



## Action blind: Disturbed self-other integration in schizophrenia

Roman Liepelt<sup>a,\*</sup>, Johanna C. Schneider<sup>b</sup>, Désirée S. Aichert<sup>b</sup>, Nicola Wöstmann<sup>b</sup>, Sandra Dehning<sup>b</sup>, Hans-Jürgen Möller<sup>b</sup>, Michael Riedel<sup>b</sup>, Thomas Dolk<sup>c</sup>, Ulrich Ettinger<sup>d</sup>

<sup>a</sup> Institute for Psychology, Junior Group "Neurocognition of Joint Action", University of Muenster, Fliednerstrasse 21, Muenster 48149, Germany

<sup>b</sup> Department of Psychiatry, Ludwig-Maximilians-University, Munich, Germany

<sup>c</sup> Department of Psychology, Max-Planck-Institute for Human Cognitive and Brain Sciences, Leipzig, Germany

<sup>d</sup> Department of Psychology, University of Bonn, Bonn, Germany

### ARTICLE INFO

#### Article history:

Received 13 June 2012

Received in revised form

19 October 2012

Accepted 30 October 2012

Available online 7 November 2012

#### Keywords:

Joint action

Social Simon effect

Schizophrenia

### ABSTRACT

Recent research using individual task settings suggests that a major problem in schizophrenia is a dysfunctional theory of mind system leading to false mental state attributions. However, if a more low-level deficit to integrate own and other's actions (action blindness) is present in schizophrenia is still unknown. Using a Social Simon task, we tested if schizophrenia patients have a deficit in self-other integration. Further, we tested for a possible genetic bias of this dysfunction by studying clinically unaffected first-degree relatives of schizophrenia patients. While schizophrenia patients showed no Social Simon effect, we found a reliable Social Simon effect in healthy participants and first-degree relatives of schizophrenia patients. Joint task performance differed statistically between patients and healthy controls. We did not find any differences in the size of the Social Simon effects of relatives and healthy controls. The present findings suggest that schizophrenia patients have severe problems with self-other integration, which may lead to problems in social interactions. Since first-degree relatives of schizophrenia patients showed a reliable Social Simon effect, the evidence for a genetic bias of this social dysfunction in schizophrenia however is weak.

© 2012 Elsevier Ltd. All rights reserved.

### 1. Introduction

Schizophrenia is a mental disorder that is typically characterized by a range of neurocognitive deficits. These deficits range from inefficient executive functioning (Kerns, Nuechterlein, Braver, & Barch, 2008; Lesh, Niendam, Minzenberg, & Carter, 2011), decreased episodic memory (Aleman, Hijman, de Haan, & Kahn, 1999), altered reward and salience processing (Kapur, 2003; Walter et al. 2010), up to dysfunctional emotional regulation (Takahashia et al. 2004; for a detailed overview see Meyer-Lindenberg (2010)). More recently, abnormal social cognition (Biedermann, Frajo-Apor, & Hofer, 2012; Mier et al. 2010; Walter et al., 2010) mediated by medial prefrontal cortex (MPFC) and the temporoparietal junction (TPJ) has become a central focus in schizophrenia research. Deficits in social cognition are thought to account for the hallmark features of delusions and auditory hallucinations frequently present in schizophrenia patients (Blakemore, Wolpert, & Frith, 2002; Blakemore and Frith, 2003). Accordingly, an over-activation in MPFC in schizophrenia patients has been associated with a permanent screening of other persons'

actions or intentions (Walter et al. 2009). Also other neuroimaging findings showing abnormal (pre-) frontal functioning are consistent with the assumption that schizophrenia patients hyper-mentalize and arrive at high-level interpretations for stimuli that have objectively no social or intentional content (Biedermann et al., 2012; Walter et al., 2010).

The idea behind hyper-mentalization is that a relative complex cognitive function, theory of mind (TOM), is impaired in schizophrenia patients. TOM is defined as the ability to infer mental states of other people, such as their beliefs, thoughts, desires and intentions (Gallagher and Frith, 2003). A functioning TOM system helps to construct representations that relate oneself to other people in order to flexibly guide social interactions. Schizophrenia patients with a dysfunctional TOM system seem to over-interpret seemingly normal input information not only in social settings, but also when interpreting physical input information (Blakemore et al., 2002).

Interestingly, the TOM deficit of schizophrenia patients seems to be complementary to TOM deficits typically observed in patients with autism spectrum disorder. The TOM system of individuals with autism seems to be defective in a way that does not allow those patients to interpret social information of others—known as "mind blindness" (Baron-Cohen, 1995; Frith, 2001; Lombardo, Chakrabarti, Bullmore, Baron-Cohen, & MRC AIMS Consortium, 2011). One way to

\* Corresponding author.

E-mail address: [roman.liepelt@uni-muenster.de](mailto:roman.liepelt@uni-muenster.de) (R. Liepelt).

understand these effects in relation to the deficit present in schizophrenia patients has been proposed by Frith (1992) suggesting that people with autism do not infer mental states of others, because their cognitive defect is present from birth. In contrast the ability to infer mental states of other people in schizophrenia is relatively normal during early development, but they lose this ability during their first psychotic breakdown (Frith, 1992). As a consequence people with autism have a general problem to understand mental states of others, while schizophrenia patients still perform inferences about mental states of others; however, these inferences are often wrong. According to Frith (1992), schizophrenia patients see communicative intent where no such intent is present, resulting in delusions. Frith related the major problem in schizophrenia to so-called second order representations playing a key role in the awareness of our own and other people's goals and intentions and the reflection about others intentional states.

TOM deficits in schizophrenia patients have been differentiated from impairments in the sense of agency, the awareness that the self is causing an action (Allen et al. 2004; Schimansky, David, Rössler, & Haker, 2010). For a functioning sense of agency one needs the ability to correctly differentiate between self-produced action effects and those generated by others (Gallagher, 2000; Jeannerod, 2009; Schimansky et al., 2010). Accordingly, a disturbed sense of agency in schizophrenia patients has been explained by delusions of control. That is, schizophrenia patients cannot correctly predict the sensory consequences of their own actions (Frith, 2005; Frith, Blakemore, & Wolpert, 2000). While retaining the feeling that the actions they are performing are intentional, they assume that the intention is not their own (Frith & Corcoran, 1996). Such agency deficits in schizophrenia patients have been associated with an over-activation in TPJ (Spence et al. 1997), which often goes together with a loss of attenuation for self-generated actions (see Shergill, Bays, Frith, & Wolpert, 2003). Similar effects of attenuation loss have also been observed for the sensory consequences of speech (Ford et al. 2001), which may explain effects of auditory hallucinations in schizophrenia patients.

The aim of the present study was to test (1) if a relatively low-level social deficit concerning the integration of own and other's actions (action blindness) represents a basic deficit in schizophrenia patients and (2) if there is a genetic bias for action blindness in schizophrenia. To do so, we compared the performance in a Social Simon task (Sebanz, Knoblich, & Prinz, 2003) in a group of schizophrenia patients with the performance in an age and gender matched healthy control group. In a Social Simon task, two people share a task that is commonly used in the standard Simon paradigm (Simon, Hinrichs, & Craft, 1970). In the standard Simon task, single participants perform spatially defined responses (left and right key presses) to certain stimulus attributes (for example, left or right pointing triangles) that are randomly presented on the left or right side of a monitor. Under spatial correspondence of assigned stimulus and required response positions (e.g., left pointing triangle on the left side of the monitor and left response) participants typically show improved performance than under non-correspondence (e.g., left pointing triangle on the right side of the monitor and left response; De Jong, Liang, & Lauber, 1994; Simon & Rudell, 1967).

Sebanz et al. (2003) distributed the standard Simon task across two participants sitting next to each other, so that each participant had to respond to only one of the two stimuli by pressing one of the assigned keys. Sharing this go–nogo version of the Simon task with another person typically elicits a Stimulus–Response compatibility effect across both participants, known as the Social Simon effect (SSE; Sebanz et al., 2003; Tsai, Kuo, Jing, Hung, & Tzeng, 2006; Vlasic, Liepelt, Colzato, Prinz, & Hommel, 2010). Similar to the effect observed when one person is responsible for both responses in the standard Simon task, participants

in the Social Simon task respond faster when task-irrelevant stimulus features, such as the pointing direction of a finger (Sebanz et al., 2003) or the position of a geometric shape (Liepelt, Wenke, Fischer, & Prinz, 2011), correspond with an actor's response position. The SSE is typically explained on the basis of a common functional representational level of perception and action (Knoblich & Sebanz, 2006) providing a good measure of self-other integration (Colzato et al. 2012; Sebanz, Knoblich, & Prinz, 2005; Tsai, Kuo, Hung, & Tzeng, 2008; Wenke et al. 2011). Crucially, this is not the case when both co-actors perform the same go–nogo task in isolation (Liepelt et al., 2011; Sebanz et al., 2003). According to Theory of Event Coding (TEC) (Hommel, Müsseler, Aschersleben, & Prinz, 2001); Prinz, 1997) others' action events introduce a discrimination problem between action event representations referring to one's own action and those referring to the other person's action (Hommel, Colzato, & van den Wildenberg, 2009; Liepelt, Wenke, & Fischer, 2012). This is especially true for events that are similar to one's own action event representations. One way to resolve this discrimination problem is by means of relative spatial coding, the coding of one's own (and others') actions as left and right (Dittrich, Rothe, & Klauer, 2012; Dolk et al. 2011; Guagnano, Rusconi, & Umiltà, 2010; Liepelt, Stenzel, & Lappe, 2012). This re-introduces dimensional overlap between stimulus and response sets (Kornblum, Hasbroucq, & Osman, 1990) in a Social Simon task leading to response facilitation in compatible trials and response interference in incompatible trials and hence the SSE.

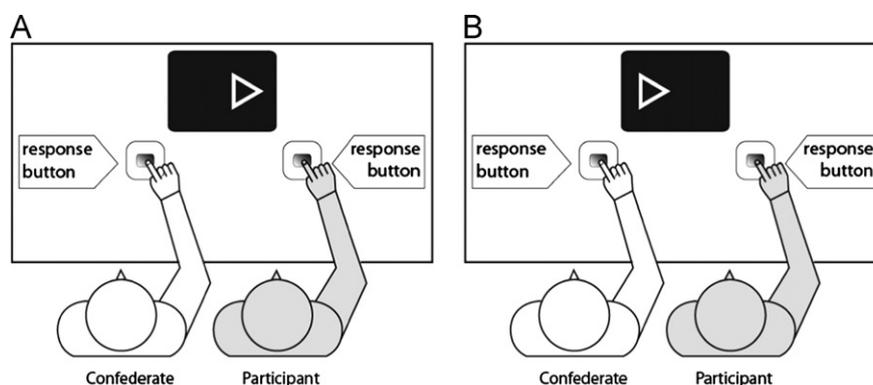
We tested three groups of participants on a Social Simon task: a group of hospitalized schizophrenia patients, a matched control group with healthy participants and a group of clinically unaffected first-degree relatives of schizophrenia patients in order to test for a genetic bias of action blindness in schizophrenia. First-degree relatives share on average 50% of their DNA sequence variations with the patients and are known to have a higher risk to suffer from schizophrenia. Therefore, behavioral or cognitive deficits observed in relatives (when compared to healthy controls without a family history of schizophrenia) are thought to reflect the genetic risk for the illness and are considered as endophenotypes (Gottesman & Gould, 2003).

If schizophrenia patients have a deficit in self-other integration (i.e., being action blind), then we expect a decreased SSE in schizophrenia patients, as compared to healthy controls. In contrast, however, if schizophrenia patients have no problems with self-other integration, we would expect no differences in the SSE between schizophrenia patients and healthy controls. Moreover, if there is a genetic bias for this deficit in schizophrenia, first-degree relatives of schizophrenia patients should show a decreased SSE as compared to healthy controls.

## 2. Method

### 2.1. Participants

Patients were recruited at the Department of Psychiatry and Psychotherapy of the University of Munich. The diagnosis of schizophrenia in the patients was made by experienced psychiatrists according to DSM-IV (American Psychiatric Association, 1994) criteria using the Structured Clinical Interview for DSM Axis I Disorders (SCID; First, Spitzer, Gibbon, & Williams, 1996). Severity of schizophrenia symptoms was rated using the Positive and Negative Syndrome Scale (PANSS; Kay, Fiszbein, & Opler, 1987) and further clinical data were collected in a semi-structured interview. Exclusion of DSM-IV Axis I diagnoses in relatives and controls was confirmed using the Mini International Neuropsychiatric Interview (MINI; Sheehan et al. 1998). An exclusion criterion for patients was a change in the type of medication 10 days or less before assessment. An exclusion criterion for all groups was the existence of neurological conditions. All participants were right-handed, had normal or corrected-to-normal vision, were naive with regard to the hypothesis of the experiment and received compensation for their participation. The ethics committee of the Faculty of Medicine of the University of Munich



**Fig. 1.** Experimental setup. Gray-shaded person indicates the participant, who is responding to his/her assigned stimulus (right pointing triangle) in a Stimulus–Response (S–R) compatible trial (A) and in an S–R incompatible trial (B).

approved the study and all participants gave their written informed consent to participate in the experiment, which was conducted in accordance with the ethical standards laid down in the 1975 Declaration of Helsinki.

## 2.2. Task and procedure

Two white triangles (11.0 × 9.0 cm) pointing to either the left or right (target stimuli) were chosen as go and nogo stimuli in a visual Social Simon task. Both triangles were presented either to the left or right (4.8 × 4.8 cm) from the midline of a 17-in. color monitor that was connected to a Pentium I PC (Fig. 1). The presentation of a left or a right pointing triangle on either the left or the right side of the screen, was followed by a white fixation cross (1.2 × 1.2 cm). At a viewing distance of 80 cm, the stimulus subtended a visual angle of 7.8° × 6.4°.

Prior to the instruction phase of the experiment, the participants (patients, relatives or controls) were seated on either the right or the left (counterbalanced across participants) next to a gender matched confederate. Both participant and confederate were asked to place their right index finger on a response button (25 cm in front and 25 cm away from the midline of the computer monitor) while placing their left hand underneath the table on their left thigh (Fig. 1).

To familiarize participants with the task, the experiment started with an instruction phase (~5 min) including the presentation of the two targets, their go–nogo assignment and a training of 10 trials in total. After the instruction phase was completed, the experimental phase started. Participants were instructed to respond as quickly and as accurately as possible to their individual target stimulus (left sitting person to left pointing triangles and right sitting person to right pointing triangles) irrespective of its location (left or right to the midline of the computer monitor) and to keep fixating the position of the fixation cross (center of a computer monitor) throughout the entire experiment.

There were two blocks of 64 trials separated by a short break. Go and nogo-signals were randomly presented within each block: 32 with a spatially compatible stimulus–response relationship (e.g. a left pointing triangle presented on the left side of the screen) and 32 with a spatially incompatible stimulus–response relationship (e.g. a left pointing triangle presented on the right side of the screen). Each trial began with the presentation of a left or a right pointing triangle to the left or the right of the screen for 400 ms. After the critical stimulus disappeared, a white fixation cross was presented for 1100 ms before the next trial began. The whole experiment took approximately 20 min.

## 3. Results

### 3.1. Clinical and sociodemographic data

Twenty-one patients (nine female; 19–60 years of age,  $M=38.7$ ,  $SD=13.0$ ), 21 healthy controls (10 female; 18–51 years of age,  $M=34.9$ ,  $SD=10.8$ ), and 21 healthy first-degree relatives of schizophrenia patients (15 female; 18–67 years of age,  $M=41.4$ ,  $SD=16.0$ ) took part in the study. All three groups were comparable regarding age,  $F(2,56)=1.14$ ,  $p > 0.05$ , and gender,  $\chi^2(2)=3.27$ ,  $p > 0.05$ .

The mean PANSS positive symptom rating at the time of the study in the patients was 14.8 ( $SD=5.3$ ), the mean negative symptom rating was 22.1 ( $SD=8.2$ ) and the mean general psychopathology rating was 37.8 ( $SD=10.9$ ). All patients were treated with atypical antipsychotics. The average number of psychiatric

hospitalizations in the patient group was 6.71 ( $SD=5.52$ ) and the mean number of years since first treatment for schizophrenia was 12.69 ( $SD=9.99$ ).

### 3.2. Reaction times

For statistical analysis, we excluded all trials in which the responses were incorrect (1.3%) and the reaction times (RTs) were faster than 150 ms or slower than 1000 ms (3.5%; Liepelt et al., 2011; Röder, Kusmirek, Spence, & Schicke, 2007). Difference scores of the RTs were calculated by subtracting the average RT of compatible trials from the average RT of incompatible trials. Due to high error rates (2.5 SDs above the mean) or difference scores over 2.5 SDs above the mean (Müller et al. 2011), we had to exclude two patients, one relative and one control participant from further analysis.

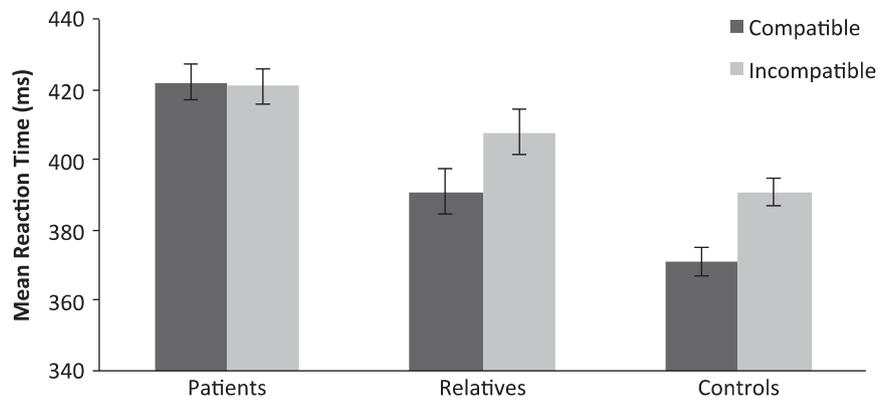
To test for a deficit in self–other integration, we performed a 2 × 2 repeated measures analysis of variance (ANOVA) with the within-subjects factor compatibility (compatible, incompatible) and group (patients, controls). We found a decreased SSE in schizophrenia patients (–1 ms,  $p > 0.05$ ) as compared to healthy controls (16 ms,  $p < 0.016$ ), as indicated by a significant interaction of compatibility × group,  $F(1,37)=4.66$ ,  $p < 0.05$ ,  $\eta^2=0.11$  (Fig. 2).

To test for a genetic bias, we compared performance in relatives and controls. We observed no significant difference of the SSE in relatives (21 ms,  $p < 0.016$ ) and controls (16 ms,  $p < 0.016$ ),  $F(1,38) < 1$  (Fig. 2), but a significant main effect of compatibility,  $F(1,38)=18.60$ ,  $p < 0.001$ ,  $\eta^2=0.33$ .

## 4. Discussion

The present study aimed at investigating whether schizophrenia patients have a deficit in self–other integration during joint action. We compared the performance of schizophrenia patients with the performance of healthy controls in a Social Simon task. A comparison of first-degree relatives with healthy controls was applied to test for a possible genetic bias of this dysfunction (Gottesman & Gould, 2003).

We observed a significant SSE in healthy controls and in first-degree relatives of schizophrenia patients. The size of the SSE in relatives and in controls did not differ statistically from each other. Importantly, the SSE in schizophrenia patients was fully absent. When comparing the sizes of the SSE in schizophrenia patients with that of healthy controls, we found a significant reduction in the SSE in schizophrenia patients relative to healthy controls. These findings are in line with the assumption that schizophrenia patients have a deficit in self–other integration.



**Fig. 2.** Mean reaction time as a function of spatial stimulus–response compatibility per group (patients, relatives, controls). Error bars represent standard errors of the mean differences.

This deficit may be related to a problem of controlling one's own actions or a problem to integrate action representations of self and other (Sebanz et al., 2003). In the latter case the deficit would primarily be a social deficit even though the first variant may also produce deficits in the integration of own and other's action events in social interactions. While the action control variant would predict an overall performance deficit also in a standard (two-choice) Simon task, the social variant of the deficit would predict a more specialized deficit in the social version of the Simon task. Accordingly, two recent studies investigating the standard Simon effect do not report differences in the size of the overall Simon effect between healthy controls and schizophrenia patients (Gastaldo, Umiltà, Bianchin, & Prior, 2002; Lalanne, van Assche, & Giersch, 2012). However, both studies report more subtle differences with respect to temporal continuity judgments (Lalanne et al., 2012) and differences in left–right hemifield processing in patients with a negative symptomatic (Gastaldo et al., 2002). Thus, considering that schizophrenia patients show a standard Simon effect one may argue that the lack of the SSE we found in schizophrenia patients is related to a more specialized deficit in integrating actions produced by self and other representing a primarily social deficit. However, more research has to be performed to test this assumption further.

One potential source of the integration deficit might relate to an inability to spatially re-code one's own actions relative to others actions in a task sharing situation (Dolk et al., 2011; Dolk, Liepelt, Villringer, Prinz, & Ragert, 2012; Hommel et al., 2009) removing the dimensional overlap between stimulus and response sets and hence the SSE. This conclusion is in accordance with the assumption that schizophrenia patients are likely to confuse the events of their own action with external events (Blakemore and Frith, 2003; Blakemore et al., 2002; Frith, 1987; Haggard, Martin, Taylor-Clarke, Jeannerod, & Franck, 2003; Voss et al. 2010). As these previous studies showed that schizophrenia patients have problems in forming representations of the predicted action consequences of their own actions, the present study is, according to our knowledge, the first to show that schizophrenia patients have a lack of integrating other's actions in the self.

Interestingly, our results on schizophrenia patients seem to be different to those observed in autistic patients who showed a full-blown SSE in a previous study (Sebanz, Knoblich, Stumpf, & Prinz, 2005) using a comparable Social Simon task. Hence, unlike schizophrenia patients, people with autism are not action blind (Sebanz, Knoblich, Stumpf et al., 2005), but seem to be mind-blind (Baron-Cohen, 1995; Frith, 2001; Lombardo et al., 2011). It seems that people with autism have a specific deficit in attributing representational states to others, whereas more low-level self-other integration processes seem to be efficient.

The present finding of a lack of an SSE in schizophrenia patients is partly in line with recent findings of Humphreys and Bedford (2011). These authors showed a relation of joint action and TOM in patients with frontal and posterior parietal lesions. In the Humphreys and Bedford (2011) study frontal and parietal lesion patients, who had severe problems with TOM tasks also showed a reduced SSE in a social Simon task using word stimuli as compared to healthy controls. When explicitly instructing these patients to take the other person's action into account the authors found that a reliable SSE reemerged in patients with parietal lesions. In patients with frontal lesions a small SSE also reemerged by instruction, which decreased again over time. Humphreys & Bedford (2011) concluded that these findings may be due to an inability to attend to appropriate social cues (posterior parietal patients) or to a problem in preserving enough processing resources to maintain the other person's actions over time (frontal patients). As we did not explicitly instruct our schizophrenia patients to attend to the other person's action we cannot draw a specific conclusion on effects of task instruction. However, as schizophrenia patients often show abnormal functioning in MPFC and TPJ (Biedermann et al., 2012; Mier et al., 2010; Walter et al., 2010), as well as a lack of an SSE, a strong relation between self-other integration and TOM should be predicted. Nevertheless, we would argue that self-other integration might also be selectively impaired. If self-other integration is disturbed in schizophrenia patients, the TOM system that further processes this incorrect incoming information may also come up with incorrect high-level interpretations about the other person's intentions (Gallagher and Frith, 2003) even if the TOM system would still be functioning. Consider for example a situation where you sit at a table opposite another person. A number of objects are located at specific locations of the table. Now you perceive an object moving from one side of the table to the other side without perceiving the other person performing an action that may have caused the object movement. Under these circumstances, even a person with a functioning TOM system would have to come up with some odd high-level interpretation to explain the situation. This could be a scenario you are confronted with when your self-other integration mechanism is disturbed. The same may be true for schizophrenia patients, whereby the problem for such a misinterpretation may not (necessarily) be a dysfunctioning TOM system, but may be the result of an inability to correctly integrate one's own and other's actions. Further research testing whether there are schizophrenia patients who selectively differ with respect to problems in self-other integration and more complex TOM abilities is needed to gain more information on this issue.

When comparing the SSE in healthy controls and clinically unaffected first-degree relatives of schizophrenia patients, sharing on average 50% of their genes with the schizophrenia patients, we did

not observe any significant differences in the size of the SSE. This finding provides no evidence for a direct genetic influence of action blindness in schizophrenia patients. As a consequence, this result suggests that the deficit observed in schizophrenia patients does not present an endophenotype (Gottesman & Gould, 2003). One may speculate that the problem with self-other integration we observed in schizophrenia patients is based on environmental etiological factors that play a causal role in the development of the illness, from neurocognitive changes associated with the illness expression itself, or from influences of subsequent antipsychotic treatment. The fact that all patients were treated with atypical antipsychotics represents a limitation of the present study. An effect of drug treatment can therefore not be fully ruled out. However, if this would be the case one may expect a general slowing of schizophrenia patients relative to healthy controls under drug treatment (Jogems-Kosterman, Zitman, Van Hoof, & Hulstijn, 2001). However, as we found no overall differences in response speed between patients and controls it seems unlikely that drug treatment itself has caused the lack of an SSE in schizophrenia patients.

Based on the present findings showing a lack of an SSE in schizophrenia patients, without a strong genetic bias, it appears tempting for prospects of future research as well as highly relevant for rehabilitative purposes to investigate whether specific cognitive trainings forcing participants to improve the integration process between own and other's actions, with paradigms like the Social Simon task (Sebanz et al., 2003) or the imitation-inhibition task (Brass, Bekkering, Wohlschläger, & Prinz, 2000; Liepelt, von Cramon, & Brass, 2008) will lead to an improvement of social cognitive abilities in schizophrenia patients.

## 5. Conclusion

Testing for action blindness in a Social Simon task, we found a full breakdown of the Social Simon effect in schizophrenia without evidence for a direct genetic predisposition. This suggests a basic deficit in the integration of own and other's actions in schizophrenia.

## Acknowledgments

This study was supported by the following German Research Foundation grants: DFG LI 2115/1-1 awarded to Roman Liepelt and DFG ET 31/2-1 awarded to Ulrich Ettinger. The authors wish to thank two anonymous reviewers for helpful comments on a previous version of the manuscript.

## References

- American Psychiatric Association (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, D.C.: American Psychiatric Association.
- Aleman, A., Hijman, R., de Haan, E. H., & Kahn, R. S. (1999). Memory impairment in schizophrenia: A meta-analysis. *The American Journal of Psychiatry*, *156*, 1358–1366.
- Allen, P. P., Johns, L. C., Fu, C. H., Broome, M. R., Vythelingum, G. N., & McGuire, P. K. (2004). Misattribution of external speech in patients with hallucinations and delusions. *Schizophrenia Research*, *69*, 277–287.
- Baron-Cohen, S. (1995). *Mindblindness: An essay on autism and theory Of mind*. Cambridge, MA: MIT Press.
- Biedermann, F., Frajo-Apor, B., & Hofer, A. (2012). Theory of mind and its relevance in schizophrenia. *Current Opinion in Psychiatry*, *25*, 71–75.
- Blakemore, & Frith (2003). Self-awareness and action. *Current Opinion in Neurobiology*, *13*, 219–224.
- Blakemore, Wolpert, & Frith (2002). Abnormalities in the awareness of action. *Trends in Cognitive Sciences*, *6*, 237–242.
- Brass, M., Bekkering, H., Wohlschläger, A., & Prinz, W. (2000). Compatibility between observed and executed finger movements: Comparing symbolic, spatial, and imitative cues. *Brain and Cognition*, *44*, 124–143.
- Colzato, L. S., Zech, H., Hommel, B., Verdonschot, R., van den Wildenberg, W., & Hsieh, S. (2012). Loving-kindness brings loving-kindness: The impact of Buddhism on cognitive self-other integration. *Psychonomic Bulletin & Review*, *19*, 541–545.
- De Jong, R., Liang, C.-C., & Lauber, E. (1994). Conditional and unconditional automaticity: dual-process model of effects of spatial stimulus-response correspondence. *Journal of Experimental Psychology: Human Perception and Performance*, *20*, 731–750.
- Dittrich, K., Rothe, A., & Klauer, K. C. (2012). Increased spatial salience in the social Simon task: A response coding account of spatial compatibility effects. *Attention, Perception, & Psychophysics*, *74*, 911–929.
- Dolk, T., Hommel, B., Colzato, L. S., Schütz-Bosbach, S., Prinz, W., & Liepelt, R. (2011). How social is the social Simon effect? *Frontiers in Psychology*, *2*, 84. <http://dx.doi.org/10.3389/fpsyg.2011.00084>.
- Dolk, T., Liepelt, R., Villringer, A., Prinz, W., & Ragert, P. (2012). Morphometric gray matter differences of the medial frontal cortex influence the social Simon effect. *Neuroimage*, *61*, 1249–1254.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1996). *Structured clinical interview for DSM-IV Axis I disorders research version (SCID-I)*. Biometrics Research, New York State Psychiatric Institute: New York.
- Ford, J. M., Mathalon, D. H., Kalba, S., Whitfield, S., Faustman, W. O., & Roth, W. T. (2001). Cortical responsiveness during talking and listening in schizophrenia: An event-related brain potential study. *Biological Psychiatry*, *50*, 540–549.
- Frith, C. D. (1987). The positive and negative symptoms of schizophrenia reflect impairments in the perception and initiation of action. *Psychological Medicine*, *17*, 631–648.
- Frith, C. D. (1992). *The cognitive neuropsychology of schizophrenia*. Hove: Lawrence Erlbaum Associates.
- Frith, C. D. (2005). The self in action: Lessons from delusions of control. *Consciousness and Cognition*, *14*, 752–770.
- Frith, C. D., Blakemore, S. J., & Wolpert, D. M. (2000). Explaining the symptoms of schizophrenia: Abnormalities in the awareness of action. *Brain Research Reviews*, *31*, 357–363.
- Frith, C. D., & Corcoran, R. (1996). Exploring 'theory of mind' in people with schizophrenia. *Psychological Medicine*, *26*, 521–530.
- Frith, U. (2001). Mind blindness and the brain in autism. *Neuron*, *32*, 969–979.
- Gallagher, H. L., & Frith, C. D. (2003). Functional imaging of 'theory of mind'. *Trends in Cognitive Sciences*, *7*, 77–83.
- Gallagher, S. (2000). Philosophical conceptions of the self: Implications for cognitive science. *Trends in Cognitive Sciences*, *4*, 14–21.
- Gastaldo, S., Umiltà, C., Bianchin, G., & Prior, M. (2002). The Simon effect in schizophrenic patients with negative symptoms. *Cortex: A Journal Devoted to the Study of the Nervous System and Behavior*, *38*, 149–159.
- Gottesman, I. I., & Gould, T. D. (2003). The endophenotype concept in psychiatry: etymology and strategic intentions. *The American Journal of Psychiatry*, *160*, 636–645.
- Guagnano, D., Rusconi, E., & Umiltà, C. A. (2010). Sharing a task or sharing space? On the effect of the confederate in action coding in a detection task. *Cognition*, *114*, 348–355.
- Haggard, P., Martin, F., Taylor-Clarke, M., Jeannerod, M., & Franck, N. (2003). Awareness of action in schizophrenia. *Neuroreport*, *14*, 1081–1085.
- Hommel, B., Colzato, L. S., & van den Wildenberg, W. P. M. (2009). How social are task representations. *Psychological Science*, *20*, 794–798.
- Hommel, B., Müsseler, J., Aschersleben, G., & Prinz, W. (2001). The theory of event coding (TEC): A framework for perception and action planning. *Behavioral and Brain Sciences*, *24*, 849–878.
- Humphreys, G. W., & Bedford, J. (2011). The relations between joint action and theory of mind: A neuropsychological analysis. *Experimental Brain Research*, *211*, 357–369.
- Jeannerod, M. (2009). The sense of agency and its disturbances in schizophrenia: a reappraisal. *Experimental Brain Research*, *192*, 527–532.
- Jogems-Kosterman, B. J., Zitman, F. G., Van Hoof, J. J., & Hulstijn, W. (2001). Psychomotor slowing and planning deficits in schizophrenia. *Schizophrenia Research*, *48*, 317–333.
- Kapur, S. (2003). Psychosis as a state of aberrant salience: a framework linking biology, phenomenology, and pharmacology in schizophrenia. *The American Journal of Psychiatry*, *160*, 13–23.
- Kay, S. R., Fiszbein, A., & Opler, L. A. (1987). The positive and negative syndrome scale (PANSS) for schizophrenia. *Schizophrenia Bulletin*, *13*, 261–276.
- Kerns, J. G., Nuechterlein, K. H., Braver, T. S., & Barch, D. M. (2008). Executive Functioning Component Mechanisms and Schizophrenia. *Biological Psychiatry*, *64*, 26–33.
- Knoblich, G., & Sebanz, N. (2006). The social nature of perception and action. *Current Directions in Psychological Science*, *15*, 99–104.
- Kornblum, S., Hasbroucq, T., & Osman, A. (1990). Dimensional overlap: Cognitive basis for stimulus-response compatibility—A model and taxonomy. *Psychological Review*, *97*, 253–270.
- Lalanne, L., van Assche, M., & Giersch, A. (2012). When predictive mechanisms go wrong: Disordered visual synchrony thresholds in schizophrenia. *Schizophrenia Bulletin*, *38*, 506–513.
- Lesh, T. A., Niendam, T. A., Minzenberg, M. J., & Carter, C. S. (2011). Cognitive control deficits in schizophrenia: Mechanisms and meaning. *Neuropsychopharmacology: Official Publication of the American College of Neuropsychopharmacology*, *36*, 316–338.
- Liepelt, R., Stenzel, A., & Lappe, M. (2012). Specifying social cognitive processes with a social dual-task paradigm. *Frontiers in Human Neurosciences*, *6*, 86. <http://dx.doi.org/10.3389/fnhum.2012.00086>.

- Liepelt, R., von Cramon, D. Y., & Brass, M. (2008). What is matched in direct matching? Intention attribution modulates motor priming. *Journal of Experimental Psychology: Human Perception and Performance*, 34, 578–591.
- Liepelt, R., Wenke, D., & Fischer, R. (2012). Effects of feature integration in a hands-crossed version of the social Simon paradigm. *Psychological Research*, <http://dx.doi.org/10.1007/s00426-012-0425-0>.
- Liepelt, R., Wenke, D., Fischer, R., & Prinz, W. (2011). Trial-to-trial sequential dependencies in a social and non-social Simon task. *Psychological Research*, 75, 366–375.
- Lombardo, M. V., Chakrabarti, B., Bullmore, E. T., Baron-Cohen, S., & MRC AIMS Consortium (2011). Specialization of right temporo-parietal junction for mentalizing and its relation to social impairments in autism. *Neuroimage*, 56, 1832–1838.
- Meyer-Lindenberg, A. (2010). From maps to mechanisms through neuroimaging of schizophrenia. *Nature*, 468, 194–202.
- Mier, D., Sauer, C., Lis, S., Esslinger, C., Wilhelm, J., Gallhofer, B., et al. (2010). Neuronal correlates of affective theory of mind in schizophrenia-outpatients: Evidence for a baseline deficit. *Psychological Medicine*, 40, 1607–1617.
- Müller, B. C. N., Kühn, S., van Baaren, R. B., Dotsch, R., Brass, M., & Dijksterhuis, A. (2011). Perspective taking eliminates differences in co-representation of out-group members' actions. *Experimental Brain Research*, 211, 423–428.
- Prinz, W. (1997). Perception and action planning. *European Journal of Cognitive Psychology*, 9, 129–154.
- Röder, B., Kusmierek, A., Spence, C., & Schicke, T. (2007). Developmental vision determines the reference frame for the multisensory control of action. *Proceedings of the National Academy of Sciences USA*, 104, 4753–4758.
- Schimansky, J., David, N., Rössler, W., & Haker, H. (2010). Sense of agency and mentalizing: Dissociation of subdomains of social cognition in patients with schizophrenia. *Psychiatry Research*, 178, 39–45.
- Sebanz, N., Knoblich, G., & Prinz, W. (2005). How two share a task: Corepresenting stimulus–response mappings. *Journal of Experimental Psychology: Human Perception and Performance*, 31, 1234–1246.
- Sebanz, N., Knoblich, G., & Prinz, W. (2003). Representing others' actions: just like one's own? *Cognition*, 88, B11–B21.
- Sebanz, N., Knoblich, G., Stumpf, L., & Prinz, W. (2005). Far from action-blind: Representation of others' actions in individuals with autism. *Cognitive Neuropsychology*, 22, 433–454.
- Sheehan, D. V., Lecrubier, Y., Sheehan, K. H., Amorim, P., Janavs, J., Weiller, E., et al. (1998). The Mini-International Neuropsychiatric Interview (M.I.N.I.): The development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *Journal of Clinical Psychiatry*, 59(Suppl. 20), 22–33.
- Shergill, S. S., Bays, P. M., Frith, C. D., & Wolpert, D. M. (2003). Two eyes for an eye: The neuroscience of force escalation. *Science (New York, NY)*, 301, 187.
- Simon, J. R., Hinrichs, J. V., & Craft, J. L. (1970). Auditory S–R compatibility: Reaction time as a function of ear-hand correspondence and ear-response-location correspondence. *Journal of Experimental Psychology*, 86, 97–102.
- Simon, J. R., & Rudell, A. P. (1967). Auditory S–R compatibility: The effect of an irrelevant cue on information processing. *Journal of Applied Psychology*, 51, 300–304.
- Spence, S. A., Brooks, D. J., Hirsch, S. R., Liddle, P. F., Meehan, J., & Grasby, P. M. (1997). A PET study of voluntary movement in schizophrenic patients experiencing passivity phenomena (delusions of alien control). *Brain*, 120, 1997–2011.
- Takahashia, H., Koedac, M., Odaa, K., Matsudada, T., Matsushimada, E., Matsuura, M., et al. (2004). An fMRI study of differential neural response to affective pictures in schizophrenia. *Neuroimage*, 22, 1247–1254.
- Tsai, C.-C., Kuo, W. J., Hung, D. L., & Tzeng, O. J. L. (2008). Action co-representation is tuned to other humans. *The Journal of Cognitive Neurosciences*, 20, 2015–2024.
- Tsai, C.-C., Kuo, W. J., Jing, J. T., Hung, D. L., & Tzeng, O. J. L. (2006). A common coding framework in self-other interaction: Evidence from joint action task. *Experimental Brain Research*, 175, 353–362.
- Vlainic, E., Liepelt, R., Colzato, L. S., Prinz, W., & Hommel, B. (2010). The virtual co-actor: The social Simon effect does not rely on online feedback from the other. *Frontiers in Psychology*, 1, 208, <http://dx.doi.org/10.3389/fpsyg.2010.00208>.
- Voss, M., Moore, J., Hauser, M., Galliant, J., Heinz, A., & Haggard, P. (2010). Altered awareness of action in schizophrenia: a specific deficit in predicting action consequences. *Brain*, 133, 3104–3112.
- Walter, H., Ciaramidaro, A., Adenzato, M., Vasic, N., Ardito, R. B., Erk, S., et al. (2009). Dysfunction of the social brain in schizophrenia is modulated by intention type: An fMRI study. *Social Cognitive and Affective Neuroscience*, 4, 166–176.
- Walter, H., Heckers, S., Kassubek, J., Erk, S., Frasch, K., & Abler, B. (2010). Further evidence for aberrant prefrontal salience coding in schizophrenia. *Frontiers in Behavioral Neurosciences*, 3, 62, <http://dx.doi.org/10.3389/neuro.08.062.2009>.
- Wenke, D., Atmaca, S., Holländer, A., Liepelt, R., Baess, P., & Prinz, W. (2011). What is shared in joint action? Issues of co-representation, response conflict, and agent identification. *Review of Philosophy and Psychology*, 2, 147–172.