

Interpreting Sex Differences in Enamel Hypoplasia in Human and Non-Human Primates: Developmental, Environmental, and Cultural Considerations

DEBBIE GUATELLI-STEINBERG AND JOHN R. LUKACS
*Department of Anthropology, University of Oregon,
 Eugene, Oregon 97403-1218*

KEY WORDS enamel hypoplasia; sex differences; physiological stress; linear enamel hypoplasia; localized hypoplasia; environmental buffering

ABSTRACT The purpose of this review is to provide a synoptic, critical evaluation of the evidence of, and potential etiological factors contributing to, sex differences in the expression of enamel hypoplasia (EH). Specifically, this review considers theoretical expectations and empirical evidence bearing on two central issues. The first of these is the impact of a theorized inherent male vulnerability to physiological stress on sex differences in EH. The second issue is the potential contribution to sex differences in EH of intrinsic differences in male and female enamel composition and development. To address this first issue, EH frequencies by sex are examined in samples subject to a high degree of physiological stress. Based on the concept of inherent male vulnerability (or female buffering), males in stressful environments would be expected to exhibit higher EH frequencies than females. This expectation is evaluated in light of cultural practices of sex-biased investment that mediate the relationship between environmental stress and EH expression. Defects forming prenatally afford an opportunity to study this relationship without the confounding effects of sex-biased postnatal investment. Data bearing on this issue derive from previously conducted studies of EH in permanent and deciduous teeth in both modern and archaeological samples as well as from new data on Indian schoolchildren. To address the second issue, fundamental male-female enamel differences are evaluated for their potential impact on EH expression. A large sex difference in the duration of canine crown formation in non-human primates suggests that male canines may have greater opportunity to record stress events than those of females. This expectation is examined in great apes, whose canines often record multiple episodes of stress and are sexually dimorphic in crown formation times. With respect to the first issue, in most studies, sex differences in EH prevalence are statistically nonsignificant. However, when sex differences are significant, there is a slight trend for them to be greater in males than in females, suggesting a weak influence of greater male vulnerability. Cultural practices of sex-biased investment in children appear to have greater impact on EH expression than does male vulnerability/female buffering. With respect to the second issue, sex differences in the composition and development of enamel were reviewed and determined to have limited or unknown impact on EH expression. Of these factors, only the duration of crown formation was expected to affect EH expression by sex within the great apes. The data support an association between higher defect counts in the canines of great ape males relative to those of females that may be the result of longer crown formation times in the canines of great ape males. This review concludes with an assessment of the nature of the evidence currently available to examine these issues and suggests future avenues for research focused on elucidating them. *Yrbk Phys Anthropol* 42:73-126, 1999.

© 1999 Wiley-Liss, Inc.

TABLE OF CONTENTS

| | |
|---|-----|
| Background | 76 |
| Sex differences in enamel hypoplasia: A brief history | 76 |
| Enamel hypoplasias: Types, etiologies, and relationships to other stress indicators | 77 |
| Theoretical Foundations and Expected Data Trends | 80 |
| The question of enhanced female buffering | 80 |
| The question of intrinsic differences in the enamel of males and females | 83 |
| Sex chromosomes, enamel thickness, and crown size | 83 |
| Sex differences in amelogenin genes and their expression | 84 |
| Sex differences in the canalization of tooth development | 85 |
| Sex differences in duration of crown calcification | 86 |
| The relevance of intrinsic factors | 87 |
| Examination of the Evidence | 88 |
| Is there evidence of enhanced female buffering in enamel hypoplasia studies? | 88 |
| Biologically "stressed" samples | 88 |
| Low and very low birth weight neonates | 88 |
| Living samples with independent evidence of stress | 89 |
| Archaeological samples with independent evidence of stress | 90 |
| Slave populations: Afro-American and Roman | 93 |
| Historical, almshouse, and poorhouse samples | 94 |
| Low socioeconomic status groups | 94 |
| Cadavers, indigents, and unclaimed bodies | 95 |
| Summary of evidence for enhanced female buffering in biologically "stressed" groups | 96 |
| Is there evidence of female buffering from LHPC studies? | 97 |
| Samples with unknown levels of stress | 100 |
| Skeletal series with unknown levels of stress | 100 |
| Amerindian native skeletal series | 100 |
| American colonists | 102 |
| Australia | 102 |
| South Asia | 102 |
| Italy | 102 |
| Marianas Archipelago | 103 |
| Maya | 104 |
| Summary of skeletal series with unknown stress levels | 105 |
| Living samples | 106 |
| Non-human primates | 108 |
| EH incidence | 108 |
| Defect counts | 112 |
| Discussion | 114 |
| Interpreting sex differences in EH: Deciduous teeth | 114 |
| Interpreting sex differences in EH: Permanent teeth | 115 |
| Conclusions | 117 |
| Acknowledgments | 118 |
| Literature Cited | 119 |

Enamel hypoplasia (EH) has been defined as a deficiency "... in enamel thickness resulting from physiological perturbations (stress) during the secretory phase of amelogenesis [the process of enamel formation]" (Goodman and Rose, 1990, p. 59). Physiologi-

cal insults occurring during the period of enamel matrix formation can disturb enamel production or cause the death of ameloblasts (enamel-producing cells), resulting in macroscopic surface defects (Ten Cate, 1998). Defects of dental enamel are extensively used

by anthropologists in reconstructing the general health status of prehistoric skeletal samples (Goodman and Rose, 1990; Goodman and Skinner, 1992). The prevalence of a particular defect, known as linear enamel hypoplasia (LEH), often constitutes the primary basis for inferences regarding the relative frequency of "systemic growth disruptions" in one sample versus another. However, reporting the prevalence of enamel defects for a sample as a whole tends to conceal variation within the population by sex or by socio-economic status.

In the anthropological and clinical literature, EH prevalence is not regularly reported by sex. When sex differences in EH frequencies are found, they are often interpreted to reflect fundamental differences between the sexes in access to essential resources, including differential levels of parental care, nutrition, or health care. This review questions such simple and direct inferences because the complex etiology of stress markers, such as EH, often renders direct inference unreasonable. In addition, our survey of clinical and anthropological literature reveals that many studies are based on small sample sizes, lack descriptions of cultural contexts, do not differentiate between enamel defect types, or report results in very different formats, thereby impeding comparability and limiting insights into differential patterning of stress by sex. In most cases where direct inferences are made between the incidence of stress markers and the general health of a group, the complex and interacting factors of innate susceptibility (heterogeneity in frailty), oscillating cultural influences, and sample biases are not fully considered.

A recent discussion of how sex differences in human skeletal remains shed light on gender hierarchies in past peoples confronted the problem of interpreting differences in prevalence of EHs. "The interpretation of sex differences is complicated by the possibility that the sexes may be inherently different either in the degree to which the body buffers stress episodes, or the manner in which stress is recorded" (Cohen and Bennett, 1993, p. 284). Lower stress rates in females are frequently interpreted as "natural," while lower stress rates in males are

often regarded as reflecting culturally favored treatment of male children. Cohen and Bennett stated that "...much more work is needed before we can establish the 'natural' background pattern of sex differences in stress markers against which culturally induced patterns of stress can be measured" (Cohen and Bennett, 1993, p. 284). We concur with this assessment and have undertaken this review in order to investigate the interaction of biological and cultural variables in producing patterns of variation by sex in the expression of EH.

Broadly stated, this review concerns factors that influence sex differences in the expression of EH in human and non-human primates. More specifically, we ask two central questions:

- (1) Are there fundamental (innate) differences in the frequency of enamel defects in females and males that originate from one sex being better buffered biologically than the other?
- (2) Are there intrinsic differences in the enamel composition and/or enamel development of males and females that may cause differences in EH expression?

To address these questions, this study employs a broad comparative perspective, encompassing data from human and non-human primates, utilizing data from anthropological as well as clinical sources, and incorporating evidence from both deciduous (primary) and permanent (secondary) dentitions.

This review is divided into four main parts. Following the introduction is a background section that provides an overview of previous research on sex differences in EH and of different types of EH and their etiologies. The second part of the paper considers the theoretical underpinnings for the two central questions: that of enhanced female buffering against environmental insult and that of sex differences in intrinsic attributes of enamel. Based on these theoretical considerations, we describe expected trends in the data regarding sex differences in EH expression. In the third part, previously published clinical and anthropological data, as well as new data presented from the authors' research on Indian schoolchildren and non-

human primates, are examined for their conformity with expected data trends. The final part of the paper discusses the results of part three, evaluates the evidence in relation to the paper's two central questions, discusses the limits of present knowledge bearing on these questions, and suggests avenues for future research.

BACKGROUND

Sex differences in enamel hypoplasia: A brief history

A comprehensive history of research on EHs in biological anthropology, clinical investigations, and epidemiology is readily available (Goodman and Rose, 1990), and is not repeated here. The goal of this section is to trace the beginning and subsequent development of interest in documenting sex differences in EH prevalence.

The first volume widely viewed as a textbook in dental anthropology, *The Human Masticatory Apparatus* by Klatsky and Fischer (1953), is primarily devoted to issues related to evolution of the masticatory system and the decline of oral health with the rise of civilization. While this early literary landmark bridges the fields of dentistry and biological anthropology, EH as a form of developmental defect is mentioned only once in passing.

The classic landmark publication, entitled *Dental Anthropology*, includes a chapter devoted to the dental pathology of early human populations (Brothwell, 1963). Three pages discuss the prevalence of EH in archaeological skeletal samples and in fossil hominids. While differences in EH frequency are described in terms of cultural and diachronic patterns, the question of sex differences in EH is not considered or recommended as a focus for future study. In the late 1960s, neither the general survey of paleopathology by Kerley and Bass (1967), nor the paleopathological analyses of disease in ancient Nubia (Armelagos, 1969) or the prehistoric Valley of Tehuacán (Anderson, 1965) include observations or discussions of hypoplastic enamel defects.

Swärdstedt's (1966) analysis of medieval Swedish dental remains from Västerhus is the first seminal anthropological study of sex differences in EH prevalence. His meticu-

lous analysis of enamel defects includes results obtained by subdividing the sample into socio-economic levels (Länderman, Höldermand, Slave) and sex groups. This exemplary study is often cited by recent students of enamel defects as discovering an inverse association between LEH prevalence and socio-economic status (Goodman, 1998), and for documenting that males had higher frequencies of LEH than females.

Several synthetic reviews of bioarchaeology and paleopathology appeared in the 1980s, but sex differences in EH was not a central topic. For example, Buikstra's and Cook's (1980) critical history of American paleopathology includes a succinct yet optimistic section on dental defects; however, their potential in evaluating sex differences in levels of stress is not mentioned. A few years later, in Huss-Ashmore and colleague's (1982) review of nutritional inference in paleopathology, only three sources are cited on the topic of variation in EH by sex: one on modern Guatemalan school children (Infante and Gillespie, 1974), one on medieval Swedish (Swärdstedt, 1966), and one on prehistoric Native Americans (Goodman, 1976). The association between diet and developmental disturbances in teeth was reviewed by Rose and co-workers in 1985, and attention was directed to the potential value of enamel defects as retrospective markers of stress.

"Enamel defect analysis has the singular advantage that patterns of childhood nutritional inadequacy can be analyzed by sex when the permanent dentitions of adults are used." (Rose et al., 1985, p. 299)

These researchers present a synopsis of the hypoplasia data from Dickson Mounds (Goodman et al., 1980), showing that females have higher rates than males in the Late Woodland Period, but that in later periods the sexes are approximately equally affected. Differences in enamel micro-defects by sex are also discussed and interpreted to indicate sex-specific weaning practices (Rose et al., 1981).

An extensive summary review of methods for bioarchaeological interpretation of subsistence economy and behavior, by Larsen (1987), refers to a single source on sex differences in EH: Goodman et al.'s (1980)

analysis of the Dickson Mound skeletal series. In the early 1990s, three substantive review articles on EH were published as chapter contributions to: (1) the *Yearbook of Physical Anthropology* (Goodman and Rose, 1990), (2) *Advances in Dental Anthropology* (Goodman and Rose, 1991), and (3) *The Skeletal Biology of Past Peoples* (Skinner and Goodman, 1992). The main foci of these contributions include: (a) the history of enamel defect research, (b) methodological and technical problems of analysis, (c) defect etiology, and (d) establishing the chronology and timing of defect formation. The topic of sex differences in EH is addressed only in the *Yearbook* article, and only briefly, recounting Swärdstedt's (1966) analysis of the medieval Swedish sample from Västerhus (Goodman and Rose, 1990), and El-Najjar and co-workers' (1978) finding of higher enamel defect prevalence among white males than females in the Hamman-Todd skeletal series (Goodman and Rose, 1990). More recently, Larsen (1995) reviewed biological changes that accompany the shift to agriculture, noting that while dental caries rates increase generally, females are commonly affected more than males. However, although EH prevalence also increases with the adoption of agriculture (Larsen, 1995), the possibility of a differential impact by sex is not addressed.

Most recent overviews of the biological anthropology of past populations provide informative introductions to the analysis and etiology of EHs and their value in bioarchaeology (Larsen, 1997), human osteology (Mays, 1998), and paleopathology (Roberts and Manchester, 1995). The emphasis in such reviews is the relationship between changing prevalence of LEH and changes in subsistence patterns or differences in socioeconomic or ritual status. Sex differences in LEH prevalence are not usually addressed in these volumes or in recent dental anthropology publications (Hillson, 1996; Schultz et al., 1998). By contrast, Larsen's (1997) "Bioarchaeology" briefly summarizes sex differences in prevalence of enamel defects in archaeological skeletal series, and characterizes them as highly variable. This two paragraph review relies heavily on the review articles by Goodman and Rose (1990, 1991)

and on selected anthropo-epidemiological studies. Increased attention to sex differences in EH frequencies also appears in recently published epidemiological reports of LEH prevalence among historical skeletal samples (Saunders and Keenleyside, 1999), and living populations in Brazil (Santos and Coimbra, 1999) and China (Zhou and Corrucini, 1998), where LEH frequencies are reported by sex, and the problems of explaining sex differences in prevalence are discussed.

In summary, most anthropological literature devoted to stress markers lacks synoptic, critical evaluation of data bearing on the issues of sex differences in EH, and though recent research in dental anthropology frequently reports sex differences in EHs, most reviews of bioarchaeology, paleopathology, and paleodiet do not address the topic.

Enamel hypoplasias: Types, etiologies, and relationships to other stress indicators

The Fédération Dentaire Internationale Defects of Dental Enamel (DDE) Index (1982,1992) classifies hypoplastic defects into four types: pits (type 3), horizontal grooves (type 4), vertical grooves (type 5), or altogether missing enamel (type 6). Pits may be single or multiple; multiple pits may be scattered or distributed in horizontal bands (DDE Index, 1992). Horizontal defects can take the form of a single sharp line on the crown surface, a single groove or furrow, or in some cases the crown surface may be "ridged across with a washboard effect" (Hillson, 1986). Horizontal hypoplastic defects are also termed linear EH, LEH, (e.g., Goodman and Rose, 1990) or furrow-form defects (Hillson and Bond, 1997). Vertical defects are rare, with an etiology that is not completely understood (Eckhardt, 1992); they are manifested by females with X-linked amelogenesis imperfecta (Alvesalo, 1997). Missing enamel or "plane form defects" (Hillson and Bond, 1997) include localized hypoplasia of the primary canine (LHPC) (Skinner, 1986b) and interproximal contact hypoplasia (IPCH) (Lukacs, 1999a). Figure 1 contains examples of LEH and LHPC defects in human and non-human primates.

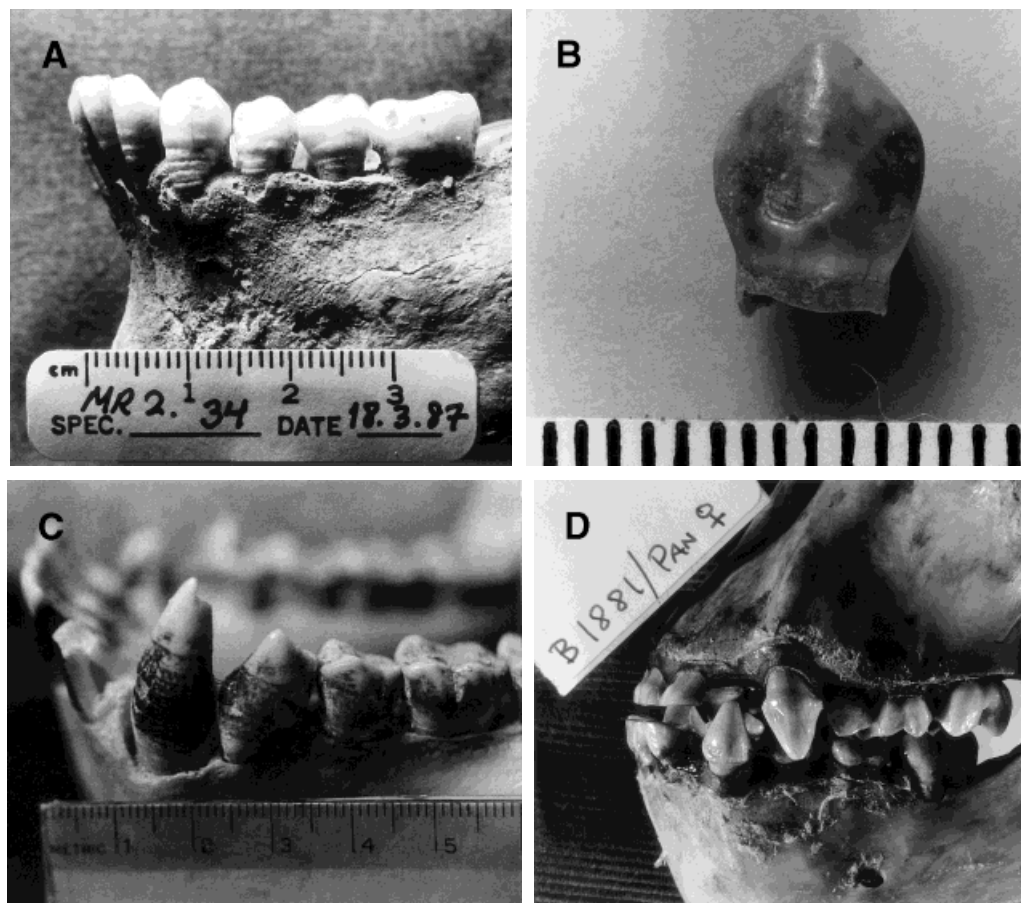


Fig. 1. Types of enamel hypoplasia. (A) MR 2. 34. multiple linear hypoplastic defects in an adolescent (14–16 years of age) from the Chalcolithic cemetery at Mehrgarh, Baluchistan Province, Pakistan (ca. 4500 BC). (B) MR 3. 158B. Localized hypoplasia of the primary canine, a plane form type of defect, on the labial surface of the deciduous left mandibular canine. Specimen is from a child (9–14 months of age) from the

Neolithic cemetery at Mehrgarh (ca. 6000 BC). (C) MCZ 37358. Eight linear defects (LEHs) are on the lower left canine of this *Pongo pygmaeus* male. Specimen is from the Museum of Comparative Zoology at Harvard University. (D) B 1881. Localized hypoplasia of primary canines in *Pan*. Note defects on both maxillary and mandibular deciduous canine teeth. From the Hamman-Todd Collection, Cleveland Museum of Natural History.

There is a strong causal link between systemic physiological stress, such as malnutrition or febrile disease, and EH (Ten Cate, 1994). Experimentation on animal models, including dogs (Mellanby 1929), mice (Kreshover 1942), rabbits (Kreshover et al. 1954), and sheep (Suckling et al. 1986), has demonstrated this connection. Mellanby (1929) discovered that vitamin D and vitamin A deficiencies could cause EH in dogs. Kreshover (1942) showed that mice infected with tuberculosis had abnormal ameloblast morphology and hypoplastic teeth. Suckling et al.

(1986) found ameloblastic changes associated with EH in sheep experimentally infected with nematode parasites.

For many years, clinical studies have found associations between hypoplastic defects and dietary deficiency and/or childhood infectious diseases. For example, in the 1700s, Bunon found enamel defects in the unerupted teeth of children who had died from rickets, scurvy, measles, or smallpox (Hillson 1992). Sarnat and Schour (1941) were able to link hypoplasias with episodes of disease in half of their sample of individu-

als from the Chicago area. More recently, Sweeney et al. (1971) found that 75% of Guatemalan children hospitalized for malnutrition had major groove defects. Goodman et al. (1991) conducted a prospective supplementation study in the 1980s in Mexico in which the control group (which did not receive nutritional supplements) had twice the LEH frequency of the supplemented group.

While nutritional and disease stress can produce EH, so can a plethora of other conditions (Cutress and Suckling, 1982; Pindborg, 1982). Nikiforouk and Fraser (1981) proposed that hypocalcemia (low serum concentration of calcium) is the unifying etiological factor among these causes of EH. However, nearly 100 conditions are associated with enamel defects (Cutress and Suckling, 1982) and not all of these are attributable to hypocalcemia. Unless fluorosis is severe, it generally causes enamel opacities, not hypoplastic defects (Goodman and Rose, 1990). Fluoride concentrations in drinking water at or higher than 4 ppm have been correlated with population increases in severe fluorosis with concomitant enamel pitting (Driscoll et al., 1983; Driscoll et al., 1986).

The multifactorial etiology of EHs has led some (e.g., Neiberger, 1990) to question the utility of EH as an indicator of nutritional stress. However, many of the etiological factors implicated in EH are uncommon systemic or inherited conditions (Cutress and Suckling, 1982), and are thus unlikely to explain the high incidences of EH observed in some human populations. The most common causes of EH in disadvantaged populations are likely to be metabolic disturbances resulting from nutritional deficiency and/or infectious disease (Skinner and Goodman, 1992). In supplementation studies (Goodman et al., 1991; May et al., 1993), it has not been possible to identify specific nutritional deficiencies resulting in EH, nor has it been possible to identify the relative importance of the synergism between malnutrition and disease (Goodman and Rose, 1991). Thus, EH is best viewed as a nonspecific indicator of systemic physiological stress occurring during dental crown formation (Goodman and Rose, 1990). Because enamel does not remodel, hypoplastic

defects provide an indelible record of metabolic disruptions (Goodman and Rose, 1990), provided that minimally worn teeth are observed. LEH, in particular, is a sensitive nonspecific indicator of systemic stress. A systemic cause is implied if linear defects can be matched on teeth forming at the same time, especially if the teeth are antimeres (Goodman and Rose, 1990).

Deciduous EHs are frequently grouped into two fundamentally different categories: LEH and localized hypoplasia of primary canines (LHPC). LHPC is easily differentiated from LEH by several criteria: (a) form of expression (nonlinear, usually circular or ovoid), (b) distribution in the dental arcade (restricted to deciduous canine teeth), and (c) location on the dental crown (confined to labial surface) (Lukacs and Walimbe, 1998). By contrast, in LEH, one or more horizontal grooves or a linear array of pits representing a deficiency of enamel formation are present on the outer enamel surface (Goodman and Rose, 1990, 1991; Hillson and Bond, 1997). LEH is more frequently observed in permanent than in deciduous teeth, and adjacent teeth are often affected. The clear distinction in the appearance of these two types of enamel defects and their dissimilar distribution in the dental arcade suggest that significantly different etiological pathways are involved. This review will consider these two categories of enamel defects separately.

LHPC, commonly associated with very low-birth weight (<1500 grams) and pre-term births, has a multifactorial etiology (Seow, 1992). Systemic illnesses causing LHPC may have an underlying common cause in calcium deficiency (but not necessarily with low blood serum calcium concentrations: Seow, 1992). Symmetrically distributed LHPC defects imply systemic illness, while isolated defects indicate trauma to the alveolus, such as occurs in infant exploratory mouthing behavior (Skinner and Hung, 1989) or with the practices of laryngoscopy and endotracheal intubation in contemporary populations (Seow, 1992). LHPC has also been associated with nutritionally disadvantaged groups, low sunlight, and low levels of retinol (Skinner et al., 1994). The etiology of IPCH, another "plane-form" defect, appears to involve physical contact

between developing tooth germs resulting from inadequate developmental space (Lukacs, 1999a).

EH incidence frequently parallels the incidence of other stress indicators. Goodman et al. (1992) report on children from Solis, Mexico in which LEH is associated with height, weight, and socio-economic status, presumably because of low intake of animal protein and poor dietary diversity. Goodman and colleagues (1980) found that the high incidence in LEH in the Middle Mississippian phase was paralleled by chronic hyperostosis (indicative of iron deficient anemia). In prehistoric skeletal remains from Schleswig-Holstein (North Germany), Harris lines and EH were associated, but not in a one-to-one correspondence (Kühl, 1992). In this same study, cribra orbitalia and EH showed no congruence of severity. Mays (1995) found a positive association between LEH and Harris lines in juvenile skeletons from a British medieval site; however, there was no association between these two stress markers in adults, presumably because of bone remodeling. May et al. (1993) suggest that enamel formation may be more sensitive than bone mineralization to changes in nutritional status. These authors found that Guatemalan children ingesting more of a nutritional supplement exhibited less LEH than children who received less supplement, but the two supplementation groups did not differ in their ossification status.

Recently, however, reports of high LEH prevalence in association with high economic or ritual status (Cucina and İşcan, 1997; Stodder, 1997) and tall adult stature (Lukacs and Pal, 1993) suggest that direct inference of health status from stress markers may be unjustified. The relationship between an individual's skeletal and dental stress markers and the nature of the causal stresses is more complex and synergistic than generally assumed (Boldsen, 1998). Inferences regarding the health of past populations are also limited by the osteological paradox (Wood et al., 1992): Groups experiencing severe stress might show low values of stress indicators simply because individuals may not survive long enough to record stress events in their teeth and bones.

THEORETICAL FOUNDATIONS AND EXPECTED DATA TRENDS

The question of enhanced female buffering

The question of whether males or females display increased sensitivity to environmental stress is an enduring and intriguing topic. In his first Epistle, Peter advocated an already commonplace attitude when he admonished men to, "live considerately with your wives, bestowing honor on the woman as the weaker sex." However, since the early 1800s, demographic and biological evidence of greater female adaptability has been forthcoming. For example, the shorter male lifespan observed by Quetelet in 1835 was interpreted as only partly due to societal roles. Sex differences in resistance to infectious diseases (Stini, 1985) and to parasite loads (Brabin, 1990) have been documented, females showing greater resistance. The X chromosome may be partly responsible for this difference (Gobel and Konopka, 1973).

The prevalent and consensus view among anthropologists is that females are better buffered against environmental stress, an opinion based on theoretical and empirical evidence. The evolution of better buffered females is adaptive given the demanding nature of reproductive functions with which the female body must cope: pregnancy, lactation, and child rearing (Stini, 1985). Research on sex differences in response to nutritional stress has led Stini (1969, 1972, 1975, 1978, 1985) to conclude that the long-term effects of protein deprivation are more pronounced in males, and that a decrease in sexual dimorphism results. Experimental protein deprivation in rhesus macaques revealed significant differences in metabolic efficiency, with females being much more efficient and gaining more weight than males when fed a low protein diet (Riopelle, 1990). The common Western cultural prejudice is that women are more vulnerable to famine than men, yet there is little physiological justification for this viewpoint (Rivers, 1988). Laboratory experiments on animals (Hoyenga and Hoyenga, 1982) and famine demographics (Ali, 1984) show that females are better able to cope with nutritional deprivation and to survive episodes of famine than

TABLE 1. Female biological superiority: Fact or fiction?¹

| Investigators | Data source | Support for female biological superiority? |
|-------------------------------------|---|--|
| Brooks and Brooks, 1994 | Longevity: life insurance records/skeletal data | None |
| Katz and Armstrong, 1994 | Marriage practices and evolution of X chromosome | None |
| Koenigsberg and Grant, 1994 | Skeletal size/shape; living stature | Yes |
| Lazenby and Pfeiffer, 1994 | Cortical bone remodeling: male (localized), female (systemic) | Qualified yes |
| Lukacs and Guatelli-Steinberg, 1994 | No significant differences in prevalence by sex; Mean defects/tooth: 5/36 comparisons M > F, 1/36 comparisons F > M, 30/36 n.s. | Qualified yes |
| Rathbun, 1994 | Skeletal pathology in 19th century African-American slaves; M > F for hypoplasia, Harris lines | Qualified yes |
| Roberts and Margerison, 1994 | Medieval skeletal data and historical records | No results |
| Saunders, 1994 | Sub-adult sex ratios from tooth size | Qualified yes |
| Stini, 1994 | Male mortality ~2× female, reversal later in life | None |
| Stinson, 1994 | Height and weight as Z scores (CDC/WHO ¹ standards), 20 growth studies | Yes |
| Van Gerven & Sheridan, 1994 | F less LEH and better growth rate despite greater nutritional stress; reversal later in life | Qualified yes |

¹ CDC: Center for Disease Control and Prevention; WHO: World Health Organization.

males. A hypothesized linkage between sex chromosomes, hormones, and energy balance may explain the observation that females display greater resistance to famine than males (Hoyenga and Hoyenga, 1982).

Stinson's (1985) review of pre- and postnatal evidence for sex differences in environmental buffering examined growth and body composition of stressed and nonstressed groups, and mortality and morbidity data at different points in the life cycle. This critical review found most types of evidence, including conventional stress markers such as EH, to be inconclusive on the issues of enhanced female buffering. The only support for increased female buffering was limited, and came from studies of late prenatal growth and mortality.

Since Stinson's review, numerous investigators have addressed the issue of better biological buffering among females. Many lend support to the concept of significant female buffering under conditions of environmental stress, among nutritionally stressed adults in rural Malawi (Dettwyler, 1992), among agriculturalists of the Peruvian Andes (Leonard, 1991), and among members of the ill-fated Donner Party (Grayson, 1990).

In 1994, Patty Stuart-Macadam organized a symposium at the American Association of Physical Anthropologists entitled,

"Female Biological Superiority: Fact or Fiction?," the purpose of which was to bring together a wide array of recent data that bear upon the question of sex differences in environmental buffering. Eleven papers were presented in this symposium. The issue of females being biologically able to better endure environmental stress was addressed using data from growth rates and stature, cortical bone remodeling, sub-adult sex ratios, morbidity, mortality, and longevity (Table 1). Two of eleven papers presented evidence supporting better environmental buffering of females, five presentations provide qualified support for it, and four studies yielded evidence neither for nor against the hypothesis.

In retrospect, that the results of this symposium were equivocal could have been predicted on the basis of the widely varying theoretical perspectives and sources of data offered by the participants. The inconclusive outcome of this symposium parallels the lack of consensus regarding sex differences in environmental buffering found by Stinson (1985) in her review.

Neither Stinson (1985) nor the 1994 symposium provided a comprehensive assessment of the evidence of female buffering from EH studies. Stinson (1985) cites only five studies dealing with dental stress indica-

tors. The 1994 symposium included just one EH study. In this review, we survey a large body of clinical and anthropological literature on EH for evidence of enhanced female buffering. According to Stinson (1985), male morbidity should be higher than that of females in more stressful environments. Males would also be expected to be less buffered against nutritional stress than females. We define "stressful" samples to include those in which there is either direct or indirect evidence of nutritional and/or disease stress. Direct indicators include historical or clinical records (e.g., infants with low birth weights) documenting physiological stress, nutritional analyses revealing caloric and/or protein deficiencies, and high population incidences of stress indicators other than EH. Indirect evidence of physiological stress derives from samples for which a high level of stress can be inferred: slave populations, almshouse and poorhouse samples, groups with low socio-economic status, and impoverished groups. Stinson (1985) makes the very important point that one cannot assume that "stressful" environments are equally experienced by the sexes, especially postnatally, when preferential investment in one sex (usually males) may occur. Thus, where possible, we report any evidence of sex-biased investment in offspring that may impact the expression of EH.

Stinson (1985) also states that the strongest evidence of male "vulnerability" to environmental insult derives from studies of prenatal mortality and growth, presumably because there is no opportunity for preferential treatment of male offspring. However, her review does not consider that deciduous teeth can provide evidence of stress during the prenatal and perinatal periods. Defects that form prenatally allow examination of the possibility of enhanced female buffering without the confounding effects of differential investment in the sexes.

A theoretical basis for predicting sex bias in enamel defects of primary teeth was advanced by Infante and Gillespie (1974) in their report on prevalence of linear EH in the deciduous anterior teeth of Guatemalan schoolchildren. The "theory of nutritional association" holds that "at birth, boys in general weigh more, have more muscle mass,

are developmentally behind, and have less subcutaneous fat than girls. Thus, boys would be expected to have greater nutritional requirements and less caloric reserves than girls at birth" (Infante and Gillespie, 1974, p. 1057). Since many defects of deciduous enamel appear to form within the first few months of birth, the theory of nutritional association predicts a higher prevalence of defects among boys than among girls. Though linear EH and localized hypoplasia of primary teeth may have different proximal etiologies, the ultimate cause of these lesions involves common underlying factors. Therefore we maintain that the nutritional association hypothesis serves as a valid one for interpreting sex differences in prevalence of deciduous EHs.

One possibility is that evidence of greater male vulnerability derives from a size effect: large body size itself, with greater caloric demand, may make an individual more vulnerable to nutritional stress, whether male or female. Yet, there is some evidence to the contrary. In a prospective study of Mexican children, some of whom were chronically undernourished, Scholl et al. (1979) did not find an association between body length at 6 months of age and the subsequent development of protein-energy malnutrition. It is important, too, to consider that surveys worldwide (Eveleth and Tanner, 1990) have shown preadolescent males to be only slightly taller and heavier than preadolescent females at the same age. Intra-sex variation is consistently much larger than small mean differences between males and females. Male-female differences in vulnerability might have more to do with body composition (muscle versus fat) than with absolute differences in stature or weight.

Based upon the foregoing theoretical considerations, we expect that across EH studies, there will be a predictable tendency in the distribution of enamel defects by sex under conditions of physiological stress. Increasing disparities in hypoplasia prevalence, with males exhibiting higher EH frequencies than females, are expected to be associated with increasing levels of environmental stress. This expectation can be best evaluated for samples in which there is evidence of environmental stress other than

the evidence from EH, and for samples in which male and female children do not differ substantially in their access to essential resources such as food and health care.

As has been noted, the available EH data are limited by a number of factors, including insufficient descriptions of cultural contexts, sources of environmental stress, and the types of enamel defects observed. We review the EH literature and present new findings bearing on the issue of enhanced female buffering in the third part of the paper.

The question of intrinsic differences in the enamel of males and females

This section examines how the following factors, which vary by sex, may influence sex differences in EH expression: sex chromosomes, enamel thickness, crown size, amelogenin gene expression, genetic canalization, and crown calcification timing. Deciduous and permanent dentitions in human and non-human primates are considered. Particular attention is focused on the canine, as it is usually the most sexually dimorphic tooth in both human (Garn et al., 1964, 1967; Kieser, 1990) and non-human primates (Greenfield, 1992, and Plavcan, 1990) and is one of the most susceptible teeth to EH (humans: Goodman and Rose, 1990; hominoids: Skinner, 1986a).

Sex chromosomes, enamel thickness, and crown size. Alvesalo et al. (1985, 1987, and 1991), Alvesalo and De La Chappelle (1981), Alvesalo and Tammissalo (1981), Alvesalo (1997), and Mayhall et al. (1998) have related metric differences in dental hard tissues to the specialized growth influences of X and Y chromosomes. Alvesalo and Tammissalo (1981) found that enamel is thinner in the permanent maxillary central incisors and canines of human 45,X (Turner's syndrome) females relative to normal male and female controls (46,XY and 46,XX), whose enamel thickness is approximately equal. 47,XXX human females have thicker enamel than normal males and females but have the same dentinal thickness as control females (Alvesalo et al., 1987), indicating that the X chromosome is active in amelogenesis but has no effect on the growth of dentin. These results, in addition to results from 45,X

females (Alvesalo and Tammissalo, 1981) and 47,XYY males (Alvesalo et al., 1985, and Mayhall et al., 1998), strongly suggest that X increases metric enamel growth somewhat more effectively than does the Y chromosome. 47,XXY (Klinefelter's syndrome) males have enamel thickness greater than that of either control females or control males and dentinal thickness that is both greater than that of normal females and smaller than that of normal males (Alvesalo et al., 1991). The authors conclude that amelogenesis is promoted by both X and Y chromosomes, but that sexual dimorphism in average tooth size is caused by a promoting effect of the Y chromosome on dentinal growth. Canines appear to be relatively stable in their development regardless of chromosomal abnormalities (Alvesalo et al. 1991). For example, there is no difference in either enamel or dentinal thickness in the maxillary canines of 47,XXY males and normal males.

The relatively greater influence of the X chromosome on amelogenesis appears to be congruent with research on the amelogenin gene (described in more detail below). The Y-chromosome amelogenin gene is expressed at 10% of the level of the X-chromosome amelogenin gene (Salido et al., 1992). The X-chromosome's differential impact on amelogenesis is also evident in individuals with X-linked amelogenesis imperfecta. In males with this condition, the enamel is thin and smooth, while in females, the enamel is of close to normal thickness with vertical grooves (Alvesalo, 1997). X-linked amelogenesis imperfecta has been associated with a nonsense mutation in the X-chromosome's amelogenin gene (Aldred et al., 1992, and Lench et al., 1994).

The implications of this body of research for sex differences in environmentally induced EH in humans are somewhat obscure. Thin enamel is considered to be less vulnerable to developmental disruption than thicker enamel (Skinner and Goodman, 1992, based on Suga, 1989; see also Boyd's comment in Backman 1997). In terms of normal males and females, however, there is only a minimal difference in maxillary canine enamel thickness. The sum of maxillary canine mesial and distal enamel layers is

2.40 mm (on average) for females and 2.31 mm (on average) for males (Alvesalo, 1997). If tall crowns are more susceptible to LEH (as suggested by Goodman and Armelagos, 1985), one might expect male crowns to be more susceptible than female crowns, particularly with respect to canines [which show 3–9% dimorphism in MD and BL diameters (Kieser, 1990) and 9% sexual dimorphism with respect to mandibular canine crown height (Plavcan, 1990)]. However, Alvesalo's research indicates that sexual dimorphism in the size of the crown is related not to enamel differences but to differences in the thickness of dentin, with normal males having thicker dentin than females (Alvesalo, 1997). Thus, it is not likely that taller male crowns would be more vulnerable to disruption than smaller female crowns as a result of enamel thickness differences.

Male deciduous teeth are also larger than female deciduous teeth (Alvesalo and Kari, 1977; De Vito and Saunders, 1990), although the degree of sexual dimorphism in size varies across populations (De Vito and Saunders, 1990). We have found no data describing to what extent these differences result from dentinal or enamel thickness differences.

Certainly, many primate species exhibit considerable sexual dimorphism in their canine crown heights (Plavcan, 1990). Walker (1984), however, found that the major difference in "bulk" between sexually dimorphic male and female primate canines is in the amount of dentin, rather than the amount of enamel. Thus, as in humans, enamel thickness does not appear to be related to canine sexual dimorphism.

Sex differences in amelogenin genes and their expression.

Amelogenins are "a heterogeneous group of low-molecular weight (20–30 kDa) hydrophobic proteins" (Ten Cate, 1998), making up 90% of the protein in the enamel matrix (Gibson et al., 1997) and functioning to control the growth and orientation of enamel crystallites (Fincham and Simmer, 1997). Sex differences in amelogenin genes, their transcription, and translational products are described below.

Lau et al. (1989) first identified amelogenin genes on both the X and Y chromo-

somes of humans (on the distal short arm of X, and near the centromere of Y). Nakahori et al. (1991) found 88.9% homology between the X and Y nucleotide sequences, while Salido et al. (1992) found that the X and Y amelogenin protein coding regions are highly conserved, with a similarity index of 93–100% (untranslated regions are less conserved). Salido et al. (1992) also found that the Y-amelogenin gene was transcribed at only 10% of the level of the X-amelogenin gene. These differences in the level of transcription, according to the authors, may reside in X-Y differences in the amelogenin gene's promoter sequences, which show only 80% base-pair similarity. Chen et al. (1998) believe that differences in the level of expression are related to sex chromosome differences in upstream (relative to amelogenin loci) regulatory regions. An additional transcriptional difference is that the splicing pattern in Y-derived mRNA differs from that of X-derived mRNA (Salido et al., 1992).

The protein products — amelogenins — themselves exhibit sexual dimorphism. Fincham et al. (1991) reported that the enamel protein complex of males contains amelogenin proteins that are not present in females. The authors wonder if these molecular differences may mean that "male enamel" has different properties than "female enamel."

It is not currently clear to what extent these differences in the level of amelogenin gene expression or in the amelogenins themselves affect normal male and female enamel differently. It does not seem that enamel thickness is affected (see above discussion). However, there may be differences in enamel quality, as amelogenin is believed to control initial enamel mineral spacing and growth (Fincham and Simmer, 1997). Potential differences resulting from the properties of "male" amelogenin, however, would not impact those primate species lacking Y-chromosome amelogenin genes: baboons, patas monkeys, green monkeys, talapoin monkeys, and tamarins (Sasaki and Shimokawa, 1995). Besides humans, great apes, capuchins, and Japanese, rhesus, and crab-eating macaques all are known to have amelogenin genes located on both X and Y chromosomes.

Sex differences in the canalization of tooth development.

Goodman and Armelagos (1985) consider differences in developmental stability to best explain inter-tooth variation in the expression of EH. They point out that patterns of stability as indicated by variation in size, shape, developmental timing, and fluctuating asymmetry indicate that the more distal teeth within each tooth class are least canalized. According to Goodman and Armelagos (1985), polar teeth within each tooth class are most vulnerable to LEH, while nonpolar teeth, presumably under weaker genetic control as the concentration gradient of the morphogen becomes attenuated, are able to respond to environmental perturbations by slowing their rates of development and decreasing their size. McKee and Lunz (1990) have shown that in individuals with maxillary central incisor LEH, I² and M² are greatly reduced in size; M² shows no signs of hypoplasia but shows this size reduction. These data lend support to the argument by Goodman and Armelagos (1985) that environmental stress causes proximal teeth in a tooth class to become hypoplastic, while distal teeth become reduced in size.

Can this argument be applied to the sexes? If, for example, the teeth of females show greater canalization than those of males, one would expect (based on Goodman and Armelagos, 1985) the more canalized teeth to be more susceptible to EH under equivalent levels of physiological stress. Garn et al. (1966) found that males have greater fluctuating asymmetry in their permanent dentition than females, a finding these researchers attributed to the fact that males are hemizygous (hence less canalized) for X-linked traits. Fluctuating asymmetry is considered to result from developmental noise (Waddington, 1957). Resistance to developmental noise and genetic canalization are "different levels of the same adaptation (Van Valen, 1962)."

Research subsequent to Garn et al. (1966), however, has not demonstrated a consistent sex difference in fluctuating asymmetry. Harris and Nweeia (1980), for example, found that female Ticuna Indians of Colombia had greater asymmetry in the MD dimension than did males (BL dimensions were not significantly different between the sexes).

The authors argue that a cultural/environmental explanation of this result is not warranted as the greater asymmetry of females occurs from birth (with the onset of I1/M2 calcification) throughout the developmental period. Townsend (1981) reviews several studies which have shown greater fluctuating asymmetry in males (Townsend and Brown, 1980; Garn, Lewis and Kerewsky, 1966 and 1967), greater fluctuating asymmetry in females (Harris and Nweeia, 1980; Niswander and Chung, 1965) and no sex difference (Perzigian, 1977; Bailit et al., 1970). Kieser and Groeneveld (1998) find nonsignificant sex differences in dental fluctuating asymmetry in the offspring of either smoking or nonsmoking parents (as a group, children of smokers have higher levels of fluctuating asymmetry than children of nonsmokers).

Townsend (1981) finds no fluctuating asymmetry difference by sex in the deciduous dentition of Australian aboriginals; neither is there a sex difference in fluctuating asymmetry in the deciduous dentition of South Australian school children (Townsend and Farmer, 1998) or Dominican mulatto children (Townsend and Garcia-Godoy, 1984). Townsend (1981) explains that his previous finding (Townsend and Brown, 1980) of greater asymmetry in the permanent dentition of males may relate to prolonged crown formation in male permanent teeth, affording more opportunity for environmental disturbances to affect crown growth. He argues that deciduous teeth do not show sex differences in fluctuating asymmetry because they are not sexually dimorphic in their developmental timing. This argument, based on the duration of crown formation, is distinct from an argument based on sex differences in canalized development.

Other indications of canalization such as variability in tooth size (Garn et al. 1964), shape (Garn et al. 1967), and timing (Anderson et al. 1975) indicate that males are more variable than females. Examining the deciduous teeth of children in the Burlington Growth Study, De Vito and Saunders (1990) found that with the exception of the canine, the crown diameters of females exhibited significantly greater variability than those of males. However, it is not clear that vari-

ability within a population is a direct indicator of the genetic control of development within an individual. Harris and Bailit (1988) found that Solomon Islands females exhibited greater odontometric correlation than males, indicating that female dental development is more "integrated" than that of males. This result points to a greater degree of canalization in females, but it is not clear that the result can be generalized to other populations. Like fluctuating asymmetry, sex differences in inter-tooth odontometric correlation may vary across groups.

In non-human primates, fluctuating asymmetry has shown interesting patterns associated with canine teeth. Nass (1982) finds that male deciduous teeth in Japanese macaques are more asymmetric than those of females, a pattern she attributes to greater canalization of female teeth. However, she also finds that male canines are much less asymmetric than their female counterparts, perhaps, she suggests, because of the relatively greater importance of male canine function. Manning and Chamberlain (1993) found that across species, fluctuating asymmetry in male canine height increases with canine size dimorphism. In their view, this correlation results because sexually selected characters are subject to directional selection with attendant increases in homozygosity and "genomic" stress. These authors also found that in species with high levels of canine sexual dimorphism and frequent and intense inter-male competition, there is a negative correlation between mean canine height and fluctuating asymmetry. Perhaps, they argue, in these species, canine symmetry functions to signal male quality and female canine symmetry is part of a correlated response [as first proposed by Greenfield (1992) for interspecific variation in female canine size]. Plavcan (1998) finds that female canine size differences across species are only partly explained by a correlated response: there is an independent effect of selection for the development of canine weaponry in species in which females exhibit "high-intensity, noncoalitional competition." There may be independent selection pressures acting on female canine symmetry as well.

Manning and Chamberlain (1994) found that canines of male lowland gorillas are sensitive to environmental stress, as measured by an association between canine fluctuating asymmetry and environmental deterioration. Because the canines of female lowland gorillas do not show this association, the authors argue that sexually selected structures are sensitive to environmental stress. If this finding is replicated in studies of other non-human primates, it might suggest that male canines are more likely than female canines to exhibit EH under stressful conditions. However, the environmental "stresses" that cause fluctuating asymmetry may differ from those that cause EH.

From the foregoing discussion, it is evident that in neither human nor non-human primates do sex differences in dental canalization show a consistent pattern. Thus, no consistent sex difference in EH can be expected on this basis.

Sex differences in the duration of crown calcification. Sex differences in the duration of crown formation for human permanent and deciduous teeth appear to be minimal. In contrast to large sex differences in root formation time, crown formation times in the permanent teeth of human males and females are practically equal (Nolla, 1960; Moorrees et al., 1963a). Moss and Moss-Salentijn (1976), based on data by Moorrees et al. (1963a), point out that of all human permanent teeth, the canine tooth shows the largest sex difference in crown calcification times, with male canines taking 70 days longer to form than those of females.

Other studies, while they have examined sex differences in dental development, have not been able to track canine crown formation differences between males and females because subjects' canine teeth had already begun to calcify (Demirjian and Levesque, 1980; Thompson et al., 1975; Anderson et al., 1975; Harris and McKee, 1990). With respect to deciduous canines, male and female crown formation times are again close to being equal (Moorrees et al., 1963b). For both males and females, the average length of time from completion of the deciduous canine's cusp outline to crown completion is

a little over 6 months (Moorrees et al., 1963b). Sex differences in human crown calcification times, then, at a maximum of 70 days for the permanent canine, are not likely to result in differences in the expression of EH.

Sex differences in the duration of crown formation in some non-human primate species, however, may be expected to result in sex differences in LEH expression. In chimpanzees (Kuykendall, 1996), and pig-tailed macaques (Sirianni and Swindler, 1985), male canines take substantially longer to form than female canines. The mandibular canines of chimpanzee females take, on average, 5 years to form, while male canines take 6.3 years to form (Kuykendall, 1996). The mean mandibular canine crown formation time in pig-tailed macaque females is 1.55 years in contrast to 3 years in males. Such large sex differences in crown formation times might lead one to expect that, all other factors being equal, in these species male canines would have a greater opportunity to record stress events (especially if they are recurrent, seasonal events) than would female canines. It has been suggested as well that within the Hominoidea, greater sexual dimorphism in canine crown height is associated with greater sexual dimorphism in crown formation times (Macho and Wood, 1995).

The relevance of intrinsic factors. In summation, there is currently little reason to expect that human sex differences in intrinsic tooth attributes (in either the deciduous or permanent dentitions) will differentially impact EH expression. In males and females with normal karyotypes, there is only a minor influence of sex-linked genetic differences on differences in enamel thickness. The potential effects of differences in the expression of amelogenin genes, and the structure of amelogenin proteins themselves, on sex differences in enamel quality are currently unknown. Crown size differences between males and females are unlikely to be related to EH expression on the basis of enamel thickness as larger male crowns have relatively thicker dentin, not enamel. The data with respect to sex differences in canalized dental development do

not uniformly point to one sex or the other as having greater genetic control over crown growth. Finally, sex differences in the duration of crown formation are minimal.

In non-human primates, there is reason to expect that differences in crown formation times between males and females may impact EH expression. There are substantial differences in canine crown formation times for males and females of pig-tailed macaques, chimpanzees, and possibly other species with pronounced canine sexual dimorphism. As the canine tooth is often the most frequently affected tooth, and is the most dimorphic in terms of calcification time, sex differences in hypoplasia expression are expected to be most pronounced for this tooth. It is currently not clear to what extent male and female canines may differ in their levels of genetic canalization. It is unlikely that taller male canines would be more vulnerable to disruption than smaller female canines as a result of enamel thickness differences, as taller male canines do not have relatively thicker enamel than female canines.

Based on these considerations, we expect that in great apes, who often exhibit multiple LEH defects, and in whom canines are sexually dimorphic in crown formation times, males should record more episodes of stress in their canine teeth than females. In the third part of this review, we examine this possibility in relation to LEH counts in male and female great apes.

EXAMINATION OF THE EVIDENCE

Is there evidence of enhanced female buffering in EH studies?

Biologically "stressed" samples. If indeed males are biologically less well buffered than females and are more susceptible to fluctuations in environmental variation, then under various conditions that theoretically "stress" homeostatic biological systems, males would be predicted to more frequently retain markers of stressful events. Within this theoretical model, a survey was conducted of bio-anthropology and clinical dental literature devoted to EH prevalence in biologically stressed human groups. This survey yielded seven separate categories of

what may be regarded as biologically "stressed" groups: (1) neonates of low and very low birth weight, (2) living samples with independent evidence of stress, (3) archaeological samples with independent evidence of stress, (4) slave populations, (5) historical poorhouse samples, (6) low socio-economic status groups, and (7) indigent individuals or unclaimed bodies. The first three categories rely upon direct evidence of stress such as that provided by clinical or historical records. Categories 4–7 involve indirect evidence, based upon frequent associations between characteristics of a group (e.g., low-socio-economic status) and nutritional and/or disease stress.

The timing and severity of weaning stress has been inferred from the chronology and prominence of enamel defects. However, we agree with Katzenberg and co-workers (1996) that this is a coincidental association and that factors of enamel prism orientation, rate of enamel secretion, as well as extrinsic factors may be involved. Therefore we have not included weaning as a subdivision of this section on stressed groups.

We recognize that our categorization of groups as "stressed" as opposed to groups we categorize as having "unknown levels of stress" (see later section) is based on our assessment of available data bearing on this issue. We encourage readers to employ alternative criteria for identifying stressed groups and to examine the evidence for enhanced female buffering in the EH literature based on diverse classifications.

As has been emphasized, an understanding of cultural practices that may differentially impact the health of male and female children is important in interpreting the relationship between the degree of stress experienced and the manifestation of hypoplastic defects forming postnatally. Many studies, unfortunately, do not provide cultural contexts. Those that do so afford the clearest opportunity to evaluate the potential impact of enhanced female buffering on EH expression. Among the Wolof of West Africa (Hrды, 1987) and the Mukogodo of Kenya (Cronk, 1993), relative to sons, daughters are given preferential access to resources and are breast-fed until later ages during childhood. However, in her review of

sex-biased parental investment, Hrды (1987, p. 99) points out that "the most widely spread bias" of differential provisioning of sons and daughters, ". . . particularly in Asia, parts of Latin America, and occasionally Europe, appears to be in favor of feeding males more." If there is evidence of female preference in cultural practices, then elevated levels of EH in male children cannot be taken as indicative of greater male vulnerability: males may show more EH in these instances as a result of parental bias in favor of females. On the other hand, if there is evidence of son preference in cultural practices relating to the provisioning and care of offspring, an elevated prevalence of EH in males would lend support to the male vulnerability hypothesis.

The variety of data sources and study groups in this section complicates comparisons since some, such as neonates and low socio-economic status groups, represent living samples analyzed by clinicians or dentists, while other samples (poorhouse, indigent, and archaeological collections) consist of recent, historic, or prehistoric skeletal samples. Given the numerous factors that influence intra- and inter-observer variation in observing and recording enamel defects, questioning the validity of this approach is reasonable. We contend that within a particular analytic category, there will be a higher degree of reliability in methods than between groups. Comparative assessment of research results will be conducted between studies within a category, with the goal of ascertaining a pattern of consistent variation in EH between the sexes. Tables 2–6 summarize the findings of these studies, listing study samples, subgroups of each sample (or categories of analysis), EH prevalence by sex within subgroups or categories, statistical significance at the 0.05 level, and sources of the data. A summary of our findings for each of the seven analytic categories will be provided prior to the final evaluation of congruence, or lack of it, between analytic groups.

Low and very low birth weight neonates.

An extensive clinical literature exists on the phenotypic characteristics of newborn infants with low (< 2500 grams), very low

TABLE 2. Enamel hypoplasia by sex in low birth weight and premature children

| Study sample | Category | EH prevalence | Significance | Data source |
|--|-----------------------------|--|---------------------------------|------------------------------|
| Royal Dental Hospital Malmö, Sweden | Preterm LBW ¹ | F = 71.8% (28/29) M = 69.0% (20/29) | $\chi^2 = 0.064$ $P = 0.800$ | Grahnén and Larsson, 1958 |
| | Control NBW ² | F = 7.1% (2/28) M = 24.2% (8/33) | Fisher's extract $P = 0.092$ | |
| Rural China near Beijing, PRC | EH all types | F = 19.4% (130/668) M = 25.0% (169/676) | $\chi^2 = 5.959$ $P = 0.015$ | Li et al., 1995 |

¹ LBW, Low birth weight.

² NBW, Normal birth weight.

(1000–1500 grams), and extremely low (<1000 grams) birth weight (Seow, 1997b). Below normal birth weight is often associated with departure from normal development resulting in an association between low birth weight and prematurity or preterm birth (Pimlott et al., 1985). Numerous investigations of EH prevalence among low birth weight (LBW) and very low birth weight (VLBW) neonates have revealed a significant inverse correlation between these variables (Johnsen et al., 1984). Early studies showed that LBW children have EH prevalences of 20–30% (Seow et al., 1987). As technological developments in neonatal care improved, and while survival of VLBW children increased, these very small children were found to have an even higher incidence of developmental problems, including EH prevalences between 43 and 96% (Seow, 1997b). The greater degree to which LBW deviates from normal birth weight, the more impaired are biological systems (respiration, circulation, hematological, immunological) essential for survival, and the higher the prevalence of enamel defects (Brook et al., 1997; Lai et al., 1997; Seow, 1997b). As birth weight decreases and “stress” levels increase, we predict that if females are better buffered from conception, they should display significantly lower rates of EH than males.

Adequately investigating this prediction is problematic because research results on neonatal enamel defects rarely report prevalence by sex (Miller and Forrester, 1959; Seow and Perham, 1990). The small number of underweight births may partly account for this pattern of data reporting, rendering subgroups by sex too small for reliable frequency data and for statistical analysis. Our

literature search found only a few studies of EH among low and very birth weight children that reported results by sex (Table 2).

An early study compared EH prevalence in a group of prematurely born children (mean birth weight = 2,057 grams) with a normal birth weight control group (mean = 3645 grams) (Grahnén and Larsson, 1958). No significant difference in prevalence of EH by sex was discovered among either the premature, physiologically stressed group, or the normal, unstressed, control group (see Table 2 for data). Enamel defects in the primary teeth of LBW children were compared to normal birth weight (NBW) controls by Fearne (et al., 1990). Characteristically, LBW children in this study exhibited a significantly higher frequency of EHs (71%) than NBW controls (15%), but no sex differences in prevalence were detected in either group (LBW or NBW) of this clinical British sample. Deciduous EHs were found to be significantly and positively associated with male sex, LBW, and prematurity in a large sample of rural Chinese children of 3 to 5 years in age (Li, 1993; Li et al., 1995). In their study of 1344 Chinese children between 3 and 5 years of age, Li et al. (1995) found that males displayed a higher incidence of EH than females. According to the authors, this result was unanticipated given the widely known cultural preference for sons in Chinese society. However, a preference for sons would not have impacted the expression of defects forming prenatally.

Living samples with independent evidence of stress. Zhou and Corruccini (1998) believe that their data, in conjunction with data gathered by King (1989), provide evidence of the effects of son preference and

male vulnerability on the incidence of LEH. These researchers examined the incidence of LEH in urban and rural areas, before, during, and after the Great Chinese Famine of 1959–1961. For their entire sample of 3014, males had a slightly greater frequency of LEH than females; however, in each subsample (rural, urban, pre-famine, famine, and post-famine) the higher LEH frequencies of males were not significantly different from the lower LEH frequencies of females. Zhou and Corruccini interpret these data as the result of son preference counteracting greater male vulnerability. They argue that in Hong Kong (King, 1989), the LEH difference between males and females is more pronounced because son preference in this city is less extreme than it is in rural areas and thus does not ameliorate the greater vulnerability of males.

Some aspects of Zhou's and Corruccini's data do not fit comfortably into this explanation. For example, if males are more vulnerable, one would expect there to be a greater sex difference in LEH during the famine period, a time of nutritional stress. However, the LEH sex difference is neither significant during the post-famine years, nor during the period of famine ($p < 0.1706$). It is possible that periods of stress interact with the practice of son preference such that physiologically stressed male children are given preferential access to resources. While not addressed by this study, this interaction does appear to occur in rural Guatemala (May et al., 1993), where male children with high morbidity are given more food than are highly morbid female children. However, in this case, girls also have significantly higher frequencies of LEH than boys.

Santos and Coimbra (1999) find that hypoplastic defects occur in 98.7% of individuals (at least one defect in an individual's anterior dentition) among Tupí-Mondé Amerindians. This high incidence is thought to have resulted from contact with Europeans, leading to infectious and parasitic disease epidemics. Males and females show no significant difference in their incidence of hypoplasia in this group, even though stresses must have been severe.

Three studies report higher frequencies of LEH in girls relative to boys (Table 4). It is interesting that these three studies derive

from the similar cultural regions of Mexico (two studies) and Guatemala (one study), Latin American countries in which male bias in offspring care is notable (Hrdy, 1987). In each of these studies, population-wide nutritional stress is documented. Two studies (Goodman et al., 1991; May et al., 1993) provide nutritional assessment of diets prior to supplementation, finding energy and/or protein intakes to be deficient.

Goodman et al. (1987) find that female children from Solis, Mexico are particularly at risk of EH during the second and third years of life, when male children may be given greater access to basic resources such as food and health care. Goodman et al. (1991) find no sex difference in the chronological pattern of LEH in children of Tezonteopan, Mexico, no interaction effect between sex and supplementation, and a slight difference in the incidence of LEH in males versus females. However, these researchers did find a statistically significant sex difference in the number of LEH-affected mandibular canines. The higher frequency of LEH on the mandibular canines of girls relative to those of boys is consistent with the fact that girls were more prone to second and third degree malnutrition. Finally, May et al. (1993) report that rural Guatemalan girls have higher LEH frequencies than boys, but our chi-square analysis difference reveals that this sex difference in LEH frequency is nonsignificant (Table 3). Nevertheless, these authors explain the higher frequency of LEH in girls as a result of son preference: females received less nutritional supplement than males, and females in the high-morbidity category received less supplement than males in the high-morbidity category. In addition, males receiving a high amount of supplementation had retarded hand-wrist ossification, possibly, the authors suggest, because males who were most often ill were receiving the most supplement. The EH results of these three studies neither refute nor support the greater male vulnerability hypothesis: girls appear to have elevated expressions of EH as a result of the preference for sons.

Archaeological samples with independent evidence of stress. Table 4 summarizes EH data from archaeological samples

TABLE 3. EH prevalence by sex in samples from living groups with independent evidence of stress

| Study sample | EH types | Subgroups | EH prevalence by sex | Significance | Data source |
|----------------------|--|--|--|---|----------------------------|
| Contemporary Chinese | LEH (individuals with one or more defects) | Prefamine | F = 47.3% (142/300) M = 50.9% (140/275) | $\chi^2 = 0.734$ $P = 0.3916$ | Zhou and Corruccini (1998) |
| | | Famine | F = 53.7% (252/469) M = 58.3% (254/436) | $\chi^2 = 1.877$ $P = 0.1706$ | |
| | | Postfamine | F = 43.3% (337/778) M = 47.8% (361/756) | $\chi^2 = 3.041$ $P = 0.0812$ | |
| | | Rural | F = 51.0% (378/741) M = 55.3% (403/729) | $\chi^2 = 2.689$ $P = 0.101$ | |
| | | Urban | F = 43.9% (354/806) M = 47.6% (351/738) | $\chi^2 = 2.058$ $P = 0.1514$ | |
| | | Total | F = 47.3% (732/1547) M = 51.4% (754/1467) | $\chi^2 = 5.015$ $P = 0.0251$ | |
| | | Tupí-Mondé Amerindians | FDI ¹ (1982) DDE ² Index (Individuals with 1+ defects) | Not given; M vs. F Chi-square comparisons were nonsignificant in 84/88 enamel zones | |
| Solis, Mexico | Modified FDI (1982) DDE Index (Individuals with one or more defects) | Females have higher frequencies of EH on all permanent teeth (except LI1) | Significance varies | Goodman et al. (1987) | |
| | | Differences for males and females are significant in several enamel zones | Significance varies | | |
| Tezonteopan, Mexico | FDI (1982) DDE Index; Focus in on LEH (Individuals with matched defects) | F = 60% M = 54.1% Frequency of defects on LC: M = 9.8% (4/41) F = 30.0% (12/40) | Significance not given $\chi^2 = 5.17$ $P = 0.022$ | Goodman et al. (1991) | |
| Rural Guatemala | FDI (1982) DDE Index; Focus is on LEH | LEH in 0–3 yr zones of maxillary canines/incisors: M = 38.5% (15/39) F = 58.3% (14/24) | $\chi^2 = 2.362$ $P = 0.124$ | May et al. (1993) | |

¹ FDI, Fédération Dentaire International.
² DDE, Developmental defects of dental enamel.

TABLE 4. Samples from archaeological skeletal series with independent evidence of stress

| Study sample | Variable | LEH prevalence | Significance | Data source |
|--|-------------|--|--------------|----------------|
| Grashopper Pueblo, Arizona (mandibular C only) | Early | F = 61.4% (43/70) M = 77.3% (34/44) | $P = 0.080$ | Fenton, 1998 |
| | Late | F = 67.9% (19/28) M = 81.3% (13/16) | $P = 0.340$ | |
| | Total | F = 62.6% (62/99) M = 78.7% (48/61) | $P = 0.030$ | |
| Northern Anasazi southwestern Colorado | Teeth | F = 66.2% M = 66.7% | n.s. | Malville, 1997 |
| | Individuals | F = 88% (44/50) M = 93% (41/44) | n.s. | |
| | Duration | Mean no. affected ½-year intervals F = 2.3 M = 2.4 | n.s. | |

in which there is independent evidence of physiological stress. Important shifts in the patterns of EH in two medieval samples from Batn El-Hajar, Nubia were reported by

Van Gerven et al. (1990). High sub-adult mortality in these series is inferred from evidence of porotic hyperostosis, iron and magnesium deficiencies, likely resulting from

infection and nutritional stress. Females in both Nubian samples exhibit lower frequencies of EHs, as well as delayed age of onset (Van Gerven et al., 1990: 418). According to the authors, the increased male prevalence of LEH agrees well with the implication of female resistance, and reaffirms prior documentation of male growth retardation in both Kulubnarti samples.

The northern Anasazi of southwestern Colorado inhabit an area plagued by a short growing season and unpredictable rainfall, conditions that might cause fluctuations in food availability. LEH prevalence was analyzed in 147 ancestral Puebloans from Montezuma County and Mesa Verde National Monument (Malville, 1997). Independent evidence of stress among the Anasazi samples varies by subgroup and period, and includes parasitic (helminth) and infectious diseases, and mild iron deficiency anemia at Mesa Verde. Faunal remains from Pueblo II sites reveal intensive processing of small mammal long bones and cannibalism, behaviors interpreted as evidence for animal protein deficiency and starvation, respectively. In addition, short and less robust long bones of males at several Pueblo II period sites suggest stunted growth. EH in this regional sample is high, with 90% (132/147) of individuals affected during the same half year growth interval on at least two anterior teeth. Alternatively, 66% (689/1041) of anterior teeth were affected, suggesting the population was stressed. When Malville examined prevalence by sex, she found no significant differences either by percentage of teeth affected, by percentage of individuals affected, or by the duration of growth disruption (percentage of half-year growth intervals affected) (Malville, 1997).

The Grasshopper Pueblo (GP) site is located approximately 2000 meters above mean sea level on the Mogollon Rim in Arizona and was occupied between 1275 and 1400 AD. Berry (1985) conducted a preliminary investigation of paleopathology at GP and found no significant difference between the sexes in prevalence of EH. More recently, Fenton (1998) re-examined skeletal and dental stress markers in the GP skeletal series, with intriguing results. The geoclimatic setting of GP insures unreliable rain-

fall, cold winters, and a short growing season; factors that combine to render subsistence agriculture a challenge and nutritional resources unreliable. Fenton found that generally EH prevalence was consistently and significantly greater among males than among females, regardless of how EH frequencies were calculated. The width of LEH defects were also determined to be consistently wider in males than in females, a finding interpreted to suggest longer duration of stressful episodes among men than among women. Independent geocological evidence suggests that environmental stress increased through time at GP, and is associated with diachronic trends in EH prevalence by sex (Fenton, 1998). Males show high EH prevalence in both early and late periods, while among women EH exhibits a significant increase through time. Consequently, in the early period male-female differences in EH are statistically significant, while in the later period they are not. Among males, hypoplasia defect width increases from early to late periods at the site, while among females, there is no change in this measure of hypoplastic defect size.

The higher male prevalence of EH at Grasshopper Pueblo and diachronic trends in EH frequency are explained by Fenton (1998) in terms of cultural patterns of behavior. These factors include sex differences in weaning age and associated stress level, as well as matrilineal kinship and matrilineal residence which may have discriminated against males. These cultural influences are exacerbated by increasingly stressful environmental conditions that suggest overpopulation and over-exploitation of food resources. Fenton's findings contradict our own prediction that if stress levels increase over time males will display higher rates of EH than females due to their greater environmental sensitivity. While we discount the likelihood of weaning stress as a probable explanatory factor for Fenton's findings (Katzenberg et al., 1996), we also find shortcomings in the matrilineal social structure argument. If male neglect follows from a matrilineal social system, why do the significant male-female differences in EH for the early GP sample not continue into the late sample? This study reveals how difficult the

task of explaining sex differences in EH becomes when multiple cultural and environmental factors are operating synergistically or antagonistically.

Slave populations: Afro-American and Roman.

Ample documentation exists for the heavy workload and poor nutritional status of Afro-American plantation slaves in the Caribbean Islands and the United States during 18th and 19th century (Steckel 1986a, 1986b, 1987). A body of anthropological literature has recently documented the biological consequences of disease and malnutrition on the skeleton and dentition of slave and Jim Crow period free black populations (Rose, 1985). These studies suffer from several methodological problems, including small sample size and poor preservation of skeletal and dental remains. Since direct evidence of the health and nutritional status of slave groups is the goal of this corpus of research, many studies report the prevalence of EH, but not all provide sex-specific prevalence data. In their analysis of skeletal and dental stress markers in the Newton Plantation slave sample from Barbados, Corruccini and co-workers (1982, 1985) focus primarily on the chronology of LEH events and its implications for weaning age. Sex differences in LEH prevalence were not reported in these studies, possibly due to the poor state of skeletal preservation.

Our survey of the literature discovered several useful sources reporting enamel defects by individual and by sex; the summary data are presented in Table 5.

In 1987, eight papers from a special symposium on Afro-American biohistory were published in the *American Journal of Physical Anthropology*, but only three directly report data on EH (Kelley and Angel, 1987; Angel et al., 1987; and Rathbun, 1987). Angel and colleagues report sex differences in EH for the First African Baptist Church sample. While their results show a bias toward greater prevalence among males, our analysis reveals that these sex differences in prevalence are not statistically significant. Rathbun's (1987) study provides more detailed attention to LEH prevalences and data are reported by sex in two ways: (1) if a single tooth displays a hypoplastic de-

fect, the individual is considered affected, and (2) teeth from two different tooth classes must display defective enamel for the individual to be counted as affected. According to Rathbun, "...a significant interruption of normal development was considered when at least two teeth of different classes expressed a defective line." (Rathbun, 1987, p. 244). Interestingly, the first analysis of less severe disruptions finds no significant difference between the sexes in LEH prevalence. However, when more severe growth disruptions are considered in this second analysis, males display a significantly higher prevalence of enamel defects than females.

A more recent report by Blakey et al. (1994) provides new data on EH by sex for 27 skeletons from four plantation sites in Maryland and Virginia. Comparative data are also presented by Blakey and co-workers (1994) for a Philadelphia sample of free Afro-American wage laborers — the First African Baptist Church (FABC) sample. The Maryland and Virginia slave samples exhibit LEH in high frequency and display significant differences in prevalence between the sexes. Blakey's analysis of the comparative FABC sample yielded results congruent with Rathbun's findings among slaves in Charleston, South Carolina. The sexes are similar in LEH prevalence when faint or mild EHs are the subject of study; however, when major growth arrests (MGA in Table 5), interpreted to record severe growth disruption, are analyzed, males are significantly more often affected than females.

A rural sample of slaves and war veterans of the Roman Imperial era from Lucius Feroniae, Rome (LFR) were comparatively analyzed for pathological dental lesions together with an urban Roman sample from the necropolis of Isola Sacra (NIS) (Manzi et al., 1997, 1999). While the overall prevalence of LEH was similar in both samples (82% LFR slaves; 81% NIS urbanites), significant sex differences were found in both series, with males displaying higher LEH prevalence than females. Individual and tooth count frequencies for EHs were subsequently reported by sex for these Italian sites, and for the 7th century AD Lombard necropolis of La Selvicciola (SLV) (Manzi et

TABLE 5. Sex differences in enamel hypoplasia among enslaved populations

| Study sample | Category ¹ | LEH prevalence | Significance | Data source |
|---|-----------------------|--|--|---------------------|
| Free Afro-American (FABC) Philadelphia, PA | | F = 47% (9/19) M = 68% (17/25) | $\chi^2 = 1.901$ $p = 0.168$ (n.s.) | Angel et al., 1987 |
| African American slaves (Maryland and Virginia) | | F = 70% (7/10) M = 100% (17/17) | Fisher's exact $p = 0.041$ | Blakey et al., 1994 |
| First African Baptist Church | Slight | F = 79.3% (23/29) M = 68.0% (17/25) | $\chi^2 = 0.894$ $p = 0.344$ (n.s.) | Blakey et al., 1994 |
| | MGA | F = 37.9 (11/29) M = 68.0% (17/25) | $\chi^2 = 4.862$ $p = 0.027$ | |
| African American slaves Charleston, SC | 1 | F = 71% (10/14) M = 100% (13/13) | Fisher's exact $p = 0.098$ (n.s.) | Rathbun, 1987 |
| | 2 | F = 50% (7/14) M = 92% (12/13) | $\chi^2 = 3.935$ $p = 0.047$ | |
| Italian series (Imperial and Medieval Rome) | NIS | F = 66.7% (12/18) M = 87.5 (35/40) | Fisher's exact $p = 0.079$ (n.s.) | Manzi et al., 1999 |
| | LFR | F = 83.3% (20/24) M = 84.0% (21/25) | Fisher's exact $p = 1.00$ (n.s.) | |
| | SLV | F = 73.3% (11/15) M = 75.9% (22/29) | Fisher's exact $p = 1.00$ (n.s.) | |

¹ Key to category abbreviations: Slight = slight hypoplastic lines; MGA = major growth arrests; 1 = individuals in whom a single tooth displays a hypoplastic defect; 2 = individuals displaying hypoplastic defects on two different tooth classes; NIS = urban Roman sample from the Necropolis of Isola Sacra; LFR = rural slaves and war veterans from Lucius Feronia Rome; SLV = Necropolis of La Selvicciola.

al., 1999). Our analysis of individual frequency data for EH by sex reveals no statistically significant differences for any of the three skeletal series (LFR, NIS, or SLV) (see Table 5). The number of individuals *not* affected by EH is small for all three groups, requiring the use of Fisher's exact test. While LFR and SLV exhibit minor differences in the frequency of LEH by sex, at Isola Sacra the statistically nonsignificant differences (>20% between the sexes, M > F) may be of biological significance.

Historical, almshouse, and poorhouse samples. Levels of physiological stress in historic, almshouse, and poorhouse skeletal samples have recently come under investigation using traditional analytic techniques of the bio-archaeologist (Grauer, 1995; Saunders and Herring, 1995). However, most studies that report data regarding EH either do not provide prevalence by sex (Higgins and Sirianni, 1995; Larsen et al., 1995), or sex specific data is based on such small samples that trends are not discernable (Elia and Wesolowsky, 1991; Murray and Perzigian, 1995; Winchell et al., 1995). However, Lanphear (1990) observed LEH on the maxillary central incisors and mandibular

canines of 296 individuals from the Monroe County Poorhouse Cemetery in upstate New York. This historic sample is unrepresentative in that the sample consists of the poorest members of the lowest socio-economic group in an industrializing population. While LEH prevalence was predictably high, 70–73% had one or more hypoplastic events [84% (153/181) were affected when the individual counting method is used], no differences in LEH were found between the sexes. The EH data for these samples, as well as for low socioeconomic status samples (see next section) are summarized in Table 6.

Low socio-economic status groups. Early epidemiological studies of poorly nourished children in unsanitary environments revealed high levels of linear EH in the deciduous maxillary incisor teeth. A series of studies among Apache in Arizona (Infante, 1974), living Maya of Guatemala (Infante and Gillespie, 1974, 1976; Sweeney et al., 1969, 1971), and Nigerian children in Africa (Enwonwu, 1973) confirm the association between low socio-economic status (SES), poor nutrition, lack of sanitation and high frequencies of EH. These populations are justifiably considered "stressed," and theo-

TABLE 6. EH prevalence by sex among poorhouse, low SES and cadaver groups

| Study sample | Variable/ group | EH prevalence | Significance | Data source |
|---|---------------------------------|--|---|-----------------------------|
| Hamman-Todd "Whites" | Permanent LEH | M > F all diffs. signif. | Varies | El-Najjar et al., 1978 |
| Hamman-Todd "Blacks" | Permanent LEH | Few significant diffs. between sexes | Varies | |
| Maya school children Guatemala | Deciduous LEH | F = 31.6% (67/212) M = 30.9% (67/217) | $\chi^2 = 0.027$ $P = 0.871$ | Infante and Gillespie, 1974 |
| Karen school children rural Thailand | Deciduous EH | F = 21.4% (34/159) M = 23.8% (44/185) | $\chi^2 = 0.281$ $P = 0.596$ | Kanchanakamol et al., 1996 |
| Monroe County Poorhouse, upstate New York | Permanent LEH | F > M but not significant | n.s. | Lanphear, 1990 |
| Republic of Cameroon West Africa | Massa n = 98 Overall n = 863 | F = 14.4% M = 32.8% F = 27.6% M = 39.2% | $\chi^2 = 2.910$ $P = 0.09$ $\chi^2 = 8.280$ $P < 0.004$ | Maunder et al., 1992 |
| Italy, 19th Century Turin | Permanent LEH | No diff. by sex values not reported | n.s. | |
| Italy, 19th Century Florence | Permanent LEH | No diff. by sex values not reported | $\chi^2 = 0.36$ $P = 0.54$ | Moggi-Cecchi et al., 1994 |

retical expectations based on the idea of nutritional association predict greater prevalence of EH in males than in females. The one study that addresses sex differences reported no significant difference by sex within village samples or with villages samples combined (Infante and Gillespie, 1974). The authors state that the absence of inter-sex differences in EH was also found by Sweeney and colleagues (1969, 1971) in their study of Guatemalan children, though this finding is not discussed in their published reports. No sex differences in prevalence of enamel defects were detected in an analysis of the deciduous anterior teeth of 344 rural Thai pre-school children (Kanchanakamol et al., 1996).

The prevalence of LEH in the permanent teeth of African school children from poor rural villages and urban private schools in Camerouns was reported by Maunder (et al., 1992). Rural village samples are similar in having little material wealth and strict childhood feeding practices that may be stress inducing. Northern rural groups have lower animal protein intake (a mean of 37 grams/day among the Massa) and greater potential for seasonal nutritional stress than southern villages, where animal protein varies between 220 and 288 grams per person/day. The urban private school sample serves as an outgroup or control sample, in which

affluent families provide children with the best health care and nutrition available.

While in rural villages LEH prevalence was significantly higher than among private school children, males exhibited a significantly higher prevalence of EHs than females. In some rural groups, such as the Massa, twice as many males (32.8%), as females (14.7%) displayed LEH. The authors suggest that "Boys may be more susceptible to undernutrition than girls, and this may be particularly consequential before six years of age." (Maunder et al., 1992). Preferential treatment of children by sex and sex bias in mortality were not investigated in this study.

Cadavers, indigents, and unclaimed bodies. Large collections of human skeletal specimens have been established at museums and medical research facilities throughout the world. Many of these series, including the Hamman-Todd at the Cleveland Museum of Natural History, and Terry Collection at the Smithsonian Institution), consist of individuals derived from the impoverished poor, whose unclaimed bodies were processed by medical students and whose skeletons became museum property. Some skeletal series are better documented than others, and provide vital information regarding individual specimen's age and sex,

health status, medical history, and age at time of death. One of the first anthropological studies to investigate the etiology of EH was based on an "indigent" sample. El-Najjar and colleagues reported LEH frequencies for Blacks and Whites in the Hamman-Todd collection and reported results in percentage of individuals affected by sex (El-Najjar et al., 1978). While White males consistently and significantly had higher frequencies of LEH than white females for all teeth, Blacks displayed few significant sex differences in LEH prevalence (P2 only).

Similar analyses of LEH prevalence have also been completed on recent skeletal series in Europe. A recent assessment of 18th and 19th Century human skeletons from Coimbra, Portugal, is based upon an "indigent" sample with identification records (Cunha, 1995). A high level of osteological "stress" markers, including Harris' lines of arrested growth, and lesions of the cranial vault confirm records indicating that this population consists of individuals of low SES. The observation of LEH in mandibular canine teeth reveals a high individual prevalence of 83.5% (116/130), yet no significant difference in LEH frequency was found between the sexes. Similar skeletal series have been examined by Moggi-Cecchi and co-workers whose analysis of LEH in skulls of "unclaimed indigents" from hospitals in Florence (Moggi-Cecchi et al., 1994) and Turin, Italy (Moggi-Cecchi et al., 1993) reveal no significant differences by sex. The actual prevalence of LEH by sex was not reported in these studies, a practice which is not uncommon when the test for sex differences is preliminary to pooling data for further analysis.

Summary of evidence for enhanced female buffering in biologically "stressed" groups. Studies of EH prevalence among groups with evidence of high levels of environmental, physiological, or cultural stress yield some provocative results and some contradictions. In modern Mesoamerican populations, several studies of EH in permanent teeth have documented higher prevalence among females than among males, a result attributed to culturally mediated preference for sons (Goodman et al, 1987,1991;

May et al., 1993). In the same cultural setting and under conditions of chronic malnutrition, several investigators have revealed that deciduous EHs of prenatal origin do not exhibit inter-sex differences in prevalence (Infante and Gillespie, 1974; Sweeney et al., 1969, 1971). These results receive confirmation from Karen school children in rural Thailand with acute and chronic malnutrition (Kanchanakamol et al., 1996), where prenataally formed EHs of maxillary deciduous incisor teeth also display nonsignificant differences in prevalence between the sexes. As a group, these results suggest that when cultural bias is absent, during prenatal development of deciduous enamel, hypoplasia prevalences by sex are equivalent. This outcome agrees with the results from LBW and premature children, whose deciduous dental hypoplasias, though high in frequency, show no significant difference between the sexes (Fearne et al., 1990; Grahnén and Larsson, 1958).

Li and colleagues' (1995) unanticipated finding of greater EH prevalence among males in rural Chinese children, a society known for preferential treatment of males, presents a contradictory picture. The expected result of greater female prevalence of EH may not have been realized because this study included all deciduous tooth classes. This protocol pools enamel zones which develop over a long period of both pre- and post-natal development, and consequently mixes the analysis of prenatal lesions that are free from cultural bias with postnatal lesions that may reflect preferential treatment of sons.

Among enslaved populations, two studies show higher EH frequency among males than among females (Blakey et al., 1994 and Rathbun, 1987). In both studies these differences were not significant when mild defects or single tooth classes were analyzed, but differences became significant ($M > F$) when severe lesions (major growth arrest lines) or significant growth disruptions (more than one tooth class affected) were inferred. At Grasshopper Pueblo, Fenton (1998) found that as environmental stress increases through time, the width of LEH bands increase especially among males. He interprets these findings as indicating longer

stress episodes among males than among females. The implication is that among Afro-American slaves (Blakey et al., 1994; Rathbun, 1987), native North Americans (Fenton, 1998), and Mesoamericans (Cohen et al., 1997, see below), when severe stress indicators are considered, males are more frequently impacted than females. These studies follow a methodological procedure developed somewhat differently by Hutchinson and Larsen (1988, 1990) and by Ensor and Irish (1995), that uses the width of linear EHs as a measure of the duration and/or intensity of stress. However, there are problems with directly inferring duration or intensity of stress from the width of LEH defects. Defect width has been shown to vary with the defect's position on the outer enamel surface. Lesions near the occlusal region will be wider than lesions near the point of maximal crown curvature, and defects near the CEJ will be narrower, given the same physiological insult (Hillson and Bond, 1997; Hillson, 1998). This variation is also influenced by the geometry of enamel formation and the angle at which prisms intersect with the outer enamel surface (Radlanski et al., 1995).

Fenton's (1998) study of Grasshopper Pueblo and Zhou and Corruccini's (1998) study of rural China both find that during periods of increased stress, sex differences in EH are reduced and not significant in comparison to periods of decreased stress levels. This result is the opposite of our initial prediction, based on the environmental sensitivity of males, that with increasing levels of stress males will exhibit significantly greater prevalence than females. Thus while some of the evidence from stressed populations is inconclusive, given the irregular attention to cultural contexts, the evidence from groups with high levels of stress and well-described cultural contexts does not strongly support expectations regarding EH frequencies based on enhanced female buffering.

Is there evidence of enhanced female buffering from LHPC studies?

We devote this section specifically to an analysis of sex differences in LHPC incidence. Accurate assessment of sex in sub-

adult human skeletons is difficult. This situation dictates that juvenile health statistics for prehistoric skeletal samples are usually reported with the sexes pooled. While early anthropological reports on LHPC frequency were based on recent and prehistoric skeletal series (Skinner, 1986b; Lukacs and Walimbe, 1998), or on late Pleistocene hominines (Skinner, 1996), the question of whether sex differences in LHPC prevalence exist can only be answered by reference to data derived from clinical and epidemiological studies. Consequently, the data for this section come from a survey of the clinical and epidemiological literature and from a recent field study of LHPC prevalence in western India conducted by Lukacs.

LHPC prevalence appears to be associated with SES. Middle class school children who presumably experienced low stress levels have very low frequencies of the trait: 0.55% (13/2380) Burnaby, Canada and 2.4% (33/1350) Vancouver, Canada (Skinner and Hung, 1989). By contrast, nutritionally disadvantaged groups with low SES are characterized by substantially higher prevalence of LHPC (30–50%): 33.2% (429/1291) Black Head Start children, Mississippi (Silberman et al., 1991); 34.5% (39/113) Harappa, Pakistan (Lukacs, 1991); 44.4% (20/45) Calcutta, India (Skinner, 1986b). If females are better buffered than males, we predict that as the level of stress increases and the overall frequency of LHPC rises, males will exhibit significantly higher LHPC prevalence than females.

A recently completed epidemiological investigation of LHPC prevalence among Indian children in rural village schools and in urban private schools in west-central India, in the state of Maharashtra, revealed significant variation between locations sampled. While all school samples are "disadvantaged" or "stressed" by western standards, some economic heterogeneity exists within and between these Indian samples. However, when sex differences in LHPC prevalence were considered, no significant differences were observed, either in the composite sample, in individual school samples, or when analyzed by location (rural, urban). The frequency of enamel defects in this contemporary sample is presented by sex in

TABLE 7. Prevalence of LHPC by sex among modern school children in western India

| Sex | Rural | | Urban | | Total | |
|---------|----------------------------|--------------|-----------------------------|--------------|-----------------------------|--------------|
| | n | Affected (%) | n | Affected (%) | n | Affected (%) |
| Female | 73 | 17 (23.3) | 48 | 7 (14.6) | 121 | 24 (19.8) |
| Male | 80 | 19 (23.8) | 87 | 16 (18.4) | 167 | 35 (21.0) |
| Total | 153 | 36 (23.5) | 135 | 23 (17.0) | 288 | 59 (20.5) |
| M vs. F | $\chi^2 = 0.005; p = .946$ | | $\chi^2 = 0.317; p = 0.573$ | | $\chi^2 = 0.054; p = 0.816$ | |

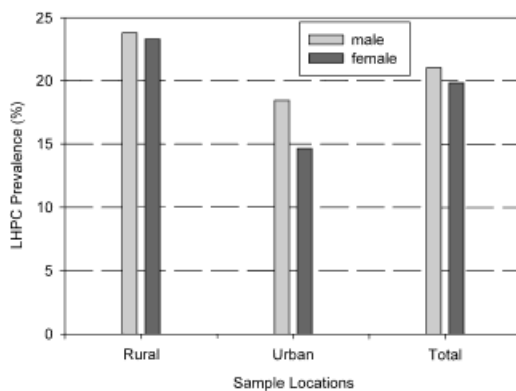


Fig. 2. Prevalence of LHPC among rural and urban school children in western India. Bar height equals the percentage of individuals with one or more canine teeth exhibiting enamel defects. The rural sample includes pooled data from two villages (Inamgaon and Walki) in western Maharashtra; the urban sample includes data from two schools in the city of Pune.

Table 7, and displayed in Figure 2. The absence of sex differences in LHPC among South Asian school children reported here is in agreement with results from an earlier pilot study of 113 rural Pakistani school children (Harappa village, Punjab Province, Pakistan) that found no sex differences in LHPC (Lukacs, 1991).

Our survey of the clinical literature on prevalence of enamel defects in deciduous teeth reveals substantial support for absence of sex differences in defect prevalence (Table 8). This is equally true for populations judged to be "stressed," and for those groups experiencing lower stress levels. Most investigations of LHPC prevalence by sex revealed no significant difference, including samples from Kentucky (Badger, 1985), Indiana (Brown and Smith, 1986), California (Nation et al., 1987), Mississippi (Duncan et al., 1988, 1994; Silberman et al., 1989, 1991), Harappa village, Punjab Province, Pakistan (Lukacs, 1991), and Vancouver, BC (Skinner and Hung, 1989; Skinner et al., 1994). Some

investigators did not report sample size or number of affected individuals, others either did not apply or document the results of statistical tests. Therefore the data in Table 8 are uneven with respect to level of detail reported.

Some studies were based on large samples, such as Skinner and Hung's (1989), which found 7 females and 6 males affected in a study sample of 2380. Unfortunately the investigators did not report the sex ratio of the study sample. However if subjects were selected at random, the sample should have had a nearly balanced sex ratio (50% male, 50% female), and sex differences in LHPC would not be significant.

Two additional studies with large sample sizes report sex differences in LHPC that are significant. Among Mississippi Whites, males exhibit a higher prevalence of LHPC in maxillary canine teeth than females, but we detected no significant sex difference in frequency among Blacks or for the total sample (Duncan et al., 1994). When Silberman and co-workers analyzed the mandibular canine, significant differences by sex ($M > F$) were reported for the pooled sample of Mississippi Black and White school children, but neither group showed significant sex differences when analyzed separately (Silberman et al., 1991). The higher rate of LHPC in maxillary canines of White males reported by Duncan et al., (1994) provides independent confirmation of Skinner's observation that "...there is a tendency... for the defect to occur primarily in the female lower jaw but in both jaws in the male" (Skinner and Hung, 1989, p. 198).

Since not all reports of enamel defect prevalence include appropriate statistical analysis, results that are suggestive, or indicative of trends, are occasionally accepted as having statistical validity. For example, Brown and Smith's (1986) data report a

TABLE 8. Sex differences in prevalence of localized hypoplasia of primary canines (LHPC)

| Study group | Subgroup | Prevalence by sex | Significance | Data source |
|---|------------------------------------|--|---------------------------------|---------------------------|
| Clinical Sample (Kentucky) | No details | F = 44.0% (13/30) M = 46.5% (12/25) | $\chi^2 = 0.120$ $P = 0.729$ | Badger, 1985 |
| Indiana University Clinical | No details | F = 27.1% (13/48) M = 42.2% (27/64) | $\chi^2 = 2.725$ $P = 0.09$ | Brown and Smith, 1986 |
| Mississippi Head Start | Fluoridated | F = 37% M = 39% | No data no statistics | Duncan et al., 1988 |
| | Non-fluoridated | F = 37% M = 36% | No data no statistics | |
| Mississippi public school children (max dc only) | Black | F = 9.9% (68/687) M = 11.3% (73/645) | $\chi^2 = 0.708$ $P = 0.400$ | Duncan et al., 1994 |
| | White | F = 5.1% (31/612) M = 7.9% (49/619) | $\chi^2 = 4.115$ $P = 0.042$ | |
| | Total | F = 7.6% (99/1299) M = 9.7% (122/1264) | $\chi^2 = 3.353$ $P = 0.067$ | |
| Rural village school children Harappa, Pakistan | South Asia | F = 29.6% (16/54) M = 39.0% (23/59) | $\chi^2 = 1.09$ $P = 0.296$ | Lukacs, 1991 |
| Loma Linda University Dental School Clinic | No details | No data by sex | n.s. | Nation et al., 1987 |
| Mississippi Head Start Black school children | Black (fluor and non) ¹ | F = 37.6% (56/149) M = 36.8% (68/185) | $\chi^2 = 0.024$ $P = 0.877$ | Silberman et al., 1989 |
| Mississippi Public School Children (mand dc only) | Black | F = 31.1% (203/652) M = 35.4% (226/639) | $\chi^2 = 2.606$ $P = 0.106$ | Silberman et al., 1991 |
| | White | F = 15.0% (87/579) M = 19.3% (118/613) | $\chi^2 = 3.730$ $P = 0.053$ | |
| | Total | F = 23.6% (290/1231) M = 27.5% (344/1252) | $\chi^2 = 5.011$ $P = 0.025$ | |
| Burnaby Kindergarten Vancouver, Canada | Mixed ethnicity | No data by sex | n.s. | Skinner and Hung, 1989 |
| Burnaby Public Schools British Columbia, Canada | Mixed ethnicity | F = 30% (12/40) M = 32% (18/56) | $\chi^2 = 0.049$ $P = 0.823$ | Skinner et al., 1994 |

¹ Fluor and non = fluoridated and non-fluoridated categories combined.

large difference in defect prevalence between females (27.1%, $n = 48$) and males (42.2%, $n = 64$), but curiously, no statistical results are presented. When we applied a chi-square test of association to their data, no significant sex difference in prevalence was found ($\chi^2 = 2.725$, $p = 0.09$), yet other investigators have referred to Brown and Smith's results as apparently indicating a greater prevalence of enamel defects among males (Silberman et al., 1989). We suggest that their findings are best interpreted as inconclusive, but deserve further inquiry and testing.

In sum, 14 of 16 comparisons (88%) listed in Tables 7 and 8 revealed that LHPC prevalence among the living populations show no significant difference by sex. Some large samples reveal a weak tendency to affect

males more than females (Duncan et al., 1994; Silberman et al., 1991). However, Duncan's (1994) "stressed" Black sub-sample displays a higher prevalence of LHPC overall than less stressed Mississippi Whites, in which male prevalence is greater than females. This finding contradicts our initial prediction that greater sex differences will be found among "stressed" study groups, with male prevalence exceeding females. Silberman and colleagues (1991) found sex differences only in the pooled Black and White composite sample.

We have cautious confidence in the results derived from the new data on LHPC prevalence in India and from the literature surveyed on sex differences in LHPC. These enamel defects are thought to form perinatally, therefore partly controlling for bias in

parental care favoring one sex more than the other. In addition, the clinical samples included in the literature review are not known to be biased in preferential treatment of neonates by sex. There is no tendency in the data for more stressed groups to show higher levels of sexual dimorphism in defect frequency than groups with lower levels of stress. The few instances in which sex differences in LHPC were found either contradicted expectations or described composite samples. We interpret these findings to suggest that with regard to LHPC formation, the sexes are approximately equally predisposed. There appears to be little evidence for better buffering of females or for greater environmental sensitivity of males.

Samples with unknown levels of stress

In this section, we review EH frequencies by sex for samples in which levels of stress are not described or cannot be reliably inferred with confidence. In this collection of studies, which must encompass a wide range of environmental and cultural variation, we do not expect to see a consistent sex difference in EH frequencies. Three categories of studies are examined, those focusing on samples of skeletal series, living populations, and non-human primates.

Skeletal series with unknown levels of stress. In many archaeologically derived skeletal series it may be difficult to ascertain if environmental stress levels or cultural sex biases were present during the childhood period of dental development. Evidence cited above, for enamel defects in the deciduous and permanent teeth of populations regarded as "stressed" by independent criteria, revealed that inter-sex differences in EH were rare. However, when sex differences were found, males always displayed significantly higher LEH prevalence than females (Blakey et al., 1994; Maunders et al., 1992; Rathbun, 1987; van Gerven et al., 1994). Below we summarize sex differences in EH for human skeletal samples from archaeological and historical contexts whose actual levels of environmental and cultural stress remain unknown or poorly documented. The goals of this section are: (1) to summarize existing literature on inter-sex differences in

EH, and (2) to integrate data derived from the literature with our findings for LEH prevalence by sex in physiologically "stressed" groups.

Amerindian native skeletal series. Several studies of EH in archaeological skeletal series report prevalence data by sex. The first widely cited application of LEH as a stress marker in prehistoric native American skeletal research was Goodman and co-worker's (1980) analysis of the series from Dickson Mounds, Illinois. Results are reported in two ways: (1) mean number of hypoplastic defects per individual, and (2) percentage of individuals with one or more hypoplastic defects. Both measures of defect prevalence reveal little difference between the sexes. For the collective sample from Dickson Mound, which includes skeletons from all cultural horizons, our chi-square analysis indicates that sex differences in EH are not significant (Table 9). Samples from each cultural horizon are small in size, suggesting that variation in LEH prevalence by sex from one horizon to another is a random sampling bias. Our application of Fisher's exact test, or chi-square with Yate's correction for continuity, shows that for each horizon sex differences in EH frequency are not statistically significant. Surprisingly, some investigators have interpreted the nonsignificant sex differences between horizons at Dickson Mound to reflect changing cultural attitudes toward care of infants (Rose et al., 1985). In the Late Woodland phase, for example, females (67%, 8/12) exhibit a higher prevalence of EH than males (17%, 1/6); but sample size is small and our application of Fisher's exact test revealed no significant difference. Nevertheless, one study concluded that, "These data suggest that males received differential nutritional care during the Late Woodland, but not during later cultural periods." (Rose et al., 1985, p. 299).

Evidence of health and disease in pre-contact Aleut and Eskimo includes the observation that LEH frequency is significantly higher in Eskimo than in Aleut series, a finding interpreted to suggest greater susceptibility to seasonal food shortages. Analysis of LEH prevalence by sex revealed no significant differences among either Aleut or Es-

TABLE 9. LEH prevalence by sex among Native North American skeletal series

| Study sample | Variable | LEH prevalence | Significance | Data source |
|--------------------------------------|-----------------------------|------------------------------------|---------------------------------|----------------------|
| Dickson Mound | % individuals affected | F = 66% (33/50) M = 62% (31/50) | $\chi^2 = 0.043$ $P = 0.835$ | Goodman et al., 1980 |
| | Mean no. LEH per individual | F = 1.24 M = 1.20 | not given | |
| Arikara Chumash | % individuals affected | F = 14% (11/78) | $P = 0.708$ | Hollimon, 1992 |
| | % individuals affected | M = 17% (16/92) | | |
| Aleut Eskimo | % individuals affected | F = 35% (179/512) | $P = 0.226$ | Keenleyside, 1998 |
| | % individuals affected | M = 31% (108/351) | | |
| Ohio River Valley | Deciduous | F = 0.0% (0/53) | $\chi^2 = 1.156$ $P = 0.282$ | Sciulli, 1978 |
| | | M = 2.2% (2/93) | | |
| Santa Barbara Channel, California | Tooth count % | F = 19.5% (33/169) | $\chi^2 = 0.063$ $P = 0.802$ | Walker, n.d. |
| | | M = 18.5% (34/184) | | |
| Santa Barbara Channel, California | Beginning age | F = 9% (1/15) | Fisher's exact $P = 0.054$ | Walker, n.d. |
| | | M = 45% (5/11) | | |
| | End age | F: mean = 3.4 years | $P > 0.05$ | |
| | | M: mean = 3.6 years | $P > 0.05$ | |
| % duration | F: mean = 4.5 years | $P > 0.05$ | | |
| | M: mean = 4.9 years | | | |
| Santa Barbara Channel, California | % duration | F = 3.7% | | |
| | | M = 3.6% | | |

kimo (Keenleyside, 1998). The influence of division of labor by sex for health status among native Americans was investigated by Hollimon (1992) for the Chumash of the Santa Barbara Channel and the Arikara of the northern plains. This study lacks statistical analysis of pathological lesions and stress indicators, and discusses small differences between the sexes as biologically significant. Hollimon found that LEH prevalence was greater among the Chumash than among the Arikara. Our analysis confirmed this observation and revealed that group differences are highly statistically significant among males ($\chi^2 = 5.826$, $p = 0.016$) and among females ($\chi^2 = 12.550$, $p < 0.001$). However, Hollimon (1992) states that Arikara males displayed more hypoplastic defects than females, when an actual difference of only 3% separates the sexes. She then concludes that Arikara males were apparently more susceptible to nutritional and disease stresses than Arikara females. Our evaluation of the data however, shows that in neither Arikara nor Chumash are sex differences in LEH prevalence statistically significant (Table 9).

Trends in the frequency of EH in 207 individuals over a 5000 year period in the prehistory of the Santa Barbara Channel area of southern California were discussed by Walker (no date). Population increase and level of inter-village interaction over time were found to be associated with a statistically significant diachronic increase in LEH. Walker found no significant sex differences in percentage of teeth affected, or in chronological timing of the first or the last hypoplastic defect, or in the duration of growth disruption. Though statistically not significant, Walker calls attention to the slight trend for females to have a higher proportion of hypoplastic teeth, and a slightly longer duration of defective enamel, and characterizes the slight female sex bias as unexpected, given an assumed greater physiological sensitivity of males to environmental perturbation.

Human remains from Carter Ranch Pueblo, an isolated small site in eastern Arizona, have been analyzed for skeletal and dental markers of health (Danforth et al., 1994). Hypoplasias were evaluated macroscopically on central incisors and canines,

and 70% of 21 individuals were scored LEH positive. No differences were found by age or by sex in either defect frequencies or in age at formation (Danforth et al., 1994).

American colonists. North American Colonial skeletal series have only recently been subjected to scrutiny by bio-archaeologists with the goal of determining health and nutritional status in the early settlements of the northeast. An historic skeletal series, comprised of 253 individuals from the First Anglican Baptist Church in Bellville, Ontario, was recently analyzed for prevalence of EHs by Saunders and Keenleyside (1999). LEH was recorded for 1714 anterior teeth in individuals whose sex was either known from parish records or assessed with a high level of accuracy from pelvic morphology. Frequencies for individuals with one or more hypoplastic defects in their 12 anterior teeth range from 17.4% to 36.1%. These low to moderate rates of hypoplasia are viewed as consistent with expectations for a developing pioneer community in which food was sufficient and chronic disease incidence low. Statistically significant differences were found between the sexes with male frequencies greater than female for the canine teeth only. Greater male susceptibility to environmental perturbation is discussed and the complicating cultural factor of preferential treatment of males considered, but the authors conclude their analysis of sex differences in EH with the statement that, "This issue should be explored further since the observed sex differences might be partly explained by variation in tooth size and rates of enamel formation (Goodman and Rose, 1990)" (Saunders and Keenleyside, 1999).

Australia. One component of Webb's analysis of paleopathology among prehistoric Australian Aboriginal hunter-gatherers was an investigation of three stress markers: cribra orbitalia, EH, and lines of arrested growth (Webb, 1995). Hypoplastic defects of the permanent maxillary canine and third molar were recorded, and frequencies were presented by sex, for six geo-climatic regions of Australia. Though Webb (1995) notes that in many regions sex differences in LEH for

canine teeth are small (central Murray), in others such as central Australia, differences are dramatic (Table 10). However, no statistical analysis is provided. Our analysis of Webb's data show that in five of the six geo-climatic zones sex differences in LEH are not significant ($p > 0.05$). Only in the tropics zone was a statistically significant sex differences in LEH prevalence found, with males exceeding females by more than 27% ($p = 0.005$).

South Asia. Archaeologically derived human skeletal samples from southern Asia provide limited insight into sex differences in EH (Table 10). Two series reveal no significant inter-sex variation in EH: one from the Iron Age site of Sarai Khola in northern Pakistan (Lukacs et al., 1989), and the other from the early Holocene of the middle Ganges Plain (Lukacs and Pal, 1993). However, at the Bronze Age urban site of Harappa, females exhibit a significantly greater prevalence of LEH than males (Lukacs, 1992). While degenerative dental lesions such as antemortem tooth loss, caries, and pulpal exposure, are also more common among females at Harappa, they are not significantly more prevalent than in males. Women's subsistence efforts are more valued in foraging societies, and patriarchal agricultural cultures of south Asia may neglect female offspring and treat males preferentially (Miller, 1981). Though this pattern fits the data for mesolithic foragers and Harappan agriculturalists, it would be premature to conclude that this causal relationship has great historical depth. Caution is therefore required until these results are supported by larger, more complete skeletal samples with better chrono-cultural representation.

Italy. Several investigations of dental paleopathology in Italy have addressed the issues of inter-sex variation in EH (Table 10). A small Neolithic series from western Liguria reveals a high frequency generally, but no significant difference by sex (Formicola, 1986–87, 1987). The rural Greek colony in Metaponto, Italy (6th–3rd century BC) exhibits a relatively high prevalence of LEH overall (78% of individuals, 88/113). These

TABLE 10. Enamel hypoplasia prevalence by sex for selected skeletal series from Australia, Europe, and India

| Prehistoric Australia | | | |
|---|--|--------------------------------------|----------------------------|
| Study sample | LEH Prevalence by Sex | Significance | Data source |
| Central Murray | F = 42.1% (45/107) M = 44.0% (80/132) | $\chi^2 = 0.099$ $P = 0.753$ | Webb, 1995 |
| Rufus River | F = 15.3% (11/72) M = 24.5% (25/102) | $\chi^2 = 2.192$ $P = 0.139$ | |
| South Coast | F = 35.7% (25/70) M = 39.8% (35/88) | $\chi^2 = 0.273$ $P = 0.602$ | |
| Desert | F = 16.1% (5/31) M = 33.8% (27/80) | $\chi^2 = 3.381$ $P = 0.066$ | |
| Tropics | F = 20.5% (9/44) M = 47.9% (35/73) | $\chi^2 = 8.842$ $P = 0.003$ | |
| East Coast | F = 36.8% (21/57) M = 50.5% (51/101) | $\chi^2 = 2.738$ $P = 0.098$ | |
| All Australia | F = 30.5% (116/381) M = 40.4% (253/626) | $\chi^2 = 10.139$ $P = 0.001$ | |
| Prehistoric Europe | | | |
| Neolithic, Italy (western Liguria) | F = 77.8% (7/9) M = 84.6% (11/13) | Fisher's exact $P = 1.00$ | Formicola, 1986-87, 1987 |
| VII-IV Century Pontecagnano, Italy | F = 22.4% M = 33.8% | n.s. | Fornaciari et al., 1985-86 |
| Greek Colony of Metaponto, Italy | F = 74.6% (53/71) M = 77.8% (28/36) | n.s. | Henneberg, 1998 |
| Medieval Norse | no significant difference by gender | n.s. | Scott et al., 1991 |
| Prehistoric South Asia | | | |
| Sarai Khola Pakistan | F = 13% (2/15) M = 33% (7/21) | Fisher's exact $P = 0.252$ (n.s.) | Lukacs et al., 1989 |
| Bronze Age Harrapa Pakistan | F = 93% (13/14) M = 53% (9/16) | Fisher's exact $P = 0.039$ | Lukacs, 1992 |
| 'Mesolithic' Ganges Plains, North India | F = 92% (11/12) M = 95% (19/20) | Fisher's exact $P = 1.00$ (n.s.) | Lukacs and Pal, 1993 |

investigators report significant differences in LEH between urban and rural groups, and between individuals with and without skeletal lesions of treponematosis. No significant in prevalence were found between the sexes (Henneberg and Henneberg, 1989; Henneberg, 1998).

Overall LEH prevalence at Pontecagnano, Italy was 22.8% (38/132 individuals) but declined over time from the pooled VII/VI century sample (45.7%) to the pooled V/IV Century (22.6%). However, when sex differences were examined, male values exceeded female rates, but the difference was not significant statistically (Fornaciari et al., 1985). LEH prevalence for the Imperial Roman slave populations discussed above also show male rates exceeding females, but these differences are generally not statistically significant (Manzi et al., 1997, 1999).

Marianas Archipelago. The recent symposium on prehistoric skeletal biology in island ecosystems included three articles with detailed discussions of LEH in the skeletal series of the Marianas Archipelago (Douglas et al., 1997; Pietrusewsky et al., 1997; and Stodder, 1997). When all degrees of severity, all tooth classes, and all types of hypoplasia are considered, the precontact Chomorro skeletal remains from the site of Apurguan, Guam exhibit EH with equal frequency in both sexes (Douglas et al., 1997). However, when the sample is age-controlled, female teeth have a significantly greater frequency of hypoplasia in the young and middle-aged categories (Table 11).

Sex differences in LEH prevalence are absent from a composite sample of Chomorro skeletal series derived from the islands of Guam, Rota, Saipan, and Tinian

TABLE 11. Enamel hypoplasia prevalence by sex for Pacific Island skeletal series

| Study group | Subgroup | Prevalence by sex | Significance | Source |
|-----------------|---------------|--|--|---------------------------|
| Mariana Islands | Apurguan | F = 25.1% (62/247) M = 20.6% (68/330) | $\chi^2 = 1.635$ $P = 0.201$ (n.s.) | Douglas et al., 1997 |
| Mariana Islands | Apurguan | F = 37.3% (31/83) M = 22.2% (24/208) | $\chi^2 = 5.258$ $P < 0.05$ | Pietruswesky et al., 1997 |
| | Fujita | F = 15.4% (4/26) M = 44.2% (19/43) | $\chi^2 = 4.822$ $P > 0.05$ | |
| | Leo Palace | F = 10.0% (1/10) M = 100% (15/15) | $\chi^2 = 17.368$ $P > 0.05$ | |
| | Guam total | F = 25.7% (36/140) M = 32.2% (58/180) | $\chi^2 = 1.608$ $P > 0.05$ | |
| | Mariana Total | F = 30.2% (48/159) M = 34.2% (69/202) | $\chi^2 = 0.640$ $P > 0.05$ | |

(Pietruswesky et al., 1997). However, inter-island variation exists in the direction of sex differences in prevalence, with adult males exceeding adult females in Fujita and Leo Palace, yet by contrast the larger Apurguan skeletal sample shows a reversal in direction of prevalence with females (37.3%) exceeding males (22.2%). The frequency and age distribution of LEH among 293 individuals from the late prehistoric Latte Period populations of Guam is discussed in detail by Stodder (1997). Chronological patterning of LEH prevalence and co-variation of LEH with skeletal markers of stress are the primary topics of this contribution. Sex differences in prevalence are not documented or discussed, but the fact that this high status sample exhibits a high frequency of LEH is noteworthy (Stodder, 1997).

Maya. The health of prehistoric Maya have been under investigation by skeletal biologists since the early 1970s, but a recent revitalization of interest has developed concerning the causes responsible for the collapse of the Classic Mayan civilization. The idea of a gradual increase in nutritional stress and decline in health leading up to the collapse has come under close scrutiny from paleodietary and paleopathological perspectives (Wright and White, 1996). Geographical and temporal variation in markers of physiological stress remain incompletely documented for Mayan skeletal series, making changes in prevalence difficult to interpret. Here we examine the issue of sex differences among Maya skeletons without assuming increasing levels of nutritional

stress in late or colonial periods. LEH prevalence by sex for Mayan skeletal series are presented in Table 12.

Saul has reported LEH frequencies in numerous Mayan skeletal series, including Altar de Sacrificios (Saul, 1972), Lubaantun (Saul, 1975), Siebal (Saul, 1973), and Tancah (Saul, 1982). His paleopathological analysis of Maya human remains from Altar de Sacrificios (Saul, 1972) provided convincing early support for the idea that poor health and nutritional stress may have had a role in the collapse of the Maya (Wright and White, 1996). In a recent analysis of LEH in the remains from Cuello, Saul and Saul (1997) report a slight trend for higher prevalence in females than males. They state that ". . . during the entire Formative Period at Cuello (not counting the two mass burials) the occurrence of linear EH was higher in females (63%) than in males (49%)" (Saul and Saul, 1997, p. 35). However, our analysis of Saul's data shows that differences in prevalence by sex are not statistically significant. This lack of statistical significance was evident whether individuals from the two mass burials were included or excluded from the analysis (see Table 12).

Among colonial period skeletons at Tipu, Belize, Cohen and associates report two different measures of LEH prevalence: (1) the mean number of hypoplastic lines per tooth, and (2) individuals with three or more hypoplastic events (Cohen et al., 1997). While males tend to display a higher mean number of hypoplastic lines per tooth in maxillary incisors and mandibular canines, the differ-

TABLE 12. LEH Prevalence by Sex Among the Maya

| Study sample | Variable | LEH prevalence | Significance | Data source |
|----------------------|--|--|---------------------------------|---------------------|
| Tipu, Belize | I ¹ (mean no. of defects/tooth) | F = 1.15 M = 1.56 | Not reported | Cohen et al., 1997 |
| | Mand. C (mean no. defects/tooth) | F = 1.47 M = 1.92 | Not reported | |
| | ≥3 LEH | F = 8.3% (3/36) M = 28.6% (16/56) | $\chi^2 = 5.477$ $P = 0.019$ | |
| Copán, Honduras | % individuals affected | F = 50% (2/4) M = 63% (5/8) | Sample too small | Hodges, n.d. |
| Cuello, Belize | Including mass burials | F = 62.5% (10/16) M = 48.7% (19/39) | $\chi^2 = 0.865$ $P = 0.352$ | Saul and Saul, 1997 |
| | Excluding mass burials | F = 62.5% (10/16) M = 58.2% (32/55) | $\chi^2 = 0.096$ $P = 0.757$ | |
| Altar de Sacrificios | % individuals affected | F = 100% (10/10) M = 86% (19/22) | Not given | Saul, 1972 |
| Lamanai, Belize | % individuals affected | No significant difference by gender | n.s. | White, 1988 |
| Lamanai, Belize | Mean no. of defects/tooth | F = 0.34 (n = 50) M = 0.39 (n = 61) | n.s. | White, 1997 |
| Copán, Honduras | % individuals affected | M slightly > F F = 43%, M = 45% | $P > 0.05$ | Whittington, 1992 |

ences are not statistically significant. When frequent growth disruptions are compared, males exhibit three or more hypoplastic lines significantly more frequently than females. This finding parallels the results for Afro-American slaves reported by Blakey et al. (1994) and by Rathbun (1987), who also found that when more severe disruptive events were considered, males had significantly more enamel defects than females.

Among the low status Maya of Copan, Honduras, LEH prevalence in males is somewhat greater than LEH prevalence in females, but the difference is not significant (Whittington, 1992). In another study of a small sample of human remains from Copan, Hodges (no date) reports slightly greater, but not significantly different, incidence among males (62.5%) than among females (50.0%). These findings are in agreement with reports of LEH among the prehistoric Maya of Lamanai, Belize, who lack significant sex differences in LEH prevalence at the age of weaning (White et al., 1994).

Postclassic and historic Maya remains from the archaeological site of Lamanai were studied by Wright (1990), who reports no significant sex difference in EH prevalence for *mild, shallow expressions* of LEH.

Summary of skeletal series with unknown stress levels. This survey of skeletal groups with unknown levels of physiological stress includes a wide range of world populations. Despite the diversity of research methods and reporting procedures, we believe intra-observer variation is sufficiently consistent within each investigation to permit the evaluation of inter-sex differences in EH. Danforth and Gilberti (1992) have found that intra-observer error in scoring LEH defects is generally low. In 37 comparisons of EH prevalence by sex among such skeletal series, the majority, 78.4% (29/37), found no significant difference. Males exceeded females in EH prevalence in 13.5% (5/37) of the comparisons, while females had a higher prevalence than males in only 8.1% (3/37) of the comparisons. This distributional pattern of sex differences in EH is very similar to results derived from 34 comparisons in groups with independent evidence of physiological stress (see "stressed" section above). In stressed groups, males and females were not significantly different in EH prevalence in 70.6% (24/34) of comparisons, males exceeded females in 17.6% (6/34), and the reverse (F > M) was true in 11.8% (4/34) of the comparisons. The similar patterning of sex differences among stressed groups and

among those with unknown stress levels may indicate that the overall severity of stress does not differentially impact the sexes. Another possible, though less likely, explanation is that groups whose stress level is undocumented or indeterminate are actually quite similar to the stressed groups and therefore yield a similar data distribution. However, since these reports do not usually provide descriptions of sex bias in treatment of offspring or differential environmental impact by sex, this inference must remain tentative.

Finally, some variability in the data is undoubtedly explained by random sampling error introduced by small study samples. In the Marianas Islands, for example, while two small samples indicate that male LEH prevalences are greater than those females, larger and age-controlled series suggest the opposite — that females have higher LEH prevalence than males — within the same cultural region.

Living samples. Studies reporting EH in the permanent teeth of living samples with unknown levels of stress are reviewed here (Table 13). Two studies, one conducted in Hong Kong (King, 1989), the other in Jordan (Al-Abassi, 1997), report statistically significantly higher frequencies of EH in boys relative to girls. In their large sample of 484 girls and 460 boys, King et al. (1989) found that girls had significantly higher frequencies of groove defects and missing enamel than boys. The authors state that multiple teeth were often affected, implying metabolic disturbance. While there is widespread cultural preference for sons in China, this preference is expressed most strongly in rural areas, and is least pronounced in urban areas such as Hong Kong (Zhou and Corruccini, 1998). While this study superficially appears to point toward male vulnerability, no evidence of population-wide stress, independent of EH, is presented. Al-Abassi (personal communication) believes that he found higher frequencies of EH in Jordanian boys (Al-Abassi, 1997) relative to girls because girls receive preferential treatment from their fathers and are less exposed to environmental stressors. Thus, while these data might also appear to support the male

vulnerability hypothesis, they can be explained by enhanced protection of female children. Lukacs and Guatelli-Steinberg (1994) found that male-female differences in the mean number of LEH lines per tooth, in samples from Northwest India, were significant in six of 36 comparisons. Five of these six significant differences were male greater than female; one was female greater than male. While this result too may suggest higher male vulnerability, it is notable that in none of the six caste/tribal groups studied were sex differences in LEH prevalence (percent of individuals affected in each group) significant.

Several studies report statistically insignificant differences of EH in boys relative to girls. These studies fall into two categories: those in which both male and female EH frequencies are low, and those studies in which the overall population incidence is high, but sex differences are insignificant. In the first group (Table 13) are studies from New Jersey (Brucker, 1943), Sweden (Crossner and Holm, 1975; Samuelson et al., 1971), South Wales (Dummer et al. 1986, 1990), the Kingdom of Tonga (Hoffman et al., 1988), Japan (Iizuka, 1976), Nigeria (Osuji, 1990), and Boston (Needlemann, et al., 1991). Interestingly, Brucker (1943) found that boys had more hypoplastic first molars than girls, perhaps suggesting (our interpretation, not Brucker's) a chronological sex difference in EH expression. These studies neither challenge nor support the male vulnerability hypothesis: the overall population frequency of EH is low, and at least in one case (Dummer et al., 1990), the asymmetric distribution of defects indicates nonsystemic causation.

The studies of El-Najjar et al. (1978), Lukacs and Guatelli-Steinberg (1994), and Lukacs and Joshi (1992), however, involve high overall population incidences of EH. El-Najjar find that Cleveland males and females (both Blacks and Whites) do not significantly differ in their expressions of EH (reported by tooth count). Lukacs and Joshi (1992) and Lukacs and Guatelli-Steinberg (1994) specifically hypothesized that high castes, particularly from North India, would provide evidence of daughter neglect through elevated LEH frequencies

TABLE 13. EH prevalence by sex in samples from living groups with unknown stress

| Study sample | EH types | Subgroup | EH prevalence by sex | Significance | Data source |
|--------------------|--|---|---|-------------------------------------|--------------------------------------|
| Northern Jordan | Pits, lines, grooves (individuals with one or more defects) | | F = 57% (108/189) M = 72% (135/188) | $\chi^2 = 8.848$ $P = 0.003$ | Al-Abassi, (1997) |
| Hong Kong | Grooves, pits, missing enamel (individuals with one or more defects) | | Horizontal grooves: F = 24.8% (114/460) M = 34.7% (168/848) Missing enamel: F = 27.4% (126/460) M = 34.7% (168/484) | $P = 0.003$ $P = 0.018$ | King et al. (1989) |
| Newark, New Jersey | Pitted, furrowed or absent enamel (individuals with one or more defects) | Blacks Whites | F = 16.6% (3/18) M = 0% (0/17) F = 3.8% (34/913) M = 4% (39/973) | n.s. n.s. | Brucker (1943) |
| Sweden | Hypoplasia not defined (individuals with one or more defects) | | F = 8% (6/70) M = 6% (5/79) | n.s. | Crossner and Holm (1975) |
| Sweden | Symmetrical "external" enamel hypoplasia | | Not given | Sex differences reported to be n.s. | Samuelson et al. (1971) |
| South Wales | FDI DDE (1982) Index: individuals with one or more defects | | Pits: F = 0.3% (1/364) M = 0.8% (3/759) Grooves: F = 0.6% (2/364) M = 0.5% (2/759) Single missing: F = 3.8% (14/364) M = 2.0% (8/759) Multiple missing: F = 3.3% (12/364) M = 3.4% (14/759) | All differences are n.s. | Dummer et al. (1986) |
| South Wales | FDI DDE (1982) Index: individuals with one or more defects | | Pits: F = 0.5% (2/398) M = 0.0% (0/383) Grooves: F = 0.3% (1/398) M = 0.0% (0/383) Single missing: F = 5.3% (21/398) M = 4.3% (17/383) Multiple missing: F = 2.8% (11/398) M = 3.3% (13/383) | All differences are n.s. | Dummer et al. (1990) |
| Kingdom of Tonga | Modified FDI DDE (1982) Index: individuals with one or more defects | | Not given | Sex differences reported to be n.s. | Hoffman et al. (1988) |
| Japan | Hypoplasia (not defined) | Individuals with one or more hypoplasias Individuals with bilateral hypoplasia | F = 4.3% (16/395) M = 6.3% (24/379) F = 1.3% (5/395) M = 2.4% (9/379) | n.s. for both sub-groups | Iizuka et al. (1976) |
| Nigeria | Hypoplasia (not defined) | | F = 6.2% (42/683) M = 4.6% (31/676) | n.s. | Osuji (1990) |
| Boston | EH in primary teeth | | F = 32.0% (90/281) M = 34.9% (80/228) | n.s. | Needleman et al. (1991) |
| Cleveland | Pits, lines, grooves; data given by tooth count | Black White | M/F differences given for incisors, canines M/F differences given for incisors, canines | All n.s. All n.s. | El-Najjar et al. (1978) |
| India | LEH (individuals with matched defects) | Variety of caste/tribal groups | Incidence varies by groups Mean defects/tooth; 5/36 comparisons M > F; 1/36 comparisons F > M | n.s. All significant | Lukacs and Gattelli-Steinberg (1994) |
| Northwest India | LEH (individuals with matched defects) | Bhils Garasias Rajputs | F = 87.7% (86/98) M = 81.4% (80/97) F = 74.4% (58/78) M = 82.4% (77/93) F = 68.0% (34/50) M = 74.4% (99/133) | All n.s. | Lukacs and Joshi (1992) |

in female children. Because they did not obtain this result, these authors suggest that greater male vulnerability serves to equalize sex difference in LEH when cultural practices cause the health of daughters to be neglected.

Non-human primates. Relative to the number and diversity of EH studies conducted in humans, the literature on EH in non-human primates is sparse. Colyer published initial findings on non-human primate EH in 1936; however, extensive research on this topic did not begin until the mid 1980s (Eckhardt, 1992; Eckhardt et al., 1992; Eckhardt and Protsch von Zieten, 1993; Guatelli-Steinberg, 1998; Guatelli-Steinberg and Lukacs, 1998; Lukacs, 1999b (in review); Moggi-Cecchi and Crovella, 1991, 1992; Newell, 1998; Skinner, 1986b; Skinner et al., 1995; Skinner and Guatelli-Steinberg, 1997; Stottlemire, 1998; Vitzthum and Wikander, 1988, Zhang, 1987). Twenty studies reporting EH frequencies were examined for information regarding sex differences. Of these, eight included information on EH incidence by sex (40%). These studies are reviewed below and summarized in Table 14.

All eight of these studies are based on skeletal collections, and as a result, behavioral evidence of preferences for male or female offspring is not known for the individuals involved. Within primate populations, there appears to be variation in sex-biased parental investment related to the dominance rank of mothers (Hrdy, 1987); however, none of these eight studies addresses this issue with behavioral data. Indications of stress other than EH are also not included in these studies, although minimal stress can be inferred in the provisioned Cayo Santiago rhesus population (Guatelli-Steinberg and Lukacs, 1998) and potential sources of disease have been identified for African ape samples (Skinner, 1986; Skinner et al. 1995).

As in humans, LEH in non-human primates shows a range of expression, from more "mild" defects with shallow depth to more "severe" groove defects. This range of expression is shown in Figure 3.

EH incidence. Statistically significant differences in the incidence of EH are evident in two of the eight studies (Guatelli-Steinberg and Lukacs, 1998; Newell, 1998). The higher frequencies of LEH in female versus male rhesus monkeys (Guatelli-Steinberg and Lukacs, 1998), based on matching LEH defects on any antimeric pair, reflects the fact that male lower P3s are affected by heavier wear than those of females. The wear differential combined with the fact that the lower P3 is preferentially affected by LEH in this sample (Guatelli-Steinberg and Lukacs, 1998), results in a higher female incidence of LEH relative to males. The sex difference in LEH incidence is not apparent when individuals with worn lower P3s are removed from the analysis. This result is therefore not significant with respect to the question of biologically meaningful differences in EH expression.

Newell's (1998) study is the first to systematically investigate sex differences in LEH in a wide variety of taxa. Newell (1998) found that across the primate order ($N = 2646$), males had a significantly greater incidence of EH than females ($p < 0.001$). Here, individuals are considered to have been affected by LEH if they had one or more defects on any permanent tooth. Anthropoid males and platyrrhine males had significantly more LEH than anthropoid females and platyrrhine females, respectively. The difference in LEH prevalence for male and female catarrhines was not significant. Within the great apes, gorillas ($N = 60$ females, 79 males) and chimpanzees ($N = 47$ females, 30 males) did not show significant LEH sex differences, while orangutans ($N = 24$ females, 24 males) did not (Fig. 4) (Newell, 1998). The majority of individual species in Newell's sample showed no significant sex difference in EH (14 species). Males had significantly more LEH than females in just two species: *Pongo pygmaeus* and *Cebus apella*. Females had significantly more LEH than males in just one species: *Presbytis rubicunda*.

Lukacs (1999b) found no significant sex differences in the LHPC incidence of great ape specimens (either collectively or by taxon) housed at the Cleveland Museum of

TABLE 14. Non-human primate enamel hypoplasia expression by sex

| Source | Species or other taxonomic group/N | Skeletal collection ^{1/} geographic source | Teeth examined/ scoring method | Defect type/overall frequency | Incidence of defects in males vs. females (asterisks denote significance at $P \leq 0.05$) | Other measures of sex differences in DDE's (asterisks denote significance at $P \leq 0.05$) | Comments |
|---|--|---|---|--|---|---|---|
| Guatelli-Steinberg (1998) | <ul style="list-style-type: none"> • <i>H. lar</i> N = 34; Males = 14 Females = 20 • <i>G. gorilla</i> N = 13 Males = 5 Females = 8 • Combined great ape sample (<i>G. gorilla</i>, <i>P. troglodytes</i>, <i>P. pygmaeus</i>) N = 27 Males = 12 Females = 15 | <ul style="list-style-type: none"> • <i>H. lar</i> MCZ/Thailand • <i>G. gorilla</i> MCZ^{1/}/Cameroon • Combined great ape sample MCZ/ —Cameroon (<i>G.g</i> and <i>P.t.</i>) —Borneo and Sumatra (<i>P.p</i>) | <p>Individuals considered to have LEH if ≥ 1 pair of matched defects on LC;</p> <p>Counts of LEH per individual = number of matched defects on an antimeric pair of LC; faint lines through grooves included;</p> <p>Minimally worn LC: < 2.5 mm estimated missing</p> | <p>LEH</p> <ul style="list-style-type: none"> • <i>H. lar</i>: 38.2% • <i>G. gorilla</i>: 38.5 • Combined great ape sample: 51.9% | <ul style="list-style-type: none"> • <i>H. lar</i> Males: 28.6% Females: 45.0% • <i>G. gorilla</i> Males: 40.0% Females: 37.5% • Combined great ape sample Males 66.7% Females 40.0% | <ul style="list-style-type: none"> • <i>H. lar</i> Number of matched defects for LC not different in m vs. f *<i>G. gorilla</i> number of matched defects for LC greater in males than females *Combined great ape sample Number of matched defects for LC greater in males than females | <p>Possible Interpretation: canine sexual dimorphism is related to LC defect counts</p> |
| Guatelli-Steinberg and Lukacs (1998) | <ul style="list-style-type: none"> • <i>M. mulatta</i> N = 360 Males = 179 Females = 181 | <ul style="list-style-type: none"> • CPRC^{2/}/Cayo Santiago (provisioned) | <p>Individuals considered to have LEH if ≥ 1 pair of matched defects on any antimeric pair; all permanent teeth examined but most defects occurred on LP3</p> | <p>LEH: faint lines, lines, and grooves. 17% of sample</p> | <ul style="list-style-type: none"> *Males 10% Females 24% | <p>None</p> | <p>Sex difference caused by wear on male LP3s obscuring faint lines (sex difference disappears when specimens with worn LP3s removed)</p> |
| Guatelli-Steinberg and Skinner (in press) | <ul style="list-style-type: none"> • Sympatric Asian primates (including cercopithecoids and hominoids) N = 97 Males = 49 Females = 46 • Sympatric West African primates (including cercopithecoids and hominoids) N = 115 Males = 55 Females = 60 | <ul style="list-style-type: none"> • Asian primates MCZ^{1/}/ Kinabatangan River • African primates Powell-Cotton/Cameroon and Congo | <ul style="list-style-type: none"> • Asian primates: Individuals considered to have LEH if ≥ 1 pair of matched defects on any antimeric pair; all permanent teeth examined; • African primates Individuals considered to have LEH if any defect occurred on permanent left UC or UII; | <p>LEH: faint lines, lines, and grooves.</p> <ul style="list-style-type: none"> • Asian Primates: 24% • African Primates: 50% | <ul style="list-style-type: none"> • Asian primates Males 31% Females 15% • African primates Males 56% Females 47% | <p>None</p> | <p>None</p> |
| Lukacs (1996) | <ul style="list-style-type: none"> • <i>Gorilla</i> N = 53 • <i>Pan</i> N = 50 • <i>Pongo</i> N = 25 | <ul style="list-style-type: none"> • Cleveland Museum of Natural History and the Smithsonian Institution • National Museum of Natural History | <ul style="list-style-type: none"> • Deciduous canines; Individuals with one or more defects considered to be affected | <p>LHPC</p> <ul style="list-style-type: none"> • <i>Gorilla</i> 88.7% • <i>Pan</i> 22.0% • <i>Pongo</i> 88.0% | <p>Sex differences in LHPC incidence not significant</p> | <p>None</p> | <p>None</p> |

(Continued)

TABLE 14. (continued)

| Source | Species or other taxonomic group/N | Skeletal collection ¹ /geographic source | Teeth examined/scoring method | Defect type/overall frequency | Incidence of defects in males vs. females (asterisks denote significance at $P \leq 0.05$) | Other measures of sex differences in DDE's (asterisks denote significance at $P \leq 0.05$) | Comments |
|---|--|---|---|--|--|---|--|
| Newell (1998) | <ul style="list-style-type: none"> All primates N = 2646 Males = 1344 Females = 1302 Anthropoids N = 2533 Males = 1290 Females = 1243 Platyrrhines N = 1018 Males = 499 Females = 519 Catarrhines N = 1516 Males = 792 Females = 724 | Variety of museum collections/locations | All permanent teeth; individuals scored as LEH-positive if they have one or more defects on any tooth | LEH Frequencies given for the number of taxa affected within larger taxonomic categories. These frequencies are not reproduced here. | <ul style="list-style-type: none"> *All Primates: males > females *Anthropoids: males > females *Platyrrhines: males > females • Catarrhines: M/F difference is non-significant | Not given | Newell gives male-female differences for 17 species; these are not reproduced here (see text for discussion) |
| Skinner (1986) | <ul style="list-style-type: none"> <i>Pan</i> N = 110 Males = 35 Females = 75 <i>Gorilla</i> N = 119 Males = 53 Females = 66 | <ul style="list-style-type: none"> Powell Cotton Museum/Cameroon | Permanent incisors and canines | LEH <ul style="list-style-type: none"> • <i>Pan</i> 58% • <i>Gorilla</i> 76% | <ul style="list-style-type: none"> • <i>Pan</i> Males 66% Females 55% • <i>Gorilla</i> Males 81% Females 71% | Range of defect counts on lower canines: <ul style="list-style-type: none"> • <i>Pan</i> Males 1–11 Females 1–6 • <i>Gorilla</i> Males 1–9 Females 1–6 | Range of defect counts is higher for males than it is for females of both genera |
| Stottlemire (1998) (published abstract) | <ul style="list-style-type: none"> <i>Pan</i> N = 98 Males = 36 Females = 62 <i>Gorilla</i> N = 229 Males = 143 Females = 86 | Hamman Collection of the Cleveland Museum of Natural History/Cameroon | Not known/Single and multiple hypoplasias recorded | Enamel Hypoplasia <ul style="list-style-type: none"> • <i>Pan</i> 80.6% • <i>Gorilla</i> 27.5% | Not given in abstract, but pers. comm. from author is that sex differences are not significant | None | None |
| Vitzthum and Wikander (1988) (published abstract) | <i>C. aethiops</i> , <i>C. mitis</i> , <i>Papio</i> , <i>Mandrillus</i> , <i>Pan</i> , <i>Gorilla</i> , <i>Pongo</i> , <i>Hylobates</i> , <i>Sivapithecus</i> , Ceboid species Total N = 2000 | Various museum collections: not specified | Not known | Enamel hypoplasia <ul style="list-style-type: none"> • Cercopithecoïd, Ceboid, gibbon taxa: 1.5–3% • African apes: 95% • <i>Sivapithecus</i>: nearly all with hypoplasia | Sex differences in incidence not significant | | |

¹ MCZ: Museum of Comparative Zoology; CPRC: Caribbean Primate Research Center.

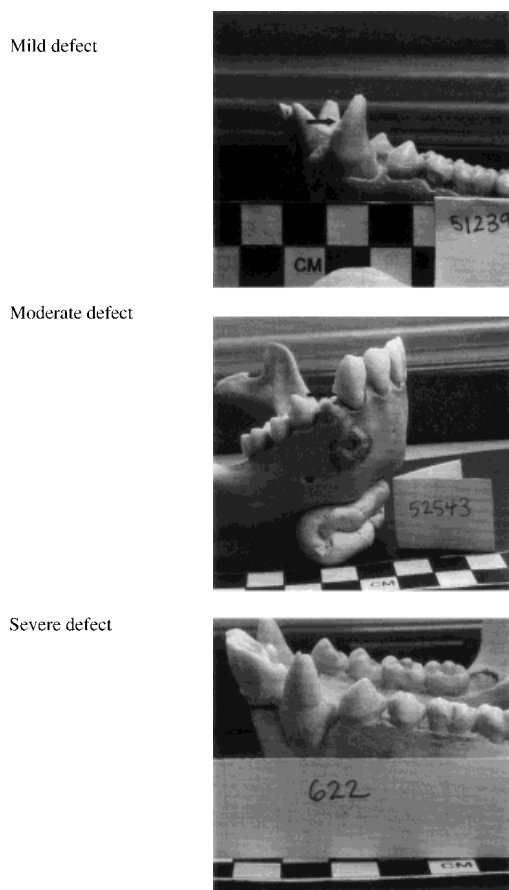


Fig. 3. Range of LEH expression in chimpanzee teeth. Defects rated as mild (LACMNH 51239), moderate (LACMNH 52543), and severe (LACMNH 622). Specimens are from the Los Angeles County Museum of Natural History (LACMNH).

Natural History and the Smithsonian Institution National Museum of Natural History (Table 15). These results were subsequently confirmed by data on LHPC prevalence derived from sympatric species of *Gorilla* and *Pan* housed in the Powell-Cotton Museum (Birchington, Kent). Examples of LHPC defects in orangutan and gorilla are shown in Figure 5. Differences in frequency of LHPC between the sexes in this sample are presented in Table 15 and are also not significant: for each genus separately, *Pan* ($n = 42$) and *Gorilla* ($n = 61$), and in the collective sample of both genera analyzed together ($p = 0.352$) (Lukacs, in preparation). A notable trend in the data from both studies is

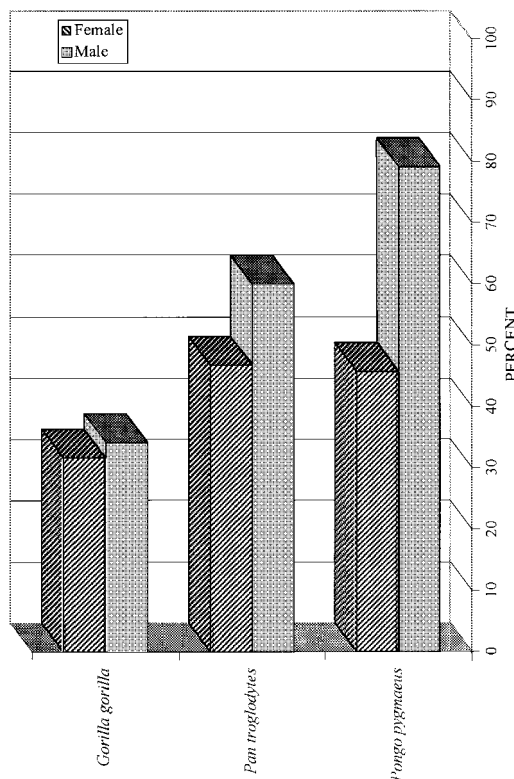


Fig. 4. LEH incidence by sex in *Gorilla*, *Pan*, and *Pongo*. (Reprinted from Newell, 1998.)

that while chimpanzees exhibit a lower overall defect frequency relative to gorillas, they display a greater, though not significant, sex difference in defect prevalence than gorilla (Fig. 6).

These studies on EH incidence are difficult to interpret because so little is known about the local environments and behavior of these primates during life. Evaluation of the male vulnerability/female buffering hypothesis in non-human primates requires this information. Eckhardt and Protsch von Zieten (1993) have shown that chimpanzee deciduous teeth are relatively free of linear defects, but do exhibit hypoplastic pits. Lukacs has documented LHPC in the deciduous teeth of orangutans, chimpanzees, and gorillas. With future research on deciduous tooth calcification in non-human primates, it may be possible to determine which hypoplastic pits and localized hypoplasias are forming in utero. If so, these prenatal de-

TABLE 15. Enamel hypoplasia in primary canine teeth of great apes

| a. CMHN and NMNH samples ¹ | | | | | | | | |
|---------------------------------------|-----------------------------|--------------|-----------------------------|--------------|-----------------------------|--------------|-----------------------------|--------------|
| Sex | Chimpanzee | | Gorilla | | Orangutan | | Total | |
| | n | Affected (%) | n | Affected (%) | n | Affected (%) | n | Affected (%) |
| Female | 17 | 4 (23.5) | 20 | 18 (90.0) | 11 | 10 (90.9) | 48 | 32 (66.7) |
| Male | 6 | 1 (16.7) | 27 | 25 (92.6) | 10 | 9 (90.0) | 43 | 35 (81.4) |
| Total | 23 | 5 (21.7) | 47 | 43 (91.5) | 21 | 19 (90.5) | 91 | 67 (73.6) |
| M vs. F | $\chi^2 = 0.051; P = 0.822$ | | $\chi^2 = 0.046; P = 0.831$ | | $\chi^2 = 0.453; P = 0.501$ | | $\chi^2 = 1.832; P = 0.176$ | |

| b. Powell-Cotton Museum sample samples ¹ | | | | | | |
|---|-----------------------------|--------------|-----------------------------|--------------|-----------------------------|--------------|
| Sex | Chimpanzee | | Gorilla | | Total | |
| | n | Affected (%) | n | Affected (%) | n | Affected (%) |
| Female | 17 | 9 (52.9) | 35 | 30 (85.7) | 52 | 39 (75.0) |
| Male | 25 | 11 (44.0) | 26 | 23 (88.5) | 51 | 34 (66.7) |
| Total | 42 | 20 (47.6) | 61 | 53 (86.9) | 103 | 73 (70.9) |
| M vs. F | $\chi^2 = 0.324; P = 0.569$ | | $\chi^2 = 0.099; P = 0.753$ | | $\chi^2 = 0.866; P = 0.352$ | |

¹ CMNH: Cleveland Museum of Natural History; NMNH: National Museum of Natural History.



Fig. 5. Examples of LHPD expression in great apes. (A) RK-1. LHPD defects in orangutan (*Pongo*). Note small ovoid defect in the maxillary canine and the larger hypoplastic lesion in the mandibular canine. (B) M690. LHPD defects in *Gorilla*. Note the well demarcated

defect in the maxillary right deciduous canine, and the paired pits on the mandibular canine. From the Powell-Cotton Museum (Quex Park), Birchington, Kent, England.

fects could allow an assessment of the male vulnerability/female buffering hypothesis uncomplicated by sex-biased parental investment after birth.

Defect counts. There is some suggestion from two studies (Guatelli-Steinberg, 1998;

Skinner, 1986) as well as from previously unpublished data provided to the authors by Jacopo Moggi-Cecchi, that male great ape canines may have higher LEH counts (numbers of lines/grooves) than those of females. Skinner (1986) found that males had a greater range of defect counts than females

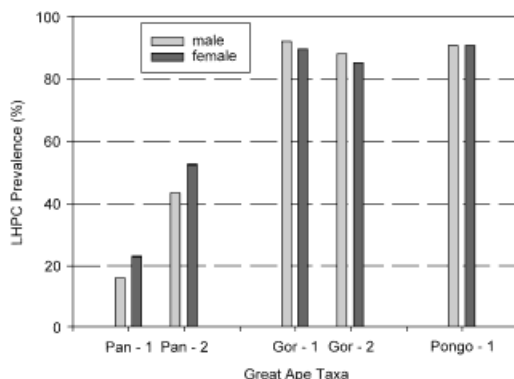


Fig. 6. LHPHC prevalence among great ape taxa. Bar height represents the percentage of individuals with one or more canine teeth exhibiting enamel defects. 1 = pooled samples from Cleveland Museum of Natural History and the Smithsonian Institution-Museum of Natural History; 2 = sample from Powell-Cotton Museum, Birchington, Kent, England.

in both chimpanzees ($N = 110$) and gorillas ($N = 119$). The highest number of defects on a male chimpanzee canine was 11 as opposed to 6 for a female's; the highest number for a gorilla male was 9 as opposed to 6 for a female. Guatelli-Steinberg (1998) found that for individuals with unworn or minimally worn lower canine antimeric pairs, sex differences in defect counts were not significant for Thailand gibbons ($N = 34$), but were significant for a small sample of Cameroon gorillas ($N = 13$) as well as for a combined great ape sample ($N = 27$). In both the gorilla and combined great ape samples, males had higher numbers of matched defect pairs on their lower canines than females ($P \leq 0.01$). Data shared by Jacopo Moggi-Cecchi, summarized in Table 16, show a similar pattern by sex in large samples of African apes. Note that the range of defect counts on the lower canine is greater for males than for females in five of the six species sampled.

These data on sex differences in LEH counts on great ape canines are preliminary, requiring further testing on larger samples with unworn canines. At the present time, these data suggest the potential for a sex difference in LEH expression that does not necessarily affect differences in incidence, but does involve differences in the number of stress episodes the canines record. Differences in crown formation times of highly

TABLE 16. Range of lower canine LEH counts in Jacopo Moggi-Cecchi's samples*

| Hominoid subspecies | Range of LEH counts on LC |
|---------------------------|--|
| <i>G. g. gorilla</i> | male 0–8 ($n = 31$) female 0–5 ($n = 19$) |
| <i>G. g. graueri</i> | male 1–7 ($n = 18$) female 1–6 ($n = 15$) |
| <i>P. paniscus</i> | male 1–8 ($n = 16$) female 1–6 ($n = 18$) |
| <i>P. t. shweinfurthi</i> | male 1–9 ($n = 24$) female 1–8 ($n = 22$) |
| <i>P. t. troglodytes</i> | male 2–5 ($n = 7$) female 2–16 ($n = 8$) |
| <i>P. t. verus</i> | male 1–14 ($n = 5$) female 1–6 ($n = 3$) |

* Gorillas from the Natural History Museum of London and Musée Royal de L'Afrique Central Tervuren/Central and West Africa; Chimpanzees from Tervuren/Central and West Africa. LEH includes thin lines as well as grooves.

sexually dimorphic canines (see section on intrinsic tooth attributes) may help explain this result: large male canines may record more stress events than smaller female canines because they have greater opportunity during development to do so. The effect of sex differences in the duration of canine enamel formation might only be expected to differentially impact defect counts when stress occurs recurrently and frequently during crown formation. Recurrent stress can be inferred for great apes who, in contrast to monkeys, often record multiple episodes of stress in their teeth (Guatelli-Steinberg and Skinner, in press; Skinner, 1986; Skinner et al, 1995). Figure 1 contains a photograph of an orangutan canine with multiple LEH defects.

Gibbon mandibular canines appear to record more episodes of stress than monkey mandibular canines, but less than the number recorded by great ape canines (Guatelli-Steinberg, 1999). The highest number of matched lower canine defect pairs for a sample of 92 gibbons was four; while the highest number for a sample of 63 great apes was eight (Guatelli-Steinberg, unpublished data). That there was no sex difference in defect counts for gibbon mandibular canines might therefore reflect two factors: the narrower range of defect counts in the gibbon sample and the smaller degree of canine sexual dimorphism relative to the great apes. While canine crown calcification data for gibbons is not currently available, the small degree of sexual dimorphism in gibbon canine crown height (Plavcan, 1990)

may imply a small sex difference in gibbon canine crown calcification (as per the suggestion of Macho and Wood regarding sex differences in hominoid canine crown calcification). Thus, it is possible that no sex differences were observed in the defect counts of gibbons, in part, because there is little difference between the sexes in the opportunity available to record stressful events.

DISCUSSION

Interpreting sex differences in EH: Deciduous teeth

The study of EH prevalence in deciduous teeth offers a unique opportunity to evaluate issues of female buffering and male environmental sensitivity in both humans and great apes. A major advantage of examining EH in deciduous teeth is that most human anterior tooth crown enamel is formed prenatally. Approximately 1/3 (canines) to 5/6 (central incisor) of the enamel of human anterior teeth forms in utero. In the absence of prenatal sex determination (ultrasound or amniocentesis) cultural bias against the fetus during gestation will not occur, particularly in rural agricultural communities where such technology is absent. This natural mechanism for "experimental" control allows us to "neutralize" potential cultural bias in parental investment by sex for the fetal period of amelogenesis. The chronology of deciduous dental development among great apes is not well known, but enamel formation in anterior teeth is at least partly prenatal, and differential treatment of the fetus by sex extremely unlikely.

The intra-uterine environment is thought to offer significant advantages to the fetus by buffering potentially stressful fluctuations in the external environment. For this reason, some investigators propose that EH in deciduous teeth will typically be low in prevalence, and by contrast, severe environmental perturbations will be required to produce EH in deciduous teeth during prenatal development (Cook and Buikstra, 1979; Sciulli, 1978). For these reasons we feel that the analysis of variation in inter-sex prevalence of EH in deciduous teeth constitutes one of the most informative sources of information on the issues of male sensitivity and female buffering.

Analysis of the two types of EH in human deciduous teeth included in this survey, LEH and LHPC, yield essentially the same result: significant differences by sex are rare. Studies of LEH among low and very low birth weight neonates and among malnourished children from "Third World" countries reveal that sex differences are usually absent. When LHPC prevalence is analyzed by sex, 18 comparisons were possible, yet only 11.1% (2/18) showed significant differences. In all cases where significant differences were discovered, males exhibited a greater prevalence of LHPC than females, though the percentage difference separating the sexes was as small as 4%.

These results are interpreted to indicate a weak tendency for males to be more environmentally sensitive to stress during fetal development than females. Limitations of data collection and presentation may actually conceal a more marked sex difference in prenatal EH than our survey detected. None of the clinical or epidemiological reports of EH in human deciduous teeth provided separate statistical analysis of LEH or LHPC by the time of formation: prenatal versus postnatal. We suspect that the pooling of pre- and postnatal EH lesions in the analysis of defect frequency may mask greater evidence of female buffering. We anticipate that future analysis of prenatal EH prevalence separately from postnatal EH will reveal more conclusive evidence of male environmental sensitivity.

A complicating factor in the analysis of sex differences in the EH expression of deciduous teeth is the possibility of differential mortality in utero. Stinson (1985) notes that female fetuses are more likely than male fetuses to survive the third trimester of "stressful" pregnancies, such as those in which mothers suffer from diseases or accidents. Under stressful conditions, then, if males have significantly greater mortality late in gestation, they might be expected to exhibit less EH than females: the most vulnerable males might not survive to record stress episodes in their teeth. If this is the case, one possibility for the lack of stronger evidence of male vulnerability in the EH literature could be the result of differential mortality effects.

EH in the deciduous teeth of great apes has rarely been studied and the LHPC lesions reported here show no significant differences by sex. This finding holds for all three taxa studied, *Gorilla*, *Pan*, *Pongo*, regardless of the overall variation in defect prevalence between taxa. Results obtained from different museums and from different original source locations also confirm the absence of significant differences by sex. Deciduous LHPC data for great apes is subject to similar but more extensive limitations than human LHPC data. The most problematic issue is incomplete data regarding the chronology of deciduous dental calcification. In the absence of baseline data on the timing of dental formation, EHs cannot be segregated into pre- and postnatal developmental periods for separate statistical analysis.

While apes and humans exhibit significant differences in LHPC prevalence, they are similar in the very low frequency of inter-sex differences. Interpreting the meaning of large inter-generic variations in LHPC frequency among hominoids and deciphering the underlying reasons why all living great apes lack significant sex differences in the trait will require more research into fundamental questions regarding defect etiology.

In sum, data for EH in the deciduous dentition of humans does not conclusively support the idea of female buffering, the most common finding is that the sexes are equally affected. Weak support was found for enhanced male sensitivity in humans, but this finding is based on a small number of studies, and may be compromised by the pooled analysis of pre- and postnatal defects. Great apes and human are similar in that sex differences LHPC are rare or absent.

Interpreting sex differences in EH: Permanent teeth

Samples with direct evidence of stress (such as that provided by historical records or nutritional assessments) and with information regarding cultural practices provide the least ambiguous opportunities for assessing the potential effect of enhanced female buffering on sex differences in EH expres-

sion. However, in the cases examined in this review, there is no clear association between high levels of stress and greater expression of EH in males. While there is a preference for male children in China (Zhou and Corrucini, 1998), sex differences in the incidence of EH are nonsignificant during a lower stress period (after the Great Chinese famine) as well as during a higher stress period (the famine years). This result argues against a strong influence of a hypothesized male vulnerability on EH expression. Detailed information about cultural practices regarding the provisioning of male and female children would help to clarify this case. As Hrdy (1987) points out, stated preferences for children of one sex or the other may not be reflected in actual practice.

Two of the clearest examples of how cultural practices can strongly impact sex differences in EH, and override the potential effect of greater male vulnerability to environmental stress, are shown in the study by Goodman et al. (1987, 1991) of children in Solis, Mexico and adolescents in Tezonteopan. These studies provide description of cultural practices favoring male children in nutritionally stressed populations and demonstrate that higher frequencies of EH in girls are statistically significant.

Archaeological samples which document both direct evidence of high stress and of cultural preferences are limited to one: Fenton's (1998) study of Grasshopper Pueblo. Fenton argues that daughters were preferred in this matrilineal society resulting in elevated frequencies of EH in males that are statistically significant. If cultural preferences remained constant through time, then during later periods of occupation, when environmental stress increases, potentially more vulnerable males would be expected to exhibit even greater differences from females in their prevalence of EH. Yet, during these later, more stressful periods, the male-female difference is statistically insignificant.

Other samples with indirect evidence of stress, such as those of slave populations and low SES, are generally inconclusive regarding the question of enhanced female buffering because cultural practices are usually not known. For example, the intriguing

studies of enslaved Afro-American samples from South Carolina (Rathbun, 1987) and Maryland/Virginia (Blakey et al., 1994) suggest that males record more episodes of "severe" disruption in their enamel than do females. Males had significantly higher EH frequencies in poor rural villages in Cameroon, while in less stressed urban groups EH frequencies were approximately equal (Maunder et al., 1992). However, since cultural practices are unknown, there is the potential of sex bias against male children. Examined as a whole, these studies with indirect evidence of stress most often show insignificant sex differences in EH prevalence. Yet, it is notable that when statistically significant sex differences are found in the studies reviewed here, they are all male greater than female (Blakey et al., 1994; El-Najjar et al. 1978; Maunder et al., 1992; Rathbun, 1987). These observations may indicate a slight influence of higher male vulnerability. On the other hand, without knowledge of cultural contexts, these observations might also reflect daughter preference. This latter possibility is not unlikely, given the prospect that parents in low status or indigent groups might favor daughters as a result of facultative adjustment of offspring care as suggested by the Trivers-Willard hypothesis (Hrdy, 1987).

This review also considered living and skeletal samples as well as non-human primates in which stress levels are unknown. These studies predominantly reveal non-significant sex differences in EH prevalence. Five studies in humans (Al-Abassi, 1997; Cohen et al., 1997; Douglas et al., 1997; Saunders and Keenleyside, 1999; Webb, 1995) and one study in non-human primates (Newell, 1987) demonstrate statistically significantly higher EH frequencies in males than females. In Al-Abassi's (1997) study, it seems that there is a cultural preference for daughters. Two studies in humans (Lukacs and Pal, 1993; Douglas et al., 1997) and two studies in non-human primates (Guatelli-Steinberg and Lukacs, 1998; Newell, 1998) involve statistically significantly higher expression of EH in females than males (although for Guatelli-Steinberg and Lukacs, 1998, this difference is not biologically significant as it results from a wear differential).

Although Newell (1998) does find that in *Presbytis rubicunda* females have higher EH frequencies than males, most species have nonsignificant sex differences in EH prevalence. Again, the overall picture from these studies with unknown stress levels seems to indicate that when sex differences are found, it is more often the case that males have higher EH frequencies than females.

That most cases of statistically significant sex differences in EH involve higher frequencies in males may suggest that there could be a slight effect of male vulnerability on the expression on EH. Most often in smaller samples, sex differences are nonsignificant. However in very large samples, for example Zhou and Corrucinni's (1998) combined Chinese sample of 3014 and Newell's (1998) combined sample of 2646 non-human primates, males have frequencies of EH that are significantly higher than those of females. It is possible that large sample size affects statistical significance in these cases: for example none of Zhou and Corrucinni's subsamples, and most of Newell's, have nonsignificant sex differences. These results again points toward a weak effect of male vulnerability on EH prevalence: the effect may be detected most clearly when sample sizes are very large.

From the foregoing discussion of sex differences in EH in permanent teeth it is evident that to be able to evaluate the impact of enhanced female buffering on EH expression, environmental stress as well as cultural child-rearing practices must be well understood and documented. To assess the effect of female buffering, two situations in which there is severe environmental stress would be most useful: those in which sons and daughters are treated relatively equally with respect to access to essential resources, and those in which there is son preference. In the latter situation, if males exhibit higher EH frequencies than females despite the preferential treatment of sons, a case could be made for greater male vulnerability. Future research on this topic might benefit by studying samples in which either of these conditions applies, and by documenting sources of environmental stress as well as relevant child-rearing practices.

Finally, the examination of the influence of sex differences in the duration of crown formation on sex differences in EH expression was examined in great ape permanent teeth. While the findings are suggestive of male great apes recording more episodes of stress relative to females, as a result of the longer period of male crown formation, these findings need to be tested on larger samples controlled for environmental variation. In addition, a possible association between estimates of canine crown formation times for individual specimens (which could be achieved by counting perikymata) and defect counts on their unworn canines could be tested for statistical significance. Within-sex testing of this relationship between canine crown formation time and defects counts would further clarify the potential effect of sex differences in crown formation time on sex differences in defect counts. The study of EH in non-human primates has only recently begun to attract attention. Future studies could also focus on EH in living primates from a variety of habitats, documenting sources of stress and patterns of sex-biased investment in offspring.

CONCLUSIONS

This review considered developmental, environmental, and cultural factors involved in interpreting sex differences in EH expression. Based on the concept of enhanced female buffering against environmental stress, a data trend of higher EH incidence in males was expected for samples in which individuals experienced physiological stress. This review also examined the possibility that intrinsic sex differences in the composition or development of enamel might differentially affect EH expression in males and females. Such enamel differences were reviewed and generally determined to have either limited or unknown impact on EH expression. Of these factors, only one, the duration of canine crown formation, was expected to affect EH expression by sex. In great apes, canine crown formation times in males are longer than those of females and thus were expected to record more episodes of stress relative to female canines. Data from previous studies as well as the authors' recent studies on Indian schoolchildren and

non-human primates were used to examine these two issues: the impact of female buffering on EH expression in humans and the impact of sex differences in canine crown formation times on EH expression in great apes. The broad, comparative perspective adopted here included data from living samples, archaeological samples and skeletal series, and considered deciduous as well as permanent teeth. A summary of the main points follows.

- Samples for which there is either direct evidence of physiological stress (e.g., from historical or clinical records) or indirect evidence of physiological stress (e.g., low socioeconomic status) do not consistently exhibit higher male incidences of EH, and thus do not indicate that female buffering has a significant impact on EH expression. For some studies, because cultural practices regarding child-rearing are not reported, it is not possible to evaluate the potential impact of enhanced female buffering/male vulnerability. For studies focusing on EH in deciduous teeth, sex-biased investment in offspring is minimized as a confounding factor in evaluating the female buffering hypothesis. However, these studies as well do not indicate that female buffering is strongly influencing EH expression.
- LHPC prevalence in "stressed" samples also lends minimal support to the female buffering hypothesis. In most cases, sex differences in LHPC prevalence are non-significant.
- In living human, skeletal, and non-human primate samples, sex differences in EH expression were most often not statistically significant.
- Over all the male-female comparisons examined here, in both human and non-human primates, when sex differences are statistically significant there is a slight trend for them to be male greater than female. The authors interpret this result to suggest a weak influence of male vulnerability on the expression of EH that is most likely to be detected in samples of very large size (>1000 individuals).
- Suggestions for further study include the recommendation that researchers incorpo-

rate detailed descriptions of the child rearing practices of, and sources of physiological stress in, their study populations. Both cultural practices as well as environmental influences must be understood in order to evaluate if and how a hypothesized greater female resistance to stress relates to the by sex distribution of EH within a population. Because defects that form prenatally provide a special opportunity to evaluate the female buffering effect, the authors suggest that defect frequencies be separately analyzed according to their time of formation (pre- or postnatal).

- Evidence reported in this review suggests that male great apes exhibit higher defect counts on their canine teeth than do females. This result requires additional testing on larger great ape samples and specific attention to the potential relationship between longer crown formation times and higher defect counts.

The question of interpreting sex differences in EH has been addressed only sporadically in the literature. Within individual studies, sex differences in EH are often attributed to environmental, cultural, or developmental variables that are incompletely documented and often, the synergism among these variables is not considered. The data examined in this review demonstrate the complex nature of factors that interact to affect the populational distribution of EH by sex.

While an understanding of how various factors combine to produce EH frequencies in the males and females of given populations is difficult to achieve, this review highlights the need for gathering particular kinds of data on cultural practices and physiological status that would help elucidate population-wide patterns.

The results of this review also suggest that the study of sex differences in enamel defects forming prenatally has the potential to clarify the ontogeny of male vulnerability in utero. Comparisons of prenatal defects by sex across populations and across primate species would allow exploration of a potential relationship between enhanced female buffering/greater male vulnerability and life

history parameters. A comparative primate perspective on prenatal enamel defects could therefore elucidate evolutionary influences on enhanced female buffering/greater male vulnerability.

Finally, this review strongly suggests that cultural practices of sex-biased parental investment after birth have more powerful effects on sex differences in EH expression than does greater male vulnerability. Evidence of higher EH frequencies in girls might therefore be used as a biological marker of preferential investment in sons. Because parents often do not report differential treatment of offspring by sex, the study of EH could be used to reveal gender disparities in access to basic resources.

The potential of enamel defects to illuminate sex differences in childhood stress will be more fully realized as greater understanding of interacting cultural, environmental, and developmental influences on EH expression is achieved. We hope that issues emphasized in this review will help to focus research efforts towards this goal.

ACKNOWLEDGMENTS

National Science Foundation Grant SBR 9615006 and University of Oregon research awards supported D.G.S.'s EH research in non-human primates. Grants and fellowships from the American Institute of Indian Studies, the National Geographic Society, the Smithsonian Institution (FCP), and the Wenner-Gren Foundation for Anthropological Research have supported J.R.L.'s research projects on the dental paleopathology of South Asia and on enamel defects in the deciduous teeth of humans and great apes. The cordial collaboration of research colleagues, G.L. Badam, and S.R. Walimbe at Deccan College (Pune), J.N. Pal and the University of Allahabad (Allahabad) facilitated studies in India. Museum personnel at several institutions made valuable research collections available for study: Terri McFadden and Maria Rutzmoser (MCZ, Harvard University), Lyman Jellema and Bruce Latimer, Cleveland Museum of Natural History (Cleveland, OH); Richard Thorington and Linda Gordon at the National Museum of Natural History, Smithsonian Institution (Washington, DC); and Malcolm P. Harman

at the Powell-Cotton Museum (Birchington, Kent, UK). We thank Rob Corruccini, Bruce Floyd, Mike Pietrusewsky, Ted Rathbun, and Mark Skinner for assistance on various aspects of the project. Special thanks are due Jacopo Moggi-Cecchi for sharing his data, and to Elizabeth Newell for sharing her graph of great ape LEH incidence. The authors also express their sincere appreciation to Dan Steinberg and Shirley Lukacs for their assistance.

LITERATURE CITED

- Al-Abbasi SA. 1997. Prevalence of EH in Jordanian children. *Am J Phys Anthropol Suppl* 24: 64.
- Aldred MJ, Crawford PJM, Roberts E, Thomas NS. 1992. Identification of a nonsense mutation in the amelogenin gene (AMELX) in a family with X-linked amelogenesis imperfecta (AIH1). *Hum. Genet* 90:413–416.
- Ali M. 1984. Women in famine: The paradox of status in India. In: Currey B, Hugo G, editors. *Famine as a geographical phenomenon*. Dordrecht: D Reidel. p 113–133.
- Alvesalo L. 1997. Sex chromosomes and human growth: a dental approach. *Hum Genet* 101: 15.
- Alvesalo L, De La Chappelle A. 1981. Tooth sizes in two males with deletions of the long arm of the Y chromosome. *Ann Hum Genet* 45:49–54.
- Alvesalo L, Kari M. 1977. Sizes of deciduous teeth in 47,XXX males. *Am J Hum Genet* 29:486–489.
- Alvesalo L, Tammisalo E. 1981. Enamel thickness in 45,X females' permanent teeth. *Am J Hum Genet* 33:464–469.
- Alvesalo L, Tammisalo E, Hakola P. 1985. Enamel thickness in 47, XYY males' permanent teeth. *Ann Hum Biol* 12:421–427.
- Alvesalo L, Tammisalo E, Therman E. 1987. 47,XXX females, sex chromosomes, and tooth crown structure. *Hum Genet* 77:345–348.
- Alvesalo L, Tammisalo E, Townsend G. 1991. Upper central incisor and canine tooth crown size in 47,XXY males. *J Dent Res* 70:1057–1060.
- Anderson JE. 1965. Human skeletons of Tehuacan. *Science* 148:496–497.
- Anderson DL, Thompson GW, Popovich F. 1975. Age of attainment of mineralization states of the permanent dentition. *J Forens Sci* 191–200.
- Angel JL, Kelley JO, Parrington M, Pinter S. 1987. Life stresses of the free Black community as represented by the First African Baptist Church, Philadelphia 1823–1841. *Am J Phys Anthropol* 74: 213–229.
- Armelagos GJ. 1969. Disease in ancient Nubia. *Science* 163:255–259.
- Backman B. 1997. Inherited enamel defects. In: *Dental enamel*, Ciba Foundation Symposium 205. Chichester: John Wiley & Sons. p 175–186.
- Badger GR. 1985. Incidence of enamel hyperplasia in primary canines. *J Dent Child* 52:57–58.
- Bailit HL, Workman PL, Niswander JD, MacLean CJ. 1970. Dental asymmetry as an indicator of genetic and environmental conditions on human populations. *Hum Biol* 42:626–638.
- Berry DR. 1985. Dental paleopathology of Grasshopper Pueblo, Arizona. In: Merbs CF, Miller RJ, Editors. *Health and disease in the prehistoric Southwest*. Tempe, AZ: Arizona State University, Anthropology Research Papers # 34. p 253–274.
- Blakey ML, Leslie TE, Reidy J. 1992. Chronological distribution of dental enamel hypoplasia in African American slaves: a test of the weaning hypothesis. *Am J Phys Anthropol Suppl* 14:50.
- Blakey ML, Leslie TE, Reidy JP. 1994. Frequency and chronological distribution of dental enamel hypoplasia. *Am J Phys Anthropol* 95:371–383.
- Boldsen JL. 1998. Body proportions in a medieval village population: effects of early childhood episodes of ill health. *Ann Hum Biol* 25(4):309–317.
- Brabin L. 1990. Factors affecting the differential susceptibility of males and females to onchocerciasis. *Acta Leiden* 59(1/2):413–426.
- Brook AH, Fearn JM, Smith JM. 1997. Environmental causes of enamel defects. *Dental Enamel*, Ciba Foundation Symposium 205. Chichester: John Wiley & Sons. p 212–215.
- Brooks ST, Brooks RH. 1994. Is longevity an indication of biological superiority, no matter the sex? *Am J Phys Anthropol Suppl* 18:60.
- Brothwell D. 1963. The macroscopic dental pathology of some earlier human populations. In: Brothwell D, Editor. *Dental anthropology*. London: Pergamon Press. p 271–288.
- Brown JD, Smith CE. 1986. Facial surface hypoplasia in primary cuspids. *J Indiana Dent Assoc* 65:13–14.
- Brucker M. 1943. Studies on the incidence and cause of dental defects in children II. hypoplasia. *J Dent Res* 22:115–121.
- Buikstra JE, Cook DC. 1980. Paleopathology: an American account. *Ann Rev Anthropol* 9:433–470.
- Chen E, Yuan ZA, Collier PM, Greene SR, Abrams WR, Gibson CW. 1998. Comparison of upstream regions of X- and Y-chromosomal amelogenin genes. *Gene* 216: 131–137.
- Cohen MN, Bennett S. 1993. Skeletal evidence for sex roles and gender hierarchies in prehistory. In: Miller BD, Editor. *Sex and gender hierarchies*. Cambridge: Cambridge University Press. p 273–296.
- Cohen MN, O'Conner K, Danforth ME, Jacobi KP, Armstrong C. 1997. Archaeology and osteology of the Tipu site. In: Whittington SL, Reed DM, Editors. *Bones of the Maya: Studies of ancient Maya skeletons*. Washington: Smithsonian Institution Press. p 78–86.
- Cook DC, Buikstra JE. 1979. Health and differential survival in prehistoric populations: Prenatal dental defects. *Am J Phys Anthropol* 51(4):649–664.
- Corruccini RS, Handler JS, Mutaw RJ, Lange FW. 1982. Osteology of a slave burial population from Barbados, West Indies. *Am J Phys Anthropol* 59(4):443–460.
- Corruccini RS, Handler JS, Jacobi KP. 1985. Chronological distribution of enamel hypoplasias and weaning in a Caribbean slave population. *Hum Biol* 57(4):699–712.
- Cronk L. 1993. Parental favoritism toward daughters. *Am Sci* 81:272–279.
- Crossner CG, Holm AK. 1975. A descriptive and comparative study of oral health in 8-year-old Swedish children. *Acta Odontol Scand* 33:135–142.
- Cucina A, Iscan MY. 1997. Assessment of enamel hyperplasia in a high status burial site. *Am J Phys Anthropol* 9:213–222.
- Cuhna E. 1995. Testing identification records: Evidence from the Coimbra identified skeletal collection (19th & 20th centuries). In: Saunders SR, Herring A, Editors. *Grave reflections: Portraying the past through cemetery studies*. Toronto: Canadian Scholar's Press. p 179–198.
- Cutress TW, Suckling GW. 1982. The assessment of noncarious defects of enamel. *Int Dent J* 32:117–122.
- Danforth ME, Cook DC, Knick SG III. 1994. The human remains from Carter Ranch Pueblo, Arizona: health in isolation. *Am Antiq* 59(1):88–101.

- Danforth ME, Gilberti JA. 1992. Patterns of inter- and intra-observer error in the microscopic scoring of linear enamel hyperplasia. In: Goodman AH, Capasso LL, Editors. Recent contributions to the study of enamel developmental defects. *J Paleopathol Monogr Publ 2*, p 61-77.
- De Vito MA, Saunders SA. 1990. A discriminant function analysis of deciduous teeth to determine sex. *J Forensic Sci* 35:845-858.
- Demirjian A, Levesque GY. 1980. Sexual differences in dental development and prediction of emergence. *J Dent Res* 59:1110-1122.
- Dettwyler KA. 1992. Nutritional status of adults in rural Mali. *Am J Phys Anthropol* 88(3):309-321.
- Douglas MT, Pietruszewsky M, Ikehara-Quebral RM. 1997. Skeletal biology of Apuruguan: A precontact Chomorro site on Guam. *Am J Phys Anthropol* 104(3): 291-313.
- Driscoll WS, Horowitz HS, Meyers R, Heifetz SB, Kingman A, Zimmerman ER. 1983. Prevalence of dental caries and dental fluorosis in areas with optimal and above-optimal water fluoride concentrations. *J Am Dent Assoc* 107:42-47.
- Driscoll WS, Horowitz HS, Meyers R, Heifetz SB, Kingman A, Zimmerman ER. 1986. Prevalence of dental caries and dental fluorosis in areas with negligible, optimal, and above-optimal fluoride concentrations in drinking water. *J Am Dent Assoc* 113:29-33.
- Dummer PM, Kingdon A, Kingdon R. 1986. Distribution of developmental defects of tooth enamel by tooth type in 11-12-year-old children in South Wales. *Comm Dent Oral Epidemiol* 14:341-344.
- Dummer PM, Kingdon A, Kingdon R. 1990. Prevalence and distribution of tooth type and surface of developmental defects of dental enamel in a group of 15- to 16-year-old children in South Wales. *Comm Dent Health* 7:369-377.
- Duncan WK, Silberman SL, Trubman A. 1988. Labial hypoplasia of primary canines in black Head Start children. *J Dent Child* 55:423-426.
- Duncan WK, Silberman SL, Trubman A, Meydrech EF. 1994. Prevalence and racial distribution of primary canine hypoplasia of the maxillary canine. *Pediatr Dent* 16:365-367.
- Eckhardt RB. 1992. Tooth crown development: Nonhuman primate perspectives on the interpretation of linear enamel hypoplasia frequencies in present and past hominid populations. In: Goodman AH, Capasso LL, Editors. Recent contributions to the study of enamel developmental defects. *J Paleopathol Monogr Publ 2*, p 293-305.
- Eckhardt RB, Protsch von Zieten R. 1993. Enamel hyperplasias as indicators of developmental stress in pongids and hominids. *Hum Evol* 8(2):93-99.
- Eckhardt RB, Protsch A, Protsch von Zieten RR. 1992. Vertical enamel hyperplasias in a Free-living population of Liberian chimpanzees: Variations in expression and frequency of incidence. In: Goodman AH, Capasso LL, Editors. Recent contributions to the study of enamel developmental defects. *J Paleopathol Monogr Publ 2*, p 107-114.
- El-Najjar MY, DeSanti MV, Ozebek L. 1978. Prevalence and possible etiology of dental EH. *Am J Phys Anthropol* 48(2):185-192.
- Elia RJ, Wesolowsky AB. 1991. Archaeological excavations at the Uxbridge Almshouse Burial Ground in Uxbridge, Massachusetts. BAR International Series 564. Oxford: British Archaeological Reports.
- Ensor BE, Irish JD. 1995. The hypoplastic area method for analyzing dental enamel hypoplasia. *Am J Phys Anthropol* 98:507-517.
- Eveleth PB, Tanner JM. 1990. Worldwide variation in human growth. Cambridge: Cambridge University Press.
- Enwonwu CO. 1973. Influence of socio-economic conditions on dental development in Nigerian children. *Archs Oral Biol* 18:95-107.
- Fearne JM, Bryan EM, Elliman AM, Brook AH, Williams DM. 1990. Enamel defects in the primary dentition of children born weighing less than 2000 g. *Br Dent J* 168:433-437.
- Fédération Dentaire Internationale. 1982. An epidemiological index of developmental defects of dental enamel (DDE). *Int Dent J* 32(2):159-167.
- Fédération Dentaire Internationale. 1992. A review of the developmental defects of enamel index (DDE Index). *Int Dent J* 42:411-426.
- Fenton TW. (1998). Dental conditions at Grasshopper Pueblo: Evidence for dietary change and increased stress. Ph.D. dissertation. University of Arizona, Tucson, Arizona.
- Fincham AG, Simmer JP. 1997. Amelogenin proteins of developing enamel. Dental enamel. Ciba Foundation Symposium 205. Chichester: John Wiley & Sons. p 118-134.
- Fincham AG, Bessem CC, Lau EC, Pavlova Z, Schuler C, Slavkin HC, Snead ML. 1991. Human developing enamel proteins exhibit a sex-linked dimorphism. *Calcif Tissue Int* 48:288-290.
- Formicola V. 1986-87. The dentition of the Neolithic sample from western Liguria, Italy. *Ossa* 13:97-108.
- Formicola V. 1987. Neolithic transition and dental changes: the cases of an Italian site. *J Hum Evol* 16:231-239.
- Fornaciari G, Brogi MG, Balducci E. 1985-86. Dental pathology of the skeletal remains of Pontecagnano, Salerno, Italy VII-IV centuries BC. *Ossa* 12:9-32.
- Garn SM, Lewis AB, Kerewsky RS. 1964. Sex differences in tooth size. *J Dent Res* 43:306.
- Garn SM, Lewis AB, Kerewsky RS. 1966. The meaning of bilateral asymmetry in the permanent dentition. *Angle Orthod* 36:55-62.
- Garn SM, Lewis AB, Kerewsky RS. 1967. Sex difference in tooth shape. *J Dent Res* 46:963-972.
- Gibson CW, Collier PM, Yuan Z, Chen E, Adeleke-Stainback P, Lim J, Rosenbloom J. 1997. Regulation of amelogenin gene expression. Dental enamel. Ciba Foundation Symposium 205. Chichester: John Wiley & Sons. p 187-199.
- Gobel FC, Konopka EA. 1973. Sex as a factor in infectious diseases. *Trans NY Acad Sci (Ser 2)* 35(4):325-346.
- Goodman AH. 1976. Enamel hypoplasia as an indicator of stress in three skeletal populations from Illinois (Abst.). *Am J Phys Anthropol* 44:181.
- Goodman AH. 1991. Stress, adaptation and enamel developmental defects. In: Ortner DJ, Aufderheide AC, Editors. Human paleopathology: Current syntheses and future options. Washington, DC: Smithsonian Institution Press. p 280-287.
- Goodman, AH. 1998. The biological consequences of inequality in antiquity. In: Goodman AH, Leatherman TL, Editors. Building a new biocultural synthesis: Political economic perspectives on human biology. Ann Arbor, MI: University of Michigan Press. p 147-169.
- Goodman AH, Armelagos GJ. 1985. Factors affecting the distribution of enamel hypoplasias within the human permanent dentition. *Am J Phys Anthropol* 68:479-493.
- Goodman AH, Rose JC. 1990. Assessment of systemic physiological perturbations from dental enamel hypoplasias and associated histological structures. *Yrbk Phys Anthropol* 33:59-110.

- Goodman AH, Rose JC. 1991. Dental enamel hyperplasias as indicators of nutritional status. In: Kelley MA, Larsen CS, Editors. *Advances in dental anthropology*. New York: Alan R. Liss. p 279–293.
- Goodman AH, Armelagos GJ, Rose JC. 1980. enamel hypoplasias as indicators of stress in three prehistoric populations from Illinois. *Hum Biol* 3:515–528.
- Goodman AH, Allen LH, Hernandez GP, Amador A, Arriola LV, Chavez A, Pelto GH. 1987. Prevalence and age at development of enamel hypoplasias in Mexican children. *Am J Phys Anthropol* 72:7–19.
- Goodman AH, Martinez C, Chavez A. 1991. Nutritional supplementation in the development of linear enamel hyperplasias in children from Tezonteopan, Mexico. *Am J Clin Nutr* 53:773–781.
- Goodman AH, Pelto GH, Allen LH, Chavez A. 1992. Socioeconomic and anthropometric correlates of linear enamel hypoplasia in children from Solis, Mexico. In: Goodman AH, Capasso LL, Editors. *Recent contributions to the study of enamel developmental defects*. *J Paleopathol Monogr Ser 2*. Chieti, Italy: Associazione Anthropologica Abruzzese. P 373–380.
- Grahnén H, Larsson, PG. 1958. Enamel defects in the deciduous dentition of prematurely born children. *Odontologisk Revy* 9:193–204.
- Grauer, A. 1995. *Bodies of evidence: Reconstructing history through skeletal analysis*. New York: Wiley-Liss, Inc.
- Grayson DK. 1990. Donner Party deaths: a demographic assessment. *J Anthropol Res* 46(3):223–242.
- Greenfield LO. 1992. Origin of the human canine: A new solution to an old enigma. *Yrbk Phys Anthropol* 35:153–185.
- Guatelli-Steinberg D. 1998. Prevalence and etiology of linear enamel hyperplasia in non-human primates. Ph.D. dissertation. University of Oregon, Eugene, Oregon.
- Guatelli-Steinberg D, Lukacs JR. 1998. Preferential expression of linear enamel hyperplasia on the sectorial premolars of Rhesus monkeys. *Am J Phys Anthropol* 107:179–186.
- Guatelli-Steinberg D, Skinner M. 1999. Prevalence and etiology of linear enamel hyperplasia (LEH) in monkeys and apes from Asia and Africa. *Folia Primatol* (in press).
- Guatelli-Steinberg D. 1999. Linear enamel hypoplasia in gibbons and other Old World Anthropoids: A graded taxonomic pattern in the expression of linear enamel hypoplasia. *Am J Phys Anthropol Suppl*. 28:141.
- Harris EF, Bailit HL. 1988. A principle components analysis of human odontometrics. *Am J Phys Anthropol* 75:87–99.
- Harris EF, Nweeia MT. 1980. Dental asymmetry as a measure of environmental stress in Ticuna Indians of Colombia. *Am J Phys Anthropol* 53:133–142.
- Harris EF, McKee JH. 1990. Tooth mineralization standards for Black and Whites from the middle southern United States. *J Forensic Sci* 35:859–872.
- Henneberg RJ. 1998. Dental health and affiliations of inhabitants of the ancient Greek colony of Metaponto, Italy (6th–3rd Century BC). Ph.D. dissertation. University of the Witwatersrand, Johannesburg, South Africa.
- Henneberg RJ, Henneberg M. 1989. Dental caries and enamel hypoplasia in a rural population of the ancient Greek colony Metaponto, Italy. *Am J Phys. Anthropol* 78:240.
- Higgins RL, Sirianni JE. 1995. An assessment of health and mortality of nineteenth century Rochester, New York using historic records and the Highland Park skeletal collection. In: A Grauer, editor. *Bodies of evidence: Reconstructing history through skeletal analysis*. New York: Wiley-Liss, Inc. p 121–136.
- Hillson S. 1986. *Teeth*. Cambridge manuals in archaeology series. Cambridge: Cambridge University Press.
- Hillson S. 1992. Dental enamel growth, perikymata, and hypoplasia in ancient tooth crowns. *J Roy Soc Med* 85:460–466.
- Hillson S. 1996. *Dental anthropology*. Cambridge: Cambridge University Press.
- Hillson S. 1998. Crown diameters, tooth crown development, and environmental factors in growth. In: Lukacs JR, Editor. *Human dental development, morphology and pathology: A tribute of Albert A. Dahlberg*. Eugene, OR: University of Oregon Anthropology Paper No 54. p 17–28.
- Hillson S, Bond S. 1997. The relationship of enamel hyperplasia to the pattern of tooth crown growth: a discussion. *Am J Phys Anthropol* 104(1):89–103.
- Hodges DC. 1987. Health and agricultural intensification in the prehistoric Valley of Oaxaca, Mexico. *Am J Phys Anthropol* 73(3):323–332.
- Hodges D (no date). *Untitled manuscript*. (Cited in Whittington, 1992).
- Hoffman MP, Cutress TW, Tomiki S. 1988. Prevalence and developmental defects of enamel in children in the Kingdom of Tonga. *NZ Dent J* 84:7–10.
- Hollimon SE. 1992. Health consequences of sexual division of labor among native Americans: The Chumash of California and the Arikara of the northern plains. In: Classen C, Editor. *Exploring gender through archaeology*. *Monographs in world archaeology*. Madison, WI: Prehistory Press. p 81–88.
- Hoyenga KB, Hoyenga KT. 1982. Gender and energy balance: sex differences in adaptations for feast and famine. *Physiol Behav* 28(3):545–563.
- Hrdy SB. 1987. Sex-biased parental investment among primates and other mammals: A critical evaluation of the Trivers-Willard hypothesis. In: Gelles RJ, Lancaster JB, Editors. *Child abuse and neglect: Biosocial dimensions*. New York: Aldine de Gruyter. p 97–147.
- Huss-Ashmore R, Goodman AH, Armelagos GJ. 1982. Nutritional inference from paleopathology. *Advan Archaeol Meth Theory* 5:395–474.
- Hutchinson DL, Larsen CS. 1988. Determination of stress episode duration from linear enamel hyperplasias: a case study from St. Catherine's Island, Georgia. *Hum Biol* 60:93–110.
- Hutchinson DL, Larsen CS. 1990. Stress and lifeway change: the evidence from enamel hypoplasias. In: Larsen CS, Editor. *The archaeology of Mission Santa Catalina de Guale: 2. Biocultural interpretations of a population in transition*. *Anthropology Papers American Museum of Natural History*, no. 68. p 50–65.
- Iizuka Y, Yasaki T, Ahiko R, Kamata M, Yokoto Y, Matsuzawa A, Hoshino T. 1976. Occurrence of idiopathic mottling of enamel II. Nonfluoride mottlings and hypoplasias in 774 children aged from nine to 14 years. *Bukk Kanagawa Dent Col* 4:51–54.
- Infante PF. 1974. Enamel hyperplasia in Apache Indian children. *Ecol Food Nut* 2:155–156.
- Infante PF, Gillespie GM. 1974. An epidemiological study of linear enamel hyperplasia of deciduous anterior teeth in Guatemalan children. *Arch Oral Biol* 19:1055–1061.
- Infante PF, Gillespie GM. 1976. Dental caries experience in the deciduous dentition of rural Guatemalan children ages 6 months to 7 years. *J Dent Res* 55(6):951–957.
- Isler R, Schoen J, Iscan MY. 1985. Dental pathology of a prehistoric human population in Florida. *Florida Scientist* 48(3):139–146.
- Jelliffe DB, Jelliffe EFP. 1971. Linear hypoplasia of deciduous teeth in malnourished children. *Am J Clin Nutr* 24:893.

- Johnsen D, Krejci C, Hack M, Fanaroff A. 1984. Distribution of enamel defects and the association with respiratory distress in very low birth weight infants. *J Dent Res* 63(1):59-64.
- Kanchanakanamol U, Tuongratanaphan S, Tuongratanaphan S, Lertpoonvilaikul W, Chittaisong C, Pattanaporn K, Navia JM, Daives GN. 1996. Prevalence of developmental enamel defects and dental caries in rural preschool Thai children. *Community Dent Health* 13(4):204-207.
- Katz SH, Armstrong DF. 1994. The biocultural evolution of human longevity: first cousin marriage practices enhance the effect of grandmothering on traits carried on the X chromosome. *Am J Phys Anthropol Suppl* 18:119.
- Katzenberg MA, Herring DA, Saunders SR. 1996. Weaning and infant mortality: Evaluating the skeletal evidence. *Yrbk Phys Anthropol* 39:177-199.
- Keenlyside A. 1998. Skeletal evidence of health and disease in precontact Alaskan Eskimos and Aleuts. *Am J Phys Anthropol* 107:51-70.
- Kelley JO, Angel L. 1987. The life stresses of slavery. *Am J Phys Anthropol* 74:199-211.
- Kerley ER, Bass WM. 1967. Paleopathology: meeting ground for many disciplines. *Science* 157:638-644.
- Kieser JA. 1990. Human adult odontometrics, Cambridge Studies in Biological Anthropology, No. 4. Cambridge: Cambridge University Press.
- Kieser JA, Groeneveld HT. 1998. Fluctuating dental asymmetry and prenatal exposure to tobacco smoke. In: Lukacs JR, Editor. Human dental development, morphology and pathology: A tribute to Albert A. Dahlberg. University of Oregon Anthropology Paper 54. p 287-297.
- King NM. 1989. Developmental defects of enamel in Chinese girls and boys in Hong Kong. *Adv Dent Res* 3:120-125.
- King NM, Wei SHY. 1992. A review of the prevalence of developmental enamel defects in permanent teeth. In: Goodman AH, Capasso LL, Editor. Recent contributions to the study of enamel developmental defects. *J Paleopathol Monogr Ser 2*. Chieti, Italy: Associazione Anthropologica Abruzzese. p 341-357.
- Klatsky M, Fischer RL. 1953. The human masticatory apparatus: an introduction to dental anthropology. Brooklyn, NY: Dental Items of Interest Publishing.
- Konigsberg LW, Grant WE. 1994. Females better buffered? Past and ethnographic present. *Am J Phys Anthropol Suppl* 18:124.
- Kreshover SJ. 1942. The histopathology of the incisor teeth of mice with experimentally induced tuberculosis. *J Dent Res* 21:27.
- Kreshover SJ, Clough OW, Hancock JA. 1954. Vaccinia infection in pregnant rabbits and its effect on maternal and fetal dental tissues. *J Am Dent Assoc* 49:549-562.
- Kühl I. 1992. Six cases of enamel hypoplasia in prehistoric cremations of Schleswig-Holstein, North Germany. In: Goodman AH, Capasso LL, Editors. Recent contributions to the study of enamel developmental defects. *J Paleopathol Monogr Publ No 2*. Teramo, Italy: Edigrafital. p 239-254.
- Kuykendall KL. 1996. Dental development in chimpanzees (*Pan troglodytes*): The timing of tooth calcification stages. *Am J Phys Anthropol* 99:135-157.
- Lai PY, Seow WK, Tudehope DI, Rogers Y. 1997. Enamel hyperplasia and dental caries in very low birth weight children: a case-controlled, longitudinal study. *Pediatr Dentistry* 19(1):42-49.
- Lanphear KM. 1990. Frequency and distribution of enamel hyperplasias in a historic skeletal series. *Am J Phys Anthropol* 81:35-43.
- Larsen CS. 1987. Bioarchaeological interpretations of subsistence economy and behavior from human skeletal remains. *Adv Archaeol Meth Theory* 10:339-445.
- Larsen CS. 1995. Biological changes in human populations with agriculture. *Annu Rev Anthropol* 24:185-213.
- Larsen CS. 1997. Bioarchaeology: Interpreting behavior from the human skeleton. Cambridge: Cambridge University Press.
- Larsen CS, Hutchinson DL. 1992. Dental evidence for physiological disruption: Biocultural interpretations from eastern Spanish borderlands. In: Goodman AH, Capasso LL, Editors. Recent contributions to the study of enamel developmental defects. *J Paleopathol Monogr Ser 2*. Chieti, Italy: Associazione Anthropologica Abruzzese. p 151-169.
- Larsen CS, Spencer CJ, Sering LE, Schoeninger MJ, Russell KF, Hutchinson DL, Williamson, MA. 1995. Cross homestead: life and death on the midwestern frontier. In: A Grauer, editor. Bodies of evidence: Reconstructing history through skeletal analysis. New York: Wiley-Liss, Inc. p 139-159.
- Lau EC, Mohandas TK, Shapiro LJ, Slavkin HC, Snead ML. 1989. Human and mouse amelogenin gene loci are on the sex chromosomes. *Genomics* 4:162-168.
- Lazenby RA, Pfeiffer SK. 1994. Sex as a covariate in the analysis of bone remodeling. *Am J Phys Anthropol Suppl* 18:127.
- Lench NJ, Brook AH, Winter GB. 1994. SSCP detection of a nonsense mutation in exon 5 of the amelogenin gene (AMGX) causing X-linked amelogenesis imperfecta (AIH1) *Hum Genet* 90:313-316.
- Leonard WR. 1991. Age and sex differences in the impact of seasonal energy stress among Andean agriculturalists. *Hum Ecol* 19(3):351-368.
- Li YH. 1993. Influence on dental caries of malnutrition, enamel hyperplasia, and colonization of mutans streptococci in rural Chinese children 3 to 5 years old. D.P.H. dissertation. University of Alabama School of Public Health, Birmingham, Alabama.
- Li Y, Navia JM, Bian JY. 1995. Prevalence and distribution of developmental defects in the primary dentition of Chinese children 3-5 years old. *Community Dent Oral Epidemiol* 23:72-79.
- Little BJ, Lanphear KM, Owsley DW. 1992. Mortuary display and status in a Nineteenth Century Anglo-American cemetery in Manassas, Virginia. *Amer Antiq* 57:397-418.
- Lukacs JR. 1991. Localized enamel hyperplasia of human deciduous canine teeth: prevalence and pattern of expression in rural Pakistan. *Hum Biol* 63(4):513-522.
- Lukacs JR. 1992. Dental paleopathology and agricultural intensification in South Asia: new evidence from Bronze Age Harappa. *Am J Phys Anthropol* 87(1):133-150.
- Lukacs JR. 1999a. Interproximal contact hypoplasia (IPCH) in primary teeth: A new enamel defect with anthropological and clinical relevance. *Am J Hum Biol* 11 (in press).
- Lukacs JR. 1996. Enamel hypoplasia in deciduous teeth of great apes: Do differences in defect prevalence imply differential levels of physiological stress? *Am J Phys Anthropol* 110:351-363.
- Lukacs JR, Guatelli-Steinberg D. 1994. Daughter neglect in India: LEH prevalence and the question of female biological superiority. *Am J Phys Anthropol Suppl* 18:132.

- Lukacs JR, Joshi MR. 1992. Enamel hypoplasia prevalence in three ethnic groups of northwest India: a test of daughter neglect and a framework for the past. In: Goodman AH, Capasso LL, Editors. Recent contributions to the study of enamel developmental defects. *J Paleopathol Monogr Publ No 2*. Teramo, Italy: Edigrafital. p 359–372.
- Lukacs JR, Pal JN. 1993. Mesolithic subsistence in north India: inferences from dental pathology and odontology. *Curr Anthropol* 34(5):745–765.
- Lukacs JR, Schultz M, Hemphill BE. 1989. Dental pathology and dietary patterns in Iron Age northern Pakistan. In: Frifelt K, Sorensen P, Editors. *South Asian archaeology 1985*. London: Curzon Press. p 475–496.
- Lukacs JR, Walimbe SR. 1998. Physiological stress in prehistoric India: New data on localized enamel hyperplasia linked to climate and subsistence change. *J Archaeol Sci* 25:571–585.
- Macho GA, Wood BA. 1995. The role of time and timing in hominid dental evolution. *Evol Anthropol* 4(1): 17–31.
- Mack ME, Coppa A. 1992. Frequency and chronological distribution of enamel hyperplasias from the Ra's alHamra5 (RH5) skeletal collection (Oman). In: Goodman AH, Capasso LL, Editors. Recent contributions to the study of enamel developmental defects. *J Paleopathol Monogr Ser 2*. Chieti, Italy: Associazione Anthropologica Abruzzese. p 131–141.
- Malville NJ. 1997. Enamel hyperplasia in ancestral Puebloan populations from southwestern Colorado: I. permanent dentition. *Am J Phys Anthropol* 102(3): 351–367.
- Manning JT, Chamberlain AT. 1993. Fluctuating asymmetry, sexual selection and canine teeth in primates. *Proc R Soc Lond B Biol Sci* 251:83–87.
- Manning JT, Chamberlain AT. 1994. Fluctuating asymmetry in gorilla canines: a sensitive indicator of environmental stress. *Proc R Soc London B Biol Sci* 225:189–193.
- Manzi G, Santandrea E, Passarello P. 1997. Dental size and shape in the Roman Imperial Age: Two examples from the area of Rome. *Am J Phys Anthropol* 102:469–479.
- Manzi G, Salvadei L, Vienna A, Passarello P. 1999. Discontinuity of life conditions at the transition from the Roman imperial age to the early middle ages: Example from central Italy evaluated by pathological dentoalveolar lesions. *Am J Hum Biol* 11(3):327–341.
- Maunder J, Goodman A, Froment A. 1992. The ecology of dental enamel hyperplasias among seven Cameroonian groups. In: Lukacs JR, Editor. *Culture, ecology and dental anthropology*. Delhi: KamlaRaj Enterprises. p 109–116.
- May RL, Goodman AH, Meindl RS. 1993. Response of bone and enamel formation to nutritional supplementation and morbidity among malnourished Guatemalan children. *Am J Phys Anthropol* 92:37–51.
- Mayhall JT, Alvesalo L, Townsend G. 1998. Dental morphology of 47, XYY males: molar cusp area, volume, shape, and linear dimensions. In: Lukacs JR, Editor. *Human dental development, morphology and pathology: A tribute to Albert A. Dahlberg*. University of Oregon Anthropology Papers, No. 54. p 29–38.
- Mays S. 1995. The relationship between Harris lines and other aspects of skeletal development in adults and juveniles. *J Archaeol Sci* 22:511–520.
- Mays S. 1998. *The archaeology of human bones*. London: Routledge.
- McKee JK, Lutz R. 1990. Correlates of enamel hyperplasia with human dental reduction. *Am J Hum Biol* 2:459–465.
- Mellanby M. 1929. Diet and teeth: an experimental study, Part I. Dental structure in dogs. Medical Research Council, Special Report Series No. 140. London: HMSO.
- Medical Research Council, Special Report Ser. No 140. London: His Majesty's Stationery Office.
- Miller BD. 1981. The endangered sex: Neglect of female children in rural North India. Ithaca, NY: Cornell University Press.
- Miller J, Forrester RM. 1959. Neonatal enamel hyperplasia: associated with haemolytic disease and with prematurity. *Br Dental J* 106(8):93–104.
- Moggi-Cecchi J, Crovella S. 1991. Occurrence of enamel hyperplasia in the dentitions of simian primates. *Folia Primatol* 57:106–110.
- Moggi-Cecchi J, Crovella S. 1992. Dental developmental defects in the dentitions of *Dryopithecus* from Spain. *Anthropol Contemp* 15:47–50.
- Moggi-Cecchi J, Crovella S, Bari A, Gonella P. 1993. Enamel hyperplasia in a 19th Century population from northern Italy. *Anthropol Anzeiger* 51:123–129.
- Moggi-Cecchi J, Pacciani E, Pinto-Cisternas J. 1994. Enamel hyperplasia and age at weaning in 19th Century Florence, Italy. *Am J Phys Anthropol* 93(3):299–306.
- Moorrees CFA, Fanning EA, Hunt EE. 1963a. Age variation of formation stages in ten permanent teeth. *J Dent Res* 42:1450–1502.
- Moorrees CFA, Fanning FA, Hunt EE. 1963b. Formation and resorption of three deciduous teeth in children. *Am J Phys Anthropol* 21:205–269.
- Moss M. 1978. Analysis of developmental processes possibly related to human dental sexual dimorphism. In: Butler BM, Joysey KM, Editors. *Development, evolution, and function of teeth*. New York: Academic Press. p 135–148.
- Moss ML, Moss-Salentijn L. 1976. Analysis of developmental processes possibly related to human dental sexual dimorphism in permanent and deciduous canines. *Am J Phys Anthropol* 46:407–414.
- Murray EA, Perzigian AJ. 1995. A glimpse of early nineteenth century Cincinnati as viewed from Potter's Field: an exercise in problem solving. In: Grauer A, editor. *Bodies of evidence: Reconstructing history through skeletal analysis*. New York: Wiley-Liss, Inc. p 173–184.
- Nakahori Y, Takenaka O, Nakagome Y. 1991. A human XY homologous region encodes "amelogenin" *Genomics* 9:264–269.
- Nass GG. 1982. Dental asymmetry as an indicator of developmental stress in a free-ranging troop of *Macaca fuscata*. In: Kurtén B, Editor. *Teeth: Form, function, and evolution*. New York: Columbia University Press. p 207–227.
- Nation WA, Marsson L, Peterson JE. 1987. Developmental enamel defects of the primary dentition in a group of California children. *J Dent Child* 4:330–334.
- Needleman HL, Allred E, Bellinger D, Leviton A, Rabinowitz M, Iverson K. 1992. Antecedents and correlates of hypoplastic enamel defects of primary incisors. *Pediatr Dent* 14:158–166.
- Needleman HL, Leviton A, Allred E. 1991. Macroscopic enamel defects of primary anterior teeth types, prevalence, and distribution. *Pediatr Dentistry* 13(4):208–216.
- Neiburger EJ. 1990. Enamel hyperplasia — A poor indicator of nutritional stress. *Am J Phys Anthropol* 82:231–233.
- Newell EA. 1998. Dental enamel hyperplasia in non-human primates: a systematic assessment of its occurrence and distribution. Ph.D. dissertation. Temple University, Philadelphia.

- Nikiforouk G, Fraser D. 1981. The etiology of enamel hyperplasia: A unifying concept. *J Pediatr* 98:888–893.
- Niswander JD, Chung CS. 1965. The effects of inbreeding on tooth size in Japanese children. *Am J Hum Genet* 17:390–398.
- Nolla CM. 1960. The development of permanent teeth. *J Dent Child* 27:254–266.
- Osuji OO. (1990) Utilization of dental services by children. *Trop Dent J* 13:97–99.
- Perzigian AJ. 1977. Fluctuating dental asymmetry: Variation among skeletal populations. *Am J Phys Anthropol* 47:81–88.
- Pietruszewsky M, Douglas MT, Ikehara-Quebral RM. 1997. An assessment of health and disease in the prehistoric inhabitants of the Mariana Islands. *Am J Phys Anthropol* 104(3):315–342.
- Pimlott JFL, Howley TP, Nikiforuk G, Fitzhardinge PM. 1985. Enamel defects in prematurely born low birth weight infants. *Pediatr Dentistry* 7(3):218–223.
- Pindborg JJ. 1982. Aetiology of developmental enamel defects not related to fluorosis. *Int Dent J* 32:123–134.
- Plavcan JM. 1990. Sexual dimorphism in the dentition of extant anthropoid primates. Ph.D. dissertation. Ann Arbor: University Microfilms.
- Plavcan JM. 1998. Correlated response, competition, and female canine size in primates. *Am J Phys Anthropol* 107(4):401–416.
- Powell ML. 1988. Status and health in Prehistory: A case study of the Moundville Chiefdom. Washington, DC: Smithsonian Institution Press.
- Radlanski RJ, Seidl W, Steding G. 1995. Prism arrangement in human dental enamel. In: Moggi-Cecchi J, Editor. *Aspects of dental biology: Palaeontology, anthropology, and evolution*. Florence: International Institute for the Study of Man. p 33–49.
- Rathbun TA. 1987. Health and disease at a South Carolina plantation 1840–1870. *Am J Phys Anthropol* 74:239–253.
- Rathbun TA. 1994. Pathology and gender in 19th Century US samples: biology or culture? *Am J Phys Anthropol* 18:165.
- Rathbun TA, Scurry JD. 1991. Status and health in colonial South Carolina: Bellview Plantation, 1738–1756. In: Powell ML, Bridges PS, Mires AMW, Editors. *What mean these bones?: Studies in southeastern bioarchaeology*. Tuscaloosa, AL: University of Alabama Press. p 148–164.
- Riopelle AJ. 1990. Postnatal protein deprivation in rhesus monkeys. *Am J Phys Anthropol* 83:239–252.
- Rivers JPW. 1988. The nutritional biology of famine. In: Harrison GA, Editor. *Famine*. Oxford: Oxford University Press. p 57–106.
- Roberts C, Manchester K. 1995. The archaeology of disease. 2nd ed. Ithaca, NY: Cornell University Press.
- Roberts CA, Margerison B. 1994. Male and female susceptibility to infectious disease: A study of British skeletal populations and historical records. *Am J Phys Anthropol Suppl* 18:170.
- Rose JC. 1985. Gone to a better land: A biohistory of a rural Black cemetery in the Post-Reconstruction South. Fayetteville, AR: Arkansas Archeological Survey, Research Series, 0882–5491; no. 25.
- Rose JC, Boyde LF, Condon KW. 1981. Enamel microdefects and subadult infections [Abstr]. *Am J Phys Anthropol* 54:270.
- Rose JC, Condon KW, Goodman AH. 1985. Diet and dentition: developmental disturbances. In: Gilbert RI Jr, Mielke JH, Editors. *The analysis of prehistoric diets*. Orlando: Academic Press. p 281–305.
- Salido EC, Yen PH, Koprivnikar K, Yu LC, Shapiro LJ. 1992. The human enamel protein gene amelogenin is expressed from both the X and Y chromosomes. *Am J Hum Genet* 50:303–316.
- Samuelson G, Grahnén H, Lindstrom G. 1971. An epidemiological study of child health and nutrition in a northern Swedish county. *Odontol Rev* 22:189–220.
- Santos RV, Coimbra CE. 1999. Hardships of contact: enamel hyperplasias in Tupi-Mondé Amerindians from the Brazilian Amazonia. *Am J Phys Anthropol* 109(1): 111–127.
- Sarnat GB, Schour I. 1941. Enamel hyperplasia (chronologic enamel aplasia) in relation to systemic disease: A chronologic, morphologic, and etiologic classification. *J Am Dent Assoc* 28/29:1989–2000/67–74.
- Sasaki S, Shimokawa H. 1995. The amelogenin gene. *Int J Dev Biol* 39:127–133.
- Saul FP. 1972. The human skeletal remains from Altar de Sacrificios: An osteobiographic analysis. *Papers Peabody Mus Archaeol Ethnol* 63(2). Cambridge, MA: Harvard University Press.
- Saul F. 1973. Disease in the Maya area: The Pre-Columbian evidence. In: Culbert TP, editor. *The classic Maya collapse*. Albuquerque: University of New Mexico Press. p 301–324.
- Saul F. 1975. Human remains from Lubaantun. In: Hammond N, editor. *Lubaantun*. Peabody Museum of Archaeology and Ethnology, No. 2. Cambridge: Harvard University Press. p 389–410.
- Saul F. 1982. Appendix II—The human skeletal remains from Tancah, Mexico. In: Miller AG, editor. *On the edge of the sea*. Washington: Dumbarton Oaks Trustees for Harvard University. p 115–128.
- Saul JM, Saul FP. 1997. The Preclassic skeletons from Cuello. In: Whittington SL, Reed DM, Editors. *Bones of the Maya: Studies of ancient Maya skeletons*. Washington, DC: Smithsonian Institution Press. p 28–50.
- Saunders SA. 1994. Tooth size as an indicator of sex differences in mortality. *Am J Phys Anthropol Suppl* 18:177.
- Saunders SR, Herring A. 1995. *Grave reflections: Portraying the past through cemetery studies*. Toronto: Canadian Scholar's Press.
- Saunders SR, Keenleyside A. 1999. Enamel hypoplasia in a Canadian historic sample. *Am J Hum Biol* 11:513–524.
- Scholl TO, Johnston FE, Cravioto J, DeLicardie ER, Lurie DS. 1979. The relationship of growth failure (chronic undernutrition) to the prevalence of clinically severe protein-energy malnutrition and to growth retardation in protein-energy malnutrition. *Am J Clin Nutr* 32:872–878.
- Schultz M, Carli-Thiele P, Schmidt-Schultz TH, Kierdorf U, Kierdorf H, Teegen WR, Kreutz K. 1998. Enamel hyperplasias in archaeological skeletal remains. In: Alt KW, Rosing FW, Teschler-Nicola M, Editors. *Dental anthropology: Fundamentals, limits, and prospects*. New York: Springer-Verlag. p 293–311.
- Sciulli PW. 1978. Developmental abnormalities of the permanent dentition in prehistoric Ohio Valley Amerindians. *Am J Phys Anthropol* 48:93–198.
- Scott GR, Halfman CM, Pedersen PO. 1991. Dental conditions of medieval Norsemen in the North Atlantic. *Acta Archaeol* 62:183–207.
- Seow WK. 1992. Dental enamel defects in low birth weight children. In: Goodman AH, Capasso LL, Editors. *Recent contributions to the study of enamel developmental defects*. *J Paleopathol Monogr Ser* 2. Chieti, Italy: Associazione Anthropologica Abruzzese. p 321–330.
- Seow WK. 1997a. Clinical diagnosis of enamel defects: pitfalls and practical guidelines. *Int Dent J* 47:173–182.

- Seow WK. 1997b. Effects of preterm birth on oral growth and development. *Austral Dent J* 42(2):85–91.
- Seow K, Humphrys C, Tudehope DI. 1987. Increased prevalence of developmental dental defects in low-birth-weight children: A controlled study. *Pediatr Dent* 9:221–225.
- Seow WK, Perham S. 1990. Enamel hyperplasia in prematurely-born children: a scanning electron microscopic study. *J Pedodont* 14:235–239.
- Silberman SL, Duncan WK, Trubman A, Meydrech EF. 1989. Primary canine hypoplasia in Head Start children. *J Publ Health Dentistry* 49:15–18.
- Silberman SL, Trubman A, Duncan WK, Meydrech EF. 1991. Prevalence of primary canine hypoplasia of the mandibular teeth. *Pediatr Dentistry* 13(6):356–360.
- Sirianni JE, Swindler D. 1985. Growth and development of the pigtailed macaque. Boca Raton, FL: CRC Press.
- Skinner MF. 1986a. Enamel hyperplasia in sympatric chimpanzee and gorilla. *Hum Evol* 1(4):289–312.
- Skinner MF. 1986b. An enigmatic hypoplastic defect of the deciduous canine. *Am J Phys Anthropol* 69(1):59–69.
- Skinner MF. 1996. Developmental stress in immature hominines from late Pleistocene Eurasia: Evidence from enamel hypoplasia. *J Archaeol Sci* 23:833–852.
- Skinner MF, Goodman AH. 1992. Anthropological uses of developmental defects of enamel. In: Saunders SR, Katzenberg MA, Editor. *Skeletal biology of past peoples: Research methods*. New York: Wiley-Liss. p 153–174.
- Skinner MF, Guatelli-Steinberg D. 1997. Distribution of linear enamel hyperplasia in sympatric monkeys and apes. *Am J Phys Anthropol Suppl* 24:213.
- Skinner MF, Hung JTW. 1989. Social and biological correlates of localized enamel hypoplasia of the human deciduous canine tooth. *Am J Phys Anthropol* 79(2):159–175.
- Skinner MF, Hadaway W, Dickie J. 1994. Effects of ethnicity and birth month on localized enamel localized enamel hyperplasia of the primary canine. *J Dentistry Child* 61(3):109–113.
- Skinner MF, Dupras TL, Moya-Sola S. 1995. Periodicity of enamel hyperplasia among Miocene *Dryopithecus* from Spain. *J Paleopathol* 7(3):197–222.
- Steckel RH. 1986a. A dreadful childhood: excess mortality among American slaves. *Soc Sci Hist* 10:427–465.
- Steckel RH. 1986b. A peculiar population: the nutrition, health and mortality of American slaves from childhood to maturity. *J Econ Hist* 46:721–741.
- Steckel RH. 1987. Growth depression and recovery: the remarkable case of American slaves. *Ann Hum Biol* 14(2):111–132.
- Stini WA. 1969. Nutritional stress and growth: Sex differences in adaptive response. *Am J Phys Anthropol* 31(3):417–426.
- Stini WA. 1971. Evolutionary implications of changing nutritional patterns in human populations. *Am Anthropol* 73:1019–1030.
- Stini WA. 1972. Reduced sexual dimorphism in upper arm muscle circumference associated with protein-deficient diet in a South American population. *Am J Phys Anthropol* 36:341–352.
- Stini WA. 1975. Adaptive strategies of human populations under nutritional stress. In: Watts ES, Johnston FE, Lasker GW, Editors. *Biosocial interrelations in population adaptation*. The Hague: Mouton. p 19–41.
- Stini WA. 1978. Human growth as an adaptive strategy. *Colloquia in anthropology* (Taos, N.M.) 2:47–62.
- Stini WA. 1985. Growth rates and sexual dimorphism in evolutionary perspective. In: Gilbert RI, Mielke JH, Editors. *The analysis of prehistoric diets*. Orlando, FL: Academic Press. p 191–226.
- Stini WA. 1994. Differences in female and male aging patterns in modern populations. *Am J Phys Anthropol Suppl* 18:188.
- Stinson S. 1985. Sex differences in environmental sensitivity during growth and development. *Yrbk Phys Anthropol* 28:123–147.
- Stinson S. 1994. Are females more buffered than males during postnatal growth? *Am J Phys Anthropol Suppl* 18:188.
- Stodder AM. 1997. Subadult stress, morbidity, and longevity in Latte Period populations on Guam, Marianas Islands. *Am J Phys Anthropol* 104(3):363–380.
- Stottlemire MM. 1998. Presence of enamel hyperplasia in wildshot chimpanzees (genus *Pan*) And gorillas (genus *Gorilla*). *Am J Phys Anthropol Suppl* 26:212.
- Suckling G, Elliot DC, Thurley DC. 1986. The macroscopic appearance and associated histological changes in the enamel organ of hypoplastic lesions of sheep incisor teeth resulting from induced parasitism. *Arch Oral Biol* 31:427–439.
- Suga S. 1989. Enamel hypomineralization viewed from the pattern of progressive mineralization of human and monkey developing enamel. *Adv Dent Res* 3:188–198.
- Swärdstedt T. 1966. Odontological aspect of a medieval population from a province in Jamtland, Mid-Sweden. Stockholm: Tiden Barnangen, AB.
- Sweeney EA, Cabrera J, Urrrtia J, Mata L. 1969. Factors associated with linear hypoplasia of human deciduous incisors. *J Dent Res* 48(2):1275–1279.
- Sweeney EA, Saffir AJ, de Leon R. 1971. Linear hypoplasia of deciduous incisor teeth in malnourished children. *Am J Clin Nutr* 24:29–31.
- Ten Cate AR. 1994. *Oral histology: Development, structure, and function*, 4th ed. St. Louis: CV Mosby.
- Ten Cate AR. 1998. *Oral histology: Development, structure, and function*, 5th ed. St. Louis: CV Mosby.
- Thompson GW, Anderson DL, Popovich F. 1975. Sexual dimorphism in dentition mineralization. *Growth* 39:289–301.
- Townsend GC. 1981. Fluctuating asymmetry in the deciduous dentition of Australian aboriginals. *J Dent Res* 60:1849–1857.
- Townsend GC, Brown T. 1980. Dental asymmetry in Australian aboriginals. *Hum Biol* 52:661–673.
- Townsend GC, Farmer A. 1998. Dental asymmetry in the deciduous dentition of South Australian aboriginals. In: Lukacs JR, Editor. *Human dental development, morphology, and pathology: A tribute to Albert A. Dahlberg*. University of Oregon Anthropology Papers 54. p 245–257.
- Townsend GC, Garcia-Godoy F. 1984. Fluctuating asymmetry in the deciduous dentition of Dominican mulatto children. *Arch Oral Biol* 29:483–486.
- van Gerven DP, Sheridan SG. 1994. Sex differences in stress response in human remains from Sudanese Nubia. *Am J Phys Anthropol Suppl* 18:201.
- van Gerven DP, Beck R, Hummert JR. 1990. Patterns of enamel hyperplasia in two medieval populations from Nubia's Batn El Hajar. *Am J Phys Anthropol* 82:413–420.
- van Valen L. 1962. A study of fluctuating asymmetry. *Evolution*. 16:125–142.
- Vitzthum VJ, Wikander R. 1988. Incidence and correlates of enamel hyperplasia in nonhuman primates. *Am J Phys Anthropol* 75:284.
- Waddington CH. 1957. *The strategy of the gene*. New York: Macmillan.
- Walker A. 1984. Mechanisms of honing in the male baboon canine. *Am J Phys Anthropol* 65:47–60.
- Walker PL. (no date) Enamel hyperplasia during 5000 years of southern California history. Health and disease in the prehistoric Southwest II. Maxwell Museum of Anthropology Papers.

- Webb S. 1995. Paleopathology of Australian Aboriginals: Health and disease across a hunter-gatherer continent. Cambridge: Cambridge University Press.
- White C. 1988. Diet and health in the ancient Maya at Lamanai, Belize. In: Kennedy BV, LeMoine GM, Editors. Diet and subsistence: Current archaeological perspectives. Calgary: University of Calgary Archaeology Association. p 288–296.
- White CD. 1997. Ancient diet at Lamanai and Pacbitun: Implications for the ecological model of collapse. In: Whittington SL, Reed DM, Editors. Bones of the Maya: Studies of ancient Maya skeletons. Washington, DC: Smithsonian Institution Press. p 171–180.
- White CD, Wright L, Pendergast DM. 1994. Biological disruption in the early colonial period at Lamanai. In: CS Larsen, Milner GR, editors. In the wake of contact: Biological responses to conquest. New York: Wiley-Liss, Inc. p 135–145.
- Whittington SL. 1992. Enamel hyperplasia in the low status Maya population of prehispanic Copan, Honduras. In: Goodman AH, Capasso LL, Editors. Recent contributions to the study of enamel developmental defects. J Paleopathol Monogr Ser 2. Chieti, Italy: Associazione Anthropologica Abruzzese. p 185–205.
- Winchell F, Rose JC, Randall WM. 1995. Health and hard times: a case study from the middle to late nineteenth century in eastern Texas. In: Grauer A, editor. Bodies of evidence: Reconstructing history through skeletal analysis. New York: Wiley-Liss, Inc. p 161–172.
- Wood JR, Milner GR, Harpending HC, Weiss KM. 1992. The osteological paradox: problems inferring health from skeletal samples. Curr Anthropol 33(4):343–358.
- Wood L. 1996. Frequency and chronological distribution of linear enamel hyperplasia in a North American colonial skeletal sample. Am J Phys Anthropol 100: 247–259.
- Wright LE. 1990. Stresses of conquest: a study of Wilson's bands and enamel hyperplasias in the Maya of Lamanai, Belize. Am J Hum Biol 2:25–35.
- Wright LE, White CD. 1996. Human biology in the classic Maya collapse: evidence from paleopathology and paleodiet. J World Prehist 10(2):147–188.
- Zhang Y. 1987. Enamel hypoplasia of *Gigantopithecus blacki*. Acta Anthropol Sinica 6:175–179.
- Zhou L, Corruccini RS. 1998. Enamel hypoplasias related to famine stress in living Chinese. Am J Hum Biol 10:723–733.