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Gert Stulp[1,2,*], Bram Kuijper[3],
Abraham P. Buunk[1,4], Thomas V. Pollet[5]
and Simon Verhulst[2]

[1]Department of Psychology, and [2]Department of Behavioural Biology, University of Groningen, Groningen, The Netherlands
[3]Department of Zoology, University of Cambridge, Cambridge, UK
[4]Royal Netherlands Academy of Arts and Sciences, Amsterdam, The Netherlands
[5]Department of Social and Organizational Psychology, VU University Amsterdam, Amsterdam, The Netherlands
*Author for correspondence (g.stulp@rug.nl).

Intralocus sexual conflict (IASC) occurs when a trait under selection in one sex constrains the other sex from achieving its sex-specific fitness optimum. Selection pressures on body size often differ between the sexes across many species, including humans: among men individuals of average height enjoy the highest reproductive success, while shorter women have the highest reproductive success. Given its high heritability, IASC over human height is likely. Using data from sibling pairs from the Wisconsin Longitudinal Study, we present evidence for IASC over height: in shorter sibling pairs (relatively) more reproductive success (number of children) was obtained through the sister than through the brother of the sibling pair. By contrast, in average height sibling pairs most reproductive success was obtained through the brother relative to the sister. In conclusion, we show that IASC over a heritable, sexually dimorphic physical trait (human height) affects Darwinian fitness in a contemporary human population.

Keywords: stature; sexual antagonism; reproductive success; genetic conflict

1. INTRODUCTION

Because of their different life histories, selection pressures often differ between the sexes [1–3]. Sex-specific selection for different trait optima, sexually antagonistic selection (SAS), can be resolved through the evolution of sexual dimorphism [3–5]. However, the sexes share most of their genome, which can constrain the evolution of sexual dimorphism, thus preventing the sexes from reaching their sex-specific optima. The resulting intralocus sexual conflict (IASC) can be an important determinant of fitness variation [3,5] and has been postulated to underlie variation in reproductive success [6] and its components [7] in human populations.

The widespread existence of sexual size dimorphism suggests that IASC is at least partly resolved in response to sex-specific selection on size [4,5]. However, IASC will persist when current levels of sexual dimorphism are insufficient to allow both sexes to attain their sex-

specific optima, leading to ongoing selection for increased sex-dimorphism [5]. Recent studies suggest that IASC could exist for human height, at least in Western populations, given that the selection pressures on height differ between the sexes. In women, a negative relationship between height and reproductive success is found, selectively favouring those women of short height (reviewed in [8]), whereas in men a curvilinear association is more prevalent (reviewed in [9]), such that average height men have more reproductive success than either shorter or taller men (see [8–10] for reviews of the potential mechanisms giving rise to these divergent selection pressures). Given the SAS on height and the high heritability of height ($h^2 > 0.8$ in industrialized countries; [11]), IASC is likely to persist.

In this study, we examined whether there is IASC over human height using data from the Wisconsin Longitudinal Study (WLS). We focus on sets of siblings (the main respondent plus one randomly selected sibling) and postulated that the relative contribution of the brother and sister to the combined reproductive success (i.e. the number of children) depends on height. More specifically, we postulate that in short sibling pairs the sister would contribute most to reproductive success, while the brother would contribute most to reproductive success in average height sibling pairs.

2. MATERIAL AND METHODS

(a) Sample

The WLS is a long-term study of a random sample of 10 317 men and women, born between 1937 and 1939, who graduated from Wisconsin high schools in 1957 [12]. Survey data were collected at several time points, covering almost 50 years of the participants' lives—see [8,9,12] for discussion of representativeness and other features of this sample. In 1975, respondents of the WLS were asked about their full siblings, and in 1977, 1994 and 2005 one of these siblings (randomly selected) was interviewed. In this study, we included data on the respondents and their selected sibling (we included siblings who were selected in 1975 and responded in 1994). Sex, number of living siblings and year of birth of the sibling were obtained in 1975. Height and the number of biological children ever born (our measure of reproductive success) were obtained by combining data from waves 1994 and 2005, whereas birth order was obtained in 2004 (original respondent) and 2005 (selected sibling). As selection pressures can vary over time and height tends to increase with year of birth, we only include full siblings who differed no more than 10 years in age with the main respondent.

(b) Analyses

We first examined whether selection pressures on the siblings were similar to those previously described [8,9], by using Poisson regressions. To examine IASC, we first standardized height within each sex and calculated the average standardized height across each sibling pair. We used this average to characterize height of both siblings, because it is a better indicator of the phenotype their shared genome codes for than the height of either sibling in isolation. We then regressed reproductive success (number of reported children ever born) against height (squared), sex and their interactions, while controlling for confounding variables (see below). On the basis of our predictions, we expected a significant interaction between height of the sibling pair and the sex of the individual in the sibling pair on reproductive success, indicating IASC. More specifically, we expected an interaction between sex and the squared term of height, because of the curvilinear effect of height found for men [9]. Poisson mixed models were used, with a random intercept at sibling pair level, to account for statistical non-independence of sibling pair members. All analyses were performed in R v. 2.13.1 [13].

3. RESULTS

Descriptive statistics for the respondents and their siblings are provided in the electronic supplementary material, table S1. In total, there were 808 brother–brother, 996

Table 1. Mixed model Poisson regression parameter estimates ($\pm$s.e.) and p-values (based on Z-value) for the effect of the average height and height^2 of the sibling pair (standardized), sex of the individual and their interactions on the number of children, while controlling for birth year (centred), birth order and the number of living siblings ($n = 6280$).

	parameter estimates ($\pm$s.e.)	p-value
intercept	0.827 (± 0.020)	<0.001
sex (ref. cat $=$ male)	0.060 (± 0.023)	0.002
pair height	-2.68×10^{-5} ($\pm 1.43 \times 10^{-2}$)	0.998
pair height^2	-0.037 (± 0.012)	0.002
sex $\times$ pair height	-0.048 (± 0.019)	0.011
sex $\times$ pair height^2	0.042 (± 0.016)	0.008
birth year	-0.032 ($\pm 2.31 \times 10^{-3}$)	<0.001
birth order	-8.36×10^{-3} ($\pm 6.38 \times 10^{-3}$)	0.190
no. living siblings	0.040 ($\pm 4.68 \times 10^{-3}$)	<0.001
random effect	parameter estimates ($\pm$s.d.)	
sibling pair	0.010 ± 0.102	

sister–sister and 1718 opposite-sex sibling pairs. Heights between siblings correlated positively (all r's > 0.45; see the electronic supplementary material, table S2).

Similar to effects observed in the primary respondents [8,9], we found SAS on height for the siblings of the respondents. In male siblings, height was curvilinearly related to the number of children when controlling for birth year (Poisson parameter estimate ($\pm$s.e.) for height: 0.22 (± 0.10); $z = 2.19$; $p = 0.028$; for height^2: -6.20×10^{-4} ($\pm 2.80 \times 10^{-4}$); $z = -2.22$; $p = 0.027$). Similarly, we found a negative effect of height on number of children in sisters (-7.63×10^{-3} ($\pm 2.33 \times 10^{-3}$); $z = -3.28$; $p = 0.001$; see the electronic supplementary material, figure S1 and table S3 for full models).

In support of IASC, we found a significant interaction between the average height of the sibling pair (squared) and the sex of the sibling on the number of children (table 1 and figure 1). In shorter sibling pairs (relatively) more reproductive success was obtained through the sister than through the brother of the sibling pair (figure 1). By contrast, in average height sibling pairs most reproductive success was obtained through the brother relative to the sister. These effects persisted after controlling for birth year, birth order and the number of siblings. Excluding these confounds, restricting the analyses to opposite-sex sibling pairs, using the standardized heights of both siblings in the sibling pair rather than their average height, or doing an analysis similar to Garver-Apgar *et al.* [7] yielded very similar results (see the electronic supplementary material, table S4–S7).

The lower number of children reported by men compared with women (see the electronic supplementary material, table S1 and figure S2) is probably a consequence of men under-reporting previous marital and non-marital births, and the under-representation of previously married men compared with previously married women [14]. However, we previously showed that male height was not associated with the number of marriages or the likelihood of marriage [9], making it unlikely that our results will be strongly affected by this limitation. Height may, however, be positively related to success in siring extra-pair children (as discussed in [9]), which are likely to be under-reported. Nevertheless, because extra-pair children generally occur at low frequency [15] and because IASC is most pronounced
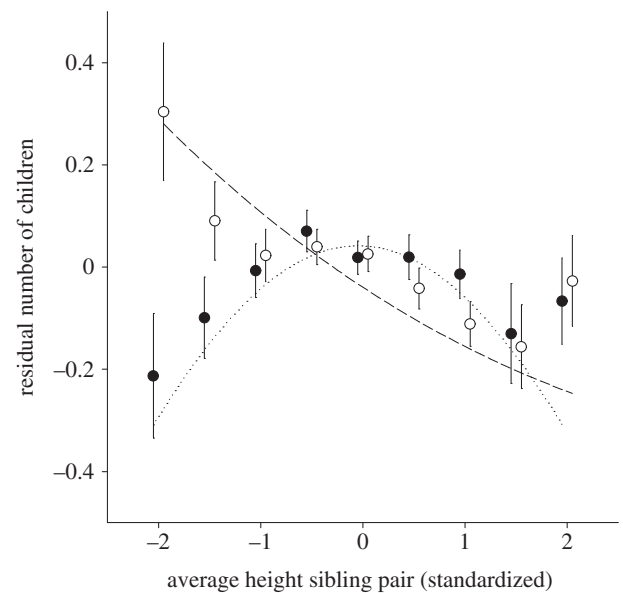
Figure 1. Average height of the sibling pair (average over heights standardized per sex) and the number of children (mean residual $\pm$ s.e.) through the brother (filled circles) and the sister (open circles; see table 1 for controls; fitted lines calculated for means of covariates). The predicted optimum for males was exactly at average standardized sibling height (0). Height was divided in bins of 0.5 s.d. Bins ≤ -2 and ≥ 2 were pooled. See electronic supplementary material, figure S2 for raw data.

in short sibling pairs (figure 1) we consider it unlikely that including extra-pair children would change our result.

A potential solution to IASC is biasing the sex-ratio of the offspring [16], but a logistic regression revealed that the average height of a sibling pair was not related to the proportion of sons in the family, providing no evidence for sex-ratio biasing in response to body size (parameter estimate ($\pm$s.e.): -0.22 (± 0.19); $\chi_1^2 = 1.32$; $p = 0.251$; $n = 3522$).

4. DISCUSSION

These results show, to our knowledge for the first time, that SAS on a human trait can result in IASC. In line with previous studies [8,9], we show that shorter women and average height men are obtaining the

highest reproductive success, indicative of SAS. These selection pressures are sufficiently different to result in significant IASC as in shorter sibling pairs more reproductive success was obtained through the sister than through the brother of the sibling pair. By contrast, in average height sibling pairs most reproductive success was obtained through the brother relative to the sister. We previously showed that the relationship between height and reproductive success in our sample is representative of contemporary Western populations [8,9] suggesting that IASC is the norm in such populations. The IASC, combined with the high heritability of height [11] indicates that neither sex is likely to evolve their sex-specific optima for height. The extent to which IASC exists in non-Western populations is difficult to assess as selection pressures may differ in these populations [8–9] and heritability's are generally lower in resource poor environments [11].

Despite substantial sexual dimorphism in human height ($\pm 8\%$; [4]), our result emphasizes that IASC is not fully resolved (in line with [5]). Why then does IASC persist over human height? First, height is a highly polygenic trait, and the result of interactions between numerous underlying genes [17] distributed over the autosomal and sex chromosomes. Therefore, sex-linkage of height genes can only partially resolve IASC [3]. Additionally, only about one-fifth of all human autosomal gene loci are estimated to be adjacent to binding sites indicative of sex-specific expression [18], rendering it likely that height loci are simply too numerous to all accumulate at upstream DNA binding sites that facilitate sex-specific expression. As a result, it seems unlikely that IASC over height will be resolved rapidly. Biasing the sex ratio provides an alternative means to resolve IASC over body size [16]. Yet, we found no evidence for a relationship between height and sex-ratio (in line with [19], using a much larger sample), indicating that, at least with respect to height, humans do not bias their sex-ratio to reduce IASC.

IASC has previously been postulated to underlie variation in reproductive success (and its components) in humans. First, male homosexuality was hypothesized to be a consequence of alleles that are beneficial to female fecundity but detrimental to male fecundity [6]. Second, variation in sibling attractiveness has also been attributed to IASC [7]: physically and hormonally masculine men and women rated their brothers as more attractive than their sisters. The latter result may be contributing to the IASC observed in our study, as height is associated with attractiveness [10]. On the basis of these results, one would predict that shorter females and average height males would be favoured by the opposite sex. We extend these studies by showing that SAS acts on a heritable, sexually dimorphic physical characteristic (human height) and results in IASC, affecting Darwinian fitness as measured by number of children.

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