species and laboratory experiments indicates that strong selection leads to maladaptive, more or less transient by-products, on account of linkage and antagonistic pleiotropy in populations out of equilibrium (e.g., see Andolfatto 2001; Lu et al. 2006). These data suggest that selection for the traits that have "made us human" (cf. Horrobin 1998), especially the neural systems underlying language and social cognition, have led to psychosis as a secondary result. Data are now available to test this hypothesis more directly, using the human haplotype map to test for selective sweeps in regions associated in genome scans with psychosis, such as 1q21 (Voight et al. 2006). Many of the selective sweeps inferred from such data (Voight et al. 2006) are remarkably recent (less than 20,000 years old). As a result, allele frequencies may be out of equilibrium, and equilibrium-based population-genetic models for explaining standing levels of variation, based on antagonistic pleiotropy or related mechanisms, do not apply.

Second, evidence for multilocus overdominance comes from multiple studies showing increased fitness, compared to the general population, in first-order relatives of schizophrenics. The study by Haukka et al. (2003) found such an effect for females, but not for males, and they cite four previous studies supporting such a difference. A stronger pattern in females fits with the less-debilitating nature of psychosis in this sex (Moriarty et al. 2001), and such a sex bias was also found by Fananas and Bertranpetit (1995) and Bassett et al. (1996). Nettle and Clegg (2006) also report an association between increased mating success and measures of schizotypy. The sample in Haukka et al. (2003) is indeed very large, but no single such study can be definitive or serve to estimate selective parameters quantitatively, given population-specific effects and the evolutionary time scale involved. The upshot is that six independent studies support a general pattern of balancing selection, apparently related to positive aspects of schizotypy.

Third, there is substantial evidence for mechanisms that can generate multilocus balancing selection on relevant aspects of cognition. The causal links between measures of schizotypy and measures of creativity and divergent thinking are much stronger than Keller & Miller (K&M) imply, and they comprise diverse evidence from functional imaging, neurophysiology, neural network modelling, genomics, and psychological experiments, as well as the biographical and survey studies discussed by Waddell (1998) (Abraham et al. 2005; Brugger 2001; Fisher et al. 2004; Folley et al. 2003; Folley & Park 2005; Hoffman et al. 2004; Lauronen et al. 2004; Nettle 2001; in press; Smalley et al. 2005). Many of these studies converge on a key role for increased right-hemisphere activation in language function (Mohr et al. 2005). They also emphasize that understanding psychosis requires analyses of its healthy analogue in components of schizotypy, given the clear pathologies and fitness reductions caused by psychosis itself, and the proposed "cliff-edged" form of the balancing fitness function (Nesse 2005).

Finally, strong recent selection on language and cognition coupled with antagonistic pleiotropy or linkage, and multilocus overdominance, are not the only possible mechanisms for the evolution and maintenance of psychosis in which selection plays a central role. A non-exclusive model involves effects of intragenomic conflict, mediated by sexual conflict or by genomic imprinting in brain development (Badcock & Crespi 2006; Burt & Trivers 2006). The clearest evidence for genomic-imprinting effects come from the oppositely imprinted disorders Prader-Willi syndrome, which engenders high rates of psychosis (Vogels et al. 2004), and Angelman syndrome, which shows a high incidence of autism (Cohen et al. 2005; Peters et al. 2004). Genome scans also demonstrate strong imprinted-gene effects in schizophrenia (Francks et al. 2003), bipolar disorder (Kennedy et al. 2003), and autism (Badcock & Crespi 2006). Genomic conflict may help maintain genetic variation via continual strong selection for divergent optima, as in host-parasite conflicts mediated by major histocompatibility complex (MHC) loci, the most polymorphic loci in the human genome. Genomic imprinting effects also provide a persuasive hypothesis for the paternal age effect on schizophrenia risk (Malaspina 2001; Sipos et al. 2004), given that mutations during spermatogenesis appear insufficient to explain such patterns (Farrer et al. 1992; Reik et al. 1993; Tiemann-Boege et al. 2002). To the extent that intragenomic conflict is involved in cognitive traits, discussions of adaptation must focus at the level of genes, as organism-level adaptive value can no longer be assumed (Burt & Trivers 2006).

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Why the adaptationist perspective must be considered: The example of morbid jealousy

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Abstract: We describe delusional disorder–jealous type ("morbid jealousy") with the adaptationist perspective used by Darwinian psychiatrists and evolutionary psychologists to explain the relatively common existence and continued prevalence of mental disorders. We then apply the "harmful dysfunction" analysis to morbid jealousy, including a discussion of this disorder as (1) an end on a continuum of normal jealousy or (2) a discrete entity.

An evolutionary psychological approach to explaining the relatively common existence and continued prevalence of mental disorder historically has required explaining a disorder's potential adaptive benefits. As Keller & Miller (K&M) note, Darwinian psychiatrists and evolutionary psychologists assume an adaptationist position, thus keeping natural selection at the etiologic forefront. If it is theoretically possible and empirically verifiable that mental disorder susceptibility alleles increased fitness in some ancestral conditions, then a balancing-selection explanation of the existence and prevalence of mental disorders may be justified.

Delusional disorder–jealous type or "morbid jealousy" is a disorder that causes individuals to misinterpret everyday actions as cues to a partner's sexual infidelity. Constant accusations of infidelity, vigilant monitoring of a partner's behavior, and restricting a partner's actions are typical of individuals diagnosed with morbid jealousy (see the *Diagnostic and Statistical Manual of Mental Disorders*, American Psychiatric Association [2000]; see also, Kingham & Gordon 2004; Shepherd 1961; Vauhkonen 1968). The benefits and costs of morbid jealousy are well documented (e.g., Buss 2000; Enoch & Trethowan 1979; Kingham & Gordon 2004; Mowat 1966; Shepherd 1961). If morbid jealousy is an extreme form of normal sexual jealousy, it is reasonable to hypothesize that morbid jealousy may thwart partner infidelity, perhaps more effectively than does normal sexual jealousy, thereby increasing the fitness of ancestral individuals with morbid jealousy. Whether the alleles associated with the costs of morbid jealousy – such as decreased daily functioning, increased risk of mate defection, and increased susceptibility to other debilitating mental disorders – would be exactly balanced through antagonistic pleiotropy by increases in the fitness payoffs of the associated benefits is unknown. Despite empirical challenges, an adaptationist perspective using balancing selection, specifically antagonistic pleiotropy, may explain the relatively common existence and continued prevalence of morbid jealousy and perhaps additional mental disorders.

Wakefield (1999a; 2005) has argued that mental disorders can only be classified as such when they are harmful dysfunctions. A *dysfunction* is a failure of a mechanism to perform as it was designed by natural selection. According to this definition, the disorder cannot be the function of a naturally selected mechanism. Therefore, a dysfunction of jealousy mechanisms would occur when they failed to motivate behaviors designed to prevent a partner's infidelity. Individuals diagnosed with morbid jealousy do deploy behaviors that function to prevent partner infidelity, even if the cues that activate the jealousy mechanisms are imagined by the individual. Perhaps morbid jealousy does not meet the dysfunction criterion and therefore should not be considered a mental disorder.

Wakefield's (1999a; 2005) harmful dysfunction analysis specifies a second criterion that must be met for a mental disorder to be considered as such. The disorder must generate harm, as defined by society. To conclude that morbid jealousy is not a disorder without assessing the associated harm would be a mistake, according to the harmful dysfunction analysis. Lives are disrupted, including the lives of the morbidly jealous individuals themselves as they constantly monitor their partner's behavior (e.g., Seeman 1979). Substantial stress is added to the relationship as morbidly jealous individuals constantly accuse their partner of infidelity (e.g., Vauhkonen 1968). Potential rivals may be derogated or attacked, partners of the morbidly jealous may be psychologically and physically abused, and sometimes this assault escalates to murder (e.g., Kingham & Gordon 2004; Mowat 1966; Shepherd 1961). Although morbid jealousy is harmful, is it more harmful than normal sexual jealousy? In fact, the greatest predictor of intimate partner homicide is sexual jealousy (Daly & Wilson 1988). It is possible that morbidly jealous individuals are more abusive toward their partners or are more likely to murder them than are individuals who experience normal sexual jealousy, but research has not investigated this possibility.

Morbid jealousy may be explained best not as a discrete categorical mental disorder, but as a continuation of normal sexual jealousy. Before this determination can be made, however, several factors must be examined (J. C. Wakefield, personal communication, March 20, 2006). First, we need to determine whether the morbid jealousy tail of a normal curve hides discrete points of jealousy disorders. For example, there are many causes of low intelligence. However, a smooth normal curve of intelligence would group these distinct causes together and would hide the individual causes of low intelligence. The same might be true of a normal sexual jealousy curve. Examining individual cases of morbid jealousy and comparing the symptoms and behaviors could help determine whether a normal sexual jealousy curve is grouping together distinct causes of morbid jealousy. If there are not multiple, distinct cases of morbid jealousy, then it could be argued that morbid jealousy is a continuation of normal sexual jealousy.

Second, we need to determine whether the morbid jealousy end of a sexual jealousy curve is fitness enhancing. Previous research has documented the adaptive benefits of normal sexual jealousy; notably, that it may prevent partner infidelity (e.g., Buss 2000). If morbid jealousy has similar adaptive benefits, this might provide further evidence that it should be viewed as part of a continuum of normal sexual jealousy.

Third, morbid jealousy may not be produced by a dysfunction of jealousy mechanisms, but instead by a dysfunction of related mechanisms. For example, individuals with morbid jealousy may have dysfunctions in mate-retention mechanisms. If this is the case, then morbid jealousy could not be considered continuous with normal sexual jealousy, as these related dysfunctions do not occur with sexual jealousy. This third issue could be investigated by examining individuals diagnosed with morbid jealousy to determine whether they have other, related dysfunctions.

Whether morbid jealousy is a discrete categorical mental illness or part of a continuum of normal sexual jealousy remains to be determined. We have discussed three research questions that could help address this question. Investigation of these questions through careful examination of individuals with morbid jealousy may lead to clarification of delusional disorder–jealous type, and may represent a model that could be used to clarify other mental disorders. Additionally, this clarification should lend support to continued use of the adaptationist approach and should provide a better understanding for the continued prevalence of disorders.

Mutations, developmental instability, and the Red Queen

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Abstract: We address two points. First, one must explain how different, rare mutations ultimately lead to common psychopathological conditions. The developmental instability model offers one solution. Second, Keller & Miller (K&M) perhaps miss the major processes other than variation fueled by rare deleterious mutations that account for interesting genetic variation in psychopathology, particularly when single alleles have non-negligible effects: Red Queen processes.

Keller & Miller (K&M) argue that much heritable variation in psychopathological conditions is fueled by deleterious mutation, rare at individual loci but ubiquitous in genomes. Ron Yeo, colleagues, and I offered a similar view a decade ago (Gangestad 1997; Gangestad & Yeo 1997; Thoma et al. 2002; Yeo & Gangestad 1993, 1998; Yeo et al. 1997, 1999), albeit less broadly applied to neurodevelopmental disorders (e.g., schizophrenia, dyslexia, attention deficit disorder). K&M do an excellent job of making this case.

Our quibbles pertain to details. We focus on two. First, mutations at many loci produce phenotypic variants much more common than individual mutations. Our model, curiously not mentioned by K&M, may explain how they do so. Second, K&M perhaps miss the most important alternative processes accounting for interesting genetic variation in psychopathology, particularly when single genes account for non-negligible (>1%) variance.

The developmental instability model. Mutations at individual loci are rare. Neurodevelopmental disorders are much more common. A successful theory must explain how different mutations can produce similar outcomes. Though K&M discuss how different "upstream," specific defects can have common "downstream" effects (sect. 6.2), they do not present a particularly compelling, specific model for how this happens.

We have suggested one route: developmental imprecision. Microcircuitry of a computer chip must be manufactured in a dust-free environment, for only then can its design be actualized. Dust that inadvertently becomes part of the chip can affect the functioning of the circuitry in random ways, disrupting design. Similarly, mutations and other developmental stresses can act as "dust" in the environment in which epigenetic processes "manufacture" an organism's phenotype, introducing *developmental instability* and deviations from naturally selected design.

Neurodevelopmental errors may disrupt adaptive coordination of a broad array of processes within developmental systems, particularly as their frequency increases. As disrupted development may channel along particular paths, different perturbations (e.g., mutations) may ultimately have common outcomes. K&M argue that more than half of all human protein-coding genes are expressed in brain tissue and, hence, neural systems capture a large amount of mutational variation. As genes that affect