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COMMENTARY

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Systemic racism, chronic health inequities, and COVID-19: A syndemic in the making?

Clarence C. Gravlee

Department of Anthropology, University of Florida, Gainesville, Florida

Correspondence
Clarence C. Gravlee, Department of Anthropology, University of Florida, 1112 Turlington Hall, Gainesville, FL 32611-7305, USA.
Email: cgravlee@ufl.edu

For a brief moment, in the early days of COVID-19, some reports heralded the new coronavirus, SARS-CoV-2, as a "great equalizer." It is unlikely that any anthropologist, human biologist, historian, or public health scientist found this idea tempting. Pandemics always follow the fault lines of society—exposing and often magnifying power inequities that shape population health even in normal times (Wade, 2020).

Soon, that stark reality became clear to all. By early April, evidence began to emerge in the United States—first in Milwaukee, then in Detroit, eventually everywhere data were disaggregated by race—that mortality from COVID-19 was disproportionately affecting Black people and communities (Johnson & Buford, 2020). During the entire course of the pandemic so far, data compiled by the non-profit APM Research Lab (2020) has shown that the crude death rate for Black Americans is more than double that for all other racialized groups. When adjusted for age, the risk of death from COVID-19 is as much as nine times higher for African Americans than it is for whites (Bassett, Chen, & Krieger, 2020).

This inequity—as appalling as it is—may still underestimate the problem, as data remain woefully incomplete. Despite calls for comprehensive, nationwide data on COVID-19 cases and deaths by race and socioeconomic status, the U.S. federal government has no such system in place, and journalists and scholars have stepped in to collate disaggregated data by race from a patchwork of state health departments. The need for better data remains.

But data alone are not enough. We also need an explicit conceptual framework to know what the numbers mean, shape the questions researchers ask, and direct attention to appropriate public health and policy responses. In the absence of such a framework, the legacy of racial-genetic determinism in American medicine makes it likely that excess Black death will be interpreted as intrinsic Black vulnerability—a pattern that has already begun to emerge (Gravlee, 2020). Here I propose that the theory of syndemics may be a useful framework for making sense of the unfolding pandemic and directing future research on COVID-19.

1 | DEFINING SYNDEMICS

Merrill Singer and colleagues (Singer, 1994, 1996; Singer & Snipes, 1992) developed the concept of syndemics in the early 1990s, in the context of research on the HIV epidemic, which was then ravaging poor, Black, and other communities of color in urban North America. Singer built on the long-standing observation that communities most impacted by new epidemics often are already facing other threats to their health. In the case of HIV among marginalized people in the U.S., those threats included "a set of closely interrelated endemic and epidemic conditions (eg, HIV, TB, STDs, hepatitis, cirrhosis, infant mortality, drug abuse, suicide, homicide, etc.), all of which are strongly influenced and sustained by a broader set of political-economic and social factors" (Singer, 1996). The crucial point, Singer argued, was that these conditions did not merely co-occur; the *syn*ergy among epi*demics* made each worse.

Syndemic theory, then, integrates two concepts: disease concentration and disease interaction (Mendenhall & Singer, 2020; Tsai & Venkataramani, 2015). Disease concentration refers to the co-occurrence or clustering of multiple epidemics as a result of large-scale, political-

economic forces and adverse social conditions. Disease interaction refers to the ways that overlapping epidemics exacerbate the health effects of adverse social conditions, either through biological interactions between disease states or through interactions between biological and social processes.

Neither disease concentration nor disease interaction is unique to syndemic thinking; the uniqueness lies in their integration. Attention to disease concentration is a common feature of most frameworks for population health, including fundamental cause theory (Link & Phelan, 1995), ecosocial theory (Krieger, 2001), and the concepts of structural violence (Farmer, 2003) and structural vulnerability (Leatherman, 2005; Quesada, Hart, & Bourgois, 2011). Indeed, more than half a century ago, Cassel (1964) argued for the relevance of social-science theory to epidemiology by highlighting social processes that lead to the clustering of seemingly unrelated diseases (in his case, tuberculosis and schizophrenia). However, like Cassel, most models of population health frame the co-occurrence of epidemics in terms of the cumulative burden of disease. What the syndemic framework adds is the prediction that overlapping epidemics are more than the sum of the parts. Both (a) biological interactions between epidemics and (b) biosocial ones between epidemics and the social conditions that shape them can result in more suffering and death than would be expected in models that treat each disease in isolation.

The focus on disease interaction also has deep, historical roots, stretching back at least to Scrimshaw, Taylor, and Gordon's (1959) work on synergism and antagonism between nutrition and infection (see also Scrimshaw, 2003). Disease interaction is usually described in terms of comorbidity and multimorbidity (van den Akker, Buntinx, & Knottnerus, 1996). These concepts, which have gained wider currency in the context of COVID-19, draw attention to common etiological pathways across disease states and to the complexity of care for patients with more than one chronic disease. Comorbidity and multimorbidity are most salient in clinical medicine but are also relevant to epidemiology and health services research (Valderas, Starfield, Sibbald, Salisbury, & Roland, 2009). Even when invoked in epidemiology, however, the focus is on the distribution of comorbid conditions (and predisposing social conditions or risk factors) at the level of the individual (Barnett et al., 2012). If syndemic theory were concerned only with biological interactions at the individual level (eg, for people who were infected with both HIV and TB), it is not clear what value it would add beyond the framework of multimorbidity. The promise of the theory lies in raising questions *across levels of analysis* about interactions among clustered epidemics and the underlying social conditions that drive them. This unique perspective is what makes syndemic theory relevant to the COVID-19 pandemic.

2 | RELEVANCE TO COVID-19

The core tenets of syndemic theory, then, are that:

1. large-scale, political-economic forces, which play out over generations, result in deep-seated social, economic, and power inequities;
2. these inequities shape the distribution of risks and resources for health, resulting in the social and spatial clustering of epidemic diseases (*disease concentration*); and
3. some overlapping epidemics have synergistic effects due to (a) biological interactions between disease states or (b) interactions between biological processes and the social, economic, and power inequities that shape the distribution of health to begin with (*disease interaction).*

COVID-19 has made these ideas feel urgent from the start. In early March, for example, the *New York Times* highlighted the intersection of social factors that increase the risk of infection in impoverished communities, such as housing density and reliance on public transportation, and "disproportionately high rates of disease and illness" that make infection more deadly (Eligon, 2020). Days later *Time* predicted that people with low incomes—disproportionately Black, Indigenous, or other people of color—would face higher exposure to the virus (because they are less likely to be able to work from home, more likely to work in service sectors where contact with strangers is routine, more likely to live in multi-family apartment buildings) and had less access to sick leave and medical care if they did become sick (Vesoulis, 2020). In mid-May, when the New York City Health Department first released data on COVID-19 deaths by ZIP code, the prediction had borne out: the highest death rates were in low-income neighborhoods with disproportionate numbers of Black and Latinx people (Schwirtz & Cook, 2020).

The spatial concentration of COVID-19 death manifests on a broader scale, too. In early April, *The Atlantic* ran a story about the demographic distinctiveness of COVID-19 mortality in the American South (Newkirk, 2020), suggesting that younger people were dying there at higher rates than in other hard-hit regions because of the legacy of slavery and Jim Crow. The suspected pathway was a higher burden of chronic diseases like hypertension and diabetes, which followed

from social and political-economic factors such as poverty, limited government investment in health care, and mass incarceration, among others.[1]

These accounts by journalists and others paint a compelling picture. The challenge for researchers is to incorporate such observations into a theory that generates testable propositions about the links between systemic racism, chronic disease, and risk of mortality from COVID-19. That work has already begun. Social scientists and public health researchers have drawn attention to the structural conditions that shape the *concentration* of COVID-19 in communities already facing higher burdens of poverty, racial inequity, and disease (Khazanchi et al., 2020; Laster Pirtle, 2020; Williams & Cooper, 2020). Medical scientists and clinicians have emphasized the *interactions* among comorbid conditions that are overrepresented among COVID-19 hospitalizations and deaths—particularly hypertension (Pranata, Lim, Huang, Raharjo, & Lukito, 2020), diabetes (Kreutz et al., 2020), and obesity (Akoumianakis & Filippatos, 2020). Syndemic theory draws together both approaches, bridging the population- and individual-level perspectives of the social and medical sciences, and it adds new questions about possible interactions between COVID-19 and pre-existing social inequities that may exacerbate suffering from chronic diseases like hypertension and diabetes.

Figure 1 offers a tentative syndemic model to guide research in this area. The right side of the model highlights the *concentration* of and possible *interactions* among COVID-19 and two chronic conditions that seem to pose particular risks for people infected with SARS-CoV-2: hypertension and diabetes (Richardson et al., 2020; Yang et al., 2020). We do not yet understand why these conditions make COVID-19 more dangerous—or even, for certain, *if* they do[2]—but suspected pathways for disease interactions include the renin-angiotensin system (Kreutz et al., 2020), the endothelium (Sardu et al., 2020), and inflammatory dysregulation (Mahmudpour, Roozbeh, Keshavarz, Farrokhi, & Nabipour, 2020). Because these systems are also involved in diabetes and hypertension, researchers are pursuing the hypothesis that those conditions interact with COVID-19 to make infection with SARS-CoV-2 more deadly.

Emerging evidence suggests that COVID-19, in turn, may exacerbate the risk of cardiometabolic disease. Rubino et al. (2020) propose that SARS-CoV-2 may have pleiotropic effects on glucose metabolism that could complicate pre-existing diabetes and lead to new onset of diabetes in people with COVID-19. Other coronaviruses, including the one that caused the original severe acute respiratory syndrome (SARS-CoV), are known to have long-term effects on cardiovascular health. People who survived SARS in 2002 to 2003 exhibited altered lipid metabolism 12 years later (Wu et al., 2017). It is too early to know whether COVID-19 will have similar effects, but it is possible that people who recover from the new coronavirus may experience long-lasting damage that increases the risks associated with hypertension and heart disease.

As we learn more about COVID-19 and its long-term sequelae, Figure 1 provides a framework to develop specific hypotheses about how it interacts with chronic conditions like hypertension and diabetes. The simultaneous attention to disease interaction and disease concentration keeps in the foreground that physiological dysregulation and possible interactions between

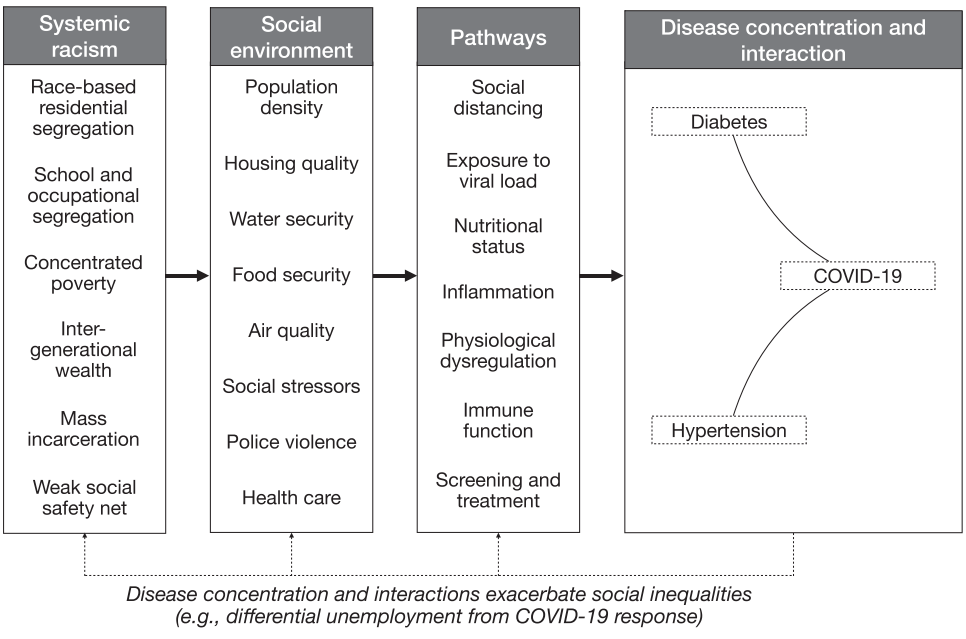

FIGURE 1 A tentative syndemic model of systemic racism, cardiometabolic disease, and COVID-19 in the United States

COVID-19 and cardiometabolic disease are socially patterned. Indeed, the same physiological systems that are the focus of interactions among hypertension, diabetes, and COVID-19 are also involved in the pathways that link neighborhood disadvantage, racial discrimination, and poverty to racial inequities in hypertension and diabetes (Cobb, Parker, & Thorpe, 2020; Dolezsar, McGrath, Herzig, & Miller, 2014; Dusendang et al., 2019; Lei, Beach, & Simons, 2018; Panza et al., 2019; Simons et al., 2018). It is plausible, therefore, that the COVID-19 pandemic in the U.S. involves both kinds of interactions put forward by syndemic theory: (a) biological interactions between overlapping diseases (ie, diabetes, hypertension, and COVID-19) and (b) biosocial ones between noxious social conditions and the biological processes involved in progression of SARS-CoV-2 infection to COVID-19 risk.

The left side of Figure 1 specifies some of the noxious social conditions. It delineates systemic racism as a set of policies and structures that spawn toxic environments and identifies behavioral and physiological pathways that mediate the social and spatial concentration of disease. Here the model departs from other conceptual diagrams of syndemic interactions, which often focus only on overlapping epidemics, "without reference to the social forces conditioning exposure" (Tsai, Mendenhall, Trostle, & Kawachi, 2017, p. 978; see also Singer, 1996). The reason for this departure is that lack of specificity about how large-scale, political-economic forces translate to individual biology impedes progress in testing the theory. My goal in specifying the conditions that shape exposure across multiple levels of analysis is to stimulate further development of testable propositions and research questions that advance the state of syndemic theory beyond a useful heuristic.

Pursuing that goal does not require new theory. Figure 1 draws on widely tested and supported models for research on racism and health (Phelan & Link, 2015; Schulz, Williams, Israel, & Lempert, 2002; Williams & Mohammed, 2013) and on the role of health equity in pandemic preparedness (Quinn & Kumar, 2014). Building on such work links syndemic theory to allied strategies for explaining racial health inequities—before, during, and likely after COVID-19—and clarifies what syndemic thinking adds to interdisciplinary efforts. The unique contribution of syndemic theory is the integrative focus on disease concentration and disease interaction in the context of large-scale, long-term, political-economic forces.

Figure 1 identifies systemic racism (Feagin, 2006) as a fundamental cause of racial inequities in disease concentration. This perspective sees the social patterning of hypertension, diabetes, and now COVID-19 as culminating from a system of racial oppression that has developed and morphed over four centuries—from settler-colonialism and chattel slavery to race-based residential segregation and mass incarceration. Systemic racism constitutes a fundamental cause (Phelan & Link, 2015) in the sense that it shapes the risk of risk through multiple, interchangeable pathways (see also Laster Pirtle, 2020). Some of those pathways lead to increased risk of diabetes, some to hypertension, some to COVID-19—and some to combinations of the three.

For example, race-based residential segregation, a result of deliberate social policy (Rothstein, 2017), has far-reaching consequences for health. It shapes the social and spatial distribution of both risks and resources, including the quality of schools, employment opportunities, density and quality of housing, availability of healthy food, exposure to pollution, threat of police violence, and access to quality health care (Williams & Collins, 2001). These aspects of the social environment, in turn, have implications for cardiometabolic conditions through unequal nutritional status, inflammation, and physiological dysregulation (eg, Lei et al., 2018; Morenoff et al., 2007). Some of these pathways (eg, inflammation) may also increase susceptibility to COVID-19, while other aspects of residential segregation may increase exposure, rather than susceptibility, to the novel coronavirus in the first place (eg, density of housing or inability to follow social-distancing guidelines leading to higher viral load).

Still other pathways have both COVID-19 and cardiometabolic disease as endpoints. For example, air pollution increases the risk of hypertension and diabetes (Coogan et al., 2012) and has been proposed as a risk factor for COVID-19 (Zhu, Xie, Huang, & Cao, 2020). The racialized structure of American labor entails differential exposure to COVID-19 (Hawkins, 2020) and to occupational stressors related to hypertension (Cuevas, Williams, & Albert, 2017). Mass incarceration unjustly impacts Black people and communities, with consequences for both COVID-19 and cardiometabolic disease. Incarcerated people face both greater exposure to SARS-CoV-2 (Akiyama, Spaulding, & Rich, 2020) and elevated risk of hypertension and heart disease (Wang et al., 2009), and nonincarcerated Black people living in neighborhoods with high rates of incarceration have higher rates of cardiometabolic disease, independent of individual- and neighborhood-level factors like poverty and rates of crime (Topel et al., 2018).

All population health frameworks draw attention to the social production of health inequities. The value added by a syndemic perspective is that it also highlights how biosocial interactions move in both directions. Not only do social inequities shape the risk of COVID-19; COVID-19 is also likely to exacerbate social inequities, further harming

health. For example, devastating job losses during the pandemic have disproportionately affected Black Americans (Gould & Wilson, 2020), and the economic fallout from COVID-19 has magnified racial inequities in income and housing (Greene & McCargo, 2020). Likewise, the online transition of K-12 and university teaching threatens to widen racial inequities in educational opportunities, given that federal policies subsidize internet access in disproportionately white, rural contexts but not in cities where residents are disproportionately Black and other people of color (Siefer & Callahan, 2020). Further, in regions where COVID-19 is concentrated, the strain on healthcare systems may compound pre-existing inequities in access to care (Williams & Rucker, 2000). Already we see evidence of racial inequities in COVID-19 treatment (Eligon & Burch, 2020), and we know that discrimination in healthcare settings adversely affects management of chronic conditions like diabetes (Peek, Wagner, Tang, Baker, & Chin, 2011).

Note that each of these scenarios—unemployment, income, housing, education, health care—involves synergies between biological and social processes at the population level. They hint at how overlapping epidemics may not merely co-occur but rather interact to make matters worse. Much of the media commentary has focused on how comorbidities like hypertension and diabetes increase the risk of COVID-19 becoming deadly. Syndemic theory alerts us, in addition, to the possibility that the pandemic could intensify racial inequities in the social and economic conditions that increase risk for hypertension and diabetes to begin with, exacerbating the toll those diseases already take on Black people and communities. The possibility of such synergistic effects—over the short and long term—underscores the relevance of syndemic thinking.

3 | CHALLENGES AND FUTURE DIRECTIONS

The concept of syndemics has a broader reach than most anthropological ideas. It gained institutional backing from the U.S. Centers for Disease Control (Milstein, 2002); was recently the focus of a special collection in one of the world's highest-impact journals (The Lancet, 2017); generates several dozen scientific articles every year (Mendenhall & Singer, 2020); is widely taught across disciplines (Singer, 2009); and is the basis of multiple interventions with real-world impact on public health (eg, Chakrapani, Kaur, Tsai, Newman, & Kumar, 2020). It is clearly an idea that matters.

Yet recent commentaries highlight shortcomings in the burgeoning literature about syndemics. A key conceptual issue, which has methodological implications, is the distinction between syndemics and overlapping epidemics that merely co-occur or are mutually causal (Tsai et al., 2017). The defining feature of a syndemic is disease *interaction* in addition to disease *concentration*. That is, true syndemics have synergistic effects that can be traced to biological interactions between disease processes (bio-bio) or between biological processes and social conditions that harm health (biosocial). Such interactions, which need to be tested explicitly, are thought to exacerbate the toll of epidemics through multiplicative, not just additive, effects.

Most studies that purport to be about syndemics do not provide evidence of such interactions. In a recent scoping review, Singer, Bulled, and Ostrach (2020) identified 188 articles about syndemics that were published during 2015 to 2019 and found that only 12% (23 articles) met the full definition of a syndemic, including evidence of disease interaction. Tsai and colleagues (Tsai, 2018; Tsai & Venkataramani, 2015) identified the analytical problems, and Mendenhall and Singer (2020) outlined research strategies that researchers are beginning to adopt to measure synergistic effects. For now, however, the central proposition of syndemic theory remains largely untested. COVID-19 presents an urgent case for specifying testable hypotheses about interactions with chronic cardiometabolic diseases that exacerbate pre-existing inequities. The model in Figure 1 is meant as a framework for developing such hypotheses as we learn more about COVID-19.

A related conceptual challenge, which again has methodological implications, concerns the level of analysis at which interactions take place. Like other frameworks for population health, syndemic theory is inherently multilevel. It proposes that large-scale, political-economic forces, which often play out over centuries, have embodied consequences for individual health (cf. Gravlee, 2009). This proposition makes syndemic theory a logical fit for multilevel analyses, which incorporate population-level and contextual effects, but existing syndemic studies have measured only individual-level factors (Tsai, 2018). This focus may result, in part, from the emphasis on biological interactions between disease states. After all, to borrow an example from Singer et al. (2020), the pathogen-pathogen interaction that makes co-infection with HIV and Hepatitis C more lethal than infection with Hepatitis C alone occurs in individual bodies. These interactions matter, but they are also captured by the concepts of co- or multimorbidity. The utility of syndemic theory is that it directs attention to possible interactions not only between *diseases* (at the individual level) but also between *epidemics* (at the population level), taking social context and political-economic inequities into account.

The tentative syndemic model of COVID-19 I offer here addresses these challenges by outlining causal pathways from large-scale social forces to individual biology and positing synergistic interactions at the individual and population level. The intersection of systemic racism, chronic health inequities, and COVID-19—apparent to journalists and other commentators—puts the onus on researchers to refine and test the syndemic model and develop public health and policy responses that account for potential synergistic effects.

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AUTHOR CONTRIBUTIONS

Clarence Gravlee: Conceptualization; writing-original draft; writing-review and editing.

ORCID

Clarence C. Gravlee https://orcid.org/0000-0002-0913-2320

ENDNOTES

[1] This kind of analysis was also common on social media like Twitter. Pulitzer Prize-winning journalist Nikole Hannah-Jones, for example, published a series of well-referenced posts that tied together the synergistic effects of (a) social conditions that make Black Americans more likely to get infected with SARS-Cov-2 (eg, higher rates of employment in service sectors where contact with strangers is routine and working from home is not an option, lower rates of home ownership resulting in increased housing density) and (b) underlying health conditions (particularly hypertension and diabetes) that make COVID-19 more dangerous once infected. https://twitter.com/nhannahjones/status/1247176506452905986.

[2] An alternative hypothesis is that hypertension and diabetes are overrepresented in people who are hospitalized or die due to COVID-19 because those conditions are associated with age, not because they are involved in the pathophysiology of COVID-19.

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