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Differentiating positive schizotypy and mania risk scales and their associations with spontaneous eye blink rate

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ABSTRACT

Positive schizotypy and mania risk scales are strongly correlated, and both are linked to alterations in striatal dopamine. Previous research has not examined whether these risk scales form distinct factors or whether they are differentially related to other measures of psychopathology risk or striatal dopamine. In the current study (N = 596), undergraduate students completed both positive schizotypy and mania risk scales as well as scales assessing related psychopathology (i.e., negative and disorganized schizotypy; self-reported manic-like episodes). Additionally, we measured spontaneous eye blink rate, which has been consistently associated with striatal dopamine levels. Positive schizotypy and mania risk factors were strongly correlated (factor correlation = 0.73). However, a two-factor model with positive schizotypy and mania risk as separate factors fit significantly better than a one-factor risk model. After removing shared variance, only positive schizotypy was positively associated with both negative and disorganized schizotypy, and only mania risk was related to self-reported manic-like episodes. Furthermore, positive schizotypy was associated with *decreased* spontaneous eye blink rate, and mania risk was associated with *increased* spontaneous eye blink rate. Overall, these results suggest that positive schizotypy and mania risk can be distinguished as separate factors and that they might be differentially associated with striatal dopamine measures.

1. Introduction

There are a number of self-report scales that attempt to assess either positive schizotypy or mania risk, and there are multiple reasons that researchers use these scales (e.g., to help understand nature of positive schizotypy and mania risk; e.g., Barrantes-Vidal et al., 2015; Eckblad and Chapman, 1986; to screen for people at clinical high risk; Kline and Schiffman, 2014). Schizotypy has been defined as a latent personality organization (Lenzenweger, 2018) and has been posited to be the underlying vulnerability for developing schizophrenia-spectrum psychopathology and is a multidimensional construct that consists of positive schizotypy, negative schizotypy, and disorganized schizotypy (Kwapil and Barrantes-Vidal, 2015). Positive schizotypy (related to but not necessarily synonymous with positive psychotic-like symptoms) is characterized by disruptions in thought content (e.g., magical ideation and delusion-like beliefs), perceptual aberration, and suspiciousness (Kwapil and Barrentes-Vidal, 2015; Lenzenweger, 2010), and individuals high on this trait are at increased risk for schizophrenia-spectrum psychopathology (Kwapil and Barrantes-Vidal, 2015). On the other hand, mania is a state of abnormally elevated or irritable mood. Symptoms of mania typically include increased risk taking and racing thoughts as well as decreased need for sleep. The extent to which positive schizotypy and mania risk are considered to be distinct has been debated, and a potentially critical gap in research related to positive schizotypy and mania risk is that previous research has rarely examined whether positive schizotypy and mania risk scales can be differentiated (Preti et al., 2015). Thus, the current research examined whether positive schizotypy and mania risk scales formed distinct factors in a confirmatory factor analysis, whether these factors were differentially related to other measures of psychopathology, and whether these factors were differentially related to a measure that in part reflects striatal dopamine.

Though there is a long history of separate positive schizotypy and mania risk measures (e.g., Chapman et al., 1978; Eckblad and Chapman, 1983, 1986; Depue et al., 1989; Youngstrom et al., 2008), there are several reasons why it is important to examine whether positive schizotypy and mania risk scales can be differentiated. First, there is evidence that positive schizotypy and mania risk scales are highly correlated (e.g. Applegate et al., 2009) with these correlations being close to or in the range for how strongly positive schizotypy scales correlate with each other and for how mania risk scales correlate with each other (e.g., Cicero and Kerns, 2010b; Miller et al., 2011). Second,

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there is a long line of evidence demonstrating that psychotic and manic disorders are highly related (e.g., high comorbidity; genetic overlap; e.g., Cross-Disorder Group of the Psychiatric Genomics Convention, 2013). Finally, both have been linked to the same neurobiological mechanism: striatal dopamine. The striatum is the primary input region of the basal ganglia (Gerfen and Surmeier, 2011) and receives major dopamine projections: from the ventral tegmental area/dorsal substantia nigra pars compacta to the ventral striatum; and from the ventral substantia nigra pars compacta to the dorsal striatum (Haber and Knutson, 2010; Haber, 2014). The dorsal striatum (for the ventral striatum; Kupchik and Kalivas, 2017) involves two different pathways, a D_1-rich Go pathway that is important in selecting responses and a D_2-rich NoGo pathway that is important in suppressing responses (Maia and Frank, 2017), with D_2 receptors often being implicated in psychosis. For instance, medication that blocks striatal dopamine D_2 receptors is the only effective medication for psychosis (Howes et al., 2012) and is as effective as any other medication for treating acute mania (Scherk et al., 2007; note however that effective treatment does not necessarily indicate a causal relationship; e.g., aspirin's effect on headaches does not mean that headaches are caused by disturbed aspirin function). Additionally, increased striatal dopamine is the best-established neurobiological correlate of psychosis (Howes et al., 2012). Further, mania is associated with alterations in the Behavioral Activation System (Johnson et al., 2012), which has long been linked to striatal dopamine (Lawrence and Brooks, 2014). Given the extensive relationship between positive schizotypy and mania, it is important to directly examine whether positive schizotypy and mania risk scales can be differentiated and whether they are differentially associated with a measure related to striatal dopamine levels.

A physiological measure that has been consistently associated with striatal dopamine levels is spontaneous eye blink rate. It is thought that the brain has a spontaneous blink generator (e.g., to help maintain eye moisture), with evidence that the spinal trigeminal complex might be involved (Kaminer et al., 2011). The striatum/basal ganglia is known to regulate spinal trigeminal complex activity, suggesting that striatal dopamine levels could then affect spontaneous eye blink rate. Consistent with this, a long line of animal and human research has found that striatal dopamine functioning is related to spontaneous eye blink rate, with decreased dopamine being associated with decreased spontaneous eye blink rate and increased dopamine being associated with increased spontaneous eye blink rate (e.g., Slagter et al., 2015). For instance, Parkinson's Disease, which involves decreased striatal dopamine, is associated with decreased spontaneous eye blink rate (Karson et al., 1982; Karson, 1983; for a recent overview of studies on spontaneous eye blink rate and dopamine, see Jongkees and Colzato, 2016). In contrast, there is evidence that spontaneous eye blink rate is increased in unmedicated individuals with psychotic disorders (Karson et al., 1990). Furthermore, animal evidence has consistently found that striatal dopamine manipulations affect spontaneous eye blink rate such that D_2 agonists have been shown to increase spontaneous eye blink rate in monkeys (Groman et al., 2014; Lawrence and Redmond, 1991) and D_2 antagonists have been shown to decrease spontaneous eye blinks rate in monkeys (Kaminer et al., 2011; Lawrence and Redmond, 1991). There is also a strong correlation between baseline spontaneous eye blink rate and D_2 receptors across subregions of the striatum (r = 0.58–0.74; Groman et al., 2014).

Thus, the current research examined whether positive schizotypy and mania risk scales could be differentiated from each other. If they could, then it would be expected that though positive schizotypy and mania risk scales might be strongly correlated, that they would still form distinct factors in a confirmatory factor analysis. In the current research, we examined whether a two-factor positive schizotypy and mania risk model fit significantly better than a one-factor model that included all positive schizotypy and mania risk scales. Further, if positive schizotypy and mania risk scales could be differentiated from each other, then it would also be expected that these latent factors would be differentially associated with other scales reflecting psychopathology risk and symptoms. In particular, one expectation was that after removing variance shared with mania risk, only positive schizotypy scales would be associated with other measures of negative and disorganized schizotypy. In contrast, another expectation was that after removing variance shared with positive schizotypy, only mania risk would be associated with measures of self-reported previous manic-like episodes. Finally, it was also expected that these positive schizotypy and mania risk latent factors would be differentially associated with spontaneous eye blink rate. Again, previous research and theory suggest that both positive schizotypy and mania risk might be associated with an increase in striatal dopamine. However, there are reasons to think that positive schizotypy scales might in fact be associated with a *decrease* in striatal dopamine. While striatal dopamine may transiently increase during an acute psychotic episode (Laruelle et al., 1999), individuals who are at risk for psychosis and are not currently experiencing a psychotic episode may have decreased tonic striatal dopamine levels (Maia and Frank, 2017). Finally, Maia and Frank posit that psychosis is associated with low levels of tonic striatal dopamine. For instance, increased tonic dopamine is associated with increased effort and faster reaction times; however, individuals with psychosis show the opposite pattern (i.e., decreased effort and slower reaction times). In addition, psychotic disorders and genetic risk for psychotic disorders are associated with decreased activation in the limbic striatum (Radua et al., 2015), which is consistent with psychosis risk being associated with decreased tonic dopamine. Hence, if positive schizotypy and mania risk scales can be differentiated from each other, then it might also be expected that these risk scales would be differentially associated with spontaneous eye blink rate, with positive schizotypy being associated with decreased spontaneous eye blink rate and mania risk with increased spontaneous eye blink rate.

2. Methods

2.1. Participants

Participants (N = 639) were undergraduates at University of Missouri. As in previous research (e.g. Karcher et al., 2015), participants (n = 23) endorsing 3 or more items on a 13-item infrequency scale (Chapman and Chapman, 1986), which measures careless and invalid responding, were excluded. Additionally, participants (n = 4) with poor performance on a very simple cognitive task (accuracy < 65% on a task deciding whether two card were same or different; additional description related to this task is in Supplemental Materials) and one participant noted by research assistants as not paying attention while filling out questionnaires (i.e., pressing random buttons very rapidly) were excluded. Lastly, 15 participants were excluded for not completing all questionnaires, resulting in a final sample size of 596 participants as seen in Table 1.

Table 1
Demographics.

Sex	
Male	47.2%
Female	52.5%
Age	
Mean (*SD*)	18.93 (0.99)
Ethnicity	
Asian American	2.5%
African American	12.3%
Caucasian	78.4%
Latino/Latina	1.8%
American Indian/Alaska Native	0.5%
Native Hawaiian/Other Pacific Islander	0.2%
Biracial	2.8%
Other	1.3%
Missing	0.3%