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RELATIONSHIPS BETWEEN FACIAL EMOTION PERCEPTION AND PSYCHOSIS PRONENESS: PERSONS WITH SCHIZOPHRENIA AND CONTROLS

by

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A thesis submitted in partial fulfillment of the requirements for the degree of Master's of Science in Clinical Psychology

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Abstract

RELATIONSHIPS BETWEEN FACIAL EMOTION PERCEPTION AND PSYCHOSIS PRONENESS: PERSONS WITH SCHIZOPHRENIA AND CONTROLS

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The University of Texas at Tyler
May 2010

It is well documented that individuals with schizophrenia demonstrate deficits in emotion processing. Though study is established in this area, the same can not yet be said for controls or at risk groups like those with a family history of schizophrenia, those with a genetic predisposition, or those high in schizotypy who are at higher risk for schizophrenia. In the proposed study I attempted to elucidate the relationships between schizotypal symptoms and facial emotion perception. The study of schizotypy and emotion perception may provide insight into the etiology of schizophrenia through better understanding of the symptom constellations and deficits that mark the disorder. Research on such high risk groups may also help to identify and understand vulnerability markers that aid in the early detection and identification of the disorder and those at risk for it. Early detection and increased understanding of the transitional phase of schizophrenia should help us in developing treatments to attenuate symptoms after onset or even abate the transition altogether. The current study reviewed schizotypy and facial emotion perception in people with schizophrenia and in controls. People with schizophrenia rated higher on schizotypy scales and worse on facial emotion recognition. No correlation was found between facial emotion perception and schizotypy in the Schizophrenia group, but an unexpected one was found in the Control group. This article explorers these relationships and discusses possible reasoning, impact, and future avenues of research.

RELATIONSHIPS BETWEEN FACIAL EMOTION PERCEPTION AND PSYCHOSIS PRONENESS: PERSONS WITH SCHIZOPHRENIA AND CONTROLS

Introduction and Overview

Schizophrenia is a disorder defined by many symptoms, including social dysfunction. Impaired social functioning is characteristic of schizophrenia (APA 2000) and these impairments are more pronounced in people with schizophrenia than those with any other psychiatric disorder (Mueser & Bellack, 1998). Many of these social functioning impairments are linked to social cognition. Social cognition is now a high priority area in the study of schizophrenia due to its importance in understanding clinical symptoms of schizophrenia and explaining functional outcomes (Green et al., 2008; Pinkham & Penn, 2006). Social Cognition has been defined as the ability to perceive the intentions and dispositions of others; the capacity to build representations of the relation between one's self and others and to use those representations flexibly to guide social behavior; a set of inter-related neurocognitive processes applied to recognition, understanding, accurate processing, and effective use of social cues and information; and mental operations underlying social interactions including perceiving, interpreting, and generating responses to the intentions

dispositions and behaviors of others (Adolphs, 2001; Brothers, 1990; Green et al., 2008; Penn et al., 1997). In this study the latter definition is used. Within this model, social cognition is comprised of 5 main constructs; Theory of Mind, Social Perception, Social Knowledge, Attributional Bias, and Emotional Processing (EP) (Green et al., 2008).

Impairments in social cognition are partially represented by consistent deficits in emotion perception (Penn, Addington, & Pinkham, 2006). Emotion perception, also called emotion identification or affect recognition, is under the larger umbrella of Emotional Processing (Green et al., 2008), and can be seen as the ability to perceive, identify, and differentiate human affects, moods, and emotions via visual and/or auditory cues. People with schizophrenia are historically poor at EP. In general they have difficulty with identification, recognition and discrimination of emotional expressions (Edwards et al., 2001; Kohler et al., 2003; Mandal, Pandey, & Prasad, 1998; Morrison, Bellack, & Mueser, 1988). In addition, individuals with schizophrenia have been shown to demonstrate abnormalities in emotional experience, emotion regulation, and expression of their own emotions (Phillips & Seidman, 2008). These deficits are not concrete, static impairments based on diagnosis. There are also differential deficits based on the stimuli and task. Schizophrenics are worse at recognition and imitation of fear versus other emotions (Gaebel & Woelwer, 1992). They rate

the emotional intensity of anger abnormally (Morrison, Bellack, & Bashore, 1988). People with schizophrenia have a bias to rate happiness more strongly during discrimination tasks (Schneider et al., 1995). They are worse at recognition of unfamiliar faces, but appear unimpaired on familiar faces (Archer, Hay, & Young, 1994). Mood induction and emotion discrimination scores have correlations with symptom scores, but these vary between the two tasks and even change direction based on the scale within the symptom measures (Schneider et al., 1995). Symptomatology has differential effects on EP tasks, evidenced by the varying correlations among symptom scales and performance. This encourages us to look to a dimensional approach that puts emphasis on relationships across a continuum, and across situations and stimuli.

If we look beyond the categorical diagnosis of schizophrenia to a symptom continuum we are better able to identify relationships within the disorder and reduce confounds, such as negative symptomology, general poor cognition, and psychosis, surrounding its study. This approach has been used previously to show that as paranoia increases along a continuum, EP deficits also increase (Combs, Michael, & Penn, 2006). Focus on the diagnosis and the large symptom constellations that accompany the diagnosis and subtypes within schizophrenia take us away from the defined study of the individual symptoms and impairments and lead us to classify and label and not solve. For example, a person with schizophrenia may have many negative symptoms and show poor EP, but would

also have general poor performance on all cognitive tasks, and not show a relative EP deficit (Kerr & Neale, 1993; Sachs et al., 2004; Salem, Kring, & Kerr, 1996). In essence we could categorize them as "with prominent negative symptoms", but this is a qualitative description that does not give us information about the relationships between specific symptoms and specific deficits. The reason this person with schizophrenia is markedly poor at EP could be related to their paranoia symptoms, their general poor cognitive capabilities, and/or their deficient social cognitive development. Until we tease out the underlying aspects we can not be certain. Even this example neglects to mention many additional underlying causes currently being considered.

Other areas of interest for underlying causes of schizophrenia spectrum dysfunctions include a structural encoding deficit, reduced amygdala activation, and other brain activation and neural substrate deficits (Gur et al., 2002; Turetsky et al., 2007; Williams et al., 2004). These point to possible misattributions of autonomic processing, abnormalities of visual processing, and other diffuse neurocognitive network deficits in the hypothalamus, mesocorticolimbic dopaminergic system, orbitofrontal cortex, dorsolateral prefrontal cortex, temporal cortex and portions of the parietal cortex. This would appear congruent with the evidence for a deficit in "social cognitive neural circuit" which includes the amygdala, fusiform gyrus, superior temporal sulcus, and prefrontal cortices (Pinkham et al., 2003). We can see here that there are numerous aspects of

schizophrenia presentation, and each of these aspects has its own deficits and symptom expression, and maybe even pathophysiology. If we disentangle the classification and look at the symptom/impairment relationships we may be able to obtain a better understanding of the processes and structures that are at work. This is why I have chosen to explore the several scales of schizotypy and EP.

Why choose EP as the one construct, out of the 5, to study? Impaired recognition of emotional expressions in others is one of the most consistent social cognitive deficits in schizophrenia (Edwards, Jackson, & Pattison, 2002). Deficits in EP among people with schizophrenia are stable over time and present across phases of the disorder, be it first episode, multiple episode, or remission (Addington & Addington, 1998; Gaebel & Woelwer, 1992; Penn & Combs. 2000; Penn et al., 2000). Emotion processing deficits are also uniquely associated with neurocognitive performance and symptom severity (Kohler et al., 2000). EP is also usable as a means to tease apart symptom constellations due strong correlations with poor emotion recognition (Combs, Michael, & Penn, 2006; Kline, Smith, & Ellis, 1992; Lewis & Garver, 1995). In addition, at risk populations, those with high genetic risk or high in schizotypy, also have poor EP that is mediated by symptom severity and stimuli presentation, modality, valence, and intensity (Phillips & Seidman, 2008). Deficits in EP contribute heavily to social impairments in schizophrenia (Hooker & Park, 2002; Ihnen et al., 1998; Kee et al., 2003), and these social deficits cause distress and hamper daily living (Meyer

& Kurtz, 2009). Emotion Processing is a key determinant of work functioning and independent living for individuals with serious mental illness (Kee et al., 2003). It is also consistently associated with community functioning, and there is good support for a relationship with social behavior in the milieu and social skill (Couture, Penn, & Roberts, 2005). We also see that deficits in social cognition are associated with impairment in ward behavior (Penn et al., 1996), and social cognition contributes unique variance to interpersonal skill beyond that of neurocognition (Pinkham & Penn, 2006). This impairment in social cues recognition in people with schizophrenia may be a possible explanation for their impaired social functioning (Zhu et al., 2007). Perhaps the most important reason to focus on EP is the simple fact that we know it can be improved (Penn & Combs, 2000; Russell, Chu, & Phillips, 2006; Silver et al., 2004).

Within the construct of EP I will herein be working specifically with the aspect of Facial Emotion Perception (FEP), which I define as the ability to perceive, identify, and differentiate human affects, moods, and emotions via visual cues of the head and face. This is measured by the Face Emotion Identification Test. Facial affect perception deficits are a crucial domain of impairment in schizophrenia that both contribute unique variance to social-skill deficits and may also temper the relationship between some aspects of neurocognition and social-skill (Meyer & Kurtz, 2009). Social cognition involves the interface of emotional and cognitive processes (Penn, Sanna, & Roberts,

2008), and there is an association between face perception measures in schizophrenia patients and social competence, including social adjustment and social skill (Mueser et al., 1996). Affect recognition is an important aspect of psychosocial functioning in stable outpatients with schizophrenia (Hofer et al., 2009). Also, affect recognition partially mediates between cognition and social functioning (Addington, Saeedi, & Addington, 2006). There are numerous studies that mark the abnormalities of FEP (Green, Williams, & Davidson, 2003; Phillips, Senior, & David, 2000; Phillips & Seidman, 2008). Even in non-clinical populations deficits in EP can be seen as vulnerability markers for schizophrenia (Green et al., 2008; Lenzenweger & O'Driscoll, 2006; Phillips & Seidman, 2008).

Like poor EP, schizotypy is also seen as a vulnerability marker for schizophrenia (Horan et al., 2008). Schizotypy can be loosely defined as a schizotypal personality organization, or more concretely as the psychological and personality organization resulting from the schizotaxic individual (one with the underlying genetic predisposition to schizophrenia and schizotypy) interacting with and developing within the world of social learning influences (Lenzenweger, 2006). Measures of schizotypy are generally based on a 3 factor model similar to schizophrenia which includes cognitive-perceptual (positive), interpersonal (negative), and disorganized (disorganized) symptom constellations (Kerns, 2006; Raine, 2006). In this study I examined the relationship between schizotypy scores, as measured by the Chapman Psychosis Proneness Scales, and FEP

scores in persons with schizophrenia and in non-clinical controls, college students. I used 5 of the Chapman scales. One is Magical Ideation, defined as belief in superstitious or magical forms of causation which are regarded as invalid by conventional standards. Two, Perceptual Aberration, which can be seen as gross distortions in the perceptions of one's own body and other perceptual distortions. Three is Physical Anhedonia, which is the absence of sensory and aesthetic pleasures like eating, touching, feeling, sex, temperature, smell, sight, and sound. Four, Social Anhedonia, is a social disinterest or schizoid asociality, separate from social anxiety. The fifth is Schizotypal Ambivalence which is a tendency to simultaneously experience divergent emotions toward situations, objects, or people. I picked 5 because, as suggested by Horan (2008), differences in longitudinal associations suggest that different scales play different roles in the development processes that lead to psychotic symptoms.

I am aware that the emotion being viewed likely plays a part in performance and though it is not analyzed in this paper, work regarding emotion specific effects is underway.

Hypotheses

Hypothesis #1

I expected to see a significant main effect of Group on FEP and schizotypy scores, where participants with schizophrenia are expected to perform worse than controls on FEP tasks, while conversely scoring higher on schizotypy scales.

Hypothesis #2

I predicted negative correlations between FEP score and all Psychosis

Proneness Scale scores within the Schizophrenia group, such that as schizotypy
scale scores increase FEP scores decrease, with the Social Anhedonia Scale
showing the strongest negative correlations with the FEP scores (Kwapil, 1998;
Miller et al., 2002). I predicted no significant correlation between the FEP and
Psychosis Proneness Scale scores within the Control group. This is based on
previous literature using the SPQ, and is an exploratory hypothesis for me, where
I was looking to see if the same holds true for the SAE. I also explored if
significant correlations existed when the groups were combined.

Hypothesis #3

Across all groups I explored if the Psychosis Proneness Scale scores account for significant variance in FEP. I expected that within the Schizophrenia group this would be seen, but not within the Control group.

Methods

Participants

Participants were comprised of people from the community with diagnoses of schizophrenia or schizoaffective disorder, recruited via newspaper advertisements and flyers placed at The Andrews Center, a community mental health center in Tyler Texas. Schizophrenics were stable, not actively psychotic, and medication compliant. They were interviewed by Dr. Dennis Combs, a licensed clinical psychologist, using a modified version of the Structured Clinical Interview for the DSM to verify diagnosis, medication compliance, stability, and substance abuse. Schizophrenic participants were paid the sum of \$30 for completion of the initial testing session.

College students were recruited mainly from undergraduate psychology classes at The University of Texas at Tyler. Participants were screened via

interview, and any current clinical diagnosis or previous psychotic diagnosis excluded them from participation. Students were offered extra credit for their participation.

Measures

Demographic information; gender, age, ethnicity, education, and medication type and dosage, were collected as part of an initial interview when participants arrived for testing. Participants were asked to wear glasses or contacts if they were prescribed, but no visual acuity tests were given.

The Face Emotion Identification Test (Kerr & Neale, 1993) served as the measure of emotion perception in this study. The FEIT is a 19 item, still frame, black and white presentation of six different emotional states: happy, sad, angry, surprised, afraid, and ashamed (each image shown for 15s). The FEIT has been widely used in schizophrenia research (Penn et al., 2000). Subjects circle their response for each item on a worksheet while watching the presentation. FEIT scores reflect emotion perception abilities, such that a higher score equates to better performance. The norms that I used were derived by Penn et al. (2000), which show a mean of 14.18 and a SD of 2.15 for controls on the FEIT. A graph showing those results is included in the appendix. The FEIT faces were taken from sets developed by Izard (1971) and Ekman (1976). The theory behind these

emotion faces is based on the idea, brought to the fore by Ekman, that there are certain facial emotion expressions that are universal across cultures. Emotion display items are picked based on a complex interplay of facial muscle movement that builds a consistent picture for each emotion.

I used the Chapman psychosis proneness scales as the measure of schizotypy, specifically the Survey of Attitudes and Experiences (SAE), which includes the Magical Ideation (Eckblad & Chapman, 1983; Horan et al., 2008), Revised Physical Anhedonia (Chapman, Chapman, & Raulin, 1976; Horan et al., 2008), Revised Social Anhedonia (Eckblad et al., 1982; Mishlove & Chapman, 1985), Perceptual Aberration (Chapman, Chapman, & Raulin, 1978; Horan et al., 2008), and Infrequency Scales. The version I used also contains a Schizotypal Ambivalence Scale, consisting of 19 additional questions, which raises the total items to 198 (Kwapil, Mann, & Raulin, 2002; Mann et al., 2008). These scales were largely developed following the steps that Jackson (1970,1971) recommended for personality scale development. This included development of items often based on Meehl's (1964) description of schizotypal behaviors and Bleuler's (1950) description of ambivalence. Items were designed and an initial test battery was given which included scales of social desirability (Crowne & Marlowe, 1964) and acquiescence (Bill, 1978; Jackson & Messick, 1962). Those items with low item-scale correlation or with high desirability or acquiescence correlations were dropped or revised. This process was repeated until a refined

group of items with low desirability and acquiescence correlations was left. The final test version would then be normed on a large sample of students. The cut off scores for deviance were usually defined as 1.91-2.0 SD above the mean. This vacillated based on gender and the test given. A uniform table of norms with cutoffs at 1.96 SD above the mean was provided by Dr. Tom Kwapil, who currently manages the scales. Cronbach's Alpha for this sample was .951. The reason for selection of the SAE and the Chapman Psychosis Proneness scales was that most current research involving schizotypy and its relation to facial emotion perception uses the Schizotypal Personality Questionnaire, and I was looking to discern if the SAE shows the same/similar patterns of deficit in relation to FEP measures.

Procedures

A master's-level clinical psychology student administered the study protocol, which was approved by the Institutional Review Board of The University of Texas at Tyler. Participants with schizophrenia were recruited for the study via newspaper advertisements and flyers placed in The Andrews Center, a community mental health center in Tyler Texas. College students were recruited mainly from undergraduate psychology classes at The University of Texas at Tyler. An initial interview was conducted with participants when they arrived for

testing to go over informed consent and risks involved with testing, as well as to garner additional information. After pre-screening all participants reviewed the informed consent on paper, and then discussed it verbally with the administrator. In the initial interview we garnered demographic data and ensured that all relevant criteria were met (ie. wearing glasses, not floridly psychotic, not intoxicated). Upon entry all participants with schizophrenia completed a full test battery, which included the SAE and FEIT, for which they were paid \$30. College students also completed a test battery, which included the SAE and FEIT, lasting approximately 2 hours, for which they received extra credit and/or received payment of \$30.

Results

The data analysis involved an initial examination of the descriptive and statistical properties of each variable to assess for normal distribution and skew. A Chi-Square test identified a significant difference in ethnicity within the Control group, $X^2(n=31)=27.548$, p<.001. There were only 2 Black and 5 Hispanic participants compared to 24 White participants. There were no differences among the dependent variables based on ethnicity within the Control group, when checked with a One Way ANOVA, but any attempt to generalize findings to an ethnically diverse population should be done so with caution. Similarly, Age

also showed significant between group differences, with the Schizophrenia Group being older than controls, p = .000. However, Age did not show a correlation with FEIT performance, p = .103. No significant differences in Ethnicity were identified within the Schizophrenia group, $X^2(n=12)=1.333$, p = .248. No significant differences in Gender were identified within groups for either the Control or Schizophrenia groups, $X^2(n=31)=1.581$, p = .209 and $X^2(n=12)=.333$, p = .564. There was a significant difference in the Mean age of participants between groups, with the Schizophrenia group being older than Controls, but age did not have a significant impact on performance. Demographics are summarized in Table 1 of the Appendix.

To assess for outliers, I computed Cook's distance and did not identify any outliers greater than ± 3SD. Skewness and Kurtosis were within acceptable limits between ±2, which suggested that the variables were relatively normally distributed. Each variable was summarized by computation of mean, standard deviation, and range, and this information was included in Table 2 in the Appendix.

Hypothesis 1

I expected to see a significant main effect of Group on Facial Emotion

Perception and schizotypy scores, where participants with schizophrenia were

expected to perform worse than controls on the FEP task, while conversely scoring higher on schizotypy scales.

Analytic Method: An independent-samples t-test (Group: Schizophrenia vs. Control) was performed to assess for between group differences on the FEIT (dependent variable). Levine's test showed that equal variances could not be assumed. The Schizophrenia group scored significantly lower than the Control group, t(33.967) = -4.080, p < .001. The Schizophrenia group obtained a mean FEIT score of 10.75 (SD=1.42), N = 12, and the Control group obtained a mean FEIT score of 13.19 (SD=2.43), N = 31. This is summarized in Table 3 of the Appendix.

A 2 (Group: Schizophrenia vs. Control) x 5 (Within subjects: Perceptual Aberration vs. Magical Ideation vs. Physical Anhedonia vs. Social Anhedonia vs. Schizotypal Ambivalence) mixed model MANOVA was conducted to examine for differences on the SAE subscales by group membership. There was a significant multivariate effect found, Wilk's Lambda = .446, F = 9.192, p < .001. This was followed by a series of univariate ANOVAs on each individual subscale from the SAE, and then followed by pairwise post hoc mean comparisons, using Bonferroni's correction. Tables for analyses and means are included in the Appendix (Table ???). When probed with univariate ANOVA's I found significant group differences for Perceptual Aberration, F(1,41) = 30.785, p < .001, $\eta^2 = .429$, Magical Ideation, F(1,41) = 34.018, p < .001, $\eta^2 = .453$, and Schizotypal

Ambivalence, F(1,41) = 7.184, p = .011, $\eta^2 = .149$, where the Schizophrenia group had higher scores on these scales. Across all subscales, participants with schizophrenia had higher scores than controls. Summary scores are presented in Table 3 of the Appendix.

Hypothesis 2

I predicted negative correlations between FEIT scores and all Psychosis Proneness Scale scores within the Schizophrenia group, such that as schizotypy scale scores increase, reflecting more psychosis traits, FEIT scores decrease. I predicted no significant correlation between the FEIT and Psychosis Proneness Scale scores within the Control group. This is based on previous literature using the SPQ, and I was looking to see if the same holds true for the SAE. I also explored if significant correlations existed when the Control and Schizophrenia groups were combined, to reflect a continuum approach to the data.

Analytic Method: Correlational analyses using the Pearson Correlation Coefficient were used to describe the magnitude and direction of the relationships. While the Social Anhedonia Scale showed the strongest correlation with the FEIT in the Schizophrenia group (r = -.262), the result was not significant, p = .411. Unexpectedly, a significant correlation and a near significant

trend were found in the Control group. Even more unexpected, both were in the positive direction, such that as schizotypy scores increased so did FEIT scores; Perceptual Aberration, r = .314, p = .086, and Magical Ideation, r = .432, p = .015. No significant correlations were found when groups were combined. Results are presented in correlation tables, for the total sample, schizophrenia group, and controls, in the Appendix (Table 4,5,6).

Hypothesis 3

Within each group and the total sample, I explored if the Psychosis

Proneness Scale scores account for significant variance in FEIT scores. I

expected that within the Schizophrenia group this would be seen, but not within the Control group or total sample.

Analytic Method: A linear regression analysis was conducted to determine the variance in the FEIT score accounted for by each of the 5 individual schizotypy scales in the schizophrenia group, the controls, and the total sample combined.

For the total sample (n = 43), when all variables were entered at the same time (direct method), the overall regression model accounted for 5% of the variance in FEIT scores (R = .228), which was not significant, p = .84. There

were no SAE subscale scores that emerged as significant predictors in the regression analysis (see Table 7).

For persons with schizophrenia only (n = 12), when all variables were entered at the same time (direct method), the overall regression model accounted for 61% of the variance in FEIT scores (R = .784), which was not significant, p = .227. Had there been significance, the SAE Social Anhedonia (p = .029) and Schizotypal Ambivalence (p = .047) subscale scores would have emerged as significant predictors in the regression analysis. (see Table 8).

For controls only (n = 31), when all variables were entered at the same time (direct method), the overall regression model accounted for 28% of the variance in FEIT scores (R = .533), which was not significant, p = .115. There were no SAE subscale scores that emerged as significant predictors in the regression analysis (see Table 9).

Discussion

The purpose of this study was to examine the relationships between facial emotion perception, as measured by the Face Emotion Identification Test, and schizotypy, as measured by 5 of the Chapman Psychosis Proneness Scales, Perceptual Aberration, Magical Ideation, Physical Anhedonia, Social Anhedonia.

and Schizotypal Ambivalence, aggregated in the Survey of Attitudes and Experiences, in a sample of persons with schizophrenia and college student controls. This study extends previous research and analysis focused on Social Cognition, Emotional Processing, and Psychosis Proneness by comparing performance on the same measures in both groups.

Both groups took the SAE and FEIT as part of a larger battery, and the Schizophrenia group was found to have significantly lower FEIT scores, and significantly higher scores on the Perceptual Aberration, Magical Ideation, and Schizotypal Ambivalence scales. This supports Hypothesis #1 and previous research that shows that people with schizophrenia are worse at facial emotion perception than controls (Morrison, Bellack, & Mueser, 1988; Mandal, Pandey, & Prasad, 1998; Edwards et al., 2001; Kohler et al., 2003), and also report higher schizotypy symptomology (Chapman, Chapman, & Raulin, 1976; Horan et al. 2008; Phillips & Seidman, 2008).

Negative correlations between schizotypy scores and FEP tasks have previously been shown in people with schizophrenia, specifically within the Social Anhedonia sphere, such that as schizotypy increases emotion perception acumen decreases (Miller et al., 2002; Kwapil, 1998). Though this was not shown in the current study, a trend was seen for the Social Anhedonia scale within the Schizophrenia group. This lack of significance is believed to be a contrivance of small sample size, and the results may attain significance with a larger sample.

Unexpectedly, Controls showed a significant correlation between the Magical Ideation scale and FEIT scores. This finding is made more unusual because the relationship is positive, such that as schizotypy increases, FEIT scores also increase. The presence of a relationship among controls and the direction, both disagree with previous research and logic. An exact explanation is not available at this time, but it is believed that the small sample size played a part, though it may also be an occurrence particular to the SAE versus what has been seen previously with the SPQ. Unreported pre-morbid or confounding conditions may also have been present. This particular relationship is an avenue for further research and exploration.

Regression analyses were performed to determine which, if any, of the psychosis proneness scales were significant predictors of performance on the FEIT. This was analyzed for each group individually and then for the groups combined. The model showed no significant predictors for any of the groups. However, within the Schizophrenia group, trends for the Social Anhedonia and Schizotypal Ambivalence scales were such that it is believed a larger sample size would reveal them both to be significant predictors of FEIT performance.

The rationale for review of sub-clinical schizotypy is based in its ability to provide insight into the etiology of and transition into Schizophrenia. By examining performance in sub-clinical populations in concurrence with schizophrenic populations, we may be able to identify symptom and deficit

constellations that can be used as vulnerability markers for subsequent onset of schizophrenia. Additionally this type of study should allow us to show the importance of a dimensional view of pathology and its mechanisms, though in this study a dimensional was not supported for Facial Emotion Perception. Our understanding of the transition into schizophrenia is key to prevention and early treatment. The evidence for a disjunction in liability and manifestation suggests underlying developmental processes interacting with environmental influences and second hits that we do not yet fully understand. Research such as what I have presented here seeks to take steps toward understanding these interacting factors so that we may one day build a stronger model of causality and prevention.

This study has several limitations, including a small sample size and the use of only one emotion perception measure. Also, use of college students, instead of a community cross section has its own implications for the efficacy of this study. However, I believe that these limits provide a solid foundation for future research would should include more numerous and more generalizable measures of emotion recognition, a community sample in conjunction with a college sample, and if possible a larger sample of people with schizophrenia. In addition measures for eye tracking, EEG, and social functioning would likely add a much more expansive view of the issue. Finally, I would like to recommend all these future avenues be explored in a longitudinal study of youth who are in high

risk groups to better identify when and where the transitional changes are taking place.

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Appendix: Tables

Table 1

Demographics & Sample Characteristics

	Schizophrenia			Con			
N	1	2		31			
Gender	Male 7 Female 5		Male 12	12 Female 19		19	
Ethnicity	White 8	Bla	ack 4	White 24	Bla	ck 2	Hispanic 5
	<u>Mean</u>	<u>N</u>	<u>SD</u>	Mean	<u>N</u>	<u>SD</u>	
Age	40.18	11	10.56	22.84	31	10.3	34
Education	12.75	12	1.49	14.68	31	1.33	3
Chlorpromazine *	864.69	12	740.49				
Equivalent							

^{*}Woods (2003)

Table 2

Descriptives: Statistics & Summary Scores

	Schizophrenia		Cont	Control		Total Sample		
	n = 1	12	n =	31	n = 4	13		
	Mean	SD	Mean	SD	Mean	S D	Range	Skew Kurt
FEIT	10.75	1.42	13.19	2.43	12.51	2.44	9 – 17	.20 -1. 16
Perceptual Aberration	13.25	7.20	4.06	3.66	6.63	6.37	0 – 26	1.19 1.08
Magical Ideation	15.83	6.45	6.35	4.00	9.00	6.39	0 – 25	.8507
Physical Anhedonia	16.50	6.52	12.90	9.24	13.91	8.64	2 – 42	1.33 1.78
Social Anhedonia	14.50	8.91	12.45	8.58	13.02	8.61	0 - 34	.6332
Schizotypal Ambivalence	10.75	5.43	6.26	4.73	7.51	5.28	1 – 19	.49 -1.04

Abbreviation: Face Emotion Identification Test (FEIT)

Table 3

Between Group Effects

Schizophrenia Control	12 31	10.75	1.42	(-4.08)			
Control	31			(-4.00)	33.97	.00	-
		13.19	2.43				
Schizophrenia	12	13.25	7.20	30 .79	1	.00	.43
Control	31	4.06	3.66				
Schizophrenia	12	15.83	6.45	34.02	1	.00	.45
Control	31	6.35	4.00				
Schizophrenia	12	16.50	6.52	1.52	1	.23	
Control	31	12.90	9.24				
Schizophrenia	12	14.50	8.91	.48	1	.49	
Control	31	12.45	8.58				
Schizophrenia	12	10.75	5.43	7.18	1	.01	.15
Control	31	6.26	4.73				
•	Schizophrenia Control Schizophrenia Control Schizophrenia Control	Schizophrenia 12 Control 31 Schizophrenia 12 Control 31 Schizophrenia 12 Control 31 Schizophrenia 12 Control 31	Schizophrenia 12 15.83 Control 31 6.35 Schizophrenia 12 16.50 Control 31 12.90 Schizophrenia 12 14.50 Control 31 12.45 Schizophrenia 12 10.75	Schizophrenia 12 15.83 6.45 Control 31 6.35 4.00 Schizophrenia 12 16.50 6.52 Control 31 12.90 9.24 Schizophrenia 12 14.50 8.91 Control 31 12.45 8.58 Schizophrenia 12 10.75 5.43	Schizophrenia 12 15.83 6.45 34.02 Control 31 6.35 4.00 Schizophrenia 12 16.50 6.52 1.52 Control 31 12.90 9.24 Schizophrenia 12 14.50 8.91 .48 Control 31 12.45 8.58 Schizophrenia 12 10.75 5.43 7.18	Schizophrenia 12 15.83 6.45 34.02 1 Control 31 6.35 4.00 Schizophrenia 12 16.50 6.52 1.52 1 Control 31 12.90 9.24 Schizophrenia 12 14.50 8.91 .48 1 Control 31 12.45 8.58 Schizophrenia 12 10.75 5.43 7.18 1	Schizophrenia 12 15.83 6.45 34.02 1 .00 Control 31 6.35 4.00 Schizophrenia 12 16.50 6.52 1.52 1 .23 Control 31 12.90 9.24 Schizophrenia 12 14.50 8.91 .48 1 .49 Control 31 12.45 8.58 Schizophrenia 12 10.75 5.43 7.18 1 .01

Note. Wilks Lambda = .446 For MANOVA

Abbreviation: Face Emotion Identification Test (FEIT)

Table 4
Summary of Correlations: Entire Sample (n=43)

	FEIT	Per Ab	Mag Id	Phy Anh	Soc Anh	Sch Amb
FEIT	-	18	~.11	02	14	07
Sig		.24	.47	.88	.36	.64
Per Ab	18	-	.81	.24	.42	.55
Sig	.24		.00*	.12	.01*	.00*
Mag ID	11	.81	-	.11	.26	.51
Sig	.47	.00*		.50	.09	.00*
Phy Anh	ı02	.24	.11	~	.62	.39
Sig	.88	.12	.50		.00*	.01*
Soc Anh	14	.42	.26	.62	-	.59
Sig	.36	.01*	.09	.00*		.00*
Sch Aml	o07	.55	.51	.39	.59	-
Sig	.64	.00*	.00*	.01*	.00*	

^{*} p<.05

Abbreviations: Face Emotion Identification Test (FEIT), Perceptual Aberration (Per Ab), Magical Ideation (Mag ID), Physical Anhedonia (Phy Anh), Social Anhedonia (Soc Anh), Schizotypal Ambivalence (Sch Amb)

Table 5
Summary of Correlations: Schizophrenia Group (n=12)

	FEIT	Per Ab	Mag Id	Phy Anh	Soc Anh	Sch Amb
FEIT	-	08	01	.02	26	.17
Sig		.80	.99	.96	.41	.60
Per Ab	08	-	.75	.50	.80	.61
Sig	.80		.01*	.10	.00*	.04*
Mag ID	01	.75	-	.28	.65	.50
Sig	.99	.01*		.38	.02*	.10
hy Anh	.02	.50	.28	-	.64	.60
Sig	.96	.10	.38		.03*	.04*
oc Anh	26	.80	.65	.64	<u></u>	.81
Sig	.41	.00*	.02*	.03*		.00*
ch Amb	.19	.61	.50	.60	.81	-
Sig	.60	.04*	.10	.04*	.00*	

^{*} p<.05

Abbreviations: Face Emotion Identification Test (FEIT), Perceptual Aberration (Per Ab), Magical Ideation (Mag ID), Physical Anhedonia (Phy Anh), Social Anhedonia (Soc Anh), Schizotypal Ambivalence (Sch Amb)

Table 6
Summary of Correlations: Control Group (n=31)

	FEIT	Per Ab	Mag Id	Phy Anh	Soc Anh	Sch Amb
FEIT	-	.31	.43	.08	08	.12
Sig		.09	.02*	.67	.68	.53
Per Ab	.31	_	.57	.01	.26	.29
Sig	.09		.00*	.94	.16	.11
M 1D	in	F.77		4.0		
Mag ID Sig	.43 .02*	.57 .00*	-	16	.03	.28
21g	.02	.00		.38	.88	.12
Phy Anh	.08	.01	~. 1 6	-	.61	.28
Sig	.67	.94	.38		.00*	.13
Soc Anh	08	.26	.03	.61	_	.51
Sig	.68	.16	.88	.00*		.00*
ich Amb	.12	.29	.28	.28	.51	-
Sig	.53	.11	.12	.13	.00*	

^{*} p<.05

Abbreviations: Face Emotion Identification Test (FEIT), Perceptual Aberration (Per Ab), Magical Ideation (Mag ID), Physical Anhedonia (Phy Anh), Social Anhedonia (Soc Anh), Schizotypal Ambivalence (Sch Amb)

Table 7
Regression Results for Predictors of FEIT: Total Sample

**				<u>.</u>
	R	\mathbb{R}^2	F	p Value
		.,	•	p value
	.23	05	41	0.4
	.2.7	,05	.41	.84

Predictor Variables	Beta (β)	t Value	<i>p</i> Value
Perceptual Aberration	24	81	.43
Magical Ideation	.07	.25	.80
Physical Anhedonia	.10	.50	.62
Social Anhedonia	17	73	.47
Schizotypal Ambivalence	.08	.37	.71

Table 8

Regression Results for Predictors of FEIT: Schizophrenia Group

R .78	R² .61	F 1.91	<i>p</i> Value .23	
Predictor Variables	Ве	eta (β)	t Value	<i>p</i> Value
Perceptual Aberration	.28	3	.57	.59
Magical Ideation	.30)	.74	.49
Physical Anhedonia	.24	Į.	.70	.51
Social Anhedonia	-1.	72	-2.86	.03
Schizotypal Ambivalence	1.1	.0	2.50	.05

Table 9

Regression Results for Predictors of FEIT: Control Group

R .53	R² .28	F 1.99	<i>p</i> Value .12					
Predictor Variables	Ве	eta (β)	t Value	<i>p</i> Value				
Perceptual Aberration	.1	7	.78	.44				
Magical Ideation	.39		.39		.39		1.81	.08
Physical Anhedonia	.36	6	1.61	.12				
Social Anhedonia	3	7	-1.51	.14				
Schizotypal Ambivalence	.04	1	.21	.83				