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Post-pandemic Inequalities: Evolutionary Anthropological Frameworks for Long-Term Impacts of the 1918 Influenza Pandemic

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ABSTRACT

The 1918 influenza pandemic was a major mortality event that is well understood in its proximate heterogeneous impacts, but its long-term impacts on inequality are less understood. Within anthropology, evolutionary frameworks such as the epidemiological transitions, biocultural anthropology, and evolutionary medicine can give meaning to ultimate explanations for pandemics' long-term consequences. I seek to identify and shape the gap in the 1918 influenza pandemic literature around the analysis of post-pandemic inequalities compared with pre-pandemic and pandemic period inequalities. I discuss six papers that address consequences on the demography and epidemiology of surviving populations and 11 papers that engage with the fetal origins hypothesis to understand unequal long-term impacts on cohorts exposed to stressful intrauterine environments during the pandemic. I contextualize existing knowledge of unequal impacts within evolutionary anthropological theory and argue that evolutionary anthropology is well suited to lead holistic research on ultimate determinants of long-term pandemic consequences.

1 | Introduction

The 1918 influenza pandemic is often considered the worst-case scenario for infectious respiratory disease pandemics and was one of the deadliest infectious disease events in human history. Despite its magnitude, the 1918 influenza pandemic has often been called "The Forgotten Pandemic" [1], and after the initial wave of research published in the decade following the pandemic's end, there was very little research published until the 21st century. More insights into this pandemic's origins, nature, and impacts have come to light since its centennial in 2018 and the onset of the COVID-19 pandemic in 2020. The influx of 1918 influenza pandemic research into many fields, including epidemiology (see Viboud and Lessler [2], and the corresponding special centennial issue in *American Journal of Epidemiology*), history [3], anthropology (e.g., [4–6]), and demography [7, 8], has brought one fact into sharper focus: the ways populations globally experienced this pandemic were highly variable, with many determinants contributing to that variation.

One feature of the 1918 influenza pandemic that has not been largely investigated is its long-term impacts, especially in populations that are known to have highly unequal experiences in sex- and age-based mortality, gradients of pandemic outcomes by socioeconomic status, differences among people with underlying chronic and/or infectious conditions, and more. As a relatively short and inarguably significant mortality event, the 1918 influenza pandemic likely had the potential to shape demographic composition and at-risk populations in the years following the end of the epidemic curve. However, while large portions of the 1918 influenza pandemic literature are dedicated to identifying unequal pandemic experiences, very little research has been dedicated to identifying changes in inequalities *after* the pandemic concluded. DeWitte and Wissler [9]

review the existing literature on detectable shifts in demographic structure and even some genetic consequences of the 1918 influenza pandemic, and the fetal origins hypothesis has been a point of interest for several decades, but post-pandemic inequalities as a consequence of mortality impacts have been relatively understudied.

In this paper, I do not seek to exclusively review the literature on inequalities during the 1918 influenza pandemic, as there have been several excellent reviews in recent years to which readers may refer for a thorough treatment of this topic (e.g., [9–14]). My aim is rather to identify and shape the gap in the literature related to inequalities *after* the 1918 influenza pandemic, specifically by discussing research that has addressed this topic directly. Specifically, I describe and engage with literature related to inequalities in mortality, morbidity, and survivorship after the pandemic, as well as the fetal origins of adult health and inequality that could be linked to exposure to a stressful *in utero* environment during the 1918 influenza pandemic. Additionally, I synthesize major themes of the research that analyzes post-pandemic inequalities compared with pre- and pandemic period inequalities and place them within evolutionary anthropological theoretical frameworks that can help explain both biological and sociocultural determinants of long-term pandemic consequences.

1.1 | Why the 1918 Influenza Pandemic?

There are six primary reasons why the 1918 influenza pandemic is an ideal historical event for which to study this question. These reasons are summarized in Table 1. First, it is inherently valuable to closely study historical events across geographic locations and among various sociocultural, international, and transnational spaces. The knowledge creation surrounding the 1918 influenza pandemic, its determinants, and its related and deeply intertwined historical events (i.e., World War I) has accelerated in recent years, and this has led to more clarity about how, by nature, pandemics are anthropogenic events that can be studied through a variety of life science, social science, and humanistic lenses. Second, there are ample early 20th century primary data sources available to study the 1918 influenza pandemic from many different perspectives. This includes not only vital death (and occasionally morbidity) records, but biological anthropologists are now engaging with skeletal samples to understand underlying frailty during and after the 1918 influenza pandemic [6, 17]. There are many new research endeavors that have dipped into these rich data sources already. Unfortunately, data availability is not ubiquitous for populations affected by the 1918 influenza pandemic, so careful research and reflection on biases are necessary.

TABLE 1 | Summary of reasons discussed in-text why the 1918 influenza pandemic is an ideal pandemic to investigate long-term evolutionary consequences.

Why study the 1918 influenza pandemic and its post-pandemic consequences?
1. It is inherently valuable to improve our knowledge of historical events.
2. Data availability is often good, though not uniformly through space and time.
3. Acute, massive mortality events can lead to detectable, long-lasting consequences in surviving populations.
4. The population that survives is larger than the population that does not[a]; what are their experiences?
5. We can compare 1918 influenza pandemic outcomes to those of other typical, non-pandemic influenza outbreaks.
6. A substantial time has passed to where we may retrospectively study long-term consequences of a century-old pandemic.

[a]This is the case almost everywhere, except when analyses are broken down into lower population levels. For example, there are villages in Alaska that were abandoned after the influenza pandemic tore through, claiming every life aside from those of some young children ([15]; see Phillips-Chan for a review of daily newspaper data [16]).

Third, the 1918 influenza pandemic was a massive mortality event, estimated to have resulted in the death of 50–100 million people on the high end [18] and around 20 million people on the low end [19]. This means for some estimates of mortality, this pandemic resulted in nearly five times as many deaths directly attributed to World War I in only 2–3 years [20]. As such, an acute mass mortality event could lead to detectable changes in demographic structure, sociocultural norms, and epidemiology in the surviving population. Pandemic research needs to closely investigate whether these shifts happened, and further, what their consequences were. Fourth, and similarly, the number of people who survived the 1918 influenza pandemic was far greater than the number who did not; there could be long-lasting detectable changes in health and epidemiology in the surviving population that could be directly or indirectly attributable to the pandemic itself, and those health impacts in the surviving populations are currently poorly understood. Without further research into questions related to long-term impacts, we are missing a significant domain of knowledge about the breadth of the pandemic's impact.

Fifth, the 1918 influenza pandemic was caused by an H1N1 influenza A virus, which is now prolific but at the time was novel [21, 22]. There are many strains of influenza circulating at any given time, and influenza viruses were common before the 1918 pandemic and are common in the present day. As a regularly circulating virus that produces a familiar disease, researchers can compare 1918 influenza pandemic outcomes to influenza outcomes from non-pandemic years. This is not a straightforward thing to do with other pandemic diseases like the recently novel SARS-CoV-2 virus that causes COVID-19 disease, AIDS, or antibiotic-resistant tuberculosis. Finally, enough time has now passed to retrospectively study the consequences of the 1918 influenza pandemic for multiple decades afterwards. This may not only lead to insights about how long the consequences of the pandemic are detectable (if they indeed are), but can provide crucial insights into how inequalities in health were impacted by this acute event. Now, 5 years into the COVID-19 pandemic, is the time to pursue more insights into how pandemics end. Evolutionary anthropological theories can provide strong insights into the nature of post-pandemic periods.

2 | Evolutionary Perspectives of Pandemic Consequences

Anthropological perspectives of population health can successfully integrate information about biology and culture over long periods of time, often by capturing evolutionary effects on demographic composition and intergenerational health transmission. Studying population health from an evolutionary perspective is an excellent way to conceptualize ultimate causes of unequal impacts. Here, I briefly outline three fundamentally evolutionary frameworks used in anthropology to describe and give meaning to observed population health inequalities: the epidemiological transitions, biocultural anthropology, and evolutionary medicine. These theoretical frameworks are not mutually exclusive, and a holistic understanding of pandemic and post-pandemic inequalities may be best achieved by drawing upon concepts from a combination of principles from each framework.

2.1 | The Epidemiological Transitions and Biocultural Anthropology

What is today referred to as the *second epidemiological transition* was first conceptualized by epidemiologist Abdel Omran in his seminal 1971 paper "The epidemiological transition: A theory of epidemiology of population change [23]." However, the concept of multiple transitions throughout recent human history (at least the last ~10,000 years) is the product of anthropological thought [24–26]. The epidemiological transitions refer to points in human history during which there were fundamental, long-term, and irreversible changes in human behavior and culture that led to changes in human health, especially cause-specific mortality [26]. As such, there are currently four distinct periods and three described transitions. The first transition aligns with the Neolithic agricultural revolution (~12,000–10,000 years ago) in about a dozen locations around the world relatively simultaneously, moving humans from the Paleolithic baseline to "The Age of Pestilence and Famine" [23]. Consequences included the widespread adoption of the agricultural mode of subsistence, the decline of a diversified diet, increased sedentism, increased population size and density, and exposure to zoonotic diseases that evolved into modern crowd diseases (e.g., influenza, smallpox, measles) [24, 27]. For groups that adopted agriculture, most of recent human history was spent in this state.

The second epidemiological transition did not begin until the mid-19th century in Western Europe and parts of North America and occurred alongside the industrial revolution [23, 28]. This period describes the shift from "The Age of Pestilence and Famine" to "The Age of Receding Pandemics," where the mortality rates from infectious diseases declined and the mortality from chronic diseases, brought on primarily by aging and organismal degeneration, increased proportionately. The concept of the second epidemiological transition was primarily formulated as an explanation for the overall mortality decline observed during the *demographic transition*: a period in which a decline in all-cause mortality was followed by a decline in fertility, leading to increases in population size and life expectancy [29–31]. The *third epidemiological transition* describes the shift into "The Age of Degenerative and Man-Made Diseases," and is characterized by new and re-emerging infectious diseases with a strong emphasis on newly recognized and truly novel pathogens in the contemporary disease-scape [25, 26].

Epidemiological transition theory is a fundamentally evolutionary theory because it involves the co-evolution of human culture, demography, and biology, considering the integral role of pathogens in that evolution [32]. On a single population level, it provides tremendous insight into population history and the dynamics of pathogens and culture over a long period of time. On a broader cross-population level, it provides opportunities to compare the trajectories of health for populations from regions that underwent the transitions and those that did not. The forces on demographic structure and dynamics are fundamentally evolutionary in nature and have inevitable consequences on human biology and health [33, 34]. For example, the second epidemiological transition in Western nations is characterized, in part, by advancements in public health practice, nutrition, infrastructure, and much later, safe and effective biomedical advances such as vaccines and antibiotics. However, these benefits are also the touchpoints of tremendous health inequities, because there is no single way to model health transitions, and the strict model of the classic transition described by Omran [23] is irrelevant for many regions outside of Western Europe and parts of North America (e.g., [35–37]). Thus, origins of baseline health inequalities are partially, though not solely, due to populations' adaptations to their specific ecologies, a process that gives rise to cultural adaptation in different ways and at different times. Epidemiological transition theory plays an important role in reminding population health researchers that nothing is static, and these changes constantly occur in the background of other emerging health issues, like novel pandemic infectious diseases. Thus, population health as described by the epidemiological transitions—at any point in time in recent human history—may provide critical contextual insights into how and why people suffer such variable consequences during pandemics.

Discussions of the epidemiological transitions can be meaningfully integrated with *biocultural anthropological theory*, as the integration and co-evolution of human biology and culture were essential to how human demography, social organization, and health shifted over time. Biocultural theory in anthropology is also a fundamentally evolutionary framework that highlights the physical embodiment of humans' macrosocial socioeconomic environments, coined "local biologies" by Margaret Lock [38, 39]. This is a popular anthropological theory that highlights the integrated and relatively plastic nature of human biology and culture [40–42]. The human "environment" includes anything from physical living spaces to plants, lands, waters, animals, and insects [43], as well as the political economic and political ecological contexts [40, 44]. Biocultural variation in humans cross-culturally can be products of the embodiment of food and water insecurity [45], stress as a product of socioeconomic stratification and structural violence [46–48], the embodiment of stress as evidenced through skeletal lesions studied by paleoepidemiologists and paleopathologists [49], and the physical markers of sex- and gender-based stressors [50].

Anthropologists who study infectious diseases and humans through an evolutionary perspective would argue that pathogens should be part of this story, as well. It is possible that infectious pathogens have been one of the strongest forces of human genome evolution [51], but humans and pathogens are evolutionary partners: we change each other. Indeed, early in the COVID-19 pandemic, Friedler [52] argued that a biocultural approach to infectious respiratory diseases was crucial to advance our understanding of how human behavior at every level—from the community to the global—impacts infectious disease dynamics. Other anthropologists have drawn upon biocultural theory to identify how and why baseline nutritional status in rural communities may have influenced tuberculosis prevalence and mortality [53], especially when tuberculosis has been identified as one of the primary pathogenic risk factors for 1918 influenza pandemic mortality [54, 55]. Zuckerman et al. [56] argue these evolutionary approaches (epidemiological transition and biocultural theory) are essential to one another. Further, van Doren [32] argues that biocultural anthropologists should more purposefully engage with the foundational concepts of epidemiological transition theory, especially the deep temporal scope and attention to population histories, especially in the context of infectious disease dynamics across hundreds—if not thousands—of years.

2.2 | Evolutionary Medicine and the Fetal Origins of Health and Disease

In reference to centuries and millennia of human-pathogen co-evolutionary history, *evolutionary medicine* within anthropology can provide keen insights into the specific tensions between humans and the invisible world of pathogens over broad temporal depth, as well. As a field, the scope of evolutionary medicine is broad, but the intention is straightforward: to apply evolutionary thinking to the broad spectrum of human health and disease [57–60]. Some of these applications include, but are far from limited to: the effects of nutritional stress on long-term population health [61]; the biological constraints of human childbirth [62]; how, despite the nonexistence of genetic markers of race, socially constructed and reinforced racialized categories have produced embodied social inequalities in health [63, 64]; allergies [65]; and chronic pain [66].

Anthropologists, especially but not limited to those who specialize in biological anthropology, are well-trained in evolutionary theory, and with their holistic approach to understanding the human condition, they are often most interested in addressing ultimate, rather than proximate, determinants of human health and disease. Evolutionary biologists have distinguished between proximate questions (those that answer "How?") and ultimate questions (those that answer "Why?"), which is a categorization that emerged from Tinbergen's "four questions": (1) What is it for? (2) How does it work? (3) Why did it develop? (4) What is its evolutionary history? [67, 68]. Biological anthropologists using evolutionary concepts to study infectious diseases in humans may spend considerable time studying the evolutionary origins of humans' relationships with viruses, bacteria, parasites, and the ecological contexts that can lead to dis-ease in humans. Though proximate explanations for why humans get sick when infected with pathogens (i.e., a description of a disease process and associated immunological responses) are extremely important as well, they are not themselves evolutionary explanations. However, those processes may be better understood when informed by evolutionary thought. Answers to proximate questions may illuminate infectious processes, human immune responses, and epidemiology of disease; answers to ultimate questions can explain human-pathogen co-evolution, social inequalities, developmental and fetal origins of disease, and susceptibility to infections based on underlying health status, among others [69]. It is not difficult to see how population health and demography determined by human behavioral changes over time (epidemiological transitions) and local biologies (described by biocultural theory) are integral pieces of this human-pathogen co-evolutionary story.

As an example, the co-evolutionary relationship between humans and diseases like tuberculosis have not only been well-established, but modern biomedical advancements have allowed scientists to observe pathogenic evolution in real time. The most common respiratory form of tuberculosis is caused by the bacterium *Mycobacterium tuberculosis*, which infects the lungs and leads to a complex immune response. In fact, this immune response is very likely an evolutionary response to the increased virulence of the pathogen and the resulting severity of tuberculosis disease [70], which when progressed can cause rapid weight loss and wasting, fatigue, severe coughing with bloody sputum, and death. Tuberculosis has been known in humans for thousands of years and has been strongly influenced by subsistence and settlement patterns, public health and sanitation, and other critical co-circulating pathogens through time [32]. Today, only 10% of people infected with *M. tuberculosis* will manifest symptoms, and only 10% of people who show symptoms will progress to death [71, 72]. However, this immunological protection against the intracellular infection that leads to tuberculosis disease has been compromised by a cultural advancement that has led to evolutionary responses in the causal pathogen: the prolific use and mis-use of antibiotics have led to the evolution of antibiotic resistance in some strains of *M. tuberculosis*, resulting in multidrug resistant and extensively drug-resistant infections in recent decades [73–75]. This adaptation to humans' biomedical cultural advancements in antibiotic use has tipped the balance back toward *M. tuberculosis*. This example, and others like it, can be cleverly illustrated by the metaphor of the Red Queen, which borrows imagery from *Through the Looking Glass* by Lewis Carroll: two opposed actors—Alice and the Red Queen—pushing and pulling on one another to gain an advantage over the other [76]. This "Red Queen Hypothesis" is particularly relevant to studies of novel pandemic diseases, as well. For example, some research on the novel H1N1 influenza A virus from 1918 suggests that, in terms of viral descendants and the makeup of typical, regularly circulating influenza viruses in the present day [21], H1N1 influenza A has been a very consequential modern virus.

The *fetal origins hypothesis of adult disease* is also an application of evolutionary medicine that is particularly relevant for studying long-term health consequences of the 1918 influenza pandemic. Though the insight on the effects of long-term health shocks *in utero* were first made by Kermack et al. in [77],

Barker's [78] comments on the persistently observed relationship between low birthweight and later health outcomes, such as cardiovascular morbidity and mortality, diabetes, and other metabolic disorders, are likely the most cited. The relationship between birthweight and later-in-life outcomes was so much stronger than the outcomes related to stressful childhoods that Barker [78] was compelled to suggest research should be directed how a stressful intrauterine environment impacts adult health. Barker [79] later published empirical results showing that infants with low birthweight for their gestational ages have significantly higher adult blood pressure, impaired glucose tolerance, and higher mortality from cardiovascular events and obstructive lung diseases. Developmental health theories are based on the assumption that there are two primary pathways of health investment throughout the life of a person: (1) the initial health endowment determined by the fetal environment, and (2) post-birth developmental investments [80]. Both levels of investment are appropriately captured by the broader theoretical framework of *developmental origins of health and disease (DOHaD)* (e.g., [81–83]), but this paper will focus specifically on how the 1918 influenza pandemic may have compromised the initial health endowment determined by the fetal environment.

In the context of the 1918 influenza pandemic, *in utero* exposure does not explicitly constitute direct fetal exposure to the pandemic virus because H1N1 influenza A does not cross the placenta ([84, 85] present two case studies during the 2009 H1N1 influenza A pandemic). Instead, *in utero* exposure most accurately refers to exposure to a stressful intrauterine environment when the mother was infected with the novel pandemic influenza virus. In this case, there are multiple pathways in which a "stressful intrauterine environment" can cause adverse health impacts on a developing fetus. Myrskylä et al. [86] provide a broad overview of the types of stress a fetus of an infected mother may have faced. First, as a basic principle of evolution and adaptation, developing fetuses recognize environmental signals and must adapt to those cues; problems arise when the stressful intrauterine environment no longer aligns with the environment in which the individual grows into an adult, thus producing a mismatch and potentially maladaptive consequences [87]. A nutritionally deprived environment *in utero*, potentially from maternal malnutrition or severe nutritional decline from either loss of appetite or vomiting and diarrhea, may prime individuals for a similar nutritionally stressed environment outside the womb. When this does not occur, overnutrition as a child or adult can increase the risk of obesity and make them more at risk of cardiovascular disease and diabetes [88–90].

Maternal infection with the pandemic influenza virus has also been a focus of fetal origins research as a potential determinant of later-in-life health. The increased risk of severe disease and symptoms in pregnant individuals has been known for some time and can partially be explained by the simultaneous demands of the immune response to the pregnancy *and* to the influenza infection [91, 92]. Elevated immune activity during novel influenza virus infection may prime the developing fetus for baseline chronic inflammation in adulthood, which is strongly linked to the development of cardiovascular disease [93, 94]. Additionally, the trimester during which the individual was exposed to 1918 influenza pandemic-related maternal stress seems to matter tremendously: as the third trimester is the primary period in which the lungs reach full development, intrauterine stress that compromises this development can lead to the development of asthma, chronic obstructive pulmonary disease, bronchopulmonary dysplasia, and congenital diaphragmatic hernia [95, 96]. Alternatively, severe intrauterine stress in the first trimester significantly increases the risk of preterm birth, which elevates the risk of adult health complications [97, 98], including shortened telomere length, which is related to the development of aging-related diseases earlier in life than expected [99]. As I will explore in the review and discussion of the fetal origins literature related to intrauterine stress during the 1918 influenza pandemic, the unequal epidemiology of the pandemic may have had substantial long-lasting unequal impacts on the individuals who survived it—not just those most at risk as younger adults, socially marginalized racial groups, age-/sex-/gender-based inequalities, or disabled individuals—but on those who lived to face consequences of the pandemic without ever being infected with the virus.

In the remaining sections of this paper, I discuss how the bibliography for the conceptual review was built and the criteria with which the primary papers of interest were selected for deeper discussion. The papers' primary results are then highlighted with special attention to the comparisons between inequalities observed in a post-pandemic period to either a pre-pandemic or pandemic period (oftentimes, both). Finally, I synthesize and discuss the results within the context of the evolutionary anthropological theories outlined here and identify key points at which further inquiry can be pursued in the future of biological anthropological pandemic studies.

3 | Methods for Identifying Literature

This paper is not a systematic review, scoping review, or meta-analysis. However, a careful and iterative literature search was performed to identify appropriate pandemic experiences as a lens through which to discuss the posed question in a conceptual review. As mentioned previously, there have been several thorough literature reviews in recent years that discuss inequalities during pandemics. These reviews were used as core papers to initiate the literature search: D'Adamo et al. [10], a scoping review identifying health and inequalities across the last four major influenza pandemics (1918, 1957, 1968, and 2009); Mamelund and Dimka [13], a narrative review of socioeconomic and racial inequalities during the COVID-19 and 1918 influenza pandemics; Mamelund et al. [100], a systematic review and meta-analysis of research on socioeconomic inequalities and pandemic influenza; Dimka et al. [11], a review and synthesis of inequalities related to sex/gender, racialized groups, disability, and pre-existing conditions during the 1918 influenza, the 2009 H1N1 influenza A, and COVID-19 pandemics; and DeWitte and Wissler [9], a review of demographic and evolutionary changes after the Black Death and 1918 influenza pandemic. The seeds for contemporary research on long-term health impacts due to fetal exposure during the 1918 influenza pandemic seem to be Almond and Mazumder [80] and Almond [101], from which many demographers and economists have drawn inspiration for applications of the fetal origins hypothesis and the 1918 influenza pandemic. During the

initial search, I used the bibliographies of the above articles to identify research related to inequalities about only the 1918 influenza pandemic; I do not consider any other pandemic in this paper.

I employed both backwards and forwards searches using these core papers for new articles that analyze inequalities in any form, but especially those related to age, sex, gender, socioeconomic status, race category, disability, and underlying conditions. Throughout this paper, I conceptualize "inequality" as heterogeneous outcomes for different subsets of a population on the basis of the above categories. This is an important expansion of the scope of inequalities investigated in some of the core literature, as some are limited only to discussing articles that formally quantify and analyze socioeconomic inequalities (e.g., [12]). This is not an inherent weakness of those approaches, but the intention in the current analysis is to identify inequalities that go beyond the scope of socioeconomic status. Characteristics such as age and sex come with important and changing social roles through the life course that cannot be extricated from explanations of differential age-based mortality or other epidemiological outcomes. These differences in social roles, how social roles shift as one ages, and the variable infectious disease exposure risks associated with those roles and behaviors, are central to an anthropological conception of pandemic experiences.

Additionally, sex is an important marker of inequality in historical studies. This is not solely due to the potential for immunological differences in males and females (i.e., females may exhibit a heightened immune response to influenza viruses but can also overreact to viral pathogens and are more prone to develop autoimmune disorders) [102], but also because researchers often infer gendered differences in behavior along the lines of recorded binary sex in vital records [103]. Although this is an imperfect interpretation, not least of all because sex and gender refer to different characteristics in humans [104], sex and gender are both complex and important characteristics that may drive differential experiences with infectious disease outbreaks via different pathways. Thus, pandemic outcomes on the basis of age, gender, and sex, along with ethnicity and socially constructed race categories, are also important markers of inequality that can exhibit dynamic patterns through time and may be affected specifically by pandemic experiences.

Since the purpose of this article is to shape the gap in the literature related to changes in inequalities after the 1918 influenza pandemic, then enough time had to pass after the end of the 1918 influenza pandemic for researchers to feasibly ask and answer this question. To my knowledge, there is no established precedent for what qualifies as "enough time" passing after the pandemic for this line of inquiry, particularly for studies of how severe mortality impacts demography and epidemiology long-term. I have considered, and ultimately chose, a cut-off of 10 years. There is geographic variation in when particular regions of the world first experienced the pandemic influenza virus. For example, Europe and parts of North America experienced the first wave of the pandemic in the spring of 1918 [1], while the pandemic did not reach Australia until 1919 [105]. Additionally, there is some evidence of other isolated areas not experiencing the pandemic influenza virus until 1920 or later (e.g., [106]). As such, although some of the early research provides important insights into inequalities during the pandemic in the form of surveys and canvases, papers, and reports that were published within 10 years of the end of the 1918 influenza pandemic (~1921) were excluded.[1] The question of what constitutes "long-term" after the 1918 influenza pandemic was considerably easier to answer when assessing research on the long-term impacts of fetal exposure during the pandemic. The fetal origins of *adult diseases* specifically stipulates that the impacts of stress in the fetal environment have consequences on *adult* health [78, 79, 120]. Thus, by nature, the research on health of adults exposed *in utero* during the main waves of the 1918 influenza pandemic is centered around observed health outcomes and socioeconomic status in 1960 and beyond. This is not an arbitrarily chosen date, but rather the earliest date at which the published research begins to assess adult outcomes.

After this literature search was completed and the time periods accounted for, there were a total of 44 articles that investigated unequal epidemiological experiences during the 1918 influenza pandemic on the basis of socioeconomic inequalities, ethnicity/racialized groups, sex/gender, and disability. Only six of these articles included analyses of a post-pandemic period and compared them to the population's respective pre-pandemic and/or pandemic periods [4, 5, 54, 121–123]. Figure 1 illustrates the time periods covered in each of these studies.

The selection of papers assessing the long-term health and socioeconomic inequalities due to fetal exposure during the 1918 influenza pandemic was slightly different. Due to the nature and specificity of the question posed ("How does exposure to a stressful fetal environment during the 1918 influenza pandemic impact adult health and livelihood?"), the gathering of articles based on a set of inclusion or exclusion criteria as outline above does not necessarily apply. Instead, I assessed articles for adherence to the question posed and the intentional framing of results within the fetal origins hypothesis. Once the gathered articles appeared to reach conceptual saturation and provided a reasonable representation of the fetal origins of adult health and disease after intrauterine stress related to the 1918 influenza pandemic, I concluded the search. In total, I will briefly discuss and synthesize 11 articles: Almond [101], Almond and Mazumder [80], Cohen et al. [159], Fletcher [160], González et al. [88], Helgertz and Bengtsson [161], Hong and Yun [162], Lin and Liu [163], Mazumder et al. [164], Myrskylä et al. [86], and Nelson [165]. Figure 2 illustrates the ranges of time these studies cover.

4 | Post-pandemic Inequalities

4.1 | Post-pandemic Inequalities Related to Pandemic Mortality, Morbidity, and Survivorship

The knowledge surrounding the demographic and epidemiological impacts of the 1918 influenza pandemic, especially its impacts *on* inequality, is very limited. In other words, for example: if sex-based inequalities were identified during the pandemic, do sex-based differences in health and social outcomes after the pandemic persist, magnify, or decline? The

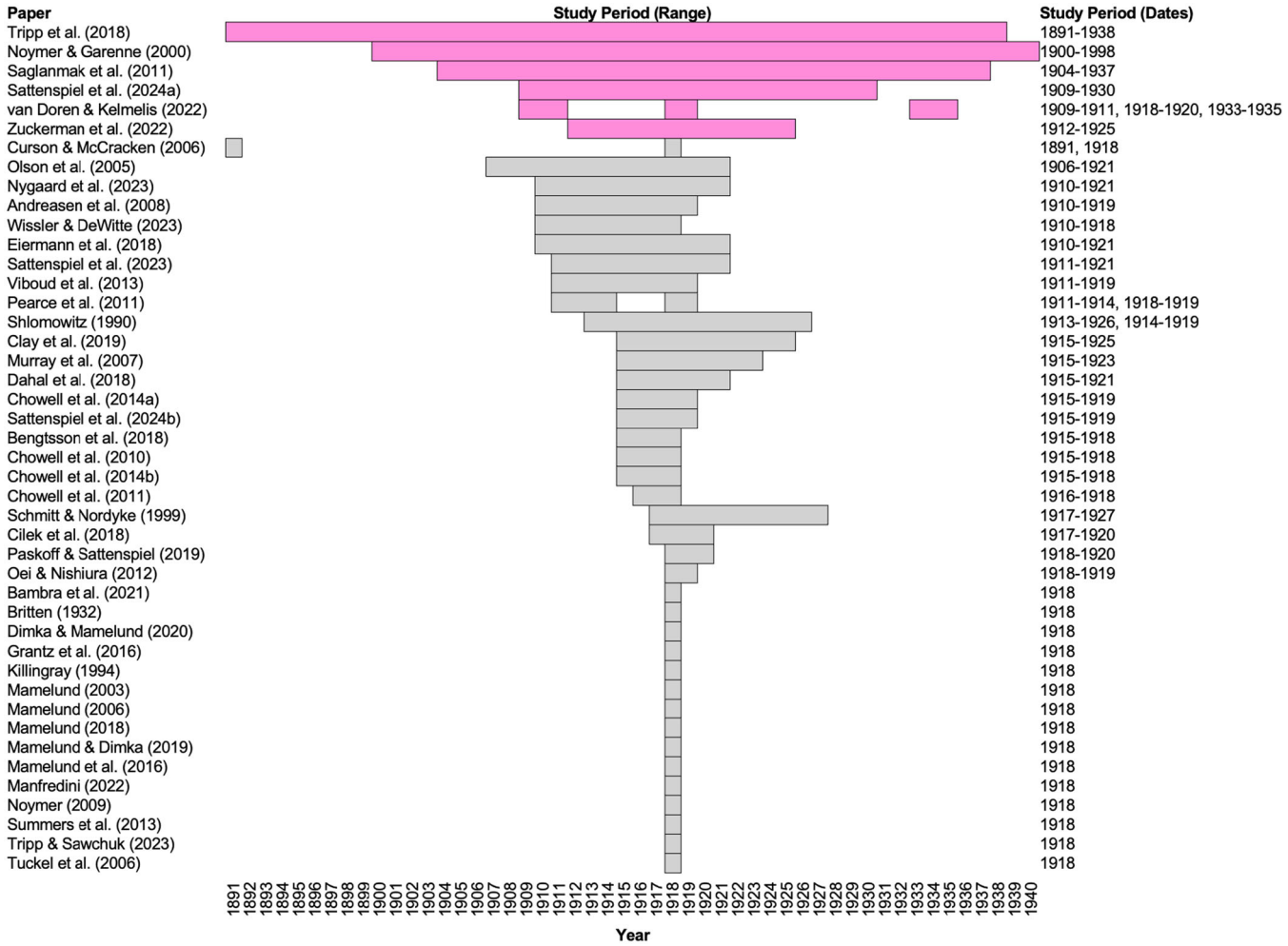

FIGURE 1 | Gantt chart of the 44 articles found during the literature search that engage with 1918 influenza pandemic inequalities on the basis of socioeconomic status, sex/gender, racialized/ethnicity groups, and disability. The six articles identified that specifically engage with post-pandemic impacts on unequal outcomes are highlighted in pink [4, 5, 54, 121–123]. The chart is grouped first by whether or not the article assesses post-pandemic to pre-pandemic and pandemic periods, then by the first year of the study period examined within the article. The exact study date ranges appear on the right side of the chart. Note that Noymer and Garenne [54] investigate changes in sex-based life expectancy through 1998, but I have truncated this timespan to 1940 for better visualization of the other papers' study periods. Articles referenced in the figure in grey correspond to references: [6, 8, 12, 55, 105, 124–158].

existing literature on post-1918 influenza pandemic inequalities is currently limited to only six papers: the impacts on life expectancy between males and females in the United States, 1900–1998 [54]; the gradual shift in age-based influenza-related mortality from people aged <65 to people aged 65+ until 1937 in Copenhagen, Denmark [121]; the identification of recrudescent influenza mortality in urban, suburban, and rural areas of the Maltese islands in 1921 and 1929 [122]; the analysis of post-pandemic demographic and epidemiological change in Newfoundland [4]; the investigation of survivorship and mortality for influenza, pneumonia, tuberculosis, and other acute respiratory diseases in 1933–1935 compared with a pre-pandemic and pandemic period in Newfoundland [5]; and the exploration of the shifts in all-respiratory, influenza, and pneumonia mortality in the MSA in 1920–1925 [123]. I address each paper in turn alphabetically by first author's last name, except for the two papers on Newfoundland [4, 5], which I discuss one after the other for thematic continuity. Table 2 is a summary table of the observations of the research detailed in the following subsections.

4.1.1 | Life Expectancy in the United States

Noymer and Garenne [54] described changes in sex-based life expectancy in the United States for nearly the entire 20th century with specific interest in if—and how—the 1918 influenza pandemic impacted those dynamics over time. Their results generally indicated that females had both higher life expectancy and lower all-cause mortality than males from 1900 to 1998, though after the 1918 influenza pandemic, females' life expectancy and mortality advantage declined. With specific attention to the role of the 1918 influenza pandemic on these measurements, Noymer and Garenne [54] found that life expectancy for both sexes fell almost 12 years in 1918, and that by 1919 life expectancy rebounded to pre-pandemic levels and continued to improve for the rest of the century. There were no lasting negative impacts on male or female life expectancy after the pandemic. Sex-based differences in life expectancy fell from 5.6 years to 1 year after 1918, and females did not regain their survivorship advantage. Similarly, in 1921, male all-cause age-standardized mortality was about 4.1 deaths per 10,000

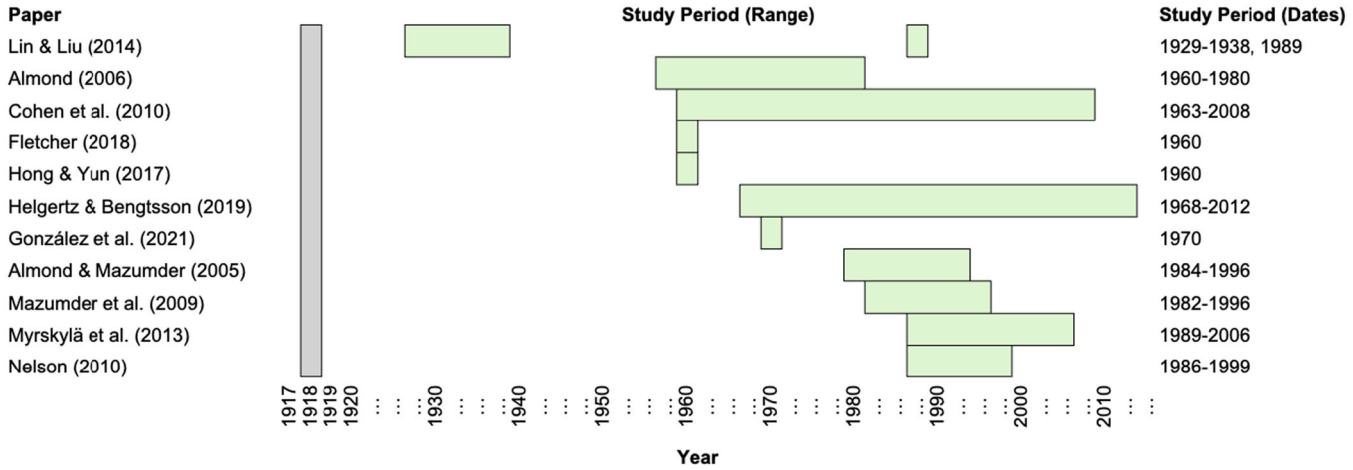

FIGURE 2 | Gantt chart of the 11 articles discussing the long-term effects of *in utero* exposure to the 1918 influenza pandemic within the framework of the fetal origins hypothesis. The 1918 influenza pandemic is highlighted as a vertical grey line to delineate the time point of exposure, and the green ranges illustrate the time periods of research on adult health and socioeconomic outcomes to fetal exposure. The exact study date ranges appear on the right side of the chart. Articles referenced in the figure correspond to references: [80, 86, 88, 101, 159–165].

individuals higher than females, and this difference fell to 0.86 deaths per 10,000 by the end of the century [54]. In this case, Noymer and Garenne [54] attributed these post-1918 life expectancy dynamics directly to the 1918 influenza pandemic through the selective mortality during the pandemic and those who were also afflicted with tuberculosis.

4.1.2 | Age-Based Mortality in Copenhagen, Denmark

Saglanmak et al. [121] investigated the changing rate ratios of excess influenza and pneumonia (P&I) mortality between individuals aged < 65 years and those aged ≥ 65 years ($RR_{<>65}$), as well as the rate ratio between seasonal post-pandemic excess mortality to the pre-pandemic baseline P&I mortality ($RR_{pre\text{-}post}$). This paper assessed these changes in excess mortality rates and compared them to the pre-pandemic and pandemic P&I mortality rates until 1937 in Copenhagen. Results indicated that 84% of deaths in 1918–19 pandemic years were in people aged 15–64, supported by 1918 and 1919 $RR_{<>65}$ of 6.5 and 2.5, respectively. $RR_{pre\text{-}post}$ was highest for individuals aged 5–14 and 15–64 in fall 1918 (124 and 67, respectively[2]) and remained high in 1919–20 (25 and 34, respectively). By 1921–22, $RR_{pre\text{-}post}$ decreased dramatically and remained between 0.1 and 1.0 until the end of the study period in 1937. The $RR_{pre\text{-}post}$ result for fall 1918 for these two age groups is substantial; for example, the $RR_{pre\text{-}post}$ for 5–14-year-olds in fall 1918 means that P&I mortality that season was 124x larger than for the baseline years. After 1920, these rate ratios returned to pre-pandemic levels, and post-pandemic P&I mortality rates were occasionally smaller than pre-pandemic rates. Unrelated to age-based excess mortality but still relevant to deeper investigations of post-pandemic patterns, Saglanmak et al. [121] additionally found that seasonal excess mortality in 1918–37 coincided with high excess influenza morbidity ($p < 0.001$), *except* for in 1923–24, when there was high excess mortality and *low* excess morbidity.

Results of the age-based analyses through the calculation of $RR_{<>65}$ help illustrate how long-term age-based inequalities observed during the pandemic did not immediately revert to pre-pandemic levels. For example, during the years of the pre-pandemic baseline, older adults (> 65 years) had a 10-fold higher risk of influenza-related death per capita than those < 65 years. After 1920, age-based excess mortality gradually shifted back toward older people: it took until 1925–26, 7 years after the emergence of the novel H1N1 influenza virus, for $RR_{<>65}$ to return to the prepandemic levels at 0.1 or below, where it remained until 1937 [121]. Some of these results may be influenced by the fact that, as people age, they move up age classes; by 1937, there were plenty of people in the ≥ 65 age category who were < 65 years during the pandemic. However, because the age classes that had the highest excess mortality during the pandemic were individuals aged 20–44 [166], the most at-risk age cohorts during the pandemic would not have aged out of the < 65 category by 1937. Thus, cohort effects observed may be slightly impacted, but these impacts are likely minimal.

4.1.3 | Demographic and Epidemiological Shifts in Newfoundland

Sattenspiel et al. [4] sought to characterize the 1918 influenza pandemic's impacts on the post-pandemic demography and epidemiology on the island of Newfoundland with existing statistical census and vital record data. Sattenspiel et al. [4] identified declines in fertility beginning in the 1930s and persistent high mortality, indicating an ambiguous position of the demographic transition on the island during this period of the 20th century. With this information, there is no clear link between the substantial mortality impact of the 1918 influenza pandemic and the demographic structures observed in the 1921 and 1935 statistical censuses, though it is clear there were some demographic shifts occurring by 1935. When this question was investigated further with mortality schedules from vital records, all-cause mortality and infectious disease mortality declined in the urbanizing regions of the island, while there were no changes in the more rural regions. Only the urbanizing region of the island exhibited consistently higher chronic disease mortality, likely due to the strong proportionate decrease in

TABLE 2 | Summary of the six core papers of the conceptual review that compared post-1918 influenza pandemic inequalities to those of the pre-pandemic or pandemic period in each respective location of study.

Reference	Study population	Location	Study period	Pandemic outcome assessed	Inequality investigated	Summary of pandemic outcomes compared with non-pandemic years
Noymer and Garenne [54]	United States	North America	1900–1998	life expectancy at birth, mortality	sex, age	Life expectancy decreased for both sexes by 11.8 years from 1917–1918; there was no lasting effect on $e_{0,}$ and survivorship rebounded quickly; after 1918, the female mortality advantage fell from 5.6 years to 1 year and did not regain pre-pandemic mortality advantage until the 1930s
Saglanmak et al. [121]	Copenhagen, Denmark	Europe	1904–1937	excess mortality, morbidity	age	Seasons associated with high excess respiratory mortality in 1918–1937 coincided with high excess influenza morbidity; in pre-pandemic years, older adults had a 10-fold higher risk of influenza-related death/capita; after the pandemic, excess mortality gradually shifted toward older people; by 1925–1926, $RR_{<>65}$ was back to pre-pandemic levels
Sattenspiel et al. [4]	Newfoundland	North America	1909–1930	mortality, birth rates, population structure	sex, age, region	No clear link between substantial mortality impact and demographic structures observed in 1921 and 1935 statistical censuses; urbanizing region exhibited consistently higher chronic disease mortality over time; fertility shifts observed due to significant decline in infectious disease mortality
Tripp et al. [122]	Maltese Islands (Malta, Gozo)	Europe	1891–1938	morbidity	sex, age, location	Pre-1918, morbidity showed relatively large peaks in 1892, 1894, and 1900 for both islands, followed by recrudescent waves from 1918 in 1921 and 1929
van Doren and Kelmelis [5]	Newfoundland	North America	1909–11, 1918–19, 1933–35	mortality, survivorship	age, region	Respiratory mortality from pre- to post-pandemic fell significantly in all but one rural region; survivorship during the pandemic was significantly lower than in pre- and post-pandemic periods; survivorship for influenza and pneumonia was significantly different among regions, and these differences became larger post-pandemic; mean and median age increased from a prepandemic range of 23.0–26.4 years to 23.6–35.7 years.
Zuckerman et al. [123]	Mississippi State Asylum (MSA)	North America	1912–1925	mortality	race, sex, age	No significant changes in post-pandemic all-respiratory or influenza and pneumonia outcomes compared with the pre-pandemic period

Note: The authors, study population, location, study period, pandemic outcome assessed, inequality investigated, and the summary of results are provided.

infectious disease mortality there [167]. The fertility shifts observed were generally due to the significant decline of infectious disease mortality rather than any observed changes in chronic disease mortality. Sattenspiel et al. [4] point out that given the substantial mortality for individuals aged 15–45 in Newfoundland [124, 168], there should be a constriction in the age-sex histograms constructed from the 1921 statistical census. Even though this constriction does appear in those aged 30–34, it does not appear in ages 45–49 for males or females in the 1935 census; if there were any substantial effects of the 1918 influenza pandemic on this age group, they were likely short-lived and were not quite substantial enough to manifest in 1935.

4.1.4 | Respiratory Disease Survivorship in Newfoundland

van Doren and Kelmelis [5] analyzed survivorship for P&I, tuberculosis, and other common respiratory diseases that most typically affected young children (bronchitis, measles, and whooping cough) on the island of Newfoundland for three different time periods: a pre-pandemic period (1909–11), the 1918 influenza pandemic (1918–19), and a post-pandemic period (1933–35). Analyses were stratified by age and geographic region to illuminate how age-based differences in these common respiratory diseases changed over the three periods between urban and rural regions. Survivorship for all respiratory causes of death was significantly lower during the pandemic, where the pre-pandemic hazard was 0.756 ($p < 0.001$) and the post-pandemic hazard was 0.945 ($p < 0.04$) compared with the pandemic baseline. This indicates that in the post-pandemic period, individuals were significantly less likely to die from a respiratory cause of death than they were in both the pre-pandemic and pandemic periods. Further, before the pandemic, survivorship for P&I was significantly different among regions, but these differences became significantly larger post-pandemic; results indicate that mean age at death from P&I rose from 23.0 years in 1909–11 to 26.4 years in 1933–35 [5].

One of the most significant results presented by van Doren and Kelmelis [5] is that survivorship from P&I *during* the 1918 influenza pandemic was essentially equal among all regions of Newfoundland, independent of their differences in population size, density, demographic structure, rurality, public health programs, access to medical care, or nutrition (although this does not mean that *mortality rates* were all equal when controlling for demographic characteristics or geography, [e.g., [124, 168]]. Therefore, it is notable that survivorship inequalities exist *outside* of the 1918 influenza pandemic, and Cox proportional hazards models indicated that there were no inequalities in P&I survivorship *during* the pandemic. It is important to know these differences exist, even if they do not occur in a pandemic context, to help give meaning to observed epidemiological patterns of respiratory diseases both inside *and* outside of the 1918 influenza pandemic.

4.1.5 | Morbidity in Malta and Gozo

Tripp et al. [122] approached inequalities in pandemic and post-pandemic experiences in the Maltese islands through investigations of differences among urban, suburban, and rural settlement patterns. There is much to say about the underlying determinants of unequal patterns of health and epidemic dynamics among urban, suburban, and rural spaces, though an exhaustive discussion is outside the scope of this paper. However, settlement type has long been a consideration for changing human health and infectious dynamics [169, 170]. Tripp et al. [122] showed that, in terms of morbidity for all settlement types in the Maltese islands, Gozo had higher morbidity than Malta, and there were significant differences in morbidity rates in rural and urban Malta versus Gozo ($p < 0.05$). A more detailed analysis in Malta suggests that it is rather the *suburban* region that suffered the highest mortality during the 1918 influenza pandemic, resulting in important questions about levels of analysis and points about settlement type definitions [125]. Pre-1918, influenza morbidity showed relatively large peaks in 1892, 1894, and 1900 for both islands, followed by recrudescent morbidity waves in 1921 and 1929, further affirming observations of recrudescence identified by Saglanmak et al. [121]. However, there were no identifiable inequalities in morbidity between Malta and Gozo for either of these post-pandemic influenza waves.

4.1.6 | Respiratory and All-Cause Mortality in the Mississippi State Asylum (MSA)

Finally, Zuckerman et al. [123] compared post-pandemic respiratory mortality to pandemic respiratory mortality by race, sex, and age in the MSA using individual death records from 1912–25. In this context, when all respiratory causes of death were pooled, there were no differences on the basis of social race category, nor were there sex-based differences, but there were age-based differences: using logistic regression and ages 0–19 as the baseline age category, there was significantly lower 40–59-year-old mortality (odds ratio [OR] = 0.390) and significantly lower > 60-year-old mortality (OR = 0.180). For solely P&I outcomes during the pandemic, there were again no race-based or sex-based differences, but there was again significantly higher probability of > 60-year-old mortality (OR = 0.313). Further, when comparing the pandemic period to non-pandemic periods, there was significantly higher pandemic mortality for both the pooled respiratory causes of death and P&I alone (OR = 1.827 and 3.058, respectively). When assessing the shifts in risk of mortality from P&I death from pre-pandemic (1912–17) to post-pandemic (1920–25), there were no significant changes in risk, either in the positive or negative direction. Notably, this article also intentionally investigated the contributions of intersectional characteristics on risk of mortality, for example, interactions between race and sex, and race and age, but the logistic regression results did not indicate any significant relationships in these control variables on the risk of mortality.

4.2 | Post-pandemic Inequalities Related to Fetal Origins

The literature that addresses the long-term impacts of exposure *in utero* is considerably larger than that of demographic and

epidemiologic impacts discussed above. Here, I discuss 11 papers that investigate the relationship between how being *in utero* during the largest waves of the 1918 influenza pandemic in the fall/winter of 1918 may have unequally influenced adult health and socioeconomic outcomes. I organize these papers into six thematic groups: socioeconomic status and family formation in the US [101, 160]; health and disability in the United States ([80, 86, 164]); mortality and life expectancy in 24 developed nations [159]; education and employment in South America [88, 165]; socioeconomic and health consequences in Sweden [161]; and education and health in Asia [162, 163]. Table 3 summarizes the body of research discussed below.

4.2.1 | Socioeconomic Status and Family Formation in the United States

Almond [101] is often cited as the first paper to take up the question of how exposure to a stressful fetal environment during the 1918 influenza pandemic can lead to inequalities in health and socioeconomic status in later life. Drawing upon information from published US decennial censuses and the Integrated Public Use Microdata Series (IPUMS) for samples of the US populations in 1960, 1970, and 1980, Almond [101] assessed differences in educational attainment, physical disability, income, socioeconomic status, and social security payments for individuals who were *in utero* during the height of the 1918 influenza pandemic compared with their immediate neighboring cohorts. The results indicated that individuals who were born in 1919 had 5 fewer months of education if their mothers were infected with influenza while pregnant and were 4%–5% less likely to complete high school compared with their surrounding cohorts; the effect on educational attainment was twice as large for non-white people. Additionally, annual wages were $700–900 lower and $2500 lower (in 2005 dollars) for sons of infected mothers, and poverty for the 1919 birth cohort was 1.5% higher than their neighboring cohort. In terms of disability, males were 6% more likely to have a work-limiting disability and 8% more likely to have a disability that prevented them from working completely compared with females in their cohort.

Fletcher [160] expanded upon Almond's [101] original work by investigating family formation among individuals born in the 1919 cohort that was *in utero* during the 1918 influenza pandemic, making this the first and—so far—only paper to specifically research this relationship. Using the same US decennial data from 1960, Fletcher [160] was interested in identifying differences in likelihood of marriage, age at first marriage, and spousal education level. The results indicated that there was about a 0.1-year reduction in years of schooling, which was small but statistically significant. The likelihood of marriage for the exposed cohort was 0.2%–0.4% lower than the neighboring cohort, and age at first marriage of the exposed cohort was no more than 1 month younger. There were sex-based differences in the likelihood of having children and the number of children per marriage: exposed females were slightly less likely to be married and get married at younger ages, and males were likely to get married at slightly older ages. Exposed females were less likely to have children and have fewer children, and exposed females have less educated male spouses, whereas exposed males did not have female spouses with any higher or lower education than expected.

4.2.2 | Health and Disability in the United States

Almond and Mazumder [80] used Survey of Income and Program Participation (SIPP) data from 1984 to 1996 to investigate the burden of chronic diseases and general health in people born 1915–23 in the US. They found that individuals born in 1919 were nearly 4% more likely to be in "fair" or "poor" health than individuals in immediately neighboring birth cohorts. They were also more likely to have a suite of functional limitations, or disabilities, including difficulty hearing (17% increase), speaking (35% increase), lifting (13% increase), and walking (17% increase). All differences were statistically significant. The 1919 birth cohort had higher rates of diabetes, stroke, cancer, heart problems, kidney problems, hypertension, and stomach problems. Interestingly, people who were likely conceived in August of 1918 and therefore spent their first trimester *in utero* during the height of the pandemic in some regions had higher risk of diabetes than any other birth cohort. This result indicates some variable risk in health outcomes by trimester of exposure.

Mazumder et al. [164] expanded upon this study by further analyzing health data on heart disease and diabetes from the National Health Interview Surveys (NHIS) by the National Center of Health Statistics for 1982–1996. They again identified a spike in heart disease for the 1919 birth cohort above levels of adjacent cohorts, followed by a downward trend in heart disease in increasingly younger cohorts. The 1919 birth cohort also showed accelerated cardiovascular aging with a 5% excess cardiovascular disease rate at ages 63–77. Individuals born in the first quarter of 1919 had 10.9% higher excess heart disease (25.4% increase in ischemic heart disease, but no identifiable effects for rheumatic or hypertensive heart disease). Further, the authors identified excess diabetes in individuals born in 1915 up to the third quarter of 1918 (14.9% excess), and then a large spike in excess diabetes for those born in the second quarter of 1919 (36.7%). Based on quarter of birth (an indication of which trimester the individual was exposed *in utero*), those born in the second quarter of 1919 had elevated heart disease ($\beta = 9.03$, $p = 0.021$), but those born in the third quarter of 1919 had significantly *decreased* heart disease ($\beta = -8.89$, $p = 0.010$) relative to those born in the first quarter of the year. There was a significant difference in heart disease for males and females born in the first quarter of 1919: males had significant excess total, ischemic, and hypertensive heart disease (23.1%, 32.7%, and 21.6%, respectively). The authors did not identify a sex-based difference in diabetes.

Myrskylä et al. [86] also used the NHIS linked with the National Death Index through December 31, 2006, from the NHIS-Linked Mortality Files. Myrskylä et al. [86] are interested in analyzing inequalities in life expectancy and excess mortality in individuals exposed to the 1918 influenza pandemic *in utero*. They found that individuals exposed in their third trimester (the second quarter of 1918 and the first quarter of 1919) had

TABLE 3 | Summary of 11 core papers of the conceptual review that investigated long-term socioeconomic and health inequalities linked to exposure to a stressed intrauterine environment during the 1918 influenza pandemic.

References	Study population	Location	Study period	Long-term Outcome(s) Assessed	Summary of long-term outcomes of fetal exposure during 1918 influenza pandemic
Almond [101]	United States	North America	1960–1980	educational attainment, physical disability, income, socioeconomic status, social security payments	Exposed less likely to finish high school (effects twice as large for non-w19hite individuals); annual income lower for men; men more likely to have work-limiting disability; social security payments higher for men; lower likelihood of high school graduation; higher poverty
Almond and Mazumder [80]	United States	North America	1984–1996	various health issues, disabilities	Exposed more likely to be in "fair" or "poor" health compared with neighboring cohorts; more likely to have functional limitations such as trouble hearing, speaking, lifting, walking; significantly higher risk of diabetes, stroke, cancer, kidney problems, hypertension, and stomach problems
Cohen et al. [159]	Many countries (24)	North America and Europe	1911–1923 birth cohorts, through 2008	mortality, life expectancy	No significant correlation between cohort-specific mortality rates and severity of pandemic in each country for 1918 or 1919; no significant changes in life expectancy
Fletcher [160]	United States	North America	1960	family formation, age at first marriage, spouse's education	Exposed had fewer years of schooling; likelihood of marriage lowered; lower age at first marriage; small reductions in likelihood of having children and number of children; exposed females less likely to be married; males experience no impact on marriage; exposed females less likely to get married and have fewer children
González et al. [88]	Argentina	South America	1970	education level, unemployment status	Exposed had significantly lower educational attainment; no relationship to employment status
Helgertz and Bengtsson [161]	Sweden	Europe	1968–2012	income attainment, occupational status, hospitalization (morbidity), mortality	Exposed had higher morbidity in ages 54–87 (hospitalizations); males exposed during 2nd trimester had higher cancer and heart disease mortality; those exposed in 2nd trimester had ~3 months shorter life expectancy; no consistent evidence of long-term socioeconomic consequences
Hong and Yun [162]	Korea	Asia	1960	education level	Exposed individuals' educational attainment fell; gap in educational attainment between exposed

(Continues)

TABLE 3 | (Continued)

References	Study population	Location	Study period	Long-term Outcome(s) Assessed	Summary of long-term outcomes of fetal exposure during 1918 influenza pandemic
					cohort and neighboring cohorts widest in low socioeconomic provinces more severely affected by influenza pandemic
Lin and Liu [163]	Taiwan	Asia	1929–1938, 1989	height, educational attainment, health outcomes	Exposed males similar in height to other males of neighboring cohort until age 14–16, at which point they were 3.5 cm shorter on average; male height difference among cohorts shrank to 1 cm by age 17; increase in maternal mortality rate (MMR) decreased average height of males; males had fewer years of education; less likely to be employed; increase in MMR reduced educational attainment; increase in MMR increased probability of respiratory disease, diabetes, and kidney disease
Mazumder et al. [164]	United States	North America	1982–1996	morbidity of heart disease and diabetes	1919 birth cohort had accelerated cardiovascular aging and 5% excess cardiovascular disease in ages 63–77; Individuals born in quarter 1 of 1919 had 10.9% higher heart disease; Individuals born in quarter 2 of 1919 had 36.7% excess diabetes; Only men had significantly higher heart disease, and no sex-based differences in diabetes morbidity
Myrskylä et al. [86]	United States	North America	1989–2006	life expectancy, excess mortality	Exposed in 3rd trimester had significantly higher excess all-cause mortality; exposed in 2nd and 3rd waves had decreased noncancer mortality and increased cancer mortality; excess all-cause mortality corresponded to decrease in life expectancy
Nelson [165]	Brazil	South America	1986–1998	college education, years of schooling, literacy, unemployment, hourly wage	Exposed in 1st and 2nd quarters of 1919 were less likely to graduate from college, less likely to be employed, less likely to be literate, have fewer years of schooling, and have lower hourly wages; males born in 1st and 2nd quarters of 1919 showed same patterns as total cohort (no differences for females)

Note: The authors, study population, location, study period, long-term outcomes assessed, and summary of the results are provided.

significantly higher all-cause excess mortality in 1989–2006 (hazard ratios [HR] = 1.08 and 1.09, respectively, $p < 0.05$). Individuals born in the third quarter of 1919 (those exposed during the second and third waves of the pandemic) had decreased non-cancer mortality (HR = 0.87, $p < 0.05$) and increased cancer mortality (HR = 1.26, $p < 0.01$). The increased all-cause excess mortality for the first quarter 1919 birth cohort corresponded to a 0.6-year decrease in life expectancy at age 70. The authors concluded that the fetal exposure to the 1918 influenza pandemic increased later-life mortality in non-cancer causes specifically, particularly for causes related to respiratory and cardiovascular causes of death.

4.2.3 | Mortality and Life Expectancy in 24 Developed Nations

Cohen et al. [159] assessed mortality and life expectancy on a much broader scope using the World Mortality Database for 24 developed nations primarily in North America and Europe from 1963–2008. They found little evidence for differences in mortality among cohorts born from 1911–1923, even when controlling for age. Specifically, there were no cohort-specific mortality rate differences for cohorts born in either 1918 ($\beta = -0.29$, $p = 0.23$) or 1919 ($\beta = 0.27$, $p = 0.26$), and analyses comparing life expectancy among cohorts yielded similarly insignificant results. To put this into context, the authors suggested that the quantity of life lost for those exposed *in utero* may have led to no more than 19 days of reduced life expectancy for females, and no change for males.

4.2.4 | Education and Employment in South America

There are two papers that investigate education and employment in South America. The first by González et al. [88] uses the 1970 National Census of Population, Households, and Housing from the Integrated Public Use Microdata Series (IPUMS) for Argentina and data from the National Hygiene Department of Argentina. They found that individuals *in utero* during the 1918 influenza pandemic had significantly lower educational attainment than their neighboring cohorts, and the most educational reduction was in the regions of Argentina with the lowest socioeconomic status (e.g., up to 0.5 fewer years of education than surrounding cohorts). They found no relationship between fetal exposure during the pandemic and employment status for either males or females. The authors noted that it was not possible to assess other employment-related outcomes (such as income) with the available data.

Nelson [165] used the Pesquisa Mensal de Emprego, conducted by the Instituto Brasileiro de Geografia e Estatistica, for the years 1986–1998 to investigate effects on college education, years of schooling, literacy, unemployment, and hourly wages in Brazil. Similarly to results found in Argentina by González et al. [88], Nelson [165] found that individuals born in the first quarter of 1919 were less likely to graduate from college (25.1%) and less likely to be employed (20.7%) later in life. Similarly, individuals born in the second quarter of 1919 were less likely to graduate from college (25.9%), less likely to be literate (12.4%), less likely to be employed (12.8%), have fewer years of schooling (0.191 years), and have lower hourly wages (24.6%) than their neighboring cohorts. Nelson [165] also identified sex-based differences in these education and socioeconomic outcomes: males born in the first quarter of 1919 were less likely to graduate from college (22.9%), less likely to be literate (7.2%), and less likely to be employed (27.1%) than their surrounding male cohort neighbors. Males born in the second quarter of 1919 were less likely to graduate college (29.3%), less likely to be literate (19.9%), and less likely to be employed (17.2%).

4.2.5 | Socioeconomic and Health Consequences in Sweden

Helgertz and Bengtsson [161] covered nearly half a century (1968–2012) in Sweden, where they used Swedish Interdisciplinary Panel (SIP) data administered at the Centre for Economic Demography at Lund University to investigate income attainment, occupational status, hospitalizations, and mortality for exposed cohorts. They found that for both males and females, fetal exposure is linked to overall higher morbidity (more hospitalizations) in people aged 54–87. Specifically, individuals exposed to a stressful environment during the second trimester were linked to increased risk of death from cancer and heart disease. Overall, effects on all-cause mortality were modest, and results indicated that there was a reduction in life expectancy for the 1919 birth cohort of about 3 months. However, the authors suggest that, with these data in Sweden, there is no consistent evidence that support any long-term consequences on socioeconomic status for individuals who were *in utero* during the height of the 1918 influenza pandemic.

4.2.6 | Education and Health in Asia

Hong and Yun [162] made use of data collected by Statistics Korea in the Microdata Integrated Services (MDIS), and analyzed a 2% microsample of the 1960 census, resulting in a sample of just over 100,000 individuals born between 1910–1929 who probably completed their education by 1960. In Korea, Hong and Yun [162] showed that the largest gaps in educational attainment for the 1919 birth cohort were in provinces most severely affected by influenza (i.e., South Chungcheong, North Gyeongsang, and South Gyeongsang). Overall, the authors estimated that the gap in years of schooling caused by the 1918 influenza pandemic in Korea for those born in 1919 was about 0.3 years, or nearly 4 months.

For available historical data in Taiwan, Lin and Liu [163] explored potential impacts on health for those who were *in utero* during the 1918 influenza pandemic. Interestingly, data available in the Taipei County's Statistical Books from 1929-1938 provided growth data through adolescence and puberty, which supplemented analyses performed using data from the 1980 Census of Taiwan and the 1989 Survey of Health and Living Status of the Elderly in Taiwan. Lin and Liu [163] found that until about age 13, the average height of males born in the 1919 cohort were statistically the same as their immediate

neighboring cohorts, but a significant gap appeared in ages 14–16 among cohorts; by age 16, individuals born in 1919 were 3.5 cm shorter. However, by age 17, the height difference between the 1919 and 1920 birth cohorts was an insignificant 1 cm. This result indicated that individuals *in utero* during the height of the 1919 influenza pandemic had a later growth spurt, with no lasting impacts on adult height. Later in life, however, analyses that relate maternal mortality rate and probability of having respiratory diseases (e.g., asthma, bronchitis, and other breathing-related diseases), diabetes, and kidney diseases showed a significant positive correlation.

Lin and Liu [163] also described some educational attainment relationships that could potentially be linked to *in utero* exposure. For example, males in the 1919 birth cohort had a 1.8% drop in years of education, which is slightly higher than the 1.4% drop observed by Almond [101]. They also observed that every 1% increase in maternal mortality rate was related to a reduction in education by 0.87 years. Finally, the authors observed that both the 1919 and 1920 birth cohorts were less likely to be employed by the 1980 census, although they acknowledge that this relationship is difficult to interpret since the age of retirement was relatively young in Taiwan (55 years).

5 | Discussion and Synthesis

The two sets of articles described in this paper cover two important domains of long-term post-pandemic impacts: those related to demographic and epidemiological consequences as a product of morbidity and mortality, and those related to adult health impacts of a stressful intrauterine environment during the pandemic. Upon description of the former set of papers, a clear difficulty emerges when attempting to develop a discussion that synthesizes all results: there are many ways to measure pandemic experiences, and each of these papers investigates a different experience. As such, with the already limited knowledge produced on post-pandemic experiences, there is little opportunity for cross-cultural/cross-population comparisons of similar quantifications of pandemic outcomes. This is one realm in which there is ample opportunity for future research, and an area to which biological anthropologists specifically can contribute knowledge due to their intellectual strengths with cross-cultural comparisons [11]. It is still valuable, however, to engage with the knowledge produced about long-term post-pandemic outcomes within the limited scope in which it currently exists.

Based on the results reported for each of the first six studies, there is no way to generalize about how the 1918 influenza pandemic affected populations demographically or epidemiologically in the long term. Noymer and Garenne's [54] results are perhaps the clearest: the 1918 influenza pandemic resulted in a very brief and dramatic decline in life expectancy at birth (nearly 12 years), followed by a similarly dramatic increase in life expectancy at birth beginning in 1919, which was *higher* than life expectancy in 1917. Life expectancy at birth then proceeded to increase for the remainder of the 20th century. Additionally, sex-based differences in life expectancy and mortality virtually disappeared after the 1918 influenza pandemic. The authors attribute this partially to selection against those who also had tuberculosis during this time period and suggest that this strong selective effect resulted in a relatively healthier population after the massive mortality wave in the fall of 1918. This type of selective mortality—that is, selection during a period of acute stress on population health, like a novel infectious disease pandemic—does have the potential to transform population health, as Noymer and Garenne [54] illustrate. These patterns are more difficult to identify for smaller populations. However, further investigation into the heterogeneity of the 1918 influenza pandemic's impacts on life expectancy on a smaller population or geographic unit than the entire United States is a worthwhile future opportunity, especially since the results may provide important insights into early 20th century demographic and health transitions. Given the results of sex-based life expectancy identified by Noymer and Garenne [54], as well as the fact that the 1918 influenza pandemic resulted in the highest excess mortality in people in their primary reproductive years (ages ~20–44) [166], we must acknowledge that, in severely impacted subpopulations, there could have been substantial impacts on fertility, social organization, migration, labor norms, and dependent care. This is evolutionarily relevant: in a thorough review of demographic biological anthropology work, DeWitte [33] describes how natural selection is heavily influenced by variations in fertility and mortality, especially during periods of rapid change [34] and the impacts of migration and gene flow [171].

Saglanmak et al.'s [121] results on the relatively slow return of total and age-based mortality back to pre-pandemic levels after the 1918 influenza pandemic provide insight into a different point of interest in long-term population health impacts after major mortality events. Saglanmak et al. [121] frame their results within the context of antigenic drift and suggest that recrudescent influenza waves of the novel H1N1 influenza A virus became less severe as time went on because people conferred immune protection against similar viruses earlier in their lives. In evolutionary theory, human-pathogen co-evolution is the adaptation of a virus to humans' immunological and cultural responses to that virus, and vice versa [172–174]. There is a delicate balance between the survival advantages of humans and pathogens, in this case the H1N1 influenza A virus, whereby humans mount increasingly adaptive immunological defenses against the once-novel virus, and the virus continues to evolve in response to those immunological defenses so that it can continue to infect, reproduce, and spread [175]. Ultimately, this process produced the phenomenon by which the novel H1N1 influenza A virus that circulated the globe in 1918–20 for the first time became the viral ancestor of H1N1 influenza A virus strains that co-circulate today as typical and somewhat predictable viral strains.

This evolutionary framing could help explain other results reported in the above discussion in a way that adds to—rather than detracts from—Noymer and Garenne's [54] explanations regarding selective mortality. For example, the increase in survivorship from respiratory diseases in Newfoundland [5] and the identification of recrudescent waves with reduced severity in post-pandemic years in the Maltese Islands [122] suggests that there were improvements in population health, potentially and partially linked to human-pathogen co-evolution, that can produce the observed improvements in epidemiological and

demographic outcomes. As discussed, van Doren and Kelmelis [5] reported significant improvements in survivorship for respiratory diseases, including influenza, from 1909–11 to 1933–35 in Newfoundland. This could be a product of selection against those with tuberculosis, those with lower underlying health status due to nutritional stress, and the cumulative effects of adaptation to the ubiquitous novel influenza virus. Researchers have not used the Newfoundland data to systematically identify recrudescent influenza waves beyond 1920—not because the recrudescent waves do not exist, but because this has not yet been a point of research. More research should be done to corroborate the results reported for Copenhagen and the Maltese Islands [121, 122].

Further, the work done in van Doren and Kelmelis [5] and Tripp et al. [122] is done from a biocultural anthropological perspective, which is not only a fundamentally evolutionary perspective but depends heavily on the synthesis of human biology and culture to produce observable population health differences [38, 40–42]. Thus, human-pathogen co-evolution is not the singular explanation for the observed differences, especially given the time frame over which these changes occur. Schmidt and Sattenspiel [167] and van Doren [103] point out that there were substantial demographic and epidemiological transitions occurring in the background in the early 20th century in Newfoundland, potentially influencing both 1918 influenza pandemic experiences and the population health of the surviving people in ways unrelated to the pandemic virus itself. In Newfoundland, public health advancements during the early 20th century, such as improved water sanitation procedures and improvements in nutrition, were key in the infectious disease decline through the 1930s and 1940s [4, 167]. Schmidt and Sattenspiel [167], specifically, show that the second epidemiological transition was underway in the urbanizing region of Newfoundland by 1939, but the full scope of the transition has not been identified there as of this writing, and it had not yet begun in the more rural regions of the island. In this way, it is possible to conceive of a situation where cultural adaptations (public health advancements) augmented baseline health, which bolstered the surviving populations' ability to immunologically adapt to the novel influenza virus in post-pandemic years.

Similarly, Zuckerman et al. [123] place their post- to pre-pandemic survivorship analyses in the MSA within the context of biocultural anthropological theory. One of the main differences between Zuckerman et al.'s [123] results and the improvements in survivorship and morbidity in Newfoundland and the Maltese Islands is that the MSA is neither a representation of the general population nor even of Mississippi, but a highly stigmatized and marginalized population. By the early 20th century, most patients in the MSA were Black and members of the labor and working classes [176]. Stigma is "... a process through which people become labeled in ways that are morally discrediting" (e.g., [177, 178]), which can involve not only barriers to accessing important healthcare [179], but also through the psychosocial and physiological stress of embodying a marginalized identity [63, 180]. Thus, stigma and marginalization are important determinants of biocultural and biosocial health disparities [181]. These social determinants of health likely had strong bearing on population health within the MSA for long before and after the 1918 influenza pandemic and may not have improved under the same biological and sociocultural pressures that produced shifts in national-level populations. Indeed, this is an illustrative example of how large-scale patterns of total life expectancy improvements and the reduction of the sex-based differences in life expectancy for the United States are likely not applicable to particular pockets of the United States, especially in the case of a specific population of systemically marginalized MSA residents.

One thing that should be considered in terms of how the 1918 influenza pandemic impacted post-pandemic population health, and even the co-evolution of humans and the novel influenza virus and its subsequent viral descendants, is that the 1918 influenza pandemic may not, in fact, have been severe enough to produce dramatic shifts. As discussed, Sattenspiel et al. [4] did not identify any substantial changes in age-sex histogram forms in the censuses following the 1918 influenza pandemic (1921, 1935). Beyond the subsequent analyses of fertility and cause-specific mortality, Sattenspiel et al. [4] concluded that, if there were any demographic impacts of the 1918 influenza pandemic in Newfoundland, they were likely short-lived, if they existed. This is not to understate the severity of the pandemic, the tragedy of up to 100 million deaths globally, or the social and biomedical upheavals that followed the pandemic. However, the 1918 influenza pandemic resulted in a global mortality of approximately 2.5% in a population of 1.8 billion with the highest death count estimates. For example, in contrast, the Black Death likely caused the deaths of one-third to half of a much smaller European population in the 14th century [182], which likely produced substantially higher stochasticity in human demographic—and possibly genetic—consequences. On the global scale, the 1918 influenza pandemic may not have produced enough mortality to substantially alter demographic trajectories. However, because of the clear heterogeneity in severity, it is worth continuing to investigate potential evolutionary impacts on a smaller population level.

In reference to the set of 11 papers on the fetal origins of adult health and socioeconomic disparities, these papers are united in that they each seek to answer the same basic question: "How does exposure to a stressful fetal environment during the 1918 influenza pandemic impact adult health and livelihood?" Thus, the goal of each of these papers is the same, and the alignment of results produces a relatively compelling picture of this phenomenon. This is distinct from the literature on demographic and epidemiological impacts of unequal morbidity and mortality on long-term outcomes, because the analysis of post-pandemic impacts is more commonly embedded within another set of analysis with variable goals. Based solely on the geographic scope of knowledge produced, the fetal origins literature discussed here is certainly broader than that of demographic and epidemiological outcomes with the inclusion of multiple papers using South American and Asian data. Overall, there are a couple generalizations that we can make about the outcomes of this set of fetal origins of inequality literature.

First, the results of these papers seem to suggest that there are variable outcomes in long-term health and socioeconomic status based on sex. Fletcher [160] found that females were less

likely to get married and get married at younger ages, while males got married at slightly older ages. Further, exposed females were less likely to have children and had fewer children if they did. Finally, exposed females tended to have less educated spouses. Nelson [165] found that males were less likely to be educated, literate, and employed, and overall had lower wages. Mazumder et al. [164] showed that males had higher risk of cardiovascular disease but otherwise no sex-based risk for diabetes, and Cohen et al. [159] reported a minimal reduction in life expectancy for females. Based on these results, exposed males and females seemed to experience elevated risks in different realms, but these inequalities vary by population.

Second, in terms of socioeconomic inequalities, although many of these articles find evidence that exposure to the 1918 influenza pandemic *in utero* eventually led to identifiable socioeconomic inequalities decades afterwards, few operationalize socioeconomic status as a predictor of long-term outcomes. That is, it is difficult to tell if the inequalities observed were overrepresented among individuals with lower socioeconomic status, which is a dimension of inequality that is well known to be associated with more severe epidemiological outcomes during the pandemic [100]. In fact, one of the primary criticisms of Almond [101] is that there was no control for the confounder of the status of the family into which the exposed infant was born [183, 184]. As a result, it is possible that the effects of exposure on later life socioeconomic inequality are overstated [183]. One of these articles does actually account for socioeconomic status in their analyses: González et al. [88] found that the regions of Argentina that experienced the greatest reduction in educational attainment were regions with relatively lower socioeconomic status.

Most of the literature of adult health and socioeconomic status after fetal exposure during the 1918 influenza pandemic comes from the field of economics. Almond and Currie [185] acknowledge that economists are primarily interested in this topic because it has implications for human capital development. Indeed, one of the most substantial contributions of economists' work on fetal origins of 1918 influenza pandemic consequences is the focus on "non-health endpoints," or the consequences unrelated to morbidity and mortality. However, evolutionary anthropological frameworks of long-term consequences might argue that what some fields consider non-health endpoints are, in fact, critical to the overall social and ecological contexts that influence human biology, and vice versa. Anthropologists Morgan Hoke and Thomas McDade [186] make this claim in their proposal for the anthropological framework of *biosocial inheritance*, which they define as "the process whereby social adversity in one generation is transmitted to the next through reinforcing biological and social mechanisms that impair health, exacerbating social and health disparities" (p. 187, 194). This framework is strongly rooted in biocultural anthropology, with some key advancements.

Biosocial inheritance acknowledges the intergenerational and transgenerational transmission of biological and social characteristics, bringing into focus how both health and socioeconomic disparities interact and compound vertically through the generations. Hoke and McDade [186] point out that poor health and low socioeconomic status, especially under the influence of power structures that create inequality (a topic popular in political economy, e.g., [187, 188]), are not mutually exclusive. Thus, what economists have identified as "non-health endpoints" of consequences of fetal exposure to stressful intrauterine environments during the 1918 influenza pandemic may rather be more holistically conceptualized as *biosocial socioeconomic consequences* of several possible pathways of inheritance (e.g., epigenetics, hypothalamic-pituitary-axis functioning, and/or metabolism). Within this framework, it is also important to recognize that the maternal environment is not a *de novo* environment, meaning the health of the mother during pregnancy is not where the story begins; rather, the intergenerational focus of biosocial inheritance (as well as other evolutionary frameworks) elevates the importance of interrogating greater temporal depth (i.e., more generations) for determinants of health. A biosocial perspective of fetal origins and long-term consequences on health and socioeconomic inequality from the 1918 influenza pandemic may more explicitly seek to integrate ecological and social contexts of the people who suffered—but survived—the pandemic, rather than simply acknowledge that maternal mortality rates and birthweight are roughly correlated with negative health and socioeconomic markers in later life.

Finally, the fetal origins literature discussed in this paper collectively helps provide a foundation for an important point about the long-term impacts of pandemics: pandemics do not only unequally impact heterogeneous populations, but as an acute stressor on maternal health (and therefore fetal environment), they may indeed help *create* inequality. It is impossible to say whether the inequalities in health and socioeconomic status between the 1919 birth cohort and their immediate neighboring cohorts identified by these 11 papers would have existed if the 1918 influenza pandemic had never happened. However, apart from the large-scale analyses that identified no compelling evidence of unequal impacts on adult health [159], the general pattern is that individuals *in utero* during the major waves of the pandemic had lower educational attainment, lower income, higher risk of cardiovascular, respiratory, and kidney disease morbidity and mortality, more disabilities, and altered family formational outcomes. Health-related fields such as public health and biomedicine may be interested in identifying, treating, and preventing these outcomes on a proximate level, which are extremely important interventions to maintaining and advancing population health. However, evolutionary anthropological approaches may help provide context for why these risks exist in the first place, and later-in-life health-related risks can be unequally transmitted intergenerationally.

6 | Conclusion

The goal of this paper was to shape the gap in the literature surrounding post-1918 influenza pandemic inequalities compared with those that existed before the pandemic and those that have been observed during the pandemic. The analysis of the existing literature within this space indicates two things: (1) There were indeed dynamics in inequalities after the 1918 influenza pandemic in the historical populations studied; and (2) There is not enough continuity in methods of analysis or populations of study to generalize about those impacts, or to

confidently describe the heterogeneity of long-term impacts. All but two papers on demographic and epidemiological consequences focused on North American populations [4, 5, 54, 123], while the other two were on European populations [121, 122]. The fetal origins of inequalities on this topic covers a slightly broader scope, including papers from South America [88, 165] and Asia [162, 163]. As of now, there is an obvious bias in our understanding of post-1918 influenza pandemic inequalities toward Western populations. Additionally, I sought to place the results discussed within evolutionary anthropological frameworks, namely the epidemiological transitions, biocultural anthropology, and evolutionary medicine. Of the six papers discussed in this conceptual review, four were first authored (for the majority, wholly authored) by anthropologists [4, 5, 122, 123] and were placed primarily within these evolutionary frameworks. As such, this topic is already an important one in the anthropology of pandemics community. None of the research on fetal origins of inequalities was done by anthropologists, despite the significant conceptual and theoretical strengths that evolutionary anthropological theory can contribute to our understanding of long-term intergenerational transmission of health and inequality.

Overall, there is a tremendous body of work from diverse fields of inquiry that investigates the nature of the 1918 influenza pandemic through a more proximate lens. There is always room to increase our knowledge of the events of the pandemic as they happened. However, there is also substantial opportunity to expand the scope of historical pandemic inquiry, especially within the field of anthropology. There is much more work to be done in: (1) Expanding the geographic breadth of research on post-1918 influenza pandemic consequences, with particular emphasis on non-Western nations that have available data; (2) Analyzing data with methods that lead to results that are directly comparable with existing results in other populations, allowing researchers to learn more about the heterogeneity of specific long-term impacts; and (3) Engaging more purposefully with evolutionary theories to answer ultimate questions related to populations' unequal and long-lasting experiences with acute pandemic pathogens.

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Ethics Statement

The author has nothing to report.

Conflicts of Interest

The author declare no conflicts of interest.

Data Availability Statement

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Endnotes

[1] The first research articles and reports published on the 1918 influenza pandemic are useful and illuminating in regard to unequal pandemic experiences. Though they are not discussed within the contexts of the review here, interested readers should see Allard [107], Armstrong and Hopkins [106], Brewer [108], Collins [109], Crampton [110], Frankel and Dublin [111], Frost [71, 112], Jordan [114], Peltier [115], and Sydenstricker [116–119]. Readers should note that Allard 107 and Peltier [115] are written in French.

[2] Note that these measurements are rate ratios; therefore, they do not have associated units.

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