



## Abnormal agency experiences in schizophrenia patients: Examining the role of psychotic symptoms and familial risk



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### ABSTRACT

Experiencing self-agency over one's own action outcomes is essential for social functioning. Recent research revealed that patients with schizophrenia do not use implicitly available information about their action-outcomes (i.e., prime-based agency inference) to arrive at self-agency experiences. Here, we examined whether this is related to symptoms and/or familial risk to develop the disease. Fifty-four patients, 54 controls, and 19 unaffected (and unrelated) siblings performed an agency inference task, in which experienced agency was measured over action-outcomes that matched or mismatched outcome-primes that were presented before action performance. The Positive and Negative Syndrome Scale (PANSS) and Comprehensive Assessment of Symptoms and History (CASH) were administered to assess psychopathology. Impairments in prime-based inferences did not differ between patients with symptoms of over- and underattribution. However, patients with agency underattribution symptoms reported significantly lower overall self-agency experiences. Siblings displayed stronger prime-based agency inferences than patients, but weaker prime-based inferences than healthy controls. However, these differences were not statistically significant. Findings suggest that impairments in prime-based agency inferences may be a trait characteristic of schizophrenia. Moreover, this study may stimulate further research on the familial basis and the clinical relevance of impairments in implicit agency inferences.

### 1. Introduction

Patients with schizophrenia often feel that they are not causing their own thoughts and actions (e.g., delusions of control or thought insertion). Such experiences may be explained by impairments in self-agency, i.e., the experience that we cause our own actions and the consequences of those actions. For example, when you press the button of an ice machine, you automatically feel that it was you who made the ice cubes fall out. Also, in more complex (social) situations this experience is crucial. For example, when you make a joke it matters whether you feel that you made the people around you laugh, or whether you think they are laughing for some other reason. By focusing on impairments in these self-agency processes and genetic vulnerability to these impairments, this study aims to contribute to a better understanding of the origin of psychotic symptoms.

Two models have been described to explain the underlying mechanisms of the experience of self-agency: motor prediction and cognitive inference. During action performance, the motor system constantly makes predictions about consequences of actions. The motor prediction

model assumes that outcomes are perceived as self-produced when a prediction matches the sensory feedback (e.g., the sound of ice cubes falling out). In case of a mismatch between the prediction and the actual outcome, people are likely to attribute agency to someone or something else (Frith et al., 2000; Wolpert and Flanagan, 2001). Several studies on schizophrenia, using various experimental paradigms based on the motor prediction model (e.g., corollary discharge or temporal binding), demonstrated impairments in agency attribution in different stages of the disease. (Daprati et al., 1997; Franck et al., 2001; Hauser et al., 2011; Hur et al., 2014; Johns et al., 2001; Maeda et al., 2012, 2013; Schimansky et al., 2010; Synofzik et al., 2010).

In situations with multiple possible outcomes and multiple possible agents, motor predictions regarding the actual outcome can no longer reliably guide feelings of self-agency and consequently, retrospective cognitive inferences of agency become important (Aarts et al., 2005; van der Weiden et al., 2013a; Wegner, 2002). The cognitive inference model assumes that people *infer* self-agency based on knowledge and beliefs regarding the effects (e.g., laughter) of their actions (e.g., making a joke) before they perform them, and regarding the influence

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of other factors (e.g., someone who is imitating you in a funny way). Similar to the matching/mismatching process within motor prediction, when an event matches (rather than mismatches) one's prior beliefs and expectations, one is likely to infer self-agency. It has been suggested that schizophrenia patients may rely more on this cognitive route towards agency due to deficits in motor prediction processes (Synofzik et al., 2013; Voss et al., 2010).

Studies on cognitive agency inferences distinguish two different routes: a goal-based (explicit) and a prime-based (implicit) route (Aarts et al., 2005; van der Weiden et al., 2013b). Goal-based agency inferences are involved in planned behavior, whereas prime-based agency inferences are involved in behavior that is instigated by environmental cues. Previous studies from our group revealed that schizophrenia patients show specific deficits in this second route, showing that they are unable to use implicitly available cues in the environment when making agency inferences (Renes et al., 2013, 2015). However, it is not known whether these impairments in making prime-based inferences relate to specific psychotic symptoms or familial risk. By combining two independent samples (Renes et al., 2013, 2015) we now have the statistical power to perform sub-group analyses in order to address these questions. In the current paper, we investigate two possible implications of schizophrenia patients' impairments in making prime-based inferences.

First, impairments in prime-based inferences may specifically explain symptoms of over- and underattribution of self-agency. One could argue that over-attributing self-agency is reflected in, for example, grandiose delusions or delusions of reference, i.e., patients feel they are capable of causing events that are actually outside of their control (Synofzik et al., 2013). To our knowledge, such symptoms of overattribution have not been studied in relation to self-agency processing yet. In contrast, delusions of control (i.e., patients experience no control over their thoughts and actions) or auditory hallucinations, (i.e., patients perceive inner speech as originating from an external source (Balconi, 2010)) may imply underattribution of self-agency (or attribution to the outside world). These symptoms are also referred to as first-rank symptoms (Carpenter et al., 1973; Heering et al., 2013) or passivity symptoms and have previously been associated with impaired agency processing (Daprati et al., 1997; Franck et al., 2001; Synofzik et al., 2010; Waters and Badcock, 2010). However, to our knowledge separate clusters consisting exclusively of symptoms of over-attribution or underattribution of agency have not been studied in experimental settings. Therefore, we examine whether prime-based agency inferences, as well as overall level of experienced self-agency are related to these clusters of symptoms. Specifically, we expect patients with symptoms of under-attribution to experience less self-agency overall, and that this may be related to a decreased sensitivity to implicit outcome-primes. Conversely, we expect patients with symptoms of over-attribution to experience more self-agency overall, and that this may be related to an increased sensitivity to implicit outcome-primes.

Second, we aim to investigate whether the disturbance in prime-based agency inferences is related to familial risk to develop schizophrenia and could serve as a vulnerability marker for the disease. Schizophrenia is highly heritable (Cardno et al., 1999; Kendler et al., 1995), and there is ample evidence that neural or cognitive features of the illness are related to the familial or genetic risk to develop the illness. Previously, it has been suggested that impairments in self-monitoring may be a vulnerability marker for psychosis (Versmissen et al., 2007). Indeed, studies in unaffected siblings of patients have shown that they perform at an intermediate level between patients and healthy controls on self-processing tasks, such as action monitoring (Hommes et al., 2012) and verbal source monitoring tasks (Brunelin et al., 2007). Here, we examine impairments in prime-based inferences as a possible vulnerability marker for schizophrenia by exploring whether unaffected siblings of schizophrenia patients perform at an intermediate level on a prime-based agency inference task, which would suggest that the familial risk to develop the illness is reflected in abnormal self-processing.

## 2. Methods

### 2.1. Subjects

A total of 54 patients with a DSM-IV diagnosis of schizophrenia, 19 unaffected (unrelated) siblings of patients with a non-affective psychotic disorder (of which 16 patients were diagnosed with schizophrenia, 1 with schizophreniform disorder, and 2 with psychotic disorder NOS), and 54 control subjects participated in the study. Patients and controls were included from two independent samples that have been described previously (sample 1 (Renes et al., 2013) and sample 2 (Renes et al., 2015)). In these samples, similar inclusion and exclusion criteria were used.

Exclusion criteria were an IQ below 70, drug or alcohol abuse in the past six months, a history of head trauma, neurological illness or endocrine dysfunction. Controls and siblings had no history of psychiatric illness. Additionally, controls had no first-degree relatives with a psychotic illness and did not use chronic medication. Within the patient group, all but three participants were receiving antipsychotic medication at time of testing.

Patients were recruited from the psychiatry department of the University Medical Center Utrecht (UMCU) and Amsterdam Medical Centre (AMC). Siblings were recruited from the Genetic Risk and Outcome of Psychosis project (Korver et al., 2012) and healthy controls were recruited via advertisements. After explaining the study procedures, participants gave written consent. They were financially compensated for study participation. The study was approved by the Human Ethics Committee of the UMCU.

### 2.2. Procedures and measures

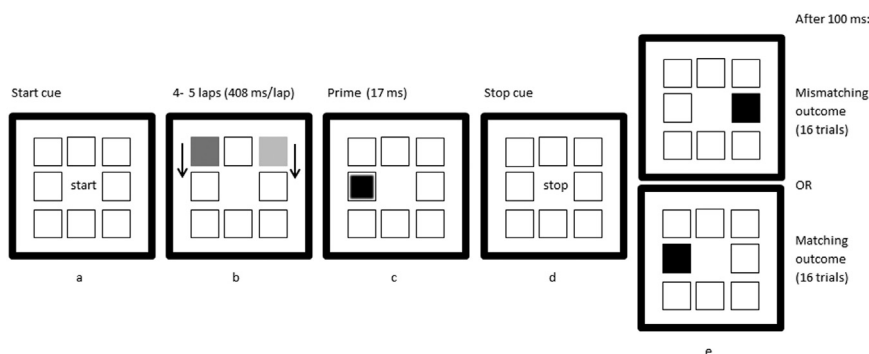
#### 2.2.1. Diagnosis and symptom levels

Lifetime and current presence and severity of symptoms and diagnosis were measured by the Comprehensive Assessment of Symptoms and History (CASH; Andreasen et al., 1992). This instrument assesses diagnoses in the affective and psychotic spectrum. Additionally, current symptom levels were assessed with the Positive and Negative Syndrome Scale (PANSS; Kay et al., 1987).

#### 2.2.2. Prime-based agency inference task

Participants learned that the study was designed to examine experiences of personal causation and how they come and go. For this purpose, we used the Wheel of Fortune task (Aarts et al., 2005; Renes et al., 2013). In this computer task, when participants pressed the s-key, two squares started moving in opposite direction along a rectangular path, consisting of eight white squares (see Fig. 1). A dark grey square, representing the participant's square, rapidly (50 ms. per tile) moved in a counter-clockwise direction. Simultaneously, a light grey square, representing the computer's square, moved in the opposite direction at the same speed. When after 4 to 5 laps "stop" appeared in the center of the screen, the moving squares became invisible to the participant and they had to press the enter-key immediately, thereby stopping the movement of their own square. This action turned one of the eight white tiles black, which represented the final position of either their own square or the computer's. After each stop, participants indicated the extent to which they felt they had caused their square to stop at that particular position [9-point scale: not at all (1)–strongly (9)].

Importantly, in reality the program always determined the outcome position and thus, actual stops occurred independently of participants' key press. In each trial, one location was subtly primed just before participants pressed the stop-key and saw the outcome location. These primes lasted for 17 ms., equal to one frame on a 60 Hz. monitor. They either matched (i.e., same location) or mismatched (i.e., three or four positions away) the outcome and were used to activate a representation of the outcome during ongoing action, without requiring a predetermined intention. The difference between agency experiences on matching and mismatching trials (i.e., matching effect) indicates patients' susceptibility to implicit agency cues. A stronger matching



**Fig. 1.** Wheel of fortune task. a) Start cue appears and participants press start-key. b) Participant's (dark grey) and computer's square (light grey) move in opposite directions. c) Just before the stop cue appears, outcome information is primed. d) At the presentation of the stop cue, squares disappear and continue to move invisibly. Participants press stop-key. e) A predetermined outcome is presented that either matches or mismatches the prime.

effect represents more use of subtly activated outcome representations when inferring self-agency.

The task comprised 32 trials that were divided in 2 blocks of 16 trials. In each block, the black square was used as an implicit prime twice on each of the eight tiles of the path, once as a match and once as a mismatch. The trials were randomly presented within each block and there was a short break (30 s) between the blocks. Participants practiced to assure they understood the task.

### 2.3. Statistical analyses

Patients, unaffected siblings, and controls were compared on age, gender, and level of own and parental education using ANOVA and a  $\chi^2$  test. For methods concerning the comparison between the two patient samples, See Supplemental Materials.

#### 2.3.1. Aim 1: Self-agency and psychotic symptoms

**2.3.1.1. Prime-based inferences in relation to symptom severity.** In patients, the relationship between matching effect (i.e., the difference in agency scores on matching and mismatching trials) and current symptom severity (PANSS positive, negative, general, and total score) was assessed using Spearman Rank correlations. Our sample of 54 patients should allow us to detect a significant moderate effect. We used Bonferroni correction for significance tests:  $\alpha=0.05/4$  symptom scores=0.0125.

**2.3.1.2. Prime-based inferences in relation to symptoms of over- and underattribution.** To examine the symptoms of underattribution (UA) or overattribution (OA) in relation to agency, patients were divided in groups based on present-state symptom levels as assessed by the CASH. OA included experiences in which agency is inaccurately attributed to the self: delusions of guilt, grandiose delusions, religious delusions, and delusions of reference. UA included symptoms in which agency is inaccurately attributed to an external source: delusions of control, thought broadcasting, thought insertion, thought withdrawal, and auditory (verbal) hallucinations. By defining the clusters on a mechanistic and not on a content level (e.g., paranoid/non-paranoid), we aim to understand the mechanisms underlying self-processing.

Participants were classified as UA/OA when a score of at least 1 (questionable) was present on at least one of the UA/OA symptoms. Four groups were created based on these symptoms: 1) patients with OA only (OA+;  $n=7$ ), 2) those with UA only (UA+;  $n=12$ ), 3) patients with OA and UA (UAOA+;  $n=18$ ), and 4) those without OA and UA (UAOA-;  $n=17$ ). One outlier from the UAOA+ group was excluded for further analyses, as the overall agency score exceeded more than three standard deviations from the group mean.

Groups were tested for differences in age, gender, years of education, and years of illness using independent samples t-tests and a  $\chi^2$  test. To test group differences regarding the ability to make implicit agency inferences,

a repeated-measures ANOVA with Group (OA+, UA+, UAOA+, and UAOA-) as between-subjects variable and Matching (matching and mismatching outcomes) as within-subjects variable was used. Assumptions for parametric testing were not violated (all  $p$ 's > 0.52).

**2.3.1.3. Overall self-agency experiences in relation to symptoms of over- and underattribution.** Overall agency was calculated by taking the mean of all trials, irrespective of the priming manipulation. As the assumption of normality was violated for this variable, a Mann-Whitney U and Kruskal-Wallis test were used to compare overall agency scores between patients and healthy controls and between the four groups regarding over- and underattribution (UA+, OA+, UAOA-, and UAOA+), respectively. Post-hoc, Mann-Whitney U tests were used (Bonferroni correction:  $\alpha=0.05/6=0.008$ ). In these analyses, OA+ and UA+ were of particular interest in examining our hypotheses.

#### 2.3.2. Aim 2: Prime-based inferences in patients, siblings, and healthy controls

Group differences in agency experiences were assessed using a repeated-measures ANOVA with Group (patients, siblings, and healthy controls) as between-subjects variable and Matching (matching and mismatching outcomes) as within-subjects variable. Assumptions for parametric testing were not violated (all  $p$ 's > 0.19). Simple effects of Matching (i.e., matching effect) within each group were also assessed by using a repeated-measures ANOVA. MANOVA was used to perform follow-up analyses in order to further assess group differences. As our sibling sample was relatively small, these analyses are exploratory.

## 3. Results

### 3.1. Demographics and clinical characteristics

No differences were found between the two patients samples regarding task performance, See Supplemental materials. Demographic and clinical characteristics of patients, unrelated and unaffected siblings, and controls are displayed in Table 1. Significantly more female participants were included in the sibling group as compared with healthy controls and patients and group differences in age were marginally significant. As expected, patients had fewer years of education than healthy controls and siblings, whereas parental years of education did not differ between groups.

#### 3.2. Aim 1: Self-agency and psychotic symptoms

##### 3.2.1. Prime-based inferences in relation to symptom severity

In patients, no significant correlations were found between matching effect and scores on the PANSS total, positive, negative, and general scale (respectively:  $r=-0.01$ ,  $p=0.93$ ;  $r=0.12$ ,  $p=0.40$ ;  $r=0.08$ ,  $p=0.59$ ;  $r=-0.003$ ,  $p=0.98$ ).

**Table 1**  
Demographic and clinical characteristics of patients (PT), unaffected (and unrelated) siblings (SIB), and healthy control subjects (HC). Means (SD) are displayed.

	Patients (n=54)	Siblings (n=19)	Controls (n=54)	Group differences
Gender (n(male/female))	48/6	12/7	47/7	$\chi^2(2)=7.56$ , $p=0.02^*$ , $\phi=0.24$ HC & PT > SIB
Age	30.8 (7.24)	34.7 (5.67)	30.1 (7.53)	$F(2,124)=3.00$ , $p=0.053$ , $\eta_p^2=0.05$
Illness duration (years)	10.54 (7.52) <sup>a</sup>	N/A	N/A	
Years of education	13.04 (1.98)	14.32 (1.95)	14.04 (2.34)	$F(2,124)=4.03$ , $p=0.02^*$ , $\eta_p^2=0.06$ HC & SIB > PT
Parental years of education	13.98 (3.18) <sup>b</sup>	13.74 (2.62)	14.04 (2.62) <sup>c</sup>	$F(2,124)=0.08$ , $p=0.93$ , $\eta_p^2=0.001$
Antipsychotic medication		N/A	N/A	
Typical	2			
Atypical	49			
None	3			
PANSS		N/A	N/A	
Positive	12.06 (4.20)			
Negative	13.80 (6.00)			
General	26.15 (7.92)			
Total	52.00 (15.25)			

\* Significant at  $p < 0.05$ .

<sup>a</sup>  $n=52$ .

<sup>b</sup>  $n=47$ .

<sup>c</sup>  $n=51$ .

**3.2.2. Prime-based inferences in relation to symptoms of over- and underattribution**

The four groups based on symptoms of over- and underattribution differed significantly on age and years of illness, but not on gender and years of education. However, as age and years of illness were not related to mean agency scores ( $r_s=-0.10$ ,  $p=0.48$ , and  $r_s=0.07$ ,  $p=0.64$ ), it was not accounted for in further analyses.

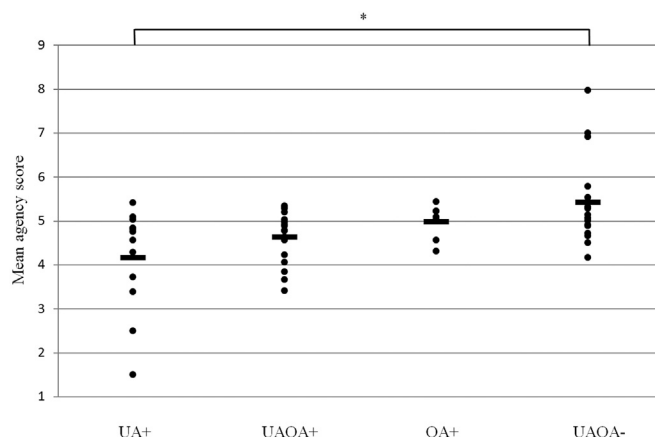
A repeated measures analysis showed no main effect of Matching,  $F(1,50)=0.56$ ,  $p=0.46$ ,  $\eta_p^2=0.01$ , indicating no difference between agency experiences on matching and mismatching trials (i.e., matching effect). Also, no group differences between UA+, OA+, UAOA+, and UAOA- were found on matching effect,  $F(3,50)=0.11$ ,  $p=0.96$ ,  $\eta_p^2=0.006$ .

**3.2.3. Overall self-agency experiences in relation to symptoms of over- and underattribution**

Overall agency ratings did not differ between patients and healthy controls,  $U=1382$ ,  $z=-0.305$ ,  $p=0.76$ ,  $r=-0.03$ . The mean agency scores within patients follow-up the expected pattern, with the lowest agency scores for UA+, followed by UAOA+, and finally OA+ and UAOA-. A Kruskal Wallis test was used to compare overall agency scores in the four subgroups based on symptoms of over- and underattribution. The four groups differed significantly,  $\chi^2(3)=10.59$ ,  $p=0.01$ . Fig. 2 and Table 2 display distributions and means of agency scores in patients. Post-hoc Mann-Whitney U tests showed that after Bonferroni correction only UA+ scored significantly lower than UAOA-. In addition, a trend level difference was found between UA+ and OA+, reflecting lower scores in UA+, and between UAOA- and UAOA+, reflecting lower scores in UAOA+.

**3.3. Aim 2: Prime-based inferences in patients, siblings, and healthy controls**

Differences in gender, age, and years of education were (marginally) significant between the three groups, but were not related to matching effect. Therefore, they are not controlled for in the repeated measures ANOVA. Fig. 3 and Table 3 show a visual display and summary statistics regarding the group differences in self-agency experiences and matching



**Fig. 2.** Level of experienced agency in groups based on symptoms of over- and underattribution. UA+=underattribution only, UAOA+=under- and overattribution, OA +=overattribution only, UAOA-=no under- or overattribution. \*significant at  $p < 0.05$  after bonferroni correction.

effects. Overall, a main effect of Matching was found, showing that agency experiences were higher in matching trials, compared with mismatching trials. There was no main effect of Group, implying that there are no differences in overall agency scores between patients, siblings and controls, irrespective of the task manipulation.

Most importantly, the analysis yielded a significant Matching by Group interaction, implicating group differences in the matching effect. Simple effects analyses, depicted in Table 3, showed that healthy controls experienced more agency on matching trials compared with mismatching trials, while it reached trend level significance in siblings and was not present in patients. The means and effect sizes of the matching effect within groups indicated that siblings ( $M=0.87$ ,  $\eta_p^2=0.17$ ) scored in between patients ( $M=0.280$ ,  $\eta_p^2=0.02$ ) and healthy controls ( $M=1.38$ ,  $\eta_p^2=0.24$ ). However, MANOVA's showed that siblings did not differ significantly from either patients or healthy controls.

In line with our previous findings on the two independent patient samples separately (Renes et al., 2013, 2015), a MANOVA revealed a significant difference between patients and healthy controls, reflecting a significant matching effect in controls, which was absent in patients.

**4. Discussion**

In the present study we found that patients with symptoms of underattribution of agency (i.e., delusions of control, thought broadcasting, thought insertion, thought withdrawal, or auditory (verbal) hallucinations) experienced less agency than patients without any symptoms of over- or underattribution. Second, the disturbances in prime-based agency infer-

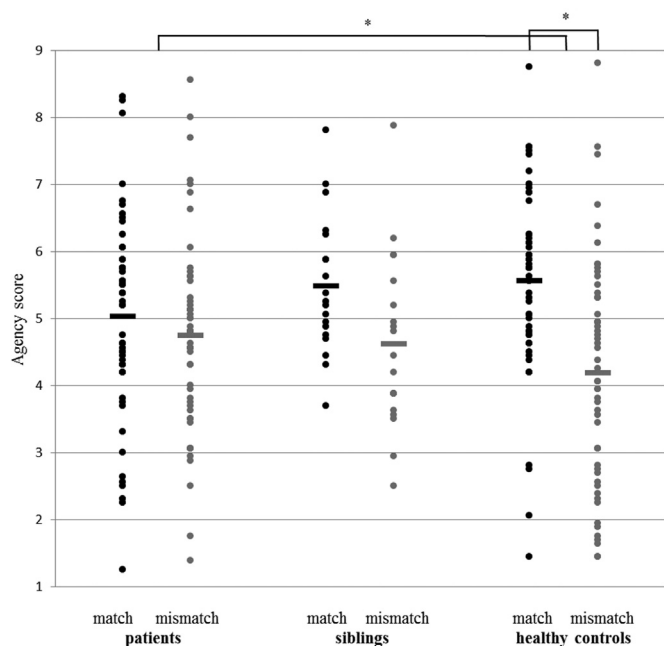
**Table 2**  
Means and post-hoc Mann-Whitney U test results regarding level of experienced agency in groups based on symptoms of over- and underattribution.

Groups	Means	Group differences		
		UA+	UAOA+	OA+
UA+ (n=12)	4.16 (1.18)	-	-	-
UAOA+ (n=17)	4.63 (1.01)	$U=80.00$ , $p=0.33$ , $r=-0.22$	-	-
OA+ (n=7)	4.98 (0.40)	$U=19.50$ , $p=0.01^*$ , $r=-0.44$	$U=38.00$ , $p=0.17$ , $r=-0.22$	-
UAOA- (n=17)	5.42 (1.00)	$U=41.00$ , $p=0.007^*$ , $r=-0.50$	$U=76.00$ , $p=0.02^*$ , $r=-0.33$	$U=47.50$ , $p=0.45$ , $r=-0.16$

\* Significant at  $p < 0.05$ .

\*\* Significant after Bonferroni correction; OA+=overattribution only, UAOA+=over- and underattribution UA+=underattribution only, UAOA-=no over- or underattribution.





**Fig. 3.** Self-agency scores in patients, siblings, and healthy controls. Mean scores on match and mismatch trials are depicted. \* Significant at  $p < 0.05$ .

ences did not differ between patients with either symptoms of over- or underattribution and were not related to severity of psychotic symptoms. Finally, the matching effect in siblings was intermediate between that of patients and healthy controls, although this did not reach significance.

**4.1. Symptoms of over- and underattribution: the role of prime-based inferences**

In the current study we differentiated between patients who did or did not experience symptoms of overattribution or underattribution. The level of experienced agency did not differ between patients who only showed symptoms of overattribution and the other three groups. This may indicate that these patients did not necessarily overattribute in the presence of other agents (e.g., a computer) and in the absence of an explicit goal to reach a specific outcome (van der Weiden et al., 2015). However, more research on this subgroup of patients is needed, as a sampling issue (i.e., the low number of patients with overattribution symptoms only) might have influenced the results.

**Table 3**  
Statistical analyses of self-agency scores and matching effects in patients, unaffected (and unrelated) siblings and healthy controls.

Mean agency scores				Main analysis: Repeated measures ANOVA ( $df=2,124$ )					
	Patients( $n=54$ )	Siblings( $n=19$ )	Controls( $n=54$ )		$F$	Sig.	$\eta_p^2$		
Match	5.03 (1.51)	5.49 (1.03)	5.58 (1.38)	Group	0.24	0.79	0.00		
Mismatch	4.75 (1.45)	4.61 (1.30)	4.19 (1.67)	Matching	14.30	< 0.001*	0.10		
Matching effect	0.28 (2.04)	0.87 (1.98)	1.38 (2.49)	Matching x Group	3.30	0.04†	0.05		
Simple effects analyses: Repeated measures ANOVA									
	Patients ( $df=1,53$ )			Siblings ( $df=1,18$ )			Healthy controls ( $df=1,53$ )		
	$F$	Sig.	$\eta_p^2$	$F$	Sig.	$\eta_p^2$	$F$	Sig.	$\eta_p^2$
Matching	1.02	0.32	0.02	3.68	0.07	0.17	16.61	< .001*	0.24
Follow-up analyses: MANOVA ( $df =2,124$ )									
	Healthy controls-Patients			Healthy controls-Siblings			Patients-Siblings		
	$t$	Sig.	Cohen's $d_s$	$t$	Sig.	Cohen's $d_s$	$t$	Sig.	Cohen's $d_s$
Matching x Group	2.56	0.01*	0.49	0.86	0.39	0.28	0.99	0.32	0.26

\* Significant at  $p < 0.05$  level.

Interestingly, confirming our hypothesis, patients with exclusively symptoms of underattribution experienced less self-agency relative to patients without symptoms of over- and underattribution and, at trend level, to patients with symptoms of overattribution only. These results suggest that patients who experience symptoms of underattribution indeed tend to under-attribute agency in situations where other possible agents are present (e.g., a computer) and where behavior is not goal-directed. Other studies that investigated impairments in agency attribution in patients with symptoms of underattribution reported contradictory results regarding the direction of the attribution bias in their experimental paradigms (Franck et al., 2001; Hauser et al., 2011; Johns et al., 2001; Schimansky et al., 2010). A possible explanation for the inconsistent findings may be the way of defining symptoms of underattribution. In our study 31% of the patients showed both overattribution and underattribution symptoms at the time of testing. Thus, in previous studies, patients selected on symptoms of underattribution may have also had symptoms of overattribution. This makes it difficult to interpret the deficits in agency processing in terms of specificity for symptoms related to agency attribution. In this context it is important to note that although symptoms of over- and underattribution can easily be distinguished on theoretical grounds, clinically it is a challenge to study these symptom clusters separately.

In our paradigm, we used a subtle manipulation (matching vs. mismatching outcome primes) to examine agency experiences that do not rely on motor prediction. This enabled us to assess the relationship between (abnormalities in) implicit agency inferences and specific symptoms, independent of the previously reported impairments in motor prediction (Daprati et al., 1997; Franck et al., 2001; Hauser et al., 2011; Hur et al., 2014; Johns et al., 2001; Maeda et al., 2012, 2013; Schimansky et al., 2010; Synofzik et al., 2010). Our findings indicate that the presence or absence of symptoms of over- and underattribution did not influence patients' ability to use implicit cues when guiding feelings of self-agency. Thus, although symptoms of underattribution were related to agency-underattribution in our task, these symptoms cannot be explained by patients' impairments in using primes when inferring self-agency.

**4.2. Symptoms of over- and underattribution: alternative explanations**

Why then, do these patients with symptoms of underattribution experience less agency? It is unlikely that goal-based agency inferences are related to underattribution symptoms, as we previously showed that patients with schizophrenia do not have problems in using goal-based inferences to guide agency experiences (Renes et al., 2013, 2015). However, as mentioned in the introduction, people do not only consider action-outcome information when inferring self-agency, but also take into account

other (e.g., agent-related) information when answering the question ‘Am I the most likely cause of this event?’ (see also: [Wegner and Wheatley, 1999](#)). Here, we address a few possible explanations for decreased agency experiences in patients with symptoms of underattribution.

#### 4.2.1. Other explanations from a cognitive level

Agency attribution is affected by several factors, for example by the presence of background beliefs ([Desantis et al., 2011](#); [Synofzik et al., 2013](#)). In fact, a patient’s strongly held belief about having no influence on causality might overrule implicit agency cues, such as the primes used in our experiment.

Also, increased attention to other (delusional) agents might decrease self-agency experiences and induce or maintain symptoms of underattribution. The amount of attention towards other agents or towards the self might fluctuate from time to time, which might also explain why symptoms of over- and underattribution can coexist. So far, the role of agent-related information in agency inferences has received little empirical attention. However, given patients’ impairments in social context processing, this may be a promising direction for future research ([Penn et al., 2002](#); [White et al., 2016](#)).

#### 4.2.2. Motor prediction

Another way to explain symptoms of over- and underattribution is by impairments in motor prediction. Specifically, when motor predictions are less precise, a mismatching outcome may be perceived as matching one’s prediction, resulting in overattribution of self-agency (see [van der Weiden et al., 2015](#)). Yet, when one is completely surprised by the outcome of one’s action because it was not predicted at all, this may result in underattribution (cf., [Blakemore et al., 2000](#)). Although there is evidence for a relation between impairments in motor prediction and symptoms of underattribution ([Franck et al., 2001](#); [Hauser et al., 2011](#); [Johns et al., 2001](#)), these results are inconsistent and it is still unclear when and how motor prediction impairments lead to underattribution (or overattribution). Future studies may shed light on this issue by distinguishing between patients with exclusively symptoms of over- or underattribution. Also, to explore whether impairments in motor-prediction or in cognitive inferences are more crucial, both processes should be tested in a single experiment.

#### 4.2.3. Ownership

The sense of agency is closely related to body ownership ([Klaver and Dijkerman, 2016](#); [Ma and Hommel, 2015](#)). In fact, a sense of body ownership is necessary to experience agency, but ownership does not necessarily require a sense of agency ([Tsakiris et al., 2007](#)). Importantly, more severe passivity symptoms (a concept that largely overlaps with our under-attribution symptoms) were associated with impairments in assessing body schema ([Graham et al., 2014](#)). Therefore, a possible cause of decreased agency experiences in patients with symptoms of underattribution might be mediated by problems in experiencing body ownership.

#### 4.3. Impairments in prime-based inferences as a trait characteristic

In our study, the absence of a relationship between the ability to make prime-based agency inferences on one hand and symptom type and severity on the other hand suggests that a reduced ability to arrive at prime-based agency inferences may be a trait characteristic of schizophrenia. In line with this notion, agency disturbances have also been found in populations at high clinical risk for psychosis ([Hauser et al., 2011](#)), indicating that these disturbances are present well before the first florid psychotic episode. Whether abnormal self-agency processing in high-risk populations is specific for those who will later develop schizophrenia has not been investigated. If the inability to make prime-based agency inferences is a trait factor, studies on self-agency processing in young high-risk individuals appear relevant to develop causal models of psychotic symptoms. So far, there is suggestive evidence that phenomenological experiences of self-disturbances might indeed be a specific predictor for

psychosis later in life ([Nelson et al., 2012](#); [Parnas et al., 2014](#)).

#### 4.4. Impairments in prime-based agency inferences as a vulnerability marker?

As a second aim of the current study, we assessed whether problems in agency inferences have a familial (and thus possibly genetic) component. Previous studies showed that unaffected siblings of patients show poorer performance on (social) cognitive tasks and neurophysiological measures ([Brunelin et al., 2007](#); [Cella et al., 2015](#); [Lavoie et al., 2013](#); [Seidman et al., 2015](#)) compared to healthy controls. [Brunelin et al. \(2007\)](#) showed that unaffected siblings performed at an intermediate level between healthy controls and schizophrenia patients on a source monitoring task, suggesting that poor source monitoring is a vulnerability marker of schizophrenia. [Hommes et al. \(2012\)](#) found similar results using an error-correction action-monitoring task. In our study, siblings also scored in between healthy controls and patients, although these differences were not statistically significant. The results of this exploratory analysis suggest that siblings do not show the same impairments in arriving at prime-based agency inferences as patients, while we can also not exclude the possibility that individuals at familial risk for schizophrenia are vulnerable to subtle impairments. Future studies should further examine this topic, for example by studying the development of agency impairments in genetic high risk populations.

#### 4.5. Future research

When investigating self-agency, we cannot disregard the role of neurocognitive impairments seen in patients with schizophrenia. Previously, it was shown that prime-based inferences do not rely on attentional control, self-reported motivation and attention, or reaction times during the task ([Renes et al., 2013](#)), and that patients are able to detect briefly presented primes ([Del Cul et al., 2006](#); [Renes et al., 2015](#)). These studies suggest that certain aspects of cognition do not relate to prime-based agency inferences. However, impaired neurocognition is one of the core deficits in schizophrenia. Hence, to exclude the possibility that impairments in prime-based inferences are due to neurocognitive impairments, we will include measures of, for instance, executive functioning and working memory performance in future studies.

#### 4.6. Conclusions

In conclusion, this study revealed that patients with symptoms of underattribution experience an overall lower level of self-agency over action-outcomes that were or were not implicitly primed. However, the ability to unconsciously use primes when inferring self-agency could not explain the decreased agency experiences in these patients. Future research on, for example, the combined influence of motor prediction and inferences might shed light on the question regarding the origin of a decreased agency experience in patients with symptoms of underattribution. Second, we found that patients show a reduced ability to use implicitly available environmental cues to inform inferences of self-agency, irrespective of severity of symptoms, suggesting that this impairment is a trait characteristic that is present in different stages of the disease. Also, we provide preliminary and suggestive evidence that individuals at increased familial risk for psychosis might perform suboptimal when making prime-based agency inferences. Siblings performed in between patients and healthy controls, suggesting that future research should further examine implicit agency inferences in individuals at increased familial risk. These first steps in unraveling impairments in making agency inferences indicate that such impairments may be an important topic in future research.

#### Conflict of interest

The authors have no conflict of interest to disclose.

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## Appendix A. Supplementary material

Supplementary data associated with this article can be found in the online version at <http://dx.doi.org/10.1016/j.psychres.2016.10.077>.

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