

Center for Studies in Demography and Ecology



Assessing Biological Mortality Bias in Deciduous Tooth Emergence

by

Darryl J. Holman
University of Washington

Kathleen A. O'Connor
University of Washington

Robert E. Jones
University of Wisconsin – Madison

ASSESSING BIOLOGICAL MORTALITY BIAS IN DECIDUOUS TOOTH EMERGENCE

Darryl J. Holman^{1,2,3}, Kathleen A. O'Connor^{1,2}, and Robert E. Jones⁴

¹Department of Anthropology,

²Center for Studies in Demography and Ecology

³Center for Statistics and the Social Sciences

University of Washington

Seattle, WA 98195

⁴Center for Demography and Ecology

University of Wisconsin—Madison

Madison, WI 53706

ABSTRACT

Traits found in a skeletal sample are frequently used to infer qualities of the living population from which the skeletons were drawn. However, traits observed in a mortality sample may not accurately represent the same traits in the living sample. The purpose of this research was to assess biological mortality bias in deciduous tooth emergence, a trait that is used for estimating chronological age in both living and skeletal samples.

Data on clinical tooth emergence were collected from longitudinal studies of Javanese, Guatemalan, and Bangladeshi children. Each sample was divided into a *living sample* for those who survived through the study and a *mortality sample* for those who died during the study. Parametric hazards analysis was used to test for differences in the timing of tooth emergence between the living and mortality samples. No mortality bias was found in the Bangladeshi and Javanese samples, although the Bangladeshi mortality sample exhibited a trend toward delayed emergence. The Guatemalan mortality sample exhibited significantly advanced emergence of the posterior dentition. We cannot rule out the possibility that this unexpected finding is an artifact of nutritional interventions used in the original study. A single analysis of all three samples found no evidence of biological mortality bias. We conclude that the timing of deciduous tooth emergence in a mortality sample (e.g. a skeletal series) can be considered representative of the timing of emergence in the living population from which the mortality sample was drawn.

INTRODUCTION

Anthropologists use skeletal samples to investigate health, disease, and the demography of past populations. One of the assumptions implicit in most such investigations is that characteristics of the skeletal sample are representative of the living population from which they were drawn. The validity of using a skeletal sample to represent the living population, however, has been long been questioned (Johnston, 1968; Armelagos et al., 1972; Merchant and Ubelaker, 1977; Wood et al. 1992). One key issue is whether factors that contributed to the deaths of the individual also affected the trait of interest (Wood et al., 1992; Saunders and Hoppa, 1993). If so, then the trait, as assessed in the mortality sample, will be biased relative to the trait in the living population, and thus not representative of the health or demographic characteristics of the living population. This has been termed biological mortality bias, defined as a physiological and morphological difference between individuals of a given age who die and those who survive (Saunders and Hoppa, 1993).¹

Biological mortality bias arises through the more general and interrelated phenomena of selective mortality and frailty differences among individuals (Wood et al. 1992). Individuals of a given age in a cemetery sample have been selected through death. As a result, they are selected for characteristics that increased risk of mortality, and will differ from the living population at that same age in these same characteristics. The mortality sample may have had lower food availability or a higher disease load, or they may have differed from the living sample in physiological characteristics like susceptibility to disease or the ability to fight infection. Biological mortality bias is an outcome of selective mortality that arises when there is a correlation between risk factors for mortality and the expression of the skeletal trait (e.g. long bone growth or tooth emergence). For example, if the rate of tooth eruption is lower for individuals who are most susceptible to diarrheal disease, and diarrheal disease leads to significant mortality, then tooth eruption will be delayed in the skeletal sample relative to the living population.

Biological mortality bias can be subdivided into two components. First, there can be mortality bias in the target trait of interest. For example, long-bone growth is frequently used to assess the health of a population based on a series of sub-adult skeletons (Buikstra and Cook, 1980; Johnston and Zimmer, 1989; Saunders, 1992). A mortality bias in age-specific long bone length would exist if, on average, children who die prior to completing their long-bone growth are developmentally behind same-aged children who survive to maturity. Hence, inferences about the underlying population would unwittingly reflect this bias toward slower growth.

A second component to biological mortality bias can arise in the traits used to age a skeletal sample. Many of the characters used to assess the health of skeletal samples are expressed relative to age. For example, growth traits must be standardized relative to chronological age in order to compare the trait in a target sample to the same trait in other populations. Dental development shows a high correlation to chronological age, and is therefore one of most accurate macro-indicators used in skeletal age estimation (Holtz, 1959; Lewis and Garn, 1960; Krogman, 1968, b; Demirjian, 1986; Saunders et al., 1993). Health is then determined by long bone growth and other osseous markers of development to see how well an individual's growth tracks with an estimate of dental age. We cannot be sure, however, that dental development is immune to biases from selective mortality. If dental development is adversely affected in the mortality sample, then our inference of health is based on biased estimates of chronological age.

¹ In addition to biological mortality, Saunders and Hoppa (1993) define two other types of mortality bias. Cultural mortality bias is variation in mortuary practice that leads to differential preservation of individuals or classes of individuals. Environmental mortality bias reflects differential effects of skeletal preservation resulting from environmental factors.

In this paper, we focus on this second aspect of biological mortality bias. We examine whether a mortality bias exists in deciduous tooth emergence. Emergence of the deciduous teeth is one type of dental development trait that is used for age estimation of children from birth to 3 years (Townsend and Hammel, 1990).

An implicit assumption in skeletal studies is that the marker used to estimate chronological age (e.g. dental development) is unaffected by malnutrition, disease, and other correlates of mortality. The timing of emergence for deciduous teeth is considered to be robust to moderate environmental insults, malnutrition, and disease (Holtz, 1959; Lewis and Garn, 1960; Demirijan, 1986). Hence, deciduous tooth emergence has been widely used to age children in populations without written age documentation (Townsend and Hammel, 1988) and as a marker of growth and development for children from birth to about age three (Jelliffe and Jelliffe, 1973).

Even if dental traits like tooth emergence are relatively buffered against nutritional and disease stress, this does not rule out the possibility that individuals who are biologically more frail may show a different pattern of dental development (Wood et al., 1992). In health-related studies, death is considered worse than disease—so that individuals who are malnourished or diseased and survive may, in part, be drawn from a population with different biological characteristics than the subset who succumb to death. This selective mortality hypothesis cannot be tested directly, because we have no direct measure of biological frailty. Instead, indirect methods must be used.

Saunders and Hoppa (1993) suggest that this problem can be investigated with historic cemeteries where comparisons can be made between skeletons and documents (e.g. Saunders et al. 1993; Lampl and Johnston, 1996). An alternative way of examining the issue, suggested by Johnston (1968), is by examining growth and development in living, non-industrial populations existing under conditions comparable to prehistoric populations.

In this paper, we use this second approach. We examine the timing of tooth emergence in three studies of growth and development in living children. If there is a biological mortality bias in deciduous tooth emergence, it would appear through differences in the timing or pattern of deciduous dental development between a sample of children who die during a prospective study, and those who do not. Thus, we compare the distributions of deciduous tooth emergence in the subsample of children who survive and the children who do not survive during the studies.

We examined dental emergence in samples from three non-industrial populations: Guatemala, Bangladesh, and Java. Parametric survival analysis was used to assess differences in emergence between children who died during the study period (the mortality sample) and those who survived to the end of the study (the living sample).

The three samples investigated in this paper provide for something of a natural experiment to examine health effects on biological mortality bias. Although all three samples are in non-industrial settings, each sample differs with respect to health and nutrition. The Bangladeshi and Javanese samples are characterized by chronic undernutrition and lack of modern health care. At the start of the Bangladesh study, a severe famine struck the country, thus compounding the chronic stress with an acute stress. The Guatemalan sample is from an intervention study that examined the effect of protein and calorie supplementation on child development; children received limited modern health care as well. We do not imply that any of these populations are representative of prehistoric or historic populations, but they do provide a natural experiment for examining whether a mortality bias exists in deciduous tooth emergence and may also reveal factors that affect the magnitude of any bias.

MATERIALS AND METHODS

Data were collected from three longitudinal studies of deciduous tooth emergence in Javanese, Guatemalan, and Bangladeshi children. In each study, clinical emergence was assessed over periodic intervals. Samples were divided into a *living sample* for those who survived through the study and a *mortality sample* for those who died during the study. For all groups, we estimated parameters and standard errors for the distributions of times to tooth emergence. We used left dentitions only, as it is statistically improper to pool sides as though they are independent observations (Holman and Jones, 1991).

Samples

In each study a tooth was considered emerged if any part of the tooth had, upon direct inspection of the mouth, pierced the gum line (clinical emergence). Designs and protocols for the three studies were broadly similar, only differing in minor characteristics such as age at recruitment, number and timing of visits, study length, and dropout patterns.

Bangladeshi children. These data were collected as part of the Meheran Growth and Development Study conducted between 1974 and 1977 in the rural village of Meheran, located about 50 km south-east of Dhaka (Khan et al., 1979; Khan and Curlin, 1981; Khan et al., 1981). Most of the children were recruited at birth, and all were less than one year of age at the beginning of the study. Dental records were prospectively collected on 397 children by trained personnel. Clinical examinations were conducted monthly for the first year of the study and quarterly for each year thereafter. Previous analyses of the dental data are given elsewhere (Khan and Curlin, 1981; Khan et al., 1981; Holman and Jones, 1998, 2003).

Health care for the village largely consisted of traditional remedies administered by unlicensed village practitioners (Bhuiya and Streatfield 1992). At the start of the study, Bangladesh experienced a severe famine that began in early 1974 and lasted until November 1975. During this time in Bangladesh, death rates increased, birth rates fell sharply, and nutritional status declined (Langsten and Chowdhury, 1984; Bairagi, 1986). About 1.5 million Bangladeshis died in the famine (Razzaque, 1989).

Guatemalan children. These data come from the Institute of Nutrition of Central America and Panama (INCAP) longitudinal study of nutrition and mental development carried out from 1968 to 1977 in four Spanish-speaking farming villages in rural Guatemala. The villages were located in the department of El Progreso, northeast of Guatemala City, in a dry, mountainous area. Most households participated in agricultural production as tenant farmers or small landowners (Habicht and Martorell, 1992).

Mothers and children were examined every 3 months from child's birth to 2 years of age and every 6 months thereafter. The sample includes dental records for 1,277 children, most of whom were enrolled in the study at birth, although some older children were recruited into the study. A description of the dental data as well as analyses of emergence can be found in Delgado et al. (1975) and Holman and Jones (1998, 2003).

Throughout the study, nutritional supplements were provided freely to all residents: two villages received a protein supplement and two received a calorie drink. Over the course of the study, health care was provided by a physician and through clinics employing paramedical personnel. Children who showed signs of malnutrition (marasmus or kwashiorkor) during the study were provided additional nutritional interventions (Habicht and Martorell, 1992; Read and Habicht, 1992). The study interventions resulted in considerable improvements in health and nutrition. Infant mortality was reduced from 139 per 1,000 births prior to 1969 to 55 per 1,000 births by 1972, while national death rates in Guatemala remained constant (Rose et al., 1992).

Javanese children. These data come from the Ngaglik project, a study of breastfeeding behavior and birth interval dynamics carried out from 1976 to 1978 in Central Java, Indonesia (Ngaglik Study Team, 1976; Hull 1978, 1979). Descriptions and analyses of the dental data can be found in Hull (1983) and Holman and Jones (1991, 1998, 2003). A cohort of 510 women were enrolled in the study, based on a recent confinement or pregnancy. Their children were recruited into the study from birth to six months of age. Survey personnel visited the women and their infants every 35 days for up to two and one-half years. Dental records from 468 children were available for analyses.

Households that participated in the study spanned a narrow socioeconomic range from low to moderate income as assessed by an economic survey (Hull 1978, 1983). Most households were involved in rural agriculture, and the mothers regularly engaged in economic activities like farming, farm labor and small-scale trading. The staple food was rice, supplemented by cassava for part of the year. Health care for the children typically consisted of homemade herbal medicines and other traditional cures. Traditional healers were rarely consulted, and then only after efforts within the household failed (Hull, 1979).

Statistical methods

Parametric survival analysis was used to assess differences in deciduous tooth emergence between the subgroup of children who died during the study period and those who survived to the end of the study. The analysis assumes that the distribution of tooth emergence follows a two-parameter lognormal distribution with the children's ages taken from eight months before birth, corresponding to the time just prior to when the dental lamina is formed (Ten Cate, 1998). Using conception (or shortly thereafter) as the starting point for tooth emergence was suggested by Kihlberg and Koski (1954) and Hayes and Mantel (1958), and has been used or discussed in a number of other studies (Magnússon, 1982; Smith et al., 1994; Holman and Jones, 1998).

Call $f(t|a, b)$ the lognormal probability density function for tooth emergence, and $S(t|a, b)$ the corresponding survival function. The mean of the distribution is $a \exp(b^2/2)$, the median is a , and the variance of the distribution is $a^2 \exp(b^2/2) [\exp(b^2/2) - 1]$. Differences between the mortality sample and the living sample in the timing of tooth emergence were assessed by treating mortality as a dummy variable and estimating the effect of that variable on hazard of tooth emergence.

Define \mathbf{x}_i as series of covariates. For the separate analyses of each sample, a single covariate, x_i , is included that is 1 if the i -th child died during the study and 0 if the child survives to the last observation. For the combined analyses, additional covariates are used to code each sample in order to control for differences among populations. We use the Guatemalan sample as the reference population and covariate $x_{Bangladesh}$ is coded as 1 if the sample is from Bangladesh and 0 otherwise; the covariate x_{Java} is coded as 1 if the sample is from Java and 0 otherwise. A vector of parameters ($\boldsymbol{\beta}$) quantify the effects of covariates on the risk of tooth emergence. For m covariates, $\mathbf{x}_i \boldsymbol{\beta} = x_{1i} \beta_1 + x_{2i} \beta_2 + \dots + x_{mi} \beta_m$. Under the proportional hazards model, the effect of $\mathbf{x}_i \boldsymbol{\beta}$ on the survival function is $S_i(t|a, b, \mathbf{x}_i \boldsymbol{\beta}) = S(t|a, b)^{\exp(\mathbf{x}_i \boldsymbol{\beta})}$ (Kalbfleish and Prentice, 1980).

The observations of tooth emergence in this study can be classified as interval censored or right censored (Holman and Jones, 1998). Briefly, interval censored observations are those for which the child's age is known at an examination time prior to emergence (t_u) and an examination time after emergence (t_e). The two times define the interval within which emergence took place. Right-censored observations are those in which the child had not emerged the tooth of interest at that child's last examination in the study. Right censored observations must be included in analyses of times to events to avoid biased results (Kalbfleish and Prentice, 1980; Elandt-Johnson and Johnson, 1980).

Estimates of the model's parameters were found by maximum likelihood. The likelihood for N observations of emergence of a particular tooth is

$$L = \prod_{i=1}^N \left[S(t_{u_i} | a, b)^{\exp(x_i, \beta)} - S(t_{e_i} | a, b)^{\exp(x_i, \beta)} \right] \quad (1)$$

For right-censored observations t_e is taken to be infinity. Maximum likelihood estimates of the parameters are the values of a , b , and β that maximize (1). The statistical package *mle* (version 2.1, Holman, 2003) was used to find parameters.

Uncertainty in the parameter estimates was found by a bootstrap method. For each tooth in each sample, 1,000 bootstrapped datasets were created and parameters were estimated for each data set. The median and the central 95% confidence intervals for the 1,000 parameter estimates were then found.

RESULTS

Table 1 through Table 3 show the parameter estimates for each sample, along with the mean emergence age for the living and mortality samples, and numbers of right-censored observations for both the living sample and the mortality sample. For the Javanese upper and lower second molars (Table 3), all observations for the mortality sample were right-censored, so it was not possible to get stable estimates for the β parameter. The study was only 2½ years long and given the small size of the mortality sample (48), it is not unexpected that all children in the mortality sample died prior to emergence of the second molars. The statistical methods, however, allow us to make use of both censored and uncensored data for all the other teeth.

For the Bangladesh sample (Table 1) and the Javanese sample (Table 3), the 95% central confidence intervals for the β parameter of each tooth enclose zero. In other words, there is no statistically significant evidence of mortality bias in the two populations with the greatest degree of nutritional stress. In the Guatemalan sample (Table 2), the β parameters for the upper and lower first molars were significantly greater than zero. This indicates that children who died during the study were advanced in emergence timing for these teeth relative to children who survived to the end of the study. This finding is surprising, but may reflect a type of biological mortality bias.

Figure 1 shows distributions for the β coefficients for each tooth based on 1,000 bootstrapped estimates. Distributions shifted to the left indicate that the mortality sample exhibited delayed tooth emergence relative to the live sample. Interestingly, the Bangladesh first and second incisors and the first molar in both jaws are consistently shifted to the left by one to two months (Table 1). This trend was not statistically significant, but it does suggest that in a severe famine, the mortality sample will have delayed emergence. In the chronically undernourished Javanese sample, there is no trend apparent, except, perhaps, delayed emergence of the lower canine and slightly accelerated emergence in the lower first incisor for the mortality sample. For the Guatemalan sample, there is a clear trend toward advanced emergence in the canines, and first and second molars of both jaws.

Results from the analysis of all samples simultaneously are given in Table 4. There were no detectible differences for the mortality sample, as the 95% central confidence intervals always enclosed zero. Even so, eight of the ten coefficients were negative, suggesting that there might be an overall slight bias toward delayed emergence in the mortality sample.

Table 4 shows that there were significant differences in the emergence of many teeth between the Guatemalan sample and the other two samples. Relative to the Guatemalan sample, the Bangladesh showed overall delayed emergence for the anterior dentition. The Javanese sample showed delays in emergence in all teeth except the lower first molar. We cannot say whether these

population differences in emergence timing arise from nutritional or other environmental factors, or whether they reflect genetic differences.

DISCUSSION

We assessed whether biological mortality bias exists for dental development in three non-industrial populations by examining differences between surviving individuals and the mortality sample. The three populations formed something of a natural experiment because of somewhat different health conditions experienced by each. The Bangladesh children were experiencing a severe famine and suffered both chronic and acute nutritional stress, the Javanese children were from rural, poor households in an undernourished population, and the Guatemalan children were provided nutritional supplements and health care. Overall, there was no systematic bias toward delayed emergence in the mortality sample, although there was a trend toward delayed emergence in the Bangladeshi sample (Table 1). The magnitude of biological mortality bias was small, however, and it would require a much larger mortality sample to detect the bias with statistical certainty.

For the Guatemalan sample, the posterior dentition was accelerated in the mortality sample relative to the living sample (Table 2). This finding was unexpected, but we propose two hypotheses for the finding. The mothers and children in this sample participated in a nutritional intervention study in which half the villages were provided with a calorie supplement and half were provided with a protein supplement. The first hypothesis is that children who were supplemented in ways that accelerated dental maturation may have been left more susceptible to mortality. For example, the children in the calorie supplementation group may have had faster dental maturation at the expense of lower immune system development or function relative to children in the protein supplementation group. That is, the observation may reflect a biological tradeoff between dental growth and other physiological processes. A second hypothesis is that the results may reflect differences in breastfeeding behavior and supplementation resulting from the nutritional intervention. Children supplemented at an earlier age are at an increased risk of morbidity and mortality because of the loss of passive immunity provided by breast milk and an increased exposure to food-borne pathogens (Popkins et al., 1990; Cunningham et al., 1991; Hanson et al., 1994; Cunningham 1995; Hanson, 2000). A related explanation is that the most well-nourished mothers may have experienced, on average, an earlier return of ovulatory cycles postpartum. Some may have become pregnant while still breastfeeding, leading to an earlier weaning age. Additional research will be needed to uncover the cause and the role, if any, of the nutritional supplementation in the Guatemalan study on this paradoxical outcome, and whether the results hold more broadly.

The overall results of this study suggest that there is little or no biological mortality bias in the timing of deciduous tooth emergence. Thus, the mortality sample (e.g. a skeletal sample) can be considered representative of the living population with respect to deciduous tooth emergence. We suspect that other dental traits (crown and root formation) may be immune from biological mortality bias, as well, but this should be verified for those traits. Our results do not generalize to other skeletal traits such as those used for studying growth. Deciduous tooth emergence has long been recognized as being better buffered against moderate nutritional stress than other skeletal traits (Jelliffe and Jelliffe, 1973). Therefore, non-dental skeletal traits may be more susceptible to biological mortality bias. Biases should be evaluated for these traits using living samples (as we have done here) or from documented cemetery samples as suggested by Saunders and Hoppa (1993).

Even though we have found little evidence for biological mortality bias in deciduous tooth emergence, this does not mean that there are no population differences in tooth emergence. Elsewhere (Holman and Jones, 1998, 2003) we have shown that substantial differences exist among populations in the timing of emergence. Table 4 reiterates these findings, and reinforces the need for

population-specific standards. Lampl and Johnston (1996) have demonstrated the need for population-specific dental development standards for aging skeletons. Holman and Yamaguchi (*in press*) found that delayed deciduous tooth emergence is associated with poor nutrition in Japanese children. If their results hold for other populations, then nutrition may explain some of the differences within populations in tooth emergence, and may lead to differences among populations with different levels of nutritional stress.

Since we found minimal mortality bias under conditions of extreme nutritional stress, it would seem that, on average, mortality acts fast enough in children that no “signature” remains in the timing of dental development. This pattern would be expected in ecological contexts where infant and child mortality result from acute infectious diseases. In Bangladesh, respiratory infections and diarrheal disease are the major cause of death for infants and children who survive the neonatal period (ICDDR,B, 1992). Jelliffe and Jelliffe (1973) and Khan et al. (1981) conclude that most childhood illnesses do not delay dental eruption. Yet, diseases of childhood can result in stunted growth (Saunders and Hoppa, 1993; King and Ulijaszek, 1999). For example, diarrheal disease is associated with stunted growth in undernourished populations (Martorell et al., 1975; Lutter et al., 1992). However, the spectrum of childhood diseases and causes of death may differ by ecological context. Since all three of our samples were in subtropical environments, our results may not apply to children in other contexts, such as arctic or temperate regions. Additional research is needed to determine whether the results can be generalized to other ecological settings.

Longitudinal studies of growth and development in living populations have provided some of the best evidence for biological mortality bias for linear growth traits (reviewed in Saunders and Hoppa, 1993). There are some problems, however, in using these studies for investigating biological mortality bias. The studies require relatively large numbers of participants because of the low level of infant and child mortality. Any future studies will require even larger samples because of contemporary research ethics that would require health intervention for children clearly at risk of mortality. This is the reason there were no un-supplemented control villages in the Guatemalan study (Habicht and Martorell, 1993). Any such interventions may further obscure the presence of mortality bias.

CONCLUSIONS

Examination of deciduous tooth emergence in three subtropical, non-industrial samples revealed no overall biases toward delayed emergence in the mortality sample relative to the living sample. The Bangladeshi and Javanese samples exhibited no evidence for significant biological mortality bias. Paradoxically, the Guatemalan sample exhibited advanced emergence for the posterior dentition in the mortality sample, although we are unable to rule out that this finding is an artifact of the nutritional interventions used in the study. We conclude that deciduous tooth emergence in a mortality sample can be considered representative of emergence in the living population from which the mortality sample was drawn.

ACKNOWLEDGMENTS

We thank Nicole Nuckley for her assistance with this project, Dr. V. Hull for facilitating work in Indonesia, and Dr. Michael Strong and the International Centre for Diarrhoeal Disease Research, Bangladesh for assistance in Bangladesh. Funding for this project was from NSF BNS8115586, NIH NICHD R01HD26899, F32HD07994, and The American Institute for Bangladesh Studies.

LITERATURE CITED

- Armstrong GJ, Mielke JH, Owen KH, VanGerven DP, Dewey JR, Mahler PE. 1972. Bone growth and development in prehistoric populations from Sudanese Nubia. *J Hum Evol* 1:89-119.
- Bairagi R. 1986. Food crisis, nutrition, and female children in rural Bangladesh. *Pop Dev Rev* 12:307-315.
- Bhuiya A, Streatfield K. 1992. A hazard logit model analysis of covariates of childhood mortality in Matlab, Bangladesh. *J Biosoc Sci* 24:447-462.
- Buikstra JE, Cook DC. 1980. Paleopathology: An American account. *Ann Rev Anthropol* 9:433-470.
- Cunningham AS. 1995. Breastfeeding: Adaptive behavior for child health and longevity. In: Stuart-Macadam P and Dettwyler KA, editors. *Breastfeeding: Biocultural Perspectives*. New York: Aldine De Gruyter. p 243-264.
- Cunningham AS, Jelliffe DB, Jelliffe EFP. 1991. Breast-feeding and health in the 1980's: A global epidemiological review. *J Pediatr* 118:659-666.
- Delgado H, Habicht J-P, Yarbrough C, Lechtig A, Martorell R, Malina RM, Klein RE. 1975. Nutritional status and the timing of deciduous tooth eruption. *Am J Clin Nutr* 28: 216-224.
- Demirjian A. 1986. Dentition. In: Falkner F, Tanner JM, editors. *Human Growth*, 2nd edition, New York: Plenum Press. p 269-98.
- Elandt-Johnson RC and Johnson NL. 1980. *Survival Models and Data Analysis*. New York: John Wiley and Sons.
- Habicht J-P, Martorell R. 1993. Objectives, research design, and implementation of the INCAP longitudinal study. *Food Nutr Bull* 14:176-190.
- Hanson LA. 2000. The mother-offspring dyad and the immune system. *Acta Paediatr* 89:252-258.
- Hanson LA, Ashraf R, Zaman S, Karlberg J, Lindblad BS, Jalil F. 1994. Breast feeding is a natural contraceptive and prevents disease and death in infants, linking infant mortality and birth rates. *Acta Paediatr* 83:3-6.
- Hayes RL, N Mantel N. 1958. Procedures for Computing the Mean Age of Eruption of Human Teeth. *J Dent Res* 37:938-947.
- Holman DJ. 2003. *mle: A programming language for building likelihood models*. Version 2.1. User's Manual and Reference Manual. <http://faculty.washington.edu/~djh/mle>.
- Holman DJ, Jones RE. 1991. Longitudinal analysis of deciduous tooth emergence in Indonesian children: I. Life table methodology. *Am J Hum Biol* 3:389-403.
- Holman DJ, Jones RE. 1998. Longitudinal analysis of deciduous tooth emergence II: Parametric survival analysis in Bangladeshi, Guatemalan, Japanese and Javanese children. *Am J Phys Anthropol* 105:209-230.
- Holman DJ, Jones RE. 2003. Longitudinal analysis of deciduous tooth emergence: III. Sexual dimorphism in Bangladeshi, Guatemalan, Japanese and Javanese children. *American Journal of Physical Anthropology* 122:269-278.

- Holman DJ, Yamaguchi K (in press). Longitudinal analysis of deciduous tooth emergence: IV. Covariate effects in Japanese children. *Am J Phys Anthropol*.
- Hotz R. 1959. The relation of dental calcification to chronological and skeletal age. *Trans Eur Orthodont Soc* 35:140-149.
- Hull VJ. 1978. A study of birth interval dynamics in rural Java. In: Mosley WH, editor. *Nutrition and Human Reproduction*. New York: Plenum. p 433-459.
- Hull VJ. 1979. Women, doctors, and family health care: Some lessons from rural Java. *Stud Fam Plan* 10:313-325.
- Hull VJ. 1983. The Ngaglik study: An inquiry into birth interval dynamics and maternal and child health in rural Java. *World Health Stat Q* 36:100-118.
- ICDDR,B. 1992. Demographic surveillance system-Matlab: registration of demographic events—1985. Dhaka, Bangladesh: International Centre for Diarrhoeal Disease Research, Bangladesh.
- Jelleffe EFP, Jelliffe DB, editors. 1973. Deciduous dental eruption, nutrition and age assessment. *J Trop Pediatrics Environ Child Health* 19(Mon. 28):193-248.
- Johnston FE. 1968. Growth of the skeleton in earlier people. In: Brothwell DR, editor. *The Skeletal Biology of Earlier Human Populations*. London: Pergamon Press. p 57-66.
- Johnston FE, Zimmer LO. 1989. In: Iscan MY, Kennedy KAR, editors. *Assessment of growth and age in the immature skeleton. Reconstruction of Life from the Skeleton*. New York: Alan R. Liss. P 11-21.
- Kalbfleisch JD, Prentice RL. 1980. *The Statistical Analysis of Failure Time Data*. New York: John Wiley and Sons.
- Khan M, Curlin GT, Chakraborty J. 1979. Growth and development studies: Rural Meheran, Comilla. *Bangladesh Med J* 7:74-90.
- Khan M, Curlin GT. 1981. Development of milk teeth in rural Meheran children of Bangladesh. Working Paper No. 8. Dhaka, Bangladesh: International Centre for Diarrhoeal Disease Research, Bangladesh.
- Khan M, Chakraborty J, Paul SR. 1981. Teeth as index to health and age in Bangladeshi children. *Nutr Rep Int* 24:963-971.
- Kihlberg J, Koski KK. 1954. On the properties of the tooth eruption curve. *Suom Hammaslaeak Toim* 50 (Suppl. 2):6-9.
- King SE, Ulijaszek SJ. 1999. Invisible insults during growth and development: contemporary theories and past populations. In: Hoppa RD, Fitzgerald CM, editors. *Human Growth in the Past: Studies from Bones and Teeth*. Cambridge: Cambridge University Press. p 161-182.
- Krogman WM. 1968. Biological timing and the dento-facial complex. Part I. *J Dent Child* 35:175-185.
- Lampl M, Johnston. 1996. Problems in the aging of skeletal juveniles: Perspectives from maturation assessments of living children. *Am J Phys Anthropol* 101:345-355.
- Langsten R, Chowdhury SA. 1984. Famine, land, and mortality in rural Bangladesh. *Pop Index* 50:426-436.

- Lewis AB, Garn SM. 1960. The relationship between tooth formation and other maturational factors. *Angle Orthodont* 30:70-77.
- Lutter CK, Habicht J-P, Rivera JA, Martorell R. 1992. The relationship between energy intake and diarrhoeal disease in their effects on child growth: Biological model, evidence, and implications for public health policy. *Food Nutr Bull* 14:36-42.
- Magnússon TE. 1982. Emergence of primary teeth and onset of dental stages in Icelandic children. *Community Dent Oral Epidemiol* 10:91-97.
- Martorell R, Yarbrough C, Lechtig A, Habicht J, Klein RE. 1975. Diarrhoeal diseases and growth retardation in preschool Guatemalan children. *Am J Phys Anthropol* 43:341-352.
- Merchant VL, Ubelaker DH. 1977. Skeletal growth of the protohistoric Arikara. *Am J Phys Anthropol* 46:61-72.
- Ngaglik Study Team. 1978. Birth interval dynamics in village Java: The methodology of the Ngaglik study. Methodology Series No. 4. Population Studies Center, Gadjah Mada University, Yogyakarta, Indonesia.
- Popkin BM, Adair L, Akin JS, Black R, Briscoe J, Flieger W. 1990. Breastfeeding and diarrhoeal morbidity. *Pediatrics* 86:874-882.
- Razzaque A. 1989. Sociodemographic differentials in mortality during the 1974-1975 famine in a rural area of Bangladesh. *J Biosoc Sci* 21:13-22.
- Read MS, Habicht J-P. 1993. History of the INCAP longitudinal study on the effects of early nutrition supplementation in child growth and development. *Food Nutr Bull* 14:169-177.
- Rose D, Martorell R, Rivera J. 1992. Infant mortality rates before, during, and after a nutrition and health intervention in rural Guatemalan villages. *Food Nutr Bull* 14:215-220.
- Saunders SR. 1992. Subadult skeletons and growth related studies. In: Saunders SR, Katzenberg MA, editors. *Skeletal Biology of Past Peoples: Research Methods*. New York: Wiley-Liss. P1-20.
- Saunders S, DeVito C, Herring A, Southern R, Hoppa R. 1993. Accuracy tests of tooth formation age estimations for human skeletal remains. *Am J Phys Anthropol* 92:173-188.
- Saunders SR, Hoppa RD. 1993. Growth deficits in survivors and non-survivors: Biological mortality bias in subadult skeletal samples. *Yrbk Phys Anthropol* 36:127-151.
- Smith BH, Crummett TL, Brandt KL. 1994. Ages of eruption of primate teeth: A compendium for aging individuals and comparing life histories. *Yrbk Phys Anthropol* 37:177-231.
- Ten Cate AR. 1998. *Oral Histology: Development, Structure and Function*. Fifth edition. St. Louis: Mosby.
- Townsend N, Hammel EA. 1990. Age estimation from the number of teeth erupted in young children: An aid to demographic surveys. *Demography* 27:165-174.
- Wood JW, Milner GR, Harpending HC, Weiss KM. 1992. The osteological paradox: Problems of inferring prehistoric health from skeletal samples. *Curr Anthropol* 33:343-369.

Table 1. Parameter estimates, mean emergence times, and percent censored observations for the Bangladeshi sample^a.

Tooth	Median parameter estimates ^b (95% central confidence interval)			Mean emergence age (months)		Percent right censored	
	<i>a</i>	<i>b</i>	β	Alive	Died	Alive	Died
<i>i</i> ¹	2.97 (2.95, 2.99)	0.162 (0.138, 0.189)	-0.509 (-1.243, 0.419)	11.8	13.2	19.5	45.5
<i>i</i> ²	3.06 (3.04, 3.08)	0.167 (0.146, 0.191)	-0.730 (-1.586, 0.279)	13.6	16.2	23.6	60.6
<i>c</i> ¹	3.36 (3.34, 3.38)	0.124 (0.109, 0.142)	-0.070 (-0.787, 0.650)	21.0	21.2	36.8	69.7
<i>m</i> ¹	3.17 (3.15, 3.18)	0.112 (0.095, 0.132)	-0.598 (-1.473, 0.315)	15.9	17.4	29.7	63.6
<i>m</i> ²	3.55 (3.53, 3.58)	0.150 (0.118, 0.185)	-0.039 (-0.589, 0.482)	27.0	27.2	40.9	72.7
<i>i</i> ₁	2.90 (2.88, 2.92)	0.144 (0.129, 0.163)	-0.702 (-1.471, 0.344)	10.4	12.3	17.0	45.5
<i>i</i> ₂	3.17 (3.15, 3.19)	0.166 (0.144, 0.188)	-0.558 (-1.306, 0.330)	16.1	18.2	29.4	66.7
<i>c</i> ₂	3.41 (3.38, 3.43)	0.145 (0.117, 0.177)	0.033 (-0.598, 0.645)	22.5	22.4	39.0	69.7
<i>m</i> ₁	3.25 (3.22, 3.27)	0.123 (0.098, 0.153)	-0.714 (-1.677, 0.645)	17.9	19.9	32.1	69.7
<i>m</i> ₂	3.56 (3.54, 3.58)	0.133 (0.100, 0.164)	0.019 (-0.803, 1.176)	27.3	27.2	41.5	72.7

^aA sample of 397 children of whom 38 died during the study.

^bBased on 1,000 bootstrapped replicates.

Table 2. Parameter estimates, mean emergence times, and percent right-censored observations for the Guatemalan sample^a.

Tooth	Median parameter estimates ^b (95% central confidence interval)			Mean emergence age (months)		Percent right censored	
	<i>a</i>	<i>b</i>	β	Alive	Died	Alive	Died
<i>i</i> ¹	2.91 (2.90, 2.92)	0.125 (0.111, 0.140)	0.026 (-0.568, 0.783)	10.6	10.5	7.6	55.3
<i>i</i> ²	2.95 (2.93, 2.96)	0.132 (0.122, 0.144)	-0.059 (-0.659, 0.738)	11.3	11.5	8.6	57.9
<i>c</i> ¹	3.30 (3.29, 3.31)	0.111 (0.103, 0.120)	0.435 (-0.378, 1.137)	19.3	18.2	14.3	71.1
<i>m</i> ¹	3.18 (3.17, 3.20)	0.098 (0.087, 0.107)	0.509 (0.056, 1.245)	16.1	15.3	12.4	63.2
<i>m</i> ²	3.58 (3.57, 3.59)	0.104 (0.097, 0.112)	0.811 (-0.276, 2.283)	28.0	25.8	21.2	86.8
<i>i</i> ₁	2.78 (2.77, 2.79)	0.140 (0.124, 0.155)	-0.243 (-0.975, 0.449)	8.3	8.8	5.4	47.4
<i>i</i> ₂	3.07 (3.06, 3.09)	0.145 (0.134, 0.155)	0.197 (-0.283, 0.768)	13.8	13.3	10.9	57.9
<i>c</i> ₂	3.33 (3.32, 3.34)	0.114 (0.106, 0.122)	0.382 (-0.332, 0.921)	20.1	19.1	14.8	73.7
<i>m</i> ₁	3.23 (3.21, 3.25)	0.191 (0.092, 0.195)	0.584 (0.120, 1.162)	17.4	16.3	12.9	63.2
<i>m</i> ₂	3.55 (3.54, 3.56)	0.120 (0.109, 0.138)	0.200 (-0.452, 0.570)	27.0	26.3	20.8	86.8

^aA sample of 1,271 children and 39 who died during the study.

^bBased 1,000 bootstrapped replicates.

Table 3. Parameter estimates, mean emergence times, and percent censored observations for the Javanese sample^a.

Tooth	Median parameter estimates ^b (95% central confidence interval)			Mean emergence age (months)		Percent right censored	
	<i>a</i>	<i>b</i>	β	Alive	Died	Alive	Died
<i>i</i> ¹	2.94 (2.93, 2.96)	0.129 (0.119, 0.141)	0.012 (-0.528, 0.495)	11.1	11.1	5.7	59.4
<i>i</i> ²	3.04 (3.02, 3.05)	0.145 (0.134, 0.155)	0.081 (-0.476, 0.731)	13.2	13.0	8.0	59.4
<i>c</i> ¹	3.34 (3.33, 3.36)	0.117 (0.107, 0.129)	-0.113 (-0.895, 0.472)	20.4	20.7	32.6	81.3
<i>m</i> ¹	3.23 (3.22, 3.24)	0.098 (0.091, 0.106)	0.053 (-0.442, 0.687)	17.4	17.3	17.0	71.9
<i>m</i> ²	3.60 (3.58, 3.62)	0.097 (0.082, 0.114)	—	28.7	—	83.7	100.0
<i>i</i> ₁	2.90 (2.88, 2.91)	0.144 (0.133, 0.155)	0.182 (-0.304, 0.629)	10.4	10.0	5.0	53.1
<i>i</i> ₂	3.18 (3.16, 3.19)	0.153 (0.142, 0.165)	0.101 (-0.453, 0.645)	16.3	16.0	16.8	68.8
<i>c</i> ₂	3.40 (3.38, 3.41)	0.123 (0.112, 0.135)	-0.918 (-1.939, 0.183)	22.1	25.8	43.7	93.8
<i>m</i> ₁	3.28 (3.27, 3.29)	0.103 (0.095, 0.112)	0.096 (-0.473, 0.819)	18.7	18.5	22.7	75.0
<i>m</i> ₂	3.59 (3.57, 3.61)	0.103 (0.088, 0.118)	—	28.3	—	81.3	100.0

^a A sample of 468 children of whom 48 died during the study.

^a Based 1,000 bootstrapped replicates.

Table 4. Parameter estimates from an analysis of all three samples.

Tooth	Median parameter estimates ^a (95% central confidence interval)				
	<i>a</i>	<i>b</i>	$\beta_{Bangladesh}$	β_{Java}	β_{Died}
<i>i</i> ¹	2.91 (2.90, 2.92)	0.125 (0.115, 0.135)	-0.572 (-0.775, -0.356)	-0.272 (-0.461, -0.105)	-0.220 (-0.695, 0.271)
<i>i</i> ²	2.95 (2.94, 2.96)	0.126 (0.117, 0.135)	-0.893 (-1.093, -0.715)	-0.643 (-0.782, -0.468)	-0.258 (-0.691, 0.239)
<i>c</i> ¹	3.30 (3.29, 3.31)	0.108 (0.102, 0.115)	-0.546 (-0.752, -0.350)	-0.407 (-0.555, -0.253)	0.064 (-0.355, 0.425)
<i>m</i> ¹	3.18 (3.17, 3.19)	0.098 (0.089, 0.104)	-0.015 (-0.325, 0.248)	-0.448 (-0.609, -0.314)	-0.179 (-1.33, 0.435)
<i>m</i> ²	3.58 (3.56, 3.59)	0.134 (0.105, 0.137)	0.054 (-0.318, 0.352)	-0.455 (-0.780, -0.156)	0.156 (-2.53, 0.927)
<i>i</i> ₁	2.79 (2.78, 2.80)	0.128 (0.118, 0.137)	-0.783 (-0.963, -0.632)	-0.692 (-0.867, -0.570)	-0.276 (-0.762, 0.220)
<i>i</i> ₂	3.08 (3.06, 3.09)	0.137 (0.130, 0.145)	-0.676 (-0.886, -0.503)	-0.674 (-0.826, -0.549)	-0.116 (-0.484, 0.280)
<i>c</i> ₂	3.34 (3.32, 3.35)	0.131 (0.106, 0.134)	-0.560 (-0.881, -0.260)	-0.469 (-0.711, -0.219)	-0.134 (-3.46, 0.464)
<i>m</i> ₁	3.27 (3.21, 3.31)	0.184 (0.090, 0.188)	0.094 (-0.636, 0.828)	0.103 (-0.678, 0.422)	-0.067 (-2.01, 0.631)
<i>m</i> ₂	3.56 (3.54, 3.57)	0.134 (0.112, 0.137)	-0.029 (-0.450, 0.256)	-0.476 (-0.778, -0.181)	-0.043 (-3.40, 0.728)

^a Based 1,000 bootstrapped replicates.

Fig. 1 Distributions of β parameter estimates for each tooth (except the Javanese second molars) in the three samples, from 1,000 bootstrapped replicates. Negative values (a lower hazard) imply a later age at emergence in the mortality sample. Vertical lines pass through zero for each tooth.

