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The Role of Mutations in Human Mating

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Chapter for *Mating Intelligence: Theoretical and*

Empirical Insights into Intimate Relationships

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1   Introduction

Evolutionary theory is the central organizing principle in the life sciences. Like other theoretical pillars in science, its value comes not only from its ability to explain existing observations according to a set of lawful principles, but also from its ability to test those explanations with new predictions. Given that there is no competing scientific explanation for complex biological design, and that human behavior is undoubtedly guided by mechanisms that are biologically complex, the question is not *whether* evolution has shaped the brain mechanisms that underlie human behavior, but rather *how* it has done so. This is not to say that everything that evolutionary psychologists have hypothesized to date is correct; the merit of these hypotheses will continue to become clearer as more data accumulate. Rather, the point is that evolutionary approaches will be central to the scientific understanding of human behavior, and indeed have already proven their scientific worth by stimulating the formation of testable and novel hypotheses in psychology (Buss, 2005).

To date, evolutionary psychologists have been focused mainly on one central aspect of evolutionary theory: adaptationism, or species-typical design features that aided ancestral fitness. There is, however, another central aspect of evolutionary theory that has been much-neglected so far in evolutionary psychology: understanding the causes and consequences of genetic variation within our species. This division between the study of adaptation and the study of genetic variation is not unique to psychology. At the beginning of the 20th century, until such luminaries as Ronald Fisher, Sewell Wright, and J. B. S. Haldane

showed otherwise, many scientists believed that Mendelian principles of heredity conflicted with the theory of natural selection (Bowler, 1989). Although the apparent conflicts between genetics and natural selection have long since been resolved, biology departments are often still divided between those studying genetics (typically at the molecular level) and those studying adaptation (typically at the organismic level).

The reason for the scientific divide between natural selection and genetics is not purely historical, however. The specific principles used to understand genetic differences are related but distinct from those used to understand genetic similarities (i.e., species-typical design features). It is no great surprise, then, that psychologists interested in adaptation have largely ignored evolutionary genetics. More surprising is the fact that behavioral geneticists and other psychologists interested in individual differences, with a few exceptions (Bailey, 2000; Eaves, Martin, Heath, Hewitt, & Neale, 1990; Gangestad & Yeo, 1997; Miller, 2000), have only rarely considered genetic variation in the light of evolutionary genetics.

Evolutionary psychology is missing an important piece of the puzzle by neglecting evolutionary genetics. In this chapter, I argue that evolutionary genetics, especially new evidence on the role that mutations play in the evolutionary process, is fundamental to understanding individual differences in behavior (e.g., variation in intelligence, personality, attractiveness, status, and courtship abilities as assessed in mate choice), as well as those species-typical adaptations that track such individual differences (e.g., mate choice systems). Thus, evolutionary-genetic principles also help to distinguish between different

conceptualizations of Mating Intelligence (MI). Finally, insofar as a theoretical approach demonstrates its worth by making testable predictions, I conclude with two illustrations of how a mutational hypothesis of individual differences can make novel predictions regarding MI.

2   On mutations and being human

Errors are inherent to life. Despite my best intentions, it is likely that a few grammatical or spelling errors have found their way into this chapter. But this chapter has only 7,300 words and 49,000 characters. If, instead, I were to write a tome of, say, 12.5 million words and 75 million characters (about 25,000 pages long—like the 25,000 genes in the human genome), not even careful writing and a full team of meticulous editors could successfully keep the work mistake-free. The probability of mistakes per event can be vanishingly small, but across enough events, mistakes become inevitable. Along with natural selection, this simple principle of probability is at the core of the evolutionary process.

Mutations are errors introduced into the structure of DNA, such as substitution of the original base-pair (A, C, G, or T) for another (called a point mutation), alterations in base-pair numbers (such as deletions or insertions), or larger changes in base-pair organization at the chromosomal level (such as translocations, inversions, or duplications). In this chapter, I focus only on point mutations (hereafter, simply *mutations*) because these are the most common (Nachman & Crowell, 2000) and best understood. Mutations most often occur during the replication of DNA prior to cell division, although the probability that a

mutation occurs during the replication of any given base-pair of DNA is low and remarkably consistent across eukaryotic life-forms: about two errors per billion base-pair copying event (Keightley & Eyre-Walker, 2000). This low error rate is a testament to billions of years of intense selection for fidelity in gene duplication, and for correction of those errors that happen to occur. Most DNA errors arise in non-germline cells, and are of little evolutionary interest because they are not transmitted to offspring (although they can result in diseases, such as cancer). However, central to the evolutionary process are those mutations that occur in sperm or egg cells, and that are then transferred to the fertilized ovum and, eventually, to every cell in the offspring's body, including the offspring's own germline cells.

Although the probability of a mutation per base pair per meiosis (cell division) is miniscule, human germline cells go through tens to hundreds of meiotic events before becoming an egg or a sperm. For each of these meiotic events, about 75 million evolutionarily important base pairs in or around the 25,000 genes (out of 3 billion base pairs overall in human genome) must be replicated. As with errors in the 25,000 page tome described above, the probability of an offspring inheriting a new mutation becomes quite high across the entire genome—current estimates are that around four in every five human offspring inherit one or more new mutations that affect the phenotype[1]. These mutations almost always harm fitness for the same reason that random changes to a computer's circuitry would almost always harm performance: entropy erodes functional complexity (Ridley, 2000).

Thus, most people reading this chapter carry one or more new mutations that impair fitness, that pervade every cell in the body, and that were not inherited from either parent. Sometimes these mutations are catastrophic to the phenotype, causing, for example, skull malformation and digit fusion (Apert's syndrome) or short-limbed dwarfism (Achondroplasia). But most new, deleterious mutations have minor, perhaps unnoticeable, phenotypic effects, such as causing one to be a little less bright, attractive, or athletic. These mutations are nevertheless significant evolutionarily, and most are destined to become extinct at some point in the future, although it may take a while for selection to eliminate them. For example, a mutation causing a 1% reduction in fitness (e.g. a 1% reduction in number of surviving offspring) will persist, on average, for about 10 generations and pass through about 100 different bodies (in multiple coexisting copies) in a large population before going extinct (García-Dorado, Caballero, & Crow, 2003). Because of this time-lag between a mutation's origin and its elimination, every population at any given time carries an encrustation of slightly old, slightly deleterious mutations. As a result, offspring do not just inherit a couple of new deleterious mutations; they also inherit from their parents an average of 500 (and perhaps many more) older, very slightly deleterious mutations in all of their cells (Fay, Wyckoff, & Wu, 2001). Humans and other animals with large genomes and long generational intervals are awash with deleterious mutations.

How might these mutations affect the phenotype? Clearly they do not usually result in Mendelian catastrophes. Elsewhere, Geoffrey Miller and I (Keller

& Miller, in press) have proposed that much of the genetic variation underlying the liability to mental disorders may be a consequence of mutations that undermine the adaptive brain mechanisms responsible for normal human behaviors. In that paper we presented several empirical observations supporting the view that mutations are important in the genetic etiology of mental disorders: 1) the apparent fitness costs of mental disorders (as manifest in reduced social and sexual success, at least in modern environments), 2) the very small effect sizes of those few susceptibility alleles that have been found to predict mental disorders so far in gene-mapping studies (suggesting that many mutations, rather than a few major genes maintained by selection, account for most mental disorder risk), 3) the increased risk of mental disorders with genetic inbreeding (which reveals the full effect of many, partially recessive, but deleterious mutations), and 4) the increased risk of mental disorders with paternal but not maternal age (the number of mutations in sperm but not eggs increases as parents age). These observations are exactly what would be predicted if most susceptibility alleles for mental disorders are actually harmful mutations that have not yet been removed by natural selection. Further, these observations are hard to reconcile with other mechanisms of genetic variation, such as balancing selection, which can favor a diversity of strategies in a population, but which tends to orchestrate their development through genes that show large effect sizes, equal average fitness, no inbreeding depression, and no paternal age effects.

Mental disorders are merely the tip of the iceberg. Individual differences in nearly every phenotype studied are related, to various degrees, to differences in peoples' genes. That is, nearly every phenotypic trait studied so far is heritable to some degree (Plomin, DeFries, McClearn, & McGuffin, 2001). We suggest that much of this genetic variation, especially in traits related to fitness, may be maladaptive—an inevitable consequence of the hundreds of individually minor deleterious mutations that everyone harbors to different degrees.

However, the idea that deleterious mutations have much impact on traits related to fitness seems to fly in the face of canonical evolutionary thought. The traditional view before about 1990 was that mutations might be common enough in traits that are peripherally related to fitness, but natural selection should ensure that they play little role in traits strongly related to fitness. As discussed in the next section, this common-sense expectation has turned out to be wrong— wrong enough to have misguided mate-choice research for many decades. Because mating intelligence is so strongly related to fitness (reproductive success), a better understanding of the evolutionary genetics of mutation may be crucial to developing a better understanding the role of human intelligence in mate attraction and mate choice.

3   Mutations and genetic variation in fitness-related traits

Alleles are the different variants (versions of DNA sequences) at genetic loci (genes positioned on chromosomes), and are the cause of genetic variation in phenotypes. Although geneticists often reserve the term *mutations* for genetic

variants with frequencies below 1% and *alleles* for genetic variants with frequencies over 1%, all alleles came into existence originally as mutation events. Thus, in this chapter, *allele* will be used as the generic term, irrespective of frequency. A single allele will have an *average*, or additive effect, on some phenotype, across all of its likely genetic contexts (all other possible alleles at other loci). Because this additive effect does not depend on specific combinations of alleles, it tends to be shared between parent and offspring. The additive genetic variation of a trait in a population, V_A, is roughly the cumulative variation of all these average effects across all alleles affecting the trait (Falconer & Mackay, 1996). One of the longest-standing expectations in evolutionary genetics, often called "Fisher's Fundamental Theorem," has been the idea that strong selection should drive the fittest alleles (those with the most fitness-positive additive effects) to "fixation" (100% prevalence), and should drive all less fit alleles to extinction (0% prevalence), causing the V_A of fitness-related traits (those that are under strong selection) to approach zero (Fisher, 1930; Haldane, 1932; Kimura, 1958). This occurs because any locus with a single allele fixated at 100% prevalence will show no locus-level genetic variation, so will contribute nothing to trait-level genetic variation. Thus, Fisher's Fundamental Theorem implied that the V_A of fitness-related traits should be very low. Empirical data initially seemed to confirm these expectations: traits that are highly relevant to fitness, such as fecundity and lifespan, had lower heritabilities (a rough index of V_A, see below) than traits less related to fitness, such as body size (Roff & Mousseau, 1987).

The expectation of minimal V_A in traits related to fitness did create some problems of its own, however. For example, what good would it do for females to choose males based on some sexually selected trait, such as long tails or deep croaks, when no genetic benefits of female choice are apparent? One common explanation for female choice—that females receive better genes by choosing males who are exceptional on these traits—relies on male traits that honestly advertise genetic *differences* in fitness. Yet how could genetic differences in fitness exist, given that fitness should be under maximal selection pressure, and should thereby show minimal amounts of V_A? This quandary became known as the "lek paradox" (Andersson, 1994; Borgia, 1979; Kirkpatrick & Ryan, 1991).

A lek is a congregation of males in certain species who compete and display for females during breeding season. It was seen as paradoxical that females in such species should care at all about choosing one male over another given that persistent female choice should erode genetic variation in male genetic quality. A common response to the paradox was to simply refute the idea that females were selecting for good genes at all: for decades, many biologists expected 'good genes' mate choice to be irrelevant, and focused on the material benefits of choosing high-quality mates—greater nuptial gifts, parental investment, survival, fertility, and so forth. But for many species, especially lekking species where females receive no material benefit or parental aid, such practical benefits of female choice could not be found (Andersson, 1994).

The crisis came to a head when biologists such as Houle (1992), Charlesworth (1987), and Price (1991) realized that fitness-related traits might

not have low V_A after all—that premature conclusion may have been an artifact of how scientists were comparing different traits' genetic variation. V_A is measured in (squared) units of whatever metric is used to measure a trait. Clearly one cannot directly compare the V_A of two traits measured on two different scales, such as the V_A of height in squared centimeters versus the V_A in squared number of offspring. To make these variances comparable (to remove their scale-dependence), V_A has traditionally been standardized by dividing it by the total phenotypic variation. The resulting measure, called narrow-sense heritability, is the proportion of phenotypic variation that is due to additive genetic effects. Heritability is used so often as an index of V_A that it is easy to forget that dividing genetic variance by total phenotypic variance is just one possible way to remove scale-dependence. Specifically, narrow-sense heritability might be misleadingly low, not because the absolute V_A is low, but because the total phenotypic variation is high (due to cumulative random effects in development and life-success), as it often is for traits highly related to fitness. For example, achieved fecundity—actual number of offspring produced by a particular organism—depends not just on genetic quality, but on luck in surviving and reproducing; whereas leg length or brain size depends relatively less on luck. Another technique for removing scale dependence, and one that is not confounded by the total phenotypic variation, is to divide a trait's variation (or technically, its standard deviation) by its mean. This metric is called the "coefficient of variation". It is usually expressed as a percentage, so coefficients of variation (CVs) can range from 0% to 100% (if a trait's standard deviation

equals its mean) to more than 100%. Traits that vary more across individuals within a species show higher CVs; for example, the CV of human brain volume is about 8%, whereas the CV of male human penis volume is about 37%, and the CV of female human breast volume is about 62% (Miller & Penke, in press).

Neither heritability nor CV is a perfect index of genetic variation: trait heritabilities are lower when trait development is more influenced by random events, and CVs are lower when trait sizes are measured as lengths rather than areas or volumes (Lande, 1977). However, Houle (1992) gave good reasons to believe that CV is often more informative. When traits were investigated using this new metric of V_A, a remarkable observation emerged: *the coefficient of additive genetic variation of fitness-related traits (such as fecundity or survival) was about five times higher than it was for traits less related to fitness (morphological traits such as bristle number or weight)* (Houle, 1992)—the opposite of what Fisher's Fundamental Theorem seemed to predict. Houle's observation, along with the paradox of the lek, created a real theoretical crisis in evolutionary genetics throughout the 1990s, the ramifications of which are only now beginning to seep into the consciousness of evolutionary psychology and the study of human mate choice.

Why would traits under the strongest selection have the highest mean-standardized V_A? It now appears that one of the most important factors affecting any traits' V_A is the *number of loci that influence the trait*, because many loci provide a larger 'target size' for mutations (Houle, 1998; Houle, Morikawa, & Lynch, 1996). Mutations tend to disrupt the functioning of more highly polygenic

traits (those influenced by many genetic loci, such as traits closely related to fitness—survival ability, sexual attractiveness, and achieved reproductive success), whereas they have less effect on simpler traits that depend on fewer loci, and that influence fitness less directly. At the same time, natural selection works to reduce the genetic variation introduced by these mutations. The end result is a balance between mutation and selection, and an equilibrium number of mutations that degrade the functioning of—and cause V_A in—every conceivable trait.

Fitness traits tend to be highly polygenic because they require the proper functioning of so many other subsidiary, 'upstream' processes (Charlesworth, 1987; Houle, 1992, 1998; Price & Schluter, 1991)—one cannot produce offspring, for instance, without first producing antibodies to fight infection, neural circuitry to feel appropriate motivations, hormones to time maturation, and so forth. Thousands of 'upstream' traits must function together to build a body capable of surviving, finding a mate, and reproducing. Indeed, the mutational target size of "fitness" is, by definition, every gene in the genome that has any fitness effect. Among the most compelling pieces of evidence for the idea that mutations are the culprit behind the high mean-standardized V_A in fitness traits are the high, positive correlations in fruit flies between a) the estimated number of loci influencing traits, b) traits' coefficients of additive genetic variation, and c) the amount of V_A that mutations contribute to traits per generation (Houle, 1998). (At the level of basic evolutionary genetics, fruit flies are surprisingly good

proxies for humans, though they have only about 14,000 genes compared to our 25,000).

Thus, fitness-related traits show high levels of V_A—not because selection favors this variation and not because selection fails to work against it—but rather because selection fights against a constant mutational headwind that replenishes genetic variation in highly polygenic traits. This explanation also clarifies why fitness-related traits have relatively low heritabilities: downstream, fitness-related traits tend to be influenced by many sources of variation: not just V_A, but also non-additive genetic variation (dominance and epistatic genetic variation, which concern interactions between alleles at a genetic locus or across loci), random environmental effects, and random developmental errors. Because natural selection can reduce V_A at a much faster rate than it can reduce these other sources of variation (Fisher, 1930; Merilä & Sheldon, 1999), the ratio of V_A to the total phenotypic variation (V_A + non-additive genetic variation + environmental variation) tends to be low in fitness-related traits—which is why they show low heritability but high CVs.

4   Mutations and sex

Under mutation-selection balance, certain individuals have a low *mutation load* (i.e., possess relatively few mutations and/or possess mutations that tend to have lower average effects), while other individuals have a higher mutational load. Individuals who have a low load of mutations will tend to have mechanisms less degraded by harmful mutations, and so will tend to have higher fitness over

evolutionary time, while those with more mutations will tend to have lower fitness. Such genetic variation in fitness is a prerequisite to all 'good genes' theories of mate selection, including those in evolutionary psychology. Evolutionary processes besides mutation-selection that maintain genetic variation in populations—such as balancing selection or drift plus recurrent neutral mutations—imply that alternative alleles have *equal* fitness, when averaged across all the genomes and environments in which those alleles could find themselves (Keller & Miller, in press). In other words, balancing selection and neutral drift produce fitness-neutral genetic variation, but they cannot produce fitness-correlated V_A (i.e., heritable variation in good genes). Aside from finding genes that complement one's own (e.g. mating with the right species, avoiding genetic inbreeding), there is no genetic advantage in choosing mates if there is no fitness-correlated V_A—given balancing selection or neutral drift, any randomly chosen mate would have about equally fit genes on average. Thus, the only plausible origin for V_A in fitness-related traits, such as sexually selected traits, is mutation-selection balance.

A mutation-selection explanation for V_A in fitness also neatly resolves the lek paradox: if sexually selected traits are highly correlated with fitness ("index handicaps") or are costly in 'fitness currency' ("strategic handicaps"), then any process that maintains V_A in fitness (i.e., mutation-selection) must also maintain V_A in the sexually selected traits that reflect fitness. Indeed, persistent sexual selection on any arbitrary trait should eventually cause that trait to correlate with fitness and, hence, to become more highly polygenic (Rowe & Houle, 1996). That

is, sexual selection tends to make sexually attractive traits more fitness-sensitive, more dependent on many genes, and more reliable indicators of overall mutation load and genetic quality.

To illustrate why this should be, consider what would occur if, over evolutionary time, females were most attracted to males with the longest fingers. As finger length increased, this trait would become increasingly costly (dealing with foot-long fingers would be a tough thing to do!). Selection would favor contingent adaptations that allow expression of the costly trait only to the degree that it pays off in fitness currency. For example, if males didn't care for finger length in female mates, then females would receive none of the sexual benefits but all of the survival costs of expressing their exaggerated-finger-length genes. In this case, selection would favor an adaptation that turns exaggerated-finger-length genes on or off depending upon whether the genes are in male or female bodies. In other words, sexual selection for a trait in only one sex should lead to sexual dimorphism, which is widely observed in nature (Darwin, 1871).

For the same reason that sexual selection leads to differences in the expression of sexual traits *between* sexes, it should also lead to differences in the expression of sexual traits *within* a sex, depending upon each individual's genetic quality (low mutation load) and phenotypic condition (overall health). Continuing with the example of sexual selection for finger length, males whose fingers are too long given their condition would have lower survival: if they had many mutations, poorly-functioning brains, and poor hand-eye coordination, they would often get their fingers cut, crushed, and burned, and they wouldn't be able

to hunt or fight effectively with such handicaps. On the other hand, males with few mutations, smarter brains, and better coordination would suffer fewer survival costs and would enjoy higher reproductive benefits from their super-sexy fingers. These differential costs and benefits of finger-length should lead selection to favor a contingent (condition-dependent) adaptation: males should grow the longest fingers possible *given* their own condition. Once such contingent mechanisms are universal, the fittest males—those least degraded by harmful mutations—would be best able to bear the costs of developing long, ornamental, self-handicapping fingers; low-fitness males would grow shorter, more practical fingers. Finger length, even if the trait were originally controlled by only a small number of genes, would become an honest signal of condition; the mutational target size of finger length increases from the genes originally only devoted to finger length, before sexual selection, to all the fitness-related genes in the genome, after sexual selection.

The end result of sexual selection is a species where an initially arbitrary trait comes to be correlated with individual mutation load, condition, and fitness. This is an interesting evolutionary property, because it suggests that whatever the origins for sexual selection preferences, be they due to Fisherian runaway, to random sensory bias, or because they genuinely reflect genetic quality from the beginning (for discussion of the possible origins of sexual selection traits, see Andersson, 1994), they will eventually become good ways to distinguish mates based upon genetic quality/mutation load (Kirkpatrick & Ryan, 1991; Rowe & Houle, 1996).

4   Mutations and mating intelligence

The relationship between mutations and sexual selection, discussed

above, is relevant to different ways of thinking about mating intelligence (MI).

Geher and Murphy (this volume) identified several ways that MI can be

conceptualized; here, I will discuss three broad categories of MI that are related,

but not identical, to those made by Geher and Murphy (2006). None of these

three conceptualizations is 'correct,' of course, but the evolutionary-genetics

principles explored above suggest that different types of mating intelligence

require different types of evolutionary explanation, fulfill different adaptive

functions, and may benefit from different names. One potential conceptualization

of MI concerns *mating preferences*—qualities that people find attractive in mates.

Understanding human mating preferences has been one of the major interests of

evolutionary psychologists, as a glance through any evolutionary psychology

journal or textbook would indicate (e.g., Buss, 2004). Different types of mating

preferences probably serve somewhat different functions—some may help

secure mates willing and able to commit material resources, some may help

secure behaviorally compatible mates, and, as detailed above, some may help

select mates of high genetic quality (Buss, 1999). Cues of genetic quality may be

favored, for example, by mate preferences for intelligence and artistic ability

(Miller, 2000), symmetrical faces and bodies (Gangestad & Thornhill, 1999b),

athleticism (Buss & Schmitt, 1993), body shape (Singh, 1993), dancing ability

(Brown et al., 2005), and facial features associated with sex-hormones (Thornhill & Gangestad, 1999).

A different conceptualization of MI concerns *mating abilities*—individual differences in those traits that people find attractive—which have been the central focus of the current chapter so far. For example, how successful are different people at attracting desirable mates? Reflecting the long standing divide between evolutionary genetics and adaptationism discussed at the beginning of this chapter, the study of mating abilities mainly concerns individual differences, while the study of mating preferences mainly concerns species-typical design. The two concepts are inherently related, of course—some species-typical mating preferences have evolved in order to track individual differences in mating ability—but their evolutionary origins and adaptive functions nevertheless require quite different types of explanations.

To simplify somewhat, we should expect much more fitness-related genetic variation in mating abilities than in mating preferences[2]. This is because mating preferences should have evolved toward those preferences that are best at discriminating between good mates, but there is no reason to believe that the genetic bases of such preferences would be any more polygenic than any other evolved preference or behavior. An evolved rule that says, "Be attracted to the longest fingers," is under selection for a *single* preference, and those alleles that code for such a preference should become fixed in the population, leading to little fitness-relevant genetic variation in the preference. Similarly, we do not expect much genetic variance in food preferences: sweet, salty, fatty tastes (indicating

ancestrally scarce nutrients) are universally attractive, whereas bitter tastes (indicating plant toxins) and rancid tastes (indicating bacterial spoilage) are universally unattractive.

By contrast, mating abilities have been under open-ended, directional (more-is-better) selection for a long time, and they have become correlated with fitness, and hence highly polygenic, for the reasons outlined above. Mating abilities will show high levels of fitness-related genetic variation, and thereby will reliably reveal the different genetic qualities of different potential mates, despite being under intense selection. Most of this genetic variation in mating abilities should be due to deleterious mutations. At the genetic level, intelligent or beautiful people do not so much have genes that cause them to be intelligent or beautiful as they lack the genes (mutations) that would make them unintelligent or unattractive.

A final conceptualization of MI concerns *individual differences in people's understandings of human mating preferences.* For example, how well do different people understand that females tend to be more interested than males in a partner's status? Note that this conceptualization of MI does not concern how well people understand or 'mind read' potential mates, which may well be related to mating abilities. Rather, it concerns how well peoples' conscious, reported beliefs about mating preferences correspond to some evidence-based standard.

It is difficult to form evolutionary predictions about this understanding-of-mating-preferences conceptualization of MI. Much in the same way that people can maximize their reproductive success without having a conscious desire for

offspring, there is no necessary connection between people's mating behavior and people's conscious beliefs about human mating preferences. Perhaps those with the best understanding of species-typical mating preferences had higher fitness, but this does not seem self-evident. A reproductively successful male could be motivated to acquire status, resources, and so forth, and yet may report a lower-than-average awareness that women find such traits desirous. Indeed, high mate-value individuals may have less understanding of how mating preferences work, because they rarely have to work very hard to attract mates or confront difficult trade-offs themselves. Moreover, if an accurate understanding of mating preferences were indeed under selection, it should show very little fitness-related genetic variation, for much the same reasons that mating preferences themselves should show little fitness-related genetic variation: such an understanding would be under strong selection, yet should be no more polygenic than any other evolved preference.

To complicate matters, there may be a lot of adaptive self-deception about mating preferences. I suspect that most of the genetic variation in people's understandings of human mating preferences is related to intelligence and personality variables (extraversion, agreeableness, etc.), while most of the modern environmental variation is related to incidental factors such as mating experience, parental and peer influences, socio-political attitudes, education, exposure to popular science, and so forth. For these reasons, I believe that evolutionary predictions of this final conceptualization of MI are not straight-forward.

5   Predictions about mating intelligence

In this section, I briefly put forward two predictions about different conceptualizations of MI, the first concerned with mating preferences, and the second with mating abilities.

Females may have a fitness advantage in mating with younger males, whose sperm is less likely to carry new, harmful mutations.

Female humans are born with their full supply of 400 or so eggs, and these eggs have gone through only 23 replications, a number which does not change as females age. By contrast, males continue to produce sperm throughout life. At age 15, sperm cells have gone through about 35 chromosomal replications, increasing to 380 by age 30, and 840 by age 50 (Crow, 2000). The probability of germ-line mutations increases with paternal age because each chromosomal replication carries a small chance of a copying error (mutation). Consistent with this point, higher paternal, but not maternal, age is associated with lower intelligence as well as many Mendelian disorders and common mental disorders (Crow, 2000). For unknown reasons, greater maternal age is associated with a higher probability of major chromosomal abnormalities (such as Down syndrome and other trisomies), but these events are very rare compared to new mutations.

Although both women and men carry the same number of old, slightly deleterious mutations, the vast majority of mutations that exist in the population were introduced by male sperm from older fathers. The proportion of existing

mutations in the population that come from males could be quantified with better information on the relationships among age, fertility, and mutation rate. The key question regarding female preferences is: how much would individual female fitness suffer over evolutionary time by having offspring with older males?

One approach is to estimate the cumulative harmfulness of new mutations per generation (which females might avoid in their offspring by mating with young males) relative to the cumulative harmfulness of old mutations per generation (which females might avoid in their offspring by mating with attractive, intelligent males). New mutations in a fathers' sperm cells will be phenotypically expressed only in the fathers' offspring and descendents; these new mutations are not phenotypically expressed in the fathers themselves, and so do not reduce a males' mating ability. The only way that females can assess the chance that a male will pass on a new mutation to her offspring is based on cues of male age.

Although old mutations are over a hundred times more common, new mutations tend to be more deleterious, since the old mutations that persist after generations of selection must have had relatively small fitness costs. Recent, albeit tentative, results suggest that about 20% of new, deleterious mutations have effects large enough to be detectable in mutation-accumulation experiments, and these detectable mutations reduce fitness by an average of about 5% (García-Dorado, López-Fanjul, & Caballero, 2004). On the other hand, evolutionary genetic theory predicts that most old mutations should reduce fitness by between 0.00005% and 0.05%. Under the simplifying assumptions that undetectable new mutations have the same fitness effects as old mutations, and

that old mutations reduce fitness by an average of, say, 0.0025%, then a given new mutation is on average 400 times ($(.05 \times .2 + .000025 \times .8) / .000025$) more deleterious to fitness than a given old mutation. Because people harbor perhaps 500+ old mutations in their genome (Fay et al., 2001), and inherit 1-2 new mutations (Eyre-Walker & Keightley, 1999), this calculation suggests that, in terms of female fitness, it is about equally important to prefer a younger male, who is less likely to pass on new mutations, as it is to prefer a high-quality male, who is less likely to pass on older mutations. The prediction that cues of youth and of genetic quality are about equally important to human females is highly speculative at this point, and could easily be wrong. While it is based upon a number of assumptions that are likely to be fairly accurate (the hominid deleterious mutation rate; the number of old, deleterious mutations per human; the average effect size of new, detectable mutations), it is also based upon some assumptions by the author that are little more than educated guesses (the average effect of old mutations; the average effect of new, undetectable mutations). Several complications, such as the non-linear acceleration in rates of paternal mutations with age (Crow, 2000), were also unaccounted for; it seems likely that below a certain age (perhaps around 35-40), the chance of a new mutation in male germline cells is still quite low, and females have little to worry about. Thus, women may not show a preference for extreme male youth so much as an aversion to extreme male age.

The goal of this exercise was to illustrate how evolutionary-genetic theory, given empirical data from seemingly distant fields, can make new, quantifiable

predictions relevant to human mate choice and mating intelligence. This exercise also shows the types of data that would be relevant in rigorously assessing the relative importance of old mutation load (affecting male mating ability) versus new mutation load (signaled by male age). Such data are increasingly available using modern genetic techniques, and more accurate estimates than the ones presented here will probably be available in a few years.

If better modeling does predict that females have an important fitness advantage in mating with younger males, it would seem to go against the standard evolutionary psychology view that males prefer youth while females do not. It is true that many females end up in long-term relationships with high-resource, older men, but this might only reflect that female reproductive success depends on many variables and trade-offs (e.g., protection and provisioning from older, higher-status males versus the increased chance of mutations in offspring that come from mating with such males). This hypothesis suggests that a) relative to females in relationships with younger males, females in relationships with older males may be more likely to have or desire extramarital affairs, b) these affairs would tend to be with younger males, and c) this tendency to seek out younger males might peak when females are at peak fertility in their menstrual cycles. Indeed, women do seem to have adaptations for seeking good-genes extra-pair partners at peak fertility (Haselton & Gangestad, 2006). Researchers might also study how cues of older male age may over-ride cues of male genetic quality in female mate choice. This could then be compared to estimates of how harmful new versus old mutations are in females' mates. If

these numbers agreed, it would support the hypothesis that paternal age is a factor in female mate-choice, and the view that mutations have been crucial in sexual selection.

The alleles that affect mating abilities will be numerous, of small effect, and difficult to find.

Whereas the first prediction concerned mating preferences, this second prediction concerns mating abilities. Furthermore, whereas the first prediction was very sensitive to quantitative assumptions about which little is definitively known, this second prediction is fairly robust and reliable. As described previously, the genetic variation in mating abilities should be due to old and new mutations at many genes (Rowe & Houle, 1996). Because mutations can occur anywhere along a locus, the coding portion of which is typically around 1,500 base pairs long, mutation-selection has created many different, lineage-specific deleterious alleles. The frequency distributions of alleles/mutations at such loci should be extremely skewed, such that besides the most common, adaptive allele, no single maladaptive allele should have a frequency greater than about 5% (Pritchard, 2001), and usually, its frequency should be much lower. These factors—many loci, and many different, lineage-specific alleles at each locus— work against current methods of gene detection using linkage and especially association studies (Terwilliger & Weiss, 1998; Weiss & Clark, 2002; Wright & Hastie, 2001). A mutation-selection hypothesis predicts slow progress in finding genes related to mating abilities.

If, as argued by Miller (2000), intelligence is a mating ability, the slow progress in finding specific genes associated with intelligence is consistent with this expectation; several likely IQ genes have been found, but none explain much of the population variation in IQ (e.g., less than 1%; Butcher et al., 2005), despite very large studies designed to find such genes. Indeed, a mutational hypothesis suggests that gene-mapping studies on mating abilities may be misconceived. Rather than a small number of alleles "for" intelligence, there may be many thousands of different alleles (mutations) "for" unintelligence. We should predict similarly slow progress in detecting genes that affect other potential mating abilities, such as physical attractiveness, fluctuating asymmetry, musical ability, sense of humor, and athleticism.

6   Summary

I end the chapter by extending an analogy introduced at the beginning of the chapter. Imagine a world in which everyone inherits two virtually identical books of code, one from mother and one from father (representing two sets of chromosomes). Each codebook contains 75 million characters (representing evolutionarily important, phenotypically expressed base pairs), 25,000 pages (genes and their surrounding regulatory regions), and 23 chapters of various length (chromosomes). The purpose of the two codebooks together is to create an intricate, self-directed machine (an individual), and on each page of each book is a section of code for assembling one aspect of the machine (some part of an adaptation). Both codebooks, in combination, average about 500+ old copying

errors committed by some great, great, great… grandparent (probably grand-father), as well as one or two newer errors, committed by the mother or, more likely, the father, when they were putting these codebooks together. Some people's codebooks have more errors, and some have fewer, and these errors are only rarely on the same pages (much less the same characters!) between different codebooks. No-one knows exactly where these errors lie in any given codebook, although they degrade the performance of each machine to various degrees.

Now imagine a great tournament, in which every machine is unleashed into a grand playing field. Machines that function the best have the highest probability of surviving. As is the custom in this imaginary world, people pass on one complete codebook to each of their children, and each new codebook is a carefully aligned pastiche of the codebooks from father and mother. Well-functioning machines are critically important, and so a core concern is to find a mate with codebooks that have the fewest and least harmful errors. To this end, machines are designed to ask other machines to do extraordinarily difficult and complicated actions to reveal how many errors exist in their codebooks. Machines with the fewest codebook errors perform the best at such tasks (show the best mating abilities), and are the most attractive (according to mate preferences for codebook quality). The best-performing machines tend to pair up with the other best-performing machines, while the worst pair up with the worst. In this way, the population-level variation in codebook errors is greatly magnified;

some codes have very few errors and make well-functioning machines, while others are so riddled with errors that they have difficulty functioning at all.

This analogy is fanciful, but it illustrates the situation that humans and other long-lived species find themselves in. Humans have evolved under intense mutational pressure; sexual selection from both sexes has been a way that our ancestors have managed this pressure. As evolutionary psychologists such as Miller (2000; 1998) and Gangestad (2000; 1999a; 1997) have hypothesized, it is likely that those traits which make up an 'attractive' mate are precisely those traits that have been under sexual selection because they reveal the mutation load that each person carries. Physical attractiveness, intelligence, athleticism, social charm, artistic abilities... all these may be attractive because they are difficult to develop and display well, and they thereby reflect an individual's mutational load. Those innate aspects of mating intelligence that make us attracted to certain types of people have been designed through millions of years of natural selection to make it likely that our offspring can keep one step ahead of the mutational beast that forever chases us.

Footnotes

[1]By comparing the sequences of chimpanzee and human protein-coding DNA, Eyre-Walker and Keightley (1999) deduced the substitution rate at neutral and non-neutral human DNA sites. They used this to estimate that about two new deleterious mutations arise on average per human per generation. Their estimate of the number of protein-coding genes in humans (60,000) has turned out to be too high, but they also did not account for mutations in regulatory (non-coding) regions of the genome, and about as much DNA in this region is evolutionarily constrained between species (Keightley & Gaffney, 2003). Accounting for both factors, it is likely that the deleterious mutation rate in the human lineage is slightly lower than two (~1.67) per individual per generation. If these mutations occur independently of one another, their frequency distribution per individual per generation should be described by a Poisson process, with mean = variance $= \lambda = 1.67$. Therefore, the probability of being born with no *new* mutations is $(e^{-\lambda} \lambda^{-0})/0! = .189$, or about one in five. All these estimates are likely to be somewhat conservative because they do not include deletions or insertions (which are rare).

[2]This is not to say that mating preferences should necessarily be less heritable than mating abilities. Not only is heritability a poor way to compare the levels of genetic variation between traits, as discussed above, but the scaling of the genetic variation must take into account its fitness effects. Specifically, the prediction is that genetic variation in mating preferences should be unrelated to fitness, while the genetic variation in mating abilities should be related to fitness.

This could be easily tested if the correct data set exists: mating abilities should

share high genetic correlations with each other and with other traits known to be

related to fitness, while mating preferences should not.

Acknowledgments

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