



Integrating molecular genetics and evolutionary psychology: Sexual jealousy and the androgen receptor (AR) gene



David M.G. Lewis^{a,*}, Laith Al-Shawaf^{b,c}, Mareike C. Janiak^d, Sarah P. Akunebu^e

^a Murdoch University, Australia

^b Bilkent University, Turkey

^c Institute for Advanced Study, Berlin, Germany

^d Rutgers University, United States

^e St. Edward's University, United States

ARTICLE INFO

Article history:

Received 22 January 2016

Received in revised form 18 October 2016

Accepted 9 November 2016

Available online 29 November 2016

Keywords:

Sexual jealousy

Androgen receptor (AR) gene

Heritability

Individual differences

Evolutionary psychology

Molecular genetics

Jealousy

Mating

ABSTRACT

Integrating evolutionary psychological and molecular genetic research may increase our knowledge of the psychological correlates of specific genes, as well as enhance evolutionary psychology's ability to explain individual differences. We tested the hypothesis that men's sexual jealousy mechanisms functionally calibrate their psychological output according to genetic variation at the androgen receptor locus. Mated men ($N = 103$) provided buccal cell samples for genotype fragment analysis and completed inventories assessing their sexually jealous cognitions and emotions. Results indicated that men with longer sequences of CAG codon repeats at the androgen receptor locus were more likely to perceive ambiguous social and environmental cues as indicative of their mates' infidelity, and experienced greater emotional upset in response to these cues. These results contribute to a growing body of research linking polymorphism at the AR locus to individual differences in psychology, and, to our knowledge, provide the first evidence pointing toward the heritability of sexual jealousy. Our discussion centers on whether the heritability of psychological differences implies direct genetic influences on the neurobiological substrate, or reflects functionally calibrated output from sex-typical and species-typical mechanisms. We conclude by describing how future research can more clearly differentiate between these alternative genetic models.

© 2016 Elsevier Ltd. All rights reserved.

Evolutionary psychologists assert that identifying the genetic basis of a psychological phenomenon is not necessary to establish that the phenomenon under investigation is the output of an evolved psychological mechanism (see Confer et al., 2010; Lewis, Al-Shawaf, Conroy-Beam, Asao, & Buss, in press; Williams, 1966). Indeed, the location of a particular allele in the molecular genetic substrate is not relevant to establishing adaptation. Rather, to show that a psychological phenomenon is the output of an evolved adaptation, one must demonstrate specialized functional design (Cosmides & Tooby, 1997; Lewis et al., in press; Williams, 1966).

This does not imply that molecular genetics cannot valuably inform evolutionary psychology. If there are compelling theoretical reasons to believe that evolved psychological mechanisms are designed to be sensitive to the downstream products of specific genetic loci, then integrating evolutionary psychological and molecular genetic research could enhance both 1) our understanding of the psychological correlates of those genes and 2) evolutionary psychology's explanatory power at

the level of individual differences in addition to sex-typical and species-typical psychological phenomena.

This paper aims to theoretically illustrate and empirically demonstrate the mutually informative potential of molecular genetics and evolutionary psychology. Specifically, the current study applied an evolutionary psychological framework to investigate individual differences in men's sexual jealousy as a function of genetic variation at the human androgen receptor locus.

1. Sexual jealousy

Ancestral men whose long-term mates were sexually unfaithful would have incurred substantial reproductive fitness costs. These include the staggering costs associated with being cuckolded and unwittingly investing in the offspring of another male, as well as the social costs of reputational damage (Buss, 2000). Selection would therefore have strongly favored the evolution of anti-infidelity adaptations in men.

Several theorists have proposed that sexual jealousy represents a coordinated suite of psychological processes designed to prevent mate defection and infidelity. Consistent with this proposal, the cognitive and

* Corresponding author at: Murdoch University, School of Psychology and Exercise Science, 90 South Street, Murdoch, Western Australia 6150, Australia.
E-mail address: d.lewis@murdoch.edu.au (D.M.G. Lewis).

affective facets of sexual jealousy exhibit evidence of design to promote one's mate's fidelity (Buss, 2002). This includes triggering information-gathering about infidelity threat (Goetz, Shackelford, Romero, Kaighobadi, & Miner, 2008; Schützwohl, 2008) and producing negative emotions in response to one's mate's social interactions – in particular with potential mate poachers (Buss, 2000). Moreover, these affective states can motivate controlling behaviors or aggressive responses (Daly, Wilson, & Weghorst, 1982) to fend off these same-sex rivals and combat other threats to the relationship (Buss, 2000; Buss, Larsen, Westen, & Semmelroth, 1992; Daly et al., 1982; Symons, 1979).

2. Evolved individual differences

A condition-dependent individual differences model (e.g., Lewis, 2015; Lukaszewski & Roney, 2011; Tooby & Cosmides, 1990; Wolf, van Doorn, Leimar, & Weissing, 2007) posits that species-typical evolved psychological mechanisms are designed to process, as input, cues ancestrally predictive of the cost-benefit tradeoffs of alternative interpersonal strategies, and produce, as output, the psychological strategy of greater probabilistic net benefit for the individual, given his or her condition (Buss & Greiling, 1999; Nettle, 2006; Wolf et al., 2007).

The output of humans' psychological mechanisms is expected to be largely the same when 1) all individuals face the same adaptive problems (Buss, 1995), 2) these adaptive problems pose similar costs to all individuals, and 3) all individuals face these adaptive problems to the same degree. However, when individuals differ in *any* of these dimensions – such as when men differentially face the risk of their mates' infidelity – we should expect the output of their shared, evolved psychological mechanisms to diverge in systematic, functional ways.

3. Individual differences in infidelity threat

An exploration of evolved female mating strategies reveals why ancestral men would have faced differential likelihoods of being cuckolded. Women's reproductive success would have been enhanced when they were able to produce genetically robust offspring and secure long-term investment from their mates. To produce offspring of high genetic quality, a woman had to copulate with a man of high genetic quality. Yet, because desirable, high genetic quality men could have increased their own reproductive success by engaging in uncommitted mating with multiple women, selection would have favored the pursuit of short-term mating strategies among these men (Gangestad & Simpson, 2000). Indeed, physically attractive men of high genetic quality are precisely those men who are least monogamous and most likely to be sexually unfaithful (e.g., see Al-Shawaf, Lewis, & Buss, 2015; Buss, 2003; Gangestad & Thornhill, 2008). Consequently, women face tradeoffs in their mate selection: they may not always have been able to reliably secure both “good genes” and long-term investment from the same man (Gangestad & Simpson, 2000). This dilemma creates the background selective conditions for the evolution of a dual female mating strategy of 1) long-term mating with men willing to commit resources and investment and 2) seeking men of high genetic quality for short-term sexual relations (Gangestad & Simpson, 2000).

Because ancestral women could not have directly observed men's genes, their detection of men's underlying genetic quality had to have been indirect – based on observable cues. Because androgens have immunosuppressive effects, androgenization may be a costly signal indicating high genetic quality (Evans, Goldsmith, & Norris, 2000; Peters, 2000; Rantala, Vainikka, & Kortet, 2003; Zahavi, 1975). Selection may thus have favored a female preference for androgenized men as short-term mating partners (see Gangestad & Simpson, 2000; Gangestad & Thornhill, 1997; Penton-Voak et al., 1999). Empirical data support this hypothesis. Evidence suggests that women prefer short-term mates who exhibit above-average levels of a wide variety of characteristics associated with androgenization, androgenization, ranging from low vocal

frequencies (see Feinberg et al., 2006) to a v-shaped torso (Hughes & Gallup, 2003).

An ancestral woman could have reaped both genetic and non-genetic benefits from engaging in a sexual affair (see Greiling & Buss, 2000), but she also could have incurred substantial costs from engaging in such liaisons. If she engaged in such an affair and was discovered, she could have lost her long-term partner, suffered reputational damage, and seen a decrease in her probability of securing future long-term mates (Forstmeier, Martin, Bolund, Schielzeith, & Kempenaers, 2011; Greiling & Buss, 2000). Selection would thus have favored extra-pair mating mechanisms in women that were only activated under conditions in which the probabilistic benefits outweighed the probabilistic costs.

The benefits of an extra-pair copulation with a man of high genetic quality would have depended on the genetic makeup of the woman's current long-term mate (Gangestad, Thornhill, & Garver-Apgar, 2005; Haselton & Gangestad, 2006; Pillsworth & Haselton, 2006). For example, a woman mated to a man of high genetic quality would have gained little genetic benefit from an extra-pair affair; the “good genes” she could have potentially obtained for her offspring would have been, at best, minimally superior to those she could have obtained by copulating with her long-term mate. Such minimal benefits are unlikely to offset the potential costs of such an affair. On the other hand, a woman mated to a man of low genetic quality could have reaped substantial genetic benefits by engaging in short-term liaisons with a man of high genetic quality (Gangestad et al., 2005; Haselton & Gangestad, 2006; Pillsworth & Haselton, 2006).

Women's short-term mating psychology may also serve additional functions besides the acquisition of high quality genes for their offspring. These additional functions include long-term mate-switching (Greiling & Buss, 2000), obtaining valuable resources (Symons, 1979), securing physical protection (Smith, 1984; see also Smuts, 1985), and elevating their social status by consorting with high-status men (Smith, 1984). Among men, androgenization is associated with the ability to effectively provide protection (Archer & Thanzami, 2009; Brewer & Riley, 2009) as well as social status and resource earnings (Newman, Guinn Sellers, & Josephs, 2005). Consequently, ancestral women would have been more likely to secure these benefits when they selected androgenized men as their short-term mating consorts. Regardless of which specific functions were served by women's extra-pair affairs (good genes, economic resources, physical protection, or more than one of these), the benefits of such an affair would have been greater, on average, for women mated to less androgenized men than for those mated to highly androgenized men (see Greiling & Buss, 2000).

This reasoning points toward the overarching hypothesis of a link between the alleles that ancestral men possessed at androgenization-linked genetic loci and their likelihood of facing sexual infidelity by their long-term mates.

4. The androgen receptor (AR) gene

The AR gene is an androgen-activated transcription factor that regulates gene expression throughout the brain and body (Bhasin, Woodhouse, & Storer, 2001; Simerly, Chang, Muramatsu, & Swanson, 1990). In humans, the AR gene is polymorphic, with the number of CAG codon repeats in the first exon ranging from nine to 31 (Alevizaki et al., 2003; Edwards, Hammond, Jin, Caskey, & Chakraborty, 1992; Lukaszewski & Roney, 2011; Simmons & Roney, 2011). Shorter sequences of CAG repeats are associated with greater expression of the AR protein (Choong, Kempainen, Zhou, & Wilson, 1996) and enhanced transcriptional activity (Chamberlain, Driver, & Miesfeld, 1994). Consequently, shorter sequences of CAG repeats translate into stronger phenotypic effects of androgens. For example, men with fewer CAG repeats exhibit a greater physiological response to testosterone than do men with a longer sequence of CAG repeats (Zitzmann & Nieschlag,

2007, see also Casella, Maduro, Lipshultz, & Lamb, 2001; von Eckardstein et al., 2001).

Because the AR gene is expressed throughout the body and brain, the number of CAG repeats should exert phenotype-wide effects: fewer CAG repeats should be associated with greater androgenization of the whole organism (Simmons & Roney, 2011). Indeed, shorter sequences of CAG repeats in men are associated with higher rates of sperm production (von Eckardstein et al., 2001) and lower levels of body fat (Nielsen et al., 2010; Zitzmann, Gromoll, von Eckardstein, & Nieschlag, 2003), as well as greater muscle mass (Nielsen et al., 2010) and upper body strength (Lukaszewski & Roney, 2011; Simmons & Roney, 2011). Moreover, these organism-wide effects are not limited to morphology; shorter CAG repeats are associated with important social, psychological, and personality outcomes, including higher levels of prestige (Simmons & Roney, 2011), greater extraversion (Lukaszewski & Roney, 2011) and increased psychological dominance (Simmons & Roney, 2011).

5. The AR gene and men's sexual jealousy

If ancestral women with less androgenized mates were more likely to engage in extra-pair affairs than women with more androgenized mates, and men's androgenization is related to polymorphism at the AR locus, then ancestral men with longer CAG repeats at the AR locus would have been at heightened risk of their mates' infidelity. Such a link between men's allelic length at the AR locus and the risk of their mates' infidelity would have created selection pressures for the evolution of a condition-dependent design feature of men's sexual jealousy mechanisms: selection should have favored sexual jealousy mechanisms that regulate their output partly as a function of a man's number of CAG repeats. We therefore hypothesized that men with longer sequences of CAG repeats should exhibit higher levels of sexual jealousy.

If men's sexual jealousy mechanisms calibrate their psychological output according to a man's number of CAG repeats, then men with a longer sequence of CAG repeats should exhibit an elevated baseline alertness to their mates' potential infidelity. "Cognitive jealousy" (Pfeiffer & Wong, 1989) – which includes psychological states such as being suspicious that one's mate is interested in having, or is surreptitiously pursuing, an intimate relationship with someone else – can functionally direct men's attention toward the potential problem of their mates' unfaithfulness. On this basis, we predicted that men with a longer sequence of CAG repeats experience jealous cognitions more frequently.

Men with a longer sequence of repeats should also have a lower threshold for jealousy in response to cues potentially indicative of their mates' infidelity. Selection would have favored sexual jealousy mechanisms that calibrated men's perceptions of the likelihood of their mates' infidelity based on cues probabilistically linked to infidelity (Nettle & Clegg, 2008). However, these cues are imperfect indicators of actual unfaithfulness (Shackelford & Buss, 1997). In trying to discriminate signals of infidelity from noise, a man could 1) correctly perceive his partner's fidelity or infidelity, 2) incorrectly perceive infidelity when his partner is faithful, or 3) fail to detect infidelity when his partner has been unfaithful. If the mates of men with longer CAG repeat lengths were more likely to be unfaithful, then, probabilistically, a given cue to infidelity would more often have reflected actual infidelity by these men's mates. Failing to direct attention to such cues would therefore have been more costly, on average, for men with longer CAG repeat lengths. Based on this logic, we predicted that men with longer sequences of CAG repeats would exhibit heightened perceptions of infidelity in response to ambiguous infidelity cues compared to men with shorter alleles rating the same cues.

Based on the same reasoning, if men with longer CAG repeat lengths and men with fewer repeats were to maintain the same baseline levels of emotional sensitivity to cues to infidelity, then men with longer repeat lengths would have more frequently failed to be emotionally jealous when cues truly did indicate their mates' infidelity. We therefore

hypothesized that men's sexual jealousy mechanisms regulate their emotional output according to variation at the AR locus. Specifically, we predicted that men with a greater number of CAG repeats would experience heightened emotional upset in response to ambiguous infidelity cues, relative to men with shorter CAG repeat sequences.

6. The current study

The current study tested the hypotheses that men with longer sequences of CAG repeats experience more jealous cognitions (Hypothesis 1), including more frequent thoughts about their mates' infidelity and elevated perceptions of infidelity in response to ambiguous cues, as well as heightened levels of emotional jealousy (Hypothesis 2), such as greater emotional distress in response to ambiguous cues.

7. Method

7.1. Participants

One hundred three men ($M_{age} = 20.22$, $SD = 2.65$, Range: 18–33) currently involved in a committed heterosexual relationship of at least six months ($M = 27.54$ months, $SD = 16.60$) participated as part of a larger study on relationship dynamics. Fifty-eight percent of the sample self-identified as "Caucasian," 23% as "Hispanic," 2% as "African American," 13% as "Asian," 1% as "Indian," and 2% as "Other." Participants were recruited from an introductory psychology subject pool, as well as by posting flyers on a university campus and advertising on social networking sites (e.g., Facebook). Participants from the introductory psychology subject pool received partial course credit for their participation.

7.2. Materials

7.2.1. Genetic sample and genotype fragment analysis

Catch-All™ Sample Collection Swabs (Soft Pack) from Epicentre (Madison, WI, USA) were used to collect buccal cells. Qiagen QIAmp DNA Mini Kits (Chatsworth, CA, USA) were used to extract genomic DNA from the swabs, and the spin protocol described in the QIAmp DNA Mini and Blood Mini Handbook (Qiagen, 2010) for DNA purification from buccal swabs was followed.

The region of interest was amplified by polymerase chain reaction (PCR) following the protocol described in Krithivas et al. (1999). The sense primer sequence was 5'-TCCAGAATCTGTCCAGAGCGTGC-3' and the antisense primer sequence was 3'-GCTGTGAAGGTTGCTGTCCTCAT-5'. Samples were denatured at 94 °C for 3 min, followed by 35 cycles of 30 s at 94 °C, 30 s at 60 °C, and 1 min at 72 °C. The final extension step consisted of 8 min at 72 °C. Gel electrophoresis was used to ensure that the PCR multiplied the target sequence. PCR products were sent to a DNA Sequencing Facility for fragment analysis. Resultant data were analyzed using SoftGenetics® GeneMarker® Software (State College, PA, USA).

7.2.2. Sexual jealousy

To capture the distinct psychological states associated with sexual jealousy, sexual jealousy was operationalized in the following ways.

Sexually jealous cognitions.

Multidimensional jealousy scale – cognitive component. The cognitive section of the Multidimensional Jealousy Scale (Pfeiffer & Wong, 1989) was used to assess individuals' frequency of jealous thoughts. Eight items use the root question "How often do you have the following thoughts about [your partner]?" Sample items include "suspect that [my partner] is secretly seeing someone of the opposite sex" and "suspect that [my partner] may be physically intimate with another member of the opposite sex behind [my] back." Participants report their frequency of experiencing these cognitions on a 7-point Likert-type scale (1 = never, 7 = all the time).

Perceptions of infidelity. An instrument displayed each of the 72 cues to infidelity presented in Shackelford and Buss (1997), and asked participants to estimate the probability of their mates' infidelity, given the observation of each cue. Estimates were made on a sliding scale ranging from 0% to 100%.

Sexually jealous emotions.

Multidimensional jealousy scale – emotional component. The emotional section of the Multidimensional Jealousy Scale assessed individuals' emotional distress in response to events potentially indicative of infidelity. Eight items ask participants how they would feel in response to events such as “[Your partner] shows a great deal of interest or excitement in talking to someone of the opposite sex.” Items are rated on a seven-point Likert-type scale (1 = very pleased, 7 = very upset).

Emotional upset. An author-generated extension of the emotional section of the MJS used the same root question (“How would you emotionally react to the following situations?”) and rating scale to assess participants' emotional upset in response to each of the 72 ambiguous cues to infidelity presented in Shackelford and Buss (1997).

7.3. Procedure

Upon arriving at the laboratory, participants were provided with an informed consent form that outlined the procedures, instruments, and measurements described above. All participants provided written informed consent.

After consenting to participate, participants were escorted to a laboratory room approved by Environmental Health & Safety (EHS) at the university for collection, handling, storage, and disposal of biological samples.

Participants were provided with a buccal swab and instructed to vigorously rub the cotton tip along the inside of both cheeks for 30 s. The swab was then air-dried for 10 min, returned to its original packaging, and frozen at -20°C until processing.

After completing buccal cell collection, participants were guided by a researcher to a private laboratory computer room to complete a survey that included the instruments described above.

8. Results

8.1. Data preparation and statistical analysis

The DNA extraction procedures resulted in successful extraction from 97 men. The number of CAG codon repeats ranged from 15 to 31 ($M = 23.7, SD = 2.75$), a distribution consistent with previous literature (e.g., Alevizaki et al., 2003; Edwards et al., 1992; Lukaszewski & Roney, 2011; Simmons & Roney, 2011).

Although the cognitive and affective components of sexual jealousy can be conceptualized as distinct psychological states, and previous literature has cleaved jealousy into distinct components at the cognitive-emotional boundary (e.g., Pfeiffer & Wong, 1989), it nonetheless was plausible that our cognitive and affective measures tapped a single underlying variable. To address this, we conducted Principal Components Analyses (rotation: direct oblimin) on the 144 items based on the cues to infidelity presented in Shackelford and Buss (1997). For the purpose of determining whether cognitive jealousy and emotional jealousy reflect distinct psychological constructs, these items were a particularly good source of data; they consisted of 72 pairs of parallel questions – one cognitive and one emotional for each cue.

Visual examination of the PCA scree plot indicated a solution consisting of between two and five components. To identify the most parsimonious of these tenable factorial solutions, we used the criterion of minimizing the total number of items that either exhibited absolute loadings of at least 0.35 on *more than one* component or failed to have such a loading on *any* component (e.g., see Al-Shawaf & Lewis, 2013). This revealed a two-factor solution that cleaved the items very neatly along a cognitive-emotional border. Seventy of the 72 items assessing

cognitive jealousy (within-instrument reliability: $\alpha = 0.98$) loaded onto the first component, and over 80% of the items assessing emotional jealousy (within-instrument reliability: $\alpha = 0.95$) loaded onto the second component. Furthermore, none of the items assessing emotional jealousy loaded onto the first component, and none of the items assessing jealous cognitions loaded onto the second component. Based on these results, which indicated that cognitive jealousy and emotional jealousy tapped distinct psychological constructs, we proceeded to test the current study's hypotheses. Because all study hypotheses were directional, we conducted one-tailed tests.

8.2. The AR gene and cognitive sexual jealousy

As described in the Method section, two distinct instruments were used to assess individuals' jealous cognitions. The cognitive component of the MJS assessed participants' frequency of sexually jealous thoughts. The Perceptions of Infidelity instrument asked participants about their perceptions of their mates' fidelity based on ambiguous cues. The former instrument assesses spontaneous sexually jealous cognitions, whereas the latter measures jealous cognitions elicited by environmental cues. However, it seemed plausible that these measures tapped a single, underlying dimension of jealous cognitions.

Somewhat consistent with this proposal, individuals' scores on the Perceptions of Infidelity instrument and the cognitive section of the MJS were positively correlated, $r(95) = 0.20, p < 0.05$. Given the conceptual reasons to believe that these two facets of cognitive jealousy reflect the output of a common underlying psychological mechanism, and the observed inter-correlation between the scales, we created a composite cognitive jealousy score by standardizing individuals' scores on these two instruments and computing the mean of these values.

In support of Hypothesis 1, men's number of CAG repeats correlated positively with their sexually jealous cognitions, $r(95) = 0.27, p < 0.01$. Given the relatively low correlation between the instruments, however, it was possible that they tapped distinct psychological dimensions. For this reason, we followed the overall finding with instrument-level analyses to determine what was driving the relationship between the AR locus and men's cognitive jealousy. These analyses revealed that the relationship between men's allelic length and their tendency to spontaneously experience sexually jealous thoughts was in the expected direction (cognitive section of the MJS, $r(95) = 0.12, p = 0.13$), but the overall link between men's cognitive jealousy and the AR locus was driven by heightened perceptions of infidelity among men with longer CAG repeat sequences in response to uncertain cues (Perceptions of Infidelity instrument, $r(95) = 0.26, p < 0.01$).

8.3. The AR gene and emotional sexual jealousy

As with our measures of jealous cognitions, we assessed men's emotional jealousy using two instruments that could capture distinct psychological dimensions, or alternatively could tap a single underlying affective dimension. Consistent with the latter proposal, men's scores on the emotional component of the MJS were strongly positively correlated with their emotional upset in response to ambiguous cues to infidelity, $r(95) = 0.64, p < 0.001$. We therefore created a composite emotional jealousy score by computing the mean of participants' standardized scores on these two instruments.

In support of Hypothesis 2, men's number of CAG codon repeats correlated positively with their emotional jealousy, $r(95) = 0.24, p < 0.01$. To parallel the cognitive jealousy analyses, we followed up this overall result with instrument-level analyses to determine which (or both) of the measures was driving the link between the AR locus and men's emotional jealousy. These analyses indicated that the relationship between men's allelic length and their emotional jealousy was manifest across both the emotional component of the Multidimensional Jealousy Scale, $r(95) = 0.19, p = 0.03$, and the instrument based on the

ambiguous cues presented in Shackelford and Buss (1997), $r(95) = 0.24, p < 0.01$.

8.4. Cognitive and emotional jealousy: independent links to the AR gene?

It was plausible that the observed bivariate correlation between men's AR allelic length and their emotional jealousy was driven by a link between the AR gene and men's jealous cognitions, and vice versa. To address this, we conducted partial correlations between men's number of CAG repeats at the AR locus and these distinct components of jealousy while controlling for the other component. Corroborating the results of the PCA, and in line with the notion that cognitive sexual jealousy and emotional sexual jealousy tap distinct psychological dimensions, over 97% of the variance in the composite cognitive jealousy and emotional jealousy variables was *non-overlapping*. In line with this degree of orthogonality, the link between men's allelic length at the AR locus and their emotional jealousy remained robust after controlling for their jealous cognitions, partial $r(94) = 0.19, p = 0.03$, as did the link between the AR gene and men's jealous cognitions after controlling for their emotional jealousy, partial $r(94) = 0.23, p = 0.01$.

9. Discussion

This study tested the overarching hypothesis that men's sexual jealousy mechanisms are calibrated to genetic polymorphism at the AR locus. In support of this hypothesis, we found that men's number of CAG codon repeats was positively associated with their sexual jealousy, and that this relationship manifested itself in both men's cognitions and emotions. The current study thereby contributes novel evidence to a growing body of recent research linking polymorphism at the AR locus to individual differences in psychology (e.g., Lukaszewski & Roney, 2011; Simmons & Roney, 2011), and more broadly, to evolutionary psychological research documenting theoretically anchored links between genetic variation and individual differences in psychology. To our knowledge, the gene-sexual jealousy links documented here also represent the first evidence pointing toward the heritability of sexual jealousy. The possibility that these gene-linked differences in psychology reflect individual differences arising from sex-typical psychological mechanisms, however, raises an important but overlooked question about the heritability of psychological differences.

9.1. Polymorphic mechanisms, or polymorphic inputs into sex-typical mechanisms?

Gene-linked psychological differences such as those observed in the current study have historically been attributed to “direct” genetic models. Under such models, heritable differences in psychology are assumed to reflect the coding of different neural substrates by different alleles (e.g., Bouchard & Loehlin, 2001; Depue & Collins, 1999; Kagan, 1998; McCrae & Costa, 2008; Nettle, 2006; Penke, Denissen, & Miller, 2007).

However, the observation of gene-linked psychological differences is equally consistent with a condition-dependent model (Lewis, 2015; Lukaszewski & Roney, 2011; Tooby & Cosmides, 1990). Individual differences in sexual jealousy may be the result of sex-typical psychological mechanisms that calibrate their output according to the conditional input they receive as a consequence of genetic variation at the AR locus.

The question that therefore looms about the relationship between genetic polymorphisms and psychological differences (e.g., Lukaszewski & Roney, 2011; Sen et al., 2004; Tochigi et al., 2006; Westberg et al., 2009) is whether these polymorphisms are associated with 1) different psychological mechanisms (and their neurobiological substrates) across individuals, or rather with 2) variable inputs into species- or sex-typical psychological mechanisms that consequently yield variable output: different psychological trait levels across individuals.

Consistent with this latter perspective, Lukaszewski and Roney (2011) demonstrate that genetic polymorphism at the AR locus is associated with individual differences in psychology, but that downstream correlates of the gene predict these psychological differences above and beyond genetic polymorphism alone. This indicates that the gene-psychology relationship cannot be solely attributed to direct genetic influence. Instead, these results point toward genes playing an important role in influencing individual differences in human psychology, but through a complex pathway in which the genes' downstream products serve as input into species-typical or sex-typical information-processing mechanisms that calibrate psychological output accordingly.

Because both “direct” genetic models and condition-dependent models lead to gene-psychology links that appear heritable in behavioral and molecular genetic studies, heritability alone cannot be used to discriminate between these two models. Rather, testing for evidence of functional, information-processing design is ultimately necessary to discriminate between these alternative, but not mutually exclusive, pathways by which genes exert their influence on psychology (see Lukaszewski & Roney, 2011). Testing for evidence of such design represents a key future direction for both molecular genetic research informed by evolutionary psychology and evolutionary psychological research informed by molecular genetics.

9.2. The AR locus as a candidate gene

The current study integrated an evolutionary psychological approach with molecular genetics by investigating covariation between the polymorphic AR locus and men's sexual jealousy. Although the candidate-gene approach has lost some of the favor with which it was once received, we believe that two key features of the current study distinguish it from the type of candidate-gene study that has fallen under criticism. One critique is that, because candidate genes are often selected based on prior knowledge of their relevance to the phenotype under investigation, they are not driven by a priori hypotheses. We acknowledge that it is questionable to call a candidate-gene study “hypothesis-driven” if there are already known links between the phenotype of interest and the candidate gene. However, the degree to which this criticism is warranted depends on the “proximity” between the phenotype under investigation and the phenotypic feature known to be associated with the candidate gene. For example, if the phenotype of interest is a neurological disease such as multiple sclerosis (MS), and there are known effects of a candidate gene (e.g., TGFB2) on the survival of dopaminergic neurons (Goris et al., 2007), then it would be dubious for a researcher to call a predicted association between TGFB2 and MS “hypothesis-driven.”¹ The known phenotypic correlate is too close to the phenotype under investigation. In the context of the current study, however, the majority of known correlates of the AR gene are not even in the same domain of knowledge. The AR locus is known to have effects on gene expression (Bhasin et al., 2001; Choong et al., 1996; Simerly et al., 1990), physiology (Casella et al., 2001; von Eckardstein et al., 2001; Zitzmann & Nieschlag, 2007), and morphology (Nielsen et al., 2010; Zitzmann et al., 2003). The current study's hypotheses were about men's psychology. In the absence of theoretically anchored evolutionary psychological reasoning, the known phenotypic correlates of the AR gene simply do not point toward an expected relationship between the AR locus and men's jealousy. Rather, truly a priori theoretical reasoning is needed to link the AR gene to the psychological features under current investigation.

A second issue with candidate-gene studies deals with their reproducibility. A principal reason that prior candidate-gene findings have not been easily reproduced is that the original samples have been genetically homogeneous (Hutchison, Stallings, McGeary, & Bryan, 2004). The

¹ Note that the criticism of such candidate-gene studies as not being hypothesis-driven does not necessarily undermine the importance or validity of the observed relationship between the candidate gene and the phenotype of interest.

present sample's diversity and representativeness (42% non-white, with at least six ethnicities represented) suggests that the current research could be free from this issue. Ideally, this would be determined empirically. We hope to see this addressed by other researchers in future work.

9.3. Limitations and future directions

9.3.1. The AR gene and sexual jealousy among women

We collected genetic samples only from men for two reasons. First, the current study's hypotheses pertained specifically to men. Second, the AR gene is located on the X chromosome. Women, but not men, can be heterozygous at this locus, and the effects of heterozygosity at this locus are not known. For example, a woman who possesses one shorter allele and one longer allele might have higher – or lower – androgen sensitivity than a woman who possesses two medium length AR gene alleles. Because men, on the other hand, possess only one copy of the AR gene, they are effectively homozygous. A man with an allele with a shorter number of CAG repeats produces androgen receptors that are more sensitive to androgens than does a man with a longer allele. For these reasons, we actively chose to examine the relationship between the AR gene and sexual jealousy specifically among men. This nonetheless represents a study limitation, and future research could benefit from employing molecular genetic methods in the investigation of individual differences in sexual jealousy among women.

9.3.2. Jealousy in response to sexual versus emotional infidelity

A substantial body of literature documents sex differences in jealousy, in particular in response to sexual versus emotional infidelity (see Buss, 2013 for a review; see also Park, Wieling, Buunk, & Massar, 2008; Fussell, Rowe, & Park, 2011; Bendixen, Kennair, & Buss, 2015). It would be ideal to separately examine the relationships between the AR gene and 1) jealousy in response to emotional infidelity and 2) jealousy in response to sexual infidelity. However, the inventories of items that served as the source for the current study's measures do not permit such a differentiation. For example, the cues to infidelity from Shackelford and Buss (1997) include items such as “her clothing style suddenly changes” and “she is unusually critical of her partner.” Such behaviors might cue emotional infidelity, sexual infidelity, or both. Consequently, the nature of these cues does not permit the differentiation between sexual and emotional infidelity. Whether allelic length is linked to jealousy in response to sexual infidelity, emotional infidelity, or both is an open question that awaits future research.

10. Conclusion

As hypothesized, men's sexual jealousy tracked polymorphism at the AR gene: men with longer CAG repeats sequences reported a) heightened perceptions of infidelity and b) greater emotional upset in response to ambiguous social and environmental cues of potential infidelity. These findings contribute novel evidence to a growing body of literature linking genetic polymorphism to individual differences in psychology. Although the current findings cannot adjudicate between a condition-dependent model and a more direct model of genetic influence, only the condition-dependent model generates the novel, theoretically anchored predictions about human psychology that provided the impetus to investigate the relationship between the AR locus and men's sexual jealousy. Indeed, in the absence of a condition-dependent evolutionary framework, this relationship could have gone undiscovered and remained unknown. We hope that this research highlights the utility of condition-dependent evolutionary models for understanding how universal psychological mechanisms can produce flexible, context-sensitive psychological outcomes. More broadly, we hope that this research illustrates both the heuristic value of an evolutionary psychological framework for molecular genetic psychological research, and the utility

of molecular genetic methods for increasing the sophistication and empirical progress of evolutionary psychology.

References

- Alevizaki, M., Cimponeriu, A. T., Garofallaki, M., Sarika, H. L., Alevizaki, C. C., Papamichael, C., ... Mavrikakis, M. (2003). The androgen receptor gene CAG polymorphism is associated with the severity of coronary artery disease in men. *Clinical Endocrinology*, *59*, 749–755.
- Al-Shawaf, L., & Lewis, D. M. G. (2013). Exposed intestines and contaminated cooks: Sex, stress, & satiation predict disgust sensitivity. *Personality and Individual Differences*, *54*, 698–702.
- Al-Shawaf, L., Lewis, D. M. G., & Buss, D. M. (2015). Disgust and mating strategy. *Evolution & Human Behavior*, *36*(3), 199–205.
- Archer, J., & Thanzami, V. (2009). The relation between mate value, entitlement, physical aggression, size, and strength among a sample of young Indian men. *Evolution & Human Behavior*, *30*(5), 315–321.
- Bendixen, M., Kennair, L. E. O., & Buss, D. M. (2015). Jealousy: Evidence of strong sex differences using both forced choice and continuous measure paradigms. *Personality and Individual Differences*, *86*, 212–216.
- Bhasin, S., Woodhouse, L., & Storer, T. W. (2001). Proof of the effect of testosterone on skeletal muscle. *Journal of Endocrinology*, *170*, 27–38.
- Bouchard, T. J., Jr., & Loehlin, J. C. (2001). Genes, evolution, and personality. *Behavior Genetics*, *31*, 243–273.
- Brewer, G., & Riley, C. (2009). Height, relationship satisfaction, jealousy and mate retention. *Evolutionary Psychology*, *7*, 477–489.
- Buss, D. M. (1995). Evolutionary psychology: A new paradigm for psychological science. *Psychological Inquiry*, *6*, 1–30.
- Buss, D. M. (2000). *The dangerous passion: Why jealousy is as necessary as love and sex*. New York: The Free Press.
- Buss, D. M. (2002). Human mate guarding. *Neuroendocrinology Letters*, *23*(4), 23–29.
- Buss, D. M. (2003). *The evolution of desire: Strategies of human mating*. New York, NY: Basic Books.
- Buss, D. M. (2013). Sexual jealousy. *Psychological Topics*, *22*, 155–182.
- Buss, D. M., & Greiling, H. (1999). Adaptive individual differences. *Journal of Personality*, *67*(2), 209–243. <http://dx.doi.org/10.1111/1467-6494.00053>.
- Buss, D. M., Larsen, R. J., Westen, D., & Semmelroth, J. (1992). Sex differences in jealousy: Evolution, physiology, and psychology. *Psychological Science*, *3*, 251–255.
- Casella, R., Maduro, M. R., Lipshultz, L. I., & Lamb, D. J. (2001). Significance of the polyglutamine tract polymorphism in the androgen receptor. *Urology*, *58*, 651–656.
- Chamberlain, N. L., Driver, E. D., & Miesfeld, R. L. (1994). The length and location of CAG trinucleotide repeats in the androgen receptor N-terminal domain affect transactivation function. *Nucleic Acids Research*, *22*, 3181–3186.
- Choong, C. S., Kempainen, J. A., Zhou, Z. X., & Wilson, E. M. (1996). Reduced androgen receptor gene expression with first exon CAG repeat expansion. *Molecular Endocrinology*, *10*, 1527–1535.
- Confer, J. C., Easton, J. A., Fleischman, D. S., Goetz, C. D., Lewis, D. M. G., Perilloux, C., et al. (2010). Evolutionary psychology: Controversies, questions, prospects, and limitations. *American Psychologist*, *65*, 110–126.
- Cosmides, L., & Tooby, J. (1997). Evolutionary psychology: A primer. Retrieved March 3, 2015, from the University of California, Santa Barbara, Center for Evolutionary Psychology Web site: <http://www.psych.ucsb.edu/research/cep/primer.html>.
- Daly, M., Wilson, M., & Weghorst, S. J. (1982). Male sexual jealousy. *Ethology and Sociobiology*, *3*, 11–27.
- Depue, R. A., & Collins, P. F. (1999). Neurobiology of the structure of personality: Dopamine, facilitation of incentive motivation, and extraversion. *Behavioral and Brain Sciences*, *22*, 491–569.
- Edwards, A., Hammond, H. A., Jin, L., Caskey, C. T., & Chakraborty, R. (1992). Genetic-variation at 5-trimeric and tetrameric tandem repeat loci in 4 human population groups. *Genomics*, *12*, 241–253.
- Evans, M. R., Goldsmith, A. R., & Norris, S. R. A. (2000). The effects of testosterone on antibody production and plumage coloration in male house sparrows (*Passer domesticus*). *Behavioral Ecology and Sociobiology*, *47*(3), 156–163. <http://dx.doi.org/10.1007/s002650050006>.
- Feinberg, D. R., Jones, B. C., Law Smith, M. J., Moore, F. R., DeBruine, L. M., Cornwell, R. E., ... Perrett, D. I. (2006). Menstrual cycle, trait estrogen level and masculinity preferences in the human voice. *Hormones and Behavior*, *49*, 215–222.
- Forstmeier, W., Martin, K., Bolund, E., Schielzeith, H., & Kempenaers, B. (2011). Female extrapair mating behavior can evolve via indirect selection on males. *Proceedings of the National Academy of Sciences*, *108*(26), 10608–10613.
- Fussell, N. J., Rowe, A. C. M., & Park, J. H. (2011). Masculinized brain and romantic jealousy: Examining the association between digit ratio (2D:4D) and between- and within-sex differences. *Personality and Individual Differences*, *51*, 107–111.
- Gangestad, S. W., & Simpson, J. A. (2000). The evolution of human mating: Trade-offs and strategic pluralism. *Behavioral and Brain Sciences*, *23*, 573–587. <http://dx.doi.org/10.1017/S0140525X0000337X>.
- Gangestad, S. W., & Thornhill, R. (1997). The evolutionary psychology of extrapair sex: The role of fluctuating asymmetry. *Evolution and Human Behavior*, *18*, 69–88.
- Gangestad, S. W., & Thornhill, R. (2008). Human oestrus. *Proceedings of the Royal Society B: Biological Sciences*, *275*(1638), 991–1000.
- Gangestad, S. W., Thornhill, R., & Garver-Apgar, C. E. (2005). Women's sexual interests across the ovulatory cycle depend on primary partner developmental instability. *Proceedings of the Royal Society of London B*, *272*(1576), 2023–2027. <http://dx.doi.org/10.1098/rspb.2005.3112>.

- Goetz, A. T., Shackelford, T. K., Romero, G. A., Kaighobadi, F., & Miner, E. J. (2008). Punishment, proprietariness, and paternity: Men's violence against women from an evolutionary perspective. *Aggression and Violent Behavior, 13*, 481–489.
- Goris, A., Williams-Gray, C. H., Foltynie, T., Brown, J., Maranian, M., Walton, A., ... Sawcer, S. J. (2007). Investigation of TGFB2 as a candidate gene in multiple sclerosis and Parkinson's disease. *Journal of Neurology, 254*(7), 846–848.
- Greiling, H., & Buss, D. M. (2000). Women's sexual strategies: The hidden dimension of extra-pair mating. *Personality and Individual Differences, 28*(5), 929–963. [http://dx.doi.org/10.1016/S0191-8869\(99\)00151-8](http://dx.doi.org/10.1016/S0191-8869(99)00151-8).
- Haselton, M. G., & Gangestad, S. W. (2006). Conditional expression of women's desires and men's mate guarding across the ovulatory cycle. *Hormones and Behavior, 49*, 509–518.
- Hughes, S. M., & Gallup, G. G., Jr. (2003). Sex differences in morphological predictors of sexual behaviors: Shoulder to hip and waist to hip ratios. *Evolution and Human Behavior, 24*, 173–178.
- Hutchison, K. E., Stallings, M., McGeary, J., & Bryan, A. (2004). Population stratification in the candidate gene study: Fatal threat or red herring? *Psychological Bulletin, 130*(1), 66–79.
- Kagan, J. (1998). *Galen's prophecy: Temperament in human nature*. New York: Basic Books.
- Krithivas, K., Yurgalevitch, S. M., Mohr, B. A., Wilcox, C. J., Batter, S. J., Brown, M., Longcope, C., ... Kantoff, P. W. (1999). Evidence that the CAG repeat in the androgen receptor gene is associated with the age-related decline in serum androgen levels in men. *Journal of Endocrinology, 162*(1), 137–142.
- Lewis, D. M. G. (2015). Evolved individual differences: Advancing a condition-dependent model of personality. *Personality and Individual Differences, 84*, 63–72.
- Lewis, D. M. G., Al-Shawaf, L., Conroy-Beam, D., Asao, K., & Buss, D. M. (2016). Evolutionary psychology: A how-to guide. *American Psychologist* (in press).
- Lukaszewski, A. W., & Roney, J. R. (2011). The origins of extraversion: Joint effects of facultative calibration and genetic polymorphism. *Personality and Social Psychology Bulletin, 37*(3), 409–421.
- McCrae, R. R., & Costa, P. T. (2008). The five factor theory of personality. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality psychology: Theory and research* (pp. 159–181) (3rd ed.). New York: Guilford Press.
- Nettle, D. (2006). The evolution of personality variation in humans and other animals. *American Psychologist, 61*(6), 622–631. <http://dx.doi.org/10.1037/0003-066X.61.6.622>.
- Nettle, D., & Clegg, H. (2008). Personality, mating strategies, and mating intelligence. In G. Geher, & G. Miller (Eds.), *Mating intelligence: Sex, relationships, and the mind's reproductive system* (pp. 121–134). New York, NY: Lawrence Erlbaum Associates.
- Newman, M., Guinn Sellers, J., & Josephs, R. (2005). Testosterone, cognition, and social status. *Hormones and Behavior, 47*, 205–211.
- Nielsen, T. L., Hagen, C., Wraae, K., Bathum, L., Larsen, R., Brixen, K., & Andersen, M. (2010). The impact of the CAG repeat polymorphism of the androgen receptor gene on muscle and adipose tissues in 20–29-year-old Danish men: Odense androgen study. *European Journal of Endocrinology, 162*, 795–804.
- Park, J., Wieling, M., Buunk, B., & Massar, K. (2008). Sex-specific relationship between digit ratio (2D:4D) and romantic jealousy. *Personality and Individual Differences, 44*, 1039–1045.
- Penke, L., Denissen, J. J. A., & Miller, G. F. (2007). The evolutionary genetics of personality. *European Journal of Personality, 21*(5), 549–587. <http://dx.doi.org/10.1002/per.629>.
- Penton-Voak, I. S., Perrett, D. I., Castles, D., Burt, M., Koyabashi, T., & Murray, L. K. (1999). Female preference for male faces changes cyclically. *Nature, 399*, 741–742.
- Peters, A. (2000). Testosterone treatment is immunosuppressive in superb fairy-wrens, yet free-living males with high testosterone are more immunocompetent. *Proceedings of the Royal Society of London B, 267*, 883–889.
- Pfeiffer, S. M., & Wong, P. T. P. (1989). Multidimensional jealousy. *Journal of Social and Personal Relationships, 6*(2), 181–196. <http://dx.doi.org/10.1177/026540758900600203>.
- Pillsworth, E. G., & Haselton, M. G. (2006). Male sexual attractiveness predicts differential ovulatory shifts in female extra-pair attraction and male mate retention. *Evolution & Human Behavior, 27*, 247–258.
- Rantala, M. J., Vainikka, A., & Kortet, R. (2003). The role of juvenile hormone in immune function and pheromone production trade-offs: A test of the immunocompetence handicap principle. *Proceedings of the Royal Society of London B, 270*, 2257–2261.
- Shackelford, T. K., & Buss, D. M. (1997). Cues to infidelity. *Personality and Social Psychology Bulletin, 23*(10), 1034–1045. <http://dx.doi.org/10.1177/01461672972310004>.
- Schützwohl, A. (2008). The disengagement of attentive resources from task- 192 irrelevant cues to sexual and emotional infidelity. *Personality and Individual Differences, 44*, 633–644.
- Sen, S., Burmeister, M., & Ghosh, D. (2004). Meta-analysis of the association between a serotonin transporter promoter polymorphism (5-HTTLPR) and anxiety-related personality disorders. *American Journal of Genetics Part B: Neuropsychiatric Genetics, 127B*(1), 85–89.
- Simerly, R. B., Chang, C., Muramatsu, M., & Swanson, L. W. (1990). Distribution of androgen and estrogen-receptor messenger RNA-containing cells in the rat brain — An in situ hybridization study. *Journal of Comparative Neurology, 294*, 76–95.
- Simmons, Z. L., & Roney, J. R. (2011). Variation in CAG repeat length of the androgen receptor gene predicts variables associated with intrasexual competitiveness in human males. *Hormones and Behavior, 60*, 306–312.
- Smith, R. L. (1984). Human sperm competition. In R. L. Smith (Ed.), *Sperm competition and the evolution of mating systems* (pp. 601–659). New York, NY: Academic Press.
- Smuts, B. B. (1985). *Sex and friendship in baboons*. New York: Aldine Publishing.
- Symons, D. (1979). *The evolution of human sexuality*. New York: Oxford University Press.
- Tochigi, M., et al. (2006). Association between dopamine D4 receptor (DRD4) exon III polymorphism and neuroticism in the Japanese population. *Neuroscience Letters, 398*(3), 333–336. <http://dx.doi.org/10.1016/j.neulet.2006.01.020>.
- Tooby, J., & Cosmides, L. (1990). On the universality of human nature and the uniqueness of the individual: The role of genetics and adaptation. *Journal of Personality, 58*(1), 17–67. <http://dx.doi.org/10.1111/j.1467-6494.1990.tb00907.x>.
- von Eckardstein, S., Syska, A., Gromoll, J., Kamischke, A., Simoni, M., & Nieschlag, E. (2001). Inverse correlation between sperm concentration and number of androgen receptor CAG repeats in normal men. *The Journal of Clinical Endocrinology and Metabolism, 86*, 2585–2590.
- Westberg, L., Henningsson, S., Landen, M., Annerbrink, K., Melke, J., Nilsson, S., ... Eriksson, E. (2009). Influence of androgen receptor repeat polymorphisms on personality traits in men. *Journal of Psychiatry and Neuroscience, 34*, 205–213.
- Williams, G. C. (1966). *Adaptation and natural selection: A critique of some current evolutionary thought*. United States: Princeton University Press.
- Wolf, M., van Doorn, G. S., Leimar, O., & Weissing, F. J. (2007). Life-history trade-offs favour the evolution of animal personalities. *Nature, 447*, 581–584. <http://dx.doi.org/10.1038/nature05835>.
- Zahavi, A. (1975). Mate selection—A selection for a handicap. *Journal of Theoretical Biology, 53*, 205–214.
- Zitzmann, M., & Nieschlag, E. (2007). Androgen receptor gene CAG repeat length and body mass index modulate the safety of long-term intramuscular testosterone undecanoate therapy in hypogonadal men. *The Journal of Clinical Endocrinology and Metabolism, 92*, 3844–3853.
- Zitzmann, M., Gromoll, J., von Eckardstein, A., & Nieschlag, E. (2003). The CAG repeat polymorphism in the androgen receptor gene modulates body fat mass and serum concentrations of leptin and insulin in men. *Diabetologia, 46*, 31–39.