To What Extent Can Evolutionary Theories Explain Mental Illness?

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Abstract: Clinical psychology, the psychiatry field, and medical conditions all tend to ignore the role of evolution in them. However, the human brain is indeed a product of natural selection. Its complexity, including both normal functions and potential dysfunctions, has developed over evolutionary time under the process of natural selection. Evolutionary psychology provides guidance for psychologists today on how to think about individual differences in terms of psychopathology. Therefore, understanding the evolutionary origin of psychiatric conditions is a crucial component of a complete understanding of psychological illnesses and their etiology. This paper explores this idea of evolution with four examples of mental illnesses, depression, postpartum depression, agoraphobia, schizophrenia and autism.

Keywords: evolution, psychiatric condition, depression, postpartum depression, agoraphobia, schizophrenia and autism

1. Introduction

The etiology of mental disorders today can be addressed in multiple ways. Both nature and nurture influences play an important role in mental disorders and illnesses. Nevertheless, more and more recent research has found that genetics plays an important role and is highly involved in mental illnesses (e.g., 90% of trait variation for autism can be accounted for with genetics; bipolar disorder, 85%; schizophrenia, 81%; unipolar depression, 37%) which indicated the solid genetic factor under mental disorders [1]. According to natural selection theory, in evolutionary time where pressures from the natural selection will be responsive by removing all alleles associated with a particular disorder. Therefore, given the idea of natural selection in which only strong genes persist, the paradox between psychological illnesses and fitness genes was raised.

There is a growing misconception of natural selection today that it can lead to perfect genes and personal traits. However, our mental and behavioral traits are complex enough for risks from heritable disorders to persist in populations despite pressures and rules of natural selection. Natural
selection tends to respond to the trade-offs between the best fit genes while maintaining risk factors of mental illnesses simultaneously. The balancing selection theory refers to an explanation of this mechanism and the pressures of developing mental disorders behind natural selection.

2. Major Depression

Given the complicated society and environment human generations are living in, evolutionary errors will occur as social sources become more dynamic where new, deleterious mutations may arise faster than mechanisms of natural selection can remove them. This brought back the idea of mutation-selection balance, stating that natural selection will remove those heritable alleles that can lead to negative mental harm from the population, which balances their dysfunction with other fitness [2]. Therefore, from an evolutionary perspective, some of the pathological symptoms and behaviors today cannot be considered disorders since they contain no variations and dysfunctions in the gene, and so failed to balance between harm and fitness. More specifically, depression, as one of the most common mental disorders in human history, cast the light on the self-defense (defensive reaction) model [3]. It can be simply explained as a normal reaction or a response to hardship in life.

Most apparent evolutionary studies in the field of depression can be divided into two categories: 'dysregulation' models and 'adaptation' models. One significant difference between them is that the 'dysregulation' model focuses on mechanisms that can activate depression, whereas the 'adaptation' model states that depression itself can be considered an adaptive behavior whether there are activation mechanisms present or not.

More specifically, the former theory 'dysregulation' contends that depression should only be based on activating events that can deeply influence one's emotion and attitude, leading to a 'dysfunction' mood. Gilbert and Allen, for example, proposed that depressive symptoms are coping strategies people use to deal with threats and challenges (activation events) in their environment where 'flight or fight' is impossible [4,5]. Similarly, Nesse proposed that some common depression symptoms, such as low mood, are simply human adaptations as a response to their surroundings their demands were not met (activating events) [6]. The 'activating mechanism' in this case was their unattainable life goal, and so they came up with this depressed mood and the following depressive symptoms [7]. In general, 'dysregulation' researchers hypothesized that most clinical depression we think of today results from various types of activating events, such as chronic over-activation or inappropriate evocation of mechanisms (in the mood or affect system) that can lead to 'dysfunction' mood and symptoms. However, although those 'dysregulation' theories discovered the primary cause and physical mechanism of depression, they failed to went further into some more personal and innate aspects such as why some people are more likely to become depressed than others under the same activation events.

Given the concerns raised by the 'dysregulation' model, a broader theory dubbed 'adaptation' was proposed. Post-partum depression is a major component of this adaptational model. More recently, Watson and Andrews combined all cases of depression, including postpartum depression, into a single adaptation stance, which they dubbed the social navigational hypothesis' (SNH). This SNH has incorporated environmental factors, such as the one mentioned above, indicating that people today live in a very different environment than our ancestors. The SNH classified depression into two functions: the social ruminative function, in which adaptationists (people) are mentally trying their alternatives for complex social problems, and the social motivate function, in which adaptationists (people) are mentally considering alternatives by acquiring information and help from social networks, especially in cases where they (adaptationists) were unwilling or unable to do so [8]. These assistances were frequently provided by their close social networks, such as friends and partners. Because the average fitness interest among partners is lower because of partners becoming more replaceable, modern society has increased the context of ruminative and
motivational functions. According to the *Ancestral neutrality explanation*, the environment that modern humans are living in today is significantly different from those where we evolved and where our ancestors lived. Therefore, as evolution persists and the environment continues to change, social dynamics increases as population grow, and it becomes even harder for people nowadays to find a befitting than they were in previous societies. Therefore, the incidence of depression increases followed by a decrease in the possibility of fitness between partners. This depression behavior, as a ruminative model, can also explain the motivational function, in which a decrease in fitness interests increases the rate of depression to motivate other social networks to help. According to Watson and Andrews, depression is more likely to be caused by social problems, particularly when a person (adapters) experiences various types of losses in their relationships or lives or is unable to achieve a specific goal. Depression, according to ruminative function theories, is a way that has been triggered or designed to solve more difficult social problems. This is related to the percentage and proportion of cognitive resources in a social problem, which can also influence information interpretation. Hagen, for example, revisited on ideas of SNH by stating that post-natal depression is a mechanism used by mothers to import energy from social networks, particularly their partners, to their babies. This special case of depression will be discussed later in the following paragraphs [9].

3. Postpartum Depression

3.1. Definition

Postpartum depression (PPD) is a serious mood disorder. Women who develop PPD have feelings of intense sadness, worry and exhaustion following childbirth. PPD is common. Approximately one in seven women can expect to experience depression in the year following childbirth. Fortunately, this disorder is treatable [10].

3.2. Causes

This article will explain it from evolutionary psychology.

Here are two perspectives:

From the perspective of maternal, Hagen argues that postpartum depression benefits the mother and is a psychological function [9]. In evolutionary psychology, there is no kinship investment for no reason. When the cost is too high and the future is not optimistic, such as low offspring viability, insufficient investment from the father or others, environmental conditions that are not conducive to raising offspring, for example, postpartum depression is like an alarm bell to remind the mother.

In addition, Postpartum depression is a distress signal from mothers, demanding the partner's commitment. For example, two people plant a fruit tree together. One person is not satisfied that the other person does nothing but enjoy the success. She simply stops investing to force the other person to invest more.

From the perspective of offspring, postpartum depression is positively correlated with the Interbirth Interval Length, meaning that the older one can benefit from postpartum depression [11]. Because the longer the birth interval age, the more conducive the concentration of resources to the offspring individuals. It was also mentioned that support from partners and society influences postpartum depression and plays a key role in the length of the labor interval.

Thus, it shows that lack of support from partners and society is the main cause of postpartum depression in evolutionary psychology.
4. **Agoraphobia**

4.1. **Characteristics**

4.1.1. **Agoraphobia**

People diagnosed with agoraphobia will intensely fear two or more: standing in line or being in a crowd, being in open spaces, being outside the home by oneself, and being in enclosed spaces [12]. According to these features, agoraphobia patients cannot go out by themselves, and they will feel intense anxiety if they are exposed to the public alone.

*Agoraphobia* is an anxiety disorder that can balance dangerous and safe signals. Sometimes, agoraphobia can be considered a “non-exposure” onset [13].

4.2. **Causes of Agoraphobia**

4.2.1. **Evolutionary Factor**

Agoraphobia is a brain evolution that creates specific mechanisms and includes specific disorders or problems [14].

Many phobias and rituals stem from pre-prepared evolutionarily ancient mechanisms rather than new hazards [15]. According to Nesse, adaptation is one explanation of agoraphobia [16]. From an evolutionary psychology perspective, adaptation is the solution to the adaptability problem, which shows the design characteristics of solving the problem. The significance of adaptability is that it has solved the problem in the past. Also, adaptability can be understood as the result of repeatedly receiving the same stimulus, resulting in a self-protection mechanism. Based on the people diagnosed with agoraphobia, the behavior which the anxiety patients have may be helpful for living, such as preventing nonspecific invaders, which may be conducive to reproduction. The agoraphobic patients may avoid exposure to the public setting, which may help them avoid cheating and hunting [17].

According to Stein and Bouwer, anxiety disorder also can be explained as neuro-evolution [18]. As their article mentioned, human anxiety disorders are mediated by specific brain-based false alarms. The false alarms may be terrible events from the past, which the brain tells the patient that it is not an excellent choice to expose to the public, and they are afraid about this. The neuroevolutionary theory of anxiety disorders can explain the false asphyxia alarm of panic attacks which may increase blood pressure and heart rate when exposed to things that the patients may be afraid of, such as exposure to a crowded center. Neuroevolutionary theory can explain agoraphobia and other types of anxiety disorders.

Agoraphobia can be explained by various evolutionary theories, including neurology, genetics, etc. For example, in the twin study by Mosing et al., there were 5440 twin pairs, and 1245 single twins took part to measure the importance of genetic and environmental etiology [19]. The result shows that in agoraphobia, genetic factors accounted for a moderate proportion of the variation, and there is a strong co-aggregation in families.

Sexual characteristics may explain why females are more likely to be diagnosed with agoraphobia than men. Bekker found in clinical and community samples that females are about four times more likely than men to be diagnosed with agoraphobia and suggest that agoraphobia is due to the relationship between dependence and gender [20]. Schmitt suggests that men are stronger and more muscular than women [21]. In order of this, we may guess that men are not accessible to afraid of danger and stay alone like women. So, they are less likely to evolve mechanisms to protect themselves from exposure to the public environment.
4.2.2. Nurturing Environment Factor

Apart from the evolutionary factors, agoraphobia can be caused by a nurturing environment, but this is not related to evolution. Nurturing environment factor is being born in dangerous environments such as natural disasters and war may have a long-term impact on children's growth and development [22].

Before discussing the nurturing factor of agoraphobia, we must know the definition of attachment theory. According to Simpson et al., attachment is an essential thing in the world for our survival [23]. De & Van “Attachment theory proposes that internal working models of attachment, that is, mental representations of attachment relationships, are shaped in childhood experiences with primary caregivers.” [24]. The study by De & Van set up a study and found the relationship between parental caregiving styles and childhood anxiety [24]. This study shows that loss of attachment from their parents can cause agoraphobia in children.

Separation anxiety in childhood can exacerbate fear disorders in adulthood. When children are young, they are fearful of separating from their parents. If they are separated from their parents, they will have separation anxiety.

5. Schizophrenia

5.1. Introduction

Schizophrenia can be regarded as a relatively common mental illness, with a high heritability rate. Its most typical characteristics are fantasy, paranoia and emotions incompatible with social environment. So far, there have been many psychology-related experiments, neurophysiology and other experts have gathered a lot of evidence to support the hypothesis of the genetic influence and evolutionary perspective of psychology with schizophrenia. Among them, HapMap analysis can be used as the evidence to explain.

5.2. HapMap Analysis

HapMap (short for "haplotype map") refers to the International HapMap Project, which to “relate variations in human DNA sequences with genes associated with health” [25]. HapMap describes “common patterns of genetic variation among people”. For schizophrenia, using HapMap could find the related haplotypes and as DTNBP1 is identified as a putative schizophrenia susceptibility gene, HapMap analyzed the extensive genotype data for DTNBP1. Dystrobrevin-binding protein 1 (DTNBP1), or Dysbindin, is thought to be essential for regulating the glutamate energy system. It is widely expressed in the brain, with reduced levels in the brain of patients with schizophrenia, glutamatergic dysfunction might contribute to the development of schizophrenia. In a study of 488 parent-offspring triplets from Bulgaria, genotyping of eight single nucleotide polymorphisms within the gene and analysis of two, three, and four marker haplotypes yielded many positive results, with six (4% of the total combinations) p < .001, which is a very small amount [17]. Therefore, it shows that the amount of DTNBP1 is a good candidate for schizophrenia risk and the severity of symptoms was decided by glutamate.

5.3. Related Disorders

Autism and schizophrenia are both neurodevelopmental disorders that share common features, including significant impairment of interpersonal relationships and cognitive dysfunction. There is an obvious commonality between schizophrenia and autism in language deficits, and both are the result of a complex interaction of genetic and environmental factors. However, the difference is that
people with autism are more inclined to have difficulty developing language skills and understanding what others say to them. People with schizophrenia are more prone to auditory hallucinations, and the most severe language changes occur at the internal conceptual level.

People with schizophrenia are also more likely to suffer from depression. Moreover, depression is the most important risk factor for patients with schizophrenia to complete suicide. For example, the “Calgary Depression Scale for Schizophrenia (CDSS) is widely used to assess depression as a distinct from negative symptoms” [26]. Antipsychotics may also be a factor in depression, as such drugs interfere with dopamine neurotransmission as a component of their activity. Dopamine is an important neurotransmitter responsible for human pleasure and reward. Therefore, if a patient with schizophrenia develops depressive symptoms, they are advised to take antidepressants at the same time. If the patient is in the acute phase of the disease, the patient should consider using atypical antipsychotics, because the depressive symptoms during this period will have adverse reactions to antipsychotics.

5.4. Diathesis-Stress Model

The neurodiathesis-stress model of schizophrenia “proposes that stress, through its effect on cortisol, acts on preexisting vulnerabilities that trigger and/or worsen schizophrenia symptoms” [27]. According to Walker and Diforio's neurodiathesis-stress model, “the constitution of schizophrenia is an abnormality of dopamine (DA) neurotransmission. Expression of this diathesis depends on stress exposure, and in particular, the neuroendocrine pathway of DA transmission is affected by cortisol release mediated by the hypothalamic-pituitary-adrenal (HPA) axis. Elevated cortisol levels associated with stress exacerbate abnormalities in neurotransmission of DA, which underlie susceptibility to schizophrenia, leading to the onset of the disease” [27]. This means elevated cortisol can lead to worsening of the disease, and antipsychotic drugs can reduce cortisol levels to alleviate the disease. Stressors are also an important factor. People with schizophrenia have more negative emotions and events than ordinary people, and this also includes their living environment, such as poverty or social marginalization. It is also because of the appearance of a stressor or sudden aggregation that causes cortisol to rise.

5.5. GWAS

GWAS, or genome-wide Association Studies, is “a method used in genetic studies to associate specific genetic variations with specific diseases” [4]. The method involves “scanning genomes from many different people and looking for genetic markers that can be used to predict the presence of disease. Once such genetic markers are identified, they can be used to understand how genes cause disease and to develop better prevention and treatment strategies” [4]. Most people because of this new research will probably focus on the benefits it brings to people, and indeed, it has many successes. For example, it has comprehensive genetic information and many tests to detect disease susceptibility genes, genetic risks and so on.

Although GWAS have made significant contributions to the study of schizophrenia and tested a very large number of genes, they still carry the risk of false positive results with other types of studies. “They drowned out the signals from real risk variants with statistical noise from lots of non-existent markers”. GWAS has many limitations, such as inability to fully account for genetic/familial risk of common diseases; It is impossible to assess rare genetic variants [22]. However, for schizophrenia, there are no common high-impact variants. It is also important to note that current GWAS testers are largely of Western European ancestry, which expanded from 16,125 to 79,366, resulting in very little understanding of genetic risk variants in non-European populations [25]. Furthermore, these differences mean that mixing people of different ancestry in a disease
cohort can severely confound the identification of causative genes—in some cases, this mixing can greatly increase the risk of false-positive results [28]. Gwas used many Europeans as participants because they could potentially be used to detect risk variants in Africans. However, the fact is that European populations are only a subset of human genetic variations, and humans vary in allele frequency, biological fitness, and other characteristics that affect the detectability and significance of risk variations [29]. Moreover, some studies have shown that no single subset of the population can completely replace or validate variations in underlying diseases in all populations.

6. Autism

6.1. Introduction

Autism Spectrum Disorders (ASD) is a neuro-developmental disorder that has key symptoms including impairment of communication and social interaction as well as limited and stereotyped behaviors and interests. Autism is classified as a pervasive developmental disorder (PDD). The broad definition of autism based on its core symptoms can be categorized into different types, which include classic autism, Asperger's syndrome, atypical autism, and other generalized developmental disorders to be classified.

The most recent data on the epidemiology of autism show that autism affects 1 percent of men in the United States, significantly more than women, and one in 70 boys may have an autism spectrum disorder. In China, the number of severe autism patients is estimated to be about 650,000, and the number of mild autism patients is about 5 million. If there is no targeted treatment or intervention, these developmental disorders will not only seriously affect the lives of autistic individuals, but also greatly increase the burden on their family and society. In the last decade, research into the treatment of autism has received increasing attention around the world.

6.2. A Genetic Protective Mechanism That Contributes to Survival

Studies dating back to the late 1970s found that viral infections in pregnant women increased the risk of autism in their offspring. Several subsequent studies have suggested a possible link between infection during pregnancy and the development of autism [30]. Known related pathogens include rubella virus, cytomegalovirus, varicella zoster virus, herpes simplex virus, and treponema. It is speculated that antibodies produced by these pathogens, which enter the fetus from the placenta, have cross-immune reactions with the developing nervous system of the fetus, interfering with the normal development of the nervous system, thus leading to the occurrence of autism.

Although autism is still a difficult problem to solve in today's society, it is believed that these individuals gave their community a powerful advantage in the early days of human society.

For example, individuals with autism are often accompanied by enhanced vision, smell and taste, as well as exceptional memory. People with Asperger's, for example, often have a high observation ability for details, a superior understanding of systems, and an exceptional focus. For example, a superior observation for detail can be used to identify different plants and animals, and a superior understanding of systems can be useful for hunting. This made some of them the chief experts in society at the time, filling positions that no one else could fit.

6.3. The Triad and Non-triad Symptoms

Individuals with autism showed obvious inadequate response to the social stimulus of spoken language. The automatic perceptual detection of vowel sound in autistic children is more delayed than that in normal children [31]. Despite perceptual gaps across multiple dimensions of auditory stimulation, individuals with autism showed stronger responses to tones.
Although the genetic function that leads to poor dynamic vision might have been a disadvantage for our hunter-gatherer ancestors, the superior ability to remember and recognize static objects would have greatly helped our ancestors.

6.4. Mutation-Selection Balance

6.4.1. An Evolutionary Paradox

As well as possessing some superior folk physics, autism, like other psychological disorders, also possesses many abnormalities (social, communication, imagination). So, if natural selection is so powerful at optimizing complex adaptations, why does it seem unable to eliminate mental disorders that tend to lead to common, harmful, heritable disorders? This brings us to the three main explanations for the apparent paradox of evolutionary genetics: (1) ancestral genes were not harmful in our ancestors (2) mutation-selection balance (3) Psychological disorders caused by mutations that inevitably contribute to gene homeostasis [32]. All three models can explain the persistent genetic variation of certain traits under certain conditions from an evolutionary psychological perspective. By introducing these three concepts into the case of autism, we can explain and analyze the high heritability of autism from the perspective of evolutionary psychology.

6.4.2. Introduction to Mutation-Selection Balance

An organism's genome is constantly being changed by mutations. Of these, the vast majority are harmful to the organism or its descendants because they reduce the viability or fertility of the carrier. So every generation, natural selection reduces the frequency of these harmful mutations. The opposing processes of mutation and selection balance each other so that the frequency in the population of deleterious mutations is maintained at an equilibrium value determined by selection intensity and mutation frequency.

For autism, although there are many negative symptoms (severe loss of thinking function due to the loss of brain thinking function caused by genetic mutation, and congenital loss of logical thinking ability such as summary, induction, analysis and judgment), its symptoms that beyond ordinary people, such as: In terms of visual recognition of static objects, excellent memory, good mechanical memory, musical ability, number sensitivity and so on, autism provides a strong balance in the process of evolutionary selection. And thus, selection is pushing alleles for mental disorders out of the population, while the continuous process of mutation keeps introducing them.

7. Conclusion

In conclusion, one main takeaway of the evolutionary approach to understanding mental illnesses refers to the mechanism of natural selection. It shapes evolution by maximizing reproductive benefits, which raises the paradox between fitness genes and later disorders.

Many psychological issues we consider ‘disorders’ today, such as the four mentioned above, are products of selection balance and trade-offs. For example, postpartum depression symptoms could be adaptive from the offspring’s fitness’ point of view by being associated with longer IBIs [11]. Agoraphobia can be understood as a mechanism to protect oneself from dangerous events in the past, which is used to be conducive to reproduction. Also, according to the research papers, females are more likely to diagnose with agoraphobia; this may be due to females being weaker than males because males are muscular. Schizophrenia is either an advantageous evolutionary condition or a disadvantageous byproduct of normal brain evolution. Lastly, considering the evolutionary approach and the mutation-selection balance, psychologists today will be able to analyze the solid genetic ability of autism from a more comprehensive and professional perspective and why autism,
as a psychological disease, can exist in the gene pool for a long time. The superior features can explain the genetic factors that contribute to the evolution of autism. Overall, still, it is clear and unpersuadable that evolutionary psychology is essential to understanding abnormal behaviors and psychological illnesses.

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References


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