File No. CI 20-01-29284

THE QUEEN'S BENCH
Winnipeg Centre

APPLICATION UNDER: *The Constitutional Questions Act*, C.C.S.M., c. 180

AND UNDER: The Court of Queen's Bench Rules, M.R. 553-88

IN THE MATTER OF: *The Public Health Act*, C.C.S.M. c. P210

B E T W E E N:

GATEWAY BIBLE BAPTIST CHURCH, PEMBINA VALLEY BAPTIST CHURCH, REDEEMING GRACE BIBLE CHURCH, THOMAS REMPEL, GRACE COVENANT CHURCH, SLAVIC BAPTIST CHURCH, CHRISTIAN CHURCH OF MORDEN, BIBLE BAPTIST CHURCH, TOBIAS TISSEN, ROSS MACKAY,

Applicants,

- and -

HER MAJESTY THE QUEEN IN RIGHT OF THE PROVINCE OF MANITOBA and DR. BRENT ROUSSIN in his capacity as CHIEF PUBLIC HEALTH OFFICER OF MANITOBA, and DR. JAZZ ATWAL in his capacity as ACTING DEPUTY CHIEF OFFICER OF HEALTH OF MANITOBA

Respondents.

AFFIDAVIT OF JASON KINDRACHUK
AFFIRMED: April 29 , 2021

DEPARTMENT OF JUSTICE
Constitutional Law Branch
1205 - 405 Broadway
Winnipeg, Manitoba
R3C 3L6

Per: Heather Leonoff

Telephone No. (204) 945-0679
Facsimile No. (204) 945-0053

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Respondents.

AFFIDAVIT OF JASON KINDRACHUK

I, JASON KINDRACHUK, of the City of Saskatoon, in the Province of Saskatchewan,

AFFIRM AS FOLLOWS:

1. I have personal knowledge of the facts and matters hereinafter deposed to by me, except where same are stated to be based upon information and belief, and those I believe to be true.

2. Attached hereto and marked as Exhibit "A" to this my Affidavit is my Reply Report.

3. I make this affidavit bona fide.

AFFIRMED before me in the City of Winnipeg, in the Province of Manitoba, through use of video conferencing as permitted by order under *The Emergency Measures Act*, this 29 day of April, 2021.

Heather Leff

A Barrister-at-law entitled to practice in and for the Province of Manitoba

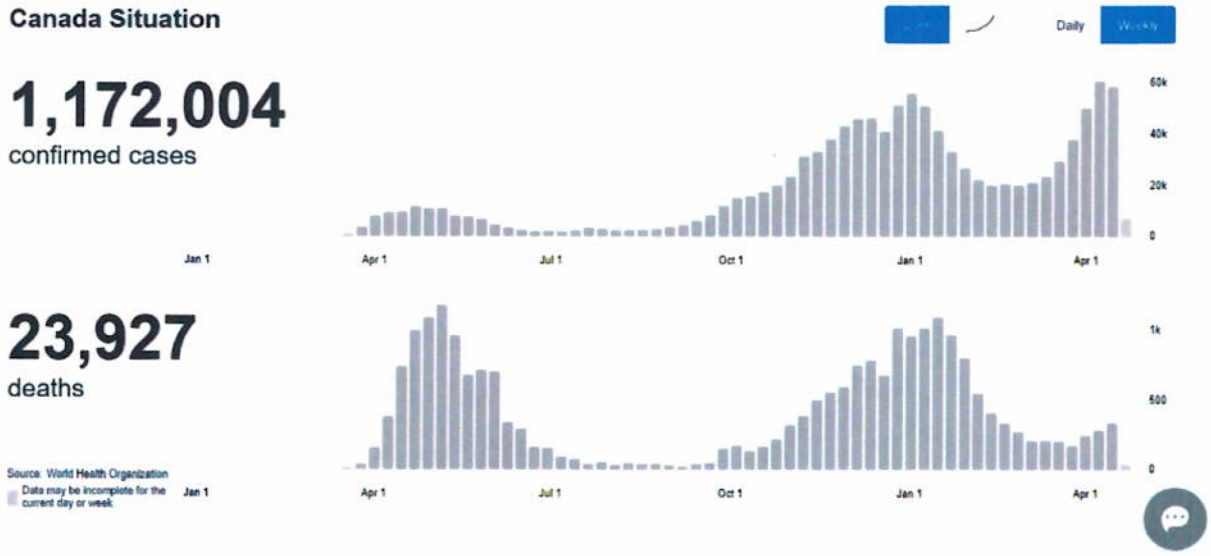
JASON KINDRACHUK

This is Exhibit "A" referred to in the Affidavit of Jason Kindrachuk affirmed before me the 29th day of April, 2021.

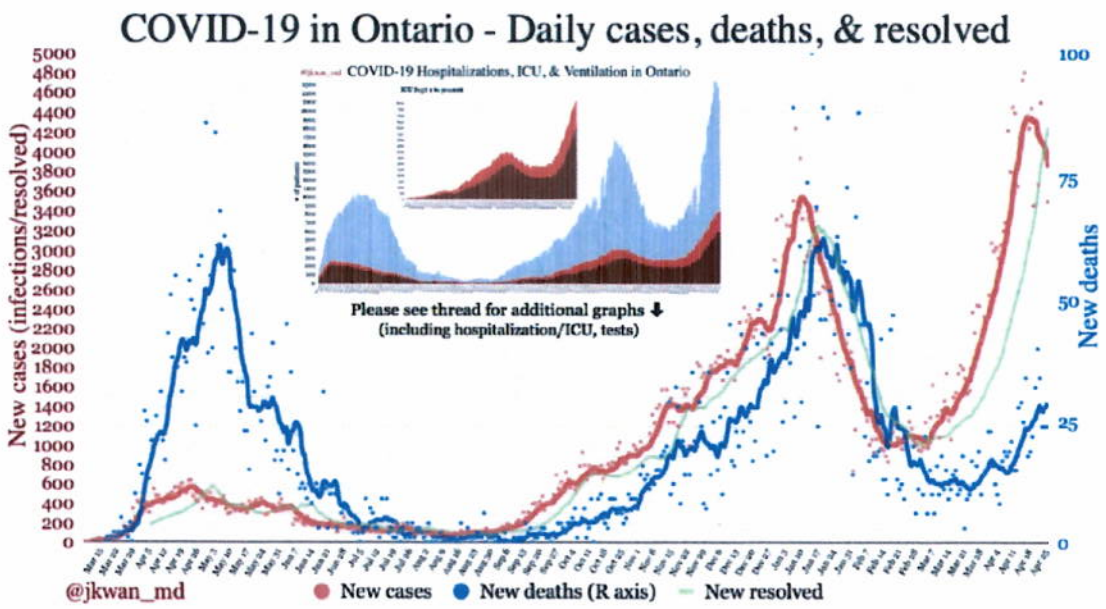
A Barrister at Law in and for the Province of Manitoba.

The resurgence of Covid-19 in Canada.

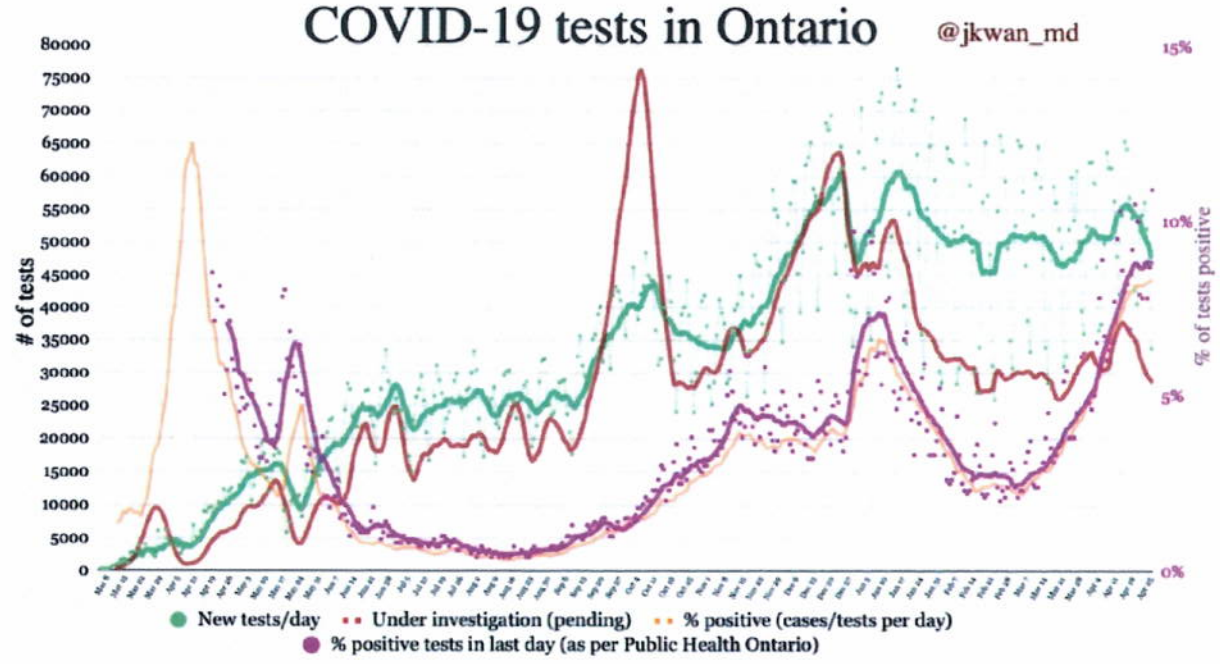
Dr. Bhattacharya asserted in his second report that epidemiological modeling and the voices of infectious disease experts in Canada (myself included) and the Public Health Agency of Canada had gotten it wrong in regards to our concerns regarding the spread of SARS-CoV-2 variants of concern within the country. Dr. Bhattacharya utilized a figure from the Financial Times to show that through March 2021, there had only been a minor increase in cases. Of particular note is that the slope of the graph in the figure provided by Dr. Bhattacharya directly suggested a clearly increasing trend in cases that had begun at a higher baseline of cases than either the first or second waves in Canada and the slope was very similar to that seen in the early stages of the second wave (Sep-Oct 2020). As of today (April 26, 2021), total nationwide cases have reached the same maxima as recorded during the second wave with many regions reporting increasing test positivity rates:



Further, hospitalizations and intensive care unit admissions continue to increase across multiple regions, most notably Ontario where hospitalizations, ICU admissions and patients requiring ventilation have exceeded those seen during the second wave.



Further, it is notable that while this current data plot of cases suggests that overall case numbers are decreasing, the test positivity rate has continued to remain elevated with no obvious decreasing trend. This suggests that many cases of infection are currently undetected and thus widespread community transmission is ongoing.



In addition, variants of concern are now responsible for the majority of reported cases with B.1.1.7 being highly over-represented within these cases (source: Public Health Ontario daily epidemiological summary April 25, 2021).

Figure 5. Number of confirmed COVID-19 cases and percent positive for mutations or VOCs: Ontario, February 7, 2021 to April 25, 2021

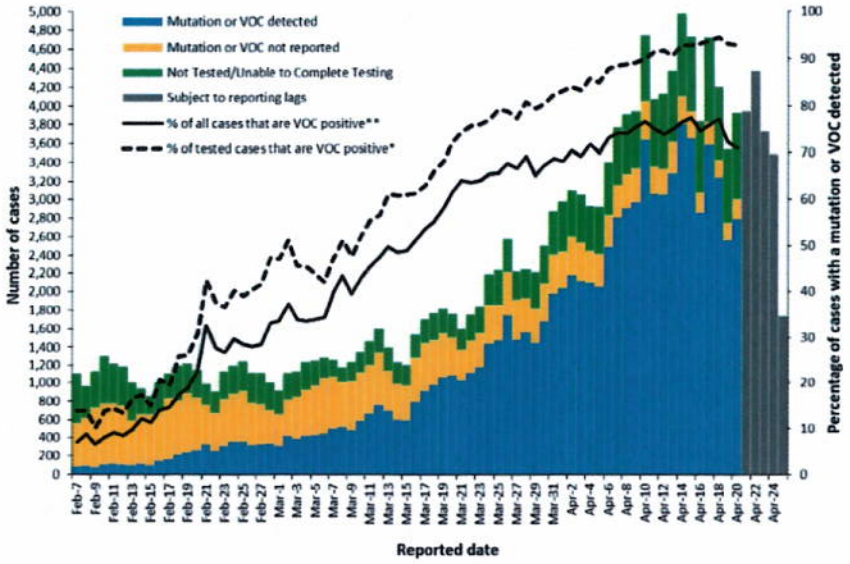


Table 7. Summary of confirmed COVID-19 cases with a mutation or VOC detected: Ontario

	Change in cases April 24, 2021	Change in cases April 25, 2021	Cumulative case count up to April 25, 2021
Variant of Concern			
Lineage B.1.1.7*	2,538	2,038	54,436
Lineage B.1.351	14	-2	162
Lineage P.1	80	4	351
Mutations			
N501Y and E484K	133	165	4,740
N501Y (E484K unknown)**	-105	56	22,794
E484K (N501Y negative)	84	137	1,816
E484K (N501Y unknown)	16	15	420

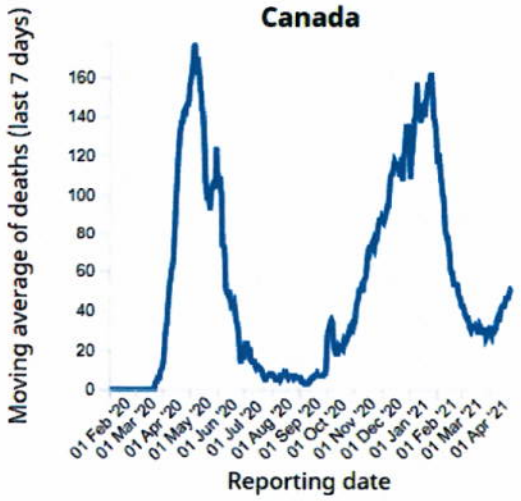
Note: Interpret the VOC and mutation trends with caution due to the varying time required to complete VOC testing and/or genomic analysis following the initial positive test for SARS-CoV-2. Due to the nature of the genomic analysis, test results may be completed in batches. Data corrections or updates can result in case records being removed and/or updated and may result in totals differing from past publicly reported case counts. Data for calculating the change in cases and the cumulative case counts uses data from the Investigation Subtype field only. Changes to the VOC testing algorithm may impact counts and trends. Further details can be found in the data caveats section.

*Includes all confirmed COVID-19 cases where lineage B.1.1.7 was identified by genomic analysis and those presumed to be B.1.1.7 based on positive N501Y and negative E484K mutation in the Investigation Subtype field

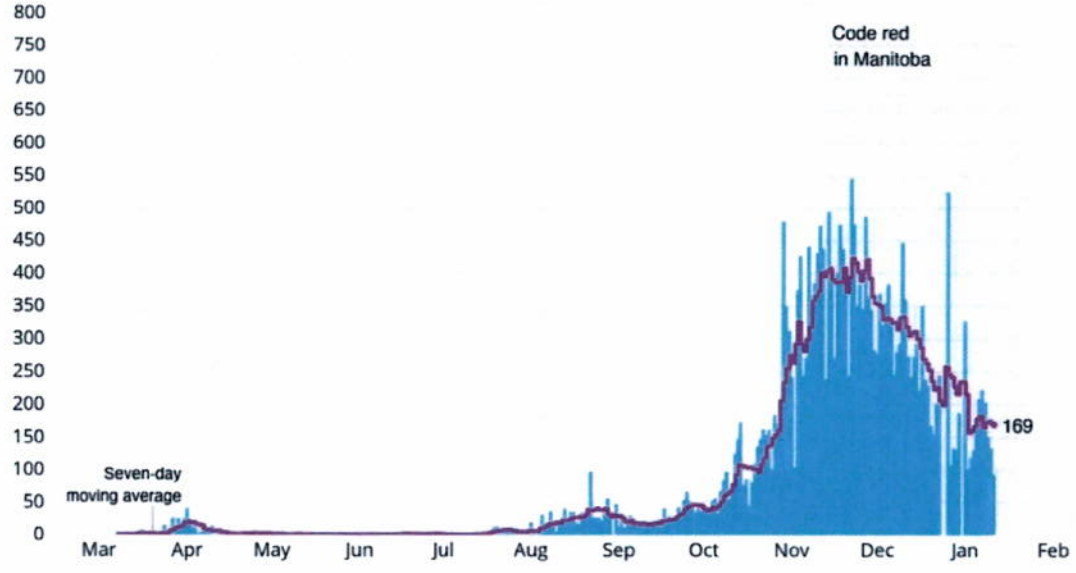
**The category 'N501Y (E484K unknown)' mainly consists of results from before the introduction of the E484K test. Counts will shift from this category into a VOC lineage category as E484K tests or genomic analysis are completed.

In the most recent affidavit response, Dr. Bhattacharya stated that "This means that the presence of a variant circulating in the population poses little additional risk of hospital overcrowding or excess mortality due to viral infection". Increasing hospitalizations across Canada due to variants of concern, and increased presentation of younger age groups requiring hospitalization as compared to prior waves, would argue against this suggestion. Further, Dr. Bhattacharya also suggested that "variants with a small infectivity advantage – but no more lethality – make up a larger fraction of a smaller number of cases is an interesting scientific observation but not important for public health policy". Once again, the nationwide data from Canada would argue that this is not an "interesting scientific observation". This is a public health crisis. Dr.

Bhattacharya further argued that the dissemination of vaccines that protect against hospitalizations and deaths had resulted in no increase in deaths during this most recent wave because of the deployment of vaccine to the vulnerable older population in Canada. This has proved to be an incorrect assumption as deaths are now increasing in Canada once again (source: Government of Canada COVID-19 daily epidemiology update April 26, 2021).

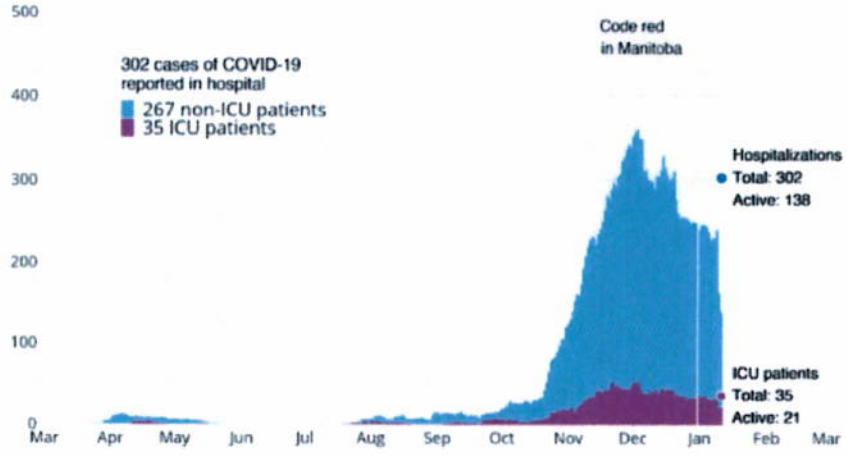


Dr. Bhattacharya also argues that the implementation of harsh lockdowns (while not discussing other non-lockdown NPIs) by Manitoba are unlikely to work to limit Covid-19 infections. Case counts and hospitalizations in Manitoba during the second wave would suggest that implementation of Code Red measures correlated with decreasing trends in both categories (source: Winnipeg Free Press and Manitoba Health)



Hospitalizations and ICU patients for COVID-19 cases in Manitoba

Shared Health counts active and long-term COVID-19 patients, those who are recovered and no longer considered contagious, separately.



WINNIPEG FREE PRESS — SOURCE: MANITOBA HEALTH (2021-01-12)

SARS-CoV-2 Virology – "Lockdowns" did not lead to the development of variants of concern

Genomic surveillance and epidemiological analysis of virus transmission patterns can help facilitate the identification of new variants where mutations have resulted in behavioral changes in the virus (e.g. transmission, virulence, immune evasion). Mutations occur frequently in RNA viruses, including coronaviruses, during the process of viral replication within an infected cell, where new copies of the virus are generated. These mutations are random events that in many cases may have no effect on the behavior of the virus. Mutations must be selectively advantageous for a variant to spread to high frequencies. However, variants can arise where the mutations impart competitive advantages to the virus including enhanced viral replication, transmission or immune evasion. Variants of concern are defined by the US Centers for Disease Control and Prevention as "A variant for which there is evidence of an increase in transmissibility, more severe disease (e.g.increased hospitalizations or deaths), significant reduction in neutralization by antibodies generated during previous infection or vaccination, reduced effectiveness of treatments or vaccines, or diagnostic detection failures". In Canada, we have seen the introduction and community transmission of three variants of concern, B.1.1.7, B.1.351 and P.1.

There has been considerable discussion on what led to the emergence of the variants of concern. It is again imperative to appreciate that RNA virus mutations occur through the process of viral replication, which requires specific machinery found within a host cell (e.g. human, nonhuman animal). Lauring and Hodcroft, experts in viral genomic surveillance, recently postulated that the selection of a variant at the population level was likely not driven by host antibodies because there are not sufficient numbers of immune individuals to push evolution of the virus in a given direction [1]. The authors also postulate that B.1.1.7 may have emerged in a chronically infected

patient due to the accumulation of mutations prior to its initial detection in early September and suggestive of prior evolution. Rambaut and colleagues provided a report on their preliminary genomic characterization of B.1.1.7 in December 2020 [2]. The authors, who are experts in genomic surveillance, highlighted that the accumulation of 14 amino-acid replacements found within B.1.1.7 prior to the initial detection of this variant was thus far unprecedented in the pandemic. In contrast to this, the authors noted that most branches in the global SARS-CoV-2 phylogenetic tree had shown relatively few mutations with a fairly consistent rate of accumulation over time (~1-2 nucleotide changes per month). However, prior studies of chronic SARS-CoV-2 infections in immunodeficient or immunocompromised patients have demonstrated high rates of mutation accumulation over short periods of time [3, 4]. Thus, the evolutionary dynamics and selective pressures exerted upon the virus population within such patients are likely very different from those found during a typical infection. Kemp and colleagues characterized the evolution of SARS-CoV-2 in a chronically-infected immunocompromised patient following multiple therapeutic treatments [5]. Multiple treatment courses with remdesivir during the first 57 days resulted in little change within the viral population; however, convalescent plasma therapy resulted in large population shifts and the emergence of a dominant variant. This demonstrated a strong selection for viral variants with reduced susceptibility to neutralizing antibodies following treatment within an immunosuppressed individual that had a chronic infection and was being treated with convalescent plasma therapy. The authors also clearly state that the "…effects of convalescent plasma on virus evolution found here are unlikely to apply in immunocompetent hosts in whom viral diversity is likely to be lower owing to better immune control". Generally, there is relatively limited within-host variation reported for SARS-CoV-2 over the course of infection [6-8]. However, factors such as prolonged infection and immunodeficiencies could result in selective pressures not encountered within those that are immune-competent. Thus the strongest evidence to date suggests that prolonged infections and compromised immune system functions likely exert selective pressures on SARS-CoV-2 resulting in a more extensive genetic changes than found during typical infections.

Covid-19 clinical symptom onset and diversity

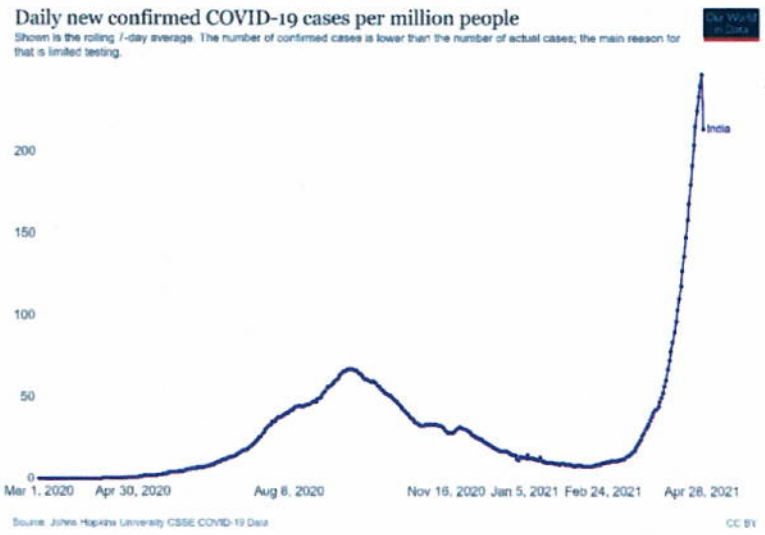
Dr. Bhattacharya argues in his second report that symptom checks are an effective means to prevent the spread of COVID-19.

Covid-19 has a diverse range of clinical presentations that range from asymptomatic infections to severe and fatal disease. The presentation of symptoms is variable within increasing severity of illness associated with older age and/or underlying health complications. Symptom and wellness checks, including temperature screening, to identify have been employed to identify infected individuals. It should be appreciated that pathogenic viruses are often able to evade early innate immune responses, the early warning system arm of our immune system that can broadly recognize different microbes. In regards to viruses, this can take the form of specific viral proteins that can inhibit or dampen these early warning systems through direct interactions. Coronaviruses can dampen the early activation of interferons, defensive molecules in our immune systems which have a central role in antiviral responses. Thus, during the early stages of viral infection, SARS-CoV-2 and other coronaviruses are able to evade early immune system

recognition and enhance infectivity. Malenfant and colleagues reported on the frequency of symptoms associated with Covid-19 in healthcare workers from March to April 2020 [9]. The authors demonstrated that Covid-19 presented with a broad spectrum of mild symptoms. While cough (51%), fever (41%), myalgia (38%) and headache (30%) were the most common initial symptoms reported, one-third of the respondents did not report fever or cough as one of their symptoms and nearly half (49%) continued to work while experiencing symptoms, some for several days. In an investigation of over 1,000 hospitalized patients, 44% of patients had fever upon admission though the half of these patients (22%) had very mild elevations in body temperature (37.6 to 38 °C) [10]. Further, according to the Clinician Guide for Covid-19 signs, symptoms and severity of disease from the Government of Canada, clinical symptoms among older adults (≥65 years old) and those with underlying health conditions may be atypical or subtle [11]. It must therefore be appreciated that symptoms are highly variable in regards to both type and severity across infected individuals and thus screening alone as a measure of case identification would likely lead to many missed cases of infection.

Herd immunity and vaccines

In January 2021, Dr. Bhattacharya co-authored an opinion piece in *The Print* [12] where he and his co-author discussed pre-existing Covid-19 immunity from prior infections within the Indian population.. In this piece Bhattacharya suggests that mathematical modelling demonstrated that "more than 50% of the Indian population may have developed immunity". Further, he states that this is corroborated by serological tests by Thyrocare which suggested that nature had silently immunized 70% of the population. This contrasts with data released on March 30, 2021, that found IgG antibodies against either the N or S1-RBD virus proteins in 26% of samples [13]. Now, it must also be considered that antibody data from Manaus, Brazil, was suggested to have potentially over-estimated seroprevalence in the population and thus arguing that immunity within the population had not actually reached the purported "herd immunity" threshold of 60-70%. If this is the case, seroprevalence data from India could also be over-estimating the actual level of SARS-CoV-2-specific IgG antibodies in the population. Over the past few weeks, India has faced a devastating wave of Covid-19 that includes broad transmission of both B.1.1.7 and a new variant of interest, B.1.617.



The herd immunity threshold (HIT) is calculated as:

$$HIT = 1 - 1/R_0$$

Where R_0 for SARS-CoV-2 has been estimated to be 2-4

Thus, HIT = 50-75%

However, increased transmissibility of variants of concern (e.g. B.1.1.7 where transmissibility is estimated to be ~50% greater) will increase the R_0 and thus drive HIT upwards. Further confounding this concept as explained by Randolph and Barreiro, "It relies on several key assumptions, including homogeneous mixing of individuals within a population and that all individuals develop sterilizing immunity—immunity that confers lifelong protection against reinfection—upon vaccination or natural infection. In real-world situations, these epidemiological and immunological assumptions are often not met, and the magnitude of indirect protection attributed to herd immunity will depend on variations in these assumptions" [14].

Thus, it must be appreciated that even with widespread transmission in regions such as Brazil and India, healthcare systems have become overwhelmed and mortality continues to increase. Randolph and Barreiro also suggested that, "Particularly in the context of attaining herd immunity to SARS-CoV-2, a regard for finite healthcare resources cannot be overstated, as this policy inherently relies on allowing a large fraction of the population to become infected. Unchecked, the spread of SARS-CoV-2 will rapidly overwhelm healthcare systems. A depletion in healthcare resources will lead not only to elevated COVID-19 mortality but also to increased all-cause mortality. This effect will be especially devastating for countries in which hospitals have limited surge capacity, where minimal public health infrastructure exists, and among vulnerable communities, including prison and homeless populations". These comments are particularly prescient given the ongoing healthcare infrastructure and resource limitations that have been encountered in both India and Brazil.

In contrast, use of vaccination and restrictions in areas such as the UK and Israel have resulted in decreasing transmission and hospitalizations, in spite of B.1.1.7 circulation. Krammer and

colleagues recently investigated the effect of vaccines on previously infected individuals [15]. Their study involved 110 participants with or without pre-existing SARS-CoV-2 immunity. Vaccinees with pre-existing immunity developed antibody titers 10-45 times as high as those without pre-existing immunity at the same time point following the first vaccine dose. A second dose of vaccine in the Covid-19 survivors had no further enhancement on antibody titers. While vaccinees with pre-existing immunity had higher frequencies of local and systemic side effects, no severe adverse events were reported.

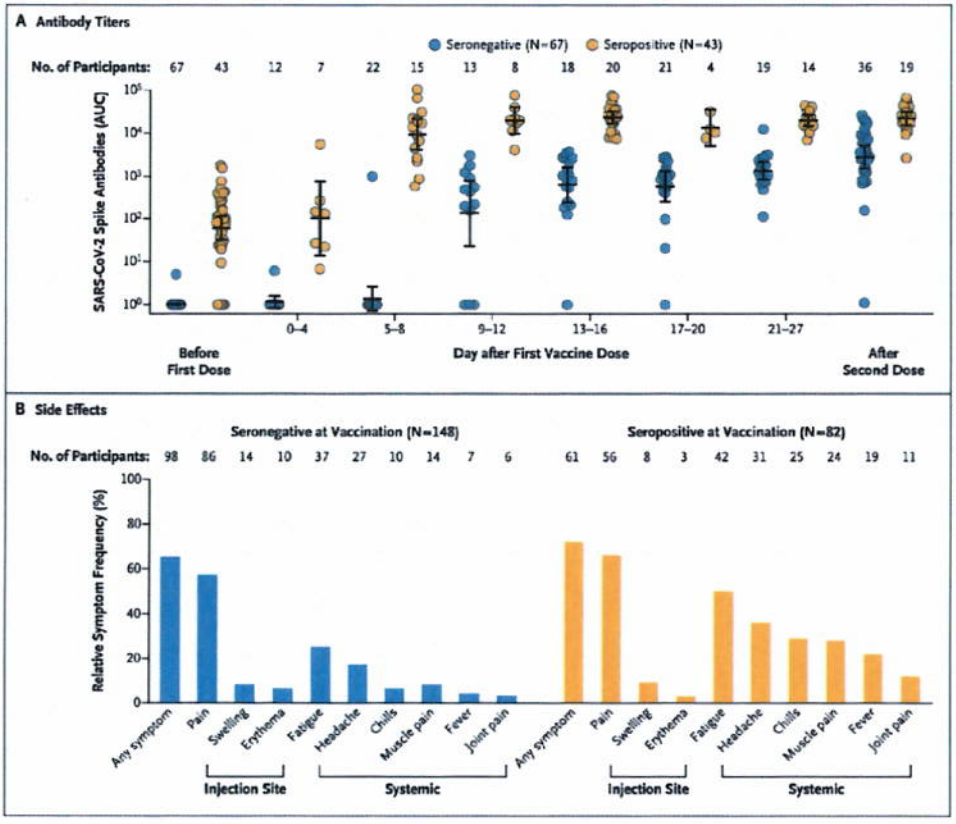


Figure: Immunogenicity and Reactogenicity of SARS-CoV-2 RNA Vaccines. Panel A shows the quantitative SARS-CoV-2 spike antibody titers (assessed by means of enzyme-linked immunosorbent assay and expressed as area under the curve [AUC]) for 110 participants. Some participants with preexisting immunity had antibody titers below detection (AUC of 1) at the time point before vaccination. Geometric means with 95% confidence intervals (not adjusted for multiple testing) are shown. Panel B shows the relative frequency of vaccine-associated side effects after the first vaccine dose (230 participants). The local side effects occurred with similar frequency among participants with preexisting immunity and among those without preexisting immunity, whereas the systemic symptoms were more common among participants with preexisting immunity. The bars represent the relative frequency of each symptom, and the numbers at the top of the graph represent the absolute numbers for a given symptom, with a given participant possibly having more than one symptom (Krammer F. et al. *NEJM.* 384: 1372-1374).

A similar investigation by Saadat et al. noted similar trends in antibody responses within vaccinees with pre-existing immunity as compared to their naïve counterparts [16]. The National Advisory Committee on Immunization (NACI) recommendation on this matter is as follows [17]:

NACI recommends that a complete series with a COVID-19 vaccine may be offered to individuals in the authorized age group without contraindications to the vaccine who have had previously PCR-confirmed SARS-CoV-2 infection. In the context of limited vaccine supply, initial doses may be prioritized for those who have not had a previously PCR-confirmed SARS-CoV-2 infection. (Discretionary NACI Recommendation)

Summary of evidence and rationale:

- Testing for previous SARS-CoV-2 infection is not needed prior to COVID-19 vaccination.
- Currently, there is a lack of evidence on potential differences in vaccine efficacy or safety between those with and without prior evidence of SARS-CoV-2 infection. In COVID-19 vaccine clinical trials to date, individuals with PCR-confirmed SARS-CoV-2 were excluded and there were only a small number of trial participants with serologic evidence of previous infection (IgG+) who had confirmed symptomatic COVID-19 during the trials, therefore efficacy in this population is uncertain.
- The immune response to SARS-CoV-2, including duration of immunity, is not yet well-understood. Reinfections with SARS-CoV-2 have been reported and research to establish the severity, frequency, and risk factors of reinfection with SARS-CoV-2 is ongoing.
- In the context of limited supply, to allow for the protection of a larger number of at-risk individuals, vaccination with a COVID-19 vaccine may be delayed for 3 months following a PCR-confirmed infection, as reinfections reported to date have been rare within the first three months following infection.
- However, if challenging from a feasibility perspective, jurisdictions may elect to disregard prior PCR-confirmed SARS-CoV-2 infection status and vaccinate everyone in a given target group.
- As a precautionary measure and in light of the need to be able to monitor for COVID-19 vaccine adverse events without potential confounding from symptoms of COVID-19 or other co-existing illnesses, and to minimize the risk of transmission of COVID-19 at an immunization venue, NACI recommends that it is prudent to wait until all symptoms of an acute illness are completely resolved before vaccinating with COVID-19 vaccine, as well as ensuring that the individual is no longer considered infectious based on current criteria.
- NACI will continue to monitor the evidence regarding vaccination in those previously infected with SARS-CoV-2 and will update recommendations as needed.

In closing, there are numerous questions that remain regarding the logic and feasibility of a natural infection-based herd immunity approach to ending the Covid-19 pandemic. While herd immunity through means other than vaccination has yet to be demonstrated for any infectious disease, there are additional concerns given our increasing understanding of SARS-CoV-2. In particular, herd immunity is impacted by behavioral, biological and environmental variables and thus should be viewed as a continuous rather

than binary (yes/no) threshold. Lastly, given the complex situations in Brazil and India where morbidity and mortality within the population from the latest pandemic wave have been exacerbated by fragile healthcare infrastructure and limitations to routine supplies (e.g. sedatives, oxygen) that will impact those requiring treatment for Covid-19 as well as other communicable and non-communicable diseases. The success of vaccination programs throughout history that have also employed nonpharmaceutical interventions and mitigation strategies, including those seen recently in the UK and Israel, argue for their use in combatting Covid-19.

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