Abnormal Orienting in Schizotypal Personality Disorder

by Adrian Raine, Deana Benishay, Todd Lencz, and Angela Scarpa

Abstract

Previous studies have found evidence for skin conductance (SC) orienting abnormalities in psychosis-prone subjects, but there have been no previous studies on subjects with a diagnosis of schizotypal personality disorder. This study assesses whether clinical schizotypal subjects show abnormal habituation to orienting stimuli. Thirteen subjects with both high scores on the Schizotypal Personality Questionnaire (SPQ) and a DSM-III-R clinical diagnosis of schizotypal personality disorder were compared to 30 controls with no such diagnosis and with low scores on the SPQ. While normals showed the expected habituation in SC orienting across trials, schizotypal subjects failed to show a decrement in responding across the first three trials. In a second study on 30 new subjects, individual differences in schizotypy correlated significantly (p = 0.47) and in the predicted direction with a dimensional measure of the orienting deficit. It is hypothesized that this retarded habituation in schizotypals reflects a deficit in preattentive template matching, which may in turn partly relate to the working memory and prefrontal deficits observed in schizotypal and schizophrenia patients.


Psychophysiological studies have shown that schizophrenia subjects, relatives of schizophrenia patients, and schizotypal subjects share similar (though not always identical) information-processing deficits (e.g., Cannon et al. 1990; Kendler et al. 1991; Cadenhead et al. 1993; Perry and Braff 1993; Thaker et al. 1993; Holzman et al. 1995). In particular, a dysfunction of attention as indexed by skin conductance (SC) orienting is a frequently reported finding in the psychophysiological study of schizophrenia; sensitization, an erratic pattern of orienting, failure to habituate, and hyporesponsivity have all been reported (Ohman 1981; Dawson and Nuechterlein 1984; Iacono 1985; Zahn 1988; Gruzelier and Raine 1994). Experimental studies have indicated that the amplitude and frequency of the SC response is a sensitive measure of information processing, reflecting the allocation of attentional resources for the processing of the eliciting stimulus and the process of template matching (Ohman 1986; Dawson et al. 1989).

Studies of psychosis-prone subjects have attempted to assess whether the same orienting abnormalities found in schizophrenia patients can be observed in nonhospitalized, ostensibly normal individuals who are at putative risk for psychosis (Venables 1993). Nine such studies have been conducted (see Raine et al. 1995 for a detailed review), all on undergraduate samples using self-report measures of both positive schizotypal symptoms (usually perceptual aberration and magical ideation) and negative aspects of psychosis-proneness (social and physical anhedonia). Although all nine studies find significant effects, findings are diverse, and there are failures to replicate (Venables 1993; Raine et al. 1995). One potentially important methodological issue in this literature is that most of these studies on schizotypy do not report SC orienting data on a trial-by-trial basis, but instead use composite indices that average across trials. This averaging procedure may wash out transient but real attentional deficits in schizotypal subjects that are not as severe as those observed in schizophrenia patients. One study by Wilkins (unpublished manuscript, 1988), which did break data down on a trial-by-trial basis, found an increase in SC orienting from trial 1 to trial 2 for high-scoring anhedonia subjects (group X trials interaction, p < 0.003). This suggests that when data are broken down by trials, schizotypy is associated with retarded habituation.

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Another important question in this field concerns whether orienting abnormalities found in psychosis-prone subjects can also be found in subjects with a full DSM-III-R (American Psychiatric Association 1987) diagnosis of schizotypal personality disorder. There is evidence that schizotypal personality disorder is genetically related to schizophrenia (Torgersen 1985; Siever et al. 1990), and research on this disorder has the potential to help clarify our understanding of the deficits that are central to the pathophysiology of schizophrenia. Hence, such deficits may be candidates for markers of schizophrenia (Iacono 1985; Raine and Lencz 1995). There have been no previous studies of SC orienting in schizotypal personality disorder as defined by DSM-III-R.

The two studies below attempt to address this gap in the literature by assessing whether diagnosed schizotypal subjects are characterized by SC orienting abnormalities. Following from Wilkins (unpublished manuscript, 1988), it was predicted that a trial-by-trial analysis of the data would reveal a significant group × trials interaction in the direction of clinically diagnosed schizotypal subjects showing more retarded habituation. In the second study, it was further predicted that retarded habituation would be significantly related to schizotypy using a dimensional design with unselected subjects.

Methods

Subjects. There were 13 subjects with schizotypal personality disorder (9 male, 4 female) and 30 controls (14 male, 16 female). Among the schizotypal patients were 5 Caucasians, 6 Asians, and 2 Hispanics; among the controls, there were 20 Caucasians, 6 Asians, 2 Hispanics and 2 African-Americans. Subjects had been selected from a pool of undergraduate students enrolled in Introduction to Psychology at the University of Southern California (see below). Informed consent was obtained from all subjects who participated in the experiment for course credit. There were no significant group differences between schizotypal subjects and controls on age (schizotypal subjects: mean = 19.0, standard deviation [SD] = 0.9; controls: mean = 19.9, SD = 2.9; p > 0.34) or IQ as estimated by an average scaled score from four subscales (digit span, arithmetic, block design, and digit symbol) of the Wechsler Adult Intelligence Scale–Revised (WAIS–R; Wechsler 1981) (schizotypal subjects: mean = 11.4, SD = 1.7; controls: mean = 11.6, SD = 1.8; p > 0.71).

Initial Subject Selection on Schizotypal Personality Questionnaire (SPQ). Subjects were administered the SPQ (Raine 1991). Subjects scoring in the top 10 percent and bottom 10 percent of the distribution of scores on the SPQ (from a total pool of several hundred subjects) were invited back for psychophysiological testing. The high and low cutoff scores on the SPQ were 41 and 12, respectively.

Full reliability and validity data for the SPQ, as well as comparisons with other schizotypy scales, are provided in Raine (1991) and Chapman et al. (1995). This 74-item self-report questionnaire takes approximately 10 minutes to complete and has been found to have high internal reliability as indicated by Cronbach’s alpha (0.91). Convergent validity, as assessed by Pearson correlations between the SPQ and other measures of schizotypal personality, ranges from 0.59 to 0.81 (Raine 1991). Discriminant validity was demonstrated by the fact that correlations between the SPQ and scales measuring nonschizotypal forms of abnormal personality were significantly lower than correlations between the SPQ and other scales of schizotypal personality. Test-retest reliability (Pearson’s r) is 0.82. Criterion validity for the SPQ is indicated by a 0.60 correlation (point-biserial) between SPQ scores and a clinical diagnosis of DSM–III–R schizotypal personality disorder and by a 0.68 correlation (Spearman’s r) between the SPQ and dimensional scores of schizotypal personality disorder derived from diagnostic interviews. The internal reliabilities of the nine SPQ subscales range from 0.71 to 0.78 (Cronbach’s α), with a mean of 0.74.

Diagnostic Interview for Schizotypal Personality Disorder. Subject volunteers from the top and bottom 10 percent were administered the Structured Clinical Interview for DSM–III–R personality disorders (SCID–II; Spitzer et al. 1987). No subject who volunteered for the study refused to be interviewed. Presence of each schizotypal trait is assessed on a 3-point scale (1 = absent, 2 = subthreshold, 3 = threshold). Subjects had to have five traits out of nine at threshold level for a DSM–III–R diagnosis of schizotypal personality disorder. Interviews were videotaped and scored by two research assistants who independently rated the subject to obtain consensus ratings (coefficient κ = 0.89). Interviewers were blind to group membership and each other’s assessment at the time ratings were conducted. Interviews took place on average 2 months following administration of the SPQ (range = 1–3 months).

As indicated above, 13 individuals scoring in the top 10 percent on the SPQ (out of a total of 22 interviewees) received a DSM–III–R diagnosis of schizotypal personality disorder. None of the 30 subjects taken from the bottom 10 percent received the diagnosis, and these subjects formed the control group.

SC Orienting. Orienting stimuli consisted of a series of six tones with an intensity of 60 decibels sound pressure
controls showed normal habituation. The interaction was
interaction (F = 2.6; df = 5,37; p < 0.05) revealed that only the
habituation. However, a significant group X trials interac-
tion of the first stimulus were also recorded. The criterion
above) was scored, while SC levels before the presenta-
tion of the first stimulus were also recorded. The criterion
to define SC nonresponse consisted of a failure to respond
to the first three orienting stimuli. Charts were scored
blind to group membership. Amplitudes were transformed
using a square root transformation to reduce skew and kur-
tosis as recommended by Venables and Christie (1980).

Results

Group Differences for Clinical Schizotypal Subjects
Versus Controls.

Orienting stimuli. SC amplitudes across the six
orienting trials for schizotypal subjects and controls are
shown in figure 1. A repeated measures multivariate
analysis of variance (MANOVA) (using the multivariate
output from the MANOVA procedure; Vasey and Thayer
1987) indicated a trend toward a main effect for trial (F =
2.2; df = 5,37; p < 0.08), which suggests that the expected
reduction in SC amplitude across trials is a function of
habituation. However, a significant group × trials interac-
tion (F = 2.6; df = 5,37; p < 0.05) revealed that only the
controls showed normal habituation. The interaction was
broken down by within- and between-group t tests using
Rom’s sequentially rejective procedure (Rom 1990;
Wilcox 1994) to control for Type I error. Paired t tests
revealed that, while the normals showed a significant
reduction in amplitude from trial 1 to trial 3 (t = 3.5, p <
0.002), the schizotypal subjects failed to show this
decrease. Although figure 1 suggests that schizotypal sub-
jects show an actual increase in response from trial 1 to
trial 3, this trend was nonsignificant. Between-group
analyses also using Rom’s procedure indicated that
schizotypal subjects showed significantly higher ampli-
tudes on trial 3 relative to controls (t = 2.7, p < 0.01); no
other comparisons were significant after alpha correction.

To provide a dimensional index of the direction and
to the extent to which subjects showed either a normal reduction
in SC orienting from trial 1 to trial 3 or a failure to reduce,
the amplitudes of the trial 1 response were subtracted
from the amplitudes of the trial 3 response. On this index,
low (negative) values indicate normal habituation while
high (zero or positive) values indicate retarded orienting.
A t test comparison (t = 2.0, p < 0.05, two-tailed) indi-
cated that the schizotypal subjects showed more abnormal
orienting with amplitude increasing across trials (mean =
0.12, SD = 0.36), as compared to controls who showed
the expected decrease over trials (mean = −0.22, SD =
0.55).

Effects of gender and ethnicity. Because groups
were unbalanced for gender, gender was entered as a fac-
tor and the above MANOVA repeated. The previously sig-
nificant group × trials interaction remained significant
(F = 3.11; df = 5.35; p < 0.02). The main effect for gender
was nonsignificant (p > 0.27), as were all interactions.
involved in gender ($p > 0.43$), indicating that the above findings were not an artifact of the excess of males in the schizotypal group.

To provide a general analysis of any ethnicity effects, subjects were divided into groups of whites and non-whites. The group $\times$ trials interaction remains significant in this analysis ($F = 2.8; df = 5.35; p < 0.03$). There were no main effects of ethnicity, nor were there any significant interactions involving ethnicity ($p > 0.10$).

**Arousal and Other Orienting Measures.** Comparisons of $t$ tests indicated that control and schizotypal subjects did not differ on SC levels (controls: mean = 1.11, SD = 0.52; schizotypal subjects: mean = 1.00, SD = 0.60; $p > 0.60$) or on spontaneous SCRs during the rest period (controls: mean = 4.7, SD = 5.1; schizotypal subjects: mean = 4.4, SD = 5.2; $p > 0.85$).

Controls and schizotypal subjects did not differ on frequency of SC orienting responses to the six-tone series (controls: mean = 1.4, SD = 1.6; schizotypal subjects: mean = 1.8, SD = 1.7; $p > 0.45$). Furthermore, there was no significant difference ($F^2 = 0.2, p > 0.66$) in overall rates of SC nonresponse in schizotypal subjects (30%) and controls (43%).

**Individual Differences in Unselected Subjects.** We attempted to extend the above finding of an orienting abnormality in clinical schizotypal subjects by taking a dimensional (as opposed to categorical) conceptualization of schizotypy using a dimensional design on a new sample of subjects. This design has the advantages of (1) assessing generalization of the previous findings from a categorical to a dimensional conceptualization of schizotypy (Lenz and Raine 1995) and (2) allowing an assessment of the links between abnormal orienting and three factors of schizotypal traits.

**Subjects and Method.** Subjects consisted of 30 volunteers (12 male, 18 female; mean age = 20.5 years) from a new sample of undergraduate students enrolled in Introduction to Psychology at the University of Southern California. Subjects volunteered in the experiment for course credit and were not selected or screened using any other criteria. All subjects were administered the SPQ, providing both a total score and subscores on three main factors of schizotypy. These subscores were derived from a confirmatory factor analysis of the nine SPQ subscales, which yielded factors labeled “cognitive-perceptual” (unusual perceptual experiences, magical thinking, ideas of reference, and paranoid ideation subscales), “interpersonal deficits” (constricted affect, no close friends, and social anxiety subscales), and “disorganization” (odd speech and odd behavior) (see Raine et al. 1994 for full details). All subjects received the same SC orienting paradigm as detailed above. As described previously, amplitude of the trial 1 response was subtracted from the amplitude of the trial 3 response to create a difference score for each subject.

**Results.** Spearman correlations between this difference score and SPQ total score and three subscores were then computed. Total SPQ scores were significantly correlated with the difference score ($r = 0.47, p < 0.004$) in the expected direction of more abnormal orienting (i.e., less habituation from trial 1 to trial 3) in those with high SPQ scores. Using a Bonferroni-corrected alpha of 0.017, significant correlations were also observed for the cognitive-perceptual subscale ($r = 0.51, p < 0.002$) and interpersonal deficits subscale ($r = 0.48, p < 0.004$), but not for the disorganized subscale ($r = 0.02, p > 0.45$). A scattergram was drawn up relating the SC difference score to SPQ scores; no evidence was found for a nonmonotonic relationship between the two variables.

The above findings do not appear to be accounted for by a relationship between SC nonresponse and high SPQ scores. Subjects who were SC nonresponders ($n = 9, 29\%$ of the sample) did not differ from responders ($n = 21$) in terms of SPQ total scores ($p > 0.12$) or the three subscale scores ($p > 0.11$).

Subjects were divided into top and bottom thirds on the total SPQ score (above 30 and below 21) to form high-scoring ($n = 10$) and low-scoring ($n = 10$) SPQ subgroups. A repeated measures MANOVA performed on the first three trials again confirmed a significant group $\times$ trials interaction ($F = 3.7; df = 2.17; p < 0.05$), with the low SPQ group showing decreasing amplitudes across trials and the high SPQ group showing increasing amplitudes across trials.

**Discussion**

The above results indicate that schizotypal subjects, diagnosed according to *DSM-III-R*, are characterized by abnormal SC orienting. Although this abnormality bears similarity to sensitization, this interpretation of the data seems less likely since the orienting tones were of low intensity. Consequently, the data seem best interpreted in terms of showing retarded habituation in schizotypal subjects. This orienting abnormality in diagnosed schizotypal subjects was again found in a second study using a dimensional design on a nonclinical sample, suggesting that the same significant findings on the clinical sample in this study and on the psychosis-prone sample of the independent study of Wilkins (unpublished manuscript, 1988) are not chance findings, and that they generalize to a dimensional conceptualization of schizotypal personality. These findings must be treated as initial and viewed with caution.
because the sample size is relatively small and because previous findings have been diverse (Venables 1993; Raine et al. 1995). Furthermore, it is clear that not all schizotypal individuals show retarded habituation. Nevertheless, findings are thought to be of potential significance because they are found in both samples and because this is the first investigation of an SC orienting abnormality in diagnosed clinical schizotypal patients who are difficult to recruit in noninstitutionalized settings. Such findings may be viewed as consistent with reports that younger, acute, unmedicated schizophrenia patients also show habituation deficits (Dawson et al. 1992b), a group that seems similar to the young unmedicated clinical schizotypal group in the current study.

An important question that needs to be addressed concerns what attentional process this deficit reflects. According to one influential information-processing model of SC orienting developed by Ohman (1979, 1986), orienting occurs when preattentive processes make a "call" for additional controlled processing. In this model, orienting reflects a change from automatic to controlled processing as a result of initial preattentive processing of the novelty and meaning of the stimulus. The normal habituation shown by controls occurs when a neural template, built up from the first presented stimulus and held in short-term store during a delay (in this study over a 35–50-second interstimulus interval), is compared with a second stimulus and a match is obtained (Siddle 1991). However, degradation in the accurate representation of the neural trace of the first stimulus (e.g., due to a working memory deficit) would result in a mismatch with the second stimulus and produce a call for processing resources to further analyze the second stimulus, producing an orienting response (Ohman 1979). Such a process could at least in part account for the failure of schizotypal subjects to show the expected decrement in amplitude across the first three trials.

The notion that this orienting abnormality of retarded habituation in schizotypal subjects may reflect a deficit in template matching, which may relate to a preattentive form of working memory, is also consistent with recent observations that schizotypy patients are characterized by working memory deficits (Holzman et al. 1995). These findings are also consonant with prior research demonstrating working memory deficits in schizophrenia patients and their relatives (Park and Holzman 1992, 1993; Park et al. 1993). Current findings and interpretations are also consistent with findings using event-related potential measures of memory templates in psychosis-prone subjects (Miller 1986), with short-term memory deficits in poor premorbid schizophrenia patients (Knight 1992), and with findings of impaired speed of information transfer from short-term visual store into short-term memory in schizotypal subjects (Merritt et al. 1986). Nevertheless, measures of what is termed "working memory" vary widely across studies, and the type of preattentive processing deficit tapped by SC orienting in this study may or may not relate to working memory. Studies that assess several measures of working memory deficits in the same sample of schizotypal subjects could better address the hypothesis that schizotypal individuals have a deficit in preattentive template matching.

At a neuroanatomical level, abnormalities in SC orienting have been related to prefrontal structural and functional deficits in schizophrenia patients, normals, and neurological patients (Tranel and Damasio 1994; Raine et al. 1991; Hazlett et al. 1993). It is possible, therefore, that SC orienting abnormalities may stem from the type of structural and functional prefrontal deficits observed in schizophrenia patients and schizotypal patients (Buchsbaum et al. 1990; Braff et al. 1991; Andreasen et al. 1992). It must be borne in mind, however, that prefrontal structural deficits are more clearly linked to reduced SC orienting than to the retarded habituation abnormality observed in this study (Raine et al. 1991; Tranel and Damasio 1994).

Consideration needs to be given to explanations of the orienting deficit in schizotypal subjects other than preattentive template matching. For example, inspection of figure 1 indicates that the data for schizotypal subjects for trials 3–6 paralleled the data for normals for trials 1–5. Schizotypal subjects appear to take two trials to reach the orienting level first shown by the normals on trial 1, after which they habituate normally. It is possible, therefore, that the deficits observed in schizotypal subjects refer specifically to the initial generation of the neural template for the stimulus. Once this template is fully formed (i.e., by trial 3 in schizotypal subjects), habituation may be executed normally. As such, it is conceivable that the key deficit may lie in template generation (and initial allocation of attentional resources) as opposed to later degradation in the accurate representation of the neural trace of the template.

Another important question concerns how this orienting abnormality could translate into clinical symptoms of the type shown by schizotypal subjects. It was found that retarded habituation was correlated with both cognitive-perceptual deficits ($\rho = 0.51$) and interpersonal deficits ($\rho = 0.48$). These data suggest that the orienting deficit is relatively pervasive to schizotypal traits with the exception of the two traits making up disorganization, which failed to correlate with the orienting deficit ($\rho = 0.02$). It is possible that template mismatching results in faulty information processing, which may underlie schizotypal symptoms such as unusual perceptual experiences in which, for example, sounds are mistaken for voices. In addition, it is possible that paying too much attention to
environmental stimuli that should normally be ignored (e.g., heightened responding on trial 3) may in part underlie schizotypal symptoms such as ideas of reference and magical thinking. We would also caution that implicit assumptions about causality from biological data and schizotypy are easy to make, yet difficult to substantiate. Consequently, we are pursuing longitudinal research on SC orienting and schizotypy to tease out temporal relationships between these constructs and further pursue specificity with subtypes of schizotypy.

Results from the first sample (clinical schizotypal subjects) were also found in the second sample (normal subjects). Although it has been theorized that schizotypal personality represents a true dichotomy (e.g., Meehl 1990), it is conceivable that schizotypal personality and its neurobiological substrate are partly dimensional in nature (e.g., Siever and Davis 1991; Claridge and Beech 1995; Raine and Lencz 1995). It may be prudent, therefore, for future researchers in this area to conduct both categorical and dimensional assessments of schizotypal personality to assess which approach produces more robust findings. This study merely demonstrates that both approaches produce essentially the same finding with respect to abnormal electrodermal orienting and cannot by itself address the important conceptual issue of the categorical versus dimensional nature of schizotypal personality (Lencz and Raine 1995). Future longitudinal studies could also address the issue of whether young adults such as undergraduates can truly meet the DSM-IV (American Psychiatric Association 1994) criteria for schizotypal personality disorder, which needs to be stable and of long duration, with its onset traced back at least to adolescence or early adulthood.

Finally, although there were no group differences on nonresponse, some comment is necessary on the relatively high rate of nonresponse in the control group (43%). One contributing factor may be the relatively low stimulus intensity employed (60 decibel). Alternatively, recent work by Dawson et al. (1992a) has also reported rates of SC nonresponse to innocuous tones of 40 percent in normal controls, while rates of approximately 45 percent using 85-decibel tones have recently been reported by Iacono et al. (1993) in their normal control groups. Thus, new evidence converges to suggest that previously accepted notions of the base rate of SC nonresponse in normals may require some revision.

References


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