Measures do not prevent deaths, transmission is not by contact, masks provide no benefit, vaccines are inherently dangerous: Review update of recent science relevant to COVID-19 policy

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Summary

The unprecedented measures of universal lockdowns, tight institutional lockdowns of care homes, universal masking of the general population, obsession with surfaces and hands, and the accelerated vaccine deployment are contrary to known science, and contrary to recent leading studies. There has been government recklessness by action and negligence by omission. Institutional measures have been needed for a long time to stem corruption in both medicine and public health policy.

The article is organized into the following sections:

- Introduction – iatrogenic pandemic of panic
- Stringency of measures has no effect on total deaths assigned to COVID-19
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Introduction - iatrogenic pandemic of panic

The health-politics context is one in which, until 2019, the reviewed science and policy consensus was that global measures such as the measures that were generally and universally applied in 2020 were [1][2]:

➔ not recommended without being justified by sufficient quantitative evidence of the local (jurisdictional) epidemiological circumstances (transmissibility, seriousness of disease, impact), and without balancing against local resulting economic, public-health and social harm

➔ for many of the measures (Contact tracing, Quarantine of exposed individuals, Entry and exit screening, Border closure), "Not recommended in any circumstances", irrespective of the severity of the pandemic viral respiratory disease (Moderate, High, or Extraordinary)

Furthermore, a major conflict of interest scandal regarding WHO flu pandemic recommendations was exposed in detail in 2010, where investigators Cohen and Carter concluded: "Key scientists advising the World Health Organization on planning for an influenza pandemic had done paid work for pharmaceutical firms that stood to gain from the guidance they wrote. These conflicts of interest have never been publicly disclosed by WHO." [4]

In 2020, none of this mattered. We entered a propaganda-driven world, with captured institutions. The precautionary principle (government must prove likely absence of harm prior to imposing dangerous policies) was turned on its head, and the burden of proof was imposed on science for a posteriori justification of unprecedented measures, swiftly imposed in an absence of and contrary to science. Unfortunately, much, or most of the science establishment complied with the new program.

Recently, there have been both dramatic events (vaccine roll out) and significant science communications, since I published my first two reviews of science relevant to COVID-19 policy, on 11 April 2020 [5] and on 3 August 2020 [6], and articles about the deadly harms of government responses, inferred from time and jurisdiction-dependent all-cause mortality data [7][8]. My first two reviews were focused on the science and politics of masks [5][6]. The present review update of recent developments is again about masks, and additionally includes key points about lockdown measures and vaccines.
Stringency of measures has no effect on total deaths assigned to COVID-19

There have been two major recent studies of global significance.

In their 21 July 2020 article “A country level analysis measuring the impact of government actions, country preparedness and socioeconomic factors on COVID-19 mortality and related health outcomes” (50 countries), Chaudhry et al. found [9]:

Rapid border closures, full lockdowns, and wide-spread testing were not associated with COVID-19 mortality per million people. (Abstract / Findings)

When COVID-19 mortality was assessed, variables significantly associated with an increased death rate per million were population prevalence of obesity and per capita GDP. In contrast, variables that was negatively associated with increased COVID-19 mortality were reduced income dispersion within the nation, smoking prevalence, and the number of nurses per million population. Indeed, more nurses within a given health care system was associated with reduced mortality. Mortality rates were also higher in those counties with an older population [...]. Lastly, government actions such as border closures, full lockdowns, and a high rate of COVID-19 testing were not associated with statistically significant reductions in the number of critical cases or overall mortality. (Section 3.4)

In their 19 November 2020 article “COVID-19 Mortality: A Matter of Vulnerability Among Nations Facing Limited Margins of Adaptation” (160 countries), De Larochelambert et al. found [10]:

Results: Higher COVID death rates are observed in the [25/65°] latitude and in the [−35/−125°] longitude ranges. The national criteria most associated with death rate are life expectancy and its slowdown, public health context (metabolic and non-communicable diseases (NCD) burden vs. infectious diseases prevalence), economy (growth national product, financial support), and environment (temperature, ultra-violet index). Stringency of the measures settled to fight pandemia, including lockdown, did not appear to be linked with death rate.

Conclusion: Countries that already experienced a stagnation or regression of life expectancy, with high income and NCD rates, had the highest price to pay. This burden was not alleviated by more stringent public decisions. Inherent factors have predetermined the COVID-19 mortality, understanding them may improve prevention strategies by increasing population resilience through better physical fitness and immunity. (Abstract)

The American Institute for Economic Research (AIER Staff) reviewed these studies and 22 further studies that make similar conclusions, in their 19 December 2020 report entitled “Lockdowns Do Not Control the Coronavirus: The Evidence” [11].

Therefore, overall, the numbers of total critical cases and total deaths were associated with the pre-existing health and societal status of the population, and this was not ameliorated by the government measures intended to slow transmission.

Importantly, in addition to studies of total-death associations, time-dependence and granularity (jurisdictional-dependence) of all-cause mortality show that the 11 March 2020 WHO declaration of a pandemic and universal recommendation to “prepare your hospitals” were followed by large numbers of deaths, probably induced by the infections and stringent lockdowns.
of unventilated care homes for sick and elderly persons.[7][8]

The mass psychology and sociology of the 2020 COVID-19 iatrogenic pandemic of propaganda are beginning to be studied by quantitative methods.[12]

Corruption of science is being exposed - Masks and PCR

A positive feature of what can be termed the current “pandemic of propaganda” is that widespread systemic corruption of establishment science is being exposed, not only via high-profile retractions of papers published in leading journals, but also through critical editorials. For example, on 13 November 2020, executive editor Kamran Abbasi put it in no uncertain terms, in the pages of the preeminent BMJ [13]:

Science is being suppressed for political and financial gain. COVID-19 has unleashed state corruption on a grand scale, and it is harmful to public health.[ref] Politicians and industry are responsible for this opportunistic embezzlement. So too are scientists and health experts. The pandemic has revealed how the medical-political complex can be manipulated in an emergency—a time when it is even more important to safeguard science.

I offer three illustrative examples.

First, systemic bias is palpable in a recent mini-saga about masks, printed in the pages of the New England Journal of Medicine [14][15][16].

Gandhi and Rutherford authored a “Perspective” article published on 29 October 2020 [14]. The authors advanced the extraordinary notion that masking lowers disease severity in those infected. They open with the propagandistic assertion that universal facial masking is “one of the pillars of COVID-19 pandemic control”. They go on to argue the fantastic: That masks can reduce the viral inoculum and thus provide asymptomatic infections in which the subject develops immunity. This alarmed respondents because the proposed mechanism is what could be termed “mask-aided naturally acquired
immunity”. Admitting any type of natural immunity, which is a hard fact of evolutionary biology, has become sacrilegious.

Two groups of researchers published rebuttals against Gandhi and Rutherford, in the same journal.

Rasmussen et al. wrote [15]:

There is insufficient evidence to support the claim that masks reduce the infectious dose of SARS-CoV-2 and the severity of COVID-19, much less that their use can induce protective immunity. [...] The suggestion that masks offer an alternative to vaccination without evidence that the benefits outweigh the great risks implicitly encourages reckless behavior.

Brosseau et al., for their part, diplomatically reset the views expounded by Gandhi and Rutherford by bringing readers back to established science and reality [16]:

Viral replication is related to dose, but disease severity is not. The epidemiology indicates that the occurrence of severe COVID-19 is associated with preexisting conditions and other risk factors, such as age, sex, and pregnancy status.[ref]

Though not yet shown in experimental models, the infectious dose of SARS-CoV-2 is probably similar to that of SARS-CoV — approximately 300 virions.[ref] Regardless of disease severity, people have high viral titers and infectious virus for at least 8 days after symptom onset. Normal talking can generate up to 3000 1-micron particles per minute in exhaled breath,[ref] and each particle could contain more than 250 virions, which means that a single minute of speaking potentially generates more than 750,000 virions. Cloth face coverings have highly variable efficacy depending on both filtering capacity and fit. Wearing a cloth face covering while being near an infected person for several minutes may not prevent the receipt of an infectious dose, which, as noted above, does not correlate with milder disease.

The authors of the original article were not deterred and replied: “more evidence is accruing to support the idea” and “there is increasing evidence both from physical sciences and from epidemiologic investigations that cloth masks (if worn properly) reduce both transmission and acquisition.”[17]

Examination of their sources shows that the authors have a generous view of what can constitute supporting “evidence”. See also [6], regarding the spin of “acquiring evidence” in the policy context of face masks.

Second, a stunning example, again about masks, is provided in the pages of Nature Medicine. Here, the “IHME COVID-19 Forecasting Team”, on 23 October 2020 (“IHME study”), declared an amazing benefit if universal masking were followed in the USA [18]:

Universal mask use could save an additional 129,574 (85,284–170,867) lives from September 22, 2020 through the end of February 2021, or an additional 95,814 (60,731–133,077) lives assuming a lesser adoption of mask wearing (85%), when compared to the reference scenario. (Abstract)

If masks provide such a large benefit, it is impossible to understand how none of the many large randomized controlled trials (RCT) with verified outcomes have detected this benefit. It is impossible to obtain the oft-repeated negative results found in the policy-grade RCT studies, if the premises and conclusions of the IHME study are correct. The IHME study was disproved prior even to its publication.

The IHME study is fatally flawed on at least two points: (1) The meta-regression used to estimate (“suggested”, in their words) that universal masking provides a
40% and more reduction in transmission is worthless, and palpably the fruit of constructive bias; (2) They used incorrect data to evaluate USA population masking compliance for the relevant time period.

The latter fatal flaw was exposed by Magness, in his report published in the Wall Street Journal, entitled “Case for Mask Mandate Rests on Bad Data” [19]:

Unfortunately, the IHME modelers’ findings contained an error that even minimal scrutiny should have caught. The projected number of lives saved, and the implied case for a mask mandate, are based on a faulty statistic. Using a months-old survey, IHME modelers assumed erroneously that the U.S. mask- adoption rate stood at only 49% as of late September, and therefore had plenty of room to increase to “universal adoption,” defined as 95%, or to a more plausible 85%. According to more recent survey findings, however, America’s mask-adoption rate has hovered around 80% since the summer.

Magness makes no mention of the IHME study’s fictitious premise that universal masking reduces transmission by 40% and more.

Third, in one of the largest scandals in the COVID-19 episode, a reverse transcription polymerase chain reaction (RT-PCR) test was hastily developed, under dubious circumstances, which is neither diagnostic of the presence of infectious viruses, nor specific to SARS-CoV-2, and deployed by States for confirmation of infection in symptomatic individuals, and for mass testing of the general asymptomatic population.

The said RT-PCR test was presented by Corman et al.[20], and their own article has:

We aimed to develop and deploy robust diagnostic methodology for use in public health laboratory settings without having virus material available.

In all these situations [all past applications of RT-PCR to “detect causative viruses from respiratory secretions”], virus isolates were available as the primary substrate for establishing and controlling assays and assay performance.

In the present case of 2019-nCoV, virus isolates or samples from infected patients have so far not become available to the international public health community. We report here on the establishment and validation of a diagnostic workflow for 2019-nCoV screening and specific confirmation, designed in absence of available virus isolates or original patient specimens. Design and validation were enabled by the close genetic relatedness to the 2003 SARS-CoV, and aided by the use of synthetic nucleic acid technology. [...] The present report describes the establishment of a diagnostic workflow for detection of an emerging virus in the absence of physical sources of viral genomic nucleic acid. Effective assay design was enabled by the willingness of scientists from China to share genome information before formal publication [...] The speed and effectiveness of the present deployment and evaluation effort were enabled by national and European research networks established in response to international health crises in recent years, demonstrating the enormous response capacity that can be released through coordinated action of academic and public laboratories [refs]. This laboratory capacity not only supports immediate public health interventions but enables sites to enrol patients during rapid clinical research responses.

The paper by Corman et al. is argued to be fatally flawed on technological and methodological grounds by an international consortium of scientists in the life sciences: See the report by Borger et al. [21]. Borger et al., among several criticisms, conclude [21]:
• These are severe design errors, since the test cannot discriminate between the whole virus and viral fragments. The test cannot be used as a diagnostic for SARS-viruses.

• Furthermore, the absence of the HE gene in both SARS-CoV1 and SARS-CoV-2 makes this gene the ideal negative control to exclude other coronaviruses. The Corman-Drosten paper does not contain this negative control, nor does it contain any other negative controls. The PCR test in the Corman-Drosten paper therefore contains neither a unique positive control nor a negative control to exclude the presence of other coronaviruses. This is another major design flaw which classifies the test as unsuitable for diagnosis.

• We find severe conflicts of interest for at least four authors, in addition to the fact that two of the authors of the Corman-Drosten paper (Christian Drosten and Chantal Reusken) are members of the editorial board of Eurosurveillance. A conflict of interest was added on July 29, 2020 (Olbert Landt is CEO of TIB-Molbiol; Marco Kaiser is senior researcher at GenExpress and serves as scientific advisor for TIB-Molbiol), that was not declared in the original version (and still is missing in the PubMed version); TIB-Molbiol is the company which was “the first” to produce PCR kits (Light Mix) based on the protocol published in the Corman-Drosten manuscript, and according to their own words, they distributed these PCR-test kits before the publication was even submitted [ref]; further, Victor Corman & Christian Drosten failed to mention their second affiliation: the commercial test laboratory “Labor Berlin”. Both are responsible for the virus diagnostics there [ref] and the company operates in the realm of real time PCR-testing.

• In light of our re-examination of the test protocol to identify SARS-CoV-2 described in the Corman-Drosten paper we have identified concerning errors and inherent fallacies which render the SARS-CoV-2 PCR test useless.

Many of the criticisms of Borger et al. [21] were already proven in detailed laboratory verifications, such as the remarkable paper by Singanayagam et al. [22], using RT-PCR with the target gene RdRp, which shows (especially their figure 3 A):

➔ The importance of the number of PCR cycles (Ct), in both clinical reporting, and clinical interpretation

➔ That except for extreme hospitalization cases (which were not studied), all the RT-PCR positives detected more than 10 days after onset of symptoms or exposure corresponded to non-infectious viruses (dead virus fragments) (no virus could be cultured in optimal cell cultures)

➔ That no time limit for detection of such non-infectious viruses (dead virus fragments) was observed, as these were obtained, with Ct=28-39, up to 60 days after onset of symptoms or exposure.

➔ That, at less than 10 days, with Ct=18-40, almost half of the “positives” were of non-infectious viruses (dead virus fragments)

➔ An operational cut-off of Ct=30, above which “positives” have less than 40% probability (<8% at Ct>35) of corresponding to viable virus, irrespective of the time relative to onset of symptoms or exposure (their Figure 2)

Such results regarding false detection of presumed viable viruses were also obtained in the more recent large study of Jaafar et al. [23] who used RT-PCR amplification of the believed to be somewhat less SARS-CoV-2-specific E gene.

Clearly, the RT-PCR test used around the world, on its own, is in effect garbage. It produces large amounts of “positives” that do not correspond to any viable
infectious virus, SARS-CoV-2 or other. This is only partly remedied if laboratories limit themselves to Ct<30, not to mention the large potential for other bad laboratory practices in the field.

Add to this the public health dishonesty of fabricating a new definition of what constitutes a “case”. A “case” is defined in medicine as an active, symptomatic and diagnosed infection. Not any more: Any “positive” in the faulty RT-PCR “test” is now counted as a “case”. The mass RT-PCR testing campaign of the general asymptomatic population, which has no clinical or epidemiological utility, thereby feeds media propaganda of fear, and disastrous consequences: Garbage-RT-PCR → meaningless-“cases” → propaganda + arbitrary-measures/great-harm + popularity of leaders[12]

**Transmission is not by contact**

On 17 September 2020, an extensive review was published by Meyerowitz et al. [24] in one of the leading medical journals in the world, the *Annals of Internal Medicine*, which concluded what should have been obvious from the start, even to the WHO: Contact transmission of viral respiratory diseases, including SARS-CoV-2, is not a thing.

In the words of Meyerowitz et al. [24]:

Strong evidence from case and cluster reports indicates that respiratory transmission is dominant, with proximity and ventilation being key determinants of transmission risk. In the few cases where direct contact or fomite transmission is presumed, respiratory transmission has not been completely excluded. Infectiousness peaks around a day before symptom onset and declines within a week of symptom onset, and no late linked transmissions (after a patient has had symptoms for about a week) have been documented. The virus has heterogeneous transmission dynamics: Most persons do not transmit virus, whereas some cause many secondary cases in transmission clusters called “superspreading events.”

(Abstract)

[...] There is currently no conclusive evidence for fomite or direct contact transmission of SARS-CoV-2 in humans.

This conclusion has far reaching implications:

- It means that “contact tracing” is an absurdity for viral respiratory diseases. No wonder the WHO in 2019 recommended that contact tracing is “Not recommended in any circumstances” (see above). Why did the WHO negate aerosol transmission for COVID-19? This is anti-science and arbitrary. [6]
It means that compulsive handwashing and surface cleaning is epidemiological nonsense, with clear negative consequences, such as massive recalls of toxic hand sanitizers [25].

It means that governments and the WHO have been negligent for more than a decade in not studying, recommending and implementing transmission-focused ventilation policies for the built environment. In fact, the WHO buried its own 2009 expert-panel report on the subject, under “water sanitation health” on its website [26], and an extensive public-domain review article was published in 2007 [27].

It means that closed door and window lockdowns of care homes for elderly persons constitute the worst possible scenario to prevent care-home epidemics. [7][8]

The reviewers Li et al. [27] concluded (their review has been cited >600 times): Ten of 40 studies reviewed were considered to be conclusive with regard to the association between building ventilation and the transmission of airborne infection. There is strong and sufficient evidence to demonstrate the association between ventilation, air movements in buildings and the transmission/spread of infectious diseases such as measles, tuberculosis, chickenpox, influenza, smallpox and SARS. (Abstract)

I have argued that it is precisely because the main transmission route is fine aerosol particles that masks cannot work to reduce transmission [5][6].

In the face of incontrovertible policy-grade evidence that masks do not reduce the wearer’s risk of being infected [5][6], the WHO and the public health complex have invented the “magical one way mask”, which prevents transmission from the wearer, while not protecting the wearer. The media has been overjoyed to propagate this fantasy, which is contrary to physics, regarding flow of aerosol-bearing air via the lowest impedance routes through and around face masks. The fantasy is the so-called “source control”, which many trained scientists have also repeated.

In fact, even a strict military grade quarantine of young healthy adults cannot prevent transmission [28].

Nurses know this. In Ontario, there have been two major administrative tribunal decisions, in 2015 and in 2018, with lengthy hearings of experts on all sides, which both concluded that nurses in several large hospitals could not be forced to wear masks, irrespective of whether they were vaccinated, because this would not protect patients [29].

“I think there is now a consensus developing in the arbitral community that there is no question that these policies really do not protect patients. The arbitrator was quite robust in describing the evidence led by the hospital as ‘insufficient, inadequate and completely unpersuasive,’” she [Sharan Basran, a lawyer for the nurses] says.
Masking in the general-population provides no detectable benefit

Since 11 April 2020, I have argued in some detail that masks don’t work, and I have dissected and exposed the disingenuous spin to the contrary.[5][6] At that time, there had not yet been a policy-grade study of masking in a general population.

On 18 November 2020, Bundgaard et al. [30] published their large randomized controlled trial (RCT) of participants selected from the general Danish population. In their words [30]:

A total of 3030 participants were randomly assigned to the recommendation to wear masks, and 2994 were assigned to control; 4862 completed the study. Infection with SARS-CoV-2 occurred in 42 participants recommended masks (1.8%) and 53 control participants (2.1%). The between-group difference was −0.3 percentage point (95% CI, −1.2 to 0.4 percentage point; P = 0.38) (odds ratio, 0.82 [CI, 0.54 to 1.23]; P = 0.33). Multiple imputation accounting for loss to follow-up yielded similar results. Although the difference observed was not statistically significant, the 95% CIs are compatible with a 46% reduction to a 25% increase in infection. (Abstract / Results)

[...] a recommendation to wear a surgical mask when outside the home among others did not reduce, at conventional levels of statistical significance, incident SARS-CoV-2 infection compared with no mask recommendation. [...] The face masks provided to participants were high-quality surgical masks with a filtration rate of 98% ref. (Discussion)

To be clear, “95% CIs are compatible with a 46% reduction to a 25% increase in infection” means that, within the bounds of uncertainty, wearing a mask could have increased the likelihood of being infected by 23%. Such is the nature of relative risk evaluation, when the comparative impact on absolute risk is too miniscule to be detected.

The authors appear to have been forced by the “peer review” process to stress that their study was not designed to test the hypothesis that I referred to above as the magical one way mask: “… and no assessment of whether masks could decrease disease transmission from mask wearers to others.”

At this stage, some fifteen (15) policy-grade RCTs later, with verified outcomes, one has to wonder what it would take for the public health complex to abandon its new-found enthusiasm for forced general-population masking, or at least to fund research on the distributed harms and societal costs of this draconian policy.

Studies on the quantifiable and potential harms of universal masking are beginning to be published, both in regular and alternative medical journals. If the “precautionary principle” was more than spin, then such studies would have been required prior to general-population masking laws and impositions.

On 6 July 2020, for example, Fikenzer et al. [31] published a rigorous study on the physiological effect of masks on 12 healthy males (age 38 ± 6 years). They concluded [31]:

Medical face masks have a marked negative impact on cardiopulmonary capacity that significantly impairs strenuous physical and occupational activities. In addition, medical masks significantly impair the quality of life of their wearer. These effects have to be considered versus the potential protective effects of face masks on viral transmissions. The quantitative data of this study may, therefore, inform medical recommendations and policy makers.
In November 2020, Borovoy et al. [32] published an extensive review of biological and medical knowledge that allows them to infer a large potential for significant harms from masking. They rightly stress the known yet underplayed role of bacteria in viral pandemics, and also review respiratory diseases arising from oral bacteria.

**Vaccines are inherently dangerous**

On 13 July 2020, an important reality check was published by Arvin et al. [33] in the pages of the leading scientific journal *Nature*, in the form of an extensive “Perspective” (review). The paper, on careful reading, is a detailed exposé about human ignorance regarding artificial interference with the human immune system. Any student of science should conclude that “we mostly don’t know anything”. The authors state this in embellished form as [33]:

Antibody-dependent enhancement (ADE) of disease is a general concern for the development of vaccines and antibody therapies because the mechanisms that underlie antibody protection against any virus have a theoretical potential to amplify the infection or trigger harmful immunopathology. This possibility requires careful consideration at this critical point in the pandemic of coronavirus disease 2019 (COVID-19), which is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Here we review observations relevant to the risks of ADE of disease, and their potential implications for SARS-CoV-2 infection. At present, there are no known clinical findings, immunological assays or biomarkers that can differentiate any severe viral infection from immune-enhanced disease, whether by measuring antibodies, T cells or intrinsic host responses. In vitro systems and animal models do not predict the risk of ADE of disease, in part because protective and potentially detrimental antibody-mediated mechanisms are the same and designing animal models depends on understanding how antiviral host responses may become harmful in humans. The implications of our lack of knowledge are twofold. First, comprehensive studies are urgently needed to define clinical correlates of protective immunity against SARS-CoV-2.
Second, because ADE of disease cannot be reliably predicted after either vaccination or treatment with antibodies—regardless of what virus is the causative agent—it will be essential to depend on careful analysis of safety in humans as immune interventions for COVID-19 move forward.

(Abstract)

Given the roll out that followed, this means that we have blindly embarked on a large-scale experiment on human subjects, without animal trials, without scientific transparency, without the possibility of informed consent, driven by pharmaceutical corporations that only want the good of humanity.

On 1 October 2020, Wehenkel [34] published a paper in which he studied 39 countries and found a large association between national influenza vaccination rate (IVR) of people 65 years and older and reported COVID-19 deaths per million inhabitants. The results are preliminary but may be a documented example of “antibody-dependent enhancement (ADE) of disease” involving COVID-19. All the highest COVID-19 death rates occurred in countries with IVR > 50% (see his figures 1 and 3). I sense a research funding opportunity to undo this finding.
Endnotes / References


My competence to review science about COVID-19

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I am retired and a former tenured Full Professor of Physics, University of Ottawa. Full Professor is the highest academic rank. During my 23-year career as a university professor, I developed new courses and taught over 2000 university students, at all levels, and in three different faculties (Science, Engineering, Arts). I supervised more than 80 junior research terms or degrees at all levels from post-doctoral fellow to graduate students to NSERC undergraduate researchers. I headed an internationally recognized interdisciplinary research laboratory, and attracted significant research funding for two decades.

I have been an invited plenary, keynote, or special session speaker at major scientific conferences some 40 times. I have published over 100 research papers in leading peer-reviewed scientific journals, in the areas of physics, chemistry, geology, bio-geochemistry, measurement science, soil science, and environmental science.

My scientific h-index impact factor is 40, and my articles have been cited more than 5,000 times in peer-reviewed scientific journals (profile at Google Scholar: https://scholar.google.ca/citations?user=1ChsRsQAAAAJ).

My personal knowledge and ability to evaluate the facts in this article are grounded in my education, research, training and experience, as follows:

i. Regarding environmental nanoparticles. Viral respiratory diseases are transmitted by the smallest size-fraction of virion-laden aerosol particles, which are reactive environmental nanoparticles. Therefore, the chemical and physical stabilities and transport properties of these aerosol particles are the foundation of the dominant contagion mechanism through air. My extensive work on reactive environmental nanoparticles is internationally recognized, and includes: precipitation and growth, surface reactivity, agglomeration, surface charging, phase transformation, settling and sedimentation, and reactive dissolution. In addition, I have taught the relevant fluid dynamics (air is a compressible fluid), and gravitational settling at the university level, and I have done industrial-application research on the technology of filtration (face masks are filters).

ii. Regarding molecular science, molecular dynamics, and surface complexation. I am an expert in molecular structures, reactions, and dynamics, including molecular complexation to biotic and abiotic surfaces. These processes are the basis of viral attachment, antigen attachment, molecular replication, attachment to mask fibers, particle charging, loss and growth in aerosol particles, and all such phenomena involved in viral transmission and infection, and in protection measures. I taught quantum mechanics at the advanced university level for many years, which is the fundamental theory of atoms, molecules and substances; and in my published
research I developed X-ray diffraction theory and methodology for characterizing small material particles.

iii. Regarding statistical analysis methods. Statistical analysis of scientific studies, including robust error propagation analysis and robust estimates of bias, sets the limit of what reliably can be inferred from any observational study, including randomized controlled trials in medicine, and including field measurements during epidemics. I am an expert in error analysis and statistical analysis of complex data, at the research level in many areas of science. Statistical analysis methods are the basis of medical research.

iv. Regarding mathematical modelling. Much of epidemiology is based on mathematical models of disease transmission and evolution in the population. I have research-level knowledge and experience with predictive and exploratory mathematical models and simulation methods. I have expert knowledge related to parameter uncertainties and parameter dependencies in such models. I have made extensive simulations of epidemiological dynamics, using standard compartmental models (SIR, MSIR) and new models.

v. Regarding measurement methods. In science there are five main categories of measurement methods: (1) spectroscopy (including nuclear, electronic and vibrational spectroscopies), (2) imaging (including optical and electron microscopies, and resonance imaging), (3) diffraction (including X-ray and neutron diffractions, used to elaborate molecular, defect and magnetic structures), (4) transport measurements (including reaction rates, energy transfers, and conductivities), and (5) physical property measurements (including specific density, thermal capacities, stress response, material fatigue...). I have taught these measurement methods in an interdisciplinary graduate course that I developed and gave to graduate (M.Sc. and Ph.D.) students of physics, biology, chemistry, geology, and engineering for many years. I have made fundamental discoveries and advances in areas of spectroscopy, diffraction, magnetometry, and microscopy, which have been published in leading scientific journals and presented at international conferences. I know measurement science, the basis of all sciences, at the highest level.