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Qualitatively Distinct Factors Contribute to Elevated Rates of Paranoia in Autism and Schizophrenia

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A converging body of clinical and empirical reports indicates that autism features elevated rates of paranoia comparable to those of individuals with paranoid schizophrenia. However, the distinct developmental courses and symptom manifestations of these two disorders suggest that the nature of paranoid ideation may differ between them in important and meaningful ways. To evaluate this hypothesis, we compared patterns of responses on the Paranoia Scale between actively paranoid individuals with schizophrenia (SCZP), individuals with schizophrenia who were not actively paranoid (SCZNP), adults with an Autism Spectrum Disorder (ASD), and healthy controls. Despite an overall similar level of heightened paranoia in the ASD and SCZP groups, discriminant correspondence analysis (DiCA) revealed that these groups were characterized by unique underlying factors. Paranoia in the SCZP group was defined by a factor based upon victimization, suspicion, and threat of harm. Whereas paranoia in the ASD group was partially characterized by this factor, it was distinguished from SCZP by an additional pattern of responses reflective of increased social cynicism. These findings indicate that paranoia in ASD is supported by qualitative factors distinct from schizophrenia and highlight mechanistic differences in the formation of paranoid ideation that may inform the development of disorder-specific treatments.

Keywords: paranoia, discriminant correspondence analysis, cynicism, social cognition

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Wing and colleagues define paranoia as "the belief that someone, some organization, or some force or power is trying to harm oneself in some way" (Wing, Cooper, & Sartorius, 1974, p. 170). Under this definition, paranoia is conceived of specifically as persecutory ideation rather than the more general meaning of paranoia as it was originally coined and used within psychiatry, namely as an encompassing term that included a variety of delusional beliefs including persecutory, grandiose, and jealous delusions (Kraepelin, 1907). This shift in the usage of the terms paranoia and paranoid are in line with current literary and popular use in which both terms have come to be synonymous with irrationally suspicious and distrustful thinking (Manschreck & Kahn, 2006). Capitalizing on this popular conceptualization of irrationality, Freeman and colleagues have more recently clarified the definition of Wing and colleagues by specifying that such beliefs should be unfounded (Freeman et al., 2008), a caveat that also emphasizes the potential abnormality and delusional aspects of paranoid ideation. In line with these definitions, paranoia is well recognized and accepted as a symptom of psychosis, and particularly schizophrenia, which includes the designation "paranoid" as a diagnostic subtype (*DSM–IV–TR*). Increased rates of paranoia are not specific to schizophrenia, however, and are also evident in anxiety and depression (van Os et al., 1999), neurological disorders such as dementia (Ballard et al., 2000), and consistent with the continuum model of paranoid ideation (e.g., Fenigstein & Vanable, 1992; Freeman et al., 2005), even subsets of nonclinical healthy individuals (Ellett, Lopes, & Chadwick, 2003; Freeman, 2007).

Elevated rates of paranoid ideation have also been noted in clinical reports of individuals with Asperger's syndrome (Hare, 1997; Wing, 1996; Woodbury-Smith, Boyd, & Szatmari, 2010), and recent empirical studies of adolescents and adults with autism spectrum disorders (ASD) have provided support for these obser-

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vations. Specifically, Abell and Hare (2005) asked a large sample of individuals with ASD to complete the Peters' Delusions Inventory (Peters, Joseph, & Garety, 1999), a self-report measure that assesses a variety of delusional beliefs. Individuals with ASD scored well above previously established norms (Peters et al., 1999) for healthy individuals, and suspicious and persecutory beliefs were among the most commonly endorsed delusional categories. Individuals with ASD also reported higher levels of distress and preoccupation related to these beliefs and greater conviction in these beliefs than those reported by the healthy normative sample. Using a more specific measure of paranoid ideation, the Paranoia Scale (Fenigstein & Vanable, 1992), both Blackshaw, Kinderman, Hare, and Hatton (2001) and North, Russell, and Gudjonsson (2008) reported elevated rates of paranoia in ASD compared to healthy controls. Two additional studies compared individuals with ASD to individuals with schizophrenia (Craig, Hatton, Craig, & Bentall, 2004; Pinkham, Hopfinger, Pelphrey, Piven, & Penn, 2008). Whereas Craig, Hatton, Craig, and Bentall (2004) found that their ASD group scored significantly higher than controls but below individuals with schizophrenia, Pinkham et al. (2008) reported nearly identical scores on the PS by individuals with ASD and those with paranoid schizophrenia.

These studies suggest overlap in paranoid ideation in ASD and schizophrenia, however, the degree and pattern of this similarity remains unexplored. Disparities in clinical presentations between individuals with these disorders, including different ages of onset, developmental course, and primary symptom manifestation (see Sasson, Pinkham, Carpenter, & Belger, 2011, for a discussion of overlap and disparity between ASD and schizophrenia), would suggest that the nature and etiology of paranoid ideation in both disorders may differ substantially. Some support for this hypothesis can be garnered by considering explanatory models of paranoid thinking in schizophrenia. These models implicate both impairments in Theory of Mind (ToM) and biased attributional style as potential contributing factors (Bentall, Corcoran, Howard, Blackwood, & Kinderman, 2001; Bentall et al., 2009; for reviews, see Freeman, 2007; Penn, Sanna, & Roberts, 2008). Deficits in ToM are well established in both schizophrenia and ASD (Bora, Yucel, & Pantelis, 2009; Yirmiya, Erel, Shaked, & Solomonica-Levi, 1998) but, the limited work that has examined attributional biases in ASD has failed to find evidence of any attributional abnormalities (Blackshaw et al., 2001; Craig et al., 2004). Such findings question the applicability of schizophrenia-based models to ASD and suggest that the paranoia seen in ASD likely stems from different mechanisms than those of schizophrenia. Thus, although some prior evidence suggests quantitative similarities in paranoia between ASD and schizophrenia, no studies have directly investigated whether qualitative differences underlie this similarity.

The identification of disorder-specific mechanisms underlying paranoia in ASD and schizophrenia would not only help reveal distinctions of a shared clinical symptom, but also inform novel avenues for targeted treatments. Perhaps not surprisingly, increased paranoia has been associated with impaired work and social functioning as well as reduced psychological well being (Martin & Penn, 2001; Olfson et al., 2002; Rossler et al., 2007), suggesting that paranoia may contribute to a reduced quality of life in individuals with these disorders. Enhancing our understanding of how patterns of paranoia differ between ASD and schizophrenia may facilitate the development of specialized interventions that result in improved psychosocial functioning for individuals with these disorders. A novel intervention targeting social–cognitive impairment and social functioning, Social Cognition and Interaction Training (SCIT: Roberts, Penn, & Combs, 2004), provides a good example of this need. SCIT was originally developed for individuals with psychotic disorders but has recently been applied to adults with autism (SCIT-A: Turner-Brown, Perry, Dichter, Bodfish, & Penn, 2008). Even though SCIT-A resulted in improved social–cognitive performance, Turner-Brown and colleagues noted that several modifications were necessary to make SCIT applicable to individuals with autism. These authors specifically cite differences in the mechanisms underlying incorrect social judgments (i.e., the tendency to jump to conclusions in individuals with schizophrenia but the tendency of individuals with autism to overvalue irrelevant information) as one area requiring modification. It is possible that a similar understanding of paranoia and the mechanisms underlying paranoia in schizophrenia and ASD may lead to interventions designed to reduce paranoid thinking that are optimized for each population.

As a first step, the present study seeks to examine potential similarities and differences in paranoid ideation between individuals with ASD and individuals with schizophrenia by conducting a detailed comparison of patterns of responses on the Paranoia Scale (PS). Here, we use discriminant correspondence analysis (DiCA: Abdi, 2007; Williams, Abdi, French, & Orange, 2010), a nonparametric factor analytic technique designed to identify the qualitative variables that discriminate between groups. It is anticipated that even though individuals with ASD and individuals with paranoid schizophrenia will share similar elevated summary scores on the PS, qualitative differences will emerge highlighting distinct disparities in the types of paranoid thoughts endorsed by both groups that reveal dissociative patterns of paranoid ideation. Such differences would provide further support that unique mechanisms underlie the increased paranoid ideation reported in ASD and schizophrenia.

Method

Participants

A total of 101 individuals participated and comprised four groups: individuals with ASD (ASD: $n = 18$), individuals with schizophrenia or schizoaffective disorder with pronounced paranoia (SCZP: $n = 24$), individuals with schizophrenia or schizoaffective disorder without paranoid symptoms (SCZNP: $n = 30$), and healthy control individuals (CON: $n = 29$). Individuals with ASD were recruited from the Dallas-Fort Worth metroplex and from the University of North Carolina (UNC) Autism Research Registry in conjunction with TEACCH (Treatment and Education of Autistic and related Communication-handicapped Children) clinics. All individuals in the ASD group had a *DSM–IV* diagnosis of Autistic Disorder or Asperger's Syndrome made by a licensed clinician experienced in the assessment and diagnosis of autism. Diagnoses were confirmed via the Autism Diagnostic Interview-Revised (ADI-R: Lord, Rutter, & Le Couteur, 1994) or the Autism Diagnostic Observation Schedule (ADOS: Lord, Rutter, DiLavore, & Risi, 1999) except in three cases, who, per TEACCH protocol,

did not complete these instruments because their clinical diagnoses were not questioned.

Individuals in the schizophrenia groups were recruited from the Schizophrenia Treatment and Evaluation Program at the University of North Carolina Neurosciences Hospital, the Schizophrenia Research Center of the University of Pennsylvania Medical Center, and Metrocare Services, a nonprofit mental health services provider organization in Dallas County, Texas. In all cases, diagnoses were confirmed via the Structured Clinical Interview for *DSM–IV* (First, Spitzer, Gibbon, & Williams, 2002) or the Diagnostic Interview for Genetic Studies (Nurnberger et al., 1994). Schizophrenia participants were placed into the SCZP and SCZNP groups based on the severity of paranoid symptoms at the time of testing as assessed by the Positive and Negative Syndrome Scale (PANSS: Kay, Opler, & Fiszbein, 1992). Participants reporting clinically significant levels of paranoia, scoring at least a 4 or above on the suspiciousness/persecution item, constituted the SCZP group, and individuals scoring a 2 or below on this item, indicating absence or only subclinical levels of paranoia, constituted the SCZNP group.

Importantly, participants were not screened for level of paranoid ideation prior to participating in the study and no attempts were made to specifically target individuals showing high or low levels of paranoia. It should also be noted that data from 48 of the participants (12 in each group) has previously been reported (Pinkham et al., 2008); however the remaining 53 subjects were recruited uniquely for this study, and as reviewed above, Pinkham et al. (2008) only reported comparable PS summary scores for the ASD and SCZP groups. No further analysis of these data was conducted, and thus the present study constitutes a novel investigation.

Measure

The Paranoia Scale is a 20-item self-report measure of paranoid ideation that was originally derived from the Minnesota Multiphasic Personality Inventory (MMPI). Participants are asked to respond to each item using a 5-point scale ranging from 1 (*not at all applicable to me*) to 5 (*extremely applicable to me*). Scores range from 20 to 100 with higher scores indicating higher levels of paranoia. The PS has good psychometric properties, including high reported internal consistency ($\alpha = .84$), adequate retest reliability ($r = .70$) and appropriate convergent and discriminant validity (Fenigstein & Vanable, 1992). Internal consistency for the present sample was excellent ($\alpha = .96$).

Statistical Analyses

To assess the first part of our hypothesis, namely that the ASD and SCZP groups would have elevated total scores on the PS relative to SCZNP and CON, we conducted a one-way ANOVA followed by Tukey's HSD post hoc comparisons. Next, we used discriminant correspondence analysis (DiCA) to assess qualitative differences in the type of paranoia endorsed by our groups. We used an implementation of DiCA available in the statistical computing language [R] (R Development Core Team, 2010, see http://www.utdallas.edu/~derekbeaton; DiCA is also available for MATLAB at http://www.utdallas.edu/~herve). Because DiCA has recently been explained in detail elsewhere (Williams et al., 2010), we will limit our current discussion to factors specific to this study as well as a brief overview of DiCA and data interpretation.

Data preprocessing. Data were first recoded into question-response levels, to account for each possible answer to every question. In doing so, each of the 20 questions is represented by five columns, one column for each possible response, yielding 100 columns. For example, a response of 1 is recoded as 1, 0, 0, 0, 0 and a response of 3 is recoded as 0, 0, 1, 0, 0. This process is called nominal (or disjunctive) coding and expresses the qualitative differences in the data (Abdi & Valentin, 2007; Lebart, Morineau, & Warwick, 1984). For example, two individuals who have an overall score of 24 are numerically identical but may have arrived at this score via very different response patterns. One individual could have obtained this score by responding with 1 to questions 1 through 19 and 5 to question 20, whereas the second individual could have responded with 5 to the first question and 1 to questions 2 through 20. Here, the identical summary scores obscure that these individuals gave highly paranoid responses to one particular, but very different, question. The use of nominal coding therefore permits examination of such differences between individuals and produces nominal variables that can be analyzed with multivariate techniques such as DiCA. For additional recoding details, please see Supplemental Materials: Recoding Example.

DiCA. DiCA is utilized when the data are observations that belong to predefined groups and when the goal is to identify the variables that best discriminate between these groups (Williams et al., 2010). DiCA is an extension of Correspondence Analysis (CA; Greenacre, 2007), and these techniques are to qualitative data what discriminant analysis and principal components analysis are (respectively) to quantitative data (Abdi & Williams, 2010a; Williams et al., 2010). As applied here, we want to perform a discriminant analysis (DA) between our groups (i.e., ASD, SCZP, SCZNP, CON) but because the data (paranoia scale) are qualitative (nominal) we required DiCA as opposed to standard DA.

In order to perform DiCA, we first create a matrix that represents each group (i.e., ASD, SCZP, SCZNP, CON) as the sum of its observations. In our case, this matrix is a 4 row by 100 column contingency table that contains the number of occurrences of each question-response level (columns) for each group (rows; see Supplemental Material: Calculation of the Contingency Table for additional information). This process constitutes the first step of DiCA, which is followed by a CA on the groups by variable matrix (which solves the discriminant problem because it maximizes the difference between groups).

Briefly, CA is specifically designed for use with qualitative data expressed either as frequencies (as in Churchill & Behan, 2010) or nominal/categorical variables (as in Chan, Gelernter, Oslin, Farrer, & Kranzler, 2011; Dumais et al., 2011). In CA, the rows (observations) are first normalized to *their relative frequency*. That is, each element (variable) of each row (observation) is expressed as the proportion of the *row* frequencies (i.e., each element of a row is divided by the total of the row). Next, the row mean (i.e., barycenter) is subtracted from each row in order to center the data. Finally, the (row normalized and centered) data matrix is decomposed with the generalized singular value decomposition. See Supplemental Material: Table Normalization and Decomposition for additional information.

DiCA—like CA or PCA—produces new variables, called factors, which combine the original variables. However, it should be

noted that CA and PCA analyze *individuals*. DiCA—like DA or MANOVA—analyzes *groups* and not individuals. Thus, power is determined differently between these analyses. PCA (and other factor analytic techniques) derives power either as a proportion of the number of observations (individuals) to measures or a large N (Costello & Osborne, 2005; though this point is arguable, see Chi, 2012). In contrast, DiCA—like DA—derives power as a function of groups and observations and is calculated in the same fashion as a MANOVA (Chi, 2012; Hwang, Schmitt, Stephanopoulos, & Stephanopoulos, 2002).

The factors produced by DiCA are orthogonal (i.e., uncorrelated) to one another. The first factor explains the maximum amount of variance in the data, and each subsequent factor explains the largest possible amount of remaining variance. The values of the factors for groups and variables are called factor scores, and when plotted, these factor scores give factor maps. These maps show the spatial relationship for groups and variables. Factor scores can also be computed for the original observations (i.e., here the participants), which can also be plotted on the factor maps.

The interpretation of factors is based on the factor maps and is facilitated by a parameter called *contribution* (which is akin to squared loadings in PCA, see Abdi & Williams, 2010c) that expresses the proportion of variance of a factor explained by a group or a variable. The interpretation is thus primarily based on groups and variables that contribute *above average* to a factor. As applied here, we have four groups and 100 variables (i.e., 20 questions with five response choices each). Therefore, a group that contributes more than (1/4 or 25%) of the variance to a factor is contributing above average, and a variable that contributes more than 1/100 (or 1%) is contributing above average. Second, as factor maps show the relationships between groups and variables along a specific factor, we can conclude that when a variable and a group fall close together, that this variable is more closely associated to this group than to the other groups. However, it is important to note that the distance between a group and a question-response level is not directly interpretable; rather, a question-response level near a group is more likely to be associated to that group than any other group.

Finally, it is important to note that the results of DiCA are descriptive (fixed-effects) and not inferential (random-effects). In order to infer from the results of DiCA, we employed four[1] statistical methods: (a) a permutation test of R^2 (Williams et al., 2010); (b) a permutation test to determine which, if any, factors are systematic and significant; (c) the Jackknife procedure (a leave-one out cross-validation approach; Williams et al., 2010, see also Abdi & Williams, 2010b); and (d) the Bootstrap (Efron & Tibshirani, 1993; Chernick, 2008).

For DiCA, R^2 is computed as between-groups variance/total variance (à la ANOVA) and represents the reliability of assignment of individuals to groups. A distribution of R^2 is computed by permuting group labels to observations. The same permutation builds a distribution of eigenvalues (variance explained) *per factor*. The distribution of R^2 and the eigenvalues can be used to test whether the original R^2 and eigenvalues are systematic or can be attributed to chance.

The Jackknife procedure leaves out each observation, in turn, and performs a new DiCA on the data without the left out observation. The factor scores of the left-out observation are then estimated from these data. This is performed for every observation.

The Bootstrap quantifies the separability of the groups and determines its significance. Statistical significance for the Bootstrap is assessed by confidence intervals displayed on the factor maps with peeled convex hulls (Greenacre, 2007). When the convex hulls of two groups do not intersect in at least one factor map, then these groups are significantly different (Abdi, Dunlop, & Williams, 2009). Additional information detailing inferential testing is provided in Supplemental Material.

Results

All four groups did not differ in gender, $\chi^2(3, N = 101) = 3.14$, $p = .37$, or ethnicity, $\chi^2(6, N = 101) = 6.12$, $p = .41$. They did however differ significantly in age, $F(3, 97) = 3.21$, $p = .027$, and level of education achieved, $F(3, 97) = 9.79$, $p < .001$. Tukey's post hoc comparisons revealed that the ASD group was significantly younger than the SCZNP group ($p = .025$), but no other direct group comparisons of age reached statistical significance. As expected, the CON group completed more years of education than all three clinical groups ($p < .02$ for all comparisons), who did not differ from each other ($p > .5$ for all comparisons).

Between the two schizophrenia groups, the SCZP group showed greater severity of both positive, $F(1, 52) = 51.61$, $p < .001$, and general symptom clusters, $F(1, 52) = 35.39$, $p < .001$. The difference in severity of general symptoms was no longer statistically significant after controlling for level of paranoia, $F(1, 51) = 3.08$, $p = .085$. The difference in positive symptom severity, however, remained, $F(1, 51) = 4.17$, $p = .046$. Groups did not differ in severity of negative symptoms $F(1, 52) = 1.83$, $p = .18$, medication dosage as indexed by Chlorpromazine equivalents, $F(1, 51) = .006$, $p = .94$; (Woods, 2003), or in the proportion of individuals diagnosed with schizophrenia versus schizoaffective disorder, $\chi^2(1, N = 54) = 1.20$, $p = .27$. Demographic and clinical information is provided in Table 1.

A one-way ANOVA comparing overall PS scores revealed a significant difference between our four groups, $F(3, 97) = 18.05$, $p < .001$. Tukey's HDS post hoc comparisons indicated that this effect was driven by significantly higher scores in the ASD and SCZP groups than in the SCZNP and CON groups ($p < .01$ for all comparisons). CON and SCZNP did not differ ($p = .67$), and importantly, mean scores for the ASD and SCZP groups did not differ ($p = .39$).

DiCA of our data produced three factors that we have named *Paranoia* (which explains 70.76% of the variance), *Cynicism* (which explains 16.48% of the variance), and *Insightful Acknowledgment* (which explains 12.75% of the variance). Recall that interpretation of the factors relies on the contribution of groups and variables (question-response levels) to each factor as well as the spatial relationships between groups and variables on each of these factors. The distance between a group and a question-response level is not directly interpretable; rather, a question-response level (e.g., 20.5)

[1] In addition to the four approaches described here, we also performed a series of split-half analyses (suggested by one of the reviewers). For the sake of brevity and clarity, we report this last analysis in the supplemental material.

Table 1
Demographic and Clinical Information

	ASD (n = 18) Mean (*SD*)	SCZP (n = 24) Mean (*SD*)	SCZNP (n = 30) Mean (*SD*)	Control (n = 29) Mean (*SD*)
Demographic Information				
Gender				
Male	17	21	25	22
Female	1	3	5	7
Ethnicity				
Caucasian	14	15	21	20
African American	2	9	7	8
Other	2	0	2	1
Age[a]	24.56 (6.0)	27.33 (5.96)	29.87 (7.18)	29.21 (5.35)
Education[b]	14.22 (2.39)	13.29 (2.44)	13.57 (2.47)	16.24 (1.70)
Clinical Information				
Symptom Severity				
Positive[c]		17.79 (3.68)	10.80 (3.45)	
Negative		12.29 (5.94)	10.50 (3.73)	
General[c]		32.38 (5.72)	24.37 (4.16)	
CPZ Equivalent		335.75 (240.40)	341.24 (287.96)	
SCZ Diagnosis				
Schizophrenia		18	26	
Schizoaffective Disorder		6	4	
Paranoia Scale[d]	49.61 (18.68)	57.17 (20.86)	34.23 (12.59)	29.72 (8.77)

Note. Medication information was missing for one patient in the SCZNP group, and no individuals in the ASD were taking antipsychotics. Symptom severity scores are presented as the sum of PANSS items for positive, negative and general symptom clusters.
[a] ASD significantly different from SCZNP at $p < .05$. [b] Controls significantly different from all clinical groups at $p < .02$. [c] SCZP significantly different from SCZNP at $p < .001$. [d] ASD and SCZP significantly different from SCZNP and controls at $p < .01$.

near a group (e.g., SCZP) indicates that this particular question-response level is more highly associated with one group (e.g., SCZP) than another group (e.g., CON). Our discussion of each factor thus focuses on contributions (to variance) and factor maps.

Factor 1—Paranoia

Factor 1 separates the SCZP and the CON groups as they are located at opposite sides of this factor (Figure 1A), and together, these two groups account for over 80% of the variance of Factor 1 (see Table 2 for the amount of variance contributed by each group to each factor). As can be seen in Figure 1A, the groups exist across a continuum on Factor 1, with SCZNP and CON on one side and SCZP on the other, and ASD in between. Factor 1 has been labeled "Paranoia" as it accounts for the majority of variance in the data and appears to reflect the original intent of the PS, namely, to assess a spectrum of severity along the hypothesized homogenous variable of paranoid ideation. This spectrum is supported by the separation of the groups (Figure 1A), and individuals (Supplemental Figure 1) in each of the groups, along this factor.

Further support for this spectrum is garnered from examination of the question-response levels that contribute above average variance to Factor 1. Supplemental Table 8 lists the above average contributing question-response levels. These questions include highly paranoid responses (i.e., 4 = *Very much applicable* and 5 = *Extremely applicable*) printed in **bold** and questions that indicate nonparanoid responses (i.e., 1 = *Not At All Applicable* and 2 = *Somewhat Applicable*) printed in regular font. Every question in the PS is represented, except Question 12. As can be seen in Figure 1A, the question-response levels with above-average contributions are largely dichotomized by paranoid and nonparanoid responses that map onto the levels of paranoid ideation seen in our groups.

Factor 2—Cynicism

Consistent with our hypothesis that qualitative differences would emerge between the types of paranoia experienced by individuals with ASD and individuals with SCZP, Factor 2 separates ASD and SCZP. These two groups occupy opposite sides of Factor 2 (Figure 1B), and together account for approximately 69% of the variance of this factor (see Table 2). Factor 2 shows that there are two types of paranoid ideation driving elevated paranoia in these clinical groups: one related to ASD and one related to SCZP. Importantly, at 46%, the ASD group is the only group to provide an above average contribution to this factor suggesting that its responses heavily influence separation on this factor.

Analysis of the question-response levels that contribute to Factor 2 and share the same side of factor space as the ASD group indicates that ASD paranoia is reflective, in some respects, of social cynicism (Bond, Leung, Au, Tong, & Chemonges-Nielson, 2004; Leung & Bond, 2004), leading us to label factor two "Cynicism." These question-response levels, such as 12.5 ("Most people make friends because friends are likely to be useful to them") and 16.4 ("I tend to be on my guard with people who are somewhat more friendly than I expected"), reflect a high level of suspiciousness about the intentions and motivations of others within social

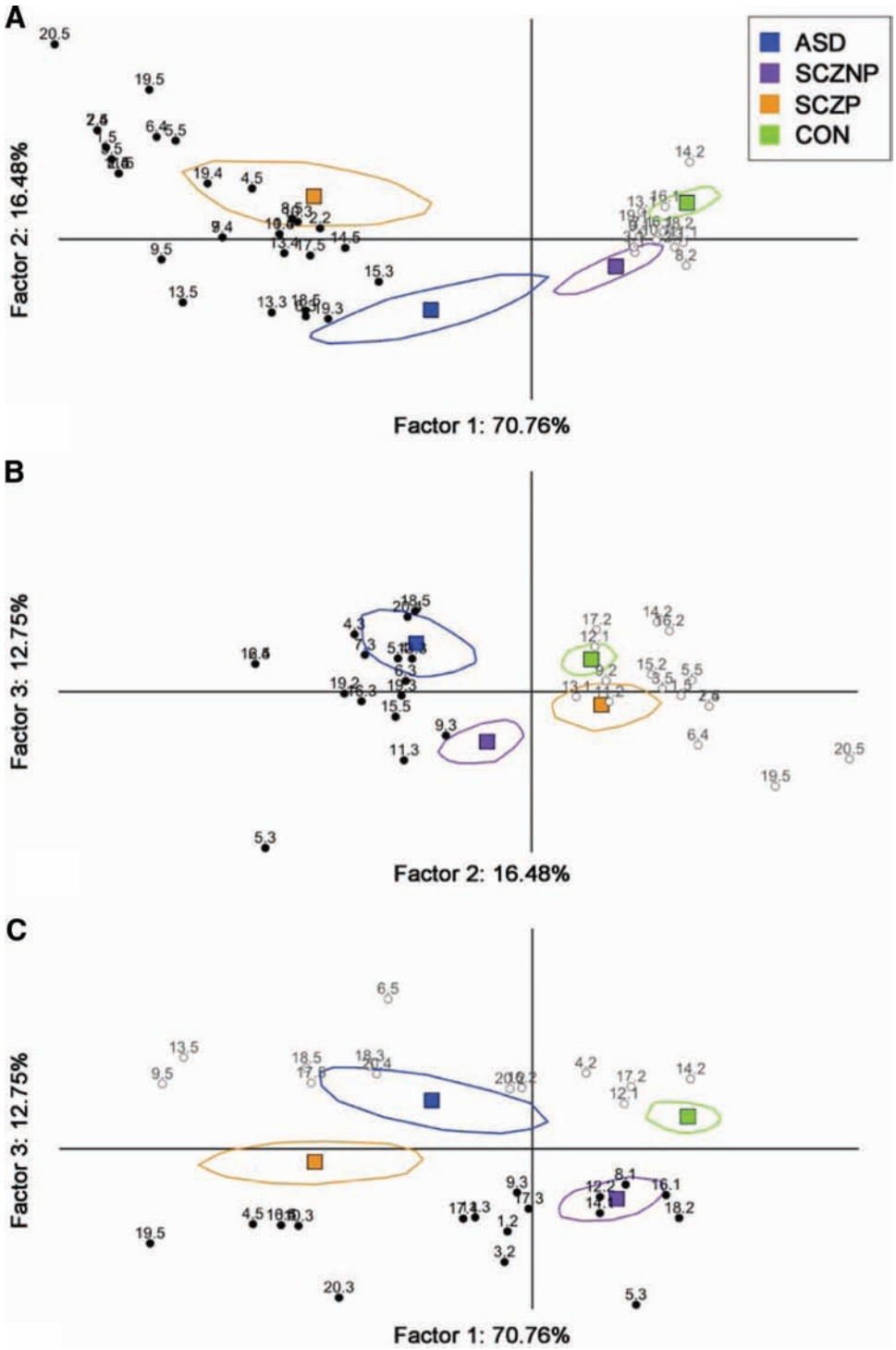

Figure 1 (*opposite*).

Table 2
Contributions of Variance by Groups to Each Factor

Group	Paranoia	Cynicism	Insight
ASD	8.17%	**46.11%**	**27.88%**
SCZNP	9.54%	11.39%	**49.35%**
SCZP	**50.91%**	22.63%	2.69%
CON	**31.37%**	19.85%	20.05%

Note. Bolded contribution values indicate that they are above average (>25%) and therefore meaningful in interpreting the factor.

situations. These levels contrast with those at the opposite end of Factor 2, such as 19.5 ("I am bothered by people outside, in cars, in stores, etc. watching me"), which capture a more traditional conceptualization of paranoia related to unfounded beliefs of being followed, persecuted or harmed. Figure 1B shows the above average question-response levels in factor space, and Supplemental Table 9 provides the response levels related to socially cynical paranoia (**bold**) and traditional paranoia (regular font).

Factor 3—Insightful Acknowledgment

Factor 3 separates our two SCZ groups from the nonpsychotic groups and is driven primarily by the SCZNP group (Figure 1C), which accounted for approximately 49% of the variance explained by this factor (see Table 2). Examination of the question-response levels contributing above average to this factor demonstrates moderate endorsements in the SCZNP group (Supplemental Table 10; Figure 1C), such as levels 5.3 ("My parents and family find more fault with me than they should"), 20.3 ("I have often found people jealous of my good ideas just because they had not thought of them first"), 18.2 ("People often disappoint me"), and 3.2 ("I believe that I have often been punished without cause"). The moderate levels of endorsement of these items, and several others, are suggestive of a realistic acknowledgment of the poorer quality of life that typically occurs as a result of mental illness and has prompted us to conceptualize this factor as "Insightful Acknowledgment."

Inferential Testing

In order to assess the significance of the relationships observed in the descriptive analysis, we also conducted four inferential tests. We used permutation tests to evaluate R^2 and the percentage of variance extracted by the factors. First, the value of $R^2 = .4207$ was significant with $p < .001$, which indicates that a priori group assignment (of individuals) was reliable and systematic. Second, we found that Factor 1 was significant at $p < .001$, Factor 2 at $p = .03$, and Factor 3 at $p = .016$. These results indicate that the factors themselves are systematic and not due to chance.

Third, the Jackknife procedure produced a confusion matrix (i.e., group assignment) for the *predicted* observations. When comparing the fixed-effects confusion matrix (Table 3, 70% overall classification, $\chi^2 = 122.44$, $p < .0004$) to the random-effects (Jackknife) confusion matrix, (as expected) the random-effect model was less impressive than the fixed-effect model. However, the assignment of new observations was still significantly better than chance (Table 4, 45% overall classification vs. 25% for chance, $\chi^2 = 44.33$, $p < .0004$). We noted that when a "new" ASD observation (i.e., left out observation) was estimated and assigned to a group, the assignment was most often to the SCZP group. Even though ASD was not perfectly classified, this result indicates that ASD *individuals* exhibit levels of paranoia above those that might be expected (i.e., as much as a clinically paranoid group: SCZP). Additionally, confusion with SCZP is likely due to the large amount of variance explained by Factor 1—Paranoia (i.e., ~71%) relative to the other two factors and that SCZP and ASD looked most similar on this factor. However, the effect for "new" individuals does not describe the group level aspects of ASD-paranoia (i.e., Factor 2—Cynicism). The group level aspects are best described by the fixed-effects with above average contributing variables for Factor 2 and bootstrap analyses.

Finally, results from the bootstrap analysis (Figures 1A-C) indicate that our groups significantly differ on all three factors. As seen, the peeled confidence convex hulls for each group do not overlap with any other group, indicating that the groups significantly differ at $p < .05$ on each factor.

Discussion

The current investigation compared patterns of paranoia in individuals with Autism Spectrum Disorders (ASD), actively paranoid patients with Schizophrenia (SCZP), patients with schizophrenia who were not actively paranoid (SCZNP), and healthy controls (CON). Individuals with ASD and those with SCZP

Figure 1 (*opposite*). DICA maps of groups and above average variables (question-response levels) on each derived factor. Groups shown in factor space with 95% peeled convex hulls confidence intervals representing the range in which 95% of all bootstrap samples would fall. Nonoverlapping hulls indicate statistical significance between groups at $p < .05$. ASD = Autism Spectrum Disorders; SCZNP = schizophrenia without paranoia; SCZP = schizophrenia with paranoia; CON = control. A) Factor 1 is displayed on the *X*-axis with above average contribution variables to the "paranoid" side of Factor 1 (i.e. left side) shown in black and above average contribution variables to the "nonparanoid" side of Factor 1 (i.e. right side) shown in gray. Placement of the groups and hulls shows clear separation between the SCZP and CON groups, and placement of variables demonstrates that strong endorsements of Paranoia Scale items are associated with the SCZP and ASD groups whereas weak endorsements are associated with the SCZNP and CON groups. Factor 2 is displayed on the *Y*-axis. B) Factor 2 is displayed on the *X*-axis with above average contribution variables to the "cynical" side of Factor 2 (i.e. left side) shown in black and above average contribution variables to the "traditional paranoia" side of Factor 2 (i.e. right side) shown in gray. Placement of the groups and hulls demonstrates clear separation of the ASD and SCZP groups along this factor. Placement of the variables demonstrates that strong endorsements of items related to suspiciousness about the intentions and motivations of others are most highly associated with the ASD group. Factor 3 appears on the *Y*-axis. C) Factor 3 is shown on the *Y*-axis with above average contribution variables to the "moderate endorsement/insightful acknowledgment" side of Factor 3 (i.e. bottom) in black, and above expected contribution question-response levels to the "extreme endorsement" side of Factor 3 (i.e. top) in gray. Placement of the groups and hulls demonstrates separation between the psychotic and nonpsychotic groups, and placement of the variables reveals that moderate endorsements of Paranoia Scale items related to the negative aspects of mental illness are associated with the SCZNP group.

Table 3
Fixed Effects Confusion Matrix

	ASD	SCZNP	SCZP	CON
ASD	**11**	3	1	1
SCZNP	4	**18**	2	2
SCZP	0	0	**16**	0
CON	3	9	5	**26**
$N = 101$	$n = 18$	$n = 30$	$n = 24$	$n = 29$

Note. The columns are the original groups and the rows the predicted groups. The majority of individuals are classified within their own groups and are classified above chance, Fisher's χ^2 exact test (2,000 permutations; R Development Core Team, 2010), $\chi^2 = 122.44$, $p < .0004$.

exhibited elevated scores on the Paranoia Scale relative to SCZNP and CON, though the two groups (ASD and SCZP) did not differ from each other. This finding corroborates previous reports of heightened paranoia in ASD; however, a discriminant correspondence analysis examining patterns of responses revealed three factors that differentiated the groups and isolated features of paranoia unique to ASD.

Factor 1 best separated the groups on a continuum of low (SCZNP and CON), middle (ASD) and high (SCZP) paranoia, suggesting continuity between ASD and SCZP such that both groups demonstrate elevated paranoid ideation as compared to SCZNP and CON. Because this factor accounts for the most variance (~71%) and is comprised of response-levels from all but one of the questions on the Paranoia Scale, it best embodies the single factor of paranoia originally conceptualized by Fenigstein and Vanable (1992) and serves as a validation of the applicability of the Paranoia Scale across multiple disorders as well as the healthy population. Factor 2 was driven by a separation between the ASD and SCZP groups and reveals a qualitative difference in the type of paranoia endorsed in autism, specifically a type of "social cynicism" that appears exclusive to the ASD group. Factor 3 differentiated the two schizophrenia groups from the nonpsychotic groups and was defined by a moderate endorsement of items related to poor quality of life, and, as such, may reflect an insightful acknowledgment of the realistic challenges and experiences of living with a severe mental illness.

The separation of ASD and SCZP on Factor 2 suggests that their similar overall scores on the Paranoia Scale are, in part, obtained from qualitatively distinct patterns of responses. Whereas individuals with SCZP were most inclined to strongly endorse items related to perceived victimization and threat of harm, individuals with ASD were unique in showing a pattern of responses that emphasized cynical bias concerning the motivations and intentions of others. This suggests mechanistic differences in the formation of paranoid ideation between these two groups. One potential mechanism, proposed by Blackshaw and colleagues (2001), suggests that paranoia in SCZP may be related to an externalizing attributional bias that occurs as a means of protecting against discrepancies between perceptions of the actual-self and the ideal-self (e.g., failing that test was not my fault, the instructor sabotaged me), but that ASD paranoia may stem to a greater degree from a social impairment that results in a failure to effectively understand social cues and the rules of social interaction. Although speculative, another potential mechanism for this distinction in the types of paranoid thoughts endorsed by these groups may be related to reports of exaggerated theory of mind biases in schizophrenia that result in the overattribution of intentionality to others (Abu-Akel & Bailey, 2000; Frith, 2004; Montag et al., 2010; Walter et al., 2009), a process that may increase and distort one's sensitivity for perceiving threat. In contrast, ASD has been related to an underattribution of mental states (Baron-Cohen, 1995; Happe, 1994) that results in difficulty predicting people's responses and behaviors. Although both social–cognitive frameworks could produce elevated rates of paranoia, the underlying mechanisms supporting the paranoia may be very different. These differences have important implications for treatment, as intervention strategies that target disorder-specific mechanisms rather than superficial similarities will likely prove to be the most efficacious.

The current study highlights dissociable contributors to paranoia in ASD and SCZP; however, it also presents an overall factor structure that more broadly refines our clinical conceptualization of paranoia. Specifically, previous research has supported the notion of one generalized concept of paranoia (Fenigstein & Vanable, 1992) or has supported the presence of paranoid subtypes derived from correlations with other personal characteristics such as levels of depression, self-esteem and anxiety (Chadwick, Trower, Juusti-Butler & Maguire, 2005; Combs et al., 2007; Trower & Chadwick, 1995). Here, however, we provide evidence of three distinct subtypes of paranoia based only upon the content of the paranoid thoughts. The first subtype aligns with a traditional definition of paranoia characterized by unfounded suspicion, perceived victimization and a fear of being harmed. In contrast, the second subtype represents a cynical paranoia in which individuals are suspicious of the motives and intentions of others rather than a threat of danger, and a belief that people are primarily self-interested. The final subtype demonstrates recognition of the challenging social circumstances that accompany mental illness and the fact that bad things do sometimes happen. Collectively, these three factors provide a comprehensive depiction of the manifestation of paranoia across multiple disorders, and suggest that feelings of paranoia and paranoid thoughts—at least as measured by the Paranoia Scale—can be generated by qualitatively distinct characteristics.

Support for these three subtypes can be garnered from the personality literature. Specifically, a factor analysis of the MMPI-2 conducted by Tellegen and colleagues (2003) demonstrated that Clinical Scale 6 (Paranoia) could be best broken down into: (a) a

Table 4
Random Effects (Jackknife) Confusion Matrix

	ASD	SCZNP	SCZP	CON
ASD	**2**	6	4	3
SCZNP	4	**10**	3	4
SCZP	9	1	**12**	0
CON	3	13	5	**22**
$N = 101$	$n = 18$	$n = 30$	$n = 24$	$n = 29$

Note. The columns are the original groups and the rows the predicted groups. Nine ASD individuals are classified as SCZP in random effects where as they were classified as ASD in fixed effects. Overall classification is above chance, Fisher's χ^2 exact test (2,000 permutations; R Development Core Team, 2010), $\chi^2 = 44.33$, $p < .0004$.

Demoralization Factor, similar to our Factor 3, which reflects the overall level of emotional discomfort and discouragement associated with psychopathology; (b) a Cynicism Factor, similar to our Factor 2, which reflects a distrust of human nature and is consistent with beliefs that others are uncaring and look out only for themselves; and (c) an Ideas of Persecution Factor, similar to our Factor 1, which is uniquely associated with paranoid thinking and is consistent with thoughts that the individual is targeted and victimized by malevolent others. Interestingly, Tellegen and colleagues also noted that their Cynicism and Ideas of Persecution factors were separated by whether the items were self- or other-referential with self-referential items loading on the Persecution factor and other-referential items loading on the Cynicism factor. Such a pattern was not as pronounced in our results; however, the majority of the items on the PS (75%) are self-referential. This resulted in a strong self-referential component in our Paranoia Factor similar to that of Tellegen and colleagues but also yielded the inclusion of both other-referential and self-referential items on Factor 2. Nevertheless, this distinction may be meaningful in understanding differences between SCZP and ASD and should be pursued in future work.

The current study also provides new information about a commonly used measure of paranoia, the Paranoia Scale. The three factor model we present extends upon the factor analysis conducted on the instrument by Fenigstein and Vanable (1992) by examining patterns of response-levels across multiple disorders, a process facilitated through the use of a statistical technique (DiCA) that is specifically designed to highlight factors that differentiate between groups. This approach not only illuminated qualitatively distinct subtypes of paranoia, but also revealed that these subtypes may be differentially associated with autism and schizophrenia. Future comparative research that continues to explore how paranoia manifests across affected disorders, as well as the efficacy of the Paranoia Scale for capturing these patterns, may prove valuable.

The present findings suggest that aspects of paranoia differ between autism and schizophrenia; however, they should be interpreted within the context of several limitations. First, results were generated using a single measure of paranoia, the Paranoia Scale. This instrument was selected for the current study because it is the most widely used measure spanning both disorders. However, more recent measures of paranoia assess additional features such as degree of conviction in paranoid thoughts and level of distress associated with these thoughts (Freeman et al., 2005; Green et al., 2008). The current study was unable to examine whether these aspects of paranoia differ between autism and schizophrenia. Further, because the Paranoia Scale is a self-report measure, it relies upon subjective feelings of paranoia that may differ from information captured through behavioral or observational methods. Future studies comparing patterns of paranoia in autism and schizophrenia may, therefore, benefit from the inclusion of multiple methodologies that assess additional aspects of paranoia. Second, levels of positive and general symptom severity differed between the two schizophrenia groups. Here, our focus was on qualitative distinctions between SCZP and ASD, but understanding the contribution of other symptoms to paranoid ideation will be important for fully understanding the nature of paranoia in schizophrenia.

Despite these limitations, findings from this study demonstrate that the overall similarity in heightened paranoia found in autism and schizophrenia is supported by qualitatively distinct features. These distinctions have significant clinical implications, as they suggest nonshared origins and patterns of paranoid ideations in the two disorders that may inform the development of disorder-specific treatment practices. Further comparative investigations are warranted.

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