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The Omicron Strain: An Overview of Its Growth and Decline in the U.S.

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ABSTRACT

The *omicron strain* was a distinctly new chapter in the battle against COVID for Americans. First identified in Botswana/South Africa, Omicron began circulating in the U.S. population in November 2021, gained dominance, and persisted through 2022 and beyond. Omicron was significantly different in its genetics, lethality, infectiousness, and susceptibility to vaccine intervention, when compared with other strains. It was less lethal than δ because it had fewer L452R S1 protein mutation, and it had an E484A mutation instead of δ's E484Q. Both of these changes made the binding site of the S1 protein (tip of the spike protein) more visible to immune system cells. However, omicron was perhaps 2.5 to 3.8 times more intrinsically infectious than δ, and was probably almost as infectious as measles. Furthermore, omicron was more contagious because it had mutated out-of-range of the original Trump-era vaccine via 28 other S1 mutations (vaccine escape). Essentially, the virus entered a virgin population in the U.S. This fact, combined with the uncontrolled movement of people within and into the U.S., a population that refuses to accept public health measures, and a generally somnolent response to COVID by the U.S. government, all took their toll on the population. For the δ-omicron dominated time interval from Aug. 15, 2021 to June 28, 2022, calculation suggest 54.3% of death (215,428) were due to seasonal temperature related effects, 28.6% (113,383 deaths) were due to domestic travel, and 17.1% (67,860 deaths) were due to medically unscreened migrants entering the U.S.

Keywords: COVID-19, omicron, cladogram, genetics, vaccine escape, migration, Fourier transform

Introduction

This publication concentrates on the time period from the end of October 2021 to June 2023. On Nov. 15, 2021, omicron appeared in the U.S population.[1] By Dec. 25, 2021, 58.6% of cases in the U.S. were omicron.[1] Between Dec. 25, 2021 and omicron's early spring decline (Apr. 24, 2022), 179,130 Americans died (most from omicron).[2] However, that wasn't the end. A second late spring to fall omicron wave followed, and by Nov. 25, 2022 another 81,959 Americans perished. Although this second omicron wave was much smaller in amplitude than the primary peak, it was also much wider in time; and much more significant than the first and second waves of the pandemic as a whole. Finally, a third omicron peak, with an even smaller amplitude, appeared at the end of 2022, and by Feb. 20, 2023 there were an additional 36,209 casualties. Total omicron deaths amounted to about 297,298 deaths, about 26% of all COVID deaths up to Feb. 20, which continued to grow! The original Trump-era vaccine was largely ineffective against this new biological terror,[1,3] contrary to "expert" opinion.[4,5] By August 31, 2022 the U.S. FDA *finally* authorized the use of an updated vaccine booster specifically targeting omicron[6] – *nine and a half months after the first detection of the omicron strain in the U.S.!* So, what went wrong *this* time? The answer is, in a few blunt words: *genes, uncontrolled migration, human factors, and public policy!* Each factor will now be discussed in detail.

The Cladogram, Genes, and Vaccine Escape

The first step in understanding omicron is to understand how it is genetically related to other COVID strains. Those strains for which there is clear evidence available indicating a significant impact on transmissibility, severity, and/or immunity (i.e. *Variants of concern* or VOCs) for the U.S. will be emphasized. Figure 1 below is called a *cladogram.* It has been simplified for clarity, but the basic relationships have been preserved. COVID entered the U.S. from two directions; on the west coast from China, and the east coast from Europe. On the west coast, an infection was introduced into Washington state by a traveler to China. However, this infection was *believed* to have been contained by the most strenuous efforts of the public health community. Although the media has done a good job of spreading the rumor that the COVID pandemic in the U.S. was ignored during the Trump presidency, the truth is exactly the opposite.[7] On the east coast the story is more complex. Starting from China as strains A and B, COVID then appeared in Pakistan as strain B.1 (defined by the mutation D614G – the nomenclature will be explained presently), then England as strain B.1.1.7 (which is called α for short). Alpha is also believed to be the first strain firmly established in the U.S., supporting an east coast point of entry, but other descendants of B.1 soon followed (Figure 1). The U.S. strains are all traceable to "branch" B.1 of trunk B of the COVID "family tree". The ε strains (B.1.427 and B.1.429, the 427th and 429th *named* descendants of B.1) are two "leaves" on B.1 that caused massive outbreaks in California. Delta (δ or B.1.617, the 617th descendant of "branch" B.1) is another "leaf" that produced many sub-strains; evolving, of course, in a very different direction from ε. The same is true for the β strain (B.1.351), the 351st descendant of "branch" B.1. The strains ε, δ, and β are relatively distantly related VOCs. However, α, γ, and ο are different. These genetically closely related strains are like three "leaves" on one "twig" (B.1.1) of "branch B.1; where α (B.1.1.7) is the seventh descendant of "twig" B.1.1 and was discovered near the beginning of the pandemic – hence the small final integer, γ (B.1.1.28, also denoted by the alias P for short) is the 28th descendant of "twig" B.1.1, and ultimately ο (B.1.1.529, called omicron) is the 529th descendant from "twig" B.1.1 and was discovered near the end of 2021 – hence the large final integer. The VOCs α, γ, and ο are more closely related than the previous triplet because the first *two* integers following B are 1's. This system of classification is called the *provisional PANGO* (Phylogenic Assignment of Named Global Outbreaks) *system* and, like the cladogram itself, is based on the gene nucleotide sequence of the all-important spike (S)

protein; which projects radially above the surface of the coronavirus outer envelope and allows the virus to attach itself to its cellular host.[8] The initial letter (B in this case) is called a *prefix*, and the trailing integers separated by decimal points (that mean "descendant of") are called a *suffix*.

However, by July 8, 2022, the omicron strain had itself split into many sub-strains. There was BA.1, BA.1.1, BA.2, BA.3, BA.4, and BA.5. What do all these double letter prefixes mean? In order to keep PANGO names from becoming too long, "aliases" are used. An example of this was the letter "P" used to represent B.1.1.28 (or γ) above. There are other aliases as well. The capital letter C ≡ B.1.1.1, and the double letter BA ≡ B.1.1.529 (or ο), so that BA.1 is the first daughter (sub-strain) of ο, while BA.1.1 is ο's granddaughter or sub-sub-strain. BA.2 is a sister strain to BA.1, and "BA.2.75" (the 75th named descendant of BA.2) is called "Centaurus".[9] Continuing through the alphabet, there is BQ.1 and its descendant BQ.1.1; both circulating on Apr, 2023.[10] When the second letter gets to Z, the first letter will have to be changed to the next available letter in the alphabet. The letter X is special, and is reserved for *recombinant strains* that combine, or exchange, genetic material from *two* or more parent strains (reassortment); so, there are the prefixes XD and XBB. In this case, the letter prefixes have nothing to do with ancestry, but reflect the *order of discovery*, and are assigned the next available letter or combination of letters. XA and XB cannot be used for COVID because A and B are used as "trunk" identifiers. The letter C cannot be used either, because C is an alias. The next available letter is D – hence XD. Double letter aliases up to AY and AZ have been taken as well.[11] BA is also taken, so XBA wouldn't work either, but XBB is allowable. Recombinant letter names can be used by themselves, like "trunk" letters, but aliases must always be followed by a decimal point and an integer. By Apr. '23, near the end of the time interval covered by this publication, the dominant COVID strains nationwide were all recombinants (87.9% XBB.1.5, 4.6% XBB.1.9.1) retaining fragments of ο in their genome.[10] However, some BA.5, BQ.1 and XBB.1 was still circulating. Hopefully, this brief summary takes some of the mystery out of PANGO names that are even confusing to biologists. The reader may have seen some of these PANGO designations in the newspapers, but they are never explained. Primarily because the journalists don't have the foggiest notion of what they mean, but they sound scientific, so PANGO designations sometimes wind up in the popular press. The author is reminded of a line from an English nursery rhyme, "even fleas have fleas that bite their backs"!

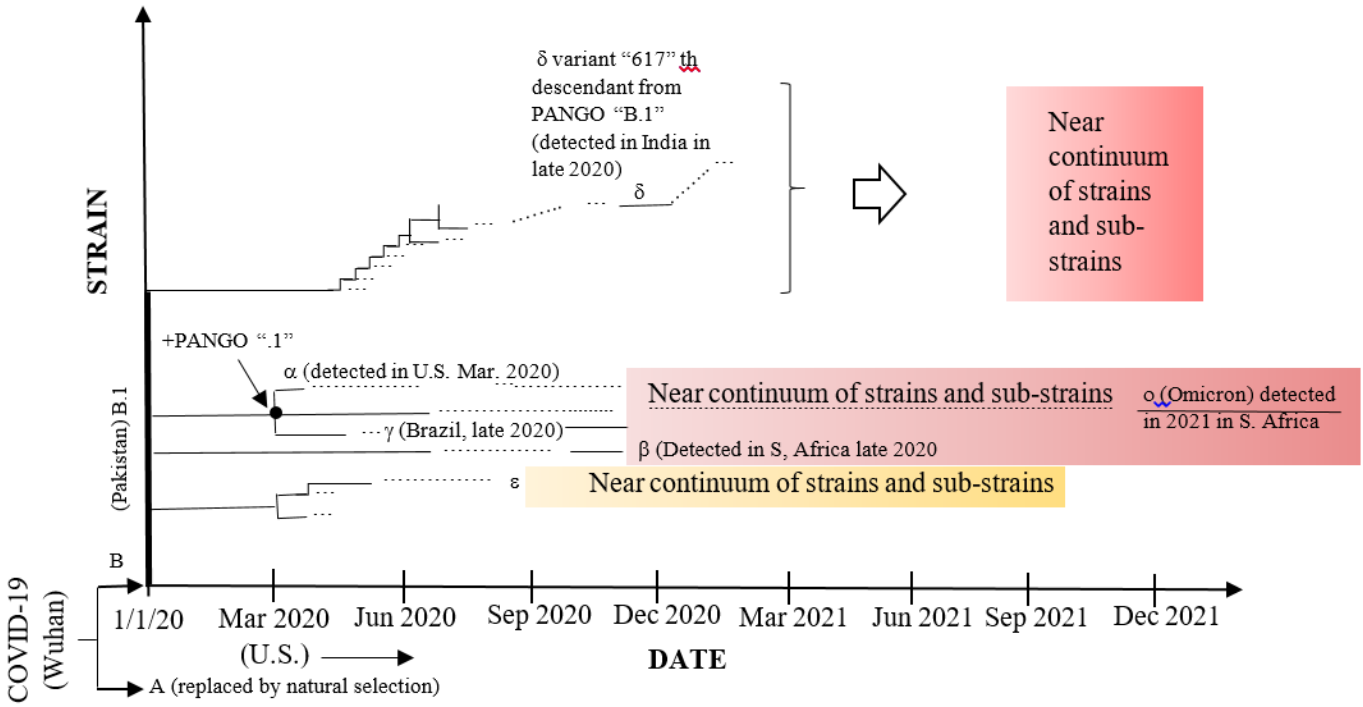
Each PANGO designation corresponds to a unique set of gene nucleotide mutations from the B "trunk" which, in turn, can produce a set of mutations in the amino acid sequence of the S-protein. Even a single point mutation (i.e. an error in a single nucleotide) in the coronavirus RNA genetic code can result in the replacement of one amino acid, of the chain of amino acids that form a protein, by another.[12] Such replacements can alter the ability of antibodies to bind to, and neutralize, the S protein; the virus' key into the interior of a host cell.

Here the author must make a brief digression into protein mutation nomenclature before proceeding. Protein mutations are identified by a code of the form X###X′, where X is the one-letter symbol for an amino acid *before* mutation, X′ is the one-letter symbol for the amino acid that X was changed into *after* mutation, and ### is a number that specifies the position of X in the amino acid chain that comprises a protein (starting from the first amino acid called the 5′ end of the viral RNA, or vRNA). The one letter symbols for each of the 20 amino acids found in human proteins are as follows: A = alanine, R = arginine, N = asparagine, D = aspartic acid, C = cysteine, E = glutamic acid, Q = glutamine, G = glycine, H = histidine, I = isoleucine, L = leucine, K = lysine, M = methionine, F = phenylalanine, P = proline, S = serine, T = threonine, W = tryptophan, Y = tyrosine, and V = valine.

The coronavirus spike protein is actually made up of two sub-proteins called S1 and S2 that are bound together to form S, and it is the S1 protein at the

tip of the spike that is most important in binding to a host cell. A few important omicron S1 mutations relative to other strains are as follows:[13] the δ L452R mutation is usually much less common for ο (globally 0.5% - 30% depending on locality), δ E484Q has become ο E484A (the 484th amino acid has a history of change that began as β E484K), δ P681R has become ο P681H, T95I is unique to both δ and ο, G142D was a δ mutation that has been lost in ο due to deletion of the 142nd amino acid, the same is true for V70F (the 70th amino acid has been deleted in ο) while the D614G mutation has been conserved by B.1, α, β, γ, δ, and ο! There are two ο mutations that are shared by δ and γ respectively; T478K and H655Y. Finally, there are many mutations that are unique to ο, these include deletion of amino acid 69, deletion of 142-144, A67V, Y145D, S371L, S373P, S375F, N440K, G446S, S477N, Q493R, G196S, Q498R, N501Y, Y505H, N679K, N764K, D796Y, N856K, Q954H, N969K.[13]

Figure 1 – The greatly simplified "cladogram" is a "Family Tree" for COVID VOCs in the U.S. The black dot is called a "node" and is needed to mark the separation point of the closely related variants of concern α, γ, and ο.[14]



In many ways, the clinical properties of the δ and ο infections can be understood together in terms of these mutations. Delta's L452R and the E484Q mutations bracket the binding site of the S1 protein.[12] Furthermore, arginine (R) of the L452R mutation is a large bulky amino acid to which one or more sugar molecules can be attached.[12] Sugar molecules can also attach themselves to glutamine (Q) of the E484Q mutation. This variable stereochemistry interferes with the binding of antibody molecules (induced by the original vaccine) to δ's S1 protein binding site, and it also makes the S1 binding site less visible to white blood cells because of its cloak of harmless sugar molecules. The cells of the human immune system normally do not recognize sugar as anything dangerous, because our body needs sugar for energy, thereby delaying the immune response. Omicron, however, has many fewer L452R mutations than δ, and that makes ο intrinsically less lethal than δ because a person that has been infected will develop natural (not vaccine induced) antibodies sooner, and they will fit the S1 binding site better. Furthermore, the Q of the δ E484Q mutation has been replaced by A in omicron. Sugar cannot attach itself to alanine (A). Therefore, the Q → A transition leaves the S1 protein binding site less disguised, again *making ο*

less lethal than δ. Although ο is less lethal, it is also more resistant to (the original) vaccine induced antibodies because D614G is the only mutation that was conserved from α by ο. The original Trump-era vaccine was designed for Wuhan (B) and α, but the antibodies that the human body produces from α-RNA fragments do not take into account the other 29 mutations (listed above) that occurred in the ο S1 protein. Again, the result is that antibodies induced by the original vaccine cannot bind well to ο S1. Serious disease might be prevented, but many virus particles (perhaps most) will escape complete neutralization. It is a lot easier for a vaccine to prevent serious disease, than all disease.[12] Essentially, ο was a new disease not covered by the original vaccine formulation; just what most biochemists had feared.[15] It is fortunate that ο was less lethal than δ because that gave the human body time to manufacture antibodies that were appropriate to this new strain – usually! The first up-dated Biden-era vaccine took into account all of omicron's 30 mutations and, if the population could have been relied upon to cooperate (it didn't), might have been theoretically capable of eradicating omicron; since preliminary trials by Pfizer indicated "that adults older than 55 had a four times greater immune response (four times the antibody levels?) compared with the original booster".[16] Furthermore, Moderna claimed their new (bivalent or two stain, α plus ο) booster "induced significantly higher neutralizing antibody titers against BA.4/BA.5," compared with the original booster.[16] Significant progress had been made, but an important question must now be asked. The dangers of *antigenic drift* (accumulating antigen [S1] changes caused by 'minor' genetic mutations) and *antigenic shifts* (major antigen changes caused by genetic reassortment), that can render a COVID vaccine formulation ineffective, were well known.[15] An annual booster *with an up-dated COVID vaccine* seemed advisable, judging from the rate of decay of antibody levels in patients and the rate of COVID mutation.[15] The first booster approved by the U.S. government was *not* δ-specific, and it was clear from the lower vaccine effectiveness against δ (only 80% vs. 95% against α), that the L452R and E484Q mutations were impeding antibodies induced by the old vaccine formulation from intimately binding to the coronavirus S1-protein.[12] The original vaccine was becoming obsolete. Clearly, a new (second) booster was going to be needed that was specific to the then newly emergent omicron strain.[12] Why did the U.S. government wait so long before approving the second (updated) booster? If they had not been so lethargic, many of the almost 300,000 deaths due to ο could have been avoided.

At this point it is natural to ask, "What about delta-omicron hybrids? Are they dangerous?" Meet the *Deltacrons*,[17] the recombinants tentatively assigned the PANGO lineage XD. Technically, XD has genes from ο BA.1 and a δ strain that is *similar* to B.1.617; hence the new letter D, with the X denoting recombination, as previously discussed. "Similar" means that some of δ's mutations are missing, while other new ones are present.[18] This new δ strain has been given the alias AY.4. XD's most characteristic S1 protein mutations are Y145H and A222V. XD is known to exhibit immune-escape properties (vaccine resistance) similar to ο BA.1.[19] It was feared that XD might combine the worst properties (lethality and transmissibility) of δ and ο.[19] There was a popular rumor circulating at the beginning of 2022 that "diseases must mutate to less and less lethal forms because it is not in the interest of a strain to kill-off its hosts". This is not necessarily true. Evolution is only concerned with *maximizing the number of progeny*. A virus kills a cell because so many daughter virus particles are produced that the cell eventually ruptures! The process is called *lysis*; and if enough cells die, the host will die also. This is what happens to AIDS patients. The alternate strategy is to produce less progeny per cell, avoid lysis, thereby enhancing host survival, and go on to infect another host. The strategy that produces the *most progeny* overall will be the one that becomes dominant in the long-run. It is not necessary for lethality to decrease monotonically to zero!

Another rumor circulating during the winter of 2022 concerning viral evolution was that "vaccines are causing the coronavirus to mutate". The truth is exactly the opposite as Anthony Fauci correctly explained, "Viruses don't mutate unless they replicate, and if you can suppress that by a very good vaccine campaign, then you could actually avoid this deleterious effect that you might get from the mutations."[20] The whole purpose of a vaccine is to inhibit viral replication.[12,15]

One final question remains, "Will there ever be an 'omega-strain'?" That is to say, given that coronavirus strains from $\alpha \rightarrow o$ progressively evolved out of range of the original vaccine, can a strain evolve that is immune to *all* vaccine intervention? Some microbes take aggressive action in their own defense. Gonorrhea is an example of the latter. This diplococcus (a bacterium) has defied all attempts at vaccine intervention because its genome encodes for a *protease*, an enzyme that cuts-up and neutralizes antibodies (which are proteins).[21] Some researchers are critical of the money spent to fund such research. However, microbes have diverse ways of sequestering genetic material from other microbes. If something really dangerous, like tuberculosis, should acquire a similar genetic sequence – heaven help the human race! The research money is well spent. Can the coronavirus ever develop, or sequester, such a sequence? The author can only say that having large unvaccinated segments of the population just encourages mutations as previously explained.

Multi-Year (Long-Term or Secular) U.S. COVID Behavior

Counting the dead is never pleasant. Yet, the diagnosis of death is certain, and offers a reliable statistic. No one ever recovers from death, and then walks away never to report the incident. However, the *cause* of death is more uncertain, since underlying factors may be involved, giving a false impression of virus lethality. Figure 2, shows total U.S. deaths as a function of time. The trend has been linear over the time interval covered by this report, although there seems to be a change in slope after Feb. 8, 2022. This change is probably due to a combination of better hospital care, new post-infection therapeutics, the lower lethality of omicron, and a drop in the number of active cases (Figure 3). Simultaneously, there has been a decrease in public cooperation concerning social distancing, mask wearing, and even acceptance of the updated booster in the fall of 2022. On Sept. 23, 2022, the Associated Press reported that only 4.4 million doses were administered even though the United States government ordered 171 million shots.[22] Many will go to waste. Those who received the old boosters, but refuse the new ones, will be unprotected since the original booster formulation was relatively ineffective against o, and whatever immunity it produced against the older strains had waned.

The situation concerning active cases in the U.S. (Figure 3) is more complex, but this *waveform* can still be understood in great detail, as will be demonstrated. There seems to be a roughly annual cycle (12 ± 2 months) that starts with a relatively small peak, followed by a larger peak, followed by a crash, followed by the beginning of a rise to the next cycle. Therefore, the 2nd wave was followed by the 3rd wave, which was followed by a crash, which was followed by the early flank of the δ wave. In the next cycle the δ peak was followed by the $o1$ peak, which was followed by a crash, which was followed by the early flank of the $o2$ peak. Each "annual" cycle seems to go from summer to summer, but they are not all exactly of the same length due to temperature irregularities from year to year, changes in the migration patterns of sick refugees due to political changes within the U.S., the season when an infection enters the U.S., and many other factors. More will be said about this in subsequent sections. By late December (2022) the $o2$ peak has passed. Everyone was bracing for the "main" ($o3$) peak. However, according to "official" statistics,[2] the $o3$ wave was a relatively minor feature – *only* 36,209 deaths.

Finally, it should be noted that from the beginning of the pandemic in the U.S. until about Jan. 31, 2022, active cases in the U.S. seemed to rise linearly on

average, like total deaths, with active case peaks rising above this "trend line" while active case crashes fell below it. The cause of this linear trend with positive slope was probably due to the onset of new infection waves within the U.S. before the previous wave (or waves) had completely finished. The result was a more or less steady buildup of active cases; an "*infection reservoir*". However, omicron broke this trend because it was so infectious that it essentially depleted (for a while) the population of susceptible hosts all at once. When they recovered (or died), the number of active cases almost fell to zero (final base-line). To give the reader a more mechanical feel for how this type of active case crash works, a mathematical demonstration will be provided in a subsequent section. Therefore, from Jan. 31, 2022 to Apr. 19, 2022, a new trend with a negative slope (a "de-trend") was in place. After Apr. 19 to June 28, 2022 the active case curve seemed to sit on the final base-line.

Figure 2 – Total U.S. deaths vs. time.[2] The death rate in the U.S. has been fairly constant at about 1% throughout the pandemic.[12,23]

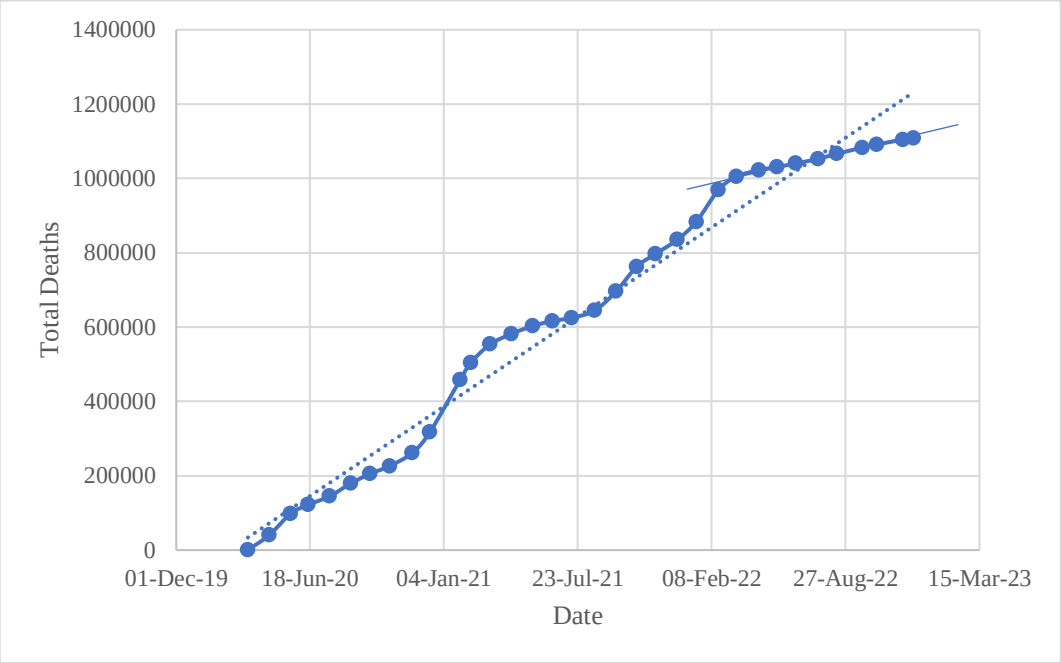
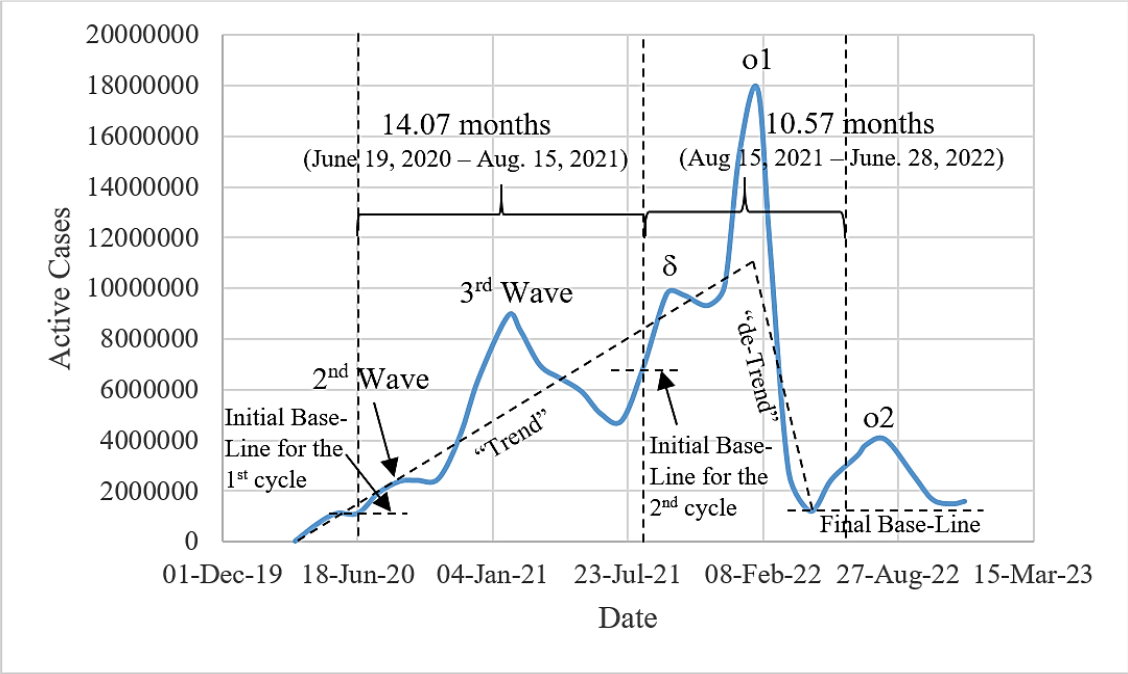


Figure 3 - Active cases (U.S.) vs. time.[2] Since there are multiple o peaks, they will be denoted serially by o1, o2, etc. This waveform is rather complicated, but it can be decomposed into understandable pieces.



"Annual" (Cyclical) U.S. COVID Behavior

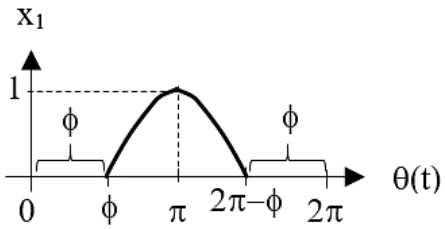
Environmental Factors that Influence Microbial Growth: Temperature, gas requirements, pH (acidity), moisture, osmotic pressure (solute concentration), light (visible and UV), and hydrostatic pressure (for deep sea *barophiles*) all influence microbial growth. However, temperature will be the most important factor for growth of the coronavirus in a live host, and its spread to other hosts. There are three different types of temperature sensitive microbes: *psychrophiles* (that thrive at temperatures below 15^0C), *mesophiles* (that thrive between 20^0C to 40^0C), and *thermophiles* (that thrive at temperatures greater than 45^0C). Of these, it is the mesophiles that are most important medically because they can survive in the human body. It is common knowledge that colds and the flu are primarily a winter (*seasonal*) phenomenon. Why? The common cold is caused by about 200 different rhinoviruses (especially) and coronaviruses (a few). That is why a vaccine has never been developed for the common cold.[21] Rhinoviruses only multiply successfully in tissues that are slightly below the human body's normal *core temperature* of 37^0C (i.e. in the 33^0C to 35^0C range).[21] Therefore, they only multiply in the extremities of the body on a cold day. However, if they get too cold they will become inert. That is why colds are passed from *person to person*, and are not caught directly from the environment on a cold day. Cold temperatures also tend to drive people indoors, making person to person contact more likely. Although many experts consider the latter behavior to be the principle cause of the winter cold and flu season, it should be noted that in the southern part of the U.S. people are more likely to be indoors during the *summer* to escape the heat by remaining in air-conditioned spaces. In this last case, human extremities (hands, fingers, toes, etc.) are subject to either hot (outdoor) temperatures, or moderate "shirt-sleeve" (indoor) temperatures such that core temperature (37^0C), or higher, can be maintained throughout the body at all times, making the development of active infection less likely. Animal hosts (migratory and domesticated) may also work as intermediate carriers in place of people. Infections spread by *animals* are called *zoonotic infections*. This is especially true of influenza. So, there is the Bird Flu, the Swine Flu, etc.[24] With this background, mathematical models can now be constructed for the annual patterns of coronavirus infection caused by *seasonal temperature changes, person to person contact (both foreign and domestic), and animals*.

Mathematical Models for Annual Infections: In this section the microbial growth factors above will be modeled by a linear combination of sine functions to mimic the "annual" cyclic behavior of the pandemic in the U.S. For this reason, the mathematical model for an "annual" infection cycle will be called a *Fourier expansion,* (or *Fourier decomposition,* or *Fourier sine series*) of the number of active cases as a function of time.[25] Furthermore, this linear sum will also be multiplied by a "ramp function", like the dotted lines in figure 3, to capture the buildup and decay of the infection reservoir (background of active cases). The word "annual" has been enclosed by quotation marks because each infection cycle varies somewhat in duration from year to year (see Figure 3). However, *on the average*, the cycle of infection must be 12 months long. Because of this cyclic temporal variability, time will be measured as an *angle* (θ), where zero radians will be identified with the commencement date (typically close to the summer solstice) of a particular cycle and 2π radians will be identified with the completion date of that cycle (also proximal to the summer solstice). Therefore, θ is a function of time t, written $\theta(t)$.

Analysis will begin with a model for body-temperature / indoor-contact effects (thermal effects). During the summer months, the number of colds, flu, and COVID, is generally low relative to the winter as discussed above. Let

$$x_1 = \begin{cases} \sin\{[(\theta - \phi)/(\pi - \phi)][\pi/2]\} & \phi < \theta < 2\pi - \phi, \text{ where } 0 < \phi < \pi \\ 0 & 0 \le \theta \le \phi \text{ or } 2\pi - \phi \le \theta \le 2\pi, \text{ where } 0 < \phi < \pi \end{cases} \quad (1)$$

Figure 4 – x_1 as a function of $\theta(t)$. The seasonal contribution to the infection cycle due to thermal effects.



Then, as a simplest model in accordance with Occam's Razor, the number of active cases during an "annual" infection cycle due to thermal effects will be proportional to x_1. Here, ϕ will be called the *climate phase angle*, and represents that warm part of the year (expressed as an angle between 0 and π radians) during which "colds" are unlikely. The climate phase angle is a function of where you are on the Earth's surface. It can be affected by proximity to the ocean, elevation above sea level, and especially latitude. Near the poles ϕ is very small, so that coronavirus infections may occur throughout the year. Whereas near the tropics ϕ may be so large that it approaches π radians, in which case x_1 is always zero (i.e. there is no thermal component to the infection cycle). Since the U.S. is in a temperate latitude, ϕ will be taken to be 90^0, or $\pi/2$ radians; meaning that coronavirus infection is unlikely (or at least less likely) for the quarter of a year from the summer solstice to the fall equinox, and from the spring equinox to the next summer solstice. Notice that as $\theta \rightarrow \pi/2$, $x_1 \rightarrow 0$ because $\sin\{[(\pi/2 - \pi/2)/(\pi - \pi/2)][\pi/2]\} = \sin(0) = 0$. Similarly, as $\theta \rightarrow 3\pi/2$, $x_1 \rightarrow 0$ because $\sin\{[(3\pi/2 - \pi/2)/(\pi - \pi/2)][\pi/2]\} = \sin\{[(2\pi/2)/(\pi/2)][\pi/2]\} = \sin(\pi) = 0$. Furthermore, at $\theta = \pi$, $x_1 = \sin\{[(\pi - \pi/2)/(\pi - \pi/2)][\pi/2]\} = \sin(\pi/2) = 1$, as expected (see Figure 4). In reality, there will always be a baseline of infection that will be addressed later.

Next, consider the spread of infection caused by travel. There are two components associated with this process. The introduction of infection into the U.S. from external sources (especially illegal immigration), and the growth of the domestic infection reservoir due to travel within the U.S. First, illegal immigration will be considered because many migrants are infected by the time they reach America's southern border, and they cannot be stopped unless arrested.

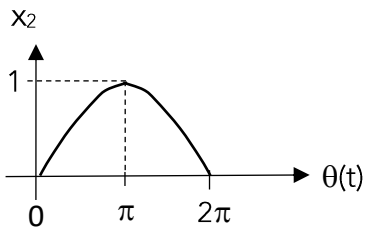
Crossing the southern border of the U.S. is not as easy as it might seem. The Sonoran and Mojave Deserts are barriers in and of themselves. During the summer months, temperatures in and around Yuma, Arizona, a popular illegal immigration corridor, can soar to 124^0F (51^0C), with an average high temperature in July of 110^0F (43^0C). The record high for April is 109 ^{0}F. Phoenix, Arizona (100 mi. north of where the author lives) is close behind. These are the "official" temperatures. However, the author can state from first-hand experience that the actual figures on the ground are higher. Therefore, migration is most practical during the cool fall and winter months between the equinoxes. While the 3 months before and after the summer solstice are to be avoided. This trend was clear in 2022.[26] Some of those who attempt the crossing on foot during the warm months will perish. However, the availability of air-conditioned four-wheel drive vehicles makes year-round migration feasible. Furthermore, there has been a steady quasi-linear increase in the number of migrants intercepted by homeland security over the last few years.[26] This trend, which contributes to the linear increase in the size of the infection reservoir in the U.S., is superimposed on the seasonal trend and will be addressed in more detail later. For now, it is only the seasonal trend that is of concern. Let

$$x_2 = \sin(\theta/2) \quad . \quad (2)$$

Then, as a simplest model, the number of active cases during an "annual" infection cycle due to illegal immigration will be proportional to x_2. When

$\theta = 0$ (or 2π) radians, typically corresponding to some date in the summer (e.g. the summer solstice), $x_2 = 0$ (i.e. there are no cases associated with those two days because anything proportional to zero is also zero). However, when $\theta = \pi$, typically corresponding to some date in the winter (e.g. the winter solstice), $x_2 = 1$. That is to say, x_2 will have its maximum value because the sine function has its maximum value at $\pi/2$ (Figure 5). Except for the two end points, x_2 is never zero. This is as it should be since there will always be a few infections due to illegal migration.

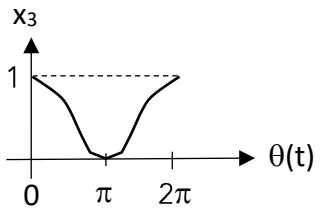
Figure 5 - x_2 as a function of $\theta(t)$. The seasonal contribution to the infection cycle due to illegal immigration.



Next, the spread of coronavirus within the U.S due to domestic travel must be addressed. As everyone knows from the variations in the price of gasoline, most domestic travel occurs during the summer months.[27] Therefore, the potential for spreading COVID via *domestic travel* will be highest in the summer, less in the fall and spring, and least in the winter - the opposite of what one would expect due to thermal effects or illegal immigration. Let

$$x_3 = 1 - \sin(\theta/2). \qquad (3)$$

Figure 6 – x_3 as a function of $\theta(t)$. The seasonal contribution to the infection cycle due to domestic travel. Notice that x_3 is zero only at $\theta = \pi$.



Then the number of active cases during an "annual" infection cycle due to domestic travel is proportional to x_3. Again, only general *seasonal* effects are of concern here, and great accuracy is not required up to this point. The secular growth in travel, and the baseline of infection due to mid-winter travel will be addressed later.

At this point in the author's narrative, it is appropriate to debunk a common myth. There have been persistent rumors that the COVID pandemic was caused by a leak in Chinese laboratory.[28] The Wuhan Institute of Virology,[28] the Zhongnan Hospital,[29] and even Chinese weapons laboratories have been targets of such accusations. There is a negligible probability that any such claims are true, no matter how "official" they may sound.[29] The pandemic originated in the Wuhan Seafood Market (Huanan Market). Epidemiologist know this for several reasons. First, half of all the initial cases involved people who worked in the market. Second, as the pandemic spread in Wuhan, statistical analysis showed the market to be the epicenter of the infection with a very high probability (>99.9%). Science is very lucky to have such detailed records. It is unique in the annals of medical history. Third, sequenced genetic material recovered from the market showed that cages within the market that held live mammals were most contaminated, as well as carts used to carry those cages and a de-feathering machine. So, not only do epidemiologists know that the Huanan Market was the source of the infection, they even know that the western part of the market, where the live animals were kept, was the point of origin where COVID (probably a descendant of wild bat coronavirus RaTG13 originally) was transferred from food animals to human hosts.[29] Furthermore, a laboratory source is not credible because the first cases to appear in laboratory workers were *after* those that occurred at the market. There are many people, including some politicians in Washington, who would like to blame the Chinese government for the pandemic. However, this is just an attempt to discredit the nominally communist *junta* in Beijing. The paths of error are various and infinite.[30] Since most Americans do not come in contact with bats or "wet markets", zoonotic infection will not be considered to be an important component of the

COVID-19 pandemic in the U.S., and will not be modeled.

Finally, the growth and decay of the domestic infection reservoir must be modeled. As previously mentioned, the onset of new infection waves prior to the complete extinction of previous waves is probably the primary cause for the build-up of active cases. However, the simple superposition of tails and peaks of different waves do not tell the whole story. The secular growth of illegal immigration and domestic travel also contribute to the build-up. Furthermore, one wave may affect another in subtle non-linear ways. For example, one wave might impart partial immunity to a population against a subsequent wave, resulting in the second peak being smaller than it might otherwise be. In fact, the non-linear interaction of infection waves may be part of the reason for the temporal variability of the "annual" cycle. The nearest physical analogue is the formation of drops from a dripping faucet. If the dripping is sufficiently slow, each drop will completely form and fall off the faucet before the next drop begins to form. The time interval between drops is regular in that case. In fact, it is so regular that Egyptian "water clocks" were built using this principle three and a half millennia ago. However, if the water flow is increased so that the next drop begins to form before the first falls off, the time interval between drops will be a little *chaotic*, as will the size of the drops. At first, only two different periods between drops will manifest themselves, but if the flow is increased still further, more periods show up, until finally the period between drops becomes unpredictable.[31] Something of this kind may be affecting the period of the "annual" infection cycle. In any event, when active cases are trending upward, a simple *multiplicative factor* (M) will be used to capture the build-up, where

$M_{growth}(\theta) = k\ \theta(t)$, and $k = 1/(2\pi) \approx 0.159$, and θ (measured in radians) is a function of time. (4)

At the start of an annual cycle $\theta = 0$, corresponding to a particular date (typically in the summer). At the end of the annual cycle $\theta = 2\pi$, corresponding to a date at the end of the cycle (typically in the summer a year later). Values of θ between 0 and 2π correspond to dates between the two end points. When $\theta = 0$, $M_{growth} = 0$, but when $\theta = 2\pi$, $M_{growth} = 1$. When $0 < \theta < 2\pi$, $0 < M_{growth} < 1$. Therefore, active cases produced by thermal effects, immigration, and domestic travel infection models can be multiplied by the M_{growth} function to produce the upward trend (infection reservoir) observed in Figure 3 between June 19, 2020 and Jan. 31, 2022. After that, infection reservoir decay can be produced by multiplying model outputs by a monotonically decreasing function $M_{decay}(\theta)$. Naturally, calculations will begin with the simplest function of this kind; a linear function with negative slope and a range that lies between 1 and 0. The use of these multiplicative functions will become clearer by their use in the next two sub-sections. In summary, M_{growth} and M_{decay} are associated with the respective growth and decline of the domestic coronavirus disease reservoir as a function of time via the three previous infection mechanisms.

The First Cycle in the U.S.: Aside from a small initial infection that lasted about 3 months, and produced 1,112,388 active infections by June 19, 2020, the first full "annual" cycle lasted from June 19, 2020 to Aug. 15, 2021 (~14.07 30-day months – see Figure 3). Let x_{Total} be *defined* by the linear combination of the infection mechanisms characterized by x_1, x_2, and x_3. Then

$$x_{Total} \equiv a_1 x_1 + a_2 x_2 + a_3 x_3, \quad (5)$$

where the relative magnitudes of the *Fourier Coefficients* a_1, a_2, and a_3 indicate the importance of the contribution that each of the three mechanisms makes to x_{Total}. During the first annual cycle, the reservoir of infection in the U.S. grew at a more or less linear rate. Therefore, an equation with a linear form is needed to model the number of active cases (denoted by y) as a function of θ, and therefore time.

$$y = (S.F.)\, M_{growth}(\theta)\, x_{Total} + b, \quad (6)$$

where b is the "initial base-level" of infection going into the first cycle (1,112,388 infections), M_{growth} is

defined by equation 4, and S.F. is a "scaling factor" to be discussed. The Fourier coefficients a_1, a_2, and a_3 may now be selected freely so that the waveform y matches the data set (number of active infections vs. time) as closely as possible, *with the caveat that the end point of the data series and the model are equal*. Otherwise, there will be an unphysical discontinuity between the end of the first cycle and the beginning of the second cycle. This subtle point will become clear when the second cycle is discussed. Note that if we define $\mathcal{M}(\theta) \equiv$ (S.F.) M_{growth} = (S.F.) k $\theta(t)$, then equation 6 can be written in the familiar linear form $y = \mathcal{M}\, x_{Total} + b$. The scaling factor S.F. is a constant chosen so that the height of the largest peak of the waveform generated by equation 6 matches the largest peak of the data set for any given cycle (here, the 1st cycle). *Goodness of Fit* will be measured by two statistics: the *mean relative error (MRE)* and the *root mean square relative error (RMSRE)* defined by

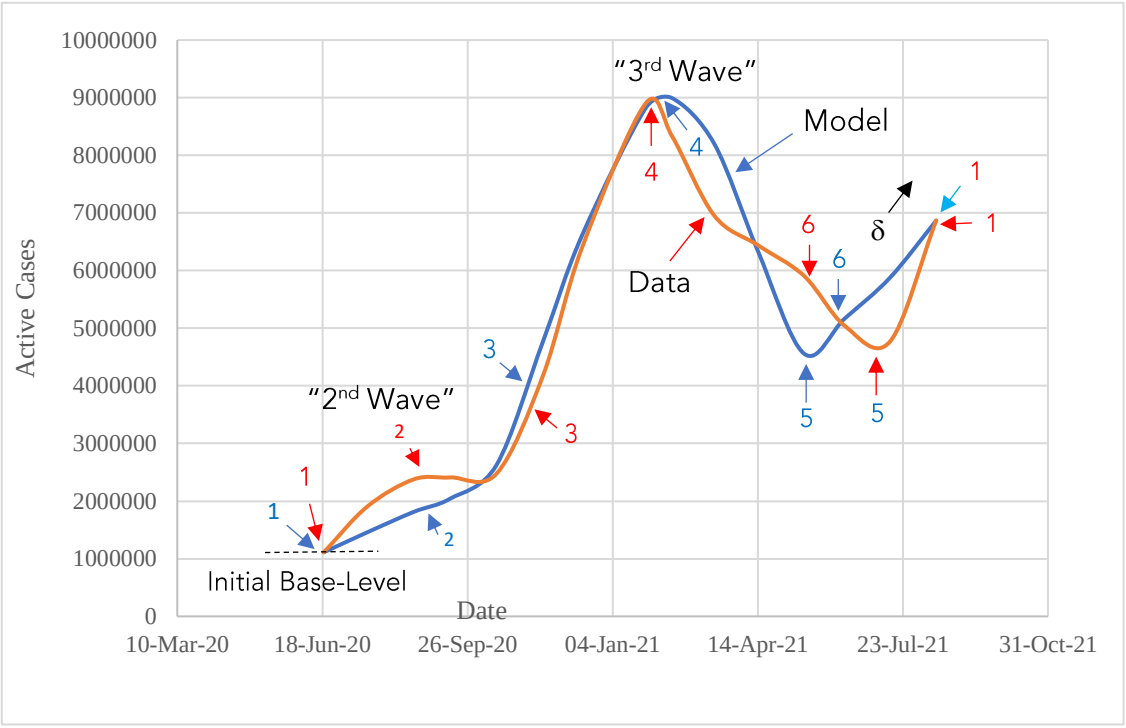
$$MRE = (\Sigma^{N}_{i=1}\, \Delta y_{rel,i}) / N, \text{ where } \Delta y_{rel,i} = (y_{theory,i} - y_{data,i}) / y_{data,i} = \text{relative error, and} \quad (7)$$

$$RMSRE = \sqrt{\{(1/N)(\Sigma^{N}_{i=1}\, \Delta y^2_{rel,i})\}} \text{ and } \Delta y^2_{rel,i} = [(y_{theory,i} - y_{data,i}) / y_{data,i}]^2 = [\text{relative error}]^2, \quad (8)$$

where $y_{data,i}$ is the ith datum of the number of active cases, $y_{theory,i}$ is the ith prediction for the number of active cases (i.e. y from equation 6), and N is the total number of data points.[32] Setting $a_1 = 1.9$, $a_2 = 0.6$, and $a_3 = 1.0$, minimizes both MRE *and* RMSRE yielding Figure 7 below. A few salient points to notice are as follows: 1) the boundary values (at the beginning and end) of both waveforms are the same, as they should be to prevent discontinuities, 2) the weak peak near Aug 18, 2020 is reproduced in both waveforms, 3) there is an outstanding match between the theory and the data on the rising (early) side of the main peak, 4) the main peak of the theoretical waveform differs by only 10 days from its position in the data, 5) the depth of the valley that lies between late June and early July 2021 in the data is almost the same for the model, although the two valleys are separated by 48 days in time, 6) the ripple in the data set near May 14, 2021 is reproduced by the model, but it lies *after* the previous minimum and not before. 7) the MRE and RMSRE between the theory and the data are about -0.83% and 14.27% respectively, the low MRE implies there is about an equal amount of data above and below the model curve – a highly desirable feature.

Although the *existence* of a credible model for the progress of the pandemic in the U.S. has been demonstrated for the first full annual cycle, that does not necessarily prove its *uniqueness*. The existence and uniqueness of solutions are what mathematicians worry about. That is to say, does there exist another set of Fourier coefficients, call them a_1', a_2', and a_3', such that the same model waveform results? The answer to this question is NO! A proof of uniqueness can be found at the end of this publication. Suppose the components of the model are changed, *then* can a model that is just as good or possibly better be found? The answer to this question is YES, but what would such a model look like and what would it take into account if not seasonal effects and person to person contact?

Figure 7 – The number of active COVID cases in the U.S. vs. date for the first complete "annual" cycle of the pandemic (June 19, 2020 to Aug 15, 2021). The blue waveform is what would be expected from theory for the progress of the infection in the U.S. population. The red waveform is what actually happened.[2] Red and blue numbers indicate critical points of comparison between theory and data, and are keyed to the text above.



There can be no doubt about the success of the proposed theoretical model, simple though it may be, because it displays all the observable features of Figure 7. As such, a few inferences can be drawn from the magnitude of the Fourier coefficients. First, the sum of the three coefficients is $a_1 + a_2 + a_3 = 1.9 + 0.6 + 1.0 = 3.5$. The percent of contribution to this total from illegal immigration alone is (0.6/3.5) x 100 = 17.1%. That is to say, the illegal immigration waveform contributes about 17.1% to the total waveform, and there is no way to escape this conclusion for *the current active case model* because the Fourier coefficients are *unique for all practical purposes*. Since the total number of deaths between June 19, 2020 and August 15, 2021 was just over 522,350, according to what was posted on Worldometer on March 17, 2023, the spread of infection due to illegal immigration must ultimately be responsible for about 89,546 deaths. Domestic travel accounts for (1/3.5) x 100 = 28.6%, or 149,243 deaths. Furthermore, the sum of the coefficients for immigration and domestic travel component waveforms is 1.6, implying that illegal immigration accounts for (0.6/1.6) x 100 = 37.5% of the infections due to the movement of people. Finally, thermal effects account for (1.9/3.5) x 100 = 54.3%, or an enormous 283, 563 dead. The sum 89,546 + 149,243 + 283,563 = 522,352 deaths, where the additional 2 deaths are due to round-off errors; a fractional death is not allowed. Therefore, all the dead are accounted for.

The Second Cycle in the U.S.: The second "annual" cycle was actually 10.57 months long and lasted from August 15, 2021 to June 28, 2022; notice that the duration of the first and second cycles together amount to about 24.64 months, or just slightly over two years. The second cycle continues the trend of secular growth in the number of active cases until January 31, 2022. Therefore, equation 6 applies,

$$y = (S.F.)' \, M_{growth}(\theta) \, x_{Total} + b', \qquad (9)$$

where b′ = 6,870,511 cases, and θ starts from 0 radians again (Aug. 15, 2021) and increases to 3.3 radians (Jan. 31, 2022; the date for which the data shows the maximum number of active infections).

The scaling factor (S.F.)' is different from the scaling factor (S.F.) used for the first cycle so that the theoretical and data peak heights match for the main ο1 peak of the second cycle. Furthermore, (S.F.)' > (S.F.) because ο is more infectious than α and resulted in more active cases. Therefore, the scaling factor for each annual cycle captures the effects of the circulating strain multiplicity factor m to be discussed more fully in the next section. After Jan. 31, 2022, the infection reservoir in the U.S. began to decay due to the rapid depletion of susceptible hosts by the highly infectious omicron strain. Between Jan. 31, and the local minimum at 4.8 radians (Apr. 19, 2022), equation 9 must be replaced by

$$y = [(S.F.)' M_{growth}(3.3)\, x_{Total} + b'] M_{decay}(\theta), \qquad (10)$$

where 3.3 radians corresponds to the peak of the active cases curve (data), and

$$M_{decay}(\theta) = -0.622(\theta) + 3.0526. \qquad (11)$$

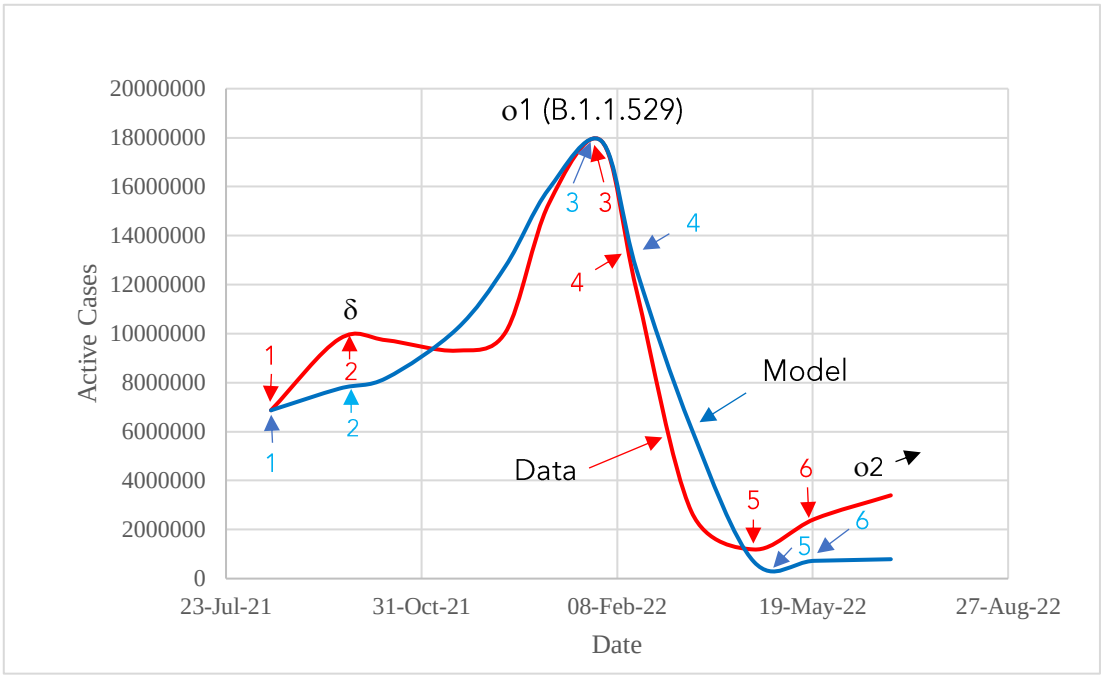
No attempt will be made to minimize the MRE or the RMSRE by changing the three Fourier coefficients (a_1, a_2, and a_3) of x_{Total} from their values established during the first cycle. Therefore, the end points of the data set and the model will not match either. However, in this case, that will not be a problem since, as will be seen, this is the end of what can be calculated. By Apr. 19, 2022 (θ = 4.8 radians), de-trending is complete. A new (final) base-line for the second cycle has been reached with $M_{decay}(4.8) = -0.622(4.8) + 3.0526 = 0.067$, which will remain constant for this third part of the second cycle as $\theta \rightarrow 6.2$ radians (June 28, 2022). However, x_{Total} continues to change with θ as usual. The comparison between y and the data set from August 15, 2021 to June 28, 2022 is shown in Figure 8 below. Notice that the theoretical piece-wise model of the data produces a continuous function without jumps. Furthermore, if the last four data points in the tails of the data and model curves are ignored due to the instability of relative errors calculated near the abscissa, MRE = 1.35% and RMSRE = 14.27% respectively – very similar to what was observed for the first cycle.

Clearly, equations 9, 10, and 11 are tracking the data even without any changes to the Fourier coefficients. The number of deaths during the second cycle was just over 396,842.[2] Taking 17.1% of this last figure to isolate the illegal immigration component yields 67,860 deaths. Deaths from domestic travel and thermal effects are 113,383 and 215,428 respectively, with all deaths accounted for. If the illegal immigration component of deaths from the first infection cycle is added to this last figure, the grand total is 67,860 + 89,546 = 157,406 deaths from June 18, 2020 to June 28, 2022.

Here, we should pause for a quick numerical check via an independent method. The number of migrants intercepted while trying to make an illegal crossing in 2021 and 2022 total about 1.4 plus 2.4 million, or 3.8 million.[26] Let's assume that the number of medically unscreened migrants that successfully enter the U.S. (either by stealth or legal device) is no greater than this – a very conservative estimate. Of these, about one third are sick.[33,34] That makes about 1,254,000 "undocumented" vectors in the U.S. Although COVID was the primary respiratory infection during the peak pandemic years due to its infectiousness, there were other diseases as well. The COVID infected, however, went on to infect between 3 and 16 other people, depending on the strain transmitted (see the next section), and assuming the infection spreads no further than these first direct contacts (very conservative). Suppose a mean of 9 is chosen. That makes a total of ~11,286,000 non-migrant COVID cases (primarily). Of these, about 1% will die. That makes ~112,860 dead. Considering the conservative approximations made, the number of deaths computed from the Fourier coefficients seems quite reasonable. The Fourier calculations, however, are much more accurate because they do not depend on guesstimates of the number of people who successfully evade the border patrol, what fraction have COVID specifically, how many other people they infect, and fluctuations in the death rate (~ ±25%). Nevertheless, "back-of- the-envelope" calculations are very convincing because they don't involve complicated mathematics or computer

simulations, and they do expose the order of magnitude of the health problem created by illegal migration.

Figure 8 - The number of active COVID cases in the U.S. vs. date for the second complete "annual" cycle of the pandemic (Aug 15, 2021 to June 28, 2022). The blue waveform is what would be expected from theory for the progress of the infection in the U.S. population. The red waveform is what actually happened.[2] The steeper decent of the data curve compared to theory after the main peak will be addressed in the next section. The numbers indicate critical points of comparison between the data and model active case curves.



The very serious question that America must answer is whether maintaining essentially open borders is worth the increase in the number of deaths due to disease? It should always be remembered that this story does not end with COVID – *not at all!* Other diseases that have entered the U.S. from abroad include polio,[35] tuberculosis (including resistant strains),[36,37] and the so-called "neglected" tropical diseases (or NTDs) from Dengue fever, to leprosy, to malaria, to Zika Virus infection.[38] Increased border security has greatly reduced border incursions back down to 2022 levels as of the end of February 2023.[26] Furthermore, border arrests seem to have continued to decrease as of August 2024 according to U.S. Customs and Border Protection.

The Third Cycle in the U.S.: No model of Active cases vs. time/date can be generated for the third cycle for several very good reasons: an incomplete data set, "under-reporting", and data tampering. The third cycle consists of two peaks; ο2 and ο3. The ο2 peak, more scientifically known as the BA.4/BA.5 dominated period, was prominent from May through September of 2022 and is shown in Figure 3. The ο2 peak was followed by a "winter" ο3 peak (XBB strains; e.g. XBB.1.5 defined by the mutation F486P) that was most prominent from about Dec. '22 to Feb. '23 and is not shown in Figure 3. By Mar. 20, '23, the U.S. was on the declining flank of the ο3 peak. According to the New York Times, the 7-day average daily new case count stood at 22,591 for the U.S.[39] At that relatively low level, the annual number of infections would be 8,245,715, or 7.8% of the total number of U.S. cases from the beginning of the pandemic until Mar. 20, '23 (105,979,978 cases; a figure equal to about one eighth of the Earth's total COVID cases). The third cycle has shown the usual infection pattern vs. time, but the devil is in the details.

First, consider the o2 peak. Because some counties stopped keeping COVID records, an underestimate in the number of active cases resulted.[40] The problem was further exacerbated by the availability of at-home testing. The discrepancy between what was being reported by the CDC and observations has come to the attention of some researchers.[41,42] Sidharthan estimates that "COVID-19 rates were likely forty times higher than CDC estimates during the BA.4/BA.5 period in the U.S." Qasmieh et al. think 24 times is a better number. These studies, based on surveys, indicate that many people are just treating themselves at home. Partial immunity induced artificially by vaccination, or naturally by a previous wild infection, means that many COVID cases will be quite mild. Nevertheless, the author thinks that estimates based on surveys are too high because non-COVID infections can produce similar symptoms. Every sniffle is not necessarily COVID! Examination of sewage proximal to o2 showed coronavirus levels at or close to their peak o1 values in many locations throughout the U.S.[40,43,44] Under the reasonable assumption that the amount of sequenced genetic material recovered from sewage is directly proportional to the number of active cases in the population sampled, the true number of active cases should be 4.5 times higher (maximum) than the number of active cases based on individual case counts reported to the CDC (see Figure 3). In any case, the true o2 peak height is certainly much higher than what has been reported by the CDC - bad news for the population, but the low CDC figures made good copy for the Biden administration.

The o3 peak has additional problems because the CDC stopped displaying "active cases" as a function of time, only "Weekly Trends in COVID-19 Cases in the United States Reported to CDC" (now called "Provisional COVID-19 Deaths, by Week, in The United States, Reported to the CDC") are presented in detail. Here, emphasis should be placed on the word 'reported'. Worldometer continued to display active cases, but changed the way they present their data beginning with the new year 2023. It appeared that they had essentially removed the pre-o1 growth trend in Figure 3. As if earlier COVID strains could not coexist with latter ones! On March 12, 2023 the author emailed Worldometer asking them why they changed their format. To date no justification, or answer of any kind, has been received. Furthermore, Worldometer showed an o3 active case peak that is only 57% as large as the o2 active case peak for the U.S. in their new format. This seemed suspect given the fact that China had been very hard hit by o3.[45] Furthermore, hospital wards across the U.S. were under stress in December 2022 due to the "Tripledemic" (COVID-19 o3, R.S.V. or Respiratory Syncytial Virus, and H3 flu).[46] Among adults and older Americans, COVID was by far the leading cause of hospitalizations for the three viruses from August to December 2022. These facts suggest that the o3 peak should be larger than that reported, but smaller than o1. So, there are definitely a few problems with the data. Under-reporting will be addressed in more detail in a subsequent section of this report. It should be noted, however, that the trends in total deaths remain unaffected by Worldometer's active case format change.

Although anecdotal, it is also worth noting that two of the author's colleagues with whom he has close association, one engineer and one mathematician, were infected by COVID during this third cycle. J. Surtees, a biochemist at the University of Buffalo in New York, was correct when she told Nature magazine in January of 2023, "I think we are truly flying blind right now. We have no idea how many cases are really out there."[47] Without knowing how the active case data was manipulated, and without knowing how many infection reports are missing, further detailed model calculations are impossible.

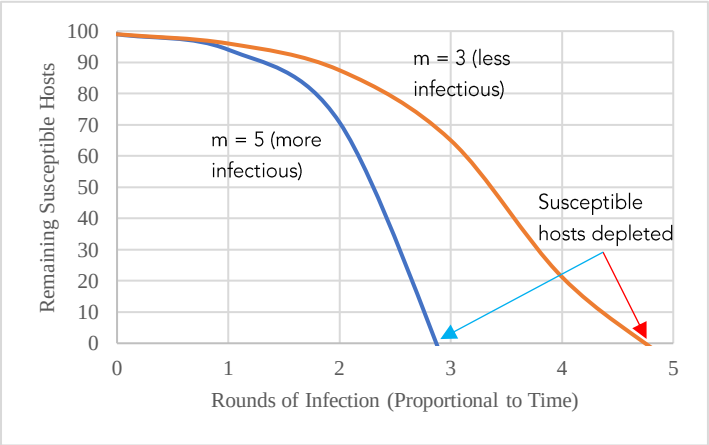
Short-Term U.S. COVID Behavior

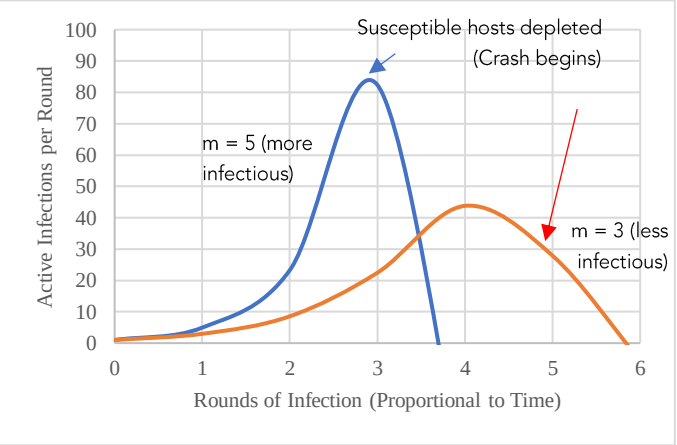
This section opens with a question, "Why does the o1 peak have such a precipitous drop after its peak value?" The same question naturally arises when we examine Figure 8 and note that the theoretical model has a slower decline from the o1 peak than the data. A monotonically decreasing non-linear function

could have been used to model the de-trending and improve the fit to data, but the same basic question remains. The answer is of central importance because it touches upon two important aspects of coronavirus science; modeling and the multiplicity factor. In the first case it is the sharp decline of the $o1$ peak that put an end to the previously growing background of infections in the U.S. (see model equations 6 and 9). In the second case, it is the high multiplicity factor m (the number of other people that each sick person will infect) of o that temporarily depleted the potentially infectious superset (i.e. {unsuccessfully vaccinated} ∪ {partially vaccinated} ∪ {unvaccinated})[12] of its weakest members.

Consider a small population of 100 susceptible people, and suppose 99 are healthy while 1 has become infected from outside the population. Suppose further that once a person is infected, they eventually recover and are completely immune after that. The small percentage of people who die will not be modeled. Furthermore, let the multiplicity factor m be constant. Then the one infected individual will eventually infect m other people, right? Well, not quite, because the original population of 100 people has now been slightly depleted by one sick person. Therefore, on the average, over several ensembles of 100 people, it will be found that only (99/100)m *new* people will be infected in this first round of infection, since the one infected person cannot infect himself. Each of these newly infected will then *try* to infect m other people. However, in the second round of infection (99/100)m+1 people are now either immune (1 person), or already sick (99m/100 people), so that for this last group reinfection is meaningless; although each of the sick can *try* to infect m others. Therefore, after the second round of infection there should be (99m/100)m *new* infections, right? Not quite, because the pool of susceptible hosts has now been further reduced by the *fraction* [100 - (99m/100 + 1)]/100, yielding {[100 - (99m/100 + 1)]/100}(99m/100)m *new* infections as an ensemble average. Therefore, the *total* number of infections is equal to 1 + (99/100)m + {[100 - (99m/100 + 1)]/100}(99m/100)m after the second round. This last formula can be simplified to 1 + (99/100)m + {…}$(99/100)m^2$, which sums the contributions of the 0^{th}, 1^{st}, and 2^{nd} rounds of infection to the total. In other words, if our original population was *infinitely large*, there would have been a total of 1 + m + m^2 cases at this point, but the terms of this sum must be *reduced* by the fractions (99/100) and {…}(99/100) that take into account the depletion in the number of susceptible hosts at each round of infection in a *finite* population. Continuing on with this series in powers of m, the total number of infections will eventually equal, or slightly exceed, the original population after n rounds. When that happens, the entire finite population of susceptible hosts is totally infected, and will eventually become immune – *active case crash!* Figures 9A and 9B show the number of susceptible hosts and active infections as a function of the round number (or time), respectively, for m = 3 and m = 5. The latter is a very high value of m for such a small population. In Figure 9B notice how "peaked" the curve is for m = 5, how it is slightly skewed (called "kurtosis"), and how the decline after the peak is precipitous. In other words, it has all the properties of the $o1$ peak in figure 3. The more infectious the strain, the greater the value of m, and the steeper the decline (cf. m = 5 and m = 3 curves). The peak height is also higher, and occurs earlier, for the more infectious strain as might be expected. For the less infectious case (m = 3), the peak involves less of the population. Finally, since the original Trump-era vaccines were relatively ineffective against o, the U.S. offered an essentially virgin population to the virus. Therefore, this active case crash model should apply.

Figure 9 – A) The number of healthy susceptible hosts from an initial population of 99 healthy hosts and one infected vector vs. the number of rounds of infection. Since each round takes a certain amount of time, the abscissa may be taken as a measure of time on a relative scale. B) The number of active infections vs. rounds of infection (or time). Active case crash is more pronounced for strains with a higher value of m.





It is important to understand that ο is a very infectious disease. The exact value of the multiplicity factor m (also called R_0 in the literature, for reproductive number) is still being debated. However, a few current figures from the literature are that ο is 2.5 to 3.8 times more infectious than δ.[48] While δ is 2 to 4 times more infectious than α, which has an average multiplicity factor m(α) of 3. Liu and Rocklöv set m(δ) = 5.08, compared to ancestral strains (e.g. α) with m(α) = 2.79.[49] That would yield at least m(ο) = 12.7 (i.e. 5.08 x 2.5), making it close to the most infectious disease known (measles is the worst with a multiplicity factor of ≈ 20). However, Liu also thinks 3.6 < m(ο) < 8.2 is the right range.[48] Kimihito Ito, working in Denmark, set m(ο) = 3.19 m(δ), with 95% confidence.[50] Again, that would make ο slightly less infectious than measles (3.19 x 5.08 = 16.2).

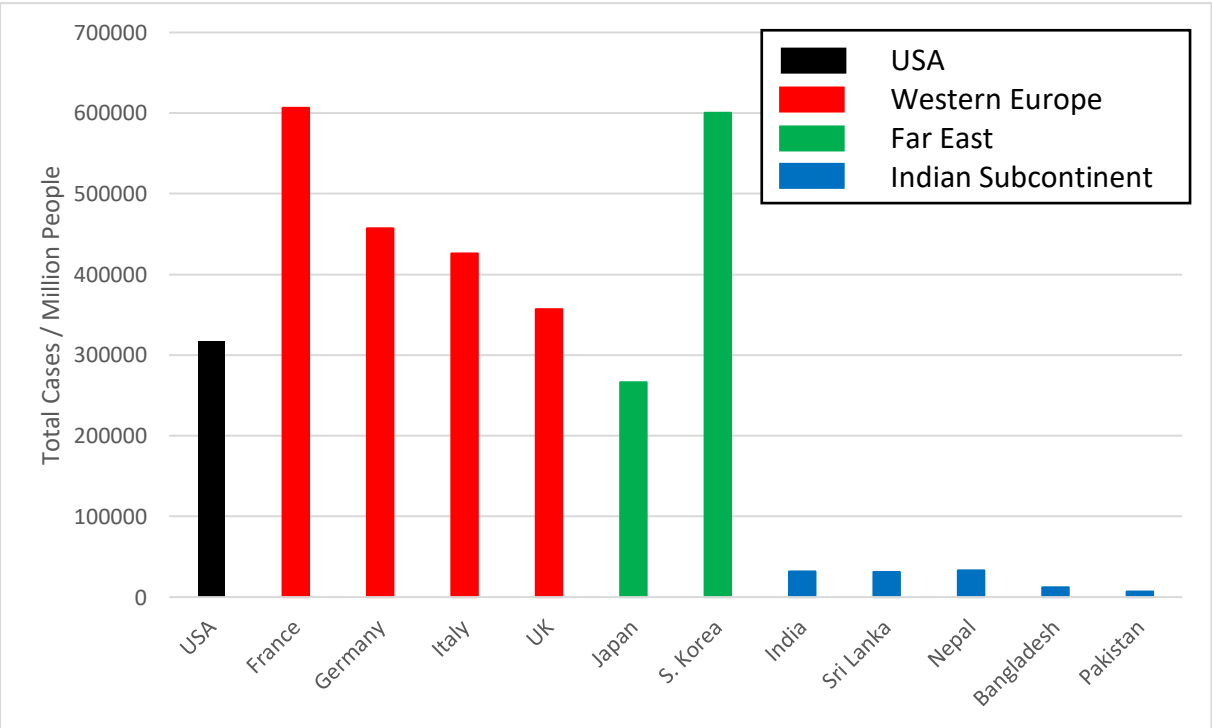
Under-Reporting

This section, like the last, opens with a question, "Why is it that as of Oct. 11, 2021, the U.S. had more cases of COVID than any other country in the world, and a shocking 45 times more active COVID cases than runner-up India, even though India has 4 times the U.S. population!" Is under-reporting by India to blame? Under-reporting affects all countries, including the U.S.; as was explained above for the third infection cycle. However, the amount of under-reporting may vary from country to country. Calculations begin by normalizing the total number of cases reported by each country by that country's population in millions. Twelve countries were selected to represent four different areas of the globe; five for the Indian subcontinent, two for the Far East, four for Europe, and of course one for the U.S. The bar graph in Figure 10 summarizes those statistics.

If one were to only consider India with respect to the seven highly industrialized temperate countries that precede it in Figure 10, one might easily conclude that India was under-reporting its case load relative to the others. However, high levels of industrialization imply cities with high population densities and people working in close proximity to one another, conditions which contribute to the spread of disease in those temperate, seasonally cold, countries. Furthermore, when the total cases per million are examined for India's neighbors, similar figures emerge. It is unlikely that they are all under-reporting in the same way. Furthermore, India has administered about as many COVID tests as the U.S. (approximately one billion from Apr. 2020 to Oct. 2023),[52] but there just doesn't seem to be as much per capita infection in that country as there is in the U.S. The reader should note that India is a technology powerhouse. They even manufacture their own vaccines. The ready availability of such intervention also inhibits COVID infection. Furthermore, it must

be remembered that case counts may vary from one climate to another, from one continent to another, and from one ethnic population to another. A warm climate tends to limit the spread of infection, as previously discussed. Isolation limits the spread of disease as well. The remoteness of the mountainous back-country in Pakistan must certainly account for some under-reporting in that country, but those people are also less likely to be infected by outsiders. India itself is a security island. To the southeast and the southwest lies the Indian Ocean, To the northeast lies the "Great Wall of India", a 4,100 Km (2,500 mile) barrier between Bangladesh and India (the 5th longest land border between two nations) that is backed-up by Indian troops. The purpose of this security wall is to stem illegal entry of insurgents, migrants, and drugs (Anon., 2006).[53] Does this sound familiar? To the northwest lies Pakistan, with whom the Indians occasionally exchange artillery rounds over Kashmir. There is a barrier along this border as well. Finally, to the north lies Nepal and the daunting Himalayas. Air travel is India's primary public contact with the outside world. For all these reasons the low per-capita incidence of COVID in India does not necessarily imply under-reporting. In the author's opinion, it is doubtful that India is greatly underestimating COVID infections in their country relative to the U.S. Here, in the U.S., two obstacles stood in the way of under-reporting by the U.S. government: Dr. Francis S. Collins and Dr. Anthony S. Fauci! Both of these leaders have since retired from government service. It should be noted that Francis Collins was "*temporarily*" replaced by Alondra Nelson, a former sociology professor at Columbia University – not a biochemist, immunologist, microbiologist, or physician. There was no immediate replacement for Anthony Fauci as of Dec. 2, 2022. It is unlikely that the errors introduced by under-reporting can ever be completely eradicated from the global data set.

Figure 10 – Total cases per million people for the 12 countries in this study. Isolated, less industrialized nations (blue) seem to have less problems with the spread of COVID. Figures from Worldometer on March 30, 2023. Per-capita infections in the U.S. are typical of those in other industrialized countries, but the U.S. is the third most populous country in the world. Therefore, the U.S. has more cases total than any country in the world. The World Health Organization now thinks China (with four times the U.S population) is close behind with 4 million less infections.[51] India, the previous runner-up, has less than half the U.S. total even though it also has four times the U.S. population.



Conclusion

Every nation, every people, every empire, eventually commits suicide. During the peak COVID years it seemed like America would follow. Not by revolution and war, not by steel and blood, not by bullets and bayonets, but by *people, drugs, and germs*. Each collapse follows a unique course, but ultimately the underlying common cause is *short-sighted, irresponsible, often fanatical leadership*. Even though we see and understand the problems, each cycle from growth to collapse repeats itself as we all stand by helplessly and watch like zombies. So irresistible, so immutable, are the tides of history. Today, we in America are ignoring our problems by failing to implement necessary far-sighted policies, on the mistaken premise that *nothing* can destroy America. An axiom of Hindu wisdom reminds us that "all compound objects must decay" – and so it is! If America is going to have a *healthy* long-term future several governmental policies must be reversed or enacted – not just for future COVID outbreaks, but for the control of other diseases as well. Now is the time to act. In Venice, the local people have a saying, "They invented brass doors after St. Mark's was robbed." (Hanno inventato le porte d'ottone dopo che il duomo di San Marco era stato derubato.) Let's not wait until it's too late – *again*!

1) *Stop* replacing capable scientists in positions of responsibility with political cronies.
2) *Continue* keeping records on COVID infections since new strains are always evolving. Without records we will not know if a new strain is spreading through the population until it is far too late to react.
3) *Fund* programs aimed at battling pandemics and disease outbreaks in general. Less than 38 days after the o1 peak, congress abruptly abandoned a 15.6 billion-dollar COVID package (for vaccines, therapeutics, tests, and suppressing the spread of variants), out of a vast 1.5 *trillion-dollar* government spending bill![54,55] And what happened to the previous money allocated for COVID relief? Much of it was *stolen*![56,57] *Accountability* is required as well as funding. There is a reason why the total number of infections was so high in the U.S., and it's not just under-reporting by other nations.
4) *Produce* an updated COVID vaccine *annually*, just as we do for influenza, and have a permanent infrastructure in place to distribute it in a timely fashion each fall. *It should be noted that in February 2023 the CDC finally decided to start a program of annual vaccine updates to begin in the fall of 2024*.[58] This decision should have been made by the Biden Administration in 2021!
5) *Maintain* a credible border policy that prevents the spread of disease, drugs, and criminals into the U.S. An open border policy is neither acceptable or responsible.
6) *Check* the temperature of all international and interstate travelers on both ground and air routes, as we check on the movement of fruits, vegetables, and plants. It should be noted that on, or about, June 10, 2022 the CDC rescinded an order requiring a negative pre-departure COVID-19 test when flying to the U.S.[59] Although more cumbersome than temperature checks, there is no reason to stop checking since COVID is admittedly still very active worldwide. The spread of COVID and other air bourn infections aboard aircraft has been reduced by the use of new cabin air disinfection technology.[60]
7) *Initiate* credible public information campaigns to get everyone vaccinated, using the same effective public relation techniques that politicians use to get themselves elected and to destroy their political enemies.
8) *Improve* the CDC website to include credible basic scientific information. Educated people are not impressed by vague non-comital statements, or a wavy line with an arrow on the end of it pointing down, and no scales, that is supposed to represent a "dumbed-down" graph!

These eight policies are what science demands. The author realizes that mounting statistics on the dead are a great embarrassment to some politicians, and an impediment to their re-election. The way to solve that problem is to enact far-sighted policies that will control disease, and not to shortsightedly stop collecting data, thereby making the problem worse for all of us. Denial does not constitute a plan of action! When President Joe Biden was elected, he promised to "follow the science" concerning this pandemic. So, let's look at the record. When Trump left office, he had been dealing with the pandemic for 1 year from its beginning, and there were about 442 thousand deaths. A vaccine only became available at the very end of Trump's tenure, testing was slow and relatively expensive, and the hunt for post infection therapeutics was in its infancy. However, the "lock-down" may have slowed the growth of the infection. Biden had the advantage of the vaccine, inexpensive reliable test kits, and FDA approved therapeutics. However, *all eight of the policies above had been violated in some way*. During Biden's first year in office, he did a little worse. There were about 458 thousand additional deaths, as the body count continued to grow linearly (Figure 2). In the author's *opinion*, after all the name-calling and hate, the 3.6% difference (relative to the mean) between these 1-year death tolls is hardly worth quibbling about due to the statistical uncertainties involved.

-Stay Well!

Proof of Uniqueness

In this section the uniqueness properties of the Fourier coefficients a_1, a_2, and a_3 will be explored for the given model of y. More precisely, *for the linearly independent functions x_1, x_2, and x_3 defined by the equations 1, 2, and 3, and a function y defined by equation 6, with x_{Total} defined by equation 5 and M_{Growth} defined by equation 4, there exists one, and only one, set of well-defined Fourier coefficients that can generate the function y over the entire closed interval from $\theta = 0$ to 2π (~1 year), except at $\theta = \pi$ (corresponding to an instant in the middle of an infection cycle) where a well-defined set of Fourier coefficients does not exist.* Proof of this theorem proceeds by assuming the opposite. That is to say, it will be assumed that there exists another set of Fourier coefficients a_1', a_2', and a_3'such that

$$(S.F.)\, k\theta\, (a_1' x_1 + a_2' x_2 + a_3' x_3) + b = y = (S.F.)\, k\theta\, (a_1 x_1 + a_2 x_2 + a_3 x_3) + b, \quad (12)$$

where S.F., k, and b are fixed constants. Therefore,

$$a_1' x_1 + a_2' x_2 + a_3' x_3 = a_1 x_1 + a_2 x_2 + a_3 x_3 . \quad (13)$$

Therefore,

$$(a_1'-a_1) x_1 + (a_2'-a_2) x_2 + (a_3'-a_3) x_3 = 0 . \quad (14)$$

Because x_1 is a "*piecewise function*" (i.e. the θ axis is partitioned into several intervals on which the function x_1 is defined differently) the remainder of this proof of uniqueness must be divided into three parts.

First consider the θ intervals $\phi < \theta < \pi$ and $\pi < \theta < 2\pi - \phi$ (where $0 < \phi < \pi$). In other words, the entire open interval $\phi < \theta < 2\pi - \phi$ with $\theta = \pi$ specifically excluded. Over this interval all three *basis functions* x_1, x_2, and x_3 are strictly positive and never zero; $\theta = \pi$ has been excluded because $x_3 = 0$ there. Defining $f \equiv \pi / (2[\pi - \phi])$ for convenience and brevity, and substituting equations 1, 2, and 3, into 14, yields after expansion and recollection of terms

$$(a_1' - a_1) \sin (f [\theta - \phi]) + \{(a_2' - a_2) - (a_3' - a_3)\} \sin (\theta/2) + (a_3' - a_3) = 0 . \quad (15)$$

The only way to satisfy equation 15 is if the constant $a_3' - a_3 = 0$. Therefore, $(a_2' - a_2) - (a_3' - a_3) = a_2' - a_2 = 0$ and $a_1' - a_1 = 0$, because both $\sin(\theta/2)$ and $\sin(f[\theta - \phi])$ are always positive over the entire open θ interval (i.e. end points at ϕ and $2\pi - \phi$ are not included) and neither function is a simple multiple of the other for any allowed value of ϕ. Therefore,

$$a_1' = a_1, \quad a_2' = a_2, \quad \text{and} \quad a_3' = a_3 . \qquad (16)$$

In other words, there exists only one set of Fourier coefficients a_1, a_2, and a_3, for the intervals over which x_1, x_2, and x_3 are positive non-zero functions. The condition $a_1'-a_1 = 0$, $a_2'-a_2 = 0$, and $a_3'-a_3 = 0$, defines the *linear independence* of the three basis functions x_1, x_2, and x_3 in equation 14.

Next, it is necessary to prove linear independence over the two half open intervals $0 < \theta \leq \phi$ and $2\pi - \phi \leq \theta < 2\pi$, where $x_1 = 0$. Over these two intervals equation 14 simplifies to

$$\{(a_2' - a_2) - (a_3' - a_3)\} \sin(\theta/2) + (a_3' - a_3) = 0. \qquad (17)$$

It is necessary for $a_3' - a_3 = 0$ to satisfy equation 17. Notice that $\theta = \pi$ is excluded from the two half open intervals because ϕ is strictly less than π by definition (see equation 1). Therefore,

$$(a_2' - a_2) \sin(\theta/2) = 0 . \qquad (18)$$

Since $\sin(\theta/2)$ is always positive, except at the excluded end points $\theta = 0$ or $\theta = 2\pi$ where it is 0 (see Figure 5), it must be true that $a_2' - a_2 = 0$ when $0 < \theta \leq \phi$ and $2\pi - \phi \leq \theta < 2\pi$ (for all $0 < \phi < \pi$). Therefore, over these open intervals $a_2' = a_2$ and $a_3' = a_3$. Therefore, a_2 and a_3 uniquely define the shape of y. Furthermore, x_2 and x_3 are linearly independent. The coefficient a_1 can be any real number from $(-\infty,+\infty)$ because x_1 is zero and contributes nothing to the shape of y over $0 < \theta \leq \phi$ and $2\pi - \phi \leq \theta < 2\pi$. Since a_1 can have an infinite number of possible values, it is said to be *undefined*.

All that remains to be proven is the uniqueness of any well-defined Fourier coefficients at the special points $\theta = 0$, π, and 2π. Since both x_1 and x_2 are zero at $\theta = 0$ and 2π, the value of y for these values of θ depends only on the one constant a_3, which by definition is unique and well-defined. While a_1 and a_2 can be any real number and are, therefore, undefined. Finally, at $\theta = \pi$, $x_3 = 1 - \sin(\pi) = 0$. Substituting $x_3 = 0$, and the equations for x_1 and x_2 with $\theta = \pi$, into equation 14, yields

$$(a_1' - a_1) \sin(f[\pi - \phi]) + (a_2' - a_2) \sin(\pi/2) = 0 . \qquad (19)$$

Since $\sin(\pi/2) = 1$ and, recalling the definition of f, $\sin(f[\pi - \phi]) = \sin(\pi/2) = 1$ (see Figures 4 and 5), equation 19 becomes

$$(a_1' - a_1) + (a_2' - a_2) = 0, \text{ or just } a_1' - a_1 = a_2 - a_2' . \qquad (20)$$

Therefore, if a_1 and a_2 are the Fourier coefficients that are used to generate y from x_1 and x_2 over the open interval $\phi < \theta < 2\pi - \phi$, then at $\theta = \pi$, there exists an infinite number of other pairs of coefficients a_1' and a_2' that can generate the same value of y, so long as the condition of equation 20 is satisfied. Therefore, at $\theta = \pi$ (ONLY), corresponding to an instant in the middle of an infection cycle, a set of well-defined Fourier coefficients *does not exist* since a_3 can be chosen arbitrarily, and there are an infinite number of ways to choose a_1' and a_2' so as to satisfy equation 20. If this argument seems obscure, don't be confused. The situation is actually very simple when one realizes that at $\theta = \pi$, $x_3 = 0$, and equation 6 becomes $y = (\text{S.F.})\, M_{growth}(\theta)\{a_1 + a_2\} + b$. Suppose, as in this report, $a_1 = 1.9$ and $a_2 = 0.6$. The sum of these numbers is 2.5. However, the same sum could be achieved if $a_1 = 2.0$ and $a_2 = 0.5$. In fact, there are an infinite number of possible choices for the real numbers a_1 and a_2 that would result in the same sum and, therefore, the same value of y, *at the single point $\theta = \pi$!* This ambiguity arises because $\theta = \pi$ is not part of an interval, $x_3 = 0$ at $\theta = \pi$, and because both $\sin(f[\theta - \phi])$ and $\sin(\theta/2)$ evaluate to unity at $\theta = \pi$.

This completes the proof of uniqueness and all its caveats and exceptions. However, two related comments are relevant. The first is that a similar proof applies when there is de-trending (decay), as in the second cycle (see Figure 3). The second is much more subtle. Suppose the analyst changes x_1, x_2, or x_3 slightly. For example, suppose $x_3 = \frac{1}{2}(1 + \cos\theta)$. This

function has the same general shape as the original choice for x_3, and it also forms a linearly independent basis set with x_1 and x_2, so the calculated Fourier coefficients are unique. What effect does this change have? The answer is that the Fourier coefficients that achieve a best fit to the data are almost exactly the same, but the MRE is worse at 6.32%! In other words, the calculation of the all-important Fourier coefficients is stable, and relatively insensitive to the shape details of x_1, x_2, and x_3! This is why the author stated that "great accuracy is not required" for the selection of these functions in the section on *Mathematical Models for Annual Infections.* At the deepest level, it is also why the author was able to simplify the calculations by choosing the simplest functions with the proper general behavior, in accordance with Occam's Razor. The reader can now fully understand how all the pieces of the infection calculations fit together!

Conflict of interest statement:

The author declares that he owns 36 shares of Moderna common stock and has no other known financial interests or any personal relationships that could appear to influence the work reported in this publication.

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