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Pandemics, past and present: The role of biological anthropology in interdisciplinary pandemic studies

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Abstract

Biological anthropologists are ideally suited for the study of pandemics given their strengths in human biology, health, culture, and behavior, yet pandemics have historically not been a major focus of research. The COVID-19 pandemic has reinforced the need to understand pandemic causes and unequal consequences at multiple levels. Insights from past pandemics can strengthen the knowledge base and inform the study of current and future pandemics through an anthropological lens. In this paper, we discuss the distinctive social and epidemiological features of pandemics, as well as the ways in which biological anthropologists have previously studied infectious diseases, epidemics, and pandemics. We then review interdisciplinary research on three pandemics–1918 influenza, 2009 influenza, and COVID-19–focusing on persistent social inequalities in morbidity and mortality related to sex and gender; race, ethnicity, and Indigeneity; and pre-existing health and disability. Following this review of the current state of pandemic research on these topics, we conclude with a discussion of ways biological anthropologists can contribute to this field moving forward. Biological anthropologists can add rich historical and cross-cultural depth to the study of pandemics, provide insights into the biosocial complexities of pandemics using the theory of syndemics, investigate the social and health impacts of stress and stigma, and address important methodological and ethical issues. As COVID-19 is unlikely to be the last global pandemic, stronger involvement of biological anthropology in pandemic studies and public health policy and research is vital.

KEYWORDS
1918 influenza, 2009 influenza, COVID-19, social inequalities, syndemics

1 | INTRODUCTION

The COVID-19 pandemic has brought into sharp relief the significant effects pandemics have on human health and societies, as well as the complex interaction of political, geographic, ecological, social, economic, demographic, and other factors that contribute to the spread of disease and disparities in outcomes. Yet, COVID-19 is only the latest in a long history of infectious diseases and pandemics that have affected human populations. Indeed, an investigation of past pandemics illustrates that, while certain factors such as the level of biomedical knowledge may vary, many contributing factors and consequences show enduring trends, such as higher levels of negative impacts on disadvantaged or marginalized populations.

Due to disciplinary interests and strengths, biological and biocultural anthropologists are ideally suited to analyze, interpret, and respond to pandemics. Nonetheless, prior to COVID-19 and despite a

substantial body of work considering infectious diseases more generally, pandemics per se have been relatively under-addressed in biological anthropology. The purpose of this paper, therefore, is to review existing scholarly literature on pandemics from a variety of related fields, and to discuss ways in which biological anthropology can extend this knowledge base. In the interest of space, our focus is on research addressing disparities in morbidity and mortality outcomes, selected because data, at least for mortality, are likely to be available with some degree of historic or even prehistoric depth, while other kinds of data (e.g., acute morbidity or long-term sequelae) may not be as accessible. Further, these outcomes are relevant to many fields, including epidemiology, public health, medicine, demography, evolutionary theory, political science, and economics, making it more likely they have been the subject of research for past pandemics. We consider disparities based on sex/gender, race/ethnicity, and health status (e.g., underlying chronic health conditions)–variables of interest to biological anthropologists as topics of research as well as of critical analysis and reflection.

In the following sections of this paper, we first establish what makes pandemics distinctive from other sorts of epidemiological events, and then highlight several theoretical approaches and examples of biological anthropological research of infectious diseases. We then review interdisciplinary work on three pandemics–1918 influenza, 2009 influenza, and COVID-19–as illustrative examples of pandemics with different magnitudes and impacts across time. These three pandemics share the qualities of a relatively quick spread of acute infectious disease to which the general population is/was considered susceptible and against which notable social and governmental action is/was taken. We conclude with a discussion of methodological and theoretical considerations for future research by biological anthropologists in pandemic studies.

TABLE 1 Glossary of epidemiological terms used throughout this paper as they apply to infectious disease

Term	Definition
age-specific rate	A rate limited to a specific age group. Calculated as the number of cases (for age-specific case rate) or deaths (for age-specific mortality rate) among the age group divided by the number of persons in that age group
case-fatality rate	Also called case-fatality ratio. Refers to the proportion of persons with a particular condition (cases) who die from that condition. The denominator is the number of persons with the condition; the numerator is the number of cause-specific deaths among those persons
epidemic wave	A period of increasing incidence, reaching a peak followed by a period of decreasing incidence
incidence	Expressed as a proportion or a rate; also called an attack rate. Refers to the frequency or fraction of persons with new cases of a disease within a specific time frame, calculated as the number of new cases divided by the size of the population at the start of the study period
outbreak	The occurrence of more cases of disease than expected in a given area or among a specific group of persons during a specific period. A common cause or connection between the cases is generally assumed. The same definition applies to an epidemic, but an outbreak is often considered more limited or localized
zoonosis	An infectious disease that is transmissible from animals to humans

Note: In epidemiology, the term 'rate' is often used more loosely to refer to proportions that are not true rates (i.e., they do not have a measure of time in the denominator).
Source: CDC (2012).

2 | DEFINING PANDEMICS

Various terms have been used to describe the spread of infectious diseases, most typically "epidemic" (Table 1 provides a glossary of additional epidemiology terms used throughout this paper). In practice, this term is often loosely defined and broadly applied to a variety of health conditions including noncommunicable diseases, as well as behaviors and ideas (Orbann, Sattenspiel, Miller, & Dimka, 2017). For example, the term epidemic has been used to describe obesity, autism, prescription drug abuse, suicide, and anti-vaccination beliefs (Anderson et al., 2020; Mannix, Lee, & Fleegler, 2020; Paulozzi et al., 2012; Wazana, Bresnahan, & Kline, 2007; WHO, 2000). However, most definitions of epidemics, particularly those involving infectious diseases, incorporate three elements. First, epidemics occur within populations bounded by geography or some other common attribute, such as occupation or cultural subgroup. Second, they are also bounded by time, usually by comparing the incidence of cases to a previous period selected as a reference or baseline. Finally, there is some assumed or identified cause, for example a virus or other pathogen (Orbann et al., 2017). In contrast, endemic refers to a disease or condition that occurs at levels that do not deviate significantly from what could be expected in a typical period of time (Antia & Halloran, 2021). The endemic state is the baseline against which an epidemic is measured; therefore, endemicity is highly relative (i.e., a condition may be endemic in one area but not another). Endemicity has recently gained global attention given the likely outcome of the COVID-19 pandemic that, rather than eradication, there will instead be persistent circulation of the virus at relatively low levels like other influenza and coronaviruses (Antia & Halloran, 2021; Torjesen, 2021).

A straightforward definition of a pandemic, therefore, is an epidemic that affects large regions, multiple countries, or even the whole world approximately simultaneously. Pandemics typically have larger and more severe consequences than more localized outbreaks or seasonal epidemics. Pandemics of acute infectious diseases typically are caused by novel pathogens or strains against which there is limited or no immunity. Thus, although definitions do not specify an absolute number of cases as a criterion, substantial morbidity and mortality can occur during pandemics, even with relatively low or moderate transmission probabilities or case fatality rates. Comparing influenza pandemics to seasonal influenza also shows that pandemics can occur in

multiple waves of infection at any time of the year. Different age-specific rates or patterns may also be seen, such as more cases among presumed healthy young adults, whereas seasonal epidemics more often affect the youngest and oldest age groups and those with underlying health conditions (Fleming, 2005; Luk, Gross, & Thompson, 2001; Nguyen-Van-Tam & Hampson, 2003; Simonsen et al., 2011).

Beyond this definition of a pandemic by epidemiological measures, however, is the political definition. Ultimately, an epidemic is when an outbreak of disease becomes a political event requiring social action; the same is true, again on a larger or even global scale, for a pandemic (Charters & Heitman, 2021; Cohen, 2011; Flegal, 2006). As seen with COVID-19, the decision to declare something a pandemic may have important implications for how the situation is handled by government bodies, policymakers, and health care workers at all levels. Additionally, it may affect how the disease is discussed in news and social media, how individuals respond, and whether and how much financial and other resources are invested in slowing or stopping the spread of disease and mitigating other social and economic effects caused by, for example, the closure of businesses and schools (Hathaway & Phillips-Robins, 2020; McKeever, 2020). As biological and social/cultural phenomena with potential for relatively sudden, global impacts, pandemics present unique challenges and raise different questions across multiple interdisciplinary fields than may be recognized in research on other sorts of epidemics or outbreaks more generally.

3 | ANTHROPOLOGICAL APPROACHES TO INFECTIOUS DISEASE

Anthropologists have long been interested in infectious diseases, and a full review of previous research is outside the scope of this paper. However, previous reviews and theoretical articles have discussed numerous examples, including many of the ones noted below. For example, Inhorn and Brown (1990) grouped work into three categories–biological, ecological, and sociocultural–while also recognizing the holistic nature of the work. Sattenspiel (2000) focused on the role of biological, environmental, and social factors in tropical infectious diseases, including human behaviors, host-pathogen relationships, and modes of transmission such as vector-borne and waterborne routes; cholera, dracunculiasis, and lymphatic filariasis were used as case studies to illustrate these points. Similarly, Larsen (2018) took a narrower focus, using tuberculosis (TB), treponematosis, dental caries, and periodontitis to illustrate issues of bioarchaeological research on infectious disease. Sattenspiel and Herring (2010) also discussed thematic trends in research, including geographic distribution and spread of infectious diseases, emerging infectious diseases, and syndemics, concluding with insights regarding the questions asked by anthropologists that overlap with, but are distinct from, epidemiologists.

Applications of anthropology to public health and collaboration with epidemiologists has been more thoroughly addressed by Manderson (1998) and Trostle and Sommerfeld (1996). Manderson (1998) focused on the role of sociocultural medical anthropology in elucidating local knowledge and concerns about infectious diseases and translating those to culturally appropriate public health interventions. Trostle and Sommerfeld (1996) discussed trends in the intersections between cultural and medical anthropology and epidemiology from a history characterized as "benign neglect" to interdisciplinary borrowing and collaboration on topics such as culture change and stress, behavior, illness classification, and social stratification. They concluded that multimethod research had become increasingly common, but that theoretical challenges and conceptual critiques remained underdeveloped, an observation that holds today. In a more recent perspective piece, van Doren (2021) emphasized the importance of biocultural anthropology to public health, specifically in how a focus on temporal depth and population health dynamics can contribute to understanding the social determinants of health.

In the following sections, we review illustrative examples of anthropological theory and research on infectious disease. As noted by several of the above authors, most notably Inhorn and Brown (1990), who argued that analysis of infectious diseases requires a bridge across or perhaps even erasure of "irrelevant" boundaries separating anthropological subfields, such research tends to be interdisciplinary, although some studies fall along more traditional boundaries. Within cultural anthropology specifically, key approaches have included ethnographic and critical medical anthropology (CMA) approaches; see, for example, Joralemon (2017) and Wiley and Allen (2017) for discussion of different approaches. Examples of infectious disease research from such perspectives include the classic work on kuru, a neurodegenerative disease caused by prions observed among the Fore of Papua New Guinea and associated, albeit not uncritically, with cannibalism (Lindenbaum, 1979, 2008; Steadman & Merbs, 1982); numerous studies on HIV/AIDS and other STIs (e.g., Green, Jurg, & Dgedge, 1993; Songwathana & Manderson, 2001); and research on vaccine hesitancy and refusal (e.g., Brunson & Sobo, 2017). Others have emphasized more nuanced analyses of, for example, gender, socioeconomic, and political issues in relation to health conditions (e.g., Farmer, 1996, 2004, 2006; Vlassoff & Manderson, 1998), reflecting CMA's central focus on power inequities. Given the venue and likely audience, a full discussion of this body of work is outside the scope of this paper. Rather, we focus the following review on biological anthropology, including bioarchaeological and biosocial/biocultural approaches.

3.1 | Biological anthropology and bioarchaeology research

Biological anthropologists, bioarchaeologists, and researchers in related fields prioritize biomedical understandings of infectious disease, often focusing on evolutionary and ecological factors contributing to the spread of pathogens and epidemics. For example, a number of authors have noted the likely substantial role of infectious diseases in human evolution and have applied insights from epidemiological

transition theory to hypothesize about the types of diseases most likely to be prevalent with different types of social organization, such as with the development of agriculture and permanent settlements (Barrett, Kuzawa, McDade, & Armelagos, 1998; Cockburn, 1971; Harper & Armelagos, 2010; Zuckerman, 2014). Further, studies have investigated infectious diseases and epidemics in nonhuman primates, the co-evolution of humans and pathogens, the role of human behavior in the emergence and spread of zoonoses, and the distribution of traits such as ABO blood types relative to the prevalence of associated infectious diseases (e.g., Genton et al., 2017; Sattenspiel, 2015; Van Blerkom, 2003; Wolfe, Daszak, Kilpatrick, & Burke, 2005).

Other work has used genetic, osteological, bioarchaeological, and paleopathological methods to address the antiquity and impacts of infectious diseases in past populations, using both specific indicators of certain diseases and nonspecific indicators of general health and stress (DeWitte & Wood, 2008; Stone, Wilbur, Buikstra, & Roberts, 2009; Wilbur, Farnbach, Knudson, & Buikstra, 2008). A classic debate centers on whether syphilis originated in the "Old" or "New World" (Arora et al., 2017; Baker & Armelagos, 1988; Crosby, 1969; Harper, Zuckerman, Harper, Kingston, & Armelagos, 2011; Livingstone, 1991). More recent work has included investigation of possible selective pressures such as smallpox that contributed to the frequency and geographic distribution of CCR5-Δ32, a variant allele for the gene that codes for the CCR5 receptor on immune system cells and confers resistance against HIV-1 infection (Galvani & Slatkin, 2003; Hummel, Schmidt, Kremeyer, Herrmann, & Oppermann, 2005).

Biological anthropology-oriented research has also used a variety of historical, archaeological, and epidemiological sources to better understand the impact of infectious diseases on immunologically naïve Indigenous populations (e.g., Hurtado, Hill, Rosenblatt, Bender, & Scharmen, 2003; Merbs, 1992; Walker, Sattenspiel, & Hill, 2015). Interdisciplinary work counters simplistic "virgin soil" epidemic narratives and includes recognition of both Indigenous agency and the effects of colonial conquest and extreme violence (e.g., Cameron, Kelton, & Swedlund, 2015). For example, colonial social structures, such as the Spanish caste system, systematically erased Indigenous identities and Indigenous people were legally discriminated against to keep them in states of structural poverty. These new and severe social inequalities likely interacted synergistically with, rather than independently from, novel pathogens (Gutiérrez, 2015).

Studies with a biological focus typically still include social and cultural variables, such as socioeconomic measures or insights from observed human behavior. A key example incorporating the environment, human behavior, and biology is the classic research on the relationship between the prevalence of malaria, the distribution of the sickle cell trait, forest clearing, and crop cultivation (Allison, 1954; Livingstone, 1958). More recent research has further explored malaria elsewhere, such as relationships between malaria, paleoenvironments, and the settlement of Oceania (Clark & Kelly, 1993); and malaria, moat-building, corralling, deforestation, and increased movement of people in Southeast Asia (King, Halcrow, Tayles, & Shkrum, 2017). New work suggests a significant malaria burden in mainland Southeast Asia prior to the adoption of agriculture (Vlok et al., 2021). Other recent work has studied environment-, culture- and behavior-specific stress-induced epigenetic modifications and their effects on chronic health conditions and acute infectious diseases (Thayer & Kuzawa, 2011), which may be further complicated by the epigenetic inheritance of intergenerational trauma (Conching & Thayer, 2019). Thus, while considerations of how environment and culture affect the physical body are not new, there have been recent advancements in biocultural methods and theory and increases in application of these approaches to human health and disease.

3.1.1 | Biosocial/biocultural integration and syndemics

As noted above, Inhorn and Brown (1990) observed the bridging role studies of infectious diseases can play between cultural and biological subfields. More recently, McElroy and Townsend (2015) and Wiley and Allen (2017) also demonstrated the ways that anthropological subfields and related disciplines can overlap theoretically and methodologically to address research questions related to health and medicine. Biocultural approaches to anthropology have gained popularity in the 21st century; however, Wiley and Cullin (2016) pointed out that among biocultural research published in major anthropological journals, there is little consensus over the precise definition of a biocultural approach. Recently, Leatherman and Goodman (2020) considered the state of biocultural anthropology incorporating critical theory, 20 years after the publication of *Building a New Biocultural Synthesis: Political-Economic Perspectives on Human Biology* (Goodman & Leatherman, 1998). While they noted that engagement with historical, political-economic, and sociocultural conditions in work described as biocultural is still lacking, they also highlighted multiple methods and theoretical approaches that indicate that biocultural integration is particularly suited for practical and applied questions related to health in human populations. Such contributions include studies of the developmental origins of health and disease, epigenetics, the microbiome, the measurement and analysis of biomarkers, and embodiment (Leatherman & Goodman, 2020), largely conducted using the laboratory- and field-based technologies of biological anthropology approaches.

Krieger (1994) first encouraged the move away from an emphasis on epidemiological methods, whose goals were to disentangle the "web of causation", to a discussion of a more broadly conceptualized ecosocial theory. Since then, ecosocial theory has become a prominent social epidemiology framework that describes how social experiences (e.g., sources of social inequalities) are literally integrated into biology, or become embodied, over the course of the lifespan (Krieger, 1999, 2014). However, syndemics, primarily developed by Singer and colleagues (e.g., Singer & Clair, 2003), is perhaps currently the most dominant theoretical framework stemming from CMA, integrating anthropological subfields, and even transcending interdisciplinary boundaries into multiple health-related fields.

Syndemics refer to the *syn*ergistic interaction of multiple epi*demics* within adverse social contexts (Singer & Clair, 2003). Both biological interactions between relevant health conditions and biosocial interactions between diseases and social conditions can have direct or indirect mutually enhancing effects, resulting in symptoms or consequences that are novel and/or worse than either condition in isolation. Historical and systemic factors leading to social inequalities, such as poverty, stigma, and deleterious environmental exposures, contribute to syndemic configurations by concentrating conditions within specific areas or populations and shaping the distribution of risks and resources (Gravlee, 2020; Singer, Bulled, & Ostrach, 2020). Numerous publications have considered syndemics associated with infectious diseases, including the earliest developed example of substance abuse, violence, and AIDS (SAVA), particularly among the urban poor (Singer, 1994, 1996; Singer & Snipes, 1992). Other studies have applied the syndemic concept to infectious disease configurations of HIV, TB, and homicide (Freudenberg, Fahs, Galea, & Greenberg, 2006); HIV and several other STIs (Singer et al., 2006); TB and helminths (Littleton & Park, 2009); tickborne diseases (Singer & Bulled, 2016); and the 1918 influenza pandemic and TB (Herring & Sattenspiel, 2007), as well as a growing body of literature related to COVID-19 (e.g., Ali, 2021; Gravlee, 2020; Horton, 2020; Singer, 2020).

In response to this emerging literature, some of which has suggested that syndemics involving pandemic diseases can be viewed as global syndemics, Singer, Bulled, and Leatherman (2021) argued that while syndemics are not themselves global, pandemic diseases can give rise to syndemics shaped by local conditions, and the concept of local biologies can help us understand the nature of disease interactions. Local biologies (now a subcategory of the more recently developed concept of situated biologies) refer to nongenetically determined biological differences among people resulting from the body's response to differing environments, some of which have profound consequences for well-being (Lock, 2017). As local biologies emphasize biosocial entanglement and continual, didactic interaction across time and space (Lock, 2017; Singer et al., 2021), this concept is a pertinent example of work that crosses traditional academic boundaries.

3.1.2 | Biological anthropology and pandemics

Several diseases studied by biological anthropologists and colleagues arguably fit the broad epidemiological and/or narrow political definitions of a pandemic. These include, but are not limited to, cholera (Sawchuk, 2001; Sawchuk, Tripp, & Samakaroon, 2020), TB and leprosy (Kelmelis & Dangvard Pedersen, 2019; Kelmelis, Price, & Wood, 2017; Stone et al., 2009), smallpox (Duggan et al., 2016), scarlet fever (Roberts & Battles, 2020; Swedlund & Donta, 2003), and polio (Battles, 2017). However, in such research, anthropologists have rarely conceptualized or theorized these diseases *as* pandemics rather than as smaller outbreaks/epidemics or as case studies with no or only minor discussion of the broader contexts of surrounding pandemics, if applicable given the period of study. Even with studies that explicitly frame the work within a pandemic context, the analysis is often at the level of smaller geographic or population scales. Consequently, there is still less emphasis placed on critical analysis of international or global, multilevel factors that contribute to and are the consequences of pandemics. Broader theoretical advances have been made recently, particularly in the realm of bioarchaeology, with the release of a special issue of *Bioarchaeology International*, which includes, among other papers, substantive discussions of demographic and evolutionary consequences of pandemics (DeWitte & Wissler, 2022), addressing intersectionality in the context of pandemics (Yaussy, 2022), the long-term sequelae of disabled pandemic survivors (Battles & Gilmour, 2022), and the social vulnerability to pandemics of institutionalized populations (Zuckerman, Emery, DeGaglia, & Gibson, 2022).

Perhaps the three pandemics that have received the most attention from biological anthropologists, alongside colleagues in other anthropological subfields and related disciplines, are HIV/AIDS, the Black Death, and the 1918 influenza pandemic. Here, we highlight examples of such work, including studies on the 1918 pandemic that address other research questions than the major themes discussed further below.

As noted above, much work especially from cultural medical anthropologists on the biological and biosocial interactions of syndemics has focused on HIV/AIDS. Additionally, biological anthropology-oriented studies have looked at, for example, genetic and ecological evidence for emergence of the virus, as well as similarities to related viruses in other primates (Wertheim & Worobey, 2009; Wolfe, Dunavan, & Diamond, 2007). Further, modeling studies have explored the spread of HIV based on population structure, behaviors, and partnerships (e.g., Goodreau et al., 2012; Katz et al., 2021; Sattenspiel, Koopman, Simon, & Jacquez, 1990). Previous reviews of anthropological work on HIV include MacQueen (1994) and Hutchinson (2001).

Research on the Black Death (referring to the first epidemic of the Second Pandemic of Plague as it occurred in Europe between 1346 and 1353) can be divided into two broad realms: genetic and bioarchaeological/paleopathological analyses. Genetic analyses have been applied to confirm the causative pathogen, *Yersinia pestis*, and reconstruct the genome (Bos et al., 2011; Haensch et al., 2010; Schuenemann et al., 2011). For example, Bos et al. (2011) confirmed that, as the reconstructed genome was ancestral to most extant strains, the Black Death was likely the main event connected to the introduction and spread of all currently circulating strains, which continue to cause sporadic zoonotic infections and outbreaks in human populations today (Barbieri et al., 2020). Further, Bos et al. (2011) concluded that the increased virulence of the pandemic pathogen was likely due to other factors than the bacterial phenotype, such as social conditions, vector dynamics, and host genetics. For example, one recent study of plague victims later in the Second Pandemic, from 16th-century Germany, found evidence of selection affecting the frequency of human genes responsible for adaptive immunity (Immel et al., 2021).

Bioarchaeological and paleopathological analyses have teased apart demographic and health information about populations affected by the Black Death, as well as compared trends before, during, and after the pandemic. DeWitte and Wissler (2022) thoroughly reviewed this research, including selective mortality with respect to factors such as age, sex, and frailty, as well as work on pre and postpandemic health, social inequality, and demography. For example, DeWitte (2015) argued that observed reductions in survival and increased mortality before the Black Death indicated the populations were stressed beforehand and thus possibly explained the high mortality rates during the pandemic. Further, as with other pandemics, mortality was not indiscriminate, and older adults and those already stressed were more likely to die. This pattern of mortality, combined with better standards of living, likely led to improvements in population health after the Black Death, an argument supported by the frequency of skeletal stress indicators (e.g., periosteal lesions), a greater number of old adults, and a positive association between age and lesions (DeWitte, 2014). Analyses from before, during, and after the Black Death in Denmark showed that urbanization had differential impacts on survivorship across the lifespan (Kelmelis & DeWitte, 2021). Further, despite potential sex differences following the pandemic (e.g., DeWitte, 2018; DeWitte & Lewis, 2021), research on whether sex was associated with previous stress, frailty, and thus mortality risks during the Black Death and subsequent recurring plagues has produced mixed results (Curtis & Roosen, 2017; DeWitte, 2009, 2010; Godde, Pasillas, & Sanchez, 2020).

Of the three pandemics discussed further in this paper, the 1918 influenza pandemic has received the most attention from biological anthropologists, occasionally in collaboration with demographers and historians. For example, research has explored the pandemic in populations in North America and elsewhere, including demographic and health impacts and potential explanations for the geographic spread of disease and regional variation in outcomes (e.g., Herring, 1993; Mamelund, Sattenspiel, & Dimka, 2013; Palmer, Sattenspiel, & Cassidy, 2007; Sattenspiel, 2011). Herring and Sattenspiel (2007) and Sattenspiel and Mamelund (2012) discussed the pandemic's impacts on parts of Canada and Alaska within the framework of syndemics, highlighting various social, ethnic, and environmental issues, as well as the role of pre-existing or co-circulating pathogens such as TB and pneumonia. Similarly, Tripp, Sawchuk, and Saliba (2018) explored the 1918 influenza pandemic on the islands of Malta and Gozo, reporting considerably different experiences possibly explained by factors such as poverty, isolation, rurality, and the roles of women and children in introducing the disease to households. Additionally, Sattenspiel and colleagues have developed agent-based and other types of models to investigate how social and geographic spaces, behaviors, demography, mobility, and other factors influence the spread of infectious diseases within small, traditional communities such as those found in early 20th-century Canada (e.g., Carpenter & Sattenspiel, 2009; Dimka & Sattenspiel, 2021; O'Neil & Sattenspiel, 2010).

Studies on the 1918 flu also have addressed questions of selective mortality and interactions with TB in response to arguments proposed by Noymer and Garenne (2000) and Noymer (2009, 2011) that the pandemic hastened the decline of and reduced sex differentials in post-pandemic TB mortality. For example, Sawchuk (2009) showed that health quickly rebounded in Gibraltar following the pandemic, with sex differentials returning to levels comparable to prepandemic estimates. van Doren and Sattenspiel (2021) analyzed yearly age-standardized TB mortality rates for the island of Newfoundland from 1900 to 1939, with the expectation that, to support the hypothesis, years soon after the pandemic should show significant declines in TB mortality rates. Female mortality did decline in 1928, which was too late to be confidently associated with the 1918 influenza pandemic, while there were no significant declines for males nor for both sexes combined. Instead, the persistent high TB mortality rates throughout the early 20th century were likely due to social factors like poor nutrition and lack of health care rather than biological interactions between the diseases. van Doren and Sattenspiel (2021) emphasized, however, that their results should not fundamentally reframe the original observations made by Noymer and Garenne. Instead, populations may exhibit different patterns of post-pandemic changes due to their cultural, demographic, and epidemiological differences.

Other work has considered factors contributing to local variation that may be driven by gendered roles, exposure, and the larger global impact of troop movement for World War I. This work also argues for the importance of more research addressing the relatively understudied first wave of the pandemic (Bogaert, 2015; Rewegan, Bogaert, Yan, Gagnon, & Herring, 2015). As described by Mamelund et al. (2016), there were important dynamics in the first wave, specifically in differences in morbidity between men and women, that help contextualize and explain the observed patterns in the major wave of the pandemic later that year. Further, Mamelund (2018) showed that there was a crossover in susceptibility of groups with different socioeconomic status (SES), where lower SES groups were hit harder in the first wave, while higher SES groups were hit harder in the second wave. More work is thus needed to better understand the underlying biological and sociocultural determinants of outcomes across waves of the pandemic.

Biological anthropology research into the COVID-19 pandemic is unsurprisingly still limited. Publications to date have been largely theoretical, with a focus on potential implications for future work and the contributions and perspectives that biological anthropologists and human biologists can provide (Brewis, Wutich, & Mahdavi, 2020; Fuentes, 2020; Gravlee, 2020; Leonard, 2020a, 2020b; McDade & Sancilio, 2020). Common themes of discussion among anthropologists and others include the marked disparities in morbidity, mortality, and access to vaccination and other health care resources, and the socioeconomic impacts of both the disease itself and related public health interventions such as lockdowns and school closures. Such disparities in outcomes and other consequences during COVID-19 and previous pandemics have been studied by researchers in many fields, offering insights into the kinds of work that might be done by biological anthropologists moving forward.

4 | EPIDEMIOLOGICAL AND HISTORICAL OVERVIEW OF SELECTED PANDEMICS

In the following review sections, we consider research investigating disparities during three pandemics with varying degrees of severity: the 1918 influenza pandemic, the 2009 H1N1 influenza pandemic, and the ongoing COVID-19 pandemic. Here, we provide brief descriptions of these pandemics to place the subsequent literature review into context. The 1918 influenza pandemic has long served as a "worst-case scenario" for pandemic preparedness planning. Estimates are that this pandemic infected up to half of the global population and may have killed 50 or even 100 million people (Johnson & Mueller, 2002; Taubenberger & Morens, 2006). Early cases appeared in Kansas (US), but other proposed origins include China and Europe (Barry, 2005; Crosby, 2003; Humphries, 2013; Oxford, 2001; Oxford & Gill, 2018; Worobey, Cox, & Gill, 2019). The cause was unknown at the time, and there were no effective vaccines, antivirals, or other treatments (Crosby, 2003; Morens, Taubenberger, Harvey, & Memoli, 2010). Broadly speaking, there were three global waves. The first wave in the spring and summer of 1918 was relatively mild, with high morbidity and relatively low mortality. The second wave, beginning in the autumn of 1918, resulted in the most deaths. A third, mild wave was generally observed in early 1919, while some areas showed an "echo" wave in 1920 that varied in size from relatively small to the largest wave of the pandemic in some locations (Chandra & Christensen, 2021; Johnson & Mueller, 2002; Patterson & Pyle, 1991; Sattenspiel, 2011). In addition to the substantial mortality overall, one of the most well-known, but still not fully explained, characteristics of the 1918 pandemic is the signature W-shaped age distribution of deaths (Figure 1), indicating unusually high mortality rates among young adults and lower than expected mortality in the elderly age classes (Gagnon et al., 2013; Luk et al., 2001; Olson, Simonsen, Edelson, & Morse, 2005).

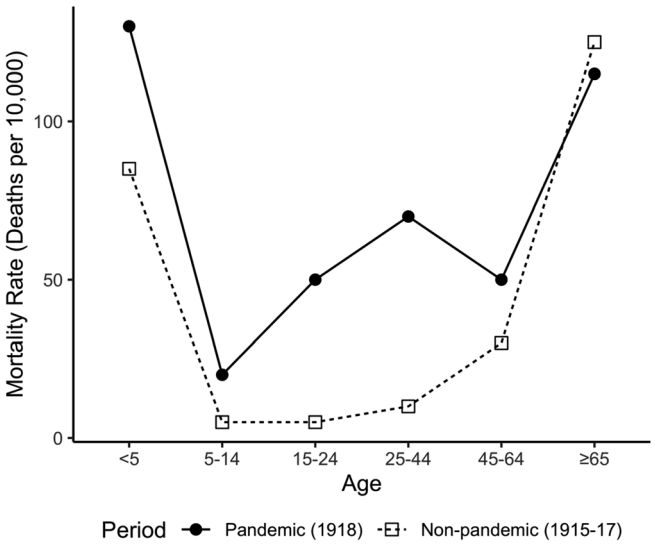

FIGURE 1 The W-shaped age distribution of influenza deaths during the 1918 pandemic, compared to the U-shaped pattern of 1915–1917. Data from Olson et al. (2005), figure 2a

The 2009 H1N1 influenza pandemic began in Mexico in March, with initial cases observed in California in April (Fowlkes et al., 2011; Khandaker et al., 2011). The pandemic quickly spread to the US and Canada, then European and other countries. The World Health Organization (WHO) declared it a pandemic on June 11, by which time cases had been confirmed across 74 countries (Cortes Garcia et al., 2012; Khandaker et al., 2011; Mytton et al., 2012; Sullivan, Jacobson, Dowdle, & Poland, 2010). The pandemic continued until August 2010 (da Costa, Saivish, Santos, de Lima Silva, & Moreli, 2020). Two to three waves were reported in different locations, and the timing of these varied as well, for example between the northern and southern hemispheres (Chowell et al., 2011; Jhung et al., 2011; Mytton et al., 2012). Genetic analyses indicated the strain was produced through the regrouping of genes from human, avian, and swine type influenza A viruses (da Costa et al., 2020). The hemagglutinin gene, which codes for an important surface antigen, had evolved from the 1918 virus, and early serologic data suggested many older adults had some cross-reactive immunity (Jhung et al., 2011). Indeed, there was an age pattern similar to 1918, with children and young adults disproportionately affected relative to those older than 65 years (Campbell et al., 2011; Fowlkes et al., 2011). While the reported number of laboratory-confirmed deaths was 18,500, a much larger mortality toll of about 201,200 respiratory deaths plus 83,300 associated cardiovascular deaths has been estimated (Dawood et al., 2012). This burden was not dramatically different from seasonal flu, which typically results in 5–15% of the population infected and approximately 291,000–646,000 deaths annually (Iuliano et al., 2018; WHO, 2021b). Nonetheless, it was a very high-profile pandemic because of the intense discussion and speculation surrounding when the "next" pandemic would occur in the years leading up to it (e.g., Taubenberger, Morens, & Fauci, 2007), as well as the severe stigmatization that emerged in the immediate wake of the pandemic (for example, accusing "illegal aliens" of bringing the flu across the Mexican border to the US and the generally racist rhetoric and political action against Mexico) (Singer, 2009).

In late December 2019, a pneumonia of unknown etiology broke out in Wuhan, Hubei Province, China, apparently associated with the Huanan Seafood Wholesale Market. A new pathogen, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), was identified on January 7 and is responsible for the disease, which came to be known as coronavirus disease 2019 (COVID-19) (Guarner, 2020; Sohrabi et al., 2020; Wu, Chen, & Chan, 2020). Genetic analyses support a zoonotic origin, although some have argued that other explanations, such as the so-called "lab-leak hypothesis", cannot be ruled out (Maxmen & Mallapaty, 2021; Thacker, 2021; Wu et al., 2020). After declaring the disease a Public Health Emergency of International Concern on January 30, the WHO characterized it as a pandemic on March 11 (Sohrabi et al., 2020; Williamson et al., 2020). Many countries around the world have experienced three or more waves of infection, with approximately 470 million cases and more than 6 million deaths reported as of this writing (Johns Hopkins University &

Medicine, 2022). Unlike the two influenza pandemics described above, increased mortality among young adults has been less of an issue, with most COVID-19 deaths occurring among the elderly (Islam et al., 2021). This age profile appears to be dependent on SES, however, as some low- and middle-income countries have reported "flatter" age-based mortality curves, suggesting higher relative mortality in the younger age classes than seen in high-income countries that report virtually no child or young adult mortality (Bonanad et al., 2020). The age profile of cases also has been affected by the introduction of vaccines, which were not initially approved for younger children, and the emergence of variant strains (e.g., Wu, 2021).

5 | EVIDENCE OF MORBIDITY AND MORTALITY DISPARITIES

In the following review of interdisciplinary literature on the three pandemics, we focus on morbidity and mortality disparities based on sex/gender, race/ethnicity, and health/disability status. These topics were chosen as (some but not the only) examples of concepts of interest to anthropologists that transcend any single pandemic or related research question, and indeed can be used to demonstrate how the relevance or salience of these variables may change over time or in different contexts. In addition to being variables commonly collected during anthropological and epidemiological research and important components of identity that may influence behavior or perceptions, they also invite opportunities for broader theoretical engagement. Each of these concepts reflect social constructs derived at least to some extent from and applied to biological characteristics; as such, they also correspond to systems of oppression, that is, sexism/patriarchy, racism, and ableism, that contribute to observed disparities. Nonetheless, as our review shows, while many epidemiological studies in particular often included more than one of the discussed factors, they typically gave little attention to these larger social issues or how they intersect. We further discuss the lack of and need for more theoretical engagement with such concepts below and in Section 6.

For each of the topics, we discuss results for each pandemic and build to integrated summaries and general observations on the literature. Overall, the volume of research and sophistication of analyses clearly have increased over time. This trend is likely due to, among other factors, the availability of individual- and population-level data, epidemiological surveillance practices and regulations in different countries, and improvements in statistical computing power. In one of the first epidemiological descriptions of the 1918 influenza pandemic, Frost (1919) acknowledged that because of the difficulty in disaggregating influenza and other respiratory causes of morbidity, especially when the seasonal curves overlap, the two things required for accurate study of an epidemiological event (systematic occurrences of the event and clear differential diagnoses) were not yet up to par in 1918. Even today, testing and diagnosis issues, asymptomatic cases, and limited access to health care result in underestimates of infections, cases, and deaths. Further, because COVID-19 is an ongoing pandemic at the time of this writing, the findings are provisional and subject to revision, although many results have shown consistency.

5.1 | Sex and gender

Sex and gender are relatively understudied in the pandemic literature, and therefore their patterns and effects on populations are not well understood. Many of the papers examined as part of this literature review use the terms "sex" and "gender" in interchangeable ways, that is, as synonyms rather than as distinct concepts. Thus, unclear definitions of the concepts of sex and gender, as well as a lack of critical evaluation of how and when they do and do not overlap, impede epidemiological research on disparities and may hinder public health interventions for the affected populations.

5.1.1 | 1918 influenza

Much of the early research on the 1918 influenza pandemic was first released in a series in the journal *Public Health Reports*, which used survey and mortality data from 12 locations in the US that were then required to report such information (Armstrong & Hopkins, 1921; Britten, 1932; Collins, 1931). Collins (1931) noted that the survey data may have had some gender bias, since women were more likely to answer survey questions and to remember their own experiences with influenza-like illnesses than those of their family members. Nonetheless, Collins (1931) reported that the incidence rates between the sexes showed small differences that were unlikely to be significant, although pneumonia incidence was reported to be much higher for males aged 10–50 years. Both mortality and case fatality were reportedly much higher in males in almost all the areas surveyed, corroborating initial observations in the US that despite a likely higher incidence of influenza and pneumonia, females were less likely to die than males (Frost, 1919; Sydenstricker, 1927).

There is often no clear answer to whether the 1918 pandemic affected males or females more severely, due to the complex ways in which age and sex interacted to increase vulnerability to the virus, as illustrated by the W-shaped age-specific mortality curve (Luk et al., 2001; Taubenberger & Morens, 2006). In one of the few studies on the 1918 influenza pandemic that analyzes age-specific mortality between males and females, Viboud et al.'s (2013) analysis in Kentucky showed that sex-based differences in mortality were highly age-dependent. Male mortality was 20–80% higher for ages 15–50, while female mortality was higher in all other groups.

In the US on a national level, age-standardized male mortality in 1918 far exceeded that of females by 174 deaths per 100,000, driven by much higher mortality in younger (ages 20–49) males, while there was a slight excess in elderly (ages 70+) female mortality (Noymer, 2011; Noymer & Garenne, 2000). Higher male mortality was generally observed outside the US as well, although results are mixed by age and epidemic wave. For example, on the island of Newfoundland, there were no statistically significant differences in sex-based

mortality rates, but logistic regression models accounting for age and region of death suggested that females aged 20–44 were consistently more likely to die during the pandemic (Paskoff & Sattenspiel, 2019). The overall pattern in Newfoundland showed that males had higher mortality during the earliest wave in the spring of 1918, followed by higher female mortality in the fall 1918 and winter 1920 waves, suggesting early exposure to the influenza virus and possibly some acquired immunity for the later waves in males (Paskoff & Sattenspiel, 2019). This overall epidemic pattern also was observed in Bergen, Norway, with higher levels of influenza-like illness for males aged 10–39 in the summer and higher levels for females aged 20–39 in the fall; similar trends were observed in Baltimore and rural areas of Maryland, as well (Mamelund, Haneberg, & Mjaaland, 2016). In Australia, the pandemic was quite delayed and did not hit the continent until 1919. It subsequently resulted in almost twice as many male deaths as female deaths. By the second wave in June and July, however, females died at slightly higher rates, again suggesting early exposure of males (Curson & McCracken, 2006). In a rare study of morbidity, on the Maltese Islands, reproductive-aged females (aged 15–44) displayed significantly higher morbidity rates than males (138.3 per 1000 vs. 97.3 per 1000 individuals, respectively) than in the pre and postreproductive age groups (this study used the categorical age classes of prereproductive, reproductive, and postreproductive rather than numbered ages) (Tripp et al., 2018).

The mixed results of research investigating sex-based differences in 1918 influenza pandemic outcomes raise issues with interpreting and explaining the observed patterns. One biological explanation for explaining higher female mortality and/or morbidity may be the potential effects of pregnancy. After the major fall wave, a survey collected information from US obstetricians on the experiences and outcomes of their pregnant patients. Results indicated no single month of pregnancy was more dangerous than the others, and that severe influenza outcomes in pregnant women were highly variable. Ultimately, the author determined that no immediate conclusions could be drawn regarding the relationship between pregnancy and the risk of severe disease or mortality during the pandemic (Harris, 1919). However, it is now well-known there are differences in immunological responses to pathogens between males and females generally and specifically in the context of pregnancy (Gabriel & Arck, 2014; Kadel & Kovats, 2018; Van Lunzen & Altfeld, 2014). Pregnancy can cause immune systems influenced by circulating sex steroid hormones and X-linked genes to develop an overactive immune response that ultimately increases risk for severe respiratory pandemic outcomes (Garenne, 2015; see Mertz et al., 2017 for a systematic review of pregnancy and influenza). Females are better protected from death during infections, but their stronger immune systems carry higher risk of the overactive immune response known as the "cytokine storm", or the overproduction of inflammatory cytokines that can result in death (Capuano, Rossi, & Paolisso, 2020; Gabriel & Arck, 2014; Klein, Hodgson, & Robinson, 2012; Scully, Haverfield, Ursin, Tannenbaum, & Klein, 2020).

More socially oriented explanations for differential outcomes include gendered behaviors. In particular, the overlap of the pandemic and World War I may account for observed excess male morbidity and mortality in general (Crosby, 2003). Viboud et al. (2013) observed that male mortality in Kentucky is likely underestimated because it does not include deaths of Kentucky residents who were out of state for training or combat, which should be considered in age- and sex-based analyses for belligerent countries. World War I cannot explain all the variation observed, however, because even nonbelligerent countries exhibited the W-shaped mortality curve and sex-based mortality patterns (Mamelund, 2011). However, because these two major historical events are so intertwined, it is impossible to entirely separate their effects (Humphries, 2013).

Gendered labor and social roles also likely influenced differential outcomes. As noted, men were likely exposed to the virus earlier in many locations, probably facilitated by the fact that men had higher workforce participation and were more likely to work outside of the home (Curson & McCracken, 2006; Mamelund et al., 2016; Paskoff & Sattenspiel, 2019; Tuckel, Sassler, Maisel, & Levkam, 2006). Further, Paskoff and Sattenspiel (2019) noted that urban/rural regional variation in social and labor roles on the island of Newfoundland was important for explaining why, in some spaces, women were the ones who experienced the most exposure due to their tight-knit social networks and caregiving behavior in their communities, rather than only with their own families.

5.1.2 | 2009 influenza

International differences in surveillance, reporting, and analyses limit comparisons of sex differences for the 2009 pandemic. For example, while a report produced by the WHO in 2010 analyzed general trends of seasonal and 2009 pandemic influenza, a major limitation was that many countries including Spain, the Netherlands, Thailand, and some aggregated European countries did not regularly stratify seasonal influenza data by sex (WHO, 2010). Further, analyses of the interaction between age and sex are uncommon, and countries such as Belgium, Italy, and Peru did not report age-stratified results despite finding no differences in the aggregate sex-based analyses (WHO, 2010).

Several studies reported no sex differences or mixed results. For example, there were no significant differences in hospitalizations for Australia, New Zealand, the United States, Mexico, or for an analysis of 19 pooled countries (WHO, 2010; Van Kerkhove et al., 2011), or in terms of cases, symptoms, or duration of illness in the first few hundred cases in the UK (McLean et al., 2010). In the earliest confirmed cases in Japan, females aged 20–79 had significantly higher case rates than males, while males under 20 and over 80 had higher rates, again suggesting an important intersection with age (Eshima et al., 2011). In another pooled analysis of fatal cases worldwide, Vaillant, La Ruche, Tarantola, and Barboza (2009) found a male/female sex ratio of 1.04 but did not report statistical significance.

Other research investigating sex-based differences have found a more consistent bias towards more severe outcomes in males during the 2009 pandemic. For example, male sex was identified as a

statistically significant independent risk factor for a positive influenza test among early cases identified in China (May–June 2009) (Cao et al., 2009). Out of almost 1000 cases in Delhi, India, at the peak of the pandemic, 64% were male (Kumar et al., 2012), and of the nearly 400 positive cases studied in a cluster of medical facilities in Japan, almost 59%, a statistically significant difference, were male (Takayama et al., 2011). In a study of hospitalizations in China, males accounted for 56% of admissions (Wang et al., 2014). From May–September 2009 in Victoria, Australia, more than half of the fatal cases (58%) were male (WHO, 2010), and in the United States, males had significantly higher excess mortality rates for almost all age groups (Nguyen & Noymer, 2013).

There was a considerable lack of discussion of reasons for the observed sex differences in the 2009 H1N1 impacts. Pregnancy was acknowledged as a significant risk factor, with early emphasis placed on vaccination priority for people in any stage of pregnancy (WHO, 2010; Özyer et al., 2011; Siston et al., 2010). In terms of gendered behaviors that may influence outcomes, women are more likely to be caregivers, work in direct contact with sick patients in healthcare facilities, and put health education on malnutrition into practice (WHO, 2010). For example, in a case study of a hospital in the Republic of Korea, most of the confirmed cases among healthcare practitioners were women and less than 40 years old, a result which likely says less about biological determinants of pandemic respiratory disease vulnerability and more about how many of the hospital's nurses and technicians in close contact with confirmed cases were women (Choi, Chung, Jeon, & Lee, 2011). It is notable that while most of the epidemiological analyses with sex-disaggregated data described above show evidence of poorer pandemic outcomes for males, those that did attempt to consider potential behavioral determinants focused on women's roles and activities.

5.1.3 | COVID-19

Although there has been ample research on the COVID-19 pandemic within its first 2 years, many of the initial conclusions are that it is too early to make generalizable statements about sex- and gender-based differences in COVID-19 outcomes. The GenderSci Lab at Harvard University has consistently reported sex-disaggregated deaths, mortality rates (crude and age-adjusted), and time series of age-adjusted sex ratios for all 50 US states and two territories (Harvard GenderSciLab, 2020). Recently, using this data, the first longitudinal study of sex disparities by state in the US during 13 months of the COVID-19 pandemic was published using weekly case and mortality rates (Danielsen et al., 2020). A guide produced by this lab for communicating sex and gender disparities acknowledges that sex-disaggregated data does not capture the complexity of biological sex and socially constructed gender (Danielsen & Noll, 2020; Harvard GenderSciLab, 2020). Worldwide, however, inconsistent reporting of data disaggregated by sex is a limitation. Most of the African and Asian continents have not reported sex-disaggregated data on cases, hospitalizations, and deaths, and much of South America only reports one or two of these measures with sex-disaggregated data (Global Health 5050, 2021). Further, while early analyses found few differences between males and females in terms of median age of cases, symptoms, or comorbidities (Jin et al., 2020), and no statistically significantly differences in exposure rates (Ruprecht et al., 2021), number of cases in the US (Klein et al., 2020; Scully et al., 2020), or case fatality rates (Ahrenfeldt, Otavova, Christensen, & Lindahl-Jacobsen, 2020; Gebhard, Regitz-Zagrosek, Neuhauser, Morgan, & Klein, 2020), pandemic outcomes are variable across the world. For example, females in South Korea accounted for about 60% of positive tests (Scully et al., 2020), most cases in a Washington State long-term care facility were female (Klein et al., 2020), elderly females in India had higher case fatality rates (Dehingia & Raj, 2021), and female risk of mortality in India increased with age (Joe, Kumar, Rajpal, Mishra, & Subramanian, 2020). In Massachusetts, absolute mortality rates were higher for males, but relative excess mortality for males and females was virtually identical (Krieger, Chen, & Waterman, 2020).

Yet, the overwhelming majority of COVID-19 research on sex-based differences in pandemic outcomes to date again shows that males are at far greater risk for almost every outcome. According to clinical classifications of severity based on, for example, respiratory rate and oxygen saturation, cases in males were more severe worldwide (Jin et al., 2020; Takahashi et al., 2020); however, Shattuck-Heidorn et al. (2021) have suggested that Takahashi et al. (2020) were uncritical in their analyses and interpretation of sex differences that were identified. Further, intubated males were more likely to acquire ventilator-associated pneumonia (Ahmed & Dumanski, 2020). Male hospitalizations in Switzerland and France outnumbered those of females 1.5-fold (Gebhard et al., 2020) and were double those of females in China (Klein et al., 2020). Male mortality in Italy was almost double that of females, with similar findings for six other European countries, China, and the Philippines (Capuano et al., 2020; Takahashi et al., 2020; Xie, Tong, Guan, Du, & Qui, 2020). Further, the risk of death increased with age, and males aged 30 and above had a significantly higher risk of mortality than females (Scully et al., 2020). Indeed, cumulative mortality rates and relative risks for males versus females were higher in 10 European regions for every age group (Ahrenfeldt et al., 2020). Nonetheless, Shattuck-Heidorn et al. (2021) recently argued for more careful and deeper engagement with the complexities of biological sex and gendered determinants of behavior that could further explain emerging differences.

With the COVID-19 pandemic, there has been more research on how gendered behaviors drive the unequal outcomes discussed above. For example, studies have found that men are less likely to engage in nonpharmaceutical interventions such as handwashing and social distancing, behaviors that are likely influenced by media consumption and perception of the degree of the public health emergency (Ahmed & Dumanski, 2020; Johnson, Sholcosky, Gabello, Ragni, & Ogonosky, 2003; Galasso et al., 2020; Mamelund, Dimka, & Bakkeli, 2021; Pedersen & Favero, 2020). Women, on the other hand, are more likely to wear a mask and are also more likely to seek health care (Haischer et al., 2020; Hearne & Niño, 2021; Howard, 2021). Yet, Scully et al. (2020) argued that the consistent observation of higher risk of death for males suggests a common biological explanation

more than any gendered determinants that contribute to risk. Testing of this hypothesis will rely on more comprehensive research in the coming years.

5.1.4 | Summary and observations

Most of the identified studies did not explicitly aim to investigate sex disparities but instead typically included these analyses as part of broader descriptions of pandemic impacts. This lack of focus on sex- and gender-based determinants of outcomes may be due to an assumption that pandemic respiratory diseases are indiscriminate (Mamelund & Dimka, 2021), so males and females are at equal risk. However, a consistent trend across pandemics suggests that, although there is variability, males most often have more severe outcomes. Indeed, Klein et al. (2020) likened COVID-19 to the 1918 influenza pandemic under the generalization that male outcomes were more severe.

Two general hypotheses are used to explain observed sex-based differences in disease prevalence and severity. The physiological hypothesis recognizes the importance of gonadal hormones and genetics in determining differences in disease manifestation, while the behavioral hypothesis emphasizes behavior that is determined by gender roles (Guerra-Silveira & Abad-Franch, 2013). While this distinction is useful for disentangling determinants, sex and gender – and thus the hypotheses – are not independent (Anker, 2007). Yet, much of the epidemiological literature lacks clear definitions of *sex* (the nonbinary spectrum of genotypic and phenotypic characteristics) and *gender* (the socially constructed roles and behaviors along the spectrum of gender identities) (Gentile, 1993; Torgrimson & Minson, 2005), and these terms are often used inconsistently and incorrectly (Gahagan, Gray, & Whynacht, 2015).

Despite the widely studied effects of gonadal hormones and sex chromosomes on immune function, most of the literature discussed here approaches sex as a binary category. However, some of the people who have suffered the most from the COVID-19 pandemic have been sexual and gender minority groups (i.e., those who do not identify with one of the binary sex or gender distinctions) (Gibb et al., 2020). Recently, biocultural anthropologists have highlighted that frameworks of biological "normalcy", which position biological, binary sexes as "normal", imply the existence of biological "abnormalcy" (DuBois & Shattuck-Heidorn, 2021; Wiley & Allen, 2017). This implication creates a tenuous situation for research on unequal pandemic outcomes for those that were born a biological sex outside the arbitrarily defined "normal" bounds of the binary, especially when sex is considered independently from the context of cultural effects on human biology. Additionally, Anker (2007) argued that, as gender roles are not homogeneous cross-culturally, a narrow description of gendered behaviors that lead to observed differences in infectious disease outcomes leads to a similarly narrow understanding of exactly how gender intersects with sex, culture, and behavior. Overall, then, a lack of clear definitions and nuanced consideration of sex and gender impedes pandemic research that seeks to explain variation in outcomes, and further fails to serve sex and gender minority communities that suffer highly unequal impacts of pandemic events. Biological and biocultural anthropologists therefore have much to contribute to the understanding of sex and gender in pandemic studies.

5.2 | Race, ethnicity, and Indigeneity

Populations studied in the research reviewed here largely fell into the general, not mutually exclusive categories of Indigenous populations, minority ethnic immigrant groups, historically marginalized racialized (i.e., non-White) groups, and "majority ethnic"/nonimmigrant populations that were sometimes used to compare to members of the same ethnic group who had emigrated elsewhere. We highlight illustrative examples of research on this topic with a particular focus on Indigenous populations, who are often overlooked compared to other racial and ethnic groups in both research and similar reviews.

5.2.1 | 1918 influenza

The approach of the centenary of the pandemic in 2018, along with concerns that "the next pandemic" would be a pandemic flu, likely contributed to increased research interest in the 1918 influenza and its impact, particularly for populations that had received less attention previously. Research on Indigenous populations around the world and Black Americans in the US illustrates the range of factors that have been explored to explain disparities, or lack thereof in some cases, in morbidity and mortality rates during the 1918 influenza pandemic, revealing a number of commonalities.

Several articles and reviews published in the past decade have helped to generate a clearer picture of the 1918 pandemic's impact with regards to Indigenous groups. Mortality rates for Indigenous populations relative to European settler populations were consistently found to be higher across a range of studies and countries/regions, including North America, Nordic countries, Australia, and the Pacific Islands (Mamelund, 2011). Generally, while potential genetic susceptibilities are often raised despite lacking scientific evidence, explanations for disparities focus on differences in exposure due to the remoteness and isolation of Indigenous communities, in terms of either prior exposure to the milder first wave in 1918 and/or lifetime exposure to nonpandemic influenza strains. When isolated Indigenous populations were hit by the 1918 flu, they suffered some of the highest observed influenza-related mortality. For example, Mamelund et al. (2013) reported mortality rates up to 90% in some Alaskan and Labrador communities. Similarly, the most isolated populations in the South Pacific had the highest influenza-related mortality in the Pacific region, both in terms of entire island populations and ethnically homogenous communities on individual islands (Shanks, Wilson, Kippen, & Brundage, 2018). Small populations in remote Arctic, Pacific, and Australian communities and limited contact meant long periods without exposure to influenza (Mamelund, 2011). Some

groups even missed exposure to the 1918 flu altogether (Mamelund, 2011; Mamelund et al., 2013; Philip & Lackman, 1962). Evidence for a lack of exposure to other influenza strains prior to 1918 comes in part from the age patterns of mortality, as remote Arctic and Pacific peoples generally had sustained high mortality in adults rather than the classic W-shaped pattern (Mamelund, 2011). A lack of previous influenza exposure combined with other factors such as crowding also was one likely explanation identified for the comparatively high mortality rate among Māori military personnel. Mortality for Māori in New Zealand was higher among both civilians (7.3 times) and military (2.3 times) (Wilson, Barnard, Summers, Shanks, & Baker, 2012). Shanks et al. (2018) found that Māori soldiers serving overseas in Europe and the Middle East had higher mortality than other New Zealand soldiers, so it was not just Māori in New Zealand who had higher flu mortality; the authors state the cause of this difference is unknown.

Conversely, the absence of disparities is also often explained by a lack of difference in previous influenza exposure. In contrast to the Sami's higher mortality in Norway, mortality for another ethnic minority, Kven (Finnish immigrants and their descendants), did not differ significantly from the ethnic Norwegian majority population (Mamelund, 2003). This finding is explained by a relatively high degree of economic and cultural assimilation of the Kven into Norwegian society, as opposed to the Sami, in the late 1910s (Mamelund, 2003). Further, isolation from previous influenza outbreaks did not explain mortality disparities everywhere in the South Pacific; for example, Shanks et al. (2018) found that it did not explain high mortality in New Zealand or Samoa.

Beyond exposure, other factors that have been proposed to explain higher 1918 flu morbidity and mortality in Indigenous populations include poor nutritional status, a lack of nursing and medical care, and concurrent infectious disease burden (e.g., Mamelund, 2011). It is beyond the scope of this review to cover the full extent of the health impacts of colonization on Indigenous peoples, much or all of which could have increased susceptibility to the 1918 flu (as well as to other diseases). However, some factors particularly relevant to the 1918 flu have received significant attention, including high rates of respiratory infections, particularly TB (Herring & Sattenspiel, 2007; Summers, Baker, & Wilson, 2018).

Research on other racial and ethnic groups has focused on Black Americans. In a working paper, Eiermann et al. (2021) report strikingly small differences in White versus non-White mortality. However, other research has found that in the autumn of 1918, Black Americans had lower morbidity and mortality but higher case fatality than the White population (Crosby, 1976, 2003; Økland & Mamelund, 2019). The data suggest that Black people had either lower exposure or a lower risk of developing disease given exposure, but a higher risk of dying if they did get sick (Økland & Mamelund, 2019). In a study of mortality rates in a community of people with disabilities institutionalized at the Mississippi State Asylum, Zuckerman et al. (2022) found that from 1918 to 1919, respiratory and influenza mortality rates were significantly elevated for Black patients compared to White patients. Reasons for these observations are unclear, but hypothesized explanations include higher exposure for Black people during the herald wave in 1918 (Crosby, 1976, 2003; Økland & Mamelund, 2019), with an alternative proposed explanation being lower exposure of Black Americans throughout the pandemic due to racial segregation (Gamble, 2010). Higher exposure in the herald wave, combined with higher case fatality throughout the pandemic, would fit with interpretations pointing to the role of poverty, systemic racism, and structural violence against Black Americans, especially in the American South (Zuckerman et al., 2022). Due to conditions of overcrowding and poor sanitation, Black Americans had increased exposure not only to pandemic influenza but also to other diseases associated with higher influenza mortality risk, including respiratory diseases such as TB and diarrheal and parasitic diseases that cause malnutrition (Zuckerman et al., 2022), while racism and segregation also limited Black peoples' access to medical care before and during the pandemic (Gamble, 2010).

The research on 1918 influenza morbidity and mortality disparities for Indigenous populations and Black Americans thus share similar profiles, with substantial focus on differential exposure patterns, as well as other factors (nutrition, co-morbidities and co-infections, health care access) traceable to structural inequalities and inequities, albeit with different histories.

5.2.2 | 2009 influenza

In contrast to the historical 1918 pandemic, much of the literature on 2009 H1N1 influenza disparities for Indigenous populations and other racial and ethnic groups focuses on hospitalization rates. The results are not straightforward, with different studies finding higher hospitalization rates for Indigenous groups alongside higher severity and mortality, lower hospitalization rates but a higher rate of severe outcomes, or higher hospitalization but little disparity in severity or mortality. For example, The ANZIC Influenza Investigators (2009) found that Indigenous groups in Australia and New Zealand were relatively overrepresented in intensive care unit (ICU) admissions for the study period of June–August 2009. Aboriginal and Torres Strait Islanders account for 2.5% of the Australian population but made up 9.7% of patients with 2009 H1N1 influenza who were admitted to Australian ICUs; similarly, Māori represent 13.6% of the New Zealand population but accounted for 25.0% of the patients admitted to New Zealand ICUs (The ANZIC Influenza Investigators, 2009). Similarly, in the US, hospitalization was significantly higher among American Indians compared to non-Hispanic Whites (Thompson et al., 2011). On the other hand, several studies in different national contexts found that Indigenous people, including Aboriginal Australians and First Nations, Inuit, and Métis in Canada, were less likely to be admitted to the hospital but were more likely to have severe outcomes such as ICU admission and/or death (Mertz et al., 2013; Helferty et al., 2010). Harris et al. (2010) showed that, unexpectedly given the prevalence of comorbidities, patients admitted for pandemic H1N1 in north Queensland, Australia, appeared less likely to be Indigenous. The authors suggested factors such as higher use of emergency

services, targeted public health efforts to test Indigenous populations, and uneven availability of clinical data among locations making for an unrepresentative sample may explain the greater proportion of Indigenous patients in the nonadmitted group (Harris et al., 2010).

Echoing the shift in disparities seen with the 1918 pandemic, variation was also observed across waves of the 2009 pandemic. For example, Helferty et al. (2010) found that the proportion of hospital admissions and deaths involving Aboriginal people (First Nations, Inuit, Métis, and unknown Aboriginal ethnicity) admitted to the hospital in Canada decreased from the first to second wave (27.8% to 6.1% and 17.6% to 8.9%, respectively), and the median age of Aboriginal patients increased from 11 to 26 years. Further, the proportion of Aboriginal patients who had underlying medical conditions was higher in the second wave, and the proportion of women of child-bearing age who were pregnant was lower (Helferty et al., 2010).

Severe cases and mortality were also higher for Indigenous populations. In a global pooled analysis, Van Kerkhove et al. (2011) noted that Indigenous populations and ethnic minorities were reported to experience a disproportionately high burden, particularly in the Americas and the Australasia-Pacific region. Zarychanski et al. (2010) reported similar findings and noted that while there is a possibility that host genetic factors may play a role in susceptibility to severe influenza infection outcomes, Indigenous groups in Canada, Australia, and New Zealand do not share a recent common ancestry. Rather, they share a history of colonization combined with persisting social inequities causing significant disparities in health. Analyses demonstrate that despite some improvements, these disparities have persisted over time; Wilson et al. (2012) found that the Māori death rate for 2009 was closer to, but still higher than, the New Zealand European death rate, compared to previous pandemics (rate ratio of 2.6, compared to 7.3 for 1918 flu and 6.2 for 1957 flu).

For other racial and ethnic groups, several studies have shown patterns of more hospitalization during the 2009 pandemic but fewer or no significant differences in terms of severe outcomes and death. A systematic review by Mertz et al. (2013) included a small number of studies on 1918 pandemic outcomes as well as nonpandemic flu and just under 100 studies for 2009. Results showed that Hispanic and Black patients were more likely to have been admitted to the hospital but were at lower risk for more severe outcomes. The authors suggested that this outcome might be due to healthcare providers' perception of an increased risk of complications among these groups, such that they were selectively admitted to hospitals. Further, while data on seasonal influenza by ethnicity was scarce, they found no significant differences in all-cause mortality among Asian, Black, or Native (Indigenous) populations compared with Whites during either seasonal or pandemic influenza (Mertz et al., 2013). Pandemic H1N1-related hospitalization was significantly higher among American Indians, Blacks and Hispanics than among non-Hispanic Whites in New Mexico, US, and was higher among persons of younger age and lower household income (Thompson et al., 2011). Similarly, Tricco, Lillie, Soobiah, Perrier, & Straus (2013) found significantly more hospitalizations among ethnic minorities versus nonethnic minorities in North America, yet no differences in ICU admissions or deaths among hospitalized patients in North America and Australia. They argued that their results suggest a similar burden of H1N1 between ethnic minorities and nonethnic minorities living in high-income countries (Tricco et al., 2013).

Several studies did show significant differences in outcomes other than hospitalization. For example, patients of South Asian (Indian, Pakistani, or Bangladeshi) ethnic minority groups in England had higher morbidity and mortality during the first wave of the pandemic. As these individuals were younger, more often male, and more often from deprived areas than cases from other ethnic groups, proposed explanations for these disparities include these factors, as well as being offered antiviral treatment less often (Trienekens, Shepherd, Pebody, Mangtani, & Cleary, 2021). Zhao, Harris, Ellis, and Pebody (2015) also found increased mortality risk in England for people in the non-White ("ethnic minority") category than those in the White ("nonethnic") category, with the highest risk among Pakistani people. Similarly, people living in areas with a higher level of deprivation had an increased risk of mortality associated with influenza infection (Zhao et al., 2015).

Spurred in part by the 2009 influenza pandemic, the past decade has seen more work on genetic and immunological susceptibility to influenza at both the individual- and population-level. Some of this work focused specifically on the H1N1 strain responsible for the pandemic (e.g., Liu et al., 2013; To et al., 2014; Zhou et al., 2012; Zúñiga et al., 2012), which also allowed some comparison with the 1918 H1N1 pandemic (see Short, Kedzierska & van de Sant, 2018), while other studies considered susceptibility to other strains (e.g., Quinones-Parra et al., 2014) or to influenza A in general (e.g., Clemens et al., 2016; Everitt et al., 2012). Of particular relevance to the issue of ethnic disparities is the work of Sambaturu et al. (2018), who modeled H1N1 influenza epidemics at the population level and considered variation in flu strains and ethnicity. This modeling found that while certain human leukocyte antigen (HLA) alleles might be associated with a stronger immune response in individuals, diversity mattered. Each person has six HLA class-I alleles comprising their HLA genotype; greater diversity in these individual levels of susceptibility would act as a barrier to influenza spread (Sambaturu et al., 2018). This work adds further dimension to considerations of differences in genetic susceptibility between populations as explanations for ethnic disparities.

5.2.3 | COVID-19

Commentaries and editorials early in the COVID-19 pandemic drew on work on past pandemics of respiratory diseases to inform assessments of potentially vulnerable populations and consequent policy decisions. For example, New Zealand's elimination strategy was based in part on research that indicated COVID-19 posed high risks to Māori and Pacific people, and these groups were also prioritized for vaccination (e.g., Steyn et al., 2020). As statistics from the first and subsequent waves became available, the predicted patterns largely bore out, with Indigenous, minority ethnic, and racialized groups disproportionately affected.

To date, research has shown that illness, hospitalization, and death is typically higher among Asian, Indigenous, and Black people than in White populations, with studies particularly focusing on US and UK contexts (e.g., de Lusignan et al., 2020; Gu et al., 2020; Holmes et al., 2020; Price-Haywood, Burton, Fort, & Seoane, 2020; Public Health England, 2020). For example, of 51 cases in Wales, individuals from Black, Asian, and Minority Ethnic (BAME) populations represented 35% of ICU admissions and 35% of deaths, despite accounting for less than 5% of the population covered by the hospital (Baumer, Phillips, Dhadda, & Szakmany, 2020). Similarly, Black people accounted for 45% of confirmed cases and 69% of deaths in Milwaukee County, WI, US, while representing only 27% of the population (Rast, Martinez, & Williams, 2020). Research in countries other than the US and UK also show disparities; for example, in a study from Brazil, Pardo (mixed ethnicity) status was the second most important risk factor for death in hospitalized patients after age (Baqui et al., 2020).

While much of what has been published to date does indicate higher risks of morbidity and mortality among Indigenous populations, such as those in the US and Mexico (e.g., Argoty-Pantoja, Robles-Rivera, Rivera-Paredez, & Salmeron, 2021; Arrazola et al., 2020; Hatcher et al., 2020), research on disparities for Indigenous populations is still limited, and questions have been raised about how much confidence should be placed in the findings. In a systematic review, Mackey et al. (2021) were only able to draw a low-confidence conclusion on higher mortality and no conclusions for other outcomes in American Indian/Alaskan Natives, Pacific Islanders, and other ethnic groups. In contrast, evidence enabled moderate to high confidence conclusions for Black and Hispanic populations and low confidence conclusions for Asians for all outcomes considered (Mackey et al., 2021).

Proposed explanations for higher COVID-19 mortality in non-White populations include a range of factors linked to racism and social inequality. For example, regarding disparities between Black and White Americans, Holmes et al. (2020) pointed to lower healthcare access, clinician bias, lower educational attainment, structural poverty, food insecurity, compromised immune systems, residential segregation, and workplace segregation. Explanations for higher case fatality among Indigenous, minority ethnic, and racialized groups include higher prevalence of chronic diseases and comorbidities (Bertocchi & Dimico, 2020; Holmes et al., 2020; Price-Haywood et al., 2020). Some authors have proposed vitamin D deficiency as a possible driver of ethnic disparities in COVID-19 outcomes (i.e., greater severity in people with darker skin pigmentation) in higher latitude settings such as the UK (Rhodes, Subramanian, Laird, Griffin, & Kenny, 2021), but studies so far have found conflicting evidence for this hypothesis (Hastie et al., 2020; Meltzer et al., 2021).

Several large initiatives have emerged to facilitate research into genetic factors influencing COVID-19 outcomes, including the COVID-19 Host Genetics Initiative (2020, 2021). Zeberg and Pääbo (2020) found that a gene cluster on chromosome 3 associated with respiratory failure after infection with SARS-CoV-2 is found in a higher proportion (~50%) of people with South Asian ethnicity than those of European ethnicity (~16%). This gene cluster is found in highest frequencies in Bangladesh (63%), which Zeberg and Pääbo (2020) suggested may at least partly explain why individuals of Bangladeshi origin in the UK have approximately twice the risk of dying from COVID-19 than the general population (Public Health England, 2020). To date, the researchers involved with the COVID-19 Host Genetics Initiative have reported 13 genome-wide significant loci associated with either infection with the virus or severe COVID-19 disease (COVID-19 Host Genetics Initiative, 2021). However, as response to viral infection is influenced by many different genes (and other variables), each genetic risk factor identified for COVID-19 is likely to have limited impact on its own (Schurr, 2020).

5.2.4 | Summary and observations

The literature across these three pandemics suggests race-related disparities were largely similar over time, in terms of worse outcomes for marginalized groups. Across different ethnic and racialized groups, common factors associated with higher morbidity and mortality include younger populations (Helferty et al., 2010; Thompson et al., 2011; Trienekens et al., 2021), and deprivation/social disparities that put certain populations at greater risk of infection (e.g., due to crowding) and severe outcomes (e.g., due to comorbidities). However, data quality is an issue, both in terms of availability and bias. For example, Esteban-Vasallo et al. (2012) noted that, except for the UK, Sweden, and the Netherlands, European countries rarely collect health data by ethnicity. Further, due to lack of access to medical care, cases and deaths in Black populations may have been undercounted during past pandemics (Gamble, 2010), and similar concerns have been raised about data on Indigenous populations (Kelm, 1999; Mallard, Pesantes, Zavaleta-Cortijo, & Ward, 2021).

Genetic/genomic studies search for specific genes conferring immunity/resistance or heightened susceptibility to these pandemic diseases or to comorbid conditions that also increase susceptibility, as well as analyze distribution of these genes in different populations. Biosocial/biocultural approaches to understanding and explaining disparities attend primarily to historical trends in socioeconomic and other conditions, in particular how racism has led to deprivation and discrimination that directly impacts morbidity and mortality via increased risk of exposure, complications, and prevalence of pre-existing or comorbid conditions. These two approaches reflect the complicated and contentious nature of concepts like race, ethnicity, and ancestry, which biological and other anthropologists helped produce and more recently have thoroughly critiqued (e.g., AAPA, 2019). Similarly, pandemic research on these topics varies widely in how they frame population groups (e.g., minority ethnic, Indigenous, BIPOC (North America), BAME (UK)). Often the categories used reflect the peculiarities of the available data and national context, which may conflate race, ethnicity, nationality, and linguistic distinctions. For example, the US census as well as research funded by the National Institutes of Health currently uses a classification system consisting of five racial and two ethnic categories (Kaneshiro, Geling, Gellert, &

Millar, 2011; NIH, 2001). The lack of standard definitions of race and ethnicity and the conflation of these concepts is a well-known problem in epidemiological research (Comstock, Castillo, & Lindsay, 2004; Kaneshiro et al., 2011; Lin & Kelsey, 2000).

Much of the literature, especially with the more recent pandemics, includes at least some discussion of socioeconomic or class issues, which are distinct from, yet deeply intertwined with, race and ethnicity, pointing to the importance of an intersectional approach to health inequalities (Bambra, Riordan, Ford, & Matthews, 2020; Gkiouleka, Huijts, Beckfield, & Bambra, 2018). Indeed, SES is by itself a major topic in health and disease studies. Mamelund and Dimka (2021) recently reviewed disparities based on SES during the 1918 influenza and COVID-19, and discussed how SES and other social factors including socially constructed race and ethnicity should be considered in the context of pandemic preparedness. Further, Nyland et al. (2015) suggested that disentangling the contributions of ethnicity and SES to influenza outcomes is a future research challenge. Accounting for the effects of SES and other confounding variables likely would, in many situations, indicate that it is social disadvantage that increases likelihood of exposure to influenza and outcome disparities; see, for example, Quinn et al. (2011), who found that racial differences in susceptibility to complications from H1N1 influenza reported in a nationally representative US survey were not seen after controlling for SES and other demographic variables. However, as with sex, much research in more clinical epidemiological and public health fields typically only briefly touches on such issues in discussion, while more frequently including race or ethnicity as analytical or control variables uncritically, reinforcing misperceptions of these categories as biologically real. Therefore, there is much need for biological anthropological work in this area.

5.3 | Pre-existing health and disability

The literature review for this section considers studies on broad general health topics such as underlying or pre-existing health, the immune system, stress, nutrition, and the microbiome. Additionally, research on specific chronic conditions, such as TB, diabetes, cardiovascular disease, asthma, and obesity, is reviewed. While such chronic diseases can fit the definition of disability as conditions or impairments of the body or mind that, as a result of the interaction of personal and environmental factors, limit activities and restrict social participation (CDC, 2020; WHO, 2021a), the term disability is often not applied to these conditions in lay usage or even the epidemiological literature discussed below. Thus, disability is considered separately, as many conditions more commonly recognized as disabilities–such as neurologic disorders, mobility and sensory impairments, intellectual and learning disabilities, and mental health conditions–are biologically, socially, and politically defined, and tend to be associated with histories of discrimination and differential treatment within health care systems and societies more generally.

5.3.1 | 1918 influenza

Overall, there has been very little research on these topics for the 1918 influenza pandemic, likely due to unavailability of data, especially at the individual level. Some pre-existing conditions may have gone undetected, the ways some conditions are diagnosed or understood have changed over time, and comorbidities may not have been recorded on death certificates at the time. For general health concerns, most relevant research has focused on the role of the immune system, typically with the aim to explain the W-shaped age mortality profile. The idea of a "honeymoon" period for children approximately aged 5–14 has been used to explain the relatively low mortality in these age groups from most infectious diseases including the 1918 pandemic compared to other age groups (e.g., Ahmed, Oldstone, & Palese, 2007; Mamelund, Haneberg, & Mjaaland, 2017). Similarly, protective immunity from exposure to previously circulating strains may explain rates observed for older age categories (Ahmed, Oldstone, & Palese, 2007). Further, priming of the immune system based on exposure to a different strain, such as the 1889 flu pandemic, in infancy may have led to a dysfunctional, pathogenic response, explaining the atypical high mortality among young adults (Gagnon et al., 2013; McAuley, Kedzierska, Brown, & Shanks, 2015; van Wijhe, Ingholt, Andreasen, & Simonsen, 2018). Most of these hypotheses rely on knowledge of immune system function and observations of broad trends, and are difficult, if not impossible, to empirically test.

Additionally, Mamelund (2011) considered nutritional status, alongside other proposed explanations, for mortality among Indigenous populations in selected areas of North America, Europe, and Oceania. He argued that nutrient-rich traditional Alaskan diets (e.g., fish and caribou) were likely beneficial to the immune system and so nutrition is not as plausible an explanation in some areas. Further, novel and innovative research using bioarchaeological analyses of skeletal lesions in documented assemblages tested the hypothesis that healthy adults were as likely to die as frail individuals during the pandemic. Analyses indicated that while more nonfrail individuals died during the pandemic compared to the prepandemic period, frail individuals still had lower survivorship and greater risk of mortality during the pandemic (Wissler, 2021).

Discussion of chronic diseases has largely been in a broad sense, such as the recognition that comorbid conditions helped form the backdrop of disease in different locations, contributing to the development of syndemics (Herring & Sattenspiel, 2007; Sattenspiel & Mamelund, 2012). However, as discussed above, much research has specifically investigated TB in relation to hypotheses about post-pandemic mortality trends (Noymer & Garenne, 2000; Sawchuk, 2009; van Doren & Sattenspiel, 2021), but it is so far unclear whether this interaction was active (i.e., a biological interaction of the pathogens and/or disease processes) rather than passive (i.e., increased TB mortality during the pandemic followed by decreased mortality in subsequent decades was more attributable to an overlap in age groups at risk of death from both diseases) (Noymer, 2009). Dahal et al. (2018) calculated excess mortality for TB during the pandemic using data from Arizona, which had a very high

prevalence of TB at the time due to the treatment-related benefits of its climate. Results showed a moderate rise in TB mortality during the fall and winter, with a notable increase among young adults in the fall wave, supporting the hypothesis that TB was a risk factor. In analysis of data from a Swiss sanatorium, Oei and Nishiura (2012) found a marginal association between TB and influenza case fatality, while there were no deaths among non-TB controls. Similarly, Mamelund and Dimka (2019) showed that while influenza morbidity was significantly higher among staff in Norwegian sanatoria, case fatality was significantly higher among the TB patients.

As with other health conditions, there is a dearth of studies considering disability as a risk factor during the 1918 pandemic. Using the same Norwegian data source as above, Dimka and Mamelund (2020) showed a similar morbidity-mortality crossover when comparing (presumed nondisabled) staff to resident patients at psychiatric institutions. High morbidity and mortality levels were also suggested by records of Norwegian schools for children with disabilities, although data were insufficient for statistical analyses (Dimka & Mamelund, 2020). In a nonpeer-reviewed blog post, Chamberlain (2020) reported that more than 80% of the population at a Pennsylvania school for children with intellectual disabilities fell ill, but any distinctions between staff and students were not clear. Further, while Zuckerman et al. (2022) analyzed mortality rates among institutionalized individuals in the Mississippi State Asylum, data were not available for comparisons to noninstitutionalized or other populations.

5.3.2 | 2009 influenza

Comparatively, a substantial body of work looked at differential outcomes based on health status during the 2009 pandemic. While very few of the identified publications investigated broad or general health topics, many studies focused on one or more specific chronic health conditions. For example, Campbell et al. (2011) found that cases with immunosuppression, and chronic kidney, neurological, and respiratory diseases were all individually associated with a 10–20-times increased risk for hospitalization, and patients with pre-existing conditions also had longer hospital stays. Similarly, the risk of admission to pediatric intensive care units for children in Ireland increased with the number of underlying conditions for both seasonal and pandemic influenza (Rebolledo et al., 2014). For patients over 16 years old in Sydney, Australia, after adjusting for age and sex, hospitalization risk factors included immune suppression, lung disease, heart disease, diabetes, and asthma. (Ward, Spokes, & McAnulty, 2011). In a sample of hospitalized patients from Montréal, diabetes tripled the risk of hospitalization and quadrupled the risk of ICU admission (Allard, Leclerc, Tremblay, & Tannenbaum, 2010).

Underlying health conditions were also associated with more severe outcomes and death. For example, in a global pooled sample of 70,000 lab-confirmed hospitalized patients, the proportion of cases with one or more chronic conditions increased across the severity levels of hospitalization, ICU admission, and death (Van Kerkhove et al., 2011). In a sample of nearly 10,000 patients in China, chronic medical conditions increased risk for severe illness across all age groups, with 33% of severe cases versus 14% of less severe cases reporting such conditions (Yu et al., 2011). However, Cortes Garcia et al. (2012) found that diabetes was not an independent risk factor for ICU admission or death in Spain, when compared to controls matched by age and sex. Similarly, national data from Spain showed that while the age-specific prevalence of diabetes was higher among hospitalized patients than the general population for most age groups, diabetes was not independently associated with dying while hospitalized (Jimenez-Garcia et al., 2013).

Research on this pandemic also indicated the need to consider a wider range of potential risk factors than those commonly recognized. For example, compared to children who died from seasonal flu in 2007–2009, children in the US who died of influenza during the 2009 pandemic were older (median of 9.4 vs. 6.2 years) and more had high-risk medical conditions (68% vs. 46% of those with information on pre-existing conditions). Notably, these analyses also investigated several conditions not then included on the Advisory Committee on Immunization Practices' (ACIP) recommendations for seasonal vaccination. These conditions, which generally had equal or higher prevalence than those that were identified as high risk, included gastrointestinal disorder, prematurity, scoliosis, and obesity (Cox, Blanton, Dhara, Brammer, & Finelli, 2011). Similarly, Louie, Jean et al. (2011) found that 80% of 541 fatal cases among Californian adults had comorbid conditions; older individuals were also more likely to have comorbidities not associated with severe flu by ACIP (e.g., chronic GI disease and hypertension). Louie, Acosta et al.'s (2011) analysis of outcomes among 534 adults was among the studies that identified obesity as a novel risk factor for influenza during this pandemic, showing that BMI ≥40 and BMI ≥45 were independently associated with death. Further, a systematic review pooling more than 25,000 lab-confirmed cases supported the association between obesity and increased risk of critical and fatal complications despite some potential confounding from treatment (Sun et al., 2016).

Fewer studies on the 2009 pandemic have looked at disabilities, with much work documenting disparities for children with neurologic diseases or intellectual disabilities. Out of 48 US children who died between April 17 and July 23, 69% had a reported underlying condition; of these, neurologic disorders including cerebral palsy, developmental delay, and Down syndrome were the most common category of conditions, at 79% of deaths (Fowlkes et al., 2011). Of 336 child deaths reported to the CDC with information on underlying health (98% of all deaths reported for the pandemic period of April 15, 2009, to September 30, 2010), 43% had neurologic disorders and 68% had one or more high-risk condition in general (Blanton et al., 2012). Hospitalization also was more frequent for children with such conditions (Launes et al., 2012). Analysis of pandemic influenza patients from Mexico showed that Down syndrome was associated with a 16-fold and 335-fold increased likelihood of hospitalization and death, respectively (Perez-Padilla et al., 2010). Indeed, Bettinger et al. (2010) noted that underlying neurologic or developmental conditions were added to the list for vaccination

recommendations in Canada in part because of data from hospitalized Canadian children during the 2009 pandemic.

5.3.3 | COVID-19

Some work to date on COVID-19 outcomes has considered measures of general health, including stress, nutrition, and characteristics of the microbiome. For example, Ramezani et al. (2020) found that levels of serum cortisol and scores on the Hospital Anxiety and Depression Scale were higher in patients who died than those who survived, even though all presented with mild to moderate cases. Using Controlling Nutritional Status scores to assess diagnosed patients, Zhou, Ma et al. (2021) found that, among other variables, nutritional status was a risk factor for adverse outcomes. Studies also have shown dysbiosis of the intestinal microbiome in correlation with COVID-19 severity, where opportunistic pathogens are enriched and beneficial symbionts are depleted (Finlay et al., 2021; Scaldaferri et al., 2020; Zuo et al., 2020). In a preprint manuscript, Hurst et al. (2021) report that nasopharyngeal microbiomes also show patterns associated with COVID-19. Species abundance varies with infection and presence or absence of symptoms, and is independently associated with age. Since the upper respiratory microbiome changes during early childhood, this finding may help explain lower susceptibility to infection and severity of cases for younger age groups observed early in the pandemic (Hurst et al., 2021).

There are also higher risks associated with specific chronic medical conditions. Logistic regression on monthly US Medicare data for hospitalization rates per 100 COVID-19 Medicare beneficiaries from January through September 2020 showed hospitalization was associated with all but two of 27 selected chronic health, mental health, and substance abuse conditions, adjusted for age, sex, race and ethnicity, and urban–rural residence (Chang, Moonesinghe, & Truman, 2021). Among hospitalized patients in Portugal, respiratory, cardiovascular, and renal diseases were associated with mortality and ICU admission; diabetes and cancer were also associated with serious outcomes when all cases in the country were analyzed (Laires et al., 2021). Systematic reviews and meta-analyses have consistently shown higher risks of more severe cases associated with obesity, hypertension, cardiovascular disease, respiratory disease, and diabetes (Matsushita et al., 2020; Popkin et al., 2020; Yang et al., 2020).

In terms of mortality, a systematic review of 25 studies involving a pooled sample of more than 65,000 patients found that conditions significantly associated with mortality risk included cardiovascular disease, hypertension, diabetes, congestive heart failure, chronic kidney disease, and cancer (Ssentongo, Ssentongo, Heilbrunn, Ba, & Chinchilli, 2020). Further, Wortham et al. (2020) analyzed deaths reported in the US between February 12 and May 18, 2020, and found that, of those with available information, about 83% of deaths among individuals younger than 65 years had one or more underlying conditions. They noted that missing data was higher for this variable than others included in the analyses, so results should be considered minimum estimates. Additionally, case–control analyses for a sample from Sardinia showed that, out of 90 deceased cases with information on underlying health (93% of total deaths), 89 had at least one underlying condition or risk factor, most commonly cardiovascular, chronic neurological, and chronic lung diseases, and diabetes mellitus (Deiana et al., 2020). Systematic reviews and meta-analyses focused on diabetes showed that this condition was associated with severe outcomes; for example, Kumar et al. (2020) found a two-fold increase in mortality and Mantovani, Byrne, Zheng, and Targher (2020) reported an approximately threefold increased risk of in-hospital mortality. Kumar et al. (2020) cautioned, however, that they could not conclude whether diabetes was an independent risk factor. Mortality was independently associated with obesity, however. For example, for hospitalized patients younger than 50, the odds ratio for mortality was 5.1 with BMI ≥40. Although this association was also significant for those older than 50, the odds ratio was lower at 1.6 (Klang et al., 2020).

Research on other conditions has found inconsistent or nonsignificant results. For example, Gao et al. (2021) reported in a letter to the editor that a meta-analysis of six studies from China indicated that TB was more prevalent, but not significantly so, in severe versus nonsevere cases, and was not associated with an increased mortality risk, although sample size could be an issue. However, risk of death was more than two times higher in patients from the Philippines with TB than in those without, and the time to death was also significantly shorter, while the time to recovery was significantly longer (Sy, Haw, & Uy, 2020). Further, some analyses found that, unlike with influenza, individuals with asthma were not more likely to have severe cases requiring hospitalization or intubation (Broadhurst et al., 2020). A meta-analysis of 11 studies with a pooled sample of nearly 108,000 COVID-19 patients showed that those with asthma were more likely to be younger and obese, had an increased risk of intubation especially if <50 years old, and had longer hospital stays if <65; however, there were no significant differences in terms of comorbidities, laboratory testing, hospitalization rates, ICU admission or development of acute respiratory distress syndrome between the groups. Additionally, asthmatic patients had better recovery and were less likely to die than nonasthmatic patients (Hussein et al., 2021).

Although under-addressed relative to other noninfectious, preexisting conditions, several studies have looked at disability as a risk factor for COVID-19. For example, the presence of chronic neurologic disorders was an independent predictor of death but not of more severe disease among a retrospective cohort of hospitalized patients in Spain, with 44.8% versus 17% mortality. Analyses indicated that the higher death rate could not be explained by a worse immune response, differences in care, or delayed presentation in emergency departments (Garcia-Azorin et al., 2020). Most identified studies have focused on intellectual and developmental disabilities (IDD), with an emphasis on institutionalized populations. In a descriptive study of 66 deaths among people with intellectual disability, approximately a third had Down syndrome and 55 lived in supported living, residential, or nursing homes (Perera et al., 2020). While Californians receiving IDD services had a lower case rate than those who did not, the case fatality rate was 2.8 times higher, with results varying significantly based on type of residence. Individuals living on their own or in a

family home had the least severe outcomes. There were higher case rates in types with more residents, likely related to exposure, and higher case fatality and mortality rates in those with 24-hour skilled nursing care, perhaps because residents in these settings have more severe disabilities and health conditions. However, the authors noted that information about age, intensity of services, or pre-existing conditions were not available, so availability of skilled nursing care served only as a proxy measure (Landes, Turk, & Wong, 2021). Case fatality rates were also higher for people with IDD living in residential group homes in New York (15%) compared to the general population of the state (7.9%) (Landes, Turk, Formica, McDonald, & Stevens, 2020).

Studies including noninstitutionalized populations also show disparities. Gleason et al. (2021) conducted a cross-sectional study across 547 health care organizations in the US from January 2019 through November 2020 and found that intellectual disability was the strongest independent risk factor for being diagnosed with COVID-19 and the strongest other than age for death (Gleason et al., 2021). The UK Office for National Statistics investigated mortality between January and November 2020 in England based on self-reported level of limitation of daily activities. Risk of death was 3.1 times greater for more disabled men ("limited a lot") and 1.9 times greater for less disabled men ("limited a little") compared to nondisabled. For women, the risks were 3.5 and 2.0 times greater, respectively. Differences were reduced but still significant for all but less disabled men after controlling for several variables including residence type and demographic and socioeconomic measures, suggesting that a range of factors jointly contribute to the increased risk of death. However, the largest effect for those with learning disabilities was associated with communal residence (Ayoubkhani & Bosworth, 2021). Further, Turk, Landes, Formica, and Goss (2020) investigated trends among more than 30,000 patients, 474 of whom had a recorded IDD, using electronic medical records from the global TriNetX COVID-19 Research Network platform, and noted distinctive age differences. Although peak percentages of overall deaths were similar for both groups, this peak occurred in the 18–74 age range for those with IDD and the 75+ range for those without. The peak percentage of cases was higher for those with IDD in the 0–17 age range (26.4% vs. 2.7%) and lower in the 75+ range (8% vs. 15.3%), and case fatality rates also showed an age-related bias (1.6% among 0–17 with IDD vs. <0.1% without, 4.5% among 18–74 with IDD vs. 2.7% without) (Turk et al., 2020). As noted earlier, pandemics often have atypical age-specific mortality curves; this study as well as others that indicate chronic health conditions increase risks in younger adult age groups may help explain these patterns.

This limited body of work clearly indicates the need for more research on different kinds of disabilities. Provocatively, Zhou, Liu, Sun, Huang, and Ye (2021) explored associations of test-confirmed COVID-19 with an array of nearly 1000 medical conditions, 30 blood biomarkers, and genetic variants for two genes related to SARS-CoV-2 infection, for 389,620 participants in the UK Biobank, a large, population-based prospective study. Results showed that the most significant risk factors for COVID-19 included Alzheimer's disease, dementia, and an overall category of delirium, dementia, amnestic, and other cognitive disorders. Associations were also found with several other, novel risk factors. However, the authors stressed that the study identified associations only; determining possible mechanisms is more complicated (Zhou, Liu et al., 2021).

5.3.4 | Summary and observations

Research on disparities in outcomes related to pre-existing health status increased dramatically for the two later pandemics, most likely due to the availability of surveillance data as well as the high prevalence of relevant conditions especially in high-income countries where many of the epidemiological studies have been performed. Probably for similar reasons, most studies also focus on hospitalized patients and/or lab-confirmed cases; many mild or asymptomatic cases might be excluded from analyses, as well as cases in populations that have no or less access to testing or health care resources. Nonetheless, results show consistent trends of worse outcomes including severe illness and death for individuals with poorer general health and specific chronic conditions, although not all studies were able to demonstrate independent associations after controlling for other factors.

Although health conditions may seem, at first glance, clearly defined or distinguishable, social factors cross-culturally and over time influence the diagnosis, surveillance, and treatment of different conditions. For example, in addition to numerous critiques against some measures of obesity such as BMI, risks associated with obesity may partially or fully reflect delays in care due to discrimination or sizeism. Additionally, data collection practices may vary for different studies (e.g., self-reported survey data or biomedical diagnoses reported by health care professionals). As Gleason et al. (2021) noted, some conditions, such as many disabilities, may be underreported. Studies on disparities related to underlying health also tend to focus on conditions previously identified as risk factors, which as Zhou, Liu et al. (2021) observed, may be due to ease of data collection, clinical experience, and prior publications. Yet, the examples of neurologic disorders and obesity during the 2009 pandemic reveal that some conditions have become, or become recognized as, risk factors. An emphasis on certain risk factors means that other conditions and affected populations may be overlooked in health research, preparedness planning, and public health responses (e.g., Landes, Stevens, & Turk, 2020; Landes, Turk, Formica, McDonald, & Stevens, 2020; Landes et al., 2021); to quote Reed, Meeks, and Swenor (2020: e423), "who counts depends on who is counted."

This review suggests several opportunities for future research, including identification and analysis of historical health data for the 1918 flu and other past pandemics, development of methods for studying nonhospitalized populations, and work on both general health measures of interest to human biologists (e.g., stress or nutrition) and chronic medical conditions and disabilities that are traditionally less studied. In particular, studies considering disability are relatively rare in both pandemic research and biological anthropology, arguably with the exception of bioarchaeology of care research

(see Tilley & Schrenk, 2017); further, such studies rarely apply relevant theoretical perspectives from disability studies (Battles & Gilmour, 2022).

6 | DISCUSSION

The above review demonstrates consistent patterns of morbidity and mortality disparities based on sex/gender, race/ethnicity, and pre-existing health/disability across three large pandemics. At the same time, various studies showed differences in the magnitude or statistical significance of disparities and in some cases their direction, for example whether males or females were more at risk in different regions. Such findings suggest that while the proximate determinants of pandemics, such as the causative pathogen, may change, ultimate determinants, likely including both social factors such as inequalities and some biological differences in host characteristics that predispose individuals for severe cases, remain relatively consistent. Pandemics are not biological events independent of social context, and compartmentalized discussion of biological and social issues should be avoided (Fuentes, 2020). Yet, despite ideally situated expertise for addressing these relationships, research on pandemics has not been a major focus relative to other health-related topics in biological anthropology.

This limited emphasis may be due to several reasons. Many recent large pandemics have been caused by influenza, which has also been the most likely candidate in pandemic preparedness. Although seasonal influenza causes substantial illness, death, and social and economic costs every year (Molinari et al., 2007), it also is often perceived as commonplace. Additionally, the SARS outbreak in 2002–2004 and the 2009 flu pandemic were *relatively* minor, or at least not as severe as feared. Therefore, investigation into epidemics and pandemics of influenza and similar diseases may not seem as urgent or attractive to researchers and funding bodies compared to other acute, emerging, and chronic conditions, or at least available research funding has not been significantly extended to the social sciences. Similarly, data on such pandemics are also easier to obtain for historical events, and studies of historical health and demography seem relatively undervalued or underemphasized in biological anthropology, bioarchaeology, and human biology compared to prehistoric or living populations.

On a more practical note, while pandemics are not completely unexpected, the exact emergence and timing of them cannot be predicted, so research activities and funding may be difficult to put into place when they occur. Biological anthropologists are also affected by travel restrictions and the massive disruptions to work and home life that typically accompany large pandemics, limiting the potential for real-time, in-person research. This limitation is further exacerbated by the ethical concerns associated with conducting anthropological research with at-risk populations during an ongoing pandemic. Finally, academic research and scientific publishing in general and in anthropology specifically tends to be a slow process, even with preprints made available online and emergency funding calls. Nonetheless, the attention shown by biological anthropologists to COVID-19, including commentary pieces and (social) media engagement, suggests that pandemics are likely to be of greater interest moving forward.

A fundamental step in the development of pandemic studies in biological anthropology will be stronger theorization of what constitutes a pandemic and how best to study one, such as how data is collected and analyzed. Pandemics are global and anthropogenic in nature and can significantly impact and interact with all aspects of human societies and biology. Anthropologists will need to consider whether, how, and when to integrate environmental, epidemiological, viral, genetic, individual, social, political, economic, and other data, in order to conceptualize and investigate these phenomena as distinct from infectious diseases or epidemics more broadly. Historical influences and trends; ecological interactions between humans, animals, and the environment; and issues of globalization and an interconnected world need to be considered, as well as the importance of foregrounding the experiences and potential vulnerabilities of marginalized communities. One of the most important contributions anthropologists can make to interdisciplinary pandemic studies is to investigate the nuanced, interacting, holistic nature of pandemics within societies, cross-culturally, and over time, such as the ways that global emergencies manifest differently in local contexts and why. We conclude this paper with discussion of several potential research directions and important considerations.

6.1 | Considerations for biological anthropology research in pandemic studies

In addition to the research directions we have highlighted in this review, numerous other angles of interdisciplinary research on the causes and consequences of pandemics are clearly of interest and relevance to anthropology. For example, with more recent pandemics, the global economic and political issues surrounding the development, production, and unequal distribution of vaccines require critical analysis. Even with research specifically on sex/gender, race/ethnicity, and underlying health, other issues besides morbidity and mortality are apparent and indeed likely play a role in differential outcomes. For example, pandemics have gendered impacts in terms of employment and economic costs, childcare and household labor, and domestic violence (Wenham, Smith, Davies et al., 2020; Wenham, Smith, & Morgan, 2020). Stigma and blame are often racialized, as seen with reports of anti-Asian bias during COVID-19 (Dionne, Hayes, & Turkmen, 2021). Further, disease exposure risks and economic and employment losses also follow racial and ethnic lines, as there is a larger presence of minority employees in essential positions or occupations that face more exposure (e.g., Hawkins, 2020). With regards to pre-existing health, much bioethical debate has centered around the allocation of limited resources based on perceptions of "quality of life" (e.g., Panocchia et al., 2021). Even pandemics of diseases with, on average, short-term illness can have long-term health consequences from the effects

of the disease itself, treatments, and public health interventions that might reduce accessibility and social support.

Rather than exploring these and other specific questions here, we instead raise broader issues that may provide inspiration for researchers to identify the ways in which their particular interests, theoretical frameworks, and methods may be used. Specifically, we discuss investigation of historical and cross-cultural context, more engagement with the syndemics framework, the social and health impacts of stress and stigma, and important methodological and ethical issues.

6.1.1 | Historical depth and cross-cultural context

Singer and Rylko-Bauer (2021, p. 23) recently wrote that without commitments and efforts in global health towards, for example, prevention and preparation, upstream causes, local specificities, and equity, "we face the possibility of a world where pandemics–like environmental disasters–become recurring events…." While this call for a paradigm shift is apt, pandemics like COVID-19 already are recurring events and will continue to be a threat into the future, even if health and economic inequalities can be reduced. Considering influenza-like illnesses alone, there have been milder pandemics beginning in 1957 and 1968, in addition to the 1889, 1918, and 2009 pandemics already mentioned, as well as smaller events not typically classified as pandemics in 1947, 1976, and 1977 (Kilbourne, 2006). School closures and other major interventions also have been associated with outbreaks of other diseases such as polio and measles (Champredon, Shoukat, Singer, Galvani, & Moghadas, 2020; Meyers & Thomasson, 2020), while recent emerging infectious diseases that may have or develop epidemic/pandemic potential have spread to at least a few regions or countries (e.g., Ebola, Zika, and Middle East Respiratory Syndrome) (CEPI, 2021; Gavi, 2021). As Gaddy (2020) noted, anthropogenic climate change is associated with vector-borne and other emerging infectious diseases, and the barriers between host species are likely lower than thought and will fall further as a result of ecological change and human encroachment into relatively untouched natural habitats. Even controlling for reporting effort, the number of emerging infectious disease events (defined as the original case or cluster of cases in human populations, and including re-emerging diseases, such as multi-drug resistant TB, and pathogens that have probably affected humans for some time but have recently increased in incidence or been identified) increased per decade between 1940 and 2004, with a peak in the 1980s associated with HIV/AIDS (Jones et al., 2008). Further, antimicrobial resistance and vaccine hesitancy or anti-vaccination movements have contributed to resurgences and outbreaks of re-emerging diseases (Harper & Armelagos, 2010). It is no wonder that pandemic preparedness planners routinely warned that another global pandemic was a matter of when, not if, and such issues highlight the importance of learning from the past.

Past infectious disease pandemics have resulted in significant morbidity and mortality from the diseases themselves and potentially from effects of public health responses and social disruptions. Lower levels of screening and treatment for other health conditions due to reduced health care access and resources or increased avoidance (Czeisler et al., 2020; Findling, Blendon, & Benson, 2020), or loss of prime-aged caretakers of younger or older individuals (Mamelund et al., 2013), also may contribute to excess morbidity or mortality associated with pandemics. These impacts, as well as long-term effects of illness or treatment, from one wave or pandemic help shape the population at risk for the next. Further, pandemics can affect fertility possibly through effects of infection but also through social and economic impacts, leading to baby booms or busts (Aassve, Cavalli, Mencarini, Plach, & Livi Bacci, 2020; Mamelund, 2004), and have potential transgenerational effects through impacts on maternal-child health (Bogin & Varea, 2020; Gildner & Thayer, 2020). Short- and long-term effects incorporating perspectives such as the Developmental Origins of Health and Disease (DOHaD) and epigenetics are therefore important topics of consideration that can only be fully studied by drawing on data from past pandemics. For example, previous research has hinted that those who were in utero during the 1918 influenza pandemic had lower SES, lower educational attainment, and increased rates of disability compared to other birth cohorts (Almond, 2006; Helgertz & Bengtsson, 2019). Historically-oriented studies that recognize the recurring pattern of pandemics and the potential for interactions of social and health factors during and between them are important for understanding demographic and ultimately evolutionary change in human populations.

Additionally, attention to historical and cross-cultural context is important for understanding patterns of spread and disparities in outcomes, as pandemics unsurprisingly often go hand-in-hand with other large-scale political, environmental, and/or social disruption. The obvious disruption of World War I may help explain variation in 1918 pandemic mortality between males and females. For example, mortality rates among males aged 15–44 were higher than those for females in areas of France farther away from the front, possibly because the men who remained in those areas during the war were more likely to be deemed medically unfit for the military and so more vulnerable to infection or complications. On the other hand, areas closer to the front or in neutral countries showed smaller differences or higher mortality among females (Zylberman, 2003). More recently, the spread and experiences of COVID-19 in the US, such as higher mortality among Black populations, cannot be easily separated from either the country's history of systemic racism or large-scale protests fueled by Black Lives Matter/racial justice issues particularly following the murder of George Floyd. Similar arguments can be made for different vaccination rates by state (Kates, Tolbert, & Orgera, 2021) and political divisions during the 2020 elections. Divorcing pandemics from their historical and social influences limits understanding of their determinants and consequences. On the contrary, consideration of such contexts may help disentangle contributing factors and elucidate consistent biological and social trends; here, more research into the relatively under-studied and milder pandemics of the mid-20th century may offer important insights.

Genetic and bioarchaeological analyses have been important means of researching infectious diseases and pandemics in prehistoric

populations, and paleodemographic methods can be used to possibly differentiate attritional death profiles from catastrophic ones related to pandemics even in the absence of other archaeological or historical evidence (Castex & Kacki, 2016; Margerison & Knüsel, 2002). Additionally, samples stored at the Armed Forces Institute of Pathology and recovered from an Inuit woman buried in permafrost in Alaska enabled isolation, identification, and analysis of the 1918 flu virus (Taubenberger, 2003; Taubenberger, Reid, Janczewski, & Fanning, 2001). As mentioned previously, bioarchaeological analyses were also used to assess health of individuals in documented skeletal collections who died before, during, and after 1918 (Wissler, 2021). These examples suggest such approaches are suitable for identifying and studying more recent as well as older pandemics.

In addition, anthropological traditions of cross-cultural comparative research and attention to smaller and geographically diverse populations would provide important insights into studies on morbidity, mortality, and other aspects of pandemics. As can be seen in the review above, the vast majority of pandemic research focuses on samples from North America; northern and western Europe, especially the UK and Norway; China and other East Asian countries; and to a lesser extent, Oceania. In contrast, fewer, if any, of the identified studies on disparities in outcomes during historical pandemics or COVID-19 have focused on, for example, countries or regions in Africa, southern and eastern Europe, and other parts of Asia. While some of this underrepresentation may be due to language barriers or literature search algorithms, there is nonetheless a lack, particularly from areas that historically have not had adequate disease surveillance and maintenance of vital records. Pandemics do not respect national borders, and the friction between international cooperation (e.g., in public health prevention and interventions) and national self-interest is an important consideration in understanding unequal outcomes, especially between low- and high-income countries. Even within countries, ethnic minorities or other subgroups may have highly dissimilar outcomes from the national averages, while differences along official boundaries such as state lines and more abstract boundaries such as rural versus urban divides also are important to consider.

6.1.2 | Syndemics

The theoretical perspective of syndemics draws specific attention to the ways that multiple health conditions and deleterious social contexts synergistically interact to enhance poor health outcomes. This perspective is also inherently multi-level, considering both individual- and population-level interactions. Gravlee (2020) recently applied this concept to illustrate how, in the context of systemic racism and inequities in social and economic conditions, a higher burden of health conditions like hypertension and diabetes contributes to disparities in outcomes during COVID-19, while the pandemic also can exacerbate these diseases in Black people. However, in offering a framework addressing causal pathways from large-scale social forces to individual biology, Gravlee (2020) also noted conceptual and methodological challenges with syndemics theory, including the complexities involved with levels of analysis and generating testable hypotheses.

Indeed, in a recent scoping review of nearly 200 publications addressing syndemics, Singer et al. (2020) concluded that only 12% of the articles met the full criteria for true syndemics. Discussion of the other categories (e.g., potential syndemics, or harmful disease clusters) indicated that many of the remaining articles tended to fail in the biological aspects. For example, the authors noted that publications classified as potential syndemics did not fully articulate biological or biological-social relationships or mechanisms of interaction. This finding represents a clear avenue for how biological anthropologists may contribute to the syndemics literature, including those involving pandemic diseases. Physiological mechanisms underlying biological interactions, especially those involving communicable diseases, are often easier to identify and describe than biosocial interactions (Singer et al., 2020). For example, lung tissue damaged by TB provides additional surface for the influenza virus to take hold, which helps explain the associations between TB and 1918 influenza mortality (Shanks & Brundage, 2012). Singer et al.'s (2021) identification of the usefulness of local biologies for syndemics suggests one approach for investigating biosocial interactions, as the concept incorporates ideas from evolutionary and developmental biology that will be familiar to biological anthropologists (e.g., developmental plasticity and epigenetics). Similarly, embodiment of stressors related to, for example, stigma and discrimination is one of the more promising avenues of research.

6.1.3 | Stigma, stress, and intersectionality

Throughout history, infectious diseases, epidemics, and pandemics have been blamed on or associated with marginalized groups such as racial and ethnic minorities, immigrants, and members of different religions. Examples include Jewish people during the Black Death, the Chinese in San Francisco with smallpox and plague, immigrants in the US with multiple diseases including TB, men who have sex with men and other high-risk populations with HIV/AIDS, and most recently anti-Asian bias and discrimination during COVID-19 (e.g., Bhanot, Singh, Verma, & Sharad, 2020; Cohn, 2007; Craddock, 1995, 1999; Dionne, Hayes, & Turkmen, 2021; Kutner et al., 2020; Markel & Stern, 2002; Shah, 2001). Heightened stigma during pandemics, as well as longer-term systemic racism and other institutional biases, may lead to biological outcomes of disease and death through several pathways, including reduced access to or avoidance of health care, compounded by inadequate care and services due to discrimination within health care systems, as well as violence and embodiment (Brewis, Wutich, & Mahdavi, 2020). Work on embodiment in biological anthropology and social epidemiology has particularly focused on the ways that individual and generational experiences of stress and discrimination, particularly related to race, can get "under the skin", contributing to disproportionately poorer outcomes for a range of biological and population health measures (Gravlee, 2009, 2020; Krieger, 1999).

As Brewis et al. (2020) observed, stigma can produce physiological responses, which while not well studied in the context of infectious diseases, likely drive or contribute to health disparities. Such responses might make individuals more prone to infection or complications or result in slower recovery. Our review corroborates that stress, as well as general health measures like nutrition, have not been well-studied with respect to outcomes during pandemics. Measures of relevant biomarkers therefore represent important research opportunities for biological anthropology and human biology. Brewis et al. (2020) also highlighted the importance of developing scales that measure social dimensions such as the impacts of stigma in space and time during epidemics.

Further, inaccurate associations of pandemic diseases with specific risk groups, including through media coverage, can prolong their spread and create further illness and death among majority and nonmarginalized populations. Members of such populations may not realize or accept that they are at risk, and thus ignore symptoms and neglect or refuse to properly follow public health recommendations. Moreover, authorities and governments may receive less pressure from constituents or approach diseases with less urgency and fewer resources if they are seen as problems restricted to minority populations with limited political power (e.g., Brewis et al., 2020).

Finally, in contrast to syndemics articles which often lack full discussion of biological mechanisms and pathways, epidemiological literature often lacks full discussion of social variables, particularly in journals for clinical or medical sciences that are overwhelmingly prolific producers of such literature and likely to strongly influence public health practices and responses. Indeed, out of 14,588 publications about COVID-19 retrieved from the Scopus database between January and May 2020, 75% were categorized into broad subjects of medicine, biochemistry and molecular biology, and immunology and microbiology, while only 4% were classified as social sciences research (Pal, 2021). As noted above, variables such as sex and race are typically treated uncritically as biological realities in epidemiological research. In addition to giving short shrift to related social factors that might produce or explain disparities, this approach also leads to the neglect of populations that do not necessarily correspond with categories commonly used in analyses, such as some intersex, nonbinary, and LGBTQ+ individuals. Further, many epidemiological studies aim to assess *independent* associations of these and other variables (e.g., SES) with health disparities. However, intersectionality perspectives recognize how multiple systems of oppression interact to create different health experiences and outcomes (Cho, Crenshaw, & McCall, 2013; Gkiouleka et al., 2018; Vaiou, 2018). For example, morbidity and mortality rates of disabled Black women are likely to be markedly different from nondisabled White men in ways that cannot be easily or fully understood by considering each of those aspects independently. Anthropologists must not only continue to critique concepts of race, gender, disability, and what constitutes "good health" (including that disability does not inherently mean poor health), but also help develop theoretical approaches and data collection and analysis methods that will enable better investigation of such intersectional issues during pandemics (see Yaussy, 2022, for a recent discussion of intersectionality in bioarchaeological research on pandemics).

6.1.4 | Data collection and ethics

As a field, biological anthropology allows for interdisciplinary tools to investigate and integrate a wide array of data on factors underlying morbidity and mortality disparities. Expertise in the field extends across multiple scales from genetic analyses through individual-, local-, and population-level variation to long-term structural and institutional systems and, ultimately, human evolution. This broad skill set, combined frequently with training in four-field anthropology and thus sociocultural aspects of human health, opens opportunities for the development and application of numerous methodological approaches. We highlight here a few key issues of data collection, analysis, and communication relevant to biological anthropological studies of pandemics.

First, as can be seen from the above review, much research on recent pandemics draws on data from hospitalized and/or lab-confirmed cases. In addition to producing a bias towards more severe cases versus mild or asymptomatic ones, this focus also creates a bias in terms of which members of populations are more likely to have access to or be able to afford testing, treatment, and other health care services. Similarly, in archival research on historical pandemics, there is typically an over-reliance on censuses and death records, which constitute "accidental" datasets, meaning they were created for purposes other than for which modern quantitative analyses use them (Swedlund & Herring, 2003). Issues inherent in archival research may include loss and damage of records, inaccurate or limited medical knowledge of the time, and under-representation of segments of the population (e.g., the poor, illiterate, or migrants, as well as women and children). Further, considering the stigma associated with some infectious diseases, recorded causes of death may even be purposely inaccurate in some cases (e.g., Hardy, 1993, on TB, and Szreter, 2014, on syphilis in the 19th and early 20th centuries). Biological anthropologists can enhance the range and quality of data used in analyses by developing field-based and other methods for collecting information on nonhospitalized cases and unrecorded deaths; evaluating potential sources of bias; and investigating local understandings of health, diagnosis, and treatment of disease, and measures of potential associated factors like SES (e.g., Weaver & Kaiser, 2020). For example, McDade and Sancilio (2020) discussed suitable field-based tests for antibodies that can identify mild or asymptomatic cases, important for both immediate epidemiological purposes as well as for broader research incorporating biosocial frameworks, environmental data, and life history approaches.

On the other hand, field-based data collection during active pandemics raises important concerns. While real-time data collection and analysis may provide substantial benefits to communities in terms of public health surveillance and responses, biological anthropologists have a far greater ethical obligation to the communities in which they

work not to introduce infectious diseases or contribute to their spread. This concern is particularly true considering many, although certainly not all, anthropologists traditionally live in and travel from higher-income areas with adequate health care resources to conduct research in smaller, less privileged communities. Even if travel is not restricted during a pandemic, biological anthropologists should explore and develop alternative modes of data collection, including but not limited to interviews via video conferencing, analyses of digitized data, and collaboration with local researchers.

Similar to concerns raised with other areas of anthropological research (e.g., issues related to NAGPRA) and in line with long-held calls for inclusion such as the phrase "nothing about us without us" common in disability communities, research on pandemics should include the voices, expertise, and active participation of relevant populations. Activities to facilitate this participation include recruiting and training students, working with and crediting collaborators, and listening and responding to the views and concerns of nonacademic members of the populations. Concerns about data ownership or sovereignty (e.g., local knowledge about prevention and treatment of infectious diseases), especially with regards to Indigenous knowledge and data, must be carefully considered, as should how best to communicate research results so they can be beneficially applied to affected populations, while avoiding misrepresentation or misunderstanding by others (Tsosie, Yracheta, Kolepenuk, & Smith, 2021). As our review demonstrates, marginalized communities are historically and consistently more likely to become ill and die during pandemics, as well as suffer other social and economic consequences. Biological anthropologists involved in pandemic studies must therefore pay close attention to the ethical issues involved in researching disparities involving these individuals and communities.

7 | CONCLUSION

Human biology, behavior, and the environment, along with historical, political, and economic influences, contribute to the emergence and spread of pandemics, as well as marked disparities in morbidity and mortality outcomes within and between different populations. Climate change, globalization, the threat of emerging infectious diseases, socioeconomic inequalities, host-pathogen co-evolution, and other factors make it likely that acute infectious disease pandemics will continue to occur. The complex, multi-level components of pandemics cannot be addressed by single fields and instead require inter- and transdisciplinary research incorporating not just clinical medicine, epidemiology, and microbiology, but also history, demography, and other social sciences. Biological anthropologists are uniquely suited to cross the boundaries of different fields while also "speaking the language" of more traditional health workers and medical researchers involved in the development of public health policy and practice. A (biological) anthropology of pandemics forces us to acknowledge that we must consider the biological, social, and epidemiological factors on multiple scales from the individual to the global, while also comparing pandemic determinants, outcomes, and long-term consequences cross-culturally and historically. The COVID-19 pandemic has reinforced observations of persistent health disparities while also revealing weaknesses in understanding and appreciating the sheer scale of pandemics and the role of humans in such global health emergencies. As COVID-19 cases and deaths hopefully decline, more and more discussion in the media and daily life turns to the question of how and whether people's lives will and should go "back to normal." One previous normal that should be avoided is the relative neglect of epidemics and pandemics in biological anthropology.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

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