Thoughts and Concerns Regarding the New Corona Virus

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Preamble

The late winter and early spring of 2020 will be earmarked in history as the era of, “The Great Corona Virus Pandemic.” It remains undecided if this designation will be one praising the collective efforts of all to defeat an invisible foe, or if it will be remembered as a public health over reaction which precipitated an economic disaster. As this article is being written in late March to mid April 2020 it would be foolhardy to predict on which side of the equation the pandemic will be judged. However, the author has had a 35 year interest in infectious diseases and has, over the last six weeks, amassed a considerable amount of literature on the new corona virus. Reading this material has identified a recurring theme, an absence of factual information on the virus. It has led to the conclusion that the world has rushed to a premature assessment of a virus about which little is known. What follows are the authors thoughts and concerns that have justified this understanding of the corona pandemic.

The New Virus

The presumptive pandemic inducing virus now named SARS-CoV-2 (previously referred to as: 2019 nCoV) was initially identified in 3 adults with severe pneumonia admitted to a hospital in Wuhan, Hubei Province, China in late 2019. (1) The article describing the investigation of the three cases was published in the New England Journal of Medicine in January 2020. (1)

Patient 1 was a 49 year old otherwise healthy woman admitted with a cough, fever and chest discomfort. Patient 2 was a 61 year old male who presented with fever and cough and increasing respiratory distress during his hospital stay. Patient 3 was a 32 year old male with clinical signs of pneumonia. Similar to patient 2, his previous medical history was not recorded. The diagnosis of pneumonia in the three patients was confirmed by CT scans. Patients 1 and 3 were discharged following a 21 day hospital stay. Unfortunately, patient 2 died 14 days after his hospital admission. (1)

In the laboratory, bronchial fluid from the patients was cultured in human respiratory epithelial cells allowing genome sequencing, real time reverse transcriptase polymerase chain reaction (RT-PCR) and isolation techniques to be used in identifying the virus as belonging to the family of widely distributed coronaviruses known to be a cause of common cold like symptoms. (1) Its unique genome sequencing resulted in the virus being referred to as a “novel” corona virus.

The investigators admit that while their labour intensive research methods had identified the new coronavirus as the “likely” cause of the pneumonia, their study did “not fulfill Koch’s postulates”- a historical method of identifying pathogens. (1)

A little later similar procedures were used by different investigators who determined that 41 of 59 patients admitted to a Wuhan hospital with pneumonia had “laboratory confirmed 2019 nCoV infection.” (2)

It is a concern that in a short space of time a new virus considered a few days before to be a likely cause became universally accepted as the confirmed cause of the pneumonia although it had not be verified as the cause of a viral pneumonia. (3) This is worrying as there ought to be independent studies performed on patients having similar signs and symptoms but in different world - wide locations whose lower
respiratory tract samples were subjected to the same investigations as the Wuhan patients. If such studies showed any variation in results from the Wuhan ones, the credibility of the latter must be questioned. Which begs the question, “How new is this novel virus?”

**Seattle Flu Study**

According to an article in the March 10th edition of the New York Times, Dr. Helen Chu, Director of the Seattle Flu Study decided to retrospectively investigate nasal swabs collected from flu patients for the presence of the novel coronavirus. Around the 25th February she identified its presence in a teenager with no travel history. Dr. Chu interpreted this as meaning that the virus was already endemic in Washington State.

It is generally accepted that coronaviruses are distributed broadly among humans and are associated with wide range of diseases. (1) Therefore, it is possible that this “new” virus is “novel” only because it has recently been identified. After all, the New World existed long before Columbus sailed west.

**An Old Virus and Its Mutations**

In the March 27th edition of the Ottawa Citizen, Dr. Francis Collins, formerly Head of the Human Genome Project, suggested that the new corona virus established a harmless symbiotic relationship with humans many centuries ago and that it gradually changed over many years to cause disease. Since the virus requires a live human host to survive, it would not be in the evolutionary best interests of the virus if the illnesses caused by such mutations were always lethal or induced immunity to it. This raises the question, “How pathogenic is this virus?”

**Koch’ Postulates**

In 1890 Koch, a German physician, described four conditions which should be met before a microorganism could be deemed a human pathogen. Over the years it was realised that these postulates were not applicable to viruses which, as intracellular bodies, were not easily purified nor cultured. While it can be difficult to prove that a virus caused a disease, it was appreciated that a need existed for rules of logic which would prevent the correlation of a virus with an illness as proof that it was the etiological agent. (4)

Koch’s original four conditions have been expanded into eight criteria that preferably should be satisfied before a causal relationship is established between a virus and infection. These are:

- Isolation of the virus in culture,
- Repeated recovery of the virus from human specimens,
- Antibody response to the virus,
- Characterization and comparison with known pathogenic viruses,
- Constant association of the virus with specific illness,
- Reproduction of the clinical illness in volunteer subjects,
- Epidemiologic studies preferably longitudinal ones,
- Prevention of disease by vaccination. (4)

There are obvious difficulties in satisfying all of these “Viral” Koch’s Postulates.
Nevertheless, the first paper on the new virus warned that it did not satisfy Koch’s Postulates. (1) A later publication seemed to suggest that the conditions had been met. (2) However, the following quotation, from a recent edition of the Journal of Medical Virology, presents a different perspective.

“But the mystery has not been completely solved yet. Until there is a formal published scientific manuscript, the facts can be argued, particularly regarding causality despite these facts having been officially announced. The data collected so far is not enough to confirm the causal relationship between the new type coronavirus and the respiratory diseases based on classical Koch’s postulates or modified ones as suggested by Fredricks and Relman. The viral-specific nucleic acids were only discovered in 15 patients, and successful viral culture was extremely limited to only a few patients. There remains considerable work to be done to differentiate between colonization, shedding, and infection. Additional strains of 2019-nCoV need to be isolated to study their homologies. It is expected that antigens and monoclonal antibodies will be developed so serology can be used to confirm previous and acute infection status.” (5)

It is a major concern that - despite the reservations noted above - it has been overwhelmingly accepted that this “new” corona virus is a human pathogen.

**Pneumonia**

Infectious diseases usually are defined by a distinguishing set of clinical signs and symptoms. For example, measles (rubeola) is characterized by fever, runny nose, sore throat, hacking cough, red eyes, presence of white (Koplik’s) spots on the oral mucosa, and the development of an itchy rash. Mumps infects the salivary gland. Chills, headache, loss of appetite and mild to moderate fever are often present prior to swelling of the salivary glands. Both are viral infections with different signs and symptoms.

On the other hand pneumonia - a lung infection - is not a single illness. Although it can be caused by bacteria, viruses and fungi, there is a commonality of signs and symptoms. These include a cough, chest pains, chills, fever and shortness of breath. Chest x-rays or CT scans, sputum and blood specimens assist in diagnosing the particular causative organism. However, in about 50% of patients the precise organism cannot be identified. (6)

In the US approximately 1 million people per year develop pneumonia, which accounts for 40-70,000 deaths per year. It is the most common fatal infection acquired in hospital usually among patients with serious chronic diseases or immune deficiencies. (7) According to the Canadian Institute for Health Information (CIHI) in the year 2017-2018 there were almost 135,000 emergency room admissions for pneumonia, a 13% increase over the previous year. (8)

**Influenza**

Numerous viruses can cause influenza although the most common strains are influenza A and influenza B. The initial signs and symptoms are those generally associated with the common cold but quickly become more severe with chills, moderate to high fever, widespread aches and pains, and in severe cases, pneumonia. The following figures are significant.

World - wide there are an estimated 1 billion cases of influenza per year, 3-5 million cases with severe illness and 250,000 to 500,000 deaths per year. (9)
The Center for Disease Control estimates that every year in the US since 2010 there have been 9,300,000 to 45,000,000 illnesses related to the flu, 140,000-810,000 hospitalizations due to the flu, and 12,000-61,000 deaths from the flu. (10)

In Canada from 2010 the annual reported number of flu cases has varied from a low of 12,194 in 2011-2012 to a high of 55,000 in 2017-2018. In Canada as of March 21st there were 41,981 case of influenza. It is estimated that on average the flu causes 12,200 hospitalizations per year in Canada and approximately 3,500 deaths. (11)

The information above shows that from year to year there are gross variations in the number of cases diagnosed as influenza and/or pneumonia. Highs and lows are to be expected. A specific geographic region might be spared one year and, in the following year, have a dramatic increase in the number of cases. Therefore sudden spikes in the number of cases should not be a reason for undue over reaction and inevitable fear and panic. It is with this understanding that the diagnosis of COVID-19 will be discussed.

**Respiratory Tract Illnesses**

The most serious but fortunately relatively rare outcome from COVID-19 is a predilection for causing - in medically compromised hosts - a respiratory tract illness which, in turn, has the potential of causing respiratory distress, pneumonia and death. Therefore, prior to describing the somewhat non-specific signs and symptoms of COVID-19 it is appropriate to discuss respiratory tract illnesses. Within the context of this article, this is most appropriately accomplished by quoting from a 2004 publication which appeared in the Proceedings of the National Academy of Sciences.

> “Acute respiratory tract infections are responsible for considerable morbidity and mortality in humans and animals, and the costs attributable to acute respiratory tract illnesses (RTI) in humans are an important burden on the national health care budgets. A variety of viruses, bacteria and fungi are associated with RTI, with most of the viruses belonging to the families of Paramyxoviridae, Orthomyxoviridae, Picornaviridae, Adenoviridae, and Coronaviridae. In a relatively large portion of samples obtained from persons suffering from RTI, no pathogens can be detected despite the use of a wide range of sensitive diagnostic assays; even in the most comprehensive studies, a causative agent (either viral or nonviral) can be identified in only 85% of the patients. In part, this may be due to the limitations of diagnostic assays, but a portion of RTI may be caused by still unknown pathogens.” (12)

With this understanding it is entirely possible that the “new” coronavirus is one of the unknown pathogens that having been causing respiratory tract illnesses of unknown etiology for years. If this is a reasonable assumption, all current attitudes and reactions to COVID-19 must be reassessed. It is unknown to what degree this idea has been explored by authoritative sources.

**COVID-19 Suspect and Confirmed Cases**

COVID-19 is the infection presumptively related to the virus SARS-CoV-2. This infection has a range of signs and symptoms similar to those associated with the common cold and influenza. A fever, dry cough, fatigue, shortness of breath, joint or muscle pains, sore throat, headache and chills are common symptoms. Patients might be asymptomatic or progress to moderate pneumonia, severe pneumonia, respiratory distress and, in a few cases, organ failure leading to death. (13)
Although COVID-19 has no distinguishing clinical features the World Health Organization (WHO) has provided descriptions of what it would consider as a “suspect” case, a “probable” case and a “confirmed” case of COVID-19. It is appreciated that different jurisdictions might alter the following definitions to suit local conditions.

The WHO defines a **suspected** case of COVID-19 as:

A. A patient with acute respiratory illness (fever, cough and/or shortness of breath), AND a history of travel to or residence in a location reporting community transmission of COVID-19 disease during the 14 days prior to symptom onset.

OR

B. A patient with acute respiratory illness AND having been in contact with a confirmed or probable COVID-19 case in the last 14 days prior to symptom onset.

OR

C. A patient with severe acute respiratory illness AND requiring hospitalization in the absence of an alternative diagnosis that fully explains the clinical presentation.

The WHO defines a **probable** case of COVID-19 as:

A. A suspect case for whom testing for the COVID-19 virus is inconclusive.

OR

B. A suspect case for whom testing could not be performed for any reason.

The WHO defines a **confirmed** case of COVID-19 as:

A. A person with laboratory confirmation of COVID-19 infection, irrespective of clinical signs and symptoms.

The WHO defines **contact** as any one of the following during 2 days before and the 14 days after the onset of symptoms of a probable or confirmed case:

1. Face to face contact with probable or confirmed case within 1 meter and for no more than 15 minutes.
2. Direct physical contact with a probable or confirmed case.
3. Direct care for a patient with probable or confirmed COVID-19 disease without using proper protective equipment.

OR

4. Other situations as indicated by local risk assessments. (14)

There are concerns with all of those definitions. For example, the diagnosis of an acute respiratory illness is entirely subjective and will depend on numerous variables. As will the decision to admit the patient to hospital and the decision to seek an alternative diagnosis. A suspect case can become a probable case for no or any reason. As explained above severe acute respiratory distress can occur for a number of reasons, yet according to criterion C for suspect cases those alternative causes can be
ignored in favour of a COVID-19 diagnosis. The figures defining contact appear arbitrary. For example would face to face contact at half a meter for 7.5 minutes be as effective as standing 2 meters apart for 30 minutes? Local risk assessments could result in a veritable soup mix of dos and don’ts. In addition, most of the definitions hinge on the presence of COVID-19 in some location.

To what degree these loose imprecise definitions will artificially inflate the number of suspect and probable cases of COVID-19 is not known. However, a confirmed case rests solely on a test result which begs the question, "How accurate is the test?"

**Testing for SARS-CoV-2**

No medical test is 100% accurate. Hundreds of thousands of people throughout the world are being subjected to screening tests for the existence of SARS-CoV-2 since its presence is, according to the WHO, definitive evidence of a COVID-19 infection. How accurate is this test?

The current test employs reverse transcriptase polymerase chain reaction (RT-PCR) techniques to identify specific aspects of the virus genetic code. This involves complex, sophisticated laboratory protocols. In 2017 Bustin wrote a lengthy comprehensive critique of investigations dependent on RT-PCR profiling. He described numerous ways in which errors in handling RNA, performing complex technical procedures and interpretation of findings produce unreliable results. (15) No matter how they are performed, such tests have a reputation of being imperfect and sometimes ambiguous. (16)

Determining the accuracy of a test requires the presence of a “gold standard.” This is a test that, “provides authoritative, and presumably indisputable, evidence that a condition does or does not exist.” (16) It is against such a gold standard (or accepted reference standard) that the sensitivity, specificity and positive value of the SARS-CoV-2 test should be assessed. To date, there is no gold standard for the new coronavirus.

Sensitivity is a measure of a test’s ability to identify true positives. A 90% sensitivity means that 10% of the patients tested will have a false negative result. Specificity is an indicator of the test’s ability to identify true negatives-individuals without the condition. A specificity of 95% means that 5% of those tested will be given a false positive result. In general, tests with a high sensitivity have a low specificity meaning that while they will identify actual cases they have a fairly high rate of false positives. The positive predictive value is the ability of a test to correctly identify, among all those who test positive, those who actually have the condition. (16) It requires knowledge of the prevalence of the condition in the population being screened. An interesting fact is that the more people tested who have no, few or a moderate number of symptoms associated with a condition, the more there will be false positives no matter the sensitivity level of the test. For example, in a population of 100 people, 1 person has the disease and the test has a 5% false positive rate and a 5% false negative rate. If all 100 are tested, 6 will test positive (1 true positive and 5 false positives). The positive predictive value of the test is 17% (1 true positive/6 positives X 100). Therefore, although the test appears accurate, it has only an 17% chance of identifying true positives.

It is a truism that, "even very accurate tests can lead to a very high false positive rate if the condition prevalence is low." (17) Therefore, when broad swaths of a population are tested including those with mild to moderate non-specific symptoms, a high percentage of false positives can be anticipated. A study from China suggests that 40-80% of asymptomatic but infected persons might be false positives.
This was based on the investigators making assumptions on disease prevalence and the test’s accuracy. (18) Interestingly, this abstract was recently withdrawn by the publisher, although the full text in Chinese does not appear to have been retracted.

At present, there is no uniform standard of testing for COVID-19. Over the world, different laboratories are testing for different genes in the nucleic acid of the virus - all of them with varying abilities to conduct such tests. The validation and specifications of these tests have not been made available and very little is known about the false positive and false negative rates for these varied tests. (17). The major reason for this is the absence of a gold standard or acceptable reference standard for SARS-CoV-2. As more tests are conducted, more positives will be identified but a decreasing percentage of these will be true positives. On 17th March Dr. Brix (White House COVID-19 Coordinator) said, “Quality testing is paramount. It doesn’t help to put out a test where 50 percent are false positives.”(17)

A recent 25th March investigation from the University of Oxford, COVID-19 Evidence Team, concludes that there is no reliable information on which to assess the false positive and false negative rates for the various RT-PCR tests used to identify SARS-CoV-2. (19)

It is a major concern that screenings are being performed on a global basis with no idea as to the accuracy of the results. Predictions and computer modelling are being performed on tests which have not been subjected to verification. As the adage says, “Garbage in, garbage out.”

The Numbers and the Curve

Each day the numbers of “confirmed” cases are announced similar to league results from a sporting event. Stating that the number of cases in a particular locale has doubled overnight is of minimal value unless accompanied by the number of tests performed to arrive at that number.

As of 14th April, the number of Canadians tested for COVID-19 was 450,717, the number “confirmed “ positive was 26,631 and the number considered negative was 420,072. The positive cases represent 5.7% of those tested who, as a condition of screening, had, at least, symptoms of the infection. It is suggested that by reporting what percentage of those screened have tested positive Canadians might not be so alarmed by media reports announcing dramatic increases in the number of cases, which are a reflection not of the rapid spread of the virus but of the numbers being tested. The paranoia would also be reduced if the numbers of those testing negative were included in the daily briefings.

The demographics of those being screened will alter the number of positives. Limiting testing to only those with severe symptoms will increase the number of positives. While including asymptomatic cases and those with very mild symptoms will decrease the number of positives. The so called “curve” can be altered simply by changing the make-up of those being tested. Unless the same extremely strict clinical criteria for testing are adopted by all screening centres and all laboratories adopt the same investigative techniques, assessing to what degree the curve has flattened will be difficult.

As an aside, the current number of positive cases represents 0.07% of the Canadian population. In the winter of 2017-2018 the percentage of Canadians with the flu was 0.14%, twice the number with COVID-19. Without vaccination the number of flu cases would have been higher.

The figures noted above do not account for the inaccuracies inherent in the test.
Death Rates

As of 14th April, 823 Canadian deaths were considered the result of COVID-19. So, how deadly is the virus? (20)

The Case Fatality Rate (CFR) is the number of deaths divided by the number of known infections. This varies widely depending on the demographics and numbers of those being screened. Assume a population of 100 infected by the virus, with 70 being asymptomatic, 30 being ill and diagnosed as positive, and 1 of those dying of the infection. The CFR is 3.3% (1/30 X 100) but the true death rate is 1.0% (1/100 X100). (21)

During the early stages of a pandemic when mild and/or asymptomatic cases of infection are not identified, the CFR will present a skewed over estimate of the number of deaths. A more accurate assessment is obtained from the Infection Fatality Rate. (21).

The Infection Fatality Rate (IFR) is the number of deaths divided by the true number of infections (confirmed and undiagnosed) in the study population. (21) This is harder to determine as it depends on knowing the prevalence of the infection in the community. Assume a population of 30 infected persons, with 10 detected (1 of whom dies), and 20 undetected. The CFR is 10% (1/10 X 100) but the IFR is 3.3% (1/ 10 detected + 20 undetected X 100).

At the present time in Canada the CFR is 3.0% (823/26,631 X 100). The IFR cannot be calculated since the total number of detected and undetected cases is unknown. However it will be considerably lower than 3.0%. Continuing to screen vulnerable patients will provide an exaggerated assessment of the pathogenicity of the virus.

If this type of information was given out at the exponentially increasing number of media briefings, it might give some reassurance to Canadians especially if compared to the death rates associated with influenza.

Deaths and the Coronavirus

Prepositions have the power to alter the number of COVID-19 related deaths. A patient dying with the corona virus is in a different category than a patient dying from the corona virus. The former represents an opportunistic infection on a compromised host, while the latter is the primary cause of death. (Many elderly men will die with prostate cancer but not from it.)

In the 19th March edition of the Telegraph, Professor Walter Ricciardi, scientific adviser to Italy’s Minister of Health, is quoted as saying, “The way in which we code deaths in our country is very generous in the sense that all the people who die in hospitals with the coronavirus are deemed to be dying of the coronavirus.” Co-morbidities are simply ignored. This might be the reason for the seemingly high number of COVID-19 deaths being recorded in Italy. More recently Professor Ricciardi issued the following clarification, “On re-evaluation by the National Institute of Health, only 12% of death certificates have shown a direct causality from coronavirus, while 88% of patients who have died have at least one-morbidity-many had two or three.” (22)
Writing in the Daily Mail on the 28th March, Peter Hitchens quotes Dr. John Lee a former UK National Health Consultant pathologist as saying, “In the current climate, anyone with a positive test for COVID-19 will certainly be known to clinical staff looking after them. If any of these patients dies, staff will have to record the COVID-19 designation on the death certificate - contrary to usual practice for most infections of this kind. There is a big difference between COVID-19 causing death, and COVID-19 being found in someone who died of other causes.”

In its 31st March update, the Oxford Covid-19 Evidence Service notes that CFRs differ between different countries due to those with severe symptoms being preferentially tested and to how deaths are attributed. (22) The Service convincingly states, “Recording the numbers of those who die with Coronavirus will inflate the CFR as opposed to those that died from Coronavirus, which will deflate the CFR. (22)

There is confusion on what is accepted as a COVID-19 death. In Canada deaths are frequently reported as being, “related to COVID-19” especially those occurring in nursing homes. The phrase carries as much significance as saying the deaths were “related to old age”. The CDC seems to suggest that phrases such as, “assumed to have caused”, or ‘assumed to have contributed to death” are to be recorded as COVID-19 deaths. These phrases do not imply that the primary cause of death was the novel coronavirus. In addition a death can be designated as being caused by COVID-19 even though other viruses and/or bacteria might have contributed to the acute respiratory distress and pneumonia. (23)

It is considered vitally important that this confusion be resolved by formulating a specific definition of what constitutes a death primarily due to the viral infection known as COVID-19.

During this crisis, Canadian authorities should announce weekly, the total number of deaths from all causes, the total number of deaths from pneumonia, the total number of deaths from seasonal influenza and the total number of deaths for which COVID-19 was the primary cause. In addition, the number of hospital beds occupied due to seasonal flu and solely because of COVID-19 should be made public.

Until the pandemic is over, it will not be possible to produce what might be considered a reasonable estimate of the CFR and IFR for any country. Current attempts to do so are based on conjecture and guess work. In his 28th March article Peter Hitchens supports this idea by noting that Professor Neil Ferguson of London’s Imperial College continues to reduce his predictions on the number of UK deaths. Originally, he was forecasting 200,000 to 500,000 deaths, but has now indicated that the deaths are unlikely to exceed 20,000.

**Personal Protective Equipment**

In 1978 the Office of Technology Assessment of the US Congress estimated that, “only 10-20% of all procedures currently used in medical practice have been shown to be efficacious by controlled trial.” (24) A 1990 report by the US National Institute of Health concluded that only 21% of medical treatment was firmly based on research generated scientific evidence. (24) Dr. M. B. Dexter, one of the founders of Evidence Based Medicine, stated in 2000 that, “…much conventional medical wisdom has been found wanting. Many techniques and interventions have been found to be either less effective than previously believed or wholly ineffective.” (25) It is with this background that the use of Personal Protective Equipment (PPE) should be assessed. Prior to doing so, it is necessary to stress that intact skin and the
mucous membranes of the mouth, nose, throat, lungs, and lacrimal fluid are effective initial barriers against invasion by pathogens.

Following the filovirus (Ebola) crisis, the Cochrane Collaboration for evidence-based care conducted a review to determine, “What are the benefits and harms of double gloves, full face protection, head cover, impermeable coveralls, particulate respirators, and rubber boots as PPE when compared with less robust PPE for health care workers caring for patients with filovirus disease.” (26) The reviewers concluded that the body of evidence supporting these PPE protocols was of, “very low quality” and that there was simply insufficient evidence to recommend their use. (26) Following an extensive review of the available literature on the prevention of influenza pandemics an Australian government report has concluded that, “there is limited evidence to support public health recommendations for the use of PPE to prevent transmission of influenza in both public healthcare and community settings.” (27)

A 2019 investigation reported that N95 respirator-type masks were no more effective at preventing COVID type influenza among health care workers as were medical masks. (28) A 2016 review of face shields found that the absolute paucity of appropriate studies warranted scientifically sound research into their effectiveness as part of PPE. (29)

Although the effectiveness of gloves and masks has been questioned by the author, the above reviews are significant because they illustrate that extreme and expensive PPEs maybe no more effective than surgical masks and latex gloves. The reviews emphasize the need to have the efficacy of health care procedures and recommendations supported by robust clinical evidence.

A few days ago, Dr. John Lee emphasized this plea by stating that, “But governments must remember that rushed science is almost always bad science. We have decided on policies of extraordinary magnitude without concrete evidence of excess harm already occurring, and without proper scrutiny of the science used to justify them.” (30)

There is no doubt that the use of PPEs by health care workers creates an intimidating physical barrier between them and their patients. The friendly reassuring smile and warm handshake so beneficial to those in emotional distress is replaced by a human robot encased in latex and plastic. It is feared that this loss of “the milk of human kindness” will have a deleterious effect on the caring professions and the patients they serve.

Social Distancing and Immunity

A recently released report has examined the effect of social distancing in controlling influenza pandemics. (31) Six measures were examined: isolating ill persons, contact tracing, quarantining exposed persons, school closures, work-place measures and avoiding crowds. The authors conclude that evidence supporting such measures was based on observational and simulation studies and is of a low quality. They stress that controlled experiments involving single or combined interventions are needed to clarify the use and effectiveness of social distancing, and when and for how long it should be in effect. In addition, there is a need to perform a cost-benefit assessment of social distancing.

The absence of definitive proof on the efficacy of social distancing combined with very scanty details on the pathogenicity of SARS-CoV-2 suggests that the concept of social distancing could be vulnerable to a legal challenge. This idea is supported by the realization that countries such as Sweden, Japan, South
Korea and Taiwan have seemingly curbed the spread of COVID-19 without adopting the strict social distancing practiced by Canada.

There is no doubt that social distancing will have profound social and economic effects beyond any that might curtail the spread of COVID-19. To what degree these were considered prior to the draconian social distancing measures now being adopted is simply not known. It is a question that, sooner rather than later, must be asked and answered. Government agencies might suggest that without social distancing the numbers of COVID-19 deaths would have been increased. This is a disingenuous idea as it will be extremely difficult to prove or disprove. What is disquieting, however, is the concern that any time there is a hint of a future pandemic similar dictatorial edicts will be enacted.

Consideration should be given to the idea that while social distancing might flatten the curve it could also be extending the length of the curve. It is naïve to assume that social distancing and associated restrictions will stop the spread of the virus. Some people will always become infected, how many and to what degree remains unknown. A safe, effective and economically viable vaccine will not be developed for months if not years, and might not be as equally effective in all age groups.

The aged and medically compromised, especially those in nursing and retirement homes, are particularly vulnerable to COVID-19. Therefore, the first priority should have been the implementation of proven cost effective infection prevention and control precautions in an effort to protect the vulnerable, with the realization that this would not provide 100% protection. In parallel, the public should have been given instructions on simple personal hygiene techniques and told to stay home if they have cold or flu like symptoms. Inevitably some hospitalizations would occur, but herd immunity would be developing which social distancing does not encourage. Together, herd immunity and an effective vaccine will assist in controlling the virus. It is entirely possible that these relatively simple techniques would be as effective as the draconian ones currently in use. At the very least, authorities should be asked why such an approach was rejected.

Conclusion

It is possible that the current responses to COVID-19 are based on the Precautionary Principle (PP). It is defined thus, “When an activity raises threats of harm to human health or environment precautionary measures should be taken even if some cause and effect relationships are not fully established scientifically.” (32) At first blush the PP appears to be a sensible approach. After all, “Better safe than sorry” and “Look before you leap” catchphrases are commonly applied to everyday events. However, the PP has several inherent faults succinctly described by Cass R. Sunstein - the most cited US law professor. He states that the PP offers no guidance because it either endorses taking no action (because to do so would incur more risks) or, especially in worse case possibilities, adopting wide-ranging usually limitless actions. The latter occurs because of the assumption that it is possible to identify safe and effective options while being profoundly ignorant of their probable outcomes. (32)

As a consequence of such actions, the outcomes invariably create risks or unfavourable results that necessitate further precautions. In turn, these produce an increasingly complex maze of recommendations and regulations that have little relationship to the original risk. In fact, Sunstein is of the opinion that, “The problem with the Precautionary Principle is not that it leads in the wrong direction, but that- if taken for all its worth- it leads in no direction at all.” (33) The parallels with how this crisis is playing out are remarkable and ought to be instructive.
It is entirely possible that if COVID-19 had been viewed as a flu like illnesses exhibiting similar epidemiologic and demographic variations as other similar epidemics, its direction would have - by now - been confined to history as causing a somewhat severe flu season. This is not a fanciful concept.

Writing in a current edition of the New England Journal of Medicine, Dr. A Fauci of the CDC said, “This suggests that the overall clinical consequences of COVID-19 may ultimately be more akin to those of severe seasonal influenza (which has a fatality rate of approximately 0.1%) or a pandemic influenza (similar to those in 1957 and 1968) rather than a disease similar to SARS and MERS which had case fatality rates of 9-10% and 36% respectively.” (34)

Instead, a previously unidentified virus has been given the status of a Hollywood demon destined to devastate mankind. This has occurred without verifying its pathogenicity and specifically defining its clinical parameters. The fact that unreliable test results with no established sensitivity and specificity levels have been used to formulate government and health care responses should be concerning. Tampering with the cause of death in presumptive COVID-19 infections borders on professional incompetence. Not surprisingly, the doomsday predictions relating to Covid-19 are based on little, if any, verifiable data. However, in countries where a broad spectrum of the population has been tested, death rates are remarkably low which suggests that the dire forecasts will not come to pass. There is a desperate need for a time out to reflect on what is truly known about this virus and to act accordingly.

A method of accomplishing this would be to start asking informed questions of all authorities whose diktats have radically altered our lives and our economy. A public discourse of this nature should be encouraged. It is hoped that the issues and concerns identified in this article might act as catalysts for such much needed debates.

When the history of COVID-19 is written there remains a chance that it will not describe Nietzsche’s perspective on insanity, “In individuals, insanity is rare: but in groups, parties, nations and epochs, it is the rule.”

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