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SYNTHETIC METACOGNITION AS A LINK BETWEEN BRAIN AND BEHAVIOR IN SCHIZOPHRENIA

Abstract

Deficits in metacognitive capacity in schizophrenia can be conceptualized as existing along a spectrum from more discrete to more synthetic activities. These capacities may be of great importance in schizophrenia research given their potential to mediate and moderate the impact of illness-related factors on outcome. To explore this possibility this review summarizes research on synthetic metacognition using a paradigm in which metacognitive capacity is rated on the basis of spontaneously produced personal narratives. Evidence from a review of the literature shows that these deficits are detectable in patients with schizophrenia and are related to, but not reducible to, symptom severity and poorer neurocognitive function. Independent of symptoms and neurocognition, deficits in synthetic metacognition, which are likely linked to the brain's ability to integrate information, are related to a range of outcomes including functional competence, learning potential, and insight. These deficits may also play a role in long term psychosocial functioning via their impact on the ability to sustain social functions.

Keywords

• Schizophrenia • Neurocognition • Recovery • Narrative • Metacognition • Psychosis • Quality of life • Self

Bleuler suggested that the interruption of goal-directed behavior that characterizes schizophrenia is a result of complex, biologically based processes that lead to the unbinding of associations and the collapse of higher order understanding of oneself and others [1]. In the more than 100 years since, a more detailed picture has emerged of the sequence of events that result in dysfunction in schizophrenia. Genetic vulnerabilities for schizophrenia have, for instance, been linked to abnormalities in brain development that are reflected in neurocognitive deficits, such as impairments in information processing, verbal memory and executive function [2-7]. Further, these cognitive deficits may be linked to disruption in synchronized oscillatory activity of neural networks, which would allow for the effective integration of information and hence binding of associations [8-11].

Connecting these processes to behavior, neurocognitive dysfunction has been linked with poorer social, vocational, and community function. Bowie *et al*. for instance, found that processing speed and attention/working memory were uniquely linked with social competence and that social and functional competence mediated the effects of neurocognition on community and work functioning [12]. These neurocognitive deficits have been detected prior to the onset of illness [13,14] and observed to interact with symptom severity [15]. Neurocognitive compromise has also been implicated as a predictor of poorer response to psychosocial treatments [16].

Considering forces taking place outside of the mind of individuals, research has also suggested that social and community factors such as trauma, stigma, poverty, isolation, and attachment patterns exacerbate or are exacerbated by both brain function and neurocognition on their own, and in conjunction with more biologically based paths to dysfunction [17,18]. For example, Walker and Diforio proposed "a neural diathesis-stress model" of schizophrenia in which a neural mechanism for these phenomena results from the stimulating effect of the hypothalamus-pituitary-adrenal (HPA) axis on dopamine synthesis and receptors [19]. This gain of function may lead to abnormalities in dopamine receptors, which together with hippocampal damage, significantly influences hypersensitivity to stress in persons with schizophrenia. In the context of this model, Read *et al*. [20] developed "a traumagenic neurodevelopmental model" that proposes that genetic alterations lead to predisposing vulnerability in the form of hypersensitivity to stress related to adverse life events such as child abuse, which, according to recent evidence, contributes to the development of schizophrenia, likely through the influence of traumatic events on the developing brain and neurobiological abnormalities that frequently occur in patients with schizophrenia, such as over reactivation of the HPA axis or structural changes to the brain (e.g. hippocampal damage, cerebral atrophy, reversed cerebral asymmetry, ventricular enlargement), as well as dopamine, norepinephrine, and serotonin abnormalities.

As shown in Figure 1, this work, which initiated with Bleuler [1], offers a path from brain to behavior, linking genetics, brain

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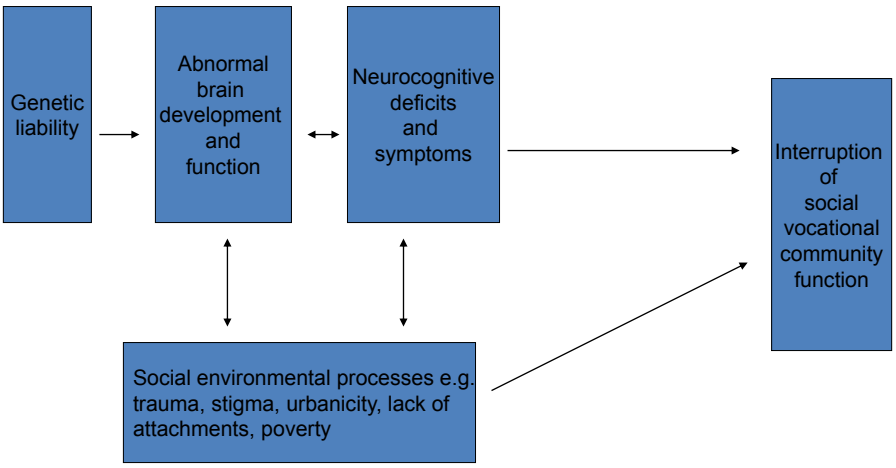

Figure 1. A path from gene to brain to dysfunctional behavior in schizophrenia.

development, cognition, symptoms, social and community experiences, and ultimately the interruption of goal directed behavior. This larger scheme provides an idea of different points for clinical intervention but also allows for a more nuanced inquiry into the proximate psychological factors affecting function in schizophrenia.

One specific area of interest concerns what is happening in the space in Figure 1 that lies in between manifestations of illness and psychosocial dysfunction. Certainly, it is not surprising that phenomena such as diminished neurocognitive capacity are related to impaired function because thought is needed for function. But exactly how does this occur? What psychological factors mediate the impact of symptoms and neurocognitive deficits on behavior? Are there phenomena specific to schizophrenia that play a role in dysfunction at this point in the model? Answers to these questions seem essential in order to develop treatments that intervene at the level most proximate to function. If we know how neurocognitive impairments and symptoms culminate in dysfunction, it may be possible to design treatments for persons with treatment-resistant cognitive deficits or symptoms, or for those whose impairments persist after cognitive deficits or symptoms have been resolved.

To address this issue, a program of research will be reviewed that has examined one set of processes which may mediate and/or moderate the impact of illness related variables such as symptoms and neurocognitive deficits on real world function. We propose that psychosocial dysfunction may result from a feedback loop in which symptoms and neurocognitive deficits influence and are influenced by deficits in synthetic forms of metacognition. We suggest that deficits in synthetic metacognition leave persons unable to make meaning of both illness related phenomenon and psychosocial challenges. Without possessing a sufficiently complex account of themselves and others, these individuals may be unable to make sense of what is happening. They may thus not know why they should take certain courses of action and hence social and vocational function may languish.

It has been proposed that phenomena such as neurocognitive deficits affect function because they leave persons less able to know how to perform basic functional and interpersonal tasks. Neurocognitive deficits may limit the rate at which persons can learn new skills, as well as accurately detect and respond to the demands of the environment [21]. In further iterations of this model, a lack of ability to perform tasks has been thought to lead to defeatism and withdrawal [22]. For instance, persons with deficits in executive function might incorrectly identify the motives or intentions of others and so not be able to know how to interact appropriately. They might similarly be unsure what actions are needed to achieve a certain end in a work setting and so struggle with an instrumental task and decide to give up. This work is certainly tied to an emerging set of interventions that try to teach people how to perform certain tasks, such as how to deliver a compliment or carry out a specific work task when under stress.

In this review we will not contradict that skill deficits are important to understanding outcome but add to it with the consideration of an additional possibility that deficits in metacognitive capacity interfere with the ability to know why to do certain things and not just how to do them. Harkening back to existential psychiatry, we will suggest that many with schizophrenia often fail to take certain action not only because they do not know how, or because they do not expect they will be successful, but often because they do not know *why* they should take those actions [23,24]. In daily life persons seek to repair rifts in relationships and endure difficult times at work because the hopes and dreams they possess make such repairs and persistence meaningful. Applied to schizophrenia then we wish to suggest, for instance, that metacognitive

deficits do not remove knowledge of how to make a compliment in a social encounter but may leave persons without an idea of why to deliver a compliment and so they may offer no compliment. Persons with schizophrenia might similarly not persist at work when under stress, not because they do not know how to persist but because of a lack of having any larger sense of themselves which would supply a reason for persisting. Figure 2 provides an illustration of how metacognitive deficits could be incorporated into standard models of the path from brain to behavior in schizophrenia.

To explore the possibilities offered by figure two, this review will first offer a definition of metacognition as a spectrum of mental activities which can involve synthesizing experience into integrated representations of self and other and can be a subject for reflection and subsequent revision. We suggest that disruptions in this form of metacognition closely parallel what Bleuler [1] described as the fundamental disturbance in schizophrenia. Second, we discuss recent efforts to operationalize and measure synthetic metacognitive deficits in schizophrenia and detail research offering preliminary confirmation of their potential links with both neurocognition as well as functional outcomes. Limitations and directions for future research and clinical implications are also explored. Of note, as portrayed in Figure 2, metacognition may potentially play a mediating role in the link between social phenomena and function, but this will not be discussed in the present review.

The concept of metacognition

Metacognition refers to a mental act in which persons forms a thought or idea about their own mental activities. The construct was first used in the education literature to capture learners' awareness of their own learning and what conditions best enabled learning, and then spread to different dimensions of experience such as self-regulation [25-27]. The psychological literature has similarly used the term to describe a broad range of phenomena. As we have summarized elsewhere [28], metacognition applied to human development and psychopathology has been used to describe a range of different mental activities. These activities may vary from one another according to the extent to which they involve focusing on discrete mental activities such as thinking about a specific isolated thought [e.g. 29] versus the integration or synthesis of a range of different experiences into a complex representation of self and others and then a reflection about that larger representation [30,31]. Intuitively, these discrete and synthetic metacognitive activities influence one another but as such are not reducible to one another. For instance, an awareness of discrete elements is needed to form larger ideas of oneself while awareness of oneself is needed to make meaning of discrete experience with the flow of daily life. Further, human beings' larger reflection about themselves and others has long been seen as more than a sum of psychological facts but rather emerging from a particular way of connecting and meaningfully situating those facts in relation to one another [32].

The distinction between discrete and synthetic forms of metacognition maps well onto a range of schizophrenia research [28,33]. Deficits have been found in the ability to detect specific mental activities such as behaviors, emotions and memories [34-36]. Looking at larger psychological phenomena, others have found difficulties reflecting on broader cognitive habits, such as reasoning style [37] as well as the ability to pull together autobiographical memories into a meaningful whole [38-40]. Importantly, the idea of a spectrum may also help clarify slightly different applications of the term metacognition with some authors concerned with the detection of errors [34] or reflections about specific beliefs, [29] and others, as will be explored in this review, with reflections about various integrations of thoughts and feelings into representations of self.

A rationale for why synthetic metacognition may be linked with psychosocial function in schizophrenia

In this review we focus on more synthetic forms of metacognition as a mediator of the effects of

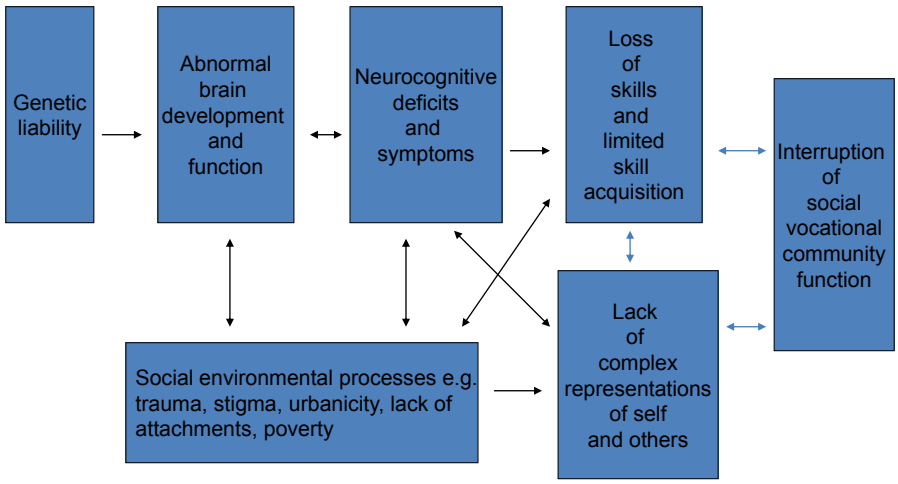

Figure 2. A path from gene to brain to dysfunctional behavior mediated by deficits in metacognitive capacity.

illness-related phenomena on function for three reasons, although we do not wish to suggest that more discrete forms of metacognitive activity would be less important or valid. First, synthetic metacognitive activity, in general, allows for meaning to be bestowed upon experience and activity. Synthetic forms of metacognition lend the potential for different meanings to be made, for instance, of conflicts at work or with others. For example, more pain is likely if one loses a job that is a cherished part of one's life vs. just a way to make money to support something else. More joy may come from one interaction vs. another because of the meaning one person has within one's life (e.g. a very promising junior colleague who might carry one's work forward vs. an acquaintance).

Second, the meaning bestowed on activity provides a context for reasoning about why one response makes more sense than another in the face of conflict or pain. As an illustration, if holding a certain job is an integral part of one's identity given longstanding career aspirations, one may find a reason why one should tolerate frustration with colleagues and supervisors rather than quit and find a new job. However, this would not make sense if the job is merely a way to make money. Similarly there would be far more reason to attempt to salvage a relationship with another person after a falling out if that relationship was seen as deeply connected with one's own life story. Thus, in the face of deficits in metacognitive capacity, a patient with schizophrenia might see less reason to persist or cope in the face of distressing developments if those events were without the personal meaning that is imbued by rich and complex understanding of oneself and others. Therefore, metacognitive deficits may result in there being no reason to undertake a course of action, regardless of whether or not a person knows how to undertake that action. In fact, without a reason to perform that action, there may be no search to figure out how to undertake that action.

Beyond the general connection between metacognition and responding to challenge, there is also a historic reason to think about schizophrenia as involving at its core a disturbance in the ability to synthesize material into complex wholes. As discussed also by Moskowitz [41], when Bleuler [1] proposed disturbances of associations as the core feature of schizophrenia, he did not refer merely to confusion due to the intrusion of unrelated ideas into thought but to the loss of a fundamental ability to form "associational synthesis" (p. 44), which reduced the understanding of oneself as an agent to a set of fragments which no longer served as a guide for goal directed activity. This lack of synthesis, as noted above, was assumed to have an organic origin but also to serve as a proximate cause of dysfunction as Bleuler [1] indeed detailed in accounts of schizophrenia patients no longer able to function socially or vocationally. Applied to the research presented above, it seems intuitively plausible that the feedback loop between brain development, social forces, symptoms, and neurocognition might well culminate in the loss of the ability to synthesize associations into larger images of oneself and others which, then, as exposed in Figure 2, may play into the larger feedback loop which results in dysfunction.

Assessing synthetic aspects of metacognition in schizophrenia using the Metacognition Assessment Scale Abbreviated (MAS-A)

To date one barrier to exploring the possibility that deficits in synthetic metacognitive activites play a role in the path from brain to behavior in schizophrenia concerns the issue of its measurement. Laboratory tasks have been developed to assess discrete aspects of metacognition, for instance to determine whether participants can detect they have made an error, made a certain action or recognize they do or do not know something [42-46]. These type of assessments, while useful for determining the effectiveness of error detection, are not useful for assessing more synthetic abilities which are a matter of complexity, flexibility, and adaptiveness.

In response, we have recently developed a method to assess synthetic metacognitive abilities from a spontaneously generated speech sample in which persons discuss their lives and personal understanding of the trials they have faced. That speech sample is obtained through a semi-structured interview called the Indiana Psychiatry Illness Interview (IPII). The IPII asks participants for their account or narrative of who they are as a person and also of their experience with psychiatric challenges. It thus allows for a life story to be told, in which there are opportunities for participants to spontaneously reveal how they think about themselves.

To quantify synthetic metacognitive capacity within IPII narratives, the Metacognition Assessment Scale – Abbreviated [MAS-A; 47] is used. The MAS-A contains four scales: "Self-reflectivity," or the comprehension of one's own mental states, "Understanding of others' minds," or the comprehension of other individuals' mental states, "Decentration," which is the ability to see the world as existing with others having independent motives, and "Mastery," which is the ability to use one's mental states to respond to social and psychological dilemmas. It is assumed that the metacognitive capacities assessed by each scale are semi-independent. Higher scores reflect abilities to perform increasingly complex synthetic acts within the domain captured by that scale. For instance, higher scores on Self-reflectivity would suggest a capacity to form more integrated representations of oneself, while higher scores on Mastery would suggest the capacity to use more complex forms of metacognition to respond to psychological and social challenges.

Acceptable levels of inter-rater reliability and internal consistency have been reported along with evidence of stability of MAS-A assessments across a 6-month interval [47-49]. Evidence that these procedures capture difficulties specific to psychosis includes findings that participants with schizophrenia have lower scores on all of the MAS-A subscales compared to others who also have significant medical and social adversity but not psychosis [50]. Concerning their validity, MAS-A scores have been linked with independent assessments of awareness of illness and cognitive insight [47,51].

Associations of metacognition with symptoms and neurocognition

To determine whether synthetic metacognitive activity may be linked with symptoms and

neurocognition, as portrayed in figure two, we have examined whether more severe symptom levels and neurocognitive deficits were linked with poorer MAS-A scores. In our first study, we correlated symptoms and neurocognitive functioning with MAS-A scores among men with schizophrenia in a non-acute phase of illness enrolled in rehabilitation [47]. The results revealed that a greater capacity for Self-reflectivity was linked with better verbal and visual memory, processing speed and premorbid intelligence. Greater capacities for Understanding the mind of the other, and Mastery were also related to better verbal memory. Concerning symptoms, higher levels of negative symptoms related to emotional withdrawal were linked with greater deficits in Self-reflectivity, Understanding the mind of the other, and Mastery.

Following up on the issue of neurocognitive capacity and metacognition, in a second sample of patients in a non-acute phase of illness we found that patients categorized as having achieved basic levels of Self-reflectivity on the MAS-A performed better on tests of executive function, working memory and social cognition [48]. In a third study [51] we turned to the issue specifically of executive function and correlated MAS-A scores with selected subtests of the Delis-Kaplan Executive Function System [D-KEFS; 52] including tests of inhibition, set shifting and mental flexibility. The sample consisted of 49 participants drawn from the two studies described above, who had completed the D-KEFS. Results revealed that the MAS-A Self-reflectivity subscale was more closely linked to D-KEFS subtests which tapped mental flexibility while D-KEFS subtests, which tested inhibition, were more closely linked to Decentration, Understanding of the other's mind, and Mastery, with correlations ranging from 0.30 to 0.47. We speculated that neurocognitive deficits contribute to deficits in synthetic metacognition. For instance, with a generalized reduction in the ability to flexibly think about daily events, persons may have difficulties forming an image of themselves as multifaceted. In contrast, without an ability to inhibit thoughts about events in the world, some may find it difficult to call to mind the perspectives of others and to detect a range of possible reactions others are having in rapidly evolving situations.

Exploring the links between metacognition and symptoms in a new sample, we compared MAS-A and symptom assessments over two time points six months apart in a group of 49 adults with schizophrenia in a stable phase of illness [49]. Correlational analyses found that the Total score of the MAS-A was correlated with both concurrent assessment of positive, negative, and disorganization symptoms as rated on the Positive and Negative Syndrome Scale [PANSS; 53]. In a multiple regression analysis, the MAS-A total score was found to predict prospective ratings of negative symptoms even after co-varying for baseline negative symptoms scores. We speculated that this may provide evidence as suggested in figure two of a bidirectional relationship between synthetic metacognition and at least some forms of symptoms. For instance, negative symptoms may dampen synthetic forms of metacognition while metacognitive deficits may be a risk factor for the emergence of negative symptoms. The findings linking negative symptoms and neurocognition with MAS-A scores in patients with prolonged psychosis have been replicated in separate Italian and Israeli samples [54,55]. McLeod *et al*. [56] have also reported metacognition rated using the MAS-A, on the basis of the Adult Attachment interview, prospectively predicted negative symptoms in 45 first episode patients in the United Kingdom above and beyond variance accounted for by premorbid function.

Metacognition and functional outcomes

Turning to the issue of synthetic metacognitive function and outcome, we first sought to see whether MAS-A scores could prospectively predict work function in a sample of 56 adults with schizophrenia enrolled in a vocational rehabilitation program. To accomplish this we divided participants who had completed a work placement program into three groups on the basis of Self-reflectivity score on the MAS-A which had been obtained prior to going to work: high (n=13), intermediate (n=21), and low Self-reflectivity (n=22) [57]. We then compared the biweekly rating of work performance completed during the six months following the initial MAS-A. Here we found that high Self-reflectivity group had significantly better work performance than either of the other two groups. That difference persisted after controlling for executive function as assessed prior to starting work. In a re-analysis of this data we also found that greater Self-reflectivity on the MAS-A was related to more accurate estimates of work ability as defined by the difference between self and supervisors assessments of work quality [58]. Links between metacognition and rehabilitation outcome were also found by Tas *et al*. [59] who reported that poorer metacognition predicted poorer response to cognitive remediation over time in a Turkish sample of 52 schizophrenia patients in a state of symptomatic remission.

In a second study we looked at the issue of social function, to assess whether we could show that metacognition, as assessed with the MAS-A, mediated the impact of neurocognitive deficits on rater assessment of the frequency of social interaction (quantity of social relationships) and on the basic building blocks which allow for social connection (quality of social relationships). Participants were 102 adults with a schizophrenia symptomatology in a post-acute phase of illness [60]. A Principal Components Analysis was used to reduce five different assessments of neurocognition, including executive function, verbal memory, visual memory, processing speed, and verbal ability into a single index and then structural equation modeling techniques were used to test the model that the capacity for metacognitive mastery mediates the impact of neurocognition upon the quality and quantity of social relationships after controlling for symptoms. Results revealed that an acceptable fit was observed between the model and data. Results persisted even after controlling for symptom and illness severity. In a third study, we followed this up by examining whether the links between metacognition and social function persisted over time [61]. Specifically, we examined the links between MAS-A scores and assessments of quality and quantity of social function conducted two times at an interval of five months apart for 72 of the original

102 participants. In a path analysis, acceptable levels of fit were found for a model in which Mastery predicted concurrent social function, and Mastery at baseline affected Mastery five months later, which similarly affected social function at that time. We interpreted the results of these studies along with the study of vocational function to provide evidence of the proximal link between synthetic metacognition and psychosocial function.

Support for these interpretations has been bolstered by another study of the relationship of metacognition with the perception of social interactions. Specifically, this study correlated MAS-A scores with assessments of social schema in a sample of 37 adults with schizophrenia in a non-acute phase of illness [62]. Social schema was measured using the Social Cognition Object Relations Scale [63], which assesses awareness of interpersonal relationships as a result of complex psychological forces, as well as the recognition that people in relationships have independent needs. Correlational analyses controlling for symptom severity and neurocognition revealed that higher levels of Mastery were linked to a greater understanding of the complex psychological forces that affect relationships and the existence of independent needs of individuals in relationships. The possible interpretation that the capacity for social connection is adversely affected by lower levels of metacognition has also been mirrored in another study, which found that greater levels of Mastery were related to self-reports of greater therapeutic alliance in cognitive behavior therapy [64].

In contrast to the studies noted above that used rater assessments of function, we also conducted studies examining the links of metacognition with self perception of illness and recovery. First, in a study of awareness of illness, we correlated assessment of awareness of symptoms, treatment needs and consequences of illness with assessments of social cognition, neurocognition and the MAS-A among 65 adults in a non-acute phase of illness [65]. After controlling for neurocognition, regressions revealed that Self-reflectivity was most closely linked to awareness of symptoms of psychosis while Mastery was most closely linked to awareness of treatment and consequence of illness. Mastery and social cognition were found to contribute independently to the prediction of awareness of consequences of illness. In terms of self-reported wellness as assessed by the Recovery Assessment Scale (RAS), with a new sample of 44 schizophrenia patients in a non-acute phase of illness, we found that those with greater levels of self-reflectivity felt less dominated by their symptoms while patients with more decentration were more able to reach out to others for help while feeling hopeful [66]. We have interpreted these findings as suggesting that synthetic metacognitive capacity may be linked to not only concrete social and vocational outcomes but also how persons appraise both illness and their achievement of health.

Finally, regarding functional competence or knowledge of how to perform tasks as indicated in Figure 2, a fourth study [67] examined the relationship between metacognition and performance on an assessment of functional skills, the UCSD Performance-Based Skills Assessment Battery (UPSA) [68]. Participants were 45 adults in a non-acute phase of schizophrenia that completed the IPII, assessments of neurocognition, symptoms and the UPSA. Correlational analyses revealed that Mastery was related to scores on the comprehension/ planning subscale of the UPSA even after controlling for symptoms and executive function. Results were interpreted as suggesting that decrements in Mastery may make some persons withdraw from more complex daily tasks such as organizing complex plans, resulting in the deficits in those functional abilities.

Discussion and limitations

In summary, decades of international research indicate that psychosocial dysfunction in schizophrenia is in part the result of the interaction of factors including symptoms and deficits in neurocognition. Less clear is how symptoms and neurocognitive deficits translate into the interruption of daily life. In this paper we sought to explore the possibility that synthetic metacognitive capacity may mediate the impact of these illness-related variables on outcome in schizophrenia. In other words, perhaps the path from brain to behavior in schizophrenia is influenced by the recursive process in which persons facing the illness reflect upon and interpret it in an ongoing inner experience. We have suggested a rationale for why this might be: metacognitive deficits seem likely linked in a bidirectional manner with symptoms and neurocognition and metacognitive deficits may leave persons less able to know why to pursue certain forms of action, resulting in the widespread interruption of goal directed activity. We have then detailed a method for assessing synthetic forms of metacognition and described findings from a number of studies that have linked synthetic metacognitive deficits with illness related phenomenon as well as multiple outcomes. These included at least one study that provided evidence that metacognitive deficits play a role as mediators.

Taken together the work reviewed provides evidence that deficits in synthetic metacognition are detectable in schizophrenia patients and that these deficits are related to outcome in a manner suggested in Figure 2. Deficits in synthetic metacognitive capacity appear to be distinct from a lack of functional skill and are not synonymous with defeatist beliefs linked with functional skill deficits [22]. Whereas a defeatist belief refers to a particular idea a person has about oneself, at issue here is the deeper ability to organize and integrate information into a larger whole, which would bestow meaning on daily activity. While this work is far from being able to offer proof that metacognitive deficits occupy the proposed role connecting brain to behavior, it appears to have offered a promising start, producing findings consistent with what one would expect if synthetic metacognition plays the role we have speculated.

As a whole this work can be seen to parallel recent converging anatomical and electrophysiological research on brain function which suggests that, as anticipated by Bleuler [1], the experience of "cognitive wholeness" is created through neural network synchronization of various association pathways [8,69]. Specifically, synchronization in the gamma and beta frequency bands has been linked to cognitive processes altered

in schizophrenia, such as perception and memory [9]. In the context of the subtitle of Crick's "Scientific search of the soul" [70] the research presented here may suggest that in schizophrenia, functional brain disintegration may unfold as psychological disintegration. Concerning treatment, this work is also consistent with other work suggesting that building metacognitive abilities through specific forms of learning during psychotherapy may also influence the brain based integrative processes as well [71,72].

There were important issues not discussed for reasons of space. This review has not dealt fully with the issue of etiology. It is unknown whether and if so how often, metacognitive deficits predate the illness and/or whether they can result from a number of different causal influences, including atrophy, loss of cognitive functioning, attachment style or exposure and response to trauma. It was also not discussed how the construct of metacognition converges and diverges with related constructs including social cognition, mentalization, mindfulness, and emotional intelligence. Future empirical and theoretical work is needed to tease apart how the phenomena studied here are related to these and other constructs. While we have explored capacity for forming and reflecting about integrated images of oneself, it should be noted that a range of work focused on autobiographical memory (e.g. [39-41]) should be considered in order to have a more complete view of the broader picture.

The research detailed here has also been most interested in links between illness related phenomenon and outcome and so the links between metacognition with social factors has been relatively neglected. We have also not fully explored the link between metacognitive disturbance and lack of functional competence. More nuanced theory is needed to develop a model of how skills deficits and metacognitive impairments impact and influence one another.

There are other limitations to the work presented here. Most of the studies discussed, though not all [54-56,59], were carried out in one laboratory. Participants also tended to be males in a later stage of illness enrolled in treatment. Replication is needed with broader samples including women, persons in an early stage of treatment and others who reject treatment. Long term longitudinal work is also needed to better understand the relationships suggested above as well as work comparing the metacognitive function of persons with schizophrenia to others with different forms of psychopathology. While we are not aware of other current methods for assessing synthetic forms of metacognition, much older work on psychotherapy and schizophrenia, such as that conducted by Carl Rogers [73], included quantitative scales for assessing personal distance from internal experience on the basis of clinical interviews. The constructs these scales assessed seem related to the central ideas of this review. Future work examining how the MAS-A may be related to these instruments could allow greater integration of the current findings into this much older and unduly neglected literature.

Clinical implications

Finally, with replication there may be important clinical implications to consider. If neurocognition and symptoms affect function through an interaction with metacognitive processes, treatment may need to do more than provide general support, education and skill remediation. Persons could be taught how to perform various functional activities but not develop any conception of why they should do so. For example, they may learn as we noted earlier how to offer compliments in a social setting but have no idea about why to and hence mere skill enhancement may not meet on its own the goal of truly enhancing patient self-direction. Suggested by the work reviewed in this paper, it may be that some with schizophrenia need assistance, possibly through a form of psychotherapy, to integrate information and form their own personal and adaptive accounts of themselves and their challenges, ultimately allowing them to take charge of their lives and find a way to achieve a fully acceptable quality of life. Recent advances in integrative forms of psychotherapy have shown promise for assisting persons to form more complex and integrated representations about themselves and others and then reflect about those representations, evolving them with time and using that knowledge to respond to psychological problems [74-78]. These forms of interventions might then allow persons to know why to persist in certain difficult life circumstances and to find the kinds of meaning in pain that allow human beings to live a rich life despite adversity.

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