Prevalence and racial distribution of primary canine hypoplasia of the maxillary canine

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Introduction

Primary canine hypoplasia (PCH) is an enamel defect located on the mesial aspect of the facial surface at approximately the junction of the gingival and the middle thirds of the tooth.[1-5] The defect exists in two forms: minimal hypoplasia consisting of brown, white, or yellow opaque areas with a smooth surface; and obvious hypoplasia having a combination of pitting, invagination, missing enamel, or a rough-textured surface.[5] In obvious hypoplasia, opaque areas may be present and there will be at least one other abnormality.

Duncan et al.[4] examined the mandibular primary canines of 334 African-American 3- to 5-year-old children, and found a prevalence of 37.1% for African-American males and females, with no apparent differences between fluoridated and nonfluoridated communities. Twenty-six percent of the defects had an associated carious lesion. Silberman et al.[3] compared the data reported by Duncan et al. with that of 37 Caucasian children examined at two Head Start centers during the same study. The prevalence noted for the Caucasian children was 11%. Silberman et al.[5] examined 2686 randomly selected Mississippi school children, African-American and Caucasian, 4 and 8 years old, and found the prevalence rate of PCH on mandibular canines to be 33.2% for African-American children and 17.2% for Caucasian children. African-Americans had a significantly ($P = 0.0001$) higher prevalence than Caucasians, and males a significantly ($P = 0.01$) higher prevalence than females. A significant ($P = 0.001$) relationship also was demonstrated between PCH and dental caries. Water fluoridation status, age, geographic location (urban or rural), and breast-feeding history were not related to PCH.

Very little has been written about PCH in the maxillary canine. Brown and Smith[6] examined all four primary canines of 112 children ages 3–10 years seen on recall visits and concluded that the mandibular teeth were affected twice as often as the maxillary. They also noted very little difference in prevalence between the maxillary canine and the left mandibular canine, but the prevalence of the right mandibular canine was remarkably higher. They did not address racial differences. A recent study[7] of 113 school children of Harappa, Punjab Province, Pakistan, noted a prevalence of PCH of 34.5% with no significant association between the defect and gender, socioeconomic status, or side of the jaw. The defect occurred significantly more frequently in the mandible than in the maxilla. The purpose of this report is to provide prevalence data for PCH affecting the maxillary primary canines in different age, gender, and race cohorts.

Methods and materials

The study population included 4- and 8-year-old (African-American and Caucasian) children in private and public preschools and elementary schools in Mississippi, selected randomly from a list of schools agreeing to participate. The total sample available for this study was 2563 children. The contingency tables were analyzed using the Grizzle, Starmer, and Koch methodology[8] and the CATMOD procedure on Statistical Analysis System (SAS).

Three investigators, standardized (kappa score = 0.67) in the use of the simplified hypoplasia index,[9] examined children under a high-intensity portable dental light with a mirror and explorer. The simplified hypoplasia index was used to overcome potential problems with more complicated indices, to simplify data collection, and to reduce procedural errors.[9] The labial surfaces of the 12 anterior teeth were examined and findings entered on a dental survey form.

Results

Prevalence of primary maxillary canine hypoplasia

Children analyzed to determine the prevalence of PCH included all African-American and Caucasian children with at least one maxillary canine present. Subjects with one or more maxillary canines diagnosed with PCH or related lesion (caries or restoration in the PCH) are described by age, race, and gender in the Table. African-American children in each age category had a significantly greater prevalence of PCH (10.4% in 4-year-olds; 11.1% in 8-year-olds) than Caucasian children (5.0% in 4-year-olds; 7.4% in 8-year-olds). Combining age groups resulted in a total prevalence of 10.6% in African-American children and 6.5% in Caucasian children.

Table. Distribution of subjects with at least one maxillary canine by race, age, and sex

		Age 4			Age 8			Total		
Race	*Gender*	*N*	*PCH*	*%*	*N*	*PCH*	*%*	*N*	*PCH*	*%*
White	Male	231	13	5.6	388	36	9.3	619	49	7.9
	Female	231	10	4.3	381	21	5.5	612	31	5.1
	Total	462	23	5.0	769	57	7.4	1231	80	6.5
Black	Male	451	48	10.6	194	25	12.9	645	73	11.3
	Female	437	44	10.1	250	24	9.6	687	68	9.9
	Total	888	92	10.4	444	49	11.1	1332	141	10.6
Both Races	Male	682	61	8.9	582	61	10.5	1264	122	9.7
	Female	668	54	8.1	631	45	7.1	1299	99	7.6
	Total	1350	115	8.5	1231	106	8.7	2563	221	8.6

Association of PCH with age, gender, and race

Univariate chi-square analysis determined a highly significant association between PCH and race ($P < 0.001$), and an association not significant between PCH and gender ($P = 0.067$) or between PCH and age ($P = 0.843$). The Grizzle, Starmet, and Koch methodology in the CATMOD procedure of SAS was performed. The first model fit to the data included the three main efforts (age, race, gender), and all first- and second-order interactions. Since none of the interactions were statistically significant at the 0.05 level, a second model was fit omitting the interactions. These results indicate statistical significance for race ($P = 0.0001$); (df = 1); ($\chi^2 = 15.47$). Gender was not significant ($P = 0.0503$); (df = 1); ($\chi^2 = 3.83$) nor was age ($P = 0.1567$); (df = 1); ($\chi^2 = 2.01$). Although this more complete analysis takes each variable into account given the presence of the other two, the results are consistent with those from the univariate analysis.

Discussion

The prevalence of PCH in maxillary canines is 6.5% for Caucasian children and 10.6% for African-American children compared with the prevalence of PCH in mandibular canine of 17% for Caucasian and 33% for African-American children.[5] The prevalence of PCH in maxillary canines is approximately 32–37% that of mandibular canines. Studies by Badger[11] and Needleman et al.[12] found an almost equal distribution of the defect between the maxillary and mandibular canines. Brown and Smith[6] however, found the prevalence of PCH to be about 2:1 in favor of the mandibular teeth, and found a higher prevalence for the mandibular defect than we found. Our study indicates a considerably higher difference between the maxillary and mandibular arches, in that mandibular PCH was about three times as prevalent as maxillary PCH.

This study was consistent with the previous study of mandibular canines,[5] in that African-American children were more likely to have maxillary PCH than Caucasian children. The ratio of mandibular PCH in African-Americans vs. Caucasians, previously was found to be almost 2:1 (33–17%). In this study, the ratio of maxillary PCH in African-Americans vs. Caucasians again approached 2:1 (10.6–6.5%). The ratio of maxillary to mandibular PCH of approximately 1:3 held true for both races. This study also concurs with the previous study in that the association between age and PCH was not significant.

The relationship between PCH and gender has been found to be significant in previous studies with males having a significantly higher prevalence of PCH. This study found that males had a higher prevalence in both ages and races, but this difference was not significant.

Little is known about the etiology of PCH. Etiologic factors that have been proposed include pressure related to breast-feeding or other trauma, nutrition, the position of the developing tooth in its crypt, and the thickness of the cortical plate over the facial surface.

The current theory[10] assumes that the labial cortical bone over the developing primary canine thins to the point where fenestration occurs at or shortly after birth, exposing the developing crown to physical trauma. Two factors — prematurity and maternal diet — have been proposed as factors causing cortical plate thinning. These factors are not mutually exclusive and are both related to infant hypocalcemia. Racial predictions may relate to nutritional deficiencies associated with low socioeconomic status. Another explanation could be a racial difference in terms of the position of the developing tooth bud. If the canine develops in a more labial position with thinner buccal plates in certain racial groups, a greater likelihood of fenestration may exist, putting the tooth at risk for PCH.

The difference in prevalence between maxillary and mandibular teeth also is difficult to explain. Perhaps the mandibular teeth are positioned more labially or have a thinner buccal plate more likely to fenestrate. Perhaps the prevalence of fenestration is the same but the mandibular canine is exposed to more trauma than the maxillary canine. Silberman et al.[5] examined the relationship between mandibular PCH and breast-feeding and did not find a significant relationship. Trauma could be related to the eruptive process itself or per-

haps to a sleeping position of the infant, where there is pressure on the facial surface of the mandibular teeth, but the maxillary teeth are protected by the zygoma.

The relationship between maxillary primary canine hypoplasia and dental caries was not specifically examined because a significant relationship between PCH and dental caries had been established.[5] The authors did not analyze the data for differences in prevalence between right and left primary canines, but they suggest it may prove interesting.

Conclusions

1. Primary canine hypoplasia occurs to a much lesser extent in the maxillary canine than in the mandibular canine (approximately one-third as often).
2. The lesion affects the maxillary primary canine of African-American children twice as frequently as Caucasian children.

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