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Cognitive control and impulsivity in deficit schizophrenia: neuropsychological and clinical characteristics

Maksymilian Bielecki, Monika Mak, Jerzy Samochowiec, Ernest Tyburski

Abstract

Over the years, various concepts concerning the nature of schizophrenia have emerged. One of the proposals put forward in the literature is the division of schizophrenia into its deficit and non-deficit subtypes. Compared to its non-deficit counterpart, the deficit type involves more severe cognitive dysfunctions. Among these, some authors enlist poorer cognitive inhibition and greater impulsivity, although their behavioral correlates still remain unclear. Structural and functional studies of the brain suggest the presence of links between impulsivity and inhibition in schizophrenia, but few studies to date have analyzed cognitive performance within these two domains in patients with the deficit type. In addition, little is known about the effectiveness of neurorehabilitation in reducing cognitive deficits in deficit schizophrenia. Thus, numerous questions in this area seem to warrant further research.

In this paper we present clinical and neurobiological correlates of cognitive control and impulsivity against neuropsychological and psychopathological symptomatology of schizophrenia. In addition, we describe selected neuropsychological tools used in the diagnosis thereof. We also touch upon potential benefits of cognitive rehabilitation in patients with schizophrenia, especially in terms of self-control.

Last but not least, this paper presents an overview of previous research and an attempt to embed them in modern theoretical models of cognitive inhibition and impulsivity, for a better understanding of the nature of cognitive dysfunction in schizophrenia seems crucial for proper diagnosis and assessment of treatment effects, and in a broader context is important for understanding its effects on quality of life and functioning of patients and their families.

deficit schizophrenia; cognitive control; cognitive inhibition; motor inhibition; impulsivity

1. INTRODUCTION

Schizophrenia is a severe neurodevelopmental disorder characterized by a variety of symptoms, commonly resulting in disability. In addition to traditional psychopathological presentation (including e.g. negative, positive or disorganized symptoms), schizophrenia involves cognitive deficits (within e.g. attention, memory, language or executive functions) [1,2,3].

Over the years, several concepts regarding the essence of schizophrenia have been put forward, from models considering it a homogeneous construct towards more multifactorial ap-

[1]Maksymilian Bielecki, [1]Monika Mak, [2]Jerzy Samochowiec, [1]Ernest Tyburski: [1]Department of Health Psychology, Pomeranian Medical University in Szczecin, Szczecin, Poland; [2]Department of Psychiatry, Pomeranian Medical University in Szczecin, Szczecin, Poland.
Corresponding address: maksbiel@gmail.com

proaches. One of such proposals is the deficit syndrome which is characterized by the presence of primary negative symptoms from the very onset of the disease [4]. The deficit type is distinguished by different risk factors [5.6], family history [7], course [8,9], response to treatment [10], neuropsychological functioning [11], and underlying neurobiological differences [12]. Various researchers deem it proper to analyse cognitive dysfunction in schizophrenia separately for individual subgroups of patients, e.g., with dominant positive or negative symptoms [13]. The multiformity of its symptoms encourages identification of more homogeneous patient populations, as proposed by Carpenter [14], who introduced the term deficit schizophrenia. This variant is dominated by the deficit syndrome (i.e., primary, persistent negative symptoms), which occurs throughout its course. Negative symptoms include: reduced emotions, verbal fluency, ability to plan, initiate and continue activities, diminished interests and to feel pleasure [14], as well as social withdrawal, poverty of speech, apathy or blunted affect [18], with previous longitudinal analyzes indicating their temporal stability [16,17,8,9,15].

Most studies indicate greater cognitive impairment in patients with deficit schizophrenia as compared to their non-deficit counterparts. However, it is not entirely clear whether there are specific features differentiating the two populations in terms of their neurocognitive profiles [11]. Some reports demonstrate a slightly greater susceptibility to cognitive interference and reduced ability to create concepts, as well as disturbances in non-verbal cognitive flexibility in patients with deficit schizophrenia [18]. Research also provides evidence of deficits in cognitive inhibition in schizophrenia, but their behavioral correlates remain largely unknown [19]. What previous findings seem to suggest is that cognitive inhibition may be a process that is relatively independent of behavioral inhibition in various schizophrenia patients. Future research projects should therefore investigate a potential relationship between cognitive and behavioral inhibition. Impulsive behavior is reported in schizophrenia [20], but there are few studies comparing impulsivity between the deficit and non-deficit patients.

Due to certain gaps in existing knowledge, this paper aims to characterize clinical and neurobiological correlates of cognitive control and impulsivity in deficit schizophrenia, and describe neuropsychological and psychopathological symptoms thereof. The review also presents models of cognitive control and impulsivity, which enable a better understanding of the essence of cognitive dysfunction in deficit schizophrenia. In addition, we also touch upon potential benefits of cognitive rehabilitation in patients with schizophrenia, especially in terms of self-control.

2. THEORETICAL MODELS OF COGNITIVE CONTROL AND IMPULSIVITY

Responsible for the control of cognitive processes and behavior, inhibition is one of the key aspects of executive performance [21]. Based on the type of stimuli and modality, different aspects of inhibition are proposed, involving emotional, cognitive and motor control [22]. Cognitive inhibition consists in refraining from action [21]. One of the key areas of executive functioning that may be more disturbed in people with deficit compared to non-deficit schizophrenia is behavioral control, responsible for achieving long-term goals and resisting short-term impulses [23]. Of note, two basic aspects thereof have been distinguished. The first one is cognitive inhibition, which directs attention straight to the goal, bypassing irrelevant stimuli that are considered distractors [24]. The other one is motor inhibition, which consists in behaviorally resisting temptations and delaying gratification to achieve superior goals in the future [25].

As a crucial process, inhibition enables cessation of actions that are non-adaptive [26]. It is defined as a control mechanism that supports the process of selecting incoming information whilst operating on two levels. On the one hand, it hinders the use of information that is irrelevant in a given situation, and on the other, it inhibits a currently undesirable activity [27, 28, 29]. The effectiveness of executive processes depends, among other things, on the capacity to inhibit all external stimuli that are irrelevant or distracting, as well as internal stimuli, such as impulses, urges or factors related to emotional processing which due to their distracting nature may reduce the quality of task performance [22].

Motor inhibition is related to the control of behavior and motor impulses. Its function is to direct behaviors in accordance with the original context, while inhibiting those that are currently undesirable [22]. Barkley [30] created a model of cognitive inhibition that corresponds to three types of situations: a) inhibition of the prepotent response to a given stimulus (enabling a conscious decision to react), b) stopping an ongoing reaction (which provides an option to postpone the evaluation of a given response) and c) interference control. Effective inhibition processes underpin self-control, delayed gratification, goal-directed action, intentional action, speech management, as well as the choice of executive behaviors [31]. Inhibition is significant for behavioral control and executive functions, which refer to the processes responsible for purposeful action. Their number, as well as related processes, depend on the adopted concept or model [32].

Opposite of inhibitory control, impulsiveness can be construed as a tendency to quick, unplanned reactions in response to emerging stimuli. Impulsive behaviors are characterized by reduced or no reflection, forethought or consideration of consequences, concerning not only oneself but also their environment [33]. There are two types of impulsivities – motor and cognitive [34]. Motor impulsiveness is associated with the inability to withhold reaction, which manifests itself via responses that might be improper, while cognitive impulsiveness is associated with a changed assessment of the effects of actions, alongside a quick, lacking detail, analysis of the collected data [35]. According to Arce [36], motor impulsivity is failure to stop a once initiated reaction in response to an incoming stimulus. Thus, impulsivity is a multidimensional theoretical construct that can be defined as a tendency to quick, unplanned reactions to internal or external stimuli, regardless of the negative consequences of these reactions to self or others [37]. Of note, excessive impulsivity might be a consequence of reduced inhibitory control [38,39]. Eysenck [40] defines impulsiveness as a trait that determines a constant tendency to take risks, unplanned actions, or make quick decisions. Impulsivity has a neurobiological basis, involving the frontal lobes, the limbic system, and the striatum. Numerous studies indicate links between high impulsivity and symptoms of damage to the frontal areas, with cognitive impulsivity largely associated with the ventromedial prefrontal cortex and behavioral impulsivity with the dorsolateral prefrontal cortex [41]. Impulsive behavior is found in both healthy and mentally or neurologically ill individuals. Notwithstanding, it is reported to assume various forms, but for the time being its precise underpinnings remain largely unknown. One likely contributor is impaired control [19]. Some authors propose that impulsiveness is associated with a certain imbalance between impulse and its control [42].

3. ASSESSMENT OF COGNITIVE CONTROL AND IMPULSIVITY

Crucial for assessing the nature and severity of impairments within inhibition and impulsiveness is the proper selection of diagnostic tools that will adequately indicate the deficit areas, and consequently allow the best choice of treatment or rehabilitation methods. Selected tools are listed and discussed in the tables below. Table 1 presents those that are used in the neuropsychological assessment of cognitive inhibition.

Table 1. Characteristics of common assessment tools for cognitive inhibition in schizophrenia

Neuropsychological tests to measure the level of cognitive inhibition	
Stroop Color Word Test	SCWT [43] in the paper version consists of three A4 sheets. The first one contains names of colors (red, green, yellow, and blue) printed in black ink. The task is to read the names of colors in the shortest possible time. The next sheet shows colored boxes, in the colors whose names are in the first part of the test. The task is to name the colors in the shortest possible time. The third sheet contains names of colors printed in an inconsistent color ink, e.g. the word „green" is printed in blue. The task is to name the font color in the shortest possible time, ignoring the content of the color word. The test measures cognitive inhibition of the prepotent response (i.e. reading). It measures interference control, i.e. inhibiting a well-learned, automatic task and replacing it with an intentional response [31]. Cognitive inhibition occurs at a deep level of cognitive processing, it is active and effortful [22].

Variations of the Stroop Color Word Test	
The Victoria Version	VST [44] whose great advantage is an abbreviated form compared to the others. Unlike other versions including several dozen items (60–112) for each part of the task, the Victoria Stroop Test (VST) has only 24 items in each of the three conditions. While administration time is shorter, the VST remains a good measure of difficulties in terms of response inhibition as it does not provide the opportunity to gain experience in performing the task. The test consists of three cards measuring 21.5 × 14 cm, each containing six rows of four positions (Helvetica, 28 points). In part D (Dots), the subject must name the colors of 24 dots printed in blue, green, red or yellow as quickly as possible. Each color is used six times and the four colors are arranged in a pseudo-random order. Unlike the original Stroop Test, Part W (Words) is similar to Part D, except that the dots are replaced with common words (when, hard and over). The task is to name the color in which the words are printed, ignoring their verbal content. Part C (Colors) is similar to parts D and W except that the stimuli are the color names „blue, green, red and yellow" printed so that the color of the ink never matches the color name (e.g. „red" is printed in blue ink). The latter task therefore requires suppressing the prepotent reading response in favor of a more complicated response that is color naming. The interference effect is determined by calculating the extra time required to name the colors relative to the time required to name the colors in the control condition.
The Comalli Version	The Comalli Version [45] consists of three white sheets measuring 24 × 24 cm. Each sheet contains 100 items (a 10×10 layout). The first card contains names of colors (red, blue and green) printed in black ink, randomly arranged in 10 rows of 10 pieces each. The second card contains red, blue and green patches randomly selected arranged in a 10 x 10 pattern. The third card contains color words printed in ink that is incongruous with the verbal content. The time to complete each of the three parts is an independent variable. The Comalli modification assumes that the card with the color words should be presented first. The rationale behind this modification is that a) people who are colorblind can be quickly identified and b) the procedure maximizes the interference effect.
The Gold Version	The Golden version [46] consists in three parts: 1. Reading words (W) 2. Naming colors (C) and 3. Naming the color of a word (CW). During the test, errors are indicated and encouraged to be corrected. The last item on each stimulus sheet is noted after 45 seconds and the score for each part consists of the number of items completed. The interference score is also calculated. Errors are not counted. The obtained scores are compared against the norms predicted based on age and years of education.

Motor inhibition measures are distinguished by a slightly different structure and procedure, therefore their description is included in Table 2.

Table 2. Characteristics of common assessment tools for motor inhibition in schizophrenia

Tool for assessment of motor inhibition	
The Go/ No-Go Task	GNG [47] measures the capacity to monitor and inhibit conflicting reactions. Different versions of the task are available, but it usually consists in responding to certain visually presented stimuli and not responding to others. Inhibition is calculated by the number of correct responses relative to the incorrect ones. The number of omission errors is also considered [32]. The task consists of three parts. First, the task is to press a button when a green square appears on the computer screen, and the correct response is rewarded with the feedback „correct!". In the second part, green and blue squares appear on screen, and the task is to limit responses only to green squares. When a blue square appears, the desired reaction is to refrain from pressing the button. Correct responses are followed with the message „correct!", and the incorrect ones result in the message „wrong!". In the third part, green and blue squares appear, and the task is again to press a button only in response to a green square. This time around responses are not followed with any feedback in the form of „correct!" or „wrong!" messages. The sum of correct responses throughout the entire task makes up the total score.

Impulsivity is commonly measured with the use of self-report scales, including a set of questions concerning its various behavioral manifestations and problems related to its control (Table 3).

Table 3. Characteristics of common questionnaires to measure o impulsivity in schizophrenia

Scales for measuring impulsivity	
The Eysenck Impulsiveness Inventory	The IVE [48] is used to test three personality traits: impulsiveness, venturesomeness and empathy. The tool consists of 54 yes/no questions. The results are presented on three scales. Impulsiveness includes 19 items, Venturesomeness – 16 items and Empathy – the remaining 19 items. The tool has satisfactory absolute stability of all scales, good internal consistency, as well as confirmed factor validity. The criterion validity was assessed by analyzing results of clinical groups.
The Barratt Impulsiveness Scale	The first version of the scale was created in 1959. The latest, eleventh version of the Barratt Impulsiveness Scale (BIS-11) [49] is a self-report questionnaire consisting of 30 items describing behavior, rated based on its frequency on a 4 point scale (rarely/never, sometimes, often, and almost always/always). Six first-order factors emerge in the structure of BIS-11, reflecting various aspects of impulsivity, including attention, motor, self-control, cognitive complexity, perseverance, and cognitive instability impulsiveness. In addition, three second-order factors were distinguished, including attentional impulsivity, motor impulsivity, and lack of planning.
The Impulsiveness Control Scale	The ICS [50] consists of 15 items to measure levels of self-control, planning and spontaneity.
Impulsivity Rating Scale	The IRS [51] contains descriptions of behavior in ordinary situations. It includes seven elements: irritability, patience/impatience, time needed for decision, capacity to stay at activities, aggressiveness, control of response, and capacity to delay.
The Empathy Questionnaire	The IVE-7 [52] was developed to describe unconscious risk taking and conscious sensation seeking.
The Sensation-Seeking Scale	The SSS [53] measures four aspects of impulsivity, i.e. disinhibition, boredom susceptibility, thrill and adventure seeking, and experience seeking.

4. NEUROCOGNITIVE DEFICITS IN SCHIZOPHRENIA

A meta-analysis by Bora [11], which compared the results of 28 studies, indicates that people with deficit and non-deficit schizophrenia have impaired cognitive functions, particularly within the domains of executive performance, memory, processing speed, attention, working memory and verbal fluency. Of note, non-deficit patients achieved significantly poorer results relative to healthy controls. However, the effect sizes of the observed deficits were relatively small compared to the results of deficit patients. In all cognitive domains, patients with deficit schizophrenia scored significantly poorer compared to their non-deficit counterparts, including results in traditional neuropsychological tasks e.g., the Wisconsin Card Sorting Test, Verbal Fluency, and the Stroop task. However, differences emerged in the degree of impairment of individual domains between the two patient groups.

Deficit schizophrenia is characterized by markedly reduced verbal fluency, including both its semantic and phonemic variants. Alogia is a typical symptom indicative of deficits in this area. People suffering from deficit schizophrenia also experience greater difficulties in social functioning [54, 55]. Greater differences occur between patients with deficit vs non-deficit schizophrenia in executive performance dependent on processing speed in that non-deficit patients achieve results similar to low-scoring healthy individuals, while their deficit counterparts tend to score relatively lower compared to healthy populations [11].

A systematic review by Tyburski et al. [18] showed that deficient patients exhibit poorer concept formation than non-deficit patients, and have a reduced capacity to use positive and negative feedback in the process of learning and responding optimally to new situations. It was also shown that patients with deficit schizophrenia score worse on non-verbal cognitive flexibility. Based on their meta-analysis, Bora et al. [11] suggest that patients with both deficit and non-deficit schizophrenia may exhibit similar-level performance in terms of executive func-

tions (particularly accuracy-based), as well as working, visual and verbal memory. Tyburski et al. [18] found that in both groups, patients performed poorer in verbal cognitive flexibility than in other executive domains. In addition, patients in both groups achieved the same level in terms of concept formation, problem solving and motor inhibition.

There is evidence of decreased cognitive inhibition in schizophrenia, but related behavioral consequences remain unclear [56]. One study indicates no differences in the performance of the Stroop Test between schizophrenia patients and healthy individuals [56]. In turn, Henik and Salo [57] suggest increased susceptibility to Stroop interference in patients with schizophrenia, which is intensified by the extended time intervals between trials. Importantly, there is no indication of the effect of medication on Stroop interference in this patient population. On the other hand, Tyburski et al. [18] observed similar motor response in patients with deficit and non-deficit schizophrenia based on the performance of the go/no-go test. The groups did not differ in inhibition of responses to irrelevant stimuli (no-go). However, the existence of greater deficits in cognitive and motor inhibition in patients with deficit schizophrenia requires further research.

Available studies [58] suggest that patients with schizophrenia differ significantly in terms of prepulse inhibition. Prepulse inhibition is an initial reaction to a startling stimulus and consists in the appearance of a weak initial impulse that reduces the magnitude of the startle reflex. Patients with schizophrenia showed a marked deficiency in prepulse inhibition. Eye blink latency was slower in patients with schizophrenia. Poor prepulse inhibition correlated with both positive and negative symptoms of schizophrenia [58].

Of note, few studies to date refer to the negative symptoms in deficit schizophrenia [59]. As noted above, even though there have been studies of cognitive inhibition and motor skills in schizophrenia, relatively few reports focus on the deficit type. Therefore, future research should aim to assess the role of negative symptoms in deficit schizophrenia.

5. IMPULSIVITY IN SCHIZOPHRENIA

Given the role of cognitive control and impulsiveness in the lives of people with schizophrenia, it is key to understand their abnormalities. Considering the importance of primary negative symptoms, it is also important to explain their specificity in deficit schizophrenia. Depending on how they are defined, negative symptoms are observed in 50-90% of patients with the first episode of schizophrenia [60] and in 20-40% of patients with the chronic course [61]. Negative symptoms lower the standard of living and disrupt social and professional functioning [62].

Impulsive, disorganized behavior is observed in schizophrenia [20]. Patients with schizophrenia obtain significantly higher impulsivity scores on the BIS scale relative to healthy individuals [63]. Some studies report a significant increase in impulsivity in various types of tasks, such as reaction to stop signal or delay discounting [63, 56]. Although research clearly indicates a correlation between outbursts of aggression and schizophrenia, it turns out not to necessarily be associated with impulsiveness, but likely related to an intended, planned action [64]. Iancu and colleagues [65] suggest links between a high level of impulsiveness and an increased risk of suicide attempts, aggression, and positive symptoms in schizophrenia. Researchers also conclude that the higher the level of impulsivity, the higher the likelihood of suicide and psychopathology (not including negative symptoms). This means that the more impulsive a person is, the more psychotic symptoms they are likely to experience [65]. Studies on adolescents suffering from schizophrenia indicate that compared to their healthy peers, they show more intense impulsive behaviors and have more difficulty with delaying gratification [66]. There are also reports on the role of impulsivity in the pathogenesis of schizophrenia. A correlation has been found between a high level of impulsiveness and such behaviors as acts of violence, aggression, suicide risk and substance abuse [67]. There is also evidence [56] of limited negative priming (excluding the Stroop effect) among violent offenders with schizophrenia. However, there was no association between reduced cognitive inhibition and impulsiveness among the participants. Although these findings should be interpreted

with caution, cognitive inhibition in schizophrenia may be unrelated to the impulsive behaviors that are commonly associated with violence [56]. Kirkpatrick and Buchanan [68] indicate that deficit patients are no different from non-deficit patients in impulsive non-conformity or severity of psychotic symptoms.

Despite the keen interest in the topic of impulsivity in schizophrenia, there are still few reports on impulsivity in deficit schizophrenia, as well as analyzes comparing impulsivity between the deficit and non-deficit patients.

6. CLINICAL CORRELATES OF COGNITIVE CONTROL AND IMPULSIVITY

Clinical factors included in schizophrenia research that may be significant for cognitive control and impulsivity include, among others, positive and negative symptoms, general psychopathology, cognitive deficits, loss of energy, psychosomatic symptoms, medications taken, disease duration, treatment discontinuation and the number of exacerbations.

Impulsiveness often contributes to poorer clinical outcomes and complicates the treatment of patients with schizophrenia. It also increases the risk of violence against others [69,70], promotes substance abuse [71] and increases the likelihood of suicide [72]. It is associated with the need for intensive and long-term hospitalization [73,74,75]. In addition, impulsiveness is one of the key factors in the selection of antipsychotic drugs [76,77,78,79,70]. Research suggests that cognitive control is a clinically significant biomarker of mental illness [80]. Patients with earlier onset schizophrenia are reported to score much higher on impulsiveness and lower on cognitive processing [81]. According to available studies, positive symptoms of schizophrenia, alongside a history of physical and sexual abuse before the age of 18, are significant predictors of greater impulsivity in patients with schizophrenia. In addition, schizophrenia is more common in males than females [82].

The Positive and Negative Syndrome Scale (PANSS) is a commonly used tool to measure positive and negative symptoms in schizophrenia spectrum disorders [83]. Research indicates the existence of a positive correlation between impulsivity, positive and negative symptoms and the overall PANSS score [82]. What is more, the age of onset is related to the cognitive factor on the PANSS Scale.

7. NEUROBIOLOGICAL CORRELATES OF COGNITIVE CONTROL AND IMPULSIVITY

The neuroanatomical correlates of inhibition and impulsiveness in patients with schizophrenia have long been sought for. Inhibition is the cognitive process of resolving cognitive conflict [84], otherwise known as interference. Interference control is exercised during the Stroop Test, involving numerous brain structures [85]. The inhibition process is executed in a sequential fashion, with an initial key role of the anterior cingulate cortex (ACC). In the ACC, interference conflict is detected, monitored and processed [86,87,84]. Information is then selected in the dorsolateral prefrontal cortex (DLPFC). The DLPFC is ultimately responsible for resolving the interference conflict [88].

Research indicates a positive correlation between impulsivity on the BIS-11 and activation of those areas of the cortex that play a crucial role in response inhibition. Namely, schizophrenic patients demonstrate blunted activation in the right ventrolateral prefrontal cortex (VLPFC), i.e., a key region for motor response inhibition [89]. In their study, Hoptman et al. [90] examined the relationship between cognitive deficits and aggression in schizophrenia. Event-related potential (ERP) and resting-state functional connectivity were measured in 21 schizophrenic patients and 21 controls during their performance of a stop signal task. The patients showed prolonged stop signal reaction time (SSRT) compared to the control group. Multiple regression analyzes showed that longer SSRTs were independently associated with patient status, reduced amplitudes of successful stop trials, and decreased anticorrelated resting-state functional connectivity between visual and frontoparietal cortical networks. Inhibition studies [91] in early-onset schizophrenic patients demonstrated specific lateralized impairments in response inhibition. These findings are consistent with reports on immature frontostriatal networks in early-onset schizophrenia, and implicate areas

such as the premotor cortex and supplementary motor area – which are thought to play a role in both voluntary initiation and inhibition of movement. This research demonstrates that in patients with early-onset schizophrenia inhibition is impaired due to lateralization [91].

Neuropsychological and neuroimaging evidence (based on e.g., positron emission tomography and functional magnetic resonance imaging) indicates a relationship between deficits in the dorsolateral prefrontal cortex (DL-PFC), activation and impulsivity of the VL-PFC and anterior cingulate cortex (ACC). Findings from other available neuroimaging studies suggest negative correlations between increased levels of impulsivity and gray matter volume in the anterior cingulate, fronto-polar, and superior parietal areas in patients with comorbid schizophrenia and addiction [92]. In turn, patients with schizophrenia and a history of violent behavior demonstrated reduced activity in the left caudate nucleus, as well as the left thalamus. Patients with no history of violence had decreased activity in the left caudate nucleus [93]. Patients with schizophrenia have also been reported to manifest deficiencies in DLPFC activation in context-sensitive tasks [94]. Reduced ACC activity correlated with the presence of the task [95], while both reduced ACC and left DL-PFC activity were observed during inhibition (Rubia et al., 2001). Schröder et al. [96] found links between hyperactivity of the parietal cortex and motor area and disorganized thinking. In addition, hypo-activity of the left paracingulate cortex was observed in patients during the performance of a Stroop task [97].

Findings [98] implicate numerous, perhaps interrelated, brain structures in the genesis of impulsive behavior. Researchers suggest that dysfunction in the emotion regulation circuitry may play a role in the emergence of impulsive/aggressive behavior. Thus, impaired white matter integrity in such a circuit may be associated with dysregulation of impulsivity and aggression. Research [64] shows a significant and strong association between reduced cortical thickness in the ventral prefrontal areas and increased impulsivity in schizophrenia. What is more, findings tend to attribute aggressive tendencies in stabilized schizophrenia patients to increased impulsivity, not psychosis.

Available data suggest a relationship between impulsivity and the volumes of various brain structures. Indeed, there appear links between increased impulsivity and reduced orbitofrontal and hippocampal volumes [99], as well as reduced gray matter volume in the ACC, frontal and superior parietal areas in people with schizophrenia [92]. Other studies on impulsivity have found a task-specific relationship between the medial superior frontal gyrus, the ACC and the cerebellum in schizophrenic patients compared to controls [100]. In turn, Kumari and colleagues demonstrated that impulsivity is associated with reduced volume of the orbitofrontal cortex, as well as the hippocampus in people suffering from schizophrenia, which may indicate problems with inhibition [101]. Although the results of structural and functional studies to date indicate a relationship between impulsivity and inhibition in schizophrenia, the distinction between its deficit and non-deficit types has been somewhat neglected in this respect.

8. NEUROPSYCHOLOGICAL REHABILITATION AND SELF-CONTROL

Abundant body of evidence points to the deterioration of social skills, poorer professional adaptation, reduced quality of life or symptom exacerbation associated with deficits in psychomotor speed, attention, information processing, memory, and especially executive functions in people suffering from schizophrenia [102,103,104,105,106]. Executive function deficits progressing over the course of the disease are believed to be a key element underlying poor functioning of schizophrenia patients [107,108,109].

Research [110] indicates that people with schizophrenia show impaired social cognition, which is manifested by difficulties in identifying emotions, connecting with others, inferring their thoughts, and providing emotional responses. These social cognitive impairments disrupt social bonding and are strong determinants of impairment in daily functioning of patients. Therefore, there is a need for therapy and rehabilitation, especially aimed at patients suffering from the deficit subtype, as it can help them achieve functional improvement and significantly im-

prove their quality of life. Thanks to the available methods, it is possible to rehabilitate individual areas – improving general functioning, overall fitness, or connectivity and activity of specific areas of the brain – which is primarily related to neuroplasticity mechanisms [111]. Although data on patients with schizophrenia are not unambiguous, there is no doubt that illness-related deficits have a significant effect on the lives of patients, and thus on the functioning of the entire society. Cognitive rehabilitation is reported as significantly linked with improvement in the cognitive functioning of deficit patients, especially in working memory, executive functions and attention [112].

Conventional drugs used in the treatment of schizophrenia have a negligible effect on the emerging cognitive deficits [113]. Due to the poor effects of pharmacological treatment, therapy based on cognitive training and rehabilitation is becoming increasingly popular among both researchers and clinicians [114, 106]. There is evidence that rehabilitation leads to improved brain activity in patients with schizophrenia who suffer from executive and cognitive deficits [115,116].

A variety of programs have been developed to enhance cognitive and executive performance of patients with schizophrenia. Selected programs and techniques used in the neuropsychological rehabilitation of people with schizophrenia and deficits within inhibition and impulsiveness are presented in table 4.

Table 4. Characteristics of common cognitive rehabilitation programs in schizophrenia

Cognitive rehabilitation program	Description
RehaCom	A rehabilitation software designed for training of cognitive functions. Initially aimed at neurorehabilitation of cognitive functions in neurosurgical and neurological patients. Studies on its effectiveness in patients with schizophrenia showed a moderate improvement in visual attention (performance of the Stroop test, selected WCST parameters and a reduced number of non-perseverative errors [117]. Some of the available studies also indicate an improvement in the PANSS score [118].
Cognitive Training (computerized using CogRehab (Psychological Software Services, Indianapolis, IN, USA); also available in paper version)	The training includes tasks involving various cognitive domains, i.e. verbal, visual memory, language, visual-motor skills, orientation, alertness, processing speed, etc. It usually lasts about 1-2 hours a day and is repeated a few times a week. The program is linked to weekly therapy sessions.
Cognitive Enhancement Therapy (CET)	Training focused on neurocognitive and socio-cognitive functioning. Its main focus is on improving the „bottom-up" processing of critical social stimuli, with "top-down" executive control over dispersed information and emotional stimulation through integrating cognitive exercises based on attention training and problem solving, active group exercises teaching multi-perspectivity, information processing and social context assessment. The CET provides a support base to enhance neural processing. The program involves weekly sessions, which last for up to two years. Evidence shows that the CET can successfully improve both social and nonsocial cognitive deficits in schizophrenia. Its effects include improvement in the terms of neurocognition, neuroplasticity, processing speed, and understanding social stimuli.
Brain Fitness Program (BFP)	Program supporting brain plasticity, restoring and strengthening hearing, perception and working memory processing based on six exercises of increasing complexity.
Method NEAR	Based on computerized training, patients discuss strategies they have learned while performing cognitive tasks and associate them with everyday activities.
Cognitive Adaptation Training CAT	A form of cognitive rehabilitation that can be done at home. A strategy that aims to train cognition in order to later solve everyday life problems that are specific to the user.

Cognitive rehabilitation is composed of a set of tasks enhancing cognitive functions, delivered via computer-assisted programs or more conventional "paper and pencil" tools [119,120].

It is normally administered to serve a specific purpose, e.g. functional adaptation, involving activities of daily living; training of specific processes targeted at a selected cognitive skill; or general stimulation, consisting of non-specific training that covers numerous cognitive skills [134]. Cognitive rehabilitation for people with schizophrenia typically relies on widely used brain injury rehabilitation programs. Cognitive skills training is part of a larger treatment approach that also involves psychoeducation, social skills training, and professional reorganization [121].

Numerous studies [122,123,124,125,126] suggest that cognitive training should focus not only on developing selected cognitive functions, but also target general brain stimulation. These data are consistent with the neurodevelopmental epistemology of brain neuroplasticity and the potential for rehabilitation through cognitive training [105]. A meta-analysis by Kluwe-Schiavon et al. [114] suggests that program length and intensity are crucial in assessing the effectiveness of cognitive rehabilitation. Available meta-analytical findings also report long-term effects of such interventions (two or even six years after cognitive training) [102,123,126,127,124]. Conventional pharmacological treatment does not offer fully satisfactory cognitive and socio-affective changes. Combined with computer-assisted training, however, it may be more effective, especially in terms of cognitive flexibility dependent on processing speed and problem solving [112]. Cognitive training is safe and can be offered to young patients. It can be construed as a preventive measure in the earlier stages of the disease. Early recognition and cognitive intervention can limit deterioration of deficits and likely improve real world functioning of patients with psychosis [111]. The benefits of cognitive rehabilitation are particularly significant in the case of chronic and severe forms of schizophrenia. Research suggests its good effects in terms of memory, attention, problem solving, social cognition, independent functioning in everyday life and social adaptation. In the case of early detection, rehabilitation can stop or limit rapid development of deficits in psychotic disorders. It is also considered a useful preventive intervention in individuals with confirmed susceptibility to the development of schizophrenia [111].

Cognitive rehabilitation programs in schizophrenia are effective tools that allow patients to improve cognitive functions, and thus contribute to better functioning in everyday life. Unfortunately, despite numerous studies devoted to the neuropsychological rehabilitation of people with schizophrenia, the issue of rehabilitation of patients with its deficit variant remains largely unexplored. This seems a particularly important area because it is these patients who have predominant, persistent and primary deficit symptoms that experience greater functional difficulties in everyday life. In addition, the presence of severe cognitive impairment may particularly hinder therapeutic success in people with the deficit form of schizophrenia.

9. CONCLUSIONS

Despite numerous studies on cognitive functioning in schizophrenia, there are still some gaps regarding the differences between its different subtypes, especially deficit and non-deficit variants, which encourages further empirical analyzes in this area.

The available data indicate that people with deficit schizophrenia are characterized by poorer cognitive inhibition and greater impulsivity, but the behavioral consequences thereof still remain unclear. Structural and functional studies of the brain show a relationship between impulsivity and inhibition in schizophrenia, but to date few have analysed them in patients with the deficit type.

Embedding research results in modern theoretical models of inhibition and impulsivity processes can be helpful in understanding the nature of their disorders in deficit schizophrenia and likely differences emerging between patients with deficit and non-deficit schizophrenia. In addition, further research is needed to determine the relationship between cognitive control mechanisms and impulsivity in people with deficit and non-deficit schizophrenia, because understanding the essence of the disease is crucial not only in the diagnostic context, but also for the effectiveness of treatment. Despite the growing availability of various forms of neurorehabilitation, still little is known about their effectiveness in reducing cognitive deficits in defi-

cit schizophrenia. The development of programs dedicated to people with this form of the disease could be of great clinical importance, and in a broader context, could significantly contribute to improving the quality of life of patients and their families.

Author contributions

All authors contributed to and have approved the final manuscript: "Cognitive control and impulsivity in deficit schizophrenia: neuropsychological and clinical characteristics".

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