Depersonalization Disorder (DPD) and Depersonalization-Derealization Syndrome — Causes and Diagnostic Criteria

See online here

Depersonalization or Derealization symptoms are common in the general population. The recurrent or almost persistent episodes of depersonalization or derealization are the cornerstone for the diagnosis of this disorder. It is reported in about 2% of the population. These recurrent episodes are characterized by experiencing unreality about one’s own self, i.e. depersonalization, or one’s surroundings, i.e. derealization. Crisis intervention therapy during the acute episodes, combined with psychodynamic therapy during the asymptomatic period is the main line of treatment for depersonalization/derealization disorder.

Overview of Depersonalization Disorder

Depersonalization is defined as an experience of unreality, detachment or being an outside observer to one’s own thoughts, feelings, and sensations. This detachment might also make the patient feel like he or she is observing their own body. Depersonalization is usually associated with altered perception, emotional or physical numbness and distorted sense of time.
Depersonalization can happen after the loss of somebody usually dear ones, heavy drinking, or the use of recreational drugs. These episodes usually last hours to days and are not persistent. The exact prevalence of depersonalization/derealization disorder in the general population is estimated to be around 2% with equal prevalence in both genders.

The main risk factors for depersonalization/derealization symptoms or depersonalization/derealization disorder are considered the same, i.e. severe depression, or anxiety. Recreational drugs, alcohol use or seizure disorder should be excluded before attributing an episode of depersonalization to depersonalization disorder.

Epidemiology of Depersonalization/Derealization Disorder

Transient episodes of depersonalization, derealization or both are common in the general population and should not be considered as pathologic.

Depersonalization can happen after the loss of somebody, heavy drinking, or the use of recreational drugs. These episodes usually last hours to days and are not persistent. The exact prevalence of depersonalization/derealization disorder in the general population is estimated to be around 2% with equal prevalence in both genders.

The main risk factors for depersonalization/derealization symptoms or depersonalization/derealization disorder are the same, i.e. severe depression, or anxiety. Recreational drugs, alcohol use or seizure disorder should be excluded before attributing an episode of depersonalization to depersonalization disorder.

Pathophysiology of Depersonalization/Derealization Disorder

After severe physical or emotional stress, depersonalization might happen as a consequence of the activation of an inhibitory hard-wired cycle in the brain. The aim of this defense mechanism is to lower anxiety by fostering the hyperarousal state of the patient. This response is the fight or flight response which is also activated when the individual perceives a threat.

When an individual comes in contact with a situation of threat to his or her own life. The issue with depersonalization/derealization disorder arises when this mode is intensified and persistently activated without the presence of a proper trigger only by mere delusion and illusion of the events.

Recent studies in the pathophysiology of depersonalization and clinical experiences have shown that the posterior cortical sensory association areas, i.e. the inferior parietal lobe, the prefrontal cortex, and the limbic system are the main brain regions involved in the mechanism. Stimulation of the inferior parietal lobe was shown to be sufficient to induce an unreal experience in patients with brain depth electrodes implanted for other purposes, i.e. for surgical evaluation of medically refractory epilepsy.

Additionally, it was shown that patients with depersonalization experiences have limbic inhibition. Patients with depersonalization/derealization disorder also have a decreased level of norepinephrine in response to anxiety compared to healthy individuals.
Unfortunately, despite our recent understanding of the pathologic neural circuits in this disorder, specific pharmacotherapy is still not existent.

<table>
<thead>
<tr>
<th>Trauma</th>
<th>Quintessential response to acute trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Organic precursors</td>
<td>Seizures, substance abuse</td>
</tr>
<tr>
<td>Neurobiology</td>
<td>Several neurotransmitter systems (NMDA, opioid, serotonin), brain regions (parietal lobe and right hemisphere) and functional brain circuits (hypo-activation of the limbic system) are associated</td>
</tr>
</tbody>
</table>

**Conceptual model**

- **Cognitive-behavioral model:** Fear is a central component with core components: dissociation of affect (no feeling) and alexithymia (difficulty identify and verbalizing emotions)
- **Psychodynamic model:** Inability to integrate various aspects of one’s self-experience

---

**Clinical Presentation of Depersonalization/Derealization Disorder**

Patients with depersonalization/derealization disorder have **persistent or recurrent episodes of depersonalization, derealization or both.**

Depersonalization episodes present with a **feeling of unreality and detachment from one’s self,** whereas, derealization is characterized by a **dreamlike perception of the surroundings.** Patients might say things, like I am no one or me, have no self when the depersonalization is severe. They represent the delusion of selflessness.

Detached patients usually show a decreased effect and are Patients with severe depersonalization might describe a split experience where they are observing their own self-doing things, i.e. out of body experience. In severe cases, the patient develops the delusion of supernatural powers observing them and their activities.

Derealization episodes are characterized by detachment and unfamiliarity with the surroundings. This indifferent behavior can stimulate the patient to feel living in an unreal world. He feels the surroundings around him are unreal and imaginary. The patient describes his or her surroundings as colorless, lifeless, or visually distorted.

Distortion and derealization from surrounding sounds and audio can also happen i.e. hallucinations may also develop in patients. Patients with this form of derealization can describe that other sounds seem to be muted, distorted, or heightened.

---

**Diagnostic Criteria for Depersonalization/Derealization Disorder**

The **DSM-5** has put strict criteria for clinicians to allow them to accurately diagnose depersonalization/derealization disorder, however, it is still believed that many cases are poorly diagnosed due to similar clinical manifestations of psychological disorders.

The **diagnostic criteria for depersonalization/derealization disorder include the following:**

A. The presence of recurrent or persistent episodes of depersonalization or derealization
B. Reality testing remains intact during such episodes
C. The recurrent or persistent episodes cause significant occupational or social
functional impairment to the patient
D. The episode cannot be explained by another medical condition such as a seizure or
drug abuse or alcohol abuse
E. The episode should not be better explained by another mental disorder such as
schizophrenia, panic disorder or dissociative personality disorder

Patients with depersonalization episodes also complain of an altered sense of time
(misperception of time) where time might seem too fast or too slow to the patient.
Additionally, patients with depersonalization episodes also have problems with
memory consolidation and recall (memory impairment).

**Note:** Patients with depersonalization/derealization disorder very commonly
have depressive or anxiety symptoms. It is important to exclude major depressive
disorder or post-traumatic stress disorder as the cause of the depersonalization episode
before establishing the diagnosis of depersonalization/derealization disorder.

**Brain Imaging in Depersonalization/Derealization Disorder**

Single-photon emission computed tomography (SPECT) studies were performed in
patients with depersonalization/derealization disorder (DDD) and healthy subjects to
understand which areas of the brain are involved when the patients are asymptomatic
and which areas are involved when they are symptomatic.

Many parts of the default mode network, i.e. thalamus, hypothalamus, insula, and others,
showed hypoperfusion in DDD patients when they are asymptomatic. When the patients
become symptomatic, the involvement of the right medial prefrontal cortex and
the right parietal-temporal regions was noted. Therefore, it is currently believed that
these two areas are the most important areas in the perception of one's own self and in
depersonalization. Future targeted treatments, i.e. deep brain stimulation, might be
useful in selected patients with DDD but extensive research is still needed.

**Brain spectroscopy studies of brain metabolites and positron emission
tomography (PET) scans** of DDD patients revealed abnormalities in the serotonergic,
opiid and glutamatergic pathways. Therefore, pharmacotherapy targeting these three
pathways has been undergoing extensive testing in patients with DDD.

**Treatment of Depersonalization/Derealization Disorder**

After evaluation of the personal and family history of the disease, the following line of
treatment is recommended:

The main treatment of DDD involves supportive psychological interventions during
the acute episodes and psychodynamic therapy during rest states when the
patients are asymptomatic.

Crisis intervention, helping with coping skills and teaching the patient new
coping skills are needed as acute interventions in patients with an acute
depersonalization or derealization episode.
Psychodynamic therapy aims to improve self-reflection and self-evaluation skills by the patient which are believed to be impaired in DDD.

Continuous, and intensive supportive interventions combined with psychodynamic therapy have been shown to improve the symptoms of DDD in most patients.

**Note:** Despite our expanding understanding of the neurobiological pathology behind DDD, **targeted therapies are still not available.** 5. Serotonin-reuptake inhibitors were tested in few and small studies and showed no efficacy in DDD despite some evidence of an impaired serotonergic pathway. In fact, one study showed that serotonin-reuptake inhibitors might induce DDD symptoms in patients being treated for major depressive disorder.

The recent evidence of an impaired endogenous opioid pathway in patients with DDD was the driving motivation behind a recent study of the efficacy of opioid-receptor antagonists in DDD. **Naltrexone, an opioid antagonist, is the first pharmacotherapy to show some direct efficacy** against the symptoms of DDD.

Patients with DDD very often have high levels of anxiety. The use of **benzodiazepines** to manage anxiety in DDD patients is discouraged because benzodiazepines were shown to induce and exacerbate DDD episodes.

**References**


[http://doi.org/10.1176/appi.books.9780890425596](http://doi.org/10.1176/appi.books.9780890425596)


**Legal Note:** Unless otherwise stated, all rights reserved by Lecturio GmbH. For further legal regulations see our [legal information page](http://www.lecturio.com/legal-information).

Notes