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Clinical picture, pathogenesis and psychometric assessment of negative symptoms of schizophrenia

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Summary

Negative symptoms of schizophrenia constitute a serious diagnostic and therapeutic problem. They substantially account for the impairment of health, social functioning and quality of life whereas treatment is difficult. In this paper the development of the concept of schizophrenia and negative symptoms is presented. The models of positive and negative symptoms, introduced in the 1980's by Timothy Crow and Nancy Andreasen, and William Carpenter's concept of so-called deficit syndrome with the criteria of the division of negative symptoms into the primary and secondary, are discussed. Current views on the pathogenesis of negative symptoms are shown with reference to neuroimaging studies, neurotransmitter alterations, neuropsychological deficits, genetic, immunological and epidemiological studies. A subsection is devoted to the diagnostics tools for negative symptoms. Chronologically, they are divided into scales of the 1st and 2nd generation. The first generation includes: the Brief Psychiatric Rating Scale (BPRS), the Scale for the Assessment of Negative Symptoms (SANS), the Positive and Negative Syndrome Scale (PANSS), the Schedule for the Deficit Syndrome (SDS), and the Proxy for Deficit Syndrome. The second generation scales, developed as a result of the recommendation by American experts in 2006, include: the Brief Negative Syndrome Scale (BNSS) and the Clinical Assessment Interview for Negative Symptoms (CAINS), also the self-assessment scales: the Motivation and Pleasure Scale – Self Report (MAP-SR) and the Self-assessment of Negative Symptoms (SNS). The BNSS and the SNS scales, whose Polish versions were elaborated in the Department of Adult Psychiatry of Poznan University of Medical Sciences, are discussed in-depth.

Key words: schizophrenia, negative symptoms, pathogenesis

Introduction

Most medical historians consider the case report referred to as *démence précoce* recorded in 1852 by Benedict Morelto to be the first formal description of schizophrenic psychosis [1]. In the subsequent period there appeared descriptions essentially corresponding to what is now known as particular subtypes of schizophrenia – the primary

madness by Griesinger (1867), catatonia by Kahlbaum (1874) and hebephrenia by Hecker (1871) [2]. In the second half of the 19th century, Emil Kraepelin subdivided mental disorders into chronic psychoses leading to an early dementia, and affective psychoses with periodical course. He defined the first group as *dementia praecox*, in other words, premature dementia. As his views evolved, the term came to encompass previously described hebephrenia, catatonia and paranoid dementia. This concept was developed in the 6th edition of his handbook from 1899 and is regarded as the birth of schizophrenia concept [3]. Given that the term *dementia praecox* assumes the onset of the pathological condition occurring in youth, irrevocably leading to dementia [4], it acquired the name of the disease from the deterministic viewpoint, describing cause-and-effect sequence which ends unfavorably. The term schizophrenia, derived from the Greek words *schizein* – "split" and *phren* – "mind, volition", was introduced into psychiatry by Eugen Bleuler. The structure of the name was not so much associated with the expression of "split" as "inconsistency" of mental processes, making distinction of symptoms between more important (axial) and less relevant ones (additional). In the Anglo-Saxon terminology, axial symptoms still function as the famous Bleulerian "four A's" – disorders of association of thought, affect, ambivalence and autism. Bleuler also pointed out that there is a clinical diversification of schizophrenia, thus he applied the term "the group of schizophrenias" [2, 4–6].

Both Kraepelin and Bleuler proposed "deficit" symptoms in terms of the deficit of intellectual as well as emotional, interpersonal or social functions, as basic symptoms of schizophrenia. However, in clinical practice, psychotic symptoms are apparently the most spectacular and diagnostic ones. It was Kurt Schneider who as first proposed clarification of diagnosis through the introduction of the so-called first-rank symptoms. Unless there is organic damage of nervous system, the presence of any of these symptoms indicates that schizophrenia is the case, although it is not pathognomonic of it. Second-rank symptoms are diagnostically less certain. According to Schneider, first-rank ("Schneiderian") symptoms involve three types of auditory hallucinations (audible thoughts, voices arguing and commenting), three kinds of sensations in the form of the psychic automatism (thought insertion, withdrawal, broadcasting) and also experiences of somatic influence, delusional perception as well as other experiences which have impact on feelings, motivation and volition [5, 7, 8].

In the 1980s appeared concepts concerning two groups of simultaneously occurring symptoms in patients with schizophrenia, that is positive symptoms (productive, psychotic) and negative (deficit) ones. At the same time, attention was paid to the separate pathogenesis and possibilities of treatment of these two groups of symptoms. British psychiatrist Timothy Crow [9] proposed the division of schizophrenia into two types: type 1 schizophrenia (with the increased levels of dopamine receptors) and type 2 schizophrenia (loss of cells together with structural abnormalities in the brain). Subsequent observations confirmed more frequent incidence of neurocognitive impairment and tardive dyskinesia in patients suffering from type 2 schizophrenia according to Crow, in comparison with type 1 schizophrenia. Likewise, Nancy Andreasen, an American psychiatrist, put forward a suggestion for the division of schizophrenia on the basis of nature of its dominant symptoms – with the dominant positive symptoms, the dominant

negative symptoms and the mixed subgroup (people who do not meet the criteria of any of the subgroups or fulfill the criteria for both at the same time). In the assessment of the authors of the concept, patients with positive symptoms have not been reported to show deficits in adaptation or in general functioning in the premorbid period, or to show cognitive impairment or brain atrophy. On the other hand, deficit in adaptation, general functioning, cognitive impairment and brain atrophy are usually present in patients with the dominant negative symptoms [10]. Another British psychiatrist, Peter Liddle, proposed distinguishing of three syndromes of chronic schizophrenia based on the neuropsychological test results – namely the psychomotor poverty syndrome (deficit syndrome), disorganization syndrome as well as reality distortion (psychotic) syndrome. Neuroimaging with the use of positron emission computer tomography made it possible for the characteristic changes in the regional cerebral blood flow to be assigned to particular syndromes [11, 12].

Negative symptoms: the concept and clinical picture

Negative symptoms (also referred to as deficit ones) include the group of symptoms related to the decrease and loss of various mental functions [5]. Essentially, they include poverty of content and form of thinking, the limitation of different forms of activity (from physical through impulsive to intentional activities) and the variously described impairment in emotional reactivity (flat, blunted, rigid, and pale emotion) [5]. Timothy Crow in his type 2 schizophrenia concept itemized the presence of negative symptoms such as flattening of affect, poverty of speech and the loss of drive. This process, according to Crow, is characterized by chronicity, the condition of "defect", the irreversibility of the process, poor therapeutic response to antipsychotic drugs, a possibility of intellectual deficit and the loss of nerve cells or structural brain abnormalities [9]. Andreasen and Olsen [10] identified and depicted the following groups of negative symptoms: affective flattening (the unchanging facial expression, paucity of expressive gestures, poor eye contact, affective nonresponsivity, lack of vocal inflections, decreased spontaneous movements), alogia (poverty of speech, poverty of content of speech, blocking of speech, increased latency of response), avolition and apathy (neglect of grooming and hygiene, impersistence at work or school, physical anergia), anhedonia and asociality (a short list of pastimes and recreational interests and activities, inability to feel intimacy and closeness, disordered relationships with friends and peers), attention disturbances (social inattentiveness, inattentiveness during mental status testing) [10].

The above models constituted the foundation for the ongoing discussion over the structure of observable symptoms of schizophrenia. The following structures have been proposed: the three-factor structure that consists of positive, negative and disorganization symptoms (thought disturbance, bizarre behavior), the four-factor structure that goes with positive symptoms, anergia (including negative symptoms), affective disturbances (depression and anxiety), and disorganization (conceptual disorganization), as well as the five factor structure entailing the following: positive and negative symptoms, activity and mood disorder, and autistic behavior [13].

In 1988, William Carpenter pointed out to the fact that the negative symptoms of schizophrenia may be primary, idiopathic, but also secondary, resulting not so much from the illness itself but from the additional factors such as treatment, mood disorders and environmental factors. In order to better distinguish these two groups of symptoms, he proposed the concept of deficit (DS) and non-deficit (NDS) schizophrenia. In accordance with the assumptions of the theory, deficit syndromes are chronic, they are present during periods of exacerbations of positive symptoms and between these exacerbations. In the non-deficit syndrome, negative symptoms are less stable and chronic, and their intensity is associated with the presence of external factors. The diagnostic criteria for DS are as follows: 1) The patient meets DSM-III (DSM-IV) criteria for schizophrenia; 2) At least two of the following six negative symptoms are present: restricted affect, diminished emotional range, poverty of speech, curbing of interest, diminished sense of purpose, diminished social drive; 3) The symptoms are not accounted by depression, anxiety, drug effect or environmental factors; 4) Two or more symptoms have been present for the preceding 12 months and these symptoms were also present during remission of psychotic symptoms. For patients who met only criterion 1, Carpenter proposes diagnosis of schizophrenia without negative symptoms. Patients who met criteria 1, 2 and possibly 4, can be diagnosed with schizophrenia with secondary negative symptoms. Patients meeting criteria 1–4 should be diagnosed with schizophrenia with deficit syndrome. Patients meeting criteria 1–3 can be diagnosed with schizophrenia with primary negative symptoms, these patients with passage of time may meet criteria for deficit schizophrenia [14, 15].

Subsequent research also demonstrated that not only does the deficit and non-deficit schizophrenia differ from one another in terms of symptoms, but they also do when it comes to the course of illness, pathophysiology, etiology as well as the improvement after the antipsychotic treatment. Deficit schizophrenia, in clinical picture, is characterized by greater social withdrawal, more anergia, less severe depressive symptoms, less hostility and dysphoria, less severe delusions and suicidal ideation, poorer insight, and less frequent psychoactive substance abuse. Deficit schizophrenia patients exhibit greater impairments on neuropsychological tests, they perform more poorly, among other things, on the Wisconsin Card Sorting Test, they show delayed visual reaction time, greater impairment in motor coordination and sensory integration. Social adjustment of deficit schizophrenia patients is poor at all developmental stages, while in patients with non-deficit schizophrenia the impairment occurs only in late adolescence and early adulthood. Deficit syndrome is more common in men, individuals who are single and those with long duration of symptoms prior to first hospitalization [16]. Long-term prognosis is worse in patients with deficit schizophrenia and, in addition, the poorer efficacy of antipsychotic drugs is shown in these patients [15, 17, 18].

Negative symptoms: pathogenesis

Studies on the neurobiological pathogenesis of negative symptoms have been conducted for 30 years. In the classical neuroimaging studies an association between enlarged ventricles and negative symptoms has been reported. However, not all studies

confirmed this relationship, some even suggest an inverse correlation [19, 20]. Currently, it is indicated that ventricular enlargement is related to cognitive impairment rather than negative symptoms. Patients with the predominance of negative symptoms have been observed to show the reduction of the prefrontal cortical volumes, temporal cortex, caudate nucleus, limbic structure, right parietal cortex, and corpus callosum. Some studies demonstrate inverse correlation between white matter volume and negative symptoms [21].

Using the functional neuroimaging studies such as positron emission tomography (PET) as well as single photon emission computer tomography (SPECT), the relationship between the intensity of negative symptoms and the decreased frontal and prefrontal metabolism was observed, both at rest and during activation. Similar results were obtained in patients with intensive negative symptoms during the first episode or in drug-naive patients, which excludes the modification of results due the chronicity of the illness or neuroleptic treatment [22, 23]. In studies that employ functional magnetic resonance imaging (fRMI), an activation deficit in the ventral striatum was recorded during reward anticipation in unmedicated subjects or those treated with first-generation antipsychotics, while in those treated with second-generation antipsychotics similar results were not obtained [24]. Moreover, the relationship between disorders of emotional processes such as poor eye contact, diminished facial emotional expression, diminished spontaneous movement along with verbal expressiveness and dysfunctions of limbic regions, among others, the left anterior cingulate cortex, right orbitofrontal cortex, left medial prefrontal cortex and left fusiform gyrus was found. The correlation between flat affect and increased amygdala activity was also observed [25, 26].

Comparative studies of persons meeting criteria for the deficit and non-deficit schizophrenia have provided interesting results. Namely, patients with non-deficit schizophrenia, the course of which by definition is milder, were noticed to have more marked ventricular enlargement and the decrease in the prefrontal volumes. These findings may suggest that structural changes in the prefrontal region are not responsible for the negative symptoms [27]. What seems characteristic of people with deficit schizophrenia is the temporal gray matter reductions and atrophy of fronto-parietal and fronto-temporal pathways. These observations suggest that the most characteristic for negative symptoms are white matter disruptions, whereas fronto-temporal-parietal circuits are responsible for emotion expression and social functioning. This is confirmed by functional neuroimaging findings, pointing out to significant metabolic disorders within the prefrontal-thalamic-parietal-network in deficit schizophrenia patients as compared to those with non-deficit schizophrenia [21, 28].

As to relationship between the incidence and intensity of negative symptoms and disturbance in neurotransmission, many observations concern dopaminergic system. Since typical antipsychotic drugs usually cause exacerbation of negative symptoms, it has been proposed that these symptoms are associated with a deficit in dopaminergic transmission in prefrontal cortex [29]. It is also postulated that there is a relationship between the intensity of negative symptoms and the decrease in prefrontal dopamine D1, D3 and D4 receptors [21]. It was also observed that while substances modulating the activity of dopaminergic system (e.g., amphetamine or apomorphine), which

increase metabolism in the cerebellum and striatum – reduced the severity of negative symptoms, while alpha metyl-paratyrosine (AMPT), decreasing the dopamine synthesis, intensified them [21, 30]. In patients with deficit as opposed to non-deficit schizophrenia, lower concentration of homovanillic acid (HVA), the major catecholamine metabolite, was reported [31].

Several studies pointed out that since nicotine stimulates the production and release of dopamine, it may reduce negative symptoms. This was confirmed by observations in which schizophrenia patients with lower β2-nicotinic acetylcholine receptor activity were characterized with greater intensity of negative symptoms, whereas heavy smokers – with lower intensity [21, 32]. Regarding the relationship between the intensity of negative symptoms and serotonin system activity, one study showed relationship between low 5-hydroxyindoleacetic acid concentration (5-HIAA), a serotonin metabolite, and the intensity of these symptoms [33]. Also, a relationship was found between the activity of glutamatergic system and negative symptoms, especially in respect of the N-methyl-D-aspartate (NMDA) receptor dysfunction [34].

The role of genetic factors in the pathogenesis of negative symptoms raises interest as well. Researches from the Szczecin center have found the relationship between negative symptoms of deficit schizophrenia and catechol-O-methyltransferase (COMT) gene polymorphism [35]. Among other genes whose relationship with negative symptoms has been demonstrated, there are: D2 dopamine receptor gene (DRD2), nicotinic acetylcholine receptor gene (CHRNA3) and dysbindin gene (DTNBP1) [21].

Positive relationship has been found between male gender and predisposition towards developing deficit syndrome [36]. Additionally, it was shown that serum folate concentration was lower in patients with deficit than non-deficit schizophrenia [37]. It has been indicated that there is a possibility of a relationship between developing deficit schizophrenia and Borna virus as well as cytomegalovirus infection [38, 39].

In neuropsychological studies it was found that deficit schizophrenia patients in comparison to non-deficit schizophrenia patients exhibited poorer performance in tests assessing frontal and parietal lobe functions, while in the temporal lobe functioning the differences have not been reported [40].

The evaluation of deficit schizophrenia prevalence depending on the season of birth indicates that, in contrast to classical observation of a predominance of winter birth in schizophrenia – individuals with deficit form of schizophrenia more often show summer birth [41].

Methods of clinical evaluation of negative symptoms

In the first tools for the evaluation of presence and intensity of schizophrenia, referred to as first-generation scales, the assessment of negative symptoms was only one aspect of a comprehensive illness description. The Brief Psychiatric Rating Scale (BPRS) as a general psychometric scale, has been used for the longest period of time. In the classical, 18-item version of this scale, negative symptoms are rated according to the three categories: emotional withdrawal (lack of spontaneous interaction, withdrawal from relations with others), motor retardation (slowed, weakened move-

ments or speech, reduced body tone) as well as blunted affect (reduced emotional tone, reduction of intensity of feelings, emotional flattening). This scale does not take account of the environmental factors, taking drugs and any other components of mental health (e.g., depression) on the form and intensity of the above-mentioned deficit symptoms [42, 43]. The Positive and Negative Syndrome Scale (PANSS) puts the psychopathological symptoms into three categories: positive, negative and general symptoms. The description of negative symptoms comprises 7 items (blunted affect, emotional withdrawal, poor rapport, passive/apathetic social withdrawal, difficulty in abstract thinking, lack of spontaneity and flow of conversation, stereotyped thinking). The assessment of these symptoms is exposed to similar reservations as in the case of the BPRS, and including abstract and stereotyped thinking disorders as part of the negative symptoms category is currently raising controversies [43, 44].

The first diagnostic scale directly referring to deficit symptoms was the Scale for Assessment of Negative Symptoms (SANS) designed in the 1980s by Nancy Andreasen. This scale covers five items: affective flattening, alogia (poverty of thinking), avolition and apathy, attention deficit, anhedonia and non-socialization. The scale does not distinguish primary from secondary negative symptoms, however, it evaluates quality of life and level of functioning, among others, grooming and hygiene, impersistence at work and school, recreational interests and activities, sexual interest and activity, and the ability to feel intimacy and closeness [43, 45, 46].

In order to separate primary and secondary negative symptoms as well as take into account the division into the deficit and non-deficit schizophrenia, American researchers (Kirkpatrick et al.) created the Schedule for the Deficit Syndrome (SDS). It is presently regarded as the "gold standard" in the assessment of deficit symptoms and is based on the interview conducted with a patient, supplemented by the information coming from medical, social and family sources. The interview should be conducted at time of clinical stability of the mental state of the interviewed. The scale includes six symptoms: restricted affect, poverty of speech, diminished sense of purpose, diminished social drive, curbing of interest, and diminished emotional range. The symptoms should be of primary nature, an examiner has to identify and exclude secondary deficit symptoms related to anxiety, depression, drug treatment, psychotic symptoms or intellectual disability. The assessed symptoms must persist throughout a year before examination [18, 47]. In order to use the data obtained during the evaluation of schizophrenia patients by means of scales such as the BPRS, the SANS or the PANSS, Kirkpatrick et al. also proposed a tool called the Proxy for Deficit Syndrome (PDS), which indirectly allows to delineate deficit symptoms on the basis of symptoms assessed using the aforementioned scales [48].

With a view to standardize views on pathogenesis, structure and the clinical picture of negative symptoms, the participants of the Consensus Development Conference of Negative Symptoms (Rockville, 2005) issued a statement in which they summed up the results of the international discussion. They considered blunted affect, alogia, asociality, anhedonia and avolition to be the negative symptoms. It was also established that creating a new diagnostic scale concerning presently accepted views on deficit symptoms was necessary. Additionally, the expert panel determined essential

assumptions during the tool creation. According to the guidelines, the scale should be concise and feasible in practical use, so that it could be used in a variety of cultures, both in clinical trials, as well as psychological and epidemiological studies. It should evaluate the symptoms currently regarded as negative, excluding those related to disorganization (such as poverty of speech and attention disturbances), maintaining a distinction between anticipatory and consummatory aspects of anhedonia as well as to differentiate between internal experience and behavior [49].

On the basis of the above guidelines, new scales for negative symptoms were created referred to as second generation scales. These include clinical scales: the Brief Negative Syndrome Scale (BNSS) and the Clinical Assessment Interview for Negative Symptoms (CAINS), along with self-evaluation scales: the Motivation and Pleasure Scale – Self Report (MAP-SR) and the Self-assessment of Negative Symptoms (SNS).

The Clinical Assessment Interview for Negative Symptoms (CAINS) is a 13-item scale assessing motivation, pleasure and emotion expression. The section on motivation and pleasure is divided into three sections. The first section assesses the social aspect – motivation for family, spouse and partner relationships, motivation for friendships and romantic relationships, frequency of pleasurable social activities (past week), frequency of expected pleasure from social pleasure (next week). The second section assesses work and school motivation (last week) and frequency of expected pleasure (next week). The third section assesses recreational motivation and pleasure – motivation for recreational activities, frequency of pleasurable recreational activities (last week) and frequency of expected pleasure from recreational activities (next week). The section on the expression of emotions is the fourth section and includes an assessment of facial expression, vocal expression, expressive gestures, and quantity of speech. All items were rated on a scale of 0–4, with higher scores reflecting greater impairment [50].

The Motivation and Pleasure Scale – Self Report (MAP-SR) is a self-assessment scale based on the CAINS. A self-report measure would provide a time efficient method for initial identification of people with elevated negative symptoms. It consists of 18 questions concerning motivation and pleasure in terms of social relation, work, school and recreational activities, self-reported by the patient in range from 0 to 4, during the last week, or, for expected activity – in the coming week. Six items assess consummatory and anticipatory pleasure related to social or work domains, six items assess feelings and motivations to be around family, romantic partners and friends, the remaining six items assess motivation and effort to engage in activities [51].

In recent years, the Brief Negative Symptoms Scale (BNSS), which was developed in 2011 by Kirkpatrick et al. [52], has become the most popular. The scale defines negative symptoms as an absence or decrease in behaviors and subjective experiences that are normally present in a person from the same culture and age group. It evaluates 5 symptoms for which there is currently a consensus on their nature as negative symptoms – anhedonia, asociality, avolition, blunted affect, and alogia. The sixth subscale, which describes psychic distress (worrying), was added. The examination is of an interview nature based on a manual including, among other things, prompts and suggested questions. All the items are rated on a 7-point scale (0–6), with anchor

points ranging from symptom being absent (0) to severe (6). The time frame for the ratings is one week. The basis for the interview is provided by the patient; the observation of the examined person also constitutes an important element, or if needed, the data obtained from external sources. Eventually, the scale includes 13 items organized into 6 subscales: anhedonia (intensity and frequency of pleasure, intensity of expected pleasure), distress (subject's experience of unpleasant or distressing emotion of any kind, such as sadness, depression, anxiety, grief, anger), asociality reported as reduced social activity accompanied by decreased interest in forming close relationships with others (behavior, internal experience), avolition reported as a reduction in the initiation of and persistence in activity (behavior, internal experience), blunted affect which refers to a decrease in the outward expression of emotion (facial expression, vocal expression, gestures), alogia reported as poverty of speech (quantity of speech, spontaneous elaboration of speech) [52].

French researchers (Dollfus et al.) developed the Self-assessment of Negative Symptoms (SNS) with an intent of paying special attention to the benefits of self-assessment of the patient. The Self-assessment enables a patient to gain better knowledge and understanding of their experiences and, consequently, makes it possible for them to participate more fully in treatment and rehabilitation. What seems extremely significant for detecting negative symptoms is that it allows to focus on the symptoms which may have gone unnoticed by the observer, particularly in the early stages of the illness. The SNS evaluates 5 negative symptoms: social withdrawal, diminished emotional range, avolition, anhedonia, and alogia. Social withdrawal assesses social relationships as well as the patient's desire to establish new relationships; diminished emotional range evaluates happiness or sadness as perceived in situations in which happiness or sadness is usually felt; avolition evaluates motivation, energy and the ability to achieve the goals; anhedonia evaluates the pleasure perceived and anticipatory pleasure; alogia (poverty of speech) depends on the subjective assessment of the examined individual. The scale includes 20 items and the evaluation is based on the previous week. To simplify completion, the number of responses was limited to 3: "strongly agree" scoring 2, "somewhat agree" scoring 1 and "strongly disagree" scoring 0. The total score is the sum of the 20 items, ranging from 0 (no negative symptoms) to 40 (severe negative symptoms). The questionnaire should be completed in about 5 minutes [53].

The listing of the first and second generation scales (both clinical and self-assessment types) for the assessment of negative symptoms of schizophrenia is presented in table 1.

Table 1. **First and second generation scales for the assessment of negative symptoms of schizophrenia**

Diagnostics tool	Authors
First generation scales	
Brief Psychiatric Rating Scale (BPRS) 3 items for negative symptoms	Overall, Gorham; 1962 [42]

table continued on the next page

Positive and Negative Syndrome Scale (PANSS) 8 items for negative symptoms	Kay, Fiszbein, Opler; 1987 [44]
Scale for Assessment of Negative Symptoms (SANS)	Andreasen; 1989 [45]
Schedule for the Deficit Syndrome (SDS)	Kirkpatrick et al.; 1989 [47]
Second generation scales	
Clinical scales	
Clinical Assessment Interview for Negative Symptoms (CAINS)	Kring et al.; 2013 [50]
Brief Negative Symptom Scale (BNSS)	Kirkpatrick et al.; 2011 [52]
Self-evaluation scales	
Motivation and Pleasure Scale – Self Report (MAP-SR)	Llerena et al.; 2013 [51]
Self-assessment of Negative Symptoms (SNS)	Dollfus et al.; 2016 [53]

The Polish versions of the Brief Negative Symptom Scale (BNSS) as well as the Self – assessment of Negative Symptoms (SNS) were developed in the Department of Adult Psychiatry, Poznan University of Medical Sciences. The back translation of the scales and descriptions from Polish into English (BNSS) and from Polish into French (SNS) have been accepted by their authors: Brian Kirkpatrick and Sonia Dollfus, respectively.

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