

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

BETWEEN:

**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, 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 MANITOBA**

Respondents.

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**AFFIDAVIT OF THOMAS WARREN  
SWORN MARCH 30, 2021**

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**JUSTICE CENTRE FOR CONSTITUTIONAL FREEDOMS  
D. Jared Brown / Allison Kindle Pejovic / Jay Cameron**



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CHRISTIAN CHURCH OF MORDEN, BIBLE BAPTIST CHURCH,  
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MANITOBA, 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  
MANITOBA**

Respondents.

**AFFIDAVIT OF THOMAS WARREN**

I, THOMAS WARREN of the City of Oakville, in the Province of  
Ontario,

MAKE OATH AND SAY 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 do verily believe to be true.



2. I am an Infectious Diseases specialist and Medical Microbiologist currently practicing in the locations of Oakville, Milton and Georgetown in Ontario.
3. I obtained my Doctor of Medicine (MD) from the University of Western Ontario in 2005, after which I completed a three-year residency in Internal Medicine through the University of Ottawa. Following my Internal Medicine residency, I completed a Fellowship in Infectious Diseases and a second residency in Medical Microbiology, both at the University of Toronto. During my residencies and fellowship I regularly taught medical students and junior residents.
4. I have practiced in these specialty areas for ten (10) years. As part of my clinical practice, I teach through my appointment as an Assistant Clinical Professor (Adjunct) at McMaster University in Hamilton, ON. This includes supervising Infectious Diseases Clinical Rotations for physician assistant students, medical students, and Infectious Diseases fellows.
5. I am currently enrolled in a Master's of Science (Epidemiology) at the London School of Hygiene and Tropical Medicine, University of London, with an expected completion date of 2022. Areas of study include the framework for understanding the epidemiology of infectious diseases and the mathematical theory underlying epidemiological studies.
6. In my medical microbiology residency I was trained to develop, use and interpret reverse transcription polymerase chain reaction (RT-PCR) testing. I have practiced as a microbiologist for ten years in a microbiology laboratory that uses a variety of PCR tests. As an infectious diseases consultant I interpret PCR test results in the context of clinical care.

7. A copy of my curriculum vitae is attached hereto and marked as **Exhibit “A”**.

8. The Applicants’ counsel contacted me about providing expert testimony in response to the Affidavit of Dr. Brent Roussin, and the Affidavits and expert reports of Dr. Jared Bullard and Dr. Jason Kindrachuk. Specifically, I have been asked to respond to their evidence in respect of important issues surrounding the virus SARS-CoV-2 and Covid-19 disease, specifically: their description, PCR testing, and asymptomatic transmission.

9. A copy of my expert report is attached hereto and marked as **Exhibit “B”**.

10. I acknowledge that in preparing this report and providing expert evidence, the Applicants’ counsel explained that my role is to assist the court to determine the matters in issue. I further acknowledge that it is my duty to provide evidence that is fair, objective and non-partisan and to opine only on matters that are within my area of expertise. This duty prevails over any obligation that I may owe to any party on whose behalf I am engaged.



11. I make this affidavit *bona fide*.

**SWORN** 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 30th day of )  
 March, 2021. )

*L. Laik*

Commissioner of Oaths in and for  
 the Province of Manitoba

My Commission Expires: *July 8/21*

*Thomas Warren*

THOMAS WARREN

THIS IS EXHIBIT "A" TO THE  
AFFIDAVIT OF THOMAS WARREN  
SWORN BEFORE ME IN THE CITY  
OF WINNIPEG THIS 30<sup>th</sup> DAY  
OF MARCH, 2021

*L. Loeb*

A COMMISSIONER OF OATHS IN AND  
FOR THE PROVINCE OF MANITOBA  
MY COMMISSION EXPIRES: *July 8/21*



# Thomas A. Warren, MD

## Employment

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- 2011 - **Infectious Diseases consultant & Medical Microbiologist**  
Halton Healthcare, Oakville ON
- 2010-2011 **Internal Medicine specialist – locum coverage**  
St. Michael's Hospital, Toronto ON  
Hamilton Health Sciences, Hamilton ON  
Lakeridge Health, Oshawa ON
- 2010-2011 **University of Toronto**  
Department of Laboratory Medicine & Pathobiology, Toronto ON  
*Resident, Medical Microbiology*
- 2008-2010 **University of Toronto**  
Department of Medicine, Division of Infectious Diseases, Toronto ON  
*Fellow, Infectious Diseases*
- 2005-2008 **University of Ottawa**  
Department of Medicine, Ottawa ON  
*Resident, Internal Medicine*
- 1997-2003 **University of Western Ontario**  
Department of Medicine, London ON  
*Computer Programmer & Web Developer*

## Education

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- 2018 - **London School of Hygiene and Tropical Medicine, University of London**  
*Master's of Science (Epidemiology)*  
Expected Completion 2022
- 2010-2011 **Royal College of Physicians & Surgeons of Canada**  
*Residency in Medical Microbiology*

2008-2010	<b>Royal College of Physicians &amp; Surgeons of Canada</b> <i>Fellowship in Infectious Diseases</i>
2005-2008	<b>Royal College of Physicians &amp; Surgeons of Canada</b> <i>Residency in Internal Medicine</i>
2001-2005	<b>University of Western Ontario</b> Schulich School of Medicine & Dentistry <i>Doctor of Medicine</i>
1997-2001	<b>University of Western Ontario</b> <i>Bachelor of Science - Honors Microbiology &amp; Immunology</i> ( <i>Scholar's Electives Program</i> ) Graduated With Distinction

### Continuing Medical Education

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2018	<b>IDEAS Foundations of Quality Improvement Program</b> May 30 McMaster University Hamilton, ON
2018	<b>Clinical Teaching Fundamentals</b> January – March McMaster University Hamilton, ON

### Peer-Reviewed Publications

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2015	<b>Warren T</b> , Lau R, Ralevski F, Rau N, Boggild AK. Fever in a visitor to Canada: a case of mistaken identity. <i>J Clin Microbiol.</i> 53:1783-1785.
2012	<b>Warren TA</b> , Yau Y, Ratjen F, Tullis E, Waters V. Serum galactomannan in cystic fibrosis patients colonized with <i>Aspergillus</i> species. <i>Medical Mycology.</i> 2012; 50: 658-660.
2010	<b>Warren TA</b> , McTaggart L, Richardson SE, Zhang SX. <i>Candida bracarensis</i> Bloodstream Infection in an Immunocompromised Patient. <i>Journal of Clinical Microbiology.</i> 2010; 48: 4677–4679.



## Abstracts & Conference Presentations

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- 2011      **Warren TA**, Yau Y, Waters V.  
Serum galactomannan in cystic fibrosis patients colonized with *Aspergillus* species.  
Poster session presented at: Association of Medical Microbiology and Infectious Disease (AMMI) Canada 2011 Annual Conference  
2011 April 7-9; Montreal, QC.
- 2010      **Warren TA**, Yau Y, Waters V.  
Serum galactomannan in cystic fibrosis patients colonized with *Aspergillus* species.  
Poster session presented at: North American Cystic Fibrosis Conference  
2010 October 21-23; Baltimore, MD.
- 2010      **Warren TA**, Govindapillai S, Tullis E, Devlin HR, Ferris W, Matukas LM.  
Evaluation of Etest Combination Testing of Antibiotics Against Isolates from Patients with Cystic Fibrosis.  
Poster session presented at: 50th Interscience Conference on Antimicrobial Agents and Chemotherapy  
2010 September 12-15; Boston, MA.
- 2010      **Warren TA**, Rotstein C, Cole EH, Singer LG, Keshavjee S4, Husain S.  
Posaconazole therapy in solid organ transplant recipients refractory to or intolerant of standard therapy.  
Poster session presented at: Canadian Society for Transplantation Annual Conference  
2010 August 12-15; Vancouver, BC.
- 2010      **Warren TA**, McTaggart L, Zhang S. *Candida bracarensis*  
Blood Stream Infection in an Immunocompromised Patient: Case Report.  
Poster session presented at: Focus on Fungal Infections  
2010 March 3-5; New Orleans, LA.
- 2007      **Warren TA**, McCarthy AE.  
A Ten-Year Retrospective Study of Vaccination Rates, Prophylactic Antibiotic Use, Serious Infection and Overwhelming Postsplenectomy Sepsis Rates in Splenectomized Patients.  
Poster session presented at: Annual Meeting of the Infectious Diseases Society of America  
2007 October 4-7; San Diego, CA.

## Awards

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2011	<b>Best Student Poster Award – 2011 Annual Conference</b> Association of Medical Microbiology and Infectious Disease (AMMI) Canada Montreal, QC
2010	<b>ASM ICAAC Infectious Diseases Fellows Grant</b> 2010 Interscience Conference on Antimicrobial Agents and Chemotherapy Boston, MA
2008	<b>Internal Medicine CanMeds Award for Communication</b> University of Ottawa, Department of Medicine Ottawa, ON
2006	<b>Resident Research Day Award of Excellence – PGY1</b> University of Ottawa, Department of Medicine Ottawa, ON
2001	<b>Laurene Paterson scholarship</b> University of Western Ontario London, ON
1997-2001	<b>Dean's Honor List</b> University of Western Ontario, Faculty of Science London, ON
1997	<b>Western Scholarship of Excellence</b> University of Western Ontario London, ON

## Appointments

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2013 -	<b>McMaster University</b> Assistant Clinical Professor (Adjunct) Department of Medicine, Faculty of Health Sciences Hamilton, ON
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## Teaching

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2012-2021	<b>Infectious Diseases – Clinical Rotations</b> Supervised physician assistant students, medical students, residents and infectious diseases fellows from the University of Toronto and McMaster University Oakville, ON
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2009	<b>Pathobiology of Disease</b> <i>Taught microbiology to second year medical students</i> University of Toronto Toronto, ON
2008	<b>Pathobiology of Disease</b> <i>Taught microbiology to second year medical students</i> University of Toronto Toronto, ON
2008	<b>Physical Skills Development Course</b> <i>Taught physical exam skills to first year medical students</i> University of Ottawa Ottawa, ON

## Memberships

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Association of Medical Microbiology and Infectious Diseases Canada

Canadian Medical Association

Canadian Medical Protective Association

College of Physicians and Surgeons of Ontario

Ontario Medical Association

Royal College of Physicians and Surgeons of Canada

THIS IS EXHIBIT "B" TO THE  
AFFIDAVIT OF THOMAS WARREN  
SWORN BEFORE ME IN THE CITY  
OF WINNIPEG THIS 30th DAY  
OF MARCH, 2021



A COMMISSIONER OF OATHS IN AND  
FOR THE PROVINCE OF MANITOBA  
MY COMMISSION EXPIRES: July 31/21



**THOMAS WARREN M.D. – RESPONDING EXPERT REPORT ON SARS-Co-V-2 and  
COVID-19, PCR TESTS, AND ASYMPTOMATIC TRANSMISSION**

March 29, 2021

## **SARS-CoV-2 and COVID-19 – Response to Dr. Roussin’s Description**

Dr. Roussin explains and describes SARS-CoV-2 and COVID-19 in his affidavit (paragraphs 20-26). Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is a novel coronavirus. There are six other coronaviruses that are known to infect humans. Four coronaviruses, HCoV-NL63, HCoV-HKU1, HCoV-229E, and HCoV-OC43 circulate worldwide and together are the second most common cause of the common cold<sup>1,2</sup>. Severe acute respiratory syndrome coronavirus 1 (SARS-CoV-1) infected 8096 people in 2003 resulting in 774 deaths<sup>3</sup>. After 2003 there has not been any further human to human transmission. Middle East respiratory syndrome coronavirus (MERS-CoV) was first identified in humans in 2012<sup>4</sup>. MERS-CoV continues to cause sporadic infection and outbreaks in the Arabian peninsula, as well as occasional other cases and outbreaks in other parts of the world linked to travelers to the Arabian peninsula<sup>5</sup>.

Bats were the source of SARS-CoV-1<sup>6</sup> and are known to be a natural reservoir for related coronaviruses<sup>7,8</sup>. SARS-CoV-2 was likely circulating in bats for decades<sup>9</sup>. In late 2019, SARS-CoV-2 was first detected in humans and is established as the cause of the disease now designated coronavirus disease 2019 (COVID-19). Approximately 10-20% of persons with SARS-CoV-2 infection are asymptomatic<sup>10,11</sup>. In those who are symptomatic, there is a wide range of illness from those with mild symptoms such as runny nose to those with severe disease affecting particularly the respiratory tract with high mortality<sup>12</sup>. Most people with SARS-CoV-2 infection are asymptomatic or have mild-moderate symptoms not requiring hospitalization. In one study of a relatively healthy population, those with COVID-19 requiring hospital care was < 2%, and the mortality rate was < 0.1%<sup>11</sup>.

## **What is PCR? – Response to Dr. Bullard**

Dr. Bullard's report describes PCR technology and the utility of PCR in the identification of SARS-CoV-2 and diagnosis of COVID-19. Polymerase chain reaction (PCR) is a technology to amplify DNA fragments<sup>13,14</sup>. It is widely used in molecular biology, biotechnology, and medicine. Real time reverse transcription-polymerase chain reaction (real time RT-PCR) is a modification of PCR with an additional step of reverse transcription (RT) of RNA to DNA to enable amplification of an RNA target rather than a DNA target.



PCR is a relatively quick and inexpensive process that is highly sensitive. The process detects genetic material (DNA or RNA), even in minute quantities, and then copies it in a series of steps (cycles) that is usually done by a machine in a fully automated process. Each cycle doubles the amount of target DNA, and the newly created DNA is labeled with a fluorescent dye for detection. If a certain level of fluorescence is surpassed and detected by the machine, the test is considered positive. The cycle of the test that passes this threshold is called the cycle threshold (Ct).

PCR is commonly used in microbiology laboratories for the diagnosis of infectious diseases. While PCR can be the best diagnostic tool to diagnose many infections, it does have important limitations that also need to be considered. It is the limitations of PCR that are inadequately addressed in Dr. Bullard's report.

### **Limitations of PCR: Response to Dr. Bullard**

The World Health Organization (WHO) recognizes the limitations of PCR and advises that "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"<sup>15</sup>. The WHO guidance goes on to say that "disease prevalence alters the predictive value of test results; as disease prevalence decreases, the risk of false positive increases. This means that the probability that a person who has a positive result (SARS-CoV-2 detected) is truly infected with SARS-CoV-2 decreases as prevalence decreases, irrespective of the claimed specificity."

In his affidavit, Dr. Bullard states, "Regardless of the particular Ct value, a positive RT-PCR result represents a true positive case of the SARS-CoV-2 virus." (paragraph 10). It is true that a positive RT-PCR result represents the identification of SARS-CoV-2 virus fragments, but a positive RT-PCR result does not necessarily indicate the entire virus is present, replication competent virus is present, or the patient has COVID-19. If the entire virus is not present in the person, or the virus is not replication competent, then the person is not infectious.

A recent study from Singapore<sup>16</sup> showed that the higher the Ct value, the larger degree of viral fragmentation and the less likely that an entire viral genome is actually present. In other words, the higher the Ct, the more likely it is that only bits of virus are being



detected, and less likely that entire virus is present in the patient. Only complete virus particles can be replication competent and therefore infectious and transmissible.

Dr. Bullard asserts that “while less likely, some individuals might still be infectious even at a higher Ct value” (paragraph 12), and “higher Ct values are associated with a lower likelihood of growing SARS-CoV-2 in cell culture, but this cannot rule that the person was or was not infectious at the time of sample collection.” (lines 164-166) Many high quality studies published in leading peer-reviewed journals<sup>17-23</sup>, including Dr. Bullard’s own study published in *Clinical Infectious Diseases*<sup>24</sup>, have convincingly shown that the higher the Ct, the less likely replication-competent virus (infectious virus) can be detected through cell culture. An editorial in *Clinical Infectious Diseases* regarding Dr. Bullard’s study concluded “that PCR positivity is likely not a reliable surrogate marker for determining the infectious status of COVID-19 patients”. A systematic review on the topic, also published in *Clinical Infectious Diseases*, concluded that test results “with high cycle threshold are unlikely to have infectious potential.”<sup>25</sup>

That a positive test does not necessarily indicate infectiousness is indicated by recommendations for stopping isolation in persons previously positive for SARS-CoV-2. The WHO<sup>26-27</sup>, CDC<sup>28</sup>, and Canadian jurisdictions<sup>29</sup> recommend discontinuing isolation of persons with COVID-19 ten days after symptom onset, and in persons who have tested positive for SARS-CoV-2 without symptoms ten days after their first positive RT-PCR result, even though it is well established that many persons in these groups will continue to have positive RT-PCR results after those time frames. Those guidelines recommend against RT-PCR testing in these groups because it is known that positive tests in these groups does not indicate infectiousness. This is a concrete application of the evidence that late in the course of SARS-CoV-2 infection, which corresponds to increasing Ct, there is no risk of transmission. In another example, Ontario uses point-of-care tests, that are less sensitive than RT-PCR, to rule out SARS-CoV-2 infection<sup>30</sup> in persons who are symptomatic without known contact with a positive case. In this case, even though a more sensitive test such as RT-PCR might detect more positives, a less sensitive test<sup>31</sup> is sufficient to rule out significant infection.

Dr. Bullard states that “It is challenging to know where in their disease trajectory an individual with a high Ct is, thus it remains essential to identify them as a case, at a minimum to identify and investigate contacts in order to minimize secondary SARS-CoV-2 spread.” (lines 209-212) What the evidence in fact shows, as outlined below, is that the higher the Ct value, the more likely it is that a person is in the later stages of the



infection, and therefore less infectious. The nearer the Ct value approaches 40, the closer the likelihood that the patient is infectious approaches zero.

A report from the Emerging Sciences Group of the Public Health Agency of Canada<sup>32</sup> concludes that in symptomatic persons there is “a peak in viral load ranging from just before to during the first week after onset of illness” and in asymptomatic persons “viable virus and viral RNA was highest during the first week of infection and declined in subsequent weeks.” In persons that are asymptomatic or mildly symptomatic, late in the course of illness the Ct value is higher<sup>21</sup> and viable virus cannot be detected though cell culture. The likelihood of a positive cell culture correlates with disease severity<sup>33</sup>, and therefore risk of infectiousness correlates with disease severity.

Dr. Bullard further asserts that “a positive test at a higher Ct value (indicating a lower viral load) may result because the individual is only at the early stages of the COVID-19 disease.” (paragraph 12); however, a January 2021 peer-reviewed study<sup>34</sup> published in the *Journal of Clinical Medicine* clearly showed that the Ct value early in the course of illness is significantly lower than the Ct value late in the course of illness. The Ct value of pre-symptomatic persons has been shown to be low, and not be significantly different from symptomatic persons; in one peer-reviewed study published in the *New England Journal of Medicine*, the Ct value was 23.1 for pre-symptomatic persons compared to 24.8 in persons with typical symptoms<sup>10</sup>. There is a clear association between Ct value and stage of infection; the higher the cycle threshold<sup>33</sup>, the more likely the patient is in the later stages of the infection, and the less likely the patient is infectious or at risk of transmitting the virus to another person.

### **SARS-CoV-2 infection versus COVID-19 disease – Response to Dr. Bullard**

It is important to recognize the difference between SARS-CoV-2 infection and COVID-19 disease. This is an important distinction that is made with many other infections. As noted in a *British Medical Journal* editorial: “Unusually in disease management, a positive test result is the sole criterion for a Covid-19 case. Normally, a test is a support for clinical diagnosis, not a substitute.”<sup>35</sup> In other words, for COVID-19, a positive test is sufficient to make the diagnosis, which is not done in other infections that are similarly mild and short-lived.

Dr. Bullard states that RT-PCR has “a specificity of greater than 99.9% (less than 1 in 1000 will have a false positive result)” (lines 135-136) and uses that to conclude that “If



the individual tests positive, they have the SARS-CoV-2 pathogen detectable and have been diagnosed with COVID-19.” (lines 217-218) A positive test means there is a 99.9% likelihood that the person has or recently had SARS-CoV-2 (virus) in their body; however, it does not mean that the person is infectious or that they have COVID-19 disease (i.e. symptoms). If we wanted to define specificity as diagnosing infectiousness or disease (i.e. symptoms or pathology), then the specificity of RT-PCR would be dramatically lower. This distinction can be better understood if we look at how PCR is used in other infections.

To cite just a few examples, positive PCR tests for Group A Streptococcus, Salmonella, E. coli O157, Campylobacter, C. difficile, Epstein-Barr Virus (EBV), Cytomegalovirus (CMV) do not necessarily indicate disease. In the absence of symptoms or other tests indicating pathological effects, persons with positive PCR tests for those infections are not considered to have disease and they are usually not treated. The distinction between infection and disease is important. Over 50% of adult Canadians will be infected with EBV or CMV for most of their life; once acquired, those infections persist lifelong and PCR tests can detect those viruses, to varying degrees, throughout their lifetime. It would be inaccurate to say that over 50% of Canadians have disease associated with those viruses even though they can be detected by PCR.

A positive PCR result also does not necessarily indicate infectiousness. At any time, about 10% of school-aged children will have throat swabs positive for Group A Streptococcus (GAS)<sup>36,37</sup>. GAS can cause pharyngitis (strep throat), scarlet fever, rheumatic fever, and necrotizing fasciitis (flesh eating disease), but a positive test in an asymptomatic person is not considered significant in most cases.

Even a positive RT-PCR result in a person living with HIV does not necessarily mean that the person is considered infectious. According to Canadian law<sup>38</sup>, “the combined effect of condom use and low viral load precludes a realistic possibility of transmission of HIV”. Similarly, the Ontario Court of Appeal stated that “viral loads below a defined level, standing on their own, are sufficient to negate the realistic possibility of HIV transmission”<sup>39</sup>.

The presence of SARS-CoV-2 virus as detected by PCR is necessary but not sufficient to indicate either infectiousness or COVID-19 disease properly defined. If a true positive is defined as the presence of complete virus, or replication competent virus (i.e. infectious virus) then the specificity of PCR is much lower and the number of false positives associated with PCR would be considered much higher.



### Asymptomatic transmission: Response to Dr. Roussin and Dr. Kindrachuk

The affidavit from Dr. Roussin and the affidavit and expert report from Dr. Kindrachuk do not adequately synthesize or contextualize the risk of transmission in asymptomatic persons compared to pre-symptomatic or symptomatic persons.

A *British Medical Journal* editorial concisely summarizes the risk of asymptomatic transmission: “The transmission rates to contacts within a specific group (secondary attack rate) may be 3-25 times lower for people who are asymptomatic than for those with symptoms.”<sup>35</sup> This is consistent with the conclusions from several peer-reviewed systematic reviews and meta-analyses<sup>40-43</sup>.

To further exemplify the risk of asymptomatic transmission, it is useful to look specifically at a few large or comprehensive studies. A very large study, published in a leading peer-reviewed journal (*Nature Communications*) in Wuhan China of 9,899,828 city residents found 300 asymptomatic cases but there were no positive tests amongst 1,174 close contacts of asymptomatic cases<sup>44</sup>. Similarly, in a very thorough study of 100 cases from Taiwan, published in the peer-reviewed *Journal of the American Medical Association Internal Medicine*, found that “none of the 9 asymptomatic case patients transmitted a secondary case.”<sup>45</sup>

Household transmission is one of the most important modes of transmission. In a meta-analysis of household transmission, published in the peer-reviewed *Journal of the American Medical Association Network Open*, which included 54 studies and 77 758 participants<sup>46</sup>, transmission from asymptomatic cases was 0.7% compared to 18% transmission from symptomatic cases. In other words, symptomatic transmission was roughly 25 times higher than asymptomatic transmission.

In conclusion, asymptomatic transmission does occur but the rates of transmission from asymptomatic persons is substantially less than from symptomatic persons and does not warrant being considered a significant contributor to the overall transmission burden. There is no justification to limit the activities of asymptomatic persons since the risk of transmission is negligible compared to symptomatic persons.

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