Quantitative Genetics, Pleiotropy, and Morphological Integration in the Dentition of *Papio hamadryas*

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Abstract

Variation in the mammalian dentition is highly informative of adaptations and evolutionary relationships, and consequently has been the focus of considerable research. Much of the current research exploring the genetic underpinnings of dental variation can trace its roots to Olson and Miller's 1958 book *Morphological Integration*. These authors explored patterns of correlation in the post-canine dentitions of the owl monkey and *Hyopsodus*, an extinct condylarth from the Eocene. Their results were difficult to interpret, as was even noted by the authors, due to a lack of genetic information through which to view the patterns of correlation. Following in the spirit of Olson and Miller's research, we present a quantitative genetic analysis of dental variation in a pedigreed population of baboons. We identify patterns of genetic correlations that provide insight to the genetic architecture of the baboon dentition. This genetic architecture indicates the presence of at least three modules: an incisor module that is genetically independent of the post-canine dentition, and a premolar module that demonstrates incomplete pleiotropy with the molar module. We then compare this matrix of genetic correlations to matrices of phenotypic correlations between the same measurements made on museum specimens of another baboon subspecies and the Southeast Asian colobine *Presbytis*. We observe moderate significant correlations between the matrices from these three primate taxa. From these observations we infer similarity in modularity and hypothesize a common pattern of genetic integration across the dental arcade in the Cercopithecoidea.

Keywords

Dental variation; Modularity; Evolution; Primates; Genetic architecture

Introduction

Fifty years ago Olson and Miller published *Morphological Integration* (1958), developing the intellectual foundation for much of the current research exploring the relationship between genotype and phenotype (Mitteroecker and Bookstein 2008). In honor of the fiftieth anniversary of Olson and Miller's seminar publication, we discuss their contributions to

dental morphological evolution specifically and follow with our study of the genetic underpinnings of morphological integration in the primate dentition, a study inspired by the 1958 book.

Olson and Miller (1958) noted that "character changes occurring in evolution of species could not be considered to be independent of each other... the interrelationships of changing characters [is] a primary point of interest" (p. 1). They divided these interrelationships into ρ-groups and ρF-groups, the former being correlations of a particular level, and the latter being correlations that result from a common function. The authors then provide several examples of how these correlations can elucidate morphological evolution.

The size and shape of the dentition is of fundamental importance in vertebrate evolution, as teeth are highly informative of an animal's diet, foraging strategy, interactions with conspecifics, and phylogenetic relationships. Additionally, teeth are primarily inorganic and thus survive well in the fossil record. For many vertebrates, all we know of them is what their teeth looked like.

Olson and Miller (1958) recognized the importance of the dentition in vertebrate evolution and consequently dedicated more than 20% of *Morphological Integration* to a discussion of ρ- and ρF-groups within tooth size variation. They studied linear measurements for all post-canine teeth of *Aotus trivirgatus* (the small South American owl monkey) and the extinct condylarth *Hyopsodus* from the Eocene of North America. Despite the former being a primate and the latter a primitive ungulate (Gingerich 1974), they have similar dental morphologies.

Olson and Miller concluded that the post-canine dentition, as a whole, is poorly integrated, but that each tooth is highly integrated. They were also the first to note that these results were difficult to interpret, largely, they state, because there are no "guiding principles" for how to frame these results (1958, p. 182). They elaborate,

> Evidence from studies of the genetics of dentition is virtually non-existant, and until the situation is remedied the uncertainties inherent in purely inferential interpretations cannot be removed. If, however, it is possible to gain additional insight into the scope of the selective unit, some progress can be made toward an understanding of the subordinate dental characters that are so important to studies of fossil mammals. Investigations directed toward this end fall within the domain of the concept of morphological integration. (1958, p. 182)

One of the most significant sources of genetic insight since 1958 has been developmental genetics. We have learned a considerable amount about the genes necessary to make a tooth, and how they interact during odontogenesis (reviewed in Jernvall and Thesleff 2000; Stock 2001; Tucker and Sharpe 2004). Our current understanding of tooth development is largely derived from gene expression and knock-out studies on mice. However, the few comparative analyses done to date demonstrate that the genetics of tooth organogenesis are likely to be highly conserved across mammals (e.g., Keränen et al. 1998; Kapadia et al. 2007; Lin et al. 2007; Miyado et al. 2007).

The next step towards achieving Olson and Miller's goal of identifying a "guiding principle" is to translate what is known about tooth developmental genetics to our understanding of how teeth vary within a population, and how this has evolved through time—connecting the genetics of organogenesis to population-level phenotypic variation (e.g., Nemeschkal 1999; Stern 2000; Hlusko 2004; Colosimo et al. 2005). This has become a productive research direction within biology (Koentges 2008), though by no means facile or straightforward (Weiss 2008).

The concept of *modularity* provides an essential tool for exploring the relationship between the genotype and phenotype, often referred to as genotype–phenotype mapping (Wagner 1996; Schlosser and Wagner 2004; Wagner et al. 2007). Modularity in the vertebrate limb is a successful example, primarily because of the patterned function and expression of *Hox* genes. *Hox* gene expression patterns have been demonstrated to correspond to morphological modularity within vertebrate autopods (Wagner and Vargas 2008), forelimbs (Reno et al. 2008), and entire limbs (Shubin et al. 1997; Shubin 2002).

However, *Hox* genes are not expressed in the first branchial arch from which the dentition derives (James et al. 2002), and therefore do not similarly pattern the dental arcade. Instead, the patterning of the dentition may be due to a combinatorial code of the *Barx, Dlx, Msx*, and *Pitx* gene families (Cobourne and Sharpe 2003). The applicability of this odontogenic combinatorial code model to non-mouse taxa, however, remains to be determined (for alternatives see Weiss et al. 1998; Stock 2001).

Another approach for identifying the genetic underpinnings of morphological integration is quantitative genetics. Lande (1979, 1980); Cheverud (1982, 1989, 1995, 1996a, b), Cheverud and colleagues (e.g., Cheverud et al. 1983; Marroig et al. 2004), and numerous others (e.g., Schluter 2000) have demonstrated the power of quantitative genetics for identifying shared genetic effects between traits (e.g., pleiotropy), and understanding how these genetic correlations can affect morphological evolution.

Quantitative genetic analyses test the hypothesis that environmental, or non-genetic factors alone can account for the phenotypic similarities seen among family members. A significant heritability estimate for one phenotype, or a genetic correlation between two phenotypes, indicates that environmental effects by themselves cannot account for the pattern of phenotypic variation seen in a population of related individuals, and as such, the degrees of interrelatedness contribute to the phenotypic similarities (i.e., genes shared due to a common ancestor, estimated via the kinship coefficient).

We employed this method to identify the genetic underpinnings of morphological integration by determining how much of a phenotypic correlation between two phenotypes results from the genetic correlation between them. In other words, we explore Olson and Miller's (1958) ρ-groups as genetic correlation groups—this is now most commonly presented in matrix form.

Phenotypic variation (Vp) is the sum of the genetic (Vg) and environmental variances (Ve), such that $Vp = Vg + Ve$. Therefore, it is evident that the higher the heritabilities of the phenotypes included in a matrix (i.e., the greater the proportion of the phenotypic variance that is due to the additive effects of genes), the more the phenotypic correlation matrix, P, will reflect the genetic correlation matrix, G (e.g., Cheverud 1988). Numerous studies have demonstrated this phenomenon, that phenotypic correlation matrices reflect the underlying genetic correlations (e.g., Cheverud 1988; Arnold 1992; Roff 1995, 1996; Koots and Gibson 1996).

The gold standard for evolutionary quantitative genetic analyses is to estimate genetic correlations for phenotypes in all populations studies (e.g., Arnold and Phillips 1999; Phillips and Arnold 1999; Steppan et al. 2002; Caruso et al. 2005; Colosimo et al. 2005), and ultimately tie these to gene expression studies (e.g., Nemeschkal 1999). But given the difficulty of establishing pedigree structure, this is not possible for most populations or species, especially those that are extinct. However, a number of other researchers have demonstrated that cautiously exploring the phenotypic correlation matrix as a proxy for the genetic correlation matrix of a population is a productive means through which to study the evolution of modularity, or morphological integration as Olson and Miller predicted in 1958

(e.g., Cheverud et al. 1989; Ackermann and Cheverud 2000, 2002; Mezey et al. 2000; Magwene 2001).

Within primates, this type of research has largely focused on the cranium (e.g., Richtsmeier et al. 1984; Kohn et al. 1993; Cheverud 1996b; Ackermann and Cheverud 2002; Marroig et al. 2004; Roseman 2004; Marroig and Cheverud 2005; Wolf et al. 2005; Ackermann 2007; Mitteroecker and Bookstein 2008; Sherwood et al. 2008), all identifying morphological integration in the cranium, and demonstrating that cranial evolution follows predictable trajectories described by the pattern of genetic correlations (e.g., Marroig and Cheverud 2005).

Although the application of quantitative genetic methods to primate dental variation is not new, the majority of published studies have analyzed data from humans (reviewed in Rizk et al. 2008). We have undertaken the most extensive quantitative genetic analysis of dental variation in a non-human primate to date, employing a captive pedigreed breeding colony of baboons housed at the Southwest National Primate Research Center (Hlusko 2004; Hlusko et al. 2002, 2004a, b, 2006; Hlusko and Mahaney 2003, 2007a, b, 2008).

Here we report on a quantitative genetic analysis of maxillary tooth size variation in this pedigreed population of baboons. These analyses reveal, for the first time, the genetic architecture of population level tooth size variation in *Papio hamadryas*. Patterns of high genetic correlations are hypothesized to underlie modularity in tooth size variation across the dental arcade. Given the highly conserved nature of most of dental developmental genetics noted above, these modules may be present in other baboons, other Old World Monkeys (OWM), primates more broadly, and possibly even other mammals.

In order to test the hypothesis of common modularity, we undertake a series of matrix comparisons. First, we compare the matrix of genetic correlations to the matrix of phenotypic correlations within the same pedigreed population to determine the degree to which the latter reflect the former in these baboons. We then compare the genetic correlation matrix to the phenotypic correlation matrices for non-related baboons and the more evolutionarily distant Southeast Asian colobine *Presbytis* to assess the degree to which the genetic underpinnings of odontometric modularity detected in the captive baboons might also be detected in other cercopithecoids.

Materials

Data for the genetic analyses were collected from a large captive, pedigreed breeding colony of baboons (>3000) housed at the Southwest National Primate Research Center (SNPRC) at the Southwest Foundation for Biomedical Research in San Antonio, Texas. This colony is comprised largely of *Papio hamdryas anubis* with some *P. h. cynocephalus*, and *P. h. hamadryas* (as defined in Jolly 1993).

The colony is maintained in pedigrees with all mating opportunities controlled. Genetic management of the colony was started over 20 years ago and allows for data collection from non-inbred animals. All non-founder animals in this study resulted from matings that were random with respect to dental, skeletal, and developmental phenotype. The female to male sex ratio is approximately 2:1. Genetic marker maps were made for 694 individuals (Rogers et al. 2000), making QTL analyses for these data possible (in preparation).

All pedigree data management and preparation was facilitated through use of the computer package PEDSYS (Dyke 1996). The animals from which data have been collected ($n = 630$) are distributed across 11 extended pedigrees that are 3–5 generations deep. The mean

number of animals with data per pedigree is 44; these individuals typically occupy the lower 2–3 generations.

All SNPRC odontometric data were collected from high resolution plaster dental casts made from living animals while they were anesthetized (for details see Hlusko et al. 2002) or from skeletal remains curated at the University of Washington under the direction of J. Cheverud. The Institutional and Animal Care and Use Committee, in accordance with the established guidelines (National Research Council 1996), approved all procedures related to the treatment of the baboons during the conduct of this study. The dental data were collected either with calipers (incisors and premolars) or from digital photographs (molars) of the casts (protocol described in detail elsewhere, Hlusko et al. 2002).

Phenotypic data consisted of standard linear size measurements of the maxillary dentition: mesiodistal length and labiolingual width for incisors, mesiodistal length and buccolingual width for premolars, mesiodistal length and mesial and distal buccolingual width for the molars. Measurements were not available for the canines as the canines are clipped or pulled for the animals' safety in captivity.

Phenotypic data for the non-pedigreed populations are from the National Science Foundation-sponsored on-line free access database of Old World Monkey dental metrics, PRIMO (http://www.nycep.org/primo/). We used two samples from this database. The first consists of 186 *Papio hamadryas* (34 categorized as *P. h. anubis* and 152 as *P. h. ursius*). The second sample is a smaller collection of *Presbytis* ($n = 25$). These specimens are all housed in museum collections (American Museum of Natural History, British Museum of Natural History, Florida State Museum, National Museum of Natural History, Senckenbergische Anatomie, and C. Jolly's collection). The same maxillary linear measurements were used for the SNPRC and the PRIMO samples; all PRIMO data were collected with calipers. Figure 1 shows a typical baboon dentition.

Analytical Methods

Statistical genetic analyses were conducted by means of a maximum likelihood based variance decomposition approach implemented in the computer package SOLAR (Almasy and Blangero 1998). The phenotypic covariance for each trait within a pedigree is modeled as $\Omega = 2\Phi\sigma_G^2 + I\sigma_E^2$, where Φ is a matrix of kinship coefficients for all relative pairs in a pedigree, σ_G^2 is the additive genetic variance, I is an identity matrix (composed of ones along the diagonal and zeros for all off-diagonal elements), and σ_E^2 is the environmental variance. The components of the phenotypic variance are additive, such that $\sigma_P^2 + \sigma_G^2 + \sigma_E^2$, enabling us to estimate heritability, or the proportion of the phenotypic variance attributable to additive genetic effects as $h^2 = \frac{\sigma_G^2}{\sigma_P^2}$. Phenotypic variance attributable to non-genetic factors is estimated as $e^2 = 1 - h^2$. The mean effects of sex and age were tested in the analyses and included in all subsequent analyses if found to significantly contribute to the phenotypic variance of a trait.

Using extensions to univariate genetic analyses that encompass the multivariate state (Hopper and Mathews 1982; Lange and Boehnke 1983; Boehnke et al. 1987), we modeled the multivariate phenotype of an individual as a linear function of the measurements on the individual's traits, the means of these traits in the populations, the covariates and their regression coefficients, plus the additive genetic values and random environmental deviations (described in detail in Mahaney et al. 1995).

From this model, we obtained the phenotypic variance–covariance matrix from which we partitioned the additive genetic and random environmental variance–covariance matrices, given the relationships (kinship coefficients) observed in the pedigree. From these two variance–covariance matrices, we estimated the additive genetic correlation, ρ_G, and the environmental correlation, ρ_E, between trait pairs. Respectively, these correlations are estimates of the additive effects of shared genes (i.e., pleiotropy) and shared environmental (i.e., unmeasured and nongenetic) factors on the variance in a trait.

We use the maximum likelihood estimates of the additive genetic and environmental correlations to obtain the total phenotypic correlation between two traits. ρ_P, as

$\rho_P = \sqrt{h_1^2}\sqrt{h_2^2}\rho_G + \sqrt{\left(1 - h_1^2\right)}\sqrt{\left(1 - h_2^2\right)}\rho_E$. The genetic correlation between trait pairs was estimated using multivariate extensions to the basic variance decomposition methods implemented in SOLAR (Almasy and Blangero 1998).

Significance of the maximum likelihood estimates for these parameters was assessed by means of likelihood ratio tests. Twice the difference of the maximum likelihoods of a general model (in which all parameters are estimated) and a restricted model (in which the value of a parameter to be tested is held constant at some value, usually zero) are compared. This difference is distributed asymptotically approximately as either a ½:½ mixture of χ^2 and a point mass at zero for tests of parameters like h^2 for which a value of zero in a restricted model is at a boundary of the parameter space, or as a χ^2 variate for tests of covariates for which zero is not a boundary value (Hopper and Mathews 1982). In both cases degrees of freedom is equal to the difference in the number of estimated parameters in the two models (Boehnke et al. 1987). However, in tests of parameters like h^2, whose value may be fixed at a boundary of their parameter space in the null model, the appropriate significance level is obtained by halving the P-value (Boehnke et al. 1987).

For bivariate models in which genetic correlations are found to be significantly greater than zero, additional tests are performed to compare the likelihood of a model in which the value of the genetic correlation is fixed at 1 or 0 to that of the unrestricted model in which the value of the genetic correlation is estimated. A significant difference between the likelihoods of the restricted and polygenic models suggests incomplete pleiotropy, i.e., not all of the additive genetic variance in the two traits is due to the effects of the same gene or genes.

Genetic correlations between traits can result from either pleiotropy or gametic phase disequilibrium (Lynch and Walsh 1998). The degree of gametic phase disequilibrium (or linkage disequilibrium, LD) is a function of a population's genetic history and demography: e.g., it will be lower in outbred populations with many unrelated founders as recombination exerts its affects each generation, higher in populations undergoing rapid expansion from a small number of founders and those resulting from recent admixture. Given a conducive set of population characteristics, the likelihood of genetic correlation between two traits being due to LD is higher for simple traits, with monogenic (or nearly so) inheritance. However, if variation in a pair of traits is attributable to the effects of multiple alleles at multiple loci, LD is not likely to be a major contributor to the genetic correlation (Lande 1980; Lynch and Walsh 1998). Therefore, we are cautiously confident that significant additive genetic correlations estimated in our analyses on pairs of complex, multifactorial dental measures from our non-inbred, extended baboon pedigrees are primarily indicative of pleiotropy rather than LD. Ongoing and planned whole genome screens and LD analyses in this population will help confirm this.

Genetic correlations between all possible pair-wise comparisons of the SNPRC dental linear metrics were used to construct a G matrix. We then calculated the elements of the P matrix for this sample of related individuals using the identity presented above. To assess the

degree of similarity between the G and P correlation matrices for this pedigreed population, and between these matrices and the phenotypic (Pearson's) correlation matrices constructed for the PRIMO data, we employed the Mantel test (Mantel 1967; Cheverud 1989), implemented in the statistical software packer R© (http://www.r-project.org/).

The Mantel test is a statistical test of correlation between two or more dissimilarity (e.g., distance) or similarity (correlation) matrices of equal rank. We assessed the significance of correlations estimated by this method empirically from this distribution of correlations obtained after permuting the rows and columns of the matrices 1000 times. This procedure reduces our reliance on assumptions concerning the statistical distributions underlying the two matrixes and mitigates the effects of non-independence of elements within the matrices.

Results

Heritability estimates for the SNPRC population are reported in Table 1. All maxillary tooth linear measurements are heritable (P 0.05) except for the mesiodistal length of the left third molar, probably the result of the relatively small sample ($n = 234$).

Additive genetic correlations for all possible pair-wise comparisons are presented in Appendix. Figure 2 is a visual composite of the right and left side results. The only phenotype pairing that did not yield a significant result on either side of the dental arcade is the width of the third premolar and the distal width of the third molar.

We found that 4 of 6 incisor:incisor correlations are significantly different from zero, with three $\rho_G > 0.80$. Only five of the 52 possible incisor:post-canine ρ_G estimates are significantly different from zero and all but one of these is below 0.50. All possible premolar:premolar ρ_G estimates are significantly different from zero; three are not significantly different from one. Eleven of the 36 pre-molar:molar analyses returned genetic correlations that are not statistically different from zero; the rest (67%) indicate significant shared genetic effects. Only 6 of the molar: molar correlations are not statistically different from zero.

A visual inspection of the SNPRC P and G matrices (Fig. 2) reveals a similar overall pattern. Mantel's similarity test yields $r = 0.873$ for the left P versus G matrices and $r = 0.717$ for the right P versus G matrices ($P < 0.001$) (for comparison, SNPRC left versus right P matrices returned an estimate of $r = 0.795$, $P < 0.001$). These matrices are also statistically similar to the PRIMO *Papio* P matrix (SNPRC G matrix, $r = 0.36$, $P < 0.001$; SNPRC P matrix, $r = 0.35$, $P = 0.004$). The SNPRC and PRIMO *Papio* matrices are also statistically similar to the PRIMO *Presbytis* P matrix (SNPRC G matrix, $r = 0.474$, $P < 0.001$; SNPRC P matrix, $r = 0.436$, $P = 0.005$ (right) and $r = 0.5792$, $P < 0.001$ (left); PRIMO *Papio* matrix, $r = 0.346$, $P = 0.016$).

Discussion

Two fundamental questions in biology concern the relationship between genes and anatomy. How does the genotype effect the phenotype and how has this relationship influenced morphological evolution as seen through the fossil record? We are currently pursuing these two research questions using the baboon dentition as a model system. The results presented here are part of this larger project.

We have undertaken a quantitative genetic analysis of dental variation in the SNPRC baboon population to establish the genetic architecture of dental variation. We find that the G and P matrices of SNPRC dental size variation correlations are significantly similar with a relatively high r value ($r = 0.639$; $P < 0.001$). This estimate is on the higher end of r values

reported for comparisons of G and P correlation matrices (Table 1, Cheverud 1988), indicating that the G matrix is highly predictive of the P matrix, as was expected and as has been reported for other populations (e.g., Cheverud 1988, 1995; Arnold 1992; Roff 1995, 1996; Koots and Gibson 1996).

The results of our comparisons of the odontometric phenotypic correlation matrices for these three cercopithecoid species represent the first step towards determining whether or not this genetic architecture is characteristic of other Old World Monkeys, extant and extinct (using the fossil record of OWM evolution in Africa). The phenotypic correlation matrices required for these analyses require large sample sizes, which are still being acquired. However, we were able to use already-collected data from the PRIMO database to undertake some preliminary analyses.

The *Papio hamadryas ursinus/anubis* and *Presbytis* P matrices are significantly similar to the SNPRC G matrix although the *r* estimates are not high (~0.35) compared to the SNRPC P and G matrix comparison. While we find it intriguing that these matrices do show statistically significant similarity, we are cautious in the biological interpretation of these results, given that the *r* values are low. Further analyses, larger sample sizes, and more control over data collection methods (these data were not collected by the authors and followed different protocols) are needed to adequately test the hypothesis that the structure of the G matrix estimated for the SNPRC population is characteristic of the P matrices of other Old World Monkeys.

With that caveat in mind, we propose the hypothesis that these initial results reveal genetic modularity within dental variation of Old World Monkeys, and perhaps of primates and mammals more generally. While speculative at this point, there is evidence from the developmental side that bolsters this interpretation.

At the end of the 19th century, Bateson (1892, 1894) compared the variation of serially homologous structures to Chladni figures, frequency interference in wave patterns. Butler (1939, 1956) adapted this concept to the dentition and proposed that classes of teeth derive from one 'type,' proposing that tooth shape variation results from identical tooth primordia reacting to different concentrations of morphogens. This is known as the *field theory* and is characterized by ultimate tooth shape being determined by extrinsic factors expressed within three separate fields (incisor, canine, post-canine).

An alternative is the *clone theory*, proposed by Osborn (1978). In this hypothesis, each tooth in a class is produced by the replication of the original type or polar tooth (i.e., the first molar for the molar field). Morphology is therefore predetermined by intrinsic factors.

The concept of dental fields has been explored primarily through analyses of phenotypic correlation (e.g., Dahlberg 1945; Van Valen 1961; Henderson and Greene 1975; Lombardi 1975). However, none of these phenotypic correlation analyses is particularly conclusive and can be argued to support both theories. The resolution between the clone and the field theories for explaining dental patterning has been hindered by the same lack of genetic information that complicated Olson and Miller's (1958) interpretations.

The field and clone models described above, and the odontogenic combinatorial code outlined in the introduction, all propose various levels of modularity within the dentition. With that in mind, it is not surprising that we find modularity in our quantitative genetic analysis. The genetic architecture of the SNPRC baboon maxillary dentition provides evidence for at least three modules that affect tooth size variation: incisors, premolar, and molar. The incisor module is genetically independent of the entire post-canine dentition, whereas the premolar and molar modules have overlapping genetic effects.

While the three developmental models outline above are distinct from each other to various degrees, our results accord with elements of each. For example, the odontogenic combinatorial code proposed for patterning the mouse dentition (Cobourne and Sharpe 2003), if found to be common to all mammals, would suggest at least two modules within the dentition: an incisor and a molar module. Our analyses demonstrate significant genetic independence between the incisors and molars, supporting Cobourne and Sharpe's hypothesis. The odontogenic code does not address premolars, as mice lack premolars and canines.

Under the clone model, where factors intrinsic to each tooth primordium determine tooth size and shape, we would hypothesize that modules do not covary. The genetic independence we found between incisors and molars also supports this model. Additionally, the lack of a genetic correlation between premolars and incisors in the SNPRC baboons accords with this prediction.

However, we find that premolar size variation does have overlapping but non-identical genetic effects with molar size variation. Additional research is needed to explore this in more detail, but this result does accord with the field theory, as extrinsic factors influencing tooth size along the post-canine dentition could result in incomplete pleiotropy. Data from developmental genetics also supports applying elements of the field theory to our understanding of how premolars and molars are genetically interrelated (e.g., Kassai et al. 2005; Salazar-Ciudad and Jernvall 2002), as does a phenotypic analysis of seal tooth shape (Jernvall 2000).

After 50 years, Olson and Miller's predictions are now being realized. Genotype–phenotype mapping provides a useful framework for understanding morphological evolution. Here specifically, we have shown that a quantitative genetic analysis of tooth size variation may well provide insight, or a "guiding principle" for how to identify and conceptualize morphological integration within the dentition of primates, and possibly mammals more broadly.

We hope to track the evolution of these genetic modules through time by exploring the P matrices of various taxa within the Old World Monkey fossil record. Ultimately, we may be able to reconstruct the phenogenetic evolution (Weiss 2005) of the primate dentition by revealing how the genotype has responded to selective pressures placed on the phenotype, by correlating phenogenetic changes with possible selective pressures (such as climate change and/or environmental and habitat shifts).

The integration of developmental genetics with what may be called "microevolution" is challenging but productive if small-scale modules are studied using a combination of developmental genetics, quantitative genetics, and morphology (e.g., Nemeschkal 1999; Stern 2000; Hlusko 2004; Colosimo et al. 2005). Our study represents a step towards achieving this larger goal, following on the path first outlined by Olson and Miller 50 years ago.

Acknowledgments

Appendix

Table 2

Bivariate statistical genetic analyses: Maximum-likelihood estimates of genetic and environmental correlations[a]

Phenotype pair	N	Correlations (MLEs)		Significance of correlations P(Hypothesis)	
		ρ_G	ρ_E	$\rho_G = 0$	$|\rho_G| = 1$
Baboon right maxillary					
I1ll v I1md	481	0.322	0.269	0.055	<0.0000001
I1ll v I2ll	492	0.893	0.215	<0.0001	0.003
I1ll v I2md	492	0.863	–0.236	<0.0001	0.005
I1ll v P3l	529	0.804	–0.246	0.008	0.28
I1ll v P3w	542	0.437	0.004	0.039	0.0008
I1ll v P4l	564	0.168	0.353	0.277	<0.0000001
I1ll v P4w	563	0.421	–0.012	0.012	<0.00001
I1ll v M1l	548	0.461	–0.039	0.002	<0.0000001
I1ll v M1mw	543	0.017	–0.195	0.926	<0.0000001
I1ll v M1dw	543	–0.246	0.086	0.175	0.0000001
I1ll v M2l	575	0.330	0.159	0.030	<0.0000001
I1ll v M2mw	576	–0.192	0.0125	0.288	0.0000001
I1ll v M2dw	572	–0.265	0.117	0.159	0.000002
I1ll v M3l	531	0.169	0.598	0.548	0.018
I1ll v M3mw	564	-0.046	0.196	0.821	<0.0000001
I1ll v M3dw	549	–0.164	0.080	0.540	0.049
I1md v I2ll	492	0.314	–0.160	0.050	<0.0000001
I1md v I2md	492	0.450	0.022	0.006	0.0000001
I1md v P3l	536	0.046	0.294	0.865	0.012
I1md v P3w	545	0.048	0.345	0.801	0.00007
I1md v P4l	566	0.021	0.295	0.901	<0.0000001
I1md v P4w	565	0.149	0.260	0.404	<0.0000001
I1md v M1l	552	0.112	0.398	0.483	<0.0000001
I1md v M1mw	547	0.059	0.295	0.752	<0.0000001
I1md v M1dw	547	–0.034	0.284	0.851	0.0000001
I1md v M2l	576	0.129	0.399	0.399	<0.0000001
I1md v M2mw	577	–0.042	0.274	0.813	<0.0000001
I1md v M2dw	573	0.175	0.089	0.342	0.0000004
I1md v M3l	535	0.200	0.489	0.422	0.021
I1md v M3mw	567	–0.132	0.251	0.485	0.0000009
I1md v M3dw	553	0.125	0.070	0.642	0.061

Phenotype pair	N	Correlations (MLEs)		Significance of correlations P(Hypothesis)	
		ρ_G	ρ_E	$\rho_G = 0$	$|\rho_G| = 1$
I2ll v I2md	475	0.816	–0.144	<0.0001	0.0001
I2ll v P3l	520	–0.060	0.183	0.811	0.011
I2ll v P3w	531	0.151	0.182	0.435	0.00006
I2ll v P4l	553	–0.160	0.738	0.272	<0.0000001
I2ll v P4w	554	0.228	–0.063	0.171	<0.0000001
I2ll v M1l	550	0.287	–0.211	0.049	<0.0000001
I2ll v M1mw	543	–0.117	–0.318	0.483	0.0000001
I2ll v M1dw	544	–0.314	–0.063	0.059	0.0000001
I2ll v M2l	573	0.112	0.483	0.457	<0.0000001
I2ll v M2mw	575	–0.276	0.104	0.109	0.0000010
I2ll v M2dw	571	–0.198	0.086	0.264	0.0000007
I2ll v M3l	519	0.335	0.065	0.180	0.028
I2ll v M3mw	564	–0.085	0.204	0.632	0.0000002
I2ll v M3dw	537	–0.249	0.161	0.320	0.048
I2md v P3l	528	0.221	0.174	0.387	0.015
I2md v P3w	538	0.163	0.162	0.414	0.00004
I2md v P4l	560	–0.006	0.564	0.968	<0.0000001
I2md v P4w	559	0.199	0.053	0.259	<0.0000001
I2md v M1l	552	0.301	0.059	0.055	<0.0000001
I2md v M1mw	545	–0.094	0.024	0.595	0.0000003
I2md v M1dw	546	–0.226	0.275	0.188	<0.0000001
I2md v M2l	575	0.174	0.244	0.261	<0.0000001
I2md v M2mw	576	–0.007	–0.124	0.968	<0.0000001
I2md v M2dw	572	0.168	–0.185	0.363	0.0000006
I2md v M3l	529	0.520	0.175	0.053	0.046
I2md v M3mw	567	0.184	–0.052	0.318	<0.0000001
I2md v M3dw	547	0.477	–0.147	0.085	0.127
P3l v P3w	336	0.706	0.102	0.041	0.116
P3l v P4l	403	0.917	0.038	0.00005	0.321
P3l v P4w	421	0.580	0.094	0.026	0.041
P3l v M1l	505	0.729	–0.152	0.002	0.059
P3l v M1mw	502	0.385	0.334	0.208	0.009
P3l v M1dw	503	0.430	0.211	0.171	0.023
P3l v M2l	547	0.640	0.192	0.005	0.019
P3l v M2mw	548	0.076	0.409	0.794	0.012
P3l v M2dw	542	0.158	0.472	0.641	0.035
P3l v M3l	358	0.859	0.018	0.009	0.272
P3l v M3mw	494	0.546	0.128	0.052	0.014
P3l v M3dw	393	0.732	0.184	0.033	0.123
P3w v P4l	406	0.576	0.120	0.001	0.000015
P3w v P4w	423	1.000	–0.098	<0.00001	nc

Phenotype pair	N	Correlations (MLEs)		Significance of correlations P(Hypothesis)	
		ρ_G	ρ_E	$\rho_G = 0$	$|\rho_G| = 1$
P3w v M1l	517	0.313	0.584	0.091	0.00001
P3w v M1mw	515	0.358	0.233	0.119	0.00004
P3w v M1dw	516	0.051	0.757	0.821	0.00002
P3w v M2l	551	0.612	0.052	0.0003	0.00001
P3w v M2mw	554	0.477	0.207	0.025	0.00008
P3w v M2dw	547	0.514	0.163	0.028	0.0009
P3w v M3l	391	0.437	0.142	0.202	0.0431
P3w v M3mw	510	0.469	–0.012	0.045	0.0026
P3w v M3dw	428	0.049	0.303	0.882	0.0653
P4l v P4w	432	0.556	0.155	0.0003	<0.0000001
P4l v M1l	537	0.569	0.141	<0.0001	<0.0000001
P4l v M1mw	535	0.536	–0.316	0.001	<0.0000001
P4l v M1dw	535	0.445	0.066	0.0096	0.0000002
P4l v M2l	556	0.729	0.262	<0.0001	<0.0000001
P4l v M2mw	559	0.468	0.167	0.003	<0.0000001
P4l v M2dw	555	0.474	0.218	0.004	<0.0000001
P4l v M3l	439	0.646	0.253	0.001	0.010
P4l v M3mw	529	0.384	0.165	0.017	<0.0000001
P4l v M3dw	468	0.610	0.171	0.012	0.113
P4w v M1l	538	0.438	0.233	0.007	<0.0000001
P4w v M1mw	536	0.528	0.218	0.003	<0.0000001
P4w v M1dw	537	0.345	0.348	0.069	<0.0000001
P4w v M2l	557	0.534	0.197	0.0007	<0.0000001
P4w v M2mw	561	0.580	0.179	0.001	0.0000004
P4w v M2dw	557	0.519	0.150	0.007	0.000005
P4w v M3l	449	0.433	0.200	0.144	0.016
P4w v M3mw	534	0.577	0.032	0.001	0.00005
P4w v M3dw	479	0.404	0.264	0.194	0.071
M1l v M1mw	458	0.591	0.112	0.00017	<0.0001
M1l v M1dw	473	0.565	0.061	0.0008	0.0000004
M1l v M2l	547	0.918	–0.044	<0.0001	0.119
M1l v M2mw	548	0.485	–0.003	0.0036	<0.0001
M1l v M2dw	547	0.371	0.211	0.033	0.0000007
M1l v M3l	495	0.744	0.400	0.0023	0.054
M1l v M3mw	555	0.578	0.073	0.0007	0.00005
M1l v M3dw	523	0.280	0.242	0.207	0.009
M1mw v M1dw	445	0.933	0.492	<0.0001	0.0008
M1mw v M2l	543	0.555	–0.127	0.0004	<0.0001
M1mw v M2mw	543	0.865	0.362	<0.0001	0.0019
M1mw v M2dw	541	0.761	0.312	0.00004	0.0005
M1mw v M3l	482	0.544	–0.063	0.065	0.037

Phenotype pair	N	Correlations (MLEs)		Significance of correlations P(Hypothesis)	
		ρ_G	ρ_E	$\rho_G = 0$	$|\rho_G| = 1$
M1mw v M3mw	535	0.770	0.042	<0.0001	0.00055
M1mw v M3dw	502	0.208	0.448	0.49	0.037
M1dw v M2l	544	0.466	−0.297	0.0027	<0.0001
M1dw v M2mw	543	0.790	0.152	<0.0001	0.00018
M1dw v M2dw	542	0.737	0.298	0.00005	0.000007
M1dw v M3l	485	0.167	0.267	0.638	0.022
M1dw v M3mw	534	0.583	0.064	0.002	0.00008
M1dw v M3dw	523	0.286	0.232	0.192	0.009
M2l v M2mw	536	0.693	0.202	<0.0001	0.000006
M2l v M2dw	535	0.619	0.296	0.00011	<0.0001
M2l v M3l	514	0.947	0.346	<0.0001	0.303
M2l v M3mw	551	0.673	0.084	0.00005	0.0003
M2l v M3dw	548	0.267	0.476	0.212	0.012
M2mw v M2dw	531	0.821	0.734	0.00001	0.0000005
M2mw v M3l	542	0.625	0.180	0.031	0.060
M2mw v M3mw	552	0.880	0.335	<0.0001	0.068
M2mw v M3dw	549	0.397	0.576	0.303	0.154
M2dw v M3l	321	0.520	0.681	0.113	0.007
M2dw v M3mw	543	0.523	0.487	0.011	0.0000005
M2dw v M3dw	539	0.709	0.596	0.031	0.117
M3l v M3mw	453	0.608	0.669	0.018	0.012
M3l v M3dw	321	0.520	0.681	0.113	0.007
M3mw v M3dw	446	0.564	0.716	0.048	0.015
Baboon left maxillary					
I1ll v I1md	471	0.447	0.233	0.007	<0.0001
I1ll v I2ll	484	0.921	0.332	<0.0001	0.067
I1ll v I2md	471	0.654	−0.08	<0.001	0.0017
I1ll v P3l	531	0.626	−0.131	0.065	0.19
I1ll v P3w	537	0.524	−0.090	0.076	0.049
I1ll v P4l	565	0.177	0.174	0.347	<0.0001
I1ll v P4w	568	0.519	−0.228	0.008	0.0004
I1ll v M1l	557	0.080	0.339	0.67	<0.0001
I1ll v M1mw	550	−0.318	0.142	0.104	<0.0001
I1ll v M1dw	549	−0.280	0.185	0.145	<0.0001
I1ll v M2l	580	0.340	0.098	0.031	0.0000003
I1ll v M2mw	579	−0.303	0.178	0.126	0.000012
I1ll v M2dw	469	1.000	0.987	<0.0001	nc
I1ll v M3l	538	0.511	0.097	0.131	0.132
I1ll v M3mw	566	−0.174	0.108	0.532	0.0062
I1ll v M3dw	549	−0.280	0.185	0.145	<0.0001
I1md v I2ll	485	0.367	−0.053	0.018	<0.0000001

Phenotype pair	N	Correlations (MLEs)		Significance of correlations P(Hypothesis)	
		ρ_G	ρ_E	$\rho_G = 0$	$|\rho_G| = 1$
I1md v I2md	485	0.482	0.109	0.004	0.0000073
I1md v P3l	533	0.0346	0.297	0.907	0.0576
I1md v P3w	539	−0.070	0.250	0.780	0.0071
I1md v P4l	566	0.129	0.389	0.423	<0.0000001
I1md v P4w	568	0.372	−0.146	0.014	<0.0000001
I1md v M1l	557	0.133	0.484	0.353	<0.0000001
I1md v M1mw	550	0.149	0.087	0.335	<0.0000001
I1md v M1dw	549	0.085	0.235	0.561	<0.0000001
I1md v M2l	580	0.226	0.466	0.076	<0.0000001
I1md v M2mw	579	0.108	0.260	0.470	<0.0000001
I1md v M2dw	575	0.348	−0.067	0.022	0.0000001
I1md v M3l	540	0.991	−0.333	0.001	0.491
I1md v M3mw	567	0.194	0.043	0.379	0.002
I1md v M3dw	548	0.446	−0.079	0.067	0.047
I2ll v I2md	471	0.660	−0.079	0.0004	0.002
I2ll v P3l	524	0.414	0.024	0.187	0.095
I2ll v P3w	534	0.071	0.147	0.792	0.007
I2ll v P4l	582	0.001	0.590	0.995	<0.0000001
I2ll v P4w	565	0.175	−0.005	0.332	<0.0000001
I2ll v M1l	558	0.107	−0.007	0.540	<0.0000001
I2ll v M1mw	551	−0.343	0.303	0.059	<0.0000001
I2ll v M1dw	550	−0.296	0.349	0.089	<0.0000001
I2ll v M2l	579	0.181	0.208	0.226	<0.0000001
I2ll v M2mw	578	−0.303	0.256	0.096	0.0000004
I2ll v M2dw	574	0.022	−0.031	1.00	<0.0000001
I2ll v M3l	532	0.113	0.158	0.688	0.054
I2ll v M3mw	566	−0.443	0.242	0.074	0.026
I2ll v M3dw	542	−0.438	0.548	0.087	0.029
I2md v P3l	529	0.250	0.217	0.459	0.069
I2md v P3w	536	0.223	0.064	0.430	0.0061
I2md v P4l	583	0.362	0.410	0.054	0.0000004
I2md v P4w	565	0.243	−0.034	0.218	0.0000001
I2md v M1l	559	0.230	0.064	0.228	0.0000014
I2md v M1mw	553	−0.088	0.108	0.663	0.0000003
I2md v M1dw	552	−0.076	0.070	0.694	0.0000001
I2md v M2l	580	0.093	0.517	0.566	0.0000001
I2md v M2mw	579	−0.130	0.296	0.498	0.0000003
I2md v M2dw	575	0.103	0.134	0.600	0.0000001
I2md v M3l	538	0.595	0.022	0.129	0.224
I2md v M3mw	567	0.131	0.045	0.624	0.002
I2md v M3dw	547	0.350	0.137	0.207	0.016

Phenotype pair	N	Correlations (MLEs)		Significance of correlations *P*(Hypothesis)	
		ρ_G	ρ_E	$\rho_G = 0$	$|\rho_G| = 1$
P3l v P3w	348	0.517	0.329	0.201	0.055
P3l v P4l	428	0.554	0.207	0.036	0.023
P3l v P4w	465	0.568	0.454	0.013	0.006
P3l v M1l	519	0.514	0.177	0.066	0.056
P3l v M1mw	516	0.511	0.190	0.065	0.026
P3l v M1dw	516	0.472	0.124	0.136	0.077
P3l v M2l	553	0.517	0.274	0.035	0.037
P3l v M2mw	555	−0.117	0.548	0.67	0.022
P3l v M2dw	550	−0.068	0.484	1.00	0.027
P3l v M3l	374	−0.182	0.577	0.700	0.093
P3l v M3mw	499	−0.002	0.315	0.031	0.996
P3l v M3dw	401	−0.440	0.421	0.487	0.254
P3w v P4l	427	0.416	0.406	0.108	0.009
P3w v P4w	442	0.865	0.487	<0.0001	0.063
P3w v M1l	525	0.505	0.309	0.024	0.002
P3w v M1mw	519	0.550	0.420	0.025	0.013
P3w v M1dw	519	0.366	0.559	0.140	0.007
P3w v M2l	554	0.451	0.349	0.030	0.0009
P3w v M2mw	558	0.572	0.261	0.008	0.0008
P3w v M2dw	552	0.350	0.284	0.139	0.0009
P3w v M3l	411	−0.527	0.432	0.284	0.237
P3w v M3mw	511	0.411	0.221	0.224	0.004
P3w v M3dw	441	−0.686	0.436	0.171	0.281
P4l v P4w	451	0.620	0.241	0.0001	<0.0001
P4l v M1l	549	0.663	0.167	<0.0001	<0.0001
P4l v M1mw	548	0.498	0.149	0.003	<0.0001
P4l v M1dw	549	0.520	0.177	0.001	<0.0001
P4l v M2l	561	0.663	0.431	<0.0001	<0.0001
P4l v M2mw	565	0.445	0.108	0.008	<0.0001
P4l v M2dw	560	0.523	−0.042	0.001	<0.0001
P4l v M3l	463	0.402	0.432	0.122	0.023
P4l v M3mw	537	0.466	0.115	0.035	0.0006
P4l v M3dw	494	0.271	0.384	0.318	0.008
P4w v M1l	550	0.600	−0.128	<0.0001	<0.0001
P4w v M1mw	548	0.773	−0.125	0.0001	0.0001
P4w v M1dw	549	0.665	0.017	<0.0001	<0.0001
P4w v M2l	561	0.628	−0.093	<0.0001	<0.0001
P4w v M2mw	565	0.682	0.060	<0.0001	<0.0001
P4w v M2dw	561	0.515	0.222	0.0019	<0.0001
P4w v M3l	478	0.251	0.250	0.453	0.069
P4w v M3mw	541	0.691	0.255	0.0008	0.0015

Phenotype pair	N	Correlations (MLEs)		Significance of correlations P(Hypothesis)	
		ρ_G	ρ_E	$\rho_G = 0$	$|\rho_G| = 1$
P4w v M3dw	506	0.133	0.458	0.698	0.014
M1l v M1mw	474	0.669	−0.123	<0.0001	<0.0001
M1l v M1dw	474	0.714	−0.399	<0.0001	<0.0001
M1l v M2l	554	0.928	−0.153	<0.0001	0.093
M1l v M2mw	555	0.602	−0.086	<0.0001	<0.0001
M1l v M2dw	553	0.498	0.104	0.002	<0.0001
M1l v M3l	516	0.896	0.202	<0.0001	0.123
M1l v M3mw	542	0.839	0.023	<0.0001	0.040
M1l v M3dw	537	0.326	0.208	0.145	0.002
M1mw v M1dw	465	0.866	0.705	<0.0001	<0.0001
M1mw v M2l	553	0.627	−0.336	<0.0001	<0.0001
M1mw v M2mw	553	0.890	0.071	<0.0001	<0.0001
M1mw v M2dw	551	0.801	0.164	<0.0001	0.0013
M1mw v M3l	510	0.675	0.099	0.014	0.125
M1mw v M3mw	539	0.885	0.181	<0.0001	0.080
M1mw v M3dw	513	0.624	0.008	0.009	0.044
M1dw v M2l	553	0.615	−0.539	<0.0001	<0.0001
M1dw v M2mw	553	0.780	0.120	<0.0001	<0.0001
M1dw v M2dw	550	0.800	0.303	<0.0001	<0.0001
M1dw v M3l	509	0.485	0.339	0.117	0.064
M1dw v M3mw	540	0.718	0.259	0.0006	0.005
M1dw v M3dw	513	0.605	0.186	0.010	0.020
M2l v M2mw	548	0.677	−0.029	<0.0001	<0.0001
M2l v M2dw	547	0.604	0.203	<0.0001	<0.0001
M2l v M3l	548	0.828	0.286	0.0003	0.196
M2l v M3mw	552	0.878	−0.012	<0.0001	0.095
M2l v M3dw	551	0.549	0.000	0.0029	0.0011
M2mw v M2dw	541	0.903	0.474	<0.0001	0.0023
M2mw v M3l	549	0.904	−0.041	0.0008	0.342
M2mw v M3mw	557	1.00	0.347	<0.0001	nc
M2mw v M3dw	553	0.746	0.092	0.0001	0.0159
M2dw v M3l	542	0.903	0.064	0.002	0.349
M2dw v M3mw	549	0.867	0.272	<0.0001	0.060
M2dw v M3dw	545	0.914	0.149	<0.0001	0.19
M3l v M3mw	456	0.697	0.505	0.112	0.189
M3l v M3dw	347	0.697	0.508	0.078	0.106
M3mw v M3dw	443	0.457	0.693	0.26	0.0058

[a]*MLE* maximum likelihood estimate; *P(Hypothesis)* probability of the hypothesis (indicated in columns below) being true given the available pedigreed data; *nc* not computable; all phenotypes indicated with * in Table 1 were i-normalized in the bivariate analyses

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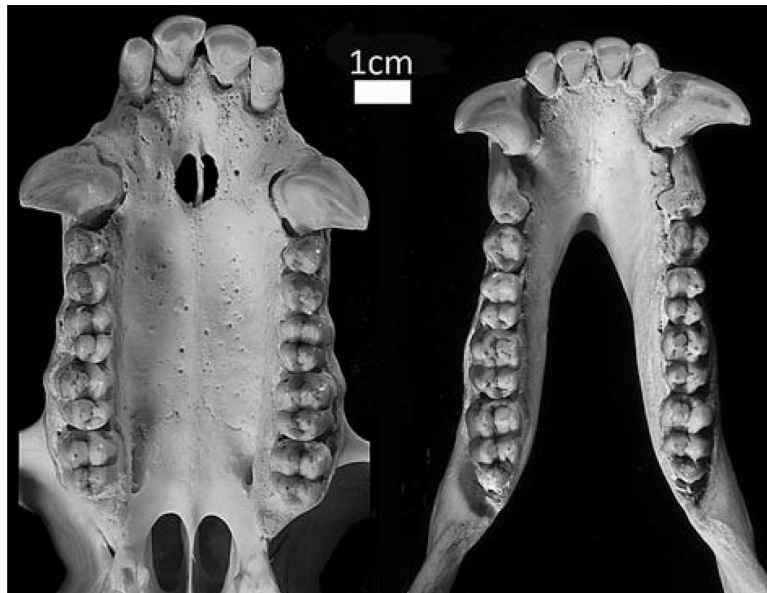
Fig. 1.
Occlusal view of the *Papio hamadryas* maxillary (*left*) and mandibular (*right*) dental arcades
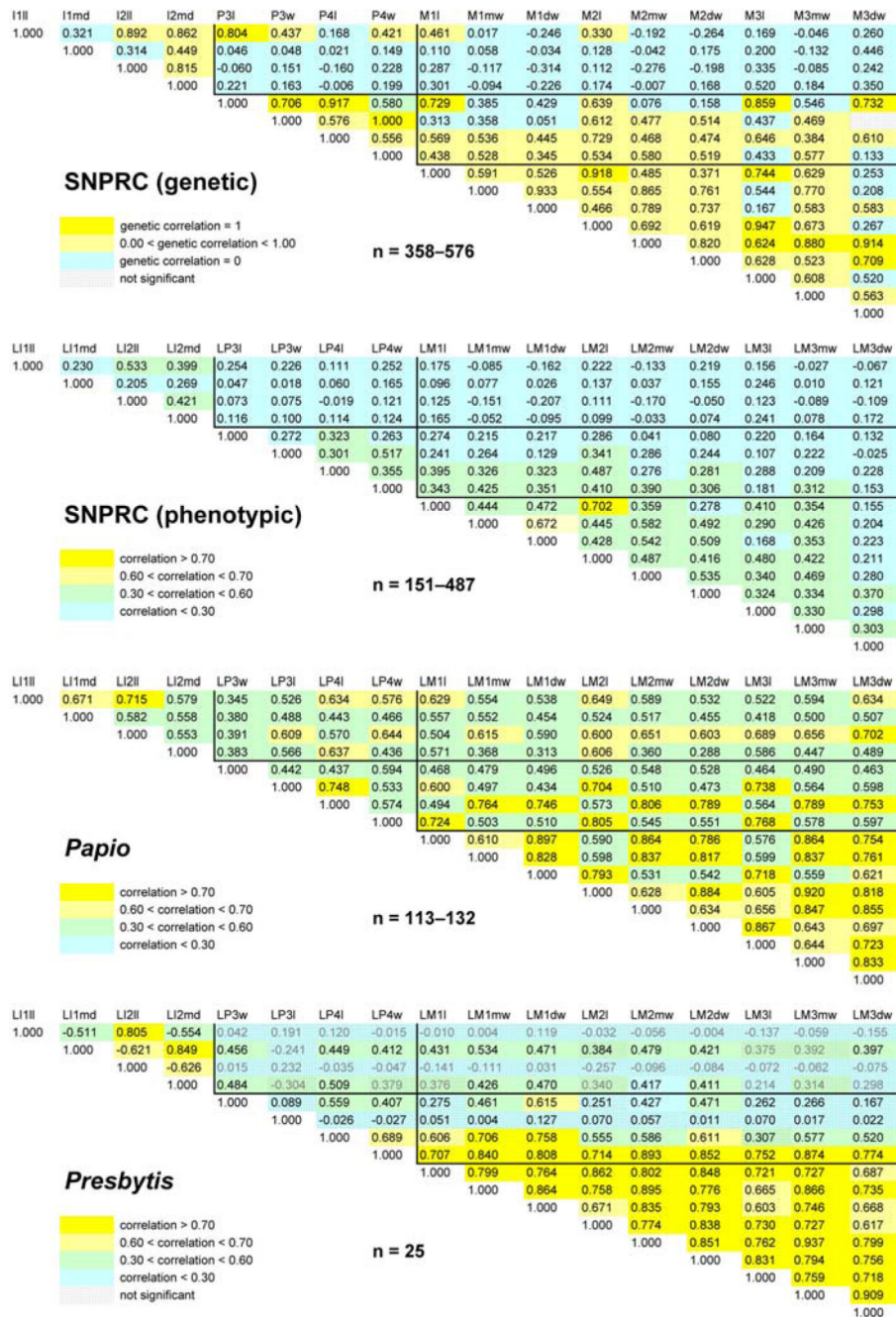
Fig. 2.
Genetic and phenotypic correlation matrices. Correlations were estimated as described in the text. The SNPRC genetic correlation matrix is a composite of the left and right sides of the maxillary dental arcade. The SNPRC phenotypic correlation matrix shows the average between the left and right side estimates

Table 1

Polygenic models for individual tooth measurements[a]

Trait	Mean	Var	n	Kurtosis	P-value	Total h^2	Total c^2	Total e^2	Residual $h^2 \pm$ SE
Baboon right maxillary									
I1ll	9.07	1.08	473	−0.3274	<0.0001	0.49	0.194	0.32	0.605 ± 0.12
I1md	9.51	0.55	480	0.4816	<0.0001	0.51	0.125	0.37	0.578 ± 0.11
I2ll	7.98	1.09	463	0.5029	<0.0001	0.51	0.204	0.28	0.642 ± 0.11
I2md	7.05	0.91	474	0.5982	<0.0001	0.52	0.141	0.33	0.611 ± 0.11
P3l	6.71	0.31	276	−0.1182	0.006	0.25	0.201	0.55	0.316 ± 0.15
P3w *	7.82	0.44	317	0.7641	<0.0001	0.43	0.346	0.22	0.659 ± 0.20
P4l	7.63	0.27	400	0.4849	<0.0001	0.48	0.295	0.23	0.680 ± 0.12
P4w *	8.51	0.38	430	0.0152	<0.0001	0.37	0.368	0.26	0.591 ± 0.12
M1l *	10.68	0.40	471	0.2626	<0.0001	0.44	0.336	0.23	0.659 ± 0.11
M1mw	8.38	0.30	438	0.7627	<0.0001	0.55	0.184	0.27	0.672 ± 0.14
M1dw *	7.87	0.29	439	0.4530	<0.0001	0.62	0.190	0.19	0.763 ± 0.16
M2l *	12.47	0.69	531	1.4037	<0.0001	0.46	0.425	0.12	0.798 ± 0.11
M2mw *	9.88	0.47	530	0.5056	<0.0001	0.39	0.291	0.32	0.544 ± 0.12
M2dw	8.85	0.40	517	0.5223	<0.0001	0.37	0.305	0.32	0.533 ± 0.13
M3l *	12.62	0.83	183	0.0403	0.013	0.24	0.483	0.28	0.455 ± 0.26
M3mw *	9.97	0.75	444	0.9430	<0.0001	0.35	0.381	0.27	0.562 ± 0.13
M3dw	8.50	0.56	286	0.0408	0.021	0.22	0.345	0.44	0.331 ± 0.19
Baboon left maxillary									
I1ll	8.96	1.06	469	0.5843	<0.0001	0.37	0.176	0.46	0.446 ± 0.11
I1md	9.58	0.48	471	0.0452	<0.0001	0.55	0.156	0.29	0.654 ± 0.10
I2ll *	7.12	0.60	481	0.3304	<0.0001	0.54	0.099	0.36	0.595 ± 0.12
I2md	5.62	0.48	471	0.3270	<0.0001	0.36	0.212	0.43	0.452 ± 0.11
P3l *	6.69	0.34	287	−0.1619	0.017	0.20	0.148	0.65	0.236 ± 0.14
P3w	7.75	0.41	323	0.5493	0.004	0.18	0.388	0.43	0.292 ± 0.14

Trait	Mean	Var	n	Kurtosis	P-value	Total h^2	Total c^2	Total e^2	Residual $h^2 \pm$ SE
P4l [*]	7.65	0.28	418	0.5649	<0.0001	0.34	0.285	0.37	0.478 ± 0.10
P4w [*]	8.52	0.37	454	−0.0675	<0.0001	0.42	0.303	0.27	0.608 ± 0.12
M1l [*]	10.66	0.37	470	−0.1161	<0.0001	0.47	0.379	0.15	0.751 ± 0.12
M1mw	8.38	0.30	458	0.5261	<0.0001	0.56	0.221	0.22	0.722 ± 0.11
M1dw	7.89	0.27	454	0.3962	<0.0001	0.62	0.206	0.17	0.786 ± 0.12
M2l	12.55	0.69	539	0.6799	<0.0001	0.44	0.479	0.08	0.847 ± 0.10
M2mw	9.90	0.45	539	0.7125	<0.0001	0.49	0.276	0.23	0.676 ± 0.11
M2dw [*]	8.92	0.39	530	0.2218	<0.0001	0.39	0.302	0.31	0.557 ± 0.11
M3l [*]	12.49	0.87	234	0.8855	0.07	0.13	0.432	0.44	0.231 ± 0.19
M3mw [*]	9.98	0.61	440	0.3233	0.002	0.15	0.373	0.48	0.234 ± 0.11

[a] Total c^2 = amount of phenotypic variance attributable to covariates. Total h^2 = (Residual h^2)(1 − Total c^2), Total e^2 = [1 − (Total c^2 + Total h^2)]

[*]
values were I-normalized to reduce kurtosis. Kurtosis values reported are for the i-normalized trait. All data are presented in mm but were analyzed as multiples of 10 to raise the variance above 1.0