association with urban upbringing (Pedersen & Mortensen 2001) – which implies instability of links with social groups – attest to the importance of intraspecific aggression and our fear thereof in schizophrenia; as do hallucinations of *other people's* voices during psychosis, which in their content are often critical and derogative (Birchwood et al. 2000; Linn 1977), reflecting patients' fears of rejection and marginalisation.

Evolutionary psychiatry is dead – Long liveth evolutionary psychopathology

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Abstract: Keller & Miller (K&M) propose that many psychiatric disorders are best explained in terms of a genetic watershed model. This view challenges traditional evolutionary accounts of psychiatric disorders, many of which have tried to argue in support of a presumed balanced polymorphism, implying some hidden adaptive advantage of the alleles predisposing people to psychiatric disorders. Does this mean that evolutionary ideas are no longer viable to explain psychiatric disorders? The answer is *no*. However, K&M's critical evaluation supports the view that psychiatric disorders are not categorically distinct from normalcy, and that evolutionary psychopathology should be grounded in rigorous empirical testing.

Keller & Miller's (K&M's) proposal of how to best conceptualize the genetic underpinnings of psychiatric disorders will certainly induce an uproar among those evolutionary psychiatrists who believe that psychiatric disorders reflect adaptations to selectively relevant problems in the human evolutionary history. Adaptive properties have been assigned to a great diversity of psychiatric problems, such as anxiety disorders, depression, and even vaguely defined disorders such as schizophrenia, either on account of their mere prevalence in the population or due to a presumed function of susceptibility alleles in heterogeneous carriers (for a critique, cf. Dubrovsky 2002). In contrast, K&M's suggestion that many psychiatric disorders emerge if the number of "unfavourable" alleles is sufficiently great in an individual, thereby rendering this individual vulnerable to environmental stress, challenges the adaptationist perspective on psychiatric disorders, because it would seem that the search for genes predisposing people to psychiatric disorders and for a hidden adaptive advantage of such genes could be a fruitless endeavour.

Does K&M's account discredit an evolutionary approach to psychopathology altogether? My answer is no. However, K&M's thoughtful review of possible genetic models of psychiatric disorders unveils several weaknesses of the adaptationist perspective in psychiatry.

First, the adaptationist perspective holds that some psychiatric disorders, while perhaps being maladaptive themselves, convey adaptive advantages in first-degree relatives of the affected individuals outweighing the reproductive disadvantage of the symptom carriers, similar to the oft-cited example of sickle-cell anaemia. Such a hypothesis could, however, be empirically tested only if such disorders were well defined at the phenomenological, neurophysiological, and genetic levels (sickle-cell anaemia is monogenetically transmitted). This is not the case for almost all psychiatric disorders, as there is a broad overlap between psychiatric disorders at all levels. For example, abundant research now points to the fact that schizophrenia and bipolar affective disorder share several clinical features (e.g., thought disorders) and neurophysiological characteristics (e.g., elevated dopamine levels), and run in the same families (i.e., genetic susceptibility).

Second, some evolutionary psychiatrists assert that psychiatric disorders themselves were adaptive responses to adverse environmental conditions. For instance, depression could reduce social stress by causing a person to assume a subordinate behavioural strategy instead of fighting for resources, if the chance of succeeding in contest is perceived to be low. This view is much more delicate, because it is plausible for minor forms of reactive depression, for example, after loss of social status or after divorce. It becomes, however, much more difficult to explain severe, melancholic, or "endogenous" depression, which, rather than being adaptive, appears to be similar to what ethologists call *vacuum behaviour* (behaviour occurring without appropriate stimulus). This example may illustrate the difficulty in drawing a line between an adaptive "second-best" strategy and maladaptive behaviour, based on severity of the symptomatology or the presence or absence of stimuli (Nettle 2004). In any event, it needs to be emphasised that sadness is not depression, and that suspiciousness does not equal delusional thinking, although in both examples the former may develop into the latter. The problem of continuity between normalcy and pathology has not yet been acknowledged by many evolutionary psychiatrists, perhaps because the majority of them still think in categorical dimensions.

Ironically, K&M's review of the possible genetic underpinnings of psychiatric disorders from an evolutionary perspective strengthens the view that environmental factors are perhaps at least equally important for explaining psychopathology in evolutionary terms (this is not to say that genetic variation does not play a role in psychopathology). If it is true that every one of us carries several hundreds of "unfavourable" alleles rendering all of us more or less susceptible to developing psychiatric disorders, it would be much more advisable to carefully investigate potentially unfavourable environmental conditions eliciting psychopathology. For example, Belsky et al. (1990) have proposed an intriguing model of how behaviour rendered "deviant" in modern environments may emerge if infants grow up under adverse conditions, including harsh parental rearing styles, lack of sufficient resources, and so on. The model basically suggests that, under favourable circumstances, parents could afford to invest heavily in their offspring, hence promoting in children the development of an *inner working model* of trustworthiness; whereas it might also have been adaptive for offspring in adverse ancestral conditions to assume a view that the world is an insecure and unpredictable place, where (in terms of reproductive fitness) it was advisable to focus on immediate resource extraction, including the exploitation of others. From a psychopathological perspective, the extremes of the latter "strategy" (no conscious reflection implied) may fall into the categories of antisocial personality disorder (ASPD) or borderline personality disorder (BPD), both of which can be seen as pathological (and therefore maladaptive) exaggerations of an adaptive mechanism.

Another strength of evolutionary psychopathology is certainly to help clarify sex differences in behaviour. With respect to the foregoing example, ASPD is much more common in males, whereas BPD is more often diagnosed in females, although the precipitating condition may be analogous or even identical. This might reflect sex-dependent competitiveness in different social arenas. Similarly, traditional psychiatry has been unable to explain sex differences in the content of delusional disorders, where evolutionary psychopathology can offer testable hypotheses about why erotomania reflects the pathological exaggeration of a female mating strategy, whereas delusional jealousy reflects the pathology of a male mating strategy (Brüne 2003). There is no psychiatric framework other than evolutionary psychopathology to explain such differences between the sexes (Brüne 2002).

In summary, evolutionary psychopathology (as a field) would do better if it chose a more symptom-based or syndromal approach rather than clinging to nosological categories that have proven more and more outdated – except for the purpose of bringing evolutionary issues of psychopathology to a broader audience of psychiatrists and clinical psychologists. This is one important message from K&M's thought-provoking thesis.

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