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Sexual dimorphism in primate aerobic capacity: a phylogenetic test

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Abstract

Male intrasexual competition should favour increased male physical prowess. This should in turn result in greater aerobic capacity in males than in females (i.e. sexual dimorphism) and a correlation between sexual dimorphism in aerobic capacity and the strength of sexual selection among species. However, physiological scaling laws predict that aerobic capacity should be lower per unit body mass in larger than in smaller animals, potentially reducing or reversing the sex difference and its association with measures of sexual selection. We used measures of haematocrit and red blood cell (RBC) counts from 45 species of primates to test four predictions related to sexual selection and body mass: (i) on average, males should have higher aerobic capacity than females, (ii) aerobic capacity should be higher in adult than juvenile males, (iii) aerobic capacity should increase with increasing sexual selection, but also that (iv) measures of aerobic capacity should co-vary negatively with body mass. For the first two predictions, we used a phylogenetic paired *t*-test developed for this study. We found support for predictions (i) and (ii). For prediction (iii), however, we found a negative correlation between the degree of sexual selection and aerobic capacity, which was opposite to our prediction. Prediction (iv) was generally supported. We also investigated whether substrate use, basal metabolic rate and agility influenced physiological measures of oxygen transport, but we found only weak evidence for a correlation between RBC count and agility.

Introduction

Male and female mammals differ in many fundamental ways related to basic reproductive features. Female investment in offspring necessitates the metabolically expensive production of ova, a long gestation period and an often even more costly period of lactation, whereas in many mammalian species, males donate only sperm and seminal fluid as a contribution to their offspring. These differences in parental investment have resulted in further sexual differentiation (known as sexual dimorphism) through the process of sexual selection (Darwin, 1871; Trivers, 1972; Clutton-Brock & Parker, 1992; Andersson, 1994; but see Kokko & Jennions, 2008). Sexual dimorphism has long attracted the attention of biologists, leading to studies of mate choice, male intrasexual selection and sexual coercion (e.g. Hamilton & Zuk, 1982; Smuts & Smuts, 1993; Andersson, 1994; Eberhard, 1996; Lindenfors & Tullberg, 1998; Lindenfors, 2002; Thorén *et al.*, 2006; Fairbairn *et al.*, 2007).

Although we know much about the behavioural and morphological characters produced by sexual selection, we know less about the underlying physiological differences between the sexes. Understanding these physiological differences could provide insights to the mechanisms that drive sexual selection. As a step towards addressing these questions, we investigated whether male and female nonhuman primates differ in measures of aerobically crucial components of their blood. Specifically, we analysed haematocrit and red blood cell (RBC)

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counts, which are known to play a central role in oxygen transport (Widmaier *et al.*, 2008). Males have been reported to have higher RBC counts and haematocrit than females in humans and some other mammals, although in other species these measures are sexually monomorphic (Glucksman, 1978). In primates, intrasexual competition among males for access to resources and mates can be intense in some species (e.g. Kappeler & van Schaik, 2004), which leads to us to predict that, on average, males should have higher RBC counts and haematocrit proportions than females of the same species because of sexual selection acting directly on aerobic capacity. Furthermore, as male competition generally occurs among adult males rather than among juveniles, we also predicted that adult males would have higher values of these measures than juvenile male conspecifics (but see Dejours, 1975).

We further tested the hypothesis that sexual selection can account for sexual differences in physiological metrics of aerobic capacity by investigating whether measures of aerobic capacity co-varied with the degree of sexual selection. Intrasexual physical competition between males over access to females should increase the need for aerobic capacity in males through the demands imposed by physical prowess. Thus, we expected to find a positive correlation between the degree of sexual selection and haematological variables important for aerobic capacity.

Other factors may also influence patterns of RBC counts and haematocrit. In particular, previous research has demonstrated negative correlations between overall body mass and RBC counts for both mammals (Preston *et al.*, 2009) and birds (Bennett & Hawkey, 1988). A negative correlation between the size and number of cells also has been reported (Bennett & Hawkey, 1988; Hawkey *et al.*, 1991). Thus, we also investigated whether body mass can account for variation in haematological traits in primates. Specifically, we predicted that larger-bodied species will have lower RBC counts and lower haematocrit, based on the fact that metabolic rate scales to body mass with an exponent less than one (i.e. Kleiber's law, Kleiber, 1961; Schmidt-Nielsen, 1975, 1990; Calder, 1984), whereas total blood volume scales approximately linearly with body mass (Stahl, 1967; West *et al.*, 1997). Thus, per unit of body mass, a smaller-bodied animal has higher metabolic requirements than a larger one, producing a negative relationship between body mass and haematological traits related to aerobic capacity. Metabolic scaling rules therefore also predict that juveniles and females, which are smaller than males, should have higher RBC counts and haematocrit than males. Note, however, that this latter prediction is opposite to the prediction derived from the sexual selection hypothesis for increased male physical prowess.

In addition to body mass, we investigated other factors that may influence RBC counts and haematocrit, including substrate use, basal metabolic rate and agility. Because of potentially higher energy usage, we predicted that aerobic capacity increases with greater use of arboreal substrates, increased metabolic rate and greater agility.

Materials and methods

Data collection

We gathered data on haematological traits for 45 primate species from the International Species Information System (ISIS, Minnesota Zoological Garden, Apple Valley, MN, USA). These samples were obtained by ISIS, which is an international zoo and aquarium organization, from healthy zoo animals for the purpose of constructing physiological references values (i.e. normal ranges) for different species. For each species, mean values are provided by sex and age classes, along with data on total sample size, number of animals sampled, standard deviation and ranges of variation.

In the analyses presented here, we focused on two widely used physiological correlates of aerobic performance: haematocrit and RBC counts. Combining age and sex classes, the data on RBC come from an average of 300.2 samples per species (range 32–1059) in an average of 22.7 zoological institutions (range 3–75), whereas data on haematocrit come from an average of 365.9 samples (range 49–1242) in an average of 22.7 zoological institutions (range 3–75). Haematocrit and RBC counts are related. Haematocrit (a unitless measure; L/L) is the proportion of a standardized blood sample that contains RBC after centrifugation, whereas RBC count is simply the erythrocyte count $\times 10^{12}$ L^{-1} (Widmaier *et al.*, 2008). However, we do not expect haematocrit and RBC to co-vary perfectly because haematocrit is also influenced by the size of the red blood cells (i.e. individuals with the same number of RBCs might have different haematocrit readings if the cells differ in size). It is well known that physical training increases haematocrit levels in humans (e.g. Faulkner *et al.*, 1967) and that low haematocrit is an indicator of anaemia. Thus, we assume that higher values of haematocrit and RBC counts indicate increased aerobic capacity.

We used sexual dimorphism in body mass as one of our measures of the degree of sexual selection. Sex-specific adult body masses were obtained from Smith & Jungers (1997). These data show that there is large variation in primate body mass dimorphism, with species ranging from monomorphic to extremely dimorphic. We used ratios of male to female values as our measures of sexual dimorphism in body mass, which ranged from 0.90 in the red-handed tamarin (*Saguinus midas*) to 2.45 in the mandrill (*Mandrillus sphinx*). For analyses in which logarithm transformation was required to satisfy distributional assumptions (in particular, the correlation analyses), we $\log_{10}$-transformed all variables prior to

analysis, including the male–female body mass ratio [log(M/F)].

In addition to body mass dimorphism, we also used mating system as a proxy of sexual selection. Here, we classified each species into one of three groups using mating system codes from Lindenfors (2002). From most to least polygynous, these groups were uni-male, multi-male and monogamous. Males of highly polygynous species (uni-male) are expected to be under intense sexual selection, whereas males of monogamous species should experience relatively less sexual selection (Lindenfors & Tullberg, 1998). Although coded discretely, in reality mating system probably varies on a continuum and was thus treated as a continuous character when necessary for subsequent analyses.

We used body mass dimorphism and mating system classification as our proxy measures of the intensity of intrasexual selection. Other measures exist, such as operational sex ratio (Mitani *et al.*,1996), harem size (Lindenfors *et al.*, 2002) or competition levels (Plavcan & van Schaik, 1992; Plavcan *et al.*, 1995; Plavcan, 2004; but see Lindenfors, 2002); however, all of these are available only for a limited subset of the species in our study. Importantly, numerous prior studies (including those cited previously) have consistently found a strong relationship between sexual size dimorphism and the aforementioned more direct aspects of sexual selection.

We also obtained data on substrate use (% terrestriality, unpublished comparative database from C. Nunn; see Nunn & van Schaik, 2002), basal metabolic rate (I. Capellini, pers. comm.) and agility, the latter indicated by both ordered discrete scores and morphological measures (i.e. semi-circular canal radius) from Spoor *et al.* (2007).

We used an updated version of a recent mammal phylogeny (Bininda-Emonds *et al.*, 2007, 2008) to test our evolutionary prediction for the evolution of aerobic capacity while taking phylogenetic information into account. This phylogeny was estimated using the matrix representation with parsimony 'super-tree' method (Baum, 1992; Ragan, 1992). Bininda-Emonds *et al.* (2007, 2008) combined previously published phylogenies based on both molecular and morphological data, thus making it the phylogeny supported by the most comprehensive data set. They estimated branch lengths proportional to time using a combination of molecular and fossil data (Bininda-Emonds *et al.*, 2007). Figure 1 shows the 45 species primate tree used in our analyses.

Phylogenetic paired *t*-test

To test the predictions involving differences in age and sex classes, we developed a phylogenetic paired *t*-test to test for consistent evolutionary differences in metabolic characters across species. A paired *t*-test is appropriate to our study because we are primarily

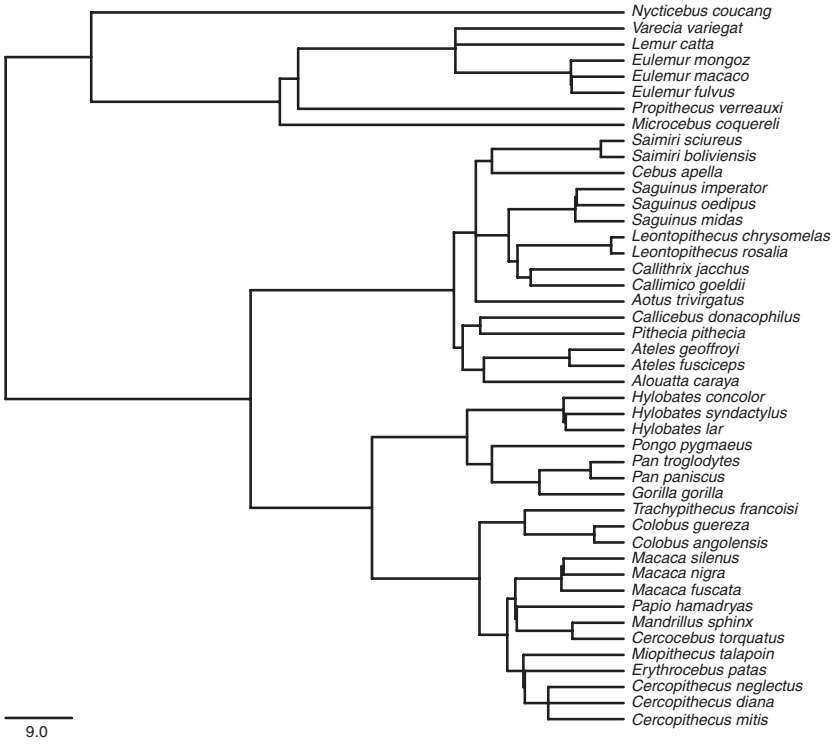

Fig. 1 A phylogenetic tree for the 45 primate species included in this study, derived from Bininda-Emonds *et al.* (2007, 2008). Branch lengths are scaled to be proportional to time in millions of years.

interested not in whether males and females (or adults and juveniles) differ from each other on average among species, but whether they differ in a consistent manner within each species across the primates in our study. An assumption of the standard paired *t*-test is that each calculated difference can be treated as an independent draw from an underlying normally distributed pool of such differences. However, when the differences are calculated for multiple species related by a phylogenetic tree, they are nonindependent because of shared evolutionary history (Felsenstein, 1985; Harvey & Pagel, 1991).

A phylogenetic paired *t*-test can be conducted like a regular *t*-test after incorporating the dependence of species descended according to a bi- or multifurcating phylogenetic history. For each trait, we started by computing the $N \times 1$ vector, $\mathbf{d}$, containing the differences between males and females or adults and juveniles (depending on the specific test) for each metabolic character in each species. We then computed the $N \times N$ matrix, $\mathbf{C}$, containing the heights above the root of the most recent common ancestor of each pair of the $N = 45$ species in the study (e.g. Rohlf, 2001; Revell & Harmon, 2008). (Juvenile samples were in some cases available for only 42 or 43 of the 45 species, so $N = 42$ or $N = 43$ for those comparisons.) This matrix provides the basis for our model of nonindependence among species. Finally, we calculated the $N \times N$ matrix $\mathbf{E}$ that contained the sampling variances of the difference for each species on its diagonal. We estimated the sampling variances for the differences between means by summing the sampling variances (the standard error squared) of each mean because the expected variance of a difference between two independent random variables is just the sum of their variances separately. The standard deviations and sample sizes for each species are available in the ISIS data. When the sample size (n) for an age–sex class of a species was $n = 1$ (as it was in only a very small number of cases), we estimated the standard error for that species as the average standard deviation calculated across all species for which $n > 1$.

Estimating the mean difference between paired observations obtained with sampling error for species related by a phylogenetic tree is a generalized least squares (GLS) estimation problem (Martins & Hansen, 1997; Garland & Ives, 2000; Rohlf, 2001). For this, we need to estimate an $N \times N$ matrix that is proportional to the variance–covariance matrix for our observations in $\mathbf{d}$. Although our data for species are nonindependent because they share common ancestry, we did not want to assume a particular model for phylogenetic signal *a priori* because a difference between two variables that both exhibit statistical dependence that is highly correlated with the phylogeny might not itself exhibit such signal. Thus, to simultaneously estimate the effect of phylogeny and sampling error (following Pagel, 1999b; Freckleton *et al.*, 2002; Ives *et al.*, 2007) along with our phylogenetic generalized mean difference, $\bar{d}$, we maximized the following equation for the log-likelihood:

$$\log(L) = -(\mathbf{d} - \bar{d}\mathbf{1})'[\sigma^2(\mathbf{C}_\lambda + \varepsilon\mathbf{E})]^{-1}(\mathbf{d} - \bar{d}\mathbf{1})/2 - \log(|\sigma^2(\mathbf{C}_\lambda + \varepsilon\mathbf{E})|)/2 - N \cdot \log(2\pi)/2.$$

This equation is based on the multivariate normal distribution. Here, $\mathbf{C}_\lambda$ is an $N \times N$ matrix containing the same diagonal elements as $\mathbf{C}$, but off-diagonals (i.e. $i \neq j$) $C_{\lambda,ij} = \lambda \cdot C_{ij}$, where λ is to be estimated (Freckleton *et al.*, 2002). λ can be considered a measure of the phylogenetic signal, and thus substituting $\mathbf{C}_\lambda$ for $\mathbf{C}$ in phylogenetic analyses incorporates phylogenetic nonindependence only insofar as it exists in our phenotypic data. ε is a scaling parameter for the sampling error matrix, $\mathbf{E}$, also to be estimated using likelihood. The error parameter ε is $\varepsilon = \sigma_\varepsilon^2/\sigma^2$ from Ives *et al.* (2007; and thus $\varepsilon \cdot \sigma^2 = \sigma_\varepsilon^2$). For a given λ and ε, we can obtain the corresponding phylogenetic mean difference, $\bar{d}$, and variance, σ^2, that maximize the likelihood using the following conditional analytic solutions (based on GLS estimating equations; e.g. Rohlf, 2001):

$$\bar{d} = [\mathbf{1}'(\mathbf{C}_\lambda + \varepsilon\mathbf{E})^{-1}\mathbf{1}]^{-1}[\mathbf{1}'(\mathbf{C}_\lambda + \varepsilon\mathbf{E})^{-1}\mathbf{d}] \text{ and}$$

$$\sigma^2 = (\mathbf{d} - \bar{d}\mathbf{1})'(\mathbf{C}_\lambda + \varepsilon\mathbf{E})^{-1}(\mathbf{d} - \bar{d}\mathbf{1})/N.$$

The latter is a maximum likelihood (ML), but not an unbiased, estimator of σ^2, which corresponds to the 'evolutionary rate' for the phenotypic difference (O'Meara *et al.*, 2006). To obtain the unbiased estimator, we will multiply by $N/(N-p)$, where p is the number of estimated parameters. In the full phylogenetic model, estimation of the variance relies on estimating three other parameters (d, λ and ε), so $p = 3$. If fewer parameters are estimated, then p decreases accordingly.

We can next estimate the standard error of the phylogenetic mean difference as $\mathrm{SE}(\bar{d}) = \sqrt{\sigma_u^2[\mathbf{1}'(\mathbf{C}_\lambda+\varepsilon\mathbf{E})^{-1}\mathbf{1}]^{-1}}$, where σ_u^2 is the phylogenetic variance, which we can compute from σ^2 using the bias correction given above. We then compared the *t*-ratio, calculated as:

$$t(\text{d.f.} = N - p) = \frac{\bar{d}}{\mathrm{SE}(\bar{d})},$$

to a *t*-distribution with $N - p$ degrees of freedom. As before, p is the number of parameters that are estimated in the calculation of t. For the full model, these parameters are σ_u^2, λ and ε. As in a standard *t*-test, the calculation of $\bar{d}$ does not consume a degree of freedom (because we are testing the null hypothesis that our observed sample mean difference, $\bar{d}$, is equal to a hypothesized population mean difference, $\bar{d}_0$ – in this case $\bar{d}_0 = 0.0$). The standard paired *t*-test has an assum-

ption of normality of the paired differences, **d**. However, because of nonindependence created by the phylogeny, in our method **d** is only expected to be normally distributed after phylogenetic transformation (e.g. following Butler *et al.*, 2000; Rohlf, 2001).

We provide MATLAB code to implement this procedure in the electronic supporting information.

Simulation test of the phylogenetic method

To test the phylogenetic paired *t*-test (which was developed here to test for consistent age and sex differences across species), we performed 1000 phylogenetic stochastic simulations of two co-evolving characters with an expected $\bar{d} = 0.0$ (i.e. no mean difference), an expected $\lambda = 1.0$ and an expected $\sigma^2_\varepsilon = \varepsilon \cdot \sigma^2$ of 1.0. We then analysed the data using three procedures: the standard paired *t*-test; the phylogenetic *t*-test where λ was optimized but ε was set to zero; and, finally, the full phylogenetic model incorporating error. We performed these simulations on the 45 taxon primate tree used in this study. Example likelihood surfaces for the two numerically optimized parameters, λ and ε, from one such analysis are given in Fig. 2. Table 1 provides a summary of the results from our simulation tests. Some salient features of this table include the lowest absolute value for $\bar{d}$ in the full model (in our simulations, $\bar{d}$ was assigned an expected value of 0.0); the lowest type I error rate (0.067) for the full model; and, finally, (more or less) unbiased parameter estimates for λ and σ^2_ε in the full model (Table 1). Although lowest in the full model, the variance of *t* in simulation ($\mathrm{var}(t) = 1.91$) is still much higher than its expected value of $df/(df - 2) = 1.05$. This seems to be because of the presence of a small number of very large, outlying values for *t*, almost invariably corresponding to very poor maximum likelihood estimation of λ and/or ε. With the 10% most extreme values of *t* excluded, the variance of *t* decreases to 1.17, much closer to its expectation. We hypothesize that the sampling distribution of our maximum likelihood parameter estimates are only asymptotically normal and unbiased, as is common for maximum likelihood estimates (Lynch & Walsh, 1998). To test this, we conducted an additional 1000 simulations on larger ($n = 100$) stochastic phylogenies. We found that the type I error rate of the *t*-test for the full model (0.056) had decreased towards its nominal level of 0.05. We also found that parameter estimation was substantially improved over that obtained from simulations on the small phylogeny (for example, the mean maximum likelihood estimates $\overline{\mathrm{MLE}(\lambda)} = 0.992$ and $\overline{\mathrm{MLE}(\sigma^2_\varepsilon)} = 0.998$; compare to Table 1).

Tests of correlated evolution

We tested for correlated evolution in aerobic capacity, sexual selection, body mass and other ecological and physiological variables. We first tested whether the traits individually showed evidence of phylogenetic signal based on the parameter λ (Pagel, 1999a; Freckleton *et al.*, 2002). As λ was significantly different from 0.0 in all tests, we used phylogeny-based methods to assess the predictions involving correlated trait change. We calculated linear regressions using phylogenetic GLS (Grafen, 1989; Garland & Ives, 2000; Rohlf, 2001; Revell, 2009). For each regression model, we estimated the multivariate λ using maximum likelihood (Freckleton *et al.*, 2002;

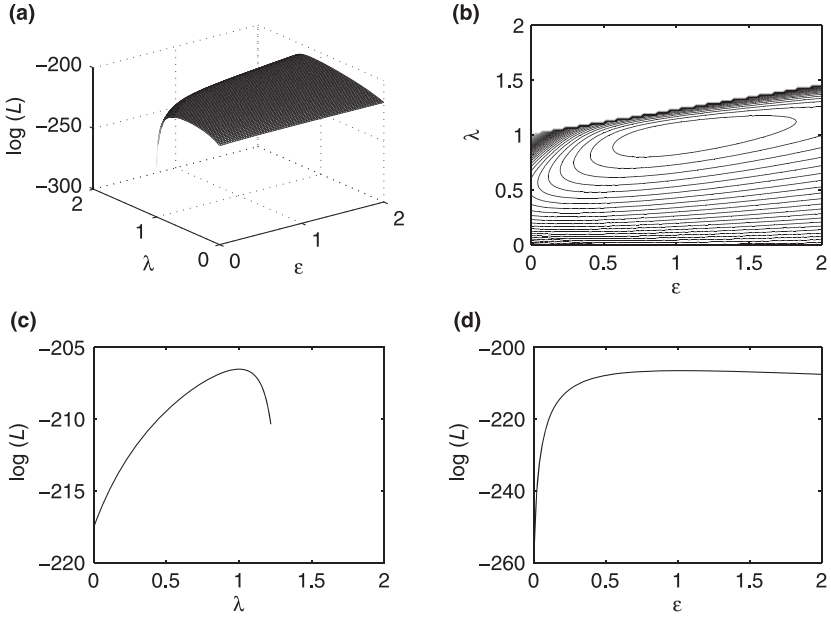

Fig. 2 Example likelihood surfaces for parameter estimation in the phylogenetic paired *t*-test. Surfaces were obtained by simulations on the primate phylogeny from this study with $\lambda = 1.0$ and $\varepsilon = 1.0$. (a) and (b) show different visualization of the multidimensional likelihood surface for λ and ε, whereas (c) and (d) show slices of the likelihood surface for λ and ε separately, taken along the maximum likelihood dimension of ε and λ, respectively. Note that $\varepsilon = \sigma^2_\varepsilon / \sigma^2$.

Table 1 Summary of the test of the paired *t*-tests used in this study. SD of the parameter estimates among simulations is given in parentheses. The three tests are a nonphylogenetic *t*-test, also ignoring simulated sampling error in the estimation of species means; a phylogenetic *t*-test ignoring sampling error; and a phylogenetic *t*-test that incorporates sampling error. Parameters were estimated using maximum likelihood, as described in the main text. Mean parameter values showing a dash (—) are not estimated in the specified model (i.e. they are fixed at zero for the analysis). Note that the generating values for $\bar{d}$, λ and σ^2_ε were $\bar{d} = 0.0$, $\lambda = 1.0$ and $\sigma^2_\varepsilon = 1.0$.

Test	$\bar{d}$	*t* (d.f. = *N* – *p*)	Type I error	MLE(λ)	MLE(σ^2_ε)
Nonphylogenetic/nonerror	0.152 (5.87)	0.143 (5.45)	0.727	–	–
Phylogenetic/nonerror	0.112 (4.58)	0.095 (1.47)	0.088	0.879 (0.193)	–
Phylogenetic/error	0.078 (4.57)	0.057 (1.38)	0.067	0.961 (0.139)	0.993 (0.727)

Revell, 2009). We performed all phylogenetic regression analyses using the R (R Development Core Team, 2008) package APE utilizing the corPagel phylogenetic correlation structure (Paradis *et al.*, 2004).

We also repeated all analyses using standard (nonphylogenetic) procedures. As these analyses produced results that were largely congruent with the phylogenetic tests, we focus on the phylogenetic results herein.

Results

All traits individually showed significant evidence for phylogenetic signal (Table 2). Haematocrit and RBC counts were also significantly correlated in both sexes after controlling for phylogeny ($r = 0.987$, $P < 0.0001$ in males; $r = 0.981$, $P = 0.0001$ in females). Haematocrit and RBC counts scaled negatively with body mass in both sexes, although the slope for haematocrit in females was not statistically significant (Table 3).

We used phylogenetic paired *t*-tests, as described above, to test our first two predictions involving sexual selection. These predictions were (i) that males have higher physiological measures of aerobic capacity and (ii) that aerobic capacity increases ontogenetically in males and females. For each trait and comparison, we computed the standard (nonphylogenetic) paired *t*-test; the phylogenetic *t*-test where λ is optimized but in which σ^2_ε (the effect of error) is set to zero; and, finally, the full phylogenetic model incorporating error. Figure 3 shows contour plots of the likelihood surfaces for λ and $\varepsilon = \sigma^2_\varepsilon/\sigma^2$ in our analyses of haematocrit (Fig. 3a) and RBC count differences among species between males and females. Also given by Fig. 3 are the locations of the maximum likelihood optima for the two parameters that are numerically estimated in these analyses (λ and $\varepsilon = \sigma^2_\varepsilon/\sigma^2$).

Table 2 Individual measures of phylogenetic signal (λ) for the physiological traits in this study. Phylogenetic signal was significantly nonzero for all but one trait.

Variable	λ	2 LR	*P*	*N*
Female haematocrit	0.722	30.680	< 0.001	45
Male haematocrit	0.250	2.579	0.108	45
Haematocrit dimorphism	0.626	15.752	< 0.001	45
Female RBC count	0.962	45.533	< 0.001	45
Male RBC count	0.900	31.446	< 0.001	45
RBC count dimorphism	0.544	5.706	0.017	45

P-values indicate significance levels when testing whether $\lambda = 0$ in a likelihood ratio test with one degree of freedom.

Table 3 Interspecific allometric scaling coefficients and model fit for the relationship between body mass and aerobic capacity in male and female primates.

Test	Haematocrit	RBC counts
Regressed on body mass in males	$\lambda = 0.161$	$\lambda = 0.912$
	$\hat{b} = -0.006$	$\hat{b} = -0.056$
	$P = 0.029$	$P = 0.002$
Regressed on body mass in females	$\lambda = 0.707$	$\lambda = 0.963$
	$\hat{b} = -0.007$	$\hat{b} = -0.058$
	$P = 0.072$	$P = 0.007$

Among adults, male primates exhibited higher haematocrit and higher RBC counts than females (Table 4). For the species in our study, adult males also had higher haematocrit and higher RBC counts than juvenile male conspecifics. In females, no corresponding significant difference between the age classes was found (Table 5). The differences between males and females, as well as between adults and juveniles, are shown graphically in Fig. 4. Thus, in analysing data from 45 primate species, we found support for the first two predictions: on average, males have significantly higher physiological measures of aerobic capacity than female conspecifics, and adult males have higher physiological aerobic capacity than juvenile males. Both findings are consistent with *a priori* predictions of sexual selection for increased aerobic capacity and opposite to predictions based on allometric scaling of metabolism.

In tests of our final prediction that aerobic capacity increases with increasing sexual selection, we found that haematocrit and RBC counts actually scaled negatively with sexual size dimorphism across species (Table 6). This pattern was opposite to our expectation based on sexual selection theory. Including both body mass and body mass dimorphism simultaneously in a multivariate regression model rendered body mass nonsignificant, whereas the relationship between physiological aerobic capacity and body mass dimorphism remained significantly negative. These multivariate regression results

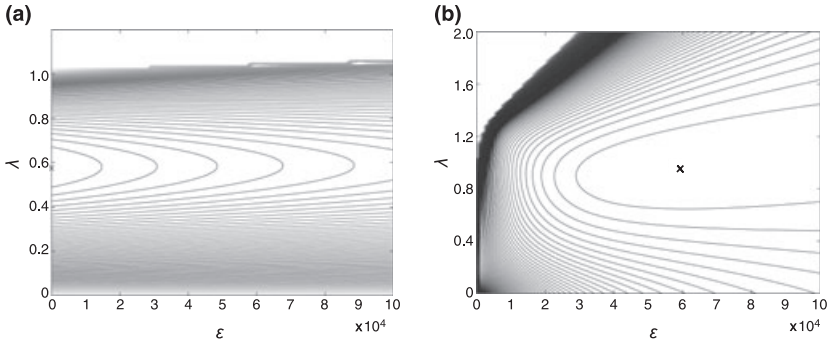

Fig. 3 Likelihood surfaces for λ and ε in the phylogenetic paired *t*-test. (a) The difference between male and female haematocrit measures and (b) the difference between male and female RBC measures.

Table 4 Sexual differences in haematocrit and RBC counts among 45 primate species. Results are based on paired *t*-tests conducted on species mean values. For haematocrit, the best fitting model included phylogeny but not error in the estimation of species means; however, for RBC count, the best fitting model included both phylogeny and error. Our results indicate that males have significantly higher haematocrit and RBC counts than females, regardless of the model.

Trait and comparison	Test	MLEs ($\lambda, \sigma^2_\varepsilon$); log($L$)	$\bar{d}$	t	d.f. ($N - p$)	P
Haematocrit M–F	Nonphylogenetic/nonerror	log(L) = 96.02	0.038	8.793	44	< 0.0001
	Phylogenetic/nonerror	λ = 0.581; log(L) = 102.42	0.030	2.343	43	0.0238
	Phylogenetic/error	λ = 0.581; $\sigma^2_\varepsilon = 0.0$; log($L$) = 102.42	0.030	2.316	42	0.0255
RBC M–F	Nonphylogenetic/nonerror	log(L) = −21.75	0.482	8.157	44	< 0.0001
	Phylogenetic/nonerror	λ = 0.343; log(L) = −20.57	0.432	2.959	43	0.0050
	Phylogenetic/error	λ = 0.964; $\sigma^2_\varepsilon = 8.20$; log($L$) = −19.21	0.360	4.431	42	< 0.0001

M, males; F, females.

Table 5 Ontogenetic differences in haematocrit and RBC counts. Among species, nonphylogenetic and phylogenetic analyses suggest that haematocrit and RBC counts are generally higher in adult males than in male juvenile conspecifics. The same difference is not found in females.

Trait and comparison	Test	MLEs ($\lambda, \sigma^2_\varepsilon$); log($L$)	$\bar{d}$	t	d.f. ($N - p$)	P
Haematocrit female A–J	Nonphylogenetic/nonerror	log(L) = 83.15	0.008	1.449	44	0.1543
	Phylogenetic/nonerror	λ = 1.00; log(L) = 97.51	0.034	1.177	43	0.2455
	Phylogenetic/error	λ = 1.37; $\sigma^2_\varepsilon = 3.76$; log($L$) = 109.53	0.012	1.373	42	0.1769
Haematocrit males A–J	Nonphylogenetic/nonerror	log(L) = 86.02	0.036	7.165	42	< 0.0001
	Phylogenetic/nonerror	λ = 0.083 log(L) = 86.04	0.035	4.508	41	< 0.0001
	Phylogenetic/error	λ = 0.0; $\sigma^2_\varepsilon = 2.27$; log($L$) = 86.54	0.036	7.285	40	< 0.0001
RBC female A–J	Nonphylogenetic/nonerror	log(L) = −25.21	–0.05	–0.78	42	0.4420
	Phylogenetic/nonerror	λ = 1.02; log(L) = −16.03	0.224	0.489	41	0.6278
	Phylogenetic/error	λ = 1.07; $\sigma^2_\varepsilon = 1.01$; log($L$) = −7.63	0.162	0.487	40	0.6290
RBC male A–J	Nonphylogenetic/nonerror	log(L) = −37.20	0.259	2.830	41	0.0072
	Phylogenetic/nonerror	λ = 0.0; log(L) = −37.20	0.259	2.795	40	0.0079
	Phylogenetic/error	λ = 0.0; $\sigma^2_\varepsilon = 9.22$; log($L$) = −32.72	0.245	3.947	39	0.0003

A, adults; J, juveniles.

should be treated cautiously, however, because we also found strong collinearity between overall body mass and size dimorphism (in particular, our variance inflation factor, VIF = 1/tolerance, was > 10; Quinn & Keough, 2002).

When we substituted a three-state mating system variable in place of body mass dimorphism, we again found a negative correlation between mating system and haematocrit for both males ($\hat{b} = -0.022$; $P = 0.020$) and females ($\hat{b} = -0.022$; $P = 0.014$). Similarly, the regressions of RBC counts on mating system were significantly negative for both males ($\hat{b} = -0.114$; $P = 0.004$) and females ($\hat{b} = -0.108$; $P = 0.006$). Neither haematocrit dimorphism nor RBC count dimorphism scaled significantly with sexual size dimorphism or with mating system. As was the case with our other indicator of sexual selection, including both body mass and mating system simultaneously in a multiple regression model produced severe collinearity between the independent variables (VIF > 10). Thus, although our first two

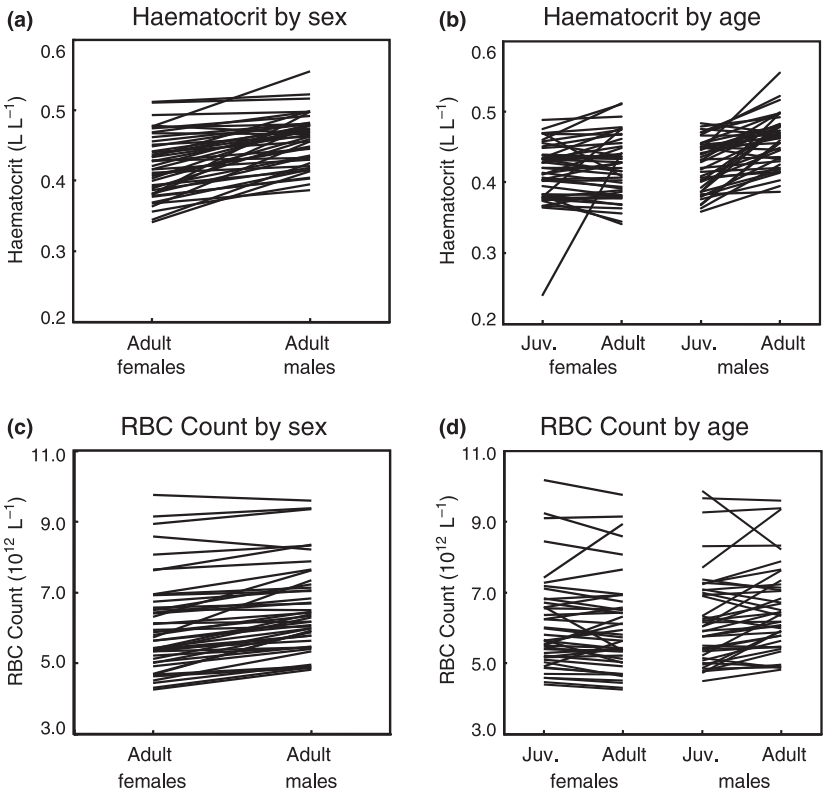

Fig. 4 Sex and age differences in haematocrit and RBC counts for the 45 species in our study. Plots show lines connecting different age or sex classes for each of the species used in the analysis. Analysed phylogenetically, adult males have significantly higher haematocrit and RBC counts, when compared to females and juvenile males. Figure panels are (a) haematocrit by sex; (b) haematocrit by age and sex; (c) RBC count by sex; and (d) RBC count by age and sex.

Table 6 Sexual selection on aerobic capacity. Regression coefficients for sexual selection (measured with body mass dimorphism) and haematocrit or RBC counts reveal a negative pattern, contrary to our *a priori* expectation.

Test	Haematocrit	RBC counts
Regressed on body mass dimorphism in males	$\lambda = -0.141$ $\hat{b} = -0.059$ $P < 0.001$	$\lambda = 0.874$ $\hat{b} = -0.272$ $P < 0.001$
Regressed on body mass dimorphism in females	$\lambda = 0.626$ $\hat{b} = -0.053$ $P < 0.001$	$\lambda = 0.947$ $\hat{b} = -0.266$ $P < 0.001$

predictions based on sexual selection were confirmed by phylogenetic tests, our final prediction, that measures of sexual selection and physiological aerobic capacity dimorphism should be positively correlated, was opposite to our empirical finding.

To investigate alternative explanations for this pattern, we examined several other variables that we hypothesized are related to aerobic capacity in primates. These variables involved locomotor and energetic benefits of RBC or haematocrit, as well as substrate use, basal metabolic rate and morphological and behavioural measures of agility. We found some evidence that aerobic capacity co-varies with agility. In particular, when we treated the ordered discrete scores of agility from Spoor *et al.* (2007) as a continuous variable in a regression on physiological aerobic capacity, we found significant positive relationships between haematocrit or RBC and agility (haematocrit females $\hat{b} = 0.004$; $t = 2.877$ $P = 0.008$; haematocrit males $\hat{b} = 0.005$; $t = 3.726$ $P = 0.001$; RBC females $\hat{b} = 0.023$; $t = 3.589$ $P = 0.001$; RBC males $\hat{b} = 0.019$; $t = 3.070$ $P = 0.005$). However, similar analyses based on morphological measures of agility (i.e. semi-circular canal radius from Spoor *et al.*, 2007) were not significant. None of the other alternative variables were significantly correlated with RBC or haematocrit. All included variables were, however, correlated with body mass, creating collinearity in our multivariate regression models (VIF > 10).

Discussion

Among our sample of 45 primate species, males generally have higher haematocrit and higher RBC counts than females. We also found that adult males have higher haematocrit and higher RBC counts than juvenile males, whereas there was no significant difference between adult and juvenile females. Thus, we identified a persistent pattern of greater aerobic capacity among primate species in adult males, when compared to other age–sex categories. This corresponds with similar patterns previously reported for humans and a few other mammals (Glucksman, 1978).

Our results were confirmed using both ordinary paired *t*-tests, in which species were treated as independent data in the analysis, and with phylogenetic paired *t*-tests. Our phylogenetic paired *t*-test is based on phylogenetic GLS, and it is, as far as we know, an application unique to this study. A phylogenetic paired *t*-test is appropriate to interspecific data because these data generally violate a central assumption of the standard paired *t*-test; that is, the assumption that each observation is an independent draw from the same, uncorrelated underlying distribution.

We also investigated the relationship between physiological measures of aerobic capacity and sexual selection using two different measures of aerobic capacity (haematocrit and RBC count) and using two different measures of sexual selection (sexual size dimorphism and mating system on an ordinal scale). Here, we expected to find that aerobic capacity increased with increasing sexual selection. Instead, we found the opposite pattern, specifically a negative relationship between aerobic capacity and sexual selection. The relationships between haematocrit and body mass and between RBC counts and body mass were also both negative, which is in line with expectations from Kleiber's law that metabolic rate scales to body mass with an exponent of 0.75 (Schmidt-Nielsen, 1975; Calder, 1984). This law, along with the observation that blood volume scales linearly with body mass, leads to the ancillary prediction that larger-bodied individuals will require fewer red blood cells and haematocrit per unit of blood. In a multivariate analysis in which we also included body mass dimorphism, body mass became nonsignificant, although collinearity between body mass and mass dimorphism leads us to be cautious in interpreting the coefficients of this model.

Testing the hypothesis that haematocrit and RBC counts are tied to differing degrees of sexual selection showed that both haematocrit and RBC counts were significantly negatively correlated with the degree of sexual selection. This finding is contrary to our *a priori* expectation that aerobic capacity should increase as a function of increasing sexual selection. Furthermore, sexual dimorphism in haematocrit and RBC count did not correlate with any measure of sexual selection. Sexual dimorphism in aerobic capacity thus appears to be caused by some factor(s) other than sexual selection on physical prowess. In this regard, we investigated several variables potentially correlated with aerobic capacity, including substrate use, basal metabolic rate and agility. Of these, we found some support for the hypothesis that aerobic capacity co-varies positively with agility. However, this pattern could not be fully substantiated because of a strong negative correlation between agility and body mass and collinearity between mass and sexual dimorphism.

Previous studies have investigated interspecific variation in red blood cell counts in mammals and other vertebrates. In a nonphylogenetic analysis, Bennett & Hawkey (1988) found that the correlation coefficient between body mass and RBC counts is negative for both mammals and birds, but only statistically significant in birds. Using nested analysis of variance, they also showed that most of the variation in RBC counts exists above the species level (i.e. among genera and families). In a more recent study, Preston *et al.* (2009) examined variation in RBC counts across 24 species of mammals using phylogenetically independent contrasts. Like us, they found a negative association between RBC counts and body mass. Researchers also have documented a negative correlation between the size and number of cells, with larger numbers of RBCs associated with smaller cell size (Bennett & Hawkey, 1988; Hawkey *et al.*, 1991). In contrast to the paucity of comparative research on physiological measures of aerobic capacity, many more studies have considered the ecological correlates of white blood cell counts and platelets (Nunn *et al.*, 2000, 2003, 2009; Nunn, 2002; Semple *et al.*, 2002; Anderson *et al.*, 2004).

Although we hypothesized sexual selection on aerobic function *a priori*, we were unable to conclusively tie aerobic capacity dimorphism to sexual selection. To help identify the generality and the causes of sexual dimorphism in aerobic capacity, it might be useful to investigate other mammalian orders, and perhaps also other nonmammalian homoeotherms such as birds. Phylogenetic comparative studies of a broader range of taxa than were included in this study might also reveal new factors that influence measures of aerobic capacity. Other factors involved in effective tissue oxygenation, such as the size of red blood cells and circulation rates, may provide further clues to the selective factors that are operating on aerobic capacity in wild organisms (see also Hawkey *et al.*, 1991). Thus, the potential for interesting results from future comparative studies of aerobic measures is great, particularly in analyses that take into account phylogenetic relatedness.

To investigate differences in measures of aerobic function among age–sex classes, we developed a new phylogenetic paired *t*-test that is based on a GLS estimator for the mean difference between the paired groups in the analysis. We also provide computer code in the electronic supporting information so that others can implement the method. GLS is appropriate to this problem because we would normally use the ordinary least squares mean difference and standard error to compute the test statistic, and GLS is a class of statistical model that is useful when the residual errors are correlated among observations (Rencher & Schaalje, 2008). Our method also incorporated error variance in the estimation of species means following Ives *et al.* (2007).

Using simulations, we demonstrate that the phylogenetic paired *t*-test has better statistical properties than analogous nonphylogenetic tests when the data come

from species that are correlated because of phylogenetic relations, including a more than 10-fold lower type I error rate over the nonphylogenetic method. We did, however, find that the type I error rate of the test remained slightly elevated over its nominal level (Table 1). Many of the false-positive results contributing to this elevated type I error corresponded with incorrect estimation of the parameters of the phylogenetic model, λ (the scaling parameter of phylogenetic covariance; Pagel, 1999a) and σ^2_ε (the error variance; Ives *et al.*, 2007), which were obtained using maximum likelihood optimization. The sampling distribution of a maximum likelihood estimator is generally only asymptotically unbiased (Lynch & Walsh, 1998), leading us to suspect that the performance of our method would be improved for larger phylogenies. Indeed, simulations on $n = 100$ taxon phylogenies showed considerable improvement both in maximum likelihood parameter estimation and in type I error. Future studies on small phylogenies might consider obtaining a null distribution for t by performing numerical simulations (e.g. Garland *et al.*, 1993) using the MLEs of λ and σ^2_ε, while setting the phylogenetic mean difference to $\bar{d} = 0.0$. This procedure would be quite computationally intensive as one would need to perform ML estimation for each simulated dataset to obtain estimates of λ and σ^2_ε, but it might decrease the type I error rate of the test to its nominal level.

In summary, in a study of 45 species broadly sampled from the primate tree, we found that primate males have higher measures of aerobic capacity than females and that adult males have higher measures of aerobic capacity than juvenile males. This corresponds with similar patterns previously reported for humans and a few other mammals (Glucksman, 1978). When testing whether aerobic capacity is a sexually selected character, however, we found negative correlations between aerobic capacity and both body mass and the degree of sexual selection. Thus, sexual dimorphism in physiological measures related to aerobic capacity may be caused by some factor(s) other than sexual selection on physical prowess. Our only indication as to what this factor might be came from the observation that RBC counts and haematocrit scale positively with measures of agility (but not with morphological measures, i.e. the semicircular canals, which also co-vary with agility; Spoor *et al.*, 2007). Unfortunately, because agility also correlates strongly with body mass, we were unable to disentangle the effects of these two variables in multivariate analyses. Future research would benefit from expanding the taxonomic scope to include nonprimates and greater consideration of additional physiological parameters related to aerobic function.

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Supporting information

Additional Supporting Information may be found in the online version of this article:

Data S1 Computer code for phylogenetic paired *t*-test.

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