Chapter 9

Mental Disorders as Catastrophic Failures of Mating Intelligence

Andrew Shaner
University of California, Los Angeles and the Veterans Affairs Greater Los Angeles Healthcare System

Geoffrey Miller
University of New Mexico

Jim Mintz
University of California, Los Angeles and the Veterans Affairs Greater Los Angeles Healthcare System

Some animals attract mates by displaying indices of genetic quality known as *sexually selected fitness indicators* (Andersson, 1994). Peacocks, for example, vibrate their showy tails as peahens hunt for the male with the biggest tail. That's because his big tail indicates that he has the genes most likely to produce high fitness in her offspring. Similarly, some human mental abilities, such as language, music, dance, art, and humor, may function as fitness indicators—the human equivalents of peacock tails (Miller, 2000). If so, those mental abilities must vary greatly in quality and that variation must include low-fitness, unattractive extremes—the human equivalents of small peacock tails. Why? Because fitness indicators can be used for mate selection only if some beaus have high-quality attractive versions and others don't; the more a trait varies across individuals, the more it can be used to select the fittest mate.

Our thesis is that some human mental disorders represent the low-fitness extremes of traits that evolved, at least in part, as sexually selected fitness indicators. In this chapter, we explore that proposition and some of its ramifications. Specifically, we'll discuss schizophrenia as a catastrophic failure of mating intelligence (as manifest in courtship ability), and anti-schizophrenia stigmatization as a possibly adaptive form of mating intelligence (as manifest in mate choice). Then, we'll explore whether fitness-indicator theory may apply to other mental disorders, including severe anxiety, depression, and mania, and whether they can be considered break-downs in mating intelligence. Finally, we'll discuss how fitness indicators arising outside the mating context may explain other mental disorders such as autism.

SCHIZOPHRENIA AS AN EVOLUTIONARY PARADOX

Schizophrenia strikes about 1 percent of people worldwide, producing delusions, hallucinations, disorganized speech, bizarre behavior, and emotional blunting. Typically beginning in late adolescence or early adulthood, it often leads to social isolation and severe lifelong disability (American Psychiatric Association, 2000). Schizophrenia is an evolutionary paradox (reviewed in Brune, 2004) as it markedly reduces reproductive success (Haukka, Suvisaari, & Lonnqvist, 2003) *and* is highly heritable (e.g., Cardno et al., 1999). So why hasn't selection eliminated the responsible genes? How can it persist at such a high prevalence—far in excess of the rate possible from a single deleterious mutation (Wilson, 1997)?

One possibility, originally suggested by Julian Huxley, Ernst Mayr, and colleagues (1964), and recently reviewed in detail (Brune, 2004), is that the same genes that cause schizophrenia in some people produce advantages in their relatives. These hidden adaptive benefits might enhance survival and reproduction, offset the evolutionary disadvantage of schizophrenia, and thereby perpetuate the responsible genes within the gene pool. However, no survival benefits have been confirmed in relatives, and while some studies have found that relatives of schizophrenics have more children than expected, other larger studies have not (reviewed in Haukka et al., 2003). Moreover, behavior-genetic modeling shows that schizophrenia is not due to a single gene or even just a few genes, as one might expect from a hidden-benefits model (Keller & Miller, in press; Riley & McGuffin, 2000). Decades of schizophrenia gene-hunting through linkage and association studies have also failed to find any major-risk genes. Consequently, investigators have concluded that schizophrenia is probably due to many genes, each accounting for a small percentage of cases (McDonald & Murphy, 2003).

This polygenic model leads to a second (and widely accepted) explanation for the persistence of schizophrenia. If schizophrenia is sufficiently polygenic, that is, if alleles (genetic differences) at many loci (chromosomal locations) are involved in its etiology, and if the penetrance (power to cause schizophrenia) of these susceptibility alleles is low, then new mutations could maintain an overall frequency of susceptibility alleles at a level sufficient to produce schizophrenia in one percent of the population (see Pritchard, 2001).

But why would *so many* genes predispose individuals to schizophrenia? Why would human mental functioning be so vulnerable to mutations at so many loci? A partial answer is that the brain systems that fail in schizophrenia are unusually vulnerable to "developmental instability" (DI). When manifest in body growth, DI results in right-left asymmetries and minor physical anomalies; when manifest in brain development, DI results in abnormal lateralization, unusual brain anatomy, lower intelligence, and psychopathology (Prokosch, Yeo, & Miller, 2005; Yeo, Gangestad, Edgar, & Thoma, 1999).

But traits needed for survival tend to develop reliably despite mutations and environmental hazards (Pomiankowski & Moller, 1995; Rowe & Houle, 1996). Why would human mental functioning be an exception? The answer may be that the brain systems which go awry in schizophrenia evolved not because they increase the odds of survival, but because they are useful in sexual courtship and competition, they increase the odds of mating, and they thereby enhance reproductive success. In other words, if a well-functioning brain is an attractive human characteristic that affects mate choice, schizophrenia may be evolutionarily analogous to a small, dull peacock's tail. More technically, it may be the low-fitness, unsuccessful extreme of a sexually selected fitness indicator that evolved in humans by mutual mate choice (Shaner, Miller, & Mintz, 2004).

That single sentence, if true, would explain many puzzling and otherwise apparently unrelated facts about schizophrenia, including why it begins in adolescence and early adulthood, why it reduces reproductive success, why it is highly heritable, why the genes underlying it are so hard to find, why it's worse in males, why it's associated with environmental hazards and abnormal brain development, why dopamine blockers are therapeutic, and even why affected individuals are so socially stigmatized.

Moreover, the hypothesis leads to some surprising and testable predictions. One, for example, is that genetic and environmental causes of schizophrenia will number in the hundreds or thousands, each accounting for no more than a few percent of cases. Another prediction is that the responsible genes will comprise a wide variety of fitness-reducing mutations that remain lineage-specific (localized in particular populations), and therefore will not replicate well across populations. A third prediction is

that drugs which reduce courtship behaviors in animals (e.g., a drug that stops peacocks from displaying their tails to peahens) may improve schizophrenia in humans.

To explain our hypothesis, we'll first review how sexually selected traits may serve as fitness indicators. Next, we'll explain how schizophrenia can be viewed as the unattractive extreme of such a trait. Finally, we'll show how this view can have so much explanatory and predictive power.

SEXUALLY SELECTED FITNESS INDICATORS

Darwin argued that traits which improve survival are more likely than others to be passed on to offspring, and that this selection process could account for the evolution of new adaptations and new species (Darwin, 1859). But he was troubled by the large number of traits that have no survival value or that might even impair survival—traits such as peacock tails, elk antlers, and human music. He suggested that they evolved for a different purpose—acquiring mates (Darwin, 1871). He wrote:

> All animals present individual differences, and as man can modify his domesticated birds by selecting the individuals which appear to him the most beautiful, so the habitual or even occasional preference by the female of the more attractive males would almost certainly lead to their modification; and such modification might in the course of time be augmented to almost any extent, compatible with the existence of the species. (pp. 750–751)

Traits that improved mating success, he argued, also stood a better chance of being passed on to offspring, and this process could account for the evolution of new species. Darwin discussed two mechanisms of sexual selection: "Contests" between males over females, which favor "weapons" such as elk antlers, and "mate choice" by females, which favors male "ornaments" such as peacock tails. More recently, biologists have identified additional sexual-selection mechanisms including endurance rivalry, scramble competition, and sperm competition (Andersson, 1994).

What makes a trait attractive to potential mates? Darwin didn't know, but subsequent evolutionary theorists have suggested several possibilities (which can act simultaneously). Traits may become attractive because they advertise health, fertility, vigor, longevity, parenting ability, optimal genetic distance, good genes, and/or simply the prospect of passing on attractiveness itself (Andersson, 1994).

How can an ornament, such as a peacock's tail, advertise genetic quality or fitness? If healthier birds tend to grow brighter feathers, then the brightness of feathers would indicate fitness (Fisher, 1915). Moreover, the offspring of females who prefer brighter feathers would inherit the

father's genes for better fitness and the mother's genes for preferring bright feathers. Across generations, the increasing co-occurrence within individuals of the preference genes and the fitness genes would lead to a powerful positive-feedback process that could fuel the rapid evolution of brighter feathers—a process termed "runaway sexual selection" (Fisher, 1930).

Why would healthier birds have brighter feathers or bigger tails? One possibility is a mechanism called "the handicap principle" (Zahavi, 1975). A peacock's tail takes considerable energy to grow, maintain, and display. This cost could make it a reliable indicator of fitness, because only the fittest peacocks can afford the energy necessary to grow large and colorful tails. As a result, peahens would evolve a preference for the extravagant extreme. The handicap principle and several related mechanisms produce extravagant traits in theoretical models (Andersson, 1994; Hasson, 1989; Michod & Hasson, 1990)—even in monogamous species (Hooper & Miller, submitted). Moreover, empirical work has shown that some sexually selected traits bear the three hallmarks of fitness indicators (Andersson, 1994): (1) they vary greatly in size, loudness, complexity, or other qualities across individuals; (2) that variance correlates with underlying fitness and condition; and (3) potential mates prefer the high-fitness extreme.

But this leads to another question—why don't all peacocks have big, beautiful tails? Tails vary greatly in size and complexity, and that variation is somewhat heritable. However, in a group of peafowl, the one or two peacocks with the most elaborate tails sire virtually all the offspring (Petrie, Halliday, & Sanders, 1991). Why don't the genes for big tails proliferate and why don't the genes for less elaborate tails disappear? This question is called "the paradox of the lek" (Kirkpatrick & Ryan, 1991)—a lek being the clearing in which male birds display their ornaments as females inspect and choose—not unlike a singles bar. Recently, several investigators have suggested a common potential resolution of the "lek paradox" (Houle & Kondrashov, 2002; Kotiaho, Simmons, & Tomkins, 2001; Michod & Hasson, 1990; Pomiankowski & Moller, 1995; Rowe & Houle, 1996). This resolution, discussed subsequently, is at the heart of the explanatory and predictive power of our hypothesis regarding schizophrenia.

THE LEK PARADOX RESOLVED

The resolution requires a distinction between "good" and "bad" genes. "Good genes" are those versions of genes ("alleles") best suited to an animal's current ecological niche, and to the rest of its species-typical genome. Individuals with "good genes" grow better bodies and brains, find more

food, resist more parasites, avoid more predators, survive longer, and, thereby, leave more offspring. However, to reproduce, they must make sperm or ova. In that process, they must copy DNA, and DNA cannot be copied perfectly. Copying errors produce new versions of genes that are (almost always) less well-suited to the niche. These altered genes are called *fitness-reducing mutations* or "bad genes." They reduce the chances that offspring will survive and reproduce. In every generation, copying errors supply new "bad genes." For example, the average human child has two to four new harmful mutations that neither parent had (Eyre-Walker & Keightley, 1999). Selection immediately removes fatal mutations, and quickly removes very harmful mutations. Mildly harmful mutations, however, can persist for many generations. A mutation causing a 1 percent reduction in fitness will persist in the population for 100 generations, on average (Falconer, 1996). The balance between mutation and selection leads to an equilibrium frequency of "bad genes" in a population (Keller & Miller, in press). For example, the average human carries 500 to 2,000 old mutations inherited from his or her ancestors—mutations that have not yet been eliminated by selection (Fay, Wyckoff, & Wu, 2001; Sunyaev et al., 2001). The number and type of "bad genes" (referred to, in composite, as "mutation load") varies across individuals and is responsible for most of the heritable variation in fitness (Houle & Kondrashov, 2002; Michod & Hasson, 1990; Rowe & Houle, 1996).

Mutation load reduces fitness and is the key to resolving the lek paradox. In panel "a" of Figure 9–1, we've modeled variation in fitness as a normal distribution with a mean of 50 and standard deviation of 10. Panel "b" shows a hypothetical relationship between fitness and the ultimate attractiveness of a sexually selected trait. Panel "c" shows the result of applying the function in "b" to the distribution in "a." For now, apply the figure to the attractiveness of peacock tails. Ignore the dashed lines in panels "b" and "c" as well as the reference to schizophrenia in panel "c."

Imagine that we could pick out the peacock embryos lucky enough to have been conceived with very few "bad genes" (i.e., a low mutation load) and therefore high fitness, say "75" on the fitness scale in panels "a" and "b." These embryos have "good genes" for precise cell migration, efficient feeding, parasite resistance, predator evasion, and any other process that can ultimately affect tail size. Thus, embryos with "good genes" for general fitness tend to develop into adult peacocks with very large and elaborate tails at about "7" on the attractiveness scale.

However, most peacock embryos contain some "bad genes" and end up with somewhat smaller, less elaborate tails. A few peacock embryos at the low-fitness extreme of the distribution contain more than their share of bad genes. Imagine we could pick out embryos with a fitness score of "35." The "bad genes" in these embryos are so numerous or so severe that they interfere with several of the hundreds of developmental processes that can

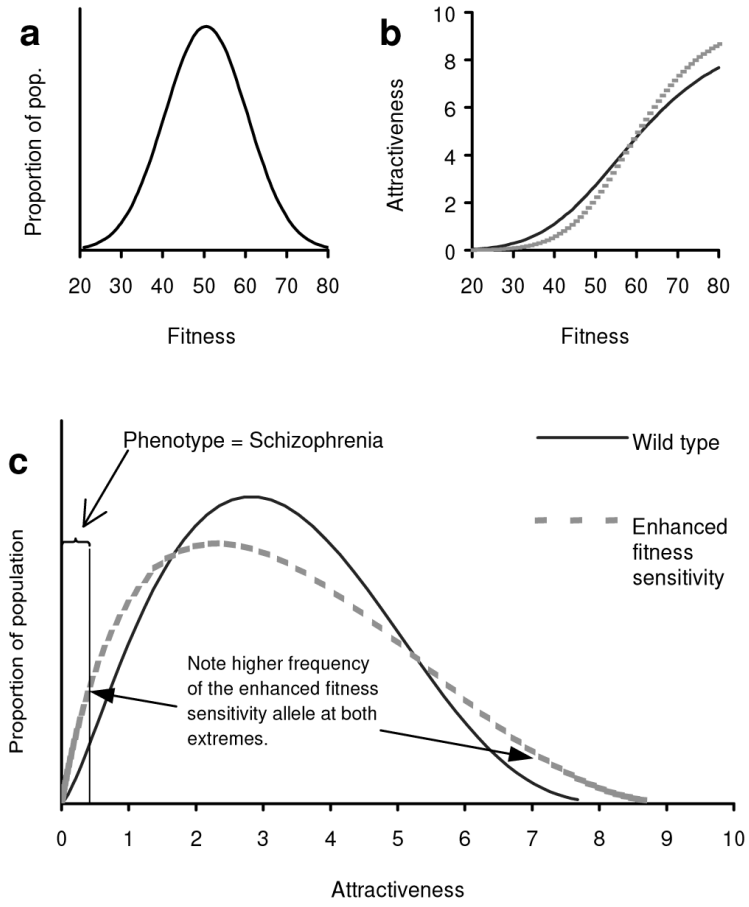

Figure 9–1. Hypothetical relationships among fitness, the attractiveness of an indicator trait, and the prevalence of schizophrenia.

a. Fitness (i.e., genetic quality) in the general population as a simple normal distribution displayed as T-scores with mean set at 50 and SD = 10. **b.** Attractiveness (on an arbitrary scale from zero to 10) expressed as two similar sigmiodal functions of fitness. **c.** Attractiveness in the general population. This is the result of applying the functions in "b" to the distribution in "a." We assumed that half the population has the wild type indicator and half have the enhanced fitness sensitivity indicator. Arbitrary parameters were set for both functions to illustrate how the "enhanced-sensitivity" function could produce greater proportions of the population at both the attractive and unattractive extremes. We chose a threshold that defines an unattractive extreme (which we hypothesize is identical to schizophrenia) containing one percent of the total population. In this illustration, that one percent comprises about one quarter percent with the wild type indicator and three quarters percent with the enhanced-sensitivity indicator.

affect tail size. By impairing anything from embryonic cell migration to adult feather preening, they disrupt tail development or maintenance enough that these peacocks tend to grow small, dull tails, at less than "1" on the attractiveness scale.

Thus, the tail's sensitivity to fitness converts otherwise subtle variation in fitness into obvious variation in attractiveness. But, if only those peacocks with the most attractive tails get to mate, why are there any offspring with unattractive tails in the next generation? This is the lek paradox.

The answer may lie in the new "bad genes" that arise during the formation of ova and (especially) sperm. The risk of a copying error in any one gene is very low. But so many genes influence tail size that there is high risk that at least one is copied incorrectly in each gamete. This is especially a problem in males, since sperm production involves many more cell-copying events than egg production does in females. For example, mature human females carry eggs that have gone through only about 20 DNA replications, whereas age-30 males carry sperm that have gone through about 380 DNA replications, and age-50 males carry sperm that have gone through about 840 DNA-replications (Crow, 2000). Thus, mutation load rises rapidly with paternal age, but not maternal age. This onslaught of new mutations in every generation—especially from older males—restores the distribution of heritable fitness in panel "a" and ensures a wide range of tail sizes, including small, dull ones, in every generation. This is a potential resolution of the "lek paradox."(Houle & Kondrashov, 2002; Kotiaho et al., 2001; Michod & Hasson, 1990; Pomiankowski & Moller, 1995; Rowe & Houle, 1996).

FITNESS INDICATORS IN ANIMALS INCLUDING HUMANS

Sexually selected fitness indicators have been found in a wide range of species. Many are bodily traits like the peacock's tail (Petrie, 1994). Others are behavioral (Andersson, 1994). For example, in several bird species, females prefer males with louder, more complex, and more numerous songs, and these measures correlate with various indices of fitness including nestling development (Nowicki, Hasselquist, Bensch, & Peters, 2000), immune function (Garamszegi, Moller, & Erritzoe, 2003), and longevity (Forstmeier, Kempenaers, Meyer, & Leisler, 2002).

Even insects use behavioral fitness indicators. At one point during fruit fly courtship, the female turns to face her pursuing suitor. He vibrates his wings in a characteristic pattern called "wing song." This vibrational song varies greatly among males within a population. It also varies

between geographically separated populations of the same species (Paillette, Bizat, & Joly, 1997)—as does human song. If the female likes her suitor's song, she allows him to mount her. If she doesn't like the song, she vibrates her wings in a characteristic rejection sound that is exactly the same all over the world. In *Drosophila montana*, females prefer a higher-frequency song (which requires faster, more energetic movements of the male's wings), and song frequency correlates with the survival rate of the male's progeny from egg to adulthood (Hoikkala, Aspi, & Suvanto, 1998).

When female choice predominates, indicators evolve in males only. However, when there is mutual choice, as exists in many socially monogamous birds and primates, indicators can evolve in both sexes (Andersson, 1994). For example, both male and female crested auklets sport a crest of feathers above their beaks, and both males and females prefer mates with larger crests (Jones & Hunter, 1993).

Are any human traits sexually selected? Recent evidence suggests that several human body traits may have evolved as fitness indicators through mate choice, including long head hair, expressive faces, everted lips, and hairless skin (Miller, 2000). Also, female choice has increased male height, upper-body muscularity, and facial masculinity (Perrett, May, & Yoshikawa, 1994), and male choice has increased female breast, hip, and buttock size (Etcoff, 1999). Thus, the mate-choice preferences contained within the brains of each sex in our species likely shaped the bodies of the opposite sex.

Of course, people looking for mates focus on far more than bodily traits. In courtship we play, dance, sing, embrace, and, most of all, we talk (and we talk *a lot*). On average, it takes about three months of frequent sex before a couple conceives its first child. Assume they talk just two hours per day at the typical rate of three words per second. In the three months before conceiving a child, they will exchange about a million words each—enough to fill six 500-page books (Miller, 2000).

What is the point of all this talk? One possibility is that courtship talk serves to reveal the quality of our genes. Not only can mate choice sculpt the bodies of the opposite sex, it can also influence the evolution of their brains and behavior. Preferences in the brains of women may have molded the brain structures underlying male verbal courtship behavior. Similarly, preferences in the brains of men may have affected female verbal courtship (Miller, 2000).

The idea that the human brain has played an active role in its own evolution is not new. Darwin (1871) argued that once the brain of any animal evolved the powers of mate choice, love, jealousy, and the appreciation of beauty, these would cause the brains of the opposite sex to evolve such mental traits as courage, pugnacity and perseverance, as well as bodily traits including size and strength, musical organs, bright colors, and ornaments.

Building on Darwin's insight, Miller (2000c) proposed that many human mental and behavioral abilities, such as language, music, humor and art—and the brain systems that support them—may have evolved as fitness indicators through mutual mate choice. For example, suppose that in our hominid ancestors, the brain systems responsible for primitive language were already somewhat sensitive to the fitness of the individual in which they develop. This might have been so because the necessary brain systems were sufficiently complex or energetically demanding that only hominids with the best genes for general fitness could grow those brain systems well. If so, then those who preferred verbally skilled mates would have secured better genes (with fewer mutations) for their offspring. Moreover, their offspring would inherit their parents' genes both for verbal skill and for preferring verbally skilled mates. The increasing correlation of these three kinds of genes—general fitness, indicator, and mate preference—would result in the rapid evolution of language as a fitness indicator. However, the process is not restricted to verbal skill. Any skill with some initial fitness-sensitivity could become the focus of mate choice and evolve, by this mechanism, into a far more elaborate fitness indicator. This reasoning sets the stage for our argument that schizophrenia represents a set of courtship mechanisms gone badly awry.

SCHIZOPHRENIA AS THE LOW FITNESS EXTREME OF A FITNESS INDICATOR

Suppose that every human embryo carries genetic instructions to grow and maintain complex brain systems for a particular set of courtship behaviors. For the moment, don't worry about exactly what those behaviors are. Just imagine that the brain systems needed to produce the behaviors are so complex or demanding of energy that their development and function are highly sensitive to overall genetic quality and environmental hazards. They grow correctly and perform best in the few individuals whose genes and environments are far above average. All others grow the systems with errors. The severity of errors depends on overall genetic quality and exposure to environmental hazards. This leads to great variation in the construction of the brain systems and great variation in their effectiveness during courtship—variation that correlates with underlying fitness.

At one extreme, those with high fitness and favorable environmental conditions develop and maintain the systems well, and display a highly effective version of this courtship behavior. They will show high mating intelligence, construed as display ability and behavioral attractiveness. The vast majority—who carry some fitness-reducing mutations or

encounter some environmental hazards—develop the systems with some errors, and display less effective versions of the behavior. An average mutation load leads to an average-quality fitness indicator. Those with high mutation loads and poor environments develop more fundamental errors in the brain systems that function as fitness indicators. They display an ineffective, unattractive version of this courtship behavior—very low mating intelligence and behavioral attractiveness. In 1 percent of the population, developmental errors are so severe that the brain systems produce the symptoms of schizophrenia rather than behaviors recognizable as courtship.

Note that we use the word "attractive" in the technical evolutionary-biology sense. It means "having the power to attract" mates, and not necessarily "pleasing or charming" in an aesthetic or moral sense. Indeed, by this definition, "attractive" behaviors could be deceptive or manipulative. Similarly, we use "unattractive" to mean lacking the power to attract mates; it does not mean undeserving of attention, concern, and care.

Now return to Figure 9–1 and apply it, not to peacock tails, but to the human courtship behaviors that go awry in schizophrenia. Imagine that we could pick out those human embryos with a fitness score of "75" (panel "a"). As their brains develop (both before and after birth), these embryos have the "good genes" needed to ensure precise cell migration, differentiation, synaptogenesis, and programmed cell death, despite environmental threats to these processes such as malnutrition, hypoxia, and infection. As a result, their brain systems for our presumed "particular set of courtship behaviors" develop well and produce, on average, a highly attractive version of the behaviors—around "7" on the attractiveness scale (which could also be interpreted as a 'mating intelligence' scale).

Now imagine we could pick out human embryos with a fitness score of "35." As they attempt to grow these complex and fitness-sensitive brain systems, their "bad genes" interfere with crucial developmental processes. As adults, their aberrant brain systems perform these courtship behaviors poorly, and they score, on average, less than "1" on the attractiveness scale. In some, the behavior is so disrupted that it no longer resembles courtship and, instead, shows the characteristic symptoms of schizophrenia. "Bad genes," thus, may be responsible for the persistence of schizophrenia, just as they are responsible for the persistence of small, dull peacock tails.

Further, our hypothesis suggests a second kind of gene (in addition to fitness-reducing mutations or "bad genes"), which could also increase the risk for schizophrenia. Ornaments may evolve through the successive accumulation of genes that increase fitness sensitivity (Hasson, 1989; Pomiankowski & Moller, 1995; Rowe & Houle, 1996). Suppose that this applies to the evolution of the brain systems that go awry in schizophre-

nia. If such genes persist, then some families and lineages will show higher fitness-sensitivity than others. They may produce more geniuses (individuals with very high-quality mental fitness indicators), but also more individuals with schizophrenia (individuals with very low-quality mental fitness indicators). This is not necessarily because their overall average fitness is higher or lower than average, but because they have alleles that increase neuro-developmental risk-seeking. They go for broke. Sometimes, this pays off with astonishing creativity or brilliance, but sometimes it leads to disastrous psychosis. Albert Einstein, John Nash, and James B. Watson all got the Nobel Prize, and they all had sons with severe schizophrenia.

WHAT COURTSHIP ABILITIES GO WRONG IN SCHIZOPHRENIA?

The question is difficult to answer. Biologists usually analyze fitness indicators starting from the attractive extreme, observing that individuals with the brightest feathers or loudest calls attract the most mates. In contrast, our theorizing begins at the other extreme, with reports that schizophrenia markedly reduces marriage rates and reproduction (reviewed in Haukka et al., 2003). We speculate that the symptoms of schizophrenia (including delusions, disorganized speech, blunted affect, poor sense of humor, and social awkwardness) reduce reproductive success by impairing courtship ability—by undermining mating intelligence. If so, what is the normal mental adaptation that goes wrong in schizophrenia?

One possibility is creative verbal courtship. The behavioral symptoms of schizophrenia might be extremely aberrant versions of uniquely human verbal courtship behaviors. By "verbal courtship" we mean more than successful pick-up lines by males to attract females. Instead, we imagine a complex verbal "dance" of mutual mate choice and display, in which each potential mate tries to talk in ways that will be interesting and attractive, given the other's beliefs, desires, interests, and attitudes. This requires fluent coordination among many psychological adaptations, including those for listening, perspective-taking, personality-assessment, planning, and talking. These brain systems are probably very complex and their development may therefore be vulnerable to mutations at many loci, and to a wide range of environmental hazards.

Return to Figure 9–1 once more and imagine that the x-axis of panel "c" represents the attractiveness of verbal courtship. Embryos drawn from the high-fitness extreme can correctly develop the complex brain systems needed for successful verbal courtship. Suppose that, as adults, these complex brain systems can generate many possible conversational gambits and critique, practice, and improve the gambits using an evolving model

of the potential mate's mind. The end results include interesting utterances, enjoyable conversation, high mating intelligence, and ultimately, high mating success.

Good conversation requires rapid, semi-conscious planning of one's utterances, including internal self-criticism. Disrupted development might impair the effectiveness and accuracy of this process of internal critique. The internal-utterance critic might fail to appreciate which ideas others will believe and what sequence of ideas others will be able to follow. This could explain why the speech of people with schizophrenia usually contains delusions and is often disorganized. If this internal-utterance critic develops aberrantly, so it connects too strongly to auditory systems, it might be experienced as derogatory auditory hallucinations. For example, many people with schizophrenia hear an insulting voice commenting disdainfully on their thoughts and behaviors. Often, this internal voice is experienced as older, higher-status, and better-educated (Nayani & David, 1996).

Language abnormalities are common in schizophrenia (reviewed in Covington et al., 2005), and people with schizophrenia have deficits in verbal humor, and in the ability to represent the beliefs, thoughts and intentions of other people (reviewed in Brune, 2005). However, schizophrenia tends to disrupt many other courtship-related skills in addition to verbal courtship. So, some of its symptoms may reflect low-fitness extremes of other fitness indicators. These may include (1) capacities for musical rhythm and dance (schizophrenia impairs sense of rhythm and motor coordination (reviewed in Boks, Russo, Knegtering, & van den Bosch, 2000)), (2) capacities for humor (schizophrenia impairs sense of humor, wit, and joke-production ability), (3) capacities for happy socializing (schizophrenia leads to social withdrawal, flat affect, and anhedonia), and (4) capacities for empathic Theory of Mind (schizophrenia impairs perspective-taking accuracy, increases paranoia, and increases selfishness and narcissism). Thus, in many ways, schizophrenia is the mirror-image of mating intelligence—it is what happens when many courtship abilities go amiss in parallel.

SEXUAL RIVALRY AND SCHIZOPHRENIA

To illustrate our hypothesis, we have focused on mate choice. However, some sexually selected traits evolved both as weapons *and* ornaments. For example, elk with the biggest antlers win contests over females (Berglund, Bisazza, & Pilastro, 1996). In addition, females prefer males with larger antlers (Fiske, Rintamaki Pekka, & Karvonen, 1998). Suppose human language evolved for both contests and courtship. Those who could model the minds of potential mates and produce more attractive verbal gambits

could have used the same brain systems to model the minds of sexual rivals and produce more intimidating verbal gambits (Miller, 2000). For example, a low mutation load might allow individuals to develop complex brain systems that enable them to detect sexual rivals, subconsciously generate many possible intimidating gambits, and subconsciously critique, practice, and improve the gambits using a constantly updated model of the rival's mind. The end result is successful intimidation and high mating success. Disrupted development might lead to inaccurate detection of rivals—expressed as persecutory delusions and insulting or threatening auditory hallucinations. This may explain why the typical auditory hallucination in schizophrenia—a short, obscene, coarse, or sexually toned insult (Nayani & David, 1996)—closely resembles a derogatory remark to or about a sexual rival (Buss & Dedden, 1990). Disrupted development might also lead to poor attempts at intimidation—expressed as grandiose delusions, which, in this context, can be viewed as overly obvious bragging. Thus, schizophrenia may represent severely impaired mating intelligence in both the domains of inter-sexual attraction and intra-sexual rivalry.

THE CONTINUUM OF PSYCHOSIS AND CONTINUOUS VARIATION IN INDICATOR QUALITY

One of the implications of our hypothesis is that schizophrenia is not so discrete a condition as one might suppose. Across individuals, sexually selected fitness indicators vary greatly and continuously in size, color intensity, loudness, pitch, etc. One peacock in a population must have the smallest tail, but several more have tails nearly as small. This may explain why the symptoms of schizophrenia appear to lie on the same continuum with the experiences of people in general (diagnosed with a mental disorder or not) (Strauss, 1969).

For example, several disorders known in composite as the *schizophrenia spectrum* are genetically linked to schizophrenia (Parnas et al., 1993). The most-studied is schizotypal personality disorder (SPD), which includes multiple oddities of perception, thought, emotion and behavior, but not psychotic symptoms (American Psychiatric Association, 2000). Compared with the general population, SPD is five times more common among the close relatives of people with schizophrenia (Kendler et al., 1993; Parnas et al., 1993). If the SPD phenotype lies adjacent to schizophrenia, near the unattractive extreme of the same indicator trait (see Figure 9–1), this would explain several facts about SPD including its association with developmental abnormalities similar to those found in schizophrenia (e.g., Takahashi et al., 2004), and its frequent improvement with dopamine antagonists (Koenigsberg et al., 2003).

In addition, a surprisingly large number of individuals without mental disorders have experienced hallucinations and delusions (Eaton, Romanoski, Anthony, & Nestadt, 1991; Strauss, 1969). In one study (van Os, Hanssen, Bijl, & Ravelli, 2000), non-mentally-ill people who reported these symptoms resembled those with schizophrenia in that they were more likely to be young, single, city dwellers with less education, poorer quality of life, and blunting of affect. In another study, delusions and hallucinations were common among patients attending a medical care clinic (even among those with no psychiatric treatment history) (Verdoux et al., 1998). These symptoms were most common in people 18 to 29 years old, and became less common with increasing age. This age distribution resembles the distribution of schizophrenia's age at onset (Hafner, Maurer, Loffler, & Riecher-Rossler, 1993), and suggests a shared mechanism. That mechanism could be the development of the sexually selected fitness indicator we propose.

EXPLANATORY AND PREDICTIVE POWER OF THE FITNESS INDICATOR MODEL

All of our explanations and predictions depend on seven generic properties of sexually selected fitness indicators. Moreover, it doesn't matter whether the relevant fitness indicator is verbal courtship, verbal intimidation, sense of humor, or rhythmic dance, because our basic explanations and predictions apply to the low-fitness extremes of *any* sexually selected fitness indicator. They apply not only to schizophrenia but also to small dull peacock tails, and low-frequency wing song in fruit flies. If our predictions hold up to empirical scrutiny, then further research will clarify which fitness indicators go awry in schizophrenia.

1. Indicators are displayed during courtship.

This leads to the general prediction that anything which stimulates courtship will precipitate or worsen schizophrenia. Peahens can't see that a peacock has a small, dull tail until the peacock matures, courts peahens, and unfurls its tail. So, if schizophrenia is analogous to a small tail, it should not be apparent to others until the age at which courtship and sexual competition usually begin, and when mating intelligence becomes important. This may explain schizophrenia's typical onset in adolescence and early adulthood. Although neurodevelopmental precursors of schizophrenia appear long before puberty (Woods, 1998), schizophrenia itself is rare before puberty, and most cases begin between the ages of 15 and 26 (Hafner et al., 1993)—a time of peak mating effort in those without schizophrenia.

Of course, other maturational hypotheses are consistent with adolescent onset. Ours, however, leads to several specific predictions. First, because the age at onset of courtship varies across populations, we predict that the average age of onset of schizophrenia will be correlated across different human groups (e.g., sexes, ethnic groups, races, and birth cohorts) with the average age at which courtship begins. For example, if courtship begins 3 years earlier in one ethnic group than another (e.g., as indexed by mean age at first kiss or first sexual intercourse), we predict that schizophrenia will also begin 3 years earlier in that ethnic group. Note that it does not matter whether the difference in age at onset of courtship is due to genes or culture or both—the prediction still holds.

Second, the lifetime course of schizophrenia symptoms should parallel age-specific changes in mating effort in the general population. Symptoms should peak in severity at the age of peak mating effort, and often spontaneously remit as mating effort declines in the 40s and 50s. Likewise, having children in a stable, supportive, sexual relationship should often reduce symptom intensity, as mating effort gives way to parenting effort.

Third, situations that stimulate courtship and sexual competition in normal individuals (for example, dating, falling in love, being derogated by a sexual rival, getting divorced) should precipitate or worsen schizophrenia. Sexual interest in a potential mate, coupled with being verbally derogated by a sexual rival, should be a particularly powerful trigger for a psychotic 'first break.' More subtle forms of sexual competition (e.g., for wealth and status) should also precipitate or exacerbate symptoms of schizophrenia. Such social stimulation of courtship and sexual competition might explain the high rate of schizophrenia among immigrants (Cantor-Graae & Selten, 2005) and city dwellers (Marcelis, Navarro-Mateu, Murray, Selten, & van Os, 1998). Racial and ethnic discrimination might force immigrants to compete harder for wealth and status (a form of sexual competition), while cities might function as vast leks, providing frequent encounters with both potential mates and sexual rivals.

Fourth, drugs that block courtship should improve schizophrenia. If we are correct that schizophrenic behaviors are dysfunctional versions of courtship behaviors, then drugs that increase or decrease courtship behaviors in normal individuals should have the same effect on schizophrenic behaviors. For example, in a wide range of species including crabs, birds, rats, flies, monkeys and humans, dopaminergic drugs alter courtship. Dopamine agonists, like amphetamine, stimulate courtship, while antagonists, like haloperidol, inhibit courtship (Chang et al., 2005; Melis & Argiolas, 1995; Wood, 1995). Consistent with our prediction, dopamine agonists, like amphetamine, worsen schizophrenia, while dopamine antagonists, like haloperidol, improve it (Kahn, 1995). This suggests that other drugs which reduce courtship behaviors may prove therapeutic for

schizophrenia. Investigators could find them by developing animal models of courtship and searching for drugs that block courtship but leave other behaviors unaffected. For example, drugs that specifically block the recently discovered ultrasonic courtship song of male mice (Holy & Guo, 2005) may also reduce the symptoms of schizophrenia.

2. Indicators affect the probability of mating.

As the unattractive extreme of a fitness indicator, schizophrenia should impair the ability to attract and retain mates. This notion could explain reduced rates of marriage (15–73 percent of normal) and reproduction (30–70 percent of normal) among individuals with schizophrenia (reviewed in Haverkamp, Propping, & Hilger, 1982). The reduced rate of reproduction among those with schizophrenia is probably due to failure to attract a mate, rather than physiological infertility, because those who do marry report nearly normal numbers of children (Nanko & Moridaira, 1993).

3. Indicators show predictable sex differences.

Even in socially monogamous species like humans, males show higher reproductive skew and higher variation in reproductive success. Compared with females, a higher proportion of males attract multiple mates, and a higher proportion of males attract no mates. Thus, males are subject to somewhat stronger sexual selection, and they court and compete earlier, more frequently, and more intensely (Andersson, 1994; Miller, 2000). This may explain why schizophrenia begins earlier and is more often severe in males (Jablensky, 2000)—despite minimal sex differences in schizophrenia's overall prevalence. The genetic, hormonal, and neurophysiological sex differences that accelerate and amplify male mating effort also amplify any abnormality, such as schizophrenia, which represents the unattractive extreme of the indicator.

4. The development of indicators is sensitive to fitness and condition.

This quality of fitness indicators permits them to perform their main evolutionary function—to convert otherwise subtle variation in genetic fitness into obvious variation in attractiveness, and thereby to make it easier for the opposite sex to choose high-quality mates. This may explain several facts about schizophrenia. (1) Abnormal brain development is common (Woods, 1998; Yeo et al., 1999) because fitness indicators reveal poor fitness through disordered development. (2) Polygenic inheritance underlies

schizophrenia (McDonald & Murphy, 2003; Tsuang, Stone, & Faraone, 2001) because an indicator must have a large "mutational target" in the genome (see Keller, this volume). That is, it must be sensitive to many loci to adequately reflect overall heritable fitness (Houle, 1998; Houle & Kondrashov, 2002). (3) Schizophrenia is associated with environmental hazards such as prenatal exposure to infection (e.g., Brown et al., 2004), famine (e.g., St. Clair et al., 2005), and hypoxia (Cannon et al., 2002), because the environmental sensitivity of fitness indicators amplifies their ability to reveal bad genes. (4) Compared with those in the general population, people with schizophrenia have a higher rate of death (at all ages)—mostly from a wide range of physical illnesses, not just suicide and drug abuse (e.g., Osby, Correia, Brandt, Ekbom, & Sparen, 2000)—because the same "bad genes" that reduce fitness and cause physical illnesses also disrupt the fitness indicator and cause schizophrenia.

More speculatively, the idea of fitness-sensitivity offers an additional and complementary explanation (see prediction one, above) for the high rate of schizophrenia among immigrants and city dwellers—sensitivity to fitness and condition favors locally adapted individuals. Theoretically, peacocks living in the environment to which their ancestors adapted should grow larger, more attractive tails than immigrant peacocks whose ancestors were better-adapted to a different environment. That's because the fitness distribution for the immigrant population (in their new environment) is shifted lower (to the left in Figure 9–1, panel a). Indeed, the females of several species prefer locally adapted males, and simulations show that the local condition-dependence of sexually selected traits could account for the evolution of such preferences (Proulx, 2001; Reinhold, 2004).

Afro-Caribbean immigrants to the U.K. and the Netherlands are physiologically adapted to the pathogens, parasites, toxins, and other ecological challenges of their homelands, more so than they are to the ecological challenges of Northern Europe. During development (both before and after birth), they encounter environmental hazards to which their immune systems and other development-stabilizing systems are not adapted. These hazards impair condition and thereby interfere with development of the relevant brain systems. This might account for their unusually high rate of schizophrenia.

The same logic may explain why those born in cities develop schizophrenia at a higher rate than those born in rural environments. Compared with rural environments, cities may contain new and rapidly changing environmental hazards (e.g., more virulent pathogens and nastier neurotoxins). If so, the fitness distribution for those born in cities would be shifted lower (to the left in Figure 9–1, panel "a"), thereby increasing the proportion with schizophrenia.

5. "Bad genes" cause most of the heritable variation in the attractiveness of indicators.

As we showed earlier, embryos with the most "bad genes" are the most likely to grow small tails or, according to our hypothesis, develop schizophrenia. Thus, we predict that most of the genes responsible for schizophrenia will be mutations that reduce general fitness (e.g., by impairing embryonic cell migration or immunity). Because they reduce fitness, they are evolutionarily transient (selection removes them eventually), and they can't spread widely across human populations (Keller & Miller, in press) Instead, they remain lineage-specific until they are ultimately removed. Such "bad genes" may explain why schizophrenia persists at such a high prevalence, and why, despite its high heritability, decades of gene-hunting have found so few susceptibility alleles that replicate across populations (McDonald & Murphy, 2003; Tsuang et al., 2001).

6. Genes may increase the fitness-sensitivity of indicators.

This prediction concerns a second type of susceptibility gene that should replicate better across populations. Evolutionary biologists have proposed that extravagant traits evolved through the successive accumulation of genes that increase fitness sensitivity (Hasson, 1989; Pomiankowski & Moller, 1995; Rowe & Houle, 1996). That is, the preference of ancestral peahens for the peacock with the biggest tail favored genes that produced larger tails in high-fitness peacocks, even at the cost of smaller tails in low-fitness peacocks. A series of such genes spread throughout the population (i.e., went to fixation) such that modern peacock tails are large and highly sensitive to fitness.

Suppose that a given courtship trait underlying schizophrenia evolved in the same manner. Such a courtship trait (perhaps verbal courtship) would have become increasingly elaborate and fitness-sensitive, until further increases imposed a net disadvantage. If this limit were identical in all human populations, then there would be no genetic differences affecting fitness sensitivity. All of the variation in the courtship trait and in the rate of schizophrenia would arise from differences in genetic quality (i.e., "bad genes," mutation load) and exposure to environmental hazards.

However, the optimal degree of fitness sensitivity depends on factors that differ between human populations, and that continue to change within populations. For example, higher rates of polygyny and/or extrapair copulations ("infidelity") lead to more intense sexual competition, which would favor higher fitness-sensitivity. The rates of polygyny and infidelity have probably varied geographically and temporally across

human evolution. So, different modern human populations may include different proportions of higher fitness-sensitivity alleles (that were favored by more intense sexual competition), and lower fitness-sensitivity alleles (that were favored by less intense sexual competition as occurs in monogamy). These fitness-sensitivity alleles are further mixed by migration and mating between groups.

Alleles for higher fitness-sensitivity would produce more successful courtship among the few with high overall fitness, at the expense of even less successful courtship, and an increased rate of schizophrenia, among less fit individuals. The dashed curve in Figure 9–1 labeled "enhanced fitness-sensitivity allele," in panels "b" and "c" shows the effect of such an allele on the relationship between fitness and attractiveness and on the distribution of attractiveness. Such sensitivity-boosting alleles would be more common among people with schizophrenia and in their relatives. This allows us to make several predictions.

First, compared with the general population, the relatives should show higher variance in the relevant courtship trait, such as verbal-courtship ability. They should also show higher variance in the anatomical and neurophysiological bases (or endophenotypes (Cadenhead & Braff, 2002)) for that trait. Second, they should show higher variance in observer-rated sexual charisma, psychological attractiveness, and mating success. In some populations, the increased variance will result in a net increase in reproductive success among relatives, but this depends on whether the current rates of polygyny and infidelity are high enough to favor enhanced fitness-sensitivity alleles. This may explain the higher-than-average reproduction rates sometimes observed in unaffected relatives of schizophrenics (reviewed in Haukka et al., 2003), and in those with mild schizotypy (Nettle & Clegg, 2006). Third, we should find a higher prevalence of schizophrenia, especially among males, in historically polygynous populations with high reproductive skew, in which increased sexual competition would have favored enhanced fitness-sensitivity alleles.

To find such genes, investigators should begin with endophenotypes (quantifiable aspects of brain structure and physiology) that (1) are abnormal in schizophrenia, (2) have the highest variance in the general population, (3) have even higher variance among the relatives of schizophrenics, and (4) are plausibly related to mating intelligence. More specifically, investigators should focus on endophenotypes that have a high coefficient of additive genetic variation, indicating that many mutation-vulnerable loci are responsible for the phenotypic variation (Houle, 1992; Miller & Penke, in press).

7. Mate preferences co-evolve with the indicator.

Well-developed versions of an indicator trait are perceived as sexually attractive, and poorly developed versions of the trait are perceived as

sexually unattractive (Andersson, 1994; Kokko, Brooks, Jennions Michael, & Morley, 2003). If schizophrenia is the unattractive extreme of an indicator, this would explain why people with schizophrenia suffer so much stigmatization across cultures (Dickerson, Sommerville, Origoni, Ringel, & Parente, 2002). This view also predicts that anti-schizophrenia bias should increase after puberty (when mate-choice systems mature), and should be more severe in females (who are typically choosier about their sexual partners), especially when females are ovulating (when it is most important to focus on indicators of "good genes") (Gangestad, Thornhill, & Garver, 2002; Penton-Voak et al., 1999). Thus, the stigmatization of the mentally ill could be viewed as an adaptive component of mating intelligence, in the form of discriminative mate choice. Such stigmatization, like that of the physically handicapped or mentally retarded, may be morally unwarranted, politically undesirable, and socially oppressive—but it may have a hidden adaptive logic that explains its pervasiveness as part of human nature.

APPLICATIONS TO OTHER MENTAL DISORDERS

Are any other mental disorders analogous to small, dull peacock tails?

One possibility concerns emotional instability (Costa et al., 1992), a basic component of many psychological disorders. Emotional stability likely requires complex and energetically demanding brain systems to 'stay cool' (coordinate adaptive behavior without losing focus) while simultaneously detecting, analyzing, and responding to a wide range of challenges (especially the challenges of courtship and sexual competition). Such brain systems might be sensitive to fitness, such that coolness or emotional stability would vary according to mutation load. A preference for mates with this ability (as documented in 62 cultures by Schmitt et al., 2004) could have fueled the evolution of calm self-control as a fitness indicator. At one extreme, those with high fitness (low mutation load) might grow these brain systems well and appear cool under the most stressful situations (think James Bond). At the other extreme, in those with low fitness, a wide range of fitness-reducing mutations might interfere with the development of these brain systems such that individuals appeared much more anxious in a much wider variety of situations (think Woody Allen). That is, they would appear neurotic. Indeed, neuroticism is another name for the low extreme of emotional stability, which is one of the 'Big Five' personality traits (Costa, McCrea, & Psychological Assessment Resources Inc., 1992). Extreme neuroticism is seen in generalized anxiety disorder and in many phobias. From this point of view, coolness evolved through sexual selection to advertise that we are not neurotic, and are likely to have better

genes. A similar case could be made for depression as the unattractive extreme of happiness as a sexually selected fitness indicator.

But do any mental disorders other than schizophrenia behave as we expect of the unattractive extremes of fitness indicators? To address this question, we believe it is useful to consider particular disorders *vis à vis* the seven generic properties of fitness indicators described above. Many common mental disorders meet the criteria for heritability, prevalence, and stigmatization, but less is known about mating success, sex differences, age profiles, and paternal age effects. For instance, bipolar disorder meets some of the criteria. It begins in adolescence and early adulthood (Costello et al., 2002), affects about one percent of the population (Bauer & Pfennig, 2005), is highly heritable (Kieseppa, Partonen, Haukka, Kaprio, & Lonnqvist, 2004), and depends on many genes (Kennedy, Farrer, Andreasen, Mayeux, & St. George-Hyslop, 2003) However, its effect on lifetime reproductive success is largely unknown. Indeed, those with bipolar could be viewed as implementing the risk-seeking strategy by cycling between high-mating-intelligence states (mild mania) and low-mating-intelligence states (depression) within their life-times. If the reproductive payoffs of occasional mania out-weigh the reproductive costs of depression, bipolar disorder can persist evolutionarily. The difference between bipolar and 'normal' individuals may be that bipolar people have endogenously driven mania cycles, whereas 'normal' individuals have more context-sensitive mania states of high mating intelligence, which they call 'being in love.'

If other mental disorders are the low-fitness extremes of fitness indicators, this may explain why people with one mental disorder often have another mental disorder, far more often than would be expected by chance. Indeed, our hypothesis predicts three sources of co-morbidity: overlapping "mutational targets," overlapping environmental risk factors, and the power of sexual selection to concentrate "bad genes" in an unlucky minority of the population. An indicator's "mutational target" comprises all the genetic loci that can potentially affect the trait's quality (see Keller, this volume) (Keller & Miller, in press). Suppose the mutational targets of two fitness-indicator traits overlap, such that some of the mutant alleles that disrupt one indicator also disrupt the other. This would increase the odds that an individual carrying those mutant alleles develops both disorders. Comorbidity could also arise if two different fitness indicators share sensitivity to the same environmental risk factors, such as prenatal infection, birth trauma, starvation, head injury, or social isolation.

A third reason for the high co-morbidity of mental disorders is that sexual selection leads to assortative mating, which aggregates "bad genes" in a subset of the population (J. F. Crow & Kimura, 1979). If both height and intelligence are preferred by both sexes, then genes for height and

intelligence will tend to end up in the lucky offspring of highly desired parents—but genes for being short and simple will also end up together, in the unlucky offspring of less-desired parents (see Kanazawa & Kovar, 2004). To the extent that mental disorders are associated with fitness-reducing alleles that are avoided in mate choice, they too will aggregate in the lower-fitness offspring of less-desirable parents. This is true even without overlapping mutational targets or shared environmental sensitivities. This effect may be augmented if "bad genes" also undermine mating intelligence construed as mate-choice accuracy—so that individuals with mental disorders are less sexually discriminating (i.e., less biased, more accepting) against others with mental disorders.

A corollary of these explanations for comorbidity is that individual mental disorders may be our way of lumping together several conditions that co-occur more frequently than not. For example, schizophrenia symptoms form natural groups or clusters often labeled "positive," "negative," and "disorganized" (Arndt, Alliger, & Andreasen, 1991). These natural groups may represent the unattractive extremes of different sexually selected fitness indicators. In this view, the unattractive extremes often appear in the same individuals because the relevant mutational targets and environmental sensitivities overlap extensively, and because "bad genes" are concentrated in a subset of the population. Because the unattractive extremes are more likely to occur together than separately, they appear to be a syndrome—one that Bleuler (1911) labeled 'schizophrenia.' This is important because it suggests the possibility that various symptoms occur together in schizophrenia, not because they share any proximate causes or common endophenotyopes, but because they share an ultimate cause—sexual selection for fitness indicators.

PARENTAL SELECTION AND CHILDHOOD MENTAL DISORDERS

Sexual selection is not the only form of directional selection that can produce fitness indicators. In many species, siblings must compete for scarce parental resources. At the same time, parents must allocate scarce resources to those offspring most likely to survive and reproduce. This conflict has produced a vast array of bodily and behavioral traits in both parents and offspring (reviewed extensively in Mock & Parker, 1997). In some species, offspring have evolved traits that advertise fitness and thereby attract parental care and feeding. For example, healthy barn swallow nestlings beg for food with wide-open mouths (or gapes) colored bright red. The color fades to dull yellow when nestlings are sick because they must divert the crucial carotenoid pigments to immune function instead of gape color. Thus, gape color serves as a fitness indicator, and

parents preferentially feed nestlings with bright red gapes (Saino et al., 2000). In North American coots, bright orange filaments cover the heads of chicks, only to be shed just before fledging. Experimental manipulation of the filaments shows that they attract parental feeding and may serve as a fitness indicator (Lyon, Eadie, & Hamilton, 1994).

Could any childhood-onset mental disorders be the equivalent of dull gapes in barn swallow nestlings or dull filaments in coot chicks? One possibility is autism. Suppose that the ability of very young children to charm their parents—through language, facial expression, creative play, and coordinated social interaction—evolved as a parentally selected fitness indicator (Miller, 2000, p. 216–217). More articulate, expressive, playful, and socially engaged children would give a reliable warranty of their genetic and phenotypic quality, so would solicit higher parental investment. Young children would vary greatly in their ability to charm parents and that variation would correlate with underlying fitness. Autism could represent the least charming, low-fitness extreme of this variation—accounting not only for the typical symptoms of autism, but also for the extreme frustration and alienation experienced by parents of autistic children.

Such a view would lead to explanations and predictions much like those regarding schizophrenia, since the evolutionary mechanism is quite similar, except that parents rather than mates make the selection. Evolutionary biologists have discovered many other adaptations for sibling rivalry and for allocating parental resources that might help explain the behavior of human children and their parents. The approach might even lead to a subsequent book entitled "Parenting Intelligence."

CONCLUSION

Many animals, including humans, prefer mates with better-quality genes. This preference appears to have driven the evolution of bodily and behavioral displays of fitness known as sexually selected fitness indicators. Both the preference for fitness and the corresponding behavioral displays of fitness can be viewed as important components of mating intelligence. If so, then the evolutionary biology of fitness indicators may lead to a deeper understanding of human mating intelligence. Here we have argued that any behaviors which evolved as sexually selected fitness indicators will have low-fitness, unattractive, or unsuccessful extremes that may correspond to mental disorders. Just as pathology illuminates physiology, these low extremes of mating intelligence can illuminate the high extremes and the normal variation.

As an example, we have discussed schizophrenia. Because we focused on fitness-indicator theory, our hypothesis differs from previous evolu-

tionary hypotheses regarding schizophrenia in several ways. First, it does not propose that schizophrenia itself is adaptive, or that the responsible genes produce consistent fitness benefits among the relatives. Second, our model does not propose that schizophrenia arises from any typical etiology—any small set of predictable defects in genes, neurodevelopment, or neurophysiology. Rather, it predicts that most of the responsible genes will be a large number of fitness-reducing mutations with a wide range of harmful effects on development, physiology, immunity, and other vital processes. Third, our model at this stage does not predict exactly which brain systems—which forms of mating intelligence—go awry in schizophrenia, only that they will be brain systems required for sexual attraction of mates and/or sexual competition against rivals. Verbal courtship is one possibility, but none of our explanations or predictions hinge on whether the indicator trait is verbal courtship or something else.

Even without specifying the relevant courtship trait, our hypothesis explains many key features of schizophrenia, including onset in adolescence and early adulthood, greater severity and earlier age at onset in males, reduced reproductive rate, substantial heritability, polygenic basis, the failure of psychiatric genetics to find replicable risk alleles of major effect size, frequent developmental abnormalities, increased mortality, association with prenatal environmental hazards, the treatment efficacy of dopamine antagonists, and cross-culturally severe social stigmatization. It also leads to some surprising and testable predictions.

Our hypothesis also resolves the evolutionary paradox that has baffled schizophrenia researchers for decades: its persistence across generations and cultures despite impairing both survival and reproduction and despite its substantial heritability (which should have allowed selection to eliminate it). The attractive extreme of any fitness indicator is attractive precisely because its development is so easily disrupted by fitness-reducing mutations and environmental hazards. For this reason, every fitness indicator must also include a low-fitness, unattractive extreme.

If, as we propose, schizophrenia is the unattractive extreme of a fitness indicator, then schizophrenia persists as an inevitable and distinctively human side effect of sexual selection for some distinctively human mode of courtship, probably involving language and social cognition. Our hypothesis also suggests the possibility that other mental disorders (e.g., depression and anxiety disorders) are low-fitness extremes of fitness indicators, and it lays out the criteria for empirically addressing this idea. If other disorders meet these criteria, such a pattern would explain the high comorbidity of mental disorders.

We are not the first to consider sexual selection in schizophrenia. Both Crow (1995) and Randall (1998) proposed roles for sexual selection, but neither addressed its effects on the genetic and phenotypic variance of sexually selected traits. Consequently, Crow postulated a single-gene model,

and Randall proposed that females perpetuate susceptibility alleles by reproducing before illness onset. Neither model is plausible given evolutionary genetics and ancestral reproduction patterns (see Keller, this volume; Keller & Miller, in press). We may be the first to use fitness-indicator theory to explain the evolutionary origins, genetic basis, and characteristic symptoms of schizophrenia. In the context of this book, schizophrenia and other mental disorders are not just clinical curiosities; they reveal the awesome adaptive complexity of mating intelligence in normal individuals by showing how many ways it can go wrong in the unlucky few.

REFERENCES

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders : DSM-IV-TR* (4th, text revision. ed.). Washington, DC: American Psychiatric Association.

Andersson, M. (1994). *Monographs in behavior and ecology: sexual selection*. Princeton, NJ: Princeton University Press.

Arndt, S., Alliger, R. J., & Andreasen, N. C. (1991). The distinction of positive and negative symptoms. The failure of a two-dimensional model. *British Journal of Psychiatry, 158*, 317–322.

Bauer, M., & Pfennig, A. (2005). Epidemiology of bipolar disorders. *Epilepsia, 46 Suppl 4*, 8–13.

Berglund, A., Bisazza, A., & Pilastro, A. (1996). Armaments and ornaments: An evolutionary explanation of traits of dual utility. *Biological Journal of the Linnean Society, 58*(4), 385–399.

[JTP1] Bleuler, E. (1911). Dementia Praecox oder gruppe der schizophrenien. In G. Aschaffenburg (Ed.), *Handbuch der psychiatrie*. Leipzig und Wien: Franz Deuticke.

Boks, M. P. M., Russo, S., Knegtering, R., & van den Bosch, R. J. (2000). The specificity of neurological signs in schizophrenia: a review. *Schizophr Res, 43*(2–3), 109–116.

Brown, A. S., Begg, M. D., Gravenstein, S., Schaefer, C. A., Wyatt, R. J., Bresnahan, M., et al. (2004). Serologic evidence of prenatal influenza in the etiology of schizophrenia. *Arch Gen Psychiatry, 61*(8), 774–780.

[JTP2] Brune, M. (2004). Schizophrenia-an evolutionary enigma? *Neurosci Biobehav Rev, 28*(1), 41–53.

[JTP3] Brune, M. (2005). "Theory of Mind" in Schizophrenia: A Review of the Literature. *Schizophr Bull, 31*(1), 21–42.

Buss, D. M., & Dedden, L. A. (1990). Derogation of competitors. *Journal of Social and Personal Relationships, 7*, 395–422.

[JTP4] Cadenhead, K. S., & Braff, D. L. (2002). Endophenotyping schizotypy: a prelude to genetic studies within the schizophrenia spectrum. *Schizophr Res, 54*(1–2), 47–57.

[JTP5] Cannon, T. D., van Erp, T. G., Rosso, I. M., Huttunen, M., Lonnqvist, J., Pirkola, T., et al. (2002). Fetal hypoxia and structural brain abnormalities in schizophrenic patients, their siblings, and controls. *Arch Gen Psychiatry, 59*(1), 35–41.

Cantor-Graae, E., & Selten, J. P. (2005). Schizophrenia and migration: a meta-analysis and review. *Am J Psychiatry, 162*(1), 12–24. [JTP6]

Cardno, A. G., Marshall, E. J., Coid, B., Macdonald, A. M., Ribchester, T. R., Davies, N. J., et al. (1999). Heritability estimates for psychotic disorders: the Maudsley twin psychosis series. *Arch Gen Psychiatry, 56*(2), 162–168. [JTP7]

Chang, H. Y., Grygoruk, A., Brooks, E. S., Ackerson, L. C., Maidment, N. T., Bainton, R. J., and Krantz, D. E. (2005). Overexpression of the Drosophila vesicular monoamine transporter increases motor activity and courtship but decreases the behavioral response to cocaine. *Mol Psychiatry*, *11*(1), 99–113. [JTP8]

Costa, P. T., McCrea, R. R., & Psychological Assessment Resources Inc. (1992). *Revised NEO Personality Inventory (NEO PI-R) and NEO Five-Factor Inventory (NEO-FFI)*. Odessa, Fla. (P.O. Box 998, Odessa 33556): Psychological Assessment Resources.

Costello, E. J., Pine, D. S., Hammen, C., March, J. S., Plotsky, P. M., Weissman, M. M., et al. (2002). Development and natural history of mood disorders. *Biol Psychiatry, 52*(6), 529–542. [JTP9]

Covington, M. A., He, C., Brown, C., Naci, L., McClain, J. T., Fjordbak, B. S., et al. (2005). Schizophrenia and the structure of language: the linguist's view. *Schizophr Res, 77*(1), 85–98. [JTP10]

Crow, J. F. (2000). The origins, patterns and implications of human spontaneous mutation. *Nature Reviews Genetics, 1*(1), 40–47.

Crow, J. F., & Kimura, M. (1979). Efficiency of Truncation Selection. *Proceedings of the National Academy of Sciences of the United States of America, 76*(1), 396–399.

Crow, T. J. (1995). A Darwinian approach to the origins of psychosis. *British Journal of Psychiatry, 167*(1), 12–25.

Darwin, C. (1859). *On the origin of species by means of natural selection*. London: J. Murray.

Darwin, C. (1871). *The descent of man, and selection in relation to sex*. London: J. Murray.

Dickerson, F. B., Sommerville, J., Origoni, A. E., Ringel, N. B., & Parente, F. (2002). Experiences of stigma among outpatients with schizophrenia. *Schizophr Bull, 28*(1), 143–155. [JTP11]

Eaton, W. W., Romanoski, A., Anthony, J. C., & Nestadt, G. (1991). Screening for psychosis in the general population with a self-report interview. *J Nerv Ment Dis, 179*(11), 689–693. [JTP12]

Etcoff, N. L. (1999). *Survival of the prettiest: The science of beauty*. New York: Doubleday.

Eyre-Walker, A., & Keightley, P. D. (1999). High genomic deleterious mutation rates in hominids. *Nature, 397*(6717), 344–347.

Falconer, D. S. (1996). *Introduction to quantitative genetics* (4th edition). New York: Longman/Wiley.

Fay, J. C., Wyckoff, G. J., & Wu, C. I. (2001). Positive and negative selection on the human genome. *Genetics, 158*(3), 1227–1234.

Fisher, R. A. (1915). The evolution of sexual preference. *Eugenics Review, 7*, 184–192.

Fisher, R. A. (1930). *The genetical theory of natural selection*. Oxford: Clarendon Press.

Fiske, P., Rintamaki Pekka, T., & Karvonen, E. (1998). Mating success in lekking males: A meta-analysis. *Behavioral Ecology, 9*(4), 328–338.

[JTP13] Forstmeier, W., Kempenaers, B., Meyer, A., & Leisler, B. (2002). A novel song parameter correlates with extra-pair paternity and reflects male longevity. *Proc R Soc Lond B Biol Sci, 269*(1499), 1479–1485.

[JTP14] Gangestad, S. W., Thornhill, R., & Garver, C. E. (2002). Changes in women's sexual interests and their partners' mate-retention tactics across the menstrual cycle: evidence for shifting conflicts of interest. *Proc R Soc Lond B Biol Sci, 269*(1494), 975–982.

Garamszegi, L. Z., Moller, A. P., & Erritzoe, J. (2003). The evolution of immune defense and song complexity in birds. *Evolution, [print] 57*(4), 905–912.

Hafner, H., Maurer, K., Loffler, W., & Riecher-Rossler, A. (1993). The influence of age and sex on the onset and early course of schizophrenia. *British Journal of Psychiatry, 162*, 80–86.

[JTP15] Hasson, O. (1989). Amplifiers and the handicap principle in sexual selection: A different emphasis. *Proc R Soc Lond B Biol Sci, 235*(1281), 383–406.

Haukka, J., Suvisaari, J., & Lonnqvist, J. (2003). Fertility of patients with schizophrenia, their siblings, and the general population: a cohort study from 1950 to 1959 in Finland. *American Journal of Psychiatry, 160*(3), 460–463.

[JTP16] Haverkamp, F., Propping, P., & Hilger, T. (1982). Is there an increase of reproductive rates in schizophrenics? I. Critical review of the literature. *Archiv fuer Psychiatrie und Nervenkrankheiten, 232*(5), 439–450.

[JTP17] Hoikkala, A., Aspi, J., & Suvanto, L. (1998). Male courtship song frequency as an indicator of male genetic quality in an insect species, Drosophila montana. *Proc R Soc Lond B Biol Sci, 265*(1395), 503–508.

Holy, T. E., & Guo, Z. (2005). Ultrasonic Songs of Male Mice. *PLoS Biology, 3*(12).

[JTP18] Hooper, P., & Miller, G. F. (Submitted). Mutual mate choice can drive ornament evolution even under perfect monogamy. *Proc R Soc Lond B Biol Sci*.

Houle, D. (1992). Comparing evolvability and variability of quantitative traits. *Genetics, 130*(1), 195–204.

Houle, D. (1998). How should we explain variation in the genetic variance of traits? *Genetica (Dordrecht), 102–103*(0), 241–253.

[JTP19] Houle, D., & Kondrashov, A. S. (2002). Coevolution of costly mate choice and condition-dependent display of good genes. *Proc R Soc Lond B Biol Sci, 269*(1486), 97–104.

Huxley, J., Mayr, E., Osmond, H., & Hoffer, A. (1964). Schizophrenia as a genetic morphism. *Nature, 204*, 220–221.

[JTP20] Jablensky, A. (2000). Epidemiology of schizophrenia: the global burden of disease and disability. *Eur Arch Psychiatry Clin Neurosci, 250*(6), 274–285.

Jones, I. L., & Hunter, F. M. (1993). Mutual sexual selection in a monogamous seabird. *Nature, 362*(6417), 238–239.

Kahn, R. S. a. D., K.L. (1995). New Developments in Dopamine and Schizophrenia. In F. E. B. a. D. J. Kupfer (Ed.), *Psychopharmacology: The fourth generation of progress* (pp. 1193–1203.). New York: Raven Press, Ltd.

Kanazawa, S., & Kovar, J. L. (2004). Why beautiful people are more intelligent. *Intelligence, 32*(3), 227–243.

Keller, M., & Miller, G. F. (in press). Which evolutionary genetic models best explain the persistence of common, harmful, heritable mental disorders? *Behavioral and Brain Sciences*.

Kendler, K. S., McGuire, M., Gruenberg, A. M., O'Hare, A., Spellman, M., & Walsh, D. (1993). The Roscommon family study. III. Schizophrenia-related personality disorders in relatives. *Arch Gen Psychiatry, 50*(10), 781–788. [JTP21]

Kennedy, J. L., Farrer, L. A., Andreasen, N. C., Mayeux, R., & St. George-Hyslop, P. (2003). The genetics of adult-onset neuropsychiatric disease: complexities and conundra? *Science, 302*(5646), 822–826.

Kieseppa, T., Partonen, T., Haukka, J., Kaprio, J., & Lonnqvist, J. (2004). High concordance of bipolar I disorder in a nationwide sample of twins. *Am J Psychiatry, 161*(10), 1814–1821.

Kirkpatrick, M., & Ryan, M. J. (1991). The Evolution of Mating Preferences and the Paradox of the Lek. *Nature, 350*(6313), 33–38.

Koenigsberg, H. W., Reynolds, D., Goodman, M., New, A. S., Mitropoulou, V., Trestman, R. L., et al. (2003). Risperidone in the treatment of schizotypal personality disorder. *J Clin Psychiatry, 64*(6), 628–634.

Kokko, H., Brooks, R., Jennions Michael, D., & Morley, J. (2003). The evolution of mate choice and mating biases. *Proc R Soc Lond B Biol Sci, [print] 270*(1515), 653–664.

Kotiaho, J. S., Simmons, L. W., & Tomkins, J. L. (2001). Towards a resolution of the lek paradox. *Nature, 410*(6829), 684–686.

Lyon, B. E., Eadie, J. M., & Hamilton, L. D. (1994). Parental choice selects for ornamental plumage in American coot chicks. *Nature, 371*(6494), 240–243.

Marcelis, M., Navarro-Mateu, F., Murray, R., Selten, J. P., & van Os, J. (1998). Urbanization and psychosis: A study of 1942–1978 birth cohort in the Netherlands. *Psychological Medicine, 28*(4), 871–879.

McDonald, C., & Murphy, K. C. (2003). The new genetics of schizophrenia. *Psychiatr Clin North Am, 26*(1), 41–63. [JTP22]

Melis, M. R., & Argiolas, A. (1995). Dopamine and sexual behavior. *Neurosci Biobehav Rev, 19*(1), 19–38. [JTP23]

Michod, R. E., & Hasson, O. (1990). On the evolution of reliable indicators of fitness. *American Naturalist, 135*(6), 788–808.

Miller, G. F. (2000). *The mating mind: How sexual choice shaped the evolution of human nature* (1st ed.). New York: Doubleday.

Miller, G. F., & Penke, L. (in press). The evolution of human intelligence and the coefficient of additive genetic variance in human brain size. *Intelligence*.

Mock, D. W., & Parker, G. A. (1997). *The evolution of sibling rivalry.* New York: Oxford University Press.

Nanko, S., & Moridaira, J. (1993). Reproductive rates in schizophrenic outpatients. *Acta Psychiatr Scand, 87*(6), 400–404. [JTP24]

Nayani, T. H., & David, A. S. (1996). The auditory hallucination: a phenomenological survey. *Psychol Med, 26*(1), 177–189. [JTP25]

Nettle, D., & Clegg, H. (2006). Schizotypy, creativity and mating success in humans. *Proc R Soc Lond B Biol Sci*, 273(1586): 611–615. [JTP26]

Nowicki, S., Hasselquist, D., Bensch, S., & Peters, S. (2000). Nestling growth and song repertoire size in great reed warblers: Evidence for song learning as an indicator mechanism in mate choice. *Proc R Soc Lond B Biol Sci, 267*(1460), 2419–2424. [JTP27]

[JTP28] Osby, U., Correia, N., Brandt, L., Ekbom, A., & Sparen, P. (2000). Mortality and causes of death in schizophrenia in Stockholm county, Sweden. *Schizophr Res, 45*(1–2), 21–28.

Paillette, M., Bizat, N., & Joly, D. (1997). Differentiation of dialects and courtship strategies in allopatric populations of Drosophila teissieri. *Journal of Insect Physiology, 43*(9), 809–814.

[JTP29] Parnas, J., Cannon, T. D., Jacobsen, B., Schulsinger, H., Schulsinger, F., & Mednick, S. A. (1993). Lifetime DSM-III-R diagnostic outcomes in the offspring of schizophrenic mothers. Results from the Copenhagen High-Risk Study. *Arch Gen Psychiatry, 50*(9), 707–714.

Penton-Voak, I. S., Perrett, D. I., Castles, D. L., Kobayashi, T., Burt, D. M., Murray, L. K., et al. (1999). Menstrual cycle alters face preference. *Nature, 399*, 741–742.

Perrett, D. I., May, K. A., & Yoshikawa, S. (1994). Facial shape and judgments of female attractiveness. *Nature, 368*(6468), 239–242.

Petrie, M. (1994). Improved growth and survival of offspring of peacocks with more elaborative trains. *Nature, 371*(6498), 598–599.

Petrie, M., Halliday, T., & Sanders, C. (1991). Peahens Prefer Peacocks with Elaborate Trains. *Animal Behaviour, 41*(2), 323–332.

[JTP30] Pomiankowski, A., & Moller, A. P. (1995). A resolution of the lek paradox. *Proc R Soc Lond B Biol Sci, 260*(1357), 21–29.

[JTP31] Pritchard, J. K. (2001). Are rare variants responsible for susceptibility to complex diseases? *Am J Hum Genet, 69*(1), 124–137.

Prokosch, M. D., Yeo, R. A., & Miller, G. F. (2005). Intelligence tests with higher g-loadings show higher correlations with body symmetry: Evidence for a general fitness factor mediated by developmental stability. *Intelligence, 33*(2), 203–213.

Proulx, S. R. (2001). Female choice via indicator traits easily evolves in the face of recombination and migration. *Evolution, 55*(12), 2401–2411.

[JTP32] Randall, P. L. (1998). Schizophrenia as a consequence of brain evolution. *Schizophr Res, 30*(2), 143–148.

Reinhold, K. (2004). Modeling a version of the good-genes hypothesis: female choice of locally adapted males. *Organisms Diversity & Evolution, 4*(3), 157–163.

Riley, B. P., & McGuffin, P. (2000). Linkage and associated studies of schizophrenia. *American Journal of Medical Genetics, 97*(1), 23–44

[JTP33] Rowe, L., & Houle, D. (1996). The lek paradox and the capture of genetic variance by condition dependent traits. *Proc R Soc Lond B Biol Sci, 263*(1375), 1415–1421.

[JTP34] Saino, N., Ninni, P., Calza, S., Martinelli, R., De Bernardi, F., & Moller, A. P. (2000). Better red than dead: Carotenoid-based mouth coloration reveals infection in barn swallow nestlings. *Proc R Soc Lond B Biol Sci, 267*(1438), 57–61.

Schmitt, D. P., Alcalay, L., Allensworth, M., Allik, J., Ault, L., Austers, I., et al. (2004). Patterns and Universals of Adult Romantic Attachment Across 62 Cultural Regions: Are Models of Self and of Other Pancultural Constructs? *Journal of Cross-Cultural Psychology, 35*(4), 367–402.

[JTP35] Shaner, A., Miller, G., & Mintz, J. (2004). Schizophrenia as one extreme of a sexually selected fitness indicator. *Schizophr Res, 70*(1), 101–109.

St. Clair, D., Xu, M., Wang, P., Yu, Y., Fang, Y., Zhang, F., et al. (2005). Rates of adult schizophrenia following prenatal exposure to the Chinese famine of 1959–1961. *Jama, 294*(5), 557–562.

Strauss, J. S. (1969). Hallucinations and delusions as points on continua function: rating scale evidence. *Arch Gen Psychiatry, 21*(5), 581–586. [JTP36]

Sunyaev, S., Ramensky, V., Koch, I., Lathe, W., III, Kondrashov, A. S., & Bork, P. (2001). Prediction of deleterious human alleles. *Hum Mol Genet, 10*(6), 591–597. [JTP37]

Takahashi, T., Suzuki, M., Zhou, S. Y., Hagino, H., Kawasaki, Y., Yamashita, I., et al. (2004). Lack of normal gender differences of the perigenual cingulate gyrus in schizophrenia spectrum disorders. A magnetic resonance imaging study. *Eur Arch Psychiatry Clin Neurosci, 254*(5), 273–280. [JTP38]

Tsuang, M. T., Stone, W. S., & Faraone, S. V. (2001). Genes, environment and schizophrenia. *Br J Psychiatry, 178*(Suppl 40), S18–24. [JTP39]

van Os, J., Hanssen, M., Bijl, R. V., & Ravelli, A. (2000). Strauss (1969) revisited: a psychosis continuum in the general population? *Schizophr Res, 45*(1–2), 11–20. [JTP40]

Verdoux, H., van Os, J., Maurice-Tison, S., Gay, B., Salamon, R., & Bourgeois, M. (1998). Is early adulthood a critical developmental stage for psychosis proneness? A survey of delusional ideation in normal subjects. *Schizophr Res, 29*(3), 247–254. [JTP41]

Wilson, D. R. (1997). Evolutionary epidemiology: Darwinian theory in the service of medicine and psychiatry. In S. Baron-Cohen (Ed.), *The maladapted mind: classic readings in evolutionary psychopathology* (pp. 39–55). Hove, East Sussex, UK: Psychology Press.

Wood, D. E. (1995). Neuromodulation of rhythmic motor patterns in the blue crab Callinectes sapidus by amines and the peptide proctolin. *Journal of Comparative Physiology A Sensory Neural and Behavioral Physiology, 177*(3), 335–349.

Woods, B. T. (1998). Is schizophrenia a progressive neurodevelopmental disorder? Toward a unitary pathogenetic mechanism. *American Journal of Psychiatry, 155*(12), 1661–1670.

Yeo, R. A., Gangestad, S. W., Edgar, C., & Thoma, R. (1999). The evolutionary genetic underpinnings of schizophrenia: the developmental instability model. *Schizophr Res, 39*(3), 197–206.

Zahavi, A. (1975). Mate selection a selection for a handicap. *Journal of Theoretical Biology, 53*(1), 205–214.

[JTP42]

PAGE WILL BE BLANK WHEN QUERIES ARE DELETED

Comments

[JPT1] Page numbers
[JPT2] Full title?
[JPT3] Full title
[JPT4] Full title
[JPT5] Full title
[JPT6] Full title
[JPT7] Full title
[JPT8] Full title
[JPT9] Full title
[JPT10] Full title
[JPT11] Full title
[JPT12] Full title
[JPT13] Full title
[JPT14] Full title
[JPT15] Full title
[JPT16] Full title?
[JPT17] Full title
[JPT18] Full title
[JPT19] Full title
[JPT20] Full title
[JPT21] Full title
[JPT22] Full title
[JPT23] Full title
[JPT24] Full title
[JPT25] Full title
[JPT26] Full title
[JPT27] Full title
[JPT28] Full title
[JPT29] Full title
[JPT30] Full title
[JPT31] Full title
[JPT32] Full title
[JPT33] Full title
[JPT34] Full title
[JPT35] Full title
[JPT36] Full title
[JPT37] Full title
[JPT38] Full title
[JPT39] Full title
[JPT40] Full title
[JPT41] Full title
[JPT42] Full title