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Biological, psychological, and socio-structural underpinnings of dissociative identity disorder

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Abstract. Epidemiological studies revealed the prevalence and diverse symptoms of dissociative identity disorder (DID), emphasizing diagnostic complexities. Debates over its etiology, ranging from trauma to fantasy predisposition, highlight the need for ongoing research and varied therapeutic approaches. Neuroimaging has uncovered distinct structural and functional brain differences in DID patients, contributing to our understanding of its neurological basis. Despite therapeutic progress, including behavioral and pharmacological interventions, effectively DID treatments remain challenging. Integrating biological, psychological, and sociocultural perspectives is crucial for advancing DID diagnosis and treatment.

Keywords: Dissociative identity disorder, Trauma model, Theory of structural dissociation of the personality, Neural basis, Comorbidity, Treatment.

1. History

Throughout the annals of history, accounts of amnesia and dramatic changes in personality are often misinterpreted as signs of demonic possession. This interpretation had been common until the emergence of modern psychology. In 1882, Vivet's case is retrospectively recognized as one of the earliest documented instances of what is now known as dissociative identity disorder (DID), previously known as multiple personality disorder, a condition later categorized within the broader spectrum of dissociative disorders [1].

The recognition and academic interest in DID significantly has increased over time. This culminated in its formal acknowledgment in the DSM-III in 1980 as a discrete diagnostic entity, under the term "multiple personality" [2]. Currently, the latest iteration of the DSM (DSM-V-TR), published in 2022, replaced the term of "multiple personality" as "dissociative identity" and continues to affirm DID as a complex and well-established mental disorder [3]. This ongoing recognition underscores the importance of continued research, clinical understanding, and support for individuals affected by DID, as it remains a significant focus within the realms of mental health and psychology.

2. Epidemiology

There has been a limited number of epidemiology studies on DID, possibly attributed to the challenges and uncertainties of its diagnosis. The prevalence of dissociative disorders, including DID, has been estimated to be approximately 10%, with DID accounting for approximately 5% of cases in the clinical populations and in the community [4].

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Conversely, a study conducted in a community setting, specifically focusing on adult women, reported a lower prevalence rate of 1.1% for DID [5]. The prevalence in the general public was suggested to be 1% [6] while the DSM reported a 12-month prevalence of 1.5% [3].

3. Symptoms

DID is typically characterized by two primary sets of symptoms [3]. The first symptom group involves sudden shifts or disturbances in a person's sense of self and control over their actions. Those affected may experience depersonalization, uncontrolled inner voices, spontaneous emotions, and behaviors that feel foreign or involuntary, often noticeable to others [3, 7]. A defining feature of DID is the presence of distinct identities or "alters," each operating independently and with autonomy from one another. In addition to the psychological characteristics of alters, physical alterations regarding medication response, allergies, and visual acuity exists [8]. Although alter switches are typically subtle, reported physical indicators of switching may include head dropping, falling to the ground, and other minor movements [9, 10]. Additionally, while most alter switches are involuntary, there have been reports of individuals with the capacity for voluntary control in certain cases [11].

The second group of symptoms in DID is characterized by dissociative amnesia. This presents as gaps in an individual's remote memory of personal life events, lapses in reliable memory for daily activities and skills, and unexplained evidence of actions they cannot recall performing [3]. These gaps are often of traumatic or of stressful nature and cannot be explained by everyday forgetfulness [12]. Moreover, amnesia episodes are often accompanied by dissociative fugues, during which individuals may engage in sudden, unremembered travel to different locations [3].

4. Etiology

A trauma model (TM) is often used to explain the formation of DID. The model states that dissociation is a result of antecedent experiences of traumatic stress and/or severe psychological adversity typically with comorbid post-traumatic stress disorder (PTSD) [13-17]. Betrayal trauma, trauma that is resulted by individuals close to the victim and whom they rely for survival, exhibits stronger correlation with dissociation compared to non-betrayal trauma [18, 19].

Victims of betrayal trauma often manifest "betrayal" blindness, wherein they unconsciously suppress awareness of traumatic events to preserve their relationships, ultimately resulting in a compromised ability to recognize social risks [19-21]. An extreme avoidance of traumatic memories can lead to a fragmentation of one's sense of self, potentially culminating in DID and/or dissociative amnesia under numerous mediators [15, 22]. These psychological mechanisms functionally protect the victim, shielding them from the overwhelming inability to escape [23].

The Theory of Structural Dissociation of the Personality (TSDP) elaborates TM to understand DID through the division of the personality into different parts, due to repeated and overwhelming traumatic experiences, typically during early childhood [24]. TSDP proposes the personality of traumatized individuals is split into a single apparently normal part of the personality (ANP) and a single apparently emotional part (EP) of the personality. ANP is oriented towards daily functioning, while EP is oriented towards traumatic memories [24]. In DID, these parts are not fully integrated, and dissociation is the result of rapid switching between ANP and EP [25]. The theory posits that the ANP and EP(s) are developed as a result of a defense mechanism against overwhelming trauma, essentially a survival strategy to suppress emotional memory and compartmentalize the traumatic experiences, similar to that during EP, away from the part that needs to function in daily life, similar to that during ANP [24, 26]. TSDP in DID was applied in neurological research. DID patients showed blood perfusion pattern differences between EP and ANP, observed in subjects as Traumatic Personality States (TPS) and Neutral Personality States (NPS) [27]. Patients presented different biopsychological reactions, suggested by overactivation in TPS and underactivation in NPS under trauma-related stimuli (see “Neural basis” section) [25, 27].

Contrary to the TM, the fantasy model (FM), also known as the socio-cognitive model, suggests that dissociative experiences are mediated by a high level of fantasy proneness and exaggeration of

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symptoms [15, 28, 29]. According to FM, trauma is not a significant contributor as memories of trauma are subjected to imagination and exaggeration rather than the actual trauma [15, 30]. Instead, the development of DID is significant influenced by social and cultural factors, including media exposure to DID and suggestive techniques used by therapists [31, 32]. However, the fantasy proneness score of DID patients were reported to be similar to the normal population [26]. Psychobiological studies generally supported more TM than FM [26, 28].

5. Diagnosis

The DSM-V [3] listed 5 diagnostic criteria of DID: disruption of identity of by two or more distinct personality states, recurrent recall gaps, significant impairment in functioning, not a normally accepted in cultural or religious practice, and are not attributable to the physiological effects of a substance. However, the diagnostic validity of DID remain controversial [33-35]. Studies have raised concerns regarding these criteria for diagnosing DID. It is often observed that DID patients exhibit a wide range of symptoms beyond the core diagnostic features, possibly due to the high comorbidity of dissociative disorders with other psychiatric disorders [36, 37]. It has been proposed that additional DID symptoms, such as questions about dissociation or an experience of possession, should be included in the diagnostic criteria of DSM-V [36, 38].

In clinical practice, the diagnostic process employs adjunctive tools such as symptom measurement scales, including DES-II [39], SCID-D [40], MID [41], and CDS [42]. Nevertheless, these scales have been criticized for their perceived shortcomings in discriminant validity and susceptibility to experimenter and participant biases [43]. A universally accepted gold-standard criterion for diagnosing DID has yet to be established.

It is believed that the accuracy of diagnoses soley on symptom measurement scales is compromised due to the absence of dependable diagnostic tools [43]. Recent research highlighted a successful and highly accurate discrimination of brain structure patterns between DID patients and healthy controls using magnetic resonance imaging (MRI) [44]. This suggests neuroimaging approaches can be used as a diagnostic benchmark for DID.

6. Neural basis

The intricate interplay between neural mechanisms and DID underscores the complexity of this mental health condition. Numerous studies have reported differences in brain structure between individuals with DID and those without the disorder. Specifically, individuals with DID tend to have smaller volumes in hippocampus and parahippocampus gyrus [17, 49, 50]. DID patients showed lower hippocampus volume than dissociative disorder not other specified (DDNOS) [50]. Patients that recovered from DID showed more hippocampal volumes than patients with florid DID [50]. Furthermore, DID patients exhibit unusual shapes in certain parts of hippocampus, including the cornu ammonis (CA), presubiculum, and subiculum regions [49]. Among these regions, CA1 significantly contributes to dissociative amnesia and was suggested to be its potential biomarker [51, 52]. Additionally, the extent of hippocampus and parahippocampus gyrus reduction is positively associated with the severity of childhood traumatization, emotional neglect, and dissociation symptoms in DID patients [17, 50, 51]. As hippocampus have a high glucocorticoid receptor density that is sensitive to the stress hormone, cortisol, chronic traumatic stress may lead to neuron damage and explain the reduced hippocampal volumes [50, 53-56]. However, another study revealed normal hippocampal size of DID patients as controls and was significantly larger than PTSD patients [57].

Individuals with DID was reported to have smaller amygdala volumes and a higher ratio of hippocampal to amygdala volume compared to those without the disorder [17, 50]. This difference could be attributed to the amygdala being less sensitive to the effects of excessive stress hormones compared to the hippocampus [51, 58, 59]. However, more recent studies showed the amygdala volume in DID patients was not significantly different from that in controls and was significantly larger than that in PTSD patients [57, 58].

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Apart from the structural studies, several functional studies also presented differences both between DID and healthy controls as well as between distinct identity states within a DID patient [26, 60-62]. Heathy controls exhibited better working memory activation in left frontal pole and ventrolateral prefrontal cortex, whereas DID patients did not show such activation [60]. In healthy individuals, the hippocampus is actively involved in the memory retrieval process, which is lacking in DID patients [26, 61]. Behavioral performances and brain activation patterns related to working memory were reported to be dissociative identity state-dependent [60]. The activated areas of NPS and TPS were suggested to be divided into two distinct neural networks [26]. While the NPS activates areas in the cerebral cortex, TPS mainly activates subcortical areas [26]. Compared to NPS, TPS demonstrated an elevated cerebral blood flow in the amygdala, insular cortex, somatosensory areas in the parahippocampal gyrus, parietal cortex, the basal ganglia, the occipital and frontal regions, and the anterior cingulate [25, 27, 63]. TPS had increased blood perfusion in the dorsomedial prefrontal cortex, primary somatosensory cortex, and motor-related areas, while NPS had increased blood perfusion in bilateral thalamus [62]. NPS involves brain regions engaged in suppressing emotional memories in mentally healthy individuals, namely, frontal areas, cingulate cortex, and intraparietal sulcus [26, 61].

In the context of understanding the hormonal differences and neural transmission in patients with DID, it is noteworthy that the current body of research predominantly focuses on other psychological disorders, such as PTSD. This emphasis in research is relevant considering they share similar methods of development according to TM. Research showed differences in the basal functioning and reactivity of the hypothalamic-pituitary-adrenal (HPA) axis between PTSD patients and controls [64]. Various reports have revealed aberrant HPA-axis functioning in PTSD, such as higher [65] or lower cortisol level [66, 67] and elevated corticotropin releasing factor (CRF) level in the cerebrospinal fluid [68-70]. Further research is recommended to demonstrate if these differences are also present in DID patients.

Although currently there are a limited number of studies discussing the biomarkers of DID, several studies covered the biomarkers of pathological dissociation that lead to dissociative disorders, including DID. Functional biomarkers are activity levels in specific brain regions, including the dorsomedial and dorsolateral prefrontal cortex, bilateral superior frontal regions, (anterior) cingulate, posterior association areas, and basal ganglia [71, 72]. Neurostructural biomarkers include reductions in hippocampal volume, basal ganglia volume, and thalamus volume [17, 49, 50, 71]. Psychobiological markers include elevated oxytocin, prolactin levels and diminished tumor necrosis factor alpha (TND-α) levels [71, 73-77].

Although these studies provide important insight into the neural basis of DID, it is important to acknowledge that several challenges affect their validity. Firstly, a significant portion of these studies relied on the same limited pool of patient data, all obtained from a common data source, as suggested in table 1 [25, 49, 51, 58]. Secondly, these studies employed inclusion criteria that were based on the older DSM-IV diagnostic criteria for DID rather than the more recent DSM-V criteria. Thirdly, the analyzed samples exclusively consisted of female subjects due to the unavailability of male healthy controls and patients, raising concerns about the generalizability of their findings. Given these limitations, it is evident that further research is needed to address these issues and enhance the robustness of our understanding of DID from a neurological perspective.

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Table 1. Neural studies of DID. ANP: apparently normal part; CA: cornu ammonis; DES: dissociative experiences scale; DID: dissociative identity disorder; DG: dentate gyrus; EP: emotional part; fMRI: functional magnetic resonance imaging; GC: granule cell; MRI: magnetic resonance imaging; ML: molecular layer; NPS: neutral personality states; PET: positron emission tomography; PTSD: post-traumatic stress disorder; TPS: traumatic personality states

Evidence	Method	Sample size	Ref
• Less working memory activation in the left frontal pole and ventrolateral prefrontal cortex • Did not engage the parietal regions during trauma-related identity states for linear load	fMRI	Diagnosed DID (n=14), DID-simulating controls (n=16), PTSD (n=16), healthy controls (n=16), all female	[60]
• Significant differences in regional blood flow patterns for the two dissociative identity states, but not for trauma-related memory scripts. • NPS level of the dissociative identity states main effect revealed a broad pattern of brain areas that showed an increase in regional blood flow relative to the TPS	PET	11 DID patients, all female	[63]
• Decrease in perfusion when TPS listened to the trauma-related script as compared to NPS • Area with the most significant increase of activation is the parietal operculum	PET	11 DID patients, all female	[27]
• Dissociative part-dependent biopsychosocial reactions to masked neutral and angry faces. During EP, they are overactivated, and during ANP underactivated • Controls were not able to simulate genuine ANP and EP biopsychosocially.	fMRI	DID (11) and healthy controls (15), all female	[25]
• Increased activity within the left temporal gyrus, orbitofrontal cortex, and dorsomedial prefrontal cortex	fMRI	15 DID patients and 15 healthy controls, all female	[62]
• The fantasy proneness score of DID patients were reported to be similar to the normal population • While the hippocampus plays a significant role in memory retrieval in healthy individuals, its contribution is absent in memory retrieval of DID patients • NPS exhibited a significant convergence with the activated brain regions involved in suppressing emotional memories in mentally healthy individuals, namely in frontal areas, cingulate cortex, and intraparietal sulcus	PET	11 DID patients, 10 high fantasy prone DID simulating controls, 8 low fantasy prone DID controls	[26]

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Table 1. (continued).

• Smaller global hippocampal volume • Abnormal shape and smaller volume in CA 2-3, CA 4-DG, and (pre)subiculum	MRI	PTSD (n=16), PTSD-DID (n=17), and healthy controls (n=32), all female	[49]
• Patients with complex dissociative disorders have smaller volumes of hippocampus, parahippocampal gyrus, and amygdala • These volumes are associated with severity of psychoform and somatoform dissociative symptoms • Patients who recovered from DID have more hippocampal volume that patients with florid DID	MRI	10 DID-patients, 13 DDNOS-patients, 10 DID-patients who completely recovered from DID, and 20 healthy controls, all female	[50]
• No significant difference in amygdala and hippocampus size between dissociative amnesia/DID and controls • Larger amygdala and hippocampus size of dissociative amnesia/DID than PTSD	MRI	23 in-patients diagnosed with dissociative amnesia/DID or PTSD, 25 healthy controls, all female	[57]
• Smaller hippocampal and amygdala volume • Higher hippocampal to amygdala volume ratio	MRI	DID (n=15) and healthy controls (n=23), all female	[38]
• Normal amygdala morphology in DID	MRI	DID (n=32) and healthy controls (n=43), all female	[58]
• Smaller bilateral hippocampal global volume • Smaller bilateral CA1, right CA4, right GC-ML-DG, and left presubiculum • Dissociative amnesia is significantly correlated with reduced bilateral hippocampal CA1 subfield volumes and between total DES scores and left CA1 subfield	MRI	DID (n=32) and healthy controls (43), all female	[51]

7. Comorbidity

Seventy-five percent of DID patients suffered from at least 4 DSM-IV psychiatric disorders in addition to DID [78]. The DSM-IV classifies mental disorders into different axes as part of its axial system, among these axes, DID and DDNOS suffered from significantly more axis-I disorder, which are clinical disorders other than personality (and developmental) disorders (table 2) [78-80]. Eighty-two percent of adult DID patients had at least one comorbid axis-I disorder with a mean number of comorbid disorder about 7 [81]. Among which, PTSD and Major depression were believed to be the most common comorbid conditions with DID, with about 79.2% of DID patients to present PTSD and 11.2%–97.2% for depression, depending on whether the exclusion rules for psychosis in the depression diagnosis apply [78, 81-83]. In addition, some believed the diagnosis of DID can increase their risk of developing depression while the stress associated with DID symptoms can contribute to the development of anxiety disorders [84]. DID also presents a comorbidity with ADHD and anxiety disorders, such as generalized anxiety disorder (GAD), social anxiety disorder, and panic disorder [82, 83]. DID is also associated with

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substance abuse disorders due to an incapability to cope with stress from DID [85]. Lastly, borderline personality disorder (BPD) is also prevalent in DID [86].

Table 2. Comorbidity studies of DID. ADHD: attention deficient hyperactivity disorder; BPD: borderline personality disorder; DID: dissociative identity disorder, DDNOS: dissociative disorder not other specified, PTSD: post-traumatic stress disorder

Comorbidities	Results	Ref
PTSD and anxiety disorders	• 75% of DID patients suffered from at least 4 DSM-IV psychiatric disorders in addition to DID • The most prevalent comorbidity in DDNOS and DID was PTSD	[78]
Major depression, PTSD, anxiety disorders, ADHD	• All 25 patients of DID also have major depression (n=25), PTSD (n=22), anxiety disorders (n=21), ADHD (n=15)	[82]
Major depression, PTSD	• Axis I comorbidity with 107 inpatients show 88 inpatients (82.2%) received a diagnosis of a severe mental disorder. • Major depressive episode was found in 104 (97.2%) inpatients, but only 12 (11.2%) inpatients met the Diagnostic and Statistical Manual of Mental Disorders-III-Revised (DSM-III-R) criteria for this diagnosis when exclusion rules for psychosis were applied. • 57 (79.2%) inpatients were diagnosed with PTSD.	[81]
PTSD, anxiety disorders	• 80.6% of DID patients met criteria for PTSD • Anxiety disorders are also prevalent in DID patients	[83]
PTSD, substance abuse disorder	• DID co-occur with PTSD and substance abuse	[85]
BPD	• DID commonly co-occurs with BPD and vice versa • individuals who meet criteria for both disorders have more comorbidity and trauma than individuals who meet criteria for only 1 disorder	[86]

8. Treatment

Behavioral treatment for DID typically follows a three-phase strategy with the goal of supporting personality integration and eliminating the reasons for dissociation [8, 24]. The first phase aims to ensure safety, achieve stabilization, and diminish symptoms. This initial phase focuses on creating a secure attachment environment, both in therapy and individuals' life. Therapists help patients develop coping mechanisms to manage and reduce symptoms, aiming to stabilize individuals and improve their overall functioning. In the second phase, therapy delves into identifying, confronting, and processing traumatic memories. The goal is to help individuals understand and integrate these experiences, reducing the power of the trauma, and lessening the fragmentation of the self. Finally, the third phase focuses on the unification of identities and fostering rehabilitation. The final phase emphasizes the integration of various identity states into a more cohesive self and focuses on improving the individual's daily functioning and quality of life. Rehabilitation involves setting goals, building future plans, and enhancing social and occupational skills [8, 24].

There is currently no FDA-approved drug treatment for DID. There is sparse evidence for the efficacy of specific psychopharmacological agents in the treatment of dissociative disorders, such as Lamotrigine [87], Paroxetine [88], and Naloxone [89]. However, these studies did not focus on DID patients but used other conditions such as PTSD and general dissociative disorders, warranting more studies to state their efficacy and neurobiological substrates in DID [90].

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More commonly, drug treatments are prescribed to treat co-occurring mood and anxiety symptoms associated with DID [90]. In the treatment of patients diagnosed with DID, there have been reports indicating efficacy of various medications such as selective serotonin reuptake inhibitors (SSRIs), tricyclic antidepressants, monoamine oxidase inhibitors, beta blockers, clonidine, anticonvulsants, and benzodiazepines in mitigating intrusive symptoms, hyperarousal, anxiety, and mood instability [91, 92]. Other potential pharmacological interventions for DID include the utilization of prazosin to alleviate nightmares, carbamazepine to address aggression, and naltrexone for managing recurrent self-injurious behaviors [91].

9. Conclusion

The historical progression of DID is characterized by significant shifts in interpretation, diagnostic controversies, and advancements in understanding its complex nature and neurological foundations. DID has undergone a transformative journey towards formal recognition within contemporary psychology, mirroring broader developments in mental health discourse. Epidemiological research has showed the prevalence and multifaceted symptoms associated with DID. This underscores the challenge of achieving accurate diagnosis. The etiology of DID, whether attributable to trauma or predisposition to fantasy, underscores the imperative for continued investigation and therapeutic approaches. Neuroimaging endeavors have yielded valuable insights into the neural substrates of DID, unveiling structural and functional distinctions within the brains of DID patients. Despite advancements in therapies encompassing behavioral interventions and pharmacological treatments, the journey toward effective DID treatment remains challenging. Consequently, a comprehensive understanding of DID, integrating biological, psychological, and sociocultural factors, is imperative for better diagnosis and treatment of DID.

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