Self-Monitoring in Schizophrenia

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Abstract: Many patients suffering from schizophrenia feel dispossessed from some of their actions or thoughts. This dispossession could result from impaired self-monitoring (SM), defined as the ability to monitor self-willed intentions and actions. SM has been widely studied during the past decades with very different paradigms; central error correction, feedback distortion, sense of effort, and motor imagery. The present article first reviews the methods used and results obtained in investigation of SM. Second, we address what we consider to be the critical questions that must be answered in order to fully understand the role of SM deficit in schizophrenia: 1) Is SM deficit only impaired in patients with specific symptoms? 2) Is SM deficit associated with other cognitive processes that are also impaired in patients with schizophrenia? 3) Can SM impairment be characterized as a trait or a state marker? Finally, we discuss the consequences of SM investigation on diagnostic evaluation and therapeutic orientations and we propose future research that we think is essential in order to clarify the role of SM in schizophrenia.

Keywords: Internal model, first-rank schneiderian symptoms, state/trait character.

INTRODUCTION

During the last two decades the concept of dysfunctional self-monitoring (SM) in schizophrenia has become well-known and very popular. SM is defined as the neurocognitive processes that allow an individual to monitor his or her own actions. Without these processes, not only could actions not be executed correctly but actions could also not be recognized as self-generated. Several teams have been working on SM in schizophrenia and substantial experimental data has been collected. In 1992 Christopher Frith published what is now a well regarded book on the topic; Cognitive Neuropsychology of Schizophrenia [1].

A dysfunction in SM has been used to explain First-Rank symptoms (FRS) that are among the most distressing sensations encountered by patients suffering from schizophrenia. FRS are characterized by patients feeling that actions and personal states are no longer under their own control [2]. The main FRS are auditory hallucinations, thought insertion, thought broadcasting, delusions of influence, and all the feelings that another is controlling the patient's thoughts, actions or emotions (Table **1**).

Initially, Feinberg [3] proposed that FRS may be explained by a deficit in the internal monitoring of action: patients who suffer from FRS would be unable to correctly monitor their own actions and thoughts. This hypothesis was re-defined by Frith [1], who proposed instead the term of self-monitoring, i.e. the ability to monitor one's own intentions, thoughts, and actions. Action monitoring involves internal forward modeling that allows the central nervous system to represent the predicted sensory consequences of a movement before its completion [4, 5]. Such a prediction is derived from a copy of the motor command, the so-called "efference copy" [6], that can be compared with reafferent signals (i.e. sensory signals arising as a consequence of the movement) (Fig. **1**). SM deficit in patients with schizophrenia specifically concerns the predicted sensory consequences. Additionally, this deficit is formulated as a lack of awareness of these predicted consequences [7]. Although, accurate representations of predicted states (derived form internal model) are available and used by the motor system, these representations are not available to awareness. Failure to form a representation of the predicted consequences of an action would result in an impaired ability to distinguish between one's own and another's actions, resulting in patients

Table 1. First Rank Symptoms of Schizophrenia, Initially Described by K. Schneider (1959)

Current Names of Symptoms	Descriptions
Acoustico-verbal hallucinations	Voices heard arguing or commenting on patient's actions.
Audible thoughts	Patient's own thoughts heard by him/herself.
Thought broadcasting	Patient's thought are passively diffused to other people.
Thought insertion	Other people intrude their thoughts upon the patient.
Thought withdrawal	Other people actively take patient's thoughts in his/her mind.
Made affect and feelings	Experience of influences playing on patient's sensations.
Somatic passivity, delusions of influence, alien control	Experience of influences playing on patient's actions.
Delusional perception	Patient's experience of a peculiar, intense, convincing experience not shared by other people.

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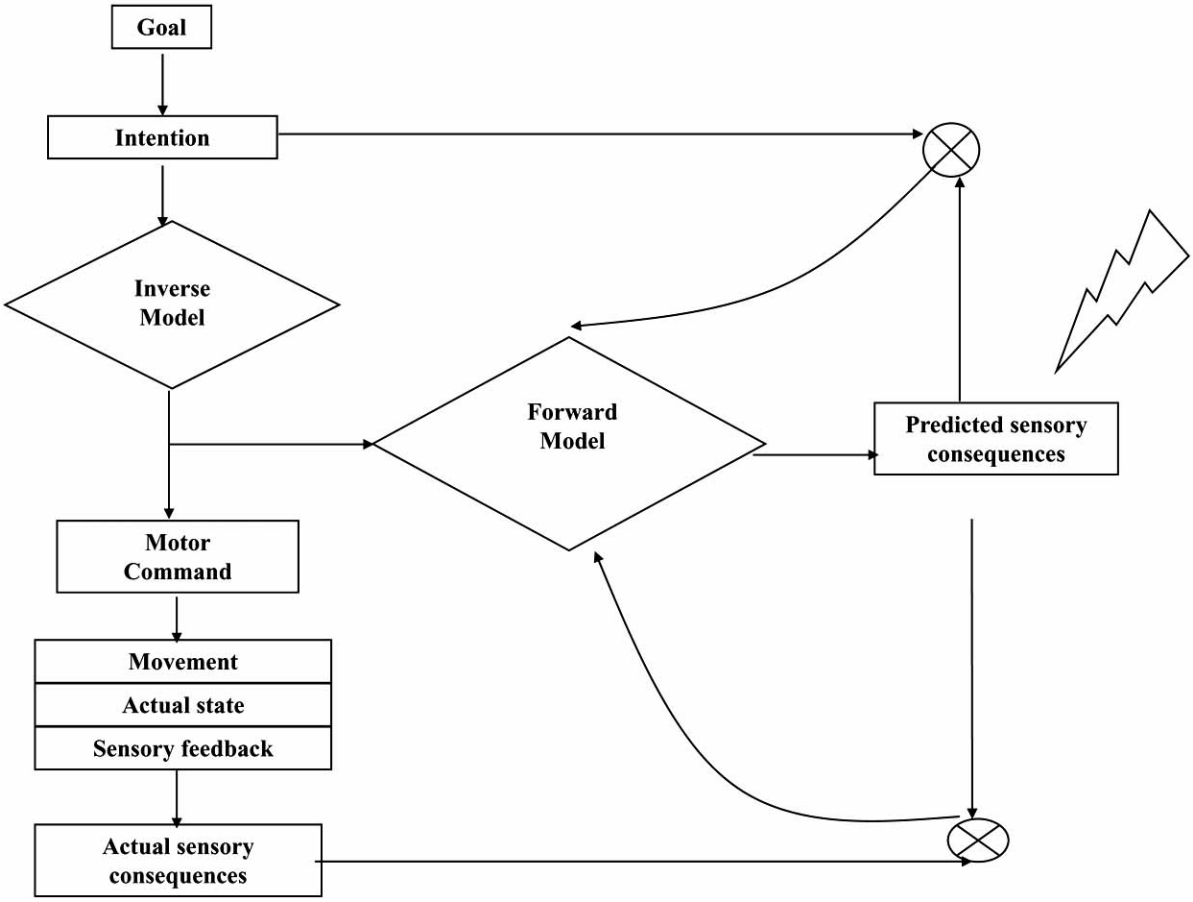

Fig. (1). The central nervous system represents the predicted sensory consequences of a movement before its completion. Such a prediction is derived from a copy of the motor command, the so-called "efference copy", that can be compared with reafferent signals (i.e. sensory signals arising as a consequence of the movement). Failure to form a representation of the predicted consequences of an action would result in an impaired ability to distinguish between one's own and another's actions, resulting in patients experiencing their own actions as being controlled by other agents (permission for use: Frith *et al*., abnormalities in the awareness and control of action, Fig. **1**, p 1773, 2000, Royal Society).

experiencing their own actions as being controlled by other agents [7] (Fig. **1**).

Different experimental paradigms have been used to evaluate SM processes. They all test the ability of patients to rely on the representations provided by the internal model. The main results of experiments based upon these studies are summarized in Table **2** and discussed in further detail below. To ensure that the studies reported in this review only deal with SM, we excluded all source-monitoring studies, defined as the ability to remember the source of the information that was obtained, because they involve both SM and memory processes, which makes it difficult to disentangle SM impairments from memory impairments.

The following parts involve first an exhaustive review of both the methods used and results obtained in SM investigation. Second, the critical questions that must be answered in order to fully understand the role of SM deficit in schizophrenia are then addressed. 1) Is SM deficit only impaired in patients with specific symptoms? 2) Is SM deficit associated with other cognitive processes that are also impaired in patients with schizophrenia? 3) Can SM impairment be characterized as a trait or a state marker?

PARADIGMS

Central Error Correction

Preventing subjects from receiving the external sensory feedback, which would normally result from their actions, forces subjects to rely on the central monitoring of these actions. If the detection of an erroneous response to a stimulus cannot be achieved in the absence of external feedback, but can be achieved when such feedback is present, it would provide evidence for defective SM. Malenka and colleagues [8] asked subjects to perform a step-function tracking task designed to prevent the use of visual signals in correcting movement errors. The subjects held a joystick and in half of the conditions the polarity control was reversed, forcing subjects to move the joystick in the direction opposite to that of the step function. The aim of this reversed polarity was to induce a large number of errors (moving the joystick in the wrong direction) that then had to be corrected. Compared with control subjects and alcoholic patients, patients with schizophrenia were the only subjects who were impaired in recognizing and correcting errors in the absence of visual feedback. Frith and Done [9] conducted a similar experiment

Table 2. Investigation of Self-Monitoring in Schizophrenia

Study	Paradigm	Subjects	Results	Relationship with Schizophrenic Symptoms (When Examined)
Malenka *et al.* (1982)	Central error correction	14 SZ, 21 NC, 12 ALC	SZ performances < NC and ALC	
Frith and Done (1989)	Central error correction	14 SZ (10 FRS and 4 NFRS, 9 BP, 6 NC	SZ NFRS performances = BP and NC	Patients with alien control were less able to make error correction in the absence of visual feedback.
Mlakar *et al.* (1994)	Central error correction	55 SC (25 FRS and 30 NFRS), 10 NC	FRS performances < NFRS and NC	Only patients with FRS showed poorer performances in conditions relying on central action monitoring.
Kopp and Rist (1994)	Central error correction	27 SZ, 27 NC, 18 ALC	SZ performances = NC and ALC (in terms of error corrections or increased correction latencies)	Neither the severity of positive symptoms nor the severity of though disorder correlated significantly with the error correction performances of SC. Error frequencies of neither subgroup were reduced compared to SC without experiences of bizarre delusions.
Delevoye-Turrell *et al.* (2003)	Central error correction	16 SZ, 16 NC	SZ performances = NC (in terms of predictive control of movement)	
Spears (1963)	Feedback distorsion (verbal feedback)	24 SZ, 21 NC, 23 OPS	SZ performances > NC SZ performances > OPS	
Sutton *et al.* (1964)	Feedback distorsion (verbal feedback)	28 SZ, 30 NC	SZ performances = females NC SZ performances > males NC	
Goldberg *et al.* (1997)	Feedback distorsion (verbal feedback)	15 SZ (10 FRS and 5 NFRS), 19NC	SZ performances < NC	SC with FRS were significantly slower, exhibiting more dysfluency than any other groups.
Cahill *et al.* (1996)	Feedback distorsion (verbal feedback)	21 SZ		Performances lower in delusional patients, but not in hallucinated patients.
Johns and McGuire (1999)	Feedback distorsion (verbal feedback)	18 SZ, 20 NC	SZ performances < NC	Performances lower in hallucinating subjects (more misattributions of their own distorted voice to another speaker) than in other patients.
Johns *et al.* (2001)	Feedback distorsion (verbal feedback)	18 SZ, 20 NC	SZ performances < NC	Performances lower in hallucinating subjects (more misattributions of their own distorted voice to another speaker) than in other patients.
Blakemore *et al.* (2000)	Feedback distorsion (tactile feedback)	38 SZ (15 FRS and 23 NFRS), 15 NC	FRS performances < NFRS and NC	Patients with auditory hallucinations and/or passivity symptoms did not distinguish between self and externally produced tactile stimulation.
Daprati *et al.* (1997)	Feedback distorsion (visual feedback)	30 SZ (13 FRS and 17 NFRS), 30 NC	SZ performances < NC	Hallucinating patients (n=13) and influenced patients (n=7) were more impaired than other patients in discriminating their own hand from an alien hand.
Franck *et al.* (2001)	Feedback distorsion (visual feedback)	24 SZ (6 influenced and 18 non influenced), 29 NC	SZ performances < NC	Influenced patients were more impaired than other patients in discriminating their own movement from a modified movement.
Fourneret *et al.* (2001)	Feedback distorsion (visual feedback)	19 SZ (10 FRS and 9 NFRS), 19 NC	FRS performances = NFRS and NC	Awareness of predicted consequences was unimpaired in patients with FRS.
Lafargue *et al.* (2006)	Sense of effort	17 SZ (6 FRS and 11 NFRS), 17 NC	FRS performances < NFRS and NC (in terms of intended effort)	All patients performed as normal subjects in processing achieved efforts (i.e. to produce an effort with one hand and reproduce it with the other one).
Danckert *et al.* (2002)	Motor imagery	10 SZ, 10 NC	SZ performances < NC	Results were not associated with the symptom profile.
Maruff *et al.* (2003)	Motor imagery	12 FRS, 12 NFRS	FRS performances < NFRS (in terms of speed-accuracy trade-off between target width and movement duration)	Imagined movements in patients with passivity phenomena were not constrained by the same biomechanical and environmental factors as real movements.
de Vignemont *et al.* (2006)	Motor imagery	13 SZ, 13 NC	SZ performances = NC (in terms of RT and accuracy of mental rotation)	Hallucinating patients made significantly more errors than non-hallucinating.
Turken *et al.* (2003)	Central error correction	8 SZ, 8 NC	SZ performances < NC	
Stirling *et al.* (1998)	Central error correction Source monitoring	35 SZ, 24 NC	SZ performances < NC	Impairments in self-monitoring were associated with the experience of symptoms of alien control.

SZ: schizophrenia group; OPS: other psychiatric group, NC: normal control group; ALC: alcoholic group; BP: bipolar disorder group; FRS: first-rank symptoms group; NFRS: non first-rank symptoms group.

but they also considered schizophrenic symptoms. Comparing patients with schizophrenia who experienced alien control of their thoughts and actions with patients without such experiences and with patients with affective psychosis, they showed that only those patients with delusions of alien control were significantly less likely to make error corrections in the absence of visual feedback. Furthermore, in the presence of visual feedback, the performance of these patients was not significantly different from patients without delusion of alien control or from patients with affective psychosis. Mlakar and colleagues [10] further extended this paradigm by examining patients with delusions and/or hallucinations and patients without these symptoms, and by introducing different conditions in which different degrees of central action monitoring were required. Although all patients with schizophrenia performed poorly in this task, only the group of patients with delusions and/or hallucinations had increased error rates as reliance on central monitoring increased. Kopp and Rist [11] used a paradigm similar to the one used by Malenka and colleagues [8] while also controlling for potential confounds such as the possibility that proprioceptive feedback or memory factors might be the information sources guiding error correction instead of central monitoring of action. The results showed that schizophrenic patients, the alcoholic patients, and the healthy subjects all relied on central monitoring of action to the same degree. Error correction rates were not lower in schizophrenic patients nor did they show a prolongation of the time needed to initiate error corrections compared with both healthy subjects and alcoholic patients. Furthermore, neither the severity of positive symptoms nor the severity of thought disorder correlated significantly with the error correction performances of schizophrenic patients. Finally, compared with schizophrenic patients without experiences of bizarre delusions, neither subgroup showed reduced frequency of errors.

The capacity to form representations of predictive consequences of actions was also tested with grip force tasks that required increasingly greater predictive control [12]. Three tasks (a lift task, a hit task, and a resist task) were used to test the predictive mechanisms required for the scaling, timing, and sequencing of voluntary motor activity. Subjects either lifted objects of various mass and texture, or used a manipulandum to either hit or resist impacts produced by a collision with a pendulum. Patients with schizophrenia were not impaired in the predictive scaling and timing of motor actions, showing that patients can form correct representations of predictions.

Feedback Distortion

Another way to study SM is to experimentally distort the subject's sensory feedback and to evaluate the consequences of this distortion on his or her subsequent performance of an action. Under conditions of distorted sensory feedback patients should behave differently from other subjects as a result of a deficit in their ability to predict the sensory consequences of their actions. This paradigm has been tested in auditory, visual, and tactile modalities.

Verbal Feedback

The first feedback distortion studies evaluated the consequences of delaying the auditory feedback (DAF paradigm) on the motor counterpart (i.e. on the changes in the parameters of the subjects' speech) while they were talking and listening to their own speech with a delay between speaking and hearing their own voice. Spears [13] showed that schizophrenic patients were neither slower nor less disturbed than patients with depression or healthy subjects. However, Sutton and colleagues [14] showed slower speech and fewer words correctly enunciated in schizophrenic patients compared to controls. Goldberg and colleagues [15] used a DAF paradigm to test for dysfluency in schizophrenic patients and normal controls. In normal subjects, delaying the auditory feedback of one's own speech resulted in subjects slowing their speech and becoming dysfluent. The authors reasoned that if there is a SM deficit in schizophrenia, then patients with auditory hallucinations and/or delusions of control should be abnormally unperturbed by the delay. The results, however, failed to support the hypothesis of a SM deficit in patients with FRS. Indeed, patients with auditory hallucinations and/or delusions of control were significantly slower, exhibiting greater dysfluency than any other group.

Another interesting aspect of sensory feedback distortion task concerns the subject's judgment of the source of the action. Cahill and colleagues [16] tested the hypothesis that under conditions of distorted external feedback of self-generated speech, patients would be deficient in their ability to identify the source of these speech sounds (i.e. themselves or someone else). In their task, the immediate auditory feedback of a patient's voice was distorted in pitch and the patient was then asked to identify the source of the voice. The magnitude of pitch distortion affected the patients' attributions; higher levels of pitch distortion were more likely to be associated with attributions to an 'other'. External attribution of the source was also found in patients with verbal hallucinations compared with controls [17, 18]. These studies also found that the content of the sentences/words spoken by the subjects had a substantial effect on the attribution; hallucinators were more likely to make errors when the words were derogatory, rather than neutral or complimentary. In conclusion, the tendency of patients to misattribute to the 'other' voices that had undergone greater pitch distortion supports the hypothesis of a deficit in SM.

Tactile Feedback

Blakemore and colleagues [19] evaluated whether patients can perceptually differentiate between tactile stimuli that are self-produced and those that are externally produced. Subjects were asked to rate a tactile sensation on the palm of their left hand that was produced either by movement of their right hand or by the experimenter. Compared to both a control group and a patient group without hallucinations or passivity experiences, only patients with these symptoms showed no difference in their perception of self-produced and externally produced tactile stimuli, suggesting that patients with auditory hallucinations and passivity experiences have an abnormal awareness of the predicted sensory consequences of their own movements.

Visual Feedback

In our laboratory we examined the capacity to visually distinguish between one's own and another person's movements. We designed a task that required subjects to execute hand movements while visualizing either their own hand or the experimenter's hand doing the same movements [20].

Hallucinating and deluded patients were less able than other schizophrenic patients to discriminate their own hand from the alien hand. These results were replicated using a visual distortion paradigm in which different amounts of spatial and temporal distortion of the visual feedback of a subject's movements were presented [21]. When compared with both controls and other patients, only those patients with passivity experiences were worse at recognizing that their movements were deviated. These results were not, however, replicated by Fourneret and colleagues [22] who used a motor task in which subjects had to make a correction of their hand trajectory toward a target. Although the visuomotor adaptation part of the task was successfully performed by all patients, only those with FRS were able to explicitly report the correction showing that awareness of predicted consequences was unimpaired in these patients.

Sense of Effort

Another approach to the investigation of SM concerns the sense of effort, defined as the phenomenological parameter when muscular force has to be estimated. The sense of effort is relevant for studying SM because it is mediated by efference copy, it requires first the processing of effort at a conceptual level and then its transformation into motor output. Patients with FRS performed worse compared with those without FRS in processing the intended effort revealing a problem in accessing awareness of the central signal of effort before this signal interacts with proprioceptive signals [23]. These results, therefore, support the hypothesis of a deficit in schizophrenia at the level of the awareness of the predicted consequences of an action.

Motor Imagery

A dysfunction of internal SM should give rise to impairment in generating motor imagery; the ability to generate internal images of intended but not executed motor movements. Subjects were tested in a visually guided pointing task that required making real or imagined back and forth movements towards a target box that could be of different widths [24]. In normal subjects, real and imagined movements are constrained by the speed-accuracy trade-off given by Fitts' law (as the target width becomes smaller, the duration of real movements increases). For both schizophrenic patients and normal controls, real movements were constrained by the speed-accuracy trade-off given by Fitts' law. Patients with passivity phenomena, however, were the only group who showed unconstrained duration of imagined movements, suggesting a specific impairment in motor imagery in these patients. Similar findings were also reported with a visually guided pointing task [25] and a mental rotation task [26].

DISCUSSION

To date, a review of the studies listed above shows converging evidence, although incomplete, for a SM deficit in schizophrenia. SM has been studied using a range of different paradigms and in heterogeneous patient groups (with or without FRS; FRS being hallucinations or delusions) (Table **2**). What first emerges is that it remains unclear whether all the paradigms that have been used actually explore the same aspect of SM. Indeed, the central error correction and sense of effort paradigms both use tasks that require an accurate representation of the action that has been undertaken and of its consequences. Whereas the feedback distortion paradigm uses tasks that require a good comparison between central and sensitive information, and motor imagery requires an intelligent body representation that includes biomechanical constraints. In order to evaluate whether the same aspect of SM is involved in the deficits observed in the studies reported here we need to compare the performances of the same patients in tasks from different paradigms.

Another question that emerges from this review concerns whether SM in schizophrenia is associated with distinct symptoms and/or with other cognitive processes that are also impaired in patients, and whether this SM deficit can be characterized as a trait or a state marker. The following paragraphs examine these issues by clarifying the questions that need to be answered in order to fully understand the role of SM in schizophrenia.

Self-Monitoring Deficit and Symptoms

SM was first put forward in a cognitive neuropsychological approach in which a SM deficit was considered as a possible causative explanation for FRS. Addressing the specific association between FRS and SM deficit requires considering two questions. First, is the whole FRS spectrum associated with SM deficit? Second, is SM deficit only impaired in patients with FRS symptoms but not in schizophrenic patients without FRS?

Asking whether a SM deficit can explain the whole FRS spectrum requires consideration of the theoretical debate about whether symptoms such as verbal hallucinations (VH), thought insertion, or thought spread can be explained by an 'action' explanation [27, 28]. Feinberg [3] proposed that the experience of conscious thought may involve a mechanism analogous to internal feedback. Some delusional experiences, such as hallucinations, might be produced by disordered internal feedback or impaired efference copy. Frith [29] also proposed that thoughts can be considered as internalized forms of actions or speech, and by consequence, the same mechanisms should apply to both FRS involving overt-movements and FRS not involving overt-movements such as VH, thought insertion, and thought control. The debate concerning VH has been attenuated since empirical research showed that when patients hallucinated, subvocalisations occurred [30, 31] with these subvocalisations arising from the activation of language mechanisms [31], supporting the claim that inner speech can be conceptualized as an action. Concerning thought insertion and thought control, recent findings by Ford & Mathalon [32] can be interpreted in support of the assumption that considers thoughts are internalized forms of actions. These authors showed electrophysiological evidence of an efference copy dysfunction in schizophrenic patients not only while they were talking but also while they were thinking (inner speech). Normal SM in verbal actions is associated with a dampening of the auditory cortex, which is reflected by the N1 component of the event related potential. In control subjects, the N1 component reflects the dampening of auditory cortex responsivity not only during talking but also during inner speech. This dampening was not observed, however, in any condition in schizophrenic patients. These results showed that the corollary discharge is abnormal in patients with schizophrenia for both

talking and inner speech, supporting the claim that cerebral processes involved in both acting and thinking may be equally dysfunctional in patients with schizophrenia. These results show that not only symptoms related to a patient's actions, but also those related to his or her thoughts might be explained by the same neurocognitive deficit.

Addressing the second question of whether a SM deficit is only associated with FRS raises two problems. The first issue is clinical, and concerns the difficulty in separating hallucinations and delusions, as these symptoms frequently co-occur and some delusional convictions are developed from patients' hallucinated hearings. Second, most studies do not permit an assessment of the deficits in patients with delusions of control compared with patients with hallucinations, as they typically group together patients that manifest either delusions, hallucinations, or both these symptoms [10, 17, 19, 20, 23, 33-36]. In spite of these difficulties, several studies have found SM impairment specifically associated with delusions of influence (passivity delusions, alien control) [9, 21, 24, 37, 38] or hallucinations [16, 18, 26]. Impaired performance on SM tasks has, however, also been observed in patients without FRS [39-43].

Upon initial reflection, these studies do not permit a clear extrapolation of a SM deficit as a possible underlying cause of all FRS. Patients with distinct FRS and with no symptom overlap need to be separated and then tested with SM paradigms. On the basis of the available data, one possible interpretation is that SM deficit takes place on a continuum; with no impairment in this ability in healthy subjects, a small impairment in patients without FRS, and a larger impairment in patients with FRS.

Self-Monitoring: Trait or State Marker?

A clear understanding of the role of a SM deficit in schizophrenia requires considering whether this impairment can be characterised as a state or a trait marker. A state marker is only observed in the presence of symptoms and its evolution correlates with the severity of the symptoms. A trait marker, however, defines impairments (either at a cognitive or a neurological level) that are present independently of the manifestation of the symptoms. To be considered as a trait marker, SM deficit needs to fulfill certain criteria. First, SM deficit should be observed in patients who are prone to experience FRS or other symptoms independently of the manifestation of these symptoms. Second, SM impairment should also be present, although less marked, in unaffected relatives of patients. Finally, SM deficit should not be associated with any other neurological or psychiatric disease. In most of the studies reviewed here, patients exhibited a SM deficit even though they did not manifest symptoms, favoring the hypothesis that SM deficit is a trait marker. Further evidence that it is a trait marker requires that a SM deficit is not present in other pathological groups. One study directly addressed the state/trait character of SM deficit by comparing the performance of schizophrenic patients with auditory hallucinations, patients with a history of (but not current) auditory hallucinations, patients with neither current nor previous hallucinations, and patients with affective psychosis and current hallucinations [44]. Subjects were tested with a DAF paradigm; they were requested to read single adjectives aloud while the source of the pitch of the on-line auditory verbal feedback was manipulated. Subjects were then asked to immediately identify the source of the speech they heard. Misidentification of the source as 'other' during distorted feedback of the speech was most likely in patients who had auditory hallucination. However, patients with a history of hallucinations did not differ from controls, and patients with hallucinations in the context of an affective disorder made more errors but not errors of misattribution. Difficulty in a SM task seems to be more related to the acute psychotic state rather than a predisposition to hallucinations as it was present in patients with affective psychosis as well as those with schizophrenia. This state marker interpretation is further supported by the finding of a misattribution of self-generated speech in healthy individuals with high levels of psychotic-like experiences, suggesting that the same cognitive impairment may underlie psychotic phenomena in healthy individuals and in patients with psychotic disorders, consistent with a continuum model of psychosis [45].

Markers of schizophrenia have also been identified at the cerebral level. Electrophysiological studies suggest that a negative component of the event-related potential, the error-related negativity (ERN), is related to SM deficit [46]. The ERN is associated with incorrect responses and may reflect a comparison process between representations of the appropriate response and the response actually made (see [47] for a review). Several research groups have observed diminished ERN amplitude in patients with schizophrenia during performance of a SM task. This decreased ERN amplitude cannot be explained by performance demands since ERN generation appears to be abnormal in schizophrenia patients even under conditions demonstrated to maximize ERN [48]. Furthermore, ERN amplitude is modulated by clinical state in schizophrenia, further supporting the idea that decreased ERN amplitude is a potentially useful marker for schizophrenia [49]. This interpretation, however, remains to be confirmed, since a recent study revealed decreased ERN amplitude in normal controls after administration of classic and atypical antipsychotics (haloperidol, olanzapine) and an antidepressant (paroxetine) [50]. SM deficit was also associated with the N1 component previously described. In control subjects, appropriate SM in verbal actions such as talking, is associated with a decreased N1 reflecting dampening of auditory cortex. In patients with schizophrenia, a dysfunctional SM was associated with abnormal N1 amplitude (no correlative decreased amplitude), suggesting no dampening of this cortex [51]. Finally, Farrer and colleagues [52] showed that SM deficit was associated with abnormal cerebral activations in the form of deactivation in the right inferior lobule in patients with FRS.

Although, the behavioral results seem to favor the state character of a SM deficit, only hallucinations were taken into account and only one kind of paradigm has been used to date. Other paradigms and other symptoms need to be tested the same way before rejecting the trait character of SM deficit. With respect to cerebral markers, the N1 component is only associated with auditory stimulation and can therefore not be considered as a general neurophysiologic marker of a SM deficit which also manifests in other sensory modalities. Furthermore, disturbances of this component in action monitoring tasks are associated with other neuropsychiatric states such as obsessive-compulsive behaviour [53], autism [54], and depressive disorder [55]. Finally, the investigations have

so far had limited success in establishing a relationship between these neurobiological indicators of SM abnormality and symptoms reports in patients [51].

On the basis of the available evidence it is not yet possible to conclude anything regarding the state/trait character of these results. Furthermore, our assumption regarding the association between one behavioural marker (i.e. SM) and one cerebral marker (i.e. ERN) may be overly simplistic. A SM deficit may be associated with several brain markers that represent different components of SM and/or different schizophrenic symptoms.

Self-Monitoring and Other Cognitive Deficits

Addressing the association between SM deficit and other cognitive impairments in schizophrenia requires considering two questions. First, does SM deficit specifically account for FRS or can other cognitive deficits also explain impaired performances of patients in SM tasks? Second, is SM impairment sufficient to fully explain FRS or does a combination of SM deficit with other cognitive impairments provide a more complete account of these symptoms?

The heterogeneity of FRS brings into question the assumption that a single dysfunctional process can explain all these symptoms. It could be argued that a more general process, that involves SM, needs to be identified. Since FRS were first considered by Schneider [2] as the consequences of a loss of boundaries between the self and others, self-recognition impairments that not only concern actions and thoughts but also some body aspects could better explain the diversity of symptoms. However, although several studies of action-monitoring and/or action recognition found a dissociation between patients with FRS and patients without FRS, only patients with FRS were impaired in distinguishing between their own actions and another's actions. In contrast, Farrer and Franck [56] showed that both groups of patients were equally impaired when asked to distinguish between their own hands and the experimenter's hand in a self-recognition task, indicating that impaired recognition of one's own body is not specifically associated with FRS [56].

Several processes involved in executive control are severely impaired in schizophrenia, and the notion of SM is close to the one of executive control with which it may share some cognitive components. Indeed, most of the tasks used with the central error correction paradigm to assess SM also tap into the same range of cognitive functions tapped by executive function tasks. Although most studies of SM in schizophrenia exclude possible explanations such as inconsistent motivation, deficits in other cognitive functions could still explain the impaired performance of schizophrenic patients with FRS. Stirling and colleagues [37] have used different SM tasks and several neuropsychological tests for evaluating IQ, recognition memory, executive functions, and attention functions. They found that patients performed worse in the SM tasks, and that their performances progressively decreased as demands on SM increased. The performance of patients on these tasks was independent, or at least in addition to, deficits in attentional, general cognitive, or recognition memory functions. Furthermore, there was a strong relationship between SM deficit and presence of alien control. These results were replicated by Turken and colleagues [57] who compared the performances of different schizophrenic patient groups on a SM task based on the work of Frith & Done [9] but modified their task in order to simultaneously assess a number of abilities considered to engage high-level executive control mechanisms: (1) conflict resolution, (2) set switching, and (3) preparatory attention. Subjects had to respond with the press of a left or a right hand button to one of four possible visual target stimuli with conditions in which the feedback was immediate or delayed (these later conditions examined SM). Stimulus-response mapping of each trial was indicated by a precue presented 1250 or 2100 msec before the target. Initial practice established a bias in favor of one of the stimulus-response mappings. Performances on trials with weaker mappings reflected an ability to suppress response bias. Comparison of performance on trials when the stimulus-response mapping switched relative to the preceding trial with those in which stimulus-response mapping remained the same assessed mental flexibility. Performance improvement with longer precue intervals assessed preparatory attention. SM impairment was observed in the patient group in the absence of significant attentional control deficits. These results argue for a dissociable dysfunction selectively affecting the mechanisms responsible for processing internal representations of one's own cognitive processes and acts.

Although these studies show a near total independence of executive control deficit relative to SM deficit in patients with schizophrenia, they do not take into account symptoms and by consequence do not allow investigation of a specific SM deficit in FRS patients with no executive control deficit contamination. Furthermore, the study by Turken and colleagues [57] only included 8 patients, too few to permit extrapolation of these results to the whole population.

A second question concerns the exclusivity of SM to explain FRS. These symptoms are characterized by their dynamic, chronic state. In most of the studies reviewed here, however, patients exhibited a SM deficit even though they did not manifest symptoms. The persistence of this deficit in the absence of a manifestation of symptoms raises the question of whether a SM deficit provides a unique contribution to the occurrence of FRS. Furthermore, it seems that this deficit may not be sufficient to explain how a misattribution of one's own actions and thoughts can happen, and it is possible that other processes may be involved in the occurrence of these symptoms. Some authors have pointed out that other cognitive deficits may work in concert with SM. For example, Allen and colleagues [36] postulated that an externalizing response bias might also participate in FRS manifestation. In their study, patients with hallucinations and delusions and patients without these symptoms were required to make self/nonself judgments while they passively listened to recordings of words spoken in their own or another person's voice. The recorded speech was manipulated by altering the pitch and the experiment was conducted a few days after the recordings were made. Patients with hallucinations and delusions were more likely than both schizophrenic patients without these symptoms and controls to misidentify their own speech as alien. The authors explained this misidentification by an externalizing bias in the processing of sensory material, with patients tending to answer alien (response bias as misattribution to other) when they fail to recognize the voice. Others factors might also intervene in FRS occurrence. For example, the emotional content of sen-

tences/words spoken by the subjects has a substantial effect on the attribution, since in DAF tasks hallucinators were more likely to make errors when the words were derogatory, rather than neutral or complimentary [17, 18]. Contextual factors could also contribute to FRS occurrence, with self-attribution disorders resulting also from a deficit in the processing of the relevant contextual information necessary to disambiguate problematic social and communicative situations [58]. FRS would thus not only result from a unique SM deficit but could also involve other impairments at psychological, cognitive, and/or emotional levels.

Implication on Diagnoses and Therapeutic

Getting around the problem of the heterogeneity of schizophrenia will require redefining the limits and coherence of this concept. For the last few decades, researches in the genetic field have been unable to identify genes involved in the expression of schizophrenia. This failure in the identification of schizophrenia genotype could be explained by the fact that schizophrenia as a whole is not a coherent phenotype, but a disorder in which multiple mechanisms explain multiple symptoms. Identifying relevant cognitive processes (for example SM deficit) will greatly assist in defining coherent groups of patients. Hence, it is necessary to make strong links between symptoms or syndromes and dysfunctional cognitive processes in order to better understand the physiopathology of schizophrenia specifically and of psychosis in general. From this perspective, it is imperative that different dimensions of schizophrenia (hallucinated, delusional, manic, negative, disorganized, etc.) are better investigated. A classification based on neurocognitive dysfunction will result in the need to revisit the psychosis spectrum and regroup patients with different disease diagnoses who, however, present similar symptoms. If these criteria are fulfilled, then it will become possible to revaluate patients' categorization.

An alteration of SM can severely affect life of patients with schizophrenia. Since impaired SM is involved in the production of FRS, it can lead to grave danger. Some patients will commit suicide in response to their voices or under the control of an external force within the framework of delusions of influence. Other patients will be aggressive towards other people for the same reasons. More generally, it has been hypothesized that self-monitoring would contribute to social cognition abilities in allowing people to control their own expressive behaviour and their self-presentation. In this perspective, a poor self-monitoring could contribute to altered social cognition and deficit in social skills observed in schizophrenia [59]. It is therefore essential to develop SM remediation in order to improve patients' social interactions and daily life. Indeed, the initial results of a recent attempt to remediate abnormal self-monitoring in schizophrenia were favourable [60]. Increasing the future efficacy of self-monitoring therapies will, however, require a clearer understanding of the role of SM deficit in schizophrenia.

CONCLUSION

Although a SM deficit in schizophrenia has been well demonstrated using a wide range of paradigms, we do not yet have answers to the questions that are essential in order to fully understand the role of SM in schizophrenia. First, there is no clear evidence of a SM deficit as a possible underlying cause of all FRS. Subgroups of patients with schizophrenia or other mental diseases, who have distinct symptoms and no symptom overlap between groups need to be separated and then tested with SM paradigms. Second, the characterisation of SM deficit as a state or a trait marker and its association with cerebral markers and symptoms remains unsatisfactory. Longitudinal studies with test-retest experiments, to evaluate the replication of the impairment over a number of different states, will also be required. Third, the relationship between SM and the other processes involved in motor planning and executive control also remains unclear. Further studies that explore this field are necessary, as well as investigations into the role of emotional and contextual data as triggering factors.

Longitudinal studies are needed in order to evaluate the evolution of SM deficits across patients at different clinical stages and across different patients with the same symptoms. The evaluation of SM in people vulnerable to schizophrenia (siblings or subjects suffering from schizotypic personality disorder) could also provide information useful for assessing the SM disorder hypothesis of schizophrenia. In conclusion, future studies will have to consider the three questions mentioned above in order to contribute to a clearer understanding of the role of SM in schizophrenia. This research field has not yet been fully explored and needs systematic, objective investigation.

ACKNOWLEDGEMENTS

We thank very much Karen Reilly for her english corrections and Valérian Chambon for his comments. This work was supported by la fondation pour la recherche médicale (FRM).

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Received: December 19, 2006 Revised: June 5, 2007 Accepted: June 13, 2007