Redefining Resilience as a Public Health Response to Stress

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Abstract

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Stress, a transdiagnostic risk factor linked to both physical and psychological illness, is important to population health and health equity. Promoting population resilience, or the capability for populations to "bounce back" in the face of stressors, may act as a public health antidote to stress-related illness by bolstering a population's capability to cope and adapt following varied stresses.

Despite extensive research into resilience at the individual level, little is known about its potential at the population level. This dissertation leverages the COVID-19 pandemic to advance the understanding of population resilience. Specific aims are: 1) Develop a structural model of population health as an alternative to the biomedical model and propose "structural resilience" as a population health outcome; 2) Compare the biomedical and structural models in the context of population-level distress during the COVID-19 pandemic; 3) Explore population psychological distress as a potential measure for the construct of "structural resilience."

REDEFINING RESILIENCE AS A PUBLIC HEALTH RESPONSE TO STRESS

INTRODUCTION

Two problems motivate this dissertation.

The first comes from the academic literature. In recent decades, growing interest in and investigation into the social determinants of health have described risk and protective factors ranging from the behavioral (e.g. barriers to adoption (Tong et al., 2020)) to the social (e.g. peer influences, social identities, isolation (Cacioppo et al., 2015; Dowd et al., 2009)), structural (e.g. built environments, political institutions, structural discrimination (Acolin et al., 2022; Krieger, 2001; Sharkey & Faber, 2014)) and environmental (e.g. air pollution, climate change (Kravitz-Wirtz et al., 2018)). It has become clear that, while healthcare and medical intervention are the cornerstone to the treatment and eradication of disease, they are neither necessary nor sufficient for illness prevention and health promotion.

This proliferation of evidence has inundated the field and produced widespread agreement that social determinants are important and urgent targets for public health intervention. As a broad-based umbrella term, the "social determinants of health" provide a unifying direction for the field (Allen et al., 2014; World Health Organization, 2008). Yet, dig into the specifics and the veneer of unity is replaced by dissent, confusion, and paralysis. *Which* social determinants deserve the greatest priority, and *why*? *How* can public health policy effectively intervene? *Who* is responsible for enacting solutions—the medical establishment, political figures, policymakers, community members, academics? And *to whom* ought attention be paid—historically underrepresented sociodemographic groups, medically high-risk individuals, city-dwellers, rural citizens, older adults, young people, sexual and gender minorities, immigrants, undocumented individuals, and the list goes on?

Evidence exists to make the case for any of the listed options. From one perspective, this may mean there is no bad option. Given the breadth and scope of the health impacts of social determinants, any action taken within any domain is bound to lead to, at the very least, some marginal improvement. This perspective, implicitly if not explicitly, guides much of the current work.

From another perspective, though, the conflicting approaches reflect an inefficient and ineffective use of limited resources that risk exacerbating tensions related to scarcity and competition—all while the ill effects of social risks on health accumulate to make the problem increasingly difficult to resolve. From this perspective, the marginal improvements of single interventions are outweighed by the steady, persistent, and mounting harms caused by a social and structural environment fundamentally at odds with human health and well-being.

This dissertation takes the latter perspective as a starting point. Its objective is to take a wide-angled view on the social determinants literature to tackle—or rather, *attempt* to tackle—the overarching problem of how to understand and improve overall population health, not as the sum of specific social or structural risks, but as a function of generalized social and structural conditions. This is the first problem.

The second problem comes from my own experiences.

For six years, I worked as a mental health therapist, advocate, and case manager for individuals eligible for services by the dubious virtue of being a victim of crime or being low-income with a severe mental illness. This meant, by extension, that I worked with individuals experiencing discrimination, food and housing insecurity, addiction, interpersonal violence, justice system involvement, administrative burden, language barriers, family conflict, and unmet medical needs. My days were filled with phone calls to hospitals, housing providers, and Social Security offices, de-escalating potentially violent clients, and mountains of paperwork. In the face of these overwhelming and interconnected challenges, I found myself in the ludicrous position of offering, at the most, one hour of individual counseling per week to clients to focus on "problem-solving," helping them to "gain coping skills," and "practice emotional regulation." I delivered, to the best of my ability, evidence-based clinical interventions targeting individual-level mechanisms in the face of overwhelming structural odds.

It was not lost on me that, after the workday, I returned home to my comfortable townhouse, less than two blocks away but serenely nestled in a quiet cul-de-sac next to a publicly maintained playground where I would take my son on weekends. For each hour of work, I earned a living wage while my clients—who arguably worked as hard as I did in our sessions—received nothing. Public support for the playground was celebrated, while public funds for their basic needs were criticized. And yet, labels aside, our time, space, and experiences were shared. We used the same roads to get to the outpatient clinic, watched the same shows on

Netflix, and were stressed out by the same hateful rhetoric. Our lives were connected, even though our social and economic environment tried to keep us separate.

I have come to see this separation as a problem, and it is the second problem that motivates this dissertation. Individual lives in modern society are inextricably linked. We rely on the same infrastructure for electricity, food, and transportation, not to mention the internet access and digital devices on which we increasingly rely. We draw on the same natural resources and experience the same climate. The geographic and technological barriers that separated ancient societies have been surmounted, and complete self-sufficiency is an illusion. Beyond a common humanity binding us together—granted, not a trivial connection—we co-exist in a common physical world.

Yet, our existences are separated by structural boundaries, political theatrics, social identities, and more. Nowhere is this contradiction more apparent than in our treatment of mental health. The causes of psychological distress, beyond the non-modifiable influences of biological predisposition or temperament, are social. Taken together, chronic and acute social stresses are estimated to explain over 40% of the variance in individual distress (Turner et al., 1995; Wheaton et al., 2013). This means that the causes of distress reside in our social and structural environment—which we share—and likely have a shared impact. Yet, mental distress is treated as an individual problem. Despite growing movement towards community treatments, the current gold standard is for diagnosed individuals to be cloistered in private rooms with individual therapists or treated in the locked wards of psychiatric hospitals, separated from purportedly "healthy" individuals.

I believe that continued separation of individual distress from the shared human experience is a problem for two reasons. First, individual-level treatments are important but ultimately temporary fixes. Without addressing the fundamental social and structural causes of distress, relapse is not merely a risk but a certainty.

Second, given the shared causes of distress, letting non-symptomatic individuals "opt-out" of addressing the common social and structural issues forces the burden on the few, creating inequity while stymying effective solutions. Distress, from the perspective of emotion theory, signals that there is a problem and motivates responsive action, as physical pain motivates protective behavior. Distress due to social or structural causes is thus a signal that social or structural issues require attention. Yet, given the choice, individuals who can may "opt-out" of

dealing with this distress. This leaves individuals who cannot opt out, for reasons that range from lack of resources to severity or immediacy of threat, left to face the issue alone.

To my clients, the harms of housing shortages were immediate, visceral, and life-threatening. To me, they were distant, hypothetical, and inconvenient. Insufficient housing leads to individuals living on the same streets and in the same parks that I may frequent. We share a common structural environment. But, if the experience becomes unpleasant, I can return home, creating psychological as well as physical distance between myself and the problem. This distance, ultimately, is an illusion. The same problem of housing shortage, which is a structural issue, impacts us both. However, the problem is only urgent for my clients, and so the burden falls disproportionately on them.

Beyond being inequitable, this approach is neither sustainable nor likely to succeed. The forces causing housing shortages, as well as those causing financial turmoil, or increased medical costs, or violent crime, and so on, are social and structural and, as a result, impact us all. Yet, the motivation to act—namely, the experience of distress—has been isolated to the few. The treatment of psychological distress as an individual issue separate from the collective experience is, I believe, at the heart of the problem.

This dissertation is my attempt to begin addressing these two problems.

The first paper, entitled “Beyond the biomedical, towards the agentic: A paradigm shift for population health,” describes a roadmap for tackling the issues raised. In it, I propose a conceptual model for population health as a function of generalized social and structural conditions, in response to the first problem. I propose the construct of “structural resilience” as the manifestation and modifiable response to the second problem of shared distress.

The second paper, entitled “Explaining population psychological distress during COVID-19: A systematic comparison of the biomedical and structural models for population health,” explores empirical evidence for distress as a shared, rather than individual, experience. This paper integrates empirical analysis with conceptual argument to make the case for the advantages of construing distress as a shared, rather than individual, phenomenon.

The third paper, entitled “Evidence for population distress as a structural predictor of vaccine hesitancy,” examines whether state-level psychological distress, as a structural predictor, is

associated with vaccine hesitancy. It is an empirical exploration into the consequences of distress as a shared structural phenomenon, guided by the model set out in the first paper.

The problems motivating this dissertation are large, and the three papers here do not constitute a solution. As I argue in the first paper, addressing these problems requires a paradigm shift in population health science. In the face of established scientific tradition, exploring this paradigm shift has been, and will continue to be, ridden with pitfalls, skeptics, and null results. This endeavor is as likely to fail as to succeed, and the possibility that I am completely and delusionally wrong haunts me daily. Yet, I continue to try. This dissertation is the result of my attempts thus far.

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TITLE: Beyond the biomedical, towards the agentic: A paradigm shift for population health science

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ABSTRACT

Life expectancy in the United States is decreasing. Health disparities are widening. Growing evidence for and integration of social and structural determinants into theory and practice has not yet improved outcomes. The COVID-19 pandemic reinforced the fact.

In this paper, we argue that the biomedical model and its underlying scientific paradigm of causal determinism, which currently dominate population health, cannot meet population health needs.

While criticism of the biomedical model is not new, this paper advances the field by going beyond criticism to recognize the need for a paradigm shift. In the first half of the paper, we present a critical analysis of the biomedical model and the paradigm of causal determinism. In the second half, we outline the agentic paradigm and present a structural model of health based on generalizable, group-level processes. We use the experience of the COVID-19 pandemic to illustrate the practical applications of our model. It will be important for future work to investigate the empirical and pragmatic applications of our structural model of population health.

KEYWORDS: *population health theory, biomedical model, causal determinism, agentic paradigm, structural resilience, congruence, COVID-19*

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INTRODUCTION

Population health science faces a problem. American life expectancy is decreasing (Woolf & Schoomaker, 2019). Life expectancy at birth remains six years lower for African Americans, compared to White Americans (Hill et al., 2022). Most recently, the COVID-19 pandemic highlighted systemic threats to morbidity and mortality that reinforced ongoing social disparities in the United States.

Growing evidence indicates that social and structural dynamics are critical to population health, perhaps even more than individual-level behaviors and treatments (Krieger, 2001; Williams, 1997). While researchers and policy-makers emphasize the role of social determinants of health, they remain guided by a biomedical model that is incompatible with population health needs.

Criticism of the biomedical model is not new (Pearce, 1996). This paper advances the field by going beyond criticism to recognize the need for a paradigm shift. From this realization emerges an alternate, structural model of population health based on generalizable, group-level processes. We use the experience of the COVID-19 pandemic to illustrate the practical applications of our model. It will be important for future work to investigate the empirical and pragmatic applications of our structural model of population health.

Paradigms, models, and theories: A taxonomy

Three related, but distinct, terms are key to this paper. Since these terms are used at times interchangeably, we offer these distinctions to clarify their use in this paper.

First, the term “paradigm,” popularized by Thomas Kuhn (1970), refers to a set of principles through which the questions and results of scientific research are organized and understood. A Kuhnian paradigm so deeply pervades a scientific enterprise as to shape perceptions of what is accepted as a truth.

“Model” refers to a simplified description of natural phenomenon, situated within a scientific paradigm and intended to support predictive reasoning and scientific inquiry. Examples include animal models in the biological sciences or models of rational decision-making in microeconomics. The current paper discusses the biomedical model, which conceptualizes health as a function of risk factors, symptoms, diagnoses, and treatments.

Finally, “theory” refers to a set of testable hypotheses regarding a specific phenomenon. A theory within the biomedical model may explain diabetes as a disease of insulin deficiency caused by

genetic predisposition and dietary habits (risk factors) and characterized by measurable blood glucose levels (symptoms).

A paradigm shift occurs when existing models are unable to account for emerging evidence. Historic examples of paradigm shifts include the shift from heliocentrism to geocentrism in astronomy or the shift from intelligent design to evolution in biology (Kuhn, 1970). In each case, existing models became increasingly unable to advance scientific inquiry.

We draw attention to the biomedical model's underlying scientific paradigm, rather than constraining discussion to the biomedical model itself, because alternative models will continue to fall short if the underlying scientific paradigm is not also questioned.

An operational definition of population health science

A "population" is a group of individuals within a shared structural environment. This shared structural environment links individual health outcomes over and above individual-level processes, thus rendering "population health" a meaningful construct distinct from the simple aggregation of individual health outcomes.

"Population health science" is the scientific study of the health of populations. This orientation towards *populations* distinguishes population health science from related fields like preventive medicine and public health. These fields are focused on promoting population health by preventing disease and disability in *individuals*, rather than populations (American College of Preventive Medicine, 2023; CDC, n.d.; CDC Foundation, 2021; Clarke, 1974). Preventive medicine specialists work within healthcare facilities and clinics, while public health practitioners work outside of the medical setting. In both cases, individuals are the targets of interventions such as medical treatments, behavior change, or environmental exposures.

Individual health is complementary to, but distinct from, population health. The structural environment links individual outcomes over and above individual-level processes to produce group-level processes. A singular focus on individual-level processes neglects parallel but distinct group-level processes. Unlike public health and preventive medicine, population health science is concerned with the group-level processes that produce population health.

SECTION I: BEYOND THE BIOMEDICAL

1.1COVID-19: A population health problem

COVID-19 mortality and morbidity were higher in the United States than in other wealthy industrialized nations (Mueller & Lutz, 2022). This was due in part to low vaccination uptake, with barely 60% of the US population having received the recommended course by December 2021, as well as lack of adherence to social distancing and masking (CDC, 2022). This excess mortality emerged despite the United State having high quality healthcare providers and facilities and producing an effective vaccine years earlier than is typical (Mueller & Lutz, 2022).

The biomedical model shaped the COVID-19 response in the United States. Understanding the failures requires a critical examination, not only of the model itself, but also its underlying scientific paradigm.

1.2 The roots of the biomedical model: Germ theory

The biomedical model's role in population health science is rooted in the success of germ theory. The role of microscopic pathogens in causing disease is now well-established in modern medicine. Empirical support obscures the fact, however, that germ theory is a *theory* that explains the specific phenomenon of infectious disease. Germ theory initially competed with alternate theories of disease, including the miasma theory, hygiene theory, and morality theory (Schwartz et al., 1999; Tomes, 1990). The story of John Snow ending the 1854 cholera outbreak in England by closing an infected water pump gained fame, not only as an example of epidemiological genius, but as pioneering evidence in support of the burgeoning "Germ Theory" (Cameron & Jones, 1983).

Germ theory posits that microorganisms external to the individual cause disease and that, in turn, eradicating these microorganisms will eradicate disease. Scientific research has since identified pathogens, characterized disease processes, and developed efficacious interventions to reduce exposure to or neutralize the adverse effects of these "germs." Previously deadly diseases, such as influenza, now present minimal threat in developed countries. Morbidity and mortality have decreased in the 20th century (Case & Deaton, 2017), and life expectancy at birth in the United States is now over 30 years longer than it was in 1900 (CDC, 1999).

The biomedical model is a generalization of germ theory's basic mechanism. It widens germ theory's specific focus on infectious microorganisms to implicate "risk factors," which range from environmental pollutants (Kravitz-Wirtz et al., 2018) and genetics (Lakhani et al., 2019) to

socioeconomic status (Glymour et al., 2014) or stress (McEwen & McEwen, 2017). Illness is understood as a constellation of symptoms. Treatments minimize exposure to risk factors or mitigate their effects to reduce symptoms.

Both germ theory and the biomedical model have advanced individual health. Yet, their empirical contributions have overshadowed critical appraisal of their underlying scientific paradigm.

1.3 The underlying paradigm: Causal determinism

Causal determinism conceives nature as comprised of a collection of independent and self-contained entities—the prototypical metaphor is of billiard balls. A causal relationship occurs when two otherwise unconnected entities come into contact. Billiard ball A slams into ball B, causing ball B to roll into the pocket. In the case of infectious diseases, an external pathogen infects the human body, causing the human body to manifest symptoms of illness.

The biomedical model is grounded in causal determinism. Health is achieved by preventing the initial collision with a risk factor or modifying the subsequent trajectory: John Snow identified an infected water pump and neutralized the threat by closing the pump (Cameron & Jones, 1983). Personal hygiene, such as hand-washing, prevents individuals from encountering infectious pathogens. Medical interventions, such as vaccination or treatment, modify the disease trajectory following infection.

Two principles of causal determinism currently organize population health science. First, a true causal effect is identified when an outcome results from a distinct and independent exposure, "all else equal" (Shimonovich et al., 2021). Methodological developments, from the Bradford-Hill criteria and the potential outcomes framework in epidemiology to treating randomized controlled trials as the gold-standard and econometric methods for causal inference (Koepsell & Weiss, 2014; Rubin, 2005), emerge from this organizing principle.

Population health scientists use "all else equal" causality to assess whether the relationship between measurable exposures (risk factors) and known diagnoses (symptom clusters) is causal. Empirical findings are organized by type of exposure or diagnostic category. Causal determinism promotes diagnosis-specific scientific inquiry.

Determinism, the second principle, assumes that outcomes can be predicted if information on all possible causes is known and that outcomes will change if antecedent causes are altered. Within the health context, determinism provides the framework for biomedical treatment. After identifying

causal effects, interventions focus on changing antecedent causes to deterministically alter health outcomes.

Research within the biomedical model is interpreted and understood through causal determinism. As the backdrop for scientific progress, causal determinism is rarely examined. Yet, an explicit description of the paradigm's underlying principles is essential to understanding the need for a paradigmatic shift within population health science.

1.4 Causal determinism and social determinants of health models

Threats to health have evolved from infectious to chronic diseases, first among industrialized nations and increasingly throughout the world (Badash et al., 2017). In response, the biomedical model and causal determinism have been generalized to non-biologic causes of disease. Theoretical and empirical developments regarding the "social determinants of health" result from this trend.

One example is the Health Belief Model (HBM) (Rosenstock, 1974), a social psychology theory that frames health behaviors as a key non-biomedical cause of health. It conceptualizes health behaviors as produced by the interaction of perceived benefits, harms, and self-efficacy (Bandura, 1986). The HBM has been influential across multiple domains, including smoking cessation, chronic disease management, vaccination, and preventive care (ex. Tong et al., 2020).

The Andersen-Newman Model (ANM) describes the predisposing, enabling, and need factors that drive health services utilization as a determinant of health outcomes (Andersen, 1995). Developed in the 1960s, the ANM represents an early attempt to bridge the health and social science fields and is most notable for its role in bringing social determinants into the purview of the health sciences. It remains one of the most-referenced theories in health services research.

Pioneering work explored the role of social networks and norms in health (ex. Rosenquist et al., 2011). Social network dynamics, relationship quality, and social capital predict outcomes ranging from smoking to obesity and depression (Umberson & Montez, 2010; Waldinger & Schulz, 2010). This research has catalyzed intervention strategies that leverage social mechanisms to promote positive health behaviors (ex. Dempsey et al., 2018).

Similarly, neighborhood effects research documents that neighborhood conditions, including disorder, housing, crime, green space, and community resources, impact individual health (Roux, 2016; Sharkey & Faber, 2014). The adage that your zip code matters more than your genetic code emerged from this work and drives research and policy from the local to federal levels in the

United States, including the Neighborhood Revitalization Initiative spearheaded by the Obama Administration (Office of Urban Affairs, n.d.).

Despite a focus on social, rather than biomedical, risk factors, causal determinism continues to shape these contributions: The "housing laws" billiard ball strikes "neighborhood conditions," which rolls towards "access to health services" to hit "the individual," sending "the individual" rolling towards the pocket of "health" or "disease" (Figure 1).

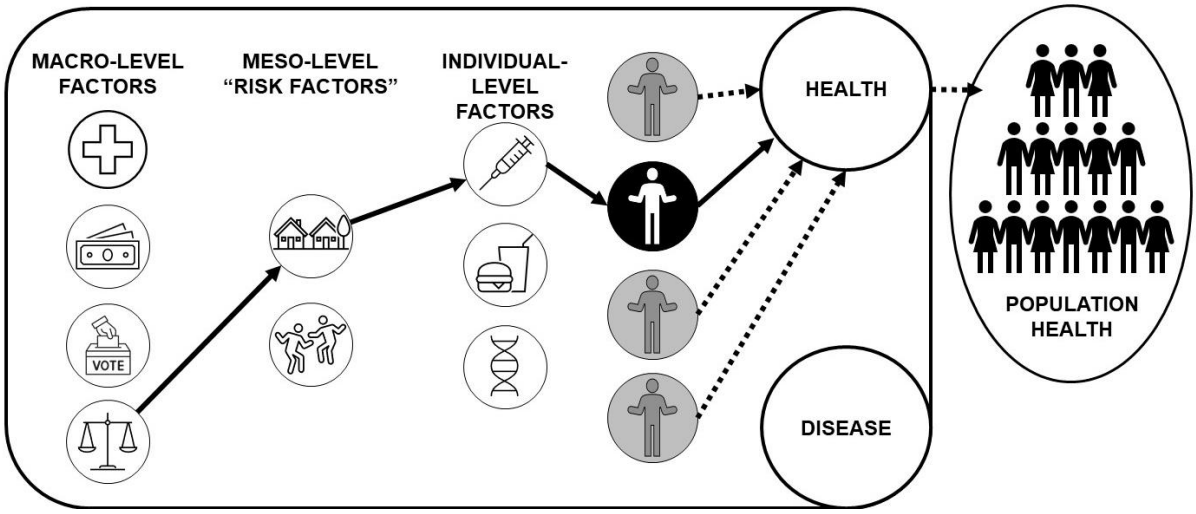

Figure 1: Social determinants and causal determinism. The "housing laws" billiard ball strikes "neighborhood conditions," which rolls towards "access to health services" to hit "the individual," sending "the individual" rolling towards the pocket of "health" or "disease." Population health is conceived as equivalent to aggregate individual health.

Treating social risks as "*determinants* of health" reflects causal determinism. Interventions focus on preventing a collision, perhaps via "neighborhood improvement" initiatives, or modifying the trajectory following collision, perhaps via education and outreach. Social determinants models posit that population health is achieved when all the individual billiard balls are successfully pushed into the "health" pocket rather than the "disease" pocket. The individual is little more than an object upon which risk factors fall and whose outcome is causally determined.

1.5 Causal determinism and population health science are incompatible

Causal determinism's organizing principles have enabled researchers to document the persistent disparities by race and class, the negative effects of poverty and trauma, and the wide-ranging impacts of social and economic policy. Yet, as in previous paradigmatic shifts, a tipping point has

been reached. More evidence is not improving outcomes. The principles of causal determinism are failing to organize the evidence base into a coherent and actionable population health science.

Causal determinism, while suitable for the study of biologic exposures and physical processes, is incompatible with the study of population health due to fundamental inconsistencies between the paradigm's organizing principles and the demands of population health.

First, population health science is concerned, not with individuals, but with populations—groups whose daily lives, behaviors, and health outcomes are bound together by shared environments, institutions, values, and norms (Keyes & Galea, 2016). It is at the population level that phenomena of social class, income inequity, or discrimination become meaningful; that neighborhood stratification, policy development, and resource allocation occur; and that population health is defined.

Consequently, the paradigmatic principle of "all else equal" causality cannot hold, because the "exposures" and "outcomes" of interest in population health are interconnected and internal to the population ecosystem. For instance, modifications to social policy have ramifications for health policy. Studies quantifying isolated effects may document causal relationships but ultimately fails to predict dynamic and interconnected real-world reactions.

Second, documented disparities by race, class, and gender across disease categories (ex. Alsan et al., 2020; Hearne & Niño, 2022) suggest that the underlying process transcends diagnoses. The focus on disease-specific pathways encouraged by causal determinism is incompatible with the rising demand for a transdiagnostic solution.

Third, efforts to predict and modify individual behavior have produced limited returns because individual behavior is not entirely causally determined. Individuals are not billiard balls. Billiard balls lack intention, meaning-making, exploration, or satisfaction. Their movements are entirely environmentally determined. By contrast, individuals can, within constraints, choose how they respond to their environment. Individual behaviors emerge from a process of choice and decision making that "differs qualitatively from their constituent elements and therefore are not reducible to them" (Bandura, 2001). This has resulted in unexpected responses to behavioral interventions.

A pathogen does not choose to infect a host, nor can it refuse to allow treatment to act on it. Research and interventions grounded in causal determinism can be effective in the biomedical context. By contrast, choice is fundamental to human behavior. A patient has the ability and right

to say no. Biomedical treatments are necessary but not sufficient for population health. Causal determinism neglects this fact.

1.6 COVID-19: A failed strategy grounded in causal determinism

The COVID-19 pandemic helps to illustrate these paradigmatic inconsistencies. Experts conceived of the pandemic in biomedical terms and prioritized pharmaceutical development and behavioral interventions. Under causal determinism, these interventions would have nudged the population towards health, as opposed to disease. However, the outcomes predicted by causal determinism were not realized.

First, the COVID-19 pandemic threatened population health over and above aggregate individual, health. The pandemic had ramifications on the shared systems of transportation, healthcare, housing, food, and others that bind the population together. Lockdowns, mask mandates, and vaccine availability aimed at encouraging individual health behaviors had social and economic ramifications. Cash transfers designed to combat the impact of low-income altered the political landscape. Online learning intended to reduce educational disparities had unintended mental health consequences. “All else equal” causal determinism failed to anticipate and respond to these interconnected processes.

Second, efforts to influence or direct human behavior during the pandemic produced backlashes. Informed by studies documenting the effectiveness of social distancing and face coverings for preventing viral spread (ex. Chu et al., 2020; Lyu & Wehby, 2020; Teslya et al., 2020), governments shut down businesses. Public health communications encouraged masking and vaccination, and social pressures and medical authority aimed to induce desired behaviors (Raifman et al., 2021).

In some circles—most notably, highly educated segments of the population—these efforts were more likely to succeed (Chen et al., 2020; Gatter & Mohapatra, 2020). However, in others, rejection of the recommendations was unexpectedly strong (Allcott et al., 2020; Berg & Lin, 2020; Iacobucci, 2021; Romer & Jamieson, 2020). Politicians disseminated messages making health behaviors central to political identity. Mask-wearing, and vaccination came to signal political affiliation (Deane et al., 2021). Causal determinism did not account for individual refusals to comply.

These problems were not about insufficient evidence. They emerged because the assumptions of causal determinism were incompatible with population health.

SECTION II: BEYOND THE BIOMEDICAL—BUT NOT FAR ENOUGH

To some, the failures of the United States' COVID-19 strategy were unsurprising. A parallel academic tradition has critiqued the biomedical model, particularly in relation to non-biologic risks, and produced a range of models intended to resolve these issues.

2.1 Structural determinants of health models

Structural determinants models begin with the metaphor of upstream/downstream or distal/proximal causes (Krieger, 2008). Upstream or distal causes of health are those that place an individual at *risk* of risks (Link & Phelan, 1995)—they shape the distribution of risks at the population, rather than individual, level. As Geoffrey Rose wrote, the causes of health in individual cases of disease are distinct from the causes of disease incidence (Rose, 1985). It is questions of disease incidence that occur at the population level.

Fundamental cause theory (Link & Phelan, 1995) implicates differential resource access via socioeconomic status as causing the persistent and widening gap in health outcomes. This "fundamental cause" of health disparities goes beyond simple causal determinism to implicate multiple, simultaneous, and co-occurring processes (Freese & Lutfey, 2011). A fundamental cause is like a force that lifts the entire pool table—changing the course of any single ball will not arrest the overall process.

Social production of disease theory, rooted in sociology, explicates the social mechanisms—including political context (Torche & Rauf, 2021), social closure (Murphy, 1984), and stratification (Sahdra & Ross, 2007)—underlying the distribution of disease in a population. Klinenberg's "social autopsy" of the Chicago heat wave (1999) is an exemplary example of this theory. Ecosocial theory, developed by Nancy Krieger (2001), moves beyond socioeconomic status to implicate social, political, and economic structures of discrimination by race, sex, and gender in the production of population health. These structural exposures get "under the skin" via embodiment, where the adverse social conditions created by structural discrimination set off stress-related biological processes that produce poor health.

The relationship between health and structural factors such as social class (Glymour et al., 2014; Lutfey & Freese, 2005; Mirowsky & Ross, 2005), race (Nguyen et al., 2020; Schell et al., 2020; Williams, 1997), and sex and gender (Davis et al., 2003; Krieger, 2003; Neitzke, 2016) is well-documented.

2.2 Going beyond, but not far enough

If the goal were to understand the drivers of population health and disparities, these structural theories would be sufficient. Yet, population health science, as a field, is defined by a common interest in, not only understanding, but also *improving* population health. In doing so, population health science bridges the impartial academic desire to understand with the interventionist medical drive to improve and emerges as a field distinct from both. Existing structural theories further understanding but leave questions of intervention largely unanswered.

These structural models, while going beyond "all else equal" causality by implicating interconnected social and structural environments, have not called determinism into question. While adherence to determinism remains, structural explanations contribute to a sense that current events are the inevitable result of historical antecedent causes. If determinism remains a fundamental organizing principle, then the mandate of population health science to change and improve outcomes must be discarded.

In other words, existing structural theories have not gone far enough. It is not enough to question "all else equal" causality without also questioning determinism. It is not enough to develop structural models without examining the scientific paradigm they rest on.

2.3 COVID-19: Understanding without action

An analysis of the COVID-19 pandemic guided by these structural models reveals the predictable nature of the poor outcomes in the United States. Consistent with fundamental cause theory, widespread economic inequalities drove social division and excess mortality (Dukhovnov & Barbieri, 2022). Disproportionate impacts among historically marginalized communities drove high mortality rates. Discriminatory social, political, and economic structures reproduced and exacerbated existing disparities, as predicted by ecosocial theory. Social tensions enhanced group identities and resulted in increased social stratification with real impacts on health (Allcott et al., 2020; Cornelson & Miloucheva, 2020).

Yet, understanding how structural forces reproduced and exacerbated disparities did not lead to actionable solutions. If high mortality in African American neighborhoods was caused by existing racism, then the deterministic solution was to eradicate racism. If gender discrimination underlie disparities in employment and income, then the deterministic solution is to eradicate discrimination.

Two problems—one superficial one fundamental—arose. The superficial problem, which activists and policy-makers struggled with throughout the pandemic, is that no proven path for eradicating structural discrimination exists. Attempts to do so, including protests for social justice or pushes to "defund the police" (Wirtshafter, 2021), met reactive backlash. This superficial problem arises from the fundamental problem, which is that under causal determinism, the antecedent causes of structural inequity are rooted in history and not currently modifiable. A commitment to effecting structural change requires a rejection of determinism to adopt an alternate framework of change.

We know that the drivers of population health are interconnected, and isolated interventions will have consequences "all else equal" causality cannot explain. We know that individual behaviors defy deterministic explanations. Above all, we know that current levels of morbidity, mortality, and health disparities are unacceptable and, as most recently illustrated by the COVID-19 pandemic, we know that a scientific paradigm of causal determinism is no longer sufficient. The time for a paradigm shift in population health science is now.

SECTION III: TOWARDS THE AGENTIC—A PARADIGM SHIFT FOR POPULATION HEALTH

3.1 COVID-19: An alternate perspective

The United States' COVID-19 public health strategy was grounded in causal determinism. This, as we have seen, did not work.

An alternate perspective views individuals as "generative, creative, proactive, and reflective, not just reactive" (Bandura, 2001). Individuals within a population are constantly "exploring, manipulating, and influencing the environment" to accomplish goals that give "meaning, direction, and satisfaction" (Bandura, 2001).

From this perspective, the novel coronavirus was a novel obstacle imposed on individuals already in the process of pursuing diverse wants and motivated to find ways to attain their wants and needs, including shelter and nutrition, psychological safety, social connection and intimacy, self-esteem and achievement, and meaning (Maslow, 1943; Seligman, 2018). Threat of infection obstructed the pursuit of individual goals. In this light, policies including lockdowns, social distancing measures, and mask mandates also represented obstacles to the pursuit of individual desires. As the pandemic progressed, the number of obstacles individuals faced and needed to incorporate into decision-making increased.

How individuals adapted to the changing environment varied. For instance, office workers who shifted to working from home were able to continue pursuing professional, economic, and social goals while adhering to COVID-19 restrictions. Essential workers were forced to make trade-offs between their need for health and for employment. Segments of the population with historic mistrust of the medical establishment found that the imposition of requirements conflicted with basic needs for self-esteem and social identity. Among these latter groups, the gauntlet of obstacles created by the health threat of infection, economic threat of unemployment, and social threat of alienation, among others, threatened to overwhelm coping abilities. This resulted in burnout, rejection of public health guidance, political backlash, and a growing need for simplicity, an example of which were the explanations offered by conspiracy theorists (Kay et al., 2009).

This alternate perspective reflects a shift from causal determinism to the agentic paradigm.

3.2 The agentic paradigm

The agentic paradigm structures scientific inquiry around the trajectories of individual lives and decisions. Individual lives are characterized by exposure to unpredictable challenges to basic needs, such as safety, shelter, and intimacy (Maslow, 1943; Seligman, 2018). These challenges, which range from unpleasant to traumatic, nevertheless provide opportunities for adaptation, learning, and growth that represent the substance from which “meaning, direction, and satisfaction” (Bandura, 2001) are derived.

In contrast to “all else equal” causality, the agentic paradigm recognizes that social and structural environments interact with individual characteristics and preferences to produce an interconnected ecosystem. A change in the ecosystem cannot be reduced to a pathway linking an independent exposure and outcome.

The resulting complexity produces ambiguity in individual decision making. At any point and even with full information of antecedent causes, there is no single optimal decision for an individual. Environmental constraints and individual needs interact to produce a menu of distinct but comparable options. In contrast to determinism, agentic action is the causal mechanism by which an individual selects among comparable options to effect change in their environment.

An agentic actor’s path, then, does not resemble a billiard ball set on an environmentally determined route. The exercise of agency is the process by which an individual decides among options to select the one most likely to advance them towards desired outcomes (Figure 2).

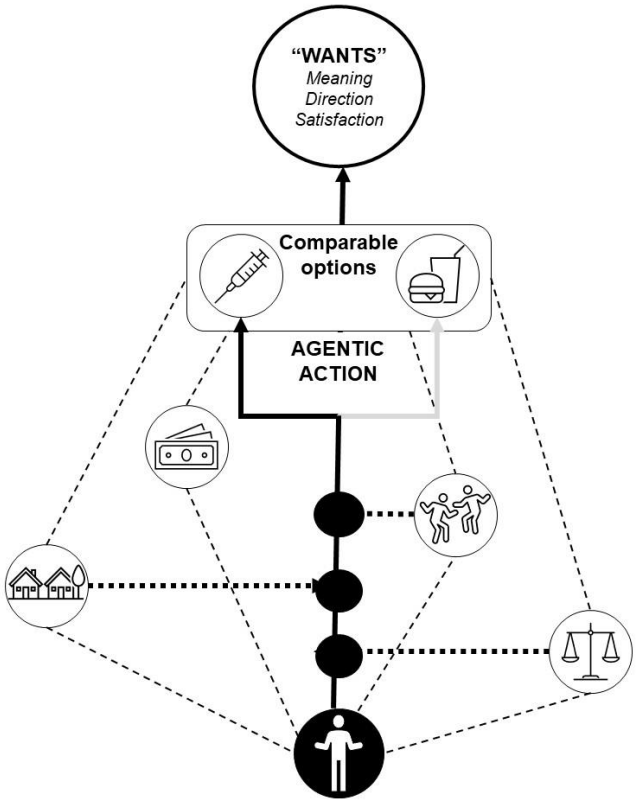

Figure 2: The agentic paradigm. Individual lives are characterized by ambiguous decisions. Social and structural conditions are interconnected and shape obstacles (black circles in the individual's path) and options. The individual, through intentionality, forethought, self-reflection, and self-regulation, exercises agentic action to select one option in the pursuit of meaning, direction, and satisfaction.

Agentic action is not causally determined. Agentic individuals respond to environmental constraints with intentionality, forethought, self-reflection, and self-regulation (Bandura, 2001). In contrast to causal determinism, the agentic paradigm takes agentic choice as a fundamental, irreducible, and non-manipulable component of individual behavior.

Approaching population health from the agentic paradigm results in a structural model that incorporates transdiagnostic concerns and multi-level pathways to produce a set of generalizable principles for both understanding and improving health outcomes.

3.3 A structural model of population health

The shift towards the agentic paradigm reframes the driving question of population health. The biomedical model asks, how do we move individuals uniformly towards "health"?

The agentic paradigm asserts there is not a uniform pathway to health. The driving question becomes: What are the characteristics of the shared environment that maximizes the chances for individuals across the population to attain meaning, direction, and satisfaction in the face of diverse and unpredictable challenges?

We propose a structural model of population health to answer this question. A structural model of health, situated within the agentic paradigm, has the following premises:

1. Individual health is defined as the transdiagnostic capacity to overcome obstacles, or individual resilience.
2. Population health is achieved by maximizing structural resilience.
3. Structural resilience is produced when the relationship between distinct but interconnected entities within a population are congruent.
4. The focus of action and intervention is on the relationships between, rather than within, entities.

3.3.1 Individual health and resilience

An extensive academic literature explores the construct of resilience. Resilience at the individual level is inferred when positive outcomes are observed following an individual's exposure to negative risk or adversity (Rutter, 2013). A trajectory of resilience is characterized by periods of distress, adaptation, and recovery following exposure to adversity (Bonanno, 2005; Kimhi et al., 2021).

Initial interest in resilience was sparked by the observation that a subset of individuals who faced adverse experiences, such as child abuse or homelessness, nevertheless evinced positive outcomes (Doty et al., 2017). Investigations, originally within the field of developmental psychology, focused on characteristics that set these individuals apart and ultimately identified a "short list" of resilience resources, including supporting caregiving, self-efficacy, and intelligence (Masten, 2001).

By organizing scientific inquiry around individual life trajectories, a structural model within the agentic paradigm departs from diagnosis-specific causal determinism to contextualize disease and recovery within a generalizable framework of overcoming life obstacles. The threat of ill health, like the threat of eviction or social rejection, is an unpredictable challenge to which the individual must adapt to pursue meaning and satisfaction. This transdiagnostic adaptive capacity is resilience. Thus, within the agentic paradigm, interest shifts from preventing specific diseases towards promoting *transdiagnostic* capacities for resilience (Badash et al., 2017).

3.3.2 Population health and structural resilience

Over time, evidence has emerged suggesting that resilience is the modal trajectory among individuals following adversity (Bonanno, 2005). Researchers increasingly viewed resilience as a generalized, rather than exceptional, capacity (Masten, 2015).

Social theorists pointed out how framing resilience as an individual-level phenomenon ignored the substantial contributions of social and structural factors to outcomes to risk inadvertently "blaming the victim" for their lack of success (Franzblau & Moore, 2001; Ungar et al., 2013). Supportive caregiving, for instance, is more likely when social policies provide parental leave, healthcare is accessible and affordable, and financial risk is limited. Studies of communities struck by natural disasters or political violence illustrated the role of social capital and collective efficacy as well as robust infrastructure in promoting resilience (Gil-Rivas & Kilmer, 2016; Hemmati et al., 2020; Isa et al., 2018; Siriwardhana & Stewart, 2013; Sousa et al., 2013). The construct of resilience came to be applied, not only at the individual level, but also at the level of neighborhoods and communities.

This work led to defining resilience from a "multi-system perspective" (Masten et al., 2021). Individual resilience results from the interaction of individual traits and behaviors, family and neighborhood support, community characteristics, and structural supports.

The structural model of population health, situated within the agentic paradigm, conceptualizes health as individual resilience. Population health, then, reflects the potential for individual resilience in the population. A multi-system perspective of resilience suggests that individual resilience emerges from the shared systems and institutions that bind a population together. We propose the term *structural resilience* to refer to the extent to which these shared systems and institutions enable individual resilience and, in turn, promote health (Figure 3).

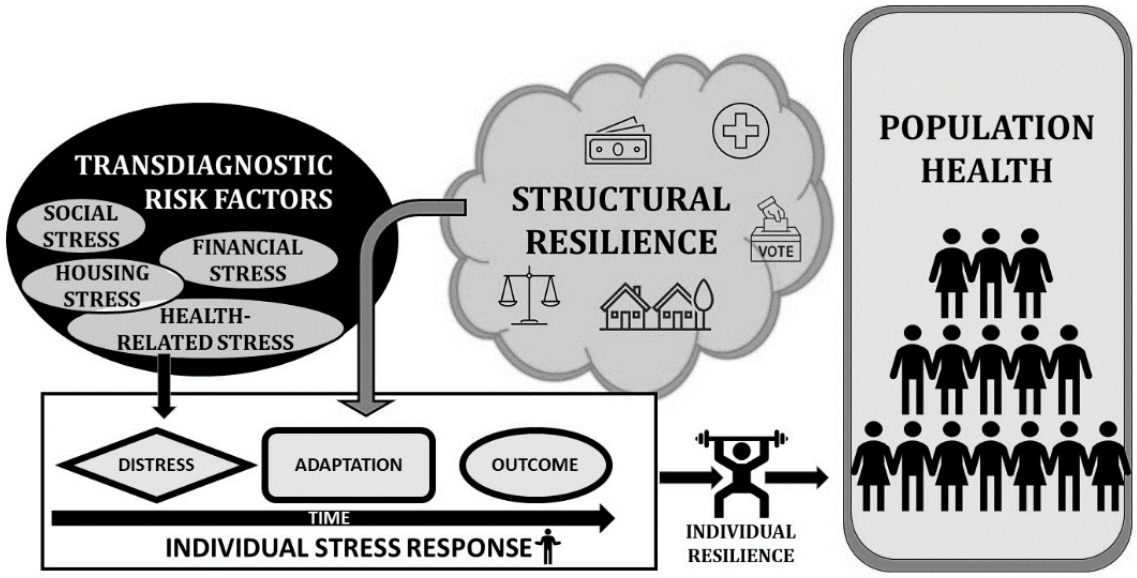

Figure 3: Structural resilience. Individuals face varied and unpredictable obstacles. While individual resilience refers to the transdiagnostic capacity to adapt and overcome, structural resilience is a distinct, group-level construct that captures whether the structural environment

The construct of structural resilience extends current perspectives on resilience. Structural resilience rejects deterministic models of individual resilience. Individual resilience, like human behavior, is not directly manipulable. The ambiguous nature of agentic choice means that successful adaptation cannot be universally operationalized—it may manifest as a college degree for one individual or leaving school for another. It runs counter to the agentic paradigm to label outcomes emerging from diverse obstacles and preferences as "not resilient" if they nevertheless contribute to the agentic pursuit of meaning and satisfaction. However, certain structural environments increase the likelihood an individual can achieve meaning and satisfaction in its varied manifestations. A population's level of structural resilience captures the extent to which the shared structural environment supports and promotes individual resilience in all its members.

Structural resilience is not a proxy for nor an intermediate step in the causal pathway towards individual resilience. It would be a mistake to evaluate structural resilience by investigating aggregate individual outcomes, such as college enrollment or income. Structural resilience exists solely at the population level (Diez Roux, 2004). It is proposed as a distinctly *population health* outcome towards which structural, in contrast to biomedical, interventions must strive.

3.3.3 Structural resilience and congruence

Congruence occurs when distinct but interconnected elements are compatible. At the population-level, structural resilience emerges when concurrent and co-existing policies, institutional structures, and social relationships are characterized by congruence. Congruence at the macro-level is achieved when the systems within a population—for instance, the healthcare, social welfare, employment, and educational systems—are aligned and oriented towards enabling individual resilience. Congruence at the meso-level is achieved when concrete living conditions are compatible with agentic needs and desires. Interpersonal congruence emerges when social relationships are characterized by a mutual commitment to each agentic individual's distinct but interconnected wants and needs. Congruence within a population produces structural resilience which, in turn, enables population health (Figure 4).

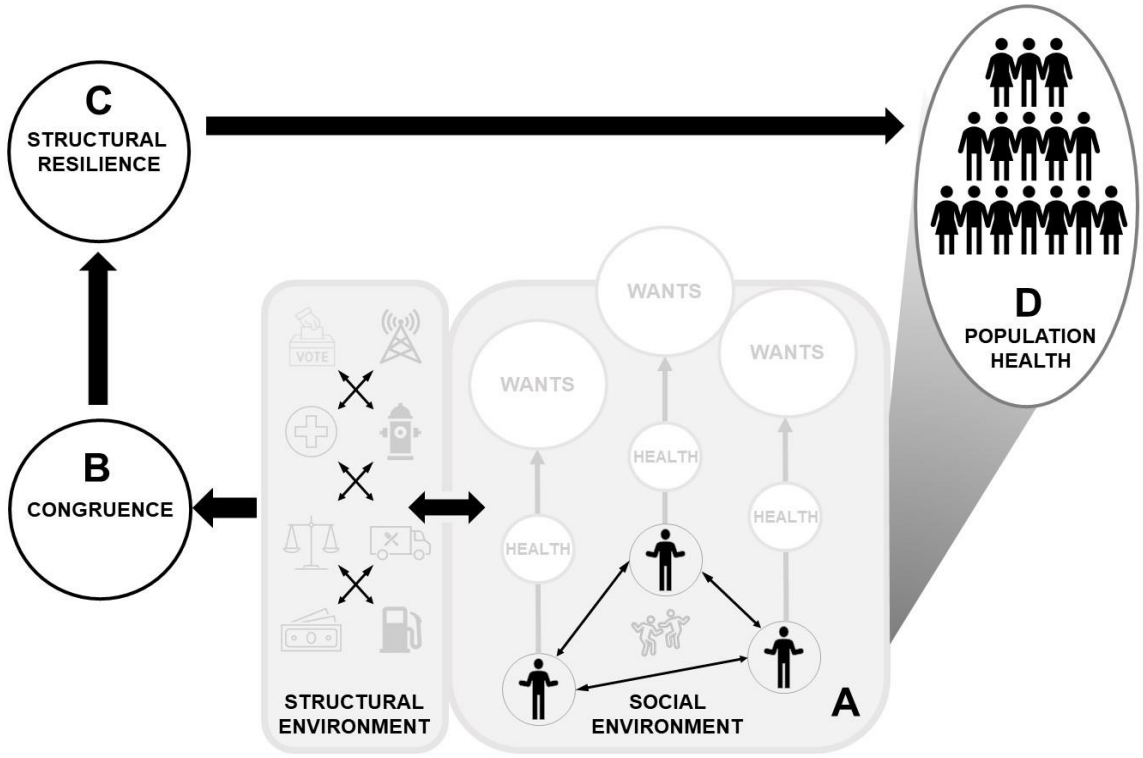

Figure 4: The structural model. The key elements of the structural model are shown in black, while elements that are not the focus are grey.

A population is comprised of agentic individuals (A). Congruence (B) occurs when elements within the population are compatible (bi-directional arrows). Congruence produces structural resilience (C), or an environment that enables rather than obstructs agentic action, which results in population health (D).

A focus on congruence departs from causal determinism. Congruence highlights the connections between elements within a population, rather than the elements themselves, as the exposure of interest. In line with the agentic paradigm, there is no single optimal structural environment for promoting population health. Rather, constraints imposed by current conditions (ex. geopolitical position, historical events, demographic trends) create a menu of options, and the choice comes down to the exercise of agency.

As a result, cultures and communities throughout history have built a diverse array of social and political institutions to meet individual needs. The extent to which a structural environment is health-promoting is related, not to any specific policy or institution, but to the relationship between them. The underlying causes of a health-promoting society are not deterministic, and health is not limited to a single biomedical definition.

3.3.4 The competing dynamics of affiliation and competition

A structural model highlights the nature of the relationships *between* agentic actors.

Such relationships are characterized by competing dynamics of *affiliation* and *competition* (Schutz, 1961). Affiliation reflects the psychosocial desire for belonging and promotes attraction and positive feelings between individuals and groups. Competition is related to the desire for power and drives mistrust. Any relationship *between* individuals can be characterized by relative and competing levels of these two drives.

Interpersonal relationships characterized by affiliation support agentic action. Positive ties allow for the pooling of resources and increase adaptive capacity across obstacles. By contrast, relationships characterized by competition promote one side's goal attainment by obstructing the other's agency.

These two drives explain the formation of communities within a population and subsequent health disparities. Subgroups with high levels of in-group affiliation and out-group competition, coupled with resource access, channel their communal adaptive capacity to influence shared structural conditions to their benefit by implementing policies, building infrastructure, and allocating resources. Such collective action results in improved health for members of the subgroup and creates a subgroup identity.

By contrast, collective action by one subgroup leads to competition for resources with other subgroups on whom worse conditions are subsequently imposed. These imposed conditions result in worsened health as well as competing subgroup identities (Figure 5). These concepts of

in- and out-group dynamics draw from the field of social psychology (Lewin, 1947) and are related to sociological constructs of closure and stratification (Murphy, 1984; Schell et al., 2020).

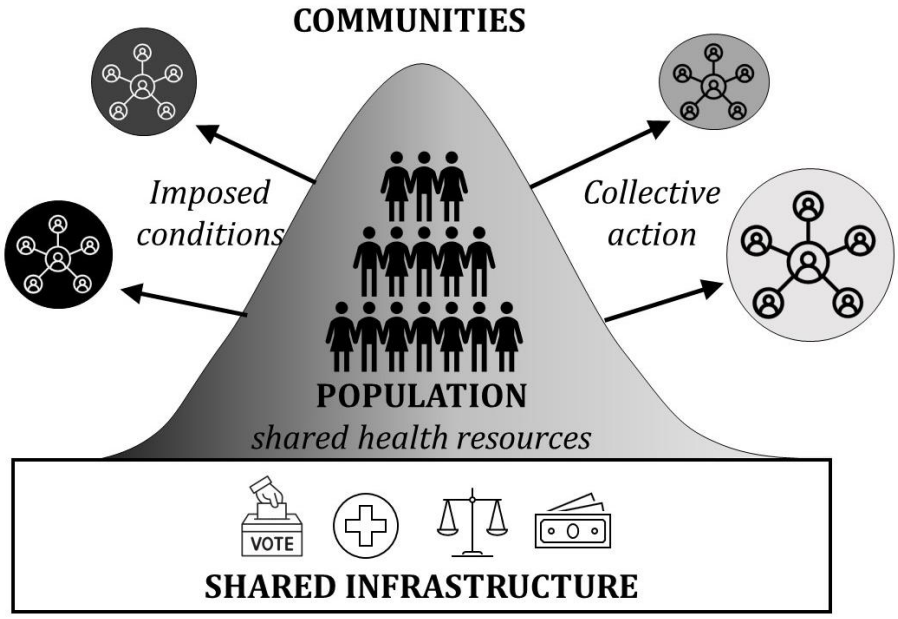

Figure 5. Within a population, affiliation and competition produce distinct communities. Increasing competition between communities increases disparities in resource access, living conditions, and health. In the structural model, increased affiliation is crucial to improved congruence, structural resilience, and population health.

Resulting communities emerge along lines such as race or class but are driven by common and generalizable group-level processes based in competing dynamics of affiliation and competition. In contrast to a focus on race or sex as specific exposures, the structural model widens the lens beyond single causal pathways to describe transdiagnostic processes underlying population health outcomes and disparities.

The agentic paradigm views individual behavior as constrained but not determined by environmental conditions. Social and structural constraints result in a menu of possible options, some of which reinforce affiliative tendencies while others promote competition. It is up to agentic choice to select one or the other. Population health emerges when individuals make choices in favor of affiliation rather than competition within a population.

Identifying affiliation and competition as driving population health both improves understanding and guides intervention. Greater levels of affiliation within a population produce a virtuous cycle of increased adaptive capacity across the population, allowing agentic individuals to work together to develop and implement policies that are congruent across systems and contribute to structural

resilience. The objective of structural interventions is to increase affiliative relationships, rather than to modify or mitigate distinct exposures, to produce population health.

3.4 COVID-19: A case study for the structural model

We return to the COVID-19 pandemic to illustrate each premise of the structural model.

3.4.1 A biomedical approach obstructed individual resilience

During the pandemic, attempts to achieve the biomedical absence of disease conflicted with the agentic transdiagnostic ability to pursue meaning, direction, and satisfaction. Lockdown strategies induced widespread social isolation and loneliness, and even the tightest "pandemic bubbles" were broken for as individuals sought connection. The moral demands of racial equity overshadowed narrow concerns of biomedical "health" as people gathered to protest the death of George Floyd. Parents struggled, feeling as if they were sacrificing their children's social and emotional well-being by subjecting them to online learning, and many became opponents of the public health recommended closures. Lack of attention to individual resilience led individuals to undertake agentic actions to achieve those things that mattered most to them, even if those desires conflicted with public health priorities.

3.4.2 Low levels of structural resilience exacerbated problems

US history is characterized by conflicts based in race, class, gender, and immigration status, among others. This history has engendered deep mistrust between social groups, strengthening distinct social identities that have been further reinforced by structural disparities within core public institutions. High income inequality, housing instability, and food insecurity at the start of the pandemic, alongside rising crime rates during the pandemic, characterized a structural environment that failed to meet basic needs. Structural discrimination, institutional inequity, and political polarization prevented marginalized communities from accessing the social and structural resources needed to protect their health. Despite historic collective action designed to insulate well-off communities, the shared infrastructures of transportation, medical care, and food production meant that the stress of the pandemic spread across the population. Low structural resilience provided little support for agentic individuals in pursuit of their wants and needs.

3.4.3 A lack of congruence hindered policy effectiveness

During the pandemic, policies across systems in America conflicted with each other and with individual needs. To curb viral spread, the healthcare and public health systems recommended

the closure of businesses and schools. As a result, the economic system suffered, leading to a push for reopening that conflicted with the public health demands. The educational system, which pivoted to online learning, conflicted with economic demands for parents to continue working. Eventually, keeping track of and following all the published guidance became impossible. Recommendations contradicted each other, and individuals resorted to picking and choosing their own set of pandemic strategies and making trade-offs between their health and other life domains.

3.4.4 Competition characterized the political environment

Uncertainty and fear during the pandemic heightened emotional responses. Increased stress on shared infrastructure highlighted structural inequity. Individuals came to rely more strongly on group identity; power, control, and competition increasingly dominated interpersonal relationships. Leaders and politicians in America viewed the pandemic opportunistically and used public health communications to increase their political power in the run-up to an election. Political messages made health behaviors central to political belonging. Mask-wearing, vaccination status, and support for pandemic response signaled political affiliation. State and local officials enacted pandemic policies based on their political identity (Adolph et al., 2022). Community leaders highlighted how school closures, lockdown policies, or fiscal stimulus set their constituents apart, contributing to the deepening divide along social identity lines. The more one social group embraced a behavior such as masking, the more opposing groups came to see it as antithetical to their identity (Deane et al., 2021). This competitive social environment obstructed population health.

CONCLUSION

This paper presented a critique of the biomedical model and causal determinism. It described a structural model of health situated within the agentic paradigm and used the COVID-19 pandemic to illustrate key concepts.

Population health science faces a problem, but solutions are both possible and pressing. The global population faces increasing threats from climate change, novel pandemics, and social unrest. Technology and globalization increasingly bind individual lives together. Causal determinism continues to drive innovative and important research, but it cannot resolve the challenges of population health.

The agentic paradigm helps chart the way forward. Treating individuals as intentional and meaning-making agentic actors illuminates directions for intervention ignored by current

approaches. The structural model's focus on structural resilience provides an actionable model for intervention, while evaluating policy congruence, rather than policy impacts "all else equal," better aligns with real-world demands.

Population health scientists today have more data, computing power, and scientific knowledge than ever before. The problem is not one of insufficient resources, but of an insufficient scientific paradigm. This paper takes steps to move beyond the biomedical model and, in doing so, makes progress towards the unifying and ultimate goal of improved population health.

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TITLE: Explaining population psychological distress during COVID-19: A systematic comparison of the biomedical and structural models for population health

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ABSTRACT

Stress is a risk factor for disease. Population health scientists have built upon the documented link to theorize explanations for social gradients of disease. Yet, empirical investigation has produced mixed results at the individual- and group-levels. The resulting "paradoxes" (ex. the Black-White mental health paradox or the Hispanic paradox) remain unresolved.

The structural model of population health highlights group-level processes, in contrast to the individual-level explanations endorsed by the biomedical model. Yet, systematic comparison of the two models for population health is sparse.

The current paper engages in such a comparison. Using data from a nationally representative sample of the US population, we use multi-level spline models to investigate trajectories of psychological distress in the early months of the COVID-19 pandemic. We detail testable hypotheses, perform empirical analysis, and discuss results from the structural and biomedical perspectives. Specifically, we examine whether individual-level differences in exposure to stressful events explain differences in distress, or whether a structural perspective better explains observed trends. We then engage in a meta-discussion to evaluate the two explanations on the grounds of accuracy, consistency, scope, simplicity, and fruitfulness.

This study is the first to suggest support for the structural model of population health by identifying a shared, group-level trajectory of distress during the pandemic. We find that, compared to individual-level biomedical explanations, the structural model better explains population psychological distress during COVID-19. Our results have implications for the design and interpretation of future population health research.

KEYWORDS: *Structural model of population health, biomedical model, population health, theory, COVID-19, psychological distress, stressful events*

INTRODUCTION

Stress and health

Over seven decades of research documents that stress is a risk factor for disease (Cohen et al., 2019; Thoits, 2010). Stress is linked to cardiovascular (Das & O'Keefe, 2006; Steptoe & Kivimaki, 2012; von Kanel, 2012), metabolic (Kuo et al., 2019; Wellen & Hotamisligil, 2005), gastrointestinal (Yaribeygi et al., 2017), and psychiatric disease (Carr, 2014; Corcoran et al., 2003) and accounts for an estimated 30% of myocardial infarction risk (Yusuf et al., 2004).

Stress refers to the subjective state of mental tension or psychological distress that emerges when demands supersede resources. Stress mediates the pathway from stressors, or adverse events, to disease (Pearlin et al., 1981). However, poor health is not a necessary outcome of adversity: Contextual factors impact whether a stressor produces stress, while coping resources influence whether stress produces distress (Wheaton et al., 2013).

Nevertheless, exposure to stressors, including chronic strains, life events, and traumas, explains between 25 and 40 percent of the variance in psychological distress and depressive symptoms (Turner, Wheaton, and Lloyd 1995). Individuals experiencing stressful life events, such as divorce or the death of a loved one, evince increased risks for multiple diseases as well as overall mortality (Cohen et al., 2007).

Population health scientists have followed social stress research with great interest, given its potential for explaining persistent and widespread health inequities by gender, race-ethnicity, and social class (Allen et al., 2014). Disparate exposure to social stressors such as discrimination, neighborhood crime, or poverty is a plausible mechanism that underlies several population health theories.

For instance, allostatic load theory hypothesizes that the cumulative burden of social stress "weathers" individual health resources to cause poor health (Geronimus et al., 2006; McEwen, 1998). Ecosocial theory describes the "embodiment" process by which racism and sexism activate biological stress processes (Krieger, 2001). The Minority Stress Model explains racial-ethnic health disparities via increased stress linked to discrimination and marginalization (Meyer & Frost, 2013).

The empirical paradox

There is abundant individual-level evidence for the social stress hypothesis. Studies document that discrimination, violence, and stigma are associated with depressive symptoms, anxiety

symptoms, and anger (ex. Louie & Upenieks, 2022; Paradies et al., 2015; Valentine & Shipherd, 2018).

These "within-group studies" compare individuals within disadvantaged social groups. The Stress Process Model, applied to the population level, would hypothesize that disadvantaged social groups exposed to higher levels of social stress would exhibit corollary higher levels of psychological distress. This is the "between-group" hypothesis.

Evidence challenges this between-group hypothesis. Despite higher social stress exposure, prevalence of mental disorders is lower in racial/ethnic minority groups compared to White Americans (Alvarez et al., 2019). Hispanic Americans face elevated social risks compared to White Americans but experience similar or better health outcomes (Ruiz et al., 2013). These unexpected findings have been called the "Black-White mental health paradox" (LaMotte et al., 2022), the "Hispanic paradox" (Ruiz et al., 2013), or the "immigrant paradox" (Alvarez et al., 2019).

A review by Schwartz and Meyer (2010) found that support for between-group differences largely rely on within-group analyses, a methodological disconnect that threatens the validity of conclusions. By contrast, between-groups analyses highlight the empirical paradox. In response, current approaches incorporate increasingly complex moderators into the causal pathway, guided by the belief that richer data coupled with more complex models will ultimately resolve the paradox.

A structural solution

We argue instead that this paradox emerges from misguided attempts to generalize individual-level mechanisms to group-level phenomena (Acolin & Fishman, 2023).

Individual-level studies are guided by the biomedical model of health, which focuses on "all else equal" causal relationships between discrete risk factors and distinct diagnoses. Biomedical investigations aim to isolate causal pathways and treat individuals as passive backdrops for biomedical processes or as objects to control for or manipulate.

Calls to move beyond the biomedical model are not new (Schwartz et al., 1999). Such calls rightly point out the mistaken focus on discrete risk factors and encourage a holistic, causal architecture approach to identify "what matters most" within a population (Keyes & Galea, 2015, 2017). We extend these critiques to challenge the biomedical model's reliance on "all else equal" causality.

Beyond its risk factors focus, the biomedical model's reliance on "all else equal" causality is incompatible with population health. Because a population is an interconnected system comprised of individuals bound together by shared institutions (political, educational, legal, etc.) and

infrastructure (transit, healthcare, food services, etc.), causes and effects are interrelated and cannot be studied "all else equal." Individuals within a population are not passive objects but engage in self-reflection, self-regulation, intentionality, and forethought (Bandura, 2001) to react to and shape their environment (Bandura, 2006).

The biomedical model alone cannot resolve the empirical paradox. We propose a structural model that complements the biomedical model to explain population-level outcomes via generalizable, group-level processes (for a more complete discussion, see Acolin & Fishman, 2023). Because group-level processes are different from individual-level mechanisms, group-level outcomes cannot be inferred from individual-level pathways. Conflicting results are not necessarily a paradox but a natural result of this distinction.

Biomedical investigations ask: Why don't group-level outcomes mirror individual-level pathways? A structural investigation asks instead: What group-level processes, beyond individual-level mechanisms, explain group-level outcomes? A structural investigation of stress and health at the population-level may improve upon existing biomedical approaches to help resolve the paradox. Due to its population health importance, systematic comparison of these two approaches is needed.

A framework for systematic comparison

The biomedical and structural models are situated within different scientific paradigms: causal determinism for the former and the agentic paradigm for the latter. Because of this, systematic comparison faces unique challenges.

A paradigm comprises the implicit and foundational assumptions on which hypotheses are built (Kuhn, 1970). The paradigm of causal determinism rests on the implicit assumptions of "all else equal" causality and physical determinism. The agentic paradigm builds notions of causality around individual agency in pursuit of meaning and satisfaction (Acolin & Fishman, 2023) .

The practice of "normal science"—framing research questions, developing hypotheses, collecting data, and interpreting results—takes place within a single scientific paradigm (Kuhn, 1970). Conventional statistical methods use a framework of "rejecting the null" to test competing hypotheses within the same paradigm. An alternate framework is needed to compare competing models within different paradigms.

Kuhn (1977) proposes an alternate framework for model comparison built on five criteria: accuracy, consistency, scope, simplicity, and fruitfulness. An accurate model is supported by

evidence. A consistent model is internally consistent—model predictions do not contradict themselves—and externally consistency with other models. A model with wide-ranging consequences that extend beyond the phenomena it was developed to explain has a broad scope. A model that is simple efficiently organizes complex phenomena, and a model that is fruitful inspires and drives further research.

The problem of stress and health presents a promising opportunity for comparing the biomedical and structural models. The individual-level association between negative events and psychological distress is well-established. The group-level manifestation of both differential exposure to stress and levels of mental health has been documented. This established empirical foundation enables scientific inquiry to turn from fact-finding to the evaluation of competing models.

The current study

The current study asks: Does the structural model improve upon the biomedical model to explain the relationship between stress and health at the population level?

We situate our study within the COVID-19 pandemic. Substantial research documents the impacts of the pandemic on psychological distress (ex. Daly & Robinson, 2021; The Lancet Infectious Diseases, 2020). Yet, the mechanisms driving distress remain unclear.

In this study, we examine the impacts of the COVID-19 public health emergency on individual- and population-level trajectories of psychological distress and compare biomedical versus structural explanations for observed patterns. To do so, we develop a set of research questions and testable hypotheses grounded in each model and will use Kuhn's criteria to systematically compare results and interpretations.

i. The biomedical model

Informed by the Stress Process Model, the biomedical hypothesis is that the pandemic caused individual psychological distress via stressful events.

To test this hypothesis, we examine whether identifiable stressful events are associated with individual psychological distress, "all else equal." We use validated measures of individual psychological distress (Kroenke et al., 2009; Löwe et al., 2010; Materu et al., 2020). However, little consensus exists on a precise definition of a "stressful event" (Cohen et al., 2019). Experts have proposed several criteria, including the event's level of threat, demands on resources, or interruption of goals. Study contexts shape operational definitions (Cohen et al., 2019; Masten et

al., 2021), and commonly studied events (ex. death of a spouse, sexual assault, or a diagnosis of imminent death) are discrete in time, relatively self-contained, straightforward to measure, and clearly linked to distress.

We follow current practice and identify three types of individual-level stressful events, as distinct from shared population-level pandemic mitigation measures such as widespread lockdowns, that arose in the pandemic context. The first is illness. President Trump declared a nationwide public health emergency on March 13, 2020. By April 10, over 18,000 confirmed deaths and over 500,000 cases had been reported in the US (CDC, 2022).

The second is school closure. In the week following President Trump's declaration, most states closed public schools. By early April, all states but Rhode Island closed K-12 public schools (Raifman et al., 2021).

The third is unemployment. Also in April, 40 states issued stay-at-home/shelter-in-place orders, and all states except South Dakota closed non-essential businesses (Camera, 2020; Raifman et al., 2021). By the end of the month, unemployment surpassed Great Depression levels to reach 14.7% (Bureau of Labor Statistics, 2020).

Not all stressors are discrete events. For instance, poverty or loneliness are neither discrete nor self-contained experiences but are also linked to distress (Berry et al., 2016; McEwen & McEwen, 2017). Such chronic strains "persist continuously for a long time" (Cohen et al., 2019) and, like stressful events, are linked to adverse outcomes. Unlike stressful events, their health impacts accumulate as a function of time exposed. While chronic strains face their own measurement challenges, their inclusion significantly increases explanatory power (Turner et al., 1995).

We address the potential confounding effect of pandemic-related chronic strains, such as loneliness or uncertainty, on the relationship between individual-level stressful events and distress in two ways. First, we limit the study period to six weeks following emergency declaration to minimize the time for chronic strains to develop. Because chronic strains may nevertheless have impacted distress during this period due to the unprecedented scope and magnitude of the pandemic, we additionally examine whether distress increased as a function of time exposed.

We also control for external influences, or confounders, that are associated with both the exposure and outcome.

Exposure to the COVID-19 pandemic occurred largely independent of individual characteristics. The novel coronavirus pandemic did "fall randomly from the sky" to impact all members of the

population (Cohen et al., 2019). At the population-level, no association between individual characteristics and exposure to the pandemic exists.

Yet, demographic characteristics may nevertheless confound the relationship between individual experience of stressful events and psychological distress (Kowal et al., 2020). For instance, compared to non-parents, parents of young children have lower levels of life satisfaction (Stanca, 2012) and were more likely to experience stress related to school closures. Women were more likely to increase time spent on caregiving and decrease employment (Madgavkar et al., 2020). College-educated workers were less likely to face layoff and, on average, have better mental health (Daly et al., 2020). Essential workers faced higher exposure risk due to in-person employment and were disproportionately non-White (Dubay et al., 2020). Young adults experienced greater threats to mental health while older adults faced greater health risks (Vahratian et al., 2021). Consequently, we control for gender, age, education, and race.

We address remaining concerns about residual confounding by testing multiple hypotheses informed by additional causal criteria (Koepsell & Weiss, 2014). Our aim is to isolate the relationship between exposure to stressful life events and individual psychological distress. Thus, we investigate the existence of a dose-response relationship, specificity in the association, and temporality to strengthen the case for causality (Shimonovich et al., 2021).

We test the following biomedical hypotheses:

> H_0: Increases in individual psychological distress did not differ by exposure to stressful events.
>
> H_a: Increases in individual psychological distress will vary by exposure to stressful events in the following ways:
>
> > H_{a1} (Dose response): Exposure to more pandemic-related stressful events will be associated with greater increases in psychological distress.
> >
> > H_{a2} (Specificity): Different types of pandemic-related stressful events will be associated with differences in the increase of psychological distress.
> >
> > H_{a3} (Temporality): Increases in psychological distress will be greatest immediately following exposure to pandemic-related stressful events.

H_{a4} (Chronic strains): Controlling for exposure to discrete stressful events, average levels of psychological distress in the population will increase as a function of time.

ii. The structural model

The structural model hypothesizes that, in response to the population-level pandemic exposure, a group-level outcome of population distress exists over and above individual-level sociodemographic characteristics or experience of stressful events. The shared structural environment is hypothesized to shape responses to the shared adverse shock to produce a common outcome.

We examine the population-level trajectory of distress over time. Because a population is a dynamic system, outcomes are not static but rather emerge from dynamic processes producing homeostatic equilibrium. A shock to the system catalyzes an immediate reaction followed by adaptation and recovery. Consequently, unlike "all else equal" investigations where temporality has two dimensions (antecedent cause and resulting effect), temporality in the structural model has three dimensions—beginning, middle, and end—characterizing a trajectory of shock and recovery.

The COVID-19 pandemic provides a unique opportunity to test the structural hypothesis. While the group-level trajectory is hypothesized to be ubiquitously present, in most cases, the timing, type, and severity of individual stressors make discerning a shared trajectory difficult. The pandemic, however, was a population-wide shock that occurred at the same time across the nation. While individual experiences of the pandemic varied, exposure to the pandemic was shared.

The current study thus tests the following structural hypotheses:

H_0: There is no group-level trajectory of psychological distress. Variation in trajectories of psychological distress are explained by differences in the individual experience of the pandemic.

H_a: There is a group-level trajectory over and above individual-level mechanisms.

METHODS

Data

We used data from the Understanding Coronavirus in America Study (UCAS) (Kapteyn et al., 2021), a publicly available tracking survey that measured the impacts of the COVID-19 pandemic via bi-weekly surveys administered to participants of its parent study, the Understanding America Study (UAS).

UAS is an ongoing longitudinal panel (2014-present) with over 8,000 current members. Panel members were recruited using random address-based sampling of American adults over age 18 using the US Postal Service Computerized Delivery Sequence file, which covers nearly 100% of US households. Twenty recruitment batches occurred between 2014-2020. Across batches, between a quarter and a third consented to participate, and between 5-10% remain active with higher attrition among those recruited prior to 2018. Surveys are completed electronically, and participants receive monetary incentives for each survey completed. If needed, participants are provided with a tablet and/or internet access.

UAS surveys were fielded monthly. Starting in March 2020, COVID-19 surveys (UCAS) were fielded bi-weekly. Participants had two weeks to complete each survey. Response rates are high; >90% of participants completed most COVID-19 surveys.

Participants

There were 7,411 unique participants with data collected between March 10 and May 25, 2020. Of these, 502 were missing pre-pandemic mental health measures and 10 had missing demographic information, leading to a study sample of 6,909 participants and 58,820 observations (average 8.5 observations/participant; over 90% of participants had 6+ observations).

Measures

Time

Our study period begins March 10, 2020, when UCAS surveys began, and ends May 25, 2020, the date of George Floyd's death.

During this period, the novel virus was of central and primary importance. Politicization of health behaviors, such as masks and vaccinations, had not yet taken root; concerns about social inequity remained secondary to public health concerns; and the political spectacle of the upcoming

presidential election were not yet center stage. On May 25, George Floyd, an African-American man in Minneapolis, died by police violence. Floyd's death and subsequent protests created an additional source of social stress (Buchanan et al., 2020). To reduce confounding by social and political concerns, we end our study period on this date.

We treat March 13, the date that President Trump declared a national public health emergency, as the reference date (time = 0) and code time in days, using positive numbers for days following and negative numbers for days preceding. To capture potentially non-linear trajectories of distress, we draw on published timelines of events (CDC, 2022; Raifman et al., 2021) to divide this time into 5 discrete time segments: Period 0 – pre-March 13 declaration; Period 1 (14 days) – March 13-27, school closures and CARES Act passed; Period 2 (14 days) — March 28-April 10, rise in cases in most states; Period 3 (14 days) – April 11-April 24, unemployment reaches 14.7%, White House announces plans for re-opening the economy; and Period 4 (30 days) – April 25-May 25, businesses re-open, and mask mandates replace stay-at-home orders.

Psychological distress

We measure psychological distress using the four-item Patient Health Questionnaire (PHQ-4). The PHQ-4 is a brief self-report questionnaire validated for measuring depression and anxiety in the general population. The questionnaire's criterion validity has been established (Kroenke et al., 2009; Löwe et al., 2010), and baseline measures in the study sample exhibit high internal consistency (Cronbach's alpha = 0.89).

The PHQ-4 asks participants how often they have been bothered by specific problems in the last two weeks (response options: Not at all, Several days, More than half the days, Nearly every day). It comprises a two-item depression scale (Item 1: Little interest or pleasure in doing things; Item 2: Feeling down, depressed, or hopeless) and a two-item anxiety scale (Item 1: Feeling nervous, anxious, or on edge; Item 2: Not being able to stop or control worrying). Possible scores range from 0 (no mental distress) to 12 (severe mental distress). We used the summed score.

Stressful event: COVID-related Illness

Exposure to illness was defined as having been personally diagnosed with or tested positive for COVID-19, or having had any close friends or family infected, hospitalized, or died from the virus, during the study period. This measure was derived from questions asked regularly as part of the core COVID-19 section of the UCAS.

The first question asks whether the individual has been tested for the coronavirus since the last survey and their result (response options: Tested positive, Tested negative, Tested but unknown

result, Not been tested). The second asks whether, regardless of testing, the individual has been diagnosed by a doctor or other healthcare professional as having or probably having the coronavirus (response options: Yes, No, Unsure). Responses of “Tested positive” to the first question or “Yes” to the second question were coded as having experienced COVID-related illness.

A second set of questions asked how many close friends and family have been infected, hospitalized, or died from the coronavirus. Non-zero responses were coded as having experienced COVID-related illness.

Responses were combined into a single binary variable capturing whether the participant experienced an illness-related stressful event (0: No, 1: Yes).

Stressful event: School closures

School closure event was defined as reporting any cancellation or suspension of school because of the coronavirus for those with school-age children. School closure was measured at Wave 3 of the survey (fielded April 15-May 12, 2020). Respondents were first asked how many members of their household are currently enrolled in preschool or daycare, primary school, middle school, or high school (response options: 0-10 for each category), and how many among them had school suspended or cancelled because of the coronavirus (response options: 0-number reported previously).

Responses were combined into a single binary variable (0: no school closure impact, 1: any school closure impact).

Stressful event: Layoff

Layoff was derived from labor status ascertained in each survey wave. This measure used responses from a question in the core labor section of the UCAS (response options: 1 – currently working, 2 – sick/other leave, 3 – unemployed-temp layoff, 4 – unemployed-looking, 5 –retired, and so not in labor force, 6 – not in labor force & not retired, 7 –unknown labor force status). Individuals whose status changed during the study period from "currently working" in one survey to “layoff,” “looking,” or “not in labor force and not retired” in the subsequent survey were coded as being laid off during the pandemic.

Stressful event operationalization: Number of events

To calculate the number of reported events during the study period, we summed the binary variables for illness, school closure, and layoff. We then created a categorical variable with levels 0: no event, 1: one event, and 2: 2+ events.

Stressful event operationalization: Type of event

To explore the impact of event-level characteristics, we excluded individuals reporting 2 or more stressful events. We coded the type of impact reported (0: no impact, 1: illness, 2: layoff, 3: school closure).

Stressful event operationalization: Timing of event

While the UCAS survey included questions about stressful events, precise dates were not included. We addressed this issue in two ways, using the subset of participants reporting 0 or 1 pandemic-related events.

First, since stressful events were related to widespread policy changes and mandates, we believed that type of event would be correlated with timing. We expected that exposure to school closure would primarily occur in late March during period 1, layoffs would occur through the month of April during periods 2 and 3, and illness would be most prominent in later periods. We tested this assumption by calculating estimated event date as the midpoint between the survey at which the event was first reported and the prior survey. We examined the correlation between event type and time and examined whether higher increases in distress corresponded to the event period.

Second, after excluding individuals reporting 2 or more stressful events, we created a time-varying variable centered on the date in which the event was first reported (time=0). For those reporting no events, we used the emergency declaration date as time 0. Days following this date were coded as positive, and days prior were coded as negative.

Additional measures

Pre-pandemic mental health

Because it is difficult to adequately produce a global measure of mental health, given its sensitivity to life events and variable presentation (Bagge et al., 2013; Thompson et al., 2021), we addressed measured pre-pandemic mental health in two ways.

First, all models included pre-pandemic depression symptoms as a control variable, derived from responses to the Composite International Diagnostic Interview (CIDI) module from previous UAS surveys (Angrisani et al., 2019). The CIDI module asks whether, in the past year, the participant had felt "sad, blue, or depressed" for at least two weeks (response options: Yes or No) and, if so, about the duration and intensity of the symptoms. Responses were combined into a single categorical variable with three levels: no depression symptoms in the previous year were coded as 0 (no depression); symptoms that lasted less than half the day most days were coded as 1 (mild depression); symptoms that lasted most of the day almost every day were coded as 2 (moderate/severe depression). We used pre-pandemic depression symptoms collected between 2015 and 2020.

Second, we modeled PHQ-4 scores over time and included, when available, PHQ-4 scores prior to March 13. The first UCAS wave was administered on March 10, meaning we have up to 3 days of pre-declaration psychological distress scores for those participants who responded during this time window. While this measure is limited, using the same dataset, Daly and Robinson (2021) found evidence that the scores reported in the UCAS from March 10-18, 2020 did not significantly differ from distress levels measured in surveys fielded in March 2017 and 2018 in comparable nationally representative samples, indicating that these early scores are reasonable estimates of pre-pandemic distress.

Education

Education was coded as a categorical variable with 4 levels (0: less than high school, 1: some college, 2: bachelor's degree, 3: graduate degree).

Age

Age was coded as a categorical variable with 3 levels (0: young adult, 26 years or younger, 1: 27-64 years old, 2: older adult, 65 years or older).

Gender

Gender was measured in the base demographic questionnaire which asked, "What is your gender?" with response options Male or Female (non-response rate: 0.02%). Gender was coded as a categorical variable with 2 levels (0: Female, 1: Male).

Race/Ethnicity

Race/ethnicity was ascertained via multiple questions where participants had the option to report identifying as White, Hispanic, African-American/Black, Asian, Pacific Islander, and American Indian.

A range of potential pathways connect race/ethnicity to pandemic-specific stressors, including structural discrimination driving income disparities, barriers to healthcare access, and increases in hate crimes. Given that we lacked sufficient data to meaningfully distinguish these pathways beyond self-reported race/ethnicity, we chose to collapse race/ethnicity responses into a single binary variable (0: White only, 1: Non-White/BIPOC).

Analytic approach

We used multilevel spline models with a random intercept to examine within-person changes in psychological distress over time. Piecewise linear splines were used to accommodate the non-linear trajectory over time. Estimated slope coefficients correspond to daily rate of change in each time segment. Negative binomial forms of the model accounted for positive skewness and over-dispersion in the psychological distress outcome. Our models used all available data, such that missing responses were dropped but participants with partial data remained in the analysis.

We follow conventions with count regression models and estimate count ratios (exponentiated coefficients) that describe the proportional change in the outcome (PHQ-4 score) associated with a 1-unit increase in the predictor (time in days), conditional on the random intercept. Figures plot the marginal estimated effect holding other variables constant at their mean value.

We first ran the spline model, with and without control variables, in the full sample with knots at 0, 14, 28, and 42 days consistent with our pre-defined periods. Our second model tested H_{a1} by including an interaction between time and number of reported events. In our third model, we excluded individuals reporting 2 or more stressful events and included an interaction between time and type of event to test H_{a2}.

Our fourth model tested H_{a3} and H_{a4} by interacting the type of event with time centered at event report date and included knots at 0, 14, and 28 days. To aid in interpreting results, we simulated data to create a reference group with a constant PHQ-4 score of 1 to represent the null hypothesis (H_0: No change in levels of distress over time). Interaction terms reflect the extent to which trajectories by event type depart from the null.

All statistical analysis were performed using R 4.0.2 (R Core Team, 2020) using the "glmmTMB" package for multi-level modeling (Brookes et al., 2017), the "lspline" package for linear piecewise splines (Bojanowski, 2022), the "ggeffects" package for marginal effects (Lüdecke, 2018), and "ggplot2" for plotting model results (Kahle, 2013).

RESULTS

Descriptive statistics

In total, 6,909 participants and 58,820 observations were included in the analysis. Of these, nearly half reported no pandemic-specific stressful event (45.5%), 40.7% reported one, and 13.9% reported two or more. On average, those who reported no pandemic-specific event were older, male, White, highly educated, and with no pre-existing depression. Demographic characteristics significantly differed by number of stressful events ($p<0.001$) (Table 1).

Figure 1 plots the average, unadjusted trajectory of psychological distress using a Loess smoother. On average, psychological distress increased until late March, decreased slightly for two weeks, and then dropped more steeply but with higher volatility for the remainder of the study period. The proposed time periods appear to capture distinct phases in the distress trajectory.

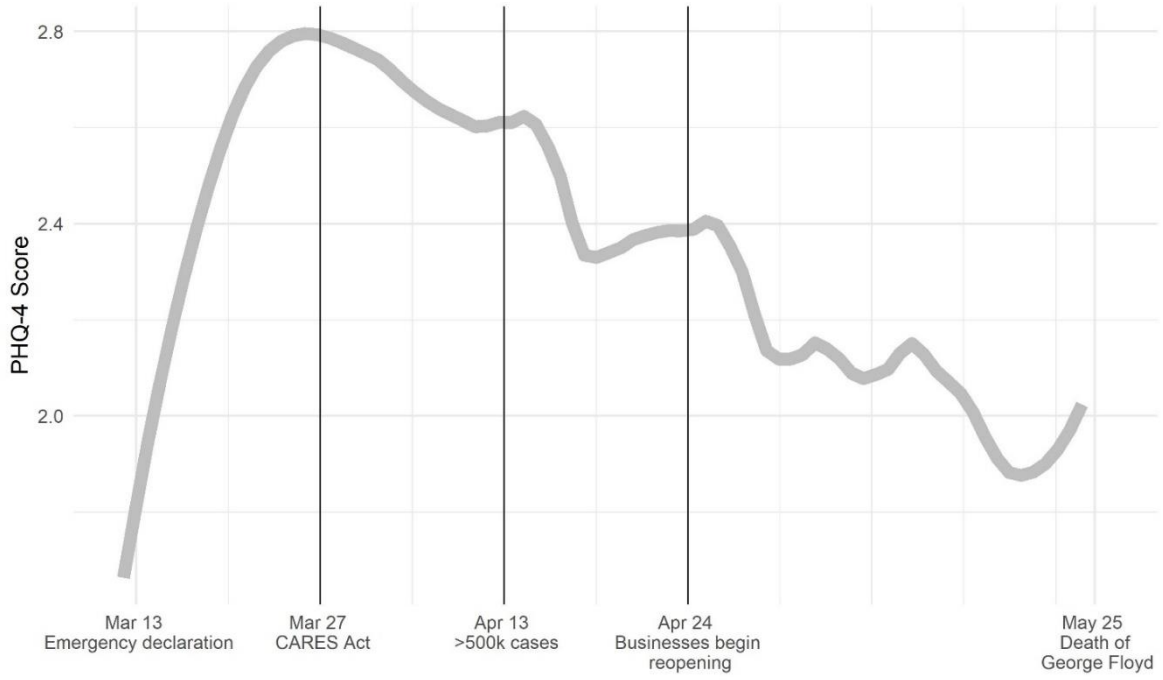

Figure 1. Loess smoothed average of population psychological distress

	Total N = 6909[a]	No events n = 3137 (45.4%)	1 event n = 2813 (40.7%)	2+ events n = 959 (13.9%)	Chi-square test
Age (Mean [SD])	**53.0 (16.1)**	56.2 (16.8)	50.5 (15.4)	44.6 (12.4)	
Male (%)	**41.8**	45.4	38.8	34.0	
BIPOC (%)[b]	**33.0**	30.7	35.5	46.2	
Hispanic/Latino	**15.6**	13.6	17.3	25.3	
Black	**9.6**	8.2	10.7	13.7	
Asian	**6.4**	6.9	6.4	5.9	P<0.001
Pacific Islander	**1.7**	1.5	1.9	2.6	
Native American	**5.0**	5.5	4.5	7.7	
Education (%)					
Less than HS	**5.0**	5.1	5.2	4.6	P<0.001
HS/GED/Some college	**53.0**	53.8	51.9	53.7	
Bachelor's degree	**24.7**	23.5	24.9	27.4	
Graduate degree	**17.3**	17.7	17.9	14.3	
Pre-COVID depression (%)					
No depression	**81.1**	82.5	80.4	79.2	P<0.001
Mild depression	**7.1**	6.4	7.4	7.9	
Moderate/Severe depression	**11.9**	11.2	12.2	12.9	

Table 1. Sample characteristics

[a] The sample size presented in the header is the denominator for all statistics reported in the column. Thus, among those reporting no impacts, 13.6%, or 427 individuals, reported Hispanic/Latino ethnicity.

[b] Each race/ethnicity category was asked separately, and participants had the option to answer yes to multiple categories. In composite variables, these individuals would be grouped together as "mixed race." To avoid presenting misleading numbers via aggregation, we present numbers for each category separately.

Model 1: Population-average trajectory of psychological distress

Consistent with Daly and Robinson (2021), we find that individuals experienced an increase in psychological distress in the days following the emergency declaration followed by a decrease beginning two weeks later (Table 2). Each day leading up to the emergency was associated with a 5% increase in PHQ4 scores. Each day in the two weeks following the emergency declaration

was associated with a 3% increase in PHQ4 scores. A reverse trend was observed in the following weeks ($p<0.001$ for all estimates).

Controlling for covariates did not change slope estimates, as would be expected when including non-time varying characteristics in a within-person longitudinal model. However, distress varied by participant characteristics. Most notably, moderate or severe levels of pre-pandemic depression were associated with nearly 3.5 times higher levels of psychological distress.

	Unadjusted	**Adjusted**	
	Exponentiated coefficients	Exponentiated coefficients	Exponentiated 95% CI
Intercept	0.92**	1.04	0.93, 1.15
Period 0 (pre-Mar 13)	1.05***	1.05***	1.02, 1.08
Period 1 (Mar 13-27)	1.03***	1.03***	1.03, 1.04
Period 2 (Mar 28-Apr 10)	0.99***	0.99***	0.99, 1.00
Period 3 (Apr 11-Apr 24)	0.99***	0.99***	0.99, 0.99
Period 4 (Apr 25-May 25)	0.99***	0.99***	0.99, 0.99
Edu (less than HS)		1.10	0.93, 1.30
Edu (some college)		0.89*	0.81, 0.98
Edu (BA)		1.10	0.99, 1.23
BIPOC		0.97	0.90, 1.04
Male		0.65**	0.61, 0.70
Age (Young adult)		1.37***	1.17, 1.59
Age (65+)		0.66***	0.61, 0.71
Pre-pandemic depression (mild)		2.38***	2.10, 2.70
Pre-pandemic depression (moderate/severe)		3.43***	3.10, 3.79

Table 2. Regression results, population average trajectory with and without controlling for covariates

* $p < .05$, ** $p < .01$, and *** $p < .001$

Model 2: Time*Number of stressful events

Model 2 results for the distress trajectory are presented in Table 3 (control variables estimates were comparable to Model 1). Figure 2 illustrates estimated marginal trajectories.

Among individuals reporting no events, PHQ-4 scores rose on average 3% per day in period 1 and decreased 1% per day after April 11. The estimated slopes for individuals reporting 1 or 2+

events do not significantly differ from those reporting no impacts (p-value ranges from 0.11 to 0.76). The only statistically significant difference in slope, emerges in the days prior to the emergency declaration, where those reporting 2+ events experienced a 12% increase in psychological distress with each day.

	No events	1 stressful events		2 stressful events	
	Exponentiated coefficient (β_1) (95%CI)	Exponentiated coefficient (β_2) (95%CI)	Estimated slope ($\beta_1 * \beta_2$)	Exponentiated coefficient (β_3) (95%CI)	Estimated slope ($\beta_1 * \beta_3$)
Period 0 (pre-Mar 13)	1.02 (0.98, 1.06)	1.03 (0.97, 1.09)	1.05	1.10** (1.02, 1.18)	1.12
Period 1 (Mar 13-27)	1.03*** (1.02, 1.04)	1.00 (0.99, 1.02)	1.04	0.99 (0.97, 1.00)	1.02
Period 2 (Mar 28-Apr 10)	0.99 (0.99, 1.00)	0.99 (0.98, 1.00)	0.99	1.00 (0.99, 1.02)	1.00
Period 3 (Apr 11-Apr 24)	0.99*** (0.99, 0.99)	1.01 (1.00. 1.01)	0.99	1.00 (0.99, 1.01)	0.99
Period 4 (Apr 25-May 25)	0.99** (0.99, 1.00)	1.00 (1.00, 1.00)	0.99	1.00 (0.99, 1.01)	1.00
Number of events		1.23*** (1.10, 1.38)		1.70*** (1.46, 1.97)	

Table 3. Model 2, estimated interaction coefficients and confidence intervals.

* p < .05, ** p < .01, and *** p < .001

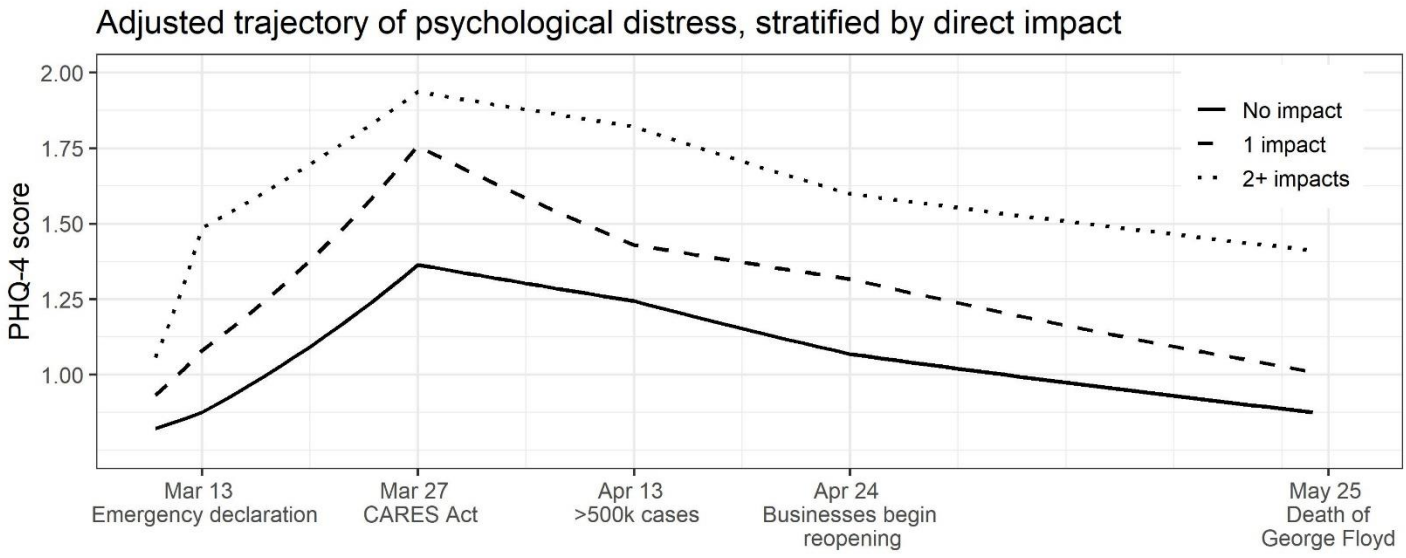

Figure 2. Adjusted trajectory of psychological distress, with number of events included as an effect modifier.

Model 3: Time*Type of stressful event

Table 4 reports sample characteristics of participants reporting only 0 or 1 stressful events (N=5,950) as well as estimated event timing. Participants reporting school closure were younger and more likely to be female, of minority race/ethnicity and with lower levels of education. Those reporting layoff were more likely to be Black and/or Hispanic males and less likely to have graduate degrees. Those reporting COVID illness events were of older age and more likely to have a bachelor's degree compared to other event types.

Table 5 reports Model 3 results. We again observe similar trajectory of increasing distress followed by gradual decrease across event types. Figure 3 illustrates the estimated marginal levels of psychological distress by group.

Descriptive statistics suggest that estimated event dates were highly correlated with period (Table 4). Most school closures happened prior to April 1, while most layoffs and illnesses occurred after March 26.

We examined whether increases in distress were greater during the period associated with the event compared to those experiencing no events. We do not find differences between school closure (period 1), while layoff and illness were associated with slightly lower slopes (period 2).

This difference is most pronounced among those experiencing layoff, where the interaction term approaches statistical significance (p-value = 0.06).

	No events n = 3137	Illness N=1312	Layoff N=294	School closure N=1207	X^2 test
Age (Mean [SD])	56.2 (16.8)	55.0 (16.3)	51.2 (15.7)	44.9 (12.0)	
Male (%)	45.4	38.3	45.2	37.8	
BIPOC (%)	30.7	30.2	36.9	40.9	
Hispanic/Latino	13.6	12.5	17.0	22.7	
Black	8.2	10.0	14.0	10.7	
Asian	6.9	6.1	7.3	6.4	P<0.001
Pacific Islander	1.5	1.7	1.8	2.2	
Native American	5.5	4.4	3.9	4.8	
Education (%)					
Less than HS	5.1	4.3	4.3	6.4	P<0.001
HS/GED/Some college	53.8	47.1	58.6	55.6	
Bachelor's degree	23.5	27.2	30.0	21.1	
Graduate degree	17.7	21.4	7.0	16.9	
Pre-COVID depression (%)					
No depression	82.5	80.1	79.5	81.0	P<0.001
Mild depression	6.4	7.9	4.8	7.5	
Moderate/Severe depression	11.2	12.0	15.7	11.4	
Estimated event date					
1st quartile		March 27	March 26	March 25	
Median		March 31	April 9	March 27	
3rd quartile		April 16	April 24	April 1	
Event reported by date					
3rd quartile		April 29	April 27	April 16	

Table 4 [a] . Sample characteristics of participants reporting only one stressful event.

[a] The notes on sample size and race/ethnicity detailed for Table 1 apply equally to this Table. Specifically, the sample size presented in the header is the denominator for all statistics reported in the column, and numbers for race/ethnicity do not sum to the percent BIPOC due to those reporting more than one category.

	None	Illness		Layoff		School closure	
	Exp(β_1) (95%CI)	Exp(β_2) (95%CI)	Exp ($\beta_1 * \beta_2$)	Exp (β_3) (95%CI)	Exp ($\beta_1 * \beta_3$)	Exp(β_4) (95%CI)	Exp ($\beta_1 * \beta_4$)
Period 0 (pre-Mar 13)	1.02 (0.98, 1.06)	1.05 (0.98, 1.12)	1.07	0.94 (0.83, 1.06)	0.95	1.03 (0.96, 1.11)	1.05
Period 1 (Mar 13-27)	1.03*** (1.02, 1.04)	1.00 (0.99, 1.02)	1.03	1.02 (0.99, 1.05)	1.05	1.00 (0.99, 1.02)	1.04
Period 2 (Mar28-Apr10)	0.99 (0.99, 1.00)	0.99 (0.98, 1.01)	0.98	0.99 (0.96, 1.01)	0.98	1.00 (0.98, 1.01)	0.99
Period 3 (Apr11-Apr24)	0.99*** (0.99, 0.99)	1.01 (1.00, 1.01)	0.99	1.01 (1.00, 1.02)	1.00	1.00 (1.00, 1.01)	0.99
Period 4 (Apr25-May 25)	0.99*** (0.99, 1.00)	1.00 (1.00, 1.00)	0.99	1.00 (0.99, 1.00)	0.99	1.00 (0.99, 1.00)	0.99
Type of event		1.40*** (1.22, 1.61)		1.13 (0.88, 1.44)		1.08 (0.93, 1.26)	

Table 5. Model 3, Type of stressful event impact.

* p < .05, ** p < .01, and *** p < .001

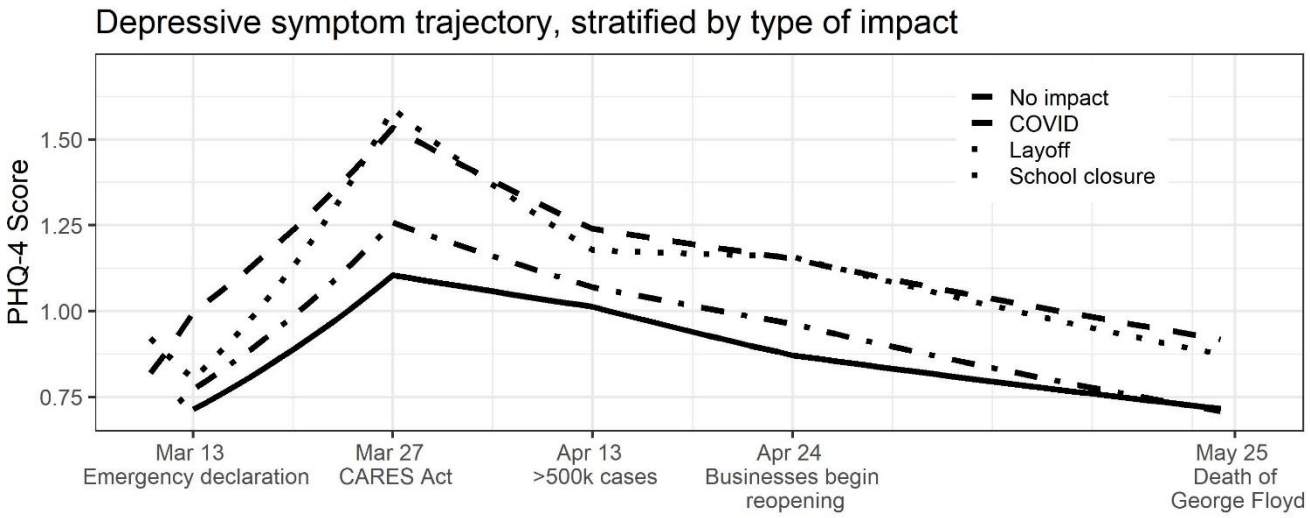

Figure 3. Adjusted trajectory of psychological distress, with type of impact included as effect modifier.

Model 4: Event time*Type of stressful event

Table 6 reports Model 4 results, and Figure 4 illustrates estimated marginal levels of distress. For those reporting one stressful event, each day leading up to the reported event was associated with 1% increase in distress. Subsequent trajectories differ, with school closure associated with a rebound in increasing distress after 28 days and layoff associated with a slower decrease in the first two weeks.

	Simulated reference group	No event	Illness	Layoff	School closure
	Exp(β_1) (95%CI)	Exp(β_2) (95%CI)	Exp (β_3) (95%CI)	Exp(β_4) (95%CI)	Exp(β5) (95%CI)
Period 0 (before event)	1.00 (0.99, 1.01)	1.02 (0.98, 1.05)	1.01* (1.00, 1.01)	1.01** (1.00, 1.02)	1.01*** (1.00, 1.01)
Period 1 (Days 0-14)	1.00 (0.97, 1.04)	1.03*** (1.02, 1.04)	1.00 (0.97, 1.02)	1.00 (0.97, 1.03)	0.99 (0.96, 1.02)
Period 2 (Days 14-28)	1.00 (0.96, 1.04)	0.99 (0.98, 1.00)	1.00 (0.97, 1.03)	0.98 (0.95, 1.02)	0.98 (0.95, 1.02)
Period 3 (Days 28+)	1.00 (0.99, 1.01)	0.99*** (0.99, 0.99)	1.00 (0.99, 1.01)	1.00 (0.99, 1.01)	1.02* (1.00, 1.03)

Table 6. Model 4, Timing of impact.

* p < .05, ** p < .01, and *** p < .001

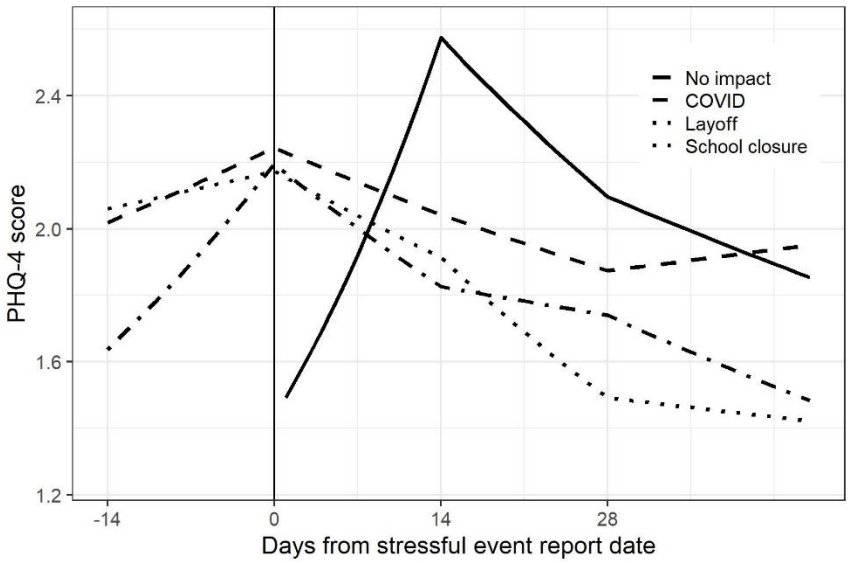

Figure 4. Adjusted trajectory of psychological distress, with time centered at the date participants first reported the stressful event, knots at 2 and 4 weeks following the event, and type of event as moderator.

DISCUSSION

Results suggest that the declaration of public health emergency on March 13 was followed by rapid increases in psychological distress across the population followed by gradual decrease. Differences in the number, type, and timing of events were not associated with change over time but were associated with distress levels: Individuals experiencing more stressful events reported more distress. In addition, age, sex, race/ethnicity, education, and pre-pandemic depression were associated with stressful events and distress, though associations for race/ethnicity and education did not reach statistical significance.

We discuss these results from the biomedical and structural perspectives below.

Discussion 1: Biomedical model

Results confirm that the COVID-19 pandemic was associated with psychological distress.

Results regarding specific hypotheses are mixed. Pandemic-related stressors were associated with higher levels of psychological distress, which supports H_{a1}. Increases in distress were comparable regardless of event type, contrary to H_{a2}. We observed differences in distress from the beginning of the study period, which counters H_{a3}. Among those experiencing stressful events, however, distress levels were greatest when the event was reported, which supports H_{a3}. Finally, psychological distress did not increase over time, inconsistent with H_{a4}.

Changes in psychological distress were comparable between those who reported *no* pandemic-related stressors and those who did. We attribute this to data quality issues. We did not have information on layoff or school closure among social contacts, and self-report may be subject to reporting bias or error. Chronic strains caused by the pandemic may have acted like acute stressors, and our data did not capture pandemic-induced loneliness and uncertainty. It will be important for future research to use improved measures specific to the pandemic context.

That psychological distress was elevated among those who experienced more stressors before the national declaration raises questions. One explanation lies in residual confounding, which future research may address by including additional measures of socioeconomic status (ex. income, wealth, social capital). It is also possible that a disproportionate number of these individuals were exposed to pandemic-related stressors, such as illness, earlier than the general population. Future research may include more precise data on the timing of stressful events.

Another explanation questions the direction of causality. While the Stress Process Model suggests social conditions leading to stressful events causes distress, the social causation

hypothesis suggests the opposite (Linton et al., 2017). Future work may explore our finding that distress increased prior to exposure.

Our results, combined with documentation of the pre-existing and rising mental health crisis in America, highlight the urgency for action. Pandemic-era policies, including expanded unemployment benefits, eviction moratoria, the child tax credit, and free school lunches, mitigated the impact of pandemic-related stressors. This study illustrated the widespread impacts of the COVID-19 pandemic on psychological distress, including via novel mechanisms conventionally treated as chronic strains. Greater understanding of specific pathways linking stress and health will be important for future study.

Discussion 2: Structural model

Following the national emergency declaration, the United States population experienced a shared, group-level trajectory of distress characterized by an increase that peaked 2-4 weeks after the declaration and returned to pre-pandemic levels within 2 months, consistent with previous research (Blanchflower & Bryson, 2022; Daly & Robinson, 2021). This trajectory emerged regardless of individual-level differences in the number, type, or timing of stressor exposure. Most strikingly, we observed this trajectory even among individuals who reported no specific pandemic-related stressors.

Our results provide empirical support for the structural model, which proposes group-level processes that supersede the individual. While these group-level processes are frequently obstructed by variation in individual-level stressful events, the shared shock of the pandemic brought them to the fore.

Our findings have implications for the study of stress and health at the population level as well as for understanding the "empirical paradox" raised in health disparities research.

First, our results suggest that the population-level relationship between stress and health in the early weeks of the pandemic supersedes individual-level pathways. Distress during this period was a shared, population-level experience. Pandemic stress was a social and structural, not individual, phenomenon.

Interestingly, distress increased more rapidly among individuals who reported more pandemic-related stressors in period 0, *prior* to the national declaration. These differences disappear by period 1, as the relative rate of increase declines, and subsequent rates are similar across groups. It is possible these individuals disproportionately experienced pandemic-related stress prior to the

national emergency declaration—the first COVID-19 case in the United States was reported on January 20, though impacts on the general population were minimal prior to March 13 (CDC, 2022). This would suggest that individual-level differences can shape trajectories in the short term, but that population-level processes supersede this variation in the longer term.

We focus on the trajectories, or rates of change over time, rather than differences in level, even though individuals who experienced more events had higher distress at all time points. This difference in levels, as opposed to rates of change, may reflect structural differences in pre-existing risk, consistent with life course theories (Ben-Shlomo & Kuh, 2002). However, when faced with a novel shock, these individuals' distress increased at rates similar to others in the population. This suggests that population-level processes shaped trajectories beyond individual differences in experiences prior to the pandemic.

Second, our results confirm documented disparities by race, gender, education, and age in exposure to stressful events and psychological distress. Consistent with the empirical paradox, BIPOC individuals in our study were more likely to experience multiple stressors but had on average lower levels of distress, though the difference did not reach statistical significance.

The pandemic was a social and structural stressor with population-wide effects. Individual-level exposure to discrete pandemic-related stressors was not associated with greater increases in distress. These results suggest that generalizing individual-level pathways to the population-level is mistaken. For individuals within a population bound together by the shared structural environment, distress supersedes the individual to affect the population more broadly.

The social stressors central to the paradox may be better understood as population-level shocks, like a pandemic, rather than discrete individual-level experiences, like illness. From the structural perspective, social stress such as discrimination increases psychological distress, not only for targeted individuals, but for all individuals within the shared environment. The fact that marginalized communities do not report higher distress is thus consistent with the structural model.

This study has limitations. While our national sample supports population-level analysis, attrition or non-response bias may threaten conclusions (Krieger et al., 2023). It will be important to examine whether our results are specific to the COVID-19 pandemic or generalizable to population stress more broadly, such as the 2008 recession or the 9/11 attacks in the United States. Future research may compare pandemic-era population distress trajectories across countries or investigate the structural model's implications for other social stresses, such as structural discrimination or income inequality (Krieger, 2000; Phelan et al., 2010).

The current study raises questions about the population-level recovery process following distress. We find that recovery occurred relatively quickly following the COVID-19 emergency declaration. How we understand this apparent recovery, to what extent the shared environment shaped a shared trajectory, and whether community characteristics moderated those trajectories remains unknown.

The structural model encourages transdiagnostic conceptualizations of health. A return to pre-pandemic distress levels is no cause for celebration when accompanied by rising death rates (Weinberger et al., 2020; Woolf et al., 2020) and increased disparities (Dukhovnov & Barbieri, 2022). It will be important to go beyond single measures like psychological distress to explore the pandemic's transdiagnostic impacts.

The structural model posits that shared social and structural resources shape group-level recovery trajectories that supersede individual-level differences. This "structural resilience" (Acolin & Fishman, 2023) departs from current conceptualizations of "resilience" as the individual-level capacity to recover from adverse shocks (Chesterman et al., 2021; Masten et al., 2021). The current study, while focused on the shared drivers of distress, nevertheless provides preliminary evidence for shared recovery. Whether and to what extent shared recovery supersedes individual-level coping resources requires further investigation.

Community-specific resources may moderate recovery trajectories. For instance, studies document the protective role of close kin relationships and positive reinforcement of racial identity specifically in African American communities (Louie & Upenieks, 2022; Taylor et al., 2013). Such differences in community-level resources may further elucidate mechanisms underlying the empirical paradox. It will be important to develop and test structural hypotheses for their impact on recovery trajectories.

Our results highlight the potential importance of group-level processes. Interventions informed by individual-level models that target vulnerable communities or specific diagnostic outcomes may miss crucial population-level mechanisms. Greater focus on the concrete role of shared institutions and infrastructure in shaping population health is needed.

Evaluating the two discussions

Accuracy: Both models explained results.

Both discussions offered plausible explanations for results.

Consistency: The structural model was more consistent.

To account for mixed results, the biomedical discussion referenced the exceptional nature of the pandemic. The need for specialized measures runs counter to the consistency criteria. Overall conclusions that pandemic stressors caused distress run counter to the mixed results to specific hypotheses.

By contrast, the same results were consistently interpreted within the structural discussion as evidence of group-level processes.

Simplicity: The structural model was simpler.

The biomedical explanation proposed *post-hoc* hypotheses to explain findings. These alternate pathways added situation-specific complexity to the basic pathway described by the Stress Process Model.

A single common mechanism explained the observed findings in the agentic explanation.

Scope: The structural model had broader scope.

The biomedical treatment of the COVID-19 pandemic as exceptional limits generalizability and, in turn, scope. The pandemic's exceptional nature raises doubts about whether policy recommendations would apply in alternate scenarios.

The structural model draws on generalizable group-level processes intended to apply across contexts. COVID-19 is not an exception to the rule but rather an opportunity to observe an otherwise generalizable phenomenon.

Fruitfulness: The structural model proposed novel hypotheses.

Future research suggested within the biomedical discussion focused on addressing study limitations to test the same underlying hypothesis. The structural discussion raised novel hypotheses that extended current results.

Conclusion

The above discussion suggests that, compared to the biomedical model, the structural model provides a better framework for the population-level relationship between stress and health.

These results are promising. Nevertheless, more research is needed to determine whether the structural model holds up to the rigor and investigation currently applied to the biomedical model.

The need for such research is urgent. The COVID-19 pandemic brought to the fore the extent to which individual lives are interconnected in the modern world. Future shared shocks, from climate change to financial crises, threaten population health. Concerted public health efforts failed to counter the pandemic's systemic threat to morbidity and mortality due to a continued reliance on the biomedical model. Further investigation into the structural model has potential to advance the field.

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TITLE: Evidence for population distress as a structural predictor of vaccine hesitancy

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ABSTRACT

OBJECTIVE: To investigate whether state-level psychological distress is associated with individual vaccine hesitancy during the COVID-19 pandemic, over and above individual distress.

METHODS: Our study took place in the United States between August 19, 2020 to July 31, 2021. We merged the Household Pulse Survey (1,387,694 observations) and the Understanding Coronavirus in America surveys (N=6,808; 99,277 observations) and to create a multi-level longitudinal dataset. We used multilevel logistic regression to test the association between within-state changes in average distress, individual distress, and individual vaccine hesitancy.

RESULTS: Individual distress was not associated with vaccine hesitancy. Conversely, a one standard deviation increase in state-level distress was associated with 5% higher odds of individual vaccine hesitancy (95% CI: 0.99, 1.12). Interactions between state-level distress and time were significant.

CONCLUSIONS: State-level distress is distinct from individual distress and may be associated with vaccine hesitancy. The shared structural environment produces conditions beyond individual-level experiences that shape population health.

POLICY IMPLICATIONS: In contrast to immutable social, political, or demographic characteristics, state-level distress may be a modifiable predictor of vaccine hesitancy. Attention to the congruence between concurrent policies, beyond single causal relationships, may guide effective policy development.

KEYWORDS: *Structural model of population health, psychological distress, vaccine hesitancy, COVID-19*

INTRODUCTION

PUBLIC HEALTH PROBLEM

Over the decades, critiques of “risk factor epidemiology” and the biomedical model’s dominance in public health have grown (Schwartz et al., 1999). Most recently, the COVID-19 pandemic highlighted the limitations of sole reliance on the biomedical model (Acolin & Fishman, 2023). For instance, while vaccination as an effective preventive measure emerged from the biomedical model, the impacts of political polarization on uptake (Roberts et al., 2022) cannot be explained, nor resolved, by a biomedical perspective.

OUR RESPONSE: THE STRUCTURAL MODEL

To address this gap, our prior theoretical work developed an alternative model for population health (Acolin & Fishman, 2023). Our proposed structural model is built on two foundational observations. First, individuals overcome varied life obstacles, including but not limited to disease, in order to pursue personally meaningful goals.. This leads us to identify resilience, or the capacity to adapt to negative shocks and stressors, as the transdiagnostic process underlying individual health in the short-term (Badash et al., 2017). The obstruction of goal attainment increases distress, while its realization produces satisfaction. Individual resilience can be observed as the trajectory of distress and recovery following adversity (Acolin et al., Forthcoming; Bonanno, 2005; Kimhi et al., 2021).

Second, individuals within a population rely on shared resources and infrastructure. We hypothesize that the shared structural environment shapes the individual achievement of goals beyond individual traits or behaviors This leads to the proposition that population health is achieved when the shared structural environment enables individual resilience and to posit that average population distress captures the structural environment’s contribution to resilience, beyond individual-level variation.

THE STRUCTURAL MODEL AND POPULATION HEALTH POLICY

Population health policymakers pursue varied objectives, such as the elimination of an infectious pathogen or the reduction of risk behaviors (e.g. alcohol consumption). Scientific research conducted within the biomedical framework provides causal evidence for the relationship between these policy targets and health outcomes. However, evidence for biomedical efficacy is not sufficient to ensure real-world effectiveness in the population.

From a structural perspective, we posit that a structural environment that enables individual resilience is a foundational precursor to policy effectiveness. This is because individuals will be more likely to work with, rather than against, policymakers to achieve shared population health goals when the structural environment promotes, rather than obstructs, individual resilience.

We propose two mechanisms for this relationship. First, the policymaker's demonstrated commitment to a supportive structural environment builds a foundation of trust that may increase the individual's willingness to trade-off personal priorities for policy goals when needed. Behaviors necessary for population health are not always in the individual's self-interest. For instance, the policy goal to eliminate an infectious pathogen requires individuals to be vaccinated. For medically low-risk individuals, the costs of vaccination outweigh the personal benefits. When the policymaker asks individuals to prioritize communal over individual benefits (Fuchs, 1974), individuals are more likely to comply if the policymaker has previously demonstrated commitment to creating an environment that supports individual well-being. This mechanism relates to the concept of *structural resilience*, which captures the extent to which the structural environment promotes communal mobilization in response to novel and unpredictable shared threats.

Second, individual adherence to public health behaviors entails costs in time, resources, and foregone opportunities that are more manageable within an environment that enables, rather than obstructs, individual resilience. We posit that, when the structural environment enables the individual pursuit of personal goals, individual capacities to handle additional population health demands are greater which, in turn, increases the likelihood of compliance. This mechanism relates to the concept of *congruence*, which highlights whether the interconnected elements of the structural environment enable or obstruct individual resilience. Both structural resilience and congruence exist only at the population-level.

Taken together, we hypothesize that lower levels of population distress will be associated with increased individual adherence to population health policy goals.

THE CURRENT STUDY: FROM THEORY TO EVIDENCE

Empirical investigation of the structural model remains limited. The current study contributes to filling this gap by exploring the structural model's proposed relationship between population distress and individual adherence to policy goals during the COVID-19 pandemic.

Population health policy goal: COVID-19 vaccination

We focus on vaccination as a key pandemic-era population health policy goal in the United States. The 2019 novel coronavirus, or 2019-nCoV, was first detected in December 2019 before spreading globally. In the United States, the Trump administration prioritized vaccination to counter the public health threat and launched Operation Warp Speed on April 30, 2020. The first viable COVID-19 vaccine, produced by Pfizer-BioNTech, received emergency use authorizations from the Food and Drug Administration on December 11, 2020. By March 13, 2021—one year after the initial emergency declaration—over 100 million COVID-19 vaccines doses had been administered in the United States (CDC, 2022a).

While other countries prioritized alternate approaches, including border lockdown and strict quarantine, vaccination was the primary goal in the United States. Yet, vaccination proved contentious. By December 2021, after a year of vaccine availability, barely 60% of the US population had completed the primary series (CDC, 2022b) compared to the 80% needed for herd immunity (World Health Organization, 2020). Investigation into whether the structural model contributes to explain vaccine hesitancy during the COVID-19 pandemic holds public health importance.

Evidence documents several individual-level predictors of vaccine hesitancy, including demographic characteristics (e.g. race, sex, age, education), perceived risk of disease, perceived benefits of vaccination, and barriers to access (Limbu et al., 2022). During the COVID-19 pandemic, political affiliation also emerged as a strong predictor of vaccine hesitancy (Roberts et al., 2022). We include these predictors as control variables to isolate the effect of population distress.

Population distress

The structural model highlights the role objective characteristics, such as shared infrastructure, administrative, or governance elements, in defining the bounds of a population (Acolin & Fishman, 2023). In the context of the United States during the COVID-19 pandemic, we operationally define population at the state level. This is because, while the federal government provided guidance and strategic oversight, the logistics and execution of pandemic policies were delegated to the state-level.

We investigate population distress as a time-varying predictor, in order to better estimate effects due to changes in the shared structural environment rather than to underlying differences between states.

Individual psychological distress

Because a key premise of the structural model is that structural conditions impact population health outcomes beyond individual-level processes, we investigate whether the effects of population-level distress supersede individual-level distress. For this reason, we also investigate the relationship between individual distress and vaccine hesitancy.

Prior literature documents that psychological distress predicted greater vaccine hesitancy in China (Xu et al., 2021) and Japan (Okubo et al., 2021; Sekizawa et al., 2022). Adults reporting symptoms of anxiety and depression were more likely to endorse false claims about the novel coronavirus (Perlis et al., 2022) and less likely to receive COVID-19 vaccination in the United States (Nguyen et al., 2022). COVID-specific anxiety positively predicted vaccine acceptance in Germany, while social and economic fears were associated with decreased acceptance (Bendau et al., 2021). A cross-national investigation found that population-levels of negative emotions, anxiety, and stress predicted variation in vaccine coverage (Vermeulen, 2023).

Yet, individual variation in goals vary, and some individuals may nevertheless prioritize vaccination due to health needs or normative values. This variation may attenuate or reverse the expected relationship, and evidence suggests this may be the case (Bai et al., 2021; Batty et al., 2022; Killgore et al., 2021). Thus, our *a priori* hypothesis is that individual psychological distress will either be associated with greater vaccine hesitancy or have no association. We do not expect an inverse relationship.

Time trends

Circumstances changed rapidly during the COVID-19 pandemic, and both psychological distress and vaccine hesitancy may have decreased over time due to secular trends rather than structural changes. Concurrent economic and political developments may confound the association between population distress and vaccine hesitancy. To isolate the effect of population distress, we control for time in our analysis.

The rapidly changing circumstances of the COVID-19 pandemic introduce a secondary hypothesis. While some components of the structural environment, such as the built environment, are relatively stable and change slowly over time, others (e.g. policy measures, political communications, geopolitical conflict) may produce rapid changes. Such rapid changes may have unintended effects by introducing novel situations or additional obstacles to which the individual must adapt. Whether and how these changes impact the relationship between population distress and individual vaccine hesitancy is unknown. For this reason, as a secondary analysis, we explore interaction effects between state-level distress and time.

Study aims

Our study has two primary aims and one exploratory aim. Our first aim is to investigate the association between population-level distress and vaccine hesitancy. We hypothesize that higher state-level distress will be associated with higher individual hesitancy, independent of time. Our second aim is to compare the effects of population- and individual-distress. We hypothesize that the effect of population distress will supersede individual distress, and that individual distress will either be positively associated or have no association with vaccine hesitancy. Our third aim is to explore the interaction effects of time. This third aim is exploratory and not guided by an *a priori* hypothesis.

DATA

We combined the Household Pulse Survey (HPS), a nationwide, repeated cross-sectional survey administered bi-weekly by the US Census Bureau (United States Census Bureau, 2021), and the Understanding Coronavirus in America survey (UCAS), a bi-weekly panel survey (Kapteyn et al., 2021), to create a multi-level longitudinal dataset.

Our study period goes from August 19, 2020 (when the HPS began to consistently measure psychological distress) to July 31, 2021 (the end of UCAS bi-weekly surveys). This resulted in the inclusion of 6,802 participants with 99,277 total observations from UCAS and nearly 1.4 million observations from the HPS with an average of 1,296 observations per state-wave (SD: 820).

MEASURES

Vaccine hesitancy was measured in the UCAS with the question: How likely are you to get vaccinated for coronavirus once a vaccine is available to the public? This question was included

in every wave during the survey period. Starting December 2020, it was asked only to those who remained unvaccinated. Responses were recoded to reflect vaccine hesitancy (0: Somewhat or very likely, 1: Somewhat or very unlikely, or unsure).

Psychological distress was measured using the four-item Patient Health Questionnaire (PHQ-4) (Kroenke et al., 2009) , collected in both the HPS and the UCAS for every survey wave during the study period. **Population distress** was calculated as the survey-weighted mean by state-wave. We centered state distress at the national mean and coded in increments of one national standard deviation (mean: 3.57, SD: 0.41 on the PHQ-4 scale). **Individual distress** was calculated as the summed score of the PHQ-4 individual items (Kroenke et al., 2009; Lowe et al., 2010) (Cronbach's alpha = 0.93). Possible scores range from 0 (no distress) to 12 (severe distress).

Time was coded as a dummy variable for each survey wave of the HPS and had 21 levels.

Perceived risk of COVID-19 infection was measured in each survey wave of the UCAS and included as time-varying control variables. **Political affiliation, age, gender, education, and race/ethnicity** were included as time-fixed control variables. Political affiliation was ascertained between August 3-November 3, 2020 as part of an election tracking segment of the UCAS. Remaining demographic control variables were ascertained from the most recent responses to the My Household survey, which is administered every three months to all participants. Details on these variables are reported in Appendix A.

ANALYTIC STRATEGY

We used multilevel logistic regression with random intercepts for the individual and state (Gelman & Hill, 2006) to examine the association between within-state changes in psychological distress and individual-level vaccine hesitancy. In our first model, we modeled individual vaccine hesitancy as a function of state-level distress, individual distress, time, and control variables. For our exploratory aim, we ran a second model that included an interaction between state-level distress and time. We follow conventions with logistic regression and exponentiate estimated coefficients to estimate odds ratios.

While missing responses were dropped, we used all available data so participants with partial data remained in the analysis. Resulting estimates are unbiased under the assumption that missingness occurs at random (MAR)(Gelman & Hill, 2006) .

All statistical analysis were performed using R 4.0.2 (R Core Team, 2020) using the "glmmTMB" package for multi-level modeling (Brookes et al., 2017) and the "survey" package for survey-weighted means (Lumley, 2020).

RESULTS

At the start of the study period in August 2020 (prior to vaccine availability), 60% of participants were likely to accept a vaccine, while 40% expressed hesitancy. Those who expressed hesitancy were younger, more likely to be female or non-White, to have lower educational attainment, and to be Republican. Perceived risk of COVID-19 infection was higher in the non-hesitant group, while psychological distress was comparable in the two groups (Table 1).

	Total N=6802	**Vaccine hesitant in August 2020** (39.6%)	**Not vaccine hesitant in August** 2020 (60.4%)
# observations (Mean [SD])	14.9 [4.8]	14.4 [4.97]	15.3 [4.55]
Age (Mean [SD])	51.1 [16.1]	49.0 [15.6]	52.7 [16.4]
% female	59.0%	65.2%	54.8%
% BIPOC	33.2%	36.7%	30.4%
Education			
Less than HS	4.8%	6.9%	3.3%
Some college	54.2%	64.4%	47.4%
Bachelor's degree	24.5%	19.2%	28.1%
Graduate degree	16.5%	9.5%	21.2%
Political affiliation			
Democrat	39.5%	27.2%	47.5%
Republican	32.4%	40.3%	27.2%
Other	28.1%	25.2%	32.5%
PHQ-4 scores (Mean [SD])	1.72 [2.80]	1.62 [2.80]	1.77 [2.78]
Perceived risk of COVID-19 infection (Mean [SD])	19.95 [22.0]	19.3 [22.0]	20.2 [21.8)

Table 1. Sample descriptives.

Results from our first model, minus presentation of time dummy variables, are presented in Table 2. We do not observe an association between individual distress and vaccine hesitancy. Over the study period, a one standard deviation increase in state psychological distress (≈0.40 points on the PHQ-4 scale) was associated with 5% higher odds of an individual reporting vaccine hesitancy (p=0.089, 95% CI: 0.99, 1.12).

	Odds ratio (e^{β}) (95% CI)	p-value
Predictors of interest		
State-level distress	1.05 (0.99, 1.12)	0.089
Individual PHQ-4 score	0.99 (0.97, 1.00)	0.149
Control variables		
Perceived risk of COVID-19 infection[1]	1.00 (1.00, 1.00)	<0.001
Age	0.96 (0.95, 0.96)	<0.001
Male	0.37 (0.31, 0.46)	<0.001
Edu: Some college	0.39 (0.25, 0.61)	<0.001
Edu: Bachelor's degree	0.06 (0.04, 0.10)	<0.001
Edu: Graduate degree	0.03 (0.02, 0.13)	<0.001
Race (non-White)	2.96 (2.33, 3.76)	<0.001
Political affiliation: Republican	21.38 (16.64, 27.48)	<0.001
Political affiliation: Other	8.59 (6.64, 10.90)	<0.001

Table 2. Model 1 results (excluding time dummy variables)

Results from the interaction model are presented in Figure 1, with graphical illustration of time trends. Estimated time trends were similar in both models. Overall, population distress increased until December 2020 before gradually decreasing, with vaccine hesitancy showing similar trends. We find that, from August 2020 until February 2021 and from May to June 2021, one standard deviation increase in state-level distress was associated with 20% higher odds of hesitancy (95% CI: 1.02, 1.42). This association was reversed from mid-February to mid-May

[1] The estimated odds ratio for perceived risk of infection is presented as 1.00 due to rounding. With the inclusion of more digits, the estimated odds ratio is 0.9976 (95% CI: 0.9960, 0.9992). The magnitude of the association, while statistically significant, is clearly very small. However, the direction is consistent with conceptual models relating individual perceived risk/benefit to vaccine uptake (a one percent increase in perceived risk of infection is associated with an approximately 1% lower odds of vaccine hesitancy).

2021, a period that appears to correspond with the initial period of widespread vaccine distribution (Raifman et al., 2021). Estimates for control variables were similar to Model 1.

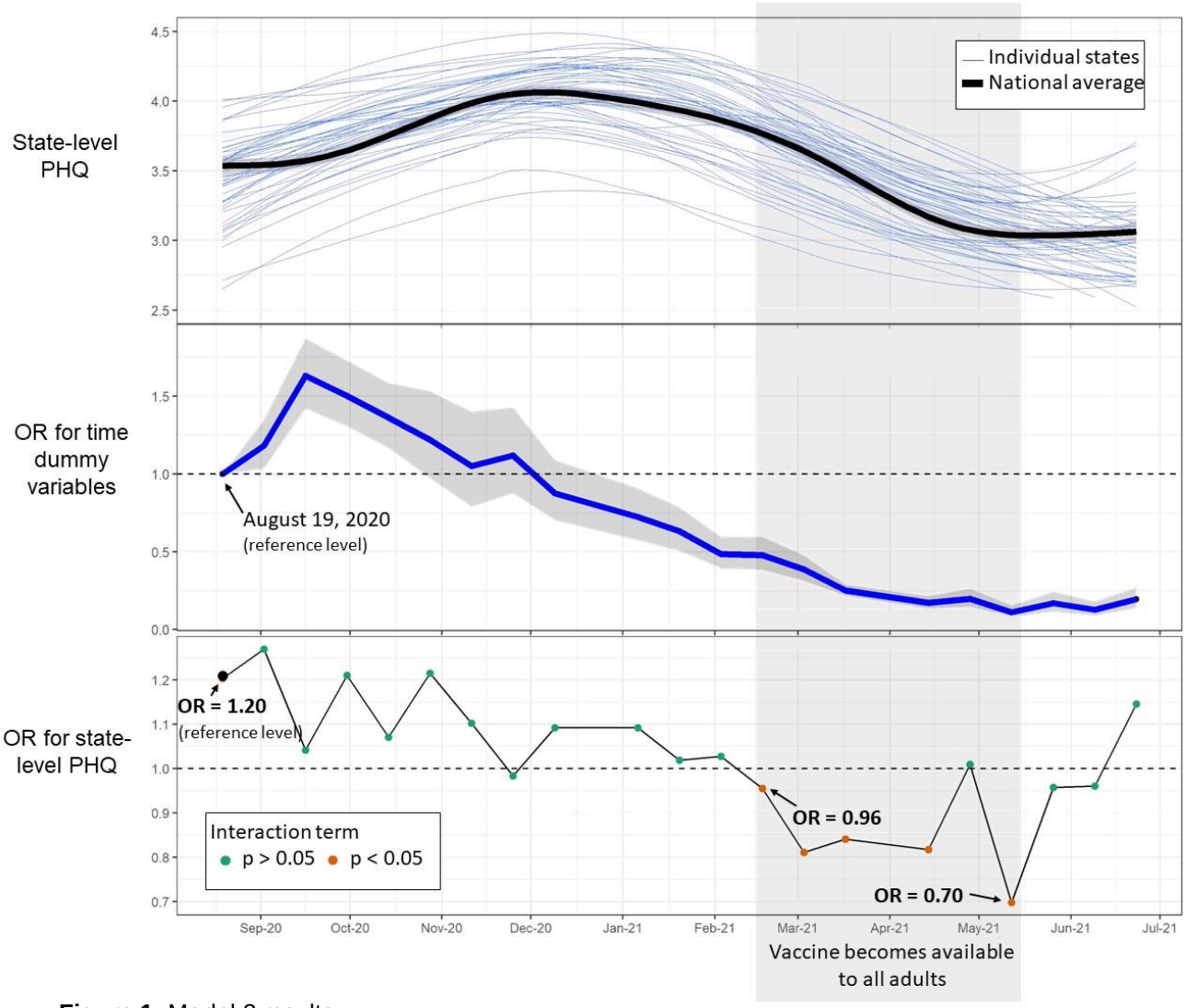

Figure 1. Model 3 results.

DISCUSSION

This study is the first to explore the structural hypothesis that state-level psychological distress is associated with individual adherence to population health policy goals. For our first aim, we find that increased within-state distress appeared to be associated with higher individual-level vaccine hesitancy. The direction of the association is consistent with our hypothesis, though the

association was not statistically significant at the 0.05 level. For our second aim, we find that the association between state-level distress supersedes the individual-level association, and that individual distress was not associated with vaccine hesitancy. For our exploratory aim, we find that the association between state-level distress and vaccine hesitancy reversed from mid-February to mid-May, which corresponds to the initial period of widespread vaccine distribution (Raifman et al., 2021).

Our findings are subject to at least four limitations. The HPS is an experimental data product. While designed to produce representative estimates at the state- and national-level, concerns about bias due to response rates or differential missingness remain (Vahratian et al., 2021). While the PHQ-4 has been validated for measuring psychological distress in the general population, concerns remain about its reliability across subpopulations (Han et al., 2008; Maffini & Wong, 2014; Materu et al., 2020; Milani et al., 2004). Our estimates of state distress levels may be biased, though changes over time within the same population remain internally valid. Though we included important individual-level control variables, residual confounding remains, as always, a risk. It will be important for our results to be replicated using alternate datasets in diverse settings, though the paucity of nationally representative longitudinal data on psychological distress may prove difficult to overcome.

We draw three general conclusions from our findings.

First, further exploration of the drivers of state-level distress and the mechanisms linking it to population health may be fruitful. We found suggestive evidence that state-level distress was associated with increased vaccine hesitancy. Further exploration of these results may contribute to improved understanding the structural drivers of vaccine hesitancy. In addition, the structural model posits that the impacts of population distress on population health is not limited to vaccine hesitancy but may be generalizable to a range of policy goals via the mechanism of structural resilience. The implications of this line of inquiry may consequently extend beyond the specifics of the COVID-19 pandemic context. Finally, because state-level distress—in contrast to immutable social, political, or demographic characteristics—may be responsive to policy, improved understanding of its contribution to population health may lead to actionable interventions.

More broadly, population health cannot be reduced to individual-level mechanisms. We found that the association between state-level distress and individual vaccine hesitancy superseded

individual distress. This finding provides empirical support for the structural model hypothesis that the shared structural environment produces conditions beyond individual-level experiences that shape population health. Future work may build on this finding to investigate whether and to what extent state-level distress relates to the concept of structural resilience.

Finally, it will be important to further explore how the overall policy landscape impacts individual behavior, beyond the isolated effect of any single policy. Exploratory analyses indicated that state-level distress interacted with time to result in a reversal of the association during the period of vaccine distribution. We interpret these results as suggestive that the congruence between policies, which can change over time, is more meaningful than any single policy in isolation. In doing so, we concur with McKee et al (2023), who similarly argue for increased attention to the relationship between concurrent health determinants as a path forward for population health. The shift in perspective represents a departure from biomedical notions of health towards a more comprehensive, structural approach.

PUBLIC HEALTH IMPLICATIONS

Because empirical investigation of the structural model is in its infancy, the primary implications of these findings regard the need for additional research. Nevertheless, preliminary implications for public health emerge.

Treating population health as distinct from, though related to, aggregate individual health will spur the development of policies targeting the structural environment rather than individual behavior. This broadens the purview of public health and will necessitate cross-sectoral collaboration.

A focus on congruence widens the lens from single policy measures to encourage thoughtful analysis of how concurrent policies work together. Policy development must combine evidence illustrating the causal impacts of single policies with context-specific expertise, and the knowledge of community members will be invaluable.

Historically, well-meaning policies have resulted in disparate impact, potentially due to a mismatch between the goals of policymakers and those of marginalized communities. To effectively achieve population health goals, policymakers must first demonstrate a commitment to creating a structural environment that supports individual goals. Population distress may

serve as a meaningful indicator of the structural contribution to individual resilience. Attention by policymakers to individual resilience across communities within the population contributes to structural resilience, which may be a necessary precursor to policy effectiveness.

The current study provides a foundation for future work with the potential to advance public health theory and practice.

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APPENDIX A: CONTROL VARIABLES

Variable	Measure	Coding
Perceived risk of COVID-19 infection	"On a scale of 0 to 100 percent, what is the chance that you will get the coronavirus in the next three months? If you're not sure, please give your best guess." Response options: 0-100	Continuous Range: 0-100
Age	"What is your date of birth?" Response options: 1910-2015 (year)	Calculated as the year the participant turned in 2020 Continuous
Gender	"What is your gender?" Response options: 1 Male 2 Female	Binary 0: Female 1: Male
Education	"What is the highest level of school that you have completed or the highest degree you have received?" Response options: 1 Less than 1st grade 2 Up to 4th grade 3 5th or 6th grade 4 7th or 8th grade 5 9th grade 6 10th grade 7 11th grade 8 12th grade-no diploma 9 High school graduate or GED 10 Some college-no degree 11 Assoc. college degree-occ/voc prog 12 Assoc. college degree-academic prog 13 Bachelor's degree 14 Master's degree 15 Professional school degree 16 Doctorate degree	Factor 0: HS or less 1: Some college 2: Bachelor's degree 3: Graduate degree
Race	"Here is a list of five race categories. Please choose all that apply." Response options:	Binary 0: White only

	1 White 2 Black or African American 3 American Indian or Alaska Native 4 Asian 5 Native Hawaiian or Other Pacific Islander	1: Any non-White
Political affiliation	“Regardless of if or how you are registered to vote, are you more closely aligned with...” Response options: 1 Democrats 2 Republicans 3 Independents (no political party) 4 Libertarians 5 Green party 6 Some other party 7 Not aligned with any political party	Factor 0: Democrats 1: Republicans 2: Other

REDEFINING RESILIENCE AS A PUBLIC HEALTH RESPONSE TO STRESS

CONCLUSION

In the introduction, I described the two problems that motivated this dissertation. I did not mention that this dissertation was also motivated by my interest in and belief that resilience posed a potential solution. This interest was apparent in my dissertation proposal, in which I proposed to develop and evaluate a measure of population resilience as a public health response to social and structural stress.

My original idea was to operationalize population resilience as the trajectory of increasing distress and subsequent recovery following identifiable shock, and then to test the hypothesis that population resilience predicted future public health response. This approach drew from prior literature identifying this trajectory as a potential indicator of individual resilience and had preliminary support from evidence suggesting that population distress levels did, in fact, adhere to the hypothesized trajectory in the months following the COVID-19 public health emergency.

I was able to estimate state-level trajectories of distress following the emergency declaration. Preliminary results suggested that these trajectories had meaningful variation and were significantly associated, both with state-level predictors (e.g. child poverty, rent burden, education levels) and with public health outcomes (state-level vaccination levels). I found suggestive evidence that measures of the trajectory behaved differently from measures of level, consistent with my original conceptual framework, and that they meaningfully distinguished subpopulations along the lines of race, gender, and political affiliation.

These preliminary analyses did not make it into the dissertation, however, because while pursuing this line of investigation, I grew increasingly concerned with what the measure was capturing. Evidence suggested that, across the board, distress levels returned to pre-pandemic levels within several months. Yet, could I truly interpret this as indicative of resilience, given concurrently rising morbidity and mortality, heightened social tensions, financial hardships, and political polarization? Did reduced population distress truly reflect “recovery”? How do I interpret the changing levels of population distress against reports of rising mental distress in the nation?

From this preliminary work, I realized that extrapolating individual-level conceptualizations of distress and resilience to the population-level might be conceptually flawed. Consequently, I put on hold the notion of population resilience as trajectory of distress following shock to wrestle

with this conceptual problem, fueled by the belief that its resolution was a necessary precursor to any further study of population resilience.

The three papers of the dissertation reflect this evolution. Though they depart from the original analytical plan, these papers nevertheless respond to the original motivating problems.

The first paper's primary contribution is to establish the necessity for treating population health as more than the sum of specific social and structural risks, which opens the way for the development of unifying strategy for addressing disparate social determinants. Its secondary contribution is to highlight the importance of respecting individual agency, which makes explicit a guiding normative value for the field. I believe these conclusions provide a foundation on which future research may build. It cannot remain the status quo to extrapolate from individual-level processes to population-level outcomes, and public health cannot continue to treat individuals as passive recipients of policy or treatment.

The proposed structural model is a tertiary contribution, and one whose staying power will be tested over time. I propose two organizing concepts for further study (structural resilience and congruence), a framework for distinguishing communities within a population, and affiliation and competition as common underlying processes in the production of health disparities. This paper goes beyond criticism of the biomedical model to propose a viable alternative, and as such represents a contribution to the literature. Yet, whether the proposed model is empirically robust remains unknown. Future work is needed to refine the conceptual premises, operationalize their measurement, and test hypothesized relationships.

The second paper's primary contribution is as an example of a systematic approach to comparing paradigmatic assumptions. Despite meaningfully shaping both the asking and answering of research questions, such assumptions frequently remain implicit. Implicit assumptions cannot be tested, which means that we cannot know if they are being wrongly applied. Yet, it may be such faulty assumptions that produce problems such as the treatment of distress as an individual issue. The second paper uses population distress during the COVID-19 pandemic as a case study to conclude in favor of the structural model. This conclusion is only a starting point. It is my hope that this paper may spur future systematic evaluations of empirical evidence on social and structural exposures and outcomes through the lens of competing paradigms.

The third paper was an experiment in empirically testing the structural model. Its primary contribution to the literature is to test a novel hypothesis. However, its contribution to my growth may be more important. As I wrestled with the results, modified my analysis plan, and wrote draft after draft, I discovered logical and empirical pitfalls of the structural model. What does structural resilience mean in practice? How might we identify whether a policy environment is congruent?

From the third paper emerge two directions for future research. The first involves better understanding population-level distress. Does population distress fit within the structural model's hypotheses and, if so, how? The second involves investigation into differences in community distress within the population. Results of exploratory analyses suggest that experiences of distress following social or structural shocks differs by race, sex, political affiliation, immigration status, or news source, and that these differences appear meaningfully associated with population health outcomes. I did not include this line of investigation into the dissertation because I did not know how to interpret these results. This line of inquiry may require additional theoretical alongside empirical work.

Ultimately, my work on this dissertation has left me with more questions than answers. The final product is exploratory rather than conclusive. Yet, in the course of this work, I have had the opportunity to explore ideas—to read widely, write frequently, and dive deeply into data. I have also learned about the practice of scientific research, about team science and constructive criticism, about data quality and coding challenges and the art of responding to reviewers. I have, in short, had the privilege of being a student. The lessons I learned in this time will guide me throughout my academic career.