15

Somatic Symptom and Related Disorders

Michael J. Zvolensky, Georg H. Eifert, and Lorra Garey

Individuals exhibiting multiple somatic symptoms often present to medical practitioners believing that they are physically ill, yet upon evaluation, they are informed that there is no known physiological source underlying their reports of distress. Although many of these patients will be satisfied with negative medical examination results, a significant subgroup will anxiously continue to worry about these physical symptoms—a phenomenon traditionally known as somatization. Somatization denotes the presence of physical symptoms (e.g., chest pain) for which a demonstrable disease process or bodily oriented pathology is not identified, but which cause distress for and impairment to the individual. Individuals who do not receive a medical diagnosis for their symptoms are likely to continue to seek help for their physical symptoms, demand more physical examinations and specialist referrals, undergo costly laboratory tests, and in rare cases, even end up on an operating table (Harth & Hermes, 2007; Warwick & Salkovskis, 1990). At the extreme, such somatization behavior can interfere with life activities and goals, resulting in clinically significant impairment—a phenomenon typically classified by the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5; American Psychiatric Association, 2013) as *somatic symptom disorder*. Yet, somatization processes frequently occur in other "somatic disorders," including conversion disorder, illness anxiety disorder, and factitious disorder, as well as many other psychiatric conditions (e.g., panic disorder, major depressive disorder; DSM-5, 2013).

Somatization is a relatively common occurrence in the general medical system. Moreover, several studies suggest that approximately 30% of somatic symptoms in primary care and related clinic settings are medically unexplained (Khan, Khan, & Kroenke, 2000; Kroenke & Price, 1993; Marple, Kroenke, Lucey, Wilder, & Lucas, 1997; Narrow, Rae, Robins, & Regier, 2002; Smith et al., 2003). Further, somatization appears to maintain cross-cultural and cross-national applicability (Zvolensky, Feldner, Eifert, Vujanovic, & Solomon, 2008).

Somatization is associated with a high degree of personal suffering measured in both human and financial terms. For example, Katon, Sullivan, and Walker (2001) analyzed several studies with community, primary care, and medical specialty samples and determined that individuals with unexplained somatic symptoms experienced severe personal distress and corresponding life impairment. Somatizing individuals have higher rates of inpatient and outpatient healthcare utilization, and incur correspondingly higher inpatient and outpatient medical costs (Barsky, Orav, & Bates, 2005; Hansen, Fink, Frydenberg, & Oxhoj, 2002). In the United States, some estimates suggest the cost associated with treating somatization at approximately $256 billion a year (Barsky et al., 2005).

Importantly, somatization does not rule out the possibility of a true physical illness. In fact, many physical healthcare problems, typically quite mild, can be found among individuals with somatization problems (e.g., hypertension). Thus, somatization denotes an excessive degree of worry about physical health and overuse of medical services, relative to the severity of *identifiable* illness. Perhaps not surprisingly, those individuals without a medical explanation for their suffering seem to experience more anxiety and depression than others with similar illness experiences that do have diagnoses (Katon et al.,

2001). This difference even held in one study that examined individuals who sacrificed extensive amounts of energy and time to their care (individuals in the top 10% of frequent primary care visitors); those without an identifiable illness reported a higher degree of physical, social, and mental impairments compared with their "diagnosable" counterparts (Vedsted, Fink, Sørensen, & Olesen, 2004). Typically, these impairments are long-standing (occur across most phases of life) and tend to be exacerbated by concurrent stressors in everyday life. Although somatization is indeed a vexing healthcare problem, this domain represents an exciting opportunity for researchers from diverse disciplines to work together in a collaborative manner to better understand the relations between "body and mind" and health and disease.

The main goal of the present chapter is to provide a contemporary overview of the process of somatization as it applies to somatic symptom disorders. To some extent, our discussion is necessarily organized around the categories and classification of somatic symptom and related disorders in DSM-5 (2013). It should be noted, however, that while the presented classifications of somatic symptom disorders are based on DSM-5, we incorporate elements of DSM-IV (American Psychiatric Association, 1994). The inclusion of the DSM-IV was necessary, given the very limited research on DSM-5 to date. Furthermore, we attempt to move beyond DSM categories toward a more function-based dimensional perspective of somatization problems. We believe that such an approach is potentially useful because it targets analyses towards fundamental biobehavioral processes and thereby provides information that is likely to be directly useful for the design of clinical interventions.

The first section of the chapter briefly reviews somatic symptom and related disorders from the DSM-5 perspective, highlighting the prevalence, nature, and the diagnostic validity of such diagnoses. We then outline how a dimensional perspective that focuses on key biobehavioral processes may be a more useful approach for understanding somatic disorders than the DSM's categorical approach. Next, we address some key vulnerability processes for somatic symptom disorders. Finally, we address how a *dimensional perspective* and a focus on dysfunctional processes related to illness behavior can be translated into treatments for somatic symptom disorders.

Classification, Prevalence, and Course of Somatic Symptom and Related Disorders

Considering the extensive DSM revisions regarding somatic symptom and related disorders, we believe it is necessary to highlight the most consequential changes early in this chapter. The somatic symptom and related disorders (DSM-5) category was previously titled *somatoform disorders* (DSM-IV). According to the DSM-5 (APA, 2013), *somatic symptom disorders* may or may not accompany diagnosed medical disorders; a diagnosis of a somatic symptom or related disorder is based on the endorsement of symptoms rather than the absence of a medical explanation. Further, DSM-5 criteria emphasizes that the diagnosis should be made in consideration of the way persons present and interpret the somatic symptoms. DSM-5 also presents updated, more distinct categories for this and related disorders, and no longer includes somatization disorder, hypochondriasis, pain disorder, or body dismorphic disorder as categories of somatic symptom disorder.

DSM-5 defines the common feature of somatic symptom disorders as the conspicuousness of somatic symptoms associated with significant distress and impairment. Abnormal and/or excessive thoughts, feelings, or behaviors regarding the somatic symptoms must result in significant distress or significantly disrupt daily life.

Although the exact prevalence of specific somatic symptom disorders is not known, it is estimated that around 5–7% of the general population has somatic symptom disorder (American Psychiatric Association, 2013). DSM-5 distinguishes between five somatic symptom and related disorders: somatic symptom disorder, illness anxiety disorder, conversion disorder, psychological factors affecting other medical conditions, and factitious disorder. We begin our discussion with a brief overview of these categories.

Somatic Symptom Disorder: Somatic symptom disorder is characterized by many physical complaints, with or without clear or known physical causes, accompanied by excessive thoughts, feelings, and behaviors regarding the physical complaints. The condition may last for many years and, in some cases, extend over the entire adult life span. To meet the DSM-5 diagnostic criteria, an individual needs to present distressing somatic symptoms or somatic symptoms that significantly interfere with the individual's ability to function in daily life, and maladaptive thoughts, feelings, or behaviors about the somatic symptoms. Additionally, symptoms must be persistent for typically more than 6 months. These symptoms lead to frequent and multiple medical consultations, complex medical history, and to alterations of the person's life-style. Physical and laboratory findings may or may not detect a plausible medical condition as the cause of the somatic symptoms. Its onset is in early adulthood, course is often chronic, and prognosis is generally regarded as poor.

Illness Anxiety Disorder: A new disorder, although somewhat similar to hypochondriasis, illness anxiety disorder is characterized by a preoccupation with having or acquiring a serious illness accompanied by substantial anxiety about health and disease. Somatic symptoms, if present, are only mild in intensity, but somatic symptoms may not be present. Patients frequently seek reassurance, check their bodies, and avoid illness-related situations. Informing patients of the absence of a disease process, or explaining the benign nature of the symptoms, does not reassure the patient, and may even heighten the individual's concerns (American Psychiatric Association, 2013).

Moreover, the renewed worry over symptoms may contribute to the individual continuing to overuse medical services. The onset of illness anxiety disorder is believed to be early to middle adulthood. The course of illness anxiety is presently unclear, but thought to be a chronic and relapsing condition.

Conversion Disorder (Functional Neurological Symptom Disorder): Conversion disorder is characterized by symptoms suggesting a neurological disorder with medical investigations and neurological examinations failing to identify a neurological or general medical disorder. At times, the particular symptoms may even be inconsistent with general neurological knowledge. Patients may present with any one or a combination of motor symptoms (e.g., paralysis), seizures or convulsions, and sensory deficits (e.g., blindness, anesthesia, and aphonia). An important requirement for the diagnosis is the temporal relation between conversion symptoms and a psychological stressor such as acute grief or victimization. Patients are typically unaware of any psychological basis for their symptoms and report being unable to control them. The diagnosis of conversion disorder is rare and difficult to establish with estimates ranging between 0.001% and 0.3% (1–300 per 100,000; American Psychiatric Association, 1994). Although conversion disorder may occur at any age, onset is typically in late childhood or early adulthood. Onset is often sudden and in response to conflicts or stressful situations such as unresolved grief and sexual trauma (Sar, Islam, & Öztürk, 2009).

Psychological Factors Affecting Other Medical Conditions: Psychological factors affecting other medical conditions are characterized by the adverse effect of one or more clinically significant psychological or behavioral factors on a medical condition. Specifically, psychological or behavioral factors such as psychological distress, patterns of interpersonal interaction, coping styles, and maladaptive health behaviors increase the risk for suffering, death, or disability (American Psychiatric Association, 2013). Psychological factors affecting other medical conditions can occur across the lifespan. The prevalence of the condition is unclear, but it is believed to be a more common diagnosis than somatic symptom disorder.

Factitious Disorder: Factitious disorder is the falsification of medical or psychological symptoms, associated with identified deception. Exaggeration, fabrication, simulation, and induction are ways in which individuals can falsify illness. Great psychological distress or functional impairment may develop in persons with factitious disorder, or those with factitious disorder may impose distress or impairment on others. Factitious disorder usually occurs in intermittent episodes. Onset is usually in early adulthood. The prevalence is estimated to be about 1% (American Psychiatric Association, 2013).

Diagnostic Validity of DSM-defined Somatic Symptom and Related Disorders As the diagnostic validity of DSM-5 has been relatively unexplored, we examine the diagnostic validity of DSM-IV. DSM-IV diagnostic validity of somatoform disorders (currently titled "Somatic Symptom and Related Disorders" in the DSM-5) in relation to each other as well as to other clinical syndromes was problematic and continues to be the focus of intense debates (Mayou, Kirmayer, Simon, & Sharpe, 2005; Löwe et al., 2008). Scholars frequently observed that conditions defined under somatoform disorders did not necessarily represent distinct conditions, and thus lead to difficulties distinguishing between symptoms of somatic disorders and physical health problems abound (Mayou et al., 2005). Developers of DSM-5 recognized and attempted to address this overlap, although it is unknown if the new classifications will provide clarity.

Furthermore, the distinctiveness of somatic disorders has been questioned repeatedly. As a category, somatic disorders continue to lack conceptual coherence and clearly defined diagnostic criteria. According to DSM-5, comorbidity is frequent and somatic symptom disorders diagnoses may accompany anxiety or depression disorders (American Psychiatric Association, 2013). Van der Feltz-Cornelis and van Balkom (2010) suggested that the DSM-5 committee completely abolish this category and simply re-categorize each subcategory so that it is subsumed under cosyndromal disorders (i.e., depressive disorders and anxiety disorders). Further, while categories are used to group items that share characteristics, scholars suggests that this category lacks a unifying principle (Sykes, 2012). The lack of a unifying principle adds further support for the abolishment of this category.

The general diagnostic criteria overlap between psychiatric disorders associated with somatic symptom disorders and somatic symptom disorders, leads to high rates of multiple diagnoses. Depression and somatization, for instance, are four times more likely to co-occur than not (Leiknes, Finset, Moum, & Sandanger, 2008). Löwe and colleagues (2008) reviewed several studies and concluded that overlap with depressive and/or anxiety disorders occurred in as many as 26–59% of cases. Mayou and colleagues (2005) point out that the subcategories are unreliable and arbitrary. Many of the subcategories fail to achieve acceptable standards of reliability, which can negatively affect patient's rights to medical-legal and insurance entitlements.

Moreover, Bass and Murphy (1995) questioned the distinction between somatic symptom disorders and personality disorders, given the high rates of comorbidity between the two. More recent research by Garcia-Campayo, Alda, Sobradiel, Olivan, and Pascual (2007) showed that individuals with somatic disorders were 2.2 times more likely to have a personality disorder than other individuals with psychiatric diagnoses. Chaturvedi, Desai and Shaligram (2006) note that somatization and *abnormal illness behavior* are intricately related. Perhaps new diagnostic standards should pay greater attention to an individual's actions, but then again, abnormal illness

behavior can develop anywhere, irrespective of a person's psychiatric diagnosis.

First (2011) argued that proposed revisions to DSM-5, which were eventually slightly revised, accepted, and integrated, present ambiguous and overlapping criteria for assessing somatic symptom disorder. Specifically, criteria B lists three items that are conceptually similar, leading to endorsement of multiple, related items. Given that DSM-5 retains the arbitrary threshold for number of symptoms to determine severity, clinicians may inappropriately specify the severity of the disorder due to the conceptual overlap in criteria.

Cultural influences on somatization processes are well documented (Ryder et al., 2008; Zvolensky et al., 2008). Based on a review of data from cross-cultural studies, Escobar and colleagues (2001) initially concluded that there is "considerable cultural variation in the expression of somatizing syndromes" (p. 226). Kirmayer and Sartorious (2009) have explained this variation as a function of different "symptom schemas" that reflect cultural conceptions of suffering and distress, and which are rooted in cultural causal explanations (e.g. cellular biology in Western medicine; the balance of the body's basic constituents—fire, earth, metal, water, wood—in traditional Korean culture; preservation of vital energy in Indian culture, etc.; p. 23). Thus, the specific symptoms of certain disorders often appear to be more a function of the individual's culture than of some underlying (distinct) biologically based disease process. For example, it would be helpful for clinicians to know that for some individuals of Indian descent, the experience of semen in the urine ("*dhat*") is frequently not a delusion but a somatization related to fatigue and depression, a representation of feeling sapped of "vital energy"; likewise, it would also be helpful to know that epigastric burning among individuals of Korean descent may not be heartburn but a somatic experience of intense, culturally inappropriate emotion ("*hwa-byong*" or "fire illness") (Kirmayer & Sartorious, 2009). Symptom lists for Europe and North America would, by contrast, focus on the most frequent areas of concern in these locations, heart disease and cancer (see López and Guarnaccia, Chapter 4 in this volume).

Mayou and colleagues (2005) note that the terms "somatoform" or "somatization" are often unacceptable to patients because they imply that the patients' symptoms are in their mind and "not real." Patients doubt that clinicians appreciate the reality and authenticity of their symptoms. At a more fundamental level, Mayou and associates (2005) criticized the distinction between somatic and psychological problems as inherently dualistic, which does not translate well into other cultures that have a less dualistic view of mind and body (e.g., Asia and China) than Western culture. The notion that problems can be neatly divided into those that can and cannot be explained by disease is indeed unlikely at best. Several authors also point to potential pathophysiological mechanisms that may underlie unexplained physical symptoms such as chest pain (Sharpe & Bass, 1992; Pilgrim & Wyss, 2008; Yilmaz et al., 2008). We often just do not understand them very well—and that is what we should tell patients, rather than using terms such as non-organic when describing their symptoms. We also need to recognize that there is frequently a reciprocal relation or "looping effect" between health anxiety and somatic symptoms, in which attending to sensations increases their salience and intensity (Kirmayer & Sartorious, 2009). This could occur either indirectly, through psychological processes, or directly, involving the behaviors designed to reduce anxiety over physical symptoms paradoxically increasing physical symptoms (e.g. constantly checking/scratching a lump increasing its size and irritation).

Although the DSM-5 attempts to address issues such as overlap and lack of clarity regarding boundaries for diagnoses, it still has many issues to overcome. DSM-5 recognizes the possible comorbidity between each category of somatic symptom disorders and other disorders, and proposes that concurrent diagnoses should be explored. Further, the differential diagnosis section tries to disentangle the overlapping features; however, the lack of clear, solid boundaries may result in patients being inappropriately diagnosed with a mental disorder and thus receiving inappropriate treatment (Sykes, 2012).

Toward a Dimensional Framework for Understanding Pathogenic Processes In light of the long-standing diagnostic problems with the somatic disorder category, Mayou and colleagues (2005) suggested abolishing this category in DSM-5 because the concept and category has consistently failed clinicians and patients alike. They suggested redistributing the somatic disorders to other diagnostic categories. More recently, Van der Feltz-Cornelis and Van Balkom (2010) suggested a similar notion. Löwe and colleagues (2008), on the other hand, have argued against a complete abolition of the somatic disorder category on the basis that, faulty or not, it has come to have huge personal, clinical, and societal importance, and abolishing the category would effectively abandon a large group of patients and exclude them from treatment. They argued for revisions that would integrate somatic disorders into the greater medical field, so that mental and physical health workers have identical, interdisciplinary diagnostic and treatment standards.

We previously argued (Eifert, Lejuez & Bouman, 1998; Eifert, Zvolensky & Lejuez, 2000) that overlap between related categories is not a problem of "comorbidity" or inaccurate definitions, but rather, a result of similar psychological processes involved in these conditions. Accordingly, we suggest adopting a dimensional approach to understanding illness-related concerns that can identify key biobehavioral processes. To illustrate this approach, we discuss focal dimensions of health anxiety, a psychological process that characterizes, in part, many somatic symptom disorders as well as related conditions

(e.g., panic disorder). We view health anxiety as a psychological process where persons present with problems that fall on a continuum along four dimensions:

1. Preoccupation with the body and its functioning. Such bodily preoccupation, especially when coupled with somatic complaints, may produce a state of somatic uncertainty and form the basis for the other three dimensions of the disorder.
2. Disease suspicion or conviction. The person has the suspicion or is convinced of having a serious physical disease; suspicion and conviction are on a continuum of strength, and in rare cases the conviction may reach delusional intensity.
3. Disease fear. The person fears having a serious physical disease.
4. Safety-seeking behavior such as repeated requests for medical examinations and tests, bodily checking, verbal complaints, and seeking reassurance. The function of such behavior is to reduce worry and anxiety over physical illness (Eifert et al. 2000; Salkovskis & Warwick, 2001).

A person could obtain a high score on any one or all four dimensions of health anxiety. For example, disease suspicion/conviction may or may not be accompanied by a strong fear of the suspected disease. Clinically, this feature is most apparent in patient's resistance to medical reassurance that nothing is wrong. This is particularly evident in a study by Rief, Heitmüller, Reisberg, and Rüddel (2006), which found that when patients with medically unexplained symptoms were asked to recall meetings with their doctor, the patients remembered a higher likelihood of medical explanations than their doctors actually gave. Accordingly, a dimensional classification system could help overcome some challenges inherent to a traditional diagnostic perspective of somatic disorders. Moreover, identifying dimensions that allow a classification of illness behavior based on the function that such behavior serves, rather than just its topography, might lead to a better understanding and improved treatments of persons with somatic problems (Eifert & Lau, 2001).

General Vulnerability Processes for Abnormal Illness Behavior

Given our previous discussion as to prototypical characteristics of health anxiety, the next logical question pertains to the types of processes that increase or decrease the risk for developing abnormal illness behavior. As discussed at the onset of this chapter, just about all people experience distressing physical sensations at some point in their lives. Moreover, a substantial percentage will even experience robust internal physical (interoceptive) reactions in the form of panic attacks, limited symptom panic attacks, gastrointestinal distress, respiratory infections, strained muscles, and so on. In fact, such bodily distress is so common to the human experience that it seems almost inconceivable to imagine a person going through life without experiencing at least some significant somatic disturbance. These normal experiences of physical symptoms become problematic when they begin to interfere with a person's life due to obsessive preoccupation with them and excessive rigid behaviors designed to control, reduce, or escape from them. Such pervasive experiential avoidance behavior seems to be at the core of many anxiety-related clinical problems (Forsyth, Eifert, & Barrios, 2006; Walker & Furer, 2008)—in fact, maladaptive avoidance behavior is a core feature of all anxiety disorders (Eifert & Forsyth, 2005; see also Williams, Chapter 9 in this volume).

Although systematic knowledge about causes is lacking, factors such as parental modeling, stressful life events, biological or genetic components, and greater cultural acceptance of physical versus mental illness appear to be related to the development of somatic disorders (Heinrich, 2004). One finding, that there is a decreased likelihood of somatic symptom diagnoses when primary care physicians have a more personal and long-term relationship with patients, also suggests that open, honest doctor–patient relationships protect against somatization diagnoses (Gureje, 2004). Difficulty in tolerating emotions (experiential avoidance) has also been shown to be associated with the development of somatization symptoms (Chawla & Ostafin, 2007). Difficulty expressing emotions (alexithymia) and negative affect/neuroticism are associated with symptom increase and persistence (De Gucht, 2002).

As these studies indicate, there are a number of biopsychosocial processes that increase vulnerability for the development of somatic symptom pathology or abnormal illness behavior generally. The processes that we focus on in this section include: (1) an inherited risk for emotional responsivity to physical sensations; (2) deficits in emotion regulatory skills; and (3) language-based and observational learning.

Before this discussion, however, we need to briefly define what we mean by the term *abnormal illness behavior*. Pilowsky (1993, p. 62) defined abnormal illness behavior as the "persistence of a maladaptive mode of experiencing, perceiving, evaluating, and responding to one's own health status" despite the fact that a doctor has conducted a comprehensive assessment of relevant biological, psychological, social and cultural factors and provided the patient with feedback about the results of these assessments and opportunities for discussion and clarification of the results. Thus, abnormal illness behavior essentially refers to the disagreement between the doctor and patient about the sick role to which the patient feels entitled. The concept of abnormal illness behavior is valuable for understanding not only patients with functional somatic symptoms but also the behavioral aspects of all illness (Chaturvedi et al., 2006).

Inherited Risk for Emotional Responsivity to Physical Sensations There is consistent empirical evidence for a strong inherited component or substrate for negative emotionality. Researchers have referred to this inherited disposition by a number of different terms, including *negative affectivity*, *trait anxiety*, and *neuroticism* (Barlow, 2001; Lau, Ely, & Stevenson, 2006). These synonymous labels are intended to capture a general predisposition to experience negative affect (e.g., anxiety) and perhaps abrupt emotional reactivity (e.g., panic) to challenging or stressful life events.

Although the characteristics of negative affectivity vary across specific negative emotional experiences (e.g., panic versus anger), they all posit that high degrees of negative emotionality are associated with a lower threshold of initial affective response, slower recovery to baseline, and greater reactivation of arousal with repeated exposure to stressful events. High reactivity is generally associated with inhibited temperament, whereas low reactivity is associated with uninhibited temperament (Kagan, 1989; Letcher, Smart, Sanson, & Toumbourou, 2009). Thus, individual differences in temperament are often related to the experience and regulation of affect because they constrain or facilitate certain types of responding. For example, the temperamental characteristic of neuroticism is related to tendencies to experience low levels of self-esteem in situations of interpersonal conflict, and global and day-to-day perceptions of relationships are more dependent on self-esteem in highly neurotic individuals than in others (Denissen & Penke, 2008).

As behavioral genetic research continues to make important strides in our understanding of emotional functioning, we may eventually have specific models articulating the extent to which a specific gene or combination of genes contributes to a specific anxiety disorder or health disorder. At this stage, however, it appears that a general disposition for negative affectivity is inherited. The contribution of this inherited component is estimated to be at approximately 30% in the development of somatic-related disorders (Kendler, Walters, Truett, & Heath, 1995). No research has documented the exact proportion of explained variance for the development of a specific disorder, although there is some evidence that there are both shared and specific genetic vulnerabilities across anxiety-related disorders (Smoller, Block, & Young, 2009). It is likely that a genetic predisposition creates the biological conditions that make people prone to respond with anxiety and panic reactions to certain bodily changes and processes (Walker & Furer, 2008, p. 369) One research challenge in this domain will be to specify how genetic vulnerabilities (once they are identified) might influence the pathogenesis of a specific disorder.

Development of Emotion Self-Regulatory Skills Research has shown that early in life humans develop a repertoire of regulatory oriented skills to deal with elevated levels of bodily arousal and concomitant emotional distress. For example, parents often distract distressed infants by orienting them to other stimuli, and children eventually learn to reorient and soothe themselves (Rueda & Rothbart, 2009, p. 26). Thus, the effective management of bodily states, particularly negative emotional experiences, is a critical early step in psychological development. As children mature, they learn to approach and avoid salient environmental stimuli, and the members of society increasingly expect them to gain better emotion regulatory skills (e.g., suppression of crying in school). By adulthood, the individual is expected to have sophisticated emotional control skills and the ability to learn new regulatory skills for a variety of changing sociocultural contexts. For example, adults are not supposed to demonstrate signs of emotional distress in social, performance, or work settings. Adults without emotion regulation skills often suffer in modern society. An interesting series of studies by Kantor-Martynuska (2009) provides some insights why this may be the case. She found that highly emotionally reactive individuals were more likely to prefer music at low volumes and to recall music as having been played loudly. This suggests that reactive individuals may have an increased sensitivity to arousing stimuli (music) and a tendency to experience it as aversive.

The preceding discussion highlights the notion that emotion regulation skills are expected to increase consistently across the lifespan and are an integral component of mental health (Stewart, Zvolensky, & Eifert, 2002). When individuals lack the ability to effectively alter their emotional experiences, they are more susceptible to physical discomfort, negative affect, and anxiety (Rothbart, Ziaie, & O'Boyle, 1992). An important way in which children learn emotional regulation skills is through exercising their capacity to explore their world, both literally and metaphorically. Developmental researchers originally referred to these experiences as *mastery learning opportunities* (Rothbart, 1989). Impoverished environments where parents or caregivers (e.g., teachers) respond to children in a non-contingent manner produce more emotional distress than do environments characterized by contingent outcomes. For example, van der Bruggen, Stams, Bögels, and Paulussen-Hoogeboom (2010) found that maternal rejection mediated a relationship between parent perceptions of children's negative emotionality and the children's anxiety/depression. The researchers hypothesize that maternal rejection instills a sense of helplessness in children, making them less likely to experience mastery of their negative feelings, and thereby increasing the likelihood that negative emotions eventually lead to symptoms of anxiety and depression. Similar findings have been observed in studies of non-human animals raised in environments with little control over important outcomes (e.g., availability of food; Mineka, Gunnar, & Champoux, 1986; Roma, Champoux, & Suomi, 2006). Such findings are important to understanding somatic disorders because greater degrees of perceived uncontrollability

are predictive of the tendency to view ambiguous internal stimuli as threatening (NASA Scientific and Technical Information Program Office, 2004; Zvolensky, Eifert, Lejuez, & McNeil, 1999).

Coping with emotional distress is, of course, a multidimensional process. Research indicates that coping responses are best viewed from a hierarchical model that includes first-order and higher-order dimensions (Compas et al., 2001). Indeed, researchers increasingly suggest that coping with emotional distress involves strategic (voluntary) and automatic (involuntary) responses. Additionally, Compas, Conner-Smith, Saltzman, Thomsen, and Wadsworth (2001) have categorized these coping responses along the dimensions of engagement and disengagement. Engagement responding includes active, primary control-oriented responding aimed at altering the immediate situation in some sort of direct manner (e.g., leaving a situation that one finds uncomfortable because of cardiac-related distress and tension). *Disengagement* responding includes secondary control-oriented responding aimed at adapting to an uncontrollable situation by purposively altering one's cognitive-affective response to that situation (indirect responding). For instance, an individual might adapt to pain or other aversive bodily sensations by altering their cognitive response to such events (e.g., acceptance, distraction, reframing). Overall, it is likely that individuals will develop a variety of emotion regulatory skills across the lifespan, and these skills are likely to be a product of early learning experiences.

Language-Based Learning Aside from direct forms of learning, individuals also will experience affective responses to body-related events and sensations through the utilization of language. Language serves important symbolic functions by providing humans with emotional experiences without exposure to the actual physical stimuli or events that ordinarily elicit those responses (Luoma, Hayes, & Walser, 2007; Staats & Eifert, 1990). For instance, both "knowing what to do" and "knowing what to feel" involve verbal understanding of the relation between them. Thus, the meaning of health-related anxiety in a psychological sense represents a complex act of relating largely arbitrary verbal symbolic events with other events and psychological functions within a particular context. For instance, words such as anxiety and fear either implicitly or explicitly establish relations with other events such as "I am anxious or afraid of . . . something, some event, or someone."

The relational quality of terms denoting emotions, in turn, must be tied to descriptions of behavior and events with a variety of stimulus functions (e.g., eliciting, evoking, reinforcing, and punishing) and meanings (e.g., good, bad, pleasant, unpleasant, painful). In turn, people often describe their emotional experiences metaphorically in ways that others can understand (e.g., "When I feel anxious it's like a knife going through my chest"). These metaphorical extensions have no real counterpart inside the person. Instead, they function to communicate the meaning of emotional experience (feeling threatened to the point of fearing death) by identifying and relating events with known stimulus functions (a sharp knife can cut into a chest and cause death).

Society determines what kind of stimuli and events are placed in relation to each other and the nature of that relation (Hayes & Wilson, 1994). These arbitrary relations are learned and function in a variety of ways (Sidman, 1994; Staats & Eifert, 1990). Individuals become anxious about particular health-related experiences or "symptoms" because they read and hear about them in the specific cultural context in which they live. This may be one of the main reasons why cultural variations in somatic disorders are widespread and can be observed with such great regularity (Escobar et al., 2001). For instance, people in Western societies become anxious when they notice a fast or irregular heart beat because they have seen or heard many times that this event may be a sign of a heart attack. In Southeast Asian and African cultures, it is quite common to observe a phenomenon that has been labeled "*koro*," which describes an individual overcome with the belief that his penis—or, in females, her nipples—are retracting or shrinking, with fear that the organ will disappear (Barlow, 2002). In contrast, this phenomenon is quite rare in Western culture. Thus, the overwhelming finding from cross-cultural studies is that the somatic manifestation and expression of emotional distress is universal but the focus of somatic concern may vary in different cultures (see López and Guarnaccia, Chapter 4 in this volume).

The most important point is that persons with somatic disorders have likely developed complex repertoires of verbal and other symbolic responses that elicit negative affect and serve as discriminative stimuli for escape or avoidance behavior (Staats & Eifert, 1990). Thus, for otherwise healthy people, the sensations of a beating heart or chest pain may lead to a sequence of verbal and autonomic events that result in the belief that they are having a heart attack (Eifert, 1992). In this instance, a fast or irregular heartbeat is not just a felt beating heart. Instead, it is an acquired and verbally mediated formulation of what it *means* to have a fast or irregular heartbeat or chest pain (e.g., "I have heart disease" or "I am suffering from a heart attack"). Not only may the person respond to such sensations by rushing to an emergency room, but also any other public or private stimulus events associated with this response may now acquire similar negative functions (e.g., physical exercise, smoking, working hard). In this way, a variety of behaviors and events can come to elicit the physiological event that the person then misconstrues as dangerous.

Observational Learning It is also likely that persons who develop somatic symptom disorders have been exposed to negative health-related events to a greater degree than

persons who do not develop these disorders. For example, some studies indicate that a significant number of persons with cardiophobia, defined as an irrational fear of heart disease, have observed heart disease and its potentially lethal effects (e.g., death) in relatives and close friends (Eifert et al., 2000). These persons had been exposed directly to the physical and emotionally painful consequences of heart disease. As a result, they may also have had more exposure to heart-focused perceptions and interpretations of physical symptoms and physiological processes.

Observational learning is strongly involved in learning pain tolerance, pain ratings, and non-verbal expressions of pain (Flor, Birbaumer, & Turk, 1990). Such observational learning may increase the likelihood of expressing and interpreting arousal and pain in later life as a heart problem because socially acquired perceptions and interpretations of symptoms largely determine how people deal with illness. For instance, if one or both parents have heart disease, children might observe their parent's response to a heart problem. If the behavior that is modeled is maladaptive (e.g., excessive illness behavior), these children will not only be more likely to respond to stress with increased cardiovascular activity, but they will have also learned maladaptive labeling and interpretation of such symptoms and have fewer adaptive coping skills.

Individuals afflicted with somatic symptom disorders also report poorer overall health (Eifert et al., 2000). Learned maladaptive coping skills may contribute to this observation. For example, men with more somatic symptoms tend to drink more alcohol (Vijayasiri, Richman, & Rospenda, 2012). This maladaptive coping strategy was possibly observed as a child and then carried forward in life as a means to cope, especially given that parental alcohol use predicts adolescent alcohol use (Kerr, Capaldi, Pears, & Owen, 2012) and adolescent alcohol use in turn predicts adult alcohol use (Patrick, Wray-Lake, Finlay, & Maggs, 2010). The true underlying process between maladaptive coping skills, health behaviors, and somatic symptoms, however, is complex and more research is needed to disentangle these relations.

Taken together, research suggests a variety of factors may promote the development of the type of abnormal illness behavior found in somatic symptom disorders. These processes are likely nonspecific in the sense that they increase the chance of negative emotional responding and poor affect regulatory strategies. Exposure to specific illnesses or to persons who model the potential dangers of certain physical disorders may increase an individual's general vulnerability. Continued research in each of these general domains will improve our ability to predict who will develop a specific type of somatic disorder.

General Treatment Strategies for Somatic Symptom Disorders

Cognitive-behavioral theories and research have been helpful in providing a fledgling basis for a better understanding and treatment of persons with somatization problems. Important progress has been made in particular for persons with health anxiety (Eifert & Lau, 2001; Salkovskis & Warwick, 2001) and chronic pain (e.g., Flor et al. 1990; Kerns, Thorn, & Dixon, 2006; Schermelleh, Eifert, Moosbrugger, & Frank, 1997). We now overview these treatment strategies at a general level and how they can be applied to specific types of somatic disorders.

Psychologically distressed patients who present with unexplained somatic symptoms are high users of medical care and their doctors regard them as frustrating and difficult to treat (Mayou, 2009). There is often a mismatch between the expectations of these patients and their doctors' abilities and communication skills. For instance, terms such as *functional heart problem*, *nervous heart*, *atypical chest pain*, and *pseudoangina*, when used to "diagnose" unexplained chest pain, can easily be misinterpreted by a patient who is determined to believe that some significant cardiac disease is being described (Eifert et al., 2000). Healthcare providers often feel frustrated and emotionally drained because these patients obviously need psychological support but resent being referred to a psychologist or psychiatrist.

Patients often perceive the use of diagnostic labels such as hypochondriasis as an insult because these labels seem to imply that the patients' problems are not real and "just in their head." The controversy surrounding hypochondriasis supported the removal of it and, to a degree, the placement of it with illness anxiety disorder (Mayou et al., 2005). This new "recategorization," however, may not yield a large degree of improvement.

Wainwright, Calnan, O'Neil, Winterbottom, and Watkins (2006) attribute the patient perceived unacceptability of a hypochondriasis diagnosis to modern society's moral "hierarchy of illness" in which observable, stoically born physical pathology is elevated and legitimized, and somatization is denigrated and thought of as "faking it" or malingering (p. 79). They therefore argue that accepting and understanding, rather than refuting or arguing with the patient's symptoms, is the more important strategy for engaging the patient in a therapeutic working relationship. Lipsitt and Starcevic (2006) similarly state that it is extremely important for healthcare providers to treat the somatizing patient in a warm, respectful manner, without prejudice based on previous assessments from other clinicians. Healthcare professionals can help make the patient feel listened to and let them know that they regard their symptoms as "real" and do not view them as "crazy." They can also ease the patient's anxieties and enhance the relationship with the patient by telling the patient that worrying about their symptoms is reasonable and legitimate. A solid and trusting working relationship is essential for optimal help. In the engagement stage of treatment, patients can also be told that there may be alternative explanations for the difficulties they are experiencing (e.g. pathophysiological pathways; Pilgrim & Wyss, 2008; Yilmaz et al., 2008). The general treatment strategy

is to test such alternative or benign medical explanations for symptoms and to conduct therapy in the context of an experiment that provides an opportunity for testing alternative hypotheses (Eifert & Lau, 2001; Salkovskis, 1996).

Rather than merely telling patients there is no "organic" reason for their chest pain, an explanation of symptoms that overcomes the non-organic–organic dualism provides the patient with a more acceptable rationale and reassurance (Eifert, Hodson, Tracey, Seville, & Gunawardane, 1996). For instance, to provide a patient with a credible explanation of how anxiety and chest-wall muscle tension can result in chest pain, patients may be given a chest-focused relaxation with electromyographic feedback that literally shows them how they can change their chest tension levels. Salkovskis and Warwick (2001) found that reassurance which only informs the patients that there is nothing organically wrong with their bodies will actually increase future reassurance-seeking rather than decrease it. Appropriate reassurance and feedback that provides the patient with new and alternative explanations is the key to successful treatment and may help prevent the development of chronic somatization problems. In a controlled case study, Eifert and Lau (2001) found that behavioral experiments were not only useful in developing alternative symptom explanations but also in teaching the patient to reassure herself rather than seeking therapist reassurance. The treatment strategies and techniques targeting the dimensions of abnormal illness behavior related to heart-focused anxiety are summarized in Table 15.1.

Medical professionals need to be trained to provide alternative explanations rather than vague pseudo-medical labels and to acknowledge rather than dismiss patients' concerns. At the same time, withholding unnecessary medication and medical examinations (response prevention for safety-seeking behavior) is a crucial part of treatment. For instance, in their interviews with British physicians, Wainwright and colleagues (2006) describe how some doctors have adopted patient-centered philosophies and view diagnosis as a negotiated process that can approach psychosocial causality covertly over several sessions, thereby balancing a doctor's ethical commitment to scientifically rigorous standards of practice while accommodating a patient's resistance to psychological explanations. The researchers also found, however, that many physicians still struggle when dealing with these issues, so the focus should remain on developing programs for health care providers that help them communicate more effectively with their patients. Early work focused on arming physicians with basic psychological intervention skills, such as empathic listening to complaints and symptoms (Goldberg, Gask, & O'Dowd, 1989; Rost, Kashner, & Smith, 1995; Smith, Rost, & Kashner, 1995). Physicians were also taught not to reinforce patients' abnormal illness behavior by providing expensive diagnostic procedures, surgeries, and hospitalizations (Rost et al., 1995; Smith et al., 1995). Withholding such procedures also prevents iatrogenic diseases and counteracts patients' disease convictions. Mayou (2009) recommends the use of a stepped-care approach, which involves mixing therapeutic intervention with medical reassessment at various set intervals. This should also help to counteract patients' disease convictions while still moving them forward in their treatment. The results of these physician-focused interventions suggest that this balanced approach to patient care can reduce healthcare costs and at the same time increase the psychological adjustment of patients.

Finally, it is essential that we move patients from an almost exclusive focus on symptoms, which may or may not be changeable and/or controllable, to goals and behaviors that are controllable and changeable. After an initial empathic discussion of symptoms, treatment needs to shift toward more changeable behavioral targets, such as occupational problem solving, social skills training,

TABLE 15.1

Key Treatment Components Targeting Various Dimensions and Symptoms of Heart-Focused Anxiety (adapted from Eifert and Lau, 2001)

Dimensions	Treatment Strategy	Techniques
Preoccupation with heart	Demonstrate that chest pain/heart sensations are not dangerous	Reduce avoidance/expose to cardiac-related stimuli
Disease fear	Extinction of fear and exposure to avoided activities	Exposure to interoceptive (particularly cardioceptive) cues
		Reinforce "dangerous" behavior (e.g., strenuous exercise)
Disease conviction	Testing alternative symptom explanations	Explain impact of anxiety and tension on body (chest pain)
		Conduct behavioral experiments to test hypotheses
		Review evidence for/against heart disease
		Review evidence for cardiac vs. tension chest pain
Safety/reassurance-seeking	Extinction of help and reassurance-seeking	Review results of previous tests
		Withhold reassurance
		Refuse further tests
		Do physical exercise while preventing pulse checking
Physical (panic) symptoms	Reduce chronic tension and overbreathing	Chest muscle relaxation
		Teach slow diaphragmatic vs. thoracic breathing

Note: Although this example deals with heart-focused anxiety, the process dimensions are adaptable and applicable to other somatization problems.

and quality of life enhancement (Rief, Hiller, Geissner, & Fichter, 1995; Mayou, 2009). Behavioral activation treatment programs (Lejuez, Hopko, & Hopko, 2001) appear to be valuable therapists' tools to help move patients in their "valued direction" (Hayes, Strosahl, & Wilson, 2000) and at the same time reduce negative affect.

Acceptance and commitment therapy (ACT), a new acceptance-based behavior therapy, has shown great promise in the treatment of chronic pain (Vowles, Wetherell, & Sorrell, 2009) and anxiety disorders (Eifert & Forsyth, 2005). In future, this approach could easily be extended to people suffering from all types of health-related anxiety and concerns because it directly targets the experiential avoidance behavior that tends to interfere with people's daily functioning. ACT helps people to refocus their efforts on areas in their lives where they can affect change, and where they are in control, rather than wasting their time with fruitless attempts to control or change bodily sensations and their evaluations of them, which are very difficult, if not impossible, to change. ACT provides very useful therapeutic tools for helping people reorient their lives away from managing symptoms, pain and disability to moving forward and living a life guided by their values—what truly matters to them (McKay, Forsyth, & Eifert, 2010).

Specific Treatment Recommendations

Somatic Symptom Disorder It may be most useful to think of somatic problems as requiring management rather than treatment (e.g., Bass & Benjamin, 1993). Early diagnosis and the prevention of unnecessary medical and surgical investigation are of primary importance. For instance, a study by Hoyer and colleagues (2008) has shown that the specific assessment of heart-focused anxiety may help identify individuals with elevated levels of heart-focused anxiety who might benefit from interventions to help them adjust to the effects of surgery and lingering cardiac problems. Most somatic symptom patients have specific expectations regarding treatment goals and procedures and try to persuade their doctors to follow their wishes for further medical investigations and treatments. Bass and Murphy (1990) state that treatment often involves long-term supportive psychotherapy and must be directed toward controlling the demands on medical care as well as the treatment of symptoms and social disability. They recommend the following four steps:

a. encourage a long-term supportive relationship with only one understanding primary care physician to prevent doctor-shopping and to coordinate all actions;
b. see patients on regular appointments rather than on demand to prevent reinforcement of illness behavior;
c. view patient's physical complaints as a form of communication rather than as evidence of disease; and
d. minimize the use of psychotropic drugs and/or analgesic medication.

In general, adaptive behavior is encouraged and promoted, whereas sick role behavior is ignored as much as possible. Kashner, Rost, Cohen, Anderson, and Smith (1995) focused on coping with the nature and consequences of the physical symptoms, general problem solving, and helping patients take more control of their lives. These authors found that eight sessions of brief group therapy improved physical and mental health at 1-year follow-up.

A randomized controlled trial examining a comprehensive cognitive-behavioral approach for dealing with medically unexplained physical symptoms was conducted by Speckens and colleagues (1995). A cognitive-behavioral intervention group of 39 general medical outpatients was compared with a control group of 40 patients receiving optimized medical care. Treatment included imaginary exposure and distraction techniques to break the vicious circles of cognitive avoidance and preoccupation; activity scheduling, exposure *in vivo*, and response prevention to decrease avoidance behavior; relaxation training, breathing exercises, and physical exercises; and problem-solving or social skills training to overcome any problems in interpersonal relationships. At both 6- and 12-month follow-up, the intervention group reported lower intensity and frequency of symptoms, reduced illness behavior, less sleep impairment, and fewer limitations in social and leisure activities than did the control group.

More recently, Allen, Woolfolk, Escobar, Gara, and Hammer (2006) and Escobar and colleagues (2007) conducted randomized controlled trials investigating the efficacy of cognitive behavior therapy interventions for the treatment of somatic symptom disorder. Both treatments were administered in a primary care setting, but in contrast to earlier studies, patients had to exhibit more severe somatic symptoms. Allen and associates (2006) found that trained, independent raters reported less severe somatic symptoms at follow-ups among patients treated with cognitive behavioral therapy. Escobar and associates (2007) observed similar finding in that patients treated with cognitive behavioral therapy had significant improvements in clinician-rated physical symptoms and depression.

Conversion Disorder An important first step in the treatment of conversion symptoms is their early recognition in which a physical examination plays a crucial role. In many cases, a positive diagnosis can be made on the basis of the rather untypical or bizarre symptoms. Since conversion symptoms vary widely across patients, treatment needs to be individualized. Identifying precipitating stressors is crucial so that patients can be taught more adaptive ways of coping with these stressors. Occasionally, manipulation of the patient's social environmental is necessary to reduce the influence of secondary gain, such as attention from family and friends. Partners and significant others may have to learn how to reinforce the patients non-symptomatic behavior.

Behavior therapy has shown some efficacy in the treatment of conversion disorder, particularly for patients that

primarily exhibit motor symptoms and gait disturbances (Lipsitt & Starcevic, 2006; Speed, 1996). The program consisted of providing or withholding reinforcement depending on the patient's attempt at normalizing gait and increasing motor movement through specific exercises.

Researchers have successfully utilized a variety of other treatments, including psychotherapy and hypnotherapy, to treat conversion disorder (see Rosebush & Mazurek, 2011). Moene, Spinhaven, Hoogduin, and VanDyck (2003) administered hypnosis to patients diagnosed with conversion disorder, motor type, and observed a significant improvement in behavioral symptoms and motor disability compared with a control group. It is worth noting, however, that the efficacy of conversion disorder treatments is inconclusive and inconsistent (Kroenke, 2007; Rosebush & Mazurek, 2011).

Conclusions

Somatic symptoms, complaints, and concerns are very common in the general population. These problems are costly to the individuals concerned in terms of distress and financial expense, as well as to society in terms of lost productivity and health care costs. Compared with other common psychological dysfunctions (e.g., anxiety and depression), our present conceptual understanding of somatic symptom and related disorders is poor, and comprehensive integrative models are still lacking.

One factor that has impeded a better understanding of the somatic symptom disorders is the unsatisfactory and somewhat arbitrary nature of the DSM-IV classification. In view of the conceptual and diagnostic confusion, vagueness, and imprecision detailed in the DSM-IV, we greatly anticipate the research to be done exploring the new classifications and appropriateness of language in the new DSM-5. "Comorbidity" of somatic symptom disorders with anxiety disorders and depression is not a diagnostic problem but an indication that there are similarities in the underlying psychopathological processes (Aikens, Zvolensky, & Eifert, 2001). We have outlined some commonalities in emotional dysregulation processes particularly in relation to anxiety. A practical consequence for researchers and clinicians is to give up their focus on the individual disorders and increase their efforts at identifying the common functions of symptoms in persons with different somatic problems (Eifert, 1996; Kirmayer & Sartorious, 2009).

The complex relationships between the physical and psychological aspects of somatic symptom disorders has led to much confusion. We caution against an overreliance upon medical diagnostic procedures and medical theory. At the same time, research and service delivery would benefit from a more balanced approach. This approach should focus not just on finding or excluding somatic abnormalities but also on combining current medical knowledge and diagnostic techniques with the psychological assessments of a patient's behavior, cognitive processes, and social relationships (cf. Fink, 1996; Löwe et al., 2008). In our work with cardiac patients we have observed how a simple reliance on one source of information (medical or psychological) was inadequate for many patients. Instead, it was the combination of sophisticated medical tests and psychological information that yielded the type of knowledge that was most useful for recommending and designing the most appropriate treatment for the individual patient.

Hence, perhaps one of the most compelling conclusions arising from this chapter is that somatic symptom disorders cannot be adequately understood, assessed, and treated from a single perspective. Both the classification and research could be improved by adopting a multidisciplinary approach and an integrated biopsychosocial perspective. For example, Mayou, Bass and Sharpe (1995) proposed a multidimensional classification of patients with functional somatic symptoms along five dimensions: (1) Number and type of somatic symptoms; (2) mental state (mood and psychiatric disorder); (3) cognitions (e.g., symptom misinterpretations, disease conviction); (4) behavioral and functional impairment (illness behavior, avoidance, use of health services); and (5) pathophysiological disturbance (organic diseases, physiological mechanisms such as hyperventilation). As described in the section on health anxiety, Löwe and colleagues (2008) present an alternate diagnostic system that focuses on the presence of behaviors, some of which are abnormal illness behaviors. Individuals should be assessed for: (1) Dysfunctional cognitions; (2) excessive healthcare use; (3) selective attention to bodily signals; (4) persistent attribution of symptoms to undiagnosed conditions; (5) avoidance and decreased activity; and (6) functional impairments due to somatization. These criteria are behavioral and would be of more immediate relevance for treatment than diagnosing.

Although DSM-5 integrated some elements similar to the recommendations described above, the utility and appropriateness of the wording and classification is still unknown. The issues outlined throughout this chapter regarding DSM-IV and DSM-5 support assessing somatic symptom disorder using tools other than the DSM. Thus, rather than attempting to find the "correct diagnosis," we recommend assessment along the crucial dimensions involved in the regulation of maladaptive illness behavior and devising treatment programs based on such assessments. This may be more valuable than diagnosing according to the number and type of physical complaints.

A multicausal perspective suggests several potentially fruitful lines for future psychological research into somatic disorders, such as an increased focus on information processing behavior (attribution, attention, and memory), and environmental contingencies for illness behavior (e.g., social, occupational, medical). Psychoneuroimmunological studies may help to clarify particular aspects of the nature of the interface between pathophysiological changes and individual responses

to such changes as exemplified in some chronic pain research (cf. Flor et al., 1990). Although the past emphasis on the problems of patients with no demonstrable physical pathology was worthwhile and deserves continued attention, the gray area of persons with some organic pathology, bodily symptoms, and psychological distress deserves greater recognition and needs to be investigated more carefully.

Treatment programs and outcomes are likely to be enhanced by an improved conceptual understanding of these problems. The need for better theories and treatments is even more pressing for those somatic problems that have been particularly neglected in the past such as conversion problems. The relative success of recent cognitive-behavioral treatment programs for persons with unexplained physical symptoms, health anxiety, or chronic pain is promising. These treatment successes may help change the common perception of healthcare providers that people with such problems are just a "pain in the neck" and invariably difficult, or even impossible, to treat.

References

Aikens, J.E., Zvolensky, M.J., & Eifert, G.H. (2001). Fear of cardiopulmonary sensations in emergency room noncardiac chest pain patients. *Journal of Behavioral Medicine*. *24*, 155–67.

Allen, L. A., Woolfolk, R. L., Escobar, J. I., Gara, M. A., & Hamer, R. M. (2006). Cognitive-behavioral therapy for somatization disorder: a randomized controlled trial. *Archives of Internal Medicine*, *166*(14), 1512.

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders*. (4th ed.). Washington, DC: American Psychiatric Association.

American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders.* (5th ed.). Washington, DC: American Psychiatric Association.

Barlow, D. H. (2002). *Anxiety and its disorders: The nature and treatment of anxiety and panic* (2nd. ed.). New York, NY: Guilford.

Barsky, A., Orav, E., & Bates, D. (2005). Somatization increases medical utilization and costs independent of psychiatric and medical comorbidity. *Archives of General Psychiatry*, *62*(8), 903–10.

Bass, C. M., & Benjamin, S. (1993). The management of the chronic somatizer. *British Journal of Psychiatry*, *162*, 472–80.

Bass, C. M., & Murphy, M. R. (1990). Somatization disorder: Critique of the concept and suggestions for future research. In C. M. Bass (Ed.) *Somatization. physical symptoms and psychological illness*. Oxford: Blackwell (pp. 301–32).

Bass, C. M., & Murphy, M. R. (1995). Somatoform and personality disorders: Syndromal comorbidity and overlapping developmental pathways. *Journal of Psychosomatic Research*, *39*, 403–27.

Chaturvedi, S., Desai, G., & Shaligram, D. (2006). Somatoform disorders, somatization and abnormal illness behavior. *International Review of Psychiatry*, *18*(1), 75–80.

Chawla, N. & Ostafin, B. (2007). Experiential avoidance as a functional dimensional approach to psychopathology: An empirical view. *Journal of Clinical Psychology*, *63*(9), 871–90.

Compas, B. E., Conner-Smith, J. K., Saltzman, H., Thomsen, A. H., & Wadsworth, M. E. (2001). Coping with stress during childhood and adolescent: Problems, progress, and potential in theory and research. *Psychological Bulletin*, *127*, 87–127.

De Gucht, V. (2002). Neuroticism, alexithymia, negative affect and positive affect as predictors of medically unexplained symptoms in primary care. *Acta Neuropsychiatrica*, *14*(4), 181–5.

Denissen, J. & Penke, L. (2008). Neuroticism predicts reactions to cues of social inclusion. *European Journal of Personality*, *22*, 497–517.

Eifert, G. H. (1992). Cardiophobia: A paradigmatic behavioral model of heart-focused anxiety and non-anginal chest pain. *Behaviour Research and Therapy*, *30*, 329–45.

Eifert, G. H. (1996). More theory-driven and less diagnosis-based behavior therapy. *Journal of Behavior Therapy and Experimental Psychiatry*, *27*, 75–86.

Eifert, G. H., & Forsyth, J. F. (2005). *Acceptance and commitment therapy for anxiety disorders*. Oakland, CA: New Harbinger Publications.

Eifert, G. H., Hodson, S. E., Tracey, D. R., Seville, J. L., & Gunawardane, K. (1996). Cardiac anxiety, illness beliefs, psychological impairment: Comparing healthy heart-anxious patients with cardiac and surgical patients. *Journal of Behavioral Medicine*, *19*, 385–99.

Eifert, G. H., & Lau, A. (2001). Using behavioral experiments in the treatment of cardiophobia: A case study. *Cognitive and Behavioral Practice*, *8*, 305–17.

Eifert, G. H., Lejuez, C. W., & Bouman, T. K. (1998). Somatoform disorders. In A. S. Bellack & M. Hersen (Eds.). *Comprehensive clinical psychology* (Vol. 6, pp. 543–65). Oxford: Pergamon.

Eifert, G. H., Zvolensky, M. J., & Lejuez, C. W. (2000). Heart-focused anxiety and chest pain: A conceptual and clinical review. *Clinical Psychology: Science and Practice*, *7*, 403–17.

Escobar, J. I., Allen, L. A., Nervi, C. H., & Gara, M. A. (2001). General and cross-cultural considerations in a medical setting for patients presenting with medically unexplained symptoms. In G. J. G. Asmundson, S. Taylor, & B. Cox (Eds.), *Health anxiety: Clinical research perspectives on hypochondriasis and related disorders* (pp. 220–45). New York, NY: Wiley.

Escobar, J. I., Gara, M. A., Diaz-Martinez, A. M., Interian, A., Warman, M., Allen, L. A., . . . Rodgers, D. (2007). Effectiveness of a time-limited cognitive behavior therapy-type intervention among primary care patients with medically unexplained symptoms. *Annals of Family Medicine*, *5*(4), 328–35.

Fink, P. (1996). Somatization—Beyond symptom count. *Journal of Psychosomatic Research*, *40*, 7–10.

First, M. B. (2011). Moving beyond unexplained medical symptoms in DSM-5: Great idea, problematic execution. *Psychosomatics*, *52*(6), 594–5. doi: 10.1016/j.psym.2011.05.002

Flor, F., Birbaumer, N., & Turk, D. C. (1990). The psychobiology of chronic pain. Advances in *Behaviour Research and Therapy*, *12*, 47–84.

Forsyth, J. P., Eifert, G. H., & Barrios, V. (2006). Fear conditioning research as a clinical analog: What makes fear learning disordered? In M. G. Craske, D. Hermans, & D. Vansteenwegen (Eds.). *Fear and learning: Basic science to clinical application* (pp. 133–53). Washington, DC: American Psychological Association.

Garcia-Campayo, J., Alda, M., Sobradiel, N., Olivan, B., & Pascual, A. (2007). Personality disorders in somatization disorder patients: A controlled study in Spain. *Journal of Psychosomatic Research*, *62*(6), 675–80.

Goldberg, D., Gask, L., & O'Dowd, T. (1989). The treatment of somatization: Teaching techniques of reattribution. *Journal of Psychosomatic Research*, *33*, 689–95.

Gureje, O. (2004). What can we learn from a cross-national study of somatic distress? *Journal of Psychosomatic Research*, *56*(4), 409–12.

Hansen, M., Fink, P., Frydenberg, M., & Oxhoj, M. (2002). Use of health services, mental illness, and self-rated disability and health in medical inpatients. *Psychosomatic Medicine*, *64*, 668–75.

Harth, W. & Hermes, B. (2007). Psychosomatic disturbances and cosmetic surgery. *Journal der Deutschen Dermatologischen Gesellschaft*, *5*(9), 736–43.

Hayes, S. C., Strosahl, K., & Wilson, K. (2000). *Acceptance and commitment therapy: Understanding and treating human suffering*. New York, NY: Guilford Press.

Hayes, S. C., & Wilson, K. G. (1994). Acceptance and commitment therapy: Altering the verbal support for experiential avoidance. *Behavior Analyst*, *17*, 289–303.

Heinrich, T. (2004). Medically unexplained symptoms and the concept of somatization. *Wisconsin Medical Journal*, *103*(6), 83–7.

Hoyer, J., Eifert, G. H., Einsle, F., Zimmermann, K., Krauss, S., Knaut, M., . . . Köllner, V. (2008). Heart-focused anxiety before and after cardiac surgery. *Journal of Psychosomatic Research*, *64*, 291–7.

Kagan, J. (1989). Temperamental contributions to social behavior. *American Psychologist*, *44*, 668–74.

Kantor-Martynuska, J. (2009) The listener's temperament and perceived tempo and loudness of music. *European Journal of Personality*, *23*, 655–73.

Kashner, T. M., Rost, K., Cohen, B., Anderson, M., & Smith. G. R. (1995). Enhancing the health of somatization disorder patients. Effectiveness of short-term group therapy. *Psychosomatics*, *36*, 462–70.

Katon, W., Sullivan, M., & Walker, E. (2001). Medical symptoms without identified pathology: Relationship to psychiatric disorders, childhood and adult trauma, and personality traits. *Annals of Internal Medicine*, *134*(9.2), 917–25.

Kendler, K.S., Walters, E. E., Truett, K. R., & Heath, A. C. (1995). A twin-family study of self- report symptoms of panic, phobia, and somatization. *Behavior Genetics*, *25*, 499–515.

Kerns, R., Thorn, B., & Dixon, K. (2006). Psychological treatments for persistent pain: An introduction. *Journal of Clinical Psychology*, *62*(11), 1327–31.

Kerr, D. C., Capaldi, D. M., Pears, K. C., & Owen, L. D. (2012). Intergenerational influences on early alcohol use: independence from the problem behavior pathway. *Developmental Psychopathology*, *24*(3), 889–906. doi: 10.1017/S0954579412000430

Khan, A. A., Khan, A., & Kroenke, K. (2000). Symptoms in primary care: Etiology and outcome. *Journal of General Internal Medicine*, *15*, 76–7.

Kirmayer, L. & Sartorious, N. (2009). Cultural models and somatic syndromes. In J. Dimsdale, Y. Xin, A. Kleinman, V. Patel, W. Narrow, P. Sirovatka, & D. Regier (Eds.), *Somatic Presentations of Mental Disorders: Refining the Research Agenda for DSM-V* (pp. 19–40). Arlington, VA: American Psychiatric Association.

Kroenke, K. (2007). Efficacy of treatment for somatoform disorders: a review of randomized controlled trials. *Psychosomatic Medicine*, *69*(9), 881–8.

Kroenke, K. & Price, R. (1993). Symptoms in the community: Prevalence, classification, and psychiatric comorbidity. *Archives of Internal Medicine*, *153*, 2474–80.

Lau, J., Ely, T., & Stevenson, J. (2006). Examining the state–trait anxiety relationship: A behavioural genetic approach. *Journal of Abnormal Child Psychology*, *34(1)*, 19–27.

Leiknes, K., Finset, A., Moum, T., & Sandanger, I. (2008). Overlap, comorbidity, and stability of somatoform disorders and the use of current versus lifetime criteria. *Psychosomatics*, *49*(2), 152–62.

Lejuez, C. W., Hopko, D. R., & Hopko, S. D. (2001). A brief behavioral activation treatment for depression. *Behavior Modification*, *25*, 255–86.

Letcher, P., Smart, D., Sanson, A., & Toumbourou, J. (2009). Psychosocial precursors and correlates of differing internalizing trajectories from 3 to 15 years. *Social Development*, *18*(3), 618–46.

Lipsitt, D.R., & Starcevic, V. (2006). Psychotherapy and pharmacotherapy in the treatment of somatoform disorders. *Psychiatric Annals*, *36*, 341–50.

Löwe, B., Mundt, C., Herzog, W., Brunner, R., Backenstrass, M., Kronmüller, K., & Henningsen, P. (2008). Validity of current somatoform disorder diagnoses: Perspectives for classification in DSM-V and ICD-11. *Psychopathology*, *41*, 4–9.

Luoma, J., Hayes, S., & Walser, R. (2007). *Learning ACT: An acceptance and commitment therapy skills-training manual for therapists*. Oakland, CA: New Harbinger Publications.

Marple, R., Kroenke, K., Lucey, C., Wilder, J., & Lucas, C. (1997). Concerns and expectations in patients presenting with physical complaints: Frequency, physician perceptions and actions, and two-week outcomes. *Archives of Internal Medicine*, *157*, 1482–8.

Mayou, R. (2009). Are treatments for common mental disorders also effective for functional symptoms and disorders? In J. Dimsdale, Y. Xin, A. Kleinman, V. Patel, W. Narrow, P. Sirovatka, & D. Regier (Eds.), *Somatic Presentations of Mental Disorders: Refining the Research Agenda for DSM-V* (pp. 131–42). Arlington, VA: American Psychiatric Association.

Mayou, R., Bass, C. M., & Sharpe, M. (Eds.) (1995). *Treatment of functional somatic symptoms*. Oxford: Oxford University Press.

Mayou, R., Kirmayer, L., Simon, G., & Sharpe, M. (2005). Somatoform disorders: Time for a new approach in DSM-V. *American Journal of Psychiatry*, *162*, 847–55.

McKay, M., Forsyth, J. P., & Eifert, G. H. (2010). *Your life on purpose: How to find what matters and create the life you want*. Oakland, CA: New Harbinger.

Mineka, S., Gunnar, M., & Champoux, M. (1986). Control and early socioemotional development: Infant rhesus monkeys reared in controllable versus uncontrollable environments. *Child Development*, *57*, 1241–56.

Moene, F. C., Spinhoven, P., Hoogduin, K. A., & Dyck, R. V. (2003). A randomized controlled clinical trial of a hypnosis-based treatment for patients with conversion disorder, motor type. *International Journal of Clinical and Experimental Hypnosis*, *51*(1), 29–50.

Narrow, W., Rae, D., Robins, L., & Regier, D. (2002). Revised prevalence estimates of mental disorders in the United States: Using a clinical significance criterion to reconcile 2 surveys' estimates. *Archives of General Psychiatry*, *59*, 115–23.

NASA Scientific and Technical Information Program Office. (2004). *Stress, cognition, and human performance: Literature review and conceptual framework*. (Pub. No. 2004-212824). Hanover, MD: NASA Center for AeroSpace Information.

Patrick, M. E., Wray-Lake, L., Finlay, A. K., & Maggs, J. L. (2010). The long arm of expectancies: adolescent alcohol expectancies predict adult alcohol use. *Alcohol & Alcoholism*, *45*(1), 17–24. doi: 10.1093/alcalc/agp066

Pilgrim, T. & Wyss, T. (2008). Takotsubo cardiomyopathy or transient left ventricular apical ballooning syndrome: A systematic review. *International Journal of Cardiology*, *124*(3), 283–92.

Pilowsky, I. (1993). Aspects of abnormal illness behaviour. *Psychotherapy and Psychosomatics*, *60*, 62–74.

Rief, W., Heitmüller, A. M., Reisberg, K., & Rüddel, H. (2006). Why reassurance fails in patients with unexplained symptoms—an experimental investigation of remembered probabilities. *PLoS medicine*, *3*(8), e269. doi: 10.1371/journal.pmed.0030269

Rief, W., Hiller, W., Geissner, E., & Fichter, M. M. (1995). A two-year follow-up study of patients with somatoform disorders. *Psychosomatics*, *36*, 376–86.

Roma, P., Champoux, M., & Suomi, S. (2006). Environmental control, social context, and individual differences in behavioral and cortisol responses to novelty in infant rhesus monkeys. *Child Development*, *77*(1), 118–31.

Rosebush, P. I., & Mazurek, M. F. (2011). Treatment of conversion disorder in the 21st century: have we moved beyond the couch? *Current Treatment Options in Neurology*, *13*(3), 255–66.

Rost, K., & Kashner, T. M., & Smith, G. R. (1995). Effectiveness of psychiatric intervention with somatization disorder patients. *General Hospital Psychiatry*, *16*, 381–7.

Rothbart, M. K. (1989). Temperament and development. In G. Kohnstamm, J. Bates, & M. Rothbart, M. K. (Eds.), *Temperament in childhood* (pp. 187–248). Chichester: Wiley.

Rothbart, M. K., Ziaie, H., & O'Boyle, C. G. (1992). Self-regulation and emotion in infancy. In N. Eisenberg & R. A. Fabes (Eds.), *Emotion and its regulation in early development: New directions for child development*, (pp. 7–24). San Francisco, CA: Jossey-Bass.

Rueda, M. & Rothbart, M. (2009). The influence of temperament on the development of coping: The role of maturation and experience. In E. Skinner & M. Zimmer-Gembeck (Eds.), *Coping and the development of regulation*. *New Directions for Child and Adolescent Development*, 124, pp. 19–31. San Francisco, CA: Jossey-Bass.

Ryder, A., Yang, J., Xiongzhao, Z., Shugiao, Y., Yi, J., Heine, J., & Bagby, R. (2008). The cultural shaping of depression: Somatic symptoms in China, psychological symptoms in North America? *Journal of Abnormal Psychology*, *117*(2), 300–13.

Salkovskis, P. M. (1996). The cognitive approach to anxiety: Threat beliefs, safety seeking behavior, and the special case of health anxiety and obsessions. In P.M. Salkovskis (Ed.) *Frontiers of cognitive therapy* (pp. 49–74). New York, NY: Guilford.

Salkovskis, P. M., & Warwick, H. M. C. (2001). Making sense of hypochondriasis: A cognitive model of health anxiety. In G. J. G. Asmundson, S. Taylor, & B. Cox (Eds.), *Health anxiety: Clinical research perspectives on hypochondriasis and related disorders* (pp. 46–64). New York, NY: Wiley.

Sar, V., Islam, S., & Öztürk, E. (2009). Childhood emotional abuse and dissociation in patients with conversion symptoms. *Psychiatry and Clinical Neuroscience*, *63*(5), 670–7.

Schermelleh-Engel, K., Eifert, G. H., Moosbrugger, H., & Frank, D. (1997). Perceived competence and anxiety as determinants of maladaptive and adaptive coping strategies of chronic pain patients. *Personality and Individual Differences*, *22*, 1–10.

Sharpe, M., & Bass, C. M. (1992). Pathophysiological mechanisms in somatization. *International Review of Psychiatry*, *4*, 81–97.

Sidman, M. (1994). *Equivalence relations and behavior: A research story*. Boston, MA: Authors Cooperative.

Smith, R. C., Lein, C., Collins, C., Lyles, J. S., Given, B., Dwamena, F. C., . . . Given, C. W. (2003). Treating patients with medically unexplained symptoms in primary care. *Journal of General Internal Medicine*, *18*(6), 478–89.

Smith, G. R., Rost, K., & Kashner, T. M. (1995). A trial of the effect of a standardized psychiatric consultation of health outcomes and cost in somatizing patients. *Archives of General Psychiatry*, *52*, 238–43.

Smoller, J., Block, S., & Young, M. (2009). Genetics of anxiety disorders: The complex road from DSM to DNA. *Depression and Anxiety*, *26*(11), 965–75.

Speckens, A. E. M., van Hemert, A. M., Spinhoven, P., Hawton, K. E., Bolk, J. H., & Rooijmans, H. G. M. (1995). Cognitive behavioral therapy for medically unexplained physical symptoms: A randomised controlled trial. *British Medical Journal*, *311*, 1328–32.

Speed, J. (1996). Behavioral management of conversion disorder: Retrospective study. *Archives of Physical Medicine and Rehabilitation*, *77*, 147–54.

Staats, A. W. & Eifert, G. H. (1990). A paradigmatic behaviorism theory of emotion: Basis for unification. *Clinical Psychology Review*, *10*, 539–66.

Stewart, S. H., Zvolensky, M. J., & Eifert, G. H. (2002). The relations of anxiety sensitivity, experiential avoidance, and alexithymic coping to young adults' motivations for drinking. *Behavior Modification*, *26*, 274–96.

Sykes, R. (2012). The DSM 5 website proposals for somatic symptom disorder: three central problems. *Psychosomatics*, *53*(6), 524–31. doi: 10.1016/j.psym.2012.06.004

van der Bruggen, C., Stams, G., Bögels, S., and Paulussen-Hoogeboom, M. (2010). Parenting behaviour as a mediator between young children's negative emotionality and their anxiety/depression. *Infant and Child Development*, *19*(4), 354–65.

van der Feltz-Cornelis, C. M., & van Balkom, A. J. (2010). The concept of comorbidity in somatoform disorder—a DSM-V alternative for the DSM-IV classification of somatoform disorder. *Journal of Psychosomatic Research*, *68*(1), 97–9; author reply 99–100. doi: 10.1016/j.jpsychores.2009.09.011

Vedsted, P., Fink, P., Sørensen, H., & Olesen, F. (2004). Physical, mental and social factors associated with frequent attendance in Danish general practice. A population-based cross-sectional study. *Social Science Medicine*, *59*(4), 813–23.

Vijayasiri, G., Richman, J. A., & Rospenda, K. M. (2012). The Great Recession, somatic symptomatology and alcohol use and abuse. *Addict Behav*, *37*(9), 1019–24. doi: 10.1016/j.addbeh.2012.04.007

Vowles, K. E., Wetherell, J. L., & Sorrell, J. T. (2009). Targeting acceptance, mindfulness, and values-based action in chronic pain: Findings of two preliminary trials of an outpatient group-based intervention. *Cognitive and Behavioral Practice*, *16*, 49–58.

Wainwright, D., Calnan, M., O'Neil, C., Winterbottom, A., & Watkins, C. (2006). When pain in the arm is 'all in the head': The management of medically unexplained suffering in primary care. *Health, Risk & Society*, *8*(1), 71–88.

Walker, J. & Furer, P. (2008). Interoceptive exposure in the treatment of health anxiety and hypochondriasis. *Journal of Cognitive Psychotherapy*, *22*(4), 366–78.

Warwick, H. M. C., & Salkovskis, P. M. (1990). Hypochondriasis. *Behaviour Research and Therapy*, *28*, 105–17.

Yilmaz, A., Mahrholdt, H., Athanasiadis, A., Vogelsberg, H., Meinhardt, G., Voehringer, M., . . . Sechtem, U. (2008). Coronary vasospasm as the underlying cause for chest pain in patients with pvb19 myocarditis. *Heart*, *94*(11), 1456–63.

Zvolensky, M. J., Eifert, G. H., Lejuez, C. W., & McNeil, D. W. (1999). The effects of offset control over 20% carbon dioxide-enriched air on anxious responding. *Journal of Abnormal Psychology*, *108*, 624–32.

Zvolensky, M. J., Feldner, M. T., Eifert, G. H., Vujanovic, A. A., & Solomon, S. E. (2008). Cardiophobia: A critical analysis. *Transcultural Psychiatry*, *45*, 231–52.

16

Dissociative Disorders

Steven Jay Lynn, Scott O. Lilienfeld, Harald Merckelbach, Reed Maxwell, Jessica Baltman, and Timo Giesbrecht

From Jekyll and Hyde like shifts of feelings and behaviors to feelings of unreality, like "all the world's a stage" for actors seemingly playing a part, to amnesia for events that one should, by all rights remember, the symptoms of dissociative disorders have proven to be as fascinating as they are perplexing and controversial. Janet (1973) was perhaps the first to claim, in 1889, that dissociation (or "desagregation" as he termed it) originated in a defensive response to traumatic events and to appreciate the importance of studying dissociation in order to comprehend the full range of everyday and anomalous experiences (Cardeña, Lynn, & Krippner, 2014). Janet's contention that dissociation represents a coping strategy in response to highly aversive events continues to provoke vigorous debate, just as the notion of multiple personality disorder (now termed dissociative identity disorder) sparked Freud's skepticism in Janet's time. In this chapter, we examine the three major dissociative disorders—dissociation/derealization disorder, dissociative amnesia, and dissociative identity disorder, in turn. More specifically, we describe their symptoms, prevalence, and assessment, as well as current controversies regarding the genesis of dissociation and competing theories of their nature and origin and efforts to treat their vexing symptoms.

Dissociation and Dissociative Disorders

As described in the DSM-5 (American Psychiatric Association, 2013), dissociation can be defined as a "disruption of and/or discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control, and behavior" (p. 291). Dissociative experiences range from the mundane, such as occasional lapses or divisions in attention and memory, to fantasizing, daydreaming, becoming absorbed in movies and what is commonly referred to as "highway hypnosis" ("losing" lengthy periods of time while driving), to the profound and sometimes unpredictable shifts in consciousness that mark dissociative disorders. In some cases, it is difficult to distinguish pathological from nonpathological dissociation (Giesbrecht, Lynn, Lilienfeld, & Merckelbach, 2008; Modestin & Erni, 2004; Waller, Putnam, & Carlson, 1996; Waller & Ross, 1997), and it is not altogether clear whether milder manifestations of dissociation share biological and etiological roots with more dysfunctional manifestations of dissociation (see Cardena, 1994; Lynn et al., 2014).

Although controversy persists regarding the origins of both mild and more pathological dissociation, there is little dispute that some individuals present with symptoms that fall under the rubric of "dissociation," as codified in DSM-5. In brief, the three major disorders in DSM-5 are:

1. *Depersonalization/derealization disorder* (with depersonalization being experiences of unreality, detachment, outside observer of one's thoughts, feelings, sensations or actions, and derealization being experiences of unreality or detachment with respect to surroundings);
2. *Dissociative amnesia*, the inability to recall important autobiographical information, usually of a traumatic or stressful nature, inconsistent with ordinary forgetting; and
3. *Dissociative identity disorder* (DID; formerly called multiple personality disorder), a striking disruption of identity characterized by two or more distinct personality states and recurrent gaps in the recall of everyday events.

To merit a diagnosis of a dissociative disorder, dissociative experiences or symptoms must (a) interfere with important areas of functioning, such as work and social relationships, (b) engender subjective distress, or both. Spiegel and colleagues (2011) classified pathological dissociative symptoms as "positive" or "negative," with positive symptoms comprising flashbacks and intrusions of an "aspect of identity" into awareness, and negative symptoms comprising "deficits in memory, sense of self, and/or the ability to sense or control different parts of the body" (p. 826).

Some researchers have suggested that a dissociative subtype of schizophrenia can be identified with prominent dissociative features, including amnesia and depersonalization/derealization (see Ross, 2008; Vogel, Braungardt, Grabe, Schneider, & Klauer, 2013). However, it is not clear to what extent dissociative symptoms merely reflect nonspecific symptoms associated with substantial comorbidity due to a psychotic disorder (Laferrière-Simard, Lecomte, & Ahoundova, 2014), or whether a dissociative subtype of schizophrenia will emerge as a valid entity pending future research.

Prevalence of Pathological Dissociation: Nonclinical and Clinical Samples Studies of the prevalence of people in nonclinical populations who report clinically significant symptoms of dissociation using well-validated measures of dissociation or structured interviews provide variable estimates of often non-trivial rates of dissociation. More specifically, the rates of pathological dissociation generally range from 0.3% (Spitzer, Barnow, Freyberger, & Grabe, 2006) to 2–3% in the general population (Seedat et al., 2003; Vanderlinden, Van Dyck, Vandereycken, & Vertommen, 1991; Waller & Ross, 1997), although Sar, Akyüz, and Dogan (2007) reported an "outlier" estimate of 18.3% lifetime prevalence of dissociative disorders among Turkish women sampled in the community, a rate perhaps attributable to unknown cultural factors.

Waller and Ross (1997) conducted an analysis of pathological dissociation in the general population in two North American samples and used biometric and taxonomic statistical procedures to determine that approximately 3.3% of the population belongs to a pathological dissociative taxon, as measured by an 8-item scale (DES-T) derived from the Dissociative Experience Scale (DES; Bernstein-Carlson & Putnam, 1986). In psychopathology, a taxon is a natural class; that is, a group that differs in kind rather than in degree from normality. Nevertheless, Watson (2003) found the taxon scores to be only modestly stable (test re-test correlation $r = 0.34$ after approximately 2 months), and most individuals identified as taxon members on one occasion failed to be so classified at re-test. Generally speaking, little is known about the test–re-test reliability of diagnoses of dissociative disorders and the impact of potential low reliability on prevalence estimates.

Few studies have examined the rates of dissociative disorders in college students. Sandberg and Lynn (1992) identified 33 female college students who scored in the upper 15% on the DES, a widely used self-report measure of dissociation (Bernstein-Carlson & Putnam, 1986). Two of these students met criteria for a dissociative disorder (i.e., psychogenic amnesia, or what would today be called dissociative amnesia; multiple personality disorder, or what would today be called dissociative identity disorder), although none of the 33 students who scored below the mean on the DES met criteria for a dissociative disorder. Eight participants who scored in the upper 2% on the DES did not meet criteria for a dissociative disorder.

The prevalence of dissociation in clinical populations tends to be much higher than in nonclinical populations, although significant variability is also evident in the former samples, ranging from lows of 12–17% (Lipsanen et al., 2004; Sar, Tutkun, Alyanak, Bakim, & Barai, 2000; Saxe et al., 1993) to higher estimates of lifetime prevalence of 28–40.9% in an inpatient setting (Ross, Duffy, & Ellason, 2002) to the highest estimate 34.9% in a psychiatric emergency facility (Sar, Akyüz, & Dogan, 2007). Prevalence rates of dissociative disorders also run high in special populations, with rates reported of all dissociative disorders of 80% (35% diagnosed with DID) in exotic dancers and 55% in prostitutes (Ross, Anderson, Heber, & Norton, 1990), and 17–39% in individuals with substance use disorders (for a review, see Sar, 2011). Overall, the findings regarding gender differences in dissociative diagnoses are mixed, with some studies finding a higher prevalence among women, and other studies finding no differences (Sar, 2011).

Suicide and self-mutilatory behaviors tend to be higher (completed suicide 1–2%) among patients with dissociative disorder, although the interpretation of such elevated rates is often complicated by substantial "comorbidity" (co-occurrence) of dissociative disorders with borderline personality disorder and other serious manifestations of psychopathology, including other personality disorders, anxiety disorders, and depressive disorders (Johnson, Cohen, Kasen, & Brook, 2006; Lynn et al., 2014). Sar Akyüz, Öztürk, and Alioğlu (2013) sampled 628 women in a Turkish community and identified "dissociative depression" among 40% of depressed women, a condition marked by symptoms of depression, increased suicidality and self-mutilation, and reports of childhood trauma. Most of the women did not meet diagnostic criteria for borderline personality or posttraumatic stress disorder (PTSD).

Reasons for the disparities of prevalence rates among studies are not clear, although such rates almost certainly vary as a function of the assessment instrument used, diagnostic base rates at different facilities, the presence and nature of comorbid symptoms, and examiner beliefs and biases regarding dissociation. Generally speaking, experimenter blindness regarding patient characteristics lowers the rate of diagnoses of dissociative disorders (Friedl, Draijer, & de Jonge, 2000).

Genetics and Dissociation Research supports a genetic substrate for the propensity toward dissociation, although

the evidence is not consistent. Jang, Paris, Zweig-Frank, and Livesley (1998) reported that 48% of the variability in DES-T scores is attributable to genetic influences,, and that the remainder of the variance (52%) can be attributed to nonshared environments; that is, environmental experiences that make members of the same family different from one another. When the researchers excluded taxon items and considered nonpathological dissociation scores, genetic influences accounted for 55% of the variance, whereas nonshared environmental influences accounted for 45% of the variance. In a study of children and adolescents, Becker-Blease and colleagues (2004) found a substantial genetic (59% genes, 41% nonshared environment) contribution to dissociation scores, although Waller and Ross (1997) found no evidence for genetic influences based on their research with 280 identical twins and 148 fraternal twins. Unfortunately, no genetic studies of diagnosed patients with dissociative disorders have been conducted. Twin and adoption studies would help to clarify the extent to which the familial clustering of dissociative disorders is due to genes, shared environment (environmental factors that boost family resemblance), or both.

Physiological and Neuroimaging Findings Research has not produced definitive conclusions regarding (a) the relation between dissociation and cortisol response, a commonly used measure of stress reactivity, and (b) the relations among dissociation, cardiovascular (heart rate), and autonomic (e.g., skin conductance response) reactions in response to trauma reminders (Dalenberg et al., 2012). Similar inconsistencies are present in neuroimaging studies in which "either increased or decreased response in medial prefrontal cortex and limbic regions accompanying dissociative symptoms" has been observed (p. 579). Moreover, individuals who score highly on measures of dissociation usually fall within the normative range on standard neuropsychological tests and tests of intellectual ability (Giesbrecht et al., 2008; Schurle, Ray, Bruce, Arnett, & Carlson, 2007). Although trait dissociation, in contrast to PTSD, is associated with increases in gray matter volume in prefrontal, paralimbic, and parietal cortices, as determined by magnetic resonance imaging (MRI), the meaning of these findings is unclear (Nardo et al., 2013). In many studies in which differences in physiological responses vary as a function of dissociation, researchers have neglected to control for general distress and openness to experience, which are both moderately associated with dissociation, making it difficult to interpret the findings (Lynn et al., 2014). Nevertheless, as our discussion proceeds, we see that different dissociative disorders are related to different cognitive deficits and physiological responses.

Depersonalization/Derealization Disorder

We begin our discussion of the three major dissociative disorders with depersonalization/derealization disorder (hereafter referred to as DPD), the most common and least controversial of the dissociative disorders. Unlike earlier versions of the DSM, in which depersonalization and derealization were catalogued as separate disorders, DSM-5 groups depersonalization and derealization experiences together as DPD. The conjoining of depersonalization and derealization appears to be warranted, as they share similar features with respect to demographics, comorbidity with other disorders, and course (Simeon, 2009).

Description Ludovic Dugas is often credited with first using the term "depersonalization" in the late 1890s (Simeon & Aubugel, 2006). The hallmark of depersonalization is an altered sense of selfhood. Depersonalization symptoms are varied and span feeling as though one is outside of or observing one's body, feelings, sensations, and actions; emotional or physical numbing; time distortion; and an absent or unreal sense of self. In contrast, derealization pertains to feelings of unreality with regard to one's surroundings, in which objects or people may appear to be visually distorted or experienced as foggy, dreamlike, robotic, or lifeless. Understandably, such strange, unusual, and often unexpected experiences of depersonalization/derealization can evoke considerable anxiety, even to the extent of fearing losing control or becoming psychotic. Simeon and colleagues (2008) conducted a factor analysis which revealed that depersonalization could be described in terms of five symptom dimensions: Numbing, unreality of self, perceptual alterations, temporal disintegration, and unreality of surroundings, providing further support for combining depersonalization and derealization symptoms. Individuals with DPD may experience not only alexithymia (i.e., difficulty identifying feelings; Simeon, Giesbrecht, Knutelska, Smith, R. J., & Smith, L. M., 2009), but also feelings of unreality produced by difficulties in discriminating emotional from neutral stimuli (Quadflieg et al. 2013) and a dampening, numbing, or shutting out of emotional responses (Medford, 2012).

Diagnosis The onset of DPD varies from gradual to sudden and from chronic to episodic, and may last from hours to weeks or months and extend to years or even decades in rare cases (Simeon, 2009). Freud described experiencing transient depersonalization/derealization during a visit to the Athens Acropolis in 1904, which he found difficult to experience as real (Simeon & Aubugel, p. 11). Freud's experience is not atypical: Episodes of depersonalization/derealization are commonly brief and may occur only a few times in a person's lifetime. Estimates suggest that the annual prevalence of transient depersonalization/derealization is 23% (Aderibigbe, Bloch, & Waler, 2001), with a lifetime prevalence between 26% and 74% (Hunter, Sierra, & David, 2004).

DPD often occurs in the context of highly stressful events, unfamiliar environments, threatening social interactions, or the use of drugs such as marijuana, ketamine,

and other hallucinogens. DPD has been associated with childhood parental rejection and punishment, controlling for age and severity of depression and anxiety (Michal et al., 2009), and with emotional abuse in childhood in clinical and nonclinical samples (Michal et al., 2009; Simeon, Guralnik, Schmeidler, Sirof, & Knutelska, 2001).

However, when DPD symptoms are persistent, recurrent (at least once a month), disturbing, and/or interfere with daily functioning, and do not occur exclusively in the context of another disorder, such as an anxiety disorder or a medical condition (e.g., seizures), they qualify for a diagnosis of DPD. The first episode of depersonalization/derealization is likely to occur between adolescence (age 16; Simeon et al., 1997) and young adulthood (age 22; Baker, Hunter, & Lawrence, 2003), although treatment may not be sought until late adulthood. In approximately two-thirds of cases, the course of DPD is chronic, with symptoms present most or all of the time (Simeon, 2009). To rule out a diagnosis of schizophrenia or another psychotic condition, for a diagnosis of DPD, reality testing must remain intact during episodes of depersonalization/derealization. Moreover, DPD should not be diagnosed when symptoms are restricted to meditative or trance practices.

Prevalence and Comorbidity Estimates of the prevalence of diagnosed DPD in the general population converge in the range of 1–3% (Lynn et al., 2014). In inpatient samples, the rates of depersonalization and derealization diagnoses run as high as 16% (Hunter et al., 2004).

DPD often occurs in the presence of other disorders. For example, DPD is especially common in panic disorder, with prevalence as high as 82%. The relation between DPD and anxiety has compelled some European authors to classify DPD as an anxiety disorder (Lynn, Merckelbach et al., 2015). Moreover, depersonalization/derealization is included among the diagnostic criteria for panic disorder in DSM-5. Nevertheless, the lumping of anxiety and depersonalization/derealization has been questioned. Although one study found that the only childhood risk factor identified for adult DPD is a history of anxiety (Lee, Kwok, Hunter, Richards, & David, 2012), another found that the correlation between measures of anxiety and depersonalization in adulthood is statistically significant but low (Sierra, Medford, Wyatt, & David, 2012). Moreover, mood-stabilizing medications typically used to treat anxiety conditions have little therapeutic effect on DPD (Simeon, 2009). Additionally, DPD occurs commonly in many conditions, not identified solely with anxiety, including major depression, somatoform disorders, substance use disorders, and various personality disorders (e.g., borderline, avoidant; Belli, Ural, Vardar, Yesilyurt, & Oncu, 2012; Lynn et al., 2014). For example, the rate of depersonalization/derealization symptoms in major depression has been reported to be as high as 60% (Noyes, Hoenk, Kuperman, & Slymen, 1977). Finally, although hyperarousal may trigger episodes of depersonalization/derealization (Sterlini & Bryant, 2002), depersonalization/derealization itself may engender anxiety, as mentioned earlier, and hyperventilation may bring about symptoms of both anxiety and depersonalization/derealization (Lickel, Nelson, Hayes, Lickel, & Deacon, 2008). In sum, anxiety and depersonalization appear to be related to some extent, but are largely distinguishable (Sierra et al., 2012).

In acute stress disorder, which often precedes PTSD, depersonalization/derealization is one of 14 symptoms listed in DSM-5, and is present in as many as 30% of cases of PTSD (Michal et al., 2009). Moreover, in DSM-5, it is possible to specify whether depersonalization and derealization accompany PTSD (American Psychiatric Association, p. 274). The dissociative subtype of PTSD can be identified in civilian and noncivilian populations (Lanius et al., 2012), with the prevalence of the subtype ranging from 14% (Stein et al., 2013) to 25% (Steuwe, Lanius, & Frewen, 2012), to 30% (females; Wolf et al., 2012). The fact that dissociative symptoms follow in the wake of trauma in only a minority of cases implies that exposure to highly aversive events is but one of a number of potential variables associated with dissociation, a contention that figures in our discussion below of theories of dissociation.

Physiological and Neuroimaging Findings Although trauma exposure may be a distal cause of depersonalization, researchers have implicated a mismatch in perceptual/sensory signals and alterations in body schemas as culprits in engendering DPD. For example, investigators (Simeon et al., 2009) have used MRI and positron emission tomography of the brain to reveal that DPD patients, compared with healthy controls, exhibit abnormalities in areas of the sensory cortex related to somatosensory, visual, and auditory experiences and areas of the cortex that subserve the integration of body schemas. Simeon et al. (2009) suggested that these findings imply that DPD involves a dissociation of perceptions that give rise to the symptoms of DPD. Additionally, vestibular stimulation produced by caloric irrigation of the ear labyrinths produces feelings of depersonalization in healthy participants, including strange bodily feelings, feeling "spaced out," and not in control of the self (Jáuregui-Renaud, Green, Bronstein, & Gresty, 2006). Moreover, patients experiencing peripheral vestibular disease (Jáuregui-Renaud, Sang, Gresty, Green, & Bronstein, 2008) and patients with retinal disease (Jáuregi-Renaud, Ramos-Toledo, Bolaños, Montaño-Velazquez, & Pliego-Maldonado, 2008) are more likely to experience depersonalization/derealization compared with healthy controls. Accordingly, DPD symptoms may ensue when there is a lack of integration or mismatch between multisensory inputs (e.g., vestibular, visual proprioceptive) that impairs neural representations that generate an altered sense of reality and the self

(Aspell & Blanke, 2009; Lynn et al., 2014). Alternatively, among predisposed individuals, DPD symptoms may tend to arise whenever there are markedly unexpected bodily or perceptual experiences.

Additional evidence of a link between DPD symptoms and physiology comes from studies of out-of-body experiences—a sense of physical separation from the self—sometimes reported by people with DPD. Researchers have shown that out-of-body experiences stem from a mixing or scrambling of the senses (e.g., vision and touch) when the sense of the physical body is disrupted. When physical sensations and visual impressions combine in atypical ways and there is a disruption in somatosensory signals, it can create the experience of feeling outside of one's body (Cheyne & Girard, 2009; Terhune, 2009). Ehrrson (2007) generated an out-of-body experience in the laboratory by creating the illusion that participants' bodies were standing in front of them. This was accomplished by participants donning goggles that displayed a video image of themselves provided by a camera behind them. Participants reported that they could experience themselves being touched in a location outside their physical bodies when they were touched with a rod on the chest at the same time the experimenters used the camera set-up to make it appear that their visual image was touched (see also Aspell, Lenggenhager, & Blanke, 2009; Lenggenhager et al., 2007). Additionally, investigators have produced out-of-body-like experiences by stimulating where the brain's parietal and right temporal lobes join, the vestibular cortex, and the superior temporal gyrus (see Lynn et al., 2014). Perhaps not coincidentally, Sierra, and colleagues (2014) compared DPD patients with controls and reported gray matter changes in the frontal, temporal, and parietal lobes associated with DPD based on MRI findings. The authors note that additional research is necessary to determine whether these changes are vulnerability or disease markers.

Transcranial magnetic stimulation (TMS)—a method of inducing an electrical field in select portions of the cortex by passing a magnetic field through the skull (Barker, Jalinous, & Freeston, 1985; O'Shea & Walsh, 2007)—has shown promise in elucidating pathological variations in cortical excitability associated with depersonalization (e.g., Sierra & Berrios, 1998). Jay, Sierra, Van den Eynde, Rothwell, and David (2014) used repetitive TMS (rTMS) to evaluate a neurobiological model of depersonalization proposed by Sierra and Berrios (1998) in which the ventrolateral prefrontal cortex (vlPFC) inhibition of the insula (a brain area involved in the processing of bodily sensations) contributes to the emotional numbing and altered sense of self associated with depersonalization. Jay and colleagues hypothesized that inhibition of the vlPFC engendered by rTMS would disinhibit insula activity and would allow for increased arousal and reduced depersonalization symptoms. Among patients with medication-resistant depersonalization disorder, a single session of rTMS produced reductions in depersonalization symptoms. However, although rTMS inhibited vlPFC activity and increased insula activity as hypothesized, symptom reductions also occurred after patients received rTMS targeting the temporal parietal junction, a known neural substrate of out-of-body experiences. The authors concluded that their findings support the neurobiological model proposed by Sierra and Berrios (2000). Nevertheless, they observed that, in their uncontrolled trial, the therapeutic effects of rTMS were independent of increased arousal (i.e., insula activity) and thus nonspecific and potentially attributable to placebo effects.

Other investigations of the effects of TMS and rTMS on depersonalization symptoms include two case studies and one clinical trial. Keenan, Freund, and Pascual-Leone (1999) treated a female patient with comorbid major depression and depersonalization disorder with rTMS and reported decreases in depersonalization symptoms in tandem with increases in self-awareness. In this case, rTMS targeted the patient's right frontal lobe, which showed hyperactivity in a single PET scan. A second case study (Jiménez-Genchi, 2004) of a male patient with comorbid medication-resistant DPD and major depressive disorder showed a 28% reduction in depersonalization symptoms after six sessions of rTMS delivered to the dorsolateral prefrontal cortex. Finally, Mantovani and colleagues (2011) conducted the first uncontrolled open clinical trial examining effects of inhibitory rTMS delivered to the temporal parietal junction in patients with depersonalization disorder. The authors observed that 6 of 12 patients demonstrated symptom improvement after 3 weeks, and 5 of the 6 responders showed 68% improvement in DPD symptoms after an additional 3 weeks of treatment (see also Christopeit, Simeon, & Mantovani, 2013). Randomized and placebo controlled trials are essential to further evaluate the specific and nonspecific effects of rTMS.

Dissociative Amnesia

As mentioned earlier, in DSM-5, dissociative amnesia (formerly called psychogenic amnesia) is diagnosed when there is substantial memory loss for important autobiographical information that is not the product of a neurological or other medical condition (e.g., seizures, memory loss associated with age, or brain injury) or substance abuse. Forgetting can pertain to everyday circumstances and is not limited to amnesia for traumatic or highly stressful events. Moreover, the symptoms of dissociative amnesia cannot be attributable to another disorder, such as acute stress disorder or PTSD, somatic symptom disorder, a neurocognitive disorder, or DID. It is believed to frequently follow a traumatic event and may be classified in terms of localized amnesia related to a specific time period (e.g., a vacation), selective amnesia for some but not all events from a specific time period, generalized amnesia for all life events, continuous amnesia for new events, and systematized amnesia for specific categories

of information, such as childhood sexual abuse. Based on an analysis of 42 memory and amnesia items from the Multidimensional Inventory of Dissociation, Dell (2013) identified three amnesia factors: discovering dissociated actions, lapses of recent memory and skills, and gaps in remote memory.

Despite failure to recall important events, people with dissociative amnesia typically retain implicit memories and habits related to these events, and some people with dissociative amnesia continue to experience a sense of familiarity with respect to previously known people, objects, and places. Moreover, in cases of dissociative amnesia, perception and immediate memory are preserved, although disorientation in time and impairment in new learning may occur (Staniloiu & Markowitsch, 2012).

Dissociative amnesia varies in terms of whether (a) it is experienced in childhood or adulthood (even young children may experience significant memory losses); (b) amnestic episodes last only minutes or persist for years and range from an isolated episode to recurrent episodes; and (c) significant or minimal functional impairment occurs before, during, or after the amnestic episode. Most cases of dissociative amnesia occur in the 30s and 40s, last between 1 and 5 days, and are equally common among females and males (American Psychiatric Association, 2013; Lynn et al., 2014). In two classic studies, in 63 cases, 94% (59/63) resolved within a week, and only 6% (4/59) of people took 3 weeks or more to recover (Ables & Childer, 1935; Herman, 1938). Because of differences in symptom presentation, age of onset, and functional impairment, it is likely that dissociative amnesia comprises a heterogeneous set of conditions with different etiologies.

Prevalence and Comorbidity The prevalence rates for dissociative amnesia range from 0.2% (China), to 3.0% (Canada), to 7.3% (Turkey), with unspecified cultural factors and differences in criteria for evaluating amnesia across studies probably playing a role in disparate diagnostic base-rates across samples (Dell, 2009; Lynn et al., 2014). Ross (2009) reported that among 3000 trauma patients he treated, only one exhibited dissociative amnesia. Still, individuals in the general population often report significant forgetting of events. In one study, 20.6% of college students reported some degree of impairment associated with one or more symptoms of dissociative amnesia based on their responses to a questionnaire (Sar, Alioğlu, Akyüz, & Karabulut, 2014). Researchers have reported that dissociative amnesia often occurs in response to multiple stressors and in the presence of mood, somatoform, eating, and personality disorders (Maldonado & Spiegel, 2008).

Controversy Dissociative amnesia has attracted considerable controversy, spurred in recent decades by Loftus's (1993) critique that questioned whether memories of traumatic events are repressed or dissociated from consciousness. Spiegel (1997) contended that dissociative amnesia is "more, rather than less common after repeated episodes; involves strong affect; and is resistant to retrieval through salient cues" (p. 6). Yet intense affect and repetition ordinarily improve memory, increasing access of memory through salient cues (McNally, 2003). Traumatic or stressful memories are often better remembered than less stressful memories (Berntsen & Rubin, 2014; Lynn et al., 2014; McNally, 2004), as is the case in PTSD, when patients experience intrusive and distressing memories (Lynn et al., 2014; Porter & Peace, 2007). Studies of concentration camp survivors, witnesses of homicide, and children who have been kidnapped indicate that memories of such highly aversive events are typically remembered vividly with little or no hint of amnesia (Merckelbach, Dekkers, Wessel, & Roefs, 2003). Memories of traumatic events may be better remembered for their salience, enhanced by attendant physiological arousal (e.g., Jelicic, Geraerts, Merckelbach, & Guerrieri, 2004).

Findings of facilitated recall for emotionally charged memories render implausible many claims of crime-related amnesia—reported in 25–40% of homicide cases and severe sex offenses—in which perpetrators claim they have little or no recall of crimes they committed (Moskowitz, 2004). Van Oorsouw and Merckelbach (2010) contend that malingering probably accounts for most claims of crime-related amnesia.

Although Brown, Scheflin, and Hammond (1998) argued that the empirical case for dissociative amnesia is compelling, Kihlstrom (2005) asserted that supportive studies lack methodological rigor and that longitudinal studies are both scant and do not rule out alternate explanations for amnesia, such as seizures and traumatic brain injury (see also Piper, Pope, & Borowiecki, 2000). Additionally, researchers have suggested that it is problematic to assume that the failure to report memories associated with highly aversive events, such as childhood abuse, is produced by dissociation and have asserted that studies of corroborated traumatic events have failed to uncover evidence that people "encode trauma, yet become incapable of recalling it through the mechanism of dissociative amnesia" (Lynn et al., 2014, p. 906; see also McNally, 2004; Pope, Oliva, & Hudson, 1999).

Specifically, the failure to report memories of events such as childhood sexual abuse may occur for reasons entirely unrelated to a special dissociative mechanism. For example, individuals may not have labeled the physical contact as "abuse" at the time it occurred, and not have thought about the event for some time, or they may presently be reluctant to disclose the abuse to a particular interviewer (Loftus, Polonsky, & Fulliove, 1994; McNally, 2004). Ordinary memory mechanisms, such as motivated forgetting or memory suppression, could account for failure to recall or report important autobiographical information, particularly when the information to be recalled or reported produces anxiety, shame,

or embarrassment. Indeed, feeling greater responsibility for the abuse, or fearing negative consequences of disclosure, rather than dissociative amnesia, provide a plausible account for the nondisclosure of some cases of childhood sexual abuse. Goodman and colleagues (2003) repeatedly interviewed 175 individuals with documented child sexual abuse approximately 13 years after the event. On initial interview, 18.9% of the participants did not report the event. Yet, by the third interview, which occurred in person, only 8% of the respondents failed to disclose the abuse, implicating reluctance to report, rather than a dissociative mechanism, for failure to report.

A fine line may divide active and passive or unconscious memory suppression, as exemplified by the case of FF, who, after a series of traumatic events (i.e., serious motorcycle accident and being the victim of a robbery and shooting) and earlier problems with his business and impending divorce "decided to get away from all of these problems by coming (from Germany) to the United States and starting anew" (p.1145), after which he experienced fugue symptoms. He allegedly lost access to his native German language and confided to researchers,

> I was aware of knowing German in written and spoken form . . . It was a part of my life I just wanted to lock away in a dark chamber. I can't even say if it was an active will or passive defense . . . The point where this disorientation was replaced by neglecting the truth is not easy to find and somehow undefined.
>
> (Glisky et al., 2004, p. 1146)

Pope, Poliakoff, Parker, and Boynes (2007) argued that dissociative amnesia is a culture-bound phenomenon and does not appear in literature until the late 18th century. Only one individual from among over 100 entrants in a contest was able to find an example of dissociative amnesia appearing in a fictional or nonfictional work before 1800, and thereby claim a $1,000 prize that Pope and colleagues offered for such evidence. The recency of reports of dissociative amnesia raises troubling questions about its existence as a natural category or entity, and suggests that social, cultural, and historical factors are implicated in reports of dissociative amnesia (Lynn et al., 2014).

Dissociative Fugue In DSM-IV-TR (American Psychiatric Association, 2000), dissociative fugue was classified as a separate disorder, but in DSM-5 it is coded as a condition that can accompany dissociative amnesia. Dissociative fugue (formerly called psychogenic fugue, and based on the Latin word for flight, which has the same root as the English word "fugitive") is a puzzling, rare (0.2% of the general population; Sadock & Sadock, 2005), and reversible amnesia for personal identity that involves wandering or unplanned travel. Some individuals with fugue assume a new identity in a new location, and in so doing, may travel thousands of miles and cross national boundaries (Santos & Gago, 2010). Still, in most cases, fugue is limited in duration to days or weeks and to local travel. Cases of fugue are often associated with highly aversive events, including natural disasters and war, and with avoidance of other stressful life events, including legal, financial, and marital problems.

The association of fugue with stressful events, physical escape from anxiety-eliciting surroundings, the assumption of a "new identity," and the fact that serious psychopathology is rarely manifested in the new identity, point to the purposeful, goal-directed nature of fugue-related behaviors and the possibility of malingering and factitious disorder. Clearly, these possibilities are essential to rule out in arriving at the conclusion that fugue is present. Moreover, it is important for clinicians to evaluate the hypothesis that fugue-like behaviors arise from brain injury and other medical complications, as fugue-like behaviors have been related to prednisone use (Gifford, Murawski, Kline, & Sachar, 1976), limbic system dysfunction (Mohan, Salo & Nagaswami, 1975), cervical plexus block (Goldberg, 1995), and changes in frontal, temporal, and parietal brain activity (Hennig-Fast et al., 2008).

Fugue, like dissociative amnesia, has stirred considerable controversy and skepticism. Hacking (1998), for example, described fugue as a phenomenon that varies with historical and cultural circumstances, given that it first appeared in the 19th century. Across cultures, fugue-like symptoms, including running, fleeing, energized activity, and amnesia for the duration of the episode are manifested in such culture-bound syndromes as amok in Western Pacific cultures (the source of the popular term "running amok"); *pibloktok* in native cultures in the Arctic, and Navajo "frenzy" witchcraft (Lynn et al., 2014).

Dissociative Identity Disorder

DID is arguably the most fascinating and controversial of the dissociative disorders. The diagnostic criteria for DID have changed considerably over the years, reflecting changes in the conceptualization of dissociative symptoms. The term "multiple personality" first surfaced in DSM-II (American Psychiatric Association, 1968), listed under the diagnosis hysterical neurosis, dissociative type. In DSM-III (American Psychiatric Association, 1980), the diagnosis fell under the rubric of "dissociative disorders" and retained the term multiple personality. In DSM-IV (American Psychiatric Association, 1994), the diagnosis was renamed to "dissociative identity disorder" to emphasize changes in identity and to deemphasize the notion that individuals with DID house separate personalities. In DSM-5, dissociative amnesia was broadened to encompass gaps in memories for everyday events, no longer being largely restricted to forgetting of traumatic or stressful events. In DSM-5, the criterion that "two or more distinct identities or personality states" (p. 529) must be present was replaced with "two or more distinct personality states" (p. 292), creating clear distance from the

DSM-IV view of DID as involving the invasion or intrusion of identities or personalities (sometimes called alters) that "recurrently take control of the individual's behavior" (p. 519). The latest diagnostic scheme also specifies that the "signs and symptoms may be observed by or reported by the individual" (p. 292), and that a person is eligible for a diagnosis when there are "sudden alterations or discontinuities in sense of self or agency and recurrent dissociative amnesias" (p. 293). Given the widespread use of the terms "personalities" and "identities," and the fact that these terms encompass "personality states," we continue to use all three terms in this chapter (see also Lynn et al., 2014).

DSM-5 notes that, in some cultures, personality states may be described as an experience of possession by a spirit, supernatural entity, or outside person taking control, such that the individual begins speaking or acting in a distinctly different manner (American Psychiatric Association, 2013, p. 293). In cases of possession, the personality states must be unwanted, involuntary, recurrent, distressing or impairing, and not be a part of accepted cultural/religious practices. According to DSM-5, discontinuities supposedly associated with different personality states may involve rapid and unusual shifts in attitudes, food preferences, perceptions of the body as a small child or member of the opposite gender, perceptions of internal voices, crying, and a sense of loss of self. Still, in only a "small proportion of non-possession-form cases, manifestations of alternate identities are highly overt" (p. 292) and individuals may "often conceal, or are not fully aware of . . . amnesia or other dissociative symptoms" (p. 294).

Although DID symptoms are typically more florid (e.g., flashbacks, amnesia, fugue, neurological symptoms) in women compared with men, men exhibit more criminal or violent behavior. DSM-5 specifies that the symptoms that qualify for a diagnosis of DID "are not attributable to the physiological effects of a substance (e.g., blackouts or chaotic behavior during alcohol intoxication) or another medical condition (e.g., complex partial seizures)" (p. 292) and that "the disturbance is not a normal part of a broadly accepted cultural or religious practice" (American Psychiatric Association, 2013; p. 292).

The shifts in diagnostic criteria over the years pose significant problems for diagnosticians and will probably increase the prevalence rates of DID for the following five reasons:

1. The criteria leave open to interpretation: (a) What is a "personality state," especially when it typically is not "overt," and (b) alterations or discontinuities in the sense of self or agency may be difficult to specify because behaviors and emotions are often highly variable and experienced with little sense of personal agency in everyday life (Kirsch & Lynn, 1998).
2. Relatedly, in cases of possession, the judgment of whether personality states are "involuntary" is highly subjective.
3. In some cases, it may be difficult to differentiate "ordinary forgetting" and "memory gaps" from clinically significant amnesia.
4. Including both individuals' and outside observers' evaluation of shifts in personality states as diagnostic indicators further liberalizes the criteria for diagnosing DID.
5. Because some individuals purportedly conceal amnesia or other dissociative symptoms, the diagnosis may be highly dependent on the evaluator's impressions, judgments, and beliefs about dissociation.

Prevalence and Comorbidity The prevalence rates of DID vary widely in terms of general versus clinical populations. General population studies of participants in Turkey (Akyüz Do an, Sar, Yargic, & Tukun, 1999), Canada (Ross, 1991), and North America (Loewenstein, 1994) converge on a prevalence rate of approximately 1% for DID, with approximately equal rates among males and females (American Psychiatric Association, 2013). In contrast, in inpatient settings, with the exception of a study by Rifkin, Ghisalbert, Dimatou, Jin, and Sethi (1998), the reported prevalence rates equal (Bliss & Jeppsen, 1985) or exceed (Latz, Kramer, & Hughes, 1995; Ross et al., 2002) 10%, with Ross and colleagues (1992) reporting a 14% lifetime prevalence of DID in 100 chemically dependent patients. In adult clinical settings, females predominate, although gender equality in prevalence rate is common in child clinical settings (DSM-5). Selection and referral biases may account for the imbalanced sex ratio among adults in inpatient settings: A large proportion of males with DID are treated in forensic settings or incarcerated (Lilienfeld & Lynn, 2015; Putnam & Loewenstein, 2000).

The prevalence of DID has generated much controversy, sparked by literature reviews contending that the rates of adult and childhood DID are inflated and limited to a small number of practitioners in only a few countries (Boysen, 2011; Boysen & VanBergen, 2013; see also Piper & Merskey, 2004). For example, Boysen (2011) reported that four research groups in the United States accounted for two-thirds of all 255 child cases reported since 1980, which prompted his conclusion that childhood DID is extremely rare.

The number of reported cases of DID has increased dramatically over the years. From 1970, in which approximately 80 cases were reported, the number had skyrocketed to approximately 6,000 by 1986. Over roughly the same time period, the number of alters per patient also dramatically increased from 2 to 3 to approximately 16 (Lynn et al., 2014). Paris (2012) claimed that the diagnosis of DID is little more than a fad. Nevertheless, other researchers have vigorously challenged the rarity of DID, counterpunching that DID is massively underdiagnosed (Brand, Loewenstein, & Spiegel, 2013; Ross, 2013). Studies that implement standardized assessment of DID using structured interviews with evaluators trained to a high degree of reliability and blind to symptom status and patient history are imperative to securing more accurate

prevalence estimates. Nevertheless, estimating the prevalence of DID will remain a challenge in the absence of clear-cut validating variables for the presence or absence of DID (Lilienfeld & Lynn, 2015; Robins & Guze, 1970).

DID is comorbid with many disorders, raising the question of whether the disorder is a marker for severe psychopathology or negative emotionality (Lynn et al., 2014; North, Ryall, Ricci, & Wetzel, 1993). Indeed, many disorders typically occur in conjunction with a DID diagnosis, with one study (Ellason, Ross, & Fuchs, 1996) finding that DID patients qualified for an average of 4.5 personality disorders and eight other mental disorders. Up to three-quarters of patients with DID meet diagnostic criteria for borderline personality disorder and substance abuse, and as many as 90% of patients with DID meet criteria for major depression (see Lynn et al., 2014). DID is also comorbid with PTSD, schizoaffective disorder; schizophrenia; sexual, sleep, and eating disorders; and avoidant and obsessive-compulsive personality disorders (Lynn et al., 2014). Moreover, DID is associated with markedly increased risk of attempted suicide (more than 70% of patients so diagnosed have attempted suicide), self-mutilation, and aggressive behaviors (American Psychiatric Association, 2013).

Assessment of Dissociation

Researchers have at their disposal a number of assessment instruments to evaluate trait and state dissociation based on self-report and interview modalities. In this section, we review the most widely used and best validated measures of dissociation, starting with self-report trait measures.

First developed in 1986 (Bernstein-Carlson & Putnam, 1986) and revised in 1993 (Bernstein-Carlson & Putnam, 1993), the 28-item Dissociative Experiences Scale (DES) is today the most widely used measure of trait dissociation in both applied and research settings. Participants rate the percent of the time they experience a given symptom of dissociation from 0–100% of the time at 10% intervals. Test–re-test reliabilities (Bernstein-Carlson & Putnam, 1993) range from r = .79 to r = .95, split-half reliabilities of r = .83 to r = .93, and Cronbach's alpha = .96 (Condon & Lynn, 2014; van Ijzendoorn & Schuengel, 1996). The DES has demonstrated convergent correlations with related constructs, diagnostic interview scores, and other dissociation measures (van Ijzendoorn & Schuengel, 1996). In a multicenter study (Carlson et al., 1993), a cutoff of 30 on the DES was thought to index more pathological dissociative psychopathology. With this cutoff, the DES correctly identified 80% of participants without DID and correctly identified 74% of patients with depersonalization disorder. Although research has generated mixed findings regarding the factor structure of the DES, researchers typically use three factor analytically derived scales that measure absorption, amnesia, and depersonalization (Carlson et al., 1991; Ross, Ellason, & Anderson, 1995; Sanders & Green, 1994). In nonclinical populations, the DES distribution tends to be highly skewed and vulnerable to floor effects (Wright & Loftus, 1999). Although the DES-T derivative measure introduced earlier yields a high proportion of false positive diagnoses (Giesbrecht, Merckelbach, & Geraerts, 2007), the scale is capable of distinguishing patients with DPD from control participants and has been employed in studies of dissociation, memory, and cognition (Giesbrecht, Lynn, Lilienfeld, & Merckelbach, 2008).

Researchers have developed a 30-item Adolescent Dissociative Experiences Scale (A-DES; Armstrong, Putnam, Carlson, Libero, & Smith, 1998) designed to screen for dissociative disorders and assess changes in dissociation over time. Participants rate items grouped into four subscales (i.e., absorption and imaginative involvement, passive influence, depersonalization/derealization, and dissociative amnesia) on an 11-point Likert-type scale. Norms exist for both patients and healthy adolescents, and the total score and scales are consistent internally (α = .72–.93, total score; see Giesbrecht et al., 2008). Still, the A-DES appears to be related to anxiety as well as dissociation, so its discriminant validity is questionable (Muris, Merckelbach, & Peeters, 2003).

Researchers have developed additional self-report scales of dissociative experiences and symptoms that have been used as screening instruments and research tools. These measures possess adequate-to-excellent internal consistency, test–re-test reliability, and discriminant and convergent validity, and include the Dissociation Questionnaire (63 items, α = .96; Vanderlinden et al., 1991), which has seen the widest use, the Questionnaire of Experiences of Dissociation (Riley, 1988; 26 items, α = .80; Watson, 2003), the Dissociative Processes Scale (Harrison & Watson, 1992; Watson, 2003; 33 items; α = .93), and the Perceptual Alterations Scale (Sanders, 1986; 60 items; α = .95). Unlike the aforementioned measures, the Multidimensional Inventory of Dissociation (MID; Dell, 2006; median α = .91) assesses only pathological dissociation. The 218-item MID provides scores on 23 diagnostic scales and five validity scales that evaluate participants on criteria for DID proposed by Dell (2002). The MID possesses good-to-excellent 4–8-week test–re-test reliability; r = .82–.97), convergent validity (correlations with the DES = .90), and distinguishes among patients with DID, dissociative disorder not otherwise specified, mixed psychiatric patients, and nonclinical adults (Lynn et al., 2014).

The 29-item Cambridge Depersonalization Scale (Sierra & Berrios, 2000; α = .89) assesses the frequency and duration of depersonalization symptoms. Discriminative validity is excellent, with the scale distinguishing patients with DPD from healthy patients and patients with other disorders, including anxiety disorders and epilepsy (Sierra & Berrios, 2000).

The 20-item Somatoform Dissociation Questionnaire (SDQ-20; Nijenhuis, Spinhoven, Van Dyck, Van der Hart, & Vanderlinden, 1996; α = .95) evaluates somatoform responses (e.g., unexplained neurological symptoms, sensory loss) associated with dissociative states with no

identifiable medical basis and has demonstrated success in distinguishing outpatients with dissociative disorders from nondissociative psychiatric outpatients. A five-item scale derived from the SDQ-20 is available to screen for dissociative disorders (SDQ-5, 1997) with a high degree of sensitivity (94%) and specificity (98%) among psychiatric patients (Nijenhuis, Spinhoven, Van Dyck, Van der Hart, & Vanderlinden, 1998).

Researchers have devised so-called "state" measures to evaluate relatively transient dissociative experiences. The Clinician Administered Dissociative States Scale (CADSS; Bremner et al., 1998; α = .94; Condon & Lynn, 2014; α = .80) is composed of 19 self-report items and eight observer-rated items. The self-report items are grouped into three subscales: amnesia, depersonalization, and derealization. The CADSS is sensitive to experimental manipulations intended to increase levels of dissociation (e.g., staring at a dot). Moreover, research has garnered evidence of construct validity, including high correlations with the DES (r = .56, Condon & Lynn, 2014) and discriminative validity, including the ability to distinguish among patients with PTSD, healthy adults, and patients with schizophrenia and mood disorders (Bremner et al., 1998).

The 56-item State Scale of Dissociation (SSD; Krüger & Mace, 2002; α = .97) comprises seven subscales: derealization, depersonalization, identity alteration, identity confusion, conversion, amnesia, and hypermnesia. The SSC and the DES correlate at r = .81 among people with a dissociative disorder and r = .57 among healthy adults.

Structured interviews provide a more fine-grained analysis of dissociative symptoms in terms of psychological diagnoses, with the 250-item Structured Clinical Interview for DSM-IV Dissociative Disorders-Revised (SCID-DR; Steinberg, 1994) and the 131-item Dissociative Disorders Interview Schedule (DDIS; Ross, Heber, Norton, & Anderson, G., 1989) the most widely used to discriminate dissociative disorders from other disorders. Interrater reliability for both measures is adequate to excellent, with interrater reliability ranging from r = .68 (Ross, Heber, Norton, Anderson, D. et al., 1989) to .72–.86 (weighted κ, SCID-D; Steinberg, 1994), with the exception of lower interrater reliability (r = .56) for depersonalization disorder on the DDIS, which cannot be reliably diagnosed in this context (Ross, Anderson, Fraser, Bjornson, Miller, & Reagor, 1992). Both structured interviews have demonstrated success in distinguishing among patients with dissociative disorders and nondissociative disorders, and healthy participants. Nevertheless, the SCID poses some difficulties in discriminating among dissociative disorders, bipolar disorder, borderline personality disorder, and schizophrenia (Kihlstrom, 2005).

Models of Dissociative Disorders

No discussion of dissociation and dissociative disorders would be complete without consideration of the controversies that have riven the field of dissociative disorders. In this section, we describe the two major accounts of dissociation that vie for empirical attention and support: the posttraumatic model (PTM) and the sociocognitive model (SCM), the latter called by some the fantasy model (Dalenberg et al., 2012). We suggest that a multifactorial perspective, which provides a modicum of integration of these models, provides the most comprehensive, balanced, and accurate account of dissociative experiences and disorders.

The PTM (Dalenberg et al., 2012, 2014; Gleaves, 1996; Ross, 1997) holds that dissociation is a defensive response to severe physical, sexual, or emotional abuse and/or other highly aversive events that often date to childhood. More specifically, to ward off negative emotions that would engender or intensify anxiety and suffering, or to escape the implications of abuse (e.g., "I am unlovable, bad, and so forth"), the individual either distances him or herself from reality or a sense of selfhood, as in DPD, or develops what are variously called separate selves, identities, personality states, ego states, personalities, or alters to contain, manage, and delimit threatening memories and affect, as in DID. In the latter case, the sense of self essentially fragments, with different aspects of experience and identity somehow splitting off from consciousness, disrupting the ordinary sense of continuity of experience, behaviors, and identity, thereby engendering dissociative experiences and symptoms.

The most persuasive evidence for the PTM comes from many studies that provide evidence for positive correlations between a history of highly aversive experiences and reports of dissociative symptoms and experiences. For example, in their meta-analysis, Dalenberg and colleagues (2012) reported that after excluding studies using college samples, and studies using subscales of the Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986), they obtained an overall weighted r effect-size estimate of .32 (a medium-sized effect; Cohen, 1992). Moreover, Dalenberg et al. (2012) claimed that prospective studies of trauma, using objective measures, have yielded positive correlations between highly aversive events and later dissociation.

The SCM has challenged the central assumption of the PTM of a close link between highly aversive events and dissociation. According to this account, sociocognitive and other variables must be considered in a comprehensive account of dissociation. SCM theorists (Spanos, 1994, 1996; Lilienfeld et al., 1999; Lynn et al., 2014) have claimed that the following nine points cast doubt on the existence of a reliable link between highly aversive events and dissociation:

1. Many studies of the link between trauma and dissociation lack strong corroboration of child abuse or the index event and do not permit causal inferences, as they are based on cross-sectional designs that are subject to memory biases. In contrast, prospective studies with well-corroborated cases of abuse often, but not

always, find little or no statistical association between childhood abuse and dissociation in adolescence and adulthood (see Lynn et al., 2014, for a review).
2. As noted earlier, dissociative disorders often co-occur with manifestations of mild to severe psychopathology and negative emotionality. Nevertheless, the role of comorbid conditions is rarely examined in studies of dissociation and trauma, despite the fact that such conditions may contribute substantially to dissociation (Kwapil, Wrobel, & Pope, 2002; Muris et al., 2003) and render it difficult to isolate abuse as the central causal agent of dissociation (Lynn et al., 2014).
3. Selection and referral biases common in psychiatric samples may account for high levels of child abuse among DID patients. For example, patients who are abused are more likely than other patients to seek treatment (Pope & Hudson, 1995).
4. Reports of abuse often arise in the context of a stressful or pathogenic family environment. When perceptions of family pathology are controlled statistically, the correlations between abuse and psychopathology decrease appreciably or disappear entirely (Nash, Hulsey, Sexton, Haralson, & Lambert, 1993).
5. Correlations between highly aversive events and dissociative experiences and symptoms are highly variable and range between $r = .06$ (NS) to $r = .44$ ($P < 0.001$) in nonclinical samples, and from $r = .14$ (NS) to $r = .63$ ($P < 0.001$) in clinical samples (see Dalenberg et al., 2012). Moreover, 40% of the correlations Dalenberg et al. (2012) reported were below .30, and only 6% of the correlations equaled or exceeded .50, signifying a large effect (Lynn et al., 2014). The reasons for the differences observed among correlations are unknown.
6. Highly aversive events do not necessarily precede the onset of dissociative disorders. For example, in two studies, 39.1% (Sar et al., 2007) and 24.4% (Duffy, 2000) of DID patients reported no trauma or neglect of any kind (Lynn et al., 2014).
7. Some studies that purport to find a link between a history of trauma and dissociation (see Dalenberg et al., 2012) are compromised by a lack of blindness. More specifically, diagnoses of DID were not made blindly of trauma reports, made only after records were thoroughly reviewed, and made when standardized diagnostic interviews were not completed (Lynn et al., 2014).
8. Drugs such as ketamine and other hallucinogens produce dissociative reactions (e.g., depersonalization/derealization), implying that pathways to dissociation exist independent of exposure to highly aversive events.
9. Highly dissociative individuals tend to score highly on measures of symptom exaggeration, raising suspicions about the authenticity of some of these individuals' memories and symptoms (Lynn et al., 2014).

In sharp contrast to the PTM, the SCM requires no special "dissociative mechanism" to explain dissociative symptoms (Lynn, Knox, Fassler, Lilienfeld, & Loftus, 2004). Instead, the SCM posits that DID is largely a socially constructed or reinforced condition that occurs when people are exposed to media influences (e.g., books, film, television), broader sociocultural expectations (e.g., people cope with abuse by developing "multiple personalities"), and suggestive procedures in psychotherapy (e.g., leading questions, hypnosis, repeated questioning about abuse; Lynn, Krackow et al., 2015) that cue the presentation of DID. For example, mainstream techniques for treating DID often shape or reinforce patients' displays of "separate selves" by (a) posing questions such as, "To which part am I speaking now?" or "Is there another part of you that holds your anger?", (b) conversing with different alters, and (c) employing suggestive devices, such as charts and bulletin boards, to "map the personality system" (Putnam, 1989). Moreover, the SCM contends that certain vulnerabilities increase the likelihood of a DID diagnosis, including serious coexisting psychopathology (e.g., major depression, borderline personality disorder), ambiguous or puzzling psychological symptoms, as well as high suggestibility, fantasy proneness, cognitive failures (e.g., absent-mindedness), a disrupted sleep cycle, and negative emotionality (Giesbrecht et al., 2008, 2010; Lynn et al., 2014). The SCM draws on the following findings to support the hypothesis that sociocultural and cognitive variables provide an account of dissociation and to challenge key tenets of the PTM:

1. Over the past several decades, the media in the United States accorded prominent attention to DID (e.g., movies: *The Three Faces of Eve*, television programs: *Sybil*, *The United States of Tara*), promoting awareness of DID among clinicians and patients regarding the features of DID and its purported link with abuse. These developments coincided with greatly increased numbers of patients diagnosed with DID (Elzinga, van Dyck, & Spinhoven, 1998), as well as sharp increases in the number of alters per patient (Ross, Norton, & Wozney, 1989). In most cases of DID prior to the 1970s, only one or two personalities was the norm; yet not many years later, Ross, Heber, Norton, and Anderson (1989) observed that the mean number increased to 16 personalities.

   Curiously, that number was the same number reported by the woman who went by the pseudoname of Sybil (Acocella, 1999), whose treatment was memorialized in the bestselling book (Schreiber, 1973) by the same name and the Emmy-award winning television film starring Sally Fields. Yet the case of Sybil, which monumentally shaped the cultural narrative regarding the purported tie between dissociation and abuse, has come under critical fire, with serious and credible doubts expressed regarding whether claims of abuse in her case are genuine

and the diagnosis of DID is accurate (Nathan, 2002; Rieber, 2006).

2. The possibility that DID is co-created by patient-therapist interactions is supported by findings that (a) most DID patients show few or no clear-cut signs of this condition (alters) prior to psychotherapy (Kluft, 1984), and (b) the number of alters at the time of initial diagnosis appears to have remained stable while the number of alters has increased following psychotherapy (Lynn et al., 2014; Ross, Heber et al., 1989). Moreover, a small number of therapists distributed over only a few countries, many of whom specialize in treating DID, account for the majority of cases in the published literature and probably patients seen in clinical practice (Boysen & VanBergen, 2013; Mai, 1995). Finally, the diagnosis of DID has proliferated in other countries, such as the Netherlands, where it has been the focus of extensive media and professional attention and publicity.
3. The entrenched cultural narrative enfolding DID is captured in laboratory research demonstrating that nonclinical participants who are provided with appropriate cues and prompts can reproduce many of the overt symptoms of DID (e.g., alter personalities that respond differently to psychological tests; Spanos, Weekes, & Bertrand, 1995). Moreover, persons in laboratory studies instructed to role-play the symptoms of DID report serious and implausible abuse (e.g., satanic ritual abuse) when interviewed about their childhood, consistent with clinical and media reports regarding DID (Stafford & Lynn, 2002). Across most comparisons between people instructed to simulate or role-play DID and patients with DID, few or no significant differences have emerged on measures of self-reported dissociative experiences, memory, and event-related potentials (Boysen & van Bergen, 2014).
4. Some researchers have claimed to find striking differences among alters or personality states, including differences in pain tolerance, eyeglasses prescriptions, handedness, handwriting, allergies, and heart rates (Lilienfeld & Lynn, 2015). Yet such disparities may be attributable to mundane fluctuations in mood, differences related to conscious or unconscious enactment or role-playing of different identities, or both (Lilienfeld & Lynn, 2015). Indeed, similar intra-individual differences may arise when healthy participants (e.g., actors) are instructed to role-play alters (Boysen & Van Bergen, 2014; Merckelbach, Devilly, & Rassin, 2002) or may arise on the basis of chance (type I errors), given the many psychophysiological variables considered in many studies (Allen & Movius, 2000).
5. The SCM holds that at least some reports of childhood abuse may be exaggerated or based on inaccurate memories prompted by suggestive techniques in psychotherapy. Researchers have demonstrated that in a sizable minority or even majority of participants (25–75%; Garry, 2013), it is possible to implant false memories of events such as riding in a hot-air balloon, being the victim of bullying, and being subject to a vicious animal attack (Lynn et al., 2014), implying that false memory formation is possible in more intensive psychotherapy contexts that may extend for months, years, and even decades (Lynn et al., 2014). Moreover, psychotherapists who use hypnosis tend to consult with more DID patients compared with psychotherapists who do not use hypnosis (Powell & Gee, 1999), a finding of considerable interest as hypnosis is associated with higher rates of inaccurate memories and unwarranted recall confidence compared with nonhypnotically enhanced recall (Lynn, Boycheva, Deming, Lilienfeld, & Hallquist, 2009).
6. The propensity for pseudomemories and memory commission errors associated with dissociation may be mediated by heightened levels of fantasy proneness, suggestibility, and cognitive failures, although the findings pertaining to trait dissociation and memory errors are mixed and not consistently strong in magnitude (see Dalenberg et al., 2012).
7. Contrary to the PTM, researchers have not found consistent support for an amnesic barrier that separates identities or personality states (Dalenberg et al., 2012). When objective measures, such as event-related potentials or behavioral tasks are used, studies typically find clear evidence of transfer of information across identities or alters (see Giesbrecht et al., 2010; Lynn, Lilienfeld et al., 2015).
8. Highly dissociative individuals typically experience a breakdown in cognitive inhibition (Giesbrecht et al., 2009, 2010) and, compared with other individuals, exhibit better memory for to-be-forgotten sexual words in directed-forgetting tasks (Elzinga, De Beurs, Sergeant, Van Dyck, & Phaf, 2000). These findings constitute a strong challenge to the idea that amnesia and avoidance of threat-related information are core features of dissociation.

Objections to the PTM have not gone unchallenged by adherents of the model. Specifically, proponents of the PTM (e.g., Dalenberg et al., 2012, 2014; Gleaves, 1996) have (a) criticized the SCM as failing to provide evidence for a strong link between dissociation and fantasy and suggestibility/false memories; (b) contended that trauma accounts for variance in dissociation beyond that predicted by fantasy proneness, but not vice versa; (c) argued that some studies provide evidence of a link between trauma and dissociation, even when objective measures of trauma are used; and (d) suggested that findings from laboratory studies using role-players, for example, are not generalizable to clinical populations.

Possibilities for Rapprochement and Integration

Recently, proponents of the PTM (Dalenberg et al., 2012)

and SCM (Lynn et al., 2014) have made important concessions and converged, to some extent, in their thinking about dissociation. Consensus now exists that biological (e.g., genetic) vulnerabilities, family environment, social support, developmental factors, and psychiatric history, may play a role in dissociative experiences and symptoms. Some adherents of the PTM acknowledge that: (a) Individuals with DID come to mistakenly believe they are more than one person and that DID is in part a disorder of self-understanding (Dalenberg et al., 2012, p. 568), views compatible with the SCM; and (b) "fantasy proneness—among other factors—may lead to inaccurate trauma reports" (p. 551); (c) the effects of a pathological family environment may be difficult to isolate from the effects of trauma on dissociation; and (d) therapists should eschew suggestive methods in psychotherapy.

SCM theorists (Lynn et al., 2014), in turn, have conceded that trauma may play a nonspecific causal role in dissociation by increasing stress levels and negative emotionality, particularly with regard to depersonalization/derealization in the face of high-impact aversive events (e.g., natural disaster). Moreover, the SCM acknowledges that brief dissociative reactions may persist on a longer-term basis in individuals predisposed to negative emotionality (e.g., trait anxiety, depression), especially when accompanied by comorbid psychopathology (Lynn et al., 2014). Another possibility is that fantasy proneness, suggestibility, and cognitive failures may contribute to an overestimation of a genuine, albeit weak or modest, association between dissociation and trauma (Lynn, Lilienfeld, Merckelbach, Giesbrecht, & van der Kloet, 2012). Alternatively, early trauma may predispose individuals to develop high levels of fantasy proneness or absorption, which may increase vulnerability to the iatrogenic (therapist-induced) and cultural influences posited by the SCM, thereby increasing the likelihood that DID will be diagnosed following exposure to these influences. Finally, the SCM and PTM concur that individuals with DID typically do not consciously role-play or feign the condition, but actually come to believe they possess multiple selves. In short, the subjective experience of "multiplicity" is real (Lynn et al., 2014).

Sleep and Dissociation: An Integrative Model

Recent findings regarding the link between sleep and dissociation may pave the way of integrating elements of the PTM and the SCM in a more encompassing perspective. van der Kloet, Merckelbach, Giesbrecht, & Lynn (2012) reviewed 23 clinical and nonclinical studies using a variety of measures of dissociation that assess sleep and dissociation. With only one exception, the 23 studies yielded correlations between measures of sleep disturbance and dissociation in the range of r = .30–.55. van der Kloet and colleagues (2013) tested patients experiencing insomnia who stayed one night in a specialized sleep clinic and obtained measures of a range of sleep electroencephalograph parameters along with self-report measures of dissociation, unusual sleep experiences, sleep quality, and trauma history. Dissociative symptom levels were elevated in patients suffering from insomnia, and dissociative symptoms were correlated with unusual sleep experiences (e.g., narcoleptic symptoms, sleep paralysis, nightmares) and poor sleep quality. Moreover, longer rapid eye movement (REM) sleep periods predicted dissociation.

Additional studies suggest that dissociation and sleep-related disturbances are causally related. After one night of sleep deprivation, sleep loss engendered a substantial increase in dissociative symptoms in participants that could not be attributed to changes in mood or demand characteristics (Giesbrecht, Merckelbach, & Geraerts, 2007). van der Kloet, Merckelbach, and colleagues (2012) found that when volunteers were deprived of 36 hours of sleep, dissociative symptoms, sleepiness, and mood deterioration all followed the same oscillating pattern of the sleep–wake cycle, remaining stable during the day and increasing during the night. Moreover, feelings of sleepiness preceded an increase of dissociative symptoms and deterioration of mood. van der Kloet, Giesbrecht, and colleagues (2012) examined the relation between sleep experiences and dissociative symptoms in a mixed inpatient sample at a private clinic evaluated on arrival and at discharge 6–8 weeks later. Following participation in a sleep hygiene program, decreases in unusual sleep experiences (e.g., narcoleptic symptoms, hypnagogic imagery) accompanied a reduction in dissociative symptoms. van der Kloet, Giesbrecht, Merckelbach, and Soontiens (2015) replicated decreases in dissociation in a college sample following 4 days of implementing rules of sleep hygiene. Combined, these findings imply a causal relation between sleep experiences and dissociation.

Based on the available evidence, researchers (Giesbrecht et al., 2008; Koffel & Watson, 2009a; van der Kloet, Merckelbach et al., 2012) have proposed that individuals with a labile sleep–wake cycle and unusual sleep experiences (e.g., hypnagogic hallucinations) experience intrusions of sleep phenomena (e.g., dreamlike experiences) into waking consciousness that promote fantasy-proneness linked with dissociation (see Giesbrecht et al., 2008; Koffel & Watson, 2009) and symptoms of depersonalization/derealization (see also Soffer-Dudek & Shahar, 2011). Disruptions of the sleep–wake cycle also impair memory and attentional control, producing the attention deficits, cognitive failures, and memory fragmentation experienced by highly dissociative individuals and patients (Lynn et al., 2014). Sleep disruptions, particularly in REM sleep, which affects the processing of emotional information (Walker & van der Helm, 2009), may account for the breakdown in cognitive inhibition in an emotional context that is evident in dissociative patients (Dorahy, McCusker, Loewenstein, Colbert, & Mulholland, 2006). Deficient processing of stressful events due to sleep loss also may explain the chronically increased levels of

anxiety and physiological arousal, which Giesbrecht et al. (2010) observed in patients with DPD. Moreover, daily stress interacts with trait dissociation to predict dissociative phenomena (e.g., hypnagogic hallucinations, nightmares) associated with sleep (Soffer-Dudek & Shahar, 2011). According to Lynn and colleagues (2014), this integrative perspective may explain (a) how highly aversive events or daily stressors disrupt the sleep cycle and promote errors in memory, (b) the intrusion of dissociative experiences into consciousness (e.g., fantasy and daydreaming), and (c) cognitive failures—all of which the SCM posits increase sensitivity to sociocognitive influences (e.g., suggestive psychotherapeutic techniques, media influences) and the likelihood of a diagnosis of a dissociative disorder.

Dissociation as Hyperassociation: A Novel Hypothesis

In this section, we turn the idea of dissociation on its proverbial head by positing that the tendency to hyperassociate increases the likelihood of garnering a DID diagnosis and may be an important substrate or mechanism of dissociative symptoms. The first author's (SJL) observations of six people in clinical and forensic contexts with the diagnosis of DID is the source of this hypothesis; yet there is also some support for this notion in the literature. SJL observed that all six patients exhibited a strong tendence to hyperassociate. That is, they often responded in a rapid-fire manner with associations to their thoughts, feelings, and behaviors in response to internal and external stimuli. Such hyperassociations were often accompanied by strong affect, or occurred in response to cues that elicited strong affect. At times, such associational shifts were marked by avoidance of the topic at hand and were followed by a period of silence, whereas at other times, the flow of associations led to the discussion of emotionally charged material. Occasionally, the conversation turned so far afield from the original topic that the patient lost the thread of the discussion and occasionally reported feeling "unanchored in reality" (i.e., depersonalization/derealization) or experienced difficulty in recalling elements of the conversation, implying a lack of cognitive control or coherence of associative processes. In the DID literature, this hyperassociative phenomenon has been called "switching," which might easily imply that the person is experiencing distinct "personality states."

Spitzer and colleagues (2007) observed that rapid shifting of attention in response to negative emotions during psychotherapy is why high dissociators do not achieve gains comparable with low dissociators in treatment. Hyperreactivity with respect to schema-related triggers, such as interpersonal situations and traumas, is also manifested in borderline personality disorder, a condition that is highly comorbid with DID (Saue, Arens, Stopsack, Spitzer, & Barnow, 2014). In borderline personality disorder, identity disturbance is correlated with affective instability and mood reactivity (Koenigsberg et al., 2001), which we posit are associated with poor impulse control and hyperassociativity.

Other evidence is consistent with the hyperassociation hypothesis. van Heughten-van der Kloet, Merckelbach, and Lynn (2013) suggested that excessive REM sleep during the night and/or minor REM sleep episodes during the day "fuel the type of fluid and hyperassociative cognition that is typical for dissociative disorders" (p. 630). Chiu, Heh, Huang, Wu, and Chiu (2009) reported that when experiencing negative emotion, high dissociators are particularly adept at disengaging from one task to rapidly shift to another task. According to Soffer-Dudek's (2014) review, highly dissociative individuals, especially when confronted with negative emotion, display impairments in sustained attention, focused attention when exposed to distracting stimuli, and attentional control (e.g., decreased theta brain wave activity; Krüger, Bartel, & Fletcher, 2013), as well as deficits in inhibitory functions, implying difficulties in steering and modulating mental associations.

A particular dimension of fantasizing or fantasy proneness associated with psychopathology and the tendency to engage in vivid and compelling imagery overlaps with dissociation (Klinger, Henning, & Janssen, 2009) and can be conceptualized as a tendency to fluid thinking and hyperassociation to a degree that lacks clear boundaries and can be disconnected from reality (see also Lynn, Neufeld, Green, Sandberg, & Rhue, 1996). Starker (1979) observed that emotion-loaded imagery disrupts the normal flow of imagery that ordinarily remains intact. In extreme terms, as in schizophrenia, for example, Starker hypothesized that the emotional content of the imagery process is manifested in dissociated form as hallucinations. In short, people who hyperassociate will appear to be "dissociated," particularly in response to stressors, negative emotions, and sleep disruptions that tax limited cognitive resources and the ability to impose control over mentation.

More specifically, we suggest that aspects of fantasy proneness, cognitive failures and degraded executive control, heightened suggestibility, hyperarousal, and intrusions of sleep-related mentation into everyday consciousness, lower the threshold for hyperassociation (and thereby dissociation) and account for the link between dissociation and memory errors. The combination of (a) hyperassociative tendencies, (b) a therapist who interprets associative shifts as manifestations of dissociated selves, (c) the use of suggestive methods that reify the existence of distinct identities, (d) attempts to recover memories associated with "dissociated identities," and (e) puzzling coexisting psychopathology, may be a perfect recipe for the iatrogenic creation of DID in psychotherapy. Clearly, investigating hyperassociative mechanisms in dissociation and impulse control disorders (e.g., borderline personality, bipolar disorder) is an important avenue for future research.

Psychotherapy and Dissociation

Treatments for dissociative conditions have received scant empirical attention, almost certainly less than

interventions for most major psychological disorders. Dissociative disorders are notoriously difficult to treat with pharmacotherapies. Research on medication treatment for dissociative amnesia is nonexistent, and little is known about the response of DID patients to medications. Moreover, DPD has largely proven refractory to pharmacological treatments (Simeon, 2009).

No well-controlled psychotherapy studies of DPD exist, although case studies (e.g., family therapy, paradoxical intervention, flooding) are scattered throughout the literature (Lynn, Merckelbach et al., 2015). In a study of PTSD, women with high levels of depersonalization appeared to respond better to a multimodal cognitive processing therapy compared with cognitive therapy alone (Resick, Suvak, Johnides, Mitchell, & Iverson, 2012). TMS, a procedure discussed earlier, may be a promising treatment for DPD, but controlled studies are lacking.

Numerous case studies of the treatment of DID have been reported, representing a wide swath of therapies, and all reporting positive outcomes. Yet Brand, Classen, McNary, and Zaveri (2009) were able to identify only eight studies that evaluated treatment for DID and other dissociative disorders. None of these studies was a randomized controlled trial. Accordingly, it is not possible to evaluate the reasons for symptom reduction, stemming from many potential alternative explanations, including placebo effects, regression to the mean, and the passage of time (for additional reasons, see Lilienfeld, Ritschel, Lynn, Cautin, & Latzman, 2014).

Since their earlier review, Brand and her associates (Brand, McNary et al., 2013) reported their findings of decreased levels of dissociation, PTSD, general distress, and depression over the course of a 30-month community treatment and follow-up study of patients with DID and dissociative disorder not otherwise specified. A subsample of patients aged 18–30 progressed at a faster rate than did their older counterparts (Myrick et al., 2012). In another reanalysis of the data, patients who experienced revictimization, stressors, or both revictimization and stressors over the course of treatment fared worse than did patients who did not experience such events (Myrick, Brand, & Putnam, 2013). Although the findings of this large noncontrolled study are promising, no clear-cut conclusions can be drawn from the broader outcome research because of the lack of randomized trials, dropout rates as high as 68%, the variability in treatments provided, and the failure to document clinically meaningful changes following treatment (Lynn et al., 2014).

Proponents of the SCM have roundly criticized some PTM-based interventions for being highly suggestive in recovering supposedly repressed memories, identifying and speaking with alters, and encouraging "personalities" to interact (Lynn, Condon, & Colletti, 2013). Support for such concerns comes from a study in which the majority of patients developed "florid posttraumatic stress disorder during treatment" (Dell & Eisenhower, 1990, p. 361). Moreover, after treatment commences with some PTM-based approaches, patients tend to report an increased frequency of suicide attempts, hallucinations, severe dysphoria, and chronic crises (Lynn et al., 2014). Nevertheless, Brand and Loewenstein (2014) contended that DID treatment, including interacting with "dissociated self states," improves clinical outcomes and that depriving DID patients of such treatment may cause "iatrogenic harm" (see also Brand, Loewenstein, & Spiegel, 2014). As noted elsewhere (Lynn et al., 2014), research that compares negative sequelae across classic DID and conventional therapies (e.g., cognitive-behavioral) would be worthwhile. Finally, the intriguing findings we reported regarding the links among sleep, dissociation, and hyperassociation imply that interventions that ameliorate insomnia, reduce the frequency of nightmares, address other unusual sleep-related problems (e.g., narcolepsy), and promote cognitive control and affect regulation should also be a high treatment and research priority.

Conclusion

Despite, or perhaps because of, increased empirical attention devoted to dissociative disorders over the past few decades, controversy persists. It is nevertheless encouraging that some proponents of the SCM and PTM have now called for wide-ranging investigation into dissociative phenomena and agree that a multi-pronged investigatory approach that considers multiple determinants of dissociative disorders is the best way forward. There is little doubt that tension between competing perspectives will continue to generate provocative questions, healthy debate, and ultimately a more comprehensive and nuanced understanding of dissociative disorders.

References

Ables, M., & Childer, P. (1935). Psychogenic Loss of Personal Identity: Amnesia. *Archives of Neurology and Psychiatry*, *34*(3), 587–604.

Acocella, J. (1998, April 6). The politics of hysteria. *New Yorker*, 64–79.

Aderibigbe, Y. A., Bloch, R. M., & Walker, W. R. (2001). Prevalence of depersonalization and derealization experiences in a rural population. *Social Psychiatry and Psychiatric Epidemiology*, *36*(2), 63–9.

Akyüz, G., Dogan, O., Sar, V., L. Yargic, I., & Tutkun, H. (1999) Frequency of dissociative identity disorders in the general population in Turkey. *Comprehensive Psychiatry*, *40*(2): 151–9.

Allen, J. J. B., & Movius, H. L. II.(2000). The objective assessment of amnesia in dissociative identity disorder using event-related potentials. *International Journal of Psychophysiology*, *38*, 21–41.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed., text rev.). Washington, DC: Author.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed). Washington, DC: Author.

American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.

American Psychiatric Association (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.

Armstrong, J., Putnam, F., Carlson, E., Libero, D., & Smith, S. (1997). Development and validation of a measure of adolescent dissociation:

The Adolescent Dissociative Experiences Scale. *Journal of Nervous and Mental Disorders*, *185*, 491–7.

Aspell, J. E. & Blanke, O. (2009) Understanding the out-of-body experience from a neuroscientific perspective. In C. Murray (Ed.) *Psychological and scientific perspectives on out-of-body and near death experiences*. New York, NY: Nova Science Publishers.

Aspell, J. E, Lenggenhager, B., & Blanke, O, (2009). Keeping in touch with one's self: Multisensory mechanisms of self-consciousness. *PLoS ONE*, *4*(8): e6488. doi:10.1371/journal.pone.0006488

Baker, D., Hunter, E., Lawrence, E., Medford, N., Patel, M., Senior, C., . . . David, A. S. (2003). Depersonalisation disorder: Clinical features of 204 cases. *British Journal of Psychiatry*, *182*, 428–33.

Barker, A. T., Jalinous, R., & Freeston, I. L. (1985). Non-invasive magnetic stimulation of human motor cortex. *Lancet*, *325*(8437), 1106–7.

Becker-Blease, K. A., Deater-Deckard, K., Eley, T., Freyd, J. J., Stevenson, J., & Plomin, R. (2004). A genetic analysis of individual differences in dissociative behaviors in childhood and adolescence. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, *45*, 522–32.

Belli, H., Ural, C., Vardar, M. K., Yesilyurt, S., & Oncu, F. (2012). Dissociative symptoms and dissociative disorder comorbidity in patients with obsessive-compulsive disorder. *Comprehensive Psychiatry*, *53*(7), 975–80.

Bernstein-Carlson, E. B., & Putnam, F. W. (1986). Development, reliability, and validity of a dissociation scale. *Journal of Nervous and Mental Disease*, *174*, 727–35.

Bernstein-Carlson, E., & Putnam, F. W. (1993). An update on the Dissociative Experiences Scale. *Dissociation*, *6*, 19–27.

Berntsen, D., & Rubin, D. C. (2014). Involuntary memories and dissociative amnesia assessing key assumptions in posttraumatic stress disorder research. *Clinical Psychological Science*, *2*(2), 174–86.

Bliss, E., & Jeppsen, A. E. (1985). Prevalence of multiple personality among inpatients and outpatients. *American Journal of Psychiatry*, *14*(2), 250–1.

Boysen, G. A. (2011). The scientific status of childhood dissociative identity disorder: A review of published research. *Psychotherapy and Psychosomatics*, *80*(6), 329–34.

Boysen, G. A., & VanBergen, A. (2013). A review of published research on adult dissociative identity disorder: 2000–2010. *Journal of Nervous and Mental Disease*, *201*(1), 5–11.

Boysen, G., & VanBergen, A. (2014). The simulation of multiple personalities: A review of research comparing diagnosed and simulated dissociative identity disorder. *Clinical Psychology Review*, *34*(1), 14–28.

Brand, B., Classen, C. C., McNary, S. W., & Zaveri, P. (2009). A review of dissociative disorders treatment studies. *Journal of Nervous and Mental Disease*, *197*, 646–94.

Brand, B., & Loewenstein, R. J. (2014). Does phasic trauma treatment make patients with dissociative identity disorder treatment more dissociative? *Journal of Trauma & Dissociation*, 15(1), 52–65.

Brand, B., Lowenstein, R, & Spiegel, D. (2013). Patients with DID are found and researched more widely than Boysen and VanBergen recognized. *Journal of Nervous and Mental Disease*, *201*(5), 440.

Brand, B. L., Loewenstein, R. J., & Spiegel, D. (2014). Dispelling myths about dissociative identity disorder treatment: An empirically based approach. *Psychiatry: Interpersonal and Biological Processes*, *77*(2), 169–89.

Brand, B. L., McNary, S. W., Myrick, A. C., Classen, C. C., Lanius, R., Loewenstein, R. J., . . . Putnam, F. W. (2013). A longitudinal naturalistic study of patients with dissociative disorders treated by community clinicians. *Psychological Trauma: Theory, Research, Practice, and Policy*, *5*(4), 301–8.

Bremner, J. D., Krystal, J. H., Putnam, F. W. Southwick, S. M., Marmar, C., Charney, D. S., & Mazure, C. M. (1998). Measurement of dissociative states with the Clinician Administered Dissociative States Scale (CADSS). *Journal of Traumatic Stress*, *11*, 125–36.

Brown, D. P., Scheflin, A. W. & Hammond, D. C. (1997). *Memory, trauma, treatment, and the law*. New York, NY: W. W. Norton.

Cardeña, E., Lynn, S. J., & Krippner, S. (Eds.) (2014). *Varieties of anomalous experiences* (2nd ed.). Washington, DC: American Psychological Association.

Carlson, E. B., Putnam, F. W., Ross, C. A., Torem, M., Coons, P., Bowman, E. S. . . . Braun, B. G. (1991). Validity of the Dissociative Experiences Scale in screening for mutiple personality disorder: A multicenter study. *American Journal of Psychiatry*, *150*(7), 1030–6.

Cheyne, J. A., & Girard, T. A. (2009). The body unbound: Vestibular-motor hallucinations and out-of-body experiences. *Cortex*, *45*(2), 201–15.

Chiu, C. D., Yeh, Y. Y., Huang, Y. M., Wu, Y. C., & Chiu, Y. C. (2009). The set switching function of nonclinical dissociators under negative emotion. *Journal of Abnormal Psychology*, *118*, 214–22.

Christopeit, M., Simeon, D., & Mantovani, A. (2013). 1829-Effects of repetitive transcranial magnetic stimulation (rTMS) on specific symptom clusters in depersonalization disorder (DPD). *European Psychiatry*, *28*, 1.

Condon, L. P., & Lynn, S. J. (2014). State and trait dissociation: Evaluating convergent and discriminant validity. *Imagination, Cognition, and Personality*, *34*(1), 25–37.

Dalenberg, C. J., Brand, B. L., Loewenstein, R. J., Gleaves, D. H., Dorahy, M. J., Cardeña, E., . . . Spiegel, D. (2014). Reality versus fantasy: Reply to Lynn et al. (2014). *Psychological Bulletin*, *140*(3), 911–20.

Dalenberg, C. J., Brand, B. L., Gleaves, D. H., Dorahy, M. J., Loewenstein, R. J., Cardeña, E., . . . Spiegel, D. (2012). Evaluation of the evidence for the trauma and fantasy models of dissociation. *Psychological Bulletin*, *138*(3), 550–88.

Dell, P. F. (2002). Dissociative phenomenology of dissociative identity disorder. *Journal of Nervous and Mental Disease*, *190*(1), 10–15.

Dell, P. F. (2006). The Mulitdimensional Inventory of Dissociation (MID): A comprehensive measure of pathological dissociation. *Journal of Trauma and Dissociation*, *7*(2), 77–106.

Dell, P. F. (2009). The long struggle to diagnose multiple personality disorder (MPD): Partial MPD. In P. F. Dell, & J. A. O'Neil (Eds.), *Dissociation and the dissociative disorders: DSM-5 and beyond* (pp. 403–28). New York, NY: Routledge.

Dell, P. F. (2013). Three dimensions of dissociative amnesia. *Journal of Trauma & Dissociation*, *14*(1), 25–39.

Dell, P. F., & Eisenhower, J. W. (1990). Adolescent multiple personality disorder: A preliminary study of eleven cases. *Journal of the American Academy of Child & Adolescent Psychiatry*, *29*(3), 359–66.

Dorahy, M. J., McCusker, C. G., Loewenstein, R. J., Colbert, K., & Mulholland, C. (2006). Cognitive inhibition and interference in dissociative identity disorder: The effects of anxiety on specific executive functions. *Behaviour Research and Therapy*, *44*, 749–64.

Duffy, C. (2000). *Prevalence of undiagnosed dissociative disorders in an inpatient setting* [Doctoral dissertation]. Available from ProQuest Dissertations and Theses database. (AAT No. 3041898).

Ellason, J. W., Ross, C. A., & Fuchs, D. L. (1996). Lifetime Axis 1 and II comorbidity and childhood trauma history in dissociative identity disorder. *Psychiatry*, *59*, 255–61.

Elzinga, B. M., de Beurs, E., Sergeant, J. A., Van Dyck, R., & Phaf, R. H. (2000). Dissociative style and directed forgetting. *Cognitive Therapy and Research*, *24*, 279–95.

Elzinga, B.M., van Dyck, R., & Spinhoven, P. (1998). Three controversies about dissociative identity disorder. *Clinical Psychology and Psychotherapy*, *5*, 13–23.

Friedl, M., Draijer, N., & De Jonge, P. (2000). Prevalence of dissociative disorders in psychiatric inpatients: The impact of study characteristics. *Acta Psychiatrica Scandinavica*, *102*(6), 423–8.

Garry, M. (2013). [An analysis of false memory studies of photographs, false descriptions, and suggestions of complex events]. Unpublished raw data.

Giesbrecht, T., Merckelbach, H., & Geraerts, E. (2007). The dissociative experiences taxon is related to fantasy proneness. *Journal of Nervous and Mental Disease*, *195*, 769–72.

Giesbrecht, T., Lynn, S. J., Lilienfeld, S., & Merckelbach, H. (2008). Cognitive processes in dissociation: An analysis of core theoretical assumptions. *Psychological Bulletin*, *134*, 617–47.

Giesbrecht, T., Lynn, S. J., Lilienfeld, S., & Merckelbach, H. (2010). Cognitive processes, trauma, and dissociation: Misconceptions and misrepresentations (Reply to Bremner, 2009). *Psychological Bulletin*, *136*, 7–11.

Gifford, S., Murawski, B. J., Kline, N. S., & Sachar, E. J. (1976). An unusual adverse reaction to self-medication with prednisone: An irrational crime during a fugue-state. *International Journal of Psychiatry in Medicine*, *7*(2), 97–122.

Glisky, E. L., Ryan, L., Reminger, S., Hardt, O., Hayes, S. M., & Hupbach, A. (2004). A case of psychogenic fugue: I understand, aber ich verstehe nichts. *Neuropsychologia*, *42*(8), 1132–47.

Gleaves, D. H. (1996). The *sociocognitive model* of dissociative identity disorder: A reexamination of the evidence. *Psychological Bulletin*, *120*, 142–59.

Goldberg, M. J. (1995). Complication of cervical plexus block or fugue state? *Anesthesia & Analgesia*, *81*(5), 1108–9.

Goodman, G. S., Ghetti, S., Quas, J. A., Edelstein, R. S., Alexander, K. W., Redlich, A. D., . . . Jones, D. P. (2003). A prospective study of memory for child sexual abuse: New fndings relevant to the repressed-memory controversy. *Psychological Science*, *14*(2), 113–18.

Hacking, I. (1995). *Rewriting the soul: Multiple personality and the sciences*. Princeton, NJ: Princeton University Press.

Harrison, J. A., & Watson, D. (1992). *The dissociative processes scale*. Unpublished manuscript, Department of Psychology, University of Iowa, Iowa City.

Hennig-Fast, K., Meister, F., Frodl, T., Beraldi, A., Padberg, F., Engel, R. R., . . . Meindl, T. (2008). A case of persistent retrograde amnesia following a dissociative fugue: Neuropsychological and neurofunctional underpinnings of loss of autobiographical memory and self-awareness. *Neuropsychologia*, *46*(12), 2993–3005.

Herman, M. (1938). The use of intravenous sodium amytal in psychogenic amnesic states. *Psychiatric Quarterly*, *12*(4), 738–42.

Hunter, E. C., Sierra, M., & David, A. S. (2004). The epidemiology of depersonalisation and derealisation. *Social Psychiatry and Psychiatric Epidemiology*, *39*(1), 9–18.

Janet, P. (1973). *L'automatisme psychologique*. Paris, France: Société Pierre Janet. (Original work published 1889).

Jang, K. L., Paris, J., Zweig-Frank, H., & Livesley, W.J. (1998). Twin study of dissociative experience. *Journal of Nervous and Mental Disease*, *186*(6), 345–51.

Jáuregui-Renaud, K., Ramos-Toledo V., Aguilar-Bolanos M., Montano-Velazquez B., & Pliego-Maldonado A. (2008). Symptoms of detachment from the self or from the environment in patients with an acquired deficiency of the special senses. *Journal of Vestibular Research*, *18*, 129–37.

Jáuregui-Renaud K., Sang F. Y., Gresty M. A., Green D. A., & Bronstein A. M. (2008). Depersonalization/derealization symptoms and updating orientation in patients with vestibular disease. *Journal of Neurology, Neurosurgery, and Psychiatry*, *79*, 276–83.

Jay, E. L., Sierra, M., Van den Eynde, F., Rothwell, J. C., & David, A. S. (2014). Testing a neurobiological model of depersonalization disorder using repetitive transcranial magnetic stimulation. *Brain stimulation*, *7*(2), 252–9.

Jelicic, M., Geraerts, E., Merckelbach, H., & Guerrieri, R. (2004). Acute stress enhances memory for emotional words, but impairs memory for neutral words. *International Journal of Neuroscience*, *114*(10), 1343–51.

Jiménez-Genchi, A. M. (2004). Repetitive transcranial magnetic stimulation improves depersonalization: a case report. *CNS Spectrums*, *9*(5), 375–6.

Johnson, J. G., Cohen, P., Kasen, S., & Brook, J. S (2006) Dissociative disorders among adults in the community, impaired functioning, and axis I and II comorbidity. *Journal of Psychiatric Research*, *40*, 31–140.

Keenan, J.P., Freund, S., & Pascual-Leone, A. (1999). Repetitive transcranial magnetic stimulation and depersonalization disorder. A case study. *Proceeds and Abstracts of the Eastern Psychological Association*, *70*, 78.

Kihlstrom, J. F. (2005). Dissociative disorders. *Annual Review of Clinical Psychology*, *1*, 1–27.

Kirsch, I., & Lynn, S. J. (1998). Dissociation theories of hypnosis. *Psychological Bulletin*, *123*(1), 100–15.

Klinger, E., Henning, V.R., & Janssen, J.M. (2009). Fantasy-proneness dimensionalized: Dissociative component is related to psychopathology, daydreaming as such is not. *Journal of Research in Personality*, *43*, 506–10.

Kluft, R. P. (1984). Treatment of multiple personality disorders: A study of 33 cases. *Psychiatric Clinics of North America*, *7*, 9–29.

Koenigsberg, H. W., Harvey, P. D., Mitropoulou, V., New, A. S., Goodman, M., Silverman, J., . . . Siever, L. J. (2001). Are the interpersonal and identity disturbances in the borderline personality disorder criteria linked to the traits of affective instability and impulsivity? *Journal of Personality Disorders*, *15*(4), 358–70.

Koffel, E., & Watson, D. (2009). Unusual sleep experiences, dissociation, and schizotypy: Evidence for a common domain. *Clinical Psychology Review*, *29*, 548–59.

Krüger, C., Bartel, P. & Fletcher, L. (2013). Dissociative mental states are canonically associated with decreased temporal theta activity on spectral analysis of the EEG. *Journal of Trauma and Dissociation*, *14*(4), 473–91.

Krüger, C., & Mace, C. J. (2002). Psychometric validation of the State Scale of Dissociation (SSD). *Psychology and Psychotherapy: Theory, Research, and Practice*, *75*, 33–51.

Kwapil, T. R., Wrobel, M. J., & Pope, C. A. (2002). The five-factor personality structure of dissociative experiences. *Personality and Individual Differences*, *32*, 431–43.

Laferrière-Simard, M. C., Lecomte, T., & Ahoundova, L. (2014). Empirical testing of criteria for dissociative schizophrenia. *Journal of Trauma & Dissociation*, *15*(1), 91–107.

Lanius, R. A., Vermetten, E., Loewenstein, R. J., Brand, B., Schmahl, C., Bremner, J. D., & Spiegel, D. (2010). Emotion modulation in PTSD: Clinical and neurobiological evidence for a dissociative subtype. *American Journal of Psychiatry*, *167*(6), 640–7.

Latz, T. T., Kramer, S. I., & Hughes D. L. (1995). Multiple personality disorder among female inpatients in a state hospital. *The American Journal of Psychiatry*, *152*(9),1343–8.

Lee, W. E., Kwok, C. H., Hunter, E. C., Richards, M., & David, A. S. (2012). Prevalence and childhood antecedents of depersonalization syndrome in a UK birth cohort. *Psychiatry and Psychiatric Epidemiology*, *47*, 1–9.

Lenggenhager, B., Tadi, T., Metzinger, T., & Blanke, O. (2007). Video ergo sum: Manipulating bodily self-consciousness. *Science*, *317*(5841), 1096–9.

Lickel, J., Nelson, E., Lickel, A. H., & Deacon, B. (2008). Interoceptive exposure exercises for evoking depersonalization and derealization: A pilot study. *Journal of Cognitive Psychotherapy*, *22*(4), 321–30.

Lilienfeld, S. O., & Lynn, S. J. (2015). Dissociative identity disorder: A contemporary scientific perspective. In S. O. Lilienfeld, S. U. Lynn, & J. M. Lohr (Eds.), *Science and pseudoscience in clinical psychology* (2nd ed., pp. 113–54). New York, NY: Guilford Press.

Lilienfeld, S. O., Lynn, S. J., Kirsch, I., Chaves, J., Sarbin, T. R., Ganaway, G. K., & Powell, R. A. (1999). Dissociative identity disorder and the sociocognitive model: Recalling the lessons of the past. *Psychological Bulletin*, *125*, 507–23.

Lilienfeld, S. O., Ritschel, L.A., Lynn, S. J., Cautin, R. L., & Latzman, R. D. (2014). Why ineffective psychotherapies can appear to work: A taxonomy of causes of spurious therapeutic effectiveness. *Perspectives on Psychological Science*, *9*(4), 355–87.

Lipsanen, T., Korkeila, J., Peltola, P., Järvinen, J., Langen, K., & Lauerma, H. (2004) Dissociative disorders among psychiatric patients: Comparison with a nonclinical sample. *European Journal of Psychiatry*, *19*, 53–5.

Loewenstein, R. (1994). Diagnosis, epidemiology, clinical course, treatment, and cost effectiveness of treatment for dissociative disorders and MPD: Report submitted to the Clinton Administration Task Force on Health Care Financing Reform. *Dissociation: Progress in the Dissociative Disorders*, *3*(1), 3–11.

Loftus, E. R. (1993). The reality of repressed memories. *American Psychologist*, *48*, 518–37.