5-2021

# Enamel Histological Indicator: Growth in Incisors vs. Canines

Nisha Zaver
*University of Tennessee, Knoxville*, nzaver724@gmail.com

# Enamel Histological Indicator: Growth in Incisors vs. Canines

By: Nisha Zaver

Advisor: Dr. Murray Marks

Chancellor's Honors Program

General Dentistry Department

University of Tennessee Medical Center, Knoxville

May 2021

**Table of Contents**

Introduction…… 3

Background…… 4

Materials and Methods…… 8

Results…… 9

Discussion…… 13

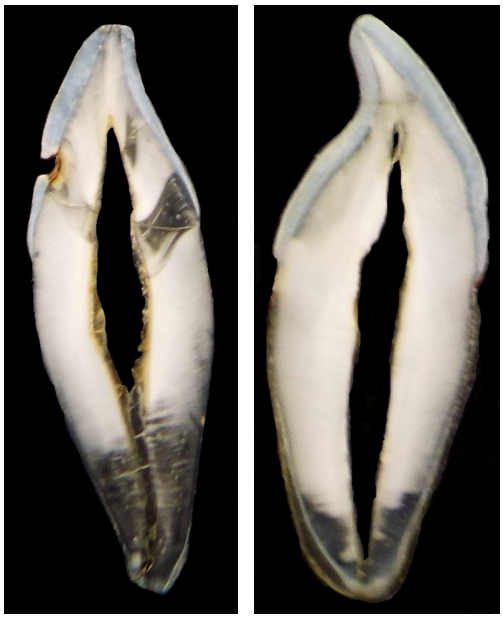
References…… 14

**Introduction**

Dental literature consists of examples of compromised odonto-skeletal growth disturbances resulting from long-term metabolic upsets, i.e., protein-calorie malnutrition, fever and infection (see v. Koenigswald and Sander, 1997; McCauley and Somerman, 2012; Thompson et. al., 2003). A metric evaluation and comparison of permanent canines and incisors with these defects has not been evaluated. I wondered if every tooth was susceptible to these defects. My project will investigate if there is a difference in histological growth disturbances between permanent incisors and canines. I will be using a sample of forty-four teeth from modern Whites (European-Americans) collected from east Tennessee oral surgeons. These teeth have been collected to identify growth disturbances in the enamel crown (i.e. accentuated Striae of Retzius) and to compare these disturbances between incisors and canines for a thesis written in 1988.

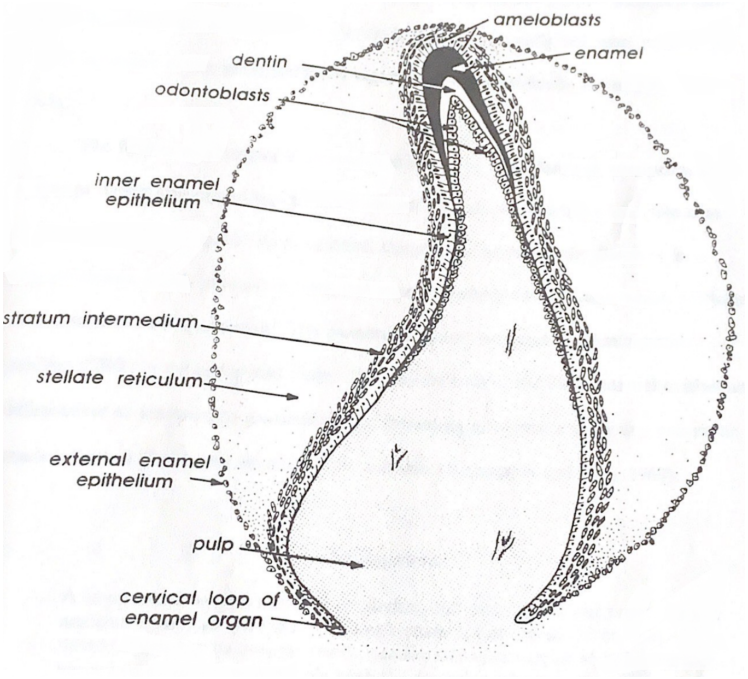
Tooth enamel keeps a permanent record of any metabolic upsets that result in growth arrest. Since the tooth crown initiates and terminates enamel formation at specific times, the mark of any defect is accurately correlated to the age of that person. Growth compromised children display skeletal (i.e. Harris lines) and dental (i.e. hypoplasias) indicators of moderate to long-term morbidity. A visual recording of the coronal region of the tooth in “ill” individuals will be able to assess if microscopic stress lines of morbidity occur more in permanent incisors or canines.

**Background**



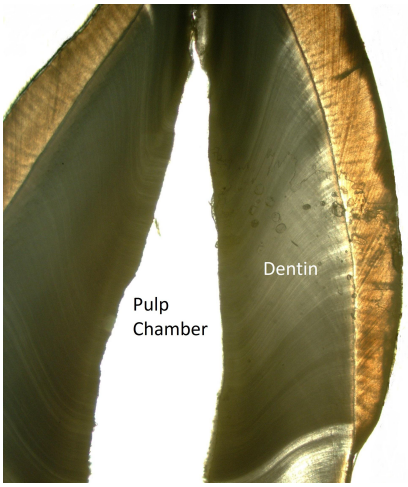
(Figure 1)

Tooth development begins with a tooth bud (Figure 2). The ingredients of this tooth bud consists of the ectoderm and mesoderm, which interact to form the basis of the enamel organ.



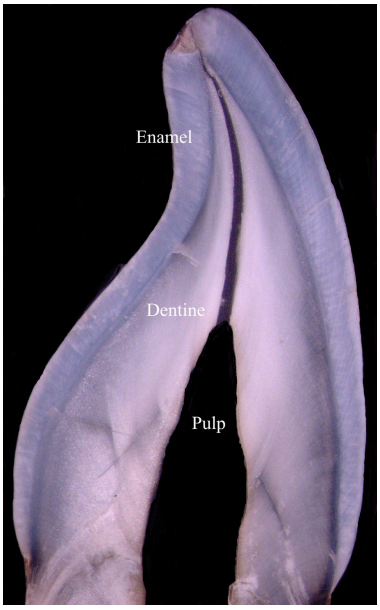

(Figure 2) (Marks, 1993)

Ectodermal ingredients give rise to enamel, while mesodermal ingredients give rise to the dentine, pulp, periodontal ligament, and alveolar bone (Figure 2).



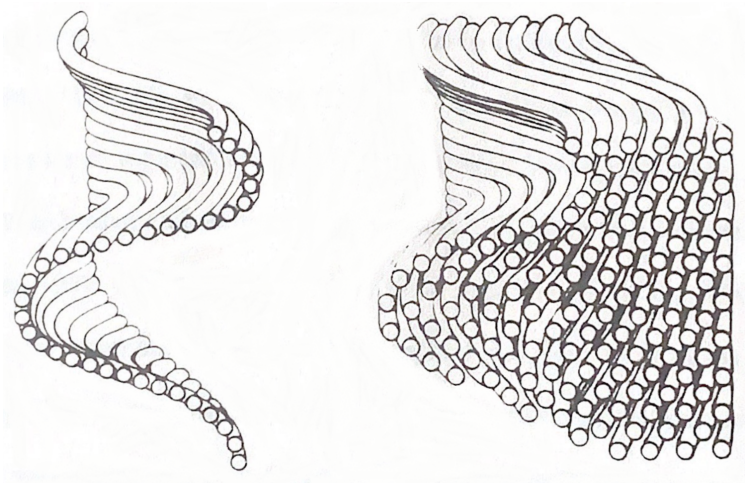

(Figure 2)

The inner enamel epithelium (IEE) of the developing enamel organ genetically determines part of crown and root. The IEE sends a signal to the other IEE cells in the basement membrane to differentiate into odontoblasts. Odontoblasts will then secrete dentine until they get to the pulp chamber and then they will stop (Figure 3).




(Figure 3)

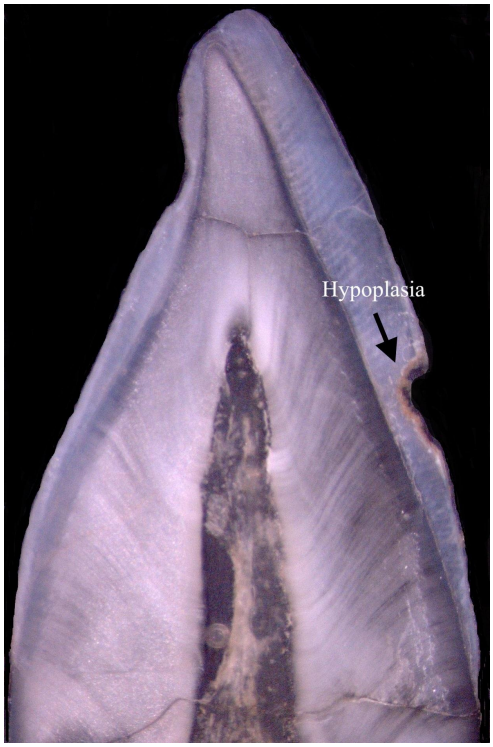
Once dentine is noted by IEE, ameloblasts are formed from IEE cells on the basement membrane as well. The thickness of enamel is how far ameloblasts travel from the basement membrane to the outer tooth surface, where the ameloblasts know where to stop. The Tomes process is important for development of enamel. It is only present prior to and during secretion and absorption of organic materials. Enamel prisms are unique structures of enamel, which is formed by secretion the behavior of Tomes process (Figure 4).



(Figure 4)

These enamel prisms will become arranged into layers of cross-sections to form Hunter-Schreger bands, which help to strengthen the enamel and prevent breakage of the tooth. The development of enamel prisms and Hunter-Schreger bands will form striae of Retzius. These are incremental growth lines that represent the start and stop in the growth of enamel. However, microscopic defects can occur within these incremental growth lines causing accentuated striae of Retzius, or Wilson bands. These are caused by acute stresses such as an illness that lasts a short period (i.e. a few weeks). Wilson bands are very apparent within the enamel of teeth, because they run parallel to Striae of Retzius (Marks, 1993). Furthermore, chronic stress causes enamel hypoplasias, which represent premature ameloblast morbidity (Figure 5). Hypoplasias are a matrix secretion

deficiency which causes altered thickness (i.e. pits). This defect can be seen with the naked eye and is caused by illnesses that last over the course of a few years.




(Figure 5)

**Materials and Methods**

Examination/documentation of developmental defects and microphotography uses a *Leica DMRX*® research microscope at 10, 25, 100 and 200x. Each thin section of both canines and incisors were detailed in a log to ensure proper documentation of Wilson bands. Wilson bands were visually counted on the labial side from the cervical margin to the cusp tip of the crown. The number 1 represents canines, while the number 2 represents incisors (Table 1). Once the total number of Wilson bands were found, each tooth was then divided into 3 sections (i.e. incisal region, mid-coronal region, and cervical region), and Wilson bands were counted on the labial side of these 3 sections. Percentages of Wilson bands were calculated by adding up Wilson bands in each section and then divided by the total numbers of bands found previously.

**Results**

Based on the data collected, incisors have more Wilson bands (Table 1). For canines, Wilson bands were more present in the mid-coronal region (42.4%), then the incisal region (39.4%), and lastly the cervical region (18.2%). On the other hand Wilson bands were more present in the cervical region (48.45% ), then the mid-coronal region (46.4%), and then the incisal region (5.15%) for incisors.

| Canines | Incisors |
|---|---|
| 1. 9004-1: 6 | 23. 5020-2: 15 |
| 2. 5521-1: 4 | 24. 0835A-2: 4 |
| 3. 6907-1: 8 | 25. 0326-2: 5 |
| 4. 0040-1: 8 | 26. 8941-2: 4 |
| 5. 4914-1: 3 | 27. 0049-2: 4 |
| 6. 7792-1: 2 | 28. 2482-2: 13 |
| 7. 1623-1: 0 | 29. 2009-2: 8 |
| 8. 1623-1: 2 | 30. 5384-2: 19 |
| 9. 9413-1: 7 | 31. 8549-2: 40+ |
| 10. 506-1: 8 | 32. 5374-2: 10 |
| 11. 6841-1: 15 | 33. 0054-2: 6 |
| 12. 6977-1: 8 | 34. 89941-2: 15 |
| 13. 4914-1: 7 | 35. 6991-2: 8 |
| 14. 1623-1: 7 | 36. 7975-2: 11 |
| 15. 4390-1: 10 | 37. 7649-2: 12 |
| 16. 5521-1: 7 | 38. 5374-2: 10 |
| 17. 8175-1: 4 | 39. 5980-2: 10 |
| 18. 1417-1: 10 | 40. 2482-2: 26 |

| | |
|---|---|
| 19. 7576-1: 10 | 41. 2830-2: 12 |
| 20. 6907-1: 4 | 42. 8623-2: 19 |
| 21. 5417-1: 3 | 43. 3596-2: 24 |
| 22. 1012-1: 0 | 44. 8623-2: 16 |

Wilson bands of canines and incisors. (Table 1)

| Tooth Identification | Incisal Region | Mid-Coronal Region | Cervical Region |
|---|---|---|---|
| 1. 4390-1 | 9 | 1 | 0 |
| 2. 506-1 | 5 | 0 | 3 |
| 3. 6907-1 | 3 | 2 | 3 |
| 4. 6907-1 | 2 | 2 | 0 |
| 5. 1417-1 | 3 | 7 | 0 |
| 6. 5417-1 | 1 | 2 | 0 |
| 7. 7576-1 | 2 | 4 | 4 |
| 8. 1012-1 | 0 | 0 | 0 |
| 9. 9004-1 | 3 | 2 | 1 |
| 10. 5521-1 | 2 | 1 | 1 |
| 11. 0040-1 | 3 | 2 | 3 |
| 12. 4914-1 | 0 | 3 | 0 |
| 13. 7792-1 | 0 | 1 | 0 |
| 14. 1623-1 | 0 | 2 | 0 |
| 15. 9413-1 | 4 | 2 | 1 |
| 16. 6841-1 | 3 | 8 | 4 |
| 17. 6977-1 | 4 | 4 | 0 |
| 18. 4914-1 | 1 | 5 | 1 |

| | | | |
|---|---|---|---|
| 19. 1623-1 | 0 | 0 | 0 |
| 20. 5521-1 | 4 | 2 | 1 |
| 21. 8175-1 | 1 | 3 | 0 |
| 22. 1623-1 | 2 | 3 | 2 |
| Totals | 52 | 56 | 24 |

Wilson bands by region of canines. (Table 2)

| Tooth Identification | Incisal Region | Mid-Coronal Region | Cervical Region |
|---|---|---|---|
| 1. 7975-2 | 3 | 4 | 4 |
| 2. 8623-2 | 2 | 6 | 11 |
| 3. 8623-2 | 0 | 7 | 9 |
| 4. 6991-2 | 2 | 4 | 2 |
| 5. 0449-2 | 0 | 3 | 1 |
| 6. 2482-2 | 1 | 12 | 13 |
| 7. 3596-2 | 0 | 10 | 14 |
| 8. 2830-2 | 0 | 7 | 5 |
| 9. 5980-2 | 2 | 6 | 2 |
| 10. 5374-2 | 0 | 5 | 5 |
| 11. 7649-2 | 0 | 5 | 7 |
| 12. 8941-2 | 1 | 2 | 1 |
| 13. 0054-2 | 0 | 1 | 3 |
| 14. 5374-2 | 0 | 5 | 5 |
| 15. 8549-2 | 1 | 17 | 16 |
| 16. 5384-2 | 2 | 9 | 6 |

| | | | |
|---|---|---|---|
| 17. 2009-2 | 0 | 5 | 3 |
| 18. 2482-2 | 0 | 6 | 7 |
| 19. 8941-2 | 0 | 2 | 2 |
| 20. 0326A-2 | 0 | 2 | 3 |
| 21. 0835A-2 | 1 | 0 | 3 |
| 22. 5020-2 | 0 | 7 | 8 |
| Totals | | | |

Wilson bands by region of incisors. (Table 3)

Calculations:

$$\frac{\#\ of\ Wilson\ Bands\ per\ region}{Total\ \#\ of\ Wilson\ Bands} \times 100$$

Canines:

$\frac{52}{132} \times 100 =$ **39.4% incisal**

$\frac{56}{132} \times 100 =$ **42.4% mid-coronal**

$\frac{24}{132} \times 100 =$ **18.2% cervical**

Incisors:

$\frac{15}{291} \times 100 =$ **5.15% incisal**

$\frac{135}{291} \times 100 =$ **46.4% mid-coronal**

$\frac{141}{291} \times 100 =$ **48.45% cervical**

**Discussion**

Further discussion of this topic is centered upon clinic applications of this research and my curiosities as a future dentist. Moreover, this research made other considerations apparent, which are listed in my questions below.

- Future research would use electron microscopy to see if enamel prisms are functioning differently than normal enamel prisms.
- Are canines more susceptible to fractures and lesions, or are incisors more susceptible to fractures and lesions?
- How do dentists treat teeth with these defects?
- Will these defects affect the orthodontics of patients?
- Is there a genetic (racial) difference in tooth susceptibility of stress markers?
- Are these defects indicators of future stress (i.e. depression, etc.) in these individuals?
- Is there a relationship between tooth defects and psychosomatics of illness (i.e. depression, PTSD, etc)?

## References

v. Koenigswald W and Sander PM (1997) (Eds.) *Tooth Enamel Microstructure – Proceedings of the Enamel Microstructure Workshop*. A.A. Balkema: Rotterdam

Marks, M. K. (1993). *Dental enamel microdefects as indicators of childhood morbidity among historic African Americans* (Doctoral dissertation, The University of Tennessee).

McCauley LK and Somerman MJ (2012) *Mineralized Tissues in Oral and Craniofacial Science, Biological Principles and Clinical Correlates.* Wiley-Blackwell: Ames, IA

Thompson JL, Krovitz GE and Nelson AJ (2003) *Patterns of Growth and Development in the Genus Homo. Cambridge Studies in Biological and Evolutionary Anthropology 37*. Cambridge University Press: Cambridge