The Daily COVID-19 Literature Surveillance Summary

July 23, 2020



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COVID-19 Daily Literature Surveillance

COVID19LST

Bringing you real time, distilled information for guiding best practices during the COVID-19 pandemic

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 (b)(Lind Library), (b) Glasziou, Trish Greenhalgh, Carl Heneghan, Alessandro Liberati, Ivan Moschetti, Bob Phillips, Hazel Thornton, Olive Goddard and Mary Hodgkinson*

EXECUTIVE SUMMARY

CLIMATE:

- Authors from Saarland University in Germany propose a method for prioritizing allocation of a SARS-CoV-2 vaccine during a resource shortage. These recommendations include a three-tier preference list for vaccine distribution: beginning with hospital workers and public safety/security officers, followed by immunocompromised/suppressed individuals such as organ transplant recipients, and finally all others in the general population in order from oldest to youngest. This idea highlights one potential method of solving the key issue of vaccine prioritization for individuals with high susceptibility to COVID-19 infection.
- A sharp peak in rates of intestinal obstruction (IO) during the lockdown period in Kerela, India (referred to as "lockdown belly") leads authors to propose four common risk factors:
  - 60 years of age or older
  - prior abdominal surgery
  - strict adherence to lockdown restrictions
  - high fiber diet of jackfruit

  Additionally, the authors suggest at least 75% of IO cases during this time may have been prevented with modifiable risk factors including diet and physical activity.
- This article explores the impact of COVID-19 on immigrants in the United States, with an emphasis on those in Houston, Texas. The most prevalent comorbidities, nationally, include obesity (71.5%), hypertension (HTN, 27.7%), and diabetes (9.6%). Many immigrants are at increased risk for COVID-19 infection as they make up a significant portion of the essential workers work force (20% in Texas) and are more likely to live in large family groups. The unemployment rate increased from 3% to 15% from February to April 2020, and concerns over food insecurity, child support, and healthcare access continue to worsen. Thus, the authors suggest the following policy recommendations in order to better support these disadvantaged communities:
  - Expand Medicaid
  - Eliminate immigration requirements for CHIP
  - Pass registration banning ICE in healthcare
  - Fund COVID-19 testing and treatment for uninsured
  - Provide immigrants and families economic relief packages

UNDERSTANDING THE PATHOLOGY:

- A cross sectional study from Columbia University that analyzed kidney biopsies of SARS-CoV-2 infected patients (n=17), revealed a spectrum of glomerular and tubular injuries, acute kidney injury (88%), and lack of SARS-CoV-2 detection in kidney cells as evidence against direct viral injury as a pathophysiologic mechanism of disease. Authors conclude that the principle nephrogenicity of SARS-CoV-2 likely results from ischemic damage and a dysregulated immune response.
-

TRANSMISSION AND PREVENTION:

- A group of psychologists found that habit reversal training (HRT) was an effective method of treating repetitive behavior problems, such as face touching, and therefore may be an effective strategy for decreasing transmission of SARS-CoV-2. The principles of HRT are centered on mindfulness and behavior modification, and authors include a practical guide to implementing these practices.
-

MANAGEMENT:

- Italian authors describe how the anti-coagulant Defibrotide may be the "drug of choice" for treating the hypercoagulable state associated with severe COVID-19 disease because of its profibrinolytic, antithrombotic, and anti-inflammatory effects. These would theoretically protect from the growing evidence implicating the role of endothelial damage and a hyperinflammatory state in SARS-CoV-2 infection.
- A literature review of 9 studies describes the effect of T cell imbalance, specifically between Treg and Th17, on uncontrolled systemic inflammation in severe COVID-19 cases in pregnancy found that:
  - Treg cells decreased and Th17 cells increased, with a consequent decrease in the Treg/Th17 cell ratio.
  - Treg cells dysregulation in COVID-19 was shown trigger hyperinflammation and tissue damage.
  - Increased Th17 is associated with fetal allograft rejection at the feto-maternal interface.
  - COVID-19 infection puts pregnant persons at higher risk for pregnancy complications.

TABLE OF CONTENTS

DISCLAIMER **2**

NOW LIVE! 2

LEVEL OF EVIDENCE **3**

EXECUTIVE SUMMARY **4**

CLIMATE **6**

An epidemic of sub acute intestinal obstruction during Covid-19 pandemic related lockdown - 'the lockdown belly' 6

GLOBAL 6

Allocation criteria for an initial shortage of a future SARS-CoV-2 vaccine and necessary measures for global immunity 6

DISPARITIES 7

Racial/Ethnic Disparities in Disease Severity on Admission Chest Radiographs among Patients Admitted with Confirmed COVID-19: A Retrospective Cohort Study 7

Disproportionate impact of the COVID-19 pandemic on immigrant communities in the United States 8

COVID-19 and the Mental Health of People From Refugee Backgrounds 10

Health literacy and COVID-19 10

Covid-19 and Disparities in Nutrition and Obesity 10

EPIDEMIOLOGY **12**

SYMPTOMS AND CLINICAL PRESENTATION 12

Pregnant Persons *12*

Third Trimester Placentas of SARS-CoV-2-Positive Women: Histomorphology, including Viral Immunohistochemistry and in Situ Hybridization 12

UNDERSTANDING THE PATHOLOGY **14**

Kidney Biopsy Findings in Patients with COVID-19 14

Viruses and asthma: the role of common respiratory viruses in asthma and its potential meaning for SARS-CoV-2 16

IN SILICO 18

ACE2 gene variants may underlie interindividual variability and susceptibility to COVID-19 in the Italian population 18

TRANSMISSION & PREVENTION **20**

DEVELOPMENTS IN TRANSMISSION & PREVENTION 20

A Surface Coating that Rapidly Inactivates SARS-CoV-2 20

PREVENTION IN THE COMMUNITY 21

Reducing risky behavior with habit reversal: A review of behavioral strategies to reduce habitual hand-to-head behavior 21

PREVENTION IN THE HOSPITAL 19

SARS-Cov-2 in peritoneal fluid: an important finding in the Covid-19 pandemic 20

MANAGEMENT **22**

ACUTE CARE 22

Multifactorial pathogenesis of COVID-19-related coagulopathy. Can defibrotide have a role in the early phases of coagulation disorders? 22

Critical Care *23*

COVID-19: what type of cytokine storm are we dealing with? 23

OBGYN 24

COVID-19 and Treg/Th17 imbalance: Potential relationship to pregnancy outcomes 24

R&D: DIAGNOSIS & TREATMENTS **25**

DEVELOPMENTS IN DIAGNOSTICS 25

Lung Ultrasound for Treatment of Patients With COVID-19: Please Report Your Settings and Mechanical Index 25

Pan-Family Assays for Rapid Viral Screening: Reducing Delays in Public Health Responses During Pandemics 25

ACKNOWLEDGEMENTS **27**

CLIMATE

AN EPIDEMIC OF SUB ACUTE INTESTINAL OBSTRUCTION DURING COVID-19 PANDEMIC RELATED LOCKDOWN - 'THE LOCKDOWN BELLY'

Philip S, [illegible] A, Kumar KS, Renny RJ, Pillai V, Manda SR.. Br J Surg. 2020 Jul 20. doi: 10.1002/bjs.11803. Online ahead of print.
Level of Evidence: Other - Expert Opinion

BLUF

In this letter to the editor, the authors hypothesize the reasons for the sharp peak in intestinal obstruction (IO) during the lockdown period (referred to as "lockdown belly") of the COVID-19 pandemic in Kerala, India compared to two years prior (Figure 1). The authors propose four common factors of those presenting with IO during this time including:
- 60 years of age or older
- prior abdominal surgery
- strict adherence to lockdown restrictions
- high fiber diet of jackfruit

In addition to encouraging additional research to better understand the epidemic of "lockdown belly", the authors suggest at least 75% of IO cases during this time may have been prevented with modifiable risk factors including diet and physical activity.

FIGURES



Figure 1: Incidence rate of intestinal obstruction by year.

GLOBAL

ALLOCATION CRITERIA FOR AN INITIAL SHORTAGE OF A FUTURE SARS-COV-2 VACCINE AND NECESSARY MEASURES FOR GLOBAL IMMUNITY

Henn W.. Vaccine. 2020 Jul 22;38(34):5396-5397. doi: 10.1016/j.vaccine.2020.06.058. Epub 2020 Jun 23.
Level of Evidence: Other - Opinion

BLUF

An opinion piece from Saarland University in Germany discusses the idea of SARS-CoV-2 vaccine shortages and proposes a three-tier preference list for vaccine distribution: beginning with hospital workers and public safety/security officers, followed by immunocompromised/suppressed individuals such as organ transplant recipients, and finally all others in the general population in order from oldest to youngest. This idea highlights one potential method of solving the key issue of vaccine prioritization for individuals with high susceptibility to COVID-19 infection.

RACIAL/ETHNIC DISPARITIES IN DISEASE SEVERITY ON ADMISSION CHEST RADIOGRAPHS AMONG PATIENTS ADMITTED WITH CONFIRMED COVID-19: A RETROSPECTIVE COHORT STUDY

Joseph NP, Reid NJ, Som A, Li MD, Hyle EP, Dugdale CM, [redacted] M, Betancourt [redacted] Deng F, Mendoza DP, Little BP, Narayan AK, Flores EJ.. Radiology. 2020 Jul 16:202602. doi: 10.1148/radiol.2020202602. Online ahead of print.
Level of Evidence: 3 - Local non-random sample

BLUF

A retrospective cohort study from Boston, MA of 326 hospitalized COVID-19 patients from March 27th, 2020 to April 10th, 2020 found that Non-White patients had a higher modified Radiographic Assessment of Lung Edema (mRALE) score on admission chest x-ray compared to White/Non-Hispanic patients (p=0.005, Figure 4). Both had similar adverse outcomes (intubation, ICU admission or deaths) with increasing mRALE scores (p<0.001) with no evidence of interaction (p=0.16). These findings could potentially assist radiologists in identifying at-risk populations to improve outcomes for ethnic and racial minorities.

ABSTRACT

Background Disease severity on chest radiographs (CXR) has been associated with higher risk of disease progression and adverse outcomes from COVID-19. Few studies have evaluated COVID-19-related racial/ethnic disparities in radiology. Purpose To evaluate whether Non-White minority patients hospitalized with confirmed COVID-19 infection presented with increased severity on admission CXR compared with White/Non-Hispanic patients. Methods Single-institution, retrospective cohort study approved by the IRB. Patients hospitalized with confirmed COVID-19 infection (3/27/20-4/10/20) were identified using the electronic medical record (EMR) (n=326, mean age: 59 years (SD: 17 years), M:F (188:138). Primary outcome was severity of lung disease on admission CXR, measured by modified Radiographic Assessment of Lung Edema (mRALE) score. Secondary outcome was a composite adverse clinical outcome of intubation, ICU admission, or death. Primary exposure was racial/ethnic category: White/Non-Hispanic versus Non-White [i.e., Hispanic, Black, Asian, Other]. Multivariable linear regression analyses were performed to evaluate the association between mRALE scores and race/ethnicity. Results Non-White patients had significantly higher mRALE scores (median 6.1, 95% CI 5.4-6.7) compared with White/Non-Hispanic patients (median 4.2, 95% CI 3.6-4.9) (unadjusted average difference 1.8, 95% CI 0.9-2.8, p<0.01). For both White (Adjusted HR, 1.3, 95%CI 1.2-1.4, p<0.001) and Non-White patients (Adjusted HR 1.2, 95%CI 1.1-1.3, p<0.001), increasing mRALE scores were associated with a higher likelihood of experiencing composite adverse outcome with no evidence of interaction (p = 0.16). Multivariable linear regression analyses demonstrated that Non-White patients presented with higher mRALE scores on admission CXR versus White/Non-Hispanic patients (adjusted average difference 1.6, 95% CI 0.5-2.7, p<0.01). Adjustment for hypothesized mediators revealed that the association between race/ethnicity and mRALE scores was mediated by limited English proficiency (p<0.01). Conclusion Non-White patients hospitalized with COVID-19 infection were more likely to present with higher severity of disease on admission CXR than White/Non-Hispanic patients, and increased severity was associated with worse outcomes for all patients.

FIGURES

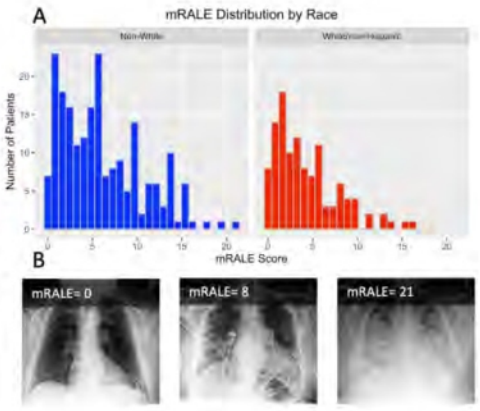


Figure 4 Title: Histogram of mRALE Score Distribution by Race. A) Distribution of mRALE categories on Admission Chest Radiograph by Race/Ethnicity. Non-White/Hispanic patients were more likely to have higher mRALE score categories compared with White/non-Hispanic patients (Coefficient 0.560, 95% CI 0.046 to 1.074 to, p = 0.033). On average, adjusted for potential confounders, Non-White patients presented with higher mRALE scores on admission CXR compared with White patients (average difference 1.60, 95%CI 0.50 to 2.71, p=0.005) in our multiple variable linear regression analyses (B) Examples of modified Radiograph Assessment of Lung Edema (mRALE) annotations for pulmonary disease severity in patients with COVID-19. The top left inset number for each image is the corresponding mRALE score, the average of annotations by multiple raters. A score of 0-4 was categorized as mild, 4.1-10 was moderate, and >10 was severe.

DISPROPORTIONATE IMPACT OF THE COVID-19 PANDEMIC ON IMMIGRANT COMMUNITIES IN THE UNITED STATES

Clark E, Fredricks K, Woc-Colburn L, Bottazzi ME, Weatherhead J.. PLoS Negl Trop Dis. 2020 Jul 13;14(7):e0008484. doi: 10.1371/journal.pntd.0008484. eCollection 2020 Jul.
Level of Evidence: 3 - Local non-random sample

BLUF

This article explores the potential impacts of COVID-19 on immigrants in the United States, with an emphasis on those in Houston, Texas. The most prevalent comorbidities, nationally, include obesity (71.5%), hypertension (HTN, 27.7%), and diabetes (9.6%). Many immigrants are at increased risk for COVID-19 infection as they make up a significant portion of the essential workers work force (20% in Texas) and are more likely to live in large family groups. The unemployment rate increased from 3% to 15% from February to April 2020, and concerns over food insecurity, child support, and healthcare access continue to worsen. Thus, the authors suggest the following policy recommendations in order to better support these disadvantaged communities:
- Expand medicaid
- Eliminate immigration requirements for CHIP
- Pass registration banning ICE in healthcare
- Fund COVID-19 testing and treatment for uninsured
- Provide immigrants and families economic relief packages

FIGURES

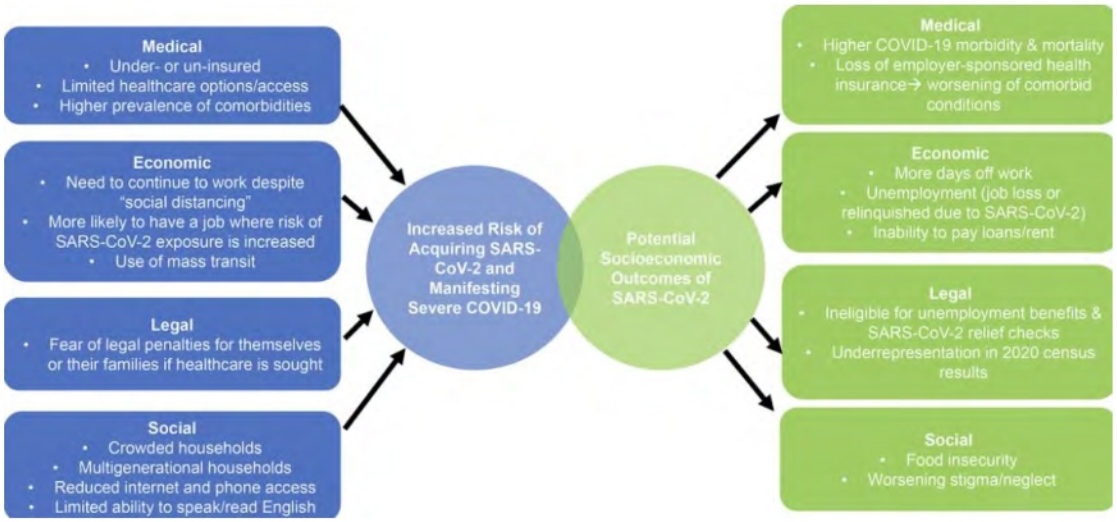


Figure 1: Risk factors and anticipated socioeconomic outcomes for the COVID-19 pandemic in vulnerable immigrant communities.

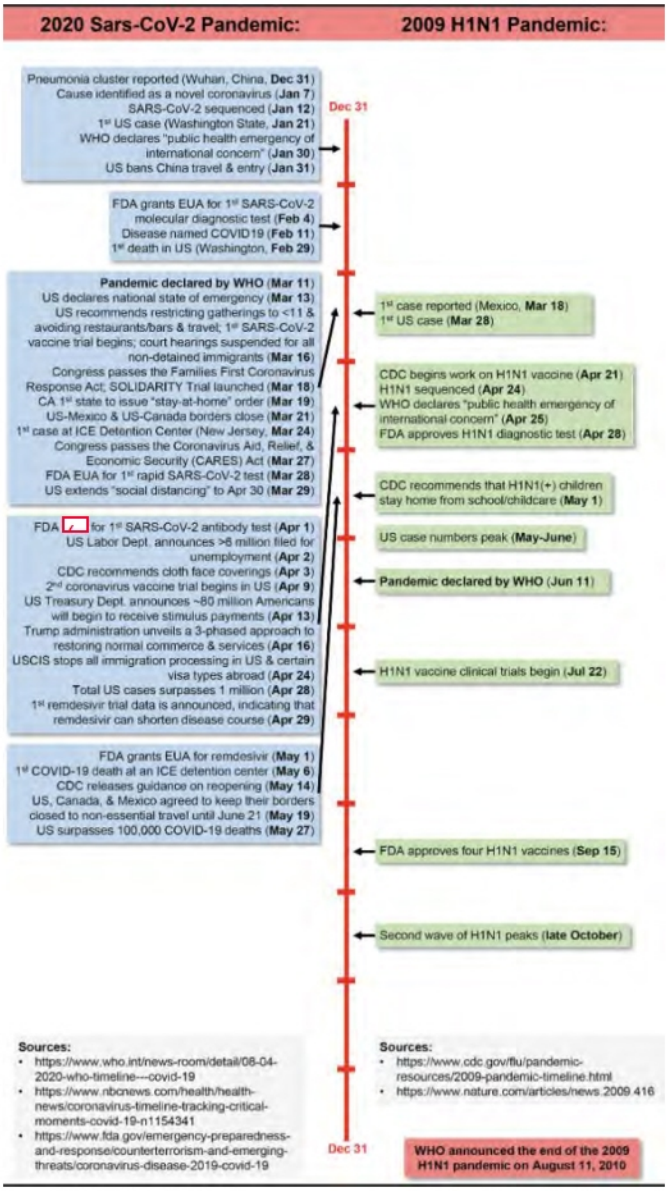


Figure 2: Comparison of COVID-19 and 2009 H1N1 influenza pandemic timelines in the context of events affecting vulnerable immigrant communities in the US. CDC, US Centers for Disease Control and Prevention; EUA, emergency use authorization;

FDA, US Food and Drug Administration; ICE, US Immigration and Customs Enforcement; USCIS, US Citizenship and Immigration Services; WHO, World Health Organization.

COVID-19 AND THE MENTAL HEALTH OF PEOPLE FROM REFUGEE BACKGROUNDS

Rees S, Fisher J.. Int J Health Serv. 2020 Jul 16:20731420942475. doi: 10.1177/0020731420942475. Online ahead of print.
Level of Evidence: Other - Expert Opinion

BLUF

Australian researchers conducted a systemically recruited cohort study to investigate the effects of COVID-19 on the mental health of refugee women from conflict-affected backgrounds (violence, insecurity, persecution, and shortage of food and medicine), and report increased stress levels from detainment, limited trust in state-run institutions, and minimal social support from the community. The researchers hope these findings will better inform healthcare providers in order to improve health care delivery to this vulnerable population.

ABSTRACT

Approximately 1 in 10 of the current 26 million people who are refugees reside in high-income countries. They have commonly experienced trauma related to violence, insecurity, persecution and shortage of food and medicine. Our research suggests that COVID-19 and its health and social sequalae may be triggering past traumatic reactions, exacerbating mental health problems and undermining functioning. The purpose of this article is to promptly communicate these anecdotal findings to general health practitioners to ensure informed and sensitive health care delivery to this vulnerable population.

HEALTH LITERACY AND COVID-19

Spring H.. Health Info Libr J. 2020 Jul 16. doi: 10.1111/hir.12322. Online ahead of print.
Level of Evidence: Other - Opinion

BLUF

This editorial argues health literacy to be a social determinant of health and promotes health literacy to empower communities to be informed and participate in their care, to address disparities, and build community resilience. The novelty of COVID-19 and social distancing has isolated people, and many have chosen to ignore the recommendations over time. The author suggests that health literacy may help to overcome these barriers and promote community safety.

ABSTRACT

In early 2020, the world experienced an unprecedented health crisis. When the pandemic of coronavirus was declared by the World Health Organization, it brought with it sudden and dramatic changes to everyday life. In the UK, the key message from the Government was to 'Stay at home, protect the NHS, save lives', sending out a clear warning that failing to stay at home would put other lives and the ability of the NHS to cope at risk. This editorial discusses COVID-19, how society responded and the vital role that health literacy plays in saving lives during a global health emergency.

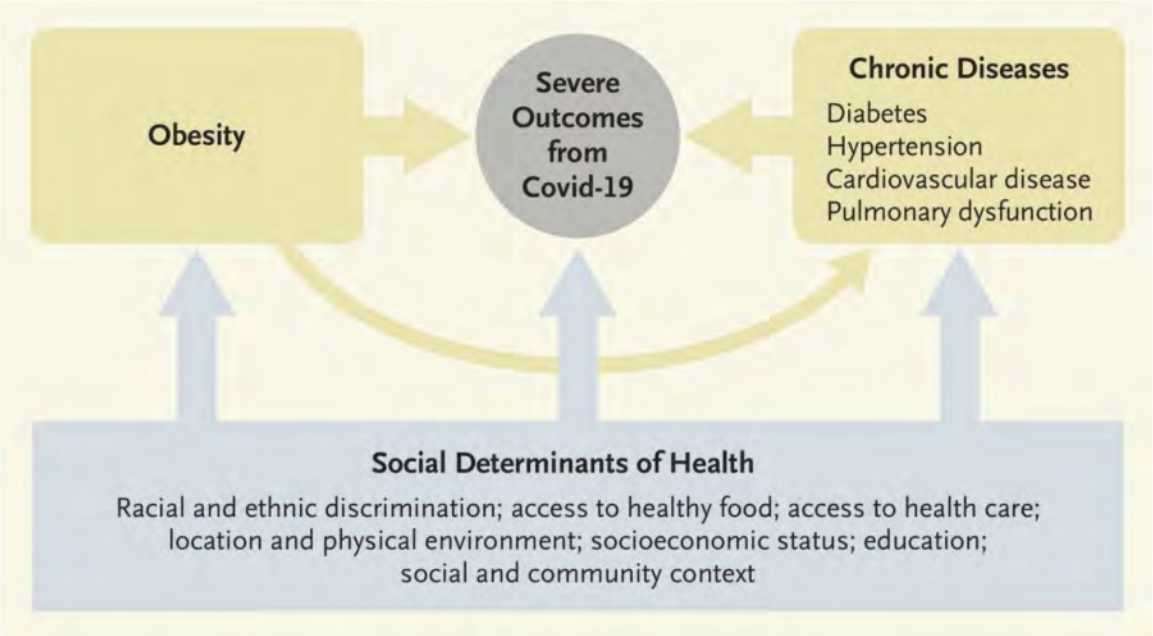
COVID-19 AND DISPARITIES IN NUTRITION AND OBESITY

Belanger MJ, Hill MA, Angelidi AM, Dalamaga M, Sowers [illegible] Mantzoros CS.. N Engl J Med. 2020 Jul 15. doi: 10.1056/NEJMp2021264. Online ahead of print.
Level of Evidence: Other - Opinion

BLUF

Public health activists remark that the socioeconomic, environmental, and educational gaps between populations (notably, Black, Latinx, and Native American households) may be contributing to disparities in nutrition, obesity, and overall worse outcomes from COVID-19 (Figure 1). The authors recommend that the U.S. health care system needs to address strategies to eliminate these health inequities in order to minimize the toll of the COVID-19 pandemic and ensure high-quality, affordable health care.

FIGURES


Obesity
Severe Outcomes from Covid-19
Chronic Diseases
Diabetes
Hypertension
Cardiovascular disease
Pulmonary dysfunction
Social Determinants of Health
Racial and ethnic discrimination; access to healthy food; access to health care; location and physical environment; socioeconomic status; education; social and community context

PREGNANT PERSONS

THIRD TRIMESTER PLACENTAS OF SARS-COV-2-POSITIVE WOMEN: HISTOMORPHOLOGY, INCLUDING VIRAL IMMUNOHISTOCHEMISTRY AND IN SITU HYBRIDIZATION

Smithgall MC, Liu-Jarin X, Hamele-Bena D, Cimic A, Mourad M, Debelenko L, Chen X.. Histopathology. 2020 Jul 21. doi: 10.1111/his.14215. Online ahead of print.
Level of Evidence: 3 - Local non-random sample

BLUF

A study comparing placental histopathology using in-situ hybridization (ISH) and immunohistochemistry (IHC) of 51 SARS-CoV-2 positive and 25 negative persons presenting in third-trimester labor at Columbia University Irving Medical Center from 23 March to 29 April 2020 found nonspecific maternal/fetal malperfusion with evidence of subchorionic thrombi (p=0.026, Figure 1A), intervillous thrombi (Figure 1B), infarction (Figure 1C), segmental avascular-villi (Figure 1D), fetal thrombotic vasculopathy (Figure 1E), and villous agglutination (p=0.003, Figure 1F). There was no evidence of direct viral changes in placental tissue with ISH (Figure 1G) or IHC (Figure 1H), and all infants born to SARS-CoV-2 positive mothers subsequently tested negative for the virus, suggesting no evidence for vertical transmission.

ABSTRACT

AIMS: The wide-variety of affected organ-systems associated with SARS-CoV-2 infection highlights the need for tissue-specific evaluation. We compared placentas from SARS-CoV-2-positive and negative women in our hospital in New York City, which became the epicenter of the COVID-19 pandemic in March 2020. While some limited studies have been published on placentas from SARS-CoV-2-positive women to date, this study, in addition to describing histomorphology, utilizes in-situ hybridization (ISH) for the S-gene encoding the spike-protein and immunohistochemistry (IHC) with the monoclonal-SARS-CoV-2 spike-antibody 1A9 for placental evaluation. METHODS AND RESULTS: In this study, 51 singleton, third-trimester placentas from SARS-CoV-2-positive women and 25 singleton, third-trimester placentas from SARS-CoV-2-negative women were examined histomorphologically using the Amsterdam Criteria as well as with ISH and/or IHC. Corresponding clinical findings and neonatal outcomes also were recorded. While no specific histomorphologic changes related to SARS-CoV-2 were noted in the placentas, evidence of maternal/fetal vascular malperfusion was identified, with placentas from SARS-CoV-2-positive women significantly more likely to show villous agglutination (p=0.003) and subchorionic thrombi (p=0.026) than placentas from SARS-CoV-2-negative women. No evidence of direct viral involvement was identified using ISH and IHC. CONCLUSIONS: In this study, third trimester placentas from SARS-CoV-2-positive women were more likely to show evidence of maternal/fetal vascular malperfusion; however, no evidence of direct viral involvement or vertical transmission was noted by ISH and IHC.

FIGURES

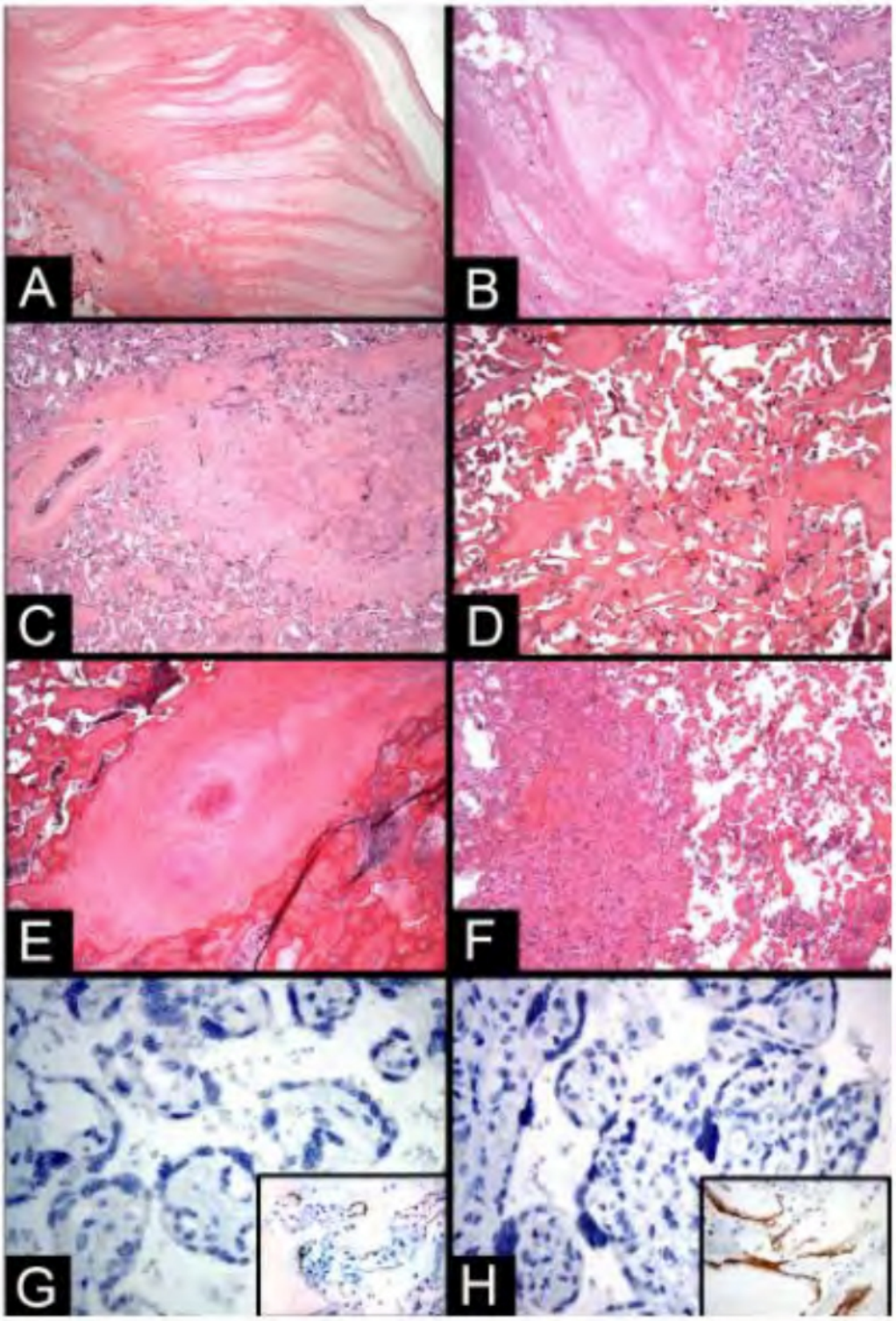


Figure 1: Subchorionic thrombi (A), intervillous thrombi (B), infarction (C), chorangiosis, segmental avascular-villi (D), fetal thrombotic vasculopathy (E), and villous agglutination (F). ISH (G) and IHC (H).

KIDNEY BIOPSY FINDINGS IN PATIENTS WITH COVID-19

Kudose S, Batal I, Santoriello D, Xu K, Barasch J, Peleg Y, Canetta P, Ratner LE, Marasa M, Gharavi AG, Stokes MB, Markowitz GS, D'Agati VD.. J Am Soc Nephrol. 2020 Jul 17:ASN.2020060802. doi: 10.1681/ASN.2020060802. Online ahead of print.

Level of Evidence: 3 - Local non-random sample

BLUF

A cross sectional study conducted in New York, United States between 13 March to 1 June 2020 by authors at Columbia University Irving Medical Center found kidney biopsies of SARS-CoV-2 infected patients (n=17; Table 1) revealed a spectrum of glomerular and tubular injuries (Figure 1, Table 3), acute kidney injury in 15 participants (88%), and lack of SARS-CoV-2 detection in kidney cells as evidence against a direct viral infection mechanism. Authors suggest SARS-CoV-2 has the potential to influence immune responses (innate and adaptive) that lead to new glomerular disease (podocytopathies and anti-glomerular basement membrane nephritis) or alloimmune conditions (lupus nephritis, membranous glomerulopathy, and allograft rejection), as well as acute tubular injury (ATI).

ABSTRACT

BACKGROUND: Coronavirus disease 2019 (COVID-19) is thought to cause kidney injury by a variety of mechanisms. To date, pathologic analyses have been limited to patient reports and autopsy series. METHODS: We evaluated biopsy samples of native and allograft kidneys from patients with COVID-19 at a single center in New York City between March and June of 2020. We also used immunohistochemistry, in situ hybridization, and electron microscopy to examine this tissue for presence of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). RESULTS: The study group included 17 patients with COVID-19 (12 men, 12 black; median age of 54 years). Sixteen patients had comorbidities, including hypertension, obesity, diabetes, malignancy, or a kidney or heart allograft. Nine patients developed COVID-19 pneumonia. Fifteen patients (88%) presented with AKI; nine had nephrotic-range proteinuria. Among 14 patients with a native kidney biopsy, 5 were diagnosed with collapsing glomerulopathy, 1 was diagnosed with minimal change disease, 2 were diagnosed with membranous glomerulopathy, 1 was diagnosed with crescentic transformation of lupus nephritis, 1 was diagnosed with anti-GBM nephritis, and 4 were diagnosed with isolated acute tubular injury. The three allograft specimens showed grade 2A acute T cell-mediated rejection, cortical infarction, or acute tubular injury. Genotyping of three patients with collapsing glomerulopathy and the patient with minimal change disease revealed that all four patients had APOL1 high-risk gene variants. We found no definitive evidence of SARS-CoV-2 in kidney cells. Biopsy diagnosis informed treatment and prognosis in all patients. CONCLUSIONS: Patients with COVID-19 develop a wide spectrum of glomerular and tubular diseases. Our findings provide evidence against direct viral infection of the kidneys as the major pathomechanism for COVID-19-related kidney injury and implicate cytokine-mediated effects and heightened adaptive immune responses.

FIGURES

Pt	Age	Sex	Race	Comorbidities	IS	Temperature, °C	SpO_2 on RA	BP	Edema	COVID-19 Manifestations	Renal Presentation/ Biopsy Indications
1	46	M	B	OSA, obesity (BMI=44)	N	37.1	94	144/100	Y	Cough, fever, sore throat	AKI, NS
2	62	M	B	HTN, prostate carcinoma	N	37.2	98	126/79	Y	Fever, myalgia, weakness	AKI on CKD, NS
3	62	M	B	HTN, DM, prostate carcinoma	N	36.8	91	122/82	N	Fever, hypoxemia, bilateral perihilar and basilar infiltrates on CXR	AKI, NRP
4	57	M	B	HTN, untreated hepatitis C virus	N	38.1	97	173/92	N	Flu-like symptoms with ground glass opacities and patchy consolidation on CXR	AKI on CKD, NRP
5	61	M	B	HTN, obesity (BMI=31.1)	N	38.5	99	134/79	N	Cough, fever	AKI, NRP
6	25	M	B	Obesity (BMI=32.2)	N	38.5	95	117/79	Y	Cough, fever, myalgia, infiltrates on CXR	AKI, NS
7	43	F	B	DM, HLD, streptococcal infection, obesity (BMI=52.5)	N	37.6	96	107/67	N	Cough, fever, sore throat, weakness, patchy LUL infiltrates on CXR	AKI
8	28	M	B	None	N	38.6	96 (on O_2)	143/62	N	Cough, fever, hypoxemia, bilateral infiltrates on CXR, elevated troponin and CPK	AKI
9	67	M	W	HTN, gout, history of tobacco use, obesity (BMI=34.9)	N	36.5	95	135/75	N	Cough, diarrhea, lethargy, multifocal infiltrates on CXR	AKI on CKD
10	51	M	B	HTN, DSA+ OHTx for NICM 1 yr ago, atrial fibrillation, CVA, BPH, HLD	Y	36.8	96	117/81	N	Predominantly nausea, vomiting, abdominal pain, with cough	AKI on CKD
11	72	M	W	HTN, DM, HLD, gout, spinal stenosis, atrial fibrillation	N	36.8	97 (on O_2)	125/73	Y	Cough, pleural effusion on CT	NS
12	70	F	B	HTN, CAD, PVD, cervical carcinoma, GERD, HLD, obesity (BMI=39.4)	N	38.1	92	118/58	Y	Cough, fever, shortness of breath	AKI, NRP
13	27	F	A	SLE with class 2 lupus nephritis	Y	36.9	93	130/80	Y	Cough, fever, shortness of breath, hypoxemia, bilateral infiltrates on CXR, required intubation and ICU admission	AKI, NS
14	48	F	B	GERD, history of tobacco use, obesity	N	37.6	98	185/80	N	Cough, myalgia, infiltrates on CXR	AKI
15	54	M	W	ESKD secondary to IgAN s/p DSA+ LURTx 1 mo ago, HTN, obesity (BMI=30.7)	Y	36.8	99	116/75	N	Asymptomatic	AKI
16	22	M	B	ESKD likely secondary to PLA2R+ MGN s/p DDRTx 2 years ago, HTN	Y	37.7	95	178/127	N	Cough, fever, bilateral infiltrates on CXR, required intubation and ICU admission	CKD
17	54	F	H	ESKD secondary to PCKD s/p DDRTx 2 months ago, HTN	Y	98.8	100	104/70	N	Fever, dry throat	AKI

BP is in millimeters of Hg. Pt, patient; IS, immunosuppression at presentation; SpO_2, oxygen saturation (percentage); RA, room air; M, man; B, black; OSA, obstructive sleep apnea; BMI, body mass index; N, no; Y, yes; NS, nephrotic syndrome; HTN, hypertension; AKI on CKD, AKI superimposed on CKD; DM, diabetes mellitus; CXR, chest x-ray; NRP, nephrotic-range proteinuria; F, woman; HLD, hyperlipidemia; LUL, left upper lobe; O_2, oxygen; CPK, creatine phosphokinase; W, white; DSA, donor-specific antibody; OHTx, orthotopic heart transplantation; NICM, nonischemic cardiomyopathy; CVA, cerebrovascular accident; BPH, benign prostatic hyperplasia; CT, computed tomography; CAD, coronary artery disease; PVD, peripheral vascular disease; GERD, gastroesophageal reflux disease; A, Asian; ICU, intensive care unit; IgAN, IgA nephropathy; s/p, status post; LURTx, living unrelated renal transplantation; MGN, membranous glomerulopathy; DDRTx, deceased donor renal transplantation; H, Hispanic; PCKD, polycystic kidney disease.

Table1. Clinical findings in patients with COVID-19 who underwent kidney biopsy.

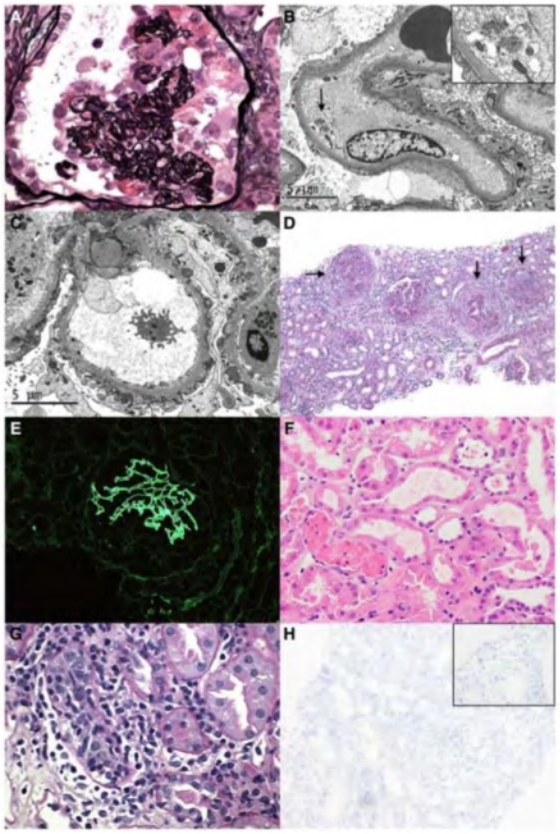


Figure 1. Kidney biopsy findings in patients with COVID-19. (A) Light microscopy demonstrates a lesion of collapsing glomerulopathy characterized by hyperplasia of glomerular epithelial cells and collapse of the underlying glomerular capillaries. Jones methanamine silver. Magnification, x 600. (B) Diffuse foot process effacement and endothelial TRIs (arrow and inset) in a patient with minimal change disease. Electron micrograph. Magnification, X 8000. (C) Subepithelial electron dense deposits in PLA2R-associated membranous glomerulopathy Electron micrograph. Magnification, X15,000. (D) Multiple glomeruli with circumferential cellular crescents (arrows) in a patient with class 4+5 lupus nephritis.Periodic acid–Schiff.Magnification, X100(E) A glomerulus compressed by a crescent with global linear GBM staining for IgG in a patient with

anti-GBM nephritis. Immunofluorescence for IgG. Magnification, X400. (F) Tubular simplification and focal shedding of degenerating epithelial cells into the tubular lumina in a patient with isolated ATI.Hematoxylin and eosin. Magnification, X 400. (G) Severe lymphocytic tubulitis in a patient with acute T cell-mediated rejection. Periodic acid–Schiff. Magnification, X 600 (H)ISH for the virus by automated method showing undetectable viral RNA in the kidney (inset shows positive lung control). Automated ISH with hematoxylin counterstain. Magnification, X 400.

Pt	Diagnosis		Light Microscopy									Electron Microscopy		
	Diagnosis	Other Findings	No. Glom	No. GS	No. Collapse	No. Noncollapsed FSGS	Hypercellularity	Microcysts	II	IFTA	VS	FPE	TRI	Viral Particles
1	Collapsing FSGS	ATI	20	0	14	0	N	Y	Focal	Mild	Mild	NA[a]	NA[a]	N
2	Collapsing FSGS	ATI	8	3	2	0	N	Y	Focal	Moderate	Moderate to severe	100	Y	N
3	Collapsing FSGS	ATI	18	4	4	1	N	Y	None	Moderate	Moderate	30	N	N
4	Collapsing FSGS	ATI	10	1	5	0	N	N	Focal	Severe	Mild	NA	NA	NA
5	Collapsing FSGS	ATI	11	3	7	0	N	Y	Focal	Severe	Moderate to severe	90	N	N
6	MCD	ATI	17	0	0	0	N	Y	None	None	None	100	Y	N
7	ATI		15	0	0	0	N	N	Focal	None	Mild	15	Y	N
8	ATI	Pigment casts	22	0	0	0	N	N	None	None	Mild	10	Y	N
9	ATI		2	1	0	0	N	N	None	Mild	Moderate	NA[a]	NA[a]	N
10	ATI		8	0	0	0	N	N	None	None	Moderate	5	N	N
11	MGN	PLA2R stain positive[b]	15	3	0	4	N	N	Focal	Mild	Mild to moderate	100	N	N
12	MGN	PLA2R stain negative[b]	3	2	0	0	N	Y	Focal	Moderate	Moderate to severe	30	Y	N
13	LN class 4+5		35	9	0	0	Mes, Endo, Crescents	N	Diffuse	Mild	Mild	90	Y	N
14	Anti-GBM GN	ATI, RBC casts	32	1	0	0	Crescents	Y	Diffuse	Mild	Moderate	NA[a]	NA[a]	N
15	TCMR grade 2A		11	1	0	0	N	N	Focal	None	Mild	NA	NA	NA
16	Infarction[c]		NA	NA	NA	NA	NA	N	Focal	Severe	NA	NA	NA	NA
17	ATI		20	1	0	0	N	N	None	None	Mild	NA	NA	NA

Pt, patient; glom, No., number; glomerulus or glomeruli; GS, global sclerosis; II, interstitial inflammation; IFTA, tubular atrophy and interstitial fibrosis; VS, vascular sclerosis; FPE, foot process effacement (percentage); N, no; Y, yes; NA, not available; MCD, minimal change disease; LN, lupus nephritis; Mes, mesangial hypercellularity; Endo, endocapillary hypercellularity; RBC, red blood cell; TCMR, T cell–mediated rejection.
[a]All glomeruli were involved by crescents in the patient with anti-GBM GN, and no glomeruli were available for ultrastructural examination in two patients with native kidney biopsies.
[b]Performed by tissue staining.
[c]Nephrectomy specimen.

Figure 1. Kidney biopsy findings in patients with COVID-19. (A) Light microscopy demonstrates a lesion of collapsing glomerulopathy characterized by hyperplasia of glomerular epithelial cells and collapse of the underlying glomerular capillaries. Jones methanamine silver. Magnification, x 600. (B) Diffuse foot process effacement and endothelial TRIs (arrow and inset) in a patient with minimal change disease. Electron micrograph. Magnification, X 8000. (C) Subepithelial electron dense deposits in PLA2R-associated membranous glomerulopathy Electron micrograph. Magnification, X15,000. (D) Multiple glomeruli with circumferential cellular crescents (arrows) in a patient with class 4+5 lupus nephritis.Periodic acid–Schiff.Magnification, X100(E) A glomerulus compressed by a crescent with global linear GBM staining for IgG in a patient with anti-GBM nephritis. Immunofluorescence for IgG. Magnification, X400. (F) Tubular simplification and focal shedding of degenerating epithelial cells into the tubular lumina in a patient with isolated ATI.Hematoxylin and eosin. Magnification, X 400. (G) Severe lymphocytic tubulitis in a patient with acute T cell-mediated rejection. Periodic acid–Schiff. Magnification, X 600 (H)ISH for the virus by automated method showing undetectable viral RNA in the kidney (inset shows positive lung control). Automated ISH with hematoxylin counterstain. Magnification, X 400.

VIRUSES AND ASTHMA: THE ROLE OF COMMON RESPIRATORY VIRUSES IN ASTHMA AND ITS POTENTIAL MEANING FOR SARS-COV-2

Novak N, Cabanillas B.. Immunology. 2020 Jul 20. doi: 10.1111/imm.13240. Online ahead of print.
Level of Evidence: Other - Review / Literature Review

BLUF

A review study conducted by allergy experts in Bonn, Germany and Madrid, Spain discussed how patients with asthma may have reduced capacity to fight off respiratory viruses due to increased Th2 inflammation and production of IL-4, IL-5, and IL-13, reduced levels of Type 1 Interferons, and increased quantities of pro-Th2 epithelial-derived cytokines (Figures 1 and 2). This authors suggest that patients with asthma who develop COVID-19 be monitored and followed up on to evaluate the sequelae and long term effects from infection with SARS-CoV-2. To note, most of the data in this publication was acquired prior to the onset of the SARS-CoV-2 pandemic.

SUMMARY

Additional points discussed:
-Asthmatic patients can have increased levels of mast cells which permit replication of respiratory viral infections.
-Two cohort studies in Wuhan found the prevalence of asthma in COVID-19 patients was not greater than that of the general population. However, a prospective cohort study in the United Kingdom found the prevalence of asthma in COVID-19 patients was 17.9% which is greater than that of the general population.
-Certain viruses may be capable of producing IgE which can exacerbate asthma symptoms.

ABSTRACT

Viral infections and atopic diseases are closely related and contribute to each other. The physiological deficiencies and immune mechanisms that underlie atopic diseases can result in a suboptimal defense against multiple viruses and promote a suitable environment for their proliferation and dissemination. Viral infections, on the other hand, can induce per se several immunological mechanisms involved in allergic inflammation capable to promote the initiation or exacerbation of atopic diseases such as atopic asthma. In a world that is affected more and more by factors that significantly impact the prevalence of atopic diseases, coronavirus disease 2019 (COVID-19) induced by the novel coronavirus severe acute respiratory syndrome (SARS-CoV-2) is having an unprecedented impact with still unpredictable consequences. Therefore, it is of crucial importance to revise the available scientific literature regarding the association between common respiratory viruses and asthma, as well as the newly emerging data about the molecular mechanisms of SARS-CoV-2 infection and its possible relation with asthma, to better understand the interrelation between common viruses and asthma and its potential meaning on the current global pandemic of COVID-19.

FIGURES

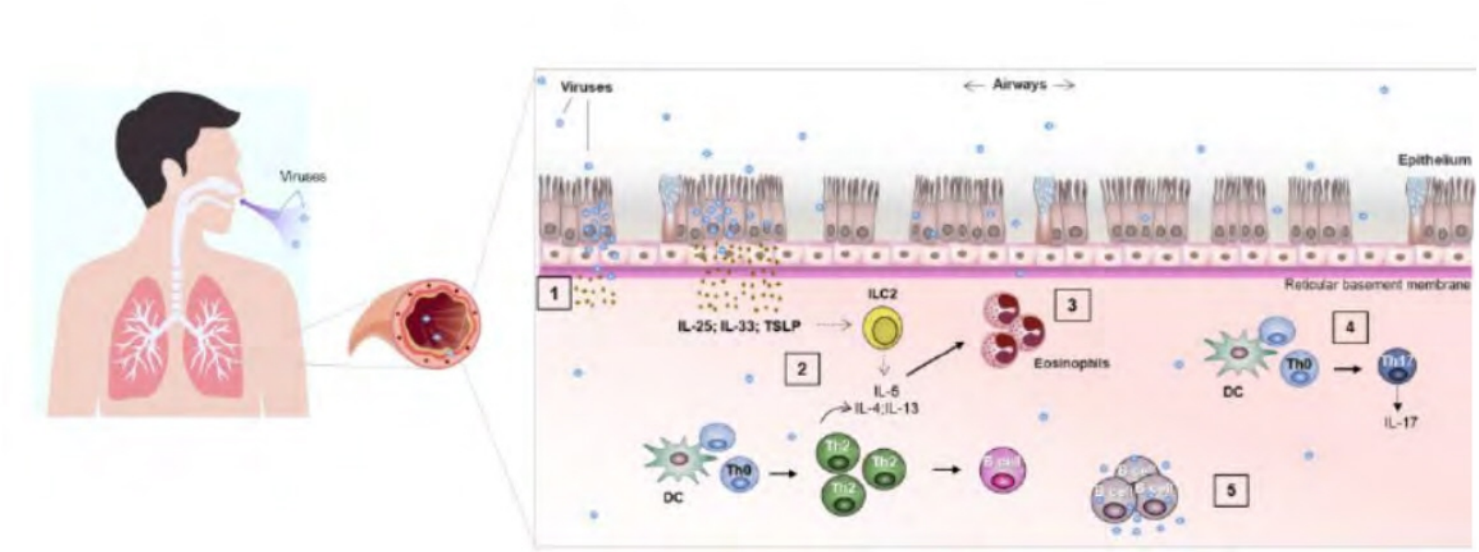


Figure 1. Mechanisms involved in the infection of respiratory viruses that can promote asthma development. Respiratory viruses can induce immunological and morphological changes that can contribute to the development of asthmatic processes. (1) Respiratory viruses replicate in the airway epithelium, which can alter the epithelial barrier integrity. (2 and 3) Infected epithelial cells promote the production of pro-inflammatory cytokines such as IL-25, IL-33, and TSLP, which induce the activation of ILC2s, DCs, Th2 cells, increasing Th2 inflammation linked to atopic asthma. (4) During respiratory viral infections, the cytokine IL-17, a proinflammatory cytokine produced by Th17 cells and linked to asthma, can increase. (5) Respiratory viruses can infect immune cells such as B cells, macrophages, or T cells which may contribute to viral replication and propagation. ILC2: group 2 innate lymphoid cells; DC: myeloid dendritic cell; TSLP: thymic stromal lymphopoietin.

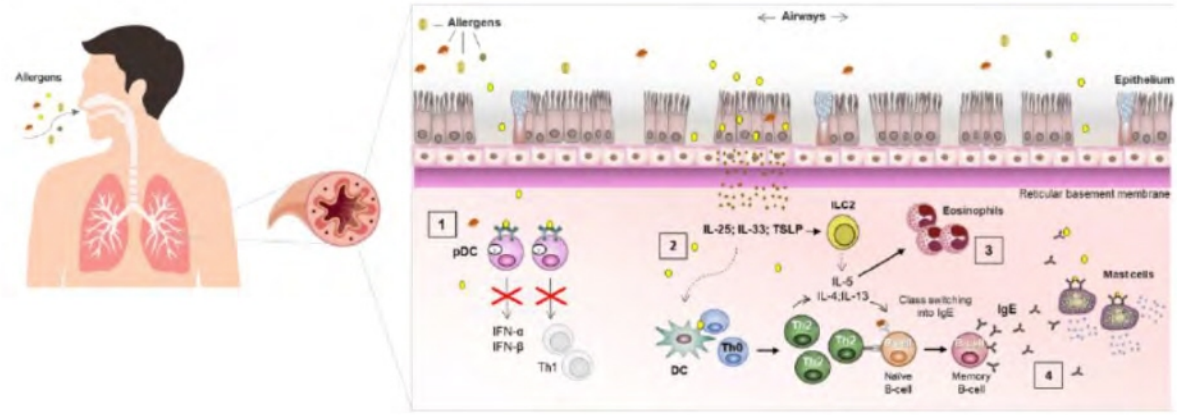


Figure 2. Pathophysiologic mechanisms of asthma that may act as facilitators of respiratory viral infections. (1) An impaired production of type I interferons (IFN-a/IFN-B) has been found in a high proportion of asthmatic patients. One of the mechanisms involves crosslinking of IgE bound to the high-affinity receptor of IgE (FCeRI) by allergens in pDCs that result in decreased TLR expression and a decline in type I interferons production. (2) Th2 inflammation in asthma linked to the production of IL-4, IL-5, and IL-13 by Th2 cells and ILC2s has been related to reduced protection against viruses. Furthermore, allergen exposure can induce production in the epithelium of cytokines that can synergistically interact with respiratory viruses. (3) Asthma is characterized by abundant infiltration of immune cells that can act as infective targets for respiratory viruses. (4) In the context of Th2 polarization in atopic asthma high amounts of specific IgE against different allergens is produced. Recent studies have proposed that specific IgE against certain viruses can also be produced. pDCs: plasmacytoid dendritic cells; ILC2: group 2 innate lymphoid cells; DC: myeloid dendritic cell; TSLP: thymic stromal lymphopoietin.

IN SILICO

ACE2 GENE VARIANTS MAY UNDERLIE INTERINDIVIDUAL VARIABILITY AND SUSCEPTIBILITY TO COVID-19 IN THE ITALIAN POPULATION

Benetti E, Tita R, Spiga O, Ciolfi A, Birolo G, Bruselles A, Doddato G, Giliberti A, Marconi C, Musacchia F, Pippucci T, Torella A, Trezza A, Valentino F, Baldassarri M, Brusco A, Asselta R, Bruttini M, Furini S, Seri M, Nigro V, Matullo G, Tartaglia M, Mari F; GEN-COVID Multicenter Study, Renieri A, Pinto AM.. Eur J Hum Genet. 2020 Jul 17. doi: 10.1038/s41431-020-0691-z. Online ahead of print.
Level of Evidence: Other - Mechanism-based reasoning

BLUF

To examine the interaction between angiotensin converting enzyme 2 (ACE2) variants and the SARS-CoV-2 spike protein, Italian investigators conducted molecular dynamics simulations of databased genomes of non-Finnish European and Italian COVID-19 patients in March 2020. Investigators identified the following:
- Several specific ACE2 variants were present in Italian/non-Finnish European populations but absent in East Asian populations that affected key interactions with SARS-CoV-2 spike protein binding to ACE2.
- When compared to an Italian control group, Italian COVID-19 patients had an overall lower degree of ACE2 gene variability.
- In support of previous studies, a genetic component to COVID-19 susceptibility was also found, proposing that inheritance of certain ACE2 variants and variable gene expression through X-inactivation could account for differential COVID-19 morbidity among sexes and ethnic groups.
- The study was limited by a narrow number of simulated ACE2-SARS-CoV-2 interactions due to technological constraints.

ABSTRACT

In December 2019, an initial cluster of interstitial bilateral pneumonia emerged in Wuhan, China. A human-to-human transmission was assumed and a previously unrecognized entity, termed coronavirus disease-19 (COVID-19) due to a novel coronavirus (SARS-CoV-2) was described. The infection has rapidly spread out all over the world and Italy has been the first European country experiencing the endemic wave with unexpected clinical severity in comparison with Asian countries. It has been shown that SARS-CoV-2 utilizes angiotensin converting enzyme 2 (ACE2) as host receptor and host proteases for cell surface binding and internalization. Thus, a predisposing genetic background can give reason for interindividual disease susceptibility and/or severity. Taking advantage of the Network of Italian Genomes (NIG), here we mined whole-exome

sequencing data of 6930 Italian control individuals from five different centers looking for ACE2 variants. A number of variants with a potential impact on protein stability were identified. Among these, three more common missense changes, p.(Asn720Asp), p.(Lys26Arg), and p.(Gly211Arg) were predicted to interfere with protein structure and stabilization. Rare variants likely interfering with the internalization process, namely p.(Leu351Val) and p.(Pro389His), predicted to interfere with SARS-CoV-2 spike protein binding, were also observed. Comparison of ACE2 WES data between a cohort of 131 patients and 258 controls allowed identifying a statistically significant (P value < 0.029) higher allelic variability in controls compared with patients. These findings suggest that a predisposing genetic background may contribute to the observed interindividual clinical variability associated with COVID-19, allowing an evidence-based risk assessment leading to personalized preventive measures and therapeutic options.

FIGURES

Table 2 Predicted changes in ACE2 protein stability as consequence of residues changes.

Wild Residue	Residue position	Mutant Residue	Predicted ΔΔG	Interaction Network around (5 Å)	Outcome
V	506	A	-2,456	*Y180, L456, R460, P500, A501, S502, L503, F504, H505, N506, S507*	*Highly Destabilizing*
V	209	G	-2,353	*Y207, E208, V209, N210, G211, V212, Y215, D216, Y217, P565, T567*	*Highly Destabilizing*
G	377	E	-2,231	*H373,* H374, E375, M376, G377, H378, I379, A380, Y381, F315, H401, V404, G405, M408	*Highly Destabilizing*
A	264	G	-1,555	*L262, P263, A264, H265, L266, L267, W271, W478, V487, V488, E489, P490, W165*	*Destabilizing*
C	498	R	-1,539	*Y497, C498, D499, P500, A501, S502, G173, R177, L176, Y180, W459, W473, M474*	*Destabilizing*
A	246	T	-1,454	*A242, Y243, V244, R245, A246, K247, L248, M249*	*Destabilizing*
G	377	W	-1,318	*H373* H374, E375, M376, G377, H378, I379, A380, Y381, F315, H401, V404, G405, M408	*Destabilizing*
L	351	V	-1,173	*W349, D350, L351, G352*, D355, R357, Y41, S44, L45, W48	*Destabilizing*
P	389	H	-1,161	*A387, Q388, P389, F390, L391, L392,* N33, T92 Q96	*Destabilizing*
T	55	A	-0,948	N53, I54, T55, E56, E57, N58, V59	*Destabilizing*
D	206	G	-0,87	*W203, G205, D206, Y207, A396, N397, E398, G399*	*Destabilizing*
K	26	R	-0,79	*E22, E23, Q24, A25, K26, T27, L29, N90, V93*	*Destabilizing*
N	580	D	-0,629	*M579, N580, V581, R582, P583, Q524*	*Destabilizing*
S	547	C	-0,611	*I544, S545*, N546, S547, T548, E549, A550, G551	*Destabilizing*
A	65	V	-0,423	*N61, M62, N63, N64, A65, G66, D67, K68, Q42, S43, S44, A46*	*Destabilizing*
H	505	R	-0,345	*L503, F504, M505, F512, Y515, Y510, S511, R273*	*Destabilizing*
T	92	V	-0,322	N90, L91, T92, V93, L95, Q96, P389, L392, S563, E564	*Destabilizing*
E	329	G	-0,302	*Q325, G326, F327, W328, E329, N330, S331*	*Destabilizing*
G	211	R	-0,283	*V209, N210, G211, V212, D213, D216*	*Destabilizing*
T	92	I	-0,155	N90, L91, T92, V93, L95, Q96, P389, L392, S563, E564	*Destabilizing*
D	494	V	-0,041	*H493, D494, E495, T496, Y497*	*Destabilizing*
Q	102	P	0,036	Q98, A99, Q102, N103, G104	(Stabilizing)

DUET program results that display predicted change in folding free energy upon ACE2 missense variant (ΔΔG in kcal/mol). In the first three columns are reported single missense variants with specific position on ACE2 protein. The residues in the first column highlighted in gray are involved in N-glycosylation pattern NxT/S, therefore those missense variants determine the loss glycosylation of Asparagine 53 and 90, respectively. In the fourth column is reported ΔΔG analysis predict effects of missense variants on protein stability using an integrated computational approach. The column "Interaction Network around (5 Å)" shows for each single missense variant the residues around 5 Å. In this column, we highlight in green residues involved in spike SARS-CoV protein interaction, in yellow residues involved in Zinc coordination and finally in magenta residues of Asn involved in N-glycosylation. The last column defines the outcome of protein stability for each single missense variant. An increasing negative value for the ΔΔG is correlated with a higher destabilizing effect, while a positive value is associated with a variant predicted as stabilizing.

A SURFACE COATING THAT RAPIDLY INACTIVATES SARS-COV-2

Behzadinasab S, Chin A, Hosseini M, Poon LLM, Ducker WA.. ACS Appl Mater Interfaces. 2020 Jul 13. doi: 10.1021/acsami.0c11425. Online ahead of print.
Level of Evidence: 5 - Mechanism-based reasoning

BLUF

A collaborative basic research study investigates SARS-CoV-2 viral titer loads on glass and stainless steel surfaces when coated with cuprous oxide (Cu2O) and found a 99.9% reduction of viral loads after one hour and retained potency after multiple exposures (Table 1), suggesting use of Cu2O as a preventative measure of coating for regularly used items.

ABSTRACT

SARS-CoV-2, the virus that causes the disease COVID-19, remains viable on solids for periods of up to one week, so one potential route for human infection is via exposure to an infectious dose from a solid. We have fabricated and tested a coating that is designed to reduce the longevity of SARS-CoV-2 on solids. The coating consists of cuprous oxide (Cu2O) particles bound with polyurethane. After one hour on coated glass or stainless steel, the viral titer was reduced by about 99.9% on average compared to the uncoated sample. An advantage of a polyurethane-based coating is that polyurethane is already used to coat a large number of everyday objects. Our coating adheres well to glass and stainless steel, as well as everyday items that people may fear to touch during a pandemic, such as a doorknob, a pen, and a credit card keypad button. The coating performs well in the cross-hatch durability test and remains intact and active after 13 days immersed in water, or after exposure to multiple cycles of exposure to virus and disinfection.

FIGURES

condition	comparison	% reduction	log reduction	95% CI*	*p*-value	Figure no.
Cu_2O/PU coating on glass	glass	>99.98	>3.64	99.95	5×10^{-4}	2
Cu_2O/PU coating on stainless steel	stainless steel	99.90	2.97	98.51	8×10^{-3}	2
PU coating on glass	glass	10	0.04	-164	0.22	S7
Cu_2O/PU on glass, stored 13 days underwater	glass†	99.96	3.39	99.56	8×10^{-4}	S10
Cu_2O/PU glass, high contact angle	glass	99.89	2.97	99.22	2×10^{-6}	S8
Cu_2O/PU glass, 5× disinfection	glass	99.89	2.95	99.79	4×10^{-8}	3

* 95% confidence limit lower bound. Upper bound set at 100%. Calculated for one-tail, assuming heteroscedastic.

† Comparison sample not stored under water.

p-values for Student's t-test calculated for one-tail, assuming heteroscedastic.

Table 1. Average reduction of SARS-CoV-2 titer on Cu_2O/PU coated solid in 1 h compared to titer on uncoated solid in 1h.

REDUCING RISKY BEHAVIOR WITH HABIT REVERSAL: A REVIEW OF BEHAVIORAL STRATEGIES TO REDUCE HABITUAL HAND-TO-HEAD BEHAVIOR

Heinicke MR, Stiede [illegible] Miltenberger RG, Woods DW.. J Appl Behav Anal. 2020 Jul 20. doi: 10.1002/jaba.745. Online ahead of print.
Level of Evidence: Other - Guidelines and Recommendations

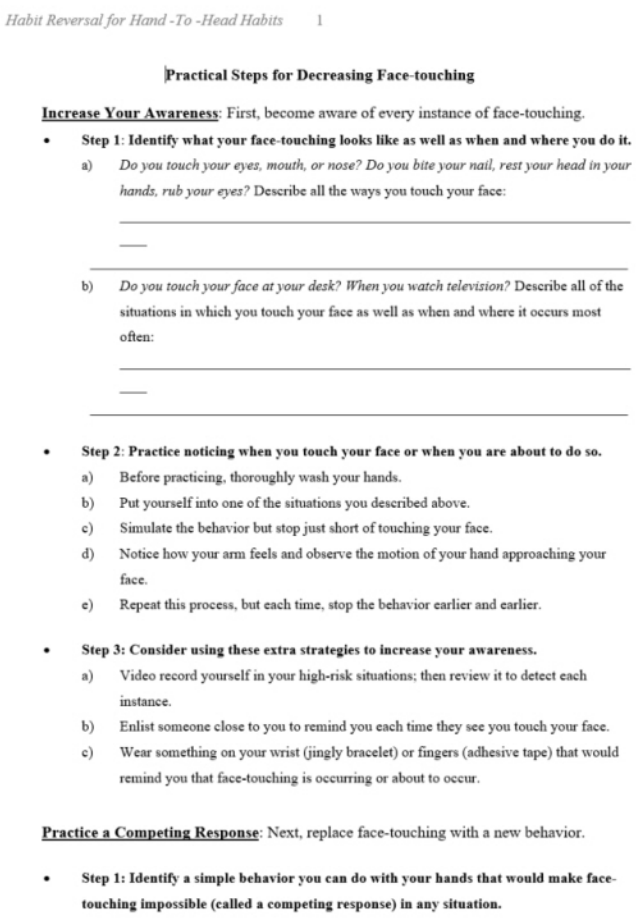
BLUF

This review by authors in psychology assessed efficacy of habit reversal training (HRT) in treating repetitive behavior problems and found HRT (increased awareness and practicing a competing response) to be effective in reducing hand-to-face or head contact, as shown in 35/39 studies examined. Authors advocate for self-administration of HRT in a wide distribution during the COVID-19 pandemic, providing recommendations/instructions for practical application (Worksheets 1 and 2) to achieve decreased face-touching and reduced disease transmission.

ABSTRACT

Habit reversal training (HRT) has been a mainstay of behavior analysts' repertoire for nearly the last 50 years. HRT has been effective in treating a host of repetitive behavior problems. In the face of the current coronavirus pandemic, HRT has practical public health importance as a possible intervention for reducing hand-to-head behaviors that increase the risk of viral infection. The current paper provides a brief review of HRT for hand-to-head habits that is designed for a broad audience and concludes with practical suggestions, based on HRT, for reducing face-touching behaviors.

FIGURES



Habit Reversal for Hand -To -Head Habits 1

Practical Steps for Decreasing Face-touching

Increase Your Awareness: First, become aware of every instance of face-touching.

- **Step 1**: **Identify what your face-touching looks like as well as when and where you do it.**
  a) *Do you touch your eyes, mouth, or nose? Do you bite your nail, rest your head in your hands, rub your eyes?* Describe all the ways you touch your face:
  b) *Do you touch your face at your desk? When you watch television?* Describe all of the situations in which you touch your face as well as when and where it occurs most often:

- **Step 2**: **Practice noticing when you touch your face or when you are about to do so.**
  a) Before practicing, thoroughly wash your hands.
  b) Put yourself into one of the situations you described above.
  c) Simulate the behavior but stop just short of touching your face.
  d) Notice how your arm feels and observe the motion of your hand approaching your face.
  e) Repeat this process, but each time, stop the behavior earlier and earlier.

- **Step 3: Consider using these extra strategies to increase your awareness.**
  a) Video record yourself in your high-risk situations; then review it to detect each instance.
  b) Enlist someone close to you to remind you each time they see you touch your face.
  c) Wear something on your wrist (jingly bracelet) or fingers (adhesive tape) that would remind you that face-touching is occurring or about to occur.

Practice a Competing Response: Next, replace face-touching with a new behavior.

- **Step 1: Identify a simple behavior you can do with your hands that would make face-touching impossible (called a competing response) in any situation.**

Worksheet 1. Habit reversal training (HRT) guidelines.

Would it be possible to make a fist and hold it in your lap? Sit on your hands? Fold your hands or arms? Describe your competing response for each situation:

_____

_____

- **Step 2: Practice your competing response.**
  a) Simulate face-touching and then immediately interrupt the behavior and use your competing response for 60 seconds.
  b) Repeat this process, but each time, interrupt the movement of your hand earlier and earlier and immediately use your competing response.ss

- **Step 3: Make the competing response a new habit in your everyday life.**
  a) Practice the exercises until you catch every movement of your hand toward your face.
  b) Continue to use the competing response during all waking hours.

- **Step 4: Consider using these extra strategies to use your competing response consistently.**
  a) Keep a tally sheet with you in high-risk situations. Each time you touch your face, it in first column, and each time you use your competing response before touching your face, it in the second column.

Continue to enlist your support person to remind you to use your competing response.

Worksheet 2. Habit reversal training (HRT) guidelines.

MANAGEMENT

ACUTE CARE

MULTIFACTORIAL PATHOGENESIS OF COVID-19-RELATED COAGULOPATHY. CAN DEFIBROTIDE HAVE A ROLE IN THE EARLY PHASES OF COAGULATION DISORDERS?

Macciò A, Madeddu C, Caocci G, La Nasa G.. J Thromb Haemost. 2020 Jul 21. doi: 10.1111/jth.15021. Online ahead of print.
Level of Evidence: Other - Expert Opinion

BLUF

A letter written by members of the Department of Medical Sciences and Public Health at the University of Cagliari in Italy discusses the connection between infection with the SARS-CoV-2 virus and endothelial damage, platelet aggregation, and systemic inflammation associated with coagulation. The authors suggest the potential use of defibrotide in the early stages of COVID-19 due to its antithrombotic and anti-inflammatory effects, in an attempt to improve prognosis via preventing disease progression.

ABSTRACT

Thrombotic complications emerged as an important issue in patients with coronavirus disease 2019 (COVID-19). Consolidated reports regarding the clinical and laboratory findings in COVID-19 patients reveal thrombocytopenia, elevated D-dimer, prolonged prothrombin time, disseminated intravascular coagulation, and pulmonary intravascular coagulation (PIC). Recently, some authors emphasized the potential role of antiphospholipid antibodies in the pathogenesis of thrombotic events in patients with severe COVID-19.

COVID-19: WHAT TYPE OF CYTOKINE STORM ARE WE DEALING WITH?

Monneret G, Benlyamani I, Gossez M, Martin JB, Martín-Fernandez M, Sesques P, Wallet F, Venet F.. J Med Virol. 2020 Jul 18. doi: 10.1002/jmv.26317. Online ahead of print.
Level of Evidence: Other - Expert Opinion

BLUF

An expert opinion from the Immunology Laboratory at Edouard Herriot Hospital, France discusses the use of toclizumab, a monoclonal antibody against the interleukin-6 (IL-6) receptor, in patients diagnosed with COVID-19. Though mean and median levels of IL-6 for COVID-19-positive patients are similar to those in patients with inflammatory diseases (chronic infection, rheumatoid arthritis, Crohn's disease, etc), they were found to still be lower when compared to diseases more often linked to a cytokine storm where tocilizumab would be more effective, like sepsis or cytokine release syndrome (Figure 1). However, the authors still advocate for its cautious use on an individualized basis for patients with COVID-19 based on extent of lymphopenia, presence of ARDS, disease chronology, and IL-6 level stratification.

ABSTRACT

We read with great interest the comment published by Andrianopoulos et al. in which the authors advocate for cautious use of tocilizumab in COVID-19 patients 1 . Tocilizumab is a monoclonal antibody against the interleukin-6 receptor which has immunosuppressive properties. Whereas accumulating results from uncontrolled trials present tocilizumab as an effective agent blocker of disease progression, some contrasting studies also progressively appear in COVID-19 literature 2-5 . This muddies the waters and makes the situation more confused. This article is protected by copyright. All rights reserved.

FIGURES

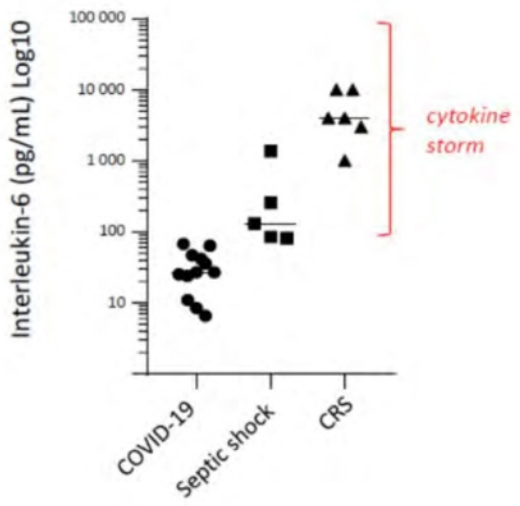


Figure 1: Median/Mean IL-6 Values in COVID-19, septic shock and cytokine release syndrome (CRS).

COVID-19 AND TREG/TH17 IMBALANCE: POTENTIAL RELATIONSHIP TO PREGNANCY OUTCOMES

Muyayalo KP, Huang DH, Zhao SJ, Xie T, Mor G, Liao AH.. Am J Reprod Immunol. 2020 Jul 14:e13304. doi: 10.1111/aji.13304. Online ahead of print.
Level of Evidence: 2 - Review / Literature Review

BLUF

A literature review summarizes the effect of Treg/Th17 cell imbalance on uncontrolled systemic inflammation in severe COVID-19 cases in pregnancy pooled from nine studies found that:
In COVID-19 positive pregnant patients:
- Treg cells decreased and Th17 cells increased, with a consequent decrease in the Treg/Th17 cell ratio.
- Treg cells dysregulation in COVID-19 was shown trigger hyperinflammation and tissue damage.
- Increased Th17 is associated with fetal allograft rejection at the feto-maternal interface.
- COVID-19 infection puts pregnant persons at higher risk for pregnancy complications.

ABSTRACT

Caused by a novel type of virus, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), coronavirus disease 2019 (COVID-19) constitutes a global public health emergency. Pregnant women are considered to have a higher risk of severe morbidity and even mortality due to their susceptibility to respiratory pathogens and their particular immunological state. Several studies assessing SARS-CoV-2 infection during pregnancy reported adverse pregnancy outcomes in patients with severe conditions, including spontaneous abortion, preterm labor, fetal distress, cesarean section, preterm birth, neonatal asphyxia, neonatal pneumonia, stillbirth, and neonatal death. However, whether these complications are causally related to SARS-CoV-2 infection is not clear. Here, we reviewed the scientific evidence supporting the contributing role of Treg/Th17 cell imbalance in the uncontrolled systemic inflammation characterizing severe cases of COVID-19. Based on the recognized harmful effects of these CD4+ T cell subset imbalances in pregnancy, we speculated that SARS-CoV-2 infection might lead to adverse pregnancy outcomes through the deregulation of otherwise tightly-regulated Treg/Th17 ratios, and to subsequent uncontrolled systemic inflammation. Moreover, we discuss the possibility of vertical transmission of COVID-19 from infected mothers to their infants, which could also explain adverse perinatal outcomes. Rigorous monitoring of pregnancies and appropriate measures should be taken to prevent and treat early eventual maternal and perinatal complications.

LUNG ULTRASOUND FOR TREATMENT OF PATIENTS WITH COVID-19: PLEASE REPORT YOUR SETTINGS AND MECHANICAL INDEX

Rosado-Mendez IM, Smargiassi A, Inchingolo R, Soldati G, Muller M, Demi L.. J Ultrasound Med. 2020 Jul 21. doi: 10.1002/jum.15389. Online ahead of print.
Level of Evidence: 5 - Mechanism-based reasoning

BLUF

A mechanism based reasoning study conducted by experts in Italy, Mexico, and the United States discussed the utilization of lung ultrasound (LUS) to evaluate acute respiratory distress syndrome (ARDS) and SARS-CoV-2 pneumonia, suggesting that this imaging could be beneficial for accurate diagnosis particularly in low-income and resource-poor settings. However, healthcare providers must be careful to avoid cavitation and tissue damage by minimizing the acoustic energy and scanning time without compromising diagnostic value.

PAN-FAMILY ASSAYS FOR RAPID VIRAL SCREENING: REDUCING DELAYS IN PUBLIC HEALTH RESPONSES DURING PANDEMICS

Erlichster M, Chana G, Zantomio D, Goudey B, Skafidas E.. Clin Infect Dis. 2020 Jul 20:ciaa1028. doi: 10.1093/cid/ciaa1028. Online ahead of print.
Level of Evidence: 5 - Mechanism-based reasoning

BLUF

An in silico study conducted in Australia using pan-family assays derived from 60 species of coronavirus indicates that these assays would "be expected to be sufficiently specific for a novel pathogen to allow for broad population screening" due to recognition of highly conserved regions of the viral genome (Figure 1). The authors suggest that the development of these pan-family assays would help detect SARS-CoV-2 with adequate sensitivity and specificity to be practical in largely affected areas with limited testing options. The utility of pan-family assays could prove useful not only for COVID-19, but also in any future pandemics.

ABSTRACT

BACKGROUND: COVID-19 has highlighted deficiencies in the testing capacity of many developed countries during the early stages of pandemics. Here we describe a strategy utilizing pan-family viral assays to improve early accessibility of large-scale nucleic acid testing. METHODS: Coronaviruses and SARS-CoV-2 were used as a case-study for assessing utility of pan-family viral assays during the early stages of a novel pandemic. Specificity of a pan-coronavirus (Pan-CoV) assay for a novel pathogen was assessed using the frequency of common human coronavirus (HCoV) species in key populations. A reported Pan-CoV assay was assessed to determine sensitivity to 60 reference coronaviruses, including SARS-CoV-2. The resilience of the primer target regions of this assay to mutation was assessed in 8893 high-quality SARS-CoV-2 genomes to predict ongoing utility during pandemic progression. RESULTS: Due to common HCoV species, a Pan-CoV assay would return false positives for as few as 1% of asymptomatic adults, but up to 30% of immunocompromised patients with respiratory disease. Half of reported Pan-CoV assays identify SARS-CoV-2 and with small adjustments can accommodate diverse variation observed in animal coronaviruses. The target region of one well established Pan-CoV assay is highly resistant to mutation compared to species-specific SARS-CoV-2 RT-PCR assays. CONCLUSIONS: Despite cross-reactivity with common pathogens, pan-family assays may greatly assist management of emerging pandemics through prioritization of high-resolution testing or isolation measures. Targeting highly conserved genomic regions make pan-family assays robust and resilient to mutation. A strategic stockpile of pan-family assays may improve containment of novel diseases prior to the availability of species-specific assays.



Figure 1. Alignment of 4 Reported Pan-Coronavirus Primer Targets and the SARS-CoV-2 Genome. IUPAC nucleotide code used where degenerate primers are reported. Each of the 5 rows shows a different region of the SARS2 genome. Primers targets with a 100% match to the SARS-CoV-2 genome highlighted with a gray box. Primers with mismatches between target sites and SARS-CoV-2 genome are not highlighted, with mismatched underlined.

ACKNOWLEDGEMENTS

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Board on Health Sciences Policy

HOST GENOMICS RESEARCH AND DATA COLLECTION EFFORTS RELATED TO COVID-19

Joint Meeting of the
Roundtable on Genomics and Precision Health
and the
Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats

Virtual Meeting July 1, 2020
Zoom: https://nasem.zoom.us/j/95540901323



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Board on Health Sciences Policy

Host Genomics Research and Data Collection Efforts Related to COVID-19

July 1, 2020

Table of Contents

Agenda 3

Agenda 4

ROUNDTABLE AND STANDING COMMITTEE INFORMATION 7

Roundtable on Genomics and Precision Health 8

Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats 10

MEETING INFORMATION 13

Speaker and Chair Biographies 14

BACKGROUND MATERIALS 17

Online links to background materials 18

Rapid Expert Consultations on the COVID-19 Pandemic 20

A blood-based host gene expression assay for early detection of viral infection 21

AGENDA

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Board on Health Sciences Policy

Host Genomics Research and Data Collection Efforts Related to COVID-19

Joint Meeting
of the
Roundtable on Genomics and Precision Health
and the
Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats

A VIRTUAL MEETING
July 1, 2020
1:00 PM – 2:45 PM ET
Zoom Link: https://nasem.zoom.us/j/95540901323

Aims for the meeting:

- To understand the scientific challenges that the field of genomics is facing
  - What has been the effect of the pandemic on genomics research and data collection and sharing? What is the role of the genomics community during the pandemic? What research is underway? What research still needs to be done?
  - What gaps and challenges in the field is this crisis highlighting? What unique contributions of the field are not being leveraged effectively?
- To explore innovative approaches to overcoming those challenges
  - What new approaches are you using to solve a problem? Could these be applied to the field of genomics? Are there lessons from genomics that are being used in light of the pandemic? What are the opportunities for connecting efforts and data sets (e.g. across institutions, genotype and phenotype data)?
- To discuss opportunities for the Genomics Roundtable and for the Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats to facilitate progress
  - Are there opportunities for the Roundtable and Standing Committee to act that align with strategic priorities?
  - Are the Roundtable and Standing Committee uniquely positioned to address specific challenges?



AGENDA:

1:00 p.m. ET Welcoming Remarks

GEOFFREY GINSBURG, *Roundtable Co-Chair*
Director, Duke Center for Applied Genomics & Precision Medicine
Professor, Medicine, Pathology, and Biomedical Engineering
Duke University Medical Center

MICHELLE PENNY, *Roundtable Co-Chair*
Vice President and Head of Genomics
Goldfinch Bio

HARVEY FINEBERG, *Standing Committee Chair*
President
Gordon and Betty Moore Foundation

1:15 p.m. **An Introduction to the Host Genetics of COVID-19**

MAXIMILIAN MUENKE
CEO
American College of Medical Genetics and Genomics (ACMG)

Understanding Host Genetics and Genomics Related to COVID-19 and Opportunities for Forming Data Sharing COVID-19 Communities

1:25 p.m. SHARON TERRY
Founder and CEO
Genetic Alliance

1:35 p.m. JOYCE TUNG
Vice President, Research
23andMe

1:45 p.m. GEOFFREY GINSBURG, *Roundtable Co-Chair*
Director, Duke Center for Applied Genomics & Precision Medicine
Professor, Medicine, Pathology, and Biomedical Engineering
Duke University Medical Center

1:55 p.m. **Discussion with Roundtable and Standing Committee Members**

2:30 p.m. **Next Steps**

Questions:

- What challenges or gaps were exposed today?
- Is there an area that should be incorporated into the Roundtable's strategic planning or the Standing Committee's work?

GEOFFREY GINSBURG, *Roundtable Co-Chair*
Director, Duke Center for Applied Genomics & Precision Medicine
Professor, Medicine, Pathology, and Biomedical Engineering
Duke University Medical Center

MICHELLE PENNY, *Roundtable Co-Chair*
Vice President and Head of Genomics
Goldfinch Bio

HARVEY FINEBERG, *Standing Committee Chair*
President
Gordon and Betty Moore Foundation

2:45 p.m. Adjourn

ROUNDTABLE AND STANDING COMMITTEE INFORMATION

The National Academies of
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Roundtable on GENOMICS and PRECISION HEALTH

The sequencing of the human genome is rapidly opening new doors to research and progress in biology, medicine, and health care. At the same time, these developments have produced a diversity of new issues to be addressed.

The National Academies of Sciences, Engineering, and Medicine has convened a Roundtable on Genomics and Precision Health (previously the Roundtable on Translating Genomic-Based Research for Health) that brings together leaders from academia, industry, government, foundations and associations, and representatives of patient and consumer interests who have a mutual concern and interest in addressing the issues surrounding the translation of genome-based research for use in maintaining and improving health. The mission of the Roundtable is to advance the field of genomics and improve the translation of research findings to health care, education, and policy. The Roundtable will discuss the translation process, identify challenges at various points in the process, and discuss approaches to address those challenges.

The field of genomics and its translation involves many disciplines, and takes place within different economic, social, and cultural contexts, necessitating a need for increased communication and understanding across these fields. As a convening mechanism for interested parties from diverse perspectives to meet and discuss complex issues of mutual concern in a neutral setting, the Roundtable: fosters dialogue across sectors and institutions; illuminates issues, but does not necessarily resolve them; and fosters collaboration among stakeholders.

To achieve its objectives, the Roundtable conducts structured discussions, workshops, and symposia. Workshop summaries will be published and collaborative efforts among members are encouraged (e.g., journal articles). Specific issues and agenda topics are determined by the Roundtable membership, and span a broad range of issues relevant to the translation process.

Issues may include the integration and coordination of genomic information into health care and public health including encompassing standards for genetic screening and testing, improving information technology for use in clinical decision making, ensuring access while protecting privacy, and using genomic information to reduce health disparities. The patient and family perspective on the use of genomic information for translation includes social and behavioral issues for target populations. There are evolving requirements for the health professional community, and the need to be able to understand and responsibly apply genomics to medicine and public health.

Of increasing importance is the need to identify the economic implications of using genome-based research for health. Such issues include incentives, cost-effectiveness, and sustainability.

Issues related to the developing science base are also important in the translation process. Such issues could include studies of gene-environment interactions, as well as the implications of genomics for complex disorders such as addiction, mental illness, and chronic diseases.

Roundtable sponsors include federal agencies, pharmaceutical companies, medical and scientific associations, foundations, and patient/public representatives. For more information about the Roundtable on Genomics and Precision Health, please visit our website at nationalacademies.org/GenomicsRT or contact Sarah Beachy at (b)(6) or by e-mail at sbeachy@nas.edu.

The National Academy of Sciences, National Academy of Engineering, and National Academy of Medicine work together as the National Academies of Sciences, Engineering, and Medicine ("the Academies") to provide independent, objective analysis and advice to the nation and conduct other activities to solve complex problems and inform public policy decisions. The Academies also encourage education and research, recognize outstanding contributions to knowledge, and increase public understanding in matters of science, engineering, and medicine.

Roundtable on Genomics and Precision Health Membership

Geoffrey Ginsburg, M.D., Ph.D. (Co-Chair) Duke University
Michelle Penny, Ph.D. (Co-Chair) Goldfinch Bio

Naomi Aronson, Ph.D., BlueCross/BlueShield Association

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Karina Bienfait, Ph.D., Merck and Co., Inc.

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Health and Medicine Division

Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats

In response to a request from the Office of Science and Technology Policy (OSTP) and the Office of the Assistant Secretary for Preparedness and Response (ASPR), the National Academies of Sciences, Engineering, and Medicine will convene a standing committee of experts to help inform the federal government on critical science and policy issues related to emerging infectious diseases and other 21st century health threats. The standing committee will include members with expertise in emerging infectious diseases, public health, public health preparedness and response, biological sciences, clinical care and crisis standards of care, risk communication, epidemiology, and regulatory issues, as well as veterinary science, One Health, ethics, and community engagement. The standing committee will provide a venue for the exchange of ideas among federal government agencies, the private sector, and the academic community, as well as other relevant stakeholders.

The standing committee will:

- Stand ready to respond on short notice to requests from the federal government to assess and consider the science and policy implications of an emerging infectious disease or significant public health threat;
- Provide a venue to enable science and policy discussions relevant to the federal government on emerging issues, research, and activities through in-depth knowledge of the sponsor's programs, goals, and objectives;
- Identify opportunities to integrate science into national preparedness and response decision making;
- Explore lessons learned and best practices from previous preparedness and response efforts, and identify opportunities to disseminate that information to a variety of stakeholders;
- Serve as a focal point for national policy discussions by experts and other leaders in the field;
- Consider, identify, and discuss strategies for addressing misinformation; and
- Respond to the federal government's needs for continuing dialog related to strategic planning and program development to address emerging infectious diseases, biosecurity, and public health and medical preparedness.

At the request of the sponsors, the standing committee will be involved in the planning, development, and oversight of related ad hoc activities undertaken by separately appointed committees operating under its auspices.

The standing committee will serve as a focal point for the discussion of scientific, technical, and policy issues relevant to emerging infectious diseases and public health preparedness and response that warrant detailed examination. Topics for discussion with the standing committee may include:

- Technical assistance and/or assessment of response to emerging infectious diseases;
- Availability of and access to information, samples, and other materials to determine the origin and evolution of emerging infectious diseases;
- International coordination and engagement;
- Technical assessment of ecological and evolutionary drivers of disease emergence;
- Approaches to proactive public messaging and strategies to address misinformation;
- Other science and policy issues relevant to emerging infectious diseases and 21st century health threats.

The committee will carry out its charge at its in-person and virtual meetings by gathering evidence from experts, deliberating, and, when necessary, by preparing short reports.

COMMITTEE SPONSORS

White House Office of Science and Technology Policy and HHS Office of Assistant Secretary for Preparedness and Response

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ADDITIONAL INFORMATION

For additional information, please visit https://www.nationalacademies.org/our-work/standing-committee-on-emerging-infectious-diseases-and-21st-century-health-threats

MEETING INFORMATION

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Board on Health Sciences Policy

Host Genomics Research and Data Collection Efforts Related to COVID-19

Joint Meeting
of the
Roundtable on Genomics and Precision Health
and the
Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats

SPEAKER AND CHAIR BIOGRAPHIES

Harvey V. Fineberg, M.D., Ph.D. ***(Standing Committee Chair)***
Dr. Fineberg is the president of the Gordon and Betty Moore Foundation, which fosters path-breaking scientific discovery, environmental conservation, improvements in patient care and preservation of the special character of the San Francisco Bay Area. He previously held the Presidential Chair for 2014-2015 as visiting professor at the University of California, San Francisco. Prior to that, he served as president of the Institute of Medicine from 2002 to 2014 and as provost of Harvard University from 1997 to 2001, following 13 years as dean of the Harvard School of Public Health. He has devoted most of his academic career to the fields of health policy and medical decision-making. His past research has focused on the process of policy development and implementation, assessment of medical technology, evaluation and use of vaccines, and dissemination of medical innovations. Fineberg chairs the board of the Carnegie Endowment for International Peace and serves on the boards of the William and Flora Hewlett Foundation and the China Medical Board. He helped found and served as president of the Society for Medical Decision Making and also served as consultant to the World Health Organization. Fineberg is co-author of the books Clinical Decision Analysis, Innovators in Physician Education and The Epidemic That Never Was, an analysis of the controversial federal immunization program against swine flu in 1976. He has co-edited several books on such diverse topics as AIDS prevention, vaccine safety, understanding risk in society and global health. He has also authored numerous articles published in professional journals. Fineberg is the recipient of several honorary degrees—the Frank A. Calderone Prize in Public Health, the Henry G. Friesen International Prize in Health Research and the Harvard Medal, awarded by the alumni association of the university from which he earned his bachelor's and doctoral degrees.

Geoffrey Ginsburg, M.D., Ph.D. (Roundtable *Co-Chair*)
Dr. Ginsburg is the founding director for the Center for Applied Genomics & Precision Medicine at the Duke University Medical Center and for MEDx, a partnership between the Schools of Medicine and Engineering to spark and translate innovation. His research addresses the challenges for translating genomic and digital information into medical practice and the integration of precision medicine into healthcare. In 2017 he received Duke's Translational Research Mentorship Award and is a finalist in the NIH/BARDA Antimicrobial Resistance Prize.

He is a member of the Advisory Council to the Director of NIH and is co-chair of the National Academies Roundtable on Genomics and Precision Health and is founder and president of the Global Genomic Medicine



Collaborative, a not for profit organization aimed creating international partnerships to advance the implementation of precision medicine. He has recently served as a member of the Board of External Experts for the NHLBI, the advisory council for the National Center for Accelerating Translational Science, the chair of the review for Genome Canada's Large Scale Applied Research Competition in Genomics and Precision Medicine, and the World Economic Forum's Global Agenda Council on the Future of the Health Sector. He is a founder of Predigen Inc and MeTree&You Inc. He was previously Vice President of Molecular Medicine at Millennium Pharmaceuticals, Inc and a faculty member at Harvard Medical School.

Maximilian Muenke, M.D., FACMG
Dr. Muenke joined the American College of Medical Genomics (ACMG) as CEO in 2019. Recognized as a highly acclaimed physician-scientist and dedicated clinical and research mentor, Dr. Muenke has more than three decades of experience to the ACMG, including 10 years on the faculty of the University of Pennsylvania School of Medicine. Since 2000, Dr. Muenke has served as Senior Investigator, Head of the Human Development Section, and Chief of the Medical Genetics Branch at the National Human Genome Research Institute (NHGRI) of the National Institutes of Health (NIH). The focus of his laboratory's research was on the delineation and identification of the underlying causes of craniofacial and related anomalies in humans including holoprosencephaly as well as Muenke syndrome, a relatively common craniosynostosis syndrome. Dr. Muenke is passionate about training the next generation of leaders in the field of genetics and genomic medicine. He has directed medical genetics training since 1994, first at the University of Pennsylvania and then at the NIH, where he was the Director of the NIH Medical Genetics and Genomic Medicine Residency and Fellowship Training Programs.

Michelle Penny, Ph.D. ***(Roundtable Co-Chair)***
Dr. Penny is a Vice President and Head of Genomics at Golfich Bio. Prior to that, she was the Head of Translational Genome Sciences at Biogen. She received her Ph.D. in Genetics from the University of Birmingham, UK in 1993. After a postdoctoral fellowship in the Virology Division at the National Institute of Medical Research, Mill Hill, London, she joined Imperial College London, Department of Medical and Community Genetics where she was a postdoctoral research scientist until taking a lectureship in Human Molecular Genetics in 1998. Her academic research career focused on the study of complex polygenic diseases particularly autoimmune disease and susceptibility to infection. In 2002, Dr. Penny joined the Clinical Pharmacogenomics group at Pfizer in Sandwich, UK, and moved to New London, CT in 2006 to take on the role of Oncology Molecular Medicine Lead until 2009 when she moved to Indianapolis to lead the Pharmacogenomics work at Eli Lilly and Company.

Sharon Terry, M.A.
Sharon Terry is President and CEO of Genetic Alliance, a large network engaging individuals, families and communities to transform health. Genetic Alliance works to provide programs, products and tools for ordinary people to take charge of their health.

As 'just a Mom' with a master's degree in Theology, she cofounded PXE International, a research advocacy organization for the genetic condition pseudoxanthoma elasticum (PXE), in response to the diagnosis of PXE in her two children in 1994. With her husband, she co-discovered the ABCC6 gene, patented it to ensure ethical stewardship in 2000, and assigned their rights to the foundation. She subsequently developed a diagnostic test and conducts clinical trials. She is the author of 140 peer-reviewed papers, of which 30 are clinical PXE studies.

In her focus at the forefront of consumer participation in genetics research, services and policy, she serves in a leadership role on many of the major international and national organizations, including the Precision Medicine Initiative Cohort Advisory Panel, Accelerating Medicines Partnership, Institute of Medicine (IOM) Science and Policy Board, the IOM Roundtable on Translating Genomic-Based Research for Health, the PubMed Central National Advisory Committee, the PhenX scientific advisory board, the Global Alliance for Genomics and Health, the International Rare Disease Research Consortium Executive Committee and as Founding President of EspeRare Foundation of Geneva, Switzerland. Terry is co-founder of the Genetic Alliance Registry and Biobank. She is on the editorial boards of several journals. She led the coalition that was instrumental in the passage of the Genetic Information Nondiscrimination Act. She received an honorary doctorate from Iona College for her

community engagement work in 2006; the Research!America Distinguished Organization Advocacy Award in 2009; and the Clinical Research Forum and Foundation's Annual Award for Leadership in Public Advocacy in 2011. She was named one of FDA's "30 Heroes for the Thirtieth Anniversary of the Orphan Drug Act" in 2013. She is co-inventor of the Platform for Engaging Everyone Responsibly (PEER). PEER received $500,000 from the Robert Wood Johnson Foundation in 2014. PEER undergirds the Community Engaged Network for All (CENA), a PCORnet member funded in 2013 for Phase I and in 2015 for Phase II. She is Co-PI of the PCORnet Coordinating Center and Chair of the PCORnet Engagment Committee.

Joyce Tung, Ph.D.
Joyce joined 23andMe in 2007 and manages the 23andMe research team, which is responsible for consumer health and ancestry research and development, academic and industry collaborations, computational analyses for therapeutics, and new research methods and tools development. While a postdoctoral fellow at Stanford University, Joyce studied the genetics of mouse and human pigmentation. She graduated from Stanford with honors and distinction with a B.S. in Biological Sciences and a minor in computer science, and earned her Ph.D. in Genetics from the University of California, San Francisco where she was a National Science Foundation graduate research fellow.

BACKGROUND MATERIALS

Links to Background Materials

Materials from 23andMe

- The information page for the external recruitment portion of our COVID-19 study
- Blog post on initial launch of our study
- Blog post on expansion of our study to enroll severe cases
- Blog post on preliminary findings

Genomewide Association Study of Severe Covid-19 with Respiratory Failure

BACKGROUND

There is considerable variation in disease behavior among patients infected with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the virus that causes coronavirus disease 2019 (Covid-19). Genomewide association analysis may allow for the identification of potential genetic factors involved in the development of Covid-19.

A Host Transcriptional Signature for Presymptomatic Detection of Infection in Humans Exposed to Influenza H1N1 or H3N2

BACKGROUND

There is great potential for host-based gene expression analysis to impact the early diagnosis of infectious diseases. In particular, the influenza pandemic of 2009 highlighted the challenges and limitations of traditional pathogen-based testing for suspected upper respiratory viral infection.

A crowdsourced analysis to identify ab initio molecular signatures predictive of susceptibility to viral infection

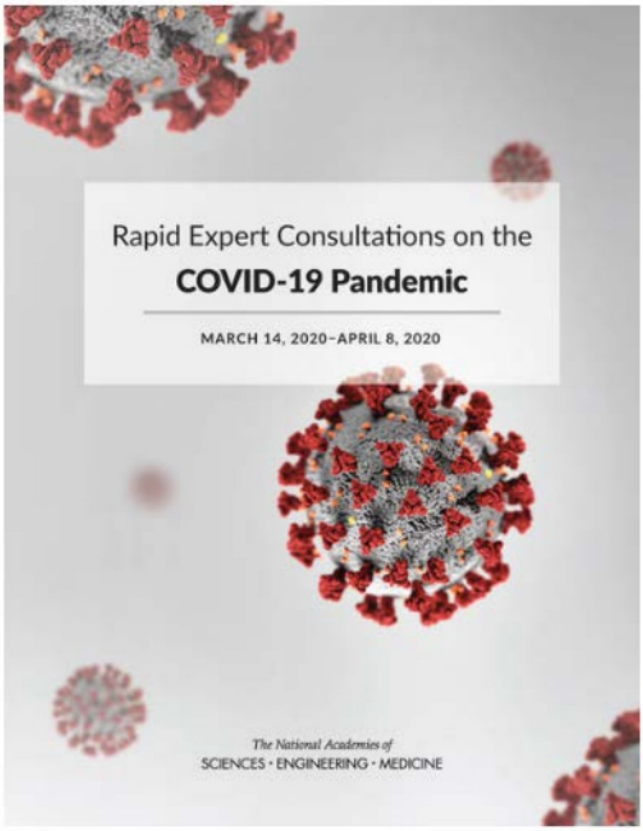
BACKGROUND

The response to respiratory viruses varies substantially between individuals, and there are currently no known molecular predictors from the early stages of infection. Here we conduct a community-based analysis to determine whether pre- or early post-exposure molecular factors could predict physiologic responses to viral exposure.

A Host-Based RT-PCR Gene Expression Signature to Identify Acute Respiratory Viral Infection

Improved ways to diagnose acute respiratory viral infections could decrease inappropriate antibacterial use and serve as a vital triage mechanism in the event of a potential viral pandemic. Measurement of the host response to infection is an alternative to pathogen-based

diagnostic testing and may improve diagnostic accuracy. We have developed a host-based assay with a reverse transcription polymerase chain reaction (RT-PCR) TaqMan low-density array (TLDA) platform for classifying respiratory viral infection.



Rapid Expert Consultations on the COVID-19 Pandemic

In response to a request from the Office of Science and Technology Policy and the Office of the Assistant Secretary for Preparedness and Response, the National Academies of Sciences, Engineering, and Medicine convened a standing committee of experts to help inform the federal government on critical science and policy issues related to emerging infectious diseases and other 21st century health threats. This set of Rapid Expert Consultations are the first of their kind and represent the best evidence available to the Committee at the time each publication was released.

This paper is in press at The Lancet Infectious Diseases

A blood-based host gene expression assay for early detection of viral infection

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Research in context

Evidence before this study

An increasing body of research has demonstrated that host gene expression patterns in peripheral blood offer a high degree of accuracy for diagnosis of acute infections at the time of clinical presentation.

Added value of this study

For the first time we demonstrate in a real-world setting that a blood-based gene expression test can accurately predict impending viral infection even before typical symptoms are present.

Implications of all the available evidence

These data show that biomarkers of viral infection are present and detectable even before clinical disease develops, opening the door to novel approaches to early treatment and outbreak management.

Abstract

Background

Early and accurate identification of persons with viral infections can be of critical importance for clinical management and public health interventions. Measuring host responses through gene signature-based biomarkers offer promise for improved diagnosis of respiratory viral infections, but little is known how early in disease such measures may be effective.

Methods

We performed a modified index-cluster study in 1465 university dormitory residents over five years. Each year, we monitored for symptomatic Index Cases (IC) and identified clusters of their Close Contacts (CC) who would be at presumed increased risk of developing symptomatic respiratory tract infection (RTI) while under observation. We then monitored each CC for 5 days for development of RTI and measured transcriptomic responses at each timepoint via a blood-based 36-gene RT-PCR assay.

Findings

Of 264 ICs with RTI symptoms, 150 (56%) had a confirmed viral etiology detected. Of their 555 CCs, 106 (19%) developed symptomatic RTI with a proven viral etiology during the observation window. The transcriptomic assay accurately predicts infection at the time of peak symptoms despite heterogeneity of hosts and nine different viral etiologies (auROC 0·96). Furthermore, these genomic biomarkers are capable of identifying early disease one, two and three days prior to peak illness when minimal to no symptoms are present (auROC 0·87, 0·86, and 0·77, respectively), and when nearly two-thirds of subjects (62%) are not yet producing detectable viral shedding.

Interpretation

Transcriptional biomarkers accurately predict and diagnose infection across diverse viral etiologies and stages of disease, and may prove useful for guiding administration of early effective therapy, quarantine decisions, and other clinical and public health interventions in the setting of endemic and pandemic infectious disease.

Funding: US Defense Advanced Research Projects Agency

Introduction

Acute viral infections are among the most common causes of acute physician visits in the United States[1]. In a given year, influenza alone affects 5-20% of the population in the US, causes over 400,000 hospitalizations, and as many as 56,000 deaths.[2,3] Outbreaks of viral infection continue to plague modern society, including heavy resource-requiring outbreaks of measles in the developed world, global pandemics like the novel H1N1 outbreak of 2009, or the deadly epidemics of SARS and MERS-CoV.[4-6] However, molecular tools that can contribute to outbreak investigation or directing early clinical management are often lacking. Traditional pathogen-focused diagnostic modalities for viral infection (culture, serology, antigen detection, PCR) offer considerable utility but can also be slow, costly, limited in terms of breadth of pathogen detection, require an *a priori* knowledge of the pathogen to be tested, may not detect emerging strains (as with the 2009 influenza pandemic[7]), or are incapable of distinguishing between active infection and colonization, all of which can minimize the impact of their use. Therefore, there is increasing interest in host-derived biomarkers to aid in the determination of the presence and/or type of infection in at-risk hosts - from single-analyte biomarkers like procalcitonin to composite, multiplex biomarker panels such as those included in a growing number of gene expression-based studies.[8-21] However, the published works on diagnostics for naturally-acquired infection focus on identifying symptomatic subjects at the time of clinical presentation for medical care[19], which is often late in the time course of many viral diseases. Identification of infectious etiologies in earlier, even pre-symptomatic phases of illness, offers the ability to optimize and deliver timely and thus more effective therapy, refine prophylaxis decisions, and guide public health interventions such as isolation and quarantine.[13,22] However, identifying subjects who have been exposed to infectious virus but are not yet exhibiting symptoms of clinical illness is logistically challenging, resulting in a dearth of literature to address these problems.

We have previously shown that during experimental influenza infection of healthy volunteers the host genomic response is robust and detectable well before typical symptoms become apparent.[16,22,23] Other

investigators have confirmed that transcriptomic markers can be indicative of early viral infection in experimental human challenge models as well as animal models of disease.[24-27] However, the mechanism of challenge infection is contrived rather than natural, typically consists of highly egg-adapted viral strains, and the severity of clinical illness does not fully replicate that seen in naturally-acquired infection, all of which can limit the broad applicability of findings from this approach.[16,22,28-31] To date there has been no significant description of the host response to infection during the pre-symptomatic phase of naturally-occurring disease. Here we have utilized a unique index-cluster study design with a focus on serial sampling of 'real world', healthy close contacts of infected individuals to enrich for cases of naturally-occurring infection during these early, post-exposure, but pre-symptomatic time points. This unique design has permitted real-world validation of a transcriptomic signature in peripheral blood that is capable of accurately identifying exposed but apparently healthy individuals who will go on to develop symptomatic viral infection.

Methods

Surveillance Cohort

Over five academic years, we enrolled 1465 Duke University undergraduate students (age 18-24, most of whom live in dormitories on campus) in a modified index-cluster study design (Figure 1). At the time of enrollment, we collected baseline biospecimens and standardized questionnaires to ascertain demographic, behavioral, and other medical characteristics. After enrollment, participating students completed a daily, web-based survey to describe the presence and severity of eight symptoms of RTI (scored 0-4) on a daily basis (see eMethods for details). We monitored the surveys daily for a 6-point increase in a subject's cumulative daily symptom score which would trigger a sample and data collection event for a potential Index Case. Suspected ICs were then assessed for confirmation of illness and biospecimen collection. Using these sentinel cases of RTI suggestive of viral etiology we then identified and monitored a prospective observational cohort of asymptomatic but potentially exposed Close Contacts (people living in the same dormitory or identified by the IC as a Close Contact), who were then monitored and sampled for up to 5 days (the Observation Window).

Sample and data collection

Confirmed IC's provided blood and nasal swab samples at the time of illness identification. Viral etiology was assessed using a commercial multiplex PCR assay (QIAGEN ResPlex II v2·0, Luminex RVP, or Biofire FilmArray RP). Healthy CCs living in proximity to an IC provided blood and respiratory samples for up to five consecutive days and were monitored for symptomatic conversion and viral shedding.

Real-time PCR

Peripheral blood was collected in PAXgene™ Blood RNA tubes (PreAnalytiX), and total RNA extracted using the PAXgene™ Blood miRNA Kit (QIAGEN). cDNA synthesis was performed using SuperScript VILO™ Master Mix (Invitrogen) according to manufacturer's instructions. Real-time PCR was performed using custom TLDA 384-well microfluidic cards with TaqMan Gene Expression Master Mix, and run on a ViiA7 Real-Time PCR System (Applied Biosystems).

Statistical Analysis

For RT-PCR data the Cq values were transformed into normalized relative quantities using reference genes - those with the most stable combined coefficient of variation across all samples - FPGS and TRAP1 were used for the first batch, and DECR1 and TRAP1 were used for the second batch, and data was normalized as previously described.[8,9] We utilized a regularized (2-norm) logistic regression model to discriminate cases from controls (see eMethods for complete details). The data was split into training and testing sets 25 times using repeated random subsampling and each of the 25 models was trained using all timepoints. We evaluated the model's performance separately at each individual timepoint. All model analyses were implemented in Python and Tensorflow.[32]

Results

Brief Description of Study

Healthy first year students at Duke University (n=1465) were enrolled into a surveillance cohort at the beginning of each academic year. After enrollment, we electronically monitored self-reported daily symptoms throughout the duration of the academic year via an online portal. When one or more subjects indicated a change in respiratory symptoms, the sample collection protocol was initiated for individuals (Index Cases, ICs) that met the clinical case definition for acute, community-acquired RTI (see Methods), as well as for a subset of other currently healthy study participants who were in close proximity to the Index Case (Close Contacts, CCs). CCs were monitored for symptoms and serially sampled for serum, whole blood PAXgene RNA, and respiratory samples for 5 days from identification of their Index Case, regardless of symptomatology (Figure 1). This design permitted enrichment for, and collection of, samples from CCs during the timeframe between exposure (which occurred at an unknown time) and subsequent development of symptomatic disease (which occurred under observation).

Prevalence, Transmission, and Clinical Outcomes of Viral Respiratory Tract Infection

We identified 264 clinical ICs, of whom 150 had a confirmed respiratory viral etiology by multiplex PCR testing of NP samples, and 555 CCs, 19% (106) of whom developed symptomatic RTI with concomitant confirmatory viral PCR. Only 57% of the CCs were infected with the same virus as their associated IC. Nine different viral pathogens were detected, and there was marked variation in viral etiologies detected from year to year (Table 1). Infection resulted in many common symptoms of upper respiratory tract infection with clinical variation amongst viral etiologies (Figure 2). For example, symptoms were almost twice as severe in individuals with influenza infection compared to all other viruses (mean symptom score of 20 for influenza vs 13 for all others ($p=0.005$)), although all subjects in the study remained well enough to be managed in the outpatient setting.

Since the exact time of exposure/transmission during naturally-acquired infection is unknown, for analysis CC days were aligned to the time of maximal illness (day of highest symptom score) for each subject (Time "T") in order to attempt to place subjects on broadly similar time scales in terms of the host response to infection. The average total symptom score for all CCs at Time T was 14·8, although this varied by viral etiology (Figures 1,2). For each individual CC, the day before their day of maximal symptoms was termed T-minus 1 ("T-1"), 48 hours prior to symptom max was "T-2" and 72 hours prior to symptom max was time "T-3". At times prior to maximal illness the average total symptom score for CCs was 7·0 (T-1), 4·1 (T-2), and 1·7 (T-3). In our studies a daily symptom score of 6 represents mild clinical illness and was the threshold for defining a 'symptomatic' day. Thus, at days T-2 and earlier, most subjects exhibited subclinical or absent illness (65% asymptomatic at Time T-3, 32% at T-2).

An RT-PCR signature identifies symptomatic subjects with naturally-acquired viral RTI

We previously developed a host response-derived transcriptomic signature of symptomatic viral infection using microarray data[30]. However, microarray and bulk RNA sequencing platforms are impractical for rapid clinical testing. We therefore migrated our prior 'pan-viral' signature to an RT-PCR platform with potential for more direct translation to point-of-care testing [8,9]. Using sparsity-imposing techniques (see eMethods), we selected 36 pre-designed TaqMan probes representing genes comprising the acute respiratory viral signature (and normalization controls) to be used on a TaqMan Low Density Array (TLDA) platform[8]. We then utilized a regularized (2-norm) logistic regression model to determine coefficients for each gene in the signature[33], such that the output of the model is a probability that a given subject will go on to have a symptomatic, viral PCR-positive event during the observation window. The performance of the RT-PCR signature for classification of symptomatic individuals was evaluated using a repeated random subsampling validation strategy on relevant participants across all study years. Overall, the RT-PCR-based host gene expression assay distinguished symptomatic, virus-infected subjects from asymptomatic, viral PCR-negative controls with 92% accuracy (Figure 3). However, accuracy varied depending on viral etiology, ranging from 99% accuracy in influenza infection (where symptoms are

typically more severe) to 92% with Coronavirus and 81% accuracy with metapneumovirus infections (eFigure 1). Interestingly, analysis of individual gene components of the model demonstrates that many individual genes performed similar to the aggregate model across all iterations (eFigure 2-3). In fact, many individual gene targets alone had mean area under Receiver Operating Characteristic (auROC) of >0·90 at the time of maximal illness with the top performing genes at time T being IFIT3 (0·97), HERC5 (0·97), and RSAD2 (0·96)

The gene expression signature identifies naturally-acquired viral RTI during the pre-symptomatic phase.

In order to evaluate performance of the gene signature early in the course of natural infection, we selected CCs with positive microbiology and at least three days of sampling prior to the development of maximal symptoms. As noted previously, the RT-PCR gene signature correctly identified symptomatic subjects with 92% accuracy on the day of maximal symptoms. This decreased to 88% on day T-1 (24 hours before maximal symptoms), and 83% accuracy on day T-2 (48 hours before maximal symptoms) (Figure 3), although performance varies from virus to virus. Only one case of influenza infection had available pre-symptomatic samples at all times, and the signature correctly classified that individual at each timepoint (T-1, T-2, T-3, and T-4). For a fixed FPR of 0·2 (specificity 0·8), the model achieves greater sensitivity than either symptoms alone or viral PCR at all timepoints other than time T and for ICs (where symptom-based diagnosis by definition has a 100% sensitivity, eFigures 4-6). Critically, 62% of subjects who would develop proven viral RTI lacked detectable viral shedding at time T-3, and 44% were still negative for virus by routine clinical testing at day T-2, times where the gene signature outperforms the clinical standards of care - symptom evaluation and viral PCR.

Discussion

The unique design of this community study has allowed us to identify individuals (CCs) at increased risk for developing viral RTI while under observation. This approach has enabled us to demonstrate for the first time that host responses to naturally-acquired respiratory viral infection, as measured by gene expression in peripheral blood, are present well before the time of maximal illness and can be used to predict those at highest risk for developing active infection.

This study demonstrates that conserved genomic responses can be used to correctly classify individuals with a broad array of naturally-acquired respiratory viral infections well before the time when they would present for clinical evaluation, and even before detectable viral shedding begins for the majority of subjects (62%). The promise of this approach is manifested by the discriminative ability of the genomic signature from two and three days prior to maximal illness, when many individuals (65% at Time T-3, 32% at T-2) have no symptoms at all. Even the mild symptoms some subjects do have at early times are clinically vague and would be typical of seasonal allergies, mild COPD flares, or even symptoms due to sequelae of chronic smoking.[34] The potential utility of genomic analysis to classify viral infection among asymptomatic individuals or those with common, nonspecific upper respiratory symptoms is profound. That this holds true across 9 different respiratory viruses, each with its own incubation times and variable clinical progression and duration, also speaks to the potential power of this approach. Additionally, it is important to note that the signature utilized represents conserved underlying antiviral response pathways that are active, and thus discriminatory, across a wide range of illness and not just at early times. This is vital as any real-world use of such a biomarker assay would often be agnostic to where in the course of infection an individual exists at the moment of testing.

While this transcriptomic signature accurately classifies a wide spectrum of viral RTIs, it performs best in a subset of viruses including influenza and coronavirus, where it performs with a high degree of accuracy even during early and pre-symptomatic phases of disease. Influenza is one of the most important viral etiologies to identify early given its importance in pandemics where existing diagnostics may perform

poorly (especially for emerging strains as in 2009[35]), where questions of triage and quarantine are paramount, and where effective therapeutic options are readily available and most effective when given early in disease. Similarly, a number of novel highly virulent coronaviruses, including SARS-CoV and MERS-CoV, have recently arisen and driven global outbreaks, highlighting another area of need for such diagnostics.[5,36].It seems likely that our ability to accurately detect exposure to these types of emerging infections will only become more critical over time. Availability of tools that can aid in assessment of those with potential exposure, especially those that offer diagnostic utility prior to the time of detectable viral shedding, would be a valuable aid to existing epidemiologic and molecular approaches in outbreak settings.

A number of other studies have described interferon response-based transcriptomic signatures of acute respiratory infections which vary by target population, pathogens, modelling approaches, and numbers of genes required to discriminate disease states. [9,10,13-18,30] Some of this work has also been validated, combined, or expanded upon by secondary *ex vivo* analysis of publicly available data. [11,12,37] While the focused signature described here clearly represents a limited subset of all discriminating genes, some observations regarding simplified, IFN-driven signatures can be made. Our data confirm that that some genes from these prior works (such as IFI44L and IFI27[10,38]) both perform well as classifiers distinguishing between viral illness and the healthy state (median auROC = 0·92 and 0·87), being strongly upregulated in cases of viral infection. The data further suggest that a number of other single targets also perform this task well including RSAD2 (0·96), IFIT3 (0·97), and IFI44 (0·93). Based on our experience and the published literature, it seems that relatively simple classification tasks - where the biological responses are highly conserved, unique in character, and of great magnitude - may be reasonably addressed with pauci-analyte biomarker panels.[10,12,39-41] However, as complexity increases by adding more powerful interhost variability (such as immunosuppression), discriminating clinically similar groups with more varied underlying pathophysiology, or discriminating multiple clinical mimics at once,

more intricate biomarker panels will likely be required to maintain high fidelity in real-world settings.[9,14,42]

Our study has several limitations. This project was designed to validate the predictive power of a small set of genes which already had proven discriminatory capability during maximal illness, and thus was not designed to discover whether additional, untested gene sets may offer improved discriminatory performance at early times. Future work with RNASeq on whole blood, single cell sequencing, or other methods will be needed to more completely define early genomic changes in the naturally-infected host. Additionally, our patient cohort was comprised of otherwise young, healthy individuals with community-acquired viral RTI of mild to moderate severity. Therefore, application to patient populations from other age groups, those with immunologic comorbidities, or with more severe viral infections will need to be evaluated. Finally, we did not identify any confirmed infections due to bacterial or other pathogens. As a result, while these transcriptomic markers show great promise for differentiating early infection and pre-symptomatic viral exposure from noninfectious states (which it may mimic at early times), this study did not directly validate the ability of these markers to differentiate viral exposure from early forms of other types of infection[9,10,14,43]. As we move towards bringing transcriptomic biomarkers to the bedside, consideration of broad applicability and performance across a wide array of clinical settings will continue to be paramount.

We have for the first time defined the pre-symptomatic and temporal dynamics of transcriptomic biomarkers characterizing the host response to naturally-acquired viral infections in humans. Clinic-ready platforms capable of operationalizing PCR-based signatures of the size tested herein already exist, some of which are CLIA-approved with point-of-care functionality, offering a proximal pathway to clinical application of these findings. Thus, analysis of the evolution of gene expression-based biomarkers over time shows promise for driving development of diagnostics for early detection of viral exposure and infection which may prove useful for guiding administration of early effective therapy, quarantine

decisions, and other clinical and public health interventions in the setting of endemic and pandemic infectious disease.

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Author contributions

MTM, ELT, GSG and CWW drafted the manuscript, which was critically revised by all remaining authors. MTM, BPN, TB, TV, GSG, and CWW helped conceive and implement the study. All authors helped acquire, analyze, or interpret data. Statistical analyses were performed by FJC, RH, SS, MN, and LP.

Figures

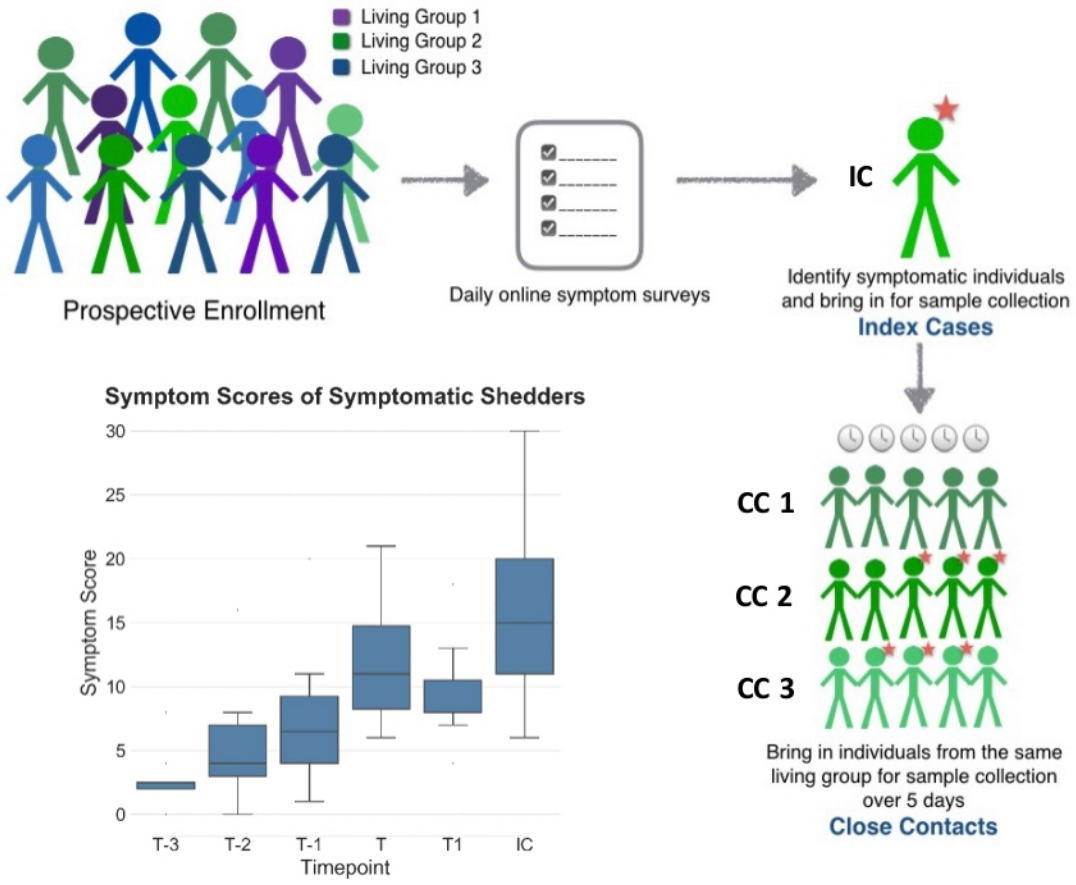

Figure 1: Study design. Students were enrolled at the beginning of the school year. Index Cases were identified when daily symptom surveys indicated potential respiratory illness. This flagged serial sampling of their Close Contacts, some of whom would also develop RTI during the (b)(6) observation window (red stars). This unique model permits blood sampling of some Close Contacts during early and presymptomatic phases of illness. Mean symptom scores of Close Contacts who developed PCR-proven RTI under observation are presented.

A

	Cough	Fever	Headache	Malaise	Nasal Congestion	Nasal Discharge	Sneezing	Sore Throat	Total Symptoms
Influenza*	3.2	1.9	2.4	3.0	2.5	2.4	1.8	2.7	20.0
RSV	2.1	0.7	1.6	2.9	2.6	2.6	1.5	2.6	16.6
Human Metapneumovirus	2.6	0.8	1.5	2.5	2.4	2.5	1.9	2.2	16.3
Rhinovirus	2.2	0.5	1.1	2.1	2.3	2.3	1.5	2.1	14.0
Coxsackie/Echovirus	2.1	0.5	1.1	2.2	2.4	2.4	1.6	1.8	14.0
Coronavirus	1.8	0.7	1.0	2.1	2.3	2.5	1.6	1.8	13.8
Parainfluenzavirus	2.3	0.5	1.2	2.3	1.8	1.7	1.3	2.3	13.4
Adenovirus	2.2	1.2	1.4	2.2	1.6	1.6	0.2	2.2	12.6
Bocavirus	1.0	0.0	0.0	1.0	2.0	2.0	1.0	1.0	8.0

B

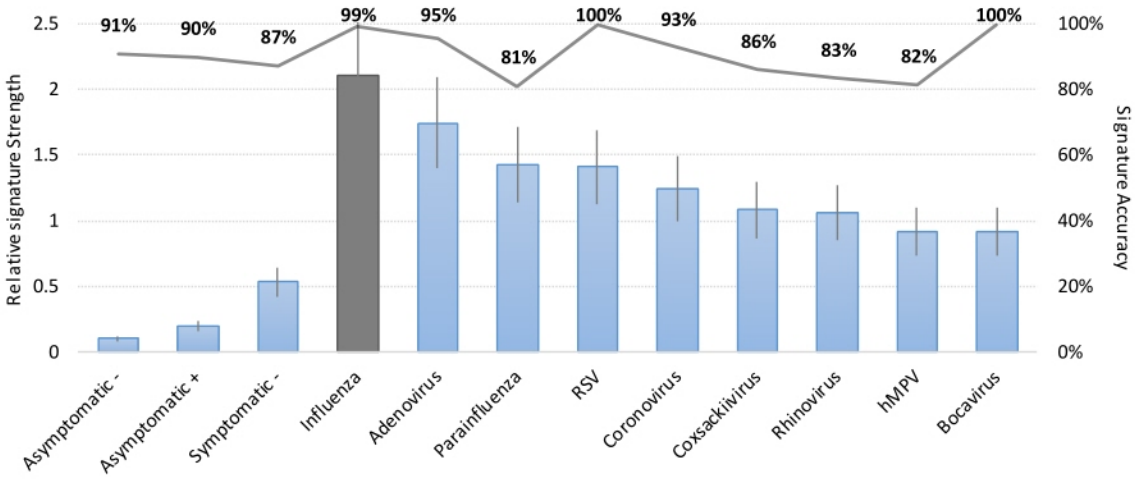

Figure 2. Clinical and genomic variation amongst viruses. For each type of viral infection, mean scores for each symptom measured at the time of maximal illness are presented (Panel A). The relative quantitative transcriptomic signature strength (bars) and accuracy (black line) at the time of maximal illness for each virus, as well as for Asymptomatic nonshedders (-), Asymptomatic shedders (+), and Symptomatic nonshedders (-) are presented in Panel B.

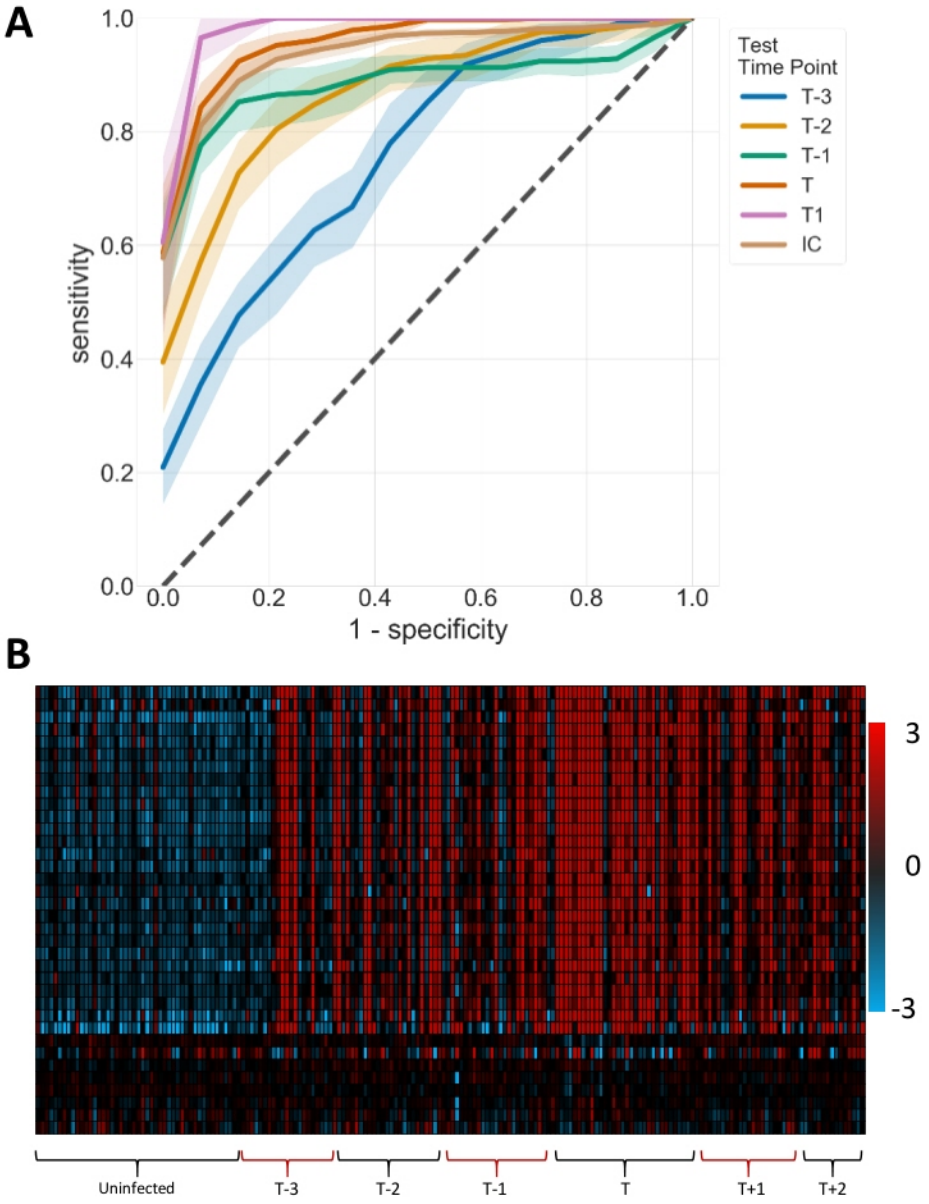

Figure 3: Signature performance across time. An RT-PCR-based 36-gene signature accurately discriminates subjects with naturally-acquired respiratory viral infection at both symptomatic and presymptomatic stages of infection (A). Heatmap of z-scores for each gene at each timepoint in subjects with proven symptomatic viral infection (Panel B), where time 'T' is the day of maximal illness.

Table 1. Viral etiologies of RTI in the Student Cohort

	2009-2010	2010-2011	2011-2012	2013-2014[a]	2014-2015	Total
Adenovirus	1	1	0	1	2	**5**
Bocavirus	0	0	0	0	2	**2**
Coronavirus	11	23	8	3	9	**54**
Coxsackie/Echovirus[b]	21	38	12	0	0	**71**
Human Metapneumovirus	2	1	3	4	1	**11**
Influenza	9	7	1	1	1	**19**
Parainfluenzavirus	3	10	1	0	7	**21**
RSV	3	1	3	0	3	**10**
Rhinovirus	20	23	29	3	37	**112**
Total	**70**	**104**	**57**	**12**	**62**	**305**

[a]truncated season, Spring enrollment only
[b]not present in 2013-2015 testing

Joint Meeting of the Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats, the Forum on Microbial Threats, and the Board on Global Health

Expert Meeting on the Impact of Globalization on Future Health Crises

Friday, September 18, 2020 3:30 p.m. – 5:30 p.m. ET
Virtual Zoom Meeting

Joint Meeting of the Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats, the Forum on Microbial Threats, and the Board on Global Health

Expert Meeting on the Impact of Globalization on Future Health Crises

TABLE OF CONTENTS

• AGENDA	**4**
• LIST OF DISCUSSION TOPICS	**7**
• ROSTERS AND BIOS	**9**
○ Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats	**11**
○ Forum on Microbial Threats	**28**
○ Board on Global Health	**42**
• CONSENSUS STUDY PROPOSAL	**50**

AGENDA

Joint Meeting of the Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats, the Forum on Microbial Threats, and the Board on Global Health

Agenda
Friday, September 18, 2020 3:30 p.m. – 5:30 p.m. ET
Virtual Zoom Meeting

Background: In response to a request from the Office of Science and Technology Policy (OSTP) and the Office of the Assistant Secretary for Preparedness and Response (ASPR), the National Academies of Sciences, Engineering, and Medicine established the Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats. ASPR, in coordination with the Office of the Director of National Intelligence (ODNI) requested an expert meeting on the impact of globalization on future health crises. Given the expertise in both the Forum on Microbial Threats and the Board on Global Health in this topic, this meeting includes members of these bodies. Globalization has improved the world in many ways, but has also introduced fragility in supply chains, adversely affected the environment, witnessed the greatest number of refugees and internally displaced persons, expanded economic disparities, and increased the ability for novel pathogens to spread quickly around the globe. Factors such as extreme weather events and social and economic disruptions, including armed conflict, have also adversely affected global interdependence.

Meeting Objectives

Discuss and consider topics for further exploration on the following:

- What changes are associated with globalization that increase the probability of crises such as pandemics and other 21st century health threats?
- What are potential mitigating factors against these threats?
- How do these different factors interact and create risks of compound events or cascading effects?
- And is there any predictability of how this is changing in frequency or severity?

FRIDAY, SEPTEMBER 18, 2020

CLOSED SESSION

SESSION I **Welcoming Remarks and Sponsors' Description of Need**

3:30 p.m. **Welcome and Charge to the Group**

Harvey Fineberg, *Standing Committee Chair*
President
Gordon and Betty Moore Foundation

3:45 p.m. **Sponsor Remarks**

David (Chris) Hassell
Acting Principal Deputy Assistant Secretary
Senior Science Advisor
The Office of the Assistant Secretary for Preparedness and Response
U.S. Department of Health and Human Services

Kathryn Brinsfield
Senior Advisor
National Counterproliferation Center
Office of the Director of National Intelligence

4:00 p.m. **Topic Introduction**

Harvey Fineberg, *Standing Committee Chair*
President
Gordon and Betty Moore Foundation

Ann Kurth, *Board on Global Health Chair*
Dean and Professor
Yale University, School of Nursing

Peter Daszak, *Forum on Microbial Threats Chair*
President
EcoHealth Alliance

SESSION II **Group Discussions on the Challenges of:**

4:20 p.m. **Prediction Modeling**

Detection Assessment
Surveillance, monitoring, rapid threat assessment that is shared and accessible

Response
Preparedness, near term and focal, long-term and dispersed

5:15 p.m. **Discussion of Next Steps with the Sponsors**

Harvey Fineberg, *Committee Chair*
President
Gordon and Betty Moore Foundation

5:30 p.m. **ADJOURN**

LIST OF DISCUSSION TOPICS

Globalization and 21st Century Health Threats

List of Discussion Topics

Globalization has improved the world in many ways, but has also introduced fragility in supply chains, adversely affected the environment, witnessed the greatest number of refugees and internally displaced persons, expanded economic disparities, and increased the ability for novel pathogens to spread quickly around the globe. Factors such as extreme weather events and social and economic disruptions, including armed conflict, have also adversely affected global interdependence. What are the changes associated with globalization that increase the probability of crises such as pandemics and other infectious disease events? What are the potential mitigating factors against these threats? How do these different factors interact and create risks of compound events or cascading effects? Is there any predictability of how this is changing in frequency or impact?

Significant Factors Related to Increasing Incidence and Consequences of Severe Health Threats

- **Human dimensions:**
  - Increased trade and dependence on foreign supplies that can be disrupted when needed most
  - Local and regional migration patterns that can spread infectious diseases (e.g., Ebola in the DRC)
  - Increased refugees and internally displaced persons
  - Socio-cultural conflicts
  - Urbanization, including sanitation and crowding
  - Rapid population growth into areas unable to support it
  - Spread of misinformation
  - Asynchronous country policies on bioethics and future human health
  - Trends in armed conflict and terrorism
  - Antimicrobial resistance and the emergence of multi-drug resistant organisms
  - Travel that can rapidly spread pathogens
- **Environment:**
  - Extreme weather events enabling emergence of transboundary zoonotic disease
  - Floods, droughts, extreme heat and other impacts of extreme weather events
  - Thawing of the permafrost and resultant environmental and societal disruption
  - Decreased food and water security
- **One Health**
  - Increased intermingling between animals and humans
  - Animal farming practices, particularly surrounding pig products
  - Changing vector and reservoir host range
  - Wet markets and contact with wildlife

Economic and Technology Issues:

- Effects of poverty on underlying health and healthcare access
- Loss of industries and national/regional capabilities
- Technology theft
- Creation of new bioweapons technology

ROSTERS AND BIOS

STANDING COMMITTEE ON EMERGING INFECTIOUS DISEASES AND 21ST CENTURY HEALTH THREATS

Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats

In response to a request from the Office of Science and Technology Policy (OSTP) and the Office of the Assistant Secretary for Preparedness and Response (ASPR), the National Academies of Sciences, Engineering, and Medicine will convene a standing committee of experts to help inform the federal government on critical science and policy issues related to emerging infectious diseases and other 21st century health threats. The standing committee will include members with expertise in emerging infectious diseases, public health, public health preparedness and response, biological sciences, clinical care and crisis standards of care, risk communication, epidemiology, and regulatory issues, as well as veterinary science, One Health, ethics, and community engagement. The standing committee will provide a venue for the exchange of ideas among federal government agencies, the private sector, and the academic community, as well as other relevant stakeholders.

The standing committee will:

- Stand ready to respond on short notice to requests from the federal government to assess and consider the science and policy implications of an emerging infectious disease or significant public health threat;
- Provide a venue to enable science and policy discussions relevant to the federal government on emerging issues, research, and activities through in-depth knowledge of the sponsor's programs, goals, and objectives;
- Identify opportunities to integrate science into national preparedness and response decision making;
- Explore lessons learned and best practices from previous preparedness and response efforts, and identify opportunities to disseminate that information to a variety of stakeholders;
- Serve as a focal point for national policy discussions by experts and other leaders in the field;
- Consider, identify, and discuss strategies for addressing misinformation; and
- Respond to the federal government's needs for continuing dialog related to strategic planning and program development to address emerging infectious diseases, biosecurity, and public health and medical preparedness.

At the request of the sponsors, the standing committee will be involved in the planning, development, and oversight of related ad hoc activities undertaken by separately appointed committees operating under its auspices.

The standing committee will serve as a focal point for the discussion of scientific, technical, and policy issues relevant to emerging infectious diseases and public health preparedness and response that warrant detailed examination. Topics for discussion with the standing committee may include:

- Technical assistance and/or assessment of response to emerging infectious diseases;
- Availability of and access to information, samples, and other materials to determine the origin and evolution of emerging infectious diseases;
- International coordination and engagement;
- Technical assessment of ecological and evolutionary drivers of disease emergence;
- Approaches to proactive public messaging and strategies to address misinformation;
- Other science and policy issues relevant to emerging infectious diseases and 21st century health threats.

The committee will carry out its charge at its in-person and virtual meetings by gathering evidence from experts, deliberating, and, when necessary, by preparing short reports.

COMMITTEE SPONSORS

HHS Office of Assistant Secretary for Preparedness and Response and Office of Science and Technology Policy

COMMITTEE ROSTER

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ADDITIONAL INFORMATION

For additional information, please visit https://www.nationalacademies.org/our-work/standing-committee-on-emerging-infectious-diseases-and-21st-century-health-threats

Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats

COMMITTEE MEMBER BIOSKETCHES

Harvey Fineberg, M.D., Ph.D. ***(Chair)***
President
Gordon and Betty Moore Foundation

Harvey Fineberg is president of the Gordon and Betty Moore Foundation. He previously served as president of the Institute of Medicine from 2002 to 2014 and as provost of Harvard University from 1997 to 2001, following 13 years as dean of the Harvard School of Public Health. Fineberg devoted most of his academic career to the fields of health policy and medical decision-making. His past research has focused on the process of policy development and implementation, assessment of medical technology, evaluation and use of vaccines, and dissemination of medical innovations. Fineberg serves on the boards of the Carnegie Endowment for International Peace and the China Medical Board. He helped found and served as president of the Society for Medical Decision Making, previously served on and chaired the board of the William and Flora Hewlett Foundation, and chaired the committee to review the performance of the World Health Organization and the functioning of the International Health Regulations (2005) during the 2009 H1N1 influenza pandemic. Fineberg is co-author of the books *Clinical Decision Analysis*, *Innovators in Physician Education* and *The Epidemic That Never Was*, an analysis of the controversial federal immunization program against swine flu in 1976. He has co-edited several books on such diverse topics as AIDS prevention, vaccine safety, understanding risk in society and global health. He has also authored numerous articles published in professional journals. Fineberg chaired the National Academies committee that produced the 2019 report on *Reproducibility and Replicability in Science*. He earned his bachelor's and doctoral degrees at Harvard and is the recipient of several honorary degrees.

Kristian Andersen, Ph.D.
Associate Professor and Director of Infectious Disease Genomics, Scripps Research Translational Institute
The Scripps Research Institute

Kristian Andersen is an associate professor in the Department of Immunology and Microbiology at Scripps Research, with joint appointments in the Department of Integrative Structural and Computational Biology, and at the Scripps Research Translational Institute. Over the past decade, his research has focused on the complex relationship between host and pathogen. Using a combination of next-generation sequencing, field work, experimentation, and computational biology he has spearheaded large international collaborations investigating the emergence, spread and evolution of deadly pathogens, including SARS-CoV-2, Zika virus, Ebola virus, West Nile virus, and Lassa virus. His work is highly cross-disciplinary and exceptionally collaborative. Kristian earned his doctoral degree from the University of Cambridge and performed postdoctoral work in Pardis Sabeti's group at Harvard University and the Broad Institute.

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William R. Kenan, Jr. Distinguished Professor
The University of North Carolina at Chapel Hill

(b)(6 (b)(6 is a William R. Kenan, Jr. Distinguished Professor in the Department of Epidemiology at the University of North Carolina. He obtained a Bachelor of Science Degree in Zoology from North Carolina State University in 1977 and a PhD in Microbiology and Immunology from North Carolina State University in 1983. He conducted postgraduate research at the University of Southern California School of Medicine in the department of Microbiology and Immunology between 1983-1986. He is a (b)(6) Weaver Scholar from the National Multiple Sclerosis Society and an Established Investigator Awardee from the American Heart Association. In addition, he is a World Technology Award Finalist, a fellow of the American Association for Microbiology, a senior editor of PloS Pathogen, and a member of the editorial board of several other specialty journals. He was a member of the National Academy Sciences Working Groups that focused on Gene Sequence Methods for Classification of Select Agents and the Risks and Benefits of Gain of Function Research, an invited speaker to the Institute of Medicine Forum on Emerging Infectious Diseases and an invited panelist for the MERS-CoV Stakeholders Workshop. His group has published over 300 papers, many in highly visible journals like PNAS, Nature Medicine, Science, PloS Medicine and PloS Pathogens. The Baric laboratory uses genetic, immunologic, molecular and biochemical approaches to study the molecular mechanisms regulating virus replication, pathogenesis, molecular evolution and cross species transmission using emerging coronaviruses, flaviviruses (Dengue) and noroviruses as model systems. We have pioneered new strategies for developing reverse genetic approaches for manipulating the SARS-CoV, SAR-CoV-2 and MERS-CoV genomes and are actively studying the role of multiple genes that function in cross species transmission, virulence, pathogenesis, viral transcription and RNA fidelity. The Baric laboratory is also identifying key neutralizing epitopes in emerging coronaviruses, dengue and noroviruses using human monoclonal antibodies and structure guided immunogen design to develop broadly active vaccines and immunotherapuetics against these pathogens. Finally, his group has developed novel animal models of human disease and identified dozens of host susceptibility loci that regulate emerging CoV pathogenesis.

Mary Bassett, M.D., M.P.H.
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Mary Bassett is the Director of the FXB Center for Health and Human Rights at Harvard University, as well as the FXB Professor of the Practice of Health and Human Rights at the Harvard School of Public Health. With more than 30 years of experience in public health, Dr. Mary Travis Bassett has dedicated her career to advancing health equity. Prior to her directorship at the FXB Center, Dr. Bassett served for four years as commissioner of Health for New York City. As commissioner, she worked to ensure that every New York City neighborhood supported the health of its residents, with the goal of closing gaps in population health across the city. Originally from New York City, Dr. Bassett lived in Zimbabwe for nearly 20 years. Previously, she was the Program Director for the African Health Initiative and the Child Well-being Program at the Doris Duke Charitable Foundation. She received her B.A. in History and Science from Harvard University and her M.D. from Columbia University's College of Physicians and Surgeons. She served her medical residency at Harlem Hospital Center, and has a master's degree in Public Health from the University of Washington, where she was a Robert Wood Johnson Clinical Scholar.

Trevor Bedford, Ph.D.
Associate Faculty Member, Vaccine and Infectious Disease Division, Public Health Sciences Division, and Human Biology Division
Fred Hutchinson Cancer Research Center

Trevor Bedford is currently Associate Member of the Vaccine and Infectious Disease Division, the Public Health Sciences Division, and the Human Biology Division at the Fred Hutchinson Cancer Research Center. Dr. Bedford uses powerful computers and complex statistical methods to study the rapid spread and evolution of viruses. Data gathered from these processes help researchers develop successful strategies for monitoring and controlling infectious diseases. His visual representations of viral family trees are used to show how the fate of dangerous outbreaks is often determined by the genetics of the infectious agent, human behavior and geography. Dr. Bedford has applied these techniques to document the worldwide spread of seasonal flu viruses. He is developing models to predict which strains of influenza are likely to be most challenging to humans — data that help inform the crucial early decisions about which strains to include in annual flu shots. He specializes in tracking the evolutionary changes of viruses such as HIV and influenza that use RNA, rather than DNA, to carry their genetic information. RNA viruses are much more prone to rapid mutation, which makes many of them particularly nimble at escaping the human immune system and difficult to stop with vaccines. He is a leading advocate for the immediate release of research analyzing viral evolution during epidemics, fresh information that could make a lifesaving difference. He received his Ph.D. in biology from Harvard University.

Georges Benjamin, M.D.
Executive Director
American Public Health Association

Georges Benjamin is well-known as a health leader, practitioner, and administrator. Dr. Benjamin has served as the executive director of the American Public Health Association, the nation's oldest and largest organization of public health professionals, since December 2002. He is a former secretary of Health for the state of Maryland. Dr. Benjamin is a graduate of the Illinois Institute of Technology and the University Of Illinois College Of Medicine. He is board-certified in internal medicine, a Master of the American College of Physicians, a fellow of the National Academy of Public Administration and a fellow emeritus of the American College of Emergency Physicians. He serves on several nonprofit boards such as Research!America, the University of Maryland Medical System and, the Reagan-Udall Foundation. He is a member of the National Academy of Medicine. In April 2016, President Obama appointed Benjamin to the National Infrastructure Advisory Council, a council that advises the president on how best to assure the security of the nation's critical infrastructure.

Donald Berwick, M.D., M.P.P., F.R.C.P., KBE
Lecturer of Health Care Policy
Harvard Medical School

Donald Berwick is president emeritus and senior fellow at the Institute for Healthcare Improvement and former administrator of the Centers for Medicare & Medicaid Services. A pediatrician by background, Dr. Berwick has served on the faculty of the Harvard Medical School and Harvard School of Public Health, and on the staffs of Boston's Children's Hospital Medical Center, Massachusetts General Hospital, and the Brigham and Women's Hospital. He has also served as vice chair of the US Preventive Services Task Force, the first "independent member" of the American Hospital Association Board of Trustees, and chair of the National Advisory Council of the Agency for Healthcare Research and Quality. He served two terms on the Institute of Medicine's (IOM's) Governing Council, was a member of the IOM's Global Health Board, and served on President Clinton's Advisory Commission on Consumer Protection and Quality in the Healthcare Industry. Recognized as a leading authority on health care quality and improvement, Dr. Berwick has received numerous awards for his contributions. In 2005, he was

appointed "Honorary Knight Commander of the British Empire" by Her Majesty, Queen Elizabeth II in recognition of his work with the British National Health Service. Dr. Berwick is the author or co-author of over 160 scientific articles and six books. He currently serves as lecturer in the Department of Health Care Policy at Harvard Medical School.

Richard Besser, M.D.
President and CEO
Robert Wood Johnson Foundation

Richard Besser is president and CEO of the Robert Wood Johnson Foundation (RWJF), a position he assumed in April 2017. Dr. Besser is the former acting director for the Centers for Disease Control and Prevention (CDC), and ABC News' former chief health and medical editor. At RWJF, Dr. Besser leads the largest private foundation in the country devoted solely to improving the nation's health. RWJF's work is focused on building a comprehensive Culture of Health that provides everyone in America with a fair and just opportunity to live the healthiest life possible. In Dr. Besser's role at ABC News, he provided medical analysis and reports for all ABC News programs and platforms. His weekly health chats on social media reached millions. Before joining ABC News in 2009, Dr. Besser worked as director of the Coordinating Office for Terrorism Preparedness and Emergency Response at the CDC. In that role, he was responsible for all the CDC's public health emergency preparedness and emergency response activities. He also served as acting director of the CDC from January to June 2009, during which time he led the CDC's response to the H1N1 influenza pandemic. He is a member of the National Academy of Medicine. He received the Surgeon General's Medallion for his leadership during the H1N1 response, and in 2011 he accepted the Dean's Medal for his contributions to public health from the Johns Hopkins Bloomberg School of Public Health. Dr. Besser received his Bachelor of Arts degree in economics from Williams College and medical degree from the University of Pennsylvania. He completed a residency and chief residency in pediatrics at Johns Hopkins University Hospital in Baltimore.

R. Alta Charo, J.D.
Warren P. Knowles Professor of Law and Bioethics
University of Wisconsin at Madison

R. Alta Charo is a member of the National Academy of Medicine and is the Warren P. Knowles Professor of Law and Bioethics at the University of Wisconsin at Madison (UW), where she is on the faculties of the law and medical schools. She teaches in the areas of bioethics, public health law and biotechnology policy and has served on UW's clinical ethics and research oversight committees. Professor Charo was a member of President Obama's transition team, focusing her attention particularly on transition issues related to NIH, FDA, stem cell policy, and women's reproductive health. From 2009 to 2011 she was on leave to serve as a senior policy advisor on emerging technology issues in the Office of the Commissioner at FDA. Her federal advisory committee service includes the 1994 NIH Human Embryo Research Panel and President Clinton's National Bioethics Advisory Commission (1996 to 2001). At the National Academies she co-chaired (with Richard Hynes) the committee on guidelines for embryonic stem cell research, and has been a member of its Board on Life Sciences, Board on Population Health and Public Health Practice, and Board on Health Sciences Policy.

Peter Daszak, Ph.D.
President and CEO
EcoHealth Alliance

Peter Daszak is President of EcoHealth Alliance, a US-based organization that conducts research and outreach programs on global health, conservation, and international development. Dr. Daszak's research has been instrumental in identifying and predicting the origins and impact of emerging diseases across the globe. He is one of the founders of the field of Conservation Medicine and has been instrumental in the

growth of EcoHealth, One Health, and now Planetary Health. Dr. Daszak is a member of the National Academy of Medicine and Chair of the NASEM's Forum on Microbial Threats. He is a member of the NRC Advisory Committee to the US Global Change Research Program, the Supervisory Board of the One Health Platform, the One Health Commission Council of Advisors, the CEEZAD External Advisory Board, the Cosmos Club, and the Advisory Council of the Bridge Collaborative. He has served on the IOM Committee on global surveillance for emerging zoonoses, the NRC committee on the future of veterinary research, the International Standing Advisory Board of the Australian Biosecurity CRC; and has advised the Director for Medical Preparedness Policy on the White House National Security Staff on global health issues. Dr. Daszak is a regular advisor to WHO on pathogen prioritization for R&D. He received his Ph.D. in parasitic infectious disease from the University of East London.

Jeffrey S. Duchin, M.D.
Health Officer and Chief, Communicable Disease Epidemiology & Immunization Section and Professor in Medicine
Public Health – Seattle & King County, WA and University of Washington, Seattle

Jeffrey Duchin is the Health Officer and the Chief of the Communicable Disease Epidemiology & Immunization Section for Public Health–Seattle & King County, Professor of Medicine in the Division of Infectious Diseases, and Adjunct Professor in the School of Public Health at the University of Washington, Seattle. Jeff currently serves on the Centers for Disease Control & Prevention's (CDC) Board of Scientific Counselors (Office of Infectious Diseases), the CDC's Advisory Committee on Immunization Practices and the Board of Directors for the Infectious Disease Society of America. Jeff has previously been a member of the National Academy of Medicine's (NAM) Forum on Microbial Threats and Forum on Medical and Public Health Preparedness, and the National Quality Forum's Adult Immunization Committee. Jeff received his medical degree from Rutgers Medical School and trained in internal medicine at Thomas Jefferson University Hospital, completed a fellowship in general internal medicine and emergency medicine at the Hospital of the University of Pennsylvania, and did his infectious disease subspecialty training at the University of Washington in Seattle. Jeff is a graduate of the CDC's Epidemic Intelligence Service (EIS) Officer training where he was assigned to the National Center for Infectious Diseases where he also completed the CDC's Preventive Medicine Residency program. Jeff worked for CDC as a medical epidemiologist in the Divisions of Tuberculosis Elimination and HIV/AIDS Special Studies Branch before assuming his current position. His peer review publications and research interests focus on communicable diseases of public health significance. For a complete listing of publications, please see PubMED.

Ellen Embrey
President/CEO
Stratitia, Inc.

Ellen Embrey is President/CEO of Stratitia, Inc., a consulting firm focused on developing meaningful and innovative strategies, and delivering supporting tools and partnerships to bring them successfully to life. Ms. Embrey brings deep expertise in health and medical issues, as well as a wealth of other experience gained during her extensive federal service. In her last federal role, she performed the duties of the Assistant Secretary of Defense for Health Affairs and the Director, TRICARE Management Activity during the presidential transition period in 2009-2010. From 2002 to 2009, Ms. Embrey was the Deputy Assistant Secretary of Defense for Force Health Protection and Readiness, leading significant changes in Department of Defense policies and programs affecting deployment and combat casualty medicine, health promotion and preventive medicine, medical readiness and public health emergency preparedness and response. For 9 months in 2001, Ms. Embrey performed the duties of Assistant Secretary of Defense for Reserve Affairs, shaping policies affecting the readiness and use of the National Guard and Reserve in both federal and state status. From 2000 to 2001, she served as Chief of Staff of that office, and from 1998 to 2001, as Deputy Assistant Secretary of Defense for Military Assistance to Civil Authorities,

developing policies that shaped the role of the National Guard and Reserve components in supporting homeland security, disaster preparedness, and national disaster response capabilities, including advising the president on such matters in the days and weeks following September 11, 2001. Between 1978 and 1997, Ms. Embrey served in senior-level policy analyst, budget analyst, program analyst, management analyst, and systems analyst positions in the Office of the Assistant Secretary of Defense for Reserve Affairs, the Defense Contract Audit Agency, and the Office of Personnel Management. Ms. Embrey was recognized with the Secretary of Defense's Distinguished Civilian Service Award in 2001 and 2004, and twice received the Meritorious Executive Presidential Rank Award in 2006 and 2009.

Baruch Fischhoff, Ph.D.
Howard Heinz University Professor, Department of Engineering and Public Policy
Carnegie Mellon University

Baruch Fischhoff is Howard Heinz University Professor, Department of Engineering and Public Policy and Institute for Politics and Strategy, Carnegie Mellon University (CMU). A graduate of the Detroit Public Schools, he holds a BS (mathematics, psychology) from Wayne State University and a PhD (psychology) from the Hebrew University of Jerusalem. He is a member of the National Academy of Sciences and of the National Academy of Medicine. He is past President of the Society for Judgment and Decision Making and of the Society for Risk Analysis. He has chaired the Food and Drug Administration Risk Communication Advisory Committee and been a member of the Eugene (Oregon) Commission on the Rights of Women, the Department of Homeland Security Science and Technology Advisory Committee and the Environmental Protection Agency Scientific Advisory Board, where he chaired the Homeland Security Advisory Committee. He has received the American Psychological Association (APA) Award for Distinguished Contribution to Psychology, CMU's Ryan Award for Teaching, an honorary Doctorate of Humanities from Lund University, and an Andrew Carnegie Fellowship. He is a Fellow of APA, the Association for Psychological Science, Society of Experimental Psychologists, and Society for Risk Analysis. His books include Acceptable Risk, Risk: A Very Short Introduction, Judgment and Decision Making, A Two-State Solution in the Middle East, Counting Civilian Casualties, and Communicating Risks and Benefits. He has co-chaired three National Academy Colloquia on the Science of Science Communication, as well as its committees on applying decision science to intelligence analysis and its committee on foundational science for cybersecurity.

Diane Griffin, M.D., Ph.D.
Professor, Department of Molecular Microbiology and Immunology
Johns Hopkins Bloomberg School of Public Health

Diane Griffin is University Distinguished Service Professor and Alfred and Jill Sommer Chair of the W. Harry Feinstone Department of Molecular Microbiology and Immunology at Johns Hopkins Bloomberg School of Public Health. Dr. Griffin is a virologist recognized for her work on the pathogenesis of viral infections. She is known particularly for her studies on measles and alphavirus encephalomyelitis that have delineated the role of the immune response in virus clearance, vaccine-induced protection from infection, tissue damage and immune suppression. Dr. Griffin was born in Iowa City, Iowa, and grew up in Oklahoma City. She graduated from Augustana College, Rock Island, Illinois with a degree in biology and from Stanford University School of Medicine in 1968 with a Ph.D. in immunology and M.D., followed by a residency in internal medicine. She was a postdoctoral fellow in virology and infectious diseases at Johns Hopkins University School of Medicine and joined the faculty in 1974. She has been president of the American Society for Virology and of the American Society for Microbiology and is a member of both the National Academy of Sciences and the National Academy of Medicine.

Robert Groves, Ph.D., M.A.
Executive Vice President and Provost
Gerard J. Campbell, S.J. Professor, Math and Statistics Department & Sociology Department
Georgetown University

Robert Groves is the Gerard J. Campbell, S.J. Professor in the Math and Statistics Department as well as the Sociology Department at Georgetown University where he has served as the Executive Vice President and Provost since 2012. Groves is a Social Statistician, who studies the Impact of Social Cognitive and Behavioral Influences on the quality of Statistical Information. His research has focused on the impact of mode of data collection on responses in sample surveys, the social and political influences on survey participation, the use of adaptive research designs to improve the cost and error properties of statistics, and public concerns about privacy affecting attitudes toward statistical agencies. He has authored or co-authored seven books and scores of peer-reviewed articles. His 1989 book, Survey Errors and Survey Costs, was named one of the 50 most influential books in survey research by the American Association of Public Opinion Research. His book, Nonresponse in Household Interview Surveys, with Mick Couper, received the 2008 AAPOR Book Award. His co-authored book, Survey Nonresponse, received the 2011 AAPOR Book Award. He served as the Director of the US Census Bureau between 2009-2012. Groves serves on several boards and advisory committees including the National Research Council Committee on National Statistics, Pew Research Center Board, the National Science Board, and the Federal Economic Statistics Advisory Committee. He is an elected member of the US National Academy of Sciences, of the National Academy of Medicine, of the American Academy of Arts and Sciences, and of the International Statistical Institute.

Margaret Hamburg, M.D.
Foreign Secretary
National Academy of Medicine

Margaret Hamburg is an internationally recognized leader in public health and medicine, and currently serves as foreign secretary of the National Academy of Medicine and chair of the NTI | bio Advisory Group. She is a former Commissioner of the U.S. Food and Drug Administration (FDA), having served for almost six years. As FDA Commissioner she was known for advancing regulatory science, streamlining and modernizing FDA's regulatory pathways, and globalization of the agency. Before joining FDA, Hamburg was founding vice president and senior scientist at the Nuclear Threat Initiative. Previous government positions include Assistant Secretary for Planning and Evaluation, U.S. Department of Health and Human Services, Health Commissioner for New York City, and Assistant Director of the National Institute of Allergy and Infectious Diseases, National Institutes of Health. She is President-elect of the American Association for the Advancement of Science (AAAS), as well as an elected member of the Council on Foreign Relations and the National Academy of Medicine. Hamburg currently sits on the boards of the Commonwealth Fund, the Simons Foundation, the Urban Institute, the Global Alliance for Vaccines and Immunization, the Parker Institute for Cancer Immunotherapy and the American Museum of Natural History. She is chair of the Joint Coordinating Group for the Coalition for Epidemic Preparedness and Innovation, and a member of the Harvard University Global Advisory Council, the Global Health Scientific Advisory Committee for the Gates Foundation, the Harvard Medical School Board of Fellows, and the World Dementia Council. Dr. Hamburg earned her B.A. from Harvard College, her M.D. from Harvard Medical School and completed her medical residency at Weill Cornell Medical Center. She is the recipient of multiple honorary degrees and numerous awards.

(b)(6) M.D.
Vice President, Technical Staff
In-Q-Tel

(b)(6) is Vice President on the Technical Staff at In-Q-Tel, a non-governmental not-for-profit strategic investor focused on enabling technologies to support national security requirements. He is a board-certified emergency physician practicing at Inova Fairfax Hospital, northern Virginia's Level I trauma center, where he led emergency preparedness response efforts in the aftermath of the 9-11 attacks and the anthrax mailings. He participates as a Medical Team Manager for Virginia Task Force One, a FEMA- and USAID-sanctioned international urban search and rescue team and has deployed to numerous catastrophic disaster events, both domestic and international. Dr. (b)(6) currently serves as the co-chair of the National Academies Forum on Medical and Public Health Preparedness and co-chaired the Institute of Medicine committees responsible for developing the work on "crisis standards of care". Dr. (b)(6) is Clinical Professor of Emergency Medicine at George Washington University. He received an AB in political science from Duke University and was awarded his medical degree from Brown University. He completed an internship in internal medicine at the Miriam Hospital in Providence, Rhode Island, and an emergency medicine residency at George Washington/Georgetown University Hospitals.

John Hick, M.D.
Associate Medical Director for EMS
Medical Director of Emergency Medicine
Hennepin County Medical Center

John Hick is a faculty emergency physician at Hennepin Healthcare and a Professor of Emergency Medicine at the University of Minnesota. Dr. Hick serves as the deputy medical director for Hennepin County Emergency Medical Services and Medical Director for Emergency Preparedness at HCMC. He is also the Vice-Chair of the Clinical Council for Life Link III helicopter service and medical director for MN TF-1 state US&R team. He served the Minnesota Department of Health as the medical director for the Office of Emergency Preparedness until becoming an Advisor to the Director of OEM at ASPR/HHS where he is the lead editor for the TRACIE healthcare disaster preparedness website. He is the founder and past chair of the Minneapolis/St. Paul Metropolitan Hospital Compact, a 32-hospital mutual aid and planning group active since 2002. He is a national speaker on hospital preparedness issues and has published numerous papers dealing with hospital preparedness for contaminated casualties, personal protective equipment, crisis standards of care, and surge capacity and was honored to serve the Institute of Medicine on their Crisis Standards of Care projects as well as the Forum on Medical and Public Health Preparedness for Disasters and Emergencies. Dr. Hick holds an M.D. from the Mayo Medical School.

Kent E. Kester, M.D.
Vice President and Head, Translational Science and Biomarkers
Sanofi Pasteur

Kent Kester is currently Vice President and Head, Translational Science and Biomarkers at Sanofi Pasteur. In this capacity, Dr. Kester leads a team of over 200 research and clinical professionals in the US and France focused on the translational development of new vaccines. During a 24-year career in the US Army, he worked extensively in clinical vaccine development and led multiple research platforms at the Walter Reed Army Institute of Research, the U.S. Department of Defense's largest and most diverse biomedical research laboratory with a major emphasis on emerging infectious diseases, an institution he later led as its Commander/Director. His final military assignment was as the Associate Dean for Clinical Research in the School of Medicine at the Uniformed Services University of the Health Sciences (USUHS). During his military service, Dr. Kester was appointed as the lead policy advisor to the US Army Surgeon General in both Infectious Diseases and in Medical Research & Development. In these

capacities, he worked extensively in the interagency environment and developed a variety of Army and DoD medical policies related to infectious diseases, both clinical and research aspects. Dr. Kester holds an undergraduate degree from Bucknell University and an M.D. from Jefferson Medical College, completing his internship and residency in internal medicine at the University of Maryland and a research fellowship in infectious diseases at the Walter Reed Army Medical Center. Currently a member of the US Government Presidential Advisory Council on Combating Antibiotic-Resistant Bacteria (PACCARB) and the Department of Veterans Affairs Health Services Research & Development Service Merit Review Board, he previously chaired the Steering Committee of the NIAID/USUHS Infectious Disease Clinical Research Program, and has served as a member of the FDA Vaccines & Related Biologics Products Advisory Committee (VRBPAC), the NIAID Advisory Council, and the CDC Office of Infectious Diseases Board of Scientific Counselors. He is the Vice Chair of the National Academy of Medicine Forum on Microbial Threats. Board-certified in both internal medicine and infectious diseases, Dr. Kester holds faculty appointments at USUHS and the University of Maryland; and is a fellow of the American College of Physicians, the Royal College of Physicians of Edinburgh, the Infectious Disease Society of America, and the American Society of Tropical Medicine and Hygiene. He is a member of the clinical faculty at the University of Maryland Shock Trauma Center in Baltimore.

Patricia King, J.D.
Professor Emerita
Georgetown University Law Center

Patricia King is Professor of Law emeritus at Georgetown University Law Center and an Adjunct Professor in the Department of Health Policy and Management, School of Hygiene and Public Health at Johns Hopkins University. She is the co-author of Cases and Materials on Law, Science and Medicine. She is a member of the National Academy of Medicine, a member of the American Law Institute, a fellow of the Hastings Center and a faculty affiliate of Georgetown's Kennedy Institute of Ethics. Her scholarship focuses on race and genomics, racial disparities in health and reproductive health. Professor King has served on numerous national advisory bodies formed to address the ethical issues generated by developments in science and technology, including the National Commission for the Protection of Human Subjects of Biomedical and Behavioral Research (1974-78), which produced the seminal "Belmont Report," the President’s Advisory Committee on Human Radiation Experiments (1994-95), the National Institutes of Health’s Embryo Research Panel (co-chair for policy, 1994), the Ethics, Legal and Social Issues Working Group of the NIH’s Human Genome Project (1989-95), and the NIH's Recombinant DNA Advisory Committee (1979-81). She has served on numerous boards and Institute of Medicine committees and is currently a member of the Board of Health Sciences Policy of the National Academies. She is also a Director of Mathematica an employee-owned company.She is a graduate of Wheaton College (Massachusetts) and has served as a Trustee and Chair of the Wheaton College Board of Trustees. In 2018 she was designated a Life Trustee by the Wheaton College Board. She graduated from Harvard Law School and is a past member of the Harvard Corporation the governing board of Harvard University. She has received honorary degrees from Wheaton College, Old Dominion University, and Harvard University.

Jonna Mazet, D.V.M., M.P.V.M., Ph.D.
Executive Director, One Health Institute
UC Davis School of Veterinary Medicine

Jonna Mazet, D.V.M, M.P.V.M., Ph.D., is a Professor of Epidemiology and Disease Ecology at the UC Davis School of Veterinary Medicine and Executive Director of the UC Davis One Health Institute. Her work focuses on global health problem solving for emerging infectious diseases and conservation challenges. She is active in international One Health education, service, and research programs, most notably in relation to disease transmission among wildlife, domestic animals, and people and the ecological drivers of disease emergence. Currently, Dr. Mazet is the Co-Director of the US Agency for

International Development's One Health Workforce – Next Generation, an $85 million educational strengthening project to empower professionals in Central/East Africa and Southeast Asia to address complex health threats, including antimicrobial resistance and zoonotic diseases. She recently served as the Global Director of PREDICT Project, a greater than $200 million viral emergence early warning project under USAID's Emerging Pandemic Threats Division. She was elected to the US National Academy of Medicine in 2013 in recognition of her successful and innovative approach to emerging environmental and global health threats and serves on the National Academies of Science, Engineering, and Medicine's Forum on Microbial Threats and chairs the Academies' One Health Action Collaborative.

Phyllis Meadows, Ph.D., M.S.N., R.N.
Senior Fellow, Health
The Kresge Foundation

Phyllis Meadows currently serves as the Senior Fellow and Program Advisor for the Kresge Foundation Health Team. In this role, she is responsible for supporting the health team in the development and implementation of investment opportunities within and across the Foundation's various programming areas. Her professional career includes leadership roles in philanthropy, academia, community health and governmental public health. She has previously served in the role of Associate Dean for Public Health Practice and Clinical Professor - Health, Management and Policy with the University of Michigan School of Public Health. She has led several initiatives to expand multi-disciplinary practice in communities, designing the University's first certification program on population health and health equity for medical residents. She is currently a Distinguished Towsley Policy Maker in Residence with the University of Michigan's Gerald Ford School of Public Policy. She has taught and developed graduate level and professional continuing education courses to address emerging health issues, including topics on health policy and public health leadership. Dr. Meadows has extensive experience in public health practice having served in various leadership roles in public health. She has held several official appointments in public health leadership at the state, county and local levels. In her most recent appointment, she served as the Chief Health Officer and Director of Health for the City of Detroit, providing leadership for the department of health, environmental health, infectious diseases, child health, clinical and dental services for the residents of Detroit. Her philanthropic experience includes positions as Program Director for the W.K. Kellogg Foundation - Youth, Education and Higher Education; and advisor for several national initiatives of the Robert Wood Johnson Foundation including the Nurse Executive Leadership Program, Partners in Nursing, and the County Roadmaps project. As a registered nurse, she has worked in both community-based health and hospitals. She currently serves as a Board Member and Advisor for several state level organizations and private foundations focusing on health.

Tara O'Toole, M.D., M.P.H.
Executive Vice President
In-Q-Tel

Tara O'Toole currently serves as Executive Vice President at In-Q-Tel. Dr. O'Toole was confirmed as the Under Secretary for Science and Technology (S&T) at the U.S. Department of Homeland Security (DHS) and served from November 4, 2009 to September 23, 2013. From 2003 to November 2009, Dr. O'Toole was the CEO and Director of the Center for Biosecurity at the University of Pittsburgh Medical Center (UPMC), and Professor of Medicine and of Public Health at the University of Pittsburgh. The Center for Biosecurity of UPMC is an independent organization dedicated to improving the country's resilience to major biological threats. Dr. O'Toole is internationally known for her work on biosecurity and on health and safety issues related to the U.S. nuclear weapons complex. Her publications in the biodefense field include articles on the response to anthrax, smallpox, and plague biological attacks; containment of contagious disease epidemics; biodefense research and development strategies; and hospital preparedness. She is the founding editor of the journal Biosecurity and Bioterrorism: Biodefense Strategy, Practice, and Science. She was a principal author and producer of Dark Winter, an influential exercise conducted in

June 2001 to alert national leaders to the dangers of bioterrorist attacks. She was also a principal writer and producer of Atlantic Storm, an international ministerial-level biosecurity exercise held in January 2005. Prior to founding the UPMC in 2003, Dr. O'Toole was one of the original members of the Johns Hopkins Center for Civilian Biodefense Strategies and served as its director from 2001 to 2003. She has served on numerous government and expert advisory committees dealing with biodefense, including panels of the Defense Science Board; the National Academy of Engineering Committee on Combating Terrorism; and the National Academy of Sciences Working Group on Biological Weapons. She served as chair of the Board of the Federation of American Scientists from 2006 to 2007, and in 2006 she was appointed to the board of Google Foundation's International Networked System for Total Early Disease Detection. From 1993 to 1997, Dr. O'Toole served as Assistant Secretary of Energy for Environment Safety and Health. In this position, she was the principal advisor to the Secretary of Energy on environmental protection and on the health and safety of the approximately 100,000 workers in the U.S. nuclear weapons complex and Department of Energy (DOE) laboratories. She developed the first overall management and safety plan for dealing with the highly enriched uranium, plutonium, spent fuel, and radioactive waste left in place when nuclear weapons production was stopped in the early 1990s. She ran the multi-agency, multimillion-dollar task force that oversaw the government's investigations into human radiation experiments conducted during the Cold War and led the U.S. delegation to Russia to establish the U.S./Russia cooperative effort to study radiation exposure and environmental hazards of the Russian nuclear weapons complex. Prior to her work at DOE, Dr. O'Toole was a senior analyst at the Congressional Office of Technology Assessment, where she directed several projects and studies, including the health impact of pollution resulting from nuclear weapons production. She also served as a consultant to industry and government in matters related to occupational and environmental health; worker participation in workplace safety protection; and organizational change. Dr. O'Toole practiced general internal medicine in community health centers in Baltimore from 1984 to 1988. She is board certified in internal medicine and occupational and environmental health. She has a bachelor's degree from Vassar College, an M.D. from the George Washington University, and a Master of Public Health degree from Johns Hopkins University. She completed internal medicine residency training at Yale University and a fellowship in Occupational and Environmental Medicine at Johns Hopkins University.

Alexandra Phelan, S.J.D., LL.M., LL.B.
Assistant Professor
Center for Global Health Science and Security
Georgetown University

Alexandra Phelan is an Assistant Professor at the Center for Global Health Science and Security in the Department of Microbiology and Immunology at Georgetown University School of Medicine. Dr. Phelan also holds an appointment as Adjunct Professor of Law at Georgetown University Law Center. Dr. Phelan works on legal and policy issues related to infectious diseases, with a particular focus on emerging and reemerging infectious disease outbreaks and international law. She has worked as a consultant for the World Health Organization, the World Bank, and Gavi: the vaccine alliance, and has advised on matters including international law and pathogen sharing, human rights law and Zika, intellectual property law, and contract law. She previously worked for a number of years as a solicitor at a firm in Melbourne, Australia and was admitted to practice to the Supreme Court of Victoria and High Court of Australia in 2010. Dr. Phelan's doctorate examined how overlap between fields of international law – in particular, global health law, international human rights law, and international environmental law – can serve as the catalyst to progressively develop international law to prevent and respond to infectious diseases. She also holds a Master of Laws, specializing in international law, from the Australian National University and a Bachelor of Biomedical Science/Bachelor of Laws (Honours) double degree from Monash University. She also holds a Diploma of Languages (Mandarin Chinese). Dr. Phelan is a General Sir John Monash Scholar and was recognized as an Associate Fellow of the Royal Commonwealth Society in 2015 for her human rights advocacy during the 2013-16 Ebola outbreak.

David Relman, M.D.
Thomas C. and Joan M. Merigan Professor in Medicine, and Chief of Infectious Diseases
Stanford University; VA Palo Alto Health Care System

David Relman is the Thomas C. and Joan M. Merigan Professor in Medicine, and Microbiology and Immunology at Stanford University, and Chief of Infectious Diseases at the Veterans Affairs Palo Alto Health Care System. Dr. Relman is also Senior Fellow at the Freeman Spogli Institute for International Studies (FSI), and served as Science Co-Director at the Center for International Security and Cooperation (2013-2017), at Stanford. He is currently director of a new Biosecurity Initiative at FSI. Relman trained at MIT and then Harvard Medical School, followed by clinical training in internal medicine and infectious diseases at the Massachusetts General Hospital in Boston, and then a postdoctoral fellowship in microbiology at Stanford. His early research focused on molecular methods for pathogen discovery and over the past 20 years, on the human microbiome. He was elected to the National Academy of Medicine in 2011. Dr. Relman served as vice-chair of the National Research Council Committee that reviewed the science performed for the FBI 2001 Anthrax Letters investigation, chair of the Forum on Microbial Threats (2007-2017), and is currently a member of the Intelligence Community Studies Board (2016-) as well as Chair of a Standing Committee tasked with examining the health-related problems of US embassy personnel stationed overseas, all at the U.S. National Academies of Science. He was a founding member of the National Science Advisory Board on Biosecurity (2005-2014), a member of the Working Group on Biodefense for the President's Council of Advisors on Science and Technology (The White House) (2016), and served as President of the Infectious Diseases Society of America (2012-2013). He holds an M.D. from Harvard Medical School.

Mark Smolinski, M.D., M.P.H.
President
Ending Pandemics

Mark Smolinski currently serves as President of Ending Pandemics. Dr. Smolinksi brings 25 years of experience in applying innovative solutions to improve disease prevention, response, and control across the globe. Mark is leading a well-knit team—bringing together technologists; human, animal, and environmental health experts; and key community stakeholders to co-create tools for early detection, advanced warning, and prevention of pandemic threats. Since 2009, Mark has served as the Chief Medical Officer and Director of Global Health at the Skoll Global Threats Fund (SGTF), where he developed the Ending Pandemics in Our Lifetime Initiative in 2012. His work at SGTF created a solid foundation for the work of Ending Pandemics, which branched out as an independent entity on January 1, 2018. Prior to SGTF, Mark developed the Predict and Prevent Initiative at Google.org, as part of the starting team at Google's philanthropic arm. Working with a team of engineers, Google Flu Trends (a project that had tremendous impact on the use of big data for disease surveillance) was created in partnership with the U.S. Centers for Disease Control. Mark has served as Vice President for Biological Programs at the Nuclear Threat Initiative, a public charity directed by CNN founder Ted Turner and former U.S. Senator Sam Nunn. Before NTI, he led an 18-member expert committee of the National Academy of Medicine on the 2003 landmark report "Microbial Threats to Health: Emergence, Detection, and Response." (b)(6) served as the sixth Luther Terry Fellow in Washington, D.C., in the Office of the U.S. Surgeon General and as an Epidemic Intelligence Officer with the U.S. Centers for Disease Control and Prevention. (b)(6) received his B.S. in Biology and M.D. from the University of Michigan in Ann Arbor. He is board-certified in preventive medicine and public health and holds an M.P.H. from the University of Arizona.

David Walt, Ph.D.
Hansjörg Wyss Professor of Biologically Inspired Engineering
Harvard Medical School

David R. Walt is the Hansjörg Wyss Professor of Bioinspired Engineering at Harvard Medical School and Professor of Pathology at Harvard Medical School and Brigham and Women's Hospital, is a Core Faculty Member of the Wyss Institute at Harvard University and is a Howard Hughes Medical Institute Professor. Previously, he was University Professor at Tufts University. His laboratory pioneered the development of microwell arrays, which revolutionized the field of genetic analysis. Dr. Walt's laboratory also introduced the idea of digital protein detection by developing a high throughput technology for performing single molecule analysis. Dr. Walt's research is aimed at applying new technologies to address unmet clinical diagnostics needs. Dr. Walt is the Scientific Founder of Illumina Inc., Quanterix Corp., and has co-founded several other life sciences startups including Ultivue, Inc., Arbor Biotechnologies, Sherlock Biosciences, and Vizgen, Inc. He has received numerous national and international awards and honors for his fundamental and applied work in the field of optical microwell arrays and single molecules. He is a member of the National Academy of Engineering, the National Academy of Medicine, a Fellow of the American Academy of Arts and Sciences, a Fellow of the American Institute for Medical and Biological Engineering, a Fellow of the National Academy of Inventors, and is inducted in the US National Inventors Hall of Fame.

FORUM ON MICROBIAL THREATS

FORUM ON MICROBIAL THREATS

BOARD ON GLOBAL HEALTH

HEALTH AND MEDICINE DIVISION

THE NATIONAL ACADEMIES OF SCIENCES, ENGINEERING, AND MEDICINE

The Forum on Microbial Threats was created in 1996 to provide a structured opportunity for discussion and scrutiny of critical—and possibly contentious—scientific and policy issues related to research on and the prevention, detection, surveillance, and responses to emerging, reemerging, and novel infectious diseases in humans, plants, and animals as well as the microbiome in health and disease. Since its creation the topics and issues that have been examined and discussed by the Forum continue to be of major global public health importance. Through public debate and private consultation, the activities of the Forum strive to facilitate discussion and inquiry into the most challenging and cross-cutting sets of challenges within and across the spectrum of "microbial threats."

Activities of the Forum are designed to examine emerging as well as long-standing challenges in microbial ecology in "health" and "disease." The Forum has been instrumental in changing the infectious disease paradigm from "the only good 'bug' is a dead 'bug'" to a more ecologically-informed view of the beneficial contributions of the microbiome in health maintenance and how these microbial communities influence and are influenced by their environmental context. The summary reports of Forum workshops have highlighted and often anticipated some of the most important infectious disease issues of the past decade, including the challenge of emerging fungal diseases and the persistent problem of antimicrobial resistance. Through dissemination to public leaders, private industry, and policymakers, these summary reports have served as useful and timely educational resources and records of these public discussions and deliberations.

Today, the complexities and challenges posed by vector and non-vector borne diseases and the corresponding trends that contribute to their emergence and reemergence continue to confound the world's public health, scientific, medical, pharmaceutical, and policymaking communities. The global vulnerability of human, plant, and animal populations has been increasingly recognized as a challenge not only to personal health, but also to public safety, economic stability and development, and national and international security. The realities of the unrelenting resurgence of once manageable diseases, the emergence of multidrug resistant infectious diseases, the emergence and spread of newly identified pathogens such as Middle East Respiratory Syndrome (MERS), the global challenge of multi-drug resistant microorganisms, the reemergence of vector-borne disease as a major, global, public health concern, and the emergence of the first global influenza pandemic of the 21st century—H1N1—serve as timely reminders of the continuing evolution and adaptation of infectious diseases and their attendant impacts on human, plant, and animal health—domestically and internationally. The activities of the Forum continue to track and anticipate these evolving challenges.

As a result of such cross-sector dialogue, priority issues for infectious disease research and public health policy have been recognized; critical issues warranting further investigation have been identified; and there have been increased opportunities for more effective collaborations and dialogue between the private and public sectors represented on the Forum, as well as between the medical, veterinary, and plant disease communities. The Forum's membership consists of individuals from a wide range of disciplines and organizations in the public and private sectors, including the public health, medical, pharmaceutical, veterinarian, academic science, agricultural, and environmental communities.

Please see the next page for a list of the Forum's current members.

FORUM MEMBERSHIP

CHAIR

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Vice President and Head
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Members of the Forum on Microbial Threats
BIOGRAPHIES



Forum Chair

Peter Daszak, Ph.D., is president of EcoHealth Alliance, a U.S.-based organization that conducts research and outreach programs on global health, conservation, and international development. Dr. Daszak's research has been instrumental in identifying and predicting the impact of emerging diseases across the globe. His achievements include identifying the bat origin of SARS, identifying the underlying drivers of Nipah and Hendra virus emergence, producing the first ever global emerging disease 'hotspots' map, developing a strategy to find out how many unknown viruses exist that could threaten to become pandemic, identifying the first case of a species extinction due to disease, and discovering the disease chytridiomycosis as the cause global amphibian declines. Dr. Daszak is a member and chair-elect of the National Academy of Sciences, Engineering and Medicine's Forum on Microbial Threats. He is a member of the National Research Council (NRC) Advisory Committee to the U.S. Global Change Research Program, the Supervisory Board of the One Health Platform, the One Health Commission Council of Advisors, the Center of Excellence for Emerging and Zoonotic Animal Diseases External Advisory Board, the Cosmos Club, and the Advisory Council of the Bridge Collaborative; he has served on the Institute of Medicine committee on global surveillance for emerging zoonoses, the NRC committee on the future of veterinary research, the International Standing Advisory Board of the Australian Biosecurity Cooperative Research Centres, and has advised the Director for Medical Preparedness Policy on the White House National Security Staff on global health issues. Dr. Daszak is a regular advisor to the World Health Organization (WHO), World Organisation for Animal Heatlh, and the Food and Agriculture Organization of the United Nations, and is actively involved in the WHO Expert group on Public Health Emergency Disease Prioritization. Dr. Daszak won the 2000 Commonwealth Scientific and Industrial Research Organisation medal for collaborative research on the discovery of amphibian chytridiomycosis, is the EHA institutional lead for USAID-EPT-PREDICT, is on the editorial boards of *Conservation Biology*, *One Health*, and *Transactions of the Royal Society of Tropical Medicine & Hygiene*, and is editor-in-chief of the journal *EcoHealth*. He has authored over 300 scientific papers, and his work has been the focus of extensive media coverage, ranging from popular press articles to television appearances.



Forum Vice Chair

Kent E. Kester, M.D., is currently vice president and head of Translational Science and Biomarkers at Sanofi Pasteur. During a 24-year career in the U.S. Army, he worked extensively in clinical vaccine development and led multiple research platforms at the Walter Reed Army Institute of Research, the U.S. Department of Defense's largest and most diverse biomedical research laboratory—an institution he later led as its commander/director. His final military assignment was as the associate dean for clinical research in the School of Medicine at the Uniformed Services University of the Health Sciences (USUHS). Dr. Kester holds an undergraduate degree from Bucknell University and an M.D. from Jefferson Medical College. He completed his internship and residency in internal medicine at the University of Maryland and a fellowship in infectious diseases at the Walter Reed Army Medical Center. A malaria vaccine researcher with over 70 scientific manuscripts and book chapters, Dr. Kester has played a major role in the development of the malaria vaccine candidate known as RTS,S. Currently

a member of the U.S. Government Presidential Advisory Council on Combating Antibiotic-Resistant Bacteria, he previously chaired the Steering Committee of the National Institute of Allergy and Infectious Diseases (NIAID)-USUHS Infectious Disease Clinical Research Program, and has served as a member of the Food and Drug Administration's Vaccines and Related Biologics Products Advisory Committee, the NIAID Advisory Council, and the U.S. Centers for Disease Control's Office of Infectious Diseases Board of Scientific Counselors. Board certified in both internal medicine and infectious diseases, he holds faculty appointments at USUHS and the University of Maryland; and is a fellow of the American College of Physicians, the Infectious Diseases Society of America, and the American Society of Tropical Medicine and Hygiene.



Forum Vice Chair

Rima F. Khabbaz, M.D., is the director of the National Center for Emerging and Zoonotic Infectious Diseases (NCEZID) at the U.S. Centers for Disease Control and Prevention (CDC). From 2010 to 2017, she was CDC's deputy director for infectious diseases and director of the Office of Infectious Diseases, where she helped lead the efforts of CDC's infectious disease national centers and advance the Agency's crosscutting infectious disease priorities including the integration of advanced molecular detection technologies into public health. During that time, she also served on an interim basis as acting director of the National Center for Immunization and Respiratory Diseases, acting director of the National Center for HIV/AIDS, Viral Hepatitis, STD, and TB Prevention, and acting director of NCEZID during leadership transitions. Her previous CDC positions include director of the National Center for Preparedness, Detection, and Control of Infectious Diseases; director, acting director, and associate director for epidemiologic science in the National Center for Infectious Diseases (NCID); and deputy director and associate director for science in the Division of Viral and Rickettsial Diseases. Her first job at CDC was an Epidemic Intelligence Service Officer in NCID's Hospital Infections Program. She later served as a medical epidemiologist in NCID's Retrovirus Diseases Branch, where she made major contributions to defining the epidemiology of the non-HIV retroviruses, specifically human T lymphotropic viruses (HTLV) I and II, in the United States and to developing guidance for counseling HTLV–infected persons. Following the hantavirus pulmonary syndrome outbreak in the southwestern United States in 1993, she led CDC's efforts to set up national surveillance for this syndrome. She also played a key role in developing and coordinating CDC's blood safety and food safety programs related to viral diseases. She has served in leadership positions during many of CDC's responses to outbreaks of new and/or reemerging infections, including Nipah, Ebola, West Nile virus, SARS, and monkeypox, and she led the CDC field team to the nation's capital during the public health response to the anthrax attacks of 2001. Dr. Khabbaz is a graduate of the American University of Beirut in Lebanon, where she obtained both her bachelor's degree in science (biology/chemistry) and her medical doctorate degree. She trained in internal medicine and completed a fellowship in infectious diseases at the University of Maryland in Baltimore. In addition to her CDC position, she serves as clinical adjunct professor of medicine (infectious diseases) at Emory University. Dr. Khabbaz is a fellow of the Infectious Diseases Society of America (IDSA), a member of the American Epidemiological Society, and a member of the American Society for Microbiology and of the American Society for Tropical Medicine and Hygiene. She is a graduate of the Public Health Leadership Institute at the University of North Carolina and the National Preparedness Leadership Initiative at Harvard University. She served on IDSA's Annual Meeting Scientific Program Committee and serves on the society's Public Health Committee. She also is a member of the National Academies of Sciences, Engineering, and Medicine's Forum on Microbial Threats.



Kevin Anderson, Ph.D., serves as a senior program manager in the Department of Homeland Security's (DHS's) Science and Technology Directorate, providing oversight and requirements for biodetection and biodiagnostics systems development for government-wide customers and stakeholders. Since joining DHS in 2003, Dr. Anderson has provided leadership for science program development, laboratory design, and strategic planning; served as a subject matter expert and advisor to the Bioterrorism Risk Assessment and Biological Threat Characterization programs; and has participated in interagency working groups and assessments which provide guidance to medical countermeasure development, a key component of the nation's biodefense strategy. Prior to joining DHS, Dr. Anderson was a principal investigator at the U.S. Army Medical Research Institute of Infectious Diseases, leading research focused on understanding basic mechanisms of viral diseases causing hemorrhagic fever and development of medical countermeasures. He received postdoctoral training in molecular virology at the University of Alabama at Birmingham and the University of North Carolina at Chapel Hill, performing basic research on human respiratory syncytial viruses, and earned Ph.D. and B.S. degrees in microbiology from Montana State University and the University of Maryland, College Park, respectively.

Timothy Burgess, M.D., is Director of the Infectious Diseases Clinical Research Program at Uniformed Services University. He is a graduate of the U. S. Naval Academy and the Indiana University School of Medicine. He trained in Internal Medicine and Infectious Diseases at National Naval Medical Center and Walter Reed Army Medical Center, and completed a Master of Public Health degree as well as a Certificate in Tropical Medicine and Travelers' Health at the Uniformed Services University. His research has included immunopathogenesis and epidemiology of dengue hemorrhagic fever, chikungunya and influenza. He currently oversees a portfolio of clinical observational and interventional studies in the areas of acute respiratory infections, skin and soft tissue infections, trauma-related infections, HIV disease, vector-borne disease and acute enteric infections. He has served as investigator and department head at the Naval Medical Research Center in Silver Spring, MD, and at Naval Medical Research Unit #2 in Jakarta; as director of basic research for the US Military's dengue vaccine development program; and as Chief of the Infectious Diseases Clinical Service at Walter Reed National Military Medical Center, Bethesda. He is a member of the faculty in the Departments of Preventive Medicine & Biostatistics and Medicine, and the Emerging Infectious Diseases interdisciplinary program, at the F. Edward Hébert School of Medicine, Uniformed Services University.

Attending the meeting with the Standing Committee for Dr. Burgess is:

Bernard Okech, Ph.D., M.S., M.P.H., *is a public health entomologist with extensive research and teaching experience in mosquito borne disease transmission. He has come to USU as the deputy director of the MPH program. Prior to joining USU, Dr. Okech was a Research Associate Professor of Environmental and Global Health and also research faculty at the Emerging Pathogens Institute at the University of Florida for more than 12 years. His research is focused on the influence of host and environmental factors on vector borne disease transmission, particularly in developing country environments. Dr. Okech's research program has been supported by funding from the DoD, NIH, USDA, USAID amongst others. Dr. Okech collaborates closely with researchers across the spectrum from local and international universities, US military, USAID, CDC, and Ministry of Health agencies in several in developing countries. These collaborations were particularly useful during the Chikungunya and Zika virus outbreaks that swept the western hemisphere in 2014/2016. During these outbreaks, Dr. Okech's team uncovered novel pathogens in mosquitoes while also providing information about future potential disease outbreaks in the population. Dr. Okech holds a Master of Science from University of Nairobi,*

Doctor of Philosophy from Kenyatta University and Master of Public Health degree from University of California Berkeley.



Marcos A. Espinal, M.D., Dr.P.H., M.P.H., is the director of the Department of Communicable Diseases and Health Analysis at the Pan American Health Organization (PAHO), Regional Office of the World Health Organization (WHO) for the Americas. Dr. Espinal, a national of the Dominican Republic, holds a medical degree from the Universidad Autónoma de Santo Domingo, Dominican Republic (1985). He has an M.P.H. (1990) and a Dr.P.H. (1995) from the University of California at Berkeley School of Public Health. His work experience includes positions in the Ministry of Health of the Dominican Republic and the National Center for Research on Maternal and Child Health; the New York City Public Health Department; and the WHO where he worked for 13 years. Before joining PAHO, Dr. Espinal served as Executive Secretary of the WHO Stop TB Partnership, a global movement aiming at the elimination of TB as a public health problem. Dr. Espinal has published more than 100 peer-reviewed publications in the field of communicable diseases. He is a recipient of the Scientific Prize of the International Union against Tuberculosis and Lung Diseases, the Walter and Elise A. Hass International Award by the University of California at Berkeley for a distinguished record of service in international health, and the Princess Chichibu Memorial Tuberculosis Global Award by the Japan Anti-Tuberculosis Association.



Eva Harris, Ph.D., is a professor in the Division of Infectious Diseases in the School of Public Health and Director of the Center for Global Public Health at the University of California, Berkeley. She has developed a multidisciplinary approach to study the molecular virology, pathogenesis, immunology, epidemiology, clinical aspects, and control of dengue, Zika, and chikungunya, the most prevalent mosquito-borne viral diseases in humans. Specifically, her work addresses immune correlates of protection and pathogenesis, viral and host factors that modulate disease severity, and virus replication and evolution, using in vitro approaches, animal models, and research involving human populations. This has been possible through a close collaboration with the Ministry of Health in Nicaragua for over 28 years. Her international work focuses on laboratory-based and epidemiological studies of dengue, chikungunya, Zika, and influenza in endemic Latin American countries, particularly in Nicaragua, where ongoing projects include clinical and biological studies of severe dengue, a pediatric cohort study of dengue, Zika, chikungunya, and influenza transmission in Managua, a household transmission study of Zika, and a recently concluded cluster-randomized controlled trial of evidence-based, community-derived interventions for prevention of dengue via control of its mosquito vector. She is also directing a study of Zika in infants and pregnancy in Nicaragua and evaluating a number of Zika diagnostic tests with her team in Nicaragua. In 1997, she received a MacArthur Award for work over the previous 10 years developing programs to build scientific capacity in developing countries to address public health and infectious disease issues. This enabled her to found a nonprofit organization in 1998, Sustainable Sciences Institute (SSI; www.sustainablesciences.org), with offices in San Francisco, Nicaragua, and Egypt, to continue and expand this work. Dr. Harris was named a Pew Scholar for her work on dengue pathogenesis. She received a national recognition award from the Minister of Health of Nicaragua for her contribution to scientific development and was selected as a "Global Leader for Tomorrow" by the World Economic Forum. In 2012, she was elected Councilor of the American Society of Tropical Medicine and Hygiene and received a Global Citizen Award from the United Nations Association. She has published over 200 peer-reviewed articles, as well as a book on her international scientific work.



Elizabeth D. Hermsen, Pharm.D., M.B.A., is the head of Global Antimicrobial Stewardship at Merck & Co., Inc. and an adjunct associate professor at the University of Nebraska Medical Center, Colleges of Pharmacy and Medicine, in Omaha, Nebraska. Dr. Hermsen received her Doctor of Pharmacy degree from the University of Nebraska Medical Center followed by a pharmacy practice residency at The Nebraska Medical Center, a fellowship in infectious diseases research at the University of Minnesota College of Pharmacy, and a master's degree in business administration at the University of Minnesota Carlson School of Management with an emphasis in health care industry. Following her fellowship, Dr. Hermsen developed and codirected the antimicrobial stewardship program at The Nebraska Medical Center and subsequently joined Cubist, where she created and led the Antimicrobial Stewardship Outreach Group. Now, in her role at Merck, she is responsible for creating and executing a strategy to advance antimicrobial stewardship through education, implementation, research, and advocacy focused on patient outcomes, population health, and the value of care. When leading the program at The Nebraska Medical Center, Dr. Hermsen developed a publicly-available antimicrobial stewardship website (www.nebraskamed.com/asp) that subsequently was featured in an article regarding top web resources for antimicrobial stewardship (Pagani L, et al. Navigating the Web in Search of Resources on Antimicrobial Stewardship in Health Care Institutions. *Clin Infect Dis*. 2009; 48:626-32.). Dr. Hermsen actively contributed to the advancement of the Society of Infectious Diseases Pharmacists (SIDP) Antimicrobial Stewardship Certificate Program during her term as SIDP president and continues to participate as a lecturer in the program. Dr. Hermsen served as a contributing member of the Antimicrobial Stewardship Knowledge & Skills Collaborative, coordinated by the Society for Healthcare Epidemiology of America; an expert panel on Antimicrobial Practice Improvement in Hospitals, coordinated by the American Society of Health-System Pharmacists; an expert panel on Hospital-based Antimicrobial Utilization Surveillance via the National Healthcare Safety Network, coordinated by the U.S. Centers for Disease Control and Prevention (CDC); the Cardinal Health Infectious Diseases Advisory Group; and an expert panel coordinated by the National Quality Forum and CDC to develop the practical tool entitled, *Antibiotic Stewardship in Acute Care: A Practical Playbook*. Dr. Hermsen is currently co-chair of the Antimicrobial Stewardship Work Package (1A) for the Innovative Medicines Institute Driving Reinvestment in Research & Development and Responsible Antibiotic Use (DRIVE-AB) initiative (www.drive-ab.eu). Dr. Hermsen is a Board Certified Pharmacotherapy Specialist with added qualifications in infectious diseases. She has contributed to the profession with numerous publications in peer-reviewed journals, book contributions, and by serving as a reviewer for several professional journals. Dr. Hermsen has also given over 100 invited presentations at state, regional, national, and international meetings.

Christopher R. Houchens, Ph.D. is the Director of the Division of Chemical, Biological, Radiological and Nuclear Countermeasures within the Biomedical Advanced Research and Development Authority (BARDA), a component of the Office of the Assistant Secretary for Preparedness and Response in the U.S. Department of Health and Human Services. His primary role is building and managing a diverse product portfolio focused on the advanced research, development and procurement of novel vaccines, prophylactics, therapeutics, diagnostics and devices as medical countermeasures against chemical, biological, radiological and nuclear threats. In this role, Dr. Houchens conducts outreach with industry to identify new partnership opportunities and participates in numerous interagency working groups across the US government with the goal of strengthening the ability and capacity of the United States to rapidly respond to naturally occurring and intentionally engineered threats to public health. Dr. Houchens received his Ph.D. in Cell and Molecular Biology from the University of Vermont and continued his training as a Research Fellow at Johns Hopkins Medical Institute and a Staff Scientist at Memorial Sloan-

Kettering Cancer Center. Prior to joining BARDA, Dr. Houchens served as a Senior Scientist at the Defense Advanced Research Projects Agency where he managed translational research and development programs to rapidly design, manufacture and evaluate novel medical countermeasures. Dr. Houchens has over 25 years of experience ranging from early stage disease research to late stage product development and approval which has provided him a thorough understanding of the lifecycle of drug development and the challenges associated with each specific phase of product development.

Michael Mair, M.P.H., serves as the acting assistant commissioner for counterterrorism policy and acting director of the Office of Counterterrorism and Emerging Threats in the Office of the Chief Scientist, U.S. Food and Drug Administration (FDA). In this capacity, Mr. Mair is responsible for providing leadership, coordination, and oversight for FDA's national and global health security and emerging threat portfolios. Mr. Mair also coordinates FDA's Medical Countermeasures Initiative (MCMi), a key component of a broad U.S. government program to improve the U.S. capacity to prepare for and respond to public health emergencies.



Jonna A. K. Mazet, D.V.M., M.P.V.M., Ph.D., earned her doctorate of veterinary medicine, master of preventative medicine, and her Ph.D. in epidemiology from the University of California, Davis. In addition to her faculty appointment in the Department of Medicine and Epidemiology in the UC Davis School of Veterinary Medicine, she serves as the Executive Director of the UC Davis One Health Institute (OHI). Dr. Mazet specializes in emerging infectious diseases and wildlife epidemiology, and as director of OHI, focuses on global health problem solving. In her role at UC Davis, she assists government agencies and the public with emerging health challenges, and is active in international One Health research programs such as tuberculosis in Africa, novel pathogen detection in less developed countries, and pathogen pollution of California coastal waters. Dr. Mazet founded California's Oiled Wildlife Care Network, the premier model wildlife emergency management system worldwide, and remains a consulting expert on wildlife emergency preparedness and response, serving on multiple government and nongovernment organization advisory panels. Dr. Mazet is the principal investigator and global director of the novel viral emergence early warning project, PREDICT, that has been developed with the USAID's Emerging Pandemic Threats Program. She leads a network of global NGOs and governmental agencies to build capacity within the PREDICT-engaged countries to develop surveillance systems and complete the necessary research to halt the next pandemic, like influenza, SARS, Ebola, and HIV that have preceded the program.



Sally A. Miller, Ph.D., is a professor of plant pathology and state extension specialist for vegetable pathology at The Ohio State University, Ohio Agricultural Research and Development Center in Wooster, Ohio. She received her B. Sc. in biology from The Ohio State University (OSU), and M.S. and Ph.D. degrees in plant pathology from the University of Wisconsin-Madison. Prior to joining The Ohio State University faculty in 1991, she was a research manager at Agri-Diagnostics Associates in Cinnaminson, New Jersey, an early developer of plant disease diagnostic assays. Dr. Miller's research is focused on the development of sustainable disease management strategies for conventional and organic vegetable crops, in open field and protected (greenhouse and high tunnel) production systems. This includes diagnosis and management of diseases caused by viruses, bacteria, fungi, oomycetes (water molds), and nematodes. Her lab diagnoses more than 300 vegetable samples from growers and home gardeners each year, at no cost to Ohio residents. Areas of research emphasis are bacterial disease management in processing, fresh market and hydroponic tomatoes, downy mildew and bacterial wilt in cucurbits, soilborne diseases in all vegetable

crops, and food safety. Outreach efforts are directed primarily to vegetable growers and extension educators, and in addition to diagnostics include providing management advice through presentations, fact sheets and other printed information, and social media (websites, blogs, and Twitter). Dr. Miller has been active in long-term international agricultural development projects on integrated pest management (IPM) and plant diagnostics in South and Southeast Asia, Ukraine, West and East Africa, and Central America, primarily under the auspices of the U.S. Agency for International Development (USAID). Current USAID-funded IPM programs are underway in East Africa (Tanzania, Kenya, and Ethiopia) and South/Southeast Asia (Bangladesh, Nepal, and Cambodia). Dr. Miller has been active in the leadership of the American Phytopathological Society, serving as president in 2015-2016. She has recently been appointed to a Food and Agriculture Organization of the United Nations expert panel on antimicrobial resistance, and serves on the executive committee and two working groups for OSU's Global One Health Initiative.



Suerie Moon, Ph.D., M.P.A., is director of research at the Global Health Centre, Graduate Institute of International and Development Studies, Geneva and adjunct lecturer on global health at the Harvard T.H. Chan School of Public Health. She has served on a number of advisory bodies, including most recently the World Health Organization Fair Pricing Forum Advisory Group, Expert Advisory Group to the United Nations Secretary General's High-Level Panel on Access to Medicines, and Proposal Review Committee of UNITAID. Prior to joining the Graduate Institute, she was study director of the Harvard-London School of Hygiene & Tropical Medicine Independent Panel on the Global Response to Ebola, and cofounded and led the Forum on Global Governance for Health, a focal point at Harvard University for research, debate, and strategic convening on issues at the intersection of global governance and health. Her research and teaching focus on global governance, the political economy of global health (focusing on innovation and access to medicines; outbreak preparedness and response; trade, investment, and intellectual property rules; and development assistance for health), the evolution of international regimes, and innovative policies for addressing global problems. She received a B.A. from Yale, an M.P.A. from Princeton, and a Ph.D. from the Harvard Kennedy School of Government.



Rafael Obregon, Ph.D., M.A., provides technical leadership and guidance on the development of standards, guidelines, and quality assurance for the application of communication for development principles and strategies across programmatic areas of the United Nations Children's Fund (UNICEF), including emergency response and humanitarian action. In this capacity, Dr. Obregon has engaged in several responses to public health emergencies and disease outbreaks, including the 2014 – 2015 West Africa Ebola Outbreak. In 2016 Dr. Obregon also served as a member of the Advisory Committee to the World Health Organization's (WHO's) International Health Regulations Emergency Committee on Zika virus and observed increase in neurological disorders and neonatal malformations. Prior to joining UNICEF, he has served as regional advisor for health communication within the Area of Family and Community Health and Child and Adolescent Health Unit at the Pan American Health Organization. Dr Obregon has also been a technical advisor, researcher, and resource/focal person for international/national cooperation agencies and government and nongovernmental organizations, particularly in health and development initiatives. His duties have focused on formative research, project design and evaluation, and capacity strengthening. Dr. Obregon has also been associate professor and guest faculty member at a number of universities, including Ohio University, the Universidad Autónoma in Barcelona, Spain, and the Universidad del Norte in Barranquilla, Colombia, where he remains as an adjunct faculty. Throughout his career, he has written

several books, book chapters, monographs, manuals, peer-reviewed journal articles and reports on public health communication, participatory communication, and capacity development. He is a member of several editorial boards including the *Journal of Health Communication*, and has been a member of several scientific committees including the World Congress on Communication and Development, convened by the World Bank, the Food and Agriculture Organization of the United Nations, and the Communication Initiative, as well as a member of the Technical Advisory Group for the Global Health Communication Partnership within the Center for Communication Programs at Johns Hopkins University. Dr. Obregon earned his Ph.D. in an interdisciplinary program in mass communications, with a concentration on international health, at the College of Communications at Pennsylvania State University in 1999. He received his Master of Arts in international affairs and communication and development from Ohio University in 1994 with a minor in public health. Additionally, he obtained a diploma in education and pedagogy through the National Apprenticeship Service in Colombia in 1990.



Kumanan Rasanathan, M.B.Ch.B., M.P.H., a public health physician with 20 years of experience in health and related sectors. He is a member of the board of Health Systems Global and currently works in the areas of health systems and maternal and child health in Cambodia. He was previously chief, Implementation Research Unit and Delivery Science Unit and Senior Adviser Health for United Nations Children's Fund in New York, working on implementation research focused on improving child service delivery, universal health coverage, district health system strengthening, health systems resilience post-Ebola, integrated community case management, the Sustainable Development Goals agenda, and multisectoral approaches to child health. Prior to this, Dr. Rasanathan worked for the World Health Organization in Geneva on primary health care and the social determinants of health, and in a number of different countries as a clinician, researcher, policy maker, program manager, and advocate. He started his public health career running Phase I and II vaccine clinical trials leading to the licensure and rollout of meningococcal B vaccine in New Zealand.



Gary A. Roselle, M.D., is the national director, Infectious Diseases Service for the Veterans Health Administration of the Department of Veterans Affairs (VA). Dr. Roselle is board certified in internal medicine and infectious diseases and a professor of medicine in the Department of Internal Medicine, Division of Infectious Diseases at the University of Cincinnati College of Medicine. Dr. Roselle serves on multiple national VA and Federal Interagency Committees. He serves as the VA representative on the U.S. Centers for Disease Control and Prevention's (CDC's) Advisory Council for the Elimination of Tuberculosis, CDC's Public Health Action Plan to Combat Antimicrobial Resistance Task Force, CDC's Healthcare Infection Control Practices Advisory Committee, and the Department of Health and Human Services' Steering Committee for the Prevention of Healthcare-Associated Infections, and the National Academies of Sciences, Engineering, and Medicine's Forum on Microbial Threats.

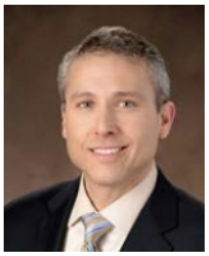


Peter A. Sands, M.P.A., is the executive director of The Global Fund to Fight AIDS, Tuberculosis, and Malaria. Since June 2015, Mr. Sands has been a research fellow at Harvard University, dividing his time between the Mossavar-Rahmani Center for Business and Government at Harvard Kennedy School and the Harvard Global Health Institute, part of the Harvard T.H. Chan School of Public Health, and working on a range of research projects in financial markets and regulation, fintech, and global health. Mr. Sands' engagement with global health issues includes: chairing the U.S. National Academy of Medicine's Commission on a Global Health Risk

Framework for the Future, which in January 2016 produced the highly influential report *The Neglected Dimension of Global Security: A Framework to Counter Infectious Disease Threats*; chairing the World Bank's International Working Group on financing preparedness, which in May 2017 published *From Panic and Neglect to Investing in Health Security: Financing Preparedness at a National Level*; authoring several papers on infectious disease crises in the *New England Journal of Medicine*, *The Lancet*, and *British Medical Journal*; being the lead non-executive director between 2011-2017 on the Board of the UK's Department of Health, which provides oversight and policy direction to the UK's National Health Service; and being an active member on both the U.S. National Academy of Science's Committee on Ensuring Access to Affordable Drugs and the Forum on Microbial Threats. Mr. Sands is a board member or advisor to several startups in the fintech and meditech arenas, such as Noble Markets (US) and Cera (UK). He was group chief executive of Standard Chartered PLC from November 2006 to June 2015. He joined the Board of Standard Chartered PLC as group finance director in May 2002, responsible for finance, strategy, risk and technology, and operations. Prior to this, Mr. Sands was a senior partner at worldwide consultants McKinsey & Co. Before joining McKinsey, he worked for the UK's Foreign and Commonwealth Office. He has served on various boards and commissions, including as a director of the World Economic Forum and co-chairman of Davos, governor of the UK's National Institute for Economic and Social Research, member of the International Advisory Board of the Monetary Authority of Singapore, member of the Browne Commission on Higher Education Funding in the UK, member of the China People's Association for Friendship with Foreign People's Global CEO Council, co-chair of the UK-India CEO Forum, board director of the Institute of International Finance, chairman of the International Monetary Conference, member of the International Advisory Board of Lingnan University, China, and trustee of the Camden Roundhouse, London. Mr. Sands graduated from Brasenose College, Oxford University with a first class degree in politics, philosophy, and economics. He also received a Master in Public Administration from Harvard University, where he was a Harkness Fellow. Mr. Sands, who grew up in Singapore and Malaysia, is married to author and bookshop owner, Betsy Tobin and has four children.



Thomas W. Scott, Ph.D., is a distinguished professor of mosquito-transmitted disease ecology and epidemiology at the University of California, Davis. He received his Ph.D. in ecology from the Pennsylvania State University and was a postdoctoral fellow in epidemiology at the Yale School of Medicine. After initially examining the relationship of mosquito ecology to pathogen transmission in Southeast Asia, Latin America, and Africa in the early 1990s, in an effort to strengthen the public health connection of his work, he began longitudinal dengue epidemiological studies in Thailand and Peru. He currently focuses on assessment of recommendations for mosquito-borne disease prevention, testing assumptions in public health policy, and developing innovative, cost-, and operationally-effective concepts for disease prevention. At UC Davis, he was director of the Center for Vector-Borne Disease Research and the Davis Arbovirus Research Unit. He chaired the Mosquito-Borne Disease Working Group in the Research and Policy in Infectious Disease Dynamics (RAPIDD) program, which developed novel mathematical modeling frameworks and reported to the Science and Technology Directorate of the Department of Homeland Security, and Fogarty International Center at the National Institutes of Health. At the World Health Organization, he chairs the Vector Control Advisory Group, which assesses the public health value of new product classes in vector control; has co-chaired the Global Vector Control Response, which aims to reduce the global burden of vector-borne diseases through locally adapted sustainable vector control; chaired the Emergency Response Consultation for Zika Virus; and serves on the International Health Regulators Roster of Experts. He is a member of the Management Committee for the Global Dengue and Aedes-transmitted Diseases Consortium.



Alan Tennenberg, M.D., M.P.H., is the Chief Medical Officer of Johnson & Johnson Global Public Health. Trained as an infectious diseases physician, Alan brings over 25 years of experience in the pharmaceutical industry, clinical medicine, public health, and academia. As Chief Medical Officer, he is responsible for building strategic relationships with key stakeholders and partners in government, academia, multi-lateral institutions, and non-governmental organizations around the world. He is a leading figure in facilitating private sector engagement to advance the Global Health Security Agenda.

Matthew Zahn, M.D., currently serves as medical director of the Division of Epidemiology and Assessment for the Orange County Health Care Agency. Dr. Zahn received his doctorate in medicine from St. Louis University School of Medicine. From 2004 through 2011, he served as medical director for the Louisville Metro Department of Public Health and Wellness. During that time, he also served as an assistant professor of pediatric infectious disease at the University of Louisville School of Medicine. Dr. Zahn has served on multiple national public health committees, including his current service as the chair of the Infectious Diseases Society of America's Public Health Workgroup.

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Center for Innovation in Global Health
Stanford University

Sarah Cleaveland, Ph.D.
Institute of Biodiversity, Animal Health and Comparative Medicine
University of Glasgow

Malick Diara, M.D., M.B.A., M.P.H.
Public Health Manager
Workplace Wellness & Infectious Disease Control Programs
Global Medicine and Occupational Health Department
ExxonMobil Corporation

Isabel Garcia, D.D.S., M.P.H.
Dean
University of Florida College of Dentistry

Lawrence O. Gostin, J.D.
University Professor and Founding Linda and Timothy O'Neill Professor of Global Health Law
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Biographies

Ann E. Kurth, Ph.D., CNM, M.P.H., FAAN is Dean, and Linda Koch Lorimer Professor, Yale University School of Nursing. Dr. Kurth is an elected Fellow of the Institute of Medicine (National Academy of Medicine) and a member of the 2014-2018 US Preventive Services Task Force, which sets screening and primary care prevention guidelines for the United States. Dr. Kurth is 2018-2020 chair of the Consortium of Universities for Global Health. An epidemiologist and clinically-trained nurse-midwife, Dr. Kurth's research focuses on HIV/reproductive health and global health system strengthening. Her work has been funded by the National Institutes of Health (NIAID, NIDA, NIMH, NICHD), the Bill & Melinda Gates Foundation, UNAIDS, CDC, HRSA, and others, for studies conducted in the United States and internationally. Dr. Kurth has consulted for the NIH, Gates Foundation, WHO, USAID and CDC, among others. Dr. Kurth has published over 200 peer-reviewed articles, book chapters, and scholarly monographs and presented at hundreds of scientific conferences and invited talks. Dr. Kurth has received awards for her science and leadership including the Friends of the National Institute of Nursing Research Ada Sue Hinshaw Research Award and the International Nurse Researcher Hall of Fame award from Sigma Theta Tau International.

Kelly Baker, Ph.D. is an Assistant Professor in Occupational and Environmental Health, and Epidemiology at the University of Iowa's College of Public Health, and is a faculty member of the interdisciplinary Sustainable Water Development Program in the College of Engineering. Her research focuses on understanding how unsafe water, sanitation and hygiene (WASH) conditions affect maternal and child health outcomes in low and middle income countries. Her Social Microbes program uses an microbial ecology systems approach to understand the complex behavioral, environmental, and spatial-temporal mechanisms that result in young children being exposed to and infected by dozens of types of common enteric pathogens in unsanitary settings. A fundamental theory underlying this research is that in highly unsanitary settings where multiple types of enteric pathogens are dynamically transmitted between humans and animal populations over space and time, the distribution of transmission risks are characterized at the simplest level by presence and concentration of a specific pathogen and at the most complex level by simultaneous exposure to multiple types of pathogens. Dr. Baker's group uses microbial exposure assessment and epidemiology approaches to identify and rank priority transmission pathways, as well as to evaluate the impact of WASH intervention trials. Other research in the Baker Group focuses on documenting how social and environmental barriers in meeting daily WASH needs affects women's reproductive health, with a particular focus on the epidemiology of preterm birth and low infant birth weight outcomes in pregnant women. Dr. Baker has advised the World Bank, World Health Organization, UNICEF and several NGOs on issues related to water quality, priority enteric pathogens, and infectious disease exposure assessment and epidemiology.

Michele Barry, M.D., is Senior Associate Dean for Global Health and Director of the Center for Innovation in Global Health at Stanford University. She is a past President of The American Society of Tropical Medicine and Hygiene and has also served on the advisory board of NIH -Fogarty Center. Areas of scholarly interest include global health workforce, clinical tropical medicine, emerging infectious

diseases, problems of underserved populations and globalization's impact upon health in the developing world. Dr. Barry is an elected member of the National Academy of Medicine.

Sarah Cleaveland. Ph.D., is a Professor of Comparative Epidemiology at the University of Glasgow, UK Institute of Biodiversity, Animal Health and Comparative Medicine. She is a veterinary epidemiologist and leads a research program investigating zoonotic and livestock diseases in Africa. Her research focuses on understanding the burden, epidemiology and control of zoonoses and has generated evidence to support the feasibility of canine rabies elimination. Professor Cleaveland is a member of the National Academy of Medicine.

Malick Diara, M.D., M.B.A., M.P.H., joined ExxonMobil in 2009 with more than 20 years of experience in international health. He is the Public Health Manager of the Exxon Mobil Corporate Medicine and Occupational Health (MOH) Department. As a member of the MOH leadership team, his responsibilities are focused on infectious disease prevention and control in ExxonMobil workplaces, and recently, on management oversight of the Company Culture of Health Program. His illustrative accomplishments include the successful establishment of the Company infectious disease outbreak management program with no operation disruption since 2010, no death due to malaria for the past 10 years with the development of a malaria drug field test that saved over 1.6 USD million within 3 years on lab costs and the launch of a global Tuberculosis control program with over 1000 TB cases averted since 2010. Prior to ExxonMobil and being based in Houston since 2009, Dr. Diara worked with private Non Profit organizations in Washington DC for 9 years and in West Africa for 12 years. With funding from USAID, the French Cooperation or the European Union, and in partnership with local authorities and organizations such as UNICEF and WHO, he supported the design, implementation and evaluation of global, national or local public health programs. Malick is a physician with a Medical Doctorate from Dakar School of Medicine in Senegal, a Master's in Business Administration from the Paris School of Business – Institut Superieur de Gestion and a Masters in Public Health from Tulane University, Louisiana.

Isabel Garcia, D.D.S., M.P.H., joined the University of Florida College of Dentistry as dean on Feb. 16, 2015, after retiring from the U.S. Public Health Service in 2014 as a rear admiral lower half. Garcia's career spans 37 years in public health, clinical practice, research, teaching and administration at the local, state and national levels. Garcia joined the National Institute of Dental and Craniofacial Research at the NIH in 1995 and held multiple leadership roles during her time there. She led NIDCR's science transfer efforts, directed the Institute's Office of Science Policy and Analysis, and served as acting NIDCR director from 2010-2011. Garcia also served as the institute's coordinator for global health and directed NIDCR's Residency in Dental Public Health program from 2005 to 2014. While with the USPHS, Garcia was deployed to help prepare a major health diplomacy mission to Central and South America, which provided care to over 85,000 people in 12 countries.
As deputy director of NIDCR from 2007-2014, she shared responsibility for the oversight and management of programs and functions within the institute — which included a staff of more than 400 scientists and administrators dedicated to research, training, science policy, health education, communications and financial management. Garcia received a doctorate in dental surgery in 1980 from Virginia Commonwealth University and a master's degree in public health from the University of Michigan in 1988. She subsequently completed a residency in dental public health at the University of Michigan and a fellowship in primary care policy from the U.S. Public Health Service. A fellow of the American College of Dentists and the Pierre Fauchard Academy, Dr. Garcia is a diplomate and Past President of the American Board of Dental Public Health and an active member of the American Dental Education Association, the International Association for Dental Research, and the American Dental Association.

Lawrence Gostin, J.D., is University Professor (Georgetown University's highest academic rank) and the Founding Linda and Timothy O'Neill Professor of Global Health Law at Georgetown University, Professor of Public Health at Johns Hopkins University, Director of the O'Neill Institute on National and Global Law, and Director of the WHO Collaborating Center on Public Health Law and Human Rights at the Georgetown University Law Center. He is a global correspondent and contributing writer for JAMA, the journal of the American Medical Association. In 2016, President Obama appointed him to a six-year term on the National Cancer Advisory Board.

Andrew Kanter, M.D., M.P.H., FACMI, is Assistant Professor of Clinical Biomedical Informatics and Clinical Epidemiology at Columbia University. Interested in application of ICT to health in the developing world, he has worked or traveled in more than 50 countries. Prior to joining Columbia, he recently spent 12 years with a private medical informatics company where he helped develop the Healthmatics EHR now being sold by Allscripts in addition to providing medical terminology and consulting services to other electronic medical record companies. He is currently appointed to Columbia University full-time in the Departments of Biomedical Informatics (College of Physicians & Surgeons) and Epidemiology (Mailman School of Public Health) and previously supported the development and implementation of the Millennium Villages Global-Network (MVG-Net) for the Millennium Villages Project (MVP). He directs the Columbia International eHealth Lab (CIEL) in the Department of Biomedical Informatics which supports eHealth work around the world. His work focuses on bringing real-world solutions to resource-poor settings to help them achieve the Millennium Development Goals.

Karestan Koenen, Ph.D., does research and teaches about trauma and posttraumatic stress disorder (PTSD). The broad goal of her work is three-fold. First, she studies why, when exposed so a similar traumatic event, some persons develop PTSD while others are resilient. She is particularly interested in how genes shape risk for PTSD. Much of this work is done through the PTSD working group of the Psychiatric Genomics Consortium that she co-leads with Kerry Ressler and Israel Liberzon. Second, she investigates how trauma and PTSD influence weight gain and alter long-term physical health including chronic diseases such as cardiovascular disease and type-2-diabetes. Third, she documents global burden of trauma and PTSD through her work with the World Mental Health Surveys. Dr. Koenen also advocates for victims of sexual violence. In May 2011, Dr. Koenen testified before the House Foreign Affairs Full Committee about the epidemic of sexual violence and victim blaming culture of the Peace Corps. She has written for the Boston Globe, the Washington Post, the Huffington Post, and the Women's Media Center's Women Under Siege Project, a journalism project founded by Gloria Steinem that investigates how rape and other forms of sexualized violence are used as tools in conflict. Dr. Koenen also consulted with award-winning documentary filmmaker Lisa Jackson on the film It Happened Here, which investigates the epidemic of sexual assault on university campuses. Dr. Koenen currently lives in Boston. When not working, she is likely taking a yoga class or spending time with her son, Lorcan.

Orin Levine, Ph.D., leads the foundation's efforts to accelerate the introduction of new vaccines and related technologies and to improve routine immunization systems. He is the Foundation's focal point for engagement with the GAVI Alliance whose mission is saving children's lives by increasing access to immunization in poor countries. Before joining the foundation's Global Development Program in 2012, Dr. Levine was a Professor of International Health, and Executive Director of the International Vaccine Access Center (IVAC) at the Johns Hopkins University's Bloomberg School of Public Health. He has also served as a Steering Committee Member of the Decade of Vaccines Collaboration and Co-Chair of its Global Access Working Group, as well as President, Committee on Global Health, American Society of

Tropical Medicine & Hygiene. Dr. Levine graduated with a Bachelor's degree from Gettysburg College and received a PhD in epidemiology from The Johns Hopkins Bloomberg School of Public Health.

Maureen Lichtveld, M.D., M.P.H., is Professor and Chair of Environmental Health Sciences in the School of Public Health and Tropical Medicine at Tulane University. Dr. Lichtveld's career in environmental public health spans more than 30 years. Her research focuses on environmentally induced diseases including asthma and cancer, health disparities, environmental health policy, disaster preparedness, and public health systems. Her global health expertise is focused on lower and middle income countries in the Caribbean, a region faced with recurring infectious disease epidemics, a significant NCD burden natural disasters, and unprecedented environmental health threats.

Gbenga Ogedegbe, M.D., M.P.H., M.S., a physician, is the Adolph & Margaret Berger Professor of Population Health & Medicine, Chief Division of Health & Behavior and Director Center for Healthful Behavior Change in the Department of Population Health at New York University School of Medicine. Gbenga is a leading expert on health disparities research; his work focuses on the implementation of evidence-based interventions for cardiovascular risk reduction in minority populations. He is Principal Investigator on numerous NIH projects, and has expanded his work globally to Sub-Saharan Africa where he is funded by the NIH to strengthen research capacity and reduce the burden of noncommunicable diseases. He has co-authored over 250 publications and his work has been recognized by receipt of several research and mentoring awards including the prestigious John M. Eisenberg Excellence in Mentorship Award from the Agency for Healthcare Research and Quality, and the Daniel Savage Science Award. He has served on numerous scientific panels including the NIH, CDC, World Health Organization, and the European Union Research Council. Prior to joining NYU, he was faculty at Cornell Weill Medical School and Columbia University College of Physicians and Surgeons.

Scott C. Ratzan, M.D., M.P.A., is Senior Fellow at the Mossavar-Rahmani Center for Business & Government at Harvard Kennedy School of Government. Dr. Ratzan has three decades of pioneering accomplishments in the U.S. and globally in health communication, health literacy and strategic diplomacy. He is the founding Editor-in-Chief of the Journal of Health Communication: International Perspectives. He has been engaged in multidisciplinary activities related to global health including as President of the ABInBev Foundation and Vice President of Global Health and Social Impact at ABInBev. Dr. Ratzan was at Johnson & Johnson for eleven years including as Vice President of Global Health at headquarters and also in Brussels as VP Government Affairs & Policy. Before his private sector engagement, he worked at the US Agency for International Development (USAID) in Washington DC, designing the framework for the Bureau of Global Health communication efforts. He launched his career in Boston spending a decade in academia as a professor and Founding Director of the Emerson-Tufts Masters Program in Health Communication. In addition to a number of publications in the global health field, he is the co-author of the definition of health literacy adopted by the US Government and incorporated in the Affordable Care Act. Recently, he has served as Co-Chair of the UN Secretary General's Every Woman Every Child Innovation Working Group, on the U.S. Centers for Disease Control and Prevention, Board of Scientific Counselors, Office of Infectious Disease and on the World Economic Forum Global Agenda Council on Well-Being and Mental Health. Dr. Ratzan has an M.D. from the University of Southern California, an M.P.A. from the Harvard Kennedy School, and an M.A. in Communication from Emerson College. His academic appointments include Adjunct Professor at Columbia University Mailman School of Public Health, Tufts University School of Medicine, and George Washington University School of Public Health.

Carlos del Rio, M.D., is a Distinguished Professor of Medicine in the Division of Infectious Diseases at Emory University School of Medicine and Executive Associate Dean for Emory at Grady. He is also

Professor of Global Health in the Department of Global Health and Professor of Epidemiology at the Rollins School of Public Health. He is also co-Director of the Emory Center for AIDS Research (CFAR) and co-PI of the Emory-CDC HIV Clinical Trials Unit and the Emory Vaccine and Treatment Evalaution Unit. Dr. del Rio is a native of Mexico where he attended medical school at Universidad La Salle, graduating in 1983. He did his Internal Medicine and Infectious Diseases residencies at Emory University. In 1989 he returned to Mexico where he was Executive Director of the National AIDS Council of Mexico (CONASIDA, the Federal agency of the Mexican Government responsible for AIDS Policy throughout Mexico), from 1992 through 1996. In November of 1996 he returned to Emory where he has been involved in patient care, teaching and research. Dr. del Rio was Chief of the Emory Medical Service at Grady Memorial Hospital from 2001 - 2009 and Chair of the Department of Global Health from 2009 - 2019. Dr. del Rio's research focuses on the early diagnosis, access to care, engagement in care, compliance with antiretrovirals and the prevention of HIV infection. He has worked for over a decade with hard-to-reach populations including substance users to improve outcomes of those infected with HIV and to prevent infection with those at risk. He is also interested in the translation of research findings into practice and policy. His international work includes collaborations in the country of Georgia, Ethiopia, Vietnam, Mexico, Kenya and Thailand. He has also worked on emerging infections such as pandemic influenza and was a member of the WHO Influenza A(H1N1) Clinical Advisory Group and of the CDC Influenza A(H1N1) Task Force during the 2009 pandemic. Dr. del Rio is a Member of the Board of Directors of the International Antiviral Society-USA (IAS-USA) and was a Board member and Chair of HIVMA of the Infectious Diseases Society of America (IDSA). He is a also the Chair of the PEPFAR Scientific Advisory Board. He is Chief Section Editor for HIV/AIDS for NEJM Journal Watch Infectious Diseases, Associate Editor for Clinical Infectious Diseases and member of the editorial board of Journal of AIDS and Global Public Health. Dr. del Rio has co-authored 30 book chapters and over 350 scientific papers. Among his many honors are the James H. Nakano Citation received in 2001 and awarded by the CDC for an outstanding scientific paper published in 2000; the Emory University Marion V. Creekmore Achievement Award for Internationalization; he was selected by the "Atlanta Magazine" as one of the 55 most influential foreign born Atlantans in 2007. In 2013 Dr. del Rio was elected to the National Academy of Medicine and in 2020 was elected as Foreign Secretary of the National Academy of Medicine.

Sarah Tishkoff, Ph.D., M.Phil., is the David and Lyn Silfen University Professor in Genetics and Biology at the University of Pennsylvania, holding appointments in the School of Medicine and the School of Arts and Sciences. Dr. Tishkoff studies genomic and phenotypic variation in ethnically diverse Africans. Her research combines field work, laboratory research, and computational methods to examine African population history and how genetic variation can affect a wide range of practical issues – for example, why humans have different susceptibility to disease, how they metabolize drugs, and how they adapt through evolution. Dr. Tishkoff is a member of the National Academy of Sciences and a recipient of an NIH Pioneer Award, a David and Lucile Packard Career Award, a Burroughs/Wellcome Fund Career Award, and a Penn Integrates Knowledge (PIK) endowed chair. She is a member of the board of directors of the American Society of Human Genetics and is on the editorial boards at PLOS Genetics, Genome Research; Evolution, Medicine, and Public Health; G3 (Genes, Genomes, and Genetics). Her research is supported by grants from the National Institutes of Health and the National Science Foundation.

CONSENSUS STUDY PROPOSAL

The National Academies of SCIENCES ENGINEERING MEDICINE



CONSENSUS STUDY PROPOSAL

Emerging Infections: Global Health Security, Disease X, and the Future Pandemic Threat

The Institute of Medicine (IOM) first tackled the issue of microbial threats in their 1992 report *Emerging Infections: Microbial Threats to Health in the United States.* This report highlighted the need for a domestic response and preparedness capacity for new and emerging infectious diseases. Since that time, the global burden of infectious disease has increased, as have threats to the United States. The 2003 IOM report, *Microbial Threats to Health: Emergence, Detection, and Response*, written in the aftermath of the anthrax attacks in the United States, served as a successor to this report and called for improved surveillance, diagnostics, vaccines, antimicrobials and a global response capacity. However, in the 16 years since this report was published, the pandemic threat has continued to grow, and our surveillance, response and prevention strategy continues to be challenged. For example:

- Outbreaks of Ebola have increased in size and number, spilled beyond borders, and threatened regional health and security
- New diseases have emerged and rapidly spread globally, including the current 2019-nCoV, Middle East Respiratory Syndrome, H1N1 triple reassortant influenza, and Zika virus
- Vaccine-preventable diseases, measles in particular, have increased in multiple countries, and many, including the United States, may soon lose elimination status
- Outbreaks of old diseases, such as plague and cholera, have re-emerged in many countries
- Antibiotic-resistant pathogens have increased, and present one of the biggest threats to global health today

With each crisis, we learn new lessons, leading to new tactics to counter the pandemic threat:

- The World Health Organization created a senior management emergency response capacity
- The World Bank created the Pandemic Emergency Financing (PEF) Facility
- The Inter-Agency Standing Committee of the UN coordinates international organizations to provide humanitarian assistance during pandemics
- The Global Preparedness Monitoring Board (GPMB) was co-convened by the World Bank Group and the World Health Organization
- The African Centers for Disease Control was established
- The Coalition for Epidemic Preparedness Innovations was launched to expand the vaccine and therapeutics pipeline for new and rare infections
- National and regional networks have been established to build capacity to control and prevent emerging infections

Despite these initiatives, we are still in 'catch-up' mode in our fight against pandemics. The global response to outbreaks is often significant, but interest wanes between them, leaving preparedness

plans, vaccine and countermeasure development at the mercy of cyclical funding patterns. We face daunting microbial diversity in nature and, in our rapidly changing planet we have provided perfect conditions for new microbes to emerge. Megatrends of demographic and environmental changes, globalized travel and trade, and lack of health capacity characterize the most important disease hotspots. Our armory of vaccines and therapeutics is based on pathogens that have already emerged, and has failed to scale up in the face of a new pandemic. Finally, there is still no agreed-upon response strategy for a global pandemic. The GPMB's first annual report released in 2019 concluded that most countries have not implemented recommendations called for over previous years and identified seven urgent actions in country leadership, preparation, financial planning and international coordination.

The National Academies of Sciences, Engineering, and Medicine proposes to convene an expert committee to provide new recommendations on our strategy to deal with the complexity of emerging diseases. It will build upon recommendations from the previous IOM reports, expert committees that reviewed our global response to SARS, H1N1, MERS, and Ebola, and the global mandates expounded by the GPMB and the GHSA. The committee will identify key trends in the intervening years and what we can expect in the future, which recommendations have been implemented, and what the remaining gaps are. It will critically review the following initiatives, issues and megatrends that have emerged since the last report to identify which most effectively impact our national and global response:

- The role of One Health in preventing, detecting and responding to disease threats
- Evaluation of recently launched programs such as the
    - Global Health Security Agenda and Joint External Evaluations
    - New International Health Regulations
    - Sustainable Development Goals
    - Pandemic Emergency Financing Facility
- The impact of climate change and other global environmental changes on infectious disease transmission and outbreak risk, and mitigation strategies to deal with them
- Use of social media and big data in detecting and responding to pandemics
- The economic impact of outbreaks and economic incentives for preparedness
- Interactions of poverty, equity, and non-communicable diseases on EIDs, and how endemic and emerging disease programs can be leveraged more effectively
- Linkages between plant diseases, agriculture, food security and human infections
- Advances in our understanding of the ecology of microbes, the socio-economic and environmental drivers of disease emergence, and how this might be used to predict and prevent pandemics.

Most importantly, this report will, for the first time, bring together lessons from over 15 years of pandemic response to identify strategies in our fight against future pandemics. Disease pandemics are no longer just a health issue – they are emergencies that need an all of government response. They require international leadership, multilateral coordination, and public support on the frontline of outbreaks, and in the countries that support the response. In the age of Disease X, this report will provide a pathway towards preventing pandemics, and identify consensus strategies to break free from the cycle of pandemic emergence and response.

From:	Singaravelu, Shalini <SSingaravelu@nas.edu>
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CC:	<baruch@andrew.cmu.edu>; 'Phyllis D. Meadows' <PDMeadows@kresge.org>; <alp81@georgetown.edu>; Diane Griffin <dgriffi6@jhmi.edu>; O'Connell, Dawn (OS/ASPR/IO) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=18214a88325a46739f963c2056d46cd1-O'Connell, <Dawn.Oconnell@hhs.gov>; <mh1898@georgetown.edu>; Wollek, Scott <SWollek@nas.edu>; Attal-Juncqua, Aurelia <AAttal-Juncqua@nas.edu>; Liao, Julie <JLiao@nas.edu>;

	Pavlin, Julie <JPavlin@nas.edu>; Soler, Robin E. (CDC/DDPHSIS/CPR/OD) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=5ea1ac76765b4b2e9f20af803583e63f-Soler, Robi <dqx4@cdc.gov>; Owolabi, Yakubu (CDC/DDPHSIS/CGH/DGHT) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=1df85ed61a634af78e292b4c64e0f04f-yakubu.owol <juu6@cdc.gov>
Subject:	Canceled: HOLD - April 2022 Virtual Meeting of the Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats
Date:	2022/04/15 14:58:42
Start Date:	2022/04/25 12:00:00
End Date:	2022/04/25 17:00:00
Importance:	High
Priority:	Urgent
Type:	Schedule.Meeting.Canceled
Location:	TBD

Dear all,

Please kindly note that this meeting has been postponed until June.

Many thanks,
Shalini

Shalini Singaravelu, MSc
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Board on Health Sciences Policy
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Academies logo-Jan 2017_email ONLY.png

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Sent Date:	2022/04/15 14:57:18
Delivered Date:	2022/04/15 14:58:42

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Subject:	One Hell of an Untouchable subjects - next pandemic
Date:	2021/06/04 14:07:47
Priority:	Normal
Type:	Note

Dear Sharyl,

Did you mean it when your tag line says *UNTOUCHABLE SUBJECTS* because I have one hell of one. Mycotoxins from indoor mold in our living environments! The same mycotoxins that are HIGHLY regulated in the food industry are NOT being recognized as a hazard in our indoor environment. I will speak in Facts that have been published by your various

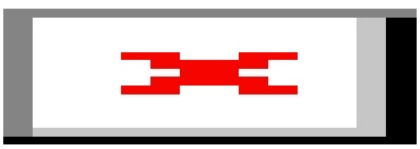
institutions which I am assuming you will accept as "Fact" since you said it. Links will be attached to the statement.

The very fact that the medical community, our politicians and our government refuses to admit that the same mycotoxins in food which are highly regulated, are the same mycotoxins produced in our indoor environments. It is a disgusting abuse of power and not serving the best interest of the people. In fact, the CDC goes one step further by **discouraging** people from testing the mold/mycotoxins in their homes. The insurance companies have excluded MOLD from their homeowner's policies because they know. Most if not all insurance companies exclude Asbestos, Lead Paint, and more recently MOLD. Why? Because all will harm you! Warren Buffet knows... Why else would he sell **USG Corporation** which remains a leading manufacturer of ceiling, floor, gypsum, roofing, sheathing, and wall products? Because the government is waiting for businesses to "Cover their Asses" before the truth gets out that Americans are being slowly poisoned. Drywall is FOOD FOR MOLD. Mold requires two things to grow, food and water. As the largest producer of Drywall, the amount of loss to USG would be astronomical when people realize this product is a hazard to their health when introduced to water. Guess who purchased USG. Knauf Int is very proficient in moving lawsuits through. Look at their court cases for Chinees Drywall and Asbestos.. Our Government is practicing Profit over People. Look at the history of lead paint. The United States did not conform to lead-based paint standards like other countries until the late 20th century. It wasn't until seven years later, in 1977, that the U.S. Consumer Product Safety Commission finally banned the use of lead-based paint completely in residential and public properties. This is also when the use of lead paint in toys and furniture was banned in the United States. How many lives could they have saved if they gave the public the knowledge that it kills you!

Mycotoxins can cause a variety of adverse health effects and pose a serious health threat to both humans and livestock. The adverse health effects of **mycotoxins** range from acute poisoning to long-term effects such as immune deficiency and cancer. https://www.**who**.int/news-room/fact-sheets/detail/mycotoxins#:~:text=Mycotoxins%20can%20cause%20a%20variety,as%20immune%20deficiency%20and%20cancer

Mycotoxins are secondary metabolites produced by microfungi that are capable of causing disease and death in humans and other animals.
Below is the summary of an article from your colleagues. It clearly states " Mycotoxins usually enter the body via ingestion of contaminated foods, but inhalation of toxigenic spores and direct dermal contact are also important routes."

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC164220/



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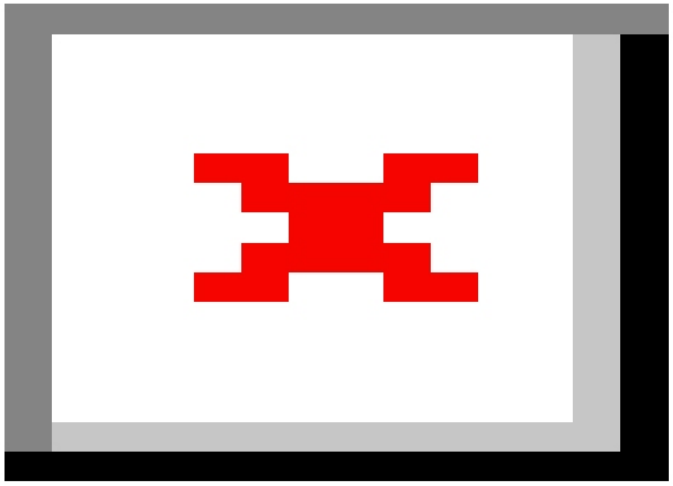
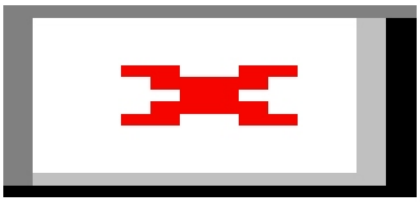


image.png

_____SCARY!_____
_____

THE CDC tells us that mold produces mycotoxins and that mycotoxins can be hazardous **YET they tell us NOT TO TEST IT?** Why wouldn't we want it tested. **California's** implied **warranty of habitability** requires a landlord to keep the premises in a condition fit for the occupation of human beings. Landlords must substantially comply with housing and building codes/standards that materially affect a tenant's safety and health. Without testing how do we know if we live in a hazard? How will we be able to get it fixed? How will our doctors know how to treat us if they don't know what we inhaled or injected?



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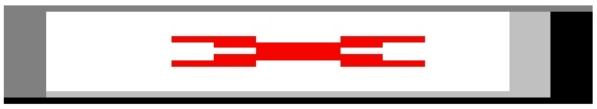

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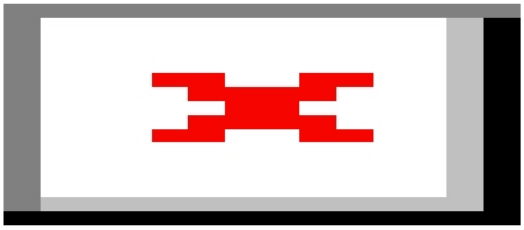

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This is the tip of the iceberg and I have been researching since I was hospitalized for 10 days in April 2014 after the Wyman Property Management in Napa California refused to even come to look at a leak for 18 months which lead to my illness. It was not until I hired a microbiologist after the hospital visit to measure the quality of my indoor air. This is when I discovered that it was NOT the mold, it is the Mycotoxins destroying my immune system as it is doing to millions of Americans.

I am an Accountant and I do realize the economic impact. The impact to the legal and medical communities will be enormous but isn't a life worth it.

How do I know all this?

I owned a Condo in Napa with my two children for 19 years, I had a job making $80,000 a year and within 2 years I lost my health due to the mycotoxins, lost my job because the ignorant doctors diagnosed me with MS and the owner fired me when he found out I was diagnosed with MS. I then Lost my home because I stopped paying my HOA fees until they fixed the leak and remediated, instead they took my home by foreclosure. I was not behind in my mortgage, they took my half a million-dollar home for Association fees, and then they blocked the 90-day redemption of my home.

It gets worse, the people who purchase the third party lien for the dues, came in the next day and remove EVERYTHING me and my children owned including textbooks and medical devices. These people failed to pay the first mortgage and only paid just when Wells Fargo was about to foreclose on the mortgage. I was not late on payments so these people DESTROYED my credit without me even knowing until it was too late Wells Fargo told me it was legal for these people to use my loan and gave them the information to pay just the amount to keep them out of foreclosure. This started the cycle of late payments being reported to my credit all over again. I tried to rent a place but my credit was destroyed! I lived in motel6 for nearly a year with my daughter, two dogs, and two birds until I ran out of money and had to work for motel 6 for $9.05 an hour. We had to rent a room in a 5 bedroom home with 8 strangers. Now the government takes care of me. This is what the Mycotoxins in Mold did to me... I am a member of ONE of the many FACEBOOK groups that have over 29,000 people in it. The fact that there is no FDA-approved **test for mycotoxins** in human urine.

Don't wait 17 years before you warn the public as you did with Lead Paint and hold property managers liable NOW for illnesses created from toxic environments. At the very least REQUIRE them to do yearly testing of indoor environments including MILITARY housing. The renter then has a choice! A choice of whether we choose to rent these places with high mycotoxins. PLUS people will have the lab report with the type of mycotoxins they have been exposed to so the medical community will finally believe that the same mycotoxins that make us sick and are highly regulated by ALL governments in food is the same mycotoxins created in our homes. We can begin to heal as a community.

The CURE is easy. See mold, run. You smell mold, run. If you cannot run immediately- OPEN all windows and sleep next to an open window when you sleep.
Avoid the 11 foods highest in mycotoxins and all animals that eat these foods like cows, pigs, and chicken, and avoid their byproducts like eggs, milk, and cheese. This is going to kill these industries which are HIGHLY subsidized by the government. Can you see the economic impact this is going to have... It is an avalanche ready to tumble down..

This is just the beginning.. I am almost done with my website and my book called "Chicken Politics". I have gone to every government agency for help where NONE was to be found. I went to my doctors but without a FEDERALLY approved test for mycotoxins in humans, the doctors cannot make an accurate diagnosis. Without a doctor's diagnosis that the indoor environment is making us ill, we cannot force the landlords to fix the issues because no court of law will recognize Mycotoxins as the cause of our illness so lawsuits are impossible because NO attorney will represent them.

If any of you see a flaw in my logic, I would be grateful for the correction. I would be happy to share my research with any of you.

The American People will only have the wool pooled over their eyes for so long. I even have pictures of 404 errors for the moved website from when I first started researching mold. I believe the government is removing and/or redirecting the links through software programs in order to hide the fact from the people.

I have a story, I have the facts and I am ready to tell the world! You have an opportunity to work with me and help me get the information out there without MASS PANIC and/or Economic impact and that does not mean REMOVING FACTS/Links from the internet.

I look forward to hearing from you!

Stacee Cootes
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Recipient:	<info@sharylattkisson.com>; <aravinda.chakravarti@nyulangone.org>; <andersen@scripps.edu>; <trevor@bedford.io>; <daszak@ecohealthalliance.org>; <dgriffi6@jhmi.edu>; <ggronvall@jhu.edu>; <tinglesby@jhu.edu>; <stanley-perman@uiowa.edu>; <kathrybr@dni.gov>; Fauci, Anthony (NIH/NIAID) [E] /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=826965b24a314ffca7eddcb6e8229aa7-anthony.fau <afauci@niaid.nih.gov>; Hassell, David (Chris) (OS/ASPR/IO) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=aedbfb0ff96e4119ac7a3b3abaf71a3d-Hassell, Da <David.Hassell@hhs.gov>; Prasher, Joanna (CDC/DDPHSIS/CPR/OD) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=e13ced68f0e44e7b81e9698d31849967-joanna.pras <mex7@cdc.gov>; (b)(6) <ian.d.watson@ostp.eop.gov>; <robert.kadlec@hhs.gov>; Conrad, Patricia (NIH/NIAID) [E] /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=3793ae43a1744d8f8aa56b600c0c975b-patricia.co <conradpa@niaid.nih.gov>; Barasch, Kimberly (NIH/NIAID) [E] /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=efc199caf0ae49039e810a8fdae070dc-kimberly.ba <kimberly.barasch@nih.gov>; <dmay@nas.edu>; <schoa@nas.edu>; <klaney@nas.edu>; <cshore@nas.edu>; <mshelton@nas.edu>; <gsymmes@nas.edu>; <lbrown@nas.edu>; <vdzau@nas.edu>; <sbeachy@nas.edu>;

	<klogan@nas.edu>; <adowney@nas.edu>; <swollek@nas.edu>; <mkearney@nas.edu>; <dkorsen@nas.edu>; <cBehney@nas.edu>; <lshern@nas.edu>; <bborel@nas.edu>; <apope@nas.edu>
Sent Date:	2021/06/04 14:06:56
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SUMMARY

Go to:

Fungi cause human illness in different ways. Mycoses are the best-known diseases of fungal etiology, but toxic secondary metabolites produced by saprophytic species are also an important health hazard. The term mycotoxin is an artificial rubric used to describe pharmacologically active mold metabolites characterized by vertebrate toxicity. They fall into several chemically unrelated classes, are produced in a strain-specific way, and elicit some complicated and overlapping toxigenic activities in sensitive species that include carcinogenicity, inhibition of protein synthesis, immunosuppression, dermal irritation, and other metabolic perturbations. Mycotoxins usually enter the body via ingestion of contaminated foods, but inhalation of toxigenic spores and direct dermal contact are also important routes.

It is difficult to prove that a disease is a mycotoxicosis. Molds may be present without producing any toxin. Thus, the demonstration of mold contamination is not the same thing as the demonstration of mycotoxin contamination. Moreover, even when mycotoxins are detected, it is not easy to show that they are the etiological agents in a given veterinary or human health problem. Nevertheless, there is sufficient evidence from animal models and human epidemiological data to conclude that mycotoxins pose an important danger to human and animal health, albeit one that is hard to pin down. The incidence of mycotoxicoses may be more common than suspected. It is easy to attribute the symptoms of acute mycotoxin poisoning to other causes; the opposite is true of etiology. It is not easy to prove that cancer and other chronic conditions are caused by mycotoxin exposure. In summary, in the absence of appropriate investigative criteria and reliable laboratory tests, the mycotoxicoses will remain diagnostically daunting diseases.

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Clin Microbiol Rev. 2003 Jul; 16(3): 497–516.
doi: 10.1128/CMR.16.3.497-516.2003

PMCID: PMC164220
PMID: 12857779

Mycotoxins






Promoting productive workplaces through safety and health research NIOSH

INDOOR ENVIRONMENTAL QUALITY

What is mold?

“Toxic Mold” & *Stachybotrys chartarum*

Certain molds are toxigenic, meaning they can produce toxins (mycotoxins), but the molds themselves are not toxic, or poisonous. Hazards presented by molds that may produce mycotoxins,



CDC does not recommend **mold testing**. The health effects of **mold** can be different for different people so you cannot rely on sampling and culturing to know if you or a member of your family might become sick. No matter what type of **mold** is present, you need to remove it.

https://www.cdc.gov › mold › control_mold

You Can Control Mold | CDC

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To:	Hassell, David (Chris) (OS/ASPR/IO) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=aedbfb0ff96e4119ac7a3b3abaf71a3d-Hassell, Da (b)(6) Sloane, Margaret (OS/ASPR/IO) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=db86d85f76a644eaa3b9a21ccef6a901-Sloane, Mar <Margaret.Sloane@hhs.gov>; (b)(6) (b)(6) 'carrie.hultberg@moore.org' <carrie.hultberg@moore.org>; Andreadis, Joanne (CDC/DDPHSIS/CPR/OD) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=2f7e7f8dcdf740329bbb1fd1a2c184fe-Andreadis, <jsa9@cdc.gov>; (b)(6) (b)(6) (b)(6) (b)(6) 'antoinette_baric@med.unc.edu' <antoinette_baric@med.unc.edu>; 'mary.bassett@health.ny.gov' <mary.bassett@health.ny.gov>; 'georges.benjamin@apha.org' <georges.benjamin@apha.org>; 'asma.shethwalayu@apha.org' <asma.shethwalayu@apha.org>; (b)(6) (b)(6) 'pdubie@ihi.org' <pdubie@ihi.org>; 'rbesser@rwjf.org' <rbesser@rwjf.org>; 'nferris@rwjf.org' <nferris@rwjf.org>; 'alta.charo@wisc.edu' <alta.charo@wisc.edu>; 'daszak@ecohealthalliance.org' <daszak@ecohealthalliance.org>; 'andre@ecohealthalliance.org' <andre@ecohealthalliance.org>; 'Gigi Gronvall' <ggronvall@jhu.edu>; 'Jeff.Duchin@kingcounty.gov' <Jeff.Duchin@kingcounty.gov>; 'stephanie.scappini@kingcounty.gov' <stephanie.scappini@kingcounty.gov>; 'bgroves@georgetown.edu' <bgroves@georgetown.edu>; 'Motrya.Calafiura@georgetown.edu' <Motrya.Calafiura@georgetown.edu>; 'eembrey@stratitia.com' <eembrey@stratitia.com>; 'peggy@hbfam.net' <peggy@hbfam.net>; 'baruch@cmu.edu' <baruch@cmu.edu>; 'dgriffi6@jhu.edu' <dgriffi6@jhu.edu>; (b)(6) (b)(6) 'Navish, Maureen' <mnavish@iqt.org>; hick.john /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=user7b974f4e (b)(6) 'pmeadows@kresge.org' <pmeadows@kresge.org>; 'KAHoffman@kresge.org' <KAHoffman@kresge.org>; 'kkester@iavi.org' <kkester@iavi.org>; 'totoole@iqt.org' <totoole@iqt.org>; (b)(6) (b)(6) 'alexandra.phelan@georgetown.edu' <alexandra.phelan@georgetown.edu>; 'Nicole Lurie' (b)(6) 'relman@stanford.edu' <relman@stanford.edu>; 'jkmazet@ucdavis.edu' <jkmazet@ucdavis.edu>; 'dmagallanes@ucdavis.edu' <dmagallanes@ucdavis.edu>; (b)(6) (b)(6) 'dwalt@bwh.harvard.edu' <dwalt@bwh.harvard.edu>
Subject:	HOLD - April 2022 Virtual Meeting of the Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats
Date:	2022/04/04 12:59:23
Start Date:	2022/04/25 12:00:00
End Date:	2022/04/25 17:00:00
Due Date:	2022/04/03 20:00:00
Priority:	Normal
Type:	Schedule.Meeting.Request
Location:	TBD
Attendees:	Hassell, David (Chris) (OS/ASPR/IO); Sloane, Margaret (OS/ASPR/IO); 'harvey.fineberg@moore.org'; 'carrie.hultberg@moore.org'; Andreadis, Joanne (CDC/DDPHSIS/CPR/OD) (b)(6)

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Dear all,

Please hold this time for the April 2022 meeting of the Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats.
An agenda with connection details and briefing materials will be shared closer to the meeting date.

Many thanks,
Shalini

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Sent Date:	2022/04/04 12:58:19
Delivered Date:	2022/04/04 12:59:23

From:	Brown, Lisa <LBrown@nas.edu>
To:	Brown, Lisa <LBrown@nas.edu>; Kadlec, Robert (OS/ASPR/IO) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=a182eda693d040d3832bae6efcf7a255-Kadlec, Rob <Robert.Kadlec@hhs.gov>; 'Alexandra Phelan (alp81@georgetown.edu)' <alp81@georgetown.edu>; 'David A Relman (relman@stanford.edu)' <relman@stanford.edu>; 'David Walt (dwalt@bwh.harvard.edu)' <dwalt@bwh.harvard.edu>; 'Diane Griffin (dgriffi6@jhmi.edu)' <dgriffi6@jhmi.edu>; 'Embrey, Ellen (eembrey@stratitia.com)' <eembrey@stratitia.com>; 'Georges Benjamin (georges.benjamin@apha.org)' <georges.benjamin@apha.org>; hick.john /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=user7b974f4e (b)(6) 'Jonna Mazet (jkmazet@ucdavis.edu)' <jkmazet@ucdavis.edu>; 'Kent Kester (Kent.Kester@sanofi.com)' <Kent.Kester@sanofi.com>; 'Kristian G. Andersen (b)(6) (b)(6) 'Mark Smolinski (b)(6) (b)(6) 'Mary Travis Bassett (mbassett@hsph.harvard.edu)' <mbassett@hsph.harvard.edu>; 'Patricia King (b)(6) (b)(6) 'Peggy Hamburg (peggy@hbfam.net)' <peggy@hbfam.net>; 'Peter Daszak (daszak@ecohealthalliance.org)' <daszak@ecohealthalliance.org>; 'Phyllis D. Meadows (PDMeadows@kresge.org)' <PDMeadows@kresge.org>; 'Richard Besser (rbesser@rwjf.org)' <rbesser@rwjf.org>; 'Tara O'Toole (totoole@iqt.org)' <totoole@iqt.org>; 'Trevor Bedford (trevor@bedford.io)' <trevor@bedford.io>; (b)(6) (b)(6) 'Donald Berwick' (b)(6) 'alta.charo@wisc.edu' <alta.charo@wisc.edu>; 'Jeff.Duchin@kingcounty.gov' <Jeff.Duchin@kingcounty.gov>; 'Baruch Fischhoff' <baruch@cmu.edu>; (b)(6) (b)(6) 'bgroves@georgetown.edu' <bgroves@georgetown.edu>; 'Harvey V. Fineberg (harvey.fineberg@moore.org)' <harvey.fineberg@moore.org>; Pope, Andrew <APope@nas.edu>; Pavlin, Julie <JPavlin@nas.edu>; Shore, Carolyn <CShore@nas.edu>; Wollek, Scott <SWollek@nas.edu>; Downey, Autumn <ADowney@nas.edu>; Fine, Emma <EFine@nas.edu>; Kahn, Benjamin <BKahn@nas.edu>; Attal-Juncqua, Aurelia <AAttal-Juncqua@nas.edu>; Feit, Monica <MFeit@nas.edu>; Liao, Julie <JLiao@nas.edu>; Dzau, Victor J. <VDzau@nas.edu>; Kanarek, Morgan <MKanarek@nas.edu>; Hassell, David (Chris) (OS/ASPR/IO) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=aedbfb0ff96e4119ac7a3b3abaf71a3d-Hassell, Da <David.Hassell@hhs.gov>; 'Waterman Paige E. EOP/OSTP' <Paige.E.Waterman@ostp.eop.gov>; Watson, Ian (OS/ASPR/SPPR) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=3bc7da05f4e949ddbf55281ba404386a-Watson, Ian <Ian.Watson@hhs.gov>; 'Motrya Calafiura' <Motrya.Calafiura@georgetown.edu>; Ferris, Nicole <nferris@rwjf.org>
Subject:	Standing Committee on EID and 21st Century Health Threats: URGENT Call on Monoclonal Antibodies
Date:	2020/11/11 11:29:00
Start Date:	2020/11/15 11:00:00
End Date:	2020/11/15 13:00:00
Priority:	Normal
Type:	Appointment
Location:	https://nasem.zoom.us/j/96818499306

Boss-
Andy Pope asked whether you could be on the start of this call to offer a couple of words. This call is in response to Victor's request, originating with the request from the SHO for California.
Note Victor will only be on the first 20-30 minutes of the call.

Chris

-----Original Appointment-----
From: Brown, Lisa <LBrown@nas.edu>
Sent: Tuesday, November 10, 2020 6:34 PM
To: Brown, Lisa; 'Alexandra Phelan (alp81@georgetown.edu)'; 'David A Relman (relman@stanford.edu)'; 'David Walt (dwalt@bwh.harvard.edu)'; 'Diane Griffin (dgriffi6@jhmi.edu)'; 'Embrey, Ellen (eembrey@stratitia.com)'; 'Georges Benjamin (georges.benjamin@apha.org)'; hick.john; 'Jonna Mazet (jkmazet@ucdavis.edu)'; 'Kent Kester (Kent.Kester@sanofi.com)'; 'Kristian G. Andersen (b)(6) 'Mark Smolinski (b)(6) 'Mary Travis Bassett (mbassett@hsph.harvard.edu)'; 'Patricia King (b)(6) 'Peggy Hamburg (peggy@hbfam.net)'; 'Peter Daszak (daszak@ecohealthalliance.org)'; 'Phyllis D. Meadows (PDMeadows@kresge.org)'; 'Richard Besser (rbesser@rwjf.org)'; 'Tara O'Toole (totoole@iqt.org)'; 'Trevor Bedford (trevor@bedford.io)'; 'rbaric@email.unc.edu'; 'Donald Berwick'; 'alta.charo@wisc.edu'; 'Jeff.Duchin@kingcounty.gov'; 'Baruch Fischhoff'; (b)(6) 'bgroves@georgetown.edu'; 'Harvey V. Fineberg (harvey.fineberg@moore.org)'; Pope, Andrew; Pavlin, Julie; Shore, Carolyn; Wollek, Scott; Downey, Autumn; Fine, Emma; Kahn, Benjamin; Attal-Juncqua, Aurelia; Feit, Monica; Liao, Julie; Dzau, Victor J.; Kanarek, Morgan; Hassell, David (Chris) (OS/ASPR/IO); 'Waterman Paige E. EOP/OSTP'; Watson, Ian (OS/ASPR/SPPR); 'Motrya Calafiura'; Ferris, Nicole
Subject: Standing Committee on EID and 21st Century Health Threats: URGENT Call on Monoclonal Antibodies
When: Sunday, November 15, 2020 11:00 AM-1:00 PM (UTC-05:00) Eastern Time (US & Canada).
Where (b)(6)

Join from PC, Mac, Linux, iOS or Android: https://nasem.zoom.us/j/96818499306

Or iPhone one-tap:
US: (b)(6)
Or Telephone:
Dial(for higher quality, dial a number based on your current location):
US: (b)(6)
Meeting ID: (b)(6)
International numbers available (b)(6)

Sender:	Brown, Lisa <LBrown@nas.edu>
Recipient:	Brown, Lisa <LBrown@nas.edu>; Kadlec, Robert (OS/ASPR/IO) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=a182eda693d040d3832bae6efcf7a255-Kadlec, Rob <Robert.Kadlec@hhs.gov>; 'Alexandra Phelan (alp81@georgetown.edu)' <alp81@georgetown.edu>;

	'David A Relman (relman@stanford.edu)' <relman@stanford.edu>; 'David Walt (dwalt@bwh.harvard.edu)' <dwalt@bwh.harvard.edu>; 'Diane Griffin (dgriffi6@jhmi.edu)' <dgriffi6@jhmi.edu>; 'Embrey, Ellen (eembrey@stratitia.com)' <eembrey@stratitia.com>; 'Georges Benjamin (georges.benjamin@apha.org)' <georges.benjamin@apha.org>; hick.john /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=user7b974f4e (b)(6) 'Jonna Mazet (jkmazet@ucdavis.edu)' <jkmazet@ucdavis.edu>; 'Kent Kester (Kent.Kester@sanofi.com)' <Kent.Kester@sanofi.com>; 'Kristian G. Andersen (b)(6) (b)(6) 'Mark Smolinski (b)(6) (b)(6) 'Mary Travis Bassett (mbassett@hsph.harvard.edu)' <mbassett@hsph.harvard.edu>; 'Patricia King (b)(6) (b)(6) 'Peggy Hamburg (peggy@hbfam.net)' <peggy@hbfam.net>; 'Peter Daszak (daszak@ecohealthalliance.org)' <daszak@ecohealthalliance.org>; 'Phyllis D. Meadows (PDMeadows@kresge.org)' <PDMeadows@kresge.org>; 'Richard Besser (rbesser@rwjf.org)' <rbesser@rwjf.org>; 'Tara O'Toole (totoole@iqt.org)' <totoole@iqt.org>; 'Trevor Bedford (trevor@bedford.io)' <trevor@bedford.io>; (b)(6) (b)(6) 'Donald Berwick' (b)(6) 'alta.charo@wisc.edu' <alta.charo@wisc.edu>; 'Jeff.Duchin@kingcounty.gov' <Jeff.Duchin@kingcounty.gov>; 'Baruch Fischhoff' <baruch@cmu.edu>; (b)(6) (b)(6) 'bgroves@georgetown.edu' <bgroves@georgetown.edu>; 'Harvey V. Fineberg (harvey.fineberg@moore.org)' <harvey.fineberg@moore.org>; Pope, Andrew <APope@nas.edu>; Pavlin, Julie <JPavlin@nas.edu>; Shore, Carolyn <CShore@nas.edu>; Wollek, Scott <SWollek@nas.edu>; Downey, Autumn <ADowney@nas.edu>; Fine, Emma <EFine@nas.edu>; Kahn, Benjamin <BKahn@nas.edu>; Attal-Juncqua, Aurelia <AAttal-Juncqua@nas.edu>; Feit, Monica <MFeit@nas.edu>; Liao, Julie <JLiao@nas.edu>; Dzau, Victor J. <VDzau@nas.edu>; Kanarek, Morgan <MKanarek@nas.edu>; Hassell, David (Chris) (OS/ASPR/IO) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=aedbfb0ff96e4119ac7a3b3abaf71a3d-Hassell, Da <David.Hassell@hhs.gov>; 'Waterman Paige E. EOP/OSTP' <Paige.E.Waterman@ostp.eop.gov>; Watson, Ian (OS/ASPR/SPPR) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=3bc7da05f4e949ddbf55281ba404386a-Watson, Ian <Ian.Watson@hhs.gov>; 'Motrya Calafiura' <Motrya.Calafiura@georgetown.edu>; Ferris, Nicole <nferris@rwjf.org>
Sent Date:	2020/11/11 11:29:00

From:	Caneva, Duane (b)(6)
To:	Dr. (b) (b) (b)(6) (b)((b)(6) (b)(6)
CC:	(b)((b) (b)(6) Evaluation Only. Created with Aspose.HTML. Copyright 2013-2020 Aspose Pty Ltd.gt;; (b)(6) (b) (b)(6) (b) (b)(6) (b)(6) (b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=user4629b831 (b)(6) Dr. (b) (b) (b)(6) BOURNE, ALEXANDRA (b)(6) (b) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) eric.mcdonald@sdcounty.ca.gov /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=04b8a65f67f9453b9749d3d366481f53-Guest_bef32 <eric.mcdonald@sdcounty.ca.gov>; (b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=474b01361a8b4944a39afbdad800591e-Guest_bea05 (b)(6) (b)((b)((b)(6) (b) (b)(6) (b)(6) (b) (b)(6) MD (b)(6) (b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=20f0e9a3ebeb4ef99d30a96386fb2627-Guest_945f5 (b)(6) (b)(6) (b) (b)(6) (b)((b)(S (b)(6) (b)(6) (b) (b)(6) McGinn, (b) (b)(6) (b)(6) (b)(Kellermann (b)(6) (b)(6) (b) (b)(6) (b)((b) (b)(6) (b)(6) (b)((b)(6) Eastman, (b)(6) (b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=d314e75d55434b198c36e0c981e6623d-Contact_753 (b)(6) (b)(6) (b)(6) (b)(6) (b)((b)((b)(6) LLogandakar (b)(6) Walters, (b)(6) (b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group (b)(6) (b)(6) (b) (b)(6) (b)(6) DC <michelle.colby@usda.gov>; (b)(6) JtCIVtUSARMY (USA) (b)(6) (b) (b)(6) MD (b)(6) Fantinato, Jessica (USDA.GOV) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=4a03b6317c3840afb4ee617008e821a6-Jessica.Fan <jessica.fantinato@usda.gov>; Martin, Greg (b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=d48a91c6dc5f401a80e245c518874f15-MartinGJ.os (b)(6) (b) (b)(6) (b)(6) (b)((b)(6) (b)(6) KAUSHIK, SANGEETA (b)(6) (b)(6) (b)((b)(6) (b)((b)(6) (b)((b)(6) (b)(6) (b) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Larry G (b)(6) Ignacio, Joselito (b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=908f39d0872d44b1b2c5e3ba11f04968-Joselito.Ig (b)(6)

	(b)((b)(6) Hassell, David (Chris) (OS/ASPR/IO) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=aedbfb0ff96e4119ac7a3b3abaf71a3d-Hassell, Da <David.Hassell@hhs.gov>; (b) (b)(6) (b)(6) (b)((b)(6) (b)(6) (b)((b)(6)) (b)(6) (b)(6) (b)(6) (b)(6) (b)((b)((b)(6) (b)((b)(6) (b)(6) WILKINSON, THOMAS (b)(6) Wolfe, Herbert (DHS.GOV) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=f2fb845c2e154d8e967ec3fdabecfbd6-Herbert.Wol (b)(6) (b)(6) (b)(6) Marinissen, Maria (HHS/OS/OGA) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=5d42fb4d94b041ee88e4f7dd5743e893-Marinissen, <Maria.Marinissen@hhs.gov>; Sutter, (b) (b)(6) (b)(6) (b)((b)(6) (b) (b) (b)(6) (b) (b)((b)(6) Krohmer, Jon (dot.gov) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=46c6513591aa4142b2c1c85354dd1816-Jon.Krohmer <Jon.krohmer@dot.gov>; (b)(6) (APHMFP - Emergency Medicine) (b)(6) You, Edward H. (WMD) (FBI) (b)(6) (b)((b)(W (b)(6)
Subject:	RE: [External] Re: Red Dawn Posturing, Begin May 5, 12:50 EDT
Date:	2020/05/15 16:29:49
Priority:	Normal
Type:	Note

DHS S&T Weekly update to the Master Question List attached.

Data dump:

Science

https://www.eurekalert.org/pub_releases/2020-05/uoa-sdt051420.php

Scientists develop tool to sequence circular DNA

New method will provide insight into genomes of bacteria and viruses, as well as extrachromosomal circular DNA in mammals and plants. University of Alberta biologists have invented a new way for sequencing circular DNA, according to a new study. The tool - called CIDER-Seq - will give other scientists rich, accurate data on circular DNA in any type of cell. While our own DNA is linear, circular DNA is common in the genomes of bacteria and viruses. Scientists have also discovered circular DNA within the nuclei of human and plant cells, called extrachromosomal circular DNA (eccDNA). Recently, research has begun to investigate the role of eccDNA in human cancer but progress has been hampered due to the lack of effective methods for studying and sequencing eccDNA.

University of Alberta
14-May-2020

https://www.the-scientist.com/news-opinion/droplets-from-speech-can-float-in-air-for-eight-minutes-study-67538?utm_campaign=TS_DAILY%20NEWSLETTER_2020&utm_source=hs_email&utm_medium=email&utm_content=87992907&_hsenc=p2ANqtz-

8aVWmtxVWrgfwS6Sdn6rdCuwqlVjjjN97FH5hsJ_EyKzj_CfTMemFPVuNub4U81QYPW28WuN8ZNrCpAL11PuhiVi3_WsgSrithAppkX6XRQztdyKQ&_hsmi=87992907

Droplets from Speech Can Float in Air for Eight Minutes: Study

The experiments did not involve SARS-CoV-2, but researchers say the results support precautions to avoid possibly spreading COVID-19 by talking.

Kerry Grens

May 15, 2020

Amid concerns that COVID-19 may be spread through aerosols, scientists have shown that tiny respiratory droplets produced when people talk can linger in the air for minutes. The results, published in *PNAS* yesterday (May 13), did not examine the transmission of viruses in spray from speech, but bolsters the idea that talking could present a risk for exposure to the novel coronavirus.

"This study builds on earlier research by the same team showing that speaking may factor into transmission of SARS-CoV-2," a spokesperson for the National Institute of Diabetes and Digestive and Kidney Diseases, where most of the study's authors are based, tells *USA Today*.
The authors used laser light sheets to capture on video the movement of small droplets emitted from a person's mouth as the speaker repeated the phrase "stay healthy" for 25 seconds. They calculated that the half-life of these particles in the air, considering the time it took them to fall 30 cm, was eight minutes. "This study is the most accurate measure of the size, number and frequency of droplets that leave the mouth during a normal conversation and shower any listeners within range," Benjamin Neuman, a virologist at TAMU-Texarkana who was not involved in the research, tells *The Washington Post*.

The researchers did not examine these respiratory droplets in the context of a SARS-CoV-2 infection. But in their paper they write that in a scenario where an infected person is in the vicinity of uninfected people, "At an average viral load of 7×10^6 per milliliter, we estimate that 1 min of loud speaking generates at least 1,000 virion-containing droplet nuclei that remain airborne for more than 8 min. These therefore could be inhaled by others and, according to IAH [independent action hypothesis], trigger a new SARS-CoV-2 infection."

The airborne particles were about 4 μm in diameter, and had dehydrated from respiratory droplets that the authors estimate were larger than 12 μm in diameter when they left the speaker's mouth.

The spokesperson for National Institute of Diabetes and Digestive and Kidney Diseases suggests to USA Today that the study's results support the CDC's recommendation to wear a mask. Although many governments have ordered or urged residents to don masks, it's not clear how well they prevent the transmission of the coronavirus. N95 masks filter 95 percent of particles larger than 0.3 μm (according to an early report describing COVID-19 infections, the SARS-CoV-2 viral particles range from 0.6 to 0.14 μm in diameter). For other types of masks, that efficiency varies considerably. An analysis of materials used in home-made masks found a wide range in their ability to block particles about 0.1 μm in size, from just 7 percent for two layers of quilters' cotton to 84 percent for a mask made from a nanofiber layer paired with cotton ripstop.

A study published in April that asked patients to cough into either surgical masks or cotton masks found neither "effectively filtered SARS–CoV-2 during coughs by infected patients." Other researchers have found that masking policies correlate with fewer COVID-19 cases, and estimate that widespread uptake of the use of masks could considerably limit infections.

A video illustrating the study "Small saliva droplets can remain airborne in an enclosed space for more than 10 minutes, NIDDK study shows" at https://www.youtube.com/watch?v=axmRl6P6xyw)

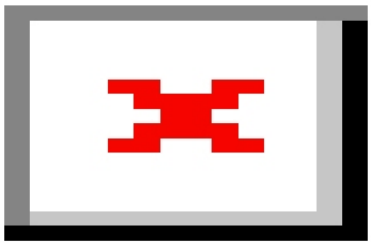
Health

https://www.technologynetworks.com/informatics/news/designing-vaccines-using-artificial-proteins-334877?utm_campaign=NEWSLETTER_TN_Breaking%20Science%20News&utm_source=hs_email&utm_medium=email&utm_content=87986690&_hsenc=p2ANqtz--0rfTHzFameJbAVaHjXCx2uZ_yV7x1M2-gqE6cYIDj-JEUfzWyptz31mAoA-JWVFGIKQkpYyRnE1rDXBrUA28xGbUWkB_frRIF--mwfuA6JvoeRjg&_hsmi=87986690

Designing Vaccines Using Artificial Proteins

May 15, 2020 | Original story from EPFL

Vaccines are one of the most effective interventions to prevent the spreading of infectious diseases. They trigger the immune system to produce antibodies that protect us against infection. However, we still lack efficacious vaccines for many important pathogens, like the flu or dengue fever. "When a vaccine doesn't work well, we tend to think that it's because the antibodies produced are not protective," says Bruno Correia, a professor at the Laboratory of Protein Design & Immunoengineering (LPDI) in EPFL's School of Engineering. "It's usually because our immune system is simply making the wrong type of antibodies". Scientists in Correia's lab have now developed a strategy to design artificial proteins that very precisely instruct the body's immune system which antibodies to produce. The study has been published in the journal *Science*.



EPFL scientists have developed a new computational approach to create artificial proteins, which showed promising results in vivo as functional vaccines. This approach opens the possibility to engineer safer and more effective vaccines. Credit: © 2020 EPFL

A disease without a vaccine

Correia's team focused on the design of de novo proteins that can result in a vaccine for the respiratory syncytial virus (RSV). RSV causes serious lung infections and is a leading cause of hospitalization in infants and the elderly, "Despite several decades of research, up to today there is still no vaccine or cure for respiratory syncytial virus," says Correia.

The artificial proteins were created in the laboratory and then tested in animal models, and triggered the immune system to produce specific antibodies against weak spots in RSV. "Our findings are encouraging because they indicate that one day we will be able to design vaccines that target specific viruses more effectively, by prompting the immune system to generate those particular antibodies," says Correia. "We still have a lot of work ahead to make the vaccine we developed more effective - this study is a first step in that direction."

Methods for creating de novo proteins have applications well beyond immunology - they can also be used in various branches of biotechnology to expand the structural and functional range of natural proteins. "We can now use the protein design tools to create proteins for other biomedical applications such as

protein-based drugs or functionalized biomaterials," concludes Sesterhenn.

Reference: F. Sesterhenn, C. Yang, J. Bonet, J. T. Cramer, X. Wen, Y. Wang, C. Chiang, L. A. Abriata, I. Kucharska, G. Castoro, S. S. Vollers, M. Galloux, E. Dheilly, S. Rosset, P. Corthésy, S. Georgeon, M. Villard, C. A. Richard, D. Descamps, T. Delgado, E. Oricchio, M. Rameix-Welti, V. Más, S. Ervin, J. F. Eléouët, S. Riffault, J. T. Bates, J. P. Julien, Y. Li, T. Jardetzky, T. Krey & B. E. Correia. *De novo protein design enables the precise induction of RSV-neutralizing antibodies.* Science

https://protect2.fireeye.com/url?k=9f97de35-c3c3c749-9f97ef0a-0cc47adc5fa2-a0f9783a25da799f&u=http://outbreaknewstoday.com/maine-lyme-disease-providers-are-already-reporting-cases-in-2020-and-the-number-will-rise-as-we-enter-the-summer-months-74705/

Maine Lyme disease: 'Providers are already reporting cases in 2020, and the number will rise as we enter the summer months'

The Maine CDC reported a record more than 2,000 Lyme disease cases in 2019 and officials announce during this Lyme Disease Awareness Month that providers are already reporting cases in 2020, and the number will rise as we enter the summer months. This has prompted health officials to advise the public to be aware of tick bites that transmit not only Lyme disease, but also anaplasmosis (a bacterial disease), babesiosis (a parasitic disease) and Powassan virus. Individuals bitten by the deer tick can acquire more than one infection. Many individuals and families are spending more time outdoors during the COVID-19 pandemic. This may put them at increased risk of exposure to tickborne pathogens. Symptoms of anaplasmosis include: fever, headache, malaise and body aches. Symptoms of babesiosis include: extreme fatigue, aches, fever, chills, sweating, dark urine, and possibly anemia. Symptoms of Powassan include: fever, headache, vomiting, weakness, confusion, loss of coordination, speech difficulties, seizures, and encephalitis and meningitis. In 2019, providers reported 685 cases of anaplasmosis, 138 cases of babesiosis and two cases of Powassan.
by News Desk
May 14, 2020

https://www.eurekalert.org/pub_releases/2020-05/wrai-nes051420.php

New evidence suggests malaria cycles are innate to the organism

Scientists from the Walter Reed Army Institute of Research joined partners at Duke University, FAU and Montana State University to publish a study providing clear evidence that malaria's characteristic cycle of fever and chills is a result of the parasite's own influence - not factors from the host. What regulated that cycle, the result of parasites bursting out of infected red blood cells in sync then re-colonizing new red blood cells, has been studied since at least the 1920s. In the current study, evidence challenges the central dogma that a cyclic pattern of parasite growth is solely dependent on cues from the host. Though the specific signals utilized remain to be elucidated, these findings raise the exciting possibility of disrupting this cycle as an antimalarial strategy.
Walter Reed Army Institute of Research
14-May-2020

ASF

https://www.thehindu.com/news/national/other-states/wild-boars-die-in-arunachal-as-african-swine-fever-kills-15000-pigs-in-assam/article31590372.ece

Wild boars die in Arunachal as African swine fever kills 15,000 pigs in Assam

As Assam prepares for mass culling to check the spread of African swine fever (ASF) that has killed almost 15,000 domesticated pigs, adjoining Arunachal Pradesh fears that the "foreign" disease may have "gone wild". This is the 1st time that ASF has been reported in India. Assam claims the disease came from China, where almost 60% of pigs have died since 2018. Officials in Arunachal Pradesh's East Siang district said 6 carcasses of wild boars including 3 piglets were found in a community forest at Lidor Soyit

upstream of Sille River. There have been unverified reports of several wild boars dying from an unknown disease in East Siang and Upper Siang districts, but the recovery of the carcasses - some partly eaten by scavengers - on Thursday made officials wary of the possibility of ASF having spread from scores of domestic pigs that have died in the State over the last 2 months. "A team of forest, veterinary officials and experts trekked about 10 km to locate the carcasses after receiving information from the villages. We suspect ASF is the cause of death but will have to await confirmation after we send blood and tissue samples to labs outside," Divisional Forest Officer (Territorial) Tashi Mize told The Hindu on Friday. Some of the carcasses appeared to have been consumed by porcupines. "ASF is confined to porcine creatures, so other animals are unlikely to be affected. But the possibility of becoming carriers of the virus could affect the wild boar population," he said.
Special Correspondent I Guwahati I may 15, 2020 13:39 IST I Updaated: May 15, 2020 14:10 IST

https://www.ndtv.com/india-news/assam-prepares-for-culling-as-african-swine-fever-kills-nearly-15-000-pigs-2229043

Assam "Prepares" For Culling As African Swine Fever Kills Nearly 15,000 Pigs

The BJP-led government is preparing for mass culling; it has demanded from the centre compensation to farmers who rear pigs. The ASF outbreak has killed nearly 15,000 pigs in Assam and is spreading in new areas despite preventive measures taken by the state government. The state government has issued a high alert in 10 affected districts. It has asked the centre to provide one-time financial package of Rs 144 crore for the farmers who rear pigs. "We are deeply concerned due to this growing crisis in Assam. The deaths are increasing every day. Now, 10 districts have been affected already. 14,919 pigs have died and the number is on the rise. We have alerted the Government of India as well," Assam Animal Husbandry Minister Atul Bora told NDTV. "Initially, 6 districts were affected but now it has spread to 10 districts out of 33, despite the fact that we took all possible steps (for prevention), but it is spreading to new areas. " We had taken biosecurity measures to make sure it doesn't spread further. We declared that area under 1-km radius (of affected areas) as containment zone, and 10-km radius as surveillance zone," the minister added. The Assam government has alerted all wildlife reserves in the state regarding the disease.
Written by Ratnadip Choudhury I Updated: May 15, 2020 08:28 am IST

https://protect2.fireeye.com/url?k=fd00f14c-a154e830-fd00c073-0cc47adc5fa2-e1faf52d0072a066&u=https://asian-agribiz.com/magazines/asian-pork/asf-update/

Concentration, contact time key to effective disinfectant use

15 May 2020 – The correct concentration and the length of time a disinfectant is in contact with a surface will better determine its effectivity against ASF and other pathogens. Pariwat Poolperm, a swine consultant from Thailand said at a webinar titled Biosecurity: The First Step to Success in Pig Farming that for disinfectants to work against ASF, they must be in contact with surfaces for at least 15-20 minutes. Dr Pariwat also said that not all disinfectants are the same. Pig producers should follow the recommended concentrations for each disinfectant and not overconcentrate or overdilute.

ASF remains a big problem in Asia

14 May 2020 – While the Covid-19 pandemic and its effects have overshadowed ASF in the news, the deadly pig virus continues to be a big problem for Asian pig producers. China and Vietnam are both working on recovery programs, but threats of ASF reinfection and the lack of breeding pigs hamper restocking efforts. Both countries have seen a spike in the number of cases in the last two weeks. In the Philippines, industry observers note that while fewer cases are being reported, a big reason for this is the movement restrictions imposed due to the pandemic. "The virus remains, and it will take some time before it can be eradicated, if at all," an industry practitioner told *Asian Agribiz*. "It is imperative the governments and local pig industries collaborate not just to control ASF spread, but also how to rebuild their industries. Pork will continue to be the preferred meat at least in China, Vietnam, and the Philippines.

Philippines reports new ASF outbreaks

13 May 2020 – The Philippines has 58 new ASF outbreaks in Luzon, the Philippine Bureau of Animal Industry (BAI) reported to the World Organization for Animal Health. The country culled another 11,074 pigs, bringing the total as of May 4 to 282,899. The outbreaks were traced to illegal transport of animals and swill feeding. An industry practitioner told *Asian Agribiz* that while the country continues its fight against ASF, the government and industry players must begin rebuilding efforts. Unfortunately, he said, nothing concrete has been made, adding that even the country does not have enough ASF test kits.

COVID-19 Webinars

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https://gallery.mailchimp.com/18fe6f8f25ec0bc7509e65e97/images/20c38cdd-bf8e-4932-9fac-481751e2ba50.gif

Wastewater Monitoring for COVID-19 Disease Surveillance

May 27, 11-1:30 PM EDT

Researchers around the world are currently exploring ways that wastewater samples can help us understand the spread of COVID-19 at a community scale. This webinar will feature a panel discussion with experts on public health and wastewater monitoring to discuss the potential value of data on SARS-CoV-2 in wastewater to inform public health management and what is needed to build a useful surveillance network

Register for Webinar

David Sedlak, UC Berkeley, Moderator

Panelists:

- Vincent Hill, Centers for Disease Control and Prevention
- Barry Liner, Water Environment Federation
- Gertjan Medema, KWR Water Research Institute, Holland
- Nicole Rowan, Colorado Department of Public Health and Environment
- Krista Wigginton, University of Michigan

Key questions for presentations and discussions:

- How can data on SARS-CoV-2 in wastewater be useful as an indicator of COVID-19 cases in a locality?
- How has wastewater disease surveillance been useful with control of other viral pathogens? What problems have been encountered and what can we learn from these experiences for COVID-19?
- What is the capacity of current wastewater monitoring technologies for detecting COVID-19 disease outbreaks (i.e., what is the recovery efficiency, detection rate relative to the loading rates)? What are the costs?
- What technical challenges need to be addressed before this strategy can be broadly implemented as a robust tool? What are the highest priority needs?
- Where might such surveillance be appropriate?
- Is this a useful investment? If so, what would the nation need to do to rapidly invest in a useful surveillance network?

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Reopening after COVID-19: Ensuring Safe Water Supplies at the Building Scale

May 27, 2:30-4:30 PM EDT

Register for Webinar

The COVID-19 pandemic has led to sweeping closures of public buildings, businesses, offices, and schools. With minimal water use, water quality in building plumbing can degrade and foster the growth of the bacteria that cause Legionnaires' disease, a severe form of pneumonia. Legionnaires' disease incidence has been increasing over the past few decades, and without appropriate water management actions, cases could increase sharply after schools and workplaces reopen. A panel of experts will discuss the state of knowledge for building water management to protect public health when reopening after COVID-19.

Ruth Berkelman, Emory University, moderator

Panelists:

- Chris Boyd, Building Water Health Program, NSF International
- David Krause, HealthCare Consulting and Contracting
- Andrew Whelton, Purdue University

Key questions for presentations and discussions:

- What are the water quality issues and related health risks associated with reopening buildings or larger office parks/campuses that have been shuttered or minimally used during the COVID-19 outbreak?
- Are there actions that could (should) be taken now to minimize current or future risk of water-related health risks?
- What guidelines are available for building owners and utilities? What are the responsibilities of various entities?
- What issues about best practices for water management remain unresolved relative to building recommissioning?
- What issues need to be addressed to improve implementation of current guidelines (e.g., communication, training)?
- https://protect2.fireeye.com/url?k=f17f3d3f-ad2b2443-f17f0c00-0cc47adc5fa2-7834c472a696ac40&u=https://mailchi.mp/nas/register-for-upcoming-webinars-earth-and-life-sciences-and-covid-19?e=a7a58840e6

Air Quality, Climate Variability, and COVID-19

May 28, 1:00-3:00 PM EDT

As the global community has raced to understand the COVID-19 pandemic, questions have arisen about how the virus is carried in aerosols, exposure risk in indoor and outdoor environments, how the changes in seasons or climate conditions might affect transmission, and how exposure to air pollution might affect mortality from the virus. This webinar will feature recent atmospheric, climate, and epidemiological research that is contributing to our understanding of the virus transmission, as well as a discussion of how agencies across the federal government are building upon existing efforts to address linkages between environmental conditions and health to understand the pandemic.

This webinar is an open session of the Board on Atmospheric Sciences and Climate spring 2020 meeting.

Register for Webinar

Coronavirus

https://gisanddata.maps.arcgis.com/apps/opsdashboard/index.html#/bda7594740fd40299423467b48e9ecf6

From CSSE Dashboard at Johns Hopkins University: 15 May 2020 at 3:32 PM EDT

Total Confirmed: 4,516,360
Global Deaths: 306,051
US deaths: 86,851
Total Test Results in US: 10,341,775

Confirmed Cases by Country/Region/Sovereignty:
1,432,045 US
262,843 Russia
238,003 United Kingdom
230,183 Spain
223,885 Italy
212,198 **Brazil**
179,630 France
175,233 Germany
146,457 Turkey
116,635 Iran
85,784 India
84,495 Peru
84,031 China
75,667 Canada

https://www.washingtonpost.com/politics/2020/05/14/3-takeaways-coronavirus-whistleblower-rick-brights-testimony/?pwapi_token=eyJ0eXAiOiJKV1QiLCJhbGciOiJIUzI1NiJ9.eyJjb29raWVuYW1lIjoid3BfY3J0aWQiLCJpc3MiOiJDYXJ0YSIsImNvb2tpZXZhbHVlIjoiNWU3YmE4NThhZTdlOGE1OTQ4ODk2NzkzIiwidGFnIjoiNWViZGFiZmZmZTFmZjY1NGMyZGQxYTY5IiwidXJsIjoiaHR0cHM6Ly93d3cud2FzaGluZ3RvbnBvc3QuY29tL3BvbGl0aWNzLzIwMjAvMDUvMTQvMy10YWtlYXdheHMtY29yb25hdmlydXMtd2hpc3RsZWJsb3dlci1yaWNrLWJyaWdodHMtdGVzdGltb255Lz91dG1fY2FtcGFpZ249d3BfdG9feW91cl9oZWFsdGgmdXRtX21lZGl1bT1lbWFpbCZ1dG1fc291cmNlPW5ld3NsZXR0ZXImd3Bpc3JjPW5sX3R5aCZ3cG1rPTEifQ.zDzXu3HqLZZn2jf9oP1z3ihfUQWvxFCFIYpZmt7xs6A&utm_campaign=wp_to_your_health&utm_medium=email&utm_source=newsletter&wpisrc=nl_tyh&wpmk=1

5 takeaways from coronavirus whistleblower Rick Bright's testimony

By Aaron Blake
May 14, 2020 at 1:38 p.m. EDT

A Trump administration vaccine expert who says he was removed from a key role for raising concerns about the federal government's coronavirus response — and its promotion of unproven drugs to treat the virus — testified Thursday before Congress.

Rick Bright became a whistleblower after being removed from his post as director of the Biomedical Advanced Research and Development Authority (BARDA), which falls under the Department of Health and Human Services.

Here are some takeaways from Bright's testimony.

1. ‘Lives were lost’ because of ‘inaction,' unheeded warnings

Bright said that early inaction by the government — particularly in the Department of Health and Human Services — had, in fact, cost lives. “That inaction has put a lot of lives at risk in our front-line health-care workers” Bright said. Bright has said that he pushed for ramping up production of medical equipment such as masks, but that it went unheeded for months after he was informed that officials didn’t think there was a “critical shortage” of masks. “I pushed that forward to the highest levels I could in HHS and got no response,” Bright said. “From that moment, I knew that we were going to have a crisis for our health-care workers because we were not taking action. We were already behind the ball. That was our last window of opportunity to turn on that production to save the lives of those health-care workers, and we didn’t act." Bright added that even today, the country is dealing with the consequences of that early negligence and that health-care workers are still more at risk than they should be. “Lives were endangered, and I believe lives were lost,” Bright said. “And not only that: We were forced to procure these supplies from other countries without the right quality standards. So even our doctors and nurses in the hospitals today are wearing N95-marked masks from other countries that are not providing the sufficient protection that a U.S.-standard N95 mask would provide them. Some of those masks are only 30 percent effective. Therefore, nurses are rushing in the hospitals thinking they’re protected, and they’re not.”

2. Administration pushed vastly expanded use of unproven drugs

One of Bright’s key claims is that he was moved to another post after raising objections to the administration pushing the use of the malaria drugs chloroquine and hydroxychloroquine to treat coronavirus. The administration allowed for the emergency use of the drugs to treat the virus, though later studies — which have yet to be peer-reviewed and were not randomized — suggest that the use of the drugs don’t help and can, in fact, have negative consequences. The FDA has now warned about the dangers of using the drugs. “My concerns were escalated when I learned that leadership in the Department of Health and Human Services were pushing to make that drug available outside of this emergency use authorization, to flood New York and New Jersey with this drug,” Bright said. Bright has cited his skepticism of the drugs for his removal from his post and said the administration wanted to make it easier for people to use them without extensive medical supervision — even people who might not even have the virus. “I believe part of the removal process for me was initiated because of a pushback that I gave when they asked me to put in place an expanded access protocol that would make chloroquine more freely available to Americans that were not under the close supervision of a physician and may not even be confirmed to be infected with the coronavirus,” Bright said.

3. Pessimism about 12- to 18-month timeline for vaccine

President Trump has been effusively optimistic about not just treatments such as the chloroquines but about the timeline for a vaccine for the virus. Shortly before Bright’s testimony Thursday, Trump even said, “I think we’re going to have a vaccine by the end of the year.” That’s even more optimistic than the 12- to 18-month timeline that medical experts such as Anthony S. Fauci, head of the National Institute of Allergy and Infectious Diseases, have said is realistic for the vaccine. But Bright said that even a year to year-and-a-half timeline might also be overly optimistic. “I still think 12 to 18 months is an aggressive schedule, and I think it’s going to take longer than that to do so,” Bright said.

4. We don’t have ‘a single point of leadership’ or ‘master plan’

As Trump increasingly criticized Fauci, Bright said the government needs to have more regard for it scientists -- and a more consistent message from the top. He said that right now the response has been hampered by not having a “single point of leadership.” “We need to install and empower leadership, and we need to unleash the voices of the scientists in our public health system in the United States so they can be heard and their guidances need to be listened to,” Bright said. “And we need to be able to convey that information to the American public so they have the truth about the real risk and dire consequences of this virus. He added: "And we don’t have a single point of leadership right now for this response, and we don’t have a master plan for this response. So those two things are absolutely critical.” Fauci has said in recent days that states that move forward with reopening their economies before meeting the guidelines from the Centers for Disease Control and Prevention are risking new outbreaks – which could set back the entire response.

5. Azar, Republicans cast Bright as a malcontent skipping work

Evaluation Only. Created with Aspose.HTML. Copyright 2013-2020 Aspose Pty Ltd.us response to date, Republicans on the committee and members of the Trump administration sought to undercut his testimony and character. Health and Human Services Secretary Alex Azar, in particular, had strong words for Bright. “Everything he is complaining about was achieved,” Azar said while standing next to Trump on the White House lawn. “Everything he talked about was done. He says he talked about the need for respirators; we procured respirators under the president’s direction.” Azar echoed Republicans in the hearing who questioned why Bright has been absent from his new posting - a narrower one focused on testing and vaccines at the National Institutes of Health — in recent weeks. “While we’re launching Operation Warp Speed,” Azar said, “he’s not showing up for work to be part of that.” Bright said that he has been on leave while dealing with “very high blood pressure” - owing in part to the stress from recent events. “I had a conversation with my physician about my hypertension and how we’ve been managing it over the last 3 weeks because this has been very stressful to be removed suddenly without explanation from my role and position as a life change for me,” Bright said.

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Global COVID-19 death toll exceeds 300,000

The number of COVID-19 deaths today passed the 300,000 mark, as another city in China went on lockdown to prevent a resurgence and more countries in Europe learned that low numbers of people were exposed in their outbreaks, meaning many are vulnerable to a second wave. Deaths climbed to 301,160 today, with cases rising to 4,413,597, according to the Johns Hopkins online dashboard.

Lisa Schnirring | News Editor | CIDRAP News| May 14, 2020

New cluster triggers new lockdown in China

The city of Jilin in northeastern China's Jilin province went on partial lockdown yesterday after a spurt of cases, which now total 21, the *South China Morning Post* reported. Officials closed schools, restricted train and bus service, and banned gatherings after 6 cases new cases were confirmed on May 12. Anyone who wants to leave the city, home to about 4M people, must be tested for COVID-19 48 hours before departure.

The first infection in the cluster was reported last week, linked to a laundry worker from Shulan, a smaller city in Jilin province. So far, investigators haven't determined how she contracted the virus. China has taken aggressive steps to prevent a resurgence, including an earlier lockdown in the city of Suifenhe on the border with Russia, and is now launching an effort to test all Wuhan residents after a cluster of cases was detected in a residential complex.

Today the country reported 3 new cases (all local -2 from Liaoning province also in northeastern China and 1 in Jilin province). In its daily update, the National Health Commission also reported 12 more asymptomatic cases, all but one of them local.

Elsewhere in Asia, Japan's Prime Minister Shinzo Abe today lifted the state of emergency early for 39 of 47 prefectures, *Kyodo News* reported. The emergency order was originally slated to expire at the end of May. However, orders for the cities of Tokyo and Osaka, home to about 50% of Japan's population, will remain in place. Abe said experts will review the situation again on May 21.

South Korea today reported 29 more cases, 20 of them linked to nightclub-related clusters in Seoul, the Korea Centers for Disease Control and Prevention said today.

Studies: Many in France, Spain still vulnerable

As major outbreaks in France and Spain decline, researchers are using seroprevalence studies to gauge how extensively populations were exposed to the virus and help inform planning for potential 2nd waves of pandemic activity.
In France, a study by Pasteur Institute researchers published in *Science* yesterday estimated that 4.4% of the country's population were infected by the COVID-19 virus, Reuters reported. The estimates were higher, between 9% and 10%, for hard-hit areas, which included eastern France and Paris.

The results suggest that without a vaccine, herd immunity won't be enough to avoid a 2nd wave as lockdown steps ease. Pasteur scientists also estimated the 55-day lockdown dramatically dropped the outbreak's reproduction number from 2.0 to 0.67.

Meanwhile, preliminary serosurvey results from Spain suggests 5% of the population was exposed to the virus, Reuters reported, citing Fernando Simon, the country's head of health emergencies.

Lockdown for Chile's capital

Chile's government yesterday ordered a lockdown for its capital Santiago, which has a population of 7M, after experiencing a 60% increase in COVID-19 infections in a 24-hour period, the *Santiago Times*Evaluation Only. Created with Aspose.HTML. Copyright 2013-2020 Aspose Pty Ltd.which today increased by 2,659 reported cases, for a total of 37,040.

In other global developments:

- Hours after Sanofi's chief executive officer was quoted as saying the US would get first access to its COVID-19 vaccine, the company walked back the statement and said when ready, it will be available in all countries, the Associated Press Sanofi is based in France, and CEO Paul Hudson's comments provoked a strong reaction from the French government. The US-based BARDA supported the development of the vaccine.
- A typhoon that struck the Philippines' eastern provinces today sent people to evacuation centers, which was complicated by COVID-19 distancing measures, Reuters reported. One city gave hundreds of evacuees face masks to wear before they were allowed in evacuation shelters, and the local officials added 2 schools as temporary shelters to better accommodate physical distancing.
- Malaysia said it will ease a ban on prayers in mosques starting tomorrow, ahead of the Eid festival, Reuters reported. Congregations will be limited to 30 or fewer. Last week the country started reopening businesses.

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Dozens of coronavirus drugs are in development — what happens next?

Drug manufacturers face supply-chain weaknesses and sourcing issues as they ramp up complex production processes to meet global demand. The world was waiting for any sign of hope in countering the COVID-19 pandemic when researchers released the 1st encouraging drips of data from a large clinical trial of the antiviral remdesivir last month. The drug, they said, reduced the time to recovery from COVID-19 by a few days - not enough to be branded a 'cure', but hopefully enough to relieve some pressure on overwhelmed health-care systems. The discovery of remdesivir's potential focused attention on the next problem in the development of COVID-19 therapeutics: ramping up complex manufacturing processes to address a global pandemic. It is likely to be one of the biggest drug-making challenges the world has ever faced. Some of the therapies being tested against COVID-19 are novel and difficult to produce. Others - even if they are relatively simple compounds that have been in use for decades - face complications such as supply-chain weaknesses as drug-makers try to scale up production. A major rate-limiting step is going to be manufacturing," says Ezekiel Emanuel (bioethicist at the University of Pennsylvania in Philadelphia). "Getting up to hundreds of millions of doses is hard." Researchers are working furiously to test a wide

variety of potential COVID-19 treatments. Those therapies span the gamut of complexity, from familiar generic medications such as the malaria drug hydroxychloroquine, to experimental small molecules like remdesivir, which was previously trialled against the Ebola virus. Scientists are also exploring antibody treatments that tamp down the body's immune response when it becomes destructive, which happens in some critically ill coronavirus patients. And if the history of infectious disease is any guide, it will take a combination of drugs - each with a distinct, even if relatively minor, impact on the disease - to tame the novel CoV. Each treatment will face different challenges when scaling up production, says Stephen Chick, who studies health-care management at INSEAD in Fontainebleau, France. "If it's successful and the technology is then adopted, you need to be prepared to deliver," says Chick. "And if you're not, you're in trouble."
Nature
Heidi Ledford
14 May 2020

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COVID-19 Update: JBS/Smithfield testing, new outbreaks
JBS will partner with TX state health officials to test the roughly 3,000 workers at its beef plant north of Amarillo. According to a report from the Texas Tribune, state officials said JBS initially rejected an offer for testing; company spokesperson Nikki Richardson stated the company were unaware of such testing being offered. The state previously dispatched personnel from its Military Department and Division of Emergency Management to run tests on the 3,500 workers at a nearby Tyson Food plant. Meanwhile, COVID-19 continues to spread among workers at JBS' beef plant in Greeley, CO. According to state data cited by KDVR, the number of confirmed cases at the plant is now 316, up from 280 earlier in the week; 6 plant employees have died, along with 1 employee from JBS' corporate offices. JBS began testing all plant workers on Monday.
By Peter Thomas Ricci on 5/15/2020

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OSHA treads lightly on COVID-19 complaints: Politico
The Department of Labor's OSHA has conducted few investigations out of the thousands of COVID-19 complaints it has received in the last several weeks, and has issued no citations, according to a report by Politico. The Beltway newsletter reported that the agency has fielded more than 3,800 complaints related to companies not adequately protecting workers from contracting the virus in crowded workplaces, but as of May 13, has opened only 281 inspections and issued no citations. OSHA, often in conjunction with the CDC, has issued a series of guidance documents on measures companies should take on employees' behalf, but has issued no directives or mandatory standards that might trigger a violation. The agency contends more regulation is unnecessary because of the enforcement options already available to it.
By Lisa M. Keefe on 5/15/2020

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Dover Air Force Base plays role in transport isolation systems for COVID-19 patients
In the ongoing fight against COVID-19, two Transport Isolation Systems, along with trained medical Airmen, arrived at Dover Air Force Base, Delaware late last month. Dover will serve as the East Coast hub for TIS decontamination in the US, thanks to its strategic location, assets and capabilities. Airmen will support and decontaminate TIS units whose aircrews are conducting COVID-19 positive patient transport missions from Africa, Europe and the Middle East to the U.S. The team is composed of members of 6 different units from across the U.S. The TIS emerged as a result of mobility requirements identified during

Operation United Assistance in support of the Ebola outbreak in 2014. This system was designed to provide in-flight medical care while containing any infectious disease, minimizing the risk to aircrew, medical attendants and the airframe. "We have two TIS modules here, because that is a standard configuration ," said Maj. Mark Dellinger, 36th Aeromedical Evacuation Squadron training flight commander. "Each has the capability of carrying 4 patients." Medical personnel assigned to the TIS mission receive multi-day training, including familiarization with the system, patient loading/unloading procedures, donning and doffing PPE, simulated in-flight patient care and infection control procedures. The TIS enables the Department of Defense to transport patients afflicted with or suspected of an infectious disease like COVID-19 from overseas to the US, providing for an expedient recovery of its personnel, as well as preventing the spread of COVID-19 to aircrews. As part of the whole-of-government response to COVID-19, the TIS mission at Dover AFB will continue for as long as required.
By Delaware Business Now
May 14, 2020

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Coronavirus (COVID-19) Update: FDA Informs Public about Possible Accuracy Concerns with Abbott ID NOW Point-of-Care Test

FDA News Release
For Immediate Release:
May 14, 2020
Today, the U.S. Food and Drug Administration is alerting the public to early data that suggest potential inaccurate results from using the Abbott ID NOW point-of-care test to diagnose COVID-19. Specifically, the test may return false negative results. "We are still evaluating the information about inaccurate results and are in direct communications with Abbott about this important issue. We will continue to study the data available and are working with the company to create additional mechanisms for studying the test. This test can still be used and can correctly identify many positive cases in minutes. Negative results may need to be confirmed with a high-sensitivity authorized molecular test," said Tim Stenzel, M.D., Ph.D., director of the Office of In Vitro Diagnostics and Radiological Health in the FDA's Center for Devices and Radiological Health.

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DoD, HHS award contract enabling prefilled syringes for future COVID-19 vaccine

The U.S. DoD and HHS awarded a $138M contract to ApiJect Systems America for Project Jumpstart and RAPID USA, 2 programs designed to expand U.S. production of medical-grade injection devices. The contract will create a U.S.-based supply chain for prefilled syringes by using Blow-Fill-Seal (BFS) aseptic plastics manufacturing technology, suitable for combatting COVID-19 when a vaccine becomes available. By upgrading existing domestic BFS facilities with installations of filling-line and technical improvements, the project will enable the manufacture of more than 100M prefilled syringes for distribution across the US by year-end 2020.
Thursday, May 14, 2020 by Dave Kovaleski
Homeland Preparedness News

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China will have to answer questions about COVID-19, says Trudeau

Countries around the world, including Canada, have questions about the origins of the COVID-19 pandemic and about China's behaviour during the early days of the outbreak that need to be asked "in

the coming months," Prime Minister Justin Trudeau said Wednesday. Speaking at his daily briefing in Ottawa, Trudeau was responding to a question about recent comments made by Canada's ambassador to China Dominic Barton. The Globe and Mail newspaper reported Wednesday that Barton told a private session of the Canadian International Council last week that China's conduct during the pandemic is damaging its own global "soft power." Earlier this month, The Associated Press reported that U.S. officials believe China covered up the extent of the CoV outbreak - and how contagious the disease is - to stock up on medical supplies needed to respond to it. The revelation comes as the Trump administration has intensified its criticism of China, with Secretary of State Mike Pompeo saying that that country was responsible for the spread of disease and must be held accountable.
Radio Canada International
By Levon Sevunts
Posted: Wednesday, May 13, 2020 13:22

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Economists: Lack of COVID-19 preparedness in line with previous findings
The delayed response of U.S. policymakers to the COVID-19 pandemic comes as no surprise to University of Wyoming Professor Jason Shogren and several of his economist colleagues at other institutions. That's because the threat of a catastrophic pandemic in 2014 - the West African Ebola outbreak - did little to change the perception of U.S. citizens regarding the importance of preparing for future outbreaks, according to research conducted by Shogren and his colleagues. "The COVID-19 pandemic has revealed that the U.S. was as unprepared as experts feared, given the responses to the Ebola scare in 2014," the economists wrote in an article that has been accepted by EcoHealth, an international journal that addresses health and sustainability challenges, including public health practices. "This lack of attention to pandemic threats is especially disturbing given the current COVID-19 and any potential future pandemics that may also have very high transmission rates, including transmission before individuals become symptomatic." Joining Shogren in the research were fellow UW economists David Aadland, David Finnoff and Alexandre Skiba, along with Jamison Pike and Peter Saszak, of the Ecohealth Alliance; and Kip Viscusi, from Vanderbilt University. Before and after the 2014 Ebola outbreak - which killed thousands of people in West Africa, harmed millions of people in that region and generated significant media coverage in the US - the researchers surveyed U.S. citizens to see if their concerns about a pandemic threat had increased, relative to risks from environmental disasters and terrorism. They were surprised to find relative complacency regarding the threat of a pandemic among the hundreds of people surveyed in 2015. The findings were unexpected because long-standing evidence has shown that people's perceptions usually are distorted toward the most recent news items.
University of Wyoming
14-May-2020

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K-State infectious disease scientist offers road map for future COVID-19 research
Researcher investigates potential therapeutic options at Biosecurity Research Institute. There are many unanswered questions about COVID-19. A Kansas State University infectious disease scientist and collaborators are offering a possible research road map to find the answers. "We need to address these challenges in a scientific manner - in a proactive manner, not in a reactive manner," said Richt, also the director of the university's Center of Excellence for Emerging and Zoonotic Animal Diseases, known as CEEZAD. "With COVID, every day something is new - what was correct yesterday, could be wrong today." Because of the rapid change of knowledge related to CoV, Richt and his collaborators wrote the article to stress importance of studying the ways that COVID-19 could spread between humans and animals. The scientists say that research should focus in several areas, including: The potential for companion animals, such as cats and dogs, to carry the virus; the economic and food security effects if the virus can spread among livestock and poultry; national security areas, especially among service

animals such as dogs that detect narcotics or explosives because COVID-19 is known to affect smell and cause hyposmia or anosmia.
Kansas State University
14 May 2020
Manhattan, KS

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Nearly quarter of a billion people in Africa will catch coronavirus and up to 190,000 could die

Health systems will struggle to cope without steps to stop spread of the virus, warn WHO experts. Nearly a quarter of a billion people across Africa will catch CoV during the1st year of the pandemic, and up to 190,000 of them will likely die, unless urgent action is taken to control the infection, reveals a predictive modelling study, accepted for publication in BMJ Global Health. These figures indicate a lower rate of exposure and viral spread than in other parts of the world, say the researchers. But the associated rise in hospital admissions, care needs, and impact on other health conditions in the region would severely strain limited health resources and worsen the impact of the virus, they warn. The WHO Africa region includes 47 countries, but excludes Djibouti, Egypt, Libya, Morocco, Somalia, Sudan and Tunisia. As of April 29, 45 countries had reported cases of SARS-CoV-2. Many mathematical models used to predict transmission and death rates in Africa have not adequately incorporated characteristics unique to the region and its individual countries. But these social, developmental, environmental and population health factors nevertheless affect the spread of the virus and the severity of COVID-19, explain the researchers.
BMJ
14-May-2020

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The newly emerged coronavirus did not spill over from scaly anteaters

Mammals known as scaly anteaters are natural hosts of coronaviruses but are not likely the direct source of the recent outbreak in humans, according to a study published May 14 in the open-access journal PLOS Pathogens by Jinping Chen of the Guangdong Institute of Applied Biological Resources, and colleagues. As noted by the authors, the large-scale surveillance of coronaviruses in these animals, called pangolins, could improve our understanding of the spectrum of coronaviruses circulating in the wild, and could help prevent and control emerging infectious diseases.
PLOS
14-May-2020

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Physicians brace for potential surge in COVID-19 cases

As health care workers begin to understand the scope of COVID-19 in their areas and how to respond to it, a new anxiety has set in: How long will the pandemic last? "The biggest stress is not knowing where we're going to go from here," Manu Jain, MD, pulmonary and critical care specialist at Northwestern Memorial Hospital, told Healio Primary Care. "Not knowing how this is going to evolve and how we're going to get back to any sense of normalcy is probably the most stressful part of this whole experience." Physicians on the front lines are often working long hours and caring for severely ill patients. Research published in JAMA Network Open showed high rates of depression (50.4%), anxiety (44.6%), insomnia (34%) and distress (71.5%) among health care workers in Wuhan, China, where the pandemic originated.
May 14, 2020
Healio

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COVID-19 disruption will lead to 28 million surgeries cancelled worldwide

Over 28 million elective surgeries across the globe could be cancelled as a result of the COVID-19 pandemic - leading to patients facing a lengthy wait for their health issues to be resolved, a new study reveals. The CovidSurg Collaborative has projected that, based on a 12-week period of peak disruption to hospital services due to COVID-19, 28.4 million elective surgeries worldwide will cancelled or postponed in 2020. The modelling study, published in the British Journal of Surgery, indicates that each additional week of disruption to hospital services will be associated with a further 2.4M cancellations. Led by researchers at the University of Birmingham, researchers collected detailed information from surgeons across 359 hospitals and 71 countries on plans for cancellation of elective surgery. This data was then statistically modelled to estimate totals for cancelled surgery across 190 countries (attached). The researchers project that worldwide 72.3% of planned surgeries would be cancelled through the peak period of COVID-19 related disruption. Most cancelled surgeries will be for non-cancer conditions. Orthopaedic procedures will be cancelled most frequently, with 6.3M orthopaedic surgeries cancelled worldwide over a 12-week period. It is also projected that globally 2.3M cancer surgeries will be cancelled or postponed.

University of Birmingham
14-May-2020

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Chest X-rays in emergency rooms can help predict severity of COVID-19 in young and middle-aged adults

Chest X-rays performed on young and middle-aged adults with COVID-19 when they arrive at the emergency room can help doctors predict who is at higher risk of severe illness and intubation, Mount Sinai researchers report. The first-of-its kind study, published in the May 14 issue of Radiology*, identifies which patients may need to be hospitalized and intubated based on the severity of CoV patterns in the lungs seen in the X-rays, using a unique scoring system to evaluate severity. The results could help physicians more quickly identify, triage, and aggressively treat these high-risk patients.*

The Mount Sinai Hospital / Mount Sinai School of Medicine
14 May 2020

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Hydroxychloroquine and azithromycin to treat COVID-19 clinical trial begins

A clinical trial has begun to evaluate whether the malaria drug hydroxychloroquine, given together with the antibiotic azithromycin, can prevent hospitalization and death from coronavirus disease 2019 (COVID-19). NIAID is sponsoring the trial, which is being conducted by the NIAID-funded AIDS Clinical Trials Group (ACTG). Teva Pharmaceuticals is donating medications for the study. The Phase 2b trial will enroll approximately 2,000 adults at participating ACTG sites across the US. Study participants must have confirmed infection with SARS-CoV-2, the virus that causes COVID-19, and be experiencing fever, cough and/or shortness of breath. The investigators anticipate that many of those enrolled will be 60 years of age or older or have a comorbidity associated with developing serious complications from COVID-19, such as cardiovascular disease or diabetes. Participants will be randomly assigned to receive short-term treatment with either hydroxychloroquine and azithromycin or matching placebos. People living with HIV and pregnant and breastfeeding women also are eligible to participate in the study. The first participant enrolled today in San Diego, CA.

By Press Release
May 14, 2020

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Treatment with interferon-α2b speeds up recovery of COVID-19 patients in exploratory study

Treatment with antivirals such as interferons may significantly improve virus clearance and reduce levels of inflammatory proteins in COVID-19 patients, according to a new study in Frontiers in Immunology. Researchers conducting an exploratory study on a cohort of confirmed COVID-19 cases in Wuhan found that treatment with interferon (IFN)-α2b significantly reduced the duration of detectable virus in the upper respiratory tract and reduced blood levels of interleukin(IL)-6 and C-reactive protein (CRP), two inflammatory proteins found in the human body. The findings show potential for the development of an effective antiviral intervention for COVID-19, which is an ongoing global pandemic caused by the novel CoV, SARS-CoV-2.

Frontiers
15-May-2020

https://protect2.fireeye.com/url?k=96fd49a1-caa950dd-96fd789e-0cc47adc5fa2-87b7d14066724e15&u=https://www.nature.com/articles/d41586-020-01430-5

Dogs caught coronavirus from their owners, genetic *analysis suggests*

But there's no evidence that dogs can pass the virus to people. *The first 2 dogs reported to have CoV probably caught the infection from their owners, say researchers who studied the animals and members of the infected households in Hong Kong. An analysis of viral genetic sequences from the dogs showed them to be identical to those in the infected people. Researchers suspected that the infection had been passed from the owners to the dogs, and the direct genomic link strongly supports that, says Malik Peiris, a virologist at the University of Hong Kong who led the study, which is published today in Nature[1]. The study showed no evidence that dogs can pass the infection to other dogs or people, but it is impossible to be certain in which direction the virus traveled "so we have to keep an open mind", says Peiris. Although the analysis confirms that people with COVID-19 can infect dogs, the probability of this happening is low, says Arjan Stegeman, a veterinary epidemiologist at Utrecht University in the Netherlands. In the study, only 2 of the 15 dogs who lived with infected people caught the disease. But other scientists say the possibility that pets might spread the virus between each other, and to people, needs to be properly investigated as part of managing future outbreaks.*

Nature
Smriti Mallapaty
14 May 2020

From: Dr. (b)(6)
Sent: Friday, May 15, 2020 3:59 PM
To: (b)(6)
Cc: Caneva, Duane (b)(6) (HMFP - Emergency Medicine) (b)(6) (DSHS) (b)(6) Dr. (b)(6) BOURNE, ALEXANDRA (b)(6) McDonald, Eric <Eric.McDonald@sdcounty.ca.gov>; Keim, MD MBA (b)(6) MD (b)(6) V.,M.D. (b)(6) S (b)(6) McGinn,

(b)(6) (b)(6) (b)(6) Kellermann (b)(6) (b)(6) (b) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) K. (b)(6) (b)(6) (b)(6) (b)(6) LLogandakar (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) DC <michelle.colby@usda.gov>; (b)(6) JtCIVtUSARMY (USA) (b)(6) (b)(6) (b)(6) MD (b)(6) Fantinato, Jessica (USDA.GOV) <jessica.fantinato@usda.gov>; Martin, Greg (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) KAUSHIK, SANGEETA (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Larry G (b)(6) Ignacio, Joselito (b)(6) (b)(6) Hassell, David (Chris) (OS/ASPR/IO) <David.Hassell@hhs.gov>; (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6)) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) WILKINSON, THOMAS (b)(6) WOLFE, HERBERT (b)(6) (b)(6) (b)(6) Marinissen, Maria (HHS/OS/OGA) <Maria.Marinissen@hhs.gov>; Sutter, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Krohmer, Jon (NHTSA) <jon.krohmer@dot.gov>; (b)(6) (APHMFP - Emergency Medicine) (b)(6) You, Edward H. (WMD) (FBI) (b)(6) (b)(6) (b)(6) W (b)(6)

Subject: Re: [External] Re: Red Dawn Posturing, Begin May 5, 12:50 EDT

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(b)(6) good point regarding community mitigation. Traffic starts to jam up again in Atlanta and many more people are going into their office. But yes, churches, schools are all closed and big events are canceled. I finished a few hundreds of business modeling and here's what I found.

1. Large group events remain difficult to open. The percentage of infection is roughly 7%-35% depending on the duration and because it is a sustained inside event, it is hard to maintain the fresh air circulation, the surface, and everything. It's cross-contamination every year. Any infection has high possibility of a hit to infect and it can be very broad in scope, depending on human behavior. Yes, university students are more proned because simply they are sick very often.

2. Across each State, there is no one-size-fit all, not within a State, and not inter-state either. What the models do show is that inter-dependence and cascading effects are certain (can be predicted very well) and hence must be taken into account as States are re-opening. The best they can do is opening strategically county-by-county, or city-by-city. And they will need to look over their shoulders on how their neigjhbours are doing as that will affect them.

3. Spitting, coughing, talking, anything to do with saliva You see the case a London transit worker died from covid-19 (five days from admitted to hospital to death) when a passenger claiming to have covid-19 on the track harrassed her by spitting on her. In US, there are people who do spit, tobacco users,

people who chew gums, or people who just spit (I saw people rolling down their car windows) and just do it. Singapore has installed fines on these actions many years ago. This habit still persists in US and this can be rather deadly in the wake of covid-19.

And public health should put restriction on spitting. If the state wants to maintain good public hygiene by deterrance, they can fine like Singapore.

Facemasks remain very critical. Essential workers must use facemasks and so are the citizens. And this should not be an option, but a must. I was surveying several stores this week - pets stores, groceries, and many employees do not use facemasks or any protecton at all and they do cough and of course they talk too. Here's a study in PNAS confirming why it is so important to avoid any droplets of saliva. I think employees need to do it to protect themselves and as customers, we do not want to receive little saliva droplets from cashiers or anyone.

https://www.pnas.org/content/early/2020/05/12/2006874117

And yes, eyes are very important to protect too. Sorry, no better news, here's *JAMA Ophthalmology* reporting tranmission through eyes,

https://protect2.fireeye.com/url?k=373a9301-6b6e8a7d-373aa23e-0cc47adc5fa2-a83e33d49eac538f&u=https://www.ajmc.com/newsroom/covid19-may-be-transmitted-through-the-eye-report-finds

So face shields are important too. Goggles are tight-fit and may be more protective. I remain a fan of the face shield especially seeing how the providers work at ease with them and are happier with it than the goggles. This is one of those cost-effective little thing that was invented and very welcome by providers.

4. Delay in tests, delay in test results, delay in everything on the graphs.
It is very hard to decipher all the testing data. First, cases are delayed in discovery. By the time the patient gets the test, there is a time-lag already. Then, it takes 2-5 days, or 7-9 days to get the results, and then posted. So everything is delay. In DeKalb, thousands of the tests never received any results back. So they won't show up anywhere. These are PUI, so their rate of positivity is high. Below are graphs from raw data, without any adjusttment in reporting etc.
Again mortality graph is one to focus.

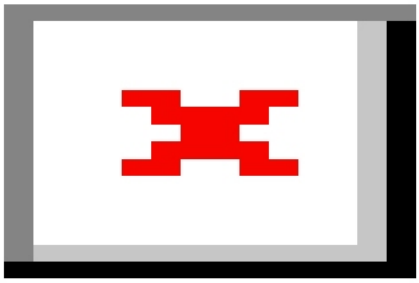


image.png

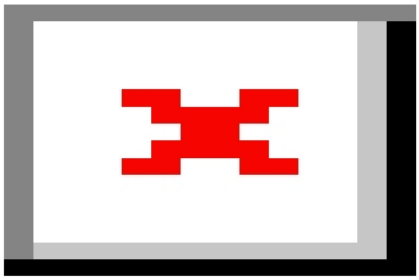
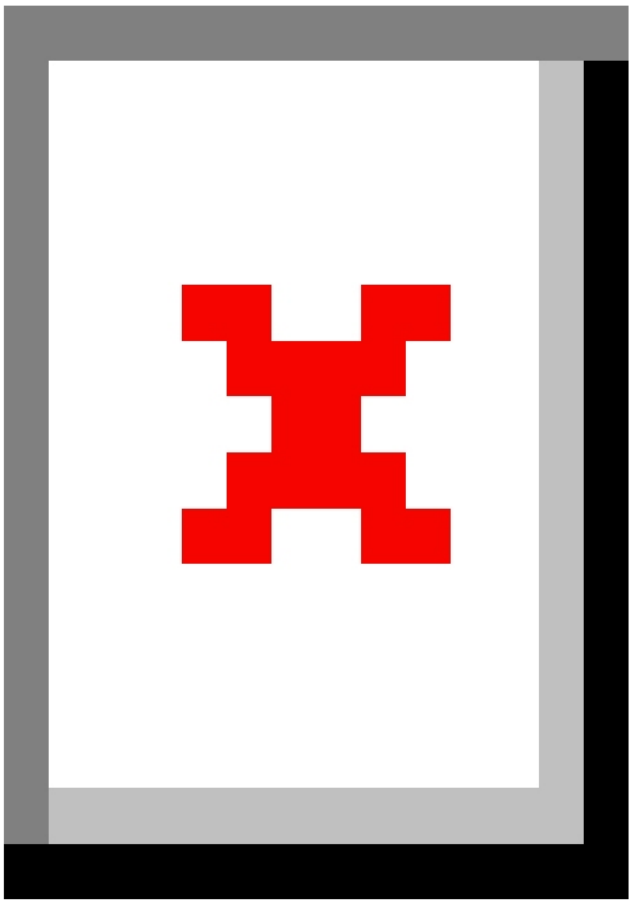
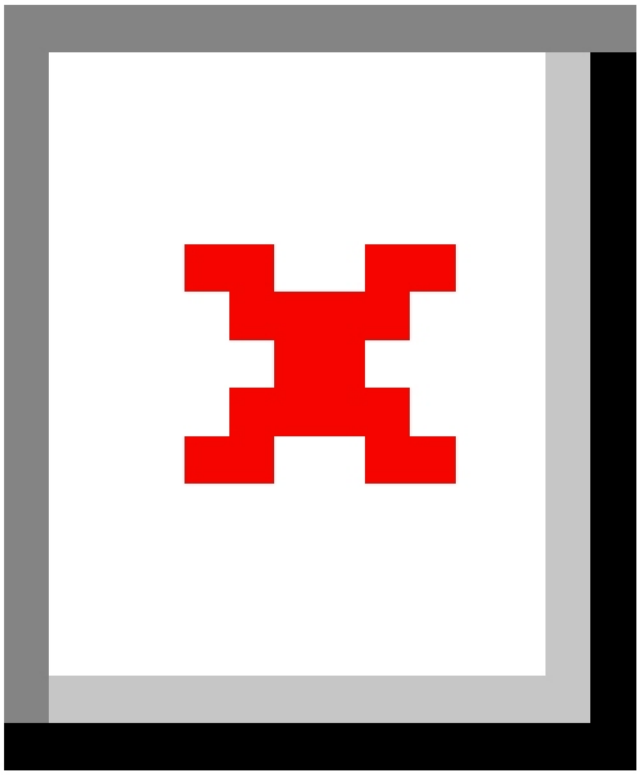


image.png

On Fri, May 15, 2020 at 9:24 AM (b)(6) (b)(6) (b)(6) wrote:
Monitoring Georgia.
First, raw data of cases and deaths reported each day with 7-day moving averages of cases, deaths, tests performed, and % positive (see attached Excel file). I already shared the LTC data from Georgia.

Second, the data from GA Public Health.





Third. Take a look at the data on https://protect2.fireeye.com/url?k=12854aa1-4ed153dd-12857b9e-0cc47adc5fa2-0391992a9ee899d4&u=https://dashboards.c19hcc.org/ for Georgia.

As I have said earlier, although we are calling what is happening as reopening, what in fact is happening is we are transitioning to the 2007 Community Mitigation Guidance (really TLC). Infected or sick individuals are still being isolated at home, I assume household contacts are still being advised to home quarantine, schools/universities/pre-school/daycare are still closed, and we continue to leverage social distancing strategies (telework; social distancing in restaurants, retail; churches closed, large gatherings canceled); and we are also wearing face masks (at least the majority of people are wearing face masks).

Sent from Mail for Windows 10

From: (b)(6) (b)(6)
Sent: Friday, May 15, 2020 7:11 AM
To: Caneva, Duane; Dr. (b) (b) (b)(6) (b)(
Cc: (b)(6) (HMFP - Emergency Medicine); (b)(6) (b)(6 (b)((b)(6) (b)(6) (DSHS); Dr. (b) (b) BOURNE, ALEXANDRA; (b)((b)(6) (b)(6) (b)(6) McDonald, Eric; (b)(Keim, MD MBA; (b)(6 (b)(6) (b)((b)(6) (b) (b)(6) MD; (b)(6) (b)(6) V.,M.D.; (b)(6), (b)((b)((b)(S; (b)(6) (b)(McGinn, (b)((b)(6) (b)(6) Kellermann; (b)(6) (b) (b)(6 (b)((b)(6) (b)(6) Eastman, (b)(6) (b)(6) (b)(6) (b)(6 (b)(6 (b)(6) LLogandakar; Walters, (b)(6) (b)(6) (b)((b)(6) DC; (b)(6) JtCIVtUSARMY (USA); (b)((b)(6) MD; Fantinato, Jessica (USA.GOV); Martin, Greg (b)(6) (b)((b)(6) (b)(, (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) (b)(6) (b)(6) (b)(6 (b)(6) (b)((b)(6) (b)(6) (b)(6) Larry G; Ignacio, Joselito; David; Hassell, David (Chris) (OS/ASPR/IO); (b) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6)); (b)(6) (b)(6) (b)(6, (b)(6 (b)((b)(6) WILKINSON, THOMAS; WOLFE, HERBERT; (b)(6) Marinissen, Maria (HHS/OS/OGA); Sutter, Mark; (b)(6) (b)(6) (b)((b)((b) (b)(6 Krohmer, Jon (NHTSA); (b)(6) (APHMFP - Emergency Medicine); You, Edward H. (WMD) (FBI); (b)(6) ((b)(6 W
Subject: RE: [External] Re: Red Dawn Posturing, Begin May 5, 12:50 EDT

LTC data by state. I have 3 data points (Apr 23, May 7 and May 14). See 1st spreadsheet for summary table. This virus is steadily moving through our LTC population in the US. Recall the estimates of 2.1M Americans who are residents in nursing homes or residential care. With 150K known cases and 30K deaths, this equates to about a 7% attack rate with a 20% fatality rate (I would assume we are learning about most cases given that nursing homes are now testing all residents).

If you are interested in learning more about long term care in the US. https://protect2.fireeye.com/url?k=c15ff247-9d0beb3b-c15fc378-0cc47adc5fa2-fc81f633b2991d7a&u=https://www.cdc.gov/nchs/data/series/sr_03/sr03_43-508.pdf There are 8.3M Americans receiving long term care services (not necessarily receiving services at a long term care facility).

Sent from Mail for Windows 10

From: (b)(6) (b)(6)
Sent: Thursday, May 14, 2020 10:55 AM
To: Caneva, Duane; Dr. (b) (b) (b)(6) (b)(

Cc: (b)(6) (HMFP - Emergency Medicine); (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (DSHS); Dr. (b) (b) BOURNE, ALEXANDRA; (b)(6) (b)(6) (b)(6) (b)(6) McDonald, Eric; (b)(6) Keim, MD MBA; (b)(6) (b)(6) (b)(6) (b)(6) (b) (b)(6) MD; (b)(6) (b)(6) V.,M.D.; (b)(6), (b)(6) (b)(6) (b)(6) S; (b)(6) (b)(6) McGinn, (b)(6) (b)(6) (b)(6) Kellermann; (b)(6) (b) (b)(6) (b)(6) (b)(6) (b)(6) Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) LLogandakar; Walters, (b)(6) (b)(6) (b)(6) (b)(6) DC; (b)(6) JtCIVtUSARMY (USA); (b)(6) (b)(6) MD; Fantinato, Jessica (USDA.GOV); Martin, Greg (b)(6) (b)(6) (b)(6) (b)(6), (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Larry G; Ignacio, Joselito; David; Hassell, David (Chris) (OS/ASPR/IO); (b) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6)); (b)(6) (b)(6) (b)(6), (b)(6) (b)(6) (b)(6) WILKINSON, THOMAS; WOLFE, HERBERT; (b)(6) Marinissen, Maria (HHS/OS/OGA); Sutter, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b) (b)(6) Krohmer, Jon (NHTSA); (b)(6) (APHMFP - Emergency Medicine); You, Edward H. (WMD) (FBI); (b)(6) (b)(6) W
Subject: RE: [External] Re: Red Dawn Posturing, Begin May 5, 12:50 EDT

We have been following any reports of reported prevalence. Spain recently reported a prevalence of 5%. Spain has a death rate of 577 deaths per million (as of yesterday). It is also at the flat part of its curve (even 2 weeks ago the death rate was 517 deaths per million). Assuming Spain has a prevalence of 5% and a death rate of 577 deaths per million, it would equate to a CFR of 1.16%. So a little higher than the CFR we have been estimating.

We haver been roughly estimating prevalence for all the states (assuming a CFR of 0.9%). But given the high percentage of nursing home deaths in a number of state (>50%), we need to do this cautiously because in a state where a high % of the deaths are in long term care, we could be overestimating prevalence.

Attached is a crude estimate of prevalence for the states. We assumed a CFR of 0.9% and time from infection onset to death of about 2 weeks. We used Apr 27 as roughly 2 weeks ago (it was the only data readily available at hand in a table) to estimate a case ascertainment rate. This is all a little crude, but helps to place some of these raw numbers of confirmed cases in perspective. We added a column of the data we have from May 7 on the % of total COVID deaths coming from LTC (those states with a % >50% are highlighted in red).

Sent from Mail for Windows 10

From: Caneva, Duane
Sent: Thursday, May 14, 2020 9:49 AM
To: Dr. (b) (b) (b)(6) (b)(6)
Cc: (b)(6) (HMFP - Emergency Medicine); (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (DSHS); Dr. (b) (b) BOURNE, ALEXANDRA; (b)(6) (b)(6) (b)(6) (b)(6) McDonald, Eric; Mark Keim, MD MBA; (b)(6) (b)(6) (b)(6) (b)(6) (b) (b)(6) MD; (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) (b)(6) (b)(6) S; (b)(6) (b)(6) McGinn, (b)(6) (b)(6) (b)(6) Kellermann; (b)(6) (b) (b)(6) (b)(6) (b)(6) (b)(6) Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Valters, (b)(6) (b)(6) (b)(6) (b)(6) DC; (b)(6) JtCIVtUSARMY (USA); (b)(6) (b)(6) MD; Fantinato, Jessica (USDA.GOV); Martin, Greg (b)(6)

(b)((b)(6) (b)(6 (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)((b)(6) (b)(6) (b)(6) Larry G; Ignacio, Joselito; (b)(6 Hassell, (b)((Chris) (OS/ASPR/IO); (b) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6)); (b)(6) (b)(6) (b)(6 (b)(6 (b)((b)(6) WILKINSON, THOMAS; WOLFE, HERBERT; (b)(6) Marinissen, Maria (HHS/OS/OGA); Sutter, (b)((b)(6) (b)(6) (b)((b)((b) (b)(6 Krohmer, Jon (NHTSA); (b)(6) (APHMFP - Emergency Medicine) (b)(6) (b)(6 ((b)(6 W
Subject: RE: [External] Re: Red Dawn Posturing, Begin May 5, 12:50 EDT

Some attachments of interest.

C19HCC Dashboard: https://protect2.fireeye.com/url?k=8c51baf9-d005a385-8c518bc6-0cc47adc5fa2-61649cccd0cf52af&u=https://dashboards.c19hcc.org/
C19HCC PPE Dashboard: https://protect2.fireeye.com/url?k=db4a14c5-871e0db9-db4a25fa-0cc47adc5fa2-f917f0177f119fac&u=https://dashboards.c19hcc.org/ppe

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From: Dr. (b) (b) (b)(6)
Sent: Wednesday, May 13, 2020 8:21 PM
Subject: Re: [External] Re: Red Dawn Posturing, Begin May 5, 12:50 EDT

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Here's the testing situation. I feel very sorry for Brazil. And my colleagues with whom I work on vaccine immunogenicity prediction are in distressed. But they have very little testing kits and very little testing power. So they are testing only the sick patients and many are positive.

On Wed, May 13, 2020 at 8:04 PM Dr. (b) (b) (b)(6) wrote:

> Bill, we must understand, US is still in the discovery phase as far as confirmed positive cases, because we are catching up on test still. I believe we have improved over time, so yes, you (b) see more confirmed cases. It is actually more important to look at fatality. Granted, counting may not be perfect. My feeling is that no country is perfect in the counting because it simply is a huge number of bodies all needed attention at a very short period of time. It is overwhelming. So everyone undercounts. Here's a little summary. You notice Positive/capita - for Iceland is very high, because they have tested a very large percentage of their small population. We are still behind in testing, And we need to test a lot more. So many people are asymptomatic, not knowing them is a disadvantage to us. Recall, not every positive case needs hospitalization. So that number as close to reality is very important because it gives us a sense of how infectious SARS-CoV-2 is. It also allows us to estimate better the CFR.

Country	Reported death	Reported positive	% death/positive	Population (M)	fatality/capita	Positive/capita
USA	83963	1411488	5.948545	334	251.3862	4226.012
Spain	27104	271095	9.997971	46.9	577.9104	5780.277
Italy	31106	222104	14.00515	50.63	614.3788	4386.806

Germany	7792	173824	4.482695	83.02	93.8569	2093.761
France	27074	140734	19.23771	66.99	404.1499	2100.821
Israel	264	16548	1.595359	8.884	29.71634	1862.674
Iceland	10	1802	0.554939	0.364134	27.46242	4948.728
(b)	33186	229705	14.44723	66.65	497.9145	3446.437

On Wed, May 13, 2020 at 7:43 PM (b)(6) (b)((b)(6) wrote:

(b)

As I understand the data, of the large countries with major impacts, 65% of the cases over last week came from the following 5 countries the U.S. (> 197,600 cases; 603 cases/million), Russia (> 70,600 cases; 489 cases/million), the U.K. (> 35,400 cases; 533 cases/million), Brazil (> 34,500 cases; 164 cases/million), and Peru (> 21,500 cases; 673 cases/million). I don't have the comparable fatality data at hand, but what I've seen has been similar.

I know that "cases" as a datapoint to track is crap because it's based on testing and the populace's threshold for seeking care, but still, we are well above the average on a rate basis. Why is this? I have a hard time believing it's just timing on application of NPIs.

-Bill

From: Dr. (b) (b) (b)(6)
Sent: Wednesday, May 13, 2020 7:34 PM
To: (b)(6) (b)((b)(6)
Cc: (b)(6) (HMFP - Emergency Medicine) (b)(6) Caneva, Duane (b)(6) (b)(6) (b)((b)(6) (b)((b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (DSHS) (b)(6) Dr. (b) (b) (b)(6) (b)(6) BOURNE, ALEXANDRA (b)(6) (b)((b)(6) (b)(6) (b)(6) (b)(6) (b)(6) McDonald, Eric <Eric.McDonald@sdcounty.ca.gov>; (b)(Keim, MD MBA (b)(6) (b)((b)(6) (b)(6) (b) (b)(6) MD (b)(6) (b)(6) (b)(6) V.,M.D. (b)(6) (b)(6) (b) (b)(6) (b)((b)(S (b)(6) (b)(6) (b) (b)(6) McGinn, (b)((b)(6) (b)(6) (b)(6) Kellermann (b)(6) (b)(6) (b) (b)(6) (b)(6 (b)((b) (b)(6) (b)(6) (b)(6 (b)(6) Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b) (b)(6) (b)(6) (b)(6) (b)(6) LLogandakar (b)(6) Walters, (b)(6) (b)(6) (b)(6) (b)((b)(6) (b)(6) DC <michelle.colby@usda.gov>; (b)(6) JtCIVtUSARMY (USA) (b)(6) (b)((b)(6) MD (b)(6) Fantinato, Jessica (USDA.GOV) <jessica.fantinato@usda.gov>; Martin, Greg (b)(6) (b)(6) (b)((b)(6) (b)(6) (b)(6 (b)(6) (b)(6) KAUSHIK, SANGEETA (b)(6) (b)(6) (b)(6) <CHRISALLEN (b)(6) (b)(6) (b)(6) (b)((b)(6) (b)(6) (b) (b)(6) (b)(6) (b)(6) (b)(6)

(b)(6) Larry G (b)(6) Ignacio, Joselito (b)(6) (b)(6) Hassell, (b)(6) (Chris) (OS/ASPR/IO) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6)) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) WILKINSON, THOMAS (b)(6) WOLFE, HERBERT (b)(6) (b)(6) (b)(6) Marinissen, Maria (HHS/OS/OGA) <Maria.Marinissen@hhs.gov>; Sutter, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Krohmer, Jon (NHTSA) <jon.krohmer@dot.gov>; (b)(6) (APHMFP - Emergency Medicine) (b)(6) You, Edward H. (WMD) (FBI) (b)(6) (b)(6) (b)(6) W (b)(6)
Subject: Re: [External] Re: Red Dawn Posturing, Begin May 5, 12:50 EDT

Bill, may I ask you how you calculate your values? I am using fatality per capita and infection per capita, we are not the highest. Is that what you are thinking? I must have misunderstood your question.

On Wed, May 13, 2020 at 7:24 PM (b)(6) (b)(6) (b)(6) wrote:

> Does anyone have insight as to why the US has the highest case and fatality rate of any large country. I haven't seen any good explanation of that. It's not that we have so much most inbound international travelers (France exceeds the US on an absolute basis – not just rate, for example, and many countries far exceed us for international arrival rates).
>
> While many feel we should have implemented NPIs earlier and harder, I don't get the sense that we were that far behind the rest of the world to explain the case and fatality rate differences.
>
> Our Chinese arrival rate compared to the rest of the world is hard to figure (the data is there, but I haven't tried to crank it).
>
> With the exception of some inner-cities, we are not a population dense as other major developed countries.
>
> So what was it (and am just missing scholarly discussion somewhere about this huge discrepancy). Everyone knew in detail why Italy blew up, but why us?
>
> (b)(6)

From: Dr. (b)(6) (b)(6)
Sent: Wednesday, May 13, 2020 5:15 PM
To: (b)(6) (HMFP - Emergency Medicine) (b)(6)
Cc: Caneva, Duane (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) <cmecher@charter.net>; (b)(6) (DSHS) (b)(6)

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Subject: Re: [External] Re: Red Dawn Posturing, Begin May 5, 12:50 EDT

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This looks very nice in the tracing and connectivity of all the cases. I assume the genetic analysis reported recently regarding origin of the US cases will give us such type of information.

(b)(6) -
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On Wed, May 13, 2020 at 4:05 PM (b)(6) (HMFP - Emergency Medicine) <gciotton@bidmc.harvard.edu> wrote:

This is terrific! Thanks Duane

Greg

Sender:	Caneva, Duane (b)(6)
Recipient:	Dr. (b) (b) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b) (b)(6) (b)(6) (HMFP - Emergency Medicine) (b)(6) (b)(6) (b) (b)(6) (b) (b)(6) (b)(6) (b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=user4629b831 (b)(6) Dr. (b) (b) (b)(6) BOURNE, ALEXANDRA (b)(6) (b) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) eric.mcdonald@sdcounty.ca.gov /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=04b8a65f67f9453b9749d3d366481f53-Guest_bef32 <eric.mcdonald@sdcounty.ca.gov>; (b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=474b01361a8b4944a39afbdad800591e-Guest_bea05 (b)(6) (b)((b)((b)(6) (b) (b)(6) (b)(6) (b) (b)(6) MD (b)(6) (b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=20f0e9a3ebeb4ef99d30a96386fb2627-Guest_945f5 (b)(6) (b)(6) (b) (b)(6) (b)((b)(S (b)(6) (b)(6) (b) (b)(6) McGinn, (b) (b)(6) (b)(6) (b)(Kellermann (b)(6) (b)(6) (b) (b)(6) (b)((b) (b)(6) (b)(6) (b)((b)(6) Eastman, (b)(6) (b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=d314e75d55434b198c36e0c981e6623d-Contact_753 (b)(6) (b)(6) (b)(6) (b)(6) (b)((b)((b)(6) LLogandakar (b)(6) Walters, (b)(6) (b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group (b)(6)

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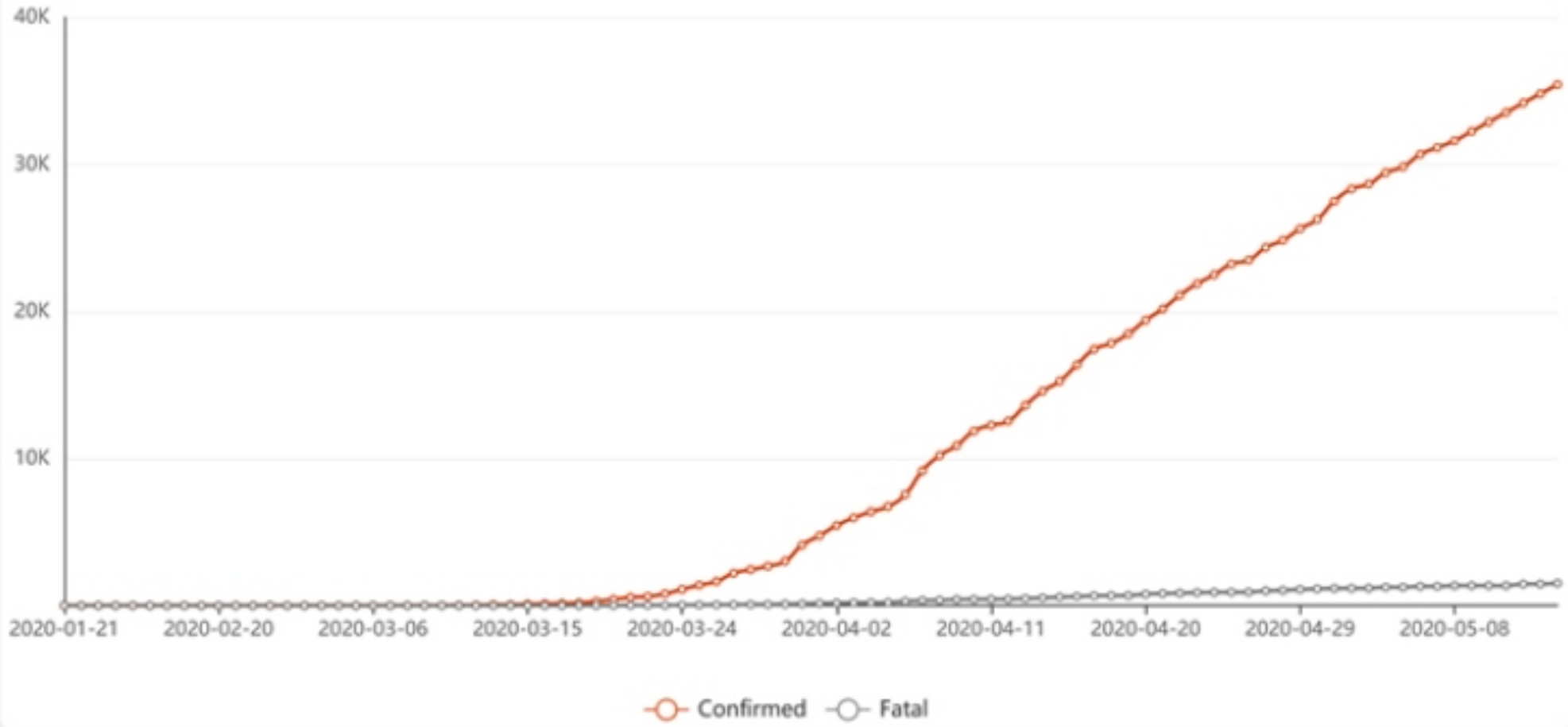

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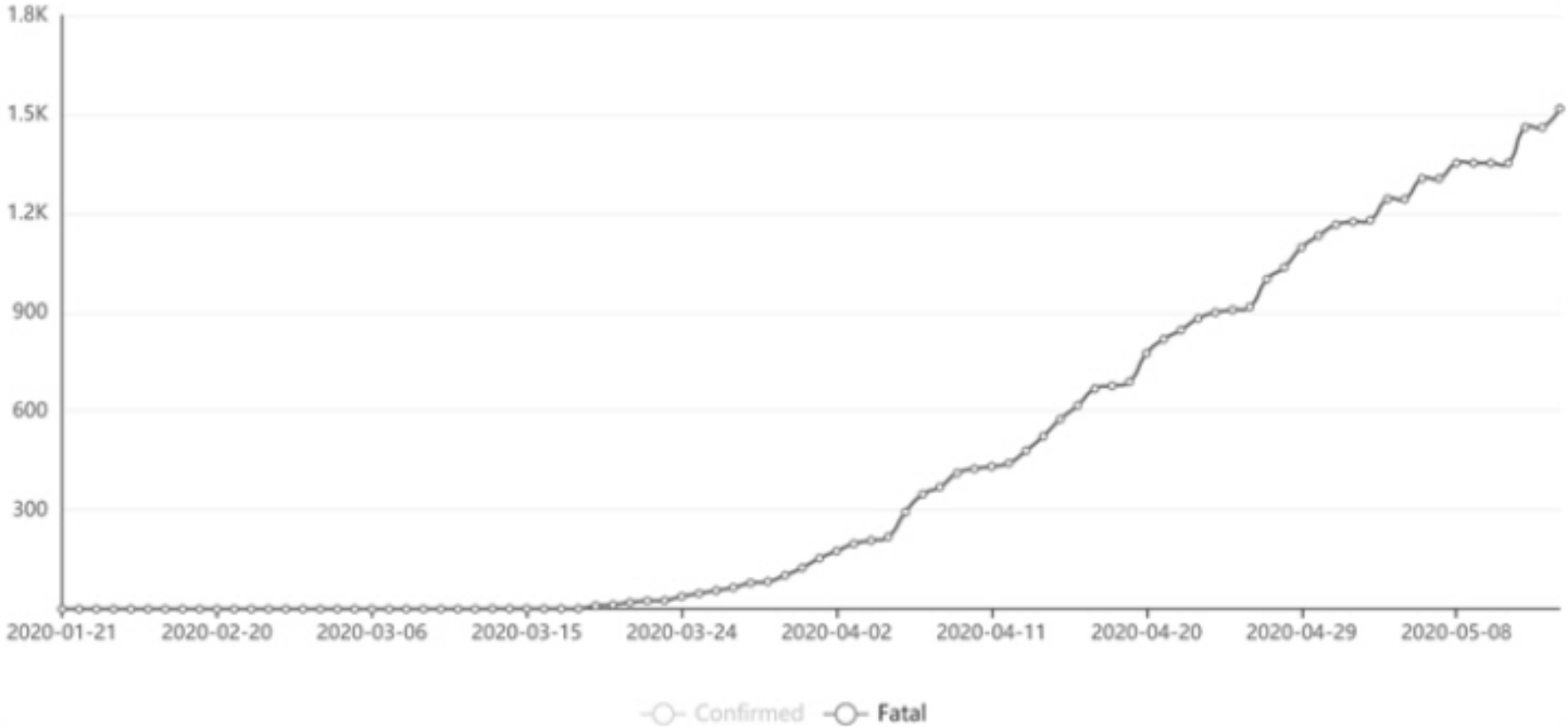
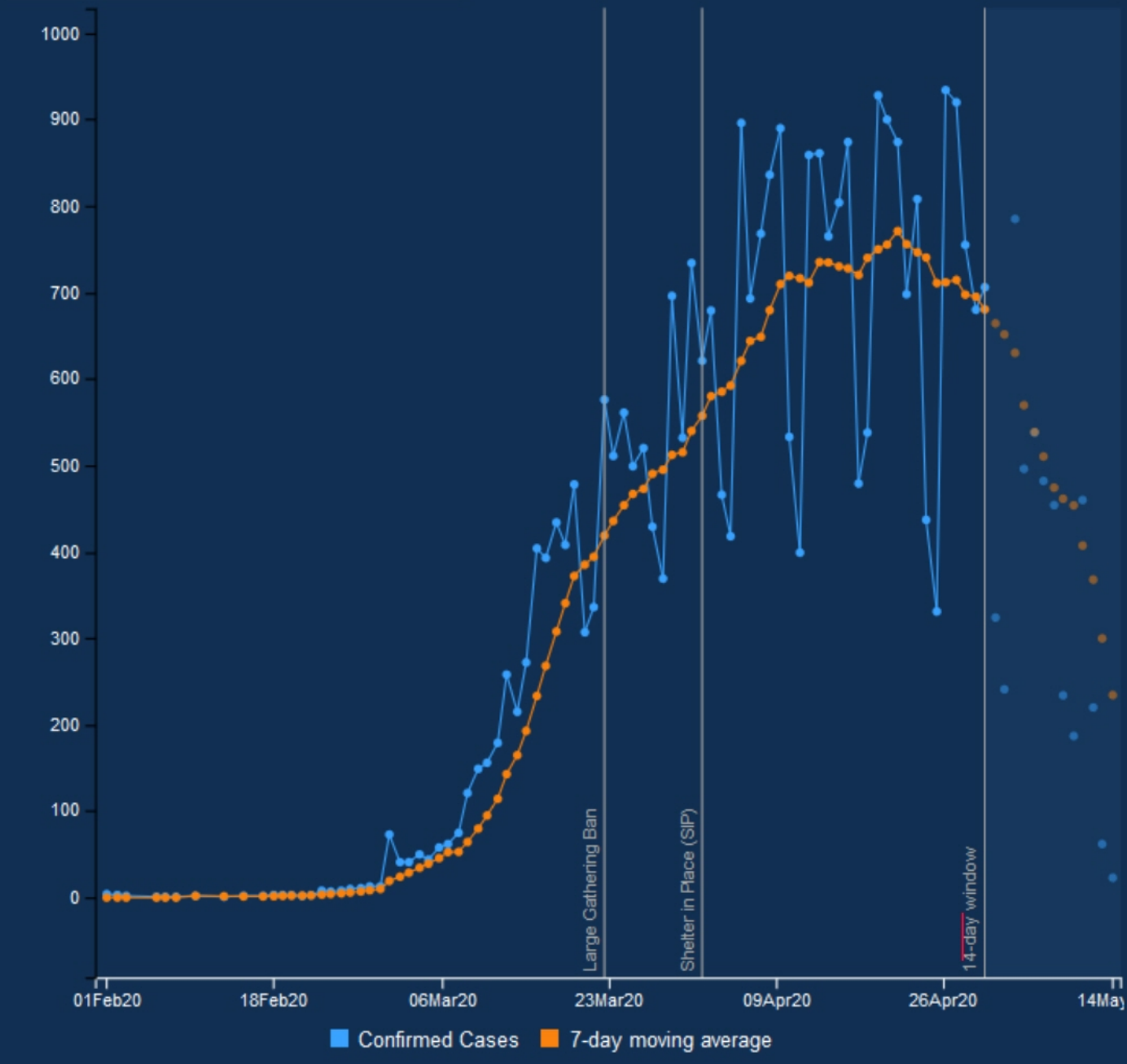


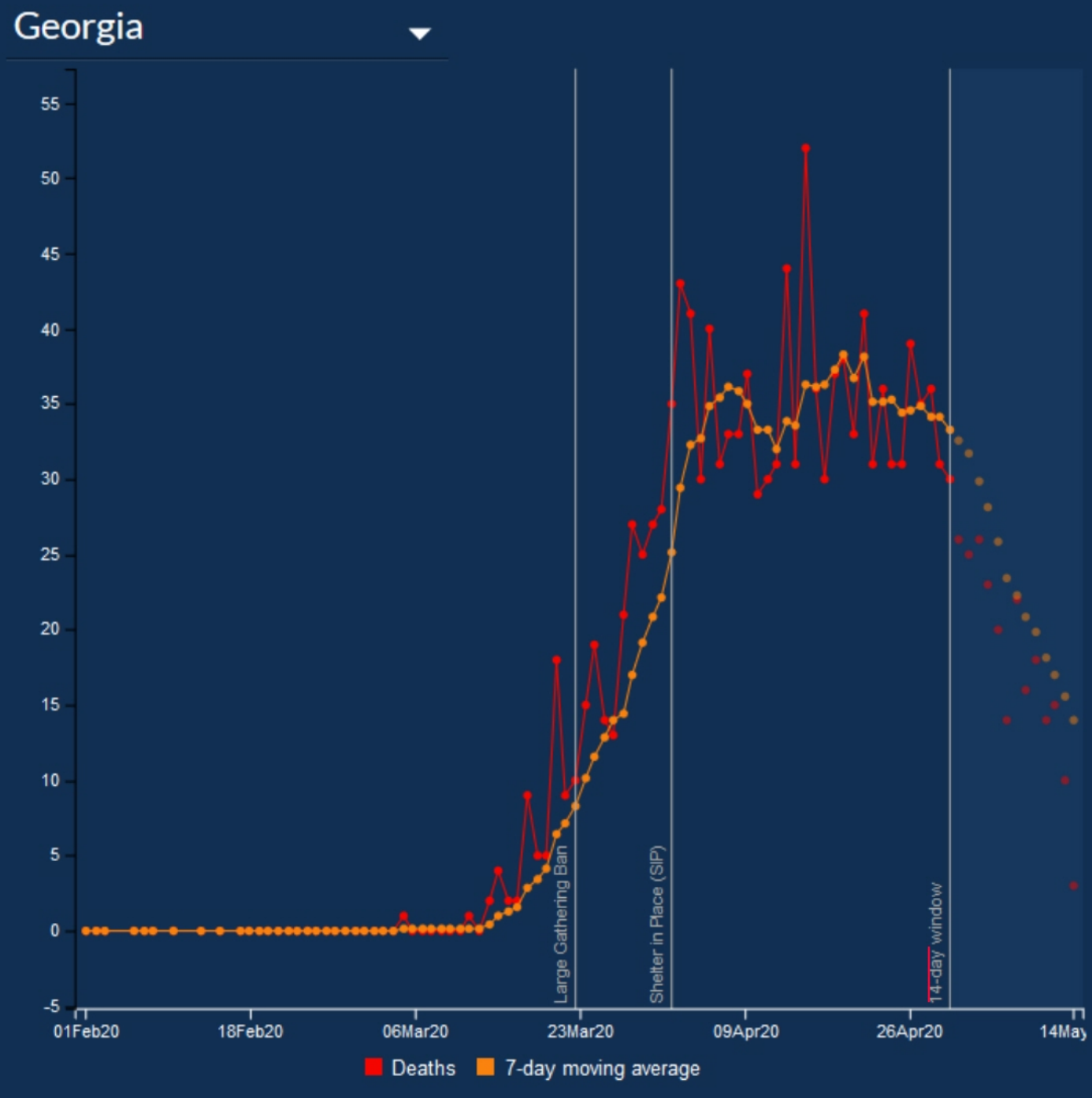
COVID-19 Cases Over Time

Georgia



** 14-day window – Confirmed cases over the last 14 days may not be accounted for due to illnesses yet to be reported or test results may still be pending.*

COVID-19 Deaths Over Time



DHS SCIENCE AND TECHNOLOGY

Master Question List for COVID-19 (caused by SARS-CoV-2)

Weekly Report

12 May 2020

For comments or questions related to the contents of this document, please contact the DHS S&T Hazard Awareness & Characterization Technology Center at (b)(6)

Homeland Security

Science and Technology

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FOREWORD

The Department of Homeland Security (DHS) is paying close attention to the evolving Coronavirus Infectious Disease (COVID-19) situation in order to protect our nation. DHS is working very closely with the Centers for Disease Control and Prevention (CDC), other federal agencies, and public health officials to implement public health control measures related to travelers and materials crossing our borders from the affected regions.

Based on the response to a similar product generated in 2014 in response to the Ebolavirus outbreak in West Africa, the DHS Science and Technology Directorate (DHS S&T) developed the following "master question list" that quickly summarizes what is known, what additional information is needed, and who may be working to address such fundamental questions as, "What is the infectious dose?" and "How long does the virus persist in the environment?" The Master Question List (MQL) is intended to quickly present the current state of available information to government decision makers in the operational response to COVID-19 and allow structured and scientifically guided discussions across the federal government without burdening them with the need to review scientific reports, and to prevent duplication of efforts by highlighting and coordinating research.

The information contained in the following table has been assembled and evaluated by experts from publicly available sources to include reports and articles found in scientific and technical journals, selected sources on the internet, and various media reports. It is intended to serve as a "quick reference" tool and should not be regarded as comprehensive source of information, nor as necessarily representing the official policies, either expressed or implied, of the DHS or the U.S. Government. DHS does not endorse any products or commercial services mentioned in this document. All sources of the information provided are cited so that individual users of this document may independently evaluate the source of that information and its suitability for any particular use. This document is a "living document" that will be updated as needed when new information becomes available.

Table of Contents

Infectious Dose – How much agent will make a healthy individual ill? 3
The human infectious dose of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is unknown by all exposure routes. SARS-CoV-2 is the cause of coronavirus disease 19 (COVID-19).
The correlation between the infectious dose and symptom severity is unknown.
Identifying the infectious dose for humans by the various routes through which we become infected is critical to the effective development of computational models to predict risk, develop diagnostics and countermeasures, and effective decontamination strategies. Animal studies are a plausible surrogate.
Transmissibility – How does it spread from one host to another? How easily is it spread? 4
SARS-CoV-2 is passed easily between humans, likely through close contact with relatively large droplets and possibly through smaller aerosolized particles.
Individuals can transmit SARS-CoV-2 to others before they have symptoms.
Undetected cases play a major role in transmission, and most cases are not reported.
Identifying the contribution of asymptomatic or pre-symptomatic transmission is important for implementing control measures. Additionally, the relative contributions of different infection sources – fomites, droplets, aerosols, and potentially feces – are unknown.
Host Range – How many species does it infect? Can it transfer from species to species? 5
SARS-CoV-2 is closely related to other coronaviruses circulating in bats in Southeast Asia. Previous coronaviruses have passed through an intermediate mammal host before infecting humans, but the identity of the SARS-CoV-2 intermediate host is unknown.
SARS-CoV-2 uses the same receptor for cell entry as the SARS-CoV-1 coronavirus that circulated in 2002/2003.
To date, mice, ferrets, mink, hamsters, cats, and primates have been shown to be susceptible to SARS-CoV-2 infection. It is unknown whether these animals can transmit infection to humans.
Several animal models have been developed to recreate human-like illness, though to date they have been infected with high dose exposures. Lower dose studies may better replicate human disease acquisition.
Incubation Period – How long after infection do symptoms appear? Are people infectious during this time? 6
The majority of individuals develop symptoms within 14 days of exposure. For most people, it takes at least 2 days to develop symptoms, and on average symptoms develop 5 days after exposure. Incubating individuals can transmit disease for several days before symptom onset. Some individuals never develop symptoms but can still transmit disease.
The incubation period is well-characterized. Patients may be infectious, however, for days before symptoms develop.
Clinical Presentation – What are the signs and symptoms of an infected person? 7
Many COVID-19 cases are asymptomatic. Most symptomatic cases are mild, but severe disease can be found in any age group.[6] Older individuals and those with underlying medical conditions are at higher risk of serious illness and death.
The case fatality rate varies substantially by patient age and underlying comorbidities.
Additional studies on vulnerable subpopulations are required.
Children are susceptible to COVID-19,[99] though generally show milder[66, 204] or no symptoms.
The true case fatality rate is unknown, as the exact number of cases is uncertain. Testing priorities and case definitions vary by location. The proportion of asymptomatic infections is not known.
Protective Immunity – How long does the immune response provide protection from reinfection? 8
Infected patients show productive immune responses, however more data is needed.
Currently, there is no evidence that recovered patients can be reinfected with SARS-CoV-2.
Understanding the duration of protective immunity is limited by small sample sizes. Animal models are plausible surrogates.
Additional research to quantify the risk of reinfection after weeks, months, and years is needed.
Clinical Diagnosis – Are there tools to diagnose infected individuals? When during infection are they effective? 9
Diagnosis relies on identifying the genetic signature of the virus in patient nose, throat, or sputum samples. These tests are relatively accurate. Confirmed cases are still underreported.
Validated serological (antibody) assays are being developed to help determine who has been exposed to SARS-CoV-2.
Serological evidence of exposure does not indicate immunity.

In general, PCR tests appear to be sensitive and specific, though confirmation of symptoms via chest CT is recommended.
The sensitivity and specificity of serological testing methods is variable, and additional work needs to be done to determine factors that affect test accuracy.
Medical Treatments – Are there effective treatments?.....10
Treatment for COVID-19 is primarily supportive care including ventilation if necessary.[128, 215] Numerous clinical trials are ongoing, but results are preliminary.[30,79] Several drugs show efficacy.
Remdesivir shows promise for reducing symptom duration in humans.[236]
Hydroxychloroquine is associated with elevated risk of cardiac arrhythmias and provides limited to no clinical benefit at this point in time.
Other pharmaceutical interventions are being investigated.
Additional clinical trial results are being released, and data from these trials are needed.
Vaccines – Are there effective vaccines?.....11
Work is ongoing to develop a SARS-CoV-2 vaccine in human and animal trials. No preliminary results are available.
Phase 2 Trials (initial testing for efficacy, continued testing for safety):
No preliminary vaccine results for humans have been released.
Non-pharmaceutical Interventions – Are public health control measures effective at reducing spread?.....12
Broad-scale control measures such as stay-at-home orders are effective at reducing movement, and modeling shows evidence that they reduce transmission.
The effect of relaxing control measures is unknown, and research is needed to help plan for easing of restrictions.
As different US states have implemented differing control measures at various times, a comprehensive analysis of social distancing efficacy has not yet been conducted.
Environmental Stability – How long does the agent live in the environment?.....13
SARS-CoV-2 can persist on surfaces for at least 3 days and on the surface of a surgical mask for up to 7 days depending on conditions. If aerosolized intentionally, SARS-CoV-2 is stable for at least several hours. The seasonality of COVID-19 transmission is unknown.
Additional testing on SARS-CoV-2, as opposed to surrogate viruses, is needed to support initial estimates of stability.
Decontamination – What are effective methods to kill the agent in the environment? 14
Soap and water, as well as common alcohol and chlorine-based cleaners, hand sanitizers, and disinfectants are effective at inactivating SARS-CoV-2 on hands and surfaces.
Methods for decontaminating N95 masks have been approved by the FDA under an Emergency Use Authorization (EUA).
Additional decontamination studies, particularly with regard to PPE and other items in short supply, are needed.
PPE – What PPE is effective, and who should be using it?.....15
The effectiveness of PPE for SARS-CoV-2 is currently unknown, and data from other related coronaviruses are used for guidance. Healthcare workers are at high risk of acquiring COVID-19, even with recommended PPE.
Most PPE recommendations have not been made on SARS-CoV-2 data, and comparative efficacy of different PPE for different tasks (e.g., intubation) is unknown. Identification of efficacious PPE for healthcare workers is critical due to their high rates of infection.
Forensics – Natural vs intentional use? Tests to be used for attribution. 16
All current evidence supports the natural emergence of SARS-CoV-2 via a bat and possible intermediate mammal species. Identifying the intermediate species between bats and humans would aid in reducing potential spillover from a natural source. Wide sampling of bats, other wild animals, and humans is needed to address the origin of SARS-CoV-2.
Genomics – How does the disease agent compare to previous strains? 17
Current evidence suggests that SARS-CoV-2 accumulates substitutions and mutations at a similar rate as other coronaviruses. Mutations and deletions in specific portions of the SARS-CoV-2 genome have not been linked to any changes in transmission or disease severity, though modeling work is attempting to identify possible changes.
Research linking genetic changes to differences in phenotype (e.g., transmissibility, virulence, progression in patients) is needed.

SARS-CoV-2 (COVID-19)	Infectious Dose – How much agent will make a healthy individual ill?
What do we know?	**The human infectious dose of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is unknown by all exposure routes. SARS-CoV-2 is the cause of coronavirus disease 19 (COVID-19).** *Work using SARS-CoV-2* • A total dose of approximately 700,000 plaque-forming units (PFU) of the novel coronavirus SARS-CoV-2 infected cynomolgus macaques via combination intranasal and intratracheal exposure (10^6 $TCID_{50}$ total dose).[274] Macaques did not exhibit clinical symptoms, but virus was shed from the nose and throat.[274] • Rhesus macaques are effectively infected with SARS-CoV-2 via the ocular conjunctival and intratracheal route at a dose of approximately 700,000 PFU (10^6 $TCID_{50}$).[96] • Rhesus macaques infected with 2,600,000 $TCID_{50}$ of SARS-CoV-2 by the intranasal, intratracheal, oral and ocular routes combined recapitulate moderate disease observed in the majority of human cases.[232] • Rhesus and cynomolgus macaques showed clinical infections at doses of 4.75 x 10^6 PFU (SARS-CoV-2 delivered through several routes), while common marmosets developed mild infections when exposed to 1.0 x 10^6 PFU intranasally.[204] • Ferrets infected with 316,000 $TCID_{50}$[163] or 600,000 $TCID_{50}$[267] of SARS-CoV-2 by the intranasal route show similar symptoms to human disease.[163, 267] Uninfected ferrets in direct contact with infected ferrets test positive and show disease as early as 2 days post-contact.[163] In one study, direct contact was required to transfer infection between ferrets,[163] however, transmission without direct contact was found in another study.[267] • Syrian Golden Hamsters infected with 100,000 PFU via the intranasal route closely resemble human respiratory infection. Uninfected hamsters in close contact with infected hamsters show symptoms within 4 days of exposure.[63] • Domestic cats exposed to 100,000 PFU of SARS-CoV-2 via the intranasal route developed severe pathological symptoms including lesions in the nose, throat, and lungs.[289] Younger cats exhibited more severe symptoms than older cats.[289] • Mice genetically modified to express the human ACE2 receptor were inoculated intranasally with 100,000 $TCID_{50}$ (~70,000 PFU) and developed pathological symptoms consistent with COVID-19.[26] **The correlation between the infectious dose and symptom severity is unknown.** • It has been suggested that higher viral loads at hospitalization correspond to more severe clinical outcomes,[200, 300] though pre- and asymptomatic individuals also show viral loads comparable to symptomatic individuals.[19, 366] *Related Coronaviruses* • The infectious dose for severe acute respiratory syndrome coronavirus 1 (SARS-CoV-1) in mice is estimated to be between 67-540 PFU (average 240 PFU, intranasal route).[93, 95] • Genetically modified mice expressing DPP4 exposed intranasally to doses of Middle East respiratory syndrome coronavirus (MERS-CoV) between 100 and 500,000 PFU show signs of infection. Infection with higher doses result in severe syndromes.[12, 80, 187, 358]
What do we need to know?	**Identifying the infectious dose for humans by the various routes through which we become infected is critical to the effective development of computational models to predict risk, develop diagnostics and countermeasures, and effective decontamination strategies. Animal studies are a plausible surrogate.** • Human infectious dose by aerosol route • Human infectious dose by surface contact (fomite) • Human infectious dose by fecal-oral route • Most appropriate animal model for SARS-CoV-2

SARS-CoV-2 (COVID-19)	Transmissibility – How does it spread from one host to another? How easily is it spread?
What do we know?	**SARS-CoV-2 is passed easily between humans, likely through close contact with relatively large droplets and possibly through smaller aerosolized particles.** • Pandemic COVID-19 has caused 4,222,968 infections and 287,809 deaths[153] in at least 187 countries and territories (as of 5/12/2020).[55, 284, 328] • In the US there are 1,354,504 confirmed COVID-19 cases across all 50 US states, with 81,076 deaths (as of 5/12/2020).[153] • Initial high-quality estimates of human transmissibility (R_0) range from 2.2 to 3.1.[213, 249, 270, 336, 357] • SARS-CoV-2 is believed to spread through close contact and droplet transmission,[59] with fomite transmission likely[155] and close-contact aerosol transmission plausible[41, 128] but unconfirmed.[327] • SARS-CoV-2 replicates in the upper respiratory tract, and infectious virus is detectable in throat and lung tissue for at least 8 days.[331] SARS-CoV-2 also infects human gut cell lines,[175] and ocular cells extracted from humans.[144] SARS-CoV-2 genetic material has been found in semen from both clinically symptomatic and recovered cases,[186] however, the infectiousness and the possibility of sexual transmission are unknown. • Contamination of patient rooms with aerosolized SARS-CoV-2 in the human respirable range (0.25-2.5 µm) indicates the potential for airborne transmission.[199] Viral RNA was detected up to 4 meters from ICU patient beds.[130] To date infectious virus has not been isolated from aerosol samples.[281] • SARS-CoV-2 may be spread by conversation and exhalation in the absence of cough, however more work is needed.[9, 185, 281] A preliminary study in China detailing a restaurant-associated outbreak supports aerosol transmission, though confirmation is needed.[192] • Experimentally infected ferrets were able to transmit SARS-CoV-2 to other ferrets by both direct contact (another ferret in same enclosure) as well as through the air (ferrets in an adjacent enclosure, separated by 10 cm).[267] • Evidence suggests that SARS-CoV-2 is not transmitted to infants during birth.[68, 72, 75, 283, 348, 350] **Individuals can transmit SARS-CoV-2 to others before they have symptoms.** • Pre-symptomatic[291, 360] or asymptomatic[24, 142, 209] patients can transmit SARS-CoV-2. At least 12% of all cases are estimated to be due to asymptomatic transmission.[101] It has been estimated that 23%,[197] 44%,[132] or as much as 56%[51] of infections may be caused by pre-symptomatic transmission. Infected patients transmit infections most often before symptoms began and within 5 days of symptom onset.[74] • Individuals may be infectious for 1-3 days prior to symptom onset,[317] and culturable virus has been found in individuals up to 6 days prior to symptom onset.[19] • Severe cases are more likely to transmit disease, and most new infections are within households of infected patients.[207] In China, it is estimated that infected individuals transmit COVID-19 to between 11.2%[34] and 16.3%[190] of household contacts, though even more may lack symptoms.[209] In New York, 38% of household contacts of infected patients became infected, with the proportion increasing with age (20% for contacts <5 years old, 55% for >65 years old).[275] **Undetected cases play a major role in transmission, and most cases are not reported.** • Models suggest up to 86% of early COVID-19 cases in China were undetected, and these infections were the source for 79% of reported cases.[189] • Models estimate that the true number of cases may be approximately 11 times greater than the reported number of cases in the UK,[349] and 5 to 10 times greater than the reported number of cases in the US.[156, 278] Preliminary estimates of the case reporting rate vary widely among countries, from roughly 1 reported case for every 3 actual cases (in Germany), to 1 in 149 (in China).[167]
What do we need to know?	**Identifying the contribution of asymptomatic or pre-symptomatic transmission is important for implementing control measures. Additionally, the relative contributions of different infection sources – fomites, droplets, aerosols, and potentially feces – are unknown.** • Capability of SARS-CoV-2 to be transmitted by contact with fomites (phones, doorknobs, surfaces, clothing, etc.) – see also Experimental Stability • Is sexual transmission possible? • Updated person to person transmission rates (e.g., R_0) as control measures take effect. • Is the R_0 estimate higher in healthcare or long-term care facilities? • When will infections peak in various cities and countries? • Are small-diameter (<5 µm) aerosol exposures capable of infecting humans? • How far do infectious aerosols (small-diameter, <5 µm) travel?

SARS-CoV-2 (COVID-19)	Host Range – How many species does it infect? Can it transfer from species to species?
What do we know?	**SARS-CoV-2 is closely related to other coronaviruses circulating in bats in Southeast Asia. Previous coronaviruses have passed through an intermediate mammal host before infecting humans, but the identity of the SARS-CoV-2 intermediate host is unknown.** • Early genomic analysis indicates similarity to SARS-CoV-1,[364] with a suggested bat origin.[82, 364] • Positive samples from the South China Seafood Market strongly suggests a wildlife source,[61] though it is possible that the virus was circulating in humans before the disease was associated with the seafood market.[29, 83, 342, 351] • Analysis of SARS-CoV-2 genomes suggests that a non-bat intermediate species is responsible for the beginning of the outbreak.[273] The identity of the intermediate host remains unknown.[191, 194-195] • Viruses similar to SARS-CoV-2 were present in pangolin samples collected several years ago.[174] **SARS-CoV-2 uses the same receptor for cell entry as the SARS-CoV-1 coronavirus that circulated in 2002/2003.** • Experiments show that SARS-CoV-2 Spike (S) receptor-binding domain binds the human cell receptor (ACE2) stronger than SARS-CoV-1,[334] potentially explaining its high transmissibility. The same work suggests that differences between SARS-CoV-2 and SARS-CoV-1 Spike proteins may limit the therapeutic ability of SARS antibody treatments.[334] • Modeling of SARS-CoV-2 Spike and ACE2 proteins suggests that SARS-CoV-2 can bind and infect human, bat, civet, monkey and swine cells.[308] Host range predictions based on structural modeling, however, are difficult,[117] and additional animal studies to better define the host range of SARS-CoV-2 are needed. • In vitro experiments suggest a broad host range for SARS-CoV-2, with more than 44 potential animal hosts, based on viral binding to species-specific ACE2 orthologs.[198] The host range is predicted to be limited primarily to mammals. • Genetic and protein analysis of primates suggests that African and Asian primates are likely more susceptible to SARS-CoV-2, while South and Central American primates are likely less susceptible,[225] confirming the SARS-CoV-2 host range is important for identifying animal reservoirs. • Changes in proteolytic cleavage of the Spike protein can also affect cell entry and animal host range, in addition to receptor binding.[226] **To date, ferrets, mink, hamsters, cats, and primates have been shown to be susceptible to SARS-CoV-2 infection. It is unknown whether these animals can transmit infection to humans.** • Animal model studies suggest that Golden Syrian hamsters, primates, and ferrets may be susceptible to infection.[63, 163] In the Netherlands, farmed mink developed breathing and gastrointestinal issues, which was diagnosed as SARS-CoV-2 infection.[2] • Domestic cats are susceptible to infection with SARS-CoV-2 (100,000 PFU via the intranasal route), and can transmit the virus to other cats via droplet or short-distance aerosol.[289] Dogs exposed to SARS-CoV-2 produced anti-SARS-CoV-2 antibodies but exhibited no clinical symptoms.[289] • Wild cats (tigers)[316] can be infected with SARS-CoV-2, although their ability to spread to humans is unknown.[214, 355] Two cases have been confirmed of pet domestic cats infected with SARS-CoV-2.[54] • Ducks, chickens, and pigs remained uninfected after experimental SARS-CoV-2 (30,000 CFU for ducks and chickens, 100,000 PFU for pigs, all via intranasal route).[289] There is currently no evidence that SARS-CoV-2 infects livestock,[149] though modeling suggests sheep, cows, pigs, and goats may be susceptible to infection by SARS-CoV-2.[173]
What do we need to know?	**Several animal models have been developed to recreate human-like illness, though to date they have been infected with high dose exposures. Lower dose studies may better replicate human disease acquisition.** • What is the intermediate host(s)? • What are the mutations in SARS-CoV-2 that allowed human infection and transmission? • What other animals can SARS-CoV-2 infect (e.g., potential wildlife reservoirs)? • Can infected animals transmit to humans (e.g., pet cats to humans)? • Can SARS-CoV-2 circulate in animal reservoir populations, potentially leading to future spillover events?

SARS-CoV-2 (COVID-19)	**Incubation Period – How long after infection do symptoms appear? Are people infectious during this time?**
What do we know?	**The majority of individuals develop symptoms within 14 days of exposure. For most people, it takes at least 2 days to develop symptoms, and on average symptoms develop 5 days after exposure. Incubating individuals can transmit disease for several days before symptom onset. Some individuals never develop symptoms but can still transmit disease.** • The best current estimate of the COVID-19 incubation period is 5.1 days, with 99% of individuals exhibiting symptoms within 14 days of exposure.[180] Fewer than 2.5% of infected individuals show symptoms sooner than 2 days after exposure.[180] • Individuals can test positive for COVID-19 even if they lack clinical symptoms.[24, 62, 129, 296, 360] • Individuals can be infectious while asymptomatic,[59, 276, 296, 360] and asymptomatic and pre-symptomatic individuals have similar amounts of virus in the nose and throat compared to symptomatic patients.[19, 162, 366] • Peak infectiousness may be during the incubation period, one day before symptoms develop.[132] Infectious virus has been cultured in patients up to 6 days before the development of symptoms.[19] • Infectious period is unknown, but possibly up to 10-14 days.[8, 189, 284] • Asymptomatic individuals are estimated to be infectious for a median of 9.5 days.[140] • On average, there are approximately 4[101] to 7.5[188] days between symptom onset in successive cases of a single transmission chain (i.e., the serial interval). Based on data from 339 transmission chains in China, the mean serial interval is 5.29 days.[100] • Children are estimated to shed virus for 15 days on average, with asymptomatic individuals shedding virus for less time (11 days) than symptomatic individuals (17 days).[206] • Most hospitalized individuals are admitted within 8-14 days of symptom onset.[362]
What do we need to know?	**The incubation period is well-characterized. Patients may be infectious, however, for days before symptoms develop.** • What is the average infectious period during which individuals can transmit the disease?

SARS-CoV-2 (COVID-19)	Clinical Presentation – What are the signs and symptoms of an infected person?
What do we know?	**Many COVID-19 cases are asymptomatic. Most symptomatic cases are mild, but severe disease can be found in any age group.[6] Older individuals and those with underlying medical conditions are at higher risk of serious illness and death.** • Approximately 18-31% of patients are asymptomatic throughout the course of their infection.[230, 238, 302] These estimates are based on studies that minimize the likelihood of including pre-symptomatic patients, which can obscure asymptomatic rates.[19] • The majority of symptomatic COVID-19 cases are mild (81%, n=44,000 cases).[296] Initial COVID-19 symptoms include fever (87.9% overall, but only 44-52% present with fever initially),[17, 129] cough (67.7%),[129], fatigue, shortness of breath, headache, and reduced lymphocyte count.[60, 65, 141] Chills, muscle pain, headache, sore throat, and loss of taste or smell[346] are also possible COVID-19 symptoms.[60] The prevalence of GI symptoms varies.[119] Neurological symptoms such as agitation and confusion may present with COVID-19,[133] and may be more common in severe cases.[88] Ocular issues[338] and skin lesions may also be symptoms of COVID-19.[38] • Complications include acute respiratory distress (ARDS, 17-29% of hospitalized patients, leading to death in 4-15% of cases),[71, 141, 310] pneumonia,[245] cardiac injury (20%),[290] secondary infection, kidney damage,[292] arrhythmia, sepsis, and shock.[129, 141, 310, 362] Most deaths are caused by respiratory failure or respiratory failure combined with myocardial (heart) damage.[277] A number of immunological indicators may help differentiate between severe and non-severe cases.[21, 104, 131, 143, 254, 294] • Approximately 15% of hospitalized patients are classified as severe,[129, 296] and approximately 5% of patients are admitted to the ICU.[129, 296] Patient deterioration can be rapid.[127] The survival rate of patients requiring mechanical ventilation varies widely (e.g., 35%,[148] 70%,[20] 75.5%[268]). • Clotting issues may be associated with severely ill COVID-19 patients[165] and those with ARDS.[88] COVID-19 patients should be monitored for possible thrombosis.[183] **The case fatality rate varies substantially by patient age and underlying comorbidities.** • Cardiovascular disease, hypertension, diabetes, and respiratory conditions all increase the CFR.[224, 296, 362] Hypertension and obesity are common in the US[119] and contribute to mortality.[18, 247] • Individuals >60 are at higher risk of death, and the CFR for individuals >85 is between 10 and 27%.[296, 362] In a small study, men exhibited more severe symptoms and died at higher rates than women.[154] The effect of comorbidities on the likelihood of severe symptoms is higher for men.[227] • Deaths due to COVID-19 are underreported. In New York City, up to 5,293 (22%) of period-specific excess deaths are unexplained and could be related to the pandemic.[240] More work is needed. **Additional studies on vulnerable subpopulations are required.** • African Americans are disproportionately represented in hospitalized populations (33% of hospitalized patients, only 18% of the base study population),[119] despite having similar rates of several underlying conditions as other groups.[126] • Pregnant women appear to develop severe symptoms at the same rate as the general population,[70, 159, 352] and current reports suggest no increase in risk of pre-term birth.[347] Most studies of COVID-19 in pregnancy represent women in later stages of pregnancy. **Children are susceptible to COVID-19,[99] though generally show milder[66, 205] or no symptoms.** • Between 21-28% of children may be asymptomatic.[205, 250, 257] A detailed study of 100 children with COVID-19 found that 21% were asymptomatic, 58% developed mild illness, 19% had moderate illness, 1% had severe illness, and 1% developed critical illness.[250] • Severe symptoms in children are possible[196] and more likely in those with complex medical histories.[285] Infant deaths have been recorded.[43, 205] • Early reports indicate the possibility of rare hyperinflammatory syndromes or shock in children (termed Pediatric Multi-System Inflammatory Syndrome[125]), putatively linked to COVID-19 infection.[271] Additional work is needed to confirm a link with COVID-19.
What do we need to know?	**The true case fatality rate is unknown, as the exact number of cases is uncertain. Testing priorities and case definitions vary by location. The proportion of asymptomatic infections is not known.** • How long does it take for infected individuals to recover outside of a healthcare setting? • What proportion of infected individuals are asymptomatic? Does this vary by age, location, or comorbidities? • Studies that test entire populations repeatedly over time and link those tests to the presence or absence of symptoms are necessary.

SARS-CoV-2 (COVID-19)	**Protective Immunity – How long does the immune response provide protection from reinfection?**
What do we know?	**Infected patients show productive immune responses, however more data is needed.** • In a limited study (n=9), hospitalized patients shed high levels of infectious virus for 7 days via the nasal-pharyngeal route, 50% of patients produced antibodies within 7 days, and all patients produced antibodies by 14 days. Antibody production did not correlate with lower viral load.[331] • In a larger study (n=175), most patients developed neutralizing antibodies within 10-15 days after disease onset. Elderly patients had significantly higher neutralizing antibody titers than younger patients.[335] In a separate study, elderly patients also showed higher viral loads than younger patients.[300] • In a study of 285 COVID-19 patients, 100% developed antiviral immunoglobulin-G within 19 days of symptom onset.[201] The neutralizing ability of these antibodies was not tested.[201] In a smaller in vitro study (n=23 patients), levels of antibodies (immunoglobulins M and G) were positively correlated with SARS-CoV-2 neutralizing ability.[300] • In a small series of 26 mild COVID-19 cases, researchers found prolonged persistence of SARS-CoV-2 antibodies and SARS-CoV-2 RNA for up to 50 days. Additionally, one patient cleared SARS-CoV-2 without developing a significant antibody response.[309] • Based on one patient, a productive immune response is generated and sustained for at least 7 days.[297] Previous studies on coronavirus immunity suggest that neutralizing antibodies may wane after several years.[46, 337] More data are needed. • A small subset of COVID-19 patients in China (8%) did not develop a serological response to infection, though the potential for reinfection in these patients is unknown.[335] Similarly, between 16.7% (for IgG) and 51.7% (for IgM) of patients in a separate study did not exhibit any immune response, in terms of production of those two types of antibodies.[295] • In a study of 221 COVID-19 patients, levels of two types of antibodies (IgM and IgG) were not associated with the severity of symptoms.[139] However, in a smaller study, patients with severe disease showed stronger antibody responses than those with non-severe symptoms.[300] • The early recovery phase of COVID-19 patients is characterized by inflammatory immune response,[319] suggesting the potential for adverse reactions after clinical improvement. **Currently, there is no evidence that recovered patients can be reinfected with SARS-CoV-2.** • Experimentally infected macaques were not capable of being reinfected after their primary infection resolved.[25] • According to the WHO, there is no evidence of re-infection with SARS-CoV-2 after recovery.[179] • Patients can test positive via PCR for up to 37 days after symptoms appear,[362] and after recovery and hospital discharge.[176] The ability of these individuals to infect others is unknown. **Similarly, there is no evidence that recovered patients are protected against reinfection with SARS-CoV-2.[324]** • Additional research is required before any conclusions can be drawn about the duration of protective immunity after SARS-CoV-2 infection.[13]
What do we need to know?	**Understanding the duration of protective immunity is limited by small sample sizes. Animal models are plausible surrogates. Additional research to quantify the risk of reinfection after weeks, months, and years is needed.** • How long does the immune response last? • Is there evidence of waning immunity? • Can humans become reinfected? • Are patients who test positive weeks after discharge from hospital capable of transmitting infection? • How does the patient immune response vary by age or disease severity? • How do different components of the immune response contribute to long-term protection?

SARS-CoV-2 (COVID-19)	Clinical Diagnosis – Are there tools to diagnose infected individuals? When during infection are they effective?
What do we know?	**Diagnosis relies on identifying the genetic signature of the virus in patient nose, throat, or sputum samples. These tests are relatively accurate. Confirmed cases are still underreported.** • The US CDC has expanded testing criteria to include symptomatic patients at clinician discretion.[28] • PCR protocols and primers have been widely shared internationally.[52, 85, 188, 288, 323, 329] PCR-based diagnostic assays are unable to differentiate between active and inactive virus. • A combination of pharyngeal (throat) RT-PCR and chest tomography is the most effective diagnostic criteria (correctly diagnoses 91.9% of infections).[265] A single throat swab detects 78.2% of infections, and duplicate tests identify 86.2% of infections.[265] • PCR tests using saliva were better able to detect SARS-CoV-2 RNA than those using nasopharyngeal swabs, and may be useful for self, at-home sampling.[340] Additional validation is needed. • Nasal and pharyngeal swabs may be less effective as diagnostic specimens than sputum and bronchoalveolar lavage fluid,[312] although evidence is mixed.[331] Combination RT-PCR and serology (antibody) testing may increase the ability to diagnose patients with mild symptoms, or identify patients at higher risk of severe disease.[359] • RT-PCR tests can identify asymptomatic cases; SARS-CoV-2 infection was identified in 2/114 individuals cleared by clinical assessment.[136] • The FDA issued an Emergency Use Authorization for an antigen-based diagnostic assay, limited to use in certified laboratories (Clinical Laboratory Improvement Amendments, CLIA).[106] • The FDA released an Emergency Use Authorization enabling laboratories to develop and use tests in-house for patient diagnosis.[110] • Tests from the US CDC are available to states.[52, 59] • Multiple rapid or real-time test kits have been produced by universities and industry, including the Wuhan Institute of Virology,[89] BGI,[33] Cepheid,[307] Abbot,[108] and Mesa Biotech.[35] • The US CDC is developing serological tests to determine what proportion of the population has been exposed to SARS-CoV-2.[158] A rapid antibody test by Cellex is now authorized by the FDA.[135, 320] • Home tests are being developed; however, none are FDA approved, nor are they useable as a diagnostic.[233-234, 248] • Artificial intelligence algorithms were able to improve the ability of radiologists to distinguish COVID-19 pneumonia from non-COVID-19 pneumonia on chest CT scans.[23] **Validated serological (antibody) assays are being developed to help determine who has been exposed to SARS-CoV-2. Serological evidence of exposure does not indicate immunity.** • Researchers found high specificity in a number of enzyme-linked immunosorbent assays (ELISA), though sample sizes for SARS-CoV-2 patients were small.[239] Additional research has shown high variability in the ability of tests (ELISA and lateral flow assays) by different manufacturers to accurately detect positive and negative cases (sensitivity and specificity, respectively).[178, 321] Lateral flow assays may be less reliable than ELISA.[10] • In one German town, serological testing has been used to identify the percent of the population already exposed to SARS-CoV-2 (14%), which can assist in public health response planning.[263] • Preliminary serological studies in Santa Clara and Los Angeles, California, estimated that 2.5-4.1% of the population has already been exposed to SARS-CoV-2 since the first confirmed cases in January,[31, 215] which is between 28 and 85 times greater than official reports. There are issues, however, with non-random study populations,[31] as well as false positive rates of the diagnostic tests themselves.[215] The false positive rate of the diagnostic assay used may account for a substantial portion of the reported infections,[31] particularly if the true proportion of positive patients is low. • In New York, initial serological testing indicates that 13.9% of the population has been exposed to COVID-19, approximately 10 times greater than the number of reported cases.[3] This is in line with other underreporting estimates in the US.[156, 278]
What do we need to know?	**In general, PCR tests appear to be sensitive and specific, though confirmation of symptoms via chest CT is recommended. The sensitivity and specificity of serological testing methods is variable, and additional work needs to be done to determine factors that affect test accuracy.** • How accurate are clinical diagnoses compared to genetic tests? • How effective are different swab specimens as diagnostic samples? • How many serological tests need to be done to obtain an accurate picture of underlying exposure?

SARS-CoV-2 (COVID-19)	Medical Treatments – Are there effective treatments?
What do we know?	**Treatment for COVID-19 is primarily supportive care including ventilation if necessary.[129, 216] Numerous clinical trials are ongoing, but results are preliminary.[30,79] Several drugs show efficacy.** • Two WHO-backed clinical trials (Solidarity and Discovery)[170] include remdesivir, hydroxychloroquine and chloroquine, ritonavir/lopinavir, and ritonavir/lopinavir and interferon-beta.[170] **Remdesivir shows promise for reducing symptom duration in humans.[237]** • Remdesivir can reduce the duration of symptoms in infected individuals, from 15 days to 11 days on average (compared to controls).[237] There is a possibility remdesivir may reduce mortality rates, though the result was not statistically significant.[237] In this trial, individuals with mild symptoms were excluded.[237] Remdesivir has received an Emergency Use Authorization from the FDA.[235] • In a clinical trial of severe COVID-19 patients, the effects of remdesivir were inconclusive due to a limitation in the study sample size.[314] For available patients, remdesivir did not reduce the time to recovery overall, but did show a tendency to reduce symptom duration for patients given the drug early.[314] This trial ended early, reducing its statistical power.[314] **Hydroxychloroquine is associated with elevated risk of cardiac arrhythmias and provides limited to no clinical benefit at this point in time.** • Several studies have found no benefit of hydroxychloroquine (with or without azithromycin) for reducing the need of intensive care,[122] reducing mortality,[211-212] or reducing the duration of symptoms.[69] Studies have documented the development of[212] or potential for cardiac arrhythmias in patients given hydroxychloroquine.[32, 228] One study suggested increased mortality rates in patients taking hydroxychloroquine alone.[211] Individuals taking hydroxychloroquine or colchicine for autoimmune disorders were not protected from COVID-19,[123] though sample sizes were limited. • Initial results purporting benefits of hydroxychloroquine and azithromycin[121] have been called into question by other researchers[146] and the journal's publishing society.[150] One small clinical trial (n=62) suggests that hydroxychloroquine can reduce recovery time compared to control group.[73] Key details are missing from this preprint.[73] **Other pharmaceutical interventions are being investigated.** • A randomized Phase II trial found that a triple combination of interferon beta-1b, lopinavir-ritonavir, and ribavirin administered early in infection reduced symptom severity, viral shedding, and hospital stay time compared to patients taking lopinavir-ritonavir alone.[147] • Limited, preliminary evidence from clinical trials supports the efficacy of favipiravir[67] (which has been approved to treat COVID-19 in China)[1] and intravenous immunoglobulin.[48] Early research found no efficacy from combination ritonavir and lopinavir.[47] • Early results from a randomized clinical trial found that high doses (600 mg, twice per day) of chloroquine diphosphate were associated with toxicity and lethality in patients with severe COVID-19.[36] The trial did not have a large enough sample size to assess chloroquine treatment efficacy.[36] • A small (n=21), observational study found benefits of tocilizumab in severe COVID-19 patients.[343] Potential benefits of immunosuppressants[7] should be weighed against potential risks.[260] • Clinical trials are underway with famotidine[37] (a heartburn reliever) and mesenchymal stromal cells (cells derived from convalescent patient bone marrow)[138] to treat ARDS. • The anticoagulant heparin is being used to mitigate risks of pulmonary embolism, potentially caused by COVID-19-associated clotting problems.[104] An observational cohort study found that systemic anticoagulant use was associated with reduced mortality rates, particularly in patients requiring mechanical ventilation.[246] • Passive antibody therapy (convalescent serum)[49] is being given to patients (FDA Investigational New Drug approval).[109] In a small trial (n=5 patients),[286] convalescent sera administration was followed by clinical improvement.[286] Large efforts are underway to broaden and enhance serum therapies.[287] • Corticosteroids are commonly given to COVID-19 patients[362] at risk of ARDS,[344] but their use is not recommended by the US CDC.[56] A small (n=115 patients) trial in China found no benefit of corticosteroids in terms of admission to the ICU or death,[311] though a small study suggested that methylprednisolone can reduce hospital stay time.[313]
What do we need to know?	**Additional clinical trial results are being released, and data from these trials are needed.** • Are convalescent plasma treatments effective in humans or animals? • Do monoclonal antibodies exhibit any efficacy in human trials? • Are viral replicase inhibitors such as beta-D-N4-hydroxycytidine effective against SARS-CoV-2?[27]

SARS-CoV-2 (COVID-19)	Vaccines – Are there effective vaccines?
What do we know?	**Work is ongoing to develop a SARS-CoV-2 vaccine in human and animal trials. No preliminary results are available.** • Multiple entities are working to produce a SARS-CoV-2 vaccine,[14] including HHS/NIH/NIAID,[134, 184] CEPI, Moderna Therapeutics, Pfizer,[105] Gilead Sciences,[4-5, 236] Sanofi,[39] and Johnson and Johnson.[157] • Vaccine candidates undergoing clinical trial are listed below. *Phase 2 Trials (initial testing for efficacy, continued testing for safety)*: • China's CanSino is the first to complete Phase 1 safety trials of their adenovirus type5 vector based Sars-CoV-2 vaccine, Ad5-nCoV, and has advanced to Phase 2 human trials.[193] *Phase 1 Trials (initial testing for safety)*: • Sinovac Biotech has reported that their inactivated virus vaccine shows protective effects in rhesus macaques, particularly at high doses.[118] The vaccine is currently in phase 1 clinical trials.[81] Moderna has a Phase 1 trial underway based on its mRNA platform, mRNA-1273. • Inovio had their IND approved by the FDA and have started their Phase 1 clinical trials on their DNA vaccine candidate INO-4800.[279] • Shenzhen Geno-Immune Medical Institute is testing its aAPC[222] and lentiviral[221] vaccines in Phase 1 clinical trials. • BioNTech and Pfizer's BNT162 program is in Phase 1/2 clinical trial for four of its mRNA vaccine candidates.[253] • University of Oxford's ChAdOx1 vaccine is in Phase 1 clinical trials. This vaccine is based on a chimpanzee adenovirus expressing SARS-CoV-2 proteins.[252] • The Beijing Institute of Biological Products/Wuhan Institute of Biological Products have initiated a Phase I trial of their inactivated vaccine candidate.[330] • Symvivo Corporation has received approval to begin a Phase I trial with their oral bacTRL-Spike vaccine candidate in Canada.[219] • Novavax is testing a recombinant spike protein nanoparticle vaccine in Phase I trials.[220] • Immunitor LLC is starting Phase I trials of a heat-inactivated vaccine derived from pooled patient plasma.[223] *Co-opting existing vaccines* • Some efforts have begun to enroll healthcare workers in clinical trials to study the efficacy of the BCG (Bacillus Calmette-Guérin) vaccine for reducing symptom severity in COVID-19 patients.[218]
What do we need to know?	**No preliminary vaccine results for humans have been released.** • Safety of candidate vaccines in humans and animals • Efficacy of candidate vaccines in humans and animals • Length of any vaccine-derived immunity • Evidence for vaccine-derived enhancement (immunopotentiation)

SARS-CoV-2 (COVID-19)	Non-pharmaceutical Interventions – Are public health control measures effective at reducing spread?
What do we know?	**Broad-scale control measures such as stay-at-home orders are effective at reducing movement, and modeling shows evidence that they reduce transmission.** • Social distancing and other policies are estimated to have reduced COVID-19 spread by 44% in Hong Kong[87] and reduced spread throughout China[166, 169, 172, 202] and Italy.[120] Restrictive lockdowns in China are estimated to have reduced disease transmission within only a few days,[365] in part, through reductions in an individual's average number of contacts.[353] • Modeling demonstrates that multifaceted restrictions and quarantines in China reduced the R_0 of SARS-CoV-2 from greater than 3 to less than 1 between January 23 and February 5.[244] Additionally, movement restrictions and other control measures helped limit the amount of time where community transmission was possible (i.e., $R_0 > 1$).[354] • Models indicate that a combination of school closures, work restrictions, and other measures are required to effectively limit transmission.[113] School closures alone appear insufficient.[151, 172] • Non-pharmaceutical interventions in China did not reduce transmission equally across all groups; transmission rates in younger individuals, particularly infants, as well as hospital workers continued to increase even while overall transmission rates declined.[244] • Anonymized cell phone location data indicate major reductions in mobility due to social distancing.[115] Mobility in major US cities declined after each public health intervention implemented.[177] Survey data from the UK suggest large reductions in individuals' physical contacts after public health restrictions were put into effect.[152] • Contact tracing to identify infected individuals reduces the amount of time infectious individuals can transmit disease in a population and increases the time between successive cases (the serial interval).[34] • In a prospective study, researchers found that a larger number of public health interventions in place was strongly associated with lower COVID-19 growth rates in the next week.[160] **The effect of relaxing control measures is unknown, and research is needed to help plan for easing of restrictions.** • Modeling indicates that COVID-19 is likely to become endemic in the US population, with regular, periodic outbreaks, and that additional social or physical distancing measures may be required for several years to keep cases below critical care capacity in absence of a vaccine or effective therapeutic.[164] Results depend critically on the duration of immunity after exposure.[164] • Modeling suggests that premature lifting of social distancing measures will substantially increase the number of local COVID-19 cases in Wuhan, China.[255] Similarly, forecasts in the US estimate a resumption of exponential case growth if social distancing measures are relaxed.[90] • In the UK, modelers are assessing the efficacy of rolling interventions, whereby social distancing measures are put into place every few weeks to keep healthcare demand below a critical point.[349] • A modeling study using Chinese data suggests that carefully balancing control measures to maintain R_0 below 1 would be more efficient than allowing R_0 to increase above 1 at any point.[181] • Robust contact tracing and case finding may be needed to control COVID-19 in the US, but would require additional staff and resources to conduct effectively.[315] • A model suggests that reducing public contacts is more important for controlling spread than reducing private (e.g., in-home) contacts.[217] • The WHO has released guidelines on public health strategy, focusing on controlling transmission, ensuring public health capacity is robust, and engaging local communities.[322] • Johns Hopkins released a report outlining how to re-open certain categories of activities (e.g., schools, restaurants, events) while reducing COVID-19 risk; the report also ranks certain activities by their contact intensity, number of contacts, and the potential to modify them to reduce risk.[272] • SARS-CoV-2 levels in wastewater may track with prevalence in the population,[339] and could be used to monitor viral elimination in an area.
What do we need to know?	**As different US states have implemented differing control measures at various times, a comprehensive analysis of social distancing efficacy has not yet been conducted.** • How many cases in the US have been averted due to social distancing restrictions? • What are plausible options for relaxing social distancing and other intervention measures without resulting in a resurgence of COVID-19 cases? • How is COVID-19 incidence changing in states that have begun easing movement and activity restrictions?

SARS-CoV-2 (COVID-19)	Environmental Stability – How long does the agent live in the environment?
What do we know?	**SARS-CoV-2 can persist on surfaces for at least 3 days and on the surface of a surgical mask for up to 7 days depending on conditions. If aerosolized intentionally, SARS-CoV-2 is stable for at least several hours. The seasonality of COVID-19 transmission is unknown.** *SARS-CoV-2 Data* • SARS-CoV-2 can persist on plastic and metal surfaces between 3 days (21-23°C, 40% RH)[304] and 7 days (22°C, 65% RH). Infectious virus can be recovered from a surgical mask after 7 days (22°C, 65% RH).[77] • SARS-CoV-2 has an aerosol half-life of 2.7 hours (particles <5 µm, tested at 21-23°C and 65% RH).[304] • SARS-CoV-2 is susceptible to heat treatment (70°C) but can persist for at least two weeks at refrigerated temperatures (4°C).[77, 262] • SARS-CoV-2 genetic material (RNA) was detected in symptomatic and asymptomatic cruise ship passenger rooms up to 17 days after cabins were vacated. The infectiousness of this material is not known.[231] • In a preliminary study, SARS-CoV-2 stability was enhanced when present with bovine serum albumin, which is commonly used to represent sources of protein found in human sputum.[251] • No strong evidence exists showing a reduction in transmission with seasonal increase in temperature and humidity.[208] Modeling suggests that even accounting for potential reductions in transmission due to weather and behavioral changes, public health interventions will still need to be in effect to limit COVID-19 transmission.[229] *Surrogate Coronavirus data*: • Studies suggest that other coronaviruses can survive on non-porous surfaces up to 9-10 days (MHV, SARS-CoV),[50, 64] and porous surfaces for up to 3-5 days (SARS-CoV)[103] in air conditioned environments (20-25°C, 40-50% RH). • Coronavirus survival tends to be higher at lower temperatures and lower relative humidity (RH),[50, 64, 259, 305] though infectious virus can persist on surfaces for several days in typical office or hospital conditions.[305] • SARS can persist with trace infectivity for up to 28 days at refrigerated temperatures (4°C) on surfaces.[50] • One hour after aerosolization approximately 63% of airborne MERS virus remained viable in a simulated office environment (25°C, 75% RH).[256] • Porous hospital materials, including paper and cotton cloth, maintain infectious SARS-CoV for a shorter time than non-porous material.[171]
What do we need to know?	**Additional testing on SARS-CoV-2, as opposed to surrogate viruses, is needed to support initial estimates of stability.** • Stability of SARS-CoV-2 in aerosol, droplets, and other matrices (mucus/sputum, feces) • Particle size distribution (e.g., droplet, large droplet, and true aerosol distribution) • Duration of SARS-CoV-2 infectivity via fomites and surfaces (contact hazard) • Stability of SARS-CoV-2 on PPE (e.g., Tyvek, nitrile, etc.) • Evidence for seasonality in transmission, or other environmental impacts (UV, temperature, humidity)

SARS-CoV-2 (COVID-19)	Decontamination – What are effective methods to kill the agent in the environment?
What do we know?	**Soap and water, as well as common alcohol and chlorine-based cleaners, hand sanitizers, and disinfectants are effective at inactivating SARS-CoV-2 on hands and surfaces.** *SARS-CoV-2* • Alcohol-based hand rubs are effective at inactivating SARS-CoV-2.[168] • Chlorine bleach (1%, 2%), 70% ethanol and 0.05% chlorhexidine are effective against live virus in lab tests.[76] • Twice-daily cleaning with sodium dichloroisocyanurate decontaminated surfaces in COVID-19 patient hospital rooms.[241] • EPA has released a list of SARS-CoV-2 disinfectants, but solutions were not tested on live virus.[11] *Other Coronaviruses* • Chlorine-based[326] and ethanol-based[84] solutions are recommended. • Heat treatment (56°C) is sufficient to kill coronaviruses,[259, 361] though effectiveness depends partly on protein in the sample.[259] • 70% ethanol, 50% isopropanol, sodium hypochlorite (0.02% bleach), and UV radiation can inactivate several coronaviruses (MHV and CCV).[280] • Ethanol-based biocides effectively disinfect coronaviruses dried on surfaces, including ethanol containing gels similar to hand sanitizer.[145, 332] • Surface spray disinfectants such as Mikrobac, Dismozon, and Korsolex are effective at reducing infectivity of the closely related SARS-CoV-1 after 30 minutes of contact.[258] • Coronaviruses may be resistant to heat inactivation for up to 7 days when stabilized in stool.[298-299] • Coronaviruses are more stable in matrixes such as respiratory sputum.[102] **Methods for decontaminating N95 masks have been approved by the FDA under an Emergency Use Authorization (EUA).** • Researchers have identified four methods capable of decontaminating N95 respirators while maintaining physical integrity (fit factor): UV radiation, heating to 70°C, and vaporized hydrogen peroxide (VHP).[114] Ethanol (70%) was associated with loss of physical integrity.[114] • Hydrogen peroxide vapor (VHP) can repeatedly decontaminate N95 respirators.[269] Devices capable of decontaminating 80,000 masks per day have been granted Emergency Use Authorization from the FDA.[106] • The FDA has issued an Emergency Use Authorization for a system capable of decontaminating 10 N95 masks at a time using devices already present in many US hospitals.[40]
What do we need to know?	**Additional decontamination studies, particularly with regard to PPE and other items in short supply, are needed.** • What is the minimal contact time for disinfectants? • Does contamination with human fluids/waste alter disinfectant efficacy profiles? • How effective is air filtration at reducing transmission in healthcare, airplanes, and public spaces? • Are landfills and wastewater treatment plants effective at inactivating SARS-CoV-2? • Is heat or UV decontamination effective to clean N95 masks, respirators and other types of PPE for multi-use?

SARS-CoV-2 (COVID-19)	PPE – What PPE is effective, and who should be using it?
What do we know?	**The effectiveness of PPE for SARS-CoV-2 is currently unknown, and data from other related coronaviruses are used for guidance. Healthcare workers are at high risk of acquiring COVID-19, even with recommended PPE.** • Healthcare worker illnesses[296] demonstrates human-to-human transmission despite isolation, PPE, and infection control.[282] • Risk of transmission to healthcare workers appears high, with up to 20% of healthcare workers in Lombardy, Italy becoming infected.[264] • Over 50% of US healthcare workers infected with COVID-19 report work in a healthcare setting as their single source of exposure.[44] • "Healthcare personnel entering the room [of SARS-CoV-2 patients] should use standard precautions, contact precautions, airborne precautions, and use eye protection (e.g., goggles or a face shield)."[57] • WHO indicates healthcare workers should wear clean long-sleeve gowns as well as gloves.[325] • Using a fluorescent simulant, researchers found contamination on exposed skin on healthcare workers while performing intubations.[112] Clothing and PPE that covers all skin may reduce exposure to pathogens.[112, 318] • Respirators (NIOSH-certified N95, EUFFP2 or equivalent) are recommended for those dealing with possible aerosols.[326] Additional protection, such as a Powered Air Purifying Respirator (PAPR) with a full hood, should be considered for high-risk procedures (i.e., intubation, ventilation).[42] • Particular attention should be paid to the potential for transmission via exhaled air during supportive respiratory procedures.[128] • There is evidence both for[199] and against[241] the detection of SARS-CoV-2 RNA via air sampling in patient rooms and other hospital areas. • Research at Johns Hopkins Center for Health Security has provided initial estimates of PPE needs in the US: 7.8 billion gloves, 668 million gowns, 360 million surgical masks, and 136 million N95 or similar respirators.[301] • KN95 respirators are, under certain conditions, approved for use under FDA Emergency Use Authorization.[107] On May 7, the FDA rescinded a number of KN95 models that no longer meet the EUA criteria and are no longer authorized.[111] **Masks may be effective at slowing transmission.** • Surgical face masks, respirators and homemade face masks may prevent transmission of coronaviruses from infectious individuals (with or without symptoms) to other individuals.[92, 182, 303] Surgical masks were associated with a significant reduction in the amount of seasonal coronavirus (not SARS-CoV-2) expressed as aerosol particles (<5 µm) compared to not wearing a mask.[182] Other preliminary work has failed to document protective efficacy of surgical or cotton masks,[22] and more SARS-CoV-2 specific research is needed. • On 4/3/2020, the US CDC recommended wearing cloth face masks in public where social distancing measures are difficult to maintain.[58] • The efficacy of homemade PPE, made with T-shirts, bandanas, or similar materials, is less than standard PPE, but may offer some protection if no other options are available.[78, 91, 266]
What do we need to know?	**Most PPE recommendations have not been made on SARS-CoV-2 data, and comparative efficacy of different PPE for different tasks (e.g., intubation) is unknown. Identification of efficacious PPE for healthcare workers is critical due to their high rates of infection.** • What is the importance of aerosol transmission (particles <5µm)? What is the effective distance of spread via droplet or aerosol? • How effective are barriers such as N95 respirators or surgical masks for SARS-CoV-2? • What is the appropriate PPE for first responders? Airport screeners? • What are proper procedures for reducing spread and transmission rates in medical facilities? • How effective are homemade masks at reducing SARS-CoV-2 transmission?

SARS-CoV-2 (COVID-19)	Forensics – Natural vs intentional use? Tests to be used for attribution.
What do we know?	**All current evidence supports the natural emergence of SARS-CoV-2 via a bat and possible intermediate mammal species.** • Genomic analysis places SARS-CoV-2 into the beta-coronavirus clade, with close relationship to bat coronaviruses. The SARS-CoV-2 virus is distinct from SARS-CoV-1 and MERS viruses.[98] • Genomic analysis suggests that SARS-CoV-2 is a natural variant and is unlikely to be human-derived or otherwise created by "recombination" with other circulating strains of coronavirus.[15, 364] • Genomic data support at least two plausible origins of SARS-CoV-2: "(i) natural selection in a non-human animal host prior to zoonotic transfer, and (ii) natural selection in humans following zoonotic transfer."[15] Both scenarios are consistent with the observed genetic changes found in all known SARS-CoV-2 isolates. • Some SARS-CoV-2 genomic evidence indicates a close relationship with pangolin coronaviruses,[333] and data suggest that pangolins may be a natural host for beta-coronaviruses.[194-195] Genomic evidence suggests a plausible recombination event between a circulating coronavirus in pangolins and bats could be the source of SARS-CoV-2.[341] Emerging studies are showing that bats are not the only reservoir of SARS-like coronaviruses.[356] Additional research is needed. • A novel bat coronavirus (RmYN02) has been identified in China with an insertion in the viral furin cleavage site. While distinct from the insertion in SARS-CoV-2, this evidence shows that such insertions can occur naturally.[363] • Additionally, "[…] SARS-CoV-2 is not derived from any previously used virus backbone," reducing the likelihood of laboratory origination,[15] and "[…] genomic evidence does not support the idea that SARS-CoV-2 is a laboratory construct, [though] it is currently impossible to prove or disprove the other theories of its origin."[15] • Work with other coronaviruses has indicated that heparan sulfate dependence can be an indicator of prior cell passage, due to a mutation in the previous furin enzyme recognition motif.[94]
What do we need to know?	**Identifying the intermediate species between bats and humans would aid in reducing potential spillover from a natural source. Wide sampling of bats, other wild animals, and humans is needed to address the origin of SARS-CoV-2.** • What tests for attribution exist for coronavirus emergence? • What is the identity of the intermediate species? • Are there closely related circulating coronaviruses in bats or other animals with the novel PRRA cleavage site found in SARS-CoV-2?

SARS-CoV-2 (COVID-19)	Genomics – How does the disease agent compare to previous strains?
What do we know?	**Current evidence suggests that SARS-CoV-2 accumulates substitutions and mutations at a similar rate as other coronaviruses. Mutations and deletions in specific portions of the SARS-CoV-2 genome have not been linked to any changes in transmission or disease severity, though modeling work is attempting to identify possible changes.** • There have been no documented cases of SARS-CoV-2 prior to December 2019. Preliminary genomic analyses, however, suggest that the first human cases of SARS-CoV-2 emerged between 10/19/2019 – 12/17/2019.[16, 29, 261] • Analysis of more than 7,000 SARS-CoV-2 genome samples provides an estimated mutation rate of $6x10^{-4}$ nucleotides per genome per year.[306] The same analysis estimates the emergence of SARS-CoV-2 in humans between October and December 2019.[306] This aligns with the first known human cases in China in early December 2019 and in Europe in late December 2019.[97] • There is currently no evidence of distinct SARS-CoV-2 phenotypes at this time.[210, 306] • Despite evidence of genomic areas under positive selection, there is no known association between particular mutations and changes in transmission or virulence.[45] • Pangolin coronaviruses are closely related to both SARS-CoV-2 and closely related bat coronaviruses. Phylogenetic analysis suggests that SARS-CoV-2 is of bat origin, but is closely related to pangolin coronavirus.[194-195] • The SARS-CoV-2 Spike protein, which mediates entry into host cells and is the major determinant of host range, is very similar to the SARS-CoV-1 Spike protein.[203] The rest of the genome is more closely related to two separate bat [203] and pangolin[195] coronaviruses. • An analysis of SARS-CoV-2 sequences from Singapore has identified a large nucleotide (382 bp) deletion in ORF-8.[293] In Arizona, researchers identified an 81-base pair deletion (removing 27 amino acids) in the ORF-7a protein, indicating that mutations can be detected by routine sentinel surveillance. The function of these deletions are unknown at this time.[137] • A recent report of virus mutations within patients needs more research.[161] Additional analysis of data suggests the results may be due to experimental methods.[124, 345] • Structural modeling suggests that observed changes in the genetic sequence of the SARS-CoV-2 Spike protein may enhance binding of the virus to human ACE2 receptors.[242] More specifically, changes to two residues (Q493 and N501) are linked with improving the stability of the virus-receptor binding complex.[242] Additionally, structural modeling identified several existing mutations that may enhance the stability of the receptor binding domain, potentially increasing binding efficacy.[243] Infectivity assays are needed to validate the genotypic changes and possible phenotypic results identified in these studies. • A key difference between SARS-CoV-2 and other beta-coronaviruses is the presence of a polybasic furin cleavage site in the Spike protein (insertion of a PRRA amino acid sequence between S1 and S2).[86] • The US CDC is launching a national genomics consortium to assess SARS-CoV-2 genomic changes over time.[53] • Preliminary phylogenetic analysis indicates at least three major SARS-CoV-2 clades that represent broad regions, though this is likely to change as additional genomes are sampled.[116] To date, there are no known differences in disease severity due to infection with different strains of SARS-CoV-2.
What do we need to know?	**Research linking genetic changes to differences in phenotype (e.g., transmissibility, virulence, progression in patients) is needed.** • Are there similar genomic differences in the progression of coronavirus strains from bat to intermediate species to human? • Are there different strains or clades of circulating virus? If so, do they differ in virulence?

Table 1. Definitions of commonly-used acronyms

Acronym/Term	Definition	Description
ACE2	Angiotensin-converting enzyme 2	Acts as a receptor for SARS-CoV and SARS-CoV-2, allowing entry into human cells
Airborne transmission	Aerosolization of infectious particles	Aerosolized particles can spread for long distances (e.g., between hospital rooms via HVAC systems). Particles generally <5 µm.
ARDS	Acute respiratory distress syndrome	Leakage of fluid into the lungs which inhibits respiration and leads to death
Attack rate	Proportion of "at-risk" individuals who develop infection	Defined in terms of "at-risk" population such as schools or households, defines the proportion of individuals in those populations who become infected after contact with an infectious individual
CCV	Canine coronavirus	Canine coronavirus
CFR	Case Fatality Rate	Number of deaths divided by confirmed patients
CoV	Coronavirus	Virus typified by crown-like structures when viewed under electron microscope
COVID-19	Coronavirus disease 19	Official name for the disease caused by the SARS-CoV-2 virus.
Droplet transmission	Sneezing, coughing	Transmission via droplets requires relatively close contact (e.g., within 6 feet)
ELISA	Enzyme-linked immunosorbent assay	Method for serological testing of antibodies
Fomite	Inanimate vector of disease	Surfaces such as hospital beds, doorknobs, healthcare worker gowns, faucets, etc.
HCW	Healthcare worker	Doctors, nurses, technicians dealing with patients or samples
Incubation period	Time between infection and symptom onset	Time between infection and onset of symptoms typically establishes guidelines for isolating patients before transmission is possible
Infectious period	Length of time an individual can transmit infection to others	Reducing the infectious period is a key method of reducing overall transmission; hospitalization, isolation, and quarantine are all effective methods
Intranasal	Agent deposited into external nares of subject	Simulates inhalation exposure by depositing liquid solution of pathogen/virus into the nose of a test animal, where it is then taken up by the respiratory system.
MERS	Middle-East Respiratory Syndrome	Coronavirus with over 2,000 cases in regional outbreak since 2012
MHV	Mouse hepatitis virus	Coronavirus surrogate
Nosocomial	Healthcare- or hospital-associated infections	Characteristic of SARS and MERS outbreaks, lead to refinement of infection control procedures
PCR	Polymerase chain reaction	PCR (or real-time [RT] or quantitative [Q] PCR) is a method of increasing the amount of genetic material in a sample, which is then used for diagnostic testing to confirm the presence of SARS-CoV-2
PFU	Plaque forming unit	Measurement of the number of infectious virus particles as determined by plaque forming assay. A measurement of sample infectivity.
PPE	Personal protective equipment	Gowns, masks, gloves, and any other measures used to prevent spread between individuals
R_0	Basic reproduction number	A measure of transmissibility. Specifically, the average number of new infections caused by a typical infectious individual in a wholly susceptible population.

Acronym/Term	Definition	Description
SARS	Severe Acute Respiratory Syndrome	Coronavirus with over 8,000 cases in global 2002-2003 outbreak
SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2	Official name for the virus previously known as 2019-nCoV.
Serial interval	Length of time between symptom onset of successive cases in a transmission chain	The serial interval can be used to estimate R_0, and is useful for estimating the rate of outbreak spread
Superspreading	One individual responsible for an abnormally large number of secondary infections	Superspreading can be caused by high variance in the distribution of secondary cases caused by a single individual; most individuals infect very few people, while some infect a large number, even with the same average number of secondary infections
$TCID_{50}$	50% Tissue Culture Infectious Dose	The number of infectious units which will infect 50% of tissue culture monolayers. A measurement of sample infectivity.
Transgenic	Genetically modified	In this case, animal models modified to be more susceptible to MERS and/or SARS by adding proteins or receptors necessary for infection

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DHS SCIENCE AND TECHNOLOGY

Master Question List for COVID-19 (caused by SARS-CoV-2)

Weekly Report

18 March 2020

For comments or questions related to the contents of this document, please contact the DHS S&T Hazard Awareness & Characterization Technology Center at (b)(6)

Homeland Security

Science and Technology

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FOREWORD

The Department of Homeland Security (DHS) is paying close attention to the evolving Coronavirus Infectious Disease (COVID-19) situation in order to protect our nation. DHS is working very closely with the Centers for Disease Control and Prevention (CDC), other federal agencies, and public health officials to implement public health control measures related to travelers and materials crossing our borders from the affected regions.

Based on the response to a similar product generated in 2014 in response to the Ebolavirus outbreak in West Africa, the DHS Science and Technology Directorate (DHS S&T) developed the following "master question list" that quickly summarizes what is known, what additional information is needed, and who may be working to address such fundamental questions as, "What is the infectious dose?" and "How long does the virus persist in the environment?" The Master Question List (MQL) is intended to quickly present the current state of available information to government decision makers in the operational response to COVID-19 and allow structured and scientifically guided discussions across the federal government without burdening them with the need to review scientific reports, and to prevent duplication of efforts by highlighting and coordinating research.

The information contained in the following table has been assembled and evaluated by experts from publicly available sources to include reports and articles found in scientific and technical journals, selected sources on the internet, and various media reports. It is intended to serve as a "quick reference" tool and should not be regarded as comprehensive source of information, nor as necessarily representing the official policies, either expressed or implied, of the DHS or the U.S. Government. DHS does not endorse any products or commercial services mentioned in this document. All sources of the information provided are cited so that individual users of this document may independently evaluate the source of that information and its suitability for any particular use. This document is a "living document" that will be updated as needed when new information becomes available.

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SARS-CoV-2 (COVID-19)	Infectious dose – how much agent will make a normal individual ill?	Transmissibility – How does it spread from one host to another? How easily is it spread?	Host range – how many species does it infect? Can it transfer from species to species?	Incubation period – how long after infection do symptoms appear? Are people infectious during this time?
What do we know?	• The human infectious dose of SARS-CoV-2, which causes coronavirus disease 19 (COVID-19) is currently unknown via all exposure routes. Animal data are used as surrogates. • Rhesus macaques are infected with SARS-CoV-2 via the ocular conjunctival and intratracheal route at a dose of 700,000 PFU (10^6 $TCID_{50}$).[51] • A total dose of 700,000 plaque-forming units (PFU) of SARS-CoV-2 infected cynomolgus macaques via a combination intranasal and intratracheal exposure (10^6 $TCID_{50}$ total dose).[109] Macaques did not exhibit clinical symptoms, but shed virus through the nose and throat.[109] • Nonhuman primate infection may not represent human infection. • Initial experiments suggest that SARS-CoV-2 can infect genetically modified mice containing the human ACE2 cell entry receptor. Infection via the intranasal route (dose: 10^5 $TCID_{50}$, approximately 70,000 PFU) causes light infection, however no virus was isolated from infected animals, and polymerase chain reaction (PCR) primers used in the study do not align well with SARS-CoV-2, casting doubt on this study.[14] • The infectious dose for SARS in mice is estimated to be between 67-540 PFU (average 240 PFU, intranasal route).[49-50] • Genetically modified mice exposed intranasally to doses of MERS virus between 100 and 500,000 PFU show signs of infection. Infection with higher doses result in severe syndromes.[7, 41, 81, 150]	• Pandemic COVID-19 has caused 214,894 infections and 8,732 deaths[72] in at least 173 countries and territories (as of 3/18/2020).[27, 114, 135] • There are 7,769 SARS-CoV-2 cases across 50 US states, with 118 deaths. (as of 3/18/2020)[72]; there is sustained community transmission of COVID-19 in the US.[17] • High-quality estimates of human transmissibility (R_0) range from 2.2 to 3.1[93, 98, 106, 142, 149] Early estimates of the attack rate in China range from 3%-10%, mainly in households.[137] • SARS-CoV-2 is believed to spread through close contact and droplet transmission,[31] with fomite transmission[73], i.e., germs left on surfaces, and close-contact aerosol transmission also plausible.[22] • SARS-CoV-2 replicates in the upper respiratory tract (e.g., throat), and infectious virus is detectable in throat and lung tissue for at least 8 days.[138] • Pre-symptomatic[151] or asymptomatic[12] patients can transmit SARS-CoV-2; between 12%[54] and 23%[90] of infections may be caused by asymptomatic or pre-symptomatic transmission. • SARS-CoV-2 is present in infected patient saliva,[124] lower respiratory sputum,[131] and feces.[86] • Social distancing and behavioral changes are estimated to have reduced COVID-19 spread by 44% in Hong Kong,[47] and a combination of non-pharmaceutical interventions (e.g., school closures, isolation) are likely required to limit transmission.[59] • Up to 86% of early COVID-19 cases in China were undiagnosed, and these infections were the source for 79% of documented cases.[84]	• Early genomic analysis indicates similarity to SARS,[154] with a suggested bat origin.[5,42, 154] • Analysis of SARS-CoV-2 genomes suggests that a non-bat intermediate species is responsible for the beginning of the outbreak.[108] The identity of the intermediate host remains unknown.[85, 87-88] • Positive samples from the South China Seafood Market strongly suggests a wildlife source,[33] though it is possible that the virus was circulating in humans before the disease was associated with the seafood market.[18, 43, 144, 148] • Experiments suggest that SARS-CoV-2 Spike (S) receptor-binding domain binds the human cell receptor (ACE2) stronger than SARS,[141] potentially explaining its high transmissibility; the same work suggests that differences between SARS-CoV-2 and SARS-CoV Spike proteins may limit the therapeutic ability of SARS antibody treatments.[141] • Modeling between SARS-CoV-2 Spike and ACE2 proteins suggests that SARS-CoV-2 can bind and infect human, bat, civet, monkey and swine cells.[129] • There is currently no experimental evidence that SARS-CoV-2 infects domestic animals or livestock, though it is expected that some animal species could be infected.	• The best current estimate of the COVID-19 incubation period is 5.1 days, with 99% of individuals exhibiting symptoms within 14 days of exposure.[79] Fewer than 2.5% of infected individuals show symptoms sooner than 2 days after exposure.[79] • The reported range of incubation periods is wide, with high-end estimates of 24,[60] 11.3,[11] and 18 days.[83] • Individuals can test positive for COVID-19 despite lacking clinical symptoms.[12, 35, 60, 120, 151] • Individuals can be infectious while asymptomatic,[31, 110, 120, 151] and asymptomatic individuals can have similar amounts of virus in their nose and throat as symptomatic individuals.[155] • Infectious period is unknown, but possibly up to 10-14 days [5, 84, 114] • On average, there are approximately 4[54] to 7.5[83] days between symptom onset in successive cases of a single transmission chain. • Most individuals are admitted to the hospital within 8-14 days of symptom onset.[153] • Patients are positive for COVID-19 via PCR for 8-37 days after symptom onset.[153] • Individuals may test positive via PCR for 5-13 days after symptom recovery and hospital discharge.[77] The ability of these individuals to infect others is unknown. • According to the WHO, there is no evidence of re-infection with SARS-CoV-2 after recovery.[78] • Experimentally infected macaques were not capable of being reinfected after their primary infection resolved.[13]

SARS-CoV-2 (COVID-19)	**Infectious dose – how much agent will make a normal individual ill?**	**Transmissibility – How does it spread from one host to another? How easily is it spread?**	**Host range – how many species does it infect? Can it transfer from species to species?**	**Incubation period – how long after infection do symptoms appear? Are people infectious during this time?**
What do we need to know?	• Human infectious dose by aerosol route • Human infectious dose by surface contact (fomite) • Human infectious dose by fecal-oral route	• Capability of SARS-CoV-2 to be transmitted by contact with fomites (doorknobs, surfaces, clothing, etc.) – see also Experimental Stability • Superspreading capacity needs to be refined • Updated person to person transmission rates (e.g., R_0) as control measures take effect • What is the underreporting rate?[71] • Can individuals become re-infected with SARS-CoV-2? • What is the difference in transmissibility among countries? • Is the R0 estimate higher in healthcare or long-term care facilities?	• What is the intermediate host(s)? • What are the mutations in SARS-CoV-2 that allowed human infection and transmission? • What animals can SARS-CoV-2 infect (e.g., pet dogs, potential wildlife reservoirs)?	• What is the average infectious period during which individuals can transmit the disease? • Are individuals infectious after hospital discharge and clinical recovery, or are positive PCR tests only detecting non-infectious virus? • Can individuals become re-infected after recovery? If so, how long after?
Who is doing experiments/has capabilities in this area?	*Capable of performing work* - DHS National Biodefense Analysis and Countermeasures Center (NBACC)	*Performing work:* - Christian Althaus (Bern) - Neil Ferguson (MRC) - Gabriel Leung, Joseph Wu (University of Hong Kong) - Sara Del Valle (Los Alamos) - Maimuna Majumder (Boston Children's Hospital) - Trevor Bedford (Fred Hutchinson Cancer Center) - Sang Woo Park (Princeton)	*Capable of performing work:* - Vincent Munster (Rocky Mountain National Laboratory) - Matthew Frieman (University of Maryland Baltimore) - Ralph Baric (University of North Carolina) - Stanley Perlman (University of Iowa) - Susan Baker (Loyola University Chicago) - Mark Denison (Vanderbilt University) - Vineet Menachery (University of Texas Medical Branch) - Jason McLellan, Daniel Wrapp, Nianshuang Wang (University of Texas) - David O'Conner (U. Wisconsin, Madison)	*Performing work:* - Chaolin Huang (Jin Yin-tan Hospital, Wuhan, China) - The Novel Coronavirus Pneumonia Emergency Response Epidemiology Team

SARS-CoV-2 (COVID-19)	Clinical presentation – what are the signs and symptoms of an infected person?	Clinical diagnosis – are there tools to diagnose infected individuals? When during infection are they effective?	Medical treatment – are there effective treatments? Vaccines?	Environmental stability – how long does the agent live in the environment?
What do we know?	• The majority of COVID-19 cases are mild (81%, N = 44,000 cases)[120] • Initial COVID-19 symptoms include fever (87.9% overall, but only 43.8% present with fever initially[60]), cough (67.7%[60]), fatigue, shortness of breath, headache, reduction in lymphocyte count.[32, 38, 68] Headache[37] and diarrhea are uncommon[68, 82] • Complications include acute respiratory distress (ARDS observed in 17-29% of hospitalized patients,[40, 67] which leads to death in 4-15% of cases[40, 68, 130]), pneumonia,[96] cardiac injury, secondary infection, kidney failure, arrhythmia, sepsis, and shock.[60,68,130, 153] • Approximately 15% of hospitalized patients were classified as severe,[60, 120] and approximately 5% of patients were admitted to the ICU.[60, 120] • Most deaths are caused by respiratory failure or respiratory failure combined with myocardial (heart) damage.[111] • The case fatality rate (CFR) depends on comorbidities; cardiovascular disease, hypertension, diabetes, and respiratory conditions all increase the CFR.[120, 153] • The CFR increases with age; individuals older than 60 are at higher risk of death,[120, 153] and >60% of confirmed fatalities have been male.[120] • Children of all ages are susceptible to COVID-19,[53] though generally present with milder symptoms.[39] Severe symptoms in children, however, are possible.[89] • In the US, 34% of hospitalizations have been individuals less than 44 years old.[4] Based on one patient, a productive immune response is generated and sustained for at least 7 days.[121]	• PCR protocols and primers have been widely shared among international researchers[26, 45, 83, 116, 132, 136] though PCR-based diagnostic assays do not differentiate between active and inactive virus. • A combination of pharyngeal (throat) RT-PCR and chest tomography are the most effective diagnostic criteria (correctly diagnosing 91.9% of infections).[104] Single throat swabs alone detect 78.2% of true infections, while duplicate tests identify 86.2% of infections.[104] • Nasal and pharyngeal swabs may be less effective as diagnostic specimens than sputum and bronchoalveolar lavage fluid.[131] • RT-PCR tests are able to identify asymptomatic cases; SARS-CoV-2 infection was identified in 2/114 individuals previously cleared by clinical assessment.[66] • The FDA released an Emergency Use Authorization enabling laboratories to develop and use tests in-house for patient diagnosis.[58] • Updated tests from the US CDC are available to states.[26, 31] • US CDC has expanded patient testing criteria to include symptomatic patients at clinician discretion.[16] • Several rapid or real-time test kits have been produced by universities and industry, including the Wuhan Institute of Virology,[48] BGI,[19] and Cepheid.[128] • The US CDC is developing serological tests to determine what proportion of the population has been exposed to SARS-CoV-2.[74] • Machine learning tools are being developed to predict severe and fatal COVID-19 cases based on CT scans.[117]	• Treatment for COVID-19 is primarily supportive care, including mechanical ventilation and antibiotics to prevent secondary infection as appropriate.[60] • Preliminary reports from two clinical trials in China suggest that favipiravir improves lung function and reduces recovery time in COVID-19 patients.[126] • Early results suggest that tocilizumab may be effective at treating severe COVID-19 cases.[145] • Press reports of a small clinical trial suggest that chloroquine is effective at reducing symptom duration.[1] • Combination lopinavir and ritonavir with standard care was no more effective than standard care alone.[24] • Corticosteroids are commonly given to COVID-19 patients[153] at risk of ARDS,[146] but their use is not recommended by the US CDC.[29] • Multiple entities are working to produce a SARS-CoV-2 vaccine,[8] including NIH/NIAID,[63, 80] Moderna Therapeutics and Gilead Sciences,[2-3, 94] and Sanofi with HHS.[21] Moderna has begun phase 1 clinical vaccine trials in humans in WA state.[107] • Regeneron Pharmaceuticals has developed potential SARS-CoV-2 antibody therapies.[99] • The development of a coronavirus fusion inhibitor in the lab suggests efficacy across multiple human coronaviruses.[143] • Takeda Pharma (Japan) is working to create antibody treatments based on infected patient plasma.[62]	*SARS-CoV-2 Data* • SARS-CoV-2 can persist on plastic and stainless steel surfaces for up to 3 days (at 21-23°C, 40% RH), with a half-life of 13-16 hours.[125] • SARS-CoV-2 has an aerosol half-life of 2.7 hours (particles <5 µm, tested at 21-23°C and 65% RH).[125] *Surrogate Coronavirus data*: • Studies suggest that other coronaviruses can survive on non-porous surfaces up to 9-10 days (MHV, SARS-CoV)[25, 36], and porous surfaces for up to 3-5 days (SARS-CoV)[56] in air conditioned environments (20-25°C, 40-50% RH) • Coronavirus survival tends to be higher at lower temperatures and lower relative humidity (RH),[25, 36, 102, 127] though infectious virus can persist on surfaces for several days in typical office or hospital conditions[127] • SARS can persist with trace infectivity for up to 28 days at refrigerated temperatures (4°C) on surfaces.[25] • Beta-coronaviruses (e.g., SARS-CoV) may be more stable than alpha-coronaviruses (HCoV-229E).[102] • No strong evidence for reduction in transmission with seasonal increase in temperature and humidity.[92] • One hour after aerosolization approximately 63% of airborne MERS virus remained viable in a simulated office environment (25°C, 75% RH)[100] • The aerosol survival of related human coronavirus (229E) was relatively high, (half-life of ~67 hours at 20°C and 50% RH), indicating ~20% of infectious virus remained after 6 days.[70] Both higher and lower RH reduced HCoV-229E survival; lower temperatures improved survival.[70]

SARS-CoV-2 (COVID-19)	Clinical presentation – what are the signs and symptoms of an infected person?	Clinical diagnosis – are there tools to diagnose infected individuals? When during infection are they effective?	Medical treatment – are there effective treatments? Vaccines?	Environmental stability – how long does the agent live in the environment?
What do we need to know?	• How long does it take for infected individuals to recover outside of a healthcare setting? • Is the reduction in CFR through time an indication of better treatment, less overcrowding, or both?	• False positive/negative rates for tests • Eclipse phase of infection (time between infection and detectable disease) in an individual	• Is GS-5734 (remdesivir) effective in vivo (already used in clinical trials under Emergency Use Authorization)?[115] • Is the GLS-5000 MERS vaccine[147] cross-reactive against SARS-CoV-2? • Efficacy of antibody treatments developed for SARS[46, 119] and MERS[34] • What is the efficacy of various MERS and SARS Phase I/II vaccines and other therapeutics? • Are viral replicase inhibitors such as beta-D-N4-hydroxycytidine effective against SARS-CoV-2?[15]	• Stability of SARS-CoV-2 in aerosol, droplets, and other matrices (mucus/sputum, feces) • Particle size distribution (e.g., droplet, large droplet and true aerosol distribution) • Duration of SARS-CoV-2 infectivity via fomites and surface (contact hazard)? • Stability of SARS-CoV-2 on PPE (e.g., Tyvek, nitrile, etc.)
Who is doing experiments/has capabilities in this area?	- Jin Yin-tan Hospital, Wuhan, China - China-Japan Friendship Hospital, Beijing, China - Peking Union Medical College, Beijing, China - Capital Medical University, Beijing, China - Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing, China - Huazhong University of Science and Technology, Wuhan, China - The Central Hospital of Wuhan, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China - Tsinghua University School of Medicine, Beijing, China - Zhongnan Hospital of Wuhan University, Wuhan, China - Peking University First Hospital, Beijing, China - Peking University People's Hospital, Beijing, China - Tsinghua University-Peking University Joint Center for Life Sciences, Beijing, China - The Fifth Medical Center of PLA General Hospital, Beijing, China	*Performing work:* - CDC - Wuhan Institute of Virology - Public Health Agency of Canada - Doherty Institute of Australia - Cepheid - BGI - Fudan University	*Performing work:* - Peter Doherty Institute for Infection and Immunity - Academy of Military Medical Sciences, Beijing, China - Tim Sheahan (University of North Carolina) - Takeda Pharma. (Japan) - Regeneron Pharmaceuticals - CureVac (Germany) *Capable of performing work:* - Ralph Baric (University of North Carolina) - Matthew Frieman (University of Maryland Baltimore) - Sanofi, with BARDA - Janssen Pharma and BARDA[64] *Funded work:* CEPI ($24 million to seven groups): NIAID/NIH: - Moderna and Kaiser Permanente for mRNA vaccine Phase I trial.[3] - University of Nebraska Medical Center Trial (multiple therapeutics including Gilead's Remdesivir).[2]	*Capable of performing work*: - Mark Sobsey (University of North Carolina) - DHS National Biodefense Analysis and Countermeasures Center (NBACC) - Defence Science and Technology Laboratory (Dstl) - Public Health Agency of Canada - CDC - EPA - NIH

SARS-CoV-2 (COVID-19)	Decontamination – what are effective methods to kill the agent in the environment?	PPE – what PPE is effective, and who should be using it?	Forensics – natural vs intentional use? Tests to be used for attribution.	Genomics – how does the disease agent compare to previous strains?
What do we know?	*SARS-CoV-2* • Twice-daily cleaning with sodium dichloroisocyanurate decontaminated surfaces in COVID-19 patient hospital rooms.[95] • Alcohol-based hand rubs are effective at inactivating SARS-CoV-2 in liquid.[75] • EPA has released a list of SARS-CoV-2 disinfectants, but solutions were not tested on live virus.[6] *Other Coronaviruses* • Chlorine-based[134] and ethanol-based[44] solutions recommended. • Heat treatment at 56°C is sufficient to kill coronaviruses,[102, 152] though effectiveness depends in part on amount of protein in contaminated media[102] • 70% ethanol, 50% isopropanol, sodium hypochlorite [bleach, 200 ppm], and UV radiation are effective at inactivating several coronaviruses (MHV and CCV)[112] • Ethanol-based biocides are effective disinfectants against coronaviruses dried on surfaces, including ethanol containing gels similar to hand sanitizer.[69, 139] • Surface spray disinfectants such as Mikrobac, Dismozon, and Korsolex are effective at reducing infectivity of the closely related SARS-CoV after 30 minutes of contact.[101] • Coronaviruses may be resistant to thermal inactivation for up to 7 days when stabilized in stool.[122-123] • Additionally, coronaviruses are more stable in matrixes such as respiratory sputum.[55] • Hydrogen peroxide vapor is expected to be effective at repeated decontamination of N95 respirators based on other pathogens.[105]	• PPE effectiveness for SARS-CoV-2 is currently unknown; SARS is used as a surrogate. • Healthcare worker illnesses (over 1,000[120]) demonstrates human-to-human transmission despite isolation, PPE, and infection control.[113] • US CDC does not recommend the use of facemasks for healthy people. Facemasks should be used by people showing symptoms to reduce the risk of others getting infected. The use of facemasks is crucial for health workers and people in close contact with infected patients (at home or in a health care facility).[28] • "Healthcare personnel entering the room [of SARS-CoV-2 patients] should use standard precautions, contact precautions, airborne precautions, and use eye protection (e.g., goggles or a face shield)"[30] • WHO indicates healthcare workers should wear clean, non-sterile, long-sleeve gowns as well as gloves.[133] • Respirators (NIOSH-certified N95, EUFFP2 or equivalent) are recommended for those dealing with possible aerosols[134] • Additional protection, such as a Powered Air Purifying Respirator (PAPR) with a full hood, should be considered for high-risk procedures (i.e., intubation, ventilation)[23] • SARS-CoV-2 transmission has occurred in hospitals inside[130] and outside of China,[61] including the US.[20] • Porous hospital materials, including paper and cotton cloth, maintain infectious SARS-CoV for a shorter time than non-porous material.[76] • Despite extensive environmental contamination, air sampling in patient rooms did not detect SARS-CoV-2.[95]	• Genomic analysis places SARS-CoV-2 into the beta-coronavirus clade, with close relationship to bat viruses. The SARS-CoV-2 virus is distinct from SARS and MERS viruses.[52] • Genomic analysis suggest that SARS-CoV-2 is a natural variant, and is therefore unlikely to be human-derived or otherwise created by "recombination" with other circulating strains of coronavirus.[9, 154] • Some genomic evidence indicates a close relationship with pangolin coronaviruses[140]; data suggests that pangolins may be a natural host for beta-coronaviruses [87-88]. Additional research is needed. • Genomic data support at least two plausible origins of SARS-CoV-2: "(i) natural selection in a non-human animal host prior to zoonotic transfer, and (ii) natural selection in humans following zoonotic transfer."[9] Either scenario is consistent the observed genetic changes found in all known SARS-CoV-2 isolates. • Additionally, "[…] SARS-CoV-2 is not derived from any previously used virus backbone," reducing the likelihood of laboratory origination,[9] and "[…] genomic evidence does not support the idea that SARS-CoV-2 is a laboratory construct, [though] it is currently impossible to prove or disprove the other theories of its origin."[9]	• There have been no documented cases of SARS-CoV-2 prior to December 2019 • Preliminary genomic analyses, however, suggest that the first human cases of SARS-CoV-2 emerged between 10/19/2019 – 12/17/2019.[10, 18, 103] • The mutation rate of SARS-CoV-2 is estimated to be similar to other RNA viruses (e.g., SARS, Ebola, Zika), and is currently calculated to be 1.04×10^{-3} substitutions per site per year (N = 116 genomes).[65] • Preliminary phylogenetic analysis identified a very close genetic similarity between SARS-CoV-2 and a Bat coronavirus (RaTG13) isolated from Yunnan Province, China; suggesting that SARS-CoV-2 originated from bats.[97] • Pangolin coronaviruses are closely related to both SARS-CoV-2 and the closely related Bat coronavirus (RaTG13); phylogenetic analysis suggested that SARS-CoV-2 is of bat origin, but is closely related to pangolin coronavirus.[87-88] • The Spike protein of SARS-CoV-2, which mediates entry into host cells and is the major determinant of host range, is very similar to the Spike protein of SARS-CoV.[91] The rest of the genome is more closely related to two separate bat [91] and pangolin[88] coronavirus. • Analysis of SARS-CoV-2 sequences from Singapore has identified a large nucleotide (382 bp) deletion in ORF-8 that may result in an attenuated (less virulent) phenotype.[118]

SARS-CoV-2 (COVID-19)	Decontamination – what are effective methods to kill the agent in the environment?	PPE – what PPE is effective, and who should be using it?	Forensics – natural vs intentional use? Tests to be used for attribution.	Genomics – how does the disease agent compare to previous strains?
What do we need to know?	• What is the minimal contact time for disinfectants? • Does contamination with human fluids/waste alter disinfectant efficacy profiles? • How effective is air filtration at reducing transmission in healthcare, airplanes and public spaces?	• Mode of aerosol transmission? Effective distance of spread via droplet or aerosol? • How effective are barriers such as N95 respirators or surgical masks? • What is the appropriate PPE for first responders? Airport screeners? • Proper procedures for reducing spread in medical facilities / transmission rate in medical settings	• What tests for attribution exist for coronavirus emergence? • What is the identity of the intermediate species? • Are there closely related circulating coronaviruses in bats or other animals with the novel PRRA cleavage site found in SARS-CoV-2?	• Are there similar genomic differences in the progression of coronavirus strains from bat to intermediate species to human? • Are there different strains or clades of circulating virus? If so, do they differ in virulence?
Who is doing experiments/has capabilities in this area?	*Capable of performing work*: - DHS National Biodefense Analysis and Countermeasures Center (NBACC)	*Generating recommendations:* - WHO - CDC - Pan-American Health Organization	*Performing genomic investigations:* - Kristian Andersen, Andrew Rambaut, Ian Lipkin, Edward Holmes, Robert Garry (Scripps, University of Edinburgh, Columbia University, University of Sydney, Tulane, Zalgen Labs [Germantown, MD]) *Capable of performing work:* - Pacific Northwest National Laboratory - DHS National Biodefense Analysis and Countermeasures Center (NBACC)	*Performing work:* - Trevor Bedford (Fred Hutchinson Cancer Research Center) - (b)(3) (b)(3) UNC - National Institute for Viral Disease Control and Prevention, Chinese Center for Disease Control and Prevention - Shandong First Medical University and Shandong Academy of Medical Sciences - Hubei Provincial Center for Disease Control and Prevention - Chinese Academy of Sciences - BGI PathoGenesis Pharmaceutical Technology, Shenzhen, China - People's Liberation Army General Hospital, Wuhan, China - Wenzhou Medical University, Wenzhou, China - University of Sydney, Sydney, NSW, Australia - The First Affiliated Hospital of Shandong First Medical University (Shandong Provincial Qianfoshan Hospital), Jinan, China

Table 1. Definitions of commonly-used acronyms

Acronym/Term	Definition	Description
Attack Rate	Proportion of "at-risk" individuals who develop infection	Defined in terms of "at-risk" population such as schools or households, defines the proportion of individuals in those populations who become infected after contact with an infectious individual
SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2	Official name for the virus previously known as 2019-nCoV.
COVID-19	Coronavirus disease 19	Official name for the disease caused by the SARS-CoV-2 virus.
CFR	Case Fatality Rate	Number of deaths divided by confirmed patients
PFU	Plaque forming unit	Measurement of the number of infectious virus particles as determined by plaque forming assay. A measurement of sample infectivity.
$TCID_{50}$	50% Tissue Culture Infectious Dose	The number of infectious units which will infect 50% of tissue culture monolayers. A measurement of sample infectivity.
HCW	Healthcare worker	Doctors, nurses, technicians dealing with patients or samples
SARS	Severe Acute Respiratory Syndrome	Coronavirus with over 8,000 cases in global 2002-2003 outbreak
MERS	Middle-East Respiratory Syndrome	Coronavirus with over 2,000 cases in regional outbreak since 2012
CoV	Coronavirus	Virus typified by crown-like structures when viewed under electron microscope
R_0	Basic reproduction number	A measure of transmissibility. Specifically, the average number of new infections caused by a typical infectious individual in a wholly susceptible population.
MHV	Mouse hepatitis virus	Coronavirus surrogate

CCV	Canine coronavirus	Canine coronavirus
Fomite	Inanimate vector of disease	Surfaces such as hospital beds, doorknobs, healthcare worker gowns, faucets, etc.
Droplet transmission	Sneezing, coughing	Transmission via droplets requires relatively close contact (e.g., within 6 feet)
Airborne transmission	Aerosolization of infectious particles	Aerosolized particles can spread for long distances (e.g., between hospital rooms via HVAC systems)
Transgenic	Genetically modified	In this case, animal models modified to be more susceptible to MERS and/or SARS by adding proteins or receptors necessary for infection
Intranasal	Agent deposited into external nares of subject	Simulates inhalation exposure by depositing liquid solution of pathogen/virus into the nose of a test animal, where it is then taken up by the respiratory system.
Incubation period	Time between infection and symptom onset	Time between infection and onset of symptoms typically establishes guidelines for isolating patients before transmission is possible
Infectious period	Length of time an individual can transmit infection to others	Reducing the infectious period is a key method of reducing overall transmission; hospitalization, isolation, and quarantine are all effective methods
Serial interval	Length of time between symptom onset of successive cases in a transmission chain	The serial interval can be used to estimate R_0, and is useful for estimating the rate of outbreak spread
Superspreading	One individual responsible for an abnormally large number of secondary infections	Superspreading can be caused by high variance in the distribution of secondary cases caused by a single individual; most individuals infect very few people, while some infect a large number, even with the same average number of secondary infections
Nosocomial	Healthcare- or hospital-associated infections	Characteristic of SARS and MERS outbreaks, lead to refinement of infection control procedures

ACE2	Angiotensin-converting enzyme 2	Acts as a receptor for SARS-CoV, allowing entry into human cells
ARDS	Acute respiratory distress syndrome	Leakage of fluid into the lungs which inhibits respiration and leads to death
PPE	Personal protective equipment	Gowns, masks, gloves, and any other measures used to prevent spread between individuals
PCR	Polymerase chain reaction	PCR (or real-time [RT] or quantitative [Q] PCR) is a method of increasing the amount of genetic material in a sample, which is then used for diagnostic testing to confirm the presence of SARS-CoV-2

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Subject:	Standing Committee on EID and 21st Century Health Threats: URGENT Call on Monoclonal Antibodies
Date:	2020/11/10 18:33:30
Start Date:	2020/11/15 11:00:00
End Date:	2020/11/15 13:00:00
Priority:	Normal
Type:	Appointment
Location:	https://nasem.zoom.us/j/96818499306
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Subject:	Today's Call/meeting info
Date:	2020/02/03 12:06:13
Priority:	Normal
Type:	Note

Thank you for participating in today's meeting of experts at the National Academies to discuss and identify what data, information and samples are needed to understand the evolutionary origins of 2019-nCoV and more effectively respond to the outbreak and resulting misinformation.

Attached for your information are:

Agenda

Scope of Work

A list of participants will be sent along shortly

Please let me know if you have any questions of problems with connecting.

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Andrew M. Pope, Ph.D.

Director

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Expert Meeting
Rapid Response for Assessment of Data Needs for 2019-nCoV

Agenda

February 3, 2020
2:00 p.m.–3:00 p.m. (ET)

Keck Center, Room 103
500 5th St NW, Washington, DC 20001

Join from PC, Mac, Linux, iOS or Android: https://nasem.zoom.us/j/777284900
Telephone: +1 646 558 8656
Meeting ID: 777 284 900
International numbers available: https://nasem.zoom.us/u/aA3S9pYwW

Meeting Objective: *Assess what data, information and samples are needed to understand the evolutionary origins of 2019-nCoV and more effectively respond to the outbreak and resulting misinformation.*

2:00 p.m. **Welcome and Introductions** (*5 mins*)

ANDREW POPE
Director, Board on Health Sciences Policy
National Academies of Sciences, Engineering, and Medicine

2:05 p.m. **Statement of Work** (*10 mins*)

KELVIN DROEGEMEIER
Director
Office of Science and Technology Policy

D. CHRISTIAN ("CHRIS") HASSELL
Senior Science Advisor
U.S. Department of Health and Human Services

2:15 p.m. **Perspective from NIH/NIAID** (*10 mins*)

ANTHONY ("TONY") S. FAUCI
Director
National Institute of Allergy and Infectious Diseases
National Institutes of Health

2:25 p.m. **Discussion of Meeting Objective** (*30 mins*)

2:55 p.m. **Determine Next Steps** (*5 mins*)

3:00 p.m. **Adjourn**

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Statement of Work

Rapid Response for Assessment of Data Needs for 2019-nCoV

February 3, 2020

Statement of Task:

In response to a request from OSTP, the NASEM will examine information and identify data requirements that would help determine the origins of 2019-nCoV, specifically from an evolutionary/structural biology standpoint. NASEM will also consider whether this should include more temporally and geographically diverse clinical isolates, sequences, etc. Although a widely-disputed paper posted on a pre-print server last week has since been withdrawn, the response to that paper highlights the need to determine these information needs as quickly as possible. As part of a broader deliberative process, this review will help prepare for future events by establishing a process for quickly assembling subject matter experts for evaluation of other potentially threatening organisms.

Workplan:

NASEM will hold a meeting of experts to assess what data, information and samples are needed to address the unknowns, in order to understand the evolutionary origins of NCoV and more effectively respond to both the outbreak and any resulting misinformation. A statement from the National Academies will be prepared and published on the Web as a "Based on Science" article that summarizes the status and needs for more and what types of data. A more in-depth examination of the issues will be established as a follow up as needed.

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Start Date:	2020/09/18 15:30:00
End Date:	2020/09/18 17:30:00
Priority:	Normal
Type:	Schedule.Meeting.Request
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Joint Meeting of the Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats, the Forum on Microbial Threats, and the Board on Global Health

Agenda
Friday, September 18, 2020 3:30 p.m. – 5:30 p.m. ET
Virtual Zoom Meeting

Background: In response to a request from the Office of Science and Technology Policy (OSTP) and the Office of the Assistant Secretary for Preparedness and Response (ASPR), the National Academies of Sciences, Engineering, and Medicine established the Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats. ASPR, in coordination with the Office of the Director of National Intelligence (ODNI) requested an expert meeting on the impact of globalization on future health crises. Given the expertise in both the Forum on Microbial Threats and the Board on Global Health in this topic, this meeting includes members of these bodies. Globalization has improved the world in many ways, but has also introduced fragility in supply chains, adversely affected the environment, witnessed the greatest number of refugees and internally displaced persons, expanded economic disparities, and increased the ability for novel pathogens to spread quickly around the globe. Factors such as extreme weather events and social and economic disruptions, including armed conflict, have also adversely affected global interdependence.

Meeting Objectives

Discuss and consider topics for further exploration on the following:

- What changes are associated with globalization that increase the probability of crises such as pandemics and other infectious disease events?
- What are potential mitigating factors against these threats?
- How do these different factors interact and create risks of compound events or cascading effects?
- And is there any predictability of how this is changing in frequency or impact?

FRIDAY, SEPTEMBER 18, 2020

CLOSED SESSION

SESSION I **Welcoming Remarks and Sponsors' Description of Need**

3:30 p.m. **Welcome and Charge to the Group**

Harvey Fineberg, *Standing Committee Chair*
President
Gordon and Betty Moore Foundation

3:45 p.m. **Sponsor Remarks**

(b)(6) **(Chris) Hassell**
Acting Principal Deputy Assistant Secretary
Senior Science Advisor
The Office of the Assistant Secretary for Preparedness and Response
U.S. Department of Health and Human Services

Kathryn Brinsfield
Senior Advisor
National Counterproliferation Center
Office of the Director of National Intelligence

4:00 p.m. **Topic Introduction**

Harvey Fineberg, *Standing Committee Chair*
President
Gordon and Betty Moore Foundation

Ann Kurth, *Board on Global Health Chair*
Dean and Professor
Yale University, School of Nursing

Peter Daszak, *Forum on Microbial Threats Chair*
President
EcoHealth Alliance

SESSION II **Group Discussions on the Challenges of:**

4:20 p.m. **Prediction Modeling**

Detection Assessment
Surveillance, monitoring, rapid threat assessment that is shared and accessible

Response
Preparedness, near term and focal, long-term and dispersed

5:15 p.m. **Discussion of Next Steps with the Sponsors**

Harvey Fineberg, *Committee Chair*
President
Gordon and Betty Moore Foundation

5:30 p.m. **ADJOURN**

SARS-CoV-2 (COVID-19)	Infectious dose – how much agent will make a normal individual ill?	Transmissibility – How does it spread from one host to another? How easily is it spread?	Host range – how many species does it infect? Can it transfer from species to species?	Incubation period – how long after infection do symptoms appear? Are people infectious during this time?
What do we know?	• The human infectious dose for novel Wuhan coronavirus (SARS-CoV-2), which causes coronavirus disease 19 (COVID-19) is currently unknown via all exposure routes. Severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS) coronaviruses (CoV) are used as surrogates. • The infectious dose for SARS in mice is estimated to be between 67-540 PFU (average 240 PFU, intranasal route).[50-51] • Genetically modified mice exposed intranasally to doses of MERS virus between 100 and 500,000 PFU show signs of infection. Infection with higher doses result in severe syndromes.[5, 41, 73, 129] • Initial experiments suggest that SARS-CoV-2 can infect genetically modified mice containing the human ACE2 cell entry receptor. Infection via the intranasal route (dose: 10^5 $TCID_{50}$) causes light infection, however no virus was isolated from infected animals, and PCR primers used in the study do not align well with SARS-CoV-2, casting doubt on this study.[11]	• The WHO has declared SARS-CoV-2 a Public Health Emergency of International Concern[116] with 95,124 cases and 3,254 deaths[68] in 75 countries (as of 3/4/2020).[22, 96, 114] • High-quality estimates of human transmissibility (R_0) range from 2.2 to 3.1[80, 86, 91, 120, 128] • Large outbreaks are occurring in Japan, Italy, Iran, South Korea, Germany France, and Spain.[110] • There are 153 SARS-CoV-2 cases across 15 US states, with 11 deaths. (as of 3/4/2020).[68] • Sustained transmission may have been occurring in the US (Seattle) for up to 5 or 6 weeks.[14] • SARS-CoV-2 transmission has occurred in hospitals inside[108] and outside of China,[58] including the US.[17] • Pre-symptomatic[130] or asymptomatic[10] patients in China can transmit SARS-CoV-2; the degree of asymptomatic transmission is still unknown. • SARS-CoV-2 is believed to spread through close contact and droplet transmission.[30] • Viable SARS-CoV-2 has been isolated from human feces; fecal-oral transmission is possible.[81, 124, 127] • Transmission via fomites has not been confirmed for SARS-CoV-2, but occurred in prior SARS[40, 121] and MERS[70] outbreaks • SARS-CoV-2 is consistently present in infected patient saliva[104] • Infants have been diagnosed with COVID-19, but no evidence exists for vertical transmission via intrauterine infection or through breastmilk.[38, 109] • China reports no evidence of super-spreading events (SSEs) within hospital patients or staff.[101]	• Early genomic analysis indicates similarity to SARS,[132] with a suggested bat origin.[5,42, 132] • Analysis of SARS-CoV-2 genomes suggests that a non-bat intermediate species is responsible for the beginning of the outbreak.[92] Although the identity of the intermediate species remains unconfirmed, pangolins may be a natural host of related viruses possibly including SARS-CoV-2.[76-77] • Positive samples from the South China Seafood Market strongly suggests a wildlife source,[32] though it is possible that the virus was circulating in humans before the disease was associated with the seafood market.[15, 43, 122, 126] • Experiments suggest that SARS-CoV-2 Spike (S) receptor-binding domain binds the human cell receptor (ACE2) stronger than SARS,[119] potentially explaining its high transmissibility; the same work suggests that differences between SARS-CoV-2 and SARS-CoV Spike proteins may limit the therapeutic ability of SARS antibody treatments.[119] • Modeling between SARS-CoV-2 Spike and ACE2 proteins suggests that SARS-CoV-2 can bind and infect human, bat, civet, monkey and swine cells.[107]	• A study of 1,099 COVID-19 patients found a median incubation period of 3 days, with a range from 0 to 24 days.[57] • Earlier estimates of the incubation period from confirmed cases were higher; 5.8 days with a range from 1.3 to 11.3 days,[9] and 5.2 days with an upper bound of 9.2-18 days.[75] • CDC estimates that the incubation period is between 2 and 14 days [27, 31] • Asymptomatic infection has been documented, where individuals do not present with clinical symptoms but are found positive via diagnostic assay.[10, 34, 57, 101, 130] • Individuals can be infectious while asymptomatic,[30, 93, 101, 130] and asymptomatic individuals have about the same amount of virus in their nose and throat as symptomatic individuals.[133] • Infectious period is unknown, but possibly up to 10-14 days [4, 96] • On average, there are 7.5 days between symptom onset in successive cases of a single transmission chain (i.e., the serial interval).[75] • The average time for individuals to first seek medical care decreased from 5.8 days after symptom onset to 4.6 days before and after January 1st, 2020, respectively, indicating an increase in seeking care behavior.[75] • China recommends 14 quarantine for *recovered* patients due to positive genetic tests days after leaving the hospital, raising the possibility of continued transmission after symptoms subside.[64]

SARS-CoV-2 (COVID-19)	Infectious dose – how much agent will make a normal individual ill?	Transmissibility – How does it spread from one host to another? How easily is it spread?	Host range – how many species does it infect? Can it transfer from species to species?	Incubation period – how long after infection do symptoms appear? Are people infectious during this time?
What do we need to know?	• Human infectious dose by aerosol route • Human infectious dose by surface contact (fomite) • Human infectious dose by fecal-oral route • Where does SARS-CoV-2 replicate in the respiratory tract?	• Capability of SARS-CoV-2 to be transmitted by contact with fomites (doorknobs, surfaces, clothing, etc.) – see also Experimental Stability • Superspreading capacity needs to be refined • Updated person to person transmission rates (e.g., R_0) as control measures take effect • Tendency for ill individuals to seek medical care due to symptoms • What is the underreporting rate?[67] • Can individuals become re-infected with SARS-CoV-2? • What is the difference in transmissibility among countries? • Is the R0 estimate higher in healthcare or long-term care facilities? • How effective are social distancing measures at reducing spread?	• What is the intermediate host(s)? • What are the mutations in SARS-CoV-2 that allowed human infection and transmission? • What animals can SARS-CoV-2 infect (e.g., pet dogs, potential wildlife reservoirs)?	• How early does asymptomatic transmission begin? • What is the average infectious period during which individuals can transmit the disease? • How long do patients continue to shed infectious virus after physical recovery? • Can individuals become re-infected after recovery? If so, how long after?
Who is doing experiments/has capabilities in this area?	*Capable of performing work* - DHS National Biodefense Analysis and Countermeasures Center (NBACC)	*Performing work:* - Christian Althaus (Bern) - Neil Ferguson (MRC) - Gabriel Leung, Joseph Wu (University of Hong Kong) - Sara Del Valle (Los Alamos) - Maimuna Majumder (Boston Children's Hospital) - Trevor Bedford (Fred Hutchinson Cancer Center) - Sang Woo Park (Princeton)	*Capable of performing work:* - Vincent Munster (Rocky Mountain National Laboratory) - Matthew Frieman (University of Maryland Baltimore) - Ralph Baric (University of North Carolina) - Stanley Perlman (University of Iowa) - Susan Baker (Loyola University Chicago) - Mark Denison (Vanderbilt University) - Vineet Menachery (University of Texas Medical Branch) - Jason McLellan, Daniel Wrapp, Nianshuang Wang (University of Texas) - David O'Conner (U. Wisconsin, Madison)	*Performing work:* - Chaolin Huang (Jin Yin-tan Hospital, Wuhan, China) - The Novel Coronavirus Pneumonia Emergency Response Epidemiology Team

SARS-CoV-2 (COVID-19)	Clinical presentation – what are the signs and symptoms of an infected person?	Clinical diagnosis – are there tools to diagnose infected individuals? When during infection are they effective?	Medical treatment – are there effective treatments? Vaccines?	Environmental stability – how long does the agent live in the environment?
What do we know?	• The majority of COVID-19 cases are mild (81%, N = 44,000 cases)[101] • Initial COVID-19 symptoms include fever (87.9% overall, but only 43.8% present with fever initially[57]), cough (67.7%[57]), fatigue, shortness of breath, headache, reduction in lymphocyte count.[31, 37, 63] Headache[36] and diarrhea are uncommon[63, 74] • Complications include acute respiratory distress (ARDS) observed in 17-29% of hospitalized patients,[39, 62] which leads to death in 4-15% of cases.[39, 63, 108] • Other complications include pneumonia (characteristic ground glass opacities[84]), acute cardiac injury, secondary infection, kidney failure, arrhythmia, and shock.[57,63,108] • Approximately 15% of hospitalized patients were classified as severe,[57, 101] and severe cases were older and more likely to have underlying disorders[57, 108]; approximately 5% of patients were admitted to the ICU.[57, 101] • Between 23-32% of cases that include pneumonia required intensive respiratory support.[63, 108] • Overactive immune cells may contribute to symptom severity.[123] • Approximately 1% of hospitalizations occur in children < 19 years old.[57, 101] • The case fatality rate (CFR) depends on patient comorbidities; no comorbidities = 0.9%, cardiovascular disease = 10.5%, diabetes = 7.3%, chronic respiratory disease = 6.3%, hypertension = 6.0%, cancer = 5.6%.[101] • The CFR is age-dependent; ≥80 years old = 14.8%, 70-79 = 8.0%, 60-69 = 3.6%, 50-59 = 1.3%, 40-49 = 0.4%, 10-39 = 0.2%, 0-9 = 0%.[101] • 63.8% of confirmed fatalities have been male.[101]	• Updated tests from the US CDC are available to states.[21, 30] • The FDA released an Emergency Use Authorization describing an accelerated policy enabling laboratories to develop and use tests in-house for patient diagnosis.[56] • The US has relaxed criteria for testing patients, no longer requires travel history or close contact with known case.[13] • US CDC has expanded patient testing criteria to include symptomatic patients at Clinician discretion.[13] CDC recommends that testing decisions should be based on local transmission, travel history, patient clinical course, close contact with infected patients, and occupational risk (e.g., Health Care Workers).[24] • SARS-CoV-2 is consistently present in infected patient saliva, suggesting that saliva may be an effective diagnostic specimen.[104] • Several RT-PCR assays have been developed to detect SARS-CoV-2 in humans.[1, 46, 113, 115] • PCR protocols and primers have been widely shared among international researchers.[21, 47, 75, 99, 111, 115] • Several rapid or real-time test kits have been produced by universities and industry, including the Wuhan Institute of Virology,[49] BGI,[16] and Cepheid.[106] • RT-PCR tests are able to identify asymptomatic cases; SARS-CoV-2 infection was identified in 2/114 individuals previously cleared by clinical assessment.[61]	• Treatment for COVID-19 is primarily supportive care including oxygen and mechanical ventilation,[29] though China has released a treatment plan[8]; over 80 clinical trials are set to run on various treatments in China.[82] • Efficacy antivirals (lopinavir, ritonavir, ribavirin, oseltamivir) is unknown[8]; however several therapeutics [Remdesivir[98] and chloroquine] inhibit SARS-CoV-2 infection in human cells *in vitro*[38] and are undergoing clinical trials in China[49] and the US.[2-3, 83] • Multiple entities are working to produce a SARS-CoV-2 vaccine, including NIH/NIAID,[59, 72] Moderna Therapeutics and Gilead Sciences,[2-3, 83] and Sanofi with HHS.[18] • The hospitalized case-fatality rate in China has decreased from 14.4% to 0.8% as of between December, 2019 and February, 2020,[101] suggesting improved treatment or increased capacity • Approximately 38% of COVID-19 patients in China received oxygen therapy, 6.1% received mechanical ventilation, 57.5% received IV antibiotics, and 35.8% received the antiviral oseltamivir.[57] • A clinical report (one patient) suggested that corticosteroids should be considered for severe patients to prevent ARDS.[123] However, US CDC recommends avoiding steroid use due to an increase in viral replication in MERS patients.[25] • Similarity in the spike proteins of SARS-CoV-2 and SARS-CoV might offer target for therapeutics,[44, 52, 71, 119, 132] as vaccines derived from spike proteins are effective at inhibiting MERS symptoms in mice.[41]	• No information yet exists regarding the environmental stability of SARS-CoV-2; SARS and MERS coronaviruses are used as surrogates instead. • Studies suggest that coronavirus can survive on non-porous surfaces up to 9-10 days (MHV, SARS-CoV)[20, 35], and porous surfaces for up to 3-5 days (SARS-CoV)[54] in air conditioned environments (20-25°C, 40-50% RH) • Coronavirus survival tends to be higher at lower temperatures and lower relative humidity (RH),[20, 35, 89, 105] though infectious virus can persist on surfaces for several days in typical office or hospital conditions[105] • SARS can persist with trace infectivity for up to 28 days at refrigerated temperatures (4°C) on surfaces.[20] • Beta-coronaviruses (e.g., SARS-CoV) may be more stable than alpha-coronaviruses (HCoV-229E).[89] • No strong evidence for reduction in transmission with seasonal increase in temperature and humidity.[79] • Survival of SARS-CoV-2 specifically is unknown, and surrogate coronavirus data need to be used at this time. • One hour after aerosolization approximately 63% of airborne MERS virus remained viable in a simulated office environment (25°C, 75% RH)[87] • The aerosol survival of related human coronavirus (229E) was relatively high, (half-life of ~67 hours at 20°C and 50% RH), indicating ~20% of infectious virus remained after 6 days.[66] Both higher and lower RH reduced HCoV-229E survival; lower temperatures improved survival.[66]

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What do we need to know?	• How long does it take for infected individuals to recover outside of a healthcare setting? • How does the CFR vary between countries? • Is the reduction in CFR through time an indication of better treatment, less overcrowding, or both?	• False positive/negative rates for tests • Eclipse phase of infection (time between infection and detectable disease) in an individual	• Is GS-5734 (remdesivir) effective in vivo (already used in clinical trials under Emergency Use Authorization)?[97] • Is the GLS-5000 MERS vaccine[125] cross-reactive against SARS-CoV-2? • Efficacy of antibody treatments developed for SARS[48, 100] and MERS[33] • What is the efficacy of various MERS and SARS Phase I/II vaccines and other therapeutics? • Are viral replicase inhibitors such as beta-D-N4-hydroxycytidine effective against SARS-CoV-2?[12]	• Stability of SARS-CoV-2 in aerosol, droplets, and other matrices (mucus/sputum, feces) • "Hang time' of the virus in air (Aerosol decay rate) • Particle size distribution (e.g., droplet, large droplet and true aerosol distribution) • Duration of SARS-CoV-2 infectivity via fomites and surface (contact hazard)? • Stability of SARS-CoV-2 on PPE (e.g., Tyvek, nitrile, etc.)
Who is doing experiments/has capabilities in this area?	- Jin Yin-tan Hospital, Wuhan, China - China-Japan Friendship Hospital, Beijing, China - Peking Union Medical College, Beijing, China - Capital Medical University, Beijing, China - Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing, China - Huazhong University of Science and Technology, Wuhan, China - The Central Hospital of Wuhan, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China - Tsinghua University School of Medicine, Beijing, China - Zhongnan Hospital of Wuhan University, Wuhan, China - Peking University First Hospital, Beijing, China - Peking University People's Hospital, Beijing, China - Tsinghua University-Peking University Joint Center for Life Sciences, Beijing, China - The Fifth Medical Center of PLA General Hospital, Beijing, China	*Performing work:* - CDC - Wuhan Institute of Virology - Public Health Agency of Canada - Doherty Institute of Australia - Cepheid - BGI	*Performing work:* - Peter Doherty Institute for Infection and Immunity - Academy of Military Medical Sciences, Beijing, China - Tim Sheahan (University of North Carolina) *Capable of performing work:* - (b)(6) (b)(6) (University of North Carolina) - (b)(6) Frieman (University of Maryland Baltimore) - Sanofi, with BARDA - Janssen Pharma and BARDA[60] *Funded work:* CEPI ($12 million to three groups): - Moderna and NIAID for mRNA platform vaccine - Inovio preparing DNA vaccine (for MERS) - University of Queensland, Australia NIAID/NIH: - Moderna and Kaiser Permanente for mRNA vaccine Phase I trial.[3] - University of Nebraska Medical Center Trial (multiple therapeutics including Gilead's Remdesivir).[2]	*Capable of performing work*: - (b)(6) Sobsey (University of North Carolina) - DHS National Biodefense Analysis and Countermeasures Center (NBACC) - Defence Science and Technology Laboratory (Dstl) - Public Health Agency of Canada - CDC - EPA - NIH

SARS-CoV-2 (COVID-19)	Decontamination – what are effective methods to kill the agent in the environment?	PPE – what PPE is effective, and who should be using it?	Forensics – natural vs intentional use? Tests to be used for attribution.	Genomics – how does the disease agent compare to previous strains?
What do we know?	• No decontamination data for SARS-CoV-2 have been identified. SARS-CoV provides a plausible surrogate, as it is a close genetic relative of SARS-CoV-2 in the beta-coronavirus clade. • Chlorine-based[113] and ethanol-based[45] solutions recommended, and the European CDC has released disinfectant guidelines for non-healthcare facilities.[55] • "The virus [SARS-CoV-2] has relatively weak viability *in vitro* and can be inactivated at 56 ° C for 30 minutes. Chlorine-containing disinfectants and 75% ethanol can effectively inactivate the virus."[131] • Heat treatment at 56°C is sufficient to kill coronaviruses,[89, 131] though effectiveness depends in part on amount of protein in contaminated media[89] • 70% ethanol, 50% isopropanol, sodium hypochlorite [bleach, 200 ppm], and UV radiation are effective at inactivating several coronaviruses (MHV and CCV)[94] • Ethanol-based biocides (including ethanol-containing gels) are effective disinfectants against coronaviruses dried on surfaces, including ethanol containing gels similar to hand sanitizer.[65, 117] • Surface spray disinfectants such as Mikrobac, Dismozon, and Korsolex are effective at reducing infectivity of the closely related SARS-CoV after 30 minutes of contact.[88] • Coronaviruses may be resistant to thermal inactivation for up to 7 days when stabilized in stool.[102-103] • Additionally, coronaviruses are more stable in matrixes such as respiratory sputum.[53]	• PPE effectiveness for SARS-CoV-2 is currently unknown; SARS is used as a surrogate. • US CDC does not recommend the use of face masks for healthy people. Face masks should be used by people showing symptoms to reduce the risk of others getting infected. The use of face masks is crucial for health workers and people in close contact with infected patients (at home or in a health care facility).[23] • "Healthcare personnel entering the room [of SARS-CoV-2 patients] should use standard precautions, contact precautions, airborne precautions, and use eye protection (e.g., goggles or a face shield)"[28] • WHO indicates healthcare workers should wear clean, non-sterile, long-sleeve gowns as well as gloves.[112] • Respirators (NIOSH-certified N95, EUFFP2 or equivalent) are recommended for those dealing with possible aerosols[113] • Additional protection, such as a Powered Air Purifying Respirator (PAPR) with a full hood, should be considered for high-risk procedures (i.e., intubation, ventilation)[19] • Healthcare worker illnesses (over 1,000[101]) demonstrates human-to-human transmission despite isolation, PPE, and infection control.[95] • Porous hospital materials, including paper and cotton cloth, maintain infectious SARS-CoV for a shorter time than non-porous material.[69] • CDC recommends face masks for individuals attempting to prevent spread of SARS-CoV-2 in the home.[26]	• Genomic analysis places SARS-CoV-2 into the beta-coronavirus clade, with close relationship to bat viruses. The SARS-CoV-2 virus is distinct from SARS and MERS viruses.[52] • Genomic analysis suggest that SARS-CoV-2 is a natural variant, and is therefore unlikely to be human-derived or otherwise created by "recombination" with other circulating strains of coronavirus.[6, 132] • Some genomic evidence indicates a close relationship with pangolin coronaviruses[118]; data suggests that pangolins may be a natural host for beta-coronaviruses [76-77]. Additional research is needed. • Genomic data support at least two plausible origins of SARS-CoV-2: "(i) natural selection in a non-human animal host prior to zoonotic transfer, and (ii) natural selection in humans following zoonotic transfer."[6] Either scenario is consistent the observed genetic changes found in all known SARS-CoV-2 isolates. • Additionally, "[…] SARS-CoV-2 is not derived from any previously used virus backbone," reducing the likelihood of laboratory origination,[6] and "[…] genomic evidence does not support the idea that SARS-CoV-2 is a laboratory construct, [though] it is currently impossible to prove or disprove the other theories of its origin."[6]	• There have been no documented cases of SARS-CoV-2 prior to December 2019 • Preliminary genomic analyses, however, suggest that the first human cases of SARS-CoV-2 emerged between 10/19/2019 – 12/17/2019.[7, 15, 90] • The mutation rate of SARS-CoV-2 is estimated to be similar to other RNA viruses (e.g., SARS, Ebola, Zika), and is currently calculated to be between $3.29 \times 10^{-4} – 2.03 \times 10^{-3}$ substitutions per site per year (median 1.07×10^{-3}),[7] though this estimate may change as more genomes are sequenced. • Preliminary phylogenetic analysis identified a very close genetic similarity between SARS-CoV-2 and a Bat coronavirus (RaTG13) isolated from Yunnan Province, China; suggesting that SARS-CoV-2 originated from bats.[85] • Pangolin coronaviruses are closely related to both SARS-CoV-2 and the closely related Bat coronavirus (RaTG13); phylogenetic analysis suggested that SARS-CoV-2 is of bat origin, but is closely related to pangolin coronavirus.[76-77] • The Spike protein of SARS-CoV-2, which mediates entry into host cells and is the major determinant of host range, is very similar to the Spike protein of SARS-CoV.[78] The rest of the genome is more closely related to two separate bat [78] and pangolin[77] coronavirus. • Protein modeling and preliminary laboratory studies suggest that SARS-CoV-2 binds to the human ACE2 receptor,[107, 119] the same cellular entry receptor used by SARS and other beta-coronaviruses.

SARS-CoV-2 (COVID-19)	Decontamination – what are effective methods to kill the agent in the environment?	PPE – what PPE is effective, and who should be using it?	Forensics – natural vs intentional use? Tests to be used for attribution.	Genomics – how does the disease agent compare to previous strains?
What do we need to know?	• What is the minimal contact time for disinfectants? • Are antiseptic wipes effective for cleaning hard, non-porous surfaces? • Does contamination with human fluids/waste alter disinfectant efficacy profiles? • How effective is air filtration at reducing transmission in healthcare, airplanes and public spaces?	• Mode of aerosol transmission? Effective distance of spread via droplet or aerosol? • Is virus detectable in aerosol samples from patient rooms? • How effective are barriers such as N95 respirators or surgical masks? • What is the appropriate PPE for first responders? • What are the proper procedures for reducing spread in medical facilities / transmission rate in medical settings?	• What tests for attribution exist for coronavirus emergence? • What is the identity of the intermediate species? • Are there closely related circulating coronaviruses in bats or other animals with the novel PRRA cleavage site found in SARS-CoV-2?	• Are there similar genomic differences in the progression of coronavirus strains from bat to intermediate species to human? • Are there different strains or clades of circulating virus? If so, do they differ in virulence?
Who is doing experiments/has capabilities in this area?	*Capable of performing work*: - DHS National Biodefense Analysis and Countermeasures Center (NBACC)	*Generating recommendations:* - WHO - CDC - Pan-American Health Organization	*Performing genomic investigations:* - Kristian Andersen, Andrew Rambaut, Ian Lipkin, Edward Holmes, Robert Garry (Scripps, University of Edinburgh, Columbia University, University of Sydney, Tulane, Zalgen Labs [Germantown, MD]) *Capable of performing work:* - Pacific Northwest National Laboratory - DHS National Biodefense Analysis and Countermeasures Center (NBACC)	*Performing work:* - Trevor Bedford (Fred Hutchinson Cancer Research Center) - (b)(6) (b)(6) UNC - National Institute for Viral Disease Control and Prevention, Chinese Center for Disease Control and Prevention - Shandong First Medical University and Shandong Academy of Medical Sciences - Hubei Provincial Center for Disease Control and Prevention - Chinese Academy of Sciences - BGI PathoGenesis Pharmaceutical Technology, Shenzhen, China - People's Liberation Army General Hospital, Wuhan, China - Wenzhou Medical University, Wenzhou, China - University of Sydney, Sydney, NSW, Australia - The First Affiliated Hospital of Shandong First Medical University (Shandong Provincial Qianfoshan Hospital), Jinan, China

Table 1. Definitions of commonly-used acronyms

Acronym/Term	Definition	Description
SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2	Official name for the virus previously known as 2019-nCoV.
COVID-19	Coronavirus disease 19	Official name for the disease caused by the SARS-CoV-2 virus.
CFR	Case Fatality Rate	Number of deaths divided by confirmed patients
PFU	Plaque forming unit	Measurement of the number of infectious virus particles as determined by plaque forming assay. A measurement of sample infectivity.
$TCID_{50}$	50% Tissue Culture Infectious Dose	The number of infectious units which will infect 50% of tissue culture monolayers. A measurement of sample infectivity.
HCW	Healthcare worker	Doctors, nurses, technicians dealing with patients or samples
SARS	Severe Acute Respiratory Syndrome	Coronavirus with over 8,000 cases in global 2002-2003 outbreak
MERS	Middle-East Respiratory Syndrome	Coronavirus with over 2,000 cases in regional outbreak since 2012
CoV	Coronavirus	Virus typified by crown-like structures when viewed under electron microscope
R_0	Basic reproduction number	A measure of transmissibility. Specifically, the average number of new infections caused by a typical infectious individual in a wholly susceptible population.
MHV	Mouse hepatitis virus	Coronavirus surrogate
CCV	Canine coronavirus	Canine coronavirus
Fomite	Inanimate vector of disease	Surfaces such as hospital beds, doorknobs, healthcare worker gowns, faucets, etc.

Droplet transmission	Sneezing, coughing	Transmission via droplets requires relatively close contact (e.g., within 6 feet)
Airborne transmission	Aerosolization of infectious particles	Aerosolized particles can spread for long distances (e.g., between hospital rooms via HVAC systems)
Transgenic	Genetically modified	In this case, animal models modified to be more susceptible to MERS and/or SARS by adding proteins or receptors necessary for infection
Intranasal	Agent deposited into external nares of subject	Simulates inhalation exposure by depositing liquid solution of pathogen/virus into the nose of a test animal, where it is then taken up by the respiratory system.
Incubation period	Time between infection and symptom onset	Time between infection and onset of symptoms typically establishes guidelines for isolating patients before transmission is possible
Infectious period	Length of time an individual can transmit infection to others	Reducing the infectious period is a key method of reducing overall transmission; hospitalization, isolation, and quarantine are all effective methods
Serial interval	Length of time between symptom onset of successive cases in a transmission chain	The serial interval can be used to estimate R_0, and is useful for estimating the rate of outbreak spread
Superspreading	One individual responsible for an abnormally large number of secondary infections	Superspreading can be caused by high variance in the distribution of secondary cases caused by a single individual; most individuals infect very few people, while some infect a large number, even with the same average number of secondary infections
Nosocomial	Healthcare- or hospital-associated infections	Characteristic of SARS and MERS outbreaks, lead to refinement of infection control procedures
ACE2	Angiotensin-converting enzyme 2	Acts as a receptor for SARS-CoV, allowing entry into human cells

ARDS	Acute respiratory distress syndrome	Leakage of fluid into the lungs which inhibits respiration and leads to death
PPE	Personal protective equipment	Gowns, masks, gloves, and any other measures used to prevent spread between individuals

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	(b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=user4629b831 (b)(6) KAUSHIK, SANGEETA (b)(6) (b)(6) (b)((b)(6) Lee, Scott (OS/ASPR/EMMO) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=userf6879348 (b)(6) Padget, Larry G (b)(6) (b) (b)(6) (b)(6) Stack, Steven J (CHFS DPH) (b)(6) Adams, Jerome (HHS/OASH) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=184fe51f7350400b8a99dabd65ac0db5-Adams, Jero (b)(6) Fantinato, Jessica (USDA.GOV) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=4a03b6317c3840afb4ee617008e821a6-Jessica.Fan <jessica.fantinato@usda.gov>; Colby, Michelle - OHS, Washington, DC <michelle.colby@usda.gov>
Subject:	RE: Red Dawn Breaking Bad, Start Feb 24
Date:	2020/02/27 16:22:04
Priority:	Normal
Type:	Note

Updated Master Question List.
Best,
Duane

From: Caneva, Duane
Sent: Thursday, February 27, 2020 4:15 PM
To: Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Additional information sources:

Forwarding information from one of our BAH team members, (b)(6) Mekaru, PhD, who has been working with colleagues at Harvard/BCH, Oxford, and Northeastern to develop a centralized repository of individual-level information on patients with laboratory-confirmed COVID-19 to include a map/data explorer visualization tool. This information is publicly available with credit to be given to the Open COVID-19 Data Curation Group on any publications/communications. Please feel free to disseminate further to anyone interested. If you have any questions, feel free to reach out. Thank you!

Map/Data Explorer: https://scarpino.shinyapps.io/Emergent_Epidemics_Lab_nCoV2019/
Link to Linelist: https://protect2.fireeye.com/url?k=b3f8f098-efacd9b3-b3f8c1a7-0cc47a6d17cc-ca3238e1b64510e4&u=https://docs.google.com/spreadsheets/d/1itaohdPiAeniCXNlntNztZ_oRvjh0HsGuJXUJWET008/edit#gid=0
Twitter Update Notifications: https://twitter.com/svscarpino/status/1230330483319484417, https://twitter.com/MOUGK
Summary (https://www.thelancet.com/journals/laninf/article/PIIS1473-3099(20)30119-5/fulltext)

- • Coronavirus disease 2019 (COVID-19) is spreading rapidly across China, and as of Feb 16, 2020, had been reported in 26 countries globally. The availability of accurate and robust

epidemiological, clinical, and laboratory data early in an epidemic is important to guide public health decision-making.1

- • Consistent recording of epidemiological information is important to understand transmissibility, risk of geographic spread, routes of transmission, and risk factors for infection, and to provide the baseline for epidemiological modelling that can inform planning of response and containment efforts to reduce the burden of disease. Furthermore, detailed information provided in real time is crucial for deciding where to prioritise surveillance.
- • Line list data are rarely available openly in real time during outbreaks. However, they enable a multiplicity of analyses to be undertaken by different groups, using various models and assumptions, which can help build consensus on robust inference. Parallels exist between this and the open sharing of genomic data.2

- • We have built a centralised repository of individual-level information on patients with laboratory-confirmed COVID-19 (in China, confirmed by detection of virus nucleic acid at the City and Provincial Centers for Disease Control and Prevention), including their travel history, location (highest resolution available and corresponding latitude and longitude), symptoms, and reported onset dates, as well as confirmation dates and basic demographics. Information is collated from a variety of sources, including official reports from WHO, Ministries of Health, and Chinese local, provincial, and national health authorities. If additional data are available from reliable online reports, they are included. Data are available openly and are updated on a regular basis (around twice a day).

- • We hope these data continue to be used to build evidence for planning, modelling, and epidemiological studies to better inform the public, policy makers, and international organizations and funders as to where and how to improve surveillance, response efforts, and delivery of resources, which are crucial factors in containing the COVID-19 epidemic.

- • The epidemic is unfolding rapidly and reports are outdated quickly, so it will be necessary to build computational infrastructure that can handle the large expected increase in case reports. Data sharing will be vital to evaluate and maintain accurate reporting of cases during this outbreak.3

From: (b)(6) (b)(6) (b)(6)
Sent: Thursday, February 27, 2020 1:29 PM
To: Dr. (b) (b) (b)(6) (b)(6) (b)(6) (b)(6)
Cc: Tracey (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Caneva, Duane (b)(6) Martin, (b)(6) J (b)(6) Walters, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR) <Daniel.Dodgen@hhs.gov>; DeBord, Kristin (OS/ASPR/SPPR) <Kristin.DeBord@hhs.gov>; Phillips, Sally (OS/ASPR/SPPR) <Sally.Phillips@hhs.gov>; (b)((b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA) (b)(6) (b) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT (b)(6) Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6)

(b)(6) (b)(6) V.,M.D. (b)(6) (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA) <Robert.Johnson@hhs.gov>; Yeskey, (b)((b)(6) Disbrow, Gary (OS/ASPR/BARDA) <Gary.Disbrow@hhs.gov>; Redd, John (OS/ASPR/SPPR) <John.Redd@hhs.gov>; Hassell, (b)((Chris) (OS/ASPR/IO) (b)(6) Hamel, Joseph (OS/ASPR/IO) <Joseph.Hamel@hhs.gov>; Dean, Charity A@CDPH (b)(6) (b)(6) (b)(6) V (b)(6) (b)(6 (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) eric.mcdonald@sdcounty.ca.gov; (b)(6 (b)((b)(6) TARANTINO, (b)(6 A (b)(6) WILKINSON, THOMAS (b)(6) (b)(6) KAUSHIK, SANGEETA (b)(6) (b)(6) (b)(6) (b)(6) (b) Scott (b)(6) Padget, Larry G (b)(6) (b)((b)(6) (b)(6) Stack, (b)(6) J (CHFS DPH) (b)(6)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

CAUTION: This email originated from outside of DHS. DO NOT click links or open attachments unless you recognize and/or trust the sender. Contact your component SOC with questions or concerns.

Excellent editorial from Jeremy Farrar about the near-term funding requirement:

https://protect2.fireeye.com/url?k=ed223e7d-b1761756-ed220f42-0cc47a6d17cc-ccb5142ee4818b7e&u=https://wellcome.ac.uk/news/global-institutions-must-act-urgently-and-decisively-tackle-covid-19

UK Chief Medical Officer Chris Whitty providing forewarning that schools may need to close for as long as two months:

> Chief medical officer: schools could shut for two months in event of pandemic
>
> Chris Whitty has been speaking about measures to reduce risk in the event that coronavirus outbreak reaches pandemic proportions. While noting that such an outcome was just one possibility, he said that there could be a "social cost" if the virus intensifies seeing mass gatherings reduced and schools closed for more than two months.

He said:

One of the things that's really clear with this virus, much more so than flu, is that anything we do we're going to have to do for quite a long period of time, probably more than two months.

"The implications of that are non-trivial, so we need to think that through carefully.

"This is something we face as really quite a serious problem for society potentially if this goes out of control. It may not but if it does globally then we may have to face that."

Whitty also said that the UK would inevitably be affected in the event of a global epidemic.

If this becomes a global epidemic then the UK will get it, and if it does not become a global epidemic the UK is perfectly capable of containing and getting rid of individual cases leading to onward transmission.

"If it is something which is containable, the UK can contain it. If it is not containable, it will be non-containable everywhere and then it is coming our way."

https://www.theguardian.com/world/live/2020/feb/27/coronavirus-news-live-updates#block-5e57f5698f086a28115b3d8d

From: Dr. (b)(6)
Sent: 27 February 2020 17:38
To: (b)(6)
Cc: Tracey (b)(6) (b)(6) (b)(6) Caneva, Duane (b)(6) Martin, (b)(6) J (b)(6) Walters, (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR) <Daniel.Dodgen@hhs.gov>; DeBord, Kristin (OS/ASPR/SPPR) <Kristin.DeBord@hhs.gov>; Phillips, Sally (OS/ASPR/SPPR) <Sally.Phillips@hhs.gov>; (b)(6) J CIV USARMY (USA) (b)(6) WOLFE, HERBERT (b)(6) Eastman, (b)(6) V.,M.D. (b)(6) Johnson, Robert (OS/ASPR/BARDA) <Robert.Johnson@hhs.gov>; Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA) <Gary.Disbrow@hhs.gov>; Redd, John (OS/ASPR/SPPR) <John.Redd@hhs.gov>; Hassell, David (Chris) (OS/ASPR/IO) <David.Hassell@hhs.gov>; Hamel, Joseph (OS/ASPR/IO) <Joseph.Hamel@hhs.gov>; Dean, Charity A@CDPH (b)(6) V

(b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6)
eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)(6) (b)(6) TARANTINO, (b)(6) A
(b)(6) WILKINSON, THOMAS (b)(6)
(b)(6) KAUSHIK, SANGEETA (b)(6) (b)(6) (b)(6)
(b)(6) (b) Scott <Scott.Lee@hhs.gov>; Padget, Larry G (b)(6)
(b)((b)(6) (b)(6) Stack, Steven J (CHFS DPH) (b)(6)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

All schools close in Hong Kong until mid April. They are quarantining a huge group of people entering from China, in separate apartment complex.

Colleagues in UK told me that their schools are on the verge of closing also.

(b)(6)
https://newton.isye.gatech.edu/DrLee/
mobile: (b)(6)

Sent with ProtonMail Secure Email.

------- Original Message -------
On Thursday, February 27, 2020 12:14 PM, (b)(6) (b)(6) (b)(6) wrote:

Japan

All schools told to be closed until April:
https://www.japantimes.co.jp/news/20.../#.XlelKkNryik

Prime Minister Abe at the government's headquarters opened shortly after 6:00 pm on March 27, and temporarily closed all elementary schools, junior high schools, and high schools nationwide from March 2 until spring break.

Prime Minister Abe noted that temporary closure of elementary and junior high schools has been taken in Hokkaido and Ichikawa City, Chiba Prefecture, and said, "Efforts have been made to prevent the spread of infection to children in each region. But the last couple of weeks is a very important time. " He said, "First and foremost, put children's health and safety first, and prepare in advance for the risk of large-scale infection caused by many children and teachers gathering for long hours on a daily basis." He stated that all elementary schools, junior high schools, high schools and special needs schools should be closed temporarily until spring break

Sent from Mail for Windows 10

From: (b)(6) (b)(6)
Sent: Thursday, February 27, 2020 6:17 AM

To: Tracey (b)(6) (b)(6) (b)(6) (b)((b)(6)
Cc: Caneva, Duane; Dr. (b) (b) Martin, (b)(6) J; Walters, (b)(6) (b)(6) (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)((b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)((Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; (b)(6 (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6 (b)(6 TARANTINO, (b)(6 A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) (b) Scott; Padget, Larry G; (b)((b)(6) Stack, (b)(6) J (CHFS DPH)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

About time for Japan (was wondering what they were waiting for to pull this trigger)

Japan's Prime Minister Abe asks schools across the country to be closed from Monday to prevent the spread of coronavirus

Hope we are paying attention in the US and especially California.

Even NK is closing schools

North Korea postpones the opening of schools to prevent an outbreak of coronavirus

Add a few more countries to the list:

- • Denmark
- • Estonia
- • Norway
- • Romania

The outbreaks in Italy and Iran are much larger than many realize by evidence of the numbers of confirmed cases in international travelers from those areas. Cases in travelers from Italy have present in Israel, England, Denmark and Brazil. Need a pretty large outbreak for that to happen—much larger than the numbers reported.

Iran now reporting 245 cases and 26 deaths. Given the confirmed case in Canada of a traveler from Iran and the time from disease onset to death, this is already a well established and large outbreak in Iran.

Italy and Iran are about where Wuhan was 1 month ago. In a couple of weeks Wuhan was overwhelmed.

Here are some snippets re cases of travelers from Italy:

- • Israel confirms 1 new case of coronavirus; an Israeli citizen who recently came back from Italy
- • 2 new cases confirmed in England, UK; one of them came back from Italy and the other from Tenerife, Spain
- • Denmark's TV2 says one of its reporters has tested positive for coronavirus after going on holiday in northern Italy. He developed symptoms on Wednesday morning.
- • The patient is a 61-year-old man in São Paulo. He was in northern Italy from February 9 until February 21.

Sent from Mail for Windows 10

From: (b)(6) (b)(6)
Sent: Thursday, February 27, 2020 5:24 AM
To: Tracey (b)(6) (b)(6) (b)(6) (b)((b)(6)
Cc: Caneva, Duane; Dr. (b) (b) Martin, (b)(6) J; Walters, (b)(6) (b)(6) (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)((b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)((Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; (b)(6 (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6 (b)(6 TARANTINO, (b)(6 A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) (b) Scott; Padget, Larry G; (b)((b)(6) Stack, (b)(6) J (CHFS DPH)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Looked at the age specific data from the cruise ship (focusing on ages <20). Although the numbers are small, the prevalence of disease in school age kids (ages 10-19) is similar to the elderly.

What also caught my eye was the data for young adults (ages 20-49). Since this data is for the entire ship, I suspect that most in this age group are crew. Only 8% of this age group was confirmed to have infection. The total confirmed cases in this analysis was only 619 (we are now up to 744), so perhaps they have since caught up.

Sent from Mail for Windows 10

From: (b)(6) (b)(6)
Sent: Thursday, February 27, 2020 5:00 AM
To: Tracey (b)(6) (b)(6) (b)(6) (b)((b)(6)
Cc: Caneva, Duane; Dr. (b) (b) Martin, (b)(6) J; Walters, (b)(6) (b)(6) (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)((b)(6) (b)(6) Matthew J CIV USARMY (USA); (b) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6 Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)((Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6 V; (b)(6 (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6 (b)(6 TARANTINO, (b)(6 A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) (b), Scott; Padget, Larry G; (b)((b)(6) Stack, Steven J (CHFS DPH)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Details below on case in California. From the cruise ship data we would estimate there are 20-50 cases for each ICY admission (assuming ICU admissions in 2%-5% of cases). That ratio was for an aged population. Suspect that ratio might be higher for a general population. And given the time from disease onset to being on a ventilator for at least a week (since at least Feb 19 when the patient arrived at UC Davis), the outbreak has had a good head start. That would suggest we already have a significant outbreak and are well behind the curve. We are now well past the equivalent 5:45 moment at Mann Gulch. You can't outrun it. They need to be thinking NPIs locally (full TLC including school closure).

[I will send something I was looking at re the cruise ship data and kids.]

https://www.sacbee.com/news/local/article240682311.html

Latest: Coronavirus patient at UC Davis Medical Center since Feb. 19 wasn't tested for days

The Solano County resident who is the nation's first confirmed case of coronavirus from "exposure in the community" has been under the care of UC Davis Medical Center for a week, according to an internal memo obtained Wednesday night by The Sacramento Bee.

Just before 10 p.m., the hospital published the memo that was sent to employees by UC Davis Health leaders earlier in the day and outlines the timeline of the patient's admission and disclosed that several employees who were exposed to the patient self-isolate at home "out of abundance of caution."

The patient, whom the U.S. Centers for Disease Control and Prevention confirmed has tested positive the COVID-19 strain, was moved to the Sacramento teaching hospital on Feb. 19, according to the memo sent to staffers by David Lubarsky, the head of the hospital and UC Davis Health's vice chancellor of human health services, and Brad Simmons, the health system's interim CEO.

The patient was transferred to the facility from another hospital, where a medical team had already put the patient on a ventilator.

"The individual is a resident of Solano County and is receiving medical care in Sacramento County. The individual had no known exposure to the virus through travel or close contact with a known infected individual," California Department of Public Health officials said in a news release.

Because physicians at the first hospital suspected the patient had a virus, they issued an order that health care workers should wear personal protective gear when with the patient to guard against exposure to droplets, said the memo, which was first reported by the Davis Enterprise newspaper.

The UCD medical team used the proper infection protocols out of concern that the individual might have coronavirus, according to the memo, and upon the patient's admission, UCD physicians requested that public health officials perform a test to determine whether the person had COVID-19.

"We requested COVID-19 testing by the CDC, since neither Sacramento County nor CDPH is doing testing for coronavirus at this time," the memo says. "Since the patient did not fit the existing CDC criteria for COVID-19, a test was not immediately administered. UC Davis Health does not control the testing process."

On Sunday, the CDC ordered a coronavirus test on the patient, and UC Davis Health officials discovered Wednesday that the patient tested positive for the deadly respiratory illness that causes coughing, fever and shortness of breath. That prompted hospital officials to tell "a small number" of hospital workers to stay home and monitor themselves for possible infection.

"Just as when a health care worker has a small chance of exposure to other illnesses, such as TB or pertussis, we are following standard CDC protocols for determination of exposure and surveillance," the memo said. "So, out of an abundance of caution, in order to assure the health and safety of our employees, we are asking a small number of employees to stay home and monitor their temperature."

"We are handling this in the same way we manage other diseases that require airborne precautions and monitoring," the memo said, adding hospital officials are "in constant communication with the state health department and the CDC and Sacramento County Public Health about the optimal management of this patient and possible employee exposures."

UCD officials did not respond to The Bee's request for comment.

The memo ended: "We are dedicated to providing the best care possible for this patient and continuing to protect the health of our employees who care for them."

Sent from Mail for Windows 10

From: (b)(6) (b)(6)
Sent: Wednesday, February 26, 2020 9:45 PM
To: Tracey (b)(6) (b)(6) (b)(6) (b)((b)(6)
Cc: Caneva, Duane; Dr. (b) (b) Martin, (b)(6) J; Walters, (b)(6) (b)(6) (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)((b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6 Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)((Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6 V; (b)(6 (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6 (b)(6 TARANTINO, (b)(6 A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) (b), Scott; Padget, Larry G; (b)((b)(6) Stack, Steven J (CHFS DPH)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Some updates.

Singapore and Hong Kong continue to hold the line.

Singapore (+2 case) 93 cases/0 deaths /7 in ICU (still 4 kids—none currently hospitalized/2 were asymptomatic

Hong Kong (+6) 91 cases/2 deaths/ 4 critical; 2 serious (1 kid age 16)

Japan 189 cases/3 deaths/13 serious

Explosive growth in South Korea, Italy, and Iran

South Korea 1,596 cases/13 deaths/13 serious; 5 critical

Italy 453 cases/12 deaths

Iran 139 cases/19 deaths

Sent from Mail for Windows 10

From: (b)(6) (b)(6)
Sent: Wednesday, February 26, 2020 9:29 PM
To: Tracey (b)(6) (b)(6) (b)(6) (b)((b)(6)
Cc: Caneva, Duane; Dr. (b) (b) Martin, (b)(6) J; Walters, (b)(6) (b)(6) (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)((b)(6) (b)(6) Matthew J CIV USARMY (USA); (b) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6 Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)((Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6 V; (b)(6 (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6 (b)(6 TARANTINO, (b)(6 A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) (b) Scott; Padget, Larry G; (b)((b)(6) Stack, Steven J (CHFS DPH)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Update Hubei & Wuhan Hospitalizations per 100,000. Hubei curve is plateauing.

	Hubei 2019-nCoV Confirmed Hospital Data								
Date	Total Current Inpatients	Mild Disease	Severely Ill	Critically Ill	Cum Discharges	Cum Deaths	Cum Inpatients	Hubei Cum cases	Wul Cas
1/14/20	6		6				6	41	4
1/15/20	5		5			2	7	41	4
1/16/20	5		5			2	7	45	4
1/17/20	8		8			2	10	62	6
1/18/20	136	100	33	3		3	139	121	12
1/19/20	170	126	35	9		4	174	198	19
1/20/20	239	176	51	12		7	246	270	25
1/21/20						15		375	32
1/22/20	399	304	71	24		17	416	444	39
1/23/20	494	365	106	23	31	24	549	549	49
1/24/20	658	472	129	57	32	39	729	729	57
1/25/20	915		221		85	52	1,052	1,052	61
1/26/20	1,645	1,013	563	69	44	76	1,423	1,423	69
1/27/20	2,567	1,877	563	127	47	100	2,714	2,714	1,5
1/28/20	3,349	2,450	671	228	80	125	3,554	3,554	1,9
1/29/20	4,334	3,346	711	277	90	162	4,586	4,586	2,2
1/30/20	5,486	4,392	804	290	116	204	5,806	5,806	2,6
1/31/20	6,738	5,444	956	338	166	249	7,153	7,153	3,2
2/1/20	8,565	7,003	1,118	444	215	294	9,074	9,074	4,1
2/2/20	9,618	7,917	1,223	478	295	350	10,263	11,177	5,1
2/3/20	10,990	8,857	1,557	576	396	414	11,800	13,522	6,3
2/4/20	12,627	10,107	1,809	711	520	479	13,626	16,678	8,3
2/5/20	14,314	11,230	2,328	756	633	549	15,496	19,665	10,1
2/6/20	15,804	11,802	3,161	841	817	618	17,239	22,112	11,6
2/7/20	19,835	14,640	4,188	1,007	1,113	699	21,647	24,953	13,6
2/8/20	20,993	15,746	4,093	1,154	1,439	780	23,212	27,100	14,9
2/9/20	22,160	16,655	4,269	1,236	1,795	871	24,826	29,631	16,9
2/10/20	25,087	18,743	5,046	1,298	2,222	974	28,283	31,728	18,4
2/11/20	26,121	18,880	5,724	1,517	2,639	1,068	29,828	31,728	18,4
2/12/20	33,693	26,609	5,647	1,437	3,441	1,310	38,444	48,206	32,9
2/13/20	36,719	27,081	7,953	1,685	4,131	1,426	42,276	51,986	35,9
2/14/20	38,107	27,955	8,276	1,876	4,774	1,457	44,338	54,406	37,9
2/15/20	39,447	29,051	8,439	1,957	5,623	1,596	46,666	56,249	39,4

2/16/20	40,814	31,017	8,024	1,773	6,639	1,696	49,149	58,182	41,1
2/17/20	41,957	30,987	9,117	1,853	7,862	1,789	51,608	59,989	42,7
2/18/20	43,471	32,225	9,289	1,957	9,128	1,921	54,520	61,682	44,4
2/19/20	43,745	32,567	9,128	2,050	10,337	2,029	56,111	62,013	45,0
2/20/20	42,056	31,059	8,979	2,018	11,788	2,144	55,988	62,422	45,3
2/21/20	41,036	30,144	8,400	2,492	13,557	2,250	56,843	63,454	45,6
2/22/20	39,073	29,643	7,776	1,654	16,738	2,399	58,210	64,287	46,6
2/23/20	37,896	29,221	7,090	1,585	18,854	2,563	59,313	64,786	47,0
2/24/20	36,242	27,916	6,840	1,486	20,912	2,615	59,769	65,187	47,4
2/25/20	34,978	26,994	6,581	1,403	23,200	2,641	60,819	65,596	47,8

Sent from Mail for Windows 10

From: Tracey (b)(6)
Sent: Wednesday, February 26, 2020 8:35 PM
To: (b)(6) (b)(6) (b)(6) (b)(6)
Cc: Caneva, Duane; (b)(6) (b)(6) Dr. (b) (b) Martin, (b)(6) J; Walters, (b)(6) (b)(6), (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)((b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6 Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)((Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6 V; (b)(6 (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6 (b)(6 TARANTINO, (b)(6 A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) (b), Scott; Padget, Larry G; (b)((b)(6) Stack, Steven J (CHFS DPH)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

FEB 26 2020

QIAGEN announces worldwide shipments of QIAstat-Dx test kits for SARS-CoV-2

• QIAstat-Dx Respiratory 2019-nCoV Panel rapidly differentiates novel coronavirus from 21 other pathogens implicated in serious respiratory syndromes

• Panel being tested at four hospitals in China after initial evaluation on clinical samples by a leading Paris hospital

• Adds to QIAGEN's portfolio of molecular testing solutions to aid in global public health emergency

Hilden, Germany, and Germantown, Maryland, February 26, 2020 – QIAGEN (NYSE: QGEN; Frankfurt Prime Standard: QIA) today announced it has shipped its newly developed QIAstat-Dx Respiratory Panel 2019-nCoV test kit to four hospitals in China for evaluation. The new kit detects the novel coronavirus SARS-CoV-2 and adds rapid Sample to Insight syndromic testing to QIAGEN's portfolio of molecular testing solutions in the public health emergency. QIAGEN is also in the process of shipping QIAstat-Dx testing kits to public health institutions in other regions, including Europe, South-East Asia, and the Middle East.

Since January QIAGEN has been providing instruments and consumables to support detection of the virus in China and other markets. Official protocols for SARS-CoV-2 detection include QIAGEN extraction kits, reagents and instruments for real-time polymerase chain reaction (RT-PCR) workflows. Customers also are deploying QIAsymphony modular instruments and NeuMoDx integrated PCR systems to automate higher-throughput processing of their laboratory-developed tests for SARS-CoV-2.

"Our dedicated task force has moved very fast to develop and make available the QIAstat-Dx respiratory panel with SARS-CoV-2 detection. We are partnering closely with authorities and customers around the world to bring rapid, accurate diagnosis to the fight against this deadly infectious disease," said Thierry Bernard, Interim CEO of QIAGEN and Senior Vice President, Head of the Molecular Diagnostics Business Area. "As we have in past health crises such as SARS and the swine flu, QIAGEN is working hard to deliver better, faster testing solutions for hospitals and public health institutions to aid in the effort to monitor and bring the outbreak under control. Our employees' extraordinary response embodies QIAGEN's core mission to make improvements in life possible."

The QIAstat-Dx system was introduced in Europe in 2018 as a CE-product and cleared by the Food and Drug Administration (FDA) in the United States in mid-2019. It enables fast, cost-effective and easy-to-use syndromic testing with novel Sample to Insight workflows. The system streamlines molecular testing from end to end. A technician simply loads a clinical sample (such as a swab) into a single-use QIAstat-Dx cartridge and places it in the analyzer. QIAGEN chemistries for sample processing

and analysis are built in, and the QIAstat-Dx instrument delivers results in about one hour.

New QIAstat-Dx testing solution for SARS-CoV-2

The QIAstat-Dx Respiratory 2019-nCoV Panel is a new version of the existing QIAstat-Dx Respiratory Panel for differential analysis of 21 viral and bacterial pathogens in respiratory syndromes. Once the SARS-CoV-2 genome was sequenced in January, QIAGEN developed two highly sensitive assays to detect SARS-CoV-2 targeting Orb1ab and the E gene. The addition of these targets provides parallel tools for combined detection of the novel coronavirus with increased sensitivity.

The expanded QIAstat-Dx panel is currently being evaluated at the Bichat-Claude Bernard Hospital in Paris. "After more than a year of experience using the existing QIAstat-Dx Respiratory Panel to evaluate patients coming into our Emergency Department with respiratory syndromes, we are evaluating the new panel with SARS-CoV-2 against RT-PCR testing using WHO recommended protocols. The QIAstat-Dx solution can provide results in about one hour, with high sensitivity and specificity and minimal hands-on time for hospital or laboratory personnel," said Dr. Benoit Visseaux, Associate Professor of Virology at Bichat-Claude Bernard Hospital in Paris.

Amid the rapidly evolving response to the coronavirus outbreak, the regulatory status of the QIAstat-Dx Respiratory Panel will vary by location. QIAGEN will apply for emergency authorization for marketing of the new panel from the U.S., the Korean KCDC/MFDS, and FDA and China's National Medical Products Administration (NMPA); the panel will be available with CE-IVD marking in Europe and other markets.

Multiple other QIAGEN testing solutions for SARS-CoV-2

QIAGEN has moved quickly on several fronts to provide molecular testing solutions to researchers working to counter the international threat from the SARS-CoV-2 virus. Building on its strong position and experience in molecular testing for infectious diseases, QIAGEN is providing a variety of solutions:

- Enabling laboratory-developed tests (LDTs) – extraction kits, PCR enzymes and instruments, supporting in-house testing by laboratories and public health institutions. CDC guidelines for RT-PCR testing list QIAGEN's EZ1 DSP Virus kits, which run on EZ1 Advanced workstations, and QIAamp DSP Viral RNA Mini kits, which can be automated on QIAcube instruments. Chinese authorities and the Berlin Charitè protocol also include QIAGEN consumables.

- Mid- and high-throughput automation – QIAsymphony modular systems for sample preparation and PCR analysis, as well as NeuMoDx 96 and 288 systems for fully integrated PCR analysis, enabling customers to implement higher-throughput solutions for laboratory-developed RT-PCR tests. QIAGEN recently started placing NeuMoDx systems in China, for use in with LDTs in SARS-CoV-2 testing.

- Additional real-time PCR tests – two new RT-PCR tests for detection of SARS-CoV-2 have been developed at QIAGEN sites in China and the United States and will be available for Research Use Only. The company is investigating potential emergency use options for the automated PCR-based test solutions.

Further information can be also found here

About QIAGEN

QIAGEN N.V., a Netherlands-based holding company, is the leading global provider of Sample to Insight solutions that enable customers to gain valuable molecular insights from samples containing the building blocks of life. Our sample technologies isolate and process DNA, RNA and proteins from blood, tissue and other materials. Assay technologies make these biomolecules visible and ready for analysis. Bioinformatics software and knowledge bases interpret data to report relevant, actionable insights. Automation solutions tie these together in seamless and cost-effective workflows. QIAGEN provides solutions to more than 500,000 customers around the world in Molecular Diagnostics (human healthcare) and Life Sciences (academia, pharma R&D and industrial applications, primarily forensics). As of December 31, 2019, QIAGEN employed approximately 5,100 people in over 35 locations worldwide. Further information can be found athttps://protect2.fireeye.com/url?k=e354d5e7-bf00fccc-e354e4d8-0cc47a6d17cc-363bf1fc5b259e02&u=http://www.qiagen.com/.

From: (b)(6) (b)(6) (b)(6)
Sent: Wednesday, February 26, 2020 3:34 PM
To: (b)(6) (b)(6) (b)(6)
Cc: Caneva, Duane (b)(6) (b)(6) (b)(6) (b)(6) Dr. (b)(6) (b)(6) Martin, (b)(6) J (b)(6) Walters, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR) <Daniel.Dodgen@hhs.gov>; DeBord, Kristin (OS/ASPR/SPPR) <Kristin.DeBord@hhs.gov>; Phillips, Sally (OS/ASPR/SPPR) <Sally.Phillips@hhs.gov>; (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT (b)(6) Eastman, (b)(6)

(b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D. (b)(6) (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA) <Robert.Johnson@hhs.gov>; Yeskey, (b)((b)(6) Disbrow, Gary (OS/ASPR/BARDA) <Gary.Disbrow@hhs.gov>; Redd, John (OS/ASPR/SPPR) <John.Redd@hhs.gov>; Hassell, David (Chris) (OS/ASPR/IO) (b)(6) Hamel, Joseph (OS/ASPR/IO) <Joseph.Hamel@hhs.gov>; Dean, Charity A@CDPH (b)(6) (b)(6) (b)(6) V (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)((b)(6) TARANTINO, DAVID A (b)(6) WILKINSON, THOMAS (b)(6) (b)(6) KAUSHIK, SANGEETA (b)(6) (b)(6) (b)(6) (b)(6) Tracey (b)(6) (b)(6) (b)(Scott (b)(6) Padget, Larry G (b)(6) (b)((b)(6) (b)(6) Stack, Steven J (CHFS DPH) (b)(6)
Subject: Re: Red Dawn Breaking Bad, Start Feb 24

Bob K live in the White House Briefing Room now - live feed on WaPo site -

Sent from my iPhone

On 26 Feb 2020, at 23:08, (b)((b)(6) (b)(6) wrote:

Why are CDC numbers (on their website) so low? E.g., 14,000 deaths.

(b)(6)

On Feb 26, 2020, at 5:56 PM, Caneva, Duane (b)(6) wrote:

Master Question List—things to think about, updated.

Also,

https://www.washingtonpost.com/health/2020/02/25/cdc-coronavirus-test/

From: (b)(6) (b)(6) <cmecher@charter.net>
Sent: Wednesday, February 26, 2020 3:14 PM
To: Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Updated cruise ship data including data we can find by country

Country	Passengers/Crew	Cases Hospitalized in Japan	Cases Confirmed upon Repatriation	Total Confirmed Cases	ICU Admissions	Deaths	% Infecte
US	434	44	42	86			20%
Hong Kong	364	55	4	59			16%
Canada	256	47		47			18%
Australia	241	47	7	54			22%
UK	78	6	4	10			13%
Italy	35						
South Korea	14						
Israel	11	3	2	5			45%

Japan						4	
Subtotal	1,433	202	59	261			
Total	**3,711**			**744**			**20%**

Date	Event	Cumulative Number of Confirmed Cases	Cumulative Number of Deaths	Note
20-Jan	Cruise ship departs from Yokohama Japan			
25-Jan	80 year old passenger disembarks in Hong Kong			
1-Feb	80 year old passenger confirmed to have COVID-19			
	When results known, certificate of landing canceled and ship under quarantine. Tests for the virus would be administered to three groups: those with symptoms, those who got off in Hong Kong, and those who had close contact with the infected passenger.			
3-Feb	Ship arrives in port of Yokohama Japan			
5-Feb	10 passengers and crew confirmed +	10		
6-Feb	31 more passengers and crew confirmed +	41		
7-Feb	30 more passenger and crew confirmed +	61		
8-Feb	9 more passenger and crew confirmed +	70		
10-Feb	66 more passenger and crew confirmed +	136		439 tes
11-Feb	39 more passenger and crew confirmed +	175		492 tes
12-Feb	28 more passenger and crew confirmed +	203		4 in IC
13-Feb	15 more passenger and crew confirmed +	218		713 tes
14-Feb	67 more passenger and crew confirmed +	285		927 tes
15-Feb	70 more passenger and crew confirmed +	355		1,219 teste 73 asympt
16-Feb	Evaluation Only. Created with Aspose.HTML. Copyright 2013-2020 Aspose Pty Ltd.sp; (14 of the evacuees found to be +) 61 Americans remained on board 44 Americans remained hospitalized in Japan	369		

17-Feb	85 more passenger and crew confirmed +	454		1,723 tested 19 seriou
18-Feb	167 more passenger and crew confirmed +	621		3,011 te
19-Feb	2 deaths	621	2	
20-Feb	13 more passenger and crew confirmed +	634	2	3,066 tested 28 seriously 322 asymp
23-Feb	Death reported in Japan		3	
24-Feb	Japan updates total to 691; US reports 36 in US	691	3	
25-Feb	Death reported in Japan; US reports 40 in US; UK 4; Australia 7; Hong Kong 4; Israel 2; Total 744; plus 4 not on ship	744	4	3,894 tested 35 seriously 380 asymp
26-Feb	US reports total of 42 cases in US; Japan announces that 45 of 813 former passengers have symptoms and will need to be tested	746	4	

Sent from Mail for Windows 10

From: (b)(6) (b)(6)
Sent: Wednesday, February 26, 2020 1:46 PM
To: (b)(6) (b)(6) Caneva, Duane; Dr. (b) (b) Martin, (b)(6) J; Walters, (b)(6) (b)(6), (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)((b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)((Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; (b)(6 (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6 (b)(6 TARANTINO, (b)(6 A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) (b) , Scott; Padget, Larry G; (b)((b)(6) Stack, (b)(6) J (CHFS DPH)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Number of cases from Diamond Princess cruise ship rises to 42 in US (plus 44 in Japan), for total of 86.

<image001.png>

Sent from Mail for Windows 10

From: (b)(6) (b)(6)
Sent: Wednesday, February 26, 2020 12:34 PM
To: (b)(6) (b)(6) Caneva, Duane; Dr. (b) (b) Martin, (b)(6) J; Walters, (b)(6) (b)(6), (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)((b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, David (Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; (b)(6) (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)(6) TARANTINO, (b)(6) A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) Lee, Scott; Padget, Larry G; (b)((b)(6) Stack, (b)(6) J (CHFS DPH)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

I found a treasure trove of information and analysis of the Diamond Princess cruise ship outbreak. Japan's National Institute of Infectious Diseases published a Field Briefing on Feb 21, 2020 https://www.niid.go.jp/niid/en/2019-ncov-e.html

I took the material and built a slide deck.

Take a look.

Sent from Mail for Windows 10

From: (b)(6) (b)(6)
Sent: Wednesday, February 26, 2020 9:08 AM
To: (b)(6) (b)(6) Caneva, Duane; Dr. (b) (b) Martin, (b)(6) J; Walters, (b)(6) (b)(6) (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)((b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)((Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; (b)(6) (b)(6)

(b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)(6) TARANTINO, DAVID A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) Lee, Scott; Padget, Larry G; (b)((b)(6) Stack, Steven J (CHFS DPH)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Things keep getting more complicated wrt the cruise ship outbreak. Another 45 patients (out of 813 passengers who let off the ship) have developed symptoms and will require testing.

TOKYO: Dozens of passengers allowed off a coronavirus-stricken ship have developed symptoms including fever and will be asked to take tests for the virus, Japan's health minister said Wednesday.

The news came as another death linked to the virus in Japan was reported and the government urged organisers of major events in the next fortnight to consider cancelling or downsizing them to help curb the spread of infections.

The government has contacted 813 former passengers of the Diamond Princess cruise ship and found "45 people had certain symptoms", Health Minister Katsunobu Kato told parliament.

"We asked all of them (who have symptoms) to see a doctor and to take tests."

Around 970 people were allowed off the boat last week after testing negative for the virus, but several have subsequently been diagnosed with the illness.

Japan has come under increasing pressure over its handling of the crisis on the vessel.

Those allowed off the ship after a 14-day quarantine were asked to stay inside, but no formal measures restricting their movement were imposed.

Opposition lawmakers have blamed the government for failing to implement a fresh (b)(6) quarantine after the passengers left the cruise ship – as was required by countries that repatriated citizens from the boat.

Infections have also continued to rise inside Japan, and Prime Minister Shinzo Abe on Wednesday said hosting large events should be reconsidered.

"In light of the significant infection risks, we will ask that national sporting or cultural events that will attract large crowds be either cancelled, postponed or downsized for the next two weeks," Abe told a cabinet task force meeting on the outbreak.

Concerts cancelled

After the announcement, Nippon Professional Baseball Organization said its unofficial spring games through March 15 would be held in empty stadiums, before the official season opens on March 20.

Some top Japanese musicians, including all-male group Exile and female trio Perfume cancelled concerts, while Tokyo Girls Collection fashion show on Saturday will be held with no audience, according to their organisers.

The virus has also forced professional football, rugby, golf, tennis and other sports to reschedule games or to hold their events with no fans in attendance.

The government has also asked state-operated museums and theatres to consider closing or cancelling shows.

The government has repeatedly said that the coming weeks will be critical in limiting the spread of the virus in Japan.

But its measures have been largely advisory, including recommending that people work from home or commute off-peak.

The recommendations come as the local government in northern Hokkaido announced in its latest update on the virus the death of a local resident, whose name, gender and age were not revealed.

The governor of Hokkaido, where at least 38 people have been diagnosed, said he was requesting local municipalities to close public schools for one week from Thursday.

In Tokyo meanwhile, the regional education board said public high schools may start classes late to spare students travelling on packed commuter trains.

Japan has seen at least 165 infections separate from the outbreak on the cruise ship.

The outbreak has raised fears that the Olympic Games to be hosted in Tokyo this summer could be cancelled, a possibility government officials and organisers have rejected.

“We have not thought about it. We have not heard about it. We have made inquiries, and we were told there is no such plan,” Tokyo 2020 CEO Toshiro Muto told reporters.

“Our basic thinking is to conduct the Olympics and Paralympics as planned. That’s our assumption.”

Sent from Mail for Windows 10

From: (b)(6) (b)(6)
Sent: Wednesday, February 26, 2020 9:01 AM

To: (b)(6) (b)(6) Caneva, Duane; Dr. (b) (b) Martin, (b)(6) J; Walters, (b)(6) (b)(6), (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)((b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)((Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; (b)(6 (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6 (b)(6 TARANTINO, (b)(6 A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) Lee, Scott; Padget, Larry G; (b)((b)(6) Stack, (b)(6) J (CHFS DPH)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

https://www.theguardian.com/world/2020/feb/26/wuhan-nurses-plea-international-medics-help-fight-coronavirus

Moving description from the front lines of conditions in Wuhan . . . And a picture of what we will likely face soon . . .

From: (b)(6) (b)(6) (b)(6)
Sent: 26 February 2020 04:11
To: Caneva, Duane (b)(6) (b)(6) (b)(6) (b)(6) Dr. (b) (b) (b)(6) Martin, (b)(6) J (b)(6) Walters, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR) <Daniel.Dodgen@hhs.gov>; DeBord, Kristin (OS/ASPR/SPPR) <Kristin.DeBord@hhs.gov>; Phillips, Sally (OS/ASPR/SPPR) <Sally.Phillips@hhs.gov>; (b)((b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA) (b)(6) (b) (b)(6) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT (b)(6) Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D. (b)(6) (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA) <Robert.Johnson@hhs.gov>; Yeskey, (b)((b)(6) Disbrow, Gary (OS/ASPR/BARDA) <Gary.Disbrow@hhs.gov>; Redd, John (OS/ASPR/SPPR) <John.Redd@hhs.gov>; Hassell, David (Chris) (OS/ASPR/IO) <David.Hassell@hhs.gov>; Hamel, Joseph (OS/ASPR/IO) <Joseph.Hamel@hhs.gov>; Dean, Charity A@CDPH (b)(6) (b)(6) (b)(6) V (b)(6) (b)(6 (b)(6) (b)(6) (b)(6) (b) (b)(6) eric.mcdonald@sdcounty.ca.gov; (b)(6 (b)((b)(6) TARANTINO, (b)(6 A (b)(6) WILKINSON, THOMAS (b)(6) (b)(6) KAUSHIK, SANGEETA (b)(6) (b)(6) (b)(6) (b)(6) Tracey (b)(6) (b)(6) (b) Scott (b)(6) Padget, Larry G (b)(6) (b)((b)(6) (b)(6) Stack, (b)(6) J (CHFS DPH) (b)(6)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Watching ILI in a few key areas--NYC, Chicago, CA, and TX. Week 7 data (week ending Feb 15). Flu is now trending down. This data is a little old (now 10 days old). Week 8 data coming soon.

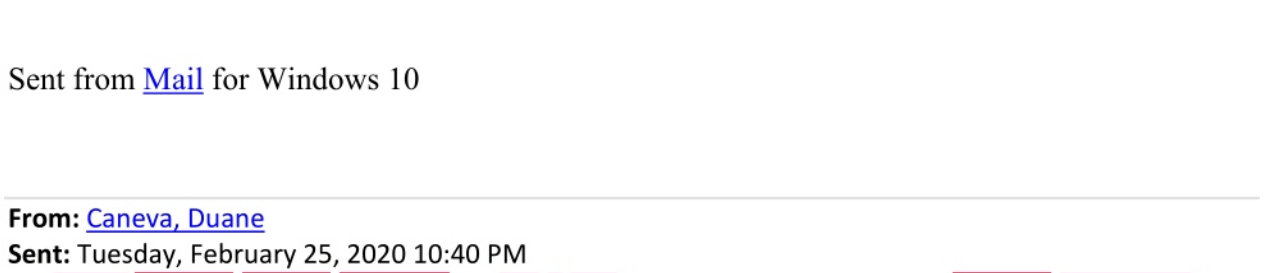
Chicago



NYC



CA



TX



Sent from Mail for Windows 10

From: Caneva, Duane
Sent: Tuesday, February 25, 2020 10:40 PM
To: (b)(6) (b)(6) (b)(6) (b)(6) Dr. (b) (b) Martin, Gregory J; Walters, (b)(6) (b)(6), (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)((b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)((Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; (b)(6) (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)(6) TARANTINO, (b)(6) A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) (b)(6) (b)(6) Lee, Scott; Padget, Larry G; (b)((b)(6) Stack, Steven J (CHFS DPH)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

ASTHO sponsored meeting hosted by White House this evening. Good discussion and input from many state and local Public Health Official partners. We are all in this together, and

preparedness and response slowly transitions to community mitigation efforts and the frontline boots on the ground.

Still only 14 cases *detected*.

Red Dawn Breaking…

From: (b)(6) (b)(6) (b)(6)
Sent:
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

https://www.sciencemag.org/news/2020/02/coronavirus-infections-keep-mounting-after-cruise-ship-fiasco-japan

TOKYO—All but a handful of the passengers of the disease-stricken Diamond Princess cruise ship berthed in Yokohama have disembarked. But for Japan, the saga is far from over. Much of the crew remains on board, enduring another 14 days of quarantine—although this time under conditions that Japanese officials hope will prevent any additional infections.

But there has been another worrisome development: As of today, eight public servants who worked on the ship to support the quarantine have tested positive for COVID-19, and more may follow. Most of the roughly 90 health ministry employees who visited the ship during the first 2-week quarantine that ended on 19 February initially returned to their normal work duties, but in light of the infections, the health ministry yesterday revised its policy and now those potentially exposed to the virus on the Diamond Princess are self-quarantining at home for 14 days, according to a ministry official who asked not to be identified.

Sent from Mail for Windows 10

From: (b)(6) (b)(6)
Sent: Tuesday, February 25, 2020 7:41 PM
To: (b)(6) (b)(6) Dr. (b) (b)
Cc: Martin, (b)(6) J; Walters, (b)(6) (b)(6) Caneva, Duane; (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)((b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6 Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)((Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6 V; (b)(6 (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6 (b)(6 TARANTINO, (b)(6 A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) (b), Scott; Padget, Larry G; (b)((b)(6)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Some updates. Will send the Hubei/Wuhan update later.

Singapore and Hong Kong continue to hold the line. I attached the case studies we did to monitor when they pulled the trigger in NPIs—lessons for us and for others.

Singapore (+1 case) 91 cases/0 deaths /7 in ICU (still 4 kids—none currently hospitalized/2 were asymptomatic

Hong Kong (+4) 85 cases/2 deaths/ 4 critical; 2 serious (still no kids reported)

Japan 170 cases/1 death/7 serious

Explosive growth in South Korea, Italy, and Iran

South Korea 977 cases/11 deaths/6 critical

99 out of 102 people in the psychiatric department of a hospital in South Korea tested positive for coronavirus infection.

https://www.bloomberg.com/news/articles/2020-02-22/nearly-all-patients-in-south-korean-psychiatric-ward-have-virus

Yesterday, the Korean government raised the alert level from Orange(Level 3) to Red (Level 4) in order to prepare for a possible nation-wide transmission. It was a proactive decision taking into consideration the pattern and speed of transmission.

Italy 322 cases/11 deaths/114 in hospital; 35 in ICU

Public events in Veneto and Lombardy are banned - All schools in Lombardy and Veneto will be closed - Venice Carnival will be shut down, shows canceled at Milan La Scala

Iran 95 cases/16 deaths

Bahrain 23 cases/0 deaths

Bahrain closes all kindergartens, schools, universities, and training centers in the country for at least 2 weeks to prevent the spread of coronavirus

New countries today

Sent from Mail for Windows 10

From: (b)(6) (b)(6)
Sent: Tuesday, February 25, 2020 5:47 PM
To: Dr. (b) (b)
Cc: (b)(6) (b)(6) Martin, (b)(6) J; Walters, (b)(6) (b)(6) Caneva, Duane; (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)((b)(6) (b)(6) Matthew J CIV USARMY (USA); (b) (b)(6) (b)(6), (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6 Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)((Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; (b)(6 (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6 (b)(6 TARANTINO, (b)(6 A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) (b), Scott; Padget, Larry G; (b)((b)(6)
Subject: Re: Red Dawn Breaking Bad, Start Feb 24

Big news today from China is the continued drop in new cases (down to just over 500 today) and the number of provinces (>20) with no new cases at all. It remains to be seen if China can hold the line but its interventions ARE working to reduce transmission dramatically and (b)(6) is right that Singapore and Hong Kong are demonstrating the value of an early, rapid, aggressive response. Whether the rest of the world, and the U.S., can mount a similarly effective response I do not know, but China has shown what can be done with NPIs.

+ (b)(6 (b)(6) from WEF. Welcome (b)(6 -

Sent from my iPhone

On 25 Feb 2020, at 18:14, Dr. (b) (b) (b)(6) wrote:

It is unclear if S. Korea will be able to setup so many temp. beds as China. They're very slow in picking up social distancing and social awareness, proper NPI. Iran is way behind in terms of medical care. I fear they will have very high mortality.

(b)(6)

https://newton.isye.gatech.edu/DrLee/

mobile: (b)(6)

Sent with ProtonMail Secure Email.

------- Original Message -------

On Tuesday, February 25, 2020 11:34 AM, (b)(6) (b)(6) (b)(6) wrote:

South Korea now has 977 cases and 10 deaths. They are about where Wuhan was on January 25th (so about 1 month behind). Wuhan was overwhelmed less than 2 weeks later. I would expect the same for South Korea with the epicenter being in Seoul.

I think Iran is about at the same point (maybe even a little ahead) of South Korea. Tehran is another very large city that will likely becomes its epicenter.

I see a few hopeful signs. Singapore and Hong Kong have done a great job thus far and have implemented NPIs very early. Both have great surveillance. They are holding the line. They are also small and islands. Japan on the other hand is struggling and hasn't been as aggressive as Singapore and Hong Kong.

The other thing that gives me hope is what I see in Hubei and Wuhan. I realize the data is a little sketchy because China has gone back and forth with the definition of cases, but I tried to smooth that over by looking at cumulative hospitalization rates per 100,000 (like we do for flu). Hubei (and Wuhan is a city within Hubei) reports each day the current number of people in the hospital (# currently in severe condition, # in critical condition), cumulative number of hospital

discharges, cumulative deaths, and cumulative cases. From this we can estimate cumulative hospitalizations and then rates. 92% of the cases have been hospitalized (up thru Feb 2nd 100% of the cases they reported were hospitalized). Knowing the number of cases in Wuhan, we have been estimating the number hospitalized assuming a similar % of the cases requiring hospitalization rate for Wuhan (that 92% of the cases are being hospitalized—that number is adjusted each day based on current data). So we really can't back out the Wuhan numbers from the Hubei numbers. The best we can do is compare Hubei totals (including Wuhan) with an estimate of Wuhan. This data is good enough to show that the Chinese appear to be slowing transmission outside of Wuhan (They were late to implement NPIs in Wuhan but were able to implement NPIs earlier in the epidemic outside of Wuhan because the outbreak had about a 2 week head-start in Wuhan).

<E0B38B2300CE43F09DC37BFDDDB81F3C.png>

We need to emulate the blue curve. If I could subtract Wuhan, this curve would be significantly lower.

Remember the goals of NPIs.

<CB432B1C32644B219725D229547BEDDC.png>

Sent from Mail for Windows 10

From: (b)(6) (b)(6)

Sent: Tuesday, February 25, 2020 10:30 AM

To: (b)(6) (b)(6) Dr. (b) (6) (b)

Cc: Martin, Gregory J; Walters, (b)(6) (b)(6) Caneva, Duane; rjglassjr@gmail.com; Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b)(6) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) , (b)(6) (b)(6) (b)(6) V.,M.D.; jwleduc@UTMB.EDU; rbaric@email.unc.edu; Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, David (Chris) (OS/ASPR/IO); Hamel, Joseph

(OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; (b)(6) (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)(6) TARANTINO, (b)(6) A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) Lee, Scott; Padget, Larry G

Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Death #4 from Diamond Princess.

https://twitter.com/BNODesk/status/1232124151789477889

From: (b)(6) (b)(6) (b)(6)

Sent: 25 February 2020 14:10

To: Dr. (b)((b)(6)

Cc: Martin, (b)(6) J (b)(6) Walters, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Caneva, Duane (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR) <Daniel.Dodgen@HHS.GOV>; DeBord, Kristin (OS/ASPR/SPPR) <Kristin.DeBord@hhs.gov>; Phillips, Sally (OS/ASPR/SPPR) <Sally.Phillips@hhs.gov>; (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA) (b)(6) (b)((b)(6) (b)(6) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT (b)(6) Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D. (b)(6) (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA) <Robert.Johnson@hhs.gov>; Yeskey, (b)(6) (b)(6) Disbrow, Gary (OS/ASPR/BARDA) <Gary.Disbrow@hhs.gov>; Redd, John (OS/ASPR/SPPR) <John.Redd@hhs.gov>; Hassell, (b)(6) (Chris) (OS/ASPR/IO) (b)(6) Hamel, Joseph (OS/ASPR/IO) <Joseph.Hamel@hhs.gov>; Dean, Charity A@CDPH (b)(6) (b)(6) (b)(6) V (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(<(b)(6) eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)(6) (b)(6) TARANTINO, (b)(6) A (b)(6) WILKINSON, THOMAS (b)(6) (b)(6) KAUSHIK, SANGEETA (b)(6) (b)(6) (b)(6) (b)(6) Tracey (b)(6) (b)(6) (b)(Scott (b)(6) Padget, Larry G (b)(6)

Subject: RE: Red Dawn Breaking Bad, Start Feb 24

We should plan assuming we won't have enough PPE—so need to change the battlefield and how we envision or even define the front lines. The frontlines for mild illness need to pivot from our usual way of dealing with ILI in our clinics and ERs to non-face-to-face alternatives for the delivery of care (by phone/telehealth/home care). It means we need to shrink the problem and think of COVID patients in two groups: (1) those with mild enough disease that they can be managed thru self-care in the home; and (2) those will more severe disease that cannot be managed at home and likely require hospitalization. The first group needs to be managed by phone/telehealth with "prescriptions" for home isolation qD X 14 days (no refills) and home isolation for household members qD X 14 days (refills allowed). And only the second group should be coming to our ERs. It is why we need to look broadly at our healthcare system (even including minute clinics at CVS/Walgreens and stand alone urgent care centers). We need to start introducing this now.

Sent from Mail for Windows 10

From: Dr. (b)(6)

Sent: Tuesday, February 25, 2020 8:47 AM

To: (b)(6) (b)(6)

Cc: Martin, (b)(6) J; Walters, (b)(6) (b)(6) (b)(6) (b)(6) Caneva, Duane; (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b)(6) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)(6) (Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; (b)(6) (b)(6) (b)(6) (b)(6) eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)(6) TARANTINO, (b)(6) A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) Lee, Scott; Padget, Larry G

Subject: RE: Red Dawn Breaking Bad, Start Feb 24

This is extremely troubling that healthcare workers, including medical leaders are contracting the COVID-19 while caring for the infected. I am very worried about the supply-chain regarding all the protective gears, medical supplies and everything that is needed to combat this disease. The demand is rising and there is no guarantee that we can continue with the supply since the supply-chain has been disrupted.

I do not know if we have enough resources to protect all frontline providers.

(b)(6)

https://newton.isye.gatech.edu/DrLee/

mobile: (b)(6)

Sent with ProtonMail Secure Email.

------- Original Message -------

On Tuesday, February 25, 2020 8:37 AM, (b)(6) (b)(6) (b)(6) wrote:

For those who cannot access Twitter but can access YouTube, here is the video.

https://www.youtube.com/watch?v=4AX4dbXIsSw

Imagine if something like this happened in the US with an equivalent national leader (watching him coughing during a press conference, rubbing his nose, and wiping the sweat from his forehead, and leaning on the podium with one hand while someone else is speaking) and then learning afterwards that he is infected with COVID. That is what has happened in Iran.

Sent from Mail for Windows 10

From: (b)(6) (b)(6)

Sent: Tuesday, February 25, 2020 8:01 AM

To: Martin, (b)(6) J; Walters, (b)(6) (b)(6) (b)(6) (b)(6) Caneva, Duane; (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b)((b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman,

(b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)(6) (Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; (b)(6) (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)(6) TARANTINO, (b)(6) A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) Dr. (b)((b)((b)(Scott

Cc: Padget, Larry G

Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Modeling isolation and social distancing (not)

https://twitter.com/BNODesk/status/1232276183305400320

Imagine how widespread it must be to see this?

Sent from Mail for Windows 10

From: (b)(6) (b)(6)

Sent: Tuesday, February 25, 2020 7:20 AM

To: Martin, (b)(6) J; Walters, (b)(6) (b)(6) (b)(6) (b)(6) Caneva, Duane; (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b)((b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)(6) (Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; (b)(6) (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)(6) TARANTINO, (b)(6) A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) Dr. (b)((b)((b)(Scott

Cc: Padget, Larry G

Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Just a word of caution. Here is what those numbers would mean. These are the projections using an attack rate of 30% (could send you the model in Excel and you can plug in whatever attack rate you like). I suspect that those CFRs are inflated because of case ascertainment issues (the actual denominator is much larger). The most recent background annual death data in the US (US Vital Statistics) is from 2017. It is useful to have those numbers of comparison.

I think those CFRs are on the high side., but don't have anything better to offer.

The best data we have for estimating CFR will be from the cruise ship, where we have a circumscribed population. That data is extremely valuable because the cruise ship had a crew of 1,045 (young and health) and 2,666 passengers (elderly). It will be important to look at each group separately. The data that I am aware of (all obtained thru open sources) is shared below this table).

AGE	2017 Census	2017 All Cause Mortality per 100,00	Actual 2017 All Cause Deaths	CFR	Projected 2019-nCoV Deaths	Ratio Projected Deaths to 2017 All Cause Deaths
<1 year	3,853,472	567.0	22,335	0.0%	0	0.00
1-4 years	16,085,388	24.3	3,880	0.0%	0	0.00
5 to 9 years	20,304,238	11.6	2,354	0.0%	0	0.00
10 to 14 years	20,778,454	15.5	3,217	0.2%	12,467	3.88
15 to 19 years	21,131,660	51.5	10,886	0.2%	12,679	1.16
20 to 24 years	22,118,635	95.6	21,139	0.2%	13,271	0.63
25 to 29 years	23,370,460	121.0	28,276	0.2%	14,022	0.50
30 to 34 years	21,972,212	145.4	31,939	0.2%	13,183	0.41
35 to 39 years	21,231,997	173.8	36,901	0.2%	12,739	0.35
40 to 44 years	19,643,373	218.4	42,895	0.4%	23,572	0.55
45 to 49 years	20,973,858	313.2	65,698	0.4%	25,169	0.38
50 to 54 years	21,401,094	488.0	104,444	1.3%	83,464	0.80
55 to 59 years	22,007,956	736.5	162,098	1.3%	85,831	0.53
60 to 64 years	19,987,702	1,050.2	209,908	3.6%	215,867	1.03
65 to 69 years	16,836,381	1,473.5	248,087	3.6%	181,833	0.73
70 to 74 years	12,847,065	2,206.9	283,523	8.0%	308,330	1.09
75 to 79 years	8,741,261	3,517.8	307,498	8.0%	209,790	0.68
80 to 84 years	5,965,290	5,871.7	350,261	14.8%	264,859	0.76
85 years and over	6,468,682	13,573.6	878,035	14.8%	287,209	0.33

Total population	325,719,178	863.8	2,813,503	1.8%	1,764,286	0.63

Attack Rate	30%

Date	Event	Cumulative Number of Confirmed Cases	Cumulative Number of Deaths	Note
20-Jan	Cruise ship departs from Yokohama Japan			
25-Jan	80 year old passenger disembarks in Hong Kong			
1-Feb	80 year old passenger confirmed to have COVID-19			
	When results known, certificate of landing canceled and ship under quarantine. Tests for the virus would be administered to three groups: those with symptoms, those who got off in Hong Kong, and those who had close contact with the infected passenger.			
3-Feb	Ship arrives in port of Yokohama Japan			
5-Feb	10 passengers and crew confirmed +	10		
6-Feb	31 more passengers and crew confirmed +	41		
7-Feb	30 more passenger and crew confirmed +	61		
8-Feb	9 more passenger and crew confirmed +	70		
10-Feb	66 more passenger and crew confirmed +	136		439 tes
11-Feb	39 more passenger and crew confirmed +	175		492 tes
12-Feb	28 more passenger and crew confirmed +	203		4 in IC
13-Feb	15 more passenger and crew confirmed +	218		713 tes
14-Feb	67 more passenger and crew confirmed +	285		927 tes
15-Feb	70 more passenger and crew confirmed +	355		1,219 tested 73 asympt
16-Feb	329 American evacuated from cruise ship (14 of the evacuees found to be +) 61 Americans remained on board 44 Americans remained hospitalized in Japan	369		
17-Feb	85 more passenger and crew confirmed +	454		1,723 tested 19 seriou
18-Feb	167 more passenger and crew confirmed +	621		3,011 te

19-Feb	2 deaths	621	2	
20-Feb	13 more passenger and crew confirmed +	634	2	3,066 tested 28 seriously 322 asymp
23-Feb	Death reported in Japan		3	
24-Feb	Japan updates total to 691; US reports 36 in US	691	3	
25-Feb	Death reported in Japan	691	4	

Total passengers and crew:

691 cases (~50% asymptomatic) / 28 seriously ill (4%) / 4 deaths (a lagging measure)

So overall, a lower limit CFR of 0.6%--this includes the young healthy crew members and the elderly passengers.

Assuming the number of seriously ill approximates the % of cases requiring ICU care (4%) and the typical mortality rates for patients with pneumonia admitted to the ICU are 15%-50%, then CFR would be estimated to be0.6%-2% overall. The ICU data is sketchy (last data point I have is from Japan only on Feb 20). If someone has actual numbers of patients admitted to the ICU, just plug in and make your won estimate.

Americans:

Total Americans 434

- 329 evacuated
- 61 remained on board
- 44 in hospitals in Japan

80 cases (44 hospitalized in Japan/36 cases in US)

Media has only reported on a single American who was in the ICU in Japan (that is from Feb 11). There is nothing being reported in the media on current numbers of cases in ICU of Americans in the US or Japan. If anyone has that data (but cannot share it), would suggest you take that number divide it by the number of known American cases (80) to estimate a % of cases requiring ICU care. Multiply that number by 15%-50% to get a range of CFRs.

A while back I shared some estimates based upon the data from the cruise ship and compared to the 2005 HHS projections of a severe pandemic. Just to put those numbers in perspective. The

2005 estimates were that 30% of the population would become ill (30% attack rate); 11% of those who became ill would require hospitalization; 1.6% of those who became ill would require ICU care; and 2% of those who became ill would die.

We are in the ballpark.

Sent from Mail for Windows 10

From: Martin, (b)(6) J

Sent: Tuesday, February 25, 2020 6:04 AM

To: Walters, (b)(6) (b)(6) (b)(6) (b)(6) Caneva, Duane; (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b)(6) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6), (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)(6) (Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; (b)(6) (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)(6) TARANTINO, (b)(6) A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) Dr. (b)((b)((b)((b), Scott; (b)(6) (b)(6)

Cc: Padget, Larry G

Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Wil,

This is what I've been using in our provider PowerPoint

Greg

Gregory J. Martin, MD

Chief, Tropical Medicine-Infectious Diseases

Bureau of Medical Services

US Department of State

2401 E St NW (SA-1)

Washington, DC 20522

BlackBerry while traveling: (b) (b)(6)

(b)(6)

From: Walters, (b)(6) (b)(6)

Sent: Tuesday, February 25, 2020 6:56 PM

To: (b)(6) (b)(6) (b)(6) (b)(6) Caneva, Duane <duane.caneva@hq.dhs.gov>; rjglassjr@gmail.com; Dodgen, Daniel (OS/ASPR/SPPR) <Daniel.Dodgen@HHS.GOV>; DeBord, Kristin (OS/ASPR/SPPR) <Kristin.DeBord@hhs.gov>; Phillips, Sally (OS/ASPR/SPPR) <Sally.Phillips@hhs.gov>; (b)(6) (b)(6) <(b)(6) (b)(6) (b)(6) J CIV USARMY (USA) (b)(6) (b)((b)(6) (b)(6) ; (b)(6) (b)(6) (b)(6) WOLFE, HERBERT (b)(6) Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D. (b)(6) (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA) <Robert.Johnson@hhs.gov>; Yeskey, (b)(6) (b)(6) Disbrow, Gary (OS/ASPR/BARDA) <Gary.Disbrow@hhs.gov>; Redd, John (OS/ASPR/SPPR) <John.Redd@hhs.gov>; Hassell, (b)(6) (Chris) (OS/ASPR/IO) (b)(6) Hamel, Joseph (OS/ASPR/IO) <Joseph.Hamel@hhs.gov>; Dean, Charity A@CDPH (b)(6) (b)(6) (b)(6) V (b)(6) Martin, (b)(6) J (b)(6) (b)(6) (b)(6) (b)(6) ; (b)(6) (b)((b)(6) eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)(6) (b)(6) TARANTINO, (b)(6) A (b)(6) WILKINSON, THOMAS (b)(6) (b)(6) KAUSHIK, SANGEETA (b)(6) (b)(6) (b)(6) (b)(6) Tracey (b)(6) <tmcNamara@westernu.edu>; Dr. (b)((b) (b)(6) (b)(Scott (b)(6) (b)(6) (b)(6) (b)(6)

Subject: Re: Red Dawn Breaking Bad, Start Feb 24

Colleagues,

Does anyone have a case fatality rate projection broken down by age?

William A. Walters, M.D., MBA

Executive Director and

Managing Director for Operational Medicine

Bureau of Medical Services

U.S. Department of State

(703) 957-9493

walterswa2@state.gov

walterswa2@state.sgov.gov

(b)(6)

From: (b)(6) (b)(6) (b)(6)
Sent: Monday, February 24, 2020 4:58:53 PM
To: (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Caneva, Duane (b)(6) (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR) <Daniel.Dodgen@HHS.GOV>; DeBord, Kristin (OS/ASPR/SPPR) <Kristin.DeBord@hhs.gov>; Phillips, Sally (OS/ASPR/SPPR) <Sally.Phillips@hhs.gov>; (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA) (b)(6) (b)(6) (b)(6) (b)(6) Walters, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT (b)(6) Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D. (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA) <Robert.Johnson@hhs.gov>; Yeskey, (b)(6) (b)(6) Disbrow, Gary (OS/ASPR/BARDA) <Gary.Disbrow@hhs.gov>; Redd, John (OS/ASPR/SPPR) <John.Redd@hhs.gov>; Hassell, David (Chris) (OS/ASPR/IO) <David.Hassell@hhs.gov>;

Hamel, Joseph (OS/ASPR/IO) <Joseph.Hamel@hhs.gov>; Dean, Charity A@CDPH (b)(6) (b)(6) (b)(6) V (b)(6) Martin, (b)(6) J (b)(6) (b)(6) (b)(6) <LBorio@iqt.org>; (b)(6) (b)((b)(6) eric.mcdonald@sdcounty.ca.gov <eric.mcdonald@sdcounty.ca.gov>; (b)(6) (b)(6) (b)(6) TARANTINO, (b)(6) A (b)(6) WILKINSON, THOMAS (b)(6) (b)(6) (b)(6) KAUSHIK, SANGEETA (b)(6) (b)(6) (b)(6) <nah2005@med.cornell.edu>; Tracey (b)(6) (b)(6) Dr. (b)(((b) <evalee-gatech@pm.me>; (b)(Scott (b)(6)
Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Several new countries announced first confirmed cases

Afghanistan

Bahrain

Iraq

Kuwait

Oman

Sent from Mail for Windows 10

From: (b)(6) (b)(6)

Sent: Monday, February 24, 2020 1:51 PM

To: (b)(6) (b)(6) (b)(6) Caneva, Duane; (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b)((b)(6) Walters, (b)(6) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)(6) (Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; Martin, (b)(6) J (b)(6) (b)(6) (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6)

David; TARANTINO, DAVID A; WILKINSON, THOMAS; (b)(6)
KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) Dr. (b)((b)(Lee, Scott (OS/ASPR/EMMO)

Subject: RE: Red Dawn Breaking Bad, Start Feb 24

The number of Americans confirmed positives from the cruise ship evacuated to the US was incorrect, it should have been 36

Sent from Mail for Windows 10

From: (b)(6) (b)(6)

Sent: Monday, February 24, 2020 1:28 PM

To: (b)(6) (b)(6) Caneva, Duane; (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b)((b)(6) Walters, (b)(6) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, David (Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; Martin, (b)(6) J (b)(6) (b)(6) (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)(6) TARANTINO, (b)(6) A; WILKINSON, THOMAS; (b)(6)
KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) Dr. (b)((b)((b)(Scott (OS/ASPR/EMMO)

Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Interesting account from China –

https://www.huffpost.com/entry/coronavirus-covid19-life-china-precautions_n_5e4eaa73c5b6df1e8be3d4d7

From: (b)(6) (b)(6) (b)(6)

Sent: 24 February 2020 18:18

To: Caneva, Duane (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR) <Daniel.Dodgen@HHS.GOV>; DeBord, Kristin (OS/ASPR/SPPR) <Kristin.DeBord@hhs.gov>; Phillips, Sally (OS/ASPR/SPPR) <Sally.Phillips@hhs.gov>; (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA) (b)(6) (b)(6) (b)(6) (b)(6) Walters, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT (b)(6) Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D. (b)(6) (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA) <Robert.Johnson@hhs.gov>; Yeskey, (b)(6) (b)(6) Disbrow, Gary (OS/ASPR/BARDA) <Gary.Disbrow@hhs.gov>; Redd, John (OS/ASPR/SPPR) <John.Redd@hhs.gov>; Hassell, David (Chris) (OS/ASPR/IO) (b)(6) Hamel, Joseph (OS/ASPR/IO) <Joseph.Hamel@hhs.gov>; Dean, Charity A@CDPH (b)(6) (b)(6) (b)(6) V (b)(6) Martin, (b)(6) J (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) <DHanfling@iqt.org>; eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)(6) (b)(6) TARANTINO, (b)(6) A (b)(6) WILKINSON, THOMAS (b)(6) (b)(6) KAUSHIK, SANGEETA (b)(6) (b)(6) (b)(6) <nah2005@med.cornell.edu>; Tracey (b)(6) (b)(6) Dr. (b)(6) (b)(6) (b)(6) Scott (OS/ASPR/EMMO) (b)(6)

Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Headlines:

Markets In Total Panic Mode As Coronavirus Cases Jump

"It's Total Panic" - Store-Shelves Empty As Virus-Spread Sparks Panic-Buying Food & Masks Across Italy

Sent from Mail for Windows 10

From: (b)(6) (b)(6)

Sent: Monday, February 24, 2020 1:01 PM

To: Caneva, Duane; (b)(6) (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b)((b)(6) Walters, (b)(6) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)(6) (Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; Martin, (b)(6) J (b)(6) (b)(6) (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6), (b)(6) TARANTINO, (b)(6) A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) Dr. (b) ((b)((b), Scott (OS/ASPR/EMMO)

Subject: RE: Red Dawn Breaking Bad, Start Feb 24

-Singapore +1 over the past 3 days (90 cases/0 deaths) 38 currently hospitalized, 7 in ICU

-Hong Kong now up to 81 cases/2 deaths

-South Korea continues to have explosive growth now up to 833 cases/7 deaths

-Japan is up to 156 cases/2 deaths

> **According to the Ministry of Health, Labor and Welfare, two new cases of infection have been confirmed on the 24th, including employees of the Ministry of Health, Labor and Welfare and quarantine officers who responded on a cruise ship. 7 staff**

members of the Ministry of Health, Labor and Welfare and quarantine officers have been confirmed +.

-Italy has 227 cases/7 deaths

-Iran 61 cases/12 deaths

Sent from Mail for Windows 10

From: Caneva, Duane

Sent: Monday, February 24, 2020 12:30 PM

To: (b)(6) (b)(6) (b)(6) Dodgen, Daniel (OS/ASPR/SPPR); DeBord, Kristin (OS/ASPR/SPPR); Phillips, Sally (OS/ASPR/SPPR); (b)(6) (b)(6) (b)(6) (b)(6) J CIV USARMY (USA); (b)((b)(6) Walters, (b)(6) (b)(6) (b)(6) (b)(6) WOLFE, HERBERT; Eastman, (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) V.,M.D.; (b)(6) (b)(6) Johnson, Robert (OS/ASPR/BARDA); Yeskey, (b)(6) Disbrow, Gary (OS/ASPR/BARDA); Redd, John (OS/ASPR/SPPR); Hassell, (b)(6) (Chris) (OS/ASPR/IO); Hamel, Joseph (OS/ASPR/IO); Dean, Charity A@CDPH; (b)(6) (b)(6) V; Martin, (b)(6) J (b)(6) (b)(6) (b)(6) (b)(6) (b)(eric.mcdonald@sdcounty.ca.gov; (b)(6) (b)(6) TARANTINO, (b)(6) A; WILKINSON, THOMAS; (b)(6) KAUSHIK, SANGEETA; (b)(6) (b)(6) Tracey (b)(6) Dr. (b) ((b)(Redd, John (OS/ASPR/SPPR); (b)(6) (b)(6) Yeskey, Kevin; Lee, Scott (OS/ASPR/EMMO)

Subject: RE: Red Dawn Breaking Bad, Start Feb 24

Singapore COVID-19 Guidance Page:

https://www.gov.sg/article/covid-19-sector-specific-advisories

Best,

Duane

From: Caneva, Duane

Sent: Monday, February 24, 2020 12:28 PM

To: Subject: Red Dawn Breaking Bad, Start Feb 24

Importance: High

All,

This is a new Red Dawn Email String. Please use this one going forward.

Best,

Duane

Duane C. Caneva, MD, MS

Chief Medical Officer

Department of Homeland Security

(b)(6) (o)

(b)(6) (c)

(b)(6)

(b)(6)

Executive Assistant: (b)(6) (b)(6) (b)(6) (b)(6)

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<2020_02_19_MQL_SARS-CoV-2_CLEARED FOR RELEASE.PDF>

Sender:	Caneva, Duane (b)(6)
Recipient:	(b)(6) (b)(6) (b)(6) Dr. (b) (b) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6) (b)(6)

(b)(6) (b)(6) (b)(6)
Martin, (b)(6) J (b)(6)
Walters, (b)(6) (b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group
(b)(6)
(b)(6)
(b)(6) (b)(6) (b)(6)
(b)(6)
Dodgen, Daniel (OS/ASPR/SPPR) /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=c23f0d7c1d634508918e1c87cf50c48c-Dodgen, Dan
<Daniel.Dodgen@HHS.GOV>;
DeBord, Kristin (OS/ASPR/SPPR) /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=d2cce28859884c1c835a9404885d6534-DeBord, Kri
<Kristin.DeBord@hhs.gov>;
Phillips, Sally (OS/ASPR/SPPR) /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=5d6ce8134846423cb83b5b28464edb60-Phillips, S
<Sally.Phillips@hhs.gov>;
(b)(6) (b)(6) (b)(6)
(b)(6) (b)(6) J CIV USARMY (USA) (b)(6)
(b)(6) (b)(6) (b)(6)
(b)(6) (b)(6) (b)(6)
Wolfe, Herbert (DHS.GOV) /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=f2fb845c2e154d8e967ec3fdabecfbd6-Herbert.Wol
(b)(6)
(b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=5d6cb05aaaff4f9fb1ee44181880376c-Guest_37b50
(b)(6)
(b)(6) (b)(6) (b)(6)
(b)(6) (b)(6) V., M.D. (b)(6)
(b)(6) (b)(6)
(b)(6)
Johnson, Robert (OS/ASPR/BARDA) /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=0851e89240324306b78740a4a60745e2-Johnson, Ro
<Robert.Johnson@hhs.gov>;
Yeskey, Kevin (OS/ASPR/IO) /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=6fe6cf13518445fd9c3a1c254e166b3f-Yeskey, Kev
<Kevin.Yeskey@hhs.gov>;
Disbrow, Gary (OS/ASPR/BARDA) /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=0fd5845defda4dc0bb45f8fac629cf09-Disbrow, Ga
<Gary.Disbrow@hhs.gov>;
Redd, John (OS/ASPR/SPPR) /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=9ba3fed4ee8646ec849a5a87136a24f6-Redd, John
<John.Redd@hhs.gov>;
Hassell, David (Chris) (OS/ASPR/IO) /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=aedbfb0ff96e4119ac7a3b3abaf71a3d-Hassell, Da
<David.Hassell@hhs.gov>;
Hamel, Joseph (OS/ASPR/IO) /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=96d2c1602dfa45e5a5e21452a098b96d-Hamel, Jose
<Joseph.Hamel@hhs.gov>;
Dean, Charity A@CDPH (b)(6)
(b)(6) (b)(6) V (b)(6)
(b)(6) (b)(6) (b)(6)
(b)(6) (b)(6) (b)(6)
eric.mcdonald@sdcounty.ca.gov /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=04b8a65f67f9453b9749d3d366481f53-Guest_bef32
<eric.mcdonald@sdcounty.ca.gov>;
(b)(6) (b)(6) (b)(6)
(b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=2b8ce179913e4ec6a251a91616b1bc4a-Guest_de2e9
(b)(6)
WILKINSON, THOMAS (b)(6)
(b)(6) /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=user4629b831 (b)(6)
KAUSHIK, SANGEETA (b)(6)
(b)(6) (b)(6) (b)(6)
Lee, Scott (OS/ASPR/EMMO) /o=ExchangeLabs/ou=Exchange Administrative Group
(FYDIBOHF23SPDLT)/cn=Recipients/cn=userf6879348 (b)(6)

	Padget, Larry G (b)(6) (b) (b)(6) (b)(6) Stack, (b)(6) J (CHFS DPH) (b)(6) Adams, (b)(6) (HHS/OASH) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=184fe51f7350400b8a99dabd65ac0db5-Adams, Jero (b)(6) Fantinato, Jessica (USDA.GOV) /o=ExchangeLabs/ou=Exchange Administrative Group (FYDIBOHF23SPDLT)/cn=Recipients/cn=4a03b6317c3840afb4ee617008e821a6-Jessica.Fan <jessica.fantinato@usda.gov>; Colby, Michelle - OHS, Washington, DC <michelle.colby@usda.gov>
Sent Date:	2020/02/27 16:19:53
Delivered Date:	2020/02/27 16:22:04
Message Flags:	Unread

SARS-CoV-2 (COVID-19)	Infectious dose – how much agent will make a normal individual ill?	Transmissibility – How does it spread from one host to another? How easily is it spread?	Host range – how many species does it infect? Can it transfer from species to species?	Incubation period – how long after infection do symptoms appear? Are people infectious during this time?
What do we know?	• The human infectious dose for novel Wuhan coronavirus (SARS-CoV-2), which causes coronavirus disease 19 (COVID-19) is currently unknown via all exposure routes. Severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS) coronaviruses (CoV) are used as surrogates. • The infectious dose for SARS in mice is estimated to be between 67-540 PFU (average 240 PFU, intranasal route)[43-44] • Genetically modified mice exposed intranasally to doses of MERS virus between 100 and 500,000 PFU show signs of infection. Infection with higher doses result in severe syndromes.[5, 34, 65, 124] • Initial experiments suggest that SARS-CoV-2 can infect genetically modified mice containing the human ACE2 cell entry receptor. Infection via the intranasal route (dose: 10^5 $TCID_{50}$) causes light infection, however no virus was isolated from infected animals, and PCR primers used in the study do not align well with SARS-CoV-2, casting doubt on this study.[11]	• SARS-CoV-2 has been declared a Public Health Emergency of International Concern by the WHO[110] with 81,332 cases and 2,770 deaths[60] in 38 countries.[19, 89, 108] (2/26/20) • While the rate of new cases in China has slowed, there are now large outbreaks in Japan, Italy, Iran and South Korea.[104] • High-quality estimates of human transmissibility (R_0) range from 2.2 to 3.1[72, 79, 84, 114, 123] • Recent studies suggest that pre-symptomatic[125] or asymptomatic[10] patients in China are able to transmit SARS-CoV-2, however the degree of asymptomatic transmission is still unknown. • SARS-CoV-2 is believed to spread between humans via the respiratory route, including droplet transmission.[24] • Viable SARS-CoV-2 has been isolated from human feces, suggesting the possibility of the fecal-oral transmission[73, 119, 122] • Transmission via fomites (contaminated surfaces) has not been confirmed for SARS-CoV-2, but contributed to the large number of nosocomial cases in prior SARS[33, 115] and MERS[62] outbreaks • SARS-CoV-2 is consistently present in infected patient saliva[97] • Infants have been diagnosed with COVID-19, but no evidence exists for vertical transmission via intrauterine infection or through breastmilk.[32, 103] • SARS-CoV-2 transmission has occurred in hospitals both inside[102] and outside of China.[51] • China reports no evidence of super-spreading events (SSEs) within hospital patients or staff.[94]	• Early genomic analysis indicates similarity to SARS,[127] with a suggested bat origin.[5,35, 127] • Analysis of SARS-CoV-2 genomes suggests that a non-bat intermediate species is responsible for the beginning of the outbreak.[85] Although the identity of the intermediate species remains unconfirmed, pangolins may be a natural host of related viruses possibly including SARS-CoV-2.[68-69] • Positive samples from the South China Seafood Market strongly suggests a wildlife source,[26] though it is possible that the virus was circulating in humans before the disease was associated with the seafood market[13, 36, 116, 121] • Experiments suggest that SARS-CoV-2 Spike (S) receptor-binding domain binds the human cell receptor (ACE2) stronger than SARS,[113] potentially explaining its high transmissibility; the same work suggests that differences between SARS-CoV-2 and SARS-CoV Spike proteins may limit the therapeutic ability of SARS antibody treatments.[113] • Modeling between SARS-CoV-2 Spike and ACE2 proteins suggests that SARS-CoV-2 can bind and infect human, bat, civet, monkey and swine cells.[101]	• A study of 1,099 COVID-19 patients found a median incubation period of 3 days, with a range from 0 to 24 days.[50] • Earlier estimates of the incubation period from confirmed cases were higher; 5.8 days with a range from 1.3 to 11.3 days,[9] and 5.2 days with an upper bound of 9.2-18 days.[67] • CDC estimates that the incubation period is between 2 and 14 days [21, 25] • Asymptomatic infection has been documented, where individuals do not present with clinical symptoms but are found positive via diagnostic assay.[10, 28, 50, 94, 125] • Individuals can be infectious while asymptomatic,[24, 86, 94, 125] and asymptomatic individuals have about the same amount of virus in their nose and throat as symptomatic individuals.[128] • Infectious period is unknown, but possibly up to 10-14 days [4, 89] • On average, there are 7.5 days between symptom onset in successive cases of a single transmission chain (i.e., the serial interval).[67] • The average time for individuals to first seek medical care decreased from 5.8 days after symptom onset to 4.6 days before and after January 1st, 2020, respectively, indicating an increase in seeking care behavior.[67] • China recommends 14 quarantine for *recovered* patients due to positive genetic tests days after leaving the hospital, raising the possibility of continued transmission after symptoms subside.[56]

SARS-CoV-2 (COVID-19)	Infectious dose – how much agent will make a normal individual ill?	Transmissibility – How does it spread from one host to another? How easily is it spread?	Host range – how many species does it infect? Can it transfer from species to species?	Incubation period – how long after infection do symptoms appear? Are people infectious during this time?
What do we need to know?	• Human infectious dose by aerosol route • Human infectious dose by surface contact (fomite) • Human infectious dose by fecal-oral route • Where does SARS-CoV-2 replicate in the respiratory tract?	• Capability of SARS-CoV-2 to be transmitted by contact with fomites (doorknobs, surfaces, clothing, etc.) – see also Experimental Stability • Superspreading capacity needs to be refined • Updated person to person transmission rates (e.g., R_0) as control measures take effect • Tendency for ill individuals to seek medical care due to symptoms • What is the underreporting rate?[59] • Proper procedures for reducing spread in medical facilities / transmission rate in medical settings • Can individuals become reinfected with SARS-CoV-2? • What is the difference in transmissibility among countries?	• What is the intermediate host(s)? • Ability of SARS-CoV-2 to bind to human ACE2 receptor (need live virus confirmation in addition to genetic similarity) • What are the mutations in SARS-CoV-2 that allowed human infection and transmission?	• How early does asymptomatic transmission begin? • What is the average infectious period during which individuals can transmit the disease? • How long do patients continue to shed infectious virus after physical recovery?
Who is doing experiments/has capabilities in this area?	*Capable of performing work* • DHS National Biodefense Analysis and Countermeasures Center (NBACC)	*Performing work:* - Christian Althaus (Bern) - Neil Ferguson (MRC) - Gabriel Leung, Joseph Wu (University of Hong Kong) - Sara Del Valle (Los Alamos) - Maimuna Majumder (Boston Children's Hospital) - Trevor Bedford (Fred Hutchinson Cancer Center) - Sang Woo Park (Princeton)	*Capable of performing work:* • Vincent Munster (Rocky Mountain National Laboratory) • Matthew Frieman (University of Maryland Baltimore) • Ralph Baric (University of North Carolina) • Stanley Perlman (University of Iowa) • Susan Baker (Loyola University Chicago) • Mark Denison (Vanderbilt University) • Vineet Menachery (University of Texas Medical Branch) • Jason McLellan, Daniel Wrapp, Nianshuang Wang (University of Texas) *Starting animal work soon:* • David O'Conner (U. Wisconsin, Madison)	*Performing work:* • Chaolin Huang (Jin Yin-tan Hospital, Wuhan, China) • The Novel Coronavirus Pneumonia Emergency Response Epidemiology Team

SARS-CoV-2 (COVID-19)	Clinical presentation – what are the signs and symptoms of an infected person?	Clinical diagnosis – are there tools to diagnose infected individuals? When during infection are they effective?	Medical treatment – are there effective treatments? Vaccines?	Environmental stability – how long does the agent live in the environment?
What do we know?	• The majority of COVID-19 cases are mild (81%, N = 44,000 cases)[94] • Initial COVID-19 symptoms include fever (87.9% overall, but only 43.8% present with fever initially[50]), cough (67.7%[50]), fatigue, shortness of breath, headache, reduction in lymphocyte count.[25, 31, 55] Headache[30] and diarrhea are uncommon[55, 66] • Complications include acute respiratory distress (ARDS), pneumonia (characteristic ground glass opacities[77]), acute cardiac injury, secondary infection, kidney failure, arrhythmia, and shock.[50,55,102] • A small study suggested that infection severely impacts the immune response, specifically causing overactive T cells (an increase of Th17 cells and CD8 T cells) which contribute to symptom severity.[117] • Approximately 15% of hospitalized patients were classified as severe,[50, 94] and severe cases were older and more likely to have underlying disorders[50, 102]; approximately 5% of patients were admitted to the ICU.[50, 94] • Approximately 1% of hospitalizations occur in children < 19 years old.[50, 94] • The case fatality rate (CFR) depends on patient comorbidities; no comorbidities = 0.9%, cardiovascular disease = 10.5%, diabetes = 7.3%, chronic respiratory disease = 6.3%, hypertension = 6.0%, cancer = 5.6%.[94] • The CFR is age-dependent; ≥80 years old = 14.8%, 70-79 = 8.0%, 60-69 = 3.6%, 50-59 = 1.3%, 40-49 = 0.4%, 10-39 = 0.2%, 0-9 = 0%.[94] • 63.8% of confirmed fatalities have been male.[94] • Patients (N = 21) were hospitalized for a mean of 17 days (range = 11 – 26).[77]	• The US CDC has developed a rapid test kit,[18, 24] though issues with inconclusive results have been reported using the CDC test kit and not all states are able to use it to diagnose potential COVID-19 patients.[49] • Several RT-PCR assays have been developed to detect SARS-CoV-2 in humans.[1, 39, 107, 109] • Multiple institutions (Hong Kong University, China CDC, U.S. CDC, National Institute of Infectious Disease Japan, the Ministry of Public Health Thailand, Charité) have developed and shared PCR protocols and primers with international researchers.[18, 40, 67, 92, 105, 109] • PAHO advises that molecular diagnosis take place in a BSL-2 laboratory.[76] • Wuhan Institute of Virology has reportedly developed a test strip for SARS-CoV-2 diagnosis[42] • BGI has developed real-time detection kits: fluorescent RT-PCR and metagenomic sequencing detection kit.[14] • Cepheid is developing an automated molecular test for use in its GeneXpert System.[100] • SARS-CoV-2 is consistently present in infected patient saliva, suggesting that saliva may be an effective diagnostic specimen.[97] • RT-PCR tests are able to identify asymptomatic cases; SARS-CoV-2 infection was identified in 2/114 individuals previously cleared by clinical assessment.[54]	• Treatment for COVID-19 is primarily supportive care including oxygen and mechanical ventilation,[23, 118] though China has released a treatment plan[8]; over 80 clinical trials are set to run on various treatments in China.[74] • Efficacy of other treatments being administered to patients in China (lopinavir, ritonavir, ribavirin, oseltamivir) is unknown[8]; however several therapeutics [Remdesivir[91] and chloroquine] inhibit SARS-CoV-2 infection in human cells *in vitro*[32] and are undergoing clinical trials in China[42] and the US.[2-3, 75] • NIH/NIAID is working with multiple partied to develop a vaccine for SARS-CoV-2,[52, 64]. Moderna Therapeutics and Gilead Sciences are involved in clinical trials for therapeutic and vaccine solutions.[2-3, 75] Sanofi will work with HHS to develop coronavirus vaccine.[15] • The hospitalized case-fatality rate has decreased from 14.4% to 0.8% as of between December, 2019 and February, 2020.[94] • Approximately 38% of COVID-19 patients in China received oxygen therapy, 6.1% received mechanical ventilation, 57.5% received IV antibiotics, and 35.8% received the antiviral oseltamivir.[50] • A clinical report (one patient) suggested that coupling corticosteroid treatment with ventilator support should be considered for severe patients to prevent ARDS.[117] • Similarity in the spike proteins of SARS-CoV-2 and SARS-CoV might offer target for therapeutics,[37, 45, 63, 113, 127] as vaccines derived from spike proteins are effective at inhibiting MERS symptoms in mice[34].	• No information yet exists regarding the environmental stability of SARS-CoV-2; SARS and MERS coronaviruses are used as surrogates instead. • Studies suggest that coronavirus can survive on non-porous surfaces up to 9-10 days (MHV, SARS-CoV)[17, 29], and porous surfaces for up to 3-5 days (SARS-CoV)[47] in air conditioned environments (20-25°C, 40-50% RH) • Coronavirus survival tends to be higher at lower temperatures and lower relative humidity (RH),[17, 29, 82, 99] though infectious virus can persist on surfaces for several days in typical office or hospital conditions[99] • SARS can persist with trace infectivity for up to 28 days at refrigerated temperatures (4°C) on surfaces.[17] • Beta-coronaviruses (e.g., SARS-CoV) may be more stable than alpha-coronaviruses (HCoV-229E).[82] • No strong evidence for reduction in transmission with seasonal increase in temperature and humidity.[71] • Survival of SARS-CoV-2 specifically is unknown, and surrogate coronavirus data need to be used at this time. • One hour after aerosolization approximately 63% of airborne MERS virus remained viable in a simulated office environment (25°C, 75% RH)[80] • The aerosol survival of related human coronavirus (229E) was relatively high, (half-life of ~67 hours at 20°C and 50% RH), indicating ~20% of infectious virus remained after 6 days.[58] Both higher and lower RH reduced HCoV-229E survival; lower temperatures improved survival.[58]

SARS-CoV-2 (COVID-19)	Clinical presentation – what are the signs and symptoms of an infected person?	Clinical diagnosis – are there tools to diagnose infected individuals? When during infection are they effective?	Medical treatment – are there effective treatments? Vaccines?	Environmental stability – how long does the agent live in the environment?
What do we need to know?	• How long does it take for infected individuals to recover outside of a healthcare setting?	• False positive/negative rates for tests • Eclipse phase of infection (time between infection and detectable disease) in an individual	• Is GS-5734 (remdesivir) effective in vivo (already used in clinical trials under Emergency Use Authorization)?[90] • Is the GLS-5000 MERS vaccine[120] cross-reactive against SARS-CoV-2? • Efficacy of antibody treatments developed for SARS[41, 93] and MERS[27] • What is the efficacy of various MERS and SARS Phase I/II vaccines and other therapeutics? • Are viral replicase inhibitors such as beta-D-N4-hydroxycytidine effective against SARS-CoV-2?[12]	• Stability of SARS-CoV-2 in aerosol, droplets, and other matrices (mucus/sputum, feces) • "Hang time' of the virus in air (Aerosol decay rate) • Particle size distribution (e.g., droplet, large droplet and true aerosol distribution) • Duration of SARS-CoV-2 infectivity via fomites and surface (contact hazard)? • Stability of SARS-CoV-2 on PPE (e.g., Tyvek, nitrile, etc.)

SARS-CoV-2 (COVID-19)	Clinical presentation – what are the signs and symptoms of an infected person?	Clinical diagnosis – are there tools to diagnose infected individuals? When during infection are they effective?	Medical treatment – are there effective treatments? Vaccines?	Environmental stability – how long does the agent live in the environment?
Who is doing experiments/has capabilities in this area?	- Jin Yin-tan Hospital, Wuhan, China - China-Japan Friendship Hospital, Beijing, China - Peking Union Medical College, Beijing, China - Capital Medical University, Beijing, China - Chinese Academy of Medical Sciences and Peking Union Medical College, Beijing, China - Huazhong University of Science and Technology, Wuhan, China - The Central Hospital of Wuhan, Tongji Medical College, Huazhong University of Science and Technology, Wuhan, China - Tsinghua University School of Medicine, Beijing, China - Zhongnan Hospital of Wuhan University, Wuhan, China - Peking University First Hospital, Beijing, China - Peking University People's Hospital, Beijing, China - Tsinghua University-Peking University Joint Center for Life Sciences, Beijing, China - The Fifth Medical Center of PLA General Hospital, Beijing, China	*Performing work:* • CDC • Wuhan Institute of Virology • Public Health Agency of Canada • Doherty Institute of Australia • Cepheid • BGI	*Performing work:* • Peter Doherty Institute for Infection and Immunity • Academy of Military Medical Sciences, Beijing, China • Tim Sheahan (University of North Carolina) *Capable of performing work:* • Ralph Baric (University of North Carolina) • Matthew Frieman (University of Maryland Baltimore) • Sanofi, with BARDA (using their recombinant DNA platform) • Janssen Pharma and BARDA[53] *Funded work:* CEPI ($12 million to three groups): • Moderna and NIAID for mRNA platform vaccine • Inovio preparing DNA vaccine (for MERS) • University of Queensland, Australia, producing viral protein from cell culture NIAID/NIH - Moderna and Kaiser Permanente for mRNA vaccine Phase I trial.[3] - University of Nebraska Medical Center Trial. Multicenter trial of multiple IND Therapeutics, including Gilead's Remdesivir. [2]	*Capable of performing work*: • Mark Sobsey (University of North Carolina) • DHS National Biodefense Analysis and Countermeasures Center (NBACC) • Defence Science and Technology Laboratory (Dstl) • Public Health Agency of Canada • CDC • EPA • NIH

SARS-CoV-2 (COVID-19)	Decontamination – what are effective methods to kill the agent in the environment?	PPE – what PPE is effective, and who should be using it?	Forensics – natural vs intentional use? Tests to be used for attribution.	Genomics – how does the disease agent compare to previous strains?
What do we know?	• No decontamination data for SARS-CoV-2 have been identified. SARS-CoV provides a plausible surrogate, as it is a close genetic relative of SARS-CoV-2 in the beta-coronavirus clade. • Chlorine-based[107] and ethanol-based[38] solutions recommended, and the European CDC has released disinfectant guidelines for non-healthcare facilities.[48] • "The virus [SARS-CoV-2] has relatively weak viability *in vitro* and can be inactivated at 56 ° C for 30 minutes. Chlorine-containing disinfectants and 75% ethanol can effectively inactivate the virus."[126] • Heat treatment at 56°C is sufficient to kill coronaviruses,[82, 126] though effectiveness depends in part on amount of protein in contaminated media[82] • 70% ethanol, 50% isopropanol, sodium hypochlorite [bleach, 200 ppm], and UV radiation are effective at inactivating several coronaviruses (MHV and CCV)[87] • Ethanol-based biocides (including ethanol-containing gels) are effective disinfectants against coronaviruses dried on surfaces, including ethanol containing gels similar to hand sanitizer.[57, 111] • Surface spray disinfectants such as Mikrobac, Dismozon, and Korsolex are effective at reducing infectivity of the closely related SARS-CoV after 30 minutes of contact.[81] • Coronaviruses may be resistant to thermal inactivation for up to 7 days when stabilized in stool.[95-96] Additionally, coronaviruses are more stable in matrixes such as respiratory sputum.[46]	• PPE effectiveness for SARS-CoV-2 is currently unknown; SARS is used as a surrogate • "Healthcare personnel entering the room [of SARS-CoV-2 patients] should use standard precautions, contact precautions, airborne precautions, and use eye protection (e.g., goggles or a face shield)"[22] • WHO indicates healthcare workers should wear clean, non-sterile, long-sleeve gowns as well as gloves.[106] • Nosocomial infections with SARS-CoV were more likely during intratracheal intubation, manipulation of oxygen masks, suction before intubation, and non-invasive ventilation[98] • Respirators (NIOSH-certified N95, EUFFP2 or equivalent) are recommended for those dealing with possible aerosols[107] • Additional protection, such as a Powered Air Purifying Respirator (PAPR) with a full hood, should be considered for high-risk procedures (i.e., intubation, ventilation)[16] • Healthcare worker illnesses (over 1,000[94]) indicate potential for human-to-human transmission despite isolation, PPE, and infection control[88] • Porous hospital materials, including paper and cotton cloth, maintain infectious SARS-CoV for a shorter time than non-porous material.[61] • CDC recommends facemasks for individuals attempting to prevent spread of SARS-CoV-2 in the home[20]	• Genomic analysis places SARS-CoV-2 into the beta-coronavirus clade, with close relationship to bat viruses. The SARS-CoV-2 virus is distinct from SARS and MERS viruses.[45] • Genomic analysis suggest that SARS-CoV-2 is a natural variant, and is therefore unlikely to be human-derived or otherwise created by "recombination" with other circulating strains of coronavirus.[6, 127] • Some genomic evidence indicates a close relationship with pangolin coronaviruses[112]; data suggests that pangolins may be a natural host for beta-coronaviruses [68-69]. Additional research is needed. • Genomic data support at least two plausible origins of SARS-CoV-2: "(i) natural selection in a non-human animal host prior to zoonotic transfer, and (ii) natural selection in humans following zoonotic transfer."[6] Either scenario is consistent the observed genetic changes found in all known SARS-CoV-2 isolates. • Additionally, "[…] SARS-CoV-2 is not derived from any previously used virus backbone," reducing the likelihood of laboratory origination,[6] and "[…] genomic evidence does not support the idea that SARS-CoV-2 is a laboratory construct, [though] it is currently impossible to prove or disprove the other theories of its origin."[6]	• There have been no documented cases of SARS-CoV-2 prior to December 2019 • Preliminary genomic analyses, however, suggest that the first human cases of SARS-CoV-2 emerged between 10/19/2019 – 12/17/2019.[7, 13, 83] • The mutation rate of SARS-CoV-2 is estimated to be similar to other RNA viruses (e.g., SARS, Ebola, Zika), and is currently calculated to be between $3.29 \times 10^{-4} – 2.03 \times 10^{-3}$ substitutions per site per year (median 1.07×10^{-3}),[7] though this estimate may change as more genomes are sequenced. • Preliminary phylogenetic analysis identified a very close genetic similarity between SARS-CoV-2 and a Bat coronavirus (RaTG13) isolated from Yunnan Province, China; suggesting that SARS-CoV-2 originated from bats.[78] • Pangolin coronaviruses are closely related to both SARS-CoV-2 and the closely related Bat coronavirus (RaTG13); phylogenetic analysis suggested that SARS-CoV-2 is of bat origin, but is closely related to pangolin coronavirus.[68-69] • The Spike protein of SARS-CoV-2, which mediates entry into host cells and is the major determinant of host range, is very similar to the Spike protein of SARS-CoV.[70] The rest of the genome is more closely related to two separate bat [70] and pangolin[69] coronavirus. • Protein modeling and preliminary laboratory studies suggest that SARS-CoV-2 binds to the human ACE2 receptor,[101, 113] the same cellular entry receptor used by SARS and other beta-coronaviruses.

What do we need to know?	• What is the minimal contact time for disinfectants? • Are antiseptic wipes effective for cleaning hard, non-porous surfaces? • Does contamination with human fluids/waste alter disinfectant efficacy profiles?	• Evidence of spread among patients within hospitals, like SARS? • Mode of aerosol transmission? • Is virus detectable in aerosol samples from patient rooms? • How effective are barriers such as N95 respirators as well as surgical masks?	• What tests for attribution exist for coronavirus emergence? • What is the identity of the intermediate species? • Are there closely related circulating coronaviruses in bats or other animals with the novel PRRA cleavage site found in SARS-CoV-2?	• Are there similar genomic differences in the progression of coronavirus strains from bat to intermediate species to human?
Who is doing experiments/has capabilities in this area?	*Capable of performing work*: • DHS National Biodefense Analysis and Countermeasures Center (NBACC)	*Generating recommendations:* • WHO • CDC • Pan-American Health Organization	*Performing genomic investigations:* • Kristian Andersen, Andrew Rambaut, Ian Lipkin, Edward Holmes, Robert Garry (Scripps, University of Edinburgh, Columbia University, University of Sydney, Tulane, Zalgen Labs [Germantown, MD]) *Capable of performing work:* • Pacific Northwest National Laboratory • DHS National Biodefense Analysis and Countermeasures Center (NBACC)	*Performing work:* • Trevor Bedford (Fred Hutchinson Cancer Research Center) • (b) (b) UNC • National Institute for Viral Disease Control and Prevention, Chinese Center for Disease Control and Prevention • Shandong First Medical University and Shandong Academy of Medical Sciences • Hubei Provincial Center for Disease Control and Prevention • Chinese Academy of Sciences • BGI PathoGenesis Pharmaceutical Technology, Shenzhen, China • People's Liberation Army General Hospital, Wuhan, China • Wenzhou Medical University, Wenzhou, China • University of Sydney, Sydney, NSW, Australia • The First Affiliated Hospital of Shandong First Medical University (Shandong Provincial Qianfoshan Hospital), Jinan, China

Table 1. Definitions of commonly-used acronyms

Acronym/Term	Definition	Description
SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2	Official name for the virus previously known as 2019-nCoV.
COVID-19	Coronavirus disease 19	Official name for the disease caused by the SARS-CoV-2 virus.
CFR	Case Fatality Rate	Number of deaths divided by confirmed patients
PFU	Plaque forming unit	Measurement of the number of infectious virus particles as determined by plaque forming assay. A measurement of sample infectivity.
$TCID_{50}$	50% Tissue Culture Infectious Dose	The number of infectious units which will infect 50% of tissue culture monolayers. A measurement of sample infectivity.
HCW	Healthcare worker	Doctors, nurses, technicians dealing with patients or samples
SARS	Severe Acute Respiratory Syndrome	Coronavirus with over 8,000 cases in global 2002-2003 outbreak
MERS	Middle-East Respiratory Syndrome	Coronavirus with over 2,000 cases in regional outbreak since 2012
CoV	Coronavirus	Virus typified by crown-like structures when viewed under electron microscope
R_0	Basic reproduction number	A measure of transmissibility. Specifically, the average number of new infections caused by a typical infectious individual in a wholly susceptible population.
MHV	Mouse hepatitis virus	Coronavirus surrogate
CCV	Canine coronavirus	Canine coronavirus
Fomite	Inanimate vector of disease	Surfaces such as hospital beds, doorknobs, healthcare worker gowns, faucets, etc.

Droplet transmission	Sneezing, coughing	Transmission via droplets requires relatively close contact (e.g., within 6 feet)
Airborne transmission	Aerosolization of infectious particles	Aerosolized particles can spread for long distances (e.g., between hospital rooms via HVAC systems)
Transgenic	Genetically modified	In this case, animal models modified to be more susceptible to MERS and/or SARS by adding proteins or receptors necessary for infection
Intranasal	Agent deposited into external nares of subject	Simulates inhalation exposure by depositing liquid solution of pathogen/virus into the nose of a test animal, where it is then taken up by the respiratory system.
Incubation period	Time between infection and symptom onset	Time between infection and onset of symptoms typically establishes guidelines for isolating patients before transmission is possible
Infectious period	Length of time an individual can transmit infection to others	Reducing the infectious period is a key method of reducing overall transmission; hospitalization, isolation, and quarantine are all effective methods
Serial interval	Length of time between symptom onset of successive cases in a transmission chain	The serial interval can be used to estimate R_0, and is useful for estimating the rate of outbreak spread
Superspreading	One individual responsible for an abnormally large number of secondary infections	Superspreading can be caused by high variance in the distribution of secondary cases caused by a single individual; most individuals infect very few people, while some infect a large number, even with the same average number of secondary infections
Nosocomial	Healthcare- or hospital-associated infections	Characteristic of SARS and MERS outbreaks, lead to refinement of infection control procedures
ACE2	Angiotensin-converting enzyme 2	Acts as a receptor for SARS-CoV, allowing entry into human cells

ARDS	Acute respiratory distress syndrome	Leakage of fluid into the lungs which inhibits respiration and leads to death
PPE	Personal protective equipment	Gowns, masks, gloves, and any other measures used to prevent spread between individuals

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Subject:	June 2022 Virtual Meeting of the Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats
Date:	2022/04/04 12:58:47
Start Date:	2022/06/17 12:00:00
End Date:	2022/06/17 15:00:00
Priority:	Normal
Type:	Appointment
Location:	https://nasem.zoom.us/j/92914027352

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Dear all,

Please find attached a draft agenda with Zoom information and briefing materials.

Topic: June 2022 Meeting of the Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats
Time: June 17, 2021 12:00 PM – 3:00 PM ET
Join from PC, Mac, Linux, iOS or Android: https://nasem.zoom.us/j/92914027352
Or iPhone one-tap (b)(6)

Many thanks,
Shalini

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Meeting of the Standing Committee on Emerging Infectious Diseases and 21st Century Health Threats

Draft Agenda – June 17, 2022, Noon – 3:00 P.M. ET

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International Numbers

FRIDAY, JUNE 17, 2022

Purpose

- Gather a broad array of information and perspectives regarding the development of a medium- (up to five years) and long-term (more than five years) COVID-19 research agenda.
- Understand U.S. Government perspectives on the opportunities, needs, and utility for a coordinated long-term research agenda for COVID-19 to coordinate and align efforts effectively to advance evidence-based practice.
- Scope potential priority topic areas to consider for a comprehensive research agenda.

SESSION I — Welcoming Remarks and Sponsor's Reflections

12:00 p.m. — **Welcoming Remarks and Overview of the Agenda**

Harvey Fineberg, *Standing Committee Chair*
President
Gordon and Betty Moore Foundation

12:15 p.m. — **Welcome and Opening Remarks from the Sponsor**

David (Chris) Hassell
Deputy Assistant Secretary – Senior Science Advisor
Office of the Assistant Secretary for Preparedness and Response
U.S. Department of Health and Human Services

SESSION II — Reflections on COVID-19 Long-Term Research Agenda and Research Priorities

12:30 p.m. — **Perspectives from Federal Partners**

Objectives:

- Understand U.S. Government perspectives on the opportunities, needs, and utility for a coordinated long-term research agenda for COVID-19;
- Explore how efforts can be coordinated and aligned across federal, state, and local counterparts to advance evidence-based practice; and

- Scope potential priority topic areas to consider for a comprehensive research agenda.

Anthony Fauci *(pre-recorded presentation)*
Director
National Institute of Allergy and Infectious Diseases

Rayvon Fouché
Division Director, Social and Economic Sciences
National Science Foundation

TBD
Centers for Disease Control and Prevention

1:00 p.m. **Committee Perspectives on Research Priorities for a Medium-to-Long-Term COVID-19 Research Agenda**

Objectives:

- Identify initial areas of research priorities or scope of the research agenda
- Solicit input on the key components of such a research agenda.

Harvey Fineberg, *Standing Committee Chair*
President
Gordon and Betty Moore Foundation

2:30 p.m. **Perspectives from Other National Academies Activities**

Objectives:

- Gather information on projects at the National Academies related to a COVID-19 research agenda; and
- Scope potential priority topic areas to consider for a comprehensive research agenda.

Robert M. Groves *(Invited)*
Co-Chair, Societal Experts Action Network

Cinnamon Dixon
Co-Chair, The Action Collaborative on Disaster Research
Forum on Medical and Public Health Preparedness for Disasters and Emergencies

Kevin Anderson
Forum Member, Forum on Microbial Threats

SESSION III MEETING WRAP UP

2:45 p.m. **Committee Debrief, Next Steps, and Future Meeting Planning**

Harvey Fineberg, *Standing Committee Chair*
President
Gordon and Betty Moore Foundation

3:00 p.m. **MEETING ADJOURNS**

DHS SCIENCE AND TECHNOLOGY

Master Question List for COVID-19 (caused by SARS-CoV-2)

Weekly Report

25 March 2020

For comments or questions related to the contents of this document, please contact the DHS S&T Hazard Awareness & Characterization Technology Center at (b)(6)

Homeland Security

Science and Technology

CLEARED FOR PUBLIC RELEASE

FOREWORD

The Department of Homeland Security (DHS) is paying close attention to the evolving Coronavirus Infectious Disease (COVID-19) situation in order to protect our nation. DHS is working very closely with the Centers for Disease Control and Prevention (CDC), other federal agencies, and public health officials to implement public health control measures related to travelers and materials crossing our borders from the affected regions.

Based on the response to a similar product generated in 2014 in response to the Ebolavirus outbreak in West Africa, the DHS Science and Technology Directorate (DHS S&T) developed the following "master question list" that quickly summarizes what is known, what additional information is needed, and who may be working to address such fundamental questions as, "What is the infectious dose?" and "How long does the virus persist in the environment?" The Master Question List (MQL) is intended to quickly present the current state of available information to government decision makers in the operational response to COVID-19 and allow structured and scientifically guided discussions across the federal government without burdening them with the need to review scientific reports, and to prevent duplication of efforts by highlighting and coordinating research.

The information contained in the following table has been assembled and evaluated by experts from publicly available sources to include reports and articles found in scientific and technical journals, selected sources on the internet, and various media reports. It is intended to serve as a "quick reference" tool and should not be regarded as a comprehensive source of information, nor as necessarily representing the official policies, either expressed or implied, of the DHS or the U.S. Government. DHS does not endorse any products or commercial services mentioned in this document. All sources of the information provided are cited so that individual users of this document may independently evaluate the source of that information and its suitability for any particular use. This document is a "living document" that will be updated as needed when new information becomes available.

SARS-CoV-2 (COVID-19)	Infectious Dose – How much agent will make a healthy individual ill?	Transmissibility – How does it spread from one host to another? How easily is it spread?	Host Range – How many species does it infect? Can it transfer from species to species?
What do we know?	**The human infectious dose of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is unknown by all exposure routes. SARS-CoV-2 is the cause of coronavirus disease 19 (COVID-19).** • A total dose of approximately 700,000 plaque-forming units (PFU) of the novel coronavirus SARS-CoV-2 was sufficient to infect cynomolgus macaques via a combination intranasal and intratracheal exposure (10^6 $TCID_{50}$ total dose).[122] Macaques did not exhibit clinical symptoms, though viral shedding occurred (nose and throat).[122] • Rhesus macaques are effectively infected with SARS-CoV-2 via the ocular conjunctival and intratracheal route at a dose of approximately 700,000 PFU (10^6 $TCID_{50}$).[57] • Initial experiments suggest that SARS-CoV-2 can infect genetically modified mice containing the human ACE2 cell entry receptor. Infection via the intranasal route (dose: 10^5 $TCID_{50}$, approximately 70,000 PFU) causes light infection, however no virus was isolated from infected animals, and PCR primers used in the study do not align well with SARS-CoV-2, casting doubt on this study.[15] • The infectious dose for severe acute respiratory syndrome coronavirus 1 (SARS) in mice is estimated to be between 67-540 PFU (average 240 PFU, intranasal route).[55-56] • Genetically modified mice exposed intranasally to doses of Middle East respiratory syndrome coronavirus (MERS) virus between 100 and 500,000 PFU show signs of infection. Infection with higher doses result in severe syndromes.[7, 46, 86, 161]	**SARS-CoV-2 is passed easily between humans, likely through close contact with relatively large droplets and possibly through smaller aerosolized particles.** • Pandemic COVID-19 has caused 468,523 infections and 21,192 deaths[77] in at least 173 countries and territories (as of 3/25/2020).[30, 127, 147] • There are 66,132 SARS-CoV-2 cases across 50 US states, with 947 deaths. (as of 3/25/2020)[77]; there is sustained community transmission of COVID-19 in the US.[18] • High-quality estimates of human transmissibility (R_0) range from 2.2 to 3.1[100, 109, 119, 153, 160] • SARS-CoV-2 is believed to spread through close contact and droplet transmission,[34] with fomite transmission[78] and close-contact aerosol transmission also plausible.[23, 67] • SARS-CoV-2 replicates in the upper respiratory tract (e.g., throat), and infectious virus is detectable in throat and lung tissue for at least 8 days.[149] • SARS-CoV-2 is present in infected patient saliva,[137] lower respiratory sputum,[143] and feces.[90] • Aerosolized virus has been detected in COVID-19 patient rooms, with particle sizes within the human respirable range (0.25 – 2.5 µm).[96] **Individuals can transmit SARS-CoV-2 to others before they have symptoms.** • Pre-symptomatic[163] or asymptomatic[13] patients can transmit SARS-CoV-2; between 12%[60] and 23%[95] of infections may be caused by asymptomatic or pre-symptomatic transmission. **Undetected cases play a major role in transmission.** • Models suggest up to 86% of early COVID-19 cases in China were undetected, and these infections were the source for 79% of reported cases.[88] **Behavior changes may limit spread.** • Social distancing and other policies are estimated to have reduced COVID-19 spread by 44% in Hong Kong.[52] Non-pharmaceutical interventions (e.g., school closures, isolation) are likely required to limit transmission.[64]	**SARS-CoV-2 is closely related to other coronaviruses circulating in bats in Southeast Asia. Previous coronaviruses have passed through an intermediate mammal host before infecting humans; the identity of the SARS-CoV-2 intermediate host is unknown.** • Early genomic analysis indicates similarity to SARS,[166] with a suggested bat origin.[5,47, 166] • Analysis of SARS-CoV-2 genomes suggests that a non-bat intermediate species is responsible for the beginning of the outbreak.[121] The identity of the intermediate host remains unknown.[89, 92-93] • Positive samples from the South China Seafood Market strongly suggests a wildlife source,[36] though it is possible that the virus was circulating in humans before the disease was associated with the seafood market.[19, 48, 155, 159] **SARS-CoV-2 uses the same receptor for cell entry as the SARS coronavirus that circulated in 2002/2003.** • Experiments suggest that SARS-CoV-2 Spike (S) receptor-binding domain binds the human cell receptor (ACE2) stronger than SARS,[152] potentially explaining its high transmissibility; the same work suggests that differences between SARS-CoV-2 and SARS-CoV Spike proteins may limit the therapeutic ability of SARS antibody treatments.[152] • Modeling between SARS-CoV-2 Spike and ACE2 proteins suggests that SARS-CoV-2 can bind and infect human, bat, civet, monkey and swine cells.[141] • There is currently no evidence that SARS-CoV-2 infects domestic animals or livestock.

SARS-CoV-2 (COVID-19)	Infectious Dose – How much agent will make a healthy individual ill?	Transmissibility – How does it spread from one host to another? How easily is it spread?	Host Range – How many species does it infect? Can it transfer from species to species?
What do we need to know?	**Identifying the infectious dose for humans by any exposure route will facilitate model development; animal studies are a plausible surrogate.** • Human infectious dose by aerosol route • Human infectious dose by surface contact (fomite) • Human infectious dose by fecal-oral route	**Identifying the contribution of asymptomatic or pre-symptomatic transmission is important for implementing control measures. Additionally, the relative contribution of different infection sources – fomites, droplets, aerosols, and potentially feces – are unknown.** • Capability of SARS-CoV-2 to be transmitted by contact with fomites (phones, doorknobs, surfaces, clothing, etc.) – see also Experimental Stability • Superspreading capacity needs to be refined • Updated person to person transmission rates (e.g., R_0) as control measures take effect • What is the underreporting rate?[76] • Can individuals become re-infected with SARS-CoV-2? • What is the difference in transmissibility among countries? • Is the R_0 estimate higher in healthcare or long-term care facilities? • How effective are social distancing measures? • When will infections peak in various cities and countries?	**Little is known about SARS-CoV-2 in non-human hosts.** • What is the intermediate host(s)? • What are the mutations in SARS-CoV-2 that allowed human infection and transmission? • What animals can SARS-CoV-2 infect (e.g., pet dogs, potential wildlife reservoirs)?

SARS-CoV-2 (COVID-19)	Incubation Period – How long after infection do symptoms appear? Are people infectious during this time?	Clinical Presentation – What are the signs and symptoms of an infected person?	Clinical Diagnosis – Are there tools to diagnose infected individuals? When during infection are they effective?
What do we know?	**The majority of individuals develop symptoms within 14 days of exposure. For most people, it takes at least 2 days to develop symptoms, and on average symptoms develop 5 days after exposure. Some individuals never develop symptoms, but can still transmit disease.** • The best current estimate of the COVID-19 incubation period is 5.1 days, with 99% of individuals exhibiting symptoms within 14 days of exposure.[84] Fewer than 2.5% of infected individuals show symptoms sooner than 2 days after exposure.[84] • The reported range of incubation periods is wide, with high-end estimates of 24,[68] 11.3,[12] and 18 days.[87] • Individuals can test positive for COVID-19 despite lacking clinical symptoms.[13, 38, 68, 133, 163] • Individuals can be infectious while asymptomatic,[34, 123, 133, 163] and asymptomatic individuals can have similar amounts of virus in their nose and throat as symptomatic individuals.[167] • Infectious period is unknown, but possibly up to 10-14 days [5, 88, 127] • On average, there are approximately 4[60] to 7.5[87] days between symptom onset in successive cases of a single transmission chain. • Most individuals are admitted to the hospital within 8-14 days of symptom onset.[165] **Currently, there is no evidence that recovered patients can be reinfected with SARS-CoV-2.** • Experimentally infected macaques were not capable of being reinfected after their primary infection resolved.[14] • According to the WHO, there is no evidence of re-infection with SARS-CoV-2 after recovery.[83] • Patients are positive for COVID-19 via PCR for 8-37 days after symptom onset.[165] • Individuals may test positive via PCR for 5-13 days after symptom recovery and hospital discharge.[82] The ability of these individuals to infect others is unknown. • According to the WHO, there is no evidence of re-infection with SARS-CoV-2 after recovery.[83]	**Most COVID-19 cases are mild, but severe disease can be found in any age group. Older individuals and those with underlying medical conditions are at higher risk of serious illness and death.** • The majority of COVID-19 cases are mild (81%, N = 44,000 cases)[133] • Initial COVID-19 symptoms include fever (87.9% overall, but only 44% - 52% present with fever initially[11, 68]), cough (67.7%[68]), fatigue, shortness of breath, headache, reduction in lymphocyte count.[35, 41, 73] Headache[40] and diarrhea are uncommon,[73, 87] though lack of appetite may be an early symptom.[107] • Complications include acute respiratory distress (ARDS observed in 17-29% of hospitalized patients,[44, 73] which leads to death in 4-15% of cases[44, 73, 142]), pneumonia,[106] cardiac injury, secondary infection, kidney failure, arrhythmia, sepsis, and shock.[68,73,142,165] • Approximately 15% of hospitalized patients were classified as severe,[68, 133] and approximately 5% of patients were admitted to the ICU.[68, 133] • Most deaths are caused by respiratory failure or respiratory failure combined with myocardial (heart) damage.[124] • The case fatality rate (CFR) depends on comorbidities; cardiovascular disease, hypertension, diabetes, and respiratory conditions all increase the CFR.[133, 165] • The CFR increases with age; individuals older than 60 are at higher risk of death,[133, 165] and >60% of confirmed fatalities have been male.[133] • Children of all ages are susceptible to COVID-19,[59] though generally present with milder symptoms[42, 98] or no symptoms at all.[98] Severe symptoms in children, however, are possible.[94] • In the US, 34% of hospitalizations have been individuals less than 44 years old.[4] • Variation in the CFR among countries may be due to demographics, testing criteria, and how COVID-19 related deaths are defined.[104] • Based on one patient, a productive immune response is generated and sustained for at least 7 days.[134]	**Diagnosis relies on identifying the genetic signature of the virus in patient nose, throat, or sputum samples. These tests are relatively accurate, but testing rates in the US are low compared to other countries. As a result, confirmed cases are underreported.** • PCR protocols and primers have been widely shared among international researchers.[29, 50, 87, 129, 144, 148] PCR-based diagnostic assays are unable to differentiate between active and inactive virus. • A combination of pharyngeal (throat) RT-PCR and chest tomography are the most effective diagnostic criteria (correctly diagnosing 91.9% of infections).[116] Single throat swabs alone detect 78.2% of true infections, while duplicate tests identify 86.2% of infections.[116] • Nasal and pharyngeal swabs may be less effective as diagnostic specimens than sputum and bronchoalveolar lavage fluid.[143] • RT-PCR tests are able to identify asymptomatic cases; SARS-CoV-2 infection was identified in 2/114 individuals previously cleared by clinical assessment.[72] • Combination RT-PCR and serology (antibody) testing may increase the ability to diagnose patients with mild symptoms, or identify patients at higher risk of severe disease.[162] • The FDA released an Emergency Use Authorization enabling laboratories to develop and use tests in-house for patient diagnosis.[63] • Updated tests from the US CDC are available to states.[29, 34] • US CDC has expanded patient testing criteria to include symptomatic patients at clinician discretion.[17] • Several rapid or real-time test kits have been produced by universities and industry, including the Wuhan Institute of Virology,[53] BGI,[21] and Cepheid.[140] • The US CDC is developing serological tests to determine what proportion of the population has been exposed to SARS-CoV-2.[79] • Machine learning tools are being developed to predict severe and fatal COVID-19 cases based on CT scans.[130]

SARS-CoV-2 (COVID-19)	Incubation Period – How long after infection do symptoms appear? Are people infectious during this time?	Clinical Presentation – What are the signs and symptoms of an infected person?	Clinical Diagnosis – Are there tools to diagnose infected individuals? When during infection are they effective?
What do we need to know?	**While the incubation period is well-characterized, less is known about how long individuals are infectious before, during, and after symptoms. Additionally, the possibility of reinfection warrants more research.** • What is the average infectious period during which individuals can transmit the disease? • Are individuals infectious after hospital discharge and clinical recovery, or are positive PCR tests only detecting non-infectious virus? • Can individuals become re-infected after recovery? If so, how long after?	**The true case fatality rate is unknown, as the exact number of cases is uncertain. Testing priorities and case definitions vary by location.** • How long does it take for infected individuals to recover outside of a healthcare setting? • Are reductions in CFR through time (e.g., China) an indication of better treatment, less overcrowding, or both? • Are pregnant women at greater risk of complications during labor?[91]	**In general, PCR tests appear to be sensitive and specific, though robust estimates of false positive/negative rates are still lacking.** • False positive/negative rates for tests • Eclipse phase of infection (time between infection and detectable disease) in an individual • With limited testing in many locations, how accurate are clinical diagnoses compared to genetic tests?

SARS-CoV-2 (COVID-19)	Medical Treatment – Are there effective treatments? Vaccines?	Environmental Stability – How long does the agent live in the environment?	Decontamination – What are effective methods to kill the agent in the environment?
What do we know?	**Treatment for COVID-19 is primarily supportive care including ventilation if necessary.[68, 101] A number of therapeutic trials are ongoing, but results are preliminary.[20]** • Preliminary reports from several clinical trials in China suggest that favipiravir improves lung function and reduces recovery time in COVID-19 patients.[43] • Early results suggest that tocilizumab may be effective at treating severe COVID-19 cases.[156] • Some evidence suggests that chloroquine is effective at reducing symptom duration.[1, 65] Hydroxychloroquine in combination with azithromycin may reduce viral load in patients compared to controls.[66] • Combination lopinavir and ritonavir with standard care was no more effective than standard care alone.[25] • JHU is pursuing an Investigational New Drug (IND) approval to provide passive antibody therapy (convalescent serum) to patients,[27] and Takeda Pharma (Japan) is working to create treatments based on patient plasma.[69] • Intravenous immunoglobulin (IVIg) may be effective at inhibiting deterioration when given at the appropriate time in severe cases. [26] • Corticosteroids are commonly given to COVID-19 patients[165] at risk of ARDS,[157] but their use is not recommended by the US CDC.[32] • Regeneron Pharmaceuticals has developed potential SARS-CoV-2 antibody therapies.[110] • The development of a coronavirus fusion inhibitor in the lab suggests efficacy across multiple human coronaviruses.[154] **Work is ongoing to develop a SARS-CoV-2 vaccine in human and animal trials. No preliminary results are available.** • Multiple entities are working to produce a SARS-CoV-2 vaccine,[8] including NIH/NIAID,[70, 85] Moderna Therapeutics and Gilead Sciences,[2-3, 103] and Sanofi with HHS.[22] Moderna has begun phase 1 clinical vaccine trials in humans in WA state.[120] • CEPI has partnered with multiple entities to develop vaccines including University of Oxford, Novavax Hong Kong University, and the Institut Pasteur.	**SARS-CoV-2 can persist on surfaces for at least 3 days depending on conditions. If aerosolized, SARS-CoV-2 is stable for at least several hours. The seasonality of COVID-19 transmission is unknown.** *SARS-CoV-2 Data* • SARS-CoV-2 can persist on plastic and stainless steel surfaces for up to 3 days (at 21-23°C, 40% RH), with a half-life of 13-16 hours.[138] • SARS-CoV-2 has an aerosol half-life of 2.7 hours (particles <5 µm, tested at 21-23°C and 65% RH).[138] • SARS-CoV-2 genetic material (RNA) was detected in symptomatic and asymptomatic cruise ship passenger rooms up to 17 days after cabins were vacated; the infectiousness of this material is not known.[102] *Surrogate Coronavirus data*: • Studies suggest that other coronaviruses can survive on non-porous surfaces up to 9-10 days (MHV, SARS-CoV)[28, 39], and porous surfaces for up to 3-5 days (SARS-CoV)[62] in air conditioned environments (20-25°C, 40-50% RH). • Coronavirus survival tends to be higher at lower temperatures and lower relative humidity (RH),[28, 39, 113, 139] though infectious virus can persist on surfaces for several days in typical office or hospital conditions[139] • SARS can persist with trace infectivity for up to 28 days at refrigerated temperatures (4°C) on surfaces.[28] • No strong evidence exists showing reduction in transmission with seasonal increase in temperature and humidity.[99] • One hour after aerosolization approximately 63% of airborne MERS virus remained viable in a simulated office environment (25°C, 75% RH)[111] • The aerosol survival of related human coronavirus (229E) was relatively high, (half-life of ~67 hours at 20°C and 50% RH), indicating ~20% of infectious virus remained after 6 days.[75] • Porous hospital materials, including paper and cotton cloth, maintain infectious SARS-CoV for a shorter time than non-porous material.[81]	**Soap and water, as well as common alcohol and chlorine-based cleaners, hand sanitizers, and disinfectants are effective at inactivating SARS-CoV-2 on hands and surfaces.** *SARS-CoV-2* • Twice-daily cleaning with sodium dichloroisocyanurate decontaminated surfaces in COVID-19 patient hospital rooms.[105] • Alcohol-based hand rubs are effective at inactivating SARS-CoV-2.[80] • EPA has released a list of SARS-CoV-2 disinfectants, but solutions were not tested on live virus.[6] *Other Coronaviruses* • Chlorine-based[146] and ethanol-based[49] solutions are recommended. • Heat treatment at 56°C is sufficient to kill coronaviruses,[113, 164] though effectiveness depends in part on amount of protein in contaminated media.[113] • 70% ethanol, 50% isopropanol, sodium hypochlorite [bleach, 200 ppm], and UV radiation are effective at inactivating several coronaviruses (MHV and CCV).[125] • Ethanol-based biocides are effective disinfectants against coronaviruses dried on surfaces, including ethanol containing gels similar to hand sanitizer.[74, 150] • Surface spray disinfectants such as Mikrobac, Dismozon, and Korsolex are effective at reducing infectivity of the closely related SARS-CoV after 30 minutes of contact.[112] • Coronaviruses may be resistant to thermal inactivation for up to 7 days when stabilized in stool.[135-136] • Additionally, coronaviruses are more stable in matrixes such as respiratory sputum.[61] • Hydrogen peroxide vapor was found to be effective with repeated decontamination of N95 respirators.[118]

SARS-CoV-2 (COVID-19)	Medical Treatment – Are there effective treatments? Vaccines?	Environmental Stability – How long does the agent live in the environment?	Decontamination – What are effective methods to kill the agent in the environment?
What do we need to know?	**In general, the efficacy of various therapeutic options for COVID-19 is unknown, though clinical trial results are beginning to be released.** • Is GS-5734 (remdesivir) effective in vivo (already used in clinical trials under Emergency Use Authorization)?[128] • Is the GLS-5000 MERS vaccine[158] cross-reactive against SARS-CoV-2? • Efficacy of antibody treatments developed for SARS[51, 132] and MERS[37] • What is the efficacy of various MERS and SARS Phase I/II vaccines and other therapeutics? • Are viral replicase inhibitors such as beta-D-N4-hydroxycytidine effective against SARS-CoV-2?[16]	**Additional testing on SARS-CoV-2, not surrogate viruses, is needed to support initial estimates of stability.** • Stability of SARS-CoV-2 in aerosol, droplets, and other matrices (mucus/sputum, feces) • Particle size distribution (e.g., droplet, large droplet and true aerosol distribution) • Duration of SARS-CoV-2 infectivity via fomites and surface (contact hazard)? • Stability of SARS-CoV-2 on PPE (e.g., Tyvek, nitrile, etc.)	**Additional decontamination studies, particularly with regard to PPE and other items in short supply, are needed.** • What is the minimal contact time for disinfectants? • Does contamination with human fluids/waste alter disinfectant efficacy profiles? • How effective is air filtration at reducing transmission in healthcare, airplanes and public spaces? • Are landfills and wastewater treatment plant processes effective at inactivating SARS-CoV-2?

SARS-CoV-2 (COVID-19)	PPE – What PPE is effective, and who should be using it?	Forensics – Natural vs intentional use? Tests to be used for attribution.	Genomics – How does the disease agent compare to previous strains?
What do we know?	**The effectiveness of PPE for SARS-CoV-2 is currently unknown, and data from other related coronaviruses are used for guidance. Healthcare workers are at high risk of acquiring COVID-19, even with recommended PPE.** • Healthcare worker illnesses (over 1,000[133]) demonstrates human-to-human transmission despite isolation, PPE, and infection control.[126] • Risk of transmission to healthcare workers appears high, with up to 20% of healthcare workers in Lombardy, Italy becoming infected.[115] • US CDC does not recommend the use of facemasks for healthy people. Facemasks should be used by people showing symptoms to reduce the risk of others getting infected. The use of facemasks is crucial for health workers and people in close contact with infected patients (at home or in a health care facility).[31] • "Healthcare personnel entering the room [of SARS-CoV-2 patients] should use standard precautions, contact precautions, airborne precautions, and use eye protection (e.g., goggles or a face shield)"[33] • WHO indicates healthcare workers should wear clean long-sleeve gowns as well as gloves.[145] • Respirators (NIOSH-certified N95, EUFFP2 or equivalent) are recommended for those dealing with possible aerosols[146] • Additional protection, such as a Powered Air Purifying Respirator (PAPR) with a full hood, should be considered for high-risk procedures (i.e., intubation, ventilation)[24] • Particular attention should be paid to the possibility of transmission via exhaled air during supportive respiratory procedures.[67] • Despite extensive environmental contamination, air sampling in patient rooms did not detect SARS-CoV-2 RNA[105] (but detected RNA in other rooms [96]) • The efficacy of "homemade" PPE, made with T-shirts, bandanas, similar materials, is less than standard PPE, but may be used if no other options are available.[45, 54, 117]	**All current evidence supports the natural emergence of SARS-CoV-2 via a bat and possible intermediate mammal species.** • Genomic analysis places SARS-CoV-2 into the beta-coronavirus clade, with close relationship to bat viruses. The SARS-CoV-2 virus is distinct from SARS and MERS viruses.[58] • Genomic analysis suggest that SARS-CoV-2 is a natural variant, and is therefore unlikely to be human-derived or otherwise created by "recombination" with other circulating strains of coronavirus.[9, 166] • Some genomic evidence indicates a close relationship with pangolin coronaviruses[151]; data suggests that pangolins may be a natural host for beta-coronaviruses [92-93]. Additional research is needed. • Genomic data support at least two plausible origins of SARS-CoV-2: "(i) natural selection in a non-human animal host prior to zoonotic transfer, and (ii) natural selection in humans following zoonotic transfer."[9] Either scenario is consistent the observed genetic changes found in all known SARS-CoV-2 isolates. • Additionally, "[…] SARS-CoV-2 is not derived from any previously used virus backbone," reducing the likelihood of laboratory origination,[9] and "[…] genomic evidence does not support the idea that SARS-CoV-2 is a laboratory construct, [though] it is currently impossible to prove or disprove the other theories of its origin."[9]	**Current evidence suggests that SARS-CoV-2 accumulates mutations at a similar rate as other coronaviruses. Mutations and deletions in specific portions of the SARS-CoV-2 genome have not been linked to any changes in transmission or disease severity.** • There have been no documented cases of SARS-CoV-2 prior to December 2019 • Preliminary genomic analyses, however, suggest that the first human cases of SARS-CoV-2 emerged between 10/19/2019 – 12/17/2019.[10, 19, 114] • The mutation rate of SARS-CoV-2 is estimated to be similar to other RNA viruses (e.g., SARS, Ebola, Zika), and is currently calculated to be 1.04×10^{-3} substitutions per site per year (N = 116 genomes).[71] • Preliminary phylogenetic analysis identified a very close genetic similarity between SARS-CoV-2 and a Bat coronavirus (RaTG13) isolated from Yunnan Province, China; suggesting that SARS-CoV-2 originated from bats.[108] • Pangolin coronaviruses are closely related to both SARS-CoV-2 and the closely related Bat coronavirus (RaTG13); phylogenetic analysis suggested that SARS-CoV-2 is of bat origin, but is closely related to pangolin coronavirus.[92-93] • The Spike protein of SARS-CoV-2, which mediates entry into host cells and is the major determinant of host range, is very similar to the Spike protein of SARS-CoV.[97] The rest of the genome is more closely related to two separate bat [97] and pangolin[93] coronavirus. • Analysis of SARS-CoV-2 sequences from Singapore has identified a large nucleotide (382 bp) deletion in ORF-8 that may result in an attenuated (less virulent) phenotype.[131]

SARS-CoV-2 (COVID-19)	PPE – What PPE is effective, and who should be using it?	Forensics – Natural vs intentional use? Tests to be used for attribution.	Genomics – How does the disease agent compare to previous strains?
What do we need to know?	**Most PPE recommendations have not been made on SARS-CoV-2 data, and comparative efficacy of different PPE for different tasks (e.g., intubation) is unknown. Identification of efficacious PPE for healthcare worker is critical due to their high rates of infection.** • What is the importance of aerosol transmission? What is the effective distance of spread via droplet or aerosol? • How effective are barriers such as N95 respirators or surgical masks? • What is the appropriate PPE for first responders? Airport screeners? • What are proper procedures for reducing spread in medical facilities / transmission rate in medical settings? • How effective are homemade masks at reducing transmission?	**Identifying the intermediate species between bats and humans would aid in reducing potential spillover from a natural source.** • What tests for attribution exist for coronavirus emergence? • What is the identity of the intermediate species? • Are there closely related circulating coronaviruses in bats or other animals with the novel PRRA cleavage site found in SARS-CoV-2?	**Research linking genetic changes to differences in phenotype (e.g., transmissibility, virulence, progression in patients) is needed.** • Are there similar genomic differences in the progression of coronavirus strains from bat to intermediate species to human? • Are there different strains or clades of circulating virus? If so, do they differ in virulence?

Table 1. Definitions of commonly-used acronyms

Acronym/Term	Definition	Description
Attack rate	Proportion of "at-risk" individuals who develop infection	Defined in terms of "at-risk" population such as schools or households, defines the proportion of individuals in those populations who become infected after contact with an infectious individual
SARS-CoV-2	Severe acute respiratory syndrome coronavirus 2	Official name for the virus previously known as 2019-nCoV.
COVID-19	Coronavirus disease 19	Official name for the disease caused by the SARS-CoV-2 virus.
CFR	Case Fatality Rate	Number of deaths divided by confirmed patients
PFU	Plaque forming unit	Measurement of the number of infectious virus particles as determined by plaque forming assay. A measurement of sample infectivity.
$TCID_{50}$	50% Tissue Culture Infectious Dose	The number of infectious units which will infect 50% of tissue culture monolayers. A measurement of sample infectivity.
HCW	Healthcare worker	Doctors, nurses, technicians dealing with patients or samples
SARS	Severe Acute Respiratory Syndrome	Coronavirus with over 8,000 cases in global 2002-2003 outbreak
MERS	Middle-East Respiratory Syndrome	Coronavirus with over 2,000 cases in regional outbreak since 2012
CoV	Coronavirus	Virus typified by crown-like structures when viewed under electron microscope
R_0	Basic reproduction number	A measure of transmissibility. Specifically, the average number of new infections caused by a typical infectious individual in a wholly susceptible population.
MHV	Mouse hepatitis virus	Coronavirus surrogate

CCV	Canine coronavirus	Canine coronavirus
Fomite	Inanimate vector of disease	Surfaces such as hospital beds, doorknobs, healthcare worker gowns, faucets, etc.
Droplet transmission	Sneezing, coughing	Transmission via droplets requires relatively close contact (e.g., within 6 feet)
Airborne transmission	Aerosolization of infectious particles	Aerosolized particles can spread for long distances (e.g., between hospital rooms via HVAC systems)
Transgenic	Genetically modified	In this case, animal models modified to be more susceptible to MERS and/or SARS by adding proteins or receptors necessary for infection
Intranasal	Agent deposited into external nares of subject	Simulates inhalation exposure by depositing liquid solution of pathogen/virus into the nose of a test animal, where it is then taken up by the respiratory system.
Incubation period	Time between infection and symptom onset	Time between infection and onset of symptoms typically establishes guidelines for isolating patients before transmission is possible
Infectious period	Length of time an individual can transmit infection to others	Reducing the infectious period is a key method of reducing overall transmission; hospitalization, isolation, and quarantine are all effective methods
Serial interval	Length of time between symptom onset of successive cases in a transmission chain	The serial interval can be used to estimate R_0, and is useful for estimating the rate of outbreak spread
Superspreading	One individual responsible for an abnormally large number of secondary infections	Superspreading can be caused by high variance in the distribution of secondary cases caused by a single individual; most individuals infect very few people, while some infect a large number, even with the same average number of secondary infections
Nosocomial	Healthcare- or hospital-associated infections	Characteristic of SARS and MERS outbreaks, lead to refinement of infection control procedures

ACE2	Angiotensin-converting enzyme 2	Acts as a receptor for SARS-CoV, allowing entry into human cells
ARDS	Acute respiratory distress syndrome	Leakage of fluid into the lungs which inhibits respiration and leads to death
PPE	Personal protective equipment	Gowns, masks, gloves, and any other measures used to prevent spread between individuals
PCR	Polymerase chain reaction	PCR (or real-time [RT] or quantitative [Q] PCR) is a method of increasing the amount of genetic material in a sample, which is then used for diagnostic testing to confirm the presence of SARS-CoV-2

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