

Anxiety and Obsessive-Compulsive Disorders

In Chapter 5, we saw that severe stress can precipitate psychological and physiological disturbances, some of which clear up when the stress is removed and some of which do not. Among the emotional consequences of stressors are fear and anxiety, although stress-related reactions may involve other emotional states as well. In this chapter, we review the *DSM-5* disorders in which fear and anxiety are central to the diagnosis. One danger in following a system such as the *DSM* too uncritically is that it can leave the impression that we are dealing with a series of discrete and separate disorders, which is not always the case. People can, and often do, show mixtures of anxiety symptom patterns that also overlap with the mood disorders. Still, anxiety symptoms are quite frequent in the United States; based on epidemiological studies (e.g., Kessler, Burgland, et al., 2005), the category of anxiety disorders is the most common of the *DSM-5* diagnoses, with lifetime prevalence rates of over 28% of the population.

6.1 Fear and Anxiety

Charles Darwin's description of fear, written more than a century ago, remains an excellent portrayal of this emotion:

Fear is often preceded by astonishment, and is so far akin to it, that both lead to the senses of sight and hearing being instantly aroused. In both cases the eyes and mouth are widely opened and the eyebrows raised. The frightened man at first stands like a statue motionless and breathless, or crouches down as if instinctively to escape observation.

The heart beats quickly and violently, so that it palpitates or knocks against the ribs; but it is very doubtful whether it then works more efficiently than usual, so as to send a greater supply of blood to all parts of the body; for the skin instantly becomes pale, as during incipient faintness. ... In connection with the disturbed action of the heart, the breathing is hurried. The salivary glands act imperfectly; the mouth becomes dry and is often opened and shut. I have also noticed that under slight fear there is a strong tendency to yawn. One of the best-marked symptoms is the trembling of all the muscles of the body; and this is often first seen in the lips. From this cause, and from dryness of the mouth, the voice becomes husky or indistinct, or may altogether fail. (Darwin, 1873/1955, pp. 290–291)

Humans, as well as most mammalian species, are born with the innate capacity to experience fear. Although neurophysiological systems in the

What are the most common mental disorders in the United States?

brain are associated with this emotion, we ordinarily infer its existence more indirectly from three kinds of data: (1) reports of subjective experiences of apprehension, such as dread,

fright, tension, inability to concentrate, and the desire to flee a particular situation, as well as physical sensations, such as a pounding heart or sinking feeling in the pit of the stomach (reports of this kind are, of course, limited to individuals who have the capacity to verbalize



(Wikimedia Commons /Charles Darwin)

these experiences); (2) behavioral manifestations, such as flight, disorganization of speech, motor incoordination, impairment of performance on complex problem-solving tasks, or immobilization, as in being “paralyzed by fear”; and (3) measurable physiological responses, such as rapid and irregular heartbeat and breathing, palmar sweating, dry mouth, dilated pupils, and muscular trembling. The physiological responses largely reflect activation of the sympathetic nervous system; however, some responses, such as diarrhea and increased frequency of urination, are produced by parasympathetic arousal. It is perhaps more accurate to say that the autonomic nervous system is thrown out of balance, with both subdivisions showing wide swings in activation.



Anyone who has seen terror expressed by a frightened young child who has not yet been taught by society to conceal fear can appreciate the reality and potential intensity of this fear. (Shutterstock)

A variation that may occur in acute fear is **fainting**. The physiological component in the fainting response, in contrast to the mixed sympathetic-parasympathetic pattern just described, is largely parasympathetic, involving abrupt dilation of the blood vessels in the viscera, slowing of the heartbeat, a drop in blood pressure, and loss of muscle tone. These effects result in a sharp decrease in the blood supply to the brain and

produce loss of consciousness. The parasympathetic-dominated fainting response is likely to occur only in strong, acute fear states.

Most of us have a pretty good idea of what an extreme fear response is like either from our own experience or from observing others. Anyone who has seen the terror expressed by a frightened young child who has not yet been taught by society to conceal fear can appreciate the reality and potential intensity of this emotion. In humans, the fear response, regardless of whatever unlearned tendencies exist for certain stimuli to elicit fear, is a highly learnable response that can become associated with almost any situation or stimulus (external or internal) that happens to be present when the fear occurs. It is also likely to be powerfully influenced by observational learning.

A distinction is usually made between the responses of fear and anxiety. Perhaps Darwin would have agreed with the notion that **fear** is an adaptive state for dealing with a real threat or danger, while **anxiety** is a chronic fear sensation that is not clearly associated with any specific stimulus.

Fainting

Loss of consciousness, slowing of heart rate, and drop in blood pressure often associated with acute fear states

Fear

A sense of dread, terror, or fright

Anxiety

A sense of worry, concern, or apprehension

Panic attack

Rapidly developing sense of intense fear and anxiety

6.2 Anxiety Disorders

6.2a Characteristic Symptoms

The *DSM-5* classifies the different anxiety disorders based in part on the occurrence of certain components of the anxiety symptom complex. One of these components is the **panic attack**, involving a rapid onset of at least four of the symptoms shown in Table 6-1 Symptoms of a Panic Attack—such as pounding heart, sensations of choking or suffocation, trembling, and fear of losing control. The attack progresses to a peak within 10 minutes of onset. People undergoing a panic attack feel intense discomfort; some think during the first such experience that they are dying. (Frequently, the sufferer describes the experience either as a “nervous breakdown” or as “losing one’s mind.”) Until children reach puberty, it is relatively rare for them to experience panic attacks; attacks are much more common among adults in the general population, where the 12-month prevalence is nearly 1 in 9 (American Psychiatric Association, 2013).

Table 6-1 DSM-5 Symptoms of a Panic Attack

Heart palpitations or accelerated heart rate
Trembling or shaking
Sweating
Chest pain
Shortness of breath or sensation of suffocating
Feelings of choking
Dizziness
Numbness or tingling sensations
Chills or heat sensations
Nausea
Derealization or depersonalization
Fear of losing control
Fear of dying

Source: American Psychiatric Association, 2013.

Panic attacks are not, themselves, a diagnosable disorder; however, they play a role in several anxiety disorders, depending on how they are experienced. Sometimes panic attacks are expected—that is, they are cued by or attached to situations—and a recurrence of the trigger (or its anticipation) can produce another attack. For example, specific phobias and social phobias may involve panic attacks associated with particular stimuli, such as blood or public attention. In other disorders (e.g., panic disorder), the attack is unexpected and seems to occur without a cue, or “out of the blue.” People who experience panic attacks, especially unexpected episodes, live in fear of having another panic attack. Many of the anxiety disorders in the *DSM-5* can involve panic attacks, but all involve intensely uncomfortable fear or anxiety as the main presenting symptom. As in the case with all *DSM* conditions, the disorders cause interference with or impairment of normal functioning.

The reliability of anxiety disorder diagnoses improved under the *DSM-IV*, with “good” reliability noted for both panic disorder and generalized anxiety disorder (Brown et al., 2001). However, the *DSM-5* field trials produced a disappointing kappa of only .20 for generalized anxiety disorder; other anxiety disorders were not assessed (Regier et al., 2013). An overview of the main anxiety disorders in the *DSM-5* is listed in Table 6-2.

Table 6-2 DSM-5 Anxiety Disorders

Disorder	Key Symptoms	Minimum Duration Required for Diagnosis	Sex Ratio
Separation anxiety disorder	Excessive anxiety concerning separation from home or caregiver	4 weeks for children/adolescents, 6 months for adults	More common in females
Selective mutism	Consistent failure to speak in certain situations but not in others	1 month	Equal
Specific phobia	Marked fear or anxiety cued by specific objects or situations	6 months	More common in females
Social anxiety disorder (Social phobia)	Marked fear or anxiety cued by social or performance situations	6 months	More common in females
Panic disorder	Recurrent unexpected panic attacks	1 month	More common in females
Agoraphobia	Marked fear or anxiety of being in situations from which escape would be difficult if incapacitating or embarrassing symptoms occur	6 months	More common in females
Generalized anxiety disorder	Excessive anxiety and worry about a number of events or activities	6 months	More common in females

Source: American Psychiatric Association, 2013.

Gender Differences in Anxiety Diagnoses

As can be seen in Table 6-2, anxiety disorders are generally much more common among females than males. In the case of some subtypes (for example, panic disorder), the female-to-male ratio may be as high as 2:1 (American Psychiatric Association, 2013). Although biased application of the diagnostic label is possible, it is unlikely that this would explain persistent differences in prevalence of this magnitude or of similar gender differences in mood disorders. (Males have much higher prevalence rates of some disorders, such as those involving violent behavior and the sexual paraphilias.) It is possible that females with anxiety disorders come to the attention of therapists more often than males do or that males underreport the incidence of anxiety. Another possible explanation for the gender differences is that females



Male-female behavior differences may be related to differential parental investment during reproduction. Because natural selection favors females who avoid dangerous situations, females would be more prone than men to experience emotions that remove them from harmful and dangerous situations. (iStock)

are differentially subjected to more frequent traumas, such as rape or sexual abuse, that could provoke anxiety-related conditions. However, precipitating traumatic events do not form the basis of diagnosis for most of the anxiety disorders. Weich, Sloggett, and Lewis (1998) did not find support for the proposal that gender differences in prevalence could be explained by the type of social roles (such as traditional caring and domestic ones) that females, rather than males, tend to occupy. It is, of course, possible that biological differences between males and females in terms of HPA reactivity confer different levels of stress and anxiety or that hormonal distinctions related to reproductive physiology are responsible for higher female prevalence in anxiety disorders (Howell, Castle, & Yonkers, 2006).

An interesting evolutionary speculation for gender differences in anxiety disorders comes from Anne Campbell (1999). Along with other evolutionary theorists, she noted that many male-female behavior differences may be related

to differential parental investment during reproduction. Although the male contribution to reproduction can be as little as a few minutes, females must carry a pregnancy, nurse the child, and usually invest years into raising offspring. According to Campbell, in tribal societies similar to early human groups, the death of either parent increases the risk of death to offspring, but the death of the mother results in a much greater risk of infant mortality than the death of the father. For that reason, natural selection would more strongly favor females who avoid dangerous or harmful situations or direct conflict, since their children would be more likely to survive. One result, she suggested, is that women would be more prone than men to experience fear and anxiety, emotions that would tend to remove them from harmful and dangerous situations. Males, on the other hand, benefit reproductively from multiple partners and from successful battles over resources and access to females. These tendencies might lead to male overrepresentation among disorders related to sexual behavior, dangerous activities, and physical aggression.

Whatever the reasons for gender differences in prevalence of mental disorders, they have been consistently reported since the *DSM-III* and will be noted as each mental disorder is described in the coming chapters. For now, we turn to a closer examination of the anxiety disorders.

6.2b Panic Disorder

Panic disorder is characterized by recurrent, spontaneous and unexpected panic attacks (see Table 6-1 *DSM-5* Symptoms of a Panic Attack), along with anxiety about future attacks and their consequences, including having a heart attack, losing control, or “going crazy.” The panic attacks are not due to another medical condition or the effects of a substance, and they have been followed by a month or more of persistent concern that the attacks will recur.

They may also be followed by significant changes in behavior related to avoiding another attack. These behavioral changes might include avoiding certain activities (such as physical exertion) and responsibilities (such as going to work), in the hopes of reducing the chance of an attack. In some cases, the sufferer is anxious about being in places from which escape would be difficult should the next panic attack begin (see agoraphobia). Up to one third of sufferers experience nocturnal panic attacks, in which they wake from sleep in a panic state (American Psychiatric Association, 2013).

Not uncommonly, those with panic disorder overreact to mild physical symptoms and often expect those symptoms to develop into serious or catastrophic events such as a stroke. Worry and apprehension about health can extend to comorbid problems including depression, generalized anxiety, and illness anxiety disorder. Sufferers may view their situation as the result of personal failure or weakness. Onset is typically between adolescence and the mid-30s, and the course is highly variable. Although almost any pattern of attacks is possible, the most common appears to be a chronic one of waxing and waning symptoms (American Psychiatric Association, 2013).

Panic disorder appears to be fairly common, affecting up to 5% of the population at some point in life (Roy-Byrne, Craske, & Stein, 2006). As noted earlier, panic disorder is more common in women by a factor of about 2:1. Especially in men, incidence appears to be much higher among homosexuals and bisexuals than among heterosexuals (Cochran, Sullivan, & Mays, 2003). The *DSM-5* cites a 12-month prevalence estimate of 2%–3% in adults and adolescents, with higher rates among American Indians and non-Latino whites than other ethnic groups. Prevalence appears to peak in adulthood and decline thereafter (American Psychiatric Association, 2013).

Causal Factors

Several biological factors may increase the risk of developing panic disorder. It is known to run in families, with first-degree relatives of panic disorder victims up to 8 times more likely to also show the disorder (American Psychiatric Association, 2000). Twin studies also support a modest genetic component, although any genetic mechanism remains unknown (Roy-Byrne et al., 2006). Panic attacks involve intense activity in parts of the brain involved in fear and emotional arousal, such as the amygdala. Thus, a possible biological hypothesis involves an oversensitivity of the brain's fear network in those who are most susceptible to the disorder, although the evidence for this as a causal factor is indirect. Another possible factor involves the inhibitory neurotransmitter GABA, which inhibits anxiety. GABA activity is low in some parts of the cortex among those with panic disorder (Goddard et al., 2004). Panic attacks can be triggered in panic disorder individuals more easily than in normal individuals by infusion of sodium lactate (resembling the lactate that is produced by the body during muscular exertion), by ingestion of stimulants such as caffeine, or by inhaling carbon dioxide (Barlow, 2002). These “physical challenge” data have been used to argue for a biological foundation for panic disorder. Increased risk of panic disorder also exists for smokers and for those with respiratory disturbances such as asthma (American Psychiatric Association, 2013).

Cognitive models (e.g., Beck & Emery, 1985) propose that panic disorder stems from a tendency to make catastrophic misinterpretations of physiological sensations. The susceptible individual may become aware of increased heart rate, which he or she then misinterprets negatively as the early signs of an onset of a panic attack. Engaging in these catastrophic thoughts and expectations then serves to generate the full-blown panic attack, in turn justifying the original catastrophic interpretation.

Bouton, Mineka, and Barlow (2001) proposed that, although catastrophic thinking may occur, a conditioning model of panic disorder more readily accounts for both the challenge study outcomes and the cognitive misinterpretations. Panic attacks are preceded by early internal physiological (**interoceptive**) cues, such as arousal and increased heart rate, as well as **exteroceptive** cues in the environment (e.g., crowds or specific social situations). The anxiety

Interoceptive

Relating to internal, physiological stimuli

Exteroceptive

Relating to external, environmental stimuli

associated with the attack becomes conditioned to those early cues, especially in people with an associative history of uncontrollable or unpredictable negative events. The conditioned anxiety elicited by the early interoceptive and exteroceptive cues serves to both predict and potentiate the developing panic symptoms. Anxiety then becomes a conditioned stimulus predicting the next panic attack, which further increases anxiety, spiraling into panic disorder.

How do different paradigms attempt to explain the development of anxiety disorders?

Psychodynamic models of panic disorder emphasize internal conflicts rather than biological predispositions or learning history as critical to the development of panic disorder, as they do with all of the anxiety disorders. Freud's original focus was on the sexual and aggressive impulses that produced anxiety in the ego. Ego defenses normally contain the anxiety through defense mechanisms, but these are overwhelmed if the unconscious conflict is too threatening. Because functions involving the unconscious are difficult to test empirically, little research exists to support a psychodynamic model of panic disorder.

Treatment for Panic Disorder

From the biological perspective, treatments for panic disorder have included minor tranquilizers such as the benzodiazepines, which are GABA enhancers and reduce anxiety and panic symptoms. However, medications such as diazepam (Valium®) and alprazolam (Xanax®) may produce **dependence**, and most patients experience a **relapse** when the medications are discontinued. Sometimes a “rebound effect” occurs (Chouinard, 2004), in which the relapse following drug discontinuation can involve anxiety more severe than that at the time treatment began. Antidepressant medications, especially the SSRIs, have been used effectively in treating panic disorder without the risk of dependence associated with the benzodiazepines. However, they do not work as quickly, requiring a few weeks before benefits are experienced. Side effects and relapse after discontinuation remain as problems.

Psychotherapy for panic disorder is effective. A meta-analysis of 17 controlled treatment studies conducted between 1990 and 1999 showed that 63% of patients who completed therapy improved, and up to 54% remained improved 2 years later (Westen & Morrison, 2001). Behavioral and cognitive-behavioral therapies typically involve exposure to feared interoceptive or exteroceptive stimuli (including bodily sensations that have predicted panic attacks), sometimes through intentional hyperventilation, exercise, or holding one's breath, with the goal of extinction of the anxiety response and the catastrophic expectations that accompany it. Controlled breathing exercises and relaxation techniques are often included, as well as various other stress-reduction, education, and self-control skills. These treatments have been quite successful and appear to be more effective and less subject to relapse than pharmacological methods (Barlow, 2002). Combination of CBT and medication produces mixed results. Adding the antidepressant imipramine to CBT provides some limited additional benefit but also increases relapse rate (Barlow, Gorman, Shear, & Woods, 2000).

6.2c Agoraphobia

A second anxiety disorder is agoraphobia (see *DSM-5*: Diagnostic Criteria for Agoraphobia). Literally from the Greek for “fear of the marketplace,” it involves not fear of open places but rather anxiety about being in places or situations from which escape would be difficult, embarrassing, or impossible in the event of having a panic attack or panic-like symptoms. Individuals might fear getting on a bus or airplane or traveling in the car because something very embarrassing (such as a bout of diarrhea) or a full-blown panic attack could occur. The fear can cause people to be reluctant to leave home (or another place where they are safe from such possibilities) or to be afraid of being at home alone when help might be needed. Obviously, agoraphobia could interfere with normal occupational, academic, and social activities and functions.

Dependence

A persistent pattern of drug use involving tolerance, withdrawal, or inability to cut down dosage

Relapse

Reoccurrence of symptoms after a period of improvement

DSM-5**Diagnostic Criteria for Agoraphobia****300.22 (F40.00) Agoraphobia**

- A. Marked fear or anxiety about two (or more) of the following five situations:
1. Using public transportation (e.g., automobiles, buses, trains, ships, planes).
 2. Being in open spaces (e.g., parking lots, marketplaces, bridges).
 3. Being in enclosed places (e.g., shops, theaters, cinemas).
 4. Standing in line or being in a crowd.
 5. Being outside of the home alone.
- B. The individual fears or avoids these situations because of thoughts that escape might be difficult or help might not be available in the event of developing panic-like symptoms or other incapacitating or embarrassing symptoms (e.g., fear of falling in the elderly; fear of incontinence).
- C. The agoraphobic situations almost always provoke fear or anxiety.
- D. The agoraphobic situations are actively avoided, require the presence of a companion, or are endured with intense fear or anxiety.
- E. The fear or anxiety is out of proportion to the actual danger posed by the agoraphobic situations and to the sociocultural context.
- F. The fear, anxiety, or avoidance is persistent, typically lasting for 6 months or more.
- G. The fear, anxiety, or avoidance causes significant distress or impairment in social, occupational, or other important areas of functioning.
- H. If another medical condition (e.g., inflammatory bowel disease, Parkinson's disease) is present, the fear, anxiety, or avoidance is clearly excessive.
- I. The fear, anxiety, or avoidance is not better explained by the symptoms of another mental disorder—for example, the symptoms are not confined to specific phobia, situational type; do not involve only social situations (as in social anxiety disorder); and are not related exclusively to obsessions (as in obsessive-compulsive disorder), perceived defects or flaws in physical appearance (as in body dysmorphic disorder), reminders or traumatic events (as in posttraumatic stress disorder), or fear of separation (as in separation anxiety disorder).

Note: Agoraphobia is diagnosed irrespective of the presence of panic disorder. If an individual's presentation meets criteria for panic disorder and agoraphobia, both diagnoses should be assigned.

Source: Reprinted with permission from the *Diagnostic and Statistical Manual of Mental Disorders*, 5th ed. (Copyright 2013). American Psychiatric Association.

In the *DSM-IV*, agoraphobia was not a separate diagnosis; rather, it was a specifier that could be linked to other anxiety conditions such as panic disorder. The *DSM-5* elevated it to a free-standing diagnosis in part because of the recognition that many people with agoraphobia do not experience panic. In those cases where both sets of symptoms occur, both diagnoses (agoraphobia and panic disorder) are now given.

The 12-month prevalence rates for agoraphobia are approximately 1.7% for adolescents and adults and 0.4% for those older than 65. Childhood onset is rare. Stressful events may be associated with the onset of agoraphobia, but there is also a relatively strong genetic link to phobias. It is often associated with depression and other anxiety disorders and is more commonly diagnosed among females than among males, who tend to show higher comorbid rates of substance use (American Psychiatric Association, 2013).

Treatment for agoraphobia has most often been studied in patients with accompanying panic disorder, due to the historical linkage between diagnoses. Promising results have been reported for behavioral and cognitive-behavioral interventions that involve controlled exposure to agoraphobic situations in an effort to promote habituation of the resulting anxiety. For example, Gloster and his colleagues (2011) employed two variants of exposure-based CBT for agoraphobia in a large sample ($N = 369$): one in which the exposure was discussed, planned, and experienced (the therapist accompanied the patient into the situation), and the other with similar interventions but without the therapist guiding the exposure. Compared to wait-list controls, both groups showed reduction in agoraphobic avoidance, with the greatest improvement in the therapist-guided group. Additional research has indicated that virtual reality exposure therapy alone may be as effective as exposure with CBT for agoraphobia.

(Malbos, Rapee, & Kavakli, 2013). Pharmacological treatment for agoraphobia usually involves SSRI antidepressants, with outcomes similar to that of CBT. For example, patients aged 60 and older in a small study ($N = 49$) were randomly assigned to receive paroxetine (an SSRI), CBT, or a wait-list control. Both treatments were effective, although CBT showed somewhat better results for those with later onset of the condition (Hendriks, Keijsers, Kampman, Hoogduin, & Oude Voshaar, 2012).

6.2d Specific Phobia

Specific phobia involves intense and persistent fear triggered by specific objects or situations (refer to the diagnostic criteria for Specific Phobia in Table 4-3). The fear reaction is excessive, resulting from either the presentation or the anticipation of the triggering stimulus. The onset of anxiety after exposure to the feared stimulus is usually immediate, includes many panic-like symptoms, and may meet the criteria for a cued panic attack. Because the fear is so distressing, individuals avoid or escape from the feared stimulus, which negatively reinforces the avoidance behavior and maintains the phobic response. Often, individuals with phobias realize that their reactions are excessive or unreasonable, but this realization does not reduce the phobic response, in part because they also overestimate the danger in their feared situations. Although many people have intense fears involving certain stimuli (such as snakes or spiders), specific phobia is not diagnosed unless it lasts at least 6 months and interferes significantly with a person's life or is associated with marked distress.

Phobic anxiety can become associated with a variety of objects or situations. Following are some common examples:

Heights: cliffs, roofs, high windows, ladders

Enclosed places: small rooms, closets, elevators, subways

Open places: halls, wide streets, squares, parks, beaches

Animals: dogs, cats, snakes, horses, spiders

Weapons: guns, knives, axes

Public gatherings: crowds, meetings, churches, theaters, stadiums

Vehicles: airplanes, trains, automobiles, buses

Natural dangers: storms, wind, lightning, darkness

The *DSM-5* includes subtype indicators in the diagnosis of specific phobia: animal type, natural environment type, blood-injection-injury type (e.g., fear of blood, mutilation, or medical procedures), situational type (e.g., fear of flying, closed spaces, or bridges), and

other types (such as fear of choking, clowns, or noises). The specific phobia diagnosis appears to be one of the most reliable in the anxiety disorder category. Brown and his colleagues (2001) reported a kappa value of .86 when it was the principal *DSM-IV* diagnosis; its reliability was not assessed in the *DSM-5* field trials, however.

Phobias are relatively common disorders, with lifetime prevalence rates of over 12% of the population (Kessler, Chiu, et al., 2005). Women are diagnosed with phobias twice as often as men. First symptoms of phobias tend to appear in childhood or early adolescence, and prevalence declines among the elderly (American Psychiatric Association, 2013). There are many different variations of phobias, as Table 6-3 illustrates. Certain phobias may be more easily acquired at different ages and may involve some differences in fear response patterns. For example, fear of heights and driving

phobias appear to be associated with later age of onset, and blood/injection phobias may be more likely to involve fainting, relative to other phobias (Antony, Brown, & Barlow, 1997).



Phobic anxiety can become associated with a variety of objects or situations. A common example is heights, such as cliffs, roofs, high windows, and ladders. (iStock)

Table 6-3 Some Variations of Specific Phobias

Name	Object(s) Feared	Name	Object(s) Feared
Acrophobia	High places	Monophobia	Being alone
Agoraphobia	Open places	Mysophobia	Contamination
Ailurophobia	Cats	Nyctophobia	Darkness
Algophobia	Pain	Ochlophobia	Crowds
Anthropophobia	Men	Pathophobia	Disease
Aquaphobia	Water	Phyrophobia	Fire
Astraphobia	Storms, thunder, lightning	Syphilophobia	Syphilis
Claustrophobia	Closed places	Thanatophobia	Death
Cynophobia	Dogs	Xenophobia	Strangers
Hematophobia	Blood	Zoophobia	Animals or a single animal

Adapted from *Abnormal Psychology: A New Look*, by M. Duke and S. Nowicki, 1986, New York, NY: Holt, Rinehart, and Winston.

Causal Factors

Specific phobias are more common in families in which other members have phobias, and monozygotic twins show higher concordance rates than dizygotic twins (Kendler, Neale, Kessler, Heath, & Eaves, 1992); both of these points support a modest genetic risk factor. As noted before, differences in HPA activity can have a genetic basis, so it is reasonable to expect that some differences in phobic response are heritable. Still, there is wide latitude for environmental effects in the formation of phobias.

The behavioral model has proposed conditioning in the etiology of phobias since Watson's demonstration with Little Albert (see Chapter 3). Essentially, the phobia results through classical conditioning when an object or situation is paired with a traumatic event or a strong sensation of fear. For example, some children are "taught" to swim by parents who throw them into the water so that they learn "naturally." A phobia of deep water might easily develop after such an episode. Struggling to keep one's head above water (the US) can produce gasping and intense fear (the UR); because of pairing, any water in which the bottom cannot be seen (CS) comes to elicit terror and a sensation of drowning (CR). Once the phobia is established through Pavlovian conditioning, future exposure to the phobic stimulus produces avoidance behavior, which is then operatively reinforced by the reduction in fear that it produces. This negative reinforcement makes future phobic avoidance more likely, thus maintaining the phobia. This model is often referred to as the **two-factor theory** of phobias (Mowrer, 1950), reflecting the involvement of both Pavlovian and operant influences.

Rachman (1977) criticized the conditioning theory as being incomplete. In early classical conditioning theory, any stimulus (object, sound, odor, and so on) might serve equally well as a CS, as long as it has been paired with the unconditioned stimulus; however, various sources of evidence suggest that this is not the case. For example, if all stimuli can serve equally well as CS, why are human phobias most commonly associated with a rather limited set of stimuli—fear of leaving home, specific animals and insects, heights, the dark, situations related to bodily injury or mutilation, and so on? Only rarely do we have phobias of pajamas, electrical outlets, or hammers, even though all of these can be associated with trauma. Pajamas, for example, are present when young children experience fear of the dark or have nightmares, but children rarely develop a pajama phobia.

How important are specific traumatic experiences in the development of phobias?



Severe storms like Hurricane Sandy, which devastated parts of the U.S. Eastern Seaboard, can produce phobic reactions. (Shutterstock)

Two-factor theory

Theory that Pavlovian conditioning establishes the phobia, while operant contingencies maintain phobic behavior

Rachman also pointed out that people who undergo repeated fear-arousing experiences may not develop phobias to surrounding stimuli. Thus, despite repeated exposure to fearsome air raids during World War II, only a very small proportion of adults or children developed phobias as a result. Rachman considered these findings as being supportive of a theory previously proposed by Seligman and Hager (1972) to the effect that humans, as well as other animals, could learn to be fearful of some stimuli more readily than they can of other stimuli. There is an innate **preparedness** to become fearful of certain stimuli, because in our evolutionary past these were associated with real dangers—animals, the dark, heights, mutilated bodies. This theory of a preparedness to become fearful of some stimuli and not others may help explain why Bregman (1934) was unable to replicate Watson and Rayner's (1920) demonstration of fear conditioning in Little Albert. Bregman attempted to condition fear in 15 infants but used biologically irrelevant objects such as geometrically shaped wooden blocks and cloth curtains, whereas Watson and Rayner used a furry rat. Bandura (1977) agreed that phobias develop more readily to some stimuli than others but argued that this can be explained by differences in the presenting nature of the stimuli rather than innate tendencies. Thus, snakes and various animals are especially likely to become phobic stimuli because they can appear at unpredictable times and places, show great mobility, and inflict injury despite self-protective efforts.

There is considerable laboratory and real-world evidence that both animal and human subjects can learn emotional reactions, including fear, by observing other subjects model these reactions (Bandura, 1977). For example, several people were treated for anxiety reactions after watching the film *The Exorcist* (Bozzuto, 1975). Likewise, monkeys raised in laboratory environments do not fear snakes until they observe the fearful response of other monkeys (Mineka, Davidson, Cook, & Keir, 1984). Various cognitive processes may also play a role in the learning and maintenance of fear. A child's interpretation of events or expectation about what is going to happen can be important in this respect. The child who hears parents, siblings, or others warn of certain dangers can begin to tell himself about these same anxiety-arousing situations. The child's potential for imaginative elaboration can multiply and maintain the stimuli.

Certainly, large individual differences would be expected in the phobic responses of people exposed to (or observing) particular traumatic events, as well as variations in prior familiarity with the stimulus, history of uncontrollable events, and prior coping experiences. These facts, as well as the issues raised by Rachman and Seligman, have been incorporated into a comprehensive contemporary learning model by Mineka and her colleagues (e.g., Mineka & Sutton, 2006; Mineka & Zinbarg, 2006), which combines early learning experiences, **vicarious conditioning**, contextual variables, genetic/temperamental influences, and basic learning principles to account for the development and maintenance of phobias. It arguably remains the most validated and plausible perspective on the etiology of phobias at this time.

The psychodynamic model is illustrated by Freud's analysis of Little Hans, described in Chapter 3. Recall that Hans had developed an animal phobia (of horses), which Freud interpreted as displaced fear of Hans's father, related to an Oedipal castration anxiety. More generally, the psychoanalytic theory of phobia formation would emphasize some initial repression of an anxiety-arousing conflict, the projection of the conflict onto the external world (Little Hans, for example, was said to have projected his wish to attack his father, and thus he believed his father wished to attack him), and then *displacement* of the anxiety onto some other target (horses, in the case of Hans).

Preparedness

Biologically based tendency to form associations between certain stimuli more readily than others

Vicarious conditioning

Conditioning based on observing the responses of other people

Treatments for Specific Phobias

From the biological perspective, acute fear reactions can be moderated with medications such as benzodiazepines, which act more rapidly than SSRIs or other antidepressants. However, in the case of specific phobia, no pharmacological intervention has been shown to be effective (Roy-Byrne & Cowley, 2002).

Nearly all empirically supported therapies for specific phobia involve exposure to the feared stimulus, as initially demonstrated by Mary Cover Jones (1924) with "Little Peter." Peter was a 3-year-old boy who was afraid of rabbits, as well as a variety of other furry creatures

and objects. In the initial sessions, Peter would cry in the presence of a caged rabbit unless it was taken at least 20 feet away. Jones began with the rabbit in a cage some 12 feet away from Peter and gradually (over several treatment sessions) brought the animal closer; at the same time, Peter was being fed his favorite candy by his mother. Eventually, Peter was able to stroke the rabbit affectionately, and even allow it to nibble at his fingers, without crying. Subsequently, Wolpe (1958, 1973) developed this approach into systematic desensitization. The many variations of exposure therapy for phobias involve extended exposure, either in vivo or in imagination, with the goal of reducing the fear response through extinction. Systematic desensitization involves maintaining a relaxed state while increasingly fear-provoking phobic stimuli are gradually presented. Participant modeling, in which the therapist first models a calm, nonphobic interaction with the feared stimulus, has been effective as well. Overall, the professional literature provides the strongest empirical support for therapies involving actual, rather than imagined, exposure, and a consensus has developed that **in vivo exposure** is the treatment of choice for specific phobia (Barlow, Raffa, & Cohen, 2002).

6.2e Social Anxiety Disorder (Social Phobia)

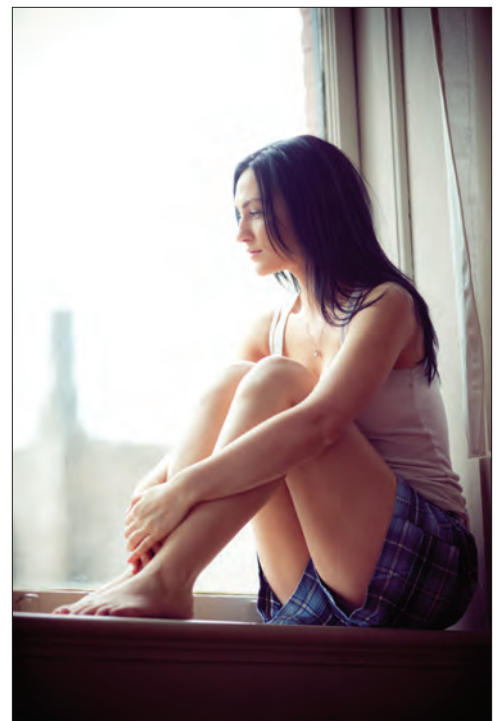
Social anxiety disorder (also called social phobia) is similar to specific phobia except that the symptoms are connected to situations in which the person is exposed to unfamiliar people or to the scrutiny of others; the person also fears acting in a way that might prove embarrassing or humiliating. For example, sufferers may fear appearing stupid, incompetent, or weak, or they may fear that others in the social setting will notice their trembling hands or their quivering voice. Exposure to these situations almost immediately triggers distressing fear symptoms, which may escalate into panic attacks. As a result, the person tends, if possible, to avoid social or performance situations to the extent that the phobia interferes with normal daily life. People with social phobia may experience fear far in advance (perhaps every day for several weeks) of an anticipated social activity.

Nearly everyone has felt some anxiety about or embarrassment in some social situations, and these occurrences do not warrant the diagnosis. For example, students may find themselves unprepared for a question from the teacher, and most people unaccustomed to public speaking would be anxious when anticipating delivering an address to a large group of people. Social anxiety disorder, however, involves shyness and social anxiety that is severe enough to interfere with normal life in terms of occupational, academic, or interpersonal functioning for at least 6 months. The diagnosis can be subtyped as “performance only” if the fear is limited to performing or speaking in public.

Incidence estimates vary widely, but the National Comorbidity Survey Replication suggested a 12.1% lifetime prevalence (Kessler, Chiu, et al., 2005) of social phobia, making it one of the most common anxiety disorders, along with specific phobia. The *DSM-5* gives the 12-month prevalence rate at 7% in the United States, noting much lower prevalence (0.5%–2.3%) in the rest of the world. Age of onset for the disorder is between 8 and 15 years in 75% of cases (American Psychiatric Association, 2013). Although it is more commonly diagnosed among women, men are represented equally in treatment settings (American Psychiatric Association, 2000), suggesting that they may be more likely to seek treatment for the condition.

Causal Factors

As in the other anxiety disorders, social anxiety disorder runs in families, supporting a modest genetic factor: First-degree relatives of sufferers have 2–6 times greater risk of developing the condition (American Psychiatric Association, 2013). Some neuroimaging data suggest that the



A person suffering from social anxiety disorder (social phobia) tends to avoid social or performance situations to the extent that the phobia interferes with normal daily life. (iStock)

In vivo exposure

Exposure to the actual feared stimulus rather than an imagined or symbolic stimulus

amygdala responds differently to novel faces rather than to familiar faces in people with social phobia (Cottraux, 2005), although that may be an effect rather than a cause of the disorder.

Learning models assume that social phobia develops in similar ways as other phobias, involving a conditioned association between social cues and unpleasant or embarrassing events or social defeats. In retrospective studies, most social phobics recall such conditioning experiences as relevant to the beginning of their symptoms (Öst & Hugdahl, 1981), although retrospective recall may not be a reliable source of information. According to the *DSM-5*, there is no information that establishes any causal relation between social anxiety disorder and childhood abuse or other childhood adversity (American Psychiatric Association, 2013). Psychodynamic models tend to emphasize internal conflicts that produce anxiety (as in the other phobias) as the probable cause of social phobia.



The SNRI venlafaxine (Effexor®) shows promise as a treatment for social anxiety. (Wikimedia Commons/Public Domain)

Treatments for Social Phobias

Pharmacologically, benzodiazepines appear to be effective in short-term relief for social anxiety (Davidson, 2004). SSRIs have also been used effectively (Roy-Byrne & Cowley, 2002). Recent work supports the SNRI venlafaxine (Effexor®) and the anticonvulsant drug pregabalin (Lyrica®) as promising, although long-term studies are still needed (Cottraux, 2005).

Cognitive-behavioral therapies have also been effective in treating social phobia. These techniques typically involve systematic exposure to feared social situations, often combined with relaxation training or desensitization. Newer CBT applications, including virtual reality technologies that provide computer-generated scenarios of public-speaking situations, also appear promising for treating social anxiety (Cottraux, 2005), as well as for other anxiety disorders.

6.2f Generalized Anxiety Disorder

Generalized anxiety disorder (GAD) is characterized by a nearly constant state of worry and apprehension about a wide variety of events or activities on most days over a 6-month period. Several symptoms of autonomic arousal are present, including disturbed sleep and concentration, muscle tension, irritability, and fatigue, to the extent that they interfere with daily life. Although people with GAD find their worries difficult to control, distressing, and physically troublesome, they do not progress into panic attacks.

People with GAD seem to be always “on edge” and worry about minor events as often as major ones. Children may worry about school, deadlines, or world events—often requiring reassurance about their performance. Adults may worry excessively about finances, health, work, and household duties. Major events, such as nuclear war or other possible catastrophes, are sources of elevated concern as well. Rarely are there periods in which all worry is absent. Comorbidity of GAD with additional diagnoses, such as depression and other anxiety disorders (including panic disorder), is fairly common. GAD is diagnosed among females twice as often as men, and 12-month prevalence is around 2.9% in U.S. adults (American Psychiatric Association, 2013). As noted earlier, the *DSM-5* diagnosis is not very reliable, with a kappa of .20, falling in the “questionable” range (Regier et al., 2013).

Causal Factors

Biological involvement in generalized anxiety includes several brain pathways that are also active in other anxiety disorders, including the amygdala, the limbic system, and the prefrontal cortex. It is assumed that inhibition of these pathways, mediated by the neurotransmitter GABA, is deficient in GAD sufferers, although there is some indication of serotonin involvement as well. Genetic evidence is mixed; its modest heritability (accounting for about one third of the risk) may be somewhat smaller than that for other anxiety disorders (American Psychiatric Association, 2013). Overlap of symptoms with mood disorders, as well

as some indications of common biological/genetic involvement between GAD and mood disorders, may indicate that these are related rather than independent clusters of disorders (Tyrer & Baldwin, 2006).

A cognitive-behavioral model of GAD would be similar to that for panic disorder: interoceptive and exteroceptive stimuli become predictive of worry and apprehension, to the point that widespread anxiety occurs as a conditioned response. This would be most likely to occur in people who have a history of uncontrollable and unpredictable events and who would also be less likely to identify periods of safety from threat, resulting in hypervigilance. It would also result in a cognitive tendency to overappraise and attend to threatening interoceptive and exteroceptive cues; such an interpretive bias can increase vulnerability to anxiety disorders (Wilson, MacLeod, Mathews, & Rutherford, 2006). In psychodynamic terms, anxiety is created by unconscious conflict, which overwhelms the ego's defenses with unacceptable impulses and produces a widespread overt anxiety reaction.

Treatments for Generalized Anxiety Disorder

Interventions, both medical and psychological, can be helpful for GAD but rarely result in total symptom remission (Tyrer & Baldwin, 2006). Effective medications include GABA stimulants such as the benzodiazepines and buspirone (which has little dependence or abuse potential) for short-term treatments. Antidepressants, especially the SSRIs, may be more helpful both in the longer term and with concomitant depression (Davidson, 2004; Gorman, 2003).

Psychotherapy for GAD can be helpful as well and appears to be at least as effective as pharmacological treatment. Reviews of the effectiveness of various treatments in controlled clinical trials report that active therapies are superior to nondirective therapies, without clear superiority for any one (Barlow et al., 2002). In a treatment comparison between cognitive-behavioral therapy and a benzodiazepine (diazepam), CBT produced better results 6 months after therapy, whereas diazepam was no more effective than placebo (Power, Simpson, Swanson, & Wallace, 1990). Some literature reviews (e.g., Westen & Morrison, 2001) found that while CBT is helpful in the short term for GAD, evidence of sustained improvement is lacking; overall, CBT is less effective for GAD than for panic disorder. On the other hand, Borkovec and Ruscio (2001) reviewed 13 well-controlled studies and concluded that CBT is consistently shown to be clinically effective for both anxiety and depression, with low dropout rates and long-term maintenance of improvement.

Which treatments are effective for anxiety disorders?

6.2g Separation Anxiety Disorder

In separation anxiety disorder, a person experiences developmentally inappropriate and excessive fear or anxiety associated with separation from home or from primary caregivers. The distress may include anxiety about getting lost or losing connection to the attachment figure, concern that the attachment figure may be harmed, or fear of being taken away or kidnapped. Associated with these fears, the person with separation anxiety disorder may experience nightmares or physical symptoms and may refuse to attend school or other activities requiring absence from home or the attachment figure. The person may refuse or resist sleeping unless the attachment figure is nearby. The disturbance must last at least 4 weeks in children and 6 months or more in adults.

Children with this disorder often come from close-knit families. They may fear specific objects or animals that could pose danger of harm or events such as travel that may result in separation. Anxiety disorders commonly co-occur, and the condition may precede the onset of panic disorder with agoraphobia. The 12-month prevalence is estimated to be 4% in children, and it appears to be more common among females (American Psychiatric Association, 2013).

Separation anxiety disorder is a strong predictor of subsequent mental disorders, especially panic disorder and major depressive disorder (Lewinsohn, Holm-Denoma, Small,

Seeley, & Joiner, 2008). At present, there are no medications approved by the FDA for treatment of separation anxiety disorder, although SSRI antidepressants are sometimes prescribed. In a small pharmacological study, the tricyclic antidepressant imipramine was not more effective than placebo in any outcome measure of separation anxiety disorder in a group of 21 children who had not responded to behavior therapy, although side effects were significantly higher in the imipramine group (Klein, Koplewicz, & Kanner, 1992). Cognitive-behavioral therapy appears to be effective for separation anxiety disorder in children (Chambless et al., 1998), and CBT with or without added family therapy was associated with long-term improvement more than 6 years after therapy (Barrett, Duffy, Dadds, & Rapee, 2001).

6.2h Selective Mutism

The central component of selective mutism is persistent failure to speak in situations where speaking is expected, such as social and school settings. The individual does speak in other situations, however. Because a person may be shy in new situations, the diagnosis is not based on the first month in a new school or other social setting. After the first month, selective mutism can be diagnosed if the failure to speak persists for a month and is not due to lack of knowledge about the language, comfort with the spoken language, embarrassment about speaking connected to a communication disorder such as stuttering, or a pervasive developmental disorder or psychotic disorder. Sometimes, children with selective mutism will communicate through hand signals or gestures. Onset is usually before age 5, and more females than males are affected. The causes are unknown, though there is some overlap with social anxiety disorder. The incidence is less than 1% of the population (American Psychiatric Association, 2013).

Some have approached the treatment of selective mutism similarly to anxiety disorders; case reports suggest antidepressants may be useful. A review of the treatment literature by Pionek Stone, Kratochwill, Sladeczek, and Serlin (2002) concluded that behavioral interventions, including reinforcement, shaping and stimulus control of speech, perhaps combined with modeling, appear to be the treatments of choice for the condition.

6.2i Other Anxiety Disorders

Anxiety disorder symptoms may also stem from general medical conditions, such as cardiovascular, respiratory, or neurological conditions, or as a result of substance use. The *DSM-5* provides diagnostic categories for these conditions when they cause significant distress or impairment in important areas of functioning.

Substance/medication-induced anxiety disorder involves panic attacks or anxiety, developing soon after intoxication with, withdrawal from, or exposure to a substance that is capable of producing the anxiety. A variety of substances can provoke anxiety or panic, including alcohol, caffeine, cannabis, cocaine, amphetamines, opioids, inhalants, and hallucinogens. The diagnosis can be coded for each, and the anxiety onset can be specified as during intoxication, during withdrawal, or after medication use. Other possible sources of the anxiety (such as an independent anxiety disorder, or a delirium) must be ruled out as well.

Anxiety disorder due to another medical condition involves panic or anxiety that can be traced to the direct pathophysiological consequence of another medical condition such as hyperthyroidism, arthritis, congestive heart failure, hypertension, or asthma. Other potential sources of anxiety, including other mental disorders, must be ruled out as well.

Other specified anxiety disorder applies to conditions of anxiety and fear that do not meet the full criteria for another anxiety disorder. For example, an individual may experience panic attacks but with fewer than four panic symptoms, or a person may experience generalized anxiety for fewer than 4 days per week.

Finally, the remaining presentations of significant anxiety or fear that are not covered by any other category may be labeled as *unspecified anxiety disorder*.

6.3 Obsessive-Compulsive and Related Disorders

Obsessions are thoughts that intrude repeatedly into awareness and are experienced as irrational, unwanted, and difficult to control or stop. **Compulsions** are actions that one is compelled to perform; they are also experienced as irrational and difficult to control. Mild forms of obsessive-compulsive experiences—like mild forms of phobic reactions—are not uncommon in normal individuals. The song that intrudes recurrently into the moment or an urge to return home to make sure the door is locked or the stove turned off are common examples.

What are obsessions and compulsions?

There can be preoccupations with certain objects or activities within many different mental disorders, as we shall see. Those basic commonalities led some researchers (e.g., Hollander & Wong, 1995) to propose that a concept of an *obsessive-compulsive spectrum* could include conditions as apparently diverse as pathological gambling, tic disorders, antisocial personality, and autism. The *DSM-5* restructured the classification of obsessive-compulsive disorder, which had been under the anxiety disorder category in both the *DSM-III* and *DSM-IV*, into a new category—the obsessive-compulsive and related disorders (see Table 6-4). Although many different types of obsessions and compulsions can be included in the spectrum, the *DSM-5* excludes certain apparently obsessive-compulsive activities that occur as part of other disorders. For example, an alcohol-dependent person may experience urges and obsessions about drinking and may appear to drink compulsively, but that preoccupation is already accounted for within a substance use diagnosis. Similarly, those obsessions or compulsions associated with eating disorders, impulse-control disorders, certain sexual disorders, or depressed persons with obsessive guilt would be excluded from the current spectrum. On this point, the *DSM-5* distinguishes between the compulsive activities of obsessive-compulsive and related disorders and those of substance dependence (for example) on the basis that the latter individuals derive pleasure from the activity, while those with obsessive-compulsive conditions do not. The diagnoses in this category can be used with specifiers—such as “with poor insight,” “absent insight/delusional beliefs,” and “tic-related”—to allow for more specificity in their use.

6.3a Obsessive-Compulsive Disorder

In obsessive-compulsive disorder (OCD), obsessions and compulsions reach a handicapping degree of severity, occupying a significant amount of time and interfering with normal social, occupational, or academic activities (see *DSM-5*: Diagnostic Criteria for Obsessive-Compulsive Disorder). Unwanted, distressing obsessions and thoughts, sometimes aggressive or sexual in nature, may alternate with thoughts or actions that counteract or inhibit them. The most

Obsession

Intrusive thought that is difficult to stop or control

Compulsion

Action that one feels compelled to perform

Table 6-4 DSM-5 Obsessive-Compulsive and Related Disorders

Disorder	Key Symptoms	Minimum Duration Required for Diagnosis	Sex Ratio
Obsessive-compulsive disorder	Recurrent obsessions or compulsions that cause distress	None	Slightly more common in females
Body dysmorphic disorder	Preoccupation with imagined or minor physical defect in appearance	None	Slightly more common in females
Hoarding disorder	Persistent difficulty discarding possessions, resulting in excessive clutter	None	More common in males
Trichotillomania (hair-pulling) disorder	Recurrent pulling out of hair	None	More common in females
Excoriation (skin-picking) disorder	Recurrent picking of skin	None	More common in females

Source: American Psychiatric Association, 2013.

common sorts of obsessions involve thoughts about contamination or repeated doubts, such as whether a necessary act has been performed; “forbidden” urges, such as the idea



As the obsessions continue to generate increasing anxiety, the person with OCD often engages in compulsive acts to neutralize the obsession and reduce anxiety—for example, excessive cleaning. (Shutterstock)

of stabbing, choking, poisoning, shooting, or otherwise injuring one’s child, parent, spouse, sibling, or self; the idea of shouting obscene words at home, work, or church; the wish that someone were dead; the thought or image of a forbidden sexual adventure, perhaps involving “perverted” sex acts; the thought of committing suicide by jumping out of a window or into the path of a truck; and the thought of contracting some disease from touching doorknobs, banisters, toilets, or other objects in public places.

At first the individual with obsessions usually attempts to ignore or suppress them. As the obsessions continue to generate increasing anxiety, the person often engages in compulsive acts to neutralize the obsession and reduce anxiety. Examples of thoughts or actions designed to counteract forbidden or distressing thoughts are almost any kind of ritual—such as washing or cleaning, counting to oneself, memorizing license plate numbers, or reciting certain words or phrases to oneself. Or the person may

DSM-5

Diagnostic Criteria for Obsessive-Compulsive Disorder

300.3 (F42.2) Obsessive-Compulsive Disorder

A. Presence of obsessions, compulsions, or both:

Obsessions are defined by (1) and (2):

1. Recurrent and persistent thoughts, urges, or images that are experienced, at some time during the disturbance, as intrusive and unwanted, and that in most individuals cause marked anxiety or distress.
2. The individual attempts to ignore or suppress such thoughts, urges, or images, or to neutralize them with some other thought or action (i.e., by performing a compulsion).

Compulsions are defined by (1) and (2):

1. Repetitive behaviors (e.g., hand washing, ordering, checking) or mental acts (e.g., praying, counting, repeating words silently) that the individual feels driven to perform in response to an obsession or according to rules that must be applied rigidly.
2. The behaviors or mental acts are aimed at preventing or reducing anxiety or distress, or preventing some dreaded event or situation; however, these behaviors or mental acts are not connected in a realistic way with what they are designed to neutralize or prevent, or are clearly excessive.

Note: Young children may not be able to articulate the aims of these behaviors or mental acts.

- B. The obsessions or compulsions are time-consuming (e.g., take more than 1 hour per day) or cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
- C. The obsessive-compulsive symptoms are not attributable to the physiological effects of a substance (e.g., a drug of abuse or medication) or another medical condition.
- D. The disturbance is not better explained by the symptoms of another mental disorder (e.g., excessive worries, as in generalized anxiety disorder; preoccupation with appearance, as in body dysmorphic disorder; difficulty discarding or parting with possessions, as in hoarding disorder; hair pulling, as in trichotillomania [hair-pulling] disorder; skin picking, as in excoriation disorder [skin-picking] disorder; stereotypies, as in stereotypic movement disorder; ritualized eating behavior, as in eating disorders; preoccupation with substances or gambling, as in substance-related and addictive disorders; preoccupation with having an illness, as in illness anxiety disorder; sexual urges or fantasies, as in paraphilic disorders; impulses, as in disruptive, impulse-control, and conduct disorders; guilty ruminations, as in major depressive disorder; thought insertion or delusional preoccupations, as in schizophrenia spectrum disorders; or repetitive patterns of behavior, as in autism spectrum disorder).

Specify if:

With good or fair insight: The individual recognizes that obsessive-compulsive disorder beliefs are definitely or probably not true or that they may or may not be true.

With poor insight: The individual thinks that obsessive-compulsive disorder beliefs are probably true.

With absent insight/delusional beliefs: The individual is completely convinced that obsessive-compulsive beliefs are true.

Specify if:

Tic-related: The individual has a current or past history of tic disorder.

follow more elaborate verbal rituals that have a scientific, philosophical, or religious basis. Other compulsive acts include excessive politeness, excessive orderliness and neatness, and inordinate attempts to schedule one's activities on a precise timetable.

The obsessions and compulsions are time consuming, occupying at least an hour per day or frequently much longer periods. Individuals may become incapacitated by endless compulsive rituals and the immobilization associated with obsessive indecision and doubting. Sufferers differ in the degree to which they are aware that the obsessions or compulsions are excessive or unreasonable; those who recognize that their obsessive-compulsive beliefs are not true receive the specification "with good insight." About 30% of those with OCD also have a tic disorder (American Psychiatric Association, 2013).

Obsessive-compulsive disorder may have a lifetime prevalence of about 2.5% of the population (12-month prevalence in the United States is 1.2%); the rate is slightly higher for females in adult onset, while childhood onset is perhaps more common among boys than girls. Onset is usually gradual, with earlier appearance of symptoms in males. If untreated, the disorder usually follows a chronic course with symptoms worsening during periods of stress (American Psychiatric Association, 2013).

Causal Factors

As with the other anxiety disorders, twin studies and familial patterns suggest a moderate genetic contribution to OCD. Some brain studies using PET scans have reported higher levels of activity in the orbital frontal cortex of the left hemisphere for those with OCD as compared with normals (e.g., Baxter, 1992). Whether this increased activity is a possible cause of, or a result of, OCD is not yet clear. The risk of OCD is also increased in children who have been subject to traumatic events, including physical and sexual abuse (American Psychiatric Association, 2013).

Behavioral models tend to employ versions of two-factor theory in the analysis of obsessive-compulsive behavior—which, according to Williams (2001), "follows the pattern predicted by two factor theory in remarkable detail" (p. 362). In the case of a hand-washing compulsion, for example, it is assumed that stimuli that trigger obsession gained their anxiety-provoking power through a pairing with strong emotions of fear or anxiety in an individual's learning history. Subsequent exposure to an anxiety-producing conditioned stimulus (such as a medical scene or a newspaper article on disease) generates anxiety that persists until it is relieved by giving in to the compulsion to wash the hands. The compulsive act is, thereby, negatively reinforced and strengthened.

In psychoanalytic terminology, obsessive persons may be said to use several defense mechanisms. They isolate feelings from intellectual content so that their obsessive thoughts and endless verbosity become detached from their emotional roots; an overintellectualized pattern of life results. Reaction formations are likewise common. For example, obsessive concern with cleanliness may be a defense against underlying urges to be dirty or sexy (sex may be perceived as dirty or aggressive). Thus, compulsive orderliness protects the person from the fear of unleashed aggression, of smashing everything in sight; excessive politeness and formality protect from urges to be cruel and sadistic. *Undoing* refers to many features of compulsive rituals in which the person attempts to "undo" the harm, real or imagined, that could result from an unacceptable impulse. Engaging in a certain mannerism (such as blinking the eyes or touching or straightening an object) helps the person feel that the dangerous impulse is canceled out. Reaction formations are similar to undoing except they are expressed in broad personality styles rather than in highly specific rituals. From a psychosexual developmental point of view, the obsessive-compulsive person has regressed,



According to psychoanalytic theory, compulsive concerns with cleanliness and orderliness represent reaction formations against anal impulses to be dirty and smelly. (iStock)

in the face of an intense Oedipal conflict, to the anal stage. Compulsive concerns with cleanliness and orderliness represent reaction formations against anal impulses to be dirty and smelly, while compulsive tendencies to inhibit emotion or to be formal or excessively good reflect reaction formations against anal-sadistic impulses, originating in the child's defiance of parental efforts to force compliance with toilet training.

Treatments for Obsessive-Compulsive Disorder

The most common medical treatment for OCD involves antidepressant medications, especially the SSRIs, some of which are effective in substantially reducing OCD symptoms (Abramowitz, 1997; Dougherty, Rauch, & Jenike, 2002). In particularly unresponsive cases, a form of psychosurgery—a **cingulotomy**, in which a small bundle of nerve fibers connecting the anterior cingulate cortex to the frontal lobes are severed—has produced improvement in some cases, including 8 of 17 patients treated by Jung and his colleagues (2006).

How effective are treatments for obsessive-compulsive and related disorders?

Psychodynamic therapists have employed interpersonal group therapies (Wells, Glickauf-Hughes, & Buzzell, 1990), as well as individual therapies, with mixed results. The treatment of choice among psychotherapies is **exposure and response prevention (ERP)**, which is the most effective intervention, producing substantial clinical improvement (Abramowitz, Foa, & Franklin, 2003). ERP requires prolonged and repeated exposure to the obsession, while the compulsive act is prevented. For example, Piacentini and Langley (2004) described the treatment of a 12-year-old boy obsessed with germs and contamination and compelled to frequently wash his hands. The ERP component of treatment required him to touch trashcans, doorknobs, and public tables and to play basketball without washing his hands for increasing periods of time. Other components of the program involved education about OCD and cognitive restructuring, common in many CBT approaches to OCD. CBT is effective for OCD whether or not medication is added (Franklin, Abramowitz, Bux, Zoellner, & Feeney, 2002), although ERP is effective with or without CBT added. In one comparison study, both treatments produced significant improvement, but ERP showed stronger effects 3 months after treatment ended (McLean et al., 2001).

6.3b Body Dysmorphic Disorder

People with body dysmorphic disorder (BDD) are preoccupied with what they consider a defect in their appearance, and they engage in repetitive acts or thoughts related to this preoccupation. The defect is either imaginary or so slight that the preoccupation is clearly excessive. The preoccupation causes significant distress or impairment in normal functioning, and it is not better accounted for by another disorder (for example, anorexia nervosa). Frequently, the preoccupations in BDD involve areas of the head and face and include wrinkles; complexion; facial proportions; the size or shape of lips, nose, eyebrows, cheeks, or ears; or thinning hair. However, the disorder can involve any area of the body, showing as excessive concern over the size or shape of hands, feet, buttocks, breasts, genitals, body size, or overall build. A specifier “with muscle dysmorphia” has been added in the *DSM-5* to capture a particular subset of sufferers who believe that their body build is too small or insufficiently muscular.

People with BDD can spend several hours each day checking the imagined defect or finding ways to cover it up or hide it. They may alternate between almost constantly looking into mirrors and avoiding them altogether due to the distress associated with viewing themselves. Their concern may lead them to isolate themselves from others or to avoid job interviews, meetings, classes, or dating. Depression, obsessive-compulsive disorder, and social phobia may co-occur, and suicidal ideation may be present. Not uncommonly, sufferers seek surgery or medical intervention and may even resort to self-surgery, although these interventions do not reduce the concern and may make the problem worse (American Psychiatric Association, 2013).

Cingulotomy

Psychosurgical technique that severs fibers connecting the anterior cingulate cortex to the frontal lobes

Exposure and response prevention (ERP)

Prolonged exposure to a fear- or anxiety-producing stimulus or situation while escape, or the compulsive act, is prevented

Based on a recent survey of U.S. households, the point prevalence of body dysmorphic disorder was estimated at 2.4% of the adult population (Koran, Abujaoude, Large, & Serpe, 2008). The prevalence may be higher in mental health settings and is up to 15% of patients in dermatological settings. BDD appears to be equally likely in males and females, begins by adolescence, and tends to have a chronic course. Muscle dysmorphia occurs almost exclusively in males; females are more likely to have comorbid eating disorders (American Psychiatric Association, 2013).

Causal Factors

Comorbidity with certain other disorders—notably, obsessive-compulsive disorder—might suggest a common causal component; there is higher prevalence of body dysmorphic disorder in those who have first-degree relatives with OCD. Childhood neglect and abuse are also risk factors for the condition (American Psychiatric Association, 2013). The current social and cultural emphasis on physical appearance likely plays a role in its development. Neziroglu, Roberts, and Yaryura-Tobias (2004) proposed a behavior model of BDD that suggests children might receive greater attention and reinforcement for their appearance than for their behavior.



People with body dysmorphic disorder may spend several hours per day checking for imagined defects. (Shutterstock)

Treatments for Body Dysmorphic Disorder

Reportedly, some BDD patients respond well to serotonin reuptake inhibitor medications (Fallon, 2004). Several recent studies have demonstrated that SSRIs work better than other pharmacological interventions for BDD, especially at higher doses and longer intervals of treatment (e.g., Hadley, Kim, Priday, & Hollander, 2006). Typical cognitive-behavioral techniques employ education, relaxation, and systematic desensitization involving graduated exposure to the parts of the body that evoke distress. Response prevention to avoid checking may be included, combined with attention training (to redirect self-observation), self-monitoring, cognitive challenging of automatic dysfunctional thinking, and developing alternative views about personal appearance. These CBT techniques have been shown effective in randomized controlled studies (Looper & Kirmayer, 2002). In fact, CBT may be as effective on BDD as it is on OCD (Cororve & Gleaves, 2001). Currently, both SSRIs and CBT are considered “first-line” treatments for body dysmorphic disorder (Phillips & Hollander, 2008), although one meta-analysis comparing their relative effectiveness concluded that CBT was more useful (Williams, Hadjistavropoulos, & Sharpe, 2006).

6.3c Hoarding Disorder

Hoarding was listed among symptoms of OCD and obsessive-compulsive personality disorder in the *DSM-IV* and received independent status as a standing diagnosis in the *DSM-5*. Historically, it was seen as a dimension of personality and received little professional attention until 1996, when studies examining the condition began to appear more frequently (Mataix-Cols et al., 2010). Recently, its prevalence in the population was estimated at 5.8% (Timpano et al., 2011). Compulsive hoarders collect items that they are later unable to discard. In fact, they have persistent difficulty in parting with personal possessions regardless of the actual value of those possessions. A review of 20 cases determined that magazines, newspapers, old clothes, junk mail, notes, and old receipts were the most commonly hoarded items, motivated primarily by the fear of discarding something that may be useful now or in the future (Winsberg, Cassic, & Koran, 1999). The hoarding causes clinically significant

distress or impairment and compromises living areas (which are typically cluttered and congested unless someone else intervenes). In addition to insight specifiers, the diagnosis can be specified “with excessive acquisition” when items continue to be collected beyond available space to store them. Hoarding disorder is 3 times more prevalent in older adults than younger adults, showing increasing severity with each decade of life. It tends to run in families: 50% of hoarders have a relative who also hoards (American Psychiatric Association, 2013).

There is little information on pharmacotherapy for hoarding. A multicomponent cognitive-behavioral intervention, using a version of ERP (that is, gradually exposing hoarders to discarding and nonacquiring situations), together with training in organizing and decision-making skills and cognitive therapy for dysfunctional beliefs, was effective in a controlled trial of 23 hoarders, compared to the same number of wait-listed subjects (Steketee, Frost, Tolin, Rasmussen, & Brown, 2010). After 26 sessions, the majority of both patients (80%) and therapists (70%) rated the condition as “improved.”

6.3d Trichotillomania (Hair-Pulling Disorder)

People with trichotillomania pull out their hair and show noticeable hair loss as a result. Episodes of hair pulling—which can be centered on any area of the body, though episodes most often involve the scalp, eyebrows, or eyelashes—occur in bouts that may be brief or may extend for hours. The likelihood of hair pulling is often higher when an individual is under stress, but it also occurs in circumstances when the person is relaxed or distracted, such as watching television. Increasing tension (or urge to resist) precedes the act, which itself is associated with pleasure, gratification, or relief. To qualify for the diagnosis, trichotillomania must cause significant distress or impairment and not be associated with a medical condition, such as skin inflammation, or another mental disorder that would better account for the activity.

Hair loss is widely variable across cases, ranging from thinning to areas of baldness. Individuals often deny their hair pulling and attempt to hide the effects; they may avoid social situations due to embarrassment. The condition is associated with mood disorders, anxiety disorders, and various obsessive-compulsive spectrum conditions, as well as personality disorders and intellectual disability. In adults, it is much more likely to be diagnosed in females. In the United States, 12-month prevalence estimates are 1%–2%, with females showing a ratio of 10:1 over males. The course is variable, and the condition may come and go, remit, or persist into adulthood (American Psychiatric Association, 2013).

Causal Factors

The causes of trichotillomania are not well understood. It appears to aggregate in families, and some preliminary data indicate multiple-gene involvement (Chamberlain, Menzies, Sahakian, & Fineberg, 2007). Its similarity to obsessive-compulsive disorder may suggest similar causal factors, but it may differ from that condition in several ways as well, especially in the absence of obsessions.

Treatments for Trichotillomania

Although some reviewers (e.g., Grant, Odlaug, & Potenza, 2007) have concluded that no medication has been shown to be consistently effective in treatment, others (e.g., Bloch et al., 2007; Chamberlain et al., 2007) reported that clomipramine, a tricyclic antidepressant, is more effective than placebo in reducing symptoms. However, SSRI antidepressants do not appear to be helpful; fluoxetine (Prozac®) does not differ from placebo. The most effective treatment for trichotillomania appears to be **habit reversal therapy (HRT)**, a behavioral intervention that combines self-monitoring of hair pulling, training in awareness of high-risk situations that trigger hair pulling, stimulus control techniques to interfere with or prevent hair pulling, and alternate response interventions to require

Habit reversal therapy (HRT)

Therapy that involves awareness training and engagement in a response that is incompatible with the compulsion

engagement in a substitute or incompatible activity when the temptation or urge to pull is present. The competing or incompatible response may include (for example) clenching a fist for a minute. Several reviews of treatment outcome in controlled studies have shown HRT to be superior to clomipramine, SSRIs, and placebo in reducing hair pulling and maintenance of gains during follow-up (Bloch et al., 2007; Chamberlain et al., 2007; Dell’Osso, Altamura, Allen, Marazziti, & Hollander, 2006).

6.3e Excoriation (Skin-Picking) Disorder

People with excoriation (skin-picking) disorder recurrently pick at their skin, causing bleeding, scarring, and infections. The condition tends to first appear in adolescence, often in association with acne. Although it can occur anywhere on the body, it most commonly involves the head and face. Significant amounts of time, sometimes several hours per day, are spent in the activity, despite repeated attempts to decrease or stop skin picking. Sufferers experience embarrassment and shame as a result of the picking, and the condition is commonly associated with comorbid anxiety and depression; its incidence may be as high as 5% in clinical samples (Stein et al., 2010). Among the general population, the lifetime prevalence in adults is 1.4%, with females showing a 3:1 ratio in prevalence over males. Excoriation disorder tends to run in families and is more common in those with OCD (American Psychiatric Association, 2013).

Treatments for Skin-Picking Disorder

There have been few studies of interventions for skin picking. Some have indicated that SSRI medications may provide a degree of relief (Bloch, Elliott, Thompson, & Koran, 2001), although other case studies have not found medication to be very useful (Christensen, 2004). Because of its similarity to trichotillomania, interventions based on competing response training or HRT have been employed in behavioral therapies. For example, Lane, Thompson, Reske, Gable, and Barton-Arwood (2006) found that occupying the hands with malleable balls of different textures was effective in reducing skin picking in a 9-year-old boy with comorbid ADHD and learning disability.

6.3f Other Obsessive-Compulsive Related Disorders

Substance/medication-induced obsessive-compulsive and related disorder can involve obsessions, compulsions, skin picking, hair pulling, or other repetitive body-focused behaviors that result from intoxication, withdrawal, or exposure to a medication. Most often these are associated with amphetamine or cocaine use, although other substances may be capable of producing the symptoms. There are only limited data about the condition, which appears to be very rare in the general population (American Psychiatric Association, 2013).

Obsessive-compulsive and related disorder due to another medical condition involves obsessive-compulsive symptoms that can be etiologically related to the presence of another medical condition, such as cerebral infarction. Its course usually follows that of the associated condition.

Other specified obsessive-compulsive and related disorder includes conditions that do not meet the full criteria for the disorders described earlier, such as body dysmorphic disorder symptoms in a person with actual physical flaws or without repetitive behaviors in response to concerns about appearance.

Finally, additional conditions, including those without sufficient information for a more specific diagnosis, could be listed as *unspecified obsessive-compulsive and related disorders* within this category.

BVT Lab

Improve your test scores. Practice quizzes are available at www.BVTLab.com.

Chapter Review

TO SUM UP ...

- Anxiety disorders are among the most common *DSM-5* diagnoses, affecting more than 28% of the population at some point in their lives. In general, anxiety disorders are more common among females than males.
- Anxiety includes the subjective feelings of fear, apprehension, dread, worry, and panic. Many of the anxiety disorders can include either panic attacks or agoraphobia in their symptom presentation.
- Panic attacks may be cued or situation bound (as in phobias or PTSD) or uncued (as in panic disorder). Agoraphobia involves anxiety about being in situations from which escape would be difficult if a panic attack occurred.
- There appears to be a moderate genetic factor in the development of anxiety disorders. However, life experiences, including exposure to unpredictable or uncontrollable events, play a substantial role in their development as well. Etiological models based on biological perspectives emphasize overactivity of fear circuits in the brain and deficiencies in anxiety-inhibition systems. Etiological models based on learning propose that anxiety becomes a conditioned response resulting from association between predictive internal and external stimuli and subsequent fear or panic, especially in those with a history of exposure to unpredictable or uncontrollable stressors. Psychoanalytic models presume that ego defenses are overwhelmed by anxiety from internal conflicts, which is then often displaced onto other objects or situations.
- Medications such as benzodiazepines are helpful for short-term relief of symptoms, while SSRIs are effective in treating many of the anxiety disorders for longer terms. No particular medications are effective for specific phobias, however.
- Several psychotherapies are available for anxiety disorders. The most effective ones involve exposure techniques, systematic desensitization, and cognitive-behavioral interventions. For several disorders—including specific phobias, obsessive-compulsive disorder, and generalized anxiety disorder—these psychotherapies may be more effective than medication and carry a lower rate of relapse after the end of therapy.
- The obsessive-compulsive spectrum of disorders includes conditions in which obsessions, sensed as nearly uncontrollable, produce increasing anxiety until a compulsive act relieves it. Effective psychological treatments include exposure and response prevention and habit reversal therapy. The most common pharmacotherapy includes the SSRI antidepressants.

KEY TERMS

Anxiety 140	Habit reversal therapy (HRT) 158
Cingulotomy 156	Interoceptive 143
Compulsion 153	In vivo exposure 149
Dependence 144	Obsession 153
Exposure and response prevention (ERP) 156	Panic attack 140
Exteroceptive 143	Preparedness 148
Fainting 140	Relapse 144
Fear 140	Two-factor theory 147
	Vicarious conditioning 148

QUESTIONS FOR STUDY

- Describe the differences between fear and anxiety. Are any symptoms unique to either one?
- Show how panic attacks are involved in various specific anxiety diagnoses.
- What are the most commonly applied treatments for anxiety disorders? Of these treatments, which are the most effective? Support your answer with research findings.
- Distinguish obsessions from compulsions. What are the most effective therapies for obsessive-compulsive disorders?

POP QUIZ

1. Anxiety disorders affect about _____ of the population at some point in their lives.
 - A. 3%
 - B. 11%
 - C. 28%
 - D. 46%
2. _____ is a chronic sensation not clearly associated with any specific stimulus.
 - A. Fear
 - B. Anxiety
 - C. Specific phobia
 - D. Astonishment

3. A panic attack progresses to a peak within _____.
 - A. 10 minutes
 - B. 30 minutes
 - C. 1 hour
 - D. 6 hours
4. As part of the criteria for panic disorder, at least one of the panic attacks has been followed by _____ of persistent concern about having additional attacks and a significant change in behavior related to the attacks.
 - A. 1 month
 - B. 3 months
 - C. 6 months
 - D. 12 months
5. Panic disorder affects up to _____ of the population at some point in life.
 - A. 35%
 - B. 20%
 - C. 15%
 - D. 5%
6. The term _____ is defined as relating to internal, physiological stimuli, while the term _____ is defined as relating to external, environmental stimuli.
 - A. interoceptive / exteroceptive
 - B. intrasensitive / extrasensitive
 - C. exteroceptive / interoceptive
 - D. extrasensitive / intrasensitive
7. As treatment for panic disorder, _____ may produce dependence; most patients experience a relapse when medications are discontinued.
 - A. antidepressants
 - B. benzodiazepines
 - C. phenothiazines
 - D. beta blockers
8. _____ therapies for panic disorder appear to be more effective and less subject to relapse.
 - A. Pharmacological
 - B. Psychodynamic
 - C. Psychosurgical
 - D. Cognitive-behavioral
9. Fear of strangers is known as _____.
 - A. monophobia
 - B. acrophobia
 - C. xenophobia
 - D. zoophobia

10. A nearly constant state of worry and concern characterizes _____.
 - A. agoraphobia
 - B. social anxiety disorder
 - C. specific phobia
 - D. generalized anxiety disorder
11. Grace checks her appearance in the mirror more than 40 times a day to make sure no hairs are out of place, her makeup is flawless, and her earrings have not moved. She brushes her hair for exactly 10 minutes on each side and has to count while brushing. If she loses count, she must start all over again. Grace probably suffers from _____.
 - A. social phobia
 - B. specific phobia
 - C. body dysmorphic disorder
 - D. obsessive-compulsive disorder
12. The most effective intervention for obsessive-compulsive disorder appears to be _____.
 - A. exposure and response prevention
 - B. relaxation training
 - C. interpersonal group therapy
 - D. habit reversal therapy
13. Obsessive-compulsive disorder, if untreated, usually follows a(n) _____ course with symptoms _____ during periods of stress.
 - A. acute / worsening
 - B. acute / maintaining the same degree
 - C. chronic / worsening
 - D. chronic / maintaining the same degree
14. Excoriation disorder involves _____.
 - A. hair pulling
 - B. skin picking
 - C. hoarding
 - D. none of the above
15. Habit reversal therapy involves all **except** _____.
 - A. systematic desensitization
 - B. self-monitoring
 - C. awareness of high-risk situations
 - D. engagement in incompatible activities

Additional study resources are available at www.BVTLab.com.