Patterns of Hypodontia among Third Molars in Contemporary American Adolescents

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ABSTRACT: Third molars (M3s) are congenitally absent (hypodontic) more frequently than any other tooth type. Causes of this enhanced variability are poorly understood, but the potential range of absence—from none through four M3s per person—provides the opportunity to examine the permutations of missing M3s within and among ethnic groups. Teenage samples of two overlapping populations (1,100 American whites; 600 American blacks) were studied here, with radiographic confirmation of each tooth's presence in the jaws. Roughly 15% of these people are missing at least one M3, but only about 2% of this sample is hypodontic for all four molars. The frequency and severity of missing M3s are significantly higher in whites than blacks. Within individuals, correspondence of occurrence is much higher within than between the jaws, but all combinations of M3 hypodontia are positive and significant statistically—implying common underlying developmental influences. While various sorts of data support a genetic influence on the risk of M3 hypodontia, patterns of inheritance suggest a multifactorial rather than a single-gene mode of inheritance. Several researchers have promoted a polygenic threshold model, and the history and application of this model are discussed. *Dental Anthropology* 2009;22(1):8-17.

Hypodontia—the congenital absence of a tooth—is not uncommon in contemporary human populations. Evidence suggests that the risk and pattern of missing teeth are under some genetic control, and it is evident that frequencies differ between sexes and among races. By far, the tooth type most likely to be congenitally missing in contemporary humans is the third molar (M3). Nanda (1954), Eidelman *et al*. (1973), Thompson *et al*. (1974), Mattheeuws *et al*. (2004) and Polder *et al*. (2004), among others, have reviewed M3 frequencies in contemporary human populations.

Various speculative ideas have been put forth to explain how a tooth can be congenitally absent and, in particular, why M3s commonly are missing (see, *e.g*., Pindborg, 1970). These mechanistic ideas predate a modern understanding of molecular signaling in tooth development (*e.g*., Matalova *et al*., 2008), but a short review is informative. As one influential example, Ashley Montagu (1940) conjectured that tooth agenesis resulted from inadequate space in the developing maxillary dental arch. Montagu was focusing specifically on the maxillary lateral incisor that forms on the lateral border of the premaxilla next to the maxillary-premaxillary suture (Behrents and Harris, 1991). Ashley Montagu's contention—which was well reasoned but unsupported by any test—was that tooth size responds to the available space of the supporting bone. Ashley Montagu speculated that, across eons—as what is now the orthognathic human face diminished in size from prognathic predecessors—tooth sizes (and, especially, size of the maxillary lateral incisors)

diminished coincident with increases in pegging and congenital absence of various tooth types. As regards the maxillary lateral incisor that is quite variable (at least in European peoples; Harris and Rathbun, 1991), Ashley Montagu concluded that this dental variability is due to the phylogenetic reduction of the premaxilla.

Ashley Montagu sidesteps the question why the canine, the other tooth adjacent to the maxillary-premaxillary suture, is, in contrast, one of the most stable tooth types. He also avoids the problem (except in his introduction) of why the mandibular incisors are not comparably variable, though sizes of the two jaws have necessarily been reduced to similar extents. Ashley Montagu's scenario—that reduced bony support leads to reduced tooth sizes—also seems at odds with the third molar located at the distal terminus of the arches also being quite variable even though these molars occur at the other end of the dental ach and form much later than the incisors (Haavikko, 1970). It seems that different agents are responsible within each tooth type.

Sofaer (*e.g*., 1969, 1979) seems to promote this same idea of inadequate formative space as a general explanation for hypodontia, though this is unsubstantiated by our current understanding of tooth morphogenesis. This conjecture also ignores the three-dimensional dispersions of the developing tooth

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germs. While it is a graphic metaphor to suppose that a formative tooth bud might be "choked" out of existence due to inadequate bony support, there are no data to support this. Instead, cytokines from the dental follicle attract osteoclasts during normal development (Marks and Cahill, 1983), and these clast cells progressively enlarge the surrounding tooth crypt to accommodate the developing tooth (Carlson, 1944). This is readily seen (and palpatable) in infants, where the buccolingual diameters of the primary tooth crypts have enlarged well beyond the incipient bony ridges, and the surfaces of the ridges are scalloped due to these out-pouchings (*e.g.*, van der Linden and Duterloo 1976). The emergence of teeth into a tight-fitting arcade of teeth as seen in the adult is not indicative of the three-dimensional arrangement of tooth crypts—plus the temporal span during which different teeth form. For example, the canine abuts against the lateral incisor in the adult, but (A) the lateral incisor forms much earlier, when there is plenty of room in the supporting jaws, and (B) when the canines do form, their positions are far apical of the other teeth.

Molecular biology now makes it clear that a tooth will fail to develop if there is no ectodermal signal to stimulate a site along the underlying mesenchyme to initiate tooth formation (Kollar and Baird, 1970a,b). This cause of hypodontia seems primarily genetic in nature, but failure of formation also can be affected by the environment. Suggestions from animal studies are that tooth buds that fail to reach a critical size will resorb—resulting in hypodontia rather than continuing to develop. Likewise, environmental stressors acting at the critical early stages of formation can simply kill off a tooth bud. Teratogenic drug actions and irradiation are well-studied examples of this (Bruce, 1950; Kaste *et al.*, 1998). Yet a third mechanism involves a genetic interruption of the cascade of molecular signals leading to tooth formation. This is obvious in the edentates (*e.g.*, armadillos, anteaters; Todd, 1918) where there is initial tooth formation, but development ceases early in the bell stage. This interruption also accounts for the "missing teeth" (absence of lateral incisors, canines, and premolars) that is characteristic of mice and other rodents. (See review by Peterkova *et al.* 2006.) The extreme example of this inhibition of tooth development probably is in birds (the class Aves), where all modern birds are tooth-less but tooth formation can be reintroduced experimentally (Chen *et al.*, 2000; Mitsiadis *et al.*, 2006). At an allelic level, it is conceivable that this sort of interruption of molecular events accounts for the variable frequencies of tooth agenesis in humans (*e.g.*, Matalova *et al.*, 2008).

Numerous clinical and physical anthropological studies have reported on the frequencies of missing M3s in humans. The purpose of the present study is to investigate the pattern of missing M3s in some detail within and among individuals in population samples. That is, there are 4 M3s distributed as left-right pairs in the two dental arches, and the issue is how hypodontia is distributed among these 4 sites. This study is restricted to M3s, though there are evident associations among tooth types (Davies, 1968; Khalaf *et al.*, 2005; Harris and Clark, 2008). As pertinent examples, Alvesalo and Portin (1969) and Woolf (1971), among others, have documented that the maxillary lateral incisor is more often affected (diminished size, pegged, absent) in individuals with hypodontic M3s versus those with developmentally intact dentitions; hypodontia is not an isolated phenomenon, even among tooth types that form at quite different ages.

MATERIALS AND METHODS

Panoramic radiographs (van der Linden and Duterloo, 1976) of 1,700 adolescents were studied. Most (1,100) were American whites, and the rest were American blacks (600), all from clinical records at the College of Dentistry, University of Tennessee, Memphis. Subjects were selected with radiographs taken between 12 and 18 years of age. These adolescents were old enough that their M3s would have begun mineralization if they were going to form (Rantanen, 1967; Harris, 2007), but the adolescents were young enough to well remember having any M3s extracted. It seems obvious that hypodontia has to be documented radiographically, especially for M3s that commonly form but do not erupt into the oral cavity. Sample sizes vary among the statistical tests described here because not every tooth's existence could be documented because of radiographic issues.

One intent was to estimate the background frequencies of M3 hypodontia in these two ethnic groups, so subjects with a recognized craniofacial syndrome, including facial clefts, were omitted since they have characteristic—often elevated—patterns of hypodontia (*e.g.*, Schalk-van der Weide, 1992; Ranta, 1983; Harris and Hullings, 1990).

Tooth formation can be viewed as a dichotomous event—a tooth has either developed or it is absent. With potentially one M3 in each quadrant, there are 16 permutations of hypodontia. Expansion of the binomial shows that there are five M3 groupings, namely (A) all 4 M3s present, (B) four arrangements with 1 tooth missing, (C) 6 arrangements with just 2 teeth missing, (D) 4 arrangements of 3 teeth missing, and (E) one situation where all 4 M3s are hypodontic. In other words, the 16 permutations are arranged in the familiar ratios of 1:4:6:4:1.

Statistical tests relied on chi-square analysis. Statistics were performed using JMP 7.0 (SAS, Cary, NC). The kappa statistic was calculated as the measure of association (Fisher and van Belle, 1993).

RESULTS

The observed frequency of M3 hypodontia for the total sample (Table 1) shows that the distribution is far from random. Despite common perceptions that hypodontia of M3 is common, most people experience development of all 4 M3s (86.8%; 1449/1670), whereas congenital absence of all 4 M3s occurred in just 1.6% of the cases. Fig. 1 shows that the distribution of severity (*i.e.*, number of congenitally absent M3s) approximates the right-end of a normal distribution, where the frequency decreases as the number of missing M3s increases. The perception that M3s frequently are absent is strongly influenced by the widespread prophylactic extraction of M3s in the late teens (*e.g.*, Eklund and Pittman, 2001).

Black-White differences

American blacks and whites have been admixing for centuries, though admixture estimates are lower in the Southeast than elsewhere in the nation because of harsher social and legal proscriptions (Williamson, 1980; Davis, 1991). Blacks have larger and morphologically more complex teeth (Richardson and Malhotra, 1975; Irish, 1997), and, evidently in an associated manner, discernibly lower frequencies of hypodontia (Harris and Clark, 2008). Stanley Garn contended in several of his publications (notably 1977) that tooth size, morphology, tempo of formation, and occurrence (in contrast to congenital absence) are positively intercorrelated features of a common underlying theme in tooth formation, not isolated phenomena—and that these features differ among tooth types controlled, at a primary level, by a tooth's position in its morphogenetic field.

Table 2 shows the distributions in each of the arches (sexes pooled). In both jaws, whites have highly significantly higher frequencies of M3 hypodontia, and the source of the significance is primarily due to deficits of bilateral absence in blacks compared to whites (as assessed from the cell chi squares).

Little is known about hypodontia in other, non-Caucasian races; most work has been done on peoples of European extraction where frequencies and the patterning of hypodontia among tooth types probably is not representative of all groups. Röse (1906) and Hrdlička (1921) each collated data from large series of peoples of diverse races—but with ill-defined criteria and without the benefit of radiography to confirm congenital absence. Still, differences in the frequencies of hypodontia are evident in these early studies. Population differences in trait frequencies are prima facie evidence for a genetic influence on the risk of hypodontia.

Sexual dimorphism

The data in Table 1 were dichotomized into cases

TABLE 1. Distributions of M3 congenital absence by race and sex

Number Missing	Whites						Blacks						Total Sample	
	Males		Females		M+F		Males		Females		M+F			
	n	%	n	%	n	%	n	%	n	%	n	%	n	%
0	396	86.09	504	80.77	900	83.03	254	95.13	294	92.45	548	93.68	1448	86.76
1	25	5.43	49	7.85	74	6.83	8	3.00	11	3.46	19	3.25	93	5.57
2	29	6.30	41	6.57	70	6.46	3	1.12	10	3.14	13	2.22	83	4.97
3	5	1.09	12	1.92	17	1.57	1	0.37	0	0.00	1	0.17	18	1.08
4	5	1.09	18	2.88	23	2.12	1	0.37	3	0.94	4	0.68	27	1.62
Sums	460		624		1,084		267		318		585		1,669	

TABLE 2. Frequencies of M3 hypodontia in American Blacks and Whites

	Whites			Blacks				
Statistic	Both Absent	One Absent	Both Present	Both Absent	One Absent	Both Present	df	Chi-square[1]
Maxilla								
%	5.0	4.0	90.9	1.7	3.2	95.1		
n	56	45	1010	10	19	561	2	12.6
Mandible								
%	4.5	3.1	92.5	1.2	1.5	97.3		
n	74	51	1534	7	9	572	2	17.8

[1]Both X^2 values are highly significant (P < 0.0001) because M3 hypodontia is more common in whites than blacks.

without M3 hypodontia and cases missing one or more M3s. This showed that hypodontia is significantly more common in girls than boys in whites (χ^2 = 5.3; df =1; P = 0.02). The source of significance (based on cell chi squares) is primarily due to the comparative deficit of hypodontia in males. 14% of males exhibit agenesis of one or more M3s, compared to 19% of females. The overall frequency is appreciably lower in American blacks (*ca.* 6% *vs.* about 16% in whites) and, with the smaller sample size of 600, the sex difference is not significant here (χ^2 = 1.8; df = 1; P = 0.1850). If the present frequencies hold, a sample size roughly three times larger (*ca.* 2,000) would be needed to achieve statistical significance in blacks.

In addition to the greater frequencies of M3 hypodontia in females, Fig. 2 shows that severity—as measured by the number of missing M3s—also is greater in females than males. This shift towards greater expression in females is more obvious in whites because of their greater incidence of M3 hypodontia overall.

Arcade effects

There are positive, statistically significant associations for M3 hypodontia between all four M3s taken pairwise; the matrix of kappa correlations (Table 3) based on the total sample shows that left-right symmetry is highest (kappa ~ 0.7) within each arch, and the inter-arch associations are appreciably lower (kappa ~ 0.3), but correlations within and between hemispheres seem equivalent. Hierarchically, the symmetry between sides is much higher than between arches, but whether the association between the arches is taken between the same or opposite quadrants seems immaterial.

A related point is that asymmetric occurrence is relatively uncommon. M3 status in one quadrant strongly predicts the same status in the antimeric site. This is anticipated since our understanding is that the same genotype affects tooth development in the left and right quadrants, with effectively the same environment in each to achieve a tooth's phenotype. Dental researchers have sought evidence for laterality or sidedness, primarily using data on crown dimensions. Documentations of laterality are few and scattered among samples (*e.g.*, Harris, 1992; Townsend *et al.*, 1999). The bulk of left-right asymmetry is expressed as random (fluctuating) asymmetry, at least with regard to size.

Figure 3 arborizes the frequencies of hypodontia by tooth type and, thereby, shows the dependencies (statistical associations) between the arches. An obvious "dose-dependent" relationship from among several of the associations is this: When both upper molars are congenitally absent, just 50% of the two mandibular molar molars are present. When just one upper molar is present, the frequency of the two lower molars being
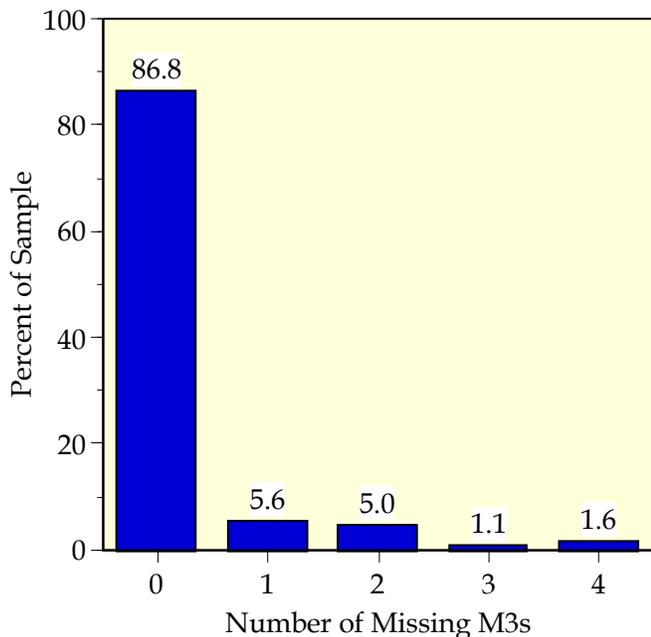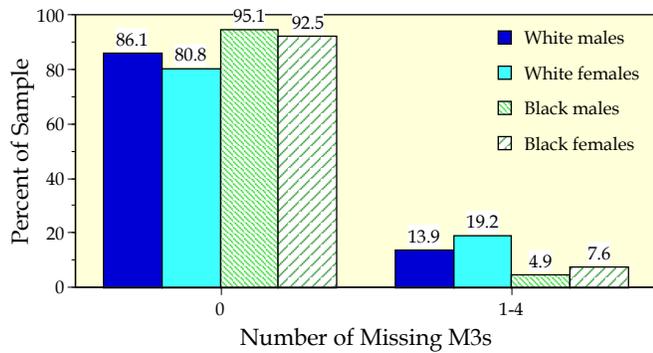

Fig. 1. Percentage distribution of M3 hypodontia in the total sample.

Fig. 2. The frequencies of the congenital absence of M3 (all expressivities combined) by race and sex. M3 hypodontia is more common in American whites than blacks, and more frequent in girls than boys in each race, though the extent of sexual dimorphism is appreciably higher in whites, perhaps because the overall incidence is higher in whites.

absent rises to 70%. And, when neither upper M3 is absent, the frequency of both lower molars being agenetic rises to a high of 94%.

Side effects

Sidedness is the interesting situation where there is preferential laterality: Does absence of a tooth on one side influence absence of the same tooth in the opposing arch? The informative cases are those where either the left or right molar is absent in the maxilla and likewise (unilateral absence) in the mandible. Unfortunately, cases of unilateral congenital absence in both dental arches are uncommon, just 7 cases in the 1,670 individuals where all 4 M3s could be scored. These 7 cases were equally distributed (3:4) as to arrangements where the ipsilateral tooth (same hemisphere) was missing in the two arches versus where the contralateral tooth (opposite hemisphere) was absent. At least with these few informative cases, there is no suggestion of sidedness.

Another way of viewing laterality is simply whether M3 is more common on one side of the mouth than the other. The maxillary left-right distribution of unilateral presence is 33 (left only) compared to 32 (right only), which is indistinguishable statistically from a random spread of 50:50. In the mandible, the left-right distribution of congenital absence is 27 (left only) compared to 34 (right only). This does not depart from a 50:50 chance occurrence (P = 0.53). Congenital absence of M3 is, then, equally distributed between sides.

DISCUSSION

Hypodontia in itself suggests a phenotypic dichotomy: the tooth either is present or absent. Features

of hypodontia, notably the increased frequencies among relatives of affected individuals (Grahnén, 1956; Brook, 1984), imply a hereditary basis for the condition, though the mode of transmission is not simple (Mendelian). Differences in population frequencies among inbred strains of laboratory animals (*e.g.*, Grüneberg, 1952; Chai and Chiang, 1962; Sofaer, 1969) and among human groups (*e.g.*, Ashley Montagu, 1940; Polder *et al.*, 2002; Harris and Clark, 2008) likewise favors some genetic basis for hypodontia. Sex differences in rates of occurrence (typically with the frequency and severity of agenesis being greater in females) is a third indicator that genes influence a person's risk (Egermark-Eriksson and Lind, 1971). The dramatic effects of some major genes, notably the suite of genes causing forms of HED (hypohidrotic ectodermal dysplasia), might also be mentioned here, but these phenotypes are characterized by oligodontia or, even, anodontia, so they do not stem from the same alleles leading to the absence of a single or just a very few teeth as occurs in most people with hypodontia (Schalk-van der Weide, 1992).

Elucidation over the past few years of specific molecular signaling factors that predispose for hypodontia, such as Pax 9, Msx 1, Msx 2, and others, greatly strengthens the argument for a genetic basis of congenital absence (*e.g.*, Mostowska *et al.*, 2003; Viera, 2004; Larmour *et al.*, 2005). These few first molecular factors to be identified are, predictably, those with clear-cut effects on the phenotypes—where affected individuals commonly are missing multiple teeth. Analytical refinements (and larger sets of family data) will lead to documentation of genes with subtler but probably more common frequencies in the general population. Work to date shows that deleterious alleles (Pax 9 and so forth) enhance the risk of hypodontia, but they do not fully determine it, and the variable expressivity among cases likely is due to (A) the individual's genetic background against which these alleles are expressed and (B) environmental conditions that modulate expression.

Quasicontinuous model

Hypodontia as expressed in most humans (with one or a few missing dental elements) has no known etiology. It is, however, common enough to warrant the attention of many dental researchers. A popular model of inheritance that accounts for the observed phenotypic distributions of the condition is quasicontinuous inheritance. The supposition is that some indefinite number of genes collectively contribute to trait expression (where "expression" here is congenital absence). This is the common polygenic model (*e.g.*, Falconer, 1989), but with a threshold (Fig. 4). The threshold is toward the lower end of the supposed underlying genotypic array. For the bulk of the population (that is above the threshold) teeth are present. It is in those comparatively few cases who

TABLE 3. Correlation coefficients (kappa) between the four third molars taken pairwise[1]

	Upper Left	Upper Right	Lower Left
Upper Right	0.66 (0.0400)		
Lower Left	0.31 (0.0437)	0.33 (0.0439)	
Lower Right	0.30 (0.0442)	0.31 (0.0444)	0.71 (0.0356)

[1]Values in parentheses are the standards errors of the estimates; all 6 correlations are highly significantly different from zero (P < 0.0001).
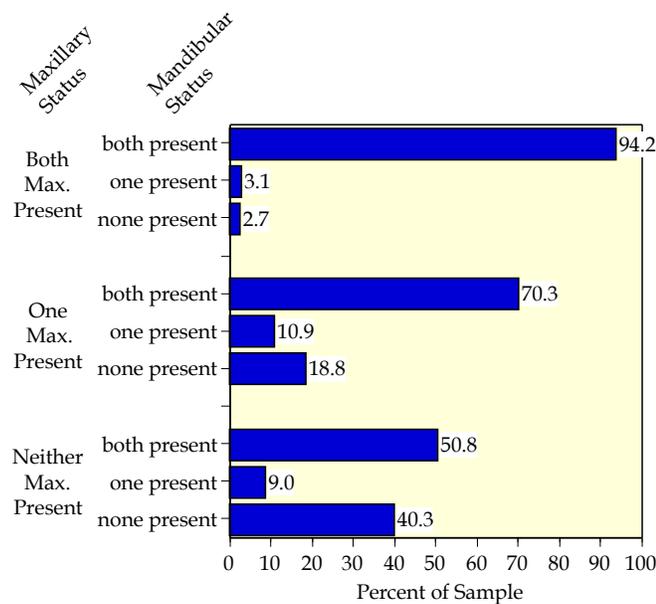
Fig. 3. Dependence between arches in the occurrence of M3s. (*Top*) When both maxillary M3s are present, 94% of cases have both mandibular M3s present. (*Middle*) when just one of the two maxillary molars is present, the frequency of both lower M3s being present drops to 70%. (*Bottom*) When both maxillary M3s are congenitally absent, the frequency of both lower M3s being present is just 40%. These associations indicate nothing about cause and effect; the same dependencies are found if the lower molars are taken as the predictive variable.

are below the threshold that hypodontia occurs.

There is an interesting but tangled history of this model. Many physical anthropologists, particularly those with interests in skeletal biology, attribute it to the work of Hans Grüneberg (1950, 1952) who marshaled the quasicontinuous model (QCM) as an explanation for the numerous minor skeletal variants he studied in mice, such as accessory foramina, ossicles, and other morphological features, that occur in some animals but not others. The utility of these "discrete" (*i.e.*, present or absent) skeletal features for phenetic studies of human skeletal series was popularized by A. C. Berry and R. J. Berry (*e.g.*, 1967, 1968, 1974).

Grüneberg's work in turn rested on the seminal studies of Sewall Wright in the 1930s. Wright (1934a,b) explored the inheritance of the number of digits on the hind feet of guinea pigs, which normally have 3 digits but may have 4, and attributed the occurrence of 4 toes to the guinea pig's genotype exceeding what he termed a "physiological threshold." Indeed, his Figure 1 (1934b, p. 544) depicts the presumed underlying polygenic model as a normal curve overlying two successive thresholds, a lower one, where poorly-formed ("vestigial") 4th toes occur, and a higher one, where the 4th toe is eumorphic. This development of a two-threshold scheme is precisely what was exploited later by Reich and others (Reich *et al.*, 1972; Corbett *et al.*, 2004) to provide practical statistical tests for distinguishing between single-gene and polygenic models of inheritance. While Wright did not formalize the QCM, he described its major features during his various breeding experiments. Denys Falconer (1965) elaborated the assumptions and statistical expectations of the QCM. Falconer described how heritability ($V_{additive}$ / V_{total}) of a trait could be estimated from trait frequencies. However, this requires family data (information on relatives of known degrees of biological relatedness). Heritability cannot be calculated from samples of cases without known relationships, so this useful aspect of the QCM generally has been ignored in skeletal biological studies, but with some noteworthy exceptions: Saunders and Popovich (1978) recorded minor skeletal variants from radiographs of siblings enrolled in the Burlington Growth Study. Sjøvold (1984, 1996) analyzed skulls of Europeans where genealogical information had been preserved. Cheverud and colleagues (*e.g.*, McGrath *et al.*, 1984; Richtsmeier *et al.*, 1984) used the unique setting of the island of Cayo Santiago (where genealogical affinities of most monkeys is known) to estimate heritability of several nonmetric bony features in macaques.

The work of Carter (notably 1969) warrants mention here because (A) he demonstrated the applicability of a threshold model for many common diseases (*e.g.*, pyloric stenosis, diabetes mellitus, spina bifida cystica, and others), which did much to familiarize the health care community with this quasicontinuous model and

(B) he listed several criteria that, when met, can be very suggestive of a polygenic threshold model.

While largely beyond the scope of this paper, it is informative to note that James (1971) pointed out that too many parameters need to be estimated than can be obtained from a QCM with one threshold. But, adequate parameters are available if two thresholds are supposed in the model, and James worked with Ted Reich (*e.g.*, Reich *et al.* 1972; Corbett *et al.* 2004) to develop tests that can distinguish between inheritance due to a single-gene model versus a polygenic model. Suarez and Spence (1974) applied a basic form of this approach to the hypodontia family data collected by Grahnén (1956), concluding that a polygenic threshold model fit the data appreciably better than expectations of the effects of single gene.
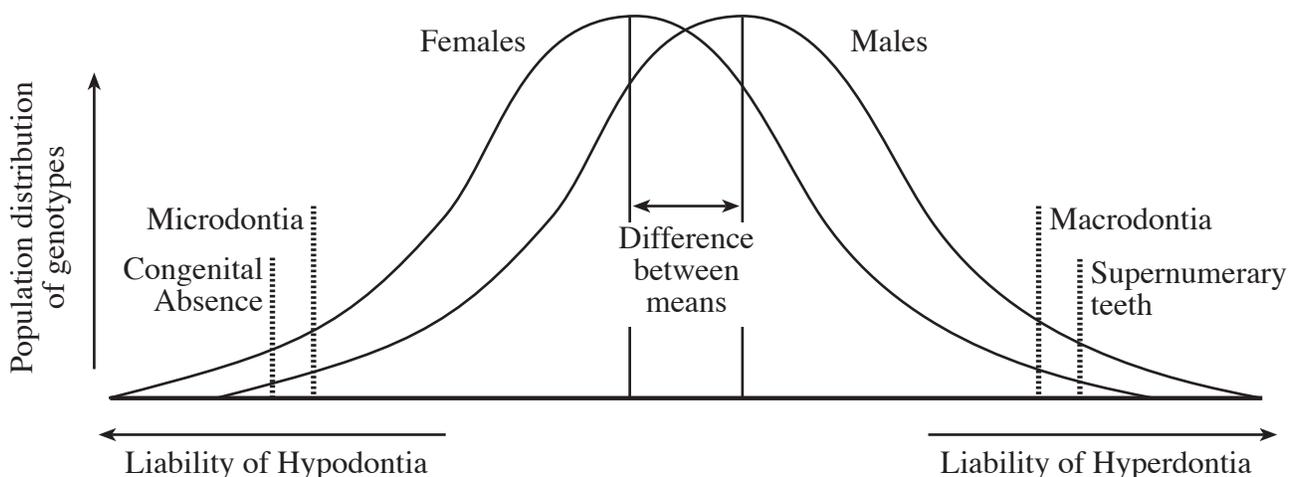
QCM and Hypodontia

Davies (1968), Sofaer (1969), Bailit (1975), and Chosack *et al.* (1975), among others, alluded to the QCM fitting observations seen in population samples, but Brook (1984) was the first to seriously develop the QCM to hypodontia (and, at the other, complementary extreme, hyperdontia). Brook emphasized the developmental interrelationships between hypodontia and tooth size. There also is a well-documented relationship between hypodontia and crown sizes of the remaining teeth; people in the population who do not have hypodontia have statistically larger teeth than those with congenital absence (Garn and Lewis, 1962; Garn *et al.*, 1962, 1963, 1970). Conversely, diminished crown sizes and microdontia are more common in those with hypodontia than in those with full complements of teeth. These clinical results are duplicated in laboratory animals (Grüneberg, 1950, 1952; Self and Leamy, 1978). The greater the extent of hypodontia, the greater the size reductions and the greater likelihood of microdontia (with associated missing cusps and simplified morphologies of the remaining teeth). Numerous studies of European groups have found higher frequencies of hypodontia in females than males (reviewed in Egermark-Eriksson and Lind, 1971). These several associations suggest that hypodontia has dentition-wide systemic effects, which is predictable since teeth form as repetitive elements (a meristic series; Bateson, 1894) using the same regulatory mechanisms controlled by the person's genotype (Kettunen and Thesleff, 1998; Jernvall and Thesleff, 2000).

Grüneburg (1952) documented differences in the frequencies of third molar hypodontia among inbred strains of mice. Mice with larger teeth had lower frequencies of M3 hypodontia than strains with smaller teeth. The same relationship is evident in humans, where African Americans (with large teeth) exhibit M3 hypodontia infrequently compared to American whites with smaller crown sizes and higher frequencies (and greater severities) of M3 hypodontia (Harris and Clark, 2008). Hyperdontia (supernumerary teeth) is, in contrast, more common in males (*e.g.*, Stafne, 1932; Khalaf *et al.*, 2004).

This collage of interrelated features recently has been extended by Uslenghi *et al.* (2006) who showed that hypodontia is associated with slowed tooth development (also see Garn *et al.* 1961).

Fig. 4. Schematic of the quasicontinuous model (modified from Brook, 1984). There is an underlying genotypic range in a population that influences a person's risk for hypodontia (left extreme) and hyperdontia (right extreme). Sex-specific distributions are shown here to reflect the greater risk of congenital absence in women versus the greater risk of supernumerary teeth in men—at least in peoples of European extraction. Sexual dimorphism appears to be lower in peoples of subSaharan extraction. The vertical bars are depicted as broken lines since a person's genotype can be modulated in either direction by the environment.

OVERVIEW

The present study assessed the phenotypic patterns of third molars (M3) congenital absence in 1,700 teenagers composing a contemporary cohort of American blacks and whites from the Southeast United States.

- There is no difference by arcade, but agenic M3s are significantly more common in females than males and in American whites compared to American blacks.
- No evidence of sidedness (preferential absence on one side) could be discerned, and asymmetry (unilateral occurrence) is fairly uncommon versus symmetric presence or absence.
- Congenital absence of one M3 is highly predictive of other missing M3s, suggesting common developmental associations that probably are modulated by the person's genetic background.
- While genes with rather severe effects on congenital absence have been documented, most cases of hypodontia are of unknown etiology, although population distributions are in concert with a quasicontinuous model of inheritance (also termed a polygenic threshold model).

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