Genetic influence on dental arch form in orthodontic patients

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The size and form of the dental arches exhibit considerable variability within and among human groups.[1-5] Form ranges from arches that are relatively short and square to others that are relatively long and tapered. Determinants of arch size and shape are not well understood. Soon after palatogenesis, the palate, ringed by the dental lamina, is roughly circular in occlusal view.[6-8] Arch form subsequently elongates anteroposteriorly, and Burdi and Lillie[7] suggested that, "Since the cartilage of the nasal capsule is the predominant tissue in the area at the time when the dental arch elongates to conform to the catenary, it appears that chondral growth is specifically responsible for arch elongation." While some authors have suggested that the shape of human arches approximates a catenary curve,[3,9-11] other researchers have fit other geometric forms, such as an ellipse, a trifocal ellipse, conic sections, a parabola, and polynomial equations.[12-17] Of note, there has been no critical evaluation of the goodness-of-fit of these competing models.

Once arch form is defined in the fetus, variability in eruptive paths of the teeth, growth of the supporting bones,[18-20] and movement of the teeth after emergence due to habits and unbalanced muscular pressures[21-25] all contribute to variation in arch size and shape.

The purpose of the present study was to inves-

Abstract

Human arch form varies considerably. This study analyzed the size and shape of the maxillary and mandibular dental arches of 320 adolescents from 155 sibships. A broad battery of measurements (k = 48) was computer-generated from Cartesian coordinates of cusp tips and line angles of the permanent teeth, and heritability estimates were generated from intraclass correlations, controlling for sex and age where indicated. Arch size has a modest genetic component, on the order of 50%, although this estimate may contain shared environmental influences. Tooth rotations have low h^2 estimates, most of them indistinguishable from zero. Arch shape, assessed as length-width ratios, also has a modest transmissible component, suggesting that arch length and width growth factors are largely independent. Highest heritability estimates, as a group, were for transverse arch widths, which averaged about 60%. Several measures of left-right asymmetry also were analyzed (k = 31), and, while the arches are systematically asymmetric (generally with left > right), there is only weak evidence of a transmissible component for directional asymmetry and essentially none for fluctuating asymmetry. In all, arch size and shape are seen to be more subject to environmental influences than to heredity. These findings direct attention toward the need to better understand what extrinsic factors modulate arch size and shape during development.

Key Words

Arch form • Palate • Jaw form • Family analysis • Heritability • Craniometry

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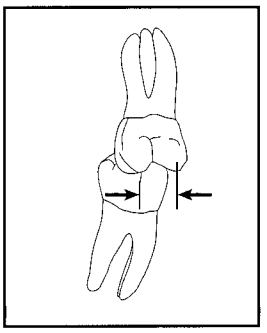

Figure 1
Schematic showing how the buccal segment relationship was measured. The value was negative when the buccal groove of the mandibular first molar was distal of the mesiobuccal cusp of the maxillary molar (i.e., a Class II molar relationship).

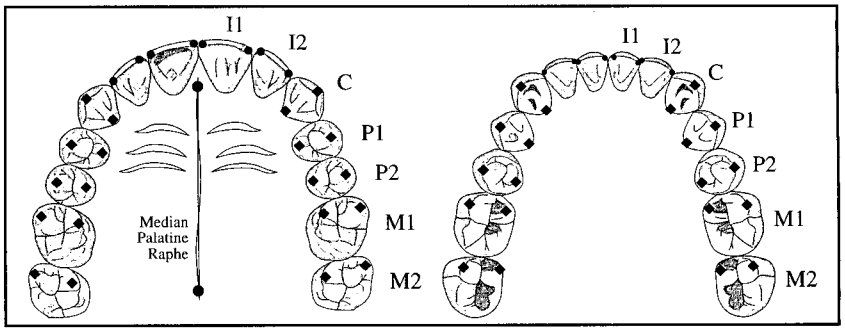

Figure 2
Schematics of the maxillary and mandibular dental arches showing locations of the two landmarks on each tooth, either cusp tips or line angles. A mesial and distal point was located on the midpalatal raphe.

tigate the role heredity plays in determining arch size and shape. The sample consisted entirely of boys and girls who subsequently received comprehensive orthodontic care. This omitted the segment of the population with ideal occlusions, but, since they are the minority[26,27] and are seldom encountered by the specialist, there is less interest in development of their arch forms. Moreover, since it appears that malocclusion, defined as tooth displacement and malrelationships, is an acquired rather than an inherited condition,[28,29] there should not be any striking difference in heritability estimates. On the other hand, exclusion of sibs not requiring treatment could inflate estimates of genetic influence since it may decrease within-sibship variance.

The full-sib genetic model was used,[30] and we began with the observation that the phenotypic variation can be partitioned into genetic and environmental components, $V_P = V_G + V_E$, where "V" is the statistical variance. It would be desirable if the "genetic" component were due just to the additive effects of the indefinitely large number of genes that collectively affect arch size (and other skeletodental dimensions[31-38]). The genetic component actually may be confounded by two potentially relevant sources, one genetic and one environmental. V_G can be rewritten as $V_G = V_A + V_D + V_{EC}$, where A is the additive genetic effect, D is due to dominance of some alleles over others, and EC is the common environment of siblings, which enhances their phenotypic similarity beyond that due to the fact that full sibs (and other first-degree relatives) share an average of 50% of their genes in common by descent. It can be shown that the correlation for a trait between full sibs is due to

$$\frac{1}{2}V_A + \frac{1}{4}V_D + V_{EC}$$

(This equation ignores epistatic and genotype-environment interactions.) This confounding of V_A with V_D and V_{EC} is not dissimilar to that encountered when using monozygotic and dizygotic twins, although the assumptions of the twin model seldom are made explicit in the clinical literature.

Shared environmental effects may be significant, but rather little is known about what specifically in the environment influences dentoskeletal growth. There are obvious examples, such as trauma, infectious disease, starvation, chronic mouthbreathing, and digit habits, but, even collectively, these do not account for much of the observed frequency of malocclusion.[23,26,39] Shared environmental effects, such as location, diet, morbidity patterns, home lifestyle, and having the same parents, will, to a greater or lesser extent, cause the growth and development of siblings to converge. This confounding is why the term "transmissibility" generally is preferable to heritability, since it takes note of the nongenetic, cultural sources of sibling similarities.[40]

Materials and methods

The sample consisted of 155 sibships, 145 of them with two members and 10 with three. All individuals were American white adolescents. All had intact permanent dentitions, although the last teeth (excluding third molars, which were not measured), were not completely erupted in a few of the youngest individuals, accounting for the variable sample sizes in the tables. Sibships were collected from several private orthodontic practices. The pretreatment orthodontic dental casts were analyzed; none had received prior treatment. All children subsequently received full-banded treatment, but no child with a craniofacial anomaly, genetic disease, or premature loss of deciduous teeth was used. It was taken at face value that the siblings were first-degree relatives as avowed by the parents. No serological or other test was used to confirm consanguinity. Pretreatment record ages ranged from 10.0 to 19.0 years with a mean of 13.5 years (SD = 1.69 yrs).

Data collection

Buccal segment relationship and incisor overjet were measured with the teeth held in maximum intercuspation according to the methods of Lundström[41] (Figure 1).

Two landmarks were located on each of the 14 teeth in each arch (Figure 2). These were the cusp tip of the protocone and paracone of the molars, the buccal and lingual cusps of the premolars, the protocone and tuberculum dentale of the canine, and the mesial and distal line angles of the incisors. On the maxillary casts the axis of the midpalatal raphe was defined by a line connecting a point marked on the raphe anterior of the incisive foramen and a second marked posterior on the raphe at the depth of the second molars (Figure 2). Marking the dental landmarks before digitizing or photocopying was advocated by Takada et al.[42] and Ho and Kerr[43] as a method of increasing accuracy.

Each cast was photocopied at 1X by placing the occlusal surface face down on a Kodak 2110 photocopier in its photographic mode. A cardboard mask covered with millimetric graph paper was used to cover the active surface of the photocopier. A small rectangle was cut into the mask to provide space for maxillary and mandibular casts. Tests were made to assess the accuracy of the method. When cusp tips were positioned in the occlusal plane, there was no measurable distortion. When teeth were apical to the occlusal plane, as in cases with a deep curve of Spee, there was some reduction in size, such that at 10 mm (which exceeded any curve of Spee in this study) reduction was 1%

Landmarks on the photocopies were digitized as Cartesian coordinates, and linear and angular measurements were computer-generated using custom software. The arch size variables can be categorized into five groupings: (1) tooth angulations, (2) arch widths, (3) arch depths, (4) arch chords, and (5) arch interrelationships. The angle formed by each maxillary tooth to the midpalatal raphe was calculated (Figure 3A). A similar, albeit manual, approach was used by Lamons and Holmes.[44] Arch widths in the upper arch were measured orthogonal to the raphe, and the left and right measurements were summed for each tooth type (Figure 3B). In the mandible, arch width was calculated as the straight-line distance between homologous cusp tips. Arch depths (Figure 3C) were measured parallel with the midpalatal raphe; five depths were defined to quantify various mesial and distal segments of the arch plus the whole arch. A different procedure, attributable to DeKock,[45]

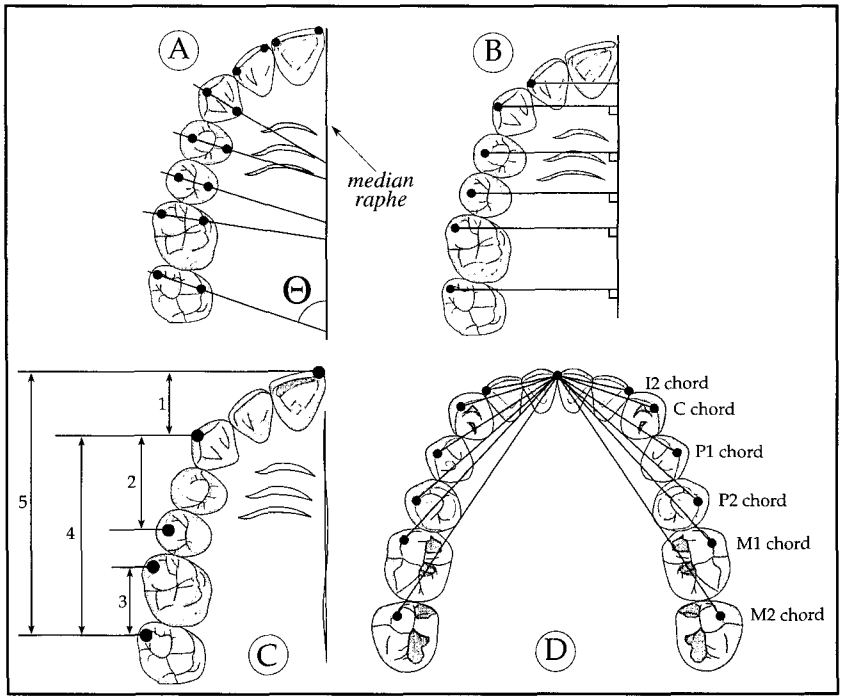

Figure 3A-D

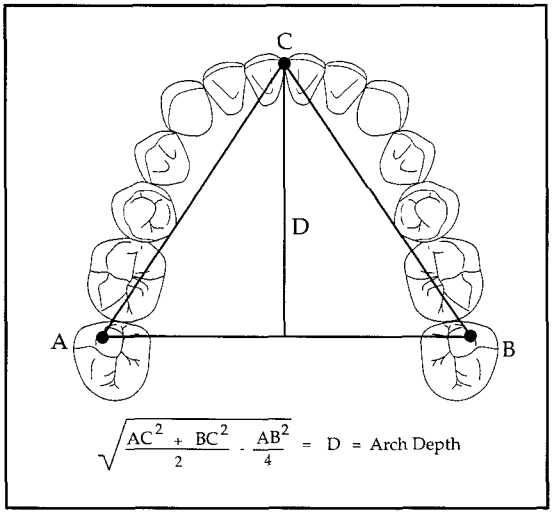

Figure 4

Figure 3A-D
Diagram showing the kinds of linear and angular measurements assessed from each set of dental study models.
A: Distolateral angle at the intersection of lines formed by a tooth's landmarks and the midpalatal raphe was measured for each tooth (except the incisors, where the mesiobuccal angles were calculated).
B: Maxillary arch widths were measured from each tooth (excluding central incisors) orthogonal to the midpalatal raphe.
C: Five maxillary depths were calculated parallel with the raphe: 1. incisor depth, 2. premolar depth, 3. molar depth, 4. buccal depth, and 5. arch depth.
D: Mandibular arch widths were the straight-line distances between homologous buccal cusp tips.

Figure 4
Sketch of a mandibular dental arch illustrating arch chord distances. In this instance, lines AC and BC are the left and right chords for the second molars. Analogous arch chords were calculated for the other five tooth types. Arch depth (D), shown here for the second molars, was calculated from the formula below the figure.

Table 1
Descriptive statistics and ANCOVA tests for sexual dimorphism and influence of age on trait size†

Variable	Males $\bar{x}$	Males SD	Females $\bar{x}$	Females SD	Sex F-ratio	Age F-ratio	h^2	Standard confidence limits Error	L_1	L_2
Tooth interrelationships (mm)										
Buccal segment relation	-1.1	2.18	-0.9	1.99	0.4	2.5	0.56*	0.12	0.32	0.79
Incisor overjet	5.1	2.53	4.4	2.17	7.7*	4.9	0.23	0.14	-0.04	0.50
Maxillary angular relationships (degrees)										
I1 angle	106.6	9.13	105.6	8.88	1.1	1.2	0.33*	0.13	0.07	0.59
I2 angle	129.2	10.72	131.2	11.06	2.4	0.3	0.15	0.14	-0.13	0.43
C angle	46.3	7.35	47.7	6.57	2.6	0.0	0.11	0.17	-0.22	0.44
P1 angle	80.2	8.05	79.7	7.25	0.3	1.6	0.39*	0.13	0.14	0.64
P2 angle	74.9	8.61	74.2	9.41	0.6	1.9	0.19	0.14	-0.09	0.46
M1 angle	58.4	6.67	57.0	6.68	3.4	0.2	0.51*	0.12	0.28	0.75
M2 angle	60.1	6.88	59.0	7.52	2.1	1.6	-0.04	0.16	-0.34	0.27
Transverse maxillary arch widths (mm)										
I2 distance	12.8	1.15	12.4	1.01	11.0*	0.2	0.62*	0.12	0.39	0.85
C distance	16.9	1.18	16.0	1.19	36.5*	1.3	0.56*	0.12	0.32	0.79
P1 distance	20.0	1.53	19.1	1.29	31.3*	0.2	0.69*	0.11	0.46	0.91
P2 distance	22.7	1.72	21.7	1.45	31.7*	0.0	0.67*	0.11	0.44	0.89
M1 distance	25.3	1.63	24.3	1.46	33.1*	0.3	0.67*	0.11	0.45	0.90
M2 distance	28.0	1.59	27.0	1.45	34.4*	3.8	0.76*	0.11	0.54	0.97
Maxillary arch chords (mm)										
I2 chord	13.8	0.89	13.1	0.93	36.8*	0.4	0.32*	0.13	0.05	0.58
C chord	18.5	1.14	17.5	1.20	50.4*	0.9	0.32*	0.13	0.06	0.59
P1 chord	25.7	1.96	24.4	1.60	40.5*	0.1	0.49*	0.12	0.24	0.74
P2 chord	32.1	2.08	30.6	1.82	47.0*	0.4	0.48*	0.12	0.23	0.72
M1 chord	38.5	2.19	36.8	2.00	52.3*	0.0	0.44*	0.13	0.19	0.69
M2 chord	49.3	2.43	47.2	2.19	61.9*	0.0	0.46*	0.13	0.21	0.70
Maxillary arch depths (mm)										
Incisor depth	9.1	2.00	8.7	1.80	3.7	9.5*	0.33*	0.13	0.06	0.59
Premolar depth	14.5	1.35	13.8	1.22	26.7*	5.6	0.43*	0.13	0.18	0.68
Molar depth	11.4	0.83	11.0	0.77	22.5*	0.6	0.47*	0.12	0.23	0.72
Buccal depth	32.1	1.80	30.6	1.74	50.4*	1.7	0.43*	0.13	0.18	0.68
Arch depth	41.1	2.72	39.3	2.56	38.1*	1.4	0.37*	0.13	0.11	0.62
Maxillary arch form										
Upper width ratio	0.60	0.04	0.59	0.04	3.4	0.1	0.40*	0.13	0.15	0.65
Upper depth ratio	0.22	0.04	0.22	0.04	0.0	12.1*	0.29*	0.14	0.03	0.56
Upper shape ratio	0.68	0.06	0.69	0.06	1.1	5.3	0.46*	0.13	0.22	0.71
Mandibular arch chords (mm)										
I2 chord	13.8	1.02	13.1	1.08	36.8*	0.1	0.31*	0.14	0.04	0.57
C chord	18.5	1.26	17.5	1.38	44.7*	0.6	0.26	0.14	-0.01	0.53
P1 chord	25.9	2.16	24.5	1.87	36.3*	0.3	0.48*	0.13	0.23	0.73
P2 chord	32.5	2.23	30.9	2.12	37.9*	0.1	0.44*	0.13	0.19	0.69
M1 chord	38.5	2.32	37.1	2.20	46.0*	0.3	0.38*	0.13	0.12	0.63
M2 chord	49.5	2.51	47.5	2.39	54.4*	0.4	0.43*	0.13	0.17	0.68
Transverse mandibular arch widths (mm)										
I2 arch width	19.2	1.55	18.7	1.41	8.7*	3.3	0.34*	0.13	0.08	0.61
C arch width	25.4	1.95	24.5	1.83	20.7*	1.9	0.48*	0.12	0.23	0.72
P1 arch width	33.0	2.67	31.6	2.30	25.7*	0.2	0.53*	0.12	0.29	0.77
P2 arch width	38.6	2.79	37.0	2.59	29.7*	1.4	0.47*	0.13	0.22	0.72
M1 arch width	43.8	2.63	42.3	2.51	26.5*	0.3	0.61*	0.12	0.38	0.85
M2 arch width	49.3	2.88	47.7	2.48	29.3*	3.9	0.43*	0.13	0.19	0.68
Mandibular arch depths (mm)										
P1 arch depth	10.4	2.01	9.8	1.73	8.4*	0.0	0.52*	0.12	0.28	0.76
P2 arch depth	17.5	1.86	16.6	1.76	18.3*	1.5	0.57*	0.12	0.34	0.81
M1 arch depth	24.0	2.04	22.9	1.85	23.2*	0.5	0.51*	0.12	0.27	0.75
M2 arch depth	35.3	2.34	33.7	2.08	39.2*	0.5	0.43*	0.13	0.18	0.68
Mandibular arch form										
Lower width ratio	0.5	0.04	0.51	0.04	0.4	9.0*	0.43*	0.13	0.18	0.68
Lower depth ratio	0.3	0.04	0.29	0.04	0.8	0.2	0.49*	0.12	0.25	0.73
Lower shape index	1.4	0.12	1.41	0.11	1.6	0.8	0.28*	0.13	0.02	0.55

†All measurements except overjet are averages of left and right sides obtained by computing the statistics on the left-right average of each case.
Tooth codes are: incisors (I), canines (C), premolars (P), and molars (M); teeth within a morphogenetic field are numbered mesially to distally.
*$p < 0.01$

was used for mandibular arch depths. First, the left and right arch chords were calculated from the mesial line angle of the central incisor in a quadrant to each tooth except the central incisor. This was done for the left and right quadrants, then the arch widths and chords were used to calculate the arch depths (Figure 4). Arch chords also were calculated for maxillary teeth (Figure 3D).

Total measurement error determined by double determinations[46] was 4%. This was the cumulative random error of cusp tip identification, photocopying, and digitization of the landmarks. This compares favorably with published determinations from other methods.

Asymmetry

Most variables were calculated separately for left and right sides, which permitted assessments of bilateral differences, both directional asymmetry (DA) and fluctuating asymmetry (FA).[47,48]

Directional asymmetry occurs when one structure preferentially outgrows the homologous structure on the contralateral side. Schultz[49] documented numerous directional asymmetries induced by preferential use of the right arm and leg in higher primates, including humans, where the right limb bones typically are longer and more robust than their antimeres.[50,51] Most individuals are right-handed,[52-54] and preferential side use during growth results in limb bone dimensions that are somewhat larger on the right. The presence of directional asymmetry was tested using a two-tailed, one-sample *t*-test of whether d = 0, where *d* is the average left-minus-right difference in the sample, and *n* is the number of individuals:

$$DA = \bar{x}_d = \frac{\Sigma(L-R)}{n}$$

Fluctuating asymmetry is assumed to result from inability of the organism to grow identical, bilaterally homologous structures. FA is present in virtually all dentoskeletal dimensions in all individuals providing the measurements are of sufficient resolution. Fluctuating asymmetry is without side-preference, however, so the distribution ought to be normal and the mean side difference, $d = \Sigma$ (L-R), will not depart significantly from zero. The measure of fluctuating asymmetry used here is:

$$FA = \Sigma \frac{|(L-R) - \bar{x}_d|}{(\frac{L+R}{2})}$$

which is essentially that used by Otremski et al.[55] The effect of directional asymmetry in this equation was removed from each subject's left-right difference. The absolute value was obtained since it is the amount of variability free from random left- and right-side differences that is desired. The denominator scales the asymmetry to mean size of the variable so comparisons can be made among measurements.[56] FA, by this formula, is the proportion of the dimension that is due to left-right asymmetry, and this can be described as a percentage of trait size.

Statistical design

The presence of statistically significant sex and/or age effects in each variable was tested with a general linear model.[57] Two runs were performed.[58] The first tested whether any variable exhibited a significant sex-by-age interaction effect. Since none did, the second run assumed a common slopes model.

Heritability estimates were obtained by fitting mixed model ANOVAs with a random effect of sibship, and sex and age were included as cofactors where indicated. The intraclass correlation (r_i) then was estimated for each trait as the ratio of among-sibship variance to the total variance, where total variance is the sum of the within- and among-sibship variances.[59] Heritability (h^2) is twice the intraclass correlation in the full-sib model.[30] The formula for the standard error of h^2 was from Swiger et al.[60] All inferential statistics were evaluated as two-tailed tests.

Results

Sex and age effects

Variables measured on the left and right sides were averaged for analysis in this section for brevity. There were 47 resulting variables; they are dealt with here in groups to avoid redundancy (Table 1).

Arch size, demarcated by the permanent dentition, was significantly larger in males than in females. Indeed, 32 of the 33 linear arch size variables exhibited a significant sex difference, which is consistent with prior studies.[61-64] Dimensions were 3% to 5% larger in males, with a consistent degree of sexual dimorphism among arch width, depth, and chord measurements.

Only three dimensions changed systematically with age. In each case (i.e., maxillary incisor depth, upper depth ratio, and lower width ratio) the "age" effect was caused by children with more tapered arch forms and more protrusive incisor segments presenting at chronologically earlier ages. These "age" effects probably are unique to orthodontic samples where those children with obvious malocclusions tend to be brought to the specialist at younger ages. There were no statistically significant age effects for the

other variables in Table 1. This may be due in part to the cross-sectional nature of the data. Longitudinal studies[65-70] have reported slight increases in arch width and decreases in arch depth during adolescence, but the changes are too subtle to be detected without serial data.

Asymmetry

Most (22/31) measures of asymmetry exhibited statistically significant directionality (Table 2). All of the left-minus-right means were positive (except for some lower arch chords), showing that the left side of the arch is slightly but systematically larger than the right. This trend included buccal segment relationship (BSR), where these children were significantly more Class II on the right side than the left (perhaps because the left side of the mandible was slightly longer than the right). Several tooth angulations also showed significant directional asymmetry, with the buccal aspects of the left teeth being rotated more to the distal than their right counterparts, a difference of about 4°, on average. While the mean left-right differences may seem trivial, several individuals possessed marked asymmetries.

Heritability estimates

Data from the full-sibling analysis are listed in Table 1. Buccal segment relationship—the sagittal relationship of the maxilla and mandible at the first molar—had a significant h^2 estimate of 56%, implying that about half the total variation in BSR is due to the genetic influence of siblings sharing half their genes in common by descent from their parents. Put a different way, it means that adolescent siblings tend to be much more alike as regards BSR than biologically unrelated adolescents. In contrast, there was no detectable familial component for overjet.

In fact, though most of the variables (42/47) had a measurable genetic component ($\alpha = 0.05$; Table 1), the level of influence varied among the types of arch variables. The highest h^2 estimates were for arch widths; the mean h^2 estimate of the collection of 12 maxillary and mandibular widths was 57%, which is significantly higher than for the other 28 variables ($Z = 3.7$; $p < 0.001$). This leaves little doubt that arch size is under appreciable genetic control—which is in concert with prior analysis of arch size[38,71] and with the other craniofacial complexes.[29,35-37,72]

Results for the tooth angulation variables were different (Table 1). Just three of the seven achieved statistical significance, and ranks for h^2 of these seven variables were significantly lower than for the other arch dimensions ($Z = 2.9$; $p = 0.004$).

Asymmetry

The sibling analysis produced little evidence for genetic control of asymmetry (Table 2). Positive findings would mean that siblings share the same direction and/or magnitude of left-right asymmetry. In fact, though, just three of the 31 variables had an h^2 estimate significantly above zero, and these three were small and scattered across the dataset rather than clustering within a type of measurement, which would imply a meaningful pattern.

There was even less evidence that the magnitude of asymmetry (i.e., fluctuating asymmetry) is clustered familially. Just 2 of the 31 h^2 estimates were statistically significant, which is about what one would expect due to Type II errors with this many univariate tests.[73] The largest h^2 estimate either for DA or FA was 0.43 for fluctuating asymmetry of M2 buccal chord distance. This implies that the degree of asymmetry (but not the direction) is significantly more similar within sibships than among them. Again, though, the paucity of significant results makes this isolated finding suspect.

Discussion

Size and form of the dental arches generally are defined by positions of the teeth,[13,17,74,75] but one certainly can have a measurable arch even in the absence of some or all of the teeth, whether the teeth are congenitally absent or lost postnatally.[15] It seems that there are combined roles of the teeth themselves and the supporting bone in determining arch size and shape. The purpose of the present analysis was to assess the relative importance of genetics in defining the dental arches in adolescents who have achieved virtually all of their adult arch size.[62,65,68,76,77] Forty-seven variables were computer-generated from each adolescent's dental study casts, but several of these were redundant because each of the seven tooth-types typically was tested within each measurement category. The battery of measurements can be reduced to five categories: (1) tooth interrelationships, (2) tooth angulations, (3) arch widths, (4) arch depths and chords, and (5) size ratios.

Of these five categories, all but tooth interrelationships contained enough variables to warrant tests among them. Tested with Kruskal-Wallis one-way analysis of variance,[78] there was a significant difference ($p < 0.001$), with the descending strengths of the h^2 estimates being arch widths > (depths and chords and ratios) > tooth angulations. The relatively high values for arch widths agree with results by Harris and Smith,[79]

Table 2
Descriptive statistics and heritability estimates for directional and fluctuating asymmetries of dental arch form variables†

		Directional asymmetry							Fluctuating asymmetry					
Variable	n	$\bar{x}$	SD	t-test	h^2	SE	L_1	L_2	$\bar{x}$	SD	h^2	SE	L_1	L_2
Buccal seg.	320	0.32	1.70	3.34*	-0.24	0.17	-0.58	0.10	1.25	1.16	0.00	0.15	-0.31	0.29
Maxillary angular relationships														
I1 angle	319	3.47	9.48	6.53*	0.08	0.15	-0.21	0.37	0.07	0.06	-0.35	0.19	-0.71	0.02
I2 angle	318	4.69	14.48	5.77*	0.08	0.15	-0.21	0.37	0.08	0.08	-0.12	0.16	-0.44	0.20
C angle	288	2.85	10.44	4.63*	0.11	0.17	-0.22	0.43	0.18	0.15	0.18	0.16	-0.14	0.50
P1 angle	319	4.72	10.37	8.13*	-0.22	0.17	-0.56	0.12	0.10	0.09	-0.14	0.17	-0.46	0.19
P2 angle	320	3.32	12.14	4.89*	0.08	0.15	-0.21	0.37	0.12	0.12	0.29*	0.13	0.03	0.55
M1 angle	320	0.46	8.67	0.95	0.32	0.13	0.06	0.58	0.12	0.10	0.14	0.14	-0.14	0.42
M2 angle	319	-0.51	9.58	0.96	0.10	0.15	-0.18	0.39	0.12	0.10	0.09	0.15	-0.20	0.38
Transverse maxillary arch widths														
I2 distance	319	0.12	1.21	1.73	0.22	0.14	-0.05	0.49	0.08	0.06	0.00	0.15	-0.30	0.30
C distance	317	0.13	1.23	1.91	-0.03	0.16	-0.34	0.28	0.06	0.05	-0.01	0.16	-0.32	0.29
P1 distance	319	0.40	1.33	5.33*	0.08	0.15	-0.22	0.37	0.05	0.04	-0.10	0.16	-0.42	0.22
P2 distance	320	0.65	1.47	7.90*	0.04	0.15	-0.25	0.34	0.05	0.04	0.04	0.15	-0.25	0.34
M1 distance	320	0.95	1.58	10.71*	-0.12	0.16	-0.44	0.20	0.05	0.04	0.15	0.14	-0.13	0.43
M2 distance	320	0.92	1.74	9.51*	-0.23	0.17	-0.57	0.11	0.05	0.04	-0.26	0.18	-0.61	0.08
Maxillary arch depths														
Incisor	317	0.34	1.67	3.66*	0.18	0.14	-0.10	0.46	0.16	0.15	0.07	0.15	-0.23	0.36
Premolar	318	0.17	1.32	2.51*	0.27	0.14	0.00	0.54	0.07	0.07	-0.01	0.16	-0.32	0.29
Molar	320	0.15	0.86	3.03*	0.28	0.13	0.00	0.55	0.06	0.05	0.11	0.15	-0.17	0.40
Buccal	318	-0.13	1.53	1.45	0.33*	0.13	0.07	0.59	0.04	0.04	0.12	0.15	-0.17	0.40
Arch	319	0.21	1.94	1.95	0.18	0.14	-0.10	0.46	0.04	0.03	0.12	0.15	-0.17	0.40
Maxillary arch chords														
I2 chord	316	0.07	0.84	1.47	-0.14	0.17	-0.47	0.19	0.05	0.04	-0.04	0.16	-0.35	0.27
C chord	315	0.07	1.20	0.98	0.01	0.16	-0.29	0.32	0.05	0.05	0.28	0.14	0.00	0.56
P1 chord	316	0.47	1.73	4.84*	0.13	0.15	-0.16	0.42	0.05	0.05	0.07	0.15	-0.23	0.36
P2 Chord	317	0.66	1.83	6.45*	0.08	0.15	-0.21	0.37	0.04	0.04	-0.05	0.16	-0.36	0.26
M1 chord	317	0.73	1.81	7.21*	0.18	0.14	-0.10	0.46	0.04	0.04	0.11	0.15	-0.18	0.40
M2 chord	317	0.56	1.60	6.24*	0.11	0.15	-0.17	0.40	0.03	0.02	-0.01	0.16	-0.32	0.29
Mandibular arch chords														
I2 chord	316	0.11	0.71	2.70*	-0.10	0.16	-0.42	0.22	0.06	0.05	-0.08	0.16	-0.40	0.24
C chord	319	0.19	1.31	2.60*	0.13	0.14	-0.16	0.41	0.07	0.08	0.04	0.15	-0.25	0.34
P1 chord	319	-0.13	1.78	1.27	0.23	0.14	-0.04	0.50	0.07	0.07	0.14	0.14	-0.14	0.42
P2 chord	317	-0.36	1.62	3.91*	0.19	0.14	-0.09	0.47	0.05	0.05	0.02	0.15	-0.29	0.32
M1 chord	318	-0.37	1.63	4.08*	0.31*	0.13	0.05	0.58	0.04	0.04	0.24	0.14	-0.03	0.51
M2 chord	319	-0.52	1.65	5.60*	0.30*	0.13	0.04	0.56	0.03	0.03	0.43*	0.13	0.18	0.68

†All variables, except angles, were measured in millimeters. Codes are: number of individuals (n), sample mean ($\bar{x}$), standard deviation (SD), *t*-tests for directional asymmetry (H_0: = 0), heritability estimate (h^2), standard error of h^2 (SE), and 95% confidence limits of h^2 (L_1, L_2).
*$p < 0.05$

who analyzed family similarities within and between generations, which allowed them to conclude that their h^2 estimates for arch size are not substantially confounded by environmental covariance. The low genetic contribution for tooth angulations (Figure 3A) was the most divergent from among the types of variables measured. It is noteworthy that tooth angulations are anatomically different than the rest of the variables, which depend on size and shape of the supporting bone rather than on positions of the teeth themselves. Prior studies[28,29,79,80] found that bone-based skeletodental variables have higher h^2 estimates than tooth-based variables such as tooth rotations, displacements, and axial inclinations, where estimates generally are indistinguishable from $h^2 = 0$. The present results are consistent with prior findings in that the orientations of the teeth (rather than their locations in supporting bone) are found to be affected primarily by the

environment. It is speculated that local environmental factors control tooth angulations, but specific influences remain elusive.[23]

Six arch form ratios were calculated, three for each arch. One was a ratio of arch widths, one a ratio of arch depths, and the third a ratio of arch width to depth. These sorts of "shape" variables have been used in prior research,[61,79,81] and the intent was to test the familial influence on shape of the dental arches. Estimated heritabilities were lower than average, with a mean of 39%, so the majority of the variability in arch shape is acquired environmentally. Heritability of 0.39 indicates that the intraclass correlation was about 0.20, so the coefficient of determination would be 4%. In other words, virtually none of the shape in one adolescent's dental arch in this sample could be predicted from examination of a sibling's arch shape. Brown et al.[62] reported low, nonsignificant correlations between arch growth in the mesiodistal and transverse dimensions, concluding that "breadth and depth were largely independent of one another, probably affected by different development processes."

Tests for significant left-right asymmetry showed that the left quadrants were larger than the right, and this sidedness was generalized, covering most lengths and widths as well as tooth angulations. Woo[2,82] and others have documented similar results for the calvaria and some facial structures. They found the right side of the calvaria to be larger, which conventionally has been attributed to greater size of the right hemisphere of the brain with accommodating and compensating growth of the calvarial bones.[52,53] In contrast, Woo[2] found that the malars and maxillae (including the palate) exhibited the opposite directionality, left > right, perhaps in compensation for side differences elsewhere or because of acquired lateralities, such as chewing-side preference. The finding in the present study that the left side of the arch is characteristically larger is, then, in keeping with the rest of the facial plan. It is noteworthy that, in spite of the pervasiveness of asymmetry in the arch dimensions, the bulk of the tests for directional (28/31) and fluctuating (29/31) asymmetry had no discernible transmissible influence ($h^2 = 0$), which agrees with other studies that have tested for a genetic basis for developmental asymmetries.[83] The scattering of significant results was only as common as expected from chance.

It is interesting that sagittal molar relationship exhibited significant directional asymmetry since it may be related to the anecdotal findings that it takes more effort to move one side of a patient's dentition into a Class I molar relationship than the other. Statistically, this sample was less Class II dentally on the left side, so the average case required more sagittal correction on the right.

Assessment of the relative importance of genetics in defining size and shape of the arches is more than an academic exercise. It directs attention to the nature of the developmental problem. If arch form were modulated predominately by the genotype, then treatment is destined to be palliative. If, in contrast, heritability is low(as shown here), with the environment playing a more important role, then research ought to focus on elucidating those factors detrimental to development of the occlusion, with the ultimate aim of prevention.

Summary

Little is known of the factors controlling dental arch size and shape. The present familial analysis suggests some interpretations. Arch dimensions are significantly larger in boys, both mediolaterally and anteroposteriorly, than in girls, a sex difference largely established prior to onset of the adolescent growth spurt. Most aberations of tooth position (e.g., rotations, displacements) have nonsignificant h^2 estimates, implying that most of the variation is due to environmentally induced, acquired factors. In contrast, arch dimensions have h^2 estimates significantly different from zero, but the magnitude varies: h^2 estimates were highest for arch widths, chords, and depths, with a mean transmissibility of 50%. So, while there are significant familial similarities in arch size, at least half of the phenotypic variation in this sample is due to environmental differences.

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