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The Interaction of Electrodermal Activity and Expressed Emotion in Predicting Symptoms in Recent-Onset Schizophrenia

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Abstract

In the present study, expressed emotion (EE) was assessed among immediate family members of 94 recent-onset schizophrenia patients at initial study entry point, and patients' electrodermal activity (EDA) was measured without the presence of family members at a baseline outpatient stabilization assessment. Psychiatric symptoms were also rated, both at the baseline outpatient test, and at one-year follow-up. Electrodermal Activity $\times$ Expressed Emotion interactions were observed at both test points. In each case, the highest levels of negative symptoms were observed among those who exhibited greater EDA and occurred in a high-EE environment. These results support the view that the combination of high family EE and sympathetic nervous system arousal confer especially high risk for poor negative symptom outcomes.

Keywords

Electrodermal activity (EDA); Expressed Emotion (EE); Positive Symptoms; Negative Symptoms; Schizophrenia; Stress

Electrodermal activity (EDA) and responsivity are stable peripheral indicators of sympathetic nervous system (SNS) function (Dawson, Schell, & Fillion, 2007), both among patients with schizophrenia and among those in the normal population (e.g., Schell et al., 2002). Heightened SNS activity, often indexed by (EDA), is frequently found in schizophrenia patients (e.g., Dawson & Nuechterlein, 1984; Dawson et al., 1994), and is related to longer hospital stays poor symptomatic and functional outcomes (Schell et al., 2005; Tarrier & Barrowclough, 1989). It is possible that stressors induce SNS activation, which then impairs cognitive processing and causes patients to further misinterpret stimuli and to interact with the environment in ways that contribute to a return of symptoms (Dawson & Nuechterlein, 1984; Nuechterlein et al., 1992a; 1992b).

One class of particularly troublesome environmental stressors for those with schizophrenia is critical or over-involved family attitudes about the patient (high expressed emotion, or EE) (Brown et al., 1972). The finding that high EE predicts psychotic relapse is well

replicated (Wearden et al., 2000). The relationship between living in a high-EE family and poor outcome may be due to the stressful nature of living in such an environment—a relationship that may be mediated by heightened SNS activation on the part of the patient (Nuechterlein & Dawson, 1984; Tarrier & Barrowclough, 1989). Indeed, higher levels of EDA are observed in patients whose families express high EE (e.g., Tarrier et al., 1979).

Our purpose in writing this paper is to examine the interaction between EDA, a stressful family environment, and the presence of positive and negative symptoms in schizophrenia patients early in the course of disorder. Our primary aim is to examine whether EDA interacts with family EE to predict symptoms of schizophrenia in order to test the hypothesis that patients who are most electrodermally reactive and who have the highest family EE levels are at particularly heightened risk for poor symptomatic outcome. To index EDR, we used skin conductance orienting responses (ORs) to innocuous environmental stimuli, since they predict both concurrent and later symptoms (Dawson, Nuechterlein, and Schell, 1992a; Dawson, Nuechterlein, Schell, and Mintz, 1992b; Schell et al., 2005).

Methods

Participants

Participants were 94 outpatients with schizophrenia (77 males, 17 females) with a mean age of 23.3 years ($SD = 4.4$) and mean education of 12.4 years ($SD = 2.1$). All had an initial onset of psychosis within two years of study entry, and were participants in Sample 1 of a longitudinal, prospective study of recent-onset schizophrenia patients entitled, “Developmental Processes in Schizophrenic Disorders” (P.I., Keith H. Nuechterlein) at the Semel Institute for Neuroscience and Human Behavior at UCLA. Patients were followed clinically in the UCLA Aftercare Research Program (Nuechterlein et al., 1992a) between 1980 and 1992. Of the 94 patients in this report, 62 (66%) contributed data to the Dawson et al. (1992a) study and 68 (72%) contributed data to the Schell et al. (2005) study.

Of the original 104 patients in Sample 1 of the Developmental Processes study, 94 patients who had EE, EDA, and symptom data at the time of their index hospitalization and at the three-month outpatient stabilization period were included in these analyses. Sixty nine of these patients also had symptom data available from a one year follow-up assessment. Patients were required to have received a diagnosis of schizophrenia ($n=81$) or schizoaffective disorder, depressed type, mainly schizophrenic ($n=13$), using Research Diagnostic Criteria (Spitzer et al., 1978). DSM-IV diagnoses were schizophrenia ($n=60$), schizoaffective disorder, depressed type ($n=8$), or schizophreniform disorder ($n=26$). Other inclusion and exclusion criteria, and a description of the treatment protocol, can be found in Nuechterlein et al. (1992a).

Design

Each patient was rated on an expanded version of the Brief Psychiatric Rating Scale (BPRS) (Lukoff et al., 1986) every two weeks. When clinically appropriate, antiparkinsonian medications with anticholinergic effects, typically benztropine, were discontinued for at least 24 hours prior to electrodermal testing, because EDA is cholinergically mediated. In 27 of the 94 cases (5 cases had missing data), we were not able to temporarily discontinue use of these medications. Patients for whom these medications were or were not discontinued did not have fewer nonspecific orienting responses (NORs) than patients off anticholinergic medications, but actually had somewhat more frequent NORs demonstrating that antiparkinsonian medication did not suppress NORs in this sample, $t(92) = 2.4$, $p = .02$, Cohen's $d = .53$). Therefore all patients were included in the analyses.

During the initial entry point into the longitudinal assessment and treatment protocol, family members were assessed with the Camberwell Family Interview (CFI; Vaughn & Leff, 1976a) for expressed emotion (EE) attitudes. EDA assessments occurred when the patient was clinically stabilized on medication, approximately three months after hospital discharge. The patient's clinical state was evaluated at the clinical stabilization point and at one-year follow up using a BPRS that covered a 2-week period.

Assessment of Expressed Emotion Attitudes as an Identification of Family Stressor

Family comments about the patient during the CFI interview were rated for criticism, hostility, and emotional over-involvement. Any family member with 6 or more critical comments, a rating of 4 or 5 on emotional over-involvement, and/or who was rated as showing hostility was considered to have high EE following criteria established by Vaughn and Leff (1976b) and Vaughn et al. (1984)¹. A family was categorized as having high-EE attitudes ($n=66$) if any of the family members were categorized as having high EE, or low EE ($n=28$) if no family members were identified as having high EE. Change in EE and living situation over time is available for only a subset of participants and is beyond the scope of this brief report.

Criteria for Defining Positive and Negative Symptoms

The BPRS was used to identify the presence of positive and negative symptoms at the baseline outpatient stabilization test and the one-year follow-up test. Each BPRS item is rated on a 1 to 7 scale according to behavioral anchor points (Ventura et al., 1993). To measure presence of positive symptoms, a thought disturbance factor score was used, comprised of the BPRS items unusual thought content, hallucinations, conceptual disorganization, and grandiosity (Guy, 1976). To measure negative symptoms, a BPRS anergia factor score was used, comprised of the BPRS items blunted affect, emotional withdrawal, motor retardation, and disorientation (Guy, 1976).

Electrodermal Testing Procedures

Electrodermal testing procedures have been described elsewhere (Dawson et al., 1994) and are only summarized here. EDA was recorded during a 1-hour laboratory session on each test occasion using standardized techniques (Dawson et al., 2007) on a Dynograph recorder (Sensor-Medics). At the beginning of each EDA testing session, recording electrodes were attached and skin conductance was recorded during a 5-min rest period. Following the rest period, participants were presented with twelve mild nonsignal tones (1000 Hz, 78 db, 1.0 s duration, 25 ms controlled rise and fall times) through headphones binaurally (intervals varying between 20, 25 and 30 s). These stimulus parameters were chosen to reproduce conditions under which skin conductance orienting and habituation abnormalities previously found in patients with schizophrenia (Bernstein et al., 1982; Dawson et al., 1992c; Gruzelier & Venables, 1972). The number of ORs (NOR), the number of skin conductance responses that began within 1 – 3 s after onset of the 12 tones, was determined for each patient from the paper tracing. ORs over .05 microseimens were included here. SCRs were hand scored from paper tracings and values were independently confirmed in most cases by co-author (M.E.D.) who had over 20 years experience in scoring analog polygraph charts. Accuracy for detecting ORs was > 95%.

¹Use of dichotomous variables based on simple cut points on a continuum provide less statistical power than use of the continuous variables (MacCallum et al., 2002). However, the use of a dichotomous variable for EE based on these three ratings follows an established convention for this construct.

Results

Low and high EE groups did not differ with respect to average age, $t(92) = .73$, $p = .47$, $d = .17$, sex, Fisher's test $p = .38$, Cohen's $w = .13$, Caucasian versus non-Caucasian, Fisher's test $p = .33$, $w = .13$, or highest grade achieved in education, $t(92) = 1.07$, $p = .29$, $d = .25$. Consistent with past findings, NORs were greater in the high EE than in the low EE group, means of 1.91 ($SD = 1.9$) and .96 ($SD = 1.6$) respectively, $t(92) = 2.49$, $p = .02$, 2-tailed, $d = .52$.

In order to determine the independent and joint contributions of the NORs and EE measures in predicting symptom levels, a multiple regression analysis was performed for both thought disorder and anergia at the initial stabilization point and at follow up. For each regression equation, NORs, EE level, and their interaction were entered as predictors. EE and NOR levels did not interact to predict thought disorder either at stabilization, Cohen's $f^2 = .01$, or at follow up, $f^2 = .006$. In contrast, in the model predicting baseline BRPS anergia the interaction was significant, $t(91) = 2.40$, $\beta = 1.1$, $p < .01$, $f^2 = .07$. NORs also interacted with EE to predict follow-up anergia, $t(89) = 2.02$, $\beta = .9$, $p = .05$, $f^2 = .05$. To aid in interpreting this interaction, the continuous NOR variable was trichotomized into nonresponders (no ORs to the tones), low responders (1 or 2 responses), and high responders (3 or more responses). As shown in Figure 1, EE was associated with higher anergia at the one-year follow-up only among high EDA responders. EE and NORs did not interact to predict thought disorder either at stabilization or at follow up.

Discussion

There are two principal findings in the present study. First, EDA responding to auditory stimuli was greater among patients from high-EE families than those from low-EE families, consistent with previous findings. These results are consistent with the neural diathesis-stress model of Walker and Diforio (1997). According to this model, when individuals with a diathesis for schizophrenia are exposed to stressors (such as those likely to be encountered in a high-EE environment), there is activation of the HPA axis, which induces dopamine and cortisol release. Physiological stress responding is also associated with activation of the sympathetic nervous system and EDA through the locus coeruleus-norepinephrine (LC/NE) – sympathetic system. The HPA axis and the SNS express “seem to participate in a positive, reverberating feedback loop, so that activation of one system tends to activate others as well” (Kaltsas & Chrousos, 2007, p. 305). EDA may provide one means of assessing stress reactivity.

The second principal finding, which is the most theoretically interesting, is that negative symptoms both at baseline and at one year follow-up were predicted by the interaction of EDA and EE. That is, high anergia was observed only among those who exhibited greater EDA and incurred high EE. Thus, our previous reports of a relationship between EDA and negative symptoms, either in the short-term (Dawson et al., 1992a) or over a one-year period (Schell et al., 2005), were due in large part to the subgroup of participants whose families expressed high EE.

There are previous reports of higher negative symptom levels in patients with families high in critical comments (e.g., Barrowclough et al., 2003), but also negative reports as well (e.g., Glynn et al., 1990). Varying levels of EDA responding naturally occurring in different samples reported in the literature might help explain such disparate results. If a sample of patients is low in EDA responding, EE might not predict negative symptom levels, but if a sample is high in EDA responding, an effect would be seen. Varying EE levels across samples might also account for discrepant findings in reports of the relationship between

EDA and negative symptom outcome (Brekke et al., 1995; Dawson et al., 1992b; Katsanis & Iacono, 1994; Schell et al., 2005).

It might at first seem paradoxical that heightened autonomic responsivity is associated with negative symptoms. It is possible that emotional restriction and social withdrawal are an attempt by some patients to reduce their autonomic overresponsiveness (Venables & Wing, 1962). The present results suggest that negative symptoms may be associated with heterogeneous processes and in some cases be an attempt by individuals with heightened autonomic responsivity to avoid environmental stress. The present results also support the view that the influence of high EE—a well replicated predictor of positive symptoms—predicts negative symptoms as well, particularly among those high in SNS responding.

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Figure 1. Relationship of nonspecific orienting responses (NORs) and Expressed Emotion level with Follow-Up Anergia