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Metacognitive Dysfunction in Schizophrenia

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1. Introduction

1.1 Metacognition

Metacognition is the high-level cognitive function that can be defined as “any knowledge or cognitive process that refers to, monitors, or controls any aspect of cognition” [1]. This definition comes from developmental psychology based on Piaget's theories when Flavell, in the 1970's, used the term metamemory in regard to an individual's ability to manage and monitor the input, storage, search and retrieval of the contents of his own memory [2]. Nowadays two areas of cognitive science are included under the umbrella concept of metacognition: the knowledge of cognition (metarepresentation) and the cognitive control of the cognitive processes (executive function).

Metarepresentation is a higher-order representation of some kind [3]. That is, a metarepresentation is a representation of a representation or, in other words, knowing about knowledge. It includes such diverse aspects as auto-consciousness/self-awareness, Theory of Mind (ToM) and other diverse “meta” constructs as metamemory, metareasoning and meta-emotion or emotional intelligence. All these areas of metacognitive knowledge have in common the concept that thinking is directly focused on representations, rather than on the external world. Metarepresentation is a determinant for the human capacity of social cognition that, in schizophrenia, is a construct composed by five domains: ToM, social perception, social knowledge, attributional bias and emotion processing [4]. Finally, constructing the narrative of self-identity largely depends on social cognition [5]. Scholl and Lesley [6] propose that ToM relies on the function of one specific brain module. Conceived in the classical way modules are defined as an innate, encapsulated and domain-specific part of the cognitive architecture, but with the characteristic of being a ‘diachronic’ module with the dynamic capacity to attain its character from the environment. This ToM module is the basic cognitive function that capacitates the four other more molar metarepresentative domains of the social human brain. Although the detailed implementation of the ToM module still remains unknown, probably it implies areas of complex cerebral connectivity [7] in the core circuit for imitation provided with neurons with mirror properties: posterior inferior frontal gyrus, ventral premotor cortex, the rostral part of the inferior parietal lobule and posterior sector of the superior temporal sulcus [8]. Analyzing data from functional neuroimaging, Gallagher and Frith [9] conclude that the ability to mentalize is mediated

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more concretely by a circumscribed region of the anterior paracingulate cortex. This region seems to be strongly associated with a more widespread network of cerebral regions involved in social cognition including the amygdala and the orbital frontal cortex.

Cognitive control, executive function or executive control, on the other hand, is the constellation of cognitive processes closely related to the frontal lobe functioning that monitors and controls cognition. It is composed of three independent but related dimensions: shifting between mental sets or tasks (Shifting), updating and monitoring of working memory contents (Updating), and inhibition of prepotent responses (Inhibition) [10]. Executive functioning is needed for any complex cognitive demand. It is genetically determined to a high degree and closely depends on dopamine neurotransmission [11] as well as the connectivity between network nodes of frontoparietal and frontostriatal circuits [12]. But, contrary to the case of ToM, and due to its heterarchical associative nature, executive function does not meet the characteristics to be implemented in one brain module. The brain cortex is functionally structured in cognits, which can be defined as “an item of knowledge about the world, the self, or the relations between them” [13]. A cognit is made up of groups of neurons and the connections between them. As more complex cognitive demands are required, the associative cognits of higher hierarchy are activated in the perception-action cycle. Cognitive functions overlap with each other from the simpler to the more complex and human-specific at the top of the hierarchy. So, the executive control in one perception-action cycle constitutes what Fuster [13] names as the high-level cognit that is broadly distributed in associative cortical areas or what Fodor years ago referred to as the one central process which does not have a modular architecture [14].

In brief, we differentiate two components in metacognition: a) the module for ToM, which is the core of social cognition and self identity and b) executive control, which is not modular but depends on broad cortical connectivity, and acts as the mediator factor integrating ToM in the global function of cognition. Executive dysfunction [15, 16] and ToM deficits [17, 18] have been confirmed as important conditions in schizophrenia. Furthermore, they are key concepts in the development of new evidence-based psychological treatments for schizophrenia, such as, Metacognitive training (MCT) [19] or Cognitive Remediation Therapy (CRT) [20], where improvement in daily functioning was predicted by better executive functioning [21] achieved by modifying metacognitive thinking strategies. So, improved knowledge of the metacognitive disturbances in schizophrenia appears to be a promising topic with high clinical and research interest.

1.2 Metacognitive deficits in schizophrenia

At the neurotransmission level, dopamine abnormalities appear to be related to schizophrenia both in metacognitive and social cognitive disorders as well as in executive disorders. Oxytocinergic and dopaminergic signalling in the amygdala result in impaired emotional salience and consequent social cognitive disturbance [22]. Dopamine presynaptic abnormality is the “final common pathway” causing the aberrant salience that is the gateway in to the psychosis [23] and is still considered the main target in the pharmacological treatment of the disease. At a higher level of analysis, social cognitive networks, executive control networks and the speech-related resting state network, have abnormal connectivity [24]. Executive function is the most global cognitive function and its deficit in schizophrenia is well known from the early states of the disease as well as in non-

affected first degree relatives [15, 16]. Moreover, executive dysfunction is closely related to the dopamine and other monoamine genetic differences that could mediate one important component of the genetic risk to the disease [11].

The impact of this deficit on the different domains of social cognition has been studied. Couture et al [25] propose that deficits in ToM could cause hostility-biased attributional style thereby leading to problems with social cognition and consequently the social functioning of the subject. This term has been partially confirmed by a path analysis study that found ToM to be a partial mediator between global neurocognition and social competence [26]. However, the hostile attributional style is only related to symptoms, ToM and social cue detection deficits being the two dimensions most closely correlated with functional outcome [27]. How much of the ToM deficit in schizophrenia is due to executive dysfunction has been a heated topic of debate, similar to what has happened in other diseases with cognitive dysfunction. For example, comparing bipolar patients to healthy controls, the worse execution of ToM tasks remains significant after controlling for executive dysfunction [28]. In another study [29], ToM problems were more closely related to attention disorders than executive dysfunction, ToM appearing as a state marker but not as a trait of the disease. So, ToM deficit seems to be dissociated from executive dysfunction in bipolar disorder. But in Parkinson's disease, the independence of ToM disorders from other cognitive dimensions is not as clear. Peron et al [30] found a significant correlation between the Stroop interference score and a subscore of the faux pas recognition test (explanation score) and argue that ToM impairments in advanced PD patients could be the consequence of general cognitive deterioration and that the dysexecutive syndrome could explain the mental state inference deficit. In a similar way, it has been proposed that ToM dysfunction in schizophrenia may reflect impairments in related non-ToM abilities, such as executive functioning, rather than representing a specific ToM deficit [17]. Those models proposing that poor performance in ToM tasks in healthy and clinical populations [31] reflect executive dysfunction argue that it could be due to the difficulty to disengage from and inhibit salient information or, on the other hand, be due to problems in the manipulation of representations of hypothetical situations. The former case relates mainly to shifting and inhibition dimensions of executive functioning and can be evaluated by the Wisconsin Card Sorting Test, while the latter indicates problems with updating working memory which is evaluated by the Tower of London task. The relationship between ToM and executive function has been systematically reviewed by Pickup [32] who refers consistently to the question indicating that specifically impaired ToM in schizophrenia actually exists reflecting dysfunction of a domain-specific cognitive system, rather than only a domain-general executive impairment. The author concludes that executive function and ToM are independent in schizophrenia, leading to the independent contribution of ToM in functioning. Furthermore, ToM impairments appear to be both a trait and state deficit.

The current state of knowledge points out that ToM constitutes a brain module that can be independently affected in schizophrenia constituting a domain-specific ToM deficit. But this fact is not incompatible with the coexistence of domain-nonspecific metacognitive deficits. Social perception, social knowledge, attributional bias and emotion processing are social cognition domains that, although based on ToM, depend on other basic and executive cognitive functions that also require wide brain connectivity. Schizophrenia has been proposed to reflect the disconnection syndrome of the highest hierarchy human networks

such as those implicated in social cognition and Theory of Mind (ToM) [7]. Multimodal network organization is abnormal in schizophrenia, as indicated by reduced hierarchy, the loss of frontal and the emergence of nonfrontal hubs, and increased connection distance [33]. These authors defined the anatomical networks, studying inter-regional correlations of high-resolution magnetic resonance image, revealing the usefulness of correlation techniques in the study of cortical functional networks. So, in conjunction with the domain-specific ToM deficit, other domain-nonspecific metacognitive dysfunction due to disconnection of associative cortical areas could exist, acting as a moderator variable on the effect of ToM deficits in the pathophysiology of schizophrenia. The association of dysfunction in the module for ToM jointly with executive dysfunctions due to cortical disconnection could increase the risk for clinical schizophrenia in vulnerable people. In this manner, the dopaminergic ToM dysfunction acts as the necessary cause or vulnerability trait of the disease, while the increased effect due to the coexistence of cortical disconnection leads to the effect from the trait of vulnerability to the state of clinical schizophrenia.

The objective of this review is to determine candidate domain-nonspecific metacognitive deficits in schizophrenia. We will investigate the association described in the schizophrenia literature between executive and metarepresentative dysfunctions and we will show that the domains of executive function and metarepresentation which are linked to a large size effect constitute a network that, when connectivity decreases, leads to domain-nonspecific metacognitive deficits.

2. Method

We have done a systematic review on PubMed and PsychoInfo in August, 30th 2011 introducing the following syntax. PubMed: Schizophrenia [MeSH Terms] AND (("dysexecutive"[All Fields] AND ("syndrome"[MeSH Terms] OR "syndrome"[All Fields])) OR executive [All Fields] OR frontal [All fields]) AND ("Theory of Mind" [All Fields] OR "metacognition" [All Fields] OR "metarepresentation" [All Fields] OR "social cognition" [All Fields]). PsychoInfo: Schizophrenia AND (dysexecutive OR executive OR frontal) AND ("Theory of Mind" OR metacognition OR metarepresentation OR "social cognition").

The obtained articles were reviewed by reading the abstracts. Articles were selected for full reading when the abstracts referred directly or indirectly to any association measure as correlation, regression, odds ratio or risk ratio between metarepresentation and executive function in schizophrenia. After that, the articles were definitively included if they provided the association measure. Finally, only the associations with large effect size were considered for analysis. The criteria for considering the effect size was that proposed by Cohen who accepts $r \geq 0.5$ (shared variance equal or greater than 0.25) as a large effect size [34]. These will be our proposed domain non-specific metacognitive deficits.

3. Results

Sixty-five articles were obtained from PubMed and 125 from PsycInfo. After abstract reading, 36 were fully read and 14 were definitively included in the analysis. Table 1 shows the article references and their main characteristics and results. All of them were published after 2000. Most of the works explores the relationship between ToM and executive function as secondary, not the main objective. Only Lysaker et al [35] specifically evaluated this

relationship. The measuring instruments used are highly variable, especially in ToM, where there is little overlap between studies. Regarding the instruments used to assess executive function, the Wisconsin Card Sorting Test (WCST) is highlighted. It is used in half of the studies. As a relational measure the authors use the correlation coefficient in 86% of the studies, one used analysis of covariance (ANCOVA) and one used linear regression. Most authors do not specify what type of correlation coefficient is chosen, except in three specific cases (two works with Pearson's correlation coefficient and one with Spearman's coefficient).

Reference Country	ToM measuring instruments	Executive function measuring instruments	Relation measuring Results
[42] Australia	Mind in the Eyes test -ME-	Reality-unknown (low-inhibition false belief - LOFB-) task. Reality-known (high-inhibition false belief - HIFB-) task.	Correlation ME/LOFB: .40* ME/HIFB: .38* *: p<.05
[43] United States	Bell Lysaker Emotion Recognition Test (BLERT) total score. Hinting Task total score. Bell Object Relations Inventory (BORI) Egocentricity Scale. Rapport item on the Quality of Life Scale (QLS).	Wisconsin Card Sorting Test (WCST).	Correlation BLERT/WCST: .184* Hinting/WCST: .260*** BORI/WCST: .169* QLS/WCST: .115 (ns) *:p< .05 ***:p< .001
[38] Germany	Ekman and Friesen's Pictures of Facial Affect. ToM novel series: <i>ToM questionnaire</i> ; <i>ToM total score</i> .	Wisconsin Card Sorting Test (WCST). Behavioural Assessment of the Dysexecutive Syndrome (BADS): <i>Key Search Test (KST)</i> ; <i>Zoo Map Test (ZMT)</i> .	Correlation ToM/WCST errors: -.114 (ns) ToM/WCST pers: -.354 (ns) ToM/KST: .605** ToM/ZMT: .492* *:p< .05; **:p< .01 Correlations with ToM total score.
[44] United States	Facial Emotion Identification Test (FEIT).	Wisconsin Card Sorting Task (WCST).	Correlation FEIT/WCST: .34 (ns)
[45] Japan	Role-play test (RPT) including fifteen parameters.	Trail Making Test (TMT) A and B: <i>differences in score B - A</i> .	Spearman's correlation RPT (Recognition of goal)/TMT: -.36* RPT(Generating thought)/TMT: -.36* *: p=.01

Reference Country	ToM measuring instruments	Executive function measuring instruments	Relation measuring Results
[41] United States	Means-Ends Problem-Solving (MEPS). Social Cue Recognition Test (SCRT): <i>concrete false-positive errors (CFPE) and abstract false-positive errors (AFPE)</i> .	Wisconsin Card Sorting Task (WCST).	Correlation MEPS/WCST: .35* SCRT(CFPE)/WCST: -.57** SCRT(AFPE)/WCST -.56** *:p< .05; **:p< .0001
[31] Australia	Picture-sequencing task: <i>False-belief mean position score (FBmps) (mentalising ability)</i> .	Computerised version of the Tower of London task: <i>Proportion of ToL problems solves in the minimum (Min); Number of moves taken beyond the minimum (Moves); Initial planning time (Init); Subsequent planning time (Sub)</i> .	Correlation FBmps /Min: .38 (ns) FBmps /Moves: -.25 (ns) FBmps /Init: -.15 (ns) FBmps /Sub: -.26 (ns)
[39] Australia	Picture-sequencing task (false-belief picture-sequencing score): FB-ps. Non-literal speech comprehension by computerized story comprehension task: <i>(metaphor hit rate): Met-hit; (irony hit rate): Iron-hit</i> .	Picture-sequencing task (<i>capture picture-sequencing score</i>): C-ps Computerized version of the Tower of London: (<i>% ToL problems solved in min. no. of moves</i>): ToL%; (<i>average no. moves taken on ToL task</i>): ToL-mov; (<i>subsequent planning time on ToL task</i>): ToL-sub.	Correlation FB-ps/ C-ps: .57** FB-ps/ ToL-mov: -.46* FB-ps/ ToL-sub: -.58** Met-hit/ToL-sub: -.44* Iron-hit/ToL-mov: -.40* Iron-hit/ToL%: -.42* *:p< .05; **:p< .01
[35] United States	Metacognition Assessment Scale (MAS): <i>Understanding one's own mind (UOwM); Understanding the others' mind (UOtM); Mastery (M); Total (T)</i> .	Delis Kaplan Executive Function System (DKEFS): <i>Design Fluency Switching (DFS); Verbal Fluency Switching (VFS); Color Word Switching (CWS); Sorting Task (ST); Word Context (WC); Twenty Questions (TQ)</i> .	Spearman's correlation UOwM/ST: .47** UOwM/WC: .47** UOwM/TQ: .32* UOtM/DFS: .30* UOtM/DFS: .31* M/VFS: .31* M/TQ: .32* T/VFS: .34* T/TQ: .43** *:p< .05; **:p< .01

Reference Country	ToM measuring instruments	Executive function measuring instruments	Relation measuring Results
[40] Germany	Computerized series of six picture stories (PS).	Wisconsin Card Sorting Test (WCST): <i>perseverative errors</i> . Behavioural Assessment of the Dysexecutive Syndrome (BADS): <i>Zoo Map Test</i> .	Parametric correlation PS/WCST(FP): .60** PS/BADS(FP): .35* PS/WCST(NFP): .49** PS/WCST(NFP) .487** *:p<.05; **:p<.01 FP: forensic patients; NFP: nonforensic patients.
[46] Italy	First-Order ToM Tasks (F-Or-T). Second-Order ToM Tasks (S-Or-T).	Tower of London test (TL). Phonemic verbal fluency test (PVF).	ANCOVA F-Or-T/TL: F= .46 (ns) F-Or-T/PVF: F= .35 (ns) S-Or-T/TL: F= .16 (ns) S-Or-T/PVF: F= .18 (ns)
[37] Italy	Comprehensive Affect Testing System (CATS).	Wisconsin Card Sorting Test (WCST).	Univariate linear regression analyses CATS/WCST: $\beta = .276, p = .04$ $\beta^* = .253, p^* = .048$ *: partial regression analyses excluding face perception.
[47] United Kingdom	Theory-of-mind (ToM) task.	Wisconsin Card Sorting Test (WCST).	Pearson's correlation ToM/WCST: -.46 p=.01
[36] Norway	Emotion perception (EP): <i>Visual emotion identification; Visual emotion discrimination; Auditory emotion identification; Auditory emotion discrimination.</i> (Z-transformed composite score based on principal components analysis is the measure of emotion perception).	Neurocognition (NC): <i>Psychomotor speed (Digit Symbol; SS); Semantic fluency (Animals & Boys' Names; SS); Executive control (Inhibition/Switching; SS); Verbal learning (CVLT-II; T-score).</i> SS: Scaled score; T-Score: Total score. (Z-transformed composite score based on principal components analysis is the measure of neurocognition).	Pearson correlation EP/NC: .67 p=.01

Table 1. Selected articles for analysis.

In connection to the results found there is great variability, generally showing positive correlations with significant results in those papers that perform multiple comparisons. Studies with negative correlations in their comparisons or no significant results are detected. Probably this high variability is conditioned by the variety of measuring instruments used by different authors, as the mention of ToM does not coincide in any author. Another factor explaining this phenomenon is probably the definition applied by different authors in the conceptualization of the term ToM and executive function, which leads to the use of different instruments as mentioned previously.

Six of the selected fourteen studies report any large effect size correlation. Three of them refer to emotion processing [36-38], two refer to ToM [39, 40] and one paper refers to social perception [41]. Studies were not detected with correlations between executive dysfunction and social knowledge or attributional bias. The results of our review show that executive dysfunction could act as a mediator or moderator variable on the clinical consequences of the domain-specific ToM deficit. In addition, emotion and social processing problems can be proposed as candidate domain-nonspecific deficits highly influenced by executive dysfunction.

3.1 Executive dysfunction influencing the effect of ToM deficits

Although ToM deficits constitute an independent and probably modular dysfunction in schizophrenia, it is not free from intermediate variables affecting its clinical manifestation. One study [40] evaluated ToM abilities using a computerized series of six picture stories depicting the cooperation of two characters, one character deceiving another, or two characters cooperating to deceive a third. It revealed that in a sample of forensic schizophrenia patients, ToM task performance inversely correlated with the amount of WCST perseverative errors ($r = -0.598$, $p < 0.001$) and with the performance on the Zoo Map Test ($r = 0.346$, $p = 0.048$), a subtest taken from the Behavioural Assessment of the Dysexecutive Syndrome (BADs). The results were similar in other samples of nonforensic schizophrenia patients. Another study testing the effect of mind-reading on the pragmatic language impairments in schizophrenia [39] observed that the performance in the mind-reading task was related to executive functioning. The task used to study mind-reading was a picture-sequencing task. Stories were presented on cards referring to four experimental story types: social-script stories testing logical reasoning about people without needing to infer mental states, mechanical stories testing physical cause-and-effect reasoning, false-belief stories testing general mind-reading, and capture stories testing inhibitory control. Executive functioning was tested using the capture picture-sequencing score to measure inhibitory control and a version of the Tower of London (ToL) task to measure planning. As a secondary result of the study, it was observed that executive dysfunction (errors in sequencing capture stories and poor ToL task performances) predicted mind-reading impairments and pragmatic comprehension deficits in schizophrenic patients. The association was $r = -0.57$ ($p < 0.01$) for false-belief picture-sequencing and capture picture-sequencing, and $r = -0.58$ ($p < 0.01$) for false-belief picture-sequencing and subsequent planning time on ToL task. But the effect of mind-reading on pragmatic comprehension deficits does not depend on executive dysfunction as this persists after controlling for executive functioning in a logistic regression model. So, in this case an independent ToM

disorder appears causing the pragmatic language deficits in addition or independently from the executive dysfunction, but is not mediated by it. The same study concludes that executive dysfunction and, beyond that, a selective difficulty with interpreting metaphors (most likely due to abnormal semantics) were associated with negative formal thought disorders, whereas positive formal thought disorders were associated with poor mind-reading. Its different clinical manifestation supports the differentiation between executive and ToM disorders.

These two studies are examples of executive dysfunction associated with ToM deficits. Executive dysfunction could affect the impact of ToM deficits in several result variables, as symptoms or functioning do. But we should determine if executive dysfunction acts as a modulating or as a mediating factor. A modulating factor modifies the size effect of the independent variable on the dependent variable. To our knowledge, the potential role of executive dysfunction as the moderator of the effect of ToM on symptoms or functioning is still unknown. On the other hand, a mediating effect is said to be present if the mediator is related to the independent and dependent variables and, furthermore, if a previously significant relationship between the independent and the dependent variables is no longer significant, or at least greatly reduced, when the mediator is controlled. This is not the case in the study of Langdon et al [39], which shows that the effect of ToM disorders on formal thought disorders persists after controlling the executive functioning.

We can conclude that, although sufficient evidence exists leading to the acceptance of the independence of the effects of ToM deficits on several clinical and functional targets, probably in some situations executive dysfunction can play a role as a modulating factor. In other cases it will be a merely coexistent deficit, but we can exclude that executive dysfunction acts as a mediator factor between ToM deficits and its functional and clinical consequences. In any case, the best way to characterise ToM deficits in schizophrenia is considering it a domain-specific deficit.

3.2 Executive dysfunction causing emotion processing problems

The result of our review suggests emotion processing problems as the most firm candidates for a domain-nonspecific deficit, defined as a deficit not depending on any single cognitive module dysfunction, but related to problems in the interaction or connection between diverse modules. Three of the selected studies address this topic.

The first of them [38] follows the proposal of Frith [48] who hypothesized that many symptoms typical of schizophrenia may be accounted for by a specific cognitive incapacity of schizophrenic patients to accurately attribute mental states to themselves or others, leading to disorders of willed action, disorders of self-monitoring and disorders of monitoring other persons thoughts and intentions. Brüne highlights the importance of differentiating non-social versus social cognitive testing in the study of schizophrenia since the latter may distinguish between patients and nonpatients better [49]. Considering that the perception of emotional states involves a ventral brain stream (amygdala and orbitofrontal cortex) whereas a dorsal pathway (superior temporal sulcus, inferior frontal regions, medial prefrontal cortex including parts of the anterior cingulate cortex) relates to ToM, the study aims to show the amount of explained variance of the patients' actual social behavioural

abnormalities which are due to emotion recognition and ToM capacities. The results reveal that schizophrenic patients differed significantly from healthy controls in the main measures of executive functioning, emotion recognition, and ToM. However, after controlling for executive functioning (as measured by perseverative errors on the WCST) and IQ, the effect disappears in happiness, fear and sadness recognition. In this case, executive functioning seems to be a mediating factor between non-social cognitive capacities and emotion perception. Furthermore, ToM and emotion recognition were not related to executive functioning in the healthy subjects of the control group while moderate associations appeared in the schizophrenic group, supporting executive functioning to be related to emotion perception and ToM only in the disease condition. The author concludes that the slowing of patient performance of ToM tasks is partly associated with their impaired understanding of the social interaction depicted in the stories, and with executive functioning. To determine the contribution of ToM and executive performance in the assignment to the schizophrenia group, a logistical regression analysis (backward-step) was done. Although ToM performance remained in the equation as the most powerful predictor of the odds of being in the group of patients, the number of perseverative errors in the computerized WCST also remained significant, but did not correlate with ToM performance in the patient group. The results of this study suggest independent contributions of ToM and executive functioning and support the differentiation we make between domain-specific and domain-nonspecific dysfunctions.

A second study [36] addressed specifically social cognition and learning potential as mediating variables between neurocognition and functional outcome in schizophrenia and confirmed that emotion perception has a mediating role in the association between neurocognition and social problem-solving. The authors propose that the mediating role that social cognition plays between neurocognition and functioning could be due to the recognition of emotional expressions in other people, probably depending on basic neurocognitive abilities such as working memory, visual scanning, and speech and face perception. To measure non-social cognition, a composite battery based on MATRICS was used, including Digit Symbol, Semantic Fluency, Inhibition/Switching and California Verbal Learning Test-II, Total List A learning. Emotion perception was assessed using the Face/Voice Emotion Identification and Discrimination Test that requires the identification of emotions from six alternatives in facial pictures and tape-recorded sentences. As functional outcome variable, a social problem-solving test was used. Emotion perception confirmed its mediating role in this study through its significant relation to both the independent (neurocognition: $r=.67$, $p<.001$) and the dependent variable (social problem solving, $r=.50$, $p<.001$) in conjunction with the elimination of the effect of neurocognition on social problem-solving when controlling for emotion perception in a regression analysis. The results are in line with other studies which have found that facial affect recognition is a partial mediator between neurocognition and social functioning (quality of life) in psychosis [50] or that social perception mediates between neurocognition and interpersonal role-playing skills [51].

The third study related to emotion processing which our review detected is by Rocca et al [37]. Its aim was to study the role of facial identity recognition in the context of cognitive functions and symptoms considering that facial emotion recognition deficits may be part of

a cognitive impairment in the domains of attention and executive functions. The executive assessment was done with the Stroop Test, the Trail Making Test and the Wisconsin Card Sorting Test (WCST). Facial emotion processing assessment used the Comprehensive Affect Testing System (CATS). The results of the study support the presence of a generalized deficit where both facial recognition and facial emotion recognition were affected in schizophrenia. Using linear regression analysis the authors conclude that executive dysfunction may explain a proportion of the variance in emotion recognition in conjunction with attention, face perception and verbal memory-learning. Executive functions explained 7% of the variance in emotion perception scores. In this case, the coincidence of diverse basic cognitive capacities is the cause of emotion perception deficits that we categorize as domain non-specific metacognitive deficits.

Emotion processing deficits appears to be the best candidate in a domain non-specific metacognitive deficit, which in part is due to executive dysfunction. Executive dysfunction has been viewed to act both as a causal factor in addition to other, as well as being a mediating factor between non-social cognition and emotion perception.

3.3 Executive dysfunction causing social processing problems

One study has been found with a relevant association between social cognition and executive functioning [41]. It studied the relationships between various domains of neurocognition and two forms of social cognition (social cue recognition and social problem solving) in patients with schizophrenia spectrum disorders. The study was based on the hypothesis that executive function is related to abstract cue recognition and social problem solving because both forms of social cognition require the ability to think in an abstract and flexible manner about diverse situations. After controlling for symptomatology, perseverative errors in the WCST accounted for 22% of the variance in a task of social cue recognition and 11% in a task of social problem-solving. The authors conclude that deviations in neurocognition may affect the ability of persons to recognize the intentions of others in common social situations, specifically when they are communicated in a more implicit manner. The social cue recognition deficits can occur via executive dysfunction, or but by other means, such as attention or memory deficits.

Social processing, similar to emotion processing, is not a simple or modular cognitive process but a complex capacity. Executive dysfunction probably affects social processing, acting as a causal or mediating factor but there is still little evidence to prove that. So, we accept deficits in social processing as a putative non-specific metacognitive deficit in schizophrenia.

3.4 Future research

ToM is a modular cognitive capacity relatively independent from its interrelation with other capacities which may be primarily altered in schizophrenia, specifically in those cases with predominance of autistic symptoms. The analysis of potential common endophenotypes shared by autism and a subgroup of schizophrenic patients appears to be an exciting area of research into metacognition and psychosis.

However, probably the most interesting area for further research is that of non-specific metacognitive disorders. First of all, a more detailed knowledge of the social cognitive domains that can be mediated or caused by executive dysfunction is necessary. Considering ToM to be the most nuclear social cognitive domain closely related to its specific module, emotion processing and social perception appear to be complex domains where the impact of connectivity pathology on executive functioning may be an important target for new studies. As executive functioning highly depends on dopaminergic neurotransmission and cortical connectivity [52], it could be found to be an important common pathway in the pathophysiology of the disease linking dopamine or glial disorders [53] to metarepresentative symptoms via executive dysfunction.

Social knowledge and attributional bias did not appear as candidates to be related to executive dysfunction or to metarepresentation disorders. We consider them to be basically related to episodic and semantic memory, being the final product of experience. Both are close to the thinking style of the patient, with his or her cognitive traits, but do not relate to the neurocognitive substrata of the disease.

Social cognition is a confirmed mediator between neurocognition and functioning. Metarepresentation is the key concept used to study social cognition and executive functioning. We propose to take into account the different nature of each of the five dimensions of social cognition in schizophrenia. ToM would be the basic cognitive module affected, which is an endophenotype and hence its dysfunction remains stable over time with little or no modification due to treatment. Executive dysfunction is due to dopamine or connectivity alterations and may act as a modulating or mediating factor when ToM disorders are the primary cause of the metacognitive disorder. Executive dysfunction could also be a causing factor independent from ToM. Emotion processing and social perception are domains more closely affected by executive dysfunction and probably both of them behave like state markers. Thus, they can be expected to fluctuate with clinical episodes and pharmacological treatments. Finally, social knowledge and attributional bias, as they relate to semantic knowledge and cognitive schemas, could be good candidates for improvement through psychotherapy. These speculations could serve as a guide for further research.

4. Conclusion

Metacognitive disorders in schizophrenia are central to the pathophysiology and psychopathology of the disease. ToM disorders are domain-specific and are related to a particular cognitive module. This chapter systematically reviews the literature on metarepresentation and executive dysfunction in schizophrenia to determine candidate domain-nonspecific metacognitive deficits. Emotion processing problems have been detected to be the best candidate. Executive dysfunction seems to be a mediating or causing factor that causes emotion processing problems which consequently lead to functioning deficits or symptom exacerbation. Social processing could be another non-specific metacognitive disorder but less evidence to sustain this term exists.

Social cognition acts as a mediator between non-social cognition and functioning. But social cognition itself is a heterogeneous construct that must be studied taking into account

metacognition, basic cognition and personality traits. An in-depth analysis of this topic could contribute to a better understanding of this new and exciting area of research in schizophrenia. We propose to consider ToM the nuclear metacognitive dimension. Emotion and social perception are domains based on ToM but highly dependant on connectivity and on executive functioning. And finally, attributional bias and social knowledge could depend on personality traits more than on metacognition.

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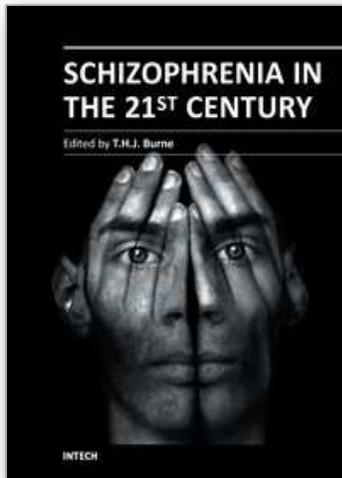
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Schizophrenia in the 21st Century

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Schizophrenia is a poorly understood but very disabling group of brain disorders. While hallucinations and delusions (positive symptoms of schizophrenia) feature prominently in diagnostic criteria, impairments of memory and attentional processing (cognitive symptoms of schizophrenia) are attracting increasing interest in modern neuropsychiatry. *Schizophrenia in the 21st Century* brings together recent findings on this group of devastating disorders. We are still a long way from having effective treatment options, particularly for cognitive symptoms, and lack effective interventions and ways to prevent this disease. This volume covers various current options for therapy, clinical research into cognitive symptoms of schizophrenia and preclinical research in animal models.

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