Report of
LEMUEL A. MOYÉ, M.D., Ph.D.

Personal Vita:

1. My name is Lem Moyé. I am over twenty-one years of age, am of sound mind, am not a party to this action, have never been convicted of a felony, and am otherwise competent to make this affidavit.

2. I have both an M.D. and a Ph.D. degree in Community Sciences - Biostatistics. My formal training has included many courses in mathematical statistics, epidemiology, and biostatistics. My CV (attached as Appendix B) further describes my relevant experience. I have been a tenured, full professor of biostatistics at the University of Texas School of Public Health in Houston, Texas, where I held a full time faculty position in the Department of Biostatistics. I retired in March 2019.

3. I have been a licensed physician in Texas continuously since 1984, and am a diplomat of the National Board of Medical Examiners. I am also licensed to practice medicine in Arizona where I currently reside. I practiced general medicine until 1992, by which time I had seen thousands of patients. I have never been disciplined or reprimanded by the State Board of Medical Examiners, nor have I ever had my license to practice medicine suspended or revoked.

4. I worked as a physician first responder in Houston, Texas in reaction to Hurricane Katrina, in which the city received hundreds of thousands of evacuees. The rectitude and honor of these citizens who have been so beaten by the weather was illuminating.

5. **Last Position**: I have been a senior investigator of The Coordinating Center for Clinical Trials, a center within the University of Texas School Of Public Health. This research center designs, executes and analyzes clinical research efforts.

6. **Research Experience**: I carried out cardiovascular research for thirty-two years designing, executing and analyzing clinical trials. I have been Principal Investigator on a grant from Schering-Plough and have been Co-Principal Investigator on two grants from Bristol Myers-Squibb. In these studies, I was responsible for the design, execution, and analysis of large scale clinical trials. In one case, the experiment involved over 2000 patients who were followed for 3.5 years, and in the other case, 4159 patients were followed for five years. In each of these enterprises, an important component of my responsibility was the reporting of adverse events from physicians. In one of these trials, I supervised the collection of adverse events that were reported to both the Sponsor and to the Federal Food and Drug Administration (F.D.A.). Each of these clinical trials has resulted in several articles that were published in the Journal of the Medical Association and *The New England Journal of Medicine* (1992 and 1996), as well as other journals in the peer reviewed medical literature.

7. I have also served as the Coordinating Center Principal Investigator and was in charge of the biostatistics group carrying out research with neurologists on the treatment of strokes, funded by the National Institute of Neurological Disorders and Strokes.

8. **Last Activities:** I was the Coordinating Center Principal Investigator for the NHBLI funded Cardiovascular Cell Therapy Research Network (CCTRN) from 2007-2019

when I retired. This National Heart, Lung, and Blood Institute funded Network designs, executes, and analyzes clinical trials to assess the role of cell therapy in heart failure, acute ischemia, and peripheral vascular disease. This particular research effort has generated over twenty manuscripts in the peer reviewed literature, including three in the *Journal of the American Medical Association (JAMA)* (2011, 2012, 2012) and one in Circulation, the Journal of the American Heart Association.

9. I have also taught every year since 1987 in biostatistics, epidemiology, and public health, advising M.P.H., M.S., and Ph.D. graduate students. I have also reviewed research grants for NIH and have given many guest lectures (curriculum vitae).

10. **Manuscripts:** I have published over two hundred and twenty manuscripts in the peer-reviewed literature that reflect my experience in the design, execution and analysis of clinical research. Included among these manuscripts are papers that provide the mathematical development of tools that improve both the design and the execution of both small and large-scale clinical trials. Specifically, I have been an Investigator on four grants from the National Institutes of Health, involving the design, execution, and analysis of clinical trials and epidemiologic studies. Each of these has led to publications in the peer-reviewed literature. I have presented papers at the International Joint Statistical Meetings.

11. I have published 10 books, nine in probability and statistics and 1 novel. The pertinent ones for this report follow:

12. My second book, entitled *Difference Equations with Public Health Applications*, co-authored with a colleague, was published by Marcel Dekker in October 2000. This book examines the role of difference-differential equations in health care. Several chapters of this book are relevant here as they derive from first principals the mathematical models for the spread of coronavirus. Specifically, pathogens such as viruses are known to be introduced into a community either by 1) an individual with the disease enters the community, 2) an individual with the virus, already in the community, spreads the virus to others in the community. Similarly patients with the virus can decline if 1) they are cured or die, and 2) the leave the community. The equations governing this activity are derived in this text.

13. In 2005, having learned of the destruction wrought in New Orleans by Hurricane Katrina, and the 700,000 New Orleans citizens forced to evacuate the city and come to Houston, I served as a physician first responder to address the critical needs of this population that was arriving in bus caravans. I was charged with the care of men, women, and children who were malnourished and dehydrated. For the first 36 hours of the experience, we had no running water, medicines, and few bandages. There was no x-ray equipment available and no medical instruments, forcing us to rely on what we brought ourselves. Based on that experience, and my interactions with the New Orleans citizens I met there, I wrote a book entitled I wrote *Caring for Katrina Survivors: A First Responder's Tribute* (Open Hand Press). This book has won five awards including the 2007 Ben Franklin Award for Best Multicultural Book of the Year.

14. **Expertise in Epidemiology and Public Health**,
As a practicing physician, I have treated patients with viral diseases caused by adenovirus, rhinoviruses, picarnovirus, and coronaviruses, hospitalized and non-hospitalized. I have treated patients with influenza including H1N1. I have treated

patients hospitalized with coxsackie viruses (i.e., myocarditis. I have treated patients suffering from rubeola, rubella, herpes simplex type I and II, and the paramyxovirus.

15. I have trained in epidemiology for 34 years. I have learned the anatomy of viruses and their pathology. I have learned of the major vectors (mosquito, animal) through which viruses attack humans. I have written specifically about the spread of contagion (bacterial, viral from human to human. Specifically in my text, *Difference Equations in Public Health*.[1] I motivated and derived in great detail and specificity the Sydney Chapman, and Andrey Kolmogorov difference-differential equations for the contagion, model, the contagion-death model, and the contagion emigration-death models. These computations are the basis of the models contemporaneously used into predict SARS-Cov-2 cases in communities, cities, counties, and states.

16. I have taught, practiced and trained in public health since I earned my MD degree in 2008. I have taught courses in public health for 31 years. I have given lectures to universities, schools of public health, pharmaceutical companies, the National Institutes of Health, the federal Food and Drug Company. I have responded to public health calls to action as was a first responder to the arrival of 700,000 New Orleans citizens.

I have the education, training, knowledge, experience, and expertise to opine on this matter involving SARS-CoV-2 and the disease COVID-19 before the court.

17. **Education:** My education consists of the following:

Year	Degree	School
1987	Ph.D. Community Sciences (Biometry)	University of Texas
1981	M.S. Statistics	Purdue University
1978	M.D.	Indiana University
1974	B.A. (mathematical sciences)	Johns Hopkins University

18. **Fees**: I currently am being retained by Gauthier, Murphy and Houghtaling LLC, Metairie, Louisiana, in this matter. My fees are $400 per hour for review of the literature, documents, and depositions. My fees are $500 per hour for deposition and trial testimony.

19. During the previous four years, I have been deposed as an expert witness in the following cases:

- *ELI LILLY AND COMPANY and ICOS Corporation. ACTAVIS LABORATORIES UT, INC., et al*., C.A. No. 1:16-cv-01119-AJT-MSN 2017
- *Sylvester Hebron et al vs. Abbvie Inc. and Abbott Laboratories Case No. 142-CC09740-01.Irwin Chaiken vs. Bristol-Myers Squibb Company et.* a. Civil Action No.:3.13-CV-04518-FLW-TJB 2017
- *Mirena Products Liability Litigation for the US District Court Southern District of New York, 2018*
- *Daniels-Feasel et. al. vs. Forest Pharmaceuticals Inc., et. al. US District Court Southern District of New York Case No 1:17-cv-04188 (LTS)(JLC)*
- *Pradaxa October 18, 2019*

- CHRISTOPHER TREJO, vs. JOHNSON & JOHNSON, et al., SUPERIOR COURT OF THE STATE OF CALIFORNIA FOR THE COUNTY OF LOS ANGELES YC058023, October 2019.

20. **Methodology:** I come to this topic of the nature of the environmental impact of SARS-CoV-2 with no *a priori* bias. To draw my conclusions, I relied on my training in medicine, my experience seeing and treating patients, my advanced training in epidemiology and biostatistics, my public health expertise, and my clinical research expertise as applied to my review of the literature. My research on this information followed the standard process of scientific inquiry. Determining whether SARS-CoV-2 impacts its surrounding environment is an exercise in critical thinking, using the "weight of the evidence" approach.

21. This "weight of the evidence" approach is the process by which a body of evidence is examined piece by piece, each component being sifted and assessed using a transparent and standard methodology. In this case, the goal is assess the relationship between SARS-CoV-2 and its environment.

22. To examine the germane question in more detail, I identify the collection of peer reviewed published papers, reflecting the universe of useful information about the state of the relationship between SARS-CoV-2 and its environment. This study-by-study review first details the study information and then distills it.

23. Following this process, I first identify the lines of evidence created in each study, assessing the methodology used by the research effort. Since the value of a research result is tightly circumscribed by the methodology used to generate it, research methodology is critical. Each manuscript is assessed on its merits. It is the rare manuscript that provides contributions to all aspects of the SARS-CoV-2/environment relationship.

24. As each manuscript is absorbed, its contributions to the SARS-CoV-2/environment relationship are assessed. This noetic approach of evidence gathering, objective reviews, and deductive reasoning, and critical reasoning has demonstrated its success repeatedly.

25. **Virology**: Virology is the study of the existence and effects of viruses. Viruses are unique in nature because they have one but only one property of life – they use nucleic acids to reproduce. Viruses are composed of one or two strands of a weak acid (either ribonucleic acid (RNA) or deoxyribonucleic acid (DNA)) surrounded a protein-lipid coat.

26. The only known function of viruses is to reproduce. They are not self-propelled but travel through the air, or water, or across solid objects to rest on a surface waiting for, and in order to be absorbed into a cell. Sometimes the spread is by injection through a violent (rabies) or relatively nonviolent (yellow fever, dengue fever). They can also spread by ingestion of fish (pancreatic necrosis virus, and Aquabinarovirus). [2] Commonly the spread is by inhalation, or tactile transfer of the virus from the surface to the eye, nose, or mouth.

27. Viruses infect many species of organisms. They infect birds (papovavirus, Budgerigar fledgling disease, psittacine polyomavirus), nonhuman mammals (herpes virus in

gorillas, parvo and rabies canine parvovirus in dogs, as well as ferrets, skunks, and raccoons), insects (phlebovirus in sandflies [3],) and fish [2]. Viruses even infect bacteria (bacteriophage) [4]

28. Viruses are – quite literally – unbelievably small. Ronald Reagan once famously compared dollars to the distance to the moon. In fact, if dollar bills were stacked on top of each other, it would take just over $3.5 trillion to get a stack high enough to reach the moon[*]. 3.5 trillion SARS-CoV2 viruses would only be 273 miles[†]. There can be more viruses in a cubic inch of water than there are stars in our Milky Way.[‡] However, despite its small size, a virus is tangible and physical. It leaves a physical presence that physically interacts with the air and solid surface.

29. **Coronaviruses:** Coronaviruses are members of family Coronaviridae that include enveloped positive sense ssRNA containing viruses, taxonomically classified amongst order Nidovirales in the realm Riboviria. They have been on the earth for thousands of years[§]. Coronaviruses infect mammals, e.g., bats, dogs and cats), birds and amphibians [5], and of course humans.

30. Like other viruses, they do not have the machinery to survive outside a living host cell and therefore cannot replicate outside of a host. [6] The riboviruses (or realm Riboviria) members replicate by utilizing RNA-dependent RNA-polymerases (RdRps). Their genetic material (RNA) serves as messenger RNA (mRNA), and is directly translated into proteins, that enwrap the original RNA. Thus, the virus can enter a cell, reproduce hundreds if not thousands of times, and emerge from the dead cell to infect other susceptible cells.

31. In addition, the coronavirus RNA, once in the cell, can have its RNA mixed with the RNA of another virus that itself is infecting the same cell (genetic recombination). Thus, the virus mutates and evolves through a series of cell infections, increasing the difficulty of tracking it and protecting against it.

32. **SARS-CoV-2** Severe Acute Respiratory Syndrome Coronovirus2 (SARS-CoV-2) has an origin in bats and jumped to humans in 2019 and is the cause of coronary virus infectious disease (Covid-2019) [7].

33. The SARS-CoV-2 virus is 125 nanometers[**] in diameter. The outer shell of the virus (the pericapsid) consists of three structural glycoproteins[††]: spike, envelope, and membrane. These three S glycoproteins attached to a lipid coating, aggregate to form a homotrimer which when they appear in numbers on the pericapsid, give the virus its crown like appearance, producing the moniker Coronavirus [8]. It is this spiked surface that comes in contact with the nonviral outside world.

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[*] The moon is 3.84×10^8 meters from earth. A dollar bill is 1.092×10^{-4} meters thick. This produces $\$3.5 * 10^{12}$.

[†] The diameter of a SARS-COV-2 virus is $1.25 * 10^{-7}$. $(3.5 \times 10^{12})(1.25 \times 10^{-7}) = 4.375 \times 10^5$. There are 1.609×10^3 meters in a mile, so this many SARS-CoV2 viruses is $(4.375 \times 10^5.)/(1.609 \times 10^{-3} = 2.719 \times 10^2 = 271.9$ miles.

[‡] A cubic inch of water is sphere with contains $(4/3)(3.14159)(1.27 \times 10^{-2}\text{m})^3 = 8.59 \times 10^{-6}\ \text{m}^{-3}$. A single SARS-COV-2 virus has a volume of $(4/3)(3.14159)(0.625 \times 10^{-7}\text{m})^3 = 1.023 \times 10^{-21}\ \text{m}^{-3}$. The ratio of volumes is 8.39×10^{15}. Our galaxy, the Milky Way, has 500 billion stars or 5.0×10^{11}.

[§] Estimates vary widely from 8,000 to 300,000,000 years. Since mankind first appeared 150,000 years ago, the coronavirus either appeared before or after his presence.

[**] A nanometer is a billionth of a meter.

[††] A glycoprotein is a long chain of amino acids to which short chained sugar moities are attached. A proteoglycan is a short protein to which long chains of sugar molecules attach.

34. The large Spike protein (S), which sits on and protrudes from the outside layer, is made up of two different domains or sections. S1 and S2. The S1 domain, contains an area which allows the binding of the virus to human cells. Specifically, this receptor binding domain binds to the extracellular peptidase domain of angiotensin I converting enzyme 2 (ACE2). This is the key to its ability to enter the cell. In addition, SARS-CoV-2 also requires a specific enzyme (transmembrane serine protease) to gain entry to the cell. The interior of the virus contains a single stranded, positive sense RNA virus.[9] and is large for a virus that uses RNA to replicate, with one of the longest genetic sequences of any RNA producing viruses (30,000 genetic bases).[10]

35. **Illness:** COVID-19 infection is commonly associated with febrile mild respiratory illness with myalgia, arthralgia, and coryza, and curiously, anosmia, or loss of smell. In many cases there is pneumonia, with some patients developing an acute respiratory distress syndrome (ARDS). In these cases, blood biochemistry measures of albumin, lactate dehydrogenase, C-reactive protein, blood cell differential indicate the disease severity. [11] Middle-aged and elderly patients with primary chronic diseases, especially high blood pressure and diabetes, have been found to be more susceptible to respiratory failure and, therefore, had poorer prognoses.

Providing respiratory support at early stages improved the disease prognosis and facilitated recovery. The ARDS in COVID-19 is due to the occurrence of cytokine storms that results in exaggerated immune response, immune regulatory network imbalance, and, finally, multiple-organ failure.

36. A suspected case of COVID-19 infection is said to be confirmed if the respiratory tract aspirate or blood samples test positive for SARS-CoV-2 nucleic acid using RT-PCR or by the identification of SARS-CoV-2 genetic sequence in respiratory tract aspirate or blood samples. [12]

37. **The Experience of Louisiana and New Orleans.** On March 6th the State of Louisiana Department of Health issued a proclamation that COVID-19 was a reportable disease. [13] On March 9, 2020, Governor John Bell Edwards stated in a declaration that the first case of presumptive positive COVID-19 occurred in a Jefferson Parish resident. On March 11, with COVID-19 effecting at least 13 residents in Louisiana, the governor declared a public health emergency, stating "the state of Louisiana has reason to believe that the COVID-19 may be spread amongst the population by various means of exposure, therefore posing a high probability of widespread exposure and significant risk of substantial future harm to a large number of Louisiana citizens". [14] This coincided with a state of emergency for the City of New Orleans being declared by its Mayor, LaToya, Cantrell.[15]

38. On March 13, the governor declared that all gatherings of greater than 250 people were banned, and closed on K through 12 level public schools. The week of March 17th, after 1 death and 3 cases in Louisiana, he focused on the New Orleans area, ordered all New Orleans restaurants and bars to shut down.

39. On March 16, 2020, Mayor LaToya Cantrell issued a "Proclamation to Promulgate Emergency Orders During the State of Emergency Due to COVID-19" providing as follows:

> "There is reason to believe that COVID-19 may be spread amongst the population by various means of exposure, including the propensity to spread person to person and the propensity to attach to surfaces for

> prolonged periods of time, thereby spreading from surface to person and causing property loss and damage in certain circumstances
>
> On March 9, 2020, the first presumptive positive case of COVID-19 was announced in the State of Louisiana, is a hospital in the City of New Orleans"

40. On March 22, 2020, Governor John Bell Edwards issued an order with "Additional Measures for COVID-19 Stay at Home" which provided that "these measures relating to closure of certain businesses and to limit the operations of non-essential businesses are necessary because of the propensity of the COVID-19 virus to spread via personal interactions and because of physical contamination of property due to its ability to attach to surfaces for prolonged periods of time" [16].

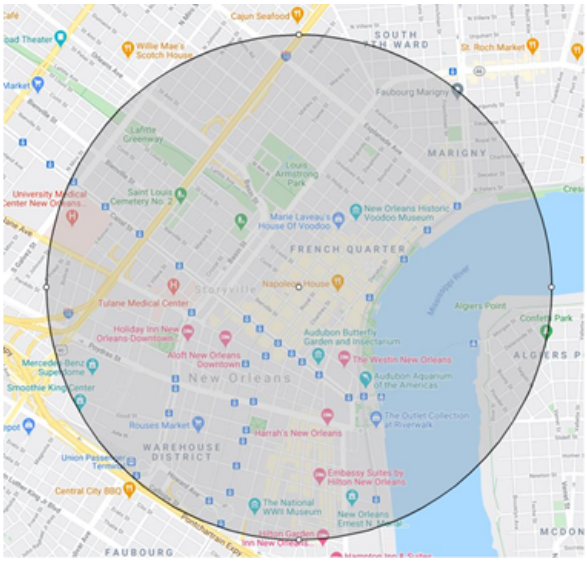
41. Cases peaked at 2728* on April 2, progressively declined to 200 on May 3, then increased to 3840 [17] on July 26.

42. On May 15th, Mayor LaToya Cantrell of New Orleans issued a proclamation that a) cancelled all public and private, nonemergency gatherings, b) all bars, health clubs, shopping malls, live performances, reception facilities, and other establishments where large crowds would gather, 3) limited all restaurants to take out and delivery only. Transitions toward normal conduct of operation would be phased. [18]

43. As of July 1, 2020, 84,241 cases were observed. It is estimated that there were 22,194 excess cases (1649 deaths) due to facilities opening before the mask mandate, and 2,428 excess cases (351 deaths) attributable to opening after the mask mandate.[19]

44. For the second time since the start of the coronavirus pandemic and in response to a surge of cases, many businesses were shuttered completely on July 24, 2020 by Mayor LaToya Cantrell as officials bracing for increased strain on hospital resources.[20] The new order, combined with statewide rules prohibiting bars from serving patrons on their premises, was essentially a full shutdown of the city bars, placing additional strains on restaurants. The mayor stated that the restrictions were particularly aimed at Bourbon Street and other areas of the city where alcohol-fueled gatherings have "gotten out of control."

* Three are a set of caveats that accompany these numbers. Cases include confirmed and probable, and are not uniformly reported seven days a week, which lead to artifactual clustering as cases that have accumulated over seven days may be reported as a combined set.



45. On September Gov. Edwards released details of his proclamation moving Louisiana's response to COVID-19 to Phase 3. [21] The new order expired on October 9. This order permitted restaurants, churches, salons, spas, gyms and other businesses will be able to open at a maximum of 75 percent of their occupancy, with social distancing in place. However, bars will remain closed to on-premises consumption in parishes with high incidence of COVID as evidenced by their test positivity rate. When re-opened, bars were able to open at 25 percent capacity, up to 50 people, indoors for customers seated for tableside service. They may have no more than 50 customers outdoors, socially distanced, seated for tableside service. No live music was be allowed.

46. The Oceana Grill was affected by the shutdown as it was the geographic zone impacted by the governor and New Orleans's mayor's declarations. In fact, its specific location on Bourbon and Conti Streets was located at one of the epicenters of the COVID19 outbreak. Besides being a bar and restaurant, patrons hold cocktail parties and French Quarter weddings at this venue [22].

47. The Oceana Grill had patrons who had SARS-CoV-2 infection. Specifically, the owner tested positive for COVID-19. In addition, there were four other instances where individuals with property access reported a positive SARS-CoV-2 positive test. These were two office employees, a maintenance worker, and prospective employee interviewing for a position. Based notice of the change in the property condition from safe to dangerous, portions of Oceana Grill were close for 24 hours. The dates of these actions on or about April 3, April 10, June 3, June 27, and August 1, 2020. [23]

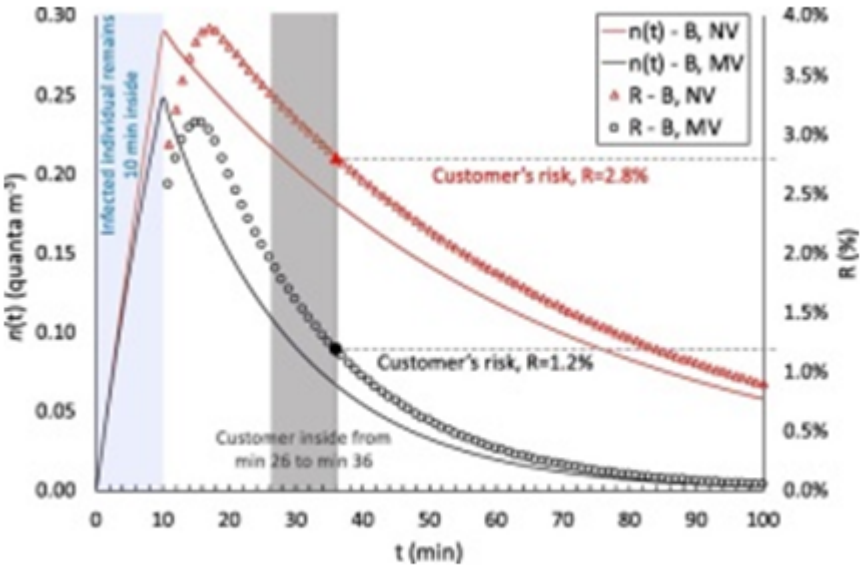
48. **Virus dispersion**. Airborne transmission is the dominant route of SARS-CoV-2 spread. The virus is transmitted mainly via small respiratory droplets containing the viral particles that the infected person exhales when coughing, sneezing or just talking. The amount of virus released by the infected individual increases as the infection progresses.

49. During the pre-Covid-19 period of time, the average guest count was 1,583 per day. During Phase 1, this dropped to less than 100 patrons per day. During Phase 2, this increased to 667, and in Phase III it was an average of 690 patrons per day. If just one patron per day is infected with SARS-CoV-2, and that patron sneezed once every half hour for four hours, then that individual has released two billion viral particles that will settle on the head, face and shoulders of patrons, and on the exposed surfaces of bars, tables, chairs, and drinking glasses. This is enough to infect others in the Oceana

Grill. With more patrons infected, the infectivity of the restaurant grows.

50. Given the established infectivity of SARS-CoV-2 and the ongoing customer patronage level of Oceana Grill, the degree of environmental exposure (air and surface) at the restaurant rose to dangerous levels. The restaurant's environment was transformed into a deleterious condition, as the virus physically transformed the air and the restaurant contents from one of safety to one of infectivity and illness. This transformation changes the structure of the surface of the restaurant contents, by a process predicted by physical law. The change in the structure is the damage so the transformation more likely than not leads to physical damage.

51. **Virus is primarily spread through the air:** The airborne dissemination of the virus was quantitated by Buonanno.[24] The figure demonstrates the quanta concentration (n(t)) and infection risk(R) trends as a function of time for two different exposure scenarios



From Buonanno G, Stabile L, Morawska L. Estimation of airborne viral emission: Quanta emission rate of SARS-CoV-2 for infection risk assessment. *Environ Int*. 2020;141:105794. doi:10.1016/j.envint.2020.105794

52. This figure demonstrates the simulated relationship between quanta of virus and time as a customer enters and then remains in an establishment (in this case, a pharmacy). As the infected customer remains, there is an increase in the environmental viral load. The longer the customer stays, the greater the number of viruses that customer sheds into the environment. Then, moving along the abscissa, as new, unexposed customers arrive in the establishment, their risk of exposure is considerable, being greater in an environment with natural ventilation then mechanical ventilation.

53. **The risk for infection is worse in poor ventilation.** The prior simulated result demonstrated high infectivity in pharmacies, supermarkets, restaurants, post offices, and banks. The infectivity in restaurants was greater due to the comingling of many people. While the infectivity is reduced in the presence of mechanical ventilation, it is still elevated.

54. Viruses spread by other means as well. For example, highly sensitive laser light scattering observations have revealed that loud speech can emit thousands of oral fluid droplets per second. Therefore, in an environment of stagnant air, droplet nuclei

generated by speaking will persist as a slowly descending cloud emanating from the speaker's mouth, with the rate of descent to services determined by the diameter of the dehydrated speech droplet nuclei [25]

55. **SARS-CoV-2 spread by air and though surface interactions**. Covid-19 is a contagious disease caused and distributed by the SARS-CoV-2 virus. This contagious disease is spread between individuals either through airborne delivery or from touching surfaces and delivering the virus to the eyes, nose, or mouth by hand. Thus, the virus transforms the air by making it physically and palpably infectious and transforms the surfaces where they land to a source of disease. This transformation is achieved by the interactions of molecules on the virus with molecules on the surfaces that compel them to link up, one to the other. The virus also alters the surface of solid objects (bars, tables, chairs, drinking glasses through the process of molecular cohesion and adhesion. This insinuation of the virus into and through the surface of the solid objects alters the surface to a wood-wax-virus hybrid that cannot be disassembled, separating the virus from the original surface. This is the physical damage to the surface.

56. **Molecular cohesion and adhesion:** Molecular cohesion is the process by which like molecules combine due to their electrostatic attraction. Examples would be surface tension. Here, the water molecules on the surface water in a glass, 1) combined together at the surface and 2) are pressed upward by the water molecules below, create a tension on the surface. This surface can be so great that insects can walk across it and sewing needles can rest on the surface.[*] Adhesion is the attraction of molecules to other, different molecules. For example, the molecules in a glass of water are electrostatically attracted to themselves (cohesion) but are also attracted to the molecules in the glass itself (adhesion).

57. **SARS-CoV-2 Adhesion**: The coronaviruses encode at least four major structural proteins, including spike glycoprotein, envelope protein, membrane protein (222aa), and nucleocapsid protein (419aa) [11]. Spike glycoprotein is an important structural protein of the coronavirus. It is a homologous trimer protein on the surface of the viral envelope involved in the process of receptor binding and membrane fusion. The S1 spike binds to anticonverting enzyme receptors (that already exist) on the host cell. S2 is responsible for fusing with the host cell membrane, permitting the virus entry to the interior of the cell. [26]

58. Thus, the virus takes advantage of attachment. If the virus is on a human cell, it attaches and attempts to push through the cell's membrane. When the SARS-CoV-2 virus is driven down by gravity and micro air currents to a bar surface, the S1 S2 projections adhere to the surface molecules of the bar attempting the chemistry to "infect" the bar. However, since infection fails, (since there is no membrane for the virus to break through), the virus simply stays adhered to the bar surface.

59. Adhesion is a physical attachment. The virus is physically attached to the bar, held there by submolecular adhesive forces (e.g., ionic bonds, covalent bonds, Van der Waals forces, and London forces, i.e., attractions between electrons and protons

[*]Surface tension is also the reason why washing hands with only water is not very effective. The flowing water cannot break the surface tension of the water molecules on our skin. The use of detergents breaks this surface tension, permitting deeper cleaner.

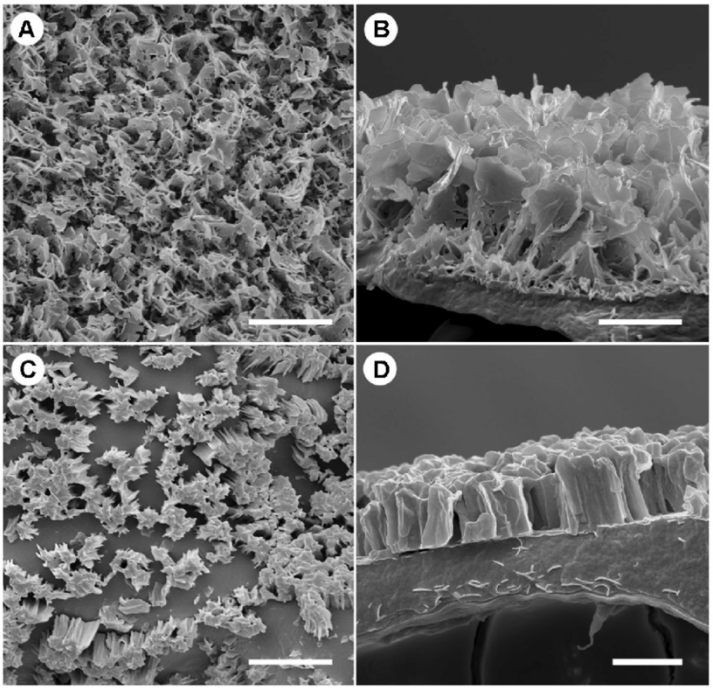
across different atoms and molecules).

60. The viral-bar adhesion may ultimately be broken by a microcurrent of air that is strong enough to break the chemical bonds between the virus and the bar, or by the use of a cleaning agent which denatures the virus, eradicating its protein coat, radically changing and destroying its molecules and thereby break the adhesion.

61. **Viruses, Dust, and Bars:** One could be forgiven for thinking initially that since dust is easily swept away by dusting, so too, viruses, also small particles, can also be dispersed. There are two reasons why this dust analogy fails: 1) size, and 2) nature of the attraction of the surface for the particle.

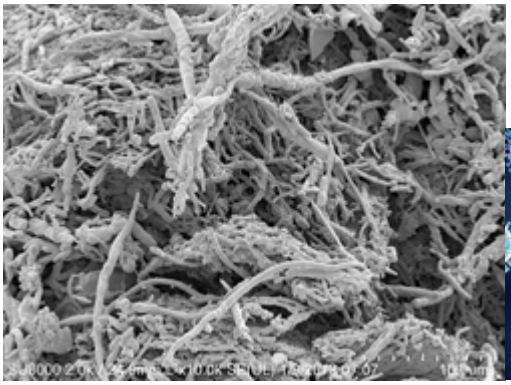
a. Size. It is easy to think of a well varnished, highly polished bar as smooth. That is an illusion.

Well-polished bars, when examined at the microscopic level are rough and craggy with deep valleys in which small particles can nestle.

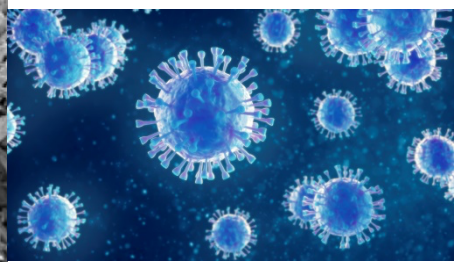


Particles that are big, rest on the top of these irregularities. Particles that are small, can nestle deeply within these crags and valleys.

Dust particles are much larger than viruses. Dust particles are 500 um in size. Compared to viruses (the SARS-CoV-2 virus is up to 4000 times smaller than a dust particle), dust particles are huge. Dust particles are much too big to fit in between the ridges and in the valleys of the surface of a "smooth" bar. SARS-CoV-2 viruses with their small size can easily slide into these irregularities.



Dust measured in micrometers



SARS-CoV-2 in nanometers

The second issue is the nature of attachment between the particle and the surface. Dust particles are heterogeneous containing collections of textile, paper, and mineral fibers. These particles create bonding by exchanging electrons with other atoms (ionic bonding).* So, in order to bond, it must find elements that are willing to engage in electron exchange. There are no such elements in a polished bar, or chair, or table. They are instead composed of organic compounds that do not exchange electrons but share them (covalent bonds). Thus, dust does not bond to the wax,

* For example, sodium and chloride exchange electron in an ionic bond that creates salt crystals.

However, SARS-CoV-2 is comprised of organic molecules and, just like wax on a bar top, is capable of sharing electrons. Covalent bonds are created between the virus and the furniture's wax. Thus, viruses due to their small size and covalent bonding, insinuate themselves in, and adhere to the bar top, making them difficult to remove. Dust, being larger and with no bonding, simple sits on top of the bar top and can be brushed off.

62. However, we must always keep in mind that viruses are not just in a dynamic environment where they are shed and land on a surface, e.g. a bar where they stay fixed. Viruses create and are caught up in a hyperdynamic environment where they are constantly being shed, landing on other individuals then landing on surfaces where the ones that stay on the superficial surface are lifted up into the air again where they may land on another surface.

63. It best to think of viruses as a continuous, turbulent river of infectious particles, being fed by the talking, coughing, sneezing infectious individuals whose viruses, ejected by the hundreds of billions, move from individual to individual, or to the air where they linger, or to surfaces where they land, and then back into the air. The entire, breathable, touchable environment is physically impacted and infected by this noxious current and transformed.

64. **Exhaled particle sizes**. While aerosols arising from human activity are heterogeneous in particle size [27], measurements show that most exhaled particles are small, (below 5 μm). There are two types of particles. in the exhaled gas, i.e., round and faceted types, based on particle shapes. The concentration of particles shows large variability across different individuals, with the presence of one potential "super emitter" out of eight participants. This ratio (12.5%) coincides approximately with the 10% of superspreading individuals for COVID-19 [28].

65. **What does it take to restore the unhealthy environment.** Van Dormelen evaluated the stability of SARS-CoV-2 and SARS-CoV-1 in aerosols and on various surfaces, estimating their decay rates. The research consisted of 10 experimental conditions involving two viruses (SARS-CoV-2 and SARS-CoV-1) in five environmental conditions (aerosols, plastic, stainless steel, copper, and cardboard). In fact, the SARS-CoV-2 remained viable in aerosols throughout the duration of our experiment (3 hours), This reduction was similar to that observed with SARS-CoV-1, from $10^{4.3}$ to $10^{3.5}$ $TCID_{50}$ per milliliter [29]

66. In addition to the direct airborne transmission of SARS-CoV-2 other routes, such as fecal excretion has contributed to the SARS-CoV-2 transmission and spread [30]. This route raises important concerns focusing on the spread of the virus in public restrooms.

67. **SARS-CoV-2 stability:** Chin reported on the stability of SARS-CoV-2 in different environmental conditions. The authors determined that the virus is highly stable at 4°C, but sensitive to heat. They further investigated the stability of this virus on different surfaces. No infectious SARS-CoV-1 could be recovered from printing and tissue papers after a 3-hour incubation; no infectious virus could be detected from treated wood and cloth on day 2. By contrast, SARS-CoV-2 was more stable on smooth surfaces. No infectious virus could be detected from treated smooth surfaces on day 4 (glass and banknote) or day 7 (stainless steel and plastic). Strikingly, a detectable level of infectious virus could still be present on the outer layer of a surgical mask on day 7.

68. They also tested the virucidal effects of disinfectants. With the exception of a 5-min incubation with hand soap, no infectious virus could be detected after a 5-min incubation at room temperature (22°C). However, the investigators also found that the SARS-CoV-2 is extremely stable in a wide range of pH values at room temperature (pH 3–10); In addition, the investigators found that while SARS-CoV-2 is susceptible to standard disinfection methods, overall, it can be highly stable in a favorable environment.[31]

69. This is a critical finding. If there was no poisoning or physical change in the environment, one might expect that no restoration of the environment would be required. To what is the environment being restored, it was unchanged prior to the restoration? However, SARS-CoV-2 does change the environment and as Chin pointed out, transforming it to infectious stability i.e., the environment remains noxious and static for days. Only restoration with specific treatments will restore the environment. Physical remediation in these circumstances is the biochemical cleaning of all surfaces to denature the billions of viruses that have adhered to them.

70. Riddle et al [32] studied environmental stability of SARS-CoV-2. They measured the risk of transmission from contaminated surfaces. All experiments were carried out in the dark, in order to negate any effects of UV light. Inoculated surfaces were incubated at 20 °C, 30 °C and 40 °C and sampled at various time points. With initial viral loads broadly equivalent to the highest titers excreted by infectious patients, viable virus was isolated for up to 28 days at 20 °C from common surfaces such as glass, stainless steel and both paper and polymer banknotes.

71. These findings demonstrate SARS-CoV-2 can remain infectious for significantly longer time periods than generally considered possible. These results could be used to inform improved risk mitigation procedures to prevent the fomite spread of COVID-19. This is significantly longer than that reported by Chin (*vide supra*) The SARS-CoV-2 virus does alter the environment including the air and surfaces.

72. Based on 1) five cases of SARS-CoV-2 positive cases involving the owner and employees of Oceana Grill and 2) the number of "hot spots" within the French Quarter and 3) the propensity of SARS-CoV-2 to disseminate airborne, it is more likely than not that based on the concentration of cases that SARS-CoV-2 was continuously in and around the restaurant for a significant period of time and caused physical damage to the property by its molecular binding to the Grill's inanimate surfaces, producing a continual contamination of the property.

73. Oceana has attempted to restore the pre-CoVid-19 environment by maneuvers e.g., airing out the property, generally cleaning surfaces with bleach-based cleaners, and closing off rooms where individuals who reported COVID-19 were located for 24 hours. While this effort is understandable, it is inadequate because 1) the effort was undone by the continued arrival of SAR-COVID-2 positive patrons and employees, and 2) the recent research of Riddle (vide supra) reveals that the virus is viable for up to 28 days and perhaps longer, invalidating the process of cordoning off a room for 24 hours.

74. The physical damage to the property at Oceana is at a molecular level. Based on five individuals who were discovered to be positive for SARS-CoV-2 from April 3 to August 1 2020, including the owner and several employees of Oceana (*vide supra*) several cases confirmed by the owner and employees of Oceana and the number of "hot spots" within the French Quarter, it's more likely than not COV2 was in and around the restaurant for a significant period of time and caused physical damage to

the property by binding to and penetrating surfaces. This is clear physical damage to property.

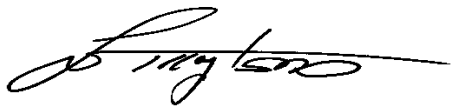
75. **Conclusions:** The SARS-CoV-2 virus is highly infectious. During its existence, the virus does physically alter the air and any surface the air or an infected human contact. This is a physical alteration, governed by the laws of chemistry, physics, and molecular biology. These chemical and physical forces determine the virus's ability to adhere to surfaces including but not limited to floors, desks, tables, chairs, stools, bottles, bar tops and sides, music equipment, microphones. This adherence cannot be seen with the naked eye because it occurs at the molecular level.

The result is a dynamic process, creating a dangerous physical condition. Specifically, this process is 1) inhabitance of the air by SARS-CoV-2, 2) transport by the air of SARS-CoV-2 to surfaces, and 3) adherence of the virus to those surfaces, rending those surfaces infective. The surfaces are damaged by the by the virus's physical contact with them.

During the course of an evening, hundreds of millions of viral particles are landing on, adhering to, and being lifted from physical surfaces. This river-like flow of infectivity converted the Oceana Grill into a dangerous and infectious nest. This is a dangerous physical condition that can only be reversed by physical restoration involving chemical resurfacing. Physical damage took place, requiring intense physical remediation. Therefore, within a reasonable degree of scientific certainty:

A. It is more likely than not that SARS-CoV-2 viral particles were in or within 1 mile of Oceana Grill
B. It is more likely than not that SARS-CoV-2 viral particles caused a property loss or damage in or within 1 mile of Oceana Grill
C. It is more likely than not that SARS-CoV-2 particles caused a property loss or damage in or within 1 mile of Oceana Grill for an extended period of time (over 72 hours) due to continuous contamination
D. It is more likely than not that SARS-CoV-2 caused a dangerous physical condition within 1 mile of Oceana Grill
E. It is more likely than not that the Mayor and Governors reasoning that SARS-CoV-2 attaches to surfaces; (2) contaminates surfaces; and (3) causes property loss and damage was scientifically supported

76. I will be working with a technician on an animated depiction of some of the issues raised in this report.



Lem Moyé, MD, PhD – 10-16-2020

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