neural development need not be expressed in brain cells per se, however, mutations across an even larger proportion of the genome may affect neurodevelopmental disorders. As this theory expects, neurodevelopmental disorders covary with markers of developmental disruption or instability (e.g., minor physical anomalies and fluctuating asymmetry; see Yeo et al. 1999).

Disorder-specific features may arise because disruptions have different outcomes depending on their timing or differentially affect specific neural systems. Alternatively, disorders may be affected by specific factors in addition to developmental instability (see Yeo et al. 1999).

Antagonistic coevolutionary processes. K&M contrast three different kinds of evolutionary models of genetic variation: *mutation-selection balance*, *neutral variation*, and *balancing selection* – maintenance of variation because selection actually favors multiple alleles (e.g., through heterozygote superiority, spatiotemporal oscillations in selection, and frequency-dependent selection). But a fourth possibility they do not mention exists: rapid evolution at loci, particularly those involved in *antagonistic coevolutionary processes*.

Species may antagonistically coevolve with other species (e.g., in predator-prey or host-pathogen roles). New adaptations in one species evoke selection on the other to evolve counteradaptations. Antagonistic coevolution (or Red Queen processes; Van Valen 1973) may persist through evolutionary time, resulting in continual change in both species. Genetic conflicts of interest between males and females, mothers and fetuses, or pairs of cooperators may fuel persistent antagonistic coevolution of genes *within a species*, as well (Rice & Holland 1997).

Red Queen processes produce predictable evolutionary outcomes: (1) *Relatively rapid evolution*: Because new adaptations in a coevolving party change selection on the other party, alleles at coevolving loci should be subject to new positive selection relatively frequently (e.g., Swanson et al. 2001; Wyckoff et al. 2000). (2) *Interindividual variation*: More often than most loci, coevolving loci should be in states in which a new, favored allele has not yet become near-fixed in the population and coexist with alleles favored previously. (3) *A non-negligible level of maladaptation within populations*: Neither conflicting party is likely to adapt perfectly to the other party. The load of maladaptation will be carried disproportionately by individuals who lack newly favored alleles.

A Red Queen process that might explain some variation in psychopathological conditions is maternal-fetal coevolution (see also Gangestad & Yeo 1997). Fetal genes maximally benefit from a greater flow of nutrients from the mother than the level maximizing maternal fitness (Trivers 1974). Maladaptive outcomes of pregnancy (e.g., maternal hypertension, gestational diabetes) may be by-products of ensuing antagonistic coevolution (Haig 1993). Maladaptive by-products could include disruption of fetal epigenesis, resulting in neurodevelopmental disorder. In this model, some fetal genes associated with poor development (e.g., developmental instability) now once were not, for they once did not coexist with current maternal genes that promote counteradaptation. (The model is akin to spatially varying selection, but not as a form of balancing selection; cf. target article, sect. 5.3.)

Some fetal genes involved in this conflict are imprinted – expressed differently depending on parent-of-origin. Imprinting is selected because fetal genes from fathers are also in conflict with those obtained from mothers (Haig 2000). Among genes that account for more than a negligible amount of variance in psychopathology, imprinted genes (or other genes involved in the imprinting process; e.g., genes that control imprinting; Burt & Trivers 2000) may be over-represented, for example, on schizophrenia, autism, or bipolar disorder (see Bah et al. 2004 [GRIK2]; Corradi et al. 2005 [GNAL]; DeLisi et al. 2002 [chromosome 22]; Francks et al. 2003 [chromosome 2]; Xu et al. 2001 [CHRNA7]); on attention-deficit hyperactivity disorder (ADHD) (see Borglum et al. 2003 [dopa decarboxylase]; Hawi et al. 2005 [9 genes putatively associated with ADHD]; Kirley et al. 2002 [TH]; Mill et al. 2004 [SNAP-25]). Only a few percent of genes are imprinted (on the mouse genome, see Luedi et al. 2005).

Findings in linkage studies have historically been fragile. Cautious interpretation is advised. Nonetheless, even if most variation in psychopathology is a result of rare mutation, some single genes probably do have non-negligible effects. One evolutionary theory K&M do not mention points toward genes involved in Red Queen processes.

Autism: Common, heritable, but not harmful

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Abstract: We assert that one of the examples used by Keller & Miller (K&M), namely, autism, is indeed common, and heritable, but we question whether it is harmful. We provide a brief review of cognitive science literature in which autistics perform superiorly to non-autistics in perceptual, reasoning, and comprehension tasks; however, these superiorities are often occluded and are instead described as dysfunctions.

We appreciate Keller & Miller (K&M) grappling with the age-old evolutionary paradox of why certain human phenotypes are so common, so heritable, but so harmful. In their treatise, K&M provide several examples of what they refer to as mental disorders, clumping together numerous phenomena, including schizophrenia, bipolar disorder, depression, phobias, panic disorders, Tourette's syndrome, obsessive-compulsive disorder, low intelligence, anorexia, and autism. We – a cognitive scientist, a research psychiatrist, and an autistic (who conducts cognitive science research) – are most interested in K&M's inclusion of autism. Therefore, we restrict ourselves to that exemplar, agreeing that autism is common and heritable but questioning whether autism is harmful.

Autism is definitely a common phenotype – even more common than K&M report. Current prevalence estimates are 200 per 100,000 for DSM-IV (American Psychiatric Association 1994) defined "autistic disorder" and around 600 per 100,000 for the entire autism "spectrum" (Chakrabarti & Fombonne 2005). A rash of public attention has spotlighted what are considered dramatic recent increases in autism prevalence, but our most reasoned logic suggests that the increases are due to purposely broadened diagnostic criteria, yoked with dramatically raised public awareness and conscientiously improved case finding (Gernsbacher et al. 2005). And when some lay spokespersons mistakenly suggest that autism first appeared in society only in the 1940s (Kennedy 2005), they are confusing the codification of the phenotype with its onset (see Frith [1989] for a convincing, albeit speculative, history of autism in society).

Autism is also a highly heritable phenotype, based on estimates from twin studies and sibling-recurrence rates. However, the existing heritability estimates warrant caution in interpretation. The twin-based estimates are derived from only a handful of studies, which are based on only a few handfuls of twins, and estimating sibling recurrence requires a reliable population prevalence rate.

But is autism a "harmful" phenotype? Primarily, K&M employ an evolutionary connotation of *harmful*, namely,

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lowered fitness (i.e., reduced fertility rates). Perhaps any extreme phenotype will be less reproductively fit, be it the low levels of intelligence that K&M include as an example or the extremely high levels of intelligence found in adults identified during adolescence by their academic precocity (Lubinski et al. 2006). Certain cognitive phenotypes might also lead to lowered fitness. The prolific inventor Nikola Tesla, who is reported to have been celibate and whose life history reveals numerous autistic traits, proclaimed:

> I do not think there is any thrill that can go through the human heart like that felt by the inventor as he sees some creation of the brain unfolding to success.... Such emotions make a man forget food, sleep, friends, love, everything.... I do not think you can name many great inventions that have been made by married men. (Pickover 1999, p. 35)

K&M also verge into the more vernacular meaning of "harmful." They refer to mental disorders as "harmful dysfunctions" (sect. 1.2, para. 2), which are "disabling" and "debilitating" (sect. 1, para. 2), which cause "human suffering" (sect. 1.1, para. 4), and which are "disastrous to survival" (sect. 1.2, para. 6). K&M view "mental disorders" such as autism as "glaring exceptions" to the "awesome power of natural selection" (sect. 2, para. 1.).

However, whereas K&M assert that Darwinian psychiatrists and evolutionary psychologists "often go to torturous lengths to find hidden adaptive benefits" (sect. 1.1, para. 3), we assert that cognitive scientists often go to torturous lengths to occlude obvious adaptive benefits. The empirical literature is replete with demonstrations of autistics' superiority in numerous perceptual, reasoning, and comprehension tasks: Across a wide range of age and measured intelligence, autistics perform significantly better than non-autistics on block design, a prominent subtest of Wechsler-type scales (Shah & Frith 1993); on embedded figures tests, which require rapid visual identification of a target figure amid a complex background (Shah & Frith 1983); on recognition memory (Toichi et al. 2002); and on sentence comprehension (Just et al. 2004); and autistics are more impervious than are non-autistics to memory distortions (Beversdorf et al. 2000) and misleading prior context (Ropar & Mitchell 2002). Such superiorities are not isolated phenomena; some theorists argue that such superiorities abound in autism (Mottron et al. 2006).

Quite compellingly, each of these statistically significant demonstrations of autistic superiority is labeled by its authors as a harmful dysfunction. Autistics' superior block-design performance is labeled "weak central coherence," symptomatic of dysfunctional "information processing in autism" (Shah & Frith 1993, p. 1351). Autistics' superior performance on embedded figures tests is considered "consistent with the cognitive-deficit theory proposed by Hermelin and O'Connor (1970) ... due to a central deficiency in information processing" (Shah & Frith 1983, p. 618). Autistics' superior recognition memory performance is attributed to deleteriously "enhanced attention to shallow aspects of perceived materials" (Toichi et al. 2002, p. 1424); their superior sentence comprehension is described as being "less proficient at semantically and syntactically integrating the words of a sentence" (Just et al. 2004, p. 1816); their superior imperviousness to memory distortions is explained by "representations in the semantic network [that] may be associated in an aberrant manner" (Beversdorf et al. 2000, p. 8736); and their superior resistance to misleading prior context is attributed to their perception being "less conceptual" (Ropar & Mitchell 2002, p. 652).

Disorders are defined by criteria that vary with cultural, societal, and medical values. As K&M write:

> Mental disorder categories may reflect a mix of historical convention, diagnostic convenience, innate categorization biases in person perception, and common final pathways of partially overlapping yet distinct dysfunctions. This suggests that *the number of loci affecting a mental disorder depends in large part on the way human minds categorize behavioral symptoms*. (target article, sect. 6.4, para. 6; emphasis in original)

We couldn't agree more. As autistic Suzanne Shaw opines:

> People say that in the world of the blind the one-eyed man is king, but I think they are mistaken. In the world of the blind the one-eyed man would be a freak, and his eye might even disable rather than enable him. Eyes are wonderful things to be sure, but they are only useful in a society that is built to require them. (http://www.as-if.org.uk/discrim.htm)

We would add that they are only useful in a society that is open to appreciating them.

Heritable mental disorders: You can't choose your relatives, but it is they who may really count

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Abstract: Keller & Miller (K&M) briefly mention and promptly dismiss the idea that genes for harmful mental disorders may confer certain advantages to affected individuals. However, the authors fail to consider that the same genes (in low doses or reduced penetrance) may be adaptive for relatives, and that this may in part explain why they are retained in the gene pool.

While Keller & Miller (K&M) here rightly address ancestral factors in developing their critique of the three evolutionary models under their consideration, they only briefly consider the idea that negative fitness effects from mental disorders (e.g., schizophrenia, bipolar disorder) may be offset by other potential benefits (e.g., creativity). They fail to consider that genes for mental disorders may be retained, even selected for, in the genome when they convey certain other advantages, perhaps in different environments or contexts, not necessarily in those directly affected by the disorder in question, but in close relatives, when present in low doses or exhibiting reduced penetrance. A model for this is the recent evidence that genes associated with a homosexual orientation in males (where retention and transmission in the genome are clearly less likely in the affected male) are also associated with increased fertility in close female relatives (Camperio-Ciani et al. 2004). It is also well established that close relatives of autistic individuals exhibit unexpectedly high frequencies of choosing certain types of career where they excel, especially those involving computation, information technology, programming, and accountancy, where social and communicatory skills are at a reduced premium (see Baron-Cohen & Belmonte 2005).

Similar factors may operate, additional to any possible compensatory effect in the affected individuals themselves (see Bradshaw [2001] for a review), with "song and dance" and ball skills associated with Tourette's syndrome, and excellence in tasks involving attention to detail or general or personal care or hygiene associated with obsessive-compulsive disorder. In uncertain times or in the case of war or threats, the capacity for suspicious and critical distrust (verging on the paranoic) associated with schizophrenia, and the diffuse attentional focus and impulsivity associated with attention deficit hyperactivity disorder (ADHD), may be beneficial. Despite the denial by K&M of a significant link between creativity and mood disorders, there is indeed considerable evidence to support such an effect – such as the study by Simeonova et al. (2005), who found that children of adults with bipolar disorder had higher scores than did controls on a measure of creativity (see also Andreasen 2005). Although K&M correctly draw our

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