Prevalence of Psychotic-like Experiences in Young Adults With Social Anxiety Disorder and Correlation With Affective Dysregulation

Marco Armando, PhD,†‡ Ashleigh Lin, PhD,‡ Paolo Girardi, MD,§ Valentino Righetti, MD,† Claudia Dario, MD,† Riccardo Saba, MD,† Franco Decrescenzo, MD,* Luigi Mazzone, PhD,* Stefano Vicari, PhD,* Maximillian Birchwood, PhD,‡ and Paolo Fiori Nastro, MD†*

Abstract: Social anxiety disorder (SAD) is associated with psychotic-like experiences (PLEs) and is a frequent diagnosis in the prodromal phases of psychosis. We investigated whether psychopathological factors could discriminate which subjects with SAD are more likely to develop PLEs. A sample of 128 young adults with SAD was split into two subsamples according to the presence of clinically relevant PLEs. Correlations between PLEs and other psychopathological markers were explored. The SAD with PLEs group showed higher level of anxiety, depression, and intolerance of uncertainty (IU) compared with the SAD without PLEs group. A limitation of this study is that the cross-sectional design precluded the analysis of causality. In our sample, the presence of PLEs is related to higher levels of depression, anxiety, and IU. The current findings are consistent with hypotheses suggesting that cognitive disturbances, together with social anxiety, may result in PLEs.

Key Words: Social anxiety disorder, psychotic-like experiences, psychotic onset.

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Social anxiety disorder (SAD) is a highly prevalent comorbid disorder in patients with established schizophrenia and schizoaffective disorder, estimated to occur in more than a third of these patients (Huppert and Smith, 2005; Pallanti et al., 2004). Individuals with comorbid SAD are at greater risk for substance/alcohol abuse, suicide attempts, greater lethality of suicide attempts, and lower social adjustment and quality of life (Pallanti et al., 2004). The common co-occurrence of SAD and psychotic symptoms is present early in psychotic illness. It is estimated that between 28% and 33% of first-episode psychosis (FEP) populations will have comorbid SAD (Birchwood et al., 2007; Lang and Stein, 2001). Even before the onset of frank psychotic symptoms, SAD represents one of the most frequent diagnoses in the prodromal phases of psychotic illness (Cannon et al., 2008; Johnstone et al., 2005).

A number of different hypotheses have been proposed to explain the origin and the nature of the association between SAD and psychosis. Three among prominent theories were recently highlighted by Michail and Birchwood (2009): a) social anxiety predates the onset of psychotic symptoms (*i.e.,* persecutory beliefs) and serves to trigger and/or maintain psychotic symptoms, b) social anxiety and psychotic symptoms develop concurrently in the early phase of psychosis and follow a similar course, c) and social anxiety develops in some individuals as a consequence of psychotic symptoms.

Evidence that social anxiety predates psychotic symptoms comes from both at-risk and psychotic populations. Social anxiety was common in the Edinburgh Genetic High Risk sample and was one of the strongest predictors of transition to frank psychosis (Johnstone et al., 2005). Similarly, in patients with psychosis, high levels of social anxiety correlate with higher levels of psychotic symptoms, particularly paranoid beliefs (Huppert and Smith, 2005; Lysaker et al., 2010a).

Despite evidence of the influence of social anxiety in the development and the maintenance of psychotic symptoms, there is still a lack of knowledge of possible pathways from social anxiety to psychotic symptoms. To better understand this, different domains have been explored. For example, some studies highlight the role played by "affective dysregulation," which is defined as the interaction of anxiety, depression, and low self-esteem that may result in maladaptive appraisal patterns of events (Bentall and Fernyhough, 2008; Bentall et al., 2009; Thewissen et al., 2007; Yung et al., 2007). These appraisals trigger a search for an explanation of their meaning, which, in turn, increases the risk for positive psychotic experiences in vulnerable individuals (van Rossum et al., 2011). Some studies have shown that affective dysregulation can play an important role in the development and the maintenance of paranoid delusions and psychotic symptoms (Smeets et al., 2012; Wigman et al., 2011c).

A second domain that has been explored as a possible explanation for the association between social anxiety and psychosis is intolerance of uncertainty (IU). This construct is related to the idea that uncertainty is unacceptable and is conceptualized as a basic dysfunctional schema that may guide information processing and appraisal (Boelen and Reijntjes, 2009; Buhr and Dugas, 2006; Freeston et al., 1994). In this regard, phenomenological, cognitive, and neurobiological models suggest that the critical factor of "sensitization" (Collip et al., 2008) in the onset of psychosis is the faulty appraisal or interpretation of experiences or events (Blankeburg, 1971; Kapur, 2003). From a phenomenological and cognitivist perspective, sensitization may be related to a pathway that begins with weakness in self-identity, becomes an IU, and subsequently leads to a response style that reflects a data-gathering reasoning bias. The end result is the faulty appraisal or interpretation of experiences or events, that is, the core of delusions (Freeman et al., 2001). Previous studies have demonstrated a correlation between psychosis, faulty appraisal, and IU (Bentall et al., 2009; Corcoran et al., 2008; Menon et al., 2008). Nevertheless, although IU is often described as a phenomenon that is closely related to anxiety and worry (Boelen and Reijntjes, 2009; Buhr and Dugas, 2006), it has rarely been directly related to psychotic experiences. Broome et al. (2007) found that young people with an at-risk mental state for

*Department of Child and Adolescence Psychiatry, Research Hospital IRCCS Bambino Gesù, Rome, Italy; †Department of Psychiatry, PhD School "Early Intervention in Psychosis," "Sapienza" University, Rome, Italy; ‡School of Psychology, University of Birmingham, Birmingham, England; and §NESMOS Department, PhD School of "Early Intervention in Psychosis," Sant'Andrea Hospital and "Sapienza" University of Rome, Rome, Italy.

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Send reprint requests to Marco Armando, PhD, Department of Child and Adolescence Psychiatry, Research Hospital IRCCS Bambino Gesù, 4 S. Onofrio Square, 00100, Rome, Italy. E-mail: marco.armando@opbg.net.

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psychosis displayed greater difficulties tolerating uncertainty than did healthy controls. Another study carried out on people with schizophrenia found that IU was associated with psychosis-related distress. IU was also associated with negative metacognitive beliefs about hallucinatory voices and paranoia, regardless of the level of current psychotic symptoms (White and Gumley, 2010).

Investigating the psychotic experiences of patients with SAD provides an excellent opportunity to better understand the mechanisms involved in the pathways from social anxiety to psychosis. Indeed, although many studies have explored the presence of SAD in patients with psychosis, there has been little investigation of psychotic symptoms in individuals with SAD. Moreover, in many studies, the association has been explored in an indirect manner. For example, investigating the association between a history of trauma and persecutory ideation and verbal hallucinations, Freeman and Fowler (2009) found that the association between trauma and paranoia was explained by levels of anxiety. In another instance, Martin and Penn (2001) examined the linear relationship between persecutory ideation and multiple clinical and social cognitive variables in the general population. They found that higher levels of paranoid ideation were significantly associated with greater depressed mood and social anxiety.

To our knowledge, only one case study has specifically investigated the presence of psychotic symptoms in three patients with SAD (Veras et al., 2011). These authors present three possible explanations for psychotic manifestations in SAD. Firstly, psychotic experiences may arise from the individual's inability to challenge the impression of being criticized by people. A second possibility is that the stressful and perpetuating nature of SAD makes some individuals more likely to present with more severe psychiatric symptoms such as delusions. Finally, it may be that in some cases, SAD is caused by a primary thought abnormality (psychotic self-reference) rather than an affective disturbance (anxious insecurity), which leads to intense concern about others' opinions.

In summary, no research has specifically investigated psychotic symptoms and their correlation with other clinical and sociodemographic variables in patients with SAD. Moreover, no study has investigated the role played by affective dysfunction in the development of psychotic-like experiences (PLEs) in this group of patients. Individuals who experience intense and frequent PLEs are five times more likely to be diagnosed with a psychotic disorder 4 years later (Hanssen et al., 2003b), particularly if the PLEs are of a persecutory nature (Armando et al., 2010). This highlights the importance of identifying which young people with SAD are most vulnerable to psychotic experiences.

The aims of this study were to investigate a large sample of young adults with SAD for a) the prevalence of PLEs compared with a healthy control group and b) the clinical (IU, anxiety, depression, general functioning, IQ, and substance abuse) and sociodemographic (sex, age, cultural background, and level of education) characteristics of SAD patients with and without PLEs. We hypothesized that the prevalence of clinically relevant PLEs will be significantly higher in those with SAD than in the healthy control group. In addition, the subsample with clinically relevant PLEs will have a different clinical picture from that of SAD patients without PLEs, characterized by higher levels of depression, anxiety, and IU, indicative of greater affective dysregulation.

METHODS

Sample

The participants in this study were 128 help-seeking young adults with SAD and 41 healthy controls, aged 19 to 25 years. The patients were recruited from the outpatient psychiatric unit of the Sapienza University Hospital in Rome between January 2008 and December 2010. The inclusion criteria were an axis I diagnosis of SAD (*Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision* [*DSM-IV-TR*]; assessed with the Structured Clinical Interview for DSM-IV Axis I Disorders [SCID-I]) and no axis II diagnosis (assessed with the SCID-II). The exclusion criteria were a previous or current psychotic disorder (affective and/or nonaffective), IQ lower than 85, and symptoms due to any organic etiological factor or related to drug use. The healthy control participants were recruited through local advertisements in the same geographic area as that of the clinical group. The exclusion criteria for the control group were the presence of any psychiatric disorder assessed with the SCID-I and the SCID-II and IQ lower than 85. The control sample was comparable with the SAD group for sex, age, and IQ (see Table 1). All the participants provided written informed consent.

TABLE 1. Sociodemographic Characteristics of the Samples

Variables	SAD + PLEs (n = 32)	SAD − PLEs (n = 96)	Control Group (n = 41)	Statistics
Age, mean ± SD, yrs	21.1 ± 4.7	20.7 ± 1.7	21.8 ± 2.9	$F(2) = 2.002$, $p = 0.138$
Sex, n (%)				
Male	12 (7.5)	20 (20.8)	12 (29.2)	$\chi^2_2 = 4.77$, $p = 0.07$
Female	20 (62.5)	76 (79.2)	29 (70.8)	
IQ, mean ± SD	92 ± 8.3	94 ± 7.1	96 ± 10.3	$F(2) = 3.163$, $p = 0.231$
Education level, n (%)				
Primary school	6 (18.7)	14 (14.6)	10 (24.4)	$\chi^2_3 = 3.52$, $p = 0.286$
Secondary school	19 (59.3)	57 (59.0)	22 (53.6)	
University or higher	7 (21.8)	25 (26.4)	9 (22.0)	
Cannabis, n (%)				
≤1 per mo	30 (91.0)	88 (92.1)	39 (95.1)	$\chi^2_2 = 2.96$, $p = 0.937$
>1 per mo	3 (9.0)	8 (7.9)	2 (4.9)	
Alcohol, n (%)				
≤1 per mo	28 (89.6)	85 (89.6)	38 (92.7)	$\chi^2_2 = 4.44$, $p = 0.816$
>1 per mo	5 (3.5)	11 (11.4)	3 (7.3)	
Other drugs, n (%)				
≤1 per mo	31 (96.0)	94 (97.7)	41 (100.0)	$\chi^2_2 = 12.76$, $p = 0.237$
>1 per mo	2 (4.0)	2 (2.3)	0 (0.0)	

Instruments

Sociodemographic and Clinical History

Basic information on the participants' sociodemographic background and clinical history (*i.e.*, familiar cultural background, level of education, previous psychiatric diagnoses, previous contact with psychiatric services, substance abuse) was collected.

PLEs, Negative Symptoms, and Related Distress

PLEs and negative symptoms were assessed using the Community Assessment of Psychic Experiences (CAPE; Stefanis et al., 2002). This self-report scale measures the lifetime prevalence of positive (PLEs), negative, and depressive symptoms on scales of both frequency (0 = never to 4 = nearly always) and distress (1 = not distressed to 4 = very distressed). Because it has been suggested that self-reporting instruments may overestimate the prevalence of PLEs (Kendler et al., 1996), a psychiatric clinician conducted clinical reinterviews to validate the self-reported PLEs. A similar methodology was previously used by Hanssen et al. (2005, 2003a).

Intolerance of Uncertainty

IU was evaluated using the Intolerance to Uncertainty Scale (IUS; Freeston et al., 1994). This is a 27-item questionnaire rated on a 5-point Likert scale. The IUS has a good internal consistency (Cronbach's $\alpha = 0.94$), item-total correlations ranging from 0.36 to 0.77, and a retest reliability of $r = 0.74$ (Buhr and Dugas, 2002, 2006; Freeston et al., 1994). The scale has previously been used in studies investigating delusion formation (Broome et al., 2007), anxiety, and depression (Boelen and Reijntjes, 2009; Buhr and Dugas, 2006).

Other Measures

Level of anxiety and depressive symptoms were evaluated using the Beck Anxiety Inventory (BAI; Beck and Steer, 1990) and the Beck Depression Inventory–II (BDI-II; Beck et al., 1996). Both are 21-item self-report instruments that assess anxiety and depressive symptoms, respectively. The SCID-I for *DSM-IV* (First et al., 1997) was used to ascertain psychiatric diagnoses. Raters were experienced clinicians, trained in using the SCID-I. Level of general functioning was measured using the General Health Questionnaire–12 (GHQ-12; Goldberg et al., 1997). The Wechsler Adult Intelligence Scale–Third Edition (Wechsler, 2001) was used to assess IQ.

Procedure

PLEs with low frequency and related low level of distress should not be considered as clinically meaningful and are not related to increased risk for further psychotic symptoms (Armando et al., 2010; Hanssen et al., 2005; Schultze-Lutter et al., 2011; van Os et al., 2009; Yung et al., 2006). To reduce the risk for misclassification, a psychiatric clinician conducted clinical interviews to validate each self-reported PLE for frequency and distress. On the basis of these considerations and in line with previous studies (Hanssen et al., 2003a; Nishida et al., 2008; van Os et al., 2001), we defined as "clinically significant PLE" any PLE that was scored both severe and distressing (*i.e.*, scored 3–4 in the severity and related distress subscales) and was confirmed by clinical interview. Consequently, two clinical subsamples were defined. The first was a subsample with SAD but without clinically significant PLEs (SAD − PLEs; *i.e.*, they scored 1–2 [never/sometimes] on any item of the CAPE positive subscale and the CAPE distress subscale related to positive symptoms). The second was a subsample with SAD and clinically significant

TABLE 2. Multiple Comparisons Between SAD + PLEs, SAD − PLEs, and the Healthy Controls

Variables	SAD + PLEs ($n = 32$)	SAD − PLEs ($n = 96$)	HC ($n = 41$)	Statistic	(I) Group	(J) Group	Mean diff	SE	p	95% CI
CAPE positive	40 ± 4.5	30 ± 3.4	23 ± 1.4	$F(2) = 226$, $p < 0.0001$	HC	SAD − PLEs	−6.7	0.62	$p < 0.0001$	−8 to −5
						SAD + PLEs	−16.7	0.78	$p < 0.0001$	−18 to −14
					SAD + PLEs	SAD − PLEs	9.9	0.67	$p < 0.0001$	8 to 11
CAPE negative	30.4 ± 5.3	26.5 ± 5.9	19.1 ± 3.7	$F(2) = 43.38$, $p < 0.0001$	HC	SAD − PLEs	−7.4	1.01	$p < 0.0001$	−9 to −5
						SAD + PLEs	−11.3	1.29	$p < 0.0001$	−14 to −8
					SAD + PLEs	SAD − PLEs	3.9	1.13	$p < 0.05$	1 to 6
BAI	25.9 ± 8.6	23.1 ± 8.1	3.6 ± 2.4	$F(2) = 125.9$, $p < 0.0001$	HC	SAD − PLEs	−19.7	1.35	$p < 0.0001$	−23 to −16
						SAD + PLEs	−22.5	1.69	$p < 0.0001$	−26 to −18
					SAD + PLEs	SAD − PLEs	6.5	1.43	$p < 0.0001$	−7 to 6
BDI-II	25.2 ± 12.4	17.2 ± 8.7	3.0 ± 2.5	$F(2) = 61.5$, $p < 0.0001$	HC	SAD − PLEs	−14.2	1.67	$p < 0.0001$	−18 to −10
						SAD + PLEs	−22.2	2.07	$p < 0.0001$	−27 to −17
					SAD + PLEs	SAD − PLEs	8.1	1.76	$p < 0.0001$	3 to 12
IUS	81.7 ± 16.5	71.4 ± 13.1	49.2 ± 12.8	$F(2) = 26.23$, $p < 0.0001$	HC	SAD − PLEs	−22.2	3.60	$p < 0.0001$	−31 to −13
						SAD + PLEs	−32.5	4.58	$p < 0.0001$	−43 to −21
					SAD + PLEs	SAD − PLEs	10.3	391	$p < 0.05$	1 to 19
GHQ-12	19.1 ± 6.4	16.3 ± 6.1	8.1 ± 4.1	$F(2) = 37.91$, $p < 0.0001$	HC	SAD − PLEs	−8.2	1.12	$p < 0.0001$	−11 to −5
						SAD + PLEs	−10.9	1.35	$p < 0.0001$	−14 to 8
					SAD + PLEs	SAD − PLEs	2.6	1.27	$p = 0.079$	1 to 5

The Hochberg's GT2 was used for post hoc comparisons.
CI indicates confidence interval; Diff, difference; HC, healthy controls.

PLEs (SAD + PLEs) scored 3 to 4 (nearly always/always) on at least one item of both the positive and the distress subscale.

Data Analysis

Analyses were conducted using the IBM Statistical Package for the Social Sciences statistics 18. As a first step, data were screened for missing values, normality, linearity, homogeneity, and outliers. Eleven participants were excluded because of missing data.

The three groups (SAD − PLEs, SAD + PLEs, and healthy control group) were compared on continuous demographic and clinical variables using analysis of variance. Comparisons on categorical variables were made using the chi-square analysis. To exclude type I and type II errors because of unequal sample sizes, homogeneity of variance was confirmed using the Levene's test. A subsequent analysis of covariance was conducted, covarying for demographic variables. The Hochberg's GT2 was used for post hoc comparisons. Correlations were conducted within the SAD + PLEs subsample to assess the relationship between the level of PLEs and the other clinical variables. All tests were two tailed, and p values less than 0.05 were considered statistically significant.

RESULTS

Prevalence of Clinically Significant PLEs in the Patients With SAD and the Healthy Controls

As predicted, the prevalence of clinically significant PLEs in the SAD group was significantly higher than in the healthy control group ($p < 0.001$). Of the participants with SAD, 32 reported clinically meaningful PLEs (SAD + PLEs; 25%) and 96 did not (SAD − PLEs; 75%). Only two participants (4.8%) in the control group showed clinically relevant PLEs. All further analyses were conducted on the three defined subsamples (SAD + PLEs, SAD − PLEs, and healthy controls).

Sociodemographic Characteristics

Age, sex, and other sociodemographic variables were homogeneous within the three groups. There were no significant differences between the three groups in terms of age, sex, education level, IQ, and drug/alcohol abuse (see Table 1).

Comparison Between SAD + PLEs, SAD − PLEs, and the Healthy Controls

We compared the three groups on clinical and functional variables. As expected, the mean scores on the CAPE intensity and distress scales were significantly different in the three subsamples ($F[2] = 226$; $p < 0.0001$), with a progressive increase from the control group (23 ± 1.4) to SAD − PLEs (30 ± 3.4) and SAD + PLEs (40 ± 4.5). Significant group differences ($p < 0.0001$) were found for all clinical and functional scores (IUS, BAI, BDI-II, CAPE-negative, and GHQ-12; see Table 2).

Post hoc analyses revealed that both clinical groups scored significantly higher than did the control group on all the clinical variables ($p < 0.0001$). Comparisons between the two clinical groups showed that the SAD + PLEs group had significantly higher scores ($p < 0.0001$) for level of IU and depressive, anxiety, and negative symptoms than those of the SAD − PLEs group (see Table 2).

Correlation Between PLEs and Other Clinical Variables in the SAD + PLEs Group

Correlations between PLEs and other clinical variables were conducted within the SAD + PLEs sample to explore which of them was most closely related to variation in the intensity of PLEs. Correlations were significant and positive for the IUS ($r = 0.520$; $p < 0.001$) and anxiety symptoms ($r = 0.575$; $p < 0.001$). Depressive and negative symptoms were not found to be correlated with the increase of PLEs (Table 3).

DISCUSSION

The first aim of this study was to investigate the prevalence of PLEs in the patients with SAD compared with the healthy controls. Twenty-four percent of the patients with SAD, compared with 5% of the healthy control group, reported clinically relevant PLEs on the CAPE. These were confirmed with clinical interviews. The prevalence of PLEs in our sample cannot be compared with others because no previous studies have investigated PLEs in SAD patients. However, the rate of PLEs in the healthy control group is significantly higher than what has been reported in previous general population studies. In a recent meta-analysis, van Os et al. (2009) estimated the median prevalence of clinically significant PLEs in the general population at 1.5% (interquartile range, 0.4%–3.0%). In the two largest cohort studies in which the distinction between clinical and subclinical psychotic phenomena was made, rates of clinically relevant symptoms were 4.2% and 3.8%, respectively (Dominguez et al., 2011; van Os et al., 2001), a prevalence similar to that of our healthy control group. It is important to acknowledge that within this literature, different measures are used to assess psychotic experiences, and clinical relevance is defined in different ways, making comparisons difficult. However, regardless of this, the prevalence of clinically significant PLEs in the SAD group was almost five times that of the healthy control group, suggesting that these are indeed very common in this clinical population.

The second aim of this study was to investigate clinical and sociodemographic differences between the SAD patients with and without clinically significant PLEs. The main finding was that the SAD patients who had clinically significant PLEs show a distinct clinical profile, characterized by higher levels of depression, IU, and negative symptoms. Moreover, within the group of patients with SAD and PLEs, there was a strong association between higher levels of PLEs and greater anxiety and IU levels. It is important to note that the level of PLEs was not related to sociodemographic variables, including cannabis or any other substance abuse.

The subsample with SAD + PLEs had higher levels of depression than those of the SAD − PLEs group. This is consistent with a number of previous studies showing a strong association between PLEs and depression in adolescents (Nishida et al., 2008; Scott et al., 2009; Wigman et al., 2011a; Yung et al., 2007) and young adults (Wigman et al., 2011b). This suggests that patients with SAD + PLEs more frequently display a combination of pessimistic thinking and low self-esteem (Bentall and Fernyhough, 2008; Bentall et al., 2009). Therefore, it seems that self-esteem and, consequently, affective dysregulation play an important role in paranoid delusions and psychotic symptoms (Smeets et al., 2012; Thewissen et al., 2007; Wigman et al., 2011a).

TABLE 3. Correlation Between PLEs and Other Clinical Variables in the SAD + PLEs Subsample

	CAPE Positive	CAPE Negative	IUS	BAI	BDI-II
CAPE positive	1				
CAPE negative	0.119	1			
IUS	0.520**	0.507**	1		
BAI	0.575**	0.215*	0.593**	1	
BDI-II	0.130	0.560**	0.619**	0.230*	1

*$p < 0.05$.
**$p < 0.01$.

Other clinical variables were found to be associated with the presence of significant psychotic experiences in SAD. In our sample, IU was significantly higher in SAD + PLEs (compared with SAD − PLEs and the healthy controls), and, within this group, there was a significant, positive correlation between the level of PLEs and IU. This finding is consistent with previous research showing that IU and related worry are associated with psychotic experiences and that social worry predicted emotional responses over and above the intensity of psychotic experiences (Morrison and Wells, 2007; White and Gumley, 2010). It has been argued that that this feature arises from a condition of IU caused by a loss of self-identity (Freeman et al., 2008; White and Gumley, 2010), which is a typical feature of the at-risk mental state (Johnstone et al., 2005). If not resolved, this condition can lead to the development of initial positive symptoms (Colbert et al., 2006), insofar as persecutory delusions (because of its intrinsic feature of absolute certainty) rule out the risk for *Ratlosigkeit* (*i.e.*, schizophrenic or schizotypal perplexity) and *wahnstimmung* (*i.e.*, delusional atmosphere or mood; Blankeburg, 1971; Conrad, 1958; Mishara, 2010). Several studies have shown an association between IU and psychotic symptoms both in nonclinical (Freeman et al., 2008) and clinical populations. Concerning the latter, Broome et al. (2007) found that young people at risk for psychosis displayed greater difficulties tolerating uncertainty than did healthy controls, whereas White and Gumley (2010) found a similar correlation in patients with psychosis, although one recent study found no association between IU and psychotic ideation in patients with psychosis (Dudley et al., 2011).

As we expected, the SAD + PLEs group reported a higher level of anxiety. Indeed, this result is in accordance with previous findings in SAD patients with FEP compared with SAD patients without FEP (Michail and Birchwood, 2009). It would be interesting to explore whether anxiety related to PLEs has different features in terms of quality and of severity. Nevertheless, the BAI is not able to distinguish between severity and quality. Further studies should investigate these differences.

The positive association between PLEs and anxiety that was evident in the current subsample with SAD + PLEs is consistent with previous findings of an association between anxiety and psychotic symptoms in clinical (Lysaker et al., 2010a, 2010b) and nonclinical (Nishida et al., 2008) samples. Lysaker et al. (2010a, 2010b) recently demonstrated that high levels of paranoid features correlated with greater social anxiety symptoms in patients with schizophrenia. Nishida et al. (2008) reported a similar association between anxiety levels and PLEs in the general population. These findings suggest that anxiety can contribute to the development and the maintenance of psychotic experiences (Freeman and Fowler, 2009; Morrison and Wells, 2007).

Interestingly, we did not find evidence of not a strong relationship between the level of PLEs and cannabis use in either of the two clinical groups. This finding contrasts previous studies that demonstrated this association (Fergusson et al., 2003; Henquet et al., 2005; Mackie et al., 2010; Wigman et al., 2011c). Mackie et al. (2010) showed that among adolescents with a trajectory of increasing PLEs during 2 years, a rise in cannabis use preceded a sharp increase in symptoms. Similarly, Henquet et al. (2005) found that the risk for PLEs among adolescents and young adults increased in a dose-response manner relative to the frequency of cannabis use. We can hypothesize that the lack of correlation in the present study can, at least partially, be related to the low prevalence of cannabis use in our sample compared with other samples with SAD (Agosti et al., 2002; Armando et al., 2012). In addition, it has recently been argued that the relationship between psychotic symptoms and cannabis depends on the specific type of cannabis and the level of cannabidiol (Schubart et al., 2011), neither of which were investigated.

The current study is, to our knowledge, the first to investigate the prevalence of PLEs in SAD and the first to examine the association between PLEs and other clinical variables in a sample of patients with SAD compared with healthy controls. By using clinical interview and self-report, we feel confident that the data attained on the presence of PLEs are accurate. A limitation of this study is that the cross-sectional design precluded the analysis of causality and the prediction of outcome. Thus, we are able to speculate only the direction of causality and the magnitude of risk. For the same reason, it is unclear why some individuals with SAD develop PLEs whereas others do not and whether PLEs are transitory or continue to exist as a part of SAD for some individuals. Future research should investigate developmental and environmental factors that may contribute to the experience of PLEs for a subgroup with SAD. Longitudinal investigations are also needed to explore the PLEs over time.

CONCLUSIONS

In summary, our findings suggest that a) clinically significant PLEs can be found in approximately one quarter of young adults with SAD; b) PLEs in patients with SAD are related to other psychopathological markers, especially IU and depressive symptoms; and c) the increase in the frequency and distress of PLEs is related to an increase in anxiety and IU levels. These findings are consistent with current hypotheses on the psychological mechanisms that underlie psychotic symptoms (Bentall et al., 2009; Morrison and Wells, 2007), particularly those that emphasize that cognitive disturbances such as IU, together with social anxiety, may lead to anomalous experiences. These experiences, in combination with affective dysregulation (high level of depression and state-related anxiety) and faulty appraisal processes, may lead to the formation of clinically significant PLEs (Krabbendam and van Os, 2005; Smeets et al., 2012; Wigman et al., 2011a). If the current findings are confirmed by longitudinal studies, there are clear clinical applications for early intervention. For example, cognitive behavioral therapies are likely to be useful in targeting faulty cognitive appraisals with the aim of avoiding the development of PLEs, but this needs to be investigated further in patients with SAD (Halperin et al., 2000; Hewitt et al., 2009). In the current study, the SAD patients with PLEs did not meet the diagnostic criteria for psychotic illness but still experienced clinically relevant and distressing psychotic experiences. As such, screening for PLEs in young adults with SAD is necessary. The specific cognitive/psychopathological mechanisms involved in this process should be considered to provide the most appropriate therapy for those patients.

DISCLOSURES

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