Understanding the Covid-19 pandemic through the lens of population health science

Sandro Galea, Katherine Keyes

Sandro Galea,[1]* Katherine Keyes[2]

[1]Dean's Office, School of Public Health, Boston University, Boston, MA.

[2]Departments of Epidemiology, Mailman School of Public Health, Columbia University, New York, NY.

*Author for correspondence

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Abstract

In a few devastating short months in 2020, the Covid-19 pandemic changed global mobility and interaction in ways that were unimaginable to many of the world's population as recently as in 2019. More than 10 million people have, at this writing, been infected by SAR-CoV-2 globally, and more than 500,000 have died of Covid-19. As our science progresses, it is becoming possible to apply the principles of population health science to help us better understand the pandemic. What does a formal approach to population health science teach us about Covid-19? Building on our previously published work about the foundations of population health, we offer a few observations—a first draft of population health science thinking—as it intersects with the Covid-19 pandemic. Of note, our collective understanding of the pathology and causes of Covid-19 are rapidly changing by the day, and thus we fully expect that this work will evolve and improve as science progresses.

Key words: epidemiology, pandemic, theory, population health

Abbreviations:

CDC: Centers for Disease Control and Prevention
HIV/AIDS: Human Immunodeficiency Virus/Acquired Immune Deficiency Syndrome
WHO: World Health Organization

Introduction

The first half of 2020 saw the emergence of the SAR-CoV-2 virus, a novel coronavirus that in a matter of weeks became a global pandemic with unparalleled consequences. It took only about three months from the time of the first diagnosis on December 31, 2019 in Wuhan, China, for the virus to be diagnosed essentially in countries all over the world. As countries scrambled to keep up with a novel pathogen with unknown characteristics, many implemented large-scale lockdowns or shelter-in-place protocols that had the effect of effectively freezing the global economy, triggering massive unemployment and plunging countries like the US in the worst economic recession in a century. Compounding the challenges of the moment in the US, long-seated social and racial injustices, manifesting in the disproportionate burden of Covid-19 experienced by persons with low-income and persons of color, came to a boil around the continued police murder and brutality toward Black men and women, and resulted in nationwide civil unrest rivaling any in the previous 50 years. These concurrent forces conspired to create a cascade of national traumas, experienced collectively, and threatening the health of the global population.

The recognition of a novel virus that posed, at the time, poorly characterized health threats, resulted in unprecedented global action. The world's attention turned to health, and to public health in particular. Massive national efforts were undertaken to promote public health, and policies were implemented in the name of public health across the world. Epidemiology become a common term on the front page of the world's newspapers, and epidemiologists suddenly found themselves thrust in the national spotlight perhaps more so than at any period since the beginning of the HIV/AIDS epidemic. This mobilization and spotlight on our field is in and of itself remarkable. It is a moment of opportunity—and of challenge—for population health science. Much remains to be learned about the specifics of the Covid-19, including characteristics of the virus itself, and about the policies and behavior changes that mitigate its spread. We expect that the coming years will result in important insights to inform and be informed by population health science that will guide how we prepare for, and mitigate, future pandemics.

We are also at a point, however, when some learnings are beginning to emerge, when we can begin to sharpen our thinking about Covid-19, seen through the lens of population health science. What does a systematic approach to population health science teach us about Covid-19? Building on a frame of our previously published work about the foundations of population health,[1] we offer a few observations as a first draft of population health science thinking as it intersects the traumas of the moment, fully expecting that this work will evolve and improve in coming years.

1. Population health is best understood by thinking beyond dichotomies

Epidemiology lends itself to a focus on categorical outcomes, aiming to understand causes of cases. By contrast, a dimensional approach aims to expand that lens beyond simple case categorization, to thinking of the fuller range of health manifestation. For example, we may think of the full spectrum of blood pressure in populations rather than considering whether blood pressure is high or low.

An infectious disease pandemic lends itself naturally to categorical thinking, as a focus on the acquisition of infection is a clear, and appropriate, priority. While this may not, at first blush, readily lend itself to dimensional thinking, Covid-19 dynamics indicate that we best serve population health by thinking dimensionally across a range of health indicators, expanding our focus beyond clearly defined categorical outcomes, even in the case of an infectious disease pandemic. This recognition frames how we may think of the causes of the pandemic and how that may shape our approaches to it.

Consider the consequences of the novel coronavirus that constitute health indicators of interest. We are first concerned with cases of infection and deaths from the virus. But deaths from the virus may be due to a disease that is uncurable, or one that overwhelms the health system. Hence, we should also be interested in hospitalization rates, and availability of acute care if it were needed to look after those sick with coronavirus. All of these are reasonable indicators of concern to population health. In addition, efforts at mitigating viral spread have now resulted in extraordinary economic shocks, second only to those experienced during the Great Depression. We know that these economic consequences will lead to health consequences.[2,3] This suggests that the economic indicators, and their health consequences should also be of concern to population health, ranging from increases in cardiovascular disease, depression, and suicide, all of which have been associated with adverse economic conditions.

Which of these health indicators matter most? They all matter, and all should be of concern for population health, as long as we understand our concern to be more than simple caseness linked to the infection, but with the full set of conditions that shape population health. This has complicated implications for policy making. If our only outcome of interest were viral caseness, it is certainly true that complete and prolonged efforts at population physical distancing are the approach best supported by population health science. But, if we recognize that the economic and social consequences have health burdens that contribute to the direct morbidity linked to viral infection, discussions of infection control should include consideration of the balance of infection spread and preservation of sustainable and thriving livelihoods. This would then suggest that age risk stratification—that could balance reducing risk among those most vulnerable to viral morbidity and mortality while preserving economic function—should gain prominence as a weight in our consideration.

Therefore, even in the case of a single infection, thinking of a dichotomous outcome (being infected or not) underlies a variety of causes and outcomes that are important both for understanding the pandemic consequences, and guiding what we may do to mitigate these consequences. A reductive concern with single dimensional caseness pushes us away from this understanding, and hides important causal observations to the detriment of approaches that may help mitigate disease. In the context of multiple national traumas, where the viral pandemic is just one piece of a larger set of conditions that threaten the moment, focusing only on single outcomes further limits us.

2. The causes of difference across populations are not the causes of difference in health within populations

Defining features of this pandemic have been both the rapid viral spread across the world, affecting essentially all countries within months, and also the incredible heterogeneity we have seen in viral spread across countries, and in the US, across states. This represents an all too apt illustration of how we may best understand the drivers of population health at different levels of influence. Let us consider two examples.

First, consider differences between two US states. The epidemic curve of states like Massachusetts and Florida, for example, have been substantially different since the epidemic started. The epidemic started in earnest in Massachusetts in March and peaked in late April, arriving by June at a much more quiescent stage. By contrast, Florida's epidemic curve emerged substantially later, becoming more pronounced as the state moved quickly to lift physical distancing guidance with the oncoming summer months. While there remains much to be analyzed and written about the differences between the two states, it is likely that some combination of idiosyncrasy, physical distancing measures, and the weight of their implementation fundamentally shaped the population curve differences between both states. Ample infectious disease modeling has, at this point, showed that physical distancing measures—ranging from stay-at-home or shelter-in-place orders, to use of personal protective equipment—ultimately determine spread of the disease, and in this case then inform inter-population differences.

And yet, the epidemics in Massachusetts and Florida are marked by similarities, particularly around the extent to which the burden of mortality is disproportionately borne by persons living in assisted-living facilities. In both states more than half the deaths were among persons living in assisted-living facilities. The preponderance of cases in these facilities likely reflects the underlying likelihood of serious illness among persons with co-morbid conditions, and the limited capacity of contact controls within these facilities nationwide. Hence, the causes of difference between these two populations were relatively distinct from the causes of similarity of the types of persons who were most affected within each of these populations. Understanding this helps focus both on the causes of pandemic spread, and on the persons most at risk who

could—with a more nuanced approach to the pandemic—have been more systematically protected, limiting the extraordinary mortality that characterized older age groups infected during this pandemic.

Second, we can consider an example moving beyond geography and thinking of particular population groups. We know that racial minorities, particularly Black Americans, are bearing a disproportionate burden of the coronavirus pandemic in the US.[4] But this observation masks different causes of incidence and of morbidity and mortality. The cause of disproportionate disease in Black Americans is, of course, greater risk of exposure to the virus. The causes of these causes are directly tied to the downstream consequences of legacies of oppression, including socioeconomic position and job class. Occupational categories disproportionately assumed by Black Americans include those that cannot be conducted remotely, resulting in a greater risk of exposure to viral transmission, be it on public transportation, or in work places that were, in the earlier part of the pandemic, poorly prepared to provide opportunities for physical distance. Physical spaces also determine risk of morbidity and mortality. For example, hospitals that serve largely still segregated neighborhoods determine extent of equipment and space availability. Hence, the causes of greater incidence are likely directly tied to contemporary economic and occupational circumstance. By contrast, the causes of conditional risk of harmful consequences of the virus, that is the greater likelihood of death among those infected, are likely quite different than the causes of incidence, including age and underlying morbidity, which then patterns the differential likelihood of serious consequences from viral infection.

Thus, the causes of differences in the rates and timing of infections between the two states (e.g. physical distancing adherence) may be distinct from the differences in rates of infection or mortality within a state (e.g. living in an assisted living facility). Meanwhile, the causes of the disproportionate incidence among Black Americans are quite different than the causes of mortality among Black Americans. Both examples show simply how the causes of difference across populations are different than the causes of difference in health within populations, pointing to different points of intervention that can mitigate the consequences of the virus.

3. The causes of population health are multilevel, accumulate throughout the life course

The coronavirus pandemic is unprecedented compared to other global health crises in recent years, and it upended global function in a way that previous epidemics, experienced more locally and less visibly through ubiquitous media, did not. Insofar as this is a novel coronavirus, the cause of Covid-19 illness is, simply, a new pathogen, one which was at the beginning poorly understood and that challenged both science and clinical approaches, resulting in substantial downstream consequences in actions taken to contain its spread. A biological perspective on the disease suggests that the experiences of 2020 stem

from a novel virus, and that, as such, a shut-down of global wet markets, where the original transmission to humans is likely to have occurred, will protect us from future pandemics. However, considered another way, the causes of the pandemic itself are far more distal than the virus entering the body.

While the original transmission may have been the first step in the pandemic, the course of the pandemic was determined by a range of factors that had little to do with the SARS-CoV-2 virus itself. First, the conditions of pandemic spread were shaped by politics and the global capacity (or lack thereof) to respond to the virus in a coordinated fashion with well-known and time-tested cost-effective public health measures. This stems both from cross-national mistrust and national self-interest, and a chronic under-investment in a public health infrastructure that can successfully mitigate the spread of a novel virus. In the US, for example, local, state health departments and the Centers for Disease Control and Prevention (CDC) are chronically underfunded, resulting in a sufficiently under-performing public health infrastructure at a time when the country needed it most.[5,6] This was as much a cause of the epidemic curve as was the virus itself and the biologic conditions on which the virus thrived.

The conditions that create a healthier world around us are determinative of the causes of viral spread, both of persons who were likely to acquire the virus and persons who were likelier to get sick when they did acquire the virus. It is abundantly clear that persons who were less able to physically distance, whose occupational category precluded them from shifting to remote work, were likelier to acquire the infection in the early days of the pandemic when efforts at personal protection were nascent. In addition, the persons who were likelier to bear most of the morbidity and the mortality of Covid-19 were persons who had pre-existing conditions that themselves are socially patterned over a lifecourse of exposure. For example, emerging data from the pandemic are showing that there is a substantial burden of mental illness, including greater than doubling of population mood anxiety disorders due to Covid-19. In addition, these data show that the burden of this mental illness is accumulating more among persons with lower incomes and who are otherwise marginalized.[7] That the burden of poor mental health falls on these groups is, in many ways, to be expected, given the increased strain of stressful life events and underinvestment in public infrastructure for treatment well before the pandemic.

Hence, social patterning of pre-existing medical conditions predisposed populations to the consequences of SARS-CoV-2. While this pandemic was novel in 2020, its population health footprint was established long before the novel coronavirus crossed over into humans, following causes that are influenced by causes at multiple levels of influence, from national and state policies, to local conditions of exposure, to forces that shape risk of other diseases that in turn predispose populations to Covid-19 infection and its consequences.

4. Improving population health may disadvantage some groups, values determine whether we prioritize efficiency or equity

One of the central stories of the Covid-19 moment is, undoubtedly, one of inequity in its consequences. Death rates from Covid-19 among Black Americans is more than twice what it is among non-Hispanic whites; rates are also higher among Native Americans. In addition, infection incidence is socially patterned with persons with lower income likelier to be infected and to experience more morbidity when they do acquire Covid-19. This is all compounded by the economic downturn that is affecting persons with lower income and people of color more than other populations. This all makes a strong case for the centrality of health inequities at the heart of the population concern with the Covid-19 pandemic. It also, however, raises the question as to how much the world embraced an approach that prioritized health equity when faced with a novel coronavirus infection.

The short answer to this question is, not particularly much. As countries all over the world scrambled to address coronavirus, efforts were put in place to stop the spread of an unknown pathogen, more or less at all costs. In the process, conditions for shut down were imposed that put essential workers at risk, without much heed to the conditions of those essential workers. We could have done this differently. For example, we could have prioritized personal protective equipment for all workers, recognizing that the riskiest and most difficult occupations are those that we de-prioritize for wages and create structural conditions under which worker safety is not valued. Commensurately, little attention was paid to congregate populations, including for example those who were incarcerated and immigrants in detention, which soon came to experience infection rates that were substantially higher than that seen in the general population.

Why have we operated in this manner globally? In no small part we have operated on a narrative that has prioritized viral suppression at all costs. We have, as a society, rushed to do everything in our power to apply blunt instruments, often limited, to mitigate the spread of a novel pathogen. This was necessary at the time perhaps but was achieved at the expense of more vulnerable populations.

Could there have been an alternative approach? We were well aware that taking rapid and drastic action to contain viral spread using the blunt instruments of whole economic shut down was going to disproportionately disadvantage those groups who were already vulnerable. Yet, we proceeded to do so anyway, being willing, in essence, to privilege overall morbidity and mortality reduction, often at the expense of disproportionate burden among the most advantaged. Were we to have seen health equity as a core concern we would have moved quickly to change complete economic shutdown, perhaps to think creatively about personal protection and risk stratification that would have continued to allow viral control, but have prioritized social and economic livelihood as a core focus.

Covid-19 highlights therefore the tradeoffs we make between equity and efficiency and how these choices depend on a set of values, how policy decisions informed by epidemiology are contingent, whether we realize it or not, on the values that inform our thinking and our action. It also should call us to account for decisions made during the Covid-19 moment and put to rest, once and for all, the notion that science-informed policy actions are value free. It calls for a population health science that is willing to engage with questions about the value that inform its work.

To what extent, for example, did the early infectious disease models that informed many of the national decisions on global lockdowns also reckon with the disproportionate burden that these approaches would have on vulnerable populations? To what extent did we create tools to allow us to weigh the consequences of our actions in terms of costs for populations that already bear the cost of lifecourse burdens imposed by unfair economic circumstance? We suggest that we did little of this during the pandemic and stand to learn to do so in future if we are to balance equity and efficiency as we think about population health after future such events. Unfortunately, this is consistent with our global history of failing to prioritize and focus on the health of marginalized populations. Covid-19 gives us an opportunity to re-envision how we create and demand equity, to create a shared understanding that the priority of population health should be creating structures for all to thrive.

5. The magnitude of an effect of exposure on disease is dependent on the prevalence of the factors that interact with the exposure

One of the central challenges that we face in population health science is a focus on isolating casual effects of single exposures on outcomes, often to the detriment of understanding co-occurring causes that interact with, and inevitably shape, the very presence and magnitude of the exposure effect itself. This is abundantly in evidence in our science in the case of the Covid-19 pandemic. We were interested centrally, perhaps understandably, in the consequences of SARS-CoV-2. In this case our exposure was the virus, and the disease was Covid-19. There was an explosion of biomedical publishing about this relation, and the causal thinking X→Y is that the virus X, is associated then with the disease, Covid-19, as Y. But to what extent is X→Y informed by Z, a co-occurring cause that interacts with X, and in whose absence the X→ Y relation may be different, or even absent?

This is, of course, the essence of biological interaction, and determines why we see heterogeneity of associations in different contexts. Understanding that this relation exists puts us on a very different path of inquiry than thinking of all potential Z variables as alternate explanation, confounders, that require being adjusted for, or being explained away, rather than being seen as part of the mechanism that explains

how the cause manifests as disease. Take, for example, in the context of SARS-CoV-2, the role of age. We now know that age, likely both as a marker of underlying co-morbidity, and of immunosenescense, is associated with greater risk of both acquiring SARS-CoV-2, and of death from Covid-19 once the disease is acquired.[8] This suggests that age interacts with the other causes of infection (exposure risks due to physical contact for example) as well as with other causes of morbidity (underlying diabetes or heart disease for example) to be a factor as important as the virus itself in determining the scale and consequences of the Covid-19 pandemic. While surveillance rapidly came to recognize the unique role that age played in this pandemic, we fell far short of recognizing the import of this observation. As a result, throughout the US more than 80% of all deaths from Covid-19 are among persons older than the age of 65.[9]

We suggest that the gap between our vital statistics observation and our action to prioritize protection of people in particular age groups would have been in part minimized by a focus on epidemiological methods that assess interaction. The absence of comprehensive interaction assessments underlies our failure to understand that the magnitude of an effect of exposure on disease is dependent on the prevalence of the factors that interact with the exposure. Countries with more older people, like Italy, had substantially worse consequences of Covid-19 than did countries with younger people.[10] This reflects, rather simply, the central importance of age as an important, even if insufficient, covariate (i.e., age by itself, without SARS-CoV-2 infection does not result in Covid-19), one that changes our understanding of the disease through its intimate interaction with the key exposure, and should accordingly inform our public health action.

6. Prevention of disease yields a greater return on investment than curing disease after it has started

Perhaps the ultimate observation of the Covid-19 pandemic is the centrality of prevention to the work of population health science, and the consequent work of public health. The Covid-19 pandemic has, as of this writing, infected more than 10 million people worldwide, and resulted in more than 500,000 deaths. In the US alone, about 3 million people have been infected, and more than 125,000 people have died. Current estimates are that the global cost of the pandemic will be about $25 trillion; in the US costs to the economy are currently estimated at $8 trillion.[11,12] These are extraordinary costs for a pathogen that was unknown as recently as at the beginning of this year. They also far outweigh the costs of prevention. Separate and apart from the human costs, measured in morbidity and mortality that would not have happened were we not experiencing this pandemic, the financial costs of the pandemic are clearly far greater than the costs that might have helped substantially defray them. For example, the World Health Organization (WHO) budget is about $5 billion annually, and the budget of the US CDC is about $11 billion annually. Clearly if we tripled the budget of the WHO and the CDC, we would still be incurring an

annual cost that is a fraction of the actual cost of the pandemic amply illustrating the benefit of prevention in this case.

There are of course other considerations when thinking about our return on investment for efforts that mitigate the consequences of this pandemic. We now know that persons with underlying co-morbidities are substantially more likely to experience adverse consequence of SARS-CoV-2 infection than persons without these co-morbidities. These co-morbidities themselves are a result of disinvestment in population health that results in preventable disease and death, that itself costs the US economy more than $1 trillion annually.[13] We know that we can reduce morbidity and mortality if we invest in reducing disease that is, at core, preventable.[14] Now we recognize that in so doing we would also be creating a population that is healthier and more robust to the consequences of this particular pandemic and almost certainly to the consequences of future pandemics. Can this make the point sufficiently for a wholesale investment in prevention once and for all?

In conclusion

We are in early stages of our understanding of the full scale of the population health implications of the events of 2020. We expect that in coming years as data emerge, our thinking about the causes and consequences of the pandemic will sharpen and clarify. However, even with this state of our understanding, the principles of population health science can help us better understand the pandemic, its causes and consequences, and how they may shape our efforts at mitigation in the current moment and with future pandemics in mind. We hope, in particular, that this approach can help inform the architecture of our thinking, providing a scaffold for future population health science.

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