Abstract: Grouping severe mental disorders into a global category is likely to lead to a "theory of everything" which forcefully explains everything and nothing. Speculation even at the phenotypic level of the single disorder cannot be fruitful, unless specific and testable models are proposed. Inclusive fitness must be incorporated in such models.

Speculation about the evolutionary origin of mental disorders is a peaceful diversion from the challenge of attempting to treat people diagnosed as having severe mental disorders. Keller & Miller (K&M) efficiently dismantle this diversionary toy. What remains of Darwinian psychiatry after their compelling criticism is an approximate model of how the genes leading to severe mental disorders had not been phased out from the general population by natural selection. However, as K&M seem well aware, "severe mental disorders" are very much influenced "by the cultural and inherent person-perception biases ... and the categorization demands of legal, medical, and research systems" (sect. 8, para. 5). So, what is the problem? Their designation of severe mental disorders as a global category predisposes to a "theory of everything" which explains everything and nothing, the most common fault of evolutionary psychopathology.

We introduce as examples several specific and testable models at the phenotypic level of the single disorder. First, let us consider sociopathy: People with sociopathy cheat in reciprocity games – they obtain resources and seldom return them. According to neo-Darwinian formulations, they challenge the cooperative subject (Axelrod & Hamilton 1981; Trivers 1971). Groups of altruists who cooperate and are unable to detect cheaters are easily exploited, leading to their extinction. However, groups of altruists who are able to detect cheaters and discriminate in their cooperative moves will protect their resources (Fehr & Fischbacher 2003). In this regard, sociopathic individuals may improve the altruists' fitness overall. Since the cognitive abilities leading to the cheater's detection are likely to be useful in all kinds of cooperative exchange (Stevens & Hauser 2004), groups of discriminative cooperators will outcompete over groups of non-discriminative cooperators. Sociopathy thus can select for individuals who are more able to detect cheating. Its permanence, thus, is of a parasitic kind: The hosts tolerate some sociopaths in their environment because the sociopaths continuously challenge the hosts' cognitive abilities, as parasites resident in our skin stimulate the immune system and act as a restraint against more virulent invaders.

Patients with schizophrenia often need permanent help: They deplete resources because they are unable to return anything. Let us assume that alleles for schizophrenia spring up in two clusters of families: the families that always help their kin, and the others who never help their kin. Some of the helped kin will arrive at reproducing their own susceptibility alleles, whereas those without help will become extinct. Since helping kin is a trait likely to favour inclusive fitness of the helper (Hamilton 1964), after some generations the alleles for schizophrenia will spread in the population, on account of their hitch-hiking on the helping trait, without adding any hidden benefit to humankind: They fixate in the universal gene pool because they are occasionally linked with some trait inherent to our more general adaptive outfit. Indeed, incidence of schizophrenia does not greatly vary across sites, confirming that the disorder is rooted in our common genetic heritage. Cultures more likely to display a helping attitude towards affected people (such as some Indian enclaves), however, are also more likely to have incidence rates that are higher than the mean (Jablensky et al. 1992), together with a more favourable outcome (Leff et al. 1992), leading in the long term to lower-than-average prevalence rates (Saha et al. 2005).

Female patients with severe anorexia nervosa, by maintaining their body weight below the threshold for ovulation, exclude themselves from reproduction; however, they often behave in a supportive way towards their kin, cooking for them what they themselves are unwilling to eat. This behaviour is assimilated to the "helping at the nest" behaviour described in the wild field and observed to improve kin's reproductive success (Arnold & Owens 1998). Indeed, what matters for the permanence of a gene set is persistence of that set in the pool, generation after generation: Humankind, for example, is thought to carry on the mitochondria of seven ancestral females (Sykes 2001). A "helping at the nest" hypothesis for anorexia nervosa is a testable one, different from the scenario of the "gene for facing famine" (cf. Guisinger 2003).

It is difficult to understand K&M's polygenic mutation-selection balance model: They consider balancing selection as a dynamics whereby two or more alternative alleles are maintained because their net fitness effects balance each other out, so the alleles are not lost by chance or genetic drift (Wilson 1998). A polygenic mutation-selection balance, therefore, is a model whereby a conditional balance is achieved: A mutation produces a decrease in fitness only given a concurrent genetic environment; in the absence of such an environment, the mutation is neutral. Past studies found that the genetic-controlled conditions increasing the risk of obstetric complications are also associated with a higher risk of schizophrenia, for example, in the case of Rh incompatibility (Hollister et al. 1996). Conversely, obstetric complications tend to recur within families, clustering in families that also show a higher representation of subjects diagnosed with schizophrenia (Walshe et al. 2005). Some time ago, we suggested that the genetics of schizophrenia might be explained in part by the genetics of the conditions increasing the risk of obstetric complications (Preti et al. 1998). Foetal brain anoxia likely to result from obstetric complications generally leads to death or to severe motor impairment: We therefore hypothesized that some brain-protecting gene would be necessary to balance the brain-damaging impact of obstetric complications (Preti & Miotto 2005). Whenever obstetric complications occur, schizophrenia develops only in the presence of the protecting gene. A subgroup of offspring of patients diagnosed with schizophrenia was found to bear a statistically reduced risk of developing schizophrenia in adulthood, indeed, as if they were carrying some protective gene (Gottesman & Erlenmeyer-Kimling 2001). Moreover, some studies reported a higher prevalence of successful creative abilities in the mathematical, visual, and spatial domains among the relatives of patients diagnosed with psychosis (Karlsson 1999). Anomalous lateralization (Dragovic & Hammond 2005) could be the link between schizophrenia and superior mathematical ability. In the absence of severe obstetric complications, protective, "left brain" genes would favour creative abilities in the visual and spatial domains; in the presence of such complications, schizophrenia would develop. Is this a possible example of the kind of polygenic balance K&M have in mind?

Behavioural ecology as a basic science for evolutionary psychiatry

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Abstract: To the evolutionarily oriented clinical psychiatrist, the discipline of behavioural ecology is a fertile basic science. Human psychology discusses variation in terms of means, standard deviations, heritabilities, and so on, but behavioural ecology deals with mutually incompatible alternative behavioural strategies, the heritable variation being maintained by negative frequency-dependent selection. I suggest that behavioural ecology should be included in the interdisciplinary dialogue recommended by Keller & Miller (K&M).

Keller & Miller (K&M) say that the heritability of mental disorders presents a problem for explanations in terms of function,

because alleles conferring function should increase in frequency to fixation. They claim the only satisfactory explanation for the high rate of mental disability is that mental disorders are caused by harmful mutations on hundreds of genes. This argument restricts evolutionary psychiatry to disorders which have zero heritability. However, the authors might have underestimated the prevalence and robustness of negative frequency-dependent selection.

I would like to draw the authors' attention to the discipline of behavioural ecology, which is the study of behaviour in relation to its function (Krebs & Davies 1993). Behavioural ecology could be said to be the basic discipline of evolutionary psychiatry. It is concerned with strategy sets, which are sets of alternative strategies for dealing with problems. For instance, the cold weather and reduced food supply of winter present a problem to many species. Sometimes all members of a species deal with the problem in the same way, but sometimes there are alternative strategies for dealing with the problem. Many bird species migrate; in some of these all the individuals migrate, in other species only a proportion migrate and the rest stay where they are. In very cold winters the rewards of migrating are greater than staying, but in mild winters the rewards of staying are greater. It is not of great concern to behavioural ecologists just how the decision, to stay or migrate, is made. It could be entirely genetic, so that a "staying" allele (or group of alleles) is competing with a "migrating" allele. Or it could be entirely environmental; for instance, it is thought that robins compete for territories in the autumn, and those birds who win territories stay and those who fail to win territories migrate. Probably for most partially migrating species the decision-making mechanism is not known. Similar considerations may apply to partially hibernating species of rodents; the territory owners stay awake, and those who do not have territories go to sleep.

Coming closer to evolutionary psychiatry, let us consider the case of pairwise contests. A rival for mates or other resources poses a problem for the individuals of most species, and various strategies have evolved to deal with it (Boone 1992; Crowley 2003). Most species have evolved the alternative strategies of escalation and de-escalation. In territorial species, you either fight or run away. In group living species, there is an alternative – appeasement – which enables one to continue living in the group, albeit at a lower social rank. Each strategy has costs and benefits. Behavioural ecologists such as Maynard Smith have studied the conditions under which alternative strategies could survive (Maynard Smith 1982; Parker 1984; Reichert 1998). Calling the escalating strategy the "hawk" strategy and the de-escalating strategy the "dove" strategy, they concluded that under certain conditions a mixture of hawk and dove is an *evolutionarily stable strategy* (ESS) in that it cannot be infiltrated and replaced by any other strategy, and in particular it cannot be replaced by a pure hawk or a pure dove strategy. Thus, variation in fighting behaviour is an ESS. The variation is maintained by negative frequency-dependent selection, because in a world of hawks it pays to be a dove, and in a world of doves it pays to be a hawk. It does not matter whether the choice between hawk and dove is genetically or environmentally determined. Nor does it matter whether the choice is a "once and for all" affair, such that type of parenting or some other variable made an individual hawk or dove, or whether each individual has the capacity to deploy both hawk and dove strategies, the choice depending perhaps on environmental cues or possibly on a random basis.

Both hawk and dove strategies have costs and benefits. We think on the whole that the costs of being a hawk tend to take the individual to the casualty department, whereas the costs of being a dove take him or her to the psychiatric clinic (Price et al. 2004). In other words, the costs of being a dove represent some of the "mental disorder susceptibility alleles" of K&M. And, since the choice between hawk and dove can be either environmental or genetic, the problem of the partial heritability of the mental disorders does not affect the argument.

Another issue is dispersal. Many species have both a maintenance phenotype, which is adapted to the natal territory, and a dispersal phenotype, which is adapted to occupying new habitats (Geist 1989). As each phenotype becomes rarer, its fitness increases, so both are maintained by negative frequency-dependent selection. During hominid evolution, rapid dispersal must have been advantageous, as receding ice sheets left new land available for occupation. However, human groups tend to be united by a common belief system which differs from the belief system of all the groups they are competing with. In order to facilitate dispersal, it may have been advantageous for an individual to undergo a change of belief system and to convert some of the group to the new belief system, and to take them off to a "promised land" (Price & Stevens 1999; Stevens & Price 2000b). Thus, two dispersal phenotypes may have evolved: One is the schizotype who has the capacity to undergo a change of belief system, and one is the suggestible or dissociative person who has the capacity to be converted from the belief system with which he or she was indoctrinated during childhood and to adopt the new belief system of a prophet or cult leader. The fitness costs of both these dispersal phenotypes would be grievous if dispersal was unsuccessful, but the benefits of successful dispersal might also be very great, leading to an adaptive radiation in a new habitat. In 45 years of psychiatric practice, I have seen many patients labelled schizophrenic who in different circumstances might have become effective cult leaders

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Bipolar disorder evolved as an adaptation to severe climate

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Abstract: Keller & Miller (K&M) assert that mental disorders could not have evolved as adaptations, but they fail to make their case against the theory of the evolutionary origin of bipolar disorder that I have proposed (Sherman 2001). Such an idea may be unorthodox, but it has considerable explanatory power and heuristic value.

In a previous publication (Sherman 2001), I proposed the theory that circular bipolar disorder (major depression with hypomania or mania) evolved as an adaptation to long severe winters and short summers, which implicates the circadian clock in its pathophysiology. This idea is inferred from theorizing and data about the seasonal effects of light and the small, but statistically significant, correlation between bipolar disorder and a pyknic, cold-adapted build. The hypothesis is consistent with recent research: Light therapy is as effective as antidepressant medications for seasonal depression, and three studies have demonstrated similar effectiveness for nonseasonal, major depression (Golden et al. 2005; Rosenthal 2006). Individuals with seasonal affective disorder, compared with healthy volunteers, generate a biological signal of change of season that is similar to the signal that mammals use to regulate seasonal changes in their behavior (Wehr et al. 2001). Other research genetically implicates the circadian clock in the pathophysiology of bipolar disorder (Yin et al. 2006).

I have suggested that circular bipolar disorder evolved among a small homogeneous population who lived in the northern temperate zone of the Old World during the ice ages. Bipolar