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Exposure, Susceptibility, and Recovery: A Framework for Examining the Intersection of the Social and Physical Environments and Infectious Disease Risk

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Despite well-documented evidence that structurally disadvantaged populations are disproportionately affected by infectious diseases, our understanding of the pathways that connect structural disadvantage to the burden of infectious diseases is limited. We propose a conceptual framework to facilitate more rigorous examination and testing of hypothesized mechanisms through which social and environmental factors shape the burden of infectious diseases and lead to persistent inequities. Drawing upon the principles laid out by Link and Phelan in their landmark paper on social conditions (*J Health Soc Behav*. 1995;(spec no.):80–94), we offer an explication of potential pathways through which structural disadvantage (e.g., racism, sexism, and economic deprivation) operates to produce infectious disease inequities. Specifically, we describe how the social environment affects an individual's risk of infectious disease by 1) increasing exposure to infectious pathogens and 2) increasing susceptibility to infection. This framework will facilitate both the systematic examination of the ways in which structural disadvantage shapes the burden of infectious disease and the design of interventions that can disrupt these pathways.

communicable diseases; disease susceptibility; disease transmission; racism; social environment; socioeconomic factors; vectorborne diseases; waterborne diseases

Abbreviations: COVID-19, coronavirus disease 2019; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.

Editor's note: *An invited commentary on this article and the authors' response will appear in an upcoming issue.*

Since the time of John Snow, epidemiologists have known that the neighborhood environment is essential to our understanding of the transmission of infectious pathogens, but we have struggled to systematically integrate the social and physical environment into our conceptualization of infectious disease risk (1). The coronavirus disease 2019 (COVID-19) pandemic has revealed yet again how infectious pathogens are distributed along lines of social disadvantage, including disadvantage at the neighborhood level (2). The stark racial and ethnic inequities in the burden of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infections, hospitalizations, and deaths are clear evidence of the role that structural disadvantage plays in determining who bears the brunt of infectious pathogens worldwide (2).

There is ample evidence that structurally disadvantaged populations are disproportionately affected by established (e.g., tuberculosis (3)) and emergent (e.g., COVID-19) infectious diseases (4). For example, from 2004 to 2012, non-Hispanic Black individuals in Michigan had an incidence rate of recently transmitted tuberculosis 25 times that of non-Hispanic Whites after controlling for nativity (3). Prior to COVID-19, tuberculosis was the leading cause of death from a single infectious pathogen globally, with an estimated 1.4 million deaths annually, disproportionately affecting those living with human immunodeficiency virus, living in poverty, and lacking access to basic infrastructure and health care (5). Similar disparities are observed with emer-

gent pathogens like SARS-CoV-2. A recent study examining the number of COVID-19 tests performed and the proportion of positive tests in New York City neighborhoods found that non-White and disadvantaged neighborhoods had both fewer tests given and a higher proportion of positive results (4). Globally, COVID-19 has disproportionately affected populations living in crowded settings with limited ability to engage in mitigation strategies or access health care (6).

Despite evidence of persistent inequities in the burden of infectious diseases, critical gaps persist in our understanding of how biological, environmental, and social factors increase both an individual's exposure and susceptibility to infectious pathogens. While exposure and susceptibility are individual-level parameters, they are rooted in the structural-level factors of racism, sexism, and economic deprivation. We refer to these collectively as structural disadvantage. Additionally, while the components of structural disadvantage do act independently, they are also interconnected and together shape population-level vulnerability and drive inequities in health outcomes. Structural racism, for example, results in increased incidence of infectious disease in racialized minority communities by determining the environments in which individuals can live, work, and play and is inextricably tied to economic deprivation (7). Racialized minority populations consistently have lower levels of vaccination coverage against diseases like influenza and pneumococcus due to financial stress and limited health-care access (8) while having higher rates of comorbidity, resulting in increased susceptibility to both infection and severe disease. The higher rates of comorbidity experienced by certain populations are themselves expressions of structural disadvantage that place individuals in unequal contexts with limited resources to promote health and well-being and prevent disease.

Macrolevel processes such as racism, sexism, and economic deprivation may be best conceptualized as "fundamental causes" of infectious disease burden, as they influence the burden of multiple infectious pathogens, they operate through different intervening pathways, they constrain resources that individuals have to mitigate risk of exposure and infection, and the resulting inequities are consistently reproduced over time (9). Fundamental cause theory was first proposed by Link and Phelan in 1995 (9). However, much of the nuance of their theoretical framework has since been lost. In fact, in their original paper, Link and Phelan proposed 2 conceptual frameworks, one of which focused on identifying the "fundamental" causes of disease (what is now known as fundamental cause theory). The other emphasized the importance of contextualizing more proximate or individual-level risk factors within the broader social context so as to understand the particular social conditions under which individual-level factors are related to disease. In proposing these 2 frameworks, Link and Phelan understood the importance of a complementary approach that acknowledges both structural disadvantages as upstream determinants of disease and the pathways that mediate the relationship between structural disadvantage and disease. Recognizing that there are root or fundamental causes of these processes does not negate the need to address mediating mechanisms. While addressing inequity at the root level is the goal, intervention on more proximal links in the chain can help reduce inequities when the intervention is designed and implemented within the larger context of a disease's social and environmental drivers.

Drawing upon the principles laid out by Link and Phelan in their landmark paper (9), we offer an explication of some potential pathways through which structural disadvantage operates to increase inequities in infectious disease burden. Specifically, we present a conceptual framework describing how the social environment impacts an individual's risk of infectious disease (Figures 1 and 2), hypothesizing that this occurs through 2 related pathways: 1) increasing exposure to infectious pathogens (Figure 1) and 2) increasing susceptibility to infection (Figure 2). We developed a set of 3 questions to guide our selection of social and environmental constructs:

Question 1: Has the relationship between this construct and infectious disease been well-documented with regards to established and/or emergent infectious diseases?

Question 2: Is there a coherent and plausible causal pathway (i.e., social, environmental, biological) through which this construct operates?

Question 3: Could an intervention be conceived of to disrupt this pathway?

We acknowledge that the resulting set of constructs and pathways is incomplete. There are many other social and environmental factors that act on these pathways that were omitted for the sake of parsimony and clarity, just as not all intersections, directionalities, and/or feedback loops between the constructs could be displayed. Collective power, for example, was omitted (at least explicitly) from the structural level, as we felt it is an inherent component of racism, sexism, and economic deprivation. While we present the exposure pathway (Figure 1) as distinct from the susceptibility pathway (Figure 2), in reality they are highly interrelated. We also delineated between structural-level and individual-level factors when there may not always be a clear distinction between the two. Indeed, in line with Link and Phelan's frameworks (9), we conceptualize these factors as occurring on a gradient whereby the structural-level factors reverberate down to affect individual-level risk.

We want to make clear, however, that this proposed framework highlights the importance of both considering mediating mechanisms and addressing structural-level factors. This is neither an explicit nor an implicit promotion of the idea that there are underlying biological differences that may explain persistent social inequities in the distribution of infectious disease, nor can these inequities be mitigated solely by intervening on individual health behaviors. There have been several salient examples that illustrate this approach since the start of the COVID-19 pandemic. For example, in a 2021 study, Marquez et al. (10) explored interventions to reduce the disproportionate burden of COVID-19 morbidity and mortality within Latinx communities likely associated with reduced access to and uptake of vaccination. They developed a low-barrier, client-centered, community-based vaccination site that also leveraged social networks to provide vaccination, thereby addressing several structural barriers to receiving vaccination in the Latinx community, including historical distrust in the medical system,

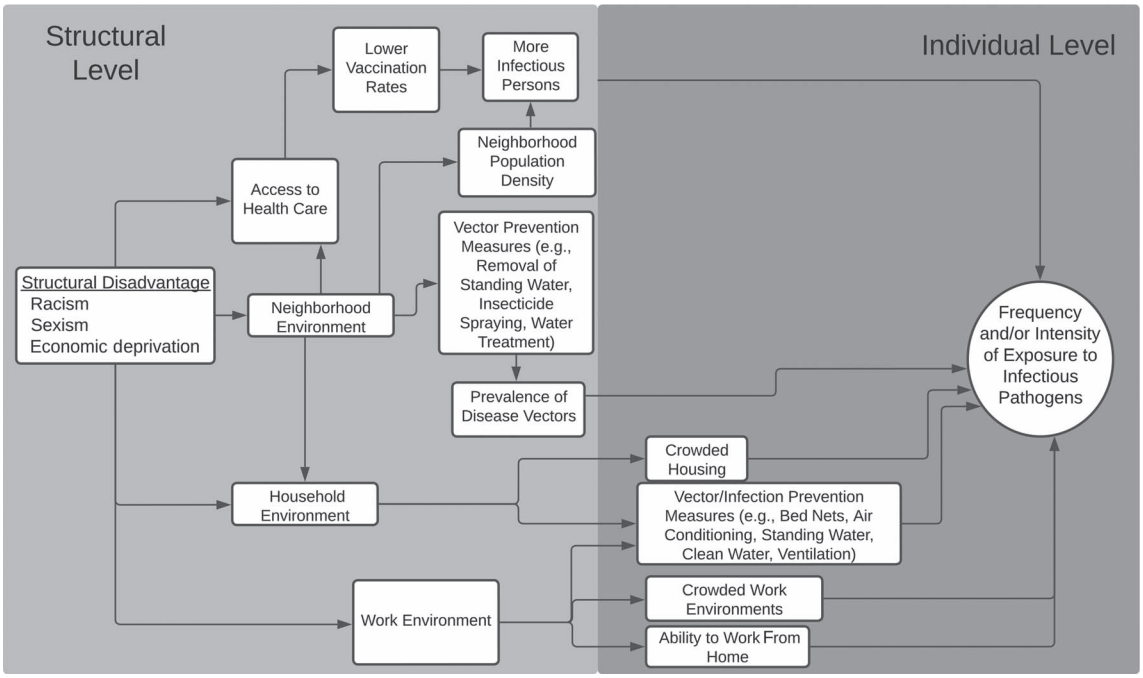

Figure 1. Conceptual framework illustrating the hypothesized mechanisms through which structural disadvantage, as defined by racism, sexism, and economic disadvantage, increases the frequency and intensity of exposure to infectious pathogens. The conceptual framework for this pathway is described at both the structural level (depicted in light gray) and the individual level (depicted in darker gray).

misinformation, and lack of access to health care. In another 2021 study, Nande et al. (11) demonstrated that the eviction moratorium that occurred in the city of Philadelphia, Pennsylvania, led to substantial reductions in individual-level risk of SARS-CoV-2. A small study in North Carolina showed that providing financial incentives (e.g., a cash card) to individuals to receive the SARS-CoV-2 vaccine effectively removed barriers to receipt of vaccination, particularly for low-income and racialized minority populations (12). Notably, these inequities in COVID-19 burden and vaccination uptake can be seen across a myriad of pathogens, suggesting that these interventions represent promising examples of how interventions designed within the broader social and environmental context can mitigate persistent health inequities.

INCREASED EXPOSURE TO INFECTIOUS PATHOGENS

Individual-level infectious disease risk is necessarily related to the frequency and intensity of a person's contact with an infectious pathogen. One's contact with infectious pathogens is a function of 3 primary spaces: the household, the neighborhood, and the work environment (Figure 1).

The household

The household environment has been a well-studied mechanism for understanding infectious disease risk, with numerous studies identifying household crowding as a risk factor for a number of infectious diseases, such as respiratory syncytial virus, tuberculosis, and cholera (13–15). Because individuals spend a substantial amount of time in their homes, environmental factors such as poor ventilation, temperature, and humidity can also inform the level of exposure a person has to infectious diseases (16). During the COVID-19 pandemic, household risk was also driven by exposure to an essential worker. Fifty-seven percent of Black adults at high risk for severe COVID-19 outcomes lived in households with at least 1 other adult who was not able to work from home, as compared with 47% of Whites at high risk for severe COVID-19 outcomes (17). The household environment is also relevant for understanding exposure to vector- and waterborne pathogens. For example, though estimates of the protective effects of household water treatment and safe water storage on diarrheal disease reduction have varied widely (18), having piped water in the home reduced the odds of childhood diarrhea by 24% in a study in Indonesia (19). Further, a study in Ethiopia found that households not using antimalarial mosquito spray had 6 times the odds of a positive malaria test as households that were sprayed (20); important risk factors for asymptomatic malaria infection also exist at the household level (21).

The neighborhood

Neighborhood environments are comprised of social, physical, and environmental factors that influence contact patterns between individuals and thereby influence the

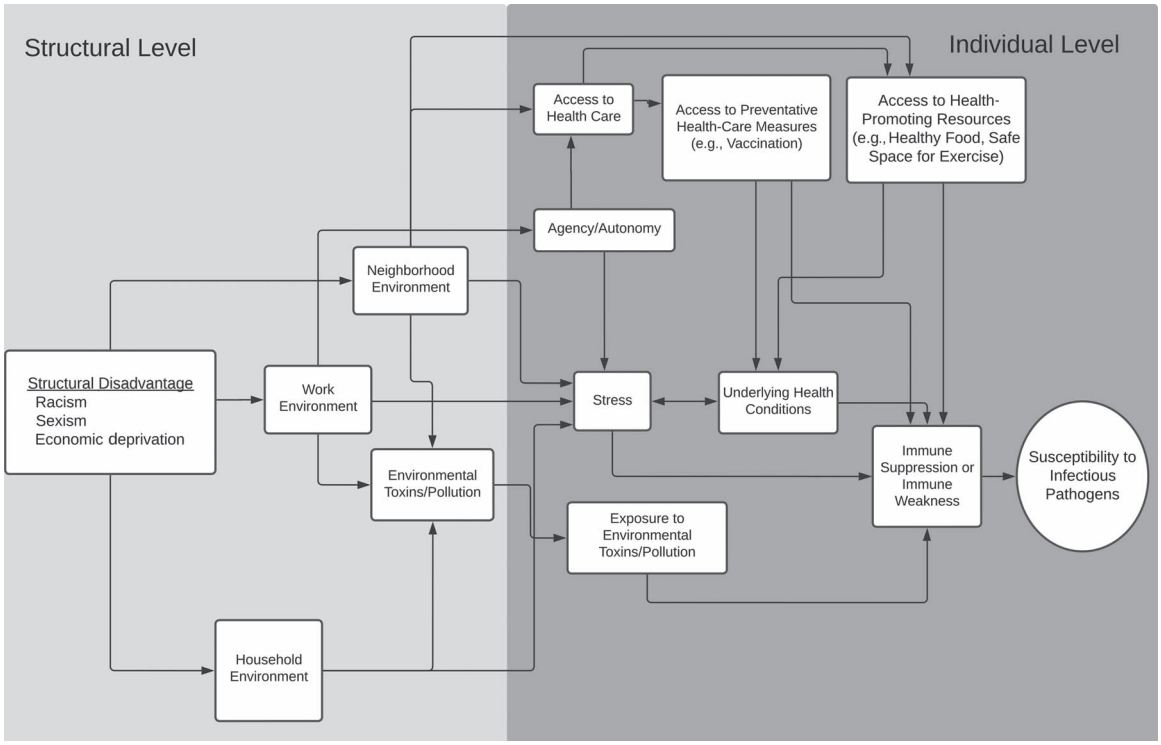

Figure 2. Conceptual framework illustrating the hypothesized mechanisms through which structural disadvantage, as defined by racism, sexism, and economic disadvantage, increases the likelihood that an individual will contract a disease given exposure to an infectious pathogen. The conceptual framework for this pathway is described at both the structural level (depicted in light gray) and the individual level (depicted in darker gray).

level of exposure to pathogens. One mechanism through which structural racism operates is by constraining where individuals are able to live and work (22, 23). Historical processes such as redlining or other forms of social segregation have led to a spatial concentration of disadvantages such as densely populated neighborhoods (24), economies reliant on lower-wage jobs deemed essential or with limited ability to work from home, more individuals working multiple jobs, a dependence on public transportation (25), and obstacles to accessing essential resources like health care (26). These factors increase exposure by increasing close contact with others, diminishing access to resources that enhance primary prevention (e.g., education, mask distribution, vaccine accessibility), and/or limiting early identification of infection and the ability to quarantine. Moreover, environmental and physical features of the neighborhood can have real impacts on individuals' exposure to vectorborne pathogens (27). For example, a study of malaria in the Democratic Republic of the Congo found that living within a 2-minute walk of an area with frequent standing water was associated with increased odds of malaria (28). For vectorborne pathogens especially, the surrounding neighborhood environment may pose a greater risk than individual-level prevention behaviors can overcome.

The work environment

Work environments have been and continue to be sources of infectious disease outbreaks. Lower-wage essential jobs in manufacturing and/or service industries are often characterized by poor and crowded working conditions (29), limited availability of protections against exposure to pathogens (30), lack of ability to work from home (31), and lack of paid sick leave, which helps mitigate illness and transmission (32, 33). Racialized minority individuals and immigrants account for a disproportionate share of the essential workforce, where they are forced to be in contact with SARS-CoV-2 with limited protections like personal protective equipment (30).

INCREASED SUSCEPTIBILITY TO INFECTIOUS PATHOGENS

While exposure to an infectious pathogen is a necessary cause of infectious disease, the underlying immune response can mitigate or promote infection once exposure has occurred. We define susceptibility as the likelihood that an individual will contract an infectious disease when exposed to a particular pathogen. A more direct link between structural disadvantage and susceptibility to infection can

be seen in the persistent disparity of vaccine coverage in racialized minority and disadvantaged populations (34, 35). Beyond this, we conceptualize 3 primary ways in which the social and physical environment increase susceptibility to an infectious pathogen: stress, underlying health conditions, and immune suppression/immune weakness (Figure 2). Notably, we hypothesize that both the stress and underlying health conditions pathways operate by modulating the immune response. However, we also believe there are social and environmental factors that act directly on the immune system, and thus this factor should be considered separately. Additionally, several key factors that operate in the exposure pathway also operate in the susceptibility pathway. For example, the work and neighborhood environments not only affect the probability of exposure to an infectious pathogen but also act in the susceptibility pathway through experiences of chronic stress. For instance, individuals with limited autonomy in their workplace exhibit higher levels of chronic stress (36), as do individuals living in disadvantaged neighborhoods (37).

Stress

Social stress theory has been widely cited as a biological mechanism explaining the link between social disadvantage and poor health outcomes (38). It posits that individuals who endure persistent social disadvantage from experiences like discrimination, joblessness, housing instability, and food insecurity are differentially exposed to chronic stress across the life course (38). Indeed, the disproportionate experience of chronic stress from racism-related discrimination is one major mechanism through which structural racism affects the health of racialized minority populations (39, 40). Chronic stress causes increased wear and tear across a number of body systems (41), including the immune system, which may in turn increase susceptibility to infection when a person is exposed to a pathogen. Studies show that chronic stress is associated with increased inflammation (42) and impaired immune function (43, 44). In a landmark study, Cohen et al. (45) empirically demonstrated this pathway, showing a clear dose-response relationship wherein participants reporting higher levels of baseline stress had increased incidence of respiratory illness following viral challenge.

Underlying health conditions

Underlying health conditions are widely cited as risk factors for a number of infectious diseases, particularly those causing respiratory infections (46). Studies of respiratory syncytial virus have found significantly higher rates of respiratory syncytial virus mortality among children with underlying health conditions in comparison with those without such conditions (15). Obesity has been associated with impaired cellular immune responses and increases in proinflammatory cytokine levels (47) and has been linked to longer viral shedding of influenza (48), increasing the potential for disease transmission. Moreover, low-income and racialized minority populations consistently report higher rates of conditions like hypertension, obesity, and diabetes and high mortality from these conditions as well (49). Again, focusing on underlying health conditions as predictors of individual susceptibility to infectious disease should not mask the root cause of the disproportionate burden of chronic health conditions: structural disadvantage.

Immune suppression/immune weakness

While both stress and underlying health conditions are likely to have an impact on the immune system, there are several other features of the social and physical environment that may directly affect the immune response. For example, living or working in areas with higher levels of air pollution can suppress the immune system and make individuals more susceptible to an infectious pathogen (50, 51). In addition, neighborhoods are critical resources for accessing health-promoting activities (e.g., healthy food, safe areas for exercise) that can prevent underlying immune suppression (52, 53). Moreover, both the neighborhood and the work environment are also points of access for health care, including the ability to get vaccinated for an infectious disease (54, 55). By limiting access to such routine medical care and preventive measures, the neighborhood and work environments can have a direct adverse effect on the strength of an individual's immune response against infection.

DISCUSSION

In this paper, we present a conceptual framework to encourage more rigorous examination and testing of pathways through which social and environmental factors cause persistent inequities in infectious disease burden. Specifically, we believe that this framework effectively demonstrates the manner in which the downstream effects of structural disadvantage shape infectious disease risk and transmission. Additionally, we have attempted to do so through diagrams that are sufficiently detailed to capture the complex interactions involved in these pathways, while retaining enough simplicity to be useful in research and policy development. We believe that such a systematic examination will facilitate the design of interventions that integrate the complex interactions between biological, social, and environmental factors that drive these inequities. For far too long, those of us in the study of infectious disease control have relied on a biomedical paradigm of disease focusing on the pathogen and host, giving little attention to the larger environment in which this interaction occurs. While it has been nearly 30 years since Link and Phelan published their seminal paper on social conditions as fundamental causes (9), we have largely forgotten their important message that contextualizing risk factors can allow for effective interventions on intermediate mechanisms. Structural disadvantage undoubtedly influences who is exposed, who is susceptible, and who can recover from an infectious disease. Though this commentary focuses on the first 2 pathways, examining how structural disadvantage influences an individual's ability to recover their health and social/economic well-being after infection is critical for future study.

Further, in studies of infectious disease transmission and typically in response to outbreaks, a compartmental model may be employed to study the mechanistic behavior of an infectious pathogen in a simulated, closed population to identify parameters like the incubation period and the reproduction number, or to assess the effect of different interventions and fit to real data. Though metapopulation models may be employed to consider spatial heterogeneity and age-structured models to account for demographic heterogeneity of key parameters, they often do not consider the larger social and physical environments that dynamically contribute to differential rates of exposure and susceptibility (56). This may be due to a variety of factors, including the lack of a theoretical framework to guide this sort of inquiry, a lack of sufficient data on the social environment, and historically an insufficient acknowledgement of the importance of social factors in studies of infectious disease (1, 57). When fitting such models to real-world populations, a lack of data and model structure (i.e., overparameterization) may limit the ability to incorporate such aspects. However, our understanding and documentation of how transmission may occur differentially across populations and environments may be improved if we consider fundamental cause theory as well as the broader social and environmental context underpinning infectious disease transmission.

We highlight recent COVID-19 studies that serve as exemplars for how to employ this sort of thinking in infectious disease research. Recent studies have demonstrated different ways of incorporating social data (e.g., mobility and race/ethnicity) into transmission models (11, 58, 59). The investigation by Ma and Lipsitch (58), in particular, showed that by incorporating underlying population heterogeneity, one could see that racial/ethnic disparities in infection rates in the New York City metropolitan area were probably driven by higher levels of exposure among racialized minority populations than among Whites and ultimately expressions of structural racism. This study offers a compelling example of how, with the acknowledgement of the ways in which structural disadvantage can affect risk of infectious disease, incorporating a single social variable, race/ethnicity, into transmission models can substantively enhance our understanding of epidemic spread. On the other hand, the study by Nande et al. (11) offers an example of how transmission modeling can be used to investigate the effects of a specific social policy (e.g., eviction moratoria) on epidemic spread. They found that evictions have a measurable impact on SARS-CoV-2 spread beyond only those households directly experiencing evictions, and demonstrated the value of incorporating specific social policies and not just the standard social variables (e.g., race/ethnicity, education, socioeconomic status).

In closing, we offer 2 recommendations to better position us in infectious disease research and control to disrupt longstanding inequities. First, we must shift the focus from individuals to the underlying systems putting individuals at risk. While medicine is necessarily individual-centered, focusing on individual behavioral change without considering the barriers introduced by structural disadvantage will only perpetuate existing inequity. For example, recommending that an individual work from home to mitigate exposure to an infectious pathogen ignores the underlying privilege that affords such opportunity. Combining such a recommendation with a program to provide personal protective equipment and safer work environments to those unable to work from home could help reduce the disparities such an intervention may introduce. The benefit of considering the larger social context can be seen in a recent pilot program which suggests that small financial incentives can effectively remove obstacles to SARS-CoV-2 vaccination that disproportionately affect racialized minority populations (e.g., lack of child care, transportation, etc.) (12). Second, we must move beyond simply documenting health inequities and identify the upstream factors or intervening pathways that are likely to be amenable to policy change. It is well documented that structurally disadvantaged populations have fewer opportunities for health and wellness and are at increased risk for many deleterious health outcomes. While fundamental cause theory calls necessary attention to the larger systems of racism and disadvantage that will affect health regardless of the intervening mechanisms, we contend that articulating the downstream mechanisms is still worthwhile so long as it supports the development of interventions that may have real effects on individuals' infectious disease risk. Even still, we assert that researchers and policymakers should consider the social and environmental context in which infectious disease burden is shaped when designing policies or interventions focused on more upstream factors.

While impactful change to such "fundamental causes" to sustainably reduce infectious disease risk may require seismic shifts in political ideologies and resource distribution, building the mechanistic evidence base from which such changes can begin to occur downstream is the first step. Delineating the mechanisms through which racism, sexism, and economic deprivation work to increase exposure and susceptibility to infectious pathogens can provide much-needed support for policy decisions meant to reduce inequities.

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