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Research Article

Mood Disorders from an Ethological Perspective: Evolutionary Psychopathology as the Basis for an Anthropological Psychiatry

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Abstract

Background: Evolutionary psychiatry posits that many mental disorders derive from the dysregulation of originally adaptive mechanisms, selected to manage threats, resources, hierarchies, and social cohesion. In this framework, depression and mania are understood as alterations of complex affective and socio-evaluative systems, shaped by human phylogenetic history and currently subjected to mismatched environmental and sociocultural conditions.

Objective: To integrate the main evolutionary, ethological, and sociobiological models applied to mood disorders, articulating them with the Innate Precipitating Mechanism (IPM) Model and contemporary sociocultural factors, especially those characteristic of postmodernity, to establish a foundation for a comparative anthropological psychiatry.

Methods: Narrative review of specialized literature in evolutionary psychiatry, affective neuroscience, ethology, sociobiology, biocultural anthropology, and comparative studies in humans and non-human primates. Relevant theoretical and empirical works on emotions, hierarchical regulation, social cognition, socioecological variability, and affective psychopathology were included.

Results: Emotions function as adaptive systems designed to coordinate physiological, cognitive, and behavioral responses to social and environmental challenges. Mood disorders emerge when these systems are activated under conditions that differ from the ancestral niche, generating mismatches in energetic, hierarchical, and socio-evaluative regulation. Price's hierarchical theory, Baron-Cohen's social cognition models, and the IPM allow for the reframing of depression and mania as functional failures of an integrated IPM-A/IPM-AA system. Cross-cultural and primatological evidence questions the universality of patterns described in WEIRD populations and underscores the impact of the postmodern context, characterized by structural uncertainty, hyper-responsibility, and self-demand.

Conclusions: Evolutionary psychiatry offers an integrative framework to reinterpret the clinical heterogeneity of mood disorders, identify subtypes with distinct evolutionary bases, and guide interventions tailored to the human emotional architecture. This approach allows for linking light rhythms, hierarchical dynamics, social support, inflammation, and social cognition with differentiated depressive and manic profiles.

INTRODUCTION

Evolutionary psychiatry is based on the premise that natural selection has configured the human emotional architecture to solve recurrent adaptive problems throughout phylogenetic history [1,2]. In this context, mental disorders are not conceived as simple "failures" of a neutral system, but as dysregulations of originally adaptive mechanisms operating outside the conditions for which they were designed [1,3,4].

Foundational works in Darwinian medicine and evolutionary psychiatry have highlighted that many psychopathological vulnerabilities stem from the interaction between selected mental mechanisms and modern environments radically different from ancestral ones [1,3-5]. Depression and bipolarity, in particular, can be understood as extreme or decontextualized expressions of emotional systems involved in regulating effort investment, social status, risk assessment, and bond maintenance [1,3].

From an evolutionary psychology perspective, the mind is viewed as a set of specialized mechanisms ("modules") designed to solve adaptive tasks such as cooperation, deception detection, mate selection, or hierarchy management [3,6]. Emotions, in this framework, are functional programs that coordinate physiology,

cognition, and behavior to solve problems associated with threat, loss, exclusion, or hierarchical conflict [2,3,6].

Evolutionary models typically group vulnerability to illness into six major sources: (1) mismatch between ancestral and current environments; (2) conflict between own and others' genetic interests; (3) infection and immune defense; (4) evolutionary constraints; (5) adaptive trade-offs; and (6) genetic variation maintained by balanced or contextual selection [1,5,7].

It follows that psychiatry, to comprehend human vulnerability to mood disorders, must integrate both proximate mechanisms (genetics, neurobiology, life experiences) and ultimate explanations (why these structures and processes were selected and the cost of their current mismatch) [1,3-5].

The objective of this manuscript is to integrate the theoretical developments of evolutionary psychiatry, ethology, sociobiology, and comparative anthropology to reinterpret affective disorders within the framework of the Innate Precipitating Mechanism (IPM) Model. It is argued that mood disorders result from the interaction between ancient systems of energetic regulation (IPM-A) and more recent social and cognitive mechanisms (IPM-AA), whose integration becomes especially vulnerable under the sociocultural conditions of postmodernity.

EMOTIONS AS EVOLUTIONARILY DESIGNED SYSTEMS

Emotions can be defined as coordinated neurobiological mechanisms that adjust physiology, motivation, and behavior in response to relevant adaptive problems[6]. Following Tinbergen's classic proposal [8], any functional trait, including an emotional state, must be analyzed at four levels: (1) proximate causal mechanisms (neurobiology, cognition); (2) ontogeny (individual development); (3) adaptive function (advantages in terms of survival and reproduction); and (4) phylogeny (evolutionary history).

Affective neuroscience has described subcortical circuits conserved across mammals that sustain basic systems such as seeking/reward, fear, rage, care, play, or separation panic [9]. These systems, organized around limbic and subcortical structures, provide the functional basis upon which complex human emotions are built.

In depression, alterations have been documented in systems involved in reward, motivation, stress response, and circadian rhythms, whose original function would have been to modulate effort investment, promote withdrawal in persistently adverse situations, and integrate environmental signals such as resource availability or social loss [1,3,4,9]. Responses that were adaptive in ancestral niches—for example, abandoning a losing fight or reducing activity in contexts of scarcity—become pathological when activated in a prolonged, intense, or decontextualized manner in contemporary settings.

CONTRIBUTIONS OF ETHOLOGY AND SOCIOBIOLOGY

Classic ethology demonstrated the existence of Fixed Action Patterns and innate releasing mechanisms activated by specific environmental stimuli [7,10]. These findings help to understand how certain social stimuli (threatening gazes, signals of rejection, loss of allies) can precipitate intense affective states of anxiety, fear, submission, or withdrawal.

Sociobiology, based on Hamilton's inclusive fitness theory [11], and subsequent developments [12], highlighted that social organization, hierarchies, and cooperation among kin and non-kin deeply influence behavior and emotion. Concepts such as dominance, defeat, coalition, or reciprocal altruism allow linking phenomena observed in social animals with patterns of human affectivity.

In numerous primate species, social defeat is associated with immobility behaviors, reduced play, loss of appetite, and isolation—patterns reminiscent of depressive symptomatology [13,14]. These parallels inspired models in which depression is interpreted as a defeat and submission response, organizing group hierarchical dynamics.

JOHN PRICE'S HIERARCHICAL THEORY: DEPRESSION AS SUBMISSION

John Price formulated one of the most influential evolutionary models of depression, proposing that it can be understood as a submission behavior following a defeat in social competition [15-17]. According to this theory, many depressive episodes are triggered when the individual perceives a significant and irreversible loss of status.

In this framework, behavioral inhibition, psychomotor slowness, intense self-criticism, and social withdrawal would function to communicate the acceptance of the new rank, reduce the risk of attacks by competitors, and facilitate group stability [15-17]. Depression is thus integrated into a temporal pattern that would include phases of struggle, frustration, surrender, and eventual acceptance of the subordinate status.

This model accurately explains depressive episodes reactive to status losses (job failure, romantic rejection,

public humiliation) and aligns with comparative data in primates [13,14]. However, it has limitations in accounting for "causeless" depressions, recurrent episodes, or conditions not linked to clear defeats. This has motivated its incorporation into integrating models like the IPM and into evolutionary classifications that distinguish depressive subtypes based on mechanisms and functions [1,3-5].

SIMON BARON-COHEN AND SOCIAL COGNITION IN AFFECTIVE DISORDERS

Baron-Cohen proposed that social cognition is organized into specialized modules for gaze detection, emotion reading, intention attribution, and Theory of Mind (ToM) [18]. Although his work focused on Autism Spectrum Disorders, his concepts are essential for understanding human affective vulnerability.

Mood disorders involve profound alterations in social perception, self-evaluation, and the interpretation of others' intentions. In depression, for example, systematic biases towards negative interpretation of ambiguous signals, overestimation of rejection, and internalization of blame are observed [1,3]. These patterns can be understood as failures in modules designed to promote cooperation and attachment, which, under dysregulated conditions, orient toward chronic self-devaluation.

In mania, conversely, hyper-positive interpretations of social signals, overconfidence, a sense of invulnerability, and grandiose perceptions of one's own status are described. This suggests that the same socio-evaluative mechanisms that are biased towards defeat in depression can suffer a dysregulation in the opposite direction, generating states of overestimation and risk [3,4] .

The integration between Theory of Mind, social cognition, and emotional models is central to the IPM, which places these modules within the IPM-AA component, responsible for the advanced regulation of status, self-image, and goal valuation.

CONTEMPORARY EVOLUTIONARY MODELS OF DEPRESSION AND MANIA

Various evolutionary models have attempted to explain the adaptive function—complete or partial—of depression. None is sufficient on its own, but together they point to several possible benefits in specific ancestral contexts:

Withdrawal from Unattainable Goals: Some authors argue that depression facilitates the abandonment of chronically unattainable goals, reducing resource investment and promoting the reevaluation of alternatives [19].

Bargaining Model: Depression has been proposed as a costly signal intended to elicit support or renegotiate social conditions in contexts of conflict or exploitation [20].

Foraging and Effort Optimization Models: From this perspective, depression appears when the energetic or cognitive cost of maintaining a behavior outweighs the expected benefits, encouraging strategic withdrawal [21].

Attachment and Grief Models: Bowlby's work described the protest–despair–detachment sequence following the loss of attachment figures, which can be considered a precursor to evolutionary models of grief depression [11,22].

Circadian and Seasonal Models: Evidence on seasonal affective disorders supports the existence of a light-dependent mood regulation system, designed to adjust activity to seasonal changes [23].

Collectively, these models suggest that depression is not a single, homogeneous syndrome, but a family of affective states that share certain symptoms but respond to distinct mechanisms and functions [1,3,4]. A similar conclusion applies to manic states, which could be related to phases of exploration, risk-taking, status seeking, or response to social success signals, even if their current costs are very high.

THE INNATE PRECIPITATING MECHANISM (IPM) MODEL AS AN INTEGRATING SYNTHESIS

The Innate Precipitating Mechanism (IPM) Model proposes that affective disorders arise from the interaction between two functional systems:

IPM-A (Ancestral Component):

- Regulates basic processes such as sleep, energy, motivation, locomotor activity, and circadian rhythms.
- Strongly modulated by photoperiod and other environmental markers.
- Homologues observed across multiple species, indicating an ancient phylogenetic history [9,23,13,14].

IPM-AA (Advanced Component):

- Relies on cortical structures and complex social cognition networks.
- Regulates status perception, rejection sensitivity, self-evaluation, shame, guilt, and goal valuation.

- Specific to humans and dependent on the integration of symbolic cognition, language, and cultural norms [3,5,6,18].

According to the IPM, clinical depression emerges when the withdrawal system is activated excessively, prolonged, or decontextualized. Mania is interpreted as an inverse dysregulation, where ambiguous signals of success or status are amplified, generating an overestimation of capacity and environmental control [1,3,4].

Bipolarity would reflect an instability in the IPM-A/IPM-AA integration, modulated by genetic, photoperiodic, immunological, and cognitive factors. This instability explains the alternation between states of extreme withdrawal and overactivation, as well as its sensitivity to seemingly minor changes in social or environmental context.

The IPM thus offers a unifying framework capable of integrating:

- Seasonal depression and circadian rhythms (IPM-A predominance) [23].
- Depressions reactive to social defeat or hierarchical conflict (IPM-A/IPM-AA interaction) [7,15-17].
- Manic episodes linked to biased interpretations of social signals (IPM-AA) [3,4,18] .

REINTERPRETING DEPRESSION: MISMATCH, MODERNITY, AND EVOLUTIONARY SUBTYPES

Depression as a Disease of Modernity

Epidemiological evidence suggests that depressive episodes defined by DSM/ICD criteria are extraordinarily infrequent in hunter-gatherer societies and increase dose-dependently with modernization [5-7,24] . This rise has been linked to:

- Sedentary lifestyle and abrupt decrease in physical activity.
- Dysregulation of sleep and chronic exposure to artificial light.
- Availability of hypercaloric foods and patterns of low-grade inflammation.
- Relative social isolation and weakening of community networks.
- Chronic stress associated with job uncertainty, precarization, and role overload [7,5].

The mismatch hypothesis proposes that emotional and physiological systems designed for environments with natural rhythms, small groups, and dense community support are now exposed to conditions that exceed their optimal operating range [7]. The result is a generalized vulnerability to affective dysfunction, mediated, among other factors, by chronic inflammation, sustained activation of the HPA axis, and epigenetic changes that predispose individuals to interpret the environment as threatening even in the absence **of immediate dangers [1,4,5,7].**

Evolutionary Subtyping of Depression

Against this backdrop, evolutionary classifications have been proposed that distinguish depressive subtypes based on:

- The proximate mechanisms that activate the episode (HPA axis, inflammation, circadian rhythms, loss of social support, hierarchical conflict, etc.).
- The ultimate possible functions of the mood change (energy conservation, social renegotiation, signaling need, withdrawal from inviable goals) or its clearly maladaptive nature [1,3-5].

Among the subtypes are (summarized):

- Infection-Induced Depression: Related to sickness behavior mediated by pro-inflammatory cytokines; considered an adaptation to conserve energy and avoid contagion [1,3,4].
- Chronic Stress Depression: A typically maladaptive state in modern contexts, linked to prolonged immune system and HPA axis activation [1,5,7].
- Loneliness Depression: Social isolation activates hyper-vigilance and inflammation responses that signaled life risk in social animals, but become chronic in individualistic societies [1,5,7].
- Hierarchical Conflict Depression: Based on Price's theory; defeat induces symptoms that function as an honest signal of submission; linked to "atypical depression" phenotypes and high rejection sensitivity [15-17].
- Postpartum Depression: Interpreted as a signal of insufficient allomaternal support; low prevalence in contexts with ample community support [25,26].
- Seasonal Depression: Conceptualized as circadian mismatch [23].
- Substance-Induced Depression: Characterized by

symptom reversibility and mechanisms linked to neuroadaptation, dopaminergic, serotonergic, and, in many cases, inflammatory dysregulation [1,3].

The evolutionary subtyping suggests that treatment will only be fully effective when the constellation of evolutionary and physiological factors underlying the episode is identified (e.g., reducing inflammation in immuno-activated depressions, restoring circadian rhythms in seasonal depressions, increasing social support in loneliness depressions, etc.) [25,26].

COMPARATIVE PERSPECTIVE AND SOCIOECOLOGICAL VARIABILITY

A key limitation of contemporary psychology and psychiatry is their reliance on WEIRD populations (Western, Educated, Industrialized, Rich, Democratic)V [24]. Most data on the prevalence, course, and treatment response of affective disorders come from these contexts, raising doubts about their generalization [5,24].

From an evolutionary perspective, it is unlikely that the affective patterns observed in WEIRD populations represent the universal expression of human emotional systems. Intracultural differences, adaptation to diverse ecological niches, and phenotypic variation associated with gene-environment interactions have shaped the IPM components throughout phylogeny [4,5,7].

A paradigmatic example is postpartum depression. Cross-cultural studies indicate that its prevalence varies markedly between traditional and modern societies [25,26]. In contexts with extensive allomaternal support networks—closer to the ancestral environment—postpartum depression appears rare; conversely, in WEIRD populations, where the family structure is nuclearized and community support decreases, the burden of care falls almost exclusively on the mother, increasing the risk of IPM-AA dysfunction and depressive episodes [25,26].

Genomic evidence further suggests that some alleles associated with psychotic disorders are found in regions subjected to recent positive selection, linked to advanced cognitive functions [27]. This suggests that certain components of the IPM-AA—especially those related to social cognition, ToM, and symbolic processing—may have conferred adaptive advantages, even at the cost of greater psychopathological vulnerability in certain contexts.

Primatological data complement this view. The presence of behaviors analogous to depressive states following maternal loss in chimpanzees [13], or phenomena similar to somatization in macaques [11], indicates that the nuclear mechanisms of IPM-A are widely shared. However, the absence of complex syndromes equivalent to human affective disorders suggests that the IPM-AA components associated with symbolic self-evaluation and autobiographical narrative are unique to Homo sapiens [13,14].

This comparative perspective reinforces the idea that human affective psychopathology arises from the interaction between ancestral modules and emergent cognitive capabilities, whose integration is unstable in modern environments [5,13,14].

DISCUSSION: POSTMODERNITY, HYPER-RESPONSIBILITY, AND ANTHROPOLOGICAL CRISIS

The evolutionary approach provides a comprehensive framework to integrate different levels of analysis — neurobiological, psychological, social, and cultural— in the understanding of affective disorders [1,3-5]. In this context, postmodernity can be understood as a radically novel socioecological environment that exacerbates IPM vulnerabilities.

Following Han's philosophical-sociological analysis, postmodernity is characterized by:

- The establishment of structural uncertainty linked to the technical potential for global destruction.
- The dissolution of transcendent frameworks of meaning and stable collective narratives.
- The substitution of external disciplinary structures by a regime of self-demand and hyper-responsibility [28,29] .

In this framework, freedom is transformed into a mandate of performance, and the subject is forced to manage their well-being, productivity, and future under a logic of permanent optimization [28]. This hyper-responsibility generates a type of suffering expressed in pathologies of exhaustion, depression, and affective disorders linked to performance [28,29].

From the IPM perspective, these dynamics can be understood as chronic overloading of the IPM-AA:

- Increased constant comparative self-evaluation.
- Increased sensitivity to perceived failure.
- Blurring of boundaries between work and personal time.
- Internalization of structural responsibilities as individual responsibilities [5,28].

Simultaneously, changes in light rhythms (artificial illumination, night work, intensive screen use) impact the IPM-A, causing sleep, energy, and circadian rhythms dysregulation [5,7,23]. The convergence of both pressures—on IPM-A and IPM-AA—favors the appearance of depressive and manic conditions in individuals with genetic or biographical vulnerabilities.

Important limitations exist in the application of evolutionary models [1,4,5]: (1) the reconstruction of the ancestral environment is necessarily inferential; (2) there is a risk of attributing adaptive functions to traits that might be neutral byproducts; and (3) the clinical heterogeneity of affective disorders implies multiple causal pathways that cannot be reduced to a single explanation. Nonetheless, the evolutionary perspective—as noted by recent reviews in evolutionary psychiatry [3-5], provides a theoretical architecture that allows emotions to be situated in their natural context and helps understand why universal mechanisms can become dysfunctional under postmodern conditions.

CONCLUSIONS

Mood disorders can be conceptualized as dysfunctions of complex emotional systems designed by evolution to coordinate the individual's energetic and social adaptation [1,3-5]. The integration of contributions from ethology, sociobiology, evolutionary psychology, and affective neuroscience, along with models such as Price's hierarchical theory, Baron-Cohen's social cognition, and the IPM, allows for the reframing of depression and bipolarity as manifestations of a single regulatory system (IPM-A/IPM-AA) that oscillates between withdrawal and activation.

This framework:

- Reinterprets the clinical heterogeneity of depression as the expression of multiple subtypes with distinct evolutionary and physiological bases.
- Links light rhythms, inflammation, hierarchical structure, social support, and socio-evaluative cognition with differentiated affective profiles.
- Points out that many current vulnerabilities stem from the mismatch between mechanisms selected in ancestral environments and postmodern conditions characterized by hyper-responsibility, structural uncertainty, and the disruption of natural rhythms [7,5,28-30].

Evolutionary psychiatry does not seek to replace traditional biological or psychological explanations but to integrate them into a coherent framework that explains why we are predisposed to affective disorders and how this vulnerability derives from the evolutionary history of the human mind [1,3-5]. This approach also offers a conceptual basis for the development of more informed, specific, and culturally sensitive therapeutic interventions, in line with an anthropological and comparative psychiatry.

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