

Emotion perception and social skill over the course of psychosis: A comparison of individuals “at-risk” for psychosis and individuals with early and chronic schizophrenia spectrum illness

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Introduction. Deficits in emotion perception and social skill have been well established in schizophrenia; however, little is known about the extent of these deficits across the course of the illness; that is, prior to illness onset and as the duration of the illness increases.

Method. We compared emotion perception (i.e., the Face Emotion Identification Task [FEIT] and Face Emotion Discrimination Task [FEDT]; Kerr & Neale, 1993) and social skill (Conversation Probe role-play test) performance in four groups: individuals “at risk” for psychosis who met criteria for a prodromal state, individuals early in the course of a schizophrenia spectrum illness (SSI), individuals with a chronic SSI, and healthy control individuals.

Results. At-risk individuals did not significantly differ from control participants on emotion perception measures; however, early and chronic SSI groups performed significantly worse than controls, although not different from one another. Conversely, there was evidence that deficits in social skill are present prior to illness onset. Consistent with the findings for the emotion perception tasks, early and chronic SSI groups showed comparable levels of social skill impairment.

Conclusion. Social skill deficits may be a vulnerability marker for schizophrenia, and it appears that the initial psychotic episode represents a critical point for the emergence of emotion perception deficits in schizophrenia spectrum illnesses.

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INTRODUCTION

Individuals with schizophrenia display impairments across a wide array of functional domains including social cognition and social skill. Within social cognition, one area that has received considerable attention is that of emotion perception. Emotion perception deficits are well established among individuals with chronic schizophrenia (for reviews see Edwards, Jackson, & Pattison, 2002; Kohler & Brennan, 2004) and are also present early in the course of the illness (Edwards, Pattison, Jackson, & Wales, 2001; Kucharska-Pietura, David, Masiak, & Phillips, 2005). These deficits have been linked to impairments in the functional domain of social skill (Ihnen, Penn, Corrigan, & Martin, 1998; Mueser et al., 1996; Pinkham & Penn, 2006) and they have been consistently found among individuals with chronic schizophrenia (Donahoe et al., 1990; Ikebuchi, Nakagome, & Takahashi, 1999; Mueser, Blanchard, & Bellack, 1995); less work has examined social skill deficits in the early stages of psychosis. It should be noted, however, that impairments in general social functioning (e.g., community functioning), which encompass social skill (Bellack, Morrison, Wixted, & Mueser, 1990; Halford & Hayes, 1995; Mueser & Bellack, 1998), are present in individuals with first episode psychosis and may even be comparable to those seen in chronic schizophrenia (Grant et al., 2001). Together, these findings suggest that functional deficits may emerge particularly early in the course of the illness.

Preliminary evidence for the presence of deficits in emotion perception and social skill early in the course of schizophrenia still leaves two important questions unanswered. First, are these deficits present prior to illness onset? If so, this may provide evidence that emotion perception and social skill are vulnerability markers for schizophrenia. And second, are these deficits stable over the course of the illness or do they worsen as illness duration increases? The first question can be addressed by utilising a sample of individuals who are considered "at risk" for the development of schizophrenia, such as individuals with prodromal symptoms (Knowles & Sharma, 2004). The prodromal stage of psychosis is defined as a period of disturbance that precedes the onset of the first psychotic episode and is marked by changes in the individual's subjective experience and behaviour, such as the emergence of attenuated or brief intermittent, psychotic symptoms (Corcoran, Malaspina, & Hercher, 2005; McGorry, McKenzie, Jackson, Waddell, & Curry, 2000). Estimates for rates of conversion to psychosis vary between 10% and 50% (Yung et al., 2006, and Mason et al., 2004, respectively), with most studies supporting a figure between these two (e.g., 27% in Brewer et al., 2003; 22% in Morrison et al., 2004; and 40% in Yung et al., 2003), suggesting that a good number of individuals with prodromal symptoms go on to develop psychosis. Thus, the risk for developing psychosis is significant, and

all of these individuals are experiencing some symptoms, albeit at subthreshold levels, of psychosis.

According to Knowles and Sharma (2004), research utilising at-risk, or prodromal, individuals provides a valuable opportunity to explore vulnerability markers for psychosis. They argue that markers identified in this group should have greater predictive power due to their proximity to illness onset. In addition, Knowles and Sharma note that the study of individuals with prodromal symptoms may be more useful than traditional genetic high-risk studies given the low conversion rates of individuals who have a first-degree relative with schizophrenia (i.e., 9%) and the fact that most individuals diagnosed with schizophrenia do not have a family member with the disorder. Therefore, because identification of prodromal individuals relies on clinical rather than genetic risk, they assert that findings with this population may be more reflective of the development of schizophrenia as a whole.

The second question can be addressed by including groups of participants who vary in illness chronicity. Using this design, Kucharska-Pietura et al. (2005) found that individuals with chronic schizophrenia had greater impairments in emotion perception than individuals early in the illness. If a similar pattern emerges for social skill, this would lend indirect support for the theory that recurrent relapses are neurotoxic (Lieberman et al., 1998, 2001). Alternately, stable deficits across illness chronicity would suggest that there is something unique about the initial psychotic episode that derails functional skills, particularly if deficits increase from the prodromal to initial psychosis stages.

The primary purpose of this study was to examine emotion perception and social skill deficits over the course of schizophrenia spectrum illnesses (SSI) in four groups of participants: individuals "at-risk" for psychosis who met criteria for prodromal state, individuals early in the course of a SSI, individuals with a chronic SSI, and healthy control individuals. Based on research showing that impairments in social functioning are present, albeit in attenuated form, prior to illness onset (reviewed in Marenco & Weinberger, 2000), we hypothesised that at-risk individuals would be impaired in social skill relative to nonclinical controls, but would still show superior functioning relative to the early and chronic SSI groups. Because emotion perception is associated with neurocognition in schizophrenia (e.g., Kee, Kern, & Green, 1998), and evidence suggests that subtle neurocognitive deficits are present in individuals with prodromal symptoms (Hawkins et al., 2004), we expected a similar pattern to emerge for emotion perception deficits. In addition, we expected greater impairments in emotion perception and social skill for individuals with chronic psychosis compared to early psychosis, which is consistent with initial work in this area (Kucharska-Pietura et al., 2005).

MATERIAL AND METHODS

Participants

Participants were individuals identified as being “at risk” for psychosis ($n = 19$), individuals early in the course of SSI ($n = 21$), individuals with chronic SSI ($n = 28$), and healthy nonclinical control individuals ($n = 21$). The at-risk group was comprised of help-seeking individuals who were recruited through the UNC-Chapel Hill PRIME (Prevention through Risk Management and Education) Clinic via community and campus advertisements and presentations, and evaluated using the Structured Interview for Prodromal Symptoms (SIPS) to ensure they met COPS (Criteria of Prodromal States) criteria for a prodromal state (Miller et al., 2002). All at-risk individuals met prodromal state criteria on the basis of attenuated positive symptoms, and two individuals also met criteria for brief intermittent psychotic symptoms. No individuals met the “trait and state” risk factor criteria defined as genetic risk with functional deterioration, which includes changes in social functioning. This is important, as any observed deficits in social skill in the at-risk group will not be an artifact of the diagnostic criteria (i.e., a decline in social functioning), but likely a reflection of true impairments in social skill. The mean duration of prodromal symptoms was 3.46 ($SD = 4.17$) years.

Individuals in the early and chronic SSI groups were outpatients recruited from the Schizophrenia Treatment and Evaluation Program (STEP) at the University of North Carolina Neurosciences Hospital and had a diagnosis of a schizophrenia-spectrum disorder based on the Structured Clinical Interview for DSM-IV (SCID-P) and chart review. Individuals were recruited via clinic postings, presentations to staff, and referrals from psychiatric residents. We recruited individuals who were experiencing only minimal symptoms at the time of the study in order to provide a strict comparison to the at-risk sample who by definition should show only subclinical levels of symptoms. To be included in the early SSI group, individuals were required to have been ill for less than 5 years, which was confirmed via review of their clinical records. The mean duration of illness for the early and chronic SSI groups were 2.0 years ($SD = 1.38$) and 17.0 years ($SD = 7.72$), respectively. Of the 21 individuals in the early SSI group, 17 had a diagnosis of schizophrenia, 2 of schizoaffective disorder, and 2 of Psychosis NOS, and of the 28 individuals in the chronic SSI group, 19 had a diagnosis of schizophrenia and 9 had a diagnosis of schizoaffective disorder.

Nonclinical participants were recruited via campus mailings to university employees and screened for history of psychopathology. Individuals could not meet past or present criteria for a SSI and could not have any first-degree relatives with a psychotic or affective disorder. Exclusion criteria for

all groups included a history of neurological injury or meeting current criteria for substance abuse or dependence.

It should be noted that data from the nonclinical controls and participants with schizophrenia spectrum illnesses were drawn from a larger study (Pinkham & Penn, 2006). The current article differs from Pinkham and Penn in the following ways: (1) the primary purpose of Pinkham and Penn was to examine the functional significance of social cognition in schizophrenia, which is a different research question from that examined in the current study; (2) the current study has included a sample with prodromal symptoms; (3) the current article has divided the clinical sample into early and more chronic subsamples, whereas Pinkham and Penn collapsed across illness chronicity; (4) the current article has included only a subsample of nonclinical controls ($N = 21$, vs. $N = 49$ in Pinkham & Penn); and (5) the social skill variables were coded differently in the current study compared to Pinkham and Penn.

Measures

Emotion identification. The Face Emotion Identification Task, FEIT; Kerr & Neale, 1993) is comprised of 19 photographs of faces expressing one of six basic emotions (happy, sad, angry, afraid, surprised, and ashamed). The participant's task is to identify which emotion the face in the picture expresses.

Emotion discrimination. The Face Emotion Discrimination Task, FEDT; Kerr & Neale, 1993) consists of 30 pairs of pictures of faces expressing basic emotions and requires the participant to determine if two faces are displaying the same or different emotions.

Reliability (Cronbach's alpha) for the FEIT and FEDT was .51 and .68, respectively, which, while below par, is consistent with, and in most cases exceeds, previous research that has used these measures (Kerr & Neale, 1993; Mueser et al., 1996; Penn et al., 2000). Additionally, the widespread use of these measures and subsequent ease of comparability with other studies supports the use of these measures here despite the moderate reliability values.

Social skill. Social skill was assessed with the Conversation Probe role-play test (CP; Penn, Hope, Spaulding, & Kucera, 1994). In the CP, the participant is informed that he/she will be interacting with an unfamiliar individual (i.e., a research confederate) for 3 minutes and that the goal of the interaction is for the two individuals to get to know one another. The confederate is instructed to be pleasant, but to limit their talking to no more than 50% of time.

The role-plays were videotaped and later rated by two independent coders, who were blind to group membership, for the following components of social skill: overall social skill, clarity (clear enunciation of speech), fluency (smoothness of verbal speech; absence of verbal interruptions), appropriate affect (communication of feeling that is appropriate to the conversation through facial expression, use of gestures, and vocal tone), flat affect (amount of communication of feeling through facial expression, etc.), gaze (eye contact), engagement (the extent to which the individual appears involved in the conversation), meshing (the smoothness of turn taking), strangeness (evidence of unusual behaviour), questions (the number of questions asked by the participant throughout the role play), and social anxiety (nervousness displayed by the participant). Role-plays completed as part of a previous study were used to train raters to adequate reliability ($ICC > .70$) on all social skill variables. Interrater reliability was assessed again after all ratings had been made, and satisfactory reliability was achieved for all social skill indices (ranging from .47 to .90), with a mean reliability coefficient of .76.

Consistent with previous research in this area (Penn, Mueser, Spaulding, Hope, & Reed, 1995), scores for the social skill variables were transformed to *z*-scores and then combined into three summary scores: "global" social skill (i.e., social skill, strangeness, and social anxiety); "specific" social skills (i.e., clarity, fluency, engagement, meshing, and questions); and "nonverbal" social skills (i.e., gaze, appropriate affect, and flat affect).

Data analytic plan

In order to minimise experiment-wise error, group differences were explored using two Multivariate Analyses of Variance (MANOVAs), one on the combined emotion perception measures and one on the combined social skill indices. Follow-up univariate and post hoc tests (Tukey's LSD) were used to explore all group differences and effect sizes are reported for all analyses.

It should be noted that equipment error (i.e., video camera malfunction) resulted in missing data for some participants; thus, the sample size for each analysis varies slightly. The number of participants included in each analysis is detailed in Table 1.

RESULTS

Clinical and demographic data

Chi-square tests and a multivariate analysis of variance (MANOVA) were conducted on demographic variables. No statistical differences existed

TABLE 1
Emotion perception and social skill performance

	<i>Controls</i> (<i>n</i> = 21) <i>Mean (SD)</i>	<i>"At-risk"</i> (<i>n</i> = 19) <i>Mean (SD)</i>	<i>Early SSI</i> (<i>n</i> = 21) <i>Mean (SD)</i>	<i>Chronic SSI</i> (<i>n</i> = 28) <i>Mean (SD)</i>
Emotion perception				
FEIT ^{b,c,d,e}	14.14 (1.68)	14.05 (1.81)	12.24 (3.27)	12.57 (2.63)
FEDT ^{c,d,e}	26.67 (2.22)	26.79 (1.99)	24.86 (3.29)	24.39 (3.65)
	<i>Controls</i> (<i>n</i> = 21) <i>Mean (SD)</i>	<i>"At-risk"</i> (<i>n</i> = 14) <i>Mean (SD)</i>	<i>Early SSI</i> (<i>n</i> = 21) <i>Mean (SD)</i>	<i>Chronic SSI</i> (<i>n</i> = 28) <i>Mean (SD)</i>
Social skill				
Global ^{a,b,c,e}	0.893 (0.384)	0.002 (.711)	−0.274 (0.691)	−0.465 (0.773)
Specific ^{b,c}	0.527 (0.440)	0.053 (.638)	−0.221 (0.707)	−0.256 (0.838)
Nonverbal ^{a,b,c}	0.660 (0.530)	0.020 (.605)	−0.174 (0.622)	−0.374 (1.00)

^aControls significantly different from at-risk at $p < .05$.

^bControls significantly different from early SSI at $p < .05$.

^cControls significantly different from chronic SSI at $p < .05$.

^dAt-risk significantly different from early SSI at $p < .05$.

^eAt-risk significantly different from chronic SSI at $p < .05$.

between the four groups on gender, $\chi^2 = 4.95$, $p = .176$, or ethnicity, $\chi^2 = 16.9$, $p = .153$; however, the groups significantly differed on the combined variables of age and years of education, Wilks' $\lambda = .323$, $F(6, 168) = 21.27$, $p < .001$. Follow-up analyses indicated that the control group was significantly older than the at-risk group ($p = .004$) and as expected, that the chronic SSI group was significantly older than all other groups ($p < .001$ for all comparisons). The chronic SSI group had completed more years of education than the at-risk group ($p = .009$), and the control group had completed more years of education than all clinical groups ($p < .01$).

Severity of symptoms was assessed in all three clinical groups with the Positive and Negative Syndrome Scale (PANSS; Kay, Opler, & Fiszbein, 1992), which was administered by research clinicians trained to adequate reliability (ICCs of $> .80$ with a gold standard rater). All participants were experiencing minimal symptoms at the time of testing. A one-way MANOVA revealed no significant difference between clinical groups on the combined variables of positive, negative, and general symptoms, Wilks' $\lambda = .849$, $F(6, 124) = 1.76$, $p = .112$. Severity of prodromal symptoms was assessed with the Scale of Prodromal Symptoms (SOPS; Miller et al., 1999).

Finally, medication differences between the clinical groups were assessed by calculating the mean daily dose in chlorpromazine equivalents based on Woods (2003). A one-way ANOVA with post hoc tests revealed significant group differences in amount of medication, $F(2, 46) = 3.28$, $p = .046$, such

that the chronic SSI group was taking more medication than the early SSI group ($p = .04$). Demographic, symptom, and medication information is provided in Table 2.

Primary analyses: Emotion perception and social skill

A one-way (group: at-risk vs. early SSI vs. chronic SSI vs. healthy controls) MANOVA conducted on the FEIT and FEDT was statistically significant, Wilks' $\lambda = .850$, $F(6, 168) = 2.38$, $p = .031$, $\eta_p^2 = .078$. Univariate analyses revealed significant group differences for both the FEIT, $F(3, 85) = 3.46$, $p = .02$, $\eta_p^2 = .109$, and the FEDT, $F(3, 85) = 3.92$, $p = .011$, $\eta_p^2 = .121$. For the FEIT, post hoc analyses indicated that both the early and chronic SSI groups performed significantly worse than the healthy controls ($p = .014$ and $p = .03$, respectively) and the at-risk group ($p = .022$ and $p = .046$, respectively), but that the performance of the at-risk group did not significantly differ from healthy controls ($p = .91$). For the FEDT, post hoc analyses revealed that the chronic SSI group performed worse than both the at-risk group ($p = .008$) and the healthy controls ($p = .009$), and that the early SSI group performed significantly worse than the at-risk group ($p = .042$). The comparison between the healthy control group and the early SSI group approached statistical significance ($p = .051$). All other group comparisons were not statistically significant (see Table 1).

A one-way (group: at-risk vs. early SSI vs. chronic SSI vs. healthy controls) MANOVA conducted on the three social skill summary scores was also statistically significant, Wilks' $\lambda = .559$, $F(9, 190) = 5.71$, $p < .001$, $\eta_p^2 = .176$. Univariate analyses revealed significant group differences in global, $F(3, 80) = 18.48$, $p < .001$, $\eta_p^2 = .409$, specific, $F(3, 80) = 6.11$, $p = .001$, $\eta_p^2 = .186$, and nonverbal, $F(3, 80) = 8.05$, $p < .001$, $\eta_p^2 = .232$, social skill.

Post hoc comparisons of global social skill demonstrated that the nonclinical control participants had significantly better social skill than all other groups ($p < .001$ for all comparisons) and that the at-risk group performed significantly better than the chronic SSI group ($p = .035$). For specific social skill, post hoc analyses revealed that nonclinical controls performed significantly better than both the early and chronic SSI groups ($p = .001$ and $p < .001$, respectively), and showed a trend-level statistical difference compared to the at-risk group ($p = .05$). Finally, post hoc analyses of nonverbal social skill showed that the nonclinical control group outperformed all clinical groups ($p = .016$ for at-risk, $p = .001$ for early SSI, and $p < .001$ for chronic SSI). The three clinical groups did not significantly differ from each other on specific or nonverbal social skill (Table 1).

To rule out potential third variable effects, bivariate correlations were conducted to determine whether the clinical and demographic factors that

TABLE 2
Descriptive statistics for sample characteristics

	<i>Controls</i> (<i>n</i> = 21) <i>Mean (SD)</i>	<i>At-risk</i> (<i>n</i> = 19) <i>Mean (SD)</i>	<i>Early SSI</i> (<i>n</i> = 21) <i>Mean (SD)</i>	<i>Chronic SSI</i> (<i>n</i> = 28) <i>Mean (SD)</i>
Ethnicity				
Caucasian	16	18	13	26
African American	4	1	6	2
Asian	0	0	1	0
Hispanic/Latino	1	0	0	0
Other	0	0	1	0
Gender				
Male	10	6	14	14
Female	11	13	7	14
Age	27.62 (4.31)	21.74 (6.01)	24.62 (4.92)	39.57 (8.38)
Education	16.67 (2.85)	12.68 (2.88)	13.71 (2.33)	14.68 (2.14)
Medication*				
Typical		0	1	0
Atypical		2	15	27
Both		0	1	1
None		17	2	0
CPZ equivalent		68.75 (44.19)	250.85 (280.17)	421.73 (271.07)
PANSS				
Positive		12.21 (3.55)	13.62 (5.25)	16.56 (6.09)
Negative		10.74 (4.65)	10.90 (3.92)	12.48 (4.26)
General		25.42 (7.34)	26.24 (5.50)	31.19 (7.58)
Depression item		2.58 (1.39)	2.29 (1.62)	3.33 (1.71)
SOPS				
Positive		9.84 (6.08)		
Negative		6.05 (6.68)		
Disorganisation		3.53 (3.69)		
General		5.42 (3.47)		

*Medication data was missing for two participants in the early SSI group due to participation in a double-blind medication study.

significantly differed between groups were also significantly correlated to performance on the emotion perception and social skill measures. First, chlorpromazine equivalent was not significantly correlated to emotion perception or social skill performance, and neither age nor education was significantly correlated to performance on the FEIT or FEDT. Within social skill, better global social skill was related to being younger ($r = -.253, p = .02$), and achieving a higher level of education was related to better performance on all three indices of social skill (global: $r = .442, p < .001$, specific: $r = .458, p < .001$, nonverbal: $r = .371, p < .001$). Given these

significant relationships, the MANOVA for social skill was repeated while controlling for age and education, resulting in a multivariate group effect that remained statistically significant, Wilks' $\lambda = .709$ $F(9, 185) = 3.129$, $p = .002$, $\eta_p^2 = .109$. Therefore, the results were unchanged after controlling for age and educational level.

Supplementary analyses

The nature of prodromal symptoms is that there will be a subgroup of individuals who convert to psychosis and a larger group who do not. This provides an additional opportunity to examine the nature of vulnerability markers in schizophrenia. Therefore, we conducted a descriptive analysis to compare the emotion perception and social skill performance of at-risk individuals who converted to psychosis ($n = 5$ for emotion perception and $n = 4$ for social skill) during the course of the study (median time to conversion = 1 year) to those who did not convert ($n = 14$ for emotion perception and $n = 10$ for social skill). Given the small sample sizes, these analyses are merely heuristic in nature, and thus, conclusions regarding the findings should be made cautiously.

On the emotion perception measures, the mean performance of these groups was virtually indistinguishable; Figure 1), which supports the conclusion that deficits in emotion perception do not seem to be characteristic of individuals at-risk for psychosis. However, the pattern of means for the three indices of social skill suggests that the performance of the individuals who converted to psychosis was lower than the individuals who did not; Figure 1), lending support for the role of impaired social skill as a vulnerability marker for psychosis.

DISCUSSION

The purpose of this study was to examine the progression of emotion perception and social skill deficits across the course of schizophrenia. Our

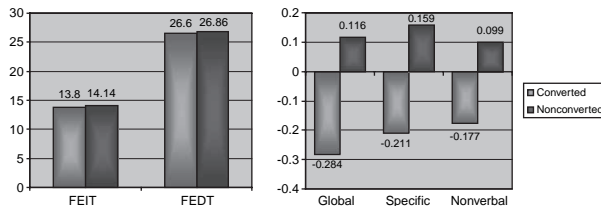


Figure 1. Emotion perception and social skill performance for at-risk group by conversion to psychosis.

results showed different patterns of impairments across these two functional domains. First, deficits in emotion perception were not observed prior to illness onset. Additionally, contrary to prediction, individuals with early SSI were as impaired in emotion perception as individuals with a chronic SSI. Second, deficits in social skill were present prior to illness onset, and consistent with the emotion perception findings, social skill was comparably impaired across early and chronic SSI groups. These findings are discussed in more detail below.

Impairments in emotion perception did not precede the onset of schizophrenia. Although unexpected, these findings are consistent with research showing that the healthy relatives of individuals with schizophrenia are not impaired on social cognitive tasks such as affect recognition (Toomey, Seidman, Lyons, Faraone, & Tsuang, 1999) and Theory of Mind (ToM; Keleman, Keri, Must, Benedek, & Janka, 2004). Therefore, deficits in emotion perception may not be a vulnerability marker for schizophrenia. Rather, they may be related to the illness itself, and the first episode may serve as a critical point at which emotion perception deficits emerge.

Impairments in emotion perception were of a comparable magnitude across individuals with schizophrenia, irrespective of whether they are early or later in the course of their illness. This pattern of findings is inconsistent with those recently reported by Kucharska-Pietura et al. (2005); however, differences in sample characteristics and emotion perception tasks may explain these discrepancies. First, Kucharska-Pietura et al. utilised samples of inpatients, whereas the present study included only outpatients. Second, Kucharska-Pietura et al. employed a task of facial emotion perception, the Facial Emotion Recognition Task, (FERT), which was more complex than the one used in the present study. Additionally, participants in Kucharska-Pietura were presented with each face for 10 s and then asked to respond during the next 10 s. This is in contrast to the present study in which participants could view, and respond to, each face for as long as they wished. Finally, the participants in the Kucharska-Pietura study showed greater levels of negative symptoms than those in the present study, and their chronic sample had more negative symptoms than the early group. Given the evidence for a relationship between negative symptoms and deficits in emotion perception (Mandal, Jain, Haque-Nizami, Weiss, & Schneider, 1999; Sachs, Steger-Wuchse, Kryspin-Exner, Gur, & Katschnig, 2004), it is possible that an interaction between symptomatology and task difficulty may have contributed to findings of greater impairment in the chronic group.

A somewhat different pattern of findings emerged with respect to social skill. Our hypothesis that individuals at-risk for psychosis would be impaired in social skill relative to nonclinical controls was supported. These findings, in conjunction with the descriptive, supplementary analyses, provide tentative support for the role of social skill as a potential vulnerability

marker for schizophrenia. The study findings are also consistent with Grant et al. (2001), who found general social functioning impairments in first episode psychosis which were comparable in magnitude to those of more chronically ill individuals. Overall, our findings suggest that social skill deficits might reflect a trait characteristic of schizophrenia (which potentially can be exacerbated once individuals formally convert to psychosis).

Overall, these findings underscore the need to target social functioning in treatment. While intervention during the prodromal phase is not without controversy (e.g., Warner, 2005), the findings indicate that for help-seeking at-risk individuals, social skills training might be a worthy supplement to CBT (Morrison et al., 2004). The case for addressing social skill deficits is even more compelling for first-episode psychosis, as these deficits remain despite symptomatic recovery (discussed in Penn, Waldheter, Mueser, Perkins, & Lieberman, 2006), clients identify social relationships as an important area of concern (Macdonald, Sauer, Howie, & Albiston, 2005; Mackrell & Lavender, 2004), there is a need to make CBT more sensitive to the functional impairments resulting from an initial psychotic episode (Addington & Gleeson, 2005), and social skills training has been shown to specifically improve social functioning in schizophrenia (Bellack, 2004; Mueser & Penn, 2004).

Future research should address the limitations of this study. First, our tasks of emotion perception and social skill were not matched for difficulty or true score variance, and thus the evidence presented here for a differential deficit must be interpreted cautiously. Second, although we had adequate power to detect group differences in our primary analyses and all statistical tests yielded moderate to large effect sizes (Cohen, 1988), our supplemental, descriptive analyses were underpowered. Thus, these results require replication with a larger sample. Third, both the early and chronic groups were comprised of participants both with schizophrenia and schizoaffective disorders. However, post hoc analyses showed that the results remained stable even after including only participants with schizophrenia, suggesting that sample heterogeneity did not affect our results. Fourth, future studies are likely to benefit from using more complex measures of emotion perception and more extensive batteries that are able to tap into subtle deficits that may not have been captured here (e.g., Kington, Jones, Watt, Hopkin, & Williams, 2000). Both the FEIT and FEDT assess perception of only basic emotions (i.e., happy, sad, etc.) and both utilise a simple correct vs. incorrect scoring system. It is possible that more nuanced measures that assess complex social emotions (i.e., guilty, arrogant, admiring, etc.) may reveal emotion perception deficits in an at-risk sample, as well as subtle changes in emotion perception abilities over the course of illness. Additionally, as cognitive ability may influence emotion perception and social skill, future work should also include a neurocognitive battery that may clarify the

potential role of cognitive deficits. Fifth, no controls for emotional state or comorbid depression were included. Although assessment of the PANSS depression item suggests that each clinical group was experiencing only subclinical levels of depression and that the clinical groups did not differ from one another, $F(2, 64) = 2.781$, $p = .07$, we cannot rule out the possibility that current emotional state, and in particular depression, may have influenced social skill performance and contributed to the deficits seen here. Finally, the cross-sectional nature of this study limits the conclusions that can be drawn concerning the actual progression of emotion perception and social skill deficits over time. Future work should capitalise on longitudinal methods to more fully examine the course of emotion perception (and other aspects of social cognition) and social skill impairments from at-risk to chronic states.

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