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Autism as the Low-Fitness Extreme of a Parentally Selected Fitness Indicator

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Abstract Siblings compete for parental care and feeding, while parents must allocate scarce resources to those offspring most likely to survive and reproduce. This could cause offspring to evolve traits that advertise health, and thereby attract parental resources. For example, experimental evidence suggests that bright orange filaments covering the heads of North American coot chicks may have evolved for this fitness-advertising purpose. Could any human mental disorders be the equivalent of dull filaments in coot chicks—low-fitness extremes of mental abilities that evolved as fitness indicators? One possibility is autism. Suppose that the ability of very young children to charm their parents evolved as a parentally selected fitness indicator. Young children would vary greatly in their ability to charm parents, that variation would correlate with underlying fitness, and autism could be the low-fitness extreme of this variation. This view explains many seemingly disparate facts about autism and leads to some surprising and testable predictions.

Keywords Autism · Sexual selection · Parental selection · Sibling rivalry · Fitness indicator

Autism is a severe developmental disorder that begins before age three and produces life-long disability. It markedly impairs social behavior and communication, and it

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produces restricted and repetitive patterns of interests and behavior (American Psychiatric Association 2000). It strikes 10 to 20 individuals per 10,000 (Chakrabarti and Fombonne 2001) and is highly heritable (Veenstra-Vanderweele et al. 2004), yet its cause is unknown and treatment is unsatisfactory (Francis 2005).

Many puzzling questions remain unanswered. If autism is heritable, why has it proven so hard to identify specific susceptibility genes? Why is autism more common in boys but more severe in girls? Why is it associated with environmental hazards, developmental abnormalities, and increased mortality? Why does it always start so early in life? This last, seemingly simple question may supply a crucial clue. Recent evidence suggests that the brain systems that go awry in autism are particularly important for a child's social interaction with its mother (Maestro et al. 2002; Zwaigenbaum et al. 2005).

In other species, many traits used by offspring to interact with parents evolved to compete with siblings for parental care and feeding (Mock and Parker 1997). Some of these traits may have evolved as "fitness indicators" that allow parents to allocate scarce resources according to a reliable estimate of each offspring's chances for long-term reproductive success. Did some aspects of the social and communicative repertoire of human offspring also evolve as fitness indicators? Could autism represent their low-fitness extreme? If so, then fitness indicator theory may help explain autism's puzzles. This paper explores that proposition.

Fitness Indicators and Mental Disorders

Fitness indicator theory (Tomkins et al. 2004) was originally developed to explain the evolution of sexually selected ornaments such as the peacock's tail. According to the "the handicap principle" (Zahavi 1975) and several related theories, a peacock's tail takes considerable energy to grow, maintain, and display. This cost makes it a reliable indicator of fitness because only peacocks in very good condition can afford the energy necessary to grow large and colorful tails. As a result, many of the same fitness-reducing mutations (i.e., "bad genes") that reduce general fitness, for example by impairing immunity or embryonic cell migration, also interfere with tail growth, maintenance, and display. Peahens evolved a preference for larger tails because it paid to mate with the fittest peacocks. The preference remains so strong that among a typical group of about 30 peacocks, the one or two with the largest tails father virtually all offspring in the next generation. This strong directional selection favored genes that could produce large tails, even if they could do so only among the few peacocks with very good genes overall. Despite strong directional selection, small tails reappear in every generation because DNA copying errors during the production of sperm and ova introduce new bad genes in every generation.

We have proposed that the power of sexual selection to maintain seemingly maladaptive variation, such as tails too small to attract mates, may explain the persistence of some common mental disorders (Shaner et al. 2007). Schizophrenia, for example, may be evolutionarily analogous to a small peacock's tail, or more technically, it may be the low-fitness extreme of a sexually selected fitness indicator that evolved in humans through mutual mate choice (Shaner et al. 2004). If correct, this view would explain schizophrenia's typical adolescent and early adult onset;

why it markedly reduces rates of marriage and reproduction; why it persists as a heritable trait despite its reproductive disadvantages; why it affects males earlier and more severely; why it is so commonly associated with neurodevelopmental abnormalities, fetal hypoxia, viral infection, and famine; why dopamine antagonists are therapeutic; and even why affected individuals are so socially stigmatized. The hypothesis also predicts that (1) specific genetic and environmental causes will number in the hundreds or thousands, each accounting for no more than a few percent of cases; (2) the responsible genes will be a wide variety of fitness-reducing mutations that are lineage-specific and that will not replicate well across populations; and (3) drugs that reduce courtship behaviors in other animals will improve schizophrenia in humans.

Parentally Selected Fitness Indicators

Sexual selection is not the only form of directional selection that could produce fitness indicators. In many species, siblings compete for scarce parental resources. At the same time, parents 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 (Mock and Parker 1997). For example, shield bug mothers cover their eggs with their bodies, placing the larger, more viable eggs in the center of the clutch where they are less vulnerable to predation. White stork parents with large clutches kill their smallest, slowest growing chicks. Barnyard piglets are born with special teeth angled outward with which they slash their siblings during contests over access to the most productive, anterior teats. Pronghorn antelope embryos with the better uterine implantation sites grow extensions of their amniotic sacs which pierce and kill their unborn siblings.

The striking overall pattern is that parents of many species create more offspring than they typically support. Evolutionary biologists have suggested four possible fitness benefits. Parents may create (1) an "optimistic" family size that can take advantage of unusually good environmental conditions, (2) extra offspring as "insurance" against loss of offspring to predators or accidents, (3) extra offspring to help the others survive (e.g., by serving as food for their siblings), and (4) extra offspring to allow parents to preferentially invest in the most promising (i.e., "progeny choice").

In "progeny choice," parents place an over-supply of offspring into an arena (e.g., a nest, den, or womb), determine which offspring have the best fitness prospects, and then kill the others through active aggression, passive neglect, or tolerance of fatal sibling competition. Whether parents choose actively or passively, the result is the same—directional selection on any trait that wins parental resources.

In response to this directional selection, the offspring of many species evolved traits that advertise physical and behavioral fitness and thereby attract parental care and feeding. For example, healthy barn swallow nestlings beg for food with wide-open mouths, or gapes, that are colored bright red. The color fades to dull yellow when nestlings are infected because they must divert the crucial carotenoid pigment molecules to serve a role in immune system function rather than gape color. Parents preferentially feed nestlings with bright red gapes. Thus, gape color may serve as a fitness indicator (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 et al. 1994). Evidence that such indicators have evolved in many other species comes from the observation that offspring solicitation of parental provisioning (e.g., "peeping" of chicks, begging calls of nestlings, ultrasonic vocalizations of rodent pups, and bleating of lambs) and the related response by parents (e.g., feeding) are both highly variable across individuals and highly heritable (around 50%) (Kolliker and Richner 2001).

Even the crying of human infants may have evolved, at least in part, as a fitness indicator (Furlow 1997; Soltis 2004). Infant cry pitch varies greatly, and higher pitch is associated with premature birth, smaller infant size, serious health problems, and more negative reactions by parents. Similarly, infant babbling may have evolved as a fitness indicator (Locke 2006) since mothers value babbling and infants who do not babble have an unusually high number of physical, sensory, cognitive, and linguistic disorders. Also, in a study of parental protection, mothers were more likely to buckle visually attractive children into grocery carts and more likely to keep attractive toddlers from wandering out of sight. This suggests the possibility that attractiveness in children may have evolved in part to attract parental protection (Harrell 2005).

Human offspring may have evolved other fitness indicators as well. This is because, like modern parents (Hrdy 1999), ancestral parents could have assessed offspring fitness and allocated resources accordingly. The finding that parents from diverse populations are more likely to neglect, abuse, and kill low-fitness infants (Soltis 2004) is supporting evidence. This behavior likely represents an extreme of continuous variation in parental investment from infanticide to life-long devotion. For example, parents also invest more in high-fitness offspring, as evidenced by longer intervals before the conception of another child (Mace and Sear 1997). Such discriminative parental solicitude (Daly and Wilson 1995) would have placed strong directional selection on fitness cues (i.e., traits which happen to provide parents with an estimate of offspring fitness; Bradbury and Vehrencamp 1998). In turn, directional selection could have converted fitness cues into fitness signals (i.e., traits that evolved for signaling and are costly to produce; Hasson 1997). Thus, discriminative parental solicitude can produce fitness indicators without causing the premature death of low-fitness offspring. Even if all offspring survive and reproduce, those displaying higher-quality fitness cues (and signals) will obtain more parental resources, grow faster and larger, and ultimately attract more or better mates.

Crucial for our hypothesis is that variation in any parentally selected fitness indicator must include an extreme that does not attract parental resources—indeed, that may be extremely aversive and distressing to parents. Also, this extreme should be heritable and harmful to survival and reproductive prospects, yet not eliminated by selection. Could any childhood-onset mental disorders represent the equivalent of dull gapes in barn swallow nestlings or dull filaments in coot chicks? One possibility is autism.

Autism as the Low-fitness Extreme of a Parentally Selected Fitness Indicator

Suppose that the ability of human offspring to charm their parents—perhaps through language, facial expression, creative play, and coordinated social interaction—

evolved as a parentally selected fitness indicator (Miller 2000). More articulate, expressive, playful, and socially engaged offspring would give a reliable warranty of their genetic and phenotypic quality and thus would solicit higher parental investment. Offspring 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 frustration and alienation experienced by parents of autistic children. Such a view leads to explanations and predictions much like those regarding schizophrenia as a low-fitness extreme of a sexually selected fitness indicator because the evolutionary mechanism of fitness-signaling is similar, except that parents, not mates, make the selection in this case.

The three essential elements of our hypothesis are: (1) Every human offspring executes genetic instructions to grow brain systems for behaviors that solicit resources from parents—a set of behaviors we will call "charm." (2) These brain systems are so complex or demand so much energy that their development is especially sensitive to genetic quality and environmental hazards. As a result, charm varies greatly across individuals and correlates with underlying fitness. At one extreme, offspring with high fitness (i.e., a low mutation load) and a favorable environment develop these brain systems well and attract a larger share of parental resources. Most offspring carry some bad genes that mildly disrupt the development of these brain systems, resulting in suboptimal charm and a smaller share of parental resources. (3) At the other extreme, a few individuals (about 10 or 20 per 10,000) with low fitness (i.e., a high mutation load), or exposed to severe environmental hazards, develop profound defects in the brain systems required for charming behaviors. It is this extreme that we recognize as autism.

Note that we use the word "charm" only in the sense that it attracts parental attention, protection, provisioning, or other resources. The behaviors that constitute charm need not be "pleasing" and could be deceptive, manipulative, or coercive. Moreover, the parental preference for the high-fitness extreme need not be conscious to the individual or morally acceptable to the society. In these regards, charm as a parentally selected fitness indicator in infants (which goes awry to produce autism) may have a lot of overlap with creative verbal intelligence as a sexually selected indicator in adults (which may go awry to produce schizophrenia) (Miller and Tal 2007; Shaner et al. 2004, 2007).

Also note that whatever trait constitutes charm, it is likely only one among many other traits that serve as fitness cues or, in some cases, signals. These other traits could be termed "charming" as well. However, here we use "charm" in a very narrow and hypothetical sense—a set of behaviors that evolved as a parentally selected fitness indicator and whose low-fitness extreme is autism.

Charm May Attract Breast-Feeding and Thereby Lengthen Birth Intervals

As we will show, our general hypothesis has considerable explanatory and predictive power even without specifying three additional elements: (1) the particular behaviors that constitute charm, (2) the specific parental resources they attract, and (3) the specific mechanism of competition with siblings over these resources. For each of these unspecified elements, there are at least several potentially overlapping

candidates. However, nearly all cases of autism begin before age three (Short and Schopler 1988), suggesting that the brain systems that go awry in autism evolved to attract resources that are critically important before age three. One such resource is breast milk. It is essential for initial growth and survival, very important for immunity (Oddy 2001), and rarely supplied beyond age three. Moreover, it suggests a special mechanism for competition with siblings.

Humans typically bear young singly, so babies rarely compete with littermates for milk, as do most other mammalian offspring. However, breast-feeding allows babies to compete with future, as-yet-unborn siblings—not over the current milk allocation, but over the interval until the next offspring arrives (Badcock 1989). This is because frequent breast-feeding acts as a powerful contraceptive (Thapa et al. 1988; World Health Organization 1998), and the longer the current baby nurses intensively, the longer it can postpone the arrival of a competitor. Such rivalry is analogous to competition over the most productive teats in pigs or over the best uterine implantation site in antelopes. The difference is that birth interval concerns the duration rather than the rate of maternal investment. So, charm could have evolved to convince mothers to continue breast-feeding, not just because milk provides nutrition and immunity, but because continued breast-feeding secures a greater share of a mother's lifetime reproductive effort.

However, human offspring have an evolutionary interest in the *eventual* arrival of a sibling because they will share many of the same segregating genes—50% if the father is the same; 25% if different. So, at some point, the current baby should reduce its demand for breast-milk enough to allow its mother to conceive a sibling (Trivers 1974). Each baby should graciously wean itself at the point when allowing the mother to invest in a younger sibling maximizes the baby's own "inclusive fitness" (Hamilton 1964)—the overall reproductive success of the baby's genes, whether those genes lie in the current baby's body or in the bodies of siblings (and other close relatives).

From the perspective of the mother's genes, though, it is better to conceive another child even sooner. A mother's reproductive success depends on the survival and reproduction of all her offspring. Because each is equally related to her, they are equally important to her reproductive success. So, mothers should have evolved to divide resources equally among offspring (Trivers 1974).

However, the current baby is 100% related to itself and only 25% to 50% related to its siblings, so its own reproductive success is much more important to its inclusive fitness than is the reproductive success of any sibling. Thus, offspring should have evolved to extract considerably more maternal resources than go to any sibling—and more than the mother would prefer to give them. This difference over the genetic value of siblings, and therefore over how to allocate maternal resources, should produce a period of "weaning conflict" between mother and child that begins when weaning is optimum for the mother's reproductive success and ends when weaning is optimum for the child's inclusive fitness (Trivers 1974).

Some Infant Social Behaviors Could Have Evolved as Fitness Indicators

How is this weaning conflict resolved? Among the possibilities is that mothers accede to a baby's demands for a larger share of resources only when

disproportionate investment would benefit the mother's reproductive success (Godfray 1995). Offspring vary in genetic quality and therefore in their potential for survival and reproduction. This could lead mothers to assess offspring fitness and allocate resources accordingly. If ancestral human parents delivered more resources to babies showing indications of superior fitness, this could have led babies to evolve traits that signal fitness. They could thereby influence how long a mother continues to breastfeed intensively enough to prevent ovulation (through lactational amenorrhea), thus delaying the appearance of a sibling rival.

For example, suppose that in our hominid ancestors, the brain systems that allow babies to respond socially to their mothers (e.g., through eye contact and social smiling) were already somewhat sensitive to fitness, perhaps because those brain systems were already sufficiently complex or energy-demanding that their development could be disrupted by genetic mutations or environmental hazards. If so, then mothers who preferentially nursed their more socially responsive babies would have allocated more resources, both milk and birth interval, to those most likely to survive and reproduce. This would have favored genes that acted in mothers to increase the preference for socially responsive babies.

In turn, maternal preferences for more socially responsive babies would have favored genes that acted in babies to increase the fitness sensitivity of their brain systems for charm. For example, consider a gene that called for earlier or more extensive development of the brain systems needed for social responsiveness—development that thereby became even more sensitive to fitness. Figure 1 illustrates the effect of such a gene (labeled "enhanced fitness sensitivity"). Babies at the high-fitness extreme would have developed the brain systems correctly, produced even more charming social behaviors, obtained more parental resources, and ultimately produced more offspring themselves.

In this scenario, individuals possessing all three kinds of genes—"good genes" for general fitness, genes that increased charm's fitness sensitivity, and genes that increased the maternal preference for charm—would have the highest reproductive success. Across generations, the increasing correlation within individuals of these three kinds of genes could have caused the rapid evolution of increasingly complex, energy-demanding, and therefore fitness-sensitive brain systems for infant social interactions with mothers. The process is analogous to that proposed for the evolution of sexually selected fitness indicators (Hasson 1989; Pomiankowski and Moller 1995; Rowe and Houle 1996).

It is even possible that some of the genes that increased the fitness sensitivity of charm in our hominid ancestors were (and still are) expressed in mothers, rather than in infants. Such genes could indirectly increase the fitness sensitivity of charm in infants by causing mothers to attempt to wean earlier. This would force infants to attempt earlier, more fitness-sensitive development of the brain systems for charm. High-fitness offspring would develop those systems well and convince mothers to continue breast-feeding for many months, while low-fitness infants would not. Mothers with such a gene, compared with those who lack it, would invest shorter birth intervals in low-fitness infants, but not necessarily longer intervals in high-fitness infants.

If this scenario is accurate and unique to humans, then we should find that greater variance in birth interval distinguishes humans from our closest hominoid relatives.

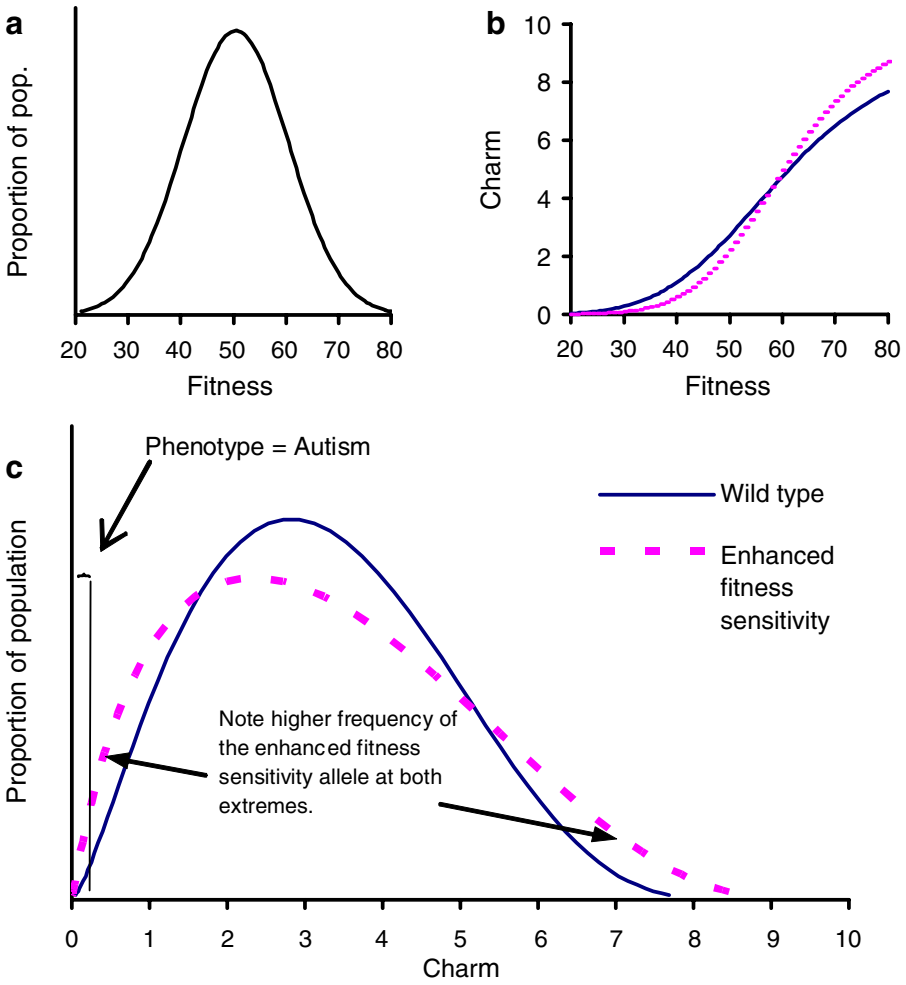

Fig. 1 Hypothetical relationships among fitness, the attractiveness of an indicator trait, and the prevalence of autism. **a** Fitness (i.e., genetic quality) in the general population as a simple normal distribution (Gaussian) displayed as *T*-scores with mean set at 50 and SD=10. **b** Charm (on an arbitrary scale from 0 to 10) expressed as two similar sigmoidal functions of fitness. **c** Charm 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 have 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 charming and uncharming extremes. We chose a threshold that defines an uncharming extreme (which we hypothesize is identical to autism) containing 0.2% of the total population. In this illustration, that 0.2% comprises about one quarter with the wild type indicator and three quarters with the enhanced sensitivity indicator

Indeed, this is exactly what Lee (1996) noted—but did not explain—in a comparison of weaning and birth intervals across a range of primate species. Compared with several other ape species, humans displayed a greater range of birth intervals due mostly to a shorter minimum birth interval. That is, humans weaned at least some infants earlier than did any other ape species.

Charm Could Involve Brain Systems that Detect and Respond to Weaning Attempts

To prevent ovulation, a baby must thwart maternal attempts to introduce other foods, reduce nighttime feedings, or reduce the frequency or total daily duration of suckling—each of which increases the odds of ovulation (Stern et al. 1986; Taylor et al. 1999; World Health Organization 1998). Charm might enable a baby to detect initial weaning attempts (e.g., slower responses to feeding requests and less maternal smiling) and respond with special efforts to convince its mother to remain close and responsive to breast-feeding requests. Such efforts might require learning which kind of cry brings mother more quickly; what emotional response to her arrival produces longer feedings; and what kind of suckling produces the greatest daily duration of nursing. Charm might allow the baby to modulate such efforts by paying close attention to its mother's gaze, facial expression, and tone of voice, and to repeat those efforts that appeared to please its mother.

Infant Social Behavior Should Correlate with Fitness

Social behaviors are good candidates for charm because (1) it is plausible that mothers would invest more in socially responsive babies, given the importance of social responsiveness and intelligence in our highly social species of group-living primate; (2) brain systems for social behavior are probably sufficiently complex or energy-demanding that their development is sensitive to fitness; (3) abnormalities in social interaction with mothers are among the earliest and most central signs of autism (Maestro et al. 2002; Zwaigenbaum et al. 2005); (4) social responsiveness may require understanding the minds of others, and autism impairs this ability (Baron-Cohen and Belmonte 2005); and (5) non-social skills (including so-called systematizing skills) are relatively spared in autism (Baron-Cohen 2002). If charm depends on social responsiveness, then the age distribution for the emergence of social responsiveness should resemble the distribution for birth interval, but shifted at least nine months earlier. Also, high-fitness (i.e., low-mutation-load) babies should perform better on measures of social responsiveness, be weaned later, attract a longer subsequent birth interval, be healthier, live longer, and leave more offspring. Consistent with this last prediction, several studies show that both longer duration of breast-feeding (Horwood et al. 2001; Vestergaard et al. 1999) and longer intervals before the next birth (Bella and Al-Almaie 2005) are associated with better offspring outcomes, including earlier attainment of developmental milestones and higher educational achievement.

Explanatory and Predictive Power of the Hypothesis

Our general hypothesis is that autism is the low-fitness extreme of a parentally selected fitness indicator. One possibility—a special case of our general claim—is that the indicator trait is social responsiveness, and that it evolved to attract breast-feeding and thereby compete with unborn siblings over birth interval. However, to test the general hypothesis, it is not necessary to specify the precise nature of the

indicator trait or the maternal resources it attracts because many of our explanations and predictions depend on nine generic properties of fitness indicators. The special case can be tested separately because it leads to additional predictions. Here we discuss the explanatory and predictive power of each of the nine properties with respect to the general hypothesis and to the special case.

1. Fitness Indicators Vary Continuously across Individuals

If autism is the low-fitness extreme of a fitness-indicator, then it is not a discrete condition. This may explain why the symptoms of autism lie on a spectrum with the traits of many individuals both with and without developmental disorders. Autistic Spectrum Disorders (ASD) include autism and two other disorders—Asperger's syndrome and nonspecific pervasive developmental disorder—in which symptoms are fewer or milder than those of autism. While one to two individuals per 1,000 has autism, another four to five have the milder forms of ASD (Chakrabarti and Fombonne 2001). Also, the two milder disorders are more common among the relatives of autistic individuals, suggesting a shared genetic etiology (Bailey et al. 1995). Moreover, autism is closely related to a measure of social responsiveness (the Social Responsiveness Scale) that varies continuously in the general population, is highly heritable, and underlies all three diagnostic criteria for autism: social deficits, language deficits, and repetitive or stereotypic behaviors (Constantino et al. 2006).

2. Offspring Display Indicators When Parents Choose or Siblings Compete

Thus, autism should appear when other children display their own higher-quality versions of charm in an effort to attract parental resources. Differences between autistic children and other children usually develop slowly, so "onset" is usually defined as the age at which parents, clinicians, or investigators can distinguish those with the disorder from those in the general population.

If the resource at stake is subsequent birth interval, infants should develop charm as mothers begin to wean and before they ovulate. Thus, the distribution of age at onset of autism will resemble that for age at weaning, and mean age at onset will not exceed mean duration of postpartum amenorrhea. Finally, differences across populations in the age at onset of autism should parallel differences in age at weaning and in the duration of postpartum amenorrhea.

Testing these predictions with existing data is problematic because (1) bottle-feeding and pacifiers might cause infants to act as if weaning has not occurred, (2) most studies define weaning as termination of breast-feeding rather than the decrease in breast-feeding that restores ovulation, (3) oral contraceptives disrupt ovulation and menses, and (4) no studies provide data on both mean age at onset of autism and mean age at weaning in the same population. However, even without data on autism itself, data on weaning, postpartum amenorrhea, or birth interval in natural-fertility populations that do not bottle-feed or use pacifiers would place plausibility limits on the mean age at onset of autism. Studies of such populations report weaning as early as 4 months and as late as 29 months (Konner 2005; Lummaa 2001; Quinlan et al. 2003; World Health Organization 1998). Moreover, breast-feeding between 6 and

18 months has the greatest effect on the duration of postpartum amenorrhea (Bongaarts 1983). Thus, some signs of autism should appear within the first 6 months after birth, with most cases beginning between 6 and 18 months, and nearly all cases by 29 months.

Consistent with this prediction, a retrospective study of home movies revealed that 6-month-olds who later developed autism had failed to develop the normal preference for social stimuli, such as faces (Maestro et al. 2002). Also, in a prospective study of at-risk infants, those who later developed autism showed marked passivity at 6 months, and by 12 months, performed poorly on measures of social interaction, including eye contact, visual tracking, orienting to name, imitation, social smiling, social interest, affect, and language development (Zwaigenbaum et al. 2005). With respect to the upper limit, parents of autistic children said they "first noticed something wrong" with their child by 24 months of age in 66% of cases and by 36 months in 94% of cases (Short and Schopler 1988).

This property of fitness indicators applies to anything that affects the timing of indicator display. For example, younger mothers should wean earlier because they have more remaining reproductive potential and can afford to be choosier about whether the current offspring warrants further resources (Trivers 1974). If so, then autism should begin earlier and be more common among those born to younger mothers. However, a countervailing effect is that birth complications—a known risk factor for autism—are more common in older mothers and would tend to increase the rate of autism among those born to older mothers. This may explain why a recent large-population-based study found that the odds of developing autism increased in both first-born children and children of older mothers (Glasson et al. 2004). In addition, the higher risk of both birth complications and autism among the offspring of older mothers may be due, at least in part, to the higher rate of new mutations in the sperm of older fathers, as we discuss under property 6 below.

Also, mothers might exercise choice by constantly modulating the extent to which they respond to infant demands. Scarce environmental resources, older dependent children, and apparently low-fitness infants should all cause mothers to begin weaning. Offspring should respond with earlier development of charm. But, earlier development would be more fitness-sensitive because rapid brain growth demands considerable energy (Aiello and Wheeler 1995) and introduces more sources of error. Greater fitness sensitivity would provide mothers with new estimates of offspring fitness, which would cause mothers to increase or decrease the pace of weaning. By increasing fitness sensitivity, earlier weaning should increase the probability of autism. Conversely, prolonging breast-feeding until it is refused by the child should decrease fitness sensitivity and reduce the probability of autism. However, we don't know which maternal behaviors signal imminent weaning, so simply ensuring breast-feeding might not suffice, whereas changing some other aspect of maternal behavior might be more effective. In some species, for example, mothers begin to wean by spending more time away from offspring (Devinney et al. 1998; Malm and Jensen 1997). If human maternal absences signal impending weaning, then avoiding such absences during the first 2 years might reduce the probability of autism. Both interventions could be studied among the younger siblings of autistic children.

3. Indicator Quality Affects Parental Resource Allocation

Autism should impair the infant's ability to attract maternal resources. If charm evolved to attract breast-feeding and delay the next pregnancy, then mothers will deliver less breast milk to autistic children, wean them earlier, and bear another child sooner. The effect would be more apparent when mothers themselves have limited resources (e.g., during a food shortage or when nursing an older child) and therefore must be even choosier about resource allocation. We know of only one study relevant to these predictions, and it showed that early weaning was more common among children with autism (Tanoue and Oda 1989).

Infant behaviors related to autism should also affect parental resource allocation. For example, poor social interaction at 12 months predicted which at-risk infants would develop autism (Zwaigenbaum et al. 2005), suggesting that social interaction ability is an important component of charm. If so, then the ability should vary greatly among infants, should correlate with underlying fitness, and should influence parental resource allocation. Moreover, if social interaction ability encourages mothers to nurse in the pattern that inhibits ovulation, then detailed recordings of suckling (Taylor et al. 1999) should show that high social interaction ability is associated with frequent suckling, greater total daily duration of suckling, and nighttime suckling—all of which inhibit ovulation.

Similarly, analysis of home movies has shown that failure to attend to social stimuli at age six months distinguished infants who later developed autism (Maestro et al. 2002), suggesting that a preference for social stimuli is an important part of charm. If so, then home movies of infants in the general population should show that preference for social stimuli varies greatly across individuals, correlates with measures of maternal resource allocation (e.g., age at weaning and birth interval), and correlates with measures of physical and mental health throughout life.

4. Indicators Show Predictable Sex Differences

Parental preferences should shape the evolution of fitness indicators differently in male and female offspring. This is because mothers should be choosier about allocating resources to sons. First, raising sons requires more maternal resources than raising daughters (Gibson and Mace 2003). For example, after raising a male calf, red deer mothers were less likely to survive the winter (Cockburn 1994), and Mongolian gerbil mothers rearing experimentally arranged all-male litters nursed more, were slower to copulate, and showed longer intervals between litters than mothers rearing all-female litters (Clark et al. 1990). Similarly, after bearing a son, human mothers wait longer to have another child (Mace and Sear 1997). Second, the reproductive success of sons varies more than that of daughters (Clutton-Brock et al. 1981). On the one hand, many low-quality males will never mate, so the expected net cost (in terms of reproductive success) of feeding the less-fit sons is higher than the cost of feeding the less-fit daughters—nearly all of whom will have some reproductive success. On the other hand, the fittest sons can have much higher reproductive success than the fittest females. So, mothers must be choosier about sons in both directions, investing most in the fittest sons, least in the least-fit sons, and somewhere in between for most daughters.

If human mothers follow this strategy, males should have evolved an even more fitness-sensitive version of charm. Male embryos should contain genetic instructions for still more complex or energy-demanding brain systems for charm, capable of producing more charming behavior among offspring with only a few bad genes (e.g., high fitness, low mutation load) even at the expense of less charming behavior, and more cases of autism, among offspring with many bad genes. This could explain why autism is four times as common in males (Skuse 2000).

Increased fitness sensitivity among males should result in earlier development of language and social skills in high-fitness males at the expense of later development in lower-fitness males. Increased fitness sensitivity is nevertheless favored if the benefit of delivering extra resources to high-fitness males outweighs the costs of limiting resource delivery to lower-fitness males. If the effect on low-fitness males is large, this would explain why, on average, boys develop language later than girls. For example, imagine that early development of social skills and language was a component of charm indicated by higher scores on the *x*-axis in panel c of Fig. 1. Higher fitness sensitivity in males would increase variance in the development of social skills (the dashed curve in panel c). The effect on mean age at attainment of such social skills would depend on the shape and position of the function in panel b.

Some evidence suggests that autism might be an extreme version of the normal male pattern in which systemizing ability exceeds empathizing ability (Baron-Cohen 2002). While autism impairs all cognitive domains, empathizing abilities are more impaired than are systematizing abilities. This is what our model would predict—the more a skill evolved to attract maternal resources, the more it will be impaired in autism. Also, on a few systematizing skills (e.g., the embedded figures task), children with Asperger's syndrome perform better than controls. A possible explanation is compensatory hypertrophy of relatively spared brain systems. That is, the brain may attempt to compensate for impaired empathizing skills by recruiting brain systems for systematizing skills, just as auditory systems hypertrophy in response to early blindness (Bavelier and Neville 2002)

5. Indicators Are Sensitive to Fitness and Condition

This property would explain several facts about autism. First, developmental abnormalities are common in autism because fitness indicators reveal fitness largely through disordered development. This may explain the increased rates in autism of minor physical anomalies (Manning et al. 2001; Rodier et al. 1997), as well as diverse abnormalities in brain development (Bauman and Kemper 2005).

Second, autism is polygenic because fitness indicators must be sensitive to mutations at many genetic loci that affect many different processes in different cell types, tissues, and organ systems (Rowe and Houle 1996). This may explain why a genome screen of a large sample of autistic sib pairs found no strong linkage results (Risch et al. 1999), and why genetic modeling of the recurrence risk in siblings suggested at least a hundred autism-susceptibility loci (Pritchard 2001).

Third, autism is associated with increased mortality (Shavelle and Strauss 1998) because of the same bad genes and environmental hazards that disrupted development of the indicator and produced autism. Moreover, excess mortality in autistic females is three times that in autistic males because the indicator trait is less

sensitive to fitness in girls. This not only reduces the rate of autism among girls, it ensures that when it does occur in girls, it is caused by worse genes and hazards, on average, and these produce higher mortality.

Fourth, autism is associated with environmental hazards, including intrauterine exposure to drugs (e.g., Stromland et al. 1994) and viruses (e.g., Ghaziuddin et al. 1992), because the environmental sensitivity of fitness indicators is what allows them to reveal bad genes. Among individuals with high genetic quality, fitness indicators develop well despite environmental hazards, but among those with low fitness, the combination of environmental hazards and bad genes interferes with indicator development. One manifestation of these hazards may be the high rate of obstetric complications among those who later develop autism (Glasson et al. 2004).

6. "Bad Genes" Cause Most of the Heritable Variation in the Attractiveness of Indicators

A balance between deleterious mutation and stabilizing selection produces an equilibrium frequency of fitness-reducing mutations in most animal populations. Thus, most heritable variation in general fitness may reflect individual differences in "mutation load" (i.e., the number of evolutionarily transient, fitness-reducing mutations; Houle and Kondrashov 2002; Michod and Hasson 1990; Rowe and Houle 1996), and embryos with the most "bad genes" are the most likely to develop poor quality indicators, including, in our hypothesis, autism. This allows us to predict that most of the genes associated with autism will carry diverse sets of low-frequency alleles (mutations) that mildly reduce general fitness, for example by impairing developmental stability (reviewed in Prokosch et al. 2005). Because they reduce fitness, they will not have spread widely across human populations but instead will be lineage-specific (Keller and Miller 2006). Such "bad genes" may explain why autism persists, and why, despite its high heritability, decades of gene-hunting have found so few susceptibility alleles that replicate across studies (Veenstra-Vanderweele et al. 2004).

This property of fitness indicators would also explain why individuals with autism, compared with their unaffected siblings, carry far more new mutations (i.e., not present in their parents) and why those mutations occur at such diverse genetic loci (Sebat et al. 2007). It would also explain why a recent analysis of autism risk (Zhao et al. 2007) concluded that new mutations cause the vast majority of cases.

Among the bad genes that cause autism, those that most impair general fitness will be the rarest, but they will also have the highest penetrance with respect to autism. This would explain why the few known genetic causes of autism are rare and are associated with severe disturbances in development, such as tuberous sclerosis and fragile X syndrome (Cohen et al. 2005).

Also, sperm production in males, especially older males, involves many more cell-copying events than egg production does in females. For example, mature human females carry eggs that have gone through only 23 DNA replications, whereas 30-year-old males carry sperm that have gone through about 380 DNA replications, and 50-year-old 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 may explain why a large retrospective study found that

the risk for autism increases with advancing paternal age (Reichenberg et al. 2006). Offspring of men age 40 or older were nearly 6 times as likely to have ASD compared with offspring of men younger than 30. Maternal age had no effect on ASD risk after adjusting for paternal age. Higher mutation load in older sperm may also explain why paternal age is an independent risk factor for birth complications (Tang et al. 2006). If so, then birth complications may be associated with autism (Glasson et al. 2004) not only because they interfere with neurodevelopment but also because some mutations lead, through separate pathways, to both birth complications and aberrant neurodevelopment, as suggested by the authors of a recent review (Kolevzon et al. 2007).

7. Some Particular Genes May Increase the Fitness Sensitivity of Indicators

Genes that boost fitness sensitivity would constitute a second type of susceptibility gene (in addition to "bad genes") that should replicate better across populations. The possibility of such genes arises from theoretical models showing that sexually selected fitness indicators could have evolved through the successive accumulation of genes that increase fitness sensitivity (Hasson 1989; Pomiankowski and Moller 1995; Rowe and Houle 1996). Parentally selected fitness indicators might have evolved in a similar fashion, though no explicit evolutionary-genetic models have been developed (Queller 1994).

Genes that increase fitness sensitivity would produce more effective charm among the few infants with high overall fitness (i.e., low mutation load), at the expense of even less effective charm, and an increased rate of autism, among less fit individuals (Fig. 1). In general, such alleles would promote the development of still more complex and energy-demanding brain systems for charm. If developed correctly, these brain systems would produce infant behaviors that are even more successful at attracting maternal resources. However, because they are more complex and energy demanding, they are even more likely to develop with serious errors if the genome contains fitness-reducing mutations or the developing individual is exposed to environmental hazards.

Increased fitness-sensitivity alleles are favored if the benefit (in terms of lifetime reproductive success) to high fitness (low mutation load) offspring from receiving extra maternal resources exceeds the cost of the reduced delivery of maternal resources to low-fitness offspring. In general, such alleles are favored when the competition for maternal resources is greater and it is therefore even more important to charm mothers. If the degree to which it is important to charm mothers has varied temporally or geographically, then genetic differences may continue to influence the fitness sensitivity of charm.

For example, the degree of genetic polyandry (i.e., the frequency with which females bear successive offspring sired by different males) varies across populations. Because greater genetic polyandry reduces the average relatedness among maternal siblings, it should increase sibling rivalry and thereby should favor higher fitness-sensitivity alleles.

If they exist, alleles that increase fitness sensitivity would be more common among those with autism and their close relatives (Fig. 1). This allows us to predict that, compared with the general population, close relatives of those with autism should show higher variance (1) in childhood charm (e.g., preference for social stimuli), (2) in

its anatomical and neurophysiological bases (i.e., endophenotypes), and (3) in its results (e.g., age at weaning and birth interval, especially following the births of boys).

Moreover, we should find a higher prevalence of autism, especially among males, in populations with historically greater sibling rivalry, such as those with historically higher rates of genetic polyandry. For example, genetic polyandry may be higher among black Africans (Rushton 1996). If so, they should suffer higher rates of autism and show higher variance in fetal growth rate, age at weaning, and birth interval. However, very few epidemiologic studies of autism have considered race, and the results are inconclusive (Dyches et al. 2004).

To find sensitivity-boosting genes, investigators should begin with traits that (1) are abnormal in autism, (2) have the highest variance in the general population, and (3) have even higher variance among the relatives of autistic children. More specifically, investigators should hunt for traits that have a high coefficient of additive genetic variance (trait genetic variance divided by phenotypic mean rather than by phenotypic variance), indicating that many mutation-vulnerable loci are responsible for the phenotypic variation (Houle 1992). Candidate traits include a wide variety of measures found to be abnormal in autism, including behavioral measures such as social responsiveness (Constantino et al. 2006), anatomical measures such as digit length ratios (Klin et al. 2002), and measures of social cognition such as recognition of socially relevant information from faces (Adolphs et al. 2001).

Note that genes that increase fitness sensitivity would be more common not only among those who score in the autistic range on a particular trait (e.g., social responsiveness) but also among those who score at the opposite extreme (see Fig. 1). This is important because linkage and association studies have generally assumed that susceptibility genes are most common among those with autism or those who score near the autistic extreme of an endophenotype. To detect genes that boost fitness sensitivity, linkage and association studies must compare those with average scores on a candidate trait with those scoring at either extreme.

Particularly important is that the brains of autistic children grow extremely fast, especially during the first year of life, reaching near adult size between ages 2 and 4, when growth stops abruptly (Redcay and Courchesne 2005). This rapid growth, which occurs during the period of weaning conflict, could be evidence of genes for earlier and more fitness-sensitive development of brain systems underlying charm. As we discussed earlier with respect to the timing of indicator display (property 2), rapid brain growth would increase the risk of developmental defects especially in low-fitness individuals, while allowing high-fitness individuals to develop charm earlier. Consider a gene that produced more rapid growth of brain systems for charm regardless of individual fitness. In low-fitness individuals, that growth would be associated with neurodevelopmental defects, impaired social skills, and a higher rate of autism. At the other extreme, high-fitness individuals would also show rapid growth but would develop superior social skills. Thus, in the general population, higher growth rates should be associated with higher variance in social responsiveness, including higher proportions at both extremes (highly autistic and highly charming). Data relevant to this prediction may already exist because pediatric clinics routinely measure both attainment of social and language milestones and head circumference (a reasonable proxy for brain size in infants and children; Redcay and Courchesne 2005).

Thus, our hypothesis predicts two distinct types of genetic differences or alleles that increase the risk of autism. First, and most important, is a large number of diverse fitness-reducing mutations (bad genes) at a large number of loci affecting a wide range of processes in multiple cell types and organ systems. This is because autism is part of a signal, which we have termed "charm," that evolved to reveal the extent to which offspring are free of such mutations. Second is a much smaller number of genetic differences that influence the fitness sensitivity of charm. These alleles fine-tune fitness sensitivity to the optimum level needed given the expected intensity of sibling rivalry. They will be present only to the extent that sibling rivalry has varied across human populations. Whereas fitness-reducing mutations will be associated with autism, genes that increase fitness sensitivity will be associated not only with autism but also with its opposite—they should be more common among unusually healthy and charming children that successfully solicit the highest levels of maternal investment.

8. Parental Preferences Coevolve with the Indicator

As offspring evolve traits that attract more parental resources, parents should evolve to be choosier about which offspring get more resources, thereby further increasing the fitness sensitivity of the indicator trait, and increasing variance in the quality of the display. This leads to three predictions regarding autism. First, it should be more common when offspring have been under evolutionary pressure to extract more resources from parents (e.g., given higher rates of genetic polyandry), or when parents have been under evolutionary pressure to be choosier about which offspring get a disproportionate share of resources (e.g., because of frequent famine). Second, women of childbearing age should be especially skilled at detecting differences in social responsiveness among infants and children, and the skill should be better developed among women from populations with historically high rates of genetic polyandry. Third, autism should be unattractive to parents and provoke some degree of parental disinvestment, ranging from mild neglect to lethal physical abuse, depending on the autism's severity, the parent's resources, and the number and fitness of current and potential offspring.

9. Parents May Imprint Genes that Influence Expression of Indicators

When females bear offspring by more than one male, fathers have an evolutionary incentive to cause their offspring to extract more maternal resources, if possible, than would be optimal for the mother's own reproductive success. They act on this motive by "genomic imprinting": silencing genes in sperm that would otherwise limit resource extraction from the mother. Mothers counter the father's strategy by silencing genes in ova that would otherwise facilitate resource extraction from themselves. The end result is a sort of tug of war in which the flag on the rope barely moves (reviewed in Moore and Haig 1991).

Rarely, one parent fails to transmit to offspring the crucial active gene, and this provides a window onto the conflict between mothers and fathers. For example, Prader-Willi syndrome (PWS), a rare genetic disorder, is caused by the absence of normally active paternal copies of maternally silenced genes at a particular chromosomal location. Such offspring should be especially poor at extracting

resources. Indeed, infants with PWS have little appetite, are unable to suckle, and sleep excessively (Haig and Wharton 2003).

Angelman Syndrome (AS), another rare genetic disorder, appears to be the mirror image of PWS. AS is caused by the absence of normally active maternal copies of paternally silenced genes in the same chromosomal region. Such offspring should be unusually good at resource extraction. Consistent with theory, children with AS are unusually sociable, smile and laugh frequently, and sleep little. One interpretation is that smiling is an important means of eliciting maternal care and feeding (Brown and Consedine 2004). However, the active copies of genes silenced by the other parent have many other effects because children who lack them have many other abnormalities as well. AS children, for example, are profoundly mentally retarded, and this may explain why, despite their superficial sociability, they also suffer higher rates of autism (Peters et al. 2004).

Since parentally selected fitness indicators are adaptations for extracting resources from parents, we expect parents to imprint the underlying genes. Fathers should silence the genes underlying charm variance in an attempt to produce greater charm in all offspring, while maternal imprints should counter this effect and maximize fitness sensitivity such that the greater the mutation load, the greater the reduction in charm. Mothers might achieve this via imprints that make it more difficult for babies to develop the brain systems underlying charm, thereby ensuring that only the fittest develop these brain systems to their fullest extent.

This may explain a curious finding among Turner syndrome females. These individuals suffer a wide range of developmental abnormalities because they have just one X chromosome (derived from either parent) and no Y chromosome. Skuse (2000) found that social and communicative skills were impaired in the direction of autism among those with a maternally derived X chromosome but were normal among those with a paternally derived X chromosome. He suggested that mothers may have silenced a gene on the X chromosome in such a way as to impair social and communicative skills, and that such an imprinted gene could explain the higher rate of autism in boys.

But why would mothers impair the social and communicative skills in their sons? Our hypothesis suggests that it is a maternal strategy to further increase the fitness sensitivity of charm in boys, and thereby better match resource allocation to offspring fitness. Imprinting of genes on the X chromosome is special (Davies et al. 2006) because sons have just one copy—always maternally derived—whereas daughters have one from each parent. So, maternal imprints on the X chromosome have a greater effect on sons because sons possess no paternally derived X chromosome to counter the effect of the maternal imprint. This differential effect may be just what mothers need to optimize their maternal resource allocation because boys are more costly to rear and more variable in reproductive success. Increasing the handicap (Zahavi 1975) in sons would boost the fitness sensitivity of charm. Such imprinting could have evolved if the benefit of allocating maternal resources preferentially to high-fitness sons exceeded the costs of impairing social and communicative skills in low-fitness sons.

Maternal imprints need not be restricted to the X chromosome. Indeed, the overall fitness-sensitive nature of charm might arise, in part, from interplay between competing parental imprints.

We are not the first to propose a role for genetic imprinting in autism. However, we are the first to propose that parental imprints may modify the fitness sensitivity of a parentally selected fitness indicator. For example, Badcock and Crespi (2006) proposed that autism arises from enhanced effects of normally active paternally derived genes and decreased effects of normally active maternally derived genes. In contrast, our hypothesis predicts the opposite—that both situations should decrease charm's fitness sensitivity and thereby reduce the risk of autism.

Conclusions

The evolutionary biology of sibling rivalry, progeny choice, and costly signaling offers a new perspective on childhood mental disorders. It led us to propose that autism is the low-fitness extreme of a parentally selected fitness indicator—a trait we call "charm." Our proposal is an adaptationist analysis of autism and not a claim that parental neglect or abuse of afflicted children is "natural" or justified in any moral sense.

Because we focused on fitness-indicator theory, our hypothesis differs from previous evolutionary hypotheses regarding autism in several ways. First, it does not propose that autism itself is adaptive, or that the responsible genes produce consistent fitness benefits among the relatives.

Second, our model does not propose that autism 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. Moreover, the harmful effects of these mutations on vital processes and, therefore, on the risk for autism will depend on exposure to environmental hazards (e.g., prenatal infection and malnutrition) that further strain mildly impaired vital processes. Thus, our hypothesis predicts that most of the genetic causes of autism will be highly pleiotropic genes that interact strongly with environmental hazards. However, because autism itself is rare (10 to 20 individuals per 10,000), it is the milder forms of dysfunctional charm that carry most of the fitness information conveyed from babies to mothers. It is charm that evolved as a fitness indicator, while autism is its most extreme low-fitness version.

Third, our model at this stage remains intentionally vague about the nature of "charm"—it does not predict exactly which brain systems go awry in autism, only that they will be brain systems required for attracting parental resources either directly or in competition with rivals. Social responsiveness that attracts breast-feeding and lengthens birth interval is one possibility, but many of our explanations and predictions do not hinge on whether the indicator trait is infant social responsiveness or something else.

The strength of our hypothesis lies not in its consistency with any particular known fact about autism. For example, other hypotheses can explain why it appears to be part of a phenotypic continuum or why it appears to be highly polygenic. However, our hypothesis explains a very wide range of facts and makes several surprising and testable predictions. In Table 1 we list the test results that would be contrary to our predictions and would falsify either the general hypothesis or the special case.

Table 1 Findings that would *falsify* the hypothesis

General hypothesis: Autism is the low-fitness extreme of parentally selected fitness indicator
• Just a few genetic abnormalities cause most cases of autism in diverse populations
• Autism is not more common in populations with historically higher rates of genetic polyandry (the frequency with which females bear offspring by different fathers)
Special case: The indicator trait is the social skill needed to prolong breast-feeding and delay the arrival of a sibling
• Across natural fertility populations, differences in age at weaning do not predict differences in age at onset of autism
• Autism is not associated with scarce environmental resources (e.g., famine)
• Autism is not associated with early weaning
• Prolonged intensive breast-feeding and increased maternal presence do not reduce the expected rate of autism among the younger siblings of those with autism
• Close relatives do not show higher variance in (a) social ability, (b) its anatomical and neurophysiological bases, or (c) the interval before birth of a sibling
• Infant social ability does not correlate positively with both underlying fitness (e.g., long-term health and reproductive success) and parental resource allocation (e.g., intensity and duration of breast-feeding)

The general progeny-choice hypothesis alone would explain (1) why autism begins in childhood, (2) why it is highly heritable, (3) why the responsible genes have been so hard to find, (4) why it is more common in boys and more severe in girls, and (5) why it is associated with environmental hazards, developmental abnormalities, and increased mortality. It predicts that (a) most of the responsible genes will be fitness-reducing mutations that remain lineage-specific and therefore will not replicate well across populations, (b) any genes that do replicate well across populations will be those that increase charm's general sensitivity to fitness, and (c) autism will be more common in populations with historically high rates of genetic polyandry.

In addition to the general hypothesis, we speculated that charm might involve infant social behaviors that prolong breast-feeding and thereby secure a longer interval before the birth of a sibling (i.e., a higher proportion of a mother's total lifetime reproductive effort). This special case of our general hypothesis would explain (1) why autism begins before age three and (2) why it impairs social behaviors so profoundly. It predicts that (a) within populations, age at onset of autism will parallel age at onset of weaning; (b) autism will be associated with scarce environmental resources and early weaning; (c) delaying weaning may protect against autism; (d) close relatives will show higher variance in social ability, higher variance in its anatomical and neurophysiological bases, and higher variance in subsequent birth interval; and (e) genes (and maternal imprints) may exist that increase variance in social responsiveness and thereby increase the risk of autism. In addition to these predictions about autism, this specific version of the hypothesis predicts that infant social ability will correlate positively with both underlying fitness and parental resource allocation (e.g., intensity and duration of breast-feeding).

Although we have focused on brain systems evolved to charm breast-feeding mothers, those same systems might develop into systems for a wide range of social behaviors evolved to attract other resources from parents (e.g., food, learning opportunities, social connections) and eventually from other individuals (e.g., mates, friends, allies). Indeed, the same brain systems used initially for charming parents might eventually develop into brain systems for charming potential mates or

intimidating sexual rivals. This suggests another reason why mothers might pay so much attention to this quality in offspring—charming offspring may develop into attractive mates.

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