



Evolutionary Psychiatry Special Interest Group (EPSIG) Newsletter January 2023

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Notes from the Editor

I hope you all had a good festive season and are raring to go in 2023. New year, new hope.

We have a fantastic line-up of evolutionary psychiatry events coming up, both in terms of free webinars through the World Psychiatric Association, as well as through our highly anticipated in person 5th Evolutionary Psychiatry International Symposium on 10th March 2023. The topics vary from childhood, to paranoia, eating disorders, compulsions and suicide. I look forward to seeing you there!

This newsletter also includes an excellent essay by Ella Greenwood, a medical student, who writes about the social risk and the propitiousness theories of depression.

FREE WPA EP Section web pages with links to all future webinars

Cas Soper's webinar **Thursday 26 January 2023**, 4pm GMT: C.A. Soper (Portugal) <u>'What Stops Us Killing Ourselves? An Evolutionary Perspective on Suicide'</u> https://www.wpanet.org/evolutionary-psychiatry

5th Evolutionary Psychiatry International Symposium 10 March 2023

- What can we learn from hunter-gatherers about child development? N. Chaudhary and A.
 Swanepoel
- What is the expected human childhood? Insights from evolutionary anthropology. W.
 Frankenhuis
- Evolutionary perspectives on paranoia and conspiracies. N. Raihani
- The evolution of suicide: The Pain and Brain theory and its implications. C.A.Soper
- Making sense of the ultimate and proximate causes of eating disorders. A. Ayton
- Evolutionary models of obsessions and compulsions. M. Kaser
- Evolutionary perspectives on human emotions. L. Al Shawaf

Please register below

https://www.rcpsych.ac.uk/events/conferences/detail/2023/03/10/default-calendar/evolutionary-psychiatry-sig-meeting-2023

Medical student essay

Essay by Ella Greenwood, a fourth year medical student at the University of Sheffield (hoping to specialise in psychiatry) interested in evolutionary approaches to understanding the mind, following her intercalated masters in cognitive science last year. She wrote this essay for a student selected project, where she focussed on evolutionary explanations for low mood and depression.

Evolutionary theories for depression: The social risk theory and the propitiousness theory

Depression is an extremely prevalent mental disorder, characterised by symptoms including persistent low mood, loss of enjoyment from previously enjoyed activities (anhedonia), reduced energy and altered appetite and sleep (American Psychiatric Association, 2013). In high income countries, it has an estimated lifetime prevalence of 14.6%, with 5.5% of the population affected per year. It is most prevalent in the second and third decades of life and is a leading cause of disability across the globe (National Institute for Health and Care Excellence, 2022). Whilst we are gaining an understanding of the underlying physiology as well as the aetiology of depression, with factors such as adverse life events, genetic vulnerability and neurochemical imbalances being implicated (Schnieder & Wisskink, 2018), we have little understanding of why this disorder is so prevalent, striking so many otherwise fit and well individuals in their prime. Researchers are increasingly turning to evolutionary theory to shed light on mental disorders, including depression. Evolutionary psychiatry is a growing academic field, aiming to deepen the understanding of mental disorders by appealing to Darwin's theory of evolution by natural selection. This research programme offers the

prospect of revolutionising psychiatry's understanding of psychopathology, by addressing the question of *why* mental disorders occur in the population (Stein, 2006).

This essay will clarify the role of evolutionary theory in explaining the occurrence of depression, before outlining and evaluating two leading competing evolutionary explanations for the disorder: the social risk theory (Allen & Badcock, 2003) and the propitiousness theory (Nesse, 2000). Whilst a range of alternative evolutionary explanations exist (for example the analytic rumination theory (Andrews & Thompson, 2009) and the social competition theory (Price et al., 2018)) this essay will focus on the social risk and propitiousness theories, as each present a plausible evolutionary account of depression supported by a body of empirical data.

Evolutionary theory and depression

Evolutionary approaches to understanding depression aim to explain the phenomenon as occurring as a result of our evolutionary past. These theories posit that at some point in our evolutionary history, environmental pressures on our ancestors resulted in the selection for genes which now make us vulnerable to the development of depression. In order to have been selected for, possession of these genes must have resulted in a trait or traits which conferred a fitness advantage. That is, the trait must have improved individuals' chances of surviving, reproducing and thus passing on their genes. Such a trait is said to be an adaptation. In the case of mental states such as moods (depression is a mood disorder), it is not the mental state in itself which is selected for, but rather the biological system which generates the mental state in response to certain circumstances (Garson, 2019).

The disabling nature of depression makes it initially difficult to see how it could have possibly conferred a survival advantage. However, whilst the proposal that clinical depression itself is an adaptative state is contentious (though a number of theorists do hold this view), the proposal that low mood is an adaptive state is considerably more widely accepted (Hagen, 2019). Whilst depression is common, low mood is universal. Low mood shares many of the same features as depression, with its major difference being duration. Theorists largely accept that the generation of the feeling of low mood (or transient depressive states) in response to specific circumstances is an adaptive mechanism, and thus it conferred our ancestors a survival advantage. The view that low mood is adaptive whilst clinical depression is dysfunctional can be explained by likening these states to defensive bodily responses such as vomiting or diarrhoea. Whilst these responses are adaptive mechanisms, as they serve the function of expelling pathogens from the body, they can in some more severe cases lead to dehydration and even death. Similarly, on this view, low mood is an adaptive response to certain situations, whilst depression is a dysregulation of this mechanism (Keller & Nesse, 2005).

There are a number of strong reasons for the belief that the low mood system is an evolutionary adaptation. The production of low mood has many of the features that evolutionary biologists identify as characteristic of an adaptation. For example, low mood is reliably caused by similar triggering events (eg. loss or failure) in humans across all cultures. It is also reliably regulated by controlling and responding to its causes (eg. avoiding failure, extra social support following loss events). Reliable causes and regulatory mechanisms are hallmarks of adaptations. Furthermore, it is

a costly and complex phenomenon, yet it still persists with high prevalence in the population, suggesting that it is in some way beneficial. Depressive episodes also strike most commonly in the reproductive years, which is a feature suggestive of an adaptation rather than a dysfunction (dysfunctions are typically more prevalent with increasing age). Whilst the fact that low mood and depressive states are unpleasant and affect normal functioning may make them appear unhelpful, the same can be said for other adaptive states such as physical pain. Despite the fact that physical pain affects normal functioning, it is clearly adaptive, as it promotes avoiding physical damage as well as attendance to and protection of injury (Keller & Nesse, 2005).

Our understanding of the functionality of depressive states have important consequences in practice. For example, should we believe that depression is nothing more than a dysfunction due to underlying brain pathology, we may be more inclined to support the use of simple drug treatments to adjust brain chemistry. However, should we view depression as a functional adaptive state, this leads us to shift our focus to what this state indicates about current environmental circumstances, and whether it is in fact those circumstances that need changing. On this latter view, development of depression is in fact a healthy response to unhealthy circumstances.

The theories covered in this essay aim to provide an evolutionary explanation for our susceptibility to low mood, in order to shed light on the prevalence of depression in the population. Each theory focuses on situations in which it posits that low mood/ depressive episodes would have provided a survival advantage, inferring that activation of these states under such circumstances explains their selection for in our evolutionary past. In other words, each theory states that the collection of depressive symptoms (such as reduced motivation, poor self-esteem, fatigue and loss of interest in activities) conferred a survival advantage in certain circumstances faced by our ancestors. The theories differ in the circumstances they posit low mood to be an adaption to. Thus, they posit differing adaptive functions of the low mood system. The following sections will outline and evaluate these proposals.

The propitiousness theory

Randalf Nesse is an influential theorist in evolutionary psychiatry. He argues that low mood (and perhaps depression in some cases) is an adaptive state, and that many instances of depression are dysregulations of this adaptive state. Nesse (2000) proposes an evolutionary theory of low mood which explains it as an adaptation to unpropitious circumstances. Unpropitious circumstances are those in which an individual is unlikely to make net gains. In other words, the outcomes of an individual's endeavours are unlikely to be worth the efforts that they put into it. An example of an unpropitious circumstance faced by our hunter-gatherer ancestors would be foraging in extreme weather conditions (such as thick snow or extreme heat), where the amount of energy expended whilst foraging would have been increased, and the rewards reaped reduced. Thus, in these situations, continued unpropitious foraging attempts would lead to net losses. Nesse argues that in these instances, depressive symptoms such as decreased belief in one's foraging ability and decreased motivation to continue foraging (due to activation of low mood) would have resulted in an adaptive conservation of energy. For instance, in this hunter-gatherer example, individuals whose mood remained unchanged by the circumstances may have continued to expend energy foraging

and eventually died (eg. of exhaustion/starvation), whilst those who became low or depressed may have given up and conserved energy until conditions improved.

Nesse also theorises that decreased mood in unpropitious circumstances not only decreases wasteful energy expenditure but inhibits potentially harmful attempts to change circumstances. For example, in our evolutionary past, risky responses to unpropitious situations may have included breaking off from the group and venturing into new territory alone in attempt to find new food sources. Nesse raises the point that drastic action in unfavourable circumstances can often take things from bad to worse. In this case, for example, becoming lost or separated from the group and having to fend for oneself. Nesse posits that the inhibitory effect of decreased mood conferred a survival advantage in suppressing high risk responses to bad situations.

Application of Nesse's theory to modern-day circumstances predicts that situations such as failure to achieve career goals or failing relationships should commonly induce low mood. In these cases, Nesse suggests that low mood reduces excessive energy/time expenditure on the unsuccessful career goal or failing relationship. It also likely inhibits drastic action such as changing career direction or abandoning the relationship. As in the outlined hunter-gatherer cases, these effects may confer an advantage. For example, persisting in a career avoids being left unemployed, whilst remaining in troubled relationships may in some cases be less risky than being alone without support.

Nesse provides a plausible explanation for why reduction in mood under certain circumstances may have increased fitness in our evolutionary past. There is empirical evidence to suggest that situations which most commonly induce depressive episodes include those which involve humiliation and entrapment (Brown et al., 1995). It is also widely applicable as many situations may involve reduced net gains or net losses, ranging from personal failings to wider events which may trigger low mood (eg. COVID pandemic, national recession etc.) Nesse's theory also accounts for the prevalence of seasonal depression, a common condition characterised by low mood in the winter months (which would have been less propitious periods for our ancestors).

However, his theory encounters a number of issues. A clear problem for Nesse's theory is that it would seem that in many cases, making a significant life change in response to bad circumstances would result in a much more favourable outcome than sticking in an unpropitious situation. Whilst persevering with some bad circumstances may sometimes avoid detrimental situations, in many cases, completely changing direction may result in improved long-term outcomes. In many instances where career aspirations are not succeeding, changing course is a sensible decision, and unhappy relationships are often better off broken off. Additionally, it is not always advantageous to reduce the amount of effort put into a difficult/ unsuccessful endeavour. For example, some endeavours produce little net gain or even net loss for a significant period but may eventually result in large rewards (for example starting a new business). Low mood induced lack of motivation and commitment in these circumstances would prove detrimental rather than beneficial in many cases. Furthermore, Nesse's theory does not account for certain effects of low mood such as its inhibition of social activity and personal neglect/ poor hygiene, or its greater prevalence in women.

The social risk theory

The social risk theory, proposed by Allen and Badcock (2003), is another influential evolutionary explanation for low mood/depression. Similar to Nesse, Allen and Badcock argue that low mood and mild depressive states occur as an adaptive mechanism, whilst severe depression occurs due to a malfunction of this system. However, they argue for a different adaptive function of low mood/depressive states to that proposed by Nesse. Whilst Nesse emphasises the role of energy expenditure in his theory, Allen and Badcock argue that the need to maintain social relations and avoid group exclusion drove the evolution of the low mood system, by inhibiting risky social behaviour.

Leading researchers working in palaeoanthropology such as Robin Dunbar (1998) emphasise the crucial importance of social factors in our ancestors' survival and chance of reproductive success. In fact, Dunbar posits that navigation of social relations and group dynamics was the driving factor behind the evolution of large brains and advanced cognitive capabilities in primates. The social risk theory holds social factors central to the selection for the low mood system. On this account, low mood evolved as a defensive social adaptation, reducing risk of social exclusion and thus improving fitness through maintenance of social bonds. Allen and Badcock argue that the tendency to feel low mood in certain situations conferred this advantage in our ancestral environment through a number of ways.

Allen and Badcock argue that every individual has what they call a 'social investment potential' (SIP) which is said to mark their value to those around them. A person's SIP is a result of the ratio of their social value (their ability to add value to a group, for example, through providing resources) and their social burden (their cost to the group, for example, their use of the groups' resources). For example, a person who possesses valuable skills, is likeable and isn't greedy, is likely to have a very high SIP. Equally, an individual who possess little skill, is lazy and is greedy with food/ resources is likely to have a very low SIP. A person who has a very low SIP, ie. their burden to the social group is greater than their benefit, risks being ostracised from the group. In the ancestral environment, where people relied on a secure position in a group for resources and mates, expulsion from the group would have been detrimental to survival and reproductive success. A person's estimation of their own SIP is said to determine self-esteem.

The social risk theory states that low mood ensues when a person's estimation of their own SIP is sufficiently low or reduced. In other words, when a person believes that they are becoming more of a social burden/ and or less valuable to those around them, low mood/ depression ensues as a defensive response. This is said to be mediated by low self-esteem. Therefore, according to Allen and Badcock, low mood is a result of low self-esteem, triggered by belief that one's social value is low. Allen and Badcock argue that activation of low mood when SIP is low serves the function of preventing further reduction in SIP, thus preventing expulsion from the group. They also argue that low mood has a role in promoting increase in SIP. Low mood is said to prevent further reduction in SIP through inhibiting risky social behaviour (eg. confrontational or exuberant behaviour) which may further aggravate social relations. It is also said to act as a sign of submission/ passivity, so the individual is not seen as a threat, which is also said to reduce the individual's risk of expulsion. Effects such as reduced appetite in low mood also act to reduce a person's social burden.

Hypersensitivity to sources of social threat aids avoidance of risky situations and further aggravating others. Finally, Allen and Badcock argue that low mood also acts as a help seeking signal to others.

Allen and Badcock's theory is generalisable to many circumstances. Many factors could decrease a persons SIP, including both direct social events (such as falling out with friends) as well as personal failings, which may reduce an individual's internalised sense of social value and thus their selfesteem. The social risk theory predicts that in modern circumstances, social factors will influence low self-esteem. There is good empirical support for this prediction (Brown, 1978; Harter, 1993; Joiner & Metalsky, 1995; Leary et al., 1995; Maccoby & Martin, 1983). There is also evidence that low selfesteem is central to depressed states (Robsen, 1988) and that depressed states cause submissive social behaviour (Allen & Gillbert, 1997) as well as negative social comparisons (Buunk & Brenninkmeyer, 2000). In addition to a citing a strong body of empirical support for their theory, Allen and Badcock provide a reason for higher rates of depression in women. They describe this as being due to women's increased need for maintaining bonds and avoiding social risk (in order to help them through vulnerable periods of pregnancy and childrearing) giving them a decreased threshold for activation of low mood. Furthermore, the social risk theory explains the link between social media use and low mood, as application of the theory suggests social media may decrease a person's estimation of their own social value, as it results in disproportionate exposure to highly attractive and successful individuals.

However, as with Nesse's theory, the social risk theory encounters a number of issues. According to this proposal, low self-esteem (through reduced SIP) is the most crucial factor in triggering low mood. However, there may be many cases in which decreased mood is not linked to low selfesteem, such as seasonal depression, or low mood/depression following a loss such as a bereavement. However, the major issue for the social risk theory is that it is not convincingly clear that low mood/ depressive states actually would reduce social risk/ risk of group exclusion. It is certainly not clear that low mood would increase a person's SIP. Whilst low mood may inhibit some risky social behaviours, it is also likely to decrease a person's value to the group by reducing their contribution through work and parenting, which is shown to be decreased in depressed individuals (Hagen, 2019). Additionally, low mood can cause behaviours such as decreased personal hygiene, messiness and reduced efforts put into physical appearance (Stewart et al., 2022) which are likely to have a negative effect on a person's group status. In the case of both modern circumstances as well as in hypothetical ancestral circumstances, it seems that low mood/ depressive states may actually decrease a person's SIP. Empirical investigation suggests this may be the case, with negative affect resulting in increased social burden and risk of ostracism. This was found to be due to expression of psychological pain resulting in others also experiencing negative affect, leading to increased burden and exclusion (Wirth et al., 2019). This is a major concern for Allen and Badcock's fundamental premise.

Conclusion

Both of the theories covered offer theoretically plausible explanations for how the low mood system could have conferred a fitness advantage and thus been selected for. However, as outlined, each theory faces significant issues. Firstly, both theories face a scope problem. Nesse's theory struggles to account for the significant social effects of low mood and cannot explain the increased prevalence

of depression in women. Equally, the social risk theory struggles to account for the non-social causes of low mood, such as seasonal depression. A successful theory of low mood should be able to account for all of its key triggers. Additionally, it is not entirely convincing in either case that the 'adaptive' mechanism outlined would have necessarily increased fitness. This issue seems to be particularly apparent for the social risk theory, suggesting Nesse's theory may be the superior proposal.

However, there is strong reason to believe that the low mood system is an evolutionary adaptation. A large difficulty faced in generating evolutionary theories, is the mismatch between modern and ancestral environments. It may be that low mood served a beneficial function to our ancestors but is now largely maladaptive. This renders the evolutionary function of low mood somewhat speculative and difficult to test. However, the more we understand about our ancestral environment, the more solid basis we have to consider how our current traits could have benefitted our ancestors. Thus, we can understand more about the way in which our modern environment, and its differences from that in which our species evolved, leaves us vulnerable to conditions such as depression. Further theoretical and empirical work in evolutionary psychiatry will likely lead us to a more successful explanation as to why we are vulnerable to depression.

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