Evolutionary Psychiatry
Special Interest Group

RC PSYCH
ROYAL COLLEGE OF PSYCHIATRISTS

ANNUAL CONFERENCE

6TH NOVEMBER 2026

ROYAL COLLEGE OF PSYCHIATRISTS
LONDON

Evolutionary Psychiatry Special Interest Group (EPSIG)

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Notes from the Editor

As you already know by now this is our 10th anniversary year. Thanks to all those who kept the show on the road and ever expanding. **We had 3964 members at the end of April!**

We are succession planning for the future, so we welcome colleagues with particular skills and willingness to contribute to EPSIG activities. We have lost count of the very helpful colleagues who contribute to meetings, papers and the newsletter, Special thanks to the executive committee members and trainees who have given of their time so generously.

We also have a fabulous 10th anniversary conference booked for **November (6th)** at the RCPsych, with some of the biggest names in Evolutionary Psychiatry. Randolph Nesse is coming over from the States and Martin Brune from Germany to speak with us (see full program below).

We also have a book review by Dr Annie Swanepoel and some past competition essays!

EPSIG Ireland

The Evolution & Psychiatry Special Interest Group (EPSIG) in Ireland continues to grow, with Gurjot Brar recently appointed as Chair. Current efforts focus on a series of projects in medical education, alongside Henry O'Connell's ongoing delivery of evolutionary psychiatry tutorials to medical students throughout the year. Their meetings are well attended, free to attend online for all

UPCOMING EVENTS— WPA EP Section Webinar Program

In collaboration with the World Psychiatric Association

25 June 6pm, Prof Samir Okasha, University of Bristol

30 July 6pm, Jay Belsky, Emeritus Prof University of California, Davis

Details of titles and FREE registration links can be found on:
https://www.wpanet.org/section-page/evolutionary-psychiatry/

EPSIG Annual Conference Programme 2026

Time	Session
9.00am	**Registration**
9.30am	**Welcome and introduction** Dr Riadh Abed, EPSIG Chair
	Plenary 1
9.40am	**Chair: Paul St John-Smith**
	The allostatic cliff edge: An evolutionary mismatch theory of mental disorders Dr Paul Keedwell, Consultant Psychiatrist, Lewisham Low Intensity Support Team, UK
10.20am	**Hypercuriosity: An evolutionary theory of ADHD** Dr Anne-Laure Le Cunff, King's College London, UK
11.05am	**Q & A**
11.25am	**Refreshment break**
	Plenary 2
11.55am	**Chair: Paul St John-Smith**
	Why all psychiatry is not evolutionary psychiatry...yet! Professor Randolph Nesse, Emeritus Professor of Psychiatry, Arizona State University and University of Michigan
12.40pm	**Q & A**
12.50pm	**Charles Darwin Essay Prize winner presentation – Higher Resident:**
1.00pm	**Lunch**
	Plenary 3
2.00pm	**Chair: Annie Swanepoel**
	Charles Darwin Essay Prize winner presentation – Core Resident:
2.10pm	**The evolution of imperfection: the science of why we aren't and can't be perfect** Professor Laurence Hurst, University of Bath, UK
2.55pm	**Q & A**
3.05pm	**Refreshment break**
	Plenary 4
3.35pm	**Chair: Annie Swanepoel**
	Evolution of music: playing off the beat in psychotherapy Professor Martin Bruene. Ruhr University Bochum, Germany
4.20pm	**Q & A**
4.30pm	**Cowering in Caves? How the archaeological record can contribute to our understanding of modern anxiety** Professor Penny Spikins, University of York
5.15pm	**Q & A**
5.25pm	**Closing comments** Dr Riadh Abed, EPSIG Chair
5.35pm	**End of conference**

Charles Darwin RCPsych EPSIG Prize 2026

Do you want to be invited to speak at an international conference?

If you are a Psychiatry core or specialty trainee, medical student or non-consultant grade psychiatrist in the UK, you are eligible to enter this Essay Competition. Two winners will be invited to speak for 10 minutes each at the in-person EPSIG International Symposium on **6th November 2026** at the RCPsych in London.

The essay submissions must be the applicant's own intellectual contribution. Originality will be valued.

Medical students, foundation doctors and staff grade doctors are all eligible to apply in their own categories with the prizes as below:

What you can win:

Invitation to give a 10-min oral presentation at the EPSIG International conference held in person on **6th November 2026** for the psychiatry core and specialty trainee winners only.

- £100 pound prize money for the winner each of five categories (medical student, foundation doctor, core trainee, specialty trainee, staff-grade doctor)
- Certificate for the runner-up in each of the above five categories
- Winners will have their essay printed in the EPSIG Newsletter that is distributed to all EPSIG members (currently over 3000 psychiatrists and other professionals worldwide)

What you need to do:

Write a 1500 to 2000-word essay (excluding references) relevant to Evolutionary Psychiatry. The essay should be supported by a review of relevant literature and must be your own intellectual work.

The title is: ***"How an evolutionary perspective can improve our understanding of … (anything to do with psychiatry)"***.

You may want to visit the EPSIG YouTube channel at Youtube.com/EPSIGUK, listen to the 'Evolving Psychiatry' podcast (on all major platforms), or read our published newsletters at epsig.org for inspiration and information.

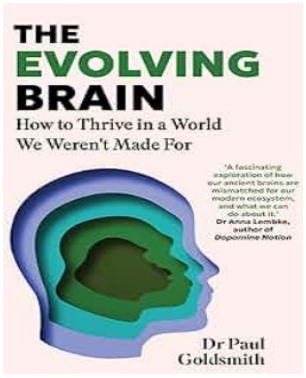
Email your submission to sigs@rcpsych.ac.uk with "Entry for EPSIG Essay prize" as title by 1st September 2026.

What we will do:

Entries will be judged by a panel of three EPSIG Executive Committee members. Criteria for judging will include clarity of expression, understanding of the evolutionary literature and evidence, critical thinking and the overall ability to convey enthusiasm and originality. The committee reserves the right not to award the prize if no entry reaching the agreed minimum standard is received. Winners and highly commended other entrants will be informed by 15th October 2026.

Book Review

The Evolving Brain: How to Thrive in a World We Weren't Made For, by Dr Paul Goldsmith



Those of us interested in evolutionary psychiatry will know the concept of evolutionary mismatch: the idea that many modern mental health conditions arise because our evolved psychology is poorly suited to contemporary environments. Work on ADHD, for instance, has drawn on the notion that traits which conferred advantage in a foraging, high-stimulation ancestral world can become liabilities in the classroom or the open-plan office. What has often been missing is a satisfying neurological account of how the mismatch actually produces the difficulties it does. Paul Goldsmith's *The Evolving Brain* fills that gap nicely.

Paul is an evolutionary neuroscientist and practising neurologist who draws on cutting-edge neuroscience and clinical case studies to build what he calls a user's guide to our ancient mind. For a psychiatric readership already sympathetic to evolutionary frameworks, this is the neurological substrate that our field has been waiting for. He shows, at the level of neural architecture and circuit function, how and why that mismatch generates suffering.

One chapter I found particularly interesting is "The Egoless Brain," where Paul looks at the neurological evidence that no discrete brain site hosts the self. The sense of a unified "I" turns out to be a functional construction rather than an anatomical fact. This resonates with what is discovered experientially in Vipassana meditation, where close attention to present-moment experience reveals the self to be fluid and constructed rather than fixed and given.

Equally valuable is Paul's treatment of the things we already advise our patients to do but sometimes struggle to explain. He gives clear neurological explanations for why physical exercise, social connection, and purposeful activity are not merely good habits but are, in the most literal sense, what our brains were built for. *Mens sana in corpore sano* turns out to have a solid neurological basis. When Paul explains why social isolation dysregulates threat-detection systems, or why sedentary behaviour conflicts with circuits shaped across millennia of movement, these everyday recommendations suddenly carry much more clinical weight.

The writing is clear and accessible throughout, and the case studies keep the neuroscience grounded. If you work in evolutionary psychiatry or are simply curious about why we struggle in the modern world, this book is well worth your time. It provides something genuinely useful: the neurological basis for how evolutionary explanations work in *Homo sapiens*.

Dr Annie Swanepoel

Previous Competition Essays

How an evolutionary perspective can improve our understanding of Tourette's syndrome

Shao Junzhe, Medical Student

Introduction

Tourette's syndrome (TS) is a neurodevelopmental disorder indicating the presence of motor or vocal tics, which are abrupt, repetitive and involuntary movements or sounds. The DSM-5 criteria requires that both multiple motor tics and one or more vocal tics should be present for more than a year, with its onset before the age of 18, to be diagnosed (American Psychiatric Association, 2013). Tics can manifest in a wide range of behaviours, from simple actions such as face grimacing, throat clearing and jerking movements, to more complex expressions like jumping, making animal noises, and repeating words or phrases—known as echolalia. In a smaller subset of patients, coprolalia (involuntarily uttering obscene words) and copropraxia (involuntarily showing rude gestures) are observed, showing a 39% and 20% prevalence rate respectively (Eapen and Robertson, 2015). While TS does not reduce life expectancy or intelligence, it may cause social problems, functional interference in tasks, psychological distress, creating a lasting impact on quality of life. (Novotny et. al, 2017). In this paper, we aim to examine TS through an evolutionary perspective, with the goal of presenting fresh insights into its origins and broader implications for the treatments in patients.

Rationale for evolutionary explanation in Tourette's syndrome

Mental disorders are known to have a very complex aetiology arising from an interplay of biological, social and psychological factors, making their diagnosis and treatment difficult among professionals. Evolutionary psychiatry seeks to address this by providing mechanistic explanations as to why traits associated with mental disorders have persisted throughout human evolution, and how they have manifested as dysfunctional vulnerabilities in the modern world through evolutionary mismatch (Nesse, 2023). Rather than viewing these traits solely as pathological, evolutionary approaches consider whether they may have represented byproducts of defunct adaptive mechanisms, having once conferred advantages in ancestral environments.

Within this framework, TS presents a strong candidate for evolutionary interpretation. TS is a relatively common disorder affecting up to 1% of the population (Kattner, 2022), with historical records suggesting its presence. While TS was formally characterized by neurologist Georges Gilles de la Tourette in 1885, descriptions of tics in medical texts dates as far back as the Ancient Greeks, by physician Aretaeus of Cappadocia (Diaz-Anzaldua and Rouleau, 2008). The persistence of tics across centuries suggests an underlying biological basis that was conserved over time.

Genetic studies provide further evidence for this. Twin studies have reported that concordance rates of TS were 53% in monozygotic twins and 5% in dizygotic twins (Price et. al, 1985). Heritability of Tourette's syndrome is high at 58% (Davis et. al, 2015), with several candidate genes and loci being identified that may be related to TS. This begs the question of why these genes and associated traits have persisted throughout human populations if they have strong evolutionary stability.

Neurobiological differences of TS

One of the widely studied mechanisms of TS involve altered dopamine regulation in the basal ganglia. Dopamine, often associated with reward and motivation, reinforces behaviours through feelings of pleasure. Brain imaging studies reveal increased dopamine release and dopaminergic

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hyperinnervation in the striatum of TS patients (Singer et. al, 2002; Maia and Conceição, 2018), possibly influencing the habit formation of tics. In fact, individuals of TS report feeling a sense of reward accompanying their ticcing actions.

While dopamine is a useful neurotransmitter in motivating behaviors essential to survival and reproduction, its role in social contexts remains less understood. The discrepancy of how these alterations of dopamine regulation maintained through evolution might have meant that these traits have worked advantageously in specific socioecological contexts in early humans and even primates (Yamaguchi et. al, 2015). An interesting take by Reser suggests that the tendency for tics were an important trait for restlessness that allowed individuals to be hypervigilant with their immediate environment (Reser. 2017). This reactivity could have improved survival by making humans more alert to dangers when hunting or foraging.

Tic behaviour in modern social contexts are seen as antisocial, indecent and irrelevant. Many TS individuals consciously engage in tic suppression to avoid humiliation especially in socially sensitive situations. Normally, executive functions in the frontal lobe influence the premotor cortex to create motor signals which then get amplified in the basal ganglia. However, TS is associated with the lack of activity in the dorsolateral prefrontal cortex of the frontal lobe, an area usually concerned with generating appropriate actions (Moriarty et. al, 1995), which might lead to an inability to filter these ticcing desires. The dopamine overactivity in basal ganglia with reduced frontal lobe control, may have favoured repetitive automatic movements controlled by the basal ganglia in these high-risk situations. A possibility that in ancestral environments speedy reactions mattered more than social norms, allowing tics to be considered the most appropriate behaviours ingrained into the basal ganglia of early humans.

Many emotional processing structures in the limbic system also found to be activated during tics, leading Leisman and Sheldon to hypothesise that there may be an emotional connection to ticking behaviours (Leisman and Sheldon, 2022). Most individuals with TS also exhibit heightened stress reactivity via the hypothalamic-pituitary-adrenal axis, with increased norepinephrine release and exacerbates tics under pressure (Cohen and Leckman, 1992). While this makes it problematic for TS individuals to communicate or function in highly stressful situations, Progovac and Benítez-Burraco offers the possibility that in these high-risk situations, tics, especially offensive tics might have served a purpose of either showing aggression or as an act of humor to relieve stress (Progovac and Benítez-Burraco, 2019). Obscene languages and gestures, as seen in coprolalia and copropraxia, are used for expressions of deep emotions such as aggression, fear, pain and frustration, which may have been used as a defense mechanism to relieve stress and pain through stress-induced analgesia (Husain et. al, 2023). Contextually, humor in profanity is also seen as a natural stress reliever by decreasing cortisol levels (Vrticka et. al, 2013). While expletives were common in many primates, its use naturally decreased with self-domestication and development of more complex linguistics.

Compensatory benefits of TS

Despite the challenges of tics, individuals with TS often demonstrate compensatory advantages. A major compensatory benefit found in individuals is that on average, they perform quicker in behavioral tests of cognitive motor control (Jackson et. al, 2011) and are significantly better at judging time intervals compared to typical individuals (Vicario et. al, 2010). Georgiou et. al revealed that TS individuals outperform controls in fast, goal directed movements, being more force efficient in their movements (Georgiou et. al, 1997).

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The study also showed that TS individuals showed reduced functional asymmetry in which both their hands showed little differences in performance compared to controls, likely due to little basal ganglia asymmetries in TS individuals. This increased coordination and motor control may have been advantageous in making precise and rapid movements in hunting scenarios. In modern sports, some TS individuals may have an advantage in competitive sports (Reser, 2017), with many famous athletes performing in professional teams. Although the presence of motor tics is still disadvantageous in modern sedentary lifestyles, individuals with TS may turn to exercise as a goal to achieve better suppression of tics chronically (Kim et. al, 2018).

Interestingly, while stress and overstimulation exacerbates tics, hyperfixation instead temporarily suppresses tics in TS. One hypothesis suggests that hyperfixation, also common in OCD and ADHD, may have been valuable in hunter-gather societies in prolonged tracking of prey, or problem -solving in high-stakes situations (Hartmann, 1993). Though disruptive in a modern classroom context, this may come in handy in workplaces with extreme focus, with many successful pilots and surgeons being documented (Sacks, 2004).

While the link between dopamine and creativity is not well-understood, some literature suggest that high level bursts of dopamine are associated with creative thoughts. In individuals with TS where dysfunction of dopamine regulation results in bursts of dopamine and subsequently uncontrolled tics, this might have resulted in enhanced creative thinking. Studies have shown that participants with TS scored higher in some areas when doing creativity tests (Wei, 2011; Zanaboni Dina et. al, 2017), although the results were not significant and require further testing. High levels of creativity are have also been reported in many single cases of individuals with TS, notably Mozart, and a musician known by 'Witty Ticky Ray' who used his tics to his advantage in jazz improvisation, as reported in Oliver Sack's book (Colautti et. al, 2021). Creativity therefore might be a crucial survival skillset in resource-poor ancestral contexts, requiring innovation in gathering food, shelter and defence.

Limitations of the evolutionary perspective

While most points in the paper are interesting and seem plausible, there is no direct proof that tics have existed in prehistoric humans or that they conferred survival advantages. Until anthropologic evidence proves tics in prehistoric humans, the evolutionary basis of TS will still be up for debate, likewise for other mental disorders.

A common criticism is that evolutionary explanations are too reductionistic, where it oversimplifies the multifactorial nature of most mental illnesses. TS is an extremely complicated disorder consisting of a wide spectrum and severity of symptoms across patients. Simplifying a disorder with an evolutionary background might not do justice to all the associated symptoms. Reser also warned that the compensatory benefits of tics might not be a fully advantageous trait in prehistoric times, but instead a result of coping mechanisms unique to TS patients adapting to the modern environment (Reser, 2017).

Another consideration is the relevance of other tic disorders, notably functional tic disorders. While tics in TS are solely linked to structural abnormalities in the brain and have a gradual onset over multiple years, functional tics show an abrupt appearance lasting days to weeks. Unexplained trends such as the sudden rise of functional tics during the COVID-19 pandemic (Heyman et. al, 2021) might include short-term causes such as psychological effects and increased social media and digital technology use. While both disorders appear almost similar symptomatically, their differences in pathophysiology might show that the aetiology of tics might not be a clear image.

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Implications in treatments and education

Traditionally, interventions such as behavior therapy have focused predominantly on the suppression of tics through habit-reversal training. However, the act of suppressing tics is mentally intensive and exhausting, which potentially increases stress and worsen tics. Antipsychotics are also prescribed to manage ticking symptoms in TS patients, however, side effects are common and their effectiveness varies within patients. Anecdotally many individuals with TS report that they are physically slower, less coordinated and slower in conversations when on medications (Sacks, 2004).

However, an evolutionary perspective encourages a reevaluation of TS, as not solely a harmful disorder with tics that need to be eradicated, but as a form of neurodiversity in psychiatry where individuals with TS might still be able to live at the fullest extent alongside neurotypicals. From a therapeutic standpoint, treatment should not aim to solely reduce tics but to also promote resilience through harnessing these adaptive strengths in a patient. Although not evidence-backed, the evolutionary mismatch hypothesis can be explained to patients during therapy to raise their motivations and encourage them to accept their diagnosis. Similarly, increasing awareness with an emphasis on the positive attributes of TS, along with more exposure of TS, may combat myths and stereotypes about the disorder and change negative attitudes within the general public. A positive outlook towards TS may be a step forward in advocating for policies to reduce discrimination and promote equal access to jobs and amenities for individuals with TS. By shifting a disadvantage to an advantage, society can provide an inclusive and empowering environment that enhances psychosocial functioning towards individuals with TS.

Conclusions

Through the current evidence describing the traits seen in TS, we derive a possibility that the aetiology presented in TS might have been backed up by evolutionary processes. Differences seen in the neurophysiology of individuals of TS might have suggested that their brains might have been wired differently favouring perhaps past environments which has caused the outbursts of ticking behaviours. Compensatory benefits have also suggested that TS might not entirely be pathological and might have conferred advantages that helped with survival and reproduction in hunter-gatherer societies. Proving that TS is in fact an adaptive condition of the past would be extremely difficult due to the scarce evidence and complicated nature of the problem, and should only be taken as exploratory and speculative. However, an evolutionary perspective would still be useful in identifying current knowledge gaps, promoting awareness and influencing current treatments for TS. In the meantime, more research would be required to fully establish a link between TS and evolution, such as looking into the epigenetics of TS in relation to different environmental stressors, cross-cultural and animal studies of TS and anthropological evidence of tics existing in prehistoric times.

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Previous Competition Essays

How an Evolutionary Perspective can improve our understanding of Depression

Krishna Prakash, Staff Grade

Abstract

Depression is one of the most common psychiatric conditions worldwide and continues to place a huge burden on individuals, families, and healthcare systems. Despite decades of research, current explanations and treatments often fall short, leaving us with high rates of relapse and treatment resistance. The conventional biomedical, psychological, and social models describe how depression develops, but they do not fully explain why this vulnerability has remained so widespread in human populations.

An evolutionary perspective offers a different way of looking at the problem. Rather than seeing depression purely as an illness or fault, it considers whether the traits we now call symptoms might once have had survival value. Several theories highlight possible adaptive functions: the social competition hypothesis suggests that withdrawal after defeat reduces conflict; the analytical rumination model proposes that low mood encourages focused problem-solving; energy conservation and immune models see fatigue and anhedonia as protective responses in times of stress or infection; attachment and signalling theories view depression as a way of eliciting support; and the mismatch hypothesis suggests that modern environments repeatedly trigger ancient responses that no longer fit.

These perspectives do not replace existing models, but they enrich them. They help reduce stigma, encourage compassion, guide therapies like behavioural activation and compassion-focused therapy, and inspire new biological and public health approaches. By placing depression in the wider story of human evolution, we gain a deeper, more humane understanding of this complex condition.

Depression is one of the most common and challenging conditions in psychiatry, affecting more than 280 million people worldwide. It contributes to a large proportion of disability and loss of quality of life, not only for the individuals themselves but also for families and societies. Despite decades of research and treatment advances, a significant number of patients do not respond to standard therapies, while many relapse even after periods of recovery. This leaves clinicians with a sense that something important is missing in our current understanding. Conventional approaches—biological, psychological, and social—are helpful, but they cannot fully explain why depression is so widespread and persistent across cultures and generations. From an evolutionary perspective, this puzzle becomes an opportunity: instead of asking only how depression works, we also ask why humans remain so vulnerable to it. This shift can generate new insights, challenge stigma, and open up more compassionate ways of thinking about depression.

When psychiatrists describe depression, they usually highlight a mixture of symptoms: sadness, loss of pleasure, fatigue, poor sleep, appetite changes, impaired concentration, and feelings of hopelessness. In severe cases, suicidal thinking or behaviour may also occur. Classification systems such as DSM-5 and ICD-11 define the disorder when symptoms are persistent and impairing. The biomedical approach often frames depression as a result of disrupted neurotransmitters like serotonin or dopamine. Psychological models emphasise patterns of negative thinking, learned helplessness, or attachment difficulties, while social approaches link depression to trauma, inequality, and stressful environments.

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Each perspective contributes something useful, but none fully answers the deeper question: why would evolution leave us prone to such a debilitating state? If depression only reduced survival and reproductive chances, natural selection would have eliminated it over time. Its ongoing presence suggests that depressive states may sometimes have served adaptive purposes, or at least reflect side effects of systems that were once beneficial.

One influential explanation is the social competition hypothesis, described by John Price and colleagues. They argued that depression may act as a biological and psychological mechanism for submission after social defeat. In many primate species, when an individual loses a contest for dominance, it withdraws, lowers its posture, and avoids further fighting. This submissive response prevents serious injury by signalling acceptance of lower rank. Humans, too, may express a version of this strategy. Feelings of worthlessness, low energy, and withdrawal in depression can be seen as echoes of an ancient survival response, designed to reduce conflict when defeat is inevitable. For example, someone passed over for promotion may develop low mood and step back from further competition. Although painful in modern life, such withdrawal may once have reduced risks and preserved group harmony.

Another important theory is the analytical rumination hypothesis, put forward by Andrews and Thomson. They suggested that depression narrows mental focus and promotes persistent thinking about difficult problems. This can look maladaptive in the clinic, where rumination traps people in cycles of negative thought. But in ancestral environments, persistent attention to a complex social or survival dilemma could have been helpful. For instance, a person facing the breakdown of an alliance or uncertainty about resource sharing might have benefited from prolonged reflection before acting. Although this mechanism can now become overextended and harmful, it may once have been an adaptive way of forcing deep analysis of high-stakes issues.

A different line of explanation comes from the energy conservation and immunological perspectives. Depression often involves psychomotor slowing, fatigue, and loss of interest in activities. From an evolutionary angle, these features may reflect an energy-saving mode triggered by stress, illness, or famine. Reducing activity and conserving resources would have improved survival when energy supplies were scarce. This idea is closely tied to the sickness behaviour model, in which inflammatory processes during infection trigger fatigue, loss of appetite, and social withdrawal to allow recovery. Some research suggests that chronic inflammation in modern societies may inappropriately trigger these ancient pathways, producing depressive symptoms even when no infection is present. This helps explain why some cases of depression are associated with raised inflammatory markers, and why anti-inflammatory agents are being investigated as treatments.

Depression has also been interpreted as a way of signalling need within social groups. Crying, sadness, and withdrawal may communicate distress and elicit care and protection from others. In small, interdependent communities, these signals could mobilise group support and strengthen bonds, increasing chances of survival and recovery. The fact that many episodes of depression are linked to bereavement, rejection, or other forms of social loss supports this view. Seen in this way, depression is not just an individual problem but also a form of communication within relationships and communities.

Finally, the mismatch hypothesis provides a broad explanation for the high rates of depression today. Our ancestors lived in small groups with close social ties, daily physical activity, exposure to natural environments, and limited social comparison. Modern life, by contrast, often involves isolation, sedentary habits, artificial environments, and constant exposure to unrealistic standards through media. Mechanisms that once protected survival may now be repeatedly triggered in situations they were never designed for. This mismatch between ancient adaptations and current living conditions may explain why depression has become so common in industrialised societies.

These evolutionary perspectives do not only offer academic interest; they also have practical implications for how we respond to depression. First, they can reduce stigma.

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If depression is understood as part of the human repertoire of responses, shaped by natural selection, then it is less likely to be seen as personal weakness or simple malfunction. Patients may feel less broken and more able to engage with treatment. Second, these perspectives align with therapies already in use. Paul Gilbert's Compassion-Focused Therapy draws explicitly on ideas of social rank and shame, aiming to activate the brain's affiliative systems. Behavioural activation, which encourages engagement in meaningful activities, can be understood as a way to counteract maladaptive energy-conservation responses. Interpersonal psychotherapy makes sense when viewed through the lens of attachment and social signalling, helping people adjust to losses and changes in key relationships.

Evolutionary models also inspire biological research. The link between inflammation and depression has prompted studies into anti-inflammatory drugs as potential treatments. Recognising overlaps between mood and immune function broadens research beyond neurotransmitters and encourages integrative models of body and mind. From a public health perspective, if mismatch plays a major role, prevention strategies should aim to create environments that restore aspects of ancestral living: strong social networks, opportunities for physical activity, healthy sleep patterns, and reduced chronic stress. Workplaces, schools, and communities could all benefit from this broader understanding of what human beings need to thrive.

However, it is important to be cautious. Evolutionary psychiatry faces criticisms. Some hypotheses can become unfalsifiable, turning into elegant but speculative 'just-so stories.' Not all depressive episodes are adaptive; severe cases can lead to disability and suicide, outcomes that clearly reduce survival and reproduction. It is likely that depression is not one thing but many, with different subtypes reflecting different mechanisms. There are also cultural differences: while some expressions of sadness and need may attract support in one society, they may lead to stigma or exclusion in another. Genetic studies suggest that vulnerability to depression is influenced by multiple small genetic factors interacting with environment, further complicating simple adaptationist stories. Thus, evolutionary accounts should not replace other perspectives but be integrated with them, and clinicians must never minimise suffering by implying that it is acceptable just because it might once have been useful.

In conclusion, depression remains one of the greatest challenges for psychiatry. Conventional models explain how depression arises, but evolutionary perspectives ask why humans are prone to it in the first place. Theories of social rank, rumination, energy conservation, social signalling, and mismatch each contribute pieces of the puzzle. Together, they suggest that what we now call depression may in part be the product of ancient systems that once supported survival but that can misfire in the modern world. For clinical practice, this view can reduce stigma, encourage compassion, shape therapies, and inspire research into new treatments and preventive strategies. By placing depression within the broader context of human evolution, we can deepen our understanding and move towards more humane and effective responses.

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Previous Competition Essays

How an Evolutionary Perspective can improve our understanding of Eating Disorder

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"It is more important to know what sort of person has a disease than to know what sort of disease a person has."

This thought-provoking aphorism, attributed to Hippocrates, emphasises the importance of understanding the broader human context to unravel the mysteries of illness.

Mental disorders, shaped by complex interactions of multiple processes, rarely fit neatly into diagnostic categories. Modern classificatory systems have moved towards dimensional approaches, but fundamental questions remain—particularly *why* such vulnerabilities arise.

Eating disorders illustrate this challenge. Their aetiology is multifactorial, involving genetic, biological, cultural, and socio-environmental influences, and their classification remains complicated by overlapping presentations and the lack of validated psychometric measures capturing this distinction. [1]

Estimates indicate that at least 1.25 million people in the UK are affected by eating disorders, while up to 6.4% of adults display some signs of disordered eating. Substantial evidence shows a global increase in the incidence of eating disorders. Between 2015 and 2021, hospital admissions for eating disorders increased by 84%, with children and young people being the most affected group. [2-4] The variation in their presentations, alongside shared epidemiological features, suggests the need to look beyond the biopsychosocial model and to incorporate a broader biological framework to understand it in totality.

Although medicine is increasingly moving towards personalised and precision approaches, the heterogeneity of eating disorders makes such applications difficult. As Erasmus Darwin suggested more than two centuries ago, "a deeper understanding of disease may emerge by tracing its origins back to evolutionary processes." [5] Evolutionary medicine offers such a framework. By distinguishing between proximate reasons and ultimate causes, it provides insights into the adaptive roots of eating behaviours.[6] Natural selection, while favouring traits that enhance survival and reproduction, may also predispose individuals to maladaptation in modern environments. Through this lens, eating disorders can be seen not merely as pathological deviations, but as exaggerated expressions of once-adaptive strategies, shaped by environmental, developmental, and social contexts.

Eating behaviour- an evolutionary paradigm:

For most of human history, survival depended on a nomadic forager lifestyle, requiring high energy expenditure in environments where food was scarce and uncertain. These conditions likely fostered adaptive preferences for energy-dense foods. Natural selection would thus have favoured individuals who consumed and conserved energy efficiently. Yet this evolutionary legacy sits uneasily with the realities of eating disorders, where behaviour departs dramatically from such adaptive tendencies.

Lutter and Nestler propose that two interrelated systems governing eating behaviour: the homeostatic system, which regulates energy balance, and the hedonic system, which drives appetite for rewarding foods such as those rich in sugar and fat. Over the course of human evolution, the hedonic system may have overridden the homeostatic one, creating a predisposition towards high-energy foods predisposing to obesogenic environments where such foods are widely available although this does not explain the restrictive eating disorders.[7] Thus, eating disorders could possibly be an offshoot of an adaptive meta-problem that emerges when evolutionary motives—such as those related to survival and mating—conflict with the modern abundance of hyper-rewarding but unhealthy food.[8]

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Theories of Eating Disorders from an Evolutionary Perspective:

Pioneers in evolutionary medicine have attempted to understand eating disorders through perspectives that go beyond traditional biomedical models. The high heritability and ethnic variations in prevalence suggest that natural selection may play a role. Several theories have been proposed, some addressing obesity, while others focus on anorexia nervosa and bulimia nervosa. Highlighting these theories provides valuable insight into how evolutionary processes may shape vulnerability to eating disorders.

For convenience, I have tried to divide the theories into 5 broad headings:

- Adaptation to food scarcity,
- Reproductive strategies
- Social competition
- Evolutionary mismatches.
- Neurobiological extensions of evolutionary models

1. **Adaptation to Food Scarcity**

Several theories argue that eating disorders stem from mechanisms that once supported survival during periods of famine. The **Thrifty Gene Hypothesis** suggests that genes promoting fat storage were favoured because they enhanced survival and reproduction during times of scarcity.[9] An extension, the **Dual Intervention Hypothesis**, posits that adiposity was regulated by a minimum threshold to prevent starvation and a maximum threshold to avoid predation. With the decline of predation, upper thresholds have relaxed, facilitating weight gain in modern contexts.[10]

Similarly, the **Seasonality Hypothesis** emphasises the disruption of seasonal eating patterns due to food abundance and the dominance of ultra-processed foods, which may have destabilised evolved metabolic systems.[11]

The **Adapted to Flee Famine Hypothesis (AFFH)** frames anorexia nervosa as an adaptive response: food restriction, denial of starvation, and hyperactivity may once have enabled migration during famine. While compelling, this model struggles to explain why individuals resist eating even in times of plenty, and why the disorder is more prevalent among women.[12] The **Insurance Hypothesis** links fat storage to buffering against food shortages, but in contexts of perceived security, food intake may paradoxically shut down, predisposing individuals to anorexia.[13]

2. Reproduction and Kin Selection

The **Reproductive Suppression Model**, one of the earliest evolutionary models proposes that anorexia reflects an adaptive mechanism for postponing reproduction during unfavourable conditions, thereby maximising lifetime reproductive success. This explains why anorexia often emerges in adolescence, a critical stage in reproductive life.[14] The **Kin Selection Hypothesis** suggests that restrictive behaviours may enhance inclusive fitness by enabling individuals to function as "helpers-at-the-nest," supporting kin survival rather than pursuing reproduction themselves. This resonates with patterns of altruism and self-sacrifice often reported among patients with anorexia.[15] The **Parental Manipulation Model** further argues that parental control over daughters' reproductive strategies may have adaptive roots in stratified societies, though it does not fully account for eating behaviours external to family context and shifts in anorexia prevalence.[16]

3. Social and Sexual Competition

A third cluster of theories situates eating disorders within the dynamics of social status and sexual selection. The **Social Threat Hypothesis** argues that anorexia is a product of competition of status in a group. Whereas plumpness once signalled wealth, thinness is now associated with prestige and self-control in Western societies.[17]

The **Sexual Competition Hypothesis (SCH)** builds on Darwin's principle of sexual selection, suggesting that women compete through inter and intersexual competition for mates by signalling reproductive potential through thinness. In ancestral environments, slenderness was a reliable cue of nubility, and fertility and natural selection would not only favour them but also those with *psychological adaptation* that signals anxiety or

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alarm if such phenotypic cues are disturbed or threatened.

In modern environments, however, this competition has intensified into maladaptive "runaway" processes, producing disordered eating.[18] This theory is supported by cross-cultural studies linking thin ideals with socioeconomic status and media exposure,[19] the socially contagious nature of eating disorders within schools, and parallels such as "reverse anorexia" among male bodybuilders.[20]

Life History Theory refines the perspective of SCH by situating eating disorders within reproductive strategies. Individuals with bulimia nervosa often pursue "fast" life history strategies, characterised by impulsivity, sensation seeking, and short-term mating. By contrast, individuals with anorexia nervosa tend to follow "slow" strategies, delaying reproduction and signalling long-term investment potential. This dichotomy helps explain why both conditions persist, despite their apparent costs, as alternative strategies within the spectrum of reproductive adaptation.[21]

4. Mismatch Theory:

A few authors understood the incompleteness of the SCH and hence proposed the **Mismatch Hypothesis**. This theory argues that mechanisms regulating food intake and mating, once -adapted, have become antagonistic in contemporary societies, creating adaptive "meta-problems" that manifest as eating disorders. This broader framework also explains the rise of other lifestyle-related diseases, such as type II diabetes and cardiovascular conditions.[8]

5. Neurobiological Extensions of Evolutionary Models

Neurobiological research conducted over the last few years, offer a biologically grounded explanation.

The **Neuroendocrine Evolutionary Model** provides a striking example of how homeostatic mechanisms may manifest maladaptively. Research on the hypothalamic arcuate nucleus has identified agouti-related protein neurons that regulate energy utilisation. Furthermore, eating behaviours stimulate serotonin and dopamine pathways, shaping cortical and subcortical networks involved in reward. Food restriction triggers dopamine release in the mesolimbic system, paradoxically reinforcing dieting behaviour and sustaining anorexia. Similarly, activation of brainstem noradrenaline neurons enhances attentional focus, conditioning anorexia as a reward-linked behaviour.[22] Critics, however, note the difficulty in distinguishing whether such neural changes are causes or consequences of starvation.

The **Extreme Female Brain (EFB) Hypothesis** (Baron-Cohen, 2002) situates eating disorders within cognitive and social-emotional processing, linking them to heightened social sensitivity and fear of negative evaluation. Emotional processing deficits, such as difficulty recognising emotions, may drive disordered eating as a maladaptive coping strategy. While valuable, this model has been criticised for reinforcing gender stereotypes and neglecting male and non-binary experiences.[23,24]

The **Psychoneuroimmunological Model** (Rantala et al.) connects eating disorders to stress, inflammation, and gut microbiota. Starvation and compulsive behaviours may exacerbate neuroinflammation, while binge eating may be linked to inflammatory processes driven by visceral adiposity. Alterations in the gut–brain axis further suggest a continuum from anorexia to bulimia shaped by stress sensitivity. Though innovative, this model remains largely speculative, with causality yet to be established.[8]

Finally, genetic studies reveal that eating disorders are highly polygenic. GWAS findings (Watson et al., 2019; Bulik et al., 2022) implicate multiple loci, though with small effect sizes, suggesting that natural variation in traits such as conscientiousness and neuroticism may predispose individuals. [25,26] Recent works by Breton et al. (2025) has highlighted circadian genes, particularly **ARNTL**, as potentially central to eating disorders. Disruptions in circadian regulation have been associated with altered locomotor activity, delayed puberty, disordered eating, and reduced fertility in animal models.[27] While intriguing, the functional significance of these associations remains to be fully established, and effect sizes in human studies remain modest.

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Engel's Biopsychosocial Model	Tinbergen's Four Questions			
	Proximate Causes		Ultimate/Evolutionary	
	Mechanism	Development	Function	Phylogeny
Biological	Dysregulation of serotonin, dopamine, opioids. Hormonal imbalances (ghrelin, leptin, HPA axis). Genetic predispositions Starvation → physiological adaptations (bradycardia, amenorrhea).	Pubertal changes influencing body image and appetite. Epigenetic effects of early adversity. Critical periods: adolescence	Energy conservation/ restriction during famine. Activity-based anorexia (increased mobility under food scarcity promote foraging). Identification of role of various hormones in appetite regulation and interaction with neurotransmitters.	Fasting/starvation responses seen across species Evolutionary trade-off between energy storage vs. reproductive suppression
Clinical Implications	Identify targets for drugs possible discovery of biomarkers	Identify individuals at risk for developing condition. Primordial prevention measures could be implemented disease onset	Psychoeducation and explanation of associated symptoms with a biological basis and how they can be avoided. Predict side effects of drugs and possible extraneous harms of treatment	Identify homologous systems in animal models for drug testing
Psychological	Distorted body image. Perfectionism, obsessive traits. Emotion dysregulation food restriction, binge, purge cycles.	Early attachment problems, adverse childhood experiences. Learned reinforcement: dieting → praise → maintenance of disordered eating. Social comparison processes sharpen in adolescence.	Control of food as a coping strategy when other domains feel uncontrollable. Restriction may temporarily reduce anxiety and provide sense of mastery.	Traits like perfectionism, sensitivity to social evaluation present in primates and humans. Cognitive biases around food/threat detection may reflect ancient survival strategies
Clinical Implications	Identify harmful thoughts, moods and behaviours as therapeutic targets. Identify the factors to break vicious cycle	intervene before harmful circumstances or psychological processes	Normalize and destigmatise; inform therapy.	Inform therapy by observing other species
Social	Peer/family pressure. Media and culture ideals of thinness.	Family dynamics (enmeshment, control). Social reinforcement (compliments on thinness). Bullying or teasing about weight.	Conforming to group norms for acceptance. Thinness as signal of discipline/ status in some cultures.	Cultural variation in body ideals across history (plumpness once signified fertility/wealth; thinness now often idealized).
Clinical Implications	Identify scenarios suitable for short term social intervention.	Identify areas for social reform, pre-empt at risk individuals and groups	Inform social reform interventions.	Identify optimum and sub-optimal social conditions over generations

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How is evolutionary theory going to make a difference in understanding eating disorder?

"Scientific advances often come from uncovering a hitherto unseen aspect of things, not so much through new instruments, but by looking at them from a different angle."

With the rising incidence of eating disorders and the limited effectiveness of current treatments, an evolutionary perspective makes us think as engineers considering *why* humans become unwell in particular ways and what evolved systems are disrupted when illness occurs. It also highlights the blurred boundaries between normative psychological states, adaptive symptoms, and pathological illness.

This approach is encapsulated in the evobiopsychosocial model,[28] which integrates Engel's biopsychosocial framework with Tinbergen's four evolutionary questions (Table on previous page).

By framing eating disorders as maladaptive amplification of once-adaptive responses in mismatched modern environments, clinicians can offer patients and families a more compassionate explanation that shifts blame away from individuals and reduce stigma Preventive strategies likewise benefit from this view: media literacy and dissonance-based interventions that challenge thin-ideal norms have proven effective. [29,30]

Applied clinically, evolutionary models have already influenced treatment. For instance, Södersten and colleagues reconceptualised eating disorders as disorders of eating behaviour than mental disorders, demonstrating that teaching patients to eat in a relaxed manner using real-time feedback produced better outcomes than standard cognitive behavioural therapy. [31]

Similarly, the **psychoneuroimmunological model** highlights the role novel therapies worth pondering such as anti-inflammatory agents, micronutrient supplementation, and even microbiota transplantation.[8] Genetic studies further implicate circadian pathways, linking anorexia nervosa with chronobiology and sleep regulation, raising the possibility of reframing therapy around functional recovery of evolved systems rather than rigid measures such as BMI thresholds.[27]

The integration of evolutionary theory into psychiatry does not replace the biopsychosocial model but enriches it, providing a deeper, unifying framework. By acknowledging both the adaptive roots and maladaptive expressions of eating behaviours, evolutionary medicine has the potential to advance diagnosis, personalise treatment, and improve prevention, offering new hope for patients affected by these debilitating disorders

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