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Haley Davis
haley.davis1@bobcats.gcsu.edu

Alfred J. Mead
Georgia College and State University, al.mead@gcsu.edu

ENAMEL HYPOPLASIA AS AN INDICATOR OF NUTRITIONAL STRESS IN JUVENILE WHITE-TAILED DEER

Haley S. Davis*
Alfred J. Mead
Department of Biological and Environmental Sciences
Georgia College and State University
Milledgeville, GA 31061
* corresponding author
haley.davis1@bobcats.gcsu.edu

ABSTRACT

Cheek teeth of 343 white-tailed deer mandibles collected from the Piedmont National Wildlife Refuge, central Georgia, were examined for the presence of enamel hypoplasia, a permanent enamel defect associated with episodes of severe physiological stress. Hypoplastic defects were observed in 27% of the individuals, with no significant difference between females (26%) and males (27%). Pit hypoplasia occurred most frequently, with most defects located on the hypoconid of the first lower molar. In white-tailed deer, the first lower molars form as fawns transition into functional ruminants and are weaned at the approximate age of 10 weeks. The presence of enamel hypoplasia at this stage in development suggests that this nutritional transition results in severe physiological stress in a large proportion of fawns each year.

Key words: Enamel hypoplasia, white-tailed deer, *Odocoileus virginianus*, nutritional stress

INTRODUCTION

The occurrence of permanent dental anomalies can serve as valuable indicators of specific events in the life history of a mammalian species. One of the primary dental abnormalities frequently studied is enamel hypoplasia, a deficiency of enamel thickness occurring during tooth crown formation. Episodic disruptions of enamel secretion result in observable bands around the circumference of the crown. These anomalous bands are classified as three main types of defects: linear, pit, and swale hypoplasias (1). Linear hypoplasia (Fig. 1A), which is considered to be the most severe form of enamel hypoplasia, materializes as a deep furrow around the tooth crown. Pit hypoplasia (Fig. 1B) is characterized by small pits on the tooth surface, and may be distributed in a wide band around the crown or merge into a linear row. Swale hypoplasia (Fig. 1C), perhaps the hardest form of enamel hypoplasia to detect, manifests as an irregular sloping around the cusp tips of teeth.

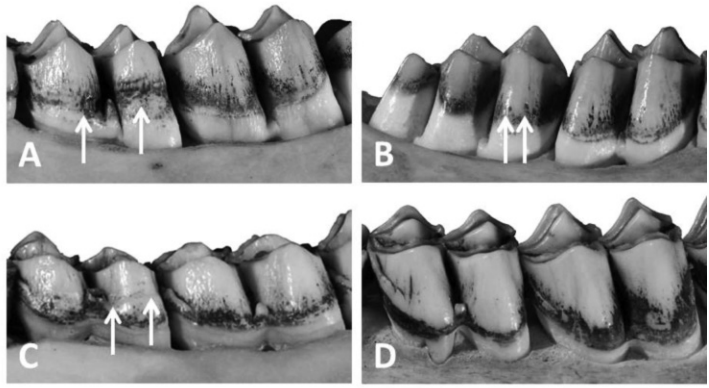

Figure 1. Buccal view of lower molars of white-tailed deer, *Odocoileus virginianus*, collected from the Piedmont National Wildlife Refuge, central Georgia. A, linear hypoplasia (arrows) on left m1, GCM 2971; B, pit hypoplasia (arrows) on right m3, GCM 2972; C, swale hypoplasia (arrows) on left m1, GCM 2973; D, left m1 and m2 lacking hypoplastic defects, GCM 2970.

Mammalian teeth are composed of two main parts, the crown and the root (1). The tooth root is anchored entirely in alveolar bone and is composed of a root canal and pulp cavity enclosed by a layer of dentine and a thin outer layer of cementum. The root canal and pulp cavity allow the passage of the blood vessels and nerves that nourish the tooth. Dentine, produced by odontoblasts, is chiefly composed of calcium phosphate minerals, fibrous proteins, and collagen. Cementum is similar in composition to bone and connects the periodontal ligament to the tooth root. Mammals are unique among vertebrates in that they have two sets of teeth: deciduous and permanent. Deciduous teeth are present in juveniles and replaced by permanent teeth as the animals are weaned and begin relying on firmer sources of food.

Enamel, which is composed primarily of tightly packed hydroxyapatite crystals, surrounds the tooth crown (1). Ameloblasts are responsible for the formation of enamel, depositing it in rings around the tooth crown beginning at the coronal surface and proceeding to the cervical region. Enamel formation occurs in two stages; the secretion stage and the maturation stage. If the ameloblasts are interrupted during either of these stages, enamel will be either anomalously thin or, in some severe cases where the ameloblast cells die, completely absent. The disruption in enamel formation may be caused by a stressor, which can be either environmental or physiological in origin. Due to the ring-like nature of enamel development and the lack of enamel remodeling throughout the life of the organism, the location of enamel hypoplasia bands can indicate the approximate age of an individual at the time the physiological stressor occurred.

The occurrence of enamel hypoplasia on mammalian teeth was first

identified in human populations. Anthropological studies of early humans suggested that the stressors causing enamel hypoplasia may have been associated with the transition from a hunting to a gathering society, the introduction of foreign pathogens, and/or weaning and nutritional stress (2). Enamel hypoplasia also has been documented in a large variety of extant and extinct mammals (3, 4, 5, 6, 7). These studies have suggested a variety of stressors, including weaning, disease, and environmental change. Mead (3) found that birth stress and the stress of cow-calf separation were likely causes of enamel hypoplasia in the Miocene rhinoceros *Teleoceras*. Birth-weaning stress events and singular environmental stressors, such as drought, were suggested as causes of enamel hypoplasia in Pleistocene *Equus* species (4). Cornay and Mead (5) suggested weaning as the hypoplasia-causing stressor in Virginia opossums, *Didelphis virginiana*, from Georgia. Fluoride toxicosis has been identified as a hypoplasia-inducing stressor in wild ungulates, including white-tailed deer, *Odocoileus virginianus*, in the northwestern United States (6).

The white-tailed deer is an abundant mammalian species ranging from southern Canada throughout most of the conterminous United States, and southward into northern South America (8). These ruminating ungulates typically weigh between 50-135 kg and live in areas that provide both woodland for cover and open areas for foraging. Survival is influenced by many factors, especially hunting pressure. The lifespan in the wild for females (does) may approach 6 years while males (bucks) seldom survive past 3.5 years. Does are normally sexually mature at 1.5 years, but depending on various ecological factors, may breed around 6 months. Does reach maximum body mass at 3-4 years. Captive bucks are capable of breeding at 6 months, but normally sexual maturity occurs at 1.5 years. Bucks reach maximum body mass at 4-5 years.

White-tailed deer breed from late October to early November, and parturition takes place in late May-early June (8). Twins are typical, although yearling does may produce only one fawn. Does have been known to have triplets if nutrients are plentiful and stress factors are low. Lactation is abbreviated in white-tailed deer. Neonates nurse 2-3 times per day for the first 2 weeks following birth, with the doe leaving the fawns in cover and returning frequently throughout the day. At 3 weeks, fawns begin grazing and moving with the mother. Fawns are functional ruminants by 8 weeks and are foraging as a family group by 10 weeks, when weaning normally occurs.

The objectives of this study were to identify and classify the types and locations of enamel hypoplasia in hunter harvested white-tailed deer from central Georgia, determine the age at which most hypoplasias form in this sample, and propose possible causative stressors.

MATERIALS AND METHODS

As part of a larger morphological study (9, 10), one side of the lower mandibles from 343 white-tailed deer (103 females, 240 males) were obtained at a hunter-check station at the Piedmont National Wildlife Refuge in Jones and Jasper Counties in central Georgia. The age and gender were recorded

for each deer from which the mandibles were extracted. Using a dissecting microscope, teeth were analyzed for the presence of enamel hypoplasia. Due to the absence of a large number of incisors, only the pre-molars and molars (cheek teeth) were examined for enamel hypoplasia. All skeletal material is housed in the Georgia College Mammal Collection (GCM).

RESULTS

Of the 343 mandibles analyzed, 93 (27.1%) displayed evidence of enamel hypoplasia (Table I). The frequency of occurrence was the same for females (27/103, 26.2%) and males (66/240, 27.5%). Many individuals showed hypoplasia on multiple teeth, resulting in 139 observed defects. Pit hypoplasia was the most observed form, accounting for 61.2% (85/139) of the defects. Swale hypoplasia was second most common (49/139, 35.3%), and linear hypoplasia was least common (5/139, 3.6%). Most of the recorded defects were located on the buccal (74.8%) rather than the lingual (25.2%) sides of the teeth. The majority of hypoplasias were observed on the hypoconids (77/139, 55.4%). Individually, hypoplasia occurred most frequently on first lower molars (m1, 58/93, 62.4%), followed by the fourth lower deciduous premolars (dp4, 18/93, 19.4%) and the second lower molars (m2, 14/93, 15.1%).

Table I. Occurrence of enamel hypoplasia in white-tailed deer lower cheek teeth (343 lower mandibles) collected from the Piedmont National Wildlife Refuge, central Georgia. Percentage of hypoplasia on teeth is based on 93 individuals. Percentage of hypoplasia on cusps is based on 139 observed defects. P = permanent tooth, D = deciduous tooth.

		Number of Occurrences	Percentage
Tooth			
	First molar (P)	58	62.4
	Fourth premolar (D)	18	19.4
	Second molar (P)	14	15.1
	Second premolar (D)	6	6.5
	Third molar (P)	5	5.4
	Third premolar (D)	5	5.4
Cusp			
	Hypoconid	77	55.4
	Protoconid	26	18.7
	Metaconid	18	12.9
	Entoconid	17	12.2

DISCUSSION

Random stressors such as disease, attempted predation, injury and drought may occur at any time during an animal's life. The high proportion of hypoplastic defects observed on m1's suggests the occurrence of a non-random, defect-inducing stressor during the juvenile development of these organisms. Possible non-random stressors include disruptions in nutrient availability associated with birth, becoming a functional ruminant, weaning, first winter food shortage, or first year dispersal. Because age at each tooth eruption is well constrained in white-tailed deer (8, 11, 12), it is possible to link these physiological stressors to certain developmental milestones in the lifespan of these organisms. The dp4 erupts during week 1 and is fully erupted at week 10, m1 erupts at week 10 and is fully erupted at week 36, and m2 erupts at week 24 and is fully erupted at week 52. Teeth, such as m1, begin development as much as 12 weeks prior to eruption (Figure 2A).

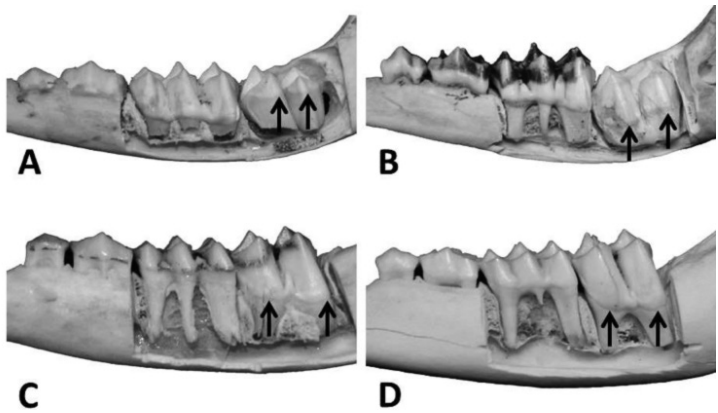

Figure 2. Buccal view, dissected mandible exposing lower molars of white-tailed deer, *Odocoileus virginianus*, collected from Georgia. A, 3-4 weeks in age, enamel fully formed on dp4, enamel maturation front near coronal end on m1 indicated by arrows, GCM 2241. B, 6-7 weeks in age, roots partially formed on dp4, enamel maturation front closer to cervical region on m1 indicated by arrows, GCM 2969. C, 12-14 weeks in age, roots fully formed on dp4, enamel fully formed on m1, cervical region indicated by arrows, GCM 2968. D, 20 weeks in age, roots nearly fully formed on m1, cervical region indicated by arrows, GCM 2967.

Approximately 27% of the white-tailed deer examined in this study displayed enamel hypoplasia. These defects were most often pits (61%) and occurred most frequently on the hypoconid (55%) of m1 (62%). As is evident in Figure 2, white-tailed deer teeth begin erupting prior to the completion of the crown. The stage of crown formation between weeks 6-7 (Fig. 2B) and weeks 12-14 (Fig. 2C) indicates that the m1 hypoplasias occurred around

weeks 9 to 10. In terms of fawn development (8, 11), neonates nurse for the first 2 weeks, begin grazing at 3 weeks, and by 8 weeks should be functional ruminants. At 10 weeks the fawns are weaned and begin moving with the family group.

If the fawn is not yet a functional ruminant at the time of weaning, it is likely that nutritional stress would be experienced. It appears that a delay in attaining functional ruminant abilities or the onset of weaning is the most likely cause of the observed m1 hypoplasia. The hypoplasia present on dp4 would form at birth or during the first week. The hypoplasia on m2 may be associated with events occurring after weaning and prior to the second summer. As seen in other extinct and extant mammals, the analysis of the occurrence of enamel hypoplasia allows for the identification of periods of nutritional stress in the modern white-tailed deer.

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