

The COVID-19 Pandemic

These last few months have been challenging. The lives lost to the C19 pandemic are terrible and tragic.

It's worth saying up front: COVID-19 is not the flu. It is both more deadly and far more transmissible.

That so many have put themselves at risk (often with less-than-ideal protection) to respond to emergencies, care for loved ones, keep us safe, build and transport life-saving supplies, stock grocery shelves, deliver food, and perform untold acts of kindness and goodness, shows the extraordinary character of the American people.

Our definition of the word "hero" has been forever changed and expanded.

Misinformation is Widespread

At the same time, the misinformation regarding COVID-19 has been astonishing.

Our most trusted media outlets have resorted to the Yellow Journalism of a century ago, printing hyperbolic and misleading headlines in search of clicks and eyeballs. Political leaders have ignored evidence-based science in a race to appear "tough on the virus." Many epidemiologists – a crowd naturally drawn to the macabre – have fanned the flames by predicting imminent catastrophe, only to see their forecasts go disastrously wrong within days.

The "experts" have badly failed us in this crisis. Their actions too often have done more harm than good.

A Fact-Based Analysis

By mid-March, it was clear the path forward for both the economy and securities markets was firmly tied to the progression of the C19 pandemic. Informed by our collective experience investing during the 2008 financial crisis, we felt it essential to develop a fact-based, internal view to help us navigate the uncertainty.

Importantly, we believed we could do it. For though the pandemic is first a question of science, in practice it is really a question of statistics, mathematics, and modeling/forecasting. These disciplines are in our sweet spot.

As we worked, it quickly became clear that never in human history has so much real-time information been available to track a pandemic. The data included (but was not limited to):

- Data on tens of millions of tests and millions of infections.
- Data on hospitalizations, ICU's, deaths, and recoveries – categorized by age, co-morbidity, and geography.
- Data on numerous drug studies (past and ongoing, in vitro and in vivo) studying treatments/therapies.
- Data on dozens of "case studies" of complete populations (cruise ships, prisons, etc.).
- Data on antibody results tracking millions in towns and cities across the globe.
- Data from real-world A/B tests as countries/states adopt varying strategies comparable in real-time.
- Data from alternative data sets that help us reach unbiased conclusions.

Was the data perfect? Not always. Many studies were pre-prints. Reporting and definitional inconsistencies complicated comparisons. Some data sets were small or difficult to access.

But that did not dissuade us. **We have long believed the greatest skill in investing is the ability to make good decisions using imperfect information.** In our judgment, there was ample data available to help us reach sound, evidence-based conclusions that could inform rational investment decision-making.

In this [Appendix A](#), we offer our analysis (via a Q&A format) and detail our investment outlook. We include a reference list in [Appendix B](#) with links to a number of articles and studies mentioned along the way.

1. Before we begin, do you have any disclaimers to share?

This analysis is not medical advice. Do not self-medicate. Please consult your doctor before taking any therapy.

This document is not an endorsement (or negative endorsement) of any politician or political entity.

Finally, events are moving quickly and we live in a moment of great uncertainty. This analysis relies on the best-available current information to reach logical conclusions. That said, we follow the data – and if the evidence changes, we reserve the right to change our views and positioning as well.

2. Please describe your high-level thesis.

We are optimistic the economy and asset prices are poised for a strong recovery.

Our thesis is in two parts. First, we believe the spread of C19 has likely peaked and is now in sustained decline. Second, we think governments and central banks will err on the side of doing “too much” rather than “too little” as they deliver record-setting stimulus, leading to robust economic growth as society re-opens.

To recapture this year’s highs, the SPX and R2000 must climb +11% and +22%, respectively (from end of May). A successful re-opening of the economy paired with interest rates at zero, Fed asset purchases in the trillions, and fiscal stimulus at “whatever it takes,” is a rocket-fueled blueprint for equity markets to surge even higher.

We see the greater risk today as being too short, not too long.

3. Let’s start with COVID-19. How has the pandemic peaked without a vaccine or herd immunity?

Many believe the only two paths out of the pandemic are either (1) a vaccine or (2) “herd immunity.”

We see this as a false choice. In fact, we believe the most likely outcome is a third and different path: that C19 has reached its “disease break point” in the US/Europe such that population-level spread is now in inexorable decline.

So let’s discuss how outbreaks end.

First, a definition. What is “herd immunity”? A population reaches herd immunity when a sufficient percentage of its members have specific resistance (are “immune”) such that the disease dies out. Specific resistance can be acquired either by recovering from the disease or via a vaccine.

The percentage required for herd immunity varies with a disease’s contagiousness. To measure contagiousness, epidemiologists use the mathematical term R_0 (pronounced “R-naught”). R_0 is the theoretical number of people who contract an illness from each infected person, assuming no existing specific resistance in a population.

For influenza (the flu), which has an R_0 between 1.5 and 1.8, the threshold for herd immunity might be 45-50%. For C19, which we think has an R_0 of 2.5-3.0, the herd immunity threshold may be 60-65%. For measles, a highly-infectious disease with an R_0 in the teens, more than 95% must have specific resistance to reach herd immunity.

One fact about herd immunity, however, is too often glossed-over: the herd immunity calculation estimates the theoretical threshold at which it is mathematically inevitable the disease will go extinct.

That is a lofty goal. So lofty, in fact, that almost no disease ever achieves it. Why? Because in practice, spread of a given disease collapses far before a population ever reaches herd immunity.

A good example is the flu. Influenza mutates easily, so each year a new vaccine (a flu shot) is required which attempts to “guess” what the flu will look like that year. Sometimes it hits. Sometimes it misses.

But a curious thing happens when the flu shot misses. That year's strain of the flu explodes, gets a lot of people sick – and then spread of the strain collapses when around 10-15% of the population is infected. The seasonal flu never comes within spitting distance of reaching the 45-50% level required for herd immunity.

And this doesn't apply just to modern seasonal illnesses. The 20th-century's greatest pandemic (the Spanish Flu of 1918) probably had an RO just above 2.0, so the herd immunity threshold was likely 55-60%. But historians estimate just 20% of people had been infected when the Spanish Flu's spread suddenly collapsed. Philadelphia saw peak deaths in mid-October 1918, but by mid-November the disease was effectively gone from the city. Spread of the Spanish Flu peaked and plunged in weeks, without ever reaching herd immunity.

So how do we explain this?

The answer: there is not one, but two levels of population "immunity" to consider.

First, **herd immunity**: the level of specific resistance in a population required for a disease to fully disappear.

Second, **the disease break point**: the level of specific resistance in a population at which spread of a disease collapses. The disease break point is generally one-third or less the threshold required for herd immunity.

The difference between the two can be understood with basic math. Herd immunity is a theoretical calculation. It relies on mathematical assumptions. For example, the herd immunity calculation assumes homogeneity of actors and outcomes: that infections, specific resistance, social graphs, and individual susceptibility are all equally distributed. If the RO is 1.8, each infected person transmits the disease to exactly 1.8 people. If 1% of a population has specific resistance, then 1% of each person's social graph has specific resistance too.

The problem is obvious. These assumptions simply do not reflect the real world. Actors and outcomes are neither homogenous nor equally distributed. In fact, the opposite is true. Some people have highly-connected social graphs (once infected, they are likely to infect many more) while others have relatively few connections (and may not infect anyone else). Some sub-populations are highly vulnerable (e.g., nursing homes) while others are highly resistant (e.g., young people). Actors and outcomes are not equally distributed. They have tremendous variance.

The disease break point model uses graph theory to better explain how outbreaks evolve in practice. The model assumes actors and outcomes are not equally distributed – and in fact assumes they are concentrated in certain individuals and sub-populations. A node with a well-connected social graph is more likely to be infected early and to transmit the disease widely. Once recovered, however, the "immune" node becomes a dead-end for future disease spread. The system spikes and then collapses far quicker than a herd immunity model (a homogenous approximation model) would predict as these "super-spreaders" become "super-suppressors."

For COVID-19, the implications are powerful. If C19's RO is 2.5-3.0 and its herd immunity threshold is 60-65%, then the disease break point would be only 15-20% specific resistance (a population's precise disease break point likely varies somewhat due to differences in susceptibility and social graphs).

Our research indicates Europe and the US reached this disease break point in March and April, respectively. We believe spread of COVID-19 in these geographies has peaked and is now in irrevocable, sustained decline.

The New Yorker recently published a memoir by a journalist who lived in Hong Kong during the 2003 SARS epidemic. The author wrote of his fear when the outbreak began, the isolation and social distancing, the gradual return to normal as the disease receded and "some internal calculation seemed to show the benefits of living our lives newly outweighed the risks," and his struggle to remember exactly when he stopped wearing a mask in public.

His story matches the science for how outbreaks end. It won't be a flip-the-switch moment. It won't be a declaration that we have a vaccine or that we've reached herd immunity. It will instead be a gradual realization that things are OK again, and we can finally, bit by bit, come out again into the light.

4. Wow - that's a lot to take in. Are you sure people who have recovered from C19 are immune?

The media has printed a number of breathless pieces on this topic. These articles are generally misleading and don't seem to understand how diseases work (they also are invariably later retracted).

This is important, so let's set things straight.

To directly reply, the simple answer is "yes." Once a person recovers from COVID-19, that person has specific resistance to the disease. As with other coronaviruses, specific resistance is likely to last years.

A better answer, however, is more nuanced. So let's go to the science.

First, to be clear: there is no such thing as immunity to a disease. A vaccine does not confer immunity. Recovering from a disease does not confer immunity. Immunity as a functional concept does not exist in the real world. That's why in this letter we are careful to use "specific resistance" instead of "immunity" when possible.

Once a virus infects a person without specific resistance, the race is on for the body to mount a response before the virus replicates to the point it becomes dangerous. It takes time to develop and produce specific antibodies that can fight back against the pathogen. This delay reduces the odds of recovery. If a person already has specific antibodies (or a template to produce them quickly), the odds of beating the virus go up dramatically.

A vaccine generally works by injecting inert antigens that mimic a disease, allowing the immune system to build up virus-fighting antibodies without actually being infected. The end-result of a vaccine is no different than having had the disease and beaten it - the person's immune system now has specific antibodies that allow for a rapid response to future virus infection, making it incredibly likely the person will successfully see off the disease.

Here's where it gets nuanced. Is it possible for a person with specific antibodies to get sick from a disease again? Is it possible to have beaten a disease and not have specific antibodies show up in a serology (antibody) test?

The answer is "yes" to both questions.

A simple example. Consider a person who had chicken pox as a child. This individual now has specific resistance. But let's say as an adult this person is infected with a disease that compromises the immune system (such as HIV/AIDS). If a person's immune system is so weak it struggles to fight off any disease, then even having antibodies or an antibody template may not be enough. With an immune system incapable of mounting a sufficient response to any pathogen, the person could be infected with chicken pox again (and it could be dangerous).

Another example. There are cases each year where an individual has pneumonia or some other respiratory tract infection (an "RTI") and beats it. The person gets better. But in the process of clearing up, a globule of virus sequestered in the lungs or sinus cavity becomes dislodged and the recoveree is whacked with a teaspoon of concentrated virus. The person becomes ill again. Viral dosage matters - and if a person receives a big enough second dose, he/she may need to mount a new defense against the same infection. It's not that the person lacks specific resistance. It's simply that the body is being poisoned with concentrated virus, which requires a response.

Could someone beat a disease and not test positive for specific antibodies? Yes, of course. But that's usually good news. It means the illness was easy to fight off, so it was unnecessary to build up many antibodies to destroy it. In fact, that's almost certainly the case with COVID-19. Some people (such as kids with hyper-active immune systems) probably beat C19 with pathogen-fighting lymphocytes (T-Cells) already present in the blood that can clear the illness before an antibody response has time to ramp up. Others may have cross-resistance to C19 from other coronaviruses (seven coronaviruses are known to infect humans; four are forms of the "common cold"). Folks in both categories have strong resistance to COVID-19, but would not test positive in a serology test.

The point is - this is the real world. It is not black-and-white. Which brings us back to the misleading headlines.

We are confident that articles about “reinfections” actually refer to either bad tests or “corner cases.”

On the former, there are three tests available for COVID-19: (1) serology tests, (2) PCR tests, and (3) antigen tests. Serology tests determine whether a person had C19 in the past (and beat it) by identifying antibodies that indicate specific resistance. These tests are hyper-accurate: serology tests from Roche and Abbott Labs claim 100% sensitivity and 99.6% specificity. Widely-used PCR tests (90-95% accurate) and less-widely-used antigen tests (80-85% accurate) determine whether a person is currently infected with COVID-19.

PCR tests are pretty good – but across a large enough sample size, they still produce a noticeable number of false positives. In addition, there could always be “corner cases” (like those described above). The panicked headlines mistake the science by conflating “reinfections” with a limited number of bad test results and the rare corner case.

Neither changes the reality. A COVID-19 recoveree has specific resistance (“immunity”) which likely lasts years.

5. How has the US reached the disease break point? Haven't only 2.3M Americans (0.7%) been infected?

This is key to our analysis. We believe the US reached its disease break point (15-20% specific resistance) in April and that population-level specific resistance in the US today may be closer to 30%.

It should be self-evident that two million identified COVID-19 cases in the US is a material undercount. The first confirmed cases in the US were in January 2020. Data from the US Centers for Disease Control and Prevention (the “CDC”) indicate a surge in unexplained US deaths with “symptoms, signs, and abnormal clinical and laboratory findings, not elsewhere classified” beginning in mid-November 2019 that correlates with the spread and then April peak of C19 in this country. We think it likely COVID-19 was in the US by October/November 2019 – and that this readily-transmissible disease had already swept across the nation a few months later.

Testing for C19, on the other hand, lagged badly. At the start of March, the entire US was only completing hundreds of tests per day. By the end of March, tests per day were still just in the tens of thousands. With high demand and so few tests available, the CDC and state governments published guidelines prioritizing who would be tested – and who would not. Universal free testing only just became a reality in some geographies in June.

The magnitude of the undercount is subject to debate – but the number is large, with many untested symptomatic infections for each confirmed case. An analysis published in April (using CDC outpatient data) by researchers at Cornell, Penn State, and Montana State estimated a 6-10x multiplier. We think this is a sensible figure.

That accounts for symptomatic infections. But what of asymptomatic infections? Humans have lived with coronaviruses for thousands of years (as mentioned, four are forms of the “common cold”) and, over time, have developed general resistance to them. As a result, coronaviruses are asymptomatic in humans 90%+ of the time. A zoonotic coronavirus (from an animal) like COVID-19 logically should be asymptomatic somewhat less often.

Complete population surveys support this view. On the Diamond Princess cruise ship, 51% of confirmed cases were asymptomatic. On the Greg Mortimer cruise ship in Uruguay, the count was 81%. Tyson Foods found 199 infections in one of its packing plants, with 198 asymptomatic (99%). After testing all servicemembers on the USS Theodore Roosevelt, General John Hyten noted the asymptomatic rate was nearly 70%. A study in Italy of 4,326 cases identified via contact tracing found 69.1% of them were asymptomatic (and never developed symptoms). A review of more than 3,300 COVID-positive inmates in four US jails found an asymptomatic rate of 96%.

Based on these and other data sets, we estimate the COVID-19 asymptomatic rate is roughly 75% of infections. For each symptomatic C19 infection, three more are asymptomatic. The multiplier is 4x.

Combining the estimates for symptomatic and asymptomatic infections yields a multiplier of 24-40x. With 2.3M confirmed cases to-date, this indicates between 55M and 92M Americans (17-28% of the population) may now have infection-acquired specific resistance to COVID-19.

The final piece to the puzzle is cross-resistance. Recent analyses from leading scientists and statisticians estimate between 40% and 80% of people may have cross-resistance to C19 due to recoveries from other coronaviruses.

We can reasonably infer that cross-resistance to C19 is most often either mild (cases are symptomatic but low-risk) or moderate (cases are asymptomatic). However, in some people the cross-resistance is likely to be strong, such that the practical effect is indistinguishable from specific resistance (e.g., the presence of antibodies that also work against C19). If we assume 5-10% of the US population has strong cross-resistance, the total estimate for Americans with specific resistance or strong cross-resistance approaches 30%.

Serology tests offer a directional double-check for our thesis. These tests provide only a partial view of population-level resistance as they do not (1) account for individuals whose lymphocytes (T-Cells) were effective without requiring specific antibodies or (2) capture those with strong cross-resistance. That said, we still see serology tests as an important indicator. A meaningful number of positive serology tests (5-15%) would be broadly confirmatory while a negligible count (1% or less) would cast doubt upon our thesis.

The results have been encouraging. In New York City, 20% tested positive for C19 antibodies. The figures were 5% in Los Angeles, 6% in Miami, 8% in Colorado, 9% in North Carolina, 11% in Madrid, and 17% in London.

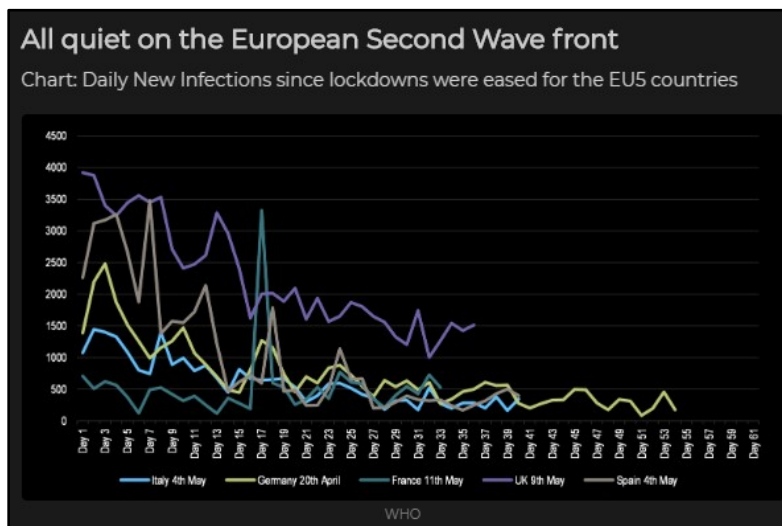
These numbers understate current resistance as antibodies don't appear until 2-3 weeks after infection and some of these surveys were completed 1-2 months ago. With serology results (even on a lag) showing 5% to 20% specific resistance – and population-level resistance likely far higher due to non-antibody resistance and cross-resistance – the evidence is strong that for the US/Europe, the disease break point is firmly in the rearview mirror.

6. As lockdowns end, won't we see a spike or "second wave" of infections?

The short answer is "no."

We don't see a scientific basis for the idea of a broad-based spike or "second wave" as society re-opens. This is consistent with two months of evidence from countries that have exited lockdown (see [Exhibit A](#)). While new infections may continue at a low level and localized outbreaks may occur in certain sub-populations, both the US and Europe are likely past their disease break points such that future COVID-19 infections will have great difficulty spreading (and be quickly contained) by the sheer force of population-level resistance.

Exhibit A



Sources: The Market Ear, WHO

Denmark’s Chief Epidemiologist Kare Molbak recently acknowledged as much. “No country has seen an actual second wave yet,” he stated at a public news briefing in May. “Some countries have seen the spread go up and down. But with the knowledge we have today, I find it very unlikely that we’ll see a second wave.”

This leads to an important observation. If a “second wave” is unlikely after lockdowns end, were lockdowns necessary in the first place? Our research indicates a counter-intuitive conclusion.

The lockdowns did not work.

And the evidence is more than convincing. We think it conclusive.

Let’s start with a story. In 1969, twelve men over-wintered in Antarctica at the British Survey Base on Adelaide Island, about 1,000 miles south of the Falkland Islands. After enduring 17 weeks in complete isolation with extraordinarily harsh outside conditions (temperatures reached -35 degrees Celsius with wind speeds of up to 100 knots), one of the men suddenly developed an upper RTI described as “a mild to moderately severe cold.” Over the next two weeks, seven more men would be affected. Symptoms on average lasted five days before recovery.

This curious, unexplained case was later detailed in a British medical journal. “The occurrence of a common cold during isolation, when the chances of introduction of a new infection from the outside are virtually nil, implies that in some way the virus persisted, either in the environment or in the men.” Even in the most extreme circumstance of quarantine and absolute isolation, a virus infected a man. Nature somehow found a way.

For the better part of a century, the epidemiological literature has recognized this by acknowledging the extraordinary difficulty of stopping disease spread in human populations.

In 2006, researchers at the Center for Biosecurity at the University of Pittsburgh Medical Center reviewed the evidence for non-pharmaceutical interventions (“**NPI’s**”) that could be employed to reduce the number of cases or deaths resulting from a flu pandemic. The researchers cited a World Health Organization (“**WHO**”) report published earlier that year and concluded some NPI’s are likely effective (isolation of sick people in hospitals and at home; hand-washing and respiratory etiquette) but that large-scale quarantine measures are inadvisable.

“There are no historical observations or scientific studies that support the confinement by quarantine of groups of possibly infected people for extended periods in order to slow the spread of influenza. A World Health Organization (WHO) Writing Group, after reviewing the literature and considering contemporary international experience, concluded that “forced isolation and quarantine are ineffective and impractical.” Despite this recommendation by experts, mandatory large-scale quarantine continues to be considered as an option by some authorities and government officials.

The interest in quarantine reflects the views and conditions prevalent more than 50 years ago, when much less was known about the epidemiology of infectious diseases and when there was far less international and domestic travel in a less densely populated world. It is difficult to identify circumstances in the past half-century when large-scale quarantine has been effectively used in the control of any disease. The negative consequences of large-scale quarantine are so extreme (forced confinement of sick people with the well; complete restriction of movement of large populations; difficulty in getting critical supplies, medicines, and food to people inside the quarantine zone) that this mitigation measure should be eliminated from serious consideration.”

Subsequent research echoed this view. In 2007, a study by the RAND Center for Domestic and International Health Security affirmed that mandatory social distancing measures are “not recommended” and there is “limited scientific evidence supporting such restrictions” in part because “predicting the effect of closing schools and workplaces is difficult, since infectious individuals may be displaced into other settings.”

The CDC’s official Pre-Pandemic Planning Guidance (published in 2007, reaffirmed in 2017) noted the “effectiveness of pandemic mitigation strategies will erode rapidly as the cumulative illness rate prior to implementation climbs above 1 percent of the population in an affected area.” A 2011 paper from the University of Oxford’s Centre for Evidence Based Medicine reviewed 67 studies regarding the effectiveness of physical interventions to interrupt or reduce the spread of respiratory viruses and also concluded that hygienic measures (frequent handwashing; isolation of the infected in hospital wards) can be effective but “there is insufficient evidence to support screening at entry ports and social distancing as a method to reduce spread during epidemics.”

In 2017, the CDC’s updated guidance on pandemic planning acknowledged the “limited empirical evidence” that community-based NPI’s are effective. And in 2019, the WHO agreed “the evidence base on the effectiveness of NPI’s in community settings is limited, and the overall quality of evidence is very low for most interventions.” The WHO listed a range of NPI’s as “not recommended” during a pandemic including home quarantine, entry and exit screening for infection in travelers, border closures, and contact tracing.

Our reading of the literature yields an important distinction: **suppression** vs. **mitigation**. Under specific conditions, a goal of disease suppression (eradication) may be merited and justify aggressive action. Such circumstances include (1) a limited number of cases, (2) widespread availability of diagnostics, (3) a defined and compliant population, and (4) available resources for implementation. With C19, this approach was feasible only in a few geographies – most notably the rich, island nations of Iceland and New Zealand where it was deployed effectively.

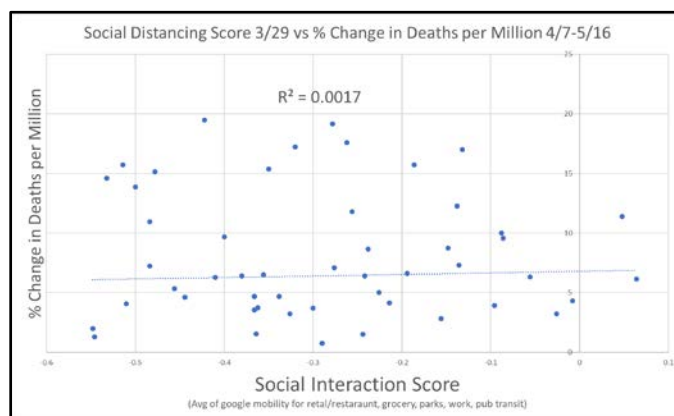
Once a disease is widespread (beyond the CDC’s 1% threshold), however, the sole course of action is disease mitigation. Halting the outbreak is impossible – it’s like trying to catch the wind. Some policy actions are sensible such as **isolating the sick, promoting hygiene and respiratory etiquette, aggressively monitoring and protecting vulnerable sub-populations, and scaling testing to support early identification and treatment of infections.** But for policies like contact tracing and lockdowns, the scientific literature is clear. They simply do not work.

Evidence from the COVID-19 pandemic is confirmatory.

Some countries had excellent results with no lockdowns (Taiwan, Iceland, South Korea). Others became “hot spots” even with aggressive lockdowns (Spain, Italy, the UK, the US). In Europe, fairly-measured per capita fatality rates for Sweden (no lockdown) and the Netherlands (half-lockdown) are indistinguishable from European peers.

The underlying data is even more compelling. We reviewed Google Mobility data on social interactions against subsequent changes to per capita fatality rates across US states. If lockdowns were effective, lower social contact should correlate with lower subsequent fatality rates. The data, however, shows nil correlation (see Exhibit B).

Exhibit B

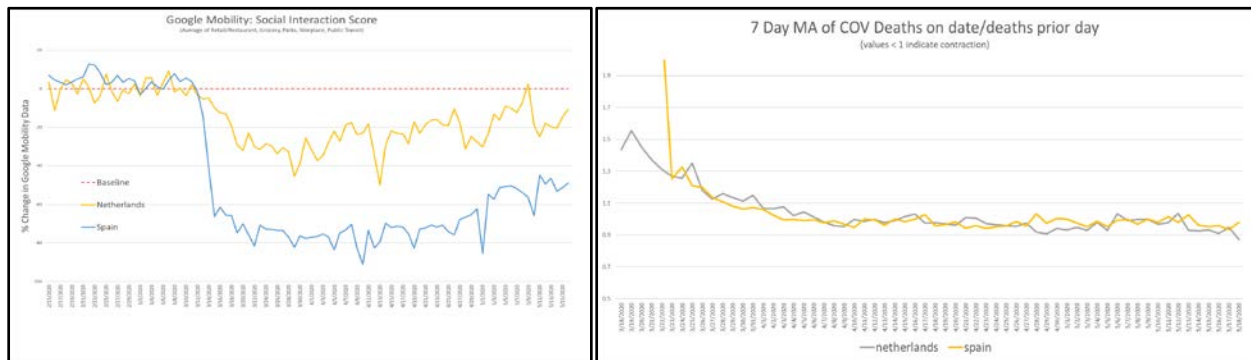


Sources: Manchester Management, Google Mobility, COVID Tracking Project

This is consistent with country-level data. The real-life A/B test of Spain and the Netherlands is particularly instructive. Both are in Western Europe. Both experienced similarly-timed disease trajectories (Spain reached one death per million on March 14; the Netherlands did so on March 16). Both provide data by incidence date (as opposed to reporting date) using similar guidelines, so the data is “clean” and comparable. Both implemented social distancing strategies within 24 hours of each other (Spain on March 14, the Netherlands on March 15).

The Google Mobility data shows a large gap in social interaction – Spain locked down hard, while the Netherlands implemented a far milder “intelligent lockdown” that maintained most social contact (see [Exhibit C](#)). The fatality data, however, shows a near-identical outcome as both countries experienced “peak deaths” on March 31 and a matching steady decline thereafter. Spain’s lockdown appears not to have reduced COVID-19 fatalities.

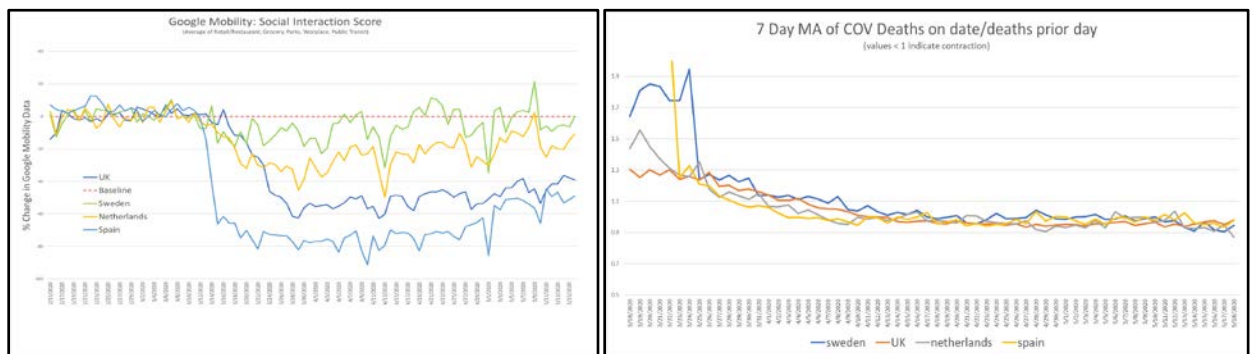
Exhibit C



Sources: Manchester Management, Google Mobility, National Public Health Agencies

To avoid the charge of cherry-picking, we add two more countries to the comparison that also report data by incident date: the UK (tough lockdown) and Sweden (no lockdown). The result? Varied social interaction scores, but near-identical COVID-19 outcomes as Spain and the Netherlands (see [Exhibit D](#)).

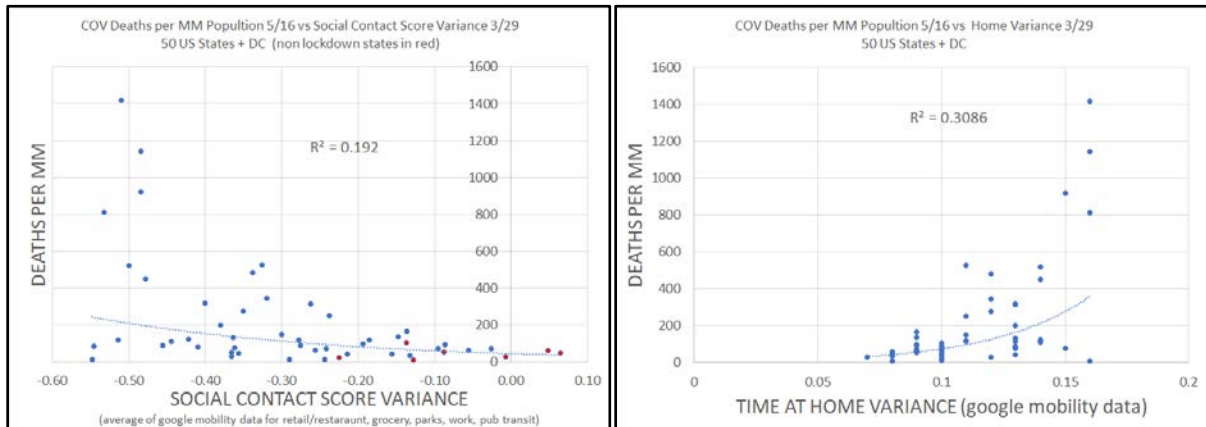
Exhibit D



Sources: Manchester Management, Google Mobility, National Public Health Agencies

A final observation from the Google Mobility data. Google breaks out social interaction by six activity types (retail/restaurant, grocery, parks, work, public transit, home). We reviewed this activity data compared to per capita COVID-19 death rates in the 50 US states. The result was powerful and non-intuitive (see [Exhibit E](#)).

Exhibit E



Sources: Manchester Management, Google Mobility, COVID Tracking Project

Counter to the prevailing narrative, engaging in activities outside the home is correlated with fewer deaths, while staying inside is correlated with more deaths. The relationship is modest but meaningful (R-Squared of 0.2-0.3).

Why would this be so? The answer, we believe, is that **viral dosage matters**.

As mentioned earlier, it is well-understood that the initial viral dose received materially affects the eventual health outcome. This is problematic for a lockdown strategy. Should one member of a household be infected, a quarantine maximizes other household members' exposure to the virus. If and when they subsequently fall ill, they likely experience higher initial viral loads – leading to worse health outcomes (more hospitalizations and deaths).

Anecdotal evidence is supportive. Reports on households with multiple ill family members often describe how the first infected person recovered quickly, but additional family members faced more serious infections. In May, New York Governor Andrew Cuomo shared that in a survey of 100 New York hospitals and 1,000 hospitalized C19 patients, 66% of new admissions had been sheltering at home. “This is shocking to us,” Cuomo said, “this is a surprise. Overwhelmingly the people were at home. We thought maybe they were taking public transportation, and we’ve taken special precautions on public transportation, but no, because these people were literally at home.”

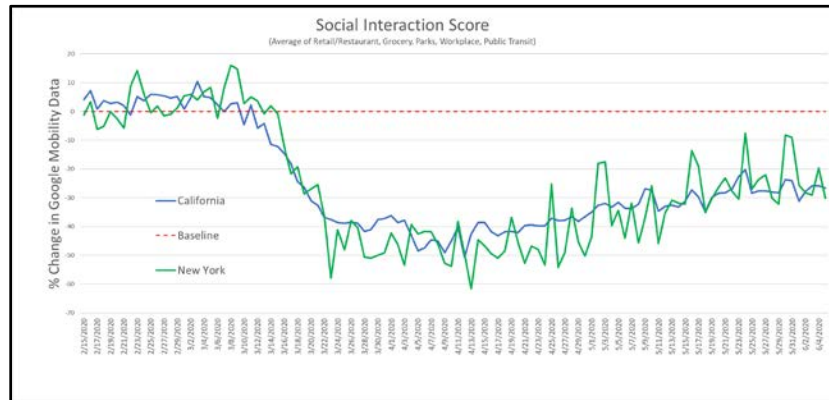
We do not find this shocking at all. In fact, it’s logical. COVID-19 is widespread. It cannot be put back in the bottle. The goal should be to minimize severe cases by protecting the vulnerable and taking sensible precautions.

Lockdowns do not do this. By confining people together and maximizing initial viral loads, lockdowns actually may lead to worse health outcomes. Correlation is not causation, so the activity data is suggestive but not confirmatory. Inverting the question, however, it is extremely rare for causation to exist without correlation – and these data sets show no correlation between lockdowns and reduced mortality. In our view, ending quarantine (especially with the disease break point behind us) is unlikely to lead to a “second wave” and may even reduce C19 fatalities.

7. What about places like Italy and NYC? Didn't California save lives vs. New York by locking down earlier?

The oft-repeated line that California saved lives relative to New York by locking down earlier and more aggressively is not supported by the evidence. Statewide stay-at-home orders were issued within 24 hours of each other (California on March 19; New York on March 20 for implementation on March 22). The Google Mobility data shows social interactions in each state moved in parallel during the pandemic (see Exhibit F).

Exhibit F



Sources: Manchester Management, Google Mobility

We do not view lockdowns as an explanatory variable in determining why some locations became C19 “hot spots” while others did not. Instead, we point to three key factors: (1) **demographics**, (2) **treatments**, and (3) **policy**.

Let’s first consider the outlier example of Northern Italy, which hit the susceptibility trifecta (making Northern Italy a particularly poor comparable to use when forecasting COVID-19 outcomes in other parts of the world).

Demographically, Northern Italy was highly vulnerable. C19 is a respiratory illness that poses a disproportionate danger to the elderly. Italy has the second-oldest population on earth. Northern Italy is tied with Poland for the worst air pollution in Europe. Households often include multiple generations. When Italians meet, they kiss each other on the cheek – twice. For COVID-19, the conditions were ideal for rapid spread to an at-risk population.

The problem was compounded by treatment missteps. The Lombardy region became a hot spot early in the pandemic (its capital Milan offered direct flights to Wuhan, China due to fashion industry ties) before its healthcare system could prepare or determine best-practice treatment protocols. In addition, the region’s hospitals generally operate at 90-95% ICU capacity (vs. 60-80% in the US), so a spike in cases quickly led to overwhelmed hospitals. This was magnified by a decision to treat most patients at hospitals (neighboring Veneto treated most patients at home with 1/3 the fatality rate). As if that wasn’t enough, Italy is known for poor hospital hygiene (30% of deaths from antibiotic-resistant bacteria in the EU each year occur in Italy). Nosocomial (in-hospital) disease spread quickly became rampant. Many of those who died likely only became infected once they reached the hospital.

But perhaps the most disastrous failure was a policy error. On March 8, in an effort to ease the burden on hospitals, Lombardy decided to send C19-positive patients back to nursing homes. The government even offered to pay €150 per patient accepted. The policy placed the ill with the vulnerable, literally spreading death. “We have become a hotbed of the disease,” said one Milan nursing home worker. “While it was immediately clear that elderly with pre-existing pathologies were the most vulnerable subjects in this epidemic, it was decided to turn care homes into COVID houses. We do not have intensive care and emergency rooms – what care could we provide?”

Unfortunately, this appalling policy was not only adopted in Italy. Which brings us back to New York.

In our view, New York’s worst-in-the-nation per capita COVID-19 death rate can be primarily attributed to (1) bad policy and (2) treatment missteps. On the former, New York issued an order on March 25 mirroring the Lombardy rule. The order has since been reversed – but the damage was done. The six US states that sent C19-positive patients to nursing homes represent less than 20% of the US population but over 60% of US COVID-19 deaths.

On the latter, New York’s treatment approach early in the outbreak was to aggressively intubate C19 patients in ICU’s. This was the wrong protocol. In a study of COVID-19 patients in NYC from March 1 to April 4, 86% of ICU patients were intubated and a staggering 78% of ICU patients died. During a similar time frame, a leading hospital

in Stockholm reported under 20% of COVID-19 patients in the ICU died. The Swedish protocol was to ventilate sparingly and focus instead on proning, oxygenation, and blood transfusions. The NYC study was published before all patient outcomes were known, so the final fatality figure was likely lower than 78% - but the directional conclusion is unchanged. Thankfully, the NYC standard-of-care in ICU's is no longer to intubate aggressively.

There are several important factors that influence the severity of the C19 pandemic in different geographies. The presence or absence of a lockdown, in our view, is not one of them.

8. What about the “second wave” that has been in the headlines?

Media outlets have recently proclaimed a “second wave” is now underway in the states of California, Florida, Arizona, and Texas. We certainly acknowledge positive tests are up in these geographies. That said, we think the singular focus on the absolute number of positive tests is simplistic and misleading – and misreads the data.

The immediate issue is that positive tests have lost predictive power. In March and April, positive tests were a useful leading indicator for future ICU admissions and deaths. Beginning in May, however, the correlation failed. Positive tests have fallen only modestly, but ICU visits and fatalities have plunged.

Why the disconnect? It all comes down to data. The headlines seem to confuse positive tests with infection spread, but the two are not the same. There are many variables that can and do distort positive test numbers.

Some states have dramatically expanded testing. Others have implemented contact tracing (which should, in theory, lead to more positives and a higher positivity rate). Some states report by specimen rather than by person, so a hospitalized patient may test positive for days in a row with each result counted as a unique positive. Some states mix PCR and serology tests together, conflating current cases with the recovered. Some states mandate that smaller facilities report positive tests, but do not require them to report negative tests. By failing to standardize COVID-19 reporting, the CDC has relegated “positive tests” to a muddled and unreliable metric.

In California, Texas, and Arizona much of the rise in positive tests relates to an increase in tests performed. Since the beginning of June, tests per day are +50% in California, +55% in Texas, and +70% in Arizona. The positivity rate has held steady at 4-6% in California for over a month – the “second wave” is simply the result of more testing.

Texas and Arizona (which have seen rises in positivity rate and ICU admits) may be seeing their “first waves” as they were yet to reach their disease break points. We also trace the higher case count to US citizens living in Mexico. The pandemic reached Mexico on a lag, and the country is now seeing its “peak” with a positivity rate above 50%. For the 1.5M American expatriates who reside there, it is common to return to the US for medical care. Recent media reports (see [Appendix B](#)) note C19-positive US citizens have been helicoptered as far north as San Francisco, with one San Diego hospital disclosing 60% of its C19 patients crossed from Mexico.

We reviewed 44 border counties (on the Mexican border or one away) with 5.7% of the US population. One month ago, these counties represented 3.5% of C19 positives in the US. Today they represent 13.1%. As the C19 outbreak in Mexico crests and declines in coming weeks, we see the pressure easing on US border states.

Florida is more complex to analyze due to data quality issues. The state has reported rapid positive test growth and a rising positivity rate. On the other hand, ICU's seem in good shape and death counts remain well below peak.

This leads to our key takeaway: positive tests only matter if they (1) lead to severe cases and deaths or (2) cause healthcare systems to be overwhelmed. For the “second wave” states, we don't think either outcome is likely.

On the former, the evidence is encouraging. The median age of a positive case in Florida has fallen from 52 to 35 since May 1. In Arizona's Maricopa County (population 4.5M), over 60% of new C19 positives are below the age of 44. Doctors and hospital administrators have reported younger patients, milder cases, and shorter hospital stays. Many C19 “hospitalizations” are folks in for unrelated elective procedures who test positive with no

symptoms. Hospitals rapidly discharge these low-risk individuals. Positive tests are up, but the likelihood of severe cases or deaths is down as these tests capture younger, healthier, and asymptomatic patients.

On the latter, we see little evidence of pressure on healthcare systems. In Florida's Miami-Dade county, available ICU beds are higher than three weeks ago. In Maricopa County, C19 ICU admissions remain 30% below the peak. In Texas, there are 4,400 C19 hospital patients with over 14,000 available hospital beds (out of 55,000 beds total, not counting surge capacity). For the US, CDC data indicates ICU's are operating at just 62% capacity with 46,000 available ICU beds. We estimate there are 9,500 COVID-19 ICU patients in the country today (7.8% of ICU capacity). For ICU's to run out of beds, severe C19 cases must jump 500% (without considering surge capacity).

We find such a scenario unlikely. The number of C19 patients currently in US ICU's is 65% below peak. In May, fatality numbers reached 2,700 in a single day. As of this writing, the 7-day trailing average just fell to a new low of 566 – a drop of 80% in barely more than one month. We empathize with every family who has lost a loved one, and recognize each of these numbers represents a person impacted by this terrible disease. But to us, the data is incredibly hopeful. Severe cases and fatalities have plummeted – and we think the trend is likely to continue.

There will always be another media article attempting to lure readers with a panicked and clickbait headline. But the facts are clear. More positive tests do not mean the infection is seeing a “second wave.” US hospitals have plenty of capacity. Fatalities have plunged and continue to reach new lows, even two months after US states began to re-open and the Google Mobility data started to rebound. We think we are headed in a promising direction.

9. Why have public health agencies like the CDC and WHO not conveyed a similar message?

In a perfect world, public health agencies would be independent and evidence-based. That is not reality. Both the CDC and WHO are fundamentally political organizations. Their leaders are politicians, subject to all kinds of outside pressures from countries, funders, pharmaceutical companies, media, and other special interest groups.

As a result, CDC and WHO guidance has often proven unreliable or outright incorrect during the pandemic. In January, the WHO repeated Chinese talking points by declaring it saw “no clear evidence of human-to-human transmission.” In March, US health officials encouraged Americans to go on cruise ships and told doctors surgical or cloth masks were “acceptable alternatives” to N95 respirator masks when treating C19 patients. The CDC and WHO have repeatedly flip-flopped on public mask-wearing, reinfections, travel restrictions, asymptomatic spread, the safety of hydroxychloroquine, and the allowable size of gatherings. Communication has been a total mess.

Unfortunately, issues at the CDC continue. Current CDC guidance lists three phases for re-opening. Phase One includes gyms, movie theaters, sit-down dining, sporting venues, places of worship, and dozens of other activities. Schools are not to re-open until Phase Two. The decision to keep schools and summer camps in the US closed is purely political. Children are highly resistant to C19 and not a primary vector for disease spread. Iceland, which has tested and traced more aggressively than any other Western nation, recently noted that “in all our data, there are only two examples where a child infected a parent.” A report by the public health agency in the Netherlands found “no indications that children younger than 12 years were the first to be infected within the family.” Schools across Europe have already re-opened. The CDC decision to delay school restarts is political, not evidence-based.

As of this writing, lakefront parks, beaches, and running trails in Chicago remain closed. COVID-19 perishes quickly when exposed to sunlight, heat, humidity, or wind. Outdoor transmission is rare. In addition, obesity and Vitamin D deficiency (from lack of sunlight) appear to be serious co-morbidities for C19 patients. Public health agencies should be actively encouraging outdoor exercise as a safe, practical approach to improving COVID-19 health outcomes. That this is even debatable shows the influence of politics over science, even on a local level.

Public health agencies simply do not have a strong track record during this pandemic. It has never been more important to do one's own homework and to rely on facts and data over headlines and “experts.”

10. Turning to your original thesis on the pandemic, why did you decide to reposition the portfolio in late-March?

Our thesis in late-March had three legs.

First, we were optimistic on **hospitals**. At the time, there was genuine fear that hospitals across the US would soon be overwhelmed with COVID-19 patients. Our analysis indicated this was unlikely and that the narrative would soon shift from “we’re running out of beds” to “we have plenty of capacity.”

Second, we were optimistic on the **infection fatality rate** (the “**IFR**”). Our research suggested that a combination of promising therapies and improvements to patient standard-of-care could lead to a meaningful decline in the IFR.

Third, we were optimistic on **economic stimulus**. We felt governments and central banks would do “whatever it takes” to support the economy (and financial markets) by unleashing overwhelming fiscal and monetary stimulus.

11. How has the first leg of your thesis played out, on hospitals?

It’s difficult to overstate the sheer panic that gripped the United States in March.

“We conservatively estimate this could require 48 million hospitalizations” in the US over the next 3-7 months, intoned University of Minnesota epidemiologist Dr. Michael Osterholm on a widely-disseminated podcast with Joe Rogan. “This is a very serious situation,” he added. “We will overwhelm the American health care system.”

Our qualitative and quantitative analysis indicated the opposite conclusion.

We spoke with hospital administrators. Their main concern was the lack of **rapid C19 tests**. Due to laboratory backlogs, C19 test results were often not available for 7-10 days (or longer). This stressed hospital resources, as patients with tests pending were treated as “presumed positives” requiring isolation, beds, masks, and time. With 85% of C19 results coming back negative, this wasted precious and significant hospital capacity.

Our research determined this was a logistics issue that would swiftly be resolved. Laboratories were expanding aggressively to clear their backlogs. Hospitals were rolling out faster tests, including newly-approved diagnostics from Danaher and Abbott that could provide results in minutes. Some hospitals we spoke with had already implemented in-house solutions with 24-hour turnarounds. It quickly became clear that the substantial burden on hospital equipment (PPE) and ICU beds from “presumed positive” patients would soon begin to abate.

We also identified the importance of **hospital surge capacity**. ICU’s generally operate at 60-80% capacity, but in a bad flu season it’s not uncommon for some hospitals to run out of beds (media articles are written on this subject every year). This is a feature, not a bug – hospitals are intentionally structured with this possibility in mind, and maintain detailed surge capacity plans to deploy as needed. By way of example, Texas Medical Center (a leading Houston-based hospital network) can increase its ICU bed count by 81%. Fewer “presumed positives” plus the deployment of surge capacity represented a powerful formula, in our view, to rapidly reduce the strain on hospitals.

Finally, we reviewed the **epidemiological forecasting models** driving public policy. We found them shockingly flawed. Politicians had gravitated to “boy who cried wolf” epidemiologists whose models seemed purpose-built to induce fear and hysteria. Our assessment indicated they had little basis in reality.

The worst of the lot was Neil Ferguson and his team at Imperial College in London. On March 16, Mr. Ferguson published a report estimating COVID-19 deaths reaching 510,000 in the UK and 2.2 million in the US. He also predicted peak ICU demand would be “over 30 times greater than the maximum supply in both countries.” The dire forecast led to cascading lockdowns first in the UK, and then across the US, as politicians panicked.

They should have looked more closely at Mr. Ferguson’s record of alarmist predictions. In 2009, Ferguson forecast tens of thousands of Brits would die from swine flu (H1N1). The actual number was 477. In 2005, Ferguson suggested 150-200 million people across the globe could die from bird flu (H5N1). The total figure was 282.

Politicians also should have looked more closely at Mr. Ferguson’s model. Ferguson assumed community NPI’s would be effective in a mitigation scenario, which had no basis in scientific literature. Ferguson assumed a 0.9% IFR, which we viewed as 4-10x too high. Ferguson assumed 81% of the population would be infected as C19 passed its herd immunity threshold, which showed a profound misunderstanding for how diseases spread in reality.

The underlying model (released to the public in May) was even worse. The code was 13-years-old, designed for influenza (not a coronavirus), failed to include important variables such as nursing homes, and was unable to produce the same results twice with identical inputs. The co-founder of a publicly-traded British software company called it a “buggy mess” and thundered that “in our commercial reality we would fire anyone for developing code like this, and any business that relied on it to produce software for sale would likely go bust.”

It is little surprise the Imperial College predictions proved a disaster. The model forecasted deaths in Sweden to be 100,000 by June. The total currently sits at 5,000. And Sweden’s definition of a C19 death is the most liberal on earth – the country cross-checks all reported deaths and if the person was diagnosed with COVID-19 in the prior 30 days, it is listed as a C19 death regardless of proximate cause (car accident, etc.). The Imperial College model missed Sweden by 20x. Its forecasts were a similar fiasco across the globe.

This is not the narrative painted by the mainstream media, but it is fact: the world locked down not due to careful, evidence-based science, but instead based on an error-riddled model with erroneous assumptions pitched by an academic with a track record for hysterical and wildly inaccurate predictions.

In the US, policymakers (especially US Governors) justified lockdowns with a model produced by the Institute for Health Metrics and Evaluation at the University of Washington (the “**IHME model**”). This model proved equally flawed. The IHME model is trend-following and extrapolates past outcomes into the future. This led to dramatic overestimates as the worst outbreaks (Wuhan, Italy, Spain) were projected onto US states. At the end of March, the IHME model predicted 104,000 hospitalizations in a week’s time. The actual figure was 39,000. The model missed by 200% in seven days. One analysis of the IHME model showed it missed the 95% confidence interval on roughly 70% of its forecasts. By definition, the model should miss such projections only 5% of the time.

A third prize goes to researchers at Columbia University who predicted 136,000 hospital beds would be required in New York City at peak demand in late-April. The actual maximum need was just over 18,000. Columbia University missed the mark by almost 700% in four weeks. The same researchers then doubled-down by predicting a sharp second-wave in Georgia after the state re-opened. Hospitalizations and deaths are lower two months later. There is little doubt Governor Cuomo had the Columbia University model top-of-mind when he said: “Now, people can speculate. People can guess. I think next week, I think two weeks, I think a month. I’m out of that business because we all failed at that business. Right? All the early national experts. Here’s my projection model. Here’s my projection model. They were all wrong. They were all wrong.”

With their models in tatters, these researchers have now turned to a new task: revisionist history. In recent weeks, (1) Columbia University published a study estimating 36,000 fewer people would have died in the US if lockdowns had been implemented a week earlier, (2) Imperial College published a study estimating 3.1 million deaths were averted due to lockdowns across 11 European countries, and (3) researchers at the University of California-Berkeley published a study saying community NPI’s prevented 500 million global C19 infections.

These “studies” are total nonsense. All three use modeled results (no actual data) based on the same models that showed zero predictive power during the pandemic. The Cal-Berkeley study was submitted on March 22. How could it judge the effect of community NPI’s when outcomes were still to be determined? That these studies were published at all (in science journals and the media) is an indictment of both academia and journalistic integrity. It is no wonder “experts” today are trusted so little by the people. Their arrogance is breathtaking.

This is not to say all epidemiologists failed the test. Public health leaders in Sweden, the Netherlands, Iceland, Denmark, and Switzerland have come through the crisis with reputations intact. Professor Sunetra Gupta, a University of Oxford epidemiologist, has been a forceful voice of reason throughout the crisis (a glowing Wall Street Journal profile of Professor Gupta is included in [Appendix B](#)). If only the world had listened to her.

In the coming months and years, epidemiologists (especially in the US) face the difficult task of unpacking how their profession failed so badly in a moment of global need – and what it can do to forge a better future.

The process has already begun in Europe. At the end of May, the Prime Minister of Norway admitted she closed schools because she panicked and “probably took many of the decisions out of fear.” The head of Norway’s public health agency confirmed that “our assessment is we could possibly have achieved the same effects and avoided some of the impacts by not locking down, but by instead keeping open but with infection control measures.”

By mid-April, it was clear that hospitals across the US not only had enough capacity, they were nearly empty. Hundreds of thousands of healthcare workers were furloughed. In NYC, the naval ship USNS Comfort and field hospitals in Central Park and the Javits Center closed after utilizing just a small fraction of their capacity.

And as for Dr. Osterholm’s “conservative” forecast of 48 million hospitalizations within 3-7 months? The total today in the US stands at less than 250,000. He missed by 200x.

12. How has the second leg of your thesis played out, on the IFR?

Conceptually, it’s important to understand **the COVID-19 infection fatality rate is not static**.

At the earliest stages of the pandemic, the COVID-19 IFR may have been 0.5-1.0% (a 1% figure was often cited). Today, however, the IFR is clearly lower. The CDC’s current “best estimate” for the IFR is 0.26%, assuming 35% of cases are asymptomatic. Using our 75% asymptomatic estimate (which we believe is better-supported by the data), the IFR falls to 0.1%. For comparison, the CDC’s midpoint estimate for the flu’s IFR from 2010-2019 is 0.09%. COVID-19 today appears to be more dangerous than the flu, but only modestly so.

How did the IFR decline so materially without a “cure”? The answer: lots of ways.

We think the standard-of-care in hospitals has improved dramatically. Early-on, doctors intubated patients to address low blood oxygen levels. Over time, it became evident the hypoxia was on a cellular level and not due to mechanical breathing issues. The standard-of-care shifted to proning, oxygenation, and blood transfusions as safer and more effective treatments. In addition, with time to prepare and better resources, hospitals have made tremendous strides reducing the nosocomial spread that initially was a primary vector for disease transmission.

Better therapies have also made a difference. **Plasma transfusions** (transferring blood from recovered patients to sick patients) have been employed for centuries to treat disease. Early in the pandemic, there was insufficient plasma for the treatment to be widely-used, but today plasma is readily available. Importantly, plasma has two potential mechanisms of action as it both provides C19-fighting antibodies/T-Cells and addresses blood hypoxia. Observational studies and anecdotal evidence suggest plasma may be an effective treatment tool.

We also are positive on the much-discussed hydroxychloroquine (“**HCQ**”) plus azithromycin (“**ZPAK**”) and zinc combination therapy (the “**combo therapy**”). HCQ has unfortunately been politicized, but our view is optimistic. The known properties of HCQ and ZPAK provide a solid basis for a hypothesis of efficacy against C19.

It appears COVID-19 deaths are not predominantly caused by C19 itself, but instead by another illness (such as pneumonia) or cytokine storms (an out-of-control overreaction by one’s own immune system). The combo therapy may be helpful on both fronts. HCQ is an anti-viral and ZPAK is a macrolide antibiotic with well-documented anti-viral properties for respiratory infections. Together, they may pack a strong one-two punch against both COVID-19 and other bacterial/viral illnesses that are present, such as pneumonia. In addition, it’s

logical the combination would deter cytokine storms, as HCQ is an immunosuppressant and ZPAK has anti-inflammatory properties. It is often the inflammation caused by a cytokine storm that constricts airways.

Importantly, both drugs are cheap, widely available in generic form, have been FDA-approved for decades, and have well-understood side effects. HCQ and ZPAK are frequently prescribed together for Lyme Disease.

Criticism of the combo therapy appears unfounded. A few studies have questioned the safety of the treatment. We note these studies prescribed chloroquine (HCQ's harsher cousin) in triple the recommended dosage. Other studies have shown no benefit when HCQ is taken prophylactically or in severe cases. Neither should be a surprise – as an anti-viral, the combo therapy is likely most effective as a first-line treatment post-infection.

Studies suggest when prescribed appropriately, the combo therapy may reduce viral loads, hospitalization rates, and deaths. Doctors have certainly noticed: three of the top-four most-prescribed treatments for C19 are HCQ, ZPAK, and plasma. President Trump (a Republican) and Chris Cuomo (a Democrat) both took the combo therapy post-exposure/infection (Cuomo deflected attention by listing “quinine”; HCQ is quinine's synthetic version).

Other factors may also contribute to the falling IFR. Epidemiology 101 suggests the most vulnerable tend to perish early in an outbreak, leaving behind a more resilient population that should naturally experience a lower IFR. Diseases also tend to weaken when they mutate (unlike in Hollywood movies) as more-severe versions kill their hosts while weaker versions spread more readily. It is possible C19's lethality is diminishing over time. Finally, with unprecedented global resources committed to improving treatments for COVID-19, we are confident health outcomes will continue to progress through new practices and new therapies.

Whatever the precise causal combination, the evidence is clear: the IFR for C19 is materially lower today. The data reflects this with C19 hospitalizations falling quicker than C19 cases, and C19 fatalities declining fastest of all.

13. How has the third leg of your thesis played out, on economic stimulus?

My father spent thirty years in the retail jewelry industry. Together with his brother (my uncle), he helped grow a small family business with a handful of local stores into a national chain with over 300 mall-based locations.

I once asked him, “if you could own any jewelry store in the country, which would you pick?”

His answer was not the store in the wealthiest neighborhood. It was not the store in the best mall. It was not the store with the most foot traffic. It was not the store in the best location. It was not the store with the best manager.

The best jewelry store, he told me, is the one in a location that just survived a hurricane. Because after a hurricane, the money pours in. The region experiences an economic boom. And yes, people buy food and toilet paper and rebuild homes and businesses. But governments err on the side of doing too much rather than too little. And when the boom arrives, a lot of people end up buying jewelry. That's the store he'd pick – ten times out of ten.

This pandemic is an unprecedented natural disaster.

It's a hurricane.

And that's how politicians are reacting to it.

The US has passed four stimulus packages totaling trillions in spend, with a fifth package likely coming soon. The Fed is spending trillions more. Across the world, previously unthinkable events are underway. Germany – the “balanced budget capital of the world” – has ditched austerity to endorse vast deficit spending at home and abroad. Germany has also joined up with France to recommend a half-trillion Euro rescue fund that looks an awful lot like a first step towards European fiscal union. Taboos are no longer. “Whatever it takes” is the watchword of the day.

Some question whether governments have enough firepower. We are confident they do.

In March, President Trump fielded a question following passage of the third US stimulus. “It’s \$6.2 trillion,” he responded. “And we can handle it easily because of who we are, what we are. It’s our money. It’s our currency.”

This statement from the President is nothing less than a full-blown endorsement of Modern Monetary Theory (“**MMT**”), the idea that deficits do not matter so long as a country controls its own currency.

Whether this is good long-term policy is irrelevant. The successful investor sets aside personal biases and sees the world as it is, not as he/she wishes it to be. The President believes the US has unlimited capacity for fiscal stimulus. Just as important, Fed Chairman Powell agrees – and echoes that the Fed is similarly unbounded. There’s “really no limit” to what the Fed can do, Powell says. “We’re not out of ammunition by a long shot.”

He’s right. In an MMT world, all things are possible. Fiscal stimulus is unlimited. Monetary stimulus is unlimited. The Fed’s arsenal is full – it can buy any asset, make any loan, backstop any entity. Interest rates can go negative. Junk bonds and even equities can be added to the Fed balance sheet. There are no limits.

This is an election year. Governments have more than enough firepower to fight this crisis.

They will not fail.

14. That’s a lot of stimulus, but the economic damage seems pretty bad. Do you really think we can recover?

More than 20 million Americans lost their jobs in April as the unemployment rate rocketed to a level not seen since the Great Depression (14.7%).

But employment figures are only part of the equation. For even as jobs plunged, US household income surged +10.5% (the largest year-on-year jump in decades) and the US personal savings rate hit 33.0% (the highest level since the US Department of Commerce began tracking the data series in 1959).

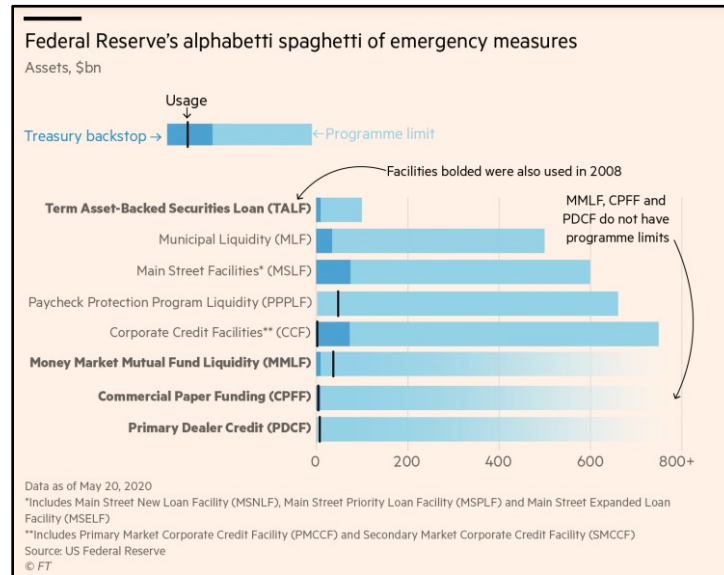
How did this happen? The answer is simple: stimulus works (at least in the short-term).

Two policies played an outsized role. The first was a \$600/week federal add-on to unemployment insurance that runs through July 31. The legislation’s goal was to broadly replace 100% of lost income during the period. The second was a direct cash payment program with an average US family of four receiving \$3,400. The combination boosted personal income at the same time spending fell due to the lockdowns. The personal balance sheet of the average American did not weaken – it strengthened materially. With extra savings in hand, a healthy US consumer (representing two-thirds of US GDP) is poised to drive a demand-led recovery as the economy re-opens.

That does not mean we’re going “back to normal” overnight. There will be winners and losers. Some businesses will restructure or go bankrupt. Some folks will lose their jobs.

But the banking system is well-capitalized – and the bulk of employment losses are in sectors likely to benefit from a cashed-up consumer and pent-up demand for goods and services (retail, hospitality, healthcare). Most importantly, governments and central banks are likely to err on the side of doing more, not less. “When the economy begins to rebound,” claims Fed Chairman Powell, “we’ll be there to make sure it’s as strong as possible.” As the Financial Times recently noted, the Fed has only just begun to deploy its sizable firepower (see [Exhibit G](#)).

Exhibit G



Source: Financial Times

We think politicians will keep pressing the pedal to the floor until they know they've won.

And even then, they will press the pedal down one more time...just in case.

15. Given your thoughts on a recovery, have you covered all your short positions?

No. Favored opportunities include structurally-challenged businesses that have benefitted from higher demand during the pandemic – but where the boost may reverse as society re-opens.

A good example is **The Clorox Company (NYSE: CLX)**. Clorox faces pressure from the long-term trend towards private-label as well as from start-up brands bypassing traditional distribution channels and going direct-to-consumer. As a result, Clorox's revenues and operating profit have grown barely +2% per year for the last decade, including acquisitions, and the company has consistently missed expectations while underperforming peers (CLX lost share in 64% of its categories in 2019). During the C19 pandemic, demand for Clorox's cleaning products (about 40% of revenues) has surged, leading to higher profit guidance and a share price at record levels. We do not think this is sustainable. At a recent price above \$200/share, investors are paying 30x earnings for a business that may see earnings decline for years from the "pandemic peak" when structural headwinds re-emerge. As one-time spring 2020 pantry-loading fades into the rearview mirror, we see downside to \$125-150/share.

Another core short position is **zooplus AG (Deutsche Bourse: Z01)**. zooplus is the leading online retailer for pet supplies in Europe. During the C19 pandemic, zooplus has benefitted from online demand and lower digital advertising costs (as "cost per click" declines in a recession). We think neither shift is permanent. Europeans tend to shop for pet supplies not in specialty stores (as Americans do) but instead at mass-market retailers. As European businesses re-open, we see sales swinging back to this traditional channel as consumers find it a better experience to shop where they are already buying. zooplus also faces strenuous competition from Amazon (which has declared European pet supplies a key focus area) and zooplus's product portfolio is commoditized. In an economic recovery, we think zooplus's race-to-the-bottom business model (profitless growth) is likely to re-appear. In this scenario, the shares could be cut in half from a current price of about 140 Euros/share.

16. What about the long book?

We are positioned for a recovery. We've recently purchased shares in a number of great businesses with strong balance sheets that we believe have material upside as the economy emerges from the pandemic.

One current theme is **US foodservice distributors** which provide food and related products (cookware, kitchen equipment, tabletop items) to restaurants, healthcare and education facilities, lodging establishments, and other clients. Foodservice distribution is a wonderful business – with thousands of suppliers and hundreds of thousands of customers, distributors have the negotiating muscle to consistently earn superior returns. Due to the lockdowns, foodservice distributors have seen their sales decline and their stock prices crater. We view this as an attractive opportunity to purchase quality long-term franchises that should rebound strongly as restaurants and other establishments re-open. Holdings include Sysco Corporation (NYSE: SYY), Performance Food Group Company (NYSE: PFGC), US Foods Holding Corp. (NYSE: USFD), and The Chefs' Warehouse, Inc. (NASDAQ: CHEF).

Another recent addition is **C&C Group PLC (LSE: CCR)**, a company we successfully owned once before in 2011-2012. C&C Group is a manufacturer and distributor of alcoholic and non-alcoholic beverages in the UK and Ireland. As roughly 75% of the company's business is to the "on-trade" (restaurants and bars), C&C Group has recently suffered a steep decline in its results. That said, C&C Group has an extraordinarily strong balance sheet – at its current rate of cash burn, the company would not run out of money until 2027 – and we are confident the UK Government will support its iconic pubs and the British people will continue to drink a good pint or three as those pubs re-open in coming weeks. We see +100% upside to the shares from today's price just above 200p.

17. It seems you are pretty optimistic! Any final thoughts?

Two years ago, I attended the Berkshire Hathaway annual meeting in Omaha, Nebraska with my mom. When the meeting opened, Warren Buffett told a story about the first time he ever purchased a stock.

The date was March 1942. "It was three months after we got involved with World War II," he said, "and we were losing – the headlines were full of bad news about the Pacific."

"But everyone knew eventually we'd win the war. Our system has worked in America since 1776. You didn't have to understand accounting or look at stock market quotations – all you had to do was figure that America would do well over time, and that American business would in turn do well and overcome difficulties."

"The overriding question is – how will American business do over our investing lifetimes? We have the greatest tailwind one could ever imagine by living in this country."

Today's headlines look scary. But we are optimistic.

This is America, the richest and most technologically advanced nation in the history of humanity. Our physical and human capital is unmatched. There is no problem we cannot overcome. In the coming weeks and months, our country will find a way to not only survive this pandemic, but emerge stronger from it.

We agree with Warren. America – and American business – will do amazingly well over our investing lifetimes. In a moment of gloom and doom, it is important to keep this in mind. This country has survived all kinds of crises over the last 250 years, and it will survive this one too. Be optimistic. America's best days still lie ahead.

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