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The role of the attachment relation in the pathogenesis of auditory hallucinations – literature overview

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Abstract

Objective: The purpose of the article is to provide an overview of the literature concerning the role of the attachment relation in the pathogenesis of auditory hallucinations. The article holistically integrates the knowledge about the attachment relation with cognitive and biological factors.

Materials and method: The literature overview was compiled using the following databases: EBSCO, PsycINFO, PubMed, Google Scholar. The databases were searched using the following keywords: „secure", „disorganized", „epigenetic", „genetic", „auditory hallucinations" and the term „monoaminergic system". These words were combined with the term „attachment style".

Results: The literature analysis suggests that neurobiological and cognitive factors mediate in the pathomechamism affiliating traumatic experience in the attachment relation with auditory hallucinations These include: dysregulation within the monoaminergic systems, atrophy of the temporal areas and cognitive distortions such as source monitoring errors and negative automatic thoughts.

Discussion and conclusions: Cognitive dysfunctions along with hereditary genetic susceptibility to auditory hallucinations may constitute factors distorting the correct interpretation of traumatic intrusions of relational experience. Future research should verify to what extent auditory hallucinations are the memory marks of a relational trauma. Verification of these hypotheses will enable better understanding of the role of the attachment styles in the development of auditory hallucinations and the formation of convictions about them.

attachment style; auditory hallucinations; voice hearing

INTRODUCTION

Hallucinations are defined as perceptual experience occurring despite lack of any outside stimuli. Hallucinations occur while awake, and their appearance is independent of the will [1]. They may affect all the senses. The subject of this article are auditory hallucinations. For some individuals, auditory hallucinations are a response to traumatic life experience. About 1% of the population experiences auditory hallucinations without requiring a psychiatric care. Another 1% of the population suffers from schizophrenia. About 75% of this clinical group experienced auditory hallucinations [2, 3]. The purpose of this article is to present the current state of knowledge regarding the role of the attachment relation in the pathogenesis of auditory hallucinations and to analyse the biological and cognitive processes involved in the development of these symptoms.

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MATERIAL AND METHODS

A systematic review of the literature was made using the following databases: EBSCO, PsycINFO, PubMed, and Google Scholar. Each database was searched using the following keywords: "secure", "disorganized", "epigenetic", "genetic", "auditory hallucinations" and "monoaminergic system". These words were combined with the term "attachment style". 512 publications were identified during the database search. Duplicates and articles unrelated to the purpose of this thematic review were excluded. On the basis of such selection, 70 publications were included in the qualitative analysis. The process of searching and selecting research papers is presented in the PRISMA scheme (Figure 1). The analysis of the literature was presented in relation to the theoretical model of Liotti and Gumley [4]. This makes it possible to present an overview of research within the broader context of the pathogenesis of positive symptoms. Therefore, the paper presents the mutual interaction of biopsychosocial risk factors for the development of auditory hallucinations.

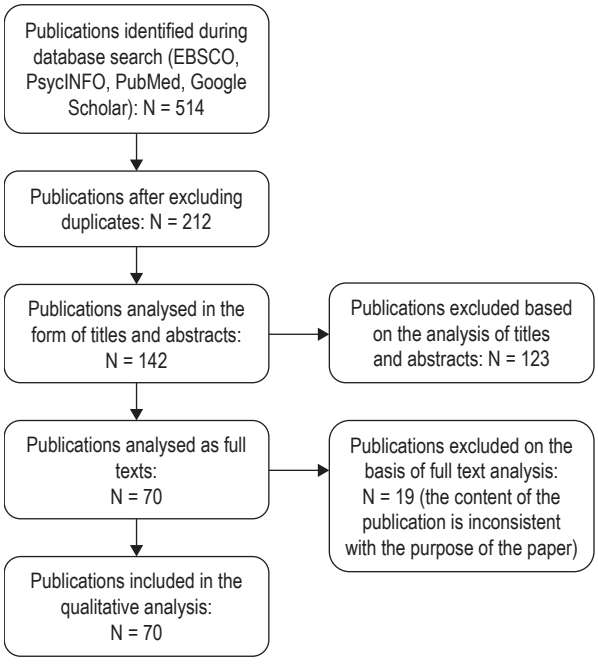

Figure 1. Summary of the research search and selection process in the PRISMA scheme.

The attachment relation and the pathogenesis of positive symptoms – the Liotti-Gumley theoretical model

According to the attachment theory [5], the memories of interactions with a parent are the grounds for forming the future operating models. The internal operating models are cognitive structures based on generalized memories of prior interactions with an attachment figure. They enable formulation of expectations regarding the future reactions of the attachment figure to the child's need for forming bonds. In the case of the safe bond style, the internal operating models create an inner sense of availability of help and encouragement from a significant other. In the case of non-safe attachment, the internal operating models create a conviction that the attachment figure will remain unapproachable, or will react negatively to a request for help [6]. The Liotti-Gumley model [4] suggests that the "mistrustful" internal operating models may increase the susceptibility for developing borderline personality disorders, PTSD, dissociative disorders and schizophrenia. A strong activation of the attachment system (e.g. caused by an actual or imaginary separation with the minder, as well as traumatic experience) may lead to a collapse of the controlling strategy and manifestation of the suppressed attachment needs. The consequences – according to Liotti and Gumley [4] – are positive and dissociative symptoms.

Among those suffering from psychoses, including schizophrenia, the prevalence of a distancing attachment style (high level of avoidance and low level of anxiety in the attachment relation) is assessed between 48% and 71%, while in healthy individuals it is 27% [7, 8]. The absorbed attachment style (low level of avoidance and high level of anxiety) concerns 12-20% of the psychotic patients, compared to 19% in healthy individuals. A safe attachment style (low level of avoidance and anxiety) is manifested by about 27-32% of the psychotic patients and about 58% of healthy individuals [7, 9]. These data indicate that the non-safe attachment styles affect schizophrenic patients to a greater extent than in the case of healthy individuals. Research [10] also suggests a relationship between the evading attachment style and positive symptoms. Therefore, the analysis of the empirical data supports

the premises of the Liotti-Gumley model [4], where a non-safe attachment relation is a potentially permanent factor of susceptibility to the occurrence of positive symptoms in the future. According to the researchers [4], this susceptibility is related to the experience of post-traumatic dissociative symptoms.

HALLUCINATIONS AND DISSOCIATIVE SYMPTOMS

A study [11] carried out on schizophrenic outpatients demonstrated that 85% of them fell victim to violence in their childhood. Moreover, it was demonstrated that the prevalence of sexual abuse experienced by schizophrenic patients was 38%, compared to 10% in the healthy population [12]. According to the study [13], schizophrenics had more traumatic experience in the attachment relation than the healthy individuals. Numerous metaanalyses and study reviews emphasize the significance of early childhood traumatic events in the development of psychotic disorders [14, 15]. Traumatic experience are also related to the occurrence of quasi-psychotic experience in healthy individuals [16, 17], and among those at risk of psychosis [18]. Moskowitz [19] claims that hallucinations, speech impediments and catatonic behaviours can be at least partially attributed to the consequences of traumatic experience, such as: depersonalization, derealization, dissociative behaviours and flashbacks.

Traumatic events in the attachment relation may cause memory disorders [20] and hallucinations, including visual, auditory and olfactory hallucinations [21]. These symptoms may occur both in post-traumatic and psychotic disorders. Auditory hallucinations in the case of dissociative disorders may be traumatic memories. For instance, the voices heard by the patients may be the comments the victim head during the abuse they experienced [22]. Both visual and auditory hallucinations among post-traumatic patients may take on a form of flashbacks to the traumatic events [23]. Studies revealed that 46-67% of patients with acute psychotic symptoms also show symptoms of post-traumatic stress disorder PTSD [24]. Positive symptoms in schizophrenia are bizarre and unreal, without any reflection in the real life [25]. Therefore, when diagnosing schizophrenia, it is justified to verify whether the delusions and hallucinations indicate emotionally-processed traumatic events. This diagnosis also involves the choice of the appropriate pharmacotherapy and psychotherapeutic interventions [21]. The separate categorization of positive and dissociative symptoms does not mean they do not contribute to the clinical picture. It is possible that dissociative symptoms affect the pathogenesis of auditory hallucinations in schizophrenia. Patients suffering from the schizophrenia spectrum disorders both manifest a higher intensity of dissociative tendencies and experience a childhood trauma more often than healthy individuals [13, 14]. Moreover, it was demonstrated that dissociation in this group of patients contributes to the positive relation between childhood traumatic experience and the susceptibility to hallucinations [26]. Studies [27] also demonstrated that the dissociative processes are related to psychotic experience, including hallucinations, which, in turn, are related to the experience of sexual abuse. Compulsions and PTSD symptoms are also mediators in the relation between childhood sexual abuse and auditory hallucinations [28]. This may indicate that dissociative symptoms and traumatic experience in attachment relationship contribute to the pathogenesis of auditory hallucinations.

COGNITIVE CONCEPTUALIZATION OF AUDITORY HALLUCINATIONS

According to the Liotti-Gumely model [4], another factor affecting the development of positive schizophrenic symptoms are the patient's cognitive deficits. Studies [29, 30] suggest that traumatic experiences are related to cognitive distortions, which is connected with the susceptibility to psychosis. Memory deficits play a particular role in this process [31]. Problems remembering traumatic experience may stem from dissociative disorders and result in an inability to recognize the experienced intrusions as arising from one's own past. Still, the relation between dissociative amnesia with the interpretation of traumatic intrusions has not been confirmed empirically.

In a hitherto-unverified model, Liotti and Gumely [4] claim that individuals experiencing

positive symptoms manifest so-called traumatic intrusion source monitoring errors. This means that these individuals often interpret the intrusions of their traumatic experience as coming from the outside (e.g. auditory hallucinations). Although this hypothesis is interesting, there are no studies which would confirm it explicitly. However, this theory is supported by the cognitive models of psychotic symptoms [32]. They work on the assumption that psychotic symptoms may result from deficits in the scope of monitoring the sources of own mental states. For instance, the patients may wrongly believe that their thougts originate from strangers.

Empirical studies – according to the cognitive models – indicate that schizophrenic patients demonstrate source monitoring errors [33]. Difficulties in the scope of differentiating between imagination and reality may already occur at the early stages of the psychosis development [29]. These deficits are a cognitive risk factor for the development of psychotic symptoms. It was demonstrated that the source monitoring errors and hallucinations are related [34]. The links between auditory hallucinations and source monitoring deficts are confirmed by meta-analyses [35, 26], but there are no studies indicating their connection with traumatic intrusions. Nevertheless, the Liotti-Gumely theoretical model [4] certainly outlines new directions for studies, which in the future may contribute to the inclusion of post-traumatic disorder symptoms in the pathogenesis of positive symptoms.

Attachment relation and auditory hallucinations from the cognitive perspective

Studies suggest a connection between the evading and anxiety attachment style and the positive symptoms. Researchers demonstrated that these relations are mediated by a low sense of self-esteem [37]. The low self-esteem, in turn, contributes to negative opinions of "I", as part of the internal operating models for attachment. These models are defined as cognitive structures which are activate in relational situations. These structures define „I", „others", and the relation between them [5]. Studies indicate that the contents of the hallucinations related to the criticism of "I" and rejection are related to intensification of evasive behaviours in attachment relations [39]. The internal operating models of non-safe attachment are also activated at the level of convictions and automatic thoughts. In the cognitive conceptualization, automatic thoughts related to low self-esteem (e.g. I'm a loser) are wrongly identified by the hallucinating individuals as originating from the outside (auditory hallucinations) [1].

The researchers also emphasize the convictions that the hallucinating individuals carry about the voices they hear. These convictions may impact the affect and behaviour stronger than the contents of the auditory hallucinations themselves. When the voices are intrusive and unpleasant, the patients react to them as they would to any other irritating manifestation. This activates convictions such as: I can't take it anymore", „I can't handle them" [1]. Among individuals with non-safe operating models of attachment, these convictions could be: "I can't count on anyone's help and understanding", "The voices are hostile towards me" [5, 4]. The relation between non-safe attachment styles with the distress felt when experiencing auditory hallucinations is mediated by the intensification of the belief that the imaginary voices are hostile [39]. This, in turn, may contribute to depressive symptoms, increase the level of distress and anger. Studies [39] also suggest a relation between the evading attachment style with the sense of obtrusiveness of the imaginary voices and the perceptible sense of threat towards them. Analysis of the studies suggests that the negative convictions about intrusive hallucinations could be the mediator in this relation [1].

A relatively new area of research is the perception of auditory hallucinations in the context between the hallucinating individual and the hallucinations themselves. Relations with the voices – just like relations with other people – can be positive, ambivalent or negative [1]. These relations may involve activation of the internal operating models for attachment [5]. Studies suggest that for some people, the voices they hear can be a source of consolation, as well as companionship. It was also demonstrated that some people experience loss once the auditory hallucinations become less intensive or completely absent [40, 41]. Attachment to the imaginary voices becomes more likely when other social rela-

tions vanish [42]. Disappearance of interpersonal contacts is frequent during schizophrenia and requires introduction of cognitive-behavioural trainings in social competence for the patients.

Non-safe attachment styles play an important role in the pathogenesis of auditory hallucinations. Liotti and Gumley [4] suggest that traumatic experience in the attachment relation and the subsequent dissociative symptoms increase the risk of developing positive symptoms. The attachment styles are connected not only with the relation to the imaginary voices and the ways of handling them, but also with the very contents of the auditory hallucinations [1, 38]. These factors, in turn, affect the intensity of the distress [39]. Attachment relations can also be described at the level of biological processes. These processes – such as activation of mesolimbic dopaminergic pathways – significantly affect the development of positive symptoms [1].

Attachment and biological determinants of auditory hallucinations

Dopamine (3,4-dihydroxyphentylamine), apart from adrenaline and noradrenaline, is classified as one of neurotransmitters called catecholamines [43]. Studies [44] carried out with an active magnetic resonance indicate dopaminergic activity within the reward system as a response to stimulation activating protective behaviours (e.g. by exposure to photographs of a child). It was also demonstrated that there is a connection between neuronal activation of mesolimbic dopaminergic pathways and the attachment relation in close relationships. Researchers [45, 36] demonstrated that the enamoured in partnership – after exposure to a photograph of the partner – are characterized by higher dopaminergic activity. This activity is increased in such areas as the anterior cingulate cortex and the ventral tegmental area. Moreover, a non-safe attachment relation in childhood is connected with a hyperactive hypothalamic-pituitary-adrenal axis. This, in turn, is related to an increased dopamine synthesis and its dysregulation in the stratum area [47]. After multiple negative stimulations in the attachment relation, there is a noticeable reinforcement of the dopaminergic response. This reinforcement was called sensitisation. This phenomenon is confirmed by animal testing. Breeding rodents in isolation causes them to produce more dopamine in the stratum [48]. It was also found that, in rodents, acute stressors activate transmission of dopamine in the stratum, causing its release and increased synthesis. For instance, an instability caused by multiple replacement of the cage companions and problems in relations with other specimen are related to an increased sensitivity to amphetamine and an increased release of dopamine in the stratum structures. Researchers [49] agree that changes within the dopaminergic system play a significant role in the attachment relation.

One of the most common theories of the positive symptom pathogenesis is the dopamine theory. It assumes that the causes of positive symptoms (delusions, hallucinations) stem from an excessive activity in the mesolimbic dopaminergic pathway [1]. Autopsies of the brains of schizophrenics revealed a significant increase in the quantity of dopamine receptor areas, particularly of type D2 [50]. The dopamine hypothesis is also supported by the effect of substances which increase the dopamine activity (i.e. amphetamine, l-dopa, cocaine) on positive symptoms [51]. Using selective radiotracers for dopamine receptors D2/3 after giving amphetamine to schizophrenic patients demonstrated that an increased release of dopamine is related to a higher induction of positive symptoms. On the other hand, reducing the dopamine levels after stopping its synthesis involves a reduction of the positive symptoms [52].

Dopamine synthesis – apart from the attachment relation – is also connected with genetic factors. Dopamine-related genes have been the subject of numerous studies in schizophrenia, particularly the dopamine receptor genes and Catechol-O-Methyl-Transferase (COMT), a key enzyme degrading dopamine [53]. Pre-clinical evidence indicate that an altered function of numerous genes upsets the dopamine system, e.g. mice with a DISC1 trauma demonstrated an increased behavioural response and increased dopamine release in the stratum after methamphetamine [54]. The TCF4 transcription factor also affects the dopamine system, activating transcription of tyrosine hydroxylase 99 [55]. The next step to explain the relationship between attachment experiences and dopamine activity are epi-

genetic changes in genes regulating its activity. In schizophrenia, epigenetic modifications have been observed, such as: DNA hypermethylation of the RELN gene promoter [56], hypermethylation of the SOX10 gene promoter [57], H3L4 hypomethylation of the GAD1 gene promoter [58], and DNA hypomethylation of the COMT gene promoter [59]. However, methylation of the OXTR, NR3C1, FKBP5, SLC6A4 and HTR3A genes is associated with insecure attachment relationship [60]. Currently, it has not been found that epigenetic processes in schizophrenia are identical with epigenetic modifications resulting from insecure attachment relationships. These hypotheses still require verification. A common genetic risk factor for the development of both positive symptoms in schizophrenia and the avoidant attachment style is the Val158Met polymorphism of the COMT gene [61]. The DRD2 gene was also found to be related to the intensity of anxiety in the relationship of attachment and the level of dopamine [62].

Interaction between the genetic and environmental factors at early stages of life is reflected in the epigenetic modifications that promote the gene expression patterns during brain development [63]. The Shore conception [64] emphasizes that lack of trustful attachment in childhood has a direct effect on the maturing right hemisphere during its critical growth period, causing its immaturity. Researchers [65] demonstrated that children with safe attachment styles, compared to those with non-safe attachment styles, were characterized by a higher volume of grey matter in the temporal sulcus, the superior temporal gyrus and the temporoparietal junction. A reduced volume of the grey matter in the temporal lobe is related to increased auditory hallucinations [66]. The pathomechanism connecting traumatic experience in the attachment relation with changes in the brain is explained by decreased neuron activation in neglected children [67]. About 30% neurons die between the ages of 7 and 14. The child's experiences dictate which synaptic links will die, and which will grow [68]. This thesis is also supported by experiments on laboratory animals [69]. Rats growing in an enriched environment have more synaptic spines on the dendrites than those growing in an impoverished environment. The latter lose a number of synaptic spines [69]. Neglected children experience no positive stimulation from the parents, which inhibits the development of neuronal areas, particularly the structures related to the regulation of affective states [64, 70]. These children experience chronic stress. Difficult experience at early stages of life – by the effects of cortisol and glutamate – are related to the atrophy of neuronal areas. In one of the studies [70], children with PTSD had, on average, 7% smaller brains compared to children from the control group. Children with PTSD also had enlarged cerebral ventricles and smaller corpus callosum. Therefore, these studies suggest that difficult experiences in the attachment relation during early childhood may be related to the atrophy of neuronal areas, including the temporal areas, involved in auditory hallucinations [66].

DISCUSSION AND CONCLUSIONS

The current state of knowledge suggests a relationship between auditory hallucinations and an insecure attachment relationship. However, the pathomechanism of this relationship has not yet been explained. Research suggests that this relationship is mediated by several groups of factors. The first group of mediators are traumatic experiences and the resulting dissociative symptoms [21, 22, 26, 27, 28]. The second group of factors mediating the genesis of symptoms are cognitive distortions [35, 36]. According to researchers, relational experiences may influence beliefs about the voices heard [37, 39]. The third group are factors related to dopaminergic activity. Higher dopaminergic activity is associated with both the attachment relationship [44, 45, 46] and the pathophysiology of hallucinations [50, 51,52]. Another group of factors mediating the relationship of attachment and auditory hallucinations are genetic factors and epigenetic modifications. Insecure attachment relationship is associated with dopamine-related genes such as DRD2 and COMT [61, 62]. Moreover, studies suggest that the insecure relationship is associated with epigenetic processes such as methylation of genes such as: OXTR, NR3C1, FKBP5, SLC6A4 and HTR3A [60]. Epigenetic modifications have also been observed in schizophrenia [56-59]. However, these are not the same modifications as those confirmed to be associated

with an insecure attachment relationship. Future research should be focused on looking for epigenetic processes mediating between the relationship of attachment and the development of positive symptoms. The last group of factors mediating the relationship between auditory hallucinations and the attachment relationship are changes in neural structures. Research suggests a relationship between traumatic experiences in the attachment relationship with gray matter volume [65], also in temporal areas, the atrophy of which correlated with the severity of auditory hallucinations [66].

The genesis of auditory hallucinations is multifactorial. It is associated with dysfunction within the temporal lobe [66] and dysregulation in the dopaminergic system [50-52]. Both genetic [53-55] and environmental factors [47, 48] play an important role in these changes. The attachment relationship is related to both the dopaminergic system [44-46], structural changes within the nervous system [64, 65] and the severity of auditory hallucinations [37, 39]. The diagnosis of internal operating models often allows for an insight into the relation of the patient with the voices being heard by him / her [40, 42]. Attachment styles are also related to the content of auditory hallucinations and beliefs about them [38, 39]. Future research should verify the hypothesis about the influence of post-traumatic intrusions on the development of positive symptoms [4]. According to this hypothesis, difficult relational experiences can manifest themselves through the styles of attachment and the content of the hallucinations experienced. Verification of these assumptions will allow not only to create more accurate models of the pathogenesis of auditory hallucinations, but also to improve therapeutic interventions.

REFERENCES

1. Beck AT, Rector NA, Stolar N, Grant P. Schizophrenia. Cognitive Theory, Research, and Therapy. Guilford: The Guildford Press; 2009.
2. Johns LC, Kompus K, Conell M, Humpson C, Lincoln TM, Longden E, Preti A, Alderson B, Badcock JC, Cella M, Fernyhough C. Auditory Verbal Hallucinations in Persons With and Without a Need for Care. Schizophrenia Bulletin. 2014; 40(4): 255-264.
3. Waters F, Fernyhough C. Hallucinations: A Systematic Review of Points of Similarity and Difference Across Diagnostic Classes. Schizophrenia Bulletin. 2017; 43(1): 32-43.
4. Liotti G, Gumley A. An Attachment Perspective on Schizophrenia: The Role of Disorganized Attachment, Dissociation and Mentalization. In: Moskowitz A, Schafer I, Dorahy MJ, editors. Psychosis, Trauma and Dissociation. New York: John Wiley & Sons; 2008. p. 117-134.
5. Bowlby J. A secure base: Parent child attachment and healthy human development. New York: Basic Books; 1988.
6. Liotti G. Trauma, Dissociation, and Disorganized Attachment: Three Strands of a Single Braid. Psychotherapy: Theory, research, practice, training. 2004; 41: 472-486.
7. Harder S. Attachment In Schizophrenia – Implications for Research, Prevention, and Treatment. Schizophrenia Bulletin. 2014; 40(6): 1189-1193.
8. Bakermans-Kranenburg MJ, Van IJzendoorn MH. The first 10000 Adult Attachment Interviews: distributions of adult attachment representations in clinical and non-clinical groups. Attachment & Human Development. 2009; 11(3): 223-263.
9. Gumley AI, Schwannauer M, Macbeth A. Insight, duration of untreated psychosis and attachment in first-episode psychosis: prospective study of psychiatric recovery over 12-month. British Journal Psychiatry, 2014; 205: 60–67.
10. Varghese D, Scott J, McGrath J. Correlates of delusion-like experiences in a non-psychotic community sample. Australian and New Zealand Journal of Psychiatry, 2008; 42: 505–508.
11. Chrzątkowski, S. Związki między psychozami a traumą interpersonalną. Wiadomości Psychiatryczne, 2007; 10(4): 249-256.
12. Worr CJ, Partridge IM. The prevalence of a history of childhood sexual abuse in an acute adult in patient population. Child Abuse Neglect, 1996; 20: 67-872.
13. Bebbington PE, Bhugra D, Brugha T. Psychosis, victimization and childhood disadvantage: eviodence from the second British National Survey of Psychiatric Merbidity. British Journal Psychiatry, 2004; 1: 185-220.
14. Misiak B, Krefft M, Bielawski T, Moustafa AA, Sasiadek MM., Frydecka D. Toward a unified theory of childhood trauma and psychosis: A comprehensive review of epidemiological, clinical, neuropsychological and biological findings. Neuroscience & Biobehavioral Reviews, 2017; 75: 393-406.
15. Gibson LE, Alloy LB, Ellman LM. Trauma and the psychosis spectrum: a review of symptom specificity and explanatory mechanisms. Clinical Psychology Review, 2016; 49: 92-105.
16. McGrath JJ, Saha S, Lim CC, Aguilar-Gaxiola S, Alonso J, Andrade LH, Bromet EJ, Bruffaerts R J, Cardoso G. Trauma and psychotic experiences: transnational data from the World Mental Health Survey. The British Journal of Psychiatry, 2017; 117(20): 55-69.
17. Sun MX, Zhang W, Guo RA, Li Y, Mwansisya TE, Zhou L, Liu C, Chen X. Psychotic-like experiences, trauma and related risk factors among “left-behind” children in China. Schizophrenia Research, 2017; 181: 43-48.

18. Thompson AD, Nelson B, Yuen HP, Lin A, Amminger GP, McGorry PD, Wood SJ, Yung AR. Sexual trauma increases the risk of developing psychosis in an ultra high-risk "prodromal" population. Schizophrenia Bulletin, 2014; 40(3): 697-706.
19. Moskowitz AK, Read J, Farrelly SI. Are psychotic symptoms traumatic in origin and dissociative in kind? In: Paul FD, John AO, editors. Dissociation and the dissociative disorders. DSM-V and Beyond. New York: Rutledge Taylor & Francis Group; 2009. p. 521-545.
20. Chu JA, Frey LM, Ganzel BL, Matthews JA. Memories of childhood abuse: dissociation, amnesia, and corroboration. The American Journal of Psychiatry, 1999; 156: 749-755.
21. Świerkosz A, Frankowska A, Turek A, Siwek M, Balachowska D, Dudek D. Dissociative or psychotic disorders? Stupor in a 23-year old female patient. Psychiatry, 2015; 12(3): 201–207.
22. Austin JA. The connection beetwen psychosis, trauma and dissociation: an explory study involving patients in forensic mental health settings. Edinburgh: The University of Edinburg; 2011.
23. Spigel D. Dissociative disorders in DSM-5. Depression and Anxiety, 2011; 28: 824-852.
24. Frame L, Morrisom A. Causes of PTSD in psychosis. Archives of General Psychiatry, 2001; 58: 305–306.
25. Gronkowski M, Śpila B, Gronkowska E, Karakuła H, Przywara G, Pawęzka J. Zaburzenia psychiczne u pacjentki wykorzystanej seksualnie w dzieciństwie – schizofrenia czy objawy złożonego PTSD (CPTSD). Psychiatria Polska, 2007; 41(4): 561-570.
26. Varese F, Barkus E, Bentall RP. Dissociation mediates the relationship between childhood trauma and hallucination-proneness. Psychological Medicine,2012; 42(5): 1025-1036.
27. Kilcommons AM, Morrison AP. Relationships between trauma and psychosis: an exploration of cognitive and dissociative factors. Acta Psychiatrica Scandinavica, 2005; 112(5): 351-359.
28. McCarthy-Jones S. Post-Traumatic Symptomatology and Compulsions as Potential Mediators of the Relation Between Child Sexual Abuse and Auditory Verbal Hallucinations. Behavioral and Cognitive Psychotherapy. 2018; 46(3): 318-331.
29. Gawęda Ł, Li E, Lavoie S, Whitford TJ, Moritz S, Nelson B. Impaired action self-monitoring and cognitive confidence among ultra-high risk for psychosis and first-episode psychosis patients. European Psychiatry, 2018; 47: 67-75.
30. Varese F, Bentall RP. The metacognitive beliefs account of hallucinatory experiences: a literature review and meta-analysis. Clinical Psychology Review, 2011; 31(5): 850-864.
31. Vinogradov S, Willis-Shore J, Poole JH. Clinical and neurocognitive aspects of source memory errors in schizophrenia. American Journal of Psychiatry, 1997; 154: 1530–1537.
32. Waters F, Allen P, Aleman A, Fernyhough C, Woodward TS, Badcock JC, Barkus E, Johns L, Varese F, Menon M. Auditory hallucinations in schizophrenia and nonschizophrenia populations: a review and integrated model of cognitive mechanisms. Schizophrenia Bulletin, 2012; 38(4): 683-693.
33. Keefe RS, Arnold M, Bayen U, Harvey P. Source monitoring deficits in patients with schizophrenia; a multinomial modelling analysis. Psychological Medicine, 1999; 29(4): 903-914.
34. Allen PP, Johns LC, Fu CHY, Broome MR, Vythelingum GN, McGuire PK. Misattribution of external speech in patients with hallucinations and delusions. Schizophrenia Research,2004; 69(2): 277-287.
35. Waters F, Woodward T, Allen P, Aleman A, Sommer I. Self-recognition deficits in schizophrenia patients with auditory hallucinations: a meta-analysis of the literature. Schizophrenia Bulletin. 2012; 38(4): 741-750.
36. Brookwell M, Bentall R, Varese F. Externalizing biases and hallucinations in source-monitoring, selfmonitoring and signal detection studies: a meta-analytic review. Psychological Medicine. 2013; 43(12): 2465-2475.
37. Wickham S, Sitko K, Bentall RP. Insecure attachment is associated with paranoia but not hallucinations in psychotic patients: the mediating role of negative self-esteem. Psychological Medicine. 2014; 45: 1-13.
38. Berry K, Wearden A, Barrowclough C, Oakland L, Bradley J. An investigation of adult attachment and the nature of relationships with voices.British Journal of Clinical Psychology. 2012; 51(3): 280–91.
39. Robson G, Mason O. Interpersonal processes and attachment in voice hearers. Behavioral Cognitive Psychology. 2015; 43(6): 655–68.
40. Chin JT, Hayward M, Drinnan A. Relating to voices: exploring the relevance of this concept to people who hear voices. Psychology Psychotherapy. 2009; 82(1): 1–17.
41. Moritz S, Favrod J, Andreou C, Morrison AP, Bohn F, Veckenstedt R. Beyond the usual suspects: positive attitudes towards positive symptoms is associated with medication noncompliance in psychosis. Schizophrenia Bulletine. 2013; 39(4): 917–22.
42. Berry K, Varese F, Bucci S. Cognitive Attachment Model of Voice: Evidence Base and Future Implications. Frontiers Psychiatry. 2017; 8: 111-124.
43. Woo-Yeon P. An affective high-performence liquid chromatographic-mass spectrometric assay for cetecholamines, as the N(O,S)-ethoxycarbonylsthyl esters, in human urine, Chromatographia, 2006; 64: 731-737.
44. Strathearn L. Maternal neglect: oxytocin, dopamine and the neurobiology of attachment. Journal of Neuroendocrinology. 2011; 23(11): 1054-1065.
45. 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.

46. Bartles A, Zeki S. The neural correlates of maternal and romantic love. Neuroimage. 2004; 21(3): 1155-1166.
47. Walker EF, Diforio D. Schizophrenia: a neural diathesis-stress model. Psychological Review. 1997; 104(4): 667-685.
48. El-Khodor BF, Boksa P. Effects of birth insult and stress at adulthood on in vivo tyrosine hydroxylase and tryptophan hydroxylase activities in rat brain. Synapse. 2003;47(1): 87–89.
49. Gordon I, Martin C, Feldman R, Leckman JF. Oxytocin and social motivation. Developmental Cognitive Neuroscience. 2011; 1(4): 471-493.
50. Crow TJ. Two dimensions of pathology in schizophrenia: Dopaminergic and non-dopaminergic. Psychopharmacology Bulletin, 1982; 18: 22-29.
51. Seligman MEP, Walker MEP, Rosenhan DL. Abnormal Psychopatholology. Norton: Norton & Co Ltd Press; 2001.
52. Abi-Dargham A, Rodenhiser J, Printz D. Increased baseline occupancy of D2 receptors by dopamine in schizophrenia. Procedings of the National Academic of Sciences of the USA., 2000; 97(14): 8104–8109.
53. Saqud M, Muck-Seler D, Mihalievic-Peles A, Vakusan-Cusa B. Catechol-O-methyl transferase and schizophrenia. Psychiatria Danubina. 2010; 22(2): 270-274.
54. Niwa M, Kamiya A, Murai R. Knockdown of DISC1 by in utero gene transfer disturbs postnatal dopaminergic maturation in the frontal cortex and leads to adult behavioral deficits. Neuron, 2010; 65(4): 480–490.
55. Yoon SO, Chikaraishi DM. Isolation of two E-box binding factors that interact with the rat tyrosine hydroxylase enhancer. Journal of Biological Chemistry, 1994; 269(28): 18453–18462.
56. Grayson DR, Jia X, Chen Y. Reelin promoter hyperme-thylation in schizophrenia. Proceedings of the National Academy Sciences of the USA, 2005; 102: 9341–9346.
57. Iwamoto K, Bundo M, Yamada K. DNA methylationstatus of SOX10 correlates with its downregulation and oligo-dendrocyte dysfunction in schizophrenia. Journal of Neuroscience, 2005; 25: 5376–5381.
58. Huang HS, Akbarian S. GAD1 mRNA expression and DNA me-thylation in prefrontal cortex of subjects with schizophrenia. PLoS ONE, 2007; 2: 800-809.
59. Abdolmaleky HM, Cheng KH, Faraone SV. Hypome-thylation of MB-COMT promoter is a major risk factor for schizophrenia and bipolar disorder. Human Molecular Genetic, 2006; 15: 3132–3145.
60. Craig F, Tenuta F, Rizzato V, Costabile A, Trabacca A, Montirosso R. Attachment-related dimensions in the epigenetic era: A systematic review of the human research. Neuroscience & Biobehavioral Reviews. 2021, 125: 654-666.
61. Erkoreka L, Zumárraga M, Macías I, Angel Gonzalez-Torres M. The COMT Val158Met polymorphism exerts a common influence on avoidant attachment and inhibited personality, with a pattern of positive heterosis. Psychiatry Research. 2018; 262: 345–347.
62. Gillath O, Shaver PR, Baek JM, Chun DS. Genetic correlates of adult attachment style. Personality and Social Psychology Bulletin. 2008; 34: 1396–1405.
63. Daskalakis NP. Schizophrenia in the Spectrum of Gene–Stress Interactions: The FKBP5 Example, Schizophrenia Bulletin, 2015; 41(2): 323–329.
64. 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.
65. Leblanc E, Degelih F, Deneault V, Beachamp MH, Bernier A. Attachment security in infancy: A preliminary study of prospective links to brain morphometry in late childhood. Frontiers in Psychology, 2017; 8: 2141-2146.
66. Gaser C, Nenadic I, Volz H, Buchel C, Sauer H. Neuroanatomy of Hearing Voices: A frontotemporal brain structural abnormality associated with auditory hallucinations in schizophrenia. Cerebral Cortex, 2004; 14(1): 91-96.
67. Lessen-Firestone J. Brain Development. New York: Newsweek Special Edition; 1997.
68. Eliot L, What's Going On in There? How the Brain and Mind Develop in the First Five Years of Life Quotes. New York: Bantam Dell Publishing Group; 2010.
69. Greenough WT. Experiential modification of the developing brain. American Scientist. 1975; 63: 37-46.
70. De Bellis MD, Keshavan MS, Clark DB, Caset BJ, Giedd JN, Boring AM, Frustaci K, Ryan ND. Developmental Traumatology Part II: Brain Development. Biological Psychiatry. 1999; 45: 1271-1284.