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Variation in CAG repeat length of the androgen receptor gene predicts variables associated with intrasexual competitiveness in human males

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ABSTRACT

An expanding body of research suggests that circulating androgens regulate the allocation of energy between mating and survival effort in human males, with higher androgen levels promoting greater investment in mating effort. Because variations in the number of CAG codon repeats in the human androgen receptor (AR) gene appear to modulate the phenotypic effects of androgens – with shorter repeat lengths associated with greater androgenic effects per unit androgen – polymorphisms in this gene may predict trait-like individual differences in the degree to which men are calibrated toward greater mating effort. Consistent with this, men in the present study with shorter CAG repeat lengths exhibited greater upper body strength and scored higher on self-report measures of dominance and prestige, all of which are argued to be indices of mating effort. Repeat length failed to predict sociosexual orientation (i.e. pursuit of short-term mating relationships), however, suggesting that the traits correlated with this polymorphism may be primarily associated with intrasexual competitiveness in the service of long-term mating effort. None of these measures of mating effort was related to baseline testosterone concentrations (either as main effects or as interactions with CAG repeat length), implying that long-term androgen exposure associated with AR gene polymorphisms may account for more variance in some androgen-dependent traits than does current testosterone concentration. These findings provide further evidence for the importance of the CAG repeat polymorphism in the AR gene in explaining a broad range of individual differences in human males.

Introduction

Polymorphisms in the human androgen receptor (AR) gene may play an important role in the explanation of individual differences in human morphology, cognition, and behavior. The number of CAG codon repeats in the first exon of this gene typically ranges from 9 to 31 in non-pathological populations, and tends to be normally distributed (e.g., Alevizaki et al., 2003; Edwards et al., 1992). Smaller numbers of CAG repeats are associated with both greater AR protein expression (Choong et al., 1996) and enhanced transcriptional activity of the AR (Chamberlain et al., 1994), suggesting that the same doses of androgen should be translated into larger phenotypic effects in men with fewer repeats. Consistent with this, physiological responses to testosterone treatment were negatively associated with CAG repeat length in a sample of hypogonadal men (Zitzmann and Nieschlag, 2007). This stronger mapping of androgen into phenotypic effects may in turn explain why shorter repeat lengths have been associated with androgen-dependent outcomes such as increased risk of prostate cancer (Casella et al., 2001) and enhanced spermatogenesis (von Eckardstein et al., 2001).

Because the AR is a ligand-activated transcription factor – i.e. it regulates the expression of various genes when bound to androgens – the AR gene could act as a dial that calibrates the expression of diverse phenotypic traits in response to androgens. AR is expressed throughout the brain and body (Bhasin et al., 2001; Simerly et al., 1990), where it regulates different genes in different cell types, such that changing the number of CAG repeats in the AR gene alone could potentially dial up or down the degree of androgenicity across the entire organism. Such whole organism effects in principle allow coordination of morphological traits, such as body size and strength, with brain mechanisms that regulate behavioral patterns in which these features are instrumental, like degree of competitiveness, status-seeking, and interpersonal dominance. As such, AR polymorphisms may explain variability within a functionally coordinated suite of traits. Identification of the specific traits in question can in turn be derived from theories of the functional roles of androgens.

A number of theorists have advanced the idea that androgens are designed to adaptively regulate energy allocation between mating effort (not just the act of pursuing or competing for mates directly, but also securing status/resources to facilitate mate acquisition) and survival effort (Bribiescas, 2001; Ellison, 2001; McIntyre et al., 2006;

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Muehlenbein and Bribiescas, 2005). In humans, androgens promote anabolic processes (e.g., the accretion and maintenance of muscle mass; Bhasin et al., 2001) and have been linked to status-seeking and dominance-related behaviors (see Archer, 2006; Josephs et al., 2006; Mazur and Booth, 1998), all of which likely facilitate intrasexual competition. Conversely, androgens appear to inhibit survival-related outcomes, such as the capacity to mobilize immune responses and sequester energy reserves in adipose tissue (for review, see Muehlenbein and Bribiescas, 2005). Furthermore, these tradeoffs can be dynamically regulated through the adjustment of circulating androgens: men's testosterone is elevated when mating effort can produce greater benefits (e.g., when single vs. partnered, Gray et al., 2002, 2004; when in the presence of potential mates, Roney et al., 2007, 2010), but declines under conditions of food shortage (Bribiescas, 2001; Trumble et al., 2010) and immune challenge (Muehlenbein et al., 2005; Muehlenbein et al., 2010; Simmons and Roney, 2009).

Given that CAG repeat number in the AR gene appears to modulate the phenotypic effects of androgens, the same distinction between mating vs. survival effort critical in crafting a functional analysis of the effects of testosterone should likewise predict the specific traits associated with polymorphisms in the AR gene. Essentially, we propose that superimposed on the state-like changes in androgens associated with life-history problems will be trait-like individual differences in the degree to which androgens are mapped into increased mating and decreased survival effort. The current research focused specifically on mating effort, and tested the broad hypothesis that men with shorter CAG repeats will on average score higher on morphological and psychological measures of such effort than will men with larger numbers of repeats.

While no studies examining the role of AR polymorphisms have been explicitly framed in terms of the tradeoff between mating and survival effort, the extant evidence is broadly consistent with the idea that shorter CAG repeats (compared to longer) predict outcomes associated with greater investment in mating effort. For men in their twenties, CAG repeat number is negatively correlated with muscle size and lean body mass (Nielsen et al., 2010) and positively correlated with measures of body fat (Nielsen et al., 2010; Zitzmann et al., 2003), though evidence suggests that these relationships may not hold among older men (Walsh et al., 2005) or men in nutritionally stressed populations (Campbell et al., 2007). Although studies testing associations between AR gene CAG repeats and personality traits have produced mixed findings (see Jonsson et al., 2001; Loehlin et al., 2005; Turakulov et al., 2004; Westberg et al., 2009), when significant effects have been found, they have been in the direction of smaller numbers of repeats predicting higher dominance-related traits, such as extraversion, assertiveness, and verbal aggression (e.g., Jonsson et al., 2001; Lukaszewski and Roney, 2011). Men with shorter CAG repeats may be more common among criminal populations (Cheng et al., 2006; Rajender et al., 2008), especially violent criminals (Rajender et al., 2008), suggesting that such men may be calibrated to engage in particularly intense intrasexual competition. Finally, physiological correlates of shorter CAG repeat lengths in men include both higher rates of sperm production (von Eckardstein et al., 2001) and larger testosterone responses to social interactions with women (Roney et al., 2010). The latter finding in particular links AR gene polymorphisms to human mating psychology, and in conjunction with the morphological correlates of CAG repeats, supports the possibility of coordinated adjustment of diverse traits associated with mating effort.

The present study provides a focused test of whether AR gene polymorphisms have coordinated effects on both physical and behavioral/psychological variables. As intrasexual competition has likely played a critical role in the dynamics of human mating (both in direct competition for women and facilitating their mate choice), men's upper body strength was examined as a measure of physical investment in mating effort. Shorter CAG repeats have been found to predict greater muscle mass in young men (Nielsen et al., 2010), but direct measures of physical strength have not been reported. Although muscle mass likely predicts physical strength, androgen-dependent variables such as rate of glucose uptake in muscle tissue (see Tsai and Sapolsky, 1996) or levels of hemoglobin (Zitzmann and Nieschlag, 2007) may alter strength per unit muscle mass. As such, actual strength (as opposed to muscle mass alone) may provide a more complete measure of physical investment in mating effort. It has been argued that upper body strength is the single most important predictor of success in men's physical conflicts (Sell et al., 2009). Winners of such contests enjoy both greater status and reproductive success among hunter-horticulturalists (von Rueden et al., 2011), supporting the idea that strength is directly relevant for mate competition under the approximate social conditions thought to characterize most of human evolution.

The degree to which men pursue social status was examined as a behavioral/psychological indicator of mating effort, as social status (and the associated command of resources) is argued to be an important factor in female mate choice (Buss, 1989). Evidence from hunter-horticultural societies supports a positive relationship between peer-nominated status and measures of mating success (Chagnon, 1988; Darwin, 1871; Irons, 1979). As status is an intrinsically scarce resource, however, men who pursued it have likely incurred significant risks due to intrasexual competition, and thus one expects on functional grounds the existence of mechanisms that calibrate status-seeking to cues of its current risk–reward ratio. Androgens appear to play a signaling role in such mechanisms insofar as they have been positively associated with decisions to compete with other individuals (Carre and McCormick, 2008; Mehta and Josephs, 2006); as such, shorter CAG repeats in the AR gene should (all else equal) similarly bias men toward greater willingness to compete for status. Measurement of status-seeking is somewhat complicated by the idea that status can be achieved through two theoretically distinct pathways: dominance, in which the application (or threat) of force is used to influence the division of resources by others, and prestige, in which resources are freely allocated to individuals based on their ability to confer benefits (Henrich and Gil-White, 2001; Johnson et al., 2007). A self-report measure with both dominance and prestige subscales (see Methods) was therefore used to measure status-seeking.

Finally, the present study assessed relationships between AR polymorphisms and self-reported measures of mating strategy. Various lines of evidence suggest that humans have had at least mildly polygynous mating systems throughout most of their history (e.g., Marlowe, 2003), in which case the pursuit of multiple partners may have been a mechanism whereby men's social status could be translated into greater reproductive success. If so, investment in physical strength, the pursuit of social status, and the desire for and pursuit of multiple sexual partners may all have been coordinated components of greater investment in mating effort. The sociosexual orientation inventory (SOI) is a validated measure of willingness to engage in sex without closeness or commitment (Simpson and Gangestad, 1991), and its items in part tap desire for sex with multiple partners. Some evidence supports positive associations between men's testosterone and their SOI scores (at least for men in relationships; see McIntyre et al., 2006), suggesting that shorter CAG repeats in the AR gene should likewise predict higher scores on this scale.

In summary, smaller numbers of CAG repeats in the AR gene were hypothesized to predict greater upper body strength, higher self-reported dominance and prestige, and higher SOI scores. Because these dependent variables could have causal relationships with each other (for instance, if greater physical strength caused greater dominance or higher prestige), exploratory analyses also tested whether individual outcome variables mediated relationships between repeat length and the other dependent measures.

Methods

Subjects

One hundred forty-nine undergraduate men participated in the study for partial fulfillment of course requirements, but failures of DNA extraction reduced the final sample size to $n = 138$ (mean age = 18.92, SD = 1.35). Sixty-four percent of the men reported being Caucasian, 21% Latino, 14% Asian, and 1% African American. All men self-identified as heterosexual.

Materials

Upper body strength was assessed using a Jamar Hydraulic Dynamometer (model 5030J1; see *Procedures*). Body weight was determined using a Tania electrical impedance scale (Tanita BC-573), and height was self-reported via questionnaire. These values for height and weight were used to calculate BMI.

As part of a larger battery of questionnaires, subjects completed the Self-Perceived Social Status Scale (SSSS; Buttermore and Kirkpatrick, in press), which includes subscales for dominance ($\alpha = 0.84$; example items: 'I demand respect from members of my peer group,' 'I am willing to use aggressive tactics') and prestige ($\alpha = 0.90$; example items: 'I have gained distinction and social prestige,' 'Others recognize me for my contributions to my social group'). The validity of this scale has been established both in relation to other scales argued to tap components of dominance and prestige (Buttermore and Kirkpatrick, in press) and with respect to willingness to deploy forceful or aggressive tactics (which correlates with the subscale of dominance, but not prestige; Johnson et al., 2007). Scores on each of these subscales were z-scored prior to analysis in the present study.

Subjects also completed the sociosexual orientation inventory (SOI), which was scored according to the algorithm presented in Simpson and Gangestad (1991). Because the SOI contains some items that inquire about an individual's history of sexual behavior, total scores on the scale are constrained by sexual opportunities, and thus may not provide a pure index of preferred mating strategy. Three items on the scale are more purely attitudinal in nature (e.g., 'I can imagine myself being comfortable and enjoying "causal" sex with different partners'), and a composite of these items was computed as an alternate index of interest in unrestricted mating.

Procedures

Participants were taking part in a larger study testing hormonal responses of men to brief social interactions with either young women or young men (see Roney et al., 2010). These interactions took place in an initial testing session; subjects returned for a second session approximately one week later to complete the procedures described below. None of the variables analyzed in the present report differed across the experimental conditions implemented in the first testing session. All data were collected in accordance with procedures approved by the UCSB institutional review board and in compliance with the Code of Ethical Principles for Medical Research Involving Human Subjects of the World Medical Association.

Subjects first provided a saliva sample via passive drool into polypropylene collection vials. They next completed a battery of questionnaires, which included the SSSS and the SOI. Measures of both grip and chest strength were collected using the dynamometer; grip strength was assessed with subjects holding the dynamometer at their sides in one hand and squeezing, chest strength by holding the dynamometer in front of their chest with two hands and pressing inward. These two measures were z-scored and then averaged to produce a composite measure of strength ($\alpha = 0.75$). Weight was recorded, then subjects swished with alcohol-based mouthwash before expectorating into collection vials. These samples contained buccal cells from which DNA could be extracted in order to assay the number of CAG repeats in the AR gene (see *Genotyping and hormone assays*). All of the second testing sessions took place between 1 PM and 5 PM.

Genotyping and hormone assays

Mouthwash samples were stored at −80 °C until being delivered to the Biological Samples Processing Core at UCLA. DNA extraction was carried out on an Autopure LS (Qiagen Inc.) using the Puregene Buccal Cell Kit (Qiagen Inc.). Genotyping and sequencing were subsequently done by the UCLA Sequencing and Genotyping Core. The primers used in PCR amplification of the DNA were: 5′-TCCAGAATCTGTTCCAGAGCGTGG-3′ (forward), and 5′-GCTCTGAAGGTTGCTGTTCCTCAT-3′ (reverse). PCR conditions were identical to those in Hernandez et al. (2008) with the following exceptions: reaction volume was reduced to 12.5 ml (using 50 ng of genomic DNA) and fragments were separated using an ABI 3730 Genetic Analyzer (Applied Biosystems). A subset of samples of varying length ($n = 6$) was fully sequenced to confirm repeat numbers using the Big Dye Terminator Sequencing Kit (Applied Biosystems). The sequencing reaction consisted of 30 cycles at 96 °C for 10 s, 50 °C for 5 s, 60 °C for 4 min and used the reverse primer.

Consistent with previously reported values, CAG repeat length ranged from 14 to 31, with an average of 21.74. Repeat length differed by ethnicity, $F(3, 134) = 2.92$, $p = 0.04$, though this effect was driven entirely by differences between the African American subjects ($n = 2$, mean = 17.5 repeats) and the other groups. Exclusion of these two participants did not alter any statistical conclusions in the paper, and all analyses were robust to controlling for ethnicity, except where otherwise indicated.

Both the saliva sample collected at the start of session 2 and another sample collected at the beginning of session 1 were assayed for testosterone. Because the two samples were collected at a similar time of day (and prior to any manipulation), they were averaged to produce a baseline estimate of circulating testosterone. This estimate was used to explore the possibility of a main effect of testosterone on the dependent variables, or an interaction between testosterone and CAG repeat number. Assays were carried out at the Biomarkers Core Laboratory at the Yerkes National Primate Research Center, Atlanta, GA, USA. The intra-assay and inter-assay CVs were 6.91% and 15.60%, respectively. Further details on the assay procedure can be found elsewhere (Roney et al., 2007, 2010).

Statistical analyses

Following convention in the CAG repeat literature (e.g., Campbell et al., 2007; Jonsson et al., 2001; Turakulov et al., 2004; Westberg et al., 2009), we divided repeat lengths into either short ($\leq$21 repeats) or long (>21 repeats) on the basis of a median split. Differences in dependent measures between the groups were examined with independent samples *t*-tests (treating CAG repeat lengths as a continuous variable in regression analyses did not change any statistical conclusions). Analysis of covariance (ANCOVA) was used to evaluate whether differences between men with short and long repeat lengths were robust to controlling for a number of other variables. Correlations between outcome variables were computed using Pearson correlation coefficients. Moderated regression analyses were carried out to examine possible interactions between CAG repeat length and baseline levels of testosterone in predicting outcome variables. Finally, bootstrapping procedures (see Preacher and Hayes, 2004; Shrout and Bolger, 2002) were employed in order to test whether effects of CAG repeats on the dependent variables may have been mediated by any of the other variables. This process estimates the indirect effect of CAG repeat number on a dependent variable — that is, the effect of CAG repeat number acting through some mediator (and only through the mediator) on the dependent

variable. In such cases, mediation is understood to have occurred when the indirect effect is non-zero (as assessed by its confidence interval). This method is more robust than the more common mediational models often employed (Preacher and Hayes, 2004), as it makes no assumptions about the distribution of any of the pathways involved. All reported significance levels are two-tailed.

Results

As predicted, men with smaller numbers of CAG repeats in the AR gene exhibited greater upper body strength, higher self-reported prestige, and higher self-reported dominance than did men with larger numbers of repeats, as shown in Table 1. Contrary to our hypothesis, however, there were no differences in total SOI scores between the long and short repeat groups. The same general pattern was obtained when using more stringent Bonferroni corrected alpha levels (accounting for the four a priori hypotheses), save that the relationship between CAG repeat length and dominance was only marginally significant. When analyses were restricted to the attitudinal items of the SOI, there was a marginally significant trend toward higher scores among men with shorter repeat lengths (see Table 1), although this trend did not survive controls for ethnicity in an ANCOVA ($p = 0.12$). In summary, these results support effects of CAG repeat length on strength and status-seeking, but not on self-reported mating strategy.

Table 1 also includes exploratory analyses demonstrating the absence of associations between CAG repeat lengths and men's height, weight, and BMI. These results suggest that the effect of repeat length on strength was not secondary to an association with overall body size. Consistent with this, the difference in strength between men with high and low repeats was still significant after controlling for each of these morphological variables in separate ANCOVAs ($ps < 0.01$).

To evaluate whether circulating androgens were associated with our dependent measures, a series of moderated regression analyses was conducted for each of the primary dependent variables listed in Table 1. In these models, CAG repeat length and baseline testosterone concentration were entered in step one, and a CAG length × testosterone interaction term was entered in step two. There were no main effects of testosterone in step 1 of the models (all $ps > 0.42$), nor were any of the testosterone × CAG interaction terms significant in step 2 (all $ps > 0.24$). As in other studies of young men (Nielsen et al., 2010; Walsh et al., 2005; Zitzmann et al., 2003; Zitzmann and Nieschlag, 2007), we found no association between baseline testosterone and number of CAG repeats (see Table 1).

Table 2 presents correlations between the primary dependent measures in the study. The significant correlations of strength with both prestige and dominance (which were uncorrelated with one another) raise the possibility that the relationship between repeat number and status-seeking might be mediated by physical strength.

Table 1
Differences in phenotypic outcomes associated with short vs. long CAG repeat length (variables in italics predicted to be higher in men with shorter CAG repeat lengths).

	CAG repeat length			
	≤21 repeats	>21 repeats	*p*	Effect size (d)
Strength[a]	0.214	−0.238	0.002	0.679
Prestige[a]	0.250	−0.172	0.008	0.504
Dominance[a]	0.151	−0.191	0.039	0.373
SOI[a]	0.019	−0.071	0.365	0.293
SOI (attitudinal)[a]	0.119	−0.188	0.063	0.335
Testosterone (pg/ml)	102.07	96.95	0.569	0.002
BMI	23.55	23.30	0.603	0.026
Height (in.)	70.15	70.25	0.839	0.012
Weight (lb)	164.40	163.00	0.754	0.002

[a] Variables transformed to Z-scores.

Table 2
Zero-order correlations between measures of mating effort and testosterone.

	Strength	Prestige	Dominance	SOI	SOI — attitudinal
Prestige	0.198*	–			
Dominance	0.191*	0.071	–		
SOI	0.172	0.135	0.076	–	
SOI — attitudinal	0.111	−0.047	0.081	0.756**	–
Testosterone	−0.011	0.136	0.046	0.083	−0.007

* $p < 0.05$.
** $p < 0.01$.

In such a scenario, AR polymorphisms lead to differences in strength, which in turn produce variation in status-seeking, rather than there being a direct link between AR variation and status-seeking. To test for the mediational role of strength, separate regression models were constructed using dominance and prestige scores as outcome variables, with CAG repeat number entered as the predictor variable and strength as the mediator. Bootstrapping procedures (see Methods) were used to evaluate the statistical significance of each model. The relationship between CAG repeat length and prestige ($\beta = -0.22$, $p = 0.007$) was not affected by the introduction of strength as a mediator ($\beta = -0.21$, $p = 0.014$); estimates of the indirect effect (mean = −0.042, 95% CI [−0.13–0.02]) confirmed that the influence of CAG on prestige through strength was not different from zero. The relationship between CAG repeat length and dominance ($\beta = -0.18$, $p = 0.03$) was mediated by strength ($\beta = -0.13$, $p = 0.117$ for CAG when strength was entered into the model), however, with the magnitude of the indirect effect greater than zero (mean = −0.087, 95% CI [−0.20 to−0.011]). The effects of AR polymorphisms on prestige-related behaviors thus seem to be largely independent of their effects on physical strength, while effects on dominance-related behaviors seem to be produced as a result of (or in concert with) effects on strength.

Discussion

The present results provide additional evidence that the CAG repeat polymorphism in the AR gene is associated with the calibration of investment in mating effort. Men with shorter CAG repeat lengths had greater upper body strength, as well as higher self-reported dominance and prestige. Although the association between repeat length and dominance was mediated by physical strength, the relationship between repeat length and prestige was not, suggesting that AR polymorphisms may have distinct effects on physical and psychological outcomes. Consistent with previous work establishing that men with shorter CAG repeats have greater muscle mass (Nielsen et al., 2010), higher levels of extraversion (Lukaszewski and Roney, 2011; Westberg et al., 2009), higher rates of sperm production (von Eckardstein et al., 2001), larger testosterone responses to potential mates (Roney et al., 2010), and higher rates of violent behavior (Rajender et al., 2008), these findings bolster the argument that polymorphisms in this single gene can dial up or down the androgenic profile of the entire organism, across both physical and psychological dimensions. Given that the traits associated with AR polymorphisms are generally implicated in intrasexual competition, furthermore, it appears that the distinction between mating and survival effort that has been used to explain the functional role of androgens (Bribiescas, 2001; Ellison, 2001; Muehlenbein and Bribiescas, 2005) may likewise provide a parsimonious framework for explaining the phenotypic correlates of variations in the AR gene.

The predicted association between CAG repeat number and SOI scores was not observed. This may be because while the SOI scale measures orientation toward short-term sexual partnerships, most of the variance in male reproductive success across human evolution may have been related to the number and quality of long-term mates

rather than the number of short-term partners. Data demonstrate that women in natural fertility populations experience fertile cycles quite rarely (Strassmann, 1997), and thus conceptive, short-term sexual opportunities would have been very limited in the small group environments thought to characterize most of human evolution. As such, higher mating effort may have been associated primarily with the pursuit of fertile long-term partners, which is a mating strategy not directly captured by the SOI.

It is interesting in this regard that dominance, prestige, and physical strength were also uncorrelated with SOI scores in the present sample (see Table 2), consistent with recent arguments suggesting that androgen-dependent, masculinized traits are more strongly associated with intrasexual competitiveness than with sexual attractiveness in human males (Puts, 2010). Rather than calibrating short-term mating effort (which is likely more strongly associated with sexual attractiveness; see Gangestad and Simpson, 2000), then, AR gene polymorphisms may primarily regulate investment in intrasexual competitiveness in the service of long-term mating effort. High levels of mating effort directed toward increasing the number and quality of long-term partners may still have involved important tradeoffs throughout most of human history, however, such as reduced survival effort or lower rates of paternal investment per individual offspring (see Marlowe, 2000). Future research could test whether AR gene polymorphisms do in fact affect mating strategy via tradeoffs in paternal quality, with shorter CAG repeats potentially predicting reduced paternal investment in favor of the pursuit of multiple long-term partners (via either concurrent polygyny or serial monogamy).

Unlike CAG repeat numbers, circulating testosterone concentrations were not correlated with any of the dependent measures. Given that shorter repeats cause greater androgen-dependent phenotypic outcomes for any given rate of androgen production (e.g., Zitzmann and Nieschlag, 2007), the CAG repeat polymorphism is likely to predict individual differences in long-term androgen exposure. As such, correlations with AR polymorphisms – but not current testosterone – implies that chronic degree of androgen exposure may predict status-seeking and physical strength more strongly than does momentary testosterone production in young men.

One reason that specific outcomes may correlate with long-term androgen exposure but not moment-to-moment testosterone concentrations is that circulating testosterone may be down-regulated when it is not necessary for courtship or competition. In olive baboons, for instance, males' testosterone concentrations and dominance rank are uncorrelated under stable social conditions, but are positively correlated during periods of social instability when dominance is in flux (Sapolsky, 1983). If similar effects occur in humans, then current testosterone may not always predict variables such as dominance (see Johnson et al., 2007), despite the fact that dominance may be predicted by long-term androgen exposure. Outcomes such as prestige and strength (and dominance, if it is influenced by strength) may be especially sensitive to long-term androgen levels insofar as they require consistent, prolonged investment (time and energy to acquire valued skills or knowledge, and somatic investment in morphology, respectively) for their successful development. These considerations may help explain why there have been mixed findings in the human literature for associations between circulating testosterone and variables like aggression (for a review, see Archer, 2006); if mechanisms regulating such outcomes are more sensitive to long-term androgen exposure, AR gene polymorphisms might have more explanatory power than do current testosterone concentrations.

No definitive account has been offered for the evolutionary origin and maintenance of the CAG repeat polymorphism in the human AR gene. Both repeat length and variability tend to increase with phylogenetic proximity to humans (Choong et al., 1998; Hong et al., 2006). Within primates, a large jump in repeat length and variance occurs when comparing apes such as orangutans (monomorphic at 12 repeats) and gorillas (7–9 repeats) to chimpanzees (14–26 repeats, with a mean of 19; Hong et al., 2006). Whatever the cause of the original CAG repeat expansion, then – whether selection for reduced androgen-dependence of male reproductive behavior or neutral processes such as genetic drift or differential species-typical mutation rates (Rubinsztein et al., 1995) – the expansion pre-dated humans, who appear to have inherited an expanded repeat range from nonhuman primate ancestors.

The nonhuman origin of the expanded human CAG repeat length does not preclude the possibility that between- and within-population variance in humans has been maintained by selection. The results of the present study show that repeat length is associated with fitness-relevant traits, and the association with mating effort in particular suggests the hypothesis that selection may act on repeat length depending on the recurrent, relative importance of mating vs. survival effort within specific populations. In ecologically harsh or unpredictable environments, for instance, a less androgenized phenotype may be ideal in order to increase relative investment in survival effort, which could select for longer CAG repeats as a means of dialing down the anabolic effects of circulating androgens (a reduction of androgen production could in principle accomplish the same result, but might also disrupt the calibration of non-AR mediated pathways whereby androgens influence rapid behavioral responses via enzymatic conversion of testosterone into neurotransmitters or other hormones; see Aikey et al., 2002; Bitran et al., 1993; Juntti et al., 2010). Perhaps consistent with this, Ariaal pastoralists, a population characterized by chronic undernutrition, have longer CAG repeats, on average, than other African populations that do not share the same history of resource scarcity (Campbell et al., 2007). A more rigorous test of this hypothesis, though, would require broader assessments of whether known between-group variation in CAG repeat length (e.g., Kittles et al., 2001) can be mapped onto estimates of the history of environmental harshness within the respective populations. With respect to the maintenance of within-group variation in CAG repeat length, on the other hand, frequency-dependent selection (in which the fitnesses of particular phenotypes depend on how rare they are in the population) may explain variance beyond that attributable to chance processes like genetic drift, since having shorter repeats than other group members may have provided competitive advantages that were balanced against the potential survival-related costs of greater androgenicity.

In conclusion, the present study provides further evidence for the importance of the AR gene CAG repeat polymorphism for the explanation of individual differences in men. This gene is particularly intriguing because it encodes for a transcription factor, which means that changes in its sequence can lead to coordinated adjustments in the expression of many other genes throughout the brain and body. The specific phenotypes associated with polymorphisms in this gene – including the status-seeking and strength variables investigated here – are unified by their relevance in making tradeoffs between mating and survival effort, with smaller numbers of CAG repeats predicting higher investment in mating effort. Future research could investigate associations between AR gene polymorphisms and measures of survival effort (e.g., pathogen load, magnitude of cellular response to immune challenge) to further elucidate the role of the AR gene in making these types of life-history tradeoffs. Extension of such a research program to non-university samples in which male intrasexual competition is more likely to involve physical aggression might also provide more direct evidence of the importance of CAG repeats in the use (and success) of such tactics.

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