Oral Health and Frailty in the Medieval English Cemetery of St Mary Graces

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ABSTRACT   The analysis of oral pathologies is routinely a part of bioarcheological and paleopathological investigations. Oral health, while certainly interesting by itself, is also potentially informative about general or systemic health. Numerous studies within modern populations have shown associations between oral pathologies and other diseases, such as cardiovascular disease, certain types of cancer, and pulmonary infections. This article addresses the question of how oral health was associated with general health in past populations by examining the relationship between two oral pathologies (periodontal disease and dental caries) and the risk of mortality in a cemetery sample from medieval England. The effects of periodontitis and dental caries on risk of death were assessed using a sample of 190 individuals from the St Mary Graces cemetery, London, dating to ~AD 1350–1538. The results suggest that the oral pathologies are associated with elevated risks of mortality in the St Mary Graces cemetery such that individuals with periodontitis and dental caries were more likely to die than their peers without such pathologies. The results shown here suggest that these oral pathologies can be used as informative indicators of general health in past populations. Am J Phys Anthropol 000:000–000, 2010.   ©2009 Wiley-Liss, Inc.

Various oral and dental pathologies are frequently analyzed as part of bioarcheological and paleopathological investigations of the skeletal remains of past populations. These pathologies include dental caries (i.e., cavities), enamel hypoplasia, periodontal disease, and antemortem tooth loss. Many biological anthropologists are interested in dental and oral health (from here subsumed under the phrase "oral health") per se and how they might reflect more general levels of health. Of particular interest to anthropologists are the ways in which oral health varies within a population according to sex, age, and social status, how diet affects oral health, and how oral health has varied over time in human populations (Moore and Corbett, 1971, 1973; Corbett and Moore, 1976; Cohen and Armelagos, 1984; Goodman et al., 1984, 1987; Hodges, 1987; Kerr, 1991; Sledzik and Moore-Jansen, 1991; Beckett and Lovell, 1994; Danforth et al., 1994; Sutter, 1995; Lukacs, 1996; Sakashita et al., 1997; Danforth, 1999; Scaronlaus, 2000; Robb et al., 2001; Steckel et al., 2002; Schollmeyer and Ii, 2004; Wols and Baker, 2004; Eshed et al., 2006; Lukacs and Largaespada, 2006; Oxenham and Tayles, 2006; Oztunc et al., 2006; Phillips, 2006; Boldsen, 2007; Lieverse et al., 2007; Paine et al., 2007; e.g., Starling and Stock, 2007; Temple and Larsen, 2007; Keenleyside, 2008; Rose and Vieira, 2008; Watson, 2008).

The advantage of oral health indicators for bioarcheological studies is that, given the highly mineralized nature of teeth, they provide a relatively durable record of the patterns of health within past populations (Roberts and Cox, 2003). Many anthropological studies examine oral pathologies as a proxy measure for general health. Several studies have demonstrated that enamel hypoplasias, enamel defects that reflect interruptions in enamel formation as a result of physiological stress, are reflective of general levels of health (e.g., Goodman and Rose, 1990; Boldsen, 2007), and many studies use enamel hypoplasia as a nonspecific indicator of stress or health in skeletal samples. But are other oral pathologies, which are also routinely examined in anthropological studies, also informative about more than just oral health? Are such oral pathologies, in fact, useful markers for patterns of systemic health? The focus of this article is the relationship between two types of oral pathology, periodontal disease and dental caries, and general health in past human populations. The analyses presented here address the question of whether certain oral pathologies are indicative of frailty (Vaupel et al., 1979), an individual's age standardized relative risk of death, in past populations. This article focuses on periodontal disease and dental caries in particular because they are the most common oral pathologies in modern populations (Rose and Vieira, 2008), and there is therefore a huge body of literature about their associations with general health (see examples below).

PERIODONTAL DISEASE

Periodontal disease (periodontitis) is characterized by a bacterial infection that causes inflammation and

destruction of gum tissue (gingiva), the periodontal ligament, root cementum, and alveolar bone. Periodontitis can be caused by a variety of pathogenic infectious agents that are found in oral biofilms (dental plaque), complex ecosystems including the oral microorganisms themselves and their byproducts surrounded by an extracellular matrix; these infectious agents include, among others, the bacteria *Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis,* and *Treponema denticola* and various herpes viruses (van Winkelhoff and Slots, 1999; Li et al., 2000; Slots, 2004). Many of the pathogens that cause periodontal disease are found in the mouths of healthy people, albeit at lower levels than are found in individuals with periodontitis (Meyer et al., 2008). Periodontal disease is very common in modern populations; for example, mild forms of periodontal disease may affect up to 90% of individuals worldwide, and moderate periodontitis affects ∼30% of adults in the United States (Slots, 2004; Pihlstrom et al., 2005). Many studies have established relationships between periodontal disease and risks of morbidity and mortality from other diseases in modern human populations. One study has found that among adults in the United States, periodontitis is associated with a 46% increased risk of all-cause mortality (DeStefano et al., 1993), and hundreds of studies have shown that periodontitis is a risk factor for such diseases as cardiovascular diseases, respiratory infections, certain types of cancer, Alzheimer's disease, diabetes mellitus, renal disease, necrotizing fasciitis, osteoporosis, and rheumatoid arthritis (e.g., see the following reviews: Hollister and Weintraub, 1993; Li et al., 2000; Kowolik et al., 2001; Slots, 2004; Pihlstrom et al., 2005; Johnson et al., 2006; Irwin et al., 2008; Williams et al., 2008).

Perhaps the best studied association between oral health and systemic disease is that existing between periodontal disease and cardiovascular diseases, including atherosclerosis and associated complications such as myocardial infarction and ischemic stroke (e.g., Joshipura et al., 1996; Dorn et al., 2000; Hujoel et al., 2000; Desvarieux et al., 2003; Joshipura et al., 2003; Grau et al., 2004; Pussinen et al., 2004a; Beck and Offenbacher, 2005; Mattila et al., 2005; Demmer and Desvarieux, 2006; Spahr et al., 2006). Cardiovascular diseases are the most common causes of death in industrialized nations, and given that both periodontal disease and cardiovascular diseases are very common throughout the world, understanding the association between the two is important from a public health perspective (Hujoel et al., 2000; Desvarieux et al., 2004). Studies of the association between periodontitis and systemic disease have attempted to determine the exact mechanism by which oral health is related to general health, i.e., whether periodontitis in some direct or indirect way causes or is caused by diseases in other areas of the body, or whether all of these diseases are caused by certain common underlying risk factors or are associated with immune response and are thus coincidentally associated. Several studies have found significant associations between periodontitis and cardiovascular disease, even after controlling for established cardiovascular risk factors that are potential confounders (e.g., age, sex, smoking, body mass, diet, and alcohol consumption) (Spahr et al., 2006). This suggests, to some, that periodontitis and cardiovascular disease are causally related (Beck and Offenbacher, 2005).

Periodontitis can cause chronic systemic infection or inflammation, and this might influence cardiovascular disease. The pathogens that cause periodontitis, or components of those pathogens such as endotoxins and outer membrane proteins, can enter the bloodstream through pockets of ulcerated gingival tissue that form as the disease progresses (Loos, 2005; Amabile et al., 2008). The body responds to the presence of such bacteria and bacterial antigens in the oral cavity and in the bloodstream through the production of proinflammatory immune cytokines, which can have systemic or distant effects (Loos, 2005; Spahr et al., 2006). Inflammatory cytokines can increase platelet aggregation in blood vessels, leading to or accelerating the progression of atherogenesis (the formation of plaques within arteries) (Amabile et al., 2008; Kamer et al., 2008; Watts et al., 2008). The oral pathogens themselves, once disseminated from the oral cavity, may also mediate coagulation directly or invade arterial walls and initiate or exacerbate the inflammatory response of atherosclerosis (Dorn et al., 2000; Demmer and Desvarieux, 2006). Chronic periodontitis may also influence lipoprotein levels and thereby favor atherogenesis (Katz et al., 2002; Pussinen et al., 2004b; Mattila et al., 2005). Periodontal pathogens have been found in coronary artery atherosclerotic plaques, which suggests that these pathogens played a direct role in the formation of such plaques (Pucar et al., 2007).

Periodontal disease appears to be a risk factor for pulmonary disease, including pneumonia and chronic obstructive pulmonary disease (which encompasses chronic bronchitis and emphysema) (Hayes et al., 1998; Terpenning, 2001; Terpenning et al., 2001; Scannapieco et al., 2003; Scannapieco and Rethman, 2003). Awano et al. (2008) found that in elderly individuals, periodontitis significantly increases the risk of mortality from bacterial pneumonia; improved oral care, including the use of antiseptic rinses and mechanical cleaning, both of which lower levels of bacteria in the oral cavity, has been shown in some studies to reduce the incidence of and mortality from pneumonia among patients in long-term care facilities (Yoneyama et al., 2002; Paju and Scannapieco, 2007; Pan et al., 2009). Dental plaque provides a reservoir of both oral and respiratory bacteria, which, in addition to causing periodontitis, can be shed into the saliva and then aspirated into the lungs, subsequently causing pulmonary infection. Pulmonary infection might also arise by spread of bacteria from sites of periodontitis in the oral cavity to the lungs via the bloodstream (i.e., bacteremia) (Paju and Scannapieco, 2007). If aspirated into the lungs, periodontal pathogens may facilitate the invasion of airway epithelia cells by respiratory pathogens, thereby assisting in an essential step in the infection process. As described above for cardiovascular disease, periodontal pathogens may also stimulate an inflammatory response, which facilitates the onset or progression of respiratory disease (Pan et al., 2009).

Periodontal disease is also associated with increased risks of certain types of cancer, including oral, esophageal, upper gastrointestinal, gastric, pancreatic, lung, and kidney cancers (Zheng et al., 1990; Stolzenberg-Solomon et al., 2003; Abnet et al., 2005; Meyer et al., 2008). For example, Michaud et al. (2008) found that periodontal disease is significantly associated with increased risks of lung, kidney, pancreatic, and hematological cancers (after controlling for smoking, diet, body mass, and other potential confounders). The observed association between periodontitis and cancers might reflect an

impaired immune system that is unable to clear an infection and also insufficiently monitors and controls tumor growth (Michaud et al., 2008). Alternatively, the inflammatory markers produced as a result of chronic periodontitis might affect cell growth control and lead to carcinogenesis (Meyer et al., 2008).

Several studies have also demonstrated associations between periodontitis and the following systemic diseases: Alzheimer's disease, obesity, diabetes, and kidney disease (Saremi et al., 2005; Khader et al., 2006; Shultis et al., 2007; Taylor and Borgnakke, 2008; Khader et al., 2009; Kshirsagar et al., 2009). As is true with respect to the diseases described in detail above, there is an ongoing effort to more clearly understand the mechanism by which periodontitis is linked to these systemic diseases. Of particular concern to many researchers is demonstrating how treatment of periodontitis might lower the risks of morbidity and mortality associated with these other diseases (Kamer et al., 2008; Watts et al., 2008).

In addition to the above diseases, periodontitis has also been linked to adverse pregnancy outcomes, including preeclampsia, spontaneous preterm birth, low birth weight, and stillbirth (Holmstrup et al., 2003; Goepfert et al., 2004; Boggess, 2005; Boggess et al., 2006; Mobeen et al., 2008; Ruma et al., 2008; Polyzos et al., 2009). According to some researchers, the association between periodontitis and adverse pregnancy outcomes is likely the result of a maternal systemic inflammatory response initiated by chronic periodontitis; this inflammatory response results in abnormal placental or fetal growth (Boggess et al., 2006; Ruma et al., 2008).

Dental caries

Dental caries is characterized by the localized demineralization of the hard tissues of the teeth (enamel, dentine, and cementum) by weak organic acids that are the byproducts resulting from bacterial fermentation of dietary carbohydrates (Larsen, 1997; Selwitz et al., 2007). The oral bacteria ultimately responsible for caries formation (primarily *Streptococcus mutans*, *Streptococcus sobrinus*, and *Lactobacillus* spp.) reside in the oral biofilm, and carious lesions form when the biofilm remains on the surface of a tooth for an extended period of time (Selwitz et al., 2007). Dental caries can affect both the crowns (coronal caries) and roots (root caries) of teeth. Dental caries begins as subsurface demineralization of the hard dental tissues beneath the biofilm, which, if unchecked, can cause cavity formation (cavitation) (Selwitz et al., 2007). Dental caries is one of the most common chronic diseases in modern populations (Selwitz et al., 2007), and according to Misra et al. (2007), it is the most common chronic disease in children worldwide.

As is true of periodontitis, several studies have found an association between dental caries and general health status (e.g., Acs et al., 1999; Joshipura et al., 2006; Ylostalo et al., 2006; Misra et al., 2007). Infection can spread from dental caries to other tissues in the mouth and elsewhere in the body, and these infections can potentially be life-threatening (Ngoenwiwatkul and Leela-Adisorn, 2009). As with periodontitis, caries can lead to a systemic inflammatory response with adverse health outcomes. For example, coronal caries can lead to inflammation of the dental pulp, which, in turn, can lead to a systemic inflammatory response that may increase the risk of coronary heart disease (Joshipura et al., 2006). The

development of caries might be the result of the body's inability to control the proliferation of cariogenic oral bacteria; as with periodontitis, the link between caries and systemic disease might reflect general inadequacies of the body's immune response (Acs et al., 1999). Some studies have shown an association between nutritional status and early childhood caries such that underweight or obese children are more likely to have severe dental caries than children of normal weight (Miller et al., 1982; Ayhan et al., 1996; Acs et al., 1999; Thomas and Primosch, 2002; Willershausen et al., 2004, 2007; Sheiham, 2006; Marshall et al., 2007; Alm, 2008; Gerdin et al., 2008; Oliveira et al., 2008; Ngoenwiwatkul and Leela-Adisorn, 2009). Acs et al. (1999) found that following comprehensive dental treatment, children with early childhood caries experienced catch-up growth, which suggests that caries negatively affects growth. Dental caries can adversely affect growth and development by interfering with normal food consumption; dental caries can cause toothaches, which negatively affect food intake and thereby contribute to low weight gain in children (Acs et al., 1999). Furthermore, the pain associated with dental caries can disrupt sleep, which can affect the production of glucosteroids and therefore adversely affect growth (Sheiham, 2006). The link between dental caries and obesity might reflect a form of malnutrition whereby individuals consume an abundance of foods that are cariogenic and full of "empty calories" but do not provide sufficient nutrients (Miller et al., 1982). Given that obesity is a risk factor for several diseases, the link observed in some studies between dental caries and obesity might also mean that caries is linked to the negative outcomes associated with obesity.

Periodontitis and dental caries in past population

The observed association between oral pathology and systemic health in modern populations raises the following question: how was oral health associated with general health in past populations? Were periodontitis and dental caries associated with other diseases and elevated risks of mortality in past populations, as they are in populations today? To address this question, this article examines the relationships between periodontitis and dental caries and the risk of mortality in a cemetery sample from medieval England. The goal is to address the question of whether these oral pathologies are indicative of frailty (an individual's age-adjusted relative risk of death) in past populations and can thus be used informatively in investigations of general heath in the past (Vaupel et al., 1979).

Numerous studies have examined patterns of periodontitis and dental caries in historic populations, and there are abundant data regarding the frequencies of these oral pathologies in various samples. Moore and Corbett examined patterns of dental caries in Britain from the Iron Age through the nineteenth century (Moore and Corbett, 1971, 1973, 1975; Corbett and Moore, 1976) and found that for much of the 2,000-year period between the Iron Age and the end of the Medieval period, overall caries prevalence was low (less than 20% of teeth, on average, were carious). Studies of various medieval populations have found frequencies of individuals with caries that range from 5 to nearly 50% (Kerr et al., 1988; Kerr, 1990; Šlaus, 2002; Vodanovic et al., 2005; Caglar et al., 2007) and percentages of teeth that are carious that range from less than 5 to nearly 25%

(Lunt, 1974; Kerr et al., 1988; Kerr, 1990; O'Sullivan et al., 1993; Watt et al., 1997; Esclassan et al., 2009). Investigations of dental caries in more recent populations, such as nineteenth-century England (O'Sullivan et al., 1993; Whittaker and Molleson, 1996), late nineteenth-/early twentieth-century Portugal (Wasterlain et al., 2009), nineteenth-century Canada (Saunders et al., 1997), and nineteenth- and early twentieth-century US (Sledzik and Moore-Jansen, 1991; Sutter, 1995; Winchell et al., 1995; Davidson et al., 2002; Higgins et al., 2002; Wols and Baker, 2004; Phillips, 2006), found frequencies of individuals with caries ranging from 27 to nearly 90% and frequencies of carious teeth ranging from 11 to 70%. Investigations of periodontitis in samples from fourth-century BC Turkey (Oztunc et al., 2006), medieval France and Scotland (Kerr, 1991; Chazel et al., 2005), eighteenth-century England (Kerr, 1994), and the late nineteenth-century US (Rose and Vieira, 2008) have found frequencies of periodontitis ranging from 18 to 100%. The above summary is not exhaustive, but even this sample of studies demonstrates the variation that has been observed with respect to dental caries and periodontitis. The possible reasons for such variation are described in the Discussion section.

These previous studies provide invaluable data regarding the patterns of oral health in past populations and the relationships between oral health and diet, oral hygiene, dental care, and other factors. But they do not explicitly address the relationship between oral pathologies and risk of death. By incorporating a model of health and mortality, this study goes beyond looking at frequencies of certain dental pathology and determines how those pathologies actually affect risks of mortality. This study examines a single population to determine the effect of these oral pathologies on risk of death, thereby providing a clearer picture of the association between oral health and general health in the past than is possible when comparing different populations.

MATERIALS AND METHODS
St Mary Graces cemetery and the skeletal sample

St Mary Graces cemetery. The St Mary Graces cemetery in London was associated with the Cistercian Abbey of St Mary Graces, which was established shortly after the Black Death ended in London in 1350 (Grainger and Hawkins, 1988). The Abbey of St Mary Graces was established on land northeast of the Tower of London (Grainger et al., 2008). The Abbey of St Mary Graces was in use until the Reformation in 1538. Monks and important lay people were buried within the Abbey's church and chapels, but the cemetery itself was used for the general population (Grainger and Hawkins, 1988; Rogers and Waldron, 2001). The St Mary Graces cemetery provides a good sample of the late Medieval population of London, as it contains individuals of all ages, both sexes, and both higher and lower socioeconomic statuses. St Mary Graces was excavated by the Museum of London Department of Greater London Archaeology (now the Museum of London Archaeology Service) in the 1980s as part of the Royal Mint site, which also revealed the East Smithfield Black Death cemetery (~1348–1350) and a Royal naval victualling yard (~1560–1785) (Hawkins, 1990). Excavation of St Mary Graces revealed 133 skeletons from within the Abbey church and chapels and 310 within the larger lay cemetery (Grainger and Hawkins, 1988).

Skeletal sample. A sample of 190 individuals from St Mary Graces was used in these analyses. This sample represents all the excavated individuals in the cemetery who were preserved well enough to be scored for skeletal indicators of age and sex (in adults) and the presence of periodontitis and dental caries.

Age and sex estimation

Age estimation. Sub-adult ages (i.e., ages less than 18 years) were estimated using the diaphyseal lengths of major long bones (only for fetal and neonatal remains between ages 10 and 50 gestational weeks), epiphyseal fusion, and dental development and eruption (Moorrees et al., 1969; Gustafson and Koch, 1974; Scheuer et al., 1980; Smith, 1991; Scheuer and Black, 2000). Adult ages (i.e., ages 18 years or older) were estimated based on tooth wear (Brothwell, 1981) and age-related changes of the pubic symphysis (Brooks and Suchey, 1990), iliac auricular surface (Lovejoy et al., 1985; Buckberry and Chamberlain, 2002), and sternal rib ends (Iscan et al., 1984, 1985).

Sex estimation. Sex was estimated using standard methods based on sexually dimorphic features of the skull and pelvis (Phenice, 1969; Brothwell, 1981; Bass, 1987; Buikstra and Ubelaker, 1994).

Oral pathology

This study examines the relationships between risk of death and each of the following oral pathologies: periodontitis (periodontal disease) and dental caries. Periodontitis is caused by a chronic oral bacterial infection resulting in gingival inflammation and the gradual destruction of periodontal tissues and alveolar bone (Irfan et al., 2001). In skeletal material, periodontitis can be identified by the loss of alveolar bone, which exposes the underlying trabecular bone and thereby produces porosity (Clarke and Hirsch, 1991; Larsen, 1997) or causes the alveolar crest (AC) to recede relative to the cemento–enamel junction (CEJ) of the associated dentition (Larsen, 1997). For this study, periodontitis was scored as present if the alveolar bone displayed porosity or if the distance between the CEJ and AC was greater than 2 mm (see Fig. 1). The alveolar bone surrounding each tooth was scored individually. However, the analyses for this study incorporated pooled mandibular scores and pooled maxillary scores to assess periodontitis at a broader scale. The use of the distance between the CEJ and the AC to indicate periodontitis is potentially problematic, given that nonpathological processes can increase the distance beyond 2 mm (Costa, 1982; Clarke et al., 1986; Clarke, 1990; Clarke and Hirsch, 1991; Hildebolt and Molnar, 1991; Varrela et al., 1995; Hillson, 1996). For example, normal dental attrition that occurs with age can be compensated for by the continuing eruption of teeth throughout life to maintain lower facial height and the occlusal level of teeth (Clarke and Hirsch, 1991; Hildebolt and Molnar, 1991). Without simultaneous growth of the AC, this supereruption of teeth results in an increase in the CEJ–AC distance (Whittaker et al., 1990; Clarke and Hirsch, 1991; Hildebolt and Molnar, 1991; Varrela et al., 1995). Similarly, continued growth of the facial skeleton in adulthood also results in continued eruption of the teeth to maintain dental articulation

Fig. 1. Example of periodontitis on the right mandible. Destruction of cortical bone has exposed the underlying trabecular bone along the alveolar margin, producing a porous appearance. ©Museum of London.

Fig. 2. Example of dental caries on the left maxillary second and third molars. ©Museum of London.

(Clarke and Hirsch, 1991; Hildebolt and Molnar, 1991). In both cases, the increased distance between the CEJ and AC occurs without any pathological destruction of the alveolar bone. Use of the "greater than 2 mm" criterion to diagnose periodontitis can lead to the overestimation of the prevalence of periodontitis in past populations, as individuals with a CEJ–AC distance greater than 2 mm caused by nonpathological processes would be incorrectly diagnosed with periodontitis. This is potentially a problem for this study because the inclusion of such false positives for periodontitis in the sample would result in an underestimation of the risk of death associated with periodontitis. The possible effect of this potential inclusion of false positives on the results of this study is described below in the Discussion section.

Dental caries is characterized by the localized demineralization of the hard tissues of the teeth and can be initially identified as white spots on the surface of the tooth and later as destruction of the enamel or dentine. For this study, caries presence was assessed for the premolars and molars; carious lesions were scored as present if destruction of enamel or dentine was visible to the naked eye (Fig. 2 shows a rather extreme example). For this sample, the severity of caries ranged from small areas involving only the destruction of enamel and destruction of dentine with or without exposure of the underlying pulp chamber to gross destruction (the crown mostly destroyed); though data on level of severity are available for this sample, the analyses described here incorporated only data on the presence or absence of caries.

Model

The risk of death associated with oral pathology within St Mary Graces was analyzed using a multistate model of morbidity and mortality that was developed for paleodemography (Usher, 2000). In the three-state version of Usher's more general model, individuals can be in one of three nonoverlapping states: State 1 includes those individuals without detectable skeletal or oral lesions, State 2 includes those with detectable lesions, and State 3 is death (see Fig. 3). In the Usher model,

individuals can only be in one state at a time, and all individuals in a sample are in one of three states at a given time. Transitions between stages occur at age-specific hazards rates and the hazards for dying from States 1 and 2 can differ. By allowing variation in the transition rates between each of the two living states and death, the model can be used to estimate the differential risk of death associated with the living states. The model therefore allows one to investigate selective mortality with respect to skeletal lesions—that is, whether individuals with lesions are at higher risks of dying than their peers without lesions. If the risk of mortality is higher for any one state, the model can help resolve one aspect of the "osteological paradox" (Wood et al., 1992), i.e., whether an individual with a certain lesion is more or less healthy than an individual without that lesion. Some researchers (e.g., Ortner, 1991; Wood et al., 1992) have suggested that skeletal lesions might sometimes actually indicate a relatively healthy individual. This is based on the fact that visible skeletal lesions do not form immediately in response to trauma or disease, but rather take some time to form to a point at which they are detectable. Individuals with skeletal lesions might therefore have been healthier than their peers without lesions, given that they were able to survive malnutrition, trauma, or disease long enough for the skeletal lesions to form. Absence of a certain skeletal lesion might, in this scenario, indicate relatively poor health, as individuals without lesions were in such poor health that they succumbed to illness, trauma, or malnutrition and died before lesions ever formed. For this study, no assumption is made about whether skeletal lesions indicate good or bad health. Rather, the difference in risk of death between individuals with and without lesions was estimated by using the Usher model to evaluate what the lesions used in this study indicate about health in the St Mary Graces population.

As is true for any study using skeletal samples, all individuals in this study are observed in State 3, and data on age at death and presence of lesions allows for the estimation of all the parameters in the Usher model.

The baseline risk of death from State 1 is specified as a Siler mortality function $h_{13}(a) = \alpha_1 e^{-\beta_1 a} + \alpha_2 + \alpha_3 e^{\beta_3 a}$, a parsimonious parametric model of mortality that fits a wide variety of human mortality patterns (Gage, 1991; Wood et al., 2002). The Siler model has three components. The first component, $\alpha_1 e^{-\beta_1 a}$, describes the juve-
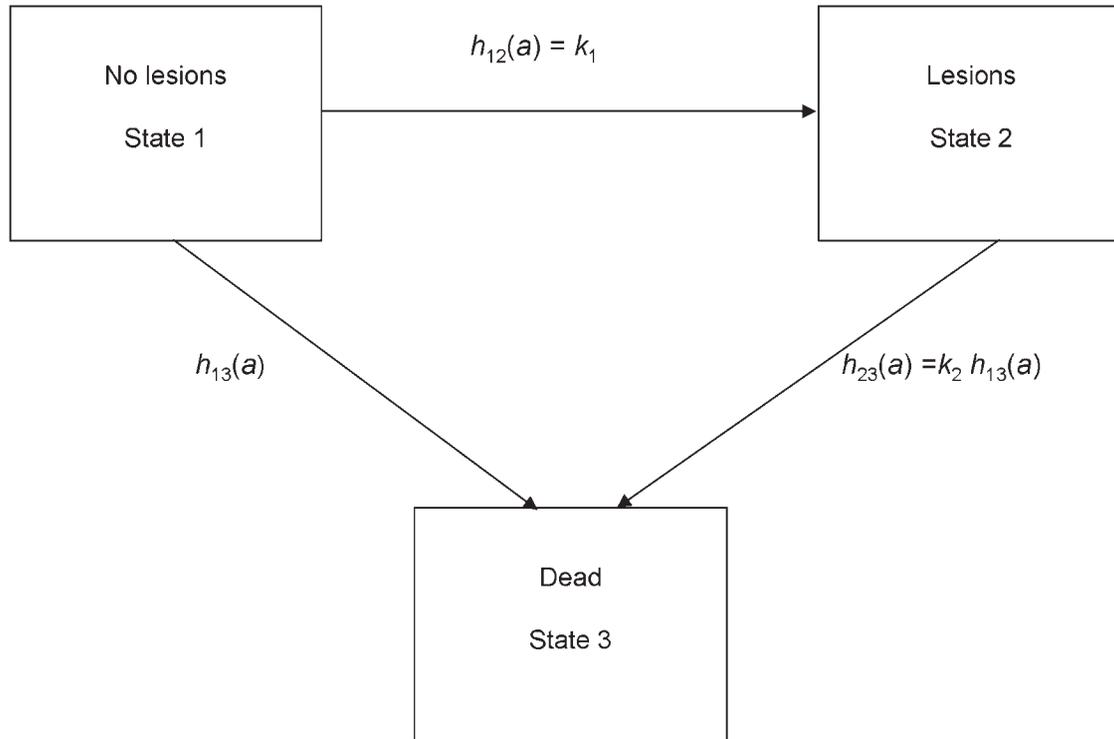
Fig. 3. Three-state model of morbidity and mortality. Individuals are born into State 1. The transitions between states occur at age-specific hazard rates $h(a)$, where a is age in years. The transition between States 1 and 3 follows the baseline Siler hazard function $h_{13}(a) = \alpha_1 e^{-\beta_1 a} + \alpha_2 + \alpha_3 e^{\beta_3 a}$, and the relative risk of death (frailty) associated with a given lesion type, k_2, acts to modify this baseline rate. The hazard of developing a detectable bony lesion, $h_{12}(a)$, is set equal to the constant k_1. Redrawn from Usher (2000).

nile risk that declines exponentially with age, where α_1 is the risk of death at birth caused by immaturity and β_1 is the rate at which this risk decreases with age a. The second component of the Siler model, α_2, is the constant age-independent or "baseline" risk that everyone within the population faces. The third component of the model, $\alpha_3 e^{-\beta_3 a}$, is the exponentially increasing senescent risk, where α_3 is the risk of death associated with senescence at the moment of birth and β_3 is the rate at which this risk increases with age (Gage, 1988). The three components of the Siler model are independent, and so surviving one component of mortality has no effect on risk of death from another component of mortality (e.g., surviving the juvenile component neither increases nor decreases an individual's risk of either the age-independent or senescent components of mortality) (Wood et al., 2002). The Siler model represents an advantage over typical life-table approaches to investigating mortality patterns, because it requires the estimation of only five parameters (Gage, 1990).

In the Usher multistate model, the hazard of developing lesions, $h_{12}(a)$, is estimated as a constant k_1, as the age of onset of conditions resulting in lesions is generally unknown in paleopathological studies. That is, for most skeletal lesions, one does not know the age at which an individual became ill or suffered some other physiological stress, nor does one know the timing of the development of the lesion. For simplicity, in this study, the age of onset of lesions is an exponential random variable. The hazard of dying from State 2, $h_{23}(a)$, is modeled as proportional to the baseline age-specific risk of dying from State 1. Under this specification, k_2 is a propor-

tional term on the Siler function and is thus independent of age; it indicates the proportional difference in risk of death between individuals with and without lesions. When k_2 is significantly larger than one, individuals with lesions faced an elevated risk of dying compared to similarly aged individuals without lesions. Because the model compares the risk of death between similarly aged individuals with and without lesions, it avoids overestimating the risk of death associated with age-progressive pathologies that are more likely to be found in older individuals. When k_2 is significantly lower than one, individuals with lesions faced a decreased risk of death compared to their age-peers without lesions. If k_2 is equal to one, individuals with and without lesions were at the same risk of death. Because of the error associated with age estimation (which was not taken into account when estimating the Usher model in this study), the k_2 parameter should be viewed as a general, qualitative measure of excess mortality associated with lesions. That is, the k_2 parameter estimates associated with various lesions are more important in terms of the patterns they reveal than their actual numerical values.

Model parameters were estimated using maximum likelihood analysis with Holman's program *mle* (Holman, 2005), using multiple start values to avoid local maxima. The model was fit separately to data on the presence of periodontitis and to data on the presence of caries. A likelihood ratio test (LRT) was used to assess the fit of the full model compared to a reduced model in which the value of the k_2 parameter was set equal to 1 ($H_0: k_2 = 1$); the LRT therefore tests the null hypothesis that lesions are not associated with elevated or decreased

Pathology	Location	n	% with pathology
Periodontitis	Mandible	182	37
	Maxilla	186	43
Dental caries	Mandibular premolars	144	15
	Maxillary premolars	118	20
	Mandibular molars	125	42
	Maxillary molars	97	55

Sample sizes refer to the number of individuals with appropriate elements preserved well enough to allow for scoring the presence of the corresponding pathologies.

risks of death such that individuals with and without oral pathologies were at the same risk of death. The LRT was computed as follows: LRT $= -2[\ln(L_{\text{reduced}}) - \ln(L_{\text{full}})]$, where LRT approximates a χ^2 distribution with $df = 1$.

RESULTS

The frequencies of dental caries and periodontitis within the St Mary Graces cemetery are shown in Table 1.

The maximum likelihood estimates of the excess mortality k_2 associated with periodontitis and premolar and molar dental caries, along with the standard errors associated with these estimates and results from the likelihood ratio tests, are shown in Table 2. These results indicate that these particular oral pathologies are, in fact, associated with increased risks of death in the St Mary Graces cemetery. It should be noted that these estimates have relatively large standard errors associated with them as shown in Table 2, and these standard errors are probably underestimated. To estimate the parameters of the model used in this study, point estimates of age were used without their corresponding errors. Because the errors associated with ages were not incorporated when the model was fit, the reported standard error might be underestimated to an unknown degree; readers should therefore view the standard error estimates with caution. However, it is encouraging that there is a consistent pattern among the results, as the k_2 estimates for all oral pathologies are greater than one. The k_2 estimates suggest that individuals with periodontitis or dental caries were at elevated risks of dying compared to similarly aged individuals without such oral pathologies. The consistent pattern of elevated risks of mortality suggests that these oral pathologies are indicative of higher frailty; however, the numerical value of the k_2 parameter should not necessarily be taken at face value given the uncertainty associated with the standard error estimates. The results of the likelihood ratio tests further suggest that these oral pathologies affected risk of death in St. Mary Graces, as they indicate that including the k_2 covariate improved the fit of the model with respect to all of the pathologies considered (though the results of the likelihood ratio tests were not significant for the models applied to periodontitis). These results suggest that oral health, as indicated by the observable oral pathologies examined here, is associated with frailty in the St Mary Graces population.

DISCUSSION

The results of this study suggest moderate levels of dental caries and periodontitis within the St Mary

Graces sample. As shown in Table 1, the frequency of premolar caries in the sample ranges from 15 to 20% and for molars the frequencies range from 42 to 55%. Most of these frequencies fall within the range of dental caries frequencies observed for other medieval skeletal samples. In a tenth- to eleventh-century Croatian sample, nearly 47% of individuals had caries (Vodanovic et al., 2005). Another study of a collection of sites in Croatia dating from 1050 to 1500 AD found that the frequency of caries ranged from 5 to 20% (Šlaus, 2002). Caglar et al. (2007) found in a thirteenth-century AD Byzantine population that ~14% of individuals had caries. Kerr et al. (1990) found that 44% of a medieval Scottish sample, from Linlithgow near Edinburgh, had caries; in another late medieval Scottish sample (1300–1600 AD) from Aberdeen, nearly 30% had caries (Kerr et al., 1988). Roberts and Cox (2003) summarize the findings from several sites in medieval Britain, and the overall prevalence of dental caries for these sites is 53%. The frequencies of caries observed in this study are not directly comparable to the results of several other investigations of dental caries in medieval populations because of differences in the reporting of data. Table 1 reports the proportions of individuals with teeth present who had at least one carious lesion, whereas numerous other studies report the percentage of carious teeth among all the teeth present (e.g., Moore and Corbett, 1973, 1975; Lunt, 1974; O'Sullivan et al., 1993; Watt et al., 1997; Esclassan et al., 2009). Moore and Corbett (1975) found that in a medieval British sample, 0–20% of premolars and molars had caries, depending on age. Of the deciduous molars present in their sample from late medieval Britain (thirteenth- to fifteenth-century AD), O'Sullivan et al. (1993) found that 23% had caries. Nearly 18% of teeth in Esclassan et al.'s (2009) sample from medieval France were carious. Only about 6% of teeth in two different medieval Scottish samples studied by Lunt (1974) and Watt et al. (1997) were carious. It is clear from all these studies, regardless of reporting method, that there was variation among these medieval samples with respect to frequencies of dental caries. The differences in caries frequencies might be the result of dietary differences; for example, higher caries frequencies are observed among individuals with diets containing more refined carbohydrates or less abrasive foods (i.e., some studies have shown that dental attrition has a negative relationship with caries) (Moore and Corbett, 1973; Maat and Van der Velde, 1987; Kerr et al., 1990; Whittaker and Molleson, 1996; Vodanovic et al., 2005; Esclassan et al., 2009). Differences in caries frequencies might also reflect differences in oral hygiene (Whittaker and Molleson, 1996; Phillips, 2006), differences in susceptibility to caries (Kerr et al., 1990), or differences in fluoride levels in the local water (Roberts and Cox, 2003). It is possible that the variation in caries might at least partly reflect sampling issues rather than real differences within past populations. For example, the observed variation might be the result of differences in the age distributions of the samples studied given that dental caries is an age-progressive process (i.e., samples with a greater proportion of older adults might have higher frequencies of dental caries) (Kerr et al., 1990; Sledzik and Moore-Jansen, 1991; Sutter, 1995; Saunders et al., 1997). Variations in the sex composition of samples might also affect the observed frequencies of caries; several studies have found higher caries rates in females when controlling for age (Whittaker and Molleson, 1996;

TABLE 2. *Maximum likelihood estimates of $\hat{k}_2$ with standard errors (s.e.) and likelihood ratio tests of $H_0:\hat{k}_2 = 1$*

Pathology	$\hat{k}_2$ (s.e.)	LRT
Periodontitis: mandible	1.2 (0.3)	0.6
Periodontitis: mandible	1.3 (0.3)	0.5
Dental caries: premolars	1.2 (0.3)	7.9*
Dental caries: molars	2.05 (0.7)	9.8*

* Significant at $\alpha = 0.05$.

Saunders et al., 1997; Lukacs, 2008; Wasterlain et al., 2009).

As shown in Table 1, the frequencies of periodontitis within St Mary Graces range from 37 to 43%. These frequencies are higher than those observed by Clarke and colleagues in various samples from premodern populations (Clarke et al., 1986; Clarke and Hirsch, 1991); in those samples, frequencies of periodontitis ranged from 8 to 11%. Clarke et al. (1986) argue that diagnosing periodontitis based on distances between the CEJ and AC greater than 2 mm will overestimate the frequency of the pathology (for reasons provided above), and so they recommend only scoring oral lesions that are more likely to be specific to periodontitis. The higher frequencies of periodontitis observed in the St Mary Graces sample might suggest that the method for detecting periodontitis used for this study is capturing individuals who did not actually have periodontal disease. However, the frequencies of periodontitis observed in this study are lower than those observed in a roughly contemporaneous sample from medieval Scotland. Kerr (1991) scored bone destruction on the interdental septa, a method he argues is more discriminating than using distances between the CEJ and AC. He found that in a sample from medieval Scotland (900–1600 AD), 72% of individuals over the age of 16 years and 91% of individuals over the age of 26 years had signs of destructive periodontitis. These results are similar to those found in a later English sample; in a predominant eighteenth-century skeletal sample from London, Kerr (1994) found that the frequency of periodontitis was 73% and higher for individuals over the age of 16 years and that all individuals above the age of 35 in the sample had periodontitis. The frequencies observed in St Mary Graces are also lower than those observed by Chazel et al. (2005) in a sample from medieval France; more than 60% of the French sample had periapical inflammation indicative of periodontitis. The variation in observed frequencies of periodontitis might result from variation in methods for identifying the pathology, differences in the age composition of the samples examined (i.e. as with caries, the frequency of periodontitis increases with age), variation in oral hygiene (Kerr, 1991, 1994; Oztunc et al., 2006), or some other factors.

As described above, the St Mary Graces cemetery provides a sample of the general population of London from the mid-fourteenth century until the Reformation in the sixteenth century. At this time, London was the largest city in Britain and one of the largest cities in Europe (Rappaport, 1989; Harding, 2002). During the fifteenth and sixteenth centuries, London experienced population growth as a result of immigration into the city. Without people moving into London, the population actually would have decreased given that mortality in the city exceeded the birth rate (Rappaport, 1989). The fourteenth-century Black Death reduced the population of London to below 50,000 people, but by the end of the sixteenth century, the population had tripled in size (Rappaport, 1989). The thousands of people living within an area not much larger than a square mile created conditions that were deleterious to health (Roberts and Cox, 2003), and according to Rappaport (1989), mortality levels were higher in London than in other parts of England by the sixteenth century. However, this period in London's history was also one of relatively high standards of living. Because of the devastating mortality of the fourteenth-century Black Death, the demand for labor in England exceeded the supply, and wages therefore increased; there was also a decrease in the price of food and rent following the epidemic (Rappaport, 1989; Dyer, 2002; Stone, 2006; Woolgar, 2006).

Despite rising standards of living, there were differences in the diets of lower and higher status individuals in London (Moore and Corbett, 1973; Dyer, 1983), all of whom are represented in the St Mary Graces cemetery. For individuals of all status levels, grains (primarily wheat, rye, and barley) provided the largest component of the diet and were consumed in the forms of bread, ale, and pottage. Before the Black Death, wealthy individuals in England consumed higher quality breads made from fine wheat flour while poorer individuals were generally limited to lower quality, coarser breads (Moore and Corbett, 1973; Stone, 2006). However, following the Black Death, because of lower grain prices, individuals of all status levels consumed greater quantities of higher quality wheat bread (Stone, 2006). Following the Black Death, there was also an increase in the amount of meat and fish in the diet, though higher status individuals consumed a greater amount and variety of both (Sykes, 2006; Woolgar, 2006). Fruits, vegetables, and dairy products were consumed by all classes (Dyer, 1983; Woolgar, 2006). Wealthy individuals ate more imported luxuries such as honey, dried fruits, spices, wine, and cane sugar (Moore and Corbett, 1973; Dyer, 1983). Because they could afford such luxuries, wealthy individuals had more sucrose in their diets, but they did not consume large quantities of it (Moore and Corbett, 1973; Whittaker and Molleson, 1996; Roberts and Cox, 2003). The late medieval English diet, regardless of status, was thus high in carbohydrates and protein, but generally did not contain the large quantities of refined carbohydrates seen in more recent populations (Moore and Corbett, 1975).

It is perhaps not surprising to find moderate frequencies of oral pathologies in a medieval English sample given that oral hygiene and dental care in medieval Europe were rudimentary at best (Newman, 2001; Anderson, 2004). According to Anderson (2004), medieval medical texts indicate that dental care in England was primarily based on herbal remedies, amulets, and charms, and though surgeries were performed occasionally to treat oral cancers and wounds, dental care was mostly noninvasive. Dental caries were thought to be caused by "toothworms" and methods believed to kill the worms, such as treatment with acid or covering the affected tooth with wax, were sometimes used (Roberts and Cox, 2003). Individuals with toothaches had the offending teeth removed, and some people rinsed their mouths with vinegar and cleaned their teeth and gums with clothes or soft sticks, but methods for removing plaque and filling caries were not widely practiced (Newman, 2001; Roberts and Cox, 2003; Anderson, 2004).

The results shown in Table 2 suggest that the oral pathologies analyzed here are associated with frailty in

the St Mary Graces sample such that individuals with periodontitis and premolar and molar dental caries were at elevated risks of death compared to their peers without such pathologies. If dental caries and periodontitis really are indicative of frailty in the St Mary Graces medieval population, the question remains whether the relationship between oral health and systemic health in past populations was similar to the relationship existing between the two in modern populations. As described above, studies of oral health in modern populations have suggested that periodontitis and dental caries are associated with diseases elsewhere in the body, and these associations might occur because of the spread of infection from the mouth to other areas of the body, chronic inflammation initiated by an oral pathology, an underlying problem with the immune system, or some other common risk factor (or perhaps some combination thereof). It is possible that periodontitis and dental caries were associated with other systemic health problems in past populations because of these same mechanisms.

Both periodontitis and dental caries might have elevated the risks of mortality within the St Mary Graces population by directly causing disease elsewhere in the body. For example, in some individuals, infections might have spread from the localized infections in the mouth to other tissues of the body and thereby caused infections that ultimately resulted in death, as apparently occurs in some cases of bacterial pneumonia. Periodontitis or dental caries might also have caused chronic systemic infection or inflammation, which subsequently influenced the development or severity of other life-threatening diseases within St Mary Graces.

The oral pathologies might reflect preexisting high frailty among individuals in St Mary Graces. As has been suggested with respect to the periodontitis and dental caries in modern populations, perhaps the oral pathologies did not directly cause systemic infection or inflammation in individuals in the St Mary Graces population, but rather they reflect inadequate immune functioning or deleteriously exaggerated immune responses, which increased an individual's risk of developing oral pathologies and a variety of other diseases that ultimately caused death.

Compromised immune functioning in the St Mary Graces population could have been the result of suboptimal nutritional status. According to Dyer (1983), typical diets in medieval England, even of higher status individuals, would have been deficient nutritionally given their minimal consumption of dairy products and fresh fruits and vegetables. As described above, some studies of modern populations have found that dental caries is more common among underweight and obese children compared to children of normal weight; that is, dental caries is possibly associated with malnutrition in modern populations. The pain of severe caries might interfere with the ability to eat and thereby contribute to abnormally low weight, and obesity can be the result of a diet composed of an abundance of highly cariogenic foods with low nutritional value. In both cases, malnutrition might result in lowered immune competence. Obesity might not have been a significant problem for the St Mary Graces population, as some argue that obesity was not highly prevalent in populations until the twentieth century (Ulijaszek and Lofink, 2006). However, perhaps in the St Mary Graces population, individuals with painful dental caries were consequently undernourished and therefore suffered reduced immune competence and were more highly susceptible to various causes of morbidity and mortality.

It is also possible that dental caries in the St Mary Graces population were caused by, rather than contributed to, poor immune functioning, as it has been suggested that in modern populations, the development of caries might be the result of the immune system's inability to control the proliferation of cariogenic oral bacteria (Acs et al., 1999). Similarly, periodontitis in St Mary Graces might reflect preexisting diseases or genetic factors, which affected immune function. Studies have shown that in individuals whose immune systems become compromised by such factors as HIV infection or chemotherapy for treatment of cancer, the severity of periodontitis increases (Beck et al., 1996). Beck et al. (1996), who study the association between periodontitis and cardiovascular disease, have hypothesized the existence of individuals with a hyperinflammatory trait who therefore exhibit abnormally high inflammatory responses to pathogens and are thus at high risk of developing both periodontal disease and cardiovascular disease. Watts et al. (2008) suggest that genetic polymorphisms for genes involved in the inflammatory process might explain the link between periodontitis and Alzheimer's disease. If, as these and other studies suggest, it is reasonable to suspect that the associations between oral health and systemic diseases in modern populations are at least partly determined by variation in genes influencing the immune system or by infection with diseases that affect immune response, such mechanisms could also have been at work in past populations. In the St Mary Graces population, there might have been some individuals who were genetically predisposed to developing dental caries, periodontitis and other diseases or who had immune systems weakened by disease and were thus more highly susceptible to oral pathologies and other causes of morbidity and mortality.

As described above, periodontitis in modern populations is associated with elevated risks of mortality and is a risk factor for several diseases, including cardiovascular disease, certain types of cancer, pulmonary infection, and diabetes. Given that the results of this study suggest that periodontitis was also associated with elevated risks of death in past populations, periodontitis might also have been associated with other systemic diseases in the past. This study does not address specifically what systemic diseases periodontitis might have been associated with in the St Mary Graces population. It is not necessarily the case that in past populations, periodontitis was associated with the same systemic diseases to which it is linked in modern populations. In fact, given that many of the diseases associated with periodontitis today are degenerative diseases such as cardiovascular disease and cancer, it is perhaps more likely that periodontitis was *not* associated with the same suite of diseases as is observed today. There is evidence that in industrialized nations, such as England, the prevalence of degenerative diseases has increased over the last few centuries such that degenerative diseases have replaced infectious diseases as the most common causes of death (Omran, 1977; Gage, 2005). This would suggest that within the St Mary Graces population of the fourteenth to sixteenth centuries, if periodontitis was in fact associated with systemic diseases, those diseases would have been predominantly infectious rather than degenerative diseases. For anthropologists interested in general

health patterns in past populations, the actual causes of morbidity and mortality with which periodontitis was associated in the past might not be as important as the apparent association between the oral pathology and elevated risk of death in the St Mary Graces population shown here. These results suggest that, regardless of the ultimate cause of death, periodontal disease is associated with increased risks of mortality and thus is potentially informative about general health in past populations. Health indices, such as that proposed by Steckel and Rose (2002), which incorporates information on length of life and the presence of certain skeletal pathologies to assess levels of health in past populations (and already includes dental caries), might benefit from the inclusion of periodontitis.

As described above, one of the criteria used in this study to identify periodontitis was the distance between the CEJ and the AC that was greater than 2 mm, a criterion that might not always indicate periodontitis. The other criterion used in this study to diagnose periodontitis, the exposure of cancellous bone as a result of the destruction of overlying cortical bone (i.e., porous alveolar bone), is one that is recommended by numerous researchers who criticize the "greater than 2 mm" criterion (Clarke et al., 1986; Clarke and Hirsch, 1991). So we are confident that the periodontitis sample used in this study does include individuals who suffered from the oral pathology. However, nonpathological processes, such as continued eruption of teeth to compensate for attrition, can result in an increase in the CEJ–AC distance beyond 2 mm (Costa, 1982; Clarke et al., 1986; Clarke, 1990; Clarke and Hirsch, 1991; Hildebolt and Molnar, 1991; Varrela et al., 1995; Hillson, 1996). It is possible that individuals with a CEJ–AC distance greater than 2 mm caused by nonpathological processes were incorrectly diagnosed with periodontitis for this study. This is potentially a problem for this study because the inclusion of such false positives for periodontitis in the sample would result in an underestimation of the risk of death associated with periodontitis. It is therefore reassuring that an elevated risk of death with the periodontitis (i.e., a k_2 estimate of greater than 1) was detected despite the possible inclusion of false positives in the sample. This result suggests that periodontitis is associated with elevated risks of mortality. The possible inclusion of false positives suggests that the true risk of mortality associated with periodontitis might have been even higher than that suggested by the results of this study. In light of the possible inclusion of false positives in the sample, in addition to the errors associated with age estimation mentioned above, the estimated value of the k_2 parameter for periodontitis shown here should not be taken at face value, but rather should be interpreted as a general indicator of an elevated risk of mortality.

CONCLUSION

The results of these analyses suggest that periodontitis and dental caries were associated with increased risks of death in the St Mary Graces population. These pathologies are associated with systemic health problems in modern populations. However, no attempt is made here to determine which particular diseases or causes of death periodontitis or dental caries were associated with in the St Mary Graces population. Future work might fruitfully examine the skeletal pathologies that are associated with periodontitis and dental caries within St Mary Graces or other skeletal samples to address this particular question. The results of this study suggest that periodontitis and dental caries can be used as informative indicators of general health in past populations.

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