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# Is There a Link Between Matriline Dominance Rank and Linear Enamel Hypoplasia? An Assessment of Defect Prevalence and Count in Cayo Santiago Rhesus Macaques (*Macaca mulatta*)

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## Abstract

Linear enamel hypoplasias are developmental defects ranging in appearance from microscopic to macroscopic furrows in enamel that encircle the tooth crown. Environmental stressors, including lack of food and infectious diseases during early periods of development, are known to induce hypoplasias in human and nonhuman primates. Social correlates of hypoplasias have not been extensively studied, however. Here, we examined the relationship between matriline dominance rank and linear enamel hypoplasia prevalence (i.e., absence or presence) and count (the total number of hypoplasias observed) in free-ranging rhesus monkeys (*Macacca mulatta*) in Cayo Santiago, Puerto Rico. We sampled 86 female offspring from low-, mid-, and high-ranking matrilines. Our results show that although hypoplasia prevalence and count were numerically higher in the combined group of low-and mid-ranking matrilines than in high-ranking matrilines, this effect was not statistically significant. There was, however, a significant negative relationship between age and hypoplasia prevalence, as well as between age and mean number of enamel defects, likely due to the attrition and abrasion of enamel that wear away shallow defects as individuals age. Future studies would benefit from using large sample sizes and collecting detailed behavioral data to determine if and when social status mediates enamel defect formation.

**Keywords** Cercopithecinae · Dominant · Primates · Stress · Subordinate

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## Introduction

Stressors in the environment, including restricted food, extreme weather events, and disease during early stages of development, can leave enduring anatomical signs on individual organisms (Benderlioglu 2010). For example, such stressors, in both clinical and experimental settings, have been linked to linear enamel hypoplasia, a developmental defect of tooth enamel (reviewed in Goodman and Rose 1990). Hypoplasias form during the secretory phase of enamel formation, manifesting on the outer enamel surface (Goodman and Rose 1990). These defects form when ameloblasts, or enamel-forming cells, either slow or cease matrix secretion as they approach the enamel surface, resulting in a thinning of enamel that appears as a line or groove (Witzel *et al.* 2008). Enamel hypoplasias have been used as an indicator of physiological stress, because these lines or grooves remain permanently observable in teeth as long as they are not worn away through attrition or abrasion (Goodman and Rose 1990). Moreover, as enamel develops in an incremental fashion, the approximate developmental timing of hypoplasias can be gleaned from their location (Reid and Dean 2000).

Experimental research in nonhuman animals and clinical research in humans suggest that various stressors, such as fever (Kreshover and Clough 1953), infectious agents (Kreshover *et al.* 1953; Suckling *et al.* 1983), and deficient nutrition (Becks and Furata 1941; Guatelli-Steinberg and Benderlioglu 2006; May *et al.* 1993; Wolbach and Howe 1933), may result in enamel defects. Some studies show that individuals with hypoplasias have lower survival rates than those without enamel defects (Amoroso *et al.* 2014; Boldsen 2007; Goodman 1996; King *et al.* 2005). Other studies linked ecological conditions, such as seasonality in rainfall and/or food availability, to the formation of linear enamel hypoplasias in free-ranging primates (Hannibal and Guatelli-Steinberg 2005; Skinner 1986; Skinner and Hopwood 2004). However, a study of capuchin monkeys (*Cebus paella*) showed that variation of temperature in different habitats, not the annual rainfall, was the main factor in linear enamel hypoplasia expression (Chollet and Teaford 2010). Research on social correlates of hypoplasias remains scant, however. One exception is a study that investigated the relationship between enamel defects and social stress, as well as rainfall, nutrition, and disease in three juvenile wild chimpanzees (*Pan troglodytes*: Smith and Boesch 2015). Although internal enamel defects were associated with slow growth, prolonged maternal dependence, and some disease outbreaks, the authors concluded that the sample was too small to test the relationship between low maternal rank, indicating high maternal stress, and the developmental enamel defects in the offspring.

Dominance rank influences how much an animal is exposed to physical and psychosocial stressors, including access to food, increased aggressive encounters between low- and high-ranking individuals, and heightened risk of injuries (Koenig 2002, Sapolsky 2005). In rhesus macaques (*Macaca mulatta*), dominance rank in females is stable and inherited by female offspring (Bercovitch and Clarke 1995; Sade *et al.* 1988). This status is generally life-long, because females stay with the social group into which they were born (Berard 1999; Drickamer and Vessey 1973; Suarez-Jimenez *et al.* 2013). In contrast, male dominance hierarchies are less stable over the lifespan and are influenced by several factors, including other mature males within the natal group and reestablishment of rank for those who emigrate after puberty (Berard 1999; Drickamer and Vessey 1973; Sprague 1998; Suarez-Jimenez *et al.* 2013).

Studies conducted with the rhesus monkeys of Cayo Santiago indicate that most of the aggression observed in the colony is directed at low-ranking members of both sexes and initiated by high-ranking individuals (Kulik *et al.* 2015). In addition, infants of low-ranking macaque mothers are more often attacked and less protected than those of high-ranking females (Berman 1983, 1986). Low-ranking juvenile offspring also experience higher rates of aggression from their peers compared to high-ranking juveniles (Colvin 1983). Taken together, both low-ranking matrilines and their offspring appear to be exposed to greater social stress than high-ranking matrilines in this colony. Supporting evidence shows that lactating low-ranking Cayo females have higher baseline stress hormone, cortisol, compared to high-ranking individuals (Maestripieri and Georgiev 2016).

Lower-ranking monkeys' access to food may also be limited by frequent aggressive encounters with higher-ranking animals resulting from high density at the site. Because food bins are spatially more concentrated than natural food sources would be, food provisioning on Cayo Santiago restricts the home range of the monkeys (Hill 1999). Observations in the colony indicate that food provisioning results in more frequent encounters and more aggression compared to wild populations (Maestripieri and Hoffman 2011). In addition, low-ranking females are regularly chased away from food bins (Bercovitch and Berard 1993).

Because social behavior may either amplify or buffer social stress for individual organisms, examining potential connections between hypoplasias and social environment may provide important insights into the occurrence of enamel defects. We aimed to explore the relationship between matrilineal dominance rank and hypoplasia prevalence and defect counts in free-ranging rhesus monkeys (*Macaca mulatta*) in Cayo Santiago. We hypothesized that the social status of the monkeys would affect enamel defects. Given that low-ranking monkeys experience more aggression than high-ranking monkeys, and thus likely greater social stress, we predicted that females born to lower-ranking matrilines would have a higher prevalence of hypoplasias and a greater number of enamel defects than those born to higher-ranking matrilines.

## Methods

### Study Sample

We studied 86 free-ranging female rhesus monkey specimens obtained from the Caribbean Primate Research Center (CPRC). The animals lived on the island colony of Cayo Santiago in Puerto Rico during 1957–1986. A regular food provisioning program started in 1956 and random removal of individuals occurred year round in 1956–1972 (Hernandez-Pacheco *et al.* 2016). In 1984, a population control measure removed three social groups, reducing the Cayo population by ca. 50% (Hernandez-Pacheco *et al.* 2016). All rank categories in our sample were assigned after food provisioning had begun.

We used the mandibular third premolars (P3s) of females only. In the Cayo Santiago rhesus monkeys, the lower P3 is the tooth type most often affected by hypoplasia, with higher frequencies in females than in males (Guatelli-Steinberg 1998; Guatelli-Steinberg and Lukacs 1998). In our sample of 45 males, only 6 individuals were

affected with hypoplasias, preventing us from adequately testing our hypothesis in males. The fact that male dominance hierarchies are also less stable than those of females in this population further justified limiting the sample to females only.

To assign dominance categories, we used three sources: 1) CPRC database, 2) Chepko-Sade and Sade (1979), and 3) Missakian (1971). These sources split matrilines into low-, middle-, or high-ranking (see also Hoffman *et al.* 2010). Ranks of female matriline groups were based on the outcome of fights among females involving all matrilines (Chapke-Sade 1967; Missakian 1971). Although Cayo Santiago rhesus matrilines form stable linear dominance hierarchies (Hoffman *et al.* 2010), the relative ranks of matrilines may change when groups fission. If matriline rank changed as a result of group fissioning during the lower P3 crown formation period for an individual, we removed the individual from analysis. We added information about matriline dominance rank to the database after we recorded all data on enamel defects to ensure that observations of hypoplasias were "blind" with respect to matriline dominance rank.

Because our sample size was small, and because both middle- and low-ranking matriline groups were subordinate to the high-ranking group, we pooled low- and middle-ranking groups. From this point forward, we refer to this pooled group as "low- and mid-ranking." Pooling these groups increased the statistical power of our comparison to the high-ranking group.

We used age at death as a covariate in the analysis of the effect of dominance rank on hypoplasias. Individuals with enamel defects may be more susceptible to stress and/or may have experienced more stress, and therefore may die young (Goodman and Armelagos 1985; Goodman and Rose 1990). Alternatively, tooth wear through time may hinder the ability to detect minor defects, and may even obliterate them. This latter proposition is supported by an association between high tooth wear and low enamel defect prevalence (Guatelli-Steinberg and Lukacs 1998).

Owing to the lack of calcification data for rhesus monkeys, we used crown calcification times for lower P3s established from radiographic data for pig-tailed macaque (*Macaca nemestrina*): from 0.9 yr. (crown initiation) to 3.1 yr. (crown completion) (Sirianni and Swindler 1985). *M. nemestrina* is the most closely related species to rhesus macaques for which calcification data are available. The CPRC database includes month and year of birth for all of the monkeys in our sample, making it possible for us to ascertain each monkey's matriline rank during this time period. Initiation may be overestimated by radiographic data, as insufficient mineral is deposited at early stages of calcification to be detected by x-ray (Beynon *et al.* 1998). However, such overestimation is not problematic here, because the earliest-forming defects generally occurred after the first third of the crown height had formed (in the middle and cervical regions), and thus were established well after initiation (Guatelli-Steinberg 1998).

### Preliminary Analyses of Hypoplasias

D. Guatelli-Steinberg observed enamel defects under diffuse lighting with a second incandescent light oriented obliquely to the specimen (Goodman and Rose 1990; Lukacs 1989). A 10× hand lens aided in the identification of the defects. We assessed defects qualitatively, not quantitatively as in some recent studies (e.g., McGrath *et al.* 2018). We identified enamel defects by the course and positioning of suspected

hypoplasia lines or grooves on the enamel surface. Specifically, we determined lines or grooves that wrapped around the tooth surface, that were parallel to perikymata (i.e., showed evidence of having formed during the course of enamel development), and, appeared wider and/or deeper than surrounding perikymata as hypoplasia. We did not include a tooth in the sample, if one-half or more of its surface was not visible due to breakage, wear, or partial eruption. We assessed reliability on 30 specimens, which we scored twice, with 1 yr. between the assessments (Guatelli-Steinberg 1998). We used Cohen's κ statistic, designed for either nominal (Cohen 1960) or dichotomous variables (Fleiss 1973), to quantify scoring agreement. This statistic uses the proportions of variables in a study to calculate the degree of scoring agreement in excess of that expected by chance (Cohen 1960; Fleiss 1973). The κ value for scoring hypoplasias by tooth was 0.64; that for individuals with defects on an antimeric pair of teeth was 0.70 (see Guatelli-Steinberg 1998, 2000; Guatelli-Steinberg and Benderlioglu 2006). Both values indicate "substantial" agreement on the scale of Landis and Koch (1977).

We used three criteria for enamel defect assessment. The first method recommended by Hillson (1996) employs the most stringent criterion to examine the effect of stress on hypoplasias by limiting defect prevalence to antimeric pairs of teeth that are forming at the same time. This method considers the nature of stress as "systemic" (Berten 1895; Hillson 1996) and evaluates defects on unmatched pairs as "local" (Franz-Odendaal 2004; Goodman and Rose 1990). Limiting the assessment of hypoplasia prevalence to only those individuals with bilateral defects reduced the sample size to 16 specimens with hypoplasias, reducing the statistical power of our analyses. We employed a second method to apply the recommendation of Hillson (1996) in a limited way by investigating whether there are any rank differences in hypoplasia prevalence (i.e., absence or presence) on either P3s. Although the first two methods assess rank differences in whether or not an individual had tooth showing hypoplasias during growth, they do not consider the number of times this developmental defect occurred. The third method investigated whether there was a rank difference in the mean number of enamel defects. In this method, a specimen is counted as hypoplasia-affected if any single P3 of the lower mandible showed linear enamel defects (Fig. 1a, b). If both sides of any P3 in the lower mandible showed hypoplasia, we included only the side of the jaw with the highest number of enamel defects to avoid double counting. This method does not limit the assessment of hypoplasias to only antimeric teeth, nor does it make a distinction between "systemic" vs. "local" stress, but includes all incidents as indicative of enamel growth disruption. It is also consistent with other studies examining the relationship between hypoplasias and stress events (e.g., Franz-Odendaal 2004).

Hypoplasia asymmetry between antimeric teeth may be related to several factors. Asymmetries in timing may be involved if, for example, one tooth is still in the cuspal stage of enamel formation where surface defects do not appear, while the other has already transitioned to lateral enamel formation. In addition, it is possible that if left and right teeth of antimeric pairs initiate and/or complete formation at slightly different times, only one tooth may record the growth disruption as a linear defect. It is also conceivable that the disruption might have occurred on one side only, consistent with "local" stress, or that one side was better able to buffer against stressors than its antimere (Franz-Odendaal 2004). Environmental stress has an adverse effect on the physiological mechanisms that maintain this buffering capacity (i.e., developmental stability), resulting in asymmetrical manifestation of bilateral traits (reviewed in

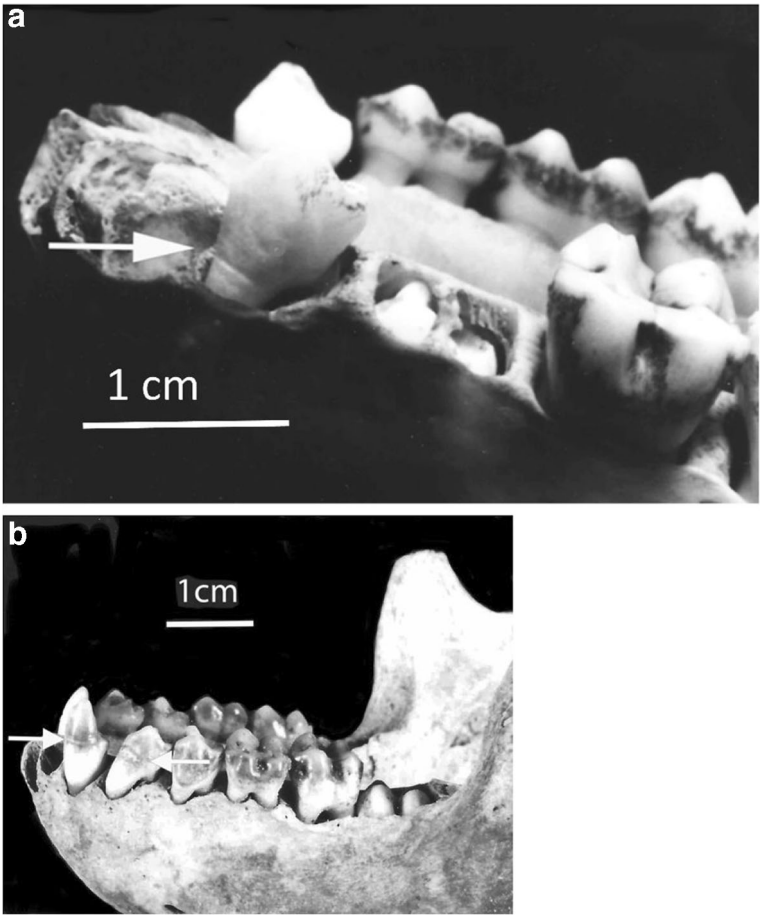


**Fig. 1** Two examples of enamel defects on P3s of Cayo Santiago rhesus monkeys whose skeletal and dental remains are housed at the Caribbean Primate Research Center. In **(a)**, an enamel defect is present on the mandibular left first premolar, which is in the process of erupting. In **(b)**, a less pronounced enamel defect is present on the mandibular left canine, possibly recording the same event that produced the hypoplasia in the premolar. White arrows point to enamel defects. Both specimens belong to low-ranking matrilines.

Benderlioglu 2010). The literature on dental fluctuating asymmetry, which demonstrates that antimeres can grow differently in response to stressors, substantiates this point (Barrett *et al.* 2012; Peiris *et al.* 2013). Even though the disruption can be less clearly attributed to a systemic cause, it may nevertheless indicate developmental instability in that enamel growth in one tooth of the pair did not proceed normally, or it may indicate physical trauma to the developing tooth (Franz-Odendaal 2004). An analysis of unilateral defects is also appropriate for the current paper, because a previous report with the same study population in Cayo Santiago indicated that female rhesus monkeys with lack of adequate food supply were twice as likely to have unilateral hypoplasia in P3s compared to the food-provisioned females (Guatelli-Steinberg and Benderlioglu 2006). That study, however, did not include dominance rank in its analyses, because rank categories were assigned by the CPRC (or other researchers) only after the food-provisioning program had begun in 1956.

### Statistical Analyses

We used SAS version 9.3 (SAS Institute Carry, NC) for all statistical analyses. A logistic regression analysis tested differences between high- and combined low- and-mid- ranking matrilines regarding hypoplasia prevalence for antimeric and unilateral defects. In these tests, binary response variables coded defect presence as "1" and absence as "0" on both members of antimeric teeth (method 1), and, then on any lower P3 (method 2). We compared high- vs. low- and mid-ranking matriline groups using 95% confidence limits for our independent variable, dominance rank, while age of the animals served as statistical control.

The next type of analysis involved comparison of enamel defects between matriline groups (method 3) employing a generalized linear model for count data. The response variable was the total number of defect counts on the lower P3 that showed the highest number of hypoplasias. Matriline rank with two levels—1) high- and 2) low- and mid-dominance—was the main explanatory variable with age serving as a covariate in this full model, which also included the intercept. We used goodness-of-fit and likelihood ratio analyses to test overall significance of a generalized linear model against a null one to ensure our data fit the full model (i.e., Cameron and Trivedi 1998). Chi-square goodness-of-fit test indicated that it was a good fit for our count data ($\chi^2 = 0.72$, df = 79, $P = 0.69$). All tests were one-tailed and results with $P \leq 0.05$ determined statistical significance.

**Data Availability** The datasets analyzed during the current study are available from the corresponding author on reasonable request.

## Ethical Note

This research adhered to the American Society of Primatologists principles for the ethical treatment of primates. We have no conflict of interest to declare.

## Results

### Dominance Rank and the Presence or Absence of Hypoplasias

A binary response (logistic regression) model testing defect presence/absence on antimeric teeth revealed no significant effect of matriline dominance rank (maximum likelihood $\chi^2 = 0.18$, df = 1, $P = 0.669$, age controlled; rate of hypoplasia-showing antimeric teeth high-rank = 15%; 5 out of 34; low-, mid-rank = 20%, 10 out of 51). Similarly, there were no significant rank effects when we tested unilateral presence of hypoplasias on either left or right lower P3 (maximum likelihood $\chi^2 = 0.24$, df = 1, $P = 0.471$, age controlled, Table I).

### Dominance Rank and Mean Enamel Defects

A maximum likelihood test showed that the main effect of matriline dominance rank regarding defect counts was not significant ($\chi^2_{1,\,79} = 3.58$, $P = 0.052$). Comparison of the

**Table I** Age, matriline dominance rank, and hypoplasia prevalence in rhesus macaques on Cayo Santiago, Puerto Rico 1957–1986

| | *N* | Mean age in years (SD) | Number of hypoplasia-affected individuals (percentage)[a] |
|---|---|---|---|
| High-ranking matriline | 29 | 7.45 (3.03) | 7 (24) |
| Low-, mid-ranking matriline | 57 | 8.59 (4.12) | 22 (39) |
| Total | 86 | | 29 (34) |

[a] A specimen is counted as hypoplasia-affected if any single P3 showed linear enamel defects

means indicated that the low- and mid-ranking group had numerically a higher mean number of enamel defects in lower P3s than the high-ranking group ($Mean_{low-,\ mid-\ rank} = 0.76$, SEM = ±0.17; $Mean_{high\ rank} = 0.50$, SEM = ±0.19, age controlled, Table II, Fig. 2).

In addition, age was a significant negative factor in predicting defect count in female rhesus monkeys. As animals aged, the total number of defects on lower P3s decreased (maximum likelihood $\chi^2_{1,79} = 4.87$, $P = 0.031$, $B = -0.07$). Parallel lines indicate this relationship was similar for all dominance categories (Fig. 3).

## Discussion

We found that although both defect prevalence and counts were numerically greater for the combined low- and mid-matriline group than for the high-matriline group of female Cayo Santiago monkeys, these differences were not statistically significant. Older individuals had significantly lower counts of enamel defects compared to younger ones regardless of their rank. This is likely due to the attrition and abrasion of enamel that increases with age, wearing away shallow enamel defects.

The present study had several limitations in comparing hypoplasia prevalence and enamel defect counts. The prevalence analysis that included monkeys with enamel defects on only antimeric lower P3s reduced the sample size to 16 out of 86 individuals (19% of the sample). Moreover, a further test of unilateral lower P3 defects to assess hypoplasia prevalence also yielded nonsignificant results potentially in part because only 34% of the specimens showed hypoplasia. Low statistical power may in part explain why defect count results were not statistically significant; alternatively, there may simply be no relationship between enamel defects and dominance rank. Although our results do not allow us to reject the null hypothesis, there was a pattern of higher average enamel defects

**Table II** Analysis of maximum likelihood estimates for enamel defect counts in rhesus macaques on Cayo Santiago, Puerto Rico 1957–1986

| Parameter | df | Estimate | Standard error | Lower confidence limit (95%) | Upper confidence limit (95%) | $\chi^2$ | *P* |
|---|---|---|---|---|---|---|---|
| Intercept | 1 | 1.71 | 0.30 | 1.12 | 2.3 | 32.71 | <0.0001 |
| Dominance rank | 1 | −0.47 | 0.25 | −0.87 | 0.99 | 3.66 | 0.052 |
| Age | 1 | −0.07 | 0.03 | −0.13 | −0.08 | 4.96 | 0.031 |

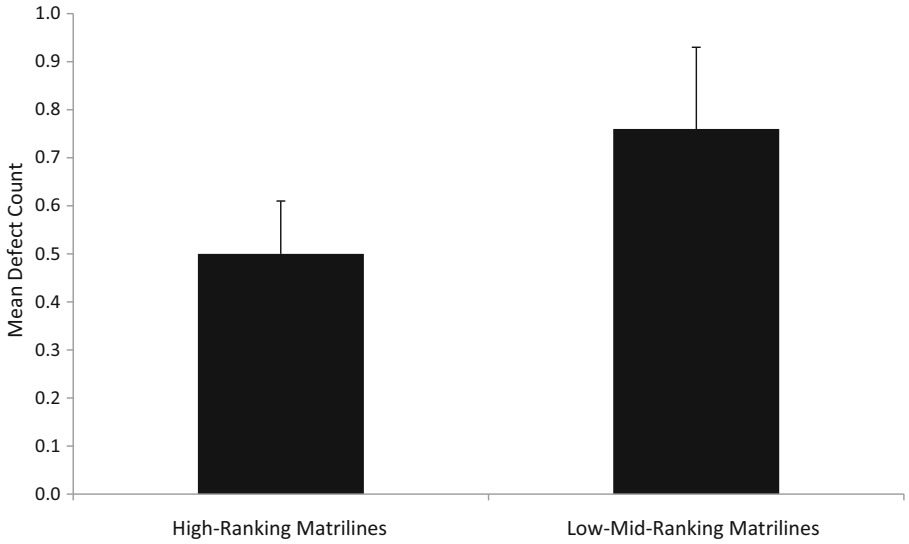


**Fig. 2** Mean count of enamel defects in lower P3s in high- and low- and mid-ranking matrilines of Cayo Santiago rhesus monkeys in 1956–1987. Error bars are the standard error of the mean.

and a higher hypoplasia prevalence on both antimeric and unilateral teeth in low- and mid-ranking matrilines than in the high-ranking ones. Twenty percent of the combined low-

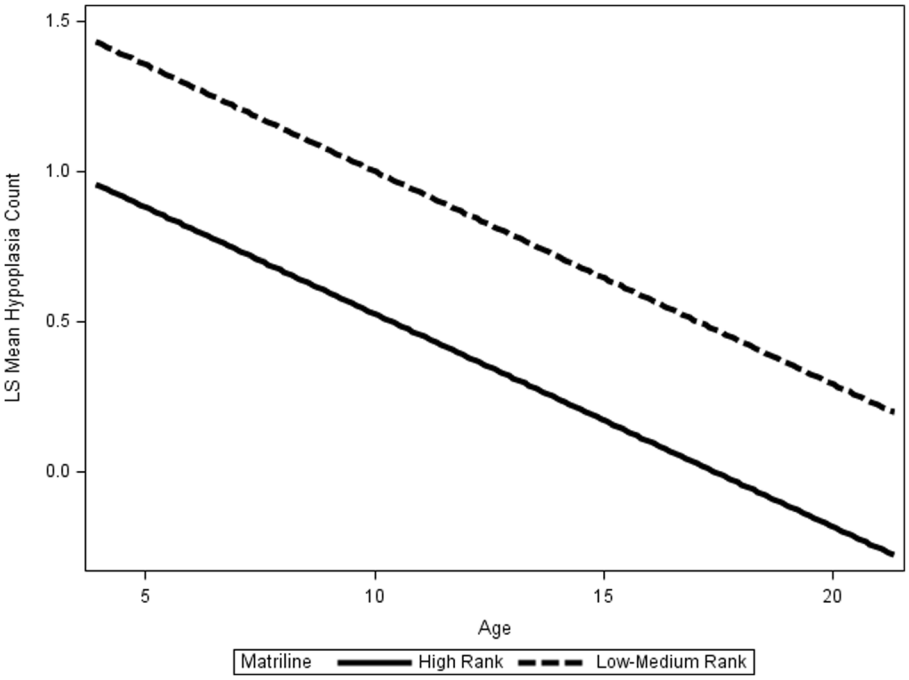


**Fig. 3** The association between age and defect counts in matriline ranks of Cayo Santiago rhesus monkeys during 1956–1987. LS-mean estimates are based on the linear combination of model parameters for (log) count data. LS means for dominance rank = −0.47 (SE = 0.25); age = −0.47 (SE = 0.03).

and mid-ranking monkey group showed hypoplasia on antimeres, in contrast to 15% of high-ranking females. Similarly, whereas 39% of the combined low- and mid-ranking group showed hypoplasia on any lower P3, the rate of hypoplasia prevalence in high-ranking matrilines was 21%. Future research would benefit from using large sample sizes on different species with clearly differentiated dominance categories in their social environment to further test the hypothesis that hypoplasias may be related to rank.

Foraging conditions at Cayo Santiago have not always been consistent. A food provisioning program commenced in 1956 and the population on the island grew at rates higher than that of wild rhesus monkeys (Hernandez-Pacheco *et al.* 2016). Furthermore, population density fluctuated as a result of colony management. For example, in 1972 and 1984, several social groups were removed and sent to different locations, thus reducing population density on the island (Hernandez-Pacheco *et al.* 2016). Such fluctuations might have affected foraging conditions and had varying effects on social stress that monkeys in our sample experienced. However, we have no data on which individuals were influenced by any of these changes, except that none of the removed subjects returned to the colony and that our study sample derived from the post-provisioning period.

Parasitic infections, other illnesses, or injuries might also have affected hypoplasia count and prevalence with respect to rank. A vaccination initiative for tetanus, which was a major cause of mortality in this colony, started in 1984. With the elimination of tetanus and population control measures, the health of the monkeys on the island significantly improved over time (Hernandez-Pacheco *et al.* 2016; Kessler and Rawlins 2016). Lack of detailed life history data on these monkeys prevents us from exploring the direct effect of these factors on hypoplasias in our sample. However, research on developmental defects of enamel in captive primates with known life histories may provide a valuable source of information on such environmental perturbations as diseases and injuries and their effect on enamel formation. Accentuated striae, internal enamel defects appearing as dark, microscopic bands that parallel the course of normal long-period increments in enamel, are particularly informative as indicators of metabolic disruption, because their timing of occurrence during development can be more precisely assessed than is possible for hypoplasias (Guatelli-Steinberg 2001). In one such study with western lowland gorillas, *Gorilla gorilla* accentuated striae in 14 permanent teeth of a captive juvenile female were associated with the exact days of this female's surgical procedures and follow-up clinic visits. Importantly, this was a "blind" study, in which the researchers examined the gorilla's medical records after they had determined the ages at which these accentuated striae had formed. Similarly, observations of accentuated striae coincided with three events in one captive female rhesus macaque: the birth of an infant, removal of all males from her social group, and maternal separation (Bowman 1991). Accentuated striae, in turn, are often, though not always (Witzel *et al.* 2008), associated with linear enamel hypoplasias (Condon 1981; Guatelli-Steinberg 2001; Hillson 1996; Smith and Boesch 2015). Over a 10-yr period, accentuated lines in three wild chimpanzees were linked to injuries and disease outbreaks, and in some cases, to linear enamel hypoplasias (Smith and Boesch 2015). New investigations on accentuated straie accompanied by separate hypoplasia assessments on the same specimens with historical records may provide valuable insights on differentiating social and environmental factors involving enamel defects in human and nonhuman animals.

Although we did not find direct evidence of the adverse effects of matriline rank on hypoplasia formation in rhesus monkeys, we did not employ direct physiological measures

to assess the level of social stress these monkeys experienced according to their rank. Therefore, we cannot refute the possibility that physiological responses to social group dynamics and stress might affect enamel formation resulting in hypoplasias, as appears to be the case with accentuated striae in gorillas (Bowman 1991) and rhesus macaques (Bowman 1991). It is also possible that social stress increases susceptibility to other stressors, such as disease, and that it is this increased susceptibility that may connect social stress to the formation of enamel defects. The evidence of social distress and decreased cellular immune response and heightened cancer risk in humans (Lutgendorf *et al.* 2005), and the higher number of illnesses and lower rate of wound healing in low-ranking baboons compared to high-ranking animals (Archie *et al.* 2012) both support this proposition. Further studies will benefit from collecting direct physiological measures with longitudinal data on stress events and hypoplasia expression in a variety of species to elucidate the potential mediating effect of social status on enamel defect formation across taxa.

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## References

Amoroso, A., Garcia, S. J., & Cardoso, H. F. V. (2014). Age at death and linear enamel hypoplasias: Testing the effects of childhood stress and adult socioeconomic circumstances in premature mortality. *American Journal of Human Biology, 26*(4), 461–468.

Archie, E. A., Altmann, J., & Alberts, S. C. (2012). Social status predicts wound healing in wild baboons. *Proceedings of the National Academy of Sciences of the USA, 109*, 9017–9022.

Barrett, C. K., Guatelli-Steinberg, D., & Sciulli, P. W. (2012). Revisiting dental fluctuating asymmetry in Neandertals and modern humans. *American Journal of Physical Anthropology, 149*, 193–204.

Becks, H., & Furata, W. (1941). Effect of magnesium deficient diets on oral and dental tissues: Changes in enamel structure. *Journal of American Dental Association, 28*, 1083–1088.

Benderlioglu, Z. (2010). Fluctuating asymmetry and steroid hormones: A review. *Symmetry, 2*(2), 541–553.

Berard, J. (1999). A four-year study of the association between male dominance rank, residency status, and reproductive activity in rhesus macaques (*Macaca mulatta*). *Primates, 40*, 159–175.

Bercovitch, F. B., & Berard, J. D. (1993). Life-history costs and consequences of rapid reproductive maturation in female rhesus macaques. *Behavioral Ecology and Sociobiology, 32*(2), 103–109.

Bercovitch, F. B., & Clarke, A. S. (1995). Dominance rank, cortisol concentrations, and reproductive maturation in male rhesus macaques. *Physiology & Behavior, 58*(2), 215–221.

Berman, C. M. (1983). Matriline difference and infant development. In R. A. Hinde (Ed.), *Primate social relationships* (pp. 132–134). Sunderland: Sinauer Associates.

Berman, C. M. (1986). Maternal lineages as tools for understanding infant social development and social structure. In R. G. Rawlings & M. J. Kessler (Eds.), *The Cayo Santiago macaques* (pp. 73–92). Albany: SUNY Press.

Berten, J. (1895). Hypoplasie des schmelzes (congenitale schmelzdefected, erosionen). *Deutsche Monatsschrift fur Zahnheilkunde, 13*, 425.

Beynon, A. D., Clayton, C. B., Ramirez Rozzi, F. V., & Reid, D. J. (1998). Radiographic and histological methodologies in estimating the chronology of crown development in modern humans and great apes: A review, with some applications for studies on juvenile hominids. *Journal of Human Evolution, 35*, 351–370.

Boldsen, J. L. (2007). Early childhood stress and adult age mortality: A study of dental enamel hypoplasia in the medieval Danish village of Tirup. *American Journal of Physical Anthropology, 132*(1), 59–66.

Bowman, J. E. (1991). Life history, growth and dental development in young primates: A study using captive rhesus macaques. PhD dissertation, University of Cambridge.

Cameron, A. C., & Trivedi, P. K. (1998). *Regression analysis of count data*. New York: Cambridge University Press.

Chepko-Sade, B. D., & Sade, D. S. (1979). Patterns of group splitting within matrilineal kinship groups: Study of social group structure in *Macaca mulatta* (Cercopithecidae, primates). *Behavioral Ecology and Sociobiology, 5*(1), 67–86.

Chollet, M. B., & Teaford, M. F. (2010). Ecological stress and linear enamel hypoplasia in *Cebus*. *American Journal of Physical Anthropology, 142*(1), 1–6.

Cohen, J. (1960). A measurement of agreement for nominal scales. *Educational Psychology and Measurement, 20*, 37–46.

Colvin, J. D. (1983). Proximate causes of male emigration at puberty in rhesus monkeys. *American Journal of Primatology, 4*(4), 328–328.

Condon, K. W. (1981). Correspondence of developmental enamel defects between the mandibular canine and first premolar. MA thesis, University of Arkansas.

Drickamer, L. C., & Vessey, S. H. (1973). Group changing in free-ranging male rhesus monkeys. *Primates, 14*, 359–368.

Fleiss, J. (1973). *Statistical methods for rates and proportions*. New York: John Wiley & Sons.

Franz-Odendaal, T. A. (2004). Enamel hypoplasia provides insights into early systemic stress in wild and captive giraffes (*Giraffa camelopardalis*). *Journal of Zoology, 263*, 197–206.

Goodman, A. H. (1996). Early life stresses and adult health: insights from dental enamel development. In C. J. K. Henry & S. J. Ulijaszek (Eds.), *Long-term consequences of early environment: growth, development, and the lifespan developmental perspective* (p. 163–182). New York: Cambridge University Press.

Goodman, A., & Armelagos, G. (1985) Disease and death at Dr. Dickson's mounds. *Natural History Magazine, 94*, 12–18.

Goodman, A. H., & Rose, J. C. (1990). Assessment of systemic physiological perturbations from dental enamel hypoplasias and associated histological structures. *Yearbook of Physical Anthropology, 33*, 59–110.

Guatelli-Steinberg, D. (1998). Prevalence and etiology of linear enamel hypoplasia in non-human primates. PhD dissertation, University of Oregon.

Guatelli-Steinberg, D. (2000). Linear enamel hypoplasia in gibbons (*Hylobates lar carpenteri*). *American Journal of Physical Anthropology, 112*(3), 395–410.

Guatelli-Steinberg, D. (2001). What can developmental defects of enamel reveal about physiological stress in nonhuman primates? *Evolutionary Anthropology, 10*(4), 138–151.

Guatelli-Steinberg, D., & Benderlioglu, Z. (2006). Brief communication: Linear enamel hypoplasia and the shift from irregular to regular provisioning in Cayo Santiago rhesus monkeys (*Macaca mulatta*). *American Journal of Physical Anthropology, 131*(3), 416–419.

Guatelli-Steinberg, D., & Lukacs, J. R. (1998). Preferential expression of linear enamel hypoplasia on the sectorial premolars of rhesus monkeys (*Macaca mulatta*). *American Journal of Physical Anthropology, 107*(2), 179–186.

Hannibal, D. L., & Guatelli-Steinberg, D. (2005). Linear enamel hypoplasia in the great apes: Analysis by genus and locality. *American Journal of Physical Anthropology, 127*(1), 13–25.

Hernandez-Pacheco, R., Delgado, D. L., Rawlins, R. G., Kessler, M. G., Ruiz-Lambides, A. V., et al (2016). Managing the Cayo Santiago rhesus macaque population: The role of density. *American Journal of Primatology, 78*(1), 167–181.

Hill, D. A. (1999). Effects of provisioning on the social behaviour of Japanese and rhesus macaques: Implications for socioecology. *Primates, 40*(1), 187–198.

Hillson, S. (1996). *Dental anthropology*. Cambridge: Cambridge University Press.

Hoffman, C. L., Ayala, J. E., Mas-Rivera, A., & Maestripieri, D. (2010). Effects of reproductive condition and dominance rank on cortisol responsiveness to stress in free-ranging female rhesus macaques. *American Journal of Primatology, 72*(7), 559–565.

Kessler, M. G., & Rawlins, R. G. (2016). A 75-year pictorial history of the Cayo Santiago rhesus monkey colony. *American Journal of Primatology, 78*(1), 6–43.

King, T., Humphrey, L. T., & Hillson, S. (2005). Linear enamel hypoplasias as indicators of systemic physiological stress: Evidence from two known age-at-death and sex populations from postmedieval London. *American Journal of Physical Anthropology, 128*(3), 547–559.

Koenig, A. (2002). Competition for resources and its behavioral consequences among female primates. *International Journal of Primatology, 23*(4), 759–783.

Kreshover, S. J., & Clough, O. W. (1953). Prenatal influences on tooth development: Artificially induced fever in rats. *Journal of Dental Research, 32*(4), 565–577.

Kreshover, S. J., Clough, O. W., & Hancock, J. A. (1953). Changes in the teeth of rabbits infected with vaccinia virus. *Journal of Dental Research, 32*(5), 661.

Kulik, L., Amici, F., Langos, D., & Widdig, A. (2015). Sex differences in the development of aggressive behavior in rhesus macaques (*Macaca mulatta*). *International Journal of Primatology, 36*, 764–789.

Landis, J. R., & Koch, G. G. (1977). Measurement of observer agreement for categorical data. *Biometrics, 33*(1), 159–174.

Lukacs, J. R. (1989). Dental paleopathology: Methods for reconstructing dietary patterns. In Y. Iscan & K. R. Kennedy (Eds.), *Reconstructing life from the skeleton* (pp. 261–286). New York: Alan R. Liss.

Lutgendorf, S. K., Sood, A. K., Anderson, B., McGinn, S., Maiseri, H., et al (2005). Social support, psychological distress, and natural killer cell activity in ovarian cancer. *Journal of Clinical Oncology, 23*, 7105–7113.

Maestripieri, D., & Georgiev, A. V. (2016). What cortisol can tell us about the costs of sociality and reproduction among free-ranging rhesus macaque females on Cayo Santiago. *American Journal of Primatology, 78*(1), 92–105.

Maestripieri, D., & Hoffman, C. L. (2011). Chronic stress, allostatic load, and aging in nonhuman primates. *Development and Psychopathology, 23*(4), 1187–1195.

May, R. L., Goodman, A. H., & Meindl, R. S. (1993). Response of bone and enamel formation to nutritional supplementation and morbidity among malnourished Guatemalan children. *American Journal of Physical Anthropology, 92*(1), 37–51.

McGrath, K., El-Zaatari, S., Guatelli-Steinberg, D., Stanton, M. A., Reid, D. J., et al (2018). Quantifying linear enamel hypoplasia in Virunga Mountain gorillas and other great apes. *American Journal of Physical Anthropology, 166*, 337–352.

Missakian, E. A. (1971). Genealogical and cross-genealogical dominance relations in a large group of free-ranging rhesus monkeys (*Macaca mulatta*) on Cayo Santiago. *American Journal of Physical Anthropology, 35*(2), 289.

Peiris, R., Satake, T., & Kanazawa, E. (2013). Fluctuating asymmetry of the permanent mandibular molars in a Japanese population. *Odontology, 101*, 15–21.

Reid, D. J., & Dean, M. C. (2000). Brief communication: The timing of linear hypoplasias on human anterior teeth. *American Journal of Physical Anthropology, 113*(1), 135–139.

Sade, D. S. (1967). Determinants of dominance in a group of free-ranging rhesus monkeys. In S. A. Altmann (Ed.), *Social communication among primates* (pp. 99–114). Chicago: University of Chicago Press.

Sade, D. S., Altmann, M., Loy, J., Hausfater, G., & Breuggeman, J. A. (1988). Sociometrics of *Macaca mulatta*: Decoupling centrality and dominance in rhesus monkey social networks. *American Journal of Physical Anthropology, 77*, 409–425.

Sapolsky, R. M. (2005). The influence of social hierarchy on primate health. *Science, 308*(5722), 648–652.

Sirianni, J., & Swindler, D. (1985). *Growth and development of the pigtailed macaque*. Boca Raton: CRC Press.

Skinner, M. F. (1986). Enamel hypoplasia in sympatric chimpanzee and gorilla. *Human Evolution, 1*, 289–312.

Skinner, M. F., & Hopwood, D. (2004). Hypothesis for the causes and periodicity of repetitive linear enamel hypoplasia in large, wild African (*Pan troglodytes* and *Gorilla gorilla*) and Asian (*Pongo pygmaeus*) apes. *American Journal of Physical Anthropology, 123*(3), 216–235.

Smith, T. M., & Boesch, C. (2015). Developmental defects in the teeth of three wild chimpanzees from the tai forest. *American Journal of Physical Anthropology, 157*(4), 556–570.

Sprague, D. S. (1998). Age, dominance rank, natal status and tenure among male macaques. *American Journal of Physical Anthropology, 105*, 511–521.

Suarez-Jimenez, B., Hathaway, A., Waters, C., Vaughan, K., Suomi, S. J., et al (2013). Effect of mother's dominance rank on offspring temperament in infant rhesus monkeys (*Macaca mulatta*). *American Journal of Primatology, 75*(1), 65–73.

Suckling, G., Elliott, D. C., & Thurley, D. C. (1983). The production of developmental defects of enamel in the incisor teeth of penned sheep resulting from induced parasitism. *Archives of Oral Biology, 28*(5), 393–399.

Witzel, C., Kierdorf, U., Schultz, M., & Kierdorf, H. (2008). Insights from the inside: Histological analysis of abnormal enamel microstructure associated with hypoplastic enamel defects in human teeth. *American Journal of Physical Anthropology, 136*(4), 400–414.

Wolbach, S. B., & Howe, P. R. (1933). The incisor teeth of albino rats and Guinea pigs in vitamin a deficiency and repair. *American Journal of Pathology, 9*(3), 275–294.