Immune disorders in Post-Vaccine-Syndrom

https://rumble.com/vqb64v-immunologische-risiken-der-corona-impfung.html

- 2006 - 2017 in eigener Praxis tätig, Schwerpunkt Onkologie & Neuroinflammation
- 2017 - 2019 Project-Manager Asia-Pacific, Integrative Oncology, Neurodegenerative Diseases
- Seit 2019 Wissenschaftlicher Leiter Mitocare GmbH, München
- Vizepräsident der DGNAME e.V.
- Seit 2009 internationale Vortragstätigkeit

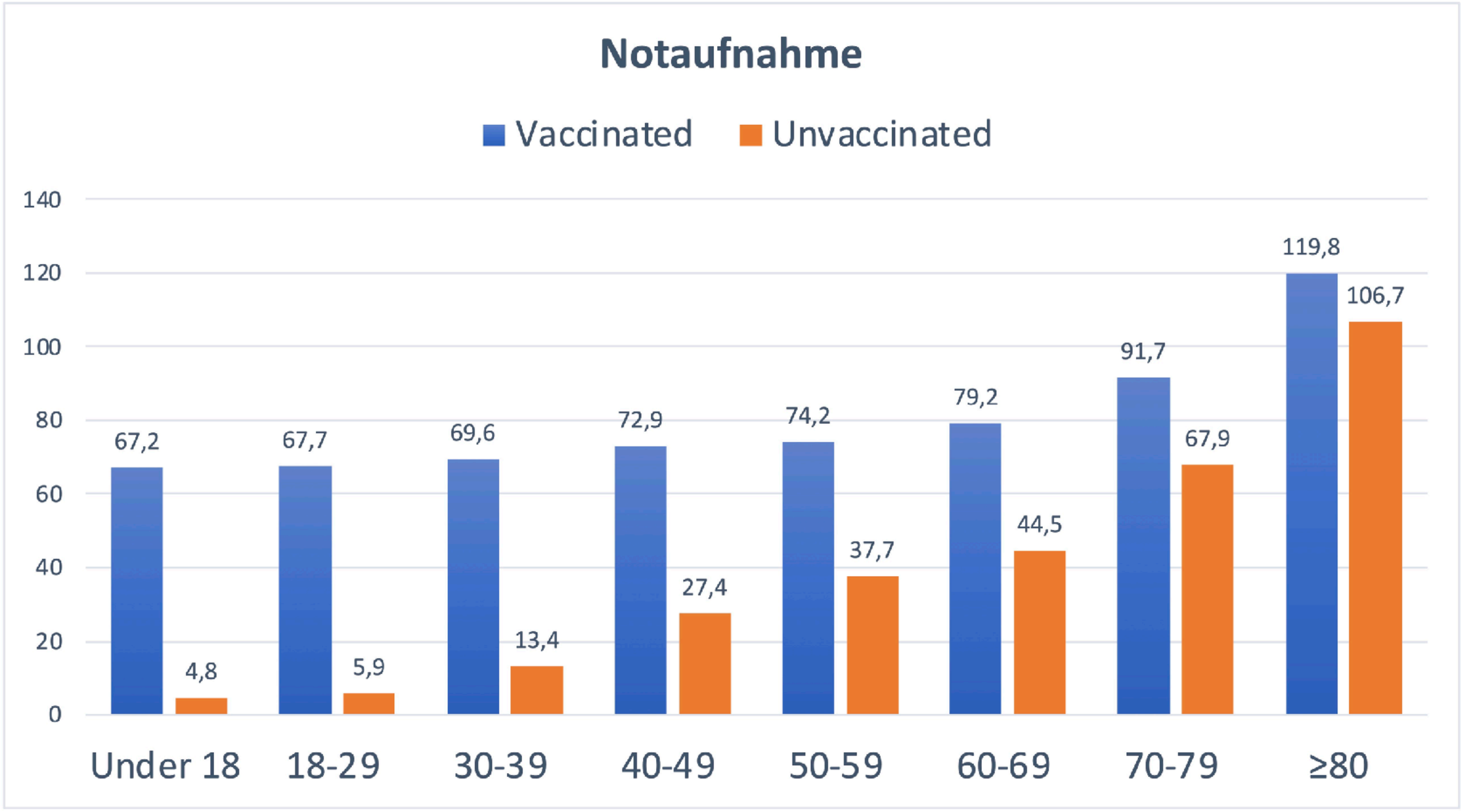
1. Warning signs?
2. Spike-induced Neuroinflammation
3. Nanoparticle-induced inflammation
4. ADE (Antibody-dependent enhancement)
5. VIDS: Vaccine-induced immune deficiency syndrome
6. Autoimmunity
7. Treatment strategies

Warning signs, any?

UK Health Security Agency

COVID-19 vaccine surveillance report
Week 42

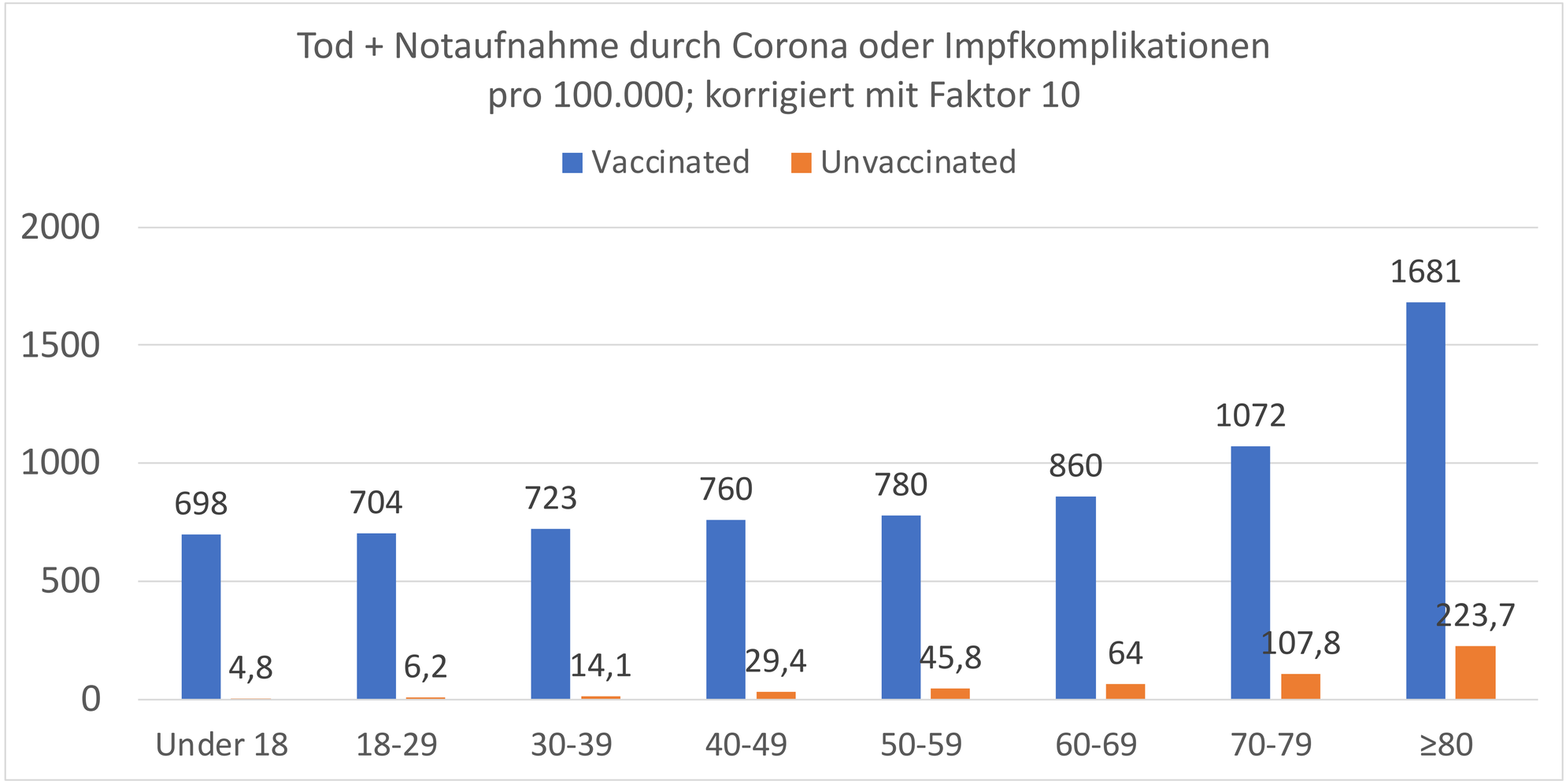
https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/1027511/Vaccine-surveillance-report-week-42.pdf


Notaufnahme
Vaccinated
Unvaccinated
140
120
100
80
60
40
20
0
67,2
4,8
67,7
5,9
69,6
13,4
72,9
27,4
74,2
37,7
79,2
44,5
91,7
67,9
119,8
106,7
Under 18
18-29
30-39
40-49
50-59
60-69
70-79
≥80

No-reporting statt Under-reporting ?

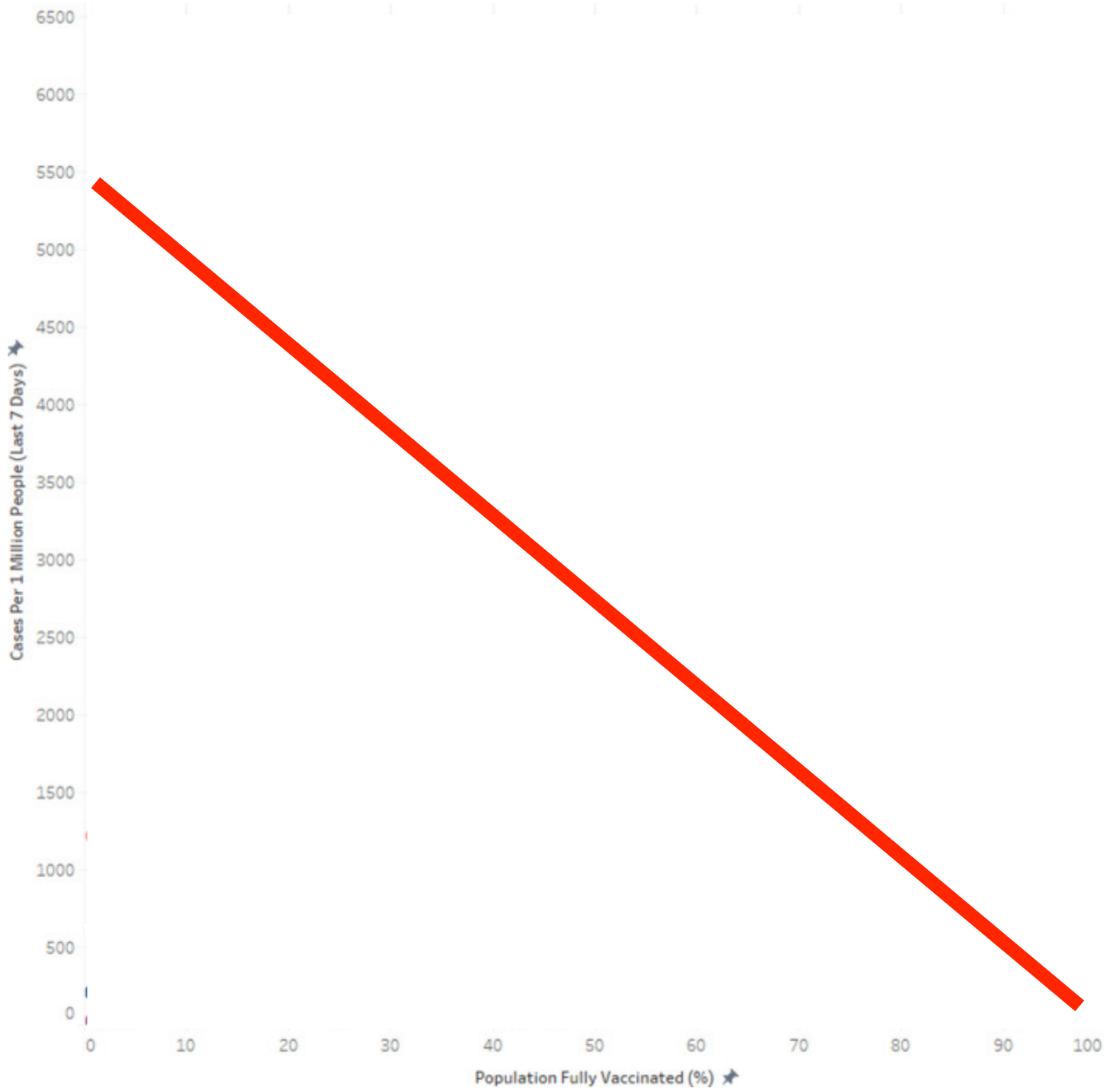
- Underreporting: x10 bis x100
- Alternative: Gar nicht berücksichtigen?
- Negative Korrelation mit Alter

ER + Tod / Corona + AE



Sind Impfdurchbrüche normal und erwartbar?

Cases / Million



% Vaccinated

Covid-Fälle gesamt

Table 2. COVID-19 cases by vaccination status between week 38 and week 41 2021

Cases reported by specimen date between week 38 and week 41 2021	Total	Unlinked*	Not vaccinated	Received one dose (1-20 days before specimen date)	Received one dose, ≥21 days before specimen date	Second dose ≥14 days before specimen date	Rates among persons vaccinated with 2 doses (per 100,000)	Rates among persons not vaccinated (per 100,000)
Under 18	397,882	24,292	351,148	10,698	11,001	-90%	314.1	3,013.6
18-29	62,885	7,512	20,902	758	8,404	-25%	462.1	615.4
30-39	92,257	7,346	21,726	636	6,545	+27%	956.7	751.1
40-49	130,904	7,297	13,022	293	3,800	+124%	1,731.3	772.9
50-59	88,020	4,790	5,399	80	1,632	+103%	1,075.3	528.6
60-69	45,155	2,614	1,872	24	617	+102%	704.1	347.1
70-79	27,360	1,559	658	12	215	+101%	537.9	267.6
≥80	11,907	854	382	7	215	+33%	406.8	304.1

Impfdurchbrüche

- Masern: > 80% Impfquote
- 76 Fälle 2020
- 12% Geimpft
- 88% Ungeimpft

CLINICAL SCIENCE

Immunogenicity and safety of the BNT162b2 mRNA COVID-19 vaccine in adult patients with autoimmune inflammatory rheumatic diseases and in the general population: a multicentre study

Victoria Furer [1,2] Tali Eviatar,[1,2] Devy Zisman,[3,4] Hagit Peleg,[5] Daphna Paran,[1,2] David Levartovsky,[2] Michael Zisapel,[1] Ofir Elalouf,[1,2] Ilana Kaufman,[1,2] Roni Meidan,[2,6] Adi Broyde,[1,2] Ari Polachek,[1,2] Jonathan Wollman,[1,2] Ira Litinsky,[1,2] Katya Meridor,[1,2] Hila Nochomovitz,[1,2] Adi Silberman,[1,2] Dana Rosenberg,[1,2] Joy Feld,[3] Amir Haddad,[3] Tal Gazzit,[3] Muna Elias,[3] Nizar Higazi,[3] Fadi Kharouf,[5,7] Gabi Shefer,[8] Orly Sharon,[8] Sara Pel,[2] Sharon Nevo,[2] Ori Elkayam[1,2]

- Deutlich reduzierte Schutzwirkung (>39%)
- Letaler Verlauf mit 1:340
- D: 1.5 Mio Patienten dieser Gruppe

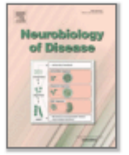
  (entspräche ca. 4.500 Todesfällen)

Neuroinflammation

ELSEVIER

Neurobiology of Disease

Volume 146, December 2020, 105131



The SARS-CoV-2 spike protein alters barrier function in 2D static and 3D microfluidic in-vitro models of the human blood–brain barrier

Tetyana P. Buzhdygan [a, b], Brandon J. DeOre [c], Abigail Baldwin-Leclair [c], Trent A. Bullock [a, b], Hannah M. McGary [a], Jana A. Khan [a], Roshanak Razmpour [a], Jonathan F. Hale [a], Peter A. Galie [c], Raghava Potula [a, b], Allison M. Andrews [a, b], Servio H. Ramirez [a, b, d]

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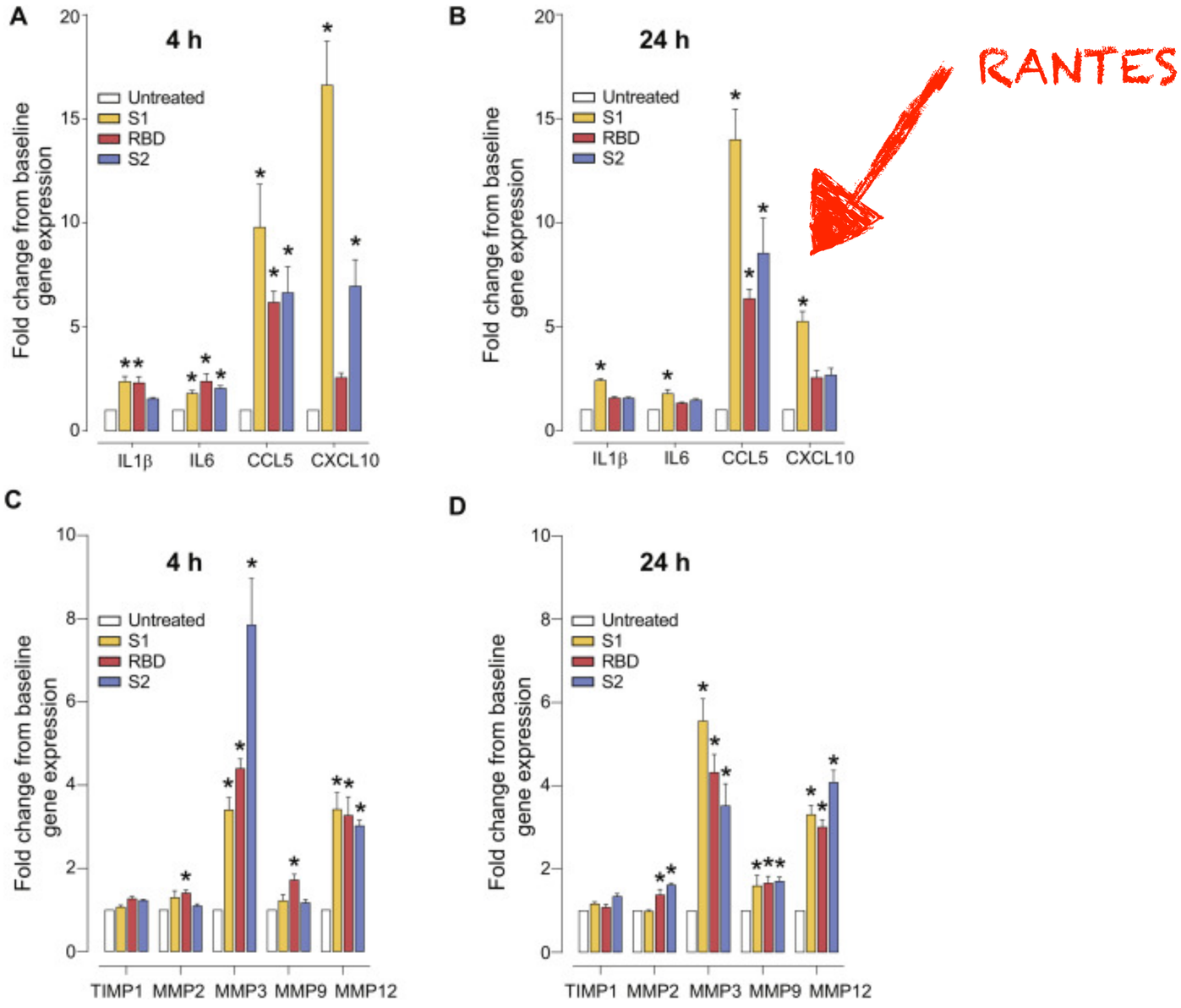
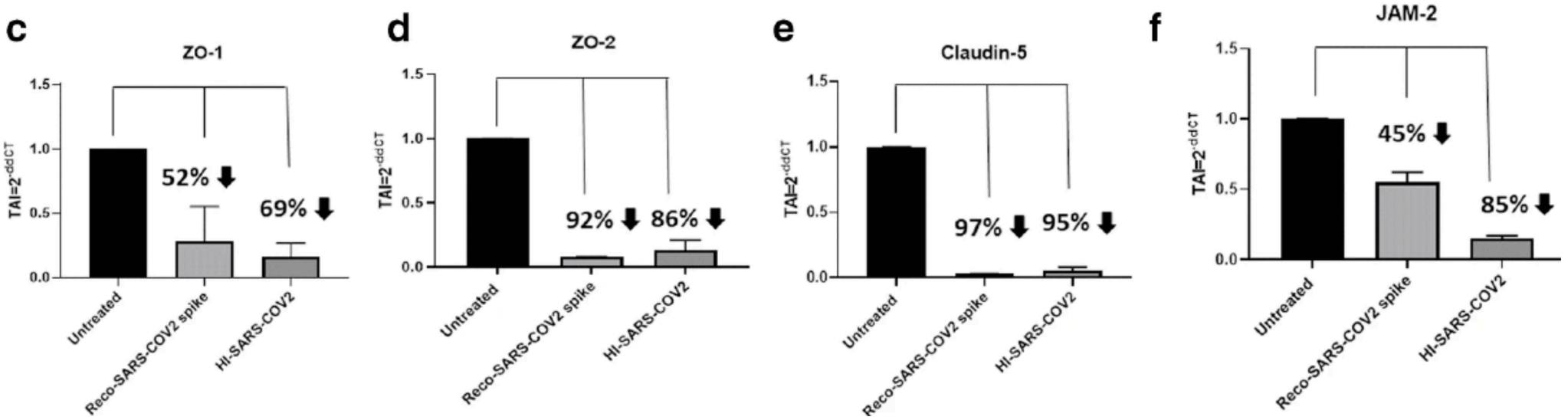
https://doi.org/10.1016/j.nbd.2020.105131

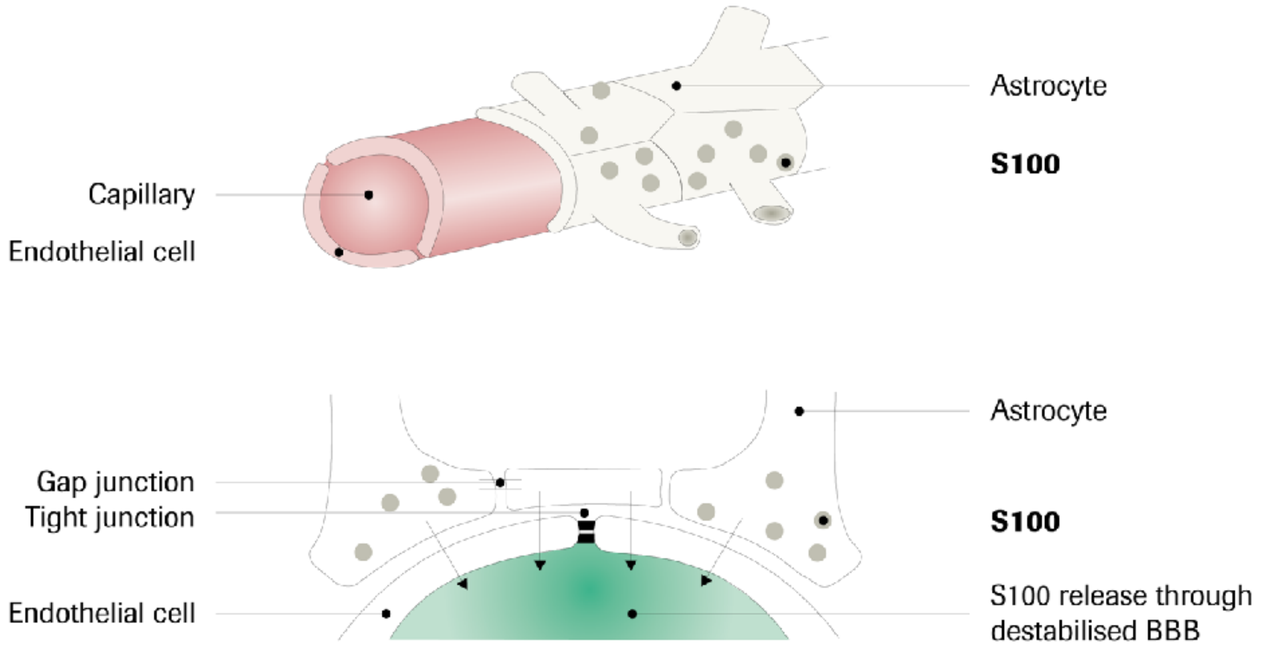


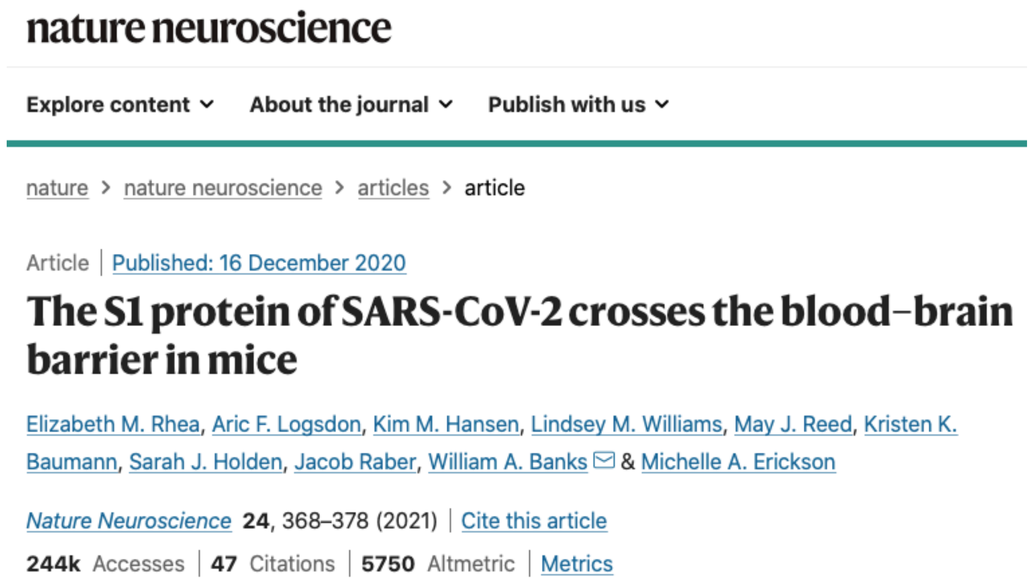
Tight-Junction-Proteine werden eliminiert



{Reynolds, 2021 #2748}

- S-100
- NSE
- a-1-Antitrypsin
- Zonulin
- TNF-a
- IL-6





nature neuroscience

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nature › nature neuroscience › articles › article

Article | Published: 16 December 2020

The S1 protein of SARS-CoV-2 crosses the blood–brain barrier in mice

Elizabeth M. Rhea, Aric F. Logsdon, Kim M. Hansen, Lindsey M. Williams, May J. Reed, Kristen K. Baumann, Sarah J. Holden, Jacob Raber, William A. Banks ✉ & Michelle A. Erickson

Nature Neuroscience **24**, 368–378 (2021) | Cite this article

244k Accesses | **47** Citations | **5750** Altmetric | Metrics

- MC >> M1
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- Rantes
- Endotheliitis
- Mitotoxischer Effekt

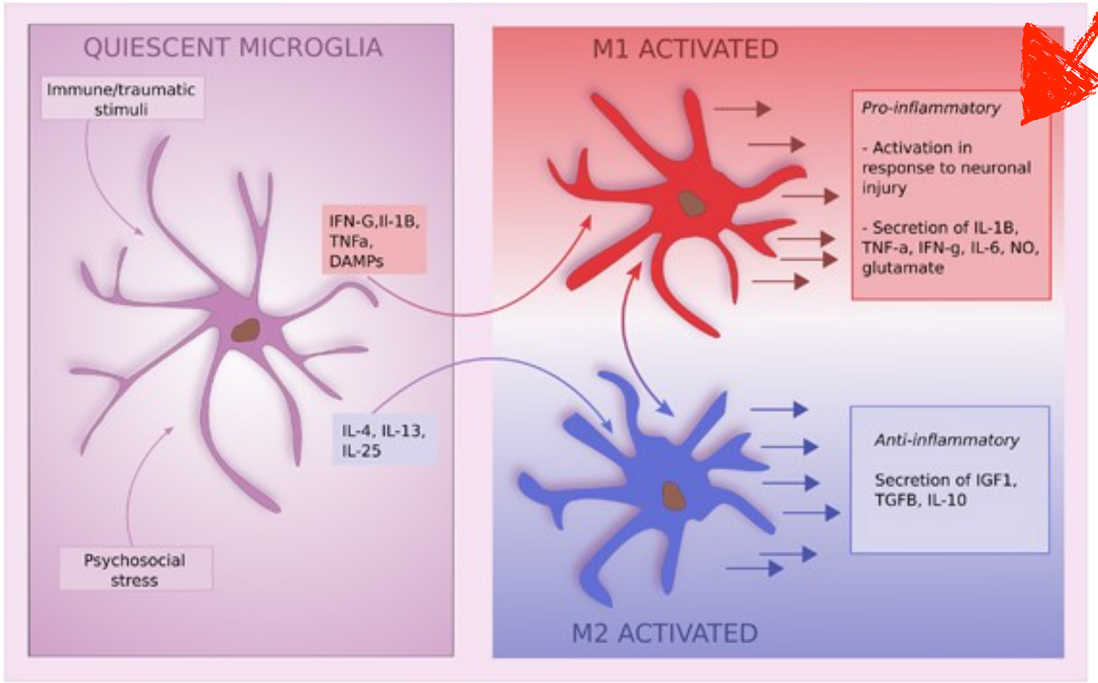
MINI REVIEW article
Front. Immunol., 20 March 2020 | https://doi.org/10.3389/fimmu.2020.00193

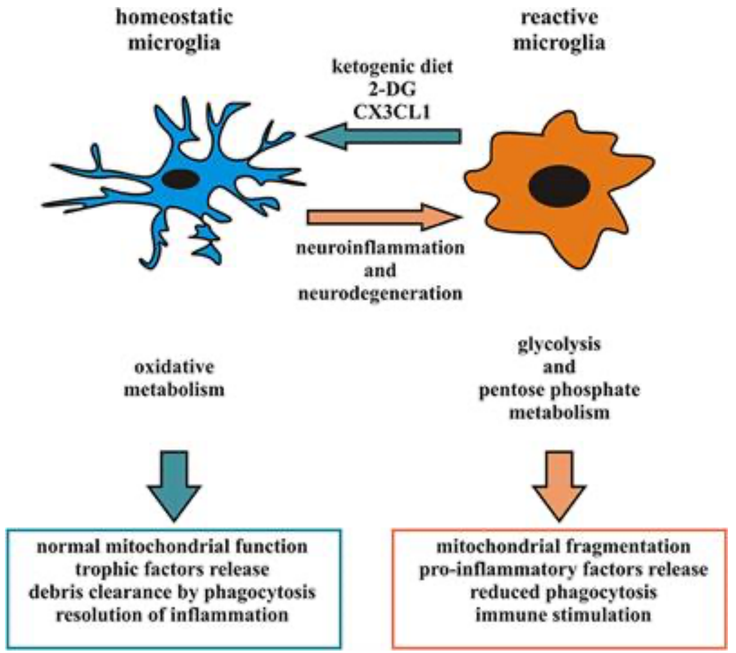
Metabolic Reprograming of Microglia in the Regulation of the Innate Inflammatory Response

Clotilde Lauro[1]* and Cristina Limatola[2,3]

[1]Department of Physiology and Pharmacology, Sapienza University of Rome, Rome, Italy
[2]Laboratory Affiliated to Istituto Pasteur Italia - Fondazione Cenci Bolognetti, Department of Physiology and Pharmacology, Sapienza University of Rome, Rome, Italy
[3]IRCCS NeuroMed, Pozzilli, Italy

IL-1B, TNF-a, IL-6, NO





Heliyon

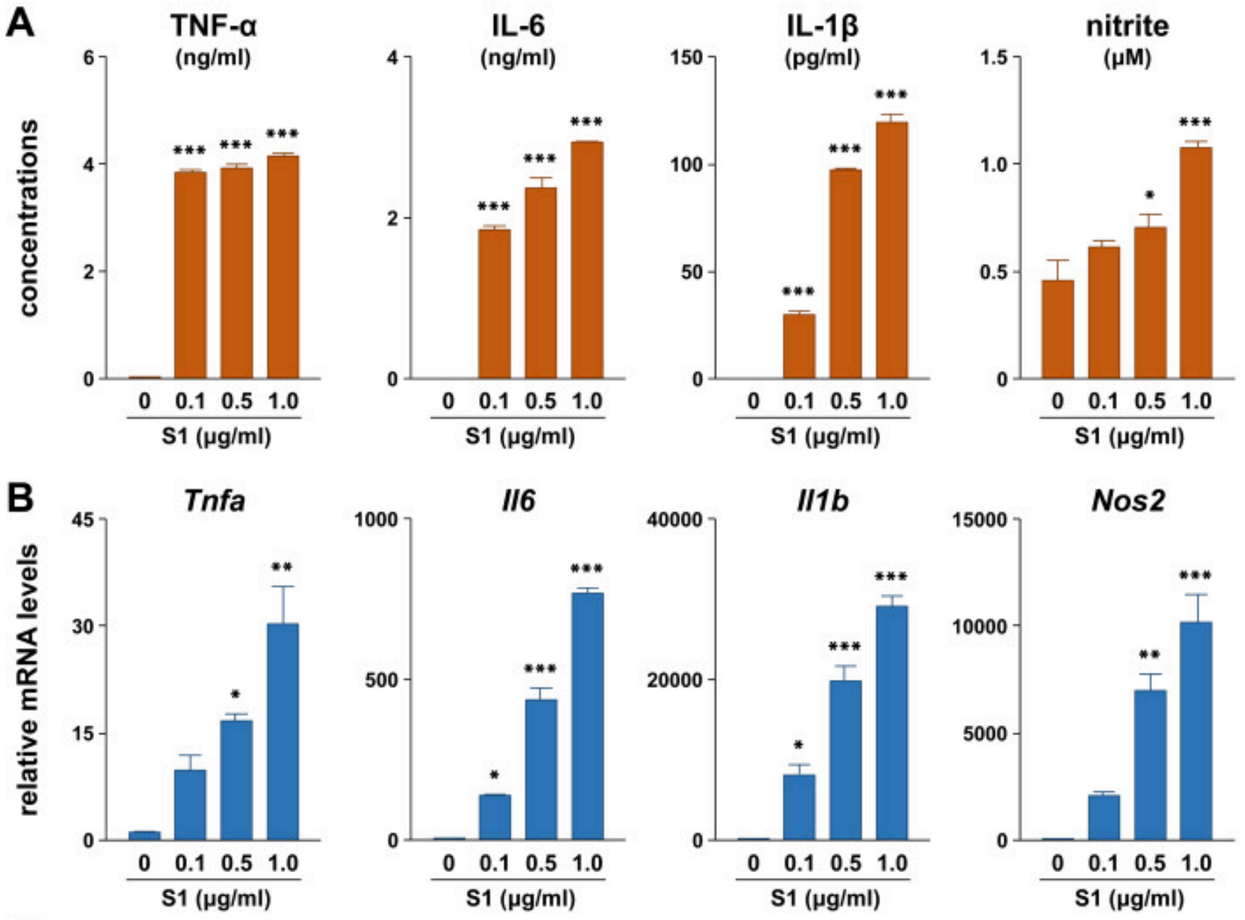
RESEARCH ARTICLE | VOLUME 7, ISSUE 2, E06187, FEBRUARY 01, 2021

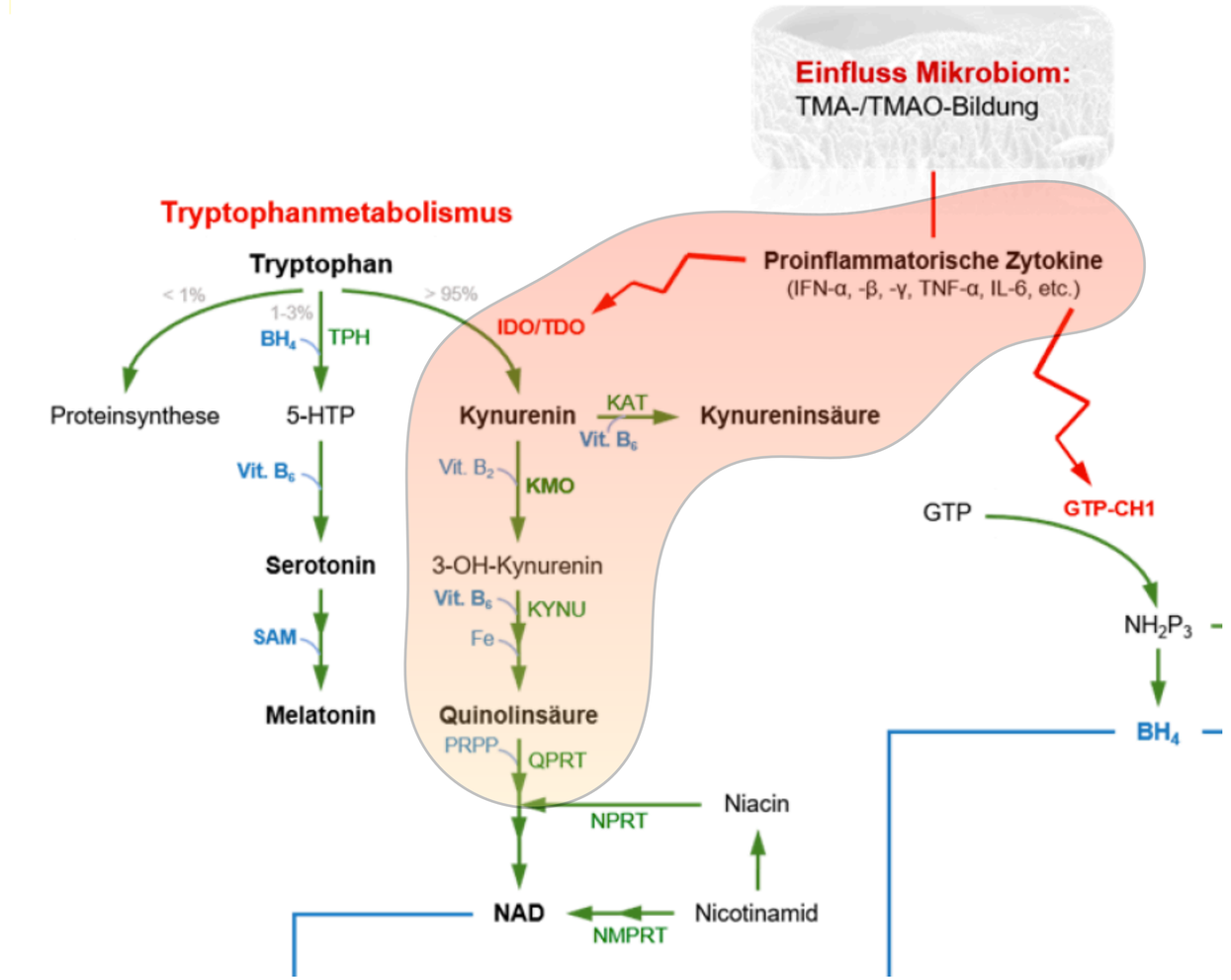
SARS-CoV-2 spike protein S1 subunit induces pro-inflammatory responses via toll-like receptor 4 signaling in murine and human macrophages

Ken Shirato • Takako Kizaki

Open Access • Published: February 02, 2021 • DOI: https://doi.org/10.1016/j.heliyon.2021.e06187 •

Check for updates





Epilepsia
Official Journal of the International League Against Epilepsy

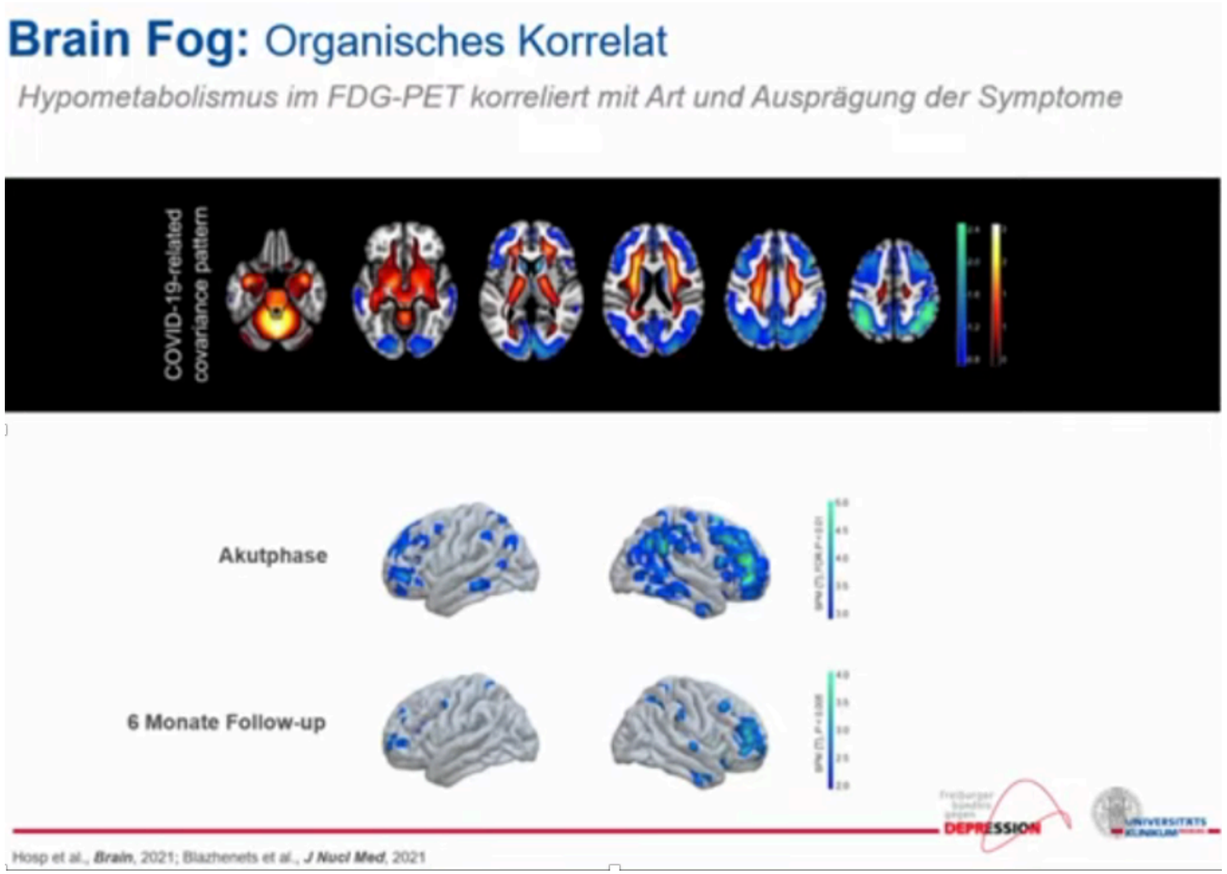
Immunity and Inflammation in Epilepsy (IIE2016) | Free Access

Neuroinflammation imaging markers for epileptogenesis

Matthias J. Koepp, Eric Årstad, Jens P. Bankstahl, Stefanie Dedeurwaerdere, Alon Friedman, Heidrun Potschka, Teresa Ravizza, William H. Theodore, Tallie Z. Baram

First published: 04 July 2017 | https://doi.org/10.1111/epi.13778 | Citations: 27

- Microvascular pathology with associated blood–brain barrier (BBB) dysfunction and neuroinflammation was shown to underlie long-lasting dysfunction of the neurovascular unit
- To identify these early pathobiologic-relevant disease-underlying processes, we developed MRI protocols as well as image and signal analysis methods to quantitatively follow microvascular pathology, specifically leaky BBB and electrophysiologic features of network dysfunction.



{Hosp, 2021 #2843}

Clinical & Experimental Immunology
The Journal of Translational Immunology
British Society for immunology

Original Article | Free Access

Intranasal delivery of dexamethasone efficiently controls LPS-induced murine neuroinflammation

G. Meneses, G. Gevorkian, A. Florentino, M. A. Bautista, A. Espinosa, G. Acero, G. Díaz, A. Fleury, I. N. Pérez Osorio, A. del Rey, G. Fragoso, E. Sciutto, H. Besedovsky,

First published: 28 July 2017 | https://doi.org/10.1111/cei.13018 | Citations: 19

Table 1. Cytokine (pg/mg protein) levels in soluble extract from brain of untreated and treated mice 4 days after lipopolysaccharide (LPS) injection

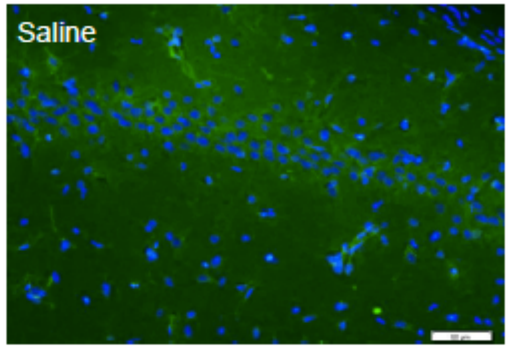
	TNF-α	IL-6	IL-1β
Non-treated	n.d.	0·72 ± 1·9[a]	12·16 ± 2·5[a]
LPS	n.d.	24·99 ± 8·7[b]	15·36 ± 1·4[b,c]
LPS + ISS i.v.	n.d.	19·44 ± 5·1[b,c]	15·03 ± 1·1[b,c]
LPS + ISS i.n.	n.d.	24·70 ± 7·9[b]	18·70 ± 6·8[b]
LPS + DX i.v.	n.d.	20·73 ± 12·6[b,c]	13·48 ± 3·1[a,b,c]
LPS + DX i.n.	n.d.	14·97 ± 5·1[c]	13·77 ± 2·9[a,c]

Intranasal Insulin Treatment of Traumatic Brain Injury

Fiona Brabazon[1], Guzal Khayrullina[1], William H. Frey[2], Kimberly R. Byrnes[1]

Department of Anatomy, Physiology and Genetics, Uniformed Services University

Department of Neurology, Oral Biology and Neuroscience, University of Minnesota



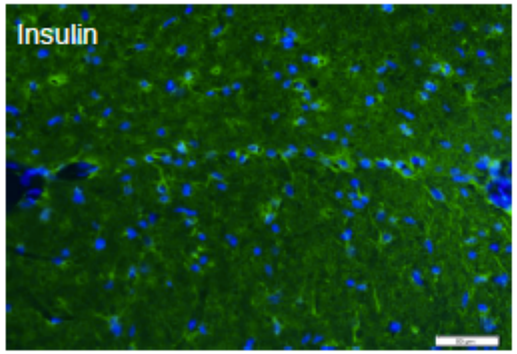
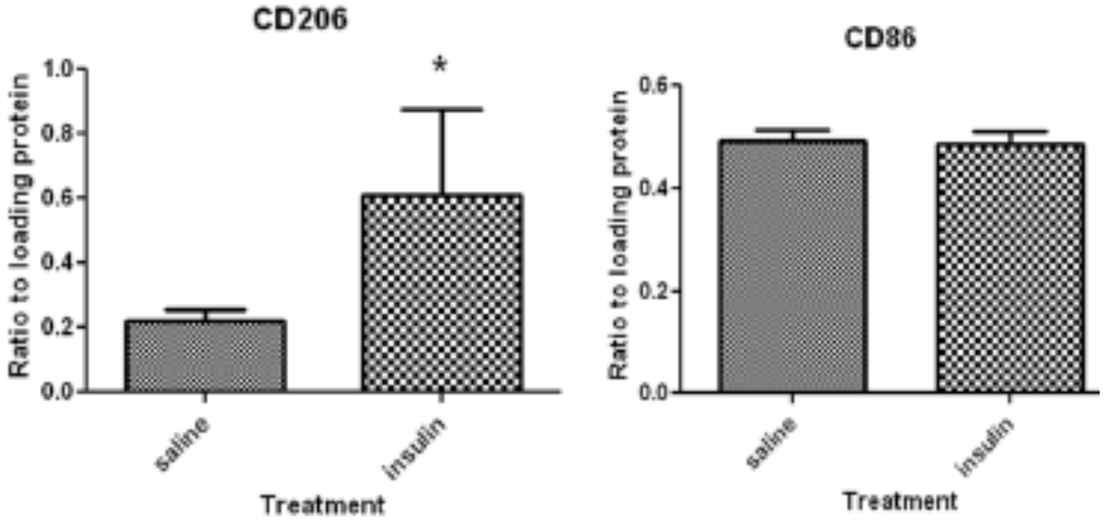


Figure 2. Intranasal Insulin increases the expression of anti-inflammatory microglia in the hippocampus



CD206 >> M2-Makrophagen

CD88 >> M1

Peptides 123 (2020) 170175

Contents lists available at ScienceDirect

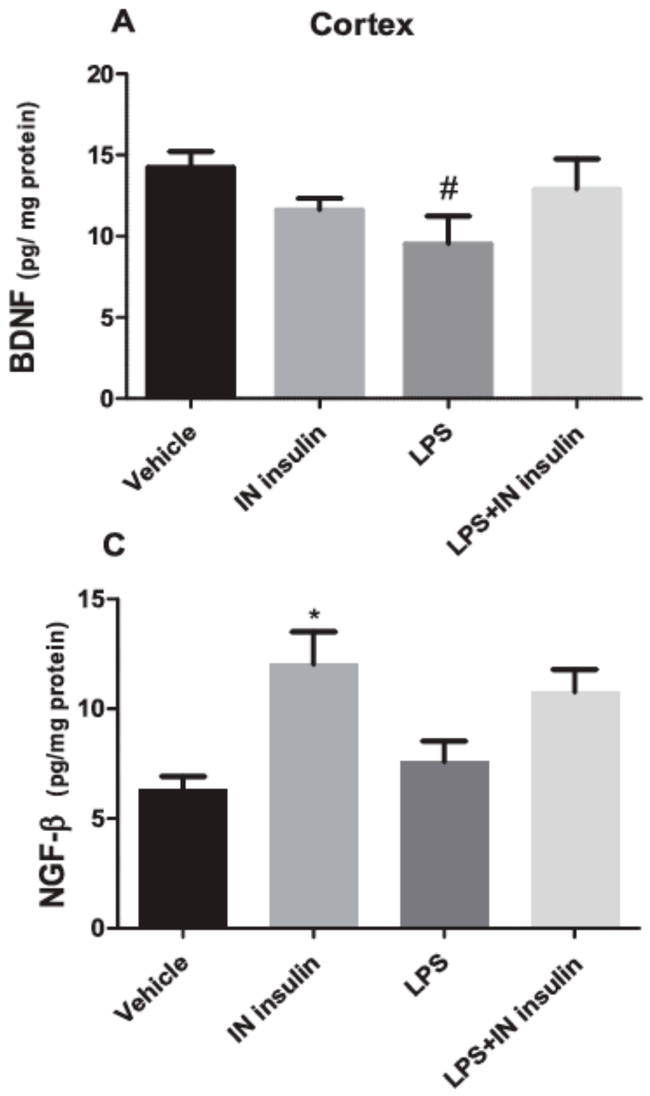
Peptides

journal homepage: www.elsevier.com/locate/peptides

Intranasal insulin treatment modulates the neurotropic, inflammatory, and oxidant mechanisms in the cortex and hippocampus in a low-grade inflammation model

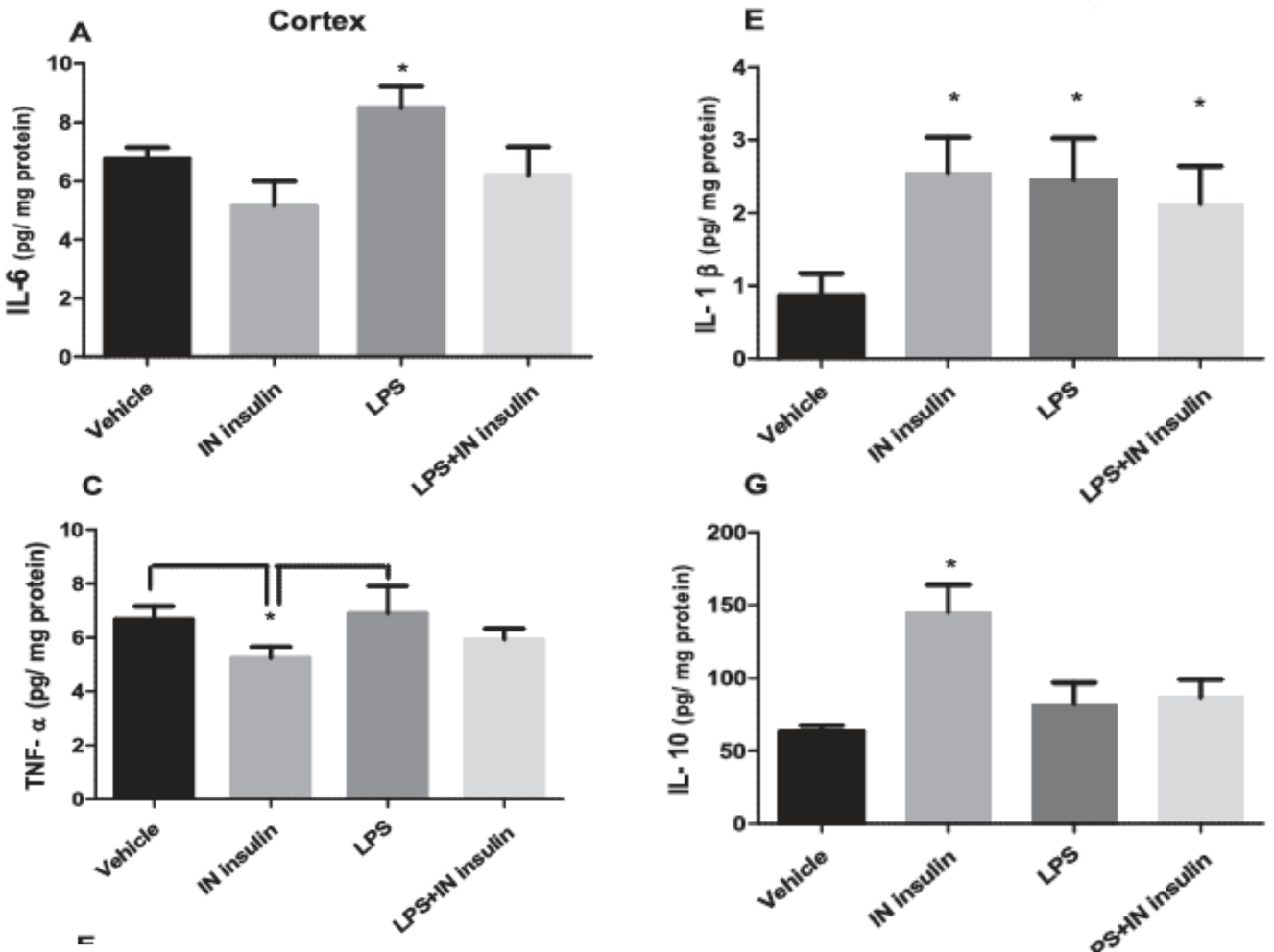
Kellen Ugioni Simon[a], Elias Wiggers Neto[a], Natalia dos Santos Tramontin[a], Paula Bortoluzzi Canteiro[a], Barbara da Costa Pereira[a], Rubya Pereira Zaccaron[a], Paulo Cesar Lock Silveira[a], Alexandre Pastoris Muller[a,b,*]

[a] Programa de Pós-Graduação em Ciências da Saúde, Universidade do Extremo Sul Catarinense (UNESC), 88806-00 Criciúma, SC, Brasil
[b] Programa de Pós Graduação em Farmacologia, Universidade Federal de Santa Catarina (UFSC), 88040-900 Florianópolis, SC, Brasil



Regenerationsleistung steigt

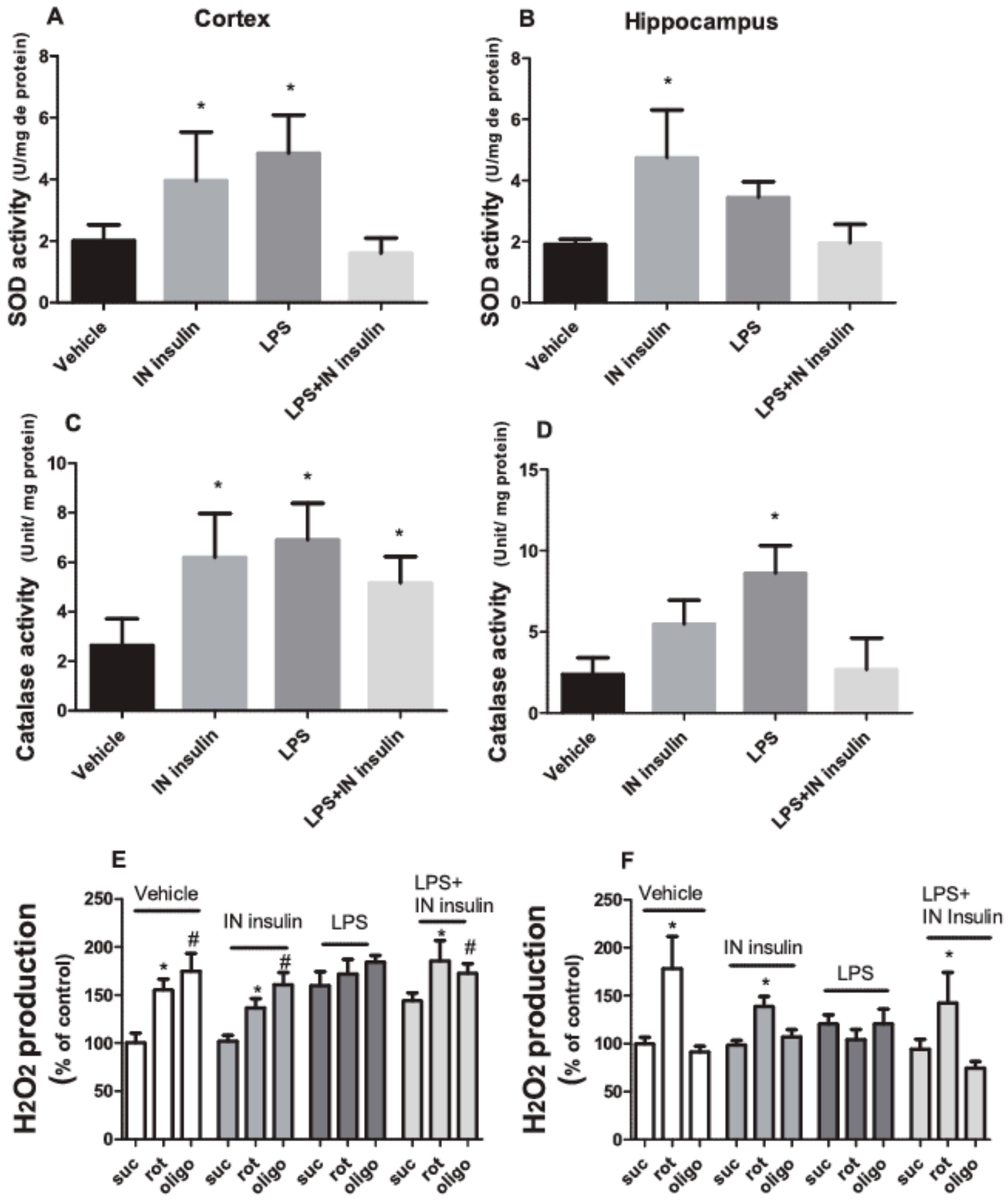
PIC sinken



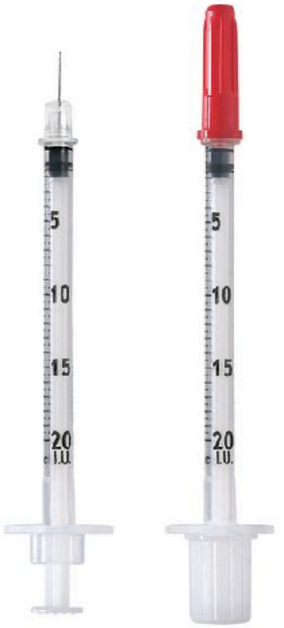
IN Insulin

CAVE:

AOC sinkt

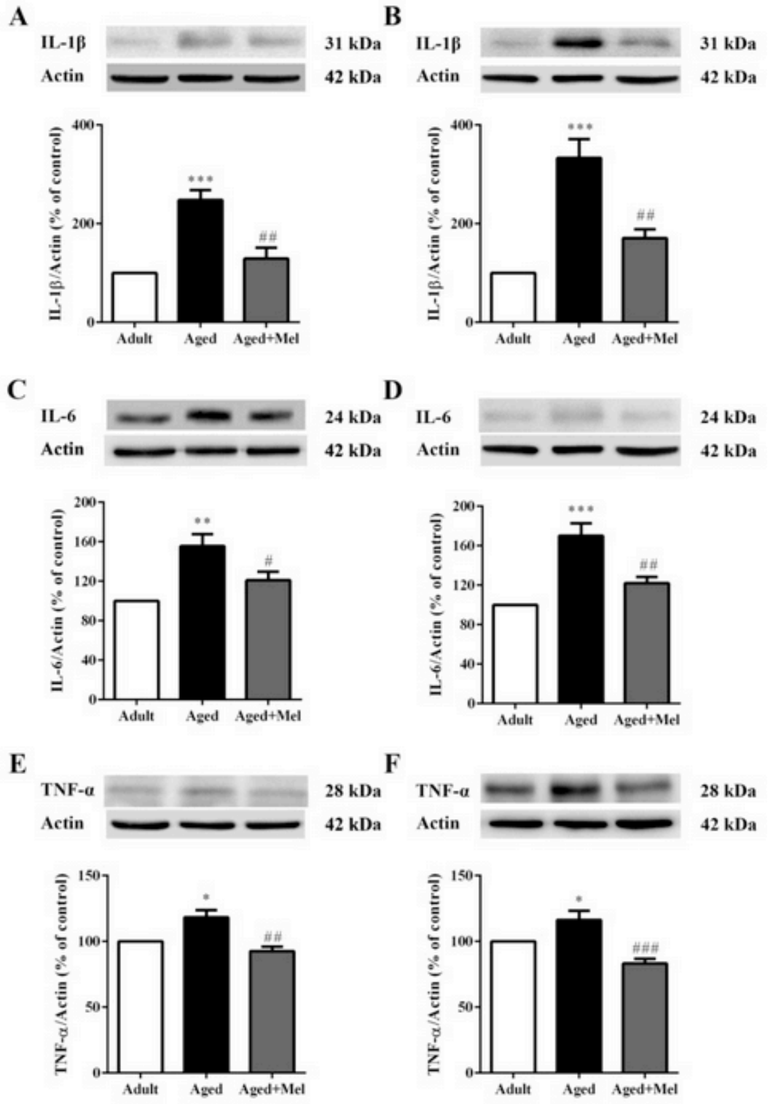


- DX (Dexamethason) IN
  - < 0,5ml / Seite / 5 Min. / d
  - Standard Dexa: 4mg/ml
  - KG x 0.1, ev. steigern auf KG x 0.2
- Insulin IN
  - 10 IU / Seite / d
  - Praktisch: 1ml/20 IU >> 2x0.5ml



High-Dose Melatonin

Hippocampus



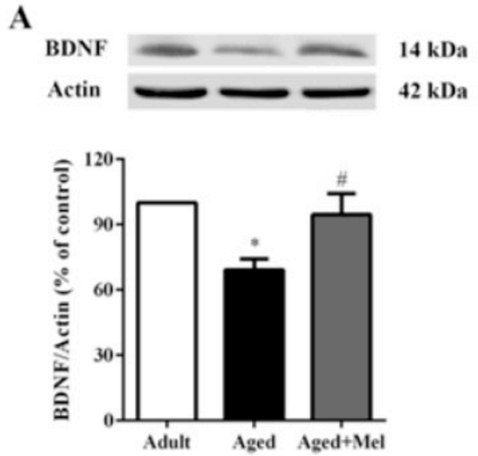
PFC

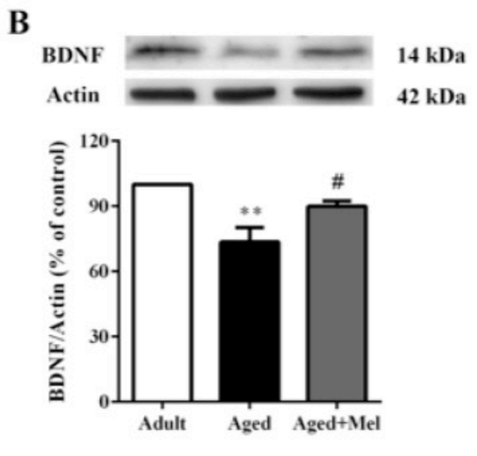
Melatonin 1mg/kg/d

{Permpoonputtana, 2018}

High-Dose Melatonin

Hippocampus





PFC

{Permpoonputtana, 2018}

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Review

Resolving neuroinflammation, the therapeutic potential of the anti-malaria drug family of artemisinin

Zhe Shi[a,1], Ying Chen[b,1], Cong Lu[c], Li-ming Dong[c], Jing-wei Lv[c], Qin-hui Tuo[a,d], Li Qin[a], Shao-wu Cheng[e], Lan-lan Bu[d], Na Lin[b], Xiao-xin Zhu[b,*], Duan-fang Liao[a,*], Xin-min Liu[a,c,*]

[a] *Division of Stem Cell Regulation and Application, Key Laboratory for Quality Evaluation of Bulk Herbs of Hunan Province, Hunan University of Chinese Medicine, Changsha 410208, Hunan, China*

[b] *Institute of Chinese Materia Medica, China Academy of Chinese Medical Sciences, Beijing 100700, China*

[c] *Research Center for Pharmacology and Toxicology, Institute of Medicinal Plant Development (IMPLAD), Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing 100193, China*

[d] *Medical School, Hunan University of Chinese Medicine, Changsha 410208, Hunan, China*

[e] *The Key Laboratory of Hunan Province for Integrated Traditional Chinese and Western Medicine on Prevention and Treatment of Cardio-Cerebral Diseases, Hunan University of Chinese Medicine, Changsha 410208, Hunan, China*

Nanoparticle-induced inflammation

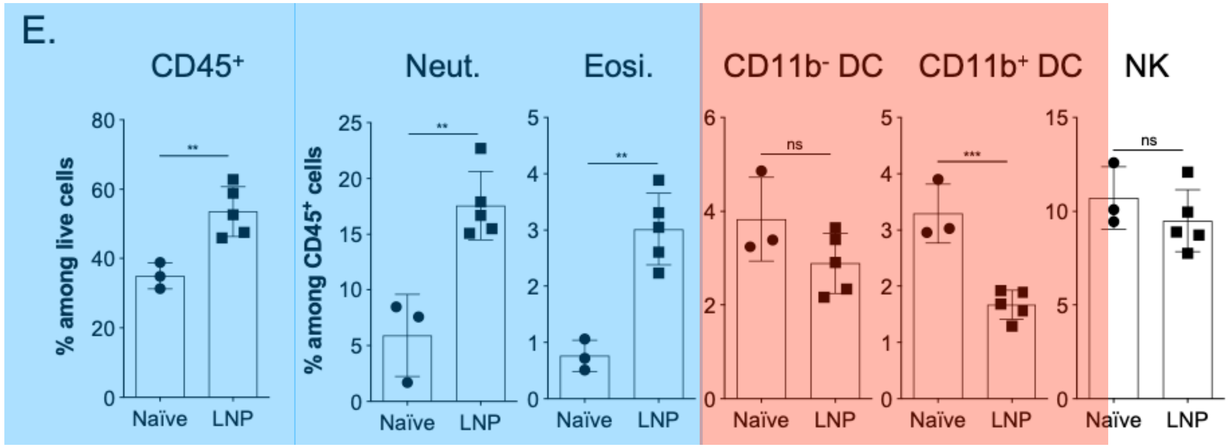
New Results

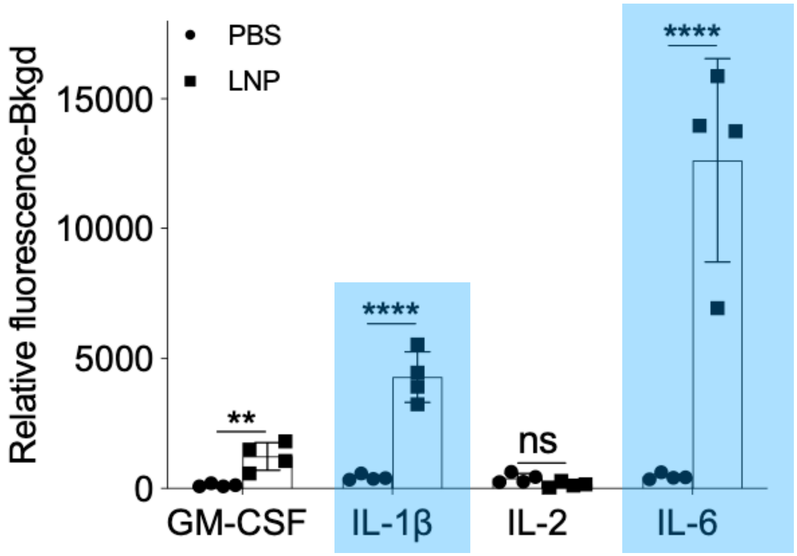
The mRNA-LNP platform's lipid nanoparticle component used in preclinical vaccine studies is highly inflammatory

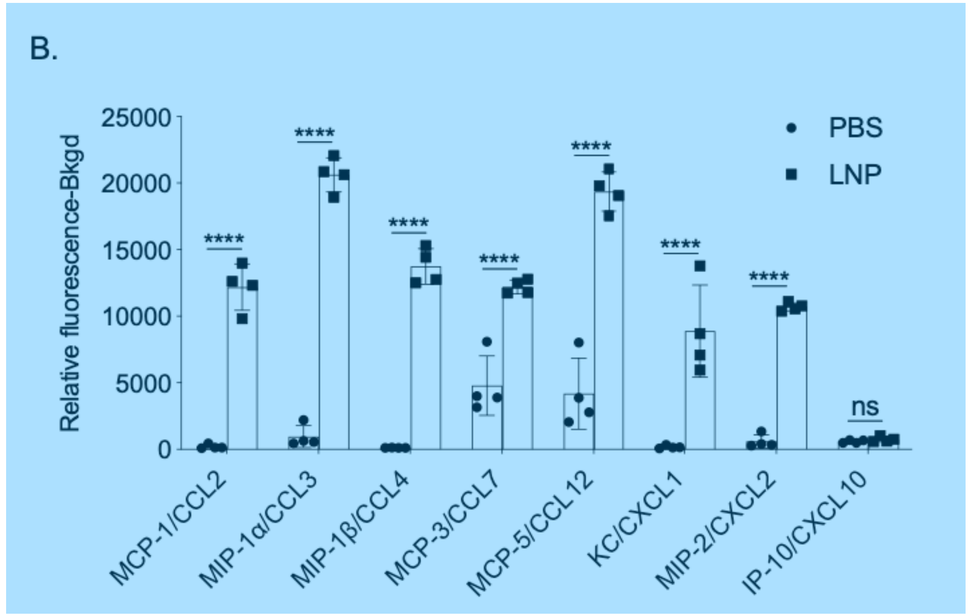
Sonia Ndeupen, Zhen Qin, Sonya Jacobsen, Henri Estanbouli, Aurélie Bouteau, Botond Z. Igyártó

doi: https://doi.org/10.1101/2021.03.04.430128

Inflammation + Immune suppression







LNP-Anreicherung im Gehirn

› Mol Ther. 2017 Jun 7;25(6):1316-1327. doi: 10.1016/j.ymthe.2017.03.035. Epub 2017 Apr 27.

Preclinical and Clinical Demonstration of Immunogenicity by mRNA Vaccines against H10N8 and H7N9 Influenza Viruses

Kapil Bahl [1], Joe J Senn [2], Olga Yuzhakov [1], Alex Bulychev [2], Luis A Brito [2], Kimberly J Hassett [1], Michael E Laska [2], Mike Smith [2], Örn Almarsson [2], James Thompson [2], Amilcar Mick Ribeiro [1], Mike Watson [1], Tal Zaks [2], Giuseppe Ciaramella [3]

Affiliations + expand

PMID: 28457665 PMCID: PMC5475249 DOI: 10.1016/j.ymthe.2017.03.035

Feature

Brain safety concerns of nanomedicines: The need for a specific regulatory framework

Bartlomiej Szabat-Iriaka [a], Marc Le Borgne [a, b]

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https://doi.org/10.1016/j.drudis.2021.06.011

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Special Issue

Prooxidant Mechanisms in Toxicology

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Volume 2013 | Article ID 942916 | https://doi.org/10.1155/2013/942916

Show citation

Mechanisms of Nanoparticle-Induced Oxidative Stress and Toxicity

Amruta Manke,[1] Liying Wang,[2] and **Yon Rojanasakul**[1]

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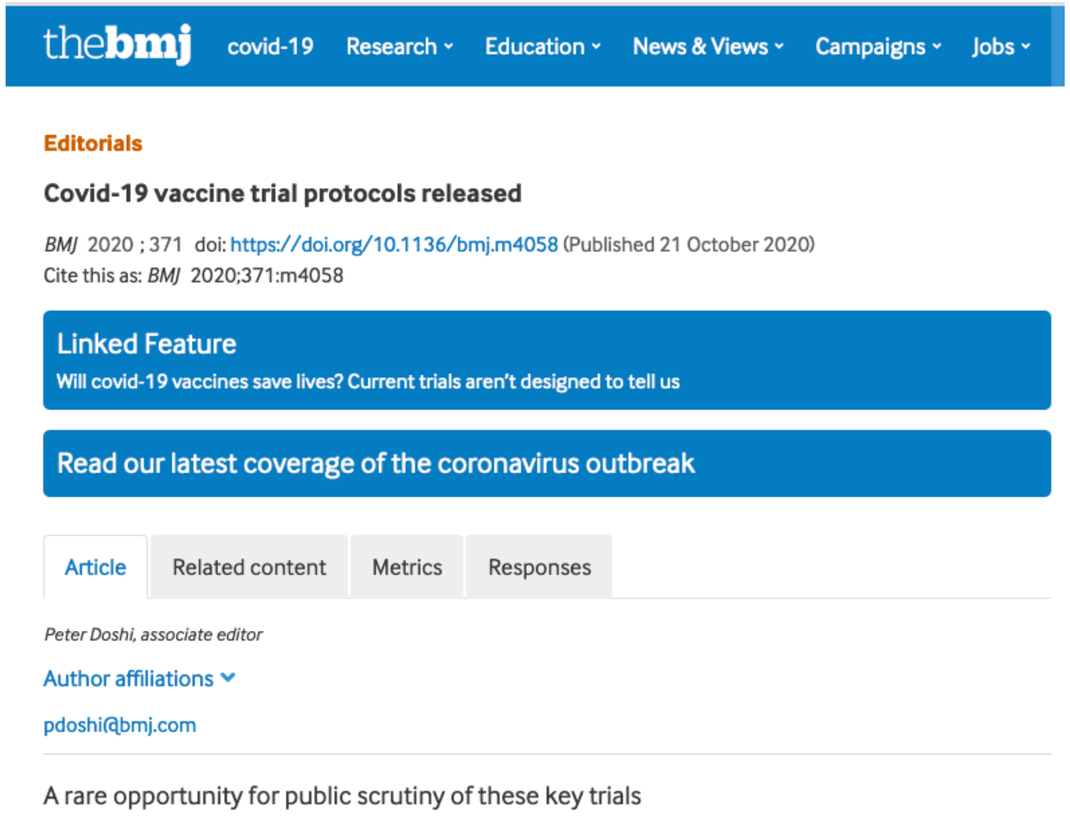
Academic Editor: Nikhat J. Siddiqi

Received	Accepted	Published
09 May 2013	16 Jul 2013	20 Aug 2013



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Editorials

Covid-19 vaccine trial protocols released

BMJ 2020 ; 371 doi: https://doi.org/10.1136/bmj.m4058 (Published 21 October 2020)
Cite this as: *BMJ* 2020;371:m4058

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Peter Doshi, associate editor

Author affiliations

pdoshi@bmj.com

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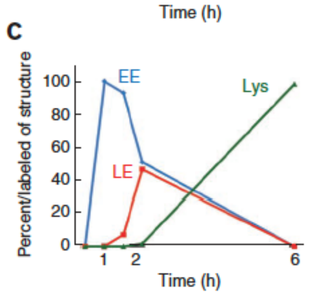
- Bone marrow
- Liver
- Lungs
- Spleen
- Kidneys

EUROPEAN MEDICINES AGENCY
SCIENCE MEDICINES HEALTH

11 March 2021
EMA/15689/2021 Corr.1*[1]
Committee for Medicinal Products for Human Use (CHMP)



Assessment report

COVID-19 Vaccine Moderna

Common name: COVID-19 mRNA Vaccine (nucleoside-modified)

Procedure No. EMEA/H/C/005791/0000

mRNA-Nachweis in allen Geweben

Autophagy for resolve?

› Autophagy. 2021 Apr;17(4):872-887. doi: 10.1080/15548627.2020.1739442. Epub 2020 Mar 15.

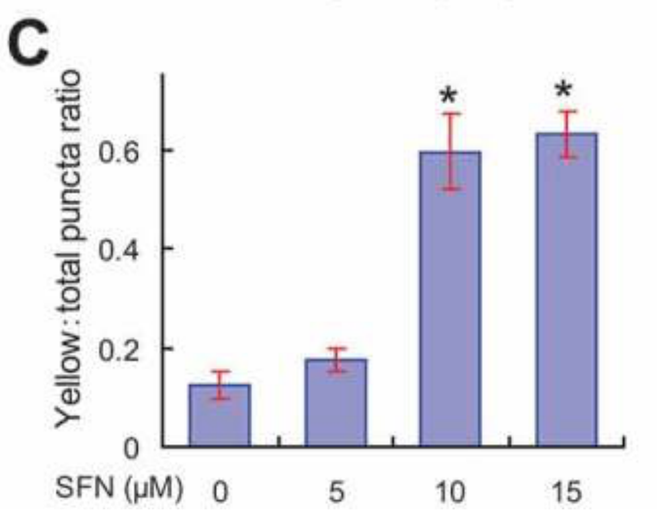
Sulforaphane Activates a lysosome-dependent transcriptional program to mitigate oxidative stress

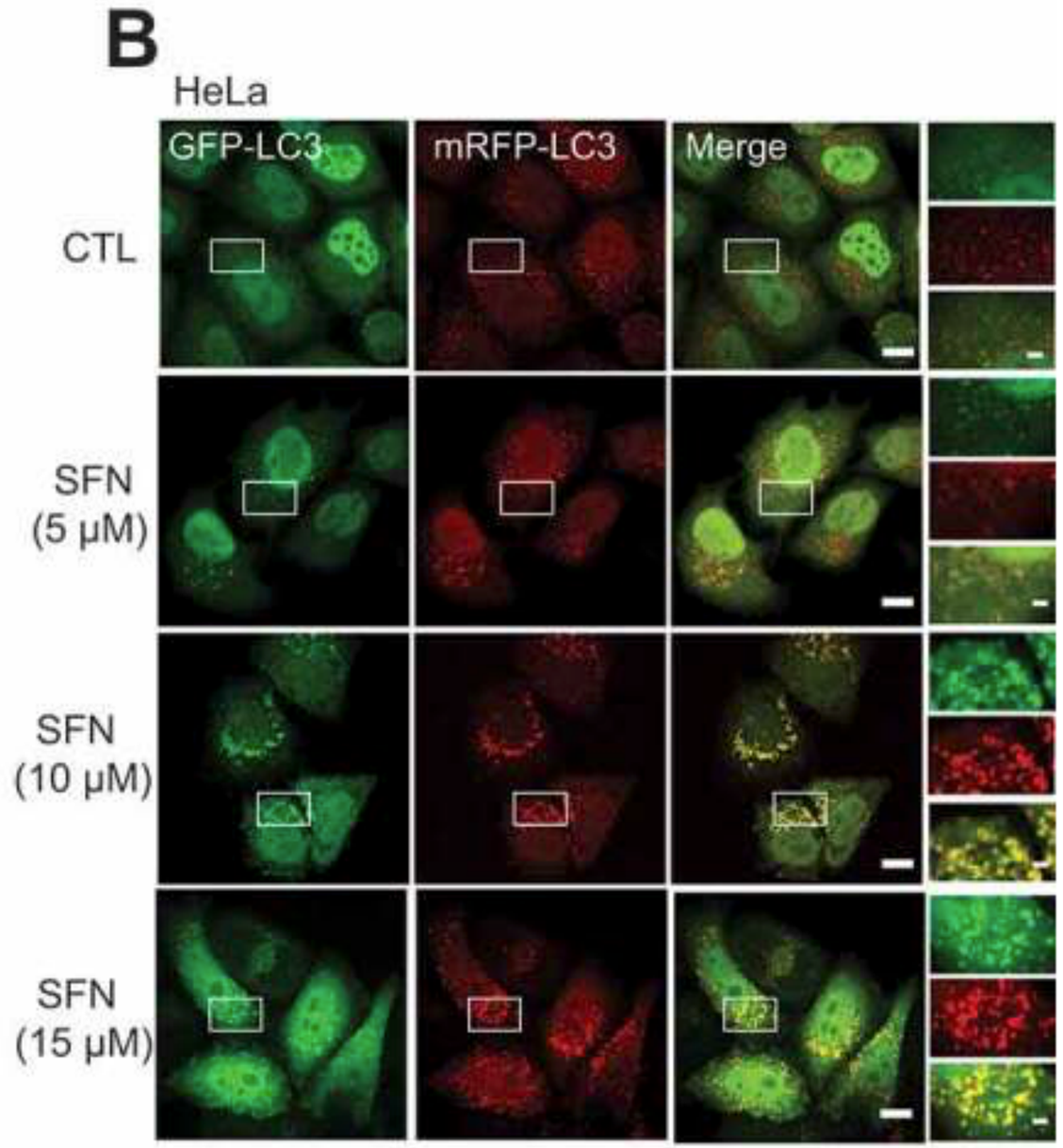
Dan Li [1 2], Rong Shao [1], Na Wang [1 2], Nan Zhou [1 2], Kaili Du [1 2], Jiahui Shi [1], Yihan Wang [1 2], Zhuangzhuang Zhao [1 2], Xin Ye [1], Xiaoli Zhang [2], Haoxing Xu [2]

Affiliations + expand

PMID: 32138578 PMCID: PMC8078734 DOI: 10.1080/15548627.2020.1739442

LC3: Lysosomale Biogenese





Review Article | Free Access

Targeting autophagy using natural compounds for cancer prevention and therapy

Shuo Deng PhD, Muthu K. Shanmugam PhD, Alan Prem Kumar PhD, Celestial T. Yap PhD, Gautam Sethi PhD, Anupam Bishayee PhD.

First published: 12 February 2019 | https://doi.org/10.1002/cncr.31978 | Citations: 112

Weitere Autophagie-Aktivatoren:

- Artemisin
- Curcumin (Lipo Curcumin)
- Resveratrol (Polyphenole)
- Tocotrienol (Mitoformula)

REVIEW article

Front. Cell Dev. Biol., 22 September 2020 | https://doi.org/10.3389/fcell.2020.555409

Natural Compounds and Autophagy: Allies Against Neurodegeneration

Alessandra Stacchiotti[1,2*] and Giovanni Corsetti[1]

[1]Division of Anatomy and Physiopathology, Department of Clinical and Experimental Sciences, University of Brescia, Brescia, Italy
[2]Interdepartmental University Center of Research "Adaptation and Regeneration of Tissues and Organs (ARTO)," University of Brescia, Brescia, Italy

Wirkstoff	Dosis (Tiermodell!)
Quercetin	5mg/kg/d
Berberine	40mg/kg/d
Melatonin	5mg/kg/d
Trehalose	2% (ca. 0,2g/kg/d)

ADE

ADE from past: HIV

nature

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nature › perspectives › article

Perspective | Published: 13 July 2020

A perspective on potential antibody-dependent enhancement of SARS-CoV-2

Ann M. Arvin, Katja Fink, Michael A. Schmid, Andrea Cathcart, Roberto Spreafico, Colin Havenar-Daughton, Antonio Lanzavecchia, Davide Corti & Herbert W. Virgin

Nature **584**, 353–363(2020) | Cite this article

126k Accesses | **94** Citations | **1120** Altmetric | Metrics

ADE from past: Dengue

- Data from the study of SARS-CoV and other respiratory viruses suggest that anti-SARS-CoV-2 antibodies could exacerbate COVID-19 through antibody-dependent enhancement (ADE)
- Previous respiratory syncytial virus and dengue virus vaccine studies revealed human clinical safety risks related to ADE, resulting in failed vaccine trials.

nature › nature microbiology › perspectives › article

Perspective | Published: 09 September 2020

Antibody-dependent enhancement and SARS-CoV-2 vaccines and therapies

Wen Shi Lee, Adam K. Wheatley, Stephen J. Kent & Brandon J. DeKosky

Nature Microbiology **5**, 1185–1191(2020) | Cite this article

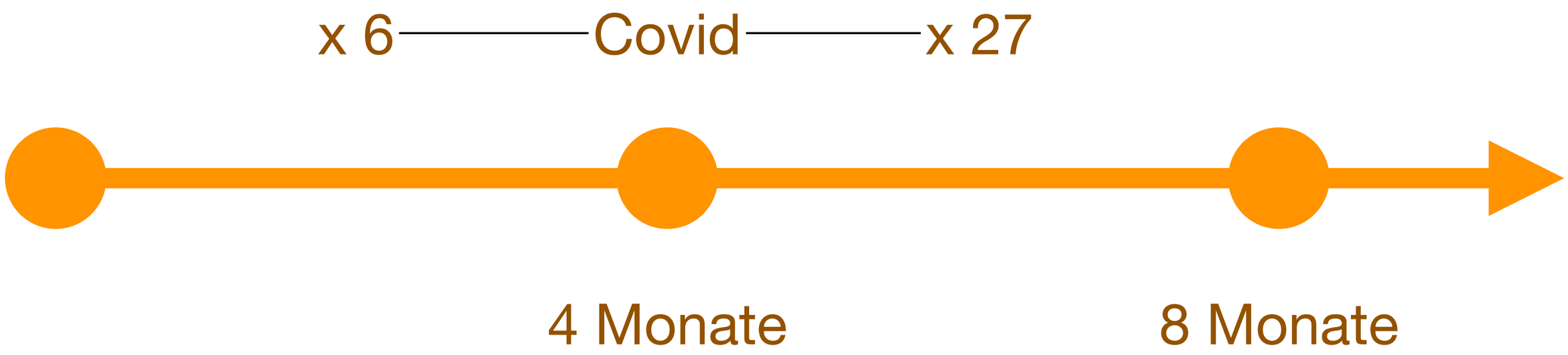
- „Impfdurchbrüche“ müssten dahingehend analysiert werden

Comments (410)

Comparing SARS-CoV-2 natural immunity to vaccine-induced immunity: reinfections versus breakthrough infections

Sivan Gazit, Roei Shlezinger, Galit Perez, Roni Lotan, Asaf Peretz, Amir Ben-Tov, Dani Cohen, Khitam Muhsen, Gabriel Chodick, Tal Patalon

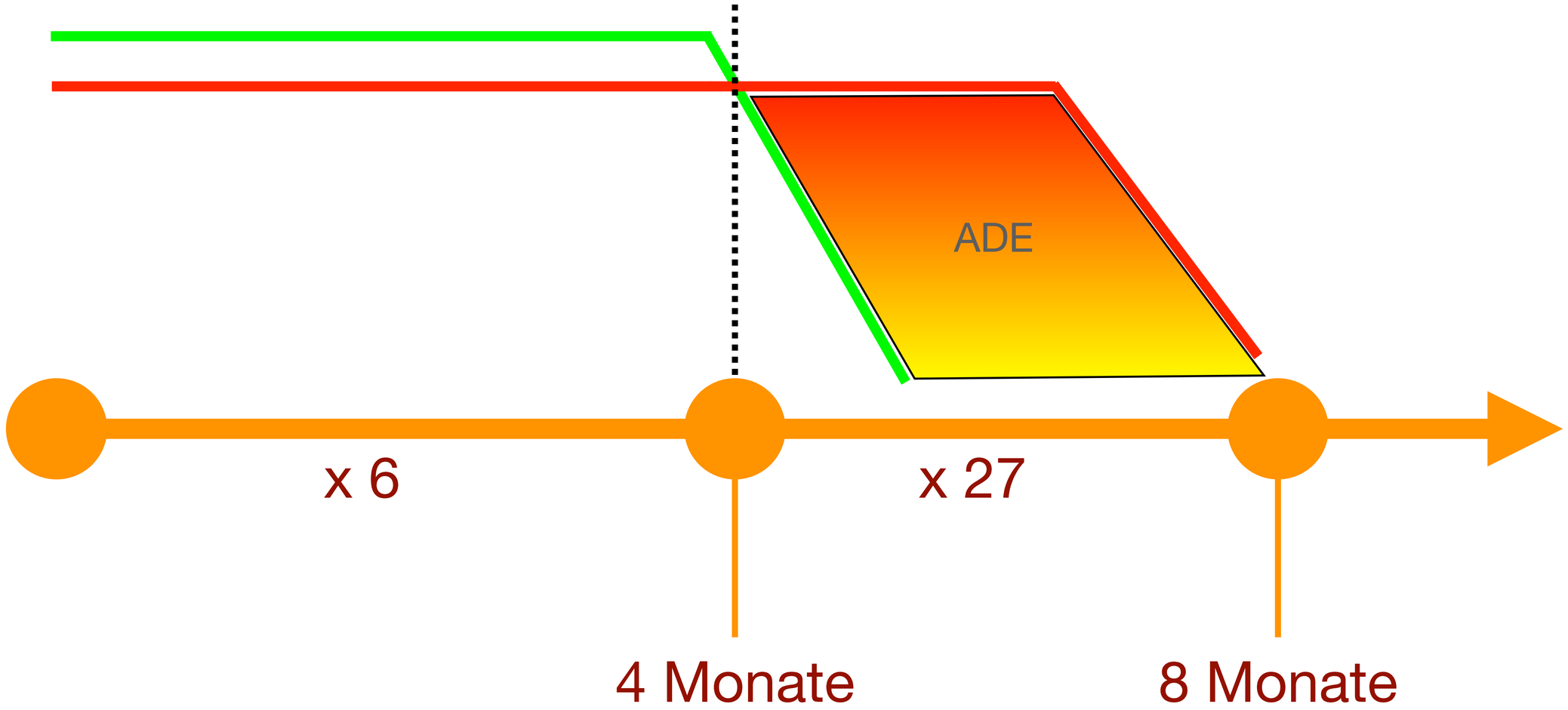
doi: https://doi.org/10.1101/2021.08.24.21262415



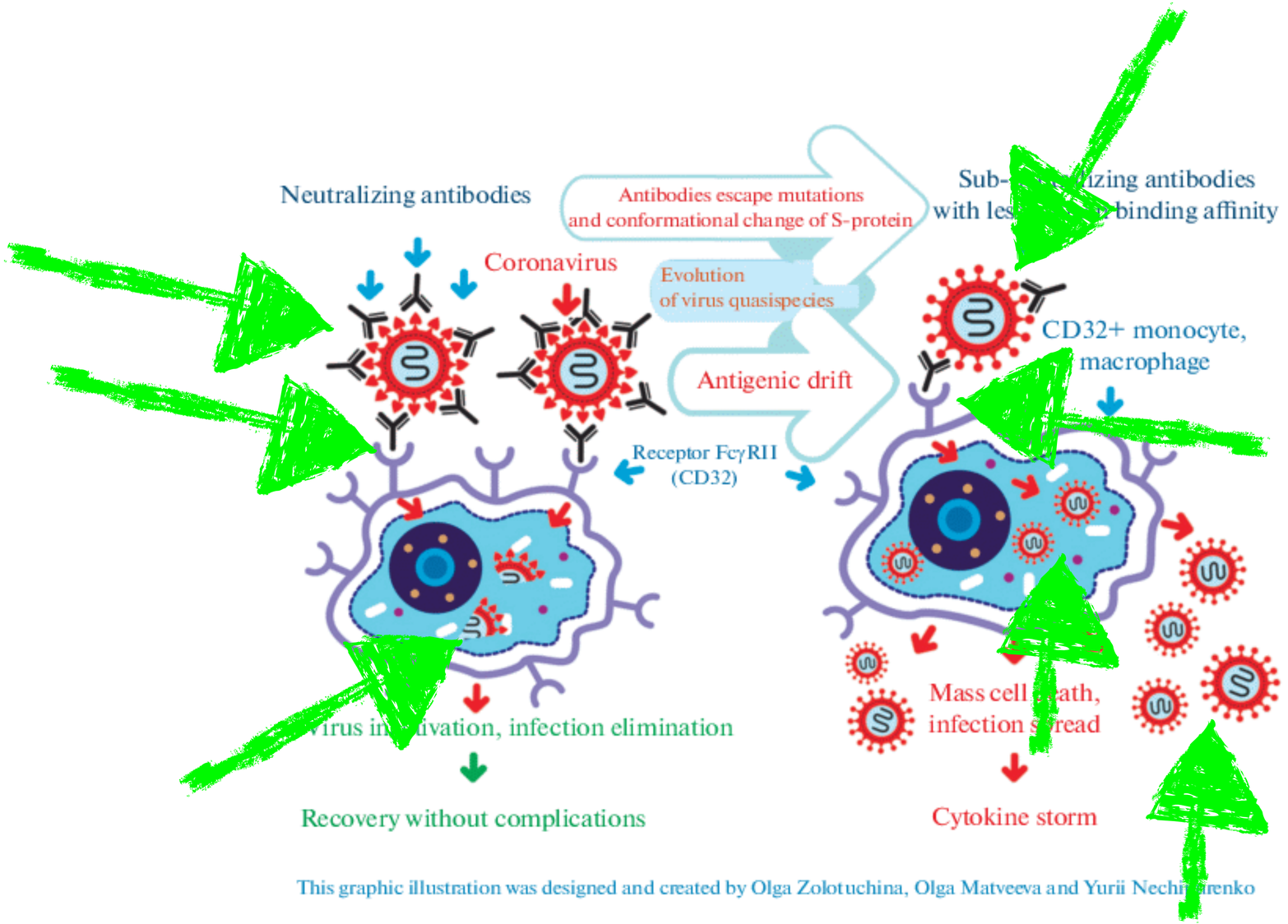
Allgemein: +100%

ADE-AB Infektionsverstärkend

N-AB Neutralisierend



ADE



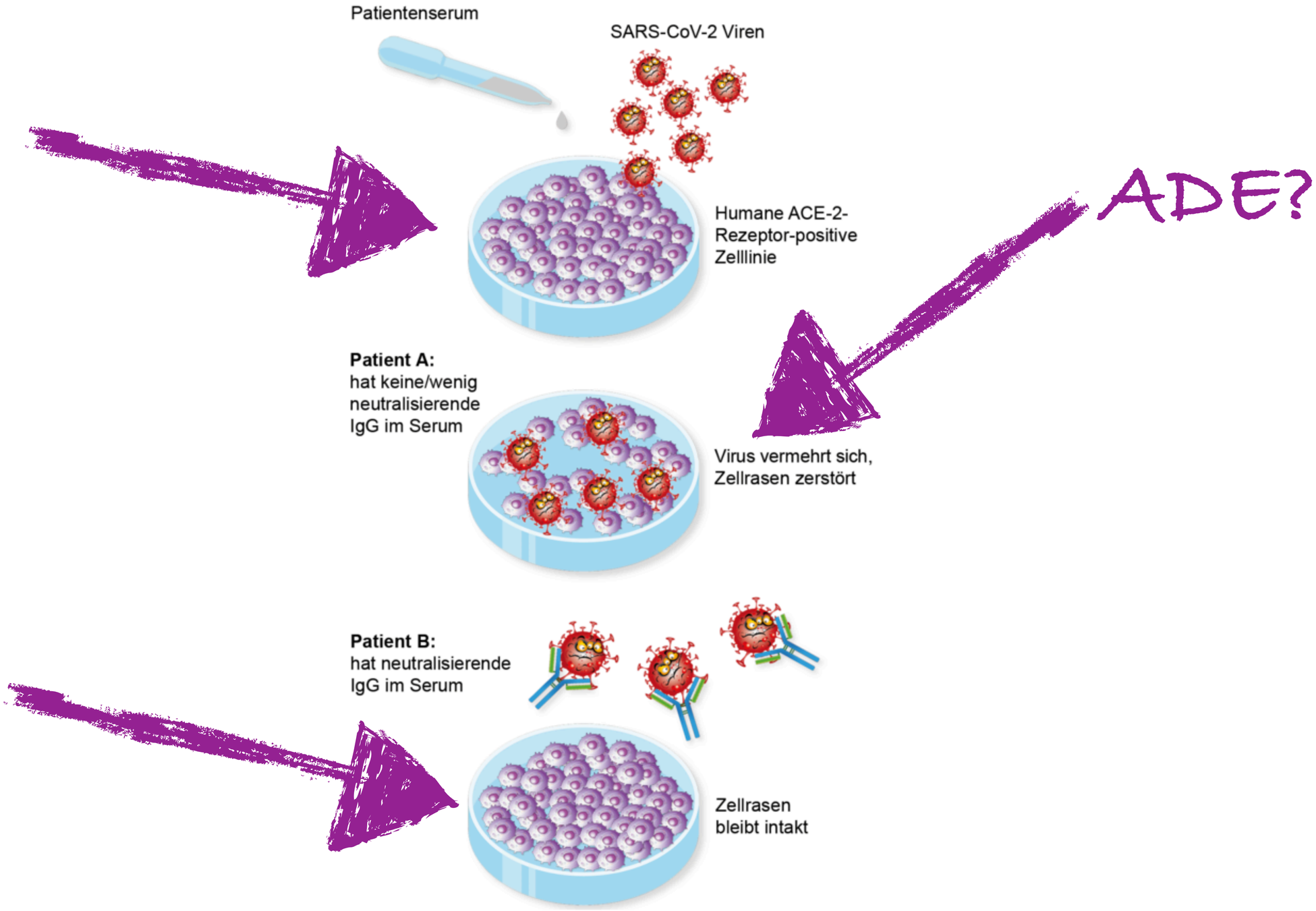


Abb. 1 Testprinzip des klassischen Plaque-Reduktions-Neutralisationstests (PRNT).

Anforderungsbogen COVID-19

SARS-CoV-2: Nachweis einer akuten oder zurückliegenden COVID-19-Infektion

Direkter Erregernachweis mit PCR-Verfahren

☐ K345 **SARS-CoV-2-qPCR Basis** Nasen- / Rachenabstrich
Erreger-Direktnachweis
(Nachweis früher Infektionen)

Test-Besonderheiten:
SARS-CoV-2-Spezifität durch Erfassung von 3 Zielgenen: E-Gen, RdRp-Gen, S-Gen.

Viruslast:
Erreger-Quantifizierung durch Angabe von CT-Werten.

Indirekter Erregernachweis
zur Sicherung einer vorausgegangenen COVID19-Infekti

☐ K341 **SARS-CoV-2 Antikörpernachweis** S
IgM-Antikörper
*(3 -7 Tage nach Symptombeginn, **meldepflichtig**)*

IgG-Antikörper
(14-21 Tage nach Symptombeginn, mögl. Immunität)

Immunität nach Infektion oder Impfung

☐ K342 **SARS-CoV-2 IgG-Antikörpernachweis (quantitativ)** S
IgG-Antikörper gegen SAR[illegible]2 zum Nachweis vorangegangener [illegible]rusinfektion
(nachweisbar ab 14. - 21. Tag nach Symptombeginn).
Sehr hohe Spezifität (99,63%).

***Achtung:** Nicht alle IgG-AK gegen SARS-CoV-2 schützen vor einer erneuten Infektion. Das tun nur neutralisierende Antikörper. Bei positivem SARS-CoV-2 IgG-Antikörpernachweis sollte daher immer im Nachgang geklärt werden, ob es sich dabei wirklich um schützende Antikörper handelt. Biovis bietet beide Tests daher als **Reflextests** an, d.h. nach jedem positiven SARS-CoV-2 IgG-AK-Test wird automatisch geprüft, ob es sich dabei um neutraliserende AK handelt.*

☐ K343 **Neutralisierende (schützende) Antikörper gegen SARS-CoV-2** ***NEU*** S
Quantitativer Nachweis schützender IgG-Antikörper gegen SARS-CoV-2 nach überstandenen Infektionen oder Impfung. Ideal zur Überprüfung eines humoralen Impfschutzes. Erfasst alle bisher bekannten Mutationen.

☐ K346A **Zellulare Immunität bei Nachweis SARS-CoV-2 spezifischer T-Zellen**
Fluoreszenz-EliSpot: Nachweis erregerspezifischer T-Effektor- und T-Gedächtniszellen gegen SARS-CoV-2 und Coronaviridae als Hinweis auf eine vorangegangene COVID-19-Infektion und möglichen Hinweis auf eine bestehende zellulare Immunität. 3CPDA/ACDB EXP

***Hintergrund:** Über Antikörpernachweise gelingt es oft nicht eine zurückliegende SARS-CoV-2 Infektion zu sichern. AK werden nicht gebildet oder AK-Spiegel nehmen wenige Wochen nach Infektion ab. Da auch das zelluläre Immunsystem an der Virusabwehr beteiligt ist, ist ein Nachweis von erregerspezifischen T-Zellen beweisend für vorausgegangene COVID-19-Infektionen und kann für einen zellulären Schutz sprechen.*

Präventive Diagnostik

Orthomolekulare Schutzfaktoren

☐ 0360 Melatonin T923

☐ E530 Vitamin C Hep

☐ E540 25 OH Vitamin D3 S

Ergänzende Faktoren

☐ E345 Zink, Selen, Vitamin A, Vitamin B6, Coenzym Q10 EDTA, Hep, S

☐ E230 Glutathionstoffwechsel EXP CPDA/ACDB
(GSH, GSSG)

Darmschleimhautimmunität, -barriere

☐ A341 Sekretorisches IgA, β-Defensin 2, α-1 Antitrypsin, Calprotectin, Zonulin Fe

☐ A712A Mikrobiom Mini mit Bacteriocin- und Enterocin-produzierenden Bakterien Fe
(z.B. Bifidobakterien, Laktobazillen, Enterokokken)

Test	Ergebnis	Einheit	Normbereich	Vorwert	Probenmaterial Methode
Infektionsdiagnostik					
SARS-CoV-2 IgG-Antikörpernachweis (quantitativ)					
SARS-CoV-2 IgG Ak (quant.)	15432	Au/ml	> 50		S NA) EIA

negativ: < 50 Au/ml
positiv: > 50 Au/ml

Neutralisierende Antikörper gegen SARS-CoV-2					
SARS-CoV-2 TrimericS IgG	>2080,0	BAU/ml	> 33,8		S NA) CLIA

negativ: < 33,8 BAU/ml
positiv: >=33,8 BAU/ml

Test	Ergebnis	Einheit	Normbereich	Vorwert	Probenmaterial Methode
Infektionsdiagnostik					
SARS-CoV-2 IgG-Antikörpernachweis (quantitativ)					
SARS-CoV-2 IgG Ak (quant.)	2138	Au/ml	> 50		S NA) EIA

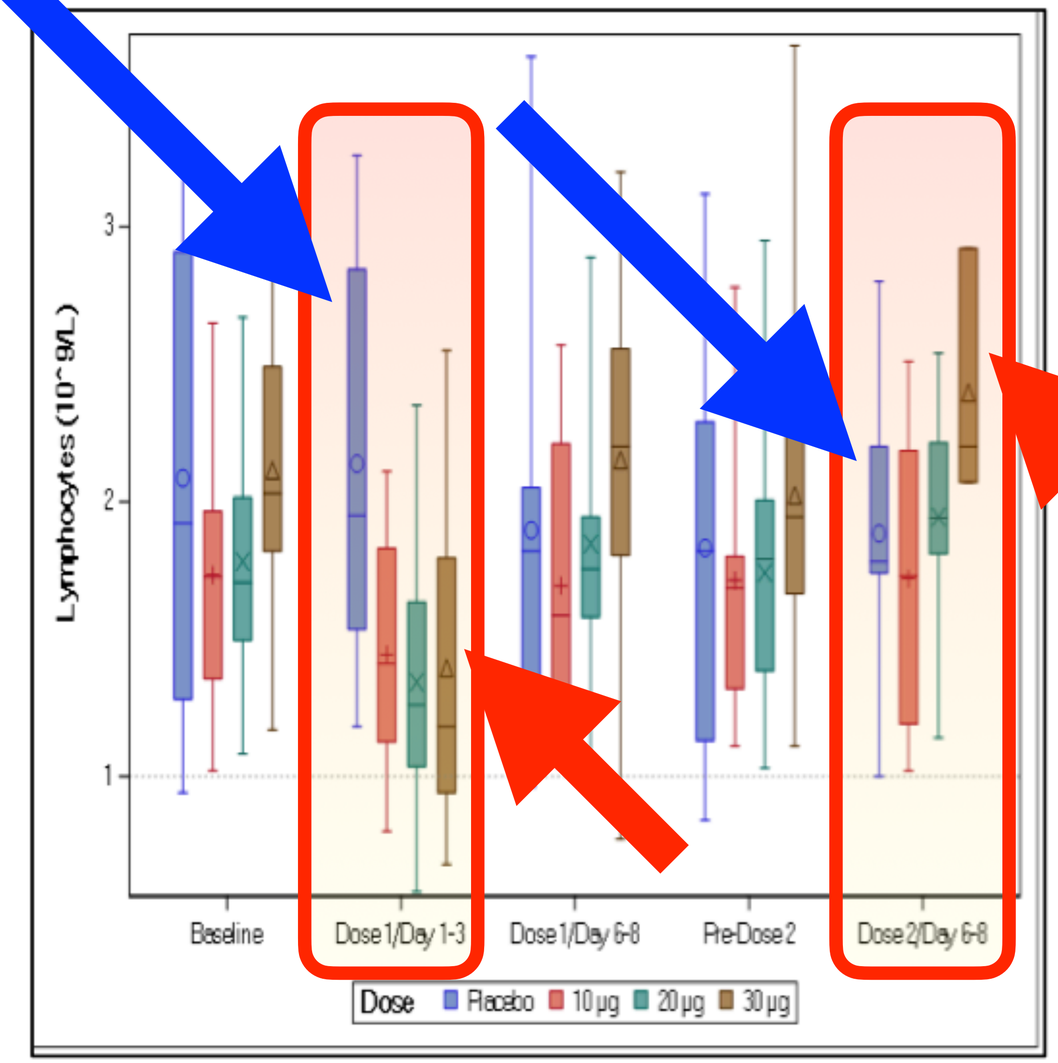
negativ: < 50 Au/ml
positiv: > 50 Au/ml

< 4% (!)

Neutralisierende Antikörper gegen SARS-CoV-2					
SARS-CoV-2 TrimericS IgG	88,6	BAU/ml	> 33,8		S NA) CLIA

negativ: < 33,8 BAU/ml
positiv: >=33,8 BAU/ml
BAU= Binding antibody units

V-AIDS



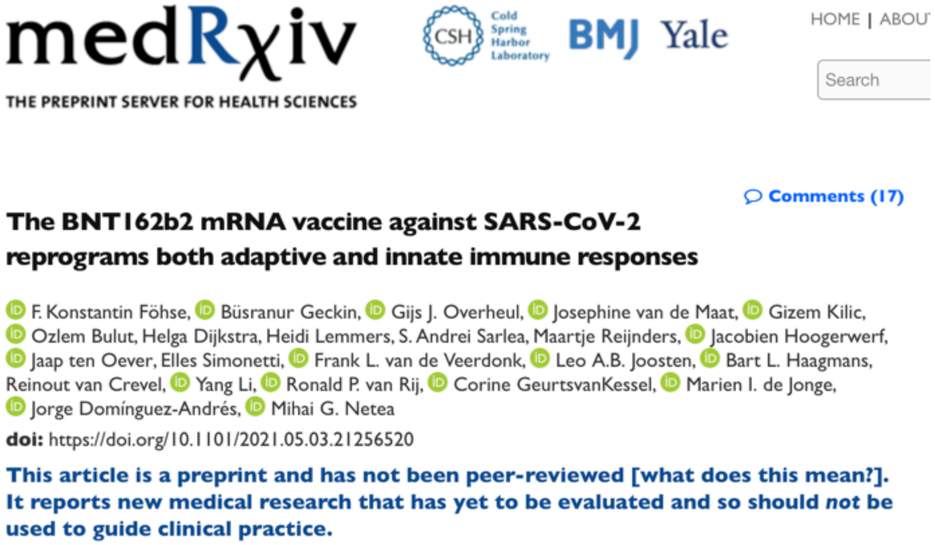
Clinical Trial > N Engl J Med. 2020 Dec 17;383(25):2439-2450.
doi: 10.1056/NEJMoa2027906. Epub 2020 Oct 14.

Safety and Immunogenicity of Two RNA-Based Covid-19 Vaccine Candidates

Edward E Walsh [1], Robert W Frenck Jr [1], Ann R Falsey [1], Nicholas Kitchin [1], Judith Absalon [1], Alejandra Gurtman [1], Stephen Lockhart [1], Kathleen Neuzil [1], Mark J Mulligan [1], Ruth Bailey [1], Kena A Swanson [1], Ping Li [1], Kenneth Koury [1], Warren Kalina [1], David Cooper [1], Camila Fontes-Garfias [1], Pei-Yong Shi [1], Özlem Türeci [1], Kristin R Tompkins [1], Kirsten E Lyke [1], Vanessa Raabe [1], Philip R Dormitzer [1], Kathrin U Jansen [1], Uğur Şahin [1], William C Gruber [1]

Affiliations + expand

PMID: 33053279 PMCID: PMC7583697 DOI: 10.1056/NEJMoa2027906



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Comments (17)

The BNT162b2 mRNA vaccine against SARS-CoV-2 reprograms both adaptive and innate immune responses

F. Konstantin Föhse, Büsranur Geckin, Gijs J. Overheul, Josephine van de Maat, Gizem Kilic, Ozlem Bulut, Helga Dijkstra, Heidi Lemmers, S. Andrei Sarlea, Maartje Reijnders, Jacobien Hoogerwerf, Jaap ten Oever, Elles Simonetti, Frank L. van de Veerdonk, Leo A.B. Joosten, Bart L. Haagmans, Reinout van Crevel, Yang Li, Ronald P. van Rij, Corine GeurtsvanKessel, Marien I. de Jonge, Jorge Domínguez-Andrés, Mihai G. Netea

doi: https://doi.org/10.1101/2021.05.03.21256520

This article is a preprint and has not been peer-reviewed [what does this mean?]. It reports new medical research that has yet to be evaluated and so should *not* be used to guide clinical practice.

- Reduzierte Immunantwort auf Bakterien und Viren
- Abnahme des TLR-Signalwegs
- Abnahme der Makrophagen-Aktivität
- Rückgang der Sekretion von TNF-a, IFN-y, IL-1, IL-6

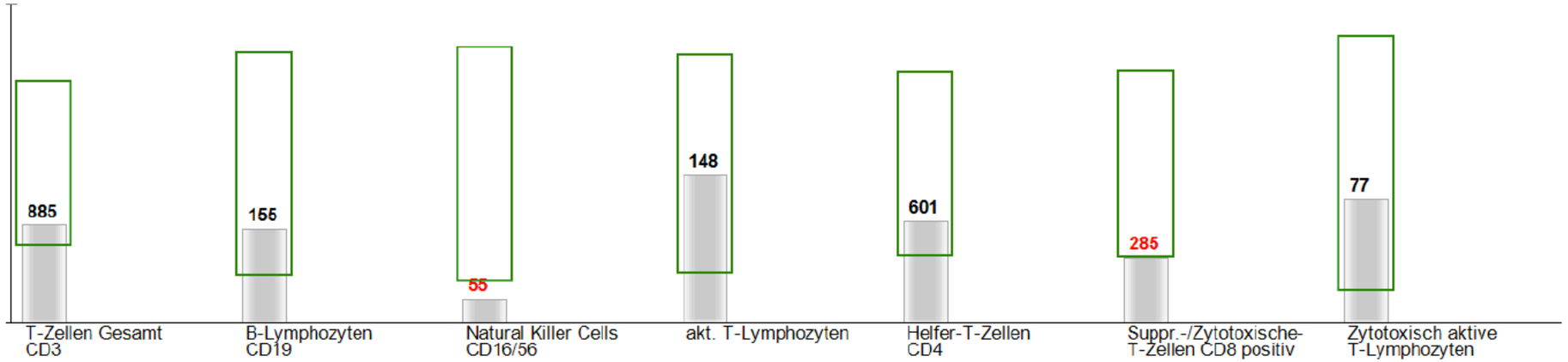
Th1/2/17

Test	Ergebnis	Einheit	Normbereich	Vorwert	Probenmaterial Methode
Immunologie und Hämatologie					
TH1/2/17 Zytokinstatus					
TH1-Zytokine (T-Helfer-, zytotox. T-Zellen)					
Interferon-gamma	19	pg/ml	500 - 3000		H NA) FLOWZY
Interleukin-2	20	pg/ml	30 - 250		H NA) FLOWZY
TNF-alpha	81	pg/ml	135 - 2100		H NA) FLOWZY
TH2-Zytokine (T-Helfer-, B-Zellen)					
Interleukin-4	4,7	pg/ml	22 - 40		H NA) FLOWZY
Interleukin-6	213	pg/ml	4000 - 8500		H NA) FLOWZY
Interferon-gamma/IL4-Ratio	3,98	Quotient	30 - 60		H NA) FLOWZY
TH2-regulatorisch (antiinflammatorisch)					
Interleukin-10	19	pg/ml	175 - 4775		H NA) FLOWZY
TH17 (Granulozyten, chronisch)					
Interleukin-17	3,94	pg/ml	0 - 25		H NA) FLOWZY

Vorläufiger Referenzbereich!

CD-profiles

Übersicht



Lymphozytendifferenzierung

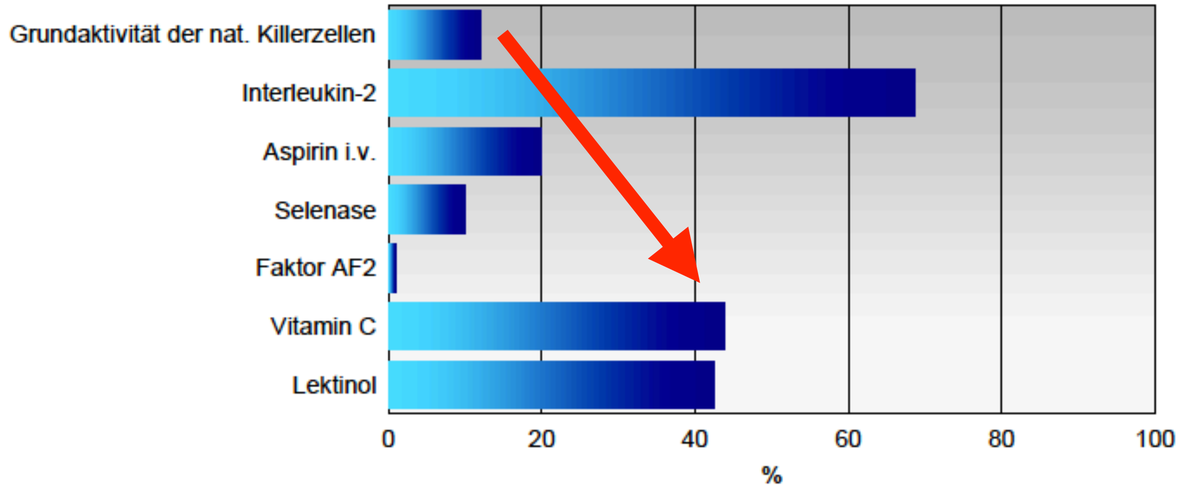
NK-activity

Immunologie und Hämatologie

Tumor killing test Standardpanel

Standard-Panel NK-Funktion

Grundaktivität der nat. Killerzellen	12,0	%	15 - 25
Interleukin-2	68,6	%	> 25
Aspirin i.v.	20,0	%	
Selenase	10,0	%	
Faktor AF2	1,0	%	
Vitamin C	43,7	%	
Lektinol	42,5	%	



NK-Modulation Biovis

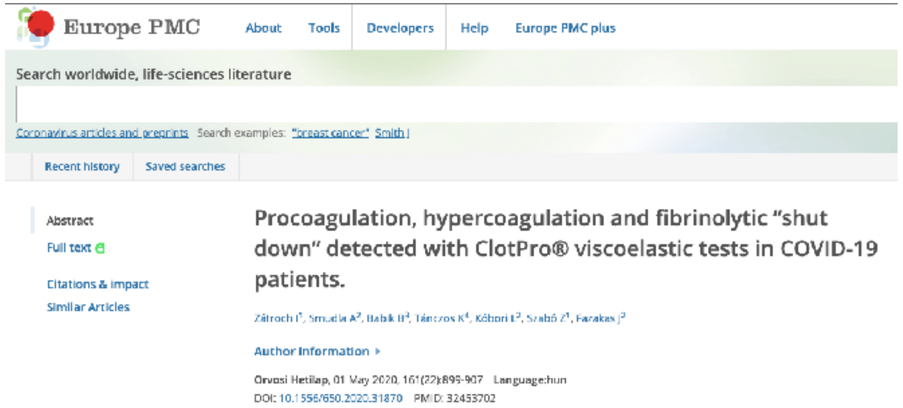
Autoimmunity

Autoimmunity: Variants

Klassische Auto-AK

Autoreaktive S-AK

GPCR-AK



- International data indicate that arterial, venous and microvascular thrombosis or disseminated intravascular coagulation occur in more than 30% of hospitalized patients with COVID-19.

- This condition is characterized by high levels of D-dimer and fibrinogen, prolonged prothrombin time and activated partial thromboplastin time.

Springer Link

Published: 09 July 2020

Profile of natural anticoagulant, coagulant factor and anti-phospholipid antibody in critically ill COVID-19 patients

Yan Zhang, Wei Cao, Wei Jiang, Meng Xiao, Yongzhe Li, Ning Tang, Zhengyin Liu, Xiaowei Yan, Yongqiang Zhao, Taisheng Li & Tienan Zhu

Journal of Thrombosis and Thrombolysis **50**, 580–586(2020) | Cite this article

2491 Accesses | **28** Citations | **2** Altmetric | Metrics

Journal of Critical Care

Volume 59, October 2020, Pages 32-34

Clinically significant anticardiolipin antibodies associated with COVID-19

Sami Hossri [a], Mahmoud Shadi [b], Zaid Hamarsha [b], Rick Schneider [b], Dany El-Sayegh [a]

Show more

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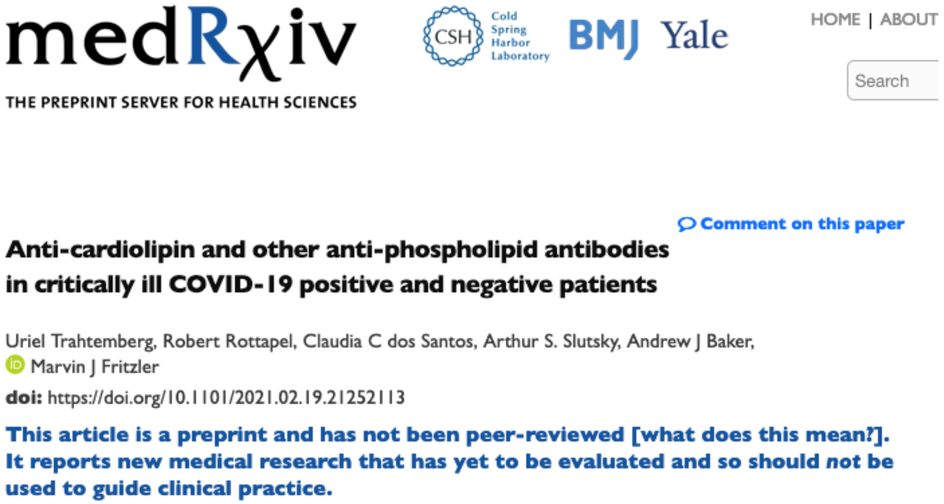
https://doi.org/10.1016/j.jcrc.2020.05.017

Get rights and content

- 40-55% of critically ill patients are (+) on phospholipid auto-antibodies



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Anti-cardiolipin and other anti-phospholipid antibodies in critically ill COVID-19 positive and negative patients

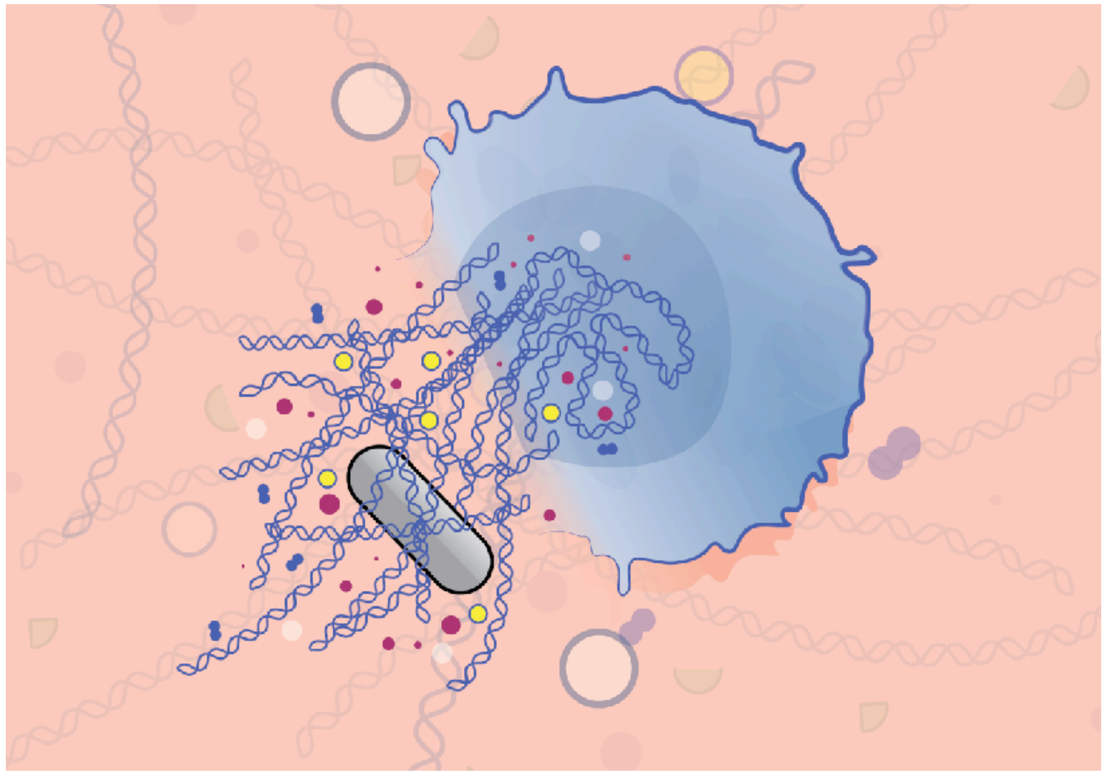
Uriel Trahtemberg, Robert Rottapel, Claudia C dos Santos, Arthur S. Slutsky, Andrew J Baker, Marvin J Fritzler

doi: https://doi.org/10.1101/2021.02.19.21252113

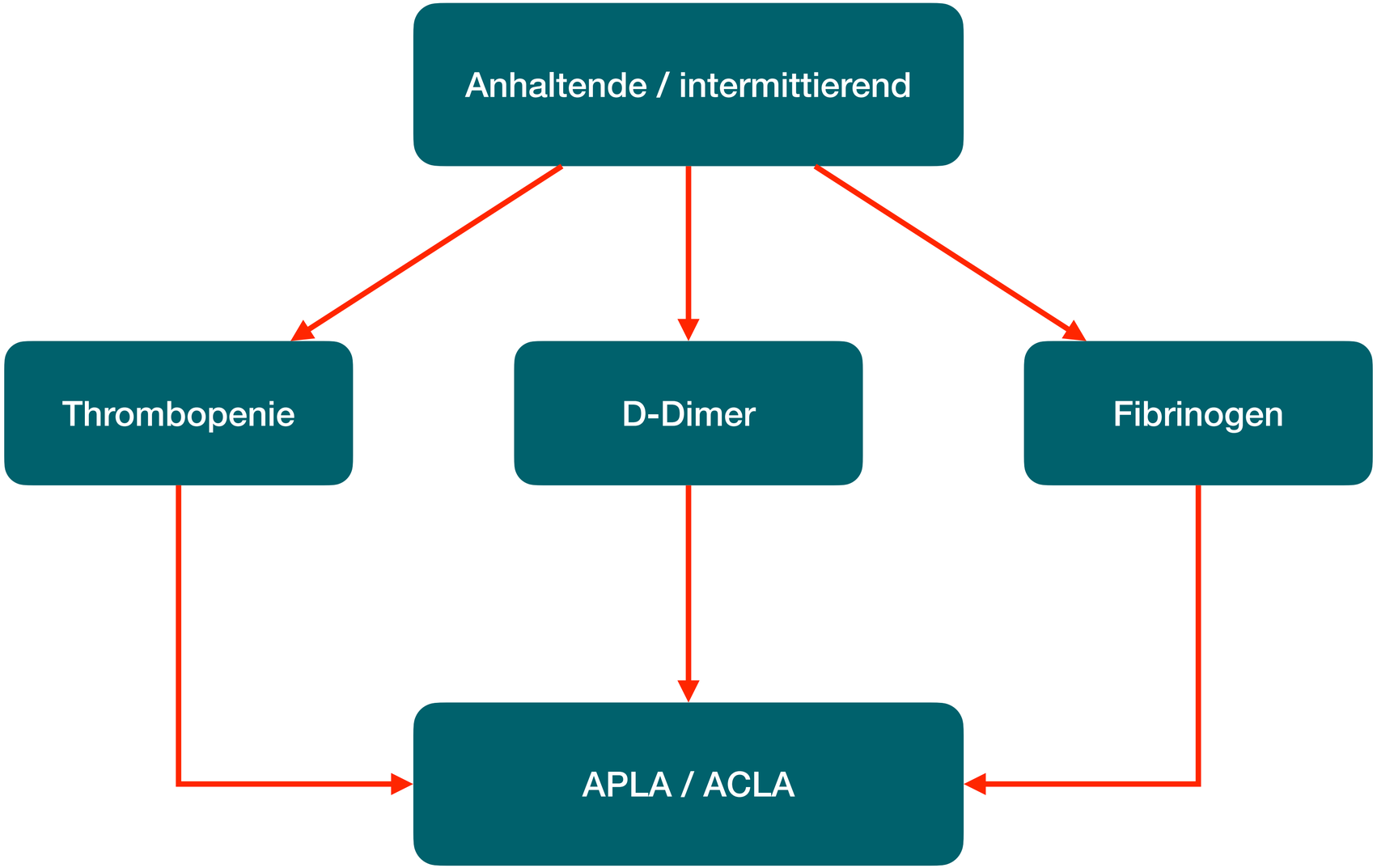
This article is a preprint and has not been peer-reviewed [what does this mean?]. It reports new medical research that has yet to be evaluated and so should *not* be used to guide clinical practice.

- APLA were predominantly IgG aCL (48%) … with a tendency toward higher frequency among the COVID+
- aCL was not associated with surrogate markers of thrombosis but IgG aCL was strongly associated with worse disease severity and higher ANA titers

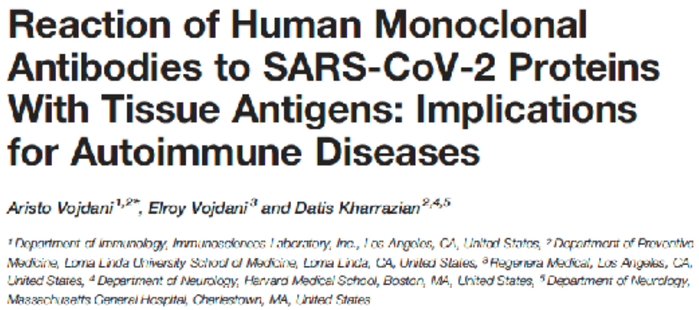
- Association of APLA with increased Calprotectin in serum
- Marker for activation of neutrophils
- Also: Myeloperoxidase-DNA-complexes, pointing at „neutrophil extracellular traps“ (NET) hinweisen.



APLA



S1-AK are auto reactive by nature



Reaction of Human Monoclonal Antibodies to SARS-CoV-2 Proteins With Tissue Antigens: Implications for Autoimmune Diseases

Aristo Vojdani[1,2], Elroy Vojdani[3] and Datis Kharrazian[2,4,5]*

[1] Department of Immunology, Immunosciences Laboratory, Inc., Los Angeles, CA, United States, [2] Department of Preventive Medicine, Loma Linda University School of Medicine, Loma Linda, CA, United States, [3] Regenera Medical, Los Angeles, CA, United States, [4] Department of Neurology, Harvard Medical School, Boston, MA, United States, [5] Department of Neurology, Massachusetts General Hospital, Charlestown, MA, United States

- SARS-CoV-2 antibodies had reactions with 28 out of 55 tissue antigens, that included
- barrier proteins, gastrointestinal, thyroid and neural tissues, and more
- similarities and homology between spike, nucleoprotein … with the human tissue antigens mitochondria M2, F-actin and TPO

S1-AK are auto reactive by nature

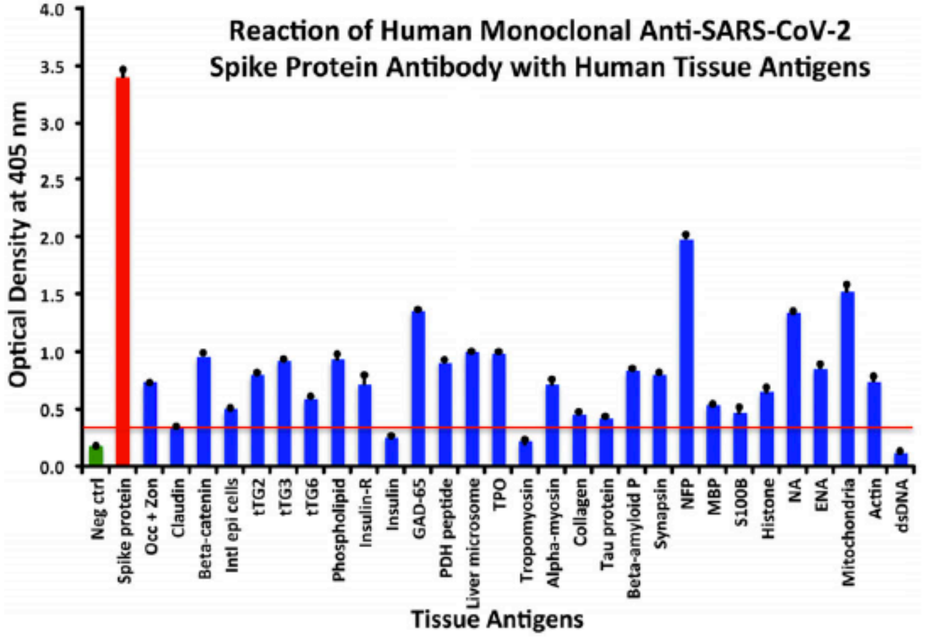


FIGURE 1 | Reaction of anti-SARS-CoV-2 spike protein human monoclonal antibody with human tissue antigens. Each bar represents the calculated

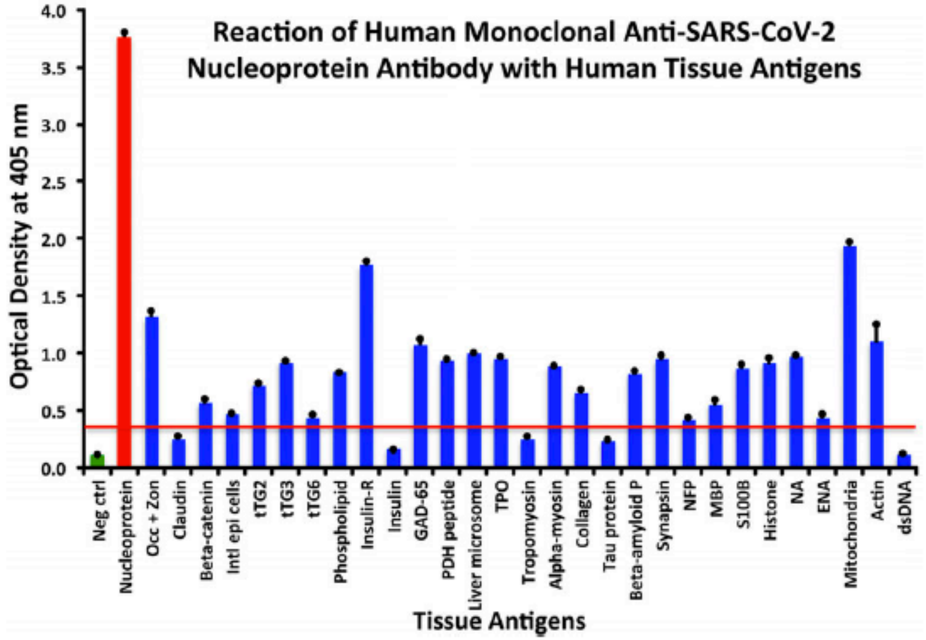


FIGURE 2 | Reaction of anti-SARS-CoV-2 nucleoprotein human monoclonal antibody with human tissue antigens. Each bar represents the calculated

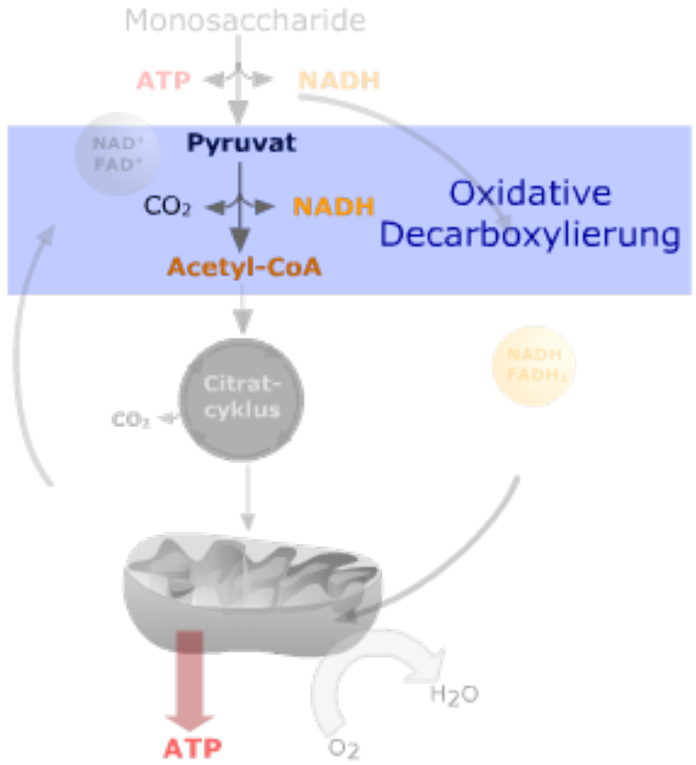
Rote Linie = Cut-off für p > 0,1

S1-AK are auto reactive by nature

Antigens	Spike protein OD	% reactivity
SARS-CoV-2	3.40	100 ++++
Actin	0.74	17.6 +
Mitochondrial antigen (M2)	1.52	41.8 +++
ENA	0.85	21.0 +
NA	1.34	36.2 ++
Histone	0.65	14.8 +
S100B	0.46	9.0 +
MBP	0.53	11.1 +
NFP	1.98	56 ++++
Synapsin	0.81	19.8 +
Beta-amyloid P	0.83	20.4 +
Tau protein	0.41	7.4 +
Collagen	0.45	8.6 +
Alpha-myosin	0.72	17.0 +
Tropomyosin	0.21	1.2 –
TPO	0.98	25.1 ++
Liver microsome	1.00	25.7 ++
PDH peptide	0.91	22.9 +
GAD-65	1.35	36.5 ++
Insulin	0.25	2.5 –
Insulin-R	0.72	17.0 +
Phospholipid	0.93	23.5 +
tTG-6	0.58	12.7 +
tTG-3	0.42	23.2 +
tTG-2	0.79	19.2 +
Int epi cells	0.49	9.9 +
Beta-catenin	0.95	24.1 +
Claudin	0.33	4.9 –
Occ + zon	0.72	17.0 +
27 other tissues*	0.34	5.2 –

AMA-M2

- Zielen auf innere und äußere Mito-Membran, u.a.
- PDH >> Entkopplung
- Citratzyklus



Leistung/Material/Versandart	Parameter			Preis in €
Intrazelluläres ATP [1)] **Material:** 1 x CPDA-Blut (8,5 ml) **Versand:** **Über Nacht ohne Kühlung**	Quantitative Bestimmung der intrazellulären ATP-Konzentration mit Angabe der möglichen mitochondrialen ATP-Bildung im Grundzustand der Immunzellen.	**PBMC**		**70,00**
ATP-Leistungstest [1)] **Material:** 1 x CPDA-Blut (8,5 ml) **Optional:** Serum (min. 1 ml) **Versand:** **Über Nacht ohne Kühlung**	Ermittlung a) der intrazellulären ATP-Konzentration, b) der mitochondrialen und glykolytischen Kapazität zur ATP-Bildung, c) von Stoffwechselverschiebungen (Crabtree-, Pasteur-, Warburgeffekt) im Grundzustand der Immunzellen. *Zusätzlich:*	**PBMC** **Thrombozyten**	☐ ☐	**139,00** **139,00**
	1. Testung auf individuell gewünschte Substanzen: Anzahl der Substanzen: ☐ Art der Substanzen: _____ **2. Testung auf Seren:** Anzahl der Seren: ☐		☐ ☐	**60,00 je Substanz/Serum/Zelltyp**
Metabolischer Phänotyp **Material:** 1 x CPDA-Blut (8,5 ml) [2)] **Versand:** **Über Nacht ohne Kühlung**	Quantitative Bestimmung des intrazellulären ATP-Gehalts mit Unterscheidung des mitochondrial und nicht-mitochondrial gebildeten ATP in den Immunzellen im Grundzustand und bei Energieanforderung (metabolisches Potenzial).	**PBMC** Thrombozyten CD8-T-Zellen Monozyten	☐ ☐ ☐ ☐	**169,00** **169,00** **219,00** **219,00**
Bioenergetischer Gesundheitsindex [1,2)] **Material:** 1 x CPDA-Blut (8,5 ml) [3)] **Versand:** **Über Nacht ohne Kühlung**	Bestimmung des mitochondrialen und nicht-mitochondrialen Sauerstoffverbrauchs, der mitochondrialen ATP-Generierung, der nicht-mitochondrialen Energiegewinnung, der mitochondrialen Reserveatmungskapazität, des Protonenlecks, des Elektronenlecks, des metabolischen Phänotyps (siehe Anforderung Metabolischer Phänotyp). *Zusätzlich:*	**PBMC** Thrombozyten CD8-T-Zellen Monozyten	☐ ☐ ☐ ☐	**199,00** **199,00** **249,00** **249,00**
	1. Testung auf individuell gewünschte Substanzen: Anzahl der Substanzen: ☐ Art der Substanzen: _____ **2. Testung auf Seren:** Anzahl der Seren: ☐		☐	**60,00 je Substanz/Serum/Zelltyp**

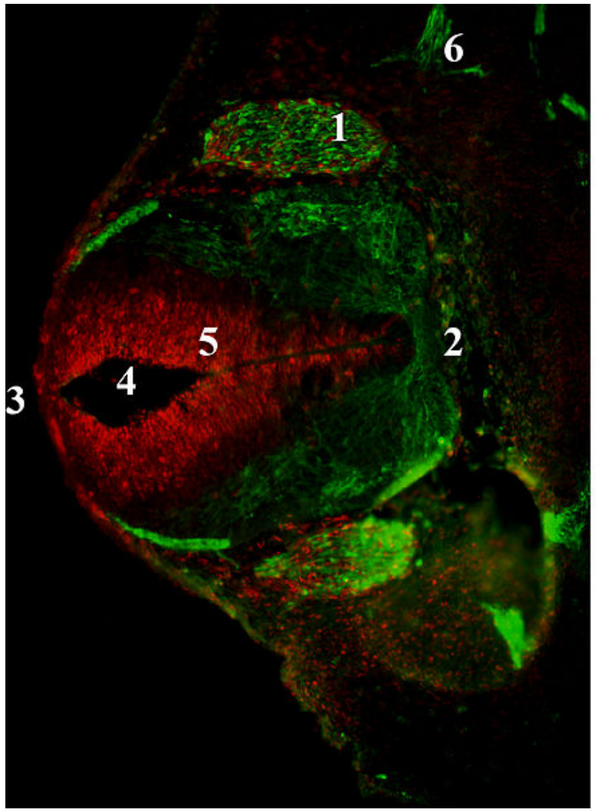
MMD

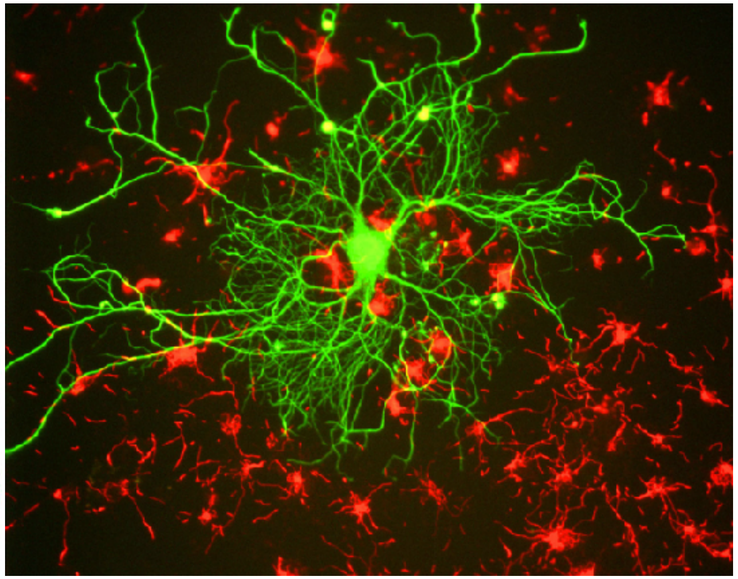
S1-AK and Mitochondria

	ohne Persönliche Wert	Serum Persönliche Wert	Zielwert (optimal)
Bioenergetischer Gesundheitsindex (BHI)	1,17	0,00	>2,5
Mitochondriale Bioenergetik			
Kopplungseffizienz in %	86,91	76,75	100
Reserveatmungskapazität in %	114,68	0,00	>400
Zelluläres Sauerstoffverbrauchsprofil			
Anteil nicht-mitochondriale Atmung zur Gesamtatmung in %	31,27	31,64	<10
Anteil des Protonenlecks zur Gesamtatmung in %	9,12	15,25	
Anteil Atmung für mitochondriale ATP-Gewinnung in %	59,61	53,11	>90
ATP-Umsatzrate (mitochondriale Sauerstoffverwertung)			
ATP-Grundumsatz in %	41,23	100,00	<20
ATP-Reserve in %	58,77	0,00	>80
Mögliche maximale Sauerstoffverbrauchsrate in pmol Sauerstoff/min	64,25	20,85	>300
Zellulärer Energie-Phänotyp			
In Ruhe	Ruhend/glykolytisch	Ruhend/glykolytisch	ruhend
Bei Energieanforderung	glykolytisch	glykolytisch	Energetisch/aerob
Metabolisches Potenzial in % -Mitochondrien-	175,85	76,50	>350
Metabolisches Potenzial in % -Glykolyse-	254,90	179,55	>350
Sauerstoffverbrauch/Glykolyse bei Energieanforderung	Starke Bevorzugung der anaeroben Glykolyse	Sehr starke Bevorzugung der anaeroben Glykolyse	

NFP

- Neurofilament-Proteine
- Zytoskelett, axonaler Transport





Further S1-AK autoreactions

- NA: Nuclear AB (vgl. ANA)
- TPO >> Autoimmun-Thyreoditis
- Leber Mikrosomen: ER-affine Vesikel mit CYP-Komplexen
- GAD-65: Glutamat-Dehydrogenase >> Glutamat-Abbau zu Glutamin
- Zusätzlich: Transglutaminase-AK (>> induzierte Zöliakie)

ELSEVIER

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Functional autoantibodies against G-protein coupled receptors in patients with persistent Long-COVID-19 symptoms

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- Auto-AK gegen G-Protein-gekoppelte Rezeptoren
- ß2-Adreno-Rp, a1-Adreno-Rp, AT1-Rp, Muscarinische Rp, etc. etc.
- 31/31 waren positiv auf diese AK, Rate betrug 2-7/patient

GPCR-AK: What to expect

Krankheit	Rezeptoren
Dilated cardiomyopathy	β1-adrenergic muscarinic M2
Peripartum cardiomyopathy	β1-adrenergic muscarinic M2
Chagas' cardiomyopathy	β1-adrenergic muscarinic M2 β2-adrenergic
Myocarditis	β1-adrenergic
Electric cardiac abnormalities	β1-adrenergic muscarinic M2 β2-adrenergic serotoninergic 5HT4
Refractory hypertension	α1-adrenergic
Idiopathic pulmonary hypertension	α1-adrenergic endothelin 1 ETA
Malignant hypertension	angiotensin II AT1
Preeclampsia	angiotensin II AT1 endothelin 1 ETA
Orthostatic hypotension	β2-adrenergic muscarinic M3
Postural orthostatic tachycardia syndrome (POTS)	β2-adrenergic muscarinic M2
Diabetes mellitus type II	α1-adrenergic
Vascular renal rejection	angiotensin II AT1
Scleroderma	angiotensin II AT1 endothelin 1 ETA
Thromboangitis obliterans	α1-adrenergic endothelin 1 ETA angiotensin II AT1
Systemic lupus erythematosus (SLE)	serotoninergic 5HT4*
Allergic asthma	β2-adrenergic*
Open angle glaucoma	β2-adrenergic
Vascular dementia / Alzheimer's dementia	α1-adrenergic β2-adrenergic endothelin 1 ETA angiotensin II AT1
Benign prostate hyperplasia	endothelin 1 ETA
Complex regional pain syndrome (CRPS)	muscarinic M2 β2-adrenergic
Sjögren's syndrome	muscarinic M3
Fatigue syndrome	β2-adrenergic muscarinic M2 muscarinic M3 muscarinic M4

GPCR-AK: What to expect

Neutralization of Autoantibodies Targeting G-Protein Coupled Receptors Improves Capillary Impairment and Fatigue Symptoms after COVID-19 Infection

9 Pages • Posted: 23 Jul 2021

- Long-Covid syndrome (LCS) … is associated with a variety of characteristics as e.g. impaired capillary microcirculation, chronic fatigue syndrome (CFS) and functional autoantibodies targeting G-protein coupled receptors (GPCR-AAb)
- report of a successful healing of LCS with BC 007 (Berlin Cures, Berlin, Germany), a DNA aptamer drug with high affinity to GPCR-AAbs that neutralizes these AAbs.
- A patient … was positively tested for GPCR-AAbs
- Within 48 h after a single BC 007 treatment, GPCR-AAbs were functionally inactivated and remained inactive during the observation period of 4 weeks.
- This observation was accompanied by a constant improvement of the patient's fatigue symptoms
- Therefore, we propose that removal of GPCR-AAb ameliorates characteristics of the Long-Covid-Syndrome such as capillary impairment, loss of taste and CFS.

GPCR-AK: What to expect

IMD Labor Berlin

Ärztlicher Befundbericht

Untersuchung	Ergebnis	Einheit	Referenz-bereich
ß1-adrenerge Rez.-AAk i.S. (ELISA)	8.0	U/ml	< 15.0
ß2-adrenerge Rez.-AAk i.S. (ELISA)	**48.6**	U/ml	< 8.0
M3-mAChR-AAk i.S. (ELISA)	**50.1**	U/ml	< 6.0
M4-mAChR-AAk i.S. (ELISA)	**> 40.0**	U/ml	< 10.7

Nachweis von Autoantikörpern (AAk) gegen ß2-adrenerge Rezeptoren und M3-/M4-muskarinerge Acetylcholinrezeptoren.

+

- ✓ ANA, ANCA
- ✓ M2-AK
- ✓ APLA / ACLA
- ✓ LKM1 (Lebermikrosomen)
- ✓ TPO-AK
- ✓ GAD-AK
- ✓ Transglutaminase-AK

Therapeutic options

Softskills: Immune-modulation

IMD Labor Berlin

Ärztlicher Befundbericht

Untersuchung	Ergebnis	Einheit	Referenz-bereich
IFN-γ (TH1)	234	pg/ml	374 - 1660
IL-4 (TH2)	544	pg/ml	28 - 141
TH1 / TH2 Ratio	0,43		6,1 - 21

Die stimulierte Zytokinfreisetzung der T-Lymphozyten zeigt einen expandierten TH2-Zell-Anteil (erhöhtes IL-4) bei gleichzeitig reduzierter TH1-Antwort (erniedrigtes IFN-γ). Die verminderte TH1 / TH2-Ratio spricht für einen TH2-Shift.

IMD Labor Berlin

Ärztlicher Befundbericht

Untersuchung	Ergebnis	Einheit
Interleukin 4 Hemmtest (PIA)		
IL4 Response	533	pg/ml
IL4 Präparat 1 Curcumin	411	pg/ml
IL4 Präparat 2 Boswellia serrata	72,2	pg/ml
IL4 Präparat 3 Resveratrol	655	pg/ml

IMD Labor Berlin

Fachbereiche | Spezielle Kompetenzen | Fortbildungen | Fachinforma

Home > Leistungsverzeichnis > Parameter

Labor Parameterverzeichnis

Interleukin- 6-Hemmtest

Primärmaterial	Heparin-Blut
Materialmenge	10 ml
Hinweise	Bitte zu testende Präparate angeben bzw. miteinsenden, keine EBM-Leistung
Methode	Vollblutstimulation
Durchführungsort	Berlin
Testdurchführung	Mo-Sa

Methode mit * = Fremdversand

IMD Labor Berlin

Ärztlicher Befundbericht

Untersuchung	Ergebnis	Einheit
TNF-alpha-Hemmtest		
Der TNF-α-Basiswert ist die Bezugsgröße, mit der die TNF-α-Werte der einzelnen Präparate verglichen werden. Werte, die niedriger als der Basalwert sind, zeigen eine antientzündliche Wirkung an. Höhere Werte sprechen für einen proentzündlichen in vitro-Effekt des jeweiligen Präparates.		
TNF-α-Basiswert (LPS stimuliert)	446	pg/ml
TNF-α Präparat 1	11,1	pg/ml
Präparat 1 Hemmung (1) Curcumin	97,5	%
TNF-α Präparat 2	387	pg/ml
Präparat 2 Hemmung (2) Boswellia serrata	13,2	%
TNF-α Präparat 3	475	pg/ml
Präparat 3 Hemmung (3) Procain	0,0	%

Vor allem Curcumin zeigt bei diesem Probanden einen deutlichen TNF-hemmenden und somit antientzündlichen Effekt. Procain hingegen hat hier keinen TNF-α hemmenden Effekt.

IMD Labor Berlin

Ärztlicher Befundbericht

Untersuchung	Ergebnis	Einheit
Effektorzelltypisierung		
IFN-γ-Basal	< 3,2	pg/ml
IL10-Basal	< 5,0	pg/ml
IL2-Basal	< 3,2	pg/ml
TNF-α-Basal	< 20,0	pg/ml
IFN-γ-Positivkontrolle	20,4	pg/ml
IL10-Positivkontrolle	155	pg/ml
IL2-Positivkontrolle	67,4	pg/ml
TNF-α-Positivkontrolle	165	pg/ml
IFN-γ-Antigen 1	7,8	pg/ml
IL10-Antigen 1	87,4	pg/ml
IL2-Antigen 1	1,8	pg/ml
TNF-α-Antigen 1 (1) Gold	62,7	pg/ml
TNF-γ-Antigen 2	0,6	pg/ml
IL10-Antigen 2	0,4	pg/ml
IL2-Antigen 2	1,1	pg/ml
TNF-α-Antigen 2 (2) Kupfer	6,7	pg/ml

Interpretation

Der Befund zeigt eine TH1-dominante Sensibilisierung auf Gold. Kupferspezifische Effektor-T-Zellen sind dagegen nicht im Blut nachweisbar, was allerdings eine latente Typ IV-Sensibilisierung nicht ausschließt.

Where it’s going

Glucokortikoide

- ➡ Einfach
- ➡ Günstig
- ➡ Schonender als Suppression

- ➡ Langfristige AE

Immunadsorption / Plasmapherese

- ➡ Sehr schnell
- ➡ Schonend
- ➡ Breite Wirkungspalette
- ➡ Wenig KI

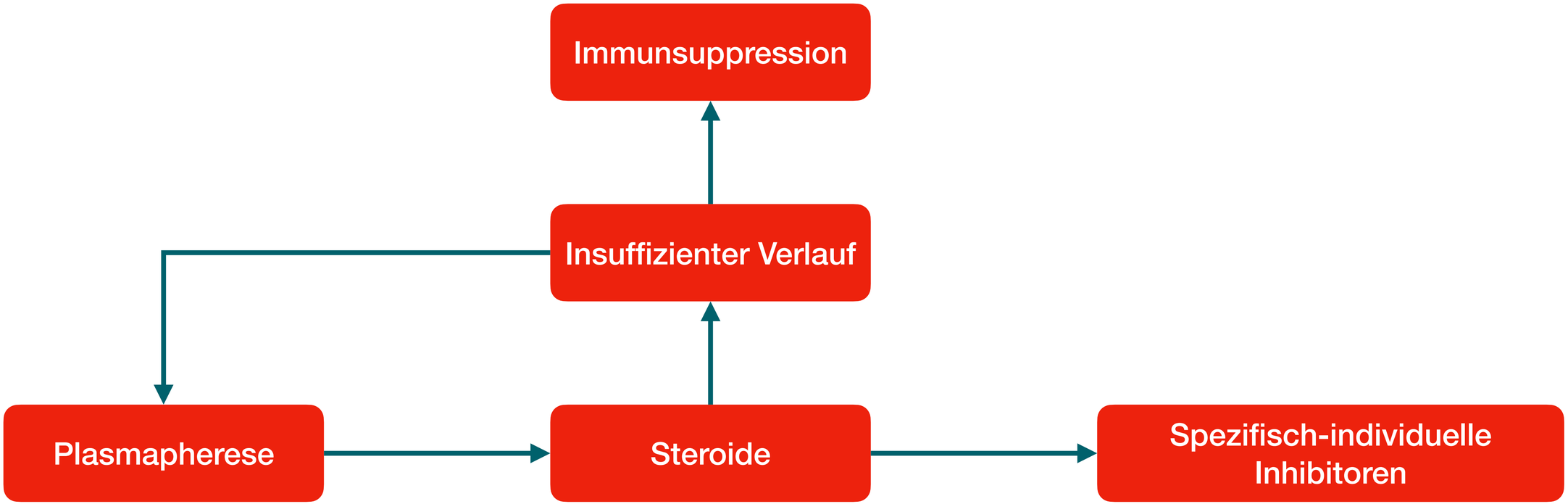
- ➡ Kosten
- ➡ Verfügbarkeit
- ➡ Logistik

Immunsuppressiva

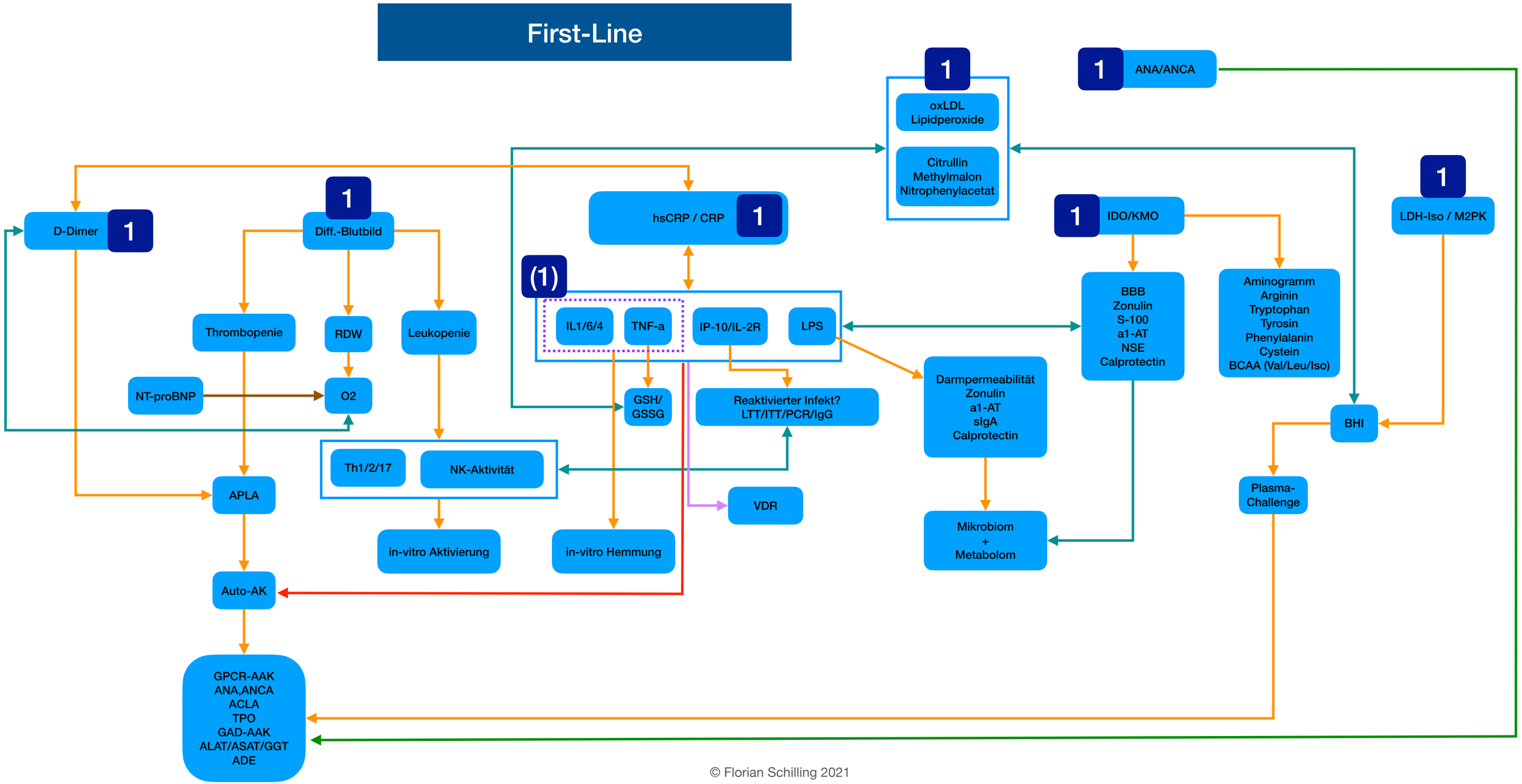
- ➡ Billiger und einfacher als P-Pherese
- ➡ Potentiell auch zelluläre Reaktionen

- ➡ Schwere AE
- ➡ Komplikationsrisiken
- ➡ Limitiert durch zahlreiche KI

Flow-chart immune therapy



First-Line



When things get tough, the tough get going

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