Anxiety disorders: causes and mechanisms as in biological, behavioral and psychoanalytic perspectives

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Introduction

Anxiety, which is defined as apprehension over an anticipated problem (Kring et al. 2010: 120) is a basic emotion recognized in humans as well as in non-human species. It arises as a response when an individual is exposed to a threatening situation where the individual feels fear and apprehension, coupled with the physiological reactions that prepare the body to defense or escape (Gelder et al. 1999: 103).

Anxiety has two variations as normal anxiety and pathological anxiety. In normal anxiety, the individual's attention is focused on the external threats. Anxiety is considered 'abnormal' if the threat experienced by the individual is more prolonged with increased severity. In pathological anxiety, while having the individual's attention focused on the response to threat, he may experience some clinical features such as tremor, rapid and shallow breathing, dry mouth, and often worry thoughts. The individual may fail to understand the causative factors of those (Gelder et al. 1999: 103).

For the ease of diagnosis and intervention, pathological anxiety is categorized as 'anxiety disorders', which are states with marked and persistent mental and physical symptoms of anxiety, which are not secondary to another disorder (Gelder et al. 1999: 104). Classification of anxiety disorders in DSM V include Separation Anxiety Disorder, Selective Mutism, Specific Phobia, Social Anxiety Disorder, Panic Disorder, Panic Attack (Specifier), Agoraphobia, Generalized Anxiety Disorder and Substance/Medication-Induced Anxiety Disorder (APA 2013).

Psychology is dedicated to investigate the underlying causative factors of abnormal behaviours through systematic observations and scientific researches. Anxiety is also discussed as overt behaviours. Behaviors are determined by multiple causative factors and thus anxiety disorders are also multifactorial in nature.

Different approaches in different paradigms have taken attempts to explain anxiety disorders and each approach emphasizes only some aspects of the condition. Therefore, it is required to consider several approaches together in order to have complete understanding about anxiety disorders. Hence, this article considers some common causative factors along

with the respective mechanisms for all the anxiety disorders as in the main three perspectives in psychology; biological, behavioural and psychoanalytic.

01. Biological approach

The biological approach, or the 'biomedical model' explains abnormal behaviours in terms of biological processes, structures and heredity. Factors such as anatomical, physiological, deficits or imbalances of biochemical in the body and genetic predisposition may cause anxiety (Eysenck 2004: 806).

Genetic predisposition of anxiety disorders

The genetic researches on anxiety disorders have grown extensively in recent times which suggest the genetic associations in producing anxiety disorders (Blanchard et al. 2011: 496). According to Hettema et al. (2005), some genes may elevate risk for several types of anxiety disorders, while other genes may elevate risk for a specific type of anxiety disorder. Kendler et al. (2001) provides an instance of having a family member with a phobia seems to increase the risk of developing not only a phobia but also developing other anxiety disorders (cited in Kring et al. 2010: 131). As some twin, family, and adoption studies indicate, some individuals are genetically more vulnerable than others in developing anxiety disorders (Eysenck 2004: 821). In similar studies, Andrews et al. (1990) supports a genetic predisposition for anxiety disorders in general (cited in Freberg 2009: 473). Maller and Reiss (1992) found that individuals with higher anxiety sensitivity were five times as likely as those who were low to suffer from panic attacks (Eysenck 2004: 835). Research suggest that anxiety can be caused by the 'S' allele of the 5-HTT and also the deficiency in the '$5HT_{1A}$' receptor and BDNF. A combination of all of the above factors may be necessary to lead a significant level of anxiety (Civjan 2012: 230). Hariri et al. (2002) detected a higher activation in right amygdala in anxious situations in healthy volunteers carrying S allele of genotype of the 5-HTTLPR (cited in Blanchard et al. 2011: 496). A norepinephrine transporter gene near chromosome 16 has found to be implicated in social anxiety. However, replicated results have not been found. It has also been suggested that multiple genes on several chromosomes are involved in the anxiety disorders. In some studies on PTSD, serotonin transporter gene on chromosome 5 appears to be to the onset of PTSD (Getz 2014: 121).

Role of neurotransmitters in producing anxiety disorders

In general, the individuals with anxiety disorders are with high or low levels of certain brain chemicals or the neurotransmitters (Eysenck 2004: 821), which are secreted in the synaptic cleft to pass signals from presynaptic to postsynaptic nerve endings. The cell membrane of the postsynaptic neuron contains receptors which are configured for specific neurotransmitters to fit into them. If a neurotransmitter fits into a receptor cite, the signal can be sent to the postsynaptic neuron. The postsynaptic neuron depends on integrating thousands of similar messages. Sometimes these messages are excitatory, leading to a creation of a nerve impulse or other times, the messages are inhibitory making the postsynaptic cell less likely to create a nerve impulse.

Once a presynaptic neuron has released neurotransmitters to the synaptic cleft, only some of them would combine with the postsynaptic receptors passing the message to the nerve successfully. The rest of the neurotransmitters in the synapse either would be broken down by the enzymes or taken back to the presynaptic neuron through a process called reuptake. Several key neurotransmitters such as dopamine, serotonin, norepinephrine, and gamma-amino butyric acid (GABA) have been identified to be responsible for abnormal behaviors. The given anxiety disorder is caused by either too much or too little of the particular transmitter in the synapse. Thus, poor functioning of the serotonin and GABA and high levels of norepinephrine are considered to be involved in producing anxiety disorders. Serotonin is also one of the most-studied neurotransmitter in studies to detect the causes of anxiety disorders. Selective serotonin reuptake inhibitors (SSRIs) are responsible for producing anxiety disorders (Kring et al. 2010: 144).

Research indicate that norepinephrine communicates with the sympathetic nervous system, producing states of high arousal and thus may be involved in anxiety disorders. It is also found out that GABA inhibits nerve impulses throughout most areas of the brain and may be involved in producing the anxiety disorders (Kring et al. 2010: 35). Latest research findings have discovered the processes how norepinephrine and GABA are produced in excess amounts. These neurotransmitters are synthesized in the neurons through a series of metabolic steps and each reaction is catalyzed by an enzyme. High or low amounts of neurotransmitters could result from an error in these metabolic steps. The amount of particular neurotransmitter is released in excess into the synapse and the remaining neurotransmitter could not be taken back (reuptake) to the presynaptic neuron would result in excess amounts of neurotransmitters are left behind in the synapse. Then, if a nerve impulse

causes more neurotransmitters to be released into the synapse the postsynaptic neuron would get a double dose of neurotransmitters and as a result, a new nerve impulse would be created. Sometimes the receptor sites on the postsynaptic neuron are too numerous or easily excited. If the excess amounts of neurotransmitters are released into the synapse there would be high chances of getting those sites been stimulated resulting anxiety in the individual (Kring et al. 2010: 120).

There is a mechanism to control the sensitivity of the postsynaptic neuron if the neuron is being activated quite frequently by neurotransmitters. On such circumstances the cell may retune the sensitivity of the receptors by releasing second messengers making it more difficult to create a nerve impulse. These second messengers help receptors to adjust themselves to norepinephrine and serotonin. This phenomenon is used to discover medications in treating anxiety disorders (Kring et al. 2010: 120).

Neurobiological factors of anxiety disorders: the role of the limbic system to produce anxiety disorders

The neurobiological factors are closely related to the limbic system which is the mid-level portion of the brain. It is considered as the emotional control center. The limbic system consists of parts of the thalamus, hypothalamus, amygdala and hippocampus, and other structures (Coon and Mitterer 2008: 69). The limbic system has a major role in producing anxiety, other emotions and motivated behaviors.

The thalamus is considered as the relay station for all sensory pathways except the olfactory. (Olfactory sensory input has direct inputs to the amygdala and entorhinal cortex) The thalamus receives nearly all impulses from different sensory organs before passing them on to the cerebral cortex, where conscious interpretations are taken place.

If the person is exposed to a threatening situation, thalamus sends the messages to the cerebral cortex. Since this is an emergency, thalamus sends messages not only to the cortex but also to the amygdala for immediate actions. Simultaneously, the other parts of the limbic system: hypothalamus and hippocampus also get activated through this mechanism. The pathways of the fear signals are called as “fear circuits”. Amygdala is a structure in the temporal lobe, responsible for emotional responses associated with physiological responses to stimuli (Whalen and Phelps 2009: 53). Amygdala is not a “thinking” part of the brain but a “reacting” part and therefore, it cannot recognize the reason why the individual is afraid of,

but it just brings the bad memories related to fight or flight response to the surface. Consequently, anxiety symptoms could be present.

Hypothalamus is the structure that controls appetite, body temperature and it also registers pain and pleasure. When there is a threat, the hypothalamus operates four specific functions as follows. They are activating the functions of the Autonomic Nervous System (ANS), stimulating the secretion of adrenocorticotrophic hormone (ACTH), producing antidiuretic (ADH) hormone and stimulating the production of thyroxin. In anxious conditions all the above functions are activated producing anxiety symptoms (Seaward 2008: 39).

02) Behavioral approach

According to behavioural approach, every human behavior is learned or conditioned response (Coon and Mitterer 2008: 486). This approach emphasizes an abnormal behavior as a result of learning and conditioning. Therefore anxiety is considered as a conditioned emotional response, acquired through the mechanism of conditioning. Behaviourists emphasize the two basic forms of learning as classical conditioning (learning by association) and operant conditioning (learning by reinforcements) (Eysenck 2004: 811).

Classical conditioning

Classical conditioning; a process of learning by association, explains how an anxiety response to nonthreatening stimuli can develop. According to classical conditioning, when something that is already feared is paired with something that is otherwise neutral, the individual learns to fear what was previously neutral (Rygh and Sanderson 2004: 11).Tthe development of a specific phobia involves classical conditioning. The conditioned stimulus is the phobic object and the unconditioned responses are fear or anxiety. For an instance, in the case of the small boy called Little Albert, he develops a phobia for rats through the mechanism of classical conditioning (pairing a loud noise with the presence of a white rat) (Eysenck 2004: 813). The Little Albert became frightened of a rat when the sight of it was paired seven times with a loud noise.

However, some theorists; Eysenk (1976, 1979), Miller (1948), Mowrer 1947, 1960) set a step forward in this model and explained that the classically conditioned fear acts act as a drive that motivates and reinforces the avoidance (cited in Lissek 2005). Classical

conditioning also stresses the incubation of the learned fear (Eysenk 1979), evolutionarily prepared aversive associations (Seligman 1971) failure to inhibit the fear response to safety cues (Davis et al. 2000), associative learning deficits (Grillon 2002), stimulus generalization (Zinbarg, 1996, Watson and Rayner 1920) and enhanced conditionability (Orr et al. 2000), which are responsible in formation and persistence of anxiety disorders.

Operant conditioning

Edward Thorndike (1905) studied the effects of consequences of behaviour rather than the association with the stimuli. He formulated a principle called the "law of effect": behavior that is followed by consequences satisfying to the organism will be repeated, and behavior that is followed by harmful or unpleasant consequences will be discouraged (Kring et al. 2010: 23).

Skinner (1938) re-named the Thorndike's "law of effect" as the "principle of reinforcement". Skinner introduced two types of reinforcements: positive and negative. Positive reinforcement means the strengthening of a tendency to respond to a pleasant event: positive reinforcer and negative reinforcement also strengthens a response but it does so via the removal of an aversive event: negative reinforcer. The anxious behavior will be continued due to positive reinforcements such as sympathy, attention or other types of rewards (Kring et al. 2010: 23).

03) Psychoanalytic Approach

Psychoanalysis introduced by Sigmund Freud is considered as the most famous model among the psychoanalytic approaches appeared during the 20th century (Eysenck 2004: 809). The psychoanalytic view of anxiety disorders explains a defense against repressed conflicts (Kring 2010: 153). Freud has divided the mind into three principal parts: *id*, *ego* and *super ego*. The *id* is present at birth and is the energy force needed to run the mind including the basic urges for food, water, elimination, warmth, affection, and sex. Its energy is biological which is called the "libido" which the individual is not able to perceive consciously and therefore it is unconscious. The *id* works on the "pleasure principle" and seeks immediate gratifications of its urges. When the *id* impulses cannot be satisfied, anxiety is produced and the *id* urges the individual to eliminate this anxiety as quickly as possible. The individual may obtain some relief by imagining in a short term basis, but this fantasizing cannot really satisfy the urges. Therefore, according to Freud, the *ego* begins to develop from the *id* to face for this

situation. This starts during the second six months in life (Kring 2010: 17). The *ego* is the rational and conscious part of the mind, which is developed during the first two years of life. The *ego* deals with the reality and therefore it works on the "reality principle". The *ego* acts as the mediator between the demands by the *id* and the reality. Also the *ego* cares of what is going on in the environment. The third part of the mind is the *super ego*, which develops at about five years of age. It is partly conscious and partly unconscious. When the child adopts many values of his or her environment (from parents, teachers and others) the *super ego* is developed. It consists of the conscience and the *ego-ideal*. The conscience is formed as a result of the punishments and it makes the child feel guilty after his bad behaviors. The *ego-ideal* is formed through the use of rewards which makes the child feel proud after behaving well.

According to Freud, anxiety disorders occur as a result of conflicts among these three parts of the mind. Mostly, conflicts occur between the *id* and the *super ego*. Since the *id* is the primary force of the mind, it needs immediate gratification, whereas the super ego relies on "moral values". The *id* impulses need to do something even it is not socially accepted and then the super ego says not to do that. In this condition, the *ego* becomes threatened by the both. These conflicts cause the individual to experience three types of anxiety: neurotic (between the *id* and the *ego*), moral (between the *id* and the *super ego*), and realistic (between the *id* and the environment). The work of *ego* is to resolve these conflicts.

The *ego* defenses itself by using various types of defense mechanisms such as repression, displacement, denial etc., which are the strategies to reduce anxiety.

The key defense mechanism of anxiety is repression. It consists of forcing, painful, threatening, or unacceptable thoughts and memories out of consciousness into the unconscious mind. The repressed ideas concern impulses or memories that the individual could not think about. According to Freud, phobias are a defense against anxiety producers when the impulses of the *id* or sexual instincts are repressed or forced into unconsciousness. This theory emerged from Freud's case study of "Little Hans" (Eysenck 2004: 861).

Discussion

It was evident that producing anxiety is multi-factorial with different approaches having different views on anxiety. The biological approach focuses on genetic and other biological factors, whereas behavioral and psychodynamic approaches focus on psychological factors. Each approach shows evidences through scientific research findings and or by rational arguments. Furthermore, all show valid reasoning in favour of their mechanisms. However, there are occasions where some theory in one approach is contradictory or disregard the same of the other approach. Therefore, a problem arises as to what to believe as right and it is challenging to draw a solid conclusion. However, it should be emphasized that neither of the approaches are complete but remains partially correct. Since any human behaviour is very complex, different views would be necessary to have a rather complete idea to explain the behavior.

Even though there some apparent conflicts, almost all the approaches believe the existence and the connection of mind and body. For instance, behaviourists do not consider about the mind but according to psychoanalytic approach the existence of the mind is the basic assumption. Biological approach is a reductionist itself and considers only biological structures and chemicals and does not pay much attention on one's psychological factors. In biological approach it is discussed how genes do their work via the environment. A question arises as to whether genes or the environment are more important. Literature provides evidences that both of these factors are important. Some also argue that nature and nurture work together, not in opposition to one another. Without the genes, any behaviour might not be possible. On the other hand, without the environment, genes could not express themselves. Human beings are quite flexible to adapt to different environments and genes are remarkably flexible at responding to different types of environments (Kring 2010).

Some genetic factors are considered to be involved in the origins of several specific phobias. Some studies reveal that the close relatives of individuals with specific phobia also had phobic conditions. It is also argued that phobias to run in families could well be due to the fact that members of the same family typically share many experiences. It is believed that although multiple genetic risk genes are there, adverse environmental effect would also be required in order to produce anxiety. Variability in maternal care is also plays a major role in such incidents (Blanchard et al. 2011: 496). However, most of the positive findings are difficult to replicate.

Although behaviorists argue that psychology should be scientific. However, as Hallam and Rachman(1976) suggest, an instance like Little Albert's in classical conditioning is unable to replicate (Eysenck 2004: 813), especially, it breaches the ethical standards.

According to behavioral approach conditioning predicts high level of anxiety by a traumatic event. However, it does not provide a detailed account of what is happening. Moreover, it does not make clear why some people develop PTSD in response to a traumatic event whereas, others do not. It also does not explain why some individuals develop PTSD rather than a specific phobia (Eysenck 2004: 837).

The psychoanalytic model proposed by Sigmund Freud was the first systematic model focused specially on psychological factors as the cause of anxiety as well as other mental disorders (Eysenck 2004: 811). According to Freud, phobias are a defense against anxiety producers when the impulses of the *id* or sexual instincts are repressed or forced into unconscious. Freud's case study of "Little Hans" gives the best instance for this scenario (Eysenck 2004: 839).

Anxiety may be produced by the interactions of the above factors mentioned in different approaches. For instance, someone may have a very high or a very low level of a given brain chemical because of genetic factors or because the individual has recently experienced a critical life event. According to another example the impact of cultural expectations can create anxiety in an individual if the demand is so high from the society (Eysenck 2004: 821).

Anxiety disorders may be produced by the interactions of the above factors and neither approach is complete but remains partially correct. It was also clear anxiety could arise due to many different causes and mechanisms. In order to overcome anxiety disorders, thinking and methods should be focused on the causes rather than overt behaviours.

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