We need a way to separate hysteria, propaganda and conspiracy theory from reality so we can gain a pragmatic perspective on the current COVID-19 pandemic caused by the SARS-CoV-2 virus...

This paper was originally written in early May 2020 and has been updated many times over a period of many months. The basics have changed very little while the accumulated statistics have continued to climb as you would expect. Thus the referenced numbers are associated with the date they were associated with.

There were 572 thousand reported COVID-19 deaths in the US as of April 25, 2021 – a per capita death rate of 0.176%. This number has issues, as I will explain later, but I will use it since it is the generally accepted published number.

Although there is no definitive official definition for the word pandemic, the meaning is generally associated with a geographically wide spread disease – typically worldwide – that sickens and kills a lot more people than you would expect from other commonly known diseases.

This paper presents a lot of information. Although I have a background in emergency medicine, microbiology, virology and infectious diseases, and I strive for demonstrable accuracy, you should be skeptical. As one teacher told me: Don’t believe it because I said it – do your own investigation and prove it to yourself.

The most recent update to this article is available here:


Introduction to Virology

A good place to start this discussion is with a little education in virology. Columbia University offers a FREE introduction to virology – covered in 26 lectures. The course is very good and is highly recommended. The class slides are provided on the class web site along with links to the lectures on YouTube. The most recent course was taught in early 2020. Here is the link to the course:

https://www.virology.ws/course/
Even if you only understand 5% of what the class covered, it would be well worth your time to sit through the class. So turn off the TV and give the social media sites a rest – take the course – watch the lectures.

**Historic Pandemics**

You should take a look at the major historic pandemics – I will use the CDC’s (United States Centers for Disease Control and Prevention) data:

[https://www.cdc.gov/flu/pandemic-resources/basics/past-pandemics.html](https://www.cdc.gov/flu/pandemic-resources/basics/past-pandemics.html)

The big recent pandemics were:

1918: H1N1 – killing an estimated 675 thousand people out of a US population of 105 million, killing roughly 0.64% of the US population. Scaled to the current US population (330 million), we would have to kill 2.1 million people to match the same death percentage.

1957: H2N2 – killing an estimated 116 thousand people out of a US population of 172 million, killing roughly 0.07% of the US population. Scaled to the current US population, we would have to kill 222 thousand people to match the same death percentage. We crossed that line around October 19, 2020 based on published data.

1968: H3N2 – killing an estimated 100 thousand people out of a US population of 206 million, killing roughly 0.05% of the US population. Scaled to the current US population, we would have to kill 160 thousand people to match the same death percentage. We crossed that line around August 6, 2020 based on published data.

These are not the only significant epidemics around the world. There were many other serious epidemics such as Ebola and HIV/Aids. The three flu epidemics cited above just escalated to what might be called pandemic status, killed a lot of people and then receded into the background after a sufficient percentage of the population had been infected and recovered from the disease.

Note that the killed percentage given above is for the entire population. However, not everyone in the population got the disease. Most estimates are that only 50% of the population got each disease. Thus, the percentage that died from the disease is estimated as roughly twice the percentage provided – i.e., 1.28%, 0.14% and 0.10%, respectively.

I was too young to remember the 1957 pandemic. However, I was in junior high school during the 1968 pandemic. There was no hysteria. The pandemic was only occasionally covered in the news. No one was very concerned. Parents and school officials did not even ask us to wash our hands more often. When people got sick, they mostly stayed home.
To be fair, COVID-19 (Coronavirus Disease of 2019) is not the flu. They are two very different diseases and the mechanisms for infection and reproduction are different.

Flu infections tend to be seasonal – peaking in the winter months and being subdued during the summer months. Thus, if a flu pandemic spans multiple years, you expect to see multiple peaks. The reason for this seasonal peaking behavior is not known but vitamin D deficiency and indoor congregating have been hypothesized. COVID-19 has shown no signs of being seasonal so how well the public follows appropriate hygiene practices will be the controlling factor for where and how high the peaks are.

It is important to emphasize that the viruses that caused the three prior pandemics are still out there – in one form or another. Like all RNA viruses, they are constantly mutating. Kids continue to be born and those kids present a new opportunity for those viruses to spread. And people travel from place to place and take their viruses with them. It is entirely possible that one of the descendants of the prior pandemics will find the right conditions and the virus will flare up as an outbreak and then recede back into the background. In fact, a significant percentage of seasonal flu is attributed to the descendants of those prior pandemic strains. SARS-CoV-2 will join the list after this pandemic.

**SARS-CoV-2**

The SARS-CoV-2 virus (Severe Acute Respiratory Syndrome Coronavirus 2, originally called the Wuhan Virus – the first documented outbreak taking place in Wuhan, China) is an RNA virus – where RNA makes up the genetic code for replication. RNA viruses are very common. For instance, the common cold and influenza (flu) are RNA viruses.

The smaller RNA viruses mutate constantly because their primitive structure has less capability to detect and repair damaged RNA sequences and are therefore more susceptible to random mutations. However, the vast majority of the mutations result in non-functional or less virulent forms of the virus. It is rare for an RNA virus to mutate to a more harmful form – but it can happen.

If you would like to see the family tree for SARS-CoV-2 or some other well-known viruses, take a look here:

**https://nextstrain.org**

The SARS-CoV-2 virus is covered with proteins that give the virus a spiked appearance under high magnification. Viruses of this type are commonly called coronaviruses due to the spiked appearance – corona translates to crown. There are currently seven coronaviruses that are known to infect people. Four of these coronaviruses are common and are estimated to cause a significant amount of annual respiratory illnesses, with colds and influenzas making up most of the rest.
The SARS-CoV-2 virus infects a cell through a process called endocytosis. A protein spike on the SARS-CoV-2 virus attaches to an ACE2 cell receptor. ACE2 (angiotensin converting enzyme 2) is often pronounced Ace Two. After a SARS-CoV-2 virus protein spike attaches to an ACE2 receptor – think lock and key, the cell brings the virus particle into the cell – at which point the viral replication process can begin. The actual process of receptor binding is a lot more complicated, but this simplistic explanation of receptor binding is sufficient for our needs.

Although the upper respiratory system has a significant concentration of environmentally exposed ACE2 cell receptors, many other organs around the body also have ACE2 receptors and are susceptible to infection. Thus it is possible for a COVID-19 infection to start in the respiratory system and then spread through the circulatory system to other organs of the body. This is why COVID-19 patients can exhibit such a wide range of symptoms. The extent of symptoms and damage to body systems can vary dramatically from one person to the next. A person’s overall health – especially your immune system status – has a huge bearing on how much damage will be done.

Other receptors may also play a part in SARS-CoV-2 virus infections. Research shows that the SARS-CoV-2 virus binds to NRP1 (neuropilin-1) receptors, which are abundant in the olfactory epithelium found within your nasal cavity and may explain why many people with COVID-19 suddenly lose their sense of smell. Research strongly suggests the SARS-CoV-2 virus binds to ICAM3 (intercellular adhesion molecule 3) receptors found in alveolar and bronchiolar epithelial cells and may explain bronchiolar mediated silent hypoxemia symptoms but further research is needed to confirm this. Research suggests that integrins (transmembrane receptors) may be a possible binding site but further research is needed to confirm this.

R₀ (capital R, subscript zero) is the basic reproduction number – the measure of how infectious a virus is – i.e., how easily the infection can spread. The SARS-CoV-2 virus (which causes COVID-19, the disease) is believed to have an R₀ of between 2 and 3. That means you need to infect 50% to 70% of the population before the virus stops spreading on its own. Thus, if we take the lower infectivity percentage, you have a roughly 50% chance of acquiring COVID-19 over the next 24 months. There’s no need to get depressed – I’m just being pragmatic here. But like so many other diseases you get during your life, you are very likely to make a full recovery.

Note that R₀ is a statistical concept – it is not constant and is affected by environmental factors. It can go up in a population with poor nutrition or go down in a well-nourished population. It can go up under conditions of squalor or down with more sanitary conditions. It can be higher in a society with a lot of physical interactions or lower in a society with less physical contact. It can be higher in a population that has never been exposed to a similar disease agent or lower in a population that has previous exposures to related diseases. And most importantly,
it goes down over time as people are infected and recover, leaving fewer vulnerable people to become infected.

I will be using the lower $R_0$ (infectivity) value of 2 throughout this article and thus the corresponding requirement to infect 50% of the population before the virus stops spreading on its own. If we use the higher value of 3, that corresponds to a requirement to infect 70% of the population before the virus stops spreading on its own. Notice that the higher infectivity results in 40% more people being infected. As a result, you would expect any associated death count to also increase by 40% because the death rate is assumed to be constant. It is left as an exercise for the reader to appropriately scale any guesstimated death counts to the higher value.

By the end of 2020, a few apparently more infectious strains of COVID-19 were documented to be spreading throughout the world population – the UK variant (B.1.1.7), the South African variant (B.1.351) and the Brazil variant (P.1). In February 2021, two California variants (B.1.427 and B.1.429) were also documented. These variants are classified by the CDC as “variants of concern.” Thus, it may now be more appropriate to use the $R_0$ value of 3. However, when this paper was originally written, the $R_0$ value of 2 seemed most appropriate and has continued to be used throughout.

The SARS-CoV-2 virus is referred to as a novel virus. That is, the SARS-CoV-2 virus is new to humans. But just because this particular virus is “new” to us is no reason to think it does not have similarities to other viruses – other coronaviruses – as far as your immune system is concerned. The fact that the vast majority of people who get COVID-19 recover with no long-term issues is a testament to the effectiveness of our immune system. It is estimated that roughly 80% of people who get COVID-19 have mild symptoms.

The closest known relative to the SARS-CoV-2 virus comes from bats in central China. But that virus does not infect people. There are two main hypothesized routes from the bat to people. The first hypothesis is that one or more zoonotic changes took place through intermediate species in the wild that resulted in an infected animal that infected people at the Wuhan wet market. The second hypothesis is that the bat virus was being studied at the Wuhan Institute of Virology, where the lab was involved in gain-of-function research that resulted in a human infectious virus and there was an accidental viral release that resulted in infected people.

Herd Immunity

There were no vaccines to inoculate the population during the three prior pandemics and thus those pandemics ran their course through the population unhindered over a roughly 2-year period. The three prior pandemics died out on their own when a sufficiently high percentage of the population had become infected and recovered from the disease. There is no reason to believe this
pandemic will be any different. It’s the way infections work. This is how Nature develops “herd immunity.” What doesn’t kill me makes me stronger.

Herd immunity is a statistical concept. It is the point at which a disease is no longer able to spread efficiently through a population and dies out – or just spreads slowly. However, different populations and their susceptibility to diseases are lumpy and not statistically even. Thus, herd immunity in one population may be different from herd immunity in the next population. Looked at another way, herd immunity is when \( R_0 \) drops below 1.

It is worth pointing out that immune response varies tremendously from one person to the next. And an individual’s resistance to infection also varies significantly over time. Although a lot is known about the immune system, there is a tremendous amount that has yet to be discovered. The immune system is capable of remembering past diseases in multiple ways but it can also loose that memory over time. How and why this happens is unknown. One thing we do know is that the single biggest determiner of the immune system’s ability to respond is lifestyle – nutrition, exercise, sleep, social well being and the like. Lifestyle also includes past challenges to the immune system – i.e., the immune system’s education process. There is no substitute for a well-maintained and functioning immune system.

Although medical technology has come a long way, there is no evidence that it is possible to develop, test and deploy a new vaccine for COVID-19 in 12 months – or even 18 months. Thus, the scenario with the highest probability is that this pandemic will run its course through the population before a vaccine can be effectively deployed. Given the relatively low death rate from COVID-19 in healthy people, allowing herd immunity to develop the old fashion way should not be a problem.

**Vaccines**

The FDA (Food and Drug Administration) issued an EUA (Emergency Use Authorization) for the Phizer-BionTech vaccine on December 11, 2020 and a second EUA for the Moderna vaccine on December 18, 2020. Both vaccines require two doses roughly 3 weeks apart. Both vaccines use mRNA (messenger RNA) technologies – mRNA wrapped in a nano lipid particle – and are similar in structure and how they work. They work by invading cells and causing the cells to make SARS-CoV-2 spike proteins.

These are not vaccines in the conventional sense of the word – these drugs are more accurately referred to as gene therapies. The briefing documents for both drugs suggest relative efficacies of roughly 95% with few known serious side affects after only a two-month phase 3 clinical trial – read the briefing documents for a list of qualifications. The efficacy numbers were derived from a small set of illnesses – 170 out of 34,922 for the Phizer-BionTech drug and 95 out of 27,817 for the Moderna drug – so the difference is quite small in absolute terms. The efficacy applies to
getting symptomatic COVID-19 but does not speak to transmission. In fact, the drugs are not designed to actually stop infection – only to reduce the severity of infection. The short length of the trial also does not address the issue of long-term efficacy or safety. The long-term efficacy and safety testing will be run using the general population. Time will tell the tale.

The FDA issued an EAU for the Janssen Biotech (Janssen Pharmaceutical Company of Johnson & Johnson) vaccine on February 27, 2021. This is a single dose vaccine. This vaccine works by using a genetically modified adenovirus to invade your cells – the modified genetic code in the adenovirus causes your cells to make SARS-CoV-2 spike proteins.

This is also not a vaccine in the conventional sense of the word – this drug is more accurately referred to as a gene therapy. The briefing document suggested a relative efficacy of roughly 66% for prevention of serious illness and death with few known serious side affects after only a two-month phase 3 clinical trial – read the briefing document for a list of qualifications. The efficacy numbers were derived from a small set of illnesses – 464 out of 39,058 – so the difference is quite small in absolute terms. The efficacy applies to getting symptomatic COVID-19 but does not speak to transmission. In fact, the drug is not designed to actually stop infection – only to reduce the severity of infection. The short length of the trial also does not address the issue of long-term efficacy or safety. The long-term efficacy and safety testing will be run using the general population. Time will tell the tale.

I would like to point out that these drugs are not “approved” but are actually “investigational” drugs – i.e., experimental. The EAU is an authorization to use an investigational drug – it is NOT an approval of the drug’s safety or efficacy. And it is important to note that not only are the pharmaceutical companies protected from liability for any adverse affects caused by their drugs under federal law (see the Public Readiness and Emergency Preparedness Act of 2005 – the PREP Act) – the required official public health emergency has been declared, you will also be required to sign away any rights to sue the pharmaceutical companies before receiving the drug. Your signature says that you are giving informed consent – are you sure you were fully informed before freely giving your consent or were your being pressured or coerced into signing? Do you realize you are also enrolling in a long-term medical study at the same time? Be sure to read the fine print before you sign.

The EUA authorization presupposes that there is a very high death rate (or some other serious consequence) to justify the experimental use. The EUA approval also presupposes that there is no other appropriate treatment. In the case of COVID-19, we will see that neither of these conditions has been met and thus it can be argued that none of these vaccines are justified in receiving an EUA.

Although attempts have been made to create an approved coronavirus vaccine over the last two decades, none has been able to get past the animal testing stages due to
adverse affects on the animal test subjects related to immune enhancement (pathogenic priming) and inflammatory responses after exposure to the wild virus – resulting in damaged and dead animals. The COVID-19 drugs will be the first mRNA gene therapies ever administered to humans – and that is after the checkered development history – and that is without any published animal study data – and that is after bypassing the normal testing and approval processes – and that is also after giving the pharmaceutical companies complete immunity. Do you really want to be a guinea pig under these circumstances?

Data for adverse COVID-19 vaccination events is available from VAERS – the Vaccine Adverse Event Reporting System. VAERS was created by the US government as part of the National Childhood Vaccine Injury Act of 1986 (NCVIA). The NCVIA protected vaccine manufacturers from liability and created a vaccine injury arbitration system – commonly referred to as Vaccine Court – to oversee limited payments for vaccine injuries for vaccines on the childhood vaccination schedule – but only if your particular injury is officially recognized – and the debate continues to this day about injuries that have been left off the list. VAERS is available at:

VAERS.hhs.gov

You should search for all “COVID” vaccine events to get an idea of what is happening. The site explains that there is a 4 to 6 week delay from when an adverse event happens to when it will likely be reported in the database. It should also be noted that submissions are voluntary and thus the data is only a subset of all adverse events.

The Harvard Pilgrim Healthcare study of VAERS conducted for the Agency for Healthcare Research and Quality (AHRQ) under the US Department of Health and Human Services (HHS) concluded: “Likewise, fewer than 1% of vaccine adverse events are reported. Low reporting rates preclude or slow the identification of “problem” drugs and vaccines that endanger public health. New surveillance methods for drug and vaccine adverse effects are needed.”

On February 20, 2021 VEARS reported 650 deaths in the US from COVID-19 vaccines out of 15,785 total COVID-19 adverse vaccination event reports – 4.1% of event reports. If you go back 4 weeks to January 24, 2021, you will see there had been 25.4 million vaccine doses given. If you go back 6 weeks to January 10, 2021, you will see there had been 11.1 million vaccine doses given. So this gives a minimum death rate per vaccine dose of between 0.0025% and 0.0059%. Two doses are required for these vaccines so you must double this to 0.005% and 0.0118%, respectively. And then you should scale the numbers to account for under reporting. Using a very conservative 10x scaling for under reporting, you get 0.05% and 0.12%, respectively. If we use the study’s estimate, we need a 100x scaling, yielding 0.5% and 1.2% respectively. This creates a realistic bracketing for a vaccine related death rate of 0.05% to 1.2%. And death is only one of the serious adverse events possible.
As a point of comparison, on February 20, 2021 the US per capita death rate for COVID-19 was 0.15%.

Thus you can see that receiving these vaccines will expose you to significant risk. Depending on how you interpret the numbers, the death rate from the COVID-19 vaccine may be of similar magnitude to the death rate from COVID-19 itself. The question is: Is the benefit of vaccination worth the risk? You will have to weight the data and make up your own mind.

On April 25, 2021 the numbers were 2,558 deaths out of 85,926 event reports – 2.98% of event reports; 117 million doses (6 weeks): 0.00219%/dose, 0.0044% for 2 doses, 0.044% to 0.44% scaled; 153 million doses (4 weeks): 0.00167%/dose, 0.0033% for 2 doses, 0.033% to 0.33% scaled. This creates a realistic bracketing for a vaccine related death rate of 0.033% to 0.44%. This result includes roughly 8 million of the J&J single-dose vaccinations – roughly 3% of all vaccines given in the US – and 121 deaths. Thus, the inclusion of the J&J data will not materially affect the results.

If you look at the reported deaths from all vaccines from January 1, 2020 to April 25, 2021 – 2,717 deaths, you will notice that 94% are from COVID-19 vaccinations. And that is after just 4 months of COVID-19 vaccinations compared to a year and a quarter for all other vaccinations. If you account for the reporting delays, we can only use 3 of the 4 months.

As of mid April 2021, it now appears that it is possible for a vaccinated person to cause adverse reactions in non-vaccinated people based on many reported cases. It is hypothesized that the vaccinated person is shedding spike proteins and those spike proteins are causing adverse reactions in some people that are in close proximity. It is not know at this time if this is due to an airborne transmission or direct physical contact transmission or indirect physical contact transmission.

There is a potential issue of molecular mimicry between the SARS-CoV-2 virus spike glycoproteins and the proteomes in humans. It is hypothesized that this may be able to interfere with placental implantation in women due to an autoimmune response in some woman, thus leading to difficulty in reproduction. This is something that needs to be researched before woman of childbearing years receive a COVID-19 vaccine.

The vaccine rollout plan is to inoculate everyone, whether they need the vaccine or not. No attempt is being made to determine who needs the vaccine. After all, if you have already had COVID-19, there is little or no benefit to getting the vaccine. If you don’t need the vaccine, there is no point incurring the risks that go along with getting the vaccine. Current data strongly suggests that the risk is much higher if you have already had COVID-19.
As I have already noted, these vaccines are arguably not designed to address true immunity but try to reduce the severity of symptomatic COVID-19. As a result, they may have little effect on the length of time needed for the pandemic to run its course because they may not contribute significantly to herd immunity.

As of mid December 2020, there are many different potential vaccines undergoing trials throughout the world – over 50 COVID-19 vaccine candidates are being worked on. How many of those vaccines will make it to large-scale distribution in a timely fashion remains to be seen. And what percentage of the population will accept those vaccines also remains to be seen.

It should be pointed out that there is a phenomena called viral interference that can take place with other vaccines – such as the annual flu vaccine – depending on how the vaccine was manufactured. If the vaccine was grown in animal tissues that contained other coronaviruses – ones that don’t infect humans, those other coronaviruses can contaminate the vaccine and lead to adverse reactions when the person becomes exposed to the wild SARS-CoV-2 virus.

**Pandemic Data**

So where are we in the current pandemic? It’s a moving target. A common place to start is with the John Hopkins data:

https://www.arcgis.com/apps/opsdashboard/index.html#/bda7594740fd40299423467b48e9ecf6  (copy/paste entire URL, remove extra space after first line)

The CDC’s weekly death data along with detailed information on the limitations of that data can be found here:

https://www.cdc.gov/nchs/nvss/vsrr/COVID19/index.htm

And if you want the Arizona data, you can go here:


As of mid March 2021, the worst per capita death rates in the general population by country are rapidly approaching 0.2%. Given the trends of the curves, there is good reason to guess the typical per capita death rate will exceed 0.2% in most European and American countries over the coming months. Whether or not the values will approach or exceed a 0.3% per capita death rate in the general population is anyone’s guess. A death rate of 0.2% is one person in 500. A death rate of 0.3% is 1 person in 333.
But before we go into too much detail about the dead folks, let’s try to unravel the numbers around the sick and recovered folks.

**Cases Versus Infections**

There is an important distinction that needs to be made – the difference between a case and an infection. A case is when someone is officially diagnosed with the infectious disease – even if that diagnosis is wrong. An infection is when someone gets the infectious disease – whether or not they are officially diagnosed with the disease. We will discuss the issues with counting cases later. The reality is we have no clue how many people have become infected by the SARS-CoV-2 virus so picking a number for infections is just a guess.

We need two important pieces of information to figure out a death rate. The first thing we need to know how many people died from the disease – the numerator. We can use one of the published figures for this number. Then we need to know how many people got the disease – the denominator. You can use the published number of cases for this number for the case fatality rate or you can guess at the number of infections for the infectious fatality rate. The quotient – the result of the division – gives us the fatality rate.

Thus we have a case fatality rate and an infection fatality rate. The case fatality rate will always be greater than the infection fatality rate because there will always be fewer cases than infections. You must always keep in mind the limitations with both of these fatality rates. They should always be viewed with suspicion.

**Cases**

If you want to know what is currently happening and the trends, use the daily or weekly rates. But as the CDC explains in detail, these numbers need to age. It takes time to gather the complete set of data. By the time the numbers are many weeks old, most of the data should be reported and the published numbers should be relatively close to where they are likely to end up. That means the data has become more stable and is less likely to change dramatically. That does not mean the data is accurate – as we are about to discuss – it just means the data is stabilizing around its likely final value.

It is important to understand what counts as a case. It all hinges on the definition of a case. A case can be “confirmed” through laboratory testing. Or a case can be only “suspected” based on symptoms. Or a case can be “inferred” due to proximity to another case. These distinctions are very important to the accuracy of the resulting numbers so let’s cover them one at a time.

A confirmed case is one that is confirmed through a laboratory test. We will take up the issue of testing accuracy in a moment, but for now we will just assume there is an accurate test. The primary use of a test is to ascertain if someone has a specific
condition so that specific condition can be treated in an appropriate manner. If you are not going to change how you treat a condition based on the result of a test, there is little point in testing for it.

An example of this would be respiratory infections. Respiratory infections can be caused by cold viruses, influenza viruses, coronaviruses and bacterium. For the vast majority of cases – i.e., for viral respiratory infections – there is no point in testing because there is no special treatment being selected. Thus, it is rare to test for respiratory viral infections because there is no specific treatment for those viral infections. If you don’t suspect a virus and you think treatment is needed that requires testing, you are probably testing for a specific infectious bacterium that can be treated by a specific antibiotic or other drug – guided by careful medical observations.

A suspected case is based on matching symptoms. The problem is, many viral infections present many similar symptoms – e.g., fever, cough, sneezing, sore throat, nausea, diarrhea, achiness, lethargy. Thus, suspected COVID-19 patients are said to have COVID-19-like symptoms or have a COVID-19-like illness. You must then ask what set of symptoms will be used to identify a suspected case of COVID-19 and distinguish it from one of the other diseases with the similar symptoms. Testing has shown that only a small percentage of people with a COVID-19-like illness test positive for COVID-19 – typically less than 15% on average and often under 7%. Thus, using suspected cases can substantially inflate the number of COVID-19 cases.

An inferred case is based on the assumption that all members of a household (or other group) must be positive if any member of a household (or other group) is positive. This assumption is wrong on average. It is common to see few other members of a group becoming infected if one other member becomes infected. Thus, using inferred cases can substantially inflate the number of COVID-19 cases.

The next issue is that different reporting areas may be using very different methods for counting cases and the methods can and do change over time. For instance, how people are being selected for testing within a reporting area can have a large impact on the number of confirmed cases. Does the area include suspected cases? Does the area include inferred cases? We quickly get to a situation where we are faced with comparing apples to oranges to bananas – the resulting numbers may have very little practical utility or medical relevance, if any.

The next issue to understand relates to testing and the accuracy and meaning of test results. False positives and false negatives are part of the testing process. Test results can be influenced by how the samples are taken, when in the infection cycle the sample is taken, the particular test used and how the test is processed.

The common PCR (Polymerase Chain Reaction) tests are testing for traces of the virus’ genetic material – they are not actually testing for the SARS-CoV-2 virus itself. Thus it is common for a PCR test to find detectable genetic material (dead viral
debris) long after the last live SARS-CoV-2 virus has been eliminated from your body. Another issue is that it is possible to have a non-target virus with similar genetic material generate a false positive. There are many different COVID-19 PCR tests in use that can be looking for different things as stand-ins for the virus.

The FDA (Food and Drug Administration) has issued EUAs (Emergency Use Authorizations) for the current COVID-19 PCR tests – they are not formally approved in the conventional sense – as would happen after rigorous testing. The PCR methodology was never designed to be used as a diagnostic tool – it was a research tool to amplify the presence of genetic material for further study.

The amount of genetic amplification – or cycle threshold (Ct) – used to process a test influences the sensitivity as well as the accuracy of the test – higher sensitivity results in lower accuracy – i.e., more false positives. Too much amplification is a common problem resulting in a lot of false positives. Suffice it to say that PCR testing has not been the reliable gold standard it was advertised to be. If you get a PCR test, you should make the lab provide the amplification used (i.e., the final Ct value) and a list of conditions that will generate a false positive so you can determine if a positive result is likely to be valid.

Research has shown that the SARS-CoV-2 virus cannot be cultured when Ct values exceed 35. The likelihood of being able to culture the virus does not become significant until the Ct value drops below 30. The likelihood of culturing the virus is approaching 100% as the Ct value drops below 20. In general, a positive test result – in the absence of symptoms – does not correlate with you being infectious.

So why would any lab use amplifications that result in Ct values over 35 – or even 30, which are likely to generate a false positive result? It boggles the mind. Yet, this has been the standard practice. Since test results are reported as a binary value – positive or negative – there is no way to assess what percentage of positive test results are likely to be false positives. But it is likely to be quite high.

The WHO (World Health Organization) published a notice on January 13, 2021 that said: “WHO guidance Diagnostic testing for SARS-CoV-2 states that careful interpretation of weak positive results is needed. The cycle threshold (Ct) needed to detect virus is inversely proportional to the patient’s viral load. Where test results do not correspond with the clinical presentation, a new specimen should be taken and retested using the same or different NAT (nucleic acid testing) technology.”

It goes on to say: “WHO reminds IVD (In Vitro Diagnostic Medical Device) users that disease prevalence alters the predictive value of test results; as disease prevalence decreases, the risk of false positive increases. This means that the probability that a person who has a positive result (SARS-CoV-2 detected) is truly infected with SARS-CoV-2 decreases as prevalence decreases, irrespective of the claimed specificity.”
Antigen tests look for viral proteins and produce faster results but have higher error rates compared to PCR tests. As you are not testing for the actual virus, a non-target virus may generate the same protein and generate a false positive.

It is important to keep in mind that testing in the US in May 2020 was generally restricted to people who were sick and showing COVID-19-like symptoms. If you were sick and chose not to seek medical help and just stayed home, you were not tested. Even if you tried to see the doctor, you still might not be tested. Thus, it was guesstimated that there were 10 times more actual infections than “confirmed” cases in the US population.

In the May 2020 timeframe, you would typically see a positive result for only 6% to 8% of symptomatic people tested. The positive rate rose to 10% to 12% by June and July 2020 during the summer peak and then dropped again before heading back up in the fall and winter. Positivity rates rose to around 25% in some places during the winter 2020/2021 peak before falling again to much lower levels. It should be noted that there were large variations from one region to the next and one time period to the next.

The time between getting a test and receiving results has been an ongoing problem. The longer it takes to receive your test result, the less useful the result will be. By the time the result is delayed by a week, the result is nearly worthless because the decision about what to do was already made without any consideration for the eventual test result. Again, you should establish the utility of a test before getting tested.

A positive result turns you into a confirmed case. However, a positive result does not mean you are infectious and a negative result does not mean you are not infectious.

If you are symptomatic – i.e., you are feeling sick and have symptoms – and you receive a positive result, it is probably good to assume the test result is correct and you should stay home and quarantine. But if you were symptomatic prior to taking the test, you should have stayed home anyway. Going out when you are sick and spreading a cold, the flu, strep or COVID-19 to family, friends and neighbors is just rude social behavior.

If you are asymptomatic – i.e., you feel fine and don’t have symptoms – and your test comes back positive, you don’t really know much. Did you get a false positive? Do you have a very mild case? Are you still in a long incubation period? There is no way to tell. 95% of people develop symptoms within 14 days of exposure. That means that 5% of people take longer than 14 days to develop symptoms. Sorry, since you took the test, you should stay home for the prescribed quarantine period.

If you are symptomatic – i.e., you are feeling sick and have symptoms – and have a negative result, you should stay home until you get better. You obviously have
something – you just don’t know what it is. You don’t want to give whatever you have to someone else in any case.

If you are asymptomatic – i.e., you feel fine and don’t have symptoms – and your test comes back negative, you really don’t know much. You might have a false negative result and be in the incubation period discussed above. Or you might have nothing. As long as you feel fine and remain free of symptoms, you can probably go on with your life without fear of infecting others. If you are asymptomatic, you are either not sick or your symptoms are sufficiently mild that you are not likely to be infectious. Remember, people with very mild symptoms for viral respiratory infections - be that a cold, the flu, a coronavirus or COVID-19 – tend to shed very few viral particles and are thus at low risk of infecting others.

As you can see, the case numbers are suspect while testing is problematic. For all of the reasons I have discussed, there is a high likelihood that the published COVID-19 case numbers are significantly overstated – even if they are a gross undercount of actual infections. Be very careful what conclusions you draw from case numbers and test results. The unscrupulous will use these results to terrorize the public and justify all kinds of mandates and controls or to otherwise push agendas that don’t benefit the public.

**Published Numbers**

Now that we have an understanding of where the published numbers are coming from – and a better understanding of the limitations of those numbers – let’s take a look at the numbers and try to figure out what is going on.

There were 1.6 million reported COVID-19 cases by May 24, 2020. It is not practical to ascertain how many of those were confirmed so we will just assume they are some combination of confirmed, suspected and inferred.

We addressed the issue of confirmed cases versus undetected infections due to under testing previously and settled on a published guesstimated scaling of 10x from May 2020 to account for under testing. Just remember that the scaling applies to confirmed cases, which is a number we don’t really have access to. We only have access to undefined case numbers that may or may not include suspected and inferred cases and may have other problems we are not aware of – such as significant false positives. So these numbers should always be viewed as suspect.

Therefore, let’s just assume – for the sake of this discussion – that there were closer to 16 million infections on May 24, 2020 – 1.6 million cases times our 10x scaling. This is a very rough guesstimate at best.

In the end, what do all those numbers mean? Not much. They are a vague guesstimate of what is going on with the infection spread. The numbers may be intellectually interesting and fun to talk about but beyond that, the numbers are of
marginal practical utility. However, the numbers can be used for political purposes so you should be very careful.

The problems with published data have continued as the pandemic has worn on. On February 16, 2021, Arizona was reporting 800 thousand cases (746 thousand confirmed, 54 thousand suspected) but only 593 thousand positive test results. That’s a discrepancy of roughly 153 thousand test results. Remember, a case is only supposed to be confirmed if there is a corresponding positive test result. Nothing on the dashboard page tries to explain how 593 thousand positive test results generates 746 thousand confirmed cases. I have used the larger reported case number for consistency because that is the number that gets used for calculating the national figures.

On July 15, 2020, the Arizona data showed that 1.8% of the 7.4 million Arizona population had tested positive for COVID-19. Of course, that assumes no one took multiple tests and tested positive multiple times – just saying. If we assume the same 10x scaling to account for under testing noted above, we can guesstimate that 18% of the Arizona population has been infected. If we assume roughly 50% of the population has to get infected for the spread to stop on its own, we can guesstimate that Arizona is one-third the way through the pandemic.

The figures rose to 2.9% and 29% respectively by September 20, 2020. By November 15, 2020, the numbers had risen to 3.7% and 37% respectively. By December 26, 2020, the numbers had risen to 6.7% and 67% respectively. By March 14, 2021, the numbers had risen to 8.2% and 82% respectively.

Another interesting data set to look at is the Arizona hospital COVID-like and influenza-like illness surveillance graphs. Each has twin graphs – one for emergency department visits and one for inpatient – I will use whichever is higher. This data spans December 2019 to February 2021.

For the influenza-like illness graphs, you see a prominent 2019/2020 winter peak around 9%, a smaller 2020 summer peak around 4% and a very low 2020/2021 winter peak around 3%. The valleys between peaks are around 2%.

Looking at the COVID-like illness graphs, we see a low level for the 2019/2020 winter of around 4%, you see the 2020 spring peak around 7%, the 2020 summer peak around 20% and the 2020/2021 winter peak around 29%. The valleys between peaks are around 4%.

You will notice the roughly 4% COVID-like illness rate existed two months before the first confirmed COVID-19 cases in Arizona. You will also notice the almost complete lack of a winter 2020/2021 flu season. Do you think there might be an issue with how symptoms are being classified?
Deaths

So let’s move on to deaths. It is important to keep in mind that not all “COVID-19 deaths” are “confirmed” deaths. So all of the issues we ran into when trying to understand what constitutes a case will also apply to what constitutes a COVID-19 death – with some additional issues thrown in.

There is a big difference between dying from COVID-19 and dying with COVID-19 – even though that distinction is not reflected in the numbers.

If you get COVID-19 and you die because of COVID-19 – for example, you get pneumonia or acute respiratory distress syndrome (ARDS) as a direct result of COVID-19 and die, you died from COVID-19.

If you have a historic health problem, such as heart disease or cancer and you die from that historic health problem but happen to test positive for COVID-19, you died with COVID-19.

Then you have the in between cases. If you have a historic medical problem and COVID-19 makes it much worse, are you dying with or from COVID-19? Your historic medical problem and COVID-19 may both share blame. As long as the COVID-19 makes a substantial contribution, you probably died from COVID-19.

This goes to the core of the issue of underlying medical conditions – also called comorbidities. Having an underlying medical condition, be it cardiovascular, cancer, respiratory, diabetes or what have you, can dramatically lower your survival rate if you also get COVID-19.

The root cause of many of these preexisting conditions typically comes down to lifestyle choices, which includes cultural influences and other modifiable environmental factors – only a tiny percentage will be genetic. And these same lifestyle choices will also influence your immune system. So the same lifestyle issues that caused your underlying medical condition are probably also responsible for reducing your immune response and making you more susceptible to COVID-19.

Finally, if you have a historical health problem and due to the pandemic you cannot get treatment for that problem and die as a result, should that count as a COVID-19 death? Many doctors and hospitals restricted medical care offerings during the pandemic. And many people were either not able to get to treatment or were too afraid to go for treatment. As a result, a lot of otherwise treatable medical conditions went untreated and a lot of additional people died. These deaths are generally not counted as COVID-19 deaths.

There is also a conflict of interest in classifying hospital patients with a respiratory illness as a COVID-19 case. As part of the Coronavirus Aid, Relief and Economic Security (CARES) Act, hospitals were paid extra for COVID-19 cases. The first round
of funding started off based on historic Medicare revenue and thus the payments varied dramatically from one state to the next. Nebraska was the highest at $379,000 per case while New York was the lowest at only $12,000 per case. By May 2020, the payment changed to $76,975 per COVID-19 case. For the July 2020 second round funding the amount was changed to $50,000 per COVID-19 case with added restrictions. For the third round of funding in December 2020, the application and compensation process got real complicated – too complicated to explain here. It should be noted that hospitals are not the only healthcare entities to qualify for similar funding.

Death certificates become the permanent historic record so any misclassification listed on a death certificate will turn into historic fact.

All of this leads to a legitimate disagreement as to how to classify any given death. As you can see, there is a significant financial incentive to fudge the diagnosis by including a COVID-19 diagnosis whenever possible. We have already seen that very few people with COVID-like illness are actually COVID-19 positive after a lab test and that lab tests can have a high false positive rate. Thus, we must conclude that the death numbers for COVID-19 are significantly overstated. How inflated the numbers are will remain for history to estimate after the pandemic is finished.

Most hospitals and other care facilities prevented family and friends from visiting COVID-19 patients during the pandemic. Even non-COVID-19 patients suffered isolation from family and friends. It is well known that social connections and visitations are important to the psychological wellbeing of all patients. Keeping patients isolated can be seen as a form of psychological torture and likely had a negative affect on many outcomes. Forcing patients to die along is unconscionable, vicious and nasty.

Data indicates that COVID-19 has an exponentially increasing risk of hospitalization and death with age. From around age 20, the risk doubles every 16 years – about 4.5% per year. Not huge but significant. And men have a risk that is roughly 50% higher compared to woman. This exponential increase in risk with age and higher male risk is common with many other diseases.

As of January 28, 2021, about 35 to 40% of US deaths due to COVID-19 were from people living in long-term care facilities. In some states, that would go as high as 80%. Long-term care facilities account for only 1% of the US population but these facilities house a high concentration of the nation’s most vulnerable people and the people in these facilities tend to be much older. Administrative procedures in these facilities were also a significant contributing cause.

It is instructive to look at the COVID-19 deaths by age group. Below is the data from February 10, 2021, when there were 476 thousand reported US COVID-19 deaths:
- Under 5 years of age: 68
- 5 to 14 years: 69
- 15 to 24 years: 626
- 25 to 34 years: 2,804
- 35 to 44 years: 7,395
- 45 to 54 years: 20,403
- 55 to 64 years: 51,786
- 65 to 74 years: 94,964
- 75 to 84 years: 123,412
- 85 and older: 141,580

Put another way, on September 10, 2020, the CDC’s estimated that the age-stratified infection fatality rates for COVID-19 should be:

- 0 to 19 years: 0.003%
- 20 to 49 years: 0.02%
- 50 to 69 years: 0.5%
- 70+ years: 5.4%

Age is a significant risk factor. It is clear that older populations will generally have a higher death rate compared to younger populations. However, the state of someone’s health is probably the most significant risk factor – even if it is more difficult to ascertain. Poorer health only generally correlates with increasing age. But even in the highest risk age group, the survival rate is estimated to be 95%.

For perspective, it was estimated that 607 thousand people died from cancer in 2019 in the US – a per capita death rate of 0.184%. Cancer is the second leading cause of death in the US. This happens year after year – yet no one gets very upset.

For a bit more perspective, the 2016 John Hopkins study estimated that 250 thousand people die each year from medical mistakes – the third leading cause of death in the US. However, due to the way medical errors are reported, some estimates have this number as high as 440 thousand deaths per year. Year after year. Why aren’t people up in arms about these preventable deaths?

The data showed that children were at very low risk from COVID-19 by summer 2020. Children generally have mild symptoms and rarely have complications requiring medical care unless they have an underlying medical condition – children have a very low rate of underlying medical conditions. Further, children pose a lower risk of passing on a COVID-19 infection as their viral loads tend to be much lower than that of an adult. Yet, most US public schools remained closed.
Predicting Deaths

The bottom line is this: How many people are going to die from COVID-19 as a result of this pandemic? What will be the eventual fatality (death) count?

Returning to our May 24, 2020 data, there were 97 thousand “COVID-19 deaths” and 1.6 million cases reported. We have already discussed why these figures are suspect but we will use them because they are the officially published data. These numbers yield a case fatality rate of 6.1%. If you assume 50% of the US population gets COVID-19 and this death percentage stays the same, we can guesstimate the pandemic will kill almost 10 million people – roughly five times the number of the 1918 pandemic when scaled to the current US population. If we use our guesstimated 10 times scaling to account for under testing, this drops to 0.61% – only about 1 million dead – roughly half that of the 1918 pandemic scaled to the current US population.

If we use the April 25, 2021 numbers – 32.1 million cases and 571 thousand deaths, we get a case fatality rate of 1.78% and can guesstimate the pandemic will kill almost 3 million people. If we use a more conservative 5 times scaling to account for under testing given the much higher testing rate, this drops to 0.35% and thus only 580 thousand people may die from this pandemic.

Still, this suggests the case fatality rate has fallen by two thirds from May 24, 2020 to April 25, 2021. That’s quite an improvement. But the numbers don’t provide any clue as to why this has happened. Is it due to the pandemic moving into a younger population with a commensurate lower death rate? Is it due to better medical procedures for treating intensive care patients? Is it due to the way numbers are accumulated and reported? Unfortunately, we don’t know.

The real death percentage can shift dramatically as the pandemic continues. As the pandemic moves through new segments of the population, those new segments may be more or less likely to die compared to previous segments of the population. As the treatment of critical care patients improves with experience, you expect the percentage of critical care patients surviving to improve. And current data shows the survival rate of critically ill COVID-19 patients has at least doubled over the last many months and is now similar to other comparable illnesses.

Sounds dreadful, doesn’t it? I’m sorry – it’s a pandemic and a lot of people are going to die. Just keep in mind that guessing at the eventual death rate is still guessing, no matter how many decimal places you use. Or put a different way, extrapolating suspect data into fanciful results is a fool’s errand.

Let’s look at a second method to guess at the eventual death count. For this method, we start with a monthly death count. The April 2020 COVID-19 death count as reported on May 29, 2020 by the CDC was roughly 50 thousand COVID-19 deaths. The data was at least a month old at the time so the data should have been
sufficiently stable. Pandemics tend to run for roughly 2 year so we can extrapolate that to 24 months with the same monthly death count. This yields 1.2 million deaths in the US.

If you compare “COVID-19 deaths” to “Pneumonia and COVID-19”, the death rate drops by half. That would lower the guess to only 600 thousand deaths using this method. Given the primary cause of death from COVID-19 is from pneumonia related issues, this may be a better number.

Note that we have assumed the pandemic will run continuously at the same level for 24 months and then suddenly stop. As of mid February 2021, the monthly death rate has averaged roughly 40 thousand deaths per month over the last 12 months. If we were to simply double this to 24 months, we get a guesstimated 980 thousand deaths by the end of the pandemic.

If you want the answer today, you must predict the future – which is notoriously unreliable.

On July 15, 2020, the Arizona data showed that 0.034% of the Arizona population had died from COVID-19. On February 21, 2021, the Arizona data showed 15,505 total COVID-19 deaths – 0.21% of the Arizona population. With 808 thousand reported cases, that is a case fatality rate of 1.9%. On the same day, the US data showed almost 500 thousand COVID-19 deaths for a per capita death rate of 0.15% and 28.8 million cases for a case fatality rate of 1.8%.

The per capita death rate graphs tells an interesting story:

https://ourworldindata.org/coronavirus-data-explorer?zoomToSelection=true&time=2020-03-01..latest&country=USA~GBR~BEL~SWE&region=World&deathsMetric=true&interval=total&perCapita=true&smoothing=0&pickerMetric=total_deaths_per_million&pickerSort=desc (copy/paste entire URL, remove extra space after each line)

Looking at the per capita death rates for early July 2020, Belgium was the highest with 0.086%. The USA was at 0.041%. The per capita death rates reported for September 16, 2020 were 0.086% for Belgium and 0.060% for the USA. By November 13, 2020, this had increased to 0.121% for Belgium and 0.074% for the USA. By December 16, 2020, this had increased to 0.158% for Belgium and 0.092% for the USA. By April 25, 2021, this had increased to 0.206% for Belgium and 0.176% for the USA.

You may be wondering why I picked Belgium for this discussion. Simple. They have consistently had the highest per capita death rate for a country with a significant population (11.6 million). This is interesting because there is a reasonable probability that any other large European or American country will achieve a
similarly high per capita death rate if given sufficient time. Thus, Belgium may be a good indication of where we are all headed.

The shape of the Belgium per capita death curve is also interesting. Around mid October 2020 there is a transition from relatively flat to significantly upward. In early November 2020 the curve reaches an inflection point while still heading upward – i.e., the rate of change in the per capita death rate has gone to zero while the per capita death rate is still high. Following the inflection point the curve begins a slow and smooth transition toward what may be a maximum value. In other words, the rate of increase is smoothly decreasing toward zero.

If you extrapolate the curve, you can guesstimate the per capita death rate will reach 0.2% to 0.25%, at which point the deaths may stop. Thus, this may be the point at which the pandemic is over for Belgium – or any other country that gets there. If you are an optimist, this means the pandemic is close to being over. If you want to be pessimistic, that may just be a plateau before the next significant increase.

Let’s move on to consider the weekly graph of all deaths against the averaged historic data of weekly deaths. Weekly numbers above the average numbers represent excess deaths that are presumably caused by some current phenomenon – such as the current pandemic. This method may do a better job of separating the folks that were going to die anyway from the folks that died primarily from COVID-19. Here is a link to the CDC data on excess deaths associated with COVID-19:

https://www.cdc.gov/nchs/nvss/vsrr/covid19/excess_deaths.htm

You will notice there was a large spike that peaked on the week ending April 11, 2020. After that, there was a rapid fall heading back toward the threshold for excess deaths line. You will notice that by the week ending June 6, 2020, excess deaths were approaching the threshold line. We ignored the last month of data – which was well below the threshold line – due to the likelihood that data is incomplete. As it turned out, this was to be just a low point in the graph that was followed by a significant broad peak during the summer months, which was followed by another low area in the early fall followed by a large spike in the late fall and early winter when many more months of data had become available.

The excess deaths graph can show all deaths, only COVID-19 deaths and non-COVID-19 deaths. There were a lot of non-COVID-19 excess deaths during the first spring peak (2020) and during and just after the second summer peak (2020). The data is probably too young to say anything about the third winter peak (2020/2021).

Current estimates are that between 25% and 50% of all excess deaths during this pandemic are not from COVID-19.

But this just begs a different question. Where did all of those non-COVID-19 excess deaths come from? Is it because the medical system is so focused on COVID-19 that
they are ignoring the common fatal disease states such as cardiovascular disease and cancer that kill a lot of people each year? How about trauma injuries? Or are people now afraid to go to their doctors for treatment? Or are people no longer able to get to their doctors for treatment? Or are people so stressed out by losing their jobs and/or being isolated that they are committing suicide? Or are people dying from excessive self-medication while trying to cope?

**Preventing Deaths**

If you look at the death data for many third world countries, you will see an interesting phenomena. The COVID-19 per capita death rates for many – if not most of these countries – are much lower than for the northern European countries and the US. Why is that? Do all of these counties suffer from bad medical data? For instance, look at India – a very crowded, mostly poor and rather unsanitary country. Yet their per capita death rate is roughly 10% of the US per capita death rate. How can that be?

Proactive early treatment is the key to preventing death for most fatal diseases. If you wait until symptoms become very bad before beginning treatment, you dramatically increase the probability of death. This is especially true if you are a high-risk patient – someone over the age of 50 with any comorbidities. In other words, hospital care for critical patients comes with a much higher fatality rate, as well as a far higher risk of long-term lung, heart, neurological and other complications for those who do survive.

Two unpredictable aspects of COVID-19 are an exaggerated inflammatory response – the cytokine storm and an exaggerated blood clotting response. Both of these exaggerated responses can result in extensive damage to body organs and death.

Early COVID-19 treatment requires a multi-drug approach to ensure the best rate of success. The treatment options involve “off-label” use of common drugs. Off-label use is when a medication is used for a purpose for which it is not currently licensed – at your doctor’s discretion. Roughly 20% of all prescriptions are for off-label uses of prescription medications so off-label use is a very common practice in medicine.

Hydroxychloroquine (HCQ) is a widely used, safe and inexpensive malaria drug that was shown to be effective against COVID-19 when used as part of a multi-drug treatment system. HCQ is also used to treat systemic lupus and rheumatoid arthritis and has several other off-label uses. HCQ has been shown to be most effective as a prophylaxis or for treating early stage COVID-19.

The Chinese mandated HCQ be used for COVID-19 in February 2020 – about three months after the pandemic started in China. The Indian government made it a national policy to recommend HCQ to be broadly used for the prevention and treatment of COVID-19 in March 2020. Both countries have a very low per capita death rate, but to be fair, there might also be other factors at play.
During the same period, the US and EU countries essentially banned the use of HCQ and all have very high per capita death rates. Why would an advanced country discourage early treatment for a disease? How many deaths might have been prevented by promoting the use of HCQ instead of actively discouraging it?

HCQ is an inexpensive “off-patent” drug in common use throughout the world with a 65-year track record and is commonly available over-the-counter (i.e., without prescription) in most countries. How inexpensive is HCQ? Around $25 per treatment in the US – about $1 per pill. The competing drug – remdesivir – has a high toxicity profile with severe side effects and costs around $3500 per treatment and can only be used in a hospital setting.

By March 2021, nearly 200 published studies have shown HCQ to be safe and effective for treating COVID-19. There have also been several fraudulent studies showing HCQ to be harmful and ineffective by using known toxic doses – which is rather immoral – or by only using it in late stage treatments when it is known to be much less effective. One fraudulent study was published in The Lancet – one of the world’s most prestigious medical journals. The study was shown to be fraudulent shortly after publication – the data for the study had been made up – that's right, fabricated – the study was never performed. The fraudulent study was quickly withdrawn. You can find the retracted fraudulent Lancet paper here:

https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(20)31180-6/fulltext

You should note that when the study was first published, it was a major front-page news story and the media made a big deal over how dangerous HCQ was. However, once the study was shown to be a fraud, the news media went silent. Such a significant fraud being published in a prestigious publication should have been big news – but instead, it was buried. So much for journalistic integrity.

Ivermectin is another inexpensive off-patent drug that has been studied and shown to be effective against COVID-19. Ivermectin is an anti-parasitic drug that has been used to treat hundreds of millions of people all over the world for head lice, skin rashes and parasitic worms with a 45-year track record. Ivermectin has been found to be more effective in late stage COVID-19 than HCQ. Ivermectin can also be used as a prophylaxis and early stage treatment for COVID-19.

Fluvoxamine is a more recent discovery and has undergone at least one small double-blind study showing safety and effectiveness against COVID-19.

Even now – in April 2021 – the official US medical position is that there is no preventative or early treatment for COVID-19. Just go home and wait for the disease to advance to the point you need to go to the hospital.
If you want early treatment, you will need to find a doctor that is both knowledgeable about COVID-19 early treatment protocols and is willing to prescribe drugs for these treatment protocols. Groups such as Association of American Physicians and Surgeons or American Frontline Doctors may be able to help you find a qualified local doctor.

One such protocol was published in the American Journal of Medicine:

https://www.amjmed.com/article/S0002-9343(20)30673-2/fulltext

This protocol was derived from an earlier protocol developed for treating the original SARS virus over 10 years ago. This protocol can also be applied to other viral respiratory infections.

Studies have shown that low levels of vitamin D correlate strongly with higher death rates. Thus, you should be able to reduce the number of COVID-19 deaths by providing appropriate levels of vitamin D. But the issue is probably not just a vitamin D deficiency. If you have a vitamin D deficiency, you probably also have deficiencies in other vitamins or minerals, which were not looked at during these studies. So there could be a larger nutritional issue at play here. In the end, poor nutrition will adversely affect your immune system and reduce your chances of a good outcome.

One can argue that if the US and other countries had been proactive with early treatments and had been willing to use these inexpensive and effective drugs for early treatment, there would be no need for COVID-19 vaccines.

**Conclusions on Death**

To conclude this talk about death, let’s ask the question: How important are these numbers? They are probably less important than you think. Once a pandemic enters a population without a concerted effort to stop it – i.e., tracking down and treating the infected, the pandemic is free to run – and it will run its course. In the end, the numbers are just going to be statistics. The numbers serve no real utilitarian medical purpose during the pandemic. However, the numbers are likely to be used for all kinds of political purposes – i.e., psychological manipulations – so be warned.

For all of the reasons I have discussed, there is a high likelihood that the published numbers of COVID-19 deaths are significantly overstated and thus include a substantial number of non-COVID-19 deaths. The argument is not whether or not people got sick or died – the argument is whether or not the sickness or death was due to COVID-19. This pandemic is probably much less severe than the published numbers indicate – specifically, the death rate from COVID-19 is likely to be much lower than advertised. Due to a high false positive rate in confirmed, suspected and inferred cases, the published death rate may be 2 to 5 times higher than actual.
Let’s return to the 1957 pandemic for a moment. Scaled to the current US population, 222 thousand people would have died. The published number of dead from COVID-19 was 572 thousand people for April 25, 2021. But as we have seen, the numbers from testing, suspected and inferred cases contain a high percentage of false positives. Thus, it may be reasonable to conclude that the COVID-19 pandemic is no worst than the 1957 pandemic and perhaps significantly better. Since no one got very upset during the 1957 pandemic, why is everyone so upset during this pandemic?

**Long Haul COVID-19**

You should also consider long-term disability caused by COVID-19 – sometimes referred to as long haul COVID-19. Just because you don’t die from the disease does not mean you will fully recover from the disease. This syndrome has been reported following cases ranging from very mild to very serious. Remember the ACE2 receptors all around the interior of your body? You may be left with damaged organs. That damage may remain for months, years or the rest of your life.

There is little published data to help assess long haul COVID-19 as of December 2020. So it is impossible to say what percentage of COVID-19 infections will turn into long haul cases. The syndrome appears to be related to a combination of vascular (blood vessel) damage, neurologic (nerve) damage and autoimmune (inflammation) damage. The combined damage can produce a wide range of symptoms that have no obvious cause and simply persist month after month.

There are no known treatments for the syndrome. And you should note that other viral infections can also produce long haul affects.

**Infection Rate**

Let’s return to the infectivity rate discussed earlier. Wouldn’t it be nice to know what percentage of the population has been infected with COVID-19 and is now part of the immune herd? Or put another way, what percentage of the population remains to be infected before the pandemic runs its course and quits?

By the beginning of October 2020, the WHO (World Health Organization) estimated that 10% of the world population had been infected. That’s 760 million out of 7.6 billion. That figure is for the world population and is only a guesstimate. The John Hopkins data for the same date shows 35 million cases worldwide and 1 million deaths worldwide. That works out to roughly 21 times more infections than cases. I used 10 times scaling to account for under testing in the US for early in the pandemic when testing was limited and 5 times scaling for later in the pandemic when a lot more testing was available.
The WHO’s estimate of infections yields an infection fatality rate of 0.13%, which seems low. But given the world’s younger average population and the wide use of HCQ and Ivermectin, perhaps it is not so far off. This places the COVID-19 pandemic in a similar category to a bad flu season.

As you can see, there are a lot of unknowns. We don’t know how many people have already acquired the disease and recovered. We don’t know how many people remain to get the disease. We don’t know what percentage of otherwise healthy people who get the disease will die from the disease. We don’t know how much the treatments for the disease – or the side affects of the disease – will improve and thus how that will affect the number of very ill people who survive instead of dying. We don’t know if a vaccine will be effectively deployed before the pandemic runs its course. It will likely be well after the pandemic is over before we have a descent estimate of what really happened.

**Preventing COVID-19**

Now let’s turn our attention to reducing our probability of catching COVID-19. Is there any magic or anything special we need to do? Not really. Just do the same old stuff that’s been taught for decades. Here’s a link to the CDC’s recommendations:


It should be emphasized that the following guidelines are good practice in general, especially during the annual flu season. We should have been doing this stuff all along.

The basic steps are very simple:

1. Get people to stay home when they have symptoms or feel sick
2. Appropriate physical distancing during interactions
3. Wash your hands as needed
4. Don’t touch your face with dirty hands
5. Get people that are particularly vulnerable to take extra precautions

The SARS-CoV-2 virus spreads most easily directly from person to person and less easily indirectly via other surfaces. This is mostly due to the amount of viable virus that can be transmitted and this determines the likelihood of transmission. In microbiology, this is the concept of the minimum infectious load needed to transmit an infection. Note that the minimum infectious load has not been determined for the SARS-CoV-2 virus but the current evidence suggests the infectivity is similar to colds and the flu.

The virus degrades outside the body and thus infectivity drops rapidly with time at normal room temperatures. Exposure to direct sunlight speeds the degradation. For this reason, most surfaces will decontaminate themselves and no disinfection is
needed. In other words, the same level of cleaning used to guard against colds and the flu should suffice.

Having a good immune system is very important to preventing any infection. Your body’s immune system is very capable. The standard guidelines of good nutrition, regular exercise, enough sleep, low stress, social connectedness and a low toxin exposure will keep your immune system in top shape. These very same guidelines also reduce the common comorbidities – i.e., things that increase the likelihood of worse outcomes – such as high blood pressure.

Some people with COVID-19 are asymptomatic – meaning that they do not feel sick and they do not show any signs of being sick. People can be asymptomatic prior to becoming symptomatic (i.e., pre-symptomatic – during the incubation period) or they may never become symptomatic because they have a very mild case. In theory, you may return to being asymptomatic as your body finishes clearing an infection. Such is life. But the same applies to most other common respiratory infections.

When looking at infectious respiratory diseases, people are generally considered to be infectious when they are symptomatic and they are generally considered to be not infectious or not very infectious when they are asymptomatic.

So how did we get to infectious asymptomatic COVID-19? Fear mongering and bad science. Fear – False Evidence Appears Real, or False Evidence made to Appear Real. There are no studies I can find to document any significant number of confirmed asymptomatic cases that are also infectious – that is not the way common infectious diseases work. In fact, a huge Chinese study in Wuhan during the summer of 2020 found no asymptomatic transmission. In any case, the transition at the end of the incubation period where you may be infectious and asymptomatic is not a significant driver of infectious spread within the population.

There will be a transition period during which you go from the incubation period to symptomatic and it might take you some time to realize you are no longer feeling well. But that will be a short transition period of perhaps an hour or two. Unless you are interacting closely with others during this period, there will be a low probability of infecting others. If you should start feeling sick, you should tell others and go home – it is the polite thing to do.

How infectious someone is depends on how much virus they are shedding and that is generally thought to correlate strongly with symptoms. This is common to many infectious diseases. Thus, if you have no symptoms you are either not diseased or not very infectious.

If you treat everybody you meet as if they are infectious, you are less likely to do the wrong thing by mistake and become infected.
Social and Physical Distancing

The historic concept of social distancing had to do with preventing social classes from mixing. The rich should not mix with the poor. The whites should not mix with the blacks. You social distance from your inferiors – however or whomever you define as your inferior. In other words, social distancing was based on prejudice. The Bogardus Scale developed by Emory Bogardus in 1924 is used to measure the willingness of people to interact with members of other social groups.

The concept of physical distancing being applied to infectious disease started in the late 1800’s when German bacteriologist Carl Flügge showed microorganisms in droplets expelled from the respiratory track was a means of disease transmission – they were called Flügge droplets. He went on in 1897 to propose using masks to stop Flügge droplets from causing infections to open wounds during surgery.

William Wells further quantified what happens to droplets with the Wells curve in 1934 – showing a relationship between droplet size, time and evaporation, formalizing the 2-meter (6.5 feet) distance for larger droplets to hit the ground while smaller particles desiccated and became airborne.

The 2-meter distance was derived from a set of assumptions about how fast droplets were expelled and the relative evaporation rate in still air. The larger droplets follow a standard parabolic ballistic curve. As you decrease the distance, the larger droplets will contact you – starting at your feet, then your legs, then your torso and even your face if you get close enough. Any further away and the larger droplets will all hit the ground. Smaller droplets dry out before hitting the ground and become airborne and are free to travel long distances. This will be explained in more detail shortly. All this hinges on statistical probabilities under assumed conditions.

Then came the Bush administration’s work on combating bioterrorism and new animal disease transfers. By 2005 there were working documents. In 2006 a high school science project on teenage social networks was integrated into a disease spread model that suggested closing schools would cut infections by two orders of magnitude. By 2007, after further studies of the 1918 flu pandemic, papers had been published suggesting shutdowns and isolation were the way to go. Arguments to the contrary that suggested the ineffectiveness of shutdowns and predicted the social and economic disaster closing most of society would cause lost out. And the rest, as they say, is history.

People have always known that staying away from the sick was protective. This is why sick people have been isolated throughout history. But it was only recently that the concept of social networks was refined into formal public health policies that isolate the healthy.
If you are going to converse with someone for an extended period of time, physical distancing can make sense. But if you are both asymptomatic and feel fine, very little will be accomplished by it.

**Masks and Respirators**

The spread of a virus through the air can take multiple forms. For instance, if you are coughing, sneezing or even just talking, you are expelling mucosal particles. Regular breathing expels relatively few mucosal particles. The mucosal particles range in size from big enough to see to very small. If you are coughing or sneezing, you should be staying home – unless you have to go to see your doctor.

The larger particles have a higher mass and travel in straighter lines, following a standard parabolic ballistic curve as they are slowed by air resistance and pulled down by gravity. These particles do not tend to travel very far before falling to the ground and are easy to catch using a course filter such as a multi-layer cloth mask.

Smaller particles have a lower mass and are more susceptible to air currents and less susceptible to gravity. The smallest of these particles are able to travel on air currents and require a much finer filter to capture.

Add to this the relative humidity. In higher humidity conditions, mucosal particles retain their water and thus their size and weight – and more of them fall to the ground. As the humidity drops, the evaporation rate goes up. Smaller particles have a higher surface area to volume ratio compared to larger particles. In other words, smaller particles shrink in size faster relative to larger particles due to evaporation. Thus, smaller particles can completely desiccate under dryer conditions and become sufficiently small and light to drift on air currents.

A mask is designed to catch and stop mucosal particles along a direct path. These particles have enough mass that they are propelled directly from your mouth or nose into the mask material and captured. Even though masks are relatively course filters, they will catch and retain larger mucosal particles as well as many of the smaller midsized mucosal particles. Thus, if you are symptomatic and infectious and can't figure out how to stay home – or you are trying to see your doctor, you wear a mask to protect others from your mucosal particles.

Masks do very little good when it comes to people who are physically distanced.

Masks are not sealed and are not fine filters and thus the smallest mucosal particles that ride on air currents will escape through the mask material or out the edges of the mask. Masks do slow and disperse the air stream from your mouth and nose, which will act to disburse any concentrated plume of virus. But this is only of concern if you are not physically distanced.
This takes us back to the concept of minimum infectious load. The more virus you are exposed to, the higher the probability you have of being infected. In general, it takes a minimum infectious load to transmit an infection. Anything less and the body can prevent the infection. Big mucosal particles can carry a much higher viral load compared to smaller particles. One big mucosal particle may be just as infectious as a bunch of smaller mucosal particles. You want to avoid either exposure.

COVID-19 and influenza viruses are small. Really small. 120 nm (nanometers) or 0.12 μm (micrometers or microns) small. Less than a wavelength of violet light small – the dividing line between violet and ultraviolet light is at 400 nm – so roughly a third of that. As a point of comparison, a human hair will average around 100 μm – roughly 800 times larger.

Smoke particles are of similar size to common respiratory viruses. A mask will NOT filter out smoke. So a mask will NOT filter out viral particles.

Thus, if you are infectious and wear a mask and have a long conversation with someone at close range, you can easily create a large enough exposure to infect the person you are talking to – even if the other person is also wearing a mask. If you are engaging in this type of behavior, you clearly do not understand the limitations of a mask.

RNA viruses such as SARS-CoV-2 degrade outside the body over a period of minutes to hours – depending on conditions. Thus, desiccated viral particles begin to deteriorate and over time lose their ability to cause a new infection. The data on how quickly this happens under different circumstances is lacking – the variation in circumstances is large – so it is not possible to make a clear definitive statement.

Masks work equally well in both directions. This is why medical people wear a mask when seeing potentially infectious patients. The mask is to stop splashed bodily fluids and larger mucosal particles expelled by the patient from entering the medical person’s mouth and nose along a direct path. They offer essentially no protection from airborne viral particles.

Note that the protection from masks is only provided for someone directly in front of your face. If you are going to cough, sneeze or talk to someone while wearing a mask, face the other person so the mask can do its job. This is a very different reflex from turning away and coughing or sneezing into your elbow when you are not wearing a mask.

Masks need to be changed on a regular basis – every couple of hours at the most. Your mask is up against your face and because of your constant breathing, the mask will become damp and become a good environment for accumulating bacteria and viruses. Every time you touch your mask, whether on the outside to adjust it or on the inside to scratch an itch, you are contaminating your mask. At the same time,
every time you touch your mask, whatever is on your mask will contaminate your hands. Damp dirty masks quickly become a source for transmitting infections. The same is true with respirators.

Remember, your goal is to reduce your viral exposure by appropriate practical methods applied consistently. Masks are not magic. Masks will not make up for bad decisions, bad habits, poor hygiene or a defective immune system. In other words, masks are highly overrated in a public setting. Conversely, if you think you need a mask to protect others – because you might be infectious, you should not be out in public.

A study of 14 randomized controlled trials with lab confirmation of infection published by the CDC in May 2020 concluded: "We did not find evidence that surgical-type face masks are effective in reducing laboratory-confirmed influenza transmission, either when worn by infected persons (source control) or by persons in the general community to reduce their susceptibility." It should be pointed out that cloth masks are even less effective than surgical-type masks.

Respirators are very different from masks. A respirator is designed to protect the person wearing the respirator from airborne contaminants in the environment. To provide this protection, the respirator must be properly fitted, sealed against your face and the seal must be tested. People have different face shapes and different respirators are designed to fit different face shapes. Thus, you must find a respirator that is designed for and fits your face shape. Then it takes training and practice to put on a respirator and validate a good seal. A respirator with a bad seal is no better than a mask and may be worse if it has an unfiltered front valve. As a result, respirators are not recommended for the general public.

Most respirators used in industry and for medical purposes are N-95 rated – meaning they stop 95% of all particles that are at least 0.3 microns (300nm) in size from being inhaled. N-95 respirators are not designed for use with tiny RNA viruses such as the SARS-CoV-2 virus that are roughly 120 nm (0.12 μm or microns) in size. N-95 respirators may reduce the inhaled viral load as these respirators have good filter efficiencies – but they will not prevent an exposure.

### Contact Tracing

Contact tracing is used to track down the origins of a disease and is commonly used to track many diseases. Properly implemented, contact tracing with treatment and isolation can theoretically stop the spread of most infectious diseases and even eliminate them from the general population.

The method is conceptually straightforward. When you discover a person with the target disease, you try to identify everybody the infected person has been in contact with over some period of time – that length of time could be the disease’s incubation period plus a few days. Then you go to all of the identified people and repeat the
process. Each identified person is evaluated and treated for the disease or placed in quarantine. In this way, you find the infected people and isolate them from the general population or otherwise treat the disease and thus prevent further spread of the infection.

Note that the CDC’s definition of a contact for the purposes of COVID-19 is spending at least 15 minutes face-to-face with someone at a distance of less than 6 feet without any personal protection equipment – such as a mask. This should give you a good idea of what it actually takes to transmit the disease. Casually passing someone on the street or stopping for a quick hello is not likely to transmit the disease.

There are many factors that can reduce the effectiveness of contact tracing. For instance, if the disease can be spread asymptomatically, there will not be any cause for an asymptomatic person to contact the health system and thus the health system will not be aware of the infectious person other than through the symptomatic people infected – think Typhoid Mary. Or, if the health system fails to make a good case to the public and engender trust in the public to encourage voluntary cooperation.

If contact tracing is to work, it must be a committed effort – if done half-heartedly, it is doomed to failure. Contact tracing must be integrated with public education. The public must be educated about the disease, how it spreads and effective methods for stopping the spread. The messaging must be truthful and consistent. Lying to the public is counterproductive and will instill mistrust throughout the public and discourage cooperation.

It is important to ask if the illness justifies the considerable effort needed to contact trace as contact tracing is quite expensive. For illnesses that have a high ongoing cost to society, contact tracing may be a good option to reduce the cost of treating an illness in the general population or preventing a high death rate. However, many illnesses – such as the common cold or flu – will see a relatively low return on investment so contact tracing is not done in these cases. COVID-19 probably falls in the latter category.

**Rapid Spread and Flattening the Curve**

The rapid spread of an infectious disease can produce a large spike in medical cases that can exceed the capacity of available local medical resources – such has hospital beds, medical equipment or medical personal. You can prevent a large spike in cases – and thus prevent overwhelming the medical system – by “flatten the curve.” The curve (the graph of the number of cases over time) is flattened by slowing the rate of spread. The same number of people may still get sick and need medical attention, but now the cases are spread over a much longer period of time so the medical system capacity is not exceeded.
How do you flatten the curve? Simple. You lower the population’s probability of transmitting the disease over the period of interest. We already covered ways for individuals to accomplish this. To extend this to the whole population may require extensive public education and perhaps the expenditure of resources to implement the practices. You also need the public’s trust and cooperation. If most people participate, this should produce the desired results.

It is important to realize that as long as the infectious disease continues to exist in the general population and as long as a large percentage of the population is vulnerable to the disease, the risk of rapid spread continues to exist. The threat of rapid spread will exist until a high enough percentage of the population has had the disease and the disease can no longer spread efficiently. In other words, until the population has developed herd immunity. The goal of good public health education is to popularize good hygiene habits and sufficient physical distancing so as to lower the probability of transmitting the infectious disease.

Another way to flatten the curve may be to provide early treatment. For many diseases, early treatment can stop or slow the progression of the disease. Early treatment usually takes few medical resources and may be able to prevent the disease from progressing to a state that requires hospitalization and other limited medical resources.

**Stopping COVID-19**

Let’s revisit the question of stopping the spread of COVID-19. We have discussed how individuals as well as populations can lower the probability of acquiring the disease. We have also mentioned early treatment. We have also discussed how contact tracing can be used to track down and isolate cases. This begs the question: Is it practical to stop a disease like COVID-19 once it has entered the general population and spread widely? Or is it better to just let the disease run its course through the population?

If you want to eradicate the disease, you must find and treat every case and completely eliminate it from the population. If even one case remains to infect a vulnerable population, the disease will be off and running – again. The cost in terms of contact tracing will be high. The benefit will be the lives saved if you are successful. The risk of failing will be high because perfection is the only acceptable outcome. Remember, to be effective, you must eradicate the disease from all populations of the world at the same time – while the population as a whole remains vulnerable.

If you let the pandemic run its course, some percentage of the world’s population will get sick and some percentage of the sick will die. The cost in terms of lives lost may be high. The benefit will be great because you now have a population that is mostly immune to the disease (herd immunity) and thus future outbreaks will be limited – the strong survived. The risk will be low as there is a natural tendency for
the disease to spread through the population and so the spread will not stop until after herd immunity has been established.

That said, if you can keep the number of infections relatively low for an extended period of time in a mostly unobtrusive way and if you assume you can eventually create a vaccine that the public will take, accepting a low long term infection rate might be a good option. But that requires a lot of assumptions.

What about the case where you are never able to develop a vaccine? There are many diseases for which no vaccine has been developed even though extensive resources have been applied to the problem over many years. What is the optimum rate of disease spread through the population to get to herd immunity? Is there an unobtrusive way to accomplish this? And at what point in the pandemic does deploying a vaccine become pointless? That is, at what point have so many people had the disease that it will be difficult to find the remaining people to give the vaccine to – without inoculating the entire population?

**Why is This Pandemic Different?**

Now we turn to an interesting sociology question. What made this pandemic different from the last three pandemics?

A lot of things have changed over the last 50 to 100 years. The world’s population and population density has increased significantly. Our transportation infrastructure allows someone to travel between most population centers of the world in a matter of hours. Social media allows lies, conspiracy theories, disinformation and propaganda to spread at an alarming rate through the population. A large percentage of the public now has underlying medical conditions – creating a lot more comorbidities and susceptibility to new diseases. Modern medical technology can keep critically ill people alive for months beyond when they would have otherwise died – monopolizing intensive care resources for extended periods. Globalization has pushed manufacturing to the cheapest resources including cheaper international labor pools and has led to vulnerable supply chains. General technology is far more advanced, and with it, our expectations for controlling our destiny. Let’s not forget our changing society, the political climate and powerful special interests. And finally, COVID-19 is not the flu.

In past pandemics, life mostly went on and people mostly went about their business. A few very large events got cancelled while other very large events took place. There was not much hysteria. People were not panicked even if they were fearful of the disease. People died and those deaths were accepted and mourned.

So many people died during the 1918 pandemic that they ran out of coffins but society generally kept going. Hospitals were filled to capacity and beyond but society soldiered on. Medical technology to keep people alive for months on end was not available so very sick people just died – requiring fewer total beds for a
similar volume of patients. A few large cities closed down entertainment establishments and even schools, but the rest of society mostly continued.

But then came COVID-19 followed by lock-downs and hysteria. Why did this happen? Why did governments choose to place their own healthy populations under house arrest instead of allowing their societies to continue functioning in a normal fashion as happened in prior pandemics? Historically, you isolate those who are sick – not the healthy.

Were lock-downs necessary to flatten the curve or were they counter productive? Would an honest, coordinated public health education program have accomplished the same thing without the hysteria, economic damage and sociological damage? Is it reasonable to think we can prevent the deaths that such a pandemic will bring without eradicating the disease through effective hygiene, contact tracing or a vaccine?

Published research has documented that the spring 2020 lock-downs across Europe produced no net reduction in deaths. The dropping death rates that took place coincident with the lock-downs had already started dropping prior to the lock-down and were the normal natural affect of infection spread that has peaked. Further, when you account for an average incubation period of 5 days and an average of 18 more days to death, you will see there is an average of 23 days from infection to death. There was no significant change in the death rates in the neighborhood of 23 days following the start of lock-downs. Finally, the very same peak and decline in death rates observed in the lock-down countries was also observed in Sweden, which did not implement any lock-downs. Other countries that avoided lock-downs had similar results.

The behavior around the peak is consistent with an increasing perceived risk that causes individuals to change their personal behavior in a way that reduces transmission. The research finds that these behavioral changes had already taken place in the weeks leading up to the lock-downs and thus the lock-downs provided no additional benefit in exchange for all of the harms the lock-downs caused.

Studies published by mid November 2020 show that lock-downs are very destructive to the general population – both economically and socially, not to mention the issue of destroyed civil rights.

But even with all of this data showing lock-downs are harmful and ineffective, governments were still imposing lock-downs in 2021.

Sweden is an example of a society that bucked the lock-down and hysteria trend and chose the more traditional route. Sweden’s approach emphasized honest public education along with individual responsibility and hygiene – the very things we have already discussed. Swedes were encouraged to work from home when practical. Children under the age of 16 continued to go to school and as of June 15,
In 2020, older children have also returned to school. Anyone who felt ill and the elderly were asked to stay home. Restaurant tables were moved further apart. Congregating at the bar was discouraged. Gatherings of over 50 people were banned. And the Swedes did not wear masks.

The Swedes understood the COVID-19 pandemic was a long-term event. The Swedes understood the pandemic was going to run its course, whether they liked it or not. The Swedish philosophy was to flatten the curve unobtrusively and ride it out.

To be fair, the Swedes have taken the position that very sick people in long-term care facilities should be allowed to die instead of keeping them alive for weeks to months using the latest life support systems. These people are provided regular end-of-life hospice services – including “comfort” medication – and allowed to die. This eliminates a huge burden on the hospital system and goes a long way to preventing COVID-19 cases from overwhelming the healthcare system.

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The Swedish per capita death rate was listed as 0.043% for June 2, 2020 – roughly halfway between the USA and Italy at the time. Sweden’s per capita death rate rose to 0.057% by September 16, 2020 while the USA’s rate had risen to 0.060%. By December 16, 2020, the Swedish per capita death rate had risen to 0.075% while the USA’s rate had risen to 0.092%. By April 25, 2021, the Swedish per capita death rate had risen to 0.137% while the USA’s rate had risen to 0.176%. Until the pandemic is over and the final numbers have been tallied, there is no point in trying to compare the Swedish outcome to the outcomes of other nations. But early results indicate the Swedes were probably right. Time will tell the tale.

The recent CHARM (COVID-19 Health Action Response for Marines) study was published in the New England Journal of Medicine on November 11, 2020 (DOI: 10.1056/NEJMo2029717). The study used Marine recruits to provide a near homogeneous group that underwent supervised quarantine, distancing, masking and environmental decontamination with regularly scheduled testing. The results showed transmission occurred in spite of implementing best-practice public health measures.

Governments around the world employed the psychology of fear appeals to take away civil rights and suspend firmly established medical science. Fear appeals have three major components: the audience, the message, and the recommended behavior. The audience was the governed population. The messages changed over time – becoming a series of messages that included: protect the hospitals (i.e., flatten the curve); millions are going to die in the US; you are going to die a lonely miserable death from COVID-19; your children will bring COVID-19 home from school and kill you and their grandparents; you are going to bring COVID-19 home to your family and kill them; the people you meet on the street are Typhoid Mary – i.e., infectious asymptomatic carriers; the person walking around without a mask is going to infect you; vaccination is the only road back to normal life; vaccination does
not prevent you from spreading the disease to others; the virus has mutated and gotten worse; vaccination is needed to prevent more mutations; you will need a vaccine passport to travel or participate in social events; you will need booster vaccination shots to protect you from the mutated virus strains; you will not be safe until the whole world has been vaccinated. The recommended behaviors were: stay isolated at home; keep your kids isolated at home; always wear a mask – or two; get your vaccine shots; only believe what the government tells you – everyone else is spreading disinformation. They want to you be afraid – very, very afraid.

Even the worldwide press mostly repeated the government propaganda without question. The major media organizations rarely questioned the validity of recommendations or mandates or any of the studies behind them. Sad stories telling of family members dying were everywhere to keep your fears and guilt front and center. Even the huge tech giants got on board by censoring material that did not agree with the government propaganda – I was able to find and review a few censored items and found them to be in agreement with pre-COVID-19 established medical science.

When I was younger, I often wondered how Hitler was able to bring down Germany so easily. How were they able to send 6 million Jews to the gas chambers without objections? Now that I have lived through this pandemic, I understand just how easy it is to herd sheep and discredit those who do not comply. We have taken the old propaganda methodologies and dramatically improved upon them using everything we have learned about human psychology in the last 70 years.

Take the Fear Formula, for example – what does Fear Formula stand for? F – frighten people – foment fear to get people’s attention. E – provide exaggerated examples – the more extreme examples usually work better. A – accuse someone – people need a scapegoat – be angry and show your animosity – antagonize your foe. R – repeat the examples and accusations over and over – move the goal posts if needed. F – fabricate fake news – it does not matter how far fetched it is – if you say it often enough, people will believe it. O – ostracize your opponents – marginalize them – denigrate them - repeatedly. R – reticule any opposing remedies – repeatedly. M – manipulate the messaging – change the meaning of their words. U – unite the uninitiated – use group-think against the agnostic – make them feel foolish if they don’t agree with you – invite them into your utopia. L – lead late – he who leads late often loose the least – your later lies linger the longest. A – applaud your actions through shameless promotion – take credit for doing good things regardless – proclaim improvements or victory even if none happened – be a great spin doctor. Credit for laying out the Fear Formula goes to Scott Jensen, MD.

What we have witnessed with the COVID-19 pandemic can only be described as unprecedented fraud on a massive scale. The corruption pervades many – if not most – aspects of our society. Do you remember the old saying: Follow the money?
It will be interesting to see what future research reveals when it looks back on the COVID-19 pandemic. What drove decisions? And what conclusions will history come to about those decisions? It will also be interesting to see what changes take place to the fabric of our society as a result of this pandemic.

Postscript

The original version of this article was written in early May 2020 in response to US Fish and Wildlife Services and USGS – along with other organizations – worrying about giving North American bats COVID-19. Why? The cavers, bat researchers and perhaps the general public were somehow going to give the North American bat population COVID-19. Then, somehow, those same bats were going to spread COVID-19 to people – or other wildlife. Or perhaps COVID-19 was going to become the new WNS bat infection that would kill bats. There is no data to even suggest these scenarios are possible but that does not stop bureaucrats from speculating up a doomsday scenario. And the probabilities of these things happening were amazingly high in their report. The human-to-human transmission vector is many orders of magnitude more efficient than any possible bat-to-human vector. The same holds true for vectors involving other wildlife. Not to mention one or more zoonotic changes to the virus are probably required before any such transmission could take place.

This is the same lousy science displayed with White Nose Syndrome (WNS) – a Eurasian bat fungal disease that has killed a lot of North American bats. Cavers and even researchers were being excluded from caves because they would somehow infect bats with WNS. The bat-to-bat transmission vector is many orders of magnitude more efficient than any possible human-to-bat vector. The only documented human-to-bat transmissions of WNS involve a white lab coat, a swab and a spore loading of 100,000 to 300,000 – i.e., a scientist intentionally infecting a bat in the lab. That large spore loading is phenomenally high – but you cannot give a bat the disease without it. Bats are very social animals and roust together as well as groom each other so bat-to-bat contact can easily achieve the spore loading needed to pass on the infection to other bats. Humans rarely interact with bats in the wild – humans walk on the ground while bats fly and hang from the ceiling – so it is nearly impossible for a human to give a bat WNS – even if the human was somehow covered in spores. There is a huge difference between what is theoretically possible and what is statistically probable. A CDC paper documenting a large number of international bat translocations years before the American WNS infection started along with the fact that the first identified infection was only 35 miles from a deep water port was dismissed out of hand. Nope, it must have been from the mud on someone’s boot who had been to Europe.

Following the initial article oriented to the bat issues, the article was rewritten as a general article for a broad audience and has been expanded and updated many times since. I hope you find it educational.