The role of attachment relationships in the pathogenesis of dissociative symptoms – literature review

Dawid Subocz

Abstract

Objective: The objective of this paper is to review the literature on the role of the attachment relationship in the pathogenesis of dissociative symptoms. The paper integrates the knowledge of environmental and genetic factors as well as neurobiological processes mediating the development of dissociative symptoms in a holistic way.

Material and method: The literature review was performed with the use of the following databases: EBSCO, PsycINFO, PubMed, Google Scholar. The databases were searched using the following keywords: "attachment style", "dissociation", "trauma". These terms were combined with the words 'secure', 'disorganised', 'epigenetic', 'genetic' and the term 'monoaminergic system'.

Results: Literature analysis suggests that the pathomechanism linking traumatic experiences in the attachment relationship with dissociative symptoms is mediated by neurobiological factors. These include: dysregulation within monoaminergic systems and atrophy of neuronal structures that play a role in affective regulation.

Discussion and conclusions: Research suggests that traumatic experiences in the attachment relationship are a risk factor for the development of dissociative symptoms in the future. There is still a lack of research verifying the relationship between attachment experiences and epigenetic changes in genes regulating the activity of monoaminergic systems. These processes may play an important role in the aetiology of many no-sological units, the development of which is mediated by dissociative symptoms. Future research should evaluate dissociation from the perspective of a transdiagnostic risk factor for the development of a broad spectrum of psychopathology.

attachment style; dissociation; neurobiology

INTRODUCTION

Dissociation can be defined as a way of coping with stress induced by external or internal factors consisting in a change in the integrating function of identity or consciousness [1]. It is a temporary narrowing of the stream of con-

Dawid Subocz: University of Szczecin, Institute of Psychology, Szczecin, Poland Correspondence address: dawid.subocz@o2.pl sciousness covering three levels: the cognitive level (variable perception of oneself and the environment), the emotional level (a sense of alienation and indifference to the environment and / or oneself) and the physiological level (increased sensitivity threshold) [2]. Dissociation changes the perception of oneself, the surrounding reality and causes a temporary disconnection or lack of contact between mental and physical processes. Due to the fact that dissociation is characterised by a diverse category of symp-

toms, it has been proposed to divide them into two categories: detachment and compartmentalisation [3]. The dissociative symptoms of detachment are related to the feeling of being alienated from one's own emotions, one's own body, from the usual sense of one's own identity, or from the normal sense of the known reality of the environment. The symptoms of detachment include the distortion of the conscious experience of "self" (depersonalisation) and the surrounding world (derealisation). The dissociative symptoms of compartmentalisation, on the other hand, prevent comparisons and semantic connections between the mental content, which should normally simultaneously enter the area of "self" consciousness. Examples of the symptoms of compartmentalisation are: dissociative amnesia (in which the memory is not available due to the dissociative process) or non-integrated states of "self" (in which it is not possible to synthesise meanings allowing to give a coherent narrative of different autobiographical episodes and semantic representations of "self") [4].

Dissociation is a mechanism that serves as the defence of the Ego and serves to deal with traumatic experiences [5]. Research confirms the relationship between dissociation and traumatic experiences in childhood [6], war experiences [7], as well as terrorist attacks [8]. It is the dissociative symptoms of detachment that are considered to be the first pathogenic consequences of trauma [4]. Traumatised persons often report symptoms of depersonalisation. During these experiences they felt as if they were standing next to each other and looking at each other like observers of their own actions. By temporarily losing awareness of one's own experiences and emotions, dissociation is a strategy that enables adaptation in a traumatic situation. The problem arises, however, when the strategy of dissociation response to traumatic events undergoes generalisation to situations with a lower level of threat. The habitual use of this mechanism causes it to become a catalyst for self-destructive behaviour and may lead to an excessive reduction in self-awareness. Many studies suggest that dissociation often precedes suicide attempts and acts of self-mutilation. The state of dissociation is so discomforting that it may result in selfmutilation in order to restore the usual clarity of sensations [5].

We still do not know what factors influence the susceptibility to dissociative reactions after traumatic stress. Some theoretical models [9-11] suggest that early experiences in the attachment relationship play an important role in the genesis of dissociative states after trauma. A secure attachment relationship – according to these assumptions – immunises the individual against dissociative reactions after traumatic experiences in the future. The aim of this paper is to present the current state of knowledge on the role of the attachment relationship in the pathogenesis of dissociative symptoms and to analyse the neurobiological processes that mediate the development of these symptoms.

The attachment relationship and dissociation – theoretical models

The basic task for the caregiver in the first year of life of the infant is to create a secure attachment with it. It depends on the psychobiological synchronisation of the mother with the child's internal states of arousal. Through visual-facial, tactile-gestural and auditory-prosodic communication, the caregiver and the infant modify their behaviour to tune in with each other. In this way, trusting attachment is created [11]. According to attachment theory [12], memories of interaction with the parent are the basis for the development of internal operating models. Internal operating models are cognitive structures based on generalised memories of prior interactions with an attachment figure. They allow for the formulation of expectations regarding the future reactions of the attachment figure to the needs of the child's bonding. In the first period of life, internal operating models are structures of non-declarative memory. They do not require language or awareness functions. During development, some internal operating models may enter the conscious level. Later bonding attempts may modify them, but they are relatively stable over time [13]. In the case of a secure attachment style, internal operating models create an internal sense of availability, help, and encouragement from a loved person. In the case of unsecured attachment, internal operating models build the belief that the attachment figure will remain inaccessible or that it will re-

Archives of Psychiatry and Psychotherapy, 2022; 2: 54-61

act negatively to a request for help [10]. If the attachment system generated "distrustful" internal operating models, the emotional responses to future traumatic and stressful experiences may become even more severe. This is because distrustful internal operating models negatively evaluate a situation in which we need help or encouragement from others. Searching for them turned out to be groundless in the past or was associated with additional negative confrontations with the attachment figures [4, 10].

Elizabeth Carlson, Tuppett Yates and Alan Srouf [9] presented four developmental patterns of dissociation. Persons with a disorganised attachment style, but without traumatic experiences, are characterised by an elevated but subclinical level of dissociation (pattern A). They have a latent dissociation predisposition that may become apparent under stress. In persons with a disorganised attachment style and severe traumatic experiences, pathological dissociation (pattern B) develops. On the other hand, a normative dissociative process is possible in a supportive and stable relationship with caregivers (pattern C). Normative dissociation is characterised by dissociative processes typical of early childhood functioning. Before developing the ability to understand complex processes or conflicting experiences, children naturally compartmentalise them. They separate content into simple categories such as good or bad, positive or negative. The early mental representations are very different from each other. The inability to compensate for them causes that during this period of development they isolate themselves from one another. This is called normative dissociation, which, through difficult emotional experience, can be transformed into a pathological type of dissociation. When persistent self organisation has developed, defensive reactions such as dissociation are used adaptively in difficult situations. In the case of severe traumatic experiences, despite stable relationships with caregivers, the child may develop a pathological dissociative process (pattern D). This development is less intense than that observed in the case of victims of traumatic experiences who did not have a stable relationship with their caregivers in childhood (pattern B). Therefore, it can be concluded that a secure and supportive attachment with the caregiver immunises the child against the development of pathological dissociation in the future [9]. This is confirmed by studies [6,8] showing that persons who experienced trauma in the attachment relationship in childhood were characterised by a greater intensity of dissociation and PTSD symptoms than those without such experiences. Among the surveyed New Yorkers, both dissociation and attachment styles were mediators of a positive relationship between the experience of physical and sexual abuse in childhood and the intensity of PTSD symptoms after the World Trade Centre attacks [8]. Another study [6], conducted on prisoners, found a relationship between dissociation and the experience of childhood abuse, including sexual abuse. These results suggest that early childhood relational traumatic experiences may influence dissociative responses in the future.

Neurobiology of attachment and the pathogenesis of dissociative symptoms

Despite the theoretical models presented above, the mechanism linking attachment with dissociative symptoms is still unknown. Research [6, 8] suggests that dissociative symptoms are the result of difficult experiences, including those related to the attachment relationship. However, the aetiology of dissociative states is multifactorial. Dissociative symptoms are related to brain injuries, e.g. hypoxia or dysconnection of neuronal areas responsible for the integration of mental representations of "self". The isolation, deactivation or hyperactivation of these structures results in the states of self-perception disintegration [14]. For example, electrical stimulation of the hippocampus results in dissociative symptoms such as depersonalisation and derealisation [15, 16]. Persons experiencing dissociation show greater activity in somatosensory areas of the brain [17]. Dissociative states are also associated with the activation of the temporal, occipital and parietal lobes when recalling traumatic experiences [18]. Dissociation is also associated with the use of psychoactive substances, which in turn affect the biochemical neuronal response that can manifest itself through dissociative states. Dissociative symptoms occur, among others, after: taking ketamine which acts by antagonising the NMDA receptor [19], cannabi-

Archives of Psychiatry and Psychotherapy, 2022; 2: 54-61

noids, including smoking marijuana [20] or taking hallucinogens such as LSD [20]. It is worth emphasising that addiction to psychoactive substances may constitute a dysfunctional form of emotional regulation resulting from the patient's development within the insecure attachment relationship [21, 22]. In an insecure attachment relationship, the child does not learn to regulate its emotions [23]. When examining addicts, it is important to assess whether dissociation is a direct result of psychoactive substances or traumatic experiences in the attachment relationship, which are associated with using drugs [21]. Attachment with the caregiver is associated not only with the development of the ability of affective regulation, but also with biochemical reactions in the child's brain, which may be a risk factor for the development of pathological dissociation. It is the analysis of neurobiological factors that allows the verification of the role that the attachment relationship plays in the development of dissociative states.

The research [24] carried out with the use of functional magnetic resonance indicates dopaminergic activity within the reward system in response to stimulation activating care behaviours (e.g. through exposure of the child's photos). The relationship between the neuronal activation of mesolimbic dopaminergic pathways and the attachment relationship in close relationships has also been demonstrated. Researchers [25, 26] showed that those in love in partner relationships are characterised by higher dopaminergic activity in such areas as the ventral tegmental area and the anterior cingulate cortex. Moreover, difficult experiences in the attachment relationship in childhood result in hyperactivity of the hypothalamic-pituitary-adrenal system, which is associated with increased dopamine synthesis and its dysregulation in the striatum [27]. Researchers [28] agree that changes in the dopaminergic system play a significant role in the relationship of attachment. The dopaminergic system also plays an important role in the pathogenesis of dissociative symptoms. Researchers [29] showed that the level of homovanillic acid – the main metabolite of dopamine - in persons suffering from eating disorders positively correlates with the severity of dissociative symptoms. The level of homovanillic acid positively correlates with the susceptibility to hypnosis, the indispensable component of which is dissociation [30]. Apart from dopamine, serotonin may also play a significant role in the pathogenesis of dissociative symptoms. This is indicated by research confirming the activation of 5HT2A and 5HT2C receptors – which are serotonin antagonists – during the occurrence of dissociative symptoms [31]. Moreover, research has shown a strong negative correlation between the level of norpinephrine in the urine and the severity of depersonalisation symptoms [32]. Attachment relationship experiences may therefore constitute an environmental risk factor for dissociation, disrupting monoaminergic systems.

Genetic studies [33] of twin pairs that used the heritability index suggested that genetic factors accounted for 45% of the variance in dissociative symptoms, while the remaining 55% of the variance explained a specific environment and measurement error. Moreover, molecular studies [33] suggest that subjects with the 5-HTTL-PR genotype report more dissociative symptoms than those who do not have this genotype and experienced trauma and depressive symptoms. Research [34] also suggests that bipolar disorder patients who experienced childhood trauma are characterised by a higher intensity of dissociative states when characterised by the Val158Met polymorphism of the COMT gene. These results [33] may suggest that genes responsible for serotonin synthesis, including 5-HTTLPR, play a role in the pathogenesis of dissociation. The genetic tendency towards dissociative states can thus be activated through traumatic experiences in the attachment relationship. Future research should analyse what types of relational experiences influence epigenetic changes in genes regulating the activity of monoaminergic systems, the deregulation of which may be manifested by dissociative states [35].

A hypothetical explanation of the effect of attachment relationships on dissociation is epigenetic modifications that program gene expression patterns during brain development [36]. Genes are responsible for the growth and direction of axons and dendrites to their correct destinations in the developing brain. However, there are not enough genes across the human genome to precisely define each synapse. By creating an excessive number of synapses, the brain forces them to compete. Only those synapses that are more active, receive more electrical impulses and release more neurotransmitters are preserved. If synapses responsible for emotional regulation are not active, they will disappear [37]. The relationship with the caregiver can therefore be perceived as an interaction causing biochemical reactions and activation of synapses responsible for the emotional development of the child. The phenomenon of attachment between mother and offspring is conditioned by oxytocin [38]. Among mothers diagnosed with insecure avoidant attachment style, decreased levels of oxytocin were found in the situation of contact with the child.

The relationship with the mother is therefore an opportunity for the child to modulate affective states, which is accompanied by biochemical reactions in both the child and the mother. It is a kind of emotional resonance, in which positive states may be strengthened. This causes the activation of synapses in the child related to the development of affective regulation, which Schore [11] locates in the structures of the right hemisphere of the brain. This author suggests that the attachment mechanism is embedded in the affective interaction between the infant's right hemisphere and the caregiver's right hemisphere. This is because the myelination of the limbic system occurs within the first 18 months [39]. Moreover, at this time there is also a rapid growth of the right hemisphere [40-42] with its connections to the limbic system [44]. According to the Schore's [11] concept, dissociative symptoms result from the development in the insecure attachment relationship, which results in the detachment of the normally integrated functions of the right hemisphere neuroautonomic system, including the orbital-frontal structures. The orbital-frontal system is important in the process of cognitive-emotional interaction [44]. This part of the emotional brain functions as an internal reflection centre. It allows for integration and assigning an emotional-motivational meaning to a cognitive sign and for the formation of relationships between emotions and thinking [45]. It also plays an important role in the relationship between emotional information and the action selection mechanism. These data suggest that victims of dissociative trauma are characterised by deficits in the cognitive and emotional structures in the right orbit-

al-frontal part of the brain, along with its cortical and subcortical connections. Research shows that when the subcortical connections within the right hemisphere function properly, it coordinates the flow of sensory information [46, 47]. However, under extreme stress, both hyperactivation and hypoactivation of the cortical parts of the right hemisphere result in a loss of the ability to integrate sensory processes. Moreover, during intense stress, the right hemisphere may lose the ability to integrate between the cortical and subcortical systems. Then, processing of the limbicautonomic information occurs only at the lower level of the right amygdala and is blocked from accessing the higher neural structures (i.e. the anterior cingulate gyrus and orbital-frontal areas). Partially processed information [48] cannot be integrated with consciousness, including the awareness of subjectively experienced emotions. It manifests itself through dissociative experiences, which, according to this concept [11], are defined as the result of the loss of the integrative abilities of the vertical emotional organisation of the right hemisphere of the brain.

According to the Schore's [11] concept, connections in the limbic system in a child, which are not supported by the caregiver in the process of emotional regulation, are exposed to the effects of toxic neurotransmitters such as glutamate and cortisol, with longer exposure time. [49]. It may also be associated with the atrophy of such neuronal structures as the hippocampus, the size of which negatively correlates with the severity of dissociative symptoms [50, 51]. Some researchers [52, 53] suggest that stress resulting from insecure attachment relationship may – by activating NMDA receptors strongly concentrated in the hippocampus - mediate dissociative symptoms. As part of this pathomechanism, the developing individual becomes susceptible in the future to reacting with dissociative states to traumatic experiences. The nervous system shaped in this way is also not able to regulate intense states of arousal. During the trauma experience, the immature prefrontal cortical parts of the limbic system are disorganised and NMDA receptors are activated, resulting in dissociation.

CONCLUSIONS

The literature analysis presented here allows for the integration of theoretical concepts with contemporary neurobiological research. It suggests that the genotype interacts with unfavourable life experiences - especially those in the attachment relationship early in life - creating individual differences in resistance to future traumatic experiences and susceptibility to dissociative reactions [36]. Dissociative symptoms at the neurobiological level are related to dysregulation within monoaminergic systems. This dysregulation results both from genetic factors responsible for the synthesis of dopamine and serotonin, and from experiences in the relationship of attachment [33]. Difficult experiences in the attachment relationship - resulting in the release of cortisol via the hypothalamic-pituitary-adrenal (HPA) axis - sensitise systems of neurotransmitters such as dopamine and serotonin, causing their excessive presynaptic synthesis and increased release [54]. The literature analysis also suggests that insecure attachment styles - mediated by epigenetic processes - may lead to the development of atrophy of neuronal structures, including the hippocampus with multiple NMDA receptors [50, 51]. The activation of these receptors under stress is directly involved in triggering dissociative symptoms [52, 53]. The development of a child in an insecure attachment relationship does not allow the development of neural connections necessary to regulate emotions, which makes it more susceptible to future traumatic experiences [11]. The next step of the researchers should be to verify the role that neurobiological processes – which lead to dissociative states in the insecure attachment relationship - play in the aetiopathogenesis of mental illness.

Traumatic experiences and dissociative symptoms are present among patients in almost all groups of psychiatric disorders. They are found in anxiety disorders [55]; personality disorders [56]; mood disorders [57]; eating disorders [58]; addictions [59]; schizophrenia and other psychoses [60]. Due to the fact that almost all diagnostic categories are related to the dissociative dimension, it is necessary to adopt a nosology dimensional perspective in order to explain dissociative pathology. It characterises psychiatric manifestations based on the main mechanism that disrupts their order. This mechanism - manifested by dissociative symptoms is most likely the disconnection of cortical and subcortical connections of the brain and dysregulation of monoaminergic systems, occurring in the etiology of most mental diseases. These changes probably occur as a result of epigenetic processes, activated under the influence of early childhood relational experiences [36]. Future research should verify the above hypotheses in detail. However, the analysis of the literature to date suggests that dissociation and insecure attachment relationship are transdiagnostic risk factors for the development of complex mental health problems.

REFERENCES

- Horowitz MJ, Markman HC, Stinson CH, Fridhandler B, Ghannam JH. A Classification Theory of Defense. In: Singer JL, editors. Repression and Dissociation. Chicago: The University of Chicago Press; 1990. p. 28 – 51.
- Orbach I. Attitudes toward the Body in Suicidal, Depressed and Normal Adolescents. Suicidal and Life – Threatening Behavior. 1995; 25(2): 211–221.
- Holmes EA, Brown RJ, Mansell W, Fearon RP, Hunter EC, Frasquilho F, Oakley DA. Are there two qualitatively distinct forms of dissociation? A review and some clinical implications. Clinical Psychology Review. 2005; 25(1): 1-23.
- Liotti G, Farina B. Traumatyczny rozwój. Etiologia, klinika i terapia wymiaru dysocjacyjnego. Fundacja Przyjaciele Martynki, Bledzew: 2016.
- Matecka M, Wycisk M. Dysocjacja: różnorodność kontekstów i znaczeń. Próba klaryfikacji. Czasopismo Psychologiczne. 2003; 9(2): 199-206.
- Akyuz G, Kugu N, Sar V, Dogan O. Trauma and dissociation among prisoners. Nord. J. Psychiatry. 2007; 6: 167-172.
- Zerach G, Greene T, Ginzburg K, Solomon Z. The Relations Between Posttraumatic Stress Disorder and Persistent Dissociation Among Ex-Prisoners of War: A Longitudinal Study. Psychological Trauma: Theory, Research, Practice, and Policy. 2014; 6(2): 99–108.
- Twaite JA, Rodriguez-Srednicki O. Childhood Sexual and Physical Abuse and Adult Vulnerability to PTSD: The Mediating Effects of Attachment and Dissociation. Journal of Child Sexual Abuse. 2004; 13(1): 17-38.
- Carlson EA, Yates TM, Sroufe LA. Dissociation and Development of the Self. In: Paul F. Dell, John A. O'Neil, editors. Dissociation and the dissociative disorders. DSM-V and Beyond. New York: Rutledge Taylor & Francis Group; 2009. p. 39-52.

Archives of Psychiatry and Psychotherapy, 2022; 2: 54–61

- Liotti G. Trauma, Dissociation, and Disorganized Attachment: Three Strands of a Single Braid. Psychotherapy: Theory, research, practice, training. 2004; 41: 472-486.
- Schore AN. Attachment Trauma and the Developing Right Brain: Origins of Pathological Dissociation. In: Dell PF, O'Neil JA, editors. Dissociation and the dissociative disorders. New York: Routledge Taylor & Francis Group; 2009. p. 107-141.
- Bowlby J. Przywiązanie. Warszawa: Wydawnictwo Naukowe PWN; 1988.
- Main M, Hesse E, Kaplan N. Predictability of attachment behaviour and representation processes at 1, 6, and 19 years of age: the Berkeley longitudinal study. In: Grossmann KE, Grossmann K, Waters E, editors. Attachment from Infancy to Adulthood: The Major Longitudinal Studies. New York: The Guilford Press; 2005. p. 245-304.
- Sack O. A journey inside the brain. The New York Review of Books. 2008; 55(4): 1-14.
- Halgern E, Walter RD, Cherlow DG, Crandall PH. Mental phenomena evoked by electrical stimulation of the hippocampal formation and amygdale. Brain.1978; 101: 83-117.
- Penfield W, Perot P. The brain's record of auditory and visual experience: a final summary and discussion, Brain.1963; 86: 595-696.
- Simeon D, Guralnik O, Hazlett S, Spiegel-Cohen J, Hollander E, Buchsbaum MS. Feeling unreal: A PET study of depersonalization disorder. American Journal of Psychiatry. 2000; 157: 1782-1788.
- Lanius RA, Williamson PC, Boksman K, Densmore M, Gupta MA., Neufeld RWJ, Gati JS, Menon RS. Brain activation during script-driven imagery induced dissociative responses in PTSD: A functional magnetic resonance imaging investigation. Biological Psychiatry. 2002; 52: 305-311.
- Currean HV, Morgan C. Cognitive, dissociative, and psychogenic effectsof ketamine in recreational users on the night of drug use and 3 days later. Addiction. 2000; 95: 575-590.
- Simeon D, Knutelska M, Nelson D, Guralnik O. Feeling unreal: a depersonalization disorder update of 117 cases. Journal of Clinical Psychiatry. 2003; 64: 990-997.
- Zdankiewicz-Ścigała E. Aleksytymia i dysocjacja jako podstawowe czynniki zjawisk potraumatycznych. Warszawa: Wydawnictwo Naukowe Scholar; 2017.
- Besharat MA, Salimian MM. The relationship between attachment styles and alexithymia: Mediating role of self-regulation. International Journal of Research Studies in Psychology. 2014; 3(4): 89-98.
- Allen JG, Fontafy P, Bateman AW. Mentalizowanie w praktyce klinicznej. Kraków: Wydawnictwo Uniwersytetu Jagiellońskiego; 2014.
- Strathearn L. Maternal neglect: oxytocin, dopamine and the neurobiology of attachment. Journal of Neuroendocrinology. 2011; 23(11): 1054-1065.

- Aaron A, Fisher H, Mashek DJ, Strong G, Li H, Brown LL. Reward, motivation, and emotion system associated with early-stage intense romantic love. Journal of Neurophysiology. 2005; 94(1): 327-337.
- Bartles A, Zeki S. The neural correlates of maternal and romantic love. Neuroimage. 2004; 21(3): 1155-1166.
- Walker EF, Diforio D. Schizophrenia: a neural diathesisstress model. Psychological Review. 1997; 104(4): 667-685.
- Gordon I, Martin C, Feldman R, Leckman JF. Oxytocin and social motivation. Developmental Cognitive Neuroscience. 2011; 1(4): 471-493.
- Demitrack AD, Putnam FW, Rubinow DR, Pigott TA, Altemus, A, Krahn DD, Gold PW. Relation of Dissociative Phenomena to Levels of Cerebrospinal Fluid Monoamine Metabolites and Beta-Endorphin in Patients With Eating Disorders: A Pilot Study. Psychiatry Research. 1993; 49: 1-10.
- Spiegel D, King R. Hypnotizability and CSF HVA levels among psychiatric patients. Biological Psychiatry. 1992; 31: 95-98.
- Simeon D, Hollander E, Stein DJ. Introduction of depersonalization by the serotonin agonist meta-chlorophenylpiperazine. Psychiatry Research. 1995; 58: 161-164.
- Simeon D, Guralnik O, Knutelska M. Hypothalamic-pituitaryadrenal axis function in dissociative disorders, PTSD, and health volunteers. Biological Psychiatry. 2001; 6: 966-973.
- Pieper S, Out D, Bekermans-Kranenburg MJ, IJzendogrn MH. Behavioral and molecular genetics of dissociation: The role of the serotonin transporter gene promoter polymorphism (5-HTTLPR). Journal of Traumatic Stress. 2011; 24(4): 373-380.
- Savitz JB, Merwe L, Newman TK, Solms M, Stein DJ, Rumesar RS. The relationship between childchood abuse and dissociation. Is it influenced by catechol-O-methyl transference (COMT) activity? International Journal of Neuropharmaology. 2008; 11: 149-161.
- Sullivan RM. The neurobiology of attachment to nurturing and abusive caregivers. Hasting Law Journal. 2012; 63(6): 1553-1570.
- Daskalakis NP. Schizophrenia in the Spectrum of Gene– Stress Interactions: The FKBP5 Example, Schizophrenia Bulletin. 2015; 41(2): 323–329.
- Elliot L. Co tam się dzieje? Jak rozwija się mózg i umysł w pierwszych pięciu latach życia. Poznań: Media Rodzina; 2003.
- Buszewicz M, Karakuła-Juhnowicz H, Pelczarska-Jamroga A. Rola oksytocyny w mechanizmie powstawania przywiązania oraz w społecznym funkcjonowaniu osób chorych na schizofrenię. Current Problems of Psychiatry. 2014; 15(4): 169-175.
- Kinney HC, Brody BA, Kolman AS, Gilles FH. Sequence of central nervous system myelination in human infancy II Patterns of myelination in autopsied infants. Journal of Neuropathology and Experimental Neurology. 1988; 47: 217-234.

Archives of Psychiatry and Psychotherapy, 2022; 2: 54-61

- Allman JM, Watson KK, Tetreault NA, Hakeem A. Intuition and autism: a possible role for Von Economo neurons. Trends in Cognitive Sciences. 2005; 9: 367-373.
- Bogolepova IN, Malofeeva LI. Characteristics of the development of speech areas 44 and 45 in the left and right hemisphere of the human brain In Elary post-natal ontogenesis. Neuroscience and Behavioral Physiology. 2001; 31: 349-354.
- Chiron C, Jambaque I, Nabbout R, Lounes R, Syrota A, Dulac O. The right brain hemisphere is dominant in human infants. Brain. 1997; 120: 1057-1065.
- Tucker DM. Developing emotions and cortical networks. In: Gunnar MR, Nelson CA, editors. Minnesota symposium on child psychology. Vol 24. Developmental behavioral neuroscience. New York: Erlbaum; 1992. p. 75-128.
- Barbas H, Saha S, Rempel-Clower N, Ghashghaei T. Serial pathways from primate prefrontal cortex to autonomic areas may influence emotional expression. BMC Neuroscience. 2003; 4.
- Joseph R. Neuropsychiatry, neuropsychology, and clinical neuroscience, second ed. Baltimore: Williams & Wilkins; 1996.
- Calvert GA, Hanses PC, Iversen SD, Brammer MJ. Detection of audio-visual integration sites in human by application of electrophysiological criteria to the BOLD effect. NeuroImage. 2001; 14: 427-483.
- Raij T, Utela K, Riita R. Audio-visual integration of letters In the human brain. Neuron. 2000; 28: 617-625.
- Whitlock FA. The etiology of hysteria. Acta Psychiatrica Scandinavica. 1967; 43: 144-162.
- Migda M. Trauma więzi a psychoterapia pacjentów z diagnozą PTSD w oparciu o mentalizację. Psychoterapia. 2015; 3(174): 99-105.
- Bremner JD, Vythilingam M, Vermetten E, Southwick SM, McGlash T, Nazeer A, Khan S, Vaccarino LV, Soufer R, Garg P, Ng CK, Staib LH, Duncan JS, Charney DS. MRI and PET study of deficits in hippocampal structure and func-

tion in women with childhood sexual abuse and posttraumatic stress disorder (PTSD). American Journaln of Psychiatry. 2003; 160: 924-932.

- Stein MB, Koverola C, Hanna C, Torchia MG, McClarty B. Hyppocampal volume in women victimized by childchood sexual abuse. Psychological Medicine. 1997; 27: 951-959.
- Bremner JD. Does stress damage the brain? Understanding trauma-related disorders from mind-body perspective. New York: W. W. Norton; 2002.
- 53. Bremner JD., Marmar C. Trauma, memory and dissociation. Washington, DC: American Psychiatric Press; 1998.
- Howes OD, Murray RM. Schizophrenia: an integrated sociodevelopmental-cognitive model, Lancet, 2014; 10(383): 1677–1687.
- Warshaw MG, Fierman E, Pratt L, Hunt M, Yonkers KA, Massion AO, Keller MB. Quality of life and dissociation in anxiety disorder patients with histories of trauma or PTSD. The American Journal of Psychiatry.1993; 150: 1512-1516.
- Dolan-Sewell RT. Co-occurrence with syndrome dis-orders. In: Livesley WJ, editors. Handbook of Personality Disorders: Theory, Research, and Treatment. New York: Guilford Press; 2001. p. 84-104.
- Lewis CV, Simons AD, Nguyen LJ, Murkami JL, Reid MW, Silva SG, MarchJ S. Impact of childchood trauma on treatment outcome in the Treatment for Adolescents with Depression Study. Journal of the American Academy of Child and Adolescent Psychiatry. 2010; 49: 132-140.
- McShane JM, Zirkel S. Dissociation In the bonge-purge cycle of bulimia nervosa. Journal of Trauma and Dissociation. 2008; 9: 463-479.
- Enoch MA. Genetic and environmental influences on the development of alcoholism: Resilience vs risk. Annals of New York Academy of Sciences. 2006; 1094: 193-201.
- Moskowitz AK, Barker-Collo S, Ellson L. Replication of dissociation-psychosis link in New Zealand students and inmates. Journal of Nervous and Mental Disease. 2005; 193(11): 722–727.