible for several neurodegenerative diseases in mammals, e.g., Creutzfeldt-Jakob disease. The novelty is that it is protein based infectious agent not involving a nucleic acid genome as found in viruses and bacteria. Prion disorders exhibit, in part, incubation periods, neuronal loss, and induce abnormal folding of specific normal cellular proteins due to enhancing reactive oxygen species associated with mitochondria energy metabolism. These agents may also induce memory, personality and movement abnormalities as well as depression, confusion and disorientation. Interestingly, some of these behavioral changes also occur in COVID-19 and mechanistically include mitochondrial damage caused by SARS-CoV-2 and subsequenct production of reactive oxygen species. Taken together, we surmise, in part, long COVID may involve the induction of spontaneous prion emergence, especially in individuals susceptible to its origin may thus explain some of its manesfestions post-acute viral infection.

Keywords SARS-CoV-2 · COVID-19 · Long COVID · Prion · Prion disorders · Mitochondria · Confusion · Depression

Scientific Situational Awareness: Is There A Problem/Threat?

Gain of Function PRION Disorders & Long COVID

Cellular and Molecular Neurobiology (2023) 43:2621–2626
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REVIEW PAPER

Potential Prion Invc
Including Behavior

George B. Stefano[1] · Pascal
Radek Ptacek[1] · Richard M.

Received: 3 March 2023 / Accepted: 22

Abstract
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e.g., Creutzfeldt-Jakob disease
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mitochondrial damage caused
surmise, in part, long COVID
tible to its origin may thus exp

Keywords SARS-CoV-2 · COV

frontiers | Frontiers in Neuroscience

TYPE Opinion
PUBLISHED 27 September 2022
DOI 10.3389/fnins.2022.1002770

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SPECIALTY SECTION
This article was submitted to
Neurodegeneration,
a section of the journal

SARS-CoV-2, long COVID, prion disease and neurodegeneration

Yuhai Zhao[1,2], Vivian R. Jaber[2] and Walter J. Lukiw[2,3,4]*

[1]Department of Cell Biology and Anatomy, Louisiana State University Health Sciences Center, New Orleans, LA, United States, [2]LSU Neuroscience Center, LSU Health Sciences Center, New Orleans, LA, United States, [3]Department of Ophthalmology, LSU Health Sciences Center, New Orleans, LA, United States, [4]Department of Neurology, LSU Health Sciences Center, New Orleans, LA, United States

KEYWORDS

Creutzfeldt-Jacob disease (CJD), long COVID-19, 'S1' spike protein, SARS-CoV-2, prion disease (PrD), Alzheimer's disease (AD), miRNA-146a, miRNA-155

Scientific Situational Awareness: Is There A Problem/Threat?

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SPECIALTY SECTION
This article was submitted to
Neurodegeneration,
a section of the journal

SARS-CoV
disease an

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Orleans, LA, United States, [2]LSU
LA, United States, [3]Department
United States, [4]Department of
United States

KEYWORDS

Creutzfeldt-Jacob disease
prion disease (PrD), Alzhei

A Potential Role of the Spike Protein in Neurodegenerative Diseases: A Narrative Review

Stephanie Seneff [1] , Anthony M. Kyriakopoulos [2] , Greg Nigh [3] , Peter A. McCullough [4]

1. Computer Science and Artificial Intelligence Laboratory, Massachusetts Institute of Technology, Cambridge, USA 2. Reasearch and Development, Nasco AD Biotechnology Laboratory, Piraeus, GRC 3. Naturopathy, Immersion Health, Portland, USA 4. Internal Medicine, Truth for Health Foundation, Tucson, USA

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Abstract

Human prion protein and prion-like protein misfolding are widely recognized as playing a causal role in many neurodegenerative diseases. Based on in vitro and in vivo experimental evidence relating to prion and prion-like disease, we extrapolate from the compelling evidence that the spike glycoprotein of SARS-CoV-2 contains extended amino acid sequences characteristic of a prion-like protein to infer its potential to cause neurodegenerative disease. We propose that vaccine-induced spike protein synthesis can facilitate the accumulation of toxic prion-like fibrils in neurons. We outline various pathways through which these proteins could be expected to distribute throughout the body. We review both cellular pathologies and the expression of disease that could become more frequent in those who have undergone mRNA vaccination. Specifically, we describe the spike protein's contributions, via its prion-like properties, to neuroinflammation and neurodegenerative diseases; to clotting disorders within the vasculature; to further disease risk due to suppressed prion protein regulation in the context of widely prevalent insulin resistance; and to other health complications. We explain why these prion-like characteristics are more relevant to vaccine-related mRNA-induced spike proteins than natural infection with SARS-CoV-2. We note with an optimism an apparent loss of prion-like properties among the current Omicron variants. We acknowledge that the chain of pathological events described throughout this paper is only hypothetical and not yet verified. We also acknowledge that the evidence we usher in, while grounded in the research literature, is currently largely circumstantial, not direct. Finally, we describe the implications of our findings for the general public, and we briefly discuss public health recommendations we feel need urgent consideration.

Scientific Situational Awareness: Is There A Problem/Threat?

ARTICLE

https://doi.org/10.1038/s41467-021-25855-2 OPEN

nature COMMUNICATIONS

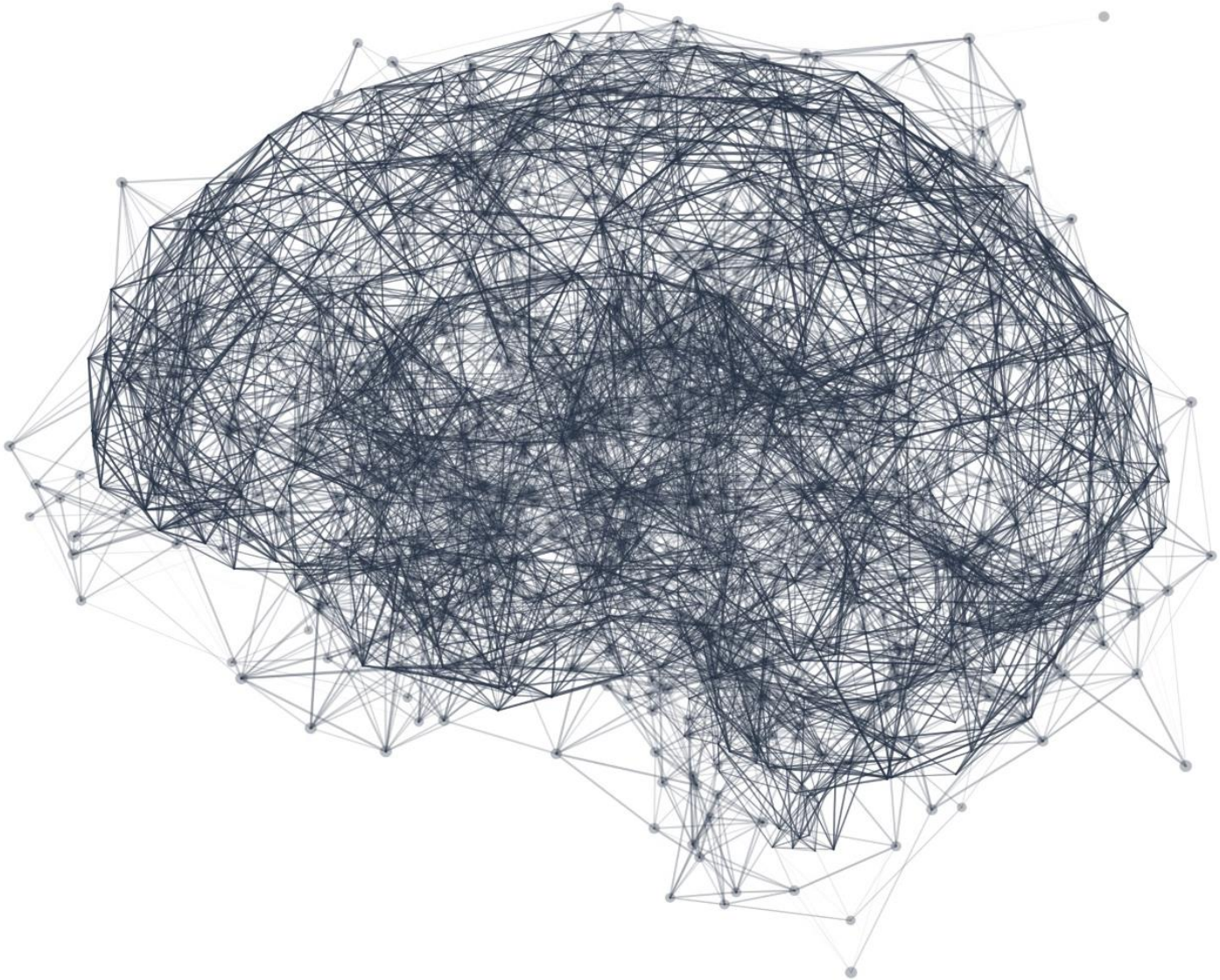
Check for up

Highly efficient intercellular spreading of protein misfolding mediated by viral ligand-receptor interactions

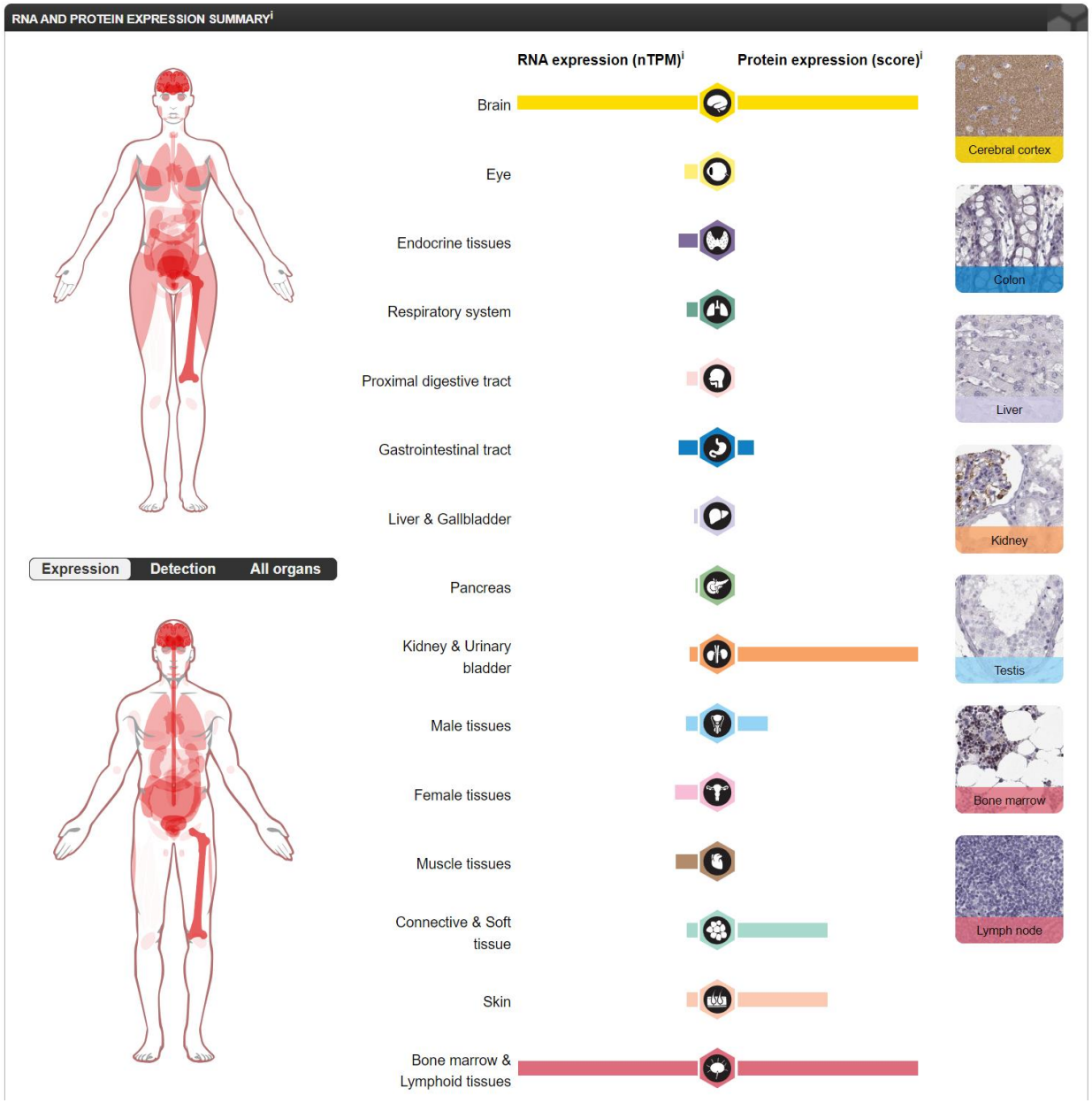
Shu Liu[1,10], André Hossinger[1], Stefanie-Elisabeth Heumüller[1], Annika Hornberger[1], Oleksandra Buravlova[1], Katerina Konstantoulea[2,3], Stephan A. Müller[4,5], Lydia Paulsen[1], Frederic Rousseau[2,3], Joost Schymkowitz[2,3], Stefan F. Lichtenthaler[4,5,6], Manuela Neumann[7,8], Philip Denner[1] & Ina M. Vorberg

Protein aggregates associated with neurodegenerative diseases have the ability to transmit to unaffected cells, thereby templating their own aberrant conformation onto soluble homotypic proteins. Proteopathic seeds can be released into the extracellular space, secreted in association with extracellular vesicles (EV) or exchanged by direct cell-to-cell contact. The extent to which each of these pathways contribute to the prion-like spreading of protein misfolding is unclear. Exchange of cellular cargo by both direct cell contact or via EV depends on receptor-ligand interactions. We hypothesized that enabling these interactions through viral ligands enhances intercellular proteopathic seed transmission. Using different cellular models propagating prions or pathogenic Tau aggregates, we demonstrate that vesicular stomatitis virus glycoprotein and SARS-CoV-2 spike S increase aggregate induction by cell contact or ligand-decorated EV. Thus, receptor-ligand interactions are important determinants of intercellular aggregate dissemination. Our data raise the possibility that viral infections contribute to proteopathic seed spreading by facilitating intercellular cargo transfer.

SARS-CoV-2 Neuroscience: NHP Alpha-Synuclein Pathology



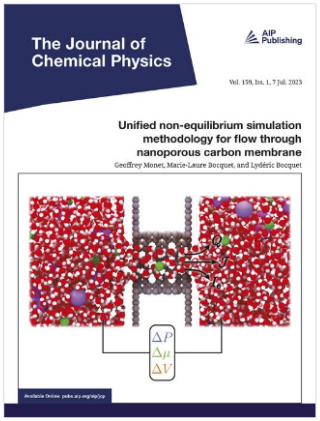
SARS-CoV-2 Neuroscience: NHP Alpha-Synuclein Distribution



SARS-CoV-2 Neuroscience: Alpha-Synuclein Amyloidogenesis

AIP Publishing The Journal of Chemical Physics

Volume 159, Issue 1
3 July 2023



Previous Article

RESEARCH ARTICLE | JULY 06 2023

SARS-COV-2 spike protein fragment eases amyloidogenesis of α-synuclein

Andrew D. Chesney ; Buddhadev Maiti ; Ulrich H. E. Hansmann

+ Author & Article Information

J. Chem. Phys. 159, 015103 (2023)
https://doi.org/10.1063/5.0157331 **Article history**

Parkinson's disease is accompanied by the presence of amyloids in the brain that are formed of α-synuclein chains. The correlation between COVID-19 and the onset of Parkinson's disease led to the idea that amyloidogenic segments in SARS-COV-2 proteins can induce aggregation of α-synuclein. Using molecular dynamic simulations, we show that the fragment FKNIDGYFKI of the spike protein, which is unique for SARS-COV-2, preferentially shifts the ensemble of α-synuclein monomer toward rod-like fibril seeding conformations and, at the same time, differentially stabilizes this polymorph over the competing twister-like structure. Our results are compared with earlier work relying on a different protein fragment that is not specific for SARS-COV-2.

SARS-CoV-2 Neuroscience: Insights From Nonhuman Primates

viruses

MDPI

Article

Brain Inflammation and Intracellular α-Synuclein Aggregates in Macaques after SARS-CoV-2 Infection

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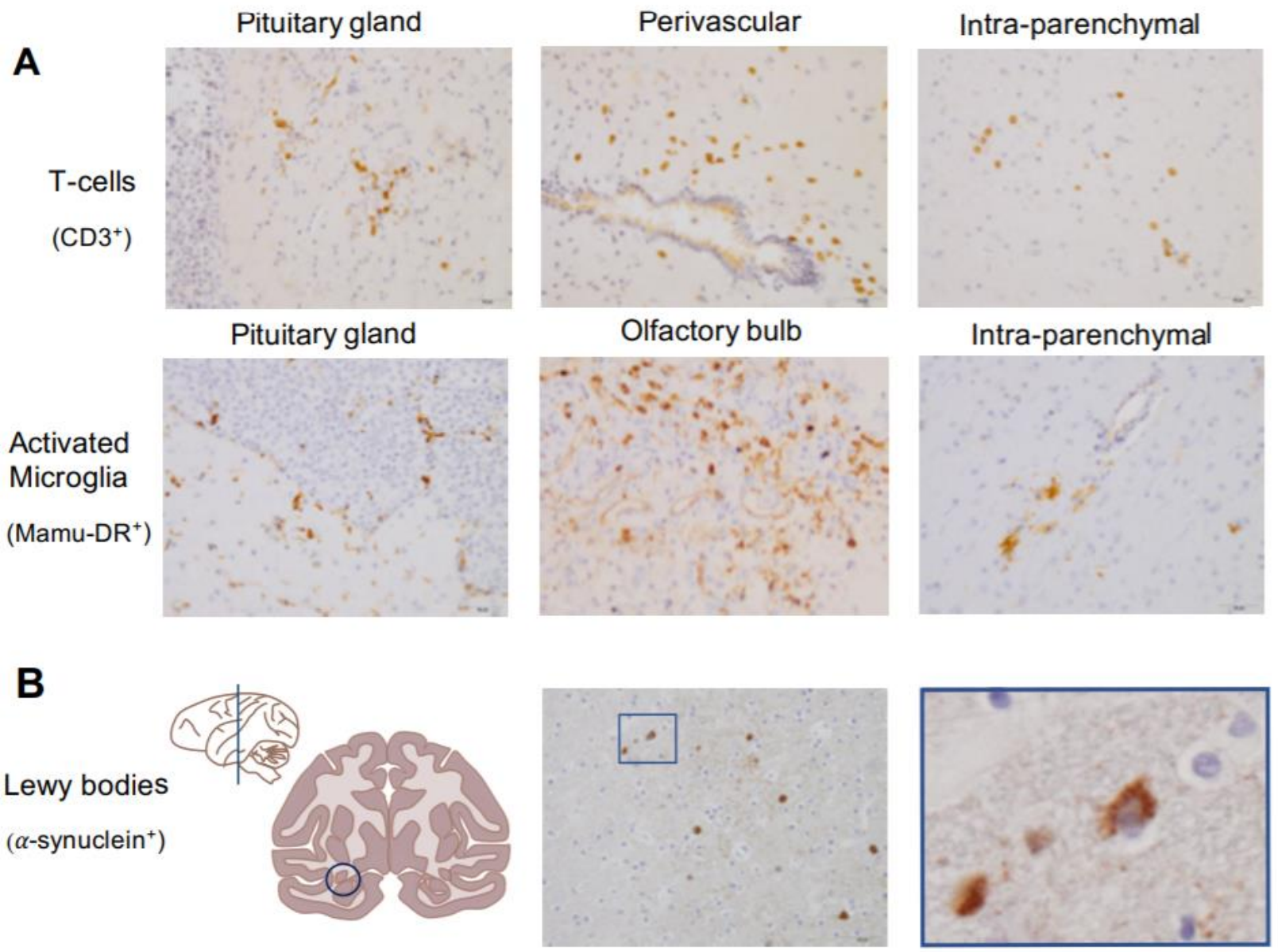
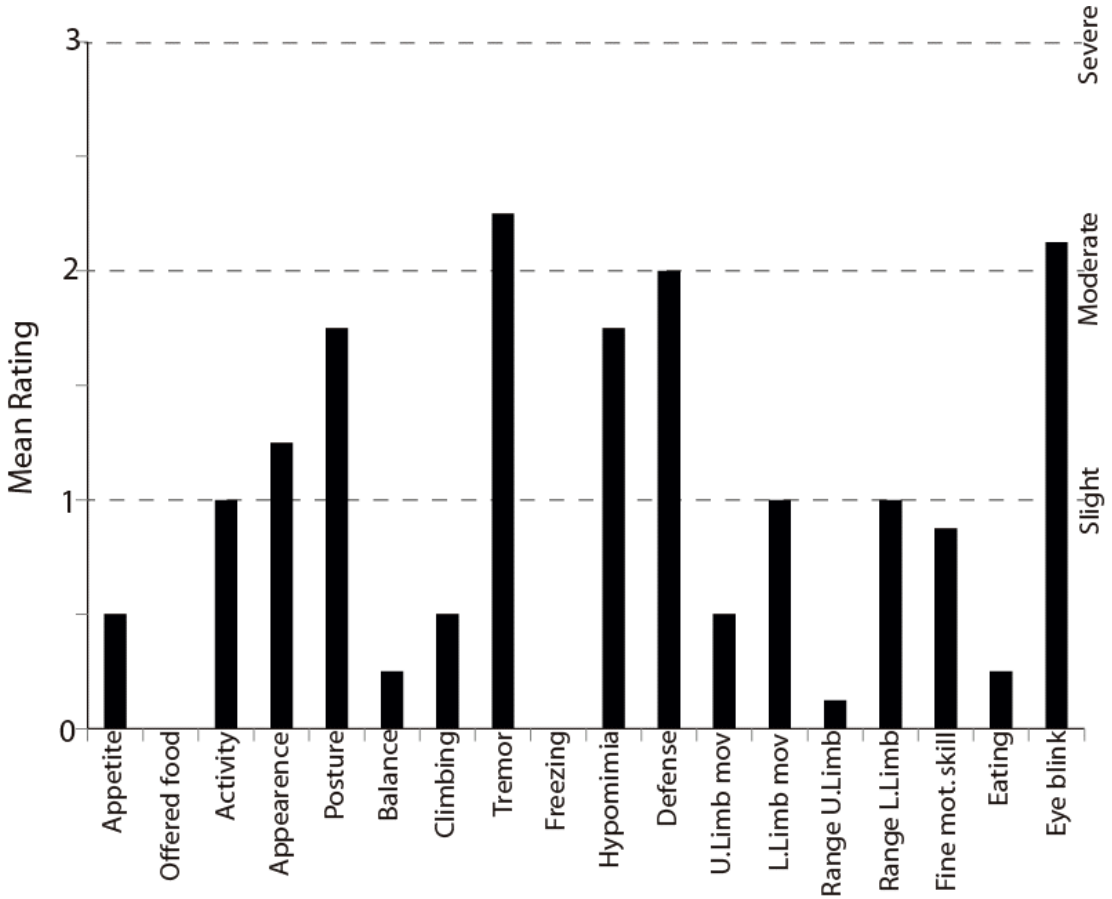
SARS-CoV-2 Neuroscience: Insights From Nonhuman Primates



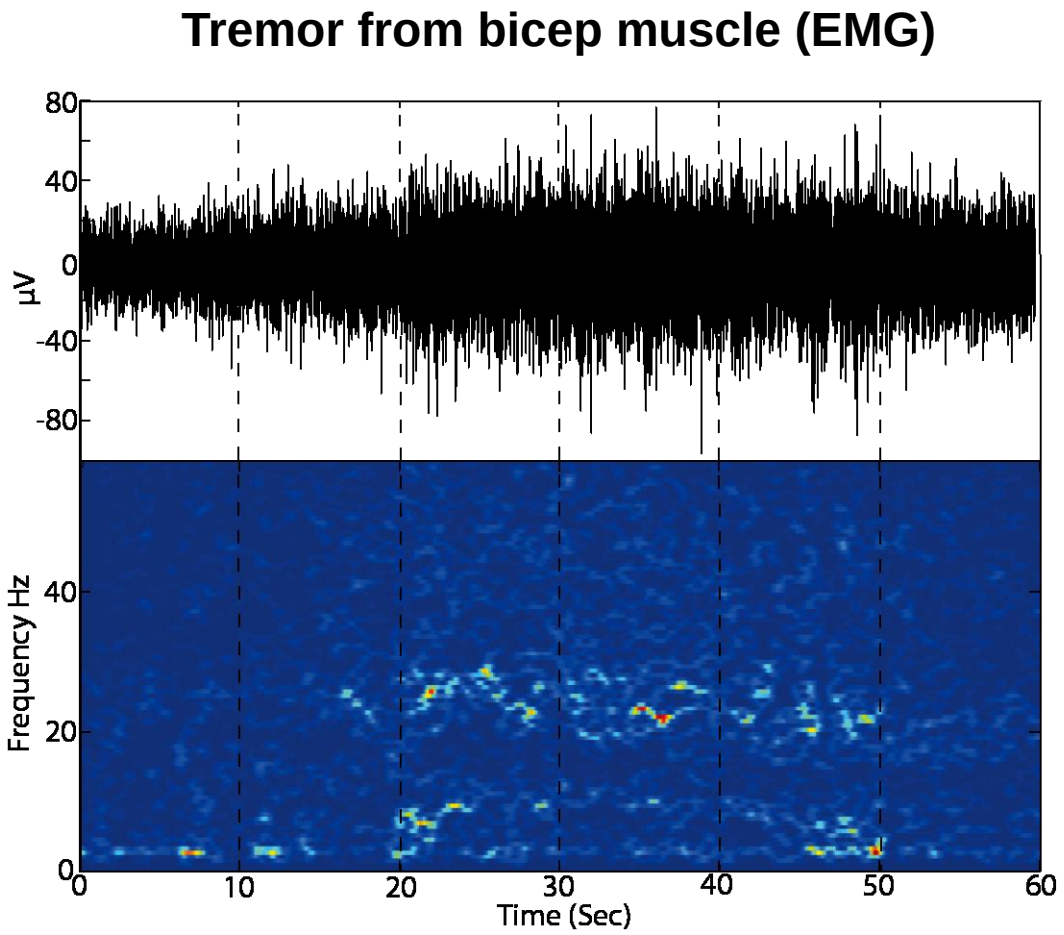
World's First In Captivity NHP Early Onset Parkinsonism

Daphne (Mfy 1823) – Kyoto Primate Research Institute (Early Onset MSA)

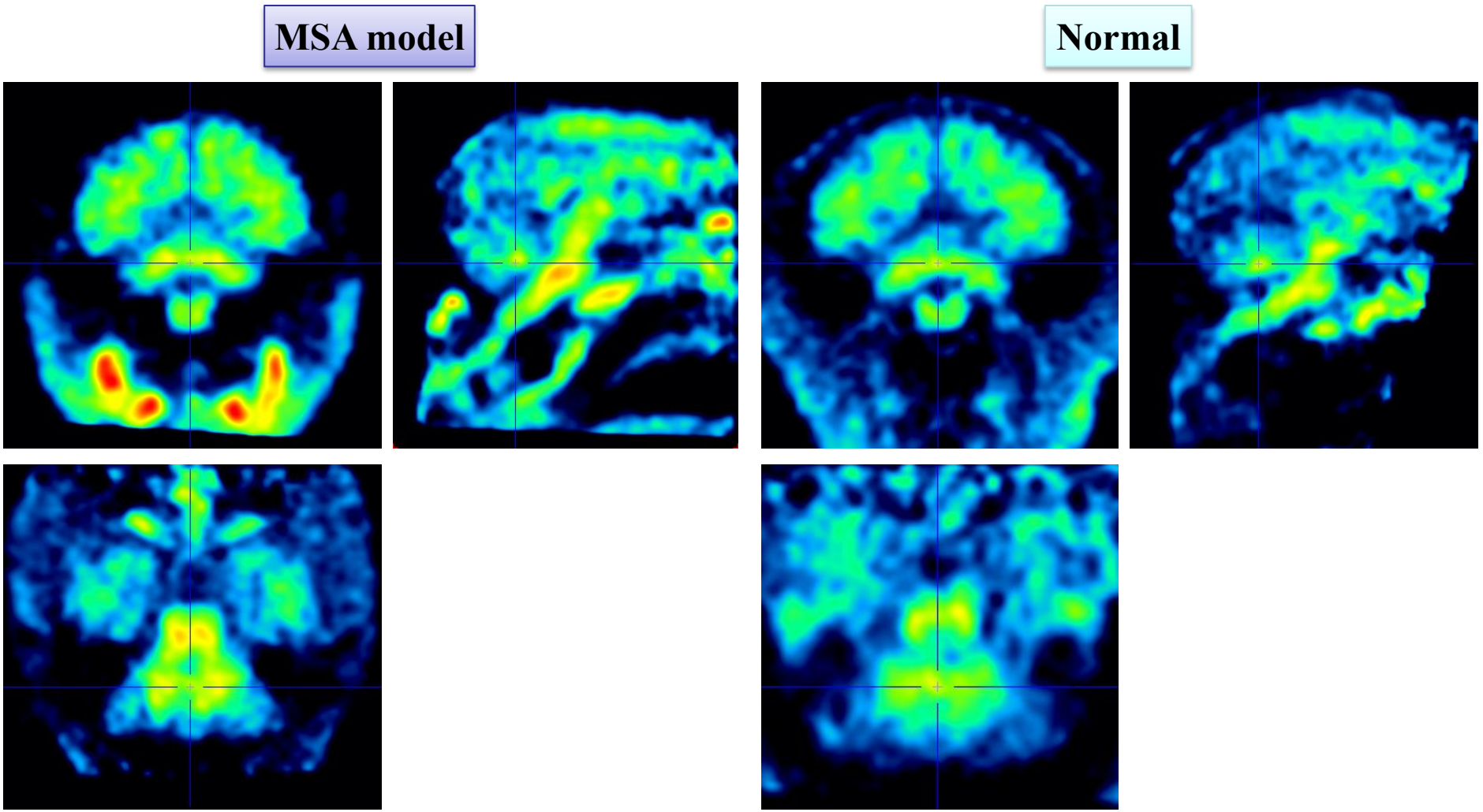
World's First In Captivity NHP Early Onset Parkinsonism



World's First In Captivity NHP Early Onset Parkinsonism



Alpha Synuclein PET Radioactive Ligand Binding

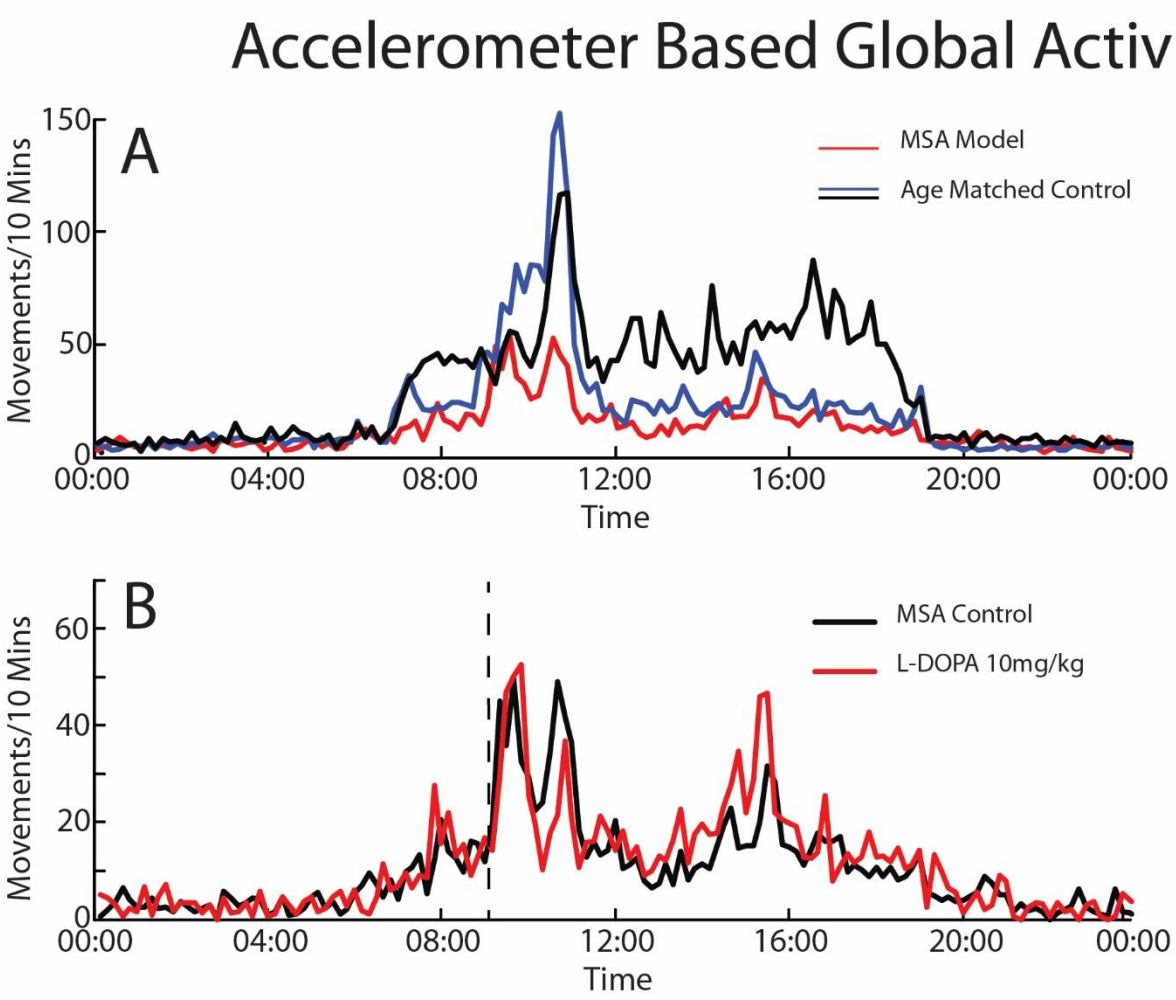


- [^{11}C]BF227 (SUV, summation 30-90min)

SUV

0.5 2

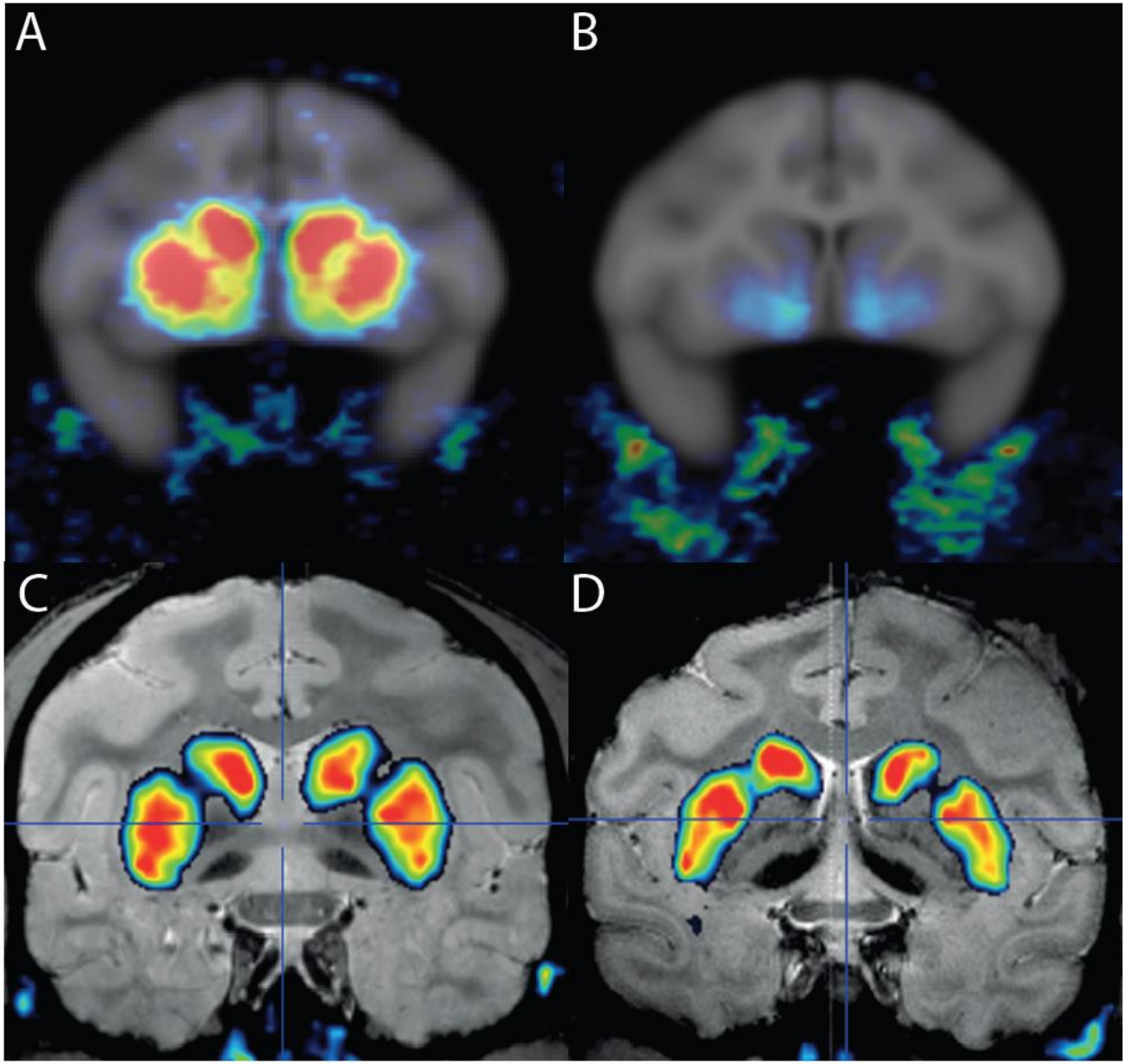
L-DOPA Challenge Confirmed Multisystem Atrophy



A. Use of an acceleromoter which continuously monitored activity over 7 days found that the MSA monkey had significantly reduced activity, especially in the morning compared to age and sex matched controls.

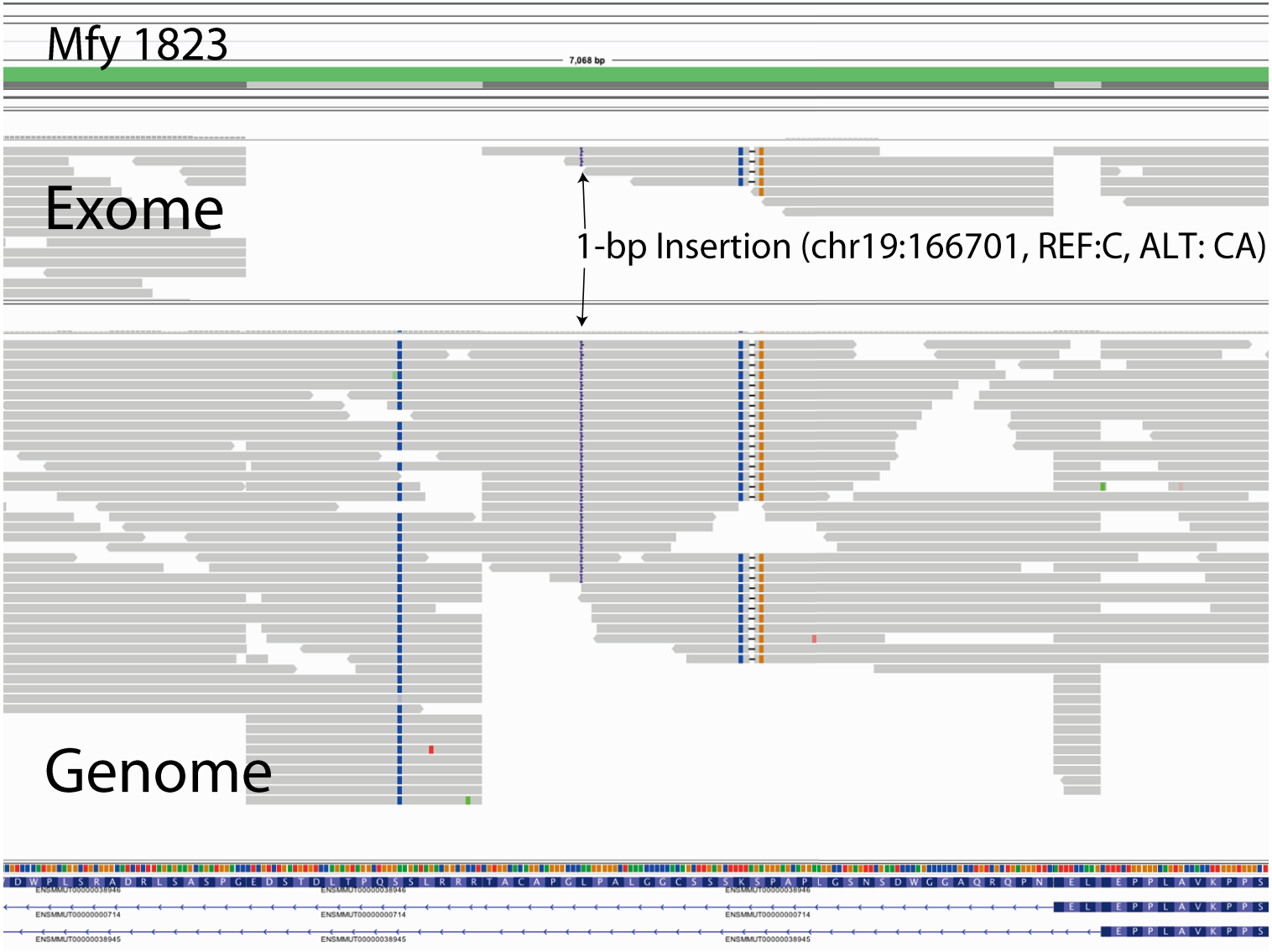
B.The MSA monkey was unresponsive to challenge with L-DOPA.

PET Raclopride Challenge Confirmed Intact DA Signaling



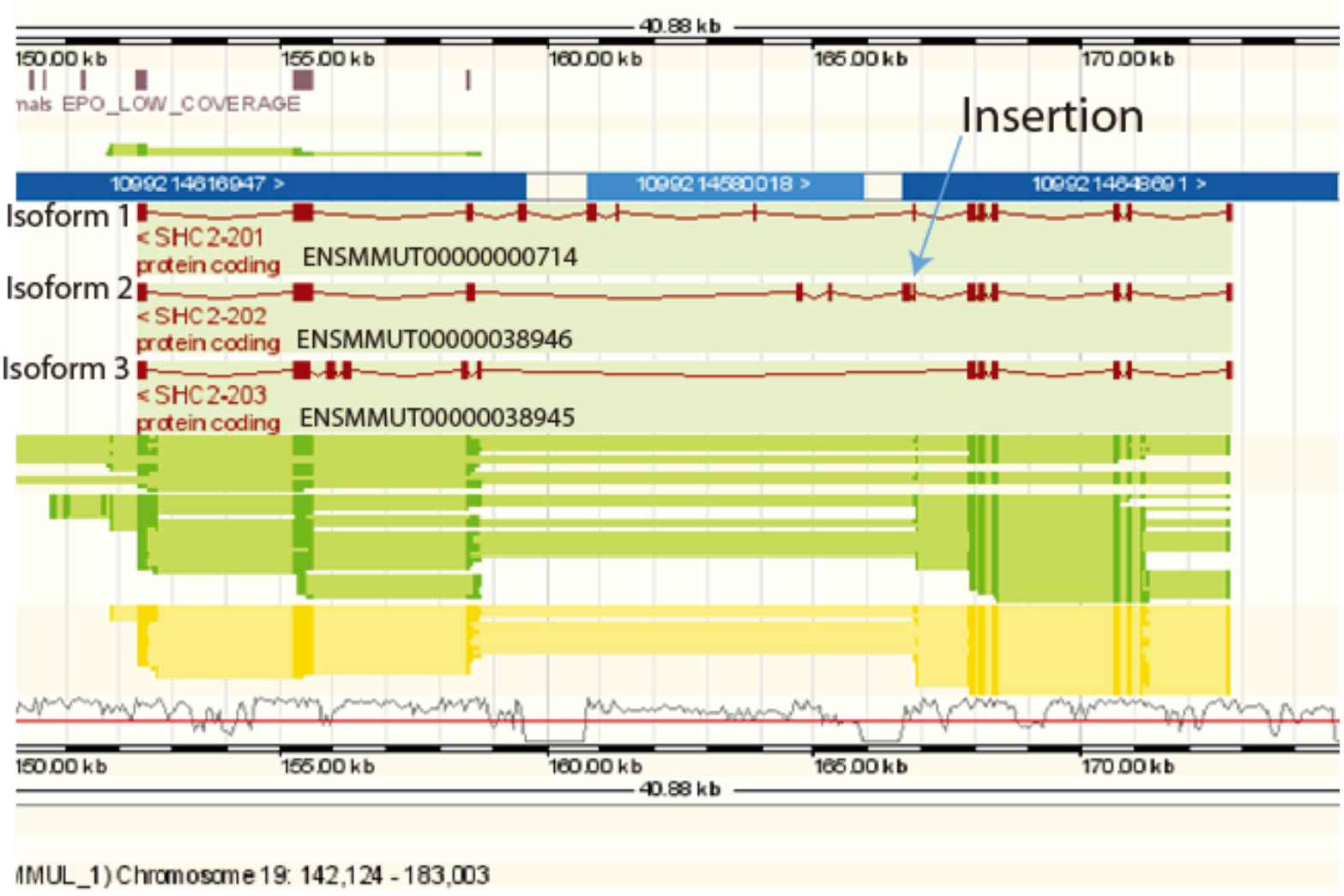
Next Generation Genome/Exome Sequencing

Next Generation Sequencing vs n = 100 NHP Control Group

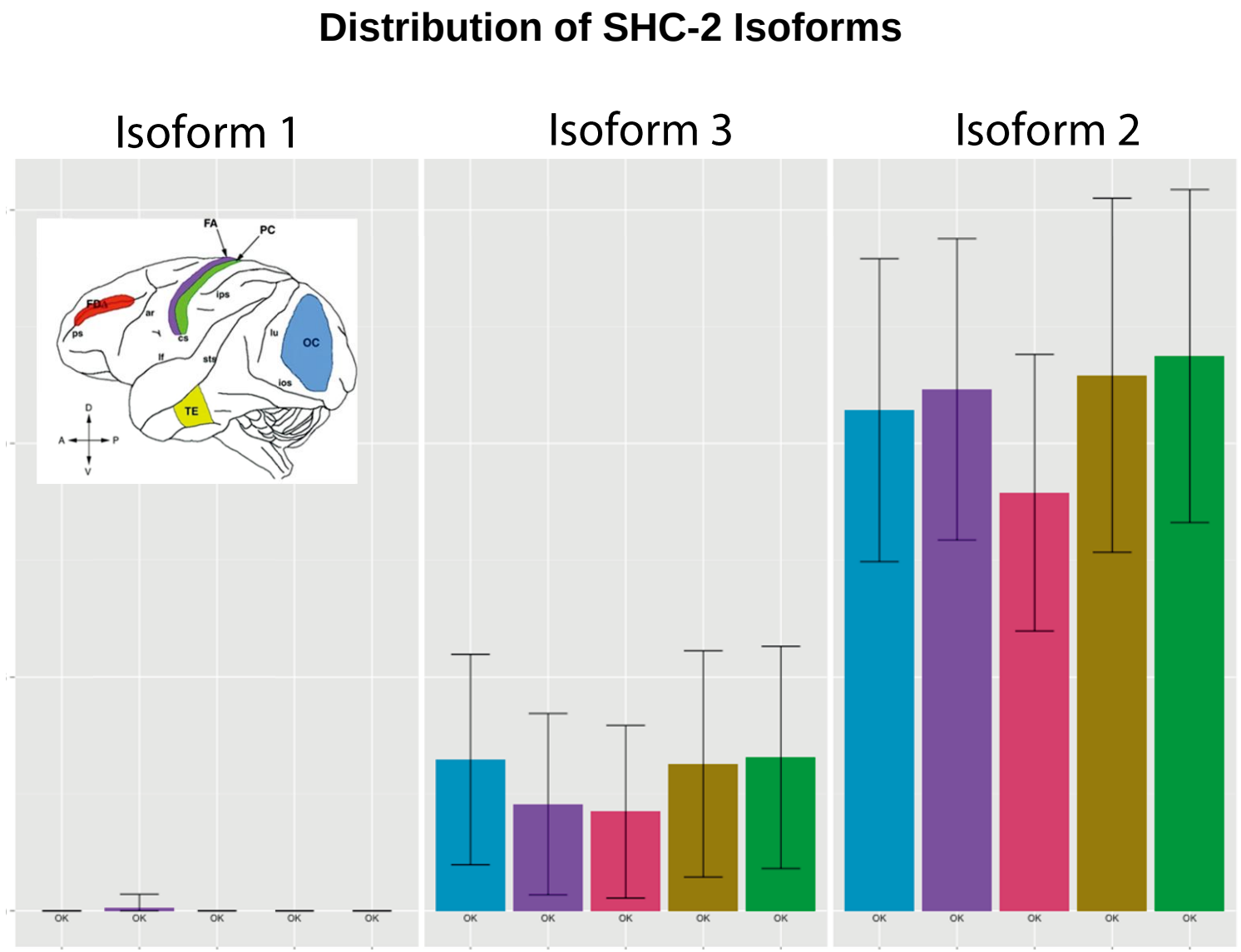


Next Generation Genome/Exome Sequencing

Identification of SHC-2 Isoforms

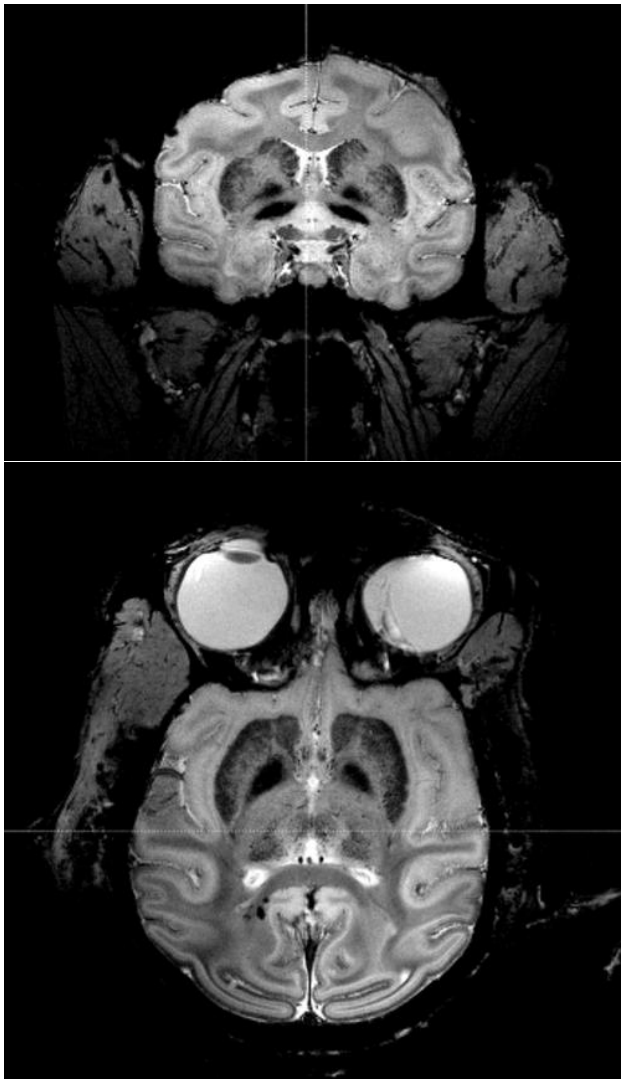


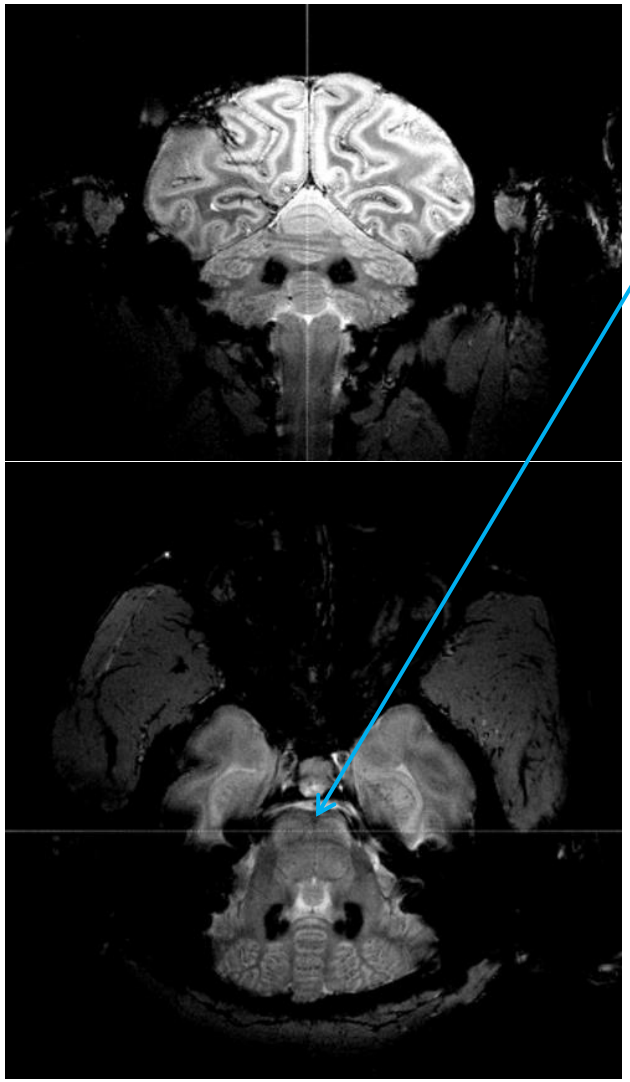
Next Generation Genome/Exome Sequencing



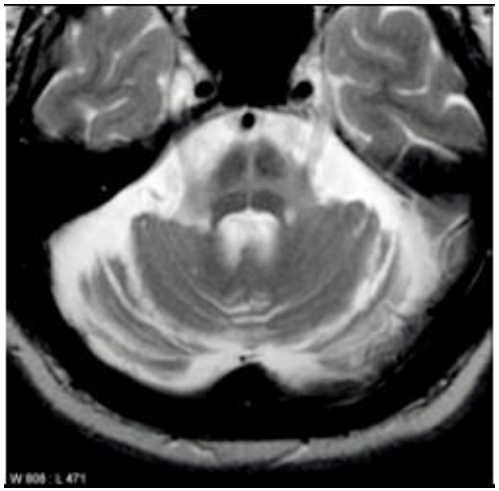
Daphne Showed Classic Pontocerebellar Tract Degeneration

High-Intensity 7-Tesla MRI





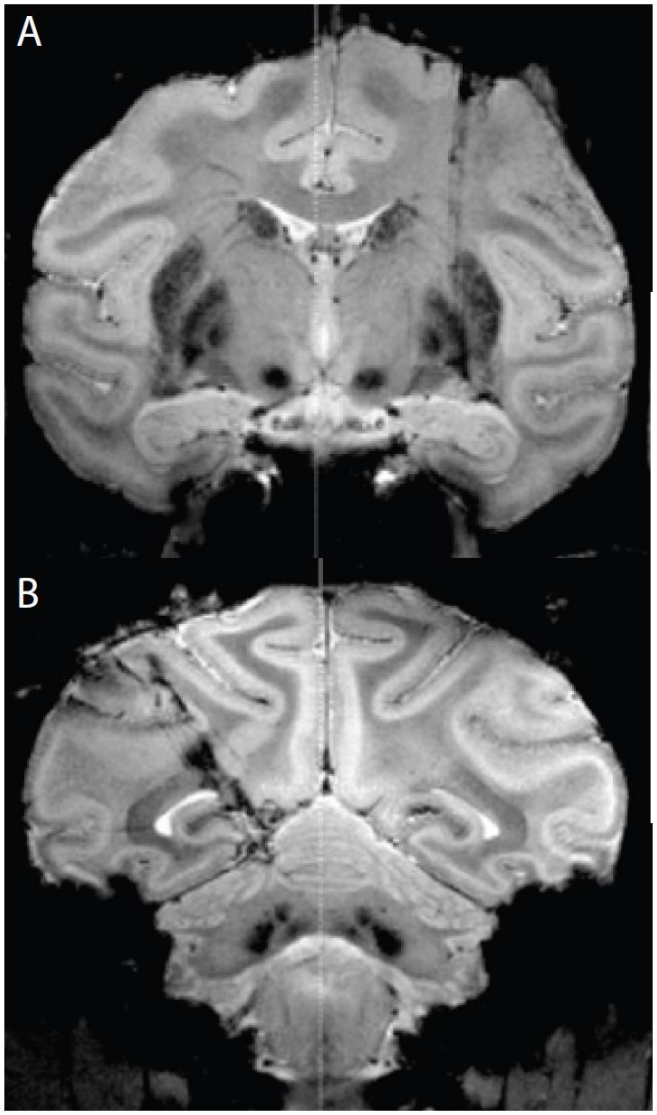
Hot cross bun sign?



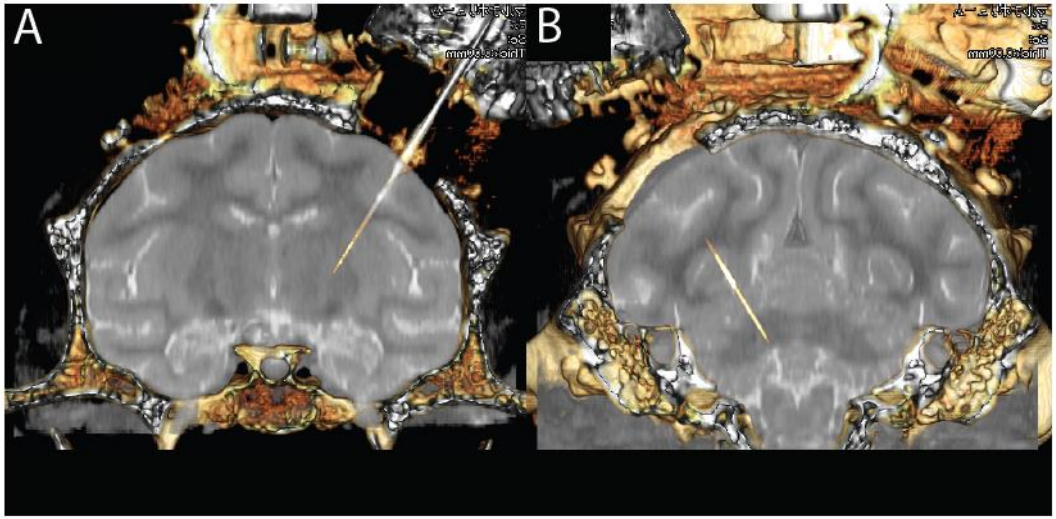
MSA Human

A. Shrivastava Radiology vol. 245, 2007

Strong T2 Hypointensity Was Prominent In All Basal Ganglia Cerebellar Nuclei



Iron Deposition Was Prominent in MSA



Synthetic Parkinsonism(MPTP) vs. Organic Parkinsonism?

Collection of follicle eggs

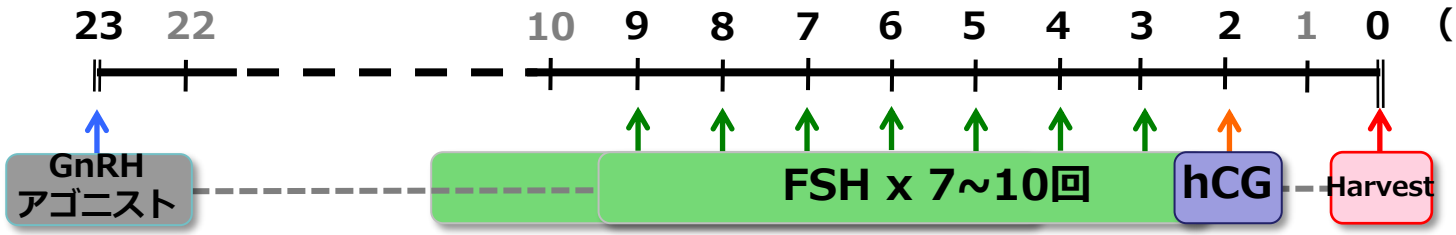
・ Follicle stimulation treatment

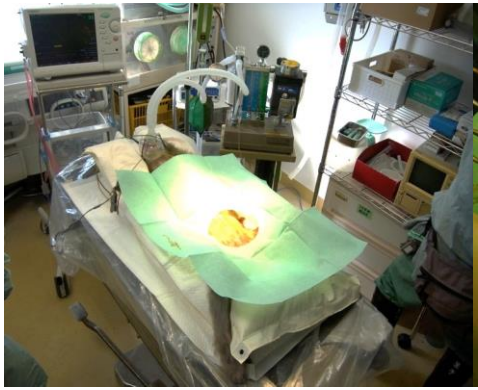
① Subcutaneous administration of 0.9 mg / head of GnRH agonist (leuprorelin) during the resting period (before and after menstruation)

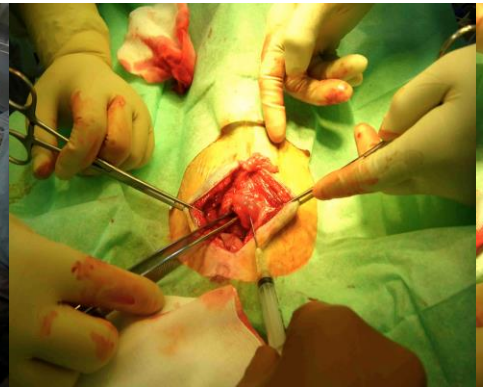
② From 2 weeks later, 5-20 IU / kg FSH (Gonapur) is intramuscularly administered for 7 to 10 consecutive days.

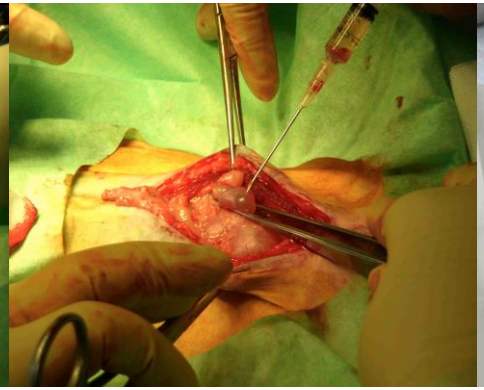
③ Intramuscular administration of 400 IU / kg hCG (gonatropin) on the day after the final day of FSH administration

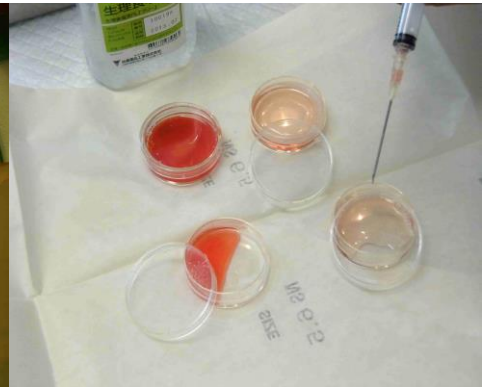
④ Suction collection from ovarian follicles 36-40 hours after hCG administration



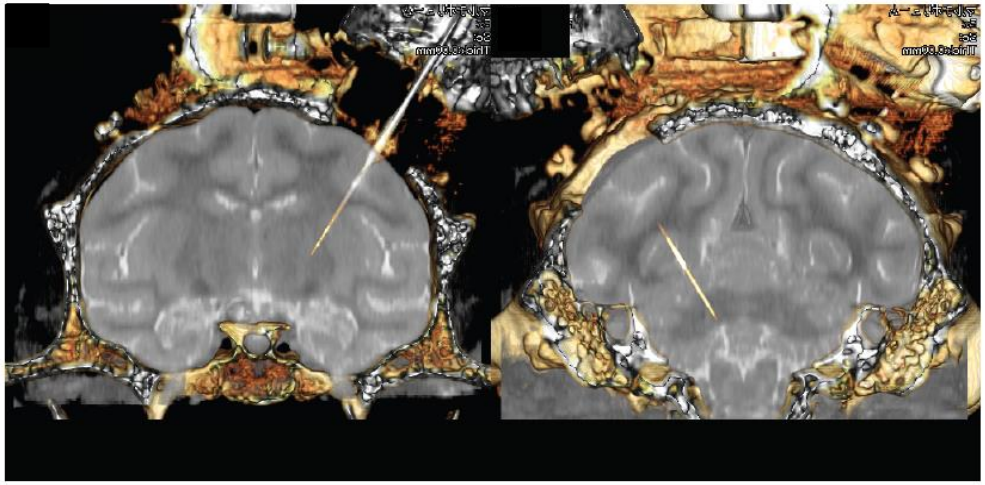




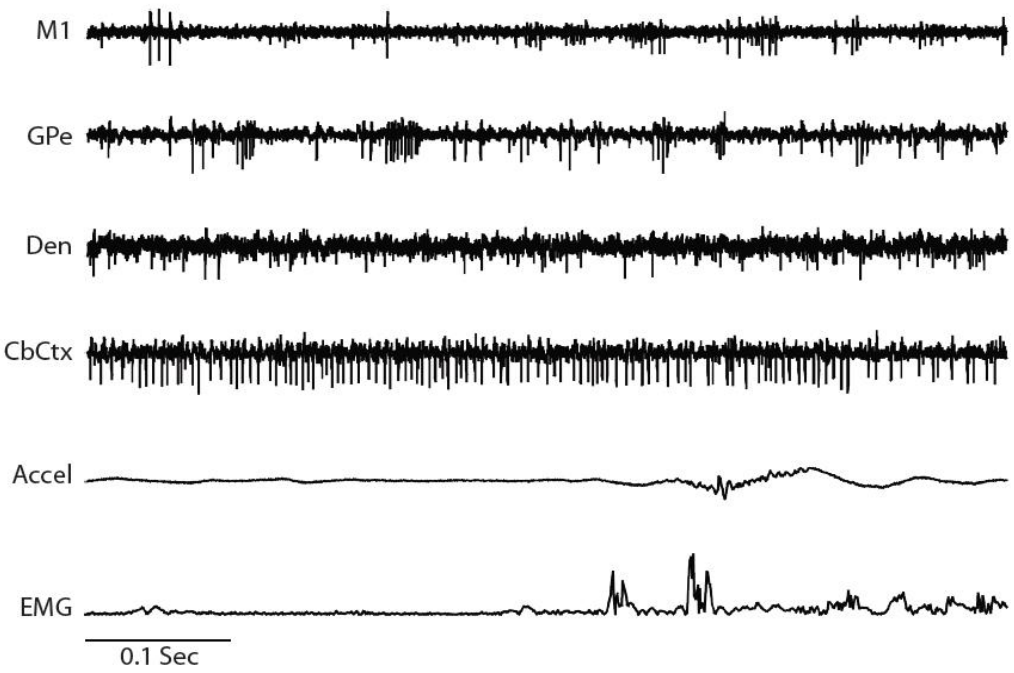




Synthetic Parkinsonism(MPTP) vs. Organic Parkinsonism?

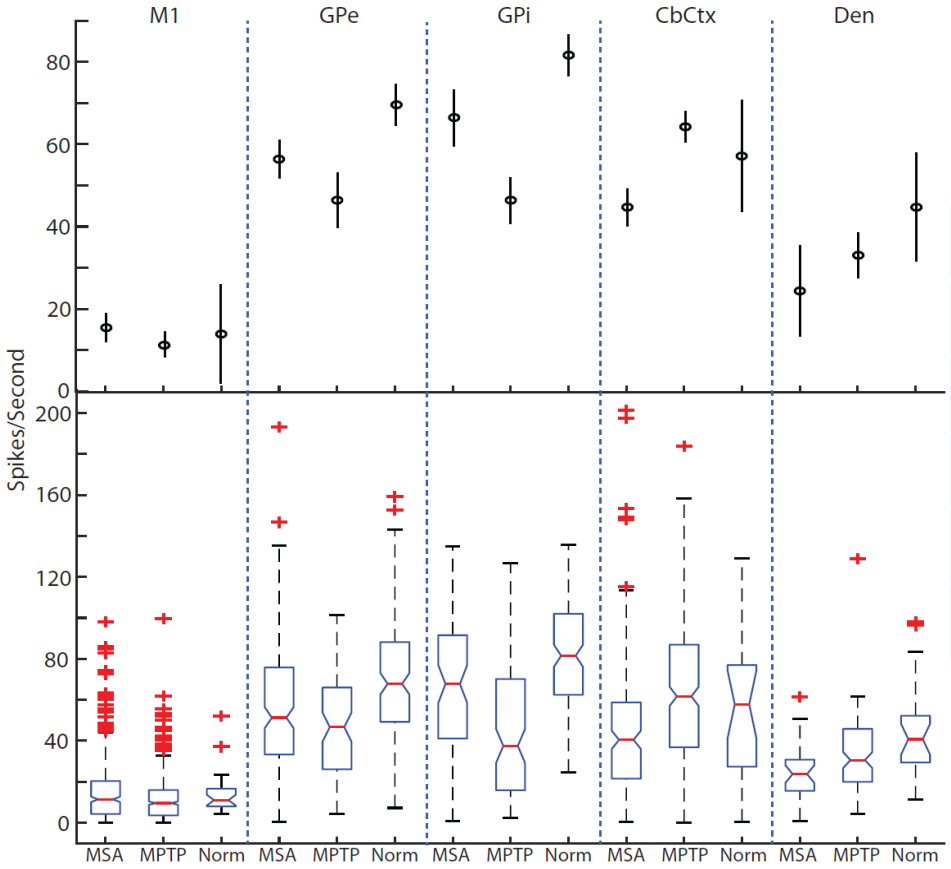


Similar recording methods, for both MPTP & SHC2 mutant, as the NHP-TS model, focusing on simultaneous recording from cortico-basal ganglia-cerebella networks.



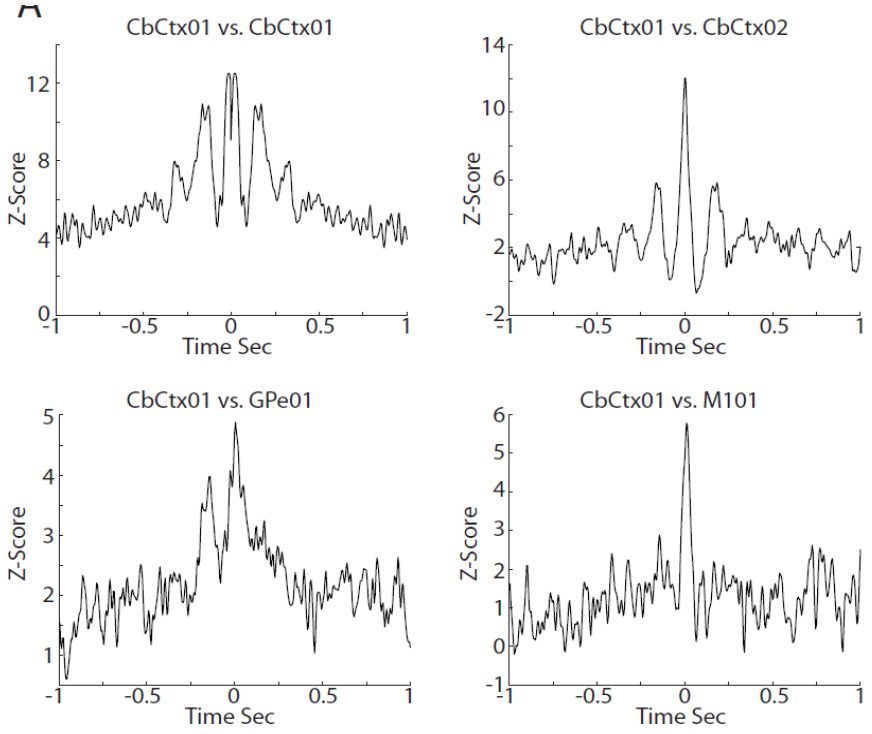
Attempted to differentiate periods of inactivity with spontaneous movement, with use of Accelerometers and EMG recording .

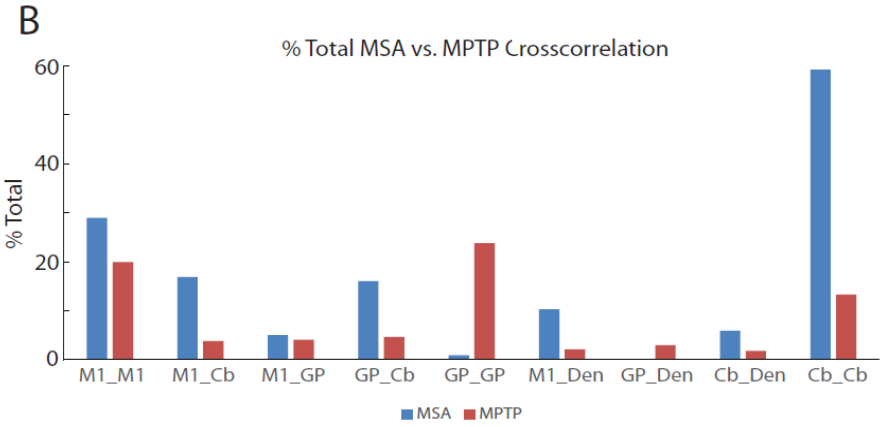
Synthetic Parkinsonism(MPTP) vs. Organic Parkinsonism?



Location	n = Norm	Mean / SD	n = MPTP	Mean / SD	n = MSA	Mean / SD
M1	35	13.9 / 10.4	141	11.3 / 16.3	80	15.3 / 15.4
GPe	41	69.6 / 28.8	88	46.5 / 25.1 *	99	56.1 / 32.0 *
GPi	32	81.6 / 23.6	45	41.6 / 33.8 *	44	64.3 / 32.4 *
CbCtx	25	57.1 / 36.7	117	61.3 / 32.6	81	48.2 / 33.5
Den	23	44.7 / 24.5	45	30.7 / 16.87	9	23.2 / 14.0

What About Hypokinetic Conditions?

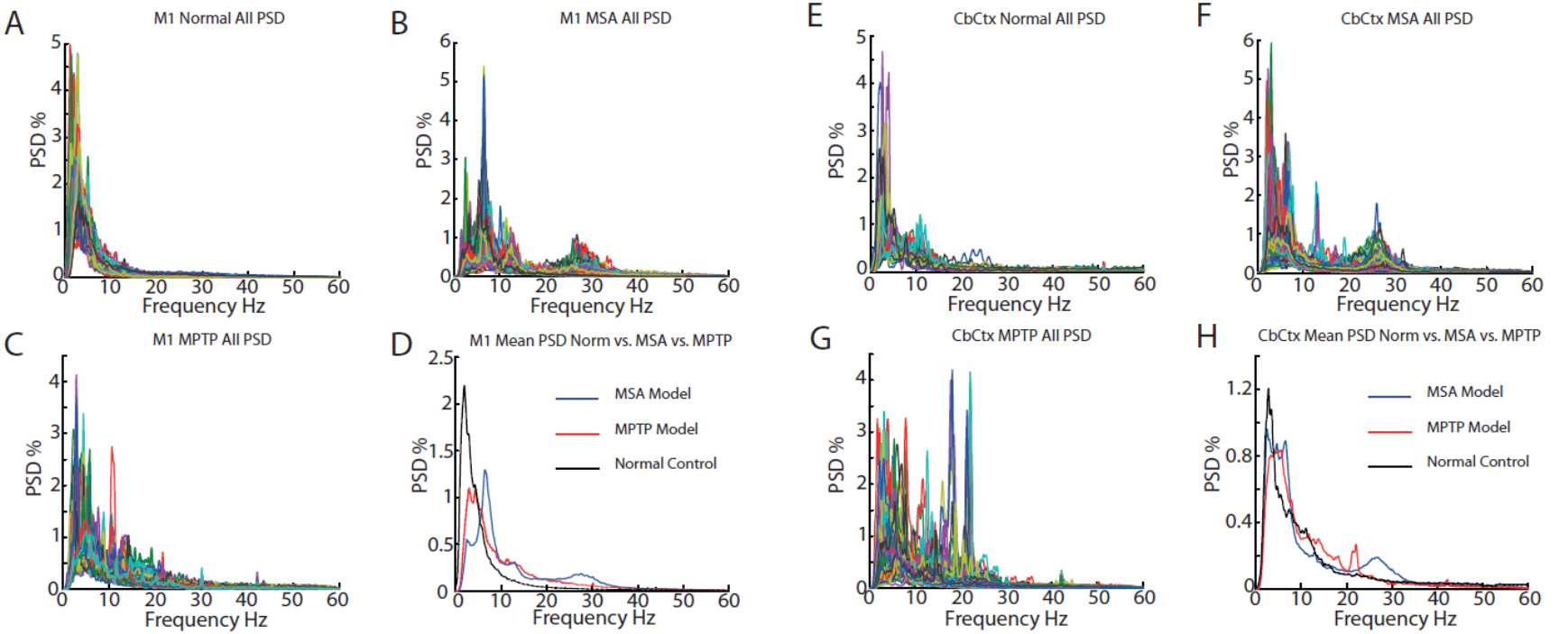




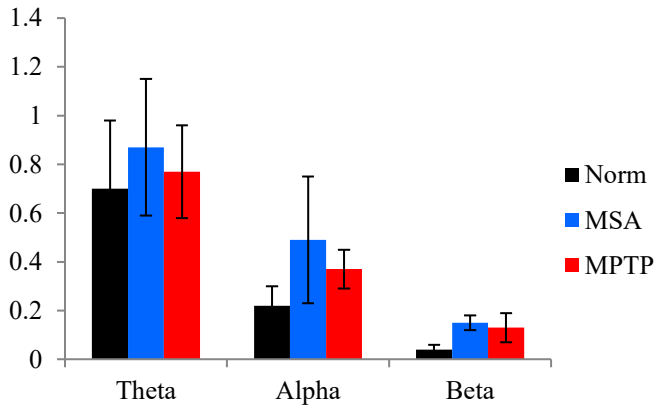
Location	n = Pairs (MPTP)	% Sig Corel (MPTP)	n = Pairs (MSA)	% Sig Corel (MSA)	MSA vs. MPTP Z-Value / P-Value
M1_M1	402	19.9	159	28.9	2.3 / 0.02 *
M1_Cb	793	3.8	243	16.9	7.1 / < 0.001 *
M1_GP	421	4.0	322	5.0	0.6 / 0.55
GP_Cb	544	4.6	175	16.0	5 / < 0.001 *
GP_GP	164	23.8	120	1.0	5.5 / < 0.001 *
M1_Den	146	2.1	39	10.2	2.4 / 0.016 *
GP_Den	274	3.0	24	0	0.7 / 0.48
Cb_Den	172	1.7	17	6.0	1.2 / 0.25
Cb_Cb	158	13.3	54	59.3	6.7 / < 0.001 *

Synthetic Parkinsonism(MPTP) vs. Organic Parkinsonism?

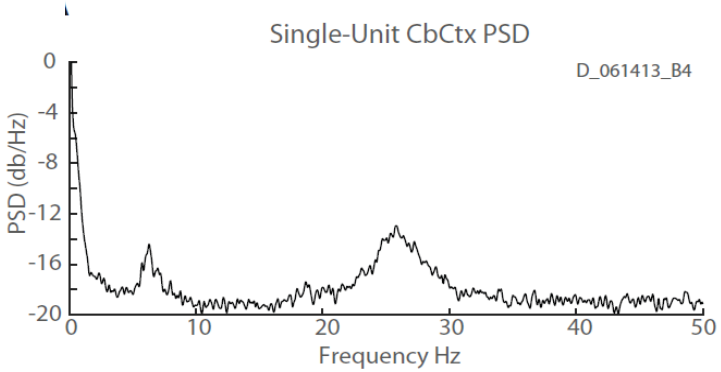
Motor Cortex

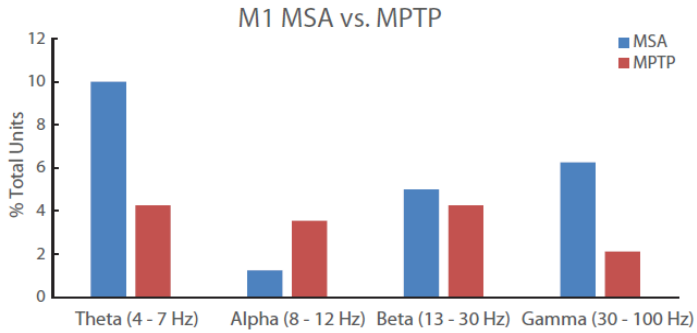


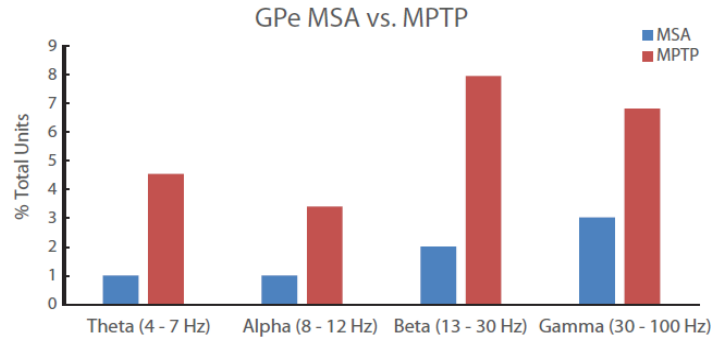
Cerebellar Cortex

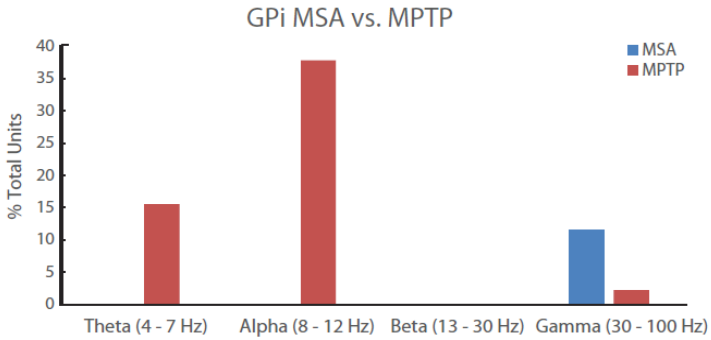


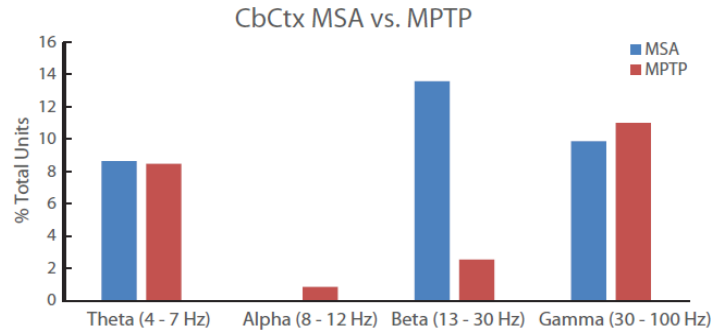
Synthetic Parkinsonism(MPTP) vs. Organic Parkinsonism?

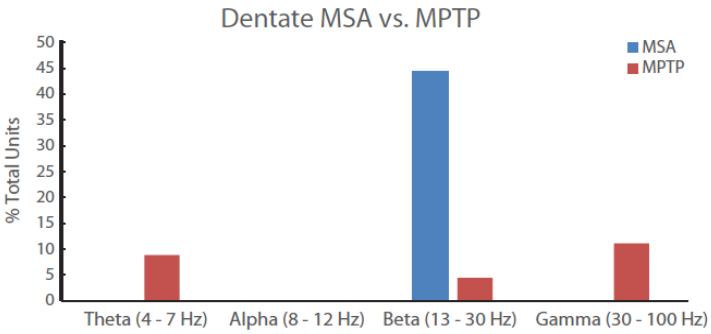












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Awareness. Research. Support.

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NIRS

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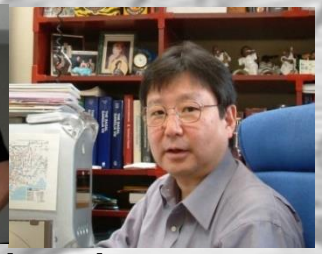
Prof. Masaki Isoda



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