The Daily COVID-19 Literature Surveillance Summary

March 08, 2021

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COVID-19 Daily Literature Surveillance

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LEVEL OF EVIDENCE

Oxford Centre for Evidence-Based Medicine 2011 Levels of Evidence

Question	Step 1 (Level 1*)	Step 2 (Level 2*)	Step 3 (Level 3*)	Step 4 (Level 4*)	Step 5 (Level 5)
How common is the problem?	Local and current random sample surveys (or censuses)	Systematic review of surveys that allow matching to local circumstances**	Local non-random sample**	Case-series**	n/a
Is this diagnostic or monitoring test accurate? (Diagnosis)	Systematic review of cross sectional studies with consistently applied reference standard and blinding	Individual cross sectional studies with consistently applied reference standard and blinding	Non-consecutive studies, or studies without consistently applied reference standards**	Case-control studies, or "poor or non-independent reference standard**	Mechanism-based reasoning
What will happen if we do not add a therapy? (Prognosis)	Systematic review of inception cohort studies	Inception cohort studies	Cohort study or control arm of randomized trial*	Case-series or case-control studies, or poor quality prognostic cohort study**	n/a
Does this intervention help? (Treatment Benefits)	Systematic review of randomized trials or *n*-of-1 trials	Randomized trial or observational study with dramatic effect	Non-randomized controlled cohort/follow-up study**	Case-series, case-control studies, or historically controlled studies**	Mechanism-based reasoning
What are the COMMON harms? (Treatment Harms)	Systematic review of randomized trials, systematic review of nested case-control studies, *n*-of-1 trial with the patient you are raising the question about, or observational study with dramatic effect	Individual randomized trial or (exceptionally) observational study with dramatic effect	Non-randomized controlled cohort/follow-up study (post-marketing surveillance) provided there are sufficient numbers to rule out a common harm. (For long-term harms the duration of follow-up must be sufficient.)**	Case-series, case-control, or historically controlled studies**	Mechanism-based reasoning
What are the RARE harms? (Treatment Harms)	Systematic review of randomized trials or *n*-of-1 trial	Randomized trial or (exceptionally) observational study with dramatic effect			
Is this (early detection) test worthwhile? (Screening)	Systematic review of randomized trials	Randomized trial	Non -randomized controlled cohort/follow-up study**	Case-series, case-control, or historically controlled studies**	Mechanism-based reasoning

*** Level may be graded down on the basis of study quality, imprecision, indirectness (study PICO does not match questions PICO), because of inconsistency between studies, or because the absolute effect size is very small; Level may be graded up if there is a large or very large effect size.**

**** As always, a systematic review is generally better than an individual study.**

How to cite the Levels of Evidence Table

OCEBM Levels of Evidence Working Group*. "The Oxford 2011 Levels of Evidence".

Oxford Centre for Evidence-Based Medicine. http://www.cebm.net/index.aspx?o=5653

** OCEBM Table of Evidence Working Group = Jeremy Howick, Iain Chalmers (James Lind Library), Paul Glasziou, Trish Greenhalgh, Carl Heneghan, Alessandro Liberati, Ivan Moschetti, Bob Phillips, Hazel Thornton, Olive Goddard and Mary Hodgkinson*

EXECUTIVE SUMMARY

Climate

- Mapping inequality in SARS-CoV-2 households shows disproportionate exposure and transmission risk for people of color and those living in poverty. Health policy experts from Stanford University in California mapped inequalities in transmission and household COVID-19 exposure risk in the United States using a publicly available dataset. They found 5.6% of the population live in high-risk households; 76% of members of these households are people of color and 58% live below 200% of the poverty line. Given these structural inequities in COVID-19 risk, authors suggest public health campaigns (testing, vaccination etc.) should focus on such households and policies facilitating prevention measures (i.e. paid time off, temporary housing for quarantine) must be implemented.

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MAPPING INEQUALITY IN SARS-COV-2 HOUSEHOLD EXPOSURE AND TRANSMISSION RISK IN THE USA

Reitsma MB, Salomon JA, Goldhaber-Fiebert JD.. J Gen Intern Med. 2021 Feb 18. doi: 10.1007/s11606-021-06603-0. Online ahead of print.

Level of Evidence: 1 - Local and current random sample surveys (or censuses)

BLUF

Health policy experts from Stanford University in California mapped inequalities in transmission and household COVID-19 exposure risk in the United States using a publicly available dataset (see summary). They found 5.6% of the population live in high-risk households (see summary for definition); most members of these households are people of color (76%) and live below 200% of the poverty line (58%)(Figure 1). Given these structural inequities in COVID-19 risk, authors suggest public health campaigns (testing, vaccination etc.) should focus on such households and policies facilitating prevention measures (i.e. paid time off, temporary housing for quarantine) must be implemented.

SUMMARY

Authors used the American Community Survey five-year (2014–2018) Public Use Microdata Sample as their data source.

High-risk households were defined as "those with (1) fewer rooms than people and (2) at least one essential worker"

FIGURES

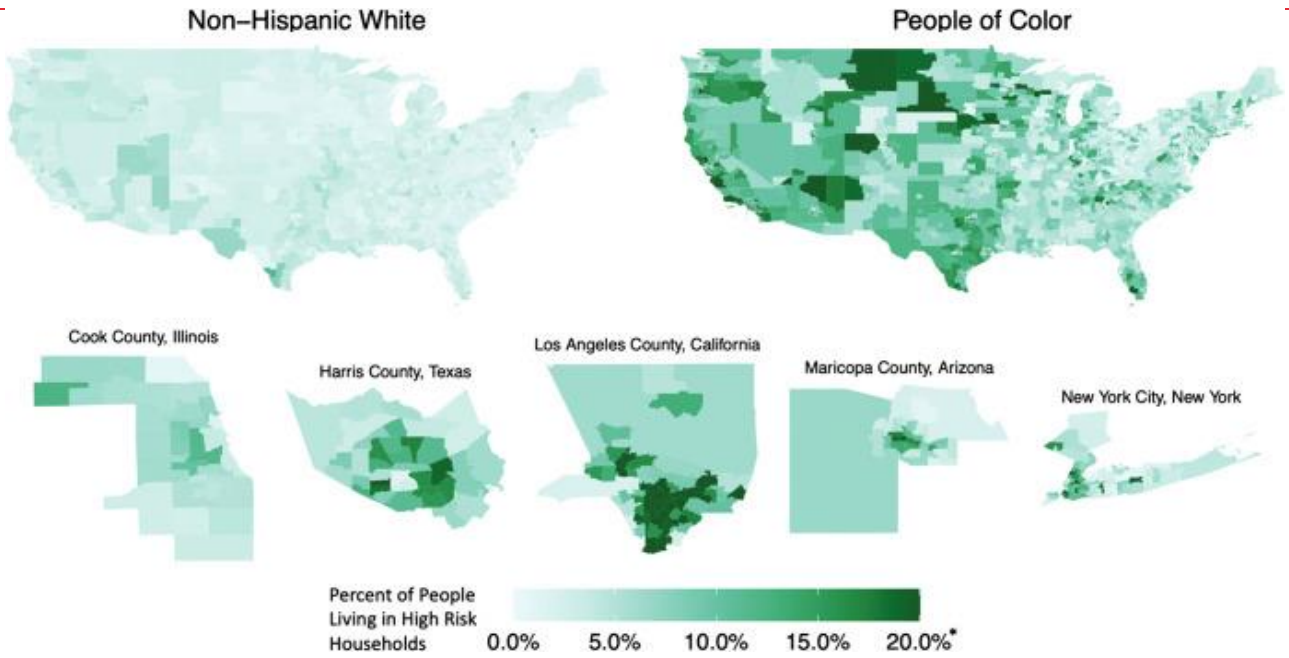

Figure 1 Percent of people living with at least one essential worker in a household with fewer rooms than people, by PUMA and race/ethnicity. Inset shows estimates for the four most-populated counties and New York City. Colors are top-coded at 20%. An asterisk indicates that 132 PUMAs were above 20% and were top-coded in the People of Color map. Three PUMAs were top-coded in the non-Hispanic White map. Thirty-nine PUMAs were top-coded in the county inset map.

ADULTS

QUANTITATIVE ASSESSMENT OF SARS-COV-2 RNAEMIA AND OUTCOME IN PATIENTS WITH CORONAVIRUS DISEASE 2019

Tang K, Wu L, Luo Y, Gong B.. J Med Virol. 2021 Feb 16. doi: 10.1002/jmv.26876. Online ahead of print.
Level of Evidence: 2 - Systematic review of inception cohort studies

BLUF

Laboratory scientists from East China Normal University in Shanghai conducted a systematic review and meta-analysis of 21 studies estimating the prevalence of SARS-CoV-2 RNAemia (see summary) and its association with disease severity (Figure 1). Despite significant heterogeneity among the studies regarding the prevalence of RNAemia (I^2=95.6%), subgroup analysis demonstrated that RNAemia was associated with severe disease (OR: 5.43, 95% CI: 3.46-8.53), ICU admission (OR: 4.28, 95% CI: 2.20-8.33), and all-cause mortality (OR: 11.07, 95% CI: 5.60-21.88) (Table 2). Authors suggest RNAemia could be used as a clinical marker to identify patients likely to develop severe COVID-19.

SUMMARY

RNAemia is defined as "the presence of viral RNA, above the technical limits of detection of PCR based assays, in blood, serum or plasma."

ABSTRACT

The disease spectrum of COVID-19 varies from asymptomatic infection to critical illness and death. Identification of prognostic marker is vital for predicting progression and clinical practice. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) RNA, known as RNAemia has been detected in blood. However, the potential clinical value of SARS-CoV-2 RNAemia remains unknown. We therefore conducted a meta-analysis using a random-effects model to estimate pooled prevalence of SARS-CoV-2 RNAemia as well as summary strength of RNAemia in association with disease severity and unfavorable clinical outcomes. A total of 21 studies involving 2181 patients were included. SARS-CoV-2 RNAemia in COVID-19 patients varied from 9.4% to 74.1%, with a pooled estimate of 34% (95% CI: 26-43%). Overall, SARS-CoV-2 RNAemia was associated with COVID-19 severity with OR of 5.43 (95% CI: 3.46-8.53). In addition, SARS-CoV-2 RNAemia was a significant risk factor for unfavorable clinical outcomes (OR = 6.54, 95% CI: 3.82-11.21). The summary OR was 4.28 (95% CI: 2.20-8.33) for ICU admission, 11.07 (95% CI: 5.60-21.88) for mortality. Furthermore, RNAemia was also a significant risk factor for invasive mechanical ventilation and multiple organ failure. SARS-CoV-2 RNAemia is associated with disease severity, ICU admission, death in COVID-19 and may serve as clinical predictor. More prospective trials in evaluating the potential of SARS-CoV-2 RNAemia as prognostic indicator are necessary.

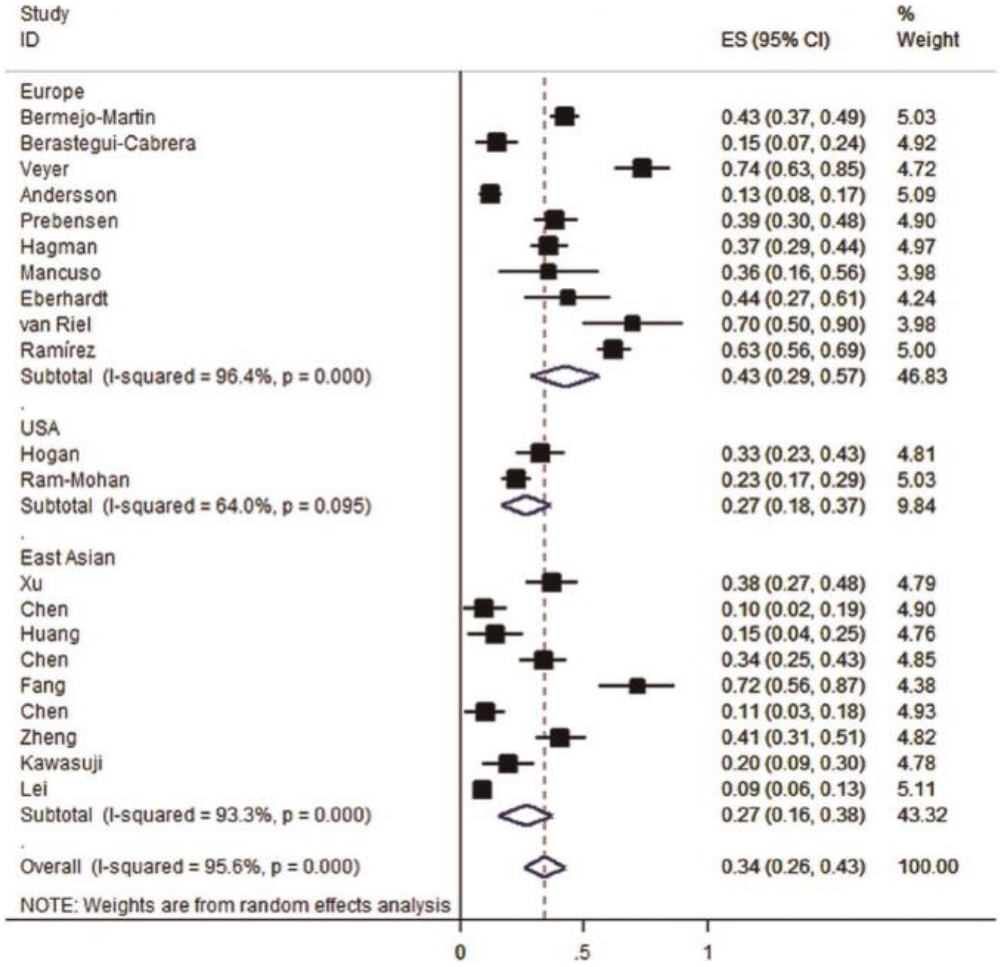

FIGURE 2 Prevalence of SARS-CoV-2 RNAemia in COVID-19 patients in different countries. Each box represents the prevalence point estimate, and its area is proportional to the weight of individual study. COVID-19, coronavirus disease 2019; SARS-CoV-2 RNAemia, severe acute respiratory syndrome coronavirus 2 RNA

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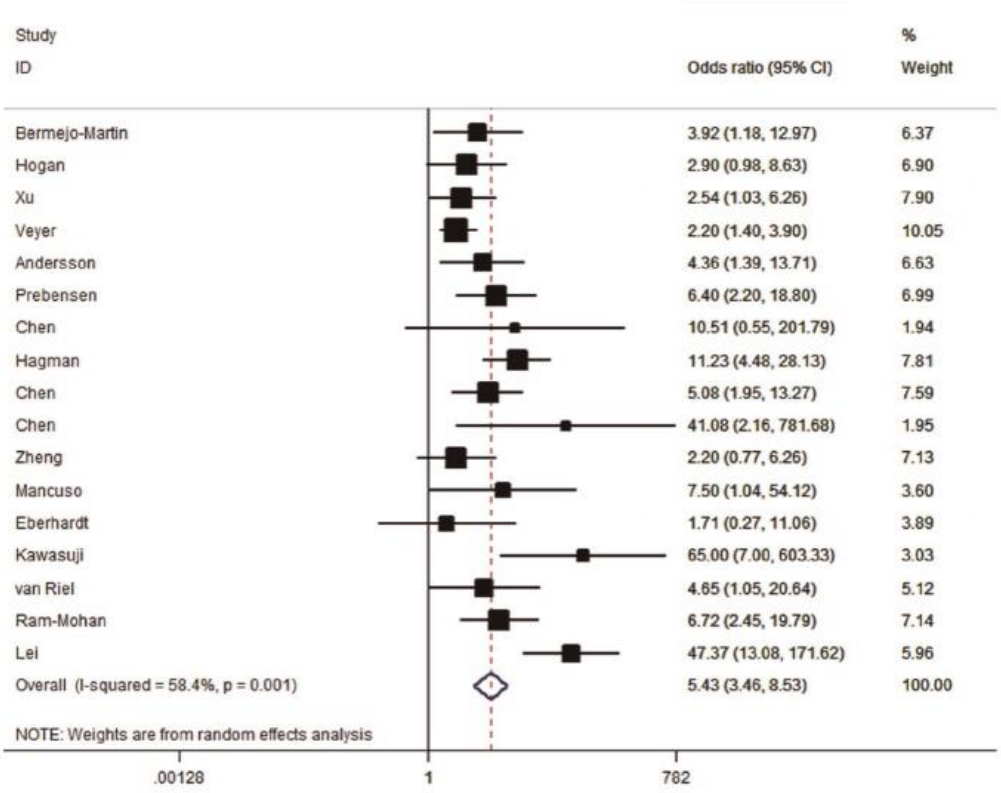

FIGURE 3 Risk for disease severity in patients with SARS-CoV-2 RNAemia versus patients without detectable SARS-CoV-2 RNA. Each box represents the OR point estimate, and its area is proportional to the weight of the individual study. OR, odds ratio; SARS-CoV-2 RNAemia, severe acute respiratory syndrome coronavirus 2 RNA

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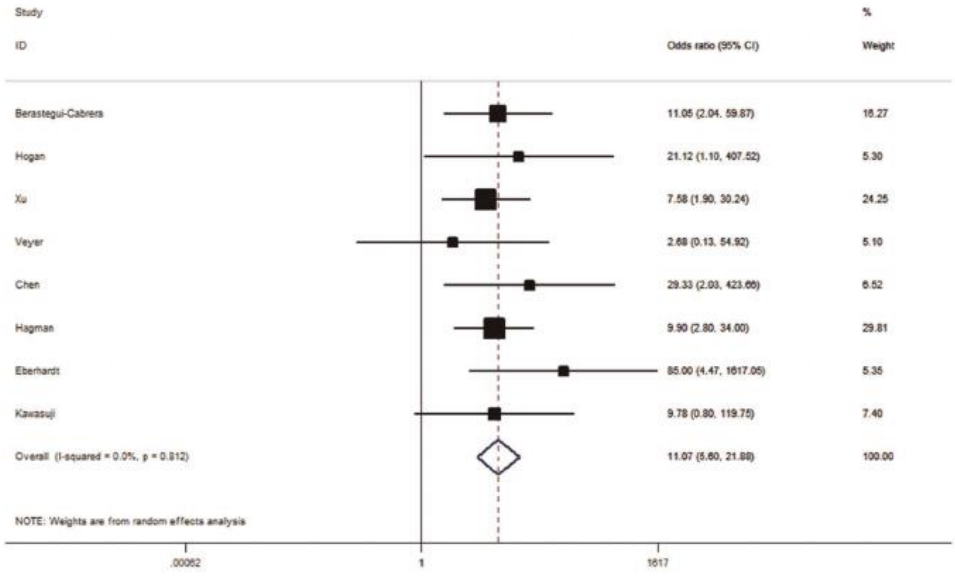


FIGURE 6 Odds ratios for all-cause mortality between the patient with and without SARS-CoV-2 RNAemia. SARS-CoV-2 RNA, severe acute respiratory syndrome coronavirus 2 RNA

FIGURE 6 Odds ratios for all-cause mortality between the patient with and without SARS-CoV-2 RNAemia. SARS-CoV-2 RNA, severe acute respiratory syndrome coronavirus 2 RNA

UNDERSTANDING THE PATHOLOGY

THE INTERPLAY BETWEEN DENDRITIC CELLS AND CD8 T LYMPHOCYTES IS A CRUCIAL COMPONENT OF SARS-COV-2 IMMUNITY

Buttenschön J, Mattner J.. Cell Mol Immunol. 2021 Jan 8. doi: 10.1038/s41423-020-00624-1. Online ahead of print.
Level of Evidence: 5 - Opinion

BLUF

Immunologists from Friedrich-Alexander Universität (FAU) Erlangen-Nürnberg in Germany comment on a recently published article which found that SARS-CoV-2 quantitatively and qualitatively impairs dendritic (DC) and CD8 T-cells (Figure 1). They summarize the findings from the paper (see summary) and explore the role of these cells in SARS-CoV-2 pathogenesis, transmission and immune function. Given waning of antibody titers in convalescent patients, authors suggest SARS-CoV-2 specific T cell responses may be more useful for determining immunogenicity in convalescent patients and vaccinated individuals rather than antibodies.

SUMMARY

The original article by Zhou, et al studied a cohort of COVID-19 patients, 8 with severe disease and 33 with mild disease. Key findings of the original paper include:
- SARS-CoV-2 significantly reduces the distribution of conventional cDC and plasmacytoid pDC
- cDC:pDC were significantly reduced in acute severe than acute mild patients
- DC and monocytes were lower in convalescent patients than the healthy donors
- Impaired antigen presentation by DCs to CD8 T cells decreased their proliferation, cytokine production(IL-2, TNFA, IFN gamma) and viral replication(Figure 1).
- DCs exhibit decreased expression of CD80, CD86.
- Both mild and severe patients developed anti-NP, anti-RBD antibodies. 23/24 convalescent patients developed anti-RBD, anti-NP antibodies which may decline in course of time.

FIGURES

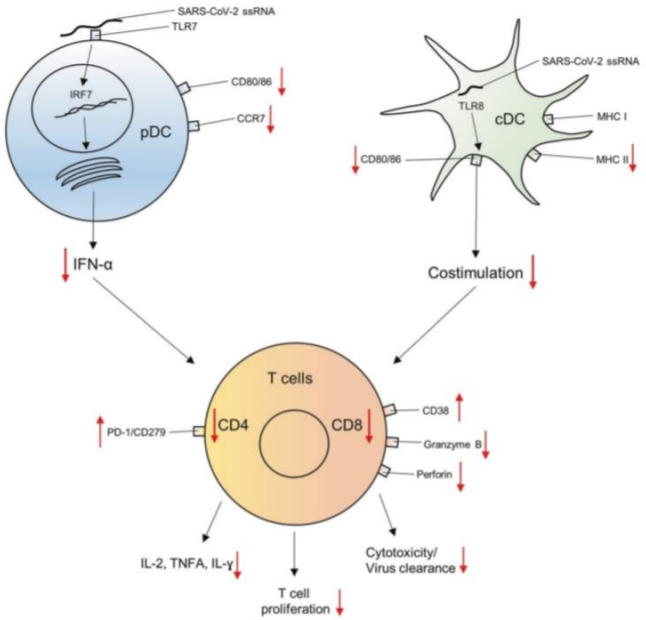


Figure 1: "A numeric reduction and impaired interaction of DCs and T lymphocytes characterizes an acute infection with SARS-CoV-2. DCs release less type I interferon and express fewer chemokines and costimulatory molecules. Subsequently, the generation and expansion of SARS-CoV-2-specific CD4 and CD8 T cells is delayed, and the release of Th1 cytokines is impaired, resulting in enhanced viral replication".

SARS-COV-2: A SYSTEMATIC REVIEW OF INDOOR AIR SAMPLING FOR VIRUS DETECTION

Borges JT, Nakada LYK, Maniero MG, Guimarães JR.. Environ Sci Pollut Res Int. 2021 Feb 25. doi: 10.1007/s11356-021-13001-w. Online ahead of print.
Level of Evidence: 5 - Review / Literature Review

BLUF

Environmental engineers from the University of Campinas conducted a systematic review of 25 publications on air sampling methods to detect SARS-CoV-2 (Table 1, Figure 1). They found solid impactors had a higher percentage of positive SARS-CoV-2 samples than liquid impactors or filters (Figure 2). Authors suggest solid impactors may be more effective than other types, but acknowledge selecting the ideal sampler type is influenced by a variety of environmental factors and will vary by scenario.

FIGURES

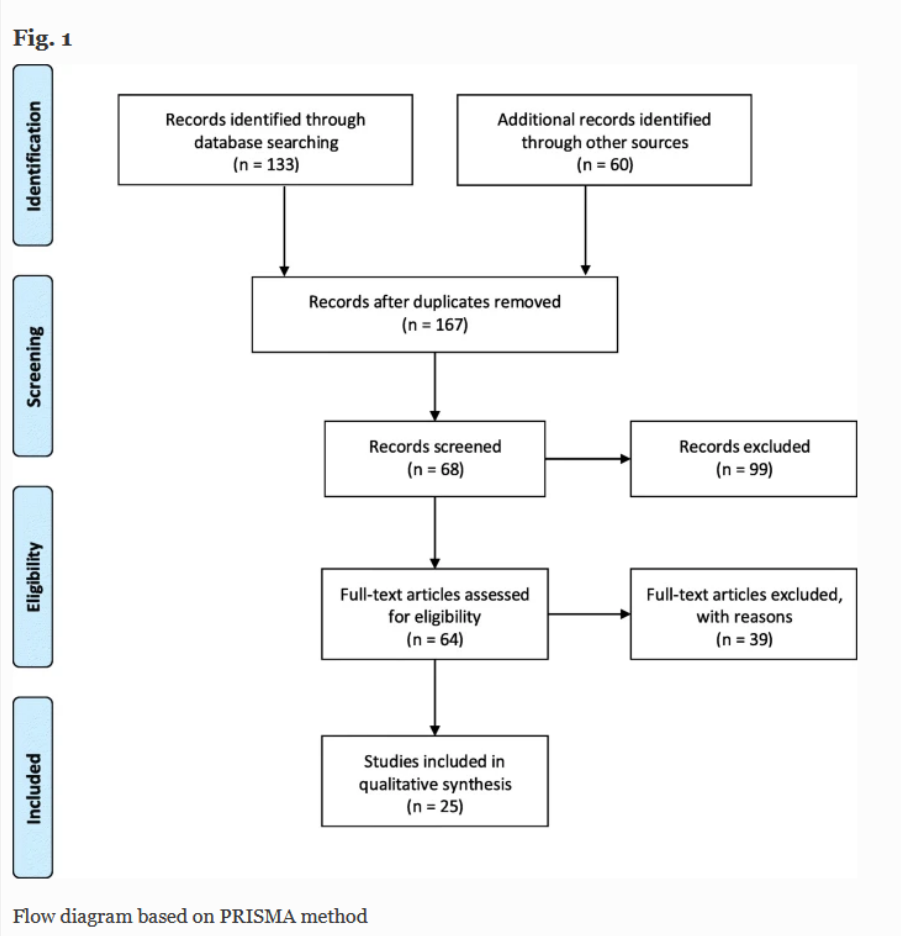


Fig. 1 Flow diagram based on PRISMA method

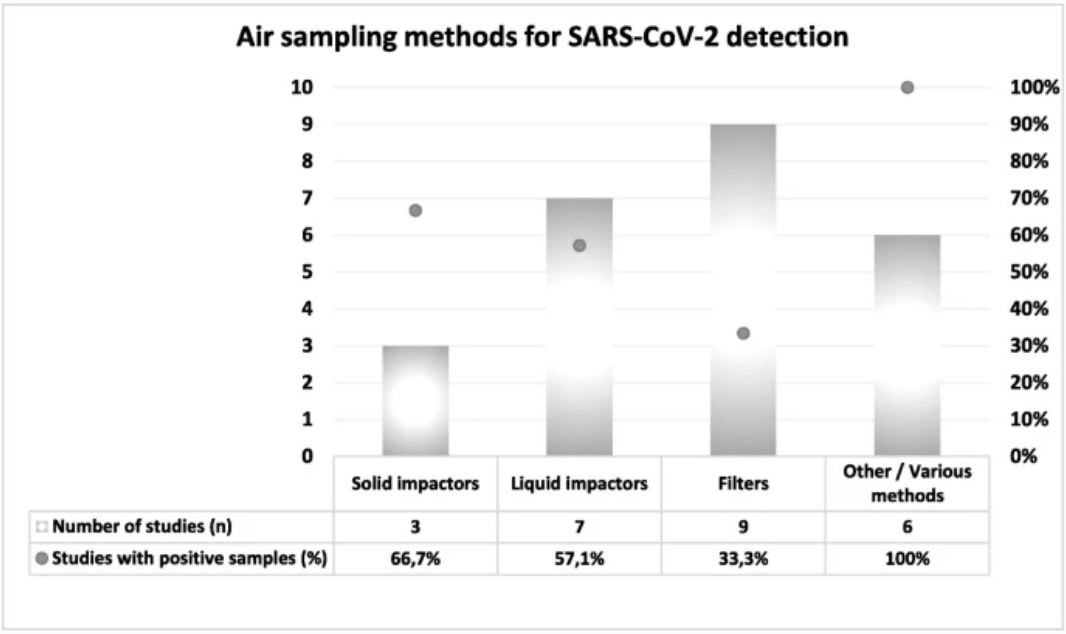
Table 1 Sample collection methods and results in investigations for the presence of viruses focusing on SARS-CoV-2 in air samples analyzed by reverse transcriptase polymerase chain reaction (RT-PCR).

From: SARS-CoV-2: a systematic review of indoor air sampling for virus detection

	Sample collection method	Result summary	Reference
Solid impactors	Air samples were collected in 3 COVID-19 patient rooms, using 6 NIOSH BC 251 bioaerosol samplers in each room, placed at different heights from the floor (1.2, 0.9, and 0.7 m), and between 1 and 2.1 m from patients. 245 surface samples were collected from 30 COVID-19 patient rooms	66.7% of the air samples were positive for SARS-CoV-2 (1.84×10^3 to 3.38×10^3 RNA copies/m^3). Rooms with viral particles in the air also presented surface contamination	Chia et al. (2020)
	33 air samples were collected for 30 min using the NIOSH BC 251 bioaerosol sampler (National Institute for Occupational Safety and Health) with air pumps (XR5000, SKC) at 3.5 L/min. The NIOSH sampler segregates air sample into large (≥ 4 μm), medium (1–4 μm), and small (< 1 μm) particles	7.7% and 82.6% of samples were positive for SARS-CoV-2 in COVID-19 respiratory investigation wards and ICUs with confirmed COVID-19 patients, respectively	Ge et al. (2020)
	28 samples were collected using 2-stage cyclone samplers (NIOSH BC 251), filter cassette containing a Teflon® filter (*D* = 37 mm and 2 μm porosity), and a sampling pump (PCXR-4, SKC, Eighty-Four, PA) at a flow rate of 3.5 L/min for 315–360 min	All samples were negative for SARS-CoV-2	Lane et al. (2020)
Liquid impactors	Air samples were collected in COVID-19 isolation rooms used for three patients with severe pneumonia, using a SKC BioSampler at 12.5 L/min and at 10 L/min, both for 20 min, placed 1.2 m from the floor, and at a 1-m distance from patients	All samples were negative for SARS-CoV-2	Ahn et al. (2020)
	10 air samples were collected during 1 h, using a vacuum pump at 1.5 L/min, in SKC sterile standard midget impingers at a height of 1.5–1.8 m from the floor and at a distance of 2–5 m from beds of patients with severe and critical symptoms	All samples were negative for SARS-CoV-2	Faridi et al. (2020)
	Air samples were collected using a SASS 2300 wetted wall cyclone sampler (Research International, Inc., https://www.resrchintl.com) at 300 L/min for 30 min	SARS-CoV-2 was detected in air at a 4-m distance from patients	Guo et al. (2020)
	Air samples were collected using a high-volume WA 400 Portable viral aerosol sampler (Dingblue Tech, Inc.), at 400 L/min for 15 min	SARS-CoV-2 was detected in 01 out of 02 air samples	Jin et al. (2020)
	Air samples were collected using the SKC impinger-type biosampler at a flow rate of 12 L/min, placed 1.5 m from the floor	02 out of 14 air samples were positive for SARS-CoV-2	Kenarkoohi et al. (2020)
	135 air samples were collected using an impinger sampler (BIO-Capturer-6, Bioenrichment Co., Hangzhou, China), for 30 min at 80 L/min, and placed 1.0–1.5 m from the floor	All samples were negative for SARS-CoV-2	Li et al. (2020)
	26 air samples were collected into 3-mL virus culture liquid (MT0301, Yocon Biology Inc., Beijing, China) using the WA-15 and WA-400 impactors (Beijing dBlueTech, Inc.) at flow rates of 15 L/min and 400 L/min, respectively	One air sample was positive for SARS-CoV-2	Ma et al. (2020)
Filters	15 air samples were collected using a dry filter air sampler (52-mm electret filters, InnovaPrep ACD-200 Bobcat, America) for 60 min at 49 L/min	All samples were negative for SARS-CoV-2	Cai et al. (2020)
	1000-L air samples from air shelters with patients were collected using the Sartorius MD8 sampler with sterile gelatin filters (*D* = 80 mm and pore size = 3 μm) (Sartorius AG, Germany) for 20 min at a flow rate of 50 L/min, being the air sampler perpendicularly positioned 10 cm from the patient's chin	All samples were negative for SARS-CoV-2	Cheng et al. (2020a)
	1000-L air samples were collected using a SAS Super ISO 180 model 86834 (VWR International PBI Srl, Milan, Italy), being the air sampler perpendicularly positioned 10 cm from the patient's chin	All samples were negative for SARS-CoV-2	Cheng et al. (2020b)
	Air samples were collected using SKC Universal pumps (with 37 mm filter cassettes and 0.3-μm Teflon® filters) for 4 h at 5 L/min, and a Sartorius MD8 microbiological sampler (with gelatin membrane filter) for 15 min at 6 m^3/h	All samples were negative for SARS-CoV-2	Ong et al. (2020)
	05 air samples were collected using an AirPort MD8 with gelatin membrane filters (Sartorius, Varedo, MB, Italy) for 40 min at 50 L/min, placed 1.5 m from the floor	All samples collected from contaminated area were positive for SARS-CoV-2	Razzini et al. (2020)
	24 high-volume air samples were collected using a Sartorius Airport MD8 air sampler at 50 L/min for 15 min, and gelatin filters (*D* = 80 mm); and 08 low-volume personal air samples were collected at 4 L/min using Personal Button Samplers (SKC, Inc.), AirChek pumps (SKC, Inc.), and gelatin filters (*D* = 25 mm)	14 out of 24 high-volume air samples were positive for SARS-CoV-2. All personal air samples were positive for SARS-CoV-2	Santarpia et al. (2020)
	06 air samples were collected using a FSC-1V air sampler (Hongrui, Suzhou, China) with filter membranes (0.22-μm pore size) for 15 min at 100 L/min, placed 1 m from the floor and 0.6-m away from patients	All samples were negative for SARS-CoV-2	Wei et al. (2020)
	04 air samples from ship cabins were collected using a Sartorius Airport MD8 air sampler at 50 L/min for 20 min and a Sartorius gelatin filter type 175 (area = 38.5 cm^2)	One air sample was positive for SARS-CoV-2	Yamagishi (2020)
	Air samples were collected in the East–West Lake Fangcang Shelter Hospital, using an air virus collection equipment (NingBo iGene TecTM) with a 0.1 μm gelatin membrane filter for 10 min at 6 m^3/h. A total of 48 air samples were collected	All samples were negative for SARS-CoV-2	Zhang et al. (2020)
Other/various sampling methods	Four bioaerosol samplers were used: an Andersen one-stage viable impactor (QuickTake-30, SKC, USA), an AirPort MD8 with gel film (Sartorius, Germany), an ASE-100 (Langsi Medical Technology, Shenzhen, China) in liquid medium, and a WA-15 (Dinglan Technology, Beijing, China)	45 out of 46 samples were negative for SARS-CoV-2	Ding et al. (2020)
	28 air samples were collected by natural sedimentation and using a microbial air sampler (MAS-100 ECO) at 100 L/min	One air sample was positive for SARS-CoV-2	Jiang et al. (2020)
	Three serial 3-h samplings using a prototype VIVAS air sampler and a BioSpot-VIVAS BSS300P. The airborne particles were collected using a water-vapor condensation method	Viable virus was isolated from air samples collected from 2 to 4.8 m away from the patients	Lednicky et al. (2020b)
	Air samples were collected using a two-stage cyclonic bioaerosol sampler (NIOSH) for 4 h at a flow rate of 3.5 L/min, into three size fractions: > 4 μm (15-mL tube), 1–4 μm (1.5–mL tube) and < 1 μm (Teflon® membrane filter with a pore size of 3.0 μm), and a cyclonic aerosol particle liquid concentrator (model W-15, Beijing Dingblue Technology Co., Ltd.) for 30 min at a flow rate of 14 L/min	02 air samples were positive for SARS-CoV-2	Lei et al. (2020)
	Aerosol samples were collected consisting of 03 different types: total suspended particles, segregated aerosol, and deposition. Total suspended particles were collected on 25 mm styrene filter cassettes (SKC Inc., USA) at 5 L/min using a portable pump (APEX2, Casella, USA). Segregated aerosol samples were collected using a cascade impactor (Sioutas impactor, SKC Inc., USA) at 9 L/min. Aerosol deposition samples were collected using filters (*D* = 80 mm) placed on the floor and intact for 7 days	19 out of 35 samples were positive for SARS-CoV-2, being 113 copies/m^3 the highest RNA concentration	Liu et al. (2020)
	Air samples were collected into conical tubes containing 5 mL of Dulbecco's minimum essential medium (DMEM) using a Coriolis μ air sampler (BERTIN INSTRUMENTS 2020)	14 out of 31 air samples were positive for SARS-CoV-2 RNA	Zhou et al. (2020)

Table 1 Sample collection methods and results in investigations for the presence of viruses focusing on SARS-CoV-2 in air samples analyzed by reverse transcriptase polymerase chain reaction (RT-PCR).

Fig. 2



Summary of air sampling mechanisms for SARS-CoV-2 detection and percentage of studies with positive samples

Fig. 2 Summary of air sampling mechanisms for SARS-CoV-2 detection and percentage of studies with positive samples

CARDIOLOGY

RESEARCHERS INVESTIGATE WHAT COVID-19 DOES TO THE HEART

Abbasi J.. JAMA. 2021 Feb 10. doi: 10.1001/jama.2021.0107. Online ahead of print.
Level of Evidence: 5 - Opinion

BLUF

This health journalism piece discusses current evidence regarding COVID-19 and cardiac injury. Topics include evidence of cardiac damage in young, mildly symptomatic patients, cardiac MRI findings (Figure 1), use of troponin to quantify myocyte damage, and the debate regarding the pathophysiology of cardiac damage. The author ends with a discussion on "long hauler" patients who continue to experience lingering symptoms of COVID-19 weeks after diagnosis and emphasizes the need to better understand the etiology of SARS-CoV-2 damage to cardiac tissue.

SUMMARY

The following are key clinical finings summarized in the article:

-Controversy still surrounds the etiology of cardiac disease after resolution of COVID-19. Researchers are still investigating if a post-viral myocarditis or other etiologies such as myopericarditis are responsible.
-A timeline of cardiac damage after COVID-19 infection is still being developed as more data is collected regarding clinical course, specifically with younger populations.
-Cardiac Resonance Imaging (CRI) is emerging as a useful modality to investigate cardiac inflammation post COVID-19 infection.
-Post-COVID-19 myocarditis in athletes has been recently highlighted as celebrity athletes, such as Boston Red Sox pitcher Eduardo Rodriguez, have been found to have residual heart strain and evidence of inflammation on CRI.
-Debate still surrounds the validity of studies using autospy results of post-COVID-19 cardiac tissue in light of small study sizes and possible confounding biases.
-New-onset chronic heart failure is a growing concern is patients with long term symptoms after recovery from COVID-19, termed "long haulers".
-Long term studies are being conducted to study cardiac inflammation and myocyte dysfunction.

FIGURES

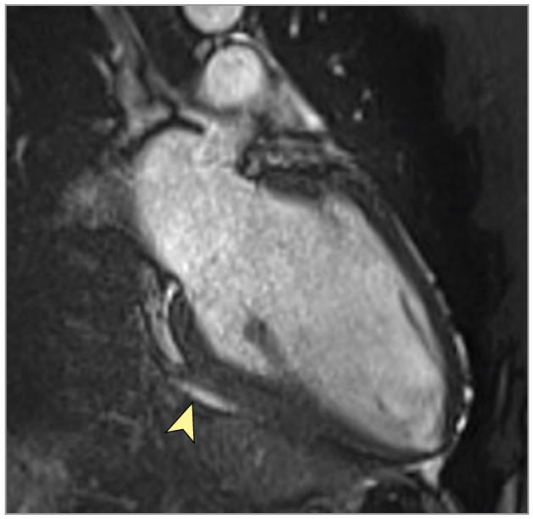


Figure 1. Cardiac magnetic resonance imaging shows pericardial effusion (indicated by arrow) in an Ohio State University competitive athlete recovering from coronavirus disease 2019. JAMA Cardiology. 2021;6(1):116-118.

ACKNOWLEDGEMENTS

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