

What is the evidence in favor of herd immunity for the COVID-19 pandemics?

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Herd immunity was first used in 1923 to explain the transmission of bacterial diseases among mice when a group of artificially immunized animals was introduced into a susceptible population¹. In the original conception of the term herd immunity, the vaccine component already existed as a fundamental ingredient of the effect. During the COVID-19 pandemic, divergences about the herd immunity threshold arose. In brief, a more conservative analysis suggests that around 60% of the population needs to be immunized by infection before herd immunity can be achieved. While analyzes considering heterogeneity in population estimates the herd immunity threshold as 40%².

Some researchers have suggested that Manaus, a Brazilian city with 1.7 million inhabitants, achieved herd immunity because of a report that screened 6,316 individuals in Manaus and 7,551 in São Paulo, a Brazilian city over 12 million inhabitants³. Since the study obtained samples from blood banks, there are some concerns about this sample's representativeness. A similar argument was used by researchers in Bergamo province (Italy), where seroprevalence reached 38.5%, and even with nearly half of the prevalence reported in Manaus

(Brazil), the researchers concluded that Bergamo was in heading toward natural herd immunity⁴. Divergences in the threshold encourage more counterintuitive approaches suggesting a herd immunity between 10% to 20%⁵. This extreme estimate suggests that the SARS-CoV-2 pandemic could achieve herd immunity by natural infection more effectively than by vaccination. Naturally, this idea does not take into account the natural history of any other pandemic.

What is the difference between a person who acquired immunity via infection and someone who acquired it through vaccination? Infected individuals get sick, need hospital beds, and transmit the disease for a long time. These characteristics explain why infected people are not practical barriers to infection. They are not able to stop the transmission of disease for a long time spreading it even asymptotically⁶. On the other hand, vaccinated individuals do not get sick, do not need hospital beds, and do not transmit the virus. It is the concept behind an effective barrier and why we have never seen any natural control of diseases in the history of pandemics. Supporting this idea, the project TYCHO⁷ hosts databases for health and makes available up to 80 years of disease, following for measles, hepatitis A, mumps, polio, whooping cough, and rubella, for instance.

From these data, we can observe that the epidemiologic history of measles in the USA reveals more than 30 years of the high prevalent disease until the vaccine introduction by 1963. After the measles vaccine introduction, the prevalence and incidence of the disease damped abruptly. That is true for almost all diseases for which we have vaccines.

There are, at least, five well-described features of respiratory virus pandemics that, although used to describe influenza pandemics, we can apply to COVID-19⁸: 1. A shift in the virus subtype, which has important implications for an influenza A pandemic, in the context of COVID-19 has limited importance^{9,10}. 2. A shift of the highest death rates to younger populations, which happened in influenza A pandemic but is not clear yet whether will be the case in the next years for COVID-19. Indeed, we are experiencing a great impact on the elderly and a mild to moderate impact on younger people¹¹. 3. Successive pandemic waves, which can be watched from different time-frames. In the context of influenza A pandemics, we frequently look for intervals from several months to years between two waves of great mortality⁸, while in the SARS-CoV-2 pandemic, we are looking to several weeks to few months. This difference in perspective of time only will receive the due importance in the next few years. 4. Higher transmissibility than that of seasonal disease, a feature that we can credit to the D614G mutation¹⁰ and the short-lasting immunity conferred by infection with seasonal coronaviruses¹². And finally, 5. Differences in impact in different geographic regions, which are observed by unevenly distributed case numbers and mortality. While the USA has a high number of cases and mortality ratio, India surpasses Brazil in the overall number of cases but not in mortality¹³.

In order to reasoning about the divergences presented in the literature, at least two important things need to be considered: first, the natural behavior of any pandemic is that over time the rate of transmission tends to slow down. It is just an expected natural fluctuation of epidemics; the cases slow down and increase again after a period of time, which translates into waves of infection. This phenomenon has nothing to do with herd immunity but is due to the non-pharmaceutical interventions (NPI) adopted by governments. As the number of fatal cases increases, the population tends to adhere to behaviors like

wearing masks and social distancing, forcing the infection rate to decrease. Indeed, we have valuable data from other respiratory infections to help us to understand what is happening with Covid-19. A study measured the incidence of metapneumovirus, rhinovirus, parainfluenzavirus, respiratory syncytial virus (RSV), and others in the Korean population and found that the NPI implemented during the Covid-19 pandemic dramatically reduced the overall incidence of respiratory infections¹⁴. The same observation was made in the United States of America, where the incidence of RSV and seasonal influenza were decreased by NPI implemented during the Covid-19 pandemic period¹⁵. There is no place for herd immunity argument in this discussion, though we are seeing essentially the same phenomenon of declination in the number of cases or fatality during a period. Why should we argue for herd immunity, in the absence of vaccination, in the case of the Covid-19 pandemic? There is anything special in SARS-CoV-2 that differs from all other respiratory disease mechanisms of infection, which authorize us to think that the virus will naturally reach herd immunity? The answer is no. We have the same epidemiologic behavior dependent on NPI for all respiratory infections. The second aspect to consider is that, during the pandemic, only a few cases (approximately 8%) are responsible for over 60% of all infections¹⁶, and asymptomatic individuals account for up to 45% of infections¹⁷. The dynamic of the SARS-CoV-2 pandemic seems to rely on superspreaders.

Finally, to reach herd immunity without vaccination is an impractical idea¹⁸. Consider the existence of reports of SARS-CoV-2 reinfection. If this phenomenon happens to be frequent, there will be no possible herd immunity¹⁹. Beyond the misleading concepts discussed here, there are ethical constraints to take into account²⁰. The discussion of whether the population will naturally get immunized by infection only should be on the screen if we are thinking about diseases whose morbidity and mortality are neglectable.

Competing interests

No financial, legal, or political competing interests with third parties (government, commercial, private foundation, etc.) were disclosed for any aspect of the submitted work (including but not limited to grants, data monitoring board, study design, manuscript preparation, statistical analysis, etc.).

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