Review of calculated SARS-CoV-2 infection fatality rates: Good CDC science versus dubious CDC science, the actual risk that does not justify the "cure" - By Prof Joseph Audie

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Review of calculated SARS-CoV-2 infection fatality rates: Good CDC science versus dubious CDC science, the actual risk that does not justify the "cure"

By Prof. Joseph Audie

26 August 2020

Introduction by Denis G. Rancourt:

Prof Joseph Audie and I have been corresponding about the ongoing COVID episode for some months, and he has previously written for the Ontario Civil Liberties Association, where I am a researcher.

Joseph Audie, PhD (biophysics), MS (biomedical engineering), BS (bioengineering) is a professor of chemistry. He has performed original drug design and discovery research and has published in scientific journals. Joseph is adept at finding errors in scientific papers in the medical field.

In this letter to me, Joseph accomplishes the following points:

- an explanation of the various kinds of fatality rates for a pathogen
- a review of the measured infection fatality rates for SARS-CoV-2
- a demonstration that a recently changed CDC evaluation is most certainly incorrect, along with an illustration of how not to do a meta-analysis
- his conclusion that "the absolute and relative 'flu-like' risk of death from a SARS-CoV-2 infection is far too low to rigorously justify governments imposing major disruptions to normal life, let alone the massive and indiscriminate 'lockdown' disruptions people have been forced to submit to and endure"

I have added a bibliography of the live links used in the text, below (in order of appearance).

Here is Joseph's letter:

Hello Dr. Rancourt,

I would like to call your attention to the CDC's second estimate for the infection fatality ratio (IFR) of SARS-CoV-2 and its reliance on a weak and error-laden meta-analysis in a failed attempt to support it.

As I am sure you know, the crude infection fatality rate (cIFR) refers to the ratio of deaths associated with or caused by a pathogen from among a sample of people with current or past infections that are typically detected using RNA or serum antibody testing. This must be distinguished from the crude case fatality rate (cCFR) which refers to the ratio of deaths associated with or caused by an infection from among a sample of infected people who also meet some medical case definition, typically having to do with the presence of clinical symptoms or hospitalization. The population IFR (pIFR) or population CFR

(pCFR) can be calculated when the sample data reflects a larger population or is adjusted to reflect a larger population.

To calculate an accurate IFR, care must be taken in calculating both the denominator (number of infections) and numerator (number of deaths). For example, according to the helpful Wikipedia account, 14 deaths and 712 RNA confirmed infections are associated with the SARS-CoV-2 outbreak on the Diamond Princess Cruise ship. Analysis of the press releases from the Japanese Ministry of Health (JMH), however, confirms 13 SARS-CoV-2 associated deaths, which implies a cIFR = 1.8%. Further analysis of the JMH press releases reveals that only 7 of the 13 people who died tested positive for SARS-CoV-2 RNA, resulting in a lower cIFR estimate of 0.98%. Additional scrutiny of the JMH press releases shows that only 4 of the deaths were directly attributed to or said to be caused by SARS-CoV-2/COVID-19, which leads to an even lower cIFR estimate of 0.56%. The average age on the Diamond Princess was about 58 years old, which does not reflect the age structure of a country like the US. Working from the Diamond Princess data available to him in early March (700 infected and 7 associated deaths), Dr. John Ioannidis of Stanford University calculated a point estimate for the pIFR of the US population to be 0.125%. The take away lesson from this exercise is that distinctions must be made (associated deaths, attributable deaths, etc.), apples and oranges (IFRs, CFRs, cIFRs, pIFRs, etc.) should not be mixed, and SARS-CoV-2 probably has a pIFR in the range of a strong influenza.

Recently, Dr. Ioannidis, who has arguably emerged as the world's leading expert on the SARS-CoV-2 IFR, published a pre-print in which he critically reviews 43 IFR estimates from 36 antibody serology studies, including his own study on Santa Clara County, CA. In the article, Dr. Ioannidis, for a variety of reasons, cautions against doing a meta-analysis on the IFR data and deriving a single best IFR estimate and instead opts for a more nuanced approach to data analysis, ultimately recommending the median pIFR as the best indicator of central tendency. Across 32 locations, Dr. Ioannidis finds median raw and corrected pIFR values of 0.27% and 0.24%, respectively. Dr. Ioannidis also provides pIFR results for 7 countries that have been reported in the media or in preliminary reports which yield median raw and corrected pIFR values of 0.15% and 0.12%, respectively.

Included in Dr. Ioannidis' review is the CDC's first official pIFR estimate of 0.26%, an estimate that is based on official CDC serology data and that can be readily calculated from officially reported CDC estimates for the population symptomatic case fatality rate (psCFR) (0.4%) and asymptomatic infection rate (35%) (0.26% = 0.4% x 0.65%). Importantly, the CDC's pIFR estimate for SARS-CoV-2 is in excellent agreement with the median corrected pIFR (0.18%) and crude mean corrected pIFR (0.35%) calculated from the 8 non-CDC pIFR studies on US states and cities reviewed by Dr. Ioannidis. For context, according to CDC data, the seasonal flu of 2010/2011 had a psCFR ≈ 0.18% which, assuming a 35% asymptomatic infection rate, implies a seasonal flu pIFR of ≈ 0.12%.

Perhaps even more importantly, the CDC's estimated pIFR of 0.26% is in excellent agreement with pIFR estimates that derive from arguably the single most rigorous pIFR study conducted to date by Professor Streeck and his team. The Streeck et al. study is based on data obtained from a spontaneous super spreading event that infected ≈ 15.5% of people in the town of Gangelt, Germany (population of 12,597) that occurred in pre-global lockdown February. The study's pIFR estimate of ≈ 0.28%, as calculated from such a natural experiment, probably provides the best available estimate for what can be thought of as the natural lethality of SARS-CoV-2 in a broadly representative population. Moreover, the estimate may represent an upper limit with respect to natural lethality, as Professor Streeck and his team were not able to confirm that all 7 recorded deaths (average age = 80.8 years) were caused by SARS-CoV-2, there were only 3 excess deaths with respect to the previous year, the number of infections does not reflect infections that could have been detected using other methods such as mucosal antibody and reactive T-

cell testing, the ability to detect infections can fall off with the passage of time, and that super spreading events tend to be on the deadlier side. Indeed, a simple excess death analysis suggests a pIFR ≈ 0.13%.

Other "natural experiments" that have occurred predominantly among working and retirement age adults in meat packing plants, prisons, and on navy ships also point to a natural pIFR ≈ 0.1%-0.3%. Several infection studies on diverse animals – cats, ferrets, macaques, hamsters - have yet to produce a single, unambiguous SARS-CoV-2 caused fatality and have universally failed to provide rigorous satisfaction of Koch's postulates, further confirming the modest natural lethality of SARS-CoV-2.

Ultimately, the tight agreement between the results of animal experiments, the natural pIFR estimates calculated by Professor Streeck and from other "natural experiment" data sets, the median pIFR values calculated across 32 and 7 global locations, respectively, the median and crude mean pIFR values calculated from the 8 independent US studies, and the pIFR calculated by the CDC from their own serology data is noteworthy and suggests the fundamental accuracy of the CDC estimate of pIFR ≈ 0.26% and that SARS-CoV-2 has the lethality of a severe influenza, like the influenza pandemic of 1957.

Given the above, it came as somewhat of a shock to learn that the CDC recently presented a second and higher pIFR estimate of 0.65%. The only justification provided by the CDC is that the pIFR is replacing the psCFR because it provides "a more directly measurable parameter for disease severity for COVID-19". This attempted justification, however, fails because, as demonstrated above, the pIFR is readily calculated from the CDC's original estimates for the psCFR and asymptomatic infection rate. In addition, the CDC does nothing to undermine let alone repudiate its original pIFR estimate of 0.26%. Indeed, the CDC's latest estimate for an asymptomatic infection rate of 40% implies an even lower pIFR estimate of 0.24%. Hence, we are left with as many as three official CDC estimates for the pIFR of SARS-CoV-2: 0.24%, 0.26% and 0.65%. The 0.65% estimate is an outlier, while the first two estimates of 0.24% and 0.26% are in excellent agreement and enjoy solid scientific support from multiple, independent studies. As will be shown below, the 0.65% estimate is based on a single meta-analysis of dubious quality that was recently published as a pre-print by Drs. Gideon Meyerowitz-Katz and Lea Merone.

The Meyerowitz-Katz and Merone article reports a point estimate for the population infection fatality ratio (pIFR) of 0.68% based on a meta-analysis of 26 studies that were retrieved from the peer reviewed and non-peer reviewed literature by 06/16/2020. The 26 studies report on the use of various methodologies – modeling, serological and observational – to estimate the infection fatality rate of SARS-CoV-2. While it represents a potentially helpful study, the study by Meyerowitz-Katz and Merone suffers from a number of errors that biases it in the direction of a high pIFR estimate which calls into question the CDC's citing it as the only support for its's new pIFR estimate of 0.65%. Indeed, it can be argued that the meta-analysis is so flawed its pIFR estimate is useless. What follows is a short overview of some of the more obvious errors that challenge the quality of the Meyerowitz-Katz and Merone meta-analysis and the accuracy of its pIFR estimate.

To their credit, Meyerowitz-Katz and Merone acknowledge that "… any meta-analysis is only as reliable as the quality of included studies …" By implication, excluding quality studies can also undermine the reliability of a meta-analysis. Both errors of commission and omission undermine the Meyerowitz-Katz and Merone study and are briefly discussed below.

Perhaps most importantly, Meyerowitz-Katz and Merone do not even mention, let alone critically engage, the IFR review article of Dr. John Ioannidis (discussed above) which was available as a first and second version pre-print on May 19 and June 8, respectively. This is a problem in its own right, as authors should contextualize their own research and critically discuss it in light of previous research.

Moreover, the omission of Dr. Ioannidis review article has important analytical implications. For example, Meyerowitz-Katz and Merone rejected the studies by Bryan et al. and Silveira et al. because they claimed "it was difficult to determine the numerator (i.e. number of deaths) associated with the seroprevalence estimate, or the denominator (i.e. population) was not well defined". Prof. Ioannidis, however, included both studies in his review and reported corrected pIFRs of 0.13% and 0.39%, respectively.

Meyerowitz-Katz also rejected the study by Sood et al. because it supposedly "explicitly warned against using its data to obtain an IFR". A reading of the Sood et al. article, however, fails to reveal such an obvious and explicit warning. Importantly, Dr. Ioannidis used the Sood et al. study to calculate a corrected pIFR of 0.18%.

Meyerowitz-Katz also excluded several studies, including blood donor sero-prevalence/IFR studies analyzed by Dr. Ioannidis, because "many studies only looked at targeted populations in their seroprevalence data, and thus could not be used as population estimators of IFR (pIFR)." Despite this, Meyerowitz-Katz included the study by Tian et al., which reports on the characteristics of a relatively small and targeted sample of hospitalized patients in Beijing, China and reports what is best characterized as a cCFR = 0.9% (as opposed to a pIFR).

Meyerowitz-Katz and Merone fail to even mention, let alone include in their analysis, the early pIFR study by Mizumoto et al. which was available as a pre-print in February and has since passed peer review and been published. Importantly, Mizumoto et al. used a modeling methodology to calculate a low pIFR estimate for Wuhan, China of 0.12%. Similarly, Meyerowitz-Katz and Merone are totally silent on the CDC's implied pIFR estimate of 0.26% (discussed above) despite the fact that it was publically available prior to 06/16/2020.

Having excluded several studies that reported relatively low pIFR values (0.13%, 0.39%, 0.18%, 0.12%, 0.26%) on dubious grounds and included at least one inappropriate study that reported a relatively high estimate of 0.9%, Meyerowitz-Katz and Merone arrive at their point estimate of 0.68% by meta-analyzing and quantitatively synthesizing results from the included 26 studies

Even had Meyerowitz-Katz and Merone included the low pIFR estimates and excluded the high and inappropriate estimate mentioned above, their meta-analysis and quantitative synthesis would still have suffered from serious problems. This is because the analyzed studies employed a range of disparate methodologies (modeling, observational, seroprevalence), ranged from highly biased to unbiased, suffered from high heterogeneity, favored Europe (with relatively high pIFRs) over other geographical locations (with relatively low IFRs), and many studies used no doubt different and many times frustratingly opaque criteria for classifying deaths. These are all red flags that undermine the case for using meta-analysis to report an unbiased and accurate point estimate for the pIFR. Indeed, in his review Prof. Ioannidis points out the many challenges of doing a meta-analysis and cautioned against such an approach, something Meyerowitz-Katz and Merone would have known had they included Prof. Ioannidis' article as part of their analysis. Ultimately, the available evidence suggests the 0.68% estimate put forth by Meyerowitz-Katz is, at best, highly biased on the high end of the pIFR range and, at worst, totally useless.

Worth noting is that the results of the Meyerowitz-Katz analysis imply a naïve or crude average pIFR for the United States of ≈ 0.58%. With the preceding in mind, it is not clear how the CDC used the results of the Meyerowitz-Katz analysis to arrive at its own pIFR estimate for the US of 0.65%, a national estimate

that is *higher* or *equal* to pIFR estimates for especially hard hit New York and New York City, for which corrected pIFR values of 0.54% and 0.65%, respectively, were calculated by Prof. Ioannidis.

To summarize: the CDC's original estimate for the pIFR = 0.26% is based on official CDC serology data, is consistent with the results of scientifically analyzed "natural experiments", and agrees nicely with the results derived from other studies that relied on international and US infection and fatality data. Additionally, the CDC has failed to provide any reason, let alone a good reason, for rejecting its estimate of pIFR = 0.26% and still makes the estimate available to the public. The only thing provided by the CDC in support of its new pIFR = 0.65% estimate is a single pre-print article reporting on a meta-analysis that suffers from numerous errors, including biased data inclusion and exclusion, resulting in a pIFR estimate that should be interpreted as either shifted to the high end of the pIFR spectrum or as totally useless. Hence, given the available evidence, the CDC's original pIFR = 0.26% estimate should be taken as the more accurate one.

In a very real sense, a discussion about whether or not the pIFR of SARS-CoV-2 is ≈ 0.26% or ≈ 0.6% is only of academic interest. Put simply, the absolute and relative "flu-like" risk of death from a SARS-CoV-2 infection is far too low to rigorously justify government's imposing any major disruptions to normal life, let alone the massive and indiscriminate "lockdown" disruptions people have been forced to submit to and endure, as such disruptions will inevitably unleash innumerable forces, including deadly forces, that will reverberate throughout society in predictable an unpredictable ways for years to come. Consider, for example, that according to one analysis even for the relatively high risk 60-69 year old demographic, a person's one year probability of dying only increases from a baseline value of 1.3% to 1.8%, assuming an age-specific SARS-CoV-2 IFR of 0.49% and absurdly high attack rate of 100%. A more reasonable but still high estimate for the increased one year probability of death from SARS-CoV-2 for 60-69 year olds, based on the Diamond Princesses' observed attack rate of ≈ 20%, would be from 1.3% to 1.4%. Does it really make sense for governments to impose unproven and massively disruptive lockdown style interventions – interventions all but guaranteed to destroy the religious, moral, legal, political, economic, educational and psychosocial fabric of a nation – in a quixotic attempt to reduce a relatively high risk person's odds of dying by 0.1%? Doesn't it make more sense for people to do what's always been done and take voluntary, evidence-based, and personalized approaches to protect such individuals that also respect absolute goods – the inalienable right to work and secure sustenance - and balance competing, relative goods – going to a concert or ballgame? The questions answer themselves. Indeed, it almost seems like a truism to point out that the imposition of coercive and unproven lockdown measures, on an entire population, amounts to a mass social experiment that is fated to fail and ultimately increase people's baseline probabilities of mortality and morbidity in myriad ways that will effectively negate and even reverse any hypothetical gains from mitigating SARS-CoV-2. There is even reason to think that lockdown measures will increase people's susceptibility to respiratory infections, including SARS-CoV-2 infections, and that thwarting microbial exchange with other humans, animals, and the natural environment could impair the proper function of people's immune systems.

SARS-CoV-2 poses a significant risk to a well-defined, vulnerable population of elderly and infirmed people and is a statistical non-issue for the vast majority of people. This is good news, for it empowers communities to adopt targeted and scientifically-based mitigation strategies, ultimately allowing everyone else to keep working to support their families, communities and the health care system, voluntarily practice standard cold and flu mitigation strategies, and ultimately acquire natural immunity, bring the epidemic to an end, preserve and perpetuate their way of life, and avoid the collateral damage wrought by imposition of the many crude and draconian interventions subsumed under the general term "lockdown".

The above analysis and policy recommendation should be seen as controversial. Indeed, it is only controversial because of mass hysteria. Rather, it follows logically from an accurate pIFR-based understanding of the "flu-like" lethality of SARS-CoV-2 and a simple relative risk analysis; it reflects common sense, universal practice, official pandemic mitigation planning, and the opinions of many experts, including a former lockdown proponent and architect; it has been demonstrated to work during the previous pandemics of 1957, 1968 and 2009; and perhaps most importantly, it has been shown to work in present day non-lockdown countries and free US states - such as Sweden, Japan, Belarus, Tanzania, Nicaragua, South Korea, and Taiwan, and North Dakota, South Dakota, Nebraska, Arkansas, Wyoming, and Iowa, respectively.

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