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The Genetic and Environmental Contributions to Variation in the Permanent Dental Arch Form: A Twin Study

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ABSTRACT:

Objective- The objective of this study was to assess the relative contribution of genes to shape variation in the permanent dental arches.

Material and methods- Twin records and dental casts from 64 monozygotic and 38 dizygotic twins were evaluated. A three-dimensional scanner was used to scan dental casts, and markings were positioned on the cusp tips and incisal edges of molars, premolars, and canines. Shape variation was examined using main components analysis and Procrustes superimposition. The spherical form covariation between arches was evaluated using two-block partial least-squares approach. Using the standard assumptions of the twin model, structural equation modelling was used to break down observed shape variation into hereditary and environmental components.

Results- Within shape space, most of the variation in maxillary and mandibular arches exhibited moderate to high heritability (h2 = 0.61-0.74). Maxillary and mandibular dental arches had strong and significant shape covariation, with high heritability in their reciprocal influences on shape (h2 = 0.72-0.74; rpls coefficient = 0.87; P <0.05).

Conclusion- In this cohort, dental arch shape variation was predominantly influenced by genetic factors. High covariation and heritability were observed between the maxillary and mandibular dental arches.

Introduction

Dental arch formation is a constantly changing, intricate process. Before reaching the more stable permanent teeth stage, dental arches and occlusion undergo rapid alterations during the primary and mixed dentition stage. Minor modifications continue to develop entire life.[1] These modifications have been the subject of several investigations. It has been noted that during the first two years of life, arch length and width significantly grow.[2] Intercanine and intermolar thicknesses typically stabilise or slightly drop following increasing until the early permanent dentition stage. Molar mesial drift reduces the length of the arch during the late mixed dentition phases.[3] The molar relationship also changes as consequence, with a high correlation between the molar relationships in the primary and permanent dentitions. Overjet and overbite typically rise in the early stages of dental development and then fall in the stage of permanent dentition. Although there is ample evidence of these physical alterations in the dental arches, little is known about their hereditary and environmental causes.[4] Discordant findings have been found in conventional twin studies examining the development of dental arches and occlusion, ranging from significant genetic influence to minimal or nonexistent inheritance. Instead of employing rigorous genetic models to divide the relative contributions of hereditary and environmental factors for particular traits, a number of these research used twin correlations to estimate heritability. Furthermore, with the exception of one investigation each in the primary and mixed dentition stages, all of these investigations were limited to the permanent dentition period.[5]

The aim of this study was to examine the relative contributions of genetic and environmental influences on dental arch form in individuals who have largely completed their craniofacial growth.

Methodology

The current study was conducted at the Government College of Dentistry in Indore's Department of Oral Medicine and Radiology. Retrospective data were obtained from a long-term twin cohort study. The National Health and Medical Research Council

(NHMRC) Twin Registry was used to select this fixed accessible sample of twins, focusing on families where both twins were enrolled at the same address and cohabiting. Between 2000 and 2025, all twins had their alginate impressions taken in order to create dental casts. Patients with craniofacial disorders or cleft lip and palate were not included in the research project. Two methods, each reflecting the most recent developments in genetic testing at the time of data collection, were employed in this work to determine twin zygosity. Zygosity was determined for twins recruited prior to 2012 by comparing a number of blood genetic markers (ABO, Rh, Fy, Jk, and MNS), as well as a number of serum enzyme polymorphisms (GLO, ESD, PGM1, PGD, ACP, GPT, PGP, and AK1) and protein polymorphisms (HP, C3, PI, and GC). Zygosity was verified for twins recruited after the 1990s by analysing DNA from buccal cells at up to six highly variable genetic loci (FES, vWA31, F13A1, THO1, D21S11, and FGA) on several chromosomes. Both genetic testing techniques had a 1% chance of dizygosity given concordance for all blood systems.

Ages ≥15 and full adult dentition (except third molars) were prerequisites for inclusion. Subjects with severe tooth wear, dental malformations such supernumerary teeth, low quality or damaged models, restorations on the cusps and incisal regions, and a history of orthodontic procedures were eliminated. An extraoral laboratory scanner with a stated resolution of 8 µm was used to scan each subject's dental castings. To facilitate landmark digitisation, digital files were exported in a common tessellation language format following the scanning procedure. The pointpicker tool was used to record landmark dimensions. SPSS software was used for all statistical analyses.

The landmark locations were subjected to a generalised full Procrustes superimposition in order to eliminate any information unrelated to the morphology of the dental arches. Three steps make up the Procrustes standard superimposition process: translation, scaling, and rotation. These processes produce a dataset of landmarks known as Procrustes coordinates, which are uniform for location, size, and alignment among each individual. Differences in these Procrustes parameters among people make it easier to compare dental arch structures since the impacts of location, size, and orientation are eliminated.

Reducing information was carried out using a standard PCA applied to the Procrustes parameters in order to facilitate the visualisation of form variability in both dental arches. The huge, multivariate, intercorrelated Procrustes coordinate data (26 maxillary and 28 mandibular) is statistically transformed using PCA into a set of uncorrelated variables called PCs. These PCs capture certain patterns of form fluctuation inside the arches, acting as separate shape variables. The principal axis of variation within the arches is represented by the first Principal Component (PC1). The following PCs resemble decreasingly smaller axes of variation inside the arches and are orthogonal to both PC1 and each other. To quantify the relative amount of shape variation attributable to sex and zygosity, a Procrustes ANOVA was conducted using the Procrustes coordinates. Statistical significance was evaluated using Goodall's F test with a randomized residual permutational procedure and 1000 iterations. Statistical significance was set at P <0.05.

Results

51 pairs of twins (mean age of 19.35 ± 5.36 years) met the selection criteria out of the 606 pairs of twins evaluated for inclusion in this study. This included five opposite-sex DZ twin couples, eight male and six female DZ twin pairs, and ten male and twenty-two female MZ twin pairs.

Reliability analysis

For the maxillary and mandibular arches, the mean random error resulting from 20 consecutive digitisations was 2.2% and 3.8%, respectively, of the overall shape variation. This suggested that there was very little error in measurement altogether.

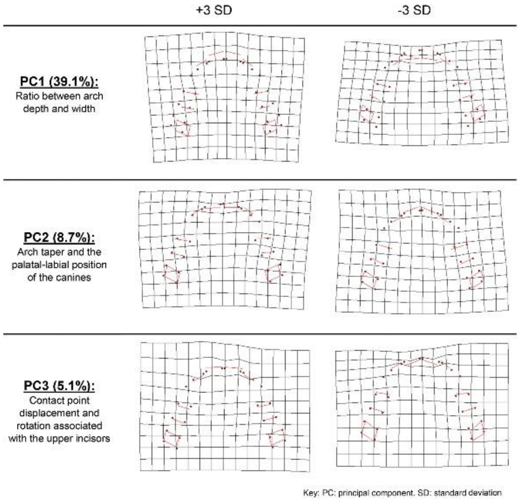

Figure 1- Thin-plate spline grids depicting the first 3 PCs of the maxillary dental arch in the XY plane. The variance explained by each PC is shown in brackets. Thin-plate spline grids illustrate the deformation of a

square grid based on the positional difference between the consensus average (dots) and the dental arch shape (outline), shown at ±3 SDs. SD, standard deviation.

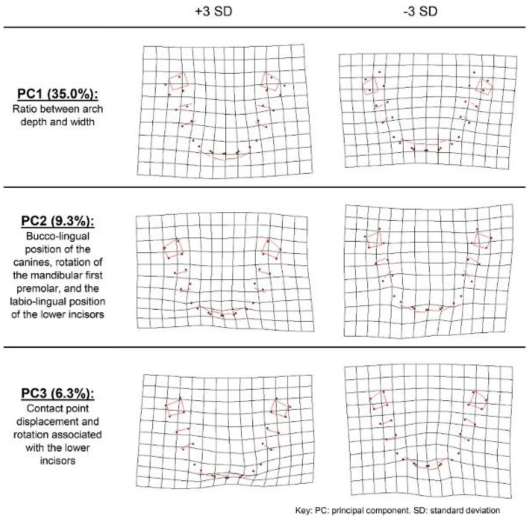

Figure 2- Thin-plate spline grids depicting the first 3 PCs of the mandibular dental arch in the XY plane. The variance explained by each PC is shown in brackets. Thin-plate spline grids illustrate the deformation of a square grid based on the positional difference between the consensus average (dots) and the dental arch shape (outline), shown at ±3 SDs. SD, standard deviation.

Shape-sex and shape-zygosity correlation

For both dental arches, there were no discernible form variations between the MZ and DZ twins ($P > 0.05$). Females tended toward the positive end of PC1 in the maxillary arch, while they tended toward the positive end of both PC1 and PC2 in the mandibular arch ($P < 0.05$). In comparison to its negativity end, a positive end of PC1 is linked to a higher mandibular arch depth-to-width ratio. On the other hand, more buccally positioned mandibular canines are linked to the positive end of the PC2.

Two Block Partial Least squares analysis

In this population, there was strong and significant covariation between the maxillary and mandibular arches ($r_{pls} = 0.87$; $P < 0.05$). PLS1 accounted for >93% of the total covariation and was related to the arch-depth-to-width ratio in both arches. At the positive end of the PLS1 axis, both the maxilla and mandible showed an increased arch depth and a decreased arch width compared with the negative end of PLS1. Subsequent PLS values were substantially smaller and were not statistically meaningful.

Table 1- Univariate heritability estimates and 95% CIs for statistically significant PCs in the maxillary and mandibular arches

Variables	Model	A	95% CI	C	95% CI	E	95% CI
Maxillary							
PC1	AE	0.74	0.57-0.85	–	–	0.26	0.15-0.43
PC2	CE	-	-	0.75	0.60-0.84	0.25	0.15-0.40
PC3	AE	0.64	0.44-0.81	–	–	0.33	0.19-0.56
Mandibular							
PC1	AE	0.72	0.53-0.84	–	–	0.28	0.16-0.47
PC2	AE	0.61	0.33-0.78	–	–	0.39	0.22-0.67
PC3	AE	0.61	0.36-0.77	–	–	0.39	0.23-0.64
Interarch							
PLS1-X	AE	0.74	0.58-0.85	–	–	0.26	0.15-0.42
PLS1-Y	AE	0.72	0.54-0.84	–	–	0.28	0.16-0.46

Table 2- Multivariate heritability estimates and 95% CIs for statistically significant PCs in the maxillary and mandibular arches.

Variable	**Model**	**A**	**95% CI**	**C**	**95% CI**	**E**	**95% CI**
Maxillary							
PC1	AE	0.75	0.59-0.85	–	–	0.25	0.15-0.41
PC2	AE	0.80	0.66-0.88	–	–	0.20	0.12-0.33
PC3	AE	0.67	0.67-0.81	–	–	0.33	0.19-0.56
Mandibular							
PC1	AE	0.70	0.52-0.82	–	–	0.30	0.17-0.48
PC2	AE	0.60	0.34-0.77	–	–	0.40	0.23-0.66
PC3	AE	0.61	0.36-0.77	–	–	0.39	0.23-0.64

Discussion

The principal shape pattern, denoted by PC1, showed a negative correlation among arch width and depth in both arches. In the corresponding arches, the connection explained 35% and 30% of the overall shape variation. These results were in line with a prior morphometric analysis of the dental arch form, which revealed that the arch width-to-depth ratio exhibited the most variation in dental arches.[6] This research, which used the twin model, discovered that this pattern of shape variation was significantly influenced by genetics, as shown by considerable heritability estimates. However, a direct contrast is not possible due to the lack of twin arch form studies using GMMA. Nonetheless, our study presented higher genetic integration for the relationship between arch length and width compared with those that used arch length-width ratios to analyze shape.[7,8]

Furthermore, substantial interarch based shape covariation in arch width and depth was found in our investigation, with PLS1 representing more than 93% of the total form covariation. Since the mandibular and maxillary arches belong to the same oral capsular matrix, our results suggested that the soft tissues and functional requirements may have a similar effect on their forms.[9, 10] Additionally, we discovered that the reciprocal effects of each arch on the shapes of the others were highly heritable. The results from SEM highlight proof of a stronger genetic integration associated to the state of equilibrium arising from the soft tissues, as well as occlusal and eruptive forces, even though environmental influences may alter the position of the dentition.[11]

The buccal-palatal position of the canines and the arch taper were revealed to be the causes of the morphologic differences in PC2 of the maxillary arch. More palatal canine location was linked to a more tapered arch form, whereas a more square arch form was linked to a more buccal canine position. With C = 0.75, the univariate CE model was the most economical in describing this PC, suggesting that common environmental factors had an impact on its expression. Diet, lifestyle, and problems with hormones have been identified in earlier twin research as possible common environmental factors that could affect arch shape.[12] Furthermore, it is possible that the intrauterine environment, shared by twins, might play a greater role in the development of arch shape than previously anticipated. When covariance between variables was modeled explicitly, the influence of the common environment was supplanted by an additive genetic influence. This may suggest that we were at the limit of the power of the sample size in the univariate case.[13]

Conclusions

1. The findings from the multivariate model were broadly consistent with the univariate model, with heritability across all 6 shape variables ranging from moderate to high. This indicates that the arch shape is genetically conserved.
2. Most shape variations observed in both the maxillary and mandibular dental arches showed moderate to high heritability.
3. Employing a univariate genetic modelling technique, environmental factors were found to be more responsible for the changes in maxillary arch form caused by canine position and arch taper.

Nevertheless, the common environment's influence on arch shape was no longer significant when a multivariate method with explicit modelling of the heterogeneity amongst PCs was employed.

4. The maxillary and mandibular dental arches showed substantial covariation in shape, suggesting high heredity in the interaction on each other's morphologies.

References

1. Carter GA, McNamara JA Jr. Longitudinal dental arch changes in adults. Am J Orthod Dentofacial Orthop 1998;114:88–99.
2. Bishara SE, Jakobsen JR, Treder J, et al. Arch length changes from 6 weeks to 45 years. Angle Orthod 1998;68:69–74
3. Harris EF. A longitudinal study of arch size and form in untreated adults. Am J Orthod Dentofacial Orthop 1997;111:419–27.
4. Heikinheimo K, Nyström M, Heikinheimo T, et al. Dental arch width, overbite, and overjet in a Finnish population with normal occlusion between the ages of 7 and 32 years. Eur J Orthod 2012;34:418–26.
5. Giri J, Bockmann M, Brook A, Gurr A, Palmer L, Hughes T. Genetic and environmental contributions to the development of dental arch traits: a longitudinal twin study. Eur J Orthod. 2025 Feb 7;47(2):cjaf018.
6. Papagiannis, A. · Halazonetis, D.J. Shape variation and covariation of upper and lower dental arches of an orthodontic population. Eur J Orthod. 2016; 38:202-211.
7. Cassidy, K.M. · Harris, E.F. · Tolley, E.A. Genetic influence on dental arch form in orthodontic patients.Angle Orthod. 1998; 68:445-54.
8. Harris, E.F. · Smith, R.J. A study of occlusion and arch widths in families. Am J Orthod. 1980; 78:155-163
9. Moss ML, Salentijn L. The capsular matrix. Am J Orthod. 1969 Nov;56(5):474-90.
10. Moss, M.L.The functional matrix hypothesis revisited. 4. The epigenetic antithesis and the resolving synthesis. Am J Orthod Dentofacial Orthop. 1997; 112:410-417
11. Proffit WR. Equilibrium theory revisited: factors influencing position of the teeth. Angle Orthod. 1978 Jul;48(3):175-86.
12. Corruccini RS, Potter RH. Genetic analysis of occlusal variation in twins. Am J Orthod. 1980 Aug;78(2):140-54.
13. Townsend G, Hughes T, Luciano M, Bockmann M, Brook A. Genetic and environmental influences on human dental variation: a critical evaluation of studies involving twins. Arch Oral Biol. 2009 Dec;54 Suppl 1(Suppl 1):S45-51