

# THE COGNITIVE THEORY OF ANXIETY

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ANXIETY AND RELATED DISORDERS



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# The Cognitive Theory of Anxiety

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Within the past three decades, the field of psychology has witnessed a cognitive revolution (Beck, 1991). Traditional behavioral methods of psychopathology and therapy (Wolpe, 1958; 1973), based upon principles of experimental research derived from the animal research paradigm, naturally favored conditioning explanations and paradigms and consequently excluded consideration of cognitive factors. Zinbarg (1990) has argued that traditional behavior therapists have continued to base their conceptualizations upon outdated models of conditioning and have not kept pace with the most recent developments in the field of conditioning. One of the most significant developments in conditioning theory over the past 20 years has been the blending of conditioning and information processing models; that is, even the basic theories upon which behavior therapy was built have subsumed cognitive variables (Zinbarg, 1990). Thus, it seemed quite natural that the emergence of cognitive and cognitive behavioral approaches (Beck, 1967; Ellis, 1962; Mahoney, 1974; Meichenbaum, 1977) would serve as an extension and expansion of black-box approaches.

The view of patients as beings whose thoughts, images, beliefs, expectancies, and assumptions play an influential role in shaping perceptions, thinking, and feeling is now firmly rooted in the field. While there is still some



controversy about whether the addition of cognitive approaches adds anything over and above behavioral approaches for selected problems (Wolpe, 1973), Michaelson (1987) suggests that the use of cognitive techniques within treatment packages may reflect a more state-of-the-art approach to treatment. Others (Simon & Fleming, 1985) have argued for the efficacy of cognitive approaches. At this point while more research is needed to clarify some of these important questions, the cognitive behavioral approach occupies an important position in our understanding of psychopathological states such as anxiety and in the design of clinically useful interventions.

While the earliest roots of the role and importance of cognition in human behavior can be traced to Eastern, Greek, and Roman philosophers (Ellis, 1989), there is little doubt that the theorizing of Aaron T. Beck (1967) has been perhaps the most influential source in guiding and nurturing the cognitive movement. Basing his earliest observations and theorizing on unipolar depression (Beck, 1967), the cognitive model enjoyed wide application to a variety of other disorders (Freeman, 1989).

In this chapter, our goal is to outline and elucidate the cognitive theory and model of anxiety and its disorders. We address the definition of anxiety and anxiety disorders, its epidemiology, basic assumptions of the cognitive theory of anxiety, the role of predisposing and precipitating factors, a

cognitive case conceptualization derived from the cognitive model, and common misconceptions about cognitive theory.

Where relevant, we have used clinical research and examples to illustrate our points.

## **ANXIETY: A DEFINITION**

Three cases that exemplify the typical characteristics of the anxious patient highlight the important aspects of the problem.

### **Case 1**

A well-built 30-year-old white male comes to your office complaining of chronic attacks of anxiety during the past year. He is a supervisor of 46 workers in an extremely stressful, high pressure job. Three weeks ago, his wife delivered their first child. His mother-in-law passed away after a long bout with cancer which has precipitated severe depression in his wife. One year ago, his older brother had a heart attack that began with chest pain. His father died suddenly from a heart attack several years earlier. His mother also had recent unsuccessful cardiac bypass surgery. One year ago at the time of his brother's heart attack, his anxiety attacks began with pain in his chest, a warm rush, and a variety of sympathetic symptoms. He has made several visits to the emergency room despite repeated reassurance from his family

physician. He continues to worry about having future attacks and to be very frightened.

## **Case 2**

A 26-year-old male supervisor at a gambling casino has recently been informed that he will be presenting an award to one of his supervisees who received an Employee of the Month distinction. The thought of standing in front of 300 people and making this presentation absolutely terrifies him. When the day arrives and he begins to make his presentation, he is unable to express himself, is extremely self-conscious, and notices several people in the front row who are laughing at his fumbling. He concludes that he has made an absolute fool of himself and subsequently develops a fear of answering the phone at home and at work. The patient reports a rather long history of interpersonal anxiety, unassertiveness, and self-consciousness in social situations.

## **Case 3**

The husband of a 68-year-old woman calls the family practice center to make an appointment with the psychologist for his wife because of her intense anxiety about a lesion under her right axilla that has a discharge. The patient is totally convinced that she has a recurrence of cancer and is extremely

frightened about being examined by a physician to the point that she has avoided medical care for 35 years. Forty years earlier, she had a double mastectomy and related therapeutic abortion. Since that time, she has extreme anxiety whenever she is in the presence of a physician. The thought of being in a medical setting, talking to a physician, or being examined by a physician who might discover something medically wrong with her creates anxiety of phobic proportions.

## **COMMON ASPECTS OF ANXIETY**

These three cases have commonalities. First, each of these individuals suffered to a significant degree from distressing levels of anxiety. Second, each person's life was compromised in some significant manner by this emotion. Third, in each instance, the individual experienced either a series of life events or some traumatic experience of relevance to their problem. Fourth, there was clear motivation to avoid those situations associated with anxiety or the very symptoms of anxiety itself. Fifth, each individual perceived a great degree of danger or threat in these situations. Finally, those situations that struck at a particular vulnerability of each patient precipitated an anxiety response.

Anxiety may be defined as a tense emotional state characterized by a variety of sympathetic symptoms including, for example, chest discomfort,

palpitations, shortness of breath. Under normal circumstances, the human nervous system is designed to prepare and mobilize the individual to fight or flee from an objective and physically dangerous threat. The hallmark of the anxious patient, however, is the presence of perceived threat and the activation of the physiological concomitants in the absence of objective real threat. In other words, the anxious disordered person sees threat and reacts to threat where no real threat exists.

While anxiety is a universal human experience and is undoubtedly a common human emotion, its evocation does not necessarily imply the presence of a clinically significant disorder. The DSMIII-R (American Psychiatric Association, 1987) is quite explicit about the definition of a disorder that “is conceptualized as a clinically significant behavioral or psychological syndrome or pattern that occurs in a person and that is associated with present distress (a painful symptom) or disability (impairment in one or more important areas of functioning) or with a significantly increased risk of suffering death, pain, disability, or an important loss of freedom (p. xxii).” In effect, a disorder implies a duration, frequency, number, and intensity of symptoms that are significant enough to interfere with a person’s quality of life. The DSMIII-R delineates the following anxiety disorders: Panic Disorder With or Without Agoraphobia, Agoraphobia Without a History of Panic Disorder, Social Phobia, Simple Phobia, Obsessive Compulsive Disorder, Posttraumatic Stress Disorder, Generalized Anxiety

Disorder, and Anxiety Disorder Not Otherwise Specified.

The National Institute of Mental Health Epidemiological Catchment Area Survey (Reiger, Myers, Kramer et al., 1984) revealed that anxiety disorders are the most common mental health problems affecting about 8.3% of the population. These disorders outrank both depression and substance abuse, yet only one of every four patients ever receives treatment.

## **BASIC ASSUMPTIONS**

The cognitive model of anxiety makes several basic assumptions about anxiety, its evocation, its medication, and significance (Beck, Emery, & Greenberg, 1985). These assumptions are crucial in understanding the phenomenon of anxiety and the nature of anxiety disorders.

1. Anxiety, an emotional response, has adaptive significance for humans when evoked in response to objective danger. (Beck, Emery, & Greenberg, 1985; Canon, 1929; Emery & Tracy, 1987; Izard & Blumberg, 1985; Lindsley, 1952; 1957; 1960; Plutchik, 1980)
2. The evocation of anxiety in response to misperceived or exaggerated danger when there is none is maladaptive. (Beck, Emery, & Greenberg, 1985; Beck & Greenberg, 1988; Foa & Kozak, 1986)
3. Anxiety disordered individuals are prone to precipitate false alarms

that create a relatively constant state of emotional tension and subjective distress. (Barlow & Cerney, 1988; Beck, Emery, & Greenberg, 1985; Beck & Greenberg, 1988)

4. During episodes of anxiety, an individual's cognitive, physiological, motivational, affective, and behavioral systems are involved. (Persons, 1989; Taylor & Arnow, 1988)
5. The cognitive system plays a vital and essential role in appraising danger and resources and activating the physiological, motivational, affective, and behavior systems, each of which serve important functions. (Beck, Emery, & Greenberg, 1985; Foa & Kozak, 1986; Lazarus, 1991)
6. The cognitive system mediates its influence through repetitive unpremeditated and rapid involuntary thoughts and/or images of which the individual is unaware (unless attention is called to them) and which the individual accepts without question. (Beck, Emery, & Greenberg, 1985; Beck & Greenberg, 1988; Emery & Tracy, 1987)
7. Automatic thoughts are derived from underlying deeper cognitive structures called schemas, also referred to as underlying beliefs or assumptions. (Emery & Tracy, 1987; Foa & Kozak, 1986; Kendall & Ingram, 1987; Persons, 1989)
8. Automatic thoughts and underlying beliefs are disorder specific and, in anxiety disordered individuals, reflect themes of threat and danger as opposed to themes of loss in depressed individuals. (Beck, Emery, & Greenberg, 1985; Beck & Rush, 1975; Beck & Weisher, 1989; Foa & Kozak, 1986; Hilbert,

1984)

9. Anxiety reactions and disorders may be more fully and parsimoniously understood by elucidating the individual's automatic thoughts, cognitive distortions and underlying assumptions. (Beck, 1976; Butler & Matthews, 1983; Deffenbacher, Zwemer, Whisman, Hill, & Sloan, 1986; Freeman, Pretzer, Fleming, & Simon, 1991; Marluzzi & Bollwood, 1989; Zwemer & Deffenbacher, 1984)
10. In trigger situations, anxiety disordered individuals have a tendency to activate danger/threat schemas by which they selectively screen in stimuli that indicate danger and screen out those stimuli that are incompatible with danger. (Beck, 1976; Beck, Emery, & Greenberg, 1985; Freeman, Pretzer, Fleming, & Simon, 1991)
11. Anxiety disordered individuals have impaired objectivity and ability to evaluate their threat bound cognitions in a realistic manner. (Beck, Emery, & Greenberg, 1985)
12. Anxiety disordered individuals exhibit systematic errors in processing information by, for example, catastrophizing, selectively abstracting, thinking dichotomously, and making arbitrary inferences. (Beck, Emery, & Greenberg, 1985)

The cognitive model of anxiety also makes explicit assumptions about the predisposing and precipitating factors that are associated with the onset of anxiety disorders. In the sections that follow, we will discuss several



predisposing and precipitating variables related to anxiety disorders. It is important to bear in mind that any combination of these factors may set the stage and provide the impetus for the development, onset, maintenance, and exacerbation of anxiety problems.

## **PREDISPOSING FACTORS**

According to cognitive model (Beck, Emery, & Greenberg, 1985), there are five possible factors that may predispose or make an individual potentially vulnerable and more prone to anxiety and anxiety disorders. These factors are: (1) genetic inheritability, (2) physical disease states, (3) psychological trauma, (4) absence of coping mechanisms, and (5) dysfunctional thoughts, beliefs, assumptions, and cognitive processing. We will discuss each of these factors in detail. As a result of individual differences, an anxiety disorder may result from a unique combination of predisposing and precipitating variables (Beck, Emery, & Greenberg, 1985).

### **Genetics**

Within recent years, the role of possible genetic factors in certain psychopathological disorders have assumed more importance. Anxiety disorders are no exception. Some authors (e.g., Sheenan, 1983) have even gone to the extreme of viewing it as a strictly biological disease necessitating

pharmacological therapy.

Cognitive therapists consider hereditary predisposition to anxiety as an important variable. Panic disorder, phobic disorders, and obsessive-compulsive disorder are more common among first-degree biological relatives of patients suffering from these disorders (American Psychiatric Association, 1987). However, the role of genetic factors in generalized anxiety disorder is less clear (Beck, Emery, & Greenberg, 1985). Nonetheless, the question about how heredity exerts an influence in anxiety disorders is important to consider in the cognitive conceptualization of anxiety. Heredity may manifest its influence by the existence of an easily aroused or labile autonomic nervous system (Barlow & Cerney, 1988). In other words, in certain anxiety conditions, a family history of the disorder may make it more likely for a patient to exhibit anxiety symptoms under the right set of conditions. Barlow and Cerney (1988), for example, have thoroughly examined the possible role of genetic factors in panic disorder. Research has supported the aggregation of panic in monozygotic twins (Torgerson, 1983) and families (Crowe, Noyes, Pauls, & Slymen, 1983; Harris, Noyes, Crowe, & Chaudry, 1983; Moran & Andrews, 1985) and is supported by clinical observation. For example, one of us (RDT) treated an elderly woman with a long history of panic disorder whose daughter exhibited an identical problem and was being treated independently. The role of genetic vulnerability cannot be fully appreciated without considering the interactive role of environmental

psychological and social factors (Barlow & Cerney, 1988). Genetics may be viewed as transmitting the biological substrate that makes the individual more vulnerable to develop clinical anxiety. As such, the cognitive model appreciates the role of possible genetic influences in anxiety disorders.

## **Physical Disease**

The cognitive model also considers the possible role of physical factors in making an individual vulnerable to an anxiety disorder. There are two issues to consider: First, ruling out possible physical causes that can mimic anxiety is essential in assessing anxiety disorders. In many instances, treating the physical problem may alleviate the symptoms. Second, however, the existence of a physical problem does not necessarily rule out the existence of an anxiety problem. A physical problem can coexist with an anxiety disorder and treating both problems may be necessary. Barlow and Cerney (1988) provide a list of nine organic conditions that are associated with anxiety symptoms and panic. These physical conditions include hypoglycemia, hyperthyroidism, hypoparathyroidism, Cushing syndrome, pheochromocytoma, temporal lobe epilepsy, hyperventilation syndrome, caffeine intoxication, audiovestibular problems, and mitral valve prolapse.

## **Mental Trauma**

The third possible predisposing factor is a mental trauma during development (Beck, Emery, & Greenberg, 1985) that can render an individual more vulnerable to experience anxiety in situations similar to the experience of the trauma. The cognitive model assumes that developmental traumas occurring in the context of high emotional arousal can result in an individual developing a threat schema. Such schemas would presumably relate to themes of danger in anxious patients and would be expected to become activated in situations that are similar to the circumstances in which the schema was learned. As Foa and Kozak (1986) noted, “A fear memory is accessed when a fearful individual is presented with fear information that matches some of the information structure in memory” (p. 23). According to their emotional processing model, fear is expressed as a memory network that incorporates information about the stimulus situations, responses, and the meaning of the stimuli and responses. Fear structures by definition involve themes of danger. One of us (RDT) treated a woman with a 25-year history of panic disorder with agoraphobia whose anxiety attacks were traced to an experience when she was locked in the trunk of a car by her brother and a friend as a prank, and learned to fear suffocating to death. Subsequently, any situations that resembled enclosed places from which escape might be difficult (e.g., planes, buses, cars, elevators) and stuffy odors were associated with the threat of suffocation which precipitated extreme anxiety.

Another predisposing factor in the development of anxiety disorders is

a deficit in coping responses. Anxiety disordered patients often exhibit deficits in adaptive coping strategies. Not only are their primary appraisals of situations more likely to result in perceptions of threat where no threat exists, but their secondary appraisals of their resources to cope with threat more often reveal inability to cope. Anxiety patients may have failed to learn adequate coping strategies or employ responses such as avoidance that strengthen their anxiety and preclude effective coping. As a result, they leave themselves vulnerable to experience anxiety in the presence of life events or other stressors.

### **Irrational Thoughts, Assumptions and Cognitive Processing Errors**

The cognitive model of anxiety places primary emphasis on the role of cognitive factors in predisposing individuals to anxiety disorders. In anxiety disordered individuals, underlying unrealistic beliefs about threat or danger are presumed to be activated by trigger events or situations that are similar to situations during which these schemas are learned. When these schemas are activated, they fuel the patient's thinking, behavior, and emotion, all of which can serve to reciprocally reinforce each other as well as the underlying schema. Persons' (1989) case conceptualization model provides an excellent approach for elucidating the central role of cognitive factors in predisposing individuals to anxiety.

## PRECIPITATING FACTORS

The cognitive model of anxiety posits a number of possible factors that may precipitate anxiety: physical disease or toxic substances, severe external stressors, long-term stress, and stressors affecting a specific emotional vulnerability of an individual.

### **Physical Problems or Toxic Substances**

Anxiety can be precipitated by the onset of a physical problem that does or does not mimic anxiety. For example, the development of anxiety after the onset of a physical problem is not an uncommon reaction during an individual's attempt to adjust to illness. Physical problems may cause symptoms such as fatigue or depression that could compromise or overtax the individual's tolerance for handling even normal, every day stressors; the result may be that previously handled stressors overburden the individual's resources. In addition, it is quite possible that a physical problem may present an individual with an array of symptoms that are viewed as signs of a serious problem when in actuality the problem is relatively benign.

Finally, there are those instances where individuals have ingested a psychoactive substance that produces some physical effect that is interpreted as threatening. In our clinical experience, we have encountered patients who developed anxiety attacks after using marijuana or cocaine, or even after

unintentionally inhaling toxic fumes. Perhaps even more interesting is the situation in which an individual consumes large quantities of caffeine. One of us (RDT) recalls the case of a young female paramedic who presented at a medical outpatient center reporting panic-like symptoms. She indicated that she had recently been seen in the emergency room after losing consciousness at home. She also claimed that she required CPR from her paramedic brother after she stopped breathing at home. What made her story even less credible in this litigious age was that she was sent home from the emergency room after spending only a short time there. Her physical and medical history were unremarkable. However, further probing revealed that she was drinking between 12 to 15 cups of caffeinated coffee on a daily basis. Her symptoms subsided with the gradual withdrawal of caffeine over time.

### **Severe External Stressors**

The occurrence of a severe stressor or life event such as loss of a loved one or loss of a job is another possible precipitant of anxiety. The role of life events (Last, Barlow, & O'Brien, 1984) in precipitating anxiety reactions is well known.

### **Chronic Insidious External Stressors**

Stressors may be cumulative over a long period of time and in a sense

may piggyback on each other. The result may be a situation in which a person's coping resources are exhausted and overwhelmed. One of us saw a woman who was experiencing profound distress following a series of events including chronic cystitis, two carpal tunnel surgeries, two car accidents, severe neck injury, cancer in her father followed by his eventual death, and home problems.

## **Vulnerability**

Stressors may also strike at an individual's particular emotional vulnerability. What may precipitate anxiety in one person may not do so in another. To partially account for this, we would infer and test whether an individual suffers from a particular vulnerability. For example, consider an individual who believes that to be worthwhile, one must be loved by everyone. As long as this individual receives acceptance from others, we may not expect him to become symptomatic. The rejection by a lover may precipitate an emotional reaction.

## **THEORY AND CASE CONCEPTUALIZATION**

The relationship between cognitive theory and case conceptualization is clearly provided by Persons (1989). Theory should guide clinicians in assessing, planning, implementing, and evaluating treatment. In this section,



we use Persons' (1989) model to highlight how the cognitive model and theory of anxiety can be helpful.

One of us (RDT) treated P, an elderly woman with a 15-year history of panic disorder. Following the cognitive model and Persons' formulation model, the following areas were identified: problem list, behavior, cognitive factors, hypothesized mechanism, relationship between mechanism and problem, current precipitants, and predicted obstacles to treatment. This conceptualization emphasizes the interaction between the predisposing and precipitating factors.

## **Cognitive-Behavioral Case Formulation Example**

### I. Problem List

#### A. Feelings

1. *Panic Attacks*: P. was experiencing the sudden onset of intense fear and accompanying panic attacks several times a week at the beginning of therapy. These attacks were interfering significantly with the quality of life and provoked a great deal of fear.
2. *Generalized Anxiety*: She also suffered from a chronic sense of generalized anxiety characterized by anticipation of the next attack. She lived in anticipation of experiencing fear provoking thoughts.

3. *Depression*: P. was experiencing a dysthymia secondary to her panic problems. She was constantly aware of the demoralizing effect that the panic attacks had upon her quality of life. There was also a sense of loss and guilt related to her view that she had changed from the person everybody knew and was now a burden on her family.

## B. Behavior

1. *Fear-Provoking Situations*: While P. exhibited no avoidance of situations, she was fearful of being alone at times. She was also fearful about the possibility of having an attack in the presence of her family. This created a great deal of anticipatory anxiety.
2. *Difficulties in Relationships with Family*: A significant source of distress is the negative effect that her problems have had on her family. Family members felt that “all she wants is attention” and attributed her problems to the fact that she “doesn’t want to live alone.” There was also some indication of resentments toward her family that she was unable to verbalize.
3. *Lack of Assertiveness*: P. had assertiveness deficits which seriously undermined her ability to obtain social reinforcement. Her dependent features also interfered with her ability to assert herself for fear of alienating family members on whom she was dependent.

## C. Cognitive

1. *Cognitive Distortions*: P. exhibited a variety of cognitive distortions, most especially catastrophizing. She tended to misinterpret her symptoms as cause for threat to her mental status. She also exhibited a variety of other distortions such as selective abstraction and jumping to conclusions. These distortions typically escalated her anxiety symptoms to panic proportions and also fueled her dysthymias.
  
2. *Suicidal Ideation*: P. reported thoughts at times of wishing she were dead and demoralization about her ability to cope with the panic for the rest of her life. She openly reported these thoughts and was willing to discuss them. On several occasions, a thorough suicide assessment was found to be negative.
  
3. *Decreased Self-Efficacy*: P. had little belief in her ability to do what she could do to resolve her problems. This may have been her reason for choosing medication initially. She had been treated with medication for such a long time that her attributions about improving were externalized.

## II. Hypothesized Mechanisms

1. *Cognitive*: P. had a variety of underlying schemas that fueled her problematic thoughts, feelings, and behaviors. Some examples of her core schema were as follows:

*Vulnerability*:

"Something terrible will happen to me at any moment, and I will lose control and go crazy."

"I will forget who I am and forget my family."

"If I experience too much anxiety, I will lose my identity."

*Dependence:*

"I am unable to cope on my own."

"If I don't please others, they will abandon me."

*View of Self Environment and Future:*

"I'll never improve."

"I'm inadequate."

"My problem is so bad that no one can help me."

2. *Social:* Although P. viewed herself as an independent person, it was very clear from her social history that she was extremely dependent on her husband. She apparently never had to rely upon herself as he "did everything for me." She was also socialized in a society that placed women in her age group in a dependent role.

3. *Biological:* A possible underlying biological mechanism in P. is suggested by the fact that one of her daughters suffered from panic disorder. This finding suggests some

possible genetic vulnerability to panic in the face of stressors.

III. *How the Mechanisms Produce the Problem:* P.'s panic attacks were possibly precipitated as a result of a variety of mechanisms including a number of stressful life events, a possible biological vulnerability, and the activation of strongly held beliefs about her vulnerability to threat. She appeared to be a rather dependent woman with low self-efficacy about her ability to cope. Her extreme dependence on her husband buffered her against the effects of stress. Following his death, a major stressor, basic schemas about her vulnerability were probably activated and fueled her negative thoughts, feelings, and behaviors, and increased the likelihood of a panic attack. When she experienced this attack, she was extremely frightened by it and interpreted it as a sign of her vulnerability to losing control and going crazy. Her distorted style of processing information exacerbated her anxiety, fueled her anticipation and generalized anxiety, demoralized her, and precipitated anxiety in certain situations. The patient found herself in a vicious cycle of precipitating the very symptoms she feared. Probably as a result of her low self-efficacy, she sought a "pill" to solve her problems. Her initial physician placed her on a low-potency benzodiazepine of questionable efficacy which may have contributed to her depression. She also exhibited several depressogenic assumptions that fueled her depression, undermined her hopefulness, and precipitated suicidal thinking. P.'s difficulties with her family related to her underlying beliefs about pleasing others and inability to assert herself.

IV. *Current Precipitants*: The original precipitant of P.'s panic related to a situation in which she misinterpreted a benign experience as a sign of an impending catastrophe. One might hypothesize that P. was socialized as a dependent weak person who is unable to cope on her own.

V. *Predicted Obstacles to Treatment*: A number of factors were hypothesized that could interfere with treatment. First, there was evidence that the patient was nonadherent with appointment keeping for her medical visits. Second, her view of medication and externalization of sources of improvement could undermine her participation in therapy.

## COMMON MISCONCEPTIONS ABOUT THE COGNITIVE MODEL OF ANXIETY

There are a variety of common misconceptions about the cognitive model of psychopathology (Freeman, Pretzer, Fleming, & Simon, 1990) and anxiety in general. Next we address each of these myths and provide a more accurate description about what the cognitive model implies.

1. *Faculty cognitions cause anxiety disorders*. This misconception is perhaps the most commonly cited unjustified criticism of the cognitive model. The cognitive model does not assume that thoughts cause anxiety disorders. Rather, this model proposes that a variety of predisposing and precipitating factors including cognitive patterns may coexist and relate to the development of anxiety disorders. Cognitions and cognitive processing are not all that are important but do represent a useful focus for intervening.

2. *The cognitive model is simply a variant of the Norman Vincent Peale power of positive thinking approach.* The cognitive model of anxiety assumes that anxiety disordered individuals have a tendency to perceive threat where no danger exists. Anxiety patients exhibit unrealistic thinking and are unlikely to respond to positive reassuring thoughts. Many of these patients have had numerous individuals including family, friends, and even their physician encourage positive thinking to no avail. The model proposes that patients must learn to evaluate the triggers for anxiety in a realistic valid manner.
  
3. *The cognitive model denies the importance of behavioral principles such as exposure in overcoming anxiety.* While the cognitive model of anxiety views that there is a basic problem in the cognitive apparatus of the patient, it is simply untrue that the model overlooks the importance of behavioral principles. In fact, Freeman & Simon have noted that the model might more appropriately be referred to as the cognitive-behavioral-emotive model. It is true that the model places primary emphasis upon the cognitive aspects but most certainly does not ignore the importance and role of behavior and emotion. Cognitive therapists freely use techniques that are designed to modify behavior (e.g., assertiveness training) and emotions (e.g., relaxation therapy).
  
4. *Applying the cognitive model is simply a matter of talking patients out of their fears and worries.* The cognitive approach actively relies upon the principles of collaborative empiricism and guided discovery. The model assumes that

the Socratic approach through which patients are lead through questioning to examine and alter faulty cognitions and underlying beliefs teaches the patient a process that they can take with them. Cognitive therapists do not talk patients out of their problems by persuading or cajoling them to adopt a new perspective. Rather, cognitive therapists talk to patients in ways that assist them in guiding themselves to think, act, and feel more realistically and adaptively.

## CONCLUSION

In summary, from our perspective, the cognitive model of anxiety appears to be both a viable and useful vehicle for furthering our understanding of the complex phenomenon of anxiety and the onset development, exacerbation, and treatment of anxiety-related disorders. Continued clinical research designed to further test and refine the hypotheses of the cognitive theory of anxiety is warranted. Likewise, we await further research aimed at more carefully delineating and clarifying the possible role of cognitive factors in the treatment of anxiety disorders.

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