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

Julia A. Sherman

*6302 Mineral Pt. Road, #303, Madison, WI 53705.*

**shermanja@earthlink.net**

**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



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depressive behaviors include lethargy, social withdrawal, and loss of interest in food and sex. These behaviors conserved energy and reduced social conflict within small groups holed up together for the long, dark winter. Symptomatic cognitive impairment makes sense in this context: winter was a time to endure, not a time for thinking or action. During the short season of fair weather, the active, outgoing, goal-oriented behaviors of hypomania helped the group make up for lost time, accomplishing tasks necessary for survival. Hypomania is a positive mood and energy state associated with productive achievement, and bipolar disorder is correlated with intelligence and upper socioeconomic level (Goodwin & Jamison 1990), factors which Keller & Miller (K&M) failed to mention. In addition to the 1% of the population with severe symptoms, the authors' major focus, another 5% show milder forms of bipolar disorder with more positive associated behaviors (Jamison 2005).

Women in the ancestral bipolar disorder group may have had a reproductive advantage because a certain level of body fat is essential for ovulation, successful pregnancy, and breast-feeding. Reviews of the literature (Anastasi & Foley 1949; Eysenck 1947, 1953; Sherman 2001) agree with Kretschmer's (1970) early-twentieth-century observation of a relationship between circular bipolar disorder and a compact, pyknic build with cold-adapted fat accumulation (also see Eiben et al. 1980; Tóth et al. 2002). Women's activity level and eating behavior were adapted to their reproductive status, and eating heartily during fair weather to store fat for winter is mirrored in what is now called summer depression. Depression is at least twice as common among women, and recent data show that it may be more heritable among women (Kendler et al. 2006).

In contrast, mania, which is more frequent and intense among males, may have evolved as a response to emergencies. During mania, individuals have a superhuman ability to go without felt need for sleep or food. They think fast and move fast, which are characteristics that aid individual and group survival. Impulsive, frequent sexual activity is also common during mania, but the authors fail to consider the possible distortion of data because of children born out of wedlock. In any case, bipolar fitness is much higher than the level of fitness associated with schizophrenia.

Skepticism that circular bipolar disorder evolved as an adaptation is understandable when it is taken out of context and considered in its most extreme form. Psychosis is only sometimes associated with bipolar disorder, and the minority who experience a psychotic episode usually recover completely from the episode. In contrast, a schizophrenic diagnosis implies psychosis, and about one-third never recover. Another challenge to circular bipolar disorder as an adaptation is the current high rate of suicide associated with the diagnosis. However, the rate of suicide now may not be relevant to a time when individuals struggled merely to live and when social conditions were more favorable, in the sense that, because their behaviors were modal, individuals with bipolar disorder were not stigmatized, victimized, or forced to function at a high level during winter months.

The authors assert that bipolar disorder could not have evolved as an adaptation because it increases with negative factors, such as paternal age (no evidence cited), brain injury, and inbreeding. Adaptations, however, are necessarily positive only for the selective environmental pressures that produce their evolution. Although the authors discuss genes by environment interaction, they fail to explore adequately the concept in this case. Space constraints prevent further discussion of other problems with this argument.

K&M emphasize overlap among mental illnesses, but their argument that bipolar disorder is correlated with depression is moot. Many people with a diagnosis of major depression later have a hypomanic or manic episode and are then correctly reclassified. In regard to the genetic overlap of schizophrenia and psychosis associated with mania, the question is unresolved. The phenotypic behavior of the two states is similar, but correct differential diagnosis *is* crucial for the patient's effective treatment.

When asked why bipolar disorder genes survive in the gene pool, James Watson replied, "Survival might often depend on not if we think two and two is four, but on being slightly wild. . . . I think when we do science we see that a little madness does help, and you propose bizarre things which everyone says can't be true" (Jamison 2005, p. 275). Theory is meant to generate new ideas, and the theory I have proposed about the evolutionary origin of bipolar disorder generates many new ideas. For example: Could public health measures that promote timely exposure to more light lessen the incidence of depression in children and adults? Does exposure to bright light during nighttime destabilize the circadian clock and stimulate pathophysiology among those with bipolar disorder susceptibility alleles? Would daytime bright light and activity combined with nighttime dark and sleep help stabilize those with bipolar disorder?

## Adaptationism and medicalization: The Scylla and Charybdis of Darwinian psychiatry

Alfonso Troisi

*Department of Neuroscience, University of Rome Tor Vergata, Rome 00161, Italy.*

**alfonso.troisi@uniroma2.it**

**Abstract:** The target article shows that the application of the evolutionary theory to psychopathology should not necessarily consist in finding hidden adaptive benefits for each psychiatric syndrome. However, in rejecting lax adaptationism, Darwinian psychiatrists should not forget that the search for adaptive behavioral polymorphisms can be a powerful antidote against the normative attitude of mainstream psychiatry and its growing tendency to medicalize human diversity.

For evolutionary psychiatrists, the existence of psychopathology is perplexing. How can Darwin's theory of natural selection explain the persistence of psychopathology that places individuals at a reproductive disadvantage? In order to resolve the paradox of common and heritable mental disorders, most evolutionary psychiatrists have endorsed the adaptationist program. In evolutionary biology, the adaptationist program is a research strategy that seeks to identify adaptations and the specific selective forces that drove their evolution in past environments. Although everyone agrees that organisms have adaptations, adaptationism as a research strategy has not enjoyed consensual affection within evolutionary biology (Andrews et al. 2002). In the 1970s, it became the target of attacks by paleontologist Stephen Jay Gould and geneticist Richard Lewontin, who argued that adaptive explanations given for most human behavioral and cognitive traits were analogous to Rudyard Kipling's "just-so" stories. In effect, adaptive stories are easy to create and hard to falsify, and the risk of using inappropriate or insufficient standards of evidence for identifying adaptations and their functions is always present in the evolutionary study of human behavior. Such a risk is even higher in Darwinian psychiatry, where it is becoming more and more popular to pick a disorder out from current nosologies (which are of dubious validity) and to invent an adaptive explanation for its existence. Evolutionary psychiatrists have been certainly inventive in their search for a hidden genetic advantage to mental disorders. Genes for schizophrenia might have made certain individuals more charismatic and shamanistic. Distractible, risk-taking individuals who are currently diagnosed as having attention deficit/hyperactivity disorder (ADHD) might have had a competitive advantage in dangerous environments where survival would depend on being response ready. Anorexia nervosa might have evolved as a strategy for conserving reproductive resources in environments in which males are perceived as scarce and female competition for males is perceived to be intense. The unfortunate result of



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