[7] Appendix materials

This appendix contains a mix of epidemiological, healthcare and economic data, and some history on the Spanish Flu.

[i] Select healthcare comparisons by country

[ii] Cumulative tests performed vs reported cases and mortality

[iii] The problem with predictions

[iv] The Spanish Flu (1918-1920) in context, and why it's a poor proxy for COVID-19

[v] A synopsis of communicable disease infectiousness and mortality

[vi] How long can viruses like COVID-19 last on hard surfaces?

[vii] Could the onset of spring and summer slow virus transmission rates?

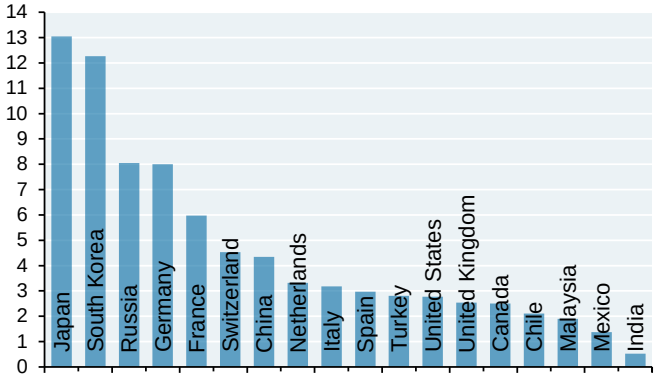
[viii] What does it take to control an outbreak? Aggressive isolation and "contact tracing"

[ix] What are "reproductive numbers" used to describe communicable diseases?

[x] Rapid response times, China and information repression

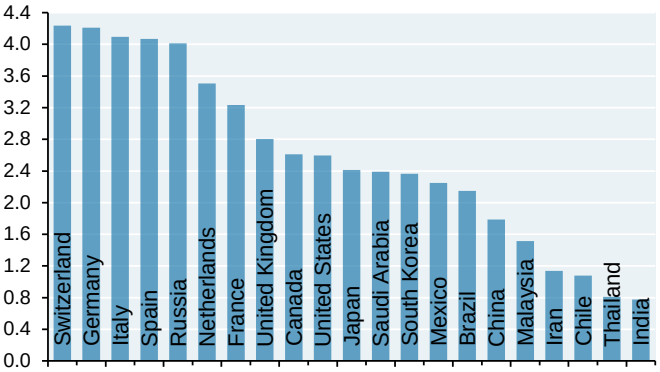
[i] Select healthcare comparisons by country

Hospital beds per 1,000 people



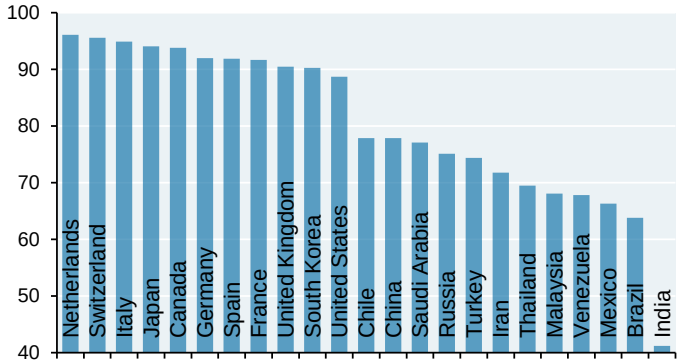
Source: Our World In Data. 2018.

Physicians per 1,000 people



Source: The World Bank. 2018.

Healthcare Access and Quality Index



Source: Institute for Health Metrics and Evaluation. 2016.

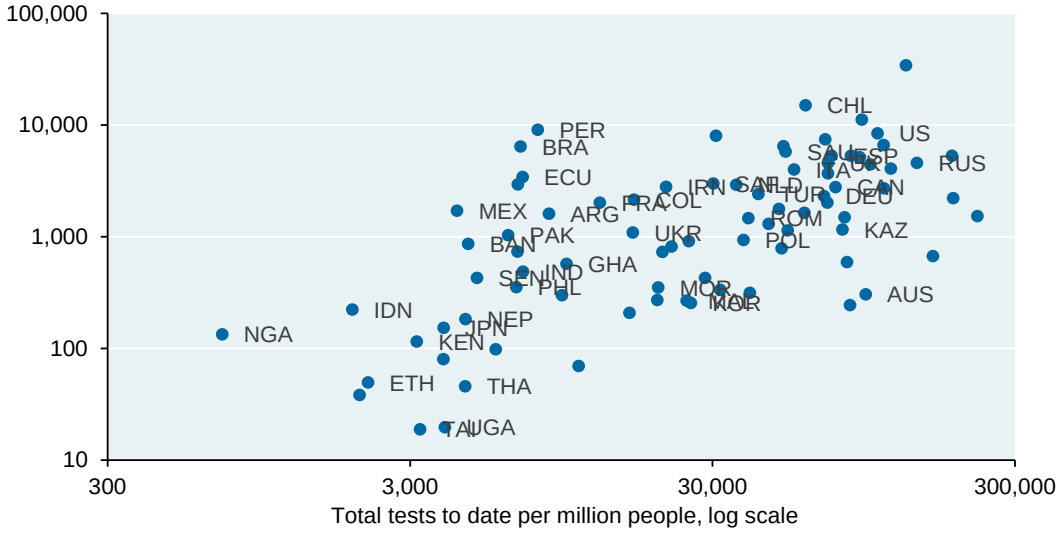
[ii] Cumulative tests performed vs reported cases and mortality

The US has finally caught up to Korea with respect to tests performed by million people…but the US should arguably be doing much *more* testing than Korea, since the US cumulative infection rate is one entire order of magnitude higher. There are important caveats to keep in mind on testing:

- Some report tested individuals and others report total tests which include the same people twice
- Some country tests are reported at regular intervals and others are not
- Some sources include "suspected" and "pending"; others do not
- Some countries only report public labs, others report public and private
- Some countries have stricter eligibility requirements to get tested than others
- For all factors, countries sometimes change their approach, rendering time series comparisons less meaningful

Testing and reported cases

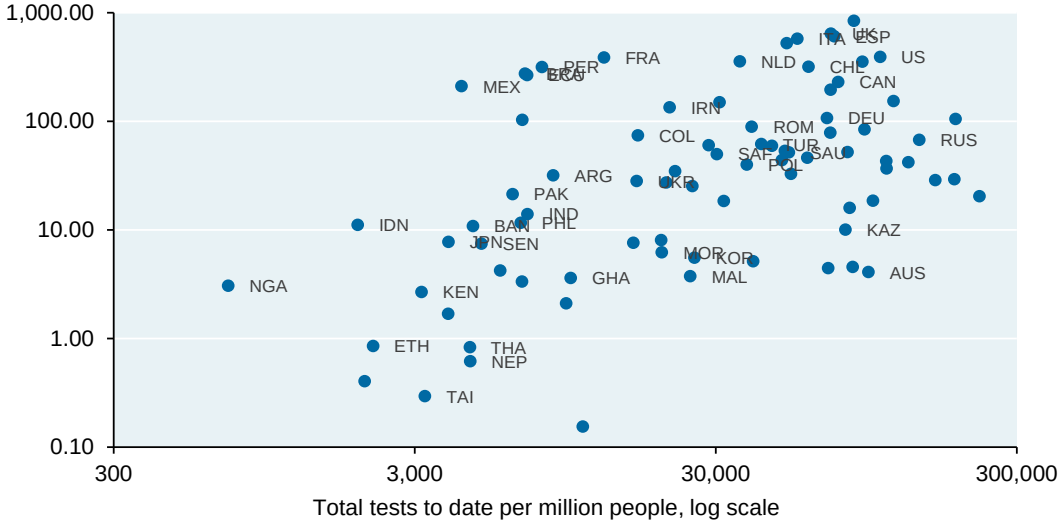
Reported cases to date per million people, log scale



Source: Our World in Data, JPMAM. July 7, 2020

Testing and mortality

Reported deaths to date per million people, log scale



Source: Our World in Data, JPMAM. July 7, 2020

[iii] The problem with predictions

Why aren't we predicting infections for COVID-19? Because by the time the models actually work, you already know the answer [Warning: only for those of you who like math]

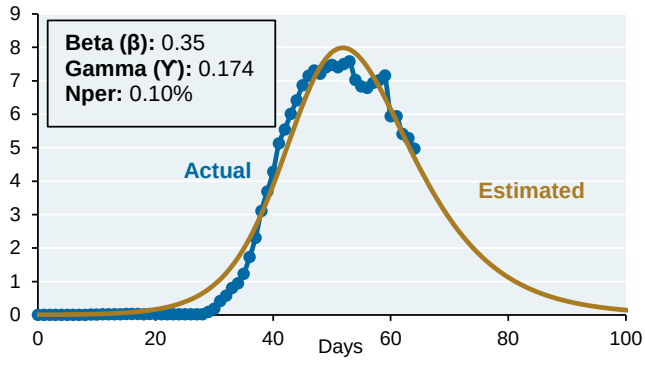
You might have seen infection prediction curves floating around for different countries. We have not found a lot of value in this exercise. The best way to explain why is with a model first applied to Korea in mid February, and then in vain to other countries.

Many epidemic outbreak models are based on the Kermack/McKendrick "SIR" model developed in the 1920's, which refers to "susceptible, infected and removed". The model estimates the number of active infections out of a given exposed population. Active infections rise based on new infections, and fall due to recoveries and mortalities. The three primary inputs are infectiousness (beta), removal rates (gamma) and the size of the exposed population as a % of the total population in a given region (Nper).

However, while this sounds very scientific, there's a lot of manual curve-fitting going on. One reason: it's hard to predict reported infections for a very infectious disease when large numbers of infected people are asymptomatic or for other reasons not reported, since the model will need to somehow reconcile fewer reported cases than it expects.

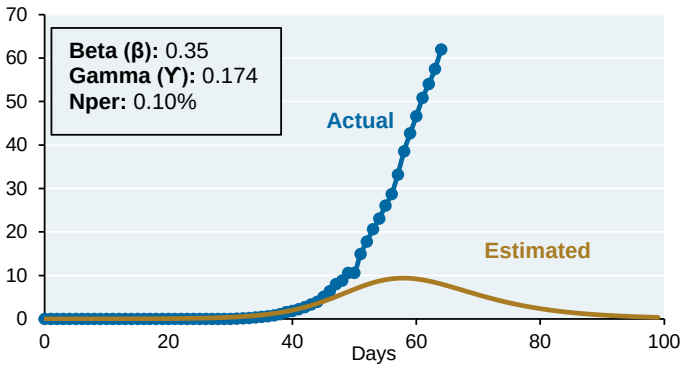
In any case, let's start with Korea. The first chart (left) shows how our model[1] could have been applied to Korea in mid-February with a given set of assumptions. Looks great, right? Don't get too excited. While it worked for Korea, the calibrated parameters proved to be **completely useless** in forecasting infections for Italy. The second chart shows what mid-February Korea parameters would have predicted for Italy (peak active infections of 9,000), compared to what has *actually* happened (62,000 active infections so far). **This massive estimation failure is not hard to understand**; the Korea parameters were fit for a country whose policy and behavioral dynamics were completely different than Italy.

A good fit for Korea in mid-February
Active infections in Korea, thousands



Source: Johns Hopkins. J.P. Morgan Asset Management. March 26, 2020.

Korea parameters: a really, really bad fit for Italy
Active infections in Italy, thousands

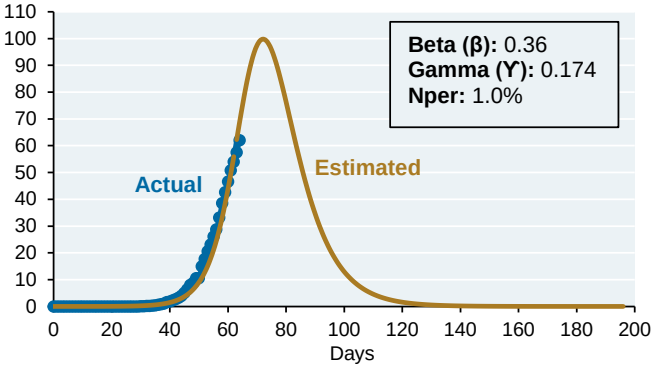


Source: Johns Hopkins. J.P. Morgan Asset Management. March 26, 2020.

[1] My son Max, who will be attending the Harvard School for Applied Computational Science in the fall, helped with this section. My models are typically written in Excel's Visual Basic. He's dismissive of VBA, so I told him that I consider VBA the programming language of the gods. His response: "yes, but it would be the programming language of gods of a society that became extinct hundreds of years ago". If you were a computer science major, you would find this exchange to be hilarious. Max writes everything in Python.

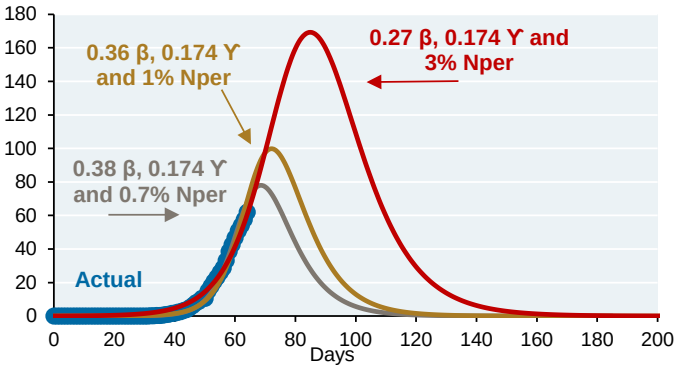
After seeing how poorly the model performed for Italy, we could have waited a couple of weeks and recalibrated its parameters to fit Italy better, which is what the next chart shows on the left. Much better fit; however, we had to increase one of the parameters by a factor of 10x (!!). **And furthermore, what good is this tail-chasing exercise**, since (a) the revised calibration may well be useless for countries other than Italy, and (b) to make matters worse, **even this new recalibrated Italy curve could be *completely* wrong too** since there are other curves with more severe infection parameters that fit the actual Italy data just as well. That's what is shown in the chart on the right; who's to say which of these curves is the right one if they all fit the actual data so far??

The model, recalibrated for Italy: 10x higher infected population, Active infections in Italy, thousands



Source: Johns Hopkins. J.P. Morgan Asset Management. March 26, 2020.

Which set of assumptions best predicts Italy? Still totally unclear, Active infections in Italy, thousands



Source: Johns Hopkins. J.P. Morgan Asset Management. March 26, 2020.

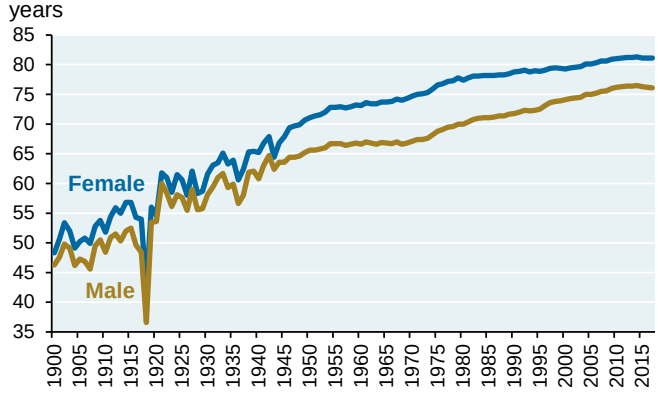
The bottom line: infection prediction models must be constantly updated to fit the observed actual infection curve in each country[2]. As a result, what you learn by fitting parameters for one country has practically no value in predicting the evolution of infections in any other country; and the predictions *within* any given country can shift wildly with the level of testing and policy changes. The best these models can do is provide a very rough estimate of potential infection trajectories for a single country assuming that public policy, testing and behaviors do not change over time, and even then, they could be totally wrong. These models are most accurate when infections are shown to have already peaked, at which point they become redundant.

[2] By the way, you don't even need a fancy SIR model to fit infection curves; we replicated the Korean infection curve with similar precision by simply using a modified version of the formula $y = \exp(-x^2)$.

[iv] The Spanish Flu (1918-1920) in context, and why it's a poor proxy for COVID-19

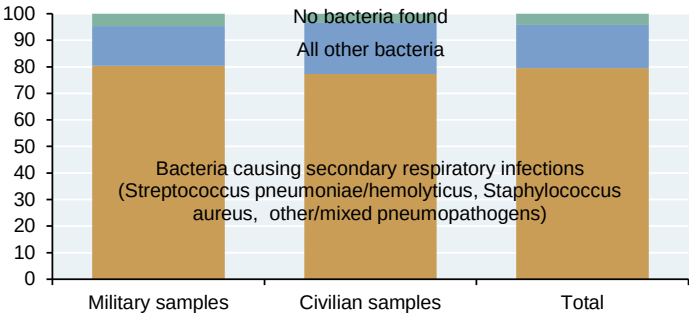
- **No vaccine was ever developed to combat the Spanish Flu**. In contrast, a SARS vaccine was developed in response to the 2002 outbreak but was never used since public health measures (closing workplaces, people working at home, etc) got the disease under control by May 2003 before the vaccine was ready[3]. Since that time, Harvard scientists have found the antibodies which block SARS and MERS from entering human cells[4], which were used to develop antibody therapies (which are different from vaccines, which are the treatment of choice and much cheaper to produce). The new COVID-19 virus shares 86% genetic similarity with SARS, so scientists aren't starting from ground zero
- **There were no antibiotics in 1918** to treat secondary bacterial infections associated with influenza. From a paper marking the 100th anniversary of Spanish Flu: "in 1918, most severe influenza-associated pneumonias were associated with **secondary bacterial infections**… high pandemic case fatality during the fall 1918 pandemic resulted primarily from increased frequency, and not increased severity, of secondary bacterial pneumonias, especially in young adults"[5] (see chart). Furthermore, without secondary bacterial pneumonia, "experts generally believed that most patients would have recovered". The first antibiotic was discovered in 1929 but mass antibiotic production did not occur until the 1940's
- The US CDC reports that lab experiments with recombinant influenza viruses containing genes from the 1918 virus suggest that the 1918 and 1918-like viruses would **be as sensitive as other virus strains** to FDA-approved anti-influenza drugs rimantadine and oseltamivir
- **As further indication of a world without antibiotics and other healthcare innovations such as anti-virals, ICU-level hospital care, ventilators, etc,** the US life expectancy for men and women ranged from 50-55 years before the onset of the Spanish Flu[6] in 1918

US life expectancy at birth



Source: US CDC; Andrew Noymer, Public Health Dep't, UC Irvine. 2020.

Spanish Flu (1918): % of cultures containing bacteria causing secondary respiratory infections/pneumonia



Source: "*Predominant Role of Bacterial Pneumonia as a Cause of Death in Pandemic Influenza*," National Institute of Allergy and Infectious Diseases, Morens et al, 2008

[3] China began clinical trials of a SARS vaccine in November 2003, while in the US, the first human SARS trials began in December 2004, conducted by the National Institute of Allergy and Infectious Diseases

[4] Harvard professor Wayne Marasco identified a single antibody out of a 27-billion antibody library that blocked the SARS virus from entering human cells. Marasco is actively testing new antibodies in search of one that will have the same effect on SARS-CoV-2 (COVID-19)

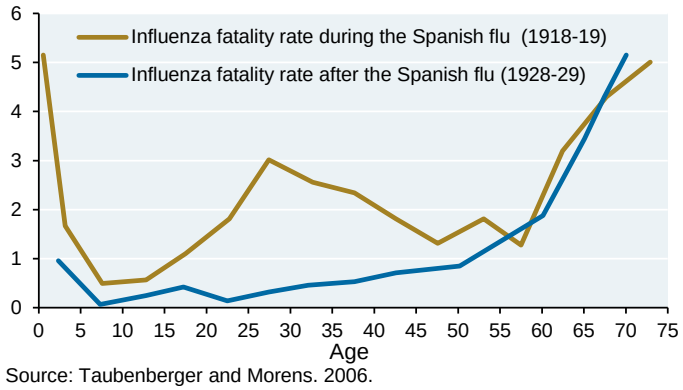
[5] "*The 1918 influenza pandemic: 100 years of questions answered and unanswered*", Taubenberger et al, Viral Pathogenesis and Evolution Section, Laboratory of Infectious Diseases, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Science Translational Medicine, July 2019

[6] Male life expectancy at birth was ~25 during the Roman Era; rose to ~33 by the Middle Ages; and hovered between 30 and 40 until the late 1800's. Starting in the 1920's, the innovations cited above ushered in the most remarkable improvement in life expectancies in the history of the world

Spanish flu mortality rates

An unusual feature of the 1918 Spanish Flu was its mortality pattern in which young adults were at high risk, something not observed in influenza outbreaks before or since. Usually, influenza mortality is "U-shaped," with higher mortality in the very young and the very old, and with low mortality in healthy persons in between. The 1918 mortality curve was "**W-shaped**", with an additional mortality peak from ages 20 to 40[7]. This has higher negative consequences for growth, employment and household formation.

Case fatality rate during and after the Spanish flu
Fatalities per 100 infected persons, US



Source: Taubenberger and Morens. 2006.

SARS vs COVID-19

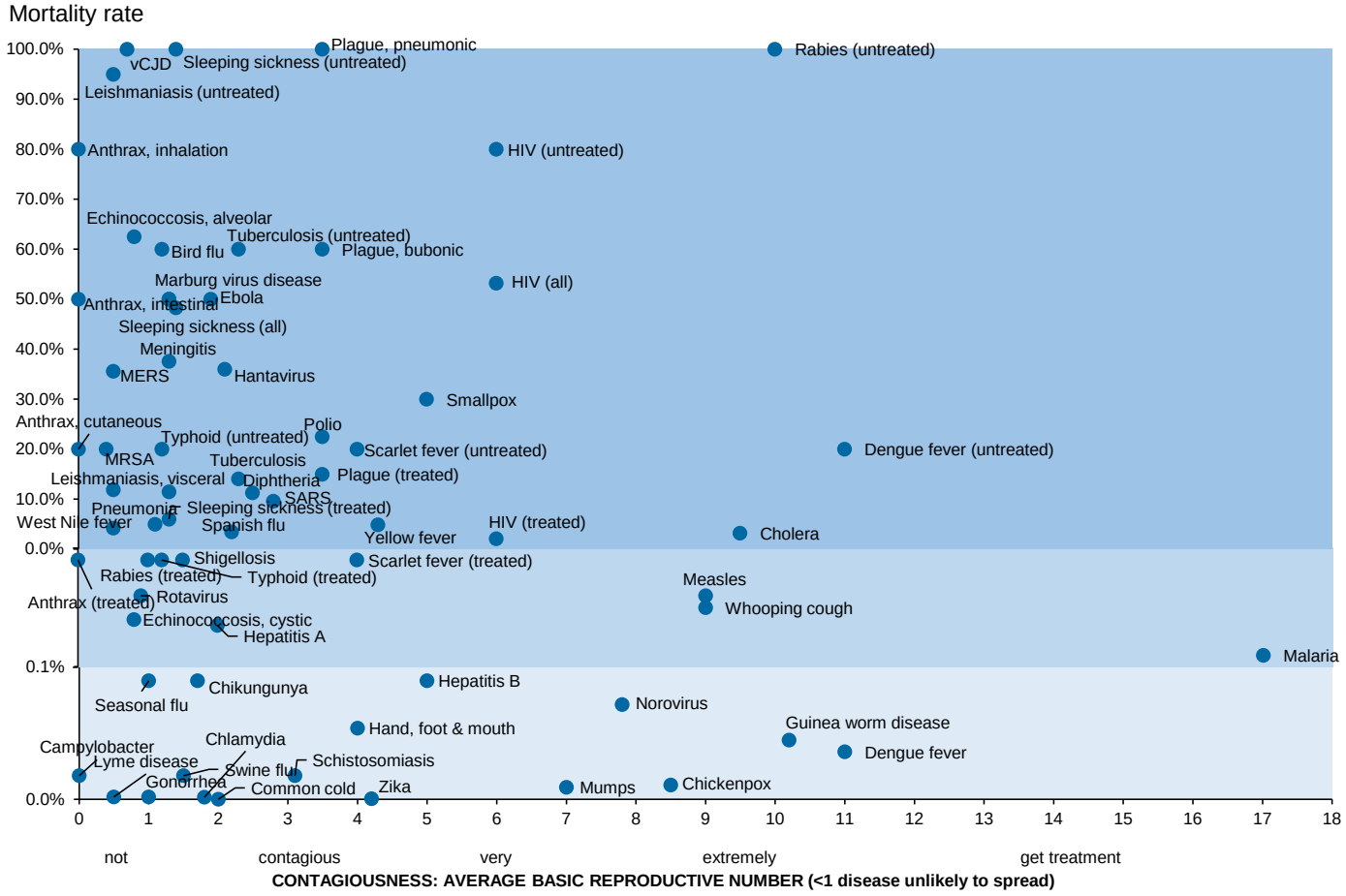
- In 2003, SARS was eventually contained by surveillance, isolation of patients, strict enforcement of quarantine of all contacts, and in some areas community-level quarantine. By interrupting human-to-human transmission, SARS was eradicated. Isolation was effective for SARS because peak infectiousness occurred ***after*** patients were already very ill with respiratory symptoms and could be easily identified. Although asymptomatic patients were reported for SARS, **no known transmission occurred from these patients**
- The new virus SARS-CoV-2 (which causes the COVID-19 disease) has 86% similarity with the 2002 SARS-CoV virus, and both have median incubation times of ~5 days and basic reproductive numbers of ~2.2. But that's where the similarities may end: a paper from the International Journal of Infectious Diseases[8] may explain why SARS-CoV-2 is spreading more rapidly. The SARS-CoV-2 serial interval (the time it takes for an infected person to become contagious) may be just 4.5 days, which is less than its incubation period (i.e., when symptoms occur). **That means that asymptomatic individuals could be contagious before they know they have the virus.** If that's the case, that's quite different than SARS, since isolation of severely ill COVID-19 patients at the time they show up at health-care facilities would be too late

[7] **More on the W-shape**: the 1918 virus had high virulence that lower only in patients born before 1889, perhaps because of exposure to a then-circulating virus capable of providing partial immune-protection against it. This might only be the case for people old enough to have been infected during that prior era. Source: "*1918 Influenza: the Mother of All Pandemics*", Taubenberger and Morens, National Institute of Health, January 2006.
[8] "*Serial interval of novel coronavirus infections*", International Journal of Infectious Diseases, March 4, 2020

[v] A synopsis of communicable disease infectiousness and mortality

In the chart below on communicable disease mortality and transmission rates (the initial basic "reproductive number"), the mortality axis (Y axis) is not linear towards the bottom in order to highlight differences in diseases with much lower mortality rates. We have not plotted COVID-19 on this chart, since its reproductive number and mortality rate are not clear yet. Many papers estimate its initial reproductive rate at 2.5, but other estimates are as high as 5.0. And on mortality, we have seen a very wide range across countries, and still do not have an accurate sense of true infection levels to allow for computation of a case fatality rate; Section 2 shows a chart with mortality rates by country to date based on reported infections.

Diseases by contagiousness (reproductive number) and mortality



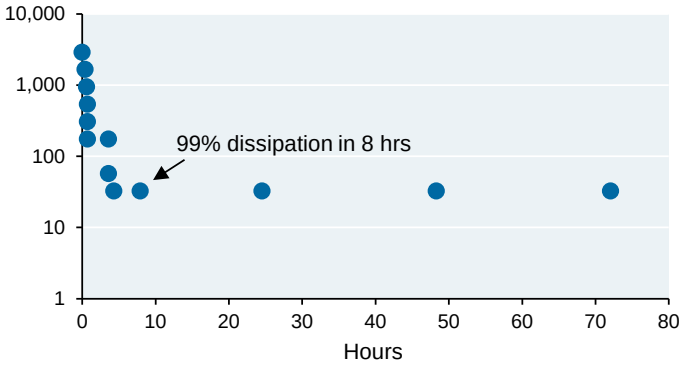
Sources include: Food and Drug Administration, National Center for Biotechnology Information, Global Health Data Exchange, Cambridge, eMedicine, Chinese Center for Disease Control, World Health Organization, ScienceDirect, AABB. Coronavirus estimate sources: Imperial College London, Chinese University of Hong Kong, York University. 2020.

[vi] How long can viruses like COVID-19 last on hard surfaces?

Hard surfaces and objects which can carry disease are called "fomites". Every disease and surface type has its own survival duration. Here's the latest research on coronaviruses and their persistence on hard surfaces.

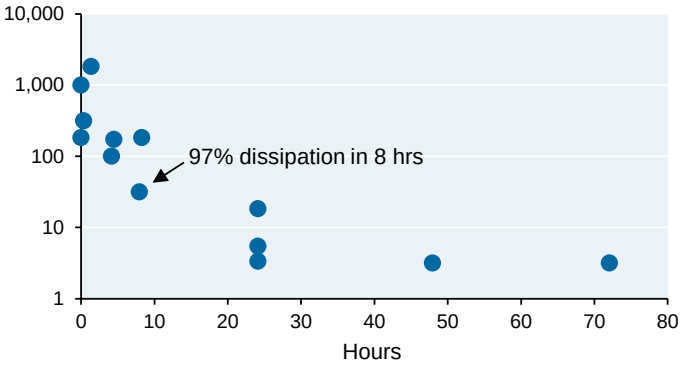
- A March 2020 report in the Journal of Hospital Infection cited 4-5 day survival persistence for both SARS and the Common Cold (sometimes referred to as HCov, or "human coronavirus") at room temperature on most surfaces, such as glass, plastic, PVC, rubber, steel, ceramic and Teflon
- However, while viruses may survive in trace amounts for several days, **they usually lose 90% of their virus infectivity after several hours**. A paper in the New England Journal of Medicine[9] tested both the SARS-CoV-2 and original SARS-CoV virus on aerosols and on various surfaces. As shown below, SARS-CoV-2 viral load intensity[10] declined by 90% with 8-24 hours, depending on the surface
- Surfaces contaminated with viruses like SARS and HCov can be disinfected in just one minute using cleaning fluids with standard concentrations of sodium hypochlorite (bleach) or ethyl alcohol (ethanol)

Decay of SARS-CoV-2 on Copper
Viral load measure, $TCID_{50}$ per ml of medium



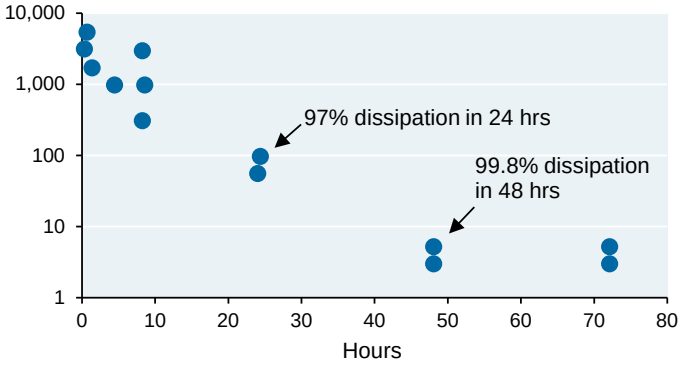
Source: van Doremalen et al, National Institutes of Health. 2020.

Decay of SARS-CoV-2 on Cardboard
Viral load measure, $TCID_{50}$ per ml of medium



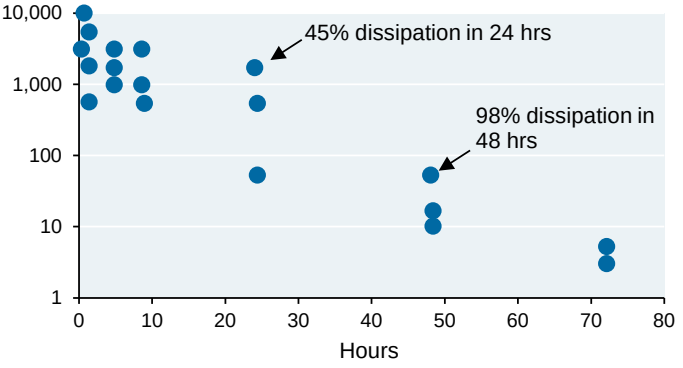
Source: van Doremalen et al, National Institutes of Health. 2020.

Decay of SARS-CoV-2 on Stainless Steel
Viral load measure, $TCID_{50}$ per ml of medium



Source: van Doremalen et al, National Institutes of Health. 2020.

Decay of SARS-CoV-2 on Plastic
Viral load measure, $TCID_{50}$ per ml of medium



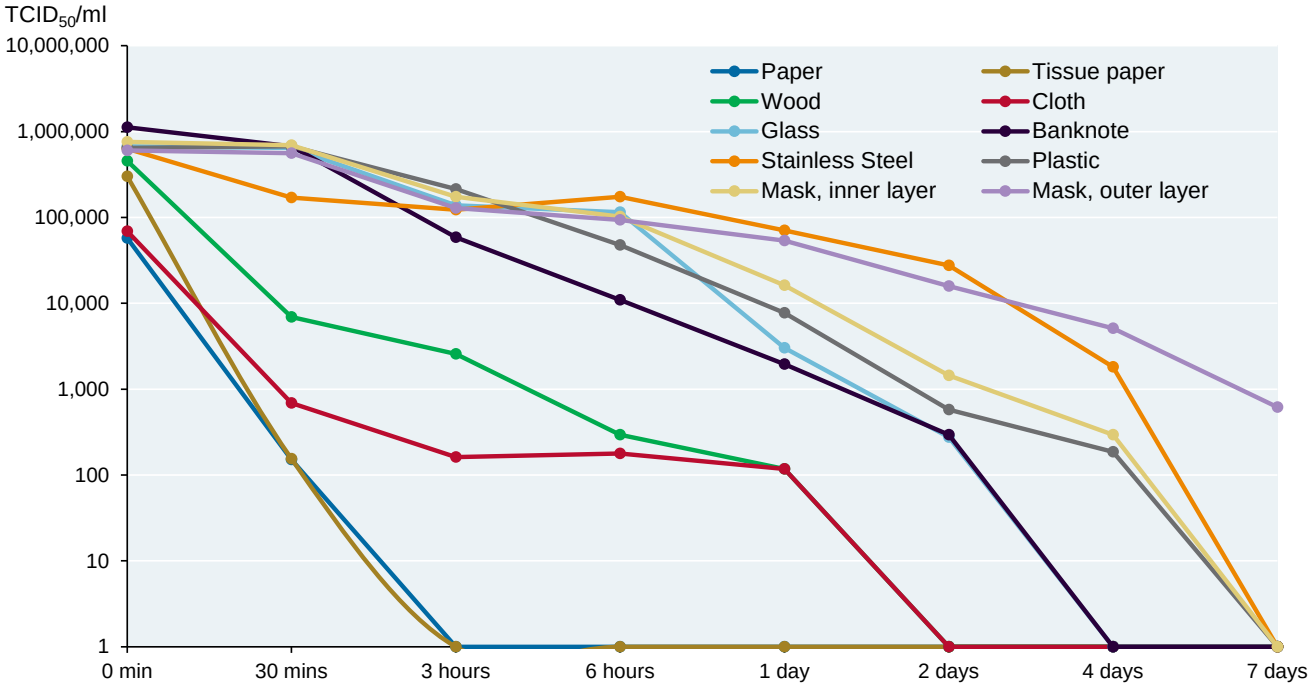
Source: van Doremalen et al, National Institutes of Health. 2020.

[9] "*Aerosol and Surface Stability of SARS-CoV-2 as Compared with SARS-CoV-1*", Doremalen, Morris et al, New England Journal of Medicine, March 17, 2020

[10] Viral load intensity is often shown as TCID 50 ("fifty percent tissue culture infective dose"). It measures the number of viruses per unit of volume that are capable of infecting 50% of cells in tissue culture

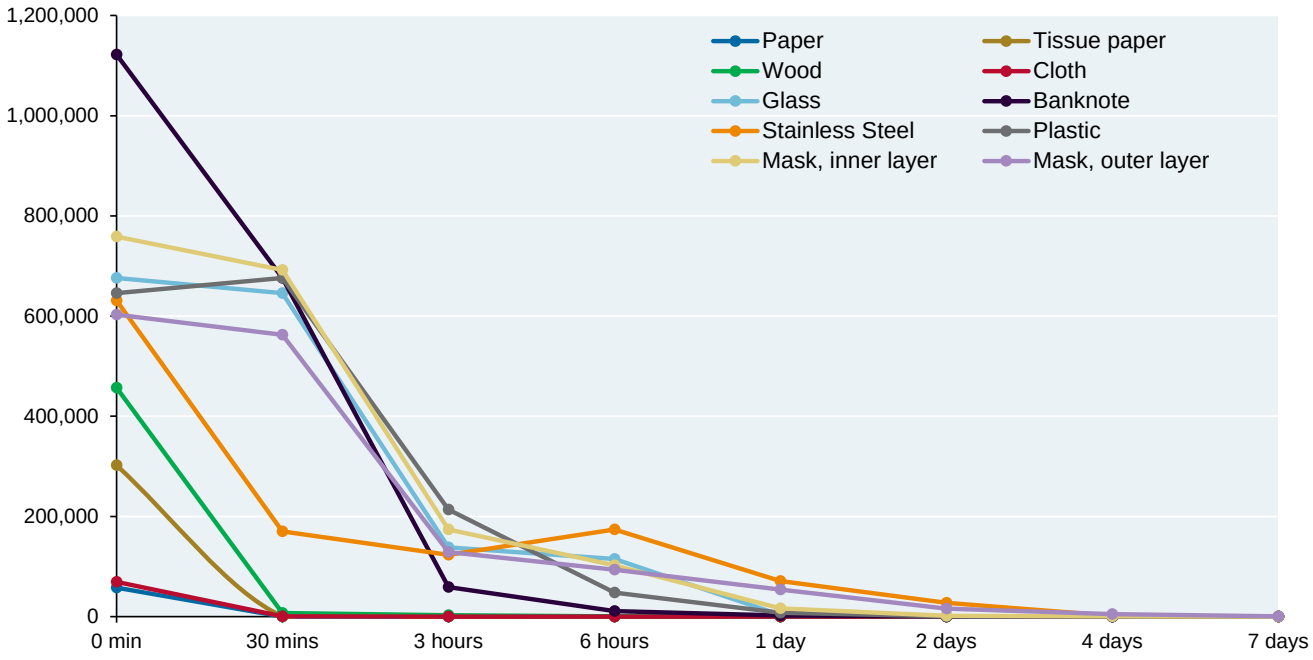
Here's another look at surface transmission, from the University of Hong Kong School of Public Health. The first chart is the standard scientist approach, which plots the data in log scale. However, remember that log scales reduce values by a factor of 10x at every unit change on the Y axis. The same chart using a linear axis appears below it so you can see how rapidly the viral loads decline after just a few hours.

SARS-CoV-2 viral load on surfaces



Source: Alex Chin et al, "*Stability of SARS-CoV-2 in different environmental conditions*", University of Hong Kong School of Public Health. March 27, 2020.

SARS-CoV-2 viral load on surfaces



Source: Alex Chin et al, "*Stability of SARS-CoV-2 in different environmental conditions*", University of Hong Kong School of Public Health. March 27, 2020.

[vii] Could the onset of spring and summer slow virus transmission rates?

There have been press articles and government statements on the possibility that COVID-19 infection rates could fall as the winter comes to an end. There are three main theories as to why the ***flu*** season in temperate regions peaks in winter months:

- More clustering of infected and uninfected people indoors due to colder temperatures
- Colder, drier air is more conducive to airborne travel of viruses; colder air allows viruses to survive for longer periods and to travel longer physical distances
- Lower levels of winter sunlight may play a role given the ability of UV light to sterilize surfaces and kill both viruses and bacteria

Some details:

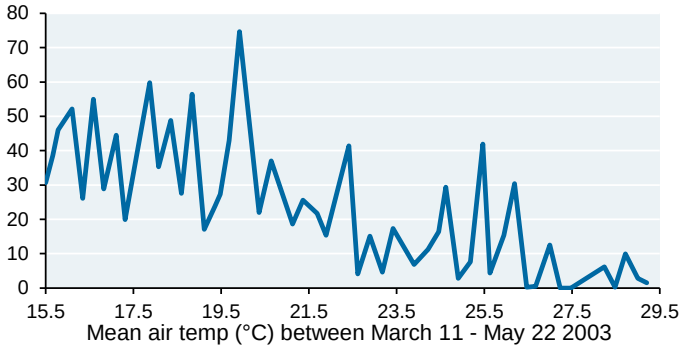
- Scientists have found that influenza peaks in periods of low humidity, low temperatures, low solar radiation and low precipitation. In other words: in cold, dry winter months
- In lab studies using animals, scientists also found that high temperatures and high humidity slowed the spread of influenza sharply, and at very high humidity levels, the virus stopped spreading completely
- During the SARS epidemic in 2003[11], infection rates declined from March to May as temperatures rose. However, there were other factors changing at the same time (changes in hospitalization rates, greater provision of gear to medical personnel, higher quarantine rates and the natural erosion of epidemic severity over time) so results were not conclusive with respect to weather in isolation. Even when combining all these factors, researchers were only able to explain two thirds of the change in SARS infection rates
- **Why might infection rates be impacted by temperature?**
    - Low winter humidity might impair the function of mucus, which traps and expels foreign bodies like viruses or bacteria. Cold, dry air can render mucus drier and less efficient at trapping a virus
    - In addition, influenza "virions" (an infective virus outside a host cell) appear to be much less stable in conditions of higher humidity, when respiratory droplets fall to the ground more quickly
- **It's not just the heat, it could be the sun as well**. Direct and scattered radiation from the sun can break down viruses that have been transmitted to surfaces ("fomites"), but is much less abundant in winter. UV light is so effective at killing bacteria and viruses that it's used in hospitals to sterilize rooms and equipment
    - One study found that in Brazil, there's a correlation between increased influenza hospital admissions and solar UV-blocking by smoke during the burning season
    - The US military reported that UV radiation sterilization virtually prevented the spread of influenza among patients in a veterans hospital, during the same time that an epidemic of influenza ravaged similar patients in nearby non-irradiated rooms
- **However, COVID-19 is not the same as influenza and SARS, and its reaction to changes in temperatures, humidity and sunlight is still unclear**. SARS did not completely subside until late May 2003, which suggests that temperature factors, if they did mitigate the disease, took time to work
- **If weather DOES play a role in COVID-19, then infection rates could FALL in the Northern Hemisphere as temperatures rise, but RISE in parts the Southern Hemisphere in June/July/August when temperatures fall there (i.e., what happens with the flu each year)**

[11] There wasn't too much weather based seasonality of the **MERS** virus, but since MERS is mostly an animal-to-human virus that is not very contagious, scientists don't believe there's a basis for weather based MERS seasonality

The 1st chart shows SARS infection rates as a function of mean air temperatures (in °C) each day from March to May. As temperatures rose, infections declined. The 2nd chart shows how influenza outbreaks (black dots) peak in periods of very low "specific humidity", measured as grams of water vapor per kilogram of air, and in periods of low precipitation. Both conditions correspond to winter months. There's also a cluster of influenza peaks during periods of *high* humidity and *high* precipitation: these mostly occur in tropic zones during summer.

SARS daily incidence and air temperature

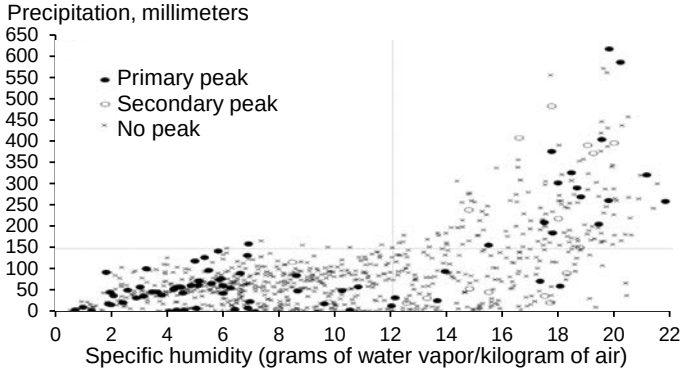
Daily incidence of SARS patients



Source: "*Environmental factors on the SARS epidemic*", Epidemiological Infections, Lin et al. 2006.

Global influenza peaks

Precipitation, millimeters



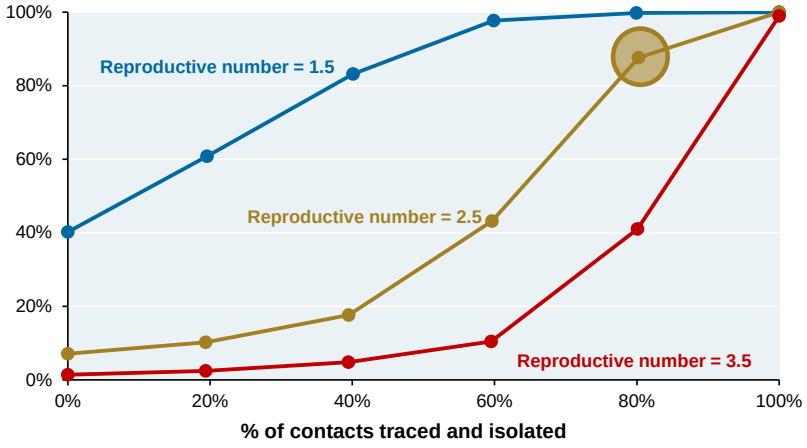
Source: "*Environmental Predictors of Seasonal Influenza Epidemics across Temperate and Tropical Climates*", PLOS Pathogens, Tamerius et al, 2013.

[viii] What does it take to control an outbreak? Aggressive isolation and "contact tracing"

The chart below gets into the question of what it might takes to control a COVID-19 outbreak[12]. Not only does the government have to arrange for isolation of infected persons, but they might also have to engage in aggressive "contact tracing", which involves finding out whom infected persons have come into contact with, and **isolating them as well** within 3-4 days. I am not sure open, Western societies will be able to execute this as aggressively as China has. Let's take an example from the chart: **in order to have a 90% chance of controlling an outbreak, if the reproductive number of COVID-19 were 2.5, 80% of the contacts of all infected individuals would have to be isolated as well (see circled dot).**

Aggressive isolation of infected individuals' traced contacts is required to control COVID-19 outbreaks

% of simulated outbreaks controlled



Source: "*Feasibility of controlling COVID-19 outbreaks by isolation of cases and contacts*", R. Eggo et al., Centre for the Mathematical Modelling of Infectious Diseases, Department of Infectious Disease Epidemiology, London School of Hygiene & Tropical Medicine, February 28, 2020.

Assumptions:

Short delay to isolation once infection is identified (3-4 days); 100% of infected persons isolated once identified; 100% of contacts isolated once identified; 15% of individuals transmit infection before onset of any symptoms

Reproductive numbers reflect observed rates of COVID-19 transmission, which have been estimated from 1.5 to 3.5. See next page for more on reproductive numbers.

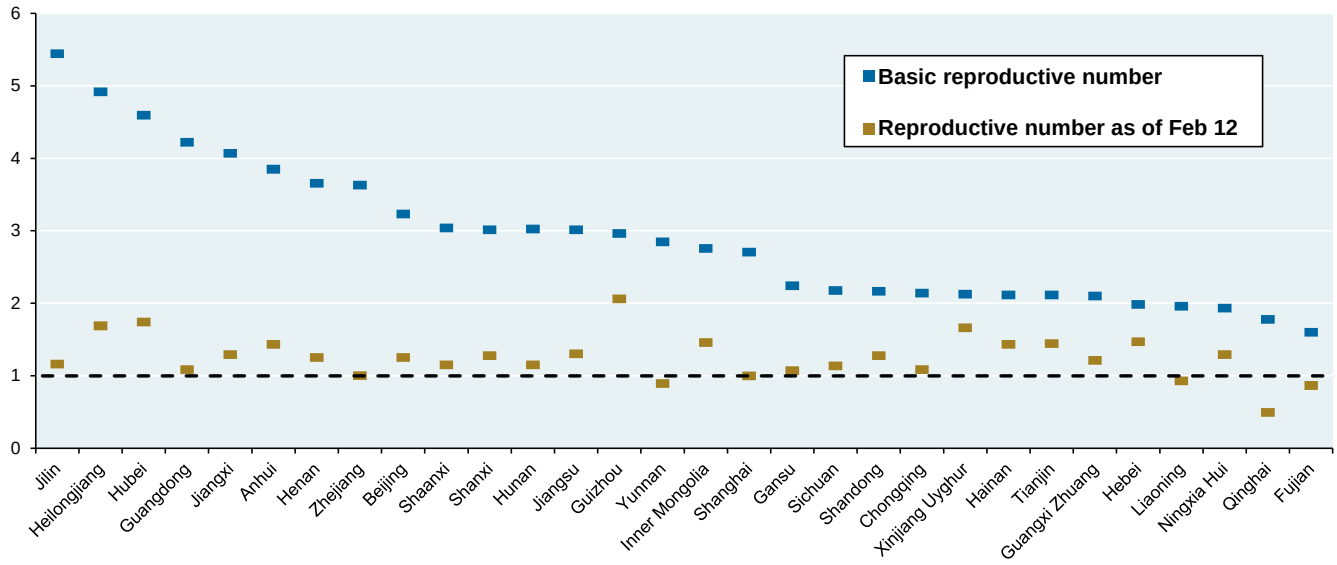
[12] A simulated outbreak is defined as controlled if there are no cases between weeks 12 and 16 after initial cases.

[ix] What are "reproductive numbers" used to describe communicable diseases?

- The basic reproductive number (R0) discussed in articles on COVID-19 is not just a reflection of the inherent transmission risk of the disease, but also of the behaviors and healthcare systems in which it spreads. It is *empirically* measured at a specific place and time, and not just predetermined by the disease itself
- For example, basic reproductive numbers measured for COVID-19 in early January differed markedly across Chinese provinces, and reflect factors such as the density of living conditions, frequency of mass gatherings, commuting patterns and isolation of infected individuals in each location
- By mid-February, a combination of quarantine, contact tracing and other restrictions reduced the observed COVID-19 reproductive number sharply in most provinces. However, for an outbreak to be controlled, the reproductive number needs to be less than 1. Most Chinese provinces were still above these levels in mid-February, but declined further since then

COVID-19 reproductive numbers in Chinese provinces

Reproductive number (the expected number of cases directly generated by one case in a population where all individuals are equally susceptible to infection)

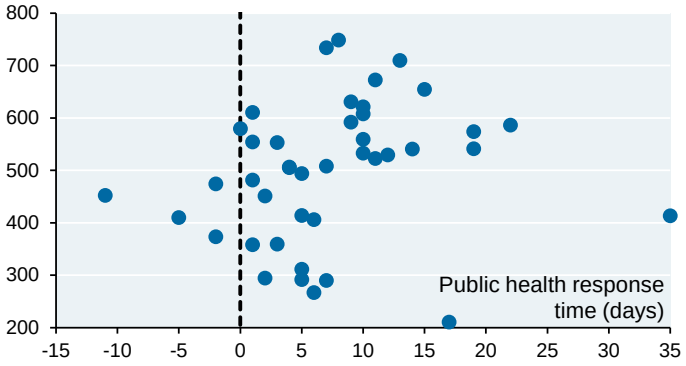


Source: Chu-Chang Ku et al, "*Epidemiological benchmarks of the COVID-19 outbreak*", University of Sheffield, School of Health and Related Research, Health Economics and Decision Science department, February 2020.

[x] Rapid response times, China and information repression

To see the importance of rapid response during a pandemic, let's go back to the Spanish Flu. There were exceptions, but longer public health response times across US cities generally resulted in higher mortality[13]. "Public health response times" represent the time between acceleration of mortality rates and non-pharmaceutical health measures (lockdowns, quarantines, school closures, cancellation of large gatherings, etc). This analysis captured the second and third waves of the Spanish Flu, and the principal time span of non-pharmaceutical interventions.

Excess pneumonia & influenza mortality in US cities, 1918-1919, Excess deaths per 100,000 population

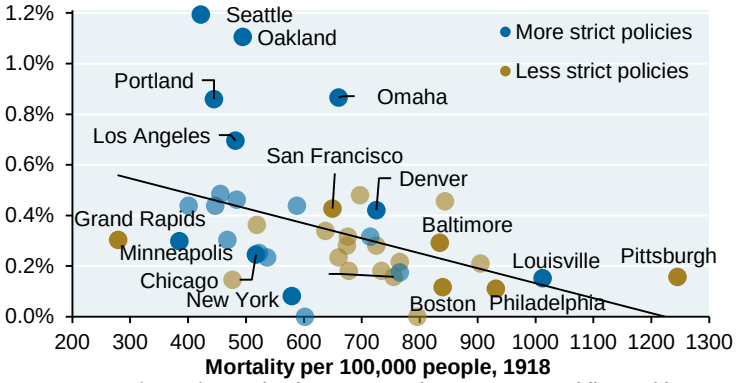


Source: Markel et al, University of Michigan. 2007.

Here's another look at the same general idea, drawn from the Spanish Flu era. The x axis shows the mortality, the y axis shows the increase in manufacturing employment and the colors of the dots illustrate the strictness of social distancing and quarantine policies. Stricter policies led to lower mortality and a faster recovery in manufacturing.

The Spanish flu: social distancing and the economy

Increase in manufacturing employment, 1914-1919



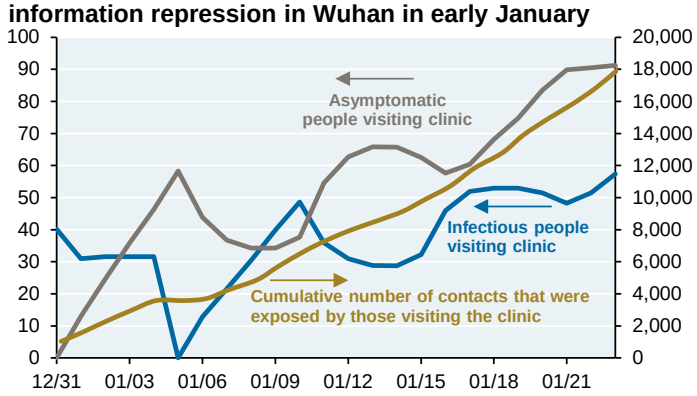
Source: Correia et al. *Pandemics Depress the Economy, Public Health Interventions Do Not: Evidence from the 1918 Flu.* March 2020.

[13] "*Non-pharmaceutical Interventions Implemented by US Cities During the 1918-1919 Influenza Pandemic*", Markel et al (Center for the History of Medicine, University of Michigan Medical School, Ann Arbor) in JAMA, 2007

That's why reporting delays and repression of virus scientists by the Chinese government played such a large role in the outbreak:

- Despite evidence of a severe virus outbreak, on December 30th, Chinese officials sent a notice requiring all hospitals not to transfer fever patients to another medical facility for treatment, and prevented them from publishing diagnosis and treatment information for COVID-19. On January 1st, the Chinese government punished doctors for privately discussing the outbreak. Then, on January 14, the Wuhan Municipal Health Commission reported that there were no new cases of pneumonia infected by coronavirus in the city, and that while the possibility of limited human-to-human transmission cannot be ruled out, the risk of continuous human-to-human transmission is low[14]
- As a result of gov't actions, during the first three weeks of January, Wuhan hospitals were not treating infected and asymptomatic outpatients as potentially infectious
- Using contact model estimates, researchers determined that the 40 infectious patients visiting clinics on Dec 31 then infected 264 other people by January 23 (when Wuhan City was shut down), who themselves came into contact with over 18,000 other people during the same time frame

The result of non-isolation of infectious outpatients and information repression in Wuhan in early January



Source: Zhang et al, Huazhong University of Science and Technology. 2020.

Related reading

"*How China's incompetence endangered the world*", Foreign Policy Magazine, Feb 15, 2020

"*Wuhan virus cover-up exposes a China built on lies*", Foreign Policy Magazine, Feb 3, 2020

"*The* New *Coronavirus and the Blindness of Authoritarianism*", Atlantic Monthly, Feb 22, 2020

[14] "*Impact of Wuhan's Epidemic Prevention Policy on the Outbreak of COVID-19 in China*", Zhang et al (Huazhong University of Science and Technology in Wuhan), Feb 2020

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