DECLARATION OF PETER A. MCCULLOUGH, MD, MPH

Pursuant to 28 U.S.C. §1746, I, Peter A. McCullough, MD, MPH, declare under the penalty of perjury of the laws of the United States of America, and state upon personal knowledge that:

1. I am an adult of sound mind, 58 years old, and make this statement voluntarily, based upon my own personal knowledge, education, and experience, and under the penalty of perjury of the laws of the United States of America.

Experience & Credentials

2. I am competent to testify to the facts and matters set forth herein. A true and accurate copy of my *curriculum vitae* is attached hereto as **Exhibit A**.

3. After receiving a bachelor's degree from Baylor University, I completed my medical degree as an Alpha Omega Alpha graduate from the University of Texas Southwestern Medical School in Dallas. I went on to complete my internal medicine residency at the University of Washington in Seattle, a cardiology fellowship including service as Chief Fellow at William Beaumont Hospital, and a master's degree in public health at the University of Michigan.

4. I am board certified in internal medicine and cardiovascular disease and hold an additional certification in clinical lipidology, and previously echocardiography. I am on the medical staff at Baylor University Medical Center and Baylor Jack and Jane Hamilton Heart and Vascular Hospital, in Dallas, Texas. I am also on staff at Baylor Heart and Vascular Institute, which promotes cardiovascular research and education. I practice internal medicine and clinical cardiology as well as teach, conduct research, and I am an active scholar in medicine with roles as an author, editorialist, and reviewer at dozens of major medical journals and textbooks. I am Professor of Medicine at Texas A & M University School of Medicine, Baylor Dallas Campus.

5. I have led clinical, education, research, and program operations at major academic centers (Henry Ford Hospital, Oakland University William Beaumont School of Medicine) as well as academically oriented community health systems. I spearheaded the clinical development of *in vitro* natriuretic peptide and neutrophil gelatinase associated lipocalin assays in diagnosis, prognosis, and management of heart and kidney disease now used worldwide. I also led the first clinical study demonstrating the relationship between severity of acute kidney injury and mortality after myocardial infarction. I have contributed to the understanding of the epidemiology of chronic heart and kidney disease through many manuscripts from the Kidney Early Evaluation Program Annual Data Report published in the American Journal of Kidney Disease and participated in clinical trial design and execution in cardiorenal applications of acute kidney injury, hypertension, acute coronary syndromes, heart failure, and chronic cardiorenal syndromes. I participated in event adjudication (involved attribution of cause of death) in trials of acute coronary syndromes, chronic kidney disease, heart failure, and data safety and monitoring of anti-diabetic agents, renal therapeutics, hematology products, and gastrointestinal treatments. I have served as the chairman or as a member of over 20 randomized trials of drugs,

devices, and clinical strategies. Sponsors have included pharmaceutical manufacturers, biotechnology companies, and the National Institutes of Health.

6. I frequently lecture and advise on internal medicine, nephrology, and cardiology to leading institutions worldwide. I am recognized by my peers for my work on the role of chronic kidney disease as a cardiovascular risk state. I have over 1,000 related scientific publications, including the "Interface between Renal Disease and Cardiovascular Illness" in Braunwald's Heart Disease Textbook. My works have appeared in the New England Journal of Medicine, Journal of the American Medical Association, and other top-tier journals worldwide. I am an associate editor of the American Journal of Cardiology and the American Journal of Kidney Diseases. I have testified before the U.S. Senate Committee on Homeland Security and Governmental Affairs, the U.S. Food and Drug Administration Cardiorenal Advisory Panel and its U.S. Congressional Oversight Committee, and the Texas Senate Committee on Health and Human Services.

7. I am a Fellow of the American College of Cardiology, the American Heart Association, the American College of Physicians, the American College of Chest Physicians, the National Lipid Association, and the National Kidney Foundation. I am also a Diplomate of the American Board of Clinical Lipidology.

8. In 2013, I was honored with the International Vicenza Award for Critical Care Nephrology for my contribution and dedication to the emerging problem of cardiorenal syndromes. I am the President of the Cardiorenal Society of America, an organization dedicated to bringing together cardiologists and nephrologists and engage in research, improved quality of care, and community outreach to patients with both heart and kidney disease.[1]

9. I am the current President of the Cardiorenal Society of America, a professional organization dedicated to advancing research and clinical care for patients who have combined heart and kidney disease. I am the Editor-in-Chief of *Cardiorenal Medicine*, a primary research journal listed by the National Library of Medicine which is the only publication with a primary focus on research concerning patients with combined heart and kidney disease. Finally, I am the Editor-in-Chief of *Reviews in Cardiovascular Medicine*, a widely read journal that publishes reviews on contemporary topics in cardiology and is also listed by the National Library of Medicine.

10. My appended *curriculum vitae* further demonstrates my academic and scientific achievements and provides a list of publications authored by me in the past 30 years.

11. Since the outset of the pandemic, I have been a leader in the medical response to the COVID-19 disaster and have published "Pathophysiological Basis and Rationale for Early Outpatient Treatment of SARS-CoV-2 (COVID-19) Infection," the first synthesis of sequenced multidrug treatment of ambulatory patients infected with SARS-CoV-2 in the *American Journal of Medicine* and updated in *Reviews in Cardiovascular Medicine*.[2] I have 40 peer-reviewed

[1] *See* http://www.cardiorenalsociety.org/.

[2] McCullough PA, Kelly RJ, Ruocco G, Lerma E, Tumlin J, Wheelan KR, Katz N, Lepor NE, Vijay K, Carter H, Singh B, McCullough SP, Bhambi BK, Palazzuoli A, De Ferrari GM, Milligan GP, Safder T, Tecson KM, Wang

publications on the COVID-19 infection cited in the National Library of Medicine. Through a window to public policymakers, I have contributed extensively on issues surrounding the COVID-19 crisis in a series of OPED's for *The Hill*. I testified on the SARS-CoV-2 outbreak in the U.S. Senate Committee on Homeland Security and Governmental Affairs on November 19, 2020. I testified on lessons learned from the pandemic response in the Texas Senate Committee on Health and Human Services on March 10, 2021, and on early treatment of COVID-19 the Colorado General Assembly on March 31, 2021. Additionally, I testified in the New Hampshire Senate on legislation concerning the investigational COVID-19 vaccine on April 14, 2020. My expertise on the SARS-CoV-2 infection and COVID-19 syndrome, like that of infectious disease specialists, is approximately 18 months old. I have formed my opinions based on my direct clinical experience with acute and convalescent COVID-19 cases as well has closely following the preprint and published literature on the outbreak. I have specifically reviewed all the key published rare cases and reports concerning possible recurrence of SARS-CoV-2.

Opinion

12. It is my opinion that SARS-CoV-2 causes an infection in humans that results in robust, complete, and durable immunity. It is also my opinion that that natural immunity is superior to vaccination-induced immunity for the reasons explained below, including because the CDC has recorded >10,000 breakthrough cases in fully vaccinated individuals. I have reviewed the available preprint and published medical literature on the topic and have formed my opinions based on these reports.

13. SARS-CoV-2 is at least 80% homologous to SARS-CoV-1 at the epitopes that would be recognized by host defenses that confer immunity.[3] The major antigen in SARS-CoV-2 is the nucleocapsid and this has >90% homology to SARS-CoV-1. The immunity to SARS-CoV-1 has been lifelong over the observation period thus far in humans which is 17 years reflecting the duration of immunity that is likely from SARS-CoV-2.[4]

DD, McKinnon JE, O'Neill WW, Zervos M, Risch HA. Pathophysiological Basis and Rationale for Early Outpatient Treatment of SARS-CoV-2 (COVID-19) Infection. Am J Med. 2021 Jan;134(1):16-22. doi: 10.1016/j.amjmed.2020.07.003. Epub 2020 Aug 7. PMID: 32771461; PMCID: PMC7410805 available at https://pubmed.ncbi.nlm.nih.gov/32771461/; McCullough PA, Alexander PE, Armstrong R, Arvinte C, Bain AF, Bartlett RP, Berkowitz RL, Berry AC, Borody TJ, Brewer JH, Brufsky AM, Clarke T, Derwand R, Eck A, Eck J, Eisner RA, Fareed GC, Farella A, Fonseca SNS, Geyer CE Jr, Gonnering RS, Graves KE, Gross KBV, Hazan S, Held KS, Hight HT, Immanuel S, Jacobs MM, Ladapo JA, Lee LH, Littell J, Lozano I, Mangat HS, Marble B, McKinnon JE, Merritt LD, Orient JM, Oskoui R, Pompan DC, Procter BC, Prodromos C, Rajter JC, Rajter JJ, Ram CVS, Rios SS, Risch HA, Robb MJA, Rutherford M, Scholz M, Singleton MM, Tumlin JA, Tyson BM, Urso RG, Victory K, Vliet EL, Wax CM, Wolkoff AG, Wooll V, Zelenko V. Multifaceted highly targeted sequential multidrug treatment of early ambulatory high-risk SARS-CoV-2 infection (COVID-19). Rev Cardiovasc Med. 2020 Dec 30;21(4):517-530. doi: 10.31083/j.rcm.2020.04.264. PMID: 33387997 available at https://pubmed.ncbi.nlm.nih.gov/33387997/.

[3] *See* Xu J, Zhao S, Teng T, et al. Systematic Comparison of Two Animal-to-Human Transmitted Human Coronaviruses: SARS-CoV-2 and SARS-CoV. Viruses. 2020;12(2):244. Published 2020 Feb 22. doi:10.3390/v12020244 available at https://pubmed.ncbi.nlm.nih.gov/32098422/.

[4] *See* Le Bert N, Tan AT, Kunasegaran K, Tham CYL, Hafezi M, Chia A, Chng MHY, Lin M, Tan N, Linster M, Chia WN, Chen MI, Wang LF, Ooi EE, Kalimuddin S, Tambyah PA, Low JG, Tan YJ, Bertoletti A. SARS-CoV-2-specific T cell immunity in cases of COVID-19 and SARS, and uninfected controls. Nature. 2020 Aug;584(7821):457-462. doi: 10.1038/s41586-020-2550-z. Epub 2020 Jul 15. PMID: 32668444 available at

14. Natural immunity that develops after infection with SARS-CoV-2 is conferred by antibodies to the nucleocapsid and to the spike protein, as well as T-helper cells, natural killer cells, B-cells, and innate immunity.[5] This robust host defense system is far more extensive than the limited library of antibodies to the spike protein that are generated in response to the currently available COVID-19 investigational vaccines which have demonstrated immunity lasting only a few months at this time. This results in more protective immunity for those who have had a natural infection as compared to those who have been vaccinated.

15. After the natural SARS-CoV-2 infection, even in cases where antibody responses have not meet the threshold for being "reactive" in the ~100 commercial assays, there is scientific evidence that cellular based immunity is present.[6] Thus, there is ample evidence to suggest the clinical infection alone, without either antibody or cellular based testing afterwards, is sufficient to identify an individual who is no longer susceptible to COVID-19.[7] Specifically, in such an individual, there is no evidence that SARS-CoV-2 can be acquired, carried, or transmitted to another individual.

16. There are no published, credible reports of reinfection with SARS-CoV-2 in humans. In the case published by Zucman and colleagues, a patient is described as having a positive nasal PCR test for SARS-CoV-2 but no symptoms and then months later having COVID-19 syndrome requiring hospitalization. It is my interpretation that rare cases such as this, in the absence of antigen and whole genome sequencing, represent a false positive nasal PCR test on one occasion and a single COVID-19 syndrome on a separate occasion.[8] Similarly,

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https://pubmed.ncbi.nlm.nih.gov/32668444/; Zuo J, Dowell AC, Pearce H, Verma K, Long HM, Begum J, Aiano F, Amin-Chowdhury Z, Hallis B, Stapley L, Borrow R, Linley E, Ahmad S, Parker B, Horsley A, Amirthalingam G, Brown K, Ramsay ME, Ladhani S, Moss P. Robust SARS-CoV-2-specific T cell immunity is maintained at 6 months following primary infection. Nat Immunol. 2021 May;22(5):620-626. doi: 10.1038/s41590-021-00902-8. Epub 2021 Mar 5. PMID: 33674800; PMCID: PMC7610739 available at https://www.nature.com/articles/s41590-021-00902-8.

[5] *See* Zuo J, Dowell AC, Pearce H, Verma K, Long HM, Begum J, Aiano F, Amin-Chowdhury Z, Hallis B, Stapley L, Borrow R, Linley E, Ahmad S, Parker B, Horsley A, Amirthalingam G, Brown K, Ramsay ME, Ladhani S, Moss P. Robust SARS-CoV-2-specific T cell immunity is maintained at 6 months following primary infection. Nat Immunol. 2021 May;22(5):620-626. doi: 10.1038/s41590-021-00902-8. Epub 2021 Mar 5. PMID: 33674800; PMCID: PMC7610739 available at https://www.nature.com/articles/s41590-021-00902-8.

[6] *See* Schwarzkopf S, Krawczyk A, Knop D, Klump H, Heinold A, Heinemann FM, Thümmler L, Temme C, Breyer M, Witzke O, Dittmer U, Lenz V, Horn PA, Lindemann M. Cellular Immunity in COVID-19 Convalescents with PCR-Confirmed Infection but with Undetectable SARS-CoV-2-Specific IgG. Emerg Infect Dis. 2021 Jan;27(1). doi: 10.3201/2701.203772. Epub 2020 Oct 15. PMID: 33058753 available at https://pubmed.ncbi.nlm.nih.gov/33058753/.

[7] *See* Schulien I, Kemming J, Oberhardt V, Wild K, Seidel LM, Killmer S, Sagar, Daul F, Salvat Lago M, Decker A, Luxenburger H, Binder B, Bettinger D, Sogukpinar O, Rieg S, Panning M, Huzly D, Schwemmle M, Kochs G, Waller CF, Nieters A, Duerschmied D, Emmerich F, Mei HE, Schulz AR, Llewellyn-Lacey S, Price DA, Boettler T, Bengsch B, Thimme R, Hofmann M, Neumann-Haefelin C. Characterization of pre-existing and induced SARS-CoV-2-specific CD8+ T cells. Nat Med. 2021 Jan;27(1):78-85. doi: 10.1038/s41591-020-01143-2. Epub 2020 Nov 12. PMID: 33184509 available at https://pubmed.ncbi.nlm.nih.gov/33184509/.

[8] *See* Zucman N, Uhel F, Descamps D, Roux D, Ricard JD. Severe reinfection with South African SARS-CoV-2 variant 501Y.V2: A case report. Clin Infect Dis. 2021 Feb 10:ciab129. doi: 10.1093/cid/ciab129. Epub ahead of

Severaj et al. have reported 1 case of their own and 34 others in the literature with a similar profile with misinterpretation of a false positive PCR leading doctors to believe a second infection was possible.[9] None of these cases of "reinfection" was confirmed by antigen and whole genome sequencing to confirm the actual presence of the SARS-CoV-2 in the setting of a clinical infection on two or more occasions in the same patient.

17. At the current number of estimate cases in the world being 164 million, if reinfection was possible in 1% of individuals, the world would have observed 1.6 million second and third cases with many requiring hospitalization and coming to clinical attention.[10] In fact, no such large volume of recurrent cases has come to clinical attention in any region of the world.

18. Raw et al. reported that in 974 individuals who received the BNT162b2/Pfizer vaccine, those with a prior history of SARS-CoV-2 or those who had positive antibodies at baseline, had a higher rate of vaccine reactions than those who were COVID-19 naive.[11]

19. Mathioudakis et al. reported that in 2002 patients who underwent vaccination with either mRNA-based, or vector-based COVID-19 vaccines, COVID-recovered patients who were needlessly vaccinated had higher rates of vaccine reactions.[12]

20. Krammer et al. reported on 231 volunteers for COVID-19 vaccination, 83 of whom had positive SARS-CoV-2 antibodies at the time of immunization. The authors found: "Vaccine recipients with preexisting immunity experience systemic side effects with a significantly higher frequency than antibody naïve vaccines (e.g., fatigue, headache, chills, fever, muscle or join pains, in order of decreasing frequency, $P < 0.001$ for all listed symptoms, Fisher's exact test, two-sided)." (https://doi.org/10.1101/2021.01.29.21250653).

21. To my knowledge, there are no studies demonstrating clinical benefit of COVID-19 vaccination in COVID-19 survivors or those who have laboratory evidence of prior infection.

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print. PMID: 33566076; PMCID: PMC7929064 available at https://academic.oup.com/cid/advance-article/doi/10.1093/cid/ciab129/6132402.

[9] *See* Selvaraj V, Herman K, Dapaah-Afriyie K. Severe, Symptomatic Reinfection in a Patient with COVID-19. R I Med J (2013). 2020 Nov 9;103(10):24-26. PMID: 33172223 available at https://pubmed.ncbi.nlm.nih.gov/33172223/.

[10] *See* https://www.worldometers.info/coronavirus/.

[11] *See* https://www.medrxiv.org/content/10.1101/2021.04.15.21252192v1.

[12] *See* https://www.medrxiv.org/content/10.1101/2021.02.26.21252096v1.

22. In sum, it is my opinion that SARS-CoV-2 causes an infection in humans that results in robust, complete, and durable immunity, and is superior to vaccine immunity. There are no studies demonstrating clinical benefit of COVID-19 vaccination in COVID-19 survivors and there are three studies demonstrating harm in such individuals. Thus, it is my opinion that the COVID-19 vaccination is contraindicated in COVID-19 survivors.

I DECLARE UNDER THE PENALTY OF PERJURY UNDER THE LAWS OF THE UNITED STATES OF AMERICA THAT THE FOREGOING INFORMATION CONTAINED IN THIS DECLARATION IS TRUE AND CORRECT.

May 28, 2021

28-MAY-2021

Peter A. McCullough, MD, MPH