Psychiatry Research 240 (2016) 181–186

Contents lists available at ScienceDirect

Psychiatry Research

journal homepage: www.elsevier.com/locate/psychres

Negative symptoms in psychometrically defined schizotypy: The role of depressive symptoms

Timothy R. Campellone *, Ori Elis, Jasmine Mote, Amy H. Sanchez, Ann M. Kring

Department of Psychology, University of California, Berkeley, 3210, Tolman Hall, Berkeley 94720-1650, CA, USA

ARTICLE INFO

Article history:
Received 27 May 2015
Received in revised form
21 February 2016
Accepted 8 April 2016
Available online 9 April 2016

Keywords:
Schizotypy
Negative symptoms
Depressive symptoms
Motivation/pleasure
Emotional expressivity
Cognitive
Somatic-affective

ABSTRACT

People high in schizotypy, a risk factor for schizophrenia-spectrum disorders, can have negative symptoms, including diminished experience of motivation/pleasure (MAP) and emotional expressivity (EXP). Additionally, people high in schizotypy often report elevated depressive symptoms, which are also associated with diminished MAP and EXP. In this study, we examined whether negative symptoms were related to schizotypy above and beyond the presence of depressive symptoms. Thirty-one people high in schizotypy and 24 people low in schizotypy were administered the Clinical Assessment Interview for Negative Symptoms (CAINS), an interview-based measure of MAP and EXP negative symptoms and completed a self-report measure of cognitive and somatic-affective depressive symptoms. People high in schizotypy had more MAP negative symptoms than people low in schizotypy, but we found no group differences in EXP negative symptoms. Importantly, the relationship between MAP negative symptoms and schizotypy was fully mediated by cognitive depressive symptoms. These findings suggest that depressive symptoms, specifically cognitive depressive symptoms, may be a pathway for motivation and pleasure impairment, in people at elevated risk for developing schizophrenia-spectrum disorders.

1. Introduction

Schizotypy refers to a set of personality traits (e.g., unusual perceptions, social isolation, odd behavior) that represent a risk for schizophrenia-spectrum disorders (e.g. Lenzenweger, 2006). Similar to people with schizophrenia, people high in schizotypy can have deficits in the experience of motivation and pleasure or MAP (Yan et al., 2011; Fervaha et al., 2014) as well as emotional expressivity or EXP (Kerns, 2006; Cohen et al., 2009), which together represent the two broad domains of negative symptoms commonly found in people with schizophrenia (Blanchard and Cohen, 2006; Foussias and Remington, 2010; Kring et al., 2013). However, the relationship between schizotypy and negative symptoms is complicated by the fact that people high in schizotypy can also experience high levels of depressive symptoms (e.g. Lewandowski et al., 2006), which are also associated with both MAP and EXP (e.g., Gaebel and Wölwer, 2004; Sherdell et al., 2012). Thus, the primary goal of this study is to examine whether negative symptoms are related to schizotypy above and beyond the presence of depressive symptoms.

There are two broad approaches to the study of schizotypy. The multidimensional approach divides schizotypy into positive and negative dimensions, which largely map onto the phenomena captured by the positive and negative symptom domains of schizophrenia (Kwapil et al., 2008). Studies using the multidimensional approach investigate how elevations in positive and/or negative schizotypy dimensions differentially relate to variables of interest. The other approach, and the approach used in this study, focuses on schizotypy as a unifying construct. Schizotypy is unifying in the sense that it bridges the divide between people at low risk for the development of schizophrenia-spectrum disorders and people with a formal schizophrenia-spectrum diagnosis. In other words, rather than looking at elevations on positive or negative schizotypy dimensions, the unifying approach compares people high and low in schizotypy by selecting participants at the extreme ends of the schizotypy distribution in order to investigate risk factors for the development of schizophrenia-spectrum disorders. These two approaches are not mutually exclusive as studies can choose to stratify their participant recruitment on elevations on a particular schizotypy dimension. In this study, we chose to collapse across schizotypy dimensions to focus on people that were high or low in overall schizotypy traits.

Negative symptoms are an important area of inquiry in schizotypy for several reasons. First, the mechanisms that cause and maintain negative symptoms are poorly understood. Second, like for people with schizophrenia (e.g. Milev et al., 2005), negative symptoms have been linked to poorer quality of life for people high in schizotypy (Cohen and Davis, 2009). Third, negative

* Corresponding author.
E-mail address: tcampellone@berkeley.edu (T.R. Campellone).

http://dx.doi.org/10.1016/j.psychres.2016.04.020
0165-1781/

symptoms represent a critical unmet treatment need among people with schizophrenia (Buchanan, et al., 2010; Elis, Caponigro, & Kring, 2013). Investigating negative symptoms in people high in schizotypy may help increase and expand our understanding of potential mechanisms underlying MAP and EXP to the schizophrenia-spectrum more generally. Indeed, previous research has shown that people high in schizotypy (not just people high in negative schizotypy) self-report diminished MAP and EXP (e.g. Yan et al., 2011; Fervaha et al., 2014; Mitchell et al., 2015).

One important consideration in studying the relationship between negative symptoms and schizotypy is the potential contributions of depressive symptoms. Indeed, diminished pleasure is also a symptom of depression (DSM-5; American Psychiatric Association, 2013) and people with depressive symptoms can also have diminished emotional expressivity (e.g. Gaebel and Wölwer, 2004). Thus, elevations in negative symptoms may reflect an overlap with depressive symptoms (Kirkpatrick et al., 2006). Further, people high in schizotypy often have impairments in MAP and EXP as well as elevations in depressive symptoms, making it unclear whether negative symptoms are related to schizotypy above and beyond the presence of depressive symptoms. One approach to avoiding issues of such comorbidity has been to exclude participants who are elevated in both schizotypy (or a particular schizotypy dimension) and depression (e.g. Cohen et al., 2012). While this approach allows for the isolation of participants who are high in schizotypy without comorbid elevations in depressive symptoms, it does not resolve whether negative symptoms are associated with schizotypy alongside depression symptoms.

Although negative symptoms and depression are related, it may be the case that MAP and EXP negative symptoms are differentially related to particular depressive symptoms. For example, negative cognitive styles such as defeatist beliefs about the ability to perform activities, common among people with elevated depressive symptoms, have been linked to deficits in motivation/pleasure negative symptoms among people with schizophrenia (e.g. Green et al., 2012). Thus, motivation/pleasure negative symptoms in people high in schizotypy may be more related to cognitive depressive symptoms (e.g. thoughts of failure, defeat, and worthlessness) compared to somatic-affective symptoms (e.g., sadness, tiredness). By excluding people who report depressive symptoms in studies of schizotypy, researchers may be excluding people that possess potential mechanisms (e.g., defeatist beliefs) that contribute to negative symptoms in the broader schizophrenia-spectrum.

In this study, we sought to investigate whether schizotypy was related to negative symptoms above and beyond the presence of depressive symptoms. We recruited participants who were high or low in schizotypy–regardless of their depressive symptoms–in order to test several hypotheses. First, in line with previous research, we hypothesized that people high in schizotypy would have more negative (MAP and EXP) and depressive symptoms (cognitive and somatic-affective) than people low in schizotypy. Second, based on recent research highlighting the role of depressive cognitions in negative symptoms in schizophrenia (e.g., Green et al., 2012), we tested whether cognitive or somatic-affective depressive symptoms mediated the relationship between level of schizotypy and each negative symptom domain. Given the findings from research in people with schizophrenia, we hypothesized that cognitive depressive symptoms would mediate the relationship between schizotypy and motivation/pleasure negative symptoms. Because the link between EXP negative symptoms and specific depressive symptoms is less clear, we conducted exploratory analyses to examine whether specific depressive symptoms mediated the relationship between schizotypy and EXP negative symptoms.

2. Methods

2.1. Participants

Undergraduate students were invited to complete an online survey that contained demographic questions as well as the Schizotypal Personality Questionnaire, Brief Revised (SPQ-BR; Cohen et al., 2010), a 32-item questionnaire used for assessing schizotypy. We received 2832 participant responses to the survey over the course of 3 semesters. To identify participants for the high (HS) and low (LS) schizotypy groups, we selected participants that were two or more standard deviations above or below the mean SPQ-BR total score in a given semester and invited people who met the inclusion criteria to participate in the main study. Our approach of deriving a HS group by selecting participants two standard deviations above the mean is similar to previous approaches (e.g. Cohen et al., 2014). Given our interest in the relationship between levels of schizotypy, negative symptoms and depressive symptoms, we used a conservative approach to recruiting our LS group. That is, while studies typically apply less stringent selection criteria for the recruitment of LS groups (e.g., lowest 15% of schizotypy scores; Williams, Henry, & Green, 2007), our LS group was comprised of participants that were two standard deviations below the mean. Sampling the high and low extremes (top and bottom 5%) of the schizotypy distribution allowed us to better assess both the relationship between schizotypy and negative symptoms as well as whether this relationship might be better accounted for depressive symptoms. Specifically, this sampling approach allowed us to isolate how the presence or relative absence of schizotypy related to MAP and EXP negative symptoms and depressive symptoms. Our final sample included 31 people in the HS group and 24 people in the LS group (see Table 1 for demographic information).

2.2. Clinical assessment

2.2.1. Clinical Assessment Interview for Negative Symptoms (CAINS)

We measured negative symptoms with the Clinical Assessment Interview for Negative Symptoms (CAINS; Kring et al., 2013). Trained masters-level graduate students served as interviewers, who rated participants' engagement and interest in motivated

Table 1
Demographic information, clinical interview, and self-report data for the high schizotypal personality trait (HS) and low schizotypal personality trait (LS) groups.

	HS (n=31)	LS (n=24)	Comparison (t or χ^2)
Age (years)	20.45 (2.6)	20.08 (1.6)	$p=0.54$
% Male	23%	25%	$p=0.83$
Racial Background (%)			
Caucasian	12.9%	44.0%	$p=0.01$
Asian	54.8%	20.0%	$p=0.01$
Black	3.2%	8.0%	ns
Hispanic	9.7%	4.0%	ns
Other/Multiple races	19.4%	24.0%	ns
SPQ-BR			
Total	125.23 (7.5)	42.20 (3.7)	$p<0.01$
Positive	44.23 (15.2)	25.60 (13.6)	$p<0.01$
Negative	35.68 (11.6)	18.69 (12.1)	$p<0.01$
Disorganized	29.68 (9.7)	15.52 (8.4)	$p<0.01$
CAINS			
MAP scale	11.35 (5.6)	6.46 (3.8)	$p<0.01$
EXP scale	2.87 (2.9)	2.00 (2.6)	$p=0.27$
BDI-II			
Somatic-Affective	11.37 (4.8)	3.96 (1.9)	$p<0.01$
Cognitive	4.96 (4.0)	1.29 (2.0)	$p<0.01$

SPQ-BR=Schizotypal Personality Questionnaire, Brief Revised, CAINS=Clinical Assessment Interview for Negative Symptoms, MAP=Motivation and Pleasure, EXP=expressivity, BDI=Beck Depression Inventory.

behavior as well as pleasure derived from social, work/school, and recreational activities over the past week using the CAINS Motivation and Pleasure (MAP) scale. The nine MAP items were rated using a 0–4 scale, with higher scores reflecting greater impairment. MAP scale internal consistency for each group was as follows: HS $\alpha=0.73$, LS $\alpha=0.68$. Interviewers rated expressivity during the interview using the CAINS Expressivity (EXP) scale. The four EXP items were rated using a 0–4 scale, with higher scores reflecting greater impairment. EXP scale internal consistency for each group was as follows: HS $\alpha=0.80$, LS $\alpha=0.83$.

2.2.2. Beck depression inventory

We measured depression with the Beck Depression Inventory (BDI; Beck and Steer, 1987), a 21-item self-report measure of depressive symptoms. BDI internal consistency for the HS $\alpha=0.84$ and the LS groups $\alpha=0.79$ were acceptable. To test our hypothesis about particular depressive symptoms, we computed two subscale scores: somatic-affective and cognitive (see Arnau et al., 2001). The 12-item somatic-affective subscale contains items related to depressive feelings (e.g. sadness, loss of pleasure, tiredness), and the cognitive subscale is comprised of 8-items measuring negative cognitions (e.g. pessimism, guilt, worthlessness).

2.3. Data analysis plan

To investigate our first hypothesis that the HS will have greater MAP and EXP negative symptoms as well as cognitive and somatic-affective depressive symptoms, we conducted multiple independent samples t-tests. All between-group comparisons were Bonferroni corrected, with the family-wise significance threshold set at $p=0.013$ to correct for multiple comparisons.

2.3.1. Mediated path analyses

To test our second hypothesis, we conducted mediated path analyses investigating whether particular depressive symptoms mediated the relationship between schizotypy group and negative symptoms. Mediated path analyses were only conducted if there were significant relationships between our predictor (schizotypy group), mediator(s) (cognitive and/or somatic-affective depressive symptoms), and criterion (MAP or EXP negative symptoms) variables. The schizotypy group variable was coded so that positive effects represent high schizotypy. For these analyses we reported direct, indirect, and total effects as unstandardized regression coefficients with standard error estimates. Direct effects refer to the effect of the independent variable (X) on the dependent variable (Y); indirect effects refer to the product of the relationship between X and the mediator (M) and the relationship between M and Y; and total effects refer to the sum of the direct and indirect effects. We used 10,000 bootstrap samples to compute bias-corrected 95% confidence intervals for all indirect effects (Preacher and Hayes, 2004). We computed the ratio of indirect to total effect as an indicator of the amount of variance accounted for by the mediator (Shrout and Bolger, 2002).

Given that many studies adopt the multidimensional approach to schizotypy, we also ran the same analyses for the positive and negative schizotypy dimension totals. Positive and negative schizotypy dimension scores were computed using the 3-factor solution identified by Cohen and colleagues (2010). Positive and negative schizotypy dimension totals can be found in Table 1.

3. Results

The HS and LS groups did not differ in age or sex composition (see Table 1). The HS group had significantly more Asian participants and significantly fewer White participants than the LS group.[1] Consistent with our first hypothesis, the HS group had more MAP negative symptoms, $t(53)=3.84$, $p<0.01$, somatic-affective depressive symptoms, $t(53)=4.06$, $p<0.01$, and cognitive depressive symptoms, $t(53)=7.00$, $p<0.01$ compared to the LS group. However, there were no group differences in EXP negative symptoms, $t(53)=1.16$, $p=0.25$. Symptom scores by group are presented in Table 1. While the CAINS was originally developed for assessing MAP and EXP negative symptoms among people with schizophrenia, we found CAINS scores conforming to a normal distribution in both schizotypy groups. To further contextualize these scores, the average MAP and EXP negative symptom scale totals from the CAINS validation study of 162 people with schizophrenia were 2.57 and 1.97, respectively.

Next, we tested the relationship between our proposed mediator and criterion variables. Both cognitive and somatic-affective depressive symptoms were associated with MAP ($r=0.58$, $p<0.01$; $r=0.54$, $p<0.01$) and EXP negative symptoms ($r=0.29$, $p=0.03$; $r=0.26$, $p=0.05$). However, because the direct path between schizotypy group and EXP was not significant, we only conducted a mediated path analysis for MAP negative symptoms for which the direct path was significant. Given that both cognitive and somatic-affective depressive symptoms were significantly related to both schizotypy group and MAP negative symptoms, we included both of these variables as potential mediators in our model.

Fig. 1a depicts the significant, direct effect of schizotypy group on MAP negative symptoms without either depressive symptom domain in the model. Fig. 1b shows the mediated path analysis with cognitive and somatic-affective depressive symptoms in the model. The coefficient and bootstrap confidence interval for the indirect effect of cognitive depressive symptoms on MAP negative symptoms was $B=0.65$, SE $=0.25$, CI [0.14 to 1.15]. The coefficient and bootstrap confidence interval for the indirect effect of somatic-affective depressive symptoms on MAP negative symptoms scores was $B=0.05$, SE $=0.22$, CI [-0.38 to 0.49]. Since the confidence interval for somatic-affective depressive symptoms contained zero, only cognitive depressive symptoms mediated the relationship between schizotypy group and MAP negative symptoms. The ratio of indirect to total effect was 0.48, indicating that cognitive depressive symptoms accounted for close to half of the effect of the relationship between schizotypy and MAP negative symptoms. Further, the amount of variance explained by the model increased from 0.30 to 0.38 with the addition of cognitive depressive symptoms, which was significant, $F\text{-}change(1,51)=6.64$, $p=0.01$. While not a primary focus of this study, we also found that cognitive depressive symptoms mediated the relationship between MAP negative symptoms and both the negative and positive schizotypy dimensions.

4. Discussion

In this study, we investigated whether schizotypy was associated with MAP and EXP negative symptoms above and beyond the presence of cognitive and somatic-affective depressive symptoms. We had three main findings. First, compared to the LS group, the HS group had greater MAP negative symptoms as well as cognitive and somatic-affective depressive symptoms. Second, the LS and HS groups did not differ in levels of EXP negative symptoms. Third, the relationship between schizotypy and MAP negative symptoms was fully mediated by cognitive depressive symptoms.

[1] Results of the mediated path analyses were the same when controlling for the racial composition of our sample.

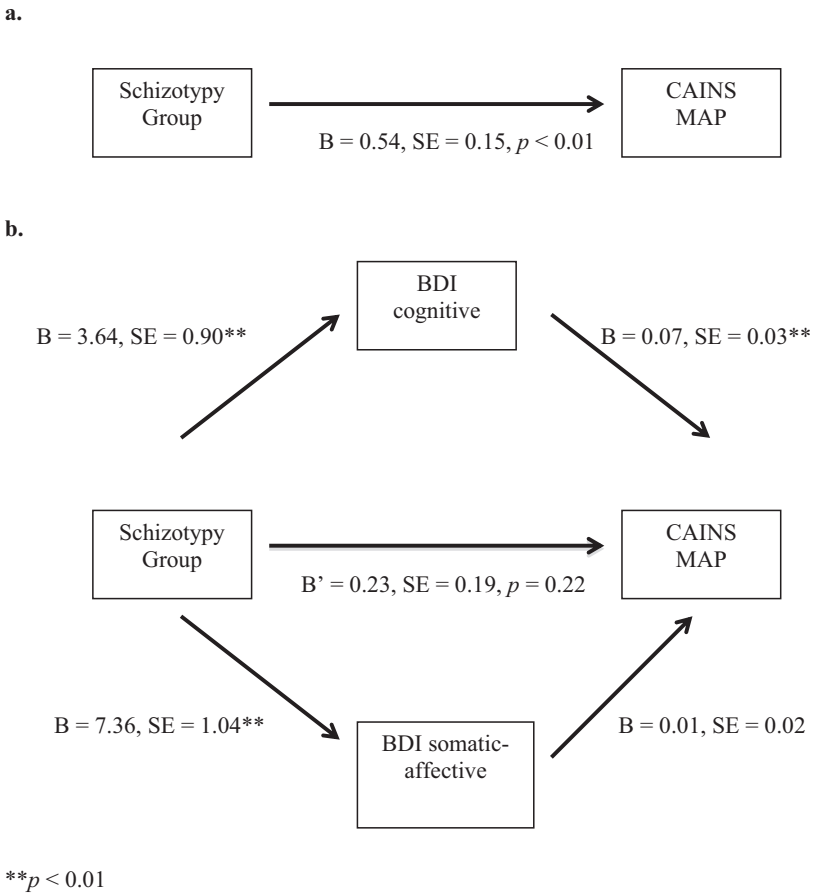

Fig. 1. Mediated path analysis testing whether somatic-affective or cognitive depression symptoms mediated the relationship between schizotypy group status and MAP negative symptoms. (a) Significant direct path between schizotypy group status and MAP negative symptoms. (b) Significant full mediation of the relationship between schizotypy group status and MAP negative symptoms by cognitive, but not somatic-affective negative symptoms.

Consistent with our hypotheses and previous work (Yan et al., 2011; Fervaha et al., 2014, the HS group had more MAP negative symptoms compared to the LS group. Our findings of greater MAP scores in the HS group is consistent with the idea of schizotypy as a unifying construct as the mean scores fell between people at low risk (LS group) and people with formal schizophrenia-spectrum diagnoses (see Kring et al., 2013).

Contrary to our hypothesis, we did not find differences in EXP negative symptoms between the LS and HS groups. This is inconsistent with previous work, which has found differences in emotional expressivity between people high and low in schizotypy (e.g. Kerns, 2006; Cohen et al., 2009; Cohen and Hong, 2011). We suggest two possible reasons for these discrepancies, one having to do with differences in eliciting emotion expressivity and the other having to do with measuring expressivity. In the present, study, we used a clinical interview to elicit emotion and then rate expressivity. It may well be that the CAINS interview was not very emotionally evocative even though we asked questions about emotional experiences, and thus may not have been sensitive enough to detect differences in emotional expressivity between people reporting high and low levels of schizotypy. Other studies assessing emotional expressivity in schizotypy have used more evocative stimuli such as emotional pictures (Cohen and Hong, 2011) or discussing positive and negative autobiographical memories (Cohen et al., 2011).

Perhaps a more plausible reason for the discrepancy has to do with measurement. Previous studies have typically only used self-report measures of expressivity (e.g. Kerns, 2006; Cohen et al., 2009; Cohen and Hong, 2011). Recent studies that also included computer-based assessment measures of emotional expressivity in people with and without schizotypy found group differences in the self-report measures only (Cohen et al., 2009; Cohen et al., 2011). Our findings support the divergence found in these studies and suggest that while people high in schizotypy may *report* being less emotionally expressive, their *behavior* appears to be just as expressive as people low in schizotypy. Given the evidence for discrepancies between self-report and behavior in people high in schizotypy, it will be important for future studies investigating emotional expressivity in schizotypy to take a multi-method approach.

Consistent with previous work, we found elevated depressive symptoms in people high in schizotypy (e.g. Lewandowski et al., 2006), and this was true for both cognitive and somatic-affective depressive symptoms. Thus, our findings extend prior work by showing that people high in schizotypy may experience elevations in both types of depressive symptoms. By computing separate cognitive and somatic-affective depressive symptom totals, we were able test whether *specific* depression symptoms might mediate the relationship between schizotypy and MAP negative symptoms. Indeed, consistent with our hypothesis, we found evidence for full mediation of the relationship between schizotypy and MAP negative symptoms by cognitive depressive symptoms, even after controlling for the effect of somatic-affective depressive symptoms. This finding is noteworthy for two reasons. First, it is in line with a growing body of evidence positioning negative cognitive styles such as those found in depression as a potential mechanism contributing to the MAP negative symptom domain (e.g. Grant and Beck, 2009). Thus, future studies seeking to understand

the development and maintenance of MAP negative symptoms should investigate the role of cognitive depressive symptoms as these may serve as a link between relative risk for developing schizophrenia-spectrum disorders and MAP negative symptoms.

Second, our finding of cognitive depressive symptoms mediating the relationship between schizotypy and MAP negative symptoms is in line with the idea that cognitive depressive symptoms may be a common pathway to motivation and pleasure impairment for psychopathology more generally. Of note, we found this same mediational result for schizotypy totals and dimensions. Previous research has found impairments in motivation and pleasure among people with other psychological disorders besides the schizophrenia spectrum, such as anxiety (DeVido et al. 2009) and mania (Pizzagalli et al., 2008), both of which are characterized by high rates of comorbidity with depression (Kessler et al., 2003; Merikangas et al., 2008). Our investigation of discrete (high or low schizotypy) as well as dimensional (cognitive and somatic-affective depressive symptoms) predictors of MAP negative symptoms is in line with recent shifts in the research framework for mental illness emphasizing common pathways to impairment (e.g. Insel et al., 2010). Thus, it may be the case that elevations in cognitive depressive symptoms, such as thoughts of failure, defeat, and worthlessness, represent a transdiagnostic risk factor for deficits in motivation and pleasure, which are also transdiagnostic in nature. Further, given that depressive symptoms are quite prevalent in the United States, with prevalence estimates ranging from 18 to 20% (e.g. Saluja et al., 2004; Shim et al., 2011), elevations in depressive symptoms may help to explain motivation and pleasure impairments experienced by people, regardless of diagnostic status.

Why did cognitive but not somatic depressive symptoms predict MAP negative symptoms? Indeed, the somatic-affective subscale included reported feelings of anhedonia, which along with symptoms of motivation represent one of the two negative symptom domains. However, the somatic-affective subscale also includes other feelings common in depression (e.g. guilt) that may not be associated with motivation and pleasure. Thus, while the BDI item assessing anhedonia was associated with MAP negative symptoms in both the LS, $r(24)=.60$, $p<0.01$, and HS groups, $r(31)=.44$, $p=.01$, this effect may have been washed out by the other symptoms included in the somatic-affective subscale.

The racial composition of our high and low schizotypy groups was not balanced, with more Asian participants in the HS group and more White participants in the LS group. While the higher number of Asian participants reflects the demographics of the UC Berkeley undergraduate population, differences in racial composition between our two groups suggest that cultural factors could have played a role in the endorsement of schizotypy. While cultural differences in self-reported schizotypy have been found in previous studies (e.g. Cohen et al., 2009), the role of culture in self-reported schizotypy remains unclear (Cohen et al., 2015). Thus, future studies should seek to unpack how ethnicity, cultural identity, and other factors may affect self-reported schizotypy.

In summary, we found that people high in schizotypy had greater MAP but not EXP negative symptoms as well as both cognitive and somatic-affective depressive symptoms than people low in schizotypy. The relationship between schizotypy and MAP negative symptoms was fully mediated by cognitive depressive symptoms. These findings suggest that depressive symptoms, and more specifically cognitive depressive symptoms, may be a pathway to motivation and pleasure impairment for people at high risk for developing schizophrenia-spectrum disorders. Future studies should investigate whether cognitive depressive symptoms might be a common pathway to impairment in other psychopathologies. Such research could point to depressive cognitions as an important target for the developing interventions for people more generally who may be experiencing decreases in motivation and pleasure.

Funding

US National Institutes of Mental Health grant 5T32MH089919 to TRC.

Contributors

TRC, OE, JM and AHS helped design the study as well as collect and analyze the data. TRC and AMK wrote the manuscript. OE, JM and AHS provided feedback on previous drafts.

Conflicts of interest

None of the authors have any conflicts to disclose.

Acknowledgments

We would like to thank all of participants who made this study possible.

References

American Psychiatric Association, 2013. Diagnostic and Statistical Manual of Mental Disorders, 5th ed. American Psychiatric Publishing, Arlington, VA.
Arnau, R.C., Meagher, M.W., Norris, M.P., Bramson, R., 2001. Psychometric evaluation of the Beck Depression Inventory-II with primary care medical patients. Health Psychol. 20, 112.
Beck, A.T., Steer, R.A., 1987. Manual for the Revised Beck Depression Inventory. San Antonio, TX; Psychological Corp.
Blanchard, J.J., Cohen, A.S., 2006. The structure of negative symptoms within schizophrenia: implications for assessment. Schizophr. Bull. 32, 238–245.
Buchanan, R.W., Kreyenbuhl, J., Kelly, D.L., Noel, J.M., Boggs, D.L., Fischer, B.A., Himelhoch, S., Fang, B., Peterson, E., Aquino, P.R., Keller, W., 2010. The 2009 PORT psychopharmacological treatment recommendations and summary statements. Schizophr. Bull. 36, 71–93.
Cohen, A.S., Auster, T.L., McGovern, J.E., MacAulay, R.K., 2014. The normalities and abnormalities associated with speech in psychometrically-defined schizotypy. Schizophr. Res. 160, 169–172.
Cohen, A.S., Davis III, T.E., 2009. Quality of life across the schizotypy spectrum: findings from a large nonclinical adult sample. Compr. Psychiatry 50, 408–414.
Cohen, A.S., Hong, S.L., 2011. Understanding constricted affect in schizotypy through computerized prosodic analysis. J. Personal. Disord. 25, 478–491.
Cohen, A.S., Iglesias, B., Minor, K.S., 2009. The neurocognitive underpinnings of diminished expressivity in schizotypy: What the voice reveals. Schizophr. Res. 109, 38–45.
Cohen, A.S., Matthews, R.A., Najolia, G.M., Brown, L.A., 2010. Toward a more psychometrically sound brief measure of schizotypal traits: Introducing the SPQ-Brief revised. J. Personal. Disord. 24, 516–537.
Cohen, A.S., Mohr, C., Ettinger, U., Chan, R.C.K., Park, S., 2015. Schizotypy as an organizing framework for social and affective sciences. Schizophr. Bull. 41, S427–S435.
Cohen, A.S., Morrison, S.C., Brown, L.A., Minor, K.S., 2012. Towards a cognitive resource limitations model of diminished expression in schizotypy. J. Abnorm. Psychol. 121, 109–118.
DeVido, J., Jones, M., Geraci, M., Hollon, N., Blair, R.J.R., Pine, D.S., Blair, K., 2009. Stimulus-reinforcement-based decision making and anxiety: impairment in generalized anxiety disorder (GAD) but not in generalized social phobia (GSP). Psychol. Med. 39, 1153.
Elis, O., Caponigro, J.M., Kring, A.M., 2013. Psychosocial treatments for negative symptoms in schizophrenia: current practices and future directions. Clin. Psych. Rev. 33 (8), 914–928.
Fervaha, G., Zakzanis, K.K., Jeffay, E., Graff-Guerrero, A., Foussias, G., Agid, O., Remington, G., 2014. Amotivation as central to negative schizotypy and their predictive value for happiness. Personal. Individ. Differ. 68, 37–42.
Foussias, G., Remington, G., 2010. Negative symptoms in schizophrenia: avolition and Occam's razor. Schizophr. Bull. 36, 359–369.
Gaebel, W., Wölwer, W., 2004. Facial expressivity in the course of schizophrenia and depression. Eur. Arch. Psychiatry Clin. Neurosci. 254, 335–342.
Grant, P.M., Beck, A.T., 2009. Defeatist beliefs as a mediator of cognitive impairment, negative symptoms, and functioning in schizophrenia. Schizophr. Bull. 35, 798–806.

Green, M.F., Hellemann, G., Horan, W.P., Lee, J., Wynn, J.K., 2012. From perception to functional outcome in schizophrenia: Modeling the role of ability and motivation. Arch. Gen. Psychiatry 69, 1216–1224.
Insel, T., Cuthbert, B., Garvey, M., Heinssen, R., Pine, D.S., Quinn, K., Sanislow, C., Wang, P., 2010. Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. Am. J. Psychiatry 167 (7), 748–751.
Kerns, J.G., 2006. Schizotypy facets, cognitive control, and emotion. J. Abnorm. Psychol. 115, 418–427.
Kessler, R.C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K.R., Rush, A.J., Walters, E.E., Wang, P.S., 2003. The epidemiology of major depressive disorder: results from the National Comorbidity Survey Replication (NCS-R). J. Am. Med. Assoc. 289, 3095–3105.
Kirkpatrick, B., Fenton, W.S., Carpenter, W., Marder, S.R., 2006. The NIMN-MATRICS consensus statement on negative symptoms. Schizophr. Bull. 32, 214–219.
Kring, A.M., Gur, R., Blanchard, J., Horan, W.P., Reise, S., 2013. The clinical assessment interview for negative symptoms (CAINS): final development and validation. Am. J. Psychiatry 170, 165–172.
Kwapil, T.R., Barrantes-Vidal, N., Silvia, P.J., 2008. The dimensional structure of the Wisconsin schizotypy scales: Factor identification and construct validity. Schizophr. Bull. 34, 444–457.
Lenzenweger, M.F., 2006. Schizotaxia, schizotypy, and schizophrenia: Paul E. Meehl's blueprint for the experimental psychopathology and genetics of schizophrenia. J. Abnorm. Psychol. 115, 195.
Lewandowski, K.E., Barrantes-Vidal, N., Nelson-Gray, R.O., Clancy, C., Kepley, H.O., Kwapil, T.R., 2006. Anxiety and depression symptoms in psychometrically defined schizotypy. Schizophr. Res. 83, 225–235.
Merikangas, K.R., Swendsen, J., Rössler, W., Ajdacic-Gross, V., Angst, J., 2008. Specificity of bipolar spectrum conditions in the comorbidity of mood and substance use disorders: results from the Zurich cohort study. Arch. Gen. Psychiatry 65, 47–52.
Milev, P., Ho, B.C., Arndt, S., Andreasen, N.C., 2005. Predictive values of neurocognition and negative symptoms on functional outcome in schizophrenia: a longitudinal first-episode study with 7-year follow-up. Am. J. Psychiatry 162, 495–506.
Mitchell, J.C., Ragsdale, K.A., Bedwell, J.S., Beidel, D.C., Cassisi, J.E., 2015. Sex differences in affective expression among individuals with psychometrically defined schizotypy: diagnostic implications. Appl. Psychophysiol. Biofeedback, 1–9.
Pizzagalli, D.A., Goetz, E., Ostacher, M., Iosifescu, D.V., Perlis, R.H., 2008. Euthymic patients with bipolar disorder show decreased reward learning in a probabilistic reward task. Biol. Psychiatry 64, 162–168.
Preacher, K.J., Hayes, A.F., 2004. SPSS and SAS procedures for estimating indirect effects in simple mediation models. Behav. Res. Methods 36, 717–731.
Saluja, G., Iachan, R., Scheidt, P.C., Overpeck, M.D., Sun, W., Giedd, J.N., 2004. Prevalence of and risk factors for depressive symptoms among young adolescents. Arch. Pedia. Adolesc. Med. 158, 760–765.
Sherdell, L., Waugh, C.E., Gotlib, I.H., 2012. Anticipatory pleasure predicts motivation for reward in major depression. J. Abnorm. Psychol. 121, 51–60.
Shrout, P.E., Bolger, N., 2002. Mediation in experimental and nonexperimental studies: new procedures and recommendations. Psychol. Methods 7, 422–445.
Yan, C., Liu, W.H., Cao, Y., Chan, R.C., 2011. Self-reported pleasure experience and motivation in individuals with schizotypal personality disorder proneness. East Asian Arch. Psychiatry 21, 115–122.