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# Dissociative Amnesia: Remembrances Under Cover

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## Abstract

The existence or questionability of “repressed memories” can be discussed as being a matter of definition. It seems, however, far-fetched to consider all “lost” memories as caused by encoding problems, brain damage, forgetfulness, failure to disclose events, and so on. We argue that dissociative amnesia (DA) (or “psychogenic amnesia,” or “functional amnesia,” or, as we favor to call it, “mnestic block syndrome”) is caused by psychic alterations, but ultimately they can be traced to changes in the physiology of the brain, as we are of the opinion that all memory processes—positive or negative—alter brain functions, sometimes more permanently, sometimes transiently. We have proven this idea using functional imaging techniques, in particular fluoro-deoxy-d-glucose positron emission tomography. Having investigated dozens of patients with severe and long-lasting DA conditions, we believe it to be disrespectful to many (but not to all) of the affected patients to question their disease condition, which can be proven to be not caused by feigning, malingering, or direct brain damage.

**Keywords:** Mnestic block syndrome; Neuropsychiatry; Autobiographical memory; Brain imaging; Dissociation; Dissociative disorders

## 1. Introduction

There is an ongoing debate on the old idea that memories can be repressed or suppressed (Dodier, Gilet, & Colombel, 2022; Erdelyi, 2006; Freud, 1898, 1899; Hartmann, 1930; Jung,

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1905; Kunzendorf & Moran, 1993/94; Loftus, 1993, 1994; Markowitsch, 2000; Otgaar et al., 2019; Suarez & Pittluck, 1975). We will argue for the existence of repressed memories on the basis of the dissociative condition named “dissociative amnesia” (DA) (Markowitsch & Staniloiu, 2016; Markowitsch, Staniloiu, Kordon, & Sarlon, 2018; Staniloiu & Markowitsch, 2014; Staniloiu & Markowitsch, 2022; Staniloiu, Markowitsch, & Kordon, 2018). By doing so, we will center on Tulving’s (2002, 2005) and Semon’s (1904) concept of the state dependency of memories, on the relation between stress and memory (Staniloiu, Kordon, & Markowitsch, 2020b), mechanisms by which DA is likely to occur (“Two-hit hypothesis”) and differential diagnostic criteria for the occurrence of DA.

We are of the opinion that our arguments favor the existence of repressed memories in the context of DA and that there are cognitive and biological bases demonstrating that repressed memories are a valid entity in the context of DA.

While most lay people would consider their memories to be reliable and stable over time, research shows that this in fact may not be the case. Especially (but not exclusively) autobiographical memories are vulnerable with respect to influences both from within the memories’ owner and by variables from outside (e.g., Kühnel, Woermann, Mertens, & Markowitsch, 2008; Loftus, 2005; Markowitsch & Staniloiu, 2022; Radulovic, Lee, & Ortony, 2018; Staniloiu, Kordon, & Markowitsch, 2020a; Wixted, Mickes, & Fisher, 2018). For memories which had been successfully encoded and consolidated in the past, but cannot be retrieved consciously in the present, the expressions *repression* and *suppression* have been used and are debated since their creation (see, e.g., the commentaries to the article of Erdelyi, 2006; *repression* seen as an unconscious and *suppression* as a conscious process, but see Panel 1 in Staniloiu & Markowitsch, 2014). Both concepts are especially linked to Sigmund Freud’s work (Langnickel & Markowitsch, 2006) and were revived in more recent years in the context of DA (Markowitsch, 2000; Markowitsch & Staniloiu, 2016) as well as in neuroscience (Anderson & Floresco, 2022; Brand et al., 2009; Kikuchi et al., 2009; Kunii, Okano, Mashiko, Yabe, & Niwa, 2012; Ross, Homan, & Buck, 1994), but also with respect to court cases of trauma and sexual abuse (e.g., DePrince & Freyd, 1997).

Already in 1982, Loftus and Burns published a paper under the headline “Mental shock can produce retrograde amnesia,” in which it was demonstrated that the perception of emotionally laden episodes—violence in a film—can lead to retrograde amnesia. (As Loftus and Burns stated initially in their paper: “Retrograde amnesia refers to the loss of memory for events that occur prior to some critical incident, such as a head injury, electroconvulsive stimulation, ...”). Similarly, Anderson and Hulbert (2021) stated that “[m]emories are, at times, too accessible for our own good” (p. 2) and, therefore, may become repressed or suppressed from conscious access. It is, therefore, important, how the environment is perceived—which depends on the interpretation of previous similar situations (Tulving, 2002, 2005).

This condition is, in fact, seen in various—usually stressful—situations, starting with the simple “tip-of-the-tongue”-phenomenon, where access to a specific word, item, or expression may be blocked, though the person is aware of knowing it in principle (Schwartz & Metcalfe, 2011). In more complex stressful situations, acting alternatives may not come to mind and the personal past is inaccessible. Already the creator of the term “amnesia” in the medical field of the 18th century, Boissier de Sauvages, referred to a “pathematic” amnesic subcategory

due to “emotional suffering or afflictions of the spirit, following strong emotions such as fear, terror or chagrin” (Langer, 2019, p. 66).

## 2. Dissociative amnesia

The completest form of a memory block is so-called generalized DA which according to DSM-5 (2013) refers to an inability to recall important autobiographic or personal information of the *total* past (usually of a traumatic or stressful nature) that is inconsistent with ordinary forgetting (p. 397f). In the ICD-11 (2021), DA “is characterised by an inability to recall important autobiographical memories, typically of recent traumatic or stressful events, that is inconsistent with ordinary forgetting.” (<https://icd.who.int/browse11/l-m/en#/http://id.who.int/icd/entity/626975732>). While generalized amnesia is considered a rarer form of DA, we have studied nearly 100 patients with the diagnosis of DA and had less than 10 who did not have the generalized form. Similarly, Pope, Schnabel, and Hudson (2023), in a bibliographic survey, found only seven out of 89 papers, published between 2011 and 2020, on DA to report localized or selective amnesia for specific events. Consequently, DA seems to be not infrequent and seems to be mostly general; some researchers consider it to be a “substitute term for repressed memory” (Otgaar et al., 2019, p. 1073). We termed this phenomenon “mnestic block syndrome” (e.g., Markowitsch, 2002; Markowitsch et al., 1999, 2000a) in order to indicate that the memories are not lost, but their retrieval is blocked.

## 3. Memory blockade and state-dependent memory

Tulving (1983, 1985, 1995) revived the concept of Semon (1904) on *state-dependent memory formation and retrieval* and combined it with the *encoding-specificity principle* (Staniloiu et al., 2020a; Tulving, 2002, 2005; Tulving & Thompson, 1973). “State-dependency” implies that the internal and external state contribute to the efficacy with which information is processed. For memory retrieval, optimal conditions apply when the environmental conditions and those of mood and physical state match those during encoding. The “encoding-specificity principle” implies that memories are linked to the context in which they are created. Another of Tulving’s terms also originated from Semon’s (1904) work and is closely related to the state-dependency—namely, the expression *ecphory* (Tulving, 1983, p. 175ff). “Ecphory” describes the process by which retrieval cues interact with stored information so that an image or a representation of the information in question appears. A mismatch between encoding and retrieval conditions may lead to a variety of memory retrieval disturbances, ranging from the common tip-of-the-tongue phenomena (see above) to complete pathological retrieval blockades.

These phenomena are strongly related to emotional processing. Already at the end of the 19th century, Sigmund Freud anticipated the concepts of ecphory and state-dependency in his descriptions, by emphasizing the importance of emotions (Freud, 1895/1950; Peper & Markowitsch, 2001).

## 4. Stress

Links between emotion and repressed memories or DA were already noted at the time of the first descriptions of DA—and named *hysteria* (Bauer, 1917; Breuer & Freud, 1895; Briquet, 1859; Mai, 1995). Initially, hysteria was strongly related to neuroticism and included as a disease in the first two editions of the *Diagnostic and Statistical Manual of Mental Diseases* (DSM); thereafter, the name was replaced by “dissociative disorders” and a stress- or trauma-related etiology was linked to the disorders.

Other terms—especially for DA—are “psychogenic amnesia” (Markowitsch, 2003) and “functional amnesia” (Markowitsch & Staniloiu, 2016). The last term implies that the disorder serves a function for the patients—though they frequently appear handicapped with respect to certain features (e.g., autobiographical memory, self-consciousness; Markowitsch & Staniloiu, 2022)—they have “secondary gains” (e.g., no longer having to work, being taken care of, not having to be responsible for personal affairs). For an outsider, nevertheless, the adverse consequences of functional amnesias seem to outweigh those of leading an unimpaired life and, therefore, an answer to this paradox is required.

The simple, but probably true answer is that these individuals lack alternatives. They are unable to cope with an adverse environment—they lack resilience and they frequently have to deal with approach-avoidance behavior. For instance, children may be led to a double bind—the child needs protection and attachment to the caretaker, but is terrified and harmed by the caretaker. This then may lead to a lack of escape strategies aside from dissociation. This mechanism has affinities to the “learned helplessness”-concept of Seligman (1972), though it certainly goes beyond it. Schauer and Elbert (2010) pointed out that after repeated situations without escape, the individuals react with tonic immobility. Such situations are strengthened by a lack of social support, denial of trauma by one’s social support system, and the like. Such consequences of lacking a proper, safe childhood and youth were described since Bettelheim (1950) (e.g., Hakamata et al., 2021; Lassri, Bregman-Hai, Soffer-Dudek, & Shahar, 2023; Y. Yang et al., 2022). That they are long-lasting, was, for example, described by Fries, Ziegler, Kurian, Jacoris, and Pollak (2005). These authors studied former orphans who had lived for their first years in poorly managed orphanages and thereafter were adopted by U.S.-American parents and then lived for the next 3–4 years together with the parents’ biological children. The authors tested the release of two binding hormones—oxytocin and vasopressin—under resting conditions and while the mother interacted with the children. They found a—compared to the parents’ biological children—reduced release of these binding hormones in the former orphans compared to the mother’s own children.

The lack of escape strategies is accompanied by symptoms of depression, feelings of worthlessness, overgeneralized self-blaming, hopelessness, loss of interest and pleasure, and an overgeneral autobiographical memory effect (“semanticizing of personal events”) (Becquet et al., 2021; Hakamata et al., 2021; Harrison et al., 2022; Sutin, Luchetti, Aschwanden, Stephan, & Terracciano, 2021; Williams et al., 2007). Of course, such an immobility in having behavioral alternatives is intimately linked to the concept of stress (Lupien, Juster, Raymond, & Marin, 2018).

Depression is also commonly found in patients with DA (Staniloiu & Markowitsch, 2014, e.g., their Panel 3: “Psychiatric history of depressive episodes” being suggestive of a diagnosis of DA; Staniloiu & Markowitsch, 2018), as, of course, is stress (Staniloiu et al., 2020b). Nevertheless, the reverse conclusion—that many depressed people should manifest DA—is not valid, as usually resilience factors counteract this idea (Staniloiu & Markowitsch, 2018).

## 5. Two-hit hypothesis of DA

The above-mentioned examples (e.g., Joy et al.: repeated flooding leading to learned helplessness) are in line with what Staniloiu and Markowitsch (2014) introduced as the *two-hit hypothesis*: This postulates an additive or synergistic interaction between psychological or physical incidents that may lead to DA. Zannas, Provençal, and Binder (2015); their fig. 1) elaborated from an epigenetic perspective on such repeated traumata which in the absence of proper resilience may lead to post-traumatic stress disorders—a frequent concomitant of DA. Huan Wang and coworkers (2022) found in a functional imaging study convincing evidence for the fact that “even mild ELS [early-life stress] might confer vulnerability to exposure to stressors later in adulthood” (p. 2123).

We found in almost all of our patients evidence for the existence of at least two “hits” which were likely to lead to DA (Fujiwara et al., 2008; Markowitsch et al., 1999; Staniloiu & Markowitsch, 2012; Staniloiu et al., 2018, 2020b). From analyzing the cases, we consider the hits to be crucial for the occurrence of DA, though, of course, other variables (e.g., depression, little satisfaction with current life circumstances, adverse environmental concomitants) contribute to the actual manifestation of DA. This also implies that counteractive variables (e.g., a “safe” environment during development, one or more reliable reference persons) can still prevent the development of DA. While these arguments appear very plausible in our eyes, they are, of course, only correlative, which lies in the nature of the findings.

## 6. Stress-related amnesia and the brain

Results from neuroanatomical studies point to numerous brain changes, accompanying stress conditions, and possibly also leading to amnesia. Studies, applying brain imaging, not always lead to consistent results, which, of course, was taken-up by the opponents of the hypothesis that stress can lead to DA and that DA implies repression of memories (Huntjens, Otgaar, Pijnenborg, & Wessel, 2022; Otgaar et al., 2019). However, when applying careful selection criteria both with respect to patients and methods, results from brain imaging become more consistent and uniform. The hippocampal formation is in the center of such studies, as it is the par excellence structure for memory processing (Tulving & Markowitsch, 1998), and numerous studies point to stress-related changes—such as volume reductions—in the hippocampus (e.g., Bremner & Wittbrodt, 2020; Weissman et al., 2022). The next important structure is the amygdala, because of its involvement in the processing of emotions (Adolphs, 1999) and its downregulation in stress-related disease conditions (e.g., Bryant

et al., 2022; Watson et al., 2022), which—for hysteria/DA—already in the 19th century was termed *belle indifference* by Janet (1892, 1893, and was confirmed in later studies such as in Reinhold & Markowitsch, 2009). Again, many studies found stress-related changes in this nuclear group (e.g., Caetano et al., 2022; Ford et al., 2022; Lippard & Nemeroff, 2020; Weissman et al., 2022). Studies on orphans with early institutional deprivation revealed changes in amygdala volumes (Mehta et al., 2009). Other brain regions, in particular the prefrontal cortex, react toward stress as well (Bremner et al., 1995; Hakamata et al., 2021; Meine, Meier, Meyer, & Wessa, 2021).

As mentioned initially with the citation of Boissier de Sauvages, extremely emotional situations may lead to amnesia. The mechanism by which this may occur was delineated already in Markowitsch (2000): Stressful life events can induce a rapid and vehement release of stress hormones (O'Brien, 1997; Shansky & Lipps, 2013)—glucocorticoids—which block the receptor channels of memory-processing regions, especially in the temporal lobes (hippocampus, amygdala) and other limbic system structures. In consequence, the access to stored engrams may be disrupted. Interestingly, the largest number of receptors for stress hormones in the brain are in the amygdala and hippocampus, and, therefore, in those areas where emotional memory is synchronized (Cahill, Babinsky, Markowitsch, & McGaugh, 1995; Markowitsch & Staniloiu, 2011a). We discussed these mechanisms, which lead to DA, under the heading “INABILITY OF ACCESS HYPOTHESIS” in Markowitsch and Staniloiu (2016).

In Markowitsch and Staniloiu (2016), we also refer to those brain regions which show the main alterations in patients with DA—the anterior temporal cortex (including the amygdala and portions of the hippocampus; Markowitsch, Emmans, Irle, Streicher, & Preilowski, 1985) and the ventrolateral prefrontal cortex of the right hemisphere. This so-called right-hemispheric temporo-frontal complex is active during retrieval of autobiographical memories (Audrain, Gilmore, Wilson, Schacter, & Martin, 2022; Fink et al., 1996; LaBar & Cabeza, 2006) and damage to it leads to retrograde autobiographical amnesia (Calabrese et al., 1996; Kroll, Markowitsch, Knight, & von Cramon, 1997; Markowitsch et al., 1993). A functional imaging study of 14 patients with DA, using [ $^{18}\text{F}$ ] fluorodeoxyglucose positron emission tomography, revealed a hypometabolic zone in exactly this right-hemispheric temporo-frontal area which is active during episodic-autobiographical memory retrieval and which, when damaged, results in amnesia of the personal past (M. Brand et al., 2009). The hypometabolism is shown in fig. 1 of M. Brand et al. (2009) (or in fig. 5 of Staniloiu and Markowitsch (2014) or in fig. 36.4 of Markowitsch and Staniloiu (2016)). These results were confirmed in other studies of patients with DA with the same and different imaging techniques (Glisky et al., 2004; Henning-Fast et al., 2008; J.-C. Mitsui, Oyanagi, Kako, & Kusumi, 2019; Sellal, Manning, Seegmuller, Scheiber, & Schoenfelder, 2002; Tramonì et al., 2009; Yang et al., 2005; for a recent review, see Taïb, Yrond, Lemesle, Péran, & Pariente, 2023). They strongly underline the existence of distinct neuroanatomical correlates of repressed memories or DA. Even if taking into account the argument of Huntjens et al. (2022) (which referred to patients with dissociative identity disorders [DIDs] and to findings of structural brain changes) that patients with DA frequently have comorbidities, the sheer number of patients with corresponding metabolic brain changes, together with the corresponding results from normal individuals (e.g., Fink et al., 1996) and from patients with organic retrograde autobiographical number

(e.g., Kroll et al., 1997) speaks for a likely causal relation with respect to DA. Furthermore, comorbidities are characteristic for patients with DA (Staniloiu & Markowitsch, 2014).

The brain changes observed in patients with DA differ considerably from those found in individuals who simulated (Markowitsch et al., 2000b) or exhibited false memories (Kühnel et al., 2008; Risius et al., 2013).

The hypometabolic changes, which induce a desynchronization between anterior temporal and prefrontal areas (Beblo et al., 2006; Ford et al., 2022; Hanson et al., 2015), speak for a potential recovery from the amnesic state and indeed we found such recovery, which was therapy-induced and resulted in a reversal from a hypometabolic to a normal metabolic brain state (Markowitsch et al., 2000a; see their fig. 1 or fig. 4 in Staniloiu & Markowitsch, 2014) (see also the discussions on such imaging results in Staniloiu & Markowitsch, 2010, and in Staniloiu, Vitcu, & Markowitsch, 2012).

From studying patients with DA, several authors (B. L. Brand, Lanius, Vermetten, Loewenstein, & Spiegel, 2012; Kikuchi et al., 2009, Kunii et al., 2012, MacDonald & MacDonald, 2009) suggested that the prefrontal cortex may exert an inhibiting influence on the hippocampus in patients with DA, leading to memory repression in the sense that the affected individuals are unable to consciously retrieve past experiences with the assumption that these experiences are still present as engrams in the brain. (Evidence for their “unconscious” or repressed presence comes from measuring physiological or autonomic reactions, such as skin conductance changes, when confronted with such information; e.g., Denburg, Jones, & Tranel, 2009; Diamond, Mayes, & Meudell, 1996.)

We conclude, therefore, in line with Cuesta, Cossini, and Politis (2021)—who reviewed the neural basis of DA—that “[t]here is enough evidence to say that DA is an objectifiable biologically based pathology” (p. 11). Their view not only is paralleled by respective statements of others (e.g., MacDonald & MacDonald, 2009), but can also be traced back to corresponding opinions of scientists from the second last century (Flechsig, 1896; Maudsley, 1870; Meynert, 1884). Maudsley, for example, stated that “[m]ental disorders are neither more nor less than nervous diseases in which mental symptoms predominate, and their entire separation from other nervous diseases has been a sad hindrance to progress” (p. 41). (Mai, 1995, cited even earlier work of Briquet, 1859, stating on p. 105 that Briquet “believed that hysteria was caused by physical changes in the brain.”) Markowitsch (1996) has taken-up and expanded this view in a review on organic and functional amnesias.

## 7. The distinct nature of DA

Patients with DA can be distinguished from other individuals with memory problems by a number of features:

1. They usually have no clear structurally measurable brain damage.
2. They do not malingering or feign memory deficits (or at least not predominantly).
3. They predominantly had stress- or trauma-related events in their past.
4. They may show additional symptoms (such as conversion symptoms).

5. The situation leading to DA may be described as “learned helplessness.”
6. The personality of patients with DA can be described as frequently labile and influenceable.
7. There are distinct brain correlates, measurable by functional imaging studies.

1) The patients are clearly distinguishable from neurological patients with memory disturbances after traumatic brain injuries, tumors, degenerative brain diseases, infarcts, and so on. The only similarity to neurological patients stems from individuals with transient global amnesia (TGA), as roughly one-third of these patients are diagnosed with TGA after emotional stress (Bartsch & Deuschl, 2010) and, therefore, after a psychic event (similarly to patients with DA). The distinguishing factors are that TGA primarily results in anterograde amnesia and lasts usually less than a day.

2) There are a number of tests available that allow distinguishing patients with DA from those who malingering retrograde amnesia (Staniloiu, Markowitsch, Schröder, & Kordon, 2023) and tests like the Test of Memory Malinger (TOMM) (Tombaugh, 1996)—which we, together with further tests, usually apply to detect malingerers—have proven their effectiveness in distinguishing fakers from dissociative patients (e.g., Vissia et al., 2016; B. L. Brand, Webermann, Snyder, & Kaliush, 2019). Nevertheless, in rare instances, an overlap may occur (Barbarotto, Laiacina, & Cocchini, 1996; Staniloiu et al., 2018a, 2020b). Otherwise, as mentioned above, there are different patterns of brain activations in individuals who have DA and individuals who simulate (Markowitsch et al., 2000b) or produce false memories (Kühnel et al., 2008; Risius et al., 2013). (However, it still should be mentioned that faking or malinger requires different task demands compared to telling the truth; see, e.g., Gleaves, Smith, Butler, & Spiegel, 2004.)

3) The hypothesis that DA is caused by stress or trauma conditions is incorporated in the definitions of the two main manuals for mental and neurological diseases (DSM-5; ICD-11) and has been favored since long. Sargant and Slater (1941) studied soldiers who mainly had been evacuated from Dunkirk at the beginning of the II. World War. They found in 1000 serial admissions that the more stress the soldiers had experienced, the higher was their rate of being amnesic. Of those having experienced “severe stress,” 35% became amnesic, of those with “moderate stress” 13%, and of those with “trifling stress” only 6%. In more recent years, several overview articles proposed the hypothesis that stressful or traumatic conditions favor the outbreak of DA (B. L. Brand et al., 2012; Dalenberg, 2006; Dalenberg et al., 2012, 2014; Loewenstein, 2018) and our patients generally confirm this view (e.g., Markowitsch et al., 1997a; Markowitsch, Fink, Thöne, Kessler, & Heiss, 1997b, 1998; Markowitsch, Thiel, Kessler, von Stockhausen, & Heiss, 1997c; Markowitsch et al., 2018; Staniloiu et al., 2018, 2020b). Similar findings were also obtained for patients with DID: Vissia et al. (2016), for example, tested several dozen of patients and controls on psychological trauma and fantasy measures and found that the genuine DID patients were not more fantasy-prone or suggestible and did not generate more false memories than the other individuals. It should be emphasized here that what a person interprets as a trauma, differs, based on life experiences and personal resilience (cf. the two-hit hypothesis above and the discussion in Radulovic et al., 2018); nevertheless, causality is difficult to verify.



Table 1  
Stressful life experiences as triggers of DA

Stressful experiences	Trigger for DA
Suppression and humiliation (verbal abuse) by alcohol-dependent parents	Disturbed family life, financial problems
Suppression, humiliation (verbal abuse) and violence by father in childhood	Suicide of husband
Severe stress from early childhood on continuing into fifth decade of life (by parents and partner)	Financial problems
Severe accidents in childhood and youth, violent parents	Falling down a staircase
Violent parents, more than one dozen surgical interventions during the last 14 years	Surgery

Note that the later trigger for DA often in some way resembles or mimics elements of the earlier trauma that likely led to the development of dissociation as a way to cope with severe stress/abuse. Composed after table 3 of Markowitsch (2008).

Typical constellations of stress- and trauma-experiences are the following:

- A young man who at age 23 witnessed the outbreak of an open fire in his house and who—as a bystander with his mother at the age of 4 years—had observed a man burning to death in his car (Markowitsch, Kessler, Van der Ven, Weber-Luxenburger, & Heiss, 1998, 2000a).
- A middle-aged man, who banged with his head against a metal door, (probably) shortly losing consciousness, and thereafter panicking that his car, money, and so on were stolen. This event occurred twice with years apart (Markowitsch & Staniloiu, 2013).
- A middle-aged man grew up as an orphan and was as a child repeatedly sexually abused and was—prior to the outbreak of his DA—threatened to be killed (or at least seriously injured) because of his illegal business (Staniloiu et al., 2018).
- A girl with a multiethnic background who had a head concussion in school due to an accident during gymnastics and later in rehabilitation fell again on her head while walking down stone stairs and thereafter had continuing DA together with a conversion syndrome (inability to move her arm) (Staniloiu et al., 2020b).

Other examples are summarized in Table 1. While such case descriptions are not universally found, absence of evidence is not evidence of absence. It is, by nature, difficult to ask an individual with amnesia about his or her past. Furthermore, there is no normative, qualitative scale of what kinds of events are stressful or not—resilience, personality dimensions, genetic and epigenetic predispositions, and other variables determine what a person experiences as stressful or traumatic (e.g., Barczak-Scarboro et al., 2022; Daskalakis et al., 2021; Fries et al., 2005; Hakamata et al., 2021; Markowitsch, 2015; Mundy et al., 2021; Sep, Joëls, & Geuze, 2022; Womersley et al., 2022; R. Yang et al., 2021; Yehuda et al., 2007; Zannas et al., 2015). (There are, of course, scales on stress and on resilience, however, one cannot say that a given stimulus is stressful as such or not.)

Alternative interpretations of dissociative disorders (“sociocognitive model,” “iatrogenic model,” “fantasy model”; see, e.g., Giesbrecht, Lynn, Lilienfeld, & Merckelbach, 2008) are not in accordance with the great majority of clinical cases with DA (see Loewenstein, 2018; Spiegel et al., 2011; Vissia et al., 2016). For these cases with usually generalized amnesia, alternative interpretations—centering on variables such as spontaneous thoughts, mind wandering, daydreaming, sleep-related versus waking experiences, deficits in metacognitive functions, hyperassociation, emotional alterations—are peripheral for the development of DA (see, e.g., Lynn et al., 2022, for interpretations of disease etiologies in patients with DID or derealization/depersonalization disorders). Of course, as mentioned in part above, additional sociocognitive, cultural, developmental, and personality variables may enforce the outbreak of DAs.

4) As mentioned above, many patients with DA show additional symptoms—from *belle indifference* to paralytic conversion disorders. This cannot astonish, as the disease condition of DA is in itself attributable to a vulnerable personality (see Staniloiu & Markowitsch, 2018a). As we wrote before, experienced stress or trauma are necessary, but not sufficient conditions for the development of DA (Markowitsch & Staniloiu, 2011b). Janet’s (1907) formulation of the mechanism of dissociation as “an inability of the personal self to bind together the various mental component in an integrated whole under its control” (p. 23) characterizes the insufficiency of these individuals which usually is attributable to a number of deviant personality variables and additional disease conditions (for details, see Staniloiu & Markowitsch, 2014, and Markowitsch & Staniloiu, 2016).

5) As mentioned and described above, *learned helplessness* can be seen as the crucial condition, inducing dissociative states, especially as it is accompanied by a number of symptoms and personality traits which characterize individuals with DA (e.g., downregulation of emotions).

6) There are many case reports of patients with DA that describe their lability and suggestibility. In fact, these features may induce the outbreak of the dissociative condition. This is obvious, for example, for the case described by Markowitsch et al. (1999), or by the case in Markowitsch et al. (1997b). The first case was strongly influenced by his wife throughout his married life, and the second case was first influenced by a dominant and authoritarian mother and then by a wife who in many respects resembled his mother. Also, during the time before entering a psychiatric clinic and during his stay there, he was under the direct influence of other persons, who told him to enter the clinic and suggested to him to start a new life in a totally different profession than he had before.

7) As described in detail above, there is now plenty of evidence for distinct brain correlates of DA. What still needs to be established is the time course of the neural changes: It can be assumed that a more transient blockade can result in complete memory recovery (as was the case for the two patients described by Lucchelli, Muggia, & Spinnler, 1995), while prolonged amnesia may permanently alter the neuropil (dendrites and axons) in the affected brain regions and then lead to incomplete or to no recovery at all (we had patients with amnesia lasting for decades). Already in 2009, we (M. Brand et al., 2009) suggested “that retrieval deficits in patients with DA are related to stress-associated dysfunctions in the inferolateral prefrontal section rather than to active and motivated forgetting of memories” (p. 37).

## 8. Conclusions

Repressed memories—defined as DA—are an entity whose existence has to be acknowledged, as other explanations fail to account for this disease which probably existed already in the old Rome as Goldsmith, Cheit, and Wood (2009) remarked, referring to Pliny the Elder (23–79 A.D.), who had mentioned that “fright” constitutes one of the causes of partial or total memory loss. Repression in its plain form is (temporary) forgetting (Brewin, 2021).

## References

- Adolphs, R. (1999). The human amygdala and emotion. *Neuroscientist*, 5, 125–137.
- Anderson, M. C., & Floresco, S. B. (2022). Prefrontal–hippocampal interactions supporting the extinction of emotional memories: The retrieval stopping model. *Neuropsychopharmacology*, 47, 180–195.
- Anderson, M. C., & Hulbert, J. C. (2021). Active forgetting: Adaptation of memory by prefrontal control. *Annual Review of Psychology*, 72, 1–36.
- Audrain, S., Gilmore, A. W., Wilson, J. M., Schacter, D. L., & Martin, A. (2022). A role for the anterior hippocampus in autobiographical memory construction regardless of temporal distance. *Journal of Neuroscience*, 42, 6445–6452.
- Barbarotto, R., Laiacina, M., & Cocchini, G. (1996). A case of simulated, psychogenic or focal pure retrograde amnesia did an entire life become unconscious? *Neuropsychologia*, 34, 575–585.
- Barczak-Scarboro, N. E., Roby, P. R., Kiefer, A. W., Bailar-Heath, M., Burke, R. J., DeLellis, S. M., Kane, S. F., Lynch, J. H., Means, G. E., Depenbrock, P. J., & Mihalik, J. P. (2022). The relationship between resilience and neurophysiological stress in Special Operations Forces combat service members. *European Journal of Neuroscience*, 55, 2804–2812.
- Bartsch, T., & Deuschl, G. (2010). Transient global amnesia: Functional anatomy and clinical implications. *Lancet Neurology*, 9, 205–214.
- Bauer, J. (1917). Hysterische Erkrankungen bei Kriegsteilnehmern [Hysterical diseases in war participants]. *Archiv für Psychiatrie und Nervenkrankheiten*, 57, 139–168.
- Beblo, T., Woermann, F. G., Mertens, M., Wingenfeld, K., Piefke, M., Rullkoetter, N., Silva-Saavedra, A., Mensebach, C., Reddemann, L., Rau, H., Markowitsch, H. J., Wulff, H., Lange, W., Berea, D., Ollech, I., & Driessen, M. (2006). Functional MRI correlates of the recall of unresolved life events in borderline personality disorder. *Psychological Medicine*, 36, 845–856.
- Becquet, C., Cogez, J., Dayan, J., Lebaï, P., Viader, F., Eustache, F., & Quinette, P. (2021). Episodic autobiographical memory impairment and differences in pronoun use: Study of self-awareness in functional amnesia and transient global amnesia. *Frontiers in Psychology*, 12, 624010. <https://doi.org/10.3389/fpsyg.2021.624010>
- Bettelheim, B. (1950). *Love is not enough: The treatment of emotionally disturbed children*. Glencoe, IL: Free Press.
- Brand, B. L., Lanius, R., Vermetten, E., Loewenstein, R. J., & Spiegel, D. (2012). Where are we going? An update on assessment, treatment, and neurobiological research in dissociative disorders as we move toward the *DSM-5*. *Journal of Trauma & Dissociation*, 13, 9–31.
- Brand, B. L., Webermann, A., Snyder, B., & Kaliush, P. (2019). Detecting clinical and simulated dissociative identity disorder with the Test of Memory Malinger. *Psychological Trauma: Theory, Research, Practice, and Policy*, 11, 513–520.
- Brand, M., Eggers, C., Reinhold, N., Fujiwara, E., Kessler, J., Heiss, W.-D., & Markowitsch, H. J. (2009). Functional brain imaging in fourteen patients with dissociative amnesia reveals right inferolateral prefrontal hypometabolism. *Psychiatry Research: Neuroimaging Section*, 174, 32–39.
- Bremner, J. D., Randall, P., Scott, T. M., Bronen, R. A., Seibyl, J. P., Southwick, S. M., Delaney, R. C., McCarthy, G., Charney, D. S., & Innis, R. B. (1995). MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. *American Journal of Psychiatry*, 152, 973–981.

- Bremner, J. D., & Wittbrodt, M. T. (2020). Stress, the brain, and trauma spectrum disorders. *International Review of Neurobiology*, 152, 1–22.
- Breuer, J., & Freud, S. (1895). *Studien über Hysterie* [Studies on hysteria]. Wien: Deuticke.
- Brewin, C. R. (2021). Tilting at windmills: Why attacks on repression are misguided. *Perspectives on Psychological Science*, 16, 443–453.
- Briquet, P. (1859). *Traité clinique et thérapeutique de l'hystérie* [Clinical and therapeutical discourse on hysteria]. Paris: J.-B. Baillière et Fils.
- Bryant, R. A. (2022). Post-traumatic stress disorder as moderator of other mental health conditions. *World Psychiatry*, 21, 310–311.
- Caetano, I., Amorim, L., Castanho, T. C., Coelho, A., Ferreira, S., Portugal-Nunes, C., Soares, J. M., Gonçalves, N., Sousa, R., Reis, J., Lima, C., Marques, P., Silva Moreira, P., João Rodrigues, A., Correia Santos, N., Morgado, P., Esteves, M., Magalhães, R., Picó-Pérez, M., & Sousa, N. (2022). Association of amygdala size with stress perception: Findings of a transversal study across the lifespan. *European Journal of Neuroscience*, 56, 5287–5298.
- Cahill, L., Babinsky, R., Markowitsch, H. J., & McGaugh, J. L. (1995). Involvement of the amygdaloid complex in emotional memory. *Nature*, 377, 295–296.
- Calabrese, P., Markowitsch, H. J., Durwen, H. F., Widlitzek, B., Haupts, M., Holinka, B., & Gehlen, W. (1996). Right temporofrontal cortex as critical locus for the ecphory of old episodic memories. *Journal of Neurology, Neurosurgery, and Psychiatry*, 61, 304–310.
- Cuesta, C., Cossini, F. C., & Politis, D. G. (2021). Las bases neurales de la Amnesia Disociativa (AD): Una revisión sistemática de la bibliografía [Systematic revision of the neural bases of dissociative amnesia]. *Vertex*, 32, 11–16.
- Dalenberg, C. (2006). Recovered memory and the Daubert criteria. Recovered memory as professionally tested, peer reviewed, and accepted in the relevant scientific community. *Trauma, Violence, & Abuse*, 7, 274–310.
- Dalenberg, C. J., Brand, B. L., Gleaves, D. H., Dorahy, M. J., Loewenstein, R. J., Cardeña, E., Frewen, P. A., Carlson, E. B., & Spiegel, D. (2012). Evaluation of the evidence for the trauma and fantasy models of dissociation. *Psychological Bulletin*, 138, 150–188.
- Dalenberg, C. J., Brand, B. L., Loewenstein, R. J., Gleaves, D. H., Dorahy, M. J., Cardeña, E., Frewen, P. A., Carlson, E. B., & Spiegel, D. (2014). Reality versus fantasy: Reply to Lynn et al. (2014). *Psychological Bulletin*, 140, 911–920.
- Daskalakis, N. P., Xu, C., Bader, H. N., Chatzinakos, C., Weber, P., Makotkine, I., Lehrner, A., Bierer, L. M., Binder, E. B., & Yehuda, R. (2021). Intergenerational trauma is associated with expression alterations in glucocorticoid- and immune-related genes. *Neuropsychopharmacology*, 46, 763–773.
- Denburg, N. L., Jones, R. D., & Tranel, D. (2009). Recognition without awareness in a patient with simultanagnosia. *International Journal of Psychophysiology*, 72, 5–12.
- DePrince, A. P., & Freyd, J. J. (1997). Do what's the dispute about? *Judges' Journal*, 36, 70–72.
- Diamond, B. J., Mayes, A. R., & Meudell, P. R. (1996). Autonomic and recognition indices of memory in amnesic and healthy control subjects. *Cortex*, 32, 439–459.
- Dodier, O., Gilet, A.-L., & Colombel, F. (2022). What do people really think of when they claim to believe in repressed memory? Methodological middle ground and applied issues. *Memory*, 30, 744–752.
- DSM-5 (American Psychiatric Association, Ed.). (2013). *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)* (5th ed.). Arlington, VA: American Psychiatric Publishing.
- Erdelyi, M. H. (2006). The unified theory of repression. *Behavioral and Brain Sciences*, 29, 499–551.
- Fink, G. R., Markowitsch, H. J., Reinkemeier, M., Bruckbauer, T., Kessler, J., & Heiss, W.-D. (1996). Cerebral representation of one's own past: neural networks involved in autobiographical memory. *Journal of Neuroscience*, 16, 4275–4282.
- Fleischig, P. (1896). *Gehirn und Seele*. Leipzig: Veit & Comp.
- Ford, J. H., Kim, S. Y., Kark, S. M., Daley, R. T., Payne, J. D., & Kensinger, E. A. (2022). Distinct stress-related changes in intrinsic amygdala connectivity predict subsequent positive and negative memory performance. *European Journal of Neuroscience*, 56, 4744–4765.

- Freud, S. (1895/1950). *Aus den Anfängen der Psychoanalyse. Anhang I: Entwurf einer Psychologie*. [The origins of psychoanalysis. Appendix I: Project of a psychology]. London: Imago Publishing.
- Freud, S. (1898). Zum psychischen Mechanismus der Vergesslichkeit. *Monatsschrift für Psychiatrie und Neurologie*, 1, 436–443.
- Freud, S. (1899). Ueber Deckerinnerungen. *Monatsschrift für Psychiatrie und Neurologie*, 2, 215–230.
- Fries, A. B., Ziegler, T. E., Kurian, J. R., Jacoris, S., & Pollak, S. D. (2005). Early experience in humans is associated with changes in neuropeptides critical for regulating social behavior. *Proceedings of the National Academy of Sciences of the United States of America*, 102, 17237–17240.
- Fujiwara, E., Brand, M., Kracht, L., Kessler, J., Diebel, A., Netz, J., & Markowitsch, H.J. (2008). Functional retrograde amnesia: A multiple case study. *Cortex*, 44, 29–45.
- Giesbrecht, T., Lynn, S. J., Lilienfeld, S. O., & Merckelbach, H. (2008). Cognitive processes in dissociation: An analysis of core theoretical assumptions. *Psychological Bulletin*, 134, 617–647.
- Gleaves, D. H., Smith, S. M., Butler, L. D., & Spiegel, D. (2004). False and recovered memories in the laboratory and clinic: A review of experimental and clinical evidence. *Clinical Psychology: Science and Practice*, 11, 3–28.
- Glisky, E. L., Ryan, L., Reminger, S., Hardt, O., Hayes, S. M., & Hupbach, A. (2004). A case of psychogenic fugue: I understand, aber ich verstehe nichts. *Neuropsychologia*, 42, 1132–1147.
- Goldsmith, R. E., Cheit, R. E., & Wood, M. E. (2009). Evidence of dissociative amnesia in science and literature: Culture-bound approaches to trauma in Pope, Poliakoff, Parker, Boynes, & Hudson (2007). *Journal of Trauma and Dissociation*, 10, 237–253.
- Hartmann, H. (1930). Gedächtnis und Lustprinzip. Untersuchungen an Korsakoffkranken. *Zeitschrift für die gesamte Neurologie und Psychiatrie*, 126, 496–519.
- Hakamata, Y., Mizukami, S., Izawa, S., Moriguchi, Y., Hori, H., Matsumoto, N., Hanakawa, T., Inoue, Y., & Tagaya, H. (2021). Childhood trauma affects autobiographical memory deficits through basal cortisol and prefrontal-extrastriate functional connectivity. *Psychoneuroendocrinology*, 127, 105172. <https://doi.org/10.1016/j.psyneuen.2021.105172>
- Hanson, J. L., Nacewicz, B. M., Sutterer, M. J., Cayo, A. A., Schaefer, S. M., Rudolph, K. D., Shirtcliff, E. A., Pollak, S. D., & Davidson, R. J. (2015). Behavioral problems after early life stress: Contributions of the hippocampus and amygdala. *Biological Psychiatry*, 77, 314–323.
- Harrison, P., Lawrence, A. J., Wang, S., Liu, S., & Xie, G., Yang, X., & Zahn, R. (2022). The psychopathology of worthlessness in depression. *Frontiers in Psychiatry*, 13, 818542. <https://doi.org/10.3389/fpsy.2022.818542>
- Henning-Fast, K., Meister, F., Frodl, T., Beraldi, A., Padberg, F., Engel, R. R., Reiser, M., Möller, H.-J., & Meindl, T. (2008). A case of persistent retrograde amnesia following a dissociative fugue: Neuropsychological and neurofunctional underpinnings of loss of autobiographical memory and self-awareness. *Neuropsychologia*, 46, 2993–3005.
- Huntjens, R. J. C., Otgaar, H., Pijnenborg, G. H. M., & Wessel, I. (2022). The elusive search for a biomarker of dissociative amnesia: A reaction to Dimitrova et al. (2022). *Psychological Medicine*, 52, 2835–2836 <https://doi.org/10.1017/S0033291722001118>
- ICD-11. (2021). *International Classification of Diseases for Mortality and Morbidity Statistics* (11th rev.). Geneva: World Health Organization.
- Janet, P. (1892). *L'état mental des hystériques (Vol. 1: Les stigmates mentaux)* (Vol. 1). [The mental state of the hysterics (Vol. The mental stigmata)]. Paris: Rueff.
- Janet, P. (1893). L'amnésie continue [Continuing amnesia]. *Revue generale des Sciences*, 4, 167–179.
- Janet, P. (1907). *The major symptoms of hysteria*. New York: Macmillan.
- Jung, C. G. (1905). Experimentelle Beobachtungen über das Erinnerungsvermögen. *Centralblatt für Nervenheilkunde und Psychiatrie*, 28, 653–666.
- Kikuchi, H., Fujii, T., Abe, N., Suzuki, M., Takagi, M., Mugikura, S., Takahashi, S., & Mori, E. (2009). Memory repression: Brain mechanisms underlying dissociative amnesia. *Journal of Cognitive Neuroscience*, 22, 602–613.

- Kroll, N., Markowitsch, H. J., Knight, R., & von Cramon, D. Y. (1997). Retrieval of old memories – The temporo-frontal hypothesis. *Brain*, 120, 1377–1399.
- Kühnel, S., Woermann, F. G., Mertens, M., & Markowitsch, H. J. (2008). Involvement of the orbitofrontal cortex during correct and false recognitions of visual stimuli. Implications for eyewitness decisions on an fMRI study using a film paradigm. *Brain Imaging and Behavior*, 2, 163–176.
- Kunii, Y., Okano, T., Mashiko, H., Yabe, H., & Niwa, S. I. (2012). Serial changes in cerebral blood flow single photon emission computed tomography findings during memory retrieval in a case of psychogenic amnesia. *Psychiatry and Clinical Neuroscience*, 66, 623–624.
- Kunzendorf, R. K., & Moran, C. (1993/94). Repression: Active censorship of stressful memories vs. source amnesia for self-consciously dissociated memories. *Imagination, Cognition and Personality*, 13, 291–302.
- LaBar, K. S., & Cabeza, R. (2006). Cognitive neuroscience of emotional memory. *Nature Reviews Neuroscience*, 7, 54–64.
- Langer, K. G. (2019). Early history of amnesia. *Frontiers in Neurology and Neuroscience*, 44, 64–74.
- Langnickel, R., & Markowitsch, H. J. (2006). Repression and the unconsciousness. *Behavioral and Brain Sciences*, 29, 524–525.
- Lassri, D., Bregman-Hai, N., Soffer-Dudek, N., & Shahar, G. (2023). The interplay between childhood sexual abuse, self-concept clarity, and dissociation: A resilience-based perspective. *Journal of Interpersonal Violence*, 38, 2313–2336. <https://doi.org/10.1177/08862605221101182>
- Lippard, E. T. C., & Nemeroff, C. B. (2020). The devastating clinical consequences of child abuse and neglect: Increased disease vulnerability and poor treatment response in mood disorders. *American Journal of Psychiatry*, 177, 20–36.
- Loewenstein, R. J. (2018). Dissociation debates: Everything you know is wrong. *Dialogues in Clinical Neuroscience*, 20, 229–242.
- Loftus, E. (1993). The reality of repressed memories. *American Psychologist*, 48, 518–537.
- Loftus, E. (1994). The repressed memory controversy. *American Psychologist*, 49, 443–445.
- Loftus, E. F. (2005). Planting misinformation in the human mind: A 30-year investigation of the malleability of memory. *Learning & Memory*, 12, 361–366.
- Loftus, E. F., & Burns, T. E. (1982). Mental shock can produce retrograde amnesia. *Memory & Cognition*, 10, 318–323.
- Lucchelli, F., Muggia, S., & Spinnler, H. (1995). The ‘Petites Madeleines’ phenomenon in two amnesic patients: Sudden recovery of forgotten memories. *Brain*, 118, 167–183.
- Lupien, S. J., Juster, R.-P., Raymond, C., & Marin, M.-F. (2018). The effects of chronic stress on the human brain: From neurotoxicity, to vulnerability, to opportunity. *Frontiers in Neuroendocrinology*, 49, 91–105.
- Lynn, S. J., Polizzi, C., Merckelbach, H., Chiu, C.-D., Maxwell, R., van Heugten, D., & Lilienfeld, S. O. (2022). Dissociation and dissociative disorders reconsidered: Beyond sociocognitive and trauma models toward a trans-theoretical framework. *Annual Review of Clinical Psychology*, 18, 259–289.
- MacDonald, K., & MacDonald, T. (2009). Peas, please: A case report and neuroscientific review of dissociative amnesia and fugue. *Journal of Trauma & Dissociation*, 10, 420–435.
- Mai, F. M. (1995). “Hysteria” in clinical neurology. *Canadian Journal of the Neurological Sciences*, 22, 101–110.
- Markowitsch, H. J. (1996). Organic and psychogenic retrograde amnesia: Two sides of the same coin? *Neurocase*, 2, 357–371.
- Markowitsch, H. J. (2000). Repressed memories. In E. Tulving (Ed.), *Memory, consciousness, and the brain: The Tallinn conference* (pp. 319–330). Philadelphia, PA: Psychology Press.
- Markowitsch, H. J. (2002). Functional retrograde amnesia – Mnestic block syndrome. *Cortex*, 38, 651–654.
- Markowitsch, H. J. (2003). Psychogenic amnesia. *Neuroimage*, 20, S132–S138.
- Markowitsch, H. J. (2008). Gedächtnis und Brain Imaging [Memory and brain imaging]. *Fortschritte der Neurologie Psychiatrie*, 76(Suppl. 1), S3–S7.
- Markowitsch, H. J. (2015). Dissoziative Amnesien – ein Krankheitsbild mit wahrscheinlicher epigenetischer Komponente [Dissociative amnesia – A clinical picture with a probable epigenetic component]. *Persönlichkeitsstörungen*, 19, 1–16.

- Markowitsch, H. J., Calabrese, P., Fink, G. R., Durwen, H. F., Kessler, J., Härting, C., König, M., Mirzaian, E. B., Heiss, W.-D., Heuser, L., & Gehlen, W. (1997a). Impaired episodic memory retrieval in a case of probable psychogenic amnesia. *Psychiatry Research: Neuroimaging Section*, 74, 119–126.
- Markowitsch, H. J., Calabrese, P., Liess, J., Haupts, M., Durwen, H. F., & Gehlen, W. (1993). Retrograde amnesia after traumatic injury of the temporo-frontal cortex. *Journal of Neurology, Neurosurgery and Psychiatry*, 56, 988–992.
- Markowitsch, H. J., Emmans, D., Irle, E., Streicher, M., & Preilowski, B. (1985). Cortical and subcortical afferent connections of the primate's temporal pole: A study of rhesus monkeys, squirrel monkeys, and marmosets. *Journal of Comparative Neurology*, 242, 425–458.
- Markowitsch, H. J., Fink, G. R., Thöne, A. I. M., Kessler, J., & Heiss, W.-D. (1997b). A PET study of persistent psychogenic amnesia covering the whole life span. *Cognitive Neuropsychiatry*, 2, 135–158.
- Markowitsch, H. J., Kessler, J., Russ, M. O., Frölich, L., Schneider, B., & Maurer, K. (1999). Mnestic block syndrome. *Cortex*, 35, 219–230.
- Markowitsch, H. J., Kessler, J., Van der Ven, C., Weber-Luxenburger, G., & Heiss, W.-D. (1998). Psychic trauma causing grossly reduced brain metabolism and cognitive deterioration. *Neuropsychologia*, 36, 77–82.
- Markowitsch, H. J., Kessler, J., Weber-Luxenburger, G., Van der Ven, C., Albers, M., & Heiss, W. D. (2000a). Neuroimaging and behavioral correlates of recovery from mnestic block syndrome and other cognitive deteriorations. *Neuropsychiatry, Neuropsychology and Behavioral Neurology*, 13, 60–66.
- Markowitsch, H. J., & Staniloiu, A. (2011a). Amygdala in action: Relaying biological and social significance to autobiographic memory. *Neuropsychologia*, 49, 718–733.
- Markowitsch, H. J., & Staniloiu, A. (2011b). Memory, autooetic consciousness, and the self. *Consciousness and Cognition*, 20, 16–39.
- Markowitsch, H. J., & Staniloiu, A. (2013). The impairment of recollection in functional amnesic states. *Cortex*, 49, 1494–1510.
- Markowitsch, H. J., & Staniloiu, A. (2016). Functional (dissociative) retrograde amnesia. In M. Hallett, J. Stone, & A. Carson (Eds.), *Handbook of clinical neurology (3rd series): Functional neurological disorders* (pp. 419–445). Amsterdam: Elsevier.
- Markowitsch, H. J., & Staniloiu, A. (2022). Behavioral, neurological and psychiatric frailty of autobiographical memory. *WIREs Cognitive Science*, e1617, 1–27. <https://doi.org/10.1002/wcs.1617>
- Markowitsch, H. J., Staniloiu, A., Kordon, A., & Sarlon, J. (2018). Minor brain damage and somatic complaints accompanied by excessive long-term amnesia: Psychological cause? In C. Pracana & M. Wang (Eds.), *Psychological applications and trends 2018* (pp. 34–38). Lisbon: InScience Press.
- Markowitsch, H. J., Thiel, A., Kessler, J., von Stockhausen, H.-M., & Heiss, W.-D. (1997c). Ecphorizing semi-conscious episodic information via the right temporopolar cortex – A PET study. *Neurocase*, 3, 445–449.
- Markowitsch, H. J., Thiel, A., Reinkemeier, M., Kessler, J., Koyuncu, A., & Heiss, W.-D. (2000b). Right amygdalar and temporofrontal activation during autobiographic, but not during fictitious memory retrieval. *Behavioural Neurology*, 12, 181–190.
- Maudsley, H. (1870). *Body and mind: An inquiry into their connection and mutual influence, specially in reference to mental disorders*. London: Macmillan and Co.
- Mehta, M. A., Golembo, N. I., Nosarti, C., Colvert, E., Mota, A., Williams, S. C. R., Rutter, M., & Sonuga-Barke, E. J. S. (2009). Amygdala, hippocampal and corpus callosum size following severe early institutional deprivation: The English and Romanian Adoptees Study Pilot. *Journal of Child Psychology and Psychiatry*, 50, 943–951.
- Meine, L. E., Meier, J., Meyer, B., & Wessa, M. (2021). Don't stress, it's under control: Neural correlates of stressor controllability in humans. *Neuroimage*, 245, 118701. <https://doi.org/10.1016/j.neuroimage.2021.118701>
- Meynert, T. (1884). *Psychiatrie. Klinik der Erkrankungen des Vorderhirns, begründet auf dessen Bau, Leistungen und Ernährung* [Psychiatry. Clinic of diseases of the forebrain, based on its construction, performance and nutrition]. Vienna: Braumüller.

- Mitsui, N., Oyanagi, Y., Kako, Y., & Kusumi, I. (2019). Natural recovery from long-lasting generalised dissociative amnesia and of cerebral blood flow. *British Medical Journal: Case Reports*, 12, e231270. <https://doi.org/10.1136/bcr-2019-231270>
- Mundy, J., Hübel, C., Gelernter, J., Levey, D., Murray, R. M., Skelton, M., Stein, M. B., Vassos, E., Breen, B., Colema, J. R. I., & Million Veteran Program, Post Traumatic Stress Disorder Working Group of the Psychiatric Genomics Consortium. (2021). Psychological trauma and the genetic overlap between posttraumatic stress disorder and major depressive disorder. *Psychological Medicine*, 52, 1–10. <https://doi.org/10.1017/S0033291721000830>
- O'Brien, J. T. (1997). The 'glucocorticoid cascade' hypothesis in man. *British Journal of Psychiatry*, 170, 199–201.
- Otgaar, H., Howe, M. L., Patihis, L., Merckelbach, H., Lynn, S. J., Lilienfeld, S. O., & Loftus, E. F. (2019). The return of the repressed: The persistent and problematic claims of long-forgotten trauma. *Perspectives on Psychological Science*, 14, 1072–1095.
- Peper, M., & Markowitsch, H. J. (2001). Pioneers of affective neuroscience and early conceptions of the emotional brain. *Journal of the History of Neuroscience*, 10, 58–66.
- Pope Jr., H. G., Schnabel, J., & Hudson, J. I. (2023). Current scientific interest in dissociative amnesia: A bibliometric analysis. *Applied Cognitive Psychology*, 37, 42–51.
- Radulovic, J., Lee, R., & Ortony, A. (2018). State-dependent memory: Neurobiological advances and prospects for translation to dissociative amnesia. *Frontiers in Behavioral Neuroscience*, 12, 259. <https://doi.org/10.3389/fnbeh.2018.00259>
- Reinhold, N., & Markowitsch, H. J. (2009). Retrograde episodic memory and emotion: A perspective from patients with dissociative amnesia. *Neuropsychologia*, 47, 2197–2206.
- Risius, U.-M., Staniloiu, A., Piefke, M., Maderwald, S., Schulte, F., Brand, M., & Markowitsch, H. J. (2013). Retrieval, monitoring and control processes: A 7 Tesla fMRI approach to memory accuracy. *Frontiers in Behavioral Neuroscience*, 7, 1–21.
- Ross, E. D., Homan, R., & Buck, R. (1994). Differential hemispheric lateralization of primary and social emotions: Implications for developing a comprehensive neurology for emotions, repression, and the subconscious. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, 7, 1–19.
- Sargant, W., & Slater, E. (1941). Amnesic syndromes in war. *Proceedings of the Royal Society of Medicine*, 34, 757–764.
- Schauer, E., & Elbert, T. (2010). The psychological impact of child soldiering. In E. Martz (Ed.), *Trauma rehabilitation after war and conflict* (pp. 311–360). New York: Springer.
- Schwartz, B. L., & Metcalfe, J. (2011). Tip-of-the-tongue (TOT) states: Retrieval, behavior, and experience. *Memory & Cognition*, 39, 737–749.
- Seligman, M. E. (1972). Learned helplessness. *Annual Review of Medicine*, 23, 407–412.
- Sellal, F., Manning, L., Seegmuller, C., Scheiber, C., & Schoenfelder, F. (2002). Pure retrograde amnesia following a mild head trauma: A neuropsychological and metabolic study. *Cortex*, 38, 499–509.
- Semon, R. (1904). *Die Mneme als erhaltendes Prinzip im Wechsel des organischen Geschehens*. Leipzig: Wilhelm Engelmann.
- Sep, M. S. C., Joëls, M., & Geuze, E. (2022). Individual differences in the encoding of contextual details following acute stress: An explorative study. *European Journal of Neuroscience*, 55, 2714–2738.
- Shansky, R. M., & Lipps, J. (2013). Stress-induced cognitive dysfunction: Hormone–neurotransmitter interactions in the prefrontal cortex. *Frontiers in Human Neuroscience*, 7, 1–6. <https://doi.org/10.3389/fnhum.2013.00123>
- Spiegel, D., Loewenstein, R. J., Lewis-Fernandez, R., Sar, V., Simeon, D., Vermetten, E., Cardeña, E., & Dell, P. F. (2011). Dissociative disorders in DSM-5. *Depression & Anxiety*, 28, 824–852.
- Staniloiu, A., Kordon, A., & Markowitsch, H. J. (2020a). Quo vadis episodic memory – Past, present, perspective. *Neuropsychologia*, 141, 107362. <https://doi.org/10.1016/j.neuropsychologia.2020.107362>
- Staniloiu, A., Kordon, A., & Markowitsch, H. J. (2020b). Stress- and trauma-related blockade of episodic-autobiographical memory processing. *Neuropsychologia*, 139, 107364. <https://doi.org/10.1016/j.neuropsychologia.2020.107364>



- Staniloiu, A., & Markowitsch, H. J. (2010). Searching for the anatomy of dissociative amnesia. *Journal of Psychology*, 218, 96–108.
- Staniloiu, A., & Markowitsch, H. J. (2012). Towards solving the riddle of forgetting in functional amnesia: Recent advances and current opinions. *Frontiers in Psychology*, 3, 403.
- Staniloiu, A., & Markowitsch, H. J. (2014). Dissociative amnesia. *Lancet Psychiatry*, 1, 226–241.
- Staniloiu, A., & Markowitsch, H. J. (2018). Dissociative amnesia – A challenge to therapy. *International Journal of Psychotherapy Practice and Research*, 1, 34–47. doi: 10.14302/issn.2574-612X.ijpr-18-2246
- Staniloiu, A., & Markowitsch, H. J. (2018b). Dissociative disorders and their clinical management (Part one: Dissociative amnesia (including its variant dissociative fugue)). *Scientific American Psychiatry*, <https://doi.org/10.2310/PSYCH.13031>
- Staniloiu, A., & Markowitsch, H. J. (2022). The fragility of remembering – Data from clinical cases. In C. Pracana & M. Wang (Eds.), *Psychological applications and trends* (pp. 129–133). Lisbon, Portugal: InScience Press.
- Staniloiu, A., Markowitsch, H. J., & Kordon, A. (2018). Psychological causes of amnesia: A study of 28 cases. *Neuropsychologia*, 110, 134–147.
- Staniloiu, A., Markowitsch, H. J., Schröder, J., & Kordon, A. (2023). Measures of episodic memory. In G. J. Boyle, Y. Stern, D. Stein, & B. Sahakian (Eds.), *The SAGE Handbook of Clinical Neuropsychology (Volume 2)* (pp. 162–178). London: Sage Publications.
- Staniloiu, A., Vitcu, I., & Markowitsch, H. J. (2012). Neuroimaging and dissociative disorders. In V. Chaudhary (Ed.), *Advances in brain imaging* (pp. 11–34). INTECH – Open Access Publication.
- Suarez, J. M., & Pittluck, A. T. (1975). Global amnesia: Organic and functional considerations. *Bulletin of the American Academy of Psychiatry and the Law*, 3, 17–24.
- Sutin, A. R., Luchetti, M., Aschwanden, D., Stephan, Y., & Terracciano, A. (2021). Sense of purpose in life, cognitive function, and the phenomenology of autobiographical memory. *Memory*, 29, 1126–1135.
- Taïb, S., Yrond, A., Lemesle, B., Péran, P., & Pariente, J. (2023). What are the neural correlates of dissociative amnesia? A systematic review of the functional neuroimaging literature. *Frontiers in Psychiatry*, 14, 1092826. <https://doi.org/10.3389/fpsy.2023.1092826>
- Tombaugh, T. N. (1996). *Test of Memory Malingering (TOMM)*. New York: Multi Health Systems.
- Tramoni, E., Aubert-Khalifa, S., Guye, M., Ranjeva, J. P., Felician, O., & Ceccaldi, M. (2009). Hypo-retrieval and hyper-suppression mechanisms in functional amnesia. *Neuropsychologia*, 47, 611–624.
- Tulving, E. (1983). *Elements of episodic memory*. Oxford: Oxford University Press.
- Tulving, E. (1985). Memory and consciousness. *Canadian Psychologist*, 26, 1–12.
- Tulving, E. (1995). Organization of memory: Quo vadis? In M. S. Gazzaniga (Ed.), *The cognitive neurosciences* (pp. 839–847). Cambridge, MA: MIT Press.
- Tulving, E. (2002). Episodic memory: From mind to brain. *Annual Review of Psychology*, 53, 1–25.
- Tulving, E. (2005). Episodic memory and autonoesis: Uniquely human? In H. Terrace & J. Metcalfe (Eds.), *The missing link in cognition: Evolution of self knowing consciousness* (pp. 3–56). New York: Oxford University Press.
- Tulving, E., & Markowitsch, H. J. (1998). Episodic and declarative memory: Role of the hippocampus. *Hippocampus*, 8, 198–204.
- Tulving, E., & Thompson, D. M. (1973). Encoding specificity and retrieval process in episodic memory. *Psychological Review*, 80, 352–373.
- Vissia, E. M., Giesen, M. E., Chalavi, S., Nijenhuis, E. R. S., Draijer, N., Brand, B. L., & Reinders, A. A. T. S. (2016). Is it trauma- or fantasy-based? Comparing dissociative identity disorder, post-traumatic stress disorder, simulators, and controls. *Acta Psychiatrica Scandinavica*, 134, 111–128.
- Wang, H., van Leeuwen, J. M. C., de Voogd, L. D., Verkes, R. J., Roozendaal, B., Fernandez, G., & Hermans, E. J. (2022). Mild early-life stress exaggerates the impact of acute stress on corticolimbic resting-state functional connectivity. *European Journal of Neuroscience*, 55, 2122–2141.
- Watson, D., Levin-Aspenson, H.-F., Waszczuk, M. A., Conway, C. C., Dalgleish, T., Dretsch, M. N., Eaton, N. R., Forbes, M. K., Forbush, K. T., Hobbs, K. A., Michelini, G., Nelson, B. D., Sellbom, M., Slade, T., South, S. C., Sunderland, M., Waldman, I., Withöft, M., Wright, A. G. C., Kotov, R., Krueger, R. F., & HiTOP Utility

- Workgroup. (2022). Validity and utility of hierarchical taxonomy of psychopathology (HiTOP): III. Emotional dysfunction superspectrum. *World Psychiatry*, 21, 26–54.
- Weissman, D. G., Lambert, H. K., Rodman, A. M., Peverill, M., Sheridan, M. A., & McLaughlin, K. A. (2022). Reduced hippocampal and amygdala volume as a mechanism underlying stress sensitization to depression following childhood trauma. *Depression and Anxiety*, 37, 916–925.
- Williams, J. M. G., Barnhofer, T., Crane, C., Hermans, D., Raes, F., Watkins, E., & Dalgleish, T. (2007). Autobiographical memory specificity and emotional disorder. *Psychological Bulletin*, 133, 122–148.
- Wixted, J. T., Mickes, L., & Fisher, R. P. (2018). Rethinking the reliability of eyewitness memory. *Perspectives on Psychological Science*, 13, 324–335.
- Womersley, J. S., Nothling, J., Toikumo, S., Malan-Müller, S., van den Heuvel, L. L., McGregor, N. W., Seedat, S., & Hemmings, S. M. J. (2022). Childhood trauma, the stress response and metabolic syndrome: A focus on DNA methylation. *European Journal of Neuroscience*, 55, 2253–2296.
- Yang, J.-C., Jeong, G.-W., Lee, M.-S., Kang, H.-K., Eun, S.-J., Kim, Y.-K., & Lee, Y.-H. (2005). Functional MR imaging of psychogenic amnesia: A case report. *Korean Journal of Radiology*, 6, 196–199.
- Yang, R., Gautam, A., Getnet, D., Daigle, B. J., Miller, S., Misganaw, B., Dean, K. R., Kumar, R., Muhie, S., Wang, K., Lee, I., Abu-Amara, D., Flory, J. D., Hood, L., Wolkowitz, O. M., Mellon, S. H., Doyle 3rd, F. J., Yehuda, R., Marmar, C. R., Ressler, K. J., Hammamieh, R., Jett, M., & PTSD Systems Biology Consortium. (2021). Epigenetic biotypes of post-traumatic stress disorder in war-zone exposed veteran and active duty males. *Molecular Psychiatry*, 26, 4300–4314.
- Yang, Y., Ma, X., Kelifa, M. O., Li, X., Chen, Z., & Wang, P. (2022). The relationship between childhood abuse and depression among adolescents: The mediating role of school connectedness and psychological resilience. *Child Abuse and Neglect*, 131, 105760. <https://doi.org/10.1016/j.chiabu.2022.105760>
- Yehuda, R., Teicher, M. H., Seckl, J. R., Grossman, R. A., Morris, A., & Bierer, L. M. (2007). Parental posttraumatic stress disorder as a vulnerability factor for low cortisol trait in offspring of holocaust survivors. *Archives of General Psychiatry*, 64, 1040–1048.
- Zannas, A. S., Provençal, N., & Binder, E. B. (2015). Epigenetics of posttraumatic stress disorder: Current evidence, challenges, and future. *Biological Psychiatry*, 78, 327–335.