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RESEARCH ARTICLE

History of Health at Cayo Santiago—An Investigation of Environmental and Genetic Influences on the Skeletal Remains of the Introduced Rhesus Macaque (*Macaca mulatta*) Colony

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ABSTRACT

The Cayo Santiago rhesus macaque colony is a renowned primate population that has experienced significant natural and anthropogenic ecological variation in their 85-year history. Demographic and familial information is also tracked and collated for the majority of monkeys. Thus, the health history of rhesus macaques at Cayo Santiago should reflect the impacts of both environmental and genetic factors. In this study, we utilized a sample of skeletal remains comprised of 2787 individuals (1571 females, 1091 males), born between 1938 and 2017 from the derived skeletal collection of the primate colony to assess variation in survivorship, pathology, bone mineral density (BMD), and dental eruption status, in the context of hurricane impacts, nutritional fluctuations, and matriline genealogy. Results demonstrated that rhesus macaques at Cayo Santiago exhibit a range of skeletal pathologies that encompass biomedical and archaeological significance, multiple etiologies, severities, locations, and types, in addition to a secular trend of declining BMD that is hypothesized to reflect decreasing physical activity levels under increasing population densities. Specifically, hurricane impacts were found to increase the rate of systemic disease, decrease BMD in young adults, and delay eruption of the primary dentition. Certain matrilines exhibited heightened levels of systemic disease at early ages while others exhibited greater rates of congenital disease. Early-life adversity, through the experience of major hurricanes, may enhance inflammatory pathways, heightening the risk of disease and accelerating the aging process leading to reduced BMD. Such impacts may underly greater levels of observed infection post-hurricane through intensification of pathogen transmission and disease rates brought on by hurricane-adaptive social strategies that favor closer proximity. Familial susceptibility to disease indicates heritable host genetic factors are likely influencing disease patterning in the population. A cluster of congenital diseases may most convincingly illustrate this, or alternatively reflects low levels of genetic diversity in the population.

1 | Introduction

The Cayo Santiago rhesus macaque (*Macaca mulatta*) population has been used extensively across multiple research disciplines over the course of their 85-year history, after being introduced to the Caribbean area in 1938 (Rawlins and Kessler 1986; Wang 2012a; Kessler and Rawlins 2016; Wang and Francis 2024 this issue). Their island habitat has created a "natural laboratory" where wild-like behaviors are able to develop within a manageable setting. A daily census tracking

Summary

- A secular trend of declining BMD and osteoarthritis corroborates with decreasing physical activity levels under increasing population densities.
- Familial susceptibility to disease indicates heritable host genetic factors are likely influencing disease patterning in the population.
- Early life hurricane experiences negatively affect growth, development, and health.

individual monkey identity, sex, age, date of birth, and matriline genealogy provides immediate demographic context to any and all population-based studies. Investigation of population biology in this context is available through their derived skeletal collection, now totaling over 3000 individuals, mostly with known sex and age at death. Numerous investigators have made use of the opportunity to study the unique Cayo Santiago population because they are well contextualized with demographic information and carry broad translational potential for both human and other nonhuman primate populations. However, many such studies employing the skeletal collection have focused on only a single aspect of skeletal health or morphology, meaning the full potential of the Cayo Santiago colony has yet to be realized. A comprehensive outlook of the history of health of the introduced rhesus colony that integrates multiple parameters of population skeletal health across family, demography, and management practices, in addition to environmental factors such as acclimation and climate natural disasters (i.e., hurricanes), has not yet been constructed, and is thus the main purpose of this study. Leveraging this integration of information, this study also attempts to unearth genetic and environmental contributors to skeletal health variation within the population to establish a model for human and nonhuman primate resilience and fragility.

Rhesus macaques have a skeletal system similar to humans, exhibiting bone diseases, anomalies, and age-related deteriorations that recapitulate those found in humans (Chiou et al. 2020; Colman 2018; Li et al. 2018), rendering them a useful model for studying human conditions (Grynpas et al. 1989; Rothschild, Hong, and Turnquist 1997; Rothschild, Hong, and Turnquist 1999; Cerroni et al. 2000; Wang, Turnquist, and Kessler 2016b; Ebersole et al. 2019). Similar physiologies and patterns of senescence to humans have yielded a significant body of research into the modeling of ageing in *M. mulatta* (Chiou et al. 2020). Individuals of the Cayo Santiago population have been found to mirror the progression of bone mineral density (BMD) in humans, increasing from infancy, peaking in adulthood, and declining with advancing age (Cerroni et al. 2000). Although, male rhesus macaques attain their peak BMD at an earlier age than females in a contrasting trend to humans (Cerroni et al. 2000). The declining BMD phase can become pathological and lead to osteopenia and eventually osteoporosis. Female rhesus macaques exhibit higher rates of fracture and osteoporosis than males, partly due decreasing estrogen levels associated with menopause, although parity has been found to mitigate the effects of BMD loss through protection of trabecular bone thickness (Turnquist et al. 2012), a phenomenon also found in humans (Cerroni et al. 2003). Cerroni et al's (2000; 2003) investigations employed the clinical definition of osteoporosis, with those with BMD values 2.5 standard deviations below an average reference sample, a subset of the Cayo population, deemed osteoporotic. Further studies have tested BMD with variable diets in the colony (Grynpas et al. 1993). Nutritional content, such as calcium, vitamin D and protein intake, has been directly linked to bone health (Mangano,Sahni, and Kerstetter 2014). From 1956 to 1969, the Cayo population was fed Purina Monkey Chow with a 15% protein content and increased to 25%, post-1969. No change in BMD was found between individuals fed either diet (Grynpas et al. 1993). However, it remains possible that the disputed link between differences in body weight may be obscuring a possible link with BMD (DeRousseau 1985; Grynpas et al. 1989; 1993).

Along with osteoporosis, other age-related diseases are well-represented in the Cayo Santiago population. Osteoarthritis and inflammatory arthritis were found in 26% and 20% of individuals over 8 years of age, respectively. (Rothschild, Hong, and Turnquist 1997; Rothschild, Hong, and Turnquist 1999; Rothschild 2005). The latter disease was found to vary by social group which are themselves divided along matrilineal genealogies. Periodontitis and odontogenic abscesses have also been found to increase with age, while both diseases along with linear enamel hypoplasia (LEH), have been found to vary between matrilines, highlighting a potential genetic predisposition and/or maternal effect in certain families (Gonzalez et al. 2016; Ebersole et al. 2019; Li et al. 2018; Guatelli-Steinberg et al. 2023). Odontogenic abscesses and LEH have additionally been found to decrease over the course of the colonies inhabitation of Cayo Santiago, purportedly as a result of the introduction of a provisioned diet in 1956 (Li et al. 2018; Benderlioglu and Guatelli-Steinberg 2019), as have greater long bone lengths been reported since the switch to a higher-protein diet in 1969 (DeRousseau and Reichs 1987). Beyond the most pervasive and destructive pathologies, the Cayo Santiago skeletal collection features important congenital defects (Rawlins and Kessler 1983), non-metric features (Cheverud and Buikstra 1981a, 1981b, 1982; Richtsmeier, Cheverud, and Buikstra 1984; Wang et al. 2006a; Francis and Wang 2024; Guatelli-Steinberg et al. 2023b), and a range of other dental abnormalities including antemortem tooth loss (AMTL), caries (Wang, Turnquist, and Kessler 2016b), and tooth wear (Guatelli-Steinberg et al. 2022). Many of the aforementioned pathologies are patterned by sex as well as age and matriline, with males broadly exhibiting greater levels of disease (Wang, Turnquist, and Kessler 2016a; Li et al. 2018; Guatelli-Steinberg et al. 2022). This may reflect diverging lifeways in regard to sexual and/or resource competition (Wang, Turnquist, and Kessler 2016b), or could be representative of reproductive patterns that lead to relatively stable body masses for females in later life stages, when many such diseases will manifest, compared to the physical wasting observed in males during this time (Turcotte et al. 2022).

Since their introduction to the island, four major hurricanes classified as Category 3 or higher have made impact at Cayo Santiago: Hurricane Hugo (Sept 18, 1989), Hurricane George (Sept 21, 1998), Hurricane Irma (Sept 06, 2017), and Hurricane

Maria (Sept 20, 2017). The documented consequences of extreme weather events are vast and varied. Along with death, destruction, and physical disease, more subtle impacts, such as those on mental health, ental have been reported in humans that have experienced such events (Schwartz et al. 2017; Scaramutti et al. 2019). In nonhuman primates, mortality patterns have reportedly increased, as have changes in regular diet and behavioral practices been reported (Behie and Pavelka 2005; Schaffner et al. 2012; Milton and Giacalone 2014). However, he long-term impact of hurricanes on the health of nonhuman primates has not been extensively studied. Some of the most notable research has so far been conducted on the Cayo Santiago macaques—detailing trends of decreased fertility (Morcillo et al. 2020), heightened developmental instability (Romero et al. 2023), delayed reproductive debut (Luevano et al. 2022), increased sociality (Testard et al. 2021), and accelerated immunological aging (Watowich et al. 2022) post-hurricane impact. Yet few have studied the direct influence on skeletal health which is possible to gauge from the collection materials.

A vast skeletal collection contextualized with known demographic data, spanning dozens of family genealogies and key environmental events, provides a rare opportunity with which to extract potential genetic and environmental drivers of overall bone health. Through pathological examination, BMD measurements, and dental development scoring, this study investigates the effects of family, hurricane impact, and nutrition within the Cayo Santiago population. We hypothesize (umbrella hypothesis) that pathology rates, BMD, and dental eruption timings will vary across families (Hypothesis 1), hurricane impact (Hypothesis 2), and nutritional status (Hypothesis 3). We specifically predict that certain matrilines, such as #065, (Rothschild, Hong, and Turnquist 1997; Rothschild, Hong, and Turnquist 1999; Gonzalez et al. 2016; Zhdanova et al. 2016; Li et al. 2018; Ebersole et al. 2019; Guatelli-Steinberg et al. 2023), those in the hurricane-exposed group (Watowich et al. 2022; Romero et al. 2023; Motes-Rodrigo et al. 2023), and pre-protein group (DeRousseau and Reichs 1987; Guatelli-Steinberg and Benderlioglu 2006) will exhibit greater levels of pathology as well as reduced levels of peak BMD in accordance with BMD's tight relationship in humans with inheritance (Beamer et al. 1996; Ralston and de Crombrugghe 2006), early-life adversity (Wuertz-Kozak et al. 2020), and nutritional status (Rizzoli and Bonjour 1999; Ralston and de Crombrugghe 2006). We further predict that dental eruption will be delayed in the hurricane-exposed group and accelerated in the protein-group, given rates of eruption between captive and wild settings (Phillips-Conroy and Jolly 1988; Zihlman, Bolter, and Boesch 2004; Zihlman, Bolter, and Boesch 2007; Wang, Turnquist, and Kessler 2016b). We further take the opportunity to investigate the composition of skeletal pathology in terms of overall prevalence, age-associated prevalence, and sex prevalence to characterize the patterns of skeletal pathologies and assess their variations over time and between different matriline families, within an environmental and genetic context.

2 | Materials and Methods

The Cayo Santiago population has been home to a colony of rhesus macaques since 1938, after 409 monkeys were translocated from northern India by Dr. Clarence Carpenter. The island of Cayo Santiago lies approximately a kilometer off the east coast of Puerto Rico. The colony are provisioned with monkey chow twice daily and have been so since 1956. In 1969, this diet was switched from a 15% protein component to 25% (DeRousseau and Reichs 1987). Population densities are high on the island (1500/15 hectares) (Newman et al. 2023) compared to forested (6/15 ha) and urban (30/15 ha) rhesus macaque groups (Fooden 2000). A number of strategies to reduce population density have taken place over the years (Hernandez-Pacheco et al. 2016), though numbers have increased from approximately 400 in 1938 to 1700 monkeys in recent years (Kanthaswamy et al. 2017). Between these dates, in the mid-1950s, the population was reduced to around 70 individuals (Kanthaswamy et al. 2017; McMillan and Duggleby 1981). As no individuals have been added to the island since, it may have produced a population bottleneck. Accordingly, circa 90% of the living residents at Cayo Santiago are descended from 15 founder females (Kanthaswamy et al. 2017). Despite this, minimal evidence of inbreeding has been uncovered in the Cayo population (Widdig et al. 2001; 2017). Documented maternal genealogies now go back over 11 generations, of which many are stored in Cayo Santiago's associated skeletal collections. The majority of skeletal material employed in this study derives from the Laboratory of Primate Morphology in the Caribbean Primate Research Center (CPRC), housed at the University of Puerto Rico Medical Campus. The sample also includes Cayo derived skeletal materials housed at the CPRC-NYU skeletal collection in the Department of Anthropology, New York University.

Between the two collections, 2787 individuals (1517 females, 1091 males) were examined (see Supporting Information S1: Table S1 for collection specimen count). All were assessed for pathology, while BMD was measured for 1249 individuals (711 females, 538 males), and dental eruption status for 1346 individuals (749 females, 585 males) (Table 1). The discrepancies in data type counts reflect the constraints of time against the duration of type collection, with a priority on individual skeletons with known demographic data including sex, age, and mother's identity. Pathology was assessed macroscopically across the entire skeleton when available. The selection of the pathologies investigated are borne out of (1) common osteological lesions within bioarchaeological studies (DeWitte and Bekvalac 2011; Mann and Hunt 2013; Villotte and Knüsel 2013; Samsel, Kacki, and Villotte 2014; Ventades et al. 2018; Buikstra 2019; Steckel et al. 2019; Wissler and DeWitte 2023), (2) studies conducted directly on the Cayo Santiago macaque population (Rothschild, Hong, and Turnquist Hong, and Turnquist 1997; 1999; Wang, Turnquist, and Kessler 2016b), (3) novel features noted during examination of the skeletal material for this study. Dental eruption was also scored for each tooth on a 0–4 scale following (Wang 2012b). BMD measurements were taken of the humerus and femur using a BeamMed Sunlight Ultrasound Bone Sonometer. These bones were used as their diaphysis provided sufficient area to take a reading from the ultrasound probe. A description of the protocol and error estimation of BMD recordings are contained within the Supporting Information and Supporting Information S1: Table S2. Dental eruption was scored for each tooth on a 0–4 scale following Wang (2012b).

TABLE 1 | Sample number breakdown for analyses of family (matriline), hurricane-exposure, and nutritional status.

	Total	M	F	UNK/Other
Sample	2787	1091	1571	125
Family	1912	794	1106	12
Family-BMD	1150	488	622	40
Hurricane	1632	634	986	12
Hurricane—Yes	484	180	304	0
Hurricane—No	1148	454	682	12
Hurricane-BMD—Yes	275	105	170	0
Hurricane-BMD—No	537	229	308	0
Hurricane-Dental—Yes	293	114	179	0
Hurricane-Dental—No	644	282	361	0
Nutrition	1538	615	755	168
Nutrition—'0'	59	15	40	4
Nutrition—'1'	282	142	133	7
Nutrition—'2'	1197	458	582	157

Note: 'UNK/Other' refers to unrecorded sex in demographic database, mixed sex, or castrated individuals. 'Family' refers to those that $N < 30$. 'BMD' stands for bone mineral density. 'Hurricane Yes' refers to the hurricane exposed group and 'Hurricane No' as the non-exposed group. Numbers following 'Nutrition' refer to their dietary status, with '0' = pre-provisioned, '1' = pre-protein group, and '2' = protein provisioned.

2.1 | Skeletal Pathology Protocol

The primary aim of this manuscript is to investigate the impact of matriline, hurricane experience, and nutritional quality on the overall health status of individuals. A major component of data used to link the skeletal material to health status is through interpretation of the patterns of skeletal pathology over time and between matrilines. Some common lesions found on the skeleton exhibit disputed etiologies and, therefore, differing outcomes for what they mean in terms of health. Others are closely linked with age and, therefore, cannot be interpreted as necessarily pathological without a known age-pathology context. While others may exhibit juxtaposing health/pathology outcomes depending on their severity and/or whether they were active at the time of death. In light of these predicaments, we record pathologies on a binary 'yes/no' basis, yet, where feasible, divide them into subcategories that include 'severity', 'activity status', and 'type'. Pathologies were also broken down into age groups to make them more comparable between families, nutritional groups, and hurricane events. Together, our categorization and subsequent interpretation of skeletal pathology data attempt to exercise caution considering the potential pitfalls in non-critically linking skeletal pathology lesions to overall health status. The list of pathologies and how and why they were collected are listed in Supporting Information S1: Table S3 and depicted in Supporting Information S1: Figures S1–S18.

2.2 | Pathology

Counts and percentages of pathologies within the sample were reported along with sex differences. Fishers Exact Tests were employed to test for differences in overall pathology frequency between sexes with $\alpha = 0.05/57 = 0.00087$. However, all statistical tests in the manuscript that yielded a p-value ≤ 0.05 were treated as potentially biologically meaningful given the scale of analysis coupled with Bonferroni corrections tendency to be overly conservative (Bender and Lange 2001). Conversely, the varying sample power of pairwise comparisons conducted within this study, and the importance of their cautious interpretation regarding health outcomes, also warranted a safeguard against Type 1 errors. All statistical analysis within this study was conducted in R Studio (R Studio Team 2020). Relative risk (RR) ratios were conducted in R Studio to test the association of pathologies with one another. The α level was set at $0.05/(56 \times 56) = 0.0000159$ (degrees of freedom = 3135) for each analysis of pooled-sex, males only, and females only. To assess pathology frequency across lifespan stages, age was coerced into intervals every 4 years starting at 0 years of age. A percentage table and bar graphs of main pathologies, made in ggplot2 (Wickham, Chang, and Wickham 2016), was used to visualize relationships between age groups and pathology frequency per group. RR ratios were also conducted across 4-year age intervals up to 28 years for each pathology, as described above. The α level was set at $0.05/(56 \times 56 \times 7) = 0.0000028$ (df = 21951) for pathology RR ratios across ages. To test for differences of survivorship between the most well represented pathologies (list in SI), Kaplan–Meier Survival Analysis was conducted. Included in this analysis are individuals exhibiting none of the concurrent pathologies, or 'no lesions', and the population average ('Pop. Total') of survivorship for visual comparison. Cox Proportional Hazard Ratios were conducted to test for pairwise differences between the average ages of pathologies, with $\alpha = 0.05$ and after Bonferroni correction $\alpha = 0.05/105 = 0.00047$ (df = 104). The same set of analysis was run for subtypes of these main pathologies, including Kaplan–Meier Survival Analysis ($\alpha = 0.05$) and pairwise Cox Proportional hazard ratios ($\alpha = 0.05/68 = 0.000732$, df = 67). Those born into social group K were removed for survival analysis as they were killed in 1972 to start up the skeletal collection (Buikstra 1975). Likewise, those derived from the CPRC-NYU collection were not included in any aspect of survival analysis.

2.3 | Family (Matriline)

To test for the effects of family on skeletal health in the Cayo Santiago population, only matrilines with $N > 30$ were included in family analyses (list in SI). This totaled 1912 individuals (1106 females, 794 males and 11 unknown or other) across 16 matrilines ranging in size from 31 to 250 individuals (Table 1). A Kruskal–Wallis test was used to assess significant age differences between families for pooled-sex, male-only, and female-only (df = 15). Fishers Exact Tests were employed to test for differences in overall pathology frequency between families. This was undertaken for pooled-sex, male-only, and female-only groups, and for pooled-sex across 4-year age intervals, with $\alpha = 0.05/57 = 0.00087$ (df = 56) for each. For survivorship comparisons, Kaplan–Meier Survival Analysis was conducted between matrilines with $N > 30$ ($\alpha = 0.05$) for pooled-sex, male-only, and female-only groups. Survival Risk tables were further constructed to visualize heterogeneity in survivorship across the lifespan for families pooled by sex, male-only, and female-only groups. For pathology comparisons, all 1912 individuals were used. A 'peak' BMD was generated for viable comparisons across variables where age-bias would attempt to be mitigated. Peak BMD was calculated as the 95% range around the maximum BMD age for each bone for males and females. The maximum was calculated using 'loess' smoothing method in R and the 'predict' function across average BMD levels to ascertain the maximum BMD age (Turcotte et al. 2022). Five percent of the overall BMD increase from age 0 to maximum BMD age was calculated then subtracted from the maximum value to provide us with the 95% range, and the ages this encapsulated, to be used as peak BMD. Across families, peak BMD was assessed for both the femur and humerus and each sex separately, using Kruskal–Wallis tests ($\alpha = 0.05$). Matrilines of $N > 20$ were included for analysis. For males this included 14 matrilines (df = 13) and 488 individuals, and for females this included 14 matrilines (df = 13) and 662 individuals. The imbalance of females over males in the sample reflects strategies to control the population by removing males (Hernandez-Pacheco et al. 2016). All subsequent BMD analysis was similarly separated by sex and bone type.

2.4 | Hurricanes

To test for the effects of the impact of major hurricanes in the Cayo Santiago population, the sample was split into a hurricane-exposed group ($N = 483$) and a non-exposed group ($N = 1150$) (Table 1). Individuals were classified in the exposed group if they were in-utero to < 4 years of age by the time one of the three major hurricanes made impact following the dates described in the introduction. This follows similar age ranges for adversity measures in the Amboseli baboons (Levy et al. 2023) and is similar to studies investigating hurricane effects at Cayo (Gonzalez, Sherer, and Hernández-Pacheco 2023; Romero et al. 2023). Comparisons between hurricane-exposed and non-exposed groups were limited to those born after 1981-01-01 to mitigate any confounding effects of nutritional changes, which are largely confined to the populations earlier years. A Wilcoxon test was used to test for significant age differences between the hurricane-exposed and non-exposed group. For pathology comparisons all 1632 individuals were used (986 females, 634 males, and 12 unknown sex), for BMD comparisons 812 individuals (478 females, 334 males) were used, and for dental eruption 937 individuals (540 females, 396 males, 1 unknown) (Table 1). Fishers Exact Tests were employed to test for differences of pathology frequency between the hurricane-exposed and non-exposed group and at each 4-year age interval as described above ($\alpha = 0.05/57 = 0.00087$ for each group and age interval). For each hurricane event, skeletal pathologies were also broken down into count and percentages to explore the relative contribution and effect of each hurricane on the population's skeletal health. Wilcoxon-tests were used to test for significant differences of peak BMD between hurricane-exposed and non-exposed groups ($\alpha = 0.05/4 = 0.0125$, df = 3) and at age categories ranging from 0–5, 5–10, 10–15, and 15+ ($\alpha = 0.05/16 = 0.0031$, df = 15). These age categories broadly correspond to life stages of rhesus macaques: "young", "young adults", "prime adults", and "aged adults" in rhesus macaques (Bernstein et al. 1991; Bercovitch 1997; Widdig et al. 2001). To test for differences in the timing of dental eruption between hurricane-exposed and non-exposed groups, median averages were compiled of each tooth in the occlusal plane (scoring '4'). The sample was limited to those below 8 years of age to coerce hurricane groups into similar age ranges and to not skew eruption averages. Cox Proportional Hazard Ratios were conducted for median age of occlusal eruption for all primary (5) and permanent (8) teeth between hurricane groups, with $\alpha = 0.05$ (df = 104).

2.5 | Nutrition

To test for the effects of nutrition, individuals in the Cayo Santiago colony were divided according to diet experienced at birth (Table 1). Those born before 1957 were the pre-provisioned group, or Group '0', between 1957 and 1970 were the pre-protein group, or Group '1', and after 1970 were the protein group, or Group '2'. Analyses for nutrition was limited to those born before 1986-01-01 to limit the potentially confounding effects of hurricane impacts. Those making up the analyses of the effects of nutrition totaled 1538 individuals, 59 in Group '0', 282 Group '1', and 1197 in Group '2'. Due to the limited sample size of the pre-provisioned group, all major analysis was between Groups '1' and '2', with '0' results used as an interpretative aid where applicable. A Kruskal–Wallis test ($\alpha = 0.05$, df = 2) and Wilcoxon post hoc tests ($\alpha = 0.05/16 = 0.0031$, df = 15) were used to test for age differences between nutritional groups. For pathology comparisons, 1538 individuals were used (755 females, 615 males, and 168 unknown or other), for BMD comparisons 702 individuals were used (375 females, 257 males), and for dental eruption 608 individuals (319 females, 280 males, and 8 unknown or other) were used (Table 1). Fishers Exact Tests were employed to test for differences of pathology frequency between the nutritional groups ($\alpha = 0.05/57 = 0.00087$). Wilcoxon-tests were used to test for significant BMD differences between nutritional groups for peak BMD levels ($\alpha = 0.05/4 = 0.0125$, df = 3), and at age categories ranging from 0–5, 5–10, 10–15, and 15+ ($\alpha = 0.05/16 = 0.0031$, df = 15.). To test for differences in the timing of dental eruption between nutrition Groups '1' and '2', the same methodology as described for hurricane group dental comparisons was conducted.

2.6 | Secular Trends

Secular trends of peak BMD for the humerus and femur bones across males and females was tested using a linear regression of peak BMD against Date of Birth. The age of individuals used in the study were defined by their age falling in the range of peak BMD for bone type and sex. Coefficients of determination (R^2) were used to test for significant temporal trends defined by Date of Birth in the colony. To test for the effects of morphological covariation of secular trends on the BMD and Date of Birth trend, BMD was tested for correlations between body weight, bone length, bone circumference, and a geometric mean of body weight, length, and circumference. Body weight was known for only a subset of individuals ($N = 101$). Therefore, we derived the body weight variable from skeletal proxies using the equations described in Francis and Wang (2023).

3 | Results

3.1 | Pathology

Pathologies ranged in frequency from 10.18% to 0.18% across the population (Table 2). Sex differences were significant for seven of the 28 'original' pathologies (not including subtypes), with males exhibiting higher rates than females for all seven (Table 2; Figure 1). Three of the seven remained significant after Bonferroni correction of the original and subtype pathologies ($p \leq 0.008$ for $\alpha = 0.05/57 = 0.0008$), including periapical cavities, temporomandibular joint arthritis (TMJA), and antemortem tooth loss (AMTL). Pathologies exhibited significant association between those of known etiology, such as periodontitis and AMTL (RR = 14.24, $p \leq 0.0001$), and those of a less-well established relationship, such as periodontitis and cribra orbitalia (RR = 8.57, $p \leq 0.0001$) (Supporting Information S1: File S1). A further breakdown of pathological association by age highlighted its interplay with disease comorbidity and mortality (Supporting Information S1: File S1). For instance, TMJA's relationship with systemic rheumatic disease is driven by both early and late-stage life associations.

Most pathologies exhibited a positive relationship with age groups (Figure 2a–l; Supporting Information S1: Table S4). Although some, such as osteomyelitis, systemic infections, and periosteal reactions, were more consistent in frequency across age groups. Those with no discernible pathology exhibited the lowest median survival age (4.48) and periodontitis the oldest (18.39) (Table 3). This same pattern holds for females while those with joint disease (18.73) exhibited the greatest average ages in males. Survival rates differed significantly between the most well-represented pathologies in the skeletal collection (Figure 3 and Table 4; Supporting Information S1: Tables S5–S6). Subsets relating to severity, type, and location of pathologies exhibit differences in survivorship are depicted in Supporting Information S1: Figure S19a–h. Differences between these subsets are not significant per the pairwise comparisons (Supporting Information S1: Table S7), except for those between systemic disease subtypes: systemic infection and systemic rheumatic disease (Supporting Information S1: Figure S19e).

3.2 | Family (Matriline)

Family differences were found between pathologies including supernumerary sutures, crowding, TMJA, developmental defects, divided zygoma (DZ), and inflammatory enthesopathy (Supporting Information S1: File S2; Table 5). Pathology differences were powerful enough to be significant between families across differing age groups (Supporting Information S1: File S2). No significant differences of average life expectancy were found between families. This includes for pooled-sex (Figure 4; Supporting Information S1: Table S8), males (Supporting Information S1: Figure S20a and Table S9), and females (Supporting Information S1: Figure S20b; Table 6). Although, variation of survivorship at different life stages is evident between families such as #065 and #022, which display signs of possible longevity and short-evity of lifespan.

BMD of the femur peaked at ages 11 and 16, and the humerus at ages 12 and 10 for males and females in the Cayo Santiago population (Table 7 and Figure 5a,b). Males exhibited a greater peak BMD than females for both bones. Peak BMD of either bone did not vary for males or females across family (Supporting Information S1: Table S11).

3.3 | Hurricane

Hurricane exposure was associated with an increase in systemic disease, supernumerary sutures, crowding, and AMTL (Table 8). The hurricane exposed group (median = 5.29) is significantly older than the non-exposed group (median = 4.49) ($p \leq 0.0001$), which may have influenced overall pathology differences strongly correlated with age, such as AMTL. Across comparative age groups, 0–4-year-olds with hurricane exposure exhibited greater levels of metopic suture retention and systemic infections (Supporting Information S1: File S2). Four to 8-year-olds exhibited greater TMJA and a decrease in periosteal reactions and AMTL. Eight to 12-year-olds were associated with a decrease in periapical cavities, trauma, AMTL, and periosteal reactions. Twelve to 16-year-olds were associated with an increase in oral defects, periapical cavities, TMJA, AMTL, joint disease, and a decrease in mechanical enthesopathy. Sixteen to 20-year-olds with hurricane experience exhibited greater levels of mechanical enthesopathies. A further breakdown of the number of individuals, sex ratio, average age, and skeletal pathologies by hurricane event (Supporting Information S1: Tables S12–S13) showcases the relatively high number and percentage of pathologies within those experiencing Hurricane Hugo in early life.

Peak BMD of either bone did not vary for males or females across hurricane exposure (Table 9). Hurricane exposure was associated with a decrease of BMD in the 'Young Adult' age group (ages 5–10) (Table 6; Supporting Information S1: Figure S21b,j). For dental eruption, females broadly exhibited later occlusal eruption of primary dentition and slightly earlier eruption of permanent dentition (Figure 6; Supporting Information S1: Table S14). Average ages for tooth stages (0–4) across sex, hurricane exposure, and nutritional groups are reported in the Supporting Information S1: File S3. Hurricane experience produced differences in the timing of dental

TABLE 2 | Pathology population and sex frequency.

	No	Yes	Pct (%)	Male No	Male Yes	Male Pct (%)	Female No	Female Yes	Female Pct (%)	Fishers *p*-value
Periodontitis	2667	120	4.31	1039	52	4.77	1454	63	4.15	0.4987
Osteomyelitis	2759	28	1	1077	14	1.28	1503	14	0.92	0.4423
Supernumerary sutures	2694	93	3.34	1040	51	4.67	1475	42	2.77	**0.0103**
Periosteal reactions	2486	301	10.8	953	138	12.65	1360	157	10.35	0.0694
Syndromes	2761	26	0.93	1079	12	1.1	1503	14	0.92	0.6922
Supernumerary teeth	2778	9	0.32	1086	5	0.46	1513	4	0.26	0.5042
Neoplasm	2782	5	0.18	1087	4	0.37	1516	1	0.07	0.1681
Cribra orbitalia	2770	17	0.61	1080	11	1.01	1512	5	0.33	**0.0397**
Porotic hyperostosis	2778	9	0.32	1086	5	0.46	1513	4	0.26	0.5042
Craniosynostosis	2781	6	0.22	1088	3	0.27	1514	3	0.2	0.6992
Crowding	2717	70	2.51	1062	29	2.66	1478	39	2.57	0.9014
Congenital dental	2777	10	0.36	1089	2	0.18	1510	7	0.46	0.3193
Oral defect	2601	186	6.67	996	95	8.71	1430	87	5.74	**0.0039**
Periapical cavity	2590	197	7.07	957	134	12.28	1456	61	4.02	***< 0.0001***
Temporomandibular joint arthritis	2694	93	3.34	1031	60	5.5	1488	29	1.91	***< 0.0001***
Trauma	2564	223	8	1006	85	7.79	1383	134	8.83	0.3531
Antemortem tooth loss	2580	207	7.43	974	117	10.72	1432	85	5.6	***< 0.0001***
Developmental defect	2747	40	1.44	1075	16	1.47	1493	24	1.58	0.8727
Tibiofibular synostosis	2766	21	0.75	1080	11	1.01	1507	10	0.66	0.3770
Radioulnar synostosis	2768	19	0.68	1084	7	0.64	1507	10	0.66	1.0000
Divided zygoma	2779	8	0.29	1085	6	0.55	1515	2	0.13	0.0749
Zygomatic deformation	2747	40	1.44	1071	20	1.83	1498	19	1.25	0.2537
Osteoarthritis	2633	154	5.53	1022	69	6.32	1437	80	5.27	0.2668
Mechanical enthesopathy	2533	254	9.11	970	121	11.09	1388	129	8.5	**0.0308**
Inflammatory enthesopathy	2588	199	7.14	992	99	9.07	1419	98	6.46	**0.0133**
Joint disease	2704	83	2.98	1053	38	3.48	1473	44	2.9	0.4268
Trauma: Healed	2667	120	4.31	1048	43	3.94	1444	73	4.81	0.3357
Trauma: Healing	2697	90	3.23	1050	41	3.76	1468	49	3.23	0.5144
Trauma: No healing	2780	7	0.25	1090	1	0.09	1511	6	0.4	0.2504
Trauma “1”	2728	59	2.12	1070	21	1.92	1482	35	2.31	0.5845
Trauma “2”	2686	101	3.62	1048	43	3.94	1460	57	3.76	0.8366
Trauma “2”	2727	60	2.15	1070	21	1.92	1478	39	2.57	0.2928
Supernumerary suture: Metopic	2761	26	0.93	1073	18	1.65	1509	8	0.53	**0.0080**
Supernumerary suture: Bregma	2729	58	2.08	1062	29	2.66	1488	29	1.91	0.2262
Periosteal reaction: Active	2628	159	5.71	1017	74	6.78	1437	80	5.27	0.1102
Periosteal reaction: Mixed	2708	79	2.83	1060	31	2.84	1469	48	3.16	0.7285
Periosteal reaction: Healed	2759	28	1	1080	11	1.01	1501	16	1.05	1.0000
Periosteal reaction “1”	2693	94	3.37	1047	44	4.03	1469	48	3.16	0.2386
Periosteal reaction “2”	2656	131	4.7	1037	54	4.95	1442	75	4.94	1.0000
Periosteal reaction “3”	2742	45	1.61	1069	22	2.02	1494	23	1.52	0.3621

(Continues)

TABLE 2 | (Continued)

	No	Yes	Pct (%)	Male No	Male Yes	Male Pct (%)	Female No	Female Yes	Female Pct (%)	Fishers *p*-value
Cribra orbitalia "1"	2781	6	0.22	1086	5	0.46	1516	1	0.07	0.0884
Cribra orbitalia "2"	2778	9	0.32	1086	5	0.46	1514	3	0.2	0.2912
Crowding "1"	2746	41	1.47	1072	19	1.74	1496	21	1.38	0.5193
Crowding "2"	2767	20	0.72	1084	7	0.64	1505	12	0.79	0.8165
Crowding "3"	2784	3	0.11	1076	0	0	1514	3	0.2	0.2714
Periapical cavity: Granuloma or acute abscess	2665	122	4.38	1004	87	7.97	1484	33	2.18	***< 0.0001***
Periapical cavity: Chronic abscess	2719	68	2.44	1043	48	4.4	1499	18	1.19	***< 0.0001***
Periapical cavity: Cyst	2763	24	0.86	1074	17	1.56	1511	6	0.4	**0.0023**
Temporomandibular joint arthritis "1"	2762	25	0.9	1074	17	1.56	1510	7	0.46	**0.0057**
Temporomandibular joint arthritis "2"	2738	49	1.76	1059	32	2.93	1501	16	1.05	***0.0006***
Temporomandibular joint arthritis "3"	2771	16	0.57	1083	8	0.73	1511	6	0.4	0.2831
Syndrome: Pathology	2774	13	0.47	1085	6	0.55	1510	7	0.46	0.7834
Syndrome: Congenital	2780	7	0.25	1088	3	0.27	1513	4	0.26	1.0000
Systemic disease	2709	78	2.8	1055	36	3.3	1477	40	2.64	0.3459
Systemic disease: Rheumatic	2742	45	1.61	1070	21	1.92	1495	22	1.45	0.3540
Systemic disease: Infection	2754	33	1.18	1076	15	1.37	1499	18	1.19	0.7239
Congenital disorder	2755	32	1.15	1078	13	1.19	1499	18	1.19	1.0000

Note: Bolded values are below 0.05, whilst bolded and italicized values are below the Bonferroni correction of 0.00047.

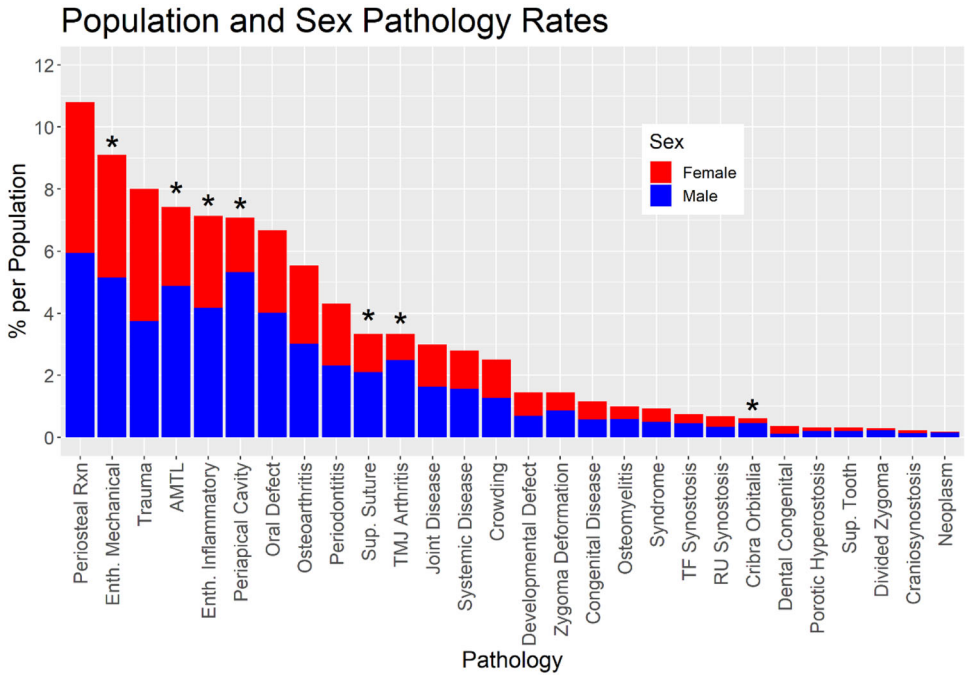


FIGURE 1 | Pathology rates and sex differences in the Cayo Santiago population. Asterisks indicate significant differences between male and females per pathology.

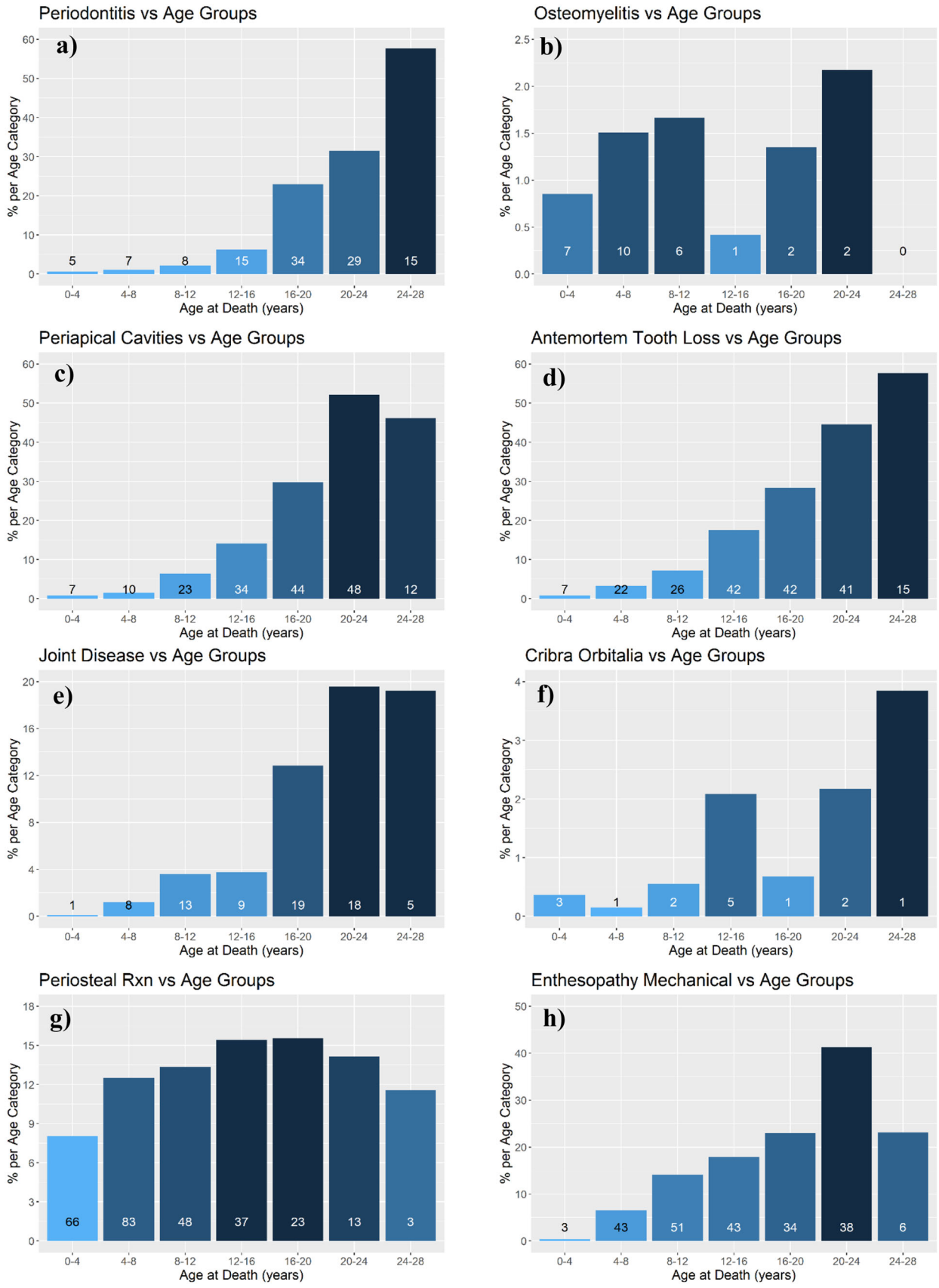


FIGURE 2 | Pathology frequency across age groups in the Cayo Santiago population. Numbers towards the base of the graphs represent N per age group: (a) Peridontitis; (b) Osteomylitis; (c) Periapical Cavities; (d) Antemortem Tooth Loss; (e) Joint Disease; (f) Cribra Orbitalia; (g) Periosteal Rxn (Reaction); (h) Enthesopathy Mechanical; (i) Trauma; (j) Enthesopathy Inflammatory; (k) TMJ Arthritis; (l) Congenital Disease; (m) Systemic Disease.

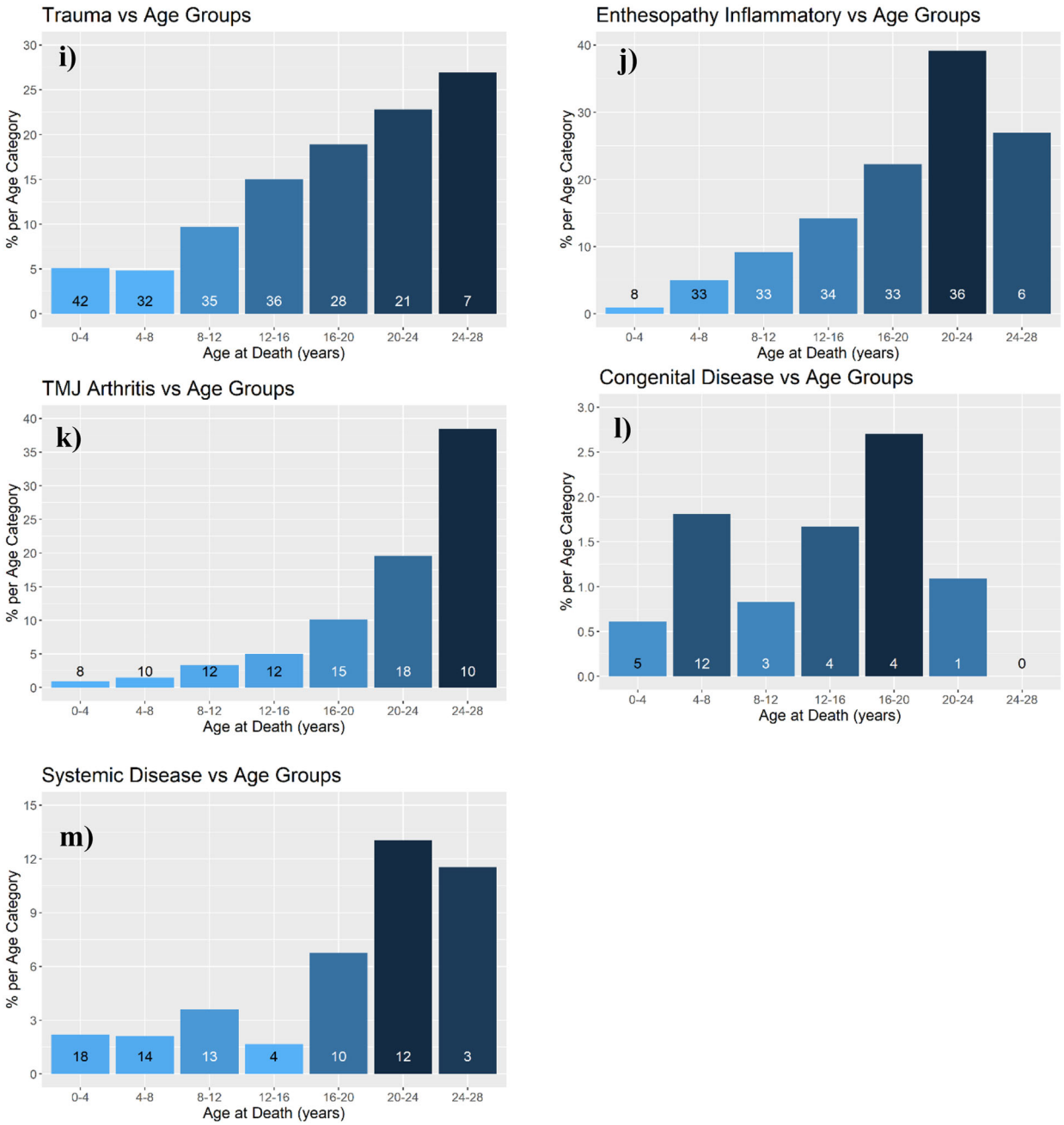


FIGURE 2 | (Continued)

eruption in the population (Figure 7; Supporting Information S1: Table S14). For males, hurricane experience was associated with delayed primary dental eruption and slightly earlier eruption of permanent teeth. For females, both primary and permanent teeth were delayed relative to those not in the hurricane group.

3.4 | Nutrition

Protein provisioned diets were associated with a decrease in periodontitis, oral defects, osteoarthritis, and mechanical enthesopathy (Table 10). The protein-diet group (median = 6.57) is significantly younger than the pre-protein group (median = 9.12) ($p \leq 0.0001$), which may have influenced overall pathology differences that are strongly correlated with age, such as OA. Across comparative age groups, 0–4-year-olds under the protein diet exhibited a decrease in syndromes, developmental defects, and congenital disorders (SI File S2). Four to 8-year-olds exhibited a decrease in OA and an increase in periosteal reactions. Eight to 12-year-olds were associated with a decrease in oral defects and OA. Twelve to 16-year-olds exhibited less periosteal reactions, oral defects, OA, and mechanical enthesopathy. Sixteen to 20-year-olds in the protein provisioned group exhibited decreased levels of periosteal reactions, oral defects, osteoarthritis, and mechanical enthesopathy.

Peak BMD of either bone did not vary for males or females across nutrition (Table 11). Those experiencing the protein

TABLE 3 | Median survival ages of pathologies for total population, males, and females (years).

Pathology	Total	Males	Females
AMTL	16.16	15.85	16.94
Congenital disease	7.17	7.02	7.98
Cribra orbitalia	12.55	12.45	13.75
Enthesopathy inflammatory	13.84	13.01	14.43
Enthesopathy mechanical	13.53	13.24	13.66
Joint disease	16.66	18.73	16.07
No lesions	4.48	3.36	5.15
Osteomyelitis	6.03	9.27	5.43
Periapical cavity	17.94	17.85	18.04
Periodontitis	18.39	18.39	18.70
Periosteal reaction	6.69	6.82	6.69
Pop. Total	5.95	5.34	6.26
Systemic disease	9.09	10.22	8.00
TMJ arthritis	15.89	17.34	8.73
Trauma	10.50	10.53	10.44

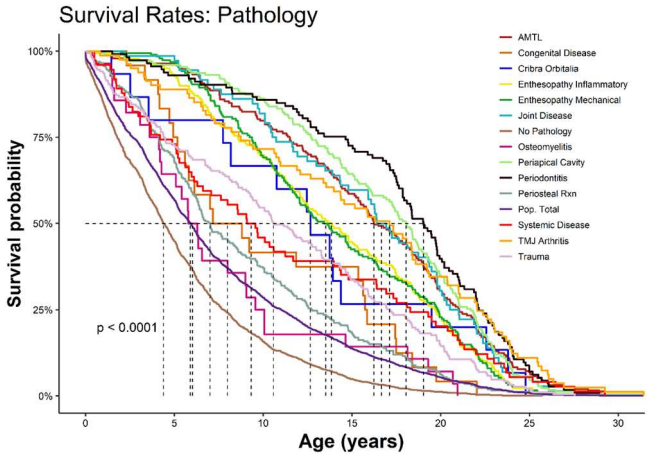


FIGURE 3 | Survival rates of well-represented pathologies in the Cayo Santiago population. Vertical dotted lines denote the 50% survivorship ages of pathologies.

provisioned diet exhibited significantly reduced BMD across sex and age groups compared to the pre-protein group (Table 12; Supporting Information S1: Figure S22). Those in the protein-provisioned group exhibited earlier eruption ages of the primary dentition and similar, or in some teeth earlier, permanent timings (Figure 8; Supporting Information S1: Table S14).

3.5 | Secular Trends

Peak BMD was found to decline dramatically over time in the colony (Figure 9). Peak BMD was found to weakly correlate to, or was not correlated with, body weight, lone bone length, long bone circumference or a geometric mean of all three (Supporting Information S1: Table S15).

4 | Discussion

The Cayo Santiago population exhibit a range of skeletal pathologies that encompass biomedical and archaeological significance, multiple etiologies, severities, locations, and types. Familial differences are observed in relation to certain pathologies across age groups indicative of a genetic contribution to skeletal health variation in the population such as the many observed in #065, which corroborates our first prediction and Hypothesis 1. Although, average of survivorship was not found to differ between families. Hurricane exposure was associated with an overall greater number of systemic diseases, reduced BMD in 'young adults', and delayed eruption of primary dentition, corroborating our second prediction and Hypothesis 2. These findings relate to the effects of hurricane-induced adversity (Testard et al. 2021; Pavez-Fox et al. 2021; Watowich et al. 2022; Luevano et al. 2022; Testard et al. 2023; Gonzalez, Sherer, and Hernández-Pacheco 2023; Romero et al. 2023; Diaz et al. 2023; Patterson et al. 2023; Motes-Rodrigo et al. 2023) on accelerated biological aging, behaviorally augmented pathogen transmission, and necessary energy reallocation. The switch to a protein-rich diet was associated with a decrease in oral defects and OA, lower BMD of the humerus and femur, and an accelerated eruption of primary dentition. This was congruent with predictions regarding pathology and dental eruption, and corroborated Hypothesis 3, but went against our prediction for the effect of protein-provisioning on BMD levels. Fewer oral defects and earlier dental eruption may reflect the effects a higher quality nutrition, similar to those seen between captive and wild populations. However, reduced OA and BMD between nutritional groups, as well as a falling peak BMD over time, is instead hypothesized to reflect a potential secular trend of decreasing physical activity, likely linked to increasing population density in more recent periods. Altogether, the health history of rhesus macaques at Cayo Santiago reflects the intertwined evo-devo mechanisms involving environmental and genetic factors that determine outcomes of growth, development, health, and diseases.

4.1 | Pathology

The frequency of recorded pathologies in the population reflects the many osteological studies that find periosteal reactions, enthesopathies, trauma, and antemortem tooth loss as some of the most ubiquitous skeletal lesions (Assis and Keenleyside 2019). Periosteal reactions can occur with any insult to the periosteum, including via trauma, infection, inflammation, and even as a byproduct of normal infant growth. Enthesopathies, AMTL, OA, and periapical cavities have strong links to aging, while trauma is expected in a despotic species roaming a spatially limited habitat. Sex differences are found in relation to certain pathologies, with males exhibiting a greater number in all sex-disparate pathologies. This may highlight different lifeways between males and females. For instance, greater antemortem tooth loss, periapical cavities,

TABLE 4 | Pairwise comparisons of pathology survival rates for total population.

Total	AMTL	Congenital disease	Cribra orbitalia	Enthesopathy inflammatory	Enthesopathy mechanical	Joint disease	No lesions	Osteo-myelitis	Periapical cavity	Periodontitis	Periosteal reaction	Pop total	Systemic disease	TMJ arthritis
Congenital disease	***0.0003***	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Cribra orbitalia	1.0000	1.0000	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Enthesopathy inflammatory	0.6129	0.1035	1.0000	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Enthesopathy mechanical	0.3428	0.0821	1.0000	1.0000	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Joint disease	1.0000	**0.0008**	1.0000	1.0000	1.0000	NA	NA	NA	NA	NA	NA	NA	NA	NA
No lesions	***< 0.0001***	0.1256	**0.0413**	***< 0.0001***	***< 0.0001***	***< 0.0001***	NA	NA	NA	NA	NA	NA	NA	NA
Osteomyelitis	***< 0.0001***	1.0000	1.0000	***< 0.0001***	***< 0.0001***	***< 0.0001***	1.0000	NA	NA	NA	NA	NA	NA	NA
Periapical cavity	1.0000	***< 0.0001***	1.0000	**0.0471**	**0.0164**	1.0000	***< 0.0001***	***< 0.0001***	NA	NA	NA	NA	NA	NA
Periodontitis	1.0000	***< 0.0001***	1.0000	**0.0076**	**0.0034**	1.0000	***< 0.0001***	***< 0.0001***	1.0000	NA	NA	NA	NA	NA
Periosteal reaction	***< 0.0001***	1.0000	1.0000	***< 0.0001***	***< 0.0001***	***< 0.0001***	***< 0.0001***	1.0000	***< 0.0001***	***< 0.0001***	NA	NA	NA	NA
Pop total	***< 0.0001***	1.0000	1.0000	***< 0.0001***	***< 0.0001***	***< 0.0001***	***< 0.0001***	1.0000	***< 0.0001***	***< 0.0001***	1.0000	NA	NA	NA
Systemic disease	0.0974	1.0000	1.0000	1.0000	1.0000	1.0000	***< 0.0001***	1.0000	**0.0252**	**0.0099**	0.0654	**0.0034**	NA	NA
TMJ arthritis	1.0000	**0.0217**	1.0000	1.0000	1.0000	1.0000	***< 0.0001***	***0.0001***	1.0000	1.0000	***< 0.0001***	***< 0.0001***	1.0000	NA
Trauma	***< 0.0001***	1.0000	1.0000	0.4619	0.6843	**0.0118**	***< 0.0001***	1.0000	***< 0.0001***	***< 0.0001***	**0.0025**	***< 0.0001***	1.0000	**0.0151**

Note: Bolded values are below 0.05, whilst bolded and italicized values are below the Bonferroni correction of 0.00047 (df = 104).

TABLE 5 | Pathologies per Family of total population, males, and females.

Pathology	Pooled sex *p*-value	Male *p*-value	Female *p*-value
Periodontitis	0.2064	0.8970	0.6296
Osteomyelitis	0.9535	0.5292	0.8720
Supernumerary sutures	**0.0010**	0.1489	**0.0174**
Periosteal reaction	0.3583	0.2753	0.7776
Syndromes	0.5077	0.9645	0.5957
Supernumerary teeth	0.8175	0.7336	0.7706
Neoplasm	0.9555	0.8820	0.5477
Cribra orbitalia	0.8955	0.7791	0.9925
Porotic hyperostosis	1.0000	1.0000	1.0000
Craniosynostosis	0.3073	0.5872	0.7216
Crowding	**0.0055**	**0.0289**	**0.0269**
Congenital dental	0.1154	0.7271	**0.0479**
Oral defect	0.3868	0.2388	0.8885
Periapical cavity	0.3138	0.3148	0.2433
Temporomandibular joint arthritis	***0.0005***	**0.0119**	0.2603
Trauma	0.5522	0.2903	0.5107
Antemortem tooth loss	0.0899	0.1394	0.6706
Developmental defect	**0.0474**	0.1519	**0.0444**
Tibiofibular synostosis	0.7171	0.3418	0.8450
Radioulnar synostosis	0.6916	0.2108	0.4812
Divided zygoma	**0.0100**	***0.0025***	0.5102
Zygomatic deformation	0.0594	0.2838	**0.0239**
Osteoarthritis	0.6291	0.4552	0.2563
Mechanical enthesopathy	0.1974	0.5842	0.1629
Inflammatory enthesopathy	**0.0389**	0.1259	0.4157
Joint disease	0.6356	0.8880	0.6221
Trauma: Healed	0.1819	0.0739	0.1639
Trauma: Healing	0.4677	0.4772	0.3908
Trauma: No healing	0.4922	0.7401	0.6321
Trauma “1”	0.4277	0.2253	0.6296
Trauma “2”	0.3913	0.3158	0.2608
Trauma “2”	0.7381	0.5607	0.8670
Supernumerary suture: Metopic	**0.0229**	0.4577	**0.0085**
Supernumerary suture: Bregma	0.0584	0.1819	0.5647
Periosteal reaction: Active	0.3588	0.4078	0.3608
Periosteal reaction: Mixed	0.2448	0.8565	0.1174
Periosteal reaction: Healed	0.2733	0.7071	0.4397
Periosteal reaction “1”	**0.0379**	0.2438	0.0644
Periosteal reaction “2”	0.8755	0.7916	0.3488
Periosteal reaction “3”	0.2713	0.8920	0.1104
Cribra orbitalia “1”	1.0000	1.0000	1.0000
Cribra orbitalia “2”	0.7456	0.5362	0.8845

(Continues)

TABLE 5 | (Continued)

Pathology	Pooled sex *p*-value	Male *p*-value	Female *p*-value
Crowding "1"	**0.0224**	**0.0179**	0.2004
Crowding "2"	0.2009	0.2638	0.5892
Crowding "3"	1.0000	1.0000	1.0000
Periapical cavity: Granuloma or acute abscess	0.4247	0.3883	0.2948
Periapical cavity: Chronic abscess	0.7041	0.5447	0.9090
Periapical cavity: Cyst	0.4937	0.3923	0.4562
Temporomandibular joint arthritis "1"	0.7346	0.6886	0.9060
Temporomandibular joint arthritis "2"	0.0814	**0.0479**	0.8650
Temporomandibular joint arthritis "3"	**0.0020**	**0.0075**	0.4098
Syndrome: Pathology	0.9630	0.9595	0.6791
Syndrome: Congenital	0.9150	0.9110	0.8260
Systemic disease	0.7796	0.8950	0.8415
Systemic disease: Rheumatic	0.9725	0.6511	0.7396
Systemic disease: Infection	0.7996	0.9030	0.8250
Congenital disorder	0.3328	0.6551	0.2983

Note: Bolded values are below 0.05, whilst bolded and italicized values are below the Bonferroni correction of 0.00087 (df = 56).

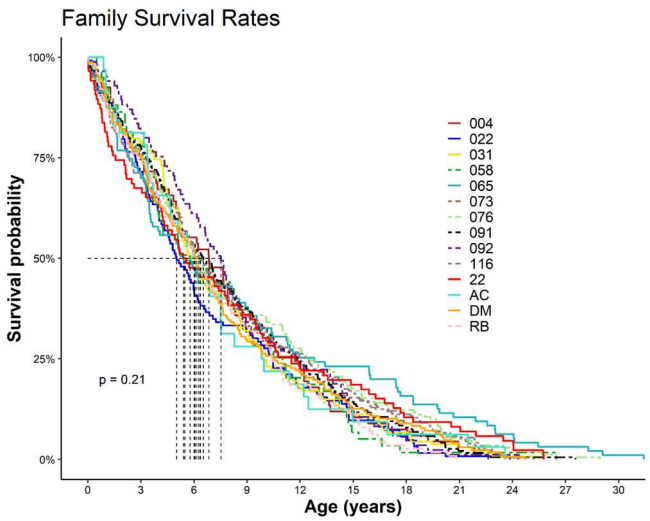


FIGURE 4 | Survival rates per family. Vertical dotted lines represent the 50% survivorship age for each matriline. Matrilines are listed in the righthand legend.

and TMJA in males may all be related to greater levels of tooth wear, instances of trauma through physical fighting, or a deterioration of health in males associated with heightened muscle atrophy and physical wasting (Li et al. 2018; Guatelli-Steinberg et al. 2022; Turcotte et al. 2022). Enthesopathies have been documented to be greater in males than females (Mariotti, Facchini, and Belcastro 2004; Milella et al. 2012; Foster, Buckley, and Tayles 2014). Age, physical activity, hormones, divisions of labor, and spondyloarthropathy are implicated in the pathogenesis of enthesopathies (Mariotti, Facchini, and Belcastro 2004; Milella et al. 2012; Foster, Buckley, and Tayles 2014). Cribra Orbitalia (CO) has exhibited slight female skews in the archaeologic record (Suby 2014), including primates (DeGusta 2010), purportedly due to lower blood iron levels in females associated with menstrual bleeding, labor, and lactation (Mittler and Van Gerven 1994). A higher male frequency in the Cayo Santiago macaques is, therefore, contra to conventional wisdom and may suggest that etiologies alternative to iron-deficiency anemia hold validity (Brickley 2018; Rothschild et al. 2021; Zdilla et al. 2022). A greater number of supernumerary sutures in males are predominantly driven by the persistence of varying amounts of metopic suture completeness, though the etiology of metopism is not well-established. Sex differences of incomplete metopic sutures have been observed more in both females (Pakdeewong and Tohno 2019), and males (Grine et al. 2024), though other studies have found no sex differences (Berry 1975; Eroğlu 2008).

Those without observable lesions exhibiting the lowest average survival rates (Figure 3 and Table 3) lends strong support to the principles of the osteological paradox (Wood et al. 1992; McFadden and Oxenham 2020; Wyatt et al. 2023). Namely, that it is possible that those exhibiting skeletal disease are more resilient than those without them as they have survived long enough for lesions to have formed, whereas those exposed to the same disease have died before this process and are thus more frail (Ortner 1991). While unknown for the majority of individuals, some of these diseases include enteritis, myocarditis, and pneumonia. Several diseases have a high average

TABLE 6 | Bone mineral density and hurricane experience across age groups.

Hurricane experience	Young			Young adult			Prime adult			Aged adult		
	Yes	No	*p*-value	Yes	No	*p*-value	Yes	No	*p*-value	Yes	No	*p*-value
Male												
Femur	4022	4090	0.79	4164	4227	**0.04**	4199	4265	0.77	4257	4180	0.11
Humerus	4055	4108	0.42	4206	4156	0.82	4156	4302	0.22	4173	4199	0.88
Female												
Femur	4050	4073	0.70	4077	4159	***< 0.01***	4129	4165	0.83	4153	4192	0.15
Humerus	3998	4064	0.26	4111	4152	0.54	4087	4145	0.77	4129	4129	0.90

Note: Bolded values are below 0.05, whilst bolded and italicized values are below the Bonferroni correction of 0.0031 (df = 15).

TABLE 7 | Peak age of bone mineral density (BMD) and the start and end of the 95% range of that peak.

	Start	End	Peak
Males			
Femur	4244.840 (9)	4234.650 (16)	4255.783 (11)
Humerus	4270.143 (10)	4265.287 (14)	4279.934 (12)
Females			
Femur	4176.351 (10)	4174.737 (21)	4187.070 (16)
Humerus	4165.258 (8)	4164.594 (11)	4172.137 (10)

Note: Bracketed values denote the age of start, end, or peak of BMD. Unit: m/s (ultrasonic speed as density proxy).

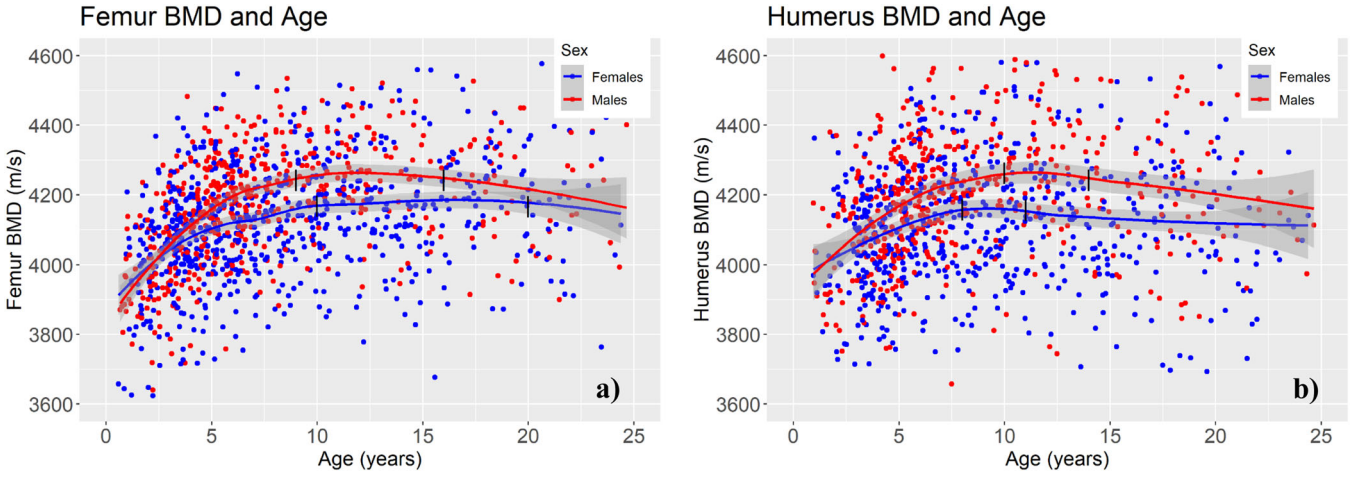


FIGURE 5 | Bone mineral density of the femur (a) and humerus (b) for males and females. Black lines denote to the 95% range of peak BMD. Black lines demarcate the start and end point of peak BMD ages.

mortality rate and do not significantly differ from those with no discernible lesions, including congenital disease and osteomyelitis. Osteomyelitis is readily diagnosed in osteological material, most common to subadults and infants, and is serious sign of ill-health given it is an infection of the bone marrow cavity with high potential to migrate throughout the body. Congenital disease is made up of several single diseases including craniosynostosis, cleft palate, and supernumerary teeth, among others. While each does not carry an obvious effect on survivorship, they may be further associated with other non-osteological defects as part of a larger disease suite. Therefore, some skeletally diagnosed diseases, such those congenitally sourced and osteomyelitis, cannot, in isolation, be associated with relative resilience (DeWitte and Stojanowski 2015).

The oldest average survivorship pathologies are associated with a degenerative, age-related, component including periodontitis, periapical cavities, and TMJA that all correspond to clinically sourced patterns. Periodontitis and TMJA have chronic and accumulative components to their etiology, meaning they are far more likely to be found in older individuals (Ebersole et al. 2016). Periapical cavities have also been postulated to increase with age due to greater tooth wear and resulting caries, fracture, and pulp exposure (Li et al. 2018). The majority of

TABLE 8 | Pathology per Hurricane experience.

Hurricane exposed individuals (H) vs. not	No (H)	Yes (H)	% (H)	No	Yes	%	*p*-value
Periodontitis	452	21	4.44	1822	94	4.91	0.7206
Osteomyelitis	462	11	2.33	1899	17	0.89	**0.0152**
Supernumerary sutures	446	27	5.71	1855	61	3.18	**0.0134**
Periosteal reaction	429	44	9.30	1686	230	12.0 s	0.1070
Syndromes	469	4	0.85	1895	21	1.10	0.8030
Supernumerary teeth	470	3	0.63	1911	5	0.26	0.1983
Neoplasm	473	0	0.00	1911	5	0.26	0.5899
Cribra orbitalia	471	2	0.42	1903	13	0.68	0.7491
Porotic hyperostosis	472	1	0.21	1908	8	0.42	1.0000
Craniosynostosis	471	2	0.42	1912	4	0.21	0.3396
Crowding	458	15	3.17	1866	50	2.61	0.5273
Congenital dental	472	1	0.21	1908	8	0.42	1.0000
Oral defect	442	31	6.55	1774	142	7.41	0.5538
Periapical cavity	442	31	6.55	1766	150	7.83	0.3833
Temporomandibular joint arthritis	453	20	4.23	1848	68	3.55	0.4954
Trauma	434	39	8.25	1754	162	8.46	0.9265
Antemortem tooth loss	435	38	8.03	1754	162	8.46	0.8529
Developmental defect	464	9	1.90	1886	30	1.57	0.5488
Tibiofibular synostosis	470	3	0.63	1900	16	0.84	1.0000
Radioulnar synostosis	468	5	1.06	1903	13	0.68	0.3773
Divided zygoma	472	1	0.21	1911	5	0.26	1.0000
Zygomatic deformation	467	6	1.27	1883	33	1.72	0.6846
Osteoarthritis	460	13	2.75	1792	124	6.47	**0.0012**
Mechanical enthesopathy	440	33	6.98	1730	186	9.71	0.0747
Inflammatory enthesopathy	436	37	7.82	1768	148	7.72	0.9236
Joint disease	460	13	2.75	1855	61	3.18	0.7660
Trauma: Healed	453	20	4.23	1824	92	4.80	0.7154
Trauma: Healing	456	17	3.59	1854	62	3.24	0.6680
Trauma: No Healing	472	1	0.21	1912	4	0.21	1.0000
Trauma “1”	462	11	2.33	1875	41	2.14	0.8601
Trauma “2”	459	14	2.96	1843	73	3.81	0.4143
Trauma “2”	460	13	2.75	1870	46	2.40	0.6222
Supernumerary suture: Metopic	461	12	2.54	1903	13	0.68	**0.0013**
Supernumerary suture: Bregma	460	13	2.75	1875	41	2.14	0.3925
Periosteal reaction: Active	452	21	4.44	1796	120	6.26	0.1561
Periosteal reaction: Mixed	457	16	3.38	1858	58	3.03	0.6582
Periosteal reaction: Healed	470	3	0.63	1893	23	1.20	0.4557
Periosteal reaction “1”	468	5	1.06	1840	76	3.97	**0.0009**
Periosteal reaction “2”	447	26	5.50	1820	96	5.01	0.6420
Periosteal reaction “3”	464	9	1.90	1883	33	1.72	0.8447
Cribra orbitalia “1”	472	1	0.21	1911	5	0.26	1.0000

(Continues)

TABLE 8 | (Continued)

Hurricane exposed individuals (H) vs. not	No (H)	Yes (H)	% (H)	No	Yes	%	*p*-value
Cribra orbitalia "2"	472	1	0.21	1910	6	0.31	1.0000
Crowding "1"	467	6	1.27	1885	31	1.62	0.6820
Crowding "2"	466	7	1.48	1903	13	0.68	0.0939
Crowding "3"	472	1	0.21	1914	2	0.10	0.4842
Periapical cavity: Granuloma or Acute Abscess	449	24	5.07	1827	89	4.65	0.7166
Periapical cavity: Chronic Abscess	464	9	1.90	1862	54	2.82	0.3362
Periapical cavity: Cyst	470	3	0.63	1897	19	0.99	0.5981
Temporomandibular joint arthritis "1"	470	3	0.63	1896	20	1.04	0.5996
Temporomandibular joint arthritis "2"	461	12	2.54	1882	34	1.77	0.2661
Temporomandibular joint arthritis "3"	468	5	1.06	1905	11	0.57	0.3392
Syndrome: Pathology	473	0	0.00	1905	11	0.57	0.1358
Syndrome: Congenital	471	2	0.42	1912	4	0.21	0.3396
Systemic disease	451	22	4.65	1862	54	2.82	0.0556
Systemic disease: Rheumatic	462	11	2.33	1884	32	1.67	0.3354
Systemic disease: Infection	462	11	2.33	1894	22	1.15	0.0741
Congenital disorder	465	8	1.69	1895	21	1.10	0.3450

Note: Bolded values are below 0.05, whilst bolded and italicized values are below the Bonferroni correction of 0.00087 (df = 56).

TABLE 9 | Peak age bone mineral density (BMD) between those experiencing hurricane impacts.

	Yes	No	*p*-value
Males			
Femur	4168	4220	0.5107
Humerus	4107	4204	0.4599
Females			
Femur	4142	4104	0.3535
Humerus	4141	4102	0.4761

Note: Bolded values are below 0.05, whilst bolded and italicized values are below the Bonferroni correction of 0.0125 (df = 3).

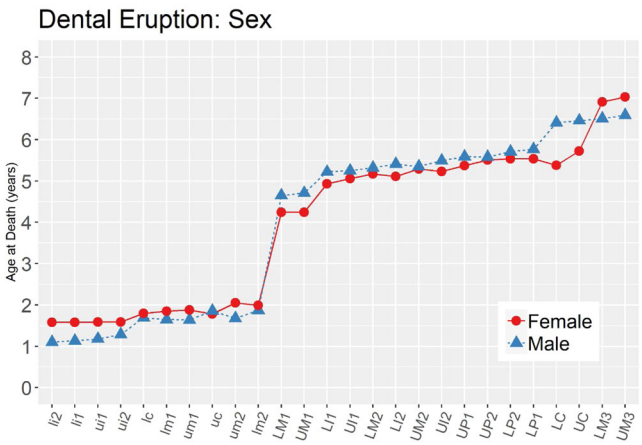


FIGURE 6 | Dental eruption in the Cayo Santiago Rhesus macaques across sexes.

pathologies increased across ages, per the survival analysis and frequencies across age groups.

RR ratios provide a chance to showcase relationships between skeletal lesions and diseases. Some are etiologically linked and intuitive, including periodontitis and AMTL (Raitapuro-Murray, Molleson, and Hughes 2014; Gilmore and Weaver 2016; Bertl et al. 2020) (RR = 14.24; $p \leq 0.0001$), trauma and limb synostosis (Antón and Polidoro 2000; Khudaverdyan, Khachatryan, and Eganyan 2016; Darton et al. 2017) (RR = 10.36; $p \leq 0.0001$), and congenital dental defects and crowding (RR = 25.9; $p < 0.0001$). Some are likely the result of their respective age distributions, such as osteoarthritis and periapical cavities (RR = 6.74; $p \leq 0.0001$), periapical cavities and supernumerary sutures (RR = 0.22; $p = 0.03$), and mechanical enthesopathies and supernumerary sutures (RR = 0.21; $p = 0.01$). However, some are unexpected and not immediately intuitive, such as cribra orbitalia and periodontitis (RR = 8.57; $p \leq 0.0001$). Six of the 17 individuals with cribra orbitalia also present with periodontitis. Their association, coupled with an elusive etiology of the former pathology, provides space to explore a potential underlying connection

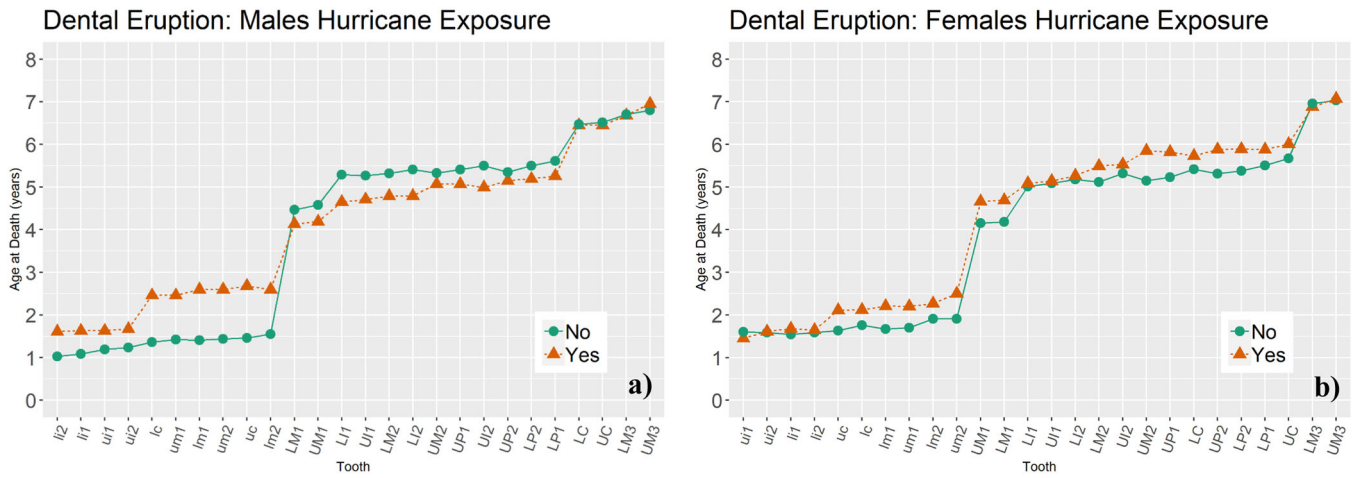


FIGURE 7 | Dental eruption in the Cayo Santiago Rhesus macaques between hurricane groups and males (a) and females (b).

TABLE 10 | Pathology per nutritional status.

Nutrition pre-protein (1) vs. post (2)	No (1)	Yes (1)	% (1)	No (2)	Yes (2)	% (2)	*p*-value
Periodontitis	252	29	10.32	778	36	3.76	***0.0006***
Osteomyelitis	278	3	1.07	802	12	1.22	0.7716
Supernumerary sutures	271	10	3.56	771	43	3.76	0.3326
Periosteal reaction	237	44	15.66	718	96	11.03	0.0980
Syndromes	279	2	0.71	805	9	1.12	0.7386
Supernumerary teeth	280	1	0.36	810	4	0.34	1.0000
Neoplasm	281	0	0.00	810	4	0.24	0.5775
Cribra orbitalia	277	4	1.42	807	7	0.54	0.4868
Porotic hyperostosis	280	1	0.36	810	4	0.39	1.0000
Craniosynostosis	280	1	0.36	812	2	0.24	1.0000
Crowding	270	11	3.91	794	20	2.54	0.2129
Congenital dental	278	3	1.07	813	1	0.24	0.0542
Oral defect	234	47	16.73	763	51	5.81	***< 0.0001***
Periapical cavity	246	35	12.46	724	90	6.59	0.5155
Temporomandibular joint arthritis	272	9	3.20	763	51	3.61	0.0666
Trauma	250	31	11.03	751	63	8.00	0.1076
Antemortem tooth loss	243	38	13.52	734	80	7.37	0.0941
Developmental defect	277	4	1.42	801	13	1.71	1.0000
Tibiofibular synostosis	279	2	0.71	805	9	0.83	0.7386
Radioulnar synostosis	275	6	2.14	811	3	0.59	**0.0111**
Divided zygoma	279	2	0.71	810	4	0.20	0.6499
Zygomatic deformation	275	6	2.14	798	16	1.46	0.8089
Osteoarthritis	220	61	21.71	792	22	2.44	***< 0.0001***
Mechanical enthesopathy	237	44	15.66	738	76	8.05	**0.0053**
Inflammatory enthesopathy	253	28	9.96	742	72	7.32	0.5507
Joint disease	270	11	3.91	788	26	2.98	0.5676
Trauma: Healed	260	21	7.47	781	33	4.20	**0.0362**
Trauma: Healing	271	10	3.56	784	30	3.37	1.0000

(Continues)

TABLE 10 | (Continued)

Nutrition pre-protein (1) vs. post (2)	No (1)	Yes (1)	% (1)	No (2)	Yes (2)	% (2)	*p*-value
Trauma: No healing	281	0	0.00	814	0	0.20	1.0000
Trauma "1"	277	4	1.42	796	18	2.24	0.6217
Trauma "2"	264	17	6.05	787	27	3.32	0.0524
Trauma "2"	271	10	3.56	796	18	2.29	0.2710
Supernumerary suture: Metopic	278	3	1.07	799	15	1.07	0.5861
Supernumerary suture: Bregma	276	5	1.78	790	24	2.39	0.3899
Periosteal reaction: Active	261	20	7.12	760	54	5.76	0.7834
Periosteal reaction: Mixed	274	7	2.49	788	26	3.22	0.6870
Periosteal reaction: Healed	275	6	2.14	809	5	0.98	**0.0379**
Periosteal reaction "1"	262	19	6.76	789	25	2.98	**0.0123**
Periosteal reaction "2"	264	17	6.05	773	41	4.98	0.5370
Periosteal reaction "3"	280	1	0.36	795	19	2.00	**0.0360**
Cribra orbitalia "1"	280	1	0.36	810	4	0.24	1.0000
Cribra orbitalia "2"	279	2	0.71	811	3	0.24	0.6074
Crowding "1"	274	7	2.49	800	14	1.42	0.4498
Crowding "2"	279	2	0.71	808	6	0.83	1.0000
Crowding "3"	281	0	0.00	814	0	0.15	1.0000
Periapical cavity: Granuloma or acute abscess	257	24	8.54	755	59	4.00	0.5134
Periapical cavity: Chronic abscess	268	13	4.63	784	30	2.20	0.4789
Periapical cavity: Cyst	277	4	1.42	800	14	0.88	1.0000
Temporomandibular joint arthritis "1"	279	2	0.71	799	15	0.98	0.2653
Temporomandibular joint arthritis "2"	275	6	2.14	789	25	1.85	0.5330
Temporomandibular joint arthritis "3"	280	1	0.36	806	8	0.63	0.4610
Syndrome: Pathology	281	0	0.00	810	4	0.54	0.5775
Syndrome: Congenital	280	1	0.36	813	1	0.24	0.4475
Systemic disease	274	7	2.49	785	29	3.32	0.4437
Systemic disease: Rheumatic	276	5	1.78	799	15	1.81	1.0000
Systemic disease: Infection	279	2	0.71	800	14	1.51	0.3851
Congenital disorder	275	6	2.14	806	8	1.07	0.2126

Note: Bolded values are below 0.05, whilst bolded and italicized values are below the Bonferroni correction of 0.00087 (df = 56).

between oral disease and the orbital porous lesions. Such a connection has not previously been found in archaeological populations and either, thus, explored in terms of etiology (Liebe-Harkort 2010; Wilham 2016). This link may have remained elusive likely due to the life stages at which both are expected to manifest and their subsequent influence on mortality: CO in childhood/adolescence and periodontitis into adulthood and old age.

Traditionally, CO has been linked with iron-deficiency anemia, as well as hemolytic and megaloblastic types. Assuming convention, clinical explanations have been put forth for a relationship between periodontitis and iron-deficient anemia—including the suppression of erythropoietin through heightened cytokine response in patients with periodontitis, leading to the development of anemia (Anumolu, Srikanth, and Paidi 2016). Anemia of inflammation (AI) has been suggested to form secondary to systemic inflammation, such as that potentially caused by periodontitis, through pro-inflammatory cytokines promoting hepcidin and increased iron-trapping in macrophages (Nibali et al. 2019). Conversely, it has been proposed the physical appearance of the lesions in the orbit defining CO are not actually porous but likely vascular in appearance and origin (Rothschild et al. 2021). Specifically, the authors considered

arteriovenous malformations, hemorrhage, hemangiomas, and subperiosteal hematomas as possible causes for CO. The link between cardiovascular disease (CVD) and periodontitis is already well-established. CVD is either linked to oral disease via periodontal pathogens directly invading the bloodstream or by augmenting the systemic level of inflammatory markers (Zardawi et al. 2021). The medical records of individuals tell us two of the 17 with CO died directly due to cardiac events, and another due to septicemia of which vascular disease is a common comorbidity. Documentation of another four individuals presenting with CO exhibited nephritis, a pancreatic tumor, ovarian cysts, and chronic enteritis, all of which are known to have a systemic or direct potential impact on vascular disease. Therefore, there is support for an etiological link between the orbital lesions characteristic of CO and periodontitis via underlying systemic diseases such as CVD.

The breakdown of pathological relationships across age groups enables us to assess RR at different points of the lifespan. For instance, the relationship between TMJA and systemic rheumatic disease is driven by both early (0–4 and 4–8 years) and later (20–24 years) age associations. This suggests that TMJA lesions in skeletal material may not solely reflect the wear-and-tear process that initiates and worsens into late adulthood, but in younger individuals more likely indicates an affected joint as part of a rheumatic disease. Observed later-life associations may likewise be accounted for this way, which would depict a bimodal relationship of TMJA lesions and rheumatic disease in the Cayo Santiago population, affecting subadult and aged adults. Alternatively, TMJA lesions in later life may be wear-and-tear related and incidental to comorbid rheumatic diseases which are more frequent in later life (Goronzy and Weyand 2012). Subadult TMJ lesions are more confidently attributed to a systemic disease, when in concurrence with other non-localized lesions, compared to later-life TMJA, given the low chances of a degenerative cause at such ages coinciding with other lesions. For known rheumatic arthropathies, such as rheumatoid arthritis (RA), the TMJ is the last joint to be affected (Sodhi et al. 2015) and is only implicated in 10%–24% of ankylosis spondylitis (Arora et al. 2013). Further work may uncover specific rheumatic diseases known to effect skeletal material (Ventades et al. 2018) and link systemic disease to potential TMJ disorders in human and nonhuman primates

Though rarely arising to statistical significance, survival analysis of pathology severity, status, and type are informative for a variety of reasons. Healed periosteal lesions are associated with greater survivorship than those with active or mixed lesions. The same has been found in the tibia of human remains indicative of periosteal lesions able to closely track frailty in a population (DeWitte 2014). Similarly, healed traumas exhibit greater survivorship than those with healing traumas throughout life. A common pattern for severity categories of periosteal reactions, trauma, and TMJA is their most severe manifestations exhibit lower survivorship than more mild versions in early life stages, with this trend reversing in later life stages. This pattern likely describes a more direct link between lesion and mortality event in early life stages and the selectiveness of the most resilient individuals in later life. Conversely, it may be that severity increases due to gradual degeneration of disease for TMJA and periosteal reactions (PRXN). Whereas in earlier life, TMJA, PRXN, and trauma are associated with systemic disease and accumulative wear and tear in later life. For trauma, later age survivorship may result from age-related bone loss and fracture severity.

TABLE 11 | Peak age bone mineral density (BMD) between those experiencing varying nutritional qualities.

	Pre-protein	Post-protein	*p*-value
Males			
Femur	4388	4251	0.2452
Humerus	4435	4303	0.4742
Females			
Femur	4286	4174	0.4812
Humerus	4321	4138	0.5187

Note: Bolded values are below 0.05, whilst bolded and italicized values are below the Bonferroni correction of 0.0125 (df = 3).

4.2 | Family (Matriline)

Average survivorship did not differ between the main ($N > 30$) matrilines (Figure 5 and Table 13), neither when analyzed separately for sex (Supporting Information S1: Figure S20 and Tables S10–S11). Matrilines exhibit clear differences in survivorship rate across different ages despite a lack of average disparity. In females for example, no individuals reach the age of 18 in for four out of 14 matrilines, whereas another matriline (#076) enjoyed a 15% survival rate at 18 years (Supporting

TABLE 12 | Bone mineral density and nutritional status across age groups.

	Young			Young adult			Prime adult			Aged adult		
Nutritional status	**'1'**	**'2'**	***p*-value**	**'1'**	**'2'**	***p*-value**	**'1'**	**'2'**	***p*-value**	**'1'**	**'2'**	***p*-value**
Male												
Femur	4288	4110	***< 0.01***	4302	4223	*0.45*	4396	4249	**0.01**	4242	4145	0.64
Humerus	4188	4065	0.06	4371	4216	**0.01**	4457	4302	**0.03**	4248	4148	0.54
Female												
Femur	4218	4097	0.41	4289	4145	0.05	4334	4183	0.22	4188	4162	0.05
Humerus	4183	4065	0.11	4309	4128	0.05	4239	4146	0.14	4196	4069	**0.01**

Note: Bolded values are below 0.05, whilst bolded and italicized values are below the Bonferroni correction of 0.0031 (df = 15).

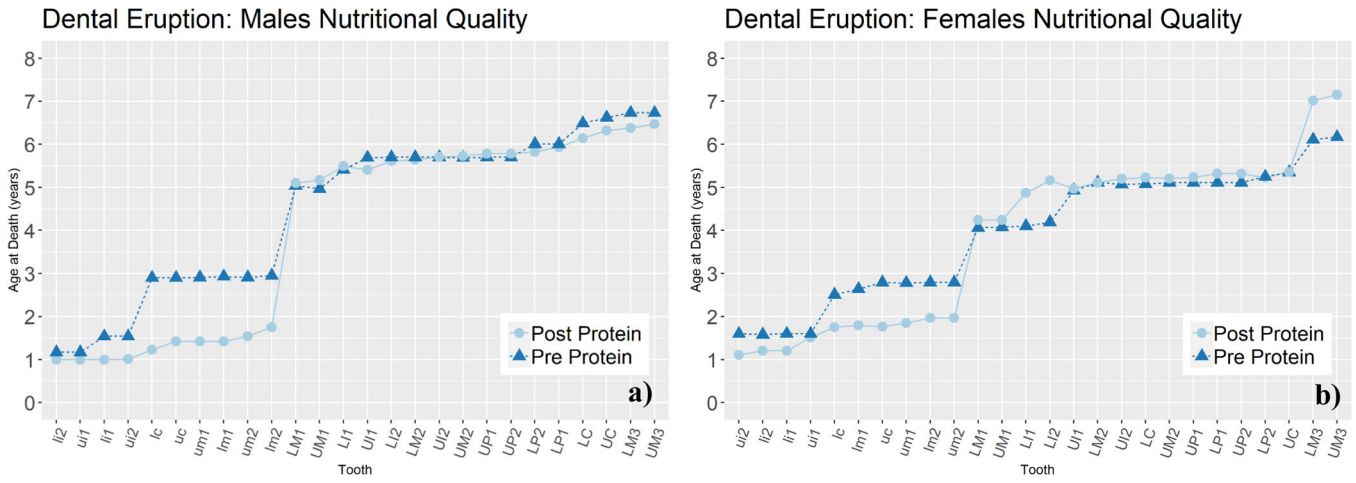


FIGURE 8 | Dental eruption in the Cayo Santiago Rhesus macaques between nutrition groups and males (a) and females (b).

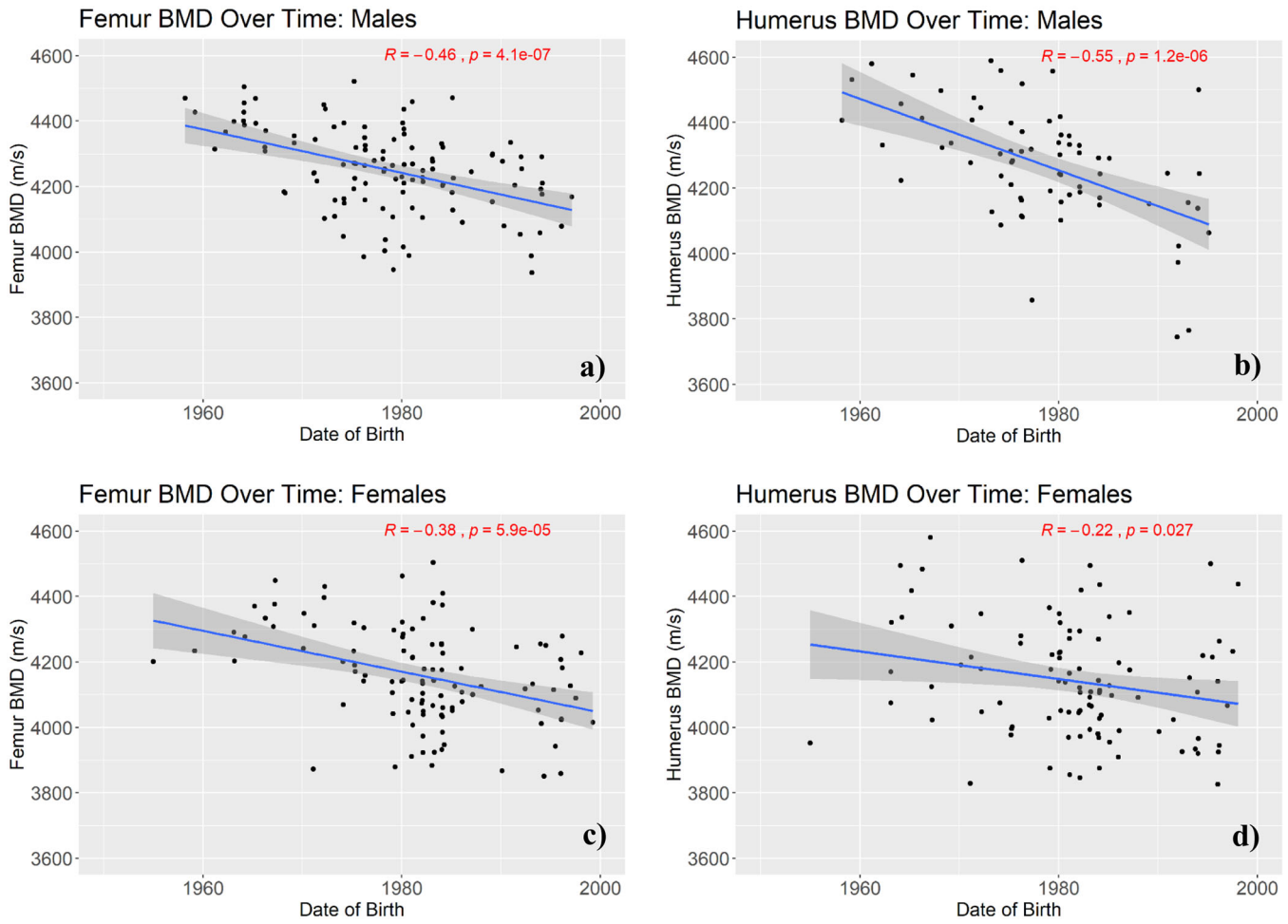
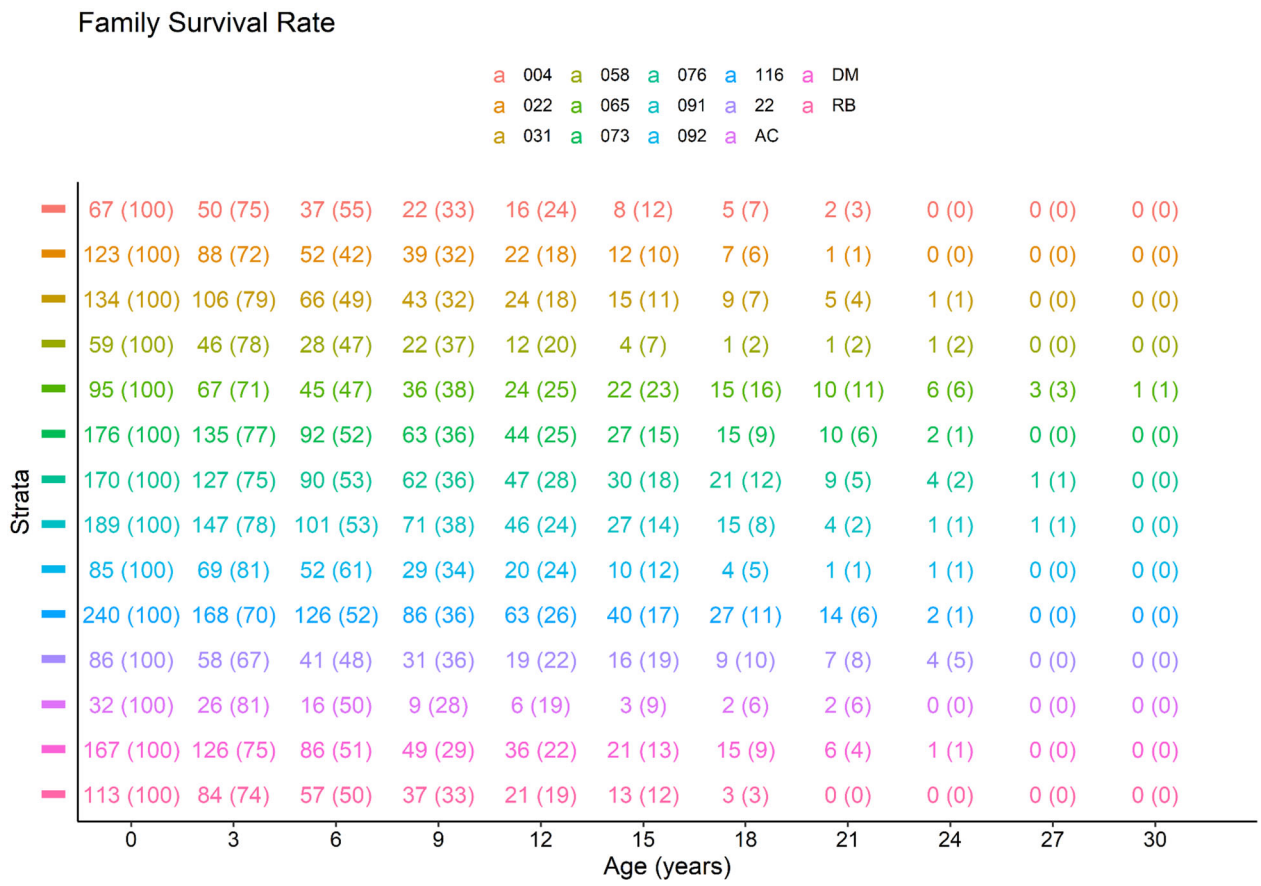


FIGURE 9 | Secular trends of peak BMD across bone type and sex in the Cayo Santiago colony: (a) Femur BMD in males; (b) Humerus BMD in males; (c) Femur BMD in females; (d) Humerus BMD in females.

Information S1: Figure S20b and Table S11). Additionally, matriline #092 enjoys relatively low subadult mortality rates in males and several members of matriline #065 exhibit extended lifespans (Figure 20a and Table 6). This may reflect a heritable component of aging in the Cayo macaques. Although though lifespan heritability is relatively minor in humans, ranging from estimates of 10%–40% (Singh et al. 2019; van den Berg et al. 2019). It is possible that ecological variation, such as exposure to hurricanes, influences lifespan (Diaz et al. 2023; Newman et al. 2023). Social status is also known to mitigate injury-related deaths (Pavez-Fox et al. 2022) and mediate inflammatory levels (Pavez-Fox et al. 2021) which is associated

TABLE 13 | Risk table of family survival across 3-year age intervals for pooled-sex.



Note: Matrilines correspond to the colors in the legend above. Values represent absolute numbers across each 3-year interval. Bracketed values indicate percentage still alive at each interval.

with molecular aging (Watowich et al. 2022). Therefore, as social hierarchies exist between matrilines (Maestripieri and Hoffman 2011), the effects of rank, trait heritability, and lifespan are likely to be tightly interlinked between matrilines.

The role of disease is another factor that should theoretically influence lifespan between matrilines. Disease expression can be influenced by heritable genetic contributions. For example, three out of the six individuals with craniosynostosis in the colony are born within a 2-year timespan of each other, are from the same matriline (#091), and all exhibit the uni-coronal form of the disease. The link between skeletal disease and lifespan is often not easily interpretable, however. For instance, matriline #065 exhibits the highest rates of periodontitis (Gonzalez et al. 2016; Ebersole et al. 2019), periapical abscesses (Li et al. 2018), TMJA, and the 2nd highest of AMTL. Yet, they are also the top-ranked matriline in their social group (Chepko-Sade and Sade 1979) and have the highest survival rate of any matriline past 13 years of age (Figure 5). Their high expression of disease is likely a product of the high proportion of their long-lived members. Following this, the variation of skeletal disease at earlier life-stages between matrilines may represent a more robust metric with which to test for a genetic component of disease expression than in looking at the entirety of the lifespan. For matriline #065, two out of the ten cases of TMJA are expressed in the 0–4 age group. Early onset of TMJA suggests that these lesions may not be solely due to socially mediated longevity, but a genuine susceptibility to TMJ disorders or disease generally. Moreover, matriline #031 exhibits relatively high levels of disease across age groups compared to other matrilines. From ages 0–12, 11.92% of individuals (13/109) exhibit systemic disease, 4.23% greater than any other matriline (next is #065 at 7.69%) across the same age interval. This suggests matriline #031 has a genetic affinity for disease. Furthermore, matriline #091 exhibits the highest rate (4.16%) of congenital disease in the population. Their high rate of craniosynostosis is accompanied by two incidences of supernumerary teeth, two with a congenitally fused vertebrae, and another with cleft palate. The individual with cleft palate is also the common maternal ancestor of all those of subsequent congenital disease within its matriline going back at most, four generations. The high concentration of birth defects following a mother with cleft palate suggests the cause for congenital disease within matriline #091 is at least partly per inheritance of chromosomal or single-gene malformations.

Major differences were also found for the rate of cranial supernumerary sutures. These differences are accentuated after limiting the frequency to those under 8 years of age, in which the vast majority are recorded. For this age bracket, frequency

ranged from 22.72% (#AC) to 0% (#DM and #062). Recently, presence of the bregmatic bone (McCloud, Francis, and Wang 2023) and pterion type (Francis and Wang 2024) have been shown to be vary along matrilines. Therefore, sutural characteristics may have a strong genetic component that is observable across matrilines in Cayo Santiago, either through direct inheritance or indirectly via cranial growth characteristics. The DZ is another type of cranial suture that appears to vary between matrilines (Wang and Dechow 2016a). Ranging from 6.45% in matriline #062 with 11/14 major matrilines not exhibiting any cases, the overall prevalence in major matrilines is relatively low. However, the two individuals of matriline #062 with the DZ are notably siblings, sharing a mother. This at least hints at DZ as a feature of inheritance. Matriline #062 additionally exhibits the highest rate of developmental defects (9.67%), which denotes qualitative asymmetry of the sagittal suture or overlapping calvaria bones. The three individuals exhibiting this feature in matriline #062 are all born within a 4-year timespan and includes a mother-offspring relationship. Though this exact feature has not previously been recorded as a pathological lesion or otherwise, it seems to also exhibit a genetic component to its presence. It has been found that reducing complexity of the sagittal suture is related to the effects of inbreeding (Alados, Escós, and Emlen 1995) which may also be causing presence of sagittal deformity here.

No differences in peak BMD were found between families. A similar finding has been made by Cerroni et al. (2000) who found natal group in the Cayo macaques showed no relationship with vertebral BMD. This suggests that peak BMD is not significantly inherited in the Cayo population.

4.3 | Hurricane

Hurricane exposed individuals exhibited overall increased rates of systemic disease, supernumerary sutures, crowding, and AMTL. While the exposed group is older than the non-exposed group, only one of these pathologies significantly correlated with increased age and thus is potentially skewing real differences between groups. The aftermath of hurricane impacts is linked to a strong uptick in disease prevalence. A weakened immune system, chronic low-grade inflammation, physical trauma, population crowding, and standing water are all factors that contribute to this phenomenon (Liang and Messenger 2018). The most devastating hurricane to have hit Cayo Santiago, Hurricane Maria in 2017, is only known to have caused approximately 50 monkey deaths (2.75% total population) (Romero et al. 2023), with a hypothesis that the colony survived by huddling on the side of the island protected by a cliff face. Closer physical proximity between Cayo residents has been found post-hurricane impact due to increased social connections, lower aggression, and sharing sporadic shade areas as 60% of the island's vegetation cover was stripped away (Testard et al. 2023). This social reorganization post-hurricane is adaptive but has also been modeled to increase disease transmission rates due to closer proximity (Motes-Rodrigo et al. 2023). The findings reported here support this assertion. Greater levels of systemic disease are found in those experiencing hurricanes than those that did not. Furthermore, those dying at 0–4 years of age after hurricane exposure exhibited significantly greater levels of systemic infection disease (4.24%; $p = 0.047$) compared those not exposed (1.26%). This suggests that behavioral modifications purposed toward enhancing survival in the aftermath of devastating hurricanes may lead to increased pathogen transmission and systemic disease in the Cayo Santiago macaques.

Systemic rheumatic disease is also greater in the hurricane exposed group, though this is not statistically significant (Table 8). Hurricane impacts at Cayo have been associated with immune dysregulation, chronic low-grade inflammation, and accelerated biological aging which is known to increase risk of autoimmune diseases (Watowich et al. 2022). The same process may be causing increased rates of observed systemic rheumatic disease in the Cayo macaques post-hurricane. Although, frequencies fluctuate across age groups (Supporting Information S1: File S2) with no clear pattern or evidence that systemic rheumatic disease initiates earlier in the hurricane exposed group.

Age-related effects of hurricane exposure are further supported by BMD analyses. The femur of young adults (ages 5–10) exhibited reduced BMD measures in those exposed to the hurricane group compared to those not in the hurricane group (Supporting Information S1: Figures S19b and S19j). Low BMD at comparative ages has been associated with early-life adversity (Gough and Godde 2019; Wuertz-Kozak et al. 2020). Specifically, low socioeconomic status in humans has been associated with an epigenetically mediated state of chronic inflammation and a resultant reduction of BMD (Brennan-Olsen et al. 2016). The impact of major hurricanes may, therefore, accelerate the skeletal aging process in a similar way that has been identified in the molecular aging process (Watowich et al. 2022): through adversity, dysregulation of the immune system and a heightened inflammatory response. However, peak BMD was not found to vary between hurricane exposed groups. Instead, we uncovered a strong secular trend of declining peak BMD over time in the Cayo Santiago population. It is possible that, although the hurricane groups are sampled from overlapping time periods, the source driving the strong secular trends is responsible for the differences observed between hurricane groups. The source of the BMD may relate to acclimation (Francis and Wang 2023). Secular trends of body weight and long bones broadly declined in the Cayo Santiago population purportedly to mitigate heat loss in response to homogenously warm climates. Body weight is a known predictor of BMD (Hoxha et al. 2014), yet peak BMD showed little to no correlation with body weight, long bone dimensions or a geometric mean of each (Supporting Information S1: Table S3). This suggests the BMD secular trend may be driven by other factors in the colony. Increasingly high populational densities over the colony's timespan (Hernández-Pacheco et al. 2013; Hernandez-Pacheco et al. 2016; Newman et al. 2023) may be influencing this trend, through reduced activity levels. Physical activity and inactivity are associated with long bone BMD in humans (Chastin et al. 2014). Therefore, as population densities have increased in the island habitat, and the number of territorial social groups remained relatively stable, each individual, on average, may be increasingly physically inactive which in turn may be reducing peak BMD values over time. This trend may be further exacerbated by vegetation destruction post-hurricanes which could diminish opportunity for arboreal locomotion and overall activity levels. Alternatively, hurricanes may be driving

the observed secular trends by reducing peak BMD in all individuals experiencing these events, not just those in-utero up to the age of 4 at time of impact. As hurricanes occurred in the second half of the colony sample time period, this explanation remains plausible.

Hurricane exposure was further associated with delayed eruption of the primary dentition. Low socioeconomic status has also been associated with delayed dental eruption (Lee, Low, and Chang 1965; Garn et al. 1973; Cardoso 2007). Similarly, captive populations frequently exhibit accelerated dental maturation relative to wild populations (Phillips-Conroy and Jolly 1988; Zihlman, Bolter, and Boesch 2004; Zihlman, Bolter, and Boesch 2007; Bolter and Zihlman 2011; Wang, Turnquist, and Kessler 2016b). A more ambient environment has been proposed as a possible explanation for this discrepancy (Wang, Turnquist, and Kessler 2016b), as has an alignment to accelerated life history events in captivity (Zihlman, Bolter, and Boesch 2004). The same logic may be applicable here. Females experiencing the hurricane impacts at Cayo are known to delay reproductive debut in favor of strategy of early-life energy allocation to growth and maintenance to maximize later reproduction potential (Luevano et al. 2022). This adaptive strategy may explain the trends of delayed eruption of primary dentition as well as female permanent dentition in the hurricane-exposed group. Whereas the acceleration of male permanent teeth may reflect a differing strategy, where male dental eruption aligns with accelerated reproductive debut in accordance with predicted shortened lifespans after early life adversity (Luevano et al. 2022; McDermott et al. 2023). Therefore, the shared pattern of delayed dental eruption in both males and females for primary teeth may represent a more immediate effect of hurricane impact, whereby energy is reallocated away from dental development and toward immediate survival and maintenance.

The partial preservation of metopic sutures out of early infancy and into adolescence and adulthood has previously been recorded in the Cayo Santiago macaques, including a male biased trend (Wang et al. 2006). Those in the hurricane exposed group exhibit greater quantities of this feature than those not exposed. Causes for the preservation of full metopic sutures are unknown and typically considered a normal anatomical variant. Suggestions include an underdeveloped frontal sinus (Guerram et al. 2014), a relationship precipitated by early nutritional deficiencies (Nikolova et al. 2018), though this relationship has been disputed (Ogut and Yildirim 2023), and partial metopism has even been associated with a larger frontal sinus volume (Grine et al. 2024). There is no obvious explanation for why hurricane impact would directly preserve the metopic suture or through abnormal frontal sinus development. Studying the relationship between early-life adversity and partial metopic suture preservation requires further investigation.

4.4 | Nutrition

Individuals born into the protein-provisioned diet consistently exhibited decreased rates of osteoarthritis and oral defects. Oral defects encompass recorded calculus, alveolar recession, caries, and the loss of tooth crown. Better nutrition would intuitively lead to better oral health as we find here, though previous analyses have found no difference in individual bone calcium content between the protein and non-protein diets at Cayo (Grynpas et al. 1993). Indeed, the lack of differences between the two dietary-groups in terms of well-represented oral pathologies including periodontitis, periapical cavities, or AMTL may be due to the fact that both are provisioned from birth. However, we do find increased crowding between ages 4–12, where it is 3.67% in the protein group and 0% in the pre-protein group. This may reflect a similar phenomenon in humans since the rise of agriculture and the post-industrial revolution, in which a shift to softer-foods has reduced the size of the mandible and thus the space for teeth to fully erupt and align (von Cramon-Taubadel 2011). Further work correlating dental crowding to masticatory dimensions may more fully elucidate such a pattern across artificial diets and generational timespans in the Cayo Santiago macaques.

Protein intake is positively associated with BMD in humans (Mangano, Sahni, and Kerstetter 2014). Therefore, it is unexpected that the protein individuals consistently exhibited lower BMD measures across age groups, though no differences of peak BMD were found. We suggest this trend is likely a consequence of the secular trend observed in peak BMD throughout the colony. Particularly, as the two groups derive from differing periods of the colony's temporal range. It is hypothesized that this phenomenon is caused by diminished activity due to consistent food provisioning, a constricted roaming area, and lack of predators, reflecting the decrease of overall bone quality from hunter-gatherers to agriculturalists (Ryan and Shaw 2015; Chirchir et al. 2017).

Those in the protein-provisioned group exhibited accelerated maturation of the primary dentition. This may similarly reflect trends of accelerated dental eruption in captive population relative to those in the wild, as discussed for hurricane impacts. A significant factor believed to drive accelerated maturation in captive populations is the regularity and caloric value of provisioned foods (Phillips-Conroy and Jolly 1988; Zihlman, Bolter, and Boesch 2004; Zihlman, Bolter, and Boesch 2007; Bolter and Zihlman 2011; Wang, Turnquist, and Kessler 2016b). It is possible the switch to protein-rich diet has produced a similar trend. Equally, it is possible that the accelerated dental maturation for the protein group corresponds to an accelerated life-history.

4.5 | Limitations of This Study

A clear limitation of the current study is the cross-sectional nature of the sample. Individual life-course heterogeneity is significant in the Cayo Santiago population (Diaz et al. 2023). Cross-sectional samples potentially lose this heterogeneity. We only possess a snapshot of individual health, and it is at the point of death which may not be representative of health over the lifetime. We, therefore, have prevalence of disease but not incidence. Moreover, prevalence of disease in skeletal material cannot be directly conflated with relative ill-health, as outlined in the osteological paradox. Future studies that link skeletal health to available medical records in the Cayo Santiago colony may be a useful resource for forensic, archaeological, and collection works for the

interpretation of health and disease. A further limitation is the relatively small sample of pre-provisioned individuals, limiting the rigor of our findings regarding the impact of nutrition on skeletal health. Another future study would also benefit from incorporating detailed population density data with BMD measurements and behavioral observations of activity levels in the Cayo Santiago population. It does appear however that BMD is declining in the population independent of altering body shapes (Francis and Wang 2023).

4.6 | Conclusion

The findings contained within this study represent a vast repository of skeletal pathology data. This record provides context to existing debates of lesion etiology and association, delineates sex-specific susceptibilities linked to behavior and life-histories, and reflects within it some of the most pervasive diseases observed clinically and in the archaeological record. Genetic and environmental signatures on pathology prevalence also contribute to our understanding of the factors that pattern skeletal disease and health. Certain matrilineal families exhibit greater pathologies on the whole and at comparable age groups, together illustrating that familial susceptibility to diseases vary in totality and for age of onset. This serves as a potential blueprint for subsequent allele-based studies of disease within the Cayo population, which will become increasingly feasible as paternity data is enriched, complementing extensive maternal records. Major hurricane events appear to have had a considerable impact on the rates of systemic disease, BMD, and dental eruption timing in the population. Infectious disease rates are placed in the context of documented social adaptations and pathogen transmission modeling post-hurricane, in the Cayo population. BMD reductions and an uptick in inflammatory disorders are postulated to reflect chronic low-grade inflammation and immune dysregulation already linked to early-life hurricane experience in the Cayo colony members. While dental eruption delays are thought to represent a reallocation of energetic resources to maintenance and increased disease load or, alternatively, are acting in tandem with delayed life-histories. The switch from a low protein to high protein provisioned diet was linked with fewer oral defects and accelerated eruption of the primary dentition. Importantly, this analysis also allowed us to uncover possible secular trends of declining peak BMD and levels of osteoarthritis which may reflect decreasing levels of physical activity via increased population density or loss of substrate complexity due to devastating hurricane impacts on vegetation.

Author Contributions

George Francis: data curation (equal), formal analysis (lead), investigation (equal), methodology (equal), visualization (lead), writing–original draft (equal), writing–review and editing (support). **Qian Wang:** conceptualization (lead), data curation (equal), formal analysis (support), investigation (equal), methodology (equal), project administration (lead), visualization (support), writing–original draft (equal), writing–review and editing (lead).

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Ethics Statement

The research was non-invasive. For research work, we obtained permission to collect skeletal data from CPRC.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

Data available upon reasonable request.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section.

Appendix

As an alternative approach to multiple pairwise comparisons, Linear Mixed Modeling (LMM) and Generalized Mixed Modeling methods were performed for each pathology. Fixed effects include hurricane exposure, age, nutrition, sex (and body weight for those of bone mineral density, osteoarthritis, and joint disease) and random effects including social group and matriline. Age and nutrition was added as interaction due to the relative old average age of those within the pre-protein provisioning group. Random effects may be interpreted relative to the value of the estimate of fixed effects as they are on the same log odds scale. Body weight was included as a variable for analysis of bone mineral density, osteoarthritis, and joint disease given the factors likely role in their disease expression. Body weight was calculated as a geometric mean of femur, tibia, humerus, and radius long bone length, and bizygomatic breadth. The variable was then scaled to enhance modeling performance.

The results of the LMM and GLMM lead to similar conclusions to the pairwise analyses in the main text and on a per-pathology basis for the most part. Hurricane remains a significant predictor of certain pathologies, including bone density levels, osteomyelitis, and supernumerary sutures. Sex is also a significant predictor of the diseases already highlighted where a male exhibits greater pathology frequencies than females including, periapical cavities, temporomandibular joint arthritis, antemortem tooth loss, and enthesopathies. Age similarly is commonly found to significantly predict pathology occurrence in this additional analysis. Nutrition is found to be significant in similar disease to the main text analyses even though there was not a cut-off of sample born later than 1986-01-01 as there was in the former. The role of social group and matriline affiliations appear to be variable, with their standard deviations greater than all fixed-effects estimates in certain pathologies (such as periodontitis), to negligible difference in others. Those pathologies without an associated analysis are those models that would not converge. This commonly happened with pathologies which were lower in sample size in the "Yes" column.

Pathology	Variable	Estimate	Std. Error	*t* value	Pr(> |t|)
BMD_Femur	(Intercept)	4310.431	31.334	137.561	0.000
	HurricaneY	−34.101	11.375	−2.997	0.002
	EAAD	−5.697	2.011	−2.832	0.004
	Nutrition2	−164.895	31.658	−5.208	0.000
	SexM	−4.442	11.543	−0.384	0.700
	BW.scaled	50.472	6.408	7.875	0.000
		St. Dev			
	Social Group	37.120			
	Matriline	33.800			
BMD_Humerus					
	(Intercept)	4327.781	39.987	108.228	0.000
	HurricaneY	−30.720	18.432	−1.666	0.103
	EAAD	−7.098	2.483	−2.858	0.004
	Nutrition2	−162.012	40.813	−3.969	0.000
	SexM	−5.358	14.886	−0.359	0.718
	BW.scaled	65.731	8.678	7.573	0.000
	EAAD:Nutrition2	3.769	2.691	1.400	0.161
		St.Dev			
	Social Group	44.720			
	Matriline	20.160			

Pathology	Variable	Estimate	Std. Error	*z* value	Pr(> |z|)	0.025	0.975
Periodontitis	(Intercept)	−6.036	0.813	−7.423	0.000	−7.629	−4.442
	HurricaneY	0.223	0.300	0.742	0.458	−0.366	0.811
	Age	0.247	0.042	5.823	0.000	0.164	0.330
	Nutrition2	−0.086	0.883	−0.097	0.923	−1.817	1.646
	SexM	0.207	0.232	0.893	0.372	−0.248	0.662
	Age:Nutrition2	−0.008	0.048	−0.175	0.861	−0.101	0.085
		St. Dev.					
	Social Group	0.479					
	Matriline	0.014					
Osteomyelitis	(Intercept)	−5.407	1.202	−4.498	0.000	−7.763	−3.051
	HurricaneY	0.987	0.407	2.425	0.015	0.189	1.785
	EAAD	0.058	0.078	0.743	0.458	−0.095	0.211
	Nutrition2	0.471	1.230	0.383	0.702	−1.940	2.882
	SexM	0.399	0.383	1.041	0.298	−0.353	1.151
	EAAD:Nutrition2	−0.050	0.085	-0.586	0.558	−0.217	0.117
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Supernumerary Sutures	(Intercept)	−2.513	0.631	−3.984	0.000	−3.749	−1.276
	HurricaneY	0.730	0.264	2.760	0.006	0.211	1.248
	EAAD	−0.170	0.083	−2.049	0.040	−0.333	−0.007

(Continues)

Pathology	Variable	Estimate	Std. Error	*z* value	Pr(> |z|)	0.025	0.975
	Nutrition2	−0.544	0.636	−0.855	0.393	−1.790	0.703
	SexM	0.483	0.225	2.150	0.032	0.043	0.924
	EAAD:Nutrition2	0.024	0.089	0.263	0.793	−0.152	0.199
		St. Dev.					
	Social Group	0.406					
	Matriline	0.775					
Periosteal Reactions	(Intercept)	−2.482	0.380	−6.537	0.000	−3.226	−1.738
	HurricaneY	−0.212	0.182	−1.165	0.244	−0.568	0.144
	EAAD	0.053	0.025	2.099	0.036	0.004	0.103
	Nutrition2	−0.015	0.413	−0.037	0.970	−0.825	0.794
	SexM	0.272	0.134	2.025	0.043	0.009	0.535
	EAAD:Nutrition2	−0.019	0.028	−0.694	0.488	−0.074	0.035
		St. Dev.					
	Social Group	0.478					
	Matriline	0.482					
Syndromes	(Intercept)	−4.477	1.533	−2.920	0.004	−7.481	−1.472
	HurricaneY	−0.137	0.593	−0.231	0.817	−1.299	1.025
	EAAD	−0.347	0.299	−1.162	0.245	−0.933	0.238
	Nutrition2	−1.686	1.434	−1.176	0.240	−4.496	1.124
	SexM	0.376	0.433	0.868	0.385	−0.473	1.225
	EAAD:Nutrition2	0.401	0.301	1.332	0.183	−0.189	0.991
		St. Dev.					
	Social Group	0.854					
	Matriline	1.230					
Supernumerary Teeth	(Intercept)	−9.867	3.668	−2.690	0.007	−17.055	−2.678
	HurricaneY	0.936	0.769	1.217	0.224	−0.571	2.443
	EAAD	0.226	0.159	1.417	0.156	−0.086	0.538
	Nutrition2	2.363	3.714	0.636	0.525	−4.917	9.643
	SexM	0.960	0.736	1.304	0.192	−0.483	2.404
	EAAD:Nutrition2	−0.110	0.167	−0.661	0.509	−0.438	0.217
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Neoplasm	NA						
Cribra Orbitalia	(Intercept)	−6.698	1.453	−4.611	0.000	−9.546	−3.851
	HurricaneY	−0.290	0.786	−0.370	0.712	−1.830	1.249
	EAAD	0.120	0.077	1.575	0.115	−0.029	0.270
	Nutrition2	0.258	1.497	0.172	0.863	−2.676	3.192
	SexM	0.955	0.563	1.696	0.090	−0.148	2.058
	EAAD:Nutrition2	−0.027	0.087	−0.307	0.759	−0.197	0.144

(Continues)

Pathology	Variable	Estimate	Std. Error	*z* value	Pr(> |z|)	0.025	0.975
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Porotic Hyperostosis	(Intercept)	−12.496	5.799	−2.155	0.031	−23.862	−1.129
	HurricaneY	−0.838	1.126	−0.744	0.457	−3.044	1.369
	EAAD	0.040	0.331	0.120	0.905	−0.610	0.689
	Nutrition2	0.786	5.378	0.146	0.884	−9.756	11.327
	SexM	1.312	0.841	1.560	0.119	−0.337	2.961
	EAAD:Nutrition2	0.016	0.338	0.046	0.963	−0.647	0.678
		St. Dev.					
	Social Group	1.139					
	Matriline	2.562					
Craniosynostosis	(Intercept)	−7.332	3.416	−2.146	0.032	−14.028	−0.636
	HurricaneY	0.334	0.937	0.357	0.721	−1.502	2.171
	EAAD	−0.445	0.550	−0.810	0.418	−1.523	0.632
	Nutrition2	−3.133	2.771	−1.131	0.258	−8.565	2.298
	SexM	0.458	0.854	0.536	0.592	−1.217	2.133
	EAAD:Nutrition2	0.373	0.558	0.669	0.503	−0.720	1.467
		St. Dev.					
	Social Group	0.000					
	Matriline	2.241					
Crowding	NA						
Congenital Dental	(Intercept)	−3.021	1.230	−2.456	0.014	−5.433	−0.610
	HurricaneY	−0.226	1.120	−0.202	0.840	−2.421	1.970
	EAAD	−0.239	0.214	−1.114	0.265	−0.659	0.181
	Nutrition2	−2.950	1.424	−2.071	0.038	−5.740	−0.159
	SexM	−0.635	0.845	−0.752	0.452	−2.291	1.021
	EAAD:Nutrition2	0.270	0.226	1.193	0.233	−0.174	0.713
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Oral Defect	(Intercept)	−3.331	0.502	−6.638	0.000	−4.314	−2.347
	HurricaneY	0.246	0.253	0.972	0.331	−0.250	0.741
	EAAD	0.103	0.028	3.728	0.000	0.049	0.157
	Nutrition2	−1.802	0.535	−3.370	0.001	−2.849	−0.754
	SexM	0.556	0.188	2.955	0.003	0.187	0.925
	EAAD:Nutrition2	0.110	0.032	3.465	0.001	0.048	0.173
		St. Dev.					
	Social Group	0.695					
	Matriline	0.804					

(Continues)

Pathology	Variable	Estimate	Std. Error	*z* value	Pr(> |z|)	0.025	0.975
Periapical Cavity	(Intercept)	−5.987	0.617	−9.696	0.000	−7.197	−4.777
	HurricaneY	−0.031	0.258	−0.122	0.903	−0.538	0.475
	EAAD	0.219	0.034	6.360	0.000	0.152	0.287
	Nutrition2	−0.331	0.636	−0.521	0.603	−1.579	0.916
	SexM	1.655	0.208	7.973	0.000	1.248	2.062
	EAAD:Nutrition2	0.043	0.038	1.113	0.266	−0.032	0.117
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Temporomandibular Joint Arthritis	(Intercept)	−7.973	1.273	−6.261	0.000	−10.469	−5.477
	HurricaneY	0.226	0.303	0.747	0.455	−0.367	0.819
	EAAD	0.222	0.060	3.678	0.000	0.104	0.341
	Nutrition2	1.841	1.280	1.438	0.150	−0.668	4.350
	SexM	1.448	0.265	5.467	0.000	0.929	1.967
	EAAD:Nutrition2	−0.038	0.063	−0.606	0.545	−0.162	0.085
		St. Dev.					
	Social Group	0.000					
	Matriline	0.751					
Trauma	(Intercept)	−3.757	0.487	−7.718	0.000	−4.711	−2.803
	HurricaneY	0.024	0.195	0.122	0.903	−0.358	0.405
	EAAD	0.125	0.029	4.305	0.000	0.068	0.181
	Nutrition2	0.604	0.502	1.202	0.229	−0.381	1.589
	SexM	0.003	0.158	0.019	0.985	−0.307	0.313
	EAAD:Nutrition2	−0.038	0.031	−1.202	0.229	−0.100	0.024
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Antemortem Tooth Loss	(Intercept)	−5.469	0.631	−8.662	0.000	−6.707	−4.232
	HurricaneY	0.113	0.231	0.488	0.625	−0.340	0.566
	EAAD	0.215	0.035	6.127	0.000	0.146	0.284
	Nutrition2	0.114	0.657	0.174	0.862	−1.174	1.402
	SexM	0.952	0.181	5.265	0.000	0.598	1.306
	EAAD:Nutrition2	0.006	0.038	0.153	0.879	−0.069	0.081
		St. Dev.					
	Social Group	0.560					
	Matriline	0.000					
Developmental Defect	(Intercept)	−0.532	0.878	−0.606	0.544	−2.252	1.188
	HurricaneY	0.107	0.410	0.260	0.795	−0.698	0.911
	EAAD	−0.965	0.378	−2.555	0.011	−1.705	−0.225
	Nutrition2	−2.789	0.906	−3.080	0.002	−4.564	−1.014

(Continues)

Pathology	Variable	Estimate	Std. Error	*z* value	Pr(> |z|)	0.025	0.975
	SexM	−0.055	0.337	−0.163	0.870	−0.716	0.606
	EAAD:Nutrition2	0.818	0.381	2.148	0.032	0.072	1.564
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Tibiofibular Synostosis	(Intercept)	−5.454	1.380	−3.952	0.000	−8.158	−2.749
	HurricaneY	−0.351	0.640	−0.549	0.583	−1.606	0.904
	EAAD	0.023	0.101	0.227	0.820	−0.175	0.221
	Nutrition2	−0.294	1.419	−0.207	0.836	−3.076	2.487
	SexM	0.502	0.464	1.082	0.279	−0.408	1.412
	EAAD:Nutrition2	0.072	0.106	0.680	0.497	−0.136	0.281
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Radioulnar Synostosis	(Intercept)	−5.724	1.253	−4.570	0.000	−8.178	−3.269
	HurricaneY	0.867	0.596	1.456	0.146	−0.301	2.035
	EAAD	0.119	0.068	1.765	0.078	−0.013	0.252
	Nutrition2	−1.571	1.440	−1.091	0.275	−4.394	1.252
	SexM	−0.169	0.527	−0.320	0.749	−1.202	0.864
	EAAD:Nutrition2	0.063	0.079	0.789	0.430	−0.093	0.218
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Divided Zygoma	NA						
Zygomatic Deformation	(Intercept)	−6.454	1.188	−5.435	0.000	−8.782	−4.127
	HurricaneY	−0.175	0.488	−0.360	0.719	−1.131	0.780
	EAAD	0.141	0.059	2.386	0.017	0.025	0.256
	Nutrition2	0.017	1.228	0.014	0.989	−2.391	2.425
	SexM	0.604	0.351	1.719	0.086	−0.085	1.292
	EAAD:Nutrition2	0.021	0.064	0.324	0.746	−0.105	0.147
		St. Dev.					
	Social Group	0.785					
	Matriline	0.530					
Osteoarthritis	(Intercept)	−2.932	0.367	−7.978	0.000	−3.652	−2.211
	HurricaneY	0.234	0.351	0.666	0.505	−0.454	0.922
	EAAD	0.124	0.023	5.384	0.000	0.079	0.169
	Nutrition2	−3.714	0.555	−6.697	0.000	−4.801	−2.627
	SexM	0.324	0.224	1.448	0.148	−0.115	0.763
	EAAD:Nutrition2	0.112	0.033	3.347	0.001	0.046	0.177
	BW.scaled	0.095	0.308	0.308	0.757	−0.510	0.700

(Continues)

Pathology	Variable	Estimate	Std. Error	*z* value	Pr(> |z|)	0.025	0.975
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Mechanical Enthesopathy	(Intercept)	−3.354	0.414	−8.106	0.000	−4.165	−2.543
	HurricaneY	−0.210	0.221	−0.949	0.343	−0.643	0.224
	EAAD	0.112	0.026	4.361	0.000	0.062	0.163
	Nutrition2	−0.817	0.440	−1.858	0.063	−1.679	0.045
	SexM	0.434	0.162	2.675	0.007	0.116	0.752
	EAAD:Nutrition2	0.062	0.029	2.160	0.031	0.006	0.119
		St. Dev.					
	Social Group	0.000					
	Matriline	0.651					
Inflammatory Enthesopathy	(Intercept)	−4.670	0.564	−8.288	0.000	−5.775	−3.566
	HurricaneY	0.057	0.220	0.261	0.794	−0.374	0.488
	EAAD	0.159	0.032	4.948	0.000	0.096	0.221
	Nutrition2	0.205	0.583	0.352	0.725	−0.937	1.347
	SexM	0.638	0.175	3.648	0.000	0.295	0.981
	EAAD:Nutrition2	0.016	0.035	0.459	0.646	−0.052	0.084
		St. Dev.					
	Social Group	0.000					
	Matriline	0.696					
Joint Disease	(Intercept)	−6.231	1.011	−6.164	0.000	−8.212	-4.250
	HurricaneY	−0.103	0.339	−0.304	0.761	−0.767	0.561
	EAAD	0.197	0.050	3.929	0.000	0.099	0.296
	Nutrition2	0.529	1.047	0.505	0.614	−1.524	2.582
	SexM	0.255	0.257	0.992	0.321	−0.249	0.760
	EAAD:Nutrition2	0.001	0.054	0.028	0.978	−0.104	0.107
	BW.Scaled	0.215	0.3109	0.694	0.487	−0.393	0.825
		St. Dev.					
	Social Group	0.526					
	Matriline	0.000					
Trauma: Healed	(Intercept)	−4.288	0.619	−6.923	0.000	−5.502	−3.074
	HurricaneY	−0.017	0.266	−0.065	0.948	−0.539	0.504
	EAAD	0.122	0.035	3.510	0.000	0.054	0.190
	Nutrition2	0.356	0.642	0.554	0.579	−0.902	1.614
	SexM	−0.042	0.210	−0.201	0.841	−0.455	0.370
	EAAD:Nutrition2	−0.029	0.038	−0.758	0.449	−0.104	0.046
		St. Dev.					
	Social Group	0.453					
	Matriline	0.000					

(Continues)

Pathology	Variable	Estimate	Std. Error	*z* value	Pr(> |z|)	0.025	0.975
Trauma: Healing	(Intercept)	−4.652	0.741	−6.281	0.000	−6.104	−3.201
	HurricaneY	0.074	0.285	0.259	0.796	−0.485	0.633
	EAAD	0.100	0.043	2.309	0.021	0.015	0.185
	Nutrition2	0.801	0.759	1.056	0.291	−0.685	2.288
	SexM	0.214	0.232	0.924	0.355	−0.240	0.669
	EAAD:Nutrition2	−0.048	0.047	−1.011	0.312	−0.140	0.045
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Trauma: No Healing	NA						
Trauma "1"	(Intercept)	−4.907	1.301	−3.772	0.000	−7.457	−2.357
	HurricaneY	0.058	0.353	0.165	0.869	−0.635	0.751
	EAAD	−0.005	0.106	−0.046	0.963	−0.213	0.203
	Nutrition2	0.652	1.317	0.495	0.621	−1.929	3.234
	SexM	0.034	0.302	0.111	0.911	−0.559	0.626
	EAAD:Nutrition2	0.060	0.109	0.552	0.581	−0.153	0.273
		St. Dev.					
	Social Group	0.000					
	Matriline	0.269					
Trauma "2"	(Intercept)	−4.261	0.612	−6.958	0.000	−5.462	−3.061
	HurricaneY	−0.157	0.307	−0.512	0.608	−0.758	0.444
	EAAD	0.110	0.035	3.107	0.002	0.041	0.180
	Nutrition2	0.287	0.638	0.450	0.652	−0.963	1.538
	SexM	0.146	0.228	0.642	0.521	−0.301	0.594
	EAAD:Nutrition2	−0.039	0.040	−0.968	0.333	−0.117	0.040
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Trauma "3"	(Intercept)	−5.124	0.852	−6.012	0.000	−6.794	−3.453
	HurricaneY	0.206	0.333	0.619	0.536	−0.447	0.859
	EAAD	0.141	0.046	3.099	0.002	0.052	0.230
	Nutrition2	0.501	0.890	0.563	0.574	−1.243	2.244
	SexM	−0.164	0.282	−0.581	0.562	−0.717	0.390
	EAAD:Nutrition2	−0.039	0.050	−0.773	0.439	−0.137	0.060
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Supernumerary Suture: Metopic	(Intercept)	−4.834	1.662	−2.908	0.004	−8.091	−1.576
	HurricaneY	1.922	0.501	3.834	0.000	0.939	2.904
	EAAD	−0.286	0.222	−1.288	0.198	−0.721	0.149

(Continues)

Pathology	Variable	Estimate	Std. Error	z value	Pr(> |z|)	0.025	0.975
	Nutrition2	−0.937	1.482	−0.632	0.527	−3.841	1.967
	SexM	1.252	0.468	2.675	0.007	0.335	2.170
	EAAD:Nutrition2	0.118	0.232	0.510	0.610	−0.336	0.572
		St. Dev.					
	Social Group	1.117					
	Matriline	0.789					
Supernumerary Suture: Bregma	(Intercept)	−1.964	0.832	−2.359	0.018	−3.595	−0.332
	HurricaneY	0.161	0.344	0.468	0.640	−0.513	0.835
	EAAD	−0.390	0.185	−2.104	0.035	−0.753	−0.027
	Nutrition2	−1.261	0.853	−1.479	0.139	−2.933	0.410
	SexM	0.217	0.281	0.771	0.441	−0.334	0.767
	EAAD:Nutrition2	0.246	0.190	1.292	0.196	−0.127	0.618
		St. Dev.					
	Social Group	0.000					
	Matriline	0.713					
Periosteal Reaction: Active	(Intercept)	−3.597	0.516	−6.970	0.000	−4.609	−2.586
	HurricaneY	−0.331	0.249	−1.328	0.184	−0.820	0.158
	EAAD	0.066	0.033	1.986	0.047	0.001	0.132
	Nutrition2	0.640	0.527	1.214	0.225	−0.393	1.674
	SexM	0.283	0.179	1.576	0.115	−0.069	0.634
	EAAD:Nutrition2	−0.049	0.037	−1.328	0.184	−0.122	0.023
		St. Dev.					
	Social Group	0.000					
	Matriline	0.425					
Periosteal Reaction: Mixed	(Intercept)	−5.046	0.931	−5.421	0.000	−6.871	−3.222
	HurricaneY	0.055	0.297	0.185	0.853	−0.526	0.636
	EAAD	0.102	0.054	1.899	0.058	−0.003	0.208
	Nutrition2	1.202	0.952	1.263	0.207	−0.664	3.067
	SexM	−0.021	0.244	−0.085	0.932	−0.500	0.458
	EAAD:Nutrition2	−0.066	0.058	−1.149	0.250	−0.179	0.047
		St. Dev.					
	Social Group	0.487					
	Matriline	0.683					
Periosteal Reaction: Healed	NA						
Periosteal Reaction “1”	(Intercept)	−3.496	0.525	−6.660	0.000	−4.525	−2.467
	HurricaneY	−1.219	0.471	−2.587	0.010	−2.142	−0.295
	EAAD	0.055	0.034	1.592	0.111	−0.013	0.122
	Nutrition2	−0.323	0.545	−0.592	0.554	−1.390	0.745

(Continues)

Pathology	Variable	Estimate	Std. Error	*z* value	Pr(> |z|)	0.025	0.975
	SexM	0.323	0.234	1.382	0.167	−0.135	0.781
	EAAD:Nutrition2	−0.012	0.040	−0.296	0.767	−0.090	0.066
		St. Dev.					
	Social Group	0.000					
	Matriline	0.552					
Periosteal Reaction "2"	NA						
Periosteal Reaction "3"	NA						
Cribra Orbitalia "1"	(Intercept)	−7.654	2.220	−3.447	0.001	−12.006	−3.302
	HurricaneY	−0.127	1.122	−0.113	0.910	−2.325	2.072
	EAAD	0.052	0.136	0.385	0.700	−0.214	0.318
	Nutrition2	−0.239	2.140	−0.112	0.911	−4.434	3.955
	SexM	1.991	1.099	1.812	0.070	−0.163	4.146
	EAAD:Nutrition2	0.029	0.149	0.194	0.846	−0.263	0.321
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Cribra Orbitalia "2"	(Intercept)	−11.513	5.058	−2.276	0.023	−21.428	−1.599
	HurricaneY	−0.195	1.121	−0.174	0.862	−2.391	2.001
	EAAD	0.313	0.201	1.557	0.120	−0.081	0.706
	Nutrition2	4.807	5.094	0.944	0.345	−5.177	14.790
	SexM	0.376	0.825	0.456	0.649	−1.240	1.992
	EAAD:Nutrition2	−0.241	0.211	−1.143	0.253	−0.654	0.172
		St. Dev.					
		0.000					
		0.000					
Crowding "1"	(Intercept)	−5.634	1.162	−4.850	0.000	−7.911	−3.357
	HurricaneY	−0.200	0.530	−0.378	0.705	−1.238	0.838
	EAAD	0.091	0.060	1.519	0.129	−0.026	0.209
	Nutrition2	−0.549	1.138	−0.482	0.630	−2.780	1.682
	SexM	0.265	0.373	0.711	0.477	−0.466	0.997
	EAAD:Nutrition2	0.069	0.067	1.026	0.305	−0.063	0.201
		St. Dev.					
	Social Group	0.667					
	Matriline	0.667					
Crowding "2"	(Intercept)	−7.584	2.723	−2.785	0.005	−12.920	−2.247
	HurricaneY	0.941	0.523	1.800	0.072	−0.084	1.965
	EAAD	0.128	0.147	0.871	0.384	−0.160	0.416
	Nutrition2	2.105	2.746	0.766	0.443	−3.277	7.486
	SexM	−0.170	0.518	−0.328	0.743	−1.184	0.845
	EAAD:Nutrition2	−0.118	0.154	−0.762	0.446	−0.420	0.185

(Continues)

Pathology	Variable	Estimate	Std. Error	*z* value	Pr(> |z|)	0.025	0.975
		St. Dev.					
	Social Group	0.801					
	Matriline	0.858					
Crowding "3"	NA						
Periapical Cavity: Granuloma or Acute Abscess	(Intercept)	−6.603	0.752	−8.781	0.000	−8.076	−5.129
	HurricaneY	0.430	0.300	1.433	0.152	−0.158	1.018
	EAAD	0.216	0.039	5.523	0.000	0.139	0.293
	Nutrition2	−0.690	0.796	−0.866	0.386	−2.250	0.871
	SexM	1.723	0.256	6.729	0.000	1.221	2.225
	EAAD:Nutrition2	0.051	0.044	1.146	0.252	−0.036	0.137
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Periapical Cavity: Chronic Abscess	(Intercept)	−7.059	1.000	−7.057	0.000	−9.019	−5.098
	HurricaneY	−0.296	0.426	−0.695	0.487	−1.131	0.539
	EAAD	0.199	0.049	4.080	0.000	0.103	0.294
	Nutrition2	−0.321	1.061	−0.303	0.762	−2.400	1.758
	SexM	1.500	0.327	4.585	0.000	0.859	2.142
	EAAD:Nutrition2	0.041	0.055	0.753	0.451	−0.066	0.149
		St. Dev.					
	Social Group	0.000					
	Matriline	0.000					
Periapical Cavity: Cyst	(Intercept)	−16.550	5.672	−2.918	0.004	−27.666	−5.433
	HurricaneY	−0.416	0.698	−0.595	0.552	−1.783	0.952
	EAAD	0.527	0.219	2.410	0.016	0.098	0.957
	Nutrition2	7.736	5.519	1.402	0.161	−3.082	18.554
	SexM	1.696	0.547	3.098	0.002	0.623	2.769
	EAAD:Nutrition2	−0.281	0.217	−1.294	0.196	−0.707	0.145
		St. Dev.					
	Social Group	0.760					
	Matriline	0.675					
Temporomandibular Joint Arthritis "1"	(Intercept)	−9.191	2.801	−3.281	0.001	−14.682	−3.701
	HurricaneY	−0.528	0.659	−0.800	0.424	−1.820	0.765
	EAAD	0.159	0.137	1.161	0.246	−0.109	0.426
	Nutrition2	1.869	2.786	0.671	0.502	−3.591	7.330
	SexM	1.680	0.531	3.162	0.002	0.639	2.721
	EAAD:Nutrition2	0.010	0.140	0.072	0.942	−0.264	0.285

(Continues)

Pathology	Variable	Estimate	Std. Error	*z* value	Pr(> |z|)	0.025	0.975
		St. Dev.					
	Social Group	0.817					
	Matriline	0.490					
Temporomandibular Joint Arthritis "2"	NA						
Temporomandibular Joint Arthritis "3"	NA						
Syndrome: Pathology	NA						
Syndrome: Congenital	NA						
Systemic Disease	(Intercept)	−7.850	1.542	−5.090	0.000	−10.873	−4.828
	HurricaneY	0.462	0.277	1.666	0.096	−0.082	1.005
	EAAD	0.247	0.071	3.497	0.000	0.108	0.385
	Nutrition2	3.499	1.548	2.260	0.024	0.465	6.533
	SexM	0.285	0.241	1.181	0.238	−0.188	0.758
	EAAD:Nutrition2	−0.164	0.073	−2.259	0.024	−0.307	−0.022
		St. Dev.					
	Social Group	0.492					
	Matriline	0.503					
Systemic Disease: Rheumatic	NA						
Systemic Disease: Infection	(Intercept)	−9.004	2.726	−3.303	0.001	−14.347	−3.661
	HurricaneY	0.634	0.382	1.662	0.097	−0.114	1.382
	EAAD	0.234	0.119	1.960	0.050	0.000	0.467
	Nutrition2	4.555	2.746	1.659	0.097	−0.827	9.936
	SexM	0.223	0.356	0.627	0.531	−0.475	0.922
	EAAD:Nutrition2	−0.248	0.124	−2.005	0.045	−0.491	−0.006
		St. Dev.					
	Social Group	0.539					
	Matriline	0.000					
Congenital Disorder	NA						