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## DISCOVERING THE BIOLOGICAL BASIS OF ABNORMAL BEHAVIOUR OF NEUROTIC DISORDERS BY ACADEMICIANS IN THE LIGHT OF NEP 2020 OF INDIA MANDATE

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### ABSTRACT

NEP advocates for multidimensional exploration of concepts, prompting scholars to delve into the biological roots of neurotic disorders and their behavioral ramifications, thus inspiring empirical research by experts. Neurotic disorders, characterized by distressing emotional states and maladaptive coping mechanisms, pose significant challenges to mental health. This paper explores the historical and contemporary perspectives on the biological foundations of neurotic disorders, with a focus on abnormal behavior within this framework. Early proponents like Haller and Kraepelin emphasized brain pathology's role, paving the way for modern biological viewpoints. Behavior genetics research reveals hereditary influences, while biochemical imbalances, notably dopamine dysregulation, contribute to neurotic disorders. Biophysical therapies such as electroconvulsive therapy underscore the biological underpinnings of mental illnesses. Examining specific disorders like phobia, panic disorder, generalized anxiety disorder (GAD), and obsessive-compulsive disorder (OCD), genetic predispositions, autonomic nervous system dysregulation, and neurological abnormalities emerge as key factors. Despite criticisms of oversimplification and subjectivity, recent research affirms the genetic basis of mental disorders, supporting the relevance of the biological model. This paper advocates for a nuanced understanding of neurotic disorders' biological underpinnings, integrating historical insights with contemporary advancements to inform effective diagnosis and treatment strategies.

**Keywords:** *Neurotic disorders, biological basis, Abnormal behavior, Genetics, NEP 2020.*

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## INTRODUCTION

NEP 2020 of India has clearly mentioned that academicians should try to explore various concepts from multiple angles and its application in the practical life situation. The concept of biological basis of neurotic disorders and its greater impact on behavior has been explored by certain academicians and they have discovered its various applications. Therefore, researchers have tried to probe into the biological basis of behaviour of neurotic disorder and the empirical study done by experts in this field.

Neurotic disorders represent a diverse category of mental health conditions characterized by distressing emotional states, maladaptive coping mechanisms, and impaired functioning in daily life. Abnormal anxiety, illogical worries, compulsive habits, and chronically low moods are just a few of the symptoms that are present in these diseases. Across all age categories and demographics, millions of people suffer from neurotic disorders, which are among the most common mental health diseases in the world (APA, 2013).

In the past, psychological disorders characterized by excessive anxiety, tension, and internal conflicts were referred to as "neurosis" when there was no significant impairment to reality testing or loss of contact with reality (Freud, 1894). The understanding and categorization of associated diseases continue to be influenced by the idea of neurosis, despite the fact that it has changed throughout time and is no longer officially acknowledged in diagnostic classification systems like the DSM (APA, 2013).

Subjective distress and functional impairment that are out of proportion to the intensity of the stressors or triggering events that cause the condition are two characteristics that distinguish neurotic disorders (Clark, 2009). People who suffer from neurotic disorders frequently feel overwhelmed by daily obstacles have severe emotional anguish, and chronic concern. Significant disruptions to work, relationships, and general quality of life can result from these symptoms (Kessler et al., 2005).

Generalized anxiety disorder (GAD), panic disorder, obsessive-compulsive disorder (OCD), and several phobic disorders are among the wide range of illnesses that fall under the umbrella of neurotic disorders (Kupfer et al., 2013). Excessive anxiety and maladaptive coping mechanisms

are common characteristics of all disorders, even though each has a distinct clinical presentation and set of diagnostic standards.

### **Abnormal Behavior in Neurotic Disorders**

Abnormal behavior, often known as psychopathology or mental illness, is defined as patterns of thoughts, feelings, and behaviors that differ from society standards and produce severe discomfort or impairment in functioning (American Psychiatric Association, 2013). This divergence from normal functioning can take many forms, including disturbances in mood, cognition, perception, and social relationships.

Cultural, societal, and historical aspects all impact our interpretation of aberrant conduct; what is abnormal in one setting may be regarded normal in another. Furthermore, anomalous conduct exists on a continuum, ranging from moderate to severe, and can be either transitory or chronic (Nolen-Hoeksema and Rector, 2015).

Now, let's explore abnormal behavior specifically within the context of neurotic disorders.

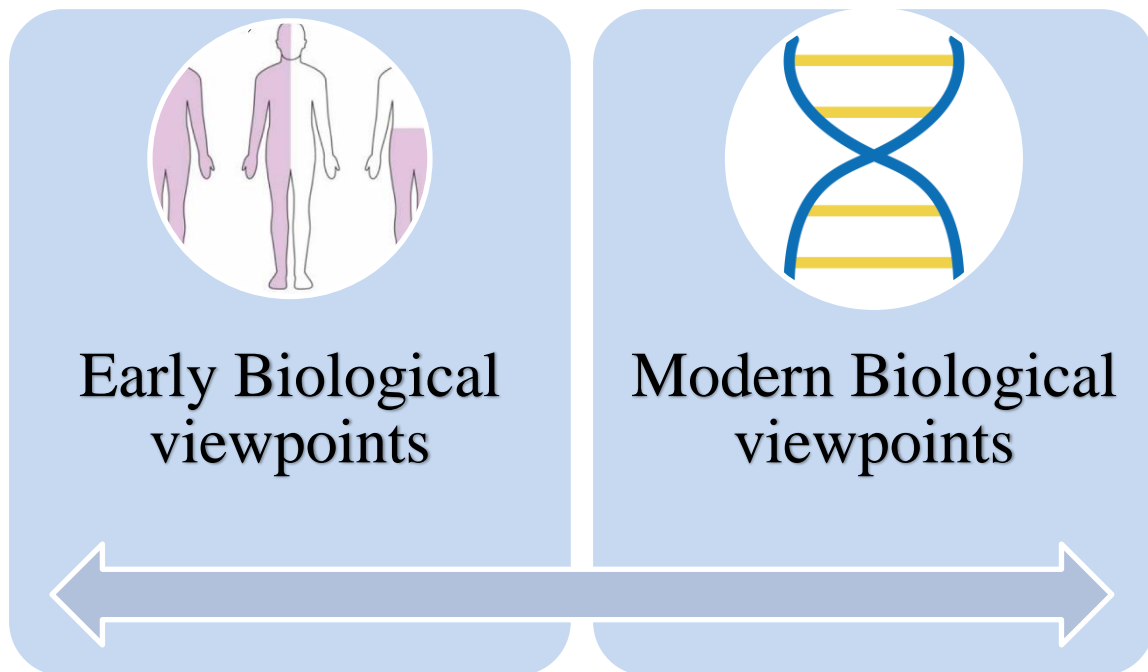
Abnormal behavior, a hallmark of neurotic disorders, encompasses a range of maladaptive coping strategies and emotional disturbances that significantly impair daily functioning (American Psychiatric Association, 2013). Within neurotic disorders like generalized anxiety disorder (GAD), obsessive-compulsive disorder (OCD), and panic disorder, individuals often exhibit excessive worry, avoidance behaviors, compulsive rituals, and emotional dysregulation (APA, 2013; Kring & Sloan, 2010). These behaviors reflect attempts to manage distressing symptoms but can exacerbate the underlying condition. For instance, individuals with OCD may engage in repetitive rituals to alleviate intrusive thoughts, while those with panic disorder may experience sudden, intense fear accompanied by physical symptoms (APA, 2013).

Understanding the biological basis of neurotic disorders is crucial for accurate diagnosis, personalized treatment, and the development of innovative therapies. By uncovering genetic, biochemical, and neurological factors underlying these conditions, clinicians can tailor interventions, reduce stigma, and improve outcomes for individuals grappling with neurotic disorders.

## BIOLOGICAL VIEWPOINTS ON ABNORMAL BEHAVIOR

In biological theories or models, abnormal behavior is explained in terms of bio-physical processes. It is sometimes also called medical model or disease model. In fact, abnormal behavior has been explained by this model at the following two levels:

(1) According to this model at the first level, abnormal behavior is considered to be a disease of the central nervous system which is either congenital (inherited) or arises from some kind of brain pathology. At this level, no importance is given to the role of any psychological factor or psychosocial environment.



(2) At the second level, which is comparatively more recent, abnormal behavior is said to be caused by imbalanced biochemical processes. When such processes within a person's body become unbalanced due to some reason, then the person becomes a victim of mental disorder.

Now we will consider the contributions of this theory a little seriously. We can broadly understand the nature of the medical model by dividing it into the following two parts:

1. **Early biological viewpoints**
2. **Modern biological viewpoints**

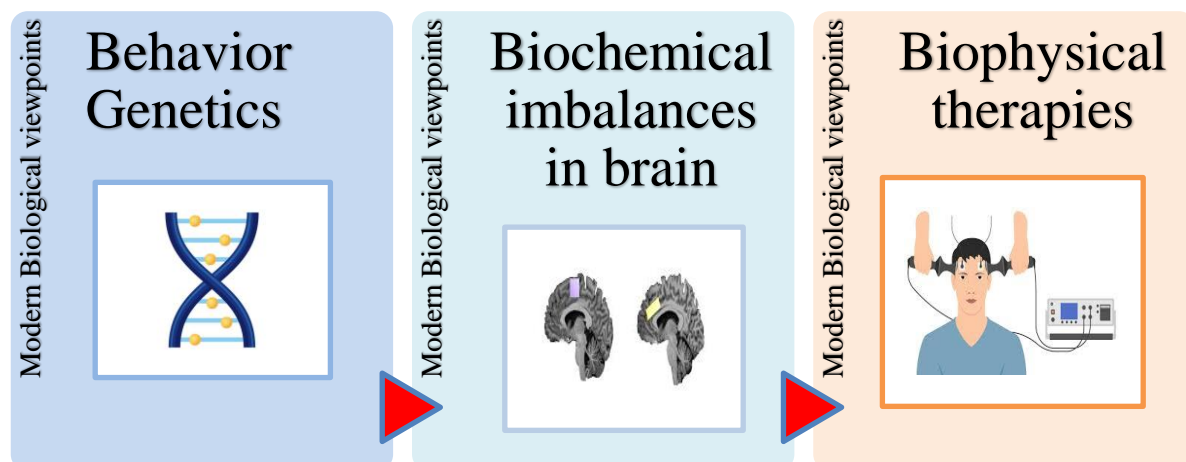
(1) **Early biological viewpoints**

Since the beginning, some scientists have been of the opinion that the cause of mental disorder or abnormal behavior is as physical as physical disease, that is, abnormal behavior is a kind of disease which affects the brain, arises due to deformity. Early proponents of this idea were **Haller and Griesinger**, who claimed in 1845 that all mental illnesses could be explained in terms of brain pathology. During the same period, mental illness like 'General Paresis' was observed by scientists, the findings of which confirmed the above ideology. In the beginning of this disease, the patient experiences weakness in arms and legs. Then delusion develops, eccentricity occurs in behavior, and then gradually paralysis occurs in the entire body and the patient dies. Some pathologies were found in the brain of such patients. Then, evidence of some brain disorders in senile mental disorders and cerebral arteriosclerosis were also found (Alzheimer, 1907), which also provides evidence of the organic basis of mental diseases.

Later, the contributions of the German psychiatrist Kraepelin played an important role in the development of the biological model. He wrote a famous textbook in 1883 that not only emphasized the role of brain pathology in the origin of mental diseases, but he also made many related contributions that helped the biological model or ideology flourish. . The most important of these contributions was his classification system of mental illnesses, which is the basis of DSM-IV. His idea was that certain symptom patterns are so persistent in an individual that they could be called a type of mental illness. His philosophy that each type of mental illness is distinct and different from the others, and that each disease is fairly predetermined and predictable, has greatly supported the medical model.

## (2) Modern Biological viewpoints

Modern research has made it clear that mental illness is not solely caused by brain pathology but by various biological factors. These factors have been studied across three main categories:



**Behavior Genetics:** Behavior Genetics in behavior studies the individual differences that occur due to genetic or hereditary factors. The entire genetic makeup of a person is made up of inherited genes. It is called genotype and all the observable characteristics of a person are called Phenotypes. On the basis of this difference between genotype and phenotype, we can easily say that different types of abnormal behavior are called deformities in genotype. There are 46 chromosomes in each zygote and the characteristics of the parents are transmitted to their offspring through thousands of genes. The study by Neil & Oltmanns (1980) highlights genetic

transmission as a crucial aspect of behavior genetics, emphasizing its role in understanding the inheritance of traits related to behavior. Paykel's study (1982) underscored the significant role of genetic factors in predisposing individuals to depression, highlighting the importance of behavior genetics in understanding the disorder. Cloninger et al. (1986) have shown that the basis of these mental diseases is hereditary, in other words, such diseases are more common in those people whose parents or family members have it. To show the importance of genetic factors in mental diseases, two methods have been proposed - family method and twin study method.

In the family method, different members of a family are matched together and a detailed study is done to find out whether if one member has a severe mental illness, then there is a possibility of the same occurring in the other one too. In the twin study method, identical twin children are compared with each other and with fraternal twin children to determine whether one child of the pair has a certain mental illness, and the other also has it. Are you seeing symptoms? Identical twins have 100% identical genetics, while fraternal twins have only 50% identical genetics. If the occurrence of a mental illness is genetic in nature, then the concordance rate in identical twins should be higher than the concordance rate in fraternal twins. This supports the view that genetic factors play an important role in the origin of physical diseases.

**Biochemical imbalances in the brain:** The medical model has also been supported by modern research that when biochemical imbalance occurs in the brain due to any reason, it also leads to mental illness. This type of imbalance occurs due to many reasons, the main ones being high fever, brain inflammation, stress, neurotransmitters etc. We explain this claim with a popular example as follows:

There is a popular hypothesis about schizophrenia which is called 'Dopamine hypothesis'. Dopamine is a type of neurotransmitter whose main function is to help in neural transmission. According to the dopamine hypothesis, when dopamine activities in the brain increase excessively, it causes psychosis. This fact has been indirectly supported by many studies, of which the study conducted by Kapur & Remington (1996) and Heinz et al. (1994) are more

famous. In their studies, schizophrenic patients were given a drug that reduced dopamine activities in the brain. With continuous use of this medicine, it was seen that dopamine activities in the brain were blocked, which reduced the symptoms of schizophrenia. Such results clearly indicate that dopamine is associated with the disease of schizophrenia. In fact, dopamine creates biochemical imbalance in the patient's brain, which causes brain degeneration and symptoms of psychosis start appearing.

**Biophysical therapy:** The biological model is also supported by the fact that a variety of biological interventions, beginning in the 1930s, have reduced the symptoms of mental illness. From this it follows that mental diseases definitely have a biological basis. Like, a popular biological therapy is electroconvulsive therapy or ECT. In this medical method, a mild electric current is passed through the patient's brain for some time. After some time it is often seen that the patient returns to normal condition. Explaining this biologically, it is said that due to electric shock, the biochemistry of the patient's brain probably gets poisoned and then after some time, a proper biochemical balance is established in it which motivates a person to behave normally instead of abnormal behaviour. In the 1950s, many types of medicines were developed which were also used to successfully treat mental diseases. For example, phenothiazines are drugs that significantly reduce confused thinking and intrusive thinking. Taking this drug causes such drastic changes in brain biochemistry that the patient generally taking this drug causes such a rapid change in brain biochemistry that the patient rapidly returns to normalcy. Such observations also reveal the fact that mental diseases have a biological basis.

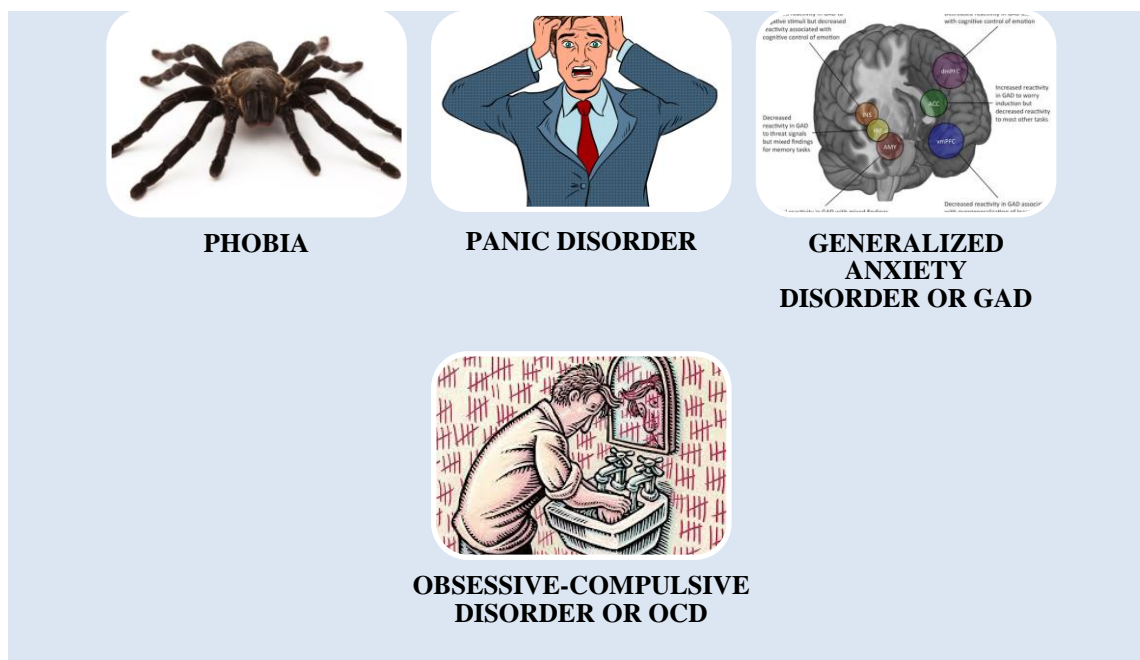
## **BIOLOGICAL THEORIES AND FACTORS OF NEUROTIC DISORDERS**

In abnormal psychology, maximum attention has been given to anxiety and the mental disorders arising from it. Anxiety refers to the unpleasant feeling of fear and apprehension. Such anxiety gives rise to a variety of mental disorders, which were earlier placed under a general diagnostic category, namely 'neurosis' or 'psychoneurosis'. Keep in mind that the term 'neuropathy' was first used by the English scientist William Cullen in his System of Nosology published in 1769 for disordered sensations of the nervous system (Kerr et al., 2008). Later, Freud and his colleagues used the term neurosis to describe mental illnesses caused by anxiety, and even today it is used by common people in almost the same sense. Many types of mental disorders were included in neurosis, which included anxiety neurosis, phobia neurosis, conversion neurosis, hypochondriacal neurosis, obsessive-compulsive neurosis etc. All of them had different symptoms or clinical nature. Whatever be the type of neurosis, two main characteristics of neurotic life-style have been described - neurotic nucleus and neurotic paradox.

The neurotic core refers to a faulty assessment of reality and the tendency to move away from stress instead of dealing with it, and the neurotic paradox refers to maintaining this particular type of lifestyle despite its maladaptive and self-destructive nature.

Psychiatrists invalidated the diagnostic category 'neurosis' in DSM because it would not be appropriate to group mental disorders with so many different characteristics or symptoms into one category because their different symptoms and causes could not be scientifically studied. In DSM, anxiety disorder refers to a disorder in which the amount of unrealistic worry and irrational fear in the client or patient is so high that his normal life behavior becomes maladapted and in this the person expresses his concerns very clearly.

The following main types of anxiety disorders have been described in DSM:



## (1) PHOBIA

**Phobia** is a very common anxiety disorder in which a person has a persistent and disproportionate amount of fear of a specific object or situation that actually poses little or no threat to the person. For example, a person who has developed a phobia of spiders cannot enter a room where a spider is present, which is an animal that poses no threat to a person. But when this fear arises, it deviate the normal behavior of the person.

**Biological Theories or Factors:** Even when exposed to the same stressful situation, some people develop phobias while others do not. The reason given for this is that in those who develop phobia, there is some biological malfunctioning (diathesis) which causes phobia in them after a stressful situation. From the research done in this area, it became clear that biological factors related to the following two areas are important:



- (i) **Autonomic nervous system:** Some studies have found evidence that phobias are more likely to occur in people whose autonomic nervous system is overstimulated by a variety of environmental stimuli. Lacey (1967) called this type of autonomic nervous system autonomic lability. According to Gabbay (1992), autonomic instability is to a great extent genetically determined. Therefore, a person's heredity plays a definite role in the occurrence of phobia.
- (ii) **Genetic factors:** There have been some studies which provide clear evidence that the possibility of developing phobia is higher in those people whose parents and close relatives have already had this type of disease. For example, according to Harris et al. (1983), direct relatives of agoraphobia patients were found to be more likely to have that disorder than direct relatives of unconscious control subjects. Noyes et al. (1986) found in their study that relatives of agoraphobia were more likely to develop panic disorder along with this disorder. Similarly, Torgersen (1983) has found in his study that the concordance rate of agoraphobia is higher in identical twin children than in fraternal twin children.

In the light of the above studies, we come to the conclusion that biological factors play a role in the origin of phobia.

## (2) PANIC DISORDER

In **Panic Disorder**, the patient has a sudden and inexplicable panic attack. When a patient has such unexpected panic attacks repeatedly, i.e. at least once or twice a week, then it is called panic disorder in DSM-IV. In this way, unexpected and frequent panic attacks occur in panic disorder.

**Biological theories or factors:** Studies have shown that Panic disorder occurs more often in those people whose family member has already had it. According to Torgersen (1983), the concordance rate of Panic deformity is much higher in identical twin children than in fraternal twin children. According to their study, if one child of a monogamous pair has panic disorder, there is a chance of it occurring in the other child that increases by 31%. But if one of the fraternal twin pair has Panic disorder, then there is almost no possibility of this disorder occurring in the other child of the pair. This clearly shows that panic disorder has a genetic basis. On the basis of their study, Charney and Heninger (1986) have stated that when the circuit of a person's brain that slows down or stops the emergency reaction is weak, if it goes away, it

increases the possibility of panic disorder occurring in it. According to Robins et al. (1986), it has also been found in patients with panic disorder that the blood flow and oxygen in some parts of their brain is more than normal.

According to the study by Ley, 1987, panic attack is related to hyperventilation or over breathing. Due to hyperventilation, the autonomic nervous system of the person gets excited due to which all the physical symptoms of panic attack occur in the patient. Some studies have also shown that when the amount of carbon dioxide in the breathing air is higher than normal, it also causes panic attacks because the higher amount of carbon dioxide is likely to cause hyper respiration.

### **(3) GENERALIZED ANXIETY DISORDER OR GAD**

**Generalized Anxiety Disorder or GAD** is another mental disorder that receives special attention in the DSM-IV. GAD is a disorder in which the patient suffers from chronic, unrealistic or excessive anxiety. This type of anxiety is traditionally called free-floating anxiety. A person suffering from GAD is always in a world of stress, anxiety and diffused uneasiness. According to DSM-IV, if a person has spent at least the last six months of his life in such a way that he has had unrealistic and excessive anxiety for most of the period, then he can definitely be called a patient of GAD. There are many clinical features or symptoms of GAD. For example, emotionally such a patient appears restless, stressed, alert, and jittery. He talks about impending dangers such as heart attack, death or loss of control, etc. He gets very upset while thinking. Cognitively, he is always expecting something bad to happen but is unable to tell what is going to happen. Many types of symptoms are seen physically also. Many emergency physical reactions have also been found in the person, the main ones being sweating, rapid heart rate, stomach upset, feeling lightheaded, hands and feet becoming very cold, etc. Such people feel tired quickly, have difficulty concentrating, exhibit irritable behavior and also complain of insomnia. Behaviorally, such people always hide themselves from others or go somewhere else to hide. Such a person finds it difficult to reach any decision and even if he takes a decision, he remains worried thinking that there must be some mistake in it.

**Biological theories or factors:** The research done so far has shown that Biological theories and factors related to Generalized Anxiety Disorder (GAD) encompass various neurobiological, genetic, and physiological aspects.

One prominent neurobiological theory suggests dysregulation within the amygdala and prefrontal cortex (PFC) circuits. The amygdala, implicated in processing threat-related information, may exhibit hyperactivity in individuals with GAD, leading to exaggerated fear responses and heightened vigilance (Roy et al., 2008). Concurrently, dysfunctional inhibition by the PFC, responsible for regulating emotional responses, may contribute to the inability to downregulate fear and worry (Etkin & Wager, 2007).

Furthermore, genetic factors play a role in the vulnerability to GAD. Family and twin studies indicate a heritable component, with estimates of heritability ranging from 30% to 40% (Hettema et al., 2001). Candidate gene studies have identified potential genetic markers associated with GAD, including variants in genes related to neurotransmitter systems such as serotonin and gamma-aminobutyric acid (GABA) (Smoller et al., 2001). Slater and Shields (1969) conducted a comparative study of seventeen pairs of identical twins and 28 sets of fraternal twins and found that 49% of identical twins had generalized anxiety disorder while only 4% of fraternal twins had generalized anxiety disorder. So, according to their study, GAD is found in identical twins more than that in fraternal twins. Torgersen (1983) found similar concordance rates for GAD in identical twins in his study. In the light of these studies, it can be said that GAD also has a hereditary basis.

Additionally, dysregulation of the hypothalamic-pituitary-adrenal (HPA) axis, the body's stress response system, is implicated in GAD. Individuals with GAD may exhibit alterations in cortisol levels, with some studies suggesting heightened baseline cortisol levels and blunted cortisol responses to stressors (Ströhle et al., 2017). These biological theories and factors provide insight into the complex interplay of neurobiological, genetic, and physiological processes underlying the development and maintenance of GAD.

#### **(4) OBSESSIVE-COMPULSIVE DISORDER OR OCD**

**Obsessive-Compulsive Disorder or OCD:** Initially, clinical psychologists and psychiatrists believed that obsession and compulsion were two independent diseases. But with later studies it became clear that these two are not independent diseases but are two aspects of the same disorder. Sometimes one aspect is dominant in the patient and sometimes the other aspect is dominant and sometimes it has been seen that both these aspects are seen in equal amounts in a balanced manner in the same patient. To understand both these aspects of this disease, we will describe it separately in the following manner-

**Obsession** is a condition in which the patient keeps repeating some illogical and inconsistent thoughts in the mind again and again, even without wanting to. The patient understands well the meaninglessness, inconsistency and illogical nature of such thoughts and also wants to get rid of them, but he remains helpless and the thoughts keep coming to his mind again and again and creating mental disturbance in him.

According to Kisker (1985), "**Obsession is a thought or thinking that is ridiculous, absurd and apparently meaningless, yet such that the patient cannot get rid of it.**"

According to Seligman & Rosenhan (1998), "**Obsessions are repetitive thoughts, images, and impulses that enter the conscious and are often abhorrent and difficult to control or eliminate.**"

By analyzing these definitions, we get the following facts about the nature of obsession-

**Obsession** is related to thoughts and images. Such thoughts or thinking are absurd and meaningless. The nature of such thoughts or thinking is repetitive and it comes to the patient's mind again and again and disturbs his mental peace. The patient has no control over not allowing such thoughts or thoughts to arise even if he wants to.

In the DSM, for such recurring thoughts or thoughts to be called obsessive thoughts, a criterion has been added that such thoughts should cause mental distress to the patient, should waste a lot of his time and should interfere with the normal functioning of the person's day-to-day life. There should be substantial disruption in work, professional work, educational work and social work. Often the thoughts and worries of obsession are related to some questions which are difficult to answer correctly as, Who is God in this world? What is the truth? Do all religions have the same origin? etc. Psychopathy may also be related to excessive doubt, indecision and procrastination, etc. The patient fails to reach any conclusion in such a situation and revisits the problem.

Compulsion is a kind of behavioral reaction in which the patient is forced to repeatedly perform an action against his will. Such actions are not only undesirable but also illogical and inconsistent. The behavior of repeatedly washing clean hands, repeatedly shaking the lock even if it is in place properly, standing on the road and noting down the numbers of passing vehicles, compulsion to steal something (Kleptomania), Compulsion to set fire (Pyromania) etc. are some examples of compulsion. Kisker (1985) has defined compulsion as follows, "When obsessions are converted into actions, they are called compulsions. People suffering from compulsions do the same thing repeatedly even though they feel that that there is no point in doing this.

**Biological theories or factors:** The research done so far has shown a clear trend that some biological factors play a decisive role in the origin of OCD. McKeon and Murray (1987) have found in their study that anxiety disorders are more prevalent in relatives of OCD patients and Lenane et al. (1990) have found in their study that about 30% of the relatives had OCD. According to Jenike's study (1986), encephalitis, head injuries and brain tumors have also been found to be important in the development of OCD.

Biomedical researchers have presented the claim on the basis of their research that OCD is a brain disease and the following three types of evidence have been discussed in its support:

**(a) Neurological signs** – There is some evidence that OCD develops from brain trauma.

Neurological examination of such patients has revealed evidence of several abnormalities such as poor motor coordination, involuntary jerks, and poor visual-motor performance. Hollander et al. (1992) have confirmed this in their research. Rapoport (1990) has mentioned a case in support of this – an eight-year-old child named Jacob was playing ball in the field. Suddenly he fell and became unconscious with brain hemorrhage. After this, he successfully underwent surgical operation on his brain and later he developed a compulsion in which he used to count everything 'seven' times, name everything 'seven' times, and on the sound of 'seven' he would use to start eating, etc. It is also clear from this case that symptoms of OCD develop due to brain injury.

Some evidence has also been found that shows that OCD is also related to epilepsy. Rapoport (1990) based on his study stated that when there was an outbreak of epilepsy in Europe between 1916 and 1918, symptoms of OCD also developed in such patients.

**(b) Brain scan abnormalities:** Brain scan studies have revealed that OCD patients have many parts of the brain in which over-activity is found. For example, activity of the 'cortical-striatal-thalamic' region is found to be higher in OCD patients. This area of the brain is concerned with reducing unnecessary repetition of inconsistent information and behavior. Naturally, when the said area of the brain becomes overactive, the patient starts having repetitive thoughts about inconsistent information and unnecessary repetition of related behavior, which is a major symptom of OCD. Robinson et al. (1995) and Baxter et al. (1999) have supported the above explanation with their respective studies.

**(c) Evidence from drug therapy:** Experts in this field, especially psychiatrists, are of the opinion that when a disease is reduced or almost eliminated by a particular drug, it automatically leads to the conclusion that It turns out that the disease has some biological

basis. For example, a special drug called clomipramine has proven to be very effective in the treatment of OCD because it reduces the symptoms of OCD significantly. Clomipramine is a type of antidepressant drug. It became clear that many biological factors are important in the origin of OCD.

## CONCLUSION

Biological model whose essence is that mental illness is an illness or disease of the body, like physical illness. This model clearly has some advantages and it has also received some criticisms which need to be considered here. Some advantages of the model are as follows:

The basis of the medical model is mature science. As a result, all its hypotheses like dopamine hypothesis, heredity, central nervous system etc. are testable.

Since the facts of the medical model are objective and the hypotheses are verifiable, hence it is trusted a lot.

The methodology of this model is well defined and is in a definite sequence which explains mental illness. The sequence of mechanisms of this model is syndrome, etiology and treatment. In other words, in this model, first a group of symptoms of the disease called syndrome is created and then its possible cause is found out and its treatment is done. Such a clear sequence to understand the nature of mental illness is not seen in any other model.

Despite these advantages, some criticisms have been made of this model, which are as follows:

- (i) When abnormal behavior is explained by the medical model, it is not possible to independently assess the cause of the symptoms of the disease. Some symptoms or behavior are categorized and given the name of a particular mental disease and then that name itself is said to be the cause of that disease. This means that the circular reasoning in the medical model is flawed. This can be explained with an example, a person who separates himself from the environment and also experiences hallucinations. On the basis of symptoms, he is diagnosed as a patient of schizophrenia, but when it is asked why the patient is having hallucinations and why he does he keeps himself away from the normal environment, then it is said that it is because of this. The behavior or symptoms are called schizophrenia. This is clearly an example of circular reasoning.
- (ii) The second criticism of the medical model is that the symptoms of physical disease are objective but the symptoms of mental disease are subjective. This criticism is related to the statement of Thomas Szasz in which it has been said that the mental

symptoms of the patient are the communications made by him about himself. When we call such reported communication symptoms, we say so only after taking a decision in a social and cultural context. For example, if a patient says that he is the Prime Minister of India, the observer will probably think that the patient is showing symptoms of paranoid schizophrenia. Even in this situation, the diagnosis will depend on how much the observer believes in the patient's statement. Again, this has a lot of scope for subjectivity.

- (iii) According to Milton and Wahler (1969), mental illness has neither specific symptoms nor a specific etiology. Whatever has been known so far about the causes of mental illness is quite limited. In such a situation, explaining mental illness through the medical model would naturally prove to be inappropriate.
- (iv) The claim of this model that personality and mental disorders are genetic is only a half-truth. Studies have made it clear that personality is partly genetic and at the same time mental illness is also partly genetic because many factors which are not genetic also have a good impact on the origin of mental diseases.

Despite these criticisms, the popularity of the medical model is considerable because the following three things have become clear from the research done in the last 20 years:

- (i) Some mental diseases are genetic.
- (ii) There are some mental diseases which are caused by biochemical disturbances and some mental diseases are neuroanatomical.
- (iii) There are definitely some medicines which cause changes in the emotions and mood of a person.

The biological perspective of abnormal psychology is clearly explaining that mental disorders are linked with chemical imbalances, structural abnormalities in the brain, neurotransmitters, as well as genetics. Biological elements such as genetics, brain structures, the endocrine system, and neurotransmitters all play an important role in the development of mental disorders. As explained by the biopsychosocial model, none of these biological factors, on their own can generate a mental disorder, but combined with environmental, psychological, and social or cultural forces, they *interact* to shape how we learn, how our brains function, which genes are expressed or turned off, and the biological foundation of a person's diathesis (or vulnerability) to developing a disorder.

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## REFERENCE

- Alzheimer, A. (1907). About a peculiar disease of the cerebral cortex. *Allgemeine Zeitschrift fur Psychiatrie und Psychisch-Gerichtlich Medicin*, 64, 146-148.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.
- Baxter, L. R., Schwartz, J. M., Mazziotta, J. C., Phelps, M. E., Pahl, J. J., Guze, B. H., ... & Selin, C. E. (1990). Cerebral glucose metabolic rates in nondepressed patients with obsessive-compulsive disorder. *The American Journal of Psychiatry*, 147(10), 1313-1314.
- Charney, D. S., & Heninger, G. R. (1986). Noradrenergic function in panic anxiety: Effects of yohimbine in healthy subjects and patients with agoraphobia and panic disorder. *Archives of General Psychiatry*, 43(10), 1042-1047.
- Clark, D. A. (2009). Cognitive theory and therapy of anxiety and depression: Convergence with neurobiological findings. *Trends in Cognitive Sciences*, 13(6), 201-206.
- Cloninger, C. R., Sigvardsson, S., & Bohman, M. (1986). Childhood personality predicts alcohol abuse in young adults. *Alcoholism: Clinical and Experimental Research*, 10(2), 150-156.
- Etkin, A., & Wager, T. D. (2007). Functional neuroimaging of anxiety: A meta-analysis of emotional processing in PTSD, social anxiety disorder, and specific phobia. *The American Journal of Psychiatry*, 164(10), 1476–1488.
- Freud, S. (1894). The neuro-psychoses of defence. In J. Strachey (Ed. & Trans.), *The standard edition of the complete psychological works of Sigmund Freud* (Vol. 3, pp. 41-61). London, UK: Hogarth Press.
- Gabbay, J. (1992). The health of the nation. *BMJ (Clinical research ed.)*, 305(6846), 129–130. <https://doi.org/10.1136/bmj.305.6846.129>
- Griesinger, W. (2011). *Patología y terapéutica de las enfermedades mentales* (1845) [Pathology and therapeutics of mental disorders (1845)]. *Vertex* (Buenos Aires, Argentina), 22(95), 74–79.
- Harris, J. E., & Brown, G. W. (1983). Psychosocial influences on the initiation and course of depressive illness: Findings from an epidemiological survey. *Journal of Affective Disorders*, 5(3), 319-332.
- Heinz, A., Knable, M. B., Coppola, R., Gorey, J., & Jones, D. W. (1994). Serotonin transporter availability correlates with the serotonin transporter binding density in limbic and paralimbic cortex regions. *Biological Psychiatry*, 36(4), 215-223.
- Hettema, J. M., Neale, M. C., & Kendler, K. S. (2001). A review and meta-analysis of the genetic epidemiology of anxiety disorders. *The American Journal of Psychiatry*, 158(10), 1568–1578.



- Hollander, E., & Greenwald, S. (1992). Behavioral and autonomic responses to cholecystokinin tetrapeptide in obsessive-compulsive disorder. *Archives of General Psychiatry*, 49(7), 559-564.
- Jenike, M. A., Baer, L., & Minichiello, W. E. (1990). *Obsessive-compulsive disorders: Theory and management*. United Kingdom: Year Book Medical Publishers.
- Kapur, S., & Remington, G. (1996). Dopamine D(2) receptors and their role in atypical antipsychotic action: Still necessary and may even be sufficient. *Biological Psychiatry*, 39(4), 135-146.
- Kerr, C. E., Milne, I., & Kaptchuk, T. J. (2008). William Cullen and a missing mind-body link in the early history of placebos. *Journal of the Royal Society of Medicine*, 101(2), 89–92. <https://doi.org/10.1258/jrsm.2007.071005>
- Kessler, R. C., Chiu, W. T., Demler, O., Merikangas, K. R., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62(6), 617-627.
- Kisker, K. P. (1985). The psychobiology of obsessive-compulsive disorder: What's new. *Journal of Anxiety Disorders*, 4(2), 129-139.
- Kraepelin, E. (1883). *Compendium der Psychiatrie*. Leipzig: Abel.
- Kring, A. M., & Sloan, D. M. (2010). *Emotion regulation and psychopathology: A transdiagnostic approach to etiology and treatment*. New York, NY: Guilford Press.
- Kupfer, D. J., Kuhl, E. A., & Regier, D. A. (2013). DSM-5 – The future arrived. *JAMA Psychiatry*, 70(5), 523-524.
- Lacey, J. I., & Lacey, B. C. (1958). Verification and extension of the principle of autonomic response-stereotypy. *The American Journal of Psychology*, 71(1), 50–73. <https://doi.org/10.2307/1419197>
- Lenane, M. C., & Swedo, S. E. (1990). Obsessive-compulsive symptoms. In *The American Journal of Psychiatry*, 147(9), 1071-1077.
- Ley, R. (1988). Panic attacks during relaxation and relaxation-induced anxiety: A hyperventilation interpretation. *Journal of Behavior Therapy and Experimental Psychiatry*, 19(4), 253-259. [https://doi.org/10.1016/0005-7916\(88\)90054-7](https://doi.org/10.1016/0005-7916(88)90054-7)
- McKeon, J., & Murray, B. (1987). Genetic and environmental influences on obsessive-compulsive disorder. *The British Journal of Psychiatry*, 150(3), 303-308.
- Milton, O., & Wahler, R. G. (Eds.). (1969). *Behavior disorders, perspectives and trends*. New York, NY: Lippincott.
- Neil, A. L., & Oltmanns, T. F. (1980). Biological factors in schizophrenia: A review of the literature. *Clinical Psychology Review*, 1(3), 297-324.
- Nolen-Hoeksema, S., & Rector, N. A. (2015). *Abnormal psychology* (6th ed.). New York, NY: McGraw-Hill Education. Top of Form
- Noyes, R., Jr, Crowe, R. R., Harris, E. L., Hamra, B. J., McChesney, C. M., & Chaudhry, D. R. (1986). Relationship between panic disorder and agoraphobia: A family study. *Archives of General Psychiatry*, 43(3), 227–232. <https://doi.org/10.1001/archpsyc.1986.01800030037004>
- Paykel, E. S. (1982). Depression: A biopsychosocial disorder. In *Psychological Medicine Monograph Supplement*, 1(3), 1-4.
- Rapoport, J. L. (1990). Obsessive-compulsive disorder in children and adolescents. *JAMA*.
- Robinson, D., Caleo, S., Allasia, S., Zucca, C., Pittoni, R., & Tarenzi, L. (1995). Functional neuroimaging in obsessive-compulsive disorder: A pilot study. *European Journal of Nuclear Medicine*, 22(11), 1203-1210.
- Roy, A. K., Shehzad, Z., Margulies, D. S., Kelly, A. M., Uddin, L. Q., Gotimer, K., ... & Milham, M. P. (2008). Functional connectivity of the human amygdala using resting state

fMRI. *NeuroImage*, 45(2), 614–626.

- Rutter, M. (2006). *Genes and behavior: Nature-nurture interplay explained*. Oxford, UK: Blackwell Publishing.
- Seligman, M., Rosenhan, D. L., & Walker, E. F. (2000). *Abnormal Psychology* (4th ed.). New York, NY: Hardcover.
- Slater, E., & Shields, J. (1969). Affective disorders and depression. *Journal of Neurology, Neurosurgery & Psychiatry*, 32(5), 297-303.
- Smoller, J. W., Block, S. R., Young, M. M., & Scherrer, J. F. (2001). Genetics of anxiety disorders: The complex road from DSM to DNA. *Depression and Anxiety*, 14(2), 85–96.
- Ströhle, A., Holsboer, F., & Ströhle, A. (2017). Circadian rhythms and the HPA axis: Clinical implications for depression and other stress-related disorders. *Dialogues in Clinical Neuroscience*, 9(4), 465–479.
- Torgersen, S. (1983). Genetics of neuroses: Neuroticism and anxiety phobia. *Acta Psychiatrica Scandinavica*, 68(2), 163-173.