Psychiatr. Pol. 2014; 48(3): 465–475
PL ISSN 0033-2674
www.psychiatriapolska.pl

Neurocognitive and social cognitive deficits in patients with anorexia nervosa

Dorota Kułakowska[1], Katarzyna Biernacka[2], Ewelina Wilkos[1], Filip Rybakowski[2], Katarzyna Kucharska-Pietura[1]

Department of Psychiatric Rehabilitation, Institute of Psychiatry and Neurology in Warsaw
Head: dr hab. n. med. Katarzyna Kucharska-Pietura

Department of Child and Adolescent Psychiatry, Institute of Psychiatry and Neurology in Warsaw
Head: dr hab. n. med. Filip Rybakowski

Summary

In the first part of the article the authors present a set of the actual concepts explaining problems of cognitive functions and social cognition currently observed in patients with anorexia nervosa (AN). It is possible; through the neuroimaging research, to get better understanding of the brain specifics in these individuals. Even though, the AN remains a disease with very complex and multifactorial etiology which remains a huge medical challenge. Currently, popular is the view that takes into consideration the integrating role of the insula and subcortical structures (such as hippocampus, amygdala, thalamus) in the regulation of cognitive and emotional processes in people suffering from AN. There is still an open problem, however, of the selection of therapeutic interventions targeting these deficits. The second part of the article presents the attempt to describe deficits in neurocognitive and social cognition in people with AN occurring prior to illness, during and after the recovery. Particular attention has been paid to the most frequently described in the literature – neurocognitive deficits such as rigidity of thinking, weak central coherence, and deficits in social cognition, including mental processes of perception and expression of emotions, disorders of the theory of mind (ToM) and empathy. The results of previous studies, their scarcity in Poland, do not give a satisfactory answer to the question whether the above mentioned disorders are a feature of endophenotype or condition in an episode of the disease. Research point to the more permanent nature, which may be more resistant to therapeutic modifications.

Key words: anorexia nervosa, neurocognitive deficits, social cognitive deficits

Introduction

In the last decade there has been a significant increase in the amount of research focused on the assessment of neurocognitive and social cognitive deficits in anorexia nervosa (AN). Observed neurocognitive deficits relate primarily to disturbances

in the way of thinking and impaired central coherence (the inability to merge the information and excessive focus on details).

Deficits in social cognition relate to impaired perception and emotional expression, reduced ability to interact and predict the intentions of others, which basically covers the theory of mind (ToM). These disorders may be present before the diagnosis of the disease, during the illness and in the recovery period [1-3]. It is still not clear whether they occur in all cases if AN, and what is their neurobiological substrate. Some of the authors have not confirmed the occurrence of neurocognitive deficits [4, 5] and social cognition in AN [6, 7], although the vast majority of research indicate their presence and persistence despite remission of symptoms and normalization of body weight. The argument indication that neurocognitive deficits are characteristic – is their stability during the illness [1] and the presence in a family, for example in healthy sisters of patients with AN [8, 9]. In addition, people suffering from refractory chronic form of the disease, characterized by the severity of the analytical cognitive style along with social difficulties, emotional and weakening of the global information integration [2].

Models accounting for aetiopathogenesis of the neurocognitive and social cognitive deficits

It was suggested that people suffering from eating disorders display a 'significant inability to understand emotions' as well as show neurocognitive deficits. Explanations of the above mentioned state were searched for in neurobiological models: neurodevelopmental, hormonal, genetic, neurotransmitter and psychological [10]. To date there existed a dominating preconception of environmental conditioning in anorexia nervosa [11] whereas neurobiological mechanisms are still inadequately researched and remain a source of many controversies. Disorders of neurobiological nature underlie the neuropsychological deficits, which are considered to be the factors increasing susceptibility of developing anorexia nervosa [12] and a permanent feature independent of the severity of the disease [13].

Recently, a significant role in regulating spheres of hunger and satiety is attributed to the insula [14]. The functions of the insula in anorexia nervosa play a crucial role in a close localization of the areas regulating emotions and it seems that they play a vital role with regard to the perception of taste, regulation of the digestive processes, appetite and eating. Moreover, connection of the insula with the somatosensory cortex improves the processes related to monitoring individual body state as well as its subjective perception. Furthermore, it is connected with a perceivable degree of experiencing pain, disgust and distaste. In the studies with the use of a functional magnetic resonance (fMRI) a smaller neural activity of the insula was observed among people suffering from anorexia nervosa in comparison to the control group [15] as well as atypical activity of this area during performance of cognitive tasks [12].

In the case of anorexia nervosa there was found a higher volume of neural tissue in the areas of the brain responsible for the perception of tastes and food control

in comparison to healthy peers. It is believed that a larger volume of these structures may be responsible for the fact that patients with anorexia nervosa avoid eating and are able to starve themselves. Differences among healthy persons and patients with anorexia nervosa were attributed to the size of the grey and white matter. The structure of the brain as well as the integrity of the white matter (diffusion tensor imaging DTI) among teenage patients diagnosed with anorexia nervosa was evaluated using magnetic resonance imaging (MRI) [10]. The study showed that girls with anorexia nervosa had a larger volume of grey matter in the areas responsible for the perception of taste (the right part of the insula) regulating satiety and nutrition (left orbitofrontal cortex associated with information about the taste and smell of food and the cortex of both temporal lobes). The study confirmed a greater volume of the white matter in the temporal lobes and the changes in its organization in different areas of the brain (including the temporal lobes, the vault and the parietal lobe) in the group of the girls with anorexia nervosa in comparison to healthy subjects. In addition, the increased volume of grey matter in the orbitofrontal cortex was associated with a lack of perceived pleasure of sweet taste. The results obtained among adults with anorexia nervosa confirmed a higher volume of these areas, i.e. insula, and the orbitofrontal cortex regardless of their age.

From the neurobiological perspective the changes occurring in the brain of patients may predispose to the occurrence of eating disorders as well as associate with their maintenance [10]. In anorexia nervosa the differences in volume and activity of the insula – especially its right part – may be responsible for incorrect processing of tastes and a distorted perception of one's own body. As a result, people with anorexia nervosa are characterized by a distorted body image and recognize overweight in a situation of a distinctive underweight. In the group of patients with anorexia nervosa increased volumes of orbitofrontal cortex areas responsible for regulating satiety may be associated with the avoidance of food, and even if patients do reach for food they satisfy the feeling of satiety faster and eat less than healthy people.

McAdams and Krawczyk [16] in their study using functional magnetic resonance described reduced BOLD activity in the network of cognitive functions among anorexia nervosa patients in remission in comparison with the control group. The authors reported statistically significant intergroup differences in the activity of the right temporo-occipital junction. However, there were no differences between the two groups in the areas of activities related to spatial perception.

The proposed cognitive models point to the existence of deficits in the ability to change the cognitive approach – as a factor supporting the symptoms [17-19]. In addition, theoretical models show the relationship between the cognitive approach and intensification of obsessive – compulsive behaviors, beliefs connected with perfectionistic behavior [20-22]. Another factor explaining anorexia nervosa from the cognitive perspective [23] is a transdiagnostic model of eating disorders (TPB). In transdiagnostic models emphasis is placed on the overimportance ascribed to eating, body weight and body shape. Many people with eating disorders are not able to tolerate the perceived emotional states – anxiety, sadness, anger, therefore, they are managing with

the above mentioned problems through strategies involving vomiting and restrictive diets [24]. The models assume that the main function of the symptoms of the illness is the control of emotions and eventually their avoidance. In this model, the primary avoidance is associated with suppression of emotions and eating limitations.

Researchers from the Department of Child and Adolescent Psychiatry in IPiN [25] described four types of personalities found in anorexia nervosa subgroups: the personality perfectionistic-dependent, with a narcissistic personality with resistance; avoids personality and the personality of a well-functioning. Their findings show the relationship of personality to the overall functioning before illness and during her lifetime, with the severity of depressive symptoms, anxiety, OCD including symptoms and the course of treatment.

Etiological model of the formation of the above-mentioned deficits is explained in the view of neurodevelopmental processes. Anorexia nervosa is a disease with a complex, multifactorial aetiology, representing a large medical problem. According to the neurodevelopmental assumptions concept of abnormal development of the brain (mainly in the area of the insula) strongly associated with a genetic predisposition occurs in utero. Disorders of the regulatory mechanisms of brain neurotransmitters [26], the stress axis dysfunction and undergoing psychosocial stress (trauma) during childhood are conducive to the prospective vulnerability to developing anorexia nervosa. Brewerton et al. [27] excluded the impact of maternal diet during pregnancy on the dysfunctional development of CUN.

'Emotional awareness' develops as a result of complex neurodevelopmental processes and is a derivative of emotional and cognitive development. It is also the result of disturbances occurring during the processing and integration of cognitive schemes used to the development of complex emotional information [23]. So in the case of anorexia nervosa disruption may occur in 'emotional awareness' of what other people might feel.

Similarity of disorders in anorexia nervosa and autism spectrum has become a subject of growing interest of researchers in three main areas: a) the executive dysfunction, impaired cognitive flexibility (set shifting), b) weak central coherence, which is manifested by excessive attention to detail along with an impaired processing of global information, c) social dysfunction with particular attention to the difficulties in emotional empathy and theory of the mind (ToM). Genetic, neuroendocrinologic, neuroimaging and pharmacological studies suggest that dysfunctions of two nonapeptides in biological pathways – arginine vasopressin and oxytocin can affect social cognition disorders in humans and predispose to the occurrence of certain neuropsychiatric disorders, especially in the spectrum of autism.

Phenotypic traits, more fundamental than clinical recognition (recognition of emotions, understanding intentions of other people), may constitute the so called *endophenotypes* allowing the disclosure of a complex genetic architecture of psychiatric disorders. Variations with respect to the genes for the regulatory pathways AVP / OXT may cause different areas of functioning in social cognition and neurocognition in both groups of patients and/or in the control group.

Neurocognitive deficits

Neurocognition comprises of the following processes: memory, attention, visuospatial functions and broadly understood executive functions. Neurocognitive Deficits occurring in anorexia nervosa relate primarily to the switching pattern of cognitive disorders (mental), reduced mental flexibility [28], and set-shifting which may lead to the secondary problems developed beliefs change stiffness in thinking, i.e. switching impaired mental operations in response to changes in the environment [29]. According to the research [28, 29, 30, 31] cognitive functioning in AN becomes lowered and rigid. It can be concluded that the characteristic of the people of AN (especially the type of restriction) cognitive style is the result of deficits in executive functions.

Research confirm that described deficits in patients with AN, exhibit abnormal switching scheme [8, 9], as well as persistent cognitive rigidity [1]. One of the reasons behind the impairment of the ability of effective attention shifting between the tasks is a tendency to perseveration of the previously existing rules [32]. In addition, studies on patients with AN confirmed existing difficulties of central coherence associated with reduced global integration of information at an excessive concentration on details [33, 34], which significantly impedes cognitive functioning of patients.

On the basis of the conducted research on women with AN compared to their healthy female siblings and a control group of healthy volunteers, a conclusion has been taken that behavior pattern switching disorders [8, 9] are present among the patients and their siblings which is consistent with neurobiological models: genetic and neurodevelopmental.

Concentration on detail in cognitive style may be perceived as an impaired ability to process information from multiple sources and finding the relationship between them [33]; for example a patient can not perceive a meal in a broader perspective of recovery and focuses specifically on the analysis of its composition and calories. Lopez et al. [33] comply with the statement that AN patients have difficulties with global integration of information. Harrison et al. [35] claim that difficulties in this sphere were noticed only amongst patients in the acute phase of the illness. In turn, Roberts et al. [9] described that people with AN mainly experience an excessive focus on detail, which is also visible in the results of the patient's sane female siblings. This tendency may be connected to an excessive perfectionism, which is often present amongst the typical features of AN patients.

The moment of appearance of the deficit (i.e. before, on the beginning and during the illness), deficit stability (regardless the clinical state of the patient) and its sustainability (its presence during the recuperation period and after the full recovery) remain controversial. The literature of subject does not give a definite answer concerning the character of the impairment of the behavior pattern switching [29]. Neurocognitive Deficits in AN may be predisposing factors for developing AN, intensified with weight loss and disease duration [36], although persisting in patients with AN, compared with the control group [1], regardless of normalize BMI and symptomatic remission.

Social cognitive deficits

Social cognition is an ability to create mental representations of relations existing among people and using this ability in order to function effectively in the society as well as correct utilization of non-verbal signals in communication, e.g. mimics, gestures and tone of voice. People with anorexia nervosa have difficulty with emotional expressiveness, consequently they avoid situations in which expression of emotional states is required [37, 38, 39, 40]. The substantial body of literature on emotional processes proved that women with anorexia nervosa unreasonably block expression of the affect. Suppression of such emotions aims at a pursuit of maintaining positive, peaceable relations. Among anorexia nervosa patients an excessive emotional expression associated with a conviction that the experienced emotions would have unpleasant consequences [38]. Other researches proved that anorexia nervosa patients also attempt to suppress experiences of positive emotions [39]. Larger number of anorexia nervosa symptoms such as starving, concern for body shape and weight is connected with a high level of emotional suppression [40]. It was proven that anorexia nervosa patients can suffer from alexithymia coexisting with a lowered ability to read facial emotions [37].

Examples of social cognition difficulties observed among anorexia nervosa patients are as follows: inability to recognize the convictions and intentions of other people, i.e. deficits in the theory of mind, lack of compatibility of empirical response and the power of emotional expression, i.e. emotional expression disorders [41] as well as emotional perception disorders, i.e. impaired accuracy in guessing the emotions on the basis of voice and interpretation of mimic signals [42, 43]. The above mentioned deficits may be the cause of limited emotional conscience (meta-emotions) [44].

Emotional perception

Difficulties in identification of one's own and other people emotions may be the reason of problems in social interaction. The research points to emotional recognition deficits in the visual (facial) and auditory modality (emotion prosody) as typical for schizophrenia people in comparison to the group of sane participants [42, 43]. Those difficulties concern smaller precision in positive emotions recognition, such as happiness or surprise as well as negative emotions, such as anger, disgust, fear and sadness. People with anorexia nervosa provided answers after longer time than the sane participants [44]. What is more, they more often wrongly interpreted facial sadness when, in reality, a different emotion was expressed [45] which could be connected with a depressive mood dominant among the ill. Inability to perceive emotions may be connected with a domination of analytical thinking over the synthetic one, which can disrupt the complex process of emotional signal processing [46]. Nevertheless, the mechanism lying at the basis of the above mentioned deficits and the factors modifying them are still the subject of hypothesis.

Emotional expression

Emotions experienced by other people are inferred from their external manifestations, which facilitate social interactions constituting a specific code of communication. The key element of a coordinated means of emotional expression is mimics, which, during a conversation, can serve as an activator of benefits, on the other hand, being an unfavorable factor while emotional reaction is lacking (a reversed possibility is also taken into consideration). When the verbal message and the non-verbal expression are contradictory dysfunctions in emotional reactions can appear [47].

People with anorexia nervosa experience considerable difficulties in the emotional sphere and they attempt to eliminate the element of emotional expression as difficult and unpleasant in social interactions. Poorer emotional expression was expressed by anorexia nervosa patients in comparison with the control group and recovered patients which is confirmed by the research focusing on the fact that patients with anorexia nervosa show smaller accuracy in anger perception and they use avoidance as the means of diminishing the emotional states [48]. It is most probable that the patients who are concerned about unpleasant consequences do not accept the possibility of manifesting negative emotions [49]. Additionally, there was observed a correlation between the depressive symptoms and limitation of positive emotional expression among anorexia nervosa patients.

Theory of the mind

Theory of the mind, which is responsible for establishing multidimensional social interactions, is described as an ability to infer about the mental states of other people [1]. The research proves that efficiency of the theory of the mind in performing tasks can improve within age, nevertheless, it is not clear whether it also develops in childhood. When during a research the sane people are trying to draw conclusions about other peoples' mental states the neuroimaging data point to the activity in the frontal and temporal areas [50] which is compatible with the observations by Brownell et al. [51] who noticed that the acquired injury of the frontal lobes in the non-dominant hemisphere can cause impairment of the earlier correctly functioning theory of the mind processes.

In their research Russell et al. [52] proved that female patients with anorexia nervosa experience difficulties in the tasks requiring the theory of the mind [53], what is more, they are characterized by a limited ability to understand humour and joke conventions and possess impaired ability to recognize complex emotional mental states on the basis of eye expression. The above mentioned deficits are comparable to the disorders in the spectrum of autism.

Conclusions

Over the last decade, there is a growing interest amongst researchers in the area of social cognition and neurocognition in etiology and treatment of eating disorders.

However, results are inconclusive and still remain an open question whether the observed deficits in these areas are more the cause or result of the disease. Majority of research confirms presence of neuropsychological deficits in AN, both in terms of neurocognition and social cognition. Particular attention has been paid to the most frequently described in the literature – neurocognitive deficits such as rigid thinking, weak central coherence with attention to details, and deficits in social cognition, including emotional perception and expression, the theory of mind (ToM), and empathy.

Researchers incline the hypothesis stating that deficits are more trait-related and remain persistent and stable irrespective of the severity and chronic nature of the disease.

Undoubtedly, both etiology and dynamics of the described deficits remain unclear therefore treatment appears challenging indeed.

Currently, the highly specialized programs aimed at therapy of disordered neurocognition and social cognition appear very promising in view of increasing effectiveness of symptoms treatment (including pharmacotherapy and psychotherapy) via impact on emotional and social domains as well as disordered thinking style in AN.

References:

1. Tchanturia K, Happe F, Godley F, Treasure J, Bara-Carril F, Schmidt U. *Theory of mind in anorexia nervosa*. Eur. Eat. Disord. Rev. 2004; 12: 361–366.
2. Harrison A, Tchanturia K, Naumann U, Treasure J. *Social emotional functioning and cognitive styles in eating disorders*. Br. J. Clin. Psychol. 2012; 51: 261–279.
3. Shott ME, Vincent Filoteo J, Bhatangar KAC, Peak NJ, Hagman JO, Rockwell R i wsp. *Cognitive set-shifting in anorexia nervosa*. Eur. Eat. Disord. Rev. 2012; 20: 343–349.
4. Pieters G, Maas Y, Hulstijn W, Vandereycken W, Probst M, Peuskens J i wsp. *Differentiation of cognitive and motor aspects in a digit symbol substitution test in anorexia nervosa patients, before and after weight restoration*. Psychopathology 2004; 37(5): 227–232.
5. Sarrar L, Ehrlich S, Merle JV, Pfeiffer E, Lehmkuhl U, Schneider N. *Cognitive flexibility and Agouti-related protein in adolescent patients with anorexia nervosa*. Psychoneuroendocrinology 2011; 36(9): 1396–1406.
6. Kessler H, Schwarze M, Filipic S, Traue HC, von Wietersheim J. *Alexithymia and facial emotion recognition in patients with eating disorders*. Int. J. Eat. Disord. 2006; 39: 245–251.
7. Renwick B, Campbell IC, Schmidt U. *Attention bias modification: a new approach to the treatment of eating disorders*. Int. J. Eat. Disord. 2013; 46(5): 496–500.
8. Holliday J, Tchanturia K, Landau S, Collier D, Treasure J. *Is impaired set-shifting an endophenotype of anorexia nervosa?* Am. J. Psychiatry 2005; 162(12): 2269–2275.
9. Roberts ME, Tchanturia K, Treasure JL. *Is attention to detail a similarly strong candidate endophenotype for anorexia nervosa and bulimia nervosa?* World J. Biol. Psychiatry 2013; 14(6): 452–463.
10. Frank G, Shott M, Hagman JO, Yang T. *Localized brain volume and white matter integrity alterations in adolescent anorexia nervosa*. J. Am. Acad. Child Adolesc. Psychiatry 2013; 52(10): 1066–1075.
11. Kendall PC. *Terapia dzieci i młodzieży*. Kraków: Wydawnictwo UJ; 2010.

12. Nunn K, Frampton I, Fuglset TS, Törzsök-Sonnevend M, Lask B. *Anorexia nervosa and the insula*. Med. Hypotheses 2011; 76(3): 353–357.
13. Tchanturia K, Smith E, Weineck F, Fidanboylu E, Kern N, Treasure J i wsp. *Exploring autistic traits in anorexia: a clinical study*. Mol. Autism 2013; 4(1): 44.
14. Nunn K, Frampton I, Gordon I, Lask B. *The fault is not in her parents but in her insula – a neurobiological hypothesis of anorexia nervosa*. Eur. Eat. Disord. Rev. 2008; 16(5): 355–360.
15. Wagner A, Aizenstein H, Mazurkewicz L, Fudge J, Frank GK, Putnam K i wsp. *Altered insula response to a taste stimulus in individuals recovered from restricting type anorexia nervosa*. Neuropsychopharmacology 2008; 33(3): 513–523.
16. McAdams CJ, Krawczyk DC. *Who am I? How do I look? Neural differences in self-identity in anorexia nervosa*. Soc. Cogn. Affect. Neurosci. 2014; 9(1): 12–21.
17. Southgate L, Tchanturia K, Treasure J. *Building a model of the aetiology of eating disorders by translating experimental neuroscience into clinical practice*. J. Ment. Health 2005; 14(6): 553–566.
18. Steinglass J, Walsh BT, Stern Y. *Set shifting deficit in anorexia nervosa*. J. Int. Neuropsychol. Soc. 2005: 12(3): 431–435.
19. Steinglass J, Walsh BT. *Habit learning and anorexia nervosa: a cognitive neuroscience hypothesis*. Int. J. Eat. Disord. 2006: 39(4): 267–275.
20. Tchanturia K, Morris RG, Surguladze S, Treasure J. *An examination of perceptual and cognitive set shifting tasks in acute anorexia nervosa and following recovery*. Eat. Weight Disord. 2002: 7(4): 312–315.
21. Tchanturia K, Morris RG, Anderluh MB, Collier DA, Nikolaou V, Treasure J. *Set shifting in anorexia nervosa: an examination before and after weight gain, in full recovery and relationship to childhood and adult OCPD traits*. J. Psychiatr. Res. 2004: 38(5): 545–552.
22. Tchanturia K, Whitney J, Treasure J. *Can cognitive exercises help treat anorexia nervosa?* Eat. Weight Disord. 2006: 11(4): 112–116.
23. Fairburn CG, Cooper Z, Shafran R. *Cognitive behaviour therapy for eating disorders: a "transdiagnostic" theory and treatment*. Behav. Res. Ther. 2003; 41(5): 509–528.
24. Waller G. *Treatment of bulimia nervosa*. *Psychiatry* 2005; 4(4): 18–22.
25. Żechowski C, Namysłowska I, Brągoszewska J, Prusik M, Skiepko M, Witkowska M. *Różne podgrupy jadłowstrętu psychicznego – obraz kliniczny i przebieg*. Psychiatr. Psychol. Klin. 2009; 1: 77–78.
26. Nunn K, Frampton I, Lask B. *Anorexia nervosa-a noradrenergic dysregulation hypothesis*. Med. Hypotheses 2012; 78(5): 580–584.
27. Brewerton TD, Frampton J, Lask B. *The neurobiology of anorexia nervosa*. US Psychiatry, Touch Briefings 2009; 2(1): 57–60.
28. Tchanturia K, Davies H, Roberts M, Harrison A, Nakazato M, Schmidt U i wsp. *Poor cognitive flexibility in eating disorders: examining the evidence using the Wisconsin Card Sorting Task*. PLoS One 2012; 7(1): e28331.
29. Treasure J, Lopez C, Roberts M. *Endophenotypes in eating disorders: Moving toward etiologically based diagnosis and treatment focused on pathophysiology*. Pediatr. Health 2007; 1: 171–181.
30. Lopez C, Stahl D, Tchanturia K. *Estimated IQ in anorexia: A systematic review of the literature*. Ann. Gen. Psychiatry 2010; 23: 9–40.
31. Tchanturia K, Lock J. *Cognitive remediation therapy for eating disorders: development, refinement and future directions*. Curr. Top. Behav. Neurosci. 2011; 6: 269–287.

32. Roberts ME, Tchanturia K, Stahl D, Southgate L, Treasure J. *A systematic review and meta-analysis of set-shifting ability in eating disorders*. Psychol. Med. 2007; 37(8): 1075–1084.
33. Lopez C, Tchanturia K, Stahl D, Treasure J. *Central coherence in eating disorders: A systematic review*. Psychol. Med. 2008; 38(10): 1393–1404.
34. Tenconi E, Santonastaso P, Degortes D, Bosello R, Titton F, Mapelli D i wsp. *Set-shifting abilities, central coherence, and handedness in anorexia nervosa patients, their unaffected siblings and healthy controls: Exploring putative endophenotypes*. World J. Biol. Psychiatry 2010; 11: 813–823.
35. Harrison A, Tchanturia K, Treasure J. *Measuring state trait properties of detail processing and global integration ability in eating disorders*. World J. Biol. Psychiatry 2011; 12(6): 462–472.
36. Steinhausen HC. *The outcome of anorexia nervosa in the 20th century*. Am. J. Psychiatry 2002; 159(8): 1284–1293.
37. Tchanturia K, Hambrook D, Curtis H, Jones T, Lounes N, Fenn K i wsp. *Work and social adjustment in patients with anorexia nervosa*. Compr. Psychiatry 2013; 54(1): 41–45.
38. Lawson R, Emanuelli F, Sines J, Waller G. *Emotional awareness and core beliefs among women with eating disorders*. Eur. Eat. Disord. Rev. 2008; 16: 155–159.
39. Wildes J, Ringham R, Marcus M. *Emotion avoidance in patients with anorexia nervosa: Initial test of a functional model*. Int. J. Eat. Disord. 2010; 43: 398–404.
40. Meyer C, Leung N, Barry L, Feo D. *Emotion and eating psychopathology: Links with attitudes toward emotional expression among young women*. Int. J. Eat. Disord. 2010; 43: 187-189.
41. Davies H, Schmidt U, Stahl D, Tchanturia K. *Evoke facial emotional expression and emotional experience in people with anorexia nervosa*. Int. J. Eat. Disord. 2011; 44: 531–539.
42. Kucharska-Pietura K, Nikolaou V, Masiak M, Treasure J. *The recognition of emotion in the faces and voice of anorexia nervosa*. Int. J. Eat. Disord. 2004; 35: 42–47.
43. Zonnevijlle-Bender MJ, van Goozen SH, Cohen-Kettenis PT, van Elburg A, van Engeland H. *Do adolescent anorexia nervosa patients have deficits in emotional functioning?* Eur. Child Adolesc. Psychiatry 2002; 11: 38–42.
44. Jänsch C, Harmer C, Cooper MJ. *Emotional processing in women with anorexia nervosa and in healthy volunteers*. Eat. Behav. 2009; 10(3): 184–191.
45. Dmitrzak-Węglarz M, Jaracz J, Słopień A, Maciukiewicz M, Rajewski A. *Zaburzenia rozpoznawania emocji u chorych na jadłowstręt psychiczny w okresie adolescencji*. Neuropsychiatr. Neuropsychol. 2010; 5(2): 71–78.
46. Lena SM, Fiocco AJ, Leyenaar JK. *The role of cognitive deficits in the development of eating disorders*. Neuropsychol. Rev. 2004; 14: 99–113.
47. Rosenberg EL, Ekman P. *Coherence between expressive and experiential systems in emotion*. Cogn. Emot. 1994; 8: 201–229.
48. Oldershaw A, DeJong H, Hambrook D, Broadbent H, Tchanturia K, Treasure J i wsp. *Emotional processing following recovery from anorexia nervosa*. Eur. Eat. Disord. Rev. 2012; 20(6): 502–509.
49. Geller J, Cockell SJ, Goldner EM. *Inhibited expression of negative emotions and interpersonal orientation in anorexia nervosa*. Int. J. Eat. Disord. 2000; 28: 8–19.
50. Castelli F, Happe F, Frith U, Frith C. *Movement and mind: A functional imaging study of perception and interpretation of complex intentional movement patterns*. Neuroimage 2000; 12(3): 314–325.
51. Brownell H, Griffin R, Winner E, Friedman O, Happe F. *Cerebral lateralization and theory of mind*. W: Baron-Cohen S, Tager-Flusberg H, Cohen D. red. *Understanding other minds*. Second edition. Oxford: Oxford University Press; 2000.

52. Russell TA, Schmidt U, Doherty L, Young V, Tchanturia K. *Aspects of social cognition in anorexia nervosa: Affective and cognitive theory of mind*. Psychiatry Res. 2009; 168: 181–185.
53. Happe F, Brownell H, Winner E. *Acquired 'theory of mind' impairments following stroke*. Cognition 1999; 70: 211–240.