An alternative model for Corona Virus Spread

www.Hunt4Answers.com

Abstract:

The 1918 Spanish flu is remarkable in the number of deaths attributed to the disease. Somewhere between 17 million and 100 million persons died of Spanish flu between February 1918 and April 1920. A consensus of reports is that the Covid-19 crisis has caused just over 1 million deaths worldwide. Data regarding Spanish flu deaths is sparse. Data availability for Covid-19 is better than Spanish flu data. However, Covid-19 data is lacking for many reasons, mostly non-medical.

This paper proffers a cohesive theory of Corona Virus Spread working with available data. Emphasis is regarding the so-called second wave of infections during the 1918 Flu Epidemic. It also offers a correlation between the 1918 Flu Epidemic and the current Covid-19 epidemic.

Background and Purpose:

The 1918 flu epidemic presented an enigma unexplained until now. Figure 1 is of the 1918 Spanish Flu expressed in daily cases per thousand. At first glance, there seem to be three peaks of activity over about two years. Further analysis reveals many more peaks leading to a unique insight into the spread of the Covid-19 virus.

The questions explored here are:

1) Why did the flu go away?

2) Why did it come back?

The answer to the first question is certainly not 'herd immunity' since millions more persons would have died to achieve herd immunity and a flu vaccine was not available. There were 550,000 Spanish flu deaths in the United States or 0.5 % of the population[1]. To achieve herd

[1] https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2740912/#:~:text=The%20estimated%20population%20of%20the,generally%20put%20at%2020%20million

immunity in the United States herd immunity would have been on the order of 5 million[2] persons assuming a 2.5 % mortality rate.

Instead there is a more reasonable answer to the first question and it requires only data presented in this report. The answer also has the einsteinian advantage of being simple. The methodology utilizes a novel approach to analysis of publicly available data. The first, and unexpected, result is that the data demonstrates the extreme similarity between the 1918 Spanish Flu and Covid-19. Historic events of the time coalesced to obscure data analysis. Compensation for historical differences will reveal an astonishing correlation between the 1918 Spanish flu epidemic and the 2019 Covid-19 epidemic. Once the correlation is established, further startling revelations are evident. If the reader considers the answer to question one reasonable, the proposed answer to question two will be stunning.

Scientists divide human immune systems into innate and adaptive components[3]. The innate system gives limited immunity to nearly every virus. Adaptive immunity is a 'learned' immunity where the body develops an immunity specific to that virus but only after exposure. Adaptive immunity is termed either passive, transferred between generations, or active, learned by exposure.

I'm going to invent a phrase, environmental immunity. It is immunity created within the individual in reaction to the environment. This is not a new concept. As a young married man, my wife was a receptionist in an ear, nose and throat medical practice. She never got the common cold, the flu or any of the transient sicknesses transferred by aerosol contact. The kids and I would get these things from school and work but even though she attended to us when we were sick, she never had a runny nose while she was a doctor's receptionist. I noticed because I was very jealous. Her job exposed her to a number of pathogens on a daily basis. Since the contact was relatively brief and remote, the dose she received was adequate to initiate an immune response but not adequate to overrun her immunity. The immune response accumulated with each exposure counteracted by antibodies. Literally, that which didn't kill her made her stronger.

This environmental immunity is well known and accepted. Environmental immunity is difficult to control and manipulate resulting in little literature on the subject. However, it is the

[2] Population of 103,000,000 persons Herd Immunity Threshold (HIT) of 50% and lethality of 5 % or 2,575,000 persons.

[3] Immunity https://en.wikipedia.org/wiki/Immunity_(medical)

only method available to create the original response to a virus by a species. Understanding the chemical mechanisms of environmental immunity is the basis of all current research.

Methods:

Primary information sources include the Bing Coronavirus page and the Wikipedia 1918 Spanish Flu page. Data analysis is by the author and based on decades of simulator experience[4]. The paper includes both source and conclusion data are included in this paper in graphic form.

The 1976 swine flu outbreak was during this time so I did research. Fortunately, the 1976 Swine flu epidemic fizzled out but my interest did not. I also read quite a bit about the 1918 flu epidemic where more people worldwide *died* than were infected by Covid-19 even though they practiced mask use and social distancing[5]. As I read and analyzed information about the various outbreaks of the flu, I could never understand multiple outbreaks during the same year. Apparently, no one else could either. Finally, I decided to employ spectral analysis similar to that used in X-ray fluorescence. The discipline involves separation of complex data peaks into constituent components for the purpose of analysis. We can analyze the relationships between the constituent peaks and analyzed them with consistent results.

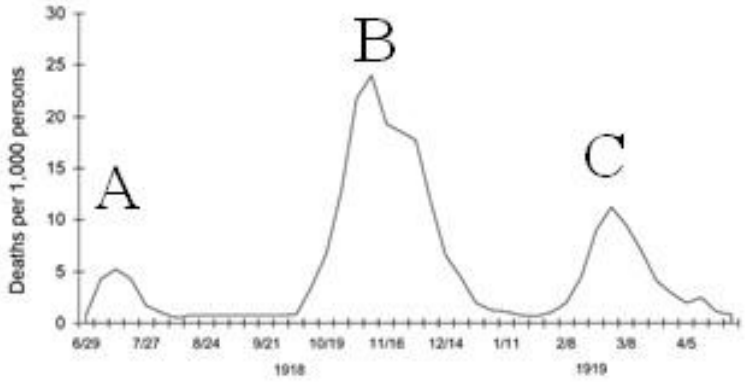

Figure 1 - 1918 Spanish flu deaths

Referring to *Figure 1 - 1918 Swine flu deaths*, the curves are actually quite complex. First, let's assume the data are Gaussian in nature[6] thus the curves are obviously the algebraic sum of multiple curves. Please notice the flat top on the first curve (A) around June, July 1918. That

[4] PCBArtist 2 with LTSpice Designer's Guide
[5] There are pictures of policemen and nurses wearing masks.
[6] The curves are not totally Gaussian but the left side (rising) slopes are. There is further discussion later in the paper.

flatness indicates that there are two overlapping peaks of approximately the same magnitude. The second curve (B) has a peak and shelf on the right side indicating a large primary peak in late October 1918 and a smaller secondary peak in mid November of 1918. The third curve (C) has a peak at approximately early March 1919 with a secondary peak in early April 1919.

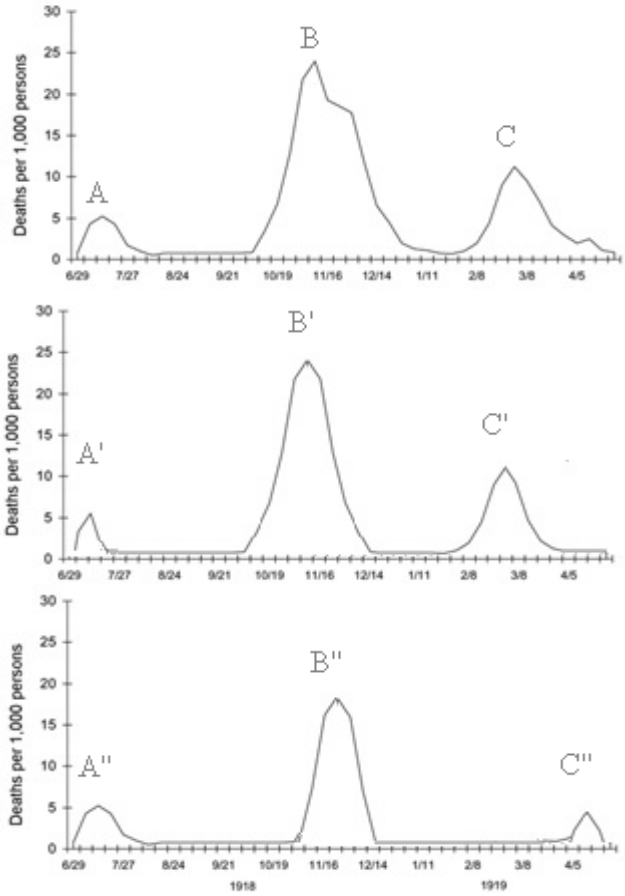

Figure 2 - 1918 Spanish flu curves devolved

Referring to *Figure 2 - 1918 Spanish flu curves*, the author has redrawn the complex curves of Figure 1 into the three sets of curves in Figure 2. After separating the complex curve into simpler components, the two sets of curves are reveled. The top display is the combined set of curves, the middle display (A', B' and C') is a set of curves related to each other by their periods or time between peaks, and the lower display (A", B" and C") is a set of curves again related to each other by their periods or time between peaks. The period between peaks in the middle set of curves (A' B' and C') is about 17 weeks. The period between peaks in the middle set of curves (A" B" and C") is about 19 weeks. The lag between B' and B" is about two weeks. The lag between C' and C" peaks is about four weeks. Peak selection was on the basis of the period

between the peaks of each derived curve. Standing alone, these revelations are not overly significant.

A reasonable interpretation of these curves might be that the data is the combination of two locations, say Europe and the United States. If we concentrate on the period between peaks of the middle and lower charts, we can see that this is the type of curves generated if the original data were a combination of the United States and Europe. Let's assume train is the dominant mode of travel for both areas. The United States is a large country with long travel times for the dominate means of travel and Europe is a smaller area with shorter travel distances and the same means of travel. With these reasonable assumptions, the middle set of curves would reflect Europe's shorter travel times and the bottom set of curves would be the United States with longer travel times. This analysis is plausible and interesting but again not especially revealing by itself.

There is more information available. If we could take the mathematic derivative or slope of the curves, we could determine the rate of growth in each area (the US or Europe.) Information sources for the 1918 flu epidemic are not adequate to perform that sort of analysis. Furthermore, the 1918 data is not limited to confirmed cases of the disease since there was no testing regime at the time. It is worth mentioning here that analysis to that level would be possible if data were time stamped indicating the day, date and time of death to compensate for delays in reporting caused by natural disasters, weekend operation and holidays.

Let's assume that the above analysis is accurate for the Spanish flu. How does it correlate to Covid-19 or SARS-2? For the purposes of this phase of the evaluation, let us gather the curves for each of the 50 states. Let us further assume that there are some generic types of spread allowing us to classify the infection rate curves as Urban, Mixed, Non-Mixed and Other. New York is a good example of Urban spread; Louisiana, of mixed spread; and Texas, of Non-Urban spread.

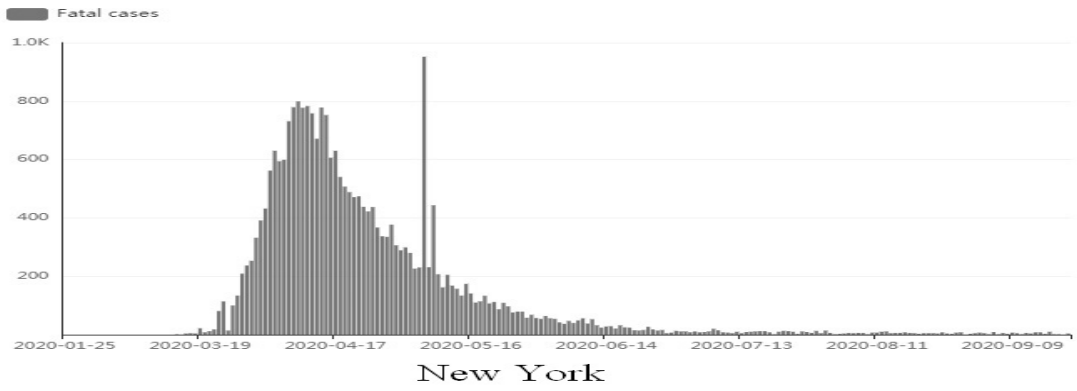

Figure 3 - New York - Urban Spread

Urban Spread

In the case of Urban Spread (see *Figure 3 - New York, Urban Spread)*, the virus spreads quickly and ends quickly without returning as a 'second wave.' Further analysis indicates that the death rate increases sharply and then the rate of growth begins to taper off. Closer examination of the rising edge reveals that the curve beginning is consistent with a double growth curve where the generational curve[7] has an exponent of about three correlating to virus effective reproduction number (R0) three. After the curve peaks, the number of cases per day diminishes asymptotically approaching zero deaths. This is consistent with antigen growth with an exponent of three. The diminished growth rate that halts growth is also with an exponent of about three as would be the case of an antigen that grows exponentially with an exponent larger than that of the virus.

Why is the right side of the curve so different from the left side of the curve? Initial virus growth is explosive as the only limit to growth is its ability to replicate. First, we have to come up with a reason the virus quits growing. The answer is the same in both the case of diminished growth on the left side of the curve and the diminished growth on the right side of the curve.

To aid in this analysis let's make the assumption that antigens can only be created in response to the specific virus that they counteract. The knee jerk reaction is 'of course'. Another assumption has to be that antigen growth rate is necessarily greater than virus growth. Again the reaction is 'of course' but still the reason for the different curve shapes might be elusive. The virus has a head start before the antigen begins multiplying. To slow virus growth, the antigen

[7] The generational portion of the curve is the beginning part when growth is at its greatest.

growth rate has to be significantly greater than the virus growth rate[8]. The author doesn't know R0 for antigens[9] but the ratio of antigen must be capable of growing at a rate greater than the virus and created in the vicinity of the virus.

The long-term implication is that reduction of virus growth (antigen generation) will always be greater than virus growth and probably by a large amount to arrest the growth of the virus.

At this point, the assumption that the curves for infection growth are Gaussian would seem inappropriate. However, if we analyze the rising and falling edges separately, the former seems (and probably is) Gaussian but the later edge is indicative of a linear-time invariant (LTI) system. An LTI system has a depletion function exponentially related to source quantity as would be the case if antigen effectiveness were dependent on virus generation.

To recap:

- When there is no antigen production a virus grows (and spreads) exponentially.
- Antigen growth begins only after there is a virus present (more about that later) and after some delay.
- Virus growth diminishes only when antigen production is greater than virus production.
- When antigen and virus growth rates are equal the curve peaks.
- Since the rate of antigen production is dependent on virus production, virus growth begins to decline as a first order linear time-invariant system thus the shapes of the right hand side of the curves for a single virus/antigen combination will always have the same general shape.
- States with large metropolitan areas have the same curve. This includes New York, Illinois, New Jersey, Massachusetts, Maryland, New Hampshire and Connecticut.

[8] An astute reader will wonder, "How can we be assured the virus can be caught in time?" This is one of those "time is of the essence" situations. If the rate of antigen creation is inadequate, virus growth will continue ad infinitum. With an unhindered growth exponent of three and one generation per hour, two virus cells will grow to 10 billion in three hours and 3 X 10^216 cells in six hours. I am using three as the R0 growth rate.

[9] There is a strong interrelation between the number of virus cells and the number of antigen cells and that is difficult to calculate because the ratio of virus-antigen reactions to total virus cells isn't known to the author.

Non-Urban Spread

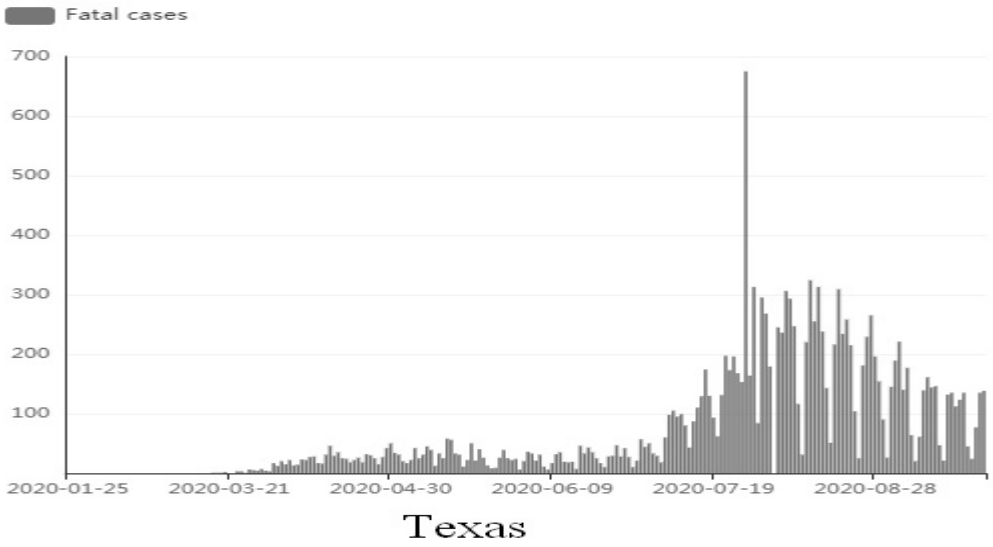

Figure 4 -Texas - Non-Urban spread

Referring to *Figure 5 -Texas - Non-Urban spread*, Texas exemplifies **Non-Urban spread** of the virus. There are very few virus deaths until early July and a peak around early August. Even though Houston, Dallas and Fort Worth are in the state, it is so large that there is still a large non-Urban population. Other states of this type are Arizona, Florida, North Carolina, Tennessee, Alabama, South Carolina, Mississippi, Arkansas, Utah, Idaho, Oregon, West (BG) Virginia, Hawaii and Montana. These states have a small or minimal peak in the early months and a pronounced 'second wave' peak. There is good evidence that this second peak reflects the spread of the disease through the non-Urban areas of those states. These curves have the same characteristics of a fast rise to a flat peak and exponential tail to a minimal death rate.

Mixed Spread

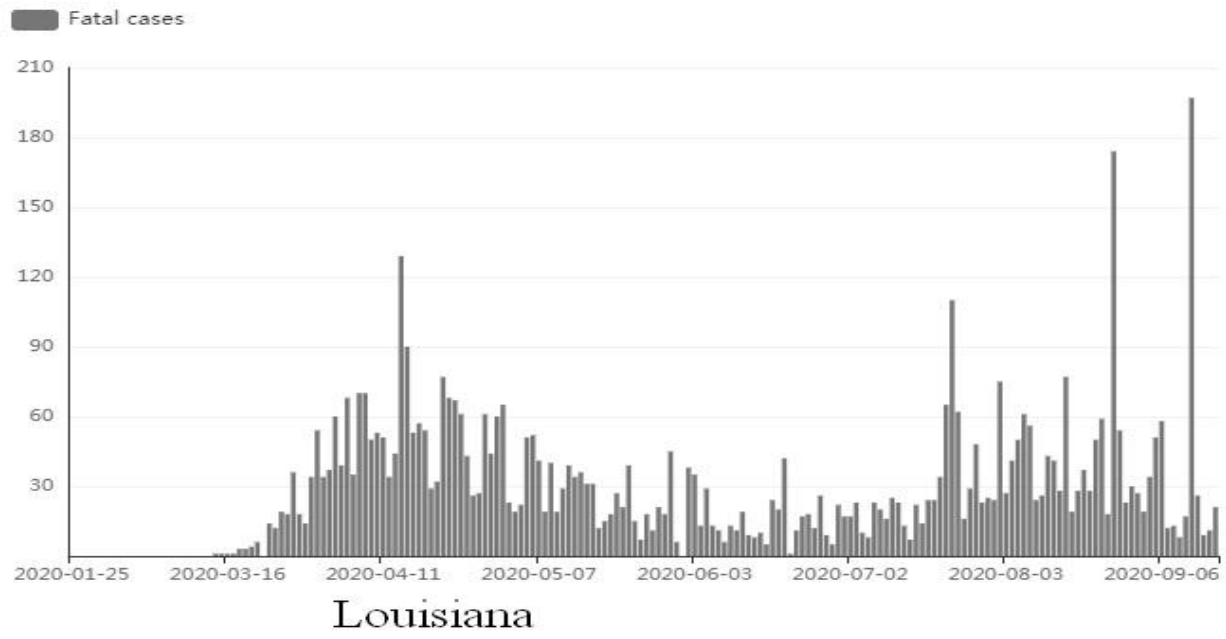

Figure 6 - Louisiana - Mixed Spread

Referring to *Figure 7 - Louisiana - Mixed Spread*, Louisiana exemplifies Mixed Spread propagation. There is an initial peak similar to the Urban spread of New York and there is a secondary peak that is similar to the Non-Urban spread of Texas. This is an algebraic (linear) combination of Urban and Non-Urban spread of the virus. In other words, this is an effect of having the Urban spread and the Non-Urban spread charted simultaneously because they are in the same state. If data for the Urban areas were displayed separately from data in the non Urban areas there would be an early peak in the former and a late peak in the latter.

The Non-Urban spread example (Texas) demonstrates that the Non-Urban build up seems slow. The author theorizes that the buildup in Urban areas (Houston, Dallas and Fort Worth) is like New York, but that cases in the other parts of the state overwhelm the Urban spread data. The slow buildup before the first peak is largely because people with minimal daily contact transmit the virus. Texas is also a commuter state minimizing the effects of mass transit. The lack of mass transit, high rises and large distance also flatten and extend the curve.

For example, Non-Urban populations such as farmers and non-factory workers often go days without leaving their property and even when they leave their property contact with others is minimal. Spread under these circumstances is extremely slow (days or weeks) compared to large Urban areas like New York. Even under those circumstances as the virus builds to a peak, it has

the same quick rise, flat top and slow decline characteristic of Urban spread virus-antigen reaction.

It is notable in that there is a minimum threshold of deaths between Urban spread and Non-Urban spread. This assumed the virus spreads very slowly in semi-rural areas and there are few opportunities for infection because of the small Urban population. You could say that there are possibly three or more curves. That might be true but as with the unclassified states, there are so few opportunities for infection analysis of this type data would be speculative at best.

To recap:

There are two basic transmission rates: Urban spread and Non-Urban spread.

Mixed Spread is a linear combination of Urban spread and Non-Urban spread.

For this paper, remaining states have statistics that are too low to evaluate.

1) The conclusion of this paper is that there is only one peak and it is happening at different times. The author also concludes that this was also true of the 1918 flu.

2) Another conclusion is that the spread is quick through Urban areas due to mass transit and buildings with elevators forcing people into close proximity for extended periods while waiting and certainly inside the elevator. The period of spread is so very quick because the effective R_0 is much larger than 3 (in the case of Covid-19) since an individual can infect many more than three people during a generation[10]. Conversely, as population density decreases so does R_0.

3) A corollary to 2 above is that R_0 is dependent on population as a first order effect. Conversely, propagation speed is a function of effective R_0.

Working with these assumptions, we can characterize rural areas as islands of people connected by relatively scarce opportunities for spread. The spread is quite slow in truly rural areas, quick in cities and towns, and very quick in large Urban areas. For the purpose of visualization, assume a knock down row of dominoes. If we place the dominoes in close proximity, the time between the falling domino and the standing domino is relatively short while dominos that are far apart will take a longer time for the falling domino to reach the standing

[10] There many values thrown about regarding the length of a generation it is difficult to determine but the value of four or five days from initial infection to zero growth. Using initial infection to zero growth is my definition and I'm sure there are other definitions. We do know that Covid-19 infects a person for a number of days. A person is not necessarily a risk to others every day a person is infected. Most calculations take a shortcut and assume R_0 refers to the initial day of infection and/or every person that becomes infected will have the same incubation period. Neither assumption is true; it only simplifies the calculations.

domino and knock it down[11]. If the dominos are set so that each one knocks down three other dominoes, the R0 for domino decimation is 3 just like Covid-19. Multiple knockdowns from a single domino are like an Urban area while one domino knocking down only one widely spaced domino would correspond to rural areas. Imagine a string of single dominoes (rural areas) that suddenly spreads out into multiple column areas (towns) connected to the next town by a single string of dominoes. This is only for visualization purposes.

Can we test this theory quickly? Yes, this would be possible if we could find an area, preferably a country that like New York is mainly metropolitan - Urban and has about the same population. Please refer to the spread curve for Sweden *Figure 6 - Fatalities in Sweden*. I have picked Sweden because it gives us a curve not affected by social distancing, masks or other artificial factors resulting in the natural spread and decline of the disease. Sweden gained public attention for a very short time because they did not take the same precautions as the other industrialized nations such as the United States.

Sweden is a country of 10 million people. Urban areas comprise 87% of the population and the population is one of the oldest in the world with an average age of 41.1 years. It is nearly perfect to compare to New York and to determine the shape of curves in areas without mitigation.

Sweden had 91,000 confirmed cases of Covid-19 over this entire year. The number of fatalities was 5,900. The percentage ratio of confirmed cases to fatal cases is therefore 6.48%. Keep in mind that Sweden has a large average age. The death percentage per capita for this YEAR is 0.0568 %.

New York City has a population of about 8,362,817 people. The City had 246,470 cases of which 23,792 resulted in fatalities. The percentage ratio of confirmed cases to fatal cases is 9.65%. The death percentage per capita is 0.28 % for the entire epidemic.

The table below compares Sweden and New York.

	Sweden	New York	Ratios
Population	10,000,000	8,362,817	83 %

[11] In fact, it is a cosine function with thresholds. There is a minimal distance between dominos such that there is not adequate inertia to affect the target domino as well as a maximum distance whereby the domino won't reach the target domino. I believe that further work will prove that cosine function of dominos also relates to Covid-19. However, it will be complicated by the presence of alternative routes.

Covid-19 cases	91,000	246,470	270 %
Covid-19 deaths	5,900	23,792	403 %
Deaths per infection	6.48 %	9.65 %	148 %
Deaths per capita	0.0568 %	0.28 %	490 %

Sweden's mitigation measures were limited to a ban on gatherings of 50 or more persons, nursing home restrictions and table-only service in bars and restaurants.

Sweden's curves

The Swedish curves are nearly an exact copy of New York's curves. The small number of total cases in Sweden account for the instantaneous variations in the data, but when averaged the shape is very, very similar to the New York curves.

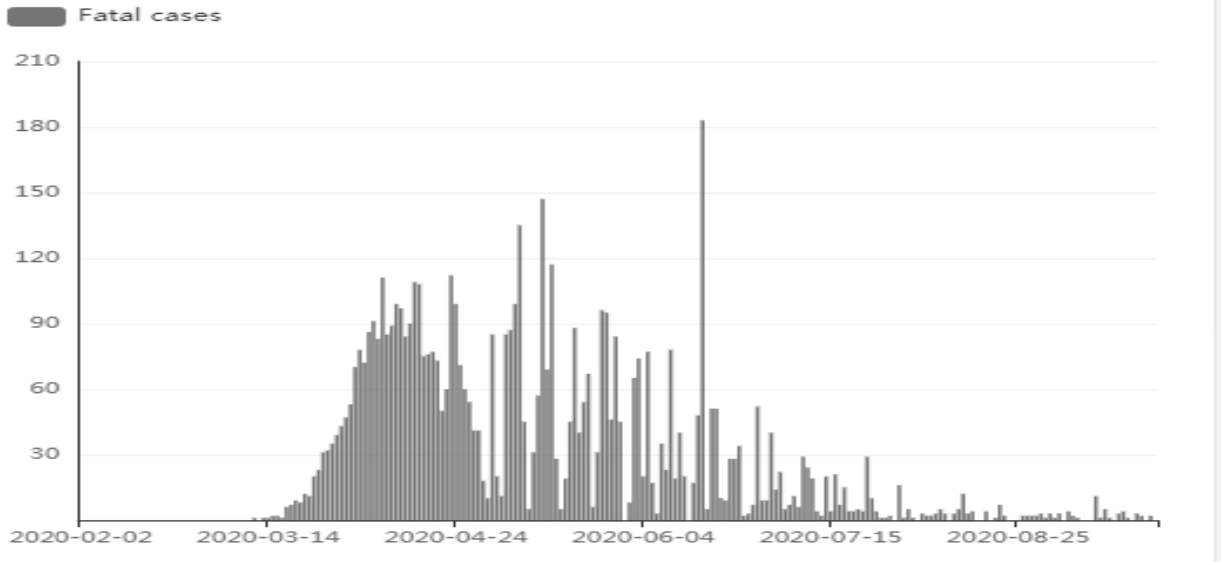

Figure 8 - Fatalities in Sweden

Preliminary conclusions

Referring to *Figure 6 Fatalities in Sweden*, it is appropriate to classify Covid-19 by population density as a first order effect. Large city death rates follow an impulse response curve

with a fast attack (increase) to a peak value that drops in a linear time-invariant manner. Small cities, towns and rural areas probably have the same basic characteristic consistent. However, there seems to be a case that the primary determinant in propagation speed is community characteristics.

This is true with or without mitigation. Sweden had 20% the number of deaths per capita compared to New York City with no government intervention such as shutdowns, mandatory masks or social distancing. If the entire state of New York is included in the analysis the Swedish example still holds true. No government intervention resulted in significantly lower death rates.

In the United States there has been no 'second curve' but rather a delay into other areas of the country that is controlled by variables such as daily activities, modes of transportation and other factors that differentiate Urban area and non-Urban area spread.

A single R_0 value is grossly not adequate for calculating virus dissemination. The R_0 for a skyscraper Urban environment is entirely different than that of automobile oriented Urban areas where there are few elevators.

The R_0 concept was probably 'borrowed' from nuclear science. That methodology is applicable only if all potential initiators (neutrons vs. virions) and targets (U-238 vs. specialized cells et. al) have fixed lifetimes (90 nanoseconds vs. hours to days), always have the same access between initiators and sources and operate in a heterogeneous environment (well defined nuclear reactor vs. the helter-skelter world[12].)

[12] To use the R0 methodology all the areas for virus transmissions would have to essentialy be the same, such as large city, mega cities, towns, agrarian areas, secularly isolated areas, tourist areas and so forth.

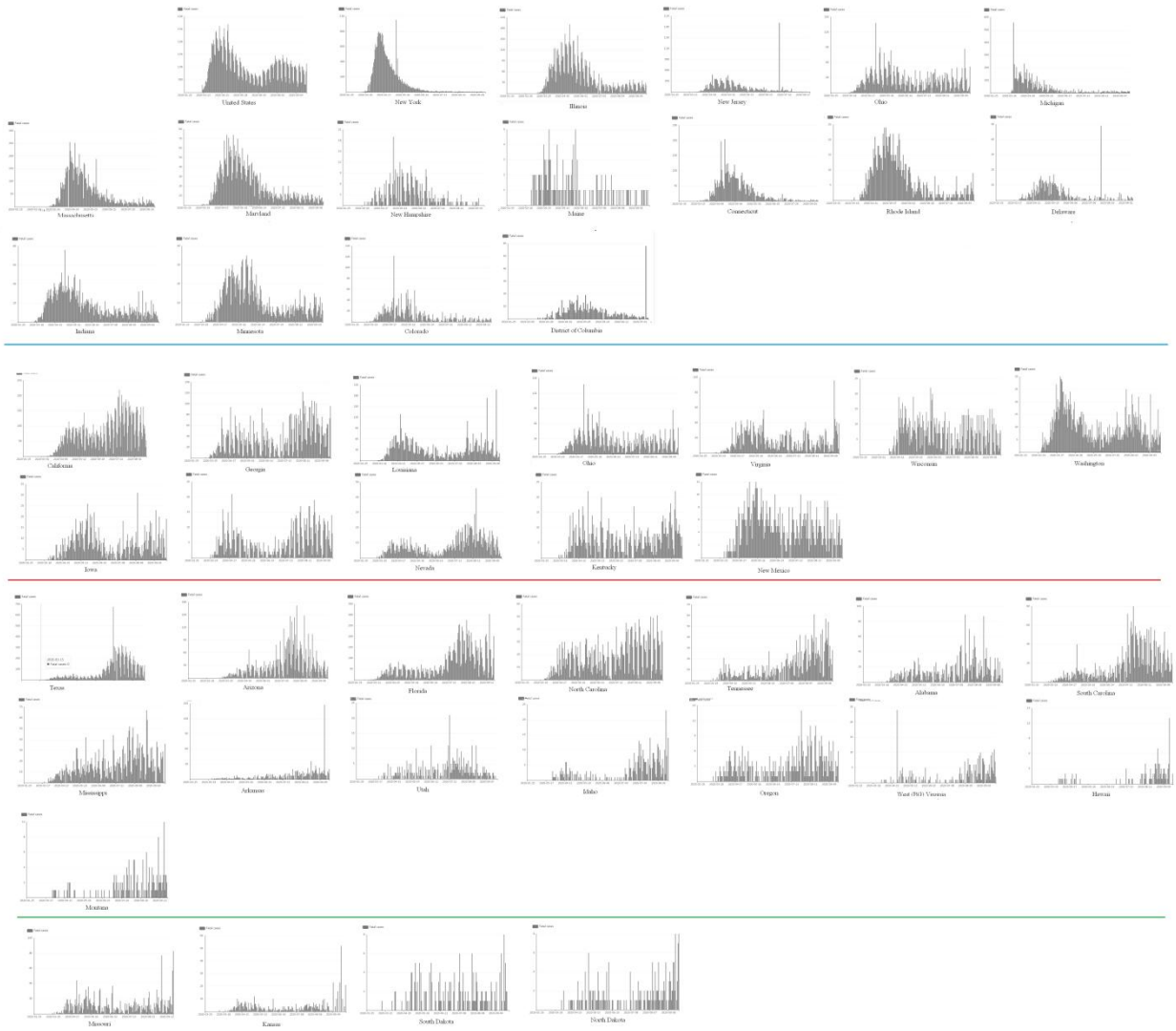

Figure 9 - Daily Covid-19 infection curves

Figure 10 - Daily Covid-19 infection curves shows all the daily infection curves for all the States and Washington DC. I collated the curves first by type and then by total number of infections in the state. Urban Spread is above the blue line, Mixed Spread is between the blue line and the red line and Non-Urban Spread is between the red line and the green line. Below the green line are areas of low statistics but if classified they would probably be mixed or non-Urban spread. In each group, larger populations are first and have the greatest quantity of cases and presumably the best statistics.

Final Conclusions

- There is not a second wave of Covid-19 in the United States and probably were no second waves in the 1918 epidemic. It is not a second wave, but rather a single wave of Covid-19 propagating throughout the country(s). At first glance, the relationship between the maximum heights of peaks A and B in the 1918 Spanish Flu indicates the non-Urban spread was much greater than the Urban spread, which is the opposite of the curves for Covid-19 in the United States. However, the swapping of the curve height proves the theory, because WWI changed the topology of the United States. Beginning the second half of August there was a buildup of soldiers trained and sent to fight in WWI. It seemed the epidemic was gone, but it was traveling through the non-Urban parts of the country. The unknowing military amassed the soldiers in the northeast in preparation for transport. Non-Urban persons not previously exposed to the virus and without immunity were crowded together for training and transport resulting in quick and widespread infections. To compound the situation those infected and too sick to travel to Europe were sent home to recuperate and make place for other soldiers. Their return home increased transmission rates in the non-Urban areas. (This is much like the seniors returned to rest homes in New York.) This also accounts for the huge third curve (C') in the US curves. The effect was of introducing the virus into non-Urban areas and then creating temporary Urban areas that were quickly overwhelmed by the virus.
- The second wave in the 1918 Spanish flu was a result of the war in Europe. If we examine the second US curve (B') relative to the first US curve (A') it is significantly higher than the second Europe curve (B") relative to the first Europe curve (A") and it is. The ratio off B' to A' is greater than B" to A". The theory is that the higher second curve B' is due to soldiers being prepared for transport in the ports of the northeast.
- Assuming this analysis is correct "Mitigation efforts" of the current Covid-19 crisis may have cost tens of thousands of lives by reducing the effectiveness of natural processes such as environmental immunity. The original shutdown was enacted to allow medical entities such as hospitals, clinics and doctors to cope with the huge

influx of cases. This theory of respiratory virus spread would indicate that once the New York crisis was under control, the country should have been opened fully to allow natural methods of immunity to prevail. This is the experience of Sweden and the mortality curves of New York as they compare so well with Sweden. Allowing natural processes to provide immunity in Sweden while protecting the vulnerable populations resulted in a lower mortality rate per capita than New York. Again, I emphasize that the average age of the Swedish population is one of the oldest in the world at 41.1 years.

- Improved practices in the collection of data for epidemics can provide clinical insight if properly collected and evaluated. Now data is logged when it is received creating a two-day lag on weekends and artificially increasing the totals on Monday and Tuesday. This would be minimized by time and date stamping each death as opposed to saying 125 died today in Alabama. The data would then give hour resolution giving detailed information regarding growth rates and latency between locations. Date and time information is ALWAYS available for the death certificate. Analysis is difficult when the peak to valley ratio of subsequent days is as much as 100 to 1 and infinite in some cases. Location information would be invaluable. Each county could provide the GPS location information for hospitals and clinics. Another idea is to pay government sources, particularly county health departments for filling out the forms. They could use the money to help the community on a grass roots level and the compensation would be exactly proportional to the effort required.
- For political reasons, the United States paid Vietnam War debts after the war because no administration wanted to publish the cost. The borrowing process was borrowing by increasing the money supply without increasing the economy. The inflated (less value) dollar made buying a house was nearly impossible because of 22% mortgages. Food prices were raising so fast individuals wanted to buy as much food as possible now because next week's groceries would be even higher. External energy sources became more costly because the US dollar was worth less. Energy, automobiles, and all other imported commodities had to be paid with the inflated currency. The US government raised some war funds by borrowed from overseas sources through the sale of bonds. When the bonds were due, they were paid with interest meaning our

foreign investors such as Japan and the OPEC countries were getting paid in discounted dollars. The dollar took a plunge and there was double-digit inflation in the United States. The recent disbursement of three trillion dollars paid out without gross domestic product coming in is simply lost. The Gross Domestic Product was about $21 Trillion in 2019. The $3 Trillion bail out alone increased the number of dollars without increasing the intrinsic value that money represents which is a 14% decrease in the value of a dollar. If there is another $2 Trillion bailout it would be a 21% decrease in the value of a dollar. This is increasing the dollar supply without increasing goods and services, which is the rote definition of inflation. A secondary factor is that the GDP has reduced by at least $1 Trillion. A tertiary factor is that the United States will struggle to keep its institutions intact with fewer taxes being paid by fewer taxpayers and have to fill the gap by printing more money adding to the devaluation of the dollar. This devaluing discounts the dollar equally for everyone. The entitled, workers, retired and wealthy will feel this pain equally. Consider food. The value of food is the same but the value of the currency is less there for the price will go up. The American experience is that once inflation begins it gains momentum. All these effects will affect the poor and fixed income persons disproportionately.

Recommendations

Whether because of external influences such as political or economic concerns or the rush to provide a useful product in the minimal time, current virus spread simulation software can be categorized as needing improvement. The general understanding is that the original intent was to provide guidance regarding management of health resources.

A completely redesigned system should be designed substituting experienced professionals using tried and true methodology without innovative or imaginative components. Medical doctors are not engineers and should understand they can benefit from the technical expertise of other disciplines.

Any resulting simulator development can proceed only after developing fully transparent specifications and testing regimes with baseline controls. While an open source approach is preferred there needs to be a single non-political, non-economic, non-aggrandizing authority that

verifies released versions of the software are adequate for specific, well-defined and professionally documented purposes.

After the basic problems of data collection and evaluation, the author further suggests an interdisciplinary approach to evaluation of simulator results. Once the core design team develops a stable and reliable tool, users can utilize innovative and imaginative methods similar to those described in this paper to develop new applications up to and including clinical analysis.

Pure conjecture

If true, the narrative above explains nearly all aspects of transportation and transmission of respiratory flu virus outbreaks. We must ask one last question. Why isn't the flu going away in the United States as it did in 1918?

The author believes there is quite possibly another mechanism in play in the body's immune mechanisms. These mechanisms are definitely beyond the knowledge of the author but lay descriptions of the operation of B cells is that B cells combine with Helper T-Cells (differentiate in the literature[13]) to create a plasma cell that has the ability to create huge numbers of antibodies. A search of the literature has not indicated that a B-cell limited to the individual. To state that another way, there is no reason to believe that B-cells are unique to individuals that created them. Consider that persons that are sick or recently recovered are generating B-cells, T-cells and antigens in abundance. Through the process of shedding, the body expels virus cells from the body through the respiratory system. This begs two questions.

1) Do humans shed dead virus cells after recovering from the virus?

2) Are antigen or antigen-related products (such as B-cells and T-cells) shed along with the dead virus products?

The first question is in regard to passing immunity between people. The basic premise for some vaccines is that dead copies of the pathogen will initiate an immune response resulting in individual immunity to the disease. What if virus shed is a mechanism that transfers immunity from infected persons to non-infected person even if there is only a short window of beneficial virus shed production? This might mean that the 1918 immunity was due to exposure to persons recovering from the virus.

[13] https://en.wikipedia.org/wiki/B_cell

The second question is in regard to passing antigens between persons. Let's make the simple assumption that the antigens are specific to a virus but universal to humans. Specifically, what if I can get antigens from you and they work the same as if I produced them? If you think about it for a while, that must be the way the mechanism works or some of the artificially produced (cloned and grown basically) vaccines would not work. The proposed mechanism assumes that future antigen production is dependent on either virus presence or on existing antigens.

Let's assume the body continues to create antigens after elimination of the virus. Isn't that the basic premise of long-term immunity? Any long-term immunity must have a mechanism for long-term reproduction because antigen cells don't last for months or years.

Let's assume the body sheds antigens in a fashion similar to the shed of virus from the body. Let's further assume that the shedding process extends past the time of infection. In case you don't see where I'm going here, there had to be a mechanism to stop the 1918 Spanish flu and the only elephant remaining in the room is the flu itself. More specifically, humans create a super-abundance of antibodies as long as there is any virus around whether alive or dead. The excess antigens are shed through the lungs and back out to other persons giving the gift of immunity.

We have analyzed all the data available in the transmission curves with this last piece of the puzzle. An exponential initial growth of the virus initiates an exponential antibody response quickly overwhelming the virus. The large rate of antigen growth might mean that that after the disease has run its course the infected person can share excess antibodies with uninfected persons and giving person-to-person immunity in the same medium in which it is spread. If so, this would be an extremely viable explanation for self-immunization of the Spanish flu.

Conjecture implications

If the conjecture above is true, isolation and masks would serve to lengthen the duration of the virus because the natural source of human immunity has been impeded. No one wants other people to die but the pain of ripping off duct tape quickly is less than the slow hair by hair tug of a well meaning friend. In math terms, when we shortened the height of the peaks (reduced the number dying per day) we increased the length of the peak. In the case of New York the close proximity assured that the effective R_0 was so high (because of so many contacts of long duration) masks were ineffective. For the rest of the country masks simply impede the flow of natural immunity available from recovering and recovered individuals to the uninfected.

This can be theoretically quantified by comparing the results in Sweden to those in New York.

To be more specific, this pure conjecture is that after a person has the disease they may expel (shed) dead virions, B-Cells, T-Cells and/or antigens which would act as a source immunity for those surrounding them that have not been infected. The stated purpose of the mask is to prevent virus spread, if this conjecture is correct the mask prevents the spread of the cure.

Again all the portion marked conjecture is truly pure conjecture. However, it does give a plausible answer to the question: "Why did Sweden have such better mortality statistics than New York when some epidemiologists predicted total failure of Sweden's methods?" What made the 1918 Spanish Flu go away? Why did Covid-19 go away in Sweden? The charts and statistics are readily available and printed in this work.

Post Script

I am publishing this on my own and no one else is involved. It is quite different from existing theory and predictions and many have staked reputations and futures on other interpretations of the available facts regarding the 1918 Flu and Covid-19 virus.

I've worked on this project since the 1970s and serendipitously found the answers only by trying to make another point. If I'm wrong, the best thing that could happen is that everyone ignores me. But, if I'm right ignore me at your peril.

You see since we have gone so far down this road what should we do? Should we take off masks and initiate block parties? No! Urban areas are probably close to immune. If one looks at the data for a county or city and it has the characteristic asymptotic tail then the disease has run its course. Some people have had not had the choice as to whether to follow the Swedish model or the US model. Most people don't know there are other successful models. It is true that those that followed the US model are getting the same results as the US is getting. If this model is correct places like New York, New Jersey, Connecticut, Rhode Island and Delaware have been through the worst in their cities but need to protect certain groups.

In my opinion, each area should have the choice of making their own decisions. I would like to warn everyone about the 'science believers' with hidden agendas on both the left and right. This is new information and decisions regarding voting shouldn't be made on the basis of past decisions at the federal level.

David L. Hunt

October 2020