Living with COVID could be harder than we think: Understanding SARS COV-2 infections in Individuals and Populations

Professor Jeremy Nicholson, PhD, DSc, FRCPath, HonFRCP, FMedSci
Director, ANPC
Pro-Vice Chancellor, Health Sciences
Murdoch University

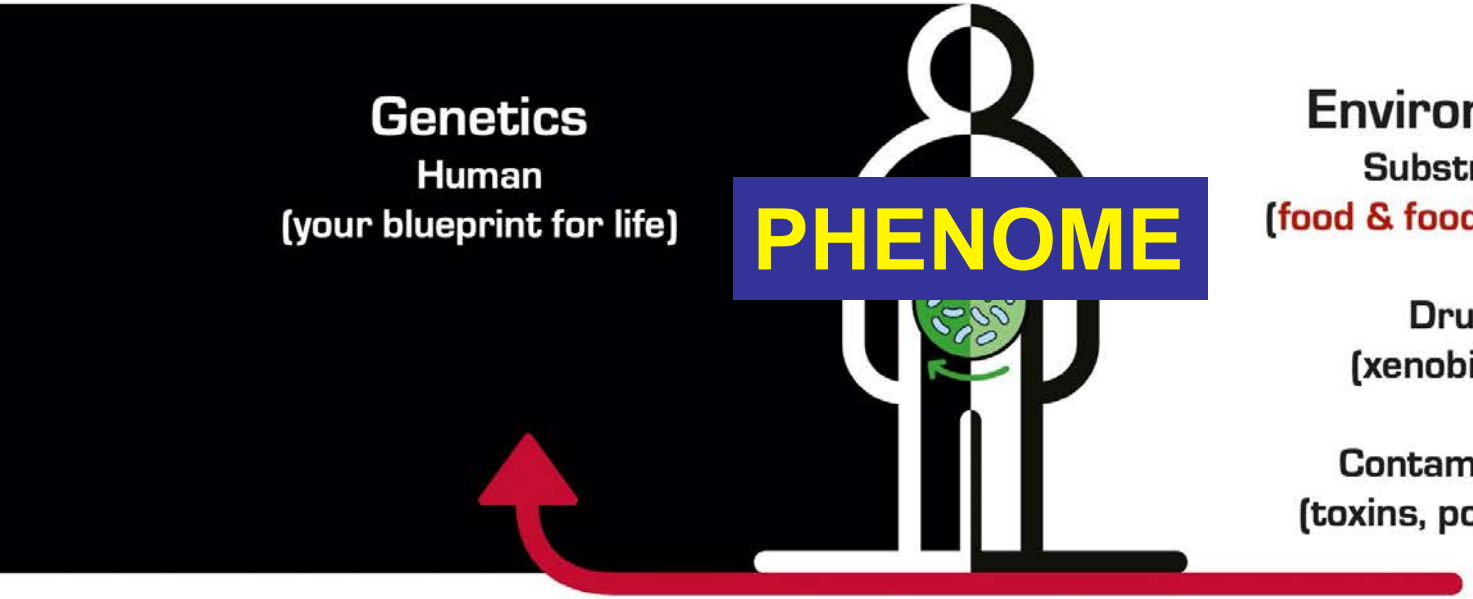
Imperial College London

"We acknowledge that we are working on the Whadjuk Noongar Boodjar and pay respect to all Noongar people and elders past, present and emerging"

Computational Medicine, Informatics and Data Visualization

One of the World's best equipped research laboratories for understanding the origins and consequences of disease

WE ARE THE PRODUCT OF OUR GENE-ENVIRONMENT INTERACTIONS



Our phenome is a description of who we are physically and chemically - our phenomes are determined by the interactions between our genes and environment (including diet and microbes)

Our Phenome links *directly* to your state of health and disease risks.... we have ways of measuring our metabolic phenotype quickly and objectively by analysing body fluids

The International Phenome Centre Network (2021)

IPCN AND GLOBAL UNMET MEDICAL NEEDS

COVID-19

Pandemic Disease Threats (Zoonoses)

Diabetes

Obesity and Malnutrition

Healthy Diet and Preventive Medicine

Cancer Stratification and Treatment Optimization

Anti-microbial Resistance

Healthy Aging and Dementias

Stroke and Cardiovascular Diseases

Global Warming-Health Interactions

Metabolic technology, protocol and method harmonization to facilitate database sharing and International Big Data mining and to address the Global Challenges of Unmet Medical Need....

yahoo!news | Yahoo News Australia

Victoria's daily Covid-19 cases soar again, nine more deaths

Brianne Tolj

Thu., 4 November 2021, 6:16 am

COVID-19 Facts

- A Systemic disease caused by the SARS CoV-2 β–coronavirus (very like SARS1)
  Dangerous new variants, infectivity, incubation times, testing and vaccine issues?

- Unmitigated, COVID-19 is ca. up to 60 X more deadly than Flu – and can be passed on asymptomatically by aerosol transmission….

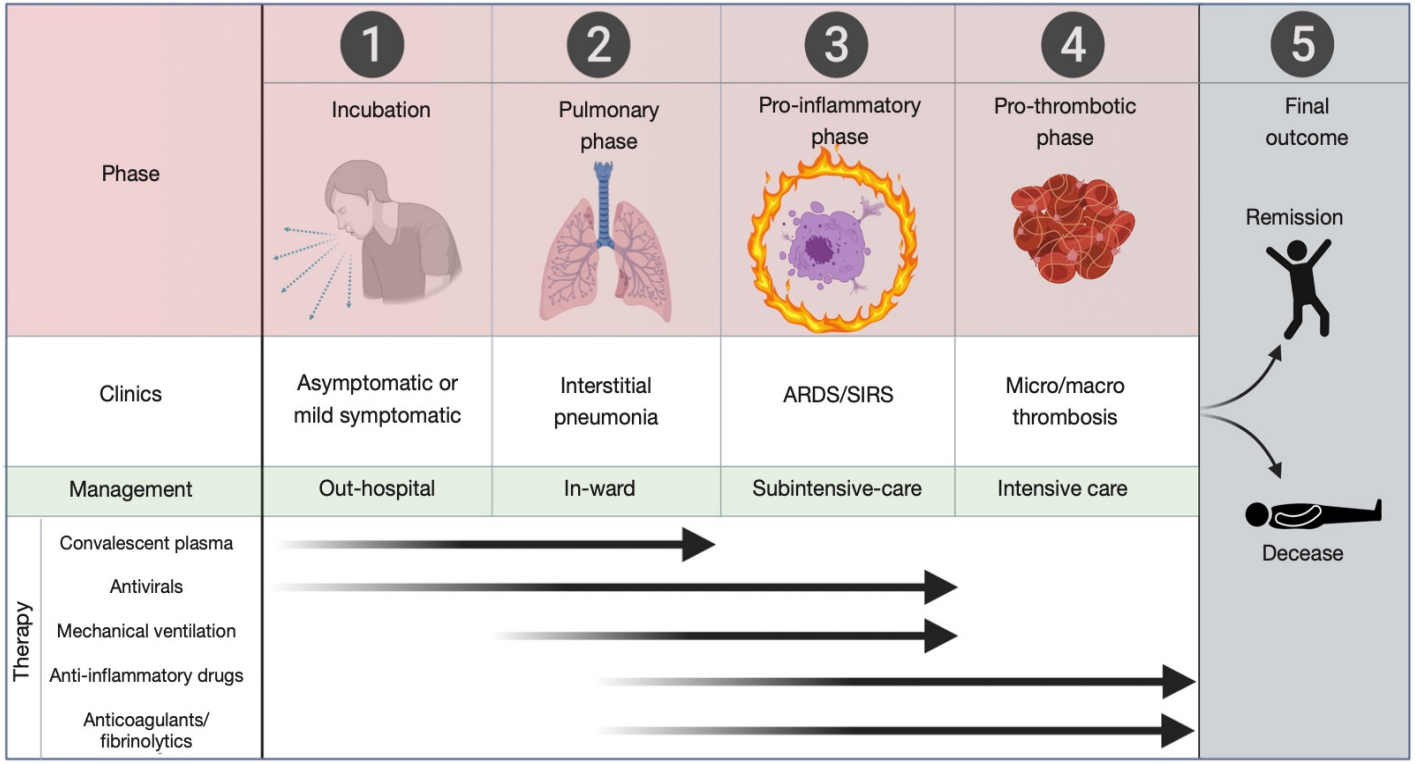
- Incubation period variable 2-12 days for Wuhan variant (newer variants are faster) Time from first symptoms to severe disease takes 8-10 days…..Poorly understood for new variants….Delta (especially Delta plus) is faster and more infectious…

- Much more than just a lung disease - complex multi-system organ failure- affects children and adults, but lung severity increases with age and background 'conditions'…. COVID makes everything you have got worse!

- Complex, incomplete and partial recovery is common- Long COVID and Post-Acute COVID-19 Syndrome (PACS). Long COVID can affect anyone and is not simply related to severity.

COVID-19: unravelling the clinical progression of nature's virtually perfect biological weapon

Ann Transl Med 2020;8(11):693 | http://dx.doi.org/10.21037/atm-20-3989



Complications and Variations

Acute respiratory illness (mild to severe)

Micro-embolism
Gastrointestinal symptoms
Dyserythropoeisis & Porphyria
Liver Dysfunction
Diabetes Adults/Kids
Cardiovascular/Heart
Renal damage
Neurological/Stroke
Skin Rashes/Sores
Kawasaki-like disease
Multiple Inflammatory Syndrome
Recovery- if so from what?
PACS and Long-term effects

Multiple Possible Time Frames of Diagnosis, Prognosis and Therapeutic Intervention Monitoring

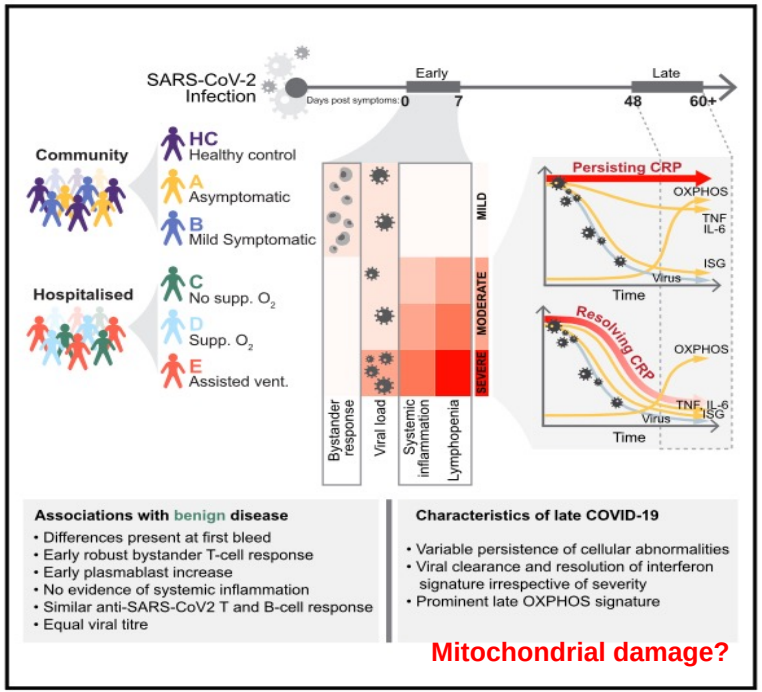
WHAT DETERMINES SEVERITY?

Immunity

Longitudinal analysis reveals that delayed bystander CD8+ T cell activation and early immune pathology distinguish severe COVID-19 from mild disease

Australian National Phenome Centre
Murdoch University

Graphical Abstract



Authors

Laura Bergamaschi, Federica Mescia, Lorinda Turner, ..., John R. Bradley, Paul A. Lyons, Kenneth G.C. Smith

Highlights

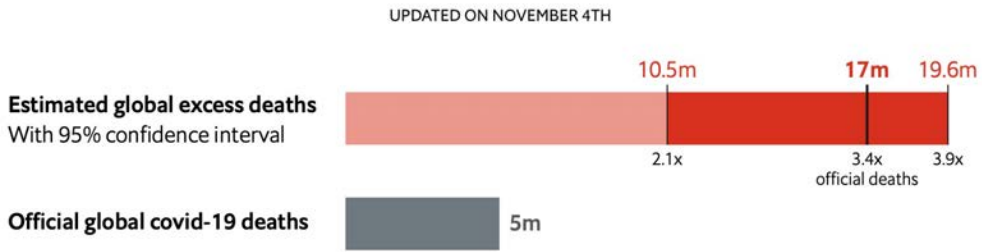
- Longitudinal analysis of COVID-19 patients with a range of disease severity
- Early bystander CD8$^+$ T cell and plasmablast responses characterize mild disease
- Pronounced systemic inflammation evident at first presentation in more severe COVID-19
- Immune/inflammatory abnormalities persist in severe disease to 60 days post symptoms

Daily chart

The number of people who have died from covid-19 is likely to be close to 17m

The pandemic's true death toll

Our daily estimate of excess deaths around the world



Coronavirus (COVID-19) Vaccinations

51.3% of the world population has received at least one dose of a COVID-19 vaccine.

7.36 billion doses have been administered globally, and **27.83 million** are now administered each day.

Only **4.4%** of people in low-income countries have received at least one dose.

Article

Sensitivity of SARS-CoV-2 B.1.1.7 to mRNA vaccine-elicited antibodies

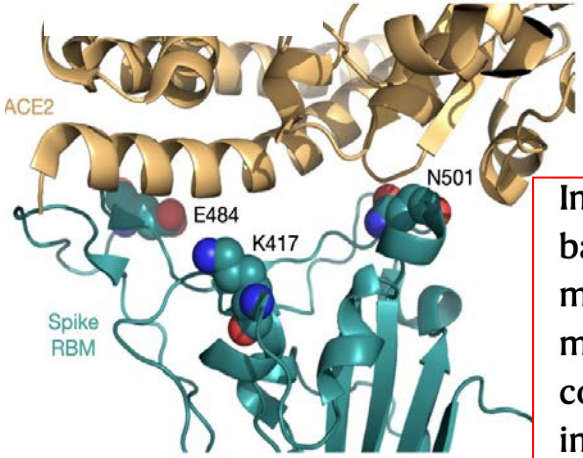
https://doi.org/10.1038/s41586-021-03412-7

Received: 26 January 2021

Accepted: 1 March 2021

Published online: 11 March 2021

Dami A. Collier[1,2,3,144], Anna De Marco[4,144], Isabella A. T. M. Ferreira[1,2,144], Bo Meng[1,2,144], Rawlings P. Datir[1,2,3,144], Alexandra C. Walls[5], Steven A. Kemp[1,2,3], Jessica Bassi[4], Dora Pinto[4], Chiara Silacci-Fregni[4], Siro Bianchi[4], M. Alejandra Tortorici[5], John Bowen[5], Katja Culap[4], Stefano Jaconi[4], Elisabetta Cameroni[4], Gyorgy Snell[6], Matteo S. Pizzuto[4], Alessandra Franzetti Pellanda[7], Christian Garzoni[7], Agostino Riva[8], The CITIID-NIHR BioResource COVID-19 Collaboration*, Anne Elmer[9], Nathalie Kingston[10], Barbara Graves[10], Laura E. McCoy[3], Kenneth G. C. Smith[1,2], John R. Bradley[2,10], Nigel Temperton[11], Lourdes Ceron-Gutierrez[12], Gabriela Barcenas-Morales[12,13], The COVID-19 Genomics UK (COG-UK) Consortium*, William Harvey[14], Herbert W. Virgin[6], Antonio Lanzavecchia[4], Luca Piccoli[4], Rainer Doffinger[12,15], Mark Wills[2], David Veesler[5], Davide Corti[4,145]✉ & Ravindra K. Gupta[1,2,15,16,17,18,145]✉



Introduction of the mutation that encodes the E484K substitution in the B.1.1.7 background to reflect a newly emerged variant of concern (VOC 202102/02) led to a more-substantial loss of neutralizing activity by vaccine-elicited antibodies and monoclonal antibodies (19 out of 31) compared with the loss of neutralizing activity conferred by the mutations in B.1.1.7 alone. The emergence of the E484K substitution in a B.1.1.7 background represents a threat to the efficacy of the BNT162b2 vaccine.

Revealed: Thousands of double jabbed over 50s have died from COVID in the last 4 weeks

Connor Parker

Fri, 12 November 2021,

Older people were vaccinated earlier in UK (and Australia)…
So now the new infective variants are in UK they have lower protection
But without any vaccination it would be ten times worse!!
BOOSTERS ARE REQUIRED! AND TUNED TO NEW VARIANTS…

Community transmission and viral load kinetics of the SARS-CoV-2 delta (B.1.617.2) variant in vaccinated and unvaccinated individuals in the UK: a prospective, longitudinal, cohort study

www.thelancet.com/infection **Published online October 28, 2021** **https://doi.org/10.1016/S1473-3099(21)00648-4**

Anika Singanayagam, Seran Hakki*, Jake Dunning*, Kieran J Madon, Michael A Crone, Aleksandra Koycheva, Nieves Derqui-Fernandez, Jack L Barnett, Michael G Whitfield, Robert Varro, Andre Charlett, Rhia Kundu, Joe Fenn, Jessica Cutajar, Valerie Quinn, Emily Conibear, Wendy Barclay, Paul S Freemont, Graham P Taylor, Shazaad Ahmad, Maria Zambon, Neil M Ferguson†, Ajit Lalvani†, on behalf of the ATACCC Study Investigators‡*

Implications of all the available evidence

Although vaccines remain highly effective at preventing severe disease and deaths from COVID-19, our findings suggest that vaccination is not sufficient to prevent transmission of the delta variant in household settings with prolonged exposures. Our findings highlight the importance of community studies to characterise the epidemiological phenotype of new SARS-CoV-2 variants in increasingly highly vaccinated populations. Continued public health and social measures to curb transmission of the delta variant remain important, even in vaccinated individuals.

Australian-first study finds most critical COVID patients have symptoms for months

By national medical reporter Sophie Scott and the Specialist Reporting Team's Lucy Kent

- **Key points:**
  - An Australian-first study has examined COVID's long-term impacts on people who were admitted to ICU
  - It found a large number struggled to complete normal activities, like walking, after their infection
  - A long COVID sufferer still has "brain fog" and trouble walking, more than 12 months after her ICU stay

Symptoms six months after ICU:

Symptom	Percentage of patients reporting
Shortness of breath	34.8 per cent
Loss of strength	21.7 per cent
Fatigue	19.1 per cent
Persistent cough	13.9 per cent
Loss of taste/smell	12.2 per cent

Contents lists available at ScienceDirect

EClinicalMedicine

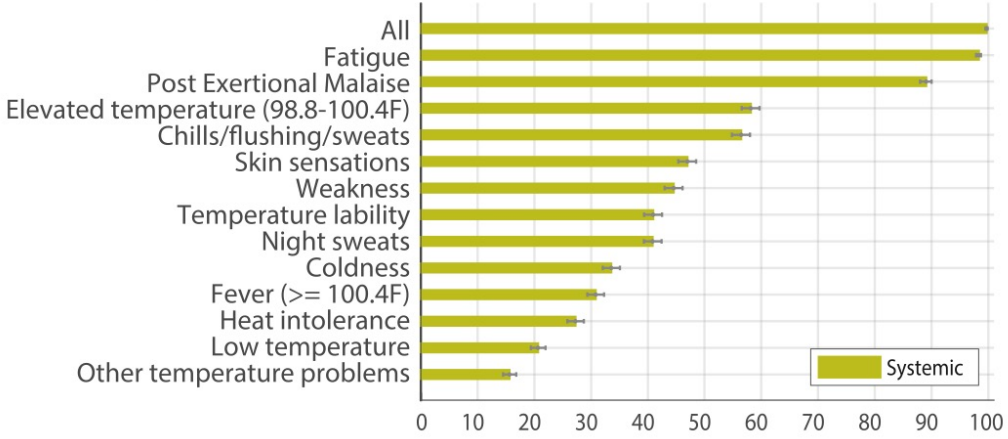
journal homepage: https://www.journals.elsevier.com/eclinicalmedicine

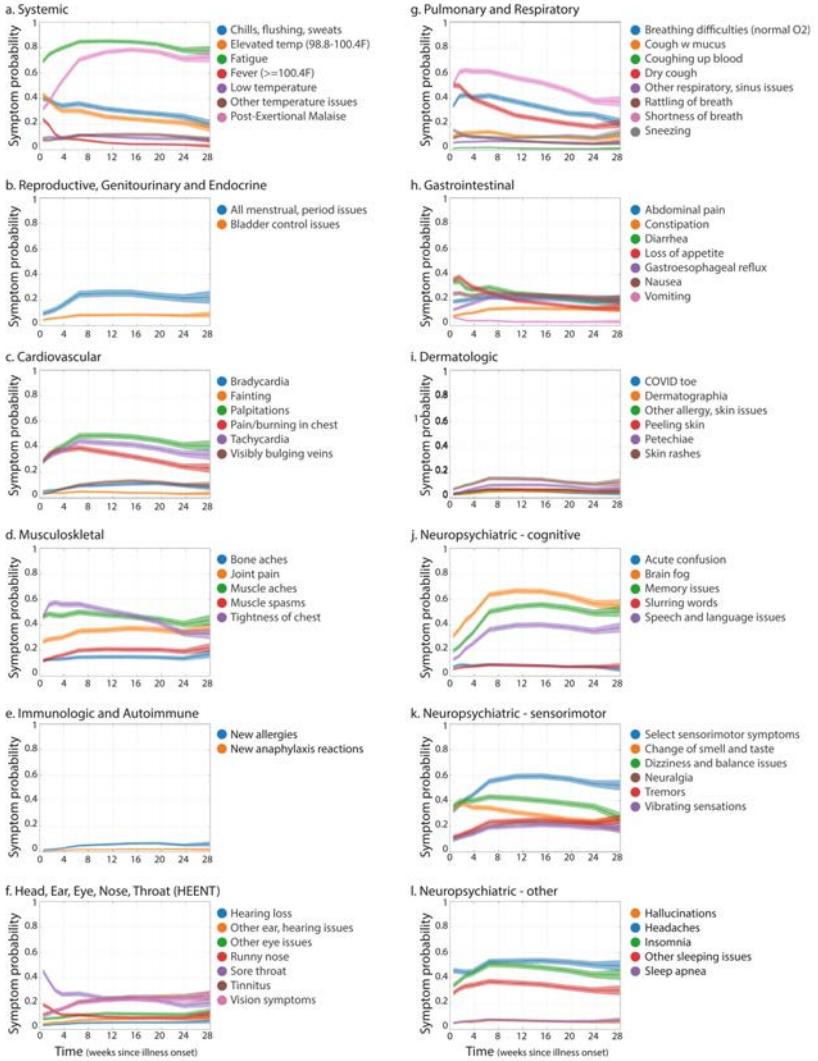
Research paper

Characterizing long COVID in an international cohort: 7 months of symptoms and their impact

Hannah E. Davis[a,1], Gina S. Assaf[a,1], Lisa McCorkell[a,1], Hannah Wei[a,1], Ryan J. Low[a,b,1], Yochai Re'em[a,c,1], Signe Redfield[a], Jared P. Austin[a,d], Athena Akrami[a,b,1,*]

Findings: For the majority of respondents (>91%), the time to recovery exceeded 35 weeks. During their illness, participants experienced an average of 55.9+/- 25.5 (mean+/-STD) symptoms, across an average of 9.1 organ systems. The most frequent symptoms after month 6 were fatigue, post-exertional malaise, and cognitive dysfunction. Symptoms varied in their prevalence over time, and we identified three symptom clusters, each with a characteristic temporal profile. 85.9% of participants (95% CI, 84.8% to 87.0%) experienced relapses, primarily triggered by exercise, physical or mental activity, and stress. 86.7% (85.6% to 92.5%) of unrecovered respondents were experiencing fatigue at the time of survey, compared to 44.7% (38.5% to 50.5%) of recovered respondents. 1700 respondents (45.2%) required a reduced work schedule compared to pre-illness, and an additional 839 (22.3%) were not working at the time of survey due to illness. Cognitive dysfunction or memory issues were common across all age groups (~88%). Except for loss of smell and taste, the prevalence and trajectory of all symptoms were similar between groups with confirmed and suspected COVID-19.





nature medicine

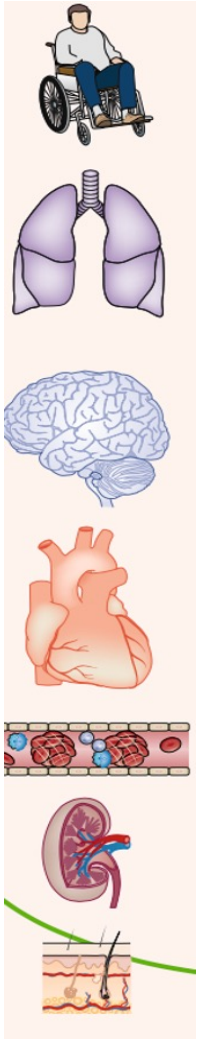
FOCUS | REVIEW ARTICLE

https://doi.org/10.1038/s41591-021-01283-z

Post-acute COVID-19 syndrome

Ani Nalbandian[1,24], Kartik Sehgal[2,3,4,24]✉, Aakriti Gupta[1,5,6], Mahesh V. Madhavan[1,5], Claire McGroder[7], Jacob S. Stevens[8], Joshua R. Cook[9], Anna S. Nordvig[10], Daniel Shalev[11], Tejasav S. Sehrawat[12], Neha Ahluwalia[13], Behnood Bikdeli[4,5,6,14], Donald Dietz[15], Caroline Der-Nigoghossian[16], Nadia Liyanage-Don[17], Gregg F. Rosner[1], Elana J. Bernstein[18], Sumit Mohan[8], Akinpelumi A. Beckley[19], David S. Seres[20], Toni K. Choueiri[2,3,4], Nir Uriel[1], John C. Ausiello[9], Domenico Accili[9], Daniel E. Freedberg[21], Matthew Baldwin[7], Allan Schwartz[1], Daniel Brodie[7], Christine Kim Garcia[7], Mitchell S. V. Elkind[10,22], Jean M. Connors[4,23], John P. Bilezikian[9], Donald W. Landry[8] and Elaine Y. Wan[1]✉

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is the pathogen responsible for the coronavirus disease 2019 (COVID-19) pandemic, which has resulted in global healthcare crises and strained health resources. As the population of patients recovering from COVID-19 grows, it is paramount to establish an understanding of the healthcare issues surrounding them. COVID-19 is now recognized as a multi-organ disease with a broad spectrum of manifestations. Similarly to post-acute viral syndromes described in survivors of other virulent coronavirus epidemics, there are increasing reports of persistent and prolonged effects after acute COVID-19. Patient advocacy groups, many members of which identify themselves as long haulers, have helped contribute to the recognition of post-acute COVID-19, a syndrome characterized by persistent symptoms and/or delayed or long-term complications beyond 4 weeks from the onset of symptoms. Here, we provide a comprehensive review of the current literature on post-acute COVID-19, its pathophysiology and its organ-specific sequelae. Finally, we discuss relevant considerations for the multidisciplinary care of COVID-19 survivors and propose a framework for the identification of those at high risk for post-acute COVID-19 and their coordinated management through dedicated COVID-19 clinics.



My COVID fears for our kids

Risk of long-term symptoms raise stakes for vaccinating children

ANDREW MILLER

> If young kids go on to develop long-term problems, they have the most years of development and quality life to lose.

MOR

You won't be surprised to hear that I was not one of the popular crowd in high school.

I was in the majority who found adolescence to be a challenge rather than a banquet of new opportunities. I had good friends who were on every A-team though, and one of the few obvious downsides of their enviable party lifestyle was the risk of "kissing disease".

Glandular fever, mononucleosis, or mono, is caused by the Epstein-Barr virus and I have been thinking about it a lot lately. One in ten diagnosed with EBV suffered badly from it for a long time, often with little sympathy for months or years.

Chronic fatigue made them sleep 18 hours a day, miss school terms, slip in academic performance, and miss out on social life and opportunities for work. Some ended up taking a gap year, sometimes two. I have heard the quiet anguish in one parent's voice who says that two years after a mild viral illness their beautiful son still can't get out of bed feeling good.

In this context the emerging spectre of "long COVID" sends a shiver up the spine, especially given the scale of this pandemic. Early reports that symptoms like brain fog and lethargy persist for months in some children have translated into news that about 7 per cent of those under 18 years old still have disabling symptoms more than three months after testing positive for SARS-Cov-2.

The National Health Service in the United Kingdom has set up 15 long COVID clinics for children costing £100 million ($180.8 million). The American Academy of Pediatrics says hese parents and kids o be believed, studied, rted and treated. plausible that this new avirus could be causing the symptoms, as opposed to other factors such as the disruption that has come from lockdowns, restrictions and loss of family members?

In WA we are fortunate to have the world leading Australian National Phenome Centre at Murdoch University.

This incredible facility is a science detective laboratory where Professor Jeremy Nicholson leads a team that is drilling down into the fine detail of the damage that COVID can do.

"COVID-19 causes significant multisystem long-term effects including liver dysfunction, kidney dysfunction, cardiovascular disease, anosmia (loss of smell) and neurological problems, and a host of other rarer complications," he says. "We're seeing that a high proportion of patients are still presenting with multiple symptoms months, and possibly years, after the acute phase."

So yes, SARS-Cov-2 could well be the culprit.

In Germany the big data, from the health records of 45 per cent of the population, analysed over two years from January 2019, is showing marked increases in many health problems in children and adults who had COVID.

This includes neurological problems, which fits with what our disease detectives expect.

How can we prevent this? In short, vaccines for all. Just last week, in a much-anticipated decision, the authorities in the US unanimously approved Pfizer vaccine for children aged 5 to 11 years. Clinics are already administering the jabs to an enthusiastic crush of kids.

This will be a major advance, and not just for them, because school-aged children are a major source of Delta variant spread. With 300 disruptive school closures last week in Victoria, it seems certain that Australia will follow suit.

Although they rarely die from COVID, children get hospitalised at a rate of about one per cent for various reasons after testing positive.

That's a lot of kids in hospital getting traumatised and potentially infecting very vulnerable patients who are in there for other serious diseases such as cancer. If young kids go on to develop long-term problems, they have the most years of development and quality life to lose.

Understandably the intense safety data required for vaccines in the 0 to 4-year-old group will take even longer to scrutinise, but given that children aged under one do worse than older kids with COVID infection, we should expect a quick turnaround on the decision to vaccinate down to six months, just as we encourage annually for influenza.

What can we do in the meantime? The McGowan Government's greatest legacy in the long run may well be preventing the compound long-term burden of disease and suffering in our children by keeping WA COVID-free for the rest of 2021.

This could allow enough time for parents to get their kids the Pfizer present that the US is having for Thanksgiving.

As for "kissing disease", Moderna has announced a vaccine is in development.

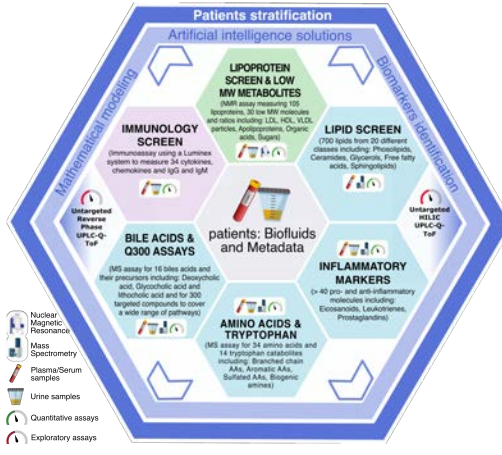
A silver lining to the horror of COVID could be that many diseases like EBV, malaria, respiratory syncytial virus and even some cancers can all be pushed back by resurgent vaccine innovation.

All wins, including for the popular kids, that we should celebrate without envy.

Dr Andrew Miller is president of the Australian Society of Anaesthetists and past president & board director AMA WA. His views are not those of any particular organisation.


BRUKER
Ascend™ 600
IVDR Ø2
SAMPLE


NO FOOD
OR DRINK
IN THIS AREA
PC2
MICROBIOLOGICAL
LABORATORY
Working apart
keeps us
together
Mass Spectry


Patients stratification
Artificial intelligence solutions
Mathematical modeling
Biomarkers identification
LIPOPROTEIN SCREEN & LOW MW METABOLITES
(NMR assay measuring 105 lipoproteins, 30 low MW molecules and ratios including: LDL, HDL, VLDL particles, Apolipoproteins, Organic acids, Sugars)
IMMUNOLOGY SCREEN
(Immunoassay using a Luminex system to measure 34 cytokines, chemokines and IgG and IgM
LIPID SCREEN
(700 lipids from 20 different classes including: Phospholipids, Ceramides, Glycerols, Free fatty acids, Sphingolipids)
Untargeted Reverse Phase UPLC-Q-ToF
Untargeted HILIC UPLC-Q-ToF
patients: Biofluids and Metadata
BILE ACIDS & Q300 ASSAYS
(MS assay for 16 biles acids and their precursors including: Deoxycholic acid, Glycocholic acid and lithocholic acid and for 300 targeted compounds to cover a wide range of pathways)
INFLAMMATORY MARKERS
(> 40 pro- and anti-inflammatory molecules including: Eicosanoids, LeuAnhmenes, Prostaglandins)
AMINO ACIDS & TRYPTOPHAN
(MS assay for 34 amino acids and 14 tryptophan catabolites including: Branched chain AAs, Aromatic AAs, Sulfated AAs, Biogenic amines)
Nuclear Magnetic Resonance
Mass Spectrometry
Plasma/Serum samples
Urine samples
Quantitative assays
Exploratory assays

Journal of proteome research

Article

Integrative Modelling of Quantitative Plasma Lipoprotein, Metabolic and Amino Acid Data Reveals a Multi-organ Pathological Signature of SARS-CoV-2 Infection

Torben Kimhofer, Samantha Lodge, Luke Whiley, Nicola Gray, Ruey Leng Loo, Nathan G. Lawler, Philipp Nitschke, Sze-How Bong, David L. Morrison, Sofina Begum, Toby Richards, Bu B. Yeap, Chris Smith, Kenneth C.G. Smith, Elaine Holmes, and Jeremy K. Nicholson

J. Proteome Res., **Just Accepted Manuscript** • DOI: 10.1021/acs.jproteome.0c00519 • Publication Date (Web): 17 Aug 2020

Journal of proteome research

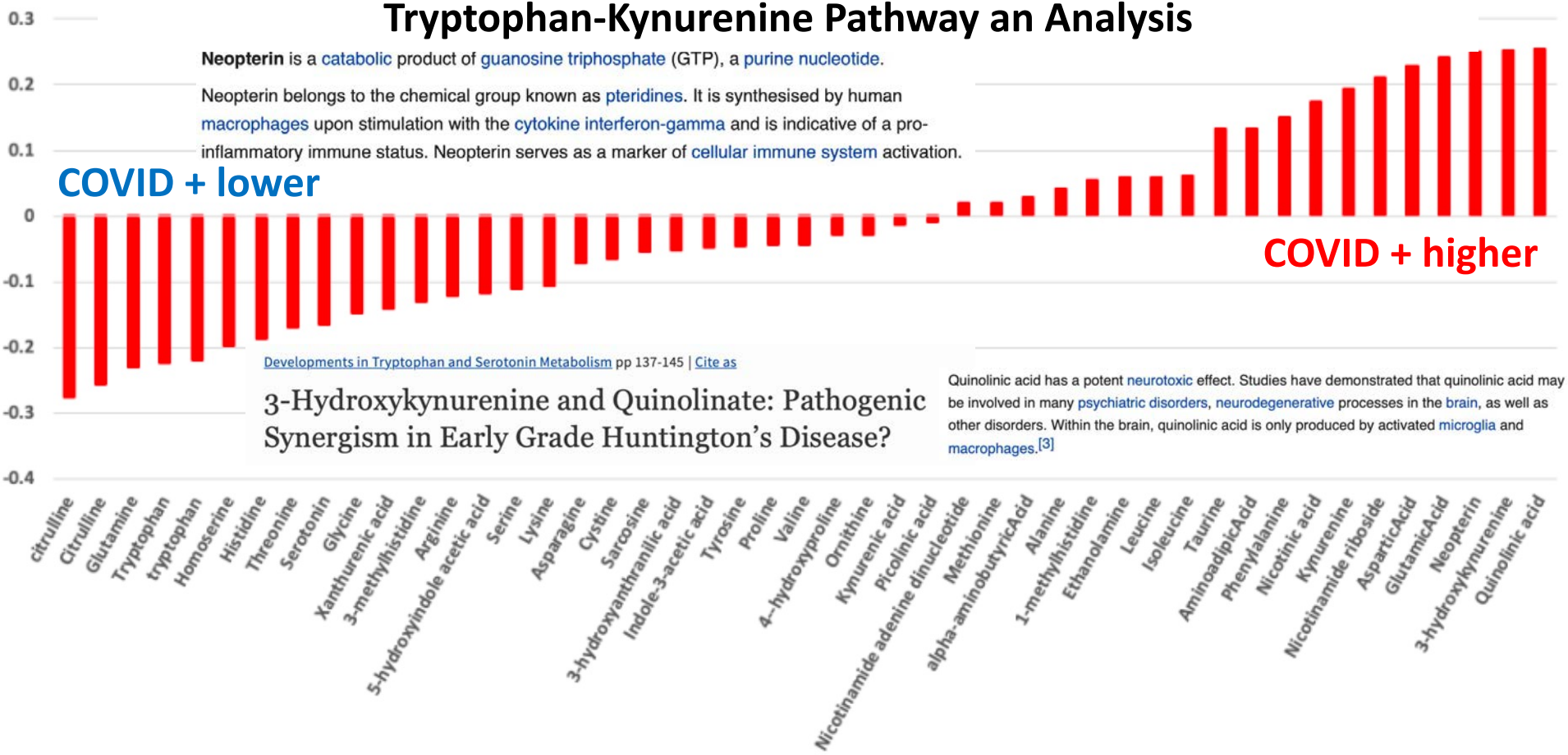
pubs.acs.org/jpr

Article

Systemic Perturbations in Amine and Kynurenine Metabolism Associated with Acute SARS-CoV-2 Infection and Inflammatory Cytokine Responses

Nathan G. Lawler,[◎] Nicola Gray,[◎] Torben Kimhofer, Berin Boughton, Melvin Gay, Rongchang Yang, Aude-Claire Morillon, Sung-Tong Chin, Monique Ryan, Sofina Begum, Sze How Bong, Jerome D. Coudert, Dale Edgar, Edward Raby, Sven Pettersson, Toby Richards, Elaine Holmes, Luke Whiley,* and Jeremy K. Nicholson*

Variable Importance Plot for Combined Amino Acid/Biogenic Amine Tryptophan-Kynurenine Pathway an Analysis



Neopterin is a catabolic product of guanosine triphosphate (GTP), a purine nucleotide.

Neopterin belongs to the chemical group known as pteridines. It is synthesised by human macrophages upon stimulation with the cytokine interferon-gamma and is indicative of a pro-inflammatory immune status. Neopterin serves as a marker of cellular immune system activation.

Developments in Tryptophan and Serotonin Metabolism pp 137-145 | Cite as

3-Hydroxykynurenine and Quinolinate: Pathogenic Synergism in Early Grade Huntington's Disease?

Quinolinic acid has a potent neurotoxic effect. Studies have demonstrated that quinolinic acid may be involved in many psychiatric disorders, neurodegenerative processes in the brain, as well as other disorders. Within the brain, quinolinic acid is only produced by activated microglia and macrophages.[3]

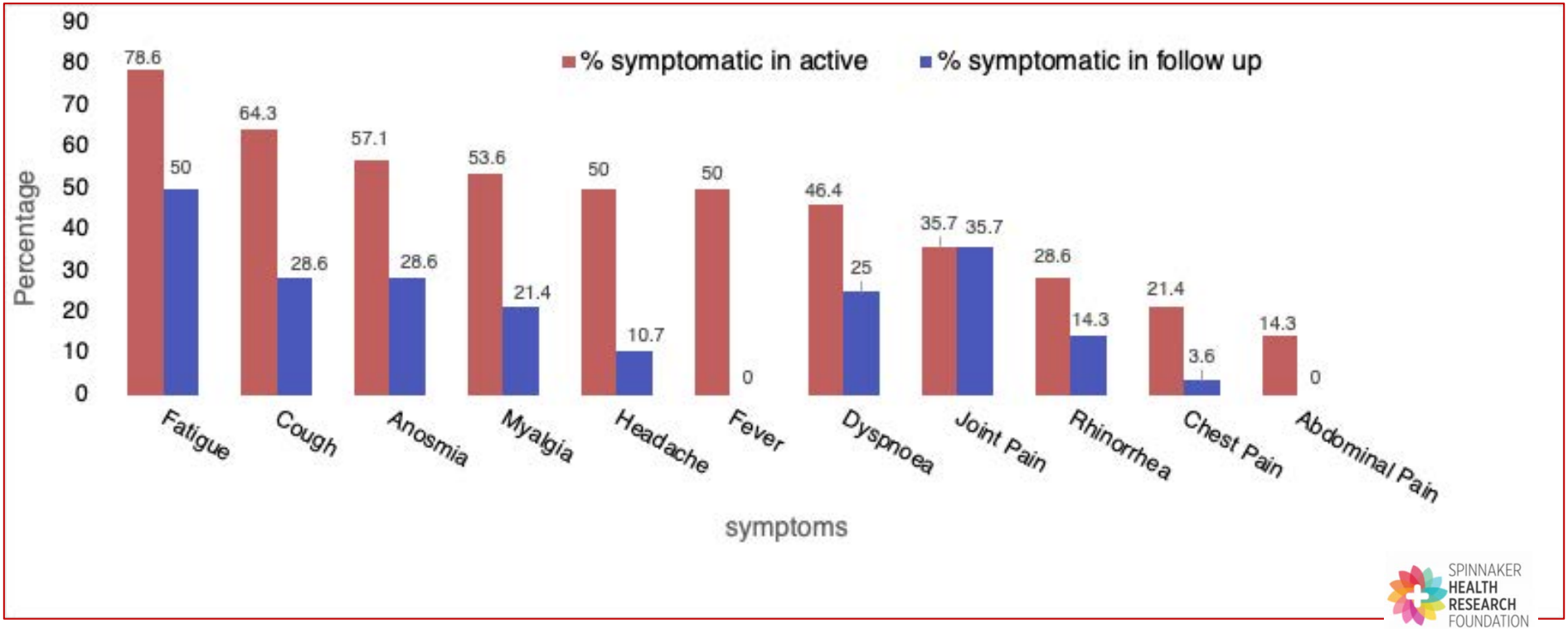
Journal of proteome research : https://doi.org/10.1021/acs.jproteome.1c00224

Incomplete Systemic Recovery and Metabolic Phenoreversion in Post-Acute-Phase Nonhospitalized COVID-19 Patients: Implications for Assessment of Post-Acute COVID-19 Syndrome

Elaine Holmes,* Julien Wist,* Reika Masuda, Samantha Lodge, Philipp Nitschke, Torben Kimhofer, Ruey-Leng Loo, Sofina Begum, Berin Boughton, Rongchang Yang, Aude-Claire Morillon, Sung-Tong Chin, Drew Hall, Monique Ryan, Sze-How Bong, Melvin Gay, Dale Edgar, John C. Lindon, Toby Richards, Bu B. Yeap, Sven Petterson, Manfred Spraul, Hartmut Schaefer, Nathan G. Lawler, Nicola Gray, Luke Whiley, and Jeremy K. Nicholson*

WA 3 month/6-month COVID-19 Follow-up Study in Non-Hospitalized Patients (57% still symptomatic)

Comparison of Early Phase Symptoms vs Persistent Symptoms (same Patients)



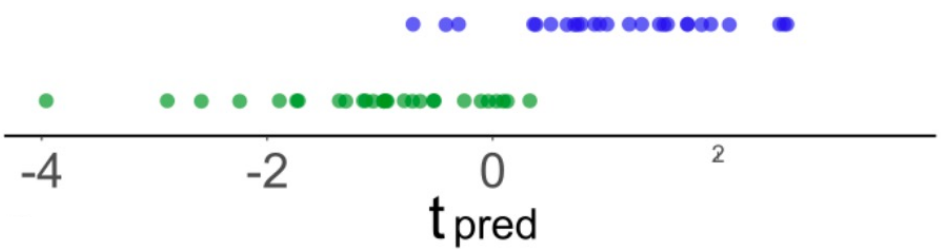
SPINNAKER HEALTH RESEARCH FOUNDATION

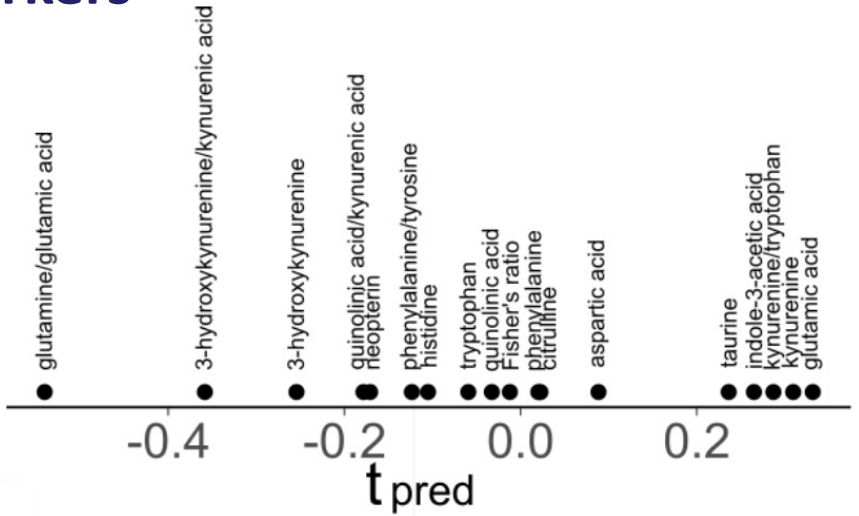
Many patients are NOT biochemically recovered- with persistent COVID-positive biomarkers

Amino acid-OPLS-model

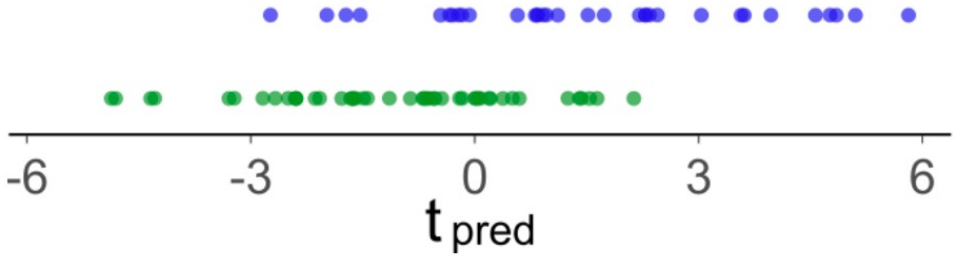
Blue-follow-up

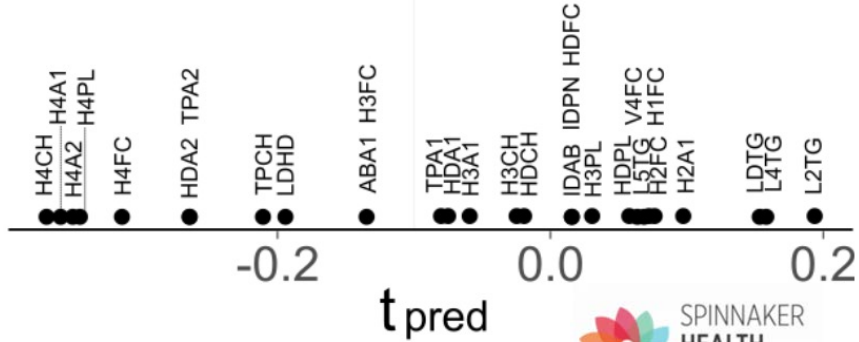
Green- Healthy





Lipoprotein-OPLS-model





SPINNAKER HEALTH RESEARCH FOUNDATION

Univariate Metabolic Analysis of 3 month follow up WA patients

Can we measure recovery from COVID-19 by Measuring return to normality of perturbed metabolites?

Red = mainly not recovered

Blue = mainly recovered

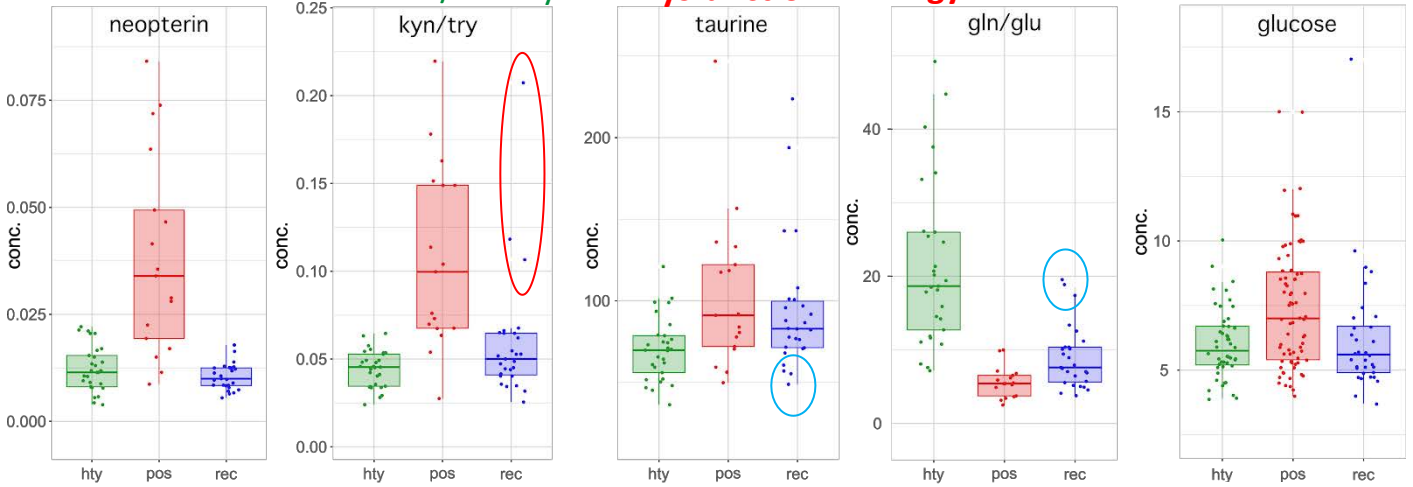
Cellular immune activation

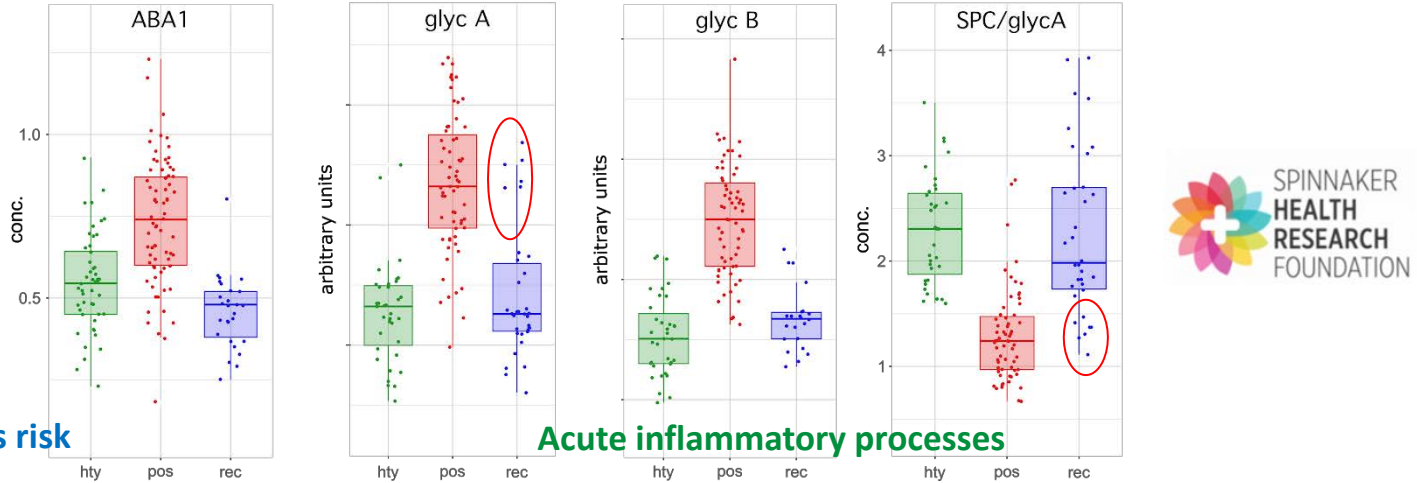
Neuro-inflammatory Liver/Kidney

Liver/Heart Dysfunction

Neuro-disorders Energy metabolism

Diabetes





Cardiovascular Atherosclerosis risk

Acute inflammatory processes

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COVID-19 - What we know from ANPC studies so far…

DISEASE DETECTION BY PLASMA PHENOCONVERSION: A series of Phenoconversion Models give High 95-99% sensitivities (low false negatives and false positives) –

SEVERITY PREDICTION IS POSSIBLE FROM EARLY SAMPLES…..

METABOLIC DATA REVEAL A COMPLEX MULTI-ORGAN SYSTEMS FAILURE PATTERN:

Diabetes, Liver Dysfunction, Porphyria, Renal Damage, Acute Systemic Inflammation, Cardiovascular and Atherosclerotic signatures, Neurological damage- Possible links to Chronic Fatigue Syndrome, Anosmia, sudden onset deafness etc.

“RECOVERY” FOR COVID-19 HAS BEEN REDEFINED FOR THE WHOLE SYSTEM:

“Recovery” varies between individuals from weeks to months or not at all. Reversibility can be directly assessed and potentially linked to other long term health effects and disease risks

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