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Evolutionary Epidemiology of Endophenotypes in the Bipolar Spectrum: Evolved Neuropsychologic Mechanisms of Social Rank

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Medicine—until quite recently—has disregarded Darwin. However, novel ethologic perspectives of evolutionary epidemiology are beginning to shed light on both proximal and ultimate causes of human behavior in its boundaries with adaptive endophenotypes, disease and therapeutics. Here we summarize how a Darwinian framework informs new insights concerning the spectrum from mania to depression.

Introduction

Niko Tinbergen—Dutch ethologist, Oxford professor, and Nobel laureate—said behavior requires four types of explanation [1]. Most usual are proximal explanations: "how" epigenetic pathways operate from gene to behavior via neuroanatomy, neurophysiology, and psychology. As the molecular genetics of bipolar disorder encounters numerous barriers, the search is now for "endophenotypes," recognizable, genetically distinct manifestations identifiable in relatives of probands [2–4]. Such endophenotypes include impairments of cognitive function [5,6], cyclothymia [7], dysmodulation of motivation and reward, and eye movement abnormalities [8]. Tinbergen [1] also required ultimate explanations: "why" or explaining how natural and sexual selection designed "evolved psychological mechanisms" that underlie all adaptive behavior, including attachment, reciprocal exchange, cheater detection, predator avoidance and social rank competition [9]. A third explanation is ontological: "what" trait development is affected by life experience. The final Tinbergian explanation is phylogenetic: "when" or delineating traits through prehistory. Bipolar psychopathology is a malfunction of these evolved systems with as yet unidentified endophenotypes [10••].

Recognition that some psychopathologies are remnants of past adaptive behaviors is so new that little of this evolutionary neuroscience is yet systematized. Several useful forays stand out: Darwin [11]; Romanes [12]; Lloyd-Morgan [13]; Huxley et al. [14]; Price [15]; Gardner [16]; Wenegrat [17]; Bailey [18]; Chance [19]; Gilbert [20]; Glantz and Pearce [21]; MacLean [22]; Wilson [23]; Williams and Nesse [24]; Stevens and Price [25]; McGuire and Troisi [26]; and Sherman [27].

Meanwhile, psychiatry is currently looking for endophenotypes underlying psychiatric conditions. It is useful to recall that MacLean [22] showed the basis for sociality is to be found in evolved psychological mechanisms. These are expressed via two opposing archetypal neuromental circuitries at three levels [22]; malfunction in any aspect of which leads to disorder. MacLean [22] identified the first level as reptilian (R)-complex, insofar as brain stem, midbrain, and part of forebrain became fully instantiated in ancestral reptilians 300 millions years ago. The second level abuts R-complex as an assemblage of transitional mammals about 250 million years ago. This Paleomammalian limbic system facilitates nursing of infants, parent-infant bonding, continuously interactive "warm-blooded" social life and the emergence of play. The third, a Neomammalian complex, from some 60 million years ago extends increasingly sophisticated domain-specific functions such as language, abstract thought, for greatly enhanced reciprocally interactive social life, including self-consciousness.