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Human fertility variation, size-related obstetrical performance and the evolution of sexual stature dimorphism

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In several animal species, change in sexual size dimorphism is a correlated response to selection on fecundity. In humans, different hypotheses have been proposed to explain the variation of sexual dimorphism in stature, but no consensus has yet emerged. In this paper, we evaluate from a theoretical and an empirical point of view the hypothesis that the extent of sexual dimorphism in human populations results from the interaction between fertility and size-related obstetric complications. We first developed an optimal evolutionary model based on extensive simulations and then we performed a comparative analysis for a total set of 38 countries worldwide. Our optimization modelling shows that size-related mortality factors do indeed have the potential to affect the extent of sexual stature dimorphism. Comparative analysis using generalized linear modelling supports the idea that maternal death caused by deliveries and complications of pregnancy (a variable known to be size related) could be a key determinant explaining variation in sexual stature dimorphism across populations. We discuss our results in relation to other hypotheses on the evolution of sexual stature dimorphism in humans.

Keywords: anthropology; fertility; maternal mortality; stature; sexual dimorphism

1. INTRODUCTION

In all human populations, men are on average taller than women but the extent of this sexual dimorphism varies between populations. Although no consensus has emerged to explain this variation, several hypotheses have been suggested. For example, well-nourished populations are more sexually dimorphic than malnourished ones, because male growth is more susceptible to nutritional deficiencies during development than female growth (Hiernaux 1968; Brauer 1982; Hamilton 1982). Sexual dimorphism could also result from natural selection acting differentially on males and females when occupying different ecological niches, e.g. different foraging strategies (Frayer 1980, 1981; Brace & Ryan 1980). The 'women's work hypothesis' (Holden & Mace 1999) suggests that sex-biased parental investment could be responsible for variation in sexual dimorphism: women would be taller, relative to men, in societies where women contribute more to food production because parents invest relatively more in their daughters, so that the growth of girls would not be compromised relative to that of boys (Holden & Mace 1999). Finally, sexual dimorphism in stature could result from sexual selection, being greater among populations with polygynous marriage because of intra-male competition for females (Trivers 1972; Alexander *et al.* 1979; but see Gray & Wolfe 1980). Thus, it seems likely that the degree of sexual dimorphism in human stature may be directly or indirectly influenced by various ecological and physiological constraints.

In this paper we evaluate from a theoretical and an empirical point of view another hypothesis, that the extent of sexual dimorphism in human populations results from the interaction between fertility (e.g. total number of offspring born to a woman passing through child-bearing age) and size-related obstetric complications. Human populations are characterized by strong variation in fertility (Jones 1990). In addition, an impressive body of gynaecological literature has shown that short maternal stature is frequently associated with serious obstetric complications and often requires an operative delivery (e.g. Caesarean section or symphyseotomy; Camilleri 1981; Adadevoh *et al.* 1989; Parsons *et al.* 1989; Sokal *et al.* 1991; Van Roosmalen & Brand 1992; Tsu 1992; Kwawukume *et al.* 1993; Moller & Lindmark 1997). The correlation between maternal height and obstetrical outcome is so strong that female stature is currently used in most antenatal programmes (e.g. UNICEF, World Health Organization) to screen pregnant women for potential risk of difficult childbirth and cephalopelvic disproportion. Knowing that body height is heritable in humans (e.g. Golden 1994; Carmichael & McGue 1995; Luo *et al.* 1998; Arinami *et al.* 1999; Magarey *et al.* 1999; Pietilainen *et al.* 1999), it seems likely that size-related obstetrical problems, interacting with variation in fertility, may influence the evolution of female stature and hence sexual dimorphism. We predict that in highly fecund populations, selective pressures for large stature in females are high and sexual dimorphism is reduced as a consequence. Conversely, in less fecund populations, sexual dimorphism would be higher because of reduced selective pressure on females for large size.

2. MATERIAL AND METHODS

(a) *Evolutionary optimization modelling*

To explain the existence of sexual stature dimorphism, and its dependence on environmental conditions from the point of view

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of evolutionary optimality, we have built a state-dependent model of evolutionarily optimal allocation of energy between growth and reproduction (Ziolko & Kozlowski 1983; Kozlowski & Teriokhin 1999). We assume that the rate of energy production in a unit of time (one year in our model) is proportional (with coefficient of proportionality a) to the body mass, w_t, at age t to some power, b, less than one (e.g. Harvey *et al.* 1991),

$$P_t = aw_t^b. \tag{1}$$

This energy is divided between growth and reproduction in proportion u_t to $1-u_t$; the rate of growth at age t is proportional to the fraction of energy allocated to growth,

$$w_{t+1} = w_t + u_t P_t, \tag{2}$$

and the reproductive gain is proportional to some function, F, of the fraction of energy allocated to reproduction,

$$G_{t+1} = G_t + F(R_t), \tag{3}$$

where

$$R_t = (1-u_t)P_t. \tag{4}$$

As a measure of evolutionary optimality we use information on the individual's lifetime reproductive success, calculated as expectation of the amount of energy allocated by the individual to reproduction throughout its entire life from zero to some maximum life span, T, which should be sufficiently large:

$$H_0 = \sum_{t=1}^{T} F(R_t)L_t. \tag{5}$$

We set $T=110$ in our computations (the probability of surviving beyond this age is less than 10^{-6} according to the Gompertz equation with typical parameters).

The survival, L_t, in the above equation is defined by

$$L_t = \exp\left(-\sum_{s=0}^{t-1} Q_s\right), \tag{6}$$

where the mortality rate, Q_s, at age s is the sum of three terms

$$Q_s = A + B\exp(C_s) + D\exp(-Ew_s). \tag{7}$$

The first two terms in this expression correspond to the equation of Gompertz–Makeham (Gompertz 1825; Makeham 1860). The first term does not depend on age and may be considered to reflect environmentally caused mortality; the second one, which increases with age, may be considered to reflect the process of physiological ageing. The third term, which we consider to reflect adult (acting after age of maturity) size-dependent mortality (i.e. obstetric-related mortality for women, other reasons such as sexual selection for males), is assumed to decrease exponentially with increasing body size, w_s. Coefficient D reflects the relative importance of size-dependent mortality and coefficient E reflects the efficiency of lowering size-related mortality by increasing body size. The problem of evolutionary optimization in our case consists of searching for the optimal strategy of dividing energy between growth and reproduction, i.e. in searching for $u^*(t,w)$ such that R is maximal for any life history starting with some initial size, w_0, and following the strategy $u^*(t,w)$. It can be shown, using, for example, the Pontryagin maximum principle (Pontryagin *et al.* 1962; Ziolko & Kozlowski 1983), that when reproductive gain and rate of growth depend linearly on u_t the strategy of optimal energy allocation only has two states: all energy should be allocated to growth (from birth up to some age of maturity, T_m) and thereafter to reproduction (after the age of maturity). The body size, W_m, reached by the age of maturity is called the size of maturity. However, humans continue to grow (up to about 25 years) after the age of maturity (which varies between 12 and 16 years). The effect of post-maturity growth emerges in the model if we assume that reproductive gain depends nonlinearly on reproductive investment, that is, that the function F in equation (3) is nonlinear, namely sigmoid

$$F(R_t) = R_t^\beta/(\alpha + R_t^\beta). \tag{8}$$

To compute optimal strategies we use the method of stochastic dynamic programming (Bellman 1957; Mangel & Clark 1988). This method consists of two steps. In the first step, we solve, iterating backwards, the so-called principal equation of dynamic programming, which in our case can be written in the form

$$H_t(w_t) = \max_{u_t}\{[H_{t+1}(w_{t+1}) + F(R_t)]\exp(-Q_t)\}, \tag{9}$$

(we assume $H_T(w_T)=0$ for all w_T). In the second step, we solve, by iterating forwards, the differential equation for body size starting from some initial size, which we set in our computations equal to 3.25, i.e. a value close to the weight at birth for a human child.

(b) *Comparative analysis*

(i) *The data set*

Data on stature of human males and females were taken from different published sources and by writing to several ministries of health where national statistics existed (our database is available upon request). These data refer to the average human stature (adults aged 18–30 years only) for both sexes per country. We collected data on male stature for 72 different countries, and on female stature for 66 different countries worldwide. This permits the calculation of sexual dimorphism within populations for 62 different populations. Since a body of variables may be responsible for inter-population variation in sexual dimorphism between countries, we considered in our analysis historical (i.e. human ethnic groups), geographical (latitude and longitude), socio-economical (global net product incomes per country and total, or only vegetable and/or only animal, calorie consumption per inhabitant), parasitological (total number of infectious diseases known in an area), two female life-history traits (mean fertility per female and mean age at menarche) and one obstetrical estimate (maternal-mortality ratio) components.

Data for socio-economical parameters were obtained from the 1992 world population data sheet (Jones 1990): the per capita gross national product (GNP) in US$ per year; calorie consumption per average inhabitant per day, categorized in vegetable-protein consumption, in animal-protein consumption, or both, taken from food balance sheets from the World Atlas© (1992). We considered mean latitude and longitude variables, which may affect variation in human stature across countries: the mean latitude and the mean longitude (in degrees) refer to the values measured at the geographical centre of each country. Since close geographical neighbours may share similar traits, e.g. inhabiting similar environments, simple cross-country comparisons are likely to be confounded by distances. The introduction of geographical similarities between countries into our generalized linear model alleviates this artefact. Parasites have been shown to play a role in the evolution of sexual dimorphism in many animals (e.g. Hamilton & Zuk 1982; Andersson 1994),

and so we collected data from both the Centers for Disease Control and the World Health Organisation for a set of 16 categories of human diseases known to affect survival (i.e. typhoid, hepatitis A, hepatitis B, malaria, schistosomiasis, filariosis, meningococcosis, yellow fever, dengue fever, cholera, trypanosomiasis, dracunculosis, chagas disease, lyme disease, cutaneous leishmaniosis and visceral leishmaniosis). Based on this information, we calculated the disease load as the total number of disease types for each country. We used two different human life-history traits likely to affect sexual dimorphism: fertility, which indicates productivity as the number of offspring born to a woman passing through child-bearing age, and age at menarche, which represents the entry of women into effective reproductive life. Data on fertility comes from Jones (1990). To obtain data on age at menarche, we performed a literature search for recent publications on this topic (see Thomas *et al.* (2001) for a summary table). The maternal-mortality ratio is defined as the number of maternal deaths (within 40 days postpartum) caused by deliveries and complications of pregnancy, child-birth and the puerperium divided by the number of live births for a given year, and expressed per 100 000 live births. Since this information may differ from source to source (e.g. it is not clear whether it includes abortion-related deaths), we used only the statistics of UNICEF in order to have a homogeneous data set (data were available for a subset of 38 countries). Furthermore, we categorized the different populations, according to Cavalli-Sforza (1997), into eight large divisions of ethnological groups: I, Africans and Nilotics (except native people from the Maghreb); II, Europeans (including people from the Middle East); III, Indians; IV, Mongoloids, Japanese and Koreans; V, Amerindians; VI, New Guineans–Papous; VII, Melanesians; VIII, Mhongs, Khmers, Thais, Filipinos, Indonesians and related tribes. In addition, a ninth category was assigned to Creoles from Caribbean islands. Ethnological divisions were introduced to control for similarity due to inheritance among related tribes (Harvey & Pagel 1991; Martins 1996). In fact, characters may be inherited as a result of either behavioural factors shared by two ethnic groups or by genetic similarity between two given tribes, or both.

(ii) *Measuring sexual dimorphism*

For the variation in sexual dimorphism between populations we used a relative measure equal to the ratio of male size to female size (Alexander *et al.* 1979; Gray & Wolfe 1980; Gaulin & Boster 1992). This differs from other measures of sexual dimorphism used in some previous studies. In particular, Harvey & Mace (1982) used residuals from the reduced major axis since a correlation may exist between the relative ratio of dimorphism and body size (Gaulin & Boster 1985; Ranta *et al.* 1994). However, we checked for relationships between both female and male stature and sexual dimorphism and they were not significant (linear regression, $r^2 = 0.007$, $p = 0.15$ and $r^2 = 0.085$, $p = 0.68$, respectively).

(iii) *Phylogenetic comparative analysis*

If a human life-history trait is associated with phylogenetic relatives, this tends to indicate that it is transmitted from ancestor to daughter populations. Thus, two closely related ethnic groups may share similarities transmitted vertically, and therefore treating these ethnic groups as independent points in statistical analyses may greatly increase the likelihood of type I errors. To correct for this potential bias, we incorporated phylogeny into the statistical analysis of variation in sexual dimorphism, using the phylogenetic regression procedure (one of the most widely used methods of controlling for similarity due to common descent; Grafen 1989; Martins 1996).

(iv) *Generalized linear models*

In order to assess the evolution of sexual dimorphism across countries, we first analysed the data univariately. Then, we used generalized linear modelling (McCullagh & Nelder 1989) to assess simultaneously which explanatory variables and/or their interaction terms better explained inter-population differences in stature between the human sexes. For these purposes, we used a generalized linear model (GLM) with a normal error, which represents the most appropriate statistical tool for analysing our data.

Our maximal model is as follows: sexual dimorphism $= a \times$ (log GNP) $+ b \times$ (log fertility) $+ c \times$ (maternal mortality ratio) $+ d \times$ (parasites) $+ e \times$ (menarche) $+ f \times$ (longitude) $+ g \times$ (latitude) $+ h \times$ (phylogeny) + (all two-way interactions between the five first terms) $+ \sigma$.

Minimal models were selected with a backward stepwise elimination procedure. We used the tolerance option at the 0.05 level, which avoids constructing highly multi-colinear models in a stepwise procedure. Here, we used the S-Plus statistical package (MathSoft, Inc. 1999; Venables & Ripley 1994). The variances for terms in the model were compared using *F*-tests. When the data suggested there were no linear trends, the explanatory variables were transformed and fitted again to try to improve their contribution to the model. As a result, both GNP and fertility variables were logarithmically transformed.

3. RESULTS

(a) *Evolutionary optimization modelling*

The evolutionary optimization model we described in § 2(a) includes a set of parameters that must be estimated before running the model. We have accomplished this estimation using different approaches. The values of parameters B and C in equation (7) were set to typical values estimated from demographic data: namely, $B = 0.000\,01$ for women and $B = 0.000\,02$ for men and $C = 0.1$ both for women and for men (e.g. Gavrilov & Gavrilova 1991). The parameter of environmental mortality, A, which varies from 0.001 for several European countries to about 0.013 for several African countries (Thomas *et al.* 2000) was set to an intermediate value of $A = 0.005$. The exponent b in equation (1) was set to 0.5 on the basis of nonlinear fitting of the data presented in Weinsier *et al.* (1992), and falls in the range of values for b typically encountered in the literature (e.g. Harvey *et al.* 1991). We could not, however, use the estimate for the parameter a in equation (1) obtained from this nonlinear fitting, first, because we used in our model units of mass (kg) as units of energy (as can be seen from equation (2)) and not units of heat (kJ) as in Weinsier *et al.* (1992) and, second, because we did not take into account in equation (1) the energy allocated to the needs of the organism other than for growth and reproduction (maintenance, repair, etc.). We assumed that these other needs constitute an approximately constant proportion of all energy produced in a unit of time and hence that they simply decrease the constant a in equation (1). We estimated this parameter indirectly, choosing a value of a that leads to plausible values of women's age and size of maturity after running

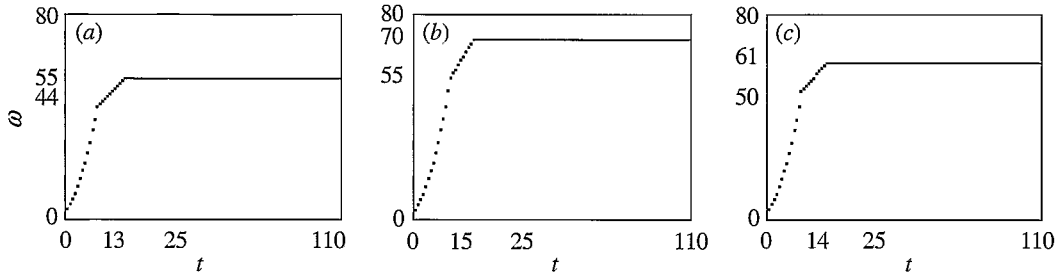

Figure 1. Examples of evolutionarily optimal dynamics of growth for different levels of size-dependent mortality. (*a*) No size-dependent mortality ('females') with maturity age $t=13$ years and maturity size $w=44$ kg ($w=55$ kg at $t=25$ years, age of termination of growth). (*b*) Strong size-dependent mortality ('males') with maturity age $t=15$ years and maturity size $w=55$ kg ($w=70$ kg at $t=25$ years, age of termination of growth). (*c*) Weak size-dependent mortality ('females') with maturity age $t=14$ years and maturity size $w=50$ kg ($w=61$ kg at $t=25$ years, age of termination of growth).

the evolutionary optimization model. The estimate of a so obtained was 0.8. Simultaneously, we estimated the parameters α and β in equation (8) as 200 and 4.5, respectively. The results of modelling with this set of parameters, assuming $D=E=0$, are presented in figure 1*a*. We observe growth up to the age of maturity at 13 years, when the body mass reaches 44 kg. Growth continues, though at a considerably reduced rate, until 25 years, when the body mass becomes equal to 55 kg.

Our next step was to show that in the presence of size-dependent mortality ($D>0$, $E>0$) optimal size at maturity and adult size increase, and hence that this mortality may explain the emergence of sexual dimorphism during human evolution. Indeed, as we can see in figure 1*b*, evolutionarily optimal mass at maturity for $D=0.01$ and $E=0.025$ is 55 kg (instead of 44 kg for $D=E=0$) and adult mass is 70 kg (instead of 55 kg); that is, we obtained values typical for men.

In a similar manner we can explain the reduction of sexual size dimorphism. For this it is sufficient to assume some size-dependent mortality in women. Such a situation is illustrated in figure 1*c* where mass at maturity for $D=0.001$ and $E=0.025$ is 50 kg (instead of 44 kg for $D=E=0$) and adult mass is 61 kg (instead of 55 kg). These results show that size-dependent mortality is indeed a factor that can strongly influence evolutionarily optimal size at maturity and adult size. Though we have based the model upon body mass, the above conclusions qualitatively remain valid for body stature because of the strong correlation between human body mass and stature (e.g. Ferembach *et al.* 1986).

(b) *Comparative analysis*

In the univariate analysis of sexual dimorphism across different human populations, several significant relationships exist between the extent of stature dimorphism and the independent variables (table 1). However, when performing a multivariate analysis, only the interaction term between maternal-mortality ratio and fertility proved to be a good predictor of sexual dimorphism in a stepwise generalized linear modelling approach (table 2 and figure 2). None of the other variables, nor their interactions, entered even in the first steps of the GLM statistical procedure. This implies that a high number of maternal deaths caused by deliveries or complications of pregnancy, and a high level of birth, both contribute statistically to low sexual dimorphism across populations. Our model was very robust, accounting for up to 20% of the total deviance.

Table 1. *Univariate analyses of sexual dimorphism in human stature against a set of variables that could possibly cause inter-country differences*

variables	r	t	p (two-tailed)
fertility (log)	0.427	−2.835	0.007
maternal-mortality ratio	0.404	−2.651	0.012
gross national product (log)	0.357	2.290	0.028
total calorie	0.317	2.114	0.041
vegetable calorie	0.180	1.156	0.255
animal calorie	0.287	1.896	0.065
parasites	0.315	−1.992	0.054
menarche	0.377	−1.991	0.058
latitude	0.232	1.876	0.069
longitude	0.129	−0.779	0.441
ethnic group I	0.425	−3.249	0.002
ethnic group II	0.243	1.738	0.089
ethnic group III	0.121	0.843	0.403
ethnic group IV	0.145	1.017	0.314
ethnic group V	0.156	1.091	0.281
ethnic group VI	0.069	−0.478	0.635
ethnic group VII	0.128	−0.895	0.375
ethnic group VIII	0.035	0.245	0.807
ethnic group IX	0.156	1.095	0.279

4. DISCUSSION

Change in sexual size dimorphism as a correlated response to selection on fertility is known in several animal species (Andersson 1994; Prenter *et al.* 1999; Reeve & Fairbairn 1999). Despite increasing evidence that female stature significantly influences obstetric performance, little attention has been devoted to the evolutionary implications of this phenomenon. However, our optimization modelling shows that size-related mortality factors indeed have the potential to affect the extent of sexual stature dimorphism. First, the action of size-related mortality can explain the overall excess of male size over that of females if we assume (e.g. Slatkin 1984; Shine 1989) that, because of divergence of ecological niches and

Table 2. *Summary of GLM for sexual dimorphism in human stature across 38 different populations*

(Results are obtained after the stepwise backward elimination procedure with a tolerance option of 0.05. A unique GLM in which only the interaction term between maternal-mortality ratio and fertility (log transformed) was obtained ($r=0.434$, $F=8.342$, $p=0.0065$). Also given are the parameter estimate, the standardized partial regression coefficient (std. b), the residual degree of freedom in the analysis (res. d.f.), t-statistics and associated probabilities. Forward stepping procedure yielded similar results.)

	parameter estimate	std. b	res. d.f.	t	$p(>\|t\|)$
intercept	1.079	0.000	38	455.646	<0.0001
maternal mortality × fertility	−0.001	−0.434	37	−2.888	0.0065

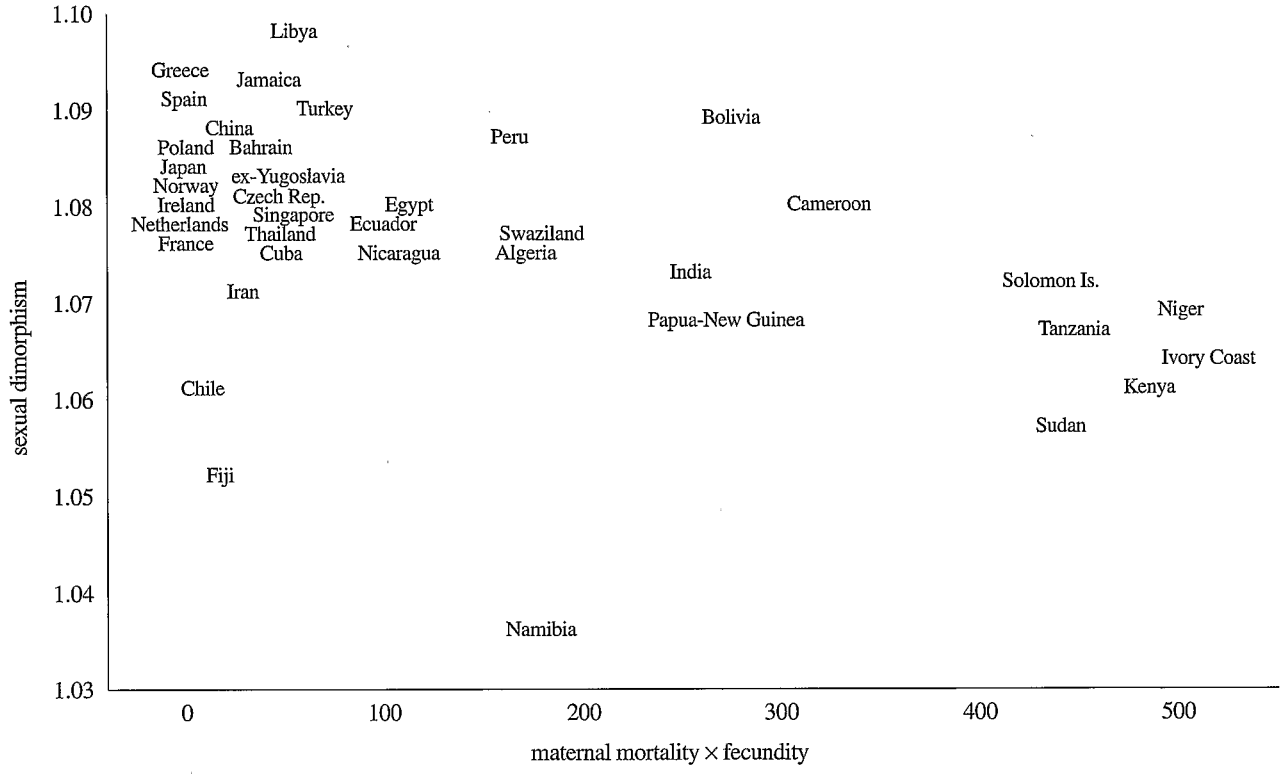

Figure 2. Sexual dimorphism in human stature in relation to the interaction term between maternal-mortality ratio and fertility (log transformed) as predicted by the GLM. For further explanation, see § 2.

reproductive strategies, the survival of males is more directly dependent on their size. Indeed, our model predicts that the observed sexual size dimorphism can be explained by an excess of the order of 0.0015 in the annual size-dependent mortality rate for men above that of women. Second, environmental variation in size-related mortality can explain changes in sexual dimorphism. In our modelling, the observed reduction of sexual size dimorphism can be explained by an increase of the order of 0.0001 in the annual size-dependent mortality for women.

Our comparative analysis using generalized linear modelling supports the idea that maternal death caused by deliveries and complications of pregnancy (a variable known to be size related) could be a key determinant explaining variation in sexual stature dimorphism between populations. Sexual dimorphism in stature was, indeed, negatively correlated with the interaction between maternal-mortality ratio and fertility. This indicates that the variation in dimorphism depends more, on a global scale, on the multiplicative effect of the maternal-mortality ratio and fertility than on the sum of the effects of each variable considered separately. This is in accordance with our expectations, since, to assess the selective pressure under study, it is necessary to multiply the risk of dying for a given birth (i.e. maternal-mortality ratio) by the number of birth events during the reproductive lifetime. Further research would be necessary to assess the importance of this selection during human evolution. However, what seems clear is that women in many countries still experience this selective pressure, for instance in rural areas in Africa where fertility values are among the highest and obstetric interventions are limited or absent.

An alternative hypothesis is that high values of the interaction between maternal-mortality ratio and fertility are a consequence rather than a cause of sexual stature dimorphism, which would then be determined by other factors. In accordance with this idea, Mascie-Taylor & Boldsen (1988) showed, from a large British national sample, that as husband–wife height differences increase,

so does the probability of having an abnormal pregnancy outcome. Further investigations would be necessary to assess this hypothesis. For instance, with the help of gynaecologists, surveys could be conducted in several countries in order to collect data on spousal physical characteristics and women's reproductive performance.

The fact that several variables have not been retained as explanatory variables in the final model allows us to discuss the relevance of other hypotheses. For instance, the 'women's work hypothesis' (Holden & Mace 1999), which stipulates that sexual dimorphism is influenced by the amount of food received by boys and girls during development (i.e. parental decisions), is not supported here. Under this hypothesis, rich countries would probably show lower levels of dimorphism than poor countries because parental discrimination against girls is less frequent in rich countries. Our results indicate the opposite trend: sexual dimorphism is higher in rich than in poor countries (for similar results, see also Eveleth 1975). The 'nutrition hypothesis' (Hiernaux 1968; Hamilton 1982), suggesting that well-nourished populations are more sexually dimorphic than malnourished ones because male growth is more susceptible to nutritional deficiencies during development than is female growth, is not supported either. Despite the higher level of dimorphism in rich countries than in poor ones, nutritional variables are not retained in the final model. However, more investigations are undoubtedly necessary to address this problem precisely. Finally, our study does not support the hypothesis that sexual-selection processes mediated by parasites influence sexual dimorphism.

It is frequently argued that comparative analyses using information from different sources may be inappropriate because data have been collected by different methods or have come from different sources. Although this argument is always applicable when no significant result is detected (i.e. the data are not precise enough to detect a potentially significant result), it is unlikely to be relevant when significant trends are found, since a biological tendency has *a priori* no reason to be correlated with background noise in the data set (Møller 1997; Lawton 1999). In conclusion, our study supports the idea that women are taller, relative to men, in populations where high levels of fertility are likely to counter-select short women because of their higher incidence of obstetrical complications. We would like, however, to underline some possible limitations in our study. If the maternal-mortality ratio responds very rapidly to recent changes in access to health care, the relationship observed between this parameter and stature dimorphism is not optimal. In addition, the sources of environmental heterogeneity at the largest scale are very variable. Thus, we must be aware that despite our efforts to control for such effects, we cannot exclude the possibility that other parameters, at a different scale, may confound our conclusion.

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