Commentary

Schizophrenia as one extreme of a sexually selected fitness indicator

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The current state of scientific and philosophical orientation for most biomedical theory and practice is not adequate to even ask, much less answer, deep questions of phylogenetic etiology (Wilson, 1993). Much biomedical science lacks a coherence to be adduced from its mongering of study design, outcome curves and other enthusiasms of the currently reigning, largely inductive science. While it is perhaps better to make a minor inductive advance than risk a crashing deductive error, theoretical inference, when robust, can reframe quantitative data or, at least, identify crisp qualitative incongruencies (Herrick, 1915, 1936) with insights, both counterintuitive and non-obvious, that would never be appreciated by mere experiment, no matter how clever.

These problems are only magnified in psychiatric research by its lack of both a foundational theory and cogently integrated intellectual aims. In certain respects, psychiatry has become a pastiche of arid facts, abstruse ideological claims and grand speculations amid a surfeit of plausible but contradictory explanations that lack an encompassing framework. It is particularly difficult to sort out historical questions (such as the past function of phenotypes and the selection of their genes in the environment of their evolution) since experimental observation is constrained, largely, to modern environmental circumstances. Of course, empirico-experimental research is welcome, but quite daunting given the scale of issues and ethics involved. Meanwhile, psychopathologists have long had available one such framework beginning with Darwin’s elegantly reasoned theory of evolution.

However, no application has yielded a clear, necessary and complete Darwinian explanation for the fact that genes linked to psychopathology, including the schizophrenia-spectrum phenotypies, appear beyond what probable mutation rates and neutral selection might maintain. It is therefore not merely speculative to consider schizophrenia may be associated with evolutionarily adaptive features—past or present—but an actual imperative from the point of view of population genetics. Among the many salient applications of evolutionary science, it is essential to a full understanding of epigenetic conditions, especially those with broad and/or high genomic frequency. That notably includes schizophrenia for which classical (pre-genomic/recombinant) epidemiology long ago established it as clearly epigenetic (McGuffin et al., 1995; Kendler and Diehl, 1993; Kety et al., 1994; Tienari et al., 1994), familial (Gottesman, 1972; Kendler and Diehl, 1993), and rather highly heritable (Matthysse and Kidd, 1981; McKusick, 1990; Tsuang et al., 1999).

Thus, Shaner et al. (in press) present extant data in a newly synthesized manner with some plausibility as to evolutionary heuristics of schizophrenia. As such, this is a laudable effort that should be of interest to readers of Schizophrenia Research and, indeed, broader audience insofar as it formulates a useful—
though by no means completely persuasive—hypothesis that schizophrenia is one extreme of a sexually selected fitness indicator. This helpful contribution comes at a time when evolutionary concepts are, at long last, making their way into the mainstream of psychiatric research—some brief contextualization of which may be helpful here.

One useful view is to consider evolutionary analyses of mental illness as either completely disadvantageous by-products of human brain evolution or that certain associated phenotypies conferred evolutionary advantage (Polimeni and Reiss, 2003). For example, Farley (1976) and Crow (1990, 1991, 1993, 1995, 1997) are among those who argue schizophrenia is an extreme variant of normal social behavior but without intrinsic Darwinian advantage. However interesting, Crow’s ideas suffer from a variety of inventive, even dubious assertions; i.e., that schizophrenia is (1) inherently linked in phylogeny to language dysfunctions that (2) specifically evolved via punctuated equilibria along (3) a single continuum of psychosis that wholly rejects any Kraepelinean taxonomic distinction for mood and psychotic disorders. Such a formulation seems little shaped by rigorous application of evolutionary genetic theory or analysis.

For his Darwinian interpretation of schizophrenia, Millar (1987) leverages that massive body of evolutionary neuroethology research that MacLean (1990) organized under the rubric of the triune brain. Here, schizophrenia derives from failed neuromental integration across the reptilian midbrain, paleo-mammalian limbic and neo-mammalian cortex. Again though plausible and broadly applicable to diverse neuromental anomalies, this approach lacks either specificity or truly robust evolutionary genetic analysis. Though not explicitly evolutionary, the cognitive dysmetria hypothesis (Andreasen et al., 1998) is quite convergent with Millar’s framework. So too, Yeo et al. (1999) model of schizophrenia as a developmental instability, though theoretical, converges with mainstream research of schizophrenia as a syndrome of composite causal factors in which moderate genetic vulnerability gives rise to diverse phenotypic expressions due to variable neurodevelopmental events. Such convergences are an important means to both enrich evolutionary models while integrating empirical findings.

However, evolutionary science is particularly tantalizing as it may cast light on how genes associated with psychopathology, including schizophrenia, were selected due to associated Darwinian advantages. But all too often, such efforts have little more scientific substance than Kipling’s ‘just-so stories’. Yet, there are scientific ways forward, particularly since evolution is itself an inherently epidemiological process. One place to begin is with the sure Darwinian deduction that common, genetically simple pathologic syndromes are likely the residua of natural selection of features that were advantageous in phylogeny. Such lineages were adaptive at least before the rise of civilization since spontaneous maladaptations cannot surpass thresholds of surprisingly low prevalence. This seems especially true in the realm of disorders that arose phylogenetically from genes expressing a variety of neurobiological sensitivities and talents. The modern environment may sometimes induce newer and less adaptive phenotypes among individuals rendered susceptible by phylogenetic quirks (Nesse and Williams, 1996).

Thus, evolutionary epidemiology is emerging as a biomathematical discipline that translates prevalence rates, as known to epidemiologists, into frequency rates, as known to geneticists (Wilson, 1992, 1993, 1998). Indeed, evolutionary epidemiological analysis is necessary to determine which gene systems have population genomic prevalence so high as to have needed a long period of advantageous natural selection in the past environment of evolutionary adaptation. It also sets the stage to proceed with Mayr’s evolutionary analytic algorithm which holds that one first must determine if a gene system was selected before then addressing how, when, where and/or why such selection occurred (with or without subsequent ‘mismatch’ problems arising in the modern environment).

For example, Huxley et al. (1964) first proposed that schizophrenia must be, in part, a heuristic evolutionary adaptation. Wilson (1998) describes this as the first published work in the evolutionary-psychiatric epidemiology as they undertook to explain the high prevalence of schizophrenia despite its disease burden and low fecundity via calculations as a genetic population polymorphism. Subsequently, investigations concerning possible adaptive advantages associated with the schizophrenic genotype have
accumulated, first gradually if more quickly in the past few years. Explanations range from one to another physiological advantage (Carter and Watts, 1971; Erlenmeyer-Kimling, 1968; Horrobin, 1998, 1999; Erlenmeyer-Kimling and Paradowski, 1966), creative intelligence (Karlsson, 1970), effects in social interaction (Kuttner et al., 1967; Kellet, 1973); polygenic influences on social skills and personality traits (Farley, 1976, 1992; Jarvik and Deckard, 1977); and/or mixed physio-social aspects (Allen and Sarich, 1988); and the effects of kinship selection and group dynamics (Stevens and Price, 2000; Polimeni and Reiss, 2002, 2003). In one way or another, these papers try to advance the ideas of Huxley et al. (1964) and inform Tsuang and Faraone’s tentative grasp of the emerging field of evolutionary epidemiology as it relates to the conventional interests of medical-psychiatric genetics.

To this body of work is now added the present paper by Shaner et al., which is not without problems. First of all, the introduction of a psychologically specific new concept of the ‘sexual selection fitness indicator (SSFI)’ is more of a piece with evolutionary psychology and its rather Cartesian ‘mental modules’ than it is steeped in clinical, much less biomedical science. Moreover, it is not always clear in operational terms how this putative mechanism relates to the similarly putative range of courtship behavior that the authors propose is linked (rather tenuously) to a range of phenotypic expression from phenomena as formal as clinical schizophrenia to the fungibilities of social suaveness.

Likewise, the ‘null hypothesis’ is not necessarily rejected by the highly theoretic approach used—even if there is an ‘SSFI’, schizophrenia may yet be (probably is) a syndrome of failed phenotypy with marginal evolutionary interest. The authors do attempt something of a formal evolutionary epidemiological analysis to first address Mayr’s question of ‘whether’ a trait was likely selected before advancing explanations of how, when, where, why and how may heuristics have arisen (Mayr, 1963). Enthusiastic evolutionary psychologists and psychopathologists routinely fail to take the first step first, but instead propound phenomenological explanations before establishing that there is an actual phenomenon! However, it is by no means clear that schizophrenia actually is an evolutionary paradox—it may well be evolutionary—developmental ‘noise’ of the machinery of selective population genetics. Still, even a disaffirmation that schizophrenia ala’ DSM is itself adaptive would constitute a useful contribution and, curiously, unrelated to confirmation or denial of evolutionary advantages perhaps associated with other ‘sub-clinical’ schizophrenoid phenotypies along a spectrum of neurodevelopmental epigenetic expression.

It remains to be seen if the authors’ introduction of a new, non-operationalized, unvalidated and possibly extraneous ancillary mechanism by way of explanation for an adaptive basis for schizophrenia will become substantiated over time or simply be a transient notion. However, the focus on sexual selection as the sole, or even principal, mechanism runs counter to the more usual matrix of selective factors that drive most evolutionary processes, particularly those that, like schizophrenia, involve complex neuromental apparatus and operations within and between conspecific organisms.

Physical anthropologists long ago argued, sometimes bitterly, whether hominoid bipedal stature was due to a need for hand use, to peer over savanna grasses, to display dominance or other driving factors. Now it is more commonly agreed that such evolutionary adaptation arises from manifold and interacting selective forces. Hence, the authors and, indeed, everyone interested in evolutionary research in neuroscience and psychopathology would do well to curb the understandable enthusiasm for singular explanations and instead focus on more robust and integrative perspectives.

Yet on the other hand, it also should be recalled that sexual selection—Darwin’s second major mode of evolution—was largely dormant for a century and has only in the past decade or so been revived among evolutionists and anthropologists as a genuine, major force in population genetics. Hence, the authors do much more than merely raise some interesting if speculative proto-theses, but have begun to develop another evolutionary scenario in the long, tangled effort to salvage something of Darwinian value in this otherwise quite tragic illness. Whatever the limitations of this current paper, the Darwinian approach has broad scope and value of increasing interest and relevance to every aspect of psychopathology, including schizophrenia research.
References


