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Etiological and Gender Perspectives of Anxiety Disorder Development

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This paper presents a comprehensive, integrative review of research on the biological, environmental, and cognitive etiology of anxiety. Causes of generalized anxiety disorder, specific and social phobias, panic disorder, and obsessive-compulsive disorder are discussed, as is the role of gender in anxiety development. Biological research concerning genetic heritability, neurobiological structure, and neurotransmitter functioning are reviewed. Environmental factors such as aversive events, parental interactions, environmental control, and learning are discussed, followed by research on cognitive distortions, attribution style, attention biases, and catastrophic cognitions. The review finds evidence of an interaction between biological, environmental, and cognitive variables, as well as a mediating influence of gender. Research is still needed to determine the processes and interactions by which anxiety develops and the reasons for gender differences in disorder prevalence. The review shows that research and analysis have been inadequate in making connections between each of the biological, environmental, and cognitive factors. Although research has clearly indicated that each of the aforementioned variables influence anxiety disorder development, most studies have examined only one aspect of anxiety etiology, disregarding the impact of other potential causes.

Anxiety is a prevalent psychological condition that encompasses several specific disorders characterized by fearfulness and worry. Meta analysis reveals that compared with nonclinical controls, individuals with anxiety disorders perceive a lower quality of life in the domains of health, occupation, home, family and especially social relationships (Olatunji, Cisler, & Tolin, 2007). Because of this impact on quality of life, treatment of anxiety is of clinical importance, and thus the mechanisms leading to anxiety development must be understood. This paper presents a comprehensive review of research on the biological, environmental and cognitive etiology of anxiety, and the interactions between the three influences. The review reflects the integrative nature of variables influencing the etiology of anxiety, and isolates variables that may account for gender discrepancies in anxiety prevalence. The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) divides anxiety disorders into twelve classifications: generalized anxiety disorder, specific phobia, social phobia, panic disorder with and without agoraphobia, agoraphobia without history of panic, obsessive-compulsive disorder, acute stress disorder, anxiety disorder due to general medical condition, posttraumatic stress disorder, substance-induced anxiety disorder, and anxiety disorder not otherwise specified. This paper reviews the etiology of anxiety, featuring research concerning all disorder categories except stress.
disorders and disorders attributed to known physical conditions. While each of the other disorders share a complex, highly debated etiology, the development of stress disorders and those linked to physical conditions are attributed to a specific, highly personalized, known cause or event (APA, 2000). Because of this inherent difference, this review concerns only anxiety as a general construct focusing on generalized anxiety disorder, specific phobia, social phobia, panic disorder, and obsessive-compulsive disorder.

Generalized anxiety disorder (GAD) is the most common anxiety disorder, characterized by uncontrollable worry focused on many facets of life (APA, 2000). Approximately two-thirds of the individuals afflicted with GAD are female (APA, 2000). Specific phobia is characterized by persistent fear of a particular stimulus, accompanied by avoidance of the stimulus (APA, 2000). Like GAD, females are overrepresented in specific phobia diagnosis, representing roughly twice as many cases as males (APA, 2000). Similarly, social phobia is characterized by intense, persistent fear of social situations in which a person feels that he or she might be evaluated or judged. Social situations are met with distress, apprehension, and avoidance. Women are impacted by this disorder more frequently than are men; however, prevalence estimates vary amongst the general population, and within clinical samples prevalence has not been shown to differ by gender (APA, 2000). The DSM-IV classifies panic as a form of anxiety. Panic disorder is characterized by recurrent, spontaneous panic attacks, coupled with persistent anxiety concerning the possibility and implications of subsequent attacks. Two classifications of panic disorders exist—panic disorder with and without agoraphobia. Agoraphobia is the fear of being in places or situations from which escape might be difficult or impossible, often occurring when the individual is out of the home alone or in a crowded area (APA, 2000). Female diagnoses are twice as frequent for panic disorder without agoraphobia, and three times as frequent for panic disorder with agoraphobia (APA, 2000). The final anxiety disorder examined here is obsessive-compulsive disorder (OCD). OCD is an anxiety disorder characterized by recurrent obsessions and/or compulsions, and unlike the aforementioned disorders, demonstrates an equal gender distribution in prevalence (APA, 2000).

Research indicates that anxiety results from the combination of several factors that interact to elicit anxious symptomology and disorder development. These factors are divided into three categories—biological, environmental, and cognitive determinants. Research concerning each of these broad categories has identified a number of specific variables that may lead to the development of anxiety disorders, either alone or in association with other characteristics.

Biological Factors

Review of biological factors include analyses of genetic predispositions assessed through family and twin studies, analyses of specific gene variants associated with anxiety, studies of neurological structures associated with anxiety, and research on neurotransmitter disparities between anxious and non-anxious individuals. As relevant, the relationship between gender and biological factors is addressed.

Genetics & Heritability

Among leading biological theories is the idea that there is a genetic predisposition to develop anxiety disorders (Leonardo & Hen, 2006). According to these theories, anxiety is a highly heritable trait passed down within families. To investigate this idea family and twin studies have been conducted.

Family studies. An indirect measure of genetic disorder heritability is epidemiology within families. According to such research, if a disorder is attributed at least in part to genetic features, then the prevalence of the disorder will be higher in families with a history of the disorder than in control families with no disorder history, sometimes referred to as a familial link. Disorder prevalence should be highest amongst immediate family
most anxiety disorders. However, because concordance rates, at best, are roughly estimated around 40% for monozygotic pairs, an interaction likely exists between genetics and other personal and environmental factors.

**Gender**

Women have been estimated three times as likely to develop an anxiety disorder as are men (Zerbe, 1995), and this disparity may be partially based in familial heritability. Eley (2001) theorized that females exhibit higher levels of anxiety heritability than do males, as they are more likely to develop anxiety symptoms in the presence of a genetic predisposition. Although theoretical, this idea in conjunction with other familial research leads one to question the possible reasons for heritability – specifically, what factors are being inherited. Eley's gender theories postulate that girls are more likely to develop the temperamental personality trait of behavioral inhibition. Behavioral inhibition is the facet of personality responsible for reactions to stress and is highlyheritable (Gray, 1991; Kagan, Reznick & Snidman, 1988). Behavioral inhibition is related to several areas of the brain that control emotions and attention, and is responsible for suppressing behavior and redirecting focus in the presence of new stimuli. The less active and sensitive a person's behavioral inhibition system is the more likely he or she is to develop anxiety, because anxious behavior is unsuppressed and the individual remains focused on the anxiety provoking stimuli (Kagan et al., 1988). Behavioral inhibition in childhood has been found related to parental behavioral inhibition (Biederman et al., 1993), as well as to social avoidance (Kagan et al., 1988) and anxiety disorder development later in life (Schwartz, Snidman, & Kagan, 1999). Research has suggested that females have a higher propensity for behavioral inhibition than do males (Muris, Merckelbach, Wessel, & van de Ven, 1999), which may explain the gender differences in anxiety heritability.

**Specific Genes**

Research has shown evidence of family heritability of anxiety disorders and possible personality characteristics; however, this research alone cannot isolate biological and environmental factors. This demonstrated relationship directs research to investigate possible genes or biological features that may be responsible for the heritability of anxiety within families. Genetic research elucidates valuable biological links, yet still does not imply causation, as interactions may exist between genetics and shared family environmental factors. Although researchers have examined the possible role of many genes in anxiety disorder development, results concerning a specific genetic variant or abnormality have raised more questions than have been answered. Several genes have been identified as possible candidates for anxiety heritability with no definitive results. Research regarding these candidate genes is often contradictory or inconclusive, and explanations regarding the expression of these genes is, as of yet, inadequate.

Some research has suggested that anxiety-related personality characteristics may be associated with allelic variations of the 5-HTT gene-linked polymorphic region (Leonard & Hen, 2006). Specifically, the personality trait of anxiety sensitivity has been linked to the 5-HTT gene responsible for serotonin transport. Anxiety sensitivity is characterized both by a fear of experiencing anxiety symptoms, and by the belief that these symptoms are predictive of harmful consequences. Anxiety sensitivity is a known risk factor for anxiety disorder development, and has been found to be associated with the 5-HTTLPR polymorphism of the serotonin transporter gene (Leonard & Hen, 2006). In addition to this, the 5-HTTLPR genotype and childhood maltreatment have been shown to interact, resulting in increased anxiety sensitivity, and consequently an increased disorder risk (Stein, Schork, & Gelernter, 2008).

While much research has been suggestive of the 5-HTTLPR polymorphism of the 5-HTT gene as a possible candidate for anxiety heritability, it has also been posited that the STin2 VNTR polymorphism of the 5-HTT gene may be responsible for anxious symptoms. Although both originating from the same gene, the 5-HTTLPR
and STin2 VNTR genotypes involve different alleles, thereby resulting in different genetic variations. There are positive correlations between the STin2 VNTR polymorphism and anxiety disorders (Ohara, Yamakawa, Nakayama, Yuasa, & Ohara, 1999), specifically associations between this genotype and OCD (Saiz et al., 2008). While relationships between these variations of the 5-HTT gene and anxiety have been demonstrated, the biological relevance of these findings is still unclear.

The Extraneuronal Monoamine Transporter (EMT) has also been identified as a possible genetic candidate responsible for anxious expression. EMT is responsible for the inactivation of both circulating and centrally released catecholamines, which are hormones that prepare the body for either a "fight" or "flight" reaction to stressful stimuli (Gründemann, Schechinger, Rappold, & Schomig, 1998). Because of its role in catecholamine inactivation, EMT may be a genetic contributor to psychiatric disorders. Research with OCD afflicted patients is indicative of an association between this disorder and EMT gene mutations; however, such research is rare and these variations alone do not account for all cases of OCD (Lazar et al., 2007). Another genetic transporter implicated in the expression of anxiety symptoms is the Vesicular Monoamine Transporter (VMAT). There are two distinct categories of VMATs, each responsible for the presynaptic packaging of specific neurotransmitters. The VMAT1 gene, which is located on chromosome 8p21, is responsible for the packaging of serotonin, and has been previously linked to psychiatric disorders including bipolar disorder (Lohoff et al., 2006) and schizophrenia (Bly, 2005). Polymorphic variations of this gene are related to both state and trait anxiety levels (Lohoff et al., 2008). The location of VMAT1 on chromosome 8p21 is of additional etiological relevance because this chromosome is related to the expression of trait anxiety (Dina et al., 2005). Although research has demonstrated associations between this gene and anxiety, genetic investigation is at an early stage.

Research and replication are necessary to provide convincing evidence of the causal effects of any specific gene. While some genes may be responsible for the heritability of anxiety and anxiety disorders, it is also possible that individuals with anxiety differ neurobiologically, and perhaps this is the feature related to inheritance.

Neurological Structure and Function

Hypothalamo-pituitary-adrenal axis and the corticotropin-releasing factor. Research has identified several areas of the brain associated with anxiety disorders. One area possibly related to anxious pathology is the Hypothalamo-Pituitary-Adrenal (HPA) axis (Heim & Nemeroff, 2001). The HPA axis is a part of the brain related to stress responding that works in conjunction with the Autonomic Nervous System to elicit reactions to threatening or stressful situations. The Autonomic Nervous System acts as the first response to stress by releasing catecholamines in the presence of stressful stimuli. Through their secretion from the Autonomic Nervous System, catecholamines activate the HPA axis, thereby beginning the secondary response to stress. This HPA activation results in the release of corticosteroids, which regulate emotional and behavioral responding to stress. This progression describes the normal reaction to stress-inducing stimuli, and is coordinated by the paraventricular nucleus. The paraventricular nucleus is responsible for stress responding through the function of the Corticotropin-Releasing Factor (CRF), which is synthesized in this region. The paraventricular nucleus and CRF are responsible for the severity of stress reactions carried out by the Autonomic Nervous System and HPA (Heim & Nemeroff, 2001).

Hypersecretion of CRF is a crucial factor in anxiety disorder development (Heim & Nemeroff, 2001). In order to determine the function and importance of CRF, animal research has been
conducted, in which CRF levels in the brains of rats have been altered. Data show that increased CRF is related to increased withdrawal and decreased social interaction (Takahashi, Kalin, Vanden Burgt, & Sherman, 1989; Dunn & File, 1987). In a similar experiment, CRF was genetically deleted from the brains of rats, causing anxious symptomology to be nonexistent (Smith et al., 1998). HPA functioning impairments also exist in human beings with anxiety (Kirby, Rice, & Valentino, 2000); however, because CRF levels cannot ethically be altered in human research, the exact causes and nature of this relationship remain unclear.

While the precise nature of CRF is difficult to investigate, research has demonstrated that increased levels of corticosterone, a hormone related to stress responding, are related to subsequent increases in anxiety. Whether exogenously administered or naturally released because of stress, corticosterone leads to increases in both anxious behavior and HPA responding in rats (McCormick, Smith, & Mathews, 2008). Such research is in an early stage, and consequently has yet to be conducted with a sample of human participants; however, response patterns found in animal research are predicted to be similar to those of human adolescents. During human adolescence, there is an increased vulnerability for anxiety development, as the adolescent brain is undergoing a reorganization of the HPA and its functioning. Physiological and psychological stress responding is in transition over this period (McCormick & Mathews, 2007), and consequently adolescents may have an increased susceptibility to the detrimental effects of aversive environmental stimuli. Experimental investigations have indicated that there may be latent expression of these negative environmental effects, as research using a rat model of adolescent social stress indicates that adolescent stress often does not manifest into anxious tendencies until adulthood (McCormick et al., 2008).

Other structural or functional processes. A study by Condren, O'Neill, Ryan, Barrett and Thakore (2002) regarding social phobia has indicated that anxiety might be related to factors such as increased cortisol responding in the HPA region. It is unclear from this research, however, if increased cortisol levels precede or result from phobic symptoms. Some studies have indicated that structural abnormalities in the HPA area may be related to the occurrence of anxiety disorders, though in research concerning the possibility of a parahippocampal association with anxious pathology no structural disparities were found between anxious and control participants (Vythilingham et al., 2000). However, anxiety, and most significantly panic disorder, were found to be related to blood flow in both parahippocampal areas (Stewart, Devous, Rush, Lane, & Bonte, 1988), increased volume in the temporal lobe (Vythilingham et al., 2000), and epileptiform discharges (Beauclair & Fontaine, 1986).

OCD. Unlike most anxiety disorders, specific links have been shown between OCD symptomatology and structural abnormalities in the brain. Research has demonstrated correlations between OCD and abnormalities in the thalamus, caudate nucleus, orbital cortex, and cingulate gyrus (Jenike et al., 1996). In addition to this, evidence of dysfunction in the orbitofrontal cortex of OCD affected patients has been demonstrated. Positron emission tomography studies have found increased rates of glucose metabolism in the orbitofrontal cortex of OCD patients, as compared with control participants (Evans, Lewis, & lobst, 2004). Similarly, research with single photon emission computed tomography technology has indicated that OCD patients experience increased blood flow in the orbitofrontal cortex (Alptekin et al., 2001). It has been theorized that these orbitofrontal cortex dysfunctions may result in cortical hyperactivity. This hyperactivity is thought to accelerate attentional and cognitive processing, resulting in the expression of the obsessions and compulsions characteristic of the disorder. Research has supported this theory, demonstrating that individuals with OCD experience an overfocusing of attention and increased cognitive arousal when compared to control participants (Saxena & Rauch, 2000).
These studies are indicative of a possible structural or neural functioning impairment that may be related to anxious pathology.

While neurobiological research has made strides in recent years, there is still much that is not understood about neurological functioning. Many of the theories regarding the role of neurobiology in anxiety development are still highly debated or difficult to methodologically research, and the exact relationships between many neurological findings and subsequent psychopathology are often hard to determine.

**Neurotransmitter Functioning**

*Serotonin.* Research has also focused on possible neurotransmitter disparities between anxious and non-anxious individuals. Neurotransmitter research has focused largely on the impact serotonin on anxious symptomology. Serotonin is a neurotransmitter shown to help regulate mood, as well as inhibit such emotions as anger and aggression. There appears to be a relationship between OCD and decreased levels of serotonin in the brain (Yaryura-Tobias & Neziroglu, 1997). Similarly, serotonin, and specifically the serotonin system, is related to GAD. The serotonin system consists of serotonin transporters (5-HT) and tryptophan hydroxylase (TPH), and is responsible for the metabolism and regulation of serotonin in the brain. Animal research has shown that 5-HT levels of serotonin transporter increase in the presence of threatening stimuli, leading to the cortical and limbic reactions associated with anxiety (You, Hu, Chen, & Zhang, 2005). A similar response was seen in GAD patients, with increased levels of 5-HT found when individuals were exposed to threatening stimuli. In addition to this, TPH, the enzyme responsible for 5-HT regulation, has been shown associated with GAD (You et al., 2005). Research with socially anxious participants has also indicated a positive correlation between pathological social phobia and a hypersensitivity of the 5-HT receptors (Schneier, Luterek, Heimberg, & Leonardo, 2004).

*Other neurotransmitters.* In addition to serotonin, studies have suggested other possible neurotransmitters related to anxiety disorders and anxious symptomology. McIntyre, Norman, Burrows, and Armstrong (1990) found evidence of a relationship between melatonin levels and panic disorder. This research demonstrated that melatonin, the neurotransmitter related to circadian cycle regulation, was significantly higher in participants with panic disorder than in control participants. As with much biological research, however, findings regarding melatonin are often contradictory. While some studies have linked panic disorder to increased melatonin levels, others have reported decreases in melatonin production in panic disorder patients. These decreased levels of brain melatonin often result in a patient experiencing subsensitivity to light, which has been used as a biological marker for panic disorder (Nathan, Burrows, & Norman, 1998). In addition to melatonin, decreased levels of dopamine, a neurotransmitter related to cognition and motor activity, might be related to social and specific phobias (Schneier et al., 2004). Further research has indicated that dysfunction of $\gamma$-aminobutyric acid (GABA) receptors in the brain may increase the likelihood of GAD development (Crestani et al., 1999). These receptors play a crucial role in the neurotransmitter system, as their functioning determines subsequent neurotransmitter functioning. The GABA system is responsible for many inhibitory functions in the brain, and dysfunction of these inhibitory processes may therefore allow certain anxious thoughts or behaviors to persist. In addition to these findings, decreased peripheral benzodiazepine receptor levels (pBR) have been found in anxiety disorder patients, as compared to control individuals (Rocca et al., 1998). Lastly, some research has found evidence of increased norepinephrine activity in individuals with anxiety disorders, specifically examining those with GAD (Khan et al., 1986). Norepinephrine is a neurotransmitter related to processes of attention, as well as stress responding. Therefore, it is logical that anxiety would be related to increased levels of the neurotransmitter responsible for stress-related responses.
While the aforementioned research is indicative of a link between specific neurotransmitters and disorder development, there is much debate concerning the exact influence of each brain chemical. As with the genetic and neurological research previously discussed, neurotransmitters do not alone account for all of the facets of anxiety development, and the nature of their contribution is clouded with uncertainty. Until we are able to better isolate and study each brain chemical, the exact effect of any one particular neurotransmitter will remain unclear.

Gender Also unclear is how these brain abnormalities or neurotransmitters may contribute to the gender disparity in anxiety prevalence. Male rats have been found more likely to experience delayed stress responding because of corticosterone levels and HPA functioning in adolescence than are female rats (McCormick et al., 2008). However, this finding does not help to explain why human female prevalence rates exceed male anxiety estimates in adulthood. To address this disparity, it has been suggested that the difference in anxiety incidence according to gender may be the result, not of brain structures or neurotransmitters, but of other brain hormones. Pigott (1999) has suggested that human female sex hormones present in puberty and during the menstrual cycle, may affect stress and anxiety levels in women. Specifically, the hormone progesterone has been shown to have destabilizing effects on mood, and may place women at an increased risk for anxiety development, or may facilitate the expression of anxious tendencies. However, this research is highly speculative and does not explain why only some women are afflicted with anxiety, despite the presence of progesterone in all females.

Environmental Factors
While empirical data has made the link between biological factors and the development of anxious pathology undeniable, the environment within which an individual interacts serves as a catalyst for these biological predispositions. Because neurobiological data are inconsistent, and because monozygotic concordance rates are well under 100%, other factors must be relevant in the development of anxiety. Early exposure to adverse experiences may reveal an innate vulnerability to anxiety, and result in the development of a stable, anxious phenotype. Similarly, subtle genetic vulnerabilities to anxiety have been shown to develop into increased susceptibility or even anxiety disorders, in the presence of significant environmental experiences (Heim & Nemeroff, 2001). Through this and similar research, it can be concluded that environmental experiences can influence susceptibility to psychopathology, as well as create a tendency for an individual to express anxious behaviors when in the presence of threatening stimuli (Gross & Hen, 2004). Because of this apparent interaction between learned and innate etiological factors, research has examined associations between anxiety development and traumatic life events, parental interactions, experiencing a lack of control, socialization, and learning, with gender serving as a moderating variable.

Aversive Events
Post traumatic stress disorder, an anxiety disorder related to the occurrence of trauma experiences such as war or violence (APA, 2000), has been the subject of much environmentally focused etiological research. Studies have shown, however, that PTSD is not alone in its etiological link to traumatic events. Research has shown that traumatic experiences occurring early in childhood may be associated with later anxiety development, regardless of disorder classification (McCaulley et al., 1997; Loewenthal et al., 1997; Murrey et al., 1993). These experiences include, but are not limited to, physical abuse, sexual abuse, loss of a loved one, parental divorce (Gross & Hen, 2004) and chronic social stress (McCormick et al., 2008).

As was previously mentioned, these traumatic experiences can influence anxiety development in two ways. First, these early experiences can influence developmental mechanisms in the brain, such that a person becomes more susceptible to future
psychopathology. This is the result of changes in areas of the brain, such as the 5-HTT gene (Lesch et al., 1997) and the hippocampal region (Khazipov et al., 2001) which are associated with fear responding. Research has also linked early trauma, specifically violent family interactions, with polymorphisms of the MAOA gene, which is responsible for coding the enzyme that metabolizes 5-HT and dopamine (Caspi et al., 2002).

In addition to influencing brain mechanisms, an individual's response to early traumatic experiences may lead to the development of a life-long tendency to react to threatening stimuli with anxiety. Traumatic experiences early in the process of brain development may interact with genetic or neurobiological factors to set an individual's personal level of trait anxiety, thereby determining how he or she will respond to stress throughout life. While research specific to this theory is limited thus far in human samples, research with mice has indicated that the interactions between mother rodents and their offspring, combined with other early environmental influences, can influence the levels of neurotransmitters and receptor proteins in the hippocampal regions (Liu, Diorio, Day, & Meaney, 2000; Weaver et al., 2001). The exact impact of these changes is still unclear; however, it has been postulated that early alterations in brain structure and functioning can influence the maturation of dendrite branches, thereby influencing lifelong fear-responding tendencies (Pokorny & Yamamoto, 1981).

**Parental Influences**

*Parent/child relationship.* Early-life attachment style seems highly related to anxious pathology. Research addresses the relationship between anxiety and secure and insecure attachment styles. Secure infant-parent attachment is positively correlated with social engagement later in childhood on into adulthood (Atili, 1989).

Insecure attachment is correlated with adult GAD (Cassidy, Lichtenstein-Phelps, Sibrava, Thomas, & Borkovec, 2009), adult trait anxiety and adult sensitivity (Watt, McWilliams, & Campbell, 2005). Furthermore, associations between maternal and child anxiety are mediated by maternal perceptions of insecure attachment (Costa & Weems, 2005). Another parent/child relationship dynamic is conceptualized as parental rejection. Aversive parent behavior such as disapproval, unresponsiveness and withdrawal juxtaposed with minimal expressions of warmth and praise are hypothesized to increase tendencies toward anxiety (Gottman, Katz, & Hooven, 1997). However, a recent meta analysis found that only 4% of the variance in childhood anxiety was accounted for by parent rejection (McLeod, Wood, & Weisz, 2007).

*Over-protective parents.* Over-protective parenting may influence child social functioning and anxiety development. Parents afflicted with anxiety, because of their own anxious thoughts and tendencies, may prevent their children from engaging in normal childhood activities for fear of physical or emotional harm (Lieb et al., 2000). When anxious parents create a restrictive environment it can influence the social interactions of their children by (a) preventing the child from developing necessary mechanisms for coping with fearful stimuli, and (b) communicating that the restricted stimuli are threatening (Costa & Weems, 2005). These children socialize significantly less than do their peers (Bogels, van Oosten, Muris, & Smulders, 2001), which may be a factor related to later social or specific phobia development. Finally, child perceptions of maternal control and overprotection are found predictive of child anxiety (Costa & Weems, 2005). Consider habituation, an essential feature of anxiety treatment. Habituation is a natural process that involves the repeated exposure of an anxious individual to their feared stimulus, resulting in the desensitization of that stimulus. By restricting their children's exposure to anxiety-inducing stimuli, anxious parents might prevent natural habituation processes from occurring.

Like restrictive environments, restrictive forms of parent-child communication may also relate to anxiety development by influencing avoidance tendencies in children. Research by Dadds, Barrett,
Rapee, and Ryan (1996) found that parent-child verbal interaction style differed based on maternal anxiety diagnosis, such that mothers of children with anxiety were less likely than mothers of children without anxiety to agree with their children, listen to them or communicate positive outcomes in ambiguous situations. This was related to strengthened avoidance tendencies in their children. It is unclear, however, if these restrictive forms of communication developed into, or because of, the child’s anxiety. Nonetheless, restricted children may feel compelled to doubt their efficacy when required to separate from their family and behave independently. Similar to habituation, some treatment models direct parents to support child independence in novel settings, leading to enhanced self-efficacy/reduced anxiety (Chorpita & Barlow, 1998). McLeod et al.’s 2007 meta-analysis suggests that the impact of over-protective parenting may account for more childhood anxiety than a poor parent child relationship, especially among diagnosed children.

Modeling. Anxious parents may influence anxiety development in their children through modeling. Parental phobic behaviors may be observed by children and imitated, leading to decreased childhood social interactions and subsequent social phobia development. Research with childhood anxiety outpatients revealed that trait anxiety levels and degree of fear in children were positively correlated with trait anxiety and fear in their parents. Modeling mediated these relationships, with similar patterns of fear expression demonstrated within mother-child pairs (Muris, Steerneman, Merckelbach, & Meesters, 1996). Similarly, anxious parenting, as measured by child perceptions of parental anxiety in parenting behaviors, has been positively correlated with child anxiety and fear in their parents. Modeling mediated these relationships, with similar patterns of fear expression demonstrated within mother-child pairs (Muris, Steerneman, Merckelbach, & Meesters, 1996). Similarly, anxious parenting, as measured by child perceptions of parental anxiety in parenting behaviors, has been positively correlated with child anxiety and fear in their parents. Modeling mediated these relationships, with similar patterns of fear expression demonstrated within mother-child pairs (Muris, Steerneman, Merckelbach, & Meesters, 1996). Similarly, anxious parenting, as measured by child perceptions of parental anxiety in parenting behaviors, has been positively correlated with child anxiety and fear in their parents. Modeling mediated these relationships, with similar patterns of fear expression demonstrated within mother-child pairs (Muris, Steerneman, Merckelbach, & Meesters, 1996). Similarly, anxious parenting, as measured by child perceptions of parental anxiety in parenting behaviors, has been positively correlated with child anxiety and fear in their parents. Modeling mediated these relationships, with similar patterns of fear expression demonstrated within mother-child pairs (Muris, Steerneman, Merckelbach, & Meesters, 1996). Similarly, anxious parenting, as measured by child perceptions of parental anxiety in parenting behaviors, has been positively correlated with child anxiety and fear in their parents. Modeling mediated these relationships, with similar patterns of fear expression demonstrated within mother-child pairs (Muris, Steerneman, Merckelbach, & Meesters, 1996). Similarly, anxious parenting, as measured by child perceptions of parental anxiety in parenting behaviors, has been positively correlated with child anxiety and fear in their parents. Modeling mediated these relationships, with similar patterns of fear expression demonstrated within mother-child pairs (Muris, Steerneman, Merckelbach, & Meesters, 1996). Similarly, anxious parenting, as measured by child perceptions of parental anxiety in parenting behaviors, has been positively correlated with child anxiety and fear in their parents. Modeling mediated these relationships, with similar patterns of fear expression demonstrated within mother-child pairs (Muris, Steerneman, Merckelbach, & Meesters, 1996). Similarly, anxious parenting, as measured by child perceptions of parental anxiety in parenting behaviors, has been positively correlated with child anxiety and fear in their parents. Modeling mediated these relationships, with similar patterns of fear expression demonstrated within mother-child pairs (Muris, Steerneman, Merckelbach, & Meesters, 1996). Similarly, anxious parenting, as measured by child perceptions of parental anxiety in parenting behaviors, has been positively correlated with child anxiety and fear in their parents. Modeling mediated these relationships, with similar patterns of fear expression demonstrated within mother-child pairs (Muris, Steerneman, Merckelbach, & Meesters, 1996). Similar

Experiences of Control

Research has shown a negative correlation between experiences of environmental control and anxiety, with lower levels of reported control predictive of increased anxious symptomatology, both in frequency and intensity. In this research, threatening or unpredictable events concerning participants’ feelings over mastery and control of such events were examined. Findings indicated that those participants who had experienced a sense of environmental control and mastery were significantly less likely to develop an anxiety disorder than were participants who thought that they were out of control or incapable of mastering threatening or unpredictable events (Bouton, Mineka, & Barlow, 2001). This research suggests that it is not only the occurrence of life trauma that may place an individual at risk for anxiety development, but also his or her cognitive perception of capability in the presence of the situation. As such, the issue of control, even though learned, might be considered a cognitive process (c.f., Hoffman, 2008).

Environmental Gender Differences

Sexual abuse. Gender disparities remain a dominant theme. Females seem to be at heightened risk to experience life trauma significant in the development of anxious pathology. Approximately 20% of women versus 7% of men have experienced sexual abuse in childhood (Pereda, Guiler, Forns, & Gomez-Benito, 2009) and the prevalence of sex abuse increases amongst women with anxiety disorders (Stein et al., 1996; Yama, Tovey, & Fogas, 1993). This indicates that traumatic life events, such as sexual abuse, are instrumental in the development of anxiety disorders (Murrey et al., 1993).

Socialization. It may be postulated that parents socialize daughters differently than they do sons. While speculative, some researchers have theorized that girls are socialized to inhibit externalizing behaviors, such as aggression, in the presence of threatening or upsetting stimuli, and are taught instead to express internalizing behaviors, such as fear, sadness, or anxiety (Lonigan &
Phillips, 2001). Similarly, the development of empathy occurs earlier in girls than in boys. This earlier ability to understand the feelings of others has been associated with the higher rates of female anxiety in adolescence (Zahn-Waxler, Cole & Barrett, 1991).

As applied to the aforementioned research on parental influence, it is possible that parents suffering with anxiety will be more likely to create restrictive environments for their daughters than for their sons. Research by Lewinsohn, Gotlib, Lewinsohn, Seeley, and Allen (1998) found that as early as age six girls were twice as likely to experience an anxiety disorder than were boys, and that this tendency continued well into adolescence and adulthood. Similarly, these data show that young girls were more likely to exhibit anxious symptoms when separated from their parental figure than were young boys. This and other research has suggested that a possible cause for these gender differences involves the internalization of environmentally taught gender roles. By observing the emotional expression of their parents, children may internalize what is acceptable behavior for their gender, and learn how to conduct themselves within a family and within society. Zamarripa, Wampold, and Gregory (2003) researched this correlation between gender role internalization and anxious and depressive symptomology, focusing specifically on the level of importance participants placed on certain stereotyped gender roles. Results found that as men’s regard for success, power, and bravery increased, so did their anxious symptomology. Similarly, as a woman’s focus on household tasks increased, so did anxious symptomology. This suggests that not only do gender roles influence male and female expressions of emotion, but when strongly internalized could lead to anxiety development.

The interaction between fathers and daughters is also relevant in child socialization and the gender disparity in anxiety prevalence. Both paternal affection (Jorm, Dear, Rodgers, & Christensen, 2003) and paternal feelings of male superiority (Silverstein & Lynch, 1998) have been linked to female anxiety. Correlations are also found between levels of paternal anxiety and anxiety in adolescent females (Bosco, Renk, Dinger, Epstein, & Phares, 2003). While parental interactions are crucial in the psychological development of both genders, the unique relationship between father and daughter seems to place females at an increased risk for anxiety development, perhaps partially accounting for the disproportionate disorder prevalence.

**Gender and control.** Leadbetter, Blatt and Quinlan (1995) speculated that women might display a lesser tendency to experience feelings of controllability over their environment than men display. Because feelings of environmental control and mastery are related to anxiety development, the greater likelihood for males to experience these feelings may contribute to their lower prevalence of anxiety. However, research concerning this theory is limited. While such environmental theories do not entirely account for the gender difference in anxiety incidence, they may act in conjunction with other etiological factors to create female-biased disorder prevalence.

Though each of the gender differences discussed throughout the present review may play a role in the development of anxiety disorders, gender differences in anxiety prevalence may be partially cultural. Even when symptoms are comparable, women are more likely to seek psychological treatment than are men (Kessler, Brown, & Brom, 1981). Thus gender differences in anxiety prevalence may be inflated by or result from a greater likelihood for women to seek treatment and therefore receive diagnoses.

**Learning Theories**

Similar to, and often overlapping the above, operant and classical learning theories concerning social interactions and traumatic life experiences have been used to account for the development of anxious psychopathology. For example, traditional learning theory speculates that phobias are the result of traumatic conditioning, in which an individual’s behavioral response of avoidance in the
presence of a traumatic stimulus has been negatively reinforced by escape or avoidance of that stimulus (Ost & Hugdahl, 1981). This theory, however, does not take into account the large population of individuals who experience trauma in life and do not develop anxious avoidance. In response, contemporary learning theory has provided two possible explanations for the tendency of only some individuals to develop phobic pathology in the presence of threatening or traumatic stimuli. First, individual differences, such as genetic vulnerability and feelings of control may mediate the relationship between trauma and phobia acquisition. This may be the case, as stronger feelings of lack of control and mastery have been found related to an increased likelihood of anxiety (Chorpita & Barlow, 1998). For example, Sanderson, Rapee and Barlow (1989) demonstrated that panic disorder patients were four times as likely to have a panic attack when manipulated to feel out of control.

A second explanation regarding the tendency for only some to develop phobias in the presence of trauma is the phenomenon known as latent inhibition. Latent inhibition describes a situation in which exposure to a neutral stimulus in an un-traumatic context, prior to its pairing with a fear eliciting unconditioned stimulus, decreases the likelihood that a person will develop a phobia of the previously neutral stimulus during subsequent traumatic conditioning (Lubow, 1998). Therefore, anxiety development may be the result of a lack of latent inhibition wherein an individual's first exposure to a neutral stimulus occurs in a traumatic context, thereby eliciting anxiety when in the presence of that stimulus in future scenarios. Individuals who have experienced parental interactions where environmental exposure has been limited may be at a greater risk for anxiety because opportunities for latent inhibition during childhood are prevented. It is also possible that disordered development is a direct result of phobic behavior reinforcement. For example, research regarding individuals with social phobia has found evidence of the reinforcement of avoidance behaviors in social situations prior to disorder development (Dadds et al., 1996). As was previously discussed, exposure to the feared stimulus through the process of habituation is necessary in order to overcome anxious tendencies. Therefore, social apprehension may develop into a phobia when avoidance of the feared situation is negatively reinforced.

In similar research, regarding patients afflicted with GAD, individual characteristics such as genetic predisposition and feelings of control were shown to mediate the learning process (Mineka & Zinbarg, 2006). As was discussed earlier, the central diagnostic feature of GAD is chronic, excessive worry. While learning theories have not yet developed an explanation for the initial occurrence of pathological worry, it has been hypothesized that worry is reinforced through the process of cognitive avoidance. This process becomes a self-sustaining cycle, in which physiological stress responding is suppressed, and worry intensified. Based upon a review of research, Borkovec, Shadick and Hopkins (1991) theorize that the act of worrying causes an individual to suppress fear imagery, thereby suppressing the physiological and somatic responses associated with fear. Because of this response suppression, the individual is physiologically unable to adapt to the feared stimulus, and the topic's threatening nature is maintained. Therefore, although the individual is engaged in worry, they are avoiding the feared stimulus on a physiological level. This, in turn, leads to increased worrying, about both the initial topic, as well as the act of worrying itself (Borkovec, Alcaine, & Behar, 2004). Individuals with GAD come to perceive worry as uncontrollable because this cognitive avoidance increases the frequency and severity of worry, leading many patients to worry not only about the feared stimulus, but also about the implications of their worrying behavior and their inability to stop. Theory suggests that this process is further intensified in those with a history of feelings of environmental uncontrollability (Mineka & Zinbarg, 2006). However, while
contemporary learning theory helps to identify the factors that perpetuate worry, the reasons why only some of those who suffer with worry develop GAD are yet to be entirely accounted for.

GAD and OCD have also been explained using conditioning paradigms. Unlike other anxiety disorders, however, research regarding the processes by which OCD is reinforced is still largely inconclusive. It has been suggested that certain verbal cues or imagery may become paired with broad, neutral ideas, leading the individual to not only experience anxiety in the presence of these specific cues, but also to generalize their fears across time and settings. An example of such a scenario involves associating an aversive stimulus with a particular place or event in which this stimulus was present. While a typical individual can differentiate this scenario from other contexts, the anxieties of a patient with OCD may generalize across situations (Mineka & Zinbarg, 2006).

Perhaps the most complex anxiety disorder to be explained with learning theories is panic disorder. In theorizing about and researching panic disorder, it is necessary not only to determine the factors influencing disorder development, but also leading to the first panic attack. Research has shown that many individuals with panic disorder can attribute their disorder to the occurrence of an initial attack. In a study of 162 patients, roughly half of the subjects reported experiencing multiple panic attacks characterized by similar symptoms and similar contexts to their first attack (Craske, Miller, Rotunda, & Barlow, 1990). This implies that either the attack itself or the event triggering the attack, if identifiable, becomes associated with specific cues, which can influence the incidence of further panic attacks. These neutral cues can be either internal or external, and because of their presence during the initial panic attack, become conditioned stimuli. The conditioning of panic disorder can be either exteroceptive, interoceptive, or both, depending on the nature of the conditioned cues. In exteroceptive conditioning, external stimuli that are perceived during the initial panic attack are conditioned to elicit further attacks. Similarly, in interoceptive conditioning, internal stimuli, such as dizziness or shortness of breath that accompany the initial attack are conditioned to elicit a similar response later (Bouton et al., 2001). Agoraphobia is thought to develop because of exteroceptive conditioning, in which environmental stimuli present during an episode of panic are conditioned to elicit a fear response later (Barlow, 2002). However, while these theories describe the process by which an initial panic attack develops into panic disorder, they do not explain why these initial attacks occur, or why only some of those who experience panic attacks develop a disorder. Research has indicated that individuals with panic disorder may exhibit a reduced extinction response. Michael, Blechert, Vriends, Margraf, and Wilhelm (2007) found that when an image was conditioned with an electric stimulus during a laboratory test, that image was appraised more negatively and as a stronger danger signal for those participants with panic disorder than for the control participants. The negative associations made with the conditioned image sustained for a significantly longer time among disordered participants than among control participants, suggesting a longer extinction phase among those with panic disorder. This may contribute to the development of panic disorder following an initial attack, but does not alone account for all variability between affected patients. Bouton et al. (2001) suggest that, like with other anxiety disorders, genetic factors and feelings of environmental control may influence the development of panic disorder, however many questions remain unanswered.

Vicarious conditioning. In addition to classical conditioning, it has also been suggested that anxiety disorders may be the result of vicarious conditioning. Vicarious conditioning theory suggests that by observing the conditioning or modeling of others' behaviors, one can also experience a similar conditioning process. Both human and animal research has indicated that simply observing the fears of others can contribute to phobia development (Cook & Mineka, 1990). This helps to explain why many specific phobias, such as the
fear of snakes, are consistent across individuals and cultures. In a similar fashion, vicarious learning can also account for components of social phobia, as observing a peer experience embarrassment or anxiety in a social situation has been shown to be predictive of later phobia development (Ost & Hugdahl, 1981).

**Cognitive Factors**

Cognitive processes such as the content of thoughts, attribution style, attention bias and catastrophic cognitions may be both a cause and a mediating variable in the development of anxiety pathology. Cognitive factors that may contribute to anxiety are presented here.

**Cognitive distortion**

Beck and colleagues have theorized about the cognitive processes underlying anxious pathology through application of the construct cognitive schemata (Beck & Clark, 1988; Beck, Emery, & Greenberg, 1985). According to Beck and colleagues, individuals develop cognitive schemata through which environmental stimuli are interpreted and emotional information processed. Those who suffer from anxiety, however, have somehow developed impaired cognitive schemata, in which environmental information is processed in an incorrect distorted manner, leading them to interpret reality as perpetually dangerous. These automatic, distorted thoughts trigger the physiological, motor, and affective components of anxiety, leading to inappropriate anxiety. The anxious states are the result of distortion in an individual's ability to accurately process information. Reardon and Williams (2007) examined the relationship between cognitive processing and psychopathology and found that the presence of impaired cognitions were predictive of anxiety and mood disorder symptoms. Beck describes the cognitive schemata as a stable, innate feature of all individuals, but did not theorize on its development or its relationship to other etiological factors, such as heritability or environment. This lack of causal explanation is a limitation found in much of the cognitive research available.

**Attribution style**

An important field of study in cognitive psychology is the role of attribution style in anxiety development. Attribution style describes both the factors to which individuals attribute events, and the stability of these attributions. Some research concerning this construct has found evidence of a correlation with anxiety, in which individuals reporting internal and stable negative attributions were more likely to experience anxious pathology (Hallam, 1985; Hibbert, 1984). In a study of attribution in both severely and mildly socially anxious participants, those with severe social anxiety experienced more symptoms that are anxious during a conversation with a confederate, and made more negative internal attributions about the conversation than did those with mild social anxiety (Lindsay & Stopa, 2008). In a similar study involving participants with OCD, a significant correlation was found between high levels of OCD and stronger negative internal attributions, specifically involving self-blaming for disorder symptoms. These attributions mediated the relationship between OCD symptoms and attempted symptom suppression (Magee & Teachman, 2007).

While such research suggests an association between attribution style and anxiety, Kenardy, Evans, and Oei (1990) found refuting evidence. In their research, a correlation between attribution style and anxiety, specifically panic disorder, was examined to determine whether methods of attribution were etiologically relevant to anxiety. Contrary to their hypotheses and previous research, Kenardy et al. found no evidence of a significant correlation between attribution style and panic disorder. Similar research found no correlations between attribution style and anxiety levels (Dowd, Claiborn, & Milne, 1985; Heimberg, Vemilyea, Dodge, Becker & Barlow, 2005). These researchers suggested the Attribution Style Questionnaire (ASQ) might have accounted for these inconclusive results, explaining that the ASQ may not be a sensitive measure when applied to studies of anxious participants. While this is a
possible limitation, the ASQ is the leading measure of attribution style and has strong psychometric properties (Peterson & Villanova, 1988). Attribution theory may only account for some specific anxiety disorders. Taylor and Wald (2003) looked at attribution style among participants with social anxiety, generalized anxiety disorder, and panic disorder, and found that among these groups, social anxiety was most strongly correlated with negative internal attributions. This suggests that associations between attribution style and anxiety may be specific to diagnosis.

Attentional Biases

Another cognitive distortion that may enhance perceived threat level is attention bias toward threat-related information. Lonigan, Vasey, Phillips and Hazen (2004) propose a model of anxiety development in children that incorporates both temperament and cognitive development. Similar to some adult theories (see below), children with a negative temperament are predisposed to attend the threat-related information. Specific to children, Lonigan et al. propose that younger children do not possess attentional skills sufficient to voluntarily alter their focus away from perceived threat. This voluntary or effortful skill only emerges after cognitive development associated with physiological maturity, specifically synaptic pruning and myelination. A recent review of the literature supports the contention that children with anxiety disorders present an attention bias to threat-related events, but it remains unclear how the development of attention skills vary across ages into adulthood (Puliafico & Kendall, 2006).

Analyses of childhood anxiety are consistent with theoretical models that propose a direct relationship between adult trait anxiety and attentional bias to threat-related words and pictures. For example, Eysenck (1997) proposes that individuals with trait anxiety are generally hypervigilant toward threat-related events in terms of memory, interpretation (c.f., Beck & Clark, 1988; Beck et al., 1985) as well as attention. Williams, Watts, Macleod and Matthews (1997) propose that trait anxiety predisposes individuals to attend to threat-related events whereas Mogg and Bradley (1998) argue that anxious individuals have a low threshold for perceiving an event as dangerous. The emerging empirical question in attention bias research is if and how the relationship between attention bias and anxiety serves to influence initial manifestation and perhaps maintenance of anxiety.

Examples of illustrative research are enlightening. Rinck and Becker (2005) found evidence of an attentional bias in women afflicted with anxiety, in which clinically anxious individuals had a tendency to direct their attention towards threatening stimuli during the early, automatic stages of information processing. Later in the information processing event, when typical individuals focus on threatening stimuli, people with anxiety unconsciously directed their attention away from threat, a form of cognitive avoidance wherein individuals with anxiety disengage from threat-related stimuli that contrasts with their initial orientation towards threatening stimuli. There is also evidence of possible disorder-specific biases in anxiety (Williams et al., 1997). For example, individuals with panic disorder may attend to stimuli associated with physical threat and individuals with social anxiety may attend to stimuli associated with social situations (Asmundson & Stein, 1994; Hope, Rapee, Heimberg, & Dombeck, 1990; Mathews & MacLeod, 1987).

These studies led researchers to question whether such findings were the result of general emotionality, in which participants are distracted simply by emotional words, regardless of specific threatening cues, or the result of schema specificity, in which participants respond only to disorder-specific cues. Investigation by Becker, Rinck, Margraf, and Roth (2001) indicated that most people with anxiety disorders exhibit patterns of schema-related attentional bias. The exception to this finding, however, is GAD, in which an effect of general emotionality has been observed. While it is possible that processes of attention will differ between GAD and other anxiety disorders, it is probable that the nature of GAD symptoms may
influence the biased attention. GAD is characterized by intense, worrisome thoughts, spanning many topics. This broad array of anxiety triggers may account, in part, for the seemingly unschematic attentional biases found in GAD patients.

Research concerning attentional biases also indicates that interactions between processes of attention and environmental stimuli may exist. Shackman, Shackman, and Pollak (2007) examined the associations between attention and abuse, and found that individuals who had experienced childhood physical abuse were significantly more likely to focus both automatic and controlled attention towards threat cues than control participants were. These findings also indicated that the amount of attention allocated to threat cues is significantly predictive of self-reported anxiety level, demonstrating the complex interactions between environmental experiences, individual differences in cognitive processing, and disorder development.

*Catastrophic Cognitions.*

The theory of catastrophic cognitions may also be of etiological relevance. Similar to theories of cognitive distortion, catastrophic cognitions are defined as cognitive misinterpretations of internal, physical symptoms. It is suggested that individuals mistakenly attribute physical sensations, such as dizziness or chest pains, to represent a serious, underlying issue. This misinterpretation leads individuals to fear what they believe to be impending physical or social doom, which in turn causes further fear and anxiety (Marks, Basoglu, Alkubaisy, Sengun & Marks, 1991).

Clark (1988) described the process by which these catastrophic cognitions influence anxious symptomology. According to this research, people first experience an internal bodily sensation, which may be the result either of a naturally occurring physical event, or of somatic anxiety symptoms. This sensation leads the individual to engage in catastrophic thinking, which increases their internal focus and inevitably causes them to notice further bodily sensations. This increased focus on internal sensations both creates anxiety-related somatic symptoms, and increases the individual’s sensitivity to normal body sensations, which would otherwise go unnoticed. This sequence intensifies, and eventually leads to the occurrence of either a panic attack or a state of anxiety. Although research has supported theories regarding the cyclical nature of catastrophic cognitions in the development of anxiety, there are limitations to this idea. First, panic and anxiety have been shown to occur without the presence of catastrophic thoughts (Zucker et al., 1989). In addition to this, the theory of catastrophic cognitions does not describe reasons why people develop these cognitive patterns.

While several studies describe and provide empirical support for theories regarding the development and maintenance of anxiety states, cognitive research is limited by its inability to explain why certain individuals experience pathological cognitions and others do not. Cognitive theories, although empirically relevant, explain the mechanisms by which people exhibit anxiety symptoms and experience state anxiety, but fail to describe the etiology of these tendencies. Despite the theoretical nature of cognitive approaches and the limited body of etiological research, cognitive therapies have been highly effective at helping treat anxiety, and therefore cognitions likely have a role in anxiety development. It is possible that patterns of cognitive functioning help to either elicit pre-existing biological vulnerabilities, or interact with environmental stimuli to help determine human responding to such stimuli. It is most likely that a complex interaction exists between the biological, environmental, and cognitive perspectives.

*Future Directions*

While researchers debate the factors affecting anxiety development, most agree that an interaction between biological, environmental, and cognitive factors exists. While research studies and reviews have examined many potential isolated causes of anxiety, the unit of analysis is important. Rather than focusing on a specific biological, learning, or cognitive process, this integrative review illustrates
the potential synthesis of disparate etiological processes and shows how the literature has been inadequate in making connections between each of the biological, environmental, and cognitive factors. Although research has clearly indicated that each of the aforementioned variables influence anxiety disorder development, most studies have examined only one aspect of anxiety etiology, disregarding the impact of other potential causes. For example, heritability research has generally examined only biologically determined, genetic influences on anxiety development. Such research often acknowledges the possibility of an environmental or cognitive impact on anxious tendencies, but little research has been conducted which takes into account multiple theoretical perspectives. This lack of comprehensive inquiry makes it difficult to draw definitive conclusions about the complex etiology of anxiety, as the nature of the interactions between biology, environment, and cognitive processes are speculative, and based largely on individual interpretation.

Future research would benefit from examination of the discussed perspectives together in unison, investigating the interactions between variables rather than conceptualizing other perspectives as uncontrolled confounding variables. This would provide a more comprehensive analysis of each possible factor in the etiology of anxiety, and better determine the relationships between peoples' biological predispositions, the environments in which they function, and their cognitive processes. Potential areas of inquiry include the interaction between (a) the 5-HTTLPR genotype and childhood maltreatment, (b) the hippocampal region and early aversive events, (c) polymorphisms of the MAOA gene and violent family interactions, (d) exposure to fear eliciting stimuli and the biological process of habituation and (e) threatening and unpredictable events and the cognitive perception of control over those events.

In addition to investigating the interactions between etiological variables, future research would also benefit from further gender-related investigation. Although some research has focused on the differences in anxiety etiology based on gender, the existing literature does not adequately explain the level of these gender disparities or the strength of their impact. It is clear that the prevalence of anxiety greatly differs by gender, with females far more affected than males. Despite this, research concerning etiology as a function of gender is limited and even completely lacking concerning certain theoretical perspectives. For instance, while some gender-related data exists concerning differences in heritability and experiences of control, no sufficient research has been conducted thus far to determine possible gender differences in anxiety conditioning or cognitive processes. In addition to this, much of the existing literature is clouded by opinion and feminist ideals, and therefore it is difficult to draw reliable, scientific conclusions from the results. Because of this lack of research, further studies should investigate not only the interactions between etiological factors, but also how gender differences contribute to the relationships between those factors.

Taken together, the review illustrates the value of idiographic assessment. The nomothetic variables influencing anxiety are wide-ranging and they cross philosophical and empirical perspectives. Clinicians can understand the importance of assessment of individual biological, learning and cognitive variables.

References


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