Exhibit 2

DECLARATION OF DR. JONATHAN LOUIS GOLOB

I, Jonathan Louis Golob, declare as follows:

1. I am an Assistant Professor at the University of Michigan School of Medicine in Ann Arbor, Michigan, where I am a specialist in infectious diseases and internal medicine. At the University of Michigan School of Medicine, I am a practicing physician and a laboratory-based scientist. My primary subspecialization is for infections in immunocompromised patients, and my recent scientific publications focus on how microbes affect immunocompromised people. I obtained my medical degree and completed my residency at the University of Washington School of Medicine in Seattle, Washington, and also completed a Fellowship in Internal Medicine Infectious Disease at the University of Washington. I am actively involved in the planning and care for patients with COVID-19. Attached as Exhibit A is a copy of my curriculum vitae.

2. COVID-19 is an infection caused by a novel zoonotic coronavirus SARS-COV-2 that has been identified as the cause of a viral outbreak that originated in Wuhan, China in December 2019. The World Health Organization has declared that COVID-19 is causing a pandemic. As of March 21, 2020, there are over 260,000 confirmed cases of COVID-19 worldwide. COVID-19 has caused over 11,000 deaths, with exponentially growing outbreaks occurring at multiple sites worldwide, including within the United States.

3. COVID-19 makes certain populations of people severely ill. People over the age of fifty are at higher risk, with those over 70 at serious risk. As the Center for Disease Control and Prevention has advised, certain medical conditions increase the risk of serious COVID-19 for people of any age. These medical conditions include: those with lung disease, heart disease, diabetes, or immunocompromised (such as from cancer, HIV, autoimmune diseases), blood disorders (including sickle cell disease), chronic liver or kidney disease, inherited metabolic disorders, stroke, developmental delay, or pregnancy.

4. For all people, even in advanced countries with very effective health care systems such as the Republic of Korea, the case fatality rate of this infection is about ten fold higher than that observed from a severe seasonal influenza. In the more vulnerable groups, both the need for care, including intensive care, and death is much higher than we observe from influenza infection: In the highest risk populations, the case fatality rate is about 15%. For high risk patients who do not die from COVID-19, a prolonged recovery is expected to be required, including the need for extensive rehabilitation for profound deconditioning, loss of digits, neurologic damage, and loss of respiratory capacity that can be expected from such a severe illness.

5. In most people, the virus causes fever, cough, and shortness of breath. In high-risk individuals as noted above, this shortness of breath can often be severe. Even in younger and healthier people, infection of this virus requires supportive care, which includes supplemental oxygen, positive pressure ventilation, and in extreme cases, extracorporeal mechanical oxygenation.

6. The incubation period (between infection and the development of symptoms) for COVID-19 is typically 5 days, but can vary from as short as two days to an infected individual never developing symptoms. There is evidence that transmission can occur before the development of infection and from infected individuals who never develop symptoms. Thus, only with aggressive testing for SARS-COV-2 can a lack of positive tests establish a lack of risk for COVID-19.

7. A lack of proven cases of COVID-19 in the context of a lack of testing is functionally meaningless for determining if there is a risk for COVID-19 transmission in a community or institution.

8. Most people in the higher risk categories will require more advanced support: positive pressure ventilation, and in extreme cases, extracorporeal mechanical oxygenation. Such care requires highly specialized equipment in limited supply as well as an entire team of care providers, including but not limited to 1:1 or 1:2 nurse to patient ratios, respiratory therapists and intensive care physicians. This level of support can quickly exceed local health care resources.

9. COVID-19 can severely damage the lung tissue, requiring an extensive period of rehabilitation and in some cases a permanent loss of respiratory capacity. The virus also seems to target the heart muscle itself, causing a medical condition called mycocarditis, or inflammation of the heart muscle. Myocarditis can affect the heart muscle and electrical system, which reduces the heart's ability to pump, leading to rapid or abnormal heart rhythms in the short term, and heart failure that limits exercise tolerance and the ability to work lifelong. There is emerging evidence that the virus can trigger an over-response by the immune system in infected people, further damaging tissues. This cytokine release syndrome can result in widespread damage to other organs, including permanent injury to the kidneys (leading to dialysis dependence) and neurologic injury.

10. There is no vaccine for this infection. Unlike influenza, there is no known effective antiviral medication to prevent or treat infection from COVID-19. Experimental therapies are being attempted. The only known effective measures to reduce the risk for a

vulnerable person from injury or death from COVID-19 are to prevent individuals from being infected with the COVID-19 virus. Social distancing, or remaining physically separated from known or potentially infected individuals, and hygiene, including washing with soap and water, are the only known effective measures for protecting vulnerable communities from COVID-19.

11. Nationally, without effective public health interventions, CDC projections indicate about 200 million people in the United States could be infected over the course of the epidemic, with as many as 1.5 million deaths in the most severe projections. Effective public health measures, including social distancing and hygiene for vulnerable populations, could reduce these numbers.

12. COVID-19 strains have specifically traced infection between residents and staff members of a skilled nursing facility in the Seattle area. This evidence suggests that COVID-19 is capable of spreading rapidly in institutionalized settings. The highest known person-to-person transmission rates for COVID-19 are in a skilled nursing facility in Kirkland, Washington and on afflicted cruise ships in Japan and off the coast of California.

13. During the H1N1 influenza ("Swine Flu") epidemic in 2009, jails and prisons were sites of severe outbreaks of viral infection. Given the avid spread of COVID-19 in skilled nursing facilities and cruise ships, it is reasonable to expect COVID-19 will also readily spread in detention centers, particularly when residents cannot engage in social distancing measures, cannot practice proper hygiene, and cannot isolate themselves from infected residents or staff. With new individuals and staff coming into the detention centers who may be asymptomatic or not yet presenting symptoms, the risk of infection rises even with symptom screening measures.

14. This information provides many reasons to conclude that vulnerable people, people over the age of 50 and people of any age with lung disease, heart disease, diabetes, or immunocompromised (such as from cancer, HIV, autoimmune diseases), blood disorders (including sickle cell disease), chronic liver or kidney disease, inherited metabolic disorders, stroke, developmental delay, or pregnancy living in an institutional setting, such as an immigration detention center, prison, or jail, with limited access to adequate hygiene facilities, limited ability to physically distance themselves from others, and exposure to potentially infected individuals from the community are at grave risk of severe illness and death from COVID-19.

Pursuant to 28 U.S.C. 1746, I declare under penalty of perjury that the foregoing is true and correct.

Executed this 23 day in March, 2020 in Ann Arbor, Michigan.

Dr. Jonathan Louis Golob

Jonathan Louis Golob, M.D. Ph.D.
Assistant Professor
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Education and Training

6/1997 – 6/2001	**Bachelor of Science**, Johns Hopkins University, Baltimore, MD Dual degree in Biomedical Engineering and Computer Science conferred June 2001.
7/2001 – 6/2011	**MSTP MD/PhD Combined Degree**, University of Washington, Seattle, WA. Ph.D. on the basic science of embryonic stem cells, specifically epigenetic regulation of differentiation Ph.D. conferred in June 2009. MD conferred in June 2011.
6/2011 – 6/2013	**Internal Medicine Residency**, University of Washington, Seattle, WA
6/2013 – 6/2017	**Infectious Diseases Fellowship**, University of Washington, Seattle, WA

Certifications and Licensure

Board Certifications

2014	Diplomate in Internal Medicine**,** American Board of Internal Medicine.
2016	Diplomate in Infectious Disease**,** American Board of Internal Medicine.

Current Medical Licenses to Practice

2013	Washington State Medical License, Physician, MD60394350
2018	Michigan State Medical License, Physician, 4301114297

Academic, Administrative, and Clinical Appointments

Academic

6/2014 – 6/2018	**Senior Fellow, Vaccine and Infectious Disease Division**, Fred Hutchinson Cancer Research Center, Seattle, WA
8/2016 – 6/2018	**Joel Meyers Endowment Fellow**, Vaccine and Infectious Disease Division, Fred Hutchinson Cancer Research Center, Seattle, WA
8/2017 – 6/2018	**Research Associate, Vaccine and Infectious Disease Division**, Fred Hutchinson Cancer Research Center, Seattle, WA
8/2017 – 6/2018	**Acting Instructor**, Division of Allergy and Infectious Diseases, Department of Medicine, University of Washington, Seattle, WA
8/2018 – Present	**Assistant Professor, Division of Infectious Diseases,** Department of Medicine, University of Michigan, Ann Arbor, MI

Clinical

12/2015 – 12/2016	**Infectious Disease Locums Physician**, Virginia Mason Medical Center, Seattle, WA
7/2017 – 6/2018	**Hospitalist Internal Medicine Physician**, Virginia Mason Medical Center, Seattle, WA
8/2017 – 6/2018	**Attending Physician**, Seattle Cancer Care Alliance, Seattle, WA
8/2017 – 6/2018	**Attending Physician**, Division of Allergy and Infectious Diseases, Department of Medicine, University of Washington, Seattle, WA
8/2018 – Present	**Attending Physician,** Division of Infectious Diseases, Department of Medicine, University of Michigan, Ann Arbor, MI

Research Interests

1. I am primarily interested in understanding how the human gut microbiome *mechanistically* affects how patients respond to treatments. I have a particular focus on patients undergoing hematopoietic cell transplant, who are at risk for recurrence of their underlying disease, treatment-related colitis (from both conditioning and graft versus host disease), and infection. In human observational trials the human gut microbiome correlates with each of these aspects. My research program uses advanced stem-cell based *in-vitro* models of the human colonic mucosa to verify if the correlations in observational trials can cause similar effects *in vitro*, and then determine by which pathways (e.g. receptors) and broad mechanisms (e.g. epigenetics) the microbes affect the host.

2. Host-microbiome interactions are contextual. A beneficial interaction in health can turn pathologic. For example, my ongoing work focused on the microbial metabolite butyrate. Butyrate enhances the health of heathy and intact colonic epithelium, acting as a substrate for cellular respiration and through receptor-mediate processes reduces cellular inflammation. However, butyrate also blocks the ability of colonic stem cells to differentiate into mature epithelium. Thus, in colitis that results in a loss of colonic crypts, an intact and butyrogenic gut microbiome results in colonic stem cells being exposed to butyrate and inhibits recovery. My ongoing work uses a primary stem-cell based model of the human colonic mucusa to establish how butyrate blocks the differentiation of colonic stem cell with a hope of generating new treatments for patients with steroid-refractory colitis.

3. I am interested in validating and improving computational tools for biological research. I have a computer science and biomedical engineering background that combined with my clinical and molecular biology training positions me optimally to understand both major aspects of computational biology: what are the needs to make biological inferences from big data, and how can tools specifically be improved to achieve such inferences.

Grants

Present and Active

ASBMT New Investigator Award J. Golob (PI) 7/2018 – 7/2020
Hematopoietic Cell Transplant Outcomes and Microbial Metabolism
Role: PI
$30,000/yr for up to two years

NIH / NIAID R01 D. Fredricks (PI) 11/2017 – 11/2021
The Gut Microbiota and Graft versus Host Disease (GVHD), AI-134808
Role: Senior / key personnel
$823,701

NIH P01 T. Schmidt (PI) Pending / Reviewed
ENGINEERING MICROBIOMES AND THEIR MOLECULAR DETERMINANTS FOR PRODUCTION OF BUTYRATE AND SECONDARY BILE ACIDS FROM RESISTANT STARCH
Role: Key Personnel

NIH / NCI R21 J. Golob (PI) Pending / Submitted
Establishing a physiologic human colonic stem/progenitor cells model of regimen-related colitis
Role: PI

NIH R21 J. Golob (PI) Pending / Submitted
Manipulating Butyrate Production by the Gut Microbiome during Chronic HIV Infection
Role: PI

Completed

Joel Meyers Endowment Fellowship 6/2016 – 6/2018
Role: Research Fellow
$63,180

DCDR Grant R. Harrington (PI) 6/2014 – 6/2018
Support for data queries into the Deidentified Clinical Data Repository
Role: PI
$1000

NIH T32 Institutional Training Grant M. Boeckh (PI) 8/2016 – 8/2017
1T32AI118690-01A1
Role: Post-Doc Trainee
$315,972

NIH T32 Institutional Training Grant W. van Voorhis (PI) 7/1/14 – 6/30/16
5T32AI007044
Role: Post-Doc Trainee
$1,527,801

Honors and Awards

2001 Tau Beta Pi Engineering Honor Society
2001 Alpha Eta Mu Beta Biomedical Engineering Honor Society

2005 ARCS Fellowship
2015 Consultant of the Month Award. University of Washington Housestaff.
2016 Joel Meyer Endowment Fellow

Membership in Professional Societies

2013 Member, Infectious Diseases Society of America
2011 Member, American Board of Internal Medicine

Bibliography

Peer-Reviewed Journals and Publications

1. Gao Z, **Golob J,** Tanavde VM, Civin CI, Hawley RG, Cheng L. High levels of transgene expression following transduction of long-term NOD/SCID-repopulating human cells with a modified lentiviral vector. *Stem Cells* 19(3): 247-59, 2001.
2. Cui Y, **Golob J**, Kelleher E, Ye Z, Pardoll D, Cheng L. Targeting transgene expression to antigen-presenting cells derived from lentivirus-transduced engrafting human hematopoietic stem/progenitor cells. *Blood* 99(2): 399-408, 2002.
3. Boursalian TE, **Golob J**, Soper DM, Cooper CJ, Fink PJ. Continued maturation of thymic emigrants in the periphery. *Nature Immunology* 5(4): 418-25, 2004.
4. Osugi T, Kohn AD, **Golob JL**, Pabon L, Reinecke H, Moon RT, Murry CE. Biphasic role for Wnt/beta-catenin signaling in cardiac specification in zebrafish and embryonic stem cells. *PNAS* 104(23): 9685-9690, 2007.
5. **Golob JL**, Paige SL, Muskheli V, Pabon L, Murry CE: Chromatin Remodeling During Mouse and Human Embryonic Stem Cell Differentiation. *Developmental Dynamics* 237(5): 1389-1398, 2008.
6. **Golob JL**, Kumar RM, Guenther MG, Laurent LC, Pabon LM, Loring JF, Young RA, Murry CE: Evidence That Gene Activation and Silencing during Stem Cell Differentiation Requires a Transcriptionally Paused Intermediate State. *PLoS ONE* 6(8): e22416, 2011.
7. **Golob JL**, Margolis E, Hoffman NG, Fredricks DN. Evaluating the accuracy of amplicon-based microbiome computational pipelines on simulated human gut microbial communities. *BMC Bioinformatics* 18(1):283, 2017.
8. MacAllister TJ, Stednick Z, **Golob JL**, Huang, ML, Pergam SA. Under-utilization of norovirus testing in hematopoietic cell transplant recipients at a large cancer center. *Am J Infect Control* pii: S0196-6553(17)30783-6. doi: 10.1016/j.ajic.2017.06.010. [Epub ahead of print], 2017.
9. **Golob JL**, Pergam SA, Srinivasan S, Fiedler TL, Liu C, Garcia K, Mielcarek M, Ko D, Aker S, Marquis S, Loeffelholz T, Plantinga A, Wu MC, Celustka K, Morrison A, Woodfield M, Fredricks DN. The Stool Microbiota at Neutrophil Recovery is Predictive for Severe Acute Graft versus Host Disease after Hematopoietic Cell Transplantation. *Clin Infect Dis* doi: 10.1093/cid/cix699. [Epub ahead of print], 2017.
10. Bhattacharyya A, Hanafi LA, Sheih A, **Golob JL**, Srinivasan S, Boeckh MJ, Pergam SA, Mahmood S, Baker KK, Gooley TA, Milano F, Fredricks DN, Riddell SR, Turtle CJ. Graft-Derived Reconstitution of Mucosal-Associated Invariant T Cells after Allogeneic Hematopoietic Cell Transplantation. *Biol Blood Marrow Transplant* pii: S1083-8791(17)30758-9. doi: 10.1016/j.bbmt.2017.10.003. Epub 2017 Oct 9.
11. Ogimi C, Krantz EM, **Golob JL**, Waghmare A, Liu C, Leisenring WM, Woodard CR, Marquis S, Kuypers JM, Jerome KR, Pergam SA, Fredricks DN, Sorror ML, Englund JA, Boeckh M. Antibiotic Exposure Prior to Respiratory Viral Infection Is Associated with Progression to Lower Respiratory Tract Disease in Allogeneic Hematopoietic Cell

Transplant Recipients. Biol Blood Marrow Transplant. 2018 May 16. pii: S1083-8791(18)30268-4. doi: 10.1016/j.bbmt.2018.05.016. [Epub ahead of print]

12. **Golob JL**, Stern J, Holte S, Kitahata MM, Crane HM, Coombs RW, Goecker E, Woolfrey AE, Harrington RD. HIV DNA levels and decay in a cohort of 111 long-term virally suppressed patients. AIDS. 2018 Sep 24;32(15):2113-2118. doi: 10.1097/QAD.0000000000001948.
13. **Golob JL**, DeMeules MM, Loeffelholz T, Quinn ZZ, Dame MK, Silvestri SS, Wu MC, Schmidt TM, Fiedler TL, Hoostal MJ, Mielcarek M, Spence J, Pergam SA, Fredricks DN. Butyrogenic bacteria after acute graft-versus-host disease (GVHD) are associated with the development of steroid-refractory GVHD. Blood Adv. 2019 Oct 8;3(19):2866–2869.

Preprint publications

1. **Golob JL** and Minot SS. Functional Analysis of Metagenomes by Likelihood Inference (FAMLI) Successfully Compensates for Multi-Mapping Short Reads from Metagenomic Samples. Preprint. doi: https://doi.org/10.1101/295352

Other Publications

1. Science Columnist and Writer for *The Stranger*, Seattle, WA, 2004 – Present
2. Freelance contributor, Ars Technica, 2016 – Present.

Abstracts (presenter underlined)

1. **Golob JL,** Srinivasan S, Pergam SA, Liu C, Ko D, Aker S, Fredricks DN. Gut Microbiome Changes in Response to Protocolized Antibiotic Administration During Hematopoietic Cell Transplantation. ID Week, Infectious Diseases Society of America, October 2015 (Oral)
2. **Golob JL**, Stern J, Holte S, Kitahata M, Crane H, Coombs R, Goecker E, Woolfrey AE, Harrington RD. HIV reservoir size and decay in 114 individuals with suppressed plasma virus for at least seven years: correlation with age and not ARV regimen. IDWeek 2016, October 26-30, 2016, New Orleans. Abstract 953 (Oral).
3. **Golob JL** , Stohs E, Sweet A, Pergam SA, Boeckh M, Fredricks DN, and Liu C. Vancomycin is Frequently Administered to Hematopoietic Cell Transplant Recipients Without a Provider Documented Indication and Correlates with Microbiome Disruption and Adverse Events. ID Week, Infectious Diseases Society of America, October 2018 (# 72504).
4. Impact of Intestinal Microbiota on Reconstitution of Mucosal-Associated Invariant T Cells after Allogeneic Hematopoietic Stem Cell Transplantation. ASH 2018 (#3393).

Invited Lectures

1. Keynote Speaker, ARCS Foundation Annual Dinner. Seattle, WA Nov 3, 2008
2. Primary Care Conference: Direct to Consumer Genetic Testing, Seattle, WA, Mar 14, 2013
3. "IRIS and TB", Harborview Medical Center Housestaff Lunchtime Conference, Seattle, WA, Jun 9, 2014
4. "Complicated Enterococcal Endocarditis", University of Washington Medical Center (UWMC) Chief of Medicine Conference, Seattle, WA, Jul 14, 2014
5. "Coccidiomycosis", UWMC Chief of Medicine Conference, Seattle, WA, Oct 7, 2014
6. "HIV and CMV encephalitis", UWMC Chief of Medicine Conference, Seattle, WA, Apr 14, 2015
7. Research Presentation for GVHD Group Meeting, Seattle, WA, Nov 2015

8. "CMV Ventriculitis", Clinical Case Presentation to the Virology Working Group, Fred Hutchinson Cancer Research Center (Fred Hutch), Seattle, WA, Nov 2015
9. "Microbiome and HCT Outcomes". 1st Infectious Disease in the Immunocompromised Host Symposium – Tribute to Joel Meyers. Fred Hutch, Seattle, WA, Jun 13 2016.
10. "Microbiome and GVHD". Infectious Disease Sciences / Virology Symposium, Fred Hutch / UW, Seattle, WA, Jan 17 2017
11. "Microbiome and GVHD". 2nd Symposium on Infectious Disease in the Immunocompromised Host. June 19 2017
12. "The Gut Microbiome Predicts GVHD. Can It Be Engineered to Protect?". St Jude. February 18th 2019