Rate of enamel formation and hypoplasia timing

• Abdulla Al-Shorman, Mohammad Alrousan, Ali Khwaileh •

Department of Anthropology, Faculty of Archaeology and Anthropology, Yarmouk University
Irbid - Jordan

Address for correspondence:
Abdulla Al-Shorman
Department of Anthropology
Faculty of Archaeology and Anthropology
Yarmouk University
Irbid - Jordan
E-mail: alshorman@yu.edu.jo

Bull Int Assoc Paleodont. 2014;8(1):203-208.

Abstract

Linear Enamel Hypoplasia (LEH) is a decrease in enamel thickness resulting from a temporary disruption in enamel formation during times of dental development. To establish a statistical relationship between the timing of enamel formation and the length of the crown, the published data of Reid and Dean (2006) were statistically analyzed using SPSS. The results indicate that the relationship between the crown length and the days of enamel formation is nonlinear as proposed by previous studies but exponential taking the form of $y = a \, . \, e^{bx}$, where *y* is the time of enamel formation in days, *a* and *b* are constants, and *x* is the crown length. Based on this equation, the timing of hypoplasia can be estimated more accurately using *x* as the distance of hypoplasia from the cemento enamel junction (CEJ). This study introduces a new equation for estimating the period of stunt growth (number of days of a hypoplastic event) depending on the horizontal shift of the mathematical function that represents the above relationship. This period may classify the LEH either as caused by acute growth disruptions or chronic and prolonged ones.

Keywords: Linear Enamel Hypoplasia; Enamel Thickness; Dental Development

Introduction

Linear Enamel Hypoplasia (LEH) is a decrease in enamel thickness resulting from a temporary disruption in enamel formation during times of dental development, where it appears as furrows on the crown's surface (1, 2). Causes of LEH include episodes of febrile disease, malnutrition, trauma, parasite infection, weaning, and congenital disorders. These systemic stresses disrupt the activity of ameloblasts as they secrete the enamel matrix (3-7). In bioarchaeological studies, the prevalence of this defect has been used to reconstruct the timing of stress in ancient societies and to understand the childhood health status (1, 8-10).

The timing of formation of this defect was calculated by measuring the distance between the defect and the cemento-enamel junction using either charts or linear regression equations (11-15). These studies assumed a constant rate of growth of tooth crown, whereas the average daily rate of enamel apposition for any maxillary premolar, for example, is 2.7 to 4.6 microns/day (16). In deciduous enamel, rates vary between 2.5 to 4.5 microns throughout/day (17). In general, the formation rate is high early in crown formation and slower later (18). Consequently, this study hypothesizes that the rate of enamel formation across the crown length is nonlinear.

Enamel has two regularly occurring markers: daily cross-striations and long-period striae of Retzius that have been used to determine crown formation times, enamel growth trajectories, and ages at death (19). Striae of Retzius reach the tooth surface as grooves called perikymata (20) and form over 7 to11 days with a mode periodicity of 8 days (21). The formation time is estimated by counting the number of cross-striations between adjacent striae of Retzius in a longitudinal tooth section (22). Histological and experimental studies show that cross striations are formed over 24-hour intervals (23-25). The study by King and his colleagues (26) stressed on the non-linearity relationship between timing of crown formation and its length. Ritzman and coworkeres (27) compared the histological method of hypoplasia timing by Reid and Dean (19) with the macroscopic method (21), where they found that the histological method yields older ages of hypoplasia but they did not suggest which method is precise. This study establishes new regression equations for enamel formation, where the time of hypoplasia formation can be estimated more precisely.

Materials and methods

The data in this study were collected by Reid and Dean (19) who analyzed samples of modern European and African human teeth to establish a rate of human dental enamel formation. Only the data on anterior teeth of modern Europeans were used in this study. These data were statistically analyzed using SPSS v. 13 where the best equation is selected based on the higher correlation coefficient.

Results and discussion

The best fit between the crown length and the time of enamel formation was obtained using an exponential function (figure 1) with a very high correlation coefficient that is close to 1 (table 1). The regression equation between the crown length and days of enamel formation in an anterior tooth takes the form of:

$$y = a \, . \, e^{bx}$$

where y is the time in days, a and b are constants, and x is the crown length.

In a hypoplastic tooth, the exponential function changes when normal enamel formation is resumed (fig. 2). If we consider for example the lower canine, the exponential function of enamel formation will be shifted vertically and horizontally according to the following equation:

$$y = k_i + 372.44 \, . \, e^{0.1555x_i + c_i}$$

where k is the vertical shift in the exponential function and represents the period of stunt growth (the period of the hypoplastic event in days), c is the horizontal shift in the exponential function and represents the width of LEH, y is the time in days, and x is the distance to LEH from CEJ in mm. The value of k is actually the difference in the crown length between the average population crown length (the average crown length of unaffected teeth) and a hypoplastic crown length assuming that the healthy crown is longer than a hypoplastic one:

The period of stunt growth (days) = the average population crown length (mm) - hypoplastic crown length (mm)

$$k_i = 372.44 \, . \, e^{0.1555x} - 372.44 \, . \, e^{0.1555x_i + c_i}$$

For example, assuming that the average length of the lower canine crown of an archaeological population is measured to be 11.00 mm, the crown length of a hypoplastic lower canine is measured to be 10.80 mm and the width of LEH is 0.05 mm, the value of k would be calculated as follows (see also figure 2 below):

$$k_i = 372.44 \, . \, e^{0.1555*11} - 372.44 \, . \, e^{0.1555*10.8+0.05} = \textit{39.31 days}$$

According to (28), stress duration is estimated by counting the number of perikymata within the occlusal wall of each defect multiplied by the circaseptan rhythm factor after (29, 21, 30). This method is invasive as it requires microscopic analysis while the regression equation above relies on macroscopic measurements of LEH and crown length. In the field of bioarchaeology, the value of k (period of stunt growth) may shed more light on the cause of LEH; acute growth disruptions may lower the value of k while chronic and prolonged disruptions do the opposite.

Conclusion

The value of crown length (x) is a population-specific value, which necessitates counting the number of brown stria of Retzius in each tooth type under the microscope and then establishing the timing schedule of enamel formation. The population-specific values (x and y) can be then used to generate population-specific exponential equations. For example, the y and x values can be established for the Byzantine populations of the Levant and later be applied on Byzantine teeth that are geographically and ethnically similar.

References

1. Hillson S. Dental Anthropology. Cambridge: Cambridge University Press. 1996.

2. Hillson S. Bond, S. Relationship of enamel hypoplasia to the pattern of tooth crwon growth: a discussion. Am J Phy Anthropol1997; 104, 89-103.

3. Skinner M. Goodman A. Anthropological uses of developmental defects of enamel. In: Skeletal biology of past peoples: research methods. Ed by S. Saunders, and K. M. New York: Wiley-Liss. 1992 p; 153-174.

4. Aldred M. Savarirayan R. Crawford P. Amelogenesis imperfecta : a classification and catalogue for the 21st century. Oral Dis 2003; 9: 19-23.

5. Goodman A. Martinez C. Chavez A. Nutritional supplementation and the development of linear enamel hypoplasias in children from Tezonteopan, Mexico. Am J Clinic Nutri 1991; 53: 773-787.

6. May R. Goodman A. Meindel R. Response of bone and enamel formation to nutritional supplementation among malnourished Guatemalan children. Am J Phy Anthropol 1993; 92: 37-51.

7. Curruccini R. Handler J. Jacobi K. Chronological distribution of enamel hypoplasias and weaning in a Caribbean slave population. Human Biology 1985; 57 (4): 699-711.

8. Temple D. Dietary variation and stress among prehistoric Jomon foragers from Japan. Am J Phy Anthropol 2007; 133: 1035-1046.

9. Pechenkina M. Roberta A. Zhijun W. Diet and health changes at the end of the Chinese Neolithic: the Yangshao/Longshan transition in Shaanxi Pietrusewsky. Am J Phy Anthropol 2002; 117: 15-36.

10. Buzon M. Health of the non-elites at Tombos: nutritional and disease stress in New Kingdom, Nubia. Am J Phy Anthropol 2006; 130: 26-37.

11. Goodman, A. and Armelagos, G. The chronological distribution of enamel hypoplasia in human permanent incisor and canine teeth. Arch Oral Biol 1985; 30 (6): 503-507.

12. Goodman, A. and Rose, J. Assessment of sysytematic physiological perturbation from dental enamel hypoplasia and associated histological structures. Yearb Phys Anthropol 1990; 33: 59-110.

13. Goodman A. Sorg R. Sources of variation in estimated ages at formation of linear enamel hypoplasia. In: Human Growth in the past. Edited by R. Hoppa, and C. Fitzgerald. Cambridge: Cambridge University Press. 1999 p; 210-240.

14. Wright L. Intertooth patterns of hypoplasia expression. Am J Phy Anthropol 1997; 102: 233-247.

15. Lovell L. Whyte I. Patterns of dental enamel defects at ancient Mendes, Egypt. Am J Phy Anthropol 1999; 110: 69-80.

16. Resnis S. Enamel apposition rate and the prism periodicity in human teeth. Scand J Dent Res 1986; 94 (5): 394-404.

17. Birch W. Dean C. Rates of enamel formation in human deciduous teeth. Front Oral Biol. 2009; 13: 116-120.

18. Shellis R. Utilization of periodic markings in enamel to obtain information on tooth growth. J Hum Evol. 1998; 35 (4-5): 387-400.

19. Reid D. Dean M. Variation in modern human enamel formation times. J Hum Evol. 2006; 50: 329-346.

20. Beynon A. (1992). Circaseptan rhythms in enamel development in modern humans and Plio-Pleistocene hominids. In:

Structure, Function and Evolution of Teeth . edited by P. Smith.. London: Freund Publishing House Ltd. 1992 p: 295-309.

21. Reid D. Ferrell R. The relationship between number of striae of Retzius and thier periodicity in imbricational enamel formation. J Hum Evol. 2006; 50: 195-202.

22. FitzGerald C. Do enamel microstructures have regular time dependency? J Hum Evol. 1998; 35: 371-386.

23. Ten Cate A. Oral histology: development, structure, and function. New York: The CV Mosby Company. 1998.

24. Smith T. Experimental determination of the periodicity of incremental features of enamel. J Anat 2006; 208: 99-113.

25. Antoine D. Hillson S. Dean, M. The developmental clock of dental enamel: a test for the periodicity of prism cross-striations in modern humans and evaluation of the most likely sources of error in histological studies of this kind. J Anat 2009; 214: 45-55.

26. King T. Hillson S. Humphrey L. A detailed study of enamel hypoplasia in post-medival adolescent of known age and sex. Arch Oral Biol 2002; 47: 29-39.

27. Ritzman T. Baker B. Gary T. Schwartz G. A Fine Line: A Comparison of Methods for Estimating Ages of Linear Enamel Hypoplasia Formation. Am J Phys Anthropol 2008: 135; 348-361.

28. Temple D. Nakatsukasa M. McGroarty J. Reconstructing patterns of systemic stress in a Jomon period subadult using incremental microatructure of enamel. J Arch Sci 2012;39: 1634-1641.

29. Guatelli-Steinberg D. Larsen C. Hutchinson D. (2004). Prevalence and duration of linear enamel hypoplasia: a comparative study of Neanderthals and Inuit foragers. J Hum Evol 2004; 47: 65-84.

30. Smith T. Reid D. Dean M. Olejniczak A. Ferrell R. Marin L. (2007). New perspectives on chimpanzee and human molar crown development. In: Dental perspectives on human evolution . edited by S. Bailey, and J. Hublin. New York: Springer. 2007 p; 177-192.

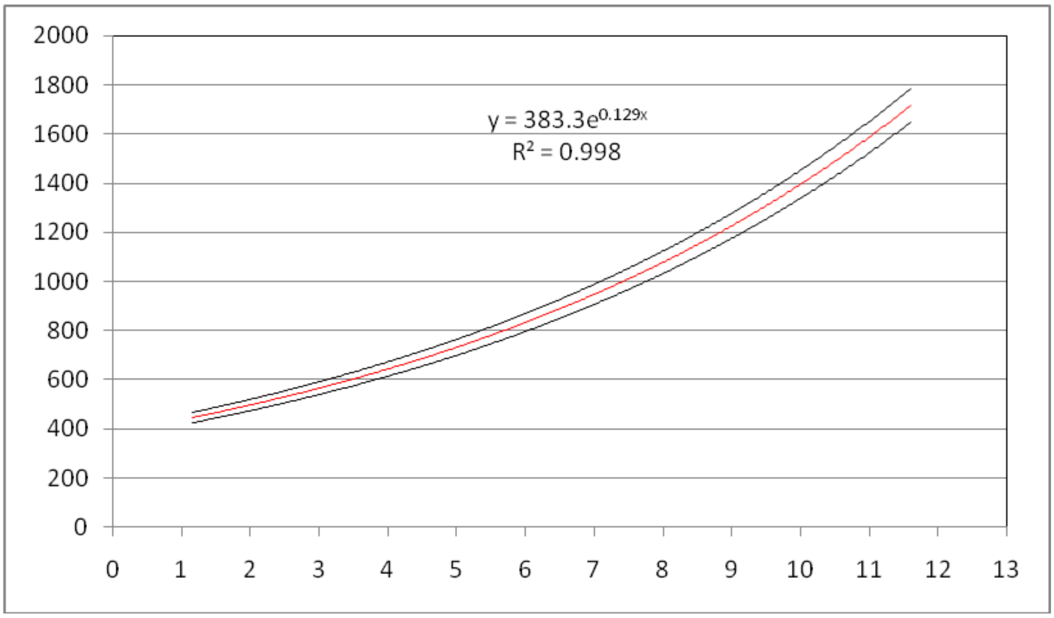

Figure 1 The rate of enamel formation for the first upper incisor.

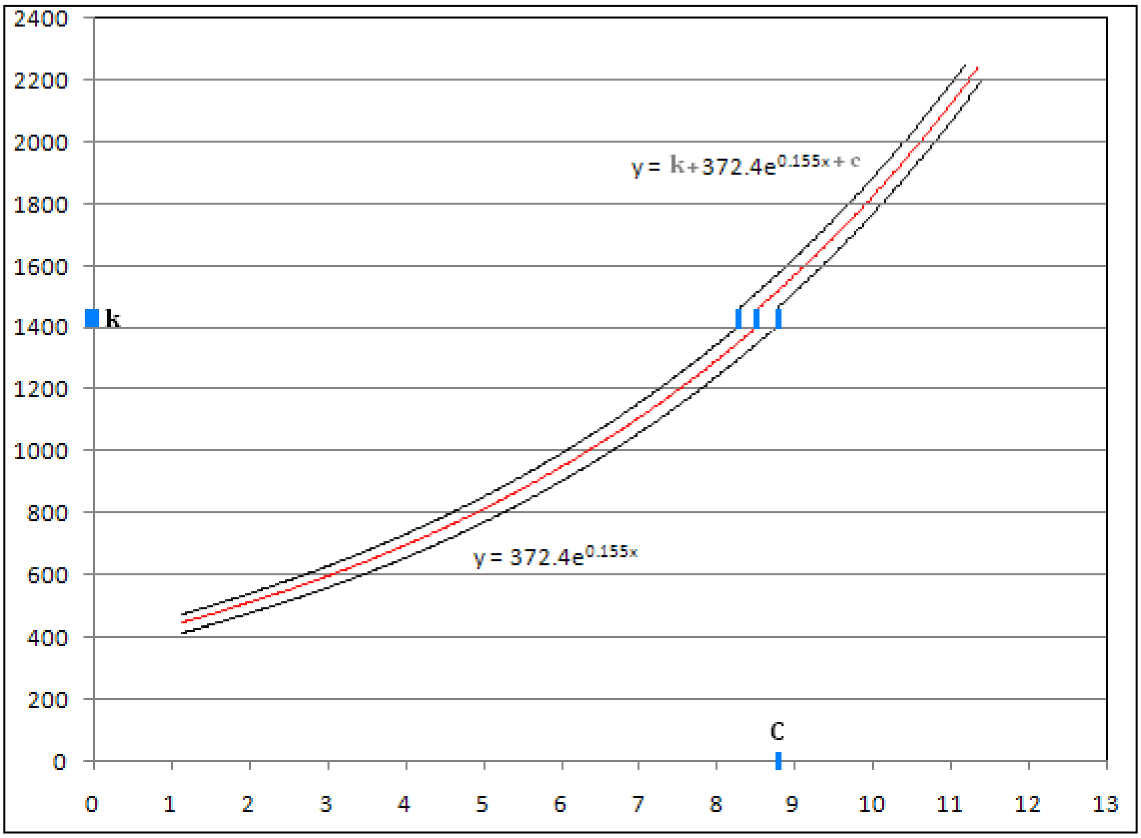

Figure 2 Lower canine vertical and horizontal shift of the regression equation.

Tooth type	Regression equation	R^2
First upper incisor	$y = 383.36 \,.\, e^{0.1292x}$	0.9981
Second upper incisor	$y = 382.19 \,.\, e^{0.1401x}$	0.9984
Upper canine	$y = 383.36 \,.\, e^{0.1384x}$	0.9981
First lower incisor	$y = 280.73 \,.\, e^{0.1568x}$	0.9954
Second lower incisor	$y = 234.55 \,.\, e^{0.1827x}$	0.9956
Lower canine	$y = 372.44 \,.\, e^{0.1555x}$	0.9964

Table 1 Regression equations for enamel formation by tooth type