Journal of Risk Analysis and Crisis Response, 2021, 11(3), 104-128
https://jracr.com/
ISSN Print: 2210-8491
ISSN Online: 2210-8505

Article

Old Lessons of Risk Assessment and Management from the COVID-19 Pandemics and Individual Infections Dynamics

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Received: October 11, 2021; Accepted: October 27, 2021; Published: October 30, 2021

Abstract: "*Certainty creates strength…Uncertainty creates weakness*" (Barry, 2005) [1]. "*The theory is this, that it would be appropriate to believe in a proposition until there is a founded reason to suppose its truth. If this view were to become commonly agreed upon, our social lives and our political system would turn out completely changed.*" (Russell, 1928) [2]. "*The best way to prevent becoming infected is to avoid being exposed to the virus*" (Source: www.astho.org/COVID-19/Q-and-A/) [3]. The recent and ongoing COVID-19 pandemic is confirming that our society is vulnerable to global risk and that science and politics are challenged by the associated high uncertainties. This makes a number of old, foundational questions on risk and its management re-emerge. In this paper, specifically for the risk posed by the current pandemic and the infection spreading phenomena driving it, we observe from data and show from theory that there are four characteristic and very human-determined timescales for infection-spread rates. Then, we conclude on the need of putting the humans in the middle/focus of risk, as they are the ones that ultimately take decisions (almost rationally) and live their outcomes. So, we argue the obvious: that is, that for managing risk, it is necessary to realize and accept rationally that risk is not absolute- it is relative and in the uncertainty of the occurrence of different events, some just have more chance of occurring than others (i.e. high versus low chance). To evaluate and compare risks, as a society we should weigh, rank and decide the intertwined balances and resulting inequalities.

Keywords: COVID-19; Pandemics; Individual Infections; Risk Assessment; Risk Management; Old Lessons

1. Introduction

The recent and ongoing COVID-19 pandemic has highlighted that there are high uncertainties to cope with and significant challenges to be faced by society and science in our global world (Aven and Zio, 2020) [4]. In managing global risk, old questions reappear strongly: What is an acceptable risk? How do we compare one risk to another? What risks should we take or not take? Who makes those decisions? And why and how should these decisions be made?

These are all important questions that affect everyone wellness and living.

In this paper, we give clear and strong statements on:

- The need of putting humans in the middle/focus, as "almost rational" beings that ultimately take decisions and live their outcomes.
- The inevitability of living under a no-zero risk condition.
- The rational value of being capable of accepting that there is never zero risk.
- The importance of comparing risks, for their prioritized acceptance.
- The understanding of what can we do to prevent/mitigate/control risks in relation to the associated uncertainty (what we know and what we do not know).
- The role and duty of the different actors involved in risk decisions (science, government, people, etc.).
- How to move forward (a framework to proceed rationally under risk).

We emphasize the obvious: in the end, what really matters is what risk we are willing to take as individuals for the products and services that we need for living and desire for our well-being.

In relation to the risk posed by the current pandemic and the infection spreading phenomena driving it, we observe from data and show from theory that there are four characteristic and very human-determined timescales for infection-spread rates.

On the other hand, in the current era of information and communication, there is a great deal of common sense and sources information available on the internet (e.g., www.CDC.gov), however accompanied by disinformation. A most interesting elementary report in this sense is Covello and Hyer (2020) [3].

While in the interest of understanding, there is of course a paucity of numbers in these popular communications and the statements are given literally clear and concise, and authoritarian. In the face of the uncertainty in the physics of infection spreading, well-meaning and professional health officials and organizations want to take every precaution possible against the spread, seeking almost zero new cases, enforce control or crisis management, and desire -as we all do- complete absence, rather than risk tolerance for accommodating the new living with the disease. We can all take precautions, and we do, but what society actually has to do is *understand how to balance the risks in order for the maximum semblance of normal life to continue for human society.*

Our previous work showed how the recovery trajectory trends in the rates of infections were of universal shape and timing; quoting Duffey and Zio (2020a) [5]:

"The strong message here is that the rational and logical approach to dealing with the risk of the occurring pandemic (as with any other risk, for that matter) is to limit own personal and potential exposure, and to minimize both the size and scale of the potentially exposed population. This is precisely what governments and contagious disease experts have been saying all along- but is also what any individual should be doing anyway while exposed to the risks of ``normal'' life".

Humans are thinking and reasoning -but not entirely rational- creatures. We have fears and desires, and take accordingly risks even when we should not. We look to authority for (and many times pretend) help, guidance and advice, but eventually take our own decisions and still make our own mistakes. We often do not know who to trust, while realizing that although we are part of a larger society we are also, in the end, on our own when it comes to our health, job, personal decisions and the risks we take.

To evaluate and compare risks for their control and management, as a society we should weigh, rank and decide the intertwined risk balances, and resulting inequalities:

Personal vs. societal - or private vs. public.

Economic vs. medical - or wealth vs. health.

Political vs. personal - or policies vs. choices.

Science vs. beliefs - or facts vs. theory

Legislation vs. voluntary - or laws vs. traditions.

The overall balance ideally should be objective and quantified technically and numerically - while recognizing that subjective and qualitative risk perceptions often dominate. To date, for COVID-19, and many other key societal issues, this perfect balance is not being achieved. The well-known risk acceptance paradox is that "events are acceptable to society until they actually happen" (Duffey, 2020) [6].

For the management of risk, it is necessary to realize and accept rationally that risk is not absolute- it is relative. This is because we are exposed to a multitude of potentially harmful events all the time- of contracting a disease, being the victim of a crime, being subject to an accident etc. In the uncertainty of the occurrence of events, some just have more chance of happening to an individual than others (i.e., high versus low chance). The outcomes of the different events may be different, also depending on the individuals, and are sometimes used to weigh or rank the risk-order of the types of events (i.e., airplane vs. car accident, hurricane vs. earthquake, chemical vs. nuclear accident). The risk for a single individual may be different from that for the overall society – i.e., some go to war, some do not, some are exposed to the consequences of an industrial accident, some are not, some are more vulnerable to a given infection than others. Our preparedness to confront with the different risks - both personally and societally- also differs, and some may be survivable (like bankruptcy) and some may not (like a terminal disease).

With regards to the COVID-19 pandemic infection, the virus does not worry or distinguish about where or when but only who, as deaths follow the usual age distribution (as expected, mostly hitting individuals with pre-existing conditions and > 65 years of age, which is a not-surprising, already well-known problem). The number of deaths in a region, then, vary by medical system, age and pre-existing propensities for pneumonia and other complications, as this has always been the case, the most vulnerable targets being mainly over 65 and the already sick or medically "challenged" individuals).

Just looking at, say, the total numbers of deaths -upsetting as each and all are- does not indicate what is really happening due to the virus, only how resistant we are or are not to its ravages, and whether the health system is keeping an accurate body count and somehow effectively coping with and curing the most severe cases. As for what an individual can do for his risk of infection and death, one must rely on "guidance" from health "authorities" whose professional job is simply to prevent you getting sick (see for example US CDC Guidance webpage https://www.cdc.gov/coronavirus/2019-ncov/).

To set some orders of magnitude for the ongoing pandemic, the average chance of being infected by COVID-19 in the USA is 1.7% as of 1st September, 2020, or one in 50 to 60 anywhere; and your chance of dying (usually from pneumonia –like bacteria) is on average about 2% of that number, although higher if you have pre-existing conditions singly or in combination (including overweight, blood pressure, drug abuse, cancer, asthma, smoking, COPD etc.). Resurgences and/or second/third or more waves do and will occur, as the virus embeds itself and its ever-evolving progeny in the community, even with successful vaccines - just like influenza does as it endlessly mutates.

As already known and as confirmed by the COVID-19 (Covello and Hyer, 2020) [3]:

- "Viruses are a threat to all people, regardless of race, ethnicity, or the country one lives in.
- Viruses do not target people from specific populations, ethnicities, or racial backgrounds.
- Viruses do not respect borders and do not discriminate among different types of people."

In the management of this situation, medical professional's focus on providing care, beds and prevention, political professionals focus on supporting economy, jobs and providing social welfare, people focus on getting on with their lives in acceptable well-being and perceived safety. For the management of the situation, response plans and emergency services are helpful but are often ad-hoc and reactive, not pro-active and pre-emptive: we diagnose the disease and treat the symptoms after it appears.

In this frame of living under risk, what really matters is what risk will we take as individuals, and not simply to blame the government or support systems for the harm they can bring to us.

Using data, we and others can show that the risk of COVID-19 infection is comparable to other accepted/acceptable risks of everyday life everywhere in the modern computerized/ industrialized world, whereas the associated small risk of death is determined by literally how healthy you are anyway if and when infected. Neither the risk of infection and/or death does depend on politics, economics, environment or societal norms - the virus is literally colorblind. This does not mean that some people, "races" or cultures may not be more susceptible - as always for these types of diseases, the obese, hi-blood pressure, diabetic, immune-system challenged, diseased, asthmatic, malnutritional etc. will be at larger risk than the others. This has been always known, - what seems not to have been known before is how well the "new" COVID-19 virus follows the traditional trends and targets- and whether the whole health and societal system can be overwhelmed by sick and dying people.

2. What We Know: Removing Mythology

COVID-19 spread mechanism and behavior are largely the same as previous pandemics and outbreaks of flu, and other viral infections (SARS, STDs...) – there is not really a new behavior and, absolutely, not racially selective: the weakest get the sickest, the most exposed likely get infected, and the infection and resulting death numbers largely depend on luck and personal physical propensity (see e.g., Glezen, 1996) [7].

A virus of any name is not politically or economically motivated or influenced, but just spreads where it does best and attacks when and where it can; so, "opportunistic" infections are random and caused by person-to-person contact(s), which is why the virus does its best in crowds and social gatherings (bars, discos, riots, protests, parties and sport arenas, …). This ubiquity is shown by the fact that infection rates are pretty much the same, independent of race, religion, country, food, pollution and society …or place on Earth (see Italy, New York, UK and Turkey, for example). We do not really know why there are "typical" curves, and predictions by complicated models based on specific assumptions turn out poor most of the time, given the high uncertainty in the underlying physical mechanisms and related representative parameters. This is shown, for example, in the comparison for California of the results of various Ro models[1], which intend to quantify the factor

[1] The conventional arbitrary fitting proportionality parameter, Ro, is not to be confused with the actual infection rate, R, due to the changing number of cases, $R = 1/(N - n)\ dn/dd$.

(denoted as "R-nought", Ro, or Reff) that is supposed to indicate if infections are growing or declining (see Figure 1). It is known that the traditional Ro medical models for infection spreading contain many empirical and freely adjustable fitting parameters, and yet…they are turning wrong in their predictions (see Figure 1).

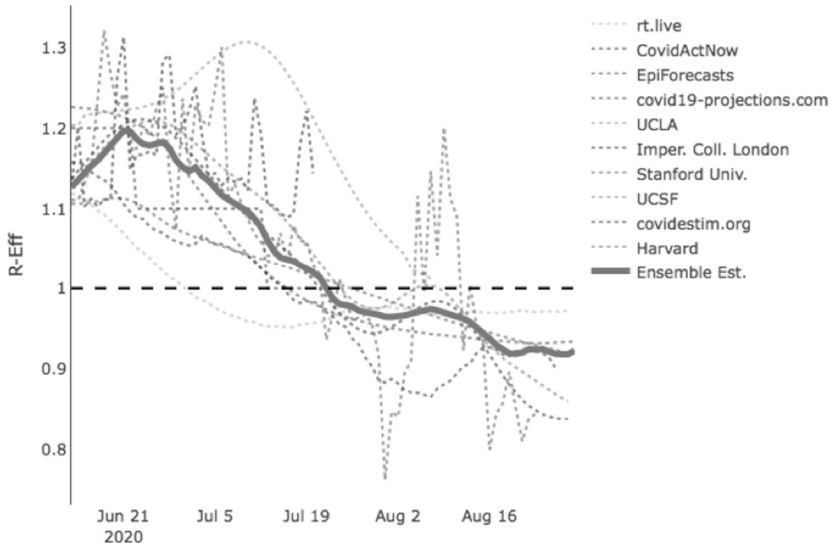

Figure 1. Ro-models' predictions for California, USA, show major differences among various high-powered models - the thick black line is the average from the ensemble: but which should one believe for active risk control? (Source: California DPH, 2020)

The infection fraction is independent of local or even national countermeasures - there is a universal relation that (as of today) the infection fraction is 1 in 18000, and it works for local regions of 100,000 people as well as for large populations like all the US. So, indeed, the virus seems not to care where you are or anything else.

In fact, we observe from the data and theory that there are four all-too-human characteristic timescales for infection spread rates, caused by:

initial rapid growth determined by the individual incubation time and rapid person-to-person contact (*5 or more days*).

peaking and, then, declining as simple personal countermeasures like improved hygiene, greater awareness and some social "distancing" reduce/slow the transmission rate (*30 or so days*).

inevitable virus spreading to the broader community causing longer term resurgences and "waves" even in isolated places, and despite monitoring, masks and quarantines (*100 or more days*).

establishing effective global response determined by human resilience to infections and pandemics (*typically a year or more*).

Resurgences (second waves) of COVID-19 occurred some 100 days (simultaneously- we postulate below why this number) after the first peak or the initial cases, independent of countermeasures and quarantines. The virus had spread and become embedded (hidden) in the communities everywhere, and even re-occurred in isolatable island states (New Zealand and Hawaii), traditional mask-wearing cultures (Japan and S Korea) and with limited countermeasures (Sweden).

Countermeasures of restricting contacts (social distancing) and extra hygiene help as always, whereas masks worn by the non-infected are largely symbolic. They are known to be relatively

ineffective other than if someone is already infected or if they are of N95 type (Barry, 2005) [1]. And, of course, noses are uncovered, food is eaten, masks forgotten and flimsy, or just rejected as inconvenient or making it hard to breathe (Rajaonah and Zio, 2020) [8]. Complete "lockdowns" of restricting freedom of movement, complete quarantines and selectively suspending business activities pose clearly a major economic risk themselves, with potential catastrophic consequences as well: we are back to the issues of comparing risks and risk acceptance. And one should consider that infections and spread are really never eliminated, despite vaccines, just like it is shown by the flu, VD, malaria, …; so, zero infections is not really a good goal to set for a rational and balanced risk management – see New Zealand. Humans have always lived with infections around them attacking their immune systems, and will continue to do so in the future.

Other facts should also be considered in the risk comparisons and risk acceptance for a rational and balanced risk management, like that (Covello and Hyer, 2020) [3] "Most COVID-19 deaths are among the elderly: 80% of deaths occurred amongst those aged 60 years or older and the highest percentage of severe outcomes were among persons aged 85 years or older. In Italy, one of the most affected European countries, nearly 90% of deaths are in those aged 60 years or older."

And in the end, if the personal risk of death from COVID-19 is less than that for other causes, it could be regarded as actually acceptable. But the problem is that what risk is acceptable for the specific root cause of pandemic has not been defined, and so we try to reflect also on this here in this paper.

3. What Do We Not Know: True Uncertainty?

As individual human beings, we pose questions to ourselves on the risk brought by the current pandemic and suggest answers based on placing our risk in perspective- comparing to the normal things we all are subjected to in life.

3.1. How Low Close to Zero Can the Risk of Getting Infected Go?

The situation is quite predictable, and what is also predictable is the "old" lesson that there is uncertainty in this. We delve into this, in the following.

3.1.1. There Is a Minimum Attainable Infection Rate That Is Not Zero.

This is what is possible/achievable with modern medicine etc. - and we know from flu that despite vaccines etc. certain vulnerable (most often corresponding to older) people will die from infection and we cannot eliminate this. We also know that the virus will also mutate, while the young and the restless will spread it anyway in parties, gatherings and normal social behavior.

Countermeasures like mandating masks (often ill-fitted) that do not work, will not work to protect us from getting infected (see Japan and Korea with traditional "mask cultures", and the physics of respirable particles) as also shown by the 1918 pandemic (Barry, 2005) [1]. They are best at stopping the infected spreading, but not the uninfected receiving; it does, however, satisfy perception as it feels like a personal risk reduction measure but it is not. On a large scale, people do not fully cover their nose, the non-medical designs have gaps and poor filtration, and there becomes a certain symbolism involved, sometimes referred to as "mask shaming" or even a fashion statement.

The rational key on this is accepting that there is no such thing as zero risk and infections will occur.

3.1.2. The Minimum Socially Acceptable Infection Rate.

Despite our hopes, the virus itself has no social conscience, no societal preferences and no idea about our "context", our ethics or "moral compass: it just infects whomever it can, when it can and where it can.

The minimum infection rate is what society and politicians and people have to put up with, and varies wildly depending on societal attitudes. As almost a matter of pride, New Zealand seeks to have zero infections and has endeavored to eliminate any cases; meanwhile, the USA just wants it to be less than what it was and compared to "normal" US societal risk coming from other risk sources, and thus considering the number of injuries on the road of about 3 million a year, or deaths by violence (50,000), automobile accidents (35,000), and from medical errors (250,000) and "normal" respirable diseases (150,000) (see e.g., CDC and https://www.hopkinsmedicine.org/news/media/releases/study_suggests_medical_errors_now_third_leading_cause_of_death_in_the_us)

Minimum rates thus far attained vary by country and by testing system/numbers but typically 100-1000 /day is observed in many places worldwide, sometimes more or less depending on the region or infection extent. Once the virus is embedded in the community, second waves and second recoveries occur even after long 50-100-day intervals of low rates (Japan, S Korea, Hawaii, Australia, USA…).

The absolute magnitude of an acceptable non-zero risk will therefore vary hugely from society-to-society, also depending on cultures, attitudes, perceptions and politics. In many places, the attainable or even or desired minimum is likely a few, a few hundred or a few thousand cases a day, depending if those values of infection rates look manageable from the societal point of view (e.g. it should be low enough to let people go on with their lives, and tourists visit and people travel if that is what society and individuals want). *The key here is acceptance of risk*, plus testing and treatments…but not lockdowns and edicts.

The rational key here is actually the "old lesson" of learning to live with an accepted risk.

3.2. How High Can the Risk of Getting Infected Go?

We have followed all the infection data trends for many countries with good reporting worldwide (e.g., Spain, France, Italy, UK, USA, Brazil, Mexico, Spain, …) and here is the full story of the trends that have so far emerged.

3.2.1. The Initial Onset Has a Universal Exponential Form.

In our earlier work (Duffey and Zio, 2020a, 2020b) [5, 9], we showed that the first infection "wave" was a simple exponential growth of the rate of infection R, with characteristic e-folding incubation timescale, G, so: $R = R_0 \exp Gd$, where, d, is the number of days since exceeding a measurable initial threshold, R_0 (which we took as 100 cases/day) at the outbreak.

The onset has a characteristic timescale governed by incubation and rapid transmission, and the initial infection rate exponential increases are all similar in timescale ($G \sim 0.14$), independent of where in the world (e-folding average characteristic incubation time of 5.5 days).

When there is a first peak, R_{M1} which nearly occurs at an identical time point during the infection evolution from the onset, differences in peak magnitude, e.g., for Italy, UK, Turkey and

New York City, depend on timing of countermeasures and medical preparedness (deaths mainly hit older folks, are due to pre-existing propensity and largely unreported) and all take about 30 days to reach peak (Figure 2). Humans incubate and recover nearly the same everywhere, and the total number of infections (the area under the curves) is about 200,000 to 500,000 by the achieved minimum, R_m, at about 130 days averaging about 2000 to 4000 per day.

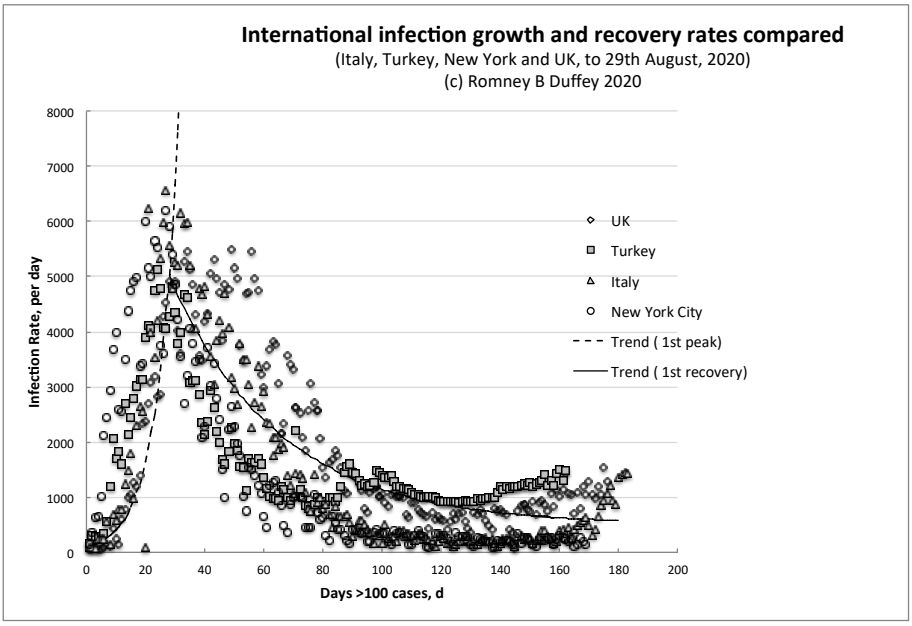

Figure 2. The typical peak and recovery trend during the COVID-19 pandemic.

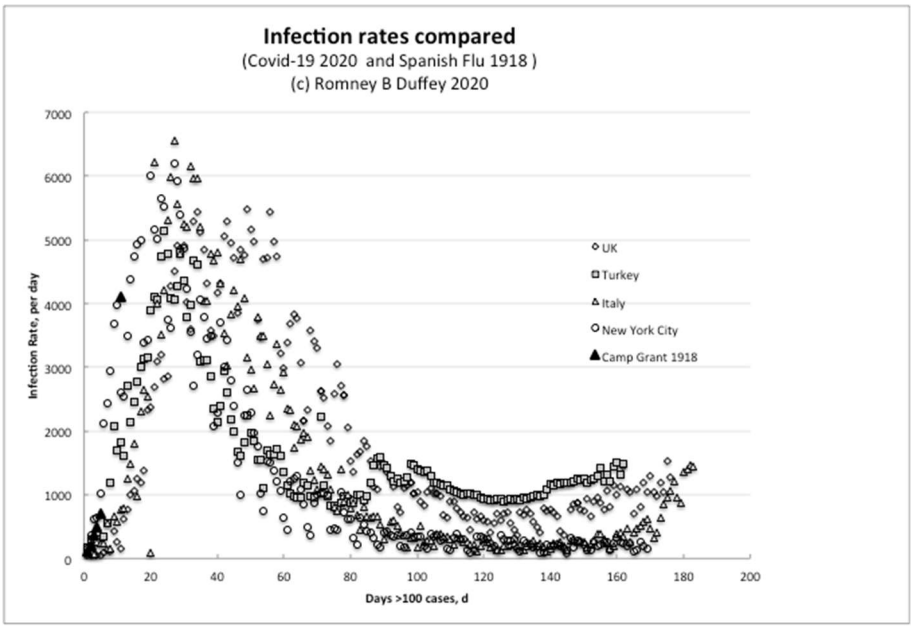

Figure 3. The growth rate in 1918 Spanish Flu and 2020 COVID-19.

The initial peak only occurs when essentially uninhibited growth is almost precisely balanced by restrictions due to the usual countermeasures, such as some form of "social distancing" and enhanced personal and public hygiene. The rise and, then, the learning decrease are clearly the same in very disparate regions/countries/cultures, but initially must be relatively localized in order to have similar rates in locations with such different populations, geographies and customs.

The double exponential pattern we found (Duffey and Zio, 2020b, 2020c) [9, 10] showed that the first infection "wave" was a simple exponential decay of the rate of infection, R, from the first peak, R_1 at day, d_{M1}, with characteristic e-folding recovery timescale, $G - k$, where $k > G$, reflecting the societal learning effect and risk management, hence: $R = R_0\, exp\,(G - k)\,(d - d_{M1})$. This is, indeed, the basic underlying universal infection rate pattern (how else to explain identical behaviors for Italy, UK, Turkey, New York City?) and it is indeed a growth vs. learning (G vs. k) balance, so zero is not possible - unless the virus runs out of hosts as immunity spreads or weakens/mutates/embeds.

Other countries or regions (especially with lower populations) had no real peak but continuing low rates, and some countries or regions do not show reaching a peak at all, their infection rate being typically a few hundred a day maximum, as the numbers are limited and hence a (G vs. k) balance establishes, which is dynamic and depends on many factors.

But there is a key "old lesson" from an earlier precedent, and importantly, the initial infection growth rate is the same as it was 100 years ago. This is shown by the limited data (solid triangles) quoted by (Barry, 2005, p 214) [1] for 36,000 soldiers at US Army Camp Grant, Illinois, in September 1918 at the very beginning of the H1N1 influenza epidemic (Figure 4).

More complete data is available for another USA Army Camp Devens, which had 45,000 soldiers and many infections at the start of the 1918 pandemic (Woolley, 1919) [11]. The daily infection rate at the Camp was recorded and tabulated by this careful military Inspector using their hospital admissions. The trend follows almost exactly the same universal curve but now with a magnitude and rate as South Korea at the beginning of the COVID-19 epidemic in 2020. That the curves, rates and characteristic timescales are essentially identical is a completely new observation that spans a century of experience and is clearly not coincidental. What the US Army camp in 1918 and S Korea in 2020 have in common is very restrictive ("island") social surroundings, homogenous social behavior with cultural uniformity, and/or imposed quarantines and discipline, resulting in rapid containment and reduction in the local region.

That fundamental trend is for highly contagious, new infections with a few thousand cases; what the CDC said about 1918 of course still applies in 2020 [12]:

"With no vaccine to protect against influenza infection and no antibiotics to treat secondary bacterial infections that can be associated with influenza infections, control efforts worldwide were limited to non-pharmaceutical interventions such as isolation, quarantine, good personal hygiene, use of disinfectants, and limitations of public gatherings, which were applied unevenly" (Source: https://www.cdc.gov/flu/pandemic-resources/1918-pandemic-h1n1.html).

These simple measures worked. There is a fundamental trend for highly contagious new infections and the infection risk shape is "universal" -so why does it peak at a few thousand per day?

The key is that the peak rates reached/reported vary depending on society/country but the characteristic timescales are unaltered.

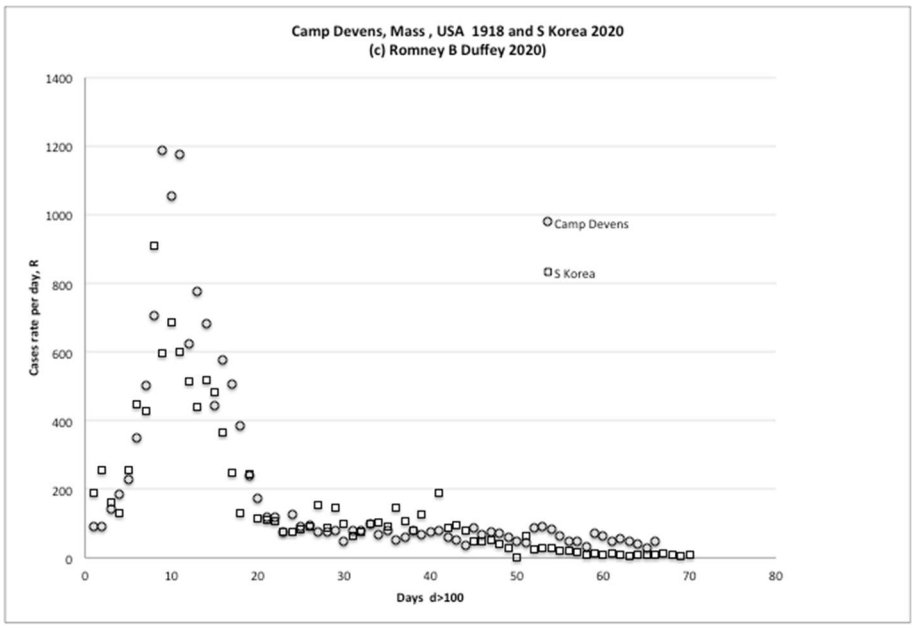

Figure 4. The rates for 1918 Army Camp and 2020 S Korea.

3.2.2. Second or More "Waves" Occur as Virus Is Embedded and Spreads in Society.

Second "waves", peaks or "spikes" have now occurred worldwide (including EU), all about 100 days or so after the first peak (including in "mask cultures" of Japan and S Korea, and even island states New Zealand, Hawaii and Australia). There are some interesting characteristics to consider.

The second wave characteristic rate of infection growth, G, is almost the same as for the first peak, but now mainly hitting younger folks as socially interacting crowds (in bars), parties (in discos), vacations (in touristic areas) and student gatherings (in schools and colleges) occur. The phenomenon varies regionally depending on who goes where (see vacationers causing spikes and second waves in Croatia, Florida, Spain…). The individual incubation timescale is unaltered but it is clear that crowds and travelers at the beginning also helped spread the infection; but once it is embedded/active in the larger community it has literally a "life" of its own, following people gatherings around.

The magnitude of the new 'second' peak rates will depend on the baseline values of prior minimum and maximum possible. The most pronounced has been in the USA and we now know the second US wave intersects the trajectories for India and Brazil, which continued to increase without a first peak due to overwhelming wider initial community spread.

These two large nations showed no initial peak – Brazil and India exhibited a slower initial rise but no "bending" of the curve. This is clearly due to the longer third characteristic time scale - the one for community spreading (diffusion and embedding) in large populations but scattered over larger areas. These slowly rising infection rates with $G = 0.05$ (for a characteristic societal transmission e-folding timescale now of 20 days) are shown in Figure 5, along with the US curves for the first peak and delayed second wave, which brings the US into a similar trajectory of 'slow spread". These Nations have several things in common- populations in excess of 200 million, land areas of over 1 million square miles, dense populations near the coasts, and thousands of miles of interior space. But, as for the first peak, there is an "old" lesson and earlier precedent for the second wave. Importantly, the second infection onset and growth rate has nearly the same resurgence pattern as

occurred 100 years ago for HINI. This is shown by the weekly infection data (circles) for 560,000 people in the major city of Copenhagen, Denmark for July, 2018 to January, 2019 during the H1N1 influenza epidemic reported and plotted[2] by Andreasen et al (2008) [13]. By comparison to the 2020 COVID-19 infection data for Japan 100 years later[3], the behavior, periodicity and timing of the infection trajectory is essentially unaltered and independent of location - because the common factor is that the host and transmission agent is the Human.

Therefore, we can we reject this argument as false: "Waves evoke predictability, however, and COVID-19 has been hard to predict. Despite the valuable lessons drawn from past influenza outbreaks, how pandemic influenza struck in 1918 is not a template for what will happen with COVID-19 in the coming months." (Webel and Freeman, 2020) [14].

The second wave peak magnitude depends on apparently many random or circumstantial factors, according to the degree of hidden community spread: so, it is unreasonable to expect a "complete" template but just a very similar one. As for the USA in 2020, the second peak is often about twice or more times the first initial peak rate and begins after the community spread characteristic timescale of about 80 to 100 days – just like in 1918. To intercompare widely differing cultures and societies, we can normalize the rate data to the first peak (Peak 1), and this calculation is shown for the disparate regions of Copenhagen in 1918, and Japan and Idaho in 2020 (Figure 6). The remarkable similarity of the trends proves the characteristic timescales of growth, recovery and community spreading dominate in many disparate places. Of course, there are exceptions, for those regions without a first peak due to *(a)* minimal initial infections or *(b)* rapid massive community spread (Figure 7). Why are the trends and shapes so similar? Why is the second peak about twice the first? And what do they have in common? It must be a universal viral characteristic property.

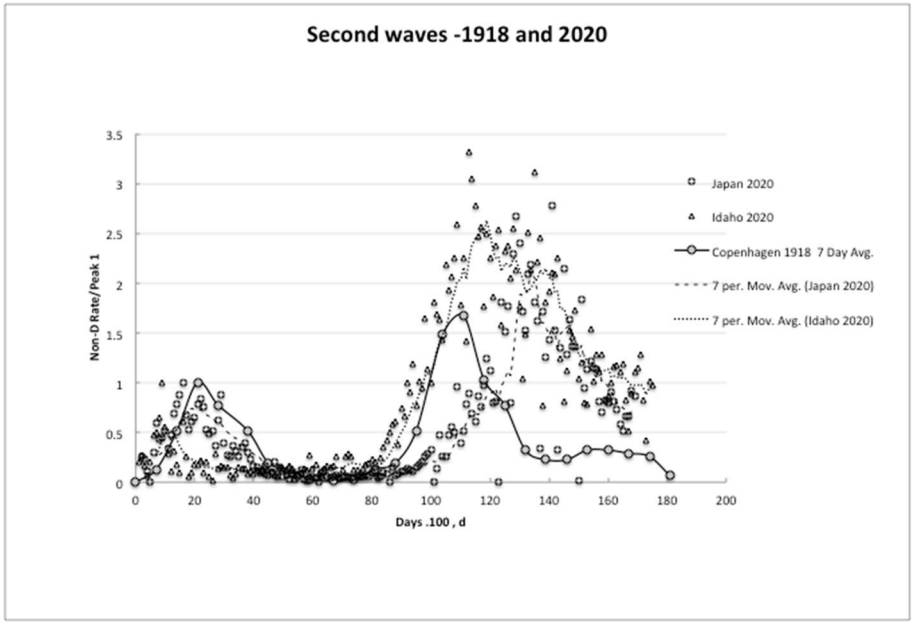

Figure 5. The similar second waves for 1918 infection and 2020 pandemic.

[2] The data were hand-transcribed from enlargements of their Figure 2 and converting the weekly rate per 10,000 to a simple daily average with an accuracy of about 5%, sufficient for showing these important major trends.
[3] Also showing the 7-day average to be consistent with weekly-reported Copenhagen data.

As is already known (Covello and Hyer, 2020) [3]: *"Effective travel restrictions and quarantine alone may not stop disease spread"*.

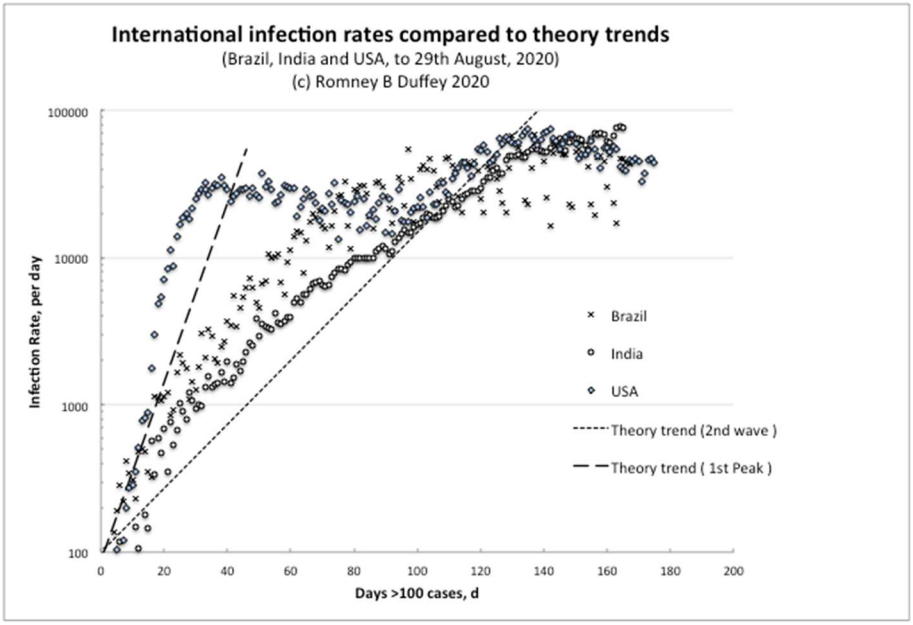

Figure 6. Extended community spread in large countries.

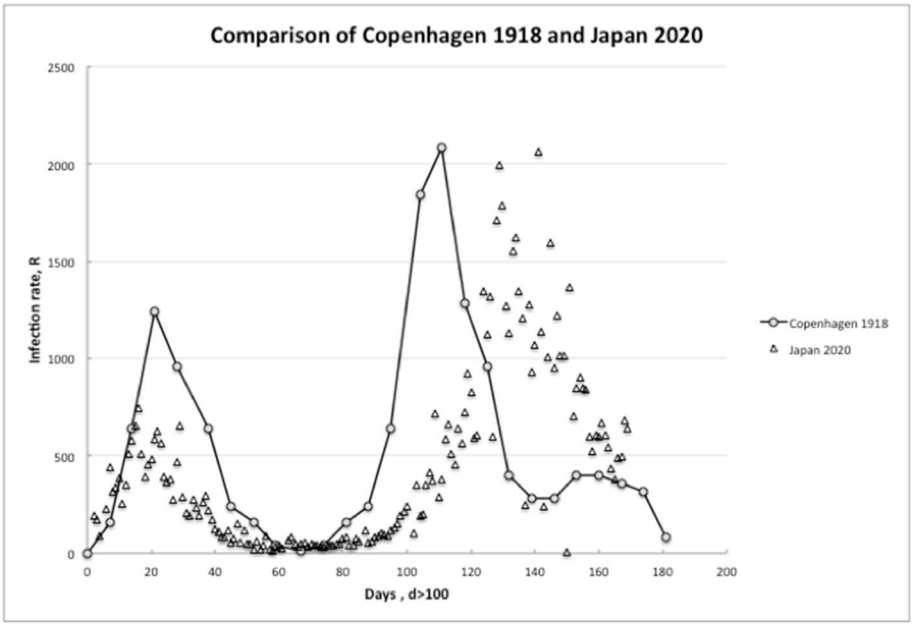

Figure 7. The universal trend in two cultures and continents.

3.2.3. No Relation of Infections to Airborne Pollution.

It is known that air pollution is a "leading risk factor" for disease worldwide (Cohen et al, 2015) [15]. There have been suggestions that COVID deaths among those most vulnerable over 65 are statistically related to PM2.5-micron air pollution levels in cities (Xiao et al, 2020) [16], with 1 μg/m^3 in PM2.5 associated with a 15% increase in the COVID-19 death rate at 95% confidence. If pollution indeed pre-weakens the immune system, lung function and asthmatics, then possibly it does also make infection numbers worse, but this is in superposition to pre-existing propensity and it will, thus,

be societally dependent and may only show up in death rate data, which is still dominated by older folks (as in Italy and USA), whereas second wave cases are mostly for the younger age group of 18-35 or so, which fill up the social gatherings.

But there was a major and completely "natural" experiment in California, with the onset of massive and record air pollution (stated as "unhealthy for everyone") due to multiple wild fires, while the pandemic was ongoing. A large step function increase in air particulates occurred and, so, we have plotted the available air quality PM2.5 concentration and COVID-19 overall infection rate data[4] for the hard-hit Bay and Silicon Valley Areas (Santa Clara County with 2 million population, and as of September 30 21000 COVID-19 cases plus extensive testing). Figure 8 shows the 7-day average lines- and uses a log scale to encompass the huge increase in PM2.5 after day 79. As can be seen, while the average "normal" prior local PM2.5 (and other indices) was a low 7.68 μg/m^3 at day 78, August 17, it increased dramatically with four days peaking over 100 μg/m^3 and averaging 31.7 μg/m^3 for more than 40 days (sufficient for COVID-19 infection, incubation and detection) and even required "stay-in" warnings that are equivalent parallels to personal quarantine or "stay-at-home" edicts.

The result is self-evident: in this inadvertent experiment, there is no correlation or relation between PM2.5 air pollution increase and any overall COVID-19 infection rate increase or greater community viral spread (a linear plot shows the same). There was also no statistically significant change in the (small) daily death rates in these intervals prior to after the sudden pollution increase.

So simple models or theories that attempt or pretend to correlate pollution with infections etc. will indeed fail to predict this trend.

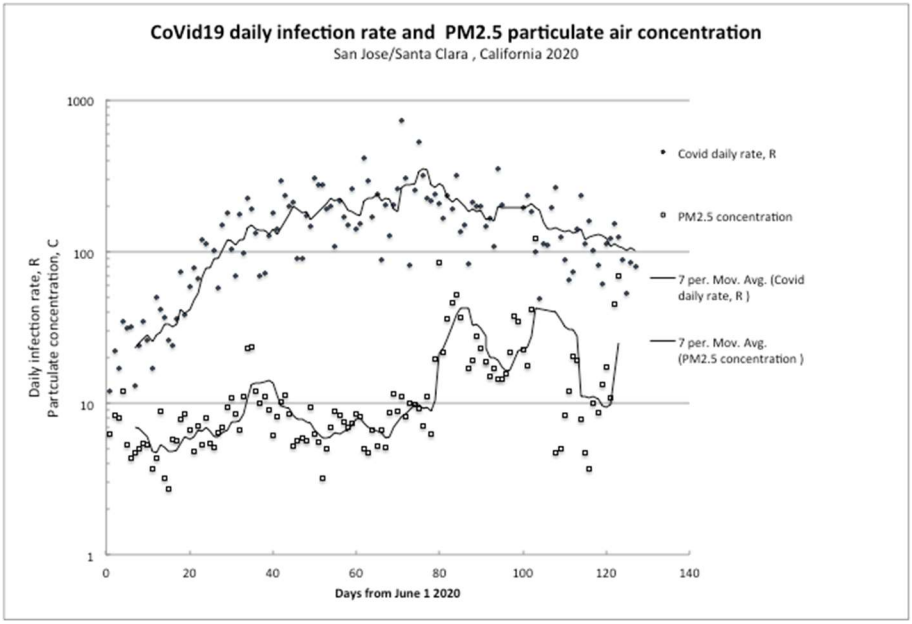

Figure 8. The lack of impact of severe respirable pollution on viral infection rates.

[4] Official data sources: www.epa.gov/outdoor-air-quality-data/pm25-continuous-monitor-comparability-assessments, ID 60850006; and data.ca.gov/dataset/covid-19-cases/resource Santa Clara.

The increased pollution timeframe well exceeds the infection incubation and growth times, and so far, there is no statistically significant increase in the infection rate – indeed, the local region daily COVID-19 infection rates have actually decreased.

The key is that we may expect there to be influences from factors that we do not expect and cannot prove, but that may affect our risk. Any conclusion on that must be confirmed by incontrovertible proof.

4. How Many Infected and How Long Will or Could Infection Last?

Now looking for example at the USA, the number of cases throughout the country is not related/relatable to region, climate, city, pollution or countermeasures. The number of regional (State) infections per population (i.e., infection opportunity) is nearly the same everywhere, and is simply proportional to the regional (State) population (see below). So, at least in the USA, COVID-19 does not worry about who, where or when…and most cases of the second wave, as expected, are among mostly younger 20-39 partygoers etc. while deaths continue to be mainly among the vulnerable subjects.

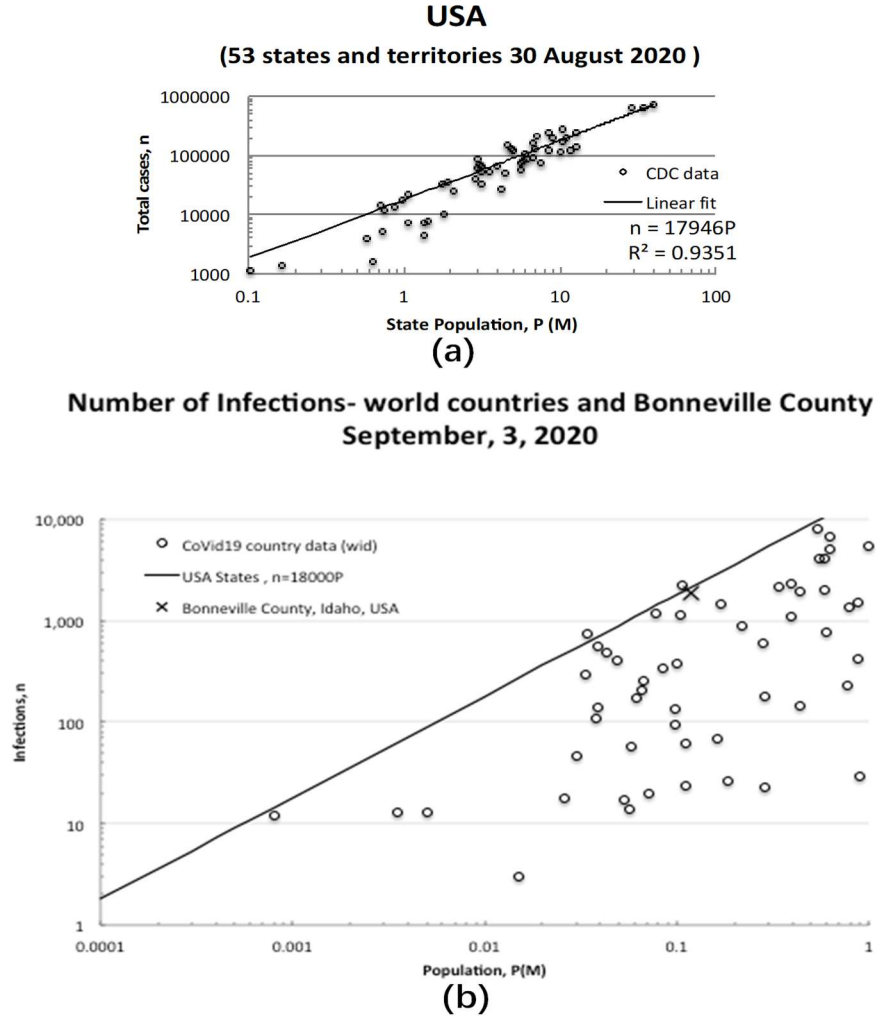

Figure 9. (a) The infection fraction for the USA; (b) The infection fraction for smaller regions.

As to the number of deaths per population, it is the same story, as that is the same in Italy as in all USA, as in Sweden and New York City, and not related/relatable to region, climate, city, pollution or countermeasures, i.e., about 500 per million.

So, again, there are no dependencies of infections or deaths on race, religion, location, masks, food, culture, travel, or lockdowns. The virus does not worry about where or when but who, so follows killing the usual age distribution of deaths (as expected mostly targeting that part of the population with pre-conditions and age >65).

Now, the data show that in open societies and democracies the number infected, n, is proportional to the population, P. This is explicitly shown by the USA data for over 50 states and regions, and has n=0.018P, or 18000 per million[5], or nearly 2%, for a range $100{,}000 < P < 40{,}000{,}000$. The highly conservative linear infection hypothesis (LHP) says that this relationship $n \sim 0.018$ P holds as an upper bound even for smaller populations of < 1 million, e.g., places like Brunei, Bhutan, island states like the Caymans, Curacao and Fiji, plus Finland and Guam, Monaco and New Caledonia, and this is confirmed as shown in the Figures 9a and 9b, using data from 22 January to 3 September, 2020.

Even more locally, Bonneville County is a subset of Idaho State, and has 119,000 people and 1884 cases as of 3 September, 2020, and is shown fitting close to the line on the graph for whole states and countries. In other words, all locations have their "fair share" of infections over the entire local population, and so the risk is pretty much the same no matter where you are, who you are or where you live.

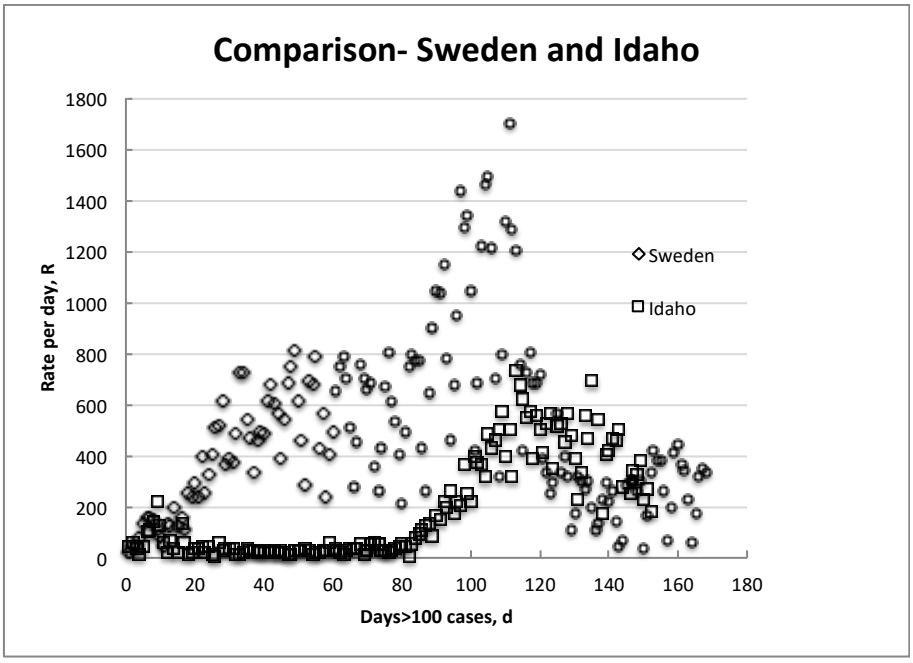

Figure 10. Infection rates independent of countermeasures in different regions.

Since the infected fraction does not depend on where you live or the countermeasures deployed, infections just spread at a certain minimum rate if enough of the human hosts are available. We can also see this in comparing Idaho (a western US State with 1.7M people and "lockdown"

[5] This infection fraction is as of 7 September, 2020, and will obviously slowly rise with more cases, but the principle of uniform or "equal infection opportunity" still holds.

countermeasures) with Sweden (a European state with 10M people), with similar climate but completely dissimilar and few countermeasures (Figure 10). Incidentally, many Swedes originally migrated to Idaho, so much so there is even a Little Sweden, and many Nordic names. The fractions and rates do not vary by more than a factor of two or three, the spread in the data are similar, but Idaho has a higher rate per capita by a factor of 5 with a lower population. How can these similarities be explained as it is not culture, geography or countermeasures?

The key answer again is that the infection risk does not matter where you live or on the countermeasure extent- viruses spread the same any way they can.

5. Gatherings, Crowds and Community Transmission: What Should We Do?

This is a controversial area, as limits on church gatherings, crowds and restaurant occupancy are hurtful to those who own or operate such activities, and to the wanted social life in general. The guidance is clear- just keep the numbers low.

As stated by the US CDC (2019) [17]:

"At a minimal-to-moderate level of community transmission, it is recommended to:

- Cancel community-wide mass gatherings (for example, >250 people); the cut off threshold is at the discretion of community leadership based on the current circumstances the community is facing and the nature of the event (https://www.cdc.gov/coronavirus/2019-ncov/downloads/community-mitigation-strategy.pdf) or move to smaller groupings.
- Cancel gatherings of more than 10 people for organizations that serve higher-risk populations.

At a substantial level of community transmission, it is recommended to cancel mass gatherings of any size."

Data is key. We searched the literature for supporting evidence, but found none. Our belief is that the guidance may be helpful in the very initial infection phase, but as can be seen from the world happening, the initial timescale rate (G-value) is completely country and countermeasure independent. Later, when the virus is embedded in the community, the spread is now slow and pervasive with or without gatherings – which occur anyway (see below).

Although there are no data found to support the (arbitrary) limits on transient gatherings, it is currently stated by medical folks (including the CDC) or on the infection rates adopted to issue mandates based on risk "levels" or 'tiers' (see e.g., "Blueprint for a safer economy" www.gov.ca.gov/2020/08/28/governor-newsom-unveils-blueprint-for-a-safer-economy-a-statewide-stringent-and-slow-plan-for-living-with-covid-19). We found a recent review (Nunan and Brassey, 2020) [18], which states:

"A survey study of mass gatherings in the United States reporting on 18 respiratory-disease outbreaks between 2009-2016 found that more than half (61%) occurred at agricultural fairs in 2012 via zoonotic transmission. Sporting, professional conferences and religious events contributed only one reported outbreak each. The most common pathogen (n=11) was influenza A H3NSv from agricultural events (probable swine flu exposure), with various other influenza A pathogens forming the remaining. None of the reported outbreaks involved single-day mass gatherings, which reflected findings from the previous systematic review The best-available evidence suggests multiple-day events with crowded communal accommodations are most associated with increased risk."

But there is real data acquired incidentally that suggest what the limits on gatherings or expected case numbers should be. During the COVID-19 pandemic, the Sturgis Motorbike Rally was a gathering of some 460,000 motorbike enthusiasts over 10 days in South Dakota in 7-16 August 2020. The attendees did so voluntarily, travelling in to overwhelm a town of just 7000, and by choice did

not rigidly adhere to mask and social distancing guidelines even knowing that crowd infection risk existed. Bikers are traditionally risk takers so this is a wonderful natural and practical example of both personal decision-making and risk-taking. Long enough for both infection and incubation, it has been reported as subsequently causing 260 traceable COVID-19 infections (https://www.npr.org/sections/coronavirus-live-updates/2020/09/02/908874086/states-report-coronavirus-cases-linked-to-sturgis-s-d-motorcycle-rally).

Assuming only about 100,000 attendees on any given day to give an upper bound, the individual infection probability is 0.0026 or a one in 390 personal chance, with the single reported death being with a pre-existing condition. This personal dynamic "transient gathering infection risk" is lower than the national average overall fulltime resident risk of 0.018 per person by at least a factor of ten, so does not count as a "super spreader" dynamic or short-term event.

Taking extreme values of the crowd being the full 500,000 for every day, the risk is even 5 times lower, and it is less risk to go to such an event. Is it possible that lockdowns and lock-ins actually increase risk by assisting domestic indoor transmissions? The results and data show that the limits on *transient*_gatherings and crowds (for sports, churches, malls etc.), proposed and enforced by mandates and guidance of 50% or 25% occupancy are low by as much a factor of 10 for achieving a comparative personal and societal risk and/or possibly avoiding one infection[6].

As we were writing this paper, a recent "study" has argued that 266,000 or so cases were caused by or related to the Sturgis gathering (e.g., see www.washingtonpost.com/health/2020/09/08/worst-case-scenerios-sturgis-rally-may-be-linked-266000-coronavirus-cases-study-says). It is based on tracking the cell phone call origins of the 460,000, attendees and ascribing essentially any increases of COVID numbers where they came from as being due to the Rally.

If 266,000 sounds a little large - in fact unbelievably large - you would be correct. For a detailed refutation of the method and numbers see https://slate.com/technology/2020/09/sturgis-rally-covid19-explosion-paper.html and also https://rapidcityjournal.com/news/local/state-and-regional/noem-study-connecting-250-000-covid-19-cases-to-sturgis-rally-is-grossly-misleading/articlel and "The Sturgis Statistical Misfire", Wall Street Journal, September 10,2020, pA18). This gigantic overestimate is a perfect example of how inexact modeling and wrong assumptions can and do provide wrong answers- and as a result drastically overstate the risk, with all political and social consequences that now arise.

It is not physically possible for that number to occur, and all we need to do is to look at the data, since we have the numbers of infections reported for all the 176 neighborhoods in New York City[7] (from February and being the highest infected city in the USA) to add to the smaller country data (see graph in Figure 11). As before, the solid line is the risk for 1 in 60 people (or 0.018 per person), for all the USA States showing infections are *always* just a proportional fraction of the total exposed population that are living together in any region, state or suburban development.

The numbers exposed and gathering in an urban city neighborhood (40,000 to 900,000) therefore now encompasses the Sturgis Rally at 460,000. An impossible worst case considers Sturgis as a place where 460,000 people gathered as a neighborhood full-time from February, so by September we

[6] Extrapolating, for comparable personal risk, a more common analogy is a sports crowd of 50,000 attendees lasting less than a day, and by adopting "normal" social distancing and hygiene precautions we may expect of the order of some 100 additional cases and a transient crowd of 500 will likely have none.

[7] Source: www1.nyc.gov/site/doh/covid/covid-19-data.page.

cannot possibly have more than about $460{,}000 \times 0.018$ cases= 8280 cases –let alone be responsible for 260,000- and indeed the reported traceable data says many less.

The reason is simply because Sturgis only lasted 10 days, not 200 days. On a purely random infection basis proportional to the time of (personal risk) exposure, we would get $8282 \times 10/200 = 414$ cases, almost exactly the 260 cases that NPR, South Dakota and others reported (not phone call contacts!). That puts the cell phone study in context - a lot of i-phone calls but not many infections- and not only further confirms the random infection hypothesis but also shows that the key variable is the time or extent of personal risk exposure. This is an obvious result- our risk simply depends on our exposure, which is why full-time medical personnel need to wear and use a lot of protective gear and ailing senior citizens should be very cautious.

The key is that the 'mandate' or guidance numbers are indeed simply overly precautionary but are not really meaningful as to the real personal risk.

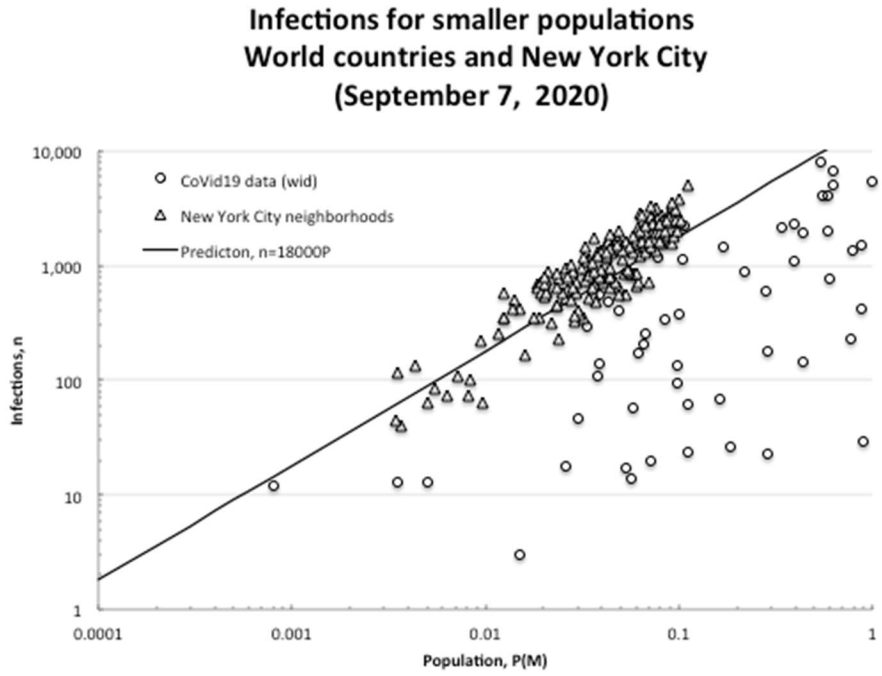

Figure 11. The infection fraction at neighborhood and mass gathering scale.

6. Risk Levels and Tiers: Comparative Risks and Real Risks

Surely, one way to compare and balance risks is to try to set societally acceptable standards for risks, but there is a problem (Duffey and Saull, 2009) [19]:

"As far as we are aware, there is no common standard for relative or acceptable risk, except what is actually practiced until something fails or goes wrong. The acceptable risk for any outcome defined or adopted on a society-wide basis is that used today by the many and varied governmental bodies responsible for licensing the construction and operation of individual technologies. The national bodies governing these incredibly disparate numerical risks such as electricity reliability commissions, building inspectorates and nuclear safety regulators, not only frame the laws and regulations, but are themselves both the judges and the juries for defining their own "acceptable" risks."

For medical systems, there is also no common standard but often recommendations or "guidance" on best practices, from which (national, regional or local) government officials often determine and pronounce what is termed "mandates" based on so-called risk levels or tiers. Their concern is largely not to overload the emergency medical system, and for that they apparently consider (daily) emergency and ICU bed count availability, although emergency temporary hospital capacity was fully supplied (and not used) in New York City nor Milan. As an example, the most recent California plan ("Blueprint for a safer economy" www.gov.ca.gov/2020/08/28/governor-newsom-unveils-blueprint-for-a-safer-economy-a-statewide-stringent-and-slow-plan-for-living-with-covid-19/) has four Tiers of risk levels using specific ranges of infection rates averaged over 7 days[8], and distinguish between even sectors in society and business differentiate depending on measures primarily intended to limit person-to-person contact. The Blueprint states:

"Criteria used to determine low/medium/high risk sectors:

- Ability to accommodate face always covering wearing (e.g., eating and drinking would require removal of face covering).
- Ability to physically distance between individuals from different households.
- Ability to limit the number of people per square foot.
- Ability to limit duration of exposure.
- Ability to limit amount of mixing of people from differing households and communities.
- Ability to limit number of physical interactions of visitors/patrons.
- Ability to optimize ventilation (e.g., indoor vs. outdoor, air exchange and filtration).
- Ability to limit activities that are known to cause increased spread (e.g., singing, shouting, heavy breathing; loud environs will cause people to raise voice)".

This is clearly over prescriptive and well beyond any measurable capability for compliance. These are all nominally designed to limit contact, but as shown by Sweden there is no effect beyond taking simple common-sense precautions.

The California Blueprint matrix with its "Tier Framework" is an illustration below: where the infection rates per 100,000 between tiers come from is not revealed or known and the risk levels of activities selected (gyms, bars, …) are not based on any data (read the full reports/guidance at the links at https://www.cdph.ca.gov/Programs/CID/DCDC/Pages/COVID-19/COVID19CountyMonitoringOverview.aspx).

Note that the Tier levels are by separate California county-by-county with populations varying from 10M to 1000, so the absolute infection numbers can be different by a factor of 10000 just for 1 case/day, while it is just a factor of 7 in rate to go from "Minimal" to "Widespread" tiers. To show the global disparity, while Los Angeles requires a 7-day average case count 700 per day to be widespread, "Tier 4", in New Zealand a 7-day average count of only 8 per day led to complete "lockdown". Being backwards thinking, equivalently, only the 22 counties in California with the smallest populations would be so affected, whereas a typical spike, fluctuation, resurgence or second wave onset can give a few hundred to a thousand per day in a city (e.g., the Los Angeles rate of 1400 per day plus or minus 335 for 17-26 August is a random example).

The key lesson here is that relative risk should be based on the increased probability or chance of the degree of harm or exposure to adverse outcomes.

[8] The 7-day averaging interval is chosen because infection data often show a clear weekly up-and-down periodicity due to testing, reporting and social habits- and the individual incubation timescales is also about 3-6 days.

So, what is the comparative upper bound of death due to COVID19 infection risk?

Choosing a large range of populations, infection numbers and social restrictions, and the most reliable WHO data (as of September 5, 2020), we find surprisingly similar death rates, just like for other infectious diseases (in Table 1):

Table 1. death rates and infectious diseases in different countries.

Country	Population (M)	Cases (M)	Deaths	Deaths per 100,000
USA	328	6.2	188,000	57
Brazil	210	4.1	125,500	59
Italy	60	0.27	35,500	59
Sweden	10	0.85	5,800	58

Since all are about the same and noting Sweden took and still takes few countermeasures, there is really no difference in the killing rate fraction of 50 to 60 per 100,000, so it does not matter who or where you are and what are the countermeasures or treatments; again, it is random and depends largely on age and propensity. Even if doubled, it is also comparable to deaths from all other infectious causes, and worldwide the number of deaths per case varies with the health system, population age, reporting protocols (co-morbidity, etc.).

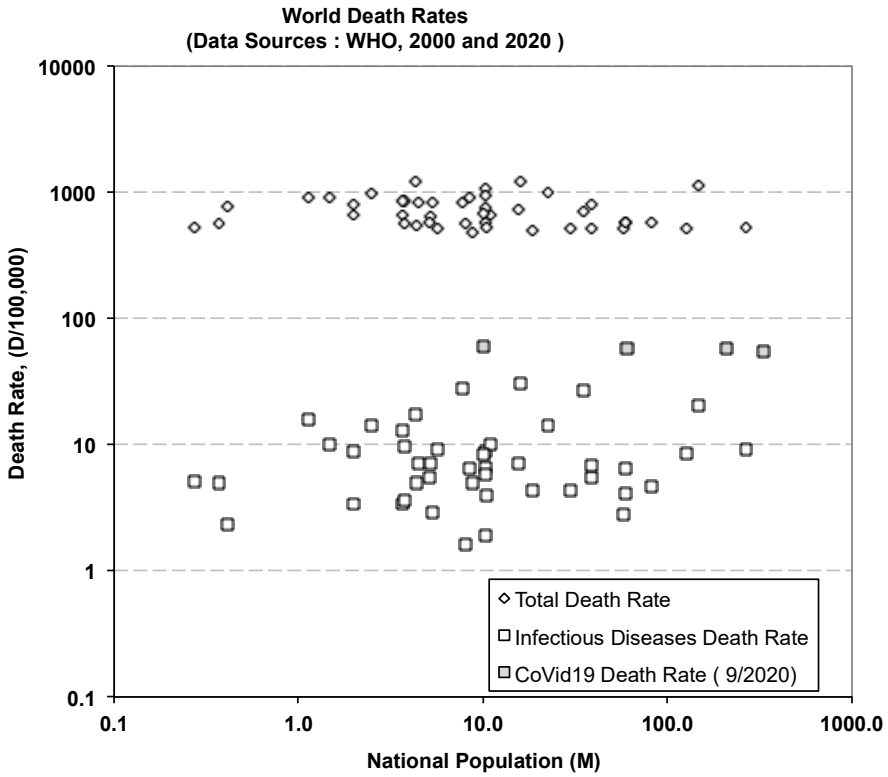

Figure 12. Relative global risk of death from infectious diseases.

The virus does not worry about where or when but who, so follows usual age distribution on most deaths (as expected mostly with pre-conditions and >65), thus supporting the ASTHO remarks that (Covello and Hyer, 2020) [3]:

- Viruses are a threat to all people, regardless of race, ethnicity, or the country one lives in.
- Viruses do not target people from specific populations, ethnicities, or racial backgrounds.
- Viruses do not respect borders and do not discriminate among different types of people.

Globally, there is approximately a 60 per 100,000 chances of death due to COVID-19 compared with global average all-causes ("normal") death rate of about 760 (+ or – 280) per 100,000, so it is a about a one in 10 chance of COVID-19 being the *primary* cause (Figure 12). This risk is comparable to being a victim of medical error or catching a serious bacterial infection while just being in hospital.

7. Discussion

From the data, there emerges a general ten-step infection framework, which is quite simple but also remarkable. These ten steps are shown in the Figure 13.

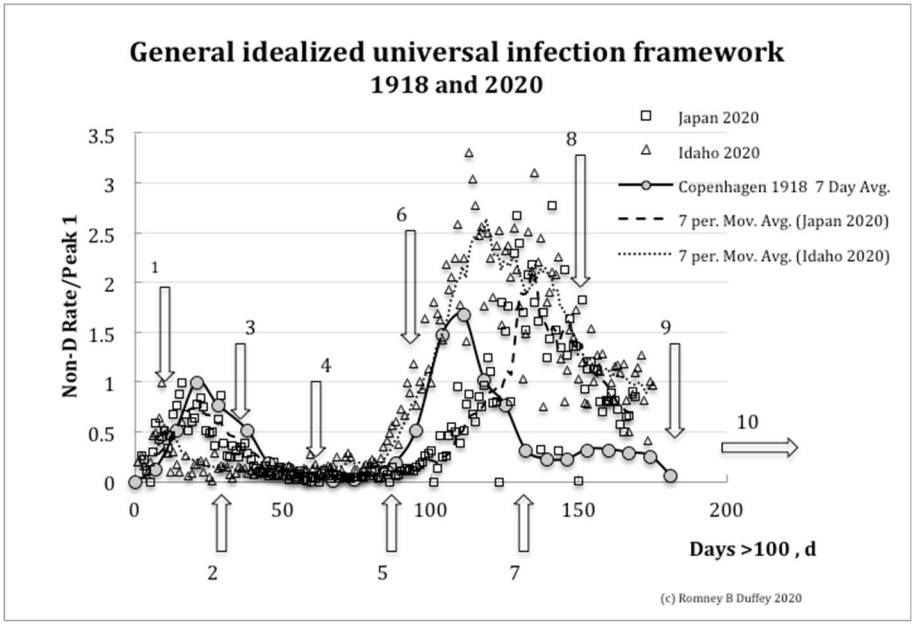

Figure 13. The general framework of infection process.

1. Initial infection exponential rise (original incubation timescale, G), $R_1 = R_0 exp(G)d$.
2. First peak in circa $d_{M1} = 20 - 30$ days, $R_{M1} = R_0 exp(G) d_{M1}$.
3. Decline due to learning timescale, k (countermeasures, hygiene, and social distancing) $R - R_m = (R_{M1} - R_m)\, exp(G - k_1)\,(d - d_{M1}), d > d_{M1}$.
4. Minimum achievable infection rate (or detection threshold), R_m.
5. Second "wave" onset at circa $(d - d_{M1}) > 80 - 100$ days (new community spread timescale) dependent on region and initial infection rates and locations.
6. Second exponential rise nearly same as first (spread due to social activities), $R_2 = R_{m2} + exp(G)(d - d_{M1})$.
7. Second peak C-times first peak at circa 100 plus 30 days, $R_{M2} \sim C R_{M1}$.
8. Decline due to learning plus new community/human host limits, $R - R_m = (R_{M2} - R_m)\, exp(G - k_2)\,(d - d_{M2})$, $d > d_{M2}$.
9. Minimum "acceptable" rate achieved again, R_m.

10. Additional waves possible (annual, seasonal, social…), or, in general, for i-communities with time varying infection spread timescales or scenarios, we simply have the sum, $\sum_i(R_i - R_{mi}) = \sum_i(R_{Mi} - R_{mi})\, exp(G - k_i)\,(d_i - d_{Mi}), d > d_{Mi}$.

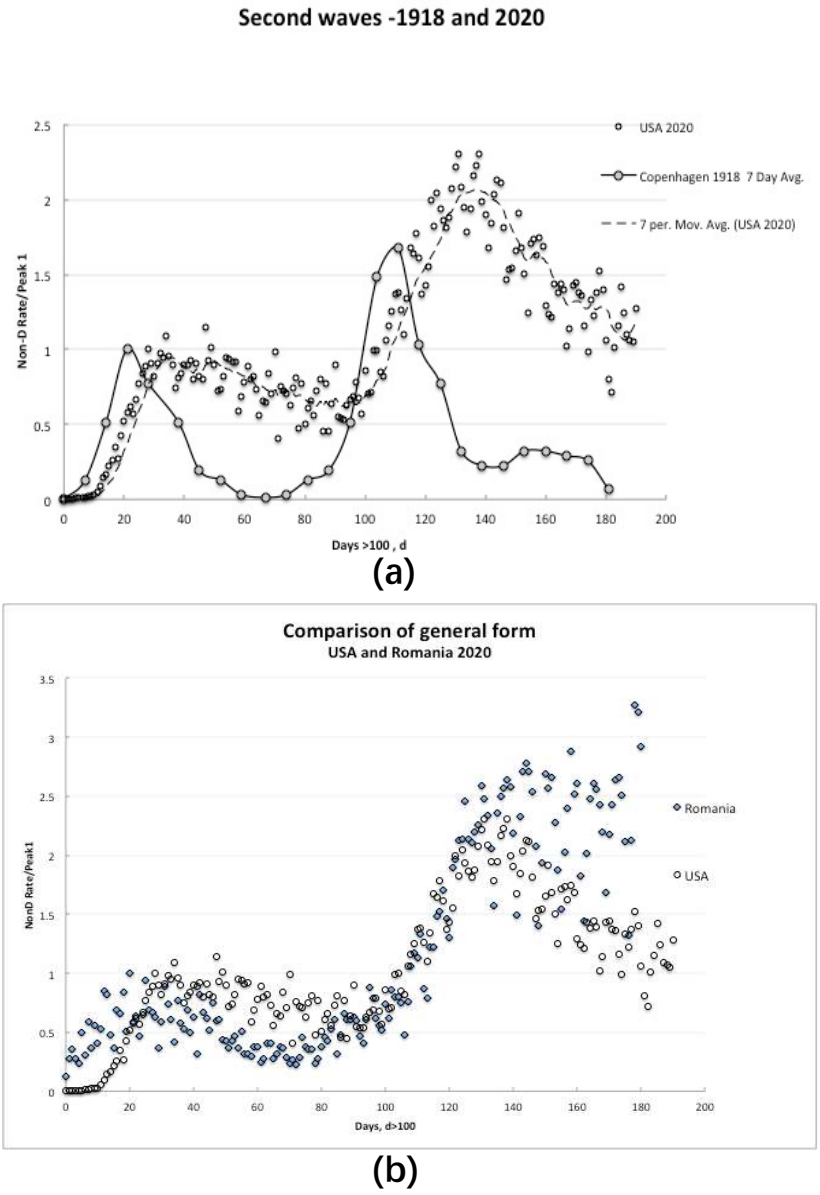

Figure 14. (a) The general scaling from 1918 to 2020, (b) The general trend in two different regions (USA and Romania).

Of course, there are exceptions- being those regions without a discernible first peak because of: (a) very small numbers due to luck: or (b) very large numbers due to rapid societal/community spread (India and Brazil). As an extreme illustration, in the USA, the second wave even overlapped the first due to slower recovery with rapid multiple inter-community spread. Once again normalizing the infection rate to the first peak, in Figure 14a, we have the remarkable comparison between the 1918 Copenhagen (13,900 cases total) and the USA 2020 (660,000 cases as of September 15, or nearly 50 times more), showing the similar rate doubling and characteristic timescales.

The "island nation" of Australia in 2020 (27,000 cases as of September), with extensive border controls, lockdowns and travel restrictions, also has similar trends to Copenhagen in 1918.

At long last we now know what to expect. Thus, generality is further clearly shown in Figure 14b by comparing the trends for Romania (population 19.4 million, peak rate RM1= 523) with the USA (population 320 million, peak rate RM1 = 32,400). Since we have successfully related viral spread trends from such wildly disparate sources and regions and timescales to a common basis, we are able to claim a general framework and understanding for infection risk prediction purposes.

8. Conclusions

For the management of risk, it is necessary to realize and accept rationally that risk is not absolute- it is relative. This is because we are exposed to a multitude of potentially harmful events. As a society we should weigh, rank and decide the intertwined risk balances, and resulting inequalities, considering what we know and what we do not know. From the data, there emerges a general ten-step infection framework, which is quite universal and important to consider for a rational and balanced management of the pandemic risk that we are experiencing. This knowledge should be accompanied by a number of lessons and key observations that have been recalled in this paper.

- We must accept that there is no such thing as zero risk and infections will occur.
- We must learn to live with an accepted risk.
- There are influences from factors that we do not expect and cannot prove, but that may affect our risk. Any conclusion on that must be confirmed by incontrovertible proof.
- 'Mandate' or guidance numbers on risk realizations tend to be overly precautionary and not meaningful as to the real personal risk.
- Relative risk should be based on the increased probability or chance of the degree of harm or exposure to adverse outcomes.

And some more specific with epidemic infections:

- Peak infection rates reached/reported vary depending on society/country but the characteristic timescales are unaltered.
- We must expect more waves superimposed to the initial ones and they all have the same characteristics.
- Infection risk does not care where you live or the countermeasure extent.

In the end, what really matters is what risk we are willing to take as individuals. We do refer to authority for guidance but eventually take our own decisions regarding risk, depending on our risk perceptions and risk-taking attitudes. We recognize that the overall balance guiding our decision should be objective and quantified technically and numerically, but subjective and qualitative risk perceptions often dominate.

Conflicts of Interest: The authors declare no conflict of interest.

Appendix A

A.1. The Probability of Infection: Positive and Negative Data

We can now make predictions for the probability of infection based on data. In any societal group, testing sample, or gathering, assume that infections are indeed random and the virus is simply opportunistic so all people have an equal chance of being infected. After any time or interval, we observe, n, positive tests or count of infections out of some total, N, and count, m, negative or non-infections out of some unknown total, M, in a total possible population numbering, $N+M$. The total infected fraction is $N/(N+M) \sim N/M = p(N)$, and the probability of observing, n, random infections/ positive tests in the sample is given by the standard hypergeometric formula:

$$p(n) = \frac{\binom{N}{n}\binom{M}{m}}{\binom{M+N}{m+n}} \qquad (A.1)$$

For a relatively small number of infections (as is presently the case for COVID-19), $M >> N$, $m >> > n$, so this becomes the usual binomial and then Poisson approximation,

$$p(n) \to \binom{n+m}{n}\left(\frac{N}{M+N}\right)^n\left(1-\frac{N}{M+N}\right)^m \approx \frac{1}{n!}\{p(N)^n\}\{1-p(N)\}^m \approx \frac{1}{n!}\{p(N)^n\}e^{-mp(N)} \qquad (A.2)$$

Now, from the analysis of the US data with extensive testing and reporting, we know the actual infected fraction, $p(N) = \frac{N}{N+M} = K$, is nearly constant over an exposed range, $100{,}000 < (N+M) < 33{,}000{,}000$ and for the USA presently $K \sim 0.018$ per person. Hence, the chance of observing any number of infections, n, is

$$p(n) \approx \frac{1}{n!}\{K^n\}e^{-mK} \qquad (A.3)$$

The probability of just one infection is

$$p(n=1) = Ke^{-1} \qquad (A.4)$$

The probability of more than one infection, $n > 1$, is the complement, $p(n > 1) = 1 - Ke^{-1}$.

Now, the maximum possible number of random infections is when $p(N) = K \approx 1$, although this strictly violates our initial assumptions just to set the order-of-magnitude, $p(n > 1\,max) = 1 - e^{-1} = 0.63$. Surprisingly the infected fraction reported for the 1918 influenza epidemic are Brazil 37%, Buenos Aires 55%, Cape Town 38%, and Japan 33% (Barry, 2005 p 363) [1]. These numbers yield a global average fraction of 40±20%, somewhat lower but broadly consistent with the maximum theoretical limit, suggesting the 1918 virus statistically simply ran out of hosts.

A.2. California Risk Tier Matrix (Source California Blueprint)

	Higher Risk → Lower Risk of Community Disease Transmission			
	Widespread Tier 1	Substantial Tier 2	Moderate Tier 3	Minimal Tier 4
Measures*				
New cases**/100,000 population per day (7 day average; 7 day lag)	>7	4-7	1-3.9	<1
Testing % Positivity (7 day average; 7 day lag)	> 8%	5-8%	2-4.9%	<2%

Figure 15. Illustration of California Risk Tier matrix (source California Blueprint).

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