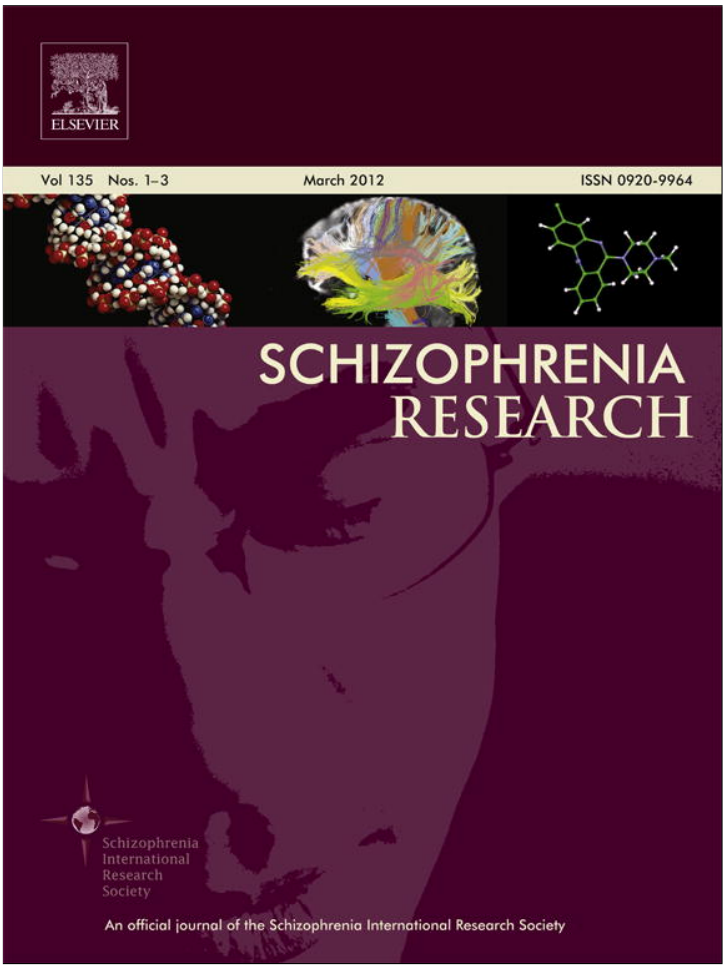

Schizophrenia Research 135 (2012) 55–61

Contents lists available at SciVerse ScienceDirect

Schizophrenia Research

journal homepage: www.elsevier.com/locate/schres

Inhibition of return and schizophrenia: A meta-analysis

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ARTICLE INFO

Article history:
Received 12 October 2011
Received in revised form 29 November 2011
Accepted 30 November 2011
Available online 4 January 2012

Keywords:
Schizophrenia
Inhibition of return
IOR
Orienting
Attention

ABSTRACT

Inhibition of return (IOR) is a phenomenon that involves inhibited or delayed orienting to previously cued locations in favor of attending to novel locations. To date, research on IOR in patients with schizophrenia has generated mixed, and seemingly conflicting, results. Some researchers report patients with schizophrenia exhibit blunted or delayed IOR, while other researchers report normal IOR, in terms of time course and magnitude. This meta-analysis summarizes the literature that has employed an IOR task in patients with schizophrenia and with controls while focusing upon a procedural feature, the use of a cue back to fixation, between the cue and target that is known to be important when executive control has been hampered in non-clinical populations. Fifteen experiments were located yielding a total sample of 362 patients with schizophrenia or schizoaffective disorder and 285 controls. Using a meta-analytic approach, results of the present analyses show that patients with schizophrenia demonstrate delayed IOR in the single cue procedure. In the cue back to fixation procedure, the time course of IOR among patients is more consistent with that of controls. Differences in measured IOR between patients with schizophrenia and controls are largely related to a deficit in endogenous disengagement of attention.

1. Introduction

1.1. Inhibition of return

Human perceptual systems have evolved mechanisms to select important information, while ignoring unimportant or uninformative information. Overt orienting (e.g., eye movements) and covert orienting (i.e., internally mediated shifts of attention) are examples of such mechanisms. Inhibition of return (IOR; Posner et al., 1985), an aftereffect of orienting, discourages orienting to previously inspected objects or locations. In general, in healthy populations, previously presented stimuli initially evoke facilitation which, after a delay, appears to transform into inhibition. In a typical trial of the model task used to explore IOR, a visual cue is presented in the periphery. This is followed, at varying intervals, by a target presented in either the cued location or another location. Depending on the interval between the cue and the target (i.e., the cue-target stimulus onset asynchrony, the SOA), facilitatory or inhibitory effects are reflected in faster or slower reaction time (RT) to stimuli presented in the previously cued locations. In some studies of IOR a second cue at fixation is used to disengage attention from the first cue. We will refer to this as the cue back to fixation procedure and contrast it with the single cue procedure.

IOR has received much attention in the field of cognitive neuropsychology (Lupiáñez et al., 2006). Since its discovery by Posner and Cohen (1984), researchers have learned much about IOR, including information about its time course, spatial coding, cause and effects, and functional significance (Klein, 2000). Researchers have also begun to examine differences in the magnitude and the time course of IOR among individuals with cognitive impairment due to brain damage or disease.

1.2. Inhibition of return and schizophrenia

Cognitive functioning is profoundly affected in individuals with schizophrenia (Antonova et al., 2004). Of the many structural and functional deficits, impairments in attention are paramount (Heinrichs, 2005). Abnormalities in the hippocampal circuitry are implicated in perseverative behavior and the failure to inhibit responses (Antonova et al., 2004), both attention-related phenomena. Dysfunctions in attention and information processing have sparked an interest in treatment options that focus on attentional processes (Tai and Turkington, 2009). For example, meta-cognitive therapy highlights the role that focusing attention on threatening information can have in the development and maintenance of the disorder (Valmaggia et al., 2007).

In an attempt to understand the mechanisms underlying the attentional deficits among patients with schizophrenia, some researchers have investigated the facilitation effects and IOR effects present in the orienting systems. This research is important as cognitive deficits often predict functional outcome (Green et al., 2004) and identifying the mechanisms responsible for attentional deficits could

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doi:10.1016/j.schres.2011.11.034

be potential markers or indicators of risk among high-risk populations (e.g., first degree relatives; Brewer et al., 2005).

To date, research on IOR in patients with schizophrenia has yielded mixed, and seemingly conflicting, results. For instance, Huey and Wexler (1994) and Gouzoulis-Mayfrank et al. (2004) found patients with schizophrenia exhibited blunted IOR when compared to healthy controls. Similarly, Kebir et al. (2010) and Larrison-Faucher et al. (2002) showed delayed onset of IOR in patients when compared to controls. Interestingly, not all research has revealed these differences in magnitude or time course of IOR in patients with schizophrenia. For example, Fuentes and colleagues have reported normal IOR, in terms of time course and size, among patients with schizophrenia (Fuentes & Santiago, 1999; Fuentes et al., 1999). Some researchers suggest that the pattern of results may depend on the task procedures used (Sapir et al., 2001; Nestor et al., 2010), the medication dosage (Gouzoulis-Mayfrank et al., 2007), or the severity/duration of illness (Liu et al., 2010). The present study further investigates claims that task differences have important effects on results.

1.3. The aim and the scope of the review

Klein (2004, 2005) proposed that IOR, which is caused by the cue, is present initially but overshadowed by lingering facilitation at the originally cued location. Only when attention is removed from the cued location (through endogenous control in the absence of a cue back or exogenously by a cue back) will the IOR be apparent. Using data from two studies that explored the time course of facilitation and IOR in patients with schizophrenia (Huey & Wexler, 1994; Sapir et al., 2001), Klein (2005) showed that when the single cue procedure was used, IOR for patients with schizophrenia was delayed relative to normal controls. He also pointed out that this time course discrepancy between patients and controls is reduced when the cue back to fixation procedure is used (though he did not illustrate this point). Klein proposed that smaller or delayed IOR in behavioral studies of patients with schizophrenia may not be evidence for a deficit in exogenous control of attention (of which, IOR is one consequence), but instead, a deficit in endogenous or voluntary control of attention. The present meta-analysis seeks to determine if Klein's (2005) preliminary findings will be replicated when many more studies of individual suffering from schizophrenia are considered. If so, this will provide support for his suggestion that "individuals with underdeveloped or degraded voluntary control over attention should be less likely to, or slower to, disengage attention from an uninformative peripheral cue - unless disengagement is accomplished exogenously via a second cue at fixation" (pp. 57) and will identify individuals with schizophrenia as being so affected.

This meta-analysis will (a) summarize previous investigations of IOR among patients with schizophrenia in order to inform the reader of the state of the current research; (b) identify patterns in the literature; and (c) suggest next steps in moving forward. Building on previous evidence (Klein, 2005), we hypothesized that patients with schizophrenia will exhibit delayed, but not absent, IOR after an uninformative peripheral cue. We hypothesized that the discrepancy in the time course of IOR between patients and controls will be reduced, if not eliminated, in studies using the cue back to fixation procedure.

2. Methods

2.1. Literature search

We searched PsycInfo and Web of Science databases to locate studies on IOR among individuals with schizophrenia. The search was limited to English articles. The databases were searched for all articles that were publish during or before 2010 using the following search criteria: schizo*, inhibition of return or IOR, and orient*. Additionally, we reviewed the reference lists of articles that were suitable for this review to ensure relevant articles were not missed. A study was included if (a) participants were diagnosed with schizophrenia or schizoaffective disorder (only one study included participants with schizoaffective disorder); (b) the study included a control group; and (c) the study used an IOR procedure involving either a single cue or a cue back to fixation, or both. We located 15 experiments, from 13 articles, that employed an IOR task in patients with schizophrenia and a control group (Carter et al., 1994; Huey & Wexler, 1994; Fuentes & Santiago, 1999; Fuentes et al., 1999; Sapir et al., 2001; Larrison-Faucher et al., 2002; Gouzoulis-Mayfrank et al., 2004, 2006, 2007; Moritz & Laudan, 2007; Sapir et al., 2007; Kebir et al., 2010; Liu et al., 2010). Eight studies used the single cue, five studies used the cue back to fixation, and two studies used both the single cue and the cue back to fixation. One study (Moritz & Laudan, 2007) used three types of pictures as cues (anxiety relevant, paranoia-relevant, and neutral). In this review, we consider only the data following (emotionally) neutral cues. Additional information from each study is presented in Table 1.

2.2. Cuing effect size calculation and analysis

Cuing effect sizes were calculated[1] for each combination of SOA and cue type (single cue or cue back) for each study as raw mean differences[2] using the *escalc* function from the *metafor* package (Viechtbauer, 2010) within R version 2.12.1 (R Development Core Team, 2010). These values were calculated by subtracting the mean of the relevant uncued condition from the mean of the corresponding cued condition. As a result, positive values represent facilitation whereas negative values represent IOR. The relevant variances were calculated from the raw data[3], tables or figures in that order of preference. Two studies (Huey and Wexler, 1994; Sapir et al., 2007) failed to report sufficient information to estimate variance so we imputed those values using the pooled variance from the remaining studies. Calculating the mean and variability of the cuing effect (uncued–cued) for any given experiment required us to estimate the correlation between these conditions. None of the included articles reported these correlations. Instead we developed a conservative estimate ($r = .85$) based upon the raw data provided to us by Kebir et al. (2010) as well as data collected within the laboratory of the third author.

Our analysis included 128 cuing effect sizes sampled from 15 experiments reported across the 13 articles summarized in Table 1. Because we were interested in comparing the time course of IOR between patients with schizophrenia and controls, with or without an intervening cue back to fixation, we coded each cuing effect size for group (control, schizophrenia), cue-target stimulus onset-asynchrony (SOA; 66 ms–1500 ms) and cue type (single cue, cue back). At this time a log-transform was applied to SOA to ensure a linear relationship with our dependent measure. These moderator variables were then centered to eliminate the possibility of collinearity (see Card, 2011). Comprehensive notes regarding cuing effect size calculations and coding practices are available from the second author upon request.

Once coding was completed, a mixed-effects model was fitted to the overall data including group, SOA and cue type as moderators. This model was generated by the *rma* function from the *metafor*

[1] Preliminary analyses indicated no consistent effects when comparing left versus right visual fields. Thus, in all subsequent analyses, we collapsed results across visual field.

[2] Adopting raw mean differences as opposed to standardized mean differences was appropriate in this case because (a) reaction time (in milliseconds) served as the dependent measure in each included comparison; and, (b) reaction time is a more meaningful metric than a standardized score which eases the interpretation of our results (see Borenstein et al., 2010).

[3] We thank Dr. Oussama Kebir and Dr. Steffan Moritz for providing data not originally included in their articles.

Table 1

Source	Patient Group	Control Group	Stimuli	IOR procedure	Task	SOAs
Carter et al. (1994)	*n*=23, M:F=19:4	*n*=14, M:F=10:4	Central fixation (X),	Single cue	Detection	100
	Mean age=32.1 (5.3)	Mean age=31.3 (5.5)	two peripheral boxes			800
	BPRS=39.57 (11.98)		Target: asterisk			
	Outpatient					
	Mean duration of illness=11.3 years (7.8)					
	Medication-free for two weeks prior to testing					
Fuentes & Santiago (1999)	*n*=16, M:F=14:2	*n*=16	Three boxes arranged horizontally			
Experiment 1	Mean age=29.25 (6.28)	Age range=21 to 55	Target: word (gato, dedo, vino, rio)	Cue back	Detection	950
	Age range=19 to 40				Words	1250
	Inpatient					
	Mean duration of illness=10.38 (5.98) years					
	Medicated at time of testing					
Fuentes et al. (1999)	*n*=14	*n*=14	Three boxes arranged horizontally	Single cue	Detection	200
			Target: asterisk			1200
	Median age=34	Median age=26.5		Cue back	Detection	1200
	Age range=20 to 55	Age range=23 to 42				
	Outpatient					
	Medicated at time of testing					
Gouzoulis-Mayfrank et al. (2004)	*n*=40, M:F=22:18	*n*=34; M:F=20:14	Central fixation (+),	Single cue	Detection	100
	Mean age=31.60 (7.99)	Mean age=30.85 (4.79)	two peripheral boxes			800
	BPRS at initial assessment=41.85 (8.77)		Target: star			
	SAPS at initial assessment=39.33 (19.95)					
	SANS at initial assessment=32.50 (23.79)					
	BPRS at follow-up=28.55 (7.4)					
	SAPS at follow-up=5.12 (7.65)					
	SANS at follow-up=28.74 (20.01)					
	Inpatient at first assessment; outpatient at follow-up					
	Mean duration of illness=4.62 (4.47) years					
	Medicated at time of testing					
Gouzoulis-Mayfrank et al. (2006)	*n*=32, M:F=26:6	*n*=16; M:F=10:6	Central fixation (+),	Single cue	Detection	100
	Mean age=31.20 (9.00)	Mean age=28.50 (3.1)	two peripheral boxes			800
	SAPS=21.40 (15.25)		Target: star			
	SANS=34.47 (25.04)					
	Inpatient					
	Mean duration of illness=4.76 (4.90) years					
	All but three medicated at time of testing					
Gouzoulis-Mayfrank et al. (2007)	*n*=15, M:F=11:4	*n*=25; M:F=15:10	Three boxes arranged horizontally	Single cue	Detection	100
			Target: star			800
	Mean age=31.20 (11.1)	Mean age=26.60 (3.3)		Cue back	Detection	800
	SAPS=48.33 (33.09)					
	SANS=19.22 (26.16)					
	Inpatient					
	Duration of illness=3 to 20 years for 7 patients; remaining 8 patients experiencing first-episode of psychosis					
	Medication-free for two weeks prior to testing					
Huey & Wexler (1994)	*n*=11, M:F=5:6	*n*=11; M:F=6:5	Four boxes forming the corners of a square, fifth box in the center	Single cue	Detection	100
	Median age=51	Median age=30	Target: asterisk			200
	Age range=22 to 66	Age range=20 to 52				700
	Outpatient					1200
	Medicated at time of testing					
Kebir et al. (2010)	*n*=14, M:F=13:1	*n*=16; M:F=15:1	Three boxes arranged horizontally with fixation (+) in center box	Single cue	Detection	200
	Mean age=26.3 (5.8)	Mean age=25.0 (5.8)	Target: star			300
	PANSS total score=53.1 (9.5)					400
	PANSS positive symptoms scale=9.5 (2.2)					500
	PANSS negative symptoms scale=17.4 (5.5)					600
	Mean duration of illness=2.84 years					700
	Medicated at time of testing					800
	Mean chlorpromazine equivalent of antipsychotic dosage=482.5 mg					900
						1000
						1100

(continued on next page)

Table 1 (*continued*)

Source	Patient Group	Control Group	Stimuli	IOR procedure	Task	SOAs
Larrison-Laucher et al. (2002)	$n=14$, M:F=11:3 Mean age=38.0 (8.0) Outpatient Mean ($n=11$) duration of illness=16.27 (8.01) years Medicated at time of testing	$n=14$; M:F=9:5 Mean age=36.2 (6.8)	Central fixation point, two peripheral boxes Target: box filled in with grey	Cue back	Localize via eye movement	66 79 106 133 159 226 305 505 705 1000
Liu et al. (2010)	First episode: $n=42$, M:F=28:14 Mean age=26.6 (6.5) PANSS total score=67.0 (12.3) PANSS positive symptoms scale=21.9 (6.1) PANSS negative symptoms scale=11.4 (6.0) Mean duration of illness=7.5 (6.3) months Medication-free at time of testing Chronic: $n=44$, M:F=21:23 Mean age=31.4 (7.0) PANSS total score=69.4 (10.7) PANSS positive symptoms scale=20.3 (5.0) PANSS negative symptoms scale=14.7 (5.5) Mean duration of illness=99.2 (49.7) months Medication-free at time of testing	$n=38$; M:F=17:21 Mean age=29.0 (8.0)	Three boxes arranged horizontally with fixation (+) in center box Target: solid circle in box	Cue back	Detection	400 500 600 700 1200 1500
Moritz & Laudan (2007)	$n=24$, M:F=16:8 Mean age=35.21 (11.29) Inpatient Medicated at time of testing Mean chlorpromazine equivalent of antipsychotic dosage=593.87 (502.35) mg	$n=34$; M:F=18:16 Mean age=35.85 (13.23)	Central fixation point, two peripheral boxes Target: black dot	Single cue	Localize via manual response	450 1100
Sapir et al. (2001) Experiment 1	$n=17$, M:F=11:6 Mean age=41.5 (14.0) Mean duration of illness=14.82 (10.74) Medicated at time of testing Mean chlorpromazine equivalent of antipsychotic dosage=460.59 (233.49) mg	$n=17$; M:F=8:9 Mean age=35.4 (12.6)	Three boxes arranged horizontally with fixation (+) in center box Target: asterisk	Single cue	Detection	100 300 1000
Sapir et al. (2001) Experiment 2	$n=16$, M:F=12:4 Mean age=33.7 (9.8) Mean duration of illness=11.09 (8.35) years Medicated at time of testing Mean ($n=14$) chlorpromazine equivalent of antipsychotic dosage=525.0 (239.19) mg	$n=16$; M:F=10:6 Mean age=35.9 (11.2)	Three boxes arranged horizontally with fixation (+) in center box Target: asterisk	Cue back	Detection	350 700 1200
Sapir et al. (2007) Experiment 1	Pre-medication: $n=10$, M:F=8:2 Mean age=41.1 (11.8) Post-medication: $n=10$, M:F=7:3 Mean age = 32.5 (4.4) Half ($n=10$) tested prior to medication delivery, half tested after medication delivery ($n=10$)	$n=10$ Mean age=46.0 (9.8)	Three boxes arranged horizontally with fixation (+) in center box Target: asterisk	Single cue	Detection	100 350 700 1200
Sapir et al. (2007) Experiment 2	Pre-medication: $n=10$, M:F=7:3 Mean age=36.8 (10.3) Post-medication: $n=10$, M:F=7:3 Mean age=36.4 (10.8) Half ($n=10$) tested prior to medication delivery, half tested after medication delivery ($n=10$)	$n=10$ Mean age=44.9 (10.5)	Three boxes arranged horizontally with fixation (+) in center box Target: asterisk	Cue back	Detection	350 700 900 1200

Note: M:F=Male to female ratio. BPRS=Brief Psychiatric Rating Scale (Overall and Gorham, 1976). SAPS=Scale for the Assessment of Positive Symptoms (Andreasen, 1984a). SANS=Scale for the Assessment of Negative Symptoms (Andreasen, 1984b). PANSS=Positive and Negative Symptoms Scale (Kay et al., 1987, 1989). Mean age and age range reported in years. Standard deviation reported in brackets when available.

package using a restricted maximum-likelihood estimator (see Viechtbauer, 2010). Cuing effect sizes within this model were weighted using the inverse of the relevant sampling variance.

3. Results

Our analysis revealed a significant main effect of group, $b = 16.57$, $CI_{95\%} = [9.40, 23.73]$, cue type $b = 14.36$ $CI_{95\%} = [7.11, 21.61]$ and log-transformed SOA, $b = -0.06$, $CI_{95\%} = [-0.07, -0.05]$. In summary, there was more IOR for controls compared to patients with schizophrenia and for experiments containing a cue back compared to those containing only a single cue. Moreover, effect sizes became more negative (revealing greater IOR) as SOA increased. More importantly, the group by cue type interaction also reached significance, $b = 15.13$, $CI_{95\%} = [0.63, 29.64]$. No other interactions were significant.[4]

To explore the nature of the cue type by group interaction, separate models were developed for the single cue and cue back conditions. For the single cue condition there were main effects of group, $b = 23.10$, $CI_{95\%} = [14.40, 31.79]$, and SOA, $b = -0.06$, $CI_{95\%} = [-0.07, -0.05]$, but no interaction, $b = 0.00$, $CI_{95\%} = [-0.02, 0.02]$. For the cue back condition, only the main effect of SOA reached significance, $b = -0.05$, $CI_{95\%} = [-0.07, -0.04]$. Neither the main effect of group, $b = 8.09$, $CI_{95\%} = [-3.62, 19.79]$, nor the group by SOA interaction, $b = -0.01$, $CI_{95\%} = [-0.04, 0.02]$, reached significance. This interaction is depicted in Fig. 1. As a means of quantifying the point at which the time course crossed the x-axis for each combination of group and cue type, we applied inverse regression to calculate a 95% fiducial interval for the x-intercept (see Draper & Smith, 1998, pp. 83–86; for a theoretical discussion, see Williams, 1959). The fiducial intervals are regarded as inverse confidence intervals for X given a Y value of 0 and are defined as the point at which the regression line crosses 0 on the X-axis bounded by the points at which the relevant confidence bands cross the X-axis. After reversing the log-transform of SOA to reveal the time course of IOR across natural time, fiducial intervals for the single cue condition indicate an earlier crossover from facilitation to inhibition for controls, $X = 293$ ms, $CI_{95\%} = [238, 350]$, compared to patients, $X = 758$ ms, $CI_{95\%} = [536, 1290]$. The presence of a cue back appears to equate the controls, $X = 241$ ms, $CI_{95\%} = [184, 297]$, and patients, $X = 313$ ms, $CI_{95\%} = [256, 373]$. These values can be visualized in Fig. 2.

4. Discussion

In this study, we hypothesized that patients with schizophrenia would exhibit delayed, but not absent, IOR; and the discrepancy in time course of IOR between patients and controls would be reduced, if not eliminated, when using the cue back to fixation procedure.

For patients with schizophrenia, results of this meta-analysis are consistent with preliminary evidence provided in Klein (2005). As Klein suggested, we can further attest that individuals with abnormalities in voluntary attention (such as patients with schizophrenia) are slower to disengage attention from a cue, unless a cue back to fixation is used to disengage attention from the previously viewed location. Results from the present study provide compelling evidence that differences in IOR from prior studies of patients with schizophrenia and controls are largely related to the IOR procedure used. Some studies included in this review have put forward ideas consistent with conclusions made here (Fuentes et al., 1999; Sapir et al., 2007; Kebir et al., 2010). In contrast, other studies (e.g., Gouzoulis-Mayfrank et al., 2004, 2006; Kebir et al., 2008) suggest patients with schizophrenia exhibit true abnormalities in IOR. In light of evidence presented in the present study, caution should be exercised when interpreting results suggesting abnormalities in IOR exist, especially in studies using single cue procedures.

For controls, results are consistent with prior suggestions that normal adults do not require the cue back to fixation to disengage their attention from the previously cued location (Klein, 2005). In fact, the equal probability of receiving a target at various locations provides enough motivation for healthy adults to place their attention in an unbiased state after each cue (Lupiáñez et al., 2006). Despite this motivation, attention is returned to "neutral" more rapidly in the control participants when there is a cue back. It is likely that this difference simply reflects the fact that exogenous orienting is more rapid than endogenous orienting (Lupiáñez et al., 2006).

Future researchers examining the time course and magnitude of IOR among patients with schizophrenia or researchers interested in identifying cognitive deficits as predictors of functional outcomes or as markers of risk should be aware that group differences in measured IOR (particularly in the absence of a cue back to fixation) might not reflect differences in IOR per se. These group differences may be more related to the efficiency or rate with which patients with schizophrenia voluntarily (endogenously) disengage their attention from the cued location back to neutral prior to the appearance of the upcoming target (Klein, 2005). In individuals with poor endogenous control over attentional processes, such as patients with schizophrenia, the endogenous disengagement of attention from a cue may be delayed. Thus, in their day-to-day lives, individuals with schizophrenia are likely to perseverate on irrelevant details of their world, potentially perpetuating their symptoms (Antonova et al., 2004). The large reduction in the time course difference between patients with schizophrenia and controls when a cue back to fixation procedure was used provides converging evidence for this view. As suggested by Klein (2005) and Klein et al., 2006, the cue back returns the attention to fixation under exogenous control. When the interest is in IOR, researchers should use this cue back procedure so as to avoid the possibility that lingering facilitation due to the slow disengagement of attention might overshadow IOR.

Although the results of the present meta-analysis will aid future understanding of IOR among patients with schizophrenia, one possible limitation should be noted. Since not all studies included in this review reported on medication dosage, severity of illness, age of illness onset, or visual field, we could not include these factors in our analyses. Our main goal was to determine if IOR procedure (single cue or cue back to fixation) provides explanatory evidence for the differences in results among prior studies. We have achieved this goal. However, as more data emerges, it would be useful to supplement these analyses with data on medication dosage, severity of illness, age of illness onset, or visual field.

Role of funding source

Aislin R. Mushquash was supported by a Canada Graduate Scholarship from the Social Sciences and Humanities Research Council. Raymond M. Klein was supported by a Natural Sciences and Engineering Council Discovery Grant.

Contributors

This manuscript was based on a project conducted by Aislin R. Mushquash as part of her Ph.D. comprehensive requirement. Aislin R. Mushquash was supervised by Raymond M. Klein. Aislin R. Mushquash was responsible for conducting the literature searches, data management, and preliminary data analyses. Jonathan M. Fawcett contributed significantly to final data analyses. Aislin R. Mushquash received guidance from Raymond M. Klein during the statistical analyses, interpretation of the results, and manuscript preparation and revisions. All authors have contributed to and have approved the final manuscript.

[4] Our model accounts for 72.87% of the heterogeneity within the observed effect sizes. Even so, a significant degree of heterogeneity remains across our measures, $Q(120) = 338.58$, $p < .01$.

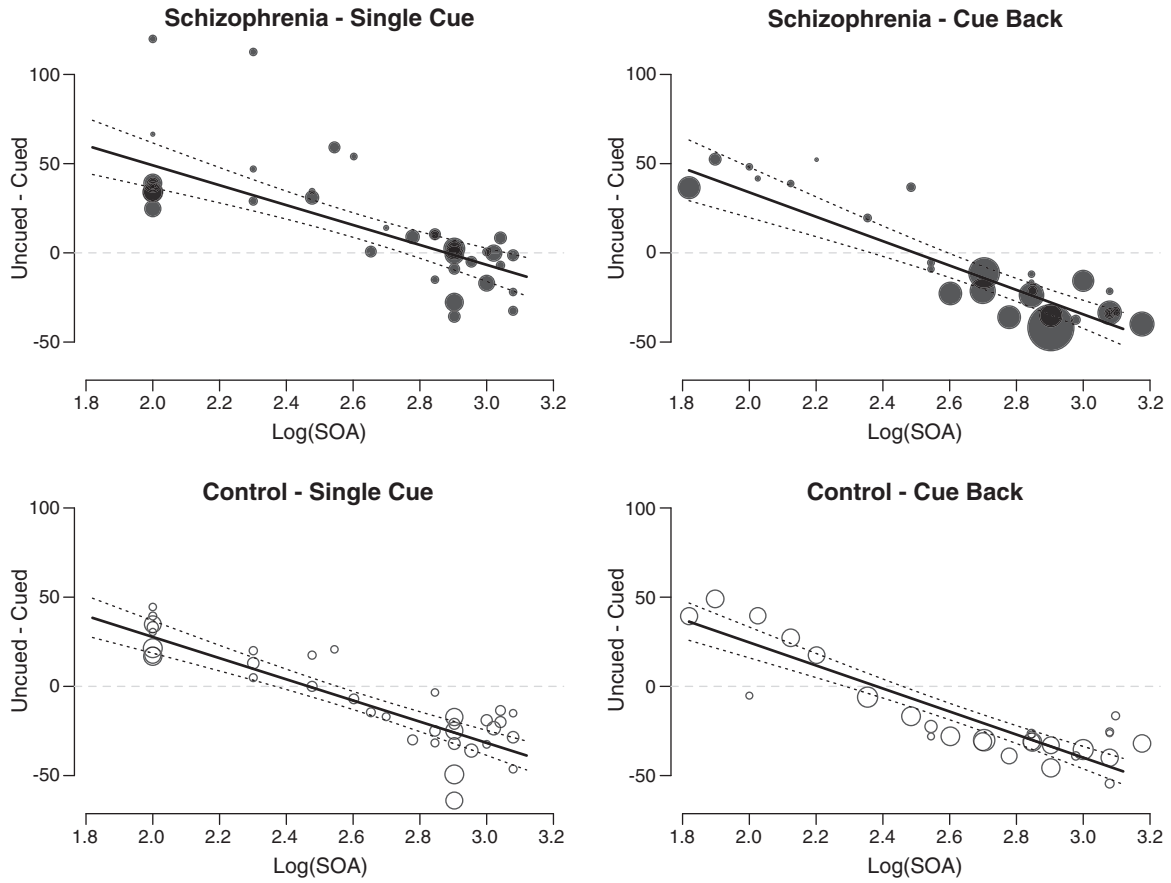

Fig. 1. The magnitude of the cuing effect (in milliseconds) as a function of the log-transformed SOA plotted separately by group (control, schizophrenia) and cue type (single cue, cue back). Solid lines represent the predicted values derived from our meta-analytical model. Dotted lines represent 95% confidence bands. The weight of each point within our model is represented graphically as the absolute size of that point in the graph.

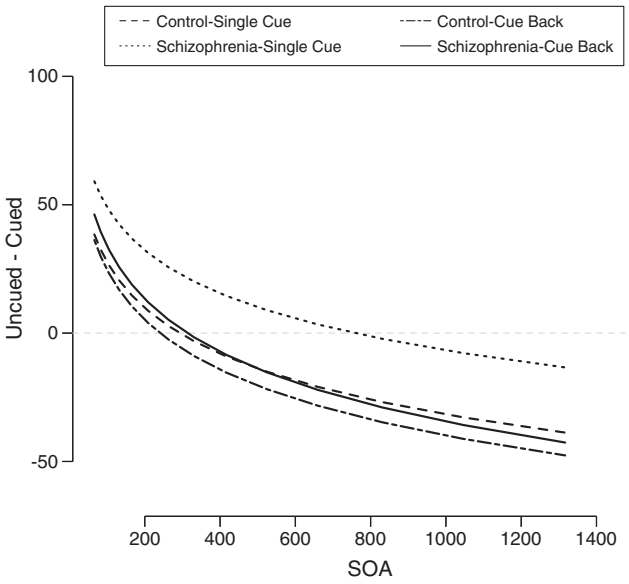

Fig. 2. The magnitude of the cuing effect (in milliseconds) as a function of the SOA (in milliseconds) plotted separately by group (control, schizophrenia) and cue type (single cue, cue back). Solid lines represent the predicted values derived from our meta-analytical model.

Conflict of interest

Aislin R. Mushquash, Jonathan M. Fawcett, and Dr. R. Klein declare that they have no conflicts of interest.

Acknowledgments

The authors wish to acknowledge John Christie for consulting on the statistical analyses.

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