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THEORETICAL RESEARCH IN ANTHROPOLOGY AND THE HISTORY OF MEDICINE

COVID-19 and the Black Death: Nutrition, frailty, inequity, and mortality

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Abstract

Introduction: COVID-19 has challenged governments, healthcare systems, and individuals, drawing attention to the limits of modern technology and the extent of social inequity. Such challenges have directed attention to historical epidemics as repositories of data that could contribute to effective public health strategies and prognostic modeling. In light of the well-established correlation between frailty and mortality from COVID-19, this paper investigates the relationship between frailty, inequity, and mortality in the setting of the Black Death of 1346 – 1353, in order to identify trends over time in populations at the greatest risk of mortality during pandemics.

Methods: A comparative review examining relationships between frailty and mortality during the fourteenth century Black Death and the current COVID-19 pandemic was conducted. Data related to the Black Death are derived from osteological analyses of remains from mass plague graves in the United Kingdom, and data related to COVID-19 are derived from the United States,

Italy, and China.

Results: Nutrition – often a consequence of socioeconomic status – plays a crucial role in pandemic mortality. During the Black Death, people with pathological indicators that can reflect undernourishment due to inadequate caloric intake were more likely to die of plague. In the COVID-19 pandemic, higher obesity rates among populations of lower socioeconomic status in the United States reveal similar relationships among nutrition, frailty, inequity, and pandemic mortality.

Conclusion: Nutrition – often a consequence of socioeconomic status – has a crucial role in risks of mortality. Our analysis underscores the importance of addressing nutrition and frailty in present and future discussions of the prevention and mitigation of pandemics.

KEY WORDS: COVID-19; frailty; medieval plague; nutrition; selective mortality

Riassunto

Introduzione: Il COVID-19 ha sfidato governi, sistemi sanitari ed individui, indirizzando l'attenzione ai limiti della tecnologia moderna ed al livello di inuguaglianza sociale. Talis fide hanno concentrato l'attenzione alle epidemie avvenute nella storia come archive di dati che potrebbero contribuire ad efficaci strategie di sanità pubblica ed a modelli prognostici. Alla luce della ben stabilita correlazione tra fragilità e mortalità da COVID-19, questo articolo indaga sulla relazione tra fragilità, inuguaglianza e mortalità nel setting della Peste Nera del 1346 – 1353, per identificare gli andamenti nel tempo nelle popolazioni a maggiore rischio di mortalità durante le pandemie.

Metodi: E' stata realizzata una revisione comparativa che esamina le relazioni tra la fragilità e la

mortalità durante la Peste Nera del quattordicesimo secolo e l'attuale pandemia da COVID-19. I dati correlate alla Peste Nera sono stati ottenuti dalle analisi osteologiche dei resti umani delle fosse comuni di peste in Gran Bretagna ed i dati correlati al COVID-19 sono stati ottenuti da Stati Uniti, Italia e Cina.

Risultati: La nutrizione – spesso una conseguenza dello status socio-economico – gioca un ruolo cruciale nella mortalità pandemica. Durante la Peste Nera, le persone con indicatori patologici che possono riflettere la denutrizione dovuta ad inadeguato introito calorico avevano più possibilità di morire di peste. Nella pandemia da COVID-19, più alti tassi di obesità tra le popolazioni con basso status socio-economico negli Stati Uniti rivelano simili relazioni tra nutrizione, fragilità, inuguaglianza e mortalità pandemica.

Conclusione: La nutrizione – spesso una conseguenza dello status socio-economico – ha un ruolo cruciale nel rischio di mortalità. La nostra analisi evidenzia l'importanza di affrontare la nutrizione e la fragilità nelle discussioni presenti e future per la prevenzione e la mitigazione delle pandemie.

TAKE-HOME MESSAGE: Nutrition – often a consequence of socioeconomic status – plays a crucial role in pandemic mortality. During the Black Death, people with pathological indicators that can reflect undernourishment due to inadequate caloric intake were more likely to die of plague. In the COVID-19 pandemic, higher obesity rates among populations of lower socioeconomic status in the United States reveal similar relationships among nutrition, frailty, inequity, and pandemic mortality.

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INTRODUCTION

Overview

COVID-19 has challenged governments, healthcare systems, and individuals, drawing attention to the limits of modern technology and the extent of social inequity. Debates about comorbidities, predisposing physiologies, social contexts, and risk factors are central to assessments of the impact of COVID-19, from management of the individual patient's condition to decision-making surrounding public health policies and risk mitigation [1–3]. Re-opening strategies are justified or rejected with reference to the people who are at greatest and lowest risk of mortality [4]. Efforts to predict patients' susceptibility and prognosis are grounded in analyses of age and comorbidities.

As healthcare providers, scientists, public policy experts, economists, and citizens try to cope with the pandemic, attention has turned toward historical epidemics as repositories of data that could contribute to effective public health strategies and prognostic modeling. However, the vast changes in social circumstances and technologies that have occurred in the centuries between the present day and, for example, the Black Death, or even the much more recent 1918 influenza pandemic, can problematize such comparisons. In this article, we seek to mitigate some of these

comparative challenges by grounding our approach in the concepts of frailty and selective mortality.

Frailty: Modern definitions

Broadly defined in modern clinical and public health contexts, frailty (also termed comorbidity and disability) may be understood as a clinical syndrome that predisposes individuals to poor health outcomes and death [5]. In this condition, return to physiologic homeostasis becomes progressively more challenging following external assaults on the body, and eventually, minor disruptions to homeostasis in frail individuals produce outcomes severer than would be observed in more robust individuals. Although normal aging represents some broad compromise of homeostatic mechanisms, such changes may be understood as less severe and more systemic relative to frailty, which Fedarko [6, 7] has argued represents disproportionate compromise in specific body systems. This imperfect distinction between normal aging and frailty is one of many aspects that make frailty difficult to study; another is that frailty is multifactorial in its constituent parts and effects, an attribute described by the phrase ‘heterogeneity in frailty’.

In modern contexts, frailty scores and studies tend to focus on individuals of advanced age and encompass both clinical observations and biomolecular data. Fried et al. developed one of the earliest frailty indices, which includes evaluation of weakness, grip strength, energy, gait speed, and activity levels [8]. A clinical diagnosis of frailty is ascribed for low values measured for ≥ 3 of these five categories, and prefrailty is diagnosed when there are low values for 1-2 categories. Other indices incorporate data related to cognition, nutrition, and psycho-social status. Biomarkers have also been used, with hyperglycemia, sarcopenia, and elevated tumor necrosis factor-alpha, interleukin-6, interleukin-2, and interleukin-1 associated with frailty in patients of

advanced age [7, 9]. Correlations have also been demonstrated between high scores on frailty indices and the presence of cardiovascular disease [6, 10] and chronic obstructive pulmonary disease (COPD) [11]. Other indices, like the Charlson Index [12], have been developed to prognose life expectancy on the basis of comorbidity (frailty) burden. With the Charlson Index, patients are scored first on the basis of age (0 points for less than 50 years, 1 point for 50-59 years, 2 points for 60-69 years, 3 points for 70-79 years, and 4 points for greater than 80 years). Patients receive additional points, on a 1-4 scale, to indicate the presence and severity of 16 other conditions (myocardial infarction, congestive heart failure, peripheral vascular disease, cerebrovascular accident/transient ischemic attack, dementia, COPD, connective tissue disease, peptic ulcer disease, liver disease, diabetes mellitus, hemiplegia, chronic kidney disease, solid tumor, leukemia, lymphoma, and AIDS). When the study was initially published in 1987, 1-year mortality rates ranged from 12% for patients with a score of 0 to 85% for patients with scores greater than 5.

Female sex is correlated with higher frailty scores: although women live longer than men, their frailty index scores are higher than those of men, even when controlling for age. A meta-analysis of longevity studies from Europe, mainland China, Ireland, Australia, Hong Kong, and Canada found that this difference between male and female frailty index scores increases with age up to the late 80s, after which it plateaus [13, 14]. Multiple hypotheses have been offered to explain this difference. Some studies suggest the higher incidence of non-lethal diseases in females [14, 15], while others highlight differences in biomarkers, including levels of inflammatory cytokines, muscle bulk, and abdominal adiposity [13, 16, 17]. Psychosocial differences, including healthcare use and rates of self-reported disability, have also been cited [18]. Recent

research has increasingly pointed to the role that complement genes play in the sex-related prevalence and severity of some chronic conditions, including schizophrenia, systemic lupus erythematosus, and Sjogren's syndrome [19].

Diet is a topic of growing interest in frailty studies, as adherence to a Mediterranean diet correlates with low frailty index scores [20, 21]. Multiple mechanisms may explain this correlation. The Mediterranean diet is high in plant-based and fish protein and low in processed and refined carbohydrates [22], possibly counterbalancing the protein-deficient states that are associated with the sarcopenia that characterizes frailty. Hyperglycemia is a biomarker of frailty [7, 9], and the Mediterranean diet is not only low in sugar but also mitigates insulin resistance [22, 23], thereby combatting hyperglycemic states. Additionally, adherence to a Mediterranean diet has been shown to reduce the risk of general cardiovascular disease, ischemic stroke, and coronary artery disease [24], and the presence of cardiovascular disease is correlated with higher frailty index scores. Obesity is also correlated with frailty, with obese and overweight adults reaching higher frailty index scores at younger ages compared to age-matched controls with normal-range BMIs [25, 26]; the Mediterranean diet has been shown in some instances to treat or to prevent obesity [27, 28]. Although the interactions between diet and frailty are multi-layered and complex, data indicate that diets generally recognized as health-promoting (such as the Mediterranean diet) are negatively correlated with frailty and comorbidity, and that markers of diets recognized as health-compromising (obesity, hyperglycemia) are positively correlated with frailty and comorbidity.

Frailty: Paleoepidemiological contexts

Although modern studies of frailty tend to focus on individuals of advanced age, studies of

epidemic disease in past populations have demonstrated relationships between skeletal markers of frailty and increased risk of death in people of all ages. In bioarchaeological contexts, frailty is often defined as the age-standardized relative risk of death [29]. This conception of frailty is appropriate to apply to samples of dead individuals, for whom the measures of frailty informative in modern clinical settings, such as grip strength or inflammatory cytokine levels, cannot be used. Acknowledged biases in skeletal samples affect assessments made about the health of the populations those skeletons are presumed to represent. Of particular concern for paleoepidemiological studies are the relatively low sensitivity and specificity of skeletal pathological lesions; in brief, many diseases produce similar (if not indistinguishable) skeletal pathologies, and often only a small proportion of people with a disease (e.g., tuberculosis) known to be capable of causing a skeletal response will actually produce bony lesions [30].

Examinations of health in the past must also contend with the issues of heterogenous frailty and selective mortality [31]. Heterogeneous frailty, or variation in susceptibility to disease or risk of death, exists for a variety of environmental, genetic, epigenetic, biological, and social factors, and much of that heterogeneity is unobservable in human skeletal remains. Mortality is selective with respect to frailty, such that the individuals who die at each age, and thus enter the skeletal samples available to bioarchaeologists, are unlikely to represent all individuals alive at that age, but rather are biased toward the most frail. In combination, these factors make it difficult to infer frailty, or health more broadly, in skeletal samples when relying on the presence or absence of skeletal lesions alone. However, careful selection of study population and the use of age-structured data and appropriate mortality models can mitigate some of these biases, enabling paleoepidemiologists to examine disease dynamics in the context of factors that exist or have

parallels in populations today [32].

Aim of the study

Using concepts of frailty from both modern medical and anthropological contexts, we highlight in particular the crucial role that nutrition – often a consequence of socioeconomic status – occupies in risks of pandemic mortality. Our analysis underscores the importance of addressing nutrition and frailty in present and future discussions of the prevention and mitigation of pandemics.

METHODS

A narrative review of primary data and research papers related to frailty and comorbidities in the Black Death of 1346 – 1353 and the COVID-19 pandemic was carried out in order to assess relationships between frailty and mortality. Data related to the Black Death were derived from osteological analyses of remains from mass plague graves in the United Kingdom; the review focused on publications related to London's East Smithfield cemetery, a known plague cemetery. Relying on skeletal data from the East Smithfield cemetery mitigates potential uncertainties associated with the assessment of frailty and selective mortality in skeletal remains; these uncertainties and the unique character of the East Smithfield assemblage are discussed further below. Data related to COVID-19 were derived from the United States, Italy, and China. Analyses of mortality patterns associated with COVID-19 in multiple global settings are being rapidly published in an ever-evolving environment; even basic epidemiological parameters related to COVID-19, including its prevalence, its attack rate, and its mortality rate are adjusted in real-time as new data are disseminated. At the time of manuscript preparation, comparisons between Black Death and COVID-19 data were based on COVID-19 epidemiological studies

that focused on assessment of comorbidity (frailty) and calculation of the case fatality rate in countries that had experienced the highest case numbers and mortality: China, Italy, and the United States.

Data on the Black Death

Paleoepidemiological analysis of the relationships between frailty at all ages and mortality in the setting of epidemic disease provides an ideal opportunity to examine health outcomes in the past and yield information that is potentially relevant to public health efforts today. Previous paleoepidemiological work has examined mortality patterns during the 14th-century Black Death (a pandemic of plague across Afro-Eurasia caused by *Yersinia pestis* [33]), specifically evaluating whether mortality was selective during one of the worst pandemics in human history [34, 35]. This research on pandemic mortality focused on the East Smithfield cemetery from London, a burial ground that was established for and used only during the Black Death *c.* 1349-1350 [36]. The cemetery was excavated in the 1980s, yielding the skeletal remains of over 600 individuals (of the estimated 2400 people originally buried there) now available for study at the Museum of London [36]. Because all of the people buried in East Smithfield died during the epidemic in London, research using this cemetery does not require diagnosis of the disease of interest via skeletal pathologies, thereby avoiding the limitations of low sensitivity and specificity of skeletal lesions. Instead, skeletal pathologies evident on the individuals interred in East Smithfield can be used to examine whether the Black Death disproportionately killed people who had previously experienced the types of physiological stress (including nutritional stress) capable of prompting a skeletal response.

Previous research focused on several skeletal pathologies, or stress markers, that reflect

conditions during development. These include linear enamel hypoplasia (lines of relatively thin enamel on the surface of a tooth crown that reflect interruption of ameloblast activity) and relatively short adult stature [37–39]. These both provide evidence of factors such as malnutrition or episodes of infectious disease during childhood or adolescence that produced growth disturbances, i.e., causing the body to temporarily shift resources from growth and development toward tissue maintenance or immune responses. Similarly, cribra orbitalia, a porous lesion on the orbital roof, typically forms during the first 12 years of life and is often attributed to childhood anemia, which can have a dietary etiology [40]. In addition to these markers of early life stress, this work also examined periosteal new bone formation, an abnormal plaque of bone that is produced in response to infection or trauma to the periosteum and can occur at any point during life [41, 42]. These skeletal markers were initially evaluated in a pre-Black Death, non-epidemic medieval skeletal sample. This sample approximates, as closely as is feasible in paleoepidemiology, a 'control' sample for the Black Death skeletal sample. Analysis with parametric hazards models, suited to the relatively small samples typical of bioarchaeology, indicated that each skeletal marker was associated with elevated risks of death under conditions of normal, medieval mortality [34]. These markers are therefore reasonable indicators of frailty for use in the analysis of selective mortality during the Black Death.

Among people buried in East Smithfield, those who exhibited these stress markers faced higher risks of death compared to their age-peers without them, indicating that the Black Death was selective with respect to frailty [34, 35]. Such variation in risk of mortality is perhaps not entirely unexpected, as most causes of death are selective with respect to frailty or underlying health condition, though the catastrophic nature of the Black Death has led some to assume otherwise.

In some cases, the etiologies associated with these stress markers were ongoing at the time of the Black Death (as evidenced, e.g., by the presence of unremodeled new bone formation) and thus represent comorbidities with plague, whereas others occurred several years before death during the epidemic. In the latter case, the stress markers might indicate an individual who had high frailty because of those early life events and/or continued exposure to disadvantageous living conditions. Many of these stress markers might have been produced by poor nutritional status, either through the direct negative effects of malnutrition on growth, or indirectly by interfering with immune responses (in the short-term) or with the development of the immune system (a long-term outcome) and thus increasing susceptibility to infection. It is important to note that some of the people who died during the Black Death might also have previously suffered through severe famines in the late 13th- early 14th century, such as the Great Famine of 1315-1317, or experienced prolonged dairy- and animal-protein deprivations caused exacerbated by the Great Bovine Pestilence, *c.* 1319-1320 [43]. In both instances, the increased scarcity of nutritious food would have exerted greater pressure on prices, resulting in those of lower socioeconomic status suffering disproportionately greater caloric deprivations.

In addition to yielding evidence of the negative effects of skeletally discernible physiological stress on risks of mortality during the Black Death, previous research on East Smithfield also examined age- and sex-related patterns of mortality. Hazards analysis revealed an increasing risk of death with age for adults during the epidemic (insufficient sample sizes of infants and children precluded informative analysis of mortality risks for non-adults) [43]. No significant difference in risk of death was detected between the sexes [44]. However, sex differentials in mortality favoring males have been estimated from historical data from plague outbreaks in the

Netherlands from the Black Death up through 1450 [45]; given the nature of the available data, it is not entirely clear whether this reflects a difference between the sexes in exposure to or susceptibility to plague.

Data on COVID-19

Analyses of mortality patterns associated with COVID-19 in multiple global settings are being rapidly published in an ever-evolving environment; even basic epidemiological parameters related to COVID-19, including its prevalence, its attack rate, and its mortality rate are adjusted in real-time as new data are disseminated. At the time of manuscript preparation, a joint World Health Organization-China study estimated an adjusted case fatality rate in mainland China of 1.4% [46]. In mid-March, the estimated case fatality rate was 7.2% in Italy [47–49] and 0.9% in South Korea [50]. A United States CDC case surveillance report spanning cases from January 22 to May 30, 2020, recorded case fatality rates of 6.0% for men and 4.8% for women. For patients with a preexisting health condition, the case fatality rate was 22.4% for men and 16.8% for women. For patients with no other health issues, the case fatality rate was 1.7% for men and 1.5% for women [51].

In most cases, infections are mild: a report from the Chinese Center for Disease Control and Prevention evaluating 44,500 confirmed infections reported that 81% of these patients had mild infections (mild or no pneumonia), 14% had severe infections (dyspnea, hypoxia, or more than 50% lung involvement on imaging within 24 – 48 hours), and 5% had critical infections (respiratory failure, shock, or multiorgan dysfunction). However, as data from China, Italy, and the United States indicate, although most infections are asymptomatic or mild, older patients are more likely to be hospitalized and more likely to die [2, 48, 51].

A study of patients in New York used the Charlson Index to assess comorbidities, frailty, and their association with mortality. When patients' comorbidities were scored with the Charlson Index, the median score for all patients was 4, corresponding to a 53% estimated 10-year survival and highlighting the high degree of frailty associated with these patients [2]. Although Charlson Index scores can theoretically reach as high as 37, scores of at least five have been associated with a 1-year mortality rate of 85% [12]. Similar trends in the presence of comorbid conditions and mortality rates associated with COVID-19 have been observed in China and in Italy [1, 52], with hypertension, diabetes, chronic obstructive pulmonary disease, cardiovascular disease, and cerebrovascular disease noted to be major risk factors. As described above, cardiovascular disease, obesity, and chronic obstructive pulmonary disease have been linked with increased frailty index scores [10, 11].

The correlations between COVID-19 and frailty are further demonstrated with reference to the nursing home population in the United States. Preliminary estimates from United States Centers for Disease Control data suggest that nearly one-third of COVID-19-related deaths in the United States have been nursing home residents or workers [53, 54]. While nursing home residents' proximity to one another has been identified as a contributory factor for higher rates of disease transmission, these outcomes are also affected by the disproportionately higher number of frail individuals living in nursing homes [6].

RESULTS AND DISCUSSION

Although the contexts and pathophysiologies associated with the Black Death and the present COVID-19 pandemic are very different, both pathogens demonstrate selective mortality patterns that share similar features. Recognizing these shared features and their implications for

individual and community health could assist in guiding public policy discussions regarding prevention and disease mitigation. We therefore highlight here features that are at least theoretically possible to change.

Nutrition and social inequity

Available skeletal data indicate that for people living in mid-fourteenth-century England, nutritional deficiencies contributed to increased mortality rates from the Black Death [55]. Following the Black Death, there is evidence of improvements in health in general in England; for example, survivorship increased and rates of some skeletal stress markers decreased in the period from 1350-1540 compared to pre-Black Death patterns [56]. Further, subsequent outbreaks of plague during the medieval period (also caused by *Y. pestis* [57]), though also quite devastating, produced lower rates of mortality [56, 58–60]. Acquired immunity likely contributed to the reduced rates of plague mortality observed after the initial outbreaks. However, such reductions in plague mortality and the broader trends in health might also be attributable to adjustments in wages and costs of living that made higher-quality, more nutritious food more available to greater numbers of people. Economic shifts after the Black Death, occurring at least in part because of the dramatic depopulation produced by epidemic, increased the standard of living for those who survived and reduced income-related disparities in access to fresh, nutritionally diverse food [61–63]. Adequate access to high-quality nutrition, which bolsters the immune system [64], was likely at least partially responsible for apparent improved general health after the Black Death. However, the effects of frailty on risks of mortality in subsequent outbreaks of plague appear to have persisted, at least over the short-term, based on analyses of individuals who died during the second outbreak of plague in England, *c.* 1361 [60].

Although food insecurity is an increasing problem both in the United States and globally, a parallel problem is equitable access to fresh, nutritious fruits and vegetables, lean protein, and foods produced without processed carbohydrates. This lack of access to nutritious food has fueled consumption of cheaper, high-calorie, processed foods – high in sugar and salt – that contribute to the development of obesity, hypertension, cardiovascular disease, and diabetes [65]. The term 'obesity epidemic' has often been applied to the rapid global growth in the number of individuals with Body Mass Index measurements in the overweight, obese, or morbidly obese ranges [66, 67], and preliminary data indicate that the collision between the obesity epidemic and the COVID-19 pandemic is responsible for a large proportion of deaths [2, 68]. The United States Centers for Disease Control, for example, classify severe obesity alongside asthma as a high-risk condition for the development of severe COVID-19-related outcomes [54]. Data from multiple countries have linked obesity to increased mortality from COVID-19: as an independent risk factor (possibly by contributing to immune system compromise [69]), as a condition that reduces biomechanical respiratory function [70], and as a physiological state that facilitates the development of other risk factors, such as cancer, cardiovascular disease, hypertension, and diabetes.

Socioeconomic contexts

Paleoepidemiological studies have indicated that nutritional deficiencies likely contributed to increased mortality during the Black Death in England. These nutritional deficiencies were produced or exacerbated by social inequalities that were increasing prior to the epidemic [56, 71]. Dramatic population growth prior to the Black Death outpaced economic growth and agricultural production; as a result, an estimated 70 percent of the English population was living

at or below the poverty line by 1290, and real wages for laborers reached a historic low two decades before the Black Death [71]. Socioeconomic status is not discernable in the medieval plague burial grounds that have been studied to date, precluding the direct assessment of the association between poverty and Black Death mortality using skeletal data. However, documentary evidence of variation in mortality by wealth, social position, or household size (which likely reflects socioeconomic status) during the Black Death and later plague outbreaks in England and other contexts has indicated that in some cases medieval and early modern plague disproportionately affected lower status and poor people [58, 60, 72–74]. Although this is not necessarily a universal or temporally consistent pattern [see, e.g., 75], in the interests of working toward alleviating some of the burden of disease today, we should perhaps privilege evidence of the existence of wealth inequalities during epidemics rather than the absence thereof. Paleoepidemiological studies have also contributed to arguments that the reductions in socioeconomic disparities emerging after the initial outbreaks may have subsequently improved the nutritional status of communities, thereby improving health for the population in general and perhaps lessening the mortality of later outbreaks. Such analyses underscore not only the nutritional components of selective mortality, but also the socioeconomic dimensions of it.

In the modern United States, the relationship between low socioeconomic status and poor nutritional status involves not only reduced access to calories, but also severely limited access to nutritious calories. The term ‘food desert’ describes an area in which there is a paucity of grocery stores selling fresh food, and ‘food swamp’ applies to an area with a high concentration of fast food and other ‘junk food’ options. Food swamps, more than food deserts, have been identified as stronger predictors of obesity, an effect increased in areas with greater income inequality [65].

This finding is supported by an Italian study showing that, even when individuals of lower socioeconomic status adhered to the Mediterranean diet, they did not see the same degree of health-protective benefits as did controls from higher-income households [76]. The lack of access to nutritious food that contributes to the development of conditions such as obesity, diabetes, and hypertension compounds the problem further once patients do develop these chronic health problems, as diet is a key component of their management and treatment [77]. Food insecurity and food inequity disproportionately affect Black individuals, Indigenous individuals, and People of Color (BIPOC): populations who experience the negative effects of racism [78,79] and have also been disproportionately affected by COVID-19 [80–82].

Strengths and limitations

Although many studies have drawn parallels between past pandemics and COVID-19 [83], we focus in particular on the role that nutrition has occupied in predisposing people and populations to selective mortality in the Black Death and COVID-19. Our discussion highlights the influence of nutritional compromise in pandemic mortality: undernourishment in the case of the Black Death, and overnourishment (contributing to diabetes, obesity, and hypertension) in the case of COVID-19. Parallels also emphasize that such disorders of nutrition – and hence, increased pandemic mortality – are related to social inequity. Limitations of this study include the challenges of comparing data sets from different time periods and different locations. Moreover, aspects of such comparisons rely on assessments of case fatality, which have been challenging to calculate for both the Black Death and for the present COVID-19 pandemic [84, 85]. Additionally, although this study relies on statistically analyzed data for both the Black Death and COVID-19, it does use statistical analysis in the comparison of the two sets of data.

CONCLUSION

The pathophysiology of COVID-19 is complex, and the mechanisms by which many factors – including frailty, diet, socioeconomic status, sex, racism, and genetics – contribute to clinical outcomes are still being vigorously investigated. Despite the present uncertainties, however, data from multiple countries have shown that metabolically-mediated conditions, including obesity and diabetes, are associated with higher mortality. Analyses of historical pandemics, including the Black Death, have similarly emphasized the role that nutrition plays in frailty and selective mortality. Moreover, paleoepidemiology research has suggested that reductions in social inequity are linked to increased availability of nutritious food, which in turn likely leads to improvements in health and reductions in mortality from infectious disease. Such studies may provide guidance about ways that governments, communities, and individuals can make changes – on structural and personal levels – to mitigate the effects of COVID-19 resurgences and of future epidemics.

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