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Table of Contents

List of Abbreviations	
COVID-19	coronavirus disease that emerged in 2019
Ct	cycle threshold
IFR	infection fatality rate
PCR	polymerase chain reaction
SARS-CoV-2	severe acute respiratory syndrome-coronavirus-2
VOCs	variants of concern

1. The Problem

Severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) can cause atypical pneumonia, known as 'coronavirus disease that was identified in 2019' (COVID-19) in a subset of individuals. For most people, COVID-19 causes, at most, mild or moderate illness. For some, SARS-CoV-2 is not even a pathogen since

it does not cause disease in them. However, for two well-defined demographics, COVID-19 can be potentially severe and even lethal. This includes individuals who are immunocompromised and the elderly, especially if co-morbidities exist. Shortly after the COVID-19 pandemic was declared in Canada, caution was exercised through the declaration of emergency orders and implementation of a what was supposed to be a short-term lockdown to allow time to: (a) assess the severity of the situation, and (b) slow the first wave of cases of COVID-19 so hospitals would not get overwhelmed. This was to be a temporary measure to 'flatten the curve', which referred to a stabilization in the daily reported cases of COVID-19 when plotted on a graph. Then, we would learn to live with the virus, like we have with the man other respiratory pathogens to which we were exposed. However, more than one year later, we have experienced cyclic emergency lockdown orders on a background of constant isolation, physical distancing, and masking measures. The overall response to the declared pandemic has not altered despite overwhelming scientific data that show the risk of severe and lethal disease is almost entirely limited to two well-defined demographics. Rather than taking a balanced approach, in which economic, physical and human resources could be focused on protecting the most vulnerable, governments have opted for a very long-term 'one-size-fits-all' approach that has had dramatic consequences for the minority of high-risk individuals as well as low-risk people, who are in the majority. What follows is a discussion some of the data that highlight where COVID-19 policies have been flawed and/or have caused harm, which, in some cases, has been irreparable.

2. Dr. Byram W. Bridle's Credentials and Role in the COVID-19 'Pandemic'

Dr. Bridle is an Associate Professor of Viral Immunology in the Department of Pathobiology at the University of Guelph. His academic appointment as an independent researcher and faculty member began in January 2012. He received a MSc and PhD in immunology and completed a post-doctoral fellowship in viral immunology. His research program focuses on the development of vaccines to prevent infectious diseases and treat cancers, as well as studying host immune responses to viruses. He teaches in several courses at the undergraduate and graduate level on the topics of immunology, virology, and cancer biology. He is also involved in training Canada's next generation of multidisciplinary researchers. With respect to COVID-19, Dr. Bridle received funding from the Ontario government (COVID-19 Rapid Research Fund, Ministry of Colleges and Universities) and federal government (Pandemic Response Challenge Program, National Research Council of Canada) to develop vaccines against COVID-19. He also holds numerous grants in support of his cancer research and basic viral immunology research programs. Since the beginning of the COVID-9 pandemic he has been actively involved in disseminating fact-based, balanced scientific information to the public and policy makers to assist people with making fully informed decisions. Additional qualifications can be found in his curriculum vitae.

3. SARS-CoV-2 is Not a Problem of Pandemic Proportions

Infection fatality rate (IFR) is a way to assess how dangerous a pathogen is. It is calculated based on the number of people that die from among the total number that were infected. Early in the declared COVID-19 pandemic, it was estimated that the IFR for SARS-CoV-2 was ~10-fold higher than for a serious outbreak of an influenza virus, or ~1%. Indeed the IFR for a bad 'flu' season can be as high as ~0.1%[1].

It is important to note that calculating an accurate IFR requires having accurate data for the denominator in the equation, which is the total number of people that have been infected. Exacerbated be a lack of testing for evidence of seroconversion (*i.e.* when pathogen-specific antibodies are present in an individual, which indicates they were infected) against SARS-CoV-2, it has been impossible to ascertain how many Canadians have been infected. However, as data have accumulated globally, the total number of infections that have occurred keeps getting re-adjusted to higher numbers. As a result, the IFR for SARS-CoV-2 has been steadily declining. Remarkably, as the data regarding total infections has become more accurate, the IFR for SARS-CoV-2 has dropped to only ~0.15%[2]. It is also possible that this IFR will drop even further as the extent of unnoticed infections is further elucidated. Indeed, a recent study found that proportion of people in British Columbia that had been exposed to SARS-CoV-2 is likely substantially higher than previously appreciated[3].

Conclusion: The IFR for SARS-CoV-2 was vastly overestimated at the beginning of the declared pandemic. It is now approaching the range of a serious influenza outbreak, but with severity of disease limited to a more restricted demographic (*i.e.* unlike influenza viruses, SARS-CoV-2 is not particularly dangerous to the very young).

4. Asymptomatic Transmission of SARS-CoV-2 is Negligible

The definition of an asymptomatic individual is a person who is known to be infected with a microorganism but fails to develop disease. Indeed, we are all 'asymptomatic carriers' in the sense that we harbor vast numbers of bacteria and viruses in our bodies. However, these normal microbiomes usually do not cause us any disease, unless we become immunosuppressed or 'safe' microbes get transferred to anatomical locations where they can potentiate disease (*e.g.* fecal to oral transfer of some strains of Escherichia coli). So, in the context of SARS-CoV-2, an asymptomatic carrier would be defined as an individual that is infected with the virus but fails to develop COVID-19.

Viral culture studies suggest that pre-symptomatic individuals can potentially shed infectious SARS-CoV-2 one to two days before the onset of symptoms and continue to be infectious up to seven days thereafter[4]. However, a study of the prevalence of SARS-CoV-2 in ~10 million people in Wuhan, China found no evidence of asymptomatic transmission[5]. In the United Kingdom, the 'Scientific Advisory Group for Emergencies' recommended that "Prioritising rapid testing of symptomatic people is likely to have a greater impact on identifying positive cases and reducing transmission than frequent testing of asymptomatic people in an outbreak area"[6]. Consequently, they have asked their government to change their testing policy by moving away from asymptomatic testing.

The World Health Organization notes that "Most PCR assays are indicated as an aid for diagnosis, therefore, health care providers must consider any result in combination with timing of sampling, specimen type, assay specifics, clinical observations, patient history, confirmed status of any contacts, and epidemiological information"[7].

On its own, a positive result on a polymerase chain reaction (PCR) test to detect SARS-CoV-2 is insufficient to diagnose COVID-19. In addition to the potential for false positive tests, true positive results can also be obtained from genomes of SARS-CoV-2 particles that are no longer infectious. An example of the latter would be an individual who has mounted a successful immune response and may have remnant

viral particles of partially degraded viral genetic material inside relatively long-lived phagocytic cells that have killed the virus. Indeed, following clearance of SARS-CoV-2 from the body, full and/or partial genomes of SARS-CoV-2 can remain for many days, even weeks. One key reason for this is that some phagocytic cells, which are a component of the innate immune system, can be long-lived. The three primary phagocytic cells in the body are neutrophils, macrophages, and dendritic cells. Neutrophils are the 'first responders' of the immune system. They rapidly infiltrate sites of SARS-CoV-2 infection and begin to phagocytose (*i.e.* consume or internalize) SARS-Cov-2 particles. The neutrophils, which are short-lived, then recruit macrophages and dendritic cells to the site of infection. Note that dendritic cells also reside at strategic sites of infection where they can immediately begin to phagocytose SARS-CoV-2. The macrophages and dendritic cells are much larger than neutrophils and can phagocytose relatively large quantities of the virus and can be relatively long-lived. One of the reasons for this is because these two cell types are critical for activating T cells and B cells, which are the key effectors against viral infections. Phagocytosis of SARS-CoV-2 is a mechanism to kill and remove the virus from the body and to activate other immunological effector cells. As such, these can be a source of SARS-CoV-2 genomes that could be amplified by a RT-PCR test. However, these genomes would not have the potential to cause COVID-19. Persistence of whole or partial genomes that are not associated with infectious particles is well-documented for a variety of other viruses, including measles[8], Middle East respiratory syndrome-coronavirus[9], and other coronaviruses[10].

Too often, a positive PCR test for the presence of SARS-CoV-2 is being used, on its own, to define positive cases of COVID-19. However, the presence of a portion of the viral genome in an individual, on its own, does not necessarily equate with disease (*i.e.* COVID-19). To be declared COVID-19, the infection would also have to be associated with expected signs and/or symptoms. The latter is known as a clinical diagnosis and would be based on evaluation by a physician, in conjunction with the test results. A gold-standard test for infectivity of a virus is a cell-based functional assay that determines the potential to cause cell death. However, such an assay is not in routine use in Canada. The absence of a test of the infection-potential of a virus further confounds any meaningful interpretation of positive results in asymptomatic people. Drawing conclusions based solely on the results of laboratory tests, would take the diagnosis of diseases would be taken out of the hands of physicians and placed into the hands of technicians employed by testing laboratories.

Positive PCR tests for SARS-CoV-2 in asymptomatic people are often based on high cycle threshold (Ct) values, which, in and of themselves, raise the question of whether these individuals harbor infectious viral particles. The low prevalence of positive PCR tests in asymptomatic people often does not differ much from the false positive rate. These issues combined with the absence of a functional cell-based assay to prove infectivity renders results of asymptomatic testing nearly impossible to interpret accurately. Indeed, the World Health Organization, agreeing with many health professionals around the world, has emphasized that spreading of SARS-CoV-2 by asymptomatic individuals is rare and an emphasis should be placed, therefore, on testing people with signs or symptoms of illness, not those who are apparently healthy[11]. Of particular concern in the context of the high cycle numbers being used by labs in Alberta (*i.e*. up to 35 cycles being defined as 'positive' by Alberta Health Services[12]), is the fact that several studies have been conducted to determine the highest Ct value at which SARS-CoV-2 could be successfully cultured in cells. The results were 25[13], 22-27[14], 30[15]. This suggests that tests with Ct values above 22-30 are almost certainly not indicative of the presence of replication-competent SARS-CoV-2. The conclusion is that it is erroneous to declare samples with high Ct values, especially those above 30, as being positive

for infectious SARS-CoV-2. It was even concluded in a study by La Scola B, *et al*., that patients testing 'positive' with Ct values above 33 could likely be discharged from hospitals[16]. This means that an unknown number of positive cases reported in Alberta were likely not true positives, especially if individuals were asymptomatic. This is further supported by evidence that asymptomatic people have detectable SARS-CoV-2-specific memory T cells after exposure to the virus, which would be inconsistent with a risk of them spreading the virus to others[17].

Importantly, false positive test results, which have a greater risk of happening among asymptomatic people, have been shown to have numerous negative consequences in terms of physical and mental health, and causes financial losses[18].

Conclusion: Testing of asymptomatic people for the presence of portions of the SARS-CoV-2 genome does not make medical nor economic sense. Positive test results cannot be interpreted in a clinically meaningful way. Also, there is no substantial evidence to suggest that people who are asymptomatic represent a substantial risk of causing COVID-19-related hospitalizations or deaths in others.

5. Individuals Who Had COVID-19 Cannot Re-Transmit the Virus

When people get infected with a respiratory pathogen, their immune system detects the virus as something that is dangerous and worth responding to. Rapid innate immune responses provide early effector mechanisms to being clearing the virus from the body. The innate arm of the immune system will also induce an adaptive immune response. The primary effectors against viruses in the adaptive arm of the immune system are cytotoxic T cells that can kill virally infected cells to prevent them from serving as a 'virus-production factory', and B cells, which can produce antibodies to neutralize the virus and prevent it from entering cells. The most notable characteristic of the adaptive immune response is that it results in the generation of immunological memory. This allows a host to respond much more rapidly and to a much greater magnitude when re-exposed to the same pathogen. The result is that the virus gets cleared so rapidly that there is usually no disease.

Note that some non-immunologists have erroneously concluded that memory conferred by natural infection with SARS-CoV-2 is not long-lasting. However, this has been based on assessments that show declining concentrations of virus-specific antibodies. The antibodies are produced by B cells. The antibodies are merely proteins in circulation with limited half-lives. They will be cleared from circulation over time. The relevant measure of memory is detection of memory B and T cells. A memory B cells can rapidly initiate the production of massive quantities of antibodies upon re-exposure to the pathogen.

Several published studies have shown that the immune response against SARS-CoV-2 infections is robust, effective, broadly targets multiple components of the virus and confers memory that lasts at least as long this aspect has been able to be studied within the context of a novel pandemic[19, 20, 21, 22, 23, 24].

Conclusion: The scientific evidence demonstrates that immune responses following infection with SARS-CoV-2 are protective and long-lasting. There is no evidence that people who previously tested positive for SARS-CoV-2 represent a substantial risk of causing COVID-19-related hospitalizations or deaths in others.

6. SARS-CoV-2 Variants of Concern

Many viruses mutate over time. This includes coronaviruses. Indeed, these viruses have an error-prone mechanism of copying their genome. This provides a strategy to adapt to novel environmental pressures. Of concern for SARS-CoV-2 is the potential for randomly generated mutants to sufficiently alter the structure of their spike protein to be able to evade the narrowly conferred spike protein-specific immunity conferred by all of the first-generation COVID-19 vaccines while maintaining the ability to infect cells. Since the beginning of the pandemic, large numbers of mutant viruses have been identified. However, three core lineages of the variants are of current concern[25]: 1. B.1.1.7, also known as the UK variant[26], 2. B.1.351, also known as the South African variant[26], 3. P.1, the Brazilian variant[27]. SARS-CoV-2 from the B1.351 lineage can largely bypass the immunity conferred by AstraZeneca's COVID-19 vaccine. However, the Pfizer and Moderna vaccines remain effective against all three lineages for the VOCs.

Some of the VOCs seem to be associated with more efficient spreading between people. This is likely due, at least in part, to the increased affinity of their spike protein for the ACE2 molecule that SARS-CoV-2 uses to enter cells. However, there is no evidence that the current VOCs are associated with a higher incidence of severe or fatal COVID-19.

Importantly, naturally acquired immunity against SARS-CoV-2 has been shown to be both long-lasting and protective. Notably, this type of immunity would be expected to be particularly protective against emerging VOCs because it is very broad, meaning that it targets multiple components of SARS-CoV-2, with both T cells and antibodies induced as effector mechanisms. Indeed, evidence of the breadth of naturally acquired immunity has recently been published[3]. In contrast, current vaccine-induced immunity targets a single protein, with a strong bias towards antibody-mediated responses. Notably, the B.1.1.7, B.1.351, and P.1 variants of SARS-CoV-2 are of concern because of their altered spike proteins, particularly in the 'receptor binding domain' (*i.e.* the portion that binds to the ACE2 molecule on host cells), which is the primary target of neutralizing antibodies. So, although there is evidence of some monoclonal antibodies failing to recognize the spike protein in some VOCs and some convalescent sera (*i.e.* sources of antibodies) being less able to neutralize the VOCs, T cells can effectively recognize conserved regions of the spike protein as well as other viral proteins.

Since SARS-CoV-2 has shown such a propensity to mutate, it is reasonable to expect this virus will become endemic. Indeed, should a variant emerge that can completely bypass the spike-specific immunity conferred by the current vaccines, additional immunizations will be required with re-designed vaccines, especially for those without naturally acquired broad-based immunity.

Conclusion: The goal in Canada should not be to get everyone vaccinated per se. Instead, the goal should be to get as many Canadians immune to SARS-CoV-2 as possible. There are two ways to achieve this: 1. Vaccination, 2. Natural acquisition of immunity. The great news is that Canada might be closer to the natural acquisition of herd immunity than what was previously appreciated[3], likely due, in large part, to the ongoing spread of the virus after the implementation of ineffective masking and misguided physical distancing policies that failed to account for the physics behind aerosol-mediated transmission of SARS-CoV-2. Like many other viruses, including other coronaviruses and influenza viruses, SARS-CoV-2 will likely become endemic, meaning that we may encounter new versions of the virus on a regular and long-term basis. As such, it is imperative that we learn to live with SARS-CoV-2 rather than attempting to hide from it; just like we have done with the other respiratory pathogens that we have accepted as a trade-off for living our lives outside the confines of lockdowns.

7. Masking Lacks Rationale in the Context of SARS-CoV-2 Spreading via Aerosols

It is now widely recognized that SARS-CoV-2 is effectively spread via aerosols coming from the respiratory system[28, 29, 30, 31, 32]. A pulmonary (*i.e.* lung-derived) aerosol is a suspension of fine water droplets suspended in exhaled air. Many people who wear glasses will be familiar with these aerosols. Indeed, when a person exhales onto the lenses of their glasses to polish them with a cloth, the liquid being deposited is due to the condensation of the lung-derived aerosol. Also, these aerosols can be readily visualized when exhaling into cold air, which causes the fine droplets to condense (*i.e.* drop out of the gaseous phase). Indeed, this condensation effect of cold air minimizes the distance that respiratory aerosols can travel since the condensed water droplets are relatively large. However, in warm air these aerosols are invisible and can potentially travel long distances depending on the rate of ambient air flow. The masks in common use among Canadians (*e.g.* surgical and cloth masks) lack standardization, users are not required to undergo fit-testing, and even if these were done, they would still lack the ability to prevent the spread of aerosols. Low-cost masks do not seal properly around the face, with leaks commonly occurring around the nose and at the joints of the jaw. Due to simple physics in which air will follow the path of least resistance, most exhaled and inhaled air will leave and enter via these gaps in the masks. This is further exacerbated by anything that increases these gaps. An example would include a beard, which would separate the mask from the chin, thereby replacing the mask material with a coarse-haired filter with massive pore sizes relative to the size of a virus. Anyone who wears glasses and a mask can attest to the venting issue around the nose, as it often causes the lenses to fog. It seems illogical to force a person's pulmonary exhaust to flow over their eyes, since this is a known route of infection for SARS-CoV-2 and could, therefore, potentiate spreading of the infection in an individual. It was shown that ocular tissues express entry receptors for SARS-CoV-2 and conjunctivitis is common among people diagnosed with COVID-19, sometimes even preceding the onset of signs and symptoms of respiratory distress[33]. As such the eyes could potentially serve as both a portal of entry and a source of person-to-person transmission.

Air venting past the ears, which is the other common location of leakage with low-cost masks, means that aerosols are generally directed behind a person. However, public health policies usually recommend that people turn away from other individuals if they must pass within proximity. If anything, this simply increases the chance of someone being exposed to pulmonary aerosols with a higher flow rate. The principles of distributing pulmonary aerosols over the eyes and behind a person also holds true for face shields. This highlights how poorly thought out masking policies are. Even if low-cost masks were properly sealed around the neck and face, SARS-CoV-2-laden aerosols and still readily pass through the relatively large pore sizes of the filtering material. Indeed, a study published in 2019 found that the low-cost masks had pore sizes ranging from 80 to 500 µm in diameter[34]. Water droplets that come from the lungs are defined as 'large droplets', 'small droplets' or 'droplet nuclei' and range in size from >60 µm, 10-60 µm, and <10 µm in diameter, respectively[35]. Coughs and sneezes will discharge droplets of all sizes. However, regular breathing and talking primarily discharges small droplets and droplet nuclei. Notably, SARS-CoV-2 has a diameter of only ~1 µm. This means that virus-laden droplets in pulmonary aerosols will have a maximum diameter of ~62 µm, with the vast majority being much smaller (remember that the pores in low-cost masks are ≥80 µm. As such, low-cost masks fail to stop the spread of SARS-CoV-2. One of the biggest challenges in relaying the science is the 'invisibility' of the microbial world. To place this into a context that is easier to picture, this would be akin to thinking that a person is locked inside a house when

the walls have huge gaping holes (*i.e.* the leakage points were there proper seals are lacking) and the front door is open (*i.e.* representing the pore size of a mask). The reality of this scenario is that the person is free to come and go as they wish.

Also, aerosols from the lungs can travel beyond two meters and the directionality will be dictated by air currents[36]. Although the viral load that a person would be exposed to from aerosols would decrease with distance, the long-range potential of aerosols highlights the arbitrariness of 2-meter physical distancing policies. Further, buildings with poor ventilation, which encompasses most buildings in Canada, facilitate the build-up of aerosols over time, which further confounds the value of two-meter distancing[37]. Finally, for the vast majority of people it is not possible to wear masks for prolonged periods of time without touching it with their fingers. For example, jaw movements associated with talking, yawning, *etc.*, causes low-cost masks to slide off the nose. Handling of masks that are dampened with aerosols promotes contamination of the fingers and anything they touch thereafter. In addition to spreading via aerosols, the other major route of transmission is via contaminated hands of infected individuals[38], which is potentiated by masking. As such, removing masking mandates and promoting traditional hand washing would be a more logical approach to reducing the spread of SARS-CoV-2.

A recent review of masking data generated during the pandemic concluded there are numerous other harms associated with masking and that it is not effective in preventing transmission of SARS-CoV-2[39]. Here are the precise conclusions from this study: "*The existing scientific evidences challenge the safety and efficacy of wearing facemask as preventive intervention for COVID-19. The data suggest that both medical and non-medical facemasks are ineffective to block human-to-human transmission of viral and infectious disease such SARS-CoV-2 and COVID-19, supporting against the usage of facemasks. Wearing facemasks has been demonstrated to have substantial adverse physiological and psychological effects. These include hypoxia, hypercapnia, shortness of breath, increased acidity and toxicity, activation of fear and stress response, rise in stress hormones, immunosuppression, fatigue, headaches, decline in cognitive performance, predisposition for viral and infectious illnesses, chronic stress, anxiety and depression.*"

Demonstration of inadequate sealing of low-cost masks around the face are shown in figures 3 and 4. The relative size of SARS-CoV-2-laden water particles and pores of low-cost masks is shown inf figure 5. Figure 6 shows how readily aerosols can pass through masks, even when having to pass through five three-ply surgical masks. Figure 7 shows the personal protective equipment required to safely work with containment level-3 pathogens such as SARS-CoV-2.

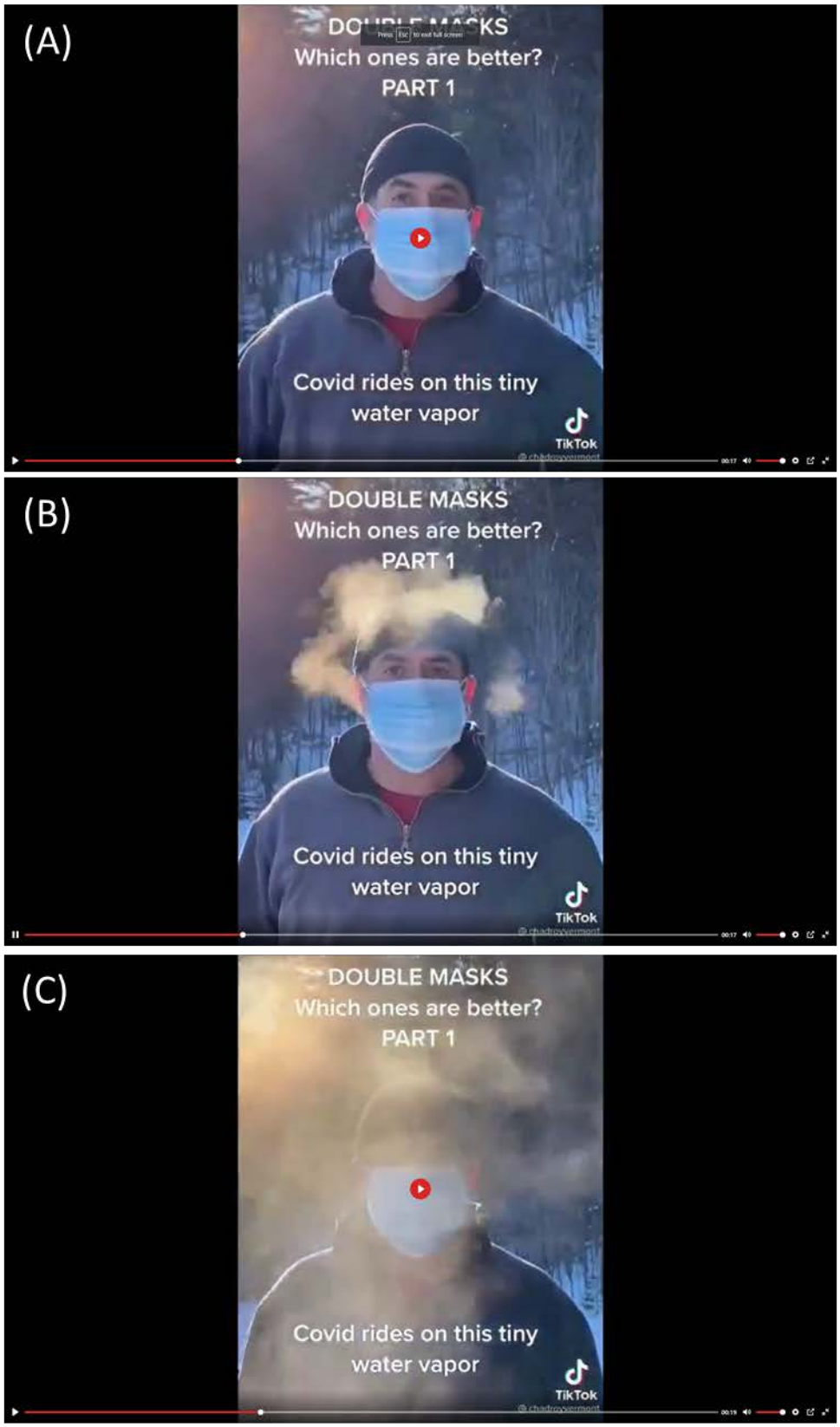

Figure 3: The leakiness of low-cost masks.
These are screen shots taken from a video showing cold-mediated condensation of a pulmonary aerosol when exhaling while wearing two three-layer surgical masks that had the metal bar pinched over the nose. (A) at the end of the inhalation. (B) During exhalation aerosol exiting the lungs is condensing in the cold air. (C) At the end of the exhalation, the profound amount of aerosol released from the mask after a single exhalation is evident.

(A)

(B)

Figure 4: The leakiness of low-cost masks.

These are screen shots taken from a video showing fogging of eyeglasses when wearing a three-layer surgical mask. (A) While inhaling, the metal bar over the nose is pinched to maximize the 'seal'. (B) During exhalation aerosol exiting the lungs is condensing on the lenses of the glasses, causing them to fog.

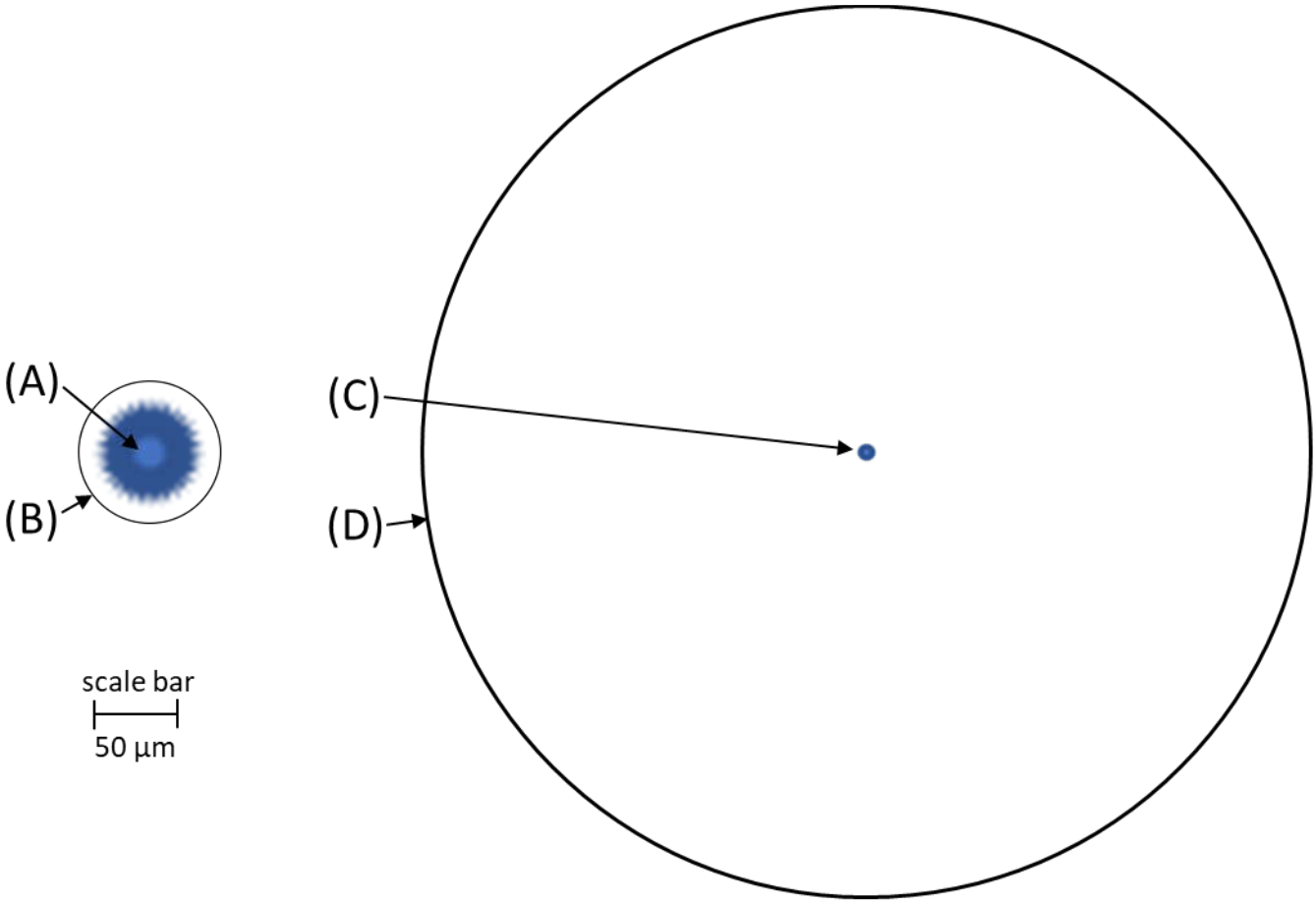

Figure 5: The relative size of SARS-CoV-2-laden water particles and pores of low-cost masks. SARS-CoV-2 particles have a diameter of ~1 µm. Water droplets in air exhaled from the lungs can be classified into three sizes. Large droplets are >60 µm, small droplets are 10-60 µm in diameter, and droplet nuclei are >10 µm in diameter. Individuals who are not coughing or sneezing will exhale an aerosol that consists almost entirely of droplet nuclei and small droplets. (A) The largest of the small droplets that are laden with SARS-CoV-2 will have a diameter of ~62 µm. (B) The smallest pore size of a low-cost mask is ~80 µm. (C) The largest of the droplet nuclei that are laden with SARS-CoV-2 will have a diameter of ~12 µm. (D) The largest pore size of a low-cost mask is ~500 µm.

= virus-laden droplet = pore in a low-cost mask

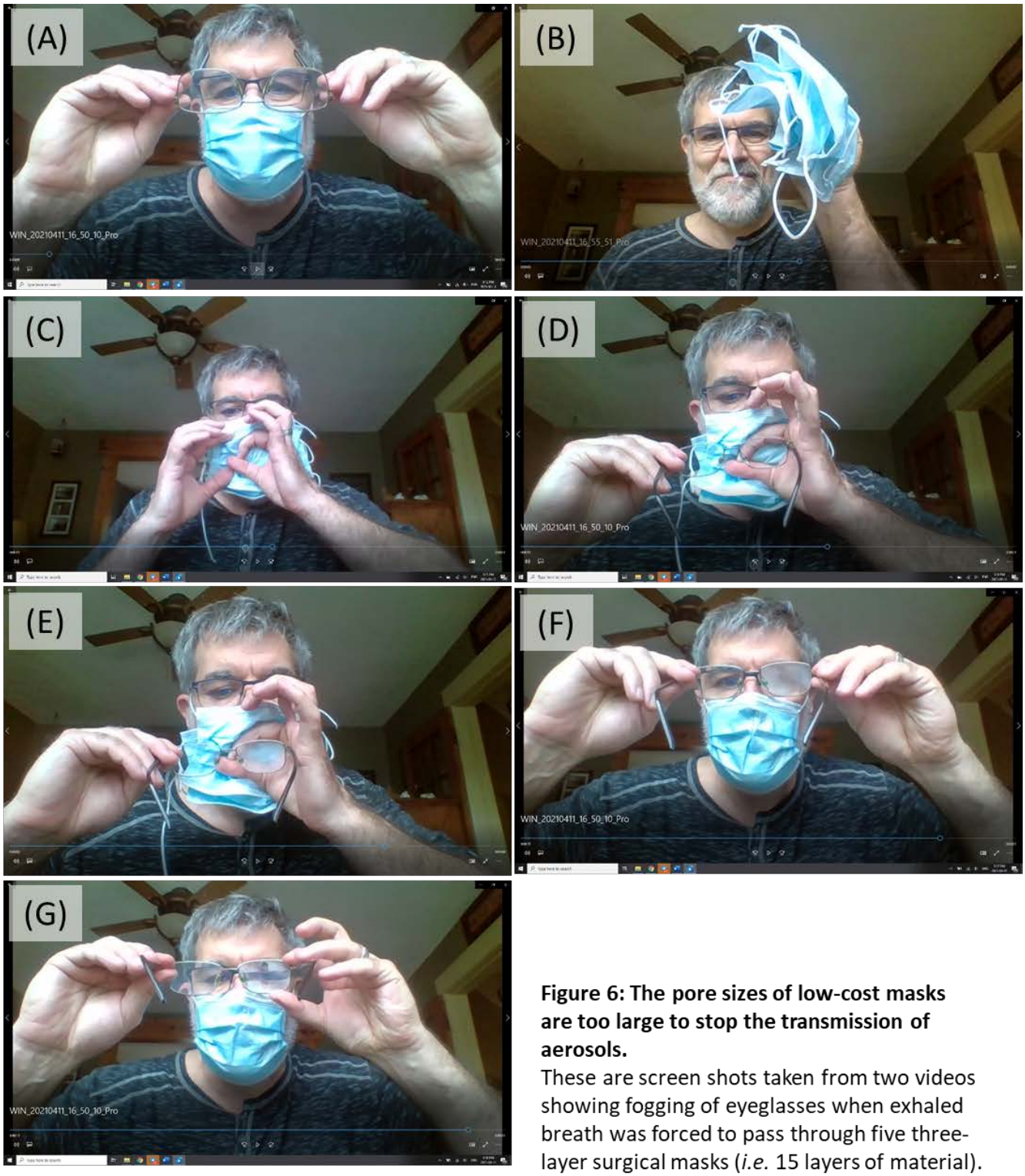

Figure 6: The pore sizes of low-cost masks are too large to stop the transmission of aerosols.

These are screen shots taken from two videos showing fogging of eyeglasses when exhaled breath was forced to pass through five three-layer surgical masks (*i.e.* 15 layers of material). (A) This image shows the clarity of the eyeglasses when no fogging is present. (B) Five surgical masks were placed sequentially over the mouth. (C) A ring was made with the finger and thumb to apply pressure around the lips and seal the mask so the only place exhaled air could exhaust was through the five three-ply surgical masks. (D) Beginning to exhale through the five masks. (E) Near the end of exhalation. (F) Post-exhalation evidence of fogging is present on the lens of the eyeglasses to the right of the image. (G) So much aerosol had condensed on the lens of the eyeglasses that a cross pattern could be drawn in the liquid.

Figure 7: Personal protective equipment required to safely work with containment level-3 pathogens such as SARS-CoV-2.

SARS-CoV-2 is defined as what is known as a 'containment level-3 pathogen' by the Public Health Agency of Canada. The personal protective equipment that they require scientists to use to ensure safe handling of SARS-CoV-2 typically includes the following: 1. Handling of SARS-CoV-2 can only be done inside a certified containment level-3 facility. 2. Anything containing SARS-CoV-2 can only be opened inside a biological safety cabinet, which is designed to provide a barrier between the virus and the scientist. 3. The scientist must wear a full body suit, including shoe covers and gloves. A head covering with a clear face shield and that seals around the neck and face must be worn. The head covering is connected by a tube that is attached to a pump that delivers filtered air into the head covering, thereby maintaining positive pressure (*i.e.* ambient air cannot flow into the head covering). Personal protective equipment that is known to prevent the wearer from being infected with a containment level-3 pathogen, such as SARS-CoV-2, is shown in figure 7.

A person wearing a low-cost mask would not be allowed to enter a containment level-3 facility due to a profound lack of protection. There is, therefore, a large discrepancy between what truly protects an individual from SARS-CoV-2 and the public health messaging surrounding cloth and surgical masks, which falsely implies a substantial amount of protection.

There are other notable harms associated with long-term masking. Although the pores sizes of low-cost masks are too large to efficiently stop the spread of SARS-CoV-2-laden aerosols, bacteria are much larger, as are dust and other environmental particles. Long-term prevention of exposure to the microbial world and natural environment in children has been associated with an increased incidence of allergies, asthma and autoimmune diseases based on an immunological principle known as the 'hygiene hypothesis'[40, 41]. Another potential harm of wearing masks is the psychological effect it has on adherence to public health protocols. The false sense of security that a mask confers causes many people to become less aware of or less concerned with the practice physical distancing. Additional problems include things like blunting social cues by preventing reading of facial body language, muffling speech (a particular concern for individuals with pre-existing speech disorders), preventing lip-reading, and exposure to hypoxia (low oxygen levels) due to slowing of gas exchange, especially when active[39].

Conclusion: Once one realizes that SARS-CoV-2 can pass through low-cost masks and travel >2 meters and sometimes much further on 'droplet nuclei' in pulmonary aerosols, it becomes readily apparent that the policies of mask-wearing and two-meter physical distancing are not adequately protective against the spread of SARS-CoV-2. If low-cost masking combined with only two-meter physical distancing does little to prevent the spread of SARS-CoV-2, it would be expected that a relatively high proportion of Canadians would have naturally acquired immunity to the virus over the past year. Indeed, this is precisely what was found in a recently published study that showed that the majority of apparently healthy adults in British Columbia have evidence of naturally acquired immunity[3]. Therefore, low-cost masking to protect against transmission of SARS-CoV-2 is futile. At the very least, liberal mask exemptions should be more commonplace.

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