

Has SARS-CoV-2 fooled the whole world?

Summary

The World Health Organization has spread fear of COVID-19 without knowing the actual circulation rate of the virus. I calculated the SARS-CoV-2 infection fatality rate (IFR) from antibody prevalence blood samples taken from donors in the Capital Region of Denmark in early April. According to my calculations, the IFR is 0.13%, making the virus approximately as dangerous as seasonal flu. This IFR figure helps explain the limited global public health impact of COVID-19. Population-based serology results, expected soon from Finland, Germany and the US, will either refute or confirm my result. I believe that WHO Assistant Director General Bruce Aylward made a major mistake in February, when he claimed, after coming back from Wuhan, that his team “did not see evidence that a large number of mild cases of the novel disease called Covid-19 are evading detection”. He also claimed that SARS-CoV-2 would be approximately as lethal as Spanish flu. I present in this paper irrefutable evidence of the extremely rapid, but undetected, spread of the SARS-CoV-2 virus. So fast has the spread been that it is likely that New York has already reached herd immunity, and that it is this, rather than the lockdown, which explains the recent abrupt end of the outbreak. The world’s economy must be reopened as soon as possible. The cure now appears to be unequivocally worse than the disease.

About the author

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Introduction

The World Health Organization (WHO) has been spreading undue fear of the newly emerged SARS-CoV-2 since the earliest days of the pandemic (1). This alarmist narrative gained momentum on 25 February when Bruce Aylward, the Assistant Director General of the WHO and the leader of an international mission to Wuhan to learn about the virus and China's response, said his team saw no evidence that a large number of mild cases of Covid-19 had evaded detection (2).

Aylward's statement represented fearmongering because it implied that many of those who became infected became ill enough to require hospitalisation. However, his suggestion was immediately rebutted by Gary Kobinger, the director of the Infectious Disease Research Center at Laval University in Quebec (2), who said it would be highly unusual for there not to be mild or symptom-free cases. He pointed to the fact that the outbreaks in other countries – including Iran and Italy – were the result of people with no symptoms travelling there from China.

Initially, the WHO based its message of fear on the high case fatality rates (CFRs) that had been observed for SARS-CoV-2 infections in China (1,2). In other words, that the CFRs of clinical cases gave an accurate assessment of how lethal SARS-CoV-2 is. They suggested that 2–4% of people infected could die, while claiming – as previously mentioned, that “no evidence that a large number of mild cases of Covid-19 had evaded detection” (2). For example, on 19 February, WHO said, based on data from China, that the CFR of COVID-19 could be 2.3% (1). This figure was also published in a high impact summary of the COVID-19 outbreak in China (3). The fearmongering was reinforced by the situation in Italy, where for a long time the daily CFR was hovering at around 10% in the daily WHO situation reports (1).

Almost four months have now elapsed since the beginning of the epidemic, and there is now an almost global lockdown, which is causing enormous economic problems. Despite this, the WHO has not been pushing its member states towards establishing the true IFR value for SARS-CoV-2 from serological surveys. The need for these surveys is now urgent (2,4). The IFR – the infection fatality rate – tells us what proportion of those infected, with or without symptoms, eventually die of COVID-19.

Materials, methods and results

Here I estimate the IFR from published blood-donor data collected in the Capital Region of Denmark in the first three days of April. Laboratory tests on these samples revealed that 27/1000 blood donors had antibodies against SARS-CoV-2. Extrapolated over the 1,848,989 population of the area, this figure implies that there were 49,923 subclinical infections (i.e. asymptomatic ones) in the Capital Region. However, by 27 March, Danish physicians had diagnosed only 1877 COVID-19 cases. This means that there were 27 times more subclinical infections than there were diagnosed COVID-19 patients in the Capital Region of Denmark

Assuming a 7-day lag from infection to antibody production and a 14-day lag from clinical infection to death, I calculate the IFR of SARS-CoV-2 to be 0.13%. The calculations are set out in Annex I. The maths is simple and should be readily understood by lay people.

Potential biases

The renowned medical mathematician Roy Anderson and his colleagues (probably wisely) gave a cautious lower boundary for SARS-CoV-2 IFR of 0.3% (5), but my figure is less than half of that. Even then, my figure may be overstated. There are two potential sources of selection bias:

- People with even slight COVID-19 symptoms would be less likely to present themselves as blood donors
- People who donate blood are healthy adults, and probably comply with the hygiene measures that are recommended to avoid COVID-19.

In other words, the blood donors were less likely to have COVID-19 infections than the population as a whole. This would tend to exaggerate my IFR figure. Thus, the real IFR of SARS-CoV-2 could conceivably be below 0.1%; in other words, even lower than that of seasonal flu (6).

There may also be a measurement bias. Antibody testing might not be sufficiently sensitive to detect an antibody response in the mildly infected. If so, it would again tend to overstate my IFR figure (7). The Danes have indicated that the sensitivity of the Wantai Elisa test they used is only 77%. Thus there might have been

65 000 people infected by 27 March. Using this value, instead of the 49,923 used above, the IFR result becomes just 0.1%, i.e. the same as that of seasonal flu. And this may still be an overestimate because of the selection biases noted above.

One other bias may tend to lead to understatement of the IFR: deaths among those infected prior to 28 March. This is what was seen on the *Diamond Princess* cruise ship, where five deaths occurred 14 days after the beginning of symptoms (1). However, a similar time-dependent error is likely to be seen in the denominator, as it is possible that mild cases will develop antibodies more slowly than clinical cases (7).

It has been suggested that cross-reacting antibodies of other coronaviruses could have resulted in false positive results among the Danish blood donors, leading to spuriously high prevalence figure in the sample. I find this possibility hard to believe: SARS-CoV-2 established a firm hyperendemic circulation in the Capital Area of Denmark, triggering the first lockdown of the whole society after Italy. Moreover, the Wantai ELISA test has very high accuracy (19), so false-positives are unlikely.

Comparison to other studies

Danish epidemiologists working in parallel with me on this blood-donor material have reached identical conclusions (9). However, a number of other studies have reached broadly similar conclusions.

Silverman and Washburne

Silverman and Washburne, who used publicly available influenza-like illness (ILI) outpatient surveillance data, concluded that IFR is around 0.1%. They say:

While an ILI surge tightly correlated with COVID case counts across the US strongly suggests that SARS-CoV-2 has potentially infected millions in the US, laboratory confirmation of our hypotheses are needed to test our findings and guide public health decisions. (8)

Finnish serosurvey

The Health and Welfare Institute of Finland, along with Helsinki University Hospital, performed a SARS-CoV-2 serosurvey in weeks 13–15 (10). Altogether, blood samples from 444 non-Covid individuals, aged 15–90 years, were tested for SARS-CoV-2 antibodies. The prevalence rates were as shown in Table 1.

Table 1 Finnish results

Week number	Prevalence	SARS-CoV-2 antibody positives / tested non-COVID-19 patients
13	0.7%	1/14
14	0%	0/150
15	3.4%	4/147

False positives were ruled out by retesting in the Health and Welfare Institute.

I calculated the IFR again, using the same logic as for the Capital Region of Denmark, and came up with a figure of 0.08% (see Annex I).

[New York obstetric data](#)

The New York–Presbyterian Allen Hospital and Columbia University Irving Medical Center implemented universal COVID-19 testing of pregnant women who were admitted to the hospital for delivery between 22 March and 4 April. They were tested for SARS-CoV-2 via nasopharyngeal swabs and quantitative PCR (11).

The SARS-CoV-2 antibody prevalence rate was 13.7% (26/215). Almost all of the women were asymptomatic. This implies that a very large number of people were infected among those who had access to these, the top hospitals in the city, with catchment areas situated in Manhattan, where COVID-19 incidence is much lower than other parts of NYC (13). Moreover, the true immunity rate could be significantly higher among these pregnant women; it has been shown that SARS-CoV-2 PCR-positivity quickly diminishes (12). If pregnant women were SARS-CoV-2 positive in Manhattan this often among wealthy New Yorkers then the number of infected people and people with immunity against SARS-CoV-2 would have been astonishingly common in all NYC at the end of March.

[Swedish obstetric data](#)

We have similar obstetric information, and for the same period, from Karolinska Hospital in Sweden (15). There, all women (N = 320) admitted to the hospital for delivery were tested for SARS-CoV-2. It turned out that 7% were infected with SARS-CoV-2 without symptoms. This again is compatible with an idea that SARS-CoV-2 had spread much much more widely in the population in Stockholm than would be suggested by the COVID-19 figures.

Discussion

My IFR figure will either be confirmed or refuted in the upcoming weeks when population serology – based on random samples – is obtained. Studies are under way in Germany and Finland (16,17), and probably elsewhere.

For lay people and journalists the reported death toll of COVID-19 appears to be very high, particularly when you couple the numbers with the horrific TV images from Wuhan, Northern Italy, Madrid, Barcelona, Paris, New York City and now also – to a certain extent - in some of the suburbs of Stockholm. The likely explanation for these tragic events is the high contact rates in multi-generational households, notably in immigrant communities (18).

However, in reality the global public health impact of the virus is limited. On average, over 150 000 people die of causes other than COVID-19 every day. The virus has increased this figure slightly, but only to a level slightly twice the death toll of the last flu season in the US (6). As a more specific example, Germany today reported a cumulative total of 3804 COVID-19 deaths. This figure is still a long way from the annual death toll (25 000) of its last flu season (20).

Since early on in the epidemic, I have wondered why the death toll has been so limited, even in circumstances where suppression and control measures are difficult to organize, for example in Iran (1). For almost a month, the daily death toll in that country has been hovering between 100 and 150 and now seems to be falling below 100. The number of active cases is rapidly diminishing. So it is likely that virus has been circulating there for two months, but with a relatively limited public health impact.

It seems that after an initial rapid rise – and the consequent surge of panic – death rates do not increase exponentially for long and are ultimately limited as social distancing measures and improvements in hygienic practices are put in place. It is known for sure that SARS-CoV-2 virus can be lethal, especially among the elderly (3). It is undoubtedly a nasty enemy if, due to its extremely rapid initial spread, it overwhelms health services or decimates communities with high contact rates between young and old. It is of particular concern in large cities, where hospitals can be quickly overwhelmed (21).

However, as already noted, the public health impacts of these tragic events remain limited, and a plausible explanation could ultimately be the low IFR of the SARS-CoV-2 virus. If this low value is correct – and as I have outlined in this paper, there is now considerable support for my view – then there are important implications. In particular, it is likely that some parts of the world have already achieved herd immunity. The abrupt decline of the death toll in New York City (14) has been ascribed to the lockdown measures taken but, as I set out in Annex II, it appears more likely that there are simply not enough uninfected people for the virus to attack. An IFR value of 0.1%, which is likely to be a conservative figure, suggests that everyone has been infected already. This implies that SARS-CoV-2 spread rapidly before almost anyone was aware of it. It is therefore likely that the virus has airborne transmission ability.

I urge the world to transition quickly away from lockdowns in an orderly fashion and establish sensible approaches to controlling the disease without causing any more economic misery (22). It may be difficult to do so, but the cure is currently much worse than the disease.

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Annex I

The IFR is calculated as:

$$\text{Number of fatalities} / \text{Number of infections}$$

(a) Denominator

The prevalence of COVID-19 antibodies in the blood donors in the Capital Region of Denmark between 1 and 3 April was 27/1000 (1). It takes around 7 days to develop antibodies against SARS-CoV-2, so these individuals were infected before 27 March.

The population of Metropolitan Copenhagen is 1,848,989.

There were thus $(27/1000) \times 1,848,989 = 49,923$ SARS-CoV-2 infections in the area before 27 March.

(b) Numerator

It takes around 14 days from symptoms to death, so the fatalities resulting from the infections would materialise by 10 April.

According to the Johns Hopkins Worldometer, up to 11 April there had been 227 COVID-19 deaths in Denmark. 31% i.e. 67 of them occurred in the Capital Region (2).

(c) Result

We can calculate that by 11 April the IFR should be $(67/49923) \times 100 = 0.13\%$. In other words, for every 10 000 SARS-CoV-2 infections there should be 13 fatalities.

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Annex II Possible herd immunity in New York City

Assume that by the time epidemic ends, there are 11000 deaths. This is approximately equal to the sum of the confirmed + probable deaths figure as at time of writing. Table 2 estimates the number and proportion of the city's 8.3 million population that have been infected already, for various possible values of IFR.

Table 2: Estimates of number of infections in New York City, based on confirmed and probable deaths

IFR (%)	Number infected millions	Proportion infected
0.01	11	100
0.02	5.5	66
0.03	3.7	37

Table 3 reworks these calculations based on only the confirmed deaths figure (6840).

Table 3: Estimates of number of infections in New York City, based on confirmed deaths only

IFR (%)	Number infected millions	Proportion infected
0.01	6.8	82
0.02	3.4	41
0.03	1.3	28

If the numbers of infected are over 60% then New York City will have probably achieved herd immunity.