

BRIEF REPORT

Is Social Attention Impaired in Schizophrenia? Gaze, but not Pointing Gestures, Is Associated With Spatial Attention Deficits

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Objective: The nature of possible impairments in orienting attention to social signals in schizophrenia is controversial. The present research was aimed at addressing this issue further by comparing gaze and arrow cues. Unlike previous studies, we also included pointing gestures as social cues, with the goal of addressing whether any eventual impairment in the attentional response was specific to gaze signals or reflected a more general deficit in dealing with social stimuli. **Method:** Patients with schizophrenia or schizoaffective disorder and matched controls performed a spatial-cuing paradigm in which task-irrelevant centrally displayed gaze, pointing finger, and arrow cues oriented rightward or leftward, preceded a lateralized target requiring a simple detection response. **Results:** Healthy controls responded faster to spatially congruent targets than to spatially incongruent targets, irrespective of cue type. In contrast, schizophrenic patients responded faster to spatially congruent targets than to spatially incongruent targets only for arrow and pointing finger cues. No cuing effect emerged for gaze cues. **Conclusion:** The results support the notion that gaze cuing is impaired in schizophrenia, and suggest that this deficit may not extend to all social cues.

Keywords: social attention, gaze cuing, symbolic cuing, schizophrenia

The notion that attentional processing may be impaired in schizophrenia has been the focus of extensive research, especially with spatial-cuing paradigms (Luck & Gold, 2008). These consist of the presentation of a spatial cue providing information concerning the location of a subsequent peripheral target stimulus requiring a response (e.g., Posner, 1980). The vast majority of studies

involving schizophrenic patients have focused on reflexive attentional orienting elicited by uninformative peripheral abrupt onsets, reporting that, relative to controls, the magnitude of spatial orienting in schizophrenics seems to be even enhanced, at least under some circumstances (e.g., Fuentes, Boucart, Alvarez, Vivas, & Zimmerman, 1999). Much less is known about reflexive orienting elicited by social cues. More generally, evidence is accumulating showing that schizophrenic patients are impaired in dealing with social stimuli, in particular in processing information conveyed by eye gaze (e.g., Hooker & Park, 2005; Tso, Mui, Taylor, & Deldin, 2012).

Looking at gaze is an essential ability for the creation of empathic contact among individuals and gaze is a key factor in the regulation of social interactions. Indeed, the specific type of relationship between the perceiver and the observed person can impact the attention devoted to gaze (e.g., Dalmaso, Pavan, Castelli, & Galfano, 2012; Pavan, Dalmaso, Galfano, & Castelli, 2011). The influence of gaze upon attention orientation can be experimentally investigated by presenting a central face with averted gaze that serves as a spatial cue for an upcoming peripheral target. In healthy participants, this gaze-cuing paradigm typically triggers reflexive shifts of attention toward the spatial location indicated by gaze (e.g., Friesen & Kingstone, 1998; Frischen, Bayliss, & Tipper, 2007; Galfano et al., 2011), followed by an inhibitory aftereffect known as inhibition of return (IOR; Frischen & Tipper, 2004), a

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Table 1
Demographic and Clinical Information of Participants

Variable	Schizophrenic patients (<i>n</i> = 18)	Healthy controls (<i>n</i> = 18)
Mean age (years)	49 (7.12)	49 (7.04)
Gender	M 9, F 9	M 9, F 9
Handedness	R 18	R 18
Mean education (years)	9.33 (2.83)	9.67 (2.63)
Mean age of illness onset (years)	28.5 (5.18)	
Mean duration of illness (years)	21 (7.24)	
Typical antipsychotic medication (%)	45.9	
Atypical antipsychotic medication (%)	54.1	

Note. Values in parentheses are *SDs*. The most frequently used typical antipsychotic medication was bromperidol, used in conjunction with aripiprazole and fluphenazine (*n* = 1), clozapine (*n* = 2), clozapine and clotiapine (*n* = 1), clozapine and olanzapine (*n* = 1), levomepromazine (*n* = 1), levomepromazine and clotiapine (*n* = 1). The most frequently used atypical antipsychotic medication was clozapine, used in conjunction with haloperidol (*n* = 3), followed by quetiapine used in conjunction with haloperidol (*n* = 1), olanzapine (*n* = 1), paliperidone (*n* = 1), and perphenazine (*n* = 1). Olanzapine was also used in conjunction with haloperidol (*n* = 1), and levomepromazine (*n* = 1). Two schizophrenic patients were also prescribed olanzapine (*n* = 1) and risperidon (*n* = 1) alone. The mean chlorpromazine (CPZ) equivalent daily dose of antipsychotic was 286.11 g (*SD* = 303.8). Pharmacological therapy included also anxiolytics (benzodiazepines).

phenomenon traditionally observed using peripheral cues (e.g., Lupiáñez, Klein, & Bartolomeo, 2006).

The few existing studies addressing gaze cuing in schizophrenic patients reported a different trend. Indeed, schizophrenics exhibit IOR for peripheral onset, but not for gaze cues (Nestor, Klein, Pomplun, Niznikiewicz, & McCarley, 2010) whereas the immediate attentional response to gaze cues is more controversial. In this regard, Langdon, Corner, McLaren, Coltheart, and Ward (2006) reported that, compared with healthy controls, schizophrenic patients showed an early rising reflexive shift of attention. At short stimulus-onset asynchrony (SOA), that is, when the time between cue onset and target onset was set to 100 ms, a gaze-cuing effect emerged only among schizophrenic patients. At longer SOAs (i.e., 300 and 800 ms), gaze cuing was reliable and undistinguished in both schizophrenics and healthy controls. A very different pattern has recently been reported by Akiyama et al. (2008). They hypothesized that abnormal gaze cuing might specifically be observed in long-term, unremitted patients, given that hyposensitivity to social stimuli is prominent in chronic schizophrenia (e.g., Hooker & Park, 2005; Tso et al., 2012). In comparing gaze cues with central arrows, Akiyama et al. (2008) observed no differences in performance between chronic schizophrenics and healthy controls with regard to arrow-driven orienting of attention. However, in line with predictions, a decreased attentional response to gaze was observed among schizophrenics, but not among healthy controls. It is important to note, experimental manipulations employed by Langdon et al. (2006) and Akiyama et al. (2008) to create gaze cues were extremely polarized. On the one hand, Langdon et al. (2006) cued participants' attention by means of a turn of the head and eyes, namely, they presented two simultaneous social cues (i.e., gaze direction and head orientation). On the other hand, Akiyama et al. (2008) used a particularly impoverished social cue (i.e., a pair of rectangular vs. elliptical eyes).

The first goal of the present study was to further explore gaze-cuing response in schizophrenic patients using a gaze cue in isolation (i.e., the spatial cue only included gaze direction) but presenting the eyes embedded in a face, to provide a more eco-

logical stimulus. Second, we aimed to explore the extent to which the altered social-cuing response, if any, is specific to gaze stimuli or extends to other socially relevant cues. In this regard, pointing gestures elicit consistent attention shifts in healthy participants and are indeed powerful social cues for communicating information and intentions among humans (e.g., Cazzato, Macaluso, Crostella, & Aglioti, 2012). Previous studies addressing social cuing in schizophrenia only focused on gaze. For this reason, including pointing gestures in a modified spatial-cuing paradigm is particularly relevant for understanding whether any eventual impairment involves social cues overall or whether abnormal processing is confined to eye gaze. Thus, our second goal was testing whether the altered attentional response demonstrated by schizophrenic patients reported in previous studies reflects gaze-specific impairments or is the consequence of a more general impaired mecha-

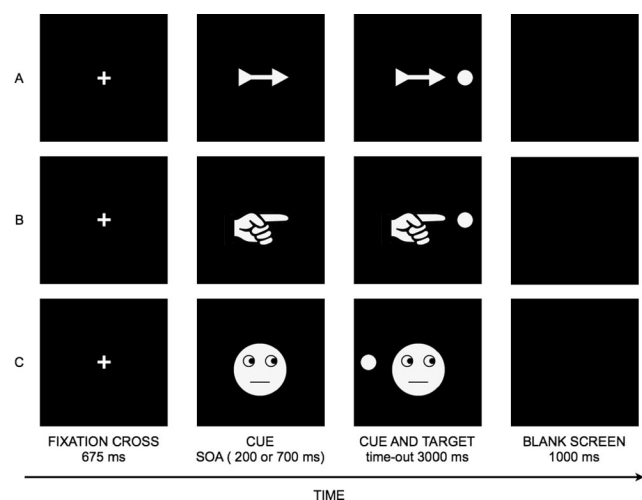


Figure 1. Illustration of stimuli (not drawn to scale) and sequence of events for a spatially congruent trial with arrow (A) and pointing finger (B) cues, and for a spatially incongruent trial with gaze cue (C).

Table 2

Mean RT (in Ms) and Reciprocal Scores (RS) Computed From Raw RT Data for Spatially Congruent and Incongruent Trials as a Function of Both Cue Type and SOA for Schizophrenic Patients and Healthy Controls

Participants	Arrows								Pointing finger			
	200				700				200			
	C		I		C		I		C		I	
	RT	RS	RT	RS	RT	RS	RT	RS	RT	RS	RT	RS
Schizophrenic Patients	706 (328)	0.001781 (0.000579)	749 (319)	0.001698 (0.000511)	710 (318)	0.001807 (0.000565)	732 (305)	0.001703 (0.000491)	711 (350)	0.001826 (0.000625)	719 (358)	0.001805 (0.000602)
Healthy Controls	501 (146)	0.002219 (0.000528)	536 (181)	0.002154 (0.000563)	518 (164)	0.002197 (0.000569)	528 (165)	0.002155 (0.000559)	505 (195)	0.002284 (0.000551)	522 (184)	0.002198 (0.000521)

Note. SDs Are Reported in brackets. C = congruent trials; I = incongruent trials.

nism in processing social stimuli. In addition to gaze and pointing cues, arrow cues were also included in our study, to obtain a direct comparison between social and symbolic stimuli (see also Akiyama et al., 2008; Spencer et al., 2011). We predicted that healthy controls would shift attention to the cued location irrespective of cue type. In contrast, previous work might lead one to predict either a similar (Langdon et al., 2006) or a decreased (Akiyama et al., 2008) gaze-cuing effect for schizophrenic patients compared with healthy controls. Any altered attentional response for both gaze and pointing gestures would support the view that schizophrenia is associated with a general deficit in social-cognitive processes. On the other hand, any eventual pattern of findings showing an altered attentional response only for gaze cues would be consistent with the view that gaze is a special social signal characterized by unique ontogenetic and phylogenetic roots (e.g., Farroni, Csibra, Simion, & Johnson, 2002). Finally, no altered attentional response was expected in schizophrenics for arrow cues, due to their symbolic nature.

Method

Participants

Eighteen outpatient clinical participants were recruited from two psychiatric clinics located in northern Italy. Fifteen patients had a diagnosis of schizophrenia and three were diagnosed with schizoaffective disorder. Diagnoses were made by a board-certified attending research team of psychiatrists using the International Classification of Diseases (ICD-10, World Health Organization, 1992). Eighteen healthy participants, selected carefully to be perfectly matched by gender, age, and education with the schizophrenic patients, participated as controls. They were interviewed and reported neither personal nor family history of psychiatric/neurological illness. Demographic and clinical information is summarized in Table 1. All participants had normal or corrected-to-normal vision, were naïve of the purposes of the experiment, and took part on a voluntary basis. The experiment was approved by the local ethics committee.

Stimuli and Apparatus

Three different stimuli were used as cues and presented in three distinct blocks of trials.¹ In the arrow-cue block, the cue was an

arrow ($3.8^\circ \times 1.6^\circ$) oriented leftward or rightward; in the pointing-finger-cue block, the cue was a schematic pointing finger ($3.8^\circ \times 2.7^\circ$) oriented leftward or rightward; in the gaze-cue block, the cue was a schematic face (6° of diameter) with gaze averted leftward or rightward. The regions in each cue that provided spatial information were identical in size ($3.8^\circ \times 1.6^\circ$). The arrow cue was drawn with a symmetric tale and head so as to be comparable with the two eyes of the face cue conveying directional information (also see Galfano et al., 2012). Participants sat approximately 57 cm from a 15-in. laptop monitor (1024×768 pixels, 60 Hz) on which stimuli were presented, using E-prime 1.1, in white against a black background.

Procedure

Each trial began with a fixation cross (1°) visible for 675 ms at the center of the screen, followed by a central cue. After either a 200-ms or 700-ms SOA, a target stimulus represented by a filled circle (1.5°) appeared 9° rightward or leftward with respect to the center of the screen. Two different SOAs were used to investigate the time course of attention shifting elicited by the different cues. Both cue and target remained visible until the participant responded or 3,000 ms were elapsed, whichever came first. The target was spatially congruent or incongruent to cue direction with the same probability. Participants were told that cue direction was uninformative with regard to target location and they were instructed to maintain fixation at the center of the screen throughout each trial. They were asked to detect the target by pressing the space bar with their dominant hand as fast as possible, and to refrain from responding and wait for the next trial when a catch trial was displayed, namely when no target was shown. Catch trials were included to prevent anticipatory responses. The red words "ERROR" and "NO RESPONSE" were presented when participants responded on catch trials (i.e., false alarms) and when they failed to respond (i.e., missed responses), respectively. Finally, a blank screen appeared for 1,000 ms (see Figure 1). Order of blocks was randomized separately for each participant, and cue type was constant within each block. Each experimental block included 96 target-present trials and 24 catch trials and was preceded by a

¹ Cue type was blocked to maximize the likelihood of obtaining significant cuing effects for each cue type. Indeed, previous evidence (Pavan et al., 2011) suggests that gaze cuing might be sensitive to contextual factors, such as the presence of other cuing stimuli within the same block of trials.

Pointing finger				Gaze							
700				200				700			
C		I		C		I		C		I	
RT	RS	RT	RS	RT	RS	RT	RS	RT	RS	RT	RS
713 (372)	0.001881 (0.000627)	748 (322)	0.001684 (0.000528)	654 (216)	0.001841 (0.000517)	657 (208)	0.001832 (0.000443)	635 (198)	0.001889 (0.000498)	641 (202)	0.001868 (0.000494)
500 (183)	0.002293 (0.000583)	522 (183)	0.002189 (0.000553)	533 (205)	0.002186 (0.000552)	532 (209)	0.002175 (0.000535)	531 (229)	0.002235 (0.000625)	545 (194)	0.002139 (0.000539)

practice block containing eight target-present trials and two catch trials. In total, each participant went through 360 experimental trials. The entire session required about 40 minutes.

Results

Data Reduction

False alarms (0.4%), missed responses (1.17%), and anticipations defined as reaction times (RTs) less than 100 ms (0.17%) were removed. Due to the low rate of errors, they were not analyzed further.

Raw data of each participant were then transformed to reciprocals to reduce the influence of extreme RTs and to obtain a more normal distribution of values (Howell, 2010, pp. 340–341; also see Slessor, Phillips, & Bull, 2008). Although analyses were carried out on reciprocal scores, for ease of interpretation, descriptive statistics are reported also as untransformed mean RTs (see Table 2).

Data Analysis

A 2 (cue–target spatial congruency: congruent vs. incongruent) \times 3 (cue type: arrow, pointing finger, gaze) \times 2 (SOA: 200 vs. 700 ms) \times 2 (group: schizophrenic patients vs. healthy controls) mixed-design repeated-measures ANOVA was performed on mean reciprocal scores. Cue–target spatial congruency was significant, $F(1, 34) = 30.685, p < .001, \eta_p^2 = .474$, indicating lower RTs for congruent than for incongruent trials. Group was also significant, $F(1, 34) = 5.392, p = .026, \eta_p^2 = .137$, indicating slower RTs for schizophrenics. Both the Cue–Target Spatial Congruency \times Cue Type interaction, $F(2, 68) = 4.453, p = .015, \eta_p^2 = .116$, and the Cue–Target Spatial Congruency \times SOA interaction, $F(1, 34) = 6.999, p = .012, \eta_p^2 = .171$, were significant. The four-way Cue–Target Spatial Congruency \times Cue Type \times SOA \times Group interaction approached statistical significance, $F(2, 68) = 2.501, p = .09, \eta_p^2 = .069$, which we found even more interesting. No other main effects or interactions were significant. Although the four-way interaction did not reach conventional levels of significance, two separate 2 (cue–target spatial congruency) \times 3 (cue type) \times 2 (SOA) repeated-measures ANOVAs were performed for the two groups, as within-participants designs provide better control of individual differences. For healthy controls, there was only a significant main effect of cue–target spatial congru-

ency, $F(1, 17) = 18.069, p = .001, \eta_p^2 = .515$, indicating lower RTs for congruent relative to incongruent trials. The Cue–Target Spatial Congruency \times Cue Type interaction was not significant, $F(2, 34) = 1.669, p = .203, \eta_p^2 = .089$. Paired two-tailed t tests comparing congruent and incongruent trials confirmed that healthy controls oriented their attention in response to arrow, $t(17) = 2.347, p = .031$, pointing finger, $t(17) = 4.975, p < .001$, and gaze, $t(17) = 2.343, p = .032$, indistinguishably. For schizophrenic patients, there was a significant main effect of cue–target spatial congruency, $F(1, 17) = 13.584, p = .002, \eta_p^2 = .444$, indicating lower RTs for congruent than for incongruent trials, as well as a significant Cue–Target Spatial Congruency \times SOA interaction, $F(1, 17) = 7.498, p = .014, \eta_p^2 = .306$. Paired two-tailed t tests indicated that schizophrenics shifted their attention to the cued location both at the shorter, $t(17) = 1.829, p = .043$, and at the longer, $t(17) = 4.147, p < .001$ SOA, although the effect was stronger in the latter case, suggesting an increased orienting response at longer intervals. It is critical to note, the Cue–Target Spatial Congruency \times Cue Type interaction was also significant, $F(2, 34) = 3.643, p = .037, \eta_p^2 = .176^2$. Paired two-tailed t tests comparing congruent and incongruent trials confirmed that schizophrenics shifted their attention in response to arrows, $t(17) = 2.499, p = .022$, and pointing fingers, $t(17) = 4.298, p < .001$, but not in response to gaze³, $t(17) = .664, p =$

² All the key findings for the purpose of the study remained significant also when additional ANOVAs collapsing across levels of SOA, were performed.

³ In the attempt to obtain further evidence about the lack of gaze cuing among schizophrenic patients, data were also submitted to Bayesian analyses. This approach helps in trying to disentangle which model (null vs. alternative hypothesis) is more strongly supported by the available data and is particularly helpful for dealing with the null hypothesis appropriately. The Bayesian information criterion (BIC) was computed following the procedure proposed by Masson (2011). This analysis showed that the posterior probability favoring the hypothesis that gaze cuing was absent in schizophrenic patients was $p_{\text{BIC}}(H_0 | D) = 0.771$. In contrast, the posterior probability favoring the hypothesis that gaze cuing was absent in healthy controls was $p_{\text{BIC}}(H_0 | D) = 0.255$. Within this framing, BIC values lower than 0.50 indicate that there is more evidence for the alternative than for the null hypothesis, whereas values higher than 0.50 indicate the opposite. According to the conventional categorization of degrees of evidence (see Masson, 2011), the obtained posterior probabilities for the null hypothesis constitute “positive” evidence for the conclusion that no gaze-cuing effect is present in schizophrenics, whereas a real cuing effect is present in the control group.

.52. To control for effects due to medication, if any, three additional analyses of covariance were performed with chlorpromazine equivalent dosage as a covariate. Cuing effects were observed for both arrows and pointing gestures whereas no gaze cuing emerged ($F = .13$). This further supports the conclusion that gaze cuing is impaired among schizophrenics.

Discussion

The purpose of the present study was twofold. First, we aimed to clarify the presence, if any, of abnormal gaze-cuing effects in schizophrenia. In this regard, the available studies in the literature have reported mixed evidence. Langdon et al. (2006) reported consistent gaze-cuing effects for schizophrenic patients using a 100-ms SOA, with performance becoming similar to that of healthy controls at longer SOAs. In contrast, Akiyama et al. (2008) recently reported overall decreased gaze-cuing effects in schizophrenic patients with respect to controls, in the presence of undifferentiated and reliable cuing effects for both groups when arrow cues were used. Our results confirmed the pattern reported by Akiyama et al. (2008), and showed that schizophrenic patients' and healthy controls' performances could be dissociated when considering gaze but not arrow cues. In addition, abnormal processing for gaze stimuli was identified by a reduced gaze-cuing effect in schizophrenics, but not in control participants. Critical factors for accounting for the discrepant results emerging from the current study and those reported by Langdon et al. (2006) may include the use of different types of gaze cues, as well as the duration of illness of the clinical samples that were tested. In this latter regard, hyposensitivity to gaze stimuli seems to become a typical schizophrenic trait only as the course of illness becomes chronic. Whereas the clinical samples included in both the present study and Akiyama et al. (2008) consisted of chronic schizophrenic patients, duration of illness in schizophrenic patients tested by Langdon et al. (2006) was shorter.

The second goal of our study was to address whether abnormal attentional processing of social stimuli was specific to gaze or not. This was tested using a pointing gesture, a social cue other than gaze, that has been shown to elicit robust attention-shifting effects (e.g., Cazzato et al., 2012). In this regard, the present results showed that, similar to healthy controls, schizophrenic patients exhibited a reliable attentional orienting effect moderated by pointing gestures. This finding seems to suggest that the attentional deficit in processing social stimuli among schizophrenic patients is selective for gaze cues.

The latter pattern of results could be explained in terms of the special status of eye gaze, among other social and nonsocial cues, suggested by previous studies. Indeed, there is abundant evidence that prioritized gaze processing can be observed very early in childhood (Farroni et al., 2002) and gaze-cuing effects are even detectable in several animal species other than humans (Shepherd, 2010), highlighting the importance of eye gaze at both an ontogenetic and phylogenetic level. It is important to note that evidence concerning a prioritized response for other social signals, such as pointing gestures, is more scarce and less unequivocal (Shepherd, 2010).

The presence of impaired gaze cuing in schizophrenia could be linked to a dysfunction in the complex neural network regulating social processing that critically involves the superior temporal

sulcus (STS) region. There is abundant neuroimaging evidence showing that the STS region is critically activated during gaze processing in healthy humans (Allison, Puce, & McCarthy, 2000). Although some studies have reported that the STS would also be engaged in processing directional information from other symbolic (Tipper, Handy, Giesbrecht, & Kingstone, 2008) and social (Sato, Kochiyama, Uono, & Yoshikawa, 2009) signals, some studies seem to suggest that involvement of the STS region would be specific to eye gaze (e.g., Kingstone, Tipper, Ristic, & Ngan, 2004). Moreover, Akiyama et al. (2006) have reported a single-case study of a brain-damaged patient with a lesion involving the right STS who showed no gaze-cuing effect whereas arrow-driven orienting was spared. There is evidence, of crucial note, of decreased activity of the STS region in schizophrenic patients (e.g., Rajarethinam, Venkatesh, Peethala, Luan Phan, & Keshavan, 2011). Hence, the observation that schizophrenics exhibit a reduced gaze-cuing response may be related to abnormal functioning in the STS region. Although symbolic (e.g., an arrow) or social signals other than gaze (e.g., a pointing gesture) can call the STS into play to some extent, it has been shown that STS activity is much more pronounced for gaze than for these other signals (e.g., Greene et al., 2011; Sato et al., 2009). Therefore, the results of the present study seem consistent overall with the available neuropsychological data.

In sum, the present findings support the notion that schizophrenics show impaired gaze-cuing effects and are consistent with the observation that gaze-driven IOR is altered in schizophrenic patients (Nestor et al., 2010). In addition, it appears that the observed deficit does not extend to other important social cues such as a pointing gesture. However, future work is needed to establish whether other types of social cuing that were not tested here are impaired in schizophrenia.

One limitation of this study is that we were unable to administer standardized measures of neuropsychological tests. Hence, the possibility of examining the contributions of neuropsychological functioning in schizophrenic patients and its relationship with symptom variables, with regard to attentional response to social versus nonsocial cues, was precluded. Although these issues have been addressed in previous work (e.g., Akiyama et al., 2008; Nestor et al., 2010) further research is needed to fully understand the extent to which abnormal response to gaze stimuli is related to neuropsychological measures and affective symptoms in schizophrenic patients.

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