MAJOR REVIEW

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BIOLOGICAL BASES OF DISSOCIATIVE AMNESIA

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SUMMARY

Dissociative amnesia is defined as the retrograde memory loss in the absence of detectable structural brain damage caused by disassociation. Although the classification criteria of dissociative amnesia are based solely on the clinical image and do not refer to biological mechanisms, recent neurobiological studies using applied modern brain imaging techniques suggest that biological mechanisms play a crucial role in this disorder. Research on this phenomenon indicated that the main biological factors that trigger dissociative amnesia are an excessive arousal of the prefrontal cortex, hippocampus and amygdalae. The prefrontal cortex is a structure, which integrates internal and external experience. In the case of an excessive arousal, its functions become deregulated, which results in the inability to register the received stimuli. The hippocampus is a very flexible structure, yet highly vulnerable. As a result of the exposure to stress factors, stress hormones are secreted, which leads to an arousal of the hippocampus. Too rapid or too long secretion of stress hormones may lead to an excessive arousal of the hippocampus, which may then lead to hippocampus damage. Meanwhile, the amygdalae primarily regulate the intensity of an emotional reaction to a traumatic event, and hence affect the arousal of other brain structures. Yet, individual differences in the predisposition to the occurrence of dissociative amnesia might be an important factor in developing this disorder; such as the predisposition to overreact to distressing factors. As a result, current research indicates that neurobiological mechanisms at least partially explain mechanisms of dissociative amnesia. Nevertheless, despite the recent progress in the identification of the biological mechanisms underlying this disorder, the research cannot be considered as completed and further investigation is needed.

Key words: amnesia, memory loss, amnestic state, neurobiology

INTRODUCTION

The term 'amnesia' originates from the Greek αμνησία 'forgetfulness' and relates to serious memory disorders, which make it impossible to learn new material or recall content previously memorised. Memory disorders may be caused by a variety of factors. Some of the factors include head traumas, cerebral circulation disorders, toxicological factors and neurological diseases of the central nervous system (e.g., cerebral degeneration process). Additionally, psychological factors have also been considered to be the cause of amnestic states (Staniloiu et al., 2010), with retrograde amnesia, which is an inability to recall previously memorised information, being most characteristic in this case (Markowitsch & Staniloiu, 2017). As a result, the term 'psychogenic amnesia' was previously used to describe this condition. Yet, according to the classification of the American Psychiatric Association – Diagnostic and Statistical Manual of Disorders (2013), this disorder is currently referred to as 'dissociative amnesia.' Dissociative amnesia (DA) is considered to be the most common dissociative disorder and is defined as the "temporary loss of recall memory caused by disassociation, which may last for a period of seconds or years [...] and is most often a result of psychological trauma. DA involves episodic autobiographical memory loss inconsistent with normal forgetfulness" (American Psychiatric Association, 2013). It is important to note that cognitive impairments, which are an essential element of DA, are a typical subsequence of traumatic events (Yasuno et al. 2000; Dąbkowska, 2007). The phenomenon of DA is hard to investigate, which could explain why there are still so many controversies about the classification of this disorder, its pathogenesis and even the correct nomenclature. The difficulties are related to a number of factors. To start with, correct differentiation between organic and dissociative memory loss can often be problematic. This results from the huge discrepancies between the clinical images of different individuals (Kritchevsky et al., 2004). Other factors impeding diagnosis include the short duration and varied course of the disorder (Andreasen & Black, 2001). Another serious methodological problem in studies on DA is difficulty with the correct classification of a traumatic event, preceding the occurrence of memory disorders. The aforementioned difficulties in studying the phenomenon of DA result in the lack of credible data concerning the frequency of the occurrence of this disorder. Thus, only partial information on the prevalence of this disorder is available. For example, Andreasen and Black (2001) reported 5-20% cases of this disorder among war veterans. For many years, studies on the mechanisms underlying DA were solely based on clinical observations of patients affected by this disorder. The introduction of a neuropsychological examination in the 1980s was a significant progress. This allowed one to describe in more details the psychological processes underlying the disorder (Kritchevsky et al., 2004). However, the biological basis of DA remained unknown. The introduction of functional methods of brain imaging was then an important step in studying the biological mechanisms of DA. Apart from the radiological methods of brain examination,

a lot of important data was acquired thanks to biochemical and neuropathological tests and examinations. These methods allowed identifying brain parts which play the key role in the mechanisms involved in reactions to the traumatic events which trigger DA. The most significant brain parts identified were: the prefrontal cortex, hippocampus and amygdalae (Tanev, 2003).

PREFRONTAL CORTEX

The prefrontal cortex seems to play a crucial role in integrating memory and emotional functions. This structure is connected with many neural paths leading from various brain parts, including, especially, the hippocampus and amygdalae (Aggleton & Passingham, 1981). As a result, all states related to cerebral activation are registered by the prefrontal cortex, independent of the fact as to whether they are cognitive (e.g., reading information in a newspaper), or emotional (agitation caused by this information). This structure combines an auditory stimulus, e.g., a barking dog, with fear of being bitten by the animal. The structure's function is combining all the received stimuli and ascribing appropriate meaning to the whole event. Meanwhile, the prefrontal cortex is a structure, which integrates internal and external experiences. This is probably due to the selection of received information. One can imagine that the 'important' information is enhanced, and the 'unimportant' is extinguished. Of course, ascribing meaning to information is subjected to an individual and depends probably on the emotional reactions related to the reception of such information (Reinhold & Markowitsch, 2009).

Special meaning is ascribed to the amygdalae in terms of the connection between the prefrontal cortex functions and emotional states. However, as was previously mentioned, the cortex is actually connected with the entire cerebrum. The links with other brain parts play a crucial role in the integration of cognitive functions, including memory with emotions. The connection between the neocortex and the limbic structures is especially significant for the memory functions, since the limbic cortex seems to have some toning influence on other prefrontal cortex areas.

Disorders of cortical functions may be a significant factor leading to the disintegration of the perceived phenomena, and hence pathological symptoms. The weakened cognitive inhibitory allows some information to be received and recorded beyond conscious control. The received information may form two independent streams or more, with only some of them being consciously processed (Dorahy, 2006). Studies conducted using contemporary brain study techniques seem to have brought evidence that the prefrontal cortex is involved in mechanisms underlying DA. Brand et al. (2009) analyzed in their study the consumption of glucose in different brain areas in individuals with this disorder, using the PET method. It turned out that memory disorders caused by a stressful situation impede the metabolic activity of the prefrontal lobes.

HIPPOCAMPUS

The hippocampus and actually the entire limbic system, were ascribed an important role in DA, especially in terms of its connections with the prefrontal cortex. The hippocampus was ascribed the main role in information storage and retrieval processes. The hippocampus seems to have a key meaning in responding to a traumatic event (Bergouignan et al., 2014). This is related to the high flexibility of this structure, which allows generating new connections among synapses in a short time. However, Weber and Reynolds (2004) pointed out that the hippocampus is also a structure which is very susceptible to all harmful factors, due to their late myelinisation. This may be very significant for the development of predispositions to overreact in response to a traumatic event, which will be elaborated on further below.

Severe or chronic stress leads to an excessive release of inflammatory cytokines and immunomodulatory molecules by glia in the hippocampus (Pearson-Leary et al., 2016). Astrocytes, microglia, and oligodendrocytes have dynamic responsibilities which substantially impact the neuronal function and activities, which directly relate to the ability to memorise information (Sajja et al., 2016).

Correct hippocampus functions are deregulated in the state of hyperarousal, in response to distressing factors (Spiegel 1997). Deregulation of its structures may lead to amnesia. The possible mechanism was partly described by McEwen (1993), who indicated the excessive and long-term deregulation of the HPA (hypothalamicpituitary – adrenal) axis to be the factor impeding the hippocampus functions. The HPA axis controls the secretion of so-called stress hormones, especially glucocorticoids, which are released in response to distressing factors. Glucocorticoids are released in physiological arousal states, triggered by distressing factors; meanwhile due to the negative feedback mechanism, they prevent further secretion and the reaction expires. It happens otherwise, when the secretion of glucocorticoids is prolonged or extremely intense. A lingering excessively high level of stress hormones leads to damage of the hippocampus structures. The hippocampus volume was observed to decrease after an experienced traumatic event (McEwen 1993), (Şar, 2014).

This is when the 'inefficient' hippocampus cannot coordinate memory processes correctly. There is a significant difference between correct conditions, when glucocorticoids affect maturation and the development of neurons, and hence learning and memory processes, and an extreme situation, when due to the excessive level of glucocorticoids they damage structures and cause disorders in brain functioning (Du et al., 2009). The phenomenon of DA may be considered as a kind of a pathological response to stress. In the case of DA, the arousal is excessive and triggers the functional deregulation. It is likely that hyperarousal of this structure deregulates its metabolic activity, which has been revealed in functional brain studies (Staniloiu et al., 2010).

However, it has to be underlined that neurobiological studies on the hippocampus structures have brought no unequivocal results so far. The relation between

the hippocampus volume and the post-stress reaction has not been indicated in all works (Tanev, 2003). In addition, an important, but still unresolved matter, remains the temporal relation between the occurrence of a traumatic event and changes in the hippocampus structure. It is worth noticing that although exposition to distressing factors probably has an impact on the hippocampus volume, still a reverse reaction is also likely, when the less efficient hippocampus enhances its response to traumatic events (Tanev, 2003).

AMYGDALAE

Another cerebral structure which is considered important when discussing DA, is amygdalae, which have numerous connections with other brain parts (Fine & Blair, 2000). Amygdalae are first of all involved in acquisition and emotional memories expression, as well as assessment of the emotional context of a stimulus (Ledoux & Muller, 1997). It can therefore be stated that amygdalae constitute the emotional basis of memory mechanisms. Amygdalae have numerous connections with other brain areas, including cortex and hippocampus structures; these connections are probably necessary to assess the meaning of the experienced stimulus. A stimulus reaching the brain is neutral, neither 'important,' nor 'unimportant;' it is the emotional factor which determines its meaning, when ascribed to it. It seems that in amygdalae information is ascribed an emotional component, and only later does it have a decisive meaning for the prefrontal cortex and affects the memorization of information. This structure probably determines, whether a stimulus reaching an organism and being registered and initially processed, will gain affective meaning, and hence be memorized.

Not only does the damage of amygdalae handicap emotional reactions, but it also causes memory disorders (Aggleton & Passingham, 1981), which further supports the relation between amygdalae and memory and emotions integration. Arousing amygdalae may probably intensify the activity of the prefrontal cortex. This hypothesis is supported by the existence of numerous connections between the amygdalae and cortex (Cunningham et al., 2002).

The mechanism, which explains the role of the amygdalae in DA, is an excessive arousal of this structure that occurs in some situations. Its intensified activity was observed in individuals, who experienced a stressful situation (Tanev, 2003). Human studies showed an increased cerebral blood flow in the amygdalae in individuals in emotional agitation states (Bergouignan et al., 2014).

It is highly probable that amygdalae determine to a high extent the intensity of an emotional reaction triggered by a traumatic event, and hence affect the level of arousal of other brain structures. The factor, which increases directly the activity of the amygdalae in reactions to stress, is probably stress hormones (Shin et al., 1997). However, the clinical effect will depend on the intensity of the HPA activation. In physiological conditions, the state of arousal will facilitate memory functions, but in certain circumstances, maybe under the impact of an excessive number of catecholamines, the amygdalae functions become dis-

torted, which affects other brain areas involved in memory mechanisms, especially the limbic system with the hippocampus and the frontal areas (Yasuno et al., 2000). Excessive activity of the amygdalae impedes the functioning of the hippocampus, which – when too aroused – becomes practically completely inactive (Weber & Reynolds, 2004). There are such situations, when the ability to select the received stimuli in the hippocampus and prefrontal cortex, is blocked.

The observation of primates reveals that damage to the prefrontal cortex or amygdalae deprives them of the ability to express the emotional states necessary for correct social functioning (Cunningham et al., 2002). This observation corresponds with studies on individuals with intensified post-stress disorders (including DA), who have deteriorated recognition of some emotions, especially fear and sadness. Psychological research has revealed a significant decrease in accuracy and sensitivity during the perception of these emotional states (Poljac et al., 2011).

MECHANISMS UNDERLYING PREDISPOSITIONS TO THE OCCURRENCE OF DISSOCIATIVE AMNESIA

Yet, not all individuals develop DA despite being exposed to a traumatic event. Therefore an important question arises: what predisposes an individual to develop this disorder? Is it possible to explain in biological terms the mechanisms underlying such a predisposition?

As Staniloiu et al. (2010) noticed some personality traits, such as narcissism, predispose to DA. In addition, individuals, who develop (dissociative) amnesia, often used to suffer from other mental disorders, especially depression and anxiety disorders. It may suggest a previous worse functioning of structures involved in the integration of emotional and memory processes. It is suggested that also people who experienced traumatic events in the past, are more predisposed to develop mental disorders, including dissociative amnesia. Contemporary brain research seems to confirm these observations. It was revealed that distressing factors decrease the hippocampus volume (McEwen, 1993). The decrease of its volume may explain predispositions to an intensified reaction in individuals who were exposed to stress in the past, especially, if a traumatic event was extremely intense or long-lasting (Roceri et al., 2002; Roceri et al., 2004). It may be assumed that the hippocampus flexibility is decreased; and the point is that a correct adaptive reaction depends on its flexibility (Şar, 2014).

Studies on factors having an impact on brain development in early life may prove very significant in order to investigate mechanisms predisposing to the development of DA (Shore, 2003). A lot of attention was paid to BDNF (brain-derived neurotropic factor). This factor is very significant for the development of neurons. It may be stated that the deficit of this factor decreases the pace of new cells development, and hence inhibits the differentiation of some brain parts. Some

empirical studies conducted on an animal model may to some extent illustrate pathogenetic processes leading to the development of DA in people.

Neurotropic factors, including BDNF, are more active in those brain areas which are very flexible, i.e., in all parts involved in memory functions, including especially the hippocampus (Huntley et al., 1992). Decreased BDNF secretion will therefore affect these structures the most. It was revealed on the animal model that a stressful situation may inhibit BDNF secretion in some brain parts, and hence decrease the pace of neuron development. Changes in BDNF concentration in response to a distressing situation depend on many additional factors; the stage of an organism's development is especially crucial. In the past it was repeatedly emphasized that early developmental stages are crucial for personality formation, and in consequence for susceptibility to traumatic factors. What was suggested in the past based on observations, seems to be confirmed in studies conducted using modern techniques.

Separation from parents is one of the strongest distressing situations in the case of a small child. In an animal model, isolation from a mother leads to a decreased BDNF level in the prefrontal cortex in young offspring. It may be assumed that the prefrontal cortex will be less differentiated, and hence its selective function will be more easily impeded in certain situations. And yet, as has been proved during experiments, these individuals become more susceptible to unfavorable factors in later stages of life; this is demonstrated by excessive reactions (Roceri et al., 2004). The observations are not only limited to the prefrontal cortex. During the maturation period, in individuals exposed to distressing factors, the secretion of BDNF and other neurotropic factors was reduced significantly not only in the prefrontal cortex, but also in the hippocampus structures. It may enhance predispositions to pathological reactions in response to distressing factors in further life (Roceri et al., 2002).

The negative impact of stress hormones on the hippocampus was mentioned before; this is especially strong when their level remains high for a long time, or, what may be the case during a traumatic event, their level sky-rockets. It turns out then that corticosterone is probably the mediator in this reaction, since it decreases the BDNF level.

It has been observed that appropriate preparation to a traumatic situation may reduce its psychological consequences to a high extent. It turns out that an increased glucocorticoids level is not always related to decreased BDNF expression. Such phenomenon was observed in experimental animals which had learnt beforehand how to avoid threats; in their case, despite the occurrence of a traumatic stimulus and an increased glucocorticoids level, the BDNF level stayed intact (Scaccianoce et al., 2003).

Although studies on BDNF provide very attractive results, it has to be remembered that they were conducted on an animal model. Considering that most of the BDNF studies were not conducted on primates, but on rodents, a very significant problem occurs related to whether these findings are transferrable to humans. Yet, a number of studies conducted on humans suggest that similar

mechanisms may also occur in humans. A particularly intriguing finding was drawn from a MRI study which revealed that patients with dissociative disorders have smaller hippocampus and amygdalae than controls (Vermetten et al., 2006).

Yet, the hippocampus, prefrontal cortex and amygdalae are not the only areas involved in mechanisms triggering dissociative amnesia. There is evidence suggesting the significance of other brain parts, too. A publication of Cloninger et al. (2008), which revealed the significance of the anterior cingulate cortex in DA, may serve as an example. It suggested that excessive arousal of the anterior cingulate cortex during a traumatic situation leads to the inhibition of motor functions and memory disorders. If the intensity of this phenomenon exceeds some physiological barrier, then pathological symptoms occur. The conclusion is that a similar mechanism to the one observed in the three aforementioned structures occurs: the case of the anterior cingulate cortex – excessive arousal of the given area leads to pathological symptoms (amnesia), due to a functional 'paralysis.' Moreover, studies reveal that the grey matter volume of the whole brain in patients with a dissociative disorder has various structural alterations in regions subserving the emergence of conscious perception (Daniels et al., 2015).

CONCLUSION

Dissociative amnesia is generally defined as the retrograde memory loss in the absence of detectable structural brain damage (Tramoni et al., 2009). However, there are significant differences in mental states among various individuals affected by this disorder. Its correct diagnosis is difficult, due to the varied clinical image, the frequent short duration of symptoms and their dynamic character (Andreasen & Black, 2001). Currently most researchers assume that the basic psychological mechanism underlying DA is dissociation, which is defined as the loss of ability to integrate various aspects of identity, memory, perception and consciousness (Dorahy, 2006). However, the phenomenon of dissociation itself is not pathological by nature (Staniloiu et al., 2010). It serves to protect personality integrity, when faced with strong and negative emotional states, especially those accompanied by fear. Yet, when a reaction to a traumatic event is excessive, DA may occur. The border between a physiological and pathological reaction is blurred and in some circumstances mechanisms triggered in response to a traumatic factor lose their adaptive character and lead to pathology (Spiegel, 1997).

The psychological observations of DA mechanisms were largely confirmed and explained in biological terms, by means of applied modern brain imaging techniques. Currently the prefrontal cortex, hippocampus and amygdalae are indicated as the main structures involved in the occurrence of this disorder. Stress hormones, whose secretion is controlled by the HPA axis, are considered to be the mediator during a traumatic event (Tanev, 2003). Dependent on the intensity of an emotional reaction, these structures may be aroused moderately, which is beneficial for memory and learning. However, excessive arousal may lead to functional disorganisation and hence amnesia (Yasuno et al.. 2000; Weber &

Reynolds 2004). Excessive arousal of the hippocampus, amygdalae or prefrontal cortex, and probably also other brain areas, leads to pathological symptoms (amnesia). The point is, these structures are so integrated that it is difficult to consider them separately. Biological mechanisms are analogical to the psychological description of a dissociative reaction (Cardeña & Spiegel, 1993; Butler et al., 1996).

Exposition to extreme situations may cause functional, and maybe also structural, changes in some brain structures, and hence additionally increase the predisposition to exhibit excessive reactions in response to traumatic events (McEwen, 1993). Exposition to traumatic factors becomes especially crucial in the period of learning and brain development (Weber & Reynolds, 2004). This observation was confirmed by experiments on an animal model, devoted to the investigation of the role of BDNF in brain development (Roceri et al., 2002; Roceri et al., 2004).

Yet, studies on biological mechanisms underlying psychogenic amnesia cannot be considered as completed. Current results are incomplete, often contradictory, and – first and foremost – there have been very few research projects focused on this phenomenon and the conducted studies were based on small populations. This is related to the significant methodological difficulties occurring in studies on this disorder. In addition, some of the quoted publications were based on animal experiments, which may not be applicable to humans. This problem is especially important in the case of psychological processes. Many interesting questions remain unanswered. For example, biological foundations responsible for the remission of symptoms are still unknown.

REFERENCES

Aggleton, J.P., & Passingham, R.E. (1981) Syndrome produced by lesions of the amygdala in monkeys, Journal of Comparative & Physiological Psychology, 95**,** 961–977.

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). Arlington, VA: American Psychiatric Publishing.

Andreasen, N.C., & Black, D.W. (2001): Introductory Textbook of Psychiatry. American Psychiatric Publishing, Washington, DC, London.

Bergouignan, L. , Nyberg, L., & H. Henrik Ehrsson, H.H. (2014) Out-of-body–induced hippocampal amnesia. Proceedings of the National Academy of Sciences USA, 11(12), 4421–4426.

Brand, M., Eggers, C., Reinhold, N., Fujiwara, E., Kessler, J., Heiss, W., & Markowitsch H. J. (2009) Functional brain imaging in 14 patients with dissociative amnesia reveals right inferolateral prefrontal hypometabolism. Psychiatry Research: Neuroimaging, 174 (1), 32–39.

Butler, L.D., Duran, R.E., Jasiukaitis, P., Koopman, C., & Spiegel, D. (1996) Hypnotizability and traumatic experience: A diathesis-stress model of dissociative symptomatology. American Journal of Psychiatry, 153, 42–63.

Cardeña, E., & Spiegel, D. (1993) Dissociative reactions to the San Francisco Bay Area earthquake of 1989. American Journal of Psychiatry, 150, 474–478.

Cloninger, C.R., & Dokucu M. (2008) Somatoform and Dissociative Disorders. The Medical Basis of Psychiatry, Part I, 181-194.

Cunningham, M.G., Bhattacharyya, S., & Benes, F.M. (2002) Amygdalo-cortical sprouting continues into early adulthood: implications for the development of normal and abnormal function during adolescence. Journal of Comparative Neurology, 453, 116–130.

Daniels, J.K., Gaebler, M., Lamke, J.P, & Walter, H. (2015) Grey matter alterations in patients with depersonalization disorder: a voxel-based morphometry study. Journal of Psychiatry & Neuroscience, 40(1), 19–27.

Dorahy, M.J. (2006) The dissociative processing style: a cognitive organization activated by perceived or actual threat in clinical dissociators. Journal of Trauma and Dissociation, 7, 29–53.

Du, J., Wang, Y., Hunter, R., Wei, Y., Blumenthal, R., & Falke, C. (2009) Dynamic regulation of mitochondrial function by glucocorticoids. Proceedings of the National Academy of Sciences of the USA 106, 3543–3548.

Fine, C., & Blair, R.J. (2000) The cognitive and emotional effects of amygdala damage. Neurocase, 6, 435–50.

Huntley, G.W., Benson, D.L., Jones, E.G., & Isackson, P.J. (1992) Developmental expression of brain derived neurotrophic factor mRNA by neurons of fetal and adult monkey prefrontal cortex. Brain Research. Developmental Brain Research, 70 (1), 53-63.

Kritchevsky, M., Chang, J., & Squire, L.R. (2004): Functional Amnesia: Clinical Description and Neuropsychological Profile of 10 Cases. Learning & Memory, 11 (2), 213-26.

Ledoux, J.E., & Muller J. (1997) Emotional memory and psychopathology. Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences, 352, 1719–1726.

Dąbkowska, M. (2007) Working Memory in Female Victims of Domestic Violence. Acta Neuropsychologica, 5(3), 90-98.

Markowitsch, H.J., & Staniloiu, A. (2017) Functional (dissociative) retrograde amnesia. Handbook of Clinical Neurology, 139, 419-445.

McEwen, B. S. (1993) Stress and the individual: Mechanisms leading to disease. Archives of Internal Medicine, 153, 2093–2101.

Pearson-Leary, J., Osborne, D.M., & McNay, E.C. (2016) Role of Glia in Stress-Induced Enhancement and Impairment of Memory. Frontiers in Integrative Neuroscience, 11(9), 63.

Poljac, E., Montagne, B., & de Haan, E.H. (2011) Reduced recognition of fear and sadness in posttraumatic stress disorder. Cortex, 2011, 47 (8), 974–980.

Reinhold, N., & Markowitsch, H.J.(2009) Retrograde episodic memory and emotion: a perspective from patients with dissociative amnesia. Neuropsychologia, 47(11), 2197-206.

Roceri, M., Cirulli, F., Pessina, C., Peretto, P., Racagni, G., & Riva, M.A. (2004) Postnatal repeated maternal deprivation produces age-dependent changes of brain-derived neurotrophic factor expression in selected rat brain regions. Biological Psychiatry, 55 (7), 708-14.

Roceri, M., Hendriks, W., Racagni, G., Ellenbroek, B.A., & Riva, M.A. (2002) Early maternal deprivation reduces the expression of BDNF and NMDA receptor subunits in rat hippocampus. Molecular Psychiatry, 7 (6), 609-16.

Sajja, V.S., Hlavac, N., & VandeVord, P.J (2016): Role of Glia in Memory Deficits Following Traumatic Brain Injury: Biomarkers of Glia Dysfunction. Frontiers in Integrative Neuroscience, 29 (10), 7.

Şar, V. (2014) The Many Faces of Dissociation: Opportunities for Innovative Research in Psychiatry. Clinical Psychopharmacology and Neuroscience, 12(3), 171–179.

Scaccianoce, S., Del Bianco, P., Caricasole, A., Nicoletti, F., & Catalani, A. (2003) Relationship between learning, stress and hippocampal brain-derived neurotrophic factor. Neuroscience, 121 (4), 825-8.

Shin, L.M., Kosslyn, S.M., McNally, R.J., Alpert, N.M., Thompson, W.L, Rauch, S.L., Macklin, M.L., & Pitman R.K. (1997) Visual imagery and perception in posttraumatic stress disorder. A positron emission tomographic investigation. Archives of General Psychiatry, 54, 233–241.

Shore, A.N. (2003) The experience-dependent maturation of a regulatory system in the orbitalprefrontal cortex and the origin of developmental psychopathology. In: Shore AN, (editor). Affect dysregulation and disorders of the self. New York: Norton, 5–35.

Spiegel, D. (1997) Trauma, Dissociation, and Memory. Annals of the New York Academy of Sciences, 821, 225–237.

Staniloiu, A., Markowitsch, H.J., & Brand, M.B. (2010) Psychogenic amnesia – A malady of the constricted self . Consciousness and Cognition, 19 (3), 778-801.

Tanev, K. (2003) Neuroimaging and neurocircuitry in post-traumatic stress disorder: what is currently known? Current Psychiatry Reports, 5 (5), 369-83.

Tramoni, E., Aubert-Khalfa, S., Guye, M., . Ranjeva, J. P, Felician, O., & Ceccaldi, M. (2009) Hypo-retrieval and hyper-suppression mechanisms in functional amnesia. Neuropsychologia, 47 (3), 611–624.

Vermetten, E., Schmahl, C., Lindner, S., Loewenstein, R.J., & Bremner, J.D. (2006) Hippocampal and amygdalar volumes in dissociative identity disorder. American Journal of Psychiatry, 163, 630–636.

Weber, D.A., & Reynolds, C.R. (2004) Clinical perspectives on neurobiological effects of psychological trauma. Neuropsychology Review, 14 (2), 115-129.

Yasuno, F., Nishikawa, T., Nakagawa, Y., Ikejiri, Y., Tokunaga, H., Mizuta, I., Shinozaki, K., Hashikawa, K., Sugita, Y., Nishimura, T., & Takeda, M. (2000) Functional anatomical study of psychogenic amnesia. Psychiatry Research, 99 (1), 43-57.

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