# I heard it through the grapevine

## On herd immunity and why it is important

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*In this article, David Napier reflects on the limitations of the 'herd immunity' concept that the British government initially supported at the beginning of the Covid-19 outbreak, only for it to be abandoned 24 hours later. Ed.*

The UK government made public on 15 March its proposed strategy to address the Covid-19 outbreak (the SARS-CoV-2 virus) by building 'herd immunity', leaving the general public mystified by a concept it was little aware of. However, the British Society for Immunology was unimpressed, stating emphatically its objections to the government's decision. In an open letter from its president, Professor Arne Akbar, the society flatly rejected the plan:

> The world faces a huge challenge in light of the SARS-CoV-2 outbreak. The UK's public health strategy differs from many other countries, with an aim to build herd immunity to protect the population. Within the immunology community, we have significant questions about this strategy. The ultimate aim of herd immunity is to stop disease spread and protect the most vulnerable in society. However, this strategy only works to reduce serious disease if, when building that immunity, vulnerable individuals are protected from becoming ill, for example through social distancing. If not, the consequences could be severe.[1]

The SARS-CoV-2 virus is surely novel, but epidemic challenges to the herd are not new, even if modern societies have altered those challenges. Some 25 years ago, I was invited to attend a series of three symposia sponsored by the Fondation Marcel Mérieux, part of the French holding company which began in 1897 as the Institut Biologique Mérieux. In that year, Mérieux, a student of Louis Pasteur, had founded the laboratory that would produce the world's first anti-tetanus vaccine, beginning its long tradition of vaccine development for infectious diseases and diagnostic technologies for disease outbreaks.

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**Fig. 1.** *A bust of Charles Mérieux (1907-2001), who devised an efficient industrial technique for mass-producing vaccines. Instituto Butantan, São Paulo, Brazil.*

### Vaccinology and immunology

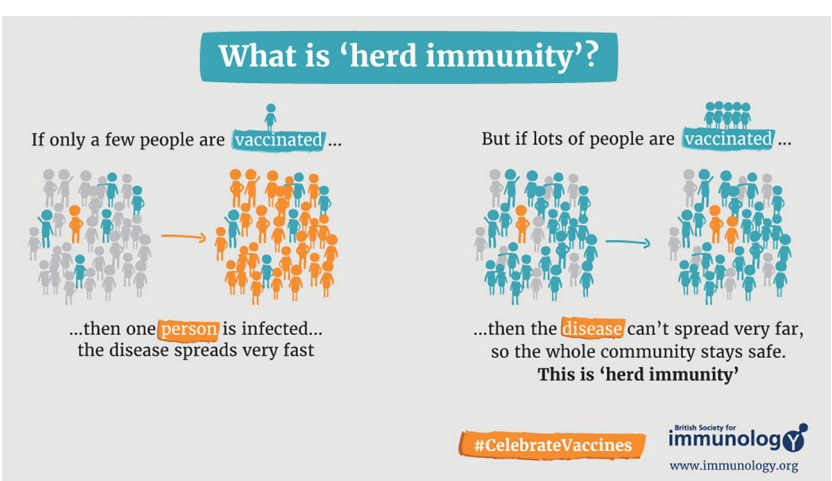
The meetings brought together a small but diverse group of thinkers working across vaccinology and immunology at both applied and theoretical levels. Nearly every participant remarked that they had never attended such interdisciplinary events. Some, surprised by this heterogeneity, stated that these get-togethers could never have occurred outside of France, where infectious disease outbreaks had long been conceptualized as not only biological, but also sociocultural – a view that aligned with my own thinking on immunology and sat well with me as an anthropologist.

My presence at the meeting was, nonetheless, an anomaly; but the organizers were broad thinkers – historians of immunology, Anne Marie Moulin, Bernardino Fantini and Stanley Plotkin, then medical and scientific director of Pasteur Merieux Connaught – and I had just published a book (Foreign bodies) that piqued their interest (Napier 1992). It was written during the 1980s, when anthropologists had yet to be involved much in the study of infectious diseases, and the book was about the assimilation of the foreign (indeed, the ideological foundation of vaccinology). In it, I pointed out that immunity had its conceptual root in the Latin, *immunis*, a legal freedom from social responsibility; and at its end I made the argument that the immune system functioned as much to assimilate difference, as it did to defend us from the 'other' – an argument I would make in more detail in *The age of immunology* (Napier 2003) and in a number of publications since then (Napier 2012, 2013, 2017).

This idea seems less novel today, and has been used both in rethinking autoimmunity (Anderson & Mackay 2014) and in cancer therapeutics (Kallikourdis 2018); but in the era before regenerative medicine and stem cell research (that is, at a time when embryology had been more or less decimated by microbiology), the argument seemed out of pace. It was only around this time, after all, that the power and potential of viral vectors would emerge to reshape our views of what viruses are and how we might employ them productively as information carriers (see for example Jooss & Chirmule 2003).

At each talk, speakers were held to a few minutes. Stanley Plotkin, the lead organizer of the events, placed a wind-up timer on the podium. After five minutes, it rang, and even Merck's public relations sponsor was pulled from the podium when his time was up. After my short talk, in which I made the aforementioned simple argument, there was an uncomfortable silence. Hilary Koprowski, the legendary inventor of the first live polio vaccine, was the first to speak. He asked if what I was saying was that eradicating viral threats was impossible. Before I could answer, he simply said: 'If so, I agree'. The late Melvin Cohn, head of the Salk Institute's theoretical immunology group, had a different response which he emphasized in later letters about my argument: 'If I accept what you are saying about assimilation, I would have to give up immunology'. At the time, that is, responding to infectious disease was a matter of 'recognizing and eliminating "nonself"' – of immunology's conceptual foundation. Indeed, the term, 'vaccinology' was not then, or even now, on any citizen's radar. Public

**Fig. 2.** *'What is "herd immunity"?', a graphic from the British Society for Immunology.*




BRITISH SOCIETY FOR IMMUNOLOGY

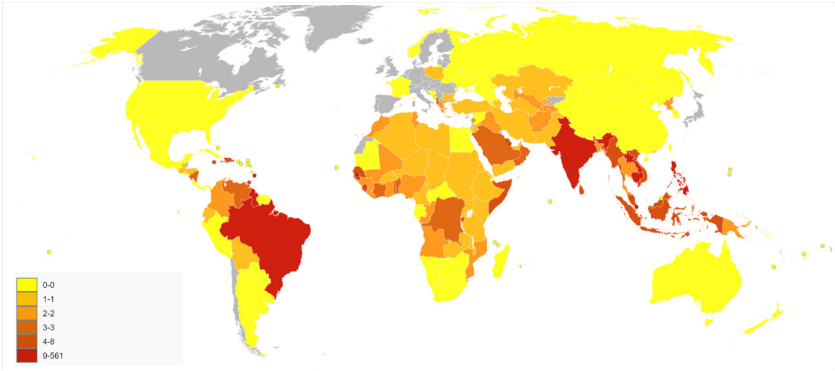



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**Fig. 3.** *Deaths from dengue per million persons in 2012. Statistics from WHO Estimated Deaths 2012 CD-ROM), grouped by deciles.*

discourse was all about invasive threats, a thing science itself had historically fuelled by conflating viruses and bacteria as equally infectious.

Because the events were small – always less than 50 people, with fewer speakers presenting – the most productive conversations happened over lunches and dinners. At one such dinner, I happened to sit next to famed Harvard HIV/AIDS researcher, Max Essex. The discussion, of course, was about HIV and immunity. But having contracted dengue fever in India in the mid-1980s, I asked him why, in his view, virologists had failed (as they continue to this day) to develop an effective dengue vaccine.

Essex perked up, saying that dengue was one of the most challenging viral diseases to understand because its mortality and morbidity remain so dependent on another thing, which is little understood, yet critical for all vaccines: herd immunity. That's because of a very strange feature of the illness: an individual can get it twice and in two different ways. The first way results in malaria-like symptoms – high fever and intense joint pain (Victorians called it 'break bone fever') – that abate after several days, only to come back again, hitting an already weakened body much harder. The second double attack happens for a period of some months after infection, a survivor who has developed immunity to one serotype becomes extremely vulnerable to at least two other of its four variants that can circulate within the herd.

What this vulnerability means is that a person with initial immunity, during those months at least, is actually more endangered by exposure to someone carrying another serotype than is someone with no dengue immunity whatsoever. Dengue morbidity, in other words, is all about what is happening in the social herd, which also explains why it continues to proliferate at an alarming rate in spite of decades of research to halt its progress.

However, funders and the researchers they sponsor want magic bullet vaccinations and the potential Nobel Prizes such discoveries ensure. While a sequential immunization for dengue has been theoretically possible for some time, nobody has pursued this strategy because most assume that herds cannot be 'controlled', and the danger of making populations more vulnerable would be too high throughout the vaccination sequence. Hence Essex's point back in 1995, and the ongoing belief today, that a person living in a dengue endemic area cannot be effectively quarantined for the period of months during which a first vaccine would leave him or her vulnerable to the other dengue serotypes.

While we wait, the disease advances alarmingly, as those sought-after magic bullet vaccines continue to fail; and the argument that a herd might discipline itself enough for a stage vaccine programme to work falls on deaf ears. This is a problem I know well, not only from having had dengue fever myself and learning about it first hand; Michael Jacobs (who was recently knighted for his work on Ebola) and I have tried for a few years now to make the case that dengue could be addressed with a two-stage vaccine protocol. The problem is that our argument keeps being rejected for publication because most researchers (that is, the expert reviewers who are committed to the magic bullet approach) don't believe that the human herd is up to the task.

Indeed, dengue continues its advance, even though a two-stage vaccine would work safely in areas *projected* to be dengue endemic, but where the local herd has yet to be exposed. Here, increased vulnerability in those critical months between vaccinations would occur only if a vaccinated person travelled to an area where dengue is already endemic. For all others remaining within the local herd, it would work without quarantine. That is, dengue's progress could be slowed dramatically, if not stopped, in as-of-yet unaffected areas by asking individuals to constrain their movement temporarily. A risk? Yes, but a small one, compared to the potential gain for the herd.

## Sociocultural drivers of the herd

So why, we might ask, does addressing the role of the herd in epidemics continue to be such a problem when we know there are concrete things we could do now to avoid future suffering? There are many reasons that go beyond the belief that our social behaviours are both unpredictable and unreliable; but there are five key reasons that might help us today as we grapple with understanding how to formulate useful responses to Covid-19.

The first has to do with scientific misinformation. For science – not common knowledge – has led us to believe that viruses invade us, which they do not. Antibiotics kill organisms, but viruses are just bits of information that the cells in our own bodies bring to life. Viruses don't invade us; but people do pass on information. The difference between living organisms and viruses may, that is, appear subtle; but it is far more than semantic. For example, there are elements of the human genome that we accept as having a viral origin via a process called reverse transcription, which allows the virus that uses this mechanism (a so-called retrovirus) to replicate itself off of the human DNA in the cell which hosts it – thereby incorporating itself into the very centre of the human genome. Other viruses use different mechanisms, but all are dependent on human cells, human enzymes (proteins which are important to biochemical reactions), and human genetic processes. Indeed, today cell biologists use viral vectors as tools to deliver genetic material. So viruses can play an important role in stabilizing what happens in the future and even in treating diseases that were in the past untreatable. Thus, viruses are not by definition only the enemies of public health.

Indeed, viruses, alone, cannot invade us, because they have no motility. We make viruses infectious by passing on cells to one another that, for better or worse, contain new information. This means that viral epidemics are profoundly herd based. It is what happens in the herd – what we do as humans together – that brings novel viral information to life. It is what the social herd does that moves viral information forward to advance our health or to make us sick. Proliferation is a social matter, in spite of the fact that bench science continues to advance the notion that viruses themselves attack us. It is not in our interest to think this way; but it is in the interest of science.

This distinction is today critical. Because our lack of social understanding – our inability to involve social science seriously in responding to Covid-19 and how it makes specific populations vulnerable – has altered the epidemic landscape profoundly.

Countries, because of this perception, close borders, even when viral information is already present within their herds. Asymptomatic carriers fear foreigners because they believe the disease stems from outside 'invaders' and, most importantly, populations forget information as easily as they learn it – meaning that as

soon as the perceived invasive threat is thought to have gone elsewhere, countries and their populations learn little or nothing about preparing for the next information overload because they think somehow, instead, they have defeated a foreign threat.

But perhaps it is not in the interest of current biomedical science to acknowledge the profound sociocultural drivers of the herd, even though before 1850, Rudolf Virchow, the so-called father of modern pathology, implored us to consider medicine as both a social and biological discipline. Here, the world's disease organizations (principally the World Health Organization, the Centers for Disease Control and Prevention and now the Gates Foundation) have, I would argue, done less than they might have to abate the belief that the recognition and elimination of nonself (i.e. the early principles of immunology) will save humanity. Indeed, viral information already residing in the human herd may protect us from the spread of one kind of viral information, while, as we will soon see, making us simultaneously vulnerable to other kinds.

Second, because information must be carried from person to person, the size and proximity of the human herd becomes critical, not only to the speed by which new viral information spreads, but to what kind of information gets spread quickly. As all epidemiologists and virologists know, the more lethal kinds of viral information (e.g. Ebola) are thankfully quite fragile (at least so far). Otherwise, the herd would be wiped out quickly and there would be no information carriers. We have, and will continue to have, Ebola outbreaks; but thus far, social containment has proven effective, if sometimes controversial, for addressing the most dangerous kinds of viral data.

But it is not only the fragility and resonance of viral information that matters; changes in the human herd itself have reframed our relationship with viruses. In the mid-19th century, 90 per cent of the world's population were farmers or at least grew their own food (compared to about 1 per cent today). By the dawn of the 20th century, still only 13 per cent of the world's population lived in cities. Setting aside profound changes in transportation and food security, more than half of the world's population today live in urban areas. Barring more pandemics, that percentage will rise to two-thirds by 2050 and 85 per cent by 2100 (European Commission n.d.).

What this means for novel kinds of viral information is that urbanization itself has profound implications for human vulnerability. More resilient, if less lethal, viruses will without doubt become commonplace, taking an increasingly higher toll on our physical health and our ability to lead productive lives. That's simply because there are more of us, and more of us herding closer together than ever. If populations continue to concentrate in urban areas, so will less lethal, but more resilient, kinds of viral information increasingly impact the human herd.

Exciting though the idea may be, it is not only because people live in close proximity to animals – thereby allowing viral information to leap from species to species – that we have new and novel pandemics. One Health is important, but not the entire picture. It's because many of us are living closer and closer to one another and in much larger numbers. Novel viruses proliferate, not only because some of us eat bats or have chickens in our kitchens, in spite of what our xenophobic inclinations would have us believe of foreigners and their 'strange' habits. The simple density of the herd is why so many viruses emerge in populated places, and why less lethal forms of viral information should be feared for their ability to debilitate the human herd physically, socially and economically.

Third, if in principle we could all stay put, an immunized herd might mean that viral information would not reach those without immunity – hence the herd itself becomes protective of its vulnerable populations. To put it another way, if the Age of Discovery had never happened, there would be legions of people in the Amazon rainforest who would not have died of the common cold. Here, containment is important for the most vulnerable of us, which is why the British Society for Immunology hastened to reject the proposal to address Covid-19 through herd immunity. But modern transport has made this kind of herd immunity obsolete, except perhaps for those parents who selfishly insist on not vaccinating their children because 'all of the other kids at school have been vaccinated'. Here, the information game is not only viral. It is also political, explaining perhaps why post-Brexit politicians might favour the approach.

However, as comforting as immediate containment may be to our limited and short-term capacity to remember the last outbreak once the current one goes temporarily into hiding, the reasons for containment have less to do with reducing the numbers of people within the herd who are positive (in Covid-19 that could eventually top 70 per cent), than they do with attending to the gross inequality that is inherent to social instability. Ask why citizens of Monrovia, Liberia and elsewhere stormed Ebola containment centres to release relatives from harm's way, and you will understand easily why social trust – the belief that the herd will promote harmony – is more important than forcing people into isolation, as much as some may applaud China's capacity to deny mobility to its citizens. Because when societies appear unstable, not only do people panic and fight over toilet paper in supermarkets, they also think mostly about themselves, even in places where caring about the common good is meant to be formally entrenched.

In one of London's highly regarded university hospitals, for example, survival among staff is also a matter of triage. With limited numbers of masks available, hospital administrators recently handed them out to other administrators and senior doctors, while front-line junior doctors and nurses – those who bear the lion's share of patient contact – were initially left with nothing. Gross self-interest, in other words, is not simply a thing endemic to capital markets, though the ongoing raiding of social welfare for private gain has now shown how cheap the emperor's clothes actually are. Typing 'percentage of growth in hospital administration' into my Google search engine, this was the first thing that appeared on my day of writing this short piece:

> Here's some food for thought: The number of physicians in the United States grew 150 percent between 1975 and 2010, roughly in keeping with population growth, while the number of healthcare administrators increased 3,200 percent for the same time period. (Cantlupe 2017)

Were they all at that unnamed London hospital, no caregiver would have access to a mask, and hospital administrators would be fighting for them amongst themselves. So much for putting money into monitoring care in the interest of saving money, rather than putting money into care.

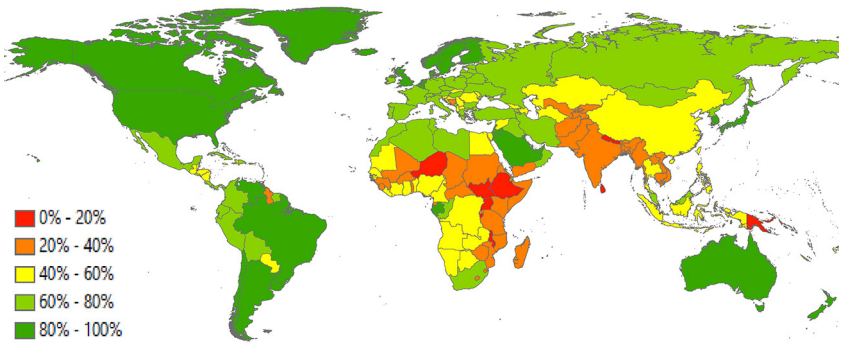


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**Fig. 4.** *The percentage of a country's population that lives in urban areas. Based on data from: United Nations, Department of Economic and Social Affairs, Population Division (2014). World Urbanization Prospects.*

**Fig. 5.** *Playground closed until further notice.*
**Fig. 6.** *Salt Lake County Parks & Recreation Covid-19 prevention sign.*
**Fig. 7.** *Thirteen noroviruses: an electron micrograph. Magnification approximately x200.*

I would like to thank the following people for reviewing an earlier draft of this essay: Rebecca Carter, Edward Fischer, Tamara Giles-Vernick, Andrew Lemert, Robert Muller, Young Su Park, Cynthia Rosenberg, Nancy Scheper-Hughes, Paul Sender, Anna-Maria Volkmann and David Wengrow.

1. https://www.immunology.org/news/bsi-open-letter-government-sars-cov-2-outbreak-response.
2. https://www.sonar-global.eu/.
3. Recombination refers to the exchange of genetic material between different organisms which leads to the production of offspring with combinations of traits that differ from those found in either parent.

Anderson, W. & I.R. Mackay 2014. *Intolerant bodies: A short history of autoimmunity*. Baltimore: Johns Hopkins University Press.

Cantlupe, J. 2017. Expert forum: The rise (and rise) of the healthcare administrator. *Athenahealth*, 7 November. https://www.athenahealth.com/knowledge-hub/practice-management/expert-forum-rise-and-rise-healthcare-administrator.

Debbink, K. et al. 2013. Emergence of new pandemic GII.4 Sydney norovirus strain correlates with escape from herd immunity. *The Journal of Infectious Diseases* 208: 1877-1887.

European Commission n.d. Urbanisation worldwide. https://ec.europa.eu/knowledge4policy/foresight/topic/continuing-urbanisation/worldwide-urban-population-growth_en.

Jooss, K. & N. Chirmule 2003. Immunity to adenovirus and adeno-associated viral vectors: Implications for gene therapy. *Gene Therapy* 10: 955-963.

Kallikourdis, M. 2018. T cell responses to tumor: How dominant assumptions on immune activity led to a neglect of pathological functions, and how evolutionary considerations can help identify testable hypotheses for improving immunotherapy. *Cancer Immunology, Immunotherapy* 67(6): 989-998.

The reason, in other words, that containment is now more important than ever is not only because of simple population growth, it's because neo-liberal advocates (always out for short-term self-interest) have invaded and ruthlessly robbed healthcare systems worldwide, even to the point of not being able to provide accurate data – and that's within and among the herds of the world's supposedly most developed nations. Containing people on boats, or in prisons, or in mental hospitals (places where it is easier to contain) may make governments appear symbolically more in control, but so doing will only slow the movement of viral information outside of those places where people remain trapped, not reverse its proliferation. Containment, that is, helps already vulnerable hospitals adjust, saving lives by slowing admissions, just as it helps policymakers who have raided the common good appear in control by 'containing a threat'.

In short, given that roughly one in seven people on the planet now lives as a migrant in a place where locals see them as an outsider, the scope for inequality and civil unrest will explode magnificently if we continue to rob social welfare as mid-level pandemics become more common. And that's a problem that will only worsen as we continue to favour paying stupid sums to those administrators who prioritize the survival of their institutions and health businesses over the interests of those needing and providing direct care within them.

This point cannot be overstated. In times of crisis, inequalities are exaggerated, not ameliorated. We must, then, prepare in advance for the herd's tendency under stress to expand its definition of 'outsider'; for under such circumstances, it will always seek to assuage any lingering guilt over its rank self-interest by feeling assured that outsiders don't belong.

It is for this reason that assessing vulnerability in real time is far more critical in responding to epidemic disease outbreaks than is trying to enforce either social quarantines or individual behaviour change. Because when new inequalities emerge, the most vulnerable may fully lack both the capability and the opportunity to respond in health-enhancing ways. Indeed, faced with new doublebinds that inequality fuels, the most vulnerable may lose the very motivation to do anything at all except what is necessary for personal survival on a day-to-day basis. That's what we call *calamity coping*; and it's why we social scientists involved in responding to infectious disease outbreaks (like members of the Sonar-Global Network)[2] have put our money on the rapid assessment of vulnerable populations, rather than on the carrot, stick and nudge theories that policymakers favour funding as a means of controlling the herd.

Fourth, and this is the 'question' we've been holding back on: what does the communality of the herd itself suggest about our ability to decrease the impact of potentially debilitating viral information? To get a grasp on this bigger social challenge we need to be aware of new work in viral epidemiology, and not only for coronaviruses.

## Epidemiology of noroviruses

Noroviruses are a good start, because they have a big impact on the human herd without often taking human lives, in turn inclining us to scapegoat and blame foreigners less. Noroviruses cause flu-like symptoms, including vomiting and diarrhoea – they are among the common winter bugs that come and go in a few days. And recent research on norovirus epidemiology has shown some surprising things about the relevance of the human herd to how viral epidemics emerge.

Work in which a group of my University College London colleagues participate has demonstrated not only that the norovirus bugs we are contending with this winter

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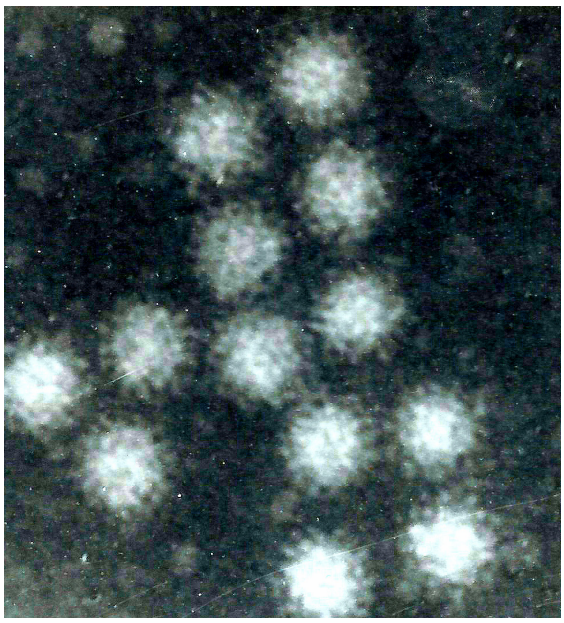
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have been around longer than we once thought, but also that their emergence may be less a function of a particular strain's mutation than a function of changes in the herd itself (Ruis et al. 2019). For not only can immunity to one virus protect us from the future effects of that same or similar viral information, but that immunity may also make us vulnerable to other kinds of viral data waiting to 'inform' the human herd, for better or worse.

As the authors claim, because vaccine development and distribution 'require identification of the sources and drivers of new pandemics', it may be critical to understand that 'pandemic noroviruses preadapt, diversify and spread worldwide years prior to emergence, strongly indicating that genetic changes are necessary but not sufficient to

Lindesmith, L.C. et al. 2012. Immunogenetic mechanisms driving norovirus GII.4 antigenic variation. *PLoS Pathogens* 8(5).
— et al. 2013. Emergence of a norovirus GII.4 strain correlates with changes in evolving blockade epitopes. *Journal of Virology* 87: 2803-2813.
Marmot, M. 2015. *The health gap: The challenge of an unequal world.* London: Bloomsbury Publishing.
Napier, A.D. 1992. *Foreign bodies*. Berkeley: University of California Press.
— 2003. *The age of immunology: Conceiving a future in an alienating world*. Chicago: University of Chicago Press.
— 2012. Nonself help: How immunology might reframe the enlightenment. *Cultural Anthropology* 27(1): 122-137.
— 2013. A new sociobiology: Immunity, alterity, and the social repertoire. *Cambridge Anthropology* 31(2): 20-43.
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Plotkin S.A. & B. Fantini (eds) 1996. *Vaccinia, vaccination, vaccinology: Jenner, Pasteur and their successors.* Paris: Elsevier.
Ruis, C. et al. 2019. Preadaptation of pandemic GII.4 noroviruses in hidden virus reservoirs years before emergence. *bioRxiv* preprint first posted online 3 June 2019.

drive a new pandemic'. Instead, they offer a different argument: 'that changes in population immunity enable pandemic emergence of a preadapted low level variant', requiring not only 'surveillance of under-sampled virus reservoirs' but vaccines that can 'elicit broad immunity' (ibid.: 2).

Reconstructing the history of norovirus genomic regions, they show that such viruses may be present 'for at least 50 years prior to the first documented pandemic' – that 'in depth analysis of sequence data shows that genetic substitutions and recombination[3] events that may be important for pandemic emergence are acquired years before such emergence occurs' (ibid.: 3).

But why are these findings important for the herd? Because the results of their tracking suggest a startling conclusion: 'that recombination events are not the proximate drivers of new norovirus pandemics' (ibid.: 5). Not only is the pandemic event 'not proximally driven by genetic changes in any of the genomic regions altering antigenicity, receptor binding or another property', but that 'what drives a variant that has been circulating widely and cryptically for years to suddenly increase in frequency, dominate outbreak worldwide and rapidly replace the preceding pandemic variant' may be 'a change in host factors' (ibid.: 5) – that is, in the state and condition of the herd itself.

> Given the importance of herd immunity in variant emergence (Debbink et al. 2013; Lindesmith et al. 2012, 2013), we hypothesize that a shift in host population immunity (potentially driven by growing immunity against the previous pandemic variant) opens a population-wide immunological niche into which the multiple circulating but hidden lineages of the new pandemic variant can expand, having acquired the necessary antigenic characteristics to do so years before. Therefore, viral genetic changes are necessary but not sufficient for pandemic emergence. Instead, a shift in host immunity combines with antigenic preadaptation to drive a new pandemic (Ruis et al. 2019: 5).

What this study indicates, furthermore, is that multiple variants 'circulate within the community and are not detected by current surveillance efforts that largely target outbreaks, predominantly in hospital and institutional settings' (ibid. : 6) – that is, in places where members of the herd are mostly quarantined. Instead, the circulation of viral information is ongoing in a potentially pandemic form, but changes in herds themselves make possible the ready assimilation of new information. Hence, the algorithmic explosion of cases in local populations during pandemics.


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**Fig. 8.** *Covid-19: rethinking risk.*

Using the amusement arcade game of whack-a-mole as a metaphor, these researchers argue that we knock one mole back into its hole only to have another pop up – attempting reactively to address what we see before us, assuming superficially we have solved a problem, without understanding at all both the biological and the social role that the herd itself plays in a new pandemic's emergence. For the condition of the host population itself – including its levels of equality or inequality; its social vulnerabilities and resiliences (Marmot 2015) – not only determines the extent to which herd immunity can work (because heightened inequality leads to increased susceptibility), but drives the biological trajectory of an epidemic. Here, our focus on addressing the immediate threat has blinded us to the broader challenge – as if we'd gambled on a single stock, while risking the strength of our portfolio.

Put simply, noroviruses are not going anywhere – we are! And the sooner we accept that the social health of the herd is as important in pandemic responsiveness as is its biological condition, the better prepared we will be in the future to assist one another – and to limit inequalities – when a different mole bearing different information emerges unexpectedly.

But this fourth point demands one more.

Fifth, and finally, if we are collectively to become more resilient in addressing whatever the next pandemic may bring, what changes need to happen now in our understanding of human herds as both biological and social collectivities – of our shared conventional understandings, call them 'cultures'?

## What changes are needed?

From the biological perspective, the changes needed are rather obvious:

First, we must at all costs stop confusing viruses with living, infectious agents. Viruses, like many types of information, are ecumenical: not entirely good, not entirely bad, but also, often changing in their substance, meaning and function. So long as we think of them as foreign invaders, we will continue to downplay the actual symbiotic relations between human herds and the viral data that circulate within those herds, making us ill and/or resilient. We will also fail to look deeper into the broad families of antigenic information that need to be attended to if we wish to limit the unexpected impact of the viral 'black swans' that episodically surprise us.

Second, if we continue to confuse viruses with living things we will not only misuse antibiotics to the point of our own extinction, but fail to limit the impact of xenophobic sentiments that drive political policies and ambitions even against the better interests of the herd. And this second point raises strategic reasons pertinent to why this confusion needs remedying.

If the organizations that put health anywhere in their names continue to ignore the social dimensions of what drives, limits, fuels and abates a pandemic – if they do not indeed take seriously the need for *health in all policies* – we will only continue to be poorly prepared for future pandemics. And if we, likewise, react to Covid-19 by naming all viruses and outsiders as 'other', we will continue to scapegoat our own neighbours, allowing the worst parts of our humanity to seek short-term preservation in place of the very diversity that is essential for our creative growth.

Indeed, changes in the herd are all about how we act together as a population. In any pandemic, these are the deciding factors, as much as biological or medical determinants on their own. For social responses and variations in responses not only determine whether we remain resilient or become vulnerable, but also determine our capacity to discover new options that are real to us that we might otherwise have never imagined. ●