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Biocultural perspectives on bioarchaeological and paleopathological evidence of past pandemics

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Abstract

Objectives: Pandemics have profoundly impacted human societies, but until relatively recently were a minor research focus within biological anthropology, especially within biocultural analyses. Here, we explore research in these fields, including molecular anthropology, that employs biocultural approaches, sometimes integrated with intersectionality and ecosocial and syndemic theory, to unpack relationships between social inequality and pandemics. A case study assesses the 1918 influenza pandemic's impacts on the patient population of the Mississippi State Asylum (MSA).

Materials and Methods: We survey bioarchaeological and paleopathological literature on pandemics and analyze respiratory disease mortality relative to sex, age, and social race amongst patient deaths ($N = 2258$) between 1912 and 1925. Logistic regression models were used to assess relationships between cause of death and odds of death during the pandemic (1918–1919).

Results: Findings include substantial respiratory mortality during the pandemic, including from influenza and influenza syndemic with pneumonia. Older patients (40–59 years, 60+ years) had lower odds ($p < 0.01$) of dying from respiratory disease than younger patients, as did female patients compared to males ($p < 0.05$). Age patterns are broadly consistent with national and state trends, while elevated mortality amongst Black and/or African American patients may reflect intersections between gender roles and race-based structural violence in the Jim Crow South.

Discussion: Future work in biological anthropology on past pandemics may benefit from explicit incorporation of biocultural frameworks, intersectionality, and ecosocial and syndemic theory. Doing so enables holistic analyses of interactions between social context, social inequality and pandemic outcomes, generating data informative for public health responses and pandemic preparedness.

KEYWORDS
bioarchaeology, biocultural, biosocial, paleopathology, pandemic

1 | INTRODUCTION

Pandemics, defined as infectious disease epidemics occurring worldwide or over an extensive area and affecting a large number of people (Last, 2001), have exerted significant, diverse effects on human societies for millennia (Sherman, 2017; Snowden, 2019; Watts, 1997). These include elevated mortality and morbidity, but also a historically contingent range of intersecting political, economic, demographic, epidemiological, ecological, social, and cultural conditions and processes, some of which occurred in the short term, while others endure for

hundreds of years (Sherman, 2017; Snowden, 2019; Watts, 1997). Amongst these, perhaps the most consistent is an interplay between pandemics and social and economic inequalities (in this paper referred to as social inequality), which directly or indirectly mediate the course and impacts of pandemics (e.g., pathogen immunity and behavioral changes) (Sherman, 2017; Snowden, 2019; Wade, 2020; Watts, 1997). Indeed, research in biological anthropology on past pandemics has shown that they "play out along the preexisting fault lines" and fragilities of societies, often having the most deleterious impacts on socially marginalized communities, and often exacerbating existing inequities, such as health disparities (Wade, 2020: 700).

When it comes to comprehending the effects of past pandemics, this phenomenon means that biological anthropology, especially its fields of bioarchaeology and paleopathology, and molecular anthropology are particularly well positioned (Dimka et al., 2022; Larsen & Crespo, 2022). Unlike historical records, which tend to represent the biological and social experiences of more privileged communities while underrepresenting those of socially marginalized groups, assemblages of skeletal individuals and the bioarchaeological record more regularly represent a wider range of populations, especially socially marginalized ones (Grauer, 2003). Further, because deceased human bodies represent direct material archives of these experiences, when data from them can be integrated with archeological, and historical and/or ethnographic data, the scope and depth of insights into health, disease, and biology of otherwise underrepresented communities is expanded past what is available from these data sets used in isolation (Herring & Swedlund, 2003), for pandemics and other conditions and processes.

Despite this, pandemics have only relatively recently emerged as a major research focus within biological anthropology, including bioarchaeology and paleopathology (Dimka et al., 2022). Here, we define bioarchaeology as the study of health, stress, disease, nutrition and diet, and lifestyle in the past through historically and/or archeologically contextualized human remains. Paleopathology is the study of disease in the past primarily through analysis of human remains. The relative scarcity of pandemic research, until very recently, is especially true for analyses that employ an explicitly biocultural approach.[1] This is puzzling because these fields focus on health and disease in the past, including broad coverage of infectious disease and understanding disease as biocultural phenomena; its biological aspects and impacts cannot be disentangled from its sociocultural and political economic dimensions (Ortner, 2003).

One cause of this relative scarcity has likely been material and methodological limitations. One major, persistent limitation is the difficulty in detecting evidence of pandemics in the bioarchaeological record. Few archeological sites and associated skeletal assemblages possess the degree of temporal specificity needed to associate them with a distinct pandemic. Further, many non-pandemic phenomena can generate catastrophic mortality[2] (vs. attritional), meaning that mass graves (i.e., plague pits) cannot always be reliably associated with pandemics (DeWitte, 2012; Grainger et al., 2008). Pandemics can also drive excess mortality, or deaths from all causes during a crisis above the numbers of deaths expected in a non-crisis period (Morens & Fauci, 2007). This means that not all individuals within an assemblage or grave associated with a pandemic may have died from the causal infection (DeWitte & Wood, 2008). These issues are compounded by the fact that most infectious diseases do not leave distinct skeletal signatures (Buikstra, 2019; Ortner, 2003).

Advances in molecular anthropology (e.g., Next Generation sequencing and in-solution capture) have enabled evolutionary and epidemiological assessment of many pathogens that are otherwise archeologically and skeletally invisible (e.g., Austin et al., 2022; Ferrari et al., 2020; Mühlemann et al., 2018; Ross et al., 2018; Vågene et al., 2018), including past pandemic pathogens, such as *Yersinia pestis* (bubonic plague) and *Vibrio cholerae* (cholera) (Devault et al., 2014; Drancourt et al., 1998). However, many bioarchaeological traces of past pandemics remain materially inscrutable.

These issues are layered on top of the material limitations (e.g., sample biases) common to all skeletal analyses (DeWitte & Stojanowski, 2015; Ortner, 1991; Wood et al., 1992). Indeed, as several researchers have explored, primarily for the 14th century Black Death and the 1918 influenza pandemic, these material limitations (e.g., heterogeneity in frailty and selective mortality) may be uniquely affected by some aspects of pandemic infectious disease (e.g., DeWitte, 2015, 2018; Kelmelis & DeWitte, 2021; Wissler, 2021). Amongst other factors, these include greater representation of socially marginalized communities in excavated assemblages and historical documented collections (vs. more privileged communities), even as these communities have been consistently more negatively impacted by pandemics (e.g., elevated mortality) (Roberts & Cox, 2003; Wissler, 2021). Worse overall health, heightened frailty and prior frailty, elevated rates of comorbidities, and other related vulnerabilities amongst these communities may further alter heterogeneity in frailty and selective mortality to pandemic infectious disease, excess mortality, and other disease sequelae relative to more buffered, privileged communities. These can be complex to identify and reconstruct in assemblages and can require nuanced and heavily contextualized analytical accommodations in paleopathological and bioarchaeological interpretations (e.g., lesion frequencies) of frailty, resiliency, disease burden, mortality, and other related concerns within past pandemics (Crespo & Lawrenz, 2015; DeWitte 2015, 2018; Kelmelis & DeWitte, 2021; Larsen & Crespo, 2022; Wissler, 2021).

In addition, biohazard and bioterrorism concerns associated with organism resurrection and/or de-extinction (El-Sayed & Kamel, 2020; Jones, 2014; Shatilovich et al., 2018; Worrall, 2019) can limit bioarchaeological work on sites associated with past pandemics as well as biomolecular research on high-risk pathogenic organisms (Wurtz et al., 2014). Further, in terms of scope, most bioarchaeological and paleopathological analyses are regionally specific, with only a few monumental studies spanning whole continents (e.g., Cohen & Armelagos 1984; Steckel & Rose, 2002; Steckel et al., 2019). Finally, relatively slow development of "big data" tools and approaches may have unintentionally hindered pandemic studies due to the necessity of collating and assessing diverse, global datasets from various disciplines.

Ideological and conceptual issues may also underlie the relative rarity of pandemic-scale investigations (Dimka et al., 2022). This issue may be attributable to the need for historical evidence to fully contextualize a relatively recent pandemic, such as the 1918 influenza pandemic, even as biological anthropology has consistently given low priority and scholarly value to work on recent historical populations (Dimka et al., 2022; see Roberts & Cox, 2003). Finally, until the COVID-19 pandemic, the global community had also not witnessed the socio-political and socio-economic disruption that a pandemic could wreck in over a century, perhaps driving scholarly disinterest. However, as biological anthropology research inspired by COVID-19 highlights (e.g., Robbins Schug & Halcrow, 2021), pandemics may become a major emergent research focus (Dimka et al., 2022).

Here, we explore research on past pandemics within bioarchaeology, paleopathology, and molecular anthropology that employs a biocultural approach, especially relative to social inequality. Further, we highlight research that employs intersectionality, ecosocial theory, and syndemic theory, sometimes in tandem with the biocultural approach, to unpack relationships between social inequality and pandemics. We demonstrate that these tools generate synthesized insights and broadened linkages between systemic, structural sources of social inequality and heightened morbidity, comorbidity, and mortality. To illustrate this, we include a case study assessing how the 1918 influenza pandemic impacted mortality within a socially vulnerable population: Individuals institutionalized at the Mississippi State Asylum (MSA) (CE 1855–1935) in Jackson, MS.

1.1 | The biocultural approach, intersectionality, and ecosocial and syndemic theory

Pandemics both past and present can be effectively and holistically understood through the synthesis of several related frameworks, approaches, and theories that are drawn from anthropology and the larger social science, as well as public health and the larger health sciences. Here, these are the biocultural approach, intersectionality, and ecosocial theory, and syndemic theory, specifically. As we demonstrate in the following sections, that is because these tools, when used alone but especially when integrated, enable identification of the pathogens that spark pandemics and interact with and affect human biology while also being transmitted and mediated by a variety of social behaviors, structures, and processes. The biocultural approach offers insights into this key interface. But further, integrated theoretical interpretation can highlight the social, economic, demographic, epidemiological, and political identities, systems, structures, and processes that intersect to distribute risk of infectious disease and its outcomes unevenly within and across human populations over time. In particular, intersectionality names the identities and their multiplicative power, illuminating pathways by which pandemics can entrench and harden existing health disparities (e.g., Lopez et al., 2021; Van Dorn et al., 2020) or, sometimes, help to loosen and ameliorate them (e.g., DeWitte, 2014a, 2014b). Notably though, while the biocultural approach speaks to the biosocial interface and intersectionality deepens our framing of the social, neither approach[3] provides guidance regarding the environment and the way in which humans mutually affect and are affected by environments around them. Bringing in ecosocial theory provides this guidance, while also exposing the diverse pathways and conditions through which inequality can impact health and susceptibility to pandemics and their outcomes. Finally, syndemic theory brings us full circle—back to the biosocial interface, now that the "social" has been more fully theorized—to address the biological-biological interface through which the distribution of pandemic mortality, morbidity, and comorbidity often mimics and exacerbates existing distributions of disease within a population. To better unpack this synthesis, the following sections define the biocultural approach, intersectionality, ecosocial theory and syndemic theory; introduce their application to pandemics; and begin to clarify how they can be integrated for application to past pandemics.

First, definitions of the biocultural (aka. biosocial) approach vary over time and across applications (Zuckerman, 2018). We define and employ[4] the approach as emphasizing the multifaceted, bidirectional, and inextricable interactions between biological, cultural, and environmental features, conditions, and processes within human phenomena (Blakey & Watkins, 2021; Zuckerman, 2018). Within this nexus, phenotypic plasticity—physiological, behavioral, and morphological changes in the phenotypic expression of a genotype in response to environmental change—and consequently human biology writ large (Pigliucci, 2001), are molded by social influences (e.g., political and economic) (Blakey & Watkins, 2021; D'Ambrosio & Colagè, 2017). These influences are in turn shaped by biological and environmental features and constraints (Holder et al., 2021). These include the adequacy—or inadequacy—of available diets, climate, exposure to pathogens, political and economic systems and structures, and societal variation in access to resources and exposure to stressors (Worthman & Costello, 2009; Zuckerman, 2018). Thus, layered social identities, environmental settings, genetics, biology, and epigenetics can affect health and disease outcomes, and biological and social factors can be intergenerationally transmitted (Castro, 2018; Hoke & Schell, 2020). As we demonstrate, these characteristics make the biocultural approach especially useful for studying past pandemics. In particular, layering historical biocultural perspectives allows insight into how generations of inequalities can become embodied and contribute to asymmetrical risk and rates of infection within populations (van Doren, 2021).

Second, intersectionality, which originated in Black feminism and critical race theory, refers to the simultaneous, synergistic interaction of categories of social identity and forms of systemic oppression (Combahee River Collective, 1983; Crenshaw, 1989; Davis, 1983; DeWitte & Yaussy, 2020; hooks, 2014 [1984]; Springer et al., 2012). Categories of social difference and identity include any aspects of identity that generate power (e.g., race, class, and gender), and associated forms of oppression (e.g., racism, classism, and sexism). As intersectionality recognizes that individuals have overlapping identities and multiple social statuses (Cole, 2009), it provides a lens through which the origins and interactions of power dynamics can be examined (Collins, 2015; Crenshaw, 1989; hooks, 2014). Intersectionality

also enables consideration of how these interactions shape social inequalities and thus health, well-being, and disease, as well as access to resources, exposure to stressors, amongst others. Critically, unlike conventional approaches to social inequality, which treat identity categories and systems of oppression as independent and exclusive, intersectionality proposes that they are mutually constitutive, reflecting overlapping structures of power, inequality, and discrimination (Bowleg 2012; Cole, 2009; Collins, 2015; Crenshaw, 1989; Davis, 1983; DeWitte & Yaussy, 2020; hooks, 2014 [1984]).

Third, ecosocial theory, which is commonly used in social epidemiology, is designed to explain social inequalities in health (Krieger, 1994, 2001; Worthman & Costello, 2009). It proposes that humans biologically embody exposures arising from societal and ecological contexts, producing population rates and distributions of health and disease (Krieger, 2012). These patterns arise through socially patterned, exposure-induced disease-causing pathways, that are mediated by physiology, behavior, and gene expression (e.g., epigenetics) and which affect the development, growth, bodily regulation, and death of individuals (Worthman & Kohrt, 2005). These interactions culminate in differential exposure, susceptibility, and resistance to disease, disability, and death (Krieger, 2012). Ecosocial theory accounts for the complex disease interactions that occur within individuals in different contexts, and how these interactions aggregate into health disparities (Krieger, 1994, 2001; Worthman & Costello, 2009). Importantly, ecology is central to ecosocial theory because of its concerns with scale, organization-level dynamic states, and mathematical modeling. These enable quantitative examinations of interactions between and within the assessed levels of causality and variables (Krieger, 2001; Peterson & Parker, 1998; Worthman & Kuzara, 2005). Notably, ecosocial theory opposes the still employed theories and frameworks that conceptualize disease etiology and health disparities as primarily innate, such as from biological race (e.g., raciogenetics) (Krieger, 2012).

Lastly, syndemic theory is increasingly becoming a mainstay in public health and medical anthropology. Singer (2009: xv) defines a syndemic (i.e., synergistic epidemic) as the concentration and deleterious interaction of two or more diseases or other health conditions in a population, especially as a result of social inequality. Syndemic theory emphasizes an encompassing experience that considers all the health and social problems suffered by an individual or community, not just the effects of a single disease (Singer, 2009: xiv). It posits that harmful social realities concentrate diseases in particular contexts (Singer, 2009; Tsai & Venkataramani, 2016). Where intersectionality highlights how interactions between multiple social identities increase disease risk and exacerbate health disparities, syndemics conceptualize disease interactions across "biological-biological" and "biological-social" interfaces. The biological-biological interface refers to co-morbid diseases interacting within the body to intensify disease burden, resulting in enhanced lethality, evolution of novel pathogens, and reduced immunity (Singer, 2009). In the biological-social interface, individuals with co-morbidities interact with their social environments so that behavior reciprocally shapes and is shaped by disease (Singer et al., 2020).

As we demonstrate below, these diverse approaches and theories can be employed alone or integrated in research on past pandemics. The biocultural approach situates these core concepts within a framework that necessitates consideration of the biological and cultural underpinnings of these phenomena. Integration with intersectionality names the structural, systemic, and multi-layered forms of oppression that disproportionately impact socially marginalized populations, further theorizing the "cultural" component of the biocultural approach. Ecosocial theory then links the biocultural, intersectional understanding of pandemic infectious disease to ecology in ways that are vital to examining the pathogens at the root of pandemics. Finally, the inclusion of syndemic theory affirms the importance of the biocultural interface but also draws attention to the biological - biological interface of human exposure to and endurance of multiple diseases that exacerbate each other's effects. Accordingly, interpretations through syndemic theory are especially key in understanding population distributions of mortality, morbidity, and comorbidity in both past and present-day pandemics.

1.2 | Biocultural research on present-day pandemics

Importantly, not all analyses of pandemics—past and present-day—that employ the ideas embedded in these theories and approaches do so *explicitly*. There are also several approaches, such as One Health (e.g., Littleton et al., 2022),[5] that are commonly used and substantially overlap with many of them. This lack of clarity or explicitness makes such research difficult to identify for review purposes. More potently, inexplicit use of the biocultural approach diminishes the possibility of their results being used to inform policymakers for public health responses and pandemic preparedness.

Most biocultural analyses of pandemics arise within medical anthropology and focus on recent and ongoing pandemics, such as HIV/AIDS (e.g., Armelagos et al., 1990; Chapman, 2020; Friedler, 2021; Gaudillière et al., 2020; Kwan et al., 2018; Ruthven, 2017; Sellen & Hadley, 2011; Smith, 2012; Young et al., 2014). These studies have mostly focused on non-biomedical health systems, culturally based understandings of disease (e.g., Inhorn & Brown, 1990; Joralemon, 2017), and the importance of local contexts in epidemic control (Kleinman et al., 2008). An increasing number assess the indirect epidemiologic effects of pandemics on well-being, including growth and development, mental health, and intergenerational and epigenetic effects, often through the Developmental Origins of Health and Disease (DOHaD) paradigm[6] (e.g., Bogin & Varea, 2020; Gibb et al., 2020; Kim, Mohamed et al., 2022; Kim, Nyengerai et al., 2022). Such analyses encourage consideration of how pandemics can affect population-level patterns of health and disease through pathways other than morbidity and mortality from the causal infection. Increasingly sophisticated applications of DOHaD to skeletal samples (e.g., Brickley et al., 2020; Gowland, 2015), and work reviewed below on the bidirectional effects of the 14th century Black Death, mean that bioarchaeologists and paleopathologists are well

positioned to apply similar assessments of pandemic-related stress to skeletal samples.

Most biocultural studies of pandemics also focus on dialectical interactions between pandemics and social inequality (Farmer, 1996, 2004a, 2004b, 2006; James, 2010; Vlassoff & Manderson, 1998). Many employ the social determinants of health (SDH), a framework for comprehending linkages between social factors that include discriminatory values and behaviors (e.g., racism) and health system biases (e.g., quality of care), in producing household, community-level, and population-level health, including differential exposures and vulnerabilities to disease, disability, and injury (CDC, 2010; Marmot, 2005; Sen & Östlin, 2010: 3). Problematically, the SDH are often used reductively, categorizing and simplifying linkages between socio-economic conditions and patterns of health and disease (Yates-Doerr, 2020). For biocultural analyses of past pandemics in bioarchaeology and paleopathology, we note that applying the SDH may also be problematic because specific social factors (e.g., socioeconomic status [SES]) can be difficult to reconstruct with certainty for skeletal individuals and samples, and are often approximated and simplified. This can obscure key findings that might help inform public health responses and pandemics preparedness. Overall, studies assessing social inequality during pandemics have focused on differential vulnerability to morbidity and mortality (heterogeneity in frailty) relative to gender, social race, ethnicity, social support and exclusion, SES, and, increasingly, sexual and gender minorities (e.g., Bentley, 2020; Brewis et al., 2020; Mamelund et al., 2019).

1.3 | Biocultural research on past pandemics

Overall, very few analyses of past pandemics or large-scale epidemics within bioarchaeology, paleopathology, and molecular anthropology explicitly employ a biocultural approach, though there are many examples of implicit use (e.g., Newman & Hodson, 2021). This is likely part of the overall scarcity of biocultural work in these fields, with relatively few researchers choosing to ask explicitly biocultural research questions, and especially in paleogenomics, being aware of the approach. Short publishing formats in paleogenomics also limit authors' ability to explore social, cultural, and environmental disease interactions, though a few researchers have persevered with biocultural paleogenomic analyses of past infectious disease events (e.g., Bos et al., 2011, 2014; Joseph & Lindo, 2022; Marciniak, 2016; Marciniak et al., 2018; Mordechai et al., 2019; Vågene et al., 2018).

Amongst the explicit biocultural works, some have taken advantage of the biocultural approach's time-depth and evolutionary perspective through cross-field analyses incorporating bioarchaeological data into medical anthropological investigations of recent pandemics. For example, Burke (2011) integrated ethnographic data on public health interventions against the late 20th century resurgence of TB with bioarchaeological evidence of the evolution of TB within human populations. Doing so enabled a multi-scalar, holistic analysis of a pandemic that could be broadly applied to other recent historical pandemics.

Biocultural analyses of past pandemics that exclusively use bioarchaeological, paleopathological, biomolecular, and historical evidence face distinct challenges. In addition to the aforementioned impediments, challenges accrue from the intervening time, limited availability and completeness of data, and inherently incomplete understandings of complex past phenomena, conditions, and processes. Building from Dressler (1995), Dufour (2006), Goodman (2014), Zuckerman (2018), Wiley and Cullin (2016), and Hoke and Schell (2020), these issues are exacerbated by the complexities of applying the biocultural approach. Amongst these is the difficulty of reconstructing theories of culture[7] and the means of operationalizing and measuring the impact of culture and culturally defined variables (e.g., gender and power dynamics) on health and other biological outcomes in ways that are accurate for past populations, as well as valid and scientifically replicable. This is especially difficult when these variables are composed of multiple, intersecting social, ecological, and economic components. Doing so requires context and condition-specific ethnographic, archeological, and/or historical data, which may be incomplete or absent, whether for recent pandemics (i.e., 1918 influenza pandemic) (Short et al., 2018) or those deeper in antiquity (e.g., the Black Death, Post-contact Indigenous population decline) (Bos & DeWitte, 2022; DeWitte, 2018; Jones et al., 2021). The highly context-specific effects of pandemics can further magnify this issue (e.g., Sattenspiel et al., 2019). Researchers must also contend with the complex biosocial and human-environmental effects of multidimensional concepts and processes (e.g., social inequality, intergenerational effects, human mobility, and population interaction) and their interactions within pandemics (Vlok & Buckley, 2022). Further, they must assess these effects in terms of pathogen ecology and biology, which can quickly evolve to outpace medical and social responses (e.g., evolution of virulence and antimicrobial resistance). Comprehending the complex interactions that occur amongst various aspects of human biology and culture, as well as between humans, animals, and their environments, and humans with pathogens, requires researchers to identify, define, and measure many different causal pathways. Doing so can be very challenging in practice, especially as we critically reflect on and work to decolonize established processes of knowledge production.

However, analyses which have overcome these challenges reveal that the biocultural approach creates unique possibilities for deeper understandings of past pandemics, as well as large-scale epidemics, and their diverse sequelae, such as physical impairment (e.g., Battles & Gilmour, 2022; Cameron et al., 2015; Crespo & Lawrenz, 2015; DeWitte & Slavin, 2013; Kelmelis & Dangvard Pedersen, 2019; Kelmelis & DeWitte, 2021; Larsen & Crespo, 2022). Within this relatively small body of work, a range of pandemics have been assessed, including those involved in post-contact Indigenous depopulation in the Americas, climate change, the Black Death, and the 1918 influenza pandemic.

1.4 | Post-contact Indigenous population decline in the Americas

Of all biocultural research on past pandemics, analyses of post-contact Indigenous population decline in the Americas have arguably

been the most impactful for realizing the promises of the biocultural approach: identifying ultimate causes within pandemics, challenging and deconstructing simplified and biologically deterministic narratives about infection and denaturalizing social inequality. For several centuries, anthropologists and historians attributed the vast decline of Indigenous populations in the centuries following first contact (Denevan, 1992; Stannard, 1993; Ubelaker, 1988) almost exclusively to the effects of infectious diseases introduced by European contact. Specifically, they proposed that the decline was caused by "virgin soil" epidemics and pandemics of acute infectious diseases that were unintentionally imported from the Old World (e.g., smallpox, cholera, influenza, and measles), to which Indigenous populations had no acquired immunity or genetic resistance (Crosby, 1976; Dobyns, 1993). These arguments have become influential, mainstream (Diamond, 2013), and naturalize Indigenous depopulation and associated societal decentralization and dispossession (e.g., acculturation and widespread substance abuse). They position it as a phenomenon beyond human control, effectively removing Indigenous people's agency within American history.

Recently, biocultural analyses have challenged and complicated this linear explanation (Cameron et al., 2015). Drawing upon extensive archeological and documentary evidence of systematic genocide; enslavement; forced labor, migration, and acculturation (e.g., Residential Schools); and other depredations, researchers have demonstrated that social conditions amplified the impacts of biological factors (Wade, 2020). Specifically, structural violence—not infectious disease—was ultimately causal to Indigenous population decline (Cameron et al., 2015). Incorporating syndemic theory, researchers have proposed that structural violence intersected with infectious disease to create a syndemic, rendering Indigenous populations more vulnerable to infectious disease, which fueled population decline and impeded societal and demographic recovery (Barrett et al., 2022; Cameron et al., 2015). Notably, this research is powered by contextualized, regional-level analyses, which enables detection of settings where infection was more or less destructive (Hull, 2015). Combined, this research denaturalizes Indigenous population decline, repositioning it as an effect of European Colonialism (i.e., genocide) (Wilcox, 2010). This has restored Indigenous agency in American history while foregrounding the moral consequences of Colonialism (Kelton et al., 2015).

Importantly, the same syndemic of infectious disease and structural violence persists for present-day Indigenous populations. Dávalos et al. (2020) highlighted the time-depth of the underpinnings of structural violence in facilitating the disproportionate burden of infectious diseases for socially marginalized populations, including Indigenous populations. Indigenous American populations continue to face disproportionate frequencies of epidemic infection, including COVID-19 and other recent pandemics, as well as NCDs, such as diabetes (Braveman et al., 2011; Jiang et al., 2018; Power et al., 2020). Through a biocultural approach and application of syndemic and ecosocial theory, these effects are revealed to be synergistic with the persistent biosocial and medico-legal effects of colonization, which are endured as interpersonal and structural discrimination, intergenerational effects, and historical trauma (Bear et al., 2018, 2019; Power et al., 2020; Singer and Rylko-Bauer, 2021). This research is critical for revealing remedies to the long-running public health crisis for Indigenous Americans, including active recognition and support of Indigenous sovereignty and land rights, restructuring health care systems, and building cultural determinants of health into health policy, practice, and research (Dávalos et al., 2020; Gamble et al., 2021; Power et al., 2020; Salmon et al., 2019; Singer and Rylko-Bauer, 2021).

1.5 | Infectious disease and climate change

Researchers combining biocultural and evolutionary approaches in studying health and disease can generate cross-cultural time depth that can elucidate complex human behavioral responses to climate change, including associated pandemics (Nystrom & Robins Schug, 2020; Zuckerman & Dafoe, 2020). Climate change affects health and disease directly and indirectly. Direct impacts include mortality from extreme weather events (e.g., heatwaves), while indirect impacts are highly complex, encompassing social, economic, ecological, physical, demographic, and political conditions and forces, overlain with stressors from climatic fluctuations (McMichael, 2012). Throughout the Holocene, climate change has been mostly indirectly associated with adverse health impacts (McMichael, 2012), including undernutrition (e.g., Robbins Schug & Blevins, 2016; Snoddy et al., 2020), chronic and/or episodic physiological stress (stress) (e.g., Binder, 2014; Williams & Larsen, 2017), and starvation (e.g., Robbins Schug & Goldman, 2014), as well as epidemics and pandemics, such as through changes in the distribution of vector species and pathogens (e.g., *Anopheles* mosquitoes and malaria) (e.g., Bourbou, 2020; Robbins Schug et al., 2013; Zuckerman & Dafoe, 2020). However, as Zuckerman and Dafoe (2020) detail, numerous material issues impede drawing direct associations between infection and climate change in the past, such as that climate data is rarely available on sub-hemispheric level. Clear associations between climate change and infectious disease are thus largely only available for the past 500 years (McMichael, 2012; Zuckerman & Dafoe, 2020).

Within this span, epidemics and pandemics have largely been linked with undernutrition, starvation, crowding, and conflict either caused or exacerbated by climate change (McMichael, 2012). These include the Little Ice Age in Europe and Asia (c. CE 1300/1650–1850) (Koepke, 2016; Koepke & Baten, 2005) and the decade-long cool, wet period that precipitated the Great Famine (CE 1315–1317/22) in Europe, which greatly increased vulnerability to the Black Death (DeWitte, 2014a, 2020; DeWitte & Slavin, 2013). These associations partially exist because stress, undernutrition (e.g., Niacin deficiency or pellagra), and famine can increase susceptibility to infectious disease through immune suppression (Himmelgreen et al., 2020). Further, biocultural analyses integrating historical and skeletal evidence have found that when these conditions, as well as the infectious diseases that occur in syndemic with them, have arisen in response to climate change, they are disproportionately born by socially marginalized populations (e.g., the poor, institutionalized) (DeWitte, 2020; Miller et al., 2020). Importantly, biocultural analyses of past infectious

disease events and climate change could reveal adaptive strategies employed in the past that could be useful for mitigating some problems moving forward (McMichael, 2012; Miller et al., 2020; Nystrom & Robins Schug, 2020; Zuckerman & Dafoe, 2020). Given escalating and amplifying climate change from anthropogenic causes (Magnan et al., 2021), evidence that climate change in the past has interfered with food security and influenced the risk of infection should warn us that anthropogenic climate change will profoundly impact human health, especially for socially marginalized populations.

1.6 | The Black Death

The Black Death (CE 1346–1353) has generated substantial biocultural research, both implicit and explicit. This pandemic caused the death of 75–200 million people throughout Eurasia and North Africa, and had profound short and long-term economic, environmental, demographic, and social impacts (Snowden, 2019). Substantial work within molecular anthropology has focused on confirming and evolutionarily exploring the Black Death's causal pathogen, *Y. pestis*, via ancient DNA (aDNA) (Bos et al., 2011; Keller et al., 2019; Morozova et al., 2020; Namouchi et al., 2018; Spyrou et al., 2016), of which several studies are implicitly biocultural. Bos et al. (2011) and Bos and DeWitte (2022) interpret the increased virulence of *Y. pestis* during the Black Death relative to modern strains as the result of synergy between genetic, social, environmental, immunological, and host/vector dynamics rather than of specific or unique genetic features. Namouchi et al. (2018) used an implicit biocultural framework to identify pathogen recirculation and transmission routes, explaining pathogen continual re-emergence and specific evolutionary relationships. While this implicit use is impactful for clarifying specific pandemic events and biological interactions, researchers have only just begun to delve into how both pathogen/pandemic and societal aspects (e.g., structural violence) have informed one another during the Black Death or other past pandemics (Bos & DeWitte, 2022).

This relative scarcity constricts the potential insights into biosocial interactions that molecular evidence could grant, and which so many biocultural analyses of past and present pandemics have demonstrated are critical to public health responses and pandemic preparedness. Crespo and Lawrenz (2015), for instance, note the continued lack of integration between assessments of the social and biological causes of heterogeneity in frailty from plague during the Black Death. They emphasize that, like social behavior, the human immune response to infectious disease, both acquired and innate, is remarkably heterogeneous and plastic (Crespo & Lawrenz, 2015); uniformitarian principles and bioarchaeological findings suggest that the same was true in the past. Observed differential morbidity and mortality thus reflects differential immune capacities amongst past individuals and across communities, which are directly linked to social and environmental pressures, including climate change, associated agricultural failures, and nutritional stress (e.g., Jang & Serra, 2014) and systemic health (Crespo & Lawrenz, 2015). Factors including host-pathogen coevolution, pathogen evolution in response to environmental conditions, anthropogenic environmental change, and the impacts of pathogens on human biology and behavior, cause Crespo and Lawrenz to call for a more integrative biocultural approach to modeling heterogeneity in frailty during the Black Death, which incorporates historical and ecological contextual data into immunological models.

Existing bioarchaeological and paleopathological research on the Black Death is prime for such an integration with molecular evidence and immunological findings (Bos & DeWitte, 2022; Larsen & Crespo, 2022). This research, while mostly implicitly biocultural, has translated extensive historical contextual data into sophisticated understandings of population health, heterogeneity in frailty, and selective mortality surrounding the Black Death (e.g., DeWitte, 2009, 2010, 2014b, 2015; DeWitte & Hughes-Morey, 2012). This work strategically considers a wide range of social, environmental, and economic variables relative to the pandemic, including social identity (e.g., gender and SES), inequality, and stratification, climate change, and urbanization at the population level and across the life course (e.g., Bos & DeWitte, 2022; DeWitte, 2015; Godde et al., 2020; Yaussy, 2022). Combined, these studies have revealed that intersecting social and economic inequalities shaped the course of the pandemic (Wade, 2020). To do so, several bioarchaeological studies of the Black Death have employed intersectionality (Yaussy, 2022); DeWitte (2018), for example, assessed the effects of sex-based variation in sensitivity to stressors, nutritional stress and pubertal timing, gender-based variation in access to dietary resources, and combinations of these factors relative to survivorship for the plague. Importantly, the models (e.g., hazard analysis) and frameworks employed by these researchers are not specific to the Black Death (see Yaussy, 2022); modified for different contexts, they can serve as templates for integrating social context, including social inequality, into intersectional interpretations of frailty, mortality, and health in other historical pandemics.

1.7 | The 1918 influenza pandemic

The 1918 influenza pandemic makes clear that historical evidence provides a crucial layer of contextual information about pathogen biology, and human biology and behavior, for historic epidemics and pandemics; with intervening time, the availability and reliability of such information decreases. Perhaps because of this evidentiary advantage, numerous implicitly and explicitly biocultural analyses have focused on the 1918 pandemic. Many of these have explored pathogen circulation and transmission routes, heterogeneity in frailty, and selective mortality and morbidity within specific social contexts. Overall, they have detected substantial intrapopulation variation during the pandemic, revealing how local factors (e.g., population density and urban vs. rural environments), poverty, and cultural, demographic, and epidemiologic differences (e.g., exposure history) can drive infection dynamics (e.g., DeWitte & Wissler 2022; Mamelund et al., 2013; Paskoff & Sattenspiel, 2019; Sattenspiel, 2011; Tripp et al., 2018; van Doren & Sattenspiel, 2021). For instance, socially marginalized

communities, such as Indigenous and Black Americans, institutionalized populations, and low SES and poor communities, suffered disproportionately high morbidity and mortality (e.g., Dimka & Mamelund, 2020; Gamble, 2010; Mamelund, 2006). Syndemic theory may explain some of this variation and disparity; socially marginalized communities, for example, were more likely to experience comorbidities (e.g., TB), as well as social, ethnic, and environmental conditions, such as systemic racism and racial inequalities in health care (e.g., segregated hospitals in the US), that exacerbated their vulnerability to influenza (Herring & Sattenspiel, 2007; Sattenspiel & Mamelund, 2012; Tripp et al., 2018). Cumulatively, this scholarship shows how social inequality drove disparities within pandemic influenza in recent history (van Doren, 2021).

Amongst other methods innovations, researchers have used agent-based modeling to unpack these dynamics within some richly documented community contexts (e.g., Carpenter & Sattenspiel, 2009; Sattenspiel et al., 2019). While these models must be calibrated to the social and historical context under study, they enable empirical investigation of how complex factors and stochasticity, including social, economic, and geographic conditions, demography, and mobility influence pathogen spread. Further, once validated, the resulting models can be applied to other directly transmitted pathogens and small communities. When paired with regionally specific historical contextual data, these and similar tools are valuable for modeling and predicting pathogen transmission in small communities (Ramirez et al., 2021; Sattenspiel et al., 2019), which is critical for effective, tailored public health responses and pandemic preparedness.

While research on the 1918 pandemic has been dominated by studies employing historical evidence, some recent, implicitly biocultural work has integrated skeletal with historical evidence. For example, Wissler (2021) combined skeletal data and closely associated historical contextual evidence to explore frailty (e.g., comorbidity) relative to pandemic mortality. The 1918 pandemic also holds disciplinary significance for molecular anthropology and paleogenomics, as the first ancient pathogen genome recovered from an historical human tissue sample derived from an influenza patient (Taubenberger et al., 1997). Importantly, subsequent reconstruction of the 1918 virus (Tumpey et al., 2005), including relative to the social determinants of health (e.g., Humphreys, 2018), has been used to directly inform public health understanding and responses to recent outbreaks (e.g., 2009 Swine Flu and SARS) and the COVID-19 pandemic.

1.8 | Case study: The 1918 influenza pandemic and related mortality within a socially vulnerable population of individuals institutionalized at the Mississippi State Asylum (MSA)

With the above context and theoretic understanding in mind, we apply the biocultural approach, ecosocial theory, syndemic theory, and intersectionality to understand the 1918 influenza pandemic's impact on mortality at the MSA. We address how mortality from respiratory infection, specifically influenza, intersected with social identity, specifically patient social race, age, and sex, amongst 2258 patient deaths at the MSA between 1912 and 1925. Social race was a primary social determinant of health in the early 20th century Jim Crow southern US (Sloan et al., 2010), and age was a well-established risk factor of the influenza pandemic (Morens & Fauci, 2007). As MSA records include primary and secondary causes of death, we assess mortality from single causes (e.g., influenza) and in syndemic. The analytical models we employ examine relationships between types of respiratory mortality and the likelihood of death during the pandemic, controlling for key demographic characteristics (e.g., age) but also assessing intersections between these identities. Based on available data, we aim to unpack some of the complexities within pandemics traditionally overlooked by strictly biological or social investigations. Our goal is to elucidate ways in which the biocultural approach can inform pandemic preparedness and broader public health measures.[8]

2 | MATERIALS AND METHODS

2.1 | Mortality patterns during the 1918 influenza pandemic

During the 1918–1919 influenza pandemic, approximately one-third of the global population—500 million individuals—was likely infected with the causal H1N1 virus strain. This strain had an estimated ≈10% fatality rate, resulting in approximately 50–100 million deaths, including ~675,000 in the US (CDC, 2019; Krishnan et al., 2020). Approximately 50% of these deaths were in young adults (20–40 years) (Morens & Fauci, 2007; van Wijhe et al., 2018). Elevated young adult mortality may have been attributable to absent cross-reactive immunity from previous exposure to pandemic influenza (Gagnon et al., 2013; Short et al., 2018; Worobey et al., 2014); a dysregulated hyperinflammatory immune response (Morens & Fauci, 2007; Short et al., 2018); undernutrition, overcrowded medical facilities, and poor hygiene, exacerbated by the economic and social devastation of World War I (WWI), which may have heightened susceptibility to secondary respiratory infections (e.g., pneumonia) (Brundage & Shanks, 2007); or a combination of these factors.

As noted above, the 1918 influenza pandemic exerted disproportionate, though varied, harm for socially marginalized communities (Mamelund et al., 2020). Amongst these, Black and/or African American (AA) communities experienced higher influenza case fatality than did White and/or European American (EA) communities (Gamble, 2010; Mamelund et al., 2020). This may have been attributable to the effects of poverty, systemic racism, and race-based structural violence. These may have created syndemics of undernutrition and comorbidity (e.g., TB and parasite infections), and exacerbated these communities' social and biological vulnerability (Franklin & Wilson, 2020) to influenza. Social inequalities may have placed Black and/or AA individuals at higher risk than White and/or EA individuals of developing—and dying from—secondary infections (e.g., pneumonia) (Økland & Mamelund, 2019). The intersections of social race and institutionalization during the 1918 pandemic in the US are not yet understood

FIGURE 1 The Mississippi State Asylum, known as the State Hospital for the Insane in the early 1900s, undated photograph. Photo credit: Mississippi Department of Archives and History

(Zuckerman et al., 2022). Nonetheless, diverse evidence suggests that segregation caused Black and/or AA communities to endure reduced access to healthcare and poorer-quality healthcare in the late 19th and early 20th centuries, resulting in worse health outcomes, even as Black and/or AA communities, including in Mississippi, resisted these forms of structural violence through independent development of community-based medical institutions (de la Cova, 2011, 2014; Franklin & Wilson, 2020; Gamble, 2010; Sano, 2010). These conditions may have directly contributed to the higher case fatality observed during the pandemic (Humphreys, 2018; Mamelund et al., 2020).

2.2 | The 1918 influenza pandemic at the Mississippi State Asylum (MSA)

The MSA provided care for approximately 30,000 individuals diagnosed with acute and chronic conditions (CE 1855–1935) in suburban Jackson, MS (Figure 1) (Lampton, 2017). Most patients were poor to laboring-class Mississippians, though some were middle class to elite (Mitchell, 1901, 1933). Throughout the 19th century, most patients were White and/or EA, but by the early 20th century the number of Black and/or AA[9] patients rose to be approximately half of patients. Living conditions varied throughout the MSA's operation, with patient health worsened by insufficient infrastructure and housing in the early 20th century, resulting in progressive overcrowding and worsening hygiene (Mitchell, 1919, 1926). Superintendents' Biennial reports maintain that Black and/or AA patients received the same care as White and/or EA patients but confirm that these burdens fell disproportionately on the former; the MSA had gender and race-segregated wards (Figure 2). By the 1920s, for example, Black and/or AA patients had effectively no outdoor leisure because of overcrowding and the importance of segregation (Mitchell, 1926). MSA records also report increased, disproportionate mortality amongst Black and/or AA patients in the early 20th century, particularly from TB and pellagra. Superintendents' reports maintained that Black and/or AA patients received the same care as White and/or EA patients, instead attributing these patterns to biologically based "racial influences" that led to greater vulnerability to TB, though also to overcrowding and poor diet prior to admission. Many Black and/or AA patients also died soon after admission, which Superintendents ascribed to insalubrious conditions in the local jails where Black and/or AA patients often awaited admission (Mitchell, 1919; Stewart, 1911). Indeed, undernutrition and endemic TB were ubiquitous within Black and/or AA communities in the early to mid-20th century in the American South, including Mississippi (de la Cova, 2011, 2014; Franklin & Wilson, 2020; Sano, 2010).

Both Mississippi and the MSA were hard hit by the 1918 pandemic (DeCesare Ross, 2017; Leathers, 1918; MSBH, 1919). Early 20th century Mississippi was primarily agricultural, with few industrial centers, and rural healthcare systems were underfunded and generally inadequate, particularly for Black and/or AA communities, despite their efforts to develop independent systems (Mitchell, 2014; Sano, 2010). Critically, WWI heightened these strains, stretching health care systems and medical professionals even thinner (DeCesare Ross, 2017; MSBH, 1919; Sano, 2010). The population was majority laboring-class to poor, with consequent poor health (Mitchell, 2014) and intensifying Jim Crow-era systemic racism and race-based structural violence further eroded health and health care within Black and/or AA communities (Franklin & Wilson, 2020; Sano, 2010). The pandemic, which caused over 6000 deaths in Mississippi (1918–1919), also had elevated transmission and higher mortality rates in Black and/or AA communities (DeCesare Ross, 2017; MSBH, 1919).

Influenza arrived at the MSA in 1918, despite staff attempting to safeguard patients by refusing admissions and visitors. Superintendents had long anticipated that if an epidemic "invade[d]" the MSA, mortality would be high because of its suburban location, distant from "sufficient medical assistance and help" (Mitchell, 1899). Indeed, Superintendent Mitchell (1919:12) wrote that "617 patients suffered" from influenza and "67 of them died" but "when I recall the fact that at this time a large majority of my employees were sick at the same time, the physicians, the nurses, the attendants, the cooks, the baker and dairyman, I feel fortunate that more did not die."

2.3 | MSA death certificates

Death Certificates (DC)[10] represent a subset of the 3697 patient deaths that occurred between 1912 and 1925, as they exist only for the 2341 patients who both died *and* were buried at the MSA; the remaining 1356 patients were buried elsewhere. DC record the name, sex, age, social race (race), cause of death (CoD), death date, and duration of stay for each patient. DCs with missing data for any independent or dependent variable of interest (i.e., CoD, age, sex, race, and death date) were excluded. This process yielded a final analytic sample size of 2258 deaths, which is consistent with the demographic breakdown of the total 2341 death certificates. Demonstrating this, most patients in the complete sample used here ($N = 2258$) and total (2341) were Black and/or AA: 85.2% in the total and 85.2% in the complete (14.8% are of White patients) (Zuckerman et al., 2022).

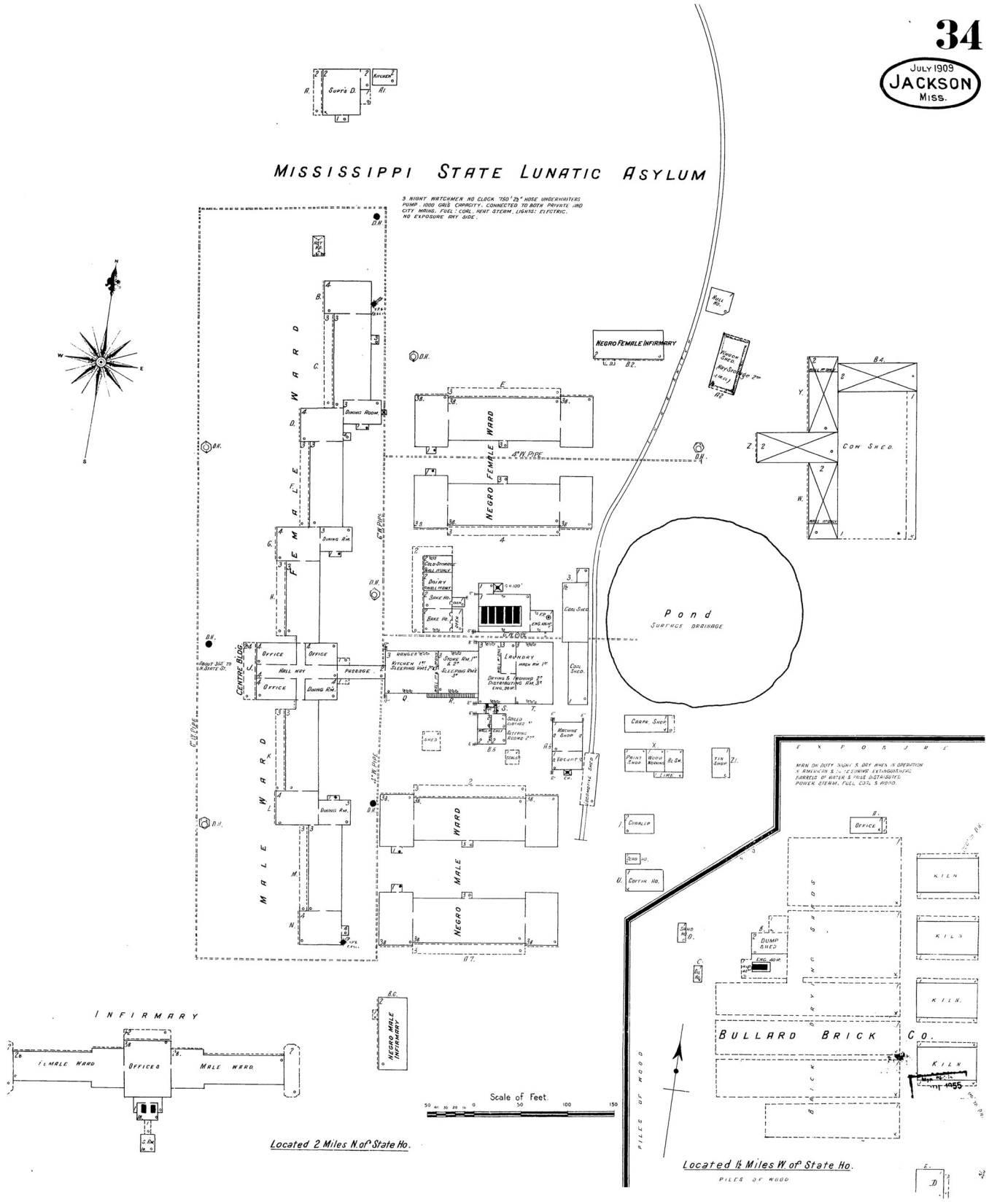

FIGURE 2 1909 MSA map, illustrating the gender and race-segregated wards and buildings. Photo credit: Mississippi Department of Archives and History

TABLE 1 Details of deaths recorded in the death certificate data 1912–1925

Covariates	Death certificate (%)
N	2258
Age (years)	
0–19	111 (4.9%)
20–39	889 (39.4%)
40–59	672 (29.8%)
60+	586 (25.9%)
Average (SD)	44.8 (18.6)
Sex	
Male	1133 (50.2%)
Female	1125 (49.8%)
Respiratory	
Respiratory	352 (15.6%)
Non-respiratory	1906 (84.4%)
Influenza	
Influenza	95 (4.2%)
All other CoD	2163 (95.8%)
Race	
Black	1923 (85.2%)
White	335 (14.8%)
Time period	
1912–1917 (pre-pandemic)	782 (34.6%)
1918–1919 (pandemic)	466 (20.6%)
1920–1925 (post-pandemic)	1010 (44.7%)

While Black and/or AA patients did account for the majority of patients at the MSA in the early 20th century, the overrepresentation is also primarily because their families were disproportionately unable to claim their bodies prior to burial—a phenomenon common to pre-WWII institutionalized populations in the US (Richardson, 1987).

2.4 | Cause of death classification

For the DC data, recorded CoD was translated into 2021 International Statistical Classification of Disease and Related Health Problems (ICD-10) adult (15–124 years) categories for the general injury or disease type. Non-respiratory and non-infectious CoD with multiple possible etiologies (e.g., "stroke"), and CoD that reflect 19th and early 20th century medical diagnostic criteria that do not align with ICD-10 categories (e.g., "traumatism") were grouped as "Unknown." Within the ICD-10 categories, subcategories were created and used for analysis (Table 1). Certain infectious and parasitic diseases were subdivided into respiratory infections and non-respiratory infections. Respiratory infection CoD were further categorized into "Influenza," other established risk factors for influenza mortality (i.e., TB) (Short et al., 2018), and established complications of influenza (i.e., pneumonia) (Short et al., 2018). Categorization continued with respiratory diseases CoDs that are established risk factors for influenza mortality (i.e., asthma) as well as nervous and circulatory system diseases CoDs that are established complications of influenza (i.e., encephalitis and myocarditis) (Short et al., 2018). Notably, multiple CoD were occasionally reported in the DC data. In these cases, the primary CoD was categorized, with two exceptions: If the primary CoD met the criteria for Unknown, but the second could be translated into an ICD-10 category (e.g., Maniacal exhaustion/Pellagra), the secondary CoD was analyzed; second, given our focus on influenza, if the secondary CoD was pneumonia or influenza it was analyzed, rather than the primary CoD.

3 | DATA ANALYSIS

Data were analyzed using R v3.6. Packages used included dplyr (Wickham et al., 2021), huxtable (Hugh-Jones, 2021), broom (Robinson et al., 2021), officer (Gohel, 2021a), flextable (Gohel, 2021b), tidyverse (Wickham et al., 2019), and gtsummary (Sjoberg et al., 2021).

3.1 | Outcome variables of interest

Four outcome variables of interest were assessed: respiratory mortality (RM), influenza or pneumonia mortality (IPM), respiratory syndemics (RS), and influenza or pneumonia syndemics (IPS). *Respiratory mortality* was defined as any CoD classified as respiratory, which included TB, asthma, influenza, and pneumonia for MSA patients. The respiratory CoD could be listed alone or with other secondary conditions, and this binary variable was "yes" for all individuals whose CoD fit the criteria. *Influenza or Pneumonia Mortality* was defined as any deaths from influenza or pneumonia according to MSA DC, including listings of the singular disease or secondary CoD. This binary variable was "yes" for all individuals who died by influenza or pneumonia. *Respiratory syndemic* was a binary categorical variable defined as any CoD wherein both a respiratory disease (i.e., TB, asthma, pneumonia, and influenza) and a secondary CoD were reported for the same patient. *Influenza/Pneumonia Syndemic* was defined as a dichotomous categorical variable with values of "yes" for patients with two CoD, one of which had to be either influenza or pneumonia.

3.2 | Predictor variables of interest

Predictor variables of interest and demographic variables were constrained to those available in the death certificates (e.g., sex, age, social race [race], cause of death [CoD], and death date).[11] *Race* was a binary categorical variable with values of "Black" or "White," and *Sex* was a binary categorical variable with values of "Male" or "Female," both based on DC data. *Age* was categorized into the following bins: 0–19, 20–39, 40–59, and 60+ years. While some data variation may have been sacrificed, binning was done to enable comparability with

TABLE 2 Logistic regression models using key variables within the MSA Death Certificate (DC) data, for all respiratory mortality outcomes (with odds ratio)

	Model 1	OR	Model 2	OR	Model 3	OR	Model 4	OR
Race (White = 1)	0.473***	1.605	0.449***	1.567	−0.197	0.821	−0.195	0.823
	(0.162)		(0.164)		(0.853)		(0.859)	
Sex (female = 1)	−0.239**	0.787	−0.224*	0.799	−0.197	0.821	−0.177	0.838
	(0.119)		(0.120)		(0.495)		(0.498)	
Adult age (20–39 yo)	−0.003	0.997	−0.018	0.982	0.048	1.049	0.015	1.016
	(0.238)		(0.240)		(0.317)		(0.319)	
Middle age (40–59 yo)	−0.748***	0.474	−0.743***	0.476	−0.945***	0.389	−0.942***	0.390
	(0.250)		(0.252)		(0.338)		(0.340)	
Old age (60+ yo)	−1.483****	0.227	−1.436****	0.238	−1.753****	0.173	−1.714****	0.180
	(0.276)		(0.277)		(0.394)		(0.396)	
Race & sex	-		-		0.351	1.420	0.135	1.145
					(1.490)		(1.503)	
Race & adult age	-		-		0.289	1.335	0.320	1.377
					(0.940)		(0.947)	
Race & middle age	-		-		0.854	2.348	0.848	2.334
					(0.928)		(0.935)	
Race & old age	-		-		1.361	3.900	1.379.	3.973
					(0.952)		(0.958)	
Sex & adult age	-		-		−0.179	0.836	−0.155	0.857
					(0.523)		(0.527)	
Sex & middle age	-		-		0.265	1.303	0.245	1.278
					(0.557)		(0.561)	
Sex & old age	-		-		−0.044	0.957	−0.051	0.951
					(0.668)		(0.671)	
Race, sex & adult age	-		-		−0.222	0.801	−0.161	0.851
					(1.588)		(1.601)	
Race, sex & middle age	-		-		−0.685	0.504	−0.465	0.628
					(1.601)		(1.614)	
Race, sex & old age	-		-		−0.619	0.538	−0.456	0.634
					(1.634)		(1.646)	
Death during pandemic	-		0.597****	1.817	-		0.602****	1.827
			(0.151)				(0.151)	
Death after pandemic	-		−0.054	0.948	-		−0.055	0.946
			(0.140)				(0.141)	
N	2258		2258		2258		2258	
AIC	1908.558		1890.392		1921.119		1902.717	

Note: Model 1 includes basic demographics. Model 2 includes basic demographics and dummy variables for the pandemic time period (1918–1919) and the post-pandemic time period (1920–1925), relative to the pre-pandemic period (1912–1917). Model 3 includes demographics with their interactions. Model 4 includes demographics with their interactions and time period dummy variables.
****$p < 0.001$; ***$p < 0.01$; **$p < 0.05$; *$p < 0.1$; .$p < 0.15$.

established analyses of the 1918 pandemic. These explore differences in outcome by age group rather than the effects of additional years of life, as 1918 pandemic mortality did not affect age in a linear fashion (e.g., Morens & Fauci, 2007; Short et al., 2018; van Wijhe et al., 2018); binning allows for age's inclusion within logistic regression models.[12] Binning also accommodates potential minimal age heaping, which is common in contemporaneous institutional records. Finally, *Year of Death* was categorized into the following periods: "Pre-Pandemic" (i.e., 1912–1917), "Pandemic" (i.e., 1918–1919), "Post-Pandemic" (i.e., 1920–1925). State-level mortality records and MSA

Superintendents' reports do not indicate that the pandemic persisted in Mississippi into 1920 (DeCesare Ross, 2017; Mitchell, 1919; MSBH, 1919).

3.3 | Model

Standard logistic regression models were used to assess relationships between period of death (i.e., Pandemic/Post-Pandemic/Pre-Pandemic) and CoD outcome variables. Race, sex, and age were also added to the models to control for confounding. Model 1 included basic demographics. Model 2 included basic demographics and dummy variables for the pandemic time period (1918–1919) and the post-pandemic time period (1920–1925), relative to the pre-pandemic period (1912–1917). Model 3 included demographic variables with interactions (e.g., race by sex, race by mid age, race by old age, etc.). Model 4, the most fully adjusted model, included demographic variables, their interaction terms, and time period dummy variables.

4 | RESULTS

As seen in the descriptive statistics provided in Table 1, MSA patients averaged 45 years of age at death. Sex was equally represented. CoD was identified as respiratory mortality linked to TB, asthma, influenza, or pneumonia for 16% of the sample. Meanwhile, 4.2% of the patients specifically had a CoD of influenza or pneumonia. Death caused by a respiratory syndemic was listed for 5.1% of patients sampled, while mortality from an influenza or pneumonia syndemic was recorded for 1.9% of patients sampled. Within this sample, 35% of deaths occurred within the pre-pandemic time period (1912–1917), 21% occurred during the pandemic (1918–1919), and 45% occurred in the post-pandemic period (1920–1925). Finally, the average year of death was 1919.[13]

Key intersectional identities (i.e., [social] race, sex, and age) were interacted with one another to control for their potential overlapping impact on the outcome being assessed. After controlling for the effects of race, sex, age, and their intersectional identities, patients who died during the pandemic had 1.84 times higher odds of a respiratory disease CoD (i.e., TB, asthma, influenza, and pneumonia) compared to before the pandemic (Table 2, Model 4, $N = 2258$). Of influenza and pneumonia mortality specifically, patients who died during the pandemic had 3.06 times higher odds of dying from influenza or pneumonia compared to before the pandemic (Table 3, Model 4, $N = 2258$). Patients who died during the pandemic had 3.91 times higher odds of a respiratory syndemic CoD listed compared to pre-pandemic period patients (Table 4, Model 4, $N = 2258$). Finally, compared with pre-pandemic patients, pandemic period patients who died had 6.25 times higher odds of having an influenza or pneumonia syndemic CoD listed (Table 5, Model 4, $N = 2258$).

Several identity variables showed statistically significant mortality differences in the most fully adjusted model (Model 4) across at least one of the following CoD categories: respiratory CoD, influenza or pneumonia CoD, respiratory syndemic CoD, or influenza/pneumonia CoD (Figure 3a,b). Older adults (60+ years) had significantly lower odds of having a respiratory CoD (OR = 0.18, $p < 0.0001$), an influenza or pneumonia CoD (OR = 0.31, $p < 0.1$), or a respiratory syndemic CoD (OR = 0.29, $p < 0.05$) compared to young patients (0–19 years). White and/or EA patients had 19 times higher odds compared to Black and/or AA patients of dying from an influenza or pneumonia syndemic (Table 5, $p < 0.05$). Finally, the addition of the intersecting identity variables in Model 4 attenuated the relationship between sex and CoD, yielding no statistically significant differences. However, Model 2, which does not include the intersecting identity variables, shows that female patients had significantly lower odds of dying from any respiratory cause (OR = 0.80, $p < 0.1$), influenza or pneumonia, (OR = 0.45, $p < 0.0001$), a respiratory syndemic (OR = 0.67, $p < 0.05$), or an influenza and pneumonia syndemic (OR = 0.38, $p < 0.01$).

5 | DISCUSSION

This case study explores the pandemic's impact within a particular socially marginalized community of individuals institutionalized at the MSA, and how vulnerability to pandemic infectious disease varied relative to patients' social identities. This provides limited insights into vulnerability to pandemic infectious disease amongst institutionalized populations, which has been scarcely addressed for the 1918 pandemic or other past pandemics (Dimka & Mamelund, 2020; Zuckerman et al., 2022). Insights are limited due to several constraints. These include some of the material and methodological limitations that have historically impeded analyses of past pandemics, as well as the challenges faced by biocultural analyses of past pandemics using historical evidence. Amongst other limitations, these include variable levels of diagnostic ambiguities and imprecision in the causes of death recorded for patients (see Beemer, 2014) and the limited range of demographic analyses recorded within the Death Certificate. High levels of incompleteness within the Death Certificates also resulted in a reduced total sample size. Constraints further include the unavailability of data representing the 1918 influenza pandemic's impacts on contemporaneous, non-institutionalized communities, including the MSA staff; Jackson, MS; the larger region; or the state of Mississippi, which curbs broader, comparative analyses. For instance, we cannot currently gain more fine-grained resolution into host risk factors in the MSA or institutionalized populations more generally. Data on morbidity and case fatality ratios from respiratory infection and influenza are also unavailable; these would enable more refined comparisons to the national-level experiences of Black and/or AA patients and White and/or EA patients during the pandemic. Comparative data on the 1918 pandemic's impacts on other institutionalized populations in the US is also currently scarce, preventing explorations of whether our findings relative to social race and sex are specific to the MSA or Mississippi or are more generalizable. Despite these limitations, our analyses could reveal how death from influenza and related causes varied by intersecting identities of

TABLE 3 Logistic regression models using key variables within the MSA DC data, for only influenza/pneumonia mortality outcomes (with odds ratio)

	Model 1	OR	Model 2	OR	Model 3	OR	Model 4	OR
Race (White = 1)	0.942****	2.565	0.922****	2.514	1.421.	4.143	1.500.	4.481
	(0.245)		(0.249)		(0.954)		(0.980)	
Sex (female = 1)	−0.806****	0.447	−0.801****	0.449	−0.881	0.414	−0.842	0.431
	(0.227)		(0.229)		(1.138)		(1.147)	
Adult age (20–39 yo)	−0.230	0.794	−0.276	0.759	0.169	1.184	0.101	1.106
	(0.406)		(0.412)		(0.559)		(0.566)	
Middle age (40–59 yo)	−0.690	0.502	−0.668	0.513	−0.267	0.765	−0.236	0.790
	(0.424)		(0.430)		(0.577)		(0.584)	
Old age (60+ yo)	−0.880**	0.415	−0.749*	0.473	−1.273*	0.280	−1.162*	0.313
	(0.438)		(0.445)		(0.686)		(0.693)	
Race & sex	-		-		1.035	2.816	0.569	1.767
					(1.809)		(1.849)	
Race & adult age	-		-		−0.708	0.493	−0.690	0.502
					(1.090)		(1.119)	
Race & middle age	-		-		−1.389	0.249	−1.496	0.224
					(1.152)		(1.178)	
Race & old age	-		-		−0.008	0.992	−0.036	0.965
					(1.153)		(1.178)	
Sex & adult age	-		-		−0.600	0.549	−0.555	0.574
					(1.206)		(1.215)	
Sex & middle age	-		-		−0.082	0.922	−0.136	0.873
					(1.251)		(1.260)	
Sex & old age	-		-		1.291	3.634	1.301	3.674
					(1.306)		(1.315)	
Race, sex & adult age	-		-		−0.013	0.987	0.116	1.123
					(1.985)		(2.026)	
Race, sex & middle age	-		-		−0.877	0.416	−0.390	0.677
					(2.218)		(2.255)	
Race, sex & old age	-		-		−1.578	0.206	−1.243	0.288
					(2.028)		(2.066)	
Death during pandemic	-		1.159****	3.187	-		1.118****	3.058
			(0.260)				(0.262)	
Death after pandemic	-		−0.222	0.801	-		−0.257	0.773
			(0.289)				(0.291)	
N	2258		2258		2258		2258	
AIC	769.732		740.490		777.046		749.204	

Note: Model 1 included basic demographics. Model 2 included basic demographics and dummy variables for the pandemic time period (1918–1919) and the post-pandemic time period (1920–1925), relative to the pre-pandemic period (1912–1917). Model 3 included demographics with their interactions. Model 4 included demographics with their interactions and time period dummy variables.
****$p < 0.001$; ***$p < 0.01$; **$p < 0.05$; *$p < 0.1$; .$p < 0.15$.

age, sex, and social race. Unpacking these dynamics involves synthesizing infectious and pandemic disease biology (e.g., pathogen transmission) with temporally, regionally, and socially specific biological and social conditions and exposures, including segregation, gender roles, and institutionalization.

As our findings show, the 1918 influenza pandemic greatly affected mortality patterns within the MSA. Independent of their multiple intersecting identities (i.e., race, sex, and age), patients who died during the pandemic period (1918–1919) were significantly more likely to have a reported respiratory illness CoD

TABLE 4 Logistic regression models using key variables within the MSA DC data, for respiratory SYNDEMIC outcome (with odds ratio)

	Model 1	OR	Model 2	OR	Model 3	OR	Model 4	OR
Race (White = 1)	0.715***	2.044	0.734***	2.083	1.421.	4.143	1.427.	4.168
	(0.245)		(0.248)		(0.954)		(0.971)	
Sex (female = 1)	−0.434**	0.648	−0.402**	0.669	0.595	1.813	0.608	1.837
	(0.199)		(0.200)		(0.741)		(0.748)	
Adult age (20–39 yo)	−0.256	0.774	−0.281	0.755	0.412	1.510	0.354	1.425
	(0.349)		(0.352)		(0.551)		(0.556)	
Middle age (40–59 yo)	−0.894**	0.409	−0.944**	0.389	−0.757	0.469	−0.806	0.446
	(0.372)		(0.376)		(0.609)		(0.613)	
Old age (60+ yo)	−1.744****	0.175	−1.756****	0.173	−1.273*	0.280	−1.254*	0.285
	(0.436)		(0.440)		(0.686)		(0.690)	
Race & sex	-		-		−0.441	0.644	−0.369	0.692
					(1.589)		(1.639)	
Race & adult age	-		-		−0.951	0.386	−0.880	0.415
					(1.086)		(1.105)	
Race & middle age	-		-		0.023	1.024	0.058	1.060
					(1.084)		(1.101)	
Race & old age	-		-		−0.550	0.577	−0.510	0.600
					(1.209)		(1.224)	
Sex & adult age	-		-		−1.265.	0.282	−1.186.	0.305
					(0.792)		(0.800)	
Sex & middle age	-		-		−0.354	0.702	−0.357	0.699
					(0.869)		(0.876)	
Sex & old age	-		-		−1.119	0.327	−1.237	0.290
					(1.122)		(1.127)	
Race, sex & adult age	-		-		−0.093	0.911	−0.326	0.722
					(1.834)		(1.879)	
Race, sex & middle age	-		-		−0.810	0.445	−0.915	0.401
					(1.849)		(1.896)	
Race, sex & old age	-		-		0.416	1.516	0.374	1.453
					(2.024)		(2.064)	
Death during pandemic	-		1.354****	3.872	-		1.363****	3.910
			(0.299)				(0.301)	
Death after pandemic	-		1.107****	3.024	-		1.107****	3.024
			(0.282)				(0.283)	
N	2258		2258		2258		2258	
AIC	877.078		854.611		888.800		866.340	

Note: Model 1 included basic demographics. Model 2 included basic demographics and dummy variables for the pandemic time period (1918–1919) and the post-pandemic time period (1920–1925), relative to the pre-pandemic period (1912–1917). Model 3 included demographics with their interactions. Model 4 included demographics with their interactions and time period dummy variables.
****$p < 0.001$; ***$p < 0.01$; **$p < 0.05$; *$p < 0.1$; .$p < 0.15$.

(influenza/pneumonia), or syndemic disease CoD than other CoD. Via syndemic theory and ecosocial theory, these results indicate that respiratory mortality was syndemic with other conditions in the MSA, clustering notably with pellagra. Pellagra, or Niacin deficiency, was endemic in the impoverished late 19th to early 20th century American South, especially amongst women, children, socially marginalized communities, like Black and/or Aas and institutionalized populations, as well as low income and poor communities (e.g., sharecroppers and factory workers) (Bollet, 1992). Pellagra's high incidence in the South was largely attributable to cotton monoculture, which undermined nutrition by displacing local food production (Clay et al., 2019). Pellagra is characterized

TABLE 5 Logistic regression models using key variables within the MSA DC data, for influenza/pneumonia SYNDEMIC outcome (with oddsratio)

	Model 1	OR	Model 2	OR	Model 3	OR	Model 4	OR
Race (White = 1)	1.490****	4.436	1.498****	4.473	2.858**	17.43	2.956**	19.22
	(0.330)		(0.335)		(1.288)		(1.315)	
Sex (female = 1)	−0.972***	0.378	−0.971***	0.379	0.556	1.743	0.607	1.834
	(0.340)		(0.343)		(1.430)		(1.439)	
Adult age (20–39 yo)	−0.296	0.744	−0.348	0.706	0.956	2.600	0.869	2.386
	(0.516)		(0.524)		(1.047)		(1.053)	
Middle age (40–59 yo)	−1.068*	0.344	−1.077*	0.340	−0.029	0.971	−0.031	0.970
	(0.558)		(0.567)		(1.104)		(1.109)	
Old age (60+ yo)	−1.898***	0.150	−1.784***	0.168	−0.764	0.466	−0.646	0.524
	(0.653)		(0.661)		(1.233)		(1.238)	
Race & sex	-		-		−0.401	0.669	−0.714	0.490
					(2.005)		(2.079)	
Race & adult age	-		-		−1.750	0.174	−1.710	0.181
					(1.422)		(1.451)	
Race & middle age	-		-		−1.627	0.197	−1.713	0.180
					(1.488)		(1.515)	
Race & old age	-		-		−2.188	0.112	−2.229	0.108
					(1.783)		(1.804)	
Sex & adult age	-		-		−2.208	0.110	−2.123	0.120
					(1.542)		(1.551)	
Sex & middle age	-		-		−1.248	0.287	−1.298	0.273
					(1.659)		(1.668)	
Sex & old age	-		-		−15.246	0.00	−15.309	0.000
					(808.3)		(792.6)	
Race, sex & adult age	-		-		1.106	3.022	1.035	2.815
					(2.272)		(2.340)	
Race, sex & middle age	-		-		0.289	1.335	0.594	1.812
					(2.471)		(2.536)	
Race, sex & old age	-		-		15.673	6,405,054	15.886	7,925,279
					(808.3)		(792.6)	
Death during pandemic	-		1.854****	6.387	-		1.832****	6.249
			(0.467)				(0.470)	
Death after pandemic	-		0.764.	2.147	-		0.767.	2.154
			(0.489)				(0.492)	
N	2258		2258		2258		2258	
AIC	409.554		392.383		421.831		405.601	

Note: Model 1 included basic demographics. Model 2 included basic demographics and dummy variables for the pandemic time period (1918–1919) and the post-pandemic time period (1920–1925), relative to the pre-pandemic period (1912–1917). Model 3 included demographics with their interactions. Model 4 included demographics with their interactions and time period dummy variables.
****$p < 0.001$; ***$p < 0.01$; **$p < 0.05$; *$p < 0.1$; .$p < 0.15$.

by dermatitis, diarrhea, and psychomotor and emotional disturbances (e.g., aggression), dementia, and in severe untreated cases, death (Bollet, 1992). Because of the aggression and dementia, pellagra was not only a common cause of institutionalization but also subsequent death at the MSA. This was especially true for Black and/or AA patients, who may have experienced worse nutrition prior to admission (e.g., Mitchell, 1919; Stewart, 1911). Along the biological-biological interface, syndemic comorbid conditions, such as pellagra, may have increased vulnerability to death from influenza at the MSA. This is because pellagra is also associated with impaired cellular immune responses (CIR) against both bacterial and viral pathogens, including those causal to respiratory infections

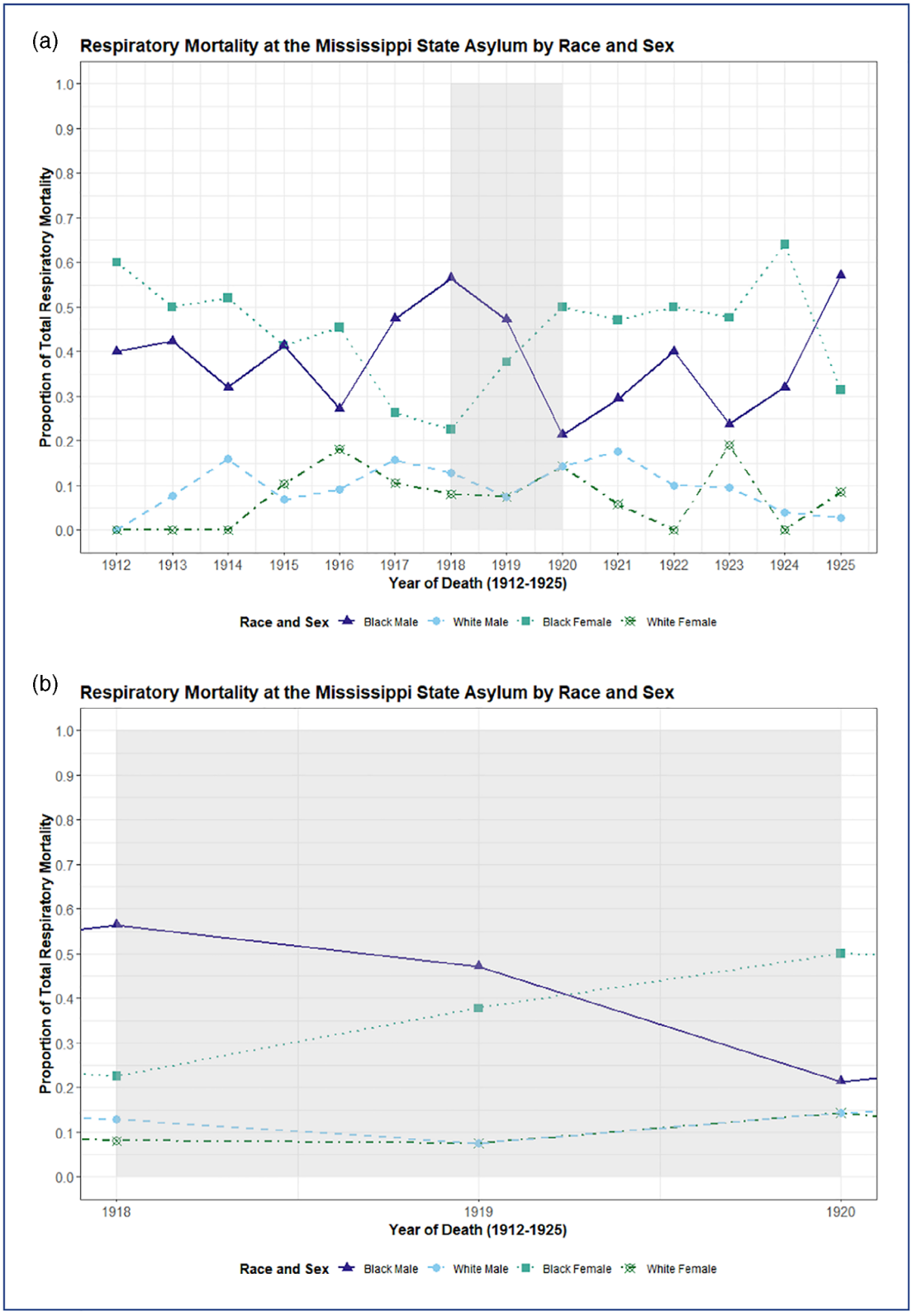

FIGURE 3 (a) Respiratory mortality at the MSA relative to race and sex, spanning the pre-pandemic, pandemic, and post-pandemic periods, and (b) in the pandemic period alone

(Kirkland, 2012; Mikkelsen & Apostolopoulos, 2019). Thus, widespread pellagra in the MSA may have increased patients' vulnerability to death from influenza and other respiratory infections.

Along the biological-social interface, compounded vulnerabilities may have occurred through various pathways, many established prior to admission. These pathways likely include poor health and comorbid

conditions prior to admission amongst most of the poor to laboring-class patients (Lampton, 2017; Mitchell, 1919; Stewart, 1911). They may also have included inadequate healthcare systems for rural, poor, and Black and/or EA communities; in both the segregated and independent systems for Black and/or AA communities, resources were scarce, resulting in generally inadequate care (Franklin & Wilson, 2020; Gamble, 2010; Sano, 2010). Further, they may have included high rates of inadequate and overcrowded residential housing, which produced high rates of respiratory infection for Black and/or AA communities and poor White and/or EA communities (i.e., TB) (de la Cova, 2011; Franklin & Wilson, 2020). Systematic experiences of chronic and/or episodic physiological stress from life-long experiences of systemic racism and poverty, and interpersonal discrimination may have further corroded overall health and resilience (Franklin & Wilson, 2020). Post-admission, the MSA's remote, suburban location, away from medical resources, and regional resource drainage by WWI likely represented additional health vulnerabilities. In addition, during the pandemic, widespread infection amongst the staff may have degraded patient care and worsened mortality, especially for those patients who were highly dependent on daily support (Zuckerman et al., 2022). Together, or in isolation, these pathways may have heightened vulnerability to influenza and other respiratory infections amongst MSA patients.

Older MSA patients (60+) had lower odds of dying from the various respiratory related health outcomes we tested, compared to younger patients (0–19 years old). This mirrors state, national, and international patterns (Morens & Fauci, 2007; MSBH, 1919). This age-based mortality pattern also aligns with other analyses of the 1918 pandemic's impacts on the MSA (Zuckerman et al., 2022). Together, these suggest that other biological and social processes, conditions, and pathways operating for institutionalized patients, as well as intersections with other aspects of their social identities (i.e., sex and race), did not markedly mediate age-based vulnerability and resistance to the 1918 influenza pandemic. Instead, the same patterns of pre- existing cross-reactive immunity, dysregulated hyperinflammatory responses, degraded immunological competence, and consequent secondary bacterial infection at play in larger populations were likely also present in the MSA patient population (see Zuckerman et al., 2022).

In contrast, respiratory mortality relative to sex and race was likely influenced by the multiple intersecting positions of MSA patients, pre- and post-admission. Studies of seasonal, epidemic, and pandemic influenza mortality have found that sex-based differences occur but often vary by region, age, and subpopulation (Dimka & Mamelund, 2020; Paskoff & Sattenspiel, 2019; WHO, 2010). Interpreted bioculturally, within intersectionality and ecosocial theory, current study results may reflect combinations of sex-based and gendered differences in vulnerability to influenza, further entangled with the embodied effects of racism. The lack of comparative regional-level data obfuscates whether the finding relates to conditions of institutionalization as well (Zuckerman et al., 2022). This is because worse infection outcomes (e.g., viral infections) in males versus females may be attributable to sex-based differences in CIR and acquired immunological responses to pathogens, driven in part by the immunomodulatory effects of sex steroid hormones (Klein & Flanagan, 2016). However, the dramatic differences shown in Figure 3 within respiratory mortality between White males and Black males, as well as White females and Black females, indicate that biology alone is not responsible for this pattern. Accordingly, the finding—including the temporal differences in mortality seen within these groups in 1918 and 1919 (Figure 3b)—may also be attributable to race and gender-based spatial aspects and differential population density within the MSA. White men and women patients lived in separate wards in the same building, whereas Black men and women patients lived in separate buildings, which may had different conditions; note that respiratory mortality was already elevated amongst Black men prior to the pandemic, for instance (Figure 3a). Especially in the early 20th century, population density was much higher in the Black and/or AA patients wards. In addition to affecting transmission of influenza and outbreak timing, these conditions may have limited staff's ability to isolate and quarantine sick patients, particularly in the Black and/or AA patients' buildings (Dimka & Mamelund, 2020; Zuckerman et al., 2022).

Notably, while White and/or EA patients were more likely to have influenza or pneumonia syndemic CoD, this is likely due to bias in the sample sizes; because White and/or EA patients represented small portions of deaths recorded in the DC, mortality from influenza within the White and/or EA patients represents a proportionately larger amount of mortality than those amongst Black and or/AA patients. Overall, this means that this finding does not reflect the odds of mortality that Black patients experienced at the MSA.

Gendered and race-based differences in exposure risk may have contributed to these higher odds of mortality amongst Black and/or AA patients. Some of these may have been mediated by activity. At the MSA, like many contemporaneous asylums (e.g., McCandless, 2013), available records suggest that patients performed regular labor as part of their treatment model (i.e., labor therapy) and for institutional upkeep. Further, they suggest that these activities were patterned along traditional gender roles in the early 20th century American South. Women largely inhabited domestic spheres in their occupational, daily, and social activities (Amott & Matthaei, 1996); at the MSA they performed domestic work inside the institution (e.g., sewing, laundry, kitchen, and occupational therapy) (United States Census, 1930). Men conducted these activities in the public sphere (Amott & Matthaei, 1996); at the MSA, they engaged in more public-facing work (e.g., farming, gardening, carpentry, and dairying) (United States Census, 1930). Notably, while White and/or EA patients performed some of this labor at the MSA, records show that Black and/or AA patients performed more institutional labor. With staff, new patients, and visitors responsible for introducing influenza into the enclosed population of the MSA in 1918, the more public roles and exposure of Black and/or AA male patients may have exacerbated their underlying biological vulnerability (Zuckerman et al., 2022).

Once infected, Black patients' risk of mortality, especially male patients, may have been heightened by pre- and post-admission biological and social conditions, both on a local and societal-level. These include ward-specific environmental conditions, such as poor

ventilation within the Black and/or AA patients wards; insalubrious conditions in local jails; and prejudice towards Black and/or AA communities held by community members, law enforcement, and MSA staff (e.g., "racial influences" on susceptibility to TB). These highly localized experiences may have exacerbated the pernicious effects of larger societal factors for Black and/or AA communities, such as likely high allostatic load from pre-institutionalization experiences of systemic poverty and racism and inadequate health care systems (de la Cova, 2011, 2014; Franklin & Wilson, 2020; Gamble, 2010; Mitchell, 1919, 1926; Stewart, 1911; Sano, 2010). Several, or all these conditions, exposures, and pathways may have operated cumulatively and intersectionally to produce the distinctive patterns of social identity relative to pandemic influenza observed here.

Our review and case study demonstrate the applicability of the biocultural approach, ecosocial theory, intersectionality, and syndemic theory for comprehending patterns of pandemic infectious disease in the past relative to social context, especially social inequality. Together, they provide inclusive, multi-directional understandings of how dialectical relationships between pathogen ecology and evolution and human biology, behavior, culture, and environments can shape pathogen exposure and pandemic outcomes, as well as the origins of pandemics (e.g., zoonoses) (see Friedler, 2021). In our case study, they reveal that while the MSA was a singular institution, its patient population did not have a singular experience of the 1918 influenza pandemic. Instead, patients' social identities and associated biological and social conditions and experiences, both pre- and post-admission, shaped their likelihood of living through the pandemic or dying from it. Further, combining these theories and approaches here not only enabled investigation of *how* influenza mortality and comorbidity were unequally distributed across and within the patient population, and the role of overlapping identities and systems of oppression in these inequities, but also *why* they were not equitably distributed, thus highlighting the loci of compounding vulnerabilities. Within our case study, insights into how syndemics within and across the patient population during the 1918 influenza pandemic profoundly impacted vulnerability and mortality also highlight the importance of considering comorbidity relative to pandemics and other infectious disease events in the past. Here, syndemics and comorbidity could be assessed courtesy of the use of historical death certificate data. However, methods development in quantitative assessments of comorbidity in the past (e.g., comorbidity indices) could further enhance the applicability of syndemic theory to paleopathological and bioarchaeological analyses of past pandemics, especially relative to social inequality. Integrating these theories and approaches enables identification of how multiple, often interconnected, patterns of pandemic infectious diseases and related health outcomes are reciprocally influenced by social inequality, as they were for MSA patients during the 1918 influenza pandemic.

6 | CONCLUSION

As we have shown above, though the diverse impacts and health outcomes of pandemics and large-scale epidemics reflect the biology of the causal pathogens, the transmission and ecology of pathogens also reflect human behaviors and social systems. Reconstructing how social context, and social inequality especially, intersected with the course, outcomes, and impacts of past pandemics is vital for identifying the long-term historical roots of many of the disparities in infection risk, morbidity, mortality, comorbidity, and indirect and intergenerational effects from recent and ongoing pandemics (Bos & DeWitte, 2022; DeWitte & Wissler, 2022). Following the biocultural approach, when such underpinnings are found, they often have shared ultimate causes. While secondary, proximate causes, like the causal pathogen, change between pandemics, the ultimate causes, like poverty and prejudice, rarely do (van Doren, 2021). Indeed, despite more than a century of intervening time, many socially vulnerable communities of persons with disabilities living in congregate settings, such as institutions, have experienced disproportionate harm and mortality rates during the COVID-19 pandemic (Shakespeare et al., 2021; Turk et al., 2020). These echo our findings at the MSA. Despite such continuities and what they forbode for the COVID-19 pandemic and future pandemics, the social lessons of biocultural analyses of past pandemics have not been considered in public health responses to pandemics and pandemic preparedness (Mamelund et al., 2020). Instead, public health responses and pandemic preparedness plans typically downplay social justice issues of social inequality and vulnerability (Mamelund, 2017; O'Sullivan & Bourgoin, 2010). However, highly socially contextualized data on the patterns of past pandemics can produce fine-grained, pragmatic, and actionable predictions of the dynamics of present and future pandemics. These include which communities will be most affected, when, and how, and which adaptive strategies proved most effective for improving human health and well-being in the past (Singer & Rylko-Bauer, 2021; van Doren, 2021). This means that moving forward, the social context-grounded data that biocultural analyses of past pandemics can provide could enable more tailored, effective, efficient, and social justice-oriented public health responses and pandemic preparedness.

AUTHOR CONTRIBUTIONS

Molly K. Zuckerman: Conceptualization (lead); formal analysis (supporting); investigation (lead); methodology (supporting); project administration (lead); resources (lead); supervision (lead); writing – original draft (lead); writing – review and editing (lead). **Anna Grace Tribble:** Conceptualization (supporting); data curation (lead); formal analysis (lead); investigation (supporting); methodology (lead); software (lead); validation (lead); visualization (lead); writing – original draft (supporting). **Rita M. Austin:** Conceptualization (supporting); investigation (supporting); writing – original draft (supporting); writing – review and editing (supporting). **Cassandra M. S. DeGaglia:** Investigation (supporting); project administration (supporting); writing – original draft (supporting); writing – review and editing (supporting). **Taylor Emery:** Resources (lead).

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CONFLICT OF INTEREST

The authors have no conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the Asylum Hill Research Consortium (AHRC). Restrictions apply to the availability of these data, which were used with permission from the AHRC for this study. Data are available from the AHRC (asylumhillproject@umc.edu) upon reasonable request.

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ENDNOTES

[1] Defined in depth in the following section.

[2] Also known as mortality crises.

[3] Intersectionality is inconsistently conceived of (e.g., theory, approach, and perspective) and definitions of it are often unclear (Collins, 2015). Many scholars consider this open-endedness and flexibility to be one of its greatest strengths, as it increases its applicability (Davis, 2008). Here, we conceive of it as an approach.

[4] The explicit definition and consistent use represents a movement forward from the very reasonable criticism raised by Wiley and Cullin (2016) that the biocultural approach is often vaguely defined and inconsistently applied.

[5] One Health is a collaborative, transdisciplinary approach that recognizes the interconnectedness of human, animal, plant, and environmental health at local, regional, national, and global levels (OHC, 2021).

[6] Research within DOHaD links early life health experiences and environmental conditions, including adverse experiences, to later life health outcomes, such as increased vulnerability to chronic disease (Gluckman et al., 2016).

[7] Defined as shared sets of values and beliefs.

[8] This research is also directly related to the research questions and subjects guiding research on the Mississippi State Asylum and those buried at the Asylum Hill cemetery ("burial population") (Mack, 2022). Individuals represented in the DC data used in this study are members of this 'burial population. These questions were developed in consultation with direct descendants of individuals buried at Asylum Hill, members of the Asylum Hill Research Consortium (AHRC), and members of the Asylum Hill Community Advisory Board. These questions include, but are not limited to: "What evidence exists for causes of death amongst the burial population?" "Are there instances of advanced disease?" and "How does the population profile of the burial population compare with the overall population of Mississippi at the time?" Additional research questions will explore questions of health, morbidity, and mortality amongst asylum patients during their confinement at the institution. Further research subjects with the potential to better illustrate the lives of the asylum residents, and of the people of Mississippi at large, during the second half of the 19th century and first third of the 20th century, include frailty and proportions of observed versus reported pathology (Mack, 2022). For more information on community engagement and outreach, and integration of research into this work, by the Asylum Hill Research Consortium, please see: https://asylumhillproject.org/. In terms of broad dissemination of results, at the least this research will be made available to the Asylum Hill Community Advisory Board, direct descendants of individuals buried at Asylum Hill, and the public through the "Research" segment on the above website.

[9] Referred to as Colored or Negro within the MSA records.

[10] Death certificates were transcribed by volunteers at the Mississippi Department of Archives and History (MDAH).

[11] Other available MA records, such as Superintendent's Biennial reports, do not include social race (see Zuckerman et al., 2022).

[12] Use of a continuous age variable also assumes linearity between age and the log odds ratio, which is inaccurate given the established W-shaped mortality age profile of the 1918 influenza pandemic (Noymer & Garenne, 2000). By contrast, using dummy variables for age categories does not assume linearity between age and the log odds ratio, enabling a clearer interpretation of regression coefficients.

[13] A power analysis is unnecessary to adequately assess the effects of interest due to the high prevalence of influenza deaths in the sample—4.2% of MSA patient deaths recorded in the Death Certificates—relative to the 0.5% of the US population estimated to have died during the pandemic (Crosby, 1989; Linder & Grove, 1943; Noymer & Garenne, 2000).

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