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Genetic Influences on the Development of Grip Strength in Adolescence

Joshua Isen, Matt McGue, and William Iacono

Department of Psychology, University of Minnesota, Twin Cities

Abstract

Enhanced physical strength is a secondary sex characteristic in males. Sexual dimorphism in physical strength far exceeds sex differences in stature or total body mass, suggesting a legacy of intense sexual selection. Upper-body strength is a particularly promising marker of intra-sexual competitiveness in young men. Consequently, it is assumed that sex-influenced gene expression contributes to the development of physical strength. It is unclear, however, whether the underlying sources of individual differences in strength development are comparable across sex. We obtained three measurements of hand-grip strength (HGS) over a six-year period spanning adolescence in male and female same-sex twins ($N = 2,513$). Biometrical latent growth models were used to partition the HGS variance at age 11 (intercept) and its growth over time (slope) into genetic and environmental components. Results demonstrated that variance around the intercept was highly heritable in both males and females (88% and 79%, respectively). In males, variance around the slope exceeded that of the intercept, while the reverse held for females. Additive genetic effects accounted for most (80%) of the variance around the slope in males, but were of less importance in females (heritability = 28%). Absolute genetic variance around the slope was nearly nine-fold higher in males. This striking disparity suggests that the developmental processes shaping HGS growth are different between the sexes. We propose that this might account for the sex-specific pattern of associations between HGS and external measures (e.g., digit ratio and physical aggression) typically reported in the literature. Our results underscore the role of endogenous androgenic influences in the development of physical strength.

Keywords

physical strength; heritability; growth; sexual dimorphism

Genetic Influences on the Development of Grip Strength in Adolescence Sexual dimorphism in body size is quite small in humans, especially when viewed within the context of other great apes (Plavcan, 2012; Plavcan & van Schaik, 1997). Although a species-typical mating system cannot appropriately characterize humans, it is apparent that men display a considerable degree of pair bonding, as reflected by the fact that men are merely 7–8% taller and 15–20% heavier than women. These observations have led some authors (e.g., Wood & Eagly, 2002) to dismiss the possibility that male-male competition for mates was a pervasive

sexual selection pressure in the Pleistocene environment of evolutionary adaptedness. Total body size, however, is a very crude indicator of a man's capacity for physical competitiveness. It is likely that reproductive fitness in men was related to fighting success, which, in turn, is more dependent on muscular strength than overall body mass. Indeed, sexual dimorphism in fat-free (muscle) mass far exceeds that for total body mass. Upper-body muscle mass is approximately 75% higher in men than women (Lassek & Gaulin, 2009).

As a major secondary sex characteristic, upper-body strength follows a different developmental course in males and females. Boys enjoy minimal advantages in physical strength over their female peers during much of childhood (Butterfield et al., 2009; Molenaar et al., 2010). Sexual dimorphism dramatically increases in middle adolescence – precisely when sexual behaviors and mating opportunities become palpable. From a functional evolutionary perspective, enhanced upper-body strength was likely shaped by recurrent male-male competition for mates in our hominid ancestors (Geary, 2010; Lassek & Gaulin, 2009; Sell, Hone, & Pound, 2012). Hand grip strength (HGS), in particular, is a potent indicator of physical competitiveness. Although it is most directly controlled by muscle fibers in the upper limbs, it is highly correlated with overall body strength (Wind, Takken, Helders & Engelbert, 2010). Furthermore, HGS contributes to proficiency in the use of thrusting tools and clubs – weapons which were highly useful in agonistic encounters during the Pleistocene (Geary, 2010; Young, 2003). Given the differing biological roles of males and females – the latter are specialized for gestation and parenting – the benefit-cost ratio of physical confrontation is much more advantageous in males. Consistent with this behavioral adaptation, large increases in lean body mass accompany secondary sexual development of males but not females (Kirchengast, 2010).

The idea that intrasexual competition was responsible for sexual dimorphism in physical strength is supported by the sex-specific pattern of correlations between HGS and various behavioral measures. In males, but not females, HGS is positively associated with a past history of physical aggression and lifetime number of sexual partners (Gallup, White, & Gallup, 2007). These associations implicate testosterone as the mediating variable, given the latter's role in affecting courtship/mating behaviors and intrasexual competition.

Previous literature demonstrates profound sex differences in the HGS of young adults, with scarcely any females exhibiting a grip strength that matches the typical (median) male. In a large community sample of adults aged 20–25, the highest performing female corresponded to the 28th percentile of the male distribution (Leyk et al., 2007). Even when examining a highly select group of female athletes (elite handball and judo players), the top female only exceeded 58% of the unselected male participants. This striking difference between typical males and females strongly suggests that sex-influenced genetic mechanisms underlie the development of HGS.

A trait is sex-influenced (or sex-limited) when the processes governing its phenotypic expression differ between males and females.¹ Phenotypic expression of a trait might differ due to an interaction of sex hormones with genes (sex-influenced gene expression) or an interaction of gender with environmental exposure (see Isen & Baker, 2008). Sex-influenced

genetic mechanisms are typically involved in the development of secondary sex characteristics, in which hormones induce change during and after puberty. Vocal pitch is a prime example; increases in sex hormones during puberty stimulate the lengthening of the pharynx and growth of the vocal cords in the larynx, most prominently in males (Fitch & Giedd, 1999).

A classic indicator of sex-influenced processes is a difference in trait variance between males and females. One of the outstanding features of HGS – and indeed, most strength characteristics – is the much higher variability observed in males. The variance in HGS of young men is at least twice that of young women (Hanten et al., 1999; Leyk et al., 2007; Massy-Westropp et al., 2011; Mathiowetz et al., 1985; Mullerpatan, Karnik, & John, 2013; Werle et al., 2009). This suggests that the processes underlying the development of physical strength are different between the sexes. Sex differences in the variance of HGS manifest at puberty, and increasingly diverge as males advance through adolescence (Mathiowetz, Wiemer, & Federman, 1986; Butterfield et al., 2009). Thus, it appears that the masculinizing agent(s) contributing to higher mean HGS also induce greater variability.

Sex-influenced processes can also be inferred on the basis of sex differences in the relative magnitude of genetic and environmental influences. It is unclear whether the excess HGS variance in males arises from genetic and/or environmental causes. The classic twin design, by exploiting differences in genetic similarity between monozygotic and dizygotic twins, can help determine the relative importance of genetic and environmental factors in the development of HGS.

If individual differences in sensitivity and exposure to androgens are important determinants of HGS, then it is reasonable to assume that the higher phenotypic variance in males is due to novel genetic effects introduced at puberty. This might lead, not only to more absolute genetic variance, but also to a greater *relative* role of genetic influences (i.e., higher heritability). On the other hand, it is possible that the greater male variance is due to increased environmental influences. For example, boys are more likely to participate in sports and other extra-curricular activities involving upper-body strength (Thomas, Nelson, & Church, 1991). There are substantial individual differences in physical training during adolescence, perhaps more so in males (given the wider opportunity afforded to them in pursuing their athletic endeavors). Greater variation in physical training could contribute to excess HGS variance via direct environmental effects. This could lead to a situation in which the overall phenotypic variance is higher in adolescent males, yet the proportion due to genetic factors (heritability) is lower.

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¹Sex-limited and sex-influenced traits are sometimes used interchangeably in the literature. However, the former should be reserved for characteristics that emerge in one sex only (e.g., breast development in girls). Most secondary sex characteristics show some representation in both sexes. For example, there are noticeable individual differences in women's vocal pitch and amount of body hair. Even if gene expression is higher in males (e.g., during puberty), the particular genes affecting these traits are likely the same in males and females. This type of gene expression is sometimes described as scalar sex-limitation (Neale & Cardon, 1992), but we refer to it as sex-influenced gene expression.

Heritability of HGS

Heritability estimates obtained from large, representative studies of adult male twins are typically in the 50–60% range (Frederiksen et al., 2002; Reed, Fabsitz, Selby, & Carmelli, 1991; Silventoinen, Magnusson, Tynelius, Kaprio, & Rasmussen, 2008). However, there is a dearth of large-scale investigations examining the genetic/environmental structure of grip strength in childhood or adolescence (see Beunen & Thomis, 2000, for a review of several studies with small sample sizes). There is also a gap in the literature regarding sex differences in the heritability of HGS. One exception is Frederiksen et al. (2002), who observed a similar pattern of genetic and environmental influences on HGS in older men and women. Although not directly indexing HGS, Peeters et al. (2005) found that genetic influences on the stability of upper-body strength during adolescence were of greater importance in males than females.

Present Study

To our knowledge, no study has yet examined genetic influences on the development of HGS in adolescence. Although a prior study examined familial influences on change in HGS over a 7-year period, the sample design (use of nuclear families) precluded a disentangling of shared environmental and genetic influences (Katzmarzyk, Gledhill, Perusse, & Bouchard, 2001). Moreover, the authors did not focus on any particular developmental period. Adolescence is a period in which physical strength characteristics begin to dramatically diverge between the sexes. As such, it provides the ideal window to delineate the genetic/environmental causes of sexual dimorphism in HGS. We used a latent growth design to model the development of HGS across a six-year period spanning from early to late adolescence. By using a large representative sample of twins from approximately age 11 to 17, we sought to determine whether the genetic and/or environmental sources of HGS growth are similar between males and females.

Method

Participants

Participants were drawn from two population-based samples of twins from the state of Minnesota. These samples were separated in time by approximately a decade, but shared highly similar testing protocols. In both cases, the twins were recruited at age 11 to take part (with their caregivers) in a comprehensive day-long laboratory visit, and later followed up at approximately 3-year intervals. One sample ($n = 1527$) comprised the younger cohort of the original Minnesota Twin Family Study (MTFS; Iacono & McGue, 2002). Their intake visit occurred between 1990 and 1996. Most of these individuals have since participated in follow-up assessments during adulthood (up to age 29), but grip strength measurements were only collected during the first three assessments in childhood/adolescence.

The more recent sample ($n = 1000$) participated in the Enrichment Study (ES) component of the MTFS (Keyes et al., 2009), starting in the years 1999–2006. As of now, these participants have returned for a maximum of two follow-up visits, henceforth referred to as “follow-up 1” (FU1) and “follow-up 2” (FU2). The MTFS was designed to investigate the

antecedents of substance use disorders and other externalizing outcomes. (The “enriched” nature of the ES sample refers to the fact that half of the participants were selected because they displayed childhood risk factors for substance dependence.) Both the original MTFs and ES samples were further subdivided into male and female cohorts. All twin pairs consisted of like-sex siblings.

Mean age at testing during the intake, FU1, and FU2 assessments was nearly identical across the four cohorts. Most participants returned to the laboratory for two follow-up visits, with total in-person return rates of 83% and 71% at FU1 and FU2, respectively. A major cause of attrition was that families relocated from the state of Minnesota, and thereby could only participate via phone assessment. Notwithstanding the slightly different recruitment strategies and 10-year gap, the two MTFs samples were highly similar with respect to most characteristics, including grip strength (data available upon request). Both samples were largely of European-American origin, but a higher proportion of the ES sample was non-Caucasian (9%) relative to the original MTFs sample (3%). This increased share of non-Caucasians is consistent with the progressively greater ethnic/racial diversity of the Minnesota population in the years covered. We combined the samples when conducting all statistical analyses.

Zygosity determination was based on an algorithm of sibling resemblance across various anthropometric indices (cephalic index, fingerprint ridge count, and ponderal index) as well as subjective evaluations of physical similarity. Molecular genetic confirmation was used to resolve any discrepancies between these estimates. A total of 788 monozygotic (MZ) and 466 dizygotic (DZ) twin pairs were identified. We obtained grip strength measurements from 2513 individuals, including five triplets. (The last-born member of each triplet set was omitted from genetic analyses).

Procedure

Grip strength was measured throughout the testing period (1990–2012) using a set of Lafayette hand dynamometers (Lafayette Instrument Company, USA). Two different dynamometer models were used, depending on the measurement occasion. At Intake, participants were tested using Model #78011, which is a pediatric hand dynamometer with a measuring range of 0–50 kg. (The maximum grip strength observed at Intake was 38 kg.) At FU1 and FU2, we administered Model #78010, which has a measuring range of 0–100 kg. All twins were tested during a day-long visit to the Minnesota Center for Twin and Family Research. Grip strength measurements occurred during the psychophysiological component of the laboratory visit, and coincided with other anthropometric measurements such as physical stature and body mass. Participants were tested in the morning hours during their Intake and FU2 assessments. At FU1, time of testing varied, such that some individuals were tested in the morning and others in the afternoon.

Both siblings in each twin pair were tested in the same room within each other’s presence. Laboratory staff first selected one twin to undergo measurement, and did not initiate testing with the second twin until all HGS measurements from the first twin were complete. During this procedure, participants positioned their arms to the side while standing. They were instructed to place their arms in a neutral position, with the elbows slightly bent and the

palms facing towards their side. Without resting their arm against their body, they were told to squeeze the hand dynamometer as tightly as possible. Four trials were administered in an alternating fashion between hands. For the 1st trial, they squeezed it with their dominant hand. Next, they switched it to their non-dominant hand. After a brief rest period, this procedure was repeated, so that a total of two measurements were obtained from each hand. Laboratory staff recorded all HGS measurements to the nearest kilogram-force unit (henceforth expressed as kg). The maximum value obtained across the four trials was used as input for all analyses.

Statistical modeling

A maximum of three data points – corresponding to HGS measurements at Intake, FU1, and FU2 – was available for each participant. In order to examine inter-individual variation in HGS development, we fit latent growth curve (LGC) models to the longitudinal data (McArdle, 1986; Meredith & Tisak, 1990). Latent growth curve models are advantageous because they allow one to conceptualize development as a single (common) growth process rather than invoking multiple changes/innovations over time (Neale & McArdle, 2000). Two latent factors – an intercept and a slope – are estimated to account for variation in the time-ordered variables. The intercept accounts for the initial HGS level, and the slope is a constant growth process responsible for change over time. As with any latent factor model, we estimated variable-specific measurement error. These time-specific variances are residual, i.e., unexplained by the intercept and slope factors.

Under the LGC framework, the intercept and slope factors have both a “fixed” mean and a variance. The “fixed” effects correspond to the initial level and the average amount of change observed in the entire group (i.e., grand means). Individual differences in the initial level and amount of change are captured by the intercept variance and slope variance, respectively. Given that children are developing from a juvenile state to a more mature one, the fixed slope parameter will undoubtedly be positive. However, some individuals (particularly girls) may fail to demonstrate gains in HGS during adolescence. The slope variance encompasses the fact that growth trajectories are not the same across individuals.

The association between individual differences in the intercept and slope can also be estimated. For example, individuals who are relatively strong at Intake might be expected to show less increase in HGS over time. Perhaps these individuals happened to experience an earlier growth spurt, and their later maturing peers will eventually “catch up” (Taeymans, Clarys, Abidi, Hebbelinck, & Duquet, 2009). If this is the case, then the intercept-slope correlation would be negative.

An important element of LGC models is the measurement of time. Interpretation of the slope depends on how one conceptualizes time. One can estimate time as a function of individuals’ chronological age or as a fixed measurement wave (Intake, FU1, and FU2). Although there was variation in participants’ ages at each time point (spanning approximately two years), certain age periods received very scant coverage. In particular, only two participants were tested within a half-year following their 13th birthday. This is typically an intensive period of physical maturation. Consequently, we observed an artificial discontinuity in mean HGS between individuals aged 12 and 13. Participants who were aged

13 (but chronologically much closer to 14) closely resembled their 14-year-old peers in grip strength. To remedy this gap in coverage, we treated each measurement wave (Intake, FU1, and FU2) as the relevant time unit. Grip strength was then residualized with respect to age. The latter step is important because, if left uncorrected, age variation will misleadingly contribute to estimates of shared environmental influences on HGS.

In LGC models, the slope parameter is akin to fitting individual regression lines to each participant's vector of data. One advantage of using a structural-equation modeling approach is that the slope can be estimated for each participant even if not all data points are present. That is, we can use full-information maximum likelihood techniques to impute intercept/slope parameters for individuals who did not participate in all three assessment waves. Imputation is appropriate if the data are assumed to be "missing at random" (Little & Rubin, 2002). This assumption is tenable as long as the missing grip strength is unrelated to the reasons for missingness. It is unlikely that the source(s) of attrition in an adolescent twin sample would be related to grip strength.

LGC modeling was implemented in Mplus Version 6 (Muthen & Muthen, 2010) by means of maximum likelihood estimation. We estimated LGC parameters separately in first-born and second-born members of each twin pair. The appropriateness of this model was determined with respect to a null model that does not assume any growth in HGS. We then evaluated the fit of more reduced (parsimonious) models using a likelihood ratio test, which is based on twice the difference in log-likelihoods between nested models. The difference in log-likelihood (goodness-of-fit) between models is distributed as chi-square. The viability of a reduced model depends on whether the gain in parsimony (i.e., number of additional degrees of freedom) outweighs the accompanying deterioration in fit. For example, we examined the hypothesis that MZ and DZ twins are derived from the same population by testing a model in which parameters were constrained to equality across zygosity status and birth order. We also tested the hypothesis that HGS development is linear over time, as well as the assumption that the slope and intercept are uncorrelated.

Genetic Analyses

After establishing an appropriate phenotypic model of HGS development, we partitioned the latent growth factor and residual variances into genetic and environmental components (see Neale & McArdle, 2000). The expected twin covariance matrices were based on standard assumptions in biometric model-fitting (Neale & Cardon, 1992). This procedure exploits the classic twin design – a natural experiment in which the level of genetic relatedness between individuals varies while the effects of family environment do not. It capitalizes on the fact that MZ and DZ co-twins are reared in the same environment, but differ in their degree of genetic similarity. Namely, we modeled the twin covariance as due to two latent factors: additive genetic (A) and shared environmental (C). Genetic factors are those that render MZ co-twins more similar to one another than DZ co-twins. Under an additive genetic model, the genetic correlation is fixed at 0.50 in DZ twins to reflect the fact that they share half of their segregating alleles on average. Since MZ twins inherit all of their genetic material in common, the MZ genetic correlation is set at unity. Shared environmental effects refer to factors that induce sibling resemblance regardless of genetic similarity, and are thus

perfectly correlated in twins reared together. Shared environmental influences on HGS might constitute family-wide factors such as household dietary quality and neighborhood access to parks/recreation. Any variance unique to individuals, including measurement error, is referred to as nonshared environmental effects (E).

The biometrical LGC model is depicted at the twin pair level in Figure 1. The time-specific residual variances, in addition to the intercept and slope variances, are partitioned into the three aforementioned ACE components. For simplicity, the association between intercept and slope is not illustrated in Figure 1, but any emergent intercept-slope correlation can be decomposed into genetic and environmental sources using a Cholesky factorization (Reynolds et al., 2005). The paths linking the growth factors to the three measurement occasions (Intake, FU1, and FU2) are factor loadings. To establish the initial measurement occasion as the intercept, all relevant factor loadings are fixed at one. Slope loadings are fixed at 0 and 1 for the Intake and FU2 variables, respectively. This allows for easier interpretation of the slope mean, which is simply reduced to the difference in HGS between the beginning and end of the study. The factor loading of the middle (FU1) time variable was estimated separately in each sex to allow for sex-specific nonlinear growth.

Results

Grip strength was symmetrically distributed within both sexes at each time point. In all cases, the mean value was within one integer of the median. Mean values of HGS and the accompanying standard deviations (unadjusted for variation in age) are listed in Table 1. Nearly two-thirds of participants ($n = 1642$) possessed data across all three assessments. Participants who failed to return to the laboratory for their follow-up assessment(s) did not significantly differ in HGS (at intake) from those who participated in all three assessments ($p = .24$, data not shown).

As a manipulation check, we examined qualitative patterns of change. Of those who possessed HGS data at both the intake and conclusion of the study ($n = 1772$), nineteen individuals (1.1%) showed a slight decline across the six-year span, and thirteen individuals showed no change. All but one of these participants was female. One participant who showed particularly steep drops in grip strength (scoring more than three *SD* units below the mean at both follow-ups) was omitted due to a diagnosis of juvenile arthritis.

There was considerable overlap between the sexes in the distribution of HGS at Intake. The similarity of boys' and girls' variances permits a straight forward calculation of effect size; Cohen's *d* was modest (~ 0.22). Put another way, 38.9% of girls met or exceeded the median male's HGS value (22 kg). The male and female distributions were very different at subsequent assessments. By age 14–15, only 6.7% of females ($n = 71$) equaled or exceeded the median male's HGS. At the final assessment, this number was reduced to six females (0.6%), of whom only one exceeded the male median of 44 kg. Over the six-year span, males doubled their HGS, while females' levels increased by 42%.

Age was a significant correlate of HGS in both boys and girls at Intake, Pearson's $r = .24$ and $.27$, respectively. Given that variation in age is an extraneous source of error rather than

a legitimate source of familial influence, we regressed HGS on participants' ages separately in males and females. The unstandardized residuals from the resultant regression equations were used as input in the latent growth curve models. In order to retain information about mean level, we added the mean predicted HGS values to these residual scores. Thus, the means are identical to those reported in Table 1, but the standard deviations are smaller because we removed variation due to age (which, in turn, captures individual differences in biological maturity). Although age was not a significant correlate of HGS at FU2 (in the case of females), we nonetheless residualized HGS with respect to age at all three assessments for the sake of consistency.

Latent Growth Curve Model

In a multi-group structural equation model, we evaluated the appropriateness of fitting growth curve parameters to the HGS data. Similar to Figure 1, we estimated the means, variances, and covariances of the intercept and slope factors as well as the time-specific residual variances. In LGC models, growth can be nonlinear, as appears to be the case in females (see Table 1). That is, more growth occurred during the three-year period between Intake and FU1 than for the period between FU1 and FU2.

Our model-fitting procedure is summarized in Table 2. We evaluated eight different hypotheses, starting with the premise that a LGC framework is viable (Model 2). The fit of this model (and all subsequent ones) was compared to a baseline "saturated" model (Model 1), where no particular structure was imposed on the data; i.e., the means, variances, and covariances of the three HGS measurements were freely estimated. We modeled separate growth curves in four groups: MZ males, DZ males, MZ females, and DZ females. Separate parameters were estimated for first-born and second-born twins in each pair. The slope loading of the second time-point was freely estimated in order to account for the possibility that growth is nonlinear. (A value of 0.50 would be expected under assumptions of linearity). The hypothesis that a LGC framework conforms to the data was tenable, $p = .15$. This outcome is not surprising, given that the HGS means and variances progressively increased over time.

We evaluated hypotheses about the tempo of growth via three submodels that are directly nested within Model 2. These submodels only differed with respect to the middle time-point, which indexes the proportion of total growth occurring by FU1. Although close to being linear in males, setting this value to 0.50 in Model 3 led to a significant decrement in fit, $p < .01$. Suffice it to say, the hypothesis of linear growth in females could also be rejected (Model 4). Based on our empirical observations, we thereby fixed the middle time point to 0.53 in males and 0.63 in females with little loss in fit (see Model 5).

In Model 6, we evaluated the hypothesis that first-born and second-born twins as well as MZ and DZ twins are derived from the same population. That is, we sought to ensure that the means and variances of the growth factors are equivalent across all four categories of twins (MZ first-born, MZ second-born, DZ first-born, and DZ second-born) within each sex. (Twin covariances were allowed to differ across MZ and DZ groups.) The gain in parsimony outweighed the resultant loss in fit ($p = .07$). Although this may seem like a statistically marginal result, we should emphasize that the root mean square error of approximation was

0.029 (90% CI: 0.00 – 0.046), indicating a close fit between the predictions of the model and the observed structure of means and variances.

In order to further simplify the LCG models, we examined whether individual differences in the intercept and slope were related. The slope-intercept correlation was positive in each sex, suggesting that individuals with higher HGS at Intake experience larger gains over time. However, the effects were statistically non-significant ($ps > 0.14$). When we fixed the slope-intercept correlation (including its cross-twin cross-trait association in each zygosity group) to zero, the drop in fit was negligible (see Model 7). Parameter estimates from this model are reported in Table 3.

The mean difference in the intercept between males and females was approximately one kg. Although the growth processes are inherently different across sex (owing to the fact that females attain a larger proportion of their total growth by age 14–15), one might still inquire as to whether the intercepts are similar. They are clearly not; both the mean and variance of the intercept were significantly higher in males, as demonstrated by Models 8–9 in Table 2. Intra-sexually, however, males' variance around the intercept was lower than their variance around the slope (see Table 3). This implies that individual differences in HGS in young men stem disproportionately from factors introduced *after* age 11. Females showed the reverse pattern; variance around the intercept was greater than that around the slope.

The slope variance in males exceeded that of females by a factor of three (21.17 vs. 6.84; see Table 3). The interpretability of this greater male variance is dependent on the proportion of total HGS variance accounted for at each time point by the intercept and slope factors. If residual variance at the final measurement occasion is proportionately higher in females, then it would limit the appropriateness of concluding that males manifest thrice the variability in HGS growth as females. This concern is allayed by the fact that the growth factors accounted for an equivalent 67% of the variance at FU2 in both sexes. (The proportion of residual variance is given as .33 in Table 3.) The proportions of residual variance at Intake and FU1, however, differed considerably between the sexes.

Intraclass twin correlations for all parameters are listed in Table 4. Differences between the MZ and DZ correlation coefficients hint at the relative importance of genetic and environmental influences. For males, MZ correlations were approximately twice as high as the corresponding DZ correlations for both the intercept and slope factors, indicating strong additive genetic influences. In females, the MZ correlation was nearly twice as high as the DZ correlation for the intercept, but less so for the slope. The residual variances followed a similar trend as the slope. In all three cases, MZ female correlations were much lower than twice the magnitude of the DZ correlations, indicating the importance of shared environmental influences. In males, there was no twin resemblance for the residual variance at Intake. At subsequent measurement occasions, MZ correlations were higher than the DZ correlations, especially at FU2.

Genetic Modeling

We partitioned the LGC variance components into genetic and environmental (ACE) sources, in the manner depicted in Figure 1, which is based on the assumptions of Model 7

from Table 2. (All free parameters were assumed to differ across sex, but were tested for equality whenever possible.) Standardized variance components from this genetic analysis are reported in Table 5. As suggested by the twin correlations, individual differences in the intercept were highly heritable in both sexes. Proportions of additive genetic influence (A) were .88 and .79 in males and females, respectively (see Table 5). These heritability estimates could be equated across sex without significant reduction in fit; $\chi^2 = 0.60$, $p = .44$. However, individual differences in the slope were highly heritable in males only. In females, the slope variance was certainly familial to some extent (only 42% of the variance was due to E), but neither estimate of A nor C was significant. The residual variances showed similar patterns, in which A and C accounted for approximately equal proportions of the variance. Genetic factors were not singly important in any instance. In males, the residual variances at FU1 and FU2 showed moderate heritability coefficients.

Absolute amounts of genetic variance reveal a starker disparity between males and females. While the heritability of HGS growth was almost three times higher in males, the absolute genetic variance exceeded that of females by nearly a factor of nine (16.94 vs. 1.92). (Unstandardized estimates of nonshared environmental variance around the intercept and slope parameters were not significantly different across sex, however, $ps > .41$.) Genetic contributions to the residual variances at FU1 and FU2 were also many times higher in males. (Nonshared environmental residual variances were significantly higher at FU1 and FU2 in males as well, $ps < .001$, although virtually identical at Intake, $p = .85$.) In fact, the absolute genetic variance in males at FU1 and FU2 greatly exceeded the *total* phenotypic variance observed in females. This phenomenon is presented in Figure 2, where the expected variance components are graphed at each measurement occasion. Using a factor regression framework, the variance in HGS at each occasion is a linear function of the variance around the intercept (V_i), variance around the slope ($V_s * t^2$), and residual variance (V_{rv}). The contribution of variance in slope at each assessment is proportional to the squared fixed time-point (t) loadings, with the latter assuming values of 0 (Intake), 0.53/0.63 (FU1), and 1 (FU2). In this manner, the additive genetic variance (V_A) in females at FU1 would be obtained as:

$$V_A = V_{Ai} + V_{As} * 0.63^2 + V_{Arv}$$

As can be seen in Figure 2, most of the genetic variance present in females during mid/late adolescence is already established by age 11–12. The age-related increase in variance is mainly due to increases in environmental effects. As a result, heritability of HGS is in the 45–50% range during mid and late adolescence – somewhat lower than the 60% heritability estimate at Intake. In males, the heritability remained consistently high across development (70–76%). This masks the fact that, in absolute terms, genetic and environmental influences increased three-fold during this period.

Adjustment for Body Mass and Stature

Grip strength development parallels growth in other physical characteristics during adolescence. In particular, physical stature and body mass are positively correlated with HGS. Given that adolescent males demonstrate greater gains in body mass and stature, it

may be informative to evaluate sex differences in HGS-specific growth, i.e., independent of changes in overall body size. For this analysis, we residualized the age-corrected HGS data with respect to physical stature and body mass at each assessment wave. Results of these regressions are reported in Table 6. Physical stature and body mass explained twice as much variance in females' HGS, relative to males, at Intake and FU2. We submitted these residual scores to the same biometrical LGM procedure conducted previously. Results of this analysis merely reinforced the observation that additive genetic variance around the slope is nearly an order of magnitude higher in males (14.52 vs. 1.55). Thus, males manifest greater individual differences in HGS growth independently of factors related to overall body size.

Discussion

Most individuals experience gains in physical strength during adolescence. However, there are substantial individual differences in the amount of growth. Surprisingly, very few longitudinal studies have investigated the genetic and environmental sources of adolescent change in physical strength characteristics (e.g., Peeters et al., 2005), and none to our awareness have specifically examined the development of HGS. We employed a biometrical latent growth model to quantify genetic and environmental influences on inter-individual variation in HGS level and change across adolescence. In line with previous studies, the total amount of growth attained between early adolescence and young adulthood was much higher in males. Importantly, males and females showed a dramatic divergence in HGS variance as they proceeded through adolescence.

Genetic influences on the development of HGS, both in absolute and proportional terms, were larger in males. Individual differences in males' HGS growth were overwhelmingly due to additive genetic factors, whereas we could not unambiguously establish a heritable basis for females' HGS development. This suggests that the greater phenotypic variance in men is due to the products of sex-influenced gene expression. A sensible interpretation is that androgen-mediated mechanisms powerfully shape the development of grip strength in males, but are subdued or lacking in females. In particular, absolute genetic influences on HGS growth were nearly nine-fold higher in males. By contrast, genetic influences on the intercept were only 50% higher in males. This developmental pattern implicates testosterone as a prime determinant of male HGS.

At first glance, this fails to explain why there is more variability in males, as opposed to simply higher mean levels. Why should higher exposure to androgens necessarily induce greater variability in males' HGS? Perhaps the reason for this is that young men exhibit a much greater range in testosterone concentrations, with inter-individual variability exceeding that of young women by at least an order of magnitude (Dabbs, 1990). Interestingly, testosterone levels are only moderately (50–60%) heritable in male adolescents (Harris, Vernon, & Boomsma, 1998; Hoekstra, Bartels, & Boomsma, 2006). While some attenuation of heritability is expected on the basis of diurnal variation and assay measurement error, it is likely that environmental influences account for some of the systematic variance in testosterone levels. This could help explain why there was greater absolute environmental variance in young men's HGS. That is, the contribution of a biological variable (testosterone) to HGS need not be transmitted entirely genetically.

Testosterone is well understood to promote the development of lean body mass and other strength-related characteristics (Bhasin et al, 2001). In early adolescent males, inter-individual variation in the development of grip strength is positively correlated with changes in testosterone concentrations over a two-year period (Hansen, Bangsbo, Twisk, & Klausen, 1999). Effects of androgens on HGS might also be exerted organizationally during fetal development. The ratio of the 2nd digit to 4th digit, a putative index of prenatal testosterone concentrations, is related to men's HGS (Fink, Thanzami, Seyde, & Manning, 2006; Hone & McCullough, 2012; Zhao, Li, Yu, & Zheng, 2012; but see Gallup et al., 2007). Notably, there is no association between 2d:4d and HGS in young women (Hone & McCullough, 2012; van Anders, 2007), perhaps because individual differences in androgen exposure among typically developing females are too restricted to influence the course of HGS development (see Collaer, Brook, Conway, Hindmarsh, & Hines, 2009, for an illustrative exception involving females with abnormally elevated adrenal androgens). Moreover, 2d:4d appears to predict physical aggression in male, but not female, youth (Butovskaya, Fedenok, Burkova, & Manning, 2013), which parallels the sex-specific nature of the association between physical aggression and HGS in young adults (Gallup et al., 2007; Sell, Tooby, & Cosmides, 2009).

We should also acknowledge an alternative explanation as to why large sex differences in HGS variance emerge at puberty. Cultural factors and leisure preferences might constrain girls' engagement in physically intense activities, particularly as they become more reproductively capable (Thomas et al., 1991). This could limit the realization of their grip strength potential, resulting in less genetic variance. Reduced participation in physical exercise also leads to attenuated stimulation of upper-limb muscle fibers, which could translate into reduced environmental influences on HGS (Morris, Naughton, Gibbs, Carlson, & Wark, 1997). Indeed, we found more nonshared environmental variance in the HGS of males than females (by a factor of nearly two) during mid and late adolescence. On the other hand, there was little evidence of shared environmental influences on HGS growth. Its absence was most apparent in males. This suggests that family-wide environmental factors related to diet and recreation exert a minimal impact on the development of HGS in the present population.

Sexual Dimorphism

Physical strength is but one of many sexually dimorphic traits in humans. One might inquire as to whether sexually dimorphic characteristics (in general) inherently lend themselves to sex differences in variance. An obvious counterexample is physical stature, in which both the phenotypic variance and heritability are quite similar between males and females at all ages (Dubois et al., 2012; Silventoinen et al., 2003). Thus, the presence of sexual dimorphism is not incompatible with the possibility that similar genetic/environmental processes operate in both sexes. For example, it is possible that men attain greater height than women primarily because the duration of the growth spurt is longer in males— even while the actual hormonal mechanisms that drive vertical skeletal growth are similar across sex. A greater focus on the raw variance components can inform our understanding of the proximate causes of sexual dimorphism. In the case of physical stature, we can safely infer that active testosterone levels (during puberty or later) cannot explain why males are taller

than females.² At the very least, we can conclude that sexual dimorphism in physical stature is caused by different factors than those shaping sexual dimorphism in HGS.

Although the developmental processes responsible for sexual dimorphism in physical strength and stature may differ, the ultimate evolutionary functions may be linked. Physical size and strength characteristics have likely undergone strong sexual selection in our hominid ancestors. In primates, larger male body size is associated with non-monogamous mating systems in which males physically compete against one another for reproductive access to females (Playcan & van Schaik, 1997). Mating patterns of archaic *Homo* species have been characterized as opportunistically polygynous. This is inferred not just on the basis of sexual dimorphism in body size, but also from other genetic/anthropometric criteria (Dupanloup et al., 2003; Nelson, Rolian, Cashmore, & Shultz, 2011). As such, male-male competition for mates was probably a major component of our evolutionary past, with enhanced physical strength providing a critical boost to males' reproductive success. It stands to reason that fighting ability (or the projection of physical threat) was the functional target of intra-sexual selection, rather than body size per se. That is, larger male body size is valuable only inasmuch that it facilitates greater physical dominance.

Another consideration pertaining to inter-male individual differences in physical strength is the link between sexual dimorphism and condition dependence (see Bonduriansky, 2007). Relative to non-sexually dimorphic characteristics, traits exaggerated by sexual selection should show greater sensitivity to environmental quality during development (i.e., more condition dependence). According to this perspective, sexual dimorphism should be enhanced under resource-rich conditions, whereas poor environments should particularly stunt the growth of sexually selected characteristics in males (more so than homologous traits in females). Despite the widespread occurrence of this phenomenon in numerous animal taxa, evidence for this in humans is equivocal (Stinson, 1985). Recent studies on sexual dimorphism in physical stature suggest that male growth is more adversely affected by socioeconomic deprivation (Nikitovic & Bogin, 2013). On the other hand, sexual dimorphism in physical stature remained constant over the course of several centuries in Sweden, despite the dramatic improvement in living conditions during the 20th Century (Gustafsson, Werdelin, Tullberg, & Lindfors, 2007).

The idea that environmental conditions modulate sexual dimorphism in physical strength is difficult to evaluate in contemporary American society, given that under privileged youth tend to develop a *heavier* body mass than their wealthier peers. This is counterintuitive because it stands in contrast to developing societies, where socioeconomic advantage is linked to greater body size and physical strength (e.g., Henneberg, Brush, & Harrison, 2001). Malnutrition is essentially non-existent in the present sample of twins. Nonetheless, we examined our twin data with respect to family socioeconomic status (SES). We averaged across the percentile ranks of household income, highest parental occupational status, and maternal/paternal educational attainment to calculate a SES composite (data not shown).

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²If anything, exogenous administration of testosterone stunts vertical skeletal growth in developing males (Zachmann et al., 1976). The irrelevance of endogenous androgen levels on height attainment is illustrated by studies of males with hypogonadism, such as the historical castrati/eunuchs and individuals with Klinefelter Syndrome (Leroi, 2004; Manning, Kilduff, & Trivers, 2012). Rather than following a female-typical pattern of vertical growth, males with hypogonadism tend to attain an exaggeratedly tall stature.

Boys from lower SES backgrounds showed *greater* growth in HGS (i.e., SES was negatively correlated with the slope factor). Since SES was unrelated to HGS in females, sexual dimorphism reached its maximum among those from the lowest SES strata. This is difficult to reconcile with the literature on condition-dependence.

Limitations

A shortcoming of this study is that we only sampled same-sex twin pairs. Thus, we were unable to evaluate qualitative models of sex-limitation. The use of opposite-sex twins could have allowed us to directly infer whether HGS development is controlled by similar genes in both sexes (Maes et al., 1996), although prenatal hormonal transfer would potentially complicate the picture. One would expect to find higher correlations in same-sex DZ twins relative to opposite-sex twins if different genes are involved in males and females. Had we sampled opposite-sex twins, the presence of a larger phenotypic correlation between same-sex twins would suggest that HGS development is the product of sex-limited (rather than merely sex-influenced) gene expression. However, there is no *a priori* reason to believe that sex-limited gene expression is at work.

Another limitation pertains to our estimation of genetic and environmental influences. These sources of variance are latent (unobserved) factors specific to a given population. The validity and precision of the genetic and environmental parameter estimates are subject to several qualifications. First, the classic twin methodology assumes that genotype-environment correlations are absent, which might not be the case. Another qualification is that one cannot simultaneously estimate non-additive genetic effects and shared environmental effects using the classic twin design. If these two sets of influences are indeed operating, then twin covariance in HGS will incorrectly be attributed to additive genetic sources. This implies that our heritability estimates could be too high.

Our failure to detect shared environmental influences on males' HGS development does not imply that family-wide environmental factors are irrelevant to HGS. Socioeconomic status is strongly linked to HGS in a South African population (Henneberg et al., 2001) and was related (inversely) to HGS in our male twin cohort. Moreover, children from smaller-bodied and/or malnourished populations are physically weaker than children from industrialized Western societies (Malina, Little, Shoup, & Buschang, 1987). Systematic disparities in HGS between these various populations are probably not wholly due to genetic differences between groups.

The present twin sample is derived from an overwhelmingly Caucasian population within a single geographical region. As such, cultural variation might not exert as much influence on the anthropometric characteristics of this sample as would be observed in more heterogeneous populations. Further, the statistical modeling employed in this study assumes the absence of gene-environment interactions, which might not be tenable. Any potential interactions between genetic factors and shared environmental factors (e.g., socioeconomic status or household diet) would be subsumed under genetic influences, thereby underestimating the shared environmentality of HGS.

Concluding Remarks

The present study was motivated by questions as to why men exhibit systematically higher HGS variance relative to women. In general, sex differences in variability are underappreciated in the HGS literature, typically superseded by issues concerning mean differences (sexual dimorphism). We believe that differences in variability are informative, as they provide clues that physical strength is shaped by different developmental processes in men and women. To our knowledge, only one previous study has examined sex differences in the genetic/environmental structure of HGS (Frederiksen et al., 2002). The authors focused on relative proportions of variance rather than absolute variances. Given the presence of large variance differences, it is unclear whether HGS can be considered comparable across sex. Even if it were the case that the heritability is similar in males and females, it would not demonstrate equivalence in terms of the underlying etiology.

This issue is underscored by recent observations that the external correlates of HGS are markedly different between young men and women. In the behavioral literature, HGS is positively correlated with levels of physical aggression and sexual experience in young men but not in young women (Gallup et al., 2007; Sell et al., 2009). Males, but not females, with higher HGS are also perceived as more socially dominant (Gallup, O'Brien, White, & Wilson, 2010). These sex-specific findings likely stem from the fact that upper-body strength in females is not predominately shaped by endogenous androgenic influences. Efforts are currently underway to determine whether the genes contributing to HGS development also exert (pleiotropic) effects on male-typical behavioral traits.

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Fig. 1.

Path diagram of a biometrical latent growth model. Left-hand side represents data from the first-born twin; right-hand side represents the second-born twin. Rectangles denote the measured grip strengths at each assessment: Intake, 1st follow-up (FU1), and 2nd follow-up (FU2). Circles denote latent variables. Fixed effects (grand means) are denoted by the triangle. Double-headed arrows represent twin covariance. Slope regression weight (t) is estimated for the middle time-point. Residual variances (RV) represent time-specific variation unexplained by the growth model; A = additive genetic factors; C = shared environmental factors; E = nonshared environmental factors. Genetic and shared environmental influences on RV are not shown due to space limitations.

Fig. 2.
Unstandardized estimates of expected genetic and environmental variances at each measurement occasion.

Table 1

Descriptive Statistics

Measurement Occasion	Males			Females		
	<i>N</i>	Age <i>M</i> $\pm$ <i>SD</i>	Grip Strength <i>M</i> $\pm$ <i>SD</i>	<i>N</i>	Age <i>M</i> $\pm$ <i>SD</i>	Grip Strength <i>M</i> $\pm$ <i>SD</i>
Intake	1229	11.78 $\pm$ 0.40	21.9 $\pm$ 4.2	1255	11.77 $\pm$ 0.46	20.8 $\pm$ 3.9
1 st Follow-up	1022	14.86 $\pm$ 0.47	33.5 $\pm$ 7.3	1052	14.82 $\pm$ 0.54	26.4 $\pm$ 4.4
2 nd Follow-up	887	17.84 $\pm$ 0.51	43.7 $\pm$ 7.7	907	17.97 $\pm$ 0.53	29.5 $\pm$ 5.1

Table 2

Model-fitting Results

Model Comparison	Hypothesis	Log-likelihood	df	Overall Fit		Fit of Nested Model	
				χ^2	p	χ^2	p
1	None: Saturated model	-17584.4	0				
2 vs. 1	Latent growth model conforms to the data ^a	-17590.4	8	12.0	.15		
3 vs. 2	Linear growth in males, i.e., middle time point = 0.5	-17598.1	12	27.4	.01	15.4	< .01
4 vs 2	Linear growth in females, i.e., middle time point = 0.5	-17630.3	12	91.8	< .01	79.8	< .01
5 vs. 2	Nonlinear growth, i.e., fixed time point at .53 in males and .63 in females	-17593.3	16	17.9	.33	5.8	.67
6 vs 5	Equal parameters across co-twins and zygosity groups	-17627.3	68	85.9	.07	68.0	.07
7 vs 6	No intercept-slope correlation	-17628.9	74	89.0	.11	3.2	.78
8 vs 7	Intercept mean is equal between males and females	-17644.8	75	120.9	< .01	31.8	< .01
9 vs 7	Intercept variance is equal between males and females	-17645.8	75	122.8	< .01	33.8	< .01

Notes. Overall fit is in reference to the saturated model. Likelihood ratio test is based on twice the difference in log-likelihood values between nested models, which is distributed as chi-square; df = degrees of freedom.

^aMiddle time point was freely estimated in all groups.

Table 3

Maximum-likelihood Estimates of Means, Variances, and Residual Variances

Parameter	Males		Females	
Growth Factors				
	Mean	Variance	Mean	Variance
Intercept	21.98 ± 0.15	15.09 ± 0.78	20.84 ± 0.14	9.50 ± 0.64
Slope	21.87 ± 0.24	21.17 ± 2.64	8.75 ± 0.17	6.84 ± 1.05
Residual Variances				
	Estimate	Proportion (1 - R ²)	Estimate	Proportion (1 - R ²)
Intake	2.36 ± 0.36	.13	5.01 ± 0.50	.34
FU1	23.84 ± 1.38	.53	7.48 ± 0.53	.38
FU2	18.12 ± 2.53	.33	8.07 ± 0.86	.33

Note. Values placed after the plus-minus signs are standard errors.

Table 4

Intraclass Twin Correlations

	MZ Males	DZ Males	MZ Females	DZ Females
Growth Factors				
Intercept	.88 (.84, .93)	.40 (.30, .50)	.84 (.79, .90)	.45 (.30, .59)
Slope	.80 (.65, .94)	.40 (.11, .69)	.58 (.36, .80)	.44 (.09, .79)
Residual Variances				
Intake	0	0	.56 (.42, .69)	.44 (.22, .65)
FU1	.62 (.54, .70)	.35 (.21, .49)	.32 (.18, .45)	.24 (.05, .43)
FU2	.49 (.28, .69)	.13 (0, .51)	.53 (.37, .70)	.39 (.13, .66)

Note. 95% confidence intervals are enclosed in parentheses.

Table 5
Standardized Estimates of Genetic and Environmental Variance Components

Parameter	Males			Females		
	A	C	E	A	C	E
Growth Factors						
Intercept	.88 ± .02	0	.12 ± .02	.79 ± .15	.05 ± .15	.16 ± .03
Slope	.80 ± .07	0	.20 ± .07	.28 ± .41	.30 ± .37	.42 ± .11
Residual Variances						
Intake	0	0	1	.24 ± .25	.32 ± .22	.45 ± .07
FU1	.53 ± .16	.09 ± .14	.39 ± .04	.15 ± .22	.17 ± .20	.69 ± .07
FU2	.49 ± .10		.51 ± .10	.29 ± .31	.25 ± .28	.46 ± .09

Notes. A = Additive genetic effects; C = shared environmental effects; E = nonshared environmental effects.

Table 6

Descriptive statistics of physical stature and body mass, and variance accounted for in age-corrected grip strength

Measurement Occasion	Males			Females		
	Stature (cm) <i>M</i> $\pm$ <i>SD</i>	Mass (kg) <i>M</i> $\pm$ <i>SD</i>	<i>R</i> ²	Stature (cm) <i>M</i> $\pm$ <i>SD</i>	Mass (kg) <i>M</i> $\pm$ <i>SD</i>	<i>R</i> ²
Intake	150.1 $\pm$ 7.6	42.9 $\pm$ 10.7	.16	151.5 $\pm$ 7.5	45.2 $\pm$ 11.7	.30
FU1	171.0 $\pm$ 8.1	63.4 $\pm$ 14.2	.18	163.6 $\pm$ 6.2	59.8 $\pm$ 13.1	.19
FU2	178.2 $\pm$ 6.8	75.2 $\pm$ 15.0	.10	165.5 $\pm$ 6.5	65.1 $\pm$ 14.6	.21

Note. *R*² is a multiple regression statistic, i.e., the squared multiple correlation of stature and mass with grip strength.