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The Latent Structure of Negative Symptoms in Individuals With Attenuated Psychosis Syndrome and Early Psychosis: Support for the 5 Consensus Domains

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Negative symptoms are prevalent in the prodromal and first-episode phases of psychosis and highly predictive of poor clinical outcomes (eg, liability for conversion and functioning). However, the latent structure of negative symptoms is unclear in the early phases of illness. Determining the latent structure of negative symptoms in early psychosis (EP) is of critical importance for early identification, prevention, and treatment efforts. In the current study, confirmatory factor analysis was used to evaluate latent structure in relation to 4 theoretically derived models: 1. a 1-factor model, 2. a 2-factor model with expression (EXP) and motivation and pleasure (MAP) factors, 3. a 5-factor model with separate factors for the 5 National Institute of Mental Health (NIMH) consensus development conference domains (blunted affect, alogia, anhedonia, avolition, and asociality), and 4. a hierarchical model with 2 second-order factors reflecting EXP and MAP, as well as 5 first-order factors reflecting the 5 consensus domains. Participants included 164 individuals at clinical high risk (CHR) who met the criteria for a prodromal syndrome and 377 EP patients who were rated on the Brief Negative Symptom Scale. *Results* indicated that the 1- and 2-factor models provided poor fit for the data. The 5-factor and hierarchical models provided excellent fit, with the 5-factor model outperforming the hierarchical model. These findings suggest that similar to the chronic phase of schizophrenia, the latent structure of negative symptom is best conceptualized in relation to the 5 consensus domains in the CHR and EP populations. Implications for early identification, prevention, and treatment are discussed.

Key words: anhedonia/avolition/asociality/blunted affect/alogia

Introduction

Given that few individuals achieve recovery after the onset of psychosis,[1–3] there has been increasing interest in early identification and prevention.[4] Psychotic disorders are typically preceded by a prodromal (ie, preillness) phase characterized by functional decline and subthreshold positive symptoms that progressively worsen over the course of several months to years.[4,5] It is now possible to reliably identify a group of clinical high-risk (CHR) youth who will go on to develop a psychotic disorder using state-of-the-art clinical interviews. Negative symptoms are one of the strongest predictors of conversion among CHR individuals, where they are highly prevalent, longitudinally stable, and one of the earliest symptoms to emerge within the prodromal phase.[6–8] Despite their clear relevance for early identification and treatment, relatively little progress has been made in studying the nature of negative symptoms among CHR individuals.

One critical, but understudied, facet of CHR phenomenology is the latent structure of negative symptoms. Latent structure refers to how the affective, motivational, and communicative behaviors that comprise negative symptoms relate to underlying factors, dimensions, or influences. Identifying the number of dimensions that underlie negative symptoms in CHR is critical for identifying the most relevant treatment targets and how to focus on early identification and monitoring evaluations. Factor analysis represents the most prominent method for identifying the number of latent dimensions subsumed within data. Conclusions about the number of latent dimensions that comprised negative symptoms based on factor analysis have undergone some evolution. Whereas early studies rightly concluded that negative

symptoms represent a separable dimension from positive and disorganized symptoms in schizophrenia and CHR populations, they erroneously suggested that negative symptoms reflect a single unitary construct. This errant conclusion resulted from factor analyzing negative symptom items along with positive, disorganized, and general psychiatric symptoms, causing negative symptom items to artificially aggregate together and appear unidimensional.[9–12]

In contrast, other studies have chosen to adopt a narrow bandwidth factor analytic approach, evaluating the structure of negative symptom items alone. This approach is less prone to systematic covariation caused by the inclusion of items from other constructs and problems with overfitting (ie, where item loading patterns are maximized for the original data but "fragile" and limited in their replicability in subsequent data sets). Studies that have favored this narrow bandwidth approach have tended to produce a 2-factor solution. Specifically, Azis et al[13] used exploratory factor analysis (EFA) to evaluate the structure of items from the negative symptom subscale of the Structured Interview for Prodromal Syndromes (SIPS).[14] Two factors emerged, reflecting emotion (items: expression of emotion, experience of emotion and self, and social anhedonia) and volition (items: avolition and occupational functioning).[13] Although informative that the construct is multidimensional, limitations associated with the SIPS negative symptom subscale (eg, construct validity, conflation of domains, and imprecision) make it unclear exactly how many domains exist and which domains those are. To accurately evaluate the latent structure of negative symptoms in CHR individuals, studies using more conceptually updated scales are needed. Chang et al[15] conducted an EFA of the Brief Negative Symptom Scale (BNSS),[16] a second-generation scale that is conceptually up-to-date and adapted for use in CHR.[17] Results supported a 2-factor structure, with dimensions reflecting motivation and pleasure (MAP: anhedonia, avolition, and asociality) and diminished expression (EXP: alogia and blunted affect). These results are similar to those obtained with EFA in people with schizophrenia.[18–22] However, it is unclear whether a 2-factor structure adequately captures the complexity of the construct in CHR individuals since evidence was derived from EFA alone. EFA is a data reduction technique that infers the presence of latent factors responsible for shared variance among a set of items, which does not specify an underlying structure or test competing models.[23] Confirmatory factor analysis (CFA) is needed to determine the latent structure of a construct because it allows for a direct comparison of competing theoretical models.

Recent CFA studies of schizophrenia indicate that the 1- and 2-factor (MAP and EXP) models provide poor fit for negative symptom data. However, 5-factor models reflecting the 5 NIMH consensus domains (anhedonia, avolition, asociality, alogia, and blunted affect) and a hierarchical model (with 2 second-order factors reflecting MAP and EXP and 5 first-order factors reflecting the 5 consensus domains) provided excellent fit, with the 5-factor model slightly outperforming the hierarchical model.[24,25] The 5-factor structure has been replicated across the 3 most contemporary negative symptom scales (BNSS, Clinical Assessment Interview for Negative Symptoms [CAINS], and Scale for the Assessment of Negative Symptoms [SANS]), across multiple cultures and languages,[26–28] and using alternate mathematical techniques (eg, network analysis).[29] These findings suggest that, in chronic schizophrenia, the 5-factor structure that has been repeatedly observed is not scale specific, unique to one culture, or the byproduct of a specific analytic strategy.[25] However, it is unclear whether the 5-factor structure also best represents the latent structure of negative symptoms in CHR individuals because CFA has yet to be completed on a second-generation rating scale that is conceptually up-to-date.

It is also unclear whether the 5-factor structure applies to those in the earliest stages of full psychotic illness. In early psychosis (EP; ie, first 5 years after illness onset), negative symptoms are highly prevalent[30,31] and a strong predictor of functional outcome and long-term clinical prognosis.[32–37] To our knowledge, only 2 studies have evaluated the structure of negative symptoms in an EP sample. Pelizza et al[38] used EFA to evaluate the structure of negative symptom items on the Positive and Negative Syndrome Scale (PANSS), finding evidence for 2 factors reflecting diminished experience and expression. However, these results are difficult to interpret because EFA was used and the PANSS items are based on outdated conceptualizations and have limited construct validity.[39] Lyne et al[40] used CFA to evaluate factors on the SANS[41] and found support for 3 factors reflecting experience, expression, and alogia/inattention. However, these findings are also difficult to interpret because the analyses included items that are not part of the modern negative symptom construct (eg, inattention subscale items and poverty of content of speech).[42] Thus, unambiguous conclusions about the structure of negative symptoms cannot be drawn in an EP sample.

The current study used CFA to evaluate competing hypotheses regarding the latent structure of negative symptoms in multiple samples of participants at CHR (ie, individuals meeting criteria for a prodromal syndrome) or in the early stages of full psychotic illness (EP; ie, ≤5 years since the first presentation of the first episode for treatment). Four theoretically derived models were evaluated based on recent CFA studies of negative symptoms in schizophrenia: 1. a 1-factor model; 2. a 2-factor model with EXP and MAP factors; 3. a 5-factor model with separate factors for the 5 NIMH consensus development conference domains (blunted affect, alogia, anhedonia, avolition, and asociality); and 4. a hierarchical model with 2 second-order factors reflecting EXP and

MAP, as well as 5 first-order factors reflecting the 5 consensus domains. Similar to past findings in chronic schizophrenia,[24–28] we hypothesized that in both the CHR and EP samples: 1. the 1- and 2-factor models would offer mediocre fit to the BNSS data, 2. 5-factor and hierarchical models would offer excellent fit, with an edge to the 5-factor model; and 3. the preferred factor solution would show evidence of equivalence across CHR and EP samples.

Method

Participants

Data for the current investigation was drawn from 4 studies that administered the BNSS to assess negative symptoms in participants with EP or those at CHR in Hong Kong and the United States. These studies were approved by the local institutional review boards, and all participants provided written informed consent. For those aged under 18 years, parental consent and participant assent were also obtained. Demographics and symptom characteristics are summarized in table 1 for the EP and CHR samples.

The CHR sample consisted of 164 total participants recruited in Hong Kong and the United States. The Hong Kong CHR group consisted of 110 help-seeking CHR participants[15] aged 15–40 years who were recruited from the EASY clinic at Queen Mary Hospital, a university-affiliated hospital and a major clinical center conducting CHR research in Hong Kong. Individuals with no past history of psychotic disorder and who fulfilled one or more of the following criteria assessed by Comprehensive Assessment of At-Risk Mental State (CAARMS)[46] were confirmed as having CHR status: 1. attenuated psychotic symptoms (APS), 2. brief limited intermittent psychotic symptoms (BLIPS), and 3. state- and trait-risk factors (vulnerability group; ie, either having a schizotypal personality disorder or family history of psychosis in a first-degree relative and recent significant functional decline). Individuals with intellectual disability, neurological disorder, a history of head injury, or current substance dependence were excluded. American CHR participants (n = 54; aged 12–27) were help-seeking individuals recruited from 2 psychosis risk evaluation programs directed by the corresponding author (G.P.S.) for studies examining negative symptom mechanisms.[47,48] These programs received referrals from local clinicians (eg, psychiatrists, psychologists, social workers, and school psychiatrists) to perform diagnostic assessment and monitoring evaluations for youth displaying psychotic experiences. Additional recruitment methods included online and print advertisements, in-person presentations to community mental health centers, and calls or in-person meetings with members of the local school system (eg, superintendent and principals). All CHR youth met criteria for a prodromal syndrome on the SIPS: 1. attenuated positive symptoms (n = 50); 2. genetic risk and deterioration syndrome (n = 3); 3. brief intermittent psychosis syndrome (n = 1). BNSS psychometric data was reported on a subset of these participants in Strauss and Chapman.[17]

Procedures

At each site, the BNSS was administered as part of larger protocols. In the CHR samples, BNSS probes were adapted to ask about social media and electronic social interactions (eg, texting and Facebook), living situations specific to youth (eg, living with parents and in dorms

Table 1. Demographic and clinical characteristics

Variable	CHR combined sample (n = 164)		Early psychosis sample (n = 377)	
	M	SD	*M*	SD
Age	20.6	5.7	37.2	9.7
Education	12.0	2.5	11.3	3.4
Male (%)	41%	--	43.5%	--
BNSS scores				
Total score	17.0	11.6	16.2	15.5
MAP average	1.94	1.3	1.61	1.4
EXP average	0.62	1.0	0.86	1.4
Anhedonia average	2.02	1.6	1.76	1.7
Asociality average	1.82	1.5	1.63	1.7
Avolition average	1.98	1.6	1.45	1.7
Blunted affect average	0.72	1.2	0.87	1.4
Alogia average	0.52	1.1	0.86	1.5

Note: CHR, clinical high risk; BNSS, Brief Negative Symptom Scale; MAP, motivation and pleasure factor; EXP, diminished expression factor.

with roommates), and recreational activities common in youth (eg, video games). Item anchors were not modified.[17] Raters at each site were trained to minimum reliability standards (interrater agreement >0.80 with gold-standard training tapes) prior to performing study procedures. Rater training consisted of an in-depth review of the manual for each measure, as well as procedures for rating the instrument. Raters watched and rated a series of initial videos that were developed either by the BNSS authors or internally by the research team. Ratings were then discussed as a group using gold-standard rationales and interviewers were instructed in interview technique. Interviewers subsequently received ongoing supervision and participated in regular (~monthly) gold-standard reliability meetings to maintain quality assurance. All raters had a bachelor's degree or higher and 1 or more years of clinical experience.

Data Analysis

First, single-sample CFAs were fitted to estimate each of the 4 alternate factor models in both the CHR and EP samples. Next, multigroup CFAs were used to test the invariance of the best fitting factor structure obtained from the single-sample CFAs. All CFA models were fitted and estimated using M*plus* Version 5.0. Similar to prior schizophrenia studies,[24–28] the Lack of Normal Distress item was not included in the CFA models because the distress item was not part of the agreed-upon NIMH consensus conference domains, and prior EFA studies reported low commonalities for this item.[19] The weighted least-squared estimator with SEs and mean- and variance-adjusted chi-square test that use a full weight matrix (WLSMV) and the maximum likelihood with robust SEs (MLR) served as estimators (the WLSMV is the program default) for single-sample CFAs. Both estimators are recommended for ordered-categorical variables and have been shown to produce accurate parameter estimates and SEs with nonnormal response distributions[49,50]. MLR model estimation was conducted with Monte Carlo-based numerical integration algorithm to designate the number of integration points. Only the WLSMV estimator was used in the estimation of multigroup factor models. The following goodness-of-fit statistics were examined to evaluate the absolute fit of each factor model to the CHR and EP data: the model chi square (χ^2), the comparative fit index (CFI), Tucker–Lewis index (TLI), root mean square error of approximation (RMSEA), and the weighted root mean-squared residual (WRMR). The Akaike's information criterion (AIC), Bayesian information criteria (BIC), and the sample-size-adjusted BIC were used to compare the relative fits of alternate factor models to the same data.

The model χ^2 is a measure of the discrepancy between the restricted covariance matrix implied by the factor model being tested and the unrestricted sample covariance matrix (ie, a model in which variables correlate freely). The CFI and TLI are incremental fit indices that measure the amount of improvement in model fit of the hypothesized factor model compared with a less restricted but nested baseline model. CFI values are normed to range from 0 to 1. TLI values (nonnormed) can exceed 1 but with both, numbers closer to 1 indicate good-fitting models. The WRMR is a residual-based index of the discrepancy between the sample variance–covariance matrix and hypothesized variance–covariance matrix implied by the fitted factor model. Unlike the CFI and TLI, which rely on an evaluation of improvement over a baseline independence model, the RMSEA is an absolute index of fit that measures the discrepancy between the hypothesized factor model and the sample data. It does this by examining how well the fitted model with optimally chosen parameter values fits the population covariance matrix. The information criteria—AIC, BIC, and adjusted BIC— are relative fit indices used for comparing 2 or more alternate nonnested models. Information criteria indices consider the fit of the hypothesized model, as well as its complexity, each imposing degrees of penalties for parameters estimated in relation to sample size. Evidence of model fit was determined according to standard interpretations of the fit indices but because the χ^2 tends to falsely reject adequate statistical model fit with large sample sizes, descriptive goodness-of-fit statistics formed the bases for evaluating each model. These include by convention a CFI/TLI value ≥0.95, and an RMSEA ≤0.08. In addition, WRMR values of 1.00 and lower suggest strong fits to data. The information criteria allow for comparisons between nonnested models with lower values indicating better model fit, so the model with the lowest values is the preferred model.[51–56]

Multigroup CFA served to test the equivalence of the preferred factor structure between EP and CHR phases of illness. The measurement invariance analysis involved iterative fitting of configural, metric, scalar, and residual invariance models to the combined CHR and EP data. Fitting these models involves the sequential imposition of equality constraints—including factor structure, loadings, intercepts, and residual variances—across the 2 groups. At each step, the more constrained model is nested within the previous model and its fit is evaluated in relation to the previous model using chi-squared difference testing. Changes in chi square ($\Delta\chi^2$), CFI (ΔCFI), TLI (ΔTLI), and RMSEA (ΔRMSEA) from the previous step served to evaluate the measurement invariance at the current step. Traditionally nonsignificant $\Delta\chi^2$ suggests that constraints imposed on the model are tenable. Given that sample size affects $\Delta\chi^2$, the analysis used other fit indices, including ΔCFI, which has the most empirical support and ΔRMSEA[57–59]. ΔCFI values not exceeding −0.01 provide evidence of measurement invariance. In

addition, a ΔRMSEA value of that not exceeding 0.015 similarly suggests invariance.

The invariance analysis examined all levels of measurement invariance across the 2 groups beginning by first evaluating a configural model. Configural invariance examines whether the factor structure—ie, item loading patterns (not the size of the loadings) are similar in the CHR and EP samples. The analysis tests metric invariance if the previous step establishes configural invariance. Metric invariance examines whether constraining the factor loadings to be equal across the CHR and EP samples is tenable. If metric invariance is established, the next step imposes additional equality constraints. Scalar invariance evaluates the tenability of constraining factor loadings and intercepts to be equal across the CHR and EP samples. Finally, if scalar equivalence is established, the final step imposes equality constraints on residual variances across both samples to test residual invariance.

Results

Results of the CFAs for EP sample are presented in tables 2 and 3, respectively. Both the 1- and 2-factor models proved to be suboptimal fits to the CHR negative symptoms data producing CFI, TLI, RMSEA, and WRMR estimates that fell outside of the acceptable range of values. The 1-factor model also proved to be poor fit for the data in the EP sample with CFI, TLI, RMSEA, and WRMR values that similarly fell short of acceptable thresholds. Although the 2-factor model produced CFIs and TLIs that exceeded the 0.95 threshold in the EP sample, both the RMSEA and the WRMR were too high for this model to be considered good fit for the data. In contrast, the CFI, TLI, RMSEA, and WRMR values obtained for the 5-factor model proved this model to be excellent fit for the CHR and EP sample. The hierarchical model similarly showed good fit for the data producing fit values that met acceptable thresholds in both samples, save its RMSEA in the CHR, which just slightly exceeded the acceptable threshold at 0.082.

Examination of the information criteria fit indices showed that the 5-factor model and the hierarchical model outperformed the unidimensional and 2-factor models. Across evaluated models, the 5-factor model produced the best (lowest) information criteria values in both the CHR and EP data.

Multigroup CFA comparing the structure of negative symptoms in the combined CHR and EP sample suggested invariance across illness phases (see table 4). Specifically, fit values from the configural model showed that the 5-factor model held across EP and CHR samples with CFI and TLI scores that exceed 0.99 and RMSEA close to the 0.08 threshold. The factor loadings were statistically significant ($P < .001$) for all items suggesting that the 5-factor structure fits well in both samples. Metric invariance model (ie, equivalence of factor loadings) similarly showed good fit with CFI and TLI that exceed 0.99 and RMSEA close to the threshold (0.09). Changes in fit

Table 2. Goodness-of-fit statistics for confirmatory factor models fitted on early psychosis sample

	Chi square	CFI/TLI	RMSEA	WRMR	Log likelihood	*k*	AIC	BIC	SSA-BIC
Model tested									
1 factor	$X^2(9) = 457.25$, $P < .001$	0.945/0.954	0.363	3.657	−4947.07	83	10 060.13	10 386.51	10 123.17
2 factor	$X^2(12) = 278.97$, $P < .001$	0.969/0.980	0.243	2.267	−4609.21	84	9386.41	9716.72	9450.21
5 factor	**$X^2(18) = 47.27$, $P < .001$**	**0.997/0.999**	**0.066**	**0.417**	**−4364.50**	**93**	**8915.01**	**9280.71**	**8985.64**
Hierarchical 5 factor	$X^2(17) = 47.98$, $P < .001$	0.996/0.998	0.070	0.608	−4507.08	85	9184.16	9518.40	9248.72

Note: The preferred models are presented in bold font. Chi square for the CFA baseline model in the sample: $X^2(8) = 8735.92$, $P < .0001$. CFI, confirmatory fit index; TLI, Tucker–Lewis index; RMSEA, root mean square error of approximation; WRMR, weighted root mean-squared residual; *k*, number of free parameters; AIC, Akaike information criterion; BIC, Bayesian information criterion. SSA-BIC, Sample Size Adjusted BIC.

Table 3. Goodness-of-fit statistics for confirmatory factor models fitted on clinical high-risk sample

	Chi square	CFI/TLI	RMSEA	WRMR	Log likelihood	*k*	AIC	BIC	SSA-BIC
Model tested									
1 factor	$X^2(10) = 304.92$, $P < .001$	0.839/0.823	0.425	2.950	−2364.04	78	4884.08	5125.39	4878.45
2 factor	$X^2(12) = 124.67$, $P < .001$	0.938/0.944	0.240	1.583	−2184.34	79	4526.67	4771.08	4520.97
5 factor	**$X^2(15) = 24.95$, $P = .051$**	**0.995/0.996**	**0.064**	**0.426**	**−2072.59**	**88**	**4321.18**	**4593.43**	**4314.84**
Hierarchical 5 factor	$X^2(11) = 22.97$, $P = .018$	0.993/0.993	0.082	0.646	−2093.88	80	4347.75	4595.25	4341.98

Note: The preferred models are presented in bold font. Chi square for the CFA baseline model in the sample: $X^2(11) = 1840.59$, $P < .0001$. Abbreviations are explained in the second footnote to table 2.

indices between the configural and metric models were not significant with the ΔCFI, ΔTLI, and ΔRMSEA falling below the cutoff values. The scalar (ie, equivalence of factor loading and intercepts) and residual (ie, equivalence of loadings, intercepts, and factor residual variances) invariance models also showed good fit for the data. Scalar and residual invariance were supported ΔCFI, ΔTLI values less than 0.01 and ΔRMSEA less than 0.015 relative to the configural and metric models.

Discussion

CFA was used to evaluate the latent structure of negative symptoms using data from the BNSS in CHR and EP participants. Results indicated that the 1- and 2-factor models provided suboptimal fit for the data. In contrast, 5-factor and hierarchical models provided excellent fit, with the 5-factor model outperforming the hierarchical and being more parsimonious. Models comparing the structure of the EP and CHR samples also supported invariance. These findings are consistent with several recent CFAs examining the latent structure of negative symptoms in adults with chronic schizophrenia, which also found that 1- and 2-factor models offered suboptimal fit and the 5-factor and hierarchical models were excellent.[24–29] Collectively, these results suggest that, across phases of psychosis illness, the 5-factor model represents the optimal conceptualization of negative symptoms.[25]

These findings have important clinical implications. First, the DSM-5 identifies an attenuated psychosis risk syndrome. Negative symptoms are not considered in this diagnosis. However, it may be beneficial to do so in future iterations, given that they are known to be highly prevalent, an important predictor of conversion, and produce a similar factor structure to overt psychosis.[60,61] Like chronic schizophrenia, our results suggest that the description and evaluation of negative symptoms should focus on 5 domains in the attenuated psychosis risk syndrome diagnosis and early episode psychosis. Second, these 5 domains reflect distinct constructs that should be evaluated separately for the purposes of early identification and monitoring. The most commonly employed CHR assessments (CAARMS, SIPS, and Schizophrenia Proneness Instrument (SPI)), do not measure negative symptoms according to modern conceptualizations (see review[62]). Their negative symptom items are impacted by conceptual confusion, imprecision, domain conflation, and inclusion of items unrelated to the construct. These scales do not measure the 5 consensus domains and may be less effective for early identification and monitoring than more conceptually updated scales. We recommend that clinical evaluations utilize newer measures like the BNSS, CAINS,[20] and Prodromal Inventory for Negative Symptoms[63] since they are the only ones capable of identifying the 5 domains at present. Third, studies are needed to explore the pathophysiological mechanisms of these 5 domains in CHR and EP participants. If differential mechanisms are identified for the 5 domains, industry and the Food and Drug Administration (FDA) would have a compelling reason to develop targeted treatments for individual domains rather than the broader negative symptom construct. The 2 dimension conceptualization may mask meaningful variance accounted for in processes underlying the individual domains that make up MAP and EXP. Clinical trials should consider using the 5 domains as distinct outcome measures given that they reflect 5 separate constructs and because continuing to evaluate efficacy in relation to 1 or 2 dimensions is likely to miss more granular changes that could occur in relation to the 5 separate domains.[25] This approach may require new conceptual models to be developed for each of the 5 domains, which might best be accomplished via a combination of animal and human neuroscience approaches with translational capability. Additionally, it may be necessary to revise standards for identifying mechanistic targets and clinical trial design procedures (eg, inclusion criteria that are domain specific based on taxometrically defined cutoffs that have yet to be established, accounting for confounding factors unique to each domain).

Certain limitations should be considered. First, our study combined data from moderately sized samples collected in the United States and Hong Kong. However, we did not have adequate power to formally test the invariance of factor structure across the sites. Although cultural differences have not been found in past CFA studies

Table 4. Goodness-of-fit estimates for measurement invariance testing across clinical high-risk and early psychosis samples

Invariance model tested	Chi square X^2 (df)	Chi-square difference test X^2(df)	CFI	CFI change	TLI	TLI change	RMSEA	RMSE change
Invariance threshold				**≤−0.010**		**≤−0.010**		**≤0.015**
Configural	$X^2(31) = 97.87$, $P < .001$	—	0.994	—	0.996	—	0.089	—
Metric	$X^2(37) = 118.49$, $P < .001$	$X^2(12) = 39.89$, $P < .001$	0.993	0.001	0.996	0.000	0.090	−0.001
Scalar	$X^2(54) = 154.56$, $P < .001$	$X^2(28) = 67.13$, $P < .001$	0.991	0.002	0.997	−0.001	0.083	0.007
Residual	$X^2(49) = 125.54$, $P < .001$	$X^2(10) = 50.91$, $P < .001$	0.993	−0.002	0.997	0.000	0.076	0.007

Note: Chi square for the CFA baseline model in the sample: $X^2(19) = 11\,711.45$, $P < .0001$. Abbreviations are explained in the second footnote to table 2.

on chronic schizophrenia,[26–28] it will be important to test cultural invariance in EP and CHR samples in the future. Second, only cross-sectional data were utilized, and it is unclear whether factor structure differs between CHR converters and nonconverters. Additional longitudinal studies are underway at each site to address this question. Third, sources of secondary negative symptoms (eg, anxiety and depression) are prevalent in the CHR population,[64] and it is unclear whether these aspects of psychopathology have an impact on factor structure in CHR individuals. We suspect that such an impact is minimal given that CHR participant results were very similar to schizophrenia patients with fewer secondary negative symptoms.[24–28] Fourth, we were unable to examine pathophysiological mechanisms associated with the 5 domains; however, this is an ongoing goal of our research groups.

Collectively, these findings suggest that the latent structure of negative symptoms is best conceptualized in relation to the 5 domains identified in the 2005 NIMH Consensus Development Conference (anhedonia, avolition, asociality, alogia, and blunted affect) in CHR and EP participants similar to chronic schizophrenia. If distinct clinical and pathophysiological correlates of these 5 domains are identified in future research, there will be important implications for early identification, prevention, and treatment in early phases of psychosis.

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