Expert Letter for Case:

Her Majesty the Queen in Right of Ontario v. Adamson Barbecue Limited and William Adamson Skelly

Court File No. CV-20-00652216-0000

My name is Gilbert G. Berdine, M.D. I received my undergraduate education from the Massachusetts Institute of Technology. I graduated in 1974 with a B.S. degree in chemistry and a 2nd B.S. degree in Life Sciences. I received my medical education from the Harvard University School of Medicine as part of the Health Sciences and Technology joint program with M.I.T. I was awarded the M.D. degree in 1978. I received my post graduate medical education from the Peter Bent Brigham Hospital in Boston, MA (now called Brigham and Women's Hospital) in the specialty of Internal Medicine and subspecialty of Pulmonary Diseases from July 1978 – June 1983. I am board certified in Internal Medicine and Pulmonary Diseases. My Pulmonary Diseases board certification pre-dates the existence of the Critical Care and Sleep Medicine Boards, so I have grandfather certification in those specialties as well.

I was a member of the faculty at University of Texas Health Sciences Center at San Antonio from 1984-1989. I was in private practice in the state of Texas from 1989-2009. I have been a member of the faculty at Texas Tech University Health Science Center in Lubbock, TX since 2009.

Since the outbreak of COVID-19 in March of 2020, I have treated many patients with COVID-19. Although I have not cared for COVID-19 patients in the Intensive Care Unit (ICU), I have cared for and am currently managing patients with COVID-19 in the following situations: asymptomatic patients with positive PCR tests managed at home; presumed COVID-19 cases with mild symptoms managed at home; presumed COVID-19 cases who required hospital care but did not require ICU care; presumed COVID-19 cases recovering from Acute Lung Injury (ALI) or acute respiratory distress syndrome (ARDS) transferred from ICU care to hospital floor care; and presumed COVID-19 survivors discharged from the hospital and receiving long term care at home. I am familiar with the clinical presentations of COVID-19. I am familiar with the difficulties of classifying cases and deaths as to whether they are associated with COVID-19 or were caused by COVID-19. I am familiar with the limitations of polymerase chain reaction (PCR) testing for the SARS-CoV-2 virus. I have reviewed manuscripts for peer-review journals on COVID-19. I have written articles related to the costs vs. benefits of lockdowns in response to COVID-19[1, 2], the dynamics of COVID-19 transmission[3-5], difficulties in distinguishing deaths FROM COVID-19 vs. deaths WITH COVID-19[6], ethical issues related to COVID-19[7], and the safety vs. efficacy of COVID-19 vaccines.[8-11] I am a co-investigator on an active research project studying the clinical features of hospitalized patients with positive PCR tests for COVID-19 including analysis of whether deaths attributed to COVID-19 are due to COVID-19 or other causes.

Diagnosis of COVID-19

There is no specific symptom, sign, or laboratory test for COVID-19. There is no way to be certain that a patient has a diagnosis of COVID-19. A review of studies reporting on clinical manifestations in COVID-19[12] concluded that the most common symptoms were fever (58.66%), cough (54.52%), dyspnea (30.82%), malaise (29.75%), fatigue (28.16%), and sputum production (25.33%). However, the prevalence of symptoms will depend on the severity of disease which will, in turn, depend on the age of

the subjects. Prior to the availability of PCR tests for COVD-19, there was no way to distinguish COVID-19 from other respiratory viruses.

Problems with PCR Testing

Polymerase chain reaction (PCR) testing for COVID-19 became available around April of 2020. Since there were no other gold standards for true positives, the PCR test became the de facto gold standard even though there was no way to determine the sensitivity and specificity of the test. Published figures for sensitivity and specificity of PCR testing for COVID-19 are guesses based on presumed true positives. A crucial part of PCR testing is chain amplification to increase the amount of material for detection. The cycle count is the number of chain amplification cycles. A cycle count of 25 is typical. Prior to January 2021, the cycle count for COVID-19 PCR tests was 35-40. This excessive level of amplification increases the likelihood that virus fragments or cellular debris are mistaken for intact virus particles. Once PCR testing was available, anyone with a positive PCR result was considered a COVID-19 case. Anyone who died following a positive PCR test was considered to be a COVID death irrespective of what actually caused the death. Medicare increased the weighting factor of Diagnosis Related Group (DRG) payments by 20% for a diagnosis of COVID.[13] U.S. Hospitals routinely perform PCR testing on every patient and include a diagnosis of COVID-19 for every patient with a positive result irrespective of the clinical situation.

PATHOLOGY OF EXPOSURE, INFECTION, DISEASE, AND DEATH

Exposure

Exposure to the SARS-CoV-2 virus responsible for COVID-19 is via the respiratory tract. Infected subjects exhale droplets contaminated with virus. These contaminated droplets are inhaled by other people. Subsequent events are determined by the viral load of the exposure and the subject's immune response. It is presumed that some people (possibly half) have pre-existing immunity from exposure to other coronaviruses in the past.[14] The initial line of defense is T-cell and IgA antibody in the respiratory tract. If the virus is defeated at this early stage, symptoms are unlikely, and there may be no production of IgG or IgM antibodies. These subjects may have no symptoms or any laboratory test confirming exposure. Tests for this type of immunity require samples of respiratory secretions such as nasal swabs. Blood tests are not helpful. Detection of T cells specific for this early stage defense are not commercially available and are usually only available from specialized research facilities. The presence of secretory IgA involved in this early defense will only be present in mucosa that was actively defending against virus; much of the respiratory mucosa may test negative. This first line of defense comes from previous exposure and natural immunity. Parenteral vaccines (jabs) lead to IgM and IgG immunity which do not provide this early protection.

Infection

If the virus gets past this initial line of defense, it infects respiratory epithelial cells, takes over the production of proteins in the cell, and uses the cellular machinery to replicate new virus particles. New virus can spread laterally to adjacent epithelium. This mechanism can lead to spread of virus from the nose, mouth, and throat to the lower respiratory tract leading to pneumonia. These patients may have a systemic response leading to fever, fatigue, malaise, leukocytosis, and the formation of IgG and/or IgM antibody. Many patients will defeat the virus at this stage without serious illness.

Serious Illness

New virus particles can also spread to deeper tissues and enter the lymphatics or blood stream. Lymphatic spread can lead to enlarged and inflamed lymph nodes. Spread via the blood stream can reach any other organ leading to multi-organ failure or sepsis. Some will develop an acute lung injury (ALI) possibly leading to acute respiratory distress syndrome (ARDS). Dissemination to multiple organs, sepsis, ALI, or ARDS would all be classified as serious illness and would probably require ICU care. ARDS is a very serious problem with mortality of 30%-50% irrespective of age. The immune response can generate non-specific symptoms. At this time, it is unclear whether the ARDS is caused by viral infection or an overreaction by the immune system: cytokine storm. The most important determinant of clinical outcome seems to be age. Young people are more likely to have either no symptoms or mild symptoms. The elderly are more likely to have ALI or ARDS. In my experience, there is no significant difference between the ARDS from COVID-19 and the ARDS from any other cause including influenza, sepsis, aspiration, or drug reactions. It has been reported that some patients with mild disease have symptoms lasting months, but this is expected for any disease that elicits a strong immune response.

Lethality of COVID-19

The government mandated lockdowns of economies in response to COVID-19 were rationalized by predictions of COVID-19 deaths made by Neil Ferguson *et al*.[15] These predictions turned out to be very wrong. A critique of the Ferguson model[16] includes that predictions of deaths in the absence of lockdowns were inflated by a factor of 10, and predictions of deaths following lockdowns were horribly low. These models made assumptions that violated principles of epidemiology known for many years.

MORTALITY CURVES AND EFFECTS OF INTERVENTIONS

Predicted Mortality Curve in Uniform Population

We will review basic principles of the predicted effects of lockdowns and confirm these basic principles with empiric data of deaths due to COVID-19.

Figure 1: Mortality Curve for a pandemic in a uniform population.

Figure 1 illustrates the dynamics of fatalities following an epidemic in a uniform population. Uniform does not mean that everyone has the same result. Uniform means that each person has the same probability of death from exposure to the virus. We cannot distinguish prognosis of outcomes between people *a priori*. Deaths are per unit of time and normalized to the peak of the mortality curve. Time is in arbitrary units. The peak mortality occurs at Time = 3. There are three regions of this curve. The Growth phase is roughly from Time = 0 to Time = 2. The Transition phase is roughly from Time = 2 to Time = 4. The Decline phase is roughly from Time = 4. The curve satisfies the equation:

$$D(t) = C(t) * F_D \text{ (Eq. 1)}$$

where D is deaths, t is time, C is active cases, and F_D is the fraction of cases that will die each time increment. We will simplify the analysis by assuming that F_D is a constant. We will, therefore, approximate D(t) as a scaled version of C(t). The change in active cases is determined by the balance of creation of new cases via transmission of virus from infected person to susceptible person, and the resolution of old cases via recovery or death. Therefore, the case curve (C(t)) is determined by the differential equation:

$$dC/dt = k_1 * P * (1-P) - k_2 * C \text{ (Eq. 2)}$$

where P is the prevalence of viral infection, k_1 is a constant combining the number of interactions between people and the probability that an interaction between an infected person and a susceptible person will result in a new infection, and k_2 is a constant representing that active cases will be resolved either by cure or death. Although the equations seem complicated, we can make some simple statements about the nature of the three phases. The math is not necessary to understand the general analysis, but the math is necessary to justify that the analysis is valid.

During the Growth phase, P is small, so (1-P) is approximately 1, so the Growth phase approximates a rising exponential with doubling time dependent on k_2. During the Transition phase, growth is

substantially less than exponential due to a decline in the fraction of susceptible persons (1-P). Growth in deaths continue to increase for some time due to increases in the prevalence of infected people, but the curve passes through an inflection point where the effect of increases in P are less important than the decline in (1-P). At the peak of the curve, new cases due to virus transmission are offset by the rising resolution of old cases, so the net change in active cases is zero. During the Decline phase, there are no new cases due to exhaustion of susceptible hosts (herd immunity), so the curve is a decaying exponential with time constant determined by k_2. The total number of deaths is the area under the curve.

Predicted Effects of Interventions on a Uniform Population

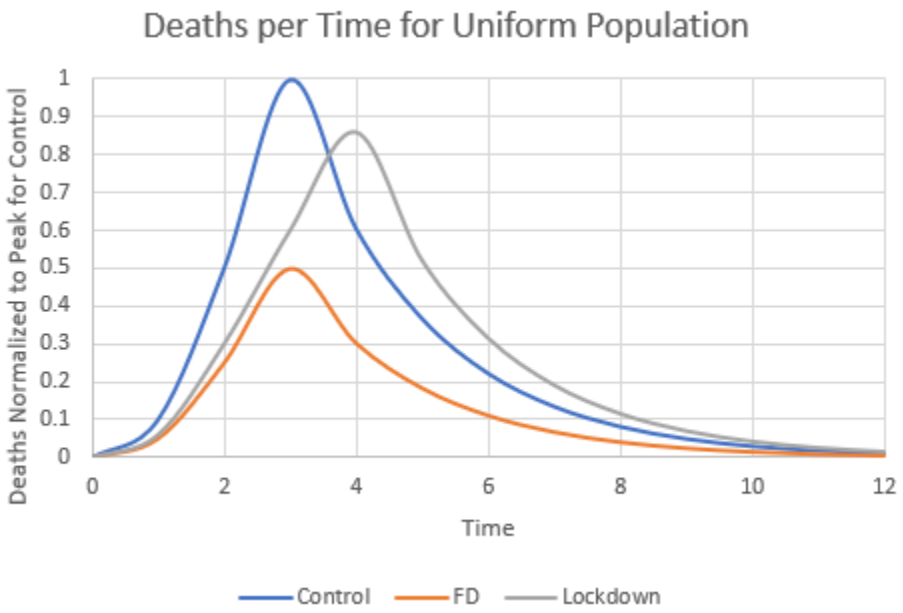

Figure 2: Mortality curves for three intervention strategies in a uniform population.

We can also infer the effects of interventions using Equations 1 and 2. Figure 2 illustrates the effects of different intervention strategies on a uniform population. The blue curve is the control group without any intervention. The total number of deaths for no intervention is 3.12.

The orange curve is an intervention that decreases F_D to ½ of the control value. An example of this type of intervention would be treating everyone with a hypothetical agent that improved immune response. This type of intervention does not change the time required to reach the peak of the curve or the dynamics of the Decline Phase. The number of deaths at each time is adjusted upward for an increase in F_D or downward for a decrease in F_D. The total number of deaths for the orange curve is 1.56 or half the total for the control group.

The grey curve illustrates the effect of lockdowns. The intent of lockdowns is to decrease the number of interactions between people, so k_1 will decrease, the duration of the Growth phase will be longer, and the time to achieve the peak in deaths will be longer. It must be noted that the total number of deaths is unchanged, however, since everyone is eventually exposed to the virus and the total number of deaths remains the total population times F_D. This was a major conceptual flaw in the Ferguson model as their

interventions presumed that lockdowns would not only prolong the Growth phase (they would), but that total deaths would decrease (they would not).

Effect of Age on Mortality – The Population is Not Uniform

According to the CDC[17] as of this writing, there were 554,064 deaths out of 30,532,965 cases for a case fatality rate of 1.81%. This number is undoubtedly inflated since many people with no symptoms or mild symptoms are not tested, and the number of deaths attributed to COVID-19 include deaths caused by other problems. As part of my active project on COVID-19, I analyzed the medical records for 45 deaths attributed to COVID-19 based on a positive PCR test and concluded that 22/45 deaths were due to other causes. Furthermore, the fatality rate is very dependent on age.

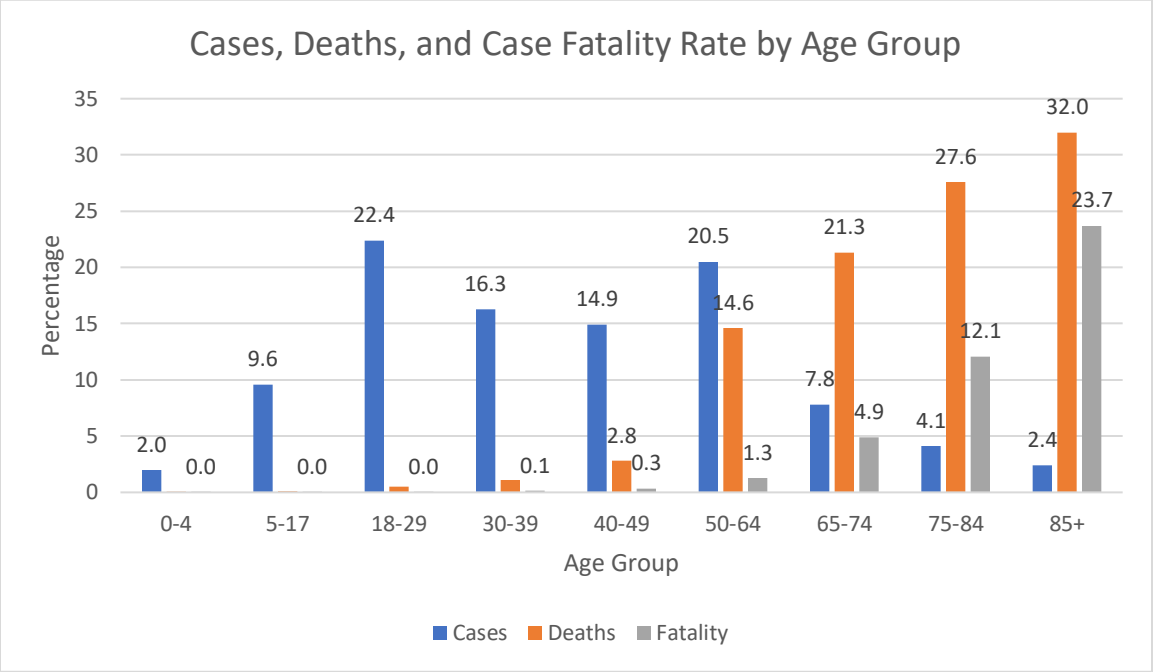

Figure 3: Percentage of Cases, percentage of deaths, and case fatality rates by Age Group. Data are from CDC.[17] Blue bars are cases as a percentage of 22,451,800 cases. Orange bars are COVID-19 deaths as a percentage of 398,179 COVID-19 deaths. Grey bars are the case fatality rates (100*deaths/cases) for each age group.

The case fatality rate is over 1000 times greater for people 85 and older compared with people under the age of 18. There is no reason to believe that the situation is much different in Canada. Any policy that treats young people the same as old people will inevitably be too restrictive on young people and too lenient on the elderly. The overall case fatality rates in Canada and the U.S. are comparable. According to Worldometer[18] as of this writing, there have been 1,014,374 cases of COVID-19 in Canada and 23,118 deaths attributable to COVID-19 in Canada for a Canadian case fatality rate of 2.28%. As was mentioned earlier, this figure is likely inflated as people without symptoms or people with mild symptoms who do not want to seek health care are unlikely to be tested.

Characteristics of a Split Population

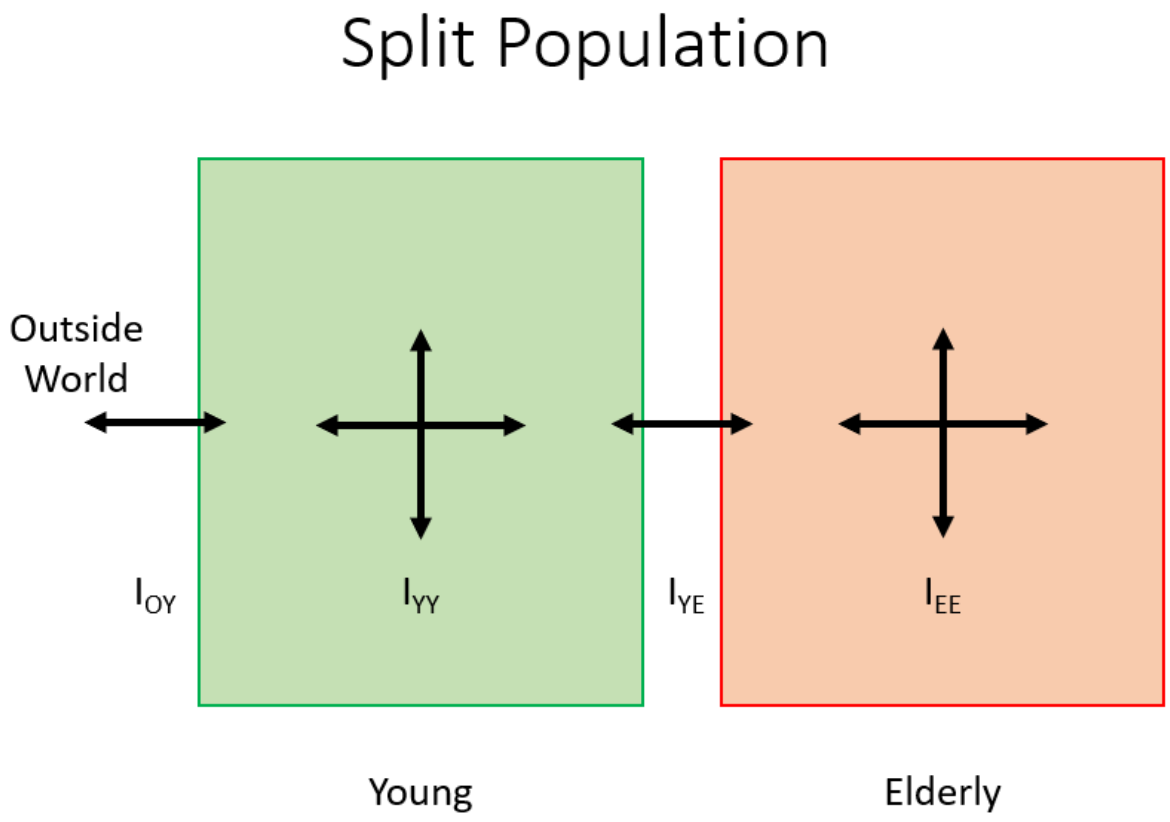

Figure 4: Schematic of Split Population

The marked effect of age on case fatality as well as the realities of how the elderly function in society require us to consider the effects of interventions on a split population. Figure 4 illustrates the simplest breakdown of our population into two separate but connected groups. The young (Green) are a large population with a very low case fatality rate. It is not possible to entirely lock down the young as even the government recognizes some activities of young people as "essential" and exempt from government lockdown. The young encounter outsiders (I_{OY}). Some of these encounters introduce virus into the young population. The young do most of the work. During such work, they encounter other young people (I_{YY}). Enough of these encounters are essential which cannot be locked down, so it is inevitable that everyone in the young (Green) group will be exposed to the virus. Eventually there will be deaths in this group defined by:

$$D_Y = N_Y * F_Y \text{ (Eq. 3)}$$

where D_Y is the number of deaths in the young population, N_Y is the total young population, and F_Y is the true case fatality rate for young people. The only means of decreasing D_Y are interventions that decrease F_Y. Lockdowns do not affect F_Y, so lockdowns cannot possibly reduce D_Y. One example of an intervention that would decrease F_Y would be an incentive to decrease obesity in the young population. D_Y will be a very small fraction of total deaths, so any attempt to decrease D_Y would have little effect on total deaths.

The elderly (Red) are low in total number but have a very high case fatality rate. The elderly are generally unemployed and receive care from young people. The elderly are not generally connected to the outside, so exposure to virus is not inevitable. There are two sources for elderly exposure: interactions between infected young people with susceptible elderly people (I_{YE}) and interactions between infected elderly people and susceptible elderly people (I_{EE}). The math for this system is much more complicated than for the uniform population. There are several domains with much different curve shapes depending on the choice of parameters. We will make generalizations to simplify the analysis, use this simplified analysis to make predictions, and compare the predictions with empiric data.

Effect of Lockdowns

Figure 4 identifies four types of human interactions. I_{OY} are interactions between people outside the jurisdiction and young people. An example would be a customs official screening people entering Canada from a foreign country. I_{OY} interactions are generally considered "essential" and would not be affected by lockdowns. I_{YE} interactions are interactions between young working people and the elderly population. An example would be a nurse in a long-term care facility. As was the case for I_{OY} interactions, I_{YE} interactions are generally considered "essential" and would not be affected by lockdowns. I_{EE} interactions are interactions between elderly people. The elderly are generally unemployed, so lockdowns of businesses would have no effect on I_{EE}. The elderly are generally neither students nor instructors, so lockdowns of schools would also have no effect on I_{EE}. The final type of interaction is I_{YY}. I_{YY} interactions are between young people. These interactions include most (if not all) interactions in workplaces. Although some elderly can be customers, we are mostly concerned about elderly people who are either home bound or receiving care in long term care facilities. Lockdowns would be expected to decrease I_{YY}. We will analyze the effect of lockdowns based on a decrease in I_{YY} without any change in the other interactions.

Maximum Number of Elderly Deaths

There is a maximum number of deaths in the elderly which will be achieved if all of the elderly are exposed to virus. This maximum number of elderly deaths is given by:

$$D_{ME} = E_E * \alpha \quad \text{(Eq. 4)}$$

Where D_{ME} is the maximum number of elderly deaths, E_E is the number of elderly people exposed to virus, and α is the probability that a single exposed elderly patient will die from COVID-19. It should be noted that the maximum value for E_E is the total number of elderly patients. α represents the deaths resulting from a single elderly exposure to COVID-19. α is related to I_{EE} and unrelated to the other interactions. α can be sufficiently high that a single exposure of COVID-19 leads to the maximum number of deaths D_{ME}. If this is true, then further analysis is moot, as any changes in I_{OY}, I_{YY}, or I_{YE} would have no effect on total elderly deaths. Since the case of excessive α is trivial, we will assume in all further analysis that α is sufficiently low to prevent a single elderly case from leading to the maximal number of elderly deaths (D_{ME}).

Compartmentalization

It is clear that lower values of α are better. α can be lowered either by improved health care methods that improve elderly outcome, or by decreases in I_{EE}, or by something that I call compartmentalization. Unlike a decrease in I_{EE} which is a decrease in how many interactions an elderly person has with other

elderly people, compartmentalization restricts how many different elderly people that an elderly person can possibly contact. An example of an increase in compartmentalization would be breaking up a large community of elderly people into many smaller groups with each group isolated from the other groups. Ships are designed with compartments such that water leaks cannot spread outside the compartment. This design allows the ship to survive limited hull ruptures until the damage can be repaired. Compartmentalization of the elderly limits how many elderly people can be infected by a single elderly case while permitting any number of elderly human interactions.

Mortality Curves for a Split Population

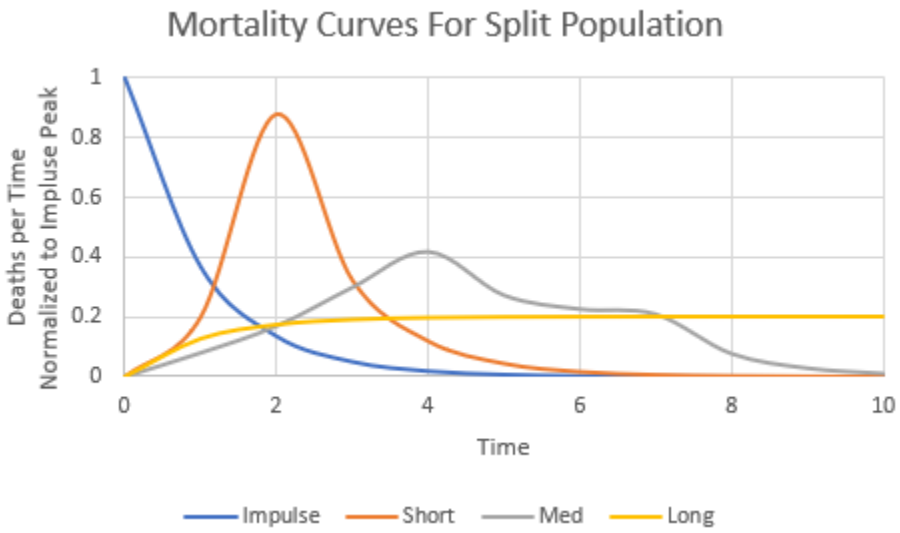

Figure 5: Mortality Curves for a Split Population. The blue curve represents mortality following an impulse of cases. The orange curve represents mortality following a short duration pulse of cases. The grey curve represents mortality following a medium duration pulse of cases. The gold curve represents mortality following a long duration pulse of cases. The total number of cases for the impulse and all the pulses are equal.

An impulse is an engineering or mathematical abstraction. It is a weird rectangle with zero width and infinite height, but the area of the rectangle is finite and is the amplitude of the impulse. An impulse with amplitude 5 has an area contained by the impulse equal to 5. For this discussion, the amplitudes of these impulses are the number of patients contained by the impulses. Imagine that at time t=0, an ambulance delivers 10 patients to a nursing home with COVID-19. The case input signal to the elderly population of our split population model is an impulse with amplitude of 10. We will predict the mortality curve for such a situation.

Equations 1 and 2 apply to this situation. Equation 2 simplifies to the differential equation:

$$dC/dt = - k_2 * C \text{ (Eq. 5)}.$$

The solution of this equation is that C(t) is a decaying exponential with time constant k_2. The boundary condition for the solution is the number of deaths at t=0. This number is determined by the probability that a case will die on any given day (α). The blue curve in Figure 5 illustrates this situation.

The step response of a system is the response to a unit step. For all t<0, the amplitude is 0, and for all t>0, the amplitude is 1. The amplitude at t=0 is undefined. The unit step is the integral of the unit impulse; the unit impulse is the derivative of the unit step. We can determine the step response of a system by integrating the impulse response; we can determine the impulse response by differentiating the step response. The step response for the mortality curve of our elderly population is another type of exponential: this one starts at 0 and the difference between the start point and end point decays exponentially. The gold curve of Figure 5 illustrates the step response.

We can determine the system response to any input from the impulse response by a technique called convolution. Without going into a lot of math, suffice it to say that the response to a rectangular pulse transitions from the impulse response for a very short pulse to the step response for a very long pulse. For our split population system, a high amplitude short duration pulse of cases entering the elderly population is produced when I_{YY} is very high. A low amplitude long duration pulse of cases entering the elderly population is produced when I_{YY} is very low. The shape of the mortality curve for our split population system depends on whether the young population saturates (achieves herd immunity) prior to reaching peak mortality, after reaching peak mortality, or not at all. The orange curve in Figure 5 is the response to a pulse where herd immunity is achieved in the young population while mortality is still rising (before the peak). As I_{YY} decreases, the peak is delayed, the peak is lower, but the area under the curve is unchanged. The mortality curve declines to zero. This shape is similar to what we analyzed for the uniform population situation. The grey curve in Figure 5 is the response to a pulse where herd immunity is achieved in the young population while mortality is declining. As I_{YY} is further reduced: the peak is further delayed; the peak is lower amplitude; the initial decline prior to herd immunity in the young population is not toward zero, but rather toward a plateau number of deaths each day; this initial plateau continues until the young population achieves herd immunity, which allows the active case number in the young population to decline to zero, which turns the pulse driving I_{YE} off, which allows the mortality curve to have a second decline to zero. The area under the curve remains unchanged since herd immunity was achieved in the young population. The gold curve in Figure 5 is the step response which is observed when herd immunity is not achieved in the young population. Since herd immunity is not achieved in the young population, the number of active cases in the young population does not decline to zero (turning the pulse off), so deaths cannot decline to zero until the maximum number of deaths occur. Further decreases in I_{YY} will have no benefit as deaths will continue to accumulate until the maximum number is achieved.

Cases vs. Deaths in a Spit Population

For a uniform population, cases are a useful proxy for deaths as the total deaths will track the total cases as long as there is no fundamental change in the lethality of the virus. The situation is much different, however, for the split population shown in Figure 4 with mortality curves shown in Figure 5. The best way to minimize total deaths is to have the virus spread rapidly through the young population (high I_{YY}) which minimizes the time to herd immunity and the time to peak deaths. A decrease in cases through lockdowns which decrease I_{YY} can actually increase mortality. Cases in the young population do not carry the same weight for mortality as cases in the elderly population. Case count cannot be used as a predictor of future deaths since very few young people die. Rapid achievement of herd immunity may result in a very large number of cases, but more cases can actually translate into fewer deaths as there is a shorter duration of time for cases to move into the elderly population through I_{YE} where the cases become deadly. For a split population, case count is a very poor metric for pandemic management;

hospitalizations or patients on ventilators are much better predictors. In some cases, there is no suitable substitute metric for deaths.

Conclusions about Split Population

The above discussion includes a lot of math. The concepts explained have been known for a long time with respect to other applications. These concepts have recently been applied in the field of epidemiology to explain apparent paradoxes where bad outcomes come from good intentions.[19] The broad points of the split population discussion are:

- Attempts at control of transmission can shift the burden of disease to older people.
- If older people have higher mortality (which they often do) these attempts at control of transmission will lead to more deaths due to a greater percentage of older people becoming infected.
- For COVID-19, the best result is rapid spread among young people to achieve herd immunity with a minimum of mortality, shortening the duration of the epidemic, minimizing the exposure of elderly people, and achieving fewer total deaths than would occur following lockdowns.

Confirmation of Predictions by Empiric Data

We cannot use the above theoretical discussion to quantitatively predict what will happen in New York, Canada, or anywhere else. People are not electrons. People are not fungible. People make unpredictable individual choices. Rather we study the theoretical framework to make general qualitative statements about the general appearance of a mortality curve if certain choices are made. Rather than working forward from a theoretical prediction to empirical data, we examine empirical data to conclude what choices could have led to the observed result.

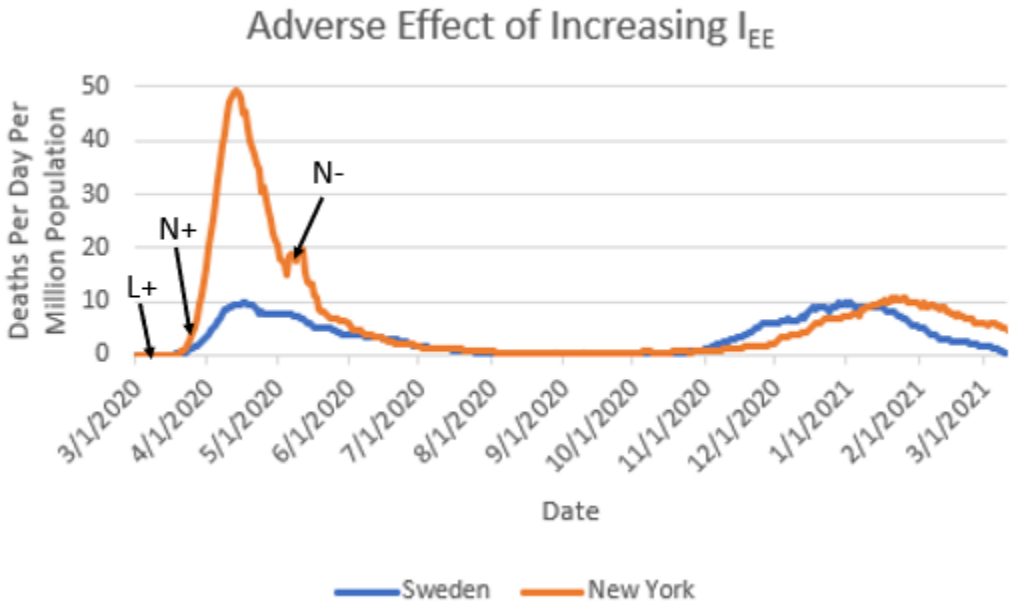

Figure 6: Mortality Curves for Sweden vs. New York State. Data are from Worldometer[18]. Data starts March 1, 2020 and ends March 10, 2021. Y-values are deaths per day per million population. The blue

curve represents Sweden. The orange curve represents New York State. L+ marks the New York state of emergency declared on March 7, 2020. N+ marks the start of the New York State nursing home order on March 25, 2020. N- marks the end of the New York State nursing home order on May 10, 2020.

Figure 6 is a striking example of increasing the adverse effect of interactions between elderly people (I_{EE}). These adverse effects can be achieved by either increasing the number of interactions between elderly people, increasing the number of deaths among elderly people from each case introduced into the elderly population, or artificially bypassing the young population and injecting infected people directly into the elderly population. Sweden serves as a control where little or no government interventions were employed. Prior to January 2021, masking and social distancing were voluntary, and the only restrictions were on gatherings of more than 50 people. In January 2021, masking and social distancing became mandatory in restaurants, bars, and gyms, but no businesses were closed. Schools were never closed. New York declared a state of emergency on March 7, 2020 and has been locked down to some degree since that date. At the time that the lockdowns were initiated, the mortality rate was less than 1 per million population. On March 25, 2020, Governor Cuomo issued the infamous order preventing nursing homes from declining patients who were PCR positive for COVID-19. This order had an immediate effect of increasing transmission of virus between elderly people in nursing homes and was effectively a large increase in I_{EE}. The mortality rate was 4.11 per million population on that date, but it would increase to almost 50 by April 13. The nursing home order was rescinded on May 10, but the damage was already done. The peak mortality occurred on April 16, 2020 in Sweden and April 13, 2020 in New York. If there was any effect of the New York lockdown through a decrease in I_{YY}, this effect was rendered moot by the nursing home order and its adverse effect on I_{EE}. The 2nd wave in the winter of 2020 has a much different picture. New York was locked down decreasing I_{YY}, but there was no nursing home order increasing I_{EE}. Swedish mortality peaked on December 31, 2020. New York mortality peaked almost a month later on January 25, 2021. This delay in the peak is consistent with a lockdown but without a nursing home order. The total mortality from March 1, 2020 through August 31, 2020 was a disaster in New York at 1,695 deaths per million population compared to 570 for Sweden. Total mortality for the 2nd wave from September 1, 2020 through February 28, 2021 was 745 deaths per million population for New York and 706 for Sweden. Sweden (no lockdowns) had superior results to New York (harsh lockdowns). The impact of the nursing home order in New York rendered any effect of the lockdown moot.

Figure 6 confirms a general point made in the theoretical discussion: any impact of a lockdown of young people (decrease in I_{YY}) should show up in the mortality curve as a delay in the peak mortality that results from an outbreak. Although a lockdown of young people (decrease in I_{YY}) should decrease the peak of the mortality curve, the total mortality (area under the curve) will either be unchanged or increased depending on how long the lockdown is maintained and whether alert fatigue develops among the population. Furthermore, one cannot compare either the peak in mortality or the total mortality between two jurisdictions with different policies and attribute the difference in mortality to the lockdown without knowing details of all the factors that determine how many deaths result from each elderly case. If the dynamics of rise to peak and decay from peak are the same for two populations, differences between the mortality curves (amplitude) cannot be attributed to the presence of lockdown or intensity of lockdown. Any effects of lockdown of young people (I_{YY}) should be apparent as a delay in the peak mortality.

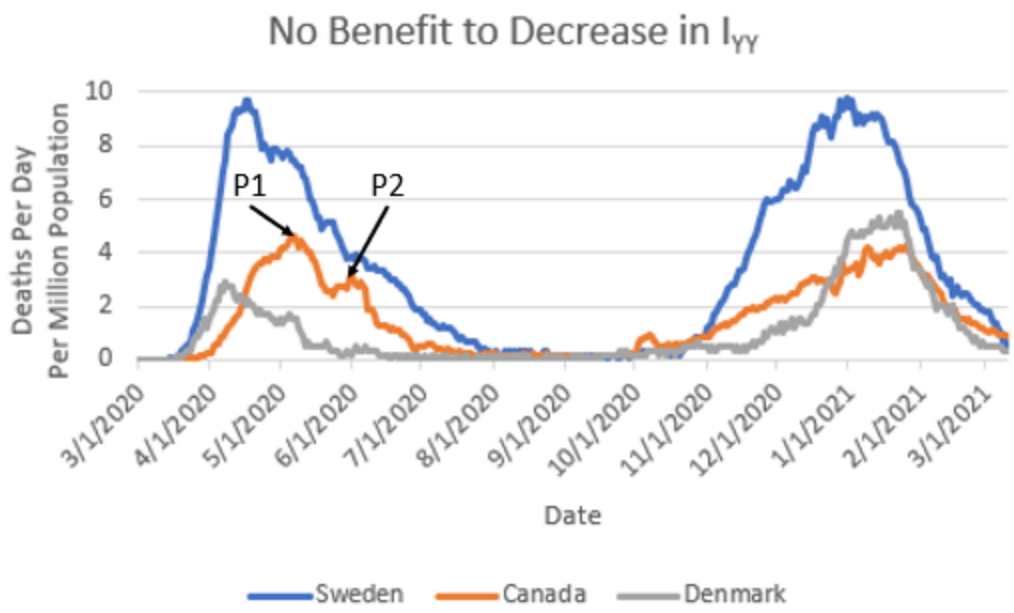

Figure 7: Mortality for Sweden (blue), Canada (orange) and Denmark (grey). Data are from Worldometer.[18] Dates begin March 1, 2020 and end March 10, 2021. Y-values are deaths per day per million population. P1 marks the 1st peak in Canadian mortality on May 6, 2020. P2 marks a later peak in Canadian mortality on May 31, 2020.

At first glance, Figure 7 seems to suggest that lockdowns improve mortality. Sweden (blue) which had no lockdowns had a higher mortality than either Canada (orange) or Denmark (grey). However, proper analysis using timing of peaks leads to the opposite conclusion. The first peak occurred on April 16, 2020 in Sweden and on April 7, 2020 in Denmark. There is no evidence that the lockdowns in Denmark had any effect on mortality by a reduction in I_{YY}. In fact, based on an earlier peak in deaths in Denmark, we cannot even conclude that the lockdowns in Denmark actually reduced I_{YY}. The improved mortality in Denmark must be due to a younger population, a healthier population, better treatment of cases, better compartmentalization of the elderly, better infection control among elderly people in long term care facilities (reduced I_{EE}), or better control of transmission of virus from young people to elderly people (reduced I_{YE}). For similar reasons, we cannot look at the lower mortality in Canada, and attribute this lower mortality to bans on indoor dining in Ontario and other provinces during March of 2020. Nobody can look at the data and make a credible claim that mortality in Canada would have been higher in the absence of lockdown.[20-24]

Pattern of Canadian Mortality and Conclusions Drawn

The gold curve in Figure 7 is COVID-19 mortality for Canada. The peak in mortality was May 6, 2020 in Canada compared with April 16, 2020 in Sweden. We can conclude that the Canadian lockdown decreased I_{YY} and delayed the development of herd immunity in Canada. There is a 2nd peak in Canadian mortality on May 31, 2020. Some might mistakenly call this a 2nd wave. This was not a 2nd wave but rather a demonstration of a medium duration pulse width illustrated in Figure 5. The shoulder seen on May 31, 2020 following the peak of the 1st wave seen on May 6, 2020 is due to prolonged percolation of

cases from young people to elderly people (I_{YE}). This slow percolation was caused by the delay in herd immunity which was a predictable result of lockdowns that decreased I_{YY}.

Empiric Confirmation of Plateaus of Death

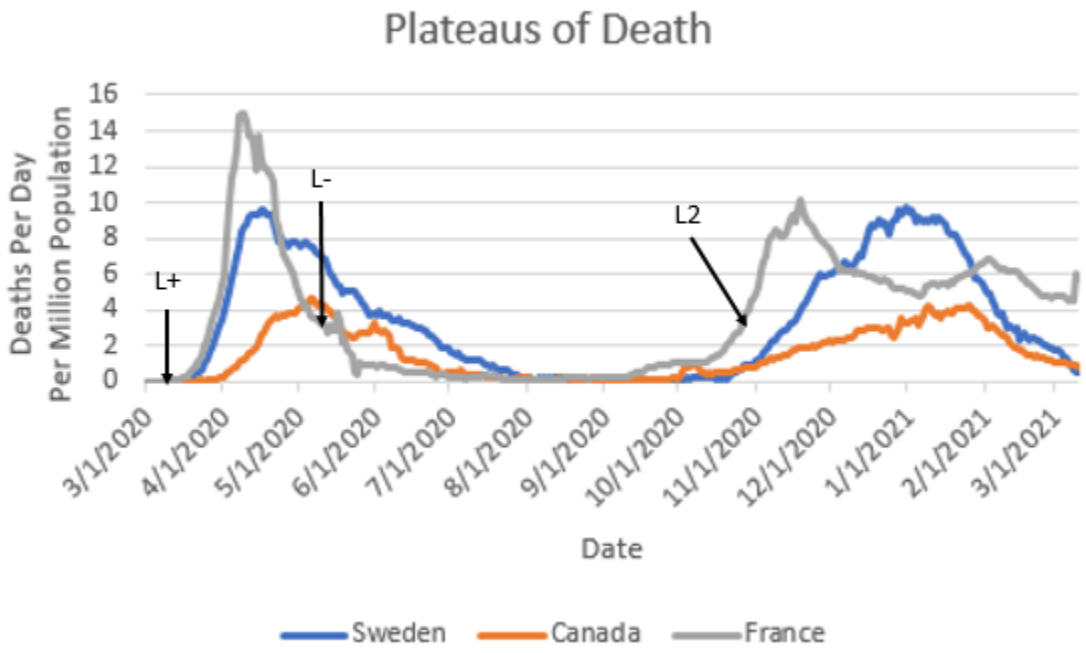

Figure 8: Example of France mortality to illustrate a plateau of death following lockdown. Data are from Worldometer.[18] Dates begin on March 1, 2020 and end on March 10, 2021. Y-values are deaths per day per million population. Blue curve is mortality for Sweden. Orange curve is mortality for Canada. Grey curve is mortality for France. L+ marks the date of school closures in France as the start of the 1st French lockdown on March 12, 2020. L- marks the end of the full 1st lockdown on May 11, 2020. Lockdown measures were relaxed in phases. L2 marks the start of the 2nd French lockdown on October 28, 2020.

The first COVID-19 case in France was reported on January 24, 2020. France reported the first COVID death outside of Asia on February 14, 2020. France closed its schools on March 12, banned public gatherings of more than 100 people on March 16, 2020, and closed all “non-essential” businesses on March 17, 2020. The lockdown was extended twice to May 11, 2020. Progressive relaxations of lockdown were permitted. On July 10, 2020 the State of Health Emergency was ended in France officially ending the 1st lockdown, though some restrictions on social distancing remained. Based on rising cases beginning in August 2020, Paris imposed mandatory masks on August 28 in public places, Paris locked down pubs, restaurants, and cafes, and France imposed a second nationwide lockdown on October 28.

The 1st lockdown in France did not delay the peak or seem to “flatten” the curve. It is possible that the French policy effectively segregated a large portion of young people who would be susceptible to future spread of the virus once the lockdowns were relaxed. This is known as the “dry tinder” effect. Similar effects were seen in Southern U.S. states including Texas. The rise in cases and deaths during August of 2020 were not a 2nd wave; rather this represented deferral of the completion of the 1st wave. This deferral was seen in many countries and U.S. states. Space does not permit an illustration of all the examples.

The mortality in France following the 2nd lockdown on October 28, 2020 was a vivid illustration of the plateau of death illustrated in Figure 5. Mortality in France still has not entered a decline phase. Mortality in France was 468 deaths per million population from March 1, 2020 through August 31, 2020 compared to 570 in Sweden. However, mortality in France was 843 from September 1, 2020 through February 28, 2021 compared to 706 in Sweden. Furthermore, daily mortality is less than 1 per million population in Sweden yet remains over 4 per million population in France. The 2nd lockdown in France created the circumstances responsible for this plateau of death.

Features of 2nd Wave During Winter of 2020

Figure 8 shows striking differences in the rate of the growth phase for Sweden, Canada, and France between the 1st Wave peaking in April 2020 for Sweden and France and May 2020 in Canada compared with the 2nd wave peaking November 19, 2020 in France, December 31, 2020 in Sweden, and January 26, 2021 in Canada. The growth phase was much more rapid during the 1st wave than during the 2nd wave in all three countries despite different government responses. In addition to government restrictions of interactions between young people (I_{YY}), there were voluntary restrictions of activity. Just because government permits a restaurant to be open to the public does not mean that the public will go to the restaurant. All other things being equal, everyone would prefer to avoid COVID-19 than become a case. The question for each person, aside from government mandates, is how much risk will an individual bear to pursue economic activity? With the media shouting out the new cases and deaths from COVID-19 together with hysterical assertions that civilization will collapse if schools open or people attend a funeral, there should be no surprise that people voluntarily abstained from interacting with each other. Voluntary suppression of I_{YY} has the same effect as government suppression of I_{YY}. In addition, one sees further stratification of the young population into those willing to take more risks and those willing to take fewer risks. These voluntary effects can slow the rise in deaths, delay the peak in deaths, and lead to death plateaus instead of a decline phase to zero just like government mandated lockdowns. In an ideal world, suppression of I_{YY} will defer deaths by prolonging the achievement of herd immunity but keep the total number of deaths equal once herd immunity is achieved. However, there is a very real effect of alert fatigue. People can remain vigilant for short periods of time, but government lockdowns cause people to develop alert fatigue and they become careless in their efforts. Masks are worn below the nose. People pretend to social distance but get closer together over time. People start to eat and drink in more places than before. I have personally witnessed this alert fatigue among health care workers who are more aware of the consequences than anyone else. Rather than the French having a 2nd wave starting in August, alert fatigue together with relaxation of government mandated lockdowns caused deaths deferred from the 1st wave to appear. The slow growth phase merged with the 2nd wave outbreak in the winter of 2020. The lockdowns in France during the 1st wave prevented herd immunity from being achieved, so France continues to have a plateau of death at this time while cases and deaths decline in much of the remaining world. This effect was less apparent in Canada but not entirely absent. Canada had a very slow rise time of its 2nd wave, a very delayed peak, and its decline phase has approached a plateau of death rather than decline to zero. The data in Figure 8 end on March 10, 2021, but the plateau in Canada persists to the current time (March 27, 2021 as of this writing).

Alternatives to Lockdowns

I have been asked to discuss alternative policies, treatments, and other remedies and compare these alternatives to indiscriminate lockdowns such as were mandated in Canada in response to COVID-19.

This is a bit awkward as I have devoted the preceding space to prove that restrictions on economic activity of young people interacting with other young people achieved more deaths than would have occurred by doing nothing. When I say that lockdowns resulted in more deaths than would have occurred by doing nothing, I am not including the deaths of despair caused by psychological harm from lockdown, delayed medical therapy for problems other than COVID-19 caused by lockdown, or deaths caused by having a lower standard of living resulting from lockdown. These issues will be covered by other experts. I am stating that the Canadian lockdowns resulted in more deaths from COVID-19 than would have occurred by doing nothing. The theory presented above demonstrates that restrictions of I_{YY} resulting from closures of schools, pubs, restaurants, gyms, and other businesses used mostly by young people could not possibly have resulted in fewer deaths from COVID-19 even in an ideal world without alert fatigue. In a real world with alert fatigue, the lockdowns certainly made deaths from COVID-19 greater than would have occurred in the absence of lockdowns.

Focus on Cases is Misguided

The obsession over cases is misguided. The best-case scenario would have been rapid spread of COVID-19 through the young population leading to large numbers of cases in people with a very low case fatality rate. The rapid growth phase would be followed by a rapid decline to zero cases. This best-case scenario did not happen because the media and so-called experts obsessed over the number of cases in young people who have a very low case fatality rate. Lockdowns of young people delayed or prevented the achievement of herd immunity, prolonged the time when young people could spread the virus to elderly people, and, when combined with alert fatigue, caused a higher number of COVID deaths spread over a longer period of time. The science and the data on this issue are quite clear.

Proper Metrics for Monitoring Effectiveness of Policy

Deaths are the best metric, but this provides information after the fact. Suitable proactive metrics would be cases in people of age 70 and over, hospitalized patients, patients requiring intensive care, or patients requiring ventilator support. I think the best metrics for a “scoreboard” to provide guidance to authorities and information to the public would be daily plots of new cases in people of age 70 and over, daily plots of active cases in people of age 70 and over, daily plots of hospitalized patients in people of age 70 and over, daily plots of patients of all ages requiring ventilator support, and daily plots of deaths.

Both cases and deaths are difficult since we have no test confirming that disease is caused by COVID-19. We can only determine whether the SARS-CoV-2 virus is present or not and the PCR test has many problems with false positives. This is the best we can do at the present time. According to the CDC, as many as 94% of patients with positive PCR tests have at least one co-morbid condition that could plausibly be the cause of illness or death.[25] I recently examined hospital records on 45 consecutive hospital deaths in patients with positive PCR tests from April 1 – September 1 at University Medical Center (UMC) in Lubbock, TX. I scored the patients as 0 (death certainly due to other cause), 1 (death more likely due to other cause than due to COVID), 2 (death equally likely to have been due to other cause or COVID), 3 (death more likely due to COVID than other cause), and 4 (death certainly due to COVID). I found that the mean score was about 2, so the number of deaths due to COVID was probably 23/45. I had two other experienced clinicians review the same information blinded to my score, and we all three agreed on the score for only 11/45 patients. This shows how difficult it is to count cases and deaths even if one is motivated by truth rather than financial incentives.

Policy Alternatives

Some policies would have decreased deaths from COVID-19. Policies leading to increased compartmentalization of vulnerable elderly people would have improved mortality. These policies limit the deaths from a single transmission of COVID-19 from a young person to an elderly person by limiting the number of elderly people who will come in contact with an infected elderly person. Note that increased compartmentalization does not mean isolation. Isolation has its own problems and should be avoided. Limiting elderly community groups to small numbers (2-3) will have improved mortality than large community groups. Small groups of 2-3 decrease the importance of masking or social distancing which can be very difficult to implement in elderly groups.

Other policies that decrease the number of deaths resulting from each elderly COVID-19 case (α in Equation 4) would improve mortality. These policies include, but are not limited to, improving nutrition, improving sleep quality, exposure to fresh air and sunshine, and improved hygiene of elderly people. The government oversees institutional care for the elderly, so these effective policies could be implemented by the government.

Policies that improve I_{EE} will mostly overlap with policies that improve compartmentalization. Policies that reduce virus transmission from interactions between young and elderly people (I_{YE}) will also reduce mortality. Some of these interactions cannot be avoided as many of the institutionalized elderly require assistance in eating, bathing and using the toilet. Social distancing is not practical for these activities. There is no substitute for careful screening of personnel involved in direct care of elderly people. Temperature checks on a daily basis is a relatively simple and non-intrusive safeguard. Rather than screening with mucosal swab PCR tests on every young caretaker every day, it is more practical to screen anyone with fever or new symptoms of cough. If, despite, screening, an elderly patient contracts COVID-19, the source can be traced to the caretaker of the elderly case, and the young caretaker can be isolated or moved to other duties while waiting for the infection to resolve. Contact tracing is made easier with smaller groups of elderly people and fewer caretakers per group. Just as small elderly groups will have lower mortality than the same number of elderly housed in large groups, lower mortality will be achieved by compartmentalization of the caregivers. A large number of caretakers who each care for only a single elderly group will have lower mortality than having a small number of caretakers who each care for a large number of elderly groups. Many nursing homes employ small numbers of respiratory therapists, physical therapists, and nurses each of whom are very specialized and provide care to the entire nursing home population. This arrangement will have higher mortality than a large number of caretakers with basic skills in respiratory care, assistance with mobility, and basic nursing who function like live-in nurses with each elderly group.

Variants of SARS-CoV-2

Like all life forms, viruses evolve. New strains of respiratory viruses appear. The CDC has been looking for the emergence of new strains of SARS-CoV-2.[26] Detection of variants require specialized testing above and beyond the usual PCR tests. The CDC processes up to 750 samples per week to monitor variants. There are three classifications of variants.[27] In order of increasing impact on clinical care, the categories are: Variants of Interest; Variants of Concern; and Variants of High Consequence. There are currently no examples of Variants of High Consequence. The Variants of Interest remain more of a curiosity than a threat.

Variants of Concern

Evolution is a process by which life forms become more successful through mutation and natural selection. More successful is defined on the basis of propagation and survival. Over time, successful mutations make respiratory viruses more contagious, more infectious, more resistant to therapy, but **LESS** lethal. This is the usual pattern of variants. There are 5 variants that the CDC lists as Variants of Concern. Most of the traits of these Variants of Concern are increased transmission, and resistance to treatment. One Variant of Concern, identified as B.1.1.7 and known as the British variant, has received publicity for increased lethality.[26]

B.1.1.7 British Variant

CNN reported that the British variant was 64% more deadly.[28] The Hill reported the British variant was up to 100% more deadly.[29] Both reports were based on a study in the British Medical Journal.[30] The inclusion criteria were age greater than 30 years of age and a positive test with symptoms. The end point was survival for 28 days. The British study found that of 54,906 patients who tested positive for the British variant, 54,680 (99.6%) survived. This was the so-called deadly variant since 54,765 (99.7%) with the original variant survived. The 64% more deadly figure cited by CNN comes from the odds ratio of 227 deaths vs. 141 deaths. The up to 100% more deadly figure cited by The Hill comes from the upper limit of the 95% confidence interval for the odds ratio. Obviously, you will generate more clicks and sell more ads by scaring people with 64% or 100% more deadly than by telling them that survival decreased from 99.7% to 99.6%. The use of odds ratios to compare two sets of rare events without providing the context that both sets of events are rare is a deceptive misuse of statistics. This practice is commonly used by the pharmaceutical industry to convince practitioners to use a new product on the basis of a seemingly large appearing odds ratio that represents a clinically inconsequential absolute benefit.

Impact of Variants of Concern

The existence of Variants of Concern, including the British variant, does not change any of my conclusions. My conclusion that closure of schools and lockdowns of restaurants, pubs, gyms, and other businesses catering primarily to young people resulted in a greater number of deaths in Canada from COVID-19 than would have occurred in the absence of lockdowns is not changed, whatsoever, by the existence of Variants of Concern including the British variant. The clinical management of patients with COVID-19 in my own medical practice has not been changed in any way by the existence of Variants of Concern including the British variant. I do not test patients for variants as this testing would not change my therapy and would have no significant change in information I give to patients including prognosis. None of my colleagues in Lubbock, TX test patients for variants for the same reasons given above.

Letter by Concerned ICU Physicians

A group of concerned intensive care unit (ICU) physicians sent a letter on April 1, 2021 to Premier Ford, Minister Elliott, and Dr. Williams.[31] This letter is an emotional appeal for government to “do something” even though no specifics about what should be done are provided. The letter does not provide any data to support assertions made.

The 1st assertion is that the use of ICU capacity as a benchmark is a “very dangerous path.” The concerned physicians do not offer a better alternative for the benchmark. The next assertion is that Variants of Concern (VOC) are spreading exponentially all over the province and will overwhelm the ICU

capacity. It is not explained how the ICU capacity benchmark will fail to signal the problem as VOC cases fill up the ICUs.

The concern about VOC is expressed as another assertion: “allowing these VOCs to spread exponentially is unethical.” Contrary to what these concerned physicians may think, the virus does not require human permission to grow exponentially. The growth of all respiratory viral outbreaks is initially exponential as explained in the section of this expert opinion labelled: Predicted Mortality Curve in Uniform Population. There is nothing special about either SARS-CoV-2 or any of the Variants of Concern with regard to exponential growth. All respiratory viral outbreaks exhibit a self-limited exponential growth phase. The greater the growth rate, the sooner the exponential growth phase ends. No government intervention could possibly prevent this exponential growth phase. This is not a matter of morality, ethics, strength of human will or any other character of human behavior; this is basic fact derived from the mathematics of combinatorial probability.

The concerned physicians offer no solution to the exponential growth phase. There is just a demand that government do something. The concerned physicians fail to acknowledge that dire consequences in other jurisdictions of the exponential growth phase of VOC failed to materialize. The concerned physicians fail to address the important point that by delaying the achievement of herd immunity, previous government mandated public health policies – including lockdowns of young people – have created the conditions that make VOC so worrisome in Ontario while in other jurisdictions the virus and all of its variants have run its course. For example, in my own community of Lubbock, TX, following the cancellation of all government mandated COVID-19 public health restrictions by Governor Abbott, both new cases and deaths have continued their declines toward zero.[32]

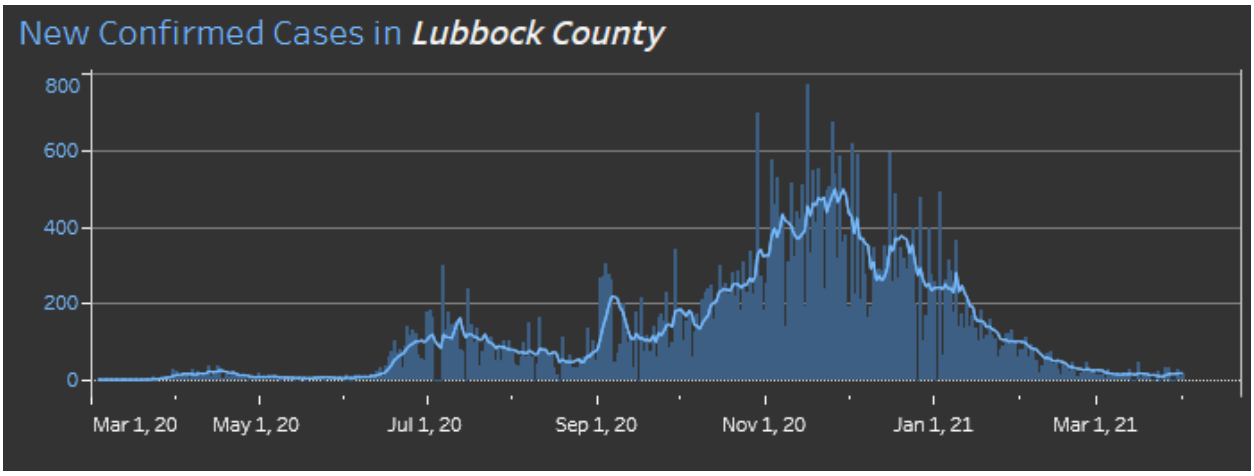

Figure 9: New Cases of COVID-19 in Lubbock County, Texas.[32] Data are from the official State of Texas COVID-19 website.

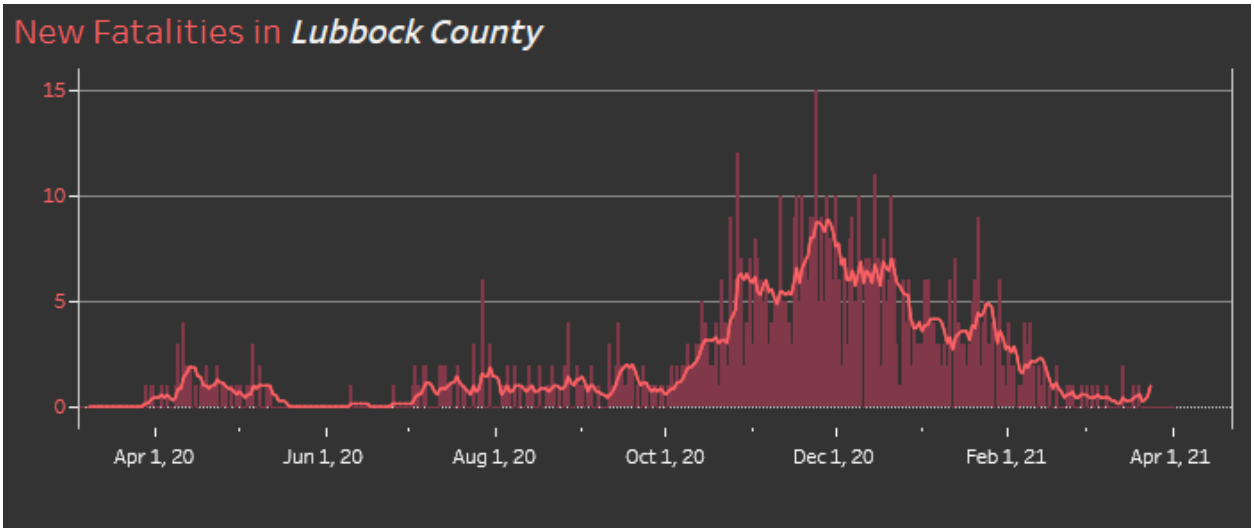

Figure 10: New Deaths in Lubbock County, TX.[32] Data are from the official State of Texas COVID-19 website.

The concerned physicians assert that: "each person who gets infected [with variants of concern] has a higher chance of hospitalization, ICU admission, and death." The only variant that has even been suggested to be more lethal is the British variant.[27] As shown above, the survival rate from the British variant is 99.6% compared with 99.7% for the original strain. Furthermore, there is no evidence that case fatality in Canada is getting worse due to this British variant. From the 7-day moving average data on the Worldometer page for Canada[33] the peak in new cases for the winter outbreak was 8,885 new cases on January 9, 2021. The peak in deaths was 163 and occurred 17 days later on January 26, 2021. This time lag of 17 days is consistent with the previous estimates of how long (on average) it takes exposure to progress to death. From these data, the reported case fatality rate can be estimated to be 1.83% at the peak of the winter outbreak. The exponential growth phase in new cases that has the concerned physicians so concerned began about March 8, 2021. There were 2,952 new cases on March 8, 2021 and 28 deaths 17 days later on March 25, 2021 for an estimated reported case fatality rate of 0.95%. Since March 8, 2021, reported new cases have increased and deaths have slightly declined, so the reported case fatality rate is falling rather than rising. Whatever the concerned physicians have seen or heard about current lethality, the data say otherwise.

Conclusions:

- There is nothing novel about COVID-19: By far, the most common cause of death from COVID-19 is acute respiratory distress syndrome (ARDS). There are literally too numerous to count distinct causes of ARDS[34] of which SARS-CoV-2 is only one. Sepsis is the leading class of causes of ARDS. Sepsis can be caused by infection, inflammation, or necrosis of any tissue or organ. ARDS can result from the inhalation of hundreds of distinct airborne toxins. ARDS can result from pneumonia caused by hundreds of distinct pathogens. The severity of illness, mortality, and course of recovery from ARDS is independent of the cause of ARDS. COVID-19 is just one of many types of ARDS.
- Concern about the B.1.1.7 or British variant of SARS-CoV-2 is exaggerated. The British variant is widespread in Texas. The CDC estimated that as of April 6, 2021 25.2% of new cases in Texas

were caused by the B.1.1.7 or British variant.[35] When Governor Abbott opened Texas up 100% and removed all state mask mandates, numerous so-called experts predicted an apocalypse of new cases and more deaths due, in part, to the rising prevalence of the British variant. The so-called experts were all wrong, however, and new cases of COVID-19 and deaths from COVID-19 continue to decline without any evidence that the relaxation of restrictions had any effect on outcomes whatsoever (see Figures 9 and 10).

- Texas is showing the rest of the world the way. Just as Sweden provided an example of courage early on in March of 2020, Texas is demonstrating to everyone what happens when lockdowns are lifted. In the month since Texas was opened up 100%, new cases of COVID-19 and deaths due to COVID-19 continue to decline. In my home of Lubbock County, new cases and deaths are near zero. None of the proponents of lockdowns, including Dr. Fauci, can explain the data in Texas. I have provided you with a detailed explanation of the data in Texas, Canada, and France. As I have discussed at length above, lockdowns do not save any lives. At best they defer deaths to some future date at enormous current cost. At worst, you end up like France with a plateau of death without any end in sight (see Figure 8).

References:

1. https://pulmonarychronicles.com/index.php/pulmonarychronicles/article/view/683/1509
2. https://mises.org/wire/covid-19-lockdowns-are-neither-necessary-nor-scientific-nor-helpful
3. https://pulmonarychronicles.com/index.php/pulmonarychronicles/article/view/755/1651
4. https://mises.org/wire/why-americans-should-adopt-sweden-model-covid-19
5. https://www.aier.org/article/lockdowns-of-young-people-lead-to-more-deaths-from-covid-19/
6. https://www.aier.org/article/covid-misclassification-what-do-the-data-suggest/
7. https://pulmonarychronicles.com/index.php/pulmonarychronicles/article/view/791/1753
8. https://mises.org/wire/mandatory-covid-19-vaccination-unethical-and-unscientific
9. https://mises.org/wire/what-covid-vaccine-hype-fails-mention
10. https://mises.org/wire/how-much-does-pfizer-vaccine-actually-reduce-risk-hospitalization
11. https://mises.org/wire/what-weve-learned-israels-covid-vaccine-program
12. https://link.springer.com/article/10.1007/s00508-020-01760-4
13. https://www.cms.gov/files/document/se20015.pdf
14. https://www.bmj.com/content/370/bmj.m3563
15. https://www.imperial.ac.uk/media/imperial-college/medicine/mrc-gida/2020-03-16-COVID19-Report-9.pdf
16. https://www.aier.org/article/the-disease-models-were-tested-and-failed-massively/
17. https://covid.cdc.gov/covid-data-tracker/#demographics
18. https://www.worldometers.info/coronavirus/
19. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2652751/
20. https://www.aier.org/article/lockdowns-do-not-control-the-coronavirus-the-evidence/
21. https://thefatemperor.com/wp-content/uploads/2020/11/1.-LANCET-LOCKDOWN-NO-MORTALITY-BENEFIT-A-country-level-analysis-measuring-the-impact-of-government-actions.pdf
22. https://www.aier.org/article/lockdowns-dont-prevent-coronavirus-spread/
23. https://www.aier.org/article/twelve-times-the-lockdowners-were-wrong/
24. https://www.aier.org/article/new-study-indicates-lockdowns-didnt-slow-the-spread-of-covid-19/

25. https://www.cdc.gov/nchs/nvss/vsrr/covid_weekly/index.htm#Comorbidities
26. https://www.cdc.gov/coronavirus/2019-ncov/cases-updates/variant-surveillance.html
27. https://www.cdc.gov/coronavirus/2019-ncov/cases-updates/variant-surveillance/variant-info.html
28. https://www.cnn.com/2021/03/10/health/coronavirus-variant-uk-more-deadly-study/index.html
29. https://thehill.com/changing-america/well-being/medical-advances/542602-covid-19-variant-from-uk-spreading-in-us-is-up
30. https://www.bmj.com/content/372/bmj.n579
31. April 1 letter by group of concerned ICU physicians
32. https://dshs.texas.gov/coronavirus/additionaldata.aspx
33. https://www.worldometers.info/coronavirus/country/canada/
34. https://www.mayoclinic.org/diseases-conditions/ards/symptoms-causes/syc-20355576
35. https://www.cdc.gov/coronavirus/2019-ncov/cases-updates/variant-proportions.html