AMERICAN JOURNAL OF PHYSICAL ANTHROPOLOGY 131:420–427 (2006)

Evolution of Genetically Correlated Traits: Tooth Size and Body Size in Baboons

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KEY WORDS primate evolution; pleiotropy; quantitative genetics; *Papio hamadryas*; dental variation

ABSTRACT Within a population, only phenotypic variation that is influenced by genes will respond to selection. Genes with pleiotropic effects are known to influence numerous traits, complicating our understanding of their evolution through time. Here we use quantitative genetic analyses to identify and estimate the shared genetic effects between molar size and trunk length in a pedigreed, breeding population of baboons housed at the Southwest National Primate Research Center. While crown area has a genetic correlation with trunk length, specific linear measurements yield different results. We find that variation in molar buccolingual width and trunk length is influenced by overlapping additive genetic effects. In contrast, mesiodistal molar length appears to be genetically independent of body size. This is the first study to demonstrate a significant genetic correlation between tooth size and body size in primates. The evolutionary implications are discussed. Am J Phys Anthropol 131:420–427, 2006.

While patterns of population-level variation are influenced by both genotype and environment, natural selection differentiates only on phenotypic variation that has a heritable component. The nature and magnitude of the genetic contribution(s) to the phenotypic variance in a trait influence to a large extent the response of that trait to selection (Fisher, 1930; Lynch and Walsh, 1998). Therefore, understanding the genetic architecture of population-level variation in a phenotype can facilitate our interpretation of its possible evolutionary significance.

Our ability to accurately interpret morphological evolution is complicated by the fact that there is not a one-to-one relationship between genotype and phenotype (Lewontin, 1974). Ignoring, for the purposes of this discussion, the effects of gene-gene interactions (epistasis) and gene-environment interactions, multiple genes can influence individual traits (polygeny), and individual genes or suites of genes can influence variation in more than one trait (i.e., genetic correlation due to pleiotropy and linkage disequilibrium). Here we focus on the latter, genetic correlation.

Studies of the evolution of phenotypic variation may be complicated if genetic correlation between characters is either unrecognized or not taken into account. This is because selection on one trait can concomitantly alter another trait, even if the two traits are seemingly functionally independent. Similarly, constraints on one trait (e.g., physical size or functional limitations) may limit the response to selection on the part of a genetically correlated, but functionally independent, character. Tooth size and body size in mammals represents one example of a trait pair whose patterns of phenotypic and genetic correlation are of great interest to evolutionary biologists.

Very few quantitative analyses of the relationship between tooth size and body size were conducted prior to the 1970s in taxa other than mice (Gould, 1975). Body size was quantified through various methods, e.g., body mass, height, cranial size, and limb bone length. Since that time, numerous studies were undertaken in an effort to understand the biological factors responsible for this phenotypic correlation within and among species. However, the results are ambiguous, because population-level estimates of tooth size:body size phenotypic correlations typically do not match those estimated in broader taxonomic studies.

For example, phenotypic correlation studies across several mammalian orders, at the family level, or even across several species, find a significant correlation between tooth size and body size (Gingerich, 1977; Goldstein et al., 1978; Creighton, 1980). Because teeth are better preserved than the rest of the skeleton, they are typically the most commonly fossilized elements known for many extinct mammalian taxa. Therefore, paleobiologists have used the high correlations between tooth size and body size as a predictive formula for estimating the body size of extinct taxa (Gingerich, 1977; Bown et al., 1994; Ciochon et al., 2001).

Grant sponsor: National Science Foundation; Grant number: BCS-0130277 and 0500179; Grant sponsor: Research Board, University of Illinois at Urbana-Champaign; Grant sponsor: National Institutes of Health/National Center for Research Resources; Grant number: P51 RR013986.

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Received 16 May 2005; accepted 24 January 2006.

DOI 10.1002/ajpa.20435
Published online 4 April 2006 in Wiley InterScience (www.interscience.wiley.com).

However, population-level studies of the relationship between tooth size and body size do not always reveal such strong correlations. Phenotypic correlation studies performed on modern human populations typically find weak or no correlation between tooth size and body size (Garn et al., 1968; Henderson and Corruccini, 1976; Anderson et al., 1977; Siegel and Gest, 1980; Kieser and Groeneveld, 1990; Lease and Harris, 2001), unless adult male and female samples are pooled without correcting for sexual size dimorphism (Anderson et al., 1977; Siegel and Gest, 1980; Perzigian, 1981; Kieser and Groeneveld, 1990). Given that higher correlations are found in primate species with significant levels of sexual dimorphism (Martin, 1971; Lauer, 1975; Swindler and Sirianni, 1975; Lavelle, 1977; Johnson, 1978), these correlations are likely an artifact of the disparity between males and females (calculating correlations from mixed samples) rather than evidence of a general pattern of covariance (Wood, 1979).

Differing allometric relationships also complicate tooth size:body size correlations. Most mammalian, including primate, postcanine teeth tend to have a negative allometric relationship with body size (Pilbeam and Gould, 1974; Gould, 1975; Wood, 1979; Creighton, 1980; Gingerich et al., 1982), whereas canine teeth are positively allometric (Wood, 1979; Leutenegger, 1982). Not all mammalian taxa conform to this relationship (Legendre and Roth, 1988), and they often deviate by dietary group (Kay, 1975; Goldstein et al., 1978). Dental traits have even more complex patterns of allometric relationships with each other (Kurtén, 1967).

Despite the low phenotypic correlations, pedigree analyses of human populations show that body size dimorphism between brother-sister pairs is related to the magnitude of tooth size dimorphism (Garn et al., 1967), and stature in one generation is related to dental crown size in the next (Garn et al., 1968). These studies suggest that tooth size and body size are influenced to some degree by the same genetic factors.

Because phenotypic correlations are complicated by factors such as sexual dimorphism, individual nutrition levels, and varying allometric relationships, analytical methods that can account for these effects will better elucidate the genetic correlation between tooth size and body size. Understanding the genetic relationship between these two traits has significant implications for evolutionary, paleontological, and neontological mammalian research.

In a study of brain size:body size allometry, Lande (1979) showed that evolutionary correlations (those found across multiple taxonomic groups) are better predicted by genetic rather than phenotypic correlations and can account for the lower intraspecific vs. interspecific phenotypic correlations. If the genotypic correlation is higher than the phenotypic correlation, then selection operating on body size can account for the correlated response in brain size in closely related species or subspecies, despite the absence of a phenotypic correlation within a population (Lande, 1979).

Quantitative genetic approaches enable both genetic and nongenetic correlations to be estimated. To date, these types of genetic analyses of the tooth size:body size relationship have been performed only with data from inbred mice. These analyses show that first, second, and third molar widths are genetically correlated with each other (Bader, 1965a,b; Wallace, 1968; Leamy and Touchberry, 1974). Molar width in mice also is genetically correlated with body mass (Leamy, 1985). These genetic correlations are higher than the phenotypic correlations, but the difference is not statistically significant (Leamy, 1985). Therefore, these data do not support the hypothesis that the genetic correlation is higher than the phenotypic correlation, and therefore cannot be called upon to explain the higher levels of interspecific correlation. However, Leamy (1985) pointed out that statistical power is difficult to achieve for these types of analyses. The lack of a significant result in the analysis of Leamy (1985) may therefore be more an absence of evidence than an evidence of absence. Similarly, the prediction by Lande (1979) of the disagreement between inter- and intraspecific phenotypic correlations has yet to be demonstrated for the tooth size:body size relationship.

The purpose of this paper is to present a quantitative genetic analysis of tooth size and body size in a population of captive, pedigreed baboons. Specifically, this study addresses the hypothesis that despite a low or nonsignificant phenotypic correlation between molar size and body size, a significant genetic correlation exists. We analyze a two-dimensional approximation of molar crown occlusal-view area and three linear measurements of molar size (mesiodistal length, mesial buccolingual width, and distal buccolingual width) to determine whether or not body size variation is genetically correlated with molar size, or some component of it.

MATERIALS AND METHODS

Data were collected from a large captive, pedigreed breeding colony of baboons (*Papio hamadryas*) housed at the Southwest National Primate Research Center (SNPRC, San Antonio, TX). The colony is maintained in pedigrees (all familial relationships known) with all matings controlled, and a female-to-male sex ratio approximating 2:1.

Genetic management of the colony was started over 20 years ago, and allows for data collection from noninbred animals. All nonfounder animals in this study resulted from matings that were random with respect to dental, skeletal, and developmental phenotype. Genetic marker maps were constructed using data obtained from approximately 1,000 individuals (Rogers et al., 2000).

All pedigree data management and preparations were conducted using the computer package PEDSYS (Dyke, 1996). The animals from which data were collected are distributed across 11 extended pedigrees. The mean number of animals with data per pedigree was 44, and these individuals typically occupied the lower, most recent two or three generations of each pedigree.

Odontometric data were collected from high-resolution plaster dental casts of 630 pedigreed, genotyped baboons. Dental casts were collected following a protocol described in detail elsewhere (Hlusko et al., 2002). Mesiodistal length, mesial buccolingual width, and distal buccolingual width for all 12 maxillary and mandibular molars were measured from digital images of each molar (Fig. 1). Data were not collected from broken or unusually or excessively worn specimens.

A two-dimensional approximation of molar crown size in occlusal view was calculated as a trapezoid: Area = $1/2(b_1 + b_2)h$, where b_1 = mesial buccolingual width, b_2 = distal buccolingual width, and h = mesiodistal length. Previous analyses showed that such an approximation is a good proxy for the actual two-dimensional molar crown area (Hlusko et al., 2002).

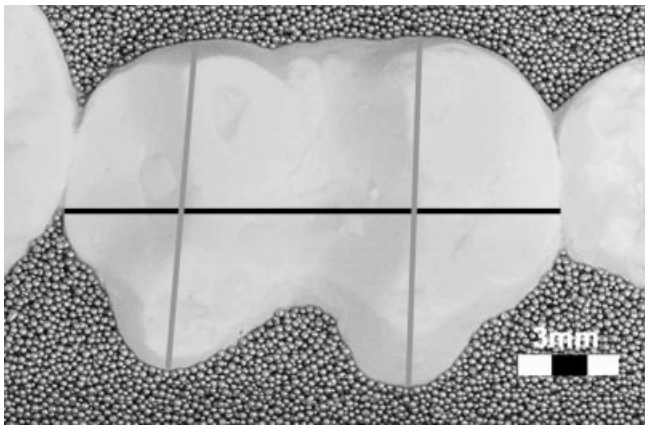

Fig. 1. Photograph of right second mandibular molar (RM_2). Mesial is at right, lingual is at top. Black line indicates mesiodistal length. Grey lines indicate two buccolingual widths measured: mesial and distal. Scale bar, 3 mm.

As a measurement of body size, we used a linear measure of trunk length, obtained using a flexible inelastic tape measure (a standard tape used in anthropometric data collection), as the distance in centimeters along the dorsum from a point on the posterior base of the skull to the caudal margin of the sacrum. The cranial and caudal points were located by palpation. Mass was measured at the same time, using a standard veterinary scale. These data were collected from animals ranging in age from 4.6–30 years.

The Institutional Animal Care and Use Committee, in accordance with established guidelines (National Research Council, 1996), approved all procedures related to the treatment of the baboons during this study.

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). In this study, the phenotypic covariance for each trait within a pedigree is modeled as $O = 2\Phi\sigma_G^2 + I\sigma_E^2$ 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. Because the components of the phenotypic variance are additive, such that $\sigma_P^2 = \sigma_G^2 + \sigma_E^2$ we estimated heritability, or the proportion of phenotypic variance attributable to additive genetic effects, as $h^2 = \sigma_G^2/\sigma_P^2$. Phenotypic variance attributable to nongenetic factors was estimated as $e^2 = 1 - h^2$. We estimated the mean effects of sex, age, body mass, and trunk length and percent subspecies admixture (i.e., as the proportion of *P. h. cynocephalus* relative to *P. h. anubis* genes) on the linear measurements recorded for each molar. Covariates found to be significant in the univariate analyses were also included in the bivariate analyses.

We tested for the effects of these covariates in two ways. First, we used likelihood ratio tests to compare the likelihoods of models in which the value of each one of these covariates was constrained to be zero to that of the general model in which all covariate effects were estimated. For the purposes of these analyses, $P \leq 0.10$ indicated a significant mean effect of the covariate. Second, because the preceding approach can occasionally fail to detect the effects of covariates in the presence of multicollinearity, we also used a Bayesian model averaging method implemented in SOLAR. With this method, we computed a statistic called the Bayesian inference criterion (BIC) for genetic models containing each possible combination (or set) of those potential covariates from the set above, and then performed additional statistical tests on the models having the highest BICs and the elements they contain (Blangero et al., 1999, 2005).

Using extensions to univariate genetic analysis that encompass the multivariate state (Hopper and Mathews, 1982; Lange and Boehnke, 1983; Boehnke et al., 1987), we followed an approach described in detail elsewhere (Mahaney et al., 1995) to model 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 population, the covariates and their regression coefficients, and the additive genetic values and random environmental deviations, as well as the genetic and environmental correlations between them. From the multivariate model of an individual's phenotype, 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.

The genetic and environmental components of the phenotypic correlation matrix are additive, like those of the corresponding variance-covariance matrix, so we could use the maximum likelihood estimates of the additive genetic and environmental correlations to obtain the total phenotypic correlation between two traits, ρ_P, as

$$\rho_P = \sqrt{h_1^2}\sqrt{h_2^2}\,\rho_G + \sqrt{(1-h_1^2)}\sqrt{(1-h_2^2)}\,\rho_E.$$

Significance of the maximum likelihood estimates for heritability and other parameters was assessed by means of likelihood ratio tests. Twice the difference of the maximum likelihoods of a general model (in which all parameters were estimated) and a restricted model (in which the value of a parameter to be tested was held constant at some value, usually zero) were compared. This difference was 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 were equal to the difference in number of estimated parameters in the two models (Boehnke et al., 1987). However, in tests of parameters like h^2, whose values may be fixed at a boundary of their parameter space in the null model, the appropriate significance level was obtained by halving the *P*-value (Boehnke et al., 1987).

For bivariate models in which genetic correlations were found to be significantly greater than zero, additional tests were performed to compare the likelihood of a model in which the value of the genetic correlation is fixed at 1.00 or zero to that of the unrestricted model in which the value of the genetic correlation is estimated.

TABLE 1. Quantitative genetics of permanent second molar (M2) crown metric traits in pedigreed baboons: maximum likelihood parameter estimates for mean, standard deviation, mean effects of significant ($P < 0.10$) covariates, and variance components[1]

Parameter	XLM2ar	XLM2l	XLM2mw	XLM2dw	XRM2ar	XRM2l	XRM2mw	XRM2dw
n	452	539	539	530	445	531	530	517
μ	132.60	13.3	10.4	9.5	130.21	13.2	10.5	9.4
σ	10.53	6.05	5.58	5.31	10.59	6.43	5.73	5.33
β *age*	−0.3897	−0.1680	−0.2293	−0.0524	−0.0891			
P	0.0008	0.02	0.002	0.09	0.0006			
β *sex*	−18.27	−10.24	−5.95	−7.47	−17.28	−10.55	−8.04	−7.23
P	<0.00001	<0.00001	<0.00001	0.00002	<0.00001	<0.00001	<0.00001	0.00001
β *mass*	0.3532	0.1909	0.1658		0.2324	0.1369		
P	0.004	0.0006	0.04		0.003	0.007		
β *trunk*					17.78			
P					<0.00001			
β *admix*	−0.0573				−0.0851			
P	0.08				0.09			
c^2	0.52	0.49	0.31	0.31	0.50	0.41	0.30	0.28
h^2_r	0.84 ± 0.11	0.86 ± 0.09	0.58 ± 0.11	0.56 ± 0.11	0.63 ± 0.13	0.73 ± 0.11	0.49 ± 0.11	0.48 ± 0.12
P	<0.00001	<0.00001	<0.00001	<0.00001	<0.00001	<0.00001	<0.00001	<0.00001
h^2 *total*	0.40	0.44	0.40	0.39	0.315	0.43	0.34	0.35
Parameter	DLM2ar	DLM2l	DLM2mw	DLM2dw	DRM2ar	DRM2l	DRM2mw	DRM2dw
n	396	485	480	471	395	490	481	475
μ	127.56	13.2	9.9	9.2	123.58	13.1	9.9	9.1
σ	10.47	5.78	5.79	5.64	10.00	5.59	5.59	5.11
β *age*								
P								
β *sex*	−22.70	−12.07	−8.58	−7.95	−16.70	−11.27	−8.66	−5.60
P	<0.00001	<0.00001	<0.00001	<0.00001	<0.00001	<0.00001	0.00003	0.00007
β *mass*						0.1188		
P						0.05		
β *trunk*								0.1787
P								0.03
β *admix*	−0.0540				−0.0728			
P	0.06				0.02			
c^2	0.50	0.48	0.32	0.29	0.51	0.50	0.31	0.34
h^2_r	0.67 ± 0.14	0.67 ± 0.11	0.51 ± 0.11	0.43 ± 0.11	0.75 ± 0.14	0.82 ± 0.10	0.76 ± 0.10	0.59 ± 0.12
P	<0.00001	<0.00001	<0.00001	<0.00001	<0.00001	<0.00001	<0.00001	<0.00001
h^2 *total*	0.34	0.35	0.35	0.31	0.37	0.41	0.52	0.39

[1] Measurements are in mm. D, mandibular; X, maxillary; L, left; R, right; l, crown length (mesiodistal diameter); mw, mesial crown width (mesial buccolingual diameter); dw, distal crown width (distal buccolingual diameter); h^2_r, proportion of residual phenotypic variance attributable to additive effects of genes ± standard error; h^2 *total*, proportion of total phenotypic variance attributable to additive effects of genes; *P*, *P*-value; c^2, proportion of total phenotypic variance attributable to the effects of significant covariates.

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.

RESULTS

Analyses were performed for all maxillary and mandibular molars. The second molar samples were the largest (n = 395–539) and returned the most precise point estimates, although the first (n = 326–470) and third (n = 232–530) molar analyses yielded similar results. In an effort to conserve space, we present only the second molar results in tabular form.

Table 1 shows the maximum likelihood parameter estimates for the heritability and significant covariate effects for all size measurements (area, mesiodistal length, and mesial and distal buccolingual widths). All measurements yielded significant ($P < 0.0001$) residual heritability estimates that are moderate to high, h^2_r = 0.43–0.86.

Covariates account for 28–52% of the total phenotypic variance. Of the potential covariates we tested, age is significant for all four left secondary maxillary molar (LM^2) measurements and the right secondary maxillary molar (RM^2) approximated area. Because teeth are altered only by wear and breakage after eruption, age acts as a proxy for wear in these analyses. Sex is a significant covariate for all measurements, in accordance with the known sexual dimorphism of baboon molar size (Swindler, 2002). Mass exerts a significant mean effect on six of the molar size measurements. Trunk length is a significant covariate in only two analyses, i.e., that of RM_2 distal width ($P = 0.03$) and RM^2 area ($P < 0.001$). Percent *P. h. cynocephalus* admixture was identified as a significant covariate only for the four approximated areas.

Random environmental effects (i.e., those due to nongenetic factors such as measurement error and unaccounted for environmental influences) contribute 7–40% (average = 0.22) to the total phenotypic variance.

Analyses of the first and third molar measurements yield similar results (not shown), with moderate to high residual heritability estimates. Covariate effects account for 17–51% of the total phenotypic variance, with age contributing significantly to three of the measurements, mass to one measurement, and trunk length to one measurement. Just as we observed in our analyses of second molar metrics, sex significantly influences all first and third molar crown measurements in these baboons. Only

TABLE 2. Permanent second molar crown metrics (DM) and trunk length in pedigreed baboons: maximum likelihood estimates of genetic and environmental correlations, likelihood ratio test results, and derived phenotypic correlations (accounting for nonindependence of data from related subjects)[1]

DM	h^2_{DM}	h^2_{Trunk}	ρ_G	$P\ (\rho_G = 0)$	$P\ (\lvert\rho_G\rvert = 1)$	ρ_E	$P\ (\rho_E = 0)$	ρ_P
Maxillary								
LM ar	0.87	0.66	0.44 ± 0.12	0.001	<0.001	−1.0	0.002	0.123
LM l	0.81	0.73	0.08 ± 0.12	0.50	<0.001	−0.23 ± 0.28	0.41	0.068
LM mw	0.61	0.78	0.48 ± 0.12	<0.001	<0.001	−0.75 ± 0.29	0.001	0.313
LM dw	0.56	0.75	0.51 ± 0.13	<0.001	<0.001	−0.58 ± 0.24	0.004	0.309
RM ar	0.67	0.66	0.45 ± 0.15	0.005	<0.001	−0.45 ± 0.25	0.04	0.144
RM l	0.71	0.74	0.07 ± 0.13	0.58	<0.001	−0.09 ± 0.24	0.70	0.055
RM mw	0.55	0.77	0.40 ± 0.13	0.004	<0.001	−0.41 ± 0.24	0.05	0.242
RM dw	0.49	0.74	0.30 ± 0.14	0.04	<0.001	−0.20 ± 0.20	0.28	0.168
Mandibular								
LM ar	0.69	0.67	0.65 ± 0.15	<0.001	0.006	−0.91 ± 0.32	<0.001	0.152
LM l	0.69	0.75	0.29 ± 0.13	0.03	<0.001	−0.47 ± 0.28	0.06	0.021
LM mw	0.55	0.76	0.45 ± 0.14	0.002	<0.001	−0.66 ± 0.26	0.003	0.266
LM dw	0.45	0.79	0.53 ± 0.15	0.001	<0.001	−0.61 ± 0.25	0.004	0.255
RM ar	0.79	0.64	0.58 ± 0.14	<0.001	<0.001	−0.78 ± 0.34	0.004	0.198
RM l	0.81	0.73	0.13 ± 0.13	0.32	<0.001	−0.29 ± 0.28	0.31	0.108
RM mw	0.77	0.73	0.23 ± 0.12	0.02	<0.001	−0.36 ± 0.28	0.17	0.186
RM dw	0.44	0.77	0.53 ± 0.15	0.001	<0.001	−0.61 ± 0.25	0.004	0.259

[1] L, left; R, right; ar, approximated area, see text for details; l, crown length (mesiodistal diameter); mw, mesial crown width (mesial buccolingual diameter); dw, distal crown width (distal buccolingual diameter); ρ_G, genetic correlation; ρ_E, nongenetic correlation; ρ_P, phenotypic correlation.

the heritability estimate for LM_3 mesiodistal length (n = 323) is not significant.

Results of bivariate analyses performed between trunk length and all second molar size measurements are shown in Table 2. Phenotypic correlations are found to be low, accounting for 3–10% of the variance in trait pairs. Environmental/nongenetic correlations are negative, with 9 of 12 estimates being significantly different from zero ($P < 0.05$).

Molar crown area and trunk length were found to be genetically correlated (ρ_G = 0.44–0.65), demonstrating that 19–24% of the additive genetic effects are shared between second molar crown size and trunk length.

When considering linear measurements of crown area, we find that all genetic correlations between molar crown width and trunk length are significant ($P < 0.05$), and range between 0.23–0.53, indicating that 5–28% of the additive genetic effects are shared by the trait pairs. For 5 of the 8 analyses pairing a buccolingual width with trunk length, more than 20% of the additive genetic covariance is due to the effects of the same gene or suite of genes.

In contrast to the buccolingual width and trunk length analyses, only one of the four genetic correlations between trunk length and molar crown mesiodistal length (LM_2) is significantly different from zero.

Bivariate quantitative genetic analyses of the same measurements from the first and third molars returned similar but less precise estimates. This is an expected result, given the smaller sample sizes for these teeth (results not shown).

Bivariate analyses between all size measurements and body mass yielded low and statistically insignificant genetic correlations.

DISCUSSION

Previous investigations of tooth size:body size relationships showed that the significant phenotypic correlations seen across broad taxonomic groups are not similarly revealed at the population level. Here, we also find low to insignificant phenotypic correlations between trunk length and molar size in this pedigreed baboon colony. However, quantitative genetic analyses revealed significant genetic correlations that were higher than their corresponding phenotypic correlations.

This finding accords with the study by Lande (1979) of brain size:body size that similarly yielded low intraspecific phenotypic correlations and higher interspecific phenotypic correlations. As is well-known, genetic correlations can exist between traits that have no phenotypic correlation (Lande, 1979). If genetic correlations are higher than phenotypic correlations, then covariance through evolutionary time and across diverse taxonomic groups would be expected, despite the apparent lack of correlation at the population level when natural selection acts on body size. Here we document relationships between traits consistent with this phenomenon for the first time in primates.

It is evident that adult trunk length is not entirely or directly determinative of tooth size, or vice versa. This is because tooth size and trunk length do not experience all the same influences on growth, i.e., first molar crowns begin to calcify before birth, and are fully formed by age 10 months; second molar crowns start mineralizing at about 1.1 years, and are complete by about 2 years (Swindler and Meekins, 1991); and adult trunk length is not attained until the animal is approximately 6 years old (Leigh and Bernstein, 2006). The correlation between tooth size and body size is probably attributable more to latent rather than specific genetic factors. The various nongenetic influences on growth and development, time differences in achieving adult states, and differences in the mean sizes of teeth and body dimensions in the two sexes probably contribute to a diminished phenotypic correlation, despite the existence of a genetic correlation.

If the genetic correlation between molar size and adult linear measures like trunk length can be "overwhelmed" by nongenetic influences on growth and development in baboons and related species with relatively prolonged ontogenetic periods, then phenotypes that reach their

ultimate morphologies at the same time ontogenetically may be expected to have higher phenotypic correlations. Obviously, this proposition remains to be tested. However, a study of the relationship between dental metrics and a humerofemoral measurement of body size in macaques found a higher correlation between the later developing third molars and body size, which Lauer (1975) tentatively attributed to exposure to the same environmental stresses at the final stages of long bone formation.

The mechanisms underlying population-level variation were demonstrated to be directly relevant to diversity at higher taxonomic levels, and are therefore useful for addressing evolutionary questions (Jernvall, 2000; Stern, 2000; Shubin, 2002; Gompel and Carroll, 2003; Kopp et al., 2003). For the degree of genetic correlation estimated for brain size and body size in mice, Lande (1979) showed that short-term evolutionary differentiation probably results from selection on body size with concomitant changes in brain size, whereas long-term diversification may result from selection acting more strongly on brain size.

The identification and quantification of genetic correlation between tooth size and body size have important implications for understanding the joint evolution of these characters. Molar crown area has a significant and high genetic correlation with trunk length. Perhaps more intriguing, though, is that when the components of this area estimation are analyzed separately, molar length and width are found to have different genetic correlations with trunk length. Our results show that approximately 10% of the total phenotypic variance in baboon molar *width* is attributable to genetic factors that also influence trunk length. In contrast, molar crown mesiodistal *length* is not genetically correlated with this measure of body size. This lack of a genetic correlation may explain the lack of a phenotypic correlation between body mass and tooth length in *Papio hamadryas anubis* reported in an earlier study (Siegel and Gest, 1980), although this is not the only possible explanation.

If these genetic differences between body size and molar crown length vs. width are found to be consistent across other taxa, we predict that molar crown mesiodistal length may show more variance between synchronic and diachronic populations than does buccolingual width when body size does not change. It is fairly common for mammalian lineages to increase molar crown length through time, as seen in the third molars of the Suidae (Harris and White, 1979) and *Theropithecus* baboons (Jablonski, 1993). However, width does not follow the same general trend. This may be due to constraints placed on molar width by jaw size and its functional interrelatedness with overall cranial size, but our results suggest that it may also result from a lack of genetic correlation between molar length and trunk length. Additional research is needed to determine whether or not the genetic architecture observed in the SNPRC baboons is characteristic of other taxa, and thereby informative of such evolutionary phenomena. As a caveat, however, it is important to note that simulation studies showed that a genetic correlation is not necessarily a good predictor of a correlated response to selection (Gromko, 1995).

As an interesting aside, in dwarf marsupial lineages from the Late Quaternary, Marshall and Corruccini (1978) found that tooth width is reduced more rapidly than length. This observation is consistent with our results for the SNPRC baboons, and may possibly result from selection for smaller body size.

Previous studies found a range of allometric relationships between molar crown area and body size. Different genetic architectures underlying molar length and width, as seen here, may explain some of this variation. Additionally, allometry within the dentition should be reconsidered in light of our results.

Our analyses do not address the relationships between body size and the size of the primary dentition. Altmann (1998) showed that juvenile baboons undergo intense selective pressure on their ability to obtain and consume certain foodstuffs. The limited odontological data available suggest that the phenotypic correlation between primary and permanent tooth size is low (Moorrees et al., 1957; Moorrees and Chadha, 1962; Steigman et al., 1982; Yuen et al., 1996; Lease, unpublished data). However, further research is needed to characterize the pleiotropic relationships between juvenile body size and primary tooth size, and between primary and permanent tooth size, because yet-unidentified genetic correlations would have significant implications for our understanding of the evolution of these features.

CONCLUSIONS

Here we present a series of bivariate quantitative genetic analyses employed to test for genetic correlations between molar crown size (an approximation of the two-dimensional occlusal-view area, mesiodistal length, and mesial and distal buccolingual widths) and trunk length (or sitting height) within a population of captive pedigreed breeding baboons housed at the Southwest National Primate Research Center. Our analyses demonstrate that while molar crown area is genetically correlated with trunk length, the linear components of this area estimate yield different results when analyzed separately. Molar buccolingual width is significantly correlated with crown-rump length. In contrast, we find no genetic correlation between crown-rump length and molar mesiodistal length. Further analyses in other populations are needed to determine how ubiquitous this genetic architecture may be within mammals. If additional studies demonstrate a similar pattern of genetic correlations, then these genetic correlations may have significantly and differentially influenced the evolution of molar crown size and shape.

ACKNOWLEDGMENTS

We thank K.D. Carey, K. Rice, and the Veterinary Staff of the Southwest Foundation for Biomedical Research and the Southwest National Primate Research Center; J. Cheverud (Washington University) for access to specimens; D.E. Newman for pedigree data management and processing; J. Rogers, A. Walker, and K. Weiss for project support and development; and L. Buchanan, T. Cannistraro, L. Holder, J. Irwin, A. Liberatore, M.L. Maas, and D. Pillie for assistance with data collection. We also thank A.G. Comuzzie for access to body size data. Thanks also go to R. Lande and three anonymous reviewers for helpful comments on the manuscript. This material is based on work supported by the National Science Foundation under award BCS-0130277 and 0500179 and the University of Illinois at Urbana-Champaign Research Board. National Institutes of Health/National Center for Research Resources P51 RR013986 supports the Southwest National Primate Research Center.

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