ALPSTEINACADEMY

Continuous Professional Development

Seminars & Webinars

1

ALPSTEINCLINIC

WELCOME

Post Covid-19 (vacc) Syndrome

Ralf Oettmeier, MD, Gais / AR, Switzerland

2

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View out from my window …

3

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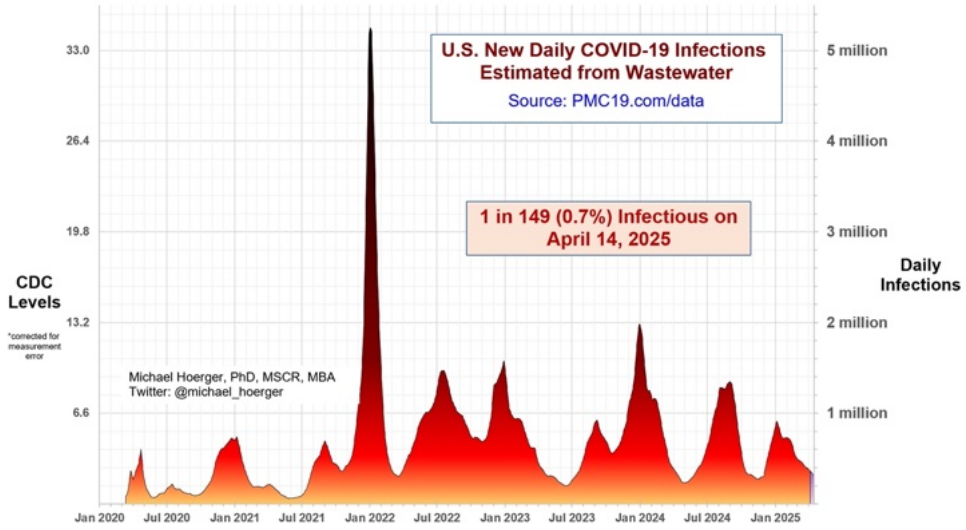
Permanent (re)-infection with Covid-19



- Approx. 5 Mio. infections per week
- 1.6% of the population is estimated to be actively infectious
- Approx. 200.000 develop Covid-associated conditions a week

4

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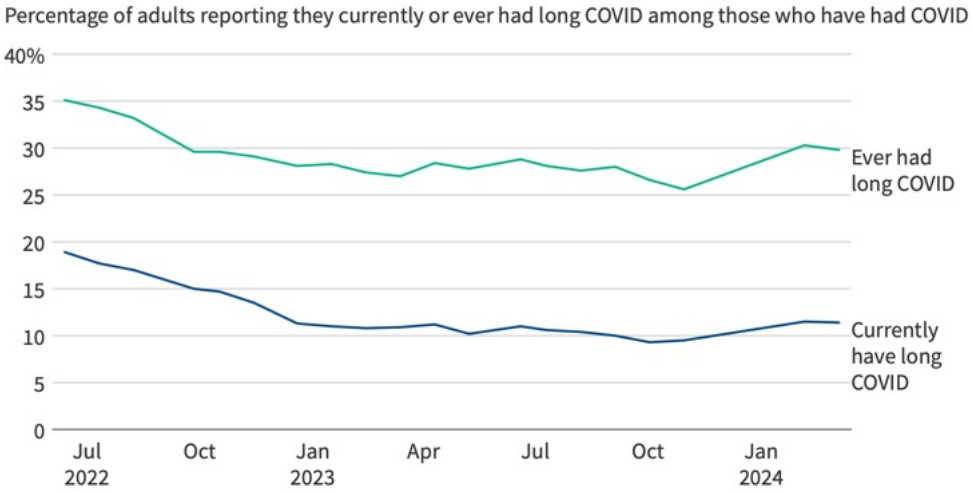
Around 3 in 10 Adults Who Have Had COVID Report Getting Long COVID



Source: https://www.kff.org/coronavirus-covid-19/issue-brief/as-recommendations-for-isolation-end-how-common-is-long-covid/

5

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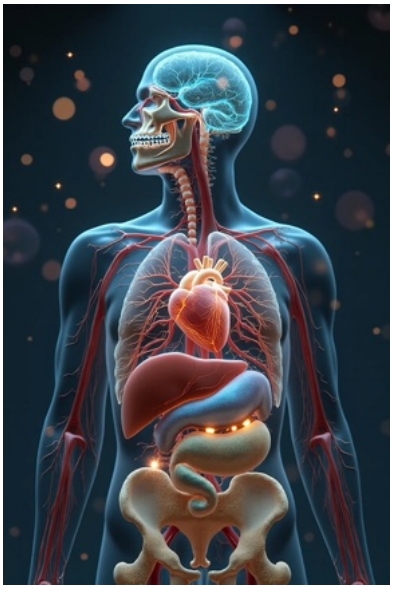
Post Covid (vacc) Syndrome

- Neurologic and mental health
- Cardiovascular conditions
- Respiratory conditions
- Blood clots and vascular issues
- Musculoskeletal conditions
- Kidney failure
- Chronic fatigue
- Etc.



6

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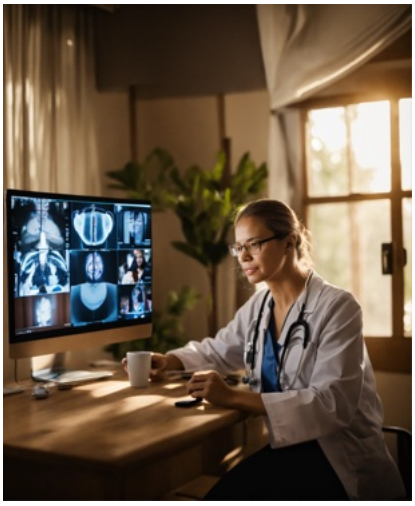
Post Covid-19 (vacc) Syndrome

What is the scientific background?

NEWS from research …



7

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Dual infection: human cells and bacteria

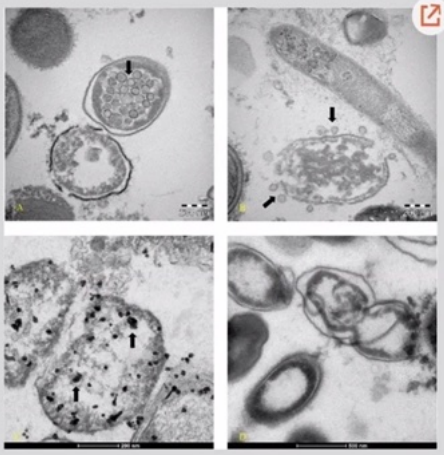


Figure 2. Transmission electron microscopy (post-embedded immunogold).

SARS-CoV-2 has a dual mechanism: it infects human cells but first infects bacterial cells in our microbiome!!

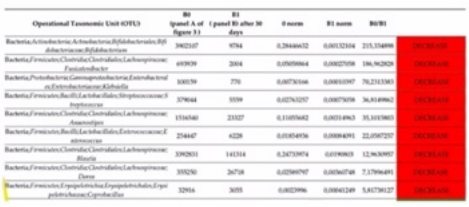
Consequence – release of toxins that go into the blood stream, disturbs the CNS and gut-brain axis

The first report on detecting SARS-CoV-2 inside human fecal-oral bacteria: A case series on asymptomatic family members and a child with COVID-19 [version 1; peer review: 1 approved with reservations]

Carlo Brogna[1], Simone Cristoni[2], [...] Marina Prisco[6], Marina Piscopo[6]

https://f1000research.com/articles/11-135

Picture below, bacteria species infected



8

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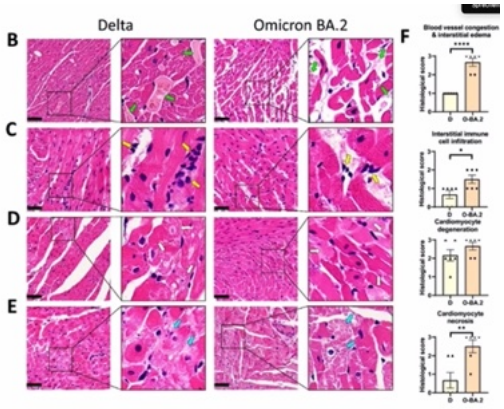
Attacking the heart muscle

SARS-CoV-2 variants divergently infect and damage cardiomyocytes in vitro and in vivo

Bobo Wing-Yee Mok, Maxwell Kwok, ... Ellen Ngar-Yun Poon + Show authors

Omicron BA.2 most efficiently infected and injured CMs in vitro and in vivo, and induced expression changes consistent with increased cardiac dysfunction, **compared to other variants tested.**

https://cellandbioscience.biomedcentral.com/articles/10.1186/s13578-024-01280-y



B myocardial blood vessel congestion and interstitial edema (green arrows), C interstitial immune cell infiltration (yellow arrows), D CM degeneration (white arrows). E CM necrosis (blue arrows). F Histological scores of pathological features scaled 0–3, where 0 indicates the absence of pathological changes.

9

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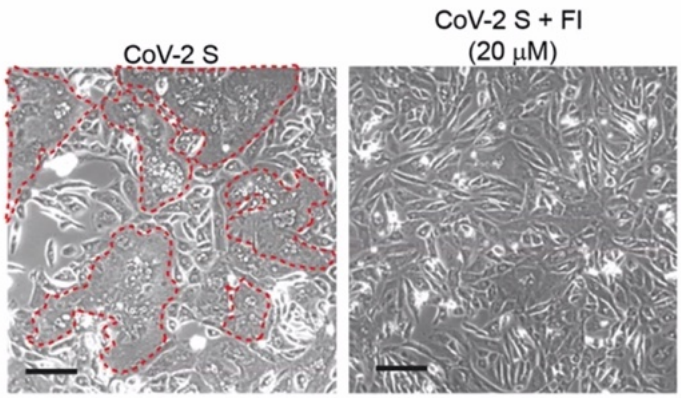
Attacking the heart muscle

Furin activation of spike is required for cardiomyocyte fusion.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC8610601/



SARS-CoV-2 spike-generated syncytia are blocked by a furin inhibitor

10

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Attacking the T-Lymphocytes

ACE2-independent infection of T lymphocytes by SARS-CoV-2

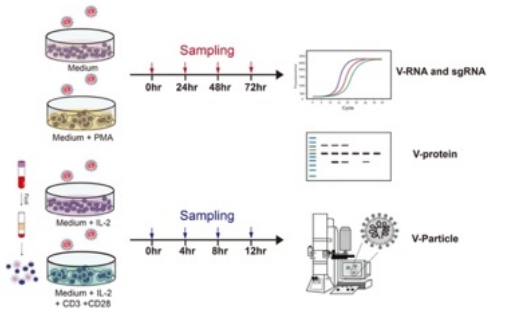
Xu-Rui Shen, Rong Geng, Qian Li, Ying Chen, Shu-Fen Li, Qi Wang, Juan Min, Yong Yang, Bei Li, Ren-Di Jiang, Xi Wang, Xiao-Shuang Zheng, Yan Zhu, Jing-Kun Jia, Xing-Lou Yang, Mei-Qin Liu, Qian-Chun Gong, Yu-Lan Zhang, Zhen-Qiong Guan, Hui-Ling Li, Zhen-Hua Zheng, Zheng-Li Shi, Hui-Lan Zhang, Ke Peng & Peng Zhou

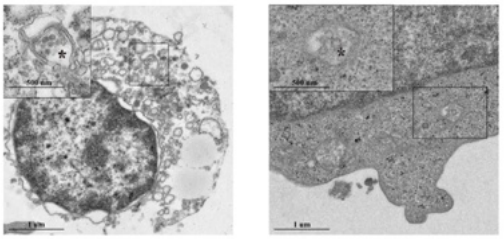
Signal Transduction and Targeted Therapy 7, Article number: 83 (2022) | Cite this article

115k Accesses | 108 Citations | 3959 Altmetric | Metrics

Abstract

SARS-CoV-2 induced marked lymphopenia in severe patients with COVID-19. However, whether lymphocytes are targets of viral infection is yet to be determined, although SARS-CoV-2 RNA or antigen has been identified in T cells from patients. Here, we confirmed that SARS-CoV-2 viral antigen could be detected in patient peripheral blood cells (PBCs) or postmortem lung T cells, and the infectious virus could also be detected from viral antigen-positive PBCs. We next prove that SARS-CoV-2 infects T lymphocytes, preferably activated CD4 + T cells in vitro. Upon infection, viral RNA, subgenomic RNA, viral protein or viral particle can be detected in the T cells. Furthermore, we show that the infection is spike-ACE2/TMPRSS2-independent through using ACE2 knockdown or receptor blocking experiments. Next, we demonstrate that viral antigen-positive T cells from patient undergone pronounced apoptosis. In vitro infection of T cells induced cell death that is likely in mitochondria ROS-HIF-1a-dependent pathways. Finally, we demonstrated that LFA-1, the protein exclusively expresses in multiple leukocytes, is more likely the entry molecule that mediated SARS-CoV-2 infection in T cells, compared to a list of other known receptors.





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11

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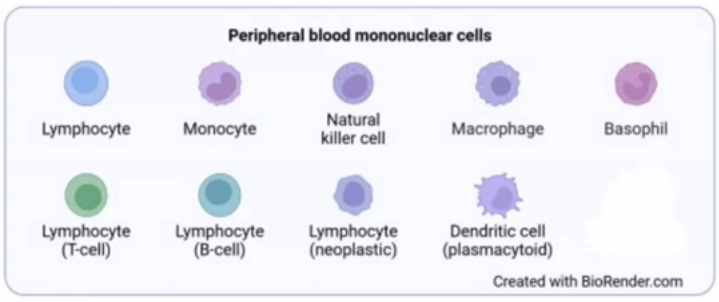
Persistent spikes in peripheral immune cells



The **persistence of SARS-CoV-2 spike proteins** in peripheral blood mononuclear cells (PBMCs) has emerged as a critical factor driving immune dysregulation, particularly through the disruption of **Th1/Th2 balance**. This imbalance, characterized by a shift from antiviral Th1 responses to pro-inflammatory and immunosuppressive Th2 dominance, may underpin chronic inflammation, impaired viral clearance, and long-term immunological complications observed in post-acute COVID-19 syndromes.

SINGLE PATIENT REPORT FROM MMD LAB GERMANY SHOWING HIGH SPIKE LEVELS IN PBMCs !!

Spikeprotein in Immunzellen (PBMC)	POSITIV 1102,58 pg/2,5x10⁶ Zellen
Spikeprotein in Immunzellen (PBMC)	POSITIV 10,04 pg/2,5x10⁶ Zellen

"SPIKE DETOX" FROM PBMCs WITHIN 6 WEEKS! 99% REDUCTION

Eliminating persistent SARS-CoV-2 spike protein from PBMCs is a viable strategy to **restore Th1/Th2 balance**, primarily by halting chronic TLR4/NF-κB signaling and reversing epigenetic Th2 polarization. However, complete immune normalization may require adjunct therapies targeting residual inflammatory pathways and exhausted T cell populations. Clinical trials focusing on spike clearance via degradation therapies are critical to mitigating long-term immunological sequelae of COVID-19.

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12

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Covid-19 virus is inducing senescence

nature › nature reviews immunology › perspectives › article

Perspective | Published: 05 October 2022

COVID-19 and cellular senescence

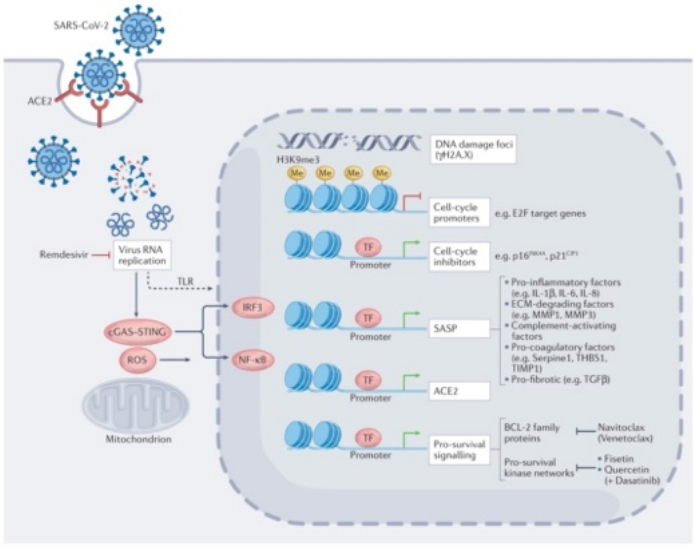
Clemens A. Schmitt , Tamar Tchkonia, Laura J. Niedernhofer, Paul D. Robbins, James L. Kirkland & Soyoung Lee

Nature Reviews Immunology **23**, 251–263 (2023) | Cite this article

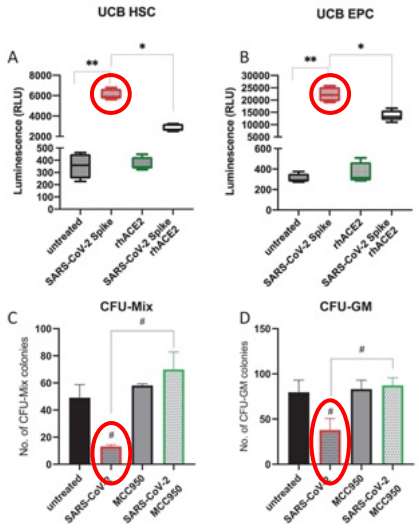
19k Accesses | **84** Citations | **237** Altmetric | Metrics

Abstract

The clinical severity of coronavirus disease 2019 (COVID-19) is largely determined by host factors. Recent advances point to cellular senescence, an ageing-related switch in cellular state, as a critical regulator of SARS-CoV-2-evoked hyperinflammation. SARS-CoV-2, like other viruses, can induce senescence and exacerbates the senescence-associated secretory phenotype (SASP), which is comprised largely of pro-inflammatory, extracellular matrix-degrading, complement-activating and pro-coagulatory factors secreted by senescent cells. These effects are enhanced in elderly individuals who have an increased proportion of pre-existing senescent cells in their tissues. SASP factors can contribute to a 'cytokine storm', tissue-destructive immune cell infiltration, endothelialitis (endotheliitis), fibrosis and microthrombosis. SASP-driven spreading of cellular senescence uncouples tissue injury from direct SARS-CoV-2-inflicted cellular damage in a paracrine fashion and can further amplify the SASP by increasing the burden of senescent cells. Preclinical and early clinical studies indicate that targeted elimination of senescent cells may offer a novel therapeutic opportunity to attenuate clinical deterioration in COVID-19 and improve resilience following infecti SARS-CoV-2 or other pathogens.



Covid-19 virus and spikes are destroying stem cells



STEM CELL BIOLOGY

An evidence that SARS-Cov-2/COVID-19 spike protein (SP) damages hematopoietic stem/progenitor cells in the mechanism of pyroptosis in Nlrp3 inflammasome-dependent manner

Magdalena Kucia, Janina Ratajczak, Kamila Bujko, Mateusz Adamiak, Andrzej Ciechanowicz, Vira Chumak, Katarzyna Brzezniakiewicz-Janus & Mariusz Z. Ratajczak

Leukemia **35**, 3026–3029 (2021) | Cite this article

Mounting evidence accumulates that hematopoietic stem/progenitor cells (HSPCs) and endothelial progenitor cells (EPCs) are damaged during severe SARS-Cov-2/COVID-19 infection [1, 2]. It has been reported that patient infected with COVID-19 are frequently presented with anemia, lymphopenia, and thrombocytopenia [1,2,3]. This negative effect of the virus on human hematopoiesis and endothelium has been reported in infected patients and demonstrated in vitro after exposure of cells to SARS-Cov-2/COVID-19 spike protein (SP) [1, 3, 4]. It is known that virus may enter cells and, directly in case of productive infection, lead to their irreversible damage. On the other hand, the interaction of viral SP with some of the receptors expressed on the cell surface may lead to their damage as well [1,2,3]. We have proposed that interaction of SP with the target cell surface receptors induces intracellular hyperactivation of Nlrp3 inflammasome which may lead to cell death by pyroptosis [5]. It is known that pyroptosis is characterized by the creation in a caspase-1 dependent manner of N-gasdermin pores in the cell membrane, which leads to the release of cytosol components to extracellular space and final cell lysis [6].

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Attacking the inner vascular layer

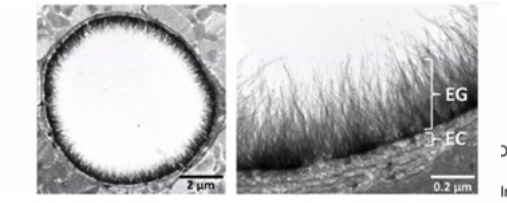
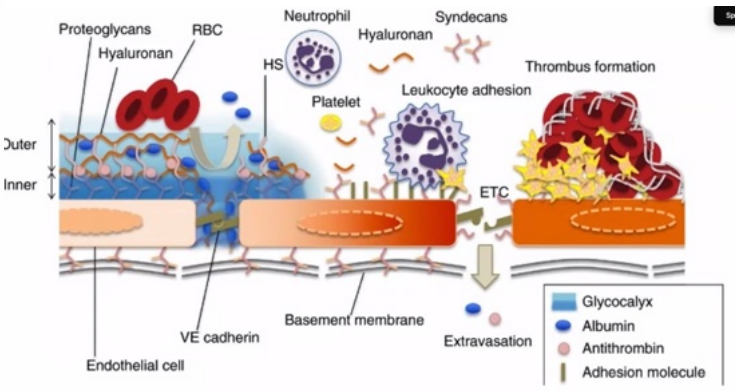


Image from: Haeren, Roel; van de Ven, Steffi; Vink,

ACE2 on the cell surface is fully enclosed by the glycocalyx layer.
Neuropilin-1 (NRP-1) is a transmembrane protein that also acts as a co-receptor for SARS CoV-2 interactions and is also fully enclosed by the glycocalyx.

Glycosaminoglycan mimetics that improve endothelial glycocalyx boundary functions have promising properties in the prevention of viral infection.



Mechanism of the vascular endothelial damage and the two-layer structure of glycocalyx by infections allowing leucocyte adhesion and thrombus formation

15

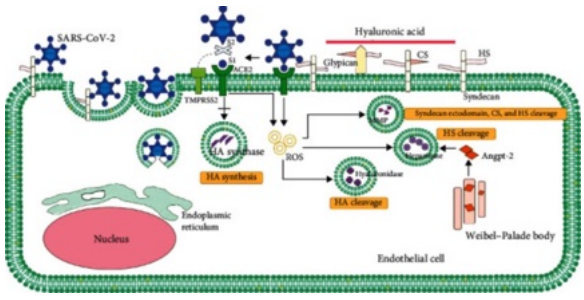
ALPSTEINCLINIC

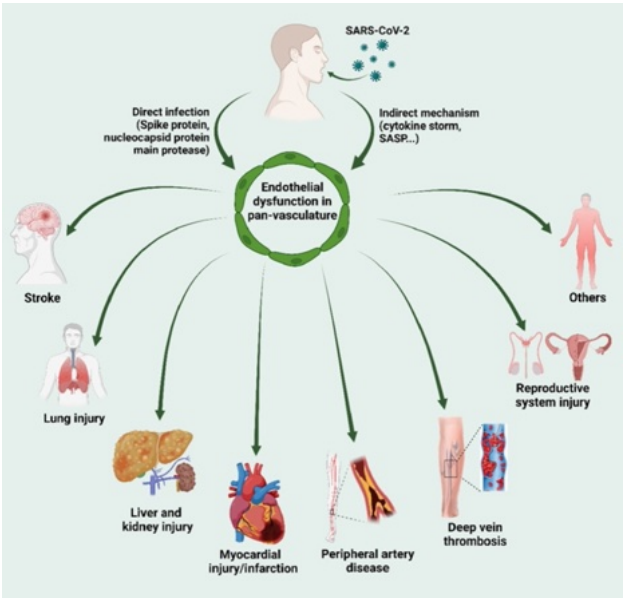
Attacking the inner vascular layer

Review > Acta Pharmacol Sin. 2023 Apr;44(4):695-709. doi: 10.1038/s41401-022-00998-0. Epub 2022 Oct 17.

Endothelial dysfunction in COVID-19: an overview of evidence, biomarkers, mechanisms and potential therapies

Suo-Wen Xu [1], Iqra Ilyas [2], Jian-Ping Weng [3]
Affiliations + expand
PMID: 36253560 PMCID: PMC9574180 DOI: 10.1038/s41401-022-00998-0



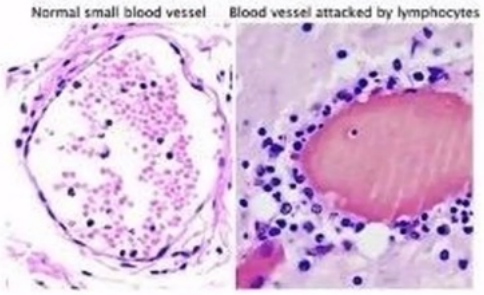


16

ALPSTEINCLINIC

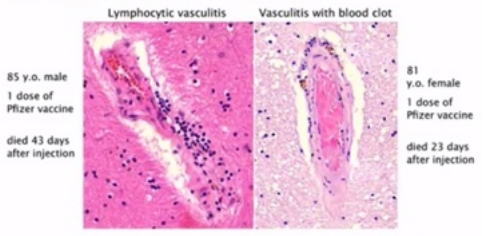
Vasculitis and thrombus formation by spike protein

Lymphocytic inflammation of a small blood vessel

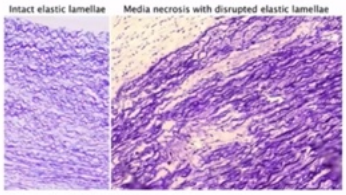


Findings by Prof. Dr. Arne Burkhardt Germany

Vasculitis of small blood vessels in the brain



Damage to elastic fibers in the arteries is irreversible



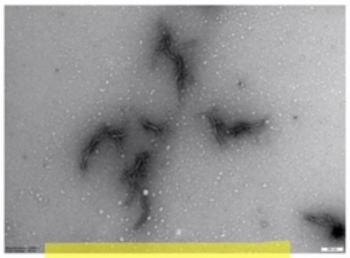
29 y.o. male, 2x Pfizer, died 67 days after second dose

ALPSTEINCLINIC

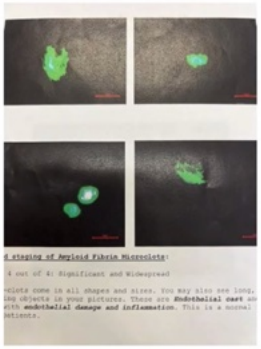
Amyloid fibres and Microclots and SARS-CoV-2 infection?

Possible discovery of mechanism behind mysterious COVID-19 symptoms

by Karin Söderlund Leifler , Linköping University



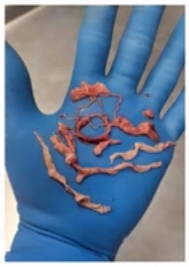
Picture of amyloid from the SARS-CoV-2 virus' spike protein, seen using an electron microscope. When the spike protein is mixed with the enzyme neutrophil elastase in test tubes, branched protein fibrils are created, which potentially can cause disturbed blood coagulation in patients with COVID-19. Credit: Sofie Nyström and Per Hammarström



Blood diagnostics - Fluorescent microscopy

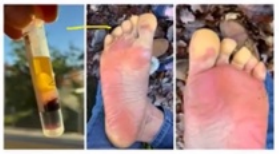
Elevated presence in the majority of patients (severity scale 1-4, in this case: 4 out of 4)

https://medicalxpress.com/news/2022-05-discovery-mechanism-



Richard Hirschman

Clot formation in blood sample of a live vaccinated patient with disrupted perfusion



Professor Dr. Arne Burckhardt

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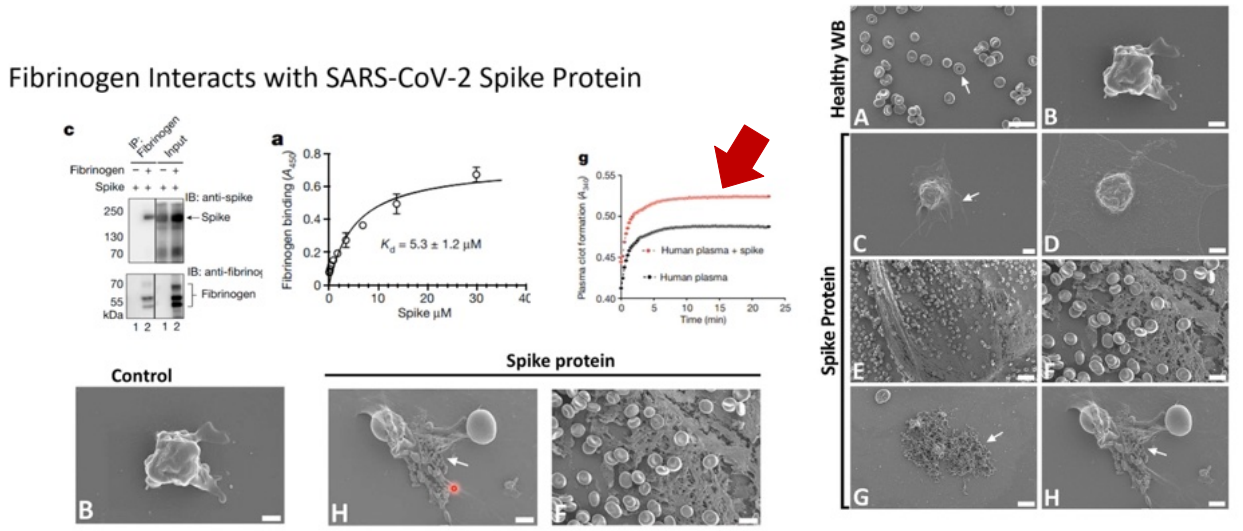
More fibrin formation induced by spike protein

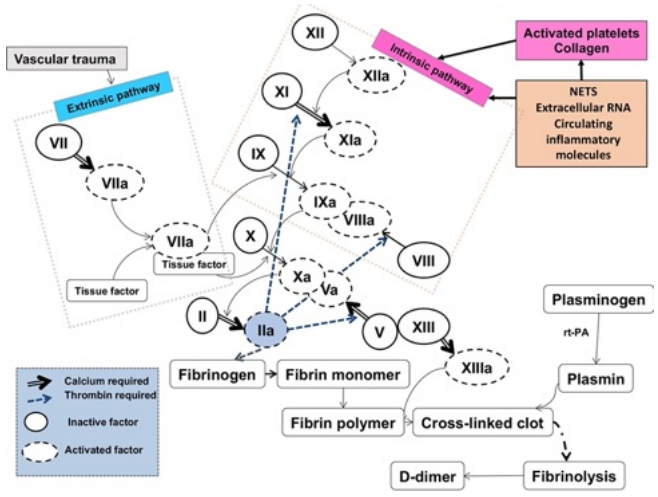


Ryue et al 2024, Nature

19

ALPSTEINCLINIC

Amyloid-Fibrin Mikroclot formation in long COVID patients



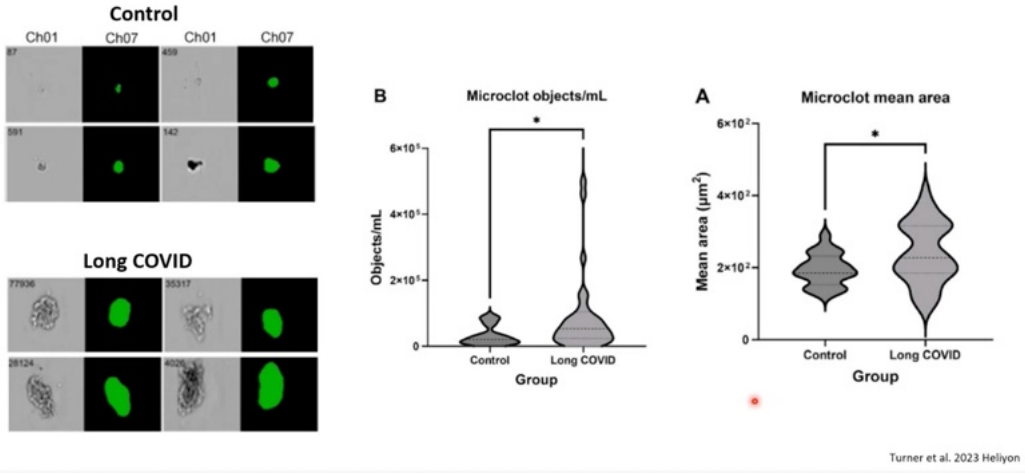
Representation of the classical blood-clotting cascades, ending in the removal of two fibrinopeptides from fibrinogen, and its self organization to produce fibrin (that may then be cross-linked).The eventual result of fibrinolysis, that acts as a record of its extent, is d-dimer. Redrawn from a CC-BY publication at [26].

20

ALPSTEINCLINIC

Long COVID and Microclot formation

Long COVID Patients Have More and Bigger Microclots Compared to Control Group



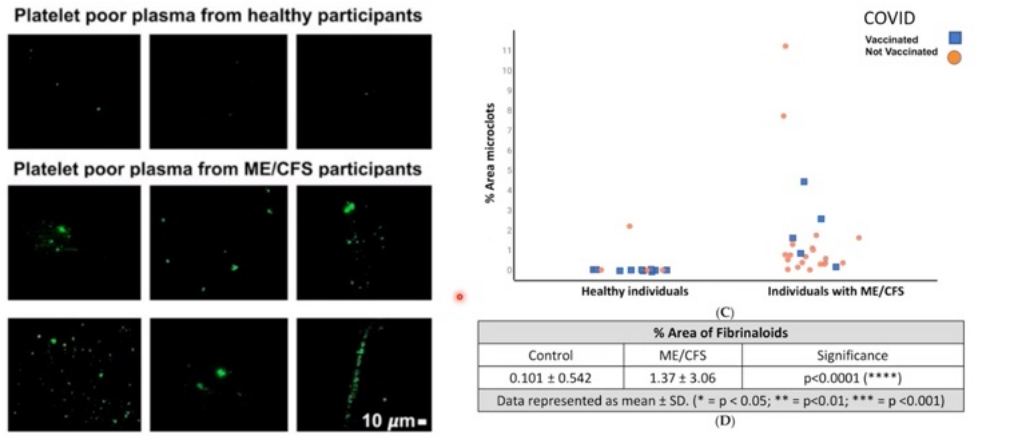
Turner et al. 2023 Heliyon

21

ALPSTEINCLINIC

More Microclot formation in patients with ME/CFS and after Covid-vaccination

Increased Microclot Presence in ME/CFS



% Area of Fibrinaloids		
Control	ME/CFS	Significance
0.101 ± 0.542	1.37 ± 3.06	p<0.0001 (****)
Data represented as mean ± SD. (* = p < 0.05; ** = p<0.01; *** = p <0.001)		

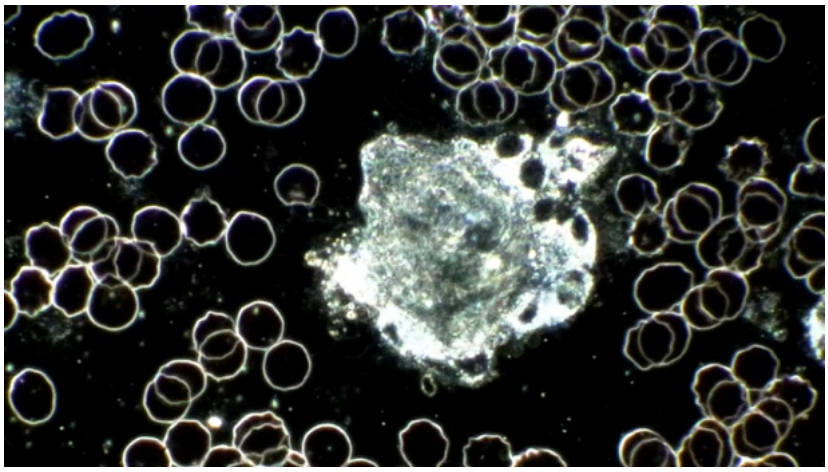
(D)

Nunes et al 2022 Pharmaceuticals

22

ALPSTEINCLINIC

“Protein deposits” in dark field microscopy



x250, after 3 hours

23

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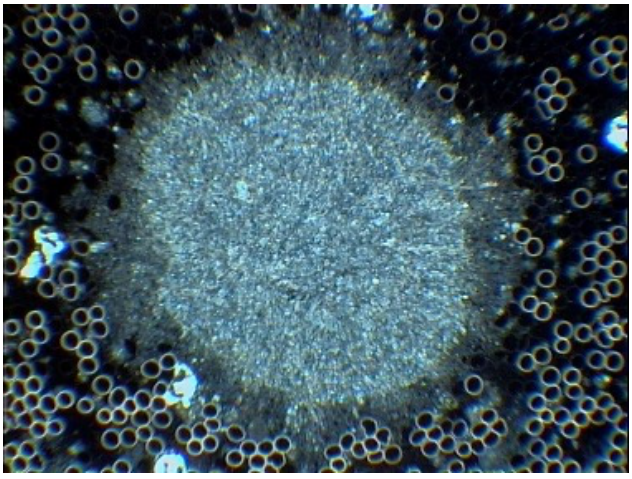
Micro thrombus in case of post-Covid syndrome



Dark field microscopy, x200

24

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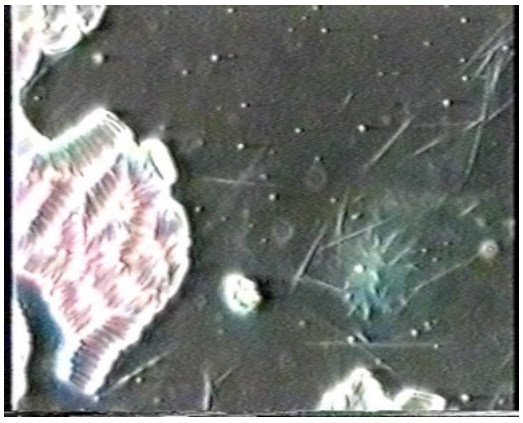
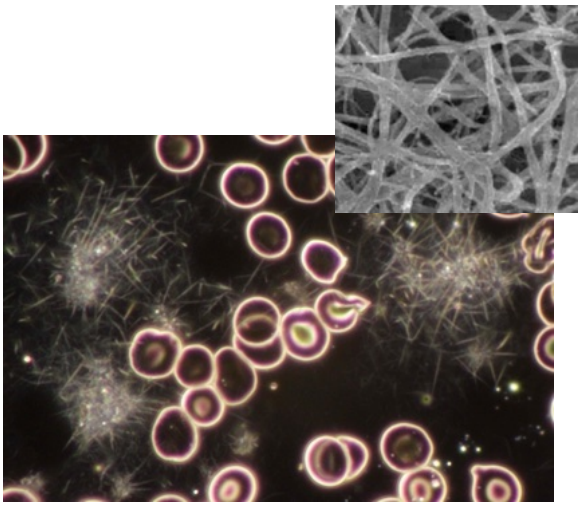
Fibrin formation in Plasma



Sludje phenomena and fibrin fibers



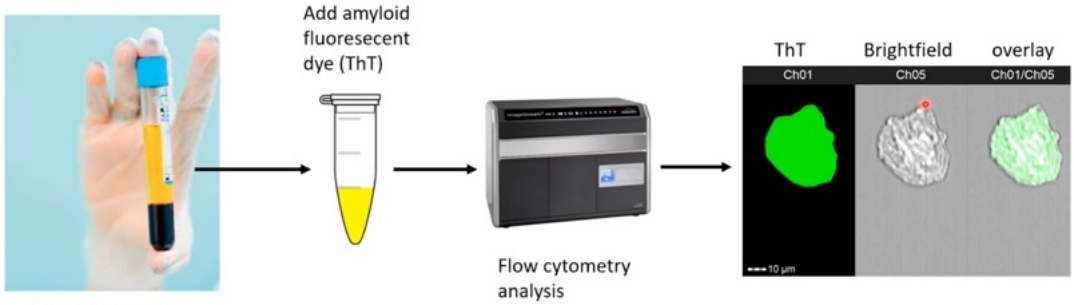
Fibrinnests / inflammation / oxidative stress

25

ALPSTEINCLINIC

Analysis of microclots from blood

Process for Microclot Analysis



Take the platelet poor plasma

Flow cytometry analysis

26

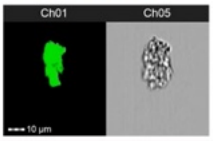
ALPSTEINCLINIC

Presentation of findings

Defining Different Categories for Microparticles

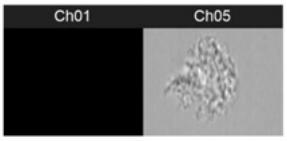
Amyloid like positive (Microclots)

Parameter	Explanation
A-TA	Total amount of amyloid fibrin microclots (ThT+) (whole size range: 0-1000 µm²)
A-TA(S)	Total amount of amyloid fibrin microclots (ThT+) of small (S) size: < 30 µm²
A-TA(M)	Total amount of amyloid fibrin microclots (ThT+) of medium (M) size: 30-55 µm²
A-TA(L)	Total amount of amyloid fibrin microclots (ThT+) of large (L) size: 55-600 µm²
A-TA(XL)	Total amount of amyloid fibrin microclots (ThT+) of extra large (XL) size: 600-1000 µm²



Amyloid like negative (Microparticles)

Parameter	Explanation
M-TA	Total amount of microparticles (ThT-) (whole size range: 0-1000 µm²)
M-TA(S)	Total amount of microparticles (ThT-) of small (S) size: < 5 µm²
M-TA(M)	Total amount of microparticles (ThT-) of medium (M) size: 5-45 µm²
M-TA(L)	Total amount of microparticles (ThT-) of large (L) size: 45-300 µm²
M-TA(XL)	Total amount of microparticles (ThT-) of extra large (XL) size: 300-1000 µm²



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27

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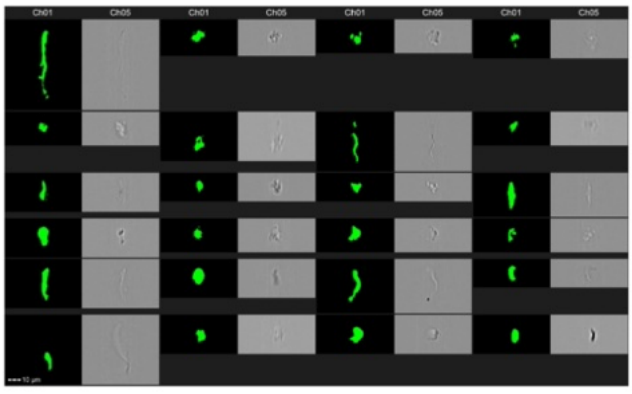
Almost normal finding

Parameter	Explanation	Your value	Unit	Reference (healthy controls, 95% CI) 5%	50% (median)	95%	Deviation from reference median
A-TA	Total amount of amyloid fibrin microclots (ThT+) (whole size range: 0-1000 µm²)	4.894	mm²	0.737	4.2365	12.191	0.66
M-TA	Total amount of microparticles (ThT-) (whole size range: 0-1000 µm²)	25.859	mm²	12.51	17.9995	45.496	7.86
AM-TA	Total amount of amyloid fibin microclots (ThT+) + total amount of microparticles (ThT-) (whole size range: 0-1000 µm²)	30.753	mm²	16.08	20.682	57.686	10.07
A-TA(S)	Total amount of amyloid fibrin microclots (ThT+) of small (S) size: < 30 µm²	0.027	mm²	0	0.008	0.1148	0.02
M-TA(S)	Total amount of microparticles (ThT-) of small (S) size: < 30 µm²	0.133	mm²	0.048	0.0665	0.1216	0.07
A-TA(M)	Total amount of amyloid fibrin microclots (ThT+) of medium (M) size: 30-55 µm²	0.632	mm²	0.047	0.78425	1.4086	-0.15
M-TA(M)	Total amount of microparticles (ThT-) of small (M) size: 5-45 µm²	19.453	mm²	5.362	9.7035	33.418	9.75
A-TA(L)	Total amount of amyloid fibrin microclots (ThT+) of large (L) size: 55-600 µm²	2.974	mm²	0.683	2.9295	7.1632	0.04
M-TA(L)	Total amount of microparticles (ThT-) of large (L) size: 45-300 µm²	5.903	mm²	2.954	5.512	15.026	0.39
A-TA(XL)	Total amount of amyloid fibrin microclots (ThT+) of extra large (XL) size: 55-600 µm²	1.261	mm²	0	0.5205	3.8277	0.74
M-TA(XL)	Total amount of microparticles (ThT-) of extra large (XL) size: 45-300 µm²	0	mm²	0.114	1.4835	6.0466	-1.48



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28

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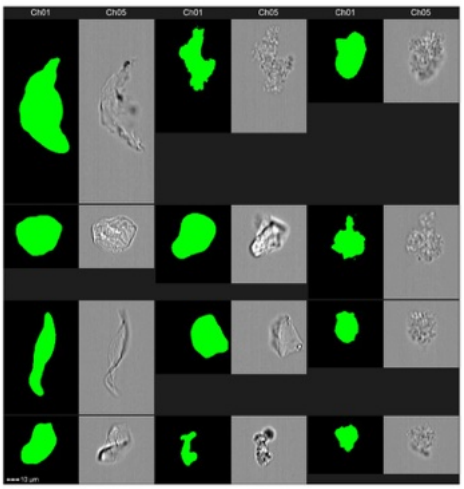
Clear pathological finding

Parameter	Explanation	Your value	Unit	Reference (healthy controls, 95% CI) 5%	50% (median)	95%	Deviation from reference median
A-TA	Total amount of amyloid fibrin microclots (ThT+) (whole size range: 0-1000 µm²)	101.958	mm²	0.737	4.2365	12.191	97.72
M-TA	Total amount of microparticles (ThT-) (whole size range: 0-1000 µm²)	141.636	mm²	12.51	17.9995	45.496	123.64
AM-TA	Total amount of amyloid fibin microclots (ThT+) + total amount of microparticles (ThT-) (whole size range: 0-1000 µm²)	243.594	mm²	16.08	20.682	57.686	222.91
A-TA(S)	Total amount of amyloid fibrin microclots (ThT+) of small (S) size: < 30 µm²	2.459	mm²	0	0.008	0.1148	2.45
M-TA(S)	Total amount of microparticles (ThT-) of small (S) size: < 30 µm²	1.152	mm²	0.048	0.0665	0.1216	1.09
A-TA(M)	Total amount of amyloid fibrin microclots (ThT+) of medium (M) size: 30-55 µm²	4.7508	mm²	0.047	0.78425	1.4086	3.97
M-TA(M)	Total amount of microparticles (ThT-) of small (M) size: 5-45 µm²	63.816	mm²	5.362	9.7035	33.418	54.11
A-TA(L)	Total amount of amyloid fibrin microclots (ThT+) of large (L) size: 55-600 µm²	86.049	mm²	0.683	2.9295	7.1632	83.12
M-TA(L)	Total amount of microparticles (ThT-) of large (L) size: 45-300 µm²	73.322	mm²	2.954	5.512	15.026	67.81
A-TA(XL)	Total amount of amyloid fibrin microclots (ThT+) of extra large (XL) size: 55-600 µm²	8.559	mm²	0	0.5205	3.8277	8.04
M-TA(XL)	Total amount of microparticles (ThT-) of extra large (XL) size: 45-300 µm²	2.927	mm²	0.114	1.4835	6.0466	1.44



29

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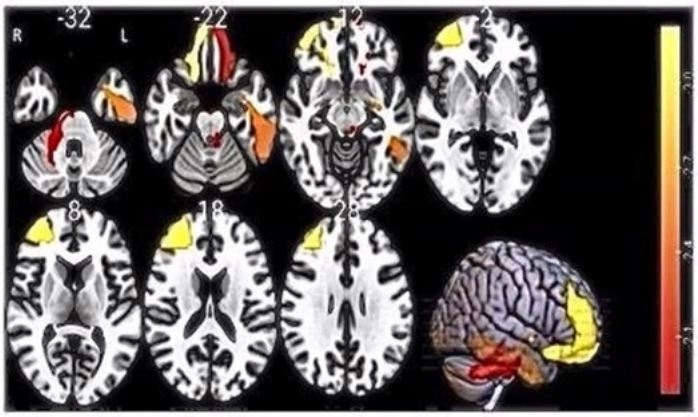
Reduced cerebral blood flow in post-COVID patients

Journal of Neuroimaging / Early View
CLINICAL INVESTIGATIVE STUDY Open Access

Cerebral blood flow in patients recovered from mild COVID-19

Souvik Sen, Roger Newman-Norlund, Nicholas Riccardi, Christopher Rorden, Sarah Newman-Norlund, Sara Sayers, Julius Fridriksson, Makenzie Logue

Areas where the COVID-19 group had significantly lower cerebral blood flow than the control group. White numbers indicate axial slice location. The color bar represents the z-scores for this contrast. Only statistically significant regions are shown. Data are derived from univariate analysis. R, right; L, left.



https://onlinelibrary.wiley.com/doi/full/10.1111/jon.13129

30

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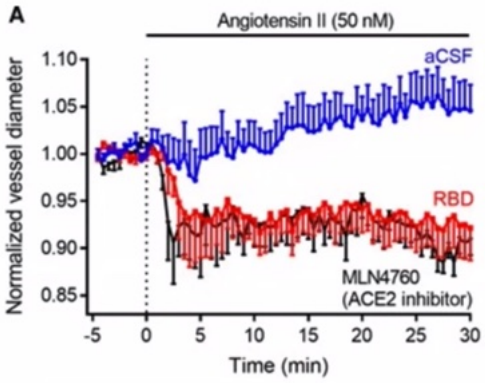
More cerebral capillary constriction in post-COVID patients

SARS-CoV-2 triggers pericyte-mediated cerebral capillary constriction

Chanawee Hirunpattarasilp, Greg James, Jaturon Kwanthongdee, Felipe Freitas, Jiandong Huo, Huma Sethi, Josef T Kittler, Raymond J Owens, Laura E McCoy, David Attwell

We found that the RBD greatly potentiated the pericyte-mediated constriction evoked in human capillaries by 50 nM angiotensin II (Fig. 4B and C). SARS-CoV-2 binding would therefore be expected to decrease human cerebral blood flow.



A) Capillary constriction at pericytes in response to 50 nM angiotensin II in the absence (n = 9) and presence (n = 9) of the RBD

https://academic.oup.com/brain/article/146/2/727/6648726

31

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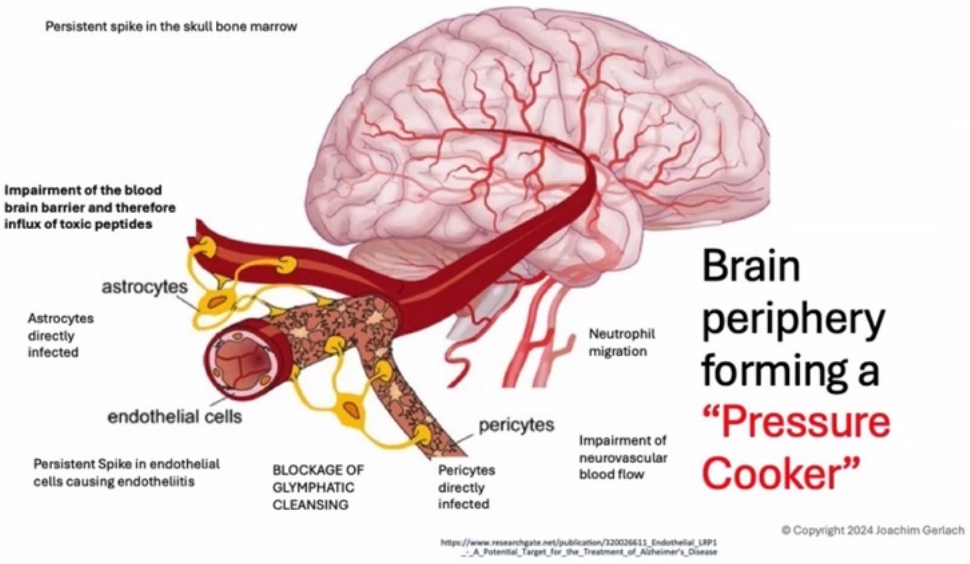
Development of more pressure in the brain in Covid-19



32

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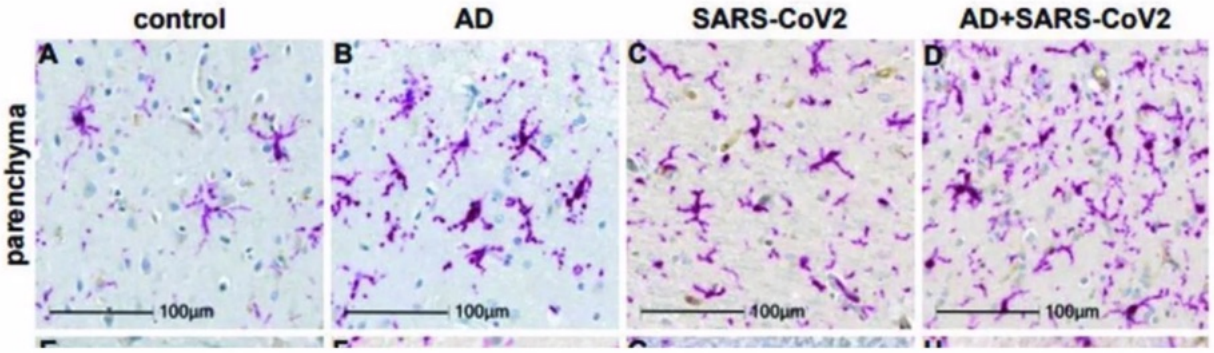
Sars-CoV-2 generates neuroinflammation an promotes neurodegeneration



Microgliosis and nodular lesions in neurological controls, SARS-CoV-2, Alzheimer's disease (AD), and SARS-CoV-2-infected AD individuals. Level of microglial activation.

33

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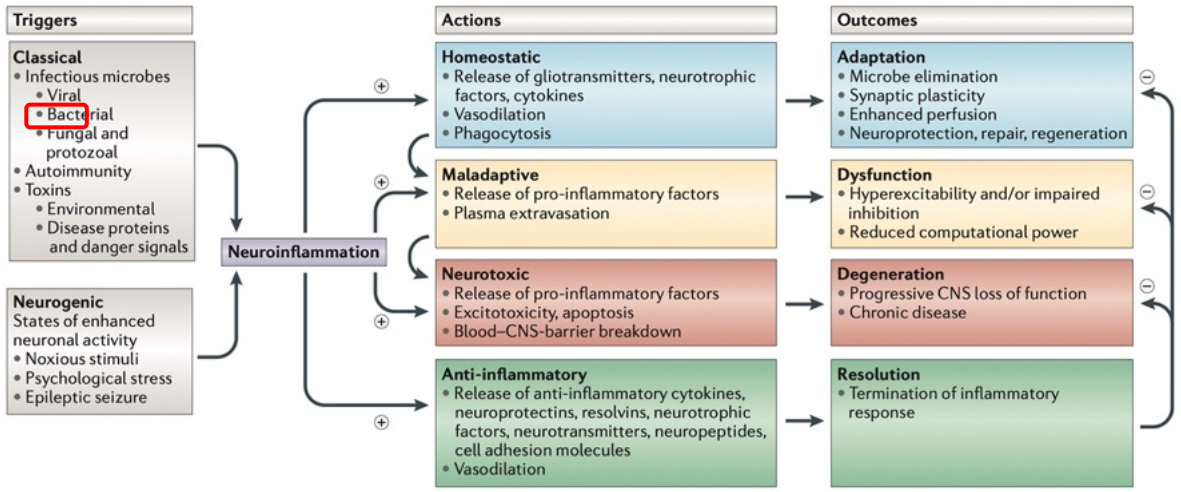
Triggers, actions and outcomes of neuroinflammation



Dimitris N. Xanthos and Jürgen Sandkühler : Neurogenic neuroinflammation: inflammatory CNS reactions in response to neuronal activity, NATURE REVIEWS | NEUROSCIENCE VOLUME 15 | JANUARY 2014 | **43**

34

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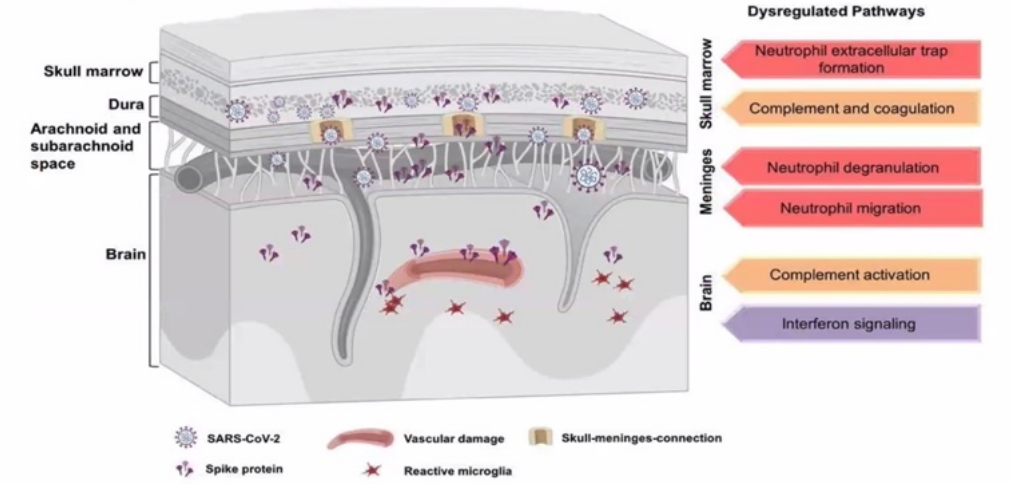
Sars-CoV-2 and spikes persistence in the brain

SARS-COV-2 / PIKE PERSISTENCE IN THE SKULL / MENINGES
Subsequent Neutrophil migration



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35

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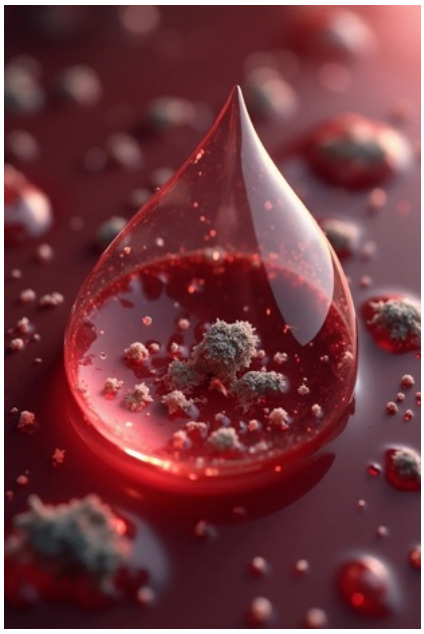
Consequences

By using integrative-biological Medicine



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36

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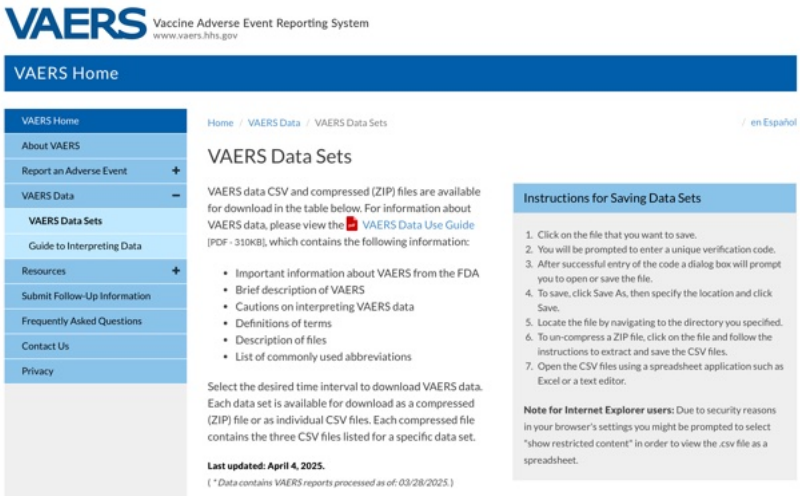
Check your batch number from Covid-vaccination

Webpage:
https://knollfrank.github.io/HowBadIsMyBatch/HowBadIsMyBatch.html



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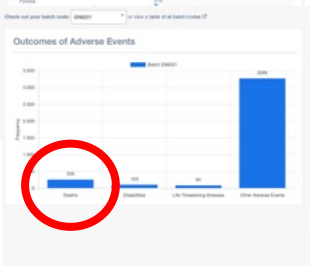
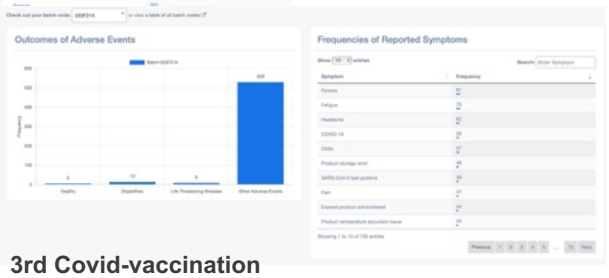
Check your batch number from Covid-vaccination

1st Covid-vaccination



2nd Covid-vaccination

64-y. old woman, Lyme disease with clear worsening after the second vaccination



3rd Covid-vaccination

Source:
https://vaers.hhs.gov/data/datasets.html

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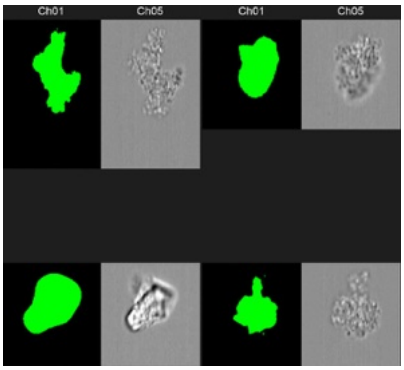
Consequences of microclot problem

1. After finding of elevated number of dry proteins / symplasts / micro thrombus and fibrin nests -> *Microclot Analysis is indicated*
2. Beside post COVID-19 also common in cases of
   - Parkinson‘s disease
   - Dementia – Alzheimer‘s disease
   - Diabetes type 2
   - Rheumatoid arthritis
3. Also, think on microplastics!
4. Biological treatment
   - SANUM: Mucokehl / Lactovis
   - Enzymes (Bromelain, WobeMugos, Nattokinase)
   - Platelet agglutination inhibitors (e.g. Fraxinus)
   - Antioxidants
   - INUSpheresis®



39

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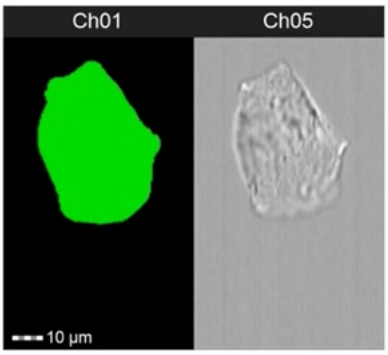
THE method to remove the Microclots …

Physically Microclots Cannot Pass Through the INUSpheresis Filter



Microclot can reach up to 10µm in diameter

INUSpheresis only allows particles smaller than 20-50 nm to pass through

40

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Clinical improvement of Long-COVID is associated with reduction in autoantibodies, lipids, and inflammation following therapeutic apheresis

Martin Achleitner[1,10], Charlotte Steenblock[1,10]✉, Juliane Dänhardt[1], Natalia Jarzebska[1], Romina Kardashi[1], Waldemar Kanczkowski[1], Richard Straube[2], Roman N. Rodionov[1], Nitzan Bornstein[1], Sergey Tselmin[1], Frank Kaiser[3], Ronald Bucher[4], Mahmoud Barbir[5], Ma-Li Wong[6,7], Karin Voit-Bak[2], Julio Licinio[6,7] and Stefan R. Bornstein[1,8,9]

85 % success rate!

In the aftermath of the COVID-19 pandemic, we are witnessing an unprecedented wave of post-infectious complications. Most prominently, millions of patients with Long-Covid complain about chronic fatigue and severe post-exertional malaise. Therapeutic apheresis has been suggested as an efficient treatment option for alleviating and mitigating symptoms in this desperate group of patients. However, little is known about the mechanisms and biomarkers correlating with treatment outcomes. Here, we have analyzed in different cohorts of Long-Covid patients specific biomarkers before and after therapeutic apheresis. In patients that reported a significant improvement following two cycles of therapeutic apheresis, there was a significant reduction in neurotransmitter autoantibodies, lipids, and inflammatory markers. Furthermore, we observed a 70% reduction in fibrinogen, and following apheresis, erythrocyte rouleaux formation and fibrin fibers largely disappeared as demonstrated by dark field microscopy. This is the first study demonstrating a pattern of specific biomarkers with clinical symptoms in this patient group. It may therefore form the basis for a more objective monitoring and a clinical score for the treatment of Long-Covid and other postinfectious syndromes.

Molecular Psychiatry; https://doi.org/10.1038/s41380-023-02084-1

Published:

Molecular Psychiatry 20.5.23

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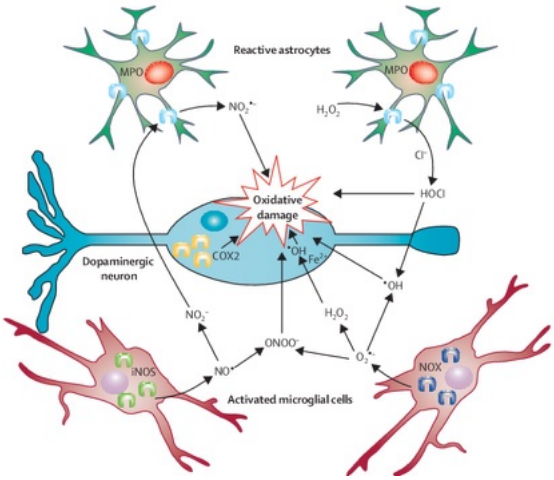
See also webinar from 2024

Biological Therapy
against neuroinflammation

- **Antioxidants** (Vit. C, E, Q10, Zinc, Selenium, OPC)
- **Frankincense** (Olibanum)
- **Alpha-lipoic acid** (600 to 1.200 mg)
- **Omega-3-Fatty acids**
- **Secondary Plant substances** (Curcuma, Resveratrol, Quercetin)
- **Galactose** (plus Glycoplan®)
- **Procaine / ProcCluster®**
- **Perfect bedroom protection**
- **INUSpheresis®**



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Reducing spikes and reduction of reinfection risk

- **Empowering the immune system:**
  - Healthy lifestyle and nutrition
  - Basic supplementation: antioxidants, Vitamin D, pre- and probiotics
  - ASLAN therapy, organopeptides (see webinar 2024)
  - SANUM immune modulation: *Utilin S D4, Recarcin D4, Bovisan D5* (weekly each)
  - Whole body hyperthermia, active fever therapy
- **Biological remedies for early signs of flue / infection**
  - Oscillococcinium, Cystus 052, Melatonin spray (intra-nasal)
- **Reduction and release of Spike proteins**
  - Curcuma / Turmeric 500 mg (2 to 3 times daily), infusion with 250 to 500 mg
  - Resveratrol (200 to 400 mg daily)
  - Artesunate (250 to 500 mg IV)
  - Quercetin (500 to 1000 mg daily)

43

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Curcumin, Spike protein and amyloid reduction

Journal of Neurochemistry JNC

Curcumin binds to the α-helical intermediate and to the amyloid form of prion protein – a new mechanism for the inhibition of PrP^{Sc} accumulation

Correction(s) for this article

Iva Hafner-Bratkovič, Jernej Gašperšič, Lojze M. Šmid, Mara Bresjanac, Roman Jerala

First published: 07 November 2007 | https://doi.org/10.1111/j.1471-4159.2007.05105.x | Citations: 114

Abstract

J. Neurochem. (2008) **104,** 1553–1564.

Conversion of the native, predominantly α-helical conformation of prion protein (PrP) into the β-stranded conformation is characteristic for the transmissible spongiform encephalopathies such as Creutzfeld–Jakob disease. Curcumin, an extended planar molecule and a dietary polyphenol, inhibits *in vitro* conversion of PrP and formation of protease resistant PrP in neuroblastoma cell lines. Curcumin recognizes the converted β-form of the PrP both as oligomers and fibrils but not the native form. Curcumin binds to the prion fibrils in the left-handed chiral arrangement as determined by circular dichroism. We show that curcumin labels the plaques of the brain sections of variant Creutzfeld–Jakob disease cases and stains the same structures as antibodies against the PrP. In contrast to thioflavin T, curcumin also binds to the α-helical intermediate of PrP present at acidic pH at stoichiometry of 1 : 1. Congo red competes with curcumin for binding to the α-intermediate as well as to the β-form of PrP but is toxic and binds also to the native form of PrP. We therefore show that the partially unfolded structural intermediate of the PrP can be targeted by non-toxic compound of natural origin.

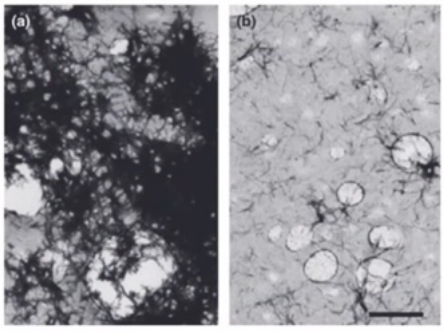

Fig. 2 Inhibition of PrP fibril formation by curcumin. PrP conversion reaction in the absence (a) or presence (b) of 80 μmol/L curcumin has been followed by electron microscopy. While in the absence of curc-

44

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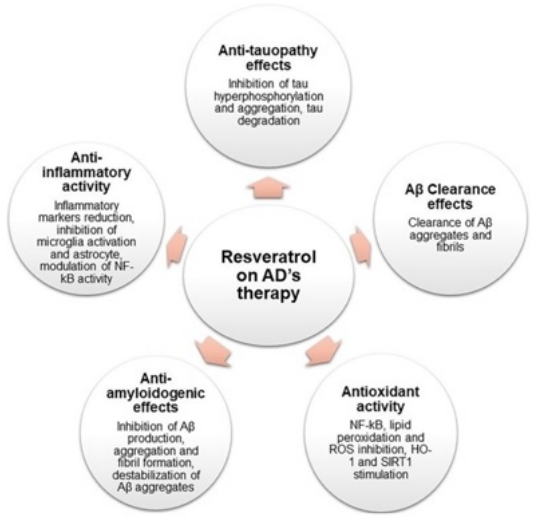
Resveratrol and Spike protein reduction



Review > Front Pharmacol. 2018 Nov 20:9:1261. doi: 10.3389/fphar.2018.01261. eCollection 2018.

Resveratrol Brain Delivery for Neurological Disorders Prevention and Treatment

Stephanie Andrade [1], Maria João Ramalho [1], Maria do Carmo Pereira [1], Joana A Loureiro [1]

Affiliations + expand

PMID: 30524273 PMCID: PMC6262174 DOI: 10.3389/fphar.2018.01261

Abstract

Resveratrol (RES) is a natural polyphenolic non-flavonoid compound present in grapes, mulberries, peanuts, rhubarb and in several other plants. Numerous health effects have been related with its intake, such as anti-carcinogenic, anti-inflammatory and brain protective effects. The neuroprotective effects of RES in neurological diseases, such as Alzheimer's (AD) and Parkinson's (PD) diseases, are related to the protection of neurons against oxidative damage and toxicity, and to the prevention of apoptotic neuronal death. In brain cancer, RES induces cell apoptotic death and inhibits angiogenesis and tumor invasion. Despite its great potential as therapeutic agent for the treatment of several diseases, RES exhibits some limitations. It has poor water solubility and it is chemically instable, being degraded by isomerization once exposed to high temperatures, pH changes, UV light, or certain types of enzymes. Thus, RES has low bioavailability, limiting its biological and pharmacological benefits. To overcome these limitations, RES can be delivered by nanocarriers. This field of nanomedicine studies how the drug administration, pharmacokinetics, and pharmacodynamics are affected by the use of nanosized materials. The role of nanotechnology, in the prevention and treatment of neurological diseases, arises from the necessity to mask the physicochemical properties of therapeutic drugs to prolong the half-life and to be able to cross the blood-brain barrier (BBB). This can be achieved by encapsulating the drug in a nanoparticle (NP), which can be made of different kinds of materials. An increasing trend to encapsulate and direct RES to the brain has been observed. RES has been encapsulated in many different types of nanosystems, as liposomes, lipid and polymeric NPs. Furthermore, some of these nanocarriers have been modified with targeting molecules able to recognize the brain areas. Then, this article aims to overview the RES benefits and limitations in the treatment of neurological diseases, as the different nanotechnology strategies to overcome these limitations.

45

ALPSTEINCLINIC

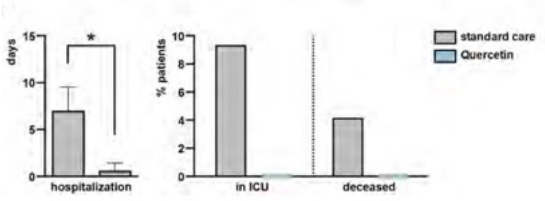
Quercetin is reducing the Covid-induced senescence

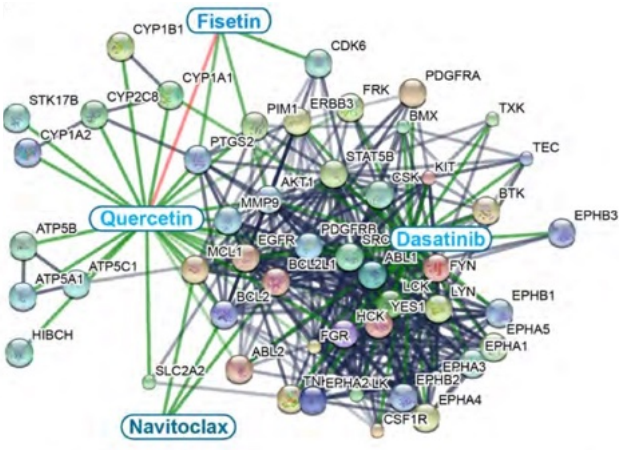
Article | Published: 13 September 2021

Virus-induced senescence is a driver and therapeutic target in COVID-19

Soyoung Lee, Yong Yu, Jakob Trimpert, Fahad Benthani, Mario Mairhofer, Paulina Richter-Pechanska, Emanuel Wyler, Dimitri Belenki, Sabine Kaltenbrunner, Maria Pammer, Lea Kausche, Theresa C. Firsching, Kristina Dietert, Michael Schotsaert, Carles Martínez-Romero, Gagandeep Singh, Séverine Kunz, Daniela Niemeyer, Riad Ghanem, Helmut J. F. Salzer, Christian Paar, Michael Mülleder, Melissa Uccellini, Edward G. Michaelis, ... Clemens A. Schmitt + Show authors

Nature **599**, 283–289 (2021) | Cite this article





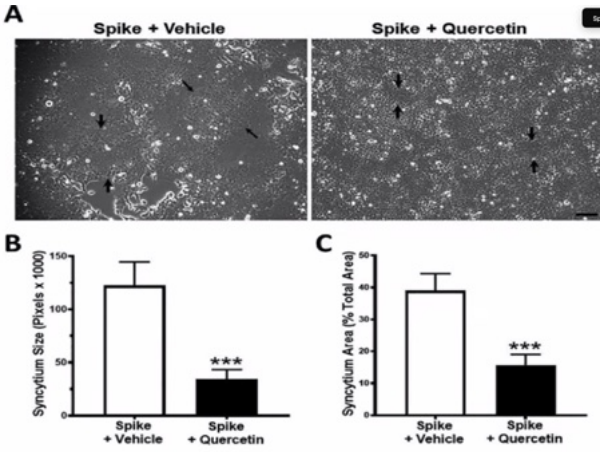
46

ALPSTEINCLINIC

Quercetin is reducing spike protein expression

The spike protein of SARS-CoV-2 induces heme oxygenase-1: Pathophysiologic implications

Raman Deep Singh [a], Michael A. Barry [b], Anthony J. Croatt [a], Allan W. Ackerman [a], Joseph P. Grande [c], Rosa M. Diaz [d], Richard G. Vile [d], Anupam Agarwal [e], Karl A. Nath [a]



SARS-CoV-2 spike protein expression in kidney cells results in syncytia formation with cellular sloughing

- Quercetin reduces syncytia size and spike protein expression

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47

48

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More Webinars till Summer 2025

Time	Speaker	Topic
22.05.25	Günther Bauer	*Principles of homotoxicological medicine*
26.06.25	Andreas Petzold	*The homeopathic pharmacy for travelling*

always Thursday, 6.30 pm, METZ

possible for Downloading for the next 24 hours

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49