Testing Evolutionary Hypotheses Regarding Individual Differences in Human Mating Strategies

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Abstract

Finding and attracting a mate is an enormous component of human life, and indeed the life of all sexually reproducing organisms. In this search, we share many common goals in attaining a partner. For instance, most people typically seek a partner who is kind, attractive, and funny. Yet, we also differ remarkably in the traits that we desire in our partners and what drives us towards choosing some mates over others. This variation represents somewhat of a Darwinian paradox, as selection typically reduces variation in traits under strong selection, such as those relating to mating strategy. To resolve this paradox, evolutionary psychologists have proposed adaptive mechanisms that cause variation in mating strategy in response to environmental contingencies. In this thesis, I present tests of a number of these hypotheses, and integrate approaches from evolutionary behavioural genetics to provide alternative explanations as to how variation in mating strategy arises.

Firstly, I review broad evolutionary approaches to explaining variation in psychological traits. Specifically, I introduce evolutionary literature regarding a proposed trade-off between parenting traits and signals of genetic quality in males that permeates approaches to various aspects of mate choice. Evidence for and against this trade-off is reviewed in the context of the maintenance of variation in facial masculinity in males and inter-partner variation in female orgasm frequency during penetrative sex. I then introduce behavioural genetics as an approach to understanding both genetic and environmental causes of variation in human mating strategy.

In my first empirical study, I present a paper testing evolutionary hypotheses regarding female variation in orgasm frequency in response to male characteristics. Where previous research has focused on women reporting on a single partner and observing male characteristics across the sample, this paper had females report on partners with whom orgasm was easy and with whom orgasm was difficult. This controls for the possibility of confounds relating to between-subject studies and increases our power to detect male traits associated with orgasm over and above between women differences. Furthermore, the study also included measures of male sexual behaviour, as these are highly likely to be related to female orgasmability. We showed mixed support for evolutionary theories regarding variation in the female orgasm, but were unable to distinguish the two leading hypotheses. Additionally, we found important contributions of male sexual behaviour to female orgasm. Consequently, little can be inferred about the role of variation in the evolution of the female orgasm. However, we demonstrate that within-subjects designs are able to detect between-partner variance with high resolution. We also demonstrate that future studies need account for male sexual behaviour when attempting to explain adaptive variation in female orgasm between different male partners.
I then present a paper providing evidence that facial dimorphism is an important factor in intrasexual competition. In this study, male participants rated male faces for dominance at increasing levels of facial masculinity (i.e., feminised and masculinised faces) and facial hair (i.e., clean-shaven, stubble, full beard). Participants also provided responses to an affective prime using the same facial images paired with words associated with either dominance or submissiveness. Explicit, but not implicit, ratings of dominance increased linearly with facial hair and facial masculinity, respectively. Facial hair and facial masculinity also interacted such that the effects of facial masculinity on dominance diminished as facial hair increased. This indicates that beards may serve to increase perceptions of dominance in the case that males lack masculine facial structure.

Twin studies are then introduced as a method of partitioning environmental and genetic causes of variance. As a demonstration of the utility of twin studies, I present an empirical paper investigating the genetic architecture of disgust sensitivity. Disgust sensitivity is believed to serve an adaptive purpose by motivating avoidance of pathogens, costly sexual encounters, and social transgressions. I observed a substantial genetic basis to variation in pathogen, sexual, and moral disgust (~50% of variation). Additional modelling revealed a common genetic basis to all three domains of disgust, and specific genes influencing each of pathogen and sexual disgust, but not moral disgust. This suggests that moral disgust may have emerged after pathogen and sexual disgust on an evolutionary timeline and co-opted existing genetic architecture.

I then apply the same twin modelling methods to the most prevalent form of partner selection in humans: assortative mating. Using data from over 6,000 families, I investigate a genetic basis to self-similarity partnering across a number of physical (height and body mass index [BMI]), and psychological traits (personality, social attitudes, religiosity, etc.). Across all traits, small but significant familial effects were observed (7%) on variation in self-similarity partnering. The remaining variance was accounted for by residual factors. The causal mechanisms of assortative mating are unknown, but genetic influences appear to relatively small which is consistent with non-human research.

These results are discussed in the context of evolutionary psychology more broadly. I provide commentary on the designs used to examine evolutionary hypotheses in this thesis and their utility in future research. I also discuss possible consequences of neglecting genetics research when exploring the evolution of individual differences. I then introduce the synthesis of behavioural genetics and evolutionary psychology as a means of furthering the field, providing a number of examples of extant theories that could benefit from the use of the classical twin design and genomic data.
Declaration by author

This thesis is composed of my original work, and contains no material previously published or written by another person except where due reference has been made in the text. I have clearly stated the contribution by others to jointly-authored works that I have included in my thesis.

I have clearly stated the contribution of others to my thesis as a whole, including statistical assistance, survey design, data analysis, significant technical procedures, professional editorial advice, and any other original research work used or reported in my thesis. The content of my thesis is the result of work I have carried out since the commencement of my research higher degree candidature and does not include a substantial part of work that has been submitted to qualify for the award of any other degree or diploma in any university or other tertiary institution. I have clearly stated which parts of my thesis, if any, have been submitted to qualify for another award.

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Publications during candidature

Publications as first author


Publications as a Contributing Author


Book chapters


Conference presentations as first author


Publications included in this thesis

Incorporated as section 3

Incorporated as section 4
Conducted analyses (50%)
Wrote manuscript (60%)

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|                          | Conducted genetics analysis (100%)  
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| Murphy, S. C.     | Conducted simulation testing (50%)  |
| Heath, A. C.      | Provided twin data (50%)  |
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| Zietsch, B. P.    | Designed experiment (50%)  
|                          | Wrote the paper (20%)  |
Contributions by others to the thesis

Brendan Zietsch and Bill von Hippel provided critical feedback on drafts of this thesis. Other than the contributions specified in regards to published papers included in this thesis, no others gave input to this thesis.

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None.
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List of abbreviations used in the thesis

A – Additive genetic variance
AIC – Akaike information criterion
BMI – Body mass index
C – Common environment
D – Non-additive genetic variance
DZ – Dizygotic
E – Residual variance/environment
H² – Broad sense heritability
h² – Narrow sense heritability
MZ – Monozygotic
rC – Common environment correlation
rE – Residual variance/environment correlation
rG – Genetic correlation
TDDS – Three-Domain Disgust Scale
TPQ – Tridimensional Personality Questionnaire
Section 1. General Introduction
1.0 Human Mating Strategy

Finding and attracting a mate is an enormous component of human life, and indeed the life of all sexually reproducing organisms. As individuals we spend hours, days, and even years attempting to find a suitable partner. Numerous industries have sprung up to assist in the process, from products designed to increase attractiveness such as perfume, make-up, and 24-hour gyms, to businesses that provide expertise and opportunity such as dating services, pick-up guides, and brothels (for the less successful). When this search yields results, the consequences are profound and reverberate across multiple aspects of human life including the social, economic, and genetic landscape. Within a romantic dyad, relationship quality is linked to both mental and physical health outcomes (Kiecolt-Glaser & Newton, 2001). Mate choice also has implications for the financial structure of society, creating economic stratification (Schwartz, 2013). Finally, as most children are born to couples (Anderson, 2006), mate choice and the mechanisms underlying mate choice will ultimately change the genetic landscape and define the future of our species (Lande, 1977; Wilson, 1973; Wright, 1921). Consequently, understanding the constants and variants of human mating strategy is of paramount importance at a multitude of levels: to increase individual prosperity, for an academic understanding of human nature, and to accurately predict the future of our society. Below, I set out a number of approaches to understanding variation in mating strategy, providing examples of how evolutionary psychology can be both beneficial and detrimental in this process, before highlighting the main shortcomings in the literature that this thesis will attempt to address. In particular, I introduce a central conflict in evolutionary psychology that revolves around variation in female preferences for sexual dimorphism. I highlight that there is little continuity between stated romantic preferences and eventual mate choice and finally, I introduce genetics as a potential cause of variation in both preferences and choice.

1.1 Investigating Human Mating Strategy

Human mate choice is an extraordinarily dynamic process influenced by biology, culture, and to a good degree mere circumstance. It is therefore unsurprising that many researchers across varied fields have sought to understand the key mechanisms underlying mate choice from various perspectives (Davidson & Moore, 2005; McGillicuddy-De Lisi & De Lisi, 2002). Nonetheless, the myriad processes underlying mating strategy including preferences, competition, and eventual choice in humans are largely opaque. One approach that has unique explanatory power is the use of evolutionary psychology principles. Given that mating strategy, and ultimately mate choice, is the mechanism by which evolution takes place (i.e., the differential reproduction of some genetic variants over others), applying evolutionary principles to patterns of mate preference, strategy, and choice may yield explanations not offered by other investigatory strategies.
1.2 Evolutionary Psychology of Mate choice

Organisms on Earth have been sexually reproducing for approximately 1.2 billion years (Butterfield, 2000). Along this journey, reproductive success stories have given rise to effective mating strategies – consistent patterns that influence mate choice across generations, ultimately shaping and indeed deriving entirely new species. Evolutionary psychology makes inferences about mate choice strategies based on pervasive adaptive problems that have appeared in a species’ evolutionary environment. To do this, evolutionary psychologists make observations regarding humans’ current mating strategies and develop hypotheses regarding the link between current behaviour and our ancestral conditions (Cosmides & Tooby, 1997). Evolution can only select for organisms that ensure their genetic material is passed on to subsequent generations. For example, in humans, infants require an enormous amount of investment, due to the relative dependence of offspring for the first decade of their life. Consequently, females should be driven to select for mates who will invest in offspring (via resource acquisition, paternal care, physical protection, etc.) increasing the chances of their survival. Males, conversely, should be driven to select mates with whom they can be assured that any mutually raised offspring are their own genetic relatives (as opposed to another male’s) (Trivers, 1972). From this, evolutionary psychologists might predict that males would be more distressed by, and should therefore be motivated to avoid, their partners engaging in sexual affairs which might result in mistakenly investing resources in another male’s offspring. In contrast, females may be more adversely affected by the prospect of a partner engaging in a romantic affair with another woman as, historically, any resources that were being contributed to rearing offspring might be diverted elsewhere. This hypothesis has largely held true (Buss, Larsen, Westen, & Semmelroth, 1992) and is relatively consistent across numerous cultures, suggesting near universality (Buss et al., 1999; Easton, Schipper, & Shackelford, 2007; Murphy, Vallacher, Shackelford, Bjorklund, & Yunger, 2006; Pietrzak, Laird, Stevens, & Thompson, 2002; Sagarin, Vaughn Becker, Guadagno, Nicastle, & Millevoi, 2003; Schützwohl, 2008; Vaughn Becker, Sagarin, Guadagno, Millevoi, & Nicastle, 2004, though see Harris, 2003).

1.3 The Darwinian Paradox of Individual Differences

There are a number of consistencies in mating strategy amongst humans that evolutionary psychology may convincingly explain. Yet, there also exists an extraordinary amount of variation in mate preferences, both within and between sexes. This variation presents a Darwinian paradox to researchers. Natural selection, via the successful reproduction of some genes over others, should typically reduce variation (though some special forms called balancing selection can maintain it), driving towards an evolved optimum. Nonetheless, humans vary on almost all quantitative traits (Houle, 1992), including those directly relating to reproductive outcomes (Hughes & Burleson,
This is strikingly exemplified by variation in female preference for sexually dimorphic traits in males.

Males and females differ from each other on a number of dimensions, both physiological and psychological, and this is reflected in mate preferences. Males tend to favour women with feminine faces: those with neotenous features including large eyes, narrow jaws, and full lips (Little, Jones, & DeBruine, 2011). Perplexingly, female preferences are not so consistent. Whereas some women prefer highly masculine males with large lower jaws, broad inner-faces, and high, robust foreheads, others prefer males with softer, more feminine features (Little, Jones, et al., 2011). Likewise, female preferences for facial hair, a sexually dimorphic trait, are highly variable (B. J. W. Dixson & Brooks, 2013; B. J. W. Dixson & Rantala, 2016; B. J. W. Dixson & Vasey, 2012; Feinman & Gill, 1977; Janif, Brooks, & Dixson, 2014; Muscarella & Cunningham, 1996; Pellegrini, 1973; Reed & Blunk, 1990; Wogalter & Hosie, 1991).

1.4 Understanding Causes of Individual Differences

Evolutionary psychologists have had limited success in accounting for this variation. Adaptation, and consequently, variation between species is generally caused by unique environmental challenges that require unique evolutionary solutions. When these strategies are successful over time, they can produce vast differences between previously similar organisms (i.e., speciation). Evolutionary psychologists frequently try to explain individual variation, and particularly variation in mating strategy, based on similar principles of environmental variation (Tooby & Cosmides, 2005). However, where evolutionary biologists typically focus on changes over many generations, evolutionary psychologists attempt to explain variation within one generation (Tooby & Cosmides, 1990). That is; what causes individuals to vary in mating strategy between ecologies and across the lifespan. A popular example of applying these principles to human mating can be found in studying women’s preferences for masculine versus feminine men.

1.5 Genetic Benefits and Parental Investment

At the centre of evolutionary explanations for this variation is a hypothesised trade-off between ‘good genes’ and ‘good dad’ characteristics (Folstad & Karter, 1992). Male facial physiognomy varies as a result of prenatal and pubertal testosterone exposure with increased testosterone corresponding to larger jawbones, thick brow ridges and prominent cheekbones (Whitehouse et al., 2015) or increased sexual dimorphism. As well as increasing facial masculinity, testosterone is also an immunosuppressant in humans (Hillgarth & Wingfield, 1997; Kanda, Tsuchida, & Tamaki, 1996; Yesilova et al., 2000). As such, high masculinity is thought to signal some underlying genetic quality because of the costs that are associated with testosterone that these males are able to sustain (Folstad & Karter, 1992). Facial masculinity and other testosterone-dependent traits, it is proposed, therefore represent a handicap and are costly signals that only
healthy individuals are capable of displaying (Zahavi, 1975). It may be advantageous for females to partner with these males for two reasons: firstly, the risk of infection during courtship is reduced and it is likely that healthy mates are better able to contribute resources and/or protection (Able, 1996; Borgia & Collis, 1989). Secondly, selecting for a disease resistant mate may confer genetic benefits to offspring that result from the coupling (Hamilton & Zuk, 1982) as immune system function in humans is substantially heritable (de Craen et al., 2005).

Testosterone is not uniquely associated with positive traits, however; highly masculine males are purported to possess traits that are negatively associated with paternal behaviour (Scott, Clark, Boothroyd, & Penton-Voak, 2013) and are typically perceived as being more dominant and aggressive (Boothroyd, Jones, Burt, & Perrett, 2007; DeBruine et al., 2006; Keating, Mazur, & Segall, 1981; Perrett et al., 1998; Swaddle & Reierison, 2002). With regard to romantic relationships, men in committed relationships are observed to have lower testosterone (Alvergne, Faurie, & Raymond, 2009; Muller, Marlowe, Bugumba, & Ellison, 2009; van Anders & Watson, 2006) whereas those with higher testosterone tend to have a higher number of sexual partners (Bogaert & Fisher, 1995; Peters, Simmons, & Rhodes, 2008; van Anders, Hamilton, & Watson, 2007). Moreover, testosterone is implicated in a range of anti-social behaviours including violence, and infidelity, as well as poor relationship outcomes such as divorce (Booth & Dabbs, 1993). In order to maximise the benefits associated with high testosterone men (i.e., genetic quality) and to minimise the costs that they may represent (e.g. infidelity or violence), women should theoretically prioritise masculine men when genetic benefits are more likely or when in an environment wherein immunocompetency is highly valued (Little, DeBruine, & Jones, 2011).

In accordance with this hypothesis, some studies have shown that female facial preferences vary predictably with environmental and reproductive conditions. More masculine faces are preferred in the context of short-term (i.e., predominantly sexual) relationships as compared to long-term relationships (Burt et al., 2007; Little, Jones, Penton-Voak, Burt, & Perrett, 2002; Penton-Voak et al., 1999; Waynforth, Delwadia, & Camm, 2005) and in response to pathogen cues (DeBruine, Jones, Crawford, Welling, & Little, 2010; Little, DeBruine, et al., 2011). Preferences in women have been observed to shift away from feminine male faces and towards more masculine faces closer to ovulation, when the chances of conception are the highest (Penton-Voak & Perrett, 2000; Penton-Voak et al., 1999). Furthermore, women’s preferences tend to shift away from masculine faces in response to cues of resource scarcity (Little, Cohen, Jones, & Belsky, 2007) and women who identify with lower socioeconomic status tend to prefer feminine faces (Lee et al., 2013).

The search for high genetic quality in romantic partners is also thought to explain variation in female sexual responsivity (Alcock, 1980; Thornhill, Gangestad, & Comer, 1995). In contrast to
males, female orgasm is inconsistent during penetrative intercourse (for review Lloyd, 2005). While substantial debate exists as to why this may be (Amundson, 2008; Barash, 2005; Barash & Lipton, 2009; Judson, 2005; Puts & Dawood, 2006; Puts, Welling, Burriss, & Dawood, 2012; Wallen, 2006; Wallen, Myers, & Lloyd, 2012; Zietsch & Santtila, 2012), some evolutionary psychologists argue that the wide variation in orgasm frequency during sex actually reflects a discriminatory function that responds to and selects for genetic quality in male sexual partners (Alcock, 1980; Baker & Bellis, 1993; Puts, Welling, et al., 2012; Smith, 1984; Thornhill et al., 1995). Genetically superior males, as signalled by facial masculinity and attractiveness (Gangestad & Scheyd, 2005), are thought to induce orgasm more frequently during sex and therefore are more likely to pass on their genes via repeated couplings. This is broadly referred to as the sire-choice hypothesis of female orgasm. In keeping with this theory, a number of studies have observed that orgasm rate is higher with more attractive and masculine partners (Andersson, 1994; Gallup Jr, Ampel, Wedberg, & Pogosjan, 2014; Grammer, Fink, Möller, & Thornhill, 2003; Shackelford et al., 2000), though it is unsurprising that women are more likely to orgasm with males they are more attracted to.

However, given the proposed trade-off between high testosterone and parenting traits, other evolutionary psychologists have posited that female orgasm may vary in response to cues of paternal investment (Barash, 1977; Beach, 1974; Eibl-Eibesfeldt, 1970; Hamburg, 1978; Morris, 1999). Under this model, known as the pair-bond hypothesis, female orgasm should respond to male traits that are likely to benefit offspring such as faithfulness, emotional warmth, and resource acquisition via traits such as earning potential (Buss & Barnes, 1986; Gallup Jr et al., 2014; Scheib, 2001). While this aspect of female orgasm variation has been investigated less than ‘good genes’ traits, some research suggests that ‘good dad’ traits may also contribute to female sexual responsiveness. Overall relationship quality, which is likely predicted by emotional investment and warmth, was found to correlate positively with greater orgasm frequency during penetrative sex (Costa & Brody, 2007), though the direction of causation in this study is difficult to establish. Additionally, Gallup Jr et al. (2014) found women reported greater orgasm frequency when their partner’s family income was higher, suggesting a role of resource provision potential. The case of the female orgasm is far from closed, however, and there a number of considerations to be taken into account.

1.6 Inconsistencies in the Evolutionary Psychology of Mating Strategy

A primary point of contention against the ‘good genes’/’good dad’ model of female preference variation is that few conclusive links have been observed between masculinity or attractiveness with genetic quality in humans (though see Markus et al., 2012). While some studies have found a relationship between masculinity and health indices, they are confounded by the use of subjective of measures of either health or masculinity, as masculine males may be prone to
underreport health issues and seek less medical help (for review see Scott et al., 2013). Further to this, a recent study found no relationship between a number of physiological health measures and mating success in males and females (Foo, Simmons, & Rhodes, 2017). Even if evolution previously favoured women who mated with healthier men, there is no current evidence for women favouring men who are actually in better health. Other research has failed to find any link between preferences for facial masculinity and preferences for perceived health (Boothroyd et al., 2005; Enlow, Moyers, & Merow, 1982). Meanwhile, current evidence regarding women’s preferences for masculine faces increasing closer to ovulation is equivocal, with two competing meta-analyses published in the same year reaching separate conclusions (Gildersleeve, Haselton, & Fales, 2014a, 2014b; Jones et al., 2017; W. Wood, Kressel, Joshi, & Louie, 2014).

Evidence regarding female orgasm variation in response to male traits is equally contentious. Most research investigating partner traits and female orgasm rely on women’s ratings of their partner (Andersson, 1994; Grammer et al., 2003) or their partner’s perceived attractiveness to others (Gallup Jr et al., 2014; Shackelford et al., 2000). Such designs introduce alternative explanations regarding causality as women may grow to view their partners more favourably if they experience more orgasms with them, which could cloud subjective reporting of attractiveness and masculinity. Other studies have avoided this limitation by having third parties rate women’s partners for attractiveness, masculinity, and dominance (Puts, Welling, et al., 2012; Thornhill et al., 1995). Using unbiased ratings of male characteristics, these studies found that the partners of women who orgasmed more often were more attractive, and Puts, Welling, et al. (2012) also found that partners who regularly elicited orgasms were rated to be more dominant and masculine.

However, even these results, and in fact any study utilising a between-subjects design, are subject to two major limitations. The first is that women are highly variable in orgasmability independent of partner (for review see Lloyd, 2005) and as a result there is limited power to cleanly detect between-partner orgasm frequency variation over and above between-woman variation. Secondly, any consistencies found across males with whom women orgasm easily may reflect the qualities of the women themselves rather than the men they are with. For instance, highly orgasmic women may tend to have sex with masculine men more often because both may be more oriented to casual sex relationships. Moreover, the sexual behaviour of males is often overlooked with regards to female orgasm, despite the obvious relevance to sexual pleasure. In sum, the evidence for female choice maintaining variation in sexually dimorphic traits in males is at best mixed.

More convincing is the evidence indicating that male facial masculinity has implications for intrasexual competition between males. Although women, on average, do not show a clear preference for masculine or feminine faces (Scott et al., 2013), facially masculine men are consistently rated as appearing more dominant and aggressive (DeBruine et al., 2006; Perrett et al.,
Masculine men are viewed as more intimidating and increasingly research indicates that intrasexual competition between men, rather than direct intersexual selection, has led to the evolution and maintenance of sexually dimorphic physiology (Archer, 2009; Puts, 2010, 2016; Puts, Bailey, & Reno, 2015).

1.7 Genetic Influences on Mating Strategy

As indicated above, evolutionary hypotheses regarding variation in mate preferences primarily focus on adaptive responses to environmental conditions and trait variation (e.g. preferences for facial masculinity in response to pathogen density, physical attractiveness, and/or short-term mating preferences). Neglected in this approach, is variance caused by genetic factors. Although humans share the majority of their genome in common with each other, there are many regions that differ from person to person (referred to as polymorphisms). The effects of these differences, although individually small (Chabris, Lee, Cesarini, Benjamin, & Laibson, 2015; Plomin & Deary, 2015), cumulatively create substantial variation, which extends to almost all human behaviour, even traits under strong selection. In fact, genetic differences account for, on average, half the variation in measured traits in humans (Polderman et al., 2015).

This is no different in the case of mate preferences (Zietsch, Verweij, & Burri, 2012). In fact, my colleagues and I have shown that approximately 40% of the variation in female’s preferences for facial masculinity in males is due to genetic effects (Zietsch, Lee, Sherlock, & Jern, 2015). Moreover, the cumulative variance in facial preferences accounted for by the same female’s sociosexuality (i.e., interest in short-term sexual relationships), pathogen disgust sensitivity (as a measure of perceived pathogen threat), conception risk, and self-rated attractiveness was less than 1% of the total variation. The same limitation can be levied at mate choice hypotheses regarding female orgasm variability. Up to 40% of the variation in orgasm frequency between women is attributable to genetic effects (Zietsch, Miller, Bailey, & Martin, 2011; Zietsch & Santtila, 2011). As such, detecting variance in orgasm frequency due to putative ‘good genes’ or ‘good dad’ traits over above variation between women is highly difficult.

1.8 The Relationship Between Mate Preference and Mate choice

A broader theoretical limitation of the evolutionary theories above is that they rely largely on self-reported preferences. It is often the case that preferences do not align with realised mate choice (for review see Eastwick, Luchies, Finkel, & Hunt, 2014), and realised mate choice and reproduction, rather than preferences, are the driving forces behind evolution. In order to better understand the causes of variation in mating strategy, it is therefore useful to study patterns of realised mate choice. Further, mate preferences may be reflected to some extent in mate-choice, however; they are likely diluted by chance and circumstance. In humans there is no trend more pervasive than assortative mating; that is the tendency for members of romantic dyads to be more
alike than would be expected by chance (Caspi, Herbener, & Ozer, 1992; Klohnen & Mendelsohn, 1998; Mascie-Taylor, 1989; Plomin, DeFries, & Roberts, 1977; Price & Vandenberg, 1980; Watson et al., 2004; Zietsch, Verweij, Heath, & Martin, 2011). Assortative mating occurs across multiple domains, but the strongest correlations occur between romantic partner’s age, religiosity, and social attitudes (Zietsch, Verweij, et al., 2011), followed by intelligence (Mascie-Taylor & Vandenberg, 1988), attractiveness (Feingold, 1988), and education (Zietsch, Verweij, et al., 2011). Despite the pervasiveness of assortative mating, the underlying mechanisms are poorly understood.
Section 2.0 Testing Evolutionary Hypotheses Regarding Individual Differences in Human Mating Strategy
From the evidence reviewed in section 1, the following limitations have been identified in regards to existing attempts to understand variation in mating strategy. Firstly, the underlying assumptions of the ‘good genes’/‘good dad’ dichotomy are yet to be convincingly established with reference to intersexual selection, and more parsimonious explanations regarding intrasexual selection have been under-studied. Secondly, hypotheses directly following from these assumptions are not consistently supported across a range of studies in a number of domains (e.g. facial preferences and female orgasm) and in the instances that they are supported alternative explanations cannot be ruled out. Thirdly, genes represent an important source of variation in almost all behavioural traits. Yet, they have been largely disregarded in evolutionary psychology literature, especially with regard to mate preference. Finally, very little focus is given to actual mate choice, as opposed to mate preference. In particular, the mechanisms behind the only consistent pattern of mating in humans, assortative mating, have to yet to be sufficiently explained. Consequently, the current thesis aims to test several hypotheses regarding variation in human mating strategy while addressing several limitations associated with the current literature.

In section 3, I present a paper testing evolutionary hypotheses regarding female variation in orgasm frequency in response to male characteristics. Where previous research has focused on women reporting on a single partner and observing male characteristics across the sample, this paper had females report on partners with whom orgasm was easy and with whom orgasm was difficult. This controls for the possibility of confounds relating to between-subject studies (as discussed in section 1.6) and increases our power to detect male traits associated with orgasm over and above between-women differences. Furthermore, the study also included measures of male sexual behaviour, as these are highly likely to be related to female orgasmability.

Section 4 of the thesis includes a paper demonstrating further evidence that facial dimorphism is an important factor in intrasexual competition. Specifically, the interaction between male facial masculinity and beardedness is investigated with regards to ratings of social dominance given by males. This relationship is also investigated in the context of implicit ratings. That is, faces of varying masculinity and hirsuteness are used as primes in a reaction time task in order to investigate whether sexually dimorphic facial characteristics are important for both ratings of explicit and implicit masculinity.

In section 5, twin studies are introduced as a method of estimating genetic causes of behavioural variation. The logic and history of the classic twin design will be discussed before an example is presented via a twin study of disgust sensitivity across three domains. Disgust sensitivity is an evolutionarily relevant behavioural domain that has links to human mating strategy (e.g. Al-Shawaf, Lewis, & Buss, 2014). Following this explanation, in section 6 I present a twin study of assortative mating. Using a large sample of Australian twins and their spouses, I use biometric
modelling to quantify the influence of genes and the environment on individuals pairing with similar partners across numerous traits including educational attainment, religiosity, income, personality, and physical characteristics. This is the first study to investigate the possibility of genetic influences on variation in assortative mating in humans.

In section 7, I synthesise the results of these papers and discuss the implications for current theory within the field of evolutionary psychology. In particular, I draw on the methodological strengths of the paradigms used in this thesis to make recommendations for future research. I integrate my findings with the best available literature and make concrete suggestions for ways in which evolutionary psychologists can use emerging approaches to untangle the Darwinian paradox of individual variation in mating strategy.
Section 3. The Evolution of Individual Differences in Female Orgasm


Abstract

The evolution of the female orgasm in humans and its role in romantic relationships is poorly understood. Whereas the male orgasm is inherently linked to reproduction, the female orgasm is not linked to obvious reproductive or survival benefits. It also occurs less consistently during penetrative sex than does the male orgasm. Mate choice hypotheses posit that the wide variation in female orgasm frequency reflects a discriminatory mechanism designed to select high-quality mates. We aimed to determine (1) whether women report that their orgasm frequency varies between partners, (2) whether this variation reflects mates’ personal characteristics, and (3) whether this variation reflects own and partner sexual behaviour during intercourse. We collected survey data from 103 women who rated (1) the extent to which their orgasm frequency varied between partners, (2) the characteristics of previous sexual partners who induced high-orgasm frequency and those who induced low-orgasm frequency, and (3) the specific behaviours during sex with those partners. This is the first study to test within-woman variation in orgasm and partner traits. Overall, women reported variation in their orgasm rates with different partners. Partners who induced high-orgasm rates were rated as more humorous, creative, warm, faithful, and better smelling than partners who induced low-orgasm rates, and also engaged in greater efforts to induce partner orgasm. Some assumptions and predictions of mate choice hypotheses of female orgasm were supported, whereas other aspects of our findings provide reasons to remain skeptical.

Keywords: evolution; sex; partner choice; mating; relationships

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3.1. Introduction

The evolutionary basis of the female orgasm in humans is poorly understood (Amundson, 2008; Barash, 2005; Barash & Lipton, 2009; Judson, 2005; Puts & Dawood, 2006; Puts, Dawood, & Welling, 2012; Zietsch & Santtila, 2012). Whereas the male orgasm is linked to ejaculation, the function (if any) of the female orgasm is unknown. Different women vary greatly in the ease with which they reach orgasm during sex, partly due to genetic differences (Zietsch, Miller, et al., 2011), and there are no clear fitness consequences of this variation (Zietsch & Santtila, 2013). However, some researchers have proposed that the female orgasm serves a discriminatory function in mate selection (Alcock, 1980; Puts, Dawood, et al., 2012; Smith, 1984; Thornhill et al., 1995). In this paper, we test some of the core assumptions and predictions of this type of hypothesis.

In essence, mate choice hypotheses are based on the proposition that due to the high gestational cost of pregnancy, as well as the ongoing cost of rearing children, it is important for women to reproduce with a mate of high quality - that is, one which will offer benefits to the woman and/or her offspring. The nature of those benefits distinguishes two versions of mate choice hypotheses: the sire-choice hypothesis, which proposes that female orgasm functions to help select mates who will provide genetic benefits to the offspring (Alcock, 1980; Baker & Bellis, 1993; Puts, Welling, et al., 2012; Smith, 1984; Thornhill et al., 1995); and the pair-bond hypothesis, under which the benefits relate to increased care and paternal investment (Barash, 1977; Beach, 1974; Eibl-Eibesfeldt, 1970; Hamburg, 1978; Morris, 1999).

Genetic benefits to offspring can, in theory, be any heritable traits with fitness benefits, but the focus has been on physical masculinity and attractiveness, which are commonly assumed to reflect genetic quality in men (Gangestad & Scheyd, 2005; though see Lee, Mitchem, et al., 2014). Several studies have found that orgasm rate is higher with more attractive partners (Andersson, 1994; Gallup Jr et al., 2014; Grammer et al., 2003; Shackelford et al., 2000). Most relied on women’s reports regarding her own partner, or women’s reports of how attractive her friends would find her partner (Gallup Jr et al., 2014; Shackelford et al., 2000), though Thornhill et al. (1995) and Puts, Welling, et al. (2012) found the same effect with independent ratings of partners. Puts, Welling, et al. (2012) also found that highly orgasmic women’s partners were rated as more dominant and masculine by online volunteers than were the partners of minimally orgasmic women.

Benefits conferred to offspring through paternal investment can include resource provision, physical protection, and infant care (Carter, 1992; Lloyd, 2005). Such benefits are more available when the father stays pair-bonded to the mother, so traits such as faithfulness and emotional warmth are often cited as important indicators of paternal investment, in addition to resource measures such as earning potential (Buss & Barnes, 1986; Gallup Jr et al., 2014; Scheib, 2001). Although the role of these traits in predicting orgasm has not been investigated thoroughly (though see Herberich,
Hothorn, Nettle, & Pollet, 2010; Pollet & Nettle, 2009), Costa and Brody (2007) found that overall relationship quality was associated with greater orgasm frequency during penetrative sex. However, neither Zietsch, Miller, et al. (2011) nor Thornhill et al. (1995) found a relationship between relationship commitment or length and orgasm frequency. In regard to resource provision, Gallup Jr et al. (2014) observed that partners’ family income was predictive of orgasm frequency.

Many traits, however, such as intelligence, could provide offspring benefits through paternal investment (e.g. via higher earning potential) as well as genetic inheritance, so we do not consider all partner traits as dividing neatly into ‘good genes’ or ‘good dad’ categories. Nevertheless, to be usefully distinct hypotheses, they must yield differential predictions regarding the kinds of traits that will be possessed by partners with whom female orgasm rate is higher versus lower. Only one study has tested the association of women’s orgasm frequency with a range of partner traits (Gallup Jr et al., 2014). The authors found that women’s orgasm frequency was predicted by partners’ attractiveness and family income. However, the study used a between-subjects design, which has two important limitations. First, power to detect associations between orgasm frequency and orgasm rates is reduced because the ‘noise’ of between-woman variation in orgasm frequency obscures the between-partner variation. Second, any associations that are found may be subject to confound - that is, highly orgasmic women may choose or attract or retain different partners than less orgasmic women. For example, the dating site OkCupid found among 42,398 site users that women who enjoy exercise have markedly greater ease of orgasm (Rudder, 2011) - such women, being fitter and healthier, may also tend to partner with more attractive men.

To address these limitations, the present study used a within-subjects design. First, we asked to what extent women actually experience variation in orgasm rate between different partners. We then compared, in women who had multiple ex-partners, the traits of the partner with whom they experienced orgasms at the highest rate (‘high-orgasm partner’) against the partner with whom they experienced orgasms at the lowest rate (‘low-orgasm partner’).

3.2 Method

Participants. In order to avoid bias in the evaluation of sexual partners, we recruited single women, as those in a relationship may feel obliged to rate their current partner more favorably than is strictly true. We therefore launched a screener survey to identify suitable participants to take part in the study. In order to qualify, participants were required to (1) be female, (2) not currently be in a relationship, (3) identify as heterosexual, and (4) have had more than two sexual partners in their lifetime. Initial screener surveys were launched on Amazon’s Mechanical Turk. Participants were offered US$0.05 to complete the screener survey, with the possibility of a larger payment if they qualified for the main survey. To avoid participants misrepresenting themselves in order to qualify for the survey, we did not advertise the selection criteria and instead excluded unsuitable
participants from further data collection. This screener survey was completed by 1,069 participants, of whom 123 qualified. Of these, 103 participants completed the full survey. Participants earned $US3.50 for completing the full study. Participant age ranged from 20 to 69 years ($M = 36.49$, $SD = 12.19$). Ninety-seven participants (93%) were from the United States, whereas six were from Australia, New Zealand, Great Britain, and Canada. The majority of participants were Caucasian (81.6%). On average, participants took 71 min to complete the survey (note that the items used in the present study constitute one component of a larger exploratory study of female sexuality and predictors of sexual and relationship satisfaction).

**Procedure.** Participants were informed that participation was voluntary and anonymous and that they could withdraw without penalty. Participants then answered questions relating to their ‘high-orgasm’ and ‘low-orgasm’ partners (counterbalanced). At the end of the survey, participants were asked if they had misrepresented any information. They were assured they would receive full payment even if they had. Fourteen women indicated that they were in a relationship at the time of the survey. These women’s data were included when analysing general sexual behaviour, but excluded when comparing high- and low-orgasm-inducing partners to prevent current partner bias. Participants were debriefed at the end of the study.

**Measures.** Participants were assessed on their general sexual preferences and behaviours as well as their sexual behaviour with a partner with whom they orgasmed easily and one with whom orgasm was difficult or absent. Additionally, participants answered a series of items regarding the characteristics of the partner and their relationship.

**Demographics.** Participants recorded basic demographic details such as their age, height, weight, and ethnicity. Relationship status and duration (where applicable) were assessed to detect partnered participants.

**General sexual behaviour.** To investigate variation in orgasm frequency, participants were first asked to report the frequency of self and partner clitoral stimulation duration during intercourse. Participants were then asked to indicate their general orgasm frequency (i.e., not with a particular partner) during sex without manual clitoral stimulation, sex with partner clitoral stimulation, and sex with self-stimulation of the clitoris. Finally, for each measure of orgasm frequency during intercourse, participants were asked to what extent their orgasm frequency changed depending on the partner. Frequency of orgasm on all items ranged from 1 (‘never’) to 6 (‘always’) whereas variation in orgasm ranged from 1 (‘always the same, doesn’t depend on who I’m with’) to 4 (‘very different depending on who I’m with’).

**Partner characteristics.** All participants then completed two identical partner characteristics sections concerning a high-orgasm partner and a low-orgasm partner (counterbalanced between participants). Participants were asked to think of a partner with whom they had orgasmed the most
easily during sex (high-orgasm) and a partner with whom they had the most difficulty orgasming during sex (low-orgasm). If participants reported no variation in their orgasm frequency, they were asked to describe their most recent and second most recent sexual partner instead of high and low. These data were later excluded from partner comparison analysis (see Results). Participants were then asked for the duration of the relationship and how long ago they last slept with this partner. This was done in order to control for the possibility that women might be biased to regard more recent (or earlier) partners more positively. Participants were then asked to rate this partner on the following traits ranging from 1 (‘much lower than average’) to 5 (‘much higher than average’).

Traits were selected based on previous claims of association with parental quality (i.e. faithfulness, warmth, earning potential, and kindness; Buss & Barnes, 1986; Pollet & Nettle, 2009; Scheib, 2001) or genetic quality. This includes physical attractiveness (Prokop & Fedor, 2011; Puts, Welling, et al., 2012), height (Prokop & Fedor, 2011), athleticism (Schulte-Hostedde, Eys, & Johnson, 2008), masculinity (Frederick & Haselton, 2007; Lassek & Gaulin, 2009), voice depth (Puts, 2005; Puts, Gaulin, & Verdolini, 2006), physical fitness (Schulte-Hostedde et al., 2008), humour (Simpson & Gangestad, 1992), creativity (Haselton & Miller, 2006; Miller, 2000), intelligence (Haselton & Miller, 2006; Miller, 2000), dominance (Simpson & Gangestad, 1992), and body odour pleasantness (Garver-Apgar, Gangestad, Thornhill, Miller, & Olp, 2006; Wedekind, Seebeck, Bettens, & Paepke, 1995). Women also reported partners’ facial hair (as a masculine trait that is easily changeable and therefore not likely to be an indicator of genetic quality), as well as their partners’ confidence, weight, penis length, and penis width. Participants were also asked to judge how attractive their friends found this partner on the same scale as other partner traits, based on findings from Sela, Weekes-Shackelford, Shackelford, and Pham (2015) and Gallup Jr et al. (2014).

**Partner sexual behaviour.** Participants then described the sexual behaviour of their high- and low-orgasm partners immediately after rating their characteristics. Participants were asked how frequently they discussed sexual positions with their partner (1 ‘never’ to 5 ‘very often’) and whether they had asked their partner to use specific positions to increase the likelihood of reaching orgasm (yes/no). The frequency of sexual activities that did not involve penetrative sex, specifically receiving oral sex, use of sex toys, and dirty talk was then assessed (1 ‘never’ to 5 ‘always’) given their possible relationship with orgasm rates. Participants were then asked to indicate the time taken in minutes (1) spent on foreplay with this partner, (2) for this partner to reach orgasm, and (3) for the participant to reach orgasm with this partner. As orgasm and clitoral stimulation can vary based on sexual position, a series of seven cartoon depictions of common sexual positions were presented and participants were asked to indicate the frequency of orgasm and self and partner manual clitoral stimulation (1 ‘never’ to 5 ‘always’) in each. The positions were face-to-face man above, face-to-
face woman above, face-to-face side position, face-to-face sitting position, rear-entry prone position, rear-entry kneeling position, and rear-entry sitting position.

**Relationship variables.** Last, participants were asked to indicate if the following statements were true or false for each of the partners they described. These included: ‘this is actually a current partner’, ‘the sex I described was non-consensual’, ‘I had sex with this partner fewer than five times’, and ‘this was an adulterous encounter’.

### 3.3 Results

**General sexual practices.** Women reported variation in orgasm during intercourse based on different sexual behaviours (see Table 3.3.1 for descriptive statistics). Orgasm frequency was significantly lower during intercourse without manual clitoral stimulation ($M = 2.75$, $SD = 1.31$) when compared to intercourse paired with self-stimulation of the clitoris ($M = 3.97$, $SD = 1.39$), $t(90) = -7.70$, $p < 0.001$, and partner stimulation of the clitoris ($M = 3.89$, $SD = 1.28$), $t(96) = -7.09$, $p < 0.001$. However, no significant differences were observed in orgasm frequency when comparing intercourse paired with self- and partner stimulation, $t(85) = 1.90$, $p = 0.061$.

<table>
<thead>
<tr>
<th>Question</th>
<th>N</th>
<th>Range</th>
<th>M</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>102</td>
<td>20-69</td>
<td>36.49</td>
<td>12.19</td>
</tr>
<tr>
<td>Frequency of self-stimulation during intercourse ($1 = Never, 6 = Always$)</td>
<td>102</td>
<td>1-6</td>
<td>2.97</td>
<td>1.14</td>
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<tr>
<td>Frequency of orgasm using self-stimulation during intercourse ($1 = Never, 6 = Always$)</td>
<td>91</td>
<td>1-6</td>
<td>3.97</td>
<td>1.39</td>
</tr>
<tr>
<td>Rate of change in orgasm using self-stimulation during intercourse ($1 = Always the same..., 4 = Very different....$)</td>
<td>91</td>
<td>1-4</td>
<td>2.64</td>
<td>0.91</td>
</tr>
<tr>
<td>Frequency of partner-stimulation during intercourse ($1 = Never, 6 = Always$)</td>
<td>102</td>
<td>1-6</td>
<td>3.38</td>
<td>1.09</td>
</tr>
<tr>
<td>Frequency of orgasm using partner-stimulation during intercourse ($1 = Never, 6 = Always$)</td>
<td>97</td>
<td>1-6</td>
<td>3.89</td>
<td>1.28</td>
</tr>
<tr>
<td>Rate of change in orgasm using partner-stimulation during intercourse ($1 = Always the same..., 4 = Very different....$)</td>
<td>96</td>
<td>1-4</td>
<td>2.80</td>
<td>0.89</td>
</tr>
<tr>
<td>Frequency of orgasm during intercourse without manual stimulation ($1 = Never, 6 = Always$)</td>
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<td>1-6</td>
<td>2.79</td>
<td>1.31</td>
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<tr>
<td>Rate of change in orgasm during intercourse without stimulation ($1 = Always the same..., 4 = Very different....$)</td>
<td>103</td>
<td>1-4</td>
<td>2.41</td>
<td>1.05</td>
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<tr>
<td>Orgasm frequency during vaginal only masturbation ($1 = Never, 6 = Always$)</td>
<td>48</td>
<td>1-6</td>
<td>3.63</td>
<td>1.41</td>
</tr>
<tr>
<td>Orgasm frequency during both vaginal and clitoral masturbation ($1 = Never, 6 = Always$)</td>
<td>99</td>
<td>1-4</td>
<td>2.18</td>
<td>0.84</td>
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</tbody>
</table>

Women reported wide variation in their orgasm frequency during intercourse (Figure 3.3.1). Variation in orgasm also differed based on the type of sexual intercourse. Women reported that their orgasms varied more between partners when self-stimulating of the clitoris during intercourse ($M = 2.64$, $SD = 0.91$) and with partner stimulation of the clitoris ($M = 2.80$, $SD = 0.89$), when compared to sex without manual clitoral stimulation ($M = 2.38$, $SD = 1.05$), $t(90) = 2.06$, $p = 0.042$ and $t(95) = 2.96$, $p = 0.004$. However, females reported greater variability in their orgasms between
partners when their partner was stimulating their clitoris compared to their own stimulation, $t(84) = 2.86, p = 0.005$.

Figure 3.3.1. Orgasm variability during intercourse (0 = always the same, doesn’t depend on whom I’m with, 2 = only slightly different depending on whom I’m with, 3 = quite different depending on whom I’m with, 4 = very different depending on who I’m with).

**Sexual partner characteristics.** Six women indicated that one of their partners was non-consensual, and 18 (17%) could not distinguish between a partner with whom they orgasmed easily or one with whom they had difficulty achieving orgasm. After excluding these data points, the final sample contained information regarding the high- and low-orgasm partners of 71 women. If a participant-reported time to orgasm with a partner was greater than 120 min (1: 1.4%), it was assumed that she did not orgasm with this partner and this response was removed. As a manipulation check, we then compared reported orgasm frequency between high- and low-orgasm males across all sexual positions. As expected, participants were more likely to orgasm in every position with a high-orgasm male ($p < 0.002$). We subsequently averaged orgasm frequency over all sexual positions and compared this between high and low partners. Again, orgasms were more frequent for high-orgasm partners on average, $t(69) = 7.591, p < 0.001$. Further comparisons of high and low partners were separated into partner characteristics (Table 3.3.2) and sexual behaviour with these partners (Table 3.3.3).

**Partner traits.** Partner humour, voice depth and facial hair length were substantially skewed and as such we first conducted non-parametric tests comparing high and low partners on these traits. Wilcoxon signed-rank tests indicated that high-orgasm partners were rated significantly higher on humour than low-orgasm males ($Z = 3.16, p < 0.002$), but no differences were observed in vocal depth ($Z = 1.43, p < 0.154$), or facial hair ($Z = 0.01, p = 0.990$). As these results did not
differ from parametric tests, we report parametric results in Table 3.3.2 for ease of interpretation. Following Bonferroni corrections for multiple comparisons (34 in total), paired t-tests indicated that a number of traits and behaviours differed between high- and low-orgasm partners at \( p \leq 0.001 \).

With this criterion, humour, attractiveness, creativity, emotional warmth, faithfulness, and body odour pleasantness were all significantly greater in high-orgasm partners (see Table 3.3.2).

Table 3.3.2 Comparing most orgasmic and least orgasmic partners on personal characteristics

<table>
<thead>
<tr>
<th>Trait</th>
<th>High-orgasm Partner M(SD)</th>
<th>Low-orgasm Partner M(SD)</th>
<th>( t )</th>
<th>( df )</th>
<th>( p )</th>
<th>( d )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Humour</td>
<td>3.72 (.91)</td>
<td>3.28 (.96)</td>
<td>3.497</td>
<td>70</td>
<td>.001*</td>
<td>0.42</td>
</tr>
<tr>
<td>Intelligence</td>
<td>3.55 (.97)</td>
<td>3.27 (.93)</td>
<td>1.944</td>
<td>70</td>
<td>.056</td>
<td>0.23</td>
</tr>
<tr>
<td>Dominance</td>
<td>3.10 (.90)</td>
<td>2.96 (1.03)</td>
<td>0.89</td>
<td>69</td>
<td>.377</td>
<td>0.10</td>
</tr>
<tr>
<td>Attractiveness</td>
<td>3.59 (.87)</td>
<td>3.04 (.84)</td>
<td>4.186</td>
<td>70</td>
<td>&lt;.001*</td>
<td>0.50</td>
</tr>
<tr>
<td>Friends find partner attractive</td>
<td>3.27 (.93)</td>
<td>3.00 (.85)</td>
<td>2.036</td>
<td>69</td>
<td>.046</td>
<td>0.24</td>
</tr>
<tr>
<td>Athleticism</td>
<td>2.91 (.99)</td>
<td>2.71 (97)</td>
<td>1.342</td>
<td>69</td>
<td>.184</td>
<td>0.16</td>
</tr>
<tr>
<td>Creativity</td>
<td>3.62 (1.03)</td>
<td>2.63 (.87)</td>
<td>7.63</td>
<td>70</td>
<td>&lt;.001*</td>
<td>0.92</td>
</tr>
<tr>
<td>Muscularity</td>
<td>3.11 (.96)</td>
<td>2.79 (.81)</td>
<td>2.266</td>
<td>70</td>
<td>.027</td>
<td>0.27</td>
</tr>
<tr>
<td>Voice Depth</td>
<td>3.11 (.69)</td>
<td>2.96 (.66)</td>
<td>1.496</td>
<td>70</td>
<td>.139</td>
<td>0.17</td>
</tr>
<tr>
<td>Fitness</td>
<td>3.08 (.94)</td>
<td>2.82 (.88)</td>
<td>1.907</td>
<td>70</td>
<td>.061</td>
<td>0.22</td>
</tr>
<tr>
<td>Emotional Warmth</td>
<td>3.40 (1.04)</td>
<td>2.77 (1.02)</td>
<td>3.797</td>
<td>69</td>
<td>&lt;.001*</td>
<td>0.46</td>
</tr>
<tr>
<td>Faithfulness</td>
<td>3.37 (1.23)</td>
<td>2.65 (1.17)</td>
<td>3.771</td>
<td>70</td>
<td>&lt;.001*</td>
<td>0.45</td>
</tr>
<tr>
<td>Earning Potential</td>
<td>3.23 (1.06)</td>
<td>2.85 (1.12)</td>
<td>2.4</td>
<td>70</td>
<td>.019</td>
<td>0.26</td>
</tr>
<tr>
<td>Height</td>
<td>3.51 (.86)</td>
<td>3.29 (.76)</td>
<td>1.635</td>
<td>69</td>
<td>.107</td>
<td>0.19</td>
</tr>
<tr>
<td>Weight</td>
<td>3.06 (.77)</td>
<td>3.03 (.70)</td>
<td>0.27</td>
<td>70</td>
<td>.788</td>
<td>0.03</td>
</tr>
<tr>
<td>Penis Length</td>
<td>3.34 (.81)</td>
<td>2.97 (.94)</td>
<td>2.714</td>
<td>70</td>
<td>.008</td>
<td>0.33</td>
</tr>
<tr>
<td>Penis Width</td>
<td>3.39 (.80)</td>
<td>2.97 (.89)</td>
<td>3.019</td>
<td>70</td>
<td>.004</td>
<td>0.36</td>
</tr>
<tr>
<td>Confidence</td>
<td>3.66 (.86)</td>
<td>3.20 (1.02)</td>
<td>3.158</td>
<td>70</td>
<td>.002</td>
<td>0.37</td>
</tr>
<tr>
<td>Kindness</td>
<td>3.48 (1.03)</td>
<td>2.96 (.87)</td>
<td>3.303</td>
<td>70</td>
<td>.002</td>
<td>0.39</td>
</tr>
<tr>
<td>Body Odour</td>
<td>3.66 (.93)</td>
<td>3.20 (.94)</td>
<td>3.626</td>
<td>70</td>
<td>.001*</td>
<td>0.42</td>
</tr>
<tr>
<td>Pleasantness</td>
<td>1.89 (1.13)</td>
<td>1.87 (1.01)</td>
<td>0.083</td>
<td>70</td>
<td>.934</td>
<td>0.01</td>
</tr>
</tbody>
</table>

*Indicates significance at Bonferroni corrected \( p \leq .001 \).

**Partner sexual behaviours.** Length of relationship, length of time since last sexual encounter, and age at last sexual encounter were all substantially skewed. As a result, non-parametric tests were used for preliminary analysis. No significant differences emerged between length of relationship with high- and low-orgasm partners (\( Z = 1.46, p = 0.145 \)), length of time since the last sexual encounter (\( Z = 0.56, p = 0.576 \)), nor age during the last sexual encounter (\( Z = 0.60, p = 0.548 \)). These results were no different from those observed using paired t-tests, which we report for ease of interpretation (Table 3.3.3). A McNemar’s paired samples test of dichotomous outcomes indicated that females were no more likely to have had sex with high- or low-orgasm partners fewer than five times, \( p = 0.388 \), nor were they more likely to have had an affair with either partner, \( p = 0.999 \).
A number of sexual behaviours differed significantly between high- and low-orgasmic partners (see Table 3.3.3). Females in the sample communicated with high-orgasm partners about sexual positions more frequently and also received oral sex from high-orgasm partners more frequently. High-orgasm males were also reported as having a greater focus on female pleasure. In addition, high-orgasm partners were more likely to use sex toys and spend more time on foreplay. Averaged over all sexual positions, high-orgasm males were also more likely to manually stimulate the clitoris. Similarly, females were more likely to stimulate their own clitoris with high-orgasm males.

### 3.4 Discussion

We collected data from sexually active females regarding their general sexual activity. We also collected their reports regarding a partner with whom orgasm came the most easily and one with whom orgasms were the most difficult to achieve. Our aim was to determine whether women were conscious of orgasm variation with different partners, whether this variation was related to mate characteristics, and whether this variation was related to different sexual practices. First, women reported (on average) variation in personal orgasm frequency between partners. Variation in orgasm frequency was also significantly greater when manual clitoral stimulation occurred. Females reported that their orgasm frequency varied more between partners when partners stimulated their clitoris, compared to when they stimulated themselves. This suggests that orgasm variation is contingent upon sexual skill in addition to mate characteristics. This group of findings
represent the first attempt to determine if women report orgasming more or less with different partners. Our results show that they do, and thus allowed us to be confident when moving on to between-partner traits and behaviours that may account for this difference.

After comparing the characteristics of high- and low-orgasm partners, a number of differences emerged in both individual characteristics and sexual behaviour. On average, high-orgasm male partners were more humorous, attractive, creative, emotionally warm, faithful and had more pleasant body odour than low-orgasm partners. These findings are somewhat consistent with previous research and with aspects of the mate choice hypothesis. High-orgasm males were higher in characteristics associated with parental investment. Emotional warmth and faithfulness are both factors that may contribute to pair bonding and may encourage a greater number of copulations within pairings as well as investment in any subsequent offspring (Buss & Barnes, 1986; Pollet & Nettle, 2009; Scheib, 2001). These traits have not previously been identified in high-orgasm partners, including research by Costa and Brody (2007) who observed that commitment, a similar relationship component, was unrelated to sexual satisfaction in a small sample of women in relationships.

High-orgasm partners were also, on average, higher in a number of characteristics that may be construed as indicative of both genetic benefits and parental investment. Both attractiveness (Mitchem et al., 2014) and creativity (e.g. musicality; Mosing et al., 2014; Trainor, Honing, Peretz, Gingras, & Fisher, 2015) are heritable traits and may confer genetic benefits to offspring (e.g. Miller, 2000).

In agreement with Gallup Jr et al. (2014), we also observed that males with whom orgasms were more frequent were rated as more humorous than low-orgasm partners. In Gallup Jr et al. (2014), ratings of partners humour were also associated with higher ratings of creativity and intelligence and indeed high-orgasm males in the present study were also considered to be more creative than low-orgasm males. It has been proposed that humour and creativity may act as an honest signal of intelligence Miller (2000) and therefore an indicator of genetic quality. Intelligence can be more difficult to gauge than humour and creativity, with only small to moderate correlations between perceived and actual intelligence scores (Borkenau, Mauer, Riemann, Spinath, & Angleitner, 2004). Meanwhile, humour and creativity are more naturalistically demonstrated in dynamic, social contexts and as such may offer a more reliable indication of intelligence.

Body odour pleasantness has also been implicated in mate choice previously and may be associated with genetic benefits. After rating the pleasantness of body odour from shirts worn by males in the previous 48 hours, it was found that females preferred the shirts of males with dissimilar immune system alleles (Wedekind et al., 1995). Not only are key components of immune function heritable (de Craen et al., 2005), but women also report lower sexual satisfaction with
partners who share the same immune system alleles (Garver-Apgar et al., 2006). It has been hypothesised that selecting mates with dissimilar immune system alleles may confer benefits to offspring as they may inherit a wider range of antibodies (for review see Havlicek & Roberts, 2009).

Although the above findings may represent partial support for aspects of mate choice hypotheses, some aspects of the findings conflict with the sire-choice hypothesis. While we found attractiveness (note that this may or may not have been interpreted by participants to refer to physical attractiveness) was rated higher in high-orgasm partners, we found no significant difference, after correcting for multiple comparisons, between high- and low-orgasm partners in terms of putative ‘good genes’ traits such as intelligence, athleticism, and fitness, nor sexually dimorphic traits such as height, dominance, muscularity, and voice depth. The pattern found in our results suggests, conversely, that women’s orgasms depended more on traits potentially representing investment and attentiveness (e.g. faithfulness, emotional warmth) than classic markers of ‘good genes’ and masculinity. Potentially more important, as will be discussed below, were what these attractive, creative, warm, and faithful partners were reported to have done in bed.

Sexual behaviour varied considerably between high-and low-orgasm partners. During intercourse, high-orgasm males were more likely to focus on female pleasure, communicate about sexual positions, use sex toys, and perform oral sex on the females in this sample. On average across all sexual positions, high-orgasm partners were also more likely to stimulate their partner’s clitoris and women appeared to be more comfortable stimulating their own clitoris with high-orgasm partners (potentially as a result of communication and high-orgasm partner’s focus on their sexual pleasure). The only two domains in which behaviour did not differ between high- and low-orgasm partners were the use of dirty talk, and length of time the male partner took to reach orgasm.

The study was not without limitations. For example, it is difficult to discern the relative independent contribution of partner characteristics to the female orgasm. For instance, greater levels of humour and creativity may simply contribute to ratings of attractiveness, which in turn increases the likelihood of orgasm. Moreover, high-orgasm partners engaged in behaviours that were more likely to elicit an orgasm than their low-orgasm counterparts, and it is unclear how this relates to the personal characteristics of those partners. We find that women report greater orgasm variation between partners when their clitoris is manually stimulated compared to when it is not. This finding hints at the fact that some men might be particularly adept at inducing orgasm during penetrative sex via clitoral stimulation relative to others. Further, it seems that women themselves are effective at inducing orgasm via self-stimulation of the clitoris more so with some partners than others. It should be noted that clitoral stimulation appears to be very important when considering between-partner ease of orgasm in general, given that high-orgasm males were more likely to stimulate the
clitoris than low-orgasm males. A future investigation of variation in orgasm frequency between high- and low-orgasm males may consider examining sex with and without clitoral stimulation separately to establish whether there are unique factors that predict female orgasmability in the absence of direct exterior clitoral stimulation.

Studies of female orgasm requiring self-report of sexual behaviour and partner characteristics can also be biased by ‘halo’ effects; either orgasm frequency may cause women to overestimate their partner’s other positive qualities or conversely, their partner’s positive qualities may cause them to overestimate their orgasm frequency. However, we do not believe this presents a substantial issue in the present study, as many socially desirable traits were not found to significantly differ between high- and low-orgasm males. For instance, intelligence is a highly socially desirable trait that is associated with numerous beneficial outcomes. Likewise, height and earning potential are substantially important in female attraction yet neither differed significantly between high- and low-orgasm partners.

Future research should also aim to replicate our findings, and compare the unique predictive strength of each characteristic. Furthermore, meaningful mediational paths might emerge. For example, emotionally warm men might focus more on women’s pleasure, stimulating their clitoris more, and thus eliciting more orgasms.

Overall, however, our findings give some support to the idea that the female orgasm helps to choose partners who are likely to be good fathers, but suggest scepticism is warranted regarding the sire-choice hypothesis, which emphasises genetic benefits to offspring. It also raises the previously ignored complication that variation in specific sexual behaviours plays at least as much of a role as do the partners’ personal characteristics in how effectively he induces orgasms.
Section 4. The Role of Facial Dimorphism in Introsexual Competition

Abstract

Sexual dimorphism in facial shape and beardedness are salient human secondary sexual traits that enhance perceptions of men’s social dominance. The majority of this evidence, however, comes from studies measuring explicit ratings. To our knowledge, few studies have tested whether facial masculinity and beardedness are implicitly associated with dominance. In the current study, we use a within-subjects design to test whether facial masculinity and beardedness drive implicit reactions and overt ratings of male dominance. Participants viewed stimuli depicting the same men when clean-shaven, with heavy stubble, and fully bearded that were morphed to be either more masculine or less masculine using computer graphic software. Participants completed an affective priming word categorisation task as well as explicit ratings of social dominance. No facilitation effects were observed for masculinised or bearded faces on implicit judgements relating to dominance. In contrast, results revealed that masculinized versions of clean-shaven, stubbled and fully bearded faces received higher explicit dominance ratings than feminized versions. However, the effects of facial masculinity were largest within clean-shaven stimuli and decreased as faces became more hirsute, suggesting that facial masculinity had diminishing returns on dominance ratings. Our results support a role for masculine facial shape and bearded-ness in explicit, but not implicit, judgments of dominance among men.

Keywords: Masculinity, Dominance, Facial masculinity, Facial hair, Affective prime

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4.1 Introduction

Human visual systems have evolved to rapidly process identity, sex, age, and emotional expression from faces (Little, Jones, et al., 2011). Although dynamic facial expressions drive many overt behavioural interactions (Blair, 2003), static facial features also provide important information that underpin aspects of mate choice and the assessment of potential rivals (Little, Jones, et al., 2011). Mate choice and same sex competition imposes sexual selection on the evolution of ornamentation and weaponry in males across a wide range of taxa (Kokko, Brooks, Jennions, & Morley, 2003) and may have shaped the evolution of sexual dimorphism in body composition (Wells, 2007), cutaneous characters (van den Berghe & Frost, 1986), vocal pitch (Puts, 2010), facial and body hair (B. J. W. Dixson & Rantala, 2016), and aspects of facial structure (Whitehouse et al., 2015) in humans.

Considerable research has focussed on whether morphological differences between men and women have been shaped by mate choice (Gangestad & Scheyd, 2005; Grammer et al., 2003) or same sex competition (Puts, 2010, 2016). Oestrogen dependent traits in women such as breast morphology (B. J. W. Dixson, Duncan, & Dixson, 2015; B. J. W. Dixson et al., 2011), gynoid fat distribution (Brooks, Shelly, Jordan, & Dixson, 2015; Singh, Dixson, Jessop, Morgan, & Dixson, 2010), lighter skin complexion (Law Smith et al., 2006) and feminine facial shape (Marcinkowska et al., 2014) provide cues to health and fertility that tend to enhance attractiveness to men. However, women’s preferences for androgen dependent masculine facial features in men are more mixed (Rhodes, 2006; Scott et al., 2013) and in some cases masculinity reduces male facial attractiveness (Perrett et al., 1998). Likewise, beardedness enhances men’s attractiveness to women in some studies (B. J. W. Dixson & Rantala, 2016; Janif et al., 2014; Pellegrini, 1973; Reed & Blunk, 1990) but not in others (B. J. W. Dixson, Tam, & Awasthy, 2013; B. J. W. Dixson & Vasey, 2012; Feinman & Gill, 1977; Muscarella & Cunningham, 1996; Wogalter & Hosie, 1991), while in other cases preferences between clean-shaven faces and those with full beards are more equivocal (B. J. W. Dixson & Brooks, 2013; Neave & Shields, 2008; Saxton, Mackey, McCarty, & Neave, 2016). Even within traits for which women typically state strong preferences, such as deeper vocal pitch (Puts et al., 2006) and muscularity (B. J. W. Dixson, Grimshaw, Ormsby, & Dixson, 2014; Frederick & Haselton, 2007), effect sizes for ratings of dominance tend to be greater than those for attractiveness (Puts, 2010). Variation among women in their preferences may be context-dependent (Scott et al., 2013; but see Zietsch, Lee, et al., 2015), becoming stronger when considering mates for short-term rather than long-term relationships and when the likelihood of conception is greater (Gildersleeve et al., 2014a; but see W. Wood et al., 2014).

Across diverse taxa, weaponry, such as claws, horns and canines can serve directly in intra-sexual contest competition (Emlen, 2008). Among the anthropoid primates, visually conspicuous secondary sexual traits provide information used by males to assess the sexual maturity, dominance
Growing evidence supports the view that intra-sexual competition has played an important role in the evolution of men's secondary sexual traits and agonistic behaviours (Archer, 2009; Puts, 2010, 2016; Puts et al., 2015). Competition among males ancestrally, when female choice for mates may have been more limited than in contemporary industrialised societies, may have shaped the evolution of many of men's secondary sexual traits (Puts et al., 2015). Cues of formidability may serve to curtail aggressive and costly fights, aid in mate guarding and ultimately translate into greater mating and reproductive success (Puts et al., 2015).

A large body of research reports that explicit judgments of men's dominance and aggressiveness are enhanced by craniofacial masculinity (DeBruine et al., 2006; Perrett et al., 1998; Spisak et al., 2012) and beardedness (B. J. W. Dixson & Brooks, 2013; B. J. W. Dixson & Vasey, 2012; Geniole, Denson, Dixson, Carré, & McCormick, 2015; Neave & Shields, 2008; Saxton et al., 2016). Further, facial masculinity is associated with measures of men's upper body strength (Fink, Neave, & Seydel, 2007; Holzleitner & Perrett, 2015; Sell et al., 2009; Windhager, Schaefer, & Fink, 2011) and behavioural dominance (Geniole et al., 2015; Pound, Penton-Voak, & Surridge, 2009). Similarly, men with beards report feeling more masculine and dominant than when clean-shaven (D. R. Wood, 1986), have higher serum levels of testosterone (Knussman & Christiansen, 1988), and endorse more stereotypical masculine gender roles in heterosexual relationships (Oldmeadow & Dixson, 2015). Men with higher self-reported social dominance and men with greater stature are also less sensitive to cues of facial dominance than men of shorter stature and lower self-reported social dominance (Watkins, Fraccaro, et al., 2010; Watkins, Jones, & Debruine, 2010). Recently, it was shown that men who received a dose of exogenous testosterone were more likely to pick a more masculine version of their face than men who received a placebo (Welling, Moreau, Bird, Hansen, & Carré, 2016). Taken together, these findings suggest that androgen dependent facial shape and beardedness are used by other males to assess age, sexual maturity, rank and dominance, supporting the view that male secondary sexual traits function in intra-sexual communications (Puts, 2010, 2016; Puts et al., 2015).

Although studies measuring explicit ratings of dominance reveal that facial masculinity and beardedness are associated with masculinity and dominance, the extent to which such judgments extend to implicit associations of male dominance remains to be determined. Given that facial features may reveal the potential threat an individual poses (Geniole et al., 2015), it would be advantageous to assess these cues in as little time as possible. For example, brain imaging has shown that the amygdala plays a role in the automatic coding of facial characteristics associated with trustworthiness (Engell, Haxby, & Todorov, 2007). Additionally, at short viewing times, participants demonstrate significantly greater cueing effects for gaze cues (i.e. following gaze) from
masculinized male faces when compared to feminized faces (Jones, Debruine, et al., 2010; though see Jones, Main, Debruine, Little, & Welling, 2010). Moreover, these effects reduced as viewing time increased. Thus, facial cues may elicit implicit, as well as explicit, appraisals of many sociosexual attributes, including dominance.

In the current study we assess possible implicit attitudes relating to perceived dominance using an affective prime task. Where explicit ratings may be subject to demand characteristics (Fazio, Jackson, Dunton, & Williams, 1995), implicit testing paradigms are thought to tap automatic and unconsciously activated attitudes by using tasks that are not transparent to participants and do not require verbalisation. Affective primes test the strength of implicit attitudes by presenting participants with a prime stimulus before quickly displaying a target word that must be categorised by the participant. In principle, if the prime stimulus evokes an attitude that is congruent with the target word, participants will respond faster on average. This congruency effect has been replicated across numerous studies (for review see Bargh, 1997; Fazio, 2001; Klauer & Musch, 2003). Affective primes have been successfully used in order to evaluate implicit attitudes associated with facial stimuli (e.g. Banse, 2001; Koranyi, Gast, & Rothermund, 2013; T. T. Li & Lu, 2014; Palermo & Schmalzl, 2006; Yang, Cao, Xu, & Chen, 2012). Here we use as priming stimuli male facial images varying in natural levels of facial hair that were experimentally manipulated to enhance or suppress masculine shape cues via computer graphic software. These stimuli were paired with target words that were either related to dominance or submissiveness selected from previous literature assessing explicit ratings of facial dominance.

We predicted that facial hair and facial masculinity would have positive main effects on explicit ratings and implicit associations, so that full beards and more masculine facial shape would be more rapidly associated with dominance and rated as looking the most dominant. We also predicted that beards and facial shape would act in concert to determine our outcome measures, with full beards with masculine facial shapes being most rapidly associated with dominance and rated as looking the most dominant.

4.2 Method

Participants. Sixty males ($M = 20.12$, $SD = 3.32$) were recruited to take part in the study. All participants were undergraduates who were fluent in English and living in Australia, 50 of whom received course credit for participating in the experiment and the remaining ten participants volunteered without course credit. 63.3 % of participants self-identified as Australian, 13.3 % were Chinese, 3.3 % were Taiwanese, 3.3 % were British, 3.3 % were Central/South American, 1.7 % were North American, 5 % were Asian, 1.7 % were Sri Lankan, 1.7 % Swedish, 1.7 % were Turkish and 1.7 % elected not to answer this question. The majority (86.7 %) were heterosexual, 5 % were bisexual, 6.7 % were homosexual and 1.6 % elected not to answer this question. Although all 60
participants completed both the explicit and implicit tasks, one participant’s data from the implicit responses was corrupted, leaving 59 participants ($M = 19.97, SD = 3.13$) for that analysis.

**Face Stimuli.** Image Set: Six men (mean age ± $SD = 23.95 ± 3.43$ years, range 20–31) of European descent were photographed when clean-shaven, with 10 days of regrowth (heavy stubble) and with at least four weeks of untrimmed growth (full beard), posing front-on with neutral facial expressions. These six identities were randomly drawn from a larger image set of 36 individuals (Janif et al., 2014) and served as stimuli in the present study.

**Masculinity Manipulation.** Facial masculinity was manipulated via JPsychomorph software (Tiddeman, Burt, & Perrett, 2001). A sexual dimorphism continuum was defined as 50th percentile of vector difference between an average male and an average female face, created by averaging 50 Caucasian male and 50 female face images, respectively, not including the stimulus identities of the current study. The average male and female faces were matched for overall colour content using the Match Colour tool in Photoshop (vCS5.1). This ensured that morphs created using this continuum would not differ in overall hue from their original image, but permitted variation of local colour cues that likely contribute to perceived facial structure.

For each stimulus identity, the three variants (clean-shaven, heavy stubble and full beard) were each then morphed (using JPsychomorph) to create two images in which masculinity was increased by 50% (by morphing parallel to the male-female vector, in the direction of the average male face) and decreased by 50% (by morphing parallel to the male-female vector, in the direction of the average female face), respectively. Six stimulus identities that had been masculinized and feminized, respectively, at three stages of facial hair resulted in a 36-image stimulus set for the current study. These images were then refined in Photoshop to ensure each had sharp edges at the sides of the neck, smooth pupils (by replacing irises in the morphs with irises from the original image) and were presented on a consistent background colour. Removal of artefacts around the neck and eyes ensured the morphs looked as much like un-manipulated photographs as the original images. Each image measured $1458 \times 2292$ pixels and was presented in grayscale (Figure 4.2.1).
Figure 4.2.1. An example of the stimuli used in this study. Images show the same individual in each of the three categories of facial hair (clean-shaven, stubble and fully bearded) manipulated to appear 50% less masculine (top panel) and 50% more masculine (bottom panel).

**Affective Prime Target Words.** All participants completed an affective priming procedure (Fazio, Sanbonmatsu, Powell, & Kardes, 1986). The affective prime portion of the study employed a three: facial hair (clean shaven, stubble, full beard) by two: facial dimorphism (masculine, feminine) by two: target word category (dominant, submissive) design. Five words relating to dominance (menace, threat, fight, violent, strong) and submissiveness (meek, timid, weak, gentle, soft) were used as target words in the affective prime. These words were sourced from terminology previously employed in literature investigating explicit perceptions of male dominance and submissiveness (Hundhammer & Mussweiler, 2012; Sanchez, Kiefer, & Ybarra, 2006; Skowronski, Sedikides, Heider, Wood, & Scherer, 2010).

**Procedure.** Prior to beginning the experiment, participants were told that they were taking part in a reaction time task. Each participant completed the study individually and the true nature of the affective prime component (implicit effects of dominance in male faces) was concealed until debriefing at the conclusion of the experiment.

**Affective Prime.** The priming task followed standard protocol for the paradigm (Fazio et al., 1986) and was controlled using Direct-RT (Jarvis, 2016). Participants were instructed to stay focused on the screen at all times throughout the task and to begin whenever they were ready. Onscreen instructions informed participants that a face would briefly flash on the screen followed
by a target word which they were to categorise as dominant or submissive using either the ‘/’
(submissive) or ‘z’ (dominant) keys on the keyboard in front of them. Participants were told to keep
their index fingers on these keys at all times throughout the experiment. Twelve practice trials
followed using two stimulus identities (masculinised and feminised at three levels of facial hair
growth: 12 images in total) that did not appear in the experiment proper. Participants then began the
six blocks of test trials.

Each block consisted of 60 trials, followed by a rest period before commencing the next
block. Each trial began with a fixation period (500 ms) of the characters ‘XXXXXXXX’ displayed
centrally followed by the affective prime: one of the 36 facial stimuli (200 ms) in the centre of the
screen. A blank screen then appeared for 100 ms, before the target word appeared in the centre of
the screen. Participants then categorised the word as dominant or submissive using the keyboard. If
a participant responded within 200 ms of the facial stimuli appearing (i.e., before the target word
appeared), a message informed them that they had responded too quickly and to wait until the target
stimulus appeared on subsequent trials. Similarly, if responses occurred after 2000 ms participants
were told that they needed to respond quicker on subsequent trials. Trials were separated by a 1000
ms inter-trial interval and continued across the six test blocks until all combinations of face primes
and target words had been rated.

Explicit Dominance Ratings. Once the affective prime task had been completed, participants
began the explicit rating task. This involved rating the same 36 faces as used in the affective prime
task, presented in random order, for dominance on a scale from 1 (not dominant) to 10 (extremely
dominant).

4.3 Results

Affective Prime. Response times for incorrect (18.1 %), and overly short (<200 ms) or long
(>1750 ms) responses (1.2 %) were excluded prior to calculating means. Correct response times
(RTs) were then averaged across each stimulus/target combination (i.e., clean-shaven, masculine,
dominant) before being entered into a 3 (facial hair) X 2 (facial dimorphism) X 2 (word category)
repeated-measures ANOVA.

There was a main effect of word category (Table 4.3.1), such that RTs to dominant words
($M = 643.97, SD = 121.52$ ms) were significantly faster than those to submissive words ($M =
660.57, SD = 137.98$ ms, $d = 0.13$). No other significant main effects or interactions emerged from
the model (Table 4.3.1).
Table 4.3.1. Repeated-measures ANOVA testing effects of facial hair, facial masculinity, and stimulus category of response times

<table>
<thead>
<tr>
<th></th>
<th>df</th>
<th>F</th>
<th>p</th>
<th>Partial Eta²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Facial hair</td>
<td>2,116</td>
<td>0.281</td>
<td>0.755</td>
<td>0.005</td>
</tr>
<tr>
<td>Facial masculinity</td>
<td>1,58</td>
<td>0.230</td>
<td>0.633</td>
<td>0.004</td>
</tr>
<tr>
<td>Stimulus category</td>
<td>1,58</td>
<td>7.467</td>
<td>0.008</td>
<td>0.114</td>
</tr>
<tr>
<td>Facial hair x facial masculinity</td>
<td>2,116</td>
<td>1.268</td>
<td>0.285</td>
<td>0.021</td>
</tr>
<tr>
<td>Facial hair x stimulus category</td>
<td>2,116</td>
<td>0.299</td>
<td>0.742</td>
<td>0.005</td>
</tr>
<tr>
<td>Facial masculinity x stimulus category</td>
<td>1,58</td>
<td>2.270</td>
<td>0.137</td>
<td>0.038</td>
</tr>
<tr>
<td>Facial hair x facial masculinity x stimulus category</td>
<td>2,116</td>
<td>1.249</td>
<td>0.291</td>
<td>0.021</td>
</tr>
</tbody>
</table>

Explicit Dominance Ratings. Explicit ratings of dominance were averaged across stimulus identities within each condition and entered as the dependent variable in a 3 (facial hair) × 2 (facial dimorphism) repeated-measures ANOVA (Table 4.3.2).

There was a significant main effect of facial hair on explicit ratings of dominance (Table 4.3.2). Faces with full beards ($M = 6.03, SD = 1.24$) were rated as more dominant than faces with stubble, ($M = 5.01, SD = 1.16; t(59) = 8.44, p < .001, d = 0.85$), and clean-shaven faces, ($M = 4.13, SD = 1.32; t(59) = 10.16, p < .001, d = 1.48$). Faces with stubble were rated as more dominant than clean-shaven faces, ($t(59) = 7.95, p < .001, d = 0.71$). There was also a significant main effect of facial masculinity (Table 4.3.2), so that dominance ratings were higher for masculine faces ($M = 5.24, SD = 1.06$) than feminised faces ($M = 4.87, SD = 1.12, d = 0.35$).

Table 4.3.2. Repeated-measures ANOVA testing effects of facial hair and facial masculinity on explicit dominance ratings

<table>
<thead>
<tr>
<th></th>
<th>df</th>
<th>F</th>
<th>p</th>
<th>Partial Eta²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Facial hair</td>
<td>2,118</td>
<td>87.797</td>
<td>&lt;0.001</td>
<td>0.598</td>
</tr>
<tr>
<td>Facial masculinity</td>
<td>1,59</td>
<td>35.841</td>
<td>&lt;0.001</td>
<td>0.378</td>
</tr>
<tr>
<td>Facial hair x Facial masculinity</td>
<td>2,118</td>
<td>4.715</td>
<td>0.011</td>
<td>0.074</td>
</tr>
</tbody>
</table>

There was also a significant facial hair × facial masculinity interaction (Table 4.3.2). Comparisons within each facial category found that masculine faces were judged as significantly more dominant than feminised faces for clean-shaven ($t(59) = 5.62, p < .001$), stubble ($t(59) = 3.69, p < .001$), and fully bearded ($t(59) = 2.91, p = .005$) faces. Comparisons across all categories revealed that masculinised faces with full beards were rated as significantly more dominant than masculinised and feminised versions of faces with stubble and masculinised and feminised versions of clean-shaven faces, (all ≥ $t(59) 7.45, all p ≤ .001$; Figure 4.3.1). Feminised faces with full beards were rated as significantly more dominant than masculinised and feminised versions of clean-shaven faces or faces with stubble (all ≥ $t(59) 5.22, all p ≤ .001$; Figure 4.3.1). Masculinised faces with stubble were rated significantly higher than feminised and masculinised clean-shaven faces,
(all $\geq t(59) 6.02$, all $p \leq .001$; Figure 4.3.1). Feminised faces with stubble were also rated higher than masculinised and feminised versions of clean-shaven faces, (all $\geq t(59) 3.32$, all $p \leq .01$; Figure 4.3.1).

Figure 4.3.1 The left panel shows the mean response times ($\pm 1$ SEM) following the dominance affective prime to faces varying in facial hair (clean-shaven, stubble and fully bearded) that had been morphed to appear more masculine (grey bars) and more feminine (white bars). The right panel shows the mean dominance ratings ($\pm 1$ SEM) to faces varying in facial hair (clean-shaven, stubble and fully bearded) that had been morphed to appear more masculine (circular symbol on solid line) and more feminine (square symbol on dashed line).

This interaction could reflect effects of facial masculinity being larger within clean-shaven faces compared to faces with any facial hair, which would suggest diminishing effects of facial masculinity on dominance ratings with advancing levels of bearded-ness. However, it could be that the additive effect of facial hair is larger for feminised compared to masculinised faces, which would further suggest diminishing returns to facial masculinity on dominance ratings as facial hair increases. Effect sizes were indeed larger for comparisons within clean-shaven faces ($d = 0.40$) compared with faces with stubble ($d = 0.27$) and full beards ($d = 0.17$). Further, when faces were feminised, effect sizes for beards vs. stubble ($d = 0.85$) and beards vs. clean-shaven faces ($d = 1.51$) were higher than the same comparisons for masculinised versions of faces ($d’s = 0.80, 1.33$ respectively). Effect sizes between stubbled and clean-shaven faces were also higher for feminised ($d = 0.74$) than masculinised faces ($d = 0.59$). This suggests diminishing returns to facial masculinity as faces become more hirsute and that additive effects of facial hair are incrementally larger within feminised than masculinised faces (Figure. 4.3.1).

**Implicit and Explicit Measures.** To test whether implicit responses to dominance were predictive of explicit ratings of dominance we correlated scores on the two measures. Participants may simply have slower reaction times across all stimuli and high dominance ratings across all stimuli. This could produce a correlation between the two measures that is unrelated to either implicit or explicit responses towards dominance in male faces. By computing difference scores, we
can calculate whether effects of facial characteristics produce consistent differences in perceptions of dominance across levels of facial hair and facial masculinity between the two measures.

We calculated the average difference between masculine and feminine face affective prime response times for each of the three levels of facial hair for dominant target words (i.e., masculine bearded face reaction times minus feminine bearded face reaction times, etc.). We then calculated the difference between explicit dominance ratings for masculine and feminine faces within each of the three levels of facial hair (i.e., ratings for masculinized clean-shaven faces minus ratings for feminised clean-shaven faces). After controlling for these effects, no significant correlations were observed between explicit and implicit measures of dominance for clean-shaven stimuli ($N = 59, r = -0.13, p = 0.320$), stimuli with stubble ($r = -0.16, p = 0.239$) or full bearded stimuli ($r = -0.08, p = 0.537$).

We repeated this process for each level of facial hair for masculinised and feminised faces. We first calculated the difference between bearded and stubbled faces, then the difference between bearded and clean-shaven faces, and finally, the difference between stubbled and clean-shaven faces for masculine and feminine faces separately for implicit and explicit responses. For masculine faces, no correlations were observed between implicit and explicit measures of the difference between bearded and stubbled faces ($r = 0.05, p = 0.711$), between bearded and clean-shaven faces, ($r = -0.02, p = 0.908$), or between stubbled and clean-shaven faces ($r = -0.04, p = 0.777$). For feminised faces, no significant correlations were observed between differences for bearded and stubbled faces ($r = -0.13, p = 0.342$), between bearded and clean faces ($r = -0.17, p = 0.200$), or between stubbled and clean-shaven faces ($r = -0.20, p = 0.132$).

We then tested whether main effects of facial hair or masculinisation correlated across implicit and explicit measures. We calculated the difference between masculinised and feminised faces across all levels of facial hair, and conversely the differences between each level of facial hair averaged across masculinised and feminised conditions. We found a significant negative correlation between the difference score of masculine and feminine faces across implicit and explicit measures, ($r = -0.28, p = 0.035$), indicating that the effect of masculinised faces on perceived dominance on one task predicted an effect on the other. However, we interpret this finding with caution, given the large number of correlations calculated and the small effect size. No significant correlations were observed for differences between bearded and stubbled faces ($r = -0.08, p = 0.535$), bearded and clean-shaven faces ($r = -0.12, p = 0.356$), or stubbled and clean-shaven faces ($r = -0.16, p = 0.233$) when averaged over masculine and feminine conditions.

To test for possible influences of non-heterosexuality on ratings, all analysis were re-run excluding participants who did not identify as heterosexual. No changes in the pattern of results were observed.
4.4 Discussion

In many mammals, weaponry like claws, horns or canines serve directly in contest competition with conspecifics (Emlen, 2008). In other cases, secondary sexual traits provide information regarding age, dominance and rank within social groups (A. F. Dixson et al., 2005; Grueter et al., 2015; Sheehan & Bergman, 2015). Converging evidence suggests men’s secondary sexual traits also play a strong role in intra-sexual communication of age, masculinity and dominance (Puts, 2010, 2016) and function during intra-sexual agonistic displays (Puts et al., 2015). We hypothesised that facial masculinity and beardedness would receive high explicit ratings of dominance and receive the most rapid associations with dominance in an affective prime task. We found that neither trait exerted significant effects on implicit responses to male dominance. Our findings have implications for understanding the role of men’s secondary sexual traits during intra-sexual assessments.

We found that beards and facial masculinity both exerted significant effects on ratings on men’s dominance, which replicates several previous studies (B. J. W. Dixson & Vasey, 2012; Neave & Shields, 2008; Perrett et al., 1998; Saxton et al., 2016). However, we found no implicit associations between men’s beardedness, facial masculinity, or their combination, on men’s implicit responses to male dominance. This result was surprising given the amount of empirical support that implicit processes underpin gaze cueing towards facial expressions of dominance (Terburg, Hooiveld, Aarts, Kenemans, & van Honk, 2011) and that androgen-dependent facial features enhance ratings of men’s formidability and dominance (Puts et al., 2015; Sell, Hone, & Pound, 2012). We did observe a main effect whereby dominance related target words were categorised quicker. This indicates a clear statistical difference in the recognition of the target words’ respective categories, confirming that there was sufficient variance in the response times to potentially detect differential effects of the primes. The predicted interaction between the targets and primes failed to emerge, however, suggesting that levels of masculinity and beardedness (beyond simply being male) in the affective primes did not differentially prime responses to dominance related words.

There are both strengths and limitations to the use of implicit measures from social psychology to study effects of facial morphology on dominance perceptions. The primary advantage of implicit measures is the circumvention of explicit and conscious responses, providing access to automatically activated attitudes (Fazio et al., 1986). However, a key limitation is that priming effects are subject to influences such as the strength of the prime and the valence associated with response categories (Fazio, 2001). With regards to the priming stimulus in the present study, there is no reason to believe that they were insufficient in their strength to elicit implicit attitudes. Previous research has found that face images can be used successfully as affective primes (e.g.
Banse, 2001; Koranyi et al., 2013; T. T. Li & Lu, 2014; Palermo & Schmalzl, 2006; Yang et al., 2012) and that complex, feature-based images are capable of being processed as primes. For example, Livingston and Brewer (2002) manipulated the physiognomy of African-American facial primes to have high prototypical features or low prototypical features. The degree of prototypicality of facial physiognomy influenced the strength of automatic evaluations in an effective priming task. Thus, not only can feature-based primes elicit automatic responses, but variation in these features also produces variation in the automatic evaluations elicited (Maddox, 2004; Maddox & Dukes, 2008). More recent research has shown priming effects of facial emotional expression (e.g. angry, fearful, or, happy), which is similarly based on feature-based processing, and that these effects were consistent across multiple face prime stimuli (Yeung, Taylor, Rubino, & Barton, 2015). Masculinity in faces is similarly the result of multiple facial characteristics (i.e., jaw size, brow ridge thickness, cheekbone height, width-to-height ratios) influencing feature-based evaluations. Given previous research, it seems unlikely that the absence of automatic evaluations in response to masculine or feminine faces is a consequence of the characteristics of the primes themselves. This is also the case for levels of facial hair, which require much more simple visual processing (i.e., attention to presence or absence of facial hair).

It is also unlikely that error in categorising target words contributed to the absence of priming effects. Although 18.1% inaccuracy is higher than in similar affective prime paradigms (e.g. 6% in Koranyi et al., 2013), it is not entirely unexpected given the relative novelty of the categories employed (i.e., dominant vs. submissive). Furthermore, we observed a main effect of target type, such that dominant words were categorised faster on average, indicating discriminant responses in both categorisation (81.9% accuracy) and latency. This is consistent with the affective priming literature, which demonstrates greater impact of negatively evaluated stimuli than positive (for review see Klauer, 1997). In the present study, dominant words are likely to have been perceived as more negative (e.g. menace, threat, fight, violent, strong), which may account for more rapid responses. Yet, this did not interact with the characteristics of the prime as would be expected in the context of implicit attitudes. It appears that while facial stimuli primes may evoke attitudes in affective prime tasks, masculine and/or bearded faces may not elicit implicit attitudes pertaining to dominance.

Analysis of explicit ratings, in contrast, revealed a significant interaction between facial hair and the facial masculinity manipulation on men’s ratings of male dominance. As predicted, masculinised versions of faces that were clean-shaven, had stubble or had full beards were rated as looking more dominant than their feminized counterparts. However, effects of masculinity on dominance ratings were reduced as faces displayed more pronounced facial hair. Thus, clean-shaven masculinized faces were rated as significantly less dominant than feminized faces with light
stubble. This diminishing return of facial masculinity suggests that facial hair may enhance sexually dimorphic features that are judged as giving men a more masculine and socially dominant appearance. It might be assumed that ancestral males were typically bearded, essentially masking sexually dimorphic craniofacial shape. Interestingly, men’s facial width-to-height ratio, a measure that is associated with ratings of men’s dominance and aggressiveness (Geniole et al., 2015), was found to predict ratings of men’s aggressive-ness in both bearded and clean-shaven versions of the same male, although bearded faces were rated as more aggressive looking than clean-shaven faces overall (Geniole & McCormick, 2015). Present day populations differ markedly in natural distribution of facial and body hair. For example, the Kung hunter-gatherers grow little facial compared to neighbouring Kavango subsistence farmers (Winkler & Christiansen, 1993), whereas Ainu hunter-gatherers of Japan remain some of the most hirsute individuals ever documented (Harvey & Brothwell, 1969). Limited cross-cultural data suggest that beards are more consistently associated with male dominance than attractiveness (B. J. W. Dixson & Vasey, 2012; Neave & Shields, 2008; Saxton et al., 2016). Yet men can easily groom or remove their beards, essentially manipulating their perceived masculinity. Although much of the variation in facial hair grooming may simply reflect trends in fashion, data from 1842 to 1971 among men in London revealed that facial hair was more common when the marriage market was more female biased (Barber, 2001), possibly because men augment their masculinity when intra-sexual competition is strongest. However, when facial hair becomes too common it is judged as less attractive than when it is rare, suggesting that negative frequency dependence may underpin some of the variation in facial hair fashions (Janif et al., 2014). Although further cross-cultural research remains a priority, mating market dynamics such as the strength of intrasexual competition appear to influence temporal fluctuations in the frequency of facial hair.

There are limitations to the explicit ratings portion of the study design that should be highlighted. Firstly, we only used six male identities in the explicit ratings of dominance. Although this is the same number as has been used in some previous studies (Saxton et al., 2016), other studies have used larger stimulus sets (Janif et al., 2014). We chose this number to avoid participant fatigue in our within-subjects design. However, we acknowledge that future studies looking in more detail at how underlying natural variation in facial morphometrics interact with beardedness to determine judgments of men’s sociosexual attributes using a larger sample of faces would be valuable. Further, there is evidence that wearing a beard changes men’s feelings of masculinity and confidence (D. R. Wood, 1986) that may have translated into greater confidence or dominance when posing neutral expressions in the photographs. Such effects have been found to influence judgments of faces in other studies. For example, t-shirt colour influences judgments of facial attractiveness despite it not being visible to raters (Roberts, Owen, & Havlicek, 2010). Given that
wearing a false beard enhances men’s self-perceived masculinity (D. R. Wood, 1986), we acknowledge that some subtle effects of confidence may have transferred onto the ratings of dominance ascribed to bearded over clean-shaven faces. Unfortunately, our study cannot account for the effects of growing or removing facial hair on men’s self-perceived confidence. Subtle differences in skin complexion and facial fatigue between the time periods in which photographs were taken could also have contributed to how facial hair was judged. Finally, our sample of raters was ethnically mixed whereas the stimuli we employed were restricted to males of European descent. Extending our study to include more diverse stimuli and raters will therefore be important. For the present, our results provide preliminary experimental evidence that facial hair plays a more salient role in driving judgments of male dominance than experimentally manipulating facial masculinity. However, the mechanisms by which men gain an advantage, if any, in mate competition by enhancing their beardedness remains a challenge for future research to tackle.
Section 5. Estimating the Effects of Genes and the Environment
Paramount to disentangling causes of variation in human mating strategy is the ability to partition the variation into that caused by the external environment (e.g. diet, pathogen density, climate, economy, etc.) and genes. For example, are mate preferences dependent upon environmental conditions or are they the result of genetic effects (e.g. Zietsch, Lee, et al., 2015)? In reality, neither nature (genes) nor nurture (everything else) can entirely account for the variation in a trait, however; in order to test hypotheses regarding selection pressures that have shaped mating strategy in humans it is often necessary to examine and quantify the contribution of each. Although recent developments in technology allow for the genotyping of unrelated individuals to estimate genetic effects, for many purposes the most powerful methods of quantifying genetic effects rely on family pedigrees (Evans & Martin, 2000). The most common of such methods relies on the natural experiment provided by identical twins.

5.1 The Classic Twin Design

In twins, nature has provided a semi-random (though see Shur, 2009) distribution of varying genetic similarity (i.e., ~50% vs. 100%) between pairs of siblings who share the same uterine and home environment. Identical twins are as close as researchers have to clones: they share their entire genome sequence as a consequence of a single egg being fertilised by a single sperm (i.e., monozygotic: MZ) before dividing into two embryos. On the other hand, non-identical twins are only as similar as siblings: they share, on average, half of the same genome and developed from separate eggs fertilised by separate sperm cells (i.e., dizygotic: DZ). In contrast to non-twin siblings, however, both identical and non-identical twins share the womb with each other. Consequently, this allows scientists to compare the phenotypic similarity of twin-pairs to their genetic similarity and make inferences about the contribution of shared genes, the shared environment, or any effects that may make the twins different from each other (Neale & Cardon, 1992).

Key to this endeavour is a number of assumptions. The first is that twins are not substantially different from non-twins in the population. If they were, it would be improper to generalise findings derived from twin studies to non-twin populations as twins may on average differ on the underlying trait. Although there are some differences between twins and non-twins that emerge in early childhood (Marceau et al., 2016; Pettersen, Nelson, Watson, & Stanley, 1993; Phillips, 1993; Record, McKeown, & Edwards, 1970), likely as a consequence of womb sharing, by age five there is very little evidence to suggest that twins and non-twins differ on physical or mental characteristics (for review see Evans & Martin, 2000; Nilsen, Bergsjo, & Nome, 1984). Although there is no substantial evidence to suggest that twins are vastly different from non-twins, twin studies that include a non-twin sibling are able to compare means and variances to the twins in the sample and check for significant deviations.
The second key assumption of the twin study is that identical twins and non-identical twins share aspects of their environment to the same degree. Twins, for the most part, will live in the same home, attend the same school, eat similar food, have similar friends, and importantly shared a uterine environment. However, if it were the case that identical twins experienced more similarity in their environments than non-identical twins and this in turn affected their behaviour, any increased similarity of identical twins relative to non-identical twins could be falsely attributed to genetic influences. To this end, identical twins are in fact treated more similarly than non-identical twins across a number of domains (Scarr, 1982), such as how they are dressed, how their rooms are kept, and whom they play with (Kendler, Heath, Martin, & Eaves, 1986), however; in order for this to be problematic this similar treatment must also cause similar behaviour in the twins.

Given the importance of this assumption for the validity of the twin study, the link between how identical twins are treated and how this affects their behaviour has been studied extensively. As such, an important caveat to the increased similarity of treatment of identical twins is that under closer inspection the more similar treatment largely occurs in response to the behaviour of the twins (Lytton, 1977), which of course tends to be more similar because of genetic similarity. Moreover, studying the effects of how identical twins are treated has revealed no links between greater similarity of environment and similarity of phenotype across cognitive testing, personality batteries, or career interests (Morris-Yates, Andrews, Howie, & Henderson, 1990). Taking advantage of families in which twin zygosity is unknown has also presented researchers with an opportunity to test the results of similar treatment. For example, if the more similar treatment of identical twins is based on parental expectations, studying non-identical twins who are believed to be identical should reveal similar results. Yet, perceived zygosity appears to have no bearing on trait similarity across a number of domains, and it appears that twins are likely to be treated according to their actual zygosity (Kendler, Neale, Kessler, Heath, & Eaves, 1993).

5.2 Quantifying Causes of Variation

On the basis of these assumptions, researchers can apply some relatively simple mathematics in order to assess the contributions of genes and the environment to any given trait. Genetic effects may be additive (i.e., the sum of allelic effects across the genome) or non-additive. Non-additive effects are the result of interactions between loci (i.e., epistasis: allele effects at one locus depend on alleles at another locus) or within loci (i.e., dominance: allele effects at a locus depend on the other allele at the same locus). Consequently, there are two mathematical models that are employed to partition causes of variation. Under an ACE model, researchers can estimate variation due to additive genetic effects, or narrow sense heritability ($h^2$ or $A$), the common environment (C), and the residual environment (E) (Neale & Cardon, 1992). Alternatively, an ADE model will provide estimates of additive genetic effects (A), non-additive genetic effects (D), and
the residual environment (E). Because D and C cannot be modelled at the same time (Eaves, 1977; Keller & Coventry, 2005), twin correlations are used to determine when an ACE or an ADE model is more appropriate. If non-identical twins correlate at less than half the magnitude of identical twins (e.g. \( MZr = 1.00, DZr = .25 \)) it is likely that non-additive genetic effects are present and an ADE model should be used. When A and D are estimated in the same model, they can be summed to give an estimate of the total genetic effects on variation in a trait (broad-sense heritability \( H^2 \)). Otherwise, it is typical to use an ACE model. One limitation of the twin model is that C and D are negatively confounded. The influence of C will increase the correlation between non-identical twins, whereas D would reduce the correlation. As such, the presence of one may mask the other.

Because of the unique nature of each variance component (i.e., A, D, C, and E), a number of predictions can be made regarding twin correlations. If additive genetic effects were the only contributing factor to trait variation, one would expect identical twins to correlate perfectly (\( r = 1.00 \)) whereas non-identical twins should correlate, on average, at half the magnitude (\( r = .50 \)), reflecting their genetic similarity. If dominance effects (i.e., a non-additive genetic effect) were the only source of influence on a trait, non-identical twins would correlate on average at .25 while identical twins would still correlate perfectly (for further explanation of non-additive effects, see Posthuma et al., 2003). The common environment (C) is mathematically defined as any non-genetic influence that causes twins to be more similar to each other and is presumed to influence identical and non-identical twins equally (see section 5.1). If the common environment were the only source of variation in a trait, both identical and non-identical twins would correlate perfectly (\( r = 1.00 \)). The common environment is typically thought to include aspects of the home such as parenting style, exposure to pets, socioeconomic status, and the religious or political orientation of the household.

Finally, the twin model can estimate the proportion of variance caused by aspects of the environment that are not shared between twins. Any influence that reduces the similarity of the twin pair will contribute to estimates of the residual environment (E), which is by definition unique to each individual. Consequently, if all variation in a trait were due to the residual environment neither identical nor non-identical twins would correlate at all (\( r = .00 \)). Precise components of the residual environment are almost impossible to identify for two reasons. Firstly, individual environmental exposures that are influential for one individual may not generalise to others (e.g. one individual may ruminate over a particular event at school whereas another may not pay it any mind, causing behavioural divergences in future). Secondly, the residual environment will include measurement error. This has two important implications: causal aspects of the residual environment are difficult to distinguish from mere error and familial effects (i.e., A, D, and, C) estimates tend to be systematically conservative because error will cause divergence between twins. Despite the
statistical partitioning that can be achieved using the classical twin model, genes and environment do not act independently of one another and frequently interact (GxE). These interactions are not modelled in the classical twin design (though they can be integrated into more sophisticated models) and subsequently manifest in estimates of the genetic effect (A/D) (Neale & Cardon, 1992).

Historically, researchers calculate the relative contribution of genes and the environment on trait variation by applying algebra to raw twin correlation data (Falconer, 1960). With computational advancements, twin modelling is now done using structural equation modelling software (e.g. OpenMx: Boker et al., 2011) which allows for the estimation of confidence intervals and the ability to compare nested models (i.e., ACE vs. AE). The estimated variance components from the twin model are standardised and sum to one and thus represent a percentage of variation accounted for by genes, the shared environment, and any residual effects (see Figure 5.2.1).

Figure 5.2.1. Structural equation model of variance components in classic twin design. Phenotypic variation is the result of the sum of latent factors A, C/D, and E.

5.3 Extending the Classic Twin Design

By extending the twin study to multiple traits at once, any variation shared between traits can be partitioned into A, C/D, and E: this is done by comparing the ratio of cross-twin cross-trait correlations between identical and non-identical twins. For example, comparing the correlation between an identical twin’s height and their co-twin’s intelligence with non-identical twins can shed
light on genetic effects that influence both traits. Extending the twin model to multiple traits allows researchers to compute a genetic correlation \((r_G)\), which represents the degree of overlap in genes influencing both traits. A corresponding statistic can also be computed for both the common environment \((r_C)\) and the residual environment \((r_E)\). The proportion of the phenotypic correlation accounted for by each of these correlations can also be computed (i.e., what percentage of covariation between traits is due to genetic or environmental influences). The classic twin study can also be expanded to include siblings of twins, which increases the statistical power to detect variance components (Dolan, Boomsma, & Neale, 1999; Posthuma & Boomsma, 2000). Non-twin siblings are assumed to share variance components with their twin siblings at the same magnitude as non-identical twins in the twin model (i.e., \(A = .50, C = 1.00, D = .25, E = .00\)).

5.4 A Multivariate Twin Study of Disgust Sensitivity

In order to appropriately test evolutionary models of mating strategy and preferences, it was necessary that I was able to use the techniques discussed in section 5. This required computing a multivariate twin model in the OpenMx structural equation modelling software package (Boker et al., 2011) run on the R programming platform (R Core Team, 2014). The phenotype investigated, disgust sensitivity, was a prime candidate trait for several reasons. Firstly, the trait is evolutionarily relevant and is proposed to link to mating behaviour (Tybur, Lieberman, & Griskevicius, 2009). Secondly, modern approaches to disgust sensitivity propose three distinct domains: pathogen, sexual, and moral (Tybur et al., 2009). This represented an opportunity to investigate the underlying genetic architecture of the three domains. Thirdly, existing theory posited that disgust sensitivity was culturally conferred from parents to their offspring (Davey, Forster, & Mayhew, 1993). Only one attempt had been made to quantify genetic causes of variation in disgust via a twin study (Rozin & Millman, 1987), however; this research took place nearly thirty years ago and subsequently relied on a far smaller sample and far less sophisticated analyses than what is currently used in twin research.

In section 5.5, I present the resulting paper from this project, which was published in Emotion (Sherlock, Zietsch, Tybur, & Jern, 2016). The analyses required an in-depth understanding of twin modelling, including the underlying matrix algebra, the programming language in which the model was executed, and the use of more advanced multivariate modelling approaches.
Abstract
Response sensitivity to common disgust elicitors varies considerably among individuals. The sources of these individual differences are largely unknown. In the current study, we use a large sample of female identical and non-identical twins (N = 1,041 individuals) and their siblings (N = 170) to estimate the proportion of variation due to genetic effects, the shared environment, and other (residual) sources across multiple domains of disgust sensitivity. We also investigate the genetic and environmental influences on the covariation between the different disgust domains. Twin modeling revealed that approximately half of the variation in pathogen, sexual, and moral disgust is due to genetic effects. An independent pathways twin model also revealed that sexual and pathogen disgust sensitivity were influenced by unique sources of genetic variation, while also being significantly affected by a general genetic factor underlying all 3 disgust domains. Moral disgust sensitivity, in contrast, did not exhibit domain-specific genetic variation. These findings are discussed in light of contemporary evolutionary approaches to disgust sensitivity.

Keywords: individual variation, behavioural genetics, evolutionary psychology, pathogen threat, sexual strategies
Received: 23 October 2014, Revised: 29 May 2015, Accepted: 2 June 2015, Published Online: 5 Oct 2015
5.5.1 Introduction

Contemporary approaches to disgust typically employ an evolutionary perspective to understand the adaptive function and origin of the emotion (Chapman, Kim, Susskind, & Anderson, 2009; Curtis, de Barra, & Aunger, 2011; D. R. Kelly, 2011; Oaten, Stevenson, & Case, 2009; Rozin, Haidt, & McCauley, 2008; Tybur et al., 2009; Tybur, Lieberman, Kurzban, & DeScioli, 2013). Such approaches frequently suggest that disgust does not have a single, general adaptive function, but can rather be divided into domains with distinct functions. For example, Tybur et al. (2009) proposed that pathogen, sexual, and moral disgust each constitute functionally specialized disgust domains, meaning that they are elicited by different types of cues, moderated by different types of contextual factors, and specialized for neutralizing different types of adaptive problems that were reliably present in the ancestral environment. Specifically, pathogen disgust is posited to motivate the avoidance of physical contact with infectious microorganisms, sexual disgust is posited to motivate the avoidance of fitness-reducing sexual behaviours, and moral disgust is posited to mitigate the costs imposed by others’ violations of social rules (for more detail, see Tybur et al., 2013).

The upsurge in recent evolutionary work on disgust has been paralleled by work investigating individual differences in a trait called disgust sensitivity, which refers to the degree to which individuals experience disgust in response to common disgust elicitors. Researchers have become interested in disgust sensitivity partly because it varies with traits ranging from psychopathology (for review see Davey, 2011; de Jong & Merckelbach, 1998; Mancini, Gragnani, & D'Olimpio, 2001; Olatunji et al., 2007), to political ideology (Inbar, Pizarro, & Bloom, 2008; Tybur, Merriman, Caldwell Hooper, McDonald, & Navarrete, 2010), to phenomena such as stigmatization (Inbar, Pizarro, Knobe, & Bloom, 2009; Lieberman, Tybur, & Latner, 2012), ethnocentrism (Navarrete & Fessler, 2006), and mate preferences (Jones et al., 2013; Lee et al., 2013).

In addition to investigating how disgust sensitivity relates to these traits, a good deal of this work has been aimed at understanding the dimensionality of disgust sensitivity itself (Haidt, McCauley, & Rozin, 1994; Olatunji et al., 2007; Tybur et al., 2009). Patterns of individual differences in sensitivity to different disgust elicitors have been shown to relate to each other in ways consistent with the adaptationist theory outlined previously. For example, in their initial development of the Disgust Scale, Haidt et al. (1994) found that although self reports of disgust toward a wide variety of pathogen sources (e.g., corpses, spoiled foods, bodily wastes, interpersonal contact) strongly covaried with each other, they did not covary with disgust toward moral violations. In their modification of the Disgust Scale, (Olatunji et al., 2007) found that many disgust responses clustered into three highly correlated factors ($r$’s = .75, .88, and .65), each of which describe cues to pathogens (e.g., bodily wastes, contact with corpses, interpersonal contact).
However, the sexual domain that was included in the original Disgust Scale did not covary strongly with these other factors, and it was eliminated from the revised Disgust Scale. Finally, Tybur et al. (2009) conducted factor analyses on a large number of disgust elicitors that were nominated by participants. A three-factor structure emerged, and these three factors appeared to reflect pathogen, sexual, and moral items. Rather than eliminating the sexual and moral items because they did not covary with the pathogen items, as had been done with previous instruments, Tybur et al. (2009) developed the Three-Domain Disgust Scale (TDDS), a 21-item instrument that measured each of these three factors.

Subsequent work has demonstrated that sex differences and correlations with personality dimensions are consistent with predictions drawn from adaptationist models (e.g. Tybur, Bryan, Lieberman, Caldwell Hooper, & Merriman, 2011; Tybur & de Vries, 2013). As would be expected, openness to experience is negatively related to pathogen and sexual disgust (Tybur et al., 2011). Further, women score much higher on the sexual factor of the TDDS, but they score only slightly higher on the pathogen and moral factors (Tybur et al., 2011; Tybur et al., 2009). These differences in sexual disgust are thought to reflect discrepancies in the fitness costs between males’ and females’ mate choice (Trivers, 1972).

At a fundamental level, investigations into the dimensionality of disgust sensitivity (and the correlation between domains of disgust sensitivity and various other traits of interest) concerns between-individual variation. In contrast, evolutionary approaches to human behaviour (including those applied to understanding disgust (e.g. Tybur et al., 2013)) have tended to focus on universals, or evolved mechanisms that calibrate each individual to their specific conditions or environmental circumstances. As such, evolutionarily informed theories of the source of individual differences has been limited and generally oriented toward environmentally induced variation (Zietsch, de Candia, & Keller, 2014). Hence, despite the upsurge in evolutionarily oriented work on disgust sensitivity, little progress has been made in understanding what causes variability between individuals, including the possible role of genetic factors. Exploring the underlying causes of this variation can provide new information regarding the nature of disgust and potentially shed light on processes leading to variation in other related traits.

**What Gives Rise to Variability in Disgust Sensitivity?**

Some researchers have argued that variability in disgust sensitivity is entirely due to environmental factors, whereas others have proposed that variation may be partly heritable, that is, caused by variation in genes. Researchers favouring a primarily environmental account have suggested that differences in disgust sensitivity across individuals result from social transmission during formative years (Kim, Ebesutani, Young, & Olatunji, 2013; Rozin et al., 2008). Similarly, others have argued that culture provides the framework for variation in sensitivity to contaminants
Children from Western countries are more likely to identify germs as a cause for illness transmission (Siegal, Pat, & Eiser, 1990), whereas individuals from non-Western countries avoid contamination because of tradition, familiarity, or social cohesion (Rogers, 1995). Interestingly, children with autism, who have impaired social learning, experience delays in developing disgust, whereas children with other development disorders do not (Kalyva, Pellizzoni, Tavano, Iannello, & Siegal, 2010). This might point to a role of individual differences in socialization in the development of disgust sensitivity.

Supporting environmental perspectives, researchers have found that parents and children score similarly on measures labelled as “food contamination,” which has similar item content to disgust sensitivity instruments, for example, “On a 9-point scale how much would you like to eat soup from a thoroughly washed dog bowl?” (Rozin, Fallon, & Mandell, 1984). Specifically, Davey et al. (1993) report that correlations between parents and offspring on these items range from .33 to .52. However, influences of genetic and environmental factors are confounded in studies that simply observe phenotypic correlations between parents and offspring. Such correlations can stem from genetic factors, environmental factors, or a combination of the two. Children could score similarly on food contagion sensitivity because they share genes with their parents, or they might simply acquire similar sensitivity through observation of their parent’s behaviour (or other parentally mediated learning processes; Davey et al., 1993).

Studies of twins can distinguish between genetic and shared environmental effects, as family environment factors are assumed to affect twin pairs equally, whereas genetic effects will vary due to differential genetic similarity between identical and non-identical twins (100% vs. 50%, respectively). Twin studies of blood-injury phobias, of which disgust responses are a key symptom (Cisler, Olatunji, & Lohr, 2009; Olatunji, Cisler, McKay, & Phillips, 2010), might hint at the presence of heritable basis to pathogen disgust. Neale et al. (1994) found a higher degree of heritability in fear of blood (56% of variance) in a large sample of twins. Similarly, Fear Survey Schedule II data collected from twin samples have shown higher concordance rates for identical twins on items relating to blood, injury, and needles (Rose & Ditto, 1983).

In contrast with these hints at genetic effects, the only study that has used a twin design to test for genetic versus environmental effects on a disgust instrument has supported a pure environmental perspective. Rozin and Millman (1987) investigated the similarity of food contamination disgust between identical and non-identical twins. Participants in this study indicated how much they would like to eat a contaminated food source on a 9-point scale. The study showed that the correlation between identical twins’ food contamination disgust sensitivity ($r = .29$) was not significantly different from non-identical twins’ scores (Rozin & Millman, 1987). The authors interpreted these results as suggesting that variability in food contagion disgust has no genetic
component and is, hence, entirely caused by environmental factors (Rozin et al., 2008; Rozin & Millman, 1987). However, Rozin and Millman (1987) initial study of heritability was conducted with fewer than 40 identical and non-identical twin pairs. Further, analytical methods available at the time did not yield standard errors and confidence intervals; such statistics would have shown that little could be concluded about the relative magnitude of genetic and environmental effects from a sample of that size.

The Current Investigation

There have been no studies to date that have effectively disentangled environmental and genetic sources of variability in disgust sensitivity. Without basic knowledge of how variability in disgust sensitivity arises, it will be difficult to maximize the knowledge that can be gleaned from the impressive body of research on the topic. In the current study, we aim to provide such basic knowledge using a large sample of identical and non-identical twins ($N = 1,041$ individuals) and their siblings ($N = 170$) to estimate the proportion of variation in pathogen, sexual, and moral disgust sensitivity that is due to genetic effects, the family environment, and other (residual) sources.

In addition, we also investigate the genetic and environmental influences on the covariation between the different disgust domains. This allows us to compare the phenotypic factor structure of disgust sensitivity with the underlying genetic architecture. We use multivariate twin modelling to estimate the extent to which each disgust domain is influenced by specific versus common genetic factors; this can inform the degree to which covariation between pathogen, sexual, and moral disgust sensitivity arises from common genes versus specific genes.

5.5.2 Method

Participants. The statistical analyses in the present study were performed on a sample of 1,903 female twins and siblings of twins (mean age = 33.12, $SD = 4.99$). This is a subsample of the population-based Genetics of Sexuality and Aggression twin sample in Finland (see Johansson et al., 2013). Data were collected in the fall of 2013, targeting women who had participated in a similar data collection in 2006, and who indicated that they would be interested in participating in survey studies in the future. We were unable to add disgust sensitivity instruments to the twin survey before data were collected on males and, hence, data were only collected on females. All data were collected through a secure online questionnaire. In total, we sent invitations to 5,197 women by postal mail. Individuals who did not respond in any way over the first 2 weeks were sent a reminder letter, followed by another reminder letter another 2 weeks later unless they responded after the first reminder. Twenty-three individuals could not be reached (because the intended recipient had, e.g., moved abroad or passed away after their addresses were obtained from the Central Population Registry of Finland). In total, 2,249 women responded, and of these, 73
individuals did not wish to participate. Thus, the final response rate was 43.5%. An additional 273 women did not complete the necessary parts of the questionnaire, resulting in the final sample of 1,903 women.

The invitation to participate in the study was accompanied by a letter explaining the voluntary nature of the study. Potential participants were informed that they are free to terminate their participation at any stage of the study without providing a reason. Written informed consent was obtained from all participants. The Ethics Committee of the Abo Akademi University (Turku, Finland) approved the research plan in accordance with the Declaration of Helsinki (World Medical Association, 2013).

For the purposes of genetic analyses, pathogen, moral, and sexual disgust sensitivity data were available from 544 identical (mean age = 32.56, SD = 2.83) and 497 non-identical (mean age = 32.6, SD = 2.84) twins. Data were also obtained from 88 and 82 siblings of monozygotic (MZ; mean age = 33.17, SD = 1.62) and dizygotic (DZ; mean age = 33.17, SD = 1.80) twins, respectively. Zygosity was determined using DNA (Johansson et al., 2013). Only data that were available for both twins were used to estimate genetic effects; however, all available data were used to estimate means, variances, and within-person between-trait covariances via full information maximum likelihood modelling.

Measures. The TDDS (Tybur et al., 2009) is a 21-item measure composed of pathogen, sexual, and moral factors. Each item describes an act, concept, or situation that typically arouses some degree of disgust in individuals. Participants rate the degree to which they find each item disgusting on a 0 to 6-point scale. Items were translated into Finnish and a panel of four individuals with excellent command of both languages subsequently reviewed the translations. Consistent with previous versions in English, Dutch, and Japanese (e.g. Quintelier, Ishii, Weeden, Kurzban, & Braeckman, 2013; Tybur & de Vries, 2013), each of the subscales had acceptable internal consistency (α > .75), and the subscales were modestly intercorrelated (~.30 to .35). Mean scores on TDDS Pathogen, Sexual, and Moral Disgust subscales, respectively, were 3.38 (SD = 1.09), 2.89 (SD = 1.17), and 4.84 (SD = 0.90).

Statistical analyses. Genetic analyses of the data were conducted in R (R Core Team, 2014) using maximum likelihood modelling procedures contained in the statistical package OpenMx (Boker et al., 2011). We controlled for the mean effects of age by including it as a covariate in all genetic analyses. Maximum-likelihood modelling in OpenMx uses chi square as an indicator of goodness of fit to the data. The change in chi square is compared against change in degrees of freedom when parameters are estimated or constrained within the model (e.g., fixing them at zero, or equating different parameters) to determine the optimal model.
Estimating genetic and environmental effects on traits. The classical twin design allows variation in a trait to be partitioned into genetic (A and D), shared environmental (C), and residual (E) sources (Neale & Cardon, 1992). Genetic effects themselves may result from additive variation (the sum of allelic effects within and across genes, i.e., A) or non-additive variation (allelic interactions such as dominance and epistasis, i.e., D). The proportion of variation in a trait due to additive genetic factors is the narrow sense heritability ($h^2$), and the proportion of variance accounted for by all genetic factors is (additive plus non-additive) the broad-sense heritability ($H^2$).

Shared environmental influences are those shared between twins; these effects will cause both identical and non-identical twins to become more similar to each other. Residual effects may be due to unique and idiosyncratic experiences not shared between the twins, measurement error, or stochastic (chance) biological effects (e.g., mutations, neoplastic transformations and cancer). The ability to partition variance in phenotypes into these components is possible because identical (MZ) twins are genetically identical, whereas non-identical (DZ) twins share only half of their segregating genes. For example, if additive genetic influences were the only cause of variation in a trait, one would expect a correlation of 1.0 between MZ twin pairs and .5 for DZ twins. Further, if non-additive genetic sources were exclusively underlying trait variation, MZ correlations would be expected to be 1.0, whereas DZ pairs would correlate at a maximum of .25 (Posthuma et al., 2003).

Non-additive genetic and shared environmental effects are confounded in the classical twin model and are unable to be estimated at the same time.

The classic twin model can be extended to a multivariate model, allowing a decomposition of variance sources over multiple traits. The multivariate model uses cross-twin and cross-trait correlations in order to partition trait covariance in the same way in which variance is partitioned in a univariate twin model. In addition, a multivariate model allows for the partitioning of an observed correlation between two variables in genetic and environmental components. Non-twin siblings can also be included in the model, which enhances statistical power (Dolan et al., 1999; Posthuma & Boomsma, 2000).

5.5.3 Results

Preliminary analyses. As expected, there were no significant differences in mean disgust sensitivity scores between MZ twins, DZ twins, and non-twin siblings. Moreover, no significant differences were observed between the correlations of non-identical twin pairs and sibling pairs, except in the case of moral disgust sensitivity, for which the correlation between non-identical twin pairs was weaker than the correlation between sibling pairs. Given that there is no plausible reason for a real effect in this direction, and given the numerous statistical tests that were conducted in the preliminary testing, this was presumed to be due to sampling error and these correlations were equated in subsequent analyses. Identical twin pairs were more similar than non-identical twin pairs.
(see Table 5.5.3.1) across the disgust domains (pathogen, \( p < .001 \); sexual, \( p = .058 \); and moral, \( p < .001 \)), indicating genetic effects on all three domains of disgust sensitivity. Indeed, for each domain, identical twin pair correlations were more than double the non-identical twin pair correlations, indicating no \( C \) variance. This means that there is no evidence that any shared environmental factors influence disgust sensitivity. As per standard practice in such circumstances, we fitted \( ADE \) models instead of \( ACE \) models.

### Table 5.5.3.1. Twin pair and twin-sibling correlations for disgust sensitivity domains

<table>
<thead>
<tr>
<th>Zygosity</th>
<th>Pathogen</th>
<th>Sexual</th>
<th>Moral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Identical twin pairs (N = 131)</td>
<td>.49 (.36,.59)</td>
<td>.41 (.28,.52)</td>
<td>.50 (.37,.60)</td>
</tr>
<tr>
<td>Non-identical twin pairs (N = 100)</td>
<td>.23 (.07,.36)</td>
<td>.20 (.02,.35)</td>
<td>-.12 (-.32,.11)</td>
</tr>
<tr>
<td>Sibling pairs (N = 73)</td>
<td>.19 (-.04,.40)</td>
<td>.31 (.10,.48)</td>
<td>.39 (.17,.55)</td>
</tr>
<tr>
<td>Non-identical twin and sibling pairs equated (N = 173)</td>
<td>.22 (.09,.34)</td>
<td>24 (.11,.36)</td>
<td>.11 (-.04,.26)</td>
</tr>
</tbody>
</table>

### Estimating genetic and environmental effects on traits.

Variances components for each trait (see Table 5.5.3.2) were estimated from univariate genetic models. All three disgust domains were then fitted to a trivariate Cholesky ADE model. Although the estimates of \( D \) were non-zero for sexual and moral disgust domains, dropping \( D \) from the model did not have a significant effect on model fit (\( \chi^2 = 4.36, p = .63 \)). As such, and for the sake of simplicity, we interpret the \( AE \) model in the knowledge that any non-additive genetic effects \( D \) are absorbed into the \( A \) estimate, which will therefore represent the broad sense heritability of each trait (Keller, Medland, & Duncan, 2010).

### Table 5.5.3.2. Estimates (and 95% confidence intervals) of the proportion of variance in disgust sensitivity accounted for by additive genetic (\( A \)), non-additive genetic (\( D \)), and residual (\( E \)) sources.

<table>
<thead>
<tr>
<th></th>
<th>Pathogen</th>
<th>Sexual</th>
<th>Moral</th>
</tr>
</thead>
<tbody>
<tr>
<td>( A )</td>
<td>.50 (.00-.60)</td>
<td>.44 (.00-.56)</td>
<td>.00 (.00-.51)</td>
</tr>
<tr>
<td>( D )</td>
<td>.00 (.00-.55)</td>
<td>.02 (.00-.54)</td>
<td>.55 (.01-.65)</td>
</tr>
<tr>
<td>( A + D )</td>
<td>.50 (.37-.61)</td>
<td>.46 (.34-.57)</td>
<td>.55 (.42-.65)</td>
</tr>
<tr>
<td>( E )</td>
<td>.50 (.39-.63)</td>
<td>.54 (.43-.66)</td>
<td>.45 (.35-.58)</td>
</tr>
</tbody>
</table>

The multivariate analysis revealed that genetic effects influence the observed (phenotypic) correlation between the three disgust domains (see Table 5.5.3.3). As can be seen, the three domains correlated positively and moderately together. The proportion of correlations between the disgust domain phenotypes due to genetic correlation can also be seen in Table 5.5.3.3. A genetic correlation indicates the extent of overlap in the genetic variation of any pair of traits, directly analogous to phenotypic correlation, which indicates the extent of overlap in observed variation of
any pair of traits. Genetic correlations can be high even if the heritability of a trait is low, because correlations only indicate the overlap in genetic effects and not their magnitude. The same principles apply to residual correlations.

**Table 5.5.3.3** Phenotypic, genetic and residual correlations and proportion of phenotypic correlation between disgust domains due to genetic correlations.

<table>
<thead>
<tr>
<th></th>
<th>Pathogen-Sexual</th>
<th>Pathogen-Moral</th>
<th>Sexual-Moral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenotypic correlation</td>
<td>.35**</td>
<td>.33**</td>
<td>.30**</td>
</tr>
<tr>
<td>Genetic correlation</td>
<td>.28*</td>
<td>.53*</td>
<td>.43*</td>
</tr>
<tr>
<td>Residual correlation</td>
<td>.41*</td>
<td>.20*</td>
<td>.22*</td>
</tr>
<tr>
<td>Proportion of phenotypic correlation due to genetic correlation</td>
<td>.40*</td>
<td>.74*</td>
<td>.65*</td>
</tr>
</tbody>
</table>

* p < .05  
** p < .01

To further assess common and specific sources of variance in the three disgust domains, we fitted an independent pathways model to the data (see Figure 5.5.3.1). This model parameterizes variation in all three disgust domains as stemming from both common and specific sources of additive genetic and residual variance. As this model is not nested within the Cholesky trivariate model, the fit to the data could not be directly compared. Instead, we compared the models’ Akaike information criterion (AIC), which allows for comparisons of non-nested models by weighing goodness-of-fit and parsimony. The AIC was equivalent between the two models, indicating equal suitability for the data. To test whether the data could be modelled even more parsimoniously, we fitted a common pathways model. This model predicts that genetic and environmental variances influence covariation between the disgust domains via a latent factor. As the common pathways model is nested within the independent pathways model (see Gillespie & Martin, 2005) we compared model fit using likelihood ratio chi-square statistics. This common pathways model fit the data significantly worse than the independent pathways model further; this indicates that genetic and environmental factors have different effects on covariation between each disgust domain, $\chi^2 (\Delta df = 2) = 6.6, p = .04$. As such, we interpret the better-fitting independent pathways model (see Figure 5.5.3.1).
Figure 5.5.3.1. Path diagram of a trivariate AE Independent Pathway Model of moral, pathogen and sexual disgust, with squared path coefficients and 95% confidence intervals. Squared path coefficients represent the proportion of variance in an observed trait accounted for by the latent factor from which the path originates. Ac and Ec represent common sources of genetic (A) and residual variance (E). A and E are sources of variance specific to each trait.

Parameter estimates of the independent pathway model showed that genetic variation in moral disgust sensitivity was primarily common (i.e., shared by all three disgust domains), whereas genetic variation in both pathogen and sexual disgust sensitivity was primarily specific to each domain. Equating moral disgust’s specific genetic path to that of pathogen and sexual disgust resulted in significantly worse model fit, $\chi^2 = 4.74, p = .03$, and $\chi^2 = 5.88, p = .02$; this suggests that the amount of genetic variance accounted for by specific and common genetic factors indeed was different for the moral domain and the other two domains.

5.5.4 Discussion

Using a large sample of female twins and their siblings, we observed that individual differences in disgust sensitivity are substantially heritable. We detected no significant effect of the shared environment of the twins. Genetic effects accounted for approximately 50% of the variation between individuals across pathogen, sexual, and moral disgust domains. All domains share a common genetic influence, which accounted for approximately 18%, 11%, and 41% of the variance in pathogen, sexual, and moral disgust sensitivity, respectively. Sensitivity to sexual and pathogen, but not moral, disgust was also subject to specific genetic influences. We note that any measurement error contributes to estimates of residual variance; this suggests that, if anything, our model likely underestimates the proportion of variance due to genetic factors. Notably, these
findings stand in direct contrast to the only previous investigation of twin similarity in disgust measures (Rozin & Millman, 1987). This discrepancy might be explained by the low power to detect genetic effects in Rozin and Millman’s (1987) study. In sum, we show, for the first time, substantial genetic effects on individual differences in disgust sensitivity.

The finding of heritable variation in disgust sensitivity runs against a weight of opinion supporting an entirely environmentally mediated development of disgust (Kim et al., 2013; Rozin et al., 2008; Rozin & Millman, 1987). Indeed, the environment shared by twins was estimated to account for almost none of the variation in any disgust phenotype. This would include various sources of parental transmission, suggesting that food contagion correlations between parents and offspring found by Davey et al. (1993) were likely due to shared genes between parents and offspring. The influence of parental style, socioeconomic status, schooling and neighbourhood type (i.e., urban or rural), household cleanliness, family pet-keeping, and so on, would also be captured by the shared environment of the twins, so these effects likely do not strongly influence disgust sensitivity.

As with pathogen disgust, the environment shared by twins had little effect on variation in sexual or moral disgust. Religiosity and political affiliations tend to be similar within all members of the family and show substantial variation due to the shared environment of twins (Hatemi, Alford, Hibbing, Martin, & Eaves, 2008; Kendler & Myers, 2009). Given the relationship between disgust sensitivity and political ideology (and, specifically, sensitivity to sexual disgust; see Inbar, Pizarro, Iyer, & Haidt, 2012; Tybur et al., 2010), it might have been expected that these influences would inform sensitivity to sexual and moral disgust. However, recent arguments have suggested that sentiments related to sexual behaviours and how resources are divided between individuals cause political and religious stances, rather than vice versa (Weeden & Kurzban, 2014). Variation in political ideology might stem from variables with no shared environment influence (e.g., disgust sensitivity) as well as factors influenced by shared environment (e.g., coalitional membership).

Up to this point, there had been no direct evidence that variation in disgust sensitivity might be caused by genes. However, sensitivity to pathogen disgust has been proposed to link with immune system function (Fessler & Navarrete, 2003; Fleischman & Fessler, 2011), which is largely heritable (for example, 53% to 86% across various cytokines; de Craen et al., 2005). Natural and sexual selection in the ancestral environment were frequently driven by the recurring threat of infectious microbes (Maynard Smith, 1978; Tooby, 1982), and direct evidence adaptation to these pervasive threats has been observed in the human genome (Fumagalli et al., 2011). Individuals who are more susceptible to infectious disease (e.g., through compromised immune function) should invest more effort in avoiding cues to pathogens, perhaps by being more disgusted by them. As such, genetic variation in sensitivity to pathogen disgust may to some extent reflect “reactive
heritability” (Tooby & Cosmides, 1990), that is, indirect heritability due to calibration to a directly heritable trait (such as immune function). Indeed, several researchers have shown that classical immune markers increase due to exposure to cues of disease, suggesting that differing pathogen sensitivity may correspond to variation in immune function (for review, see Thornhill & Fincher, 2014)

The causes of variation in sexual disgust sensitivity had been similarly opaque to those underlying pathogen disgust. Tybur et al. (2013) posit sexual disgust as a co-opted form of pathogen disgust adapted to avoid detrimental sexual partners. Variation in sociosexuality (i.e., orientation toward uncommitted sexual relationships) and number of sexual partners have both been shown to have substantial heritable components (~50 to 60%: Bailey, Kirk, Zhu, Dunne, & Martin, 2000; Zietsch et al., 2008). These behaviours also strongly correlate with variation in sensitivity to sexual disgust (Al-Shawaf et al., 2014; Tybur, Inbar, Güler, & Molho, 2015). As such, the genetic variation in sensitivity to sexual disgust that we have observed could, like sensitivity to pathogen disgust, reflect reactive heritability, with individuals following more short-term sexual strategies necessarily exhibiting less disgust toward sexual activities (Gangestad & Simpson, 2000).

Alternatively, it may be the case that sexual disgust sensitivity drives sexual strategy. In terms of moral disgust, it might have been expected that variation in individuals’ reactions to third parties’ breaches of moral standards are largely a product of the environment in which they are raised, perhaps due to the combined influences of their family’s education, religion, and political beliefs. However, our finding of substantial heritable variation in sensitivity to moral disgust (and no shared environmental influence) aligns with previous research demonstrating that various moral sentiments are influenced by genetic variation (J. M. Olson, Vernon, Harris, & Jang, 2001); for example, upward of 40% of the variation in favourable attitudes to euthanasia, capital punishment, and abortion is due to genetic effects (J. M. Olson et al., 2001).

Common genes influenced variation in all three domains of disgust sensitivity. When variation in pathogen and sexual disgust was influenced by specific genetic factors, the common genetic factor accounted for almost all of the genetic variance in sensitivity to moral disgust. The common genetic elements underlying sensitivities to pathogen and sexual disgust might stem from the pathogen risks inherent to sexual interactions. Sexual contact exposes people to pathogens; either those transmitted from non-sexual contact (e.g., influenza virus) or those that are typically transmitted during genital-genital contact (i.e., sexually transmitted infections). Individuals who are more invested in avoiding pathogens, then, might also follow sexual strategies that limit partner number and the extent of sexual content (Tybur et al., 2015). Genes that influence investment in avoiding pathogens (perhaps those that influence ability to combat pathogens) might in turn influence both sensitivities to pathogen and sexual disgust. Additionally, there were common
genetic elements influencing sensitivity to sexual disgust that did not influence sensitivity to pathogen disgust. This might stem from the fact that sexual strategies are shaped not only by pathogen avoidance but also by numerous other factors that might have genetic sources (e.g. physical attractiveness, physical dominance in men: Gangestad & Simpson, 2000; Lukaszewski, Larson, Gildersleeve, Roney, & Haselton, 2014).

Our finding that only those genes that also influence sensitivities to pathogen and sexual disgust influence sensitivity to moral disgust aligns with evidence suggesting that many facets of moral condemnation result from emotional intuitions that serve functions outside of the moral domain. For instance, many third-party behaviours that are widely sanctioned across cultures involve acts that observers find disgusting to engage in themselves, that is, elicitors of pathogen or sexual disgust (Tybur et al., 2013). This might reflect a computational architecture in which experiences of pathogen or sexual disgust act as inputs into the psychology of moral condemnation. Consistent with this, some evidence suggests that individuals who are exposed to disgust-eliciting odours (Schnall, Haidt, Clore, & Jordan, 2008) and tastes (Eskine, Kacinik, & Prinz, 2011) rate social and moral transgressions (i.e., consensual sex with a first cousin) as more immoral (though see Landy & Goodwin, 2015). At a trait level, individuals who are more sensitive to pathogen disgust also report greater moral condemnation of myriad moral acts, including those described as violating norms of harm, care, and fairness (Chapman & Anderson, 2014). If feeling pathogen or sexual disgust more frequently or intensely increases moral judgment, then those genes that lead to variation in the pathogen and sexual factors of the TDDS might also influence sensitivity to the moral factor of the TDDS.

There were some limitations of our study that are inherent to the classical twin design. One is that shared environmental effects are confounded with non-additive genetic effects, such that they cannot be both modelled for a single trait, and if both are present to equal degrees, their effects will cancel each other out. As such, we cannot rule out the presence of some shared environmental effects that have been masked by non-additive genetic effects.

Another limitation of the classical twin design is that it affords very little statistical power to distinguish additive from non-additive genetic effects, because both effects predict similar patterns of twin correlations. Although maximum likelihood estimates suggested non-additive genetic influences for sexual and moral disgust, the estimates were too imprecise to statistically distinguish them from additive genetic effects. Future twin studies of disgust with larger sample sizes, or that include data from parents, may reveal the extent to which non-additive effects influence variation in disgust sensitivity, which can be informative in inferring past evolutionary selection pressures (Merilä & Sheldon, 1999).
A further limitation is that we only investigated heritability in women. This stands in contrast with Rozin and Millman’s (1987) twin study, which used data from both males and females. Although there is no particular reason to expect great differences in heritability across the sexes, women tend to be more disgust sensitive overall (though specifically for sexual disgust: Tybur et al., 2011; Tybur et al., 2009), which raises the possibility of different processes involved in disgust sensitivity development. As such, the extent to which the same or different genes influence men and women’s disgust sensitivity could be investigated in the future, as could sex differences in the aforementioned genetic relationships between disgust domains. That said, men and women’s scores on the TDDS are equally correlated with Big Five personality traits (Tybur et al., 2011), which suggests that they might be similarly related to the processes that lead to variation in personality. Further, it is rare to find sex differences in the genetic architecture of other traits (Vink et al., 2012).

Finally, this research was conducted using a sample of Finnish twins (Johansson et al., 2013), which precludes information about sources of variation between populations. It is important to note that the variance components presented here are proportions of variation within this particular population, and it is possible that a sample with more widely varying socio-environmental contexts might yield detectable shared environmental variance in disgust sensitivity.

We investigated sources of variation in pathogen, sexual, and moral disgust sensitivity using a classical twin study design. Approximately half of the variation in each domain is due to genetic factors, with no evidence for shared environmental effects. This study is to demonstrate genetic influences on disgust sensitivity, and it further yielded novel findings about the genetic architecture underlying the three domains. Understanding sources of variation in disgust may be of benefit to the treatment of related clinical disorders such as obsessive–compulsive and sexual disorders (Olatunji & McKay, 2009; Olatunji & Sawchuk, 2005; Penn & Potts, 1999). The findings may also contribute to greater understanding of the many normal behaviours to which disgust is related, including mate preferences (Jones et al., 2013; Lee et al., 2013), political ideologies (Inbar et al., 2008; Tybur et al., 2010), and social avoidance and punishment (Inbar et al., 2009; Lieberman et al., 2012; Navarrete & Fessler, 2006).
Section 6. Causes of Variation in Human Mating

Abstract
Choice of romantic partner is an enormously important component of human life, impacting almost every facet of day-to-day existence, however; the processes underlying this choice are remarkably complex and have so far been largely resistant to scientific explanation. One consistent finding is that, on average, members of romantic dyads tend to be more alike than would be expected by chance. Selecting for self-similarity is at least partially driven by phenotypic matching wherein couples share similar phenotypes, and preferences for a number of these traits are partly genetically influenced (e.g., education, height, social attitudes and religiosity). This suggests that genetically influenced preferences for self-similarity might contribute to phenotypic matching (and thus assortative mating), but it has never been studied in actual couples. In the present study, we use a large sample of twins to model sources of variation in self-similarity between partners. Biometrical modelling revealed that very little of the variation in the tendency to assortatively mate across 14 traits was due to genetic effects (7 %) or the shared environment of twins (0 %).

Keywords: assortative mating, quantitative genetics, mate choice, self-similarity, romantic preferences
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6.1 Introduction

Choice of romantic partner is an enormously important component of human life, impacting almost every facet of day-to-day existence including physical and psychological well-being, economic decision-making and social interaction. The processes underlying this choice are remarkably complex yet one finding remains more pervasive than any other: on average, members of romantic dyads tend to be more alike than would be expected by chance (Caspi et al., 1992; Klohnen & Mendelsohn, 1998; Mascie-Taylor, 1989; Plomin et al., 1977; Watson et al., 2004; Zietsch, Verweij, et al., 2011). This is referred to as assortative mating and appears to be one of the few consistent patterns of human coupling. Age, religiosity and social attitudes correlate strongly between partners (i.e. r = 0.97, 0.72, 0.61, respectively: Zietsch, Verweij, et al., 2011), whereas intelligence (r = 0.40: Mascie-Taylor & Vandenberg, 1988), attractiveness (r = 0.39: Feingold, 1988), and education (r = 0.45: Zietsch, Verweij, et al., 2011) correlate moderately. Small correlations have also been observed between both the height and weight of romantic partners (r = \sim 0.20), while weak correlations exist for personality traits (Feingold, 1988; Hatemi et al., 2010; Koenig, McGue, & Iacono, 2009; Martin et al., 1986; Price & Vandenberg, 1980; Watson et al., 2004; Zietsch, Verweij, et al., 2011). Researchers have even identified moderate assortative mating across a range of psychiatric disorders (Agrawal et al., 2006; Boomsma et al., 2010; Grant et al., 2007; Krueger, Moffitt, Caspi, Bleske, & Silva, 1998; Nordsletten et al., 2016).

Assortative mating can restructure the genetic and social environment in a multitude of ways. For instance, assortative mating can reshape the social environment by influencing the distribution of resources across society and increasing stratification of the economy (Schwartz, 2013). When spouses match based on income and educational attainment both resources and access to resources tend to become unevenly distributed. As a result, asymmetry occurs in the incentive to mate outside of one’s economic and educational sphere such that individuals high on the distribution stand to lose more by mating down compared to individuals low on the distribution (Schwartz, 2013). Moreover, assortative mating effectively moulds the genetic landscape, increasing homozygosity in the population (Lande, 1977; Wilson, 1973; Wright, 1921) and additionally increasing genetic variance, primarily in subsequent generations following positive assortment (Bulmer, 1971). These effects are caused by linkage disequilibrium between genes of like effect (Crow & Felsenstein, 1982). Assortative mating can also produce genetic correlations between different traits such as height and intelligence (Keller et al., 2013) when both are linked to another trait (in this case overall attractiveness) for which there is assortative mating.

Despite how pervasive and consequential assortative mating is, its causes are not well understood. There is little evidence for convergence, whereby partners are not initially similar but become more similar over the course of the relationship (Caspi et al., 1992; Watson et al., 2004;
Zietsch, Verweij, et al., 2011). This suggests that assortative mating must be largely due to initial choice. One cause of this initial choice assortment for which there is clear evidence is social homogamy—that is, couples meeting through similar social backgrounds (Nagoshi, Johnson, & Ahern, 1987; C. A. Reynolds, Baker, & Pedersen, 1996, 2000). However, statistical analyses of family data indicate that social homogamy cannot fully account for assortative mating, meaning that phenotypic matching, (i.e., selection of partners based on similarity in traits) must also play a role (Nagoshi et al., 1987; C. A. Reynolds et al., 1996, 2000; Zietsch, Verweij, et al., 2011). What causes phenotypic matching, though, is not at all clear. Passive phenotypic matching can occur if individuals are more likely to meet because of their similarity on a certain trait. For example, many couples meet in the workplace, and people in the same workplace may also have more similar intelligence than a random pair of people, causing similarity between couples for intelligence. However, there is evidence that people on average prefer self-similar traits in an ideal partner, and that these preferences are partly genetically influenced (e.g. education, height, social attitudes, religiosity; Heath & Eaves, 1985; Zietch et al., 2012). This suggests that genetically influenced preferences for self-similarity might contribute to phenotypic matching. Yet, it is has not been investigated whether there are in fact genetic influences on self-similarity of actual partner choices, as opposed to stated preferences. Additionally, there is substantial debate as to whether mate preferences are related to realised partner choice. For example, some research has shown that stated preferences are not predictive of choice in the context of a speed-dating paradigm (Eastwick et al., 2014; Kurzban & Weeden, 2005; though see N. P. Li & Meltzer, 2015; N. P. Li et al., 2013).

A previous study of twins and their spouses estimated genetic influences on partner choice across numerous traits at close to zero (Zietsch, Verweij, et al., 2011). Using data from over 27,000 individuals, Zietch and colleagues (2011) investigated the similarity of identical twins’ partners when compared to non-identical twins’ across 14 different traits including height, education, income, social attitudes, and physical and personality measures. Identical twins’ partners were no more similar than non-identical twins’ partners indicating small or non-existent genetic effects on partner choice. It should be noted that the researchers also controlled for the influence of assortative mating on partner similarity between twin pairs by regressing twin’s own traits from partner traits. However, this study only aimed to investigate genetic variation in partner selection across numerous traits rather than genetic variation in selecting self-similar mates.

In the present study, we analyse this same data from a large sample of twins and their partners, for whom we have measures of height, body mass index (BMI), personality traits, social attitudes, religiosity, education, income, and age. For each trait in each twin, we calculate a score that represents the degree to which the twin’s partner is similar to the twin (with relevant statistical controls). We then use biometrical modelling to quantify the influence of genetic and environmental
factors on variation individuals’ tendency to have a partner similar to themselves. The presence of a heritable component to the variation would suggest that genetic predisposition does play a role in people’s tendency towards phenotypic matching.

6.2 Method

Participants. Two cohorts of twins were contacted for data collection, first in 1988 (see Heath, Cloninger, & Martin, 1994) and then in 1990 (see Posner, Baker, Heath, & Martin, 1996). Health and lifestyle questionnaire responses were collected from over 6,000 independent families. Available data for each measure in the questionnaire varied considerably, and twins provided information about their partners’ religiosity, educational attainment, income, and age, where partner information was missing. The use of twin-reported data for these variables increased data for twin’s partners by 110–180%. For this study, we analysed the data regarding 11,357 twins and their partners (N = 6,397, see Table 6.2.1 for details). The Queensland Institute of Medical Research Human Research Ethics Committee approved this data collection. For further details regarding the sample, zygosity determination and data collection see Heath et al. (1994).

Table 6.2.1: Sample descriptives

<table>
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<th>Twins</th>
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<th>Partners</th>
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<tr>
<td></td>
<td>Male</td>
<td>Female</td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>No.</td>
<td>4388</td>
<td>6969</td>
<td>4259</td>
<td>2138</td>
</tr>
<tr>
<td>Age (SD)</td>
<td>31.95 (18.5-77.9)</td>
<td>34.4 (16.8-74.9)</td>
<td>40.8 (18.44-77.91, SD = 12.3)</td>
<td>35.8 (16.2-74.9, SD = 13.5)</td>
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Note: Descriptive statistics vary slightly from those reported previously due to winsorisation described in Measures (Zietsch et al., 2011). Average twin age is slightly lower than partners as many younger twins do not have partners but are included in the study (see Zietsch et al., 2011)

Measures. Availability of data for both members of a twin pair and each of their partners (henceforth referred to as complete sets) varied widely for different measures. However, incomplete sets were also used in the genetic modelling as their data contributed to the estimation of means, variance, and covariances using full information maximum likelihood. Age, height and weight were self-reported and available for 2195, 439, and 426 sets, respectively. BMI was calculated based on self-reported height and weight.

Education was reported as highest level of education completed, ranging from (1) primary school/high school (8–10 years of education), (2) high school (11–12 years of education), (3) apprentice/diploma, etc., (4) technical/college, (5) university degree, and (6) university postgraduate. This was provided for 1794 complete sets. Yearly income ($AUD) was assessed using the following response options: (1) none, (2) <$5,000, (3) $5,000–$10,000, (5) $15,000–$25,000, (6) $25,000, (7) $25,000–$35,000, (7) $35,000– $50,000, and (8) >$50,000. At the time of data collection, the average full time income was ~ $25,000. Data for this question were available for 1197 complete sets.
Participants’ religiosity was indicated by frequency of church attendance (or other observances). 1765 complete sets were available with responses consisting of: (1) rarely, (2) once or twice a year, (3) every month or so, (4) once a week, and (5) more than once a week.

Participants’ social attitudes were scored based on responses to a list of topics (e.g. casual sex, immigration, birth control). Participants indicated whether they agreed or disagreed with each topic (0 or 2 depending on direction), or if they were uncertain (1). After an exploratory factor analysis of responses, 23 items were combined (absolute factor loadings >0.30) into a scale of conservative to liberal attitudes with scores ranging from 0 to 46 (for further detail see Zietsch, Verweij, et al., 2011). When three or fewer responses were missing, item scores were replaced with the mean. Participants with more than three missing responses were treated as missing. 441 complete sets were available for this scale.

To measure personality traits, participants were administered short versions of two commonly used personality inventories. Psychoticism, neuroticism, and extraversion scores were derived from 36 items (12 per trait) of the Eysenck Personality Questionnaire (EPQ-R: Eysenck, Eysenck, & Barrett, 1985). Harm avoidance (18 items), novelty seeking (19 items), reward dependence (12 items), and persistence (5 items) scores were derived from the revised Tridimensional Personality Questionnaire (TPQ: Cloninger, Przybeck, & Svrakic, 1991). Items were presented as true/false and responses were summed. If >25% of responses of a scale were missing, the scale was treated as missing. Otherwise, missing responses were replaced with the mean. To maintain normality of the data, scores were transformed into arcsine values after being converted to a proportional scale (Freeman & Tukey, 1950). Data for personality items ranged from 439 to 451 complete sets. Where both partners’ and twins’ reported data was available, correlations for religiosity, education, income, and age were 0.87, 0.84, 0.74 and 0.99, respectively (Zietsch, Verweij, et al., 2011).

Values three standard deviations above and below the mean were winsorised for all continuous variables in order to minimise departures from normality. As we analysed a measure of similarity between twins and their partners rather than scores on these measures themselves, we do not describe the original sample here. For sample descriptives, see Zietsch et al. (2011).

**Partner Self-Similarity Scores.** The aim of this study was to investigate the heritability of assortative mating on multiple traits. To do so, we required a measure of similarity between partners for each trait. However, simply taking the difference between a twin and their partner would be unsuitable given that many of the traits themselves vary due to familial influences (i.e., genetic and environmental variation shared between twins) and that the majority of traits are normally distributed within the population. Normally distributed traits will create differential likelihoods of finding a self-similar mate based on an individual’s own trait level. For example, it is more likely
that individuals who are of average intelligence will find a partner similar in intelligence to them because this is the mode of possible partner intelligence. Likewise, extremely intelligent individuals will struggle to find someone who is similarly intelligent simply because there are fewer of these individuals in the population. Because many of the traits in the current study are heritable they will be shared more strongly between identical twins than non-identical twins and so too will the probability of matching with a self-similar mate. As a result, simply analysing the heritability of the partner self-similarity via a raw difference score would result in an estimate that is biased by the heritability of the trait on which twins are matching and estimates would to some extent reflect the familial effects on the traits themselves. For example, assume mating is completely random for height. A twin who is extremely tall will likely have a co-twin who is extremely tall (because of the heritability of height), and both twins are likely to have partners much shorter than themselves (and thus large twin-partner difference scores). The same would apply for extremely short twins. Comparing correlations between identical and non-identical twin pairs would therefore give the appearance of a heritable basis to self-similarity preferences for height despite all of the twins mating at random. To control for this effect, for each twin we calculated a partner self-similarity score that controlled for the extremity of the twin’s own phenotype. The method was as follows.

Firstly, scores on all traits were standardized separately by sex. This controls for sex effects so that the difference between twins and partners is relative to the average score of their sex. Thus, an average height male will not be dissimilar from an average sized woman despite actually being taller, whereas a taller than average male would be dissimilar from an average sized female. We then calculated a partner self-similarity score for each twin for each trait by calculating the absolute difference between the twin’s sex-standardized value and their partner’s sex-standardized value. By taking the absolute difference between a twin and their partner we measure only the difference between couples regardless of direction, such that a twin who is taller than average with an average height partner is equally similar as a twin who is shorter than average with an average height partner. This score was then regressed on the twin’s age (to control for any possible effects thereof). Then, to control for the extremity of the twin’s own phenotype, we regressed the residual of the age regression on the absolute value of the twin’s own standardised score. Regressing on the absolute score controls for the degree of a twin’s deviation from the sex standardized mean for the trait. The resulting score is essentially the degree of similarity between a twin and their partner that can’t be predicted by the extremity of the trait itself.

**Simulation Testing.** To confirm that simple difference scores would be problematic and to validate our measures of assortative mating, we ran a number of simulations to ensure that we were correct to control for phenotypic extremity and are still able to detect genetic effects on self-similarity partnering. We first generated a simulation to test the hypothesis that normally distributed
traits might generate spurious estimates of heritability as described above. This involved firstly simulating a population of identical and non-identical twin pairs with correlated scores on a hypothetical variable. In this simulation, 10,000 pairs of identical twins and non-identical twins were generated with scores that correlated at $r \sim 0.70$ and $r \sim 0.45$, respectively (similar to twin correlations reported for multiple traits in Zietsch, Verweij, et al., 2011). We then generated scores for twin partners under random mating conditions (i.e., partner traits uncorrelated to the twin’s scores, $r \sim 0.00$). We then calculated a difference score by taking the absolute difference between each twin and their partners (Figure 6.2.1a), and tested the correlation of these difference scores within twin pairs. Over approximately one hundred simulated runs, identical twin similarity scores correlated more strongly ($r = 0.12, p < 0.001$) than that of non-identical twins, ($r = 0.03, p < 0.001$, respectively), despite mating completely randomly. This correlation would suggest 12% of the variance in the similarity of twins to their partners is due to genetic effects, despite no preference for self-similarity. Thus any heritability estimates would actually be attributable to familial effects on the trait itself resulting in greater similarity between twins and their partners. Controlling for this effect is the purpose of regressing out the extremity of the twins’ own scores from partner self-similarity as described earlier, so we checked that our method would work as planned. After regressing on the absolute value of the twins’ own scores (Figure 6.2.1b), the twin pair correlations for partner self-similarity scores were no longer significant for both identical, $r \sim 0.00$, and non-identical twin pairs, $r \sim 0.00$, over one hundred simulations. This demonstrates that our method of controlling for familial effects on the twin’s phenotype is successful in removing spurious estimates of heritability for partner self-similarity preferences. Our method of control additionally improves the substantial negative skew in absolute difference scores between a twin and their partner.

We then ran another simulation to test that this method would not remove genuine genetic influences on self-similarity preferences. To do this, we again simulated 10,000 identical and non-identical twin pairs. For the purposes of simulating a genetic factor influencing assortment via preference, absolute difference scores between twins and their partners were now generated such that they were correlated within twin pairs more strongly in identical ($r \sim 0.15$) than in non-identical twin pairs ($r \sim 0.07$) in accordance with a modest genetic influence of $\sim15\%$ (Figure 6.2.1c). As per the explanation above, this difference score was regressed on the absolute value of the twin’s own trait score and the residual of this regression was taken as the final index of similarity (Figure 6.2.1d). Over a hundred simulations, this process retained the majority of similarity between identical twin pairs, $r \sim 0.09, p < 0.001$, which were more than twice the size of non-identical twin pair correlations, $r \sim 0.04, p < 0.001$, consistent with the presence of genetic influences and a heritability estimate of approximately 9%.
In accordance with these successful simulations, we computed a controlled measure of partner self-similarity for each of the twin’s traits. Histograms of absolute partner differences and controlled partner differences can be seen in Supplemental Figure 1 (Appendix A) and demonstrate substantial variability in the degree of self-similarity between couples. We additionally checked that our measures of partner self-similarity were not simply reflecting Zietsch et al.’s (2011) measures of partner traits. Supplementary Table I (Appendix A) shows that correlations between the two measures (self-similarity score and partner traits) were modest or null, confirming that we are measuring a unique aspect of mate choice (i.e., self-similarity) independent of the previous study. Whereas Zietsch, Verweij, et al. (2011) conducted analysis on partner traits controlling for the twin’s own characteristics, we generate a measure of trait similarity between twins and their partners controlling for the extremity of the twins’ own traits.
Box 1. Steps taken to compute a measure of self-similarity controlling for extremity of individuals’ phenotypes

**Step 1:** Scores standardised separately by sex
- Controls for sex differences such that partner similarity is relative to average height within sex

**Step 2:** Self-similarity scores calculated by computing absolute difference between twins and partners
- Controls for direction of difference (i.e., +1 standard deviation is equally different to -1 standard deviation on any given trait)

**Step 3:** Self-similarity regressed on age
- Residual of this regression controls for any age effects

**Step 4:** Residual then regressed on absolute value of twin’s own trait
- Controls for extreme deviation of individuals’ phenotypes from sex-standardized mean.

**Estimating genetic and environmental influences on assortative mating.** Using the classical twin design, we are able to partition variance in similarity scores into that caused by genetic factors, that due to shared environmental sources, and that due to any residual sources (Neale & Cardon, 1992). Genetic causes of variation consist of additive effects (A: the sum effect of alleles across the genome) and non-additive effects (D), which include interactions within and across genes (i.e., dominance and epistasis, respectively). The proportion of variance accounted for by additive effects constitutes the narrow-sense heritability of the trait ($h^2$). The sum of A and D indicates the broad-sense heritability ($H^2$) of the trait. Family environmental factors (C) include any non-genetic effect shared by the twins. This includes factors such as socioeconomic status of the household, the shared uterine environment, and parenting style, but mathematically is defined as any non-genetic effect that contributes to correlations between twins. As C and D are confounded in the classic twin design, only one can be estimated in a given model. Which is estimated is determined by preliminary analysis of twin correlations, and D is generally presumed to be present in the case that MZ correlations are more than twice that or DZ twin pairs. Finally, other sources of variation that are unshared between twins are included in an estimate of residual influences (E). These can be environmental influences not shared by the twins, chance biological effects such as mutations, any individual experiences of the twins, and, importantly, measurement error. These variance components are standardized so as to sum to 1. Accordingly, parameter estimates of A, C/D, and E indicate the proportion of variance in a trait accounted for by each source.

Partitioning variation in a trait into these components is possible due to the identical segregating genes shared by identical (monozygotic: MZ) twins, compared with the 50% of segregating genes shared by non-identical or dizygotic (DZ) twins. For instance, if additive genetic influences were underlying variation in a trait entirely, MZ twins would correlate at 1.0 and DZ twins at 0.5. If non-additive genetic factors solely influenced trait variation, MZ twins would correlate at 1.0 and DZ twins (at most) at 0.25 (for a detailed explanation see Posthuma et al., 2003). Conversely, were shared environmental factors driving variation in a trait, both MZ and DZ
twins would correlate at 1.0. If, however, residual sources were the only influence on variation in a trait, by definition neither MZ nor DZ twin pairs would correlate at all. Trait variance is typically the result of a combination of these factors. Structural equation modelling generates estimates of these influences which best match the observed data. The classical design has limited power to distinguish non-additive and additive effects (Keller et al., 2010). Further, non-additive genetic effects are confounded with shared environmental effects. Additionally, when non-additive variance is not modelled, it is absorbed into estimates of additive genetic variance.

**Statistical analysis.** All data preparation was conducted in SPSS Statistics, version 22.0 (IBM Corp, 2013). Genetic modelling was executed using the statistical package OpenMx (Boker et al., 2011) in R (R Core Team, 2014). OpenMx employs maximum-likelihood modelling, using a goodness-of-fit index that is distributed as Chi squared. We determined the optimal model for the data by systematically constraining parameters within the model (e.g. fixing them at zero, or equating different parameters), and comparing changes in Chi squared against changes in degrees of freedom. This allowed us to test hypothesis regarding those specific parameters, for instance whether MZ and DZ correlations are significantly different from each other.

6.3 Results

Consistent with previous research, we observed assortative mating on most traits (Table 6.3.1). We conducted preliminary testing on adjusted and unadjusted partner self-similarity scores for each trait, which revealed no significant mean differences among zygosity groups or between the sexes, indicating a similar degree of assortativity between males and females and their respective partners across every trait (see Supplementary Materials [Appendix A]). In the case of several traits, variances differed significantly between males and females and between identical and non-identical twins. Twin pair correlations were also significantly different between MZ males and MZ females for religiosity (see Table 6.3.2). $\chi^2 = 10.23, p = 0.01$. In these instances, we could see no reason that these differences were due to anything other than chance over multiple model comparisons and these parameters were subsequently equated for further modelling.
To investigate genetic effects on partner self-similarity scores, we first tested whether MZ twin pair correlations were greater than DZ twin pair correlations on our phenotype-controlled measure of self-similarity preference. This was not the case for any of the traits being investigated, indicating no significant genetic effects. As a result, subsequent modelling did not include estimates of D and instead modelled C. Across all traits, univariate ACE models revealed non-significant heritability estimates ranging from 0 to 17 % of variance in partner self-similarity scores (Table 6.3.3). Shared environmental effects were also negligible, with none reaching statistical significance across any of the traits.

### Table 6.3.1: Mean difference between twins and their partners and partner correlations for each trait

<table>
<thead>
<tr>
<th>Trait</th>
<th>Number of pairs</th>
<th>Trait Standard Deviation</th>
<th>Mean couple difference †</th>
<th>Partner Correlation (r) *</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index</td>
<td>2,282</td>
<td>1.15</td>
<td>0.98</td>
<td>.14***</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>2,326</td>
<td>10.09</td>
<td>1.45</td>
<td>.20***</td>
</tr>
<tr>
<td>Education</td>
<td>6,162</td>
<td>1.53</td>
<td>1.15</td>
<td>.48***</td>
</tr>
<tr>
<td>Income</td>
<td>4,150</td>
<td>1.91</td>
<td>2.25</td>
<td>.17***</td>
</tr>
<tr>
<td>Religiosity</td>
<td>6,183</td>
<td>1.75</td>
<td>0.64</td>
<td>.74***</td>
</tr>
<tr>
<td>Attitudes</td>
<td>2,327</td>
<td>4.67</td>
<td>1.23</td>
<td>.67***</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>2,369</td>
<td>.30</td>
<td>1.10</td>
<td>.05*</td>
</tr>
<tr>
<td>Extraversion</td>
<td>2,342</td>
<td>.30</td>
<td>1.10</td>
<td>.04</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>2,367</td>
<td>.18</td>
<td>1.00</td>
<td>.16***</td>
</tr>
<tr>
<td>Harm Avoidance</td>
<td>2,346</td>
<td>.27</td>
<td>1.14</td>
<td>.03</td>
</tr>
<tr>
<td>Novelty Seeking</td>
<td>2,343</td>
<td>.21</td>
<td>1.04</td>
<td>.09***</td>
</tr>
<tr>
<td>Reward Dependence</td>
<td>2,345</td>
<td>.24</td>
<td>1.23</td>
<td>.03</td>
</tr>
<tr>
<td>Persistence</td>
<td>2,341</td>
<td>.30</td>
<td>1.10</td>
<td>.03</td>
</tr>
<tr>
<td>Age</td>
<td>6,397</td>
<td>13.51</td>
<td>0.25</td>
<td>.96***</td>
</tr>
</tbody>
</table>

† Mean difference represents the average absolute difference between twins and their partners on trait score for Education, Income and Religiosity (because they are ordinal measures), and difference in standard deviations of trait for the other measures.

◆ Partner correlations do not include parents of twins, unlike those reported in Zietsch et al. (2011) and as a result correlations differ slightly in this sample.

*p < .05

***p < .001
<table>
<thead>
<tr>
<th>Trait</th>
<th>MZM Twins</th>
<th>MZF Twins</th>
<th>MZ-Equated</th>
<th>DZM Twins</th>
<th>DZF Twins</th>
<th>DZOS Twins</th>
<th>DZ-Equated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index</td>
<td>.04 (-.16, .23)</td>
<td>.08 (-.07, .23)</td>
<td><strong>.07 (-.05, .18)</strong></td>
<td>.24 (-.14, .53)</td>
<td>.25 (.02, .44)</td>
<td>.02 (-.24, .27)</td>
<td><strong>.17 (.01, .31)</strong></td>
</tr>
<tr>
<td>Height (cm)</td>
<td>.02 (-.14, .18)</td>
<td>.03 (-.11, .18)</td>
<td><strong>.03 (-.08, .14)</strong></td>
<td>.15 (-.28, .48)</td>
<td>-.06 (-.26, .15)</td>
<td>.01 (-.24, .25)</td>
<td><strong>-.01 (-.16, .14)</strong></td>
</tr>
<tr>
<td>Education</td>
<td>.15 (.04, .25)</td>
<td>.12 (.05, .19)</td>
<td><strong>.13 (.07, .19)</strong></td>
<td>.15 (.01, .29)</td>
<td>.07 (-.02, .16)</td>
<td>.05 (-.05, .15)</td>
<td><strong>.08 (.02, .14)</strong></td>
</tr>
<tr>
<td>Income</td>
<td>.02 (-.10, .14)</td>
<td>.11 (.03, .20)</td>
<td><strong>.08 (.01, .15)</strong></td>
<td>.00 (-.16, .17)</td>
<td>.05 (-.07, .18)</td>
<td>-.02 (-.13, .09)</td>
<td><strong>.01 (-.07, .08)</strong></td>
</tr>
<tr>
<td>Religiosity</td>
<td>-.05 (-.16, .06)</td>
<td>.15 (.09, .22)</td>
<td><strong>.10 (.04, .15)</strong></td>
<td>.06 (-.10, .21)</td>
<td>.03 (-.05, .11)</td>
<td>-.04 (-.14, .06)</td>
<td><strong>.01 (-.05, .07)</strong></td>
</tr>
<tr>
<td>Attitudes</td>
<td>.14 (-.08, .34)</td>
<td>.10 (-.05, .24)</td>
<td><strong>.11 (-.01, .23)</strong></td>
<td>.08 (-.17, .31)</td>
<td>-.11 (-.32, .11)</td>
<td>-.01 (-.32, .11)</td>
<td><strong>-.02 (-.16, .12)</strong></td>
</tr>
<tr>
<td>Neuroticism</td>
<td>.00 (-.17, .17)</td>
<td>.00 (-.14, .13)</td>
<td><strong>.00 (-.11, .11)</strong></td>
<td>.46 (.09, .66)</td>
<td>.08 (-.13, .28)</td>
<td>.14 (-.09, .34)</td>
<td><strong>.15 (.00, .28)</strong></td>
</tr>
<tr>
<td>Extraversion</td>
<td>-.07 (-.26, .14)</td>
<td>-.05 (-.19, .10)</td>
<td><strong>-.05 (-.17, .07)</strong></td>
<td>-.15 (-.43, .19)</td>
<td>-.04 (-.25, .17)</td>
<td>.09 (-.13, .29)</td>
<td><strong>-.01 (-.14, .13)</strong></td>
</tr>
<tr>
<td>Psychoticism</td>
<td>-.08 (-.31, .18)</td>
<td>.05 (-.09, .18)</td>
<td><strong>.02 (-.10, .14)</strong></td>
<td>-.22 (-.53, .24)</td>
<td>-.08 (-.32, .17)</td>
<td>.01 (-.23, .24)</td>
<td><strong>-.06 (-.22, .11)</strong></td>
</tr>
<tr>
<td>Harm Avoidance</td>
<td>-.07 (-.28, .14)</td>
<td>.02 (-.12, .16)</td>
<td><strong>-.01 (-.12, .11)</strong></td>
<td>.18 (-.09, .41)</td>
<td>-.07 (-.27, .15)</td>
<td>-.02 (-.22, .19)</td>
<td><strong>.01 (-.12, .14)</strong></td>
</tr>
<tr>
<td>Novelty Seeking</td>
<td>-.06 (-.28, .16)</td>
<td>.08 (-.05, .22)</td>
<td><strong>.04 (-.07, .16)</strong></td>
<td>-.25 (-.48, .04)</td>
<td>-.03 (-.23, .16)</td>
<td>-.09 (-.29, .12)</td>
<td><strong>-.10 (-.22, .03)</strong></td>
</tr>
<tr>
<td>Reward Dependence</td>
<td>.11 (-.10, .31)</td>
<td>.07 (-.07, .21)</td>
<td><strong>.08 (-.03, .20)</strong></td>
<td>-.10 (-.36, .21)</td>
<td>-.17 (-.36, .05)</td>
<td>.07 (-.16, .28)</td>
<td><strong>-.07 (-.20, .07)</strong></td>
</tr>
<tr>
<td>Persistence</td>
<td>-.01 (-.22, .21)</td>
<td>-.06 (-.20, .08)</td>
<td><strong>-.05 (-.17, .07)</strong></td>
<td>-.01 (-.33, .31)</td>
<td>.04 (-.18, .26)</td>
<td>-.07 (-.30, .18)</td>
<td><strong>-.01 (-.16, .14)</strong></td>
</tr>
<tr>
<td>Age</td>
<td>0.09 (0.00, 0.19)</td>
<td>0.11 (0.04, 0.18)</td>
<td><strong>0.10 (0.05, 0.16)</strong></td>
<td>-0.01 (-0.15, 0.12)</td>
<td>0.07 (-0.02, 0.16)</td>
<td>0.09 (0.00, 0.18)</td>
<td><strong>0.07 (0.01, 0.12)</strong></td>
</tr>
</tbody>
</table>

**Note:** M = male, F = Female, OS = Opposite Sex, MZ = Monozygotic, DZ = Dizygotic, Equated = correlations equated across sex.
We also estimated the influence of extremity on heritability by re-running the genetic analysis without regressing self-similarity scores on the extremity of twins’ own traits. It appears that our control had the expected effect of reducing the influence of phenotypic extremity, as familial estimates were higher than in our controlled measure for 8 of 14 traits. Estimates for the remaining traits were either the same (4/14) or slightly larger (2/14). Similar estimates are likely due to non-significant correlations of similarity scores between twins. The method of control only attempts to account for extreme phenotypes that are shared between twin pairs. If the twins are already uncorrelated, the control will have no effect and there would subsequently be no spurious variance estimates as a result. Subsequent analysis was therefore conducted using our phenotype-controlled measure of self-similarity.

To investigate the possibility of genetic and environmental influences on an overall tendency towards self-similarity in partners (i.e., not specific to any one trait), we ran a multivariate model including our controlled measure of assortative mating on all traits. This allowed us to equate the influence of A and C to be equal across all of the traits to test if there is significant variation in the size of familial [i.e., genetic (A) and shared environmental effects (C)] effects between traits, while also providing an overall estimate of variance components on the tendency to assortatively mate (Table 6.3.3). No significant change in model fit was observed when estimates of A were equated across all traits, $\chi^2_{13} = 0.0, p = 0.96$, nor when C was equated across traits $\chi^2_{13} = 2.8, p = 0.96$. As A and C are partially confounded in the twin design, we have more power to detect A and C together than independently. When the contributions of both A and C were equated across traits (i.e., A equal across traits, C equal across traits) in the same model, no significant change in fit was observed, $\chi^2_{26} = 24.9, p = 0.52$. This indicates that genetic, shared environmental, and residual sources of variance are similar in magnitude for assortative mating on all of the traits we investigated. The final model shows small but significant familial influences on variation in assortative mating across the measured traits (accounting for 7% of total variation).
Table 6.3.3 Proportion of variance accounted for by additive genetic (A), shared environmental (C), and residual (E) effects on the tendency to assortatively mate for individual traits and across all traits.

<table>
<thead>
<tr>
<th>Trait</th>
<th>A (95%CI)</th>
<th>C (95%CI)</th>
<th>Familial effects [A + C] (95%CI)</th>
<th>E (95%CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index</td>
<td>.00 (.00, .20)</td>
<td>.12 (.00, .22)</td>
<td>.12 (.02, .12)</td>
<td>.88 (.78, .98)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>.02 (.00, .13)</td>
<td>.00 (.00, .11)</td>
<td>.02 (.00, .13)</td>
<td>.98 (.87, 1.00)</td>
</tr>
<tr>
<td>Education</td>
<td>.07 (.00, .19)</td>
<td>.06 (.00, .16)</td>
<td>.13 (.08, .19)</td>
<td>.87 (.81, .92)</td>
</tr>
<tr>
<td>Income</td>
<td>.08 (.00, .15)</td>
<td>.00 (.00, .12)</td>
<td>.08 (.01, .15)</td>
<td>.92 (.85, .99)</td>
</tr>
<tr>
<td>Religiosity</td>
<td>.17 (.00, .22)</td>
<td>.00 (.00, .16)</td>
<td>.17 (.12, .22)</td>
<td>.83 (.78, .88)</td>
</tr>
<tr>
<td>Attitudes</td>
<td>.09 (.00, .21)</td>
<td>.00 (.00, .15)</td>
<td>.09 (.01, .21)</td>
<td>.91 (.79, 1.00)</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>.00 (.00, .12)</td>
<td>.04 (.00, .13)</td>
<td>.04 (.00, .13)</td>
<td>.96 (.87, 1)</td>
</tr>
<tr>
<td>Extraversion</td>
<td>.00 (.00, .07)</td>
<td>.00 (.00, .06)</td>
<td>.00 (.00, .07)</td>
<td>1.00 (.93, 1.00)</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>.00 (.00, .12)</td>
<td>.00 (.00, .10)</td>
<td>.00 (.00, .12)</td>
<td>1.00 (.88, 1.00)</td>
</tr>
<tr>
<td>Harm Avoidance</td>
<td>.00 (.00, .11)</td>
<td>.01 (.00, .10)</td>
<td>.01 (.00, .11)</td>
<td>.99 (.89, 1.00)</td>
</tr>
<tr>
<td>Novelty Seeking</td>
<td>.02 (.00, .13)</td>
<td>.00 (.00, .09)</td>
<td>.02 (.00, .13)</td>
<td>.98 (.87, 1.00)</td>
</tr>
<tr>
<td>Reward Dependence</td>
<td>.05 (.00, .15)</td>
<td>.00 (.00, .10)</td>
<td>.05 (.00, .15)</td>
<td>.95 (.85, 1.00)</td>
</tr>
<tr>
<td>Persistence</td>
<td>.00 (.00, .08)</td>
<td>.00 (.00, .08)</td>
<td>.00 (.00, .08)</td>
<td>1.00 (.92, 1.00)</td>
</tr>
<tr>
<td>Age</td>
<td>.11 (.00, .16)</td>
<td>.00 (.00, .11)</td>
<td>.11 (.05, .16)</td>
<td>.89 (.84, .95)</td>
</tr>
<tr>
<td>Across all traits</td>
<td>.07 (.00, .09)</td>
<td>.00 (.00, .07)</td>
<td>.07 (.05, .09)</td>
<td>.93 (.91, .95)</td>
</tr>
</tbody>
</table>

6.4 Discussion

Modelling revealed non-significant, near-zero heritability across all 14 of the traits investigated in the study. Similarly, no significant effects of the shared environment were detected. Combined familial effects (i.e., A and C modelled together) did have a significant influence on self-similarity across several traits: more than 10% of the variation in partner similarity on age, BMI, education, and income was accounted for by the combination of genes and the shared environment. Unfortunately, we lacked the statistical power to disentangle these effects clearly. In the case of religiosity, Verweij, Abdellaoui, et al. (2014) have previously demonstrated a sizable genetic
correlation between the trait itself and preferences for the trait in a potential partner, which may drive assortativity.

Biometrical modelling revealed that very little (7%) of the variation in the tendency to assortatively mate across 14 traits was due to genetic effects when controlling for the extremity of twins’ own phenotypes. Shared environmental effects were also near-zero and non-significant. Confidence intervals indicated that genetic and shared environmental effects combined (i.e., familial effects) account for at least 5% but no more than 9% of the variance in assortative mating across all of the measured traits on average. In addition, we also quantified the degree to which familial influences may influence assortative mating via their effect on the twin’s phenotypes. At most this accounted for an additional 10% of the estimated variance in any one trait. This leaves the majority of variation in assortative mating unexplained. It should be noted that estimates of residual variance contain measurement error, though for objectively measured traits such as age, height, and BMI, this is unlikely to have contributed much to estimates of residual variance.

The absence of significant genetic effects on variation in partner self-similarity mirrors previous research on mate choice (Lykken & Tellegen, 1993; Zietsch, Verweij, et al., 2011). Zietsch, Verweij, et al. (2011) investigated the heritability of mate choice on the same traits investigated in this paper in the same sample of twins (where we investigate assortative mating on these traits) and found minimal, non-significant genetic effects. Though we used data from Zietsch, Verweij, et al.’s (2011) twin study of the heritability of mate choice and found similar results we are confident that we have measured a novel aspect of partner choice in the present study. As reported in the Methods, the measures employed in this study were minimally, or otherwise not at all correlated with measures from the previous study (see Supplementary Materials in Appendix A). The apparent lack of genetic influence on variation in both assortative mating, and mating on these traits themselves, is surprising given that almost all studied behavioural traits across thousands of studies show substantial heritable variation (Polderman et al., 2015), including a range of mate preferences (Verweij, Burri, & Zietsch, 2012, 2014; Zietsch, Lee, et al., 2015; Zietsch et al., 2012).

One explanation of the minimal heritability of mate choice, despite heritable mate preferences, may be that constraints of the mating market (e.g. an individual’s own mating value, the presence of ideal mates, or the number of competitors present (Penke, Todd, Lenton, & Fasolo, 2007)) limit the extent to which genetic influences on ideal partner preferences can be realised in an actual partner. However, individuals do tend to partner with those who are similar on a number of traits, which suggests that, to the extent that assortative mating is due to self-similarity preferences (as opposed to passive assortment), those preferences are being realised to some degree. As such, the fact that the vast majority (>90%) of variance in partner self-similarity was accounted for by residual sources of variation may point to the relative importance of propinquity in driving
assortative mating - that is, similar individuals are likely to inhabit similar environments and, as a result, are more likely to interact and ultimately pair with each other. On top of this effect, learned preferences based on past relationships, as well as simple chance effects, may also contribute to the dominant residual influence on variation in partner self-similarity.

The absence of shared environmental effects on assortative mating is also surprising. In this sample, and indeed generally, couples correlated strongly on social attitudes and religiosity. Religiosity and social values tend to be highly similar within the family and also show substantial variation due to the shared environment of the twins (Kendler & Myers, 2009; Polderman et al., 2015, respectively). Anthropological evidence also suggests that parents influence the mate choice of their offspring and could influence assortative mating by pushing for their child to partner with mates from, for example, a similar religious, social and economic background (for review see Buunk, Park, & Dubbs, 2008). Yet, the shared environment of the twins had a negligible impact on variation in assortative mating on these traits, suggesting minimal parental influences regarding similarity of these attitudinal variables in partnerships.

Crucially, this sample was limited in its power to dis-entangle genetic from shared environmental effects, given its small size; a larger sample and the addition of siblings in the twin model could potentially resolve this uncertainty by enhancing statistical power (Boomsma, Neale, & Dolan, 1999; Posthuma & Boomsma, 2000). It should be noted that although genetic effects, if they exist, on variation in mate choice must be small, they might nonetheless be meaningful over long periods of time. For example, Qvarnstrom, Brommer, and Gustafsson (2006) observed significant additive genetic variance in a large sample of birds accounting for less than 3% of variation in mate choice. Given the multivariate nature of mate-selection, this may in fact represent a substantial proportion of variation relative to other contributing factors.

Additionally, variance in the shared environment may have been limited in this study. This research was conducted with an Australian population over 25 years ago, and given the environmental influences on variation in traits such as social attitudes and religiosity, a sample with more varied socio-cultural environments may yet reveal larger shared environmental influences on variation in partner self-similarity on these traits.

Assortative mating remains one of the most pervasive phenomena of partner choice in human beings. We have for the first time investigated genetic influences on variation in partner self-similarity across multiple traits and found no significant independent influence of genes or the shared environment. We did, however, observe significant familial effects accounting for a small amount of the variation in partner self-similarity overall. Given the importance of relationship partner choice and the influences of assortative mating on the genetic, financial and social landscape via economic and cultural stratification, further work should be undertaken to
characterise the dominant non-familial causes of variation in individuals’ tendency to assortatively mate.
Section 7.0 General Discussion
7.1 Summary of Findings

In sections 3, 4, 5.5, and 6, of this thesis I presented studies testing evolutionary hypotheses regarding variation in human mating strategy across several domains. Key to this dissertation is the use of varied approaches to overcome previous methodological limitations associated with evolutionary psychology.

In section 3, I presented the first study of the female orgasm to test for within-female variation in orgasm frequency between different males. This method maximises the possibility of detecting consistent predictors of female orgasm that vary between men while controlling for traits that may vary between women. In this study, limited evidence was found for both sire-choice and pair-bonding models of female orgasm. Importantly, substantial variation was detected in the sexual behaviour of high- and low-orgasm males.

In section 4 I presented a second within-subjects design that demonstrates that sexually dimorphic characteristics in male faces are important in creating an impression of formidability in explicit but not implicit domains. Further, this experiment provides evidence that facial masculinity interacts with beardedness to influence explicit ratings of dominance. This is the first study to have employed a measure of implicit attitudes in the context of facial masculinity. Again, the use of a within-subjects design allowed us to detect variation in perceptions of dominance despite between-subjects variation. For example, some individuals may typically give lower ratings of dominance over male faces based on their own dominance. Nonetheless, we were able to add to the growing body of literature indicating that masculine characteristics in males are likely to have implications for intrasexual competition.

In section 5.5, I described the use of the classical twin design as a method to distinguish environmental and genetic variance and provided an example of its application to an evolutionarily relevant behavioural trait that may explain covariation in mating strategy. Finally, in section 6 I provided the first genetic analyses of variation in assortative mating in humans.

Taken as a whole, this thesis does not definitively establish concrete causes of individual differences in mating strategy. However, I have used several novel approaches to evolutionary hypotheses and demonstrated substantial potential for their further application to human mating research. Below, the outcomes of these studies are discussed in the broader context of the field. Additionally, I make specific suggestions regarding how these methods can best be applied to answer many of the remaining questions that exist in evolutionary psychology. Specifically, I pose a number of approaches to explaining variation in female orgasm, determining qualities relating to variation in facial shape preferences, linking preferences to realised mate choice, and taking advantage of advances in genotyping technology to resolve questions regarding genetic variation in fitness-relevant traits.
7.2 Re-evaluating Evolutionary Predictions Regarding the Female Orgasm

As genes are the mechanism through which evolution functions, genetic effects should often be considered when formulating evolutionary hypotheses. Where environmental causes of variation are invoked in evolutionary hypotheses, it’s important that confounding genetic effects are accounted for. For example, some genetically influenced traits are likely to correlate with environmental effects (e.g. family socioeconomic status and intelligence: Trzaskowski et al., 2014). Even in the event that correlations or experimental studies indicate environmental influences on mating strategy, genetic effects may outweigh any influence of the proposed environmental factors. Many evolutionarily relevant traits exhibit genetic variation, including those discussed in this thesis.

Female orgasm across multiple sexual contexts has been shown to vary substantially due to genetic differences between women. Using data from Australian twins, Dawood, Kirk, Bailey, Andrews, and Martin (2005) demonstrated that 31%, 37%, and 51% of the variation in female orgasm frequency during penile-vaginal intercourse, non-penile-vaginal sexual activity, and masturbation was due to genetic effects, respectively. Similar estimates have been observed when investigating difficulty reaching orgasm during intercourse (34% heritability) and when masturbating (45% heritability) (Dunn, Cherkas, & Spector, 2005). Compared to genetic effects, behavioural factors that are theoretically important for the evolutionary maintenance of the orgasm during intercourse, such as orientation to committed relationships or number of sexual partners, account for minimal variance (Zietsch, Miller, et al., 2011).

In the case that partner traits are found to be relevant to orgasm rates between women (e.g. Andersson, 1994; Gallup Jr et al., 2014; Grammer et al., 2003; Shackelford et al., 2000), these studies are potentially confounded by the fact that orgasm traits vary between women and may co-vary with a tendency to select a particular type of male as a sexual partner for other reasons. Using a within-subjects design controls for these factors, and my colleagues and I have demonstrated that when using this paradigm there is minimal support for either of the mate choice hypotheses (section 3).

One novel contribution of this study to the evolutionary psychology literature is that the sexual behaviour of males is highly important in distinguishing high- and low-orgasm males, in particular sexual communication and the manual stimulation of the clitoris. Both of these factors have been identified as important predictors of orgasm in large samples as well. Manual stimulation of the clitoris during vaginal intercourse has been found to account for a 20% increase in the frequency of orgasm at last sexual encounter in a sample of over 19,000 Australians (Richters, de Visser, Rissel, & Smith, 2006). In an even larger sample of 50,000 Americans, women who orgasmed more frequently were more likely to communicate what they wanted sexually from their partners, and were more likely to orgasm if manual clitoral stimulation was performed during
intercourse, amongst a host of other behaviours (Frederick, John, Garcia, & Lloyd, 2017). An incidental finding by Frederick et al. (2017) that presents difficulties for adaptationist hypotheses of the female orgasm is that lesbian women were more likely to orgasm during sexual activity than straight women (Frederick et al., 2017).

In order to establish an adaptive function for the observed variation in the female orgasm, evolutionary psychologists need to address several key aspects of the trait. Firstly, the relative contributions of male characteristics associated with either genetic quality or paternal investment models need be examined while controlling for differences in sexual behaviour, unless these behavioural differences are implicated by one or the other of the models (for instance, ‘good dad’ males may demonstrate greater focus on female pleasure). Secondly, if the female orgasm is maintained as a discriminatory tool for assessing the relative quality of male sexual partners, its frequency in non-heterosexual couples need be accounted for. Finally, if there is sufficient selection on the female orgasm as a mate choice tool to have maintained its current variation, it should not be as vulnerable as it appears to be to exploitation via females simply communicating their sexual preferences to male partners unless the orgasm is selecting for receptiveness to instruction. An alternative adaptationist position argues that the female orgasm may have been selected in order to reinforce sexual intercourse, which may in turn increase fitness via higher likelihood of conception and reproduction (Welling, 2014). Though orgasm is uncorrelated to reproductive advantage in modern humans (Zietsch, Miller, et al., 2011), this may have presented a significant advantage historically, maintaining the presence of the female orgasm. One avenue of consideration for future research would be to attempt to model the predicted fitness benefits this may provide and whether this advantage aligns with the substantial variation present in female orgasm.

### 7.3 Re-evaluating Approaches to the Genetic Benefits and Parental Investment Trade-off

Despite some evidence for the adaptive shifting of female preferences towards or away from masculine faces (see section 1.5), the link between testosterone and genetic quality and/or health has yet to be established (for review see Scott et al., 2013), and further there is no link between preferences for healthy males and preferences for testosterone dependent traits (Boothroyd et al., 2005; Enlow et al., 1982; Foo et al., 2017), though current health may be a poor proxy for immunocompetence. Female preferences do not appear to explain sexually dimorphic facial hair either, as, with the exception of a few cultures, women do not on average prefer hirsute men (A. F. Dixson, Halliwell, East, Wignarajah, & Anderson, 2003; B. J. W. Dixson, Dixson, Morgan, & Anderson, 2007; B. J. W. Dixson, Sulikowski, Gouda-Vossos, Rantala, & Brooks, 2016; Valentova, Varella, Bártová, Štěrbová, & Dixson, 2017). Moreover, my colleagues and I recently failed to replicate any effect of pathogen priming on facial masculinity and facial hair preferences (McIntosh
et al., 2017). As explored above, section 3 also failed to find consistent effects of testosterone-dependent traits on female orgasm, though these traits were not measured directly.

Though women may not, on average, prefer highly masculine faces, there are of course women who do favour masculine men (e.g. Burt et al., 2007; DeBruine, Jones, Tybur, Lieberman, & Griskevicius, 2010; Penton-Voak & Perrett, 2000; Penton-Voak et al., 1999; Waynforth et al., 2005). In such cases it is important to account for all possible causes of this variation, and not exclusively environmental factors. As in the case of female orgasm, genes have been shown to cause a substantial portion of variation in women’s preferences for facial masculinity and facial hair. As much as 38% the variation in women’s preferences for facial hair has been shown to be due to genetic effects (Verweij, Burri, et al., 2012). Moreover, genetic effects on variation in facial masculinity preferences have been investigated in the context of theoretically meaningful environmental effects. Whereas hypothetically important environmental factors accounted for less than 1% of variance in females’ preferences for facial masculinity, my colleagues and I showed that genetic effects accounted for nearly 40% of the variation (Zietsch, Lee, et al., 2015).

Although attractiveness ratings of masculine faces are highly variable, there is consistent evidence that highly masculine faces are considered more aggressive and more dominant (Boothroyd et al., 2007; DeBruine et al., 2006; Keating et al., 1981; Perrett et al., 1998; Spisak et al., 2012; Swaddle & Reierson, 2002). In section 4, we demonstrated that enhancing the masculinity of a male face significantly increased its perceived dominance. Additionally, we showed that facial hair had a similar effect, significantly increasing perceived dominance. Furthermore, we found that facial masculinity interacted with facial hair such that the relative contributions of facial masculinity to perceived dominance decreased as facial hair increased. That is: as facial hair increases, facial masculinity becomes less impactful on ratings of dominance. Consequently, beards may be used to augment visible cues of dominance in absence of highly masculine facial physiognomy. This adds to the growing body of literature that suggests that sexually dimorphic facial characteristics in human males may be the result of intrasexual selection, rather than female choice (Archer, 2009; Puts, 2010, 2016; Puts et al., 2015). Although increased perceptions of dominance may be beneficial by decreasing the likelihood of competitive advances by male conspecifics, the same visual cues are likely be harmful to intersexual selection via female choice, especially given the traits associated with higher levels of testosterone such as lower likelihood of being in a committed relationship (Alvergne et al., 2009; Muller et al., 2009; van Anders & Watson, 2006) and increased likelihood of infidelity and partner violence (Booth & Dabbs, 1993).

As such, it is possible that variation in female preferences for masculine facial traits is balanced by the advantages of partnering with a socially dominant male and the behavioural costs that may be associated with such a male, rather than a trade-off between genetic benefits and
parental investment. Further doubt is shed on the trade-off between genetic quality and parental investment in males when consulting the behavioural genetics literature, as a number of parental investment traits are also heritable and would therefore confer genetic benefits to offspring, blurring the distinction between the two aspects of the model. For example, secure attachment styles are substantially heritable, as is prosocial behaviour, parental warmth, and trustworthiness (for review see Ebstein, Israel, Chew, Zhong, & Knafo, 2010). More problematic for this model is that the same genes predisposing male offspring to develop facially masculine traits also lead females to develop facially masculine characteristics, decreasing their attractiveness, and subsequently, their reproductive fitness countering any benefits associated with ‘good-genes’ (Lee, Mitchem, et al., 2014). Any signal of genetic quality ought actually be associated with genetic benefits to offspring in order to be maintained. However, masculinity may actually be associated with genetic costs.

A prevailing question that remains in this research area is what does linearly predict facial attractiveness if facial masculinity does not? Evolutionary approaches to the attractiveness of human faces, and variation therein, propose links between features that are found to be preferred and an adaptive outcome such that attractiveness signals some inherent quality about a mate (Thornhill & Gangestad, 1999). Facial masculinity is one of the most prominently studied aspects of research in attractive male qualities, but there are a number of extant hypotheses that focus on distinct aspects of facial morphology that are putative indicators of genetic quality.

Symmetry of the face is one component of attractiveness that has been studied widely as a signal of developmental stability (i.e., genetic quality) and some studies have found that facial symmetry is preferred over asymmetry (for review see Little, Jones, et al., 2011). However, evidence for a relationship between facial asymmetry and indices of health in humans is at best mixed. Asymmetrical facial shape is positively related to self-reported respiratory disease (Thornhill & Gangestad, 2006) but few other human studies demonstrate any links between symmetry and health. In fact, a large study of English adolescents using geometric analysis of 3-dimensional facial scans found no relationship between fluctuating asymmetry and childhood health (Pound et al., 2014). It therefore seems unlikely that symmetry is an accurate signal of genetic quality or developmental stability in humans, though further research remains to be done. Facial averageness (i.e., how typical a face appears relative to other faces in the population) has also been proposed as an indicator of genetic quality (Thornhill & Gangestad, 1993). Unlike facial symmetry, facial averageness has been linked to actual measures of health in humans (Rhodes et al., 2001). In order for symmetry and averageness to function as signals of genetic quality, they need be a) heritable and b) genetically correlated to attractiveness. This would provide basis for the link between underlying genetic quality that causes averageness and or symmetry (e.g. developmental stability) and ratings of attractiveness (i.e., the perception of the signal). As with the case of facial
masculinity, there appears to be insufficient evidence to suggest that symmetry or averageness function in such a way.

In the case of facial symmetry, several studies of facial morphology have observed that asymmetry of facial features does not seem to be the product of genetic effects (Djordjevic, Jadallah, Zhurov, Toma, & Richmond, 2013; Djordjevic, Zhurov, & Richmond, 2016). Two twin studies of 3D facial shape data have found that asymmetry is primarily driven by residual sources of variance, rather than the common environment or genes (Djordjevic et al., 2013; Djordjevic et al., 2016). If asymmetry is not heritable, it is highly unlikely to be a signal of genetic quality. In contrast, facial averageness does appear to have a genetic basis. Lee et al. (2016) computed a measure of facial averageness in a large twin sample (N = 1,823) by first taking geometric landmark information for regions of the face that vary between individuals. Each individual’s landmark data was then compared to the mean of the sample while controlling for symmetry. Averageness data was then subjected to twin modelling and was found to be heritable in both males (27% of variation) and females (22% of variation). Each individual was also rated for facial attractiveness. Averageness, as predicted by theory, was also phenotypically positively correlated with attractiveness. However, this relationship did not seem to be the result of facial shape. Facial shape components associated with ratings of attractiveness did not mediate the relationship between facial averageness and overall attractiveness for males or females. Furthermore, genetic analysis of the covariation between facial attractiveness and facial averageness found no shared genetic basis to the two traits. Specifically, the genes that impact facial attractiveness are not the same genes that contribute to facial averageness. Consequently, preferences for facial averageness may not represent preferences for genetic quality, but rather sensory bias towards images that are prototypical (Halberstadt & Rhodes, 2000, 2003; Lee et al., 2016). However, due to power constraints to detect small genetic effects and some limitations associated with the photographs used in the study, confirmatory research is required.

One potential aspect of facial attractiveness that is yet to be fully explored is the relationship between desirable mate characteristics and facial morphology. For example, intelligence is a highly valued trait in mate choice, possibly as an indicator of genetic quality (Haselton & Miller, 2006; Miller, 2000). Although intelligence can be signalled in multiple different ways, such as through verbalisation (e.g. D. Reynolds, Arcy, & Gifford, 2001), there is some evidence to suggest that intelligence may be signalled somewhat accurately through facial shape (Carney, Colvin, & Hall, 2007; Zebrowitz & Rhodes, 2004; though see Borkenau & Liebler, 1995; Olivola & Todorov, 2010). Lee et al. (2017) used measured intelligence (IQ), perceived intelligence from photographs, and facial morphometric data from twins to test for a shared genetic basis to the relationship between perceived and actual intelligence. Both measured and perceived intelligence were heritable
(77% and 37% respectively), though no shared genetic variation was detected. However, significant familial covariation was detected (i.e., combined genetic and common environmental effects) suggesting the researchers lacked statistical power to differentiate small but potentially meaningful genetic correlations between actual and perceived intelligence, potentially via aspects of facial shape.

Future research should continue in this vein by testing for potential associations between facial morphology and highly desired mate characteristics. Using twins enables researchers to explore the potential genetic underpinnings of this relationship. One recent attempt has found a significant genetic relationship between facial trustworthiness and morphometric components of facial attractiveness (Lee, Wright, Martin, Keller, & Zietsch, in press). In a study of 1320 twins, heritable components of rated facial trustworthiness were genetically related to ratings of facial attractiveness. It is possible, however, that this association was driven by halo effects (Lee et al., in press). Nonetheless, each previously studied trait only contributes a small proportion of variation to attractiveness. Facial averageness, for example, only accounted for 1% and 3% of the variation in male and female attractiveness ratings in a large sample of twins (Lee et al., 2016). Consequently, future studies should employ large studies of twins investigating multiple mating-relevant traits at once (e.g. intelligence, athleticism, extraversion, etc.). This would enable testing for a genetic correlation between all traits associated with genetic quality and facial attractiveness, as well as quantifying the specific facial morphometrics that may mediate this relationship. Previous twin studies have also typically relied on 2D imaging, which may increase error in ratings of participant’s rates or in analysing morphometrics, and which in turn would reduce the capacity to detect genetic effects. More recent studies of the genetics of facial shape have used 3D scans (e.g. Djordjevic et al., 2013; Djordjevic et al., 2016), which adds an additional dimension in which to detect meaningful covariation in perceived characteristics and facial morphology. Furthermore, it would be beneficial for studies of immune function and health in relation to mate to take place in a diverse array of environments in which modern medicine, infrastructure, and resource abundance are less likely to influence results.

7.4 The Relationship Between Mating Strategies and Mate choice

As with preferences for facial shape, twin studies have demonstrated a wide range of heritability estimates for mate preferences across various different traits (e.g. Verweij, Burri, et al., 2012, 2014; Zietsch et al., 2012). However, genetic influences on mate preferences may not necessarily translate into choice of mate. In contrast to mate preferences, genetic effects appear to have a minimal impact on mate choice. Indeed, Zietsch, Verweij, et al. (2011) detected near-zero genetic influences on mate choice across numerous traits including physical characteristics (body mass index and height), social attitudes, religiosity, education and income, age, and personality in a
sample of several thousand Australian twins and their partners. In section 6, my colleagues and I used data from the same sample to test for genetic effects on variation in assortative mating, the most prevalent pattern of mate choice in humans. Over all of the studied traits, combined familial effects only accounted for between 5 and 9% of the variation in assortative mate choice, though we lacked the statistical power to further partition variance due to common environment and genes.

An argument could be made that genetic influences on mate choice would not be expected to be large in humans, given the dynamic, cultural market place in which mate choice takes place. For instance, an individual’s religion may prevent them from choosing a romantic partner who does not share their faith. Although an individual’s tendency to be religious is heritable (Bouchard, McGue, Lykken, & Tellegen, 1999; Jakubowska & Oniszczenko, 2010; Waller, Kojetin, Bouchard, Lykken, & Tellegen, 1990; Zietsch, Verweij, et al., 2011), the specific religion with which an individual identifies will likely be determined by other factors such as where they grow up and the religion of their parents (whose own tendency to be religious, but not their faith of choice, will also be genetically influenced). Yet, the incongruence of genetic influences on mate preferences and near-zero genetic influences on mate choice is not unique to humans. Similar results have been obtained from studies of pair bonding bird species. Substantial estimates of genetic effects have been observed for mate preferences (Schielzeth, Bolund, & Forstmeier, 2010), whereas the heritability of mate choice is near-zero (Hegyi et al., 2010; Qvarnstrom et al., 2006).

There are numerous reasons why, despite genetic influences on mate preference, researchers fail to detect large genetic influences on realised mate choice. The first is that the nature of preferences and choice differ in one obvious respect. Preferences are unrestrained and therefore genetic influences can be maximally expressed. In contrast, mate choice depends on mutuality of interest and availability, which may be influenced by numerous other forces. Mutual mate choice is poorly understood in humans due to its underlying complexity: at a minimum, understanding mutual mate choice requires simultaneously integrating all individuals’ preferences, their potential partners’ traits with reference to these preferences, their potential partners’ preferences, and their own traits with reference to potential partners’ preferences. Consequently, most attempts to understand mate choice typically employ a model under which males compete while females choose (for review see Stewart-Williams & Thomas, 2013). Further complicating the translation of preferences to choice is that mate selection rarely functions via a single trait. Although individual traits have of course been found to be important across multiple cultures (e.g. height, intelligence, beauty: Buss, 1989), mate selection is multivariate, often involving interactions between traits (e.g. Lee, Dubbs, von Hippel, Brooks, & Zietsch, 2014). Moreover, given that individuals will vary in mate value based on their own characteristics (such as attractiveness, kindness, intelligence, earning potential, etc.), it is unlikely that they will be able to attain a mate that is as high in mate value as
they desire. Consequently, individuals will have to engage in some satisficing behaviour whereby mate choice is constrained by optimisation across all of a potential partner’s traits and their weighted importance.

Modern approaches to multivariate mate selection have attempted to identify the sorting algorithms that predict mate choice by using simulated models and comparing these results with data obtained from real couples (e.g. Conroy-Beam & Buss, 2016a, 2016b, 2017; Conroy-Beam, Goetz, & Buss, 2016). Conroy-Beam and Buss (2016b) compared the success of seven competing mate choice algorithms designed to integrate varying preferences across 23 different traits in agent-based simulations over 200 generations. The most successful algorithm was a calculation of the Euclidean distance between an individual’s trait preference levels over all traits and potential mates’ characteristics over all traits. The Euclidean algorithm essentially calculates the shortest possible distance in multidimensional space between all of an individual’s preferences and the actual levels of those traits across multiple potential mates. This algorithm also appears to capture some of the relationship between preference and actual choice in human couples, as long-term mates tend to fall close in multidimensional space to their partner’s preferences (i.e. a shorter Euclidean distance: Conroy-Beam & Buss, 2016b). Moreover, using Euclidean distance as a metric of mate-value (i.e., the multidimensional difference between a mate and an individual’s desired traits) can predict relationship satisfaction (Conroy-Beam et al., 2016) and attraction to potential mates (Conroy-Beam & Buss, 2017). Predictors of an individual’s Euclidean distance, both in the context of their own mate value and in the context of potential mates, are currently unknown. One possible application of the classic twin design to mate choice would be to calculate the heritability of individual Euclidean values across multiple traits, in essence quantifying genetic causes of variation in an individual’s ideal mate across all possible traits rather than studying individual traits.

7.5 A Genetically Informed Perspective on Causes of Individual Differences in Mating Strategy

A consistent theme of this thesis has been a failure of evolutionary psychology to take into account the influence of genes on behavioural variation as it relates to mating strategy. A substantial portion of this may be due to mistaking genetic effects as environmental influences on behaviour. For example, a link has been proposed between father absence in early childhood and a predisposition towards short-term sexual relationships in women (Belsky, Steinberg, & Draper, 1991). The reasoning in this case is that the early life environment calibrates the motivational and physical (via early pubertal maturation) pathways of sexual development. Father absence is presumed to be an indicator of an unstable and unreliable environment in which resource provision
and relationships are likely to be inconsistent. This in turn motivates sexual strategies that maximise short-term opportunities (Belsky et al., 1991; Belsky et al., 2007; Draper & Harpending, 1982). Consistent with this hypothesis, there is substantial evidence that daughters raised without their biological father typically engage in sexual intercourse at an earlier age and are more likely to fall pregnant in their teens (Ellis et al., 2003; Hogan & Kitagawa, 1985; Kiernan & Hobcraft, 1997; Newcomer & Udry, 1987; Quinlan, 2003; Wight, Williamson, & Henderson, 2006).

As in the case of facial masculinity preferences, genes may provide a more compelling causal pathway than environmental calibration or, at the very least, may obscure the relationship between environment and behaviour. A number of behaviours that may contribute to father absence have been demonstrated to have genetic components. For instance, impulsivity (Anokhin, Golosheykin, Grant, & Heath, 2011; Anokhin, Grant, Mulligan, & Heath, 2015), sociosexual orientation (Bailey et al., 2000), number of sexual partners (Zietsch et al., 2008), and infidelity (Zietsch, Westberg, Santtila, & Jern, 2015) have all been shown to be substantially influenced by genes. Furthermore, there are genetic influences on sexual development, including age of first intercourse (Dunne et al., 1997; Mustanski, Viken, Kaprio, Winter, & Rose, 2007; Rowe, 2002) and age of first pregnancy (Waldron et al., 2007). Therefore, the relationship between father’s absence and daughter’s sexual behaviour may be the result of shared genes between the two, rather than the effect of the environment created by their father’s behaviour.

To control for the passive correlation between the genes of parents and the environment they shape via parents’ behaviour, Mendle et al. (2009) analysed data collected from the children of sister dyads. By comparing the similarity of children of sisters at varying levels of genetic relatedness (e.g. half-siblings, full siblings, twins), researchers are able to make inferences about the independent contribution of genes and the environment (Dick, Johnson, Viken, & Rose, 2000). For instance, if environmental conditions (i.e., father absence) vary across the children of siblings, but the behavioural outcome does not, presumably shared genes between the siblings and their offspring are responsible for the behavioural similarity of the offspring. Using such a model has revealed that shared genes between absent fathers and their daughters are more likely to be responsible for the latter’s early sexual maturation on a physical basis (i.e. age of menarche: Mendle et al., 2006) and sexual interest (i.e. age at first intercourse Mendle et al., 2009).

Further adaptationist arguments have been made regarding exposure to unpredictable and uncontrollable environments and a corresponding shift towards present oriented behaviour (Pepper & Nettle, 2017). Specifically, Pepper and Nettle (2017) make the claim that lower socioeconomic environments also contain cues to external mortality risk (i.e., risk of death from external sources such as violence) which cause changes in psychology in order to motivate ‘adaptive’ short-term strategies (e.g. earlier reproduction is adaptive in an environment where the future is uncertain).
However, my colleague and I have pointed out that many of the factors considered to result from lower socioeconomic environments are heritable, as is socioeconomic status itself (Sherlock & Zietsch, in press-a). Consequently, genes that influence parents’ short-term strategies will influence the behaviour of their children (via genetic inheritance) and also their environment.

When genetic causes of variation are invoked in explaining variation in behaviour, the case is often made for reactive heritability. That is, one trait is adaptively linked to another that exhibits heritability. For example, heritable variation in extraversion has been proposed to result from adaptive calibration in other traits predicting an individual’s success in social domains that are themselves heritable, namely physical strength, formidability and attractiveness (Lukaszewski & Roney, 2011). Supporting this hypothesis, phenotypic correlations have been observed between physical strength and attractiveness and extraversion (Haysom et al., 2015; Lukaszewski & Roney, 2011; von Rueden, Lukaszewski, & Gurven, 2015). To appropriately test this hypothesis, however, requires the use of genetic data.

The application of genetic modelling techniques to evolutionary psychology hypotheses lies at the centre of an emerging field known as evolutionary behavioural genetics (Zietsch, de Candia, & Keller, 2015). Using genetically informative data from large samples of both related and unrelated individuals, researchers are able to test hypothesis regarding genetic covariation between traits such as those proposed by Lukaszewski and Roney (2011). Under the reactive heritability hypothesis, the heritable variation detected in extraversion (Johnson, Vernon, & Feiler, 2008; Polderman et al., 2015) is actually the consequence of genetic factors underlying traits to which extraversion is calibrated (e.g. physical attractiveness). Using twin data, Haysom et al. (2015) demonstrated a) a lack of phenotypic correlations between height or BMI and attractiveness (both of these traits are important predictors of attractiveness in men and women respectively) and b) a lack of genetic covariation between facial attractiveness and extraversion. This indicates a unique genetic basis underlying variation in both attractiveness and extraversion that runs directly counter to a reactive heritability account for trait variation.

Evolutionary behavioural genetics studies suggest that two high profile cases of facultative calibration, in the form of facial masculinity preferences and extraversion, are unlikely to account for the observed trait variation (Haysom et al., 2015; Zietsch, Lee, et al., 2015). There are further theoretical reasons to suspect that facultative calibration is unlikely to account for a significant portion of the variation in human mating behaviour (for review see Zietsch, 2016). Genetic variation accounts for, on average, half the variation in measured human traits (Polderman et al., 2015) and genome wide association studies of these traits have further indicated that genetic variation is typically the result of many, many genes of small effect (Chabris et al., 2015). Given that a large proportion of our genome is expressed in the brain (84%: Hawrylycz et al., 2012),
psychological traits are likely vulnerable to genetic mutations (Zietsch, 2016). To the extent that these mutations accumulate and substantially harm fitness, they are likely to be selected out of the population (Eyre-Walker, 2010), but in their benign form may simply contribute to ‘noise’ (i.e., behavioural variation) in the system\(^2\) (Keller & Miller, 2006; Lande, 2007; Zhang & Hill, 2005). Recent analyses of genomic data support genetic predictions under such a model, referred to as mutation-selection balance, at least in the case of personality variation (Verweij, Yang, et al., 2012).

**7.5.1 Further understanding genetic causes of variation.** Understanding that fitness-relevant traits are heritable is in and of itself an important milestone in understanding human evolution. The precise causes of this variation remain an unanswered question within psychology and science more broadly. At the heart of the solution to this Darwinian paradox is the field of evolutionary behavioural genetics (Zietsch et al., 2014). Evolutionary behavioural genetics has already been applied to several facultative calibration hypotheses (Haysom et al., 2015; Zietsch, Lee, et al., 2015) but further testing remains to be done. For instance, Al-Shawaf et al. (2014) have proposed an adaptive relationship whereby sociosexual orientation calibrates sexual disgust in order to facilitate greater or fewer sexual encounters. Both disgust sensitivity (section 5.5) and sociosexual orientation (Bailey et al., 2000) have been shown to vary genetically. However, the genetic covariation between the traits, which is predicted by a facultative calibration hypothesis, has not been established. Similarly, pathogen disgust is hypothesised to have evolved in order to mitigate the costs of contact with disease vectors (Tybur et al., 2009) and thus may be reactively heritable to actual immune function. That is, individuals with worse immune systems may have more sensitive disgust responses to possible pathogens, and this relationship may be the result of facultative calibration (see section 5.5).

A major weakness in the current literature regarding the genetics of mating strategy is the apparent disconnect between heritable preferences and non-heritable mate choice. There are several possible approaches to this problem that follow from section 7.4. The first is to investigate the heritable basis of the Euclidean distance between individuals’ preferences and eventual mate choices. It is likely that individuals vary in their willingness to settle for mates that do not precisely match their ideal preferences (i.e., a greater Euclidean distance between their ideals and their possible mates). To some extent, this will function as a result of an individual’s own mate value via their own characteristics (e.g. attractiveness, earning potential, BMI). Another possible contributor to multivariate mate choice is an individual’s choosiness or willingness to settle, independent of his or her own mate value. For instance, some individuals with high mate value may be capable of

\(^2\) The idea of genetic variation as ‘noise’ is consistent with a proposal by Tooby and Cosmides (1990), though it has been largely neglected in subsequent research, which tends to favour adaptationist explanations of variation.
attaining other high value mates, but nonetheless select individuals who are lower in mate value. This might occur for a number of reasons; one being a fear that another high value mate may be better able to have an affair. Another important advance to be made in understanding the genetic basis of mate choice is to establish the stability of preferences over time. Following from this, genetic influences on variation in mate choice may be revealed by studying the similarity of different selected mates over time. Although genes may not account for a significant proportion of variation in mate choice on any given trait at one point in time, they may nonetheless contribute to variation in the stability of mate choice (an individual’s ‘type’ for instance) over multiple time points.

Moreover, even though the genetic effects underlying variation in mate choice appear to be small, the genetic consequences of mate choice can be profound. For instance, mating assortatively and the repeated couplings of individuals whose heritable traits are more alike than expected by chance can produce genetic linkage between traits. This can occur when two heritable traits are positively associated with attractiveness, which tends to be similar between mates. This causes positive covariance between alleles across the two traits, such that alleles that cause both traits to be higher (and therefore linked to greater attractiveness) are more likely to co-occur together (Lynch & Walsh, 1998) called gametic phase disequilibrium. Assortative mating has been shown to be partially responsible for the positive genetic correlation between intelligence and height (Keller et al., 2013). The consequences of assortative mating have not yet been fully explored in the context of other traits, which tend to be similar between partners, including social attitudes and religiosity. For religious individuals, religiosity is likely to positively scale with attractiveness. Similarly, non-religious people are likely to find non-religious people more attractive. In the same vein, conservatism is likely more attractive in a partner to individuals who are themselves conservative and vice versa for left wing or liberal ideals. There is a robust phenotypic relationship between religiosity and conservatism (Guth, Kellstedt, Smidt, & Green, 2006; N. J. Kelly & Morgan, 2008; Layman & Carmines, 1997; Layman & Green, 2006; L. R. Olson & Green, 2006) and as both religiosity (Bouchard et al., 1999; Waller et al., 1990; Zietsch, Verweij, et al., 2011) and political orientation (Alford, Funk, & Hibbing, 2005; Eaves et al., 1999; Martin et al., 1986) are heritable, assortative mating may have produced positive genetic covariance between the two traits. Shared genetic variation is typically the result of two mechanisms (which are not exclusive): firstly, pleiotropic effects of genes may influence both traits via a common predisposition (for instance towards traditionalism), or the traits may have become genetically linked via assortative mating producing repeated pairings between alleles increasing religiosity and alleles increasing conservatism. Twin studies with the addition of parental and sibling data could be used to
distinguish these sources of any potential genetic covariation between conservatism and religiosity, as well as other traits that may potentially covary as a result of assortative mating.

7.5.2 Using the entire genome to inform evolutionary psychology. Although twin studies have tremendous power to identify genetic causes of variation in a trait, they are less able to explain why genetic variation has been maintained. Three main hypotheses exist as to how genetic variation in complex traits is maintained but twin data are unfortunately insufficient to test the competing predictions generated by these theories. However, in light of increasingly sophisticated and inexpensive genotyping technology, behavioural geneticists now have greater resolution to test evolutionary predictions at the level of individual nucleotide bases (Zietsch et al., 2014). The first theory, mutation-selection balance, is described briefly above in section 7.5. The second theory as to how genetic variation is maintained is referred to as selective-neutrality or neutral-mutation drift. Under this model, genetic variation in a trait is the result of benign mutations arising at many different loci. Over time, these mutations become fixed (i.e., don’t vary between individuals) or drift out of the population and are eliminated. The final model is called balancing selection. Balancing selection is not the process by which variation is maintained necessarily, but is rather the consequence of a number of other processes under which variation is selected for rather than against. For example, the optimal level of a trait may vary over time and location or even between sexes, which balances genes that increase or decrease levels of the trait.

Each of these models makes different predictions about the genome, and specifically about the number of causal variants (polymorphisms that influence the trait), their frequency in the population, and the amount of variation caused by interactions between these causal variants. To date these methods have been used to investigate variation in personality (Verweij, Yang, et al., 2012), the consequences of inbreeding (Verweij, Abdellaoui, et al., 2014) and evolutionary history of schizophrenia (Keller et al., 2012) and have yielded impressive explanatory power. However, almost all human traits vary genetically (Polderman et al., 2015) and there is abundant opportunity to test for the causes of this variation. In particular, why do preferences vary genetically? Mate preferences almost certainly have consequences for fitness in as much as they influence the reproductive rate of some individuals over others. For instance, preferences for common traits or trait levels near the mean of the population may be more likely to be fulfilled due to higher availability. Alternatively, traits that are preferred at a lower frequency may be easier to attain in a mate due to fewer competitors also seeking this trait. As such, genetic variation underlying mate preference variation is unlikely to reflect selective neutrality or drift. However, variation in mate preferences may reflect balancing selection to some degree. For example, facially masculine males are consistently viewed as being more dominant (see section 4), which may increase the likelihood of success in competition with other males. In contrast, the genes that influence facial masculinity
are harmful for females’ attractiveness (Lee, Mitchem, et al., 2014). Consequently, the genes underlying preferences for facial masculinity may be balanced by the benefits of mating with a dominant male contrasting with the potential costs to fitness of female offspring. Under balancing selection, causal variants should be maintained at a high frequency (Curtsinger, Service, & Prout, 1994; Kopp & Hermisson, 2006; Mani, Clarke, & Shelton, 1990; Penke, Denissen, & Miller, 2007; Turelli & Barton, 2004). Under a low frequency of causal variants, balancing selection is inefficient and unstable because alleles that actually have an effect on the trait are rare in the population. Additionally, selection acting directly on the trait is likely to maintain variation at a smaller number of loci per trait (Barton & Keightley, 2002; Burger, 2000; Curtsinger et al., 1994; Kopp & Hermisson, 2006; Turelli & Barton, 2004). Without the use of genomic data, these hypotheses will be impossible to address.

### 7.6 Implications for the Future of Evolutionary Psychology

From the evidence contained in this thesis and the literature reviewed therein, a number of recommendations can be made for future researchers in the field of evolutionary psychology who wish to further understand variation in human mating strategies. Specifically, the importance of biologically informed theorising cannot be understated. Given that genes are the mechanism by which evolution functions, it is perhaps surprising how little attention is paid to the genetic literature in evolutionary psychology (e.g. Section 1.5 and 1.7). Furthermore, numerous theories in evolutionary psychology have been forwarded without convincing evidence of certain underlying biological assumptions. For example, causal links between testosterone exposure and facial shape were only established as recently as 2015 (Whitehouse et al.). Further, this study only observed links between prenatal testosterone exposure, and not adult testosterone exposure, and masculine facial shape. Yet theories regarding testosterone, facial masculinity, and genetic quality have existed since 1992 (Folstad & Karter) and there is still no convincing evidence that facial masculinity is linked to genetic quality.

One solution to this issue is consulting the biological literature, for instance behavioural genetics, where available. Researchers who are interested in explaining trait variation caused by environmental factors could potentially cross-reference the phenotype of interest with a recent meta-analysis of all twin studies conducted up to that point (Polderman et al., 2015) in order to establish whether genes may play a substantial role. Polderman et al. (2015) provide an online tool that allows researchers to search for heritability estimates of specific phenotypes. With regard to this point, twin studies allow for both the quantification of genetic and environmental influences on trait variation. The shared environment (i.e., SES, neighbourhood, aspects of the home) has been found to be far less influential in causing likeness between siblings than would be expected over a range of traits, with identical twins correlating on average at twice the magnitude of non-identical
twins suggesting almost no effect (Polderman et al., 2015). In light of this finding, a number of theoretically meaningful environmental factors (e.g. father absence: Belsky et al., 1991) are unlikely to cause substantial variance independently of genes (Mendle et al., 2009; Mendle et al., 2006). To this end, researchers should avoid making causal arguments in the presence of passive gene-environment confounds (such as those between trait impulsivity and environmental unpredictability) unless controlling for this confound with familial data (for review see McAdams et al., 2014).

In the event that causal arguments are being made regarding environmental influences on trait variation, the hypothesised environmental factors should be studied in the context of genetic influences (e.g. Zietsch, Lee, et al., 2015; Zietsch, Miller, et al., 2011). An alternative approach is to return to simple experiments in which randomisation of participant assignment between conditions controls for genetic variation. Finally, when genetic effects are known to be present, but the input of the environmental is considered to be theoretically important, within-subjects designs maximise the capacity to detect meaningful variation while also controlling for between-subjects differences (e.g. section 3).

7.7 Conclusion

In this thesis I have tested a number of evolutionary hypotheses regarding individual differences in mating strategy. However, much remains to be done in the way of understanding how humans vary in their approach to mating, the causes of this variation, and the implications of this variation for personal, reproductive, and social outcomes. In section 7, I reviewed my findings in light of the broader literature and make recommendations for the future of the field. Specifically, I recommend a greater understanding and engagement with behavioural genetic literature. I predict this will better enable scientists to a) design studies that avoid gene-environment confounds and maximise the opportunity to observe environmental variation in the presence of heritable effects and b) take advantage of technological advancements to appropriately test evolutionary hypotheses down to the level of individual nucleotide bases.

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3 Because C and D cannot be estimated at the same time, this statistic should be interpreted with caution as non-additive genetic effects (D) will decrease estimates of the shared environment (C) in an ACE model (see section 5.2).
References


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physical and psychological human traits. *PloS one*, 9(7), e103102. doi:10.1371/journal.pone.0103102


Appendix A. Section 6 Supplemental Materials

Supplementary Table I. Couple similarity scores correlated with partner trait scores used in Zietsch et al., (2011)

<table>
<thead>
<tr>
<th>Trait</th>
<th>Males (N)</th>
<th>Females (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI</td>
<td>.27*** (767)</td>
<td>.28*** (1400)</td>
</tr>
<tr>
<td>Height</td>
<td>.21*** (812)</td>
<td>.13 *** (1508)</td>
</tr>
<tr>
<td>Education</td>
<td>-.15*** (1751)</td>
<td>.22*** (3703)</td>
</tr>
<tr>
<td>Income</td>
<td>-.22*** (1338)</td>
<td>-.11*** (2396)</td>
</tr>
<tr>
<td>Religiosity</td>
<td>-.20*** (1743)</td>
<td>.41*** (3647)</td>
</tr>
<tr>
<td>Attitudes</td>
<td>.01 (804)</td>
<td>.03 (1518)</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>.20*** (824)</td>
<td>.03 (1538)</td>
</tr>
<tr>
<td>Extraversion</td>
<td>-.20*** (815)</td>
<td>-.30*** (1527)</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>.05 (827)</td>
<td>.01 (1534)</td>
</tr>
<tr>
<td>Harm avoidance</td>
<td>.17*** (820)</td>
<td>.04 (1520)</td>
</tr>
<tr>
<td>Novelty seeking</td>
<td>.00 (813)</td>
<td>.03 (1511)</td>
</tr>
<tr>
<td>Reward dependence</td>
<td>-.07* (820)</td>
<td>-.10*** (1519)</td>
</tr>
<tr>
<td>Persistence</td>
<td>-.01 (817)</td>
<td>-.22*** (1518)</td>
</tr>
<tr>
<td>Age</td>
<td>-.03 (2120)</td>
<td>.04* (4257)</td>
</tr>
</tbody>
</table>

*p < .05. **p < .01. ***p < .001
**Supplementary Table II.** Mean difference† between twins and their partners and partner correlations for each trait split by sex

<table>
<thead>
<tr>
<th>Trait</th>
<th>Number of couples</th>
<th>Mean couple difference†</th>
<th>Partner Correlation❖ (r)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male twins</td>
<td>Female twins</td>
<td>Male twins</td>
</tr>
<tr>
<td>Body mass index</td>
<td>772</td>
<td>1430</td>
<td>1.29</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>813</td>
<td>1513</td>
<td>1.77</td>
</tr>
<tr>
<td>Education</td>
<td>1975</td>
<td>4187</td>
<td>1.21</td>
</tr>
<tr>
<td>Income</td>
<td>1489</td>
<td>2661</td>
<td>2.12</td>
</tr>
<tr>
<td>Religiosity</td>
<td>2012</td>
<td>4171</td>
<td>.54</td>
</tr>
<tr>
<td>Attitudes</td>
<td>805</td>
<td>1522</td>
<td>1.21</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>825</td>
<td>1544</td>
<td>1.32</td>
</tr>
<tr>
<td>Extraversion</td>
<td>815</td>
<td>1527</td>
<td>1.35</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>828</td>
<td>1539</td>
<td>1.23</td>
</tr>
<tr>
<td>Harm Avoidance</td>
<td>821</td>
<td>1525</td>
<td>1.33</td>
</tr>
<tr>
<td>Novelty Seeking</td>
<td>818</td>
<td>1525</td>
<td>1.33</td>
</tr>
<tr>
<td>Reward Dependence</td>
<td>821</td>
<td>1524</td>
<td>1.31</td>
</tr>
<tr>
<td>Persistence</td>
<td>818</td>
<td>1523</td>
<td>1.35</td>
</tr>
<tr>
<td>Age</td>
<td>2120</td>
<td>4258</td>
<td>1.05</td>
</tr>
</tbody>
</table>

†Mean difference represents the average absolute difference between twins and their partners on trait score for Education, Income and Religiosity (because they are ordinal measures), and difference in standard deviations of trait for the other measures.

❖Partner correlations do not include parents of twins, unlike those reported in Zietsch et al. (2011) and as a result correlations differ slightly in this sample. Though some correlations differ between males and females, these are qualitative and not significant – refer to Methods for further explanation.

* $p < .05$

*** $p < .001$
**Supplementary Table III:** Proportion of variance accounted for by additive genetic (A), shared environmental (C), and residual (E) effects on the tendency to assortatively mate for unadjusted individual traits.

<table>
<thead>
<tr>
<th>Trait</th>
<th>A (95%CI)</th>
<th>C (95%CI)</th>
<th>Familial effects [A + C] (95%CI)</th>
<th>E (95%CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index</td>
<td>.00 (.00, .21)</td>
<td>.22 (.02, .31)</td>
<td>.22 (.12, .31)</td>
<td>.78 (.69, .88)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>.07 (.00, .20)</td>
<td>.02 (.00, .18)</td>
<td>.09 (.00, .20)</td>
<td>.91 (.80, 1.00)</td>
</tr>
<tr>
<td>Education</td>
<td>.07 (.00, .19)</td>
<td>.07 (.00, .16)</td>
<td>.13 (.08, .19)</td>
<td>.87 (.81, .92)</td>
</tr>
<tr>
<td>Income</td>
<td>.08 (.00, .14)</td>
<td>.00 (.00, .11)</td>
<td>.08 (.01, .14)</td>
<td>.92 (.86, .99)</td>
</tr>
<tr>
<td>Religiosity</td>
<td>.11 (.00, .16)</td>
<td>.00 (.00, .12)</td>
<td>.11 (.05, .16)</td>
<td>.89 (.84, .95)</td>
</tr>
<tr>
<td>Attitudes</td>
<td>.08 (.00, .20)</td>
<td>.00 (.00, .15)</td>
<td>.09 (.00, .20)</td>
<td>.91 (.80, 1.00)</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>.04 (.00, .17)</td>
<td>.03 (.00, .15)</td>
<td>.06 (.00, .17)</td>
<td>.94 (.83, 1.00)</td>
</tr>
<tr>
<td>Extraversion</td>
<td>.03 (.00, .15)</td>
<td>.00 (.00, .12)</td>
<td>.03 (.00, .15)</td>
<td>.97 (.85, 1.00)</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>.03 (.00, .15)</td>
<td>.00 (.00, .13)</td>
<td>.03 (.00, .15)</td>
<td>.97 (.85, 1.00)</td>
</tr>
<tr>
<td>Harm Avoidance</td>
<td>.00 (.00, .19)</td>
<td>.09 (.00, .18)</td>
<td>.09 (.00, .19)</td>
<td>.91 (.81, 1.00)</td>
</tr>
<tr>
<td>Novelty Seeking</td>
<td>.00 (.00, .09)</td>
<td>.00 (.00, .07)</td>
<td>.00 (.00, .09)</td>
<td>1.00 (.91, 1.00)</td>
</tr>
<tr>
<td>Reward Dependence</td>
<td>.06 (.00, .16)</td>
<td>.00 (.00, .12)</td>
<td>.06 (.00, .06)</td>
<td>.94 (.84, 1.00)</td>
</tr>
<tr>
<td>Persistence</td>
<td>.00 (.00, .16)</td>
<td>.05 (.00, .15)</td>
<td>.05 (.00, .16)</td>
<td>.95 (.84, 1.00)</td>
</tr>
<tr>
<td>Age</td>
<td>.11 (.00, .17)</td>
<td>.00 (.00, .12)</td>
<td>.11 (.05, .17)</td>
<td>.89 (.83, .95)</td>
</tr>
</tbody>
</table>
### Supplementary Table IV: Uncontrolled partner similarity score correlations between family members (r and 95%CI).

<table>
<thead>
<tr>
<th>Trait</th>
<th>MZM Twins</th>
<th>MZF Twins</th>
<th>MZ-Equated</th>
<th>DZM Twins</th>
<th>DZF Twins</th>
<th>DZOS Twins</th>
<th>DZ-Equated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body mass index</td>
<td>.13 (-.08, .33)</td>
<td>.17 (.02, .30)</td>
<td>.16 (.03, .27)</td>
<td>.33 (.06, .59)</td>
<td>.36 (.15, .53)</td>
<td>.19 (-.05, .40)</td>
<td>.29 (.15, .41)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>.08 (-.11, .26)</td>
<td>.11 (-.04, .25)</td>
<td>.09 (-.02, .20)</td>
<td>.10 (-.27, .42)</td>
<td>.03 (-.18, .24)</td>
<td>.08 (-.14, .28)</td>
<td>.06 (-.08, .20)</td>
</tr>
<tr>
<td>Education</td>
<td>.17 (.06, .27)</td>
<td>.12 (.05, .19)</td>
<td>.13 (.07, .19)</td>
<td>.16 (.01, .30)</td>
<td>.08 (-.01, .17)</td>
<td>.05 (-.04, .15)</td>
<td>.08 (.02, .14)</td>
</tr>
<tr>
<td>Income</td>
<td>.05 (-.07, .18)</td>
<td>.09 (.00, .18)</td>
<td>.08 (.01, .15)</td>
<td>-.05 (-.21, .11)</td>
<td>.05 (-.08, .18)</td>
<td>-.04 (-.15, .07)</td>
<td>-.01 (-.09, .06)</td>
</tr>
<tr>
<td>Religiosity</td>
<td>-.04 (-.15, .07)</td>
<td>.16 (.09, .22)</td>
<td>.10 (.05, .16)</td>
<td>.07 (-.08, .22)</td>
<td>.03 (-.05, .12)</td>
<td>-.04 (-.14, .06)</td>
<td>.01 (-.05, .07)</td>
</tr>
<tr>
<td>Attitudes</td>
<td>.11 (-.11, .32)</td>
<td>.11 (-.03, .24)</td>
<td>.11 (-.01, .22)</td>
<td>.07 (-.19, .32)</td>
<td>-.10 (-.34, .15)</td>
<td>-.03 (-.30, .26)</td>
<td>-.02 (-.17, .13)</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>.00 (-.18, .18)</td>
<td>.10 (-.04, .23)</td>
<td>.07 (-.05, .17)</td>
<td>.33 (-.04, .58)</td>
<td>-.05 (-.26, .16)</td>
<td>.04 (-.26, .16)</td>
<td>.04 (-.10, .18)</td>
</tr>
<tr>
<td>Extraversion</td>
<td>.07 (-.12, .26)</td>
<td>.03 (-.12, .18)</td>
<td>.04 (-.08, .16)</td>
<td>-.17 (-.48, .21)</td>
<td>.04 (-.16, .23)</td>
<td>-.05 (-.29, .19)</td>
<td>-.02 (-.16, .12)</td>
</tr>
<tr>
<td>Psychoticism</td>
<td>.03 (-.18, .25)</td>
<td>.04 (-.10, .18)</td>
<td>.05 (-.08, .17)</td>
<td>.18 (-.29, .53)</td>
<td>-.13 (-.36, .14)</td>
<td>-.14 (-.41, .17)</td>
<td>-.07 (-.23, .10)</td>
</tr>
<tr>
<td>Harm Avoidance</td>
<td>-.05 (-.25, .15)</td>
<td>.14 (-.01, .27)</td>
<td>.08 (-.04, .19)</td>
<td>.38 (.11, .58)</td>
<td>-.08 (-.30, .16)</td>
<td>-.04 (-.23, .15)</td>
<td>.05 (-.09, .18)</td>
</tr>
<tr>
<td>Novelty Seeking</td>
<td>-.03 (-.26, .19)</td>
<td>.00 (-.14, .13)</td>
<td>-.01 (-.13, .11)</td>
<td>-.08 (-.31, .18)</td>
<td>-.03 (-.23, .18)</td>
<td>-.07 (-.28, .14)</td>
<td>-.06 (-.18, .07)</td>
</tr>
<tr>
<td>Reward Dependence</td>
<td>.16 (-.04, .34)</td>
<td>.05 (-.10, .20)</td>
<td>.08 (-.04, .19)</td>
<td>-.03 (-.32, .27)</td>
<td>-.06 (-.25, .14)</td>
<td>.10 (-.17, .34)</td>
<td>-.01 (-.15, .13)</td>
</tr>
<tr>
<td>Persistence</td>
<td>.08 (-.14, .29)</td>
<td>-.01 (-.17, .15)</td>
<td>.02 (-.11, .15)</td>
<td>.06 (-.25, .35)</td>
<td>.11 (-.11, .32)</td>
<td>-.13 (-.37, .13)</td>
<td>.03 (-.12, .18)</td>
</tr>
<tr>
<td>Age</td>
<td>.12 (.00, .22)</td>
<td>.11 (.04, .18)</td>
<td>.11 (.01, .16)</td>
<td>.00 (-.14, .13)</td>
<td>.01 (.00, .16)</td>
<td>.01 (.00, .17)</td>
<td>.07 (.01, .12)</td>
</tr>
</tbody>
</table>

Note: M = male; F = Female; OS = Opposite Sex; MZ = Monozygotic; DZ = Dizygotic. Equated = correlations equated across sex.
Supplemental Figure I. Histograms comparing controlled vs. uncontrolled difference scores
Raw BMI Difference Scores

Controlled BMI Difference Scores

Raw Education Difference Scores

Controlled Education Difference Scores
Raw Religiosity Difference Scores

Controlled Religiosity Difference Scores

Raw Reward Dependence Difference Scores

Controlled Reward Dependence Difference Scores