An examination of neuroticism as a moderating factor in the association of positive and negative schizotypy with psychopathology in a nonclinical sample

Neus Barrantes-Vidal\textsuperscript{a,b}, Agnès Ros-Morente\textsuperscript{a}, Thomas R. Kwapi\textsuperscript{c,}\textsuperscript{*}

\textsuperscript{a} Departament de Psicologia Clínica i de la Salut, Facultat de Psicologia, Universitat Autònoma de Barcelona, 08193-Bellaterra, Barcelona, Spain

\textsuperscript{b} CIBERSAM, Spanish Ministry of Health, Instituto de Salud Carlos III-RETIC RD06/0011/0007, Red de Enfermedades Mentales (REM-TAP Network), Spain

\textsuperscript{c} Department of Psychology, University of North Carolina at Greensboro, P.O. Box 26170, Greensboro, NC 27402-6170, USA

**A B S T R A C T**

Personality traits such as neuroticism are associated with schizophrenia and schizotypy. However, studies thus far have not clarified the differential association of neuroticism with individual schizotypy dimensions and the role it plays in the expression of schizophrenia-spectrum psychopathology. 204 nonclinically ascertained participants completed self-report questionnaires assessing neuroticism and the positive and negative schizotypy dimensions, and underwent structured interviews assessing schizophrenia-spectrum psychopathology (psychotic-like experiences, negative symptoms, cluster A personality disorders and traits), mood episodes, substance abuse, and global functioning. Results indicated that neuroticism predicted positive symptoms of schizophrenia and depression, over-and-above the effects of both schizotypy dimensions. Also, neuroticism moderated the association of positive schizotypy with interview measures of psychopathology and functioning. The results of this study are consistent with other research indicating that neuroticism is etiologically relevant for schizophrenia-spectrum psychopathology and that it cannot be considered solely a `secondary effect’ of spectrum disorders. Current psychological models of psychosis can accommodate the finding of neuroticism being a shared vulnerability factor for affective and psychotic disorders.

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Negative schizotypy
Schizophrenia
Neuroticism
Moderator

1. Introduction

The dimensional view of schizophrenia posits that schizophrenia-spectrum disorders are extreme expressions of the personality disposition referred to as schizotypy, which results from the interaction of multiple genetic, neurodevelopmental, and psychosocial factors (e.g., Claridge, 1997; Meehl, 1999; van Os et al., 2009). It is assumed that there are individuals high on schizotypy who have an underlying vulnerability for schizophrenia, but who may never transition into clinical psychosis. In fact, it is presumed that the majority of individuals with schizotypy will never decompensate, although they may demonstrate mild and/or transient signs of schizophrenic-like or schizotypic adjustment including neurocognitive and biobehavioral deficits, clinical and subclinical symptoms, and social impairment. This formulation suggests that schizotypy is expressed across a dynamic continuum ranging from relative psychological health to full-blown schizophrenia. Since nonpsychotic schizotypes are hypothesized to share common neurodevelopmental pathways with schizophrenia patients, it is expected that they will exhibit mild and transient forms of the cognitive, emotional, and behavioral features of schizophrenia. Thus, the identification of schizotypic individuals should facilitate the identification of relevant etiological factors and may ultimately hasten the development of prophylactic treatment interventions. The study of schizotypy also minimizes many of the confounding effects of schizophrenia (e.g., hospitalization, medications, marginalized social status) that plague the study of patients (Lenzenweger, 2006).
Schizotypy is characterized as a multidimensional construct (Claridge et al., 1996; Mason et al., 1997; Raine et al., 1994; Stefanis et al., 2002; Vollema and van den Bosch, 1995). Positive and negative symptom schizotypy are the most consistently replicated factors (Kwapil et al., 2008). Positive schizotypy and positive symptom schizophrenia are characterized by odd beliefs and unusual perceptual experiences, which in their extreme form manifest as delusions and hallucinations. Negative schizotypy and schizophrenia are characterized by deficits such as affective flattening, anhedonia, social disinterest, and diminution of cognitive functioning. While there is not a universally agreed upon latent structure of schizotypy, the proposed factors appear consistent with those hypothesized to comprise schizophrenia (Bilder et al., 1985; Liddle, 1987; Peralta et al., 1992), supporting the hypothesis that the vulnerability to schizophrenia is expressed across the continuum of schizotypy.

Affective and anxiety symptoms are often present in schizophrenia, from prodromal to acute and chronic stages (Häfner et al., 2005). Rates of co-morbid depression and anxiety are elevated in schizophrenia and schizotypal personality disorder (American Psychiatric Association, 2000). Although, depression shares several phenotypic similarities with negative schizophrenia and schizotypy (e.g., anhedonia), depression and anxiety are more strongly associated with the positive than the negative dimension in both patient and nonpatient samples (e.g., Emsley et al., 1999; Lysaker et al., 1995; Lewandowski et al., 2006; Lenzenweger and Loranger, 1989).

Consistent with the association between mood and schizophrenia-spectrum disorders, personality traits such as neuroticism and negative affect are elevated in schizophrenia (Horan et al., 2008). Neuroticism is elevated in a wide variety of psychological disorders, but especially in syndromes that involve chronic, pervasive distress, such as major depression and generalized anxiety (Watson, 2000). Furthermore, neuroticism is a well-established risk factor for the onset of anxiety and affective disorders (Boyle et al., 1991).

Schizophrenia is characterized by high neuroticism and elevated "peculiarities" (Berenbaum and Fujita, 1994). Deviance in these emotionally-related traits may contribute to the pathoplastia of clinical symptoms, course, and associated features. Horan et al. (2008) concluded that high neuroticism occurs across all stages of schizophrenia. However, studies with schizophrenia patients cannot disentangle whether the deviance in these affective traits indexes true vulnerability for psychosis or merely represents the consequence of developing schizophrenia. The limited studies examining relations between schizotypy and neuroticism report that positive schizotypy is associated with neuroticism, but the findings were mixed regarding the relation of negative schizotypy and neuroticism (Gooding et al., 2002; Gooding, 2003; Horan et al., 2007; Kerns, 2005, 2006; Kwapil et al., 2008; Ross et al., 2002). For example, Kwapil et al. (2008) found that neuroticism was significantly associated with positive, but not negative, schizotypy in a large nonpatient sample.

Neuroticism may act as a moderating factor, that is, it may potentiate clinical outcomes among schizotypic individuals (Claridge and Davis, 2003), which may explain why only a minority of schizotypes will ever present with spectrum disorders. Thus, neuroticism might be one of several personality characteristics that influence the transition across the psychosis continuum. Meehl (1990) suggested that "polygenic potentiators" such as anxiety proneness (i.e., neuroticism), increase the likelihood of schizotypes developing clinical disorders. Based on Meehl's theorizing, Horan et al. (2007) reported that participants with higher levels of negative affectivity also had elevated levels of cluster A personality disorder symptoms. However, studies have not examined the moderating effect of neuroticism on schizotypy for predicting clinical symptoms.

The aims of the current study are to provide a preliminary examination of (i) the contribution of neuroticism over-and-above the effects of positive and negative schizotypy to explain schizophrenia-spectrum psychopathology, mood episodes, substance abuse, and general functioning; and (ii) the extent to which neuroticism moderates the effect of the schizotypy dimensions. In the present study, it is hypothesized that, (i) neuroticism will be significantly associated with interview measures of psychotic-like, schizotypal, and paranoid personality symptoms, and mood pathology, but not negative or schizoid symptoms; (ii) neuroticism will interact with positive schizotypy to moderate its association with psychopathological outcomes; and (iii) negative schizotypy will predict interview ratings of negative and schizoid symptoms, but will not be moderated by neuroticism.

2. Method

2.1. Participants

Participants included 204 undergraduates (46 men, 158 women) enrolled in introductory psychology courses at UNCG. The mean age of the sample was 20.2 (SD = 4.7). The sample was 71% Caucasian, 27% African-American, and 2% other. The participants were drawn from a pool of approximately 3000 participants who completed schizotypy questionnaires and a measure of extraversion as part of mass screening sessions during the course of three years. The sample in the present study subsequently took part in studies involving a structured interview during this time period. Note that in some cases participants were preselected based upon their scores on the schizotypy scales (either high or low scores), in other cases participants were not preselected (i.e., they were allowed to sign up for the study simply on the basis of having completed the measures in mass screening).

2.2. Materials and procedures

The schizotypy questionnaires included the Perceptual Aberration (Chapman et al., 1978), Magical Ideation (Eckblad and Chapman, 1983), Physical Anhedonia (Chapman et al., 1976) and Revised Social Anhedonia Scales (Eckblad et al., 1982), and a 13-item infrequency scale (Chapman and Chapman, 1983). Exploratory and confirmatory factor analyses of the four scales reliably produce two factors, positive and negative schizotypy, that account for 80% of the variance (Brown et al., 2007; Kwapil et al., 2008; Lewandowski et al., 2006). Participants were assigned positive and negative schizotypy dimensional scores, based upon factor loadings derived from a sample of 6137 college students (Kwapil et al., 2008). Participants completed the NEO-PI-R (Costa and
McCrae, 1992) in order to assess neuroticism. The NEO-PI-R consists of 48 items that are scored on a 5-point Likert scale from “Strongly Agree” to “Strongly Disagree”.

The interview contained portions of the Structured Clinical Interview for DSM-IV (First et al., 1995) that assesses mood episodes, substance use disorders, and demographic information. Quantitative ratings of impairment associated with alcohol and drug use were coded using the rating system described in Kwapil (1996). The modules of the International Personality Disorders Examination (IPDE; World Health Organization, 1995) that assess schizoid, paranoid, and schizotypal personality disorders were also included. These personality disorders were assessed because they are reported to be genetically related to schizophrenia (e.g., Kety et al., 1968). The IPDE provides personality disorders diagnoses and dimensional ratings of the disorders.

The Wisconsin Manual for Assessing Psychotic-like Experiences (Chapman and Chapman, 1980; Kwapil et al., 1999) was used to quantify the deviance of psychotic symptoms across clinical and subclinical deviancy. The manual provides criteria for rating seven classes of experiences from relatively normal to grossly psychotic. Kwapil et al. (1999) reported that the highest rating across the seven classes of experiences provides a useful index of clinical and subclinical deviancy, and predicts the development of psychotic disorders. The Negative Symptom Manual (Kwapil and Dickerson, 2001) was used to quantify negative symptoms of schizophrenia across clinical and subclinical deviancy. The manual includes a structured interview and rating system that assess six classes of symptoms. The Global Assessment Scale (GAS; Endicott et al., 1976) was used to assess overall functioning from marked psychopathology and impairment at the low end to superior functioning at the high end.

Although the screening questionnaires and interview measures share some overlap in content, they are not redundant with each other. The questionnaires are screening measures that tap schizophrenic experiences of a limited range and severity. The structured interviews provide detailed assessments based upon diagnostic criteria for mood episodes, and substance use and personality disorders, and based upon specific criteria for dimensional ratings of psychotic-like and negative symptoms across a broad range of severity.

The interviews were conducted by a licensed clinical psychologist and advanced graduate students in clinical psychology. The interviewers were unaware of participants’ scores on the schizotypy questionnaires.

### 3. Results

Table 1 provides descriptive data for the measures used in the study. Despite the use of a nonclinical sample, considerable variability was found on each of the measures. For example, scores on the positive and negative schizotypy dimensional scores ranged from approximately 1.5 SD below the mean to more than 4 SD above the mean.

Linear regressions were computed to examine the relations of positive schizotypy, negative schizotypy, and neuroticism with continuous criteria (e.g., ratings of psychopathology and functioning), and binary logistic regressions were computed for dichotomous diagnostic criteria. In each case, a regression analysis was computed with positive and negative schizotypy entered at the first step (indicating the unique contribution of each with the other partialed out); neuroticism was entered at the second step (revealing its effect over-and-above the schizotypy dimensions); and at the third step the positive schizotypy × neuroticism and negative schizotypy × neuroticism interaction terms were entered (examining their contribution over-and-above the main effects). In the present sample, neuroticism correlated 0.38 and 0.26 with positive and negative schizotypy, respectively.

Note that the positive schizotypy × negative schizotypy interaction and the 3-way interaction were not included in the analyses given that the focus of the investigation was on evaluating the main and moderating effects of neuroticism. Note, however, that these additional interactions did not account for significant variance of any criterion variable, as observed in several previous studies by our laboratories (e.g., Kwapil et al., 2008).

As seen in Table 2, positive, but not negative, schizotypy and neuroticism predicted psychotic-like experiences. The positive schizotypy × neuroticism interaction, but not the interaction with negative schizotypy, accounted for a significant increment in variance over the main effects. Simple slope analyses of the interaction term revealed that positive schizotypy significantly predicted psychotic-like experiences at all levels of neuroticism, but that this relation significantly strengthened as ratings of neuroticism increased. This was the case for low (β = 0.23; p = 0.02), moderate (β = 0.39; p < 0.001), and high (β = 0.55; p < 0.001) levels of neuroticism (low reflects −1 SD, medium is mean, and high is +1 SD). The moderating role of neuroticism in the association of positive schizotypy and psychotic-like experiences is shown in Fig. 1.

As hypothesized, the negative schizotypy dimensional score predicted interview ratings of negative symptoms. However, neither positive schizotypy, neuroticism, nor the interactions accounted for significant increments in variance in this measure.

### Table 1

Descriptive data for predictor and criterion variables.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Scale</th>
<th>Mean</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Predictors</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Positive schizotypy</td>
<td>z-scores</td>
<td>0.27</td>
<td>1.23</td>
<td>−1.40 to 4.20</td>
</tr>
<tr>
<td>Negative schizotypy</td>
<td>z-scores</td>
<td>0.70</td>
<td>1.36</td>
<td>−1.65 to 5.74</td>
</tr>
<tr>
<td>Neuroticism</td>
<td>T-scores</td>
<td>48.5</td>
<td>11.33</td>
<td>15.7–83.1</td>
</tr>
<tr>
<td><strong>Criteria</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychotic symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychotic-like experiences</td>
<td>0–11</td>
<td>1.27</td>
<td>2.06</td>
<td>0–9</td>
</tr>
<tr>
<td>Negative symptoms</td>
<td>0–48</td>
<td>3.95</td>
<td>4.78</td>
<td>0–22</td>
</tr>
<tr>
<td>IPDE dimensional scores</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schizotypal</td>
<td>0–18</td>
<td>1.08</td>
<td>1.62</td>
<td>0–9</td>
</tr>
<tr>
<td>Schizoid</td>
<td>0–14</td>
<td>0.91</td>
<td>1.70</td>
<td>0–9</td>
</tr>
<tr>
<td>Paranoid</td>
<td>0–14</td>
<td>0.86</td>
<td>1.69</td>
<td>0–12</td>
</tr>
<tr>
<td>Substance abuse</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drug impairment</td>
<td>0–6</td>
<td>0.56</td>
<td>1.05</td>
<td>0–5</td>
</tr>
<tr>
<td>Alcohol impairment</td>
<td>0–6</td>
<td>0.89</td>
<td>0.81</td>
<td>0–5</td>
</tr>
<tr>
<td>Functioning</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Global Assessment Scale</td>
<td>1–100</td>
<td>74.6</td>
<td>9.3</td>
<td>50–90</td>
</tr>
<tr>
<td>Affective episodes</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(percent of sample)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Major depression</td>
<td></td>
<td>14.7%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mania/hypomania</td>
<td></td>
<td>2.9%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

IPDE: International Personality Disorders Examination.
In terms of spectrum personality disorders, both schizotypy dimensions predicted schizotypal personality disorder ratings. Neuroticism was predictive over-and-above the effects of the schizotypy dimensions. The positive schizotypy × neuroticism interaction, but not the interaction with negative schizotypy, accounted for significant variance over the main effects. Simple slope analyses revealed that the relation between positive schizotypy and the dependent measure was significant at all levels of neuroticism (see Fig. 2), but strengthened as neuroticism increased, low ($\beta = 0.20$; $p = 0.03$), moderate ($\beta = 0.37$; $p < 0.001$), and high ($\beta = 0.54$; $p < 0.001$). As expected, only negative schizotypy predicted schizoid personality ratings. For paranoid personality, both positive and negative schizotypy accounted for significant variance. In addition, neuroticism also accounted for significant variance over-and-above the effects of the schizotypy dimensions. The positive schizotypy × neuroticism interaction accounted for significant variance over the main effects. Simple slope analyses revealed that the relation between positive schizotypy and paranoid ratings was significant at moderate ($\beta = 0.18$; $p = 0.01$) and high ($\beta = 0.35$; $p < 0.001$) levels of neuroticism, but not at low levels ($\beta = 0.004$; $p = 0.97$).

Regarding substance impairment, positive schizotypy (but not negative) predicted levels of drug impairment, whereas neuroticism was not predictive. However, the positive schizotypy × neuroticism interaction, but not the interaction with negative schizotypy, accounted for a significant increment in variance over the main effects. Simple slope analyses

Table 2
Relationship of schizotypy and neuroticism with interview measures of psychopathology and functioning.

<table>
<thead>
<tr>
<th>Criterion</th>
<th>Step 1</th>
<th>Step 2</th>
<th>Step 3</th>
<th>Total $R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Positive schizotypy</td>
<td>Negative schizotypy</td>
<td>Neuroticism</td>
<td>Neuroticism × pos. schizotypy</td>
</tr>
<tr>
<td>Psychotic symptoms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Experiences</td>
<td>$0.49^{***}$</td>
<td>$0.09$</td>
<td>$0.19^{**}$</td>
<td>$0.17^{**}$</td>
</tr>
<tr>
<td>Negative symptoms</td>
<td>$0.05$</td>
<td>$0.52^{***}$</td>
<td>$0.05$</td>
<td>$0.10$</td>
</tr>
<tr>
<td>PDE dimensional scores</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schizotypal</td>
<td>$0.47^{***}$</td>
<td>$0.20^{**}$</td>
<td>$0.23^{***}$</td>
<td>$0.20^{**}$</td>
</tr>
<tr>
<td>Schizoid</td>
<td>$0.04$</td>
<td>$0.43^{***}$</td>
<td>$0.06$</td>
<td>$0.13$</td>
</tr>
<tr>
<td>Paranoid</td>
<td>$0.26^{***}$</td>
<td>$0.21^{**}$</td>
<td>$0.17^{*}$</td>
<td>$0.21^{**}$</td>
</tr>
<tr>
<td>Substance abuse</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Drug impairment</td>
<td>$0.36^{***}$</td>
<td>$-0.07$</td>
<td>$0.10$</td>
<td>$0.18^{*}$</td>
</tr>
<tr>
<td>Alcohol impairment</td>
<td>$0.17^{***}$</td>
<td>$-0.08$</td>
<td>$-0.05$</td>
<td>$0.05$</td>
</tr>
<tr>
<td>Functioning</td>
<td>$-0.28^{***}$</td>
<td>$-0.34^{***}$</td>
<td>$-0.26^{***}$</td>
<td>$0.02$</td>
</tr>
</tbody>
</table>

*p < 0.05, **p < 0.01, and ***p < 0.001.

PDE: Personality Disorder Examination.

Fig. 1. Relationship between levels of positive schizotypy and psychotic-like experiences at three levels of neuroticism (low, medium, high) as indicated by simple slope analysis.

Fig. 2. Relationship between levels of positive schizotypy and schizotypal personality disorder at three levels of neuroticism (low, medium, high) as indicated by simple slope analysis.
revealed that the relationship between positive schizotypy and drug impairment was significant at moderate ($\beta = 0.28$; $p < 0.001$) and high ($\beta = 0.45$; $p < 0.001$) levels of neuroticism, but not at low levels ($\beta = 0.12$; $p = 0.25$) of the moderator variable. Positive (but not negative) schizotypy predicted alcohol impairment. Neither neuroticism nor the interactions accounted for significant increments in variance in this measure. The positive and negative schizotypy dimensions equally contributed in accounting for GAS, with neuroticism also accounting for significant variance over-and-above schizotypy dimensions. However, the interaction terms were not significant.

Binary logistic regressions were conducted to assess the prediction of mood episodes (see Table 3). Positive schizotypy and neuroticism were significantly associated with episodes of major depression; however, none of the other predictor measures was significant. Only positive schizotypy was associated with episodes of mania or hypomania.

Finally, consistent with previous studies (Goodwin et al., 2003; Krabbendam et al., 2002; van Os and Jones, 2001), we reanalyzed the data partialling out history of mood disorder at the first step. The results of these analyses, in which history of mood episodes was entered at a first step in the regression models, showed that all results remained substantively unchanged.

4. Discussion

The results of this study showed that, as hypothesized, neuroticism predicted psychotic-like experiences, schizotypal and paranoid personality disorder symptoms, depressive episodes, and poorer adjustment, over-and-above the contribution of both positive and negative schizotypy. These findings are consistent with three large-scale population-based prospective studies of risk factors for psychosis. These epidemiological studies supported the role of neuroticism as a risk factor for the development of psychosis in individuals with no lifetime history of psychosis (Goodwin et al., 2003; Krabbendam et al., 2002; van Os and Jones, 2001). Importantly, this effect persisted after controlling for co-morbid psychiatric disorders. The prospective and large-scale nature of these studies supports the hypothesis that affective traits are not merely secondary consequences of psychosis, but may be etiologically relevant for the development of psychosis.

The results of the present study also supported the hypothesis that neuroticism is a moderator of the association between positive schizotypy and schizophrenia-spectrum psychopathology and impairment. The strength of the association between positive schizotypy and psychotic-like symptoms, schizotypal and paranoid personality, and drug impairment was modified according to levels of neuroticism. In most cases, positive schizotypy was significantly associated with the psychopathology and functional ratings at all levels of neuroticism, but the strength of the association increased as neuroticism increased. Of note, this moderating effect remained significant after controlling for lifetime history of mood episodes. In contrast, as expected, negative schizotypy predicted schizoid, schizotypal, paranoid, and negative symptoms, but was not moderated by neuroticism in any of the analyses (i.e., the main effect for neuroticism remains constant across levels of negative schizotypy). This finding is consistent with research indicating that negative schizotypy is associated with diminished emotional expression and deficits in the experience and processing of emotion (Kerns, 2006); that is, neuroticism would not ‘amplify’ negative schizotypic traits because, by nature, negative schizotypy involves a reduced emotional capacity. It might be that other personality and/or social environment features would be stronger moderators for negative schizotypy, such as social support. Kerns reviewed possible explanations to account for such decreased emotionality, such as decreased physiological responses to emotional information (Simons et al., 1993), a paucity of cognitive interpretations that generate emotion and increase emotional intensity (Larsen et al., 1996), and a decreased influence of emotions on other aspects of information processing (Coffey et al., 2003).

Numerous researchers dating to back to Bleuler (1911/1950) have suggested that what we now term as negative symptoms represent the fundamental deficit in schizophrenia and that positive symptoms represent accessory symptoms that occur across a wide spectrum of psychotic disorders (e.g., Andreasen, 1982). Research examining positive and negative schizotypy dimensions in nonclinical samples has found comparable patterns of correlates. For example, Kwapiel (1998) reported that social anhedonia was specifically associated with the development of schizophrenia-spectrum disorders in a ten-year follow-up; in contrast to longitudinal findings that measures of positive schizotypy were associated with the development of wide array of disorders such as mood and nonmood psychoses, nonpsychotic mood disorders, and substance use disorders (Chapman et al., 1994). The present findings are consistent with these conjectures and research findings in that the negative schizotypy dimensional score was associated with schizophrenia-spectrum symptoms, whereas positive schizotypy was associated with a much wider array of psychopathological characteristics.

The current findings support the notion that, rather than being a risk factor for specific pathological outcomes,
neuroticism functions as an ‘emotional amplifier’ that potentiates personality traits (such as schizotypy) to augment risk of impairment (Claridge and Davis, 2001). The challenge then is to explain how this moderating effect occurs. It is suggested that cognitive and emotional processes underlying neuroticism traditionally associated with affective disorders also contribute to the liability for psychosis (Krabbendam and van Os, 2005). In this regard, several explanations have been posed that may account for the impact of negative affect in worsening the expression of schizotypy. Garety et al. (2001) suggested that the pairing of unusual experiences with negative emotions produces psychotic appraisals because these experiences become personally significant and trigger a search for an explanation. Furthermore, the presence of high distress may contribute to the maintenance of the psychotic appraisal through processes traditionally associated with anxiety, such as information-processing biases and safety-behaviors that prevent consideration of disconfirmatory evidence. In addition, the attitudinal and information-processing biases associated with neuroticism may decrease levels of social interaction and reduce the availability of resources for identifying and addressing early signs of abnormal mental states (Jones, 1999). Finally, epidemiological research indicates that transitions across the psychosis continuum are driven by emotional responses. Individuals who react with negative emotional states and/or a symptomatic coping style (i.e., concentrating on emotions/content of psychotic symptoms instead of active strategies such as problem solving, distraction or seeking help) have an increased risk for experiencing worsening psychotic experiences and developing psychotic disorders (Krabbendam and van Os, 2005).

The contribution of neuroticism to schizophrenia-spectrum personality traits and symptoms has theoretical and clinical implications. From a theoretical standpoint, it clarifies the complex relationship between personality and psychopathology in this research area. A widely accepted view of how affective and spectrum traits are connected holds that deviations found in personality are the long-term consequence of the disruptive effects of psychotic psychopathology without establishing any causal association (i.e., the so-called ‘scar’ or complication hypothesis). However, the literature reviewed above, along with the present study, suggests that this is not the only connection between affective traits and schizophrenia spectrum. This does not mean that experiencing repeated episodes of psychosis and residual symptoms does not affect personality traits causing ‘secondary’ deviancy. Indeed, this might be even more likely in psychosis than in other disorders given its pervasive nature. However, evidence indicates that temperament and personality reflect the vulnerability to particular types of pathological functioning (Barrantes-Vidal et al., 2002; Clark, 1994). From a clinical standpoint, this growing literature contradicts the view that patients with schizophrenia-spectrum disorders are fundamentally devoid of emotional experience, an assumption that has consequences for treatment (Horan et al., 2008).

The results of this study must be interpreted in light of its limitations. The use of a college student sample may limit the generalizability of the results, because more severely impaired members of this age cohort may be limited in their ability to enroll in college. However, this actually makes a more conservative test of the hypotheses. Furthermore, cross-sectional and longitudinal studies have repeatedly indicated that college students who score highly on psychometric inventories of schizotypy demonstrate schizophrenic-like impairment and heightened risk for developing schizophrenia-spectrum disorders. The predominance of women in this sample raised concerns that negative schizotypy may be underrepresented in the study, given that women with schizophrenia report fewer negative symptoms than male patients. However, both positive and negative symptoms were comparable in range and distribution in men and women. The findings are also limited by the cross-sectional design. Longitudinal studies are required to determine whether personality traits potentiate clinical outcomes in at-risk individuals.

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Contributors
Neus Barrantes-Vidal and Thomas Kwapi designed the study. Neus Barrantes-Vidal was the primary author of the manuscript. Agnês Ros-Morente participated in the manuscript preparation and data analyses. Thomas Kwapi oversaw data collection and statistical analyses. All authors contributed to and have approved the final manuscript.

Conflict of interest
None of the authors had a conflict of interest.

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