

## Toward a Neural Model of the Openness-Psychoticism Dimension: Functional Connectivity in the Default and Frontoparietal Control Networks

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**Psychosis proneness has been linked to heightened Openness to Experience and to cognitive deficits. Openness and psychotic disorders are associated with the default and frontoparietal networks, and the latter network is also robustly associated with intelligence.** We tested the hypothesis that functional connectivity of the default and frontoparietal networks is a neural correlate of the openness-psychoticism dimension. Participants in the Human Connectome Project ( $N = 1003$ ) completed measures of psychoticism, openness, and intelligence. Resting state functional magnetic resonance imaging was used to identify intrinsic connectivity networks. Structural equation modeling revealed relations among personality, intelligence, and network coherence. Psychoticism, openness, and especially their shared variance were related positively to default network coherence and negatively to frontoparietal coherence. These associations remained after controlling for intelligence. Intelligence was positively related to frontoparietal coherence. Research suggests that psychoticism and openness are linked in part through their association with connectivity in networks involving experiential simulation and cognitive control. We propose a model of psychosis risk that highlights roles of the default and frontoparietal networks. Findings echo research on functional connectivity in psychosis patients, suggesting shared mechanisms across the personality-psychopathology continuum.

**Key words:** schizotypy/personality/fMRI/intelligence/Human Connectome Project

### Introduction

Psychosis refers to a set of symptoms marked by loss of contact with reality, which are present in a range of disorders, most prominently schizophrenia. Schizophrenia symptoms can be divided into positive (hallucinations and delusions), negative (anhedonia and social withdrawal),

and disorganized (confused thought, speech, and behavior) clusters.<sup>1,2</sup> In addition to studying psychosis in its severe manifestations, one can also study symptoms in the general population. Psychotic-like experiences (hallucinations and delusions) occur throughout the general population, even in the absence of disorder, and are distributed dimensionally.<sup>3,4</sup> Persistent psychotic-like characteristics, in the absence of severe mental illness, are often described as schizotypy, which has been conceptualized as part of schizophrenia's extended phenotype.<sup>5</sup> Schizotypy can also be divided into positive, negative, and disorganized traits. Positive schizotypy presents a promising phenotype for studying mechanisms of risk for psychosis and has been referred to as psychoticism.<sup>6</sup> (This construct should not be confused with Eysenck's Psychoticism, a misleadingly named trait reflecting impulsive nonconformity, low agreeableness, and low conscientiousness, but not psychosis proneness.<sup>7,8</sup>) Advantages of studying psychoticism include sampling a broader range of the variables of interest, greater capacity of the target population to participate, and fewer confounds stemming from comorbidity and medications.

Studying normal personality traits in relation to psychoticism can give us a fuller picture of mechanisms and risk factors for psychosis. One promising approach integrates psychoticism with the Five Factor Model or Big Five, a well-established, empirically based model describing personality in terms of Extraversion, Neuroticism, Agreeableness, Conscientiousness, and Openness/Intellect.<sup>9</sup> Major dimensions of risk for psychopathology correspond structurally and conceptually to the Big Five, suggesting that mental disorders typically involve extreme and maladaptive forms of normal personality traits.<sup>10,11</sup> Psychoticism has historically been the psychopathology dimension most difficult to locate within the Big Five,<sup>12–16</sup> but recent research demonstrates that this is due to differential associations with the subfactors of Openness/Intellect. Psychoticism is

specifically associated with openness but unrelated or even negatively related to intellect.<sup>17–19</sup> Although often also positively related to Neuroticism, psychoticism loads onto a separate factor with openness, especially when measured separately from intellect.<sup>20–23</sup> Openness encompasses artistic and aesthetic interests, fantasy proneness, and individual differences in perceptual engagement, whereas Intellect reflects intellectual confidence and engagement with abstract or semantic information and is positively associated with IQ.<sup>18,19</sup> The positive association between psychoticism and openness may arise from the fact that both involve sensitivity of pattern detection, with features like unusual perceptual experiences and magical ideation representing a tendency toward false positives, also known as “*apophenia*.<sup>18,24,25</sup> As a maladaptive form of openness, *apophenia* (and hence psychoticism) reflects “openness to implausible patterns.”<sup>18</sup> Although evidence is beginning to emerge that the neurocognitive mechanisms of psychosis risk overlap with those of openness,<sup>24,25</sup> further research is critical. In the current work, we investigated patterns of functional connectivity associated with psychoticism and openness.

A substantial literature relates psychotic symptoms to abnormal patterns of structural and functional connectivity.<sup>26,27</sup> Whereas structural connectivity refers to the state of the brain’s white matter pathways, functional connectivity refers to patterns of temporal synchrony among brain regions.<sup>28–30</sup> Measuring patterns of functional connectivity in concert with clinical or subclinical assessments may provide a useful mechanistic approach for understanding both full-blown psychosis and psychosis risk throughout the population.

Many studies examining functional connectivity in the psychosis spectrum report atypical connectivity in the default network (DN). Central hubs of the DN are in medial prefrontal cortex and posterior cingulate cortex, with additional nodes in the hippocampus and the parietal and temporal cortices.<sup>31</sup> The DN is involved in many cognitive operations, including episodic memory, future-directed thought, and understanding the mental states of others—in short, anything that requires simulation of experience rather than attention to sensory input. Increased connectivity and activity of the DN have been observed in schizophrenia and in people at high risk for psychosis, in resting-state designs<sup>32–37</sup> and tasks.<sup>36,38–40</sup> Other studies, however, have reported decreased DN connectivity in psychosis.<sup>41–43</sup> Nonetheless, comprehensive reviews suggest that a majority of studies report increased connectivity.<sup>44</sup>

Importantly, the relation of schizophrenia to DN connectivity appears to be specifically linked to positive symptoms.<sup>36,38</sup> Relatives of those with schizophrenia show increased DN connectivity,<sup>33</sup> which is also seen among individuals who report higher levels of mind-wandering<sup>45</sup> and creativity.<sup>46,47</sup> Perhaps not surprisingly, then, DN connectivity is also positively related to multiple facets of openness, even after controlling for intelligence.<sup>25,48,49</sup>

This positive association has been found for global efficiency and connectivity of the DN,<sup>25,48,49</sup> but more specifically with connectivity in its core subsystem, consisting of posterior cingulate and medial prefrontal cortex.<sup>49,50</sup>

Although the DN is perhaps the most studied network in relation to psychosis and openness, portions of the network originally conceptualized as its counterpart—the task positive network—have also been investigated. One neural network is particularly associated with cognitive control and intelligence and is known as the frontoparietal control network (FPCN). The FPCN has primary nodes in dorsolateral prefrontal cortex, lateral parietal cortex, and dorsal anterior cingulate cortex, appears to be responsible for voluntary control of attention, and exhibits reduced connectivity in psychosis.<sup>51</sup> A more substantial literature has linked psychosis to disrupted function of the dorsolateral prefrontal cortex.<sup>52–54</sup> Intelligence shows positive relations to FPCN connectivity<sup>55,56</sup> and prominent frameworks for conceptualizing the neural basis of intelligence, such as Parieto-Frontal Integration Theory, underscore the importance of the FPCN.<sup>57</sup> Although intellect has been linked to greater performance-related activity in the FPCN,<sup>58</sup> possible relations with openness remain unclear.

When studying the neural correlates of traits involved in risk for disorder, it is important to consider intelligence, so as to ensure patterns of connectivity are not merely corresponding to broader deficits.<sup>25</sup> Low intelligence is a common risk factor for psychopathology, including psychosis.<sup>59,60</sup> As noted above, psychoticism also tends to be weakly inversely associated with intelligence and specific cognitive domains (e.g., social cognition, attention, and working memory).<sup>18,19,61–67</sup> Working memory, the ability to manipulate information in short-term memory, is thought to be a core mechanism underlying intelligence, a theory supported both by their strong correlation and shared neural correlates.<sup>68,69</sup> In the current research, we examined whether the neural correlates of intelligence were related to those of psychoticism and openness.

A large number of studies have identified the DN and FPCN as implicated in psychosis. Other research has connected psychoticism to openness, but few studies have investigated psychoticism and normal-range personality together with their neural correlates. The current study examined functional networks associated with psychoticism, openness, and intelligence, using resting state fMRI data from the Human Connectome Project (HCP). We hypothesized that psychoticism and openness would be associated positively with DN coherence and negatively with FPCN coherence. Furthermore, we anticipated intelligence would be positively associated with FPCN coherence.

## Methods and Materials

### Participants

Our sample included 1003 participants (534 females), from the HCP, between ages 22 and 37 ( $M = 28.7$ ,

$SD = 3.7$ ). Participants completed self-report measures and underwent four resting state fMRI scans (for 1-h total scan time). Exclusion criteria included a history of severe psychiatric, neurological, or medical disorders. However, participants were not excluded on the basis of mild psychopathology. Given population estimates, 15%–20% of participants would likely warrant a DSM-5 diagnosis.<sup>70</sup> Informed consent was obtained,<sup>71</sup> and study protocols were approved by the Institutional Review Board of Washington University in St. Louis (IRB # 201204036; “Mapping the Human Connectome: Structure, Function, and Heritability”).

### *Self-report Measures*

**NEO Five-Factor Inventory.** The NEO Five-Factor Inventory (NEO-FFI) is a measure of the Big Five. It consists of 60 items taken from the longer NEO Personality Inventory, Revised (NEO PI-R)<sup>72</sup> and uses a five-point Likert scale. The NEO-FFI does not include subscales for openness and intellect; in order to create an openness aspect scale, correlations of items from the NEO-FFI Openness to Experience scale were examined in relation to openness and intellect from the Big Five Aspect Scales (BFAS).<sup>73</sup> Previous work has been done to extract a similar openness aspect scale using the full NEO PI-R, based on item-associations with BFAS in three samples<sup>74</sup>; this latter scale has been used in previous research examining relations of openness and intellect with psychoticism.<sup>17</sup> Items from this NEO PI-R openness scale that are also included in the NEO-FFI were selected to create our FFI openness scale. Items included “Sometimes when I am reading poetry or looking at a work of art, I feel a chill or wave of excitement,” “I am intrigued by the patterns I find in art and nature,” “I don’t like to waste my time daydreaming (reversed),” and “Poetry has little or no effect on me (reversed).” In the current study, validation was done using the Eugene Springfield Community Sample, where all items in our FFI openness scale had a correlation with BFAS openness greater than 0.30 and this correlation was at least 0.15 greater than for Intellect. There was a very strong positive correlation between latent variables indicated by FFI and BFAS openness items ( $r = 1.0$ ,  $P < .001$ ), and the latent correlation between FFI openness and BFAS intellect was significantly smaller ( $r = .41$ ,  $P = < .001$ ).

At the request of reviewers, who were concerned about our use of a nonstandard openness scale, we additionally ran all models involving openness using an Openness latent variable that included all 12 items from the NEO-FFI. Because the NEO-FFI Openness scale is tilted toward openness rather than intellect to begin with, this latent variable should still be informative for our hypotheses. Direction and significance of all effects remained the same in these analyses, indicating that our measurement model was not unduly influencing results.

**Achenbach Self-report.** <sup>75</sup> To measure psychoticism, participants were administered 123 items from the Achenbach self-report (ASR), an instrument used to assess dimensions of psychopathology, which uses a three-point Likert scale. Items used in the current study include those corresponding to psychoticism<sup>74</sup>: “I hear sounds or voices that other people think aren’t there,” “I see things that other people think aren’t there,” “I do things that other people think are strange,” and “I have thoughts that other people would think are strange.”

Despite limited breadth, these items are thought to provide an adequate measure of psychotic-like experiences in the general population and have previously been linked to increased psychosis risk, altered functional connectivity, cannabis use, and suicidal ideation.<sup>76–81</sup> In the current data set, these ASR items predict family history of schizophrenia diagnosis ( $\beta = .15$ ,  $P = < .001$ ), social cognitive deficits, and behavioral metrics of apophenia—false positive cognitions and perceptions<sup>22</sup>—above variance in these constructs explained by IQ.

### *Intelligence Measures*

Intelligence measures were taken from two batteries: the NIH Toolbox<sup>82</sup> and Penn Computerized Neurocognitive Battery.<sup>83</sup> In a Picture Vocabulary test, participants selected which picture from multiple-choices most closely matched the meaning of a presented word. In a Matrix Reasoning test, participants completed a set of visual patterns (matrices) using one image from a set of multiple-choice options. For a List Sorting working memory test, participants were required to remember and sort a list of items presented verbally or visually.

### *Neuroimaging and Derivation of Networks*

Resting state fMRI data were collected using a 3T Siemens Skyra scanner. After preprocessing,<sup>84</sup> artifacts were removed using ICA+FIX.<sup>85,86</sup> Mean head movement was also calculated and included as a covariate, as this index can correlate with variables of interest.<sup>87,88</sup> Interparticipant registration of the cortex was carried out using areal feature-based alignment and the Multimodal Surface Matching algorithm.<sup>89,90</sup> Each data set was temporally demeaned and underwent variance normalization.<sup>91</sup> Data were entered into a group-PCA,<sup>91,92</sup> the results of which were fed into group-ICA using FSL’s MELODIC,<sup>91,93</sup> applying spatial-ICA at a dimensionality of 50.

After components were derived, we examined their association with canonical networks, by computing percentage of overlap with networks derived by Yeo,<sup>30</sup> focusing on the DN and FPCN. Of note, ICA components can overlap, whereas Yeo’s maps were nonoverlapping parcellations. This provides a more realistic depiction of brain organization, as many regions are involved in multiple networks.<sup>94</sup> Despite showing larger local extent due

to overlap, our components were smaller than Yeo's due to the higher dimensionality (50 vs. 7). Components with the highest overlap were visually inspected (further described in our supplement) to make sure they were centered on the correct networks. This yielded five FPCN and seven DN components (Figure 1).

#### Computation of Network Coherence Variables

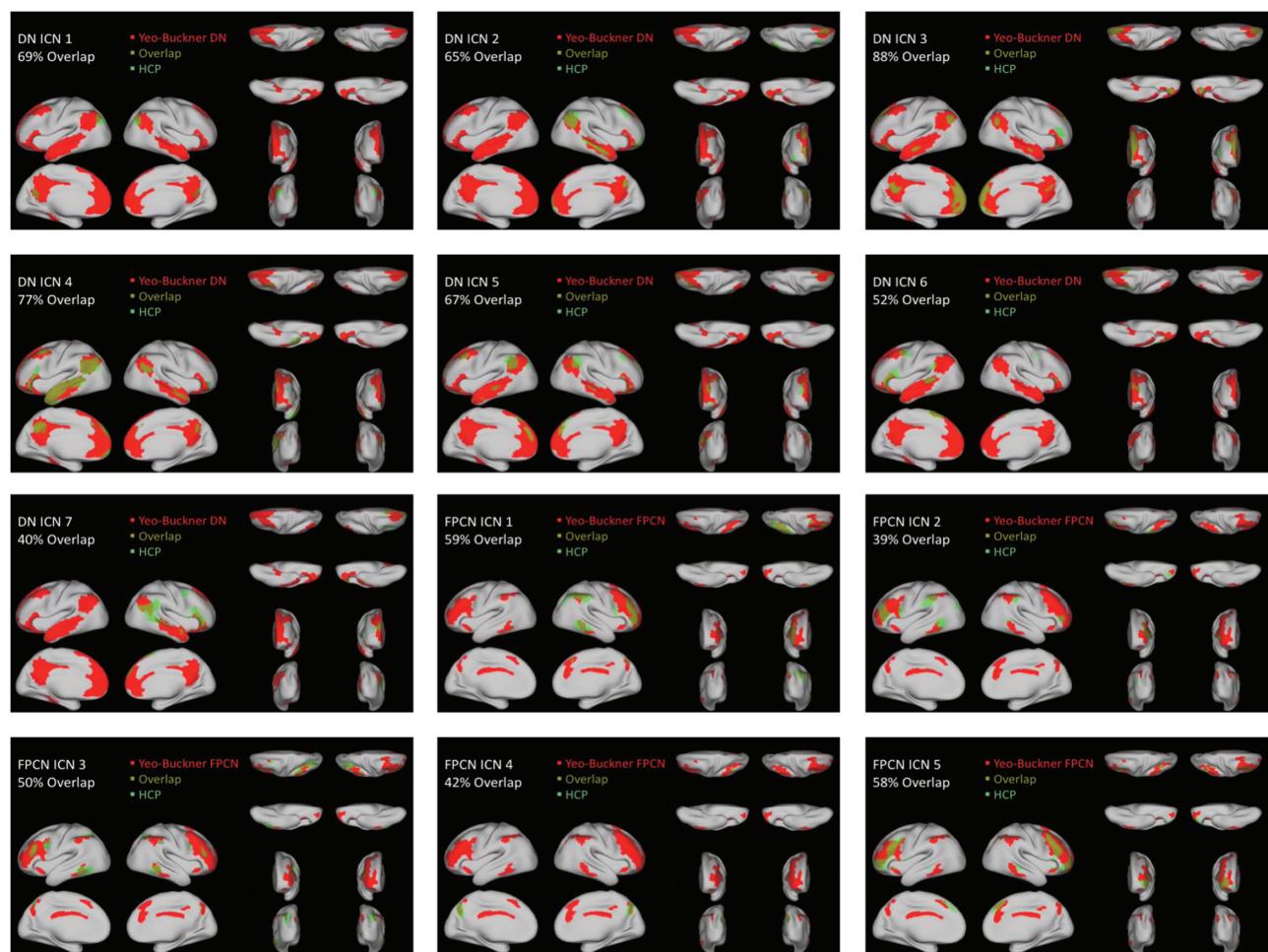
After identifying networks, we used two-stage dual regression to compute network coherence for each ICA component, for each participant.<sup>95,96</sup> Coherence quantifies connectivity within each ICA component and corresponds to the average correlation of each voxel within a network with the time series of that network. Previous studies have linked coherence to personality traits (both pathological and normal-range).<sup>97–99</sup>

First, node-time series were estimated using the standard “dual-regression stage-1” approach, in which the full set of ICA maps were used as spatial regressors against participants’ full time series data, estimating one time series per

ICA map.<sup>95</sup> Next, in stage-2 dual regression, participant-specific time series were used as temporal regressors onto each participant’s resting state data. This allowed us to derive a set of participant-specific spatial maps. Network coherence was then computed for each ICA component: group-level component maps (thresholded at  $z_{\max} > .30$ ) were binarized and applied as masks to each participant-level map,<sup>96</sup> and the mean correlation between each voxel within each participant-level spatial map and the mean time-series for all voxels in that component was calculated for each participant, for each component, giving coherence values for each of our 12 components. Finally, overall coherence scores for FPCN and DN were computed by averaging scores from corresponding components (further explained in our supplement).

#### Statistical Approach

Descriptive statistics were calculated for self-report measures and intelligence. Variables with skewness  $> 2.0$  (which included only the psychoticism items)



**Figure 1.** Visualizations of default and frontoparietal network components.

were logarithmically transformed. Structural equation modeling (SEM) was used to assess relations among latent factors. In each model, corresponding items or tasks were allowed to load onto latent variables for Psychoticism, Openness, and Intelligence. Due to the semantic similarity of two psychoticism items—"I do things that other people think are strange" and "I have thoughts that other people would think are strange"—and their high degree of correlation ( $r = .61, P < .001$ ), we made an a priori decision to allow their residuals to correlate, which significantly improved model fit ( $\Delta\chi^2 = 265.1, P < .001$ ).

SEMs with full information maximum likelihood estimation were used to test associations of Psychoticism, Openness, and Intelligence with DN and FPCN coherence. First, a model was fit to examine the relation between Openness and Psychoticism. For subsequent models, behavioral or self-report latent variables were used as criterion variables that were predicted by a set of observed variables: gender, age, head movement, DN coherence, and FPCN coherence. Predictors were allowed to correlate. Five models were created to examine effects of DN and FPCN coherence on (1) Psychoticism, (2) Openness, (3) shared variance of Openness and Psychoticism, and (4) Intelligence. For the first three models, a second iteration was fit with latent Intelligence as a covariate. Finally, scatter plots were used to visualize the relations of our Openness-psychoticism latent variable with DN and FPCN coherence (residualized by regression on coherence of the other network).

## Results

Descriptive statistics are presented in [Supplementary Table S1](#) and zero-order correlations are presented in [Supplementary Table S2](#). SEM fit indices are presented in [Table 1](#) and model results are presented in [Table 2](#). For all models, all manifest variables had significant loadings on corresponding latent variables. Latent Psychoticism was

significantly positively correlated with latent Openness ( $r = .61, P < .001$ ).

In our first neural model, Psychoticism was negatively related to FPCN coherence but positively related to DN coherence. Similarly, DN coherence positively predicted and FPCN coherence negatively predicted Openness. These associations remained significant when controlling for Intelligence. The same patterns of association appeared, with even stronger regression weights, when testing associations of coherence with shared variance of openness and psychoticism ([Figure 2](#)). Scatter plots showing relations of latent Openness-psychoticism to residualized coherence variables are presented in [Figure 3](#). Intelligence was positively associated with FPCN coherence but not associated with DN coherence. All regression paths from DN and FPCN coherence variables to criterion variables remained significant after estimation using robust (Huber-White) standard errors<sup>100</sup> and false-discovery-rate corrections.<sup>101</sup>

## Discussion

As hypothesized, openness, psychoticism, and their shared variance were associated with increased DN coherence and decreased FPCN coherence. Intelligence showed a positive relation to FPCN coherence but no relation to DN coherence. Controlling for intelligence did not eliminate significant associations of coherence with openness and psychoticism. These findings suggest that similar biological mechanisms may underlie psychosis-proneness across traditional risk indicators and associated normal-range personality traits. Findings are in line with schizophrenia research, as a number of studies have demonstrated increased DN activity and connectivity in psychosis patients,<sup>44</sup> as well as decreased FPCN function.<sup>51</sup> To the best of our knowledge, however, this is the first study to examine associations between network coherence, psychoticism, and openness in a large community sample.

Although our results are in line with patterns of functional connectivity reported in the existing literature, it

**Table 1.** Fit statistics for structural equation models

Models	$\chi^2$	P	RMSEA	95% C.I.	TLI	CFI
Model 1(Psychoticism and O)	71.2	< .001	.050	[.038, .062]	.960	.974
Model 2a (Psychoticism, DN, FPCN)	49.8	< .001	.048	[.035, .061]	.962	.983
Model 2b (Psychoticism, DN, FPCN, IQ)	131.5	< .001	.046	[.038, .055]	.948	.971
Model 3a (O, DN, FPCN)	75.5	< .001	.053	[.041, .066]	.955	.979
Model 3b (O, DN, FPCN, IQ)	244.5	< .001	.067	[.059, .075]	.898	.941
Model 4a (O-Psychoticism, DN, FPCN)	186.2	< .001	.046	[.039, .054]	.947	.964
Model 4b (O-Psychoticism, DN, FPCN, IQ)	384.7	< .001	.054	[.049, .060]	.907	.934
Model 5 (IQ, DN, FPCN)	55.7	< .001	.062	[.046, .078]	.943	.980

Notes: O = Openness, O-Psychoticism = Shared variance of Openness and Psychoticism, DN = Functional coherence of the default network, FPCN = Functional coherence of the frontoparietal control network.

**Table 2.** Structural equation model analyses—aggregate DN and FPCN coherence variables

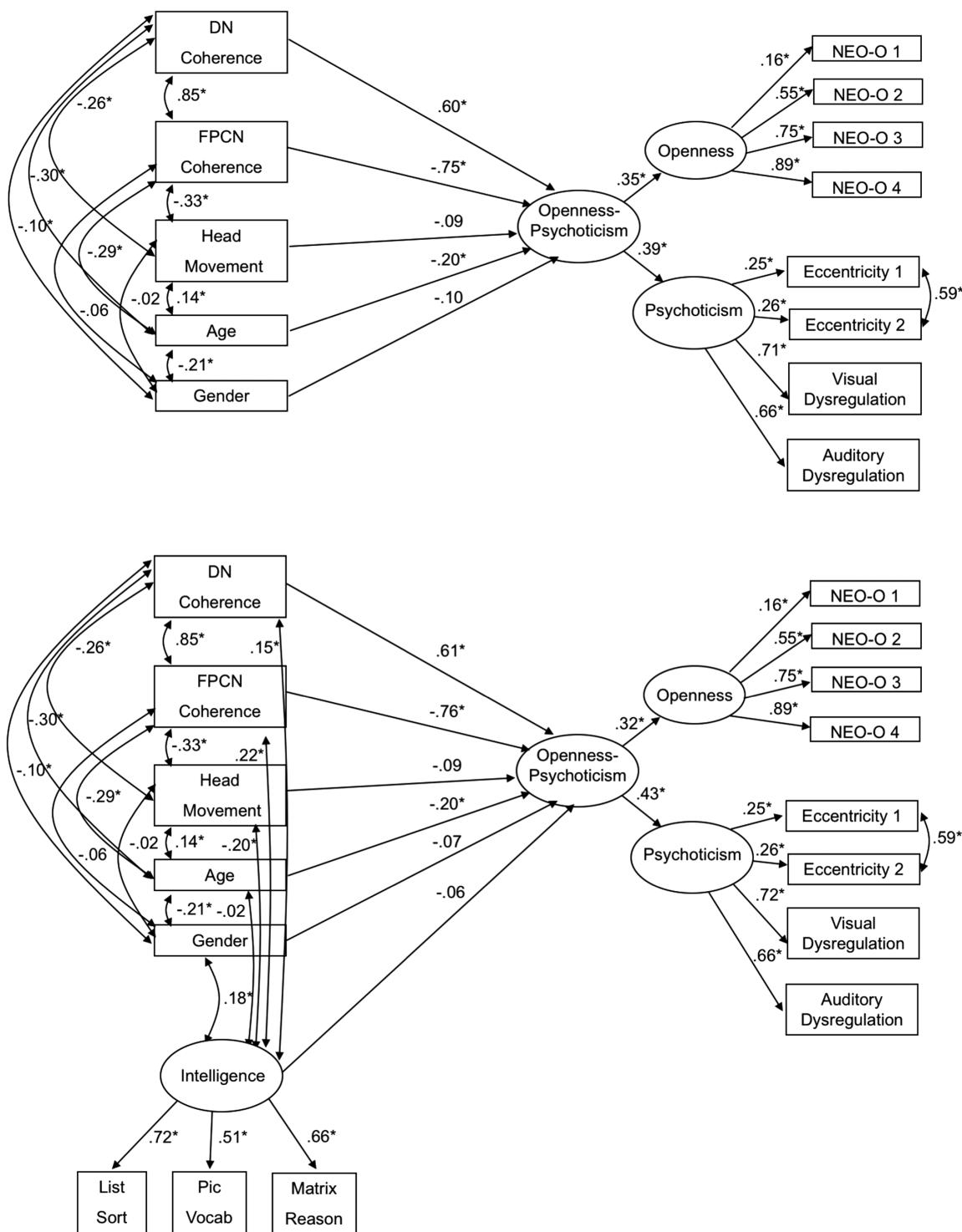
Models	Model 1			Model 2		
	<i>z</i>	$\beta$	<i>P</i>	<i>z</i>	$\beta$	<i>P</i>
<b>Psychoticism</b>						
Gender	0.4	.01	.715	0.9	.04	.385
Age	-2.3	-.09	.023	-2.1	-.08	.039
Head Movement	-0.4	-.02	.681	-0.8	-.04	.422
DN Coherence	2.8	.25	.006	2.2	.24	.031
FPCN Coherence	-3.6	-.32	< .001	-2.7	-.29	.007
Intelligence				-2.3	-.13	.021
<b>Openness</b>						
Gender	-2.2	-.08	.030	-2.5	-.10	.013
Age	-1.7	-.06	.089	-1.9	-.07	.053
Head Movement	-1.2	-.04	.245	-0.8	-.03	.415
DN Coherence	2.7	.21	.008	2.7	.21	.006
FPCN Coherence	-3.0	-.25	.003	-3.2	-.28	.001
Intelligence				1.9	.09	.056
<b>Psychoticism-Openness (Shared Variance)</b>						
Gender	-1.4	-.10	.176	-0.6	-.07	.578
Age	-2.3	-.20	.019	-2.3	-.20	.019
Head Movement	-1.1	-.09	.263	-1.0	-.09	.310
DN Coherence	3.0	.60	.003	2.4	.61	.015
FPCN Coherence	-3.3	-.75	.001	-2.8	-.76	.005
Intelligence				-0.3	-.06	.762
<b>Intelligence</b>						
Gender	5.7	.21	< .001			
Age	2.6	.10	.010			
Head Movement	-3.4	-.14	.001			
DN Coherence	-0.8	-.07	.410			
FPCN Coherence	3.7	.27	< .001			

is worth discussing them in the context of two preliminary studies using HCP data.<sup>76,79</sup> Using subsamples of 229 and 468 participants, respectively, one study found increased visual connectivity and decreased DN connectivity among participants endorsing psychoticism items,<sup>76</sup> and the other found psychoticism was negatively associated with global efficiency of the cingulo-opercular network and DN.<sup>79</sup> Our finding of positive association between DN and psychoticism may at first seem contradictory, but several factors may account for this discrepancy. One explanation is that these earlier studies did not control for variance shared among networks, which is likely artifactual.<sup>87</sup> Indeed, our results did not show a significant zero-order relation between DN coherence and psychoticism, but controlling for FPCN coherence revealed a significant positive association. This is an example of statistical suppression,<sup>102</sup> and it can be interpreted as meaning that if one examined individuals with equivalent levels of FPCN coherence, one would expect to find that individuals higher in Openness or psychoticism would have higher DN coherence. Furthermore, our use of a larger sample and latent variable modeling will have improved our statistical power, relative to those earlier studies.

Our findings suggest that psychoticism and openness reflect heightened coherence in the DN but lower levels

of FPCN coherence, possibly reflecting a tendency toward spontaneous self-generated thought coupled with reduced cognitive control. Such patterns are also reflected in studies showing that DN connectivity is positively related to positive symptoms, among patients with psychosis.<sup>43</sup> In terms of FPCN function, psychosis is not only linked specifically to dysfunction of prefrontal cortex, but it is also associated with broad cognitive deficits that have directly been tied to FPCN dysfunction.<sup>103</sup> Taken together, these neurocognitive associations with psychosis-proneness may suggest an increased default-network tendency toward erroneous thoughts and perceptions (false positives), coupled with diminished reality testing to screen out false positives. Future research including more comprehensive measurement of psychotic symptoms would be useful to confirm these speculations.

Importantly, the pattern of FPCN coherence that emerged for psychoticism and openness was also present, in the opposite direction, for intelligence. Given the prevalence of intelligence and working memory deficits in psychosis and its extended phenotype, it is not surprising similar patterns of connectivity would underlie these constructs. Mind wandering and related processes associated with openness, psychoticism, and the DN may directly compete with demands of intelligence and working

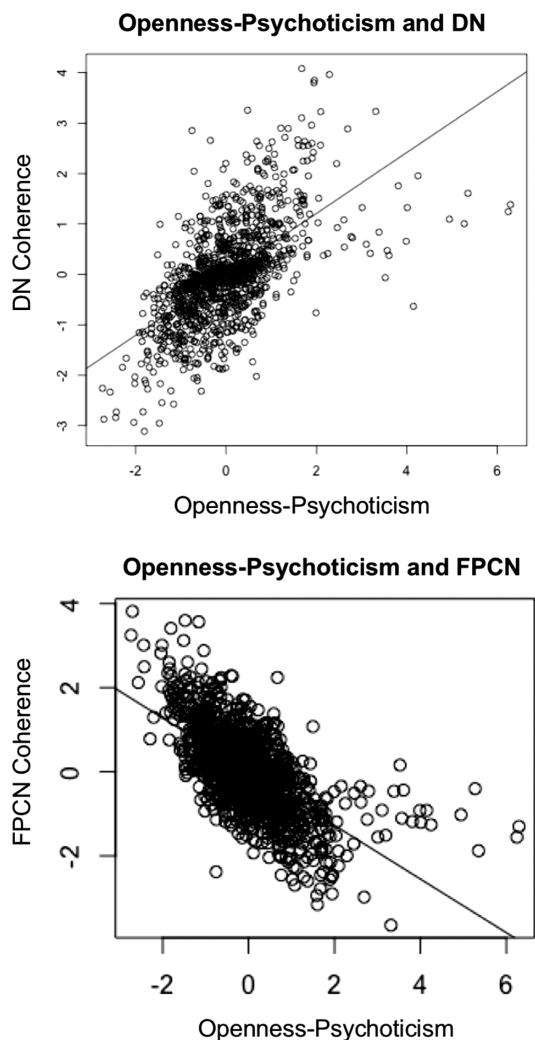


**Figure 2.** Structural equation models of Openness-psychoticism predicted by DN and FPCN coherence.

memory, leading to an inverse association with FPCN coherence. Indeed, research suggests that disrupted connectivity of the FPCN is associated with positive symptoms and working memory deficits among psychosis patients.<sup>104</sup> In short, impaired cognitive function and associated neural correlates may play an important role in

the broader mechanisms of psychosis and its extended phenotype.

The current study provides important novel insights into the measurement of neurobehavioral characteristics of the openness-psychoticism dimension, a key step in better characterizing possible mechanisms underlying



**Figure 3.** Scatter plots of Openness-psychoticism and residualized coherence variables.

risk factors for psychosis. Because psychotic symptoms are transdiagnostic features seen across a number of disorders, elucidating their neural mechanisms may eventually help facilitate more effective methods for assessment and treatment. Such an approach is in line with the National Institute of Mental Health's Research Domain Criteria initiative,<sup>105</sup> the Hierarchical Taxonomy of Psychopathology's conceptualization of psychiatric illness,<sup>11</sup> and a new theory that psychopathology is typically caused by extremity in normal personality mechanisms that interferes with goal-directed functioning.<sup>10</sup> Investigation of psychoticism, openness, and their relation to individual differences in functional brain networks and cognitive symptoms is a promising avenue for continued research into the etiology of risk for psychosis. Of particular relevance are theories positing psychosis and autism as diametrical disorders, involving divergent patterns of FPCN-DN coordination and associated cognitive processes.<sup>106–108</sup>

## Limitations

Although the current study had multiple strengths, some limitations are worth noting. First, although the Achenbach Self Report is a reliable, well-established measure, its items measuring psychoticism are limited. Use of more extensive measures of psychoticism—such as the Personality Inventory for DSM-5—could allow more robust testing of associations with neural variables. Second, our measures of openness and psychoticism were self-reported and could be usefully supplemented by clinician ratings or peer-reports. Finally, although the current study demonstrates links between neural and behavioral metrics in a population without severe mental illness, we cannot tell how well they would generalize to clinical populations. Further research should be undertaken to examine the roles of functional connectivity and cognitive deficits in those with active psychosis.

## Conclusion

Current findings suggest that psychoticism and openness are linked in part through their association with altered connectivity in neural networks associated with experiential simulation and cognitive control. It is increasingly recognized that risk for psychosis is distributed throughout the population dimensionally and that understanding subclinical indicators of risk in the general population is crucial to developing better models of psychosis etiology.<sup>3,4,109</sup> Assessing the coherence of intrinsic connectivity networks may provide a useful transdiagnostic approach to elucidating cognitive and neural mechanisms involved in psychosis and related phenomena. Our results advance understanding of the neural mechanisms of psychoticism that are shared with openness, a personality trait that appears to contribute specific risk for psychosis. This study adds to a growing body of research characterizing the underlying biology of transdiagnostic psychiatric features through the use of large, nonpatient samples and also underscores the continuity between normal personality variation and risk for psychopathology.

## Supplementary Material

Supplementary material is available at <https://academic.oup.com/schizophreniabulletin/>.

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