

Factor Structure and Construct Validity of the Scale for the Assessment of Negative Symptoms

Steven L. Sayers
Allegheny University of the Health Sciences

Patrick J. Curran
University of California, Los Angeles

Kim T. Mueser
Dartmouth Medical School

Confirmatory factor analysis (CFA) was used to examine the underlying structure of the negative symptoms of schizophrenia as measured by the Scale for the Assessment of Negative Symptoms (SANS). Schizophrenia patients ($N = 457$) were assessed with the SANS on at least 1 of 2 occasions: (a) 2–4 weeks after an index hospitalization, and (b) after a clinical stabilization period that lasted 3–6 months. Results of an exploratory factor analysis conducted for the first assessment ($n = 401$) were largely supported by the CFAs conducted on the data at the second assessment ($n = 345$). The CFA solution included 3 factors: Diminished Expression, Inattention–Alogia, and Social Amotivation. Analysis of patients' clinical characteristics, treatment outcome, chronicity of the illness, premorbid history, and social adjustment supported the validity of the 3 factors.

The “negative symptoms” of schizophrenia, such as blunted affect or asociality, have become the focus of increased inquiry in the last one and a half decades. Several theoretical and methodological developments have spurred this research, most nota-

bly the speculations that schizophrenia entails two separate underlying etiological processes corresponding to negative and positive symptoms (Andreasen, Flaum, Swayze, Tyrrell, & Arndt, 1990; Crow, 1980, 1985; Strauss, Carpenter, & Bartko, 1974). Research in this area has also been facilitated by the development of instruments to assess negative symptoms (e.g., Andreasen, 1982) and by proposals for subtyping patients based on either the predominance of positive–negative symptoms (Andreasen, 1982, 1985) or the presence of long-term “primary” negative symptoms (Carpenter, Heinrichs, & Wagman, 1988).

There are several reasons why negative symptoms have been the focus of intense research in recent years. First, negative symptoms tend to be more stable over time than positive symptoms (McGlashan & Fenton, 1992; Mueser, Douglas, Bellack, & Morrison, 1991), which suggests that they are more fundamental to schizophrenia. Second, negative symptoms are associated with both poor premorbid social functioning and are predictive of worse outcome in schizophrenia (Mueser, Bellack, Morrison, & Wixted, 1990; Pogue-Geile, 1989; Pogue-Geile & Zubin, 1988). Third, there is an accumulation of evidence showing that negative symptoms tend to be related to neurocognitive deficits and structural brain anomalies (Andreasen, Roy, & Flaum, 1995). Recognition of the importance of negative symptoms to schizophrenia is reflected by the fact that these symptoms are now included as part of the diagnostic criteria of the most recent (fourth) edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)* (American Psychiatric Association, 1994). It is not clear, however, whether negative symptoms are associated with patients' premorbid functioning and clinical outcome as a unitary construct or whether only specific types of negative symptoms have these associations. For instance, symptoms such as social amotivation and social anhedonia may be linked to poor premorbid functioning, whereas affective flattening or blunting may be linked to poor

Steven L. Sayers, Department of Psychiatry, Allegheny University of the Health Sciences; Patrick J. Curran, Department of Psychology, University of California, Los Angeles; Kim T. Mueser, Department of Psychiatry and Department of Community and Family Medicine, Dartmouth Medical School.

The Treatment Strategies in Schizophrenia (TSS) Cooperative Agreement Program is a multicenter clinical trial carried out by five research teams in collaboration with the Division of Clinical Research of the National Institute of Mental Health (NIMH), Rockville, Maryland. The NIMH principal collaborators are Nina R. Schooler, Samuel J. Keith, Joanne B. Severe, and Susan M. Matthews. The principal investigators and co-principal investigators at the five sites are as follows: Albert Einstein School of Medicine and Hillside Hospital–Long Island Jewish Medical Center, Glen Oaks, New York (UO1 MH39992)—John M. Kane, Jeffrey A. Lieberman, and Margaret Woerner; Medical College of Pennsylvania and Eastern Pennsylvania Psychiatric Institute, Philadelphia, Pennsylvania (UO1 MH39998)—Alan S. Bellack and George M. Simpson; Cornell University Medical College and Payne Whitney Clinic, New York, New York (UO1 MH40007)—Ira D. Glick and Allen J. Frances; University of California, San Francisco and San Francisco General Hospital (UO1 MH40042)—William A. Hargreaves and Marc Jacobs; Emory University and Grady Memorial Hospital, Atlanta, Georgia (UO1 MH40597)—Philip T. Ninan and Rosalind M. Mance. All prolixin decanoate and prolixin hydrochloride were provided courtesy of Bristol-Meyers Squibb.

We thank Jack Blanchard and Nina R. Schooler for their comments on an earlier version of this article.

Correspondence concerning this article should be addressed to Steven L. Sayers, Department of Psychiatry, Eastern Pennsylvania Psychiatric Institute, Allegheny University of the Health Sciences, 3200 Henry Avenue, Philadelphia, Pennsylvania 19129. Electronic mail may be sent via Internet to sayers@medcolpa.edu.

clinical outcome. There may be different neurocognitive or structural brain anomalies associated with these different types of symptoms.

The first question that has arisen in this area of research has been whether there is empirical support for the construct of negative symptoms. Existing research has been supportive of the negative symptom construct as a cluster of symptoms that are separate from positive symptoms (e.g., Lenzenweger, Dworkin, & Wethington, 1991; Schuldberg, Quinlan, Morgenstern, & Glazer, 1990). In addition, the current evidence argues against a bipolar continuum of positive versus negative symptoms (cf. Andreasen & Olsen, 1982), given that positive and negative symptoms are generally positively correlated (Czobor, Bitter, & Volavka, 1991). Although these studies demonstrate that negative and positive symptoms can be distinguished from each other, they also show that there is limited consensus regarding which symptoms should be considered as part of the negative symptom cluster. For example, some studies of negative symptoms suggest that attentional impairment correlates only at a moderately low level with most of the other negative symptoms (Lenzenweger et al., 1991; Walker, Harvey, & Perlman, 1988) and does not tend to load on the same factors as the other negative symptoms (Schuldberg et al., 1990). Furthermore, the dimensionality of negative symptoms has been explored in few studies. Thus, a great deal of inquiry is still needed regarding negative symptoms of schizophrenia. Clearly, researchers need reliable and valid measures that accurately reflect the underlying structure and composition of negative symptoms.

One of the most widely used measures of negative symptoms is the Scale for the Assessment of Negative Symptoms (SANS; Andreasen, 1982). Despite its development over a decade ago, few large-sample studies have examined the psychometric properties of the SANS using multiple sites to address issues of uniformity of the use of the scale (see Mueser, Sayers, Schooler, Mance, & Haas, 1994). Indeed, there have been few studies of the structure of the SANS, and the existing studies have not adequately tested the viability of the five rationally derived factors of the measure: Affective Flattening or Blunting, Alogia, Avolition–Apathy, Anhedonia–Asociality, and Inattention. Most studies of dimensionality have included positive symptoms in the analysis of the negative symptoms in order to examine the constituents of, and the relationship between, positive and negative symptoms (Lenzenweger et al., 1991; Liddle, 1987; Miller, Arndt, & Andreasen, 1993; Schuldberg et al., 1990; Walker et al., 1988). Generally, these studies either have resulted in support for negative symptoms as a unitary construct or, as noted above, have led to questions about the inclusion of symptoms such as attentional impairment and inappropriate affect under the construct of negative symptoms.

An exception to the studies cited above is that of Keefe et al. (1992). They used confirmatory factor analysis (CFA) to test the structure of negative symptoms in a sample of 130 hospitalized schizophrenia patients rated with the SANS. Keefe et al. (1992) found that a three-factor model fit the structure of the SANS best: The first factor was called Diminished Expression (e.g., unchanging facial expression, affective nonresponsivity), the second factor was termed Social Amotivation (e.g., inability to feel intimacy), and the third factor, Disorganization, included only two items—"inappropriate affect" and "poverty of

content of speech." There are two problems with this third factor. First, two-item factors are highly unstable (Boomsma, 1982). Second, as noted above, many questions have been raised about whether one of the items, "inappropriate affect" should be considered a negative symptom (Miller, Arndt, & Andreasen, 1993), and this item has been dropped from recent versions of the SANS (e.g., Mueser et al., 1994). Another limitation of Keefe et al.'s analysis is that it was restricted to the 13 negative symptoms examined by Liddle (1987) because their study sought to test Liddle's model. Thus, this analysis did not examine the factor structure of the full SANS.

When the structure of negative symptom measures is examined in the context of positive symptoms, it is more likely that negative symptoms will emerge as a unitary factor. For example, when positive and negative symptoms are included in a factor analysis, factors tend to coalesce, organized around positive or negative symptom dimensions first. Although negative symptoms may have further dimensionality, too few factors may be retained for detection of this dimensionality. It should be noted that an in-depth examination of the dimensionality of negative symptoms on the SANS does not determine the exact role for negative symptoms in a larger theoretical organization or classification of symptoms. For example, Nicholson and Neufeld (1993) recently proposed a two-factor model of the schizophrenias: one dimension based on the overall severity of the disorder (largely in reference to the patient's level of paranoia), and the other dimension based on the current severity of symptoms. These theoretical constructions depend on the development of symptom measures such as the SANS to examine potential correlates of these symptoms with premorbid functioning and course.

To our knowledge, only two studies have examined the structure of the full SANS without also including positive symptom measures: those by Mueser et al. (1994) and Peralta and Cuesta (1995). In the study by Mueser et al. (1994), an exploratory factor analysis of the SANS was performed using data from 207 schizophrenia patients involved in the multisite study Treatment Strategies for Schizophrenia (TSS; Schooler, Keith, Severe, & Matthews, 1989). Patients were assessed 2–4 weeks after hospital admission for an index episode. Only the 19 items that were not global items were included. (Global items represent the rationally derived structure of the SANS.) The exploratory principal axis factor analysis with an oblique rotation found three underlying factors that generally represented the following subscales: (a) the Affective Flattening–Blunting subscale, (b) the Avolition–Apathy and Anhedonia–Asociality subscales, and (c) the Alogia and Inattention subscales. The intercorrelations of the factors were in the moderate range, from .46 to .55 ($ps < .05$). These findings suggest that although the rationally derived five-factor structure of the SANS was not replicated, the measure indeed has an underlying multidimensional structure.

Peralta and Cuesta (1995) examined several models with CFA, using a Spanish-language version of the SANS administered to 253 schizophrenia patients. The models tested included the unidimensional model, the original five-factor rationally derived model, and several variants of two- and three-factor models. The authors interpreted their findings as supporting the original five-factor model, without the inappropriate affect item present in the original version of the SANS. Several important

aspects of the study warrant attention. First, it may be important to fully address the cross-cultural issues with the individual symptoms on the SANS before generalizations can be made about the structure of the SANS across language. Second, none of the models in the Peralta and Cuesta (1995) study fit compellingly well. The best models in the article had relatively high $\chi^2:df$ ratios (Bentler & Chou, 1988), and the fit indices were less than the recommended minimum cutoff of .90 (Bentler & Bonett, 1980). Last, the five-factor model included one factor with only two indicators; as mentioned above, two-indicator models tend to be unstable (Boomsma, 1982; McDonald, 1985).

In the current study we examined the structure of negative symptoms as measured by the SANS. We used CFA techniques to test the initial findings reported in Mueser et al. (1994). Whereas exploratory analyses can suggest a structure of a negative symptom measure, CFAs can help an investigator test the appropriateness of a specified structure. This study provided an opportunity to test the structure of the SANS in a large cohort of carefully diagnosed schizophrenia patients. We also examined the loading of the items on each factor and suggested ways in which the SANS might be improved.

Method

Treatment Strategies for Schizophrenia (TSS) Study

The participants were patients with schizophrenia, schizoaffective disorder, or schizophreniform disorder who were participants at one of five sites in the TSS study. The TSS study was a National Institute of Mental Health (NIMH) collaborative study (Schooler et al., 1989) that examined the effects of three neuroleptic medication maintenance strategies and two different approaches to family treatment. Patients were recruited following a symptom exacerbation and treated with fluphenazine decanoate (FPZ) and supplemental medications as indicated for a stabilization phase of the study (usually lasting 3–6 months). Patients were considered stabilized when over a 1-month period, no psychotic symptom (i.e., conceptual disorganization, grandiosity, hallucinatory behavior, and unusual thought content) on the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962, 1988; Woerner, Mannuzza, & Kane, 1988) was greater than moderate. Alternatively, patients who had more severe symptoms that were stable over time, and whose treatment team judged they could be clinically managed in a dosage reduction study, were also considered “stabilized.” Patients whose symptoms could be adequately stabilized on FPZ (12.5–50 mg every 2 weeks) as the only neuroleptic medication and who required no other major psychotropic medications (i.e., antidepressants, lithium, anticonvulsants) were then randomized to one of three dosage maintenance conditions—standard therapeutic dosage levels, low dosage levels, and targeted dose (in which patients received medication only when they began to have a symptom exacerbation)—and to one of two family treatments (see Keith, Bellack, Frances, Mance, & Matthews, 1989; Schooler et al., 1989).

Patients were assessed with the SANS 2–4 weeks after admission for the index episode by the TSS study psychiatrist trained in the use of the SANS (Time 1) and then again at the point of entry into the double-blind phase of the study (3–6 months later, Time 2). Patients who were not sufficiently stabilized to enter the double-blind phase of the study were assessed again and terminated from the study. Patients were monitored on an ongoing basis, and when patients showed prodromal signs of relapse, open-labeled medication was added, which consisted of oral FPZ; FPZ was administered by injection if compliance was a factor.

This supplemental medication was discontinued at the discretion of the treating psychiatrist.

Participants

Inclusion criteria for the TSS study included (a) being between the ages of 18 and 55; (b) being willing to take FPZ injections and not receive (or be willing to be withdrawn from) other major psychotropic medications; (c) having a minimum of 4 hr per week of contact with the family of origin (or legal guardian); (d) being willing to provide consent to participate in the dosage maintenance part of the study and the family intervention and having at least one relative willing to participate in the family treatments; and (e) having had a psychiatric hospitalization or symptom exacerbation within the past 3 months. Exclusion criteria included (a) current pregnancy; (b) current hospitalization or relapse precipitated by alcohol or drug abuse; (c) current or recent (past 3 months) dependence on alcohol, barbiturates, stimulants, or narcotics; and (d) epilepsy or organic brain syndrome. This information was gathered through a review of the patient's chart, structured clinical interviews with the patient, and interviews with the patient's relatives.

The participants included in the current study were 457 patients enrolled in the TSS project who had SANS assessments at the initial assessment after their index hospitalization (Time 1) or at the end of the stabilization period at the point of entry into the double-blind treatment study (Time 2). All patients met the criteria in the revised third edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R*; American Psychiatric Association, 1987) for schizophrenia ($N = 350$), schizophreniform disorder ($N = 29$), or schizoaffective disorder ($N = 78$) as determined by the Structured Clinical Interview for *DSM-III-R*, PD Version (SCID-PD; Spitzer, Williams, Gibbon, & First, 1988). The sample was 66% men, 50% African American, and 41% White, and 83% had never married.

We conducted a multivariate analysis of variance (MANOVA) to determine whether the patients differed in age and psychiatric history by diagnosis. The MANOVA was significant, indicating overall differences, $F(10, 866) = 4.35, p < .001$. Univariate analyses of variance indicated that the groups differed only in age and number of prior psychiatric hospitalizations ($ps < .01$). The mean ages and number of hospitalizations by patient diagnosis were as follows: schizophrenia, 30.1 years ($SD = 7.5$) and 3.5 hospitalizations ($SD = 4.4$); schizophreniform disorder, 24.1 years ($SD = 3.9$) and 0.9 hospitalizations ($SD = 2.2$); and schizoaffective disorder, 29.1 years ($SD = 7.1$) and 5.2 hospitalizations ($SD = 7.4$). Patients with schizophreniform disorder were younger and had less extensive psychiatric histories than the patients in the other two diagnostic groups ($ps < .01$). Patients did not differ by diagnosis in the age of onset of first psychiatric symptoms (range = 19.7 years for schizoaffective disorder to 21.8 years for schizophreniform disorder) or age at first hospitalization (range = 22.5 years for schizoaffective disorder to 23.1 years for schizophrenia).

SANS data were available for 401 patients at Time 1 and 345 patients at Time 2. Not all participants continued in the study after being recruited; thus some patients did not have completed SANS ratings at the second assessment. We examined the demographic differences between patients who had assessments at Time 1 and no assessments at Time 2 ($N = 112$) and those patients who had assessments at both Time 1 and Time 2 ($N = 289$). Patients who were available only at the Time 1 assessment were not significantly different from those who had both assessments in age, age of onset of first psychiatric symptom, age of first hospitalization, number of previous hospitalizations, or total months of psychiatric hospitalization (all $ps > .05$). SANS data for 56 additional patients were available at Time 2 (with no Time 1 data) because SANS assessments were added to the assessment battery after the inception of the TSS study. These 56 patients also were not significantly different on demographic variables from the 289 patients who had SANS data at both Time 1 and Time 2 assessments (all $ps > .05$).

Measures

The TSS study used a comprehensive battery of assessments. The present investigation concerned only the SANS assessments and the chronicity, history, and demographic variables presented above.

Negative symptom assessments. The Scale for the Assessment of Negative Symptoms (Modified) (SANS; Andreasen, 1982, 1984) consists of 24 items grouped into five conceptually related subscales: Affective Flattening or Blunting, Alogia, Avolition–Apathy, Anhedonia–Asociality, and Inattention. Each cluster is represented by at least two discrete items and a globally rated item representing the severity of the entire subscale. For all analyses, we used only the 19 nonglobal items because their inclusion might improperly favor the factor analytic solution dictated by this rationally derived structure of the SANS.

The version of the SANS was modified for use in the TSS study and differed in several respects from that developed by Andreasen (1982). First, SANS ratings were made based solely on an interview and observations during the interview, rather than on an interview combined with an examination of available medical records and discussions with other medical providers (e.g., nurses), as originally suggested by Andreasen. In order to increase the standardization of how symptoms were elicited in the SANS interview, one of the TSS collaborators (R. M. Mance) developed a set of probe questions for items that are based on patient response rather than observation; these questions were routinely incorporated into the interview (see Mueser et al., 1994). Second, the SANS interview assessed negative symptoms over the past week, rather than the past month, as in Andreasen's (1982) version. Andreasen noted that investigators must determine the time frame for the SANS on the basis of their own study design. Also, we believed that the quality of information about recent psychopathology obtained from an interview would decline sharply if the patient was required to recall symptoms from more than 1 week ago. These changes were made so that the SANS could be administered in a fashion similar to that of another measure of psychopathology used in the study, the BPRS. Third, the "inappropriate affect" item from the Affective Flattening or Blunting subscale was dropped because it has been found not to correlate significantly with the overall subscale score (Andreasen, 1982; Moscarelli et al., 1987). Fourth, SANS ratings were made on 5-point Likert-type scales (1 to 5), rather than the 6-point scales used by Andreasen (1982). Fifth, because the SANS was administered on multiple occasions in the study, a list of words was developed to use as alternates to spelling the word "WORLD" in Item 23 (FIRST, BRING, FRANK, STORY, WORDS).

Several investigators have demonstrated good reliability of the SANS, with intraclass correlation coefficients ranging from .70 to .83 in Andreasen's (1982) study and from .53 to .88 in Schuldberg et al.'s (1990) study. Twenty-four-month test–retest reliability for the global items in the Schuldberg et al. (1990) study ranged from .25 to .53; the 24-month test–retest reliability for the composite of all of the items was .50. In the current study, training in the SANS was conducted initially at a meeting attended by all the psychiatrists who were working on the TSS project, and the reliability was assessed with videotaped interviews conducted throughout the study. Monthly conference calls including the psychiatrists and NIMH staff were conducted in order to discuss issues pertaining to the use of these scales. Reliability analyses conducted on these data supported moderately good reliability, although somewhat lower than that in studies performed at a single hospital. The intraclass correlations were positive and significant for all items except "physical anergia" and low but significant for "poverty of content of speech" (.08; the intraclass correlations for all of the other items ranged from .26 to .94). The 3–6-month test–retest reliability findings were good, with the correlations for the composites of the subscales ranging from .37 to .54. The test–retest correlations did not differ across the five sites of the TSS study. (See Mueser et al., 1994, for an in-depth examination of the reliability of the SANS in the TSS study.)

Patient's clinical characteristics and family history. Several other

clinical characteristics were assessed using structured interviews, usually about 1 month after the patient's discharge from the index hospitalization. History was assessed with the Social and Psychiatric History (SPH) form, which was created for the TSS study. Ratings were based on both chart review and an interview with a family member who had the most contact with the patient. The following variables were examined in the current study: (a) behavior problems in childhood (1 = *yes*, 2 = *no*), (b) highest age at which the patient functioned like his or her peers, (c) patient's functioning prior to the onset of psychiatric symptoms (rated from 1 = *very well* to 3 = *poorly*), (d) age at the appearance of the first psychiatric symptoms, (e) age at the first psychiatric hospitalization, (f) number of hospitalizations prior to the index hospitalization, and (g) number of months in hospitalizations prior to the index hospitalization. Family psychiatric history of the patient's mother, father, and sibling (with the worst history if more than one sibling had psychiatric history) was also assessed with the SPH form. Possible ratings ranged from 1 (*no psychiatric history*) to 8 (*chronic hospitalizations totaling 5 years or more*).

Social adjustment was assessed with a modified version of the Social Adjustment Scale—Patient Version (SAS; Schooler, Hogarty, & Weissman, 1978); the following variables were used in the current study: instrumental role functioning, social leisure activities, household adjustment, extended family, and general adjustment. Each item was rated on a 5-point scale ranging from 1 to 5, with high scores representing severe dysfunction; each item has behavioral anchors specific to the content of the item. Training on the SPH and the SAS was conducted by TSS project staff, and monthly conference calls across sites and with NIMH staff directing the TSS study were conducted in order to discuss the use of these scales. Videotaped SAS interviews were conducted by each of the raters, the tapes were reviewed by NIMH staff, and the tapes were also rated by all of the other raters. Monthly conference calls were used to discuss discrepancies in ratings of the videotaped interviews as well as issues that were raised in the use of the scale. All raters on these scales were blind to the ratings on patients' symptom assessments.

Diagnosis was assessed with the SCID-PD (Spitzer et al., 1988) during the index hospitalization by project staff. Training for the structured diagnostic interview was conducted by the developers of the SCID through live training sessions, as well as through their training videotapes. All diagnosticians were experienced clinicians who were closely supervised by doctoral-level staff. Diagnosticians used patient interviews, chart information, and information from family members to make the diagnostic assessment. There was no formal assessment of reliability, but all sites provided ongoing supervisory support for clarification of diagnostic issues. Another rating used in the current study from the diagnostic interview concerned the severity of the patient's illness at the height of the index episode. The range of possible ratings was from 1 (*normal, not at all ill*) to 7 (*among the most extremely ill patients*).

Data Analysis

Our goal in the current study was to use CFA techniques to examine the structure of the SANS, using data assessed at the index hospitalization of the TSS study (Time 1). First, we performed CFAs on the SANS using the unitary factor model, as well as the rationally derived five-factor structure specified in the development of the measure. We did this in order to examine the adequacy of these models and to have benchmarks against which to compare other models.

Second, to move beyond this rationally derived model, we sought to cross-validate the factor structure of the SANS that had been identified from a previous unrestricted exploratory factor analysis (EFA) that had used a subsample from the TSS study (Mueser et al., 1994). For the present analyses we repeated the EFA at Time 1, using both the data examined by Mueser et al. (1994) and the additional 194 TSS partici-

pants at the baseline (Time 1) assessment period. As before, only the 19 nonglobal items were included in the EFA in order to prevent biasing of these exploratory results. Also, the analysis in Mueser et al. (1994) included only schizophrenia patients, whereas schizophrenia, schizophreniform disorder, and schizoaffective disorder patients were included in the present analyses. Thus, prior to testing the structure of the SANS at the second assessment using CFA, we reestimated the Time 1 EFA based on all 401 of the available participants.

Third, we estimated a CFA model using data for all 345 participants with SANS ratings available at Time 2 in order to test the factor structure suggested by the EFA. After an initial test of this model, alterations were made in the factor model as suggested by both the reliability results reported in Mueser et al. (1994) and the modification indices (MIs) of the CFA.

Last, we performed validation analyses to obtain empirical support for the best model from these procedures. We conducted correlational analyses between the factor scores from the results of the best model with variables representing the patient's premorbid functioning, age of onset, family history, social functioning, and chronicity. In addition, to examine the role of the dimensions in this solution to treatment outcome in the TSS treatment study, we used the factor scores as covariates in a survival analysis that examined the number of days until the first use of open-label (supplemental) medication with patients experiencing increased symptomatology.

All CFA models were estimated with the PC version of LISREL-7 (Jöreskog & Sörbom, 1988) based on the sample covariance matrix. Note that unlike EFA models, CFA models are considered restricted factor analysis. That is, each item is constrained to load on one and only one factor. We used several criteria for determining the fit of each CFA model. First, we used the Tucker-Lewis fit index (TLI; Tucker & Lewis, 1973) and the comparative fit index (CFI; Bentler, 1990) as recommended by Gerbing and Anderson (1993). Indices were required to exceed the recommended .90 cutoff (Bentler & Bonett, 1980) before a good fit was interpreted. Second, a "rule of thumb" chi-square to degrees of freedom ratio of about 2:1 was interpreted as indicating a good fit (cf. Wheaton, 1988). Third, we used visual inspection of the plot of the deviations of the residuals from normality in order to determine whether additional alteration of the model was needed. Last, the MIs provided by LISREL-7 were used as a guide for making alterations in each model.

Results

Examination of the Unidimensional Factor Model and the Rationally Derived Five-Factor Model

Some investigators have advocated a unidimensional model of negative symptoms, which prompted us to conduct preliminary CFA testing of this model using data assessed at the index hospitalization of the TSS study (Time 1). This model fit extremely poorly; $\chi^2(152, N = 401) = 1,467.28, p < .001$; TLI = 0.60; CFI = 0.65; $\chi^2:df = 9.7:1$. Inspection of the MIs indicated that substantial modification would be needed to improve the model (e.g., there were many correlated item errors) and suggested that there was little support for the unidimensional model using the SANS.

We used a CFA to estimate a model that followed the original five-factor structure of the SANS using the available data of the 401 participants at Time 1. Again, using the scales' 19 nonglobal items, we allowed the items to load on their respective factors as indicated in Table 1 and constrained them from loading on any additional factor. All five factors were allowed to intercorrelate. The initial estimation resulted in a poorly and im-

properly fitting solution in which one of the residual item variances was negative; this is known as a Heywood case (McDonald, 1985): $\chi^2(142, N = 401) = 640.1, p < .001$; TLI = 0.80; CFI = 0.87; $\chi^2:df = 3.7:1$. Heywood cases can be explained several ways, but the most common explanation points to the existence of a factor represented by fewer than three items with sufficiently large loadings (Boomsma, 1982; McDonald, 1985). Because the Inattention factor is represented only by Items 22 and 23, it is likely that any SANS model with this two-item factor would be unstable across samples and could provide poor estimates of the factor loadings (McDonald, 1985).¹

Time 1 Exploratory Factor Analysis

We conducted this EFA on the data from the 401 patients at Time 1 in precisely the same manner as our previous analysis presented in Mueser et al. (1994) was conducted. The analysis was computed with SAS PROC FACTOR and used maximum likelihood estimation with squared multiple correlations as communality estimates (SAS Institute, 1990). A three-factor solution was extracted based on examination of the scree plot and use of the Kaiser-Guttman rule (e.g., eigenvalues greater than 1.0). This three-factor solution was first rotated using an orthogonal varimax rotation and was then rotated again using an oblique promax rotation. The results of reanalysis of the SANS based on the full Time 1 sample were nearly identical to our previous results. As in Mueser et al. (1994), the three factors that resulted corresponded largely to the Affective Flattening or Blunting subscale (with the addition of the "poverty of speech" item), the Alogia and Inattention subscales, and the Avolition-Apathy and Anhedonia-Asociality subscales. We termed these factors, respectively, Diminished Expression, Inattention-Alogia, and Social Amotivation (see Keefe et al., 1992). The intercorrelations of the factors ranged from .46 to .50 ($ps < .05$). The only change in the solution with the current larger sample was that the loading for Item 4 ("poor eye contact") switched from the Diminished Expression factor in our original EFA to the Inattention-Alogia factor in the current EFA. This was not an unexpected finding given that this item was found to load nearly equally on these two factors in the original EFA. (The orderings of the "second" and "third" factors were switched from the original EFA to the current EFA, indicating that the Inattention-Alogia factor accounted for more variance than the Social Amotivation factor in the current EFA. This is an inconsequential difference in the current context.) Given the larger sample used in the current EFA, we chose to adopt the latter factor structure as the more appropriate solution. This factor structure is presented in Table 1.

¹ We also estimated the five-factor model using the data for the 345 participants at Time 2, in order to answer the inevitable question as to the fit of the five-factor model at Time 2. The initial model fit poorly: $\chi^2(142, N = 345) = 479.98, p < .001$; TLI = 0.87; CFI = 0.89; $\chi^2:df = 3.4:1$. After allowing the estimation of several correlated errors, however, we found the model fit well: $\chi^2(139, N = 345) = 263.32, p < .001$; TLI = 0.95; CFI = 0.96; $\chi^2:df = 1.89:1$. This result does not mitigate against the problem that solutions with two-item factors can lead to poorly estimated factor loadings even with large sample sizes (McDonald, 1985, p. 80).

Table 1

Original SANS Subscales and Final Exploratory Factor Analysis Solution Based on the Full Time 1 Sample (N = 401)

Original SANS subscale	SANS item	Diminished Expression	Inattention-Alogia	Social Amotivation
Affective Flattening/Blunting	1. Unchanging facial expressions	.85	.04	.04
Affective Flattening/Blunting	2. Decreasing spontaneous movement	.86	-.11	-.03
Affective Flattening/Blunting	3. Paucity of expressive gesture	.93	-.16	.00
Affective Flattening/Blunting	4. Poor eye contact	.24	.37	.08
Affective Flattening/Blunting	5. Affective nonresponsivity	.76	.07	.09
Affective Flattening/Blunting	6. Lack of vocal inflections	.85	.06	-.03
Alogia	8. Poverty of speech	.50	.36	-.00
Alogia	9. Poverty of content of speech	-.22	.39	.14
Alogia	10. Blocking	-.12	.56	.07
Alogia	11. Increased latency of response	.26	.63	-.09
Avolition/Apathy	13. Grooming and hygiene	-.17	.15	.31
Avolition/Apathy	14. Impersistence at work or school	-.12	.07	.60
Avolition/Apathy	15. Physical anergia	.11	.07	.59
Avolition/Apathy	17. Recreational interests and activities	.06	-.02	.72
Asociality/Anhedonia	18. Sexual interest and activities	.02	-.08	.51
Asociality/Anhedonia	19. Ability to feel intimacy and closeness	-.00	-.05	.77
Asociality/Anhedonia	20. Relationships with friends and peers	.00	-.05	.77
Inattention	22. Social inattentiveness	-.14	.85	-.03
Inattention	23. Inattentiveness during Mental Status testing	-.06	.40	-.10

Note. Item numbers presented are those on the version of the SANS used in this study. Items 7, 12, 16, 21, and 24 are not listed because they are globally rated items that were excluded from the analyses (see text). Loadings greater than .30 are presented in bold text for increased readability. SANS = Scale for the Assessment of Negative Symptoms.

Time 2 Confirmatory Factor Analysis

We next attempted to cross-validate the factor structure of the SANS identified in the EFA at Time 1 by performing a CFA with the same sample at Time 2.² Of course this is not a cross-validation in the true sense of the word (e.g., confirming the structure based on a completely independent sample), but confirming the structure based on the same sample at a second measurement period would nevertheless increase our confidence in the purely exploratory results found at Time 1.

The CFA was based on the sample covariance matrix for the 345 patients with SANS data at Time 2 and was defined to reflect the EFA solution presented in Table 1. Each item loaded on its corresponding factor, and all three factors were allowed to correlate. This initial model was estimated and resulted in the following model chi-square: $\chi^2(149, N = 345) = 558.9, p < .001$; TLI = 0.85; CFI = 0.87; $\chi^2:df = 3.7:1$. Given the highly significant model chi-square, a high chi-square to degree of freedom ratio, and fit indices falling below the recommended .90 cutoff (Bentler & Bonett, 1980), we concluded that this initial model did not adequately fit the observed data.

Next, we examined MIs to determine possible model misspecifications, because if a model is misspecified, the tests of specific path coefficients may be incorrect (see MacCallum, 1986). On the basis of the MIs we defined a number of conditions that must be met prior to freeing a parameter. First, only correlated measurement errors were considered, and no items were allowed to cross-load on a second factor. Second, any parameter to be freed had to be strongly consistent with theory. Finally, freeing the parameter had to decrease the overall model chi-square by at least 6.6 ($p < .01$). The first parameter to fit these criteria was freed, the model was reestimated, and the MIs were again examined. We repeated this process until no param-

eters fit the above criteria. A total of four parameters were freed. These were correlated measurement errors between Items 2 and 3, 8 and 11, 9 and 13, and 19 and 20 (see Table 1). This final model was estimated and was found to fit the observed data moderately well: $\chi^2(145, N = 345) = 302.1, p < .001$; TLI = 0.94; CFI = 0.95; $\chi^2:df = 2.1:1$. All items loaded positively and significantly on their corresponding factors. The interfactor correlations were .82 between the Diminished Expression and Inattention-Alogia factors, .61 between the Inattention-Alogia and Social Amotivation factors, and .57 between the Diminished Expression and Social Amotivation factors.

We concluded that this three-factor model best explained the interrelations observed among all 19 SANS items. However, this "best"-fitting model still did not fit the observed data compellingly well. There were several large standardized residuals, and the plot of the deviations of these residuals from normality indicated some non-normality in their distribution. In addition, there were several large and significant MIs indicating the need to free cross-loadings of the items on the factors. One of the two largest of these MIs involved cross-loadings between Item 8 ("poverty of speech") and the Inattention-Alogia factor (MI = 21.9; this item was specified to load on Diminished Expression); the other cross-loading involved Item 9 ("poverty of content of speech") and the Diminished Expression factor (MI = 10.6; this item was specified to load on Inattention-Alogia). These indicators suggest that Items 8 and 9 are not uniquely associated with any one factor and thus are not as useful in identifying discrete dimensions of negative symptoms. Items 8 and 9 were also involved in two of the four post hoc

² Readers interested in obtaining the variance-covariance matrix used for these analyses should contact Steven L. Sayers.

correlated measurement errors. It is interesting to note that one of these items was somewhat problematic in our previous analyses (Mueser et al., 1994). Item 9 had a low item-to-subscale correlation ($r = .25$) and a low intraclass correlation (.08), suggesting problems with use of the item. Most problematically, Items 8 and 9 are not logically independent. In order to show a lack of content of speech, one must show adequate production of speech. We decided that, taken together, these items, particularly Item 9, may have been contributing to the poor fit of the overall model. To further explore this issue, we dropped Item 9 and reestimated the CFA model using the remaining 18 SANS items.

Confirmatory Factor Analysis of 18 SANS Items

The CFA model was estimated as before with the exception of the deleted item (note that the four correlated errors previously freed were set back to zero). This initial model was estimated and was found to fit the data poorly: $\chi^2(132, N = 345) = 474.5$, $p < .001$; TLI = 0.86; CFI = 0.92; $\chi^2:df = 3.6:1$. MIs were again examined, and one correlated error was estimated between Items 2 and 3 (note that this was one of the same correlated errors freed in the initial CFA model). This model was estimated and was found to fit the data somewhat better: $\chi^2(131, N = 345) = 344.5$, $p < .001$; TLI = 0.92; CFI = 0.93; $\chi^2:df = 2.6:1$. However, examination of the two largest MIs suggested correlated errors between items on two different factors (Items 8 and 11; MI = 40.7) and the need to allow Item 8 to load on the Inattention-Alogia factor in addition to the Diminished Expression factor (Item 8; MI = 39.9). However, we wished to avoid cross-loadings and correlated errors across two different factors because they were not theoretically defensible. Examination of the initial EFA from Time 1 indicated that Item 8 loaded $> .30$ on both Inattention-Alogia and Diminished Expression, which was somewhat consistent with the original placement of Item 8 ("poverty of speech") on the Alogia subscale. Thus, we decided to reexamine the model fit with Item 8 loading on the Inattention-Alogia factor instead of the Diminished Expression factor.

As with the models tested earlier, the model was reestimated with the previously freed parameters set back to zero. This model fit poorly: $\chi^2(132, N = 345) = 437.39$, $p < .001$; TLI = 0.88; CFI = 0.90; $\chi^2:df = 3.3:1$. However, examination of the MIs led to freeing the measurement errors for the following pairs of items: 2 and 3, 13 and 17, and 19 and 20. This final model fit well: $\chi^2(129, N = 345) = 250.58$, $p < .001$; TLI = 0.95; CFI = 0.96; $\chi^2:df = 1.9:1$. Examination of the standardized residuals, plots of the deviations of the residuals from normality, and MIs suggested no further problems with specification error. As before, all items loaded positively and significantly on their corresponding factors. The interfactor correlations were .83 between the Diminished Expression and Inattention-Alogia factors, .56 between the Inattention-Alogia and Social Amotivation factors, and .56 between the Diminished Expression and Social Amotivation factors. It should be noted that the analyses of the 18 items of the SANS are data driven and thus exploratory in nature. Nevertheless, these analyses provide compelling evidence that this three-factor structure best explains the observed interrelations among the subset of 18 SANS items.³

Table 2

Correlations of SANS Factor Scores at Time 2 With Patients' Clinical Characteristics

Clinical characteristic	Diminished Expression	Social Amotivation	Inattention-Alogia
Child behavior problems ^a	-.06	-.16**	-.05
Highest age like peers ^b	.00	-.08	-.09
Functioning prior to onset ^c	.08	.17**	.10
Age at first illness ^d	.07	-.04	-.02
Age at first hospitalization ^e	.11*	.01	.03
No. of hospitalizations ^f	-.17**	.00	-.03
No. of months of hospitalization ^g	-.14**	.01	.00
Psychiatric history—mother ^h	.03	-.02	.05
Psychiatric history—father ^h	-.06	-.04	-.05
Psychiatric history—sibling ^h	-.06	-.04	-.07
Severity at height of episode ⁱ	.18*	.18*	.35**

Note. SANS = Scale for the Assessment of Negative Symptoms.

^a 1 = yes, 2 = no ($N = 342$). ^b Highest age at which patient functioned like peers ($N = 342$). ^c Functioning ratings ranged from 1 = very well to 3 = poorly ($N = 345$). ^d Age of first psychiatric illness ($N = 341$). ^e Age at first psychiatric hospitalization ($N = 341$). ^f Number of hospitalizations prior to index hospitalization ($N = 345$). ^g Total number of months of hospitalization prior to index hospitalization. ^h Psychiatric history ratings ranged from 1 = no psychiatric history to 8 = chronic hospitalizations totaling more than 5 years (N for mothers' history = 342; N for fathers' history = 325; N for siblings with the worst psychiatric history = 320). ⁱ Severity of illness at the height of the current episode: 1 = normal, not at all ill; 7 = among the most extremely ill patients ($N = 182$).

* $p < .05$. ** $p < .01$.

Validation Analyses for the 18-Item Solution

We sought to obtain preliminary validation evidence for the 18-item solution presented above by examining the relations between patients' scores on the three factors and clinical characteristics such as premorbid functioning, chronicity, severity of illness, social functioning and treatment outcome. For each patient we computed three factor scores using unit-weighting of the 18 items on their respective factors. Correlations were then computed between patients' scores on these factors and the clinical characteristics, as shown in Table 2.

³ A more conservative approach than switching the "poverty of speech" item (Item 8) to the Inattention-Alogia factor might have been to delete both Items 8 and 9 and then reestimate the model. After conducting this analysis (and after allowing the estimation of several correlated errors), we found the model fit well: $\chi^2(114, N = 345) = 221.7$, $p < .001$; TLI = 0.95; CFI = 0.96; $\chi^2:df = 1.94:1$. However, given the centrality of the "poverty of speech" item in the construct of negative symptoms, and given that its psychometric characteristics were adequate (Mueser et al., 1994), we considered it more useful to retain this key symptom in the analysis.

The correlational results suggested that all of the dimensions were related to greater severity of illness during the patient's worst episode. However, only Social Amotivation was associated with poorer premorbid functioning, as indicated by significant correlations with the presence of childhood behavior problems and poor functioning prior to the onset of psychiatric illness. It is interesting that Diminished Expression was associated with relatively less chronicity, as indicated by significant correlations with the patient's age at first hospitalization, the number of previous hospitalizations, and the number of months in previous hospitalizations. The highest age that the patient functioned like his or her peers and the family psychiatric history variables were not significantly related to any of the SANS dimensions.

Table 3 presents the results of the correlational analyses of the patients' SANS factor scores with the patients' measures of social functioning from the modified SAS. Social Amotivation was moderately and consistently positively associated with the patients' social dysfunction. The Diminished Expression factor score was not significantly correlated with any of the measures of social adjustment, and Inattention–Alogia was correlated with only one of the SAS ratings.

To examine whether the SANS dimensions derived in the present study predicted treatment outcome in the TSS study from which this subsample was drawn, we conducted a survival analysis that paralleled the analyses conducted on the TSS data by Schooler et al. (1996). That study clearly demonstrated the superiority of the standard dose strategy for patients' outcomes, followed in turn by the low dose strategy, and then by the targeted dose strategy. There were no differences in patients' outcome between the two family treatment conditions. Although all of the indicators of relapse yielded similar findings, one of the most sensitive and consistent measures proved to be the survival time marked from the beginning of the double-blind phase until the TSS treatment staff felt it was clinically necessary to use open-label supplemental medication. Therefore, in the present study we examined the utility of the SANS dimensions for predicting patients' outcome on the basis of this measure, over and above the main effects of the medication treatment condition.

Our survival analysis used the 291 patients with complete SANS data at Time 2, who were drawn from the 313 patients who were successfully stabilized and entered into the double-

blind phase of the TSS study. We conducted the analysis using the Kaplan–Meier estimates of the survival function, entering the medication condition and the patients' factor scores as covariates. Because in the TSS study there was no difference in outcome between the family treatment conditions, we ignored this factor in the current analyses. The model allows for right-censoring of the data, which occurs because a portion of the patients “survived” (i.e., did not relapse) before the end of the 2-year treatment period of the study. Parallel to the findings in Schooler et al. (1996), the tests associated with the covariate for the medication condition were highly significant: both the Wilcoxon test, $\chi^2(2, N = 291) = 34.73, p < .001$, which weights early failures more heavily, and the log rank test, $\chi^2(2, N = 291) = 55.33, p < .001$, which weights later failures more heavily (SAS Institute, 1990). The univariate Wilcoxon and log rank tests associated with the scores for the Diminished Expression factor were significant: $\chi^2(1, N = 291) = 4.31, p < .05$, and $\chi^2(1, N = 291) = 4.18, p < .001$, respectively. The findings suggested that higher scores on the Diminished Expression factor were associated with patients' greater survival times. None of the other univariate tests for the Social Amotivation and Inattention–Alogia factor were significant (all $ps > .05$). None of the tests of the simple effects of the covariates within each medication condition (e.g., standard dose condition) were significant, but these results yielded trends that were generally in the same direction as the aggregate effects of all three treatment conditions.

A forward stepwise entry of the factors into the model resulted in a significant log rank test for the model, $\chi^2(2, N = 291) = 8.91, p < .01$. The two significant covariates included Diminished Expression, $\chi^2(1, N = 291) = 4.18, p < .001$, and Social Amotivation, $\chi^2(1, N = 291) = 4.73, p < .001$. These findings indicated that after we adjusted for Diminished Expression, higher Social Amotivation scores were associated with patients' shorter survival times to the point when open-label supplemental medications were needed (i.e., worse outcomes). Inattention–Alogia was not a significant covariate in this analysis. A parallel stepwise analysis of these covariates with the Wilcoxon test was not significant.

Discussion

The results of the current CFA are consistent with the results of the previous EFA, which suggests that the SANS has an internal structure that is somewhat cohesive. The three factors found for the SANS included Diminished Expression, Inattention–Alogia, and Social Amotivation. Notably, the “poor eye contact” item loaded on the Inattention–Alogia factor instead of the Affective Flattening or Blunting subscale, which is entailed within the Diminished Expression factor. The original five-factor model proved to be unstable because of the paucity of items representing one of the factors. Furthermore, a well-fitting, three-factor model received some tentative support. With the exception of the “poor eye contact” item, none of the items in our best-fitting solution were associated with a factor to the exclusion of their original cluster of items specified on the SANS. Thus, although the rationally derived five-factor model provided an excellent original framework for examining nega-

Table 3
Correlations of SANS Factor Scores at Time 2 With Patients' Social Functioning

Social functioning	Diminished Expression	Social Amotivation	Inattention–Alogia
Instrumental role performance ^a	.06	.32***	.05
Household adjustment ^b	.09	.29***	.12*
Extended family functioning ^c	.07	.25***	.01
Social/leisure functioning ^b	.07	.25***	.06
General adjustment ^b	.07	.30***	.07

Note. Higher scores indicate poorer functioning. SANS = Scale for the Assessment of Negative Symptoms.

^a $N = 261$. ^b $N = 263$.

* $p < .05$. *** $p < .001$.

tive symptoms, a similar and more parsimonious three-factor model might suffice.

An interesting set of findings emerged concerning the Social Amotivation factor that suggests that this factor may represent a key underlying etiological process. The Social Amotivation factor was correlated with the other two latent factors at lower levels (r s were .56 and .56) than these factors (Diminished Expression and Inattention-Alogia) were correlated with each other ($r = .82$). Further support for the importance of Social Amotivation was the finding that it was uniquely associated with the existence of child behavior problems, poor premorbid functioning, social adjustment, and—after adjustment for Diminished Expression—shorter survival times before additional medication was needed for restabilization. On the other hand, Diminished Expression was associated with fewer prior hospitalizations, fewer months of prior hospitalizations, and longer survival times. All three dimensions had low but significant correlations with greater severity of the disorder during the index episode. These findings suggest that although Social Amotivation may be related to the other two factors, it may indeed reflect a different component of negative symptom deficits. Specifically, Diminished Expression may primarily reflect expressive deficits, whereas Social Amotivation may reflect deficits in the experience of emotion (thus making social contact less rewarding).

These results might be relevant to studies that suggest that expressive and experiential deficits in schizophrenia patients are unrelated and thus represent separate underlying processes (Berenbaum, & Oltmanns, 1992; Blanchard, Bellack, & Mueser, 1994; Kring, Kerr, Smith, & Neale, 1993). This is interesting in light of other findings that “negative” symptoms are associated with poor neuroleptic response (Kane & Mayerhoff, 1989), poor prognosis (Fenton & McGlashan, 1991), and poor premorbid functioning (Peralta, Cuesta, & de Leon, 1991); these lines of evidence may indeed be relevant only for patients with little social drive and ability to experience pleasure during social contact. Indeed, this idea is consistent with other studies showing that a low number of social contacts and poor social adjustment are predictive of relapse and rehospitalization in schizophrenia (Harrow, Westermeyer, Silverstein, Strauss, & Cohler, 1986; Jonsson & Nyman, 1991; Rajkumar & Thara, 1989; Strauss & Carpenter, 1977). Future researchers might take this into account when examining neuropsychological deficits, morphology data such as ventricular size, and genetic evidence that may have implications for the etiology and development of schizophrenia and schizoaffective disorder.

As noted by Dworkin (1992), the distinction between expressive and experiential dysfunction is often clouded when investigators attempt to measure patients’ apparent deficits in the experience of emotion using the same interview or role-play behavior that is used to assess social skill or competence (see also Dworkin et al., 1993). This is an important distinction in that Diminished Expression is likely to be more easily detected, whereas Social Amotivation requires more inquiry and data gathering about the patient’s social drive, social activities, and emotional experiences. If Social Amotivation is indeed more important to the patient’s premorbid functioning and course of the illness, the expressive-experiential distinction becomes paramount.

Our three-factor solution converges somewhat with the three-factor solution of Keefe et al. (1992). The Diminished Expression and Social Amotivation factors in the current study were very similar to two of the factors in Keefe et al.’s (1992) study with some exceptions. “Physical anergia” loaded on Social Amotivation in the current study rather than on Diminished Expression as in Keefe et al.’s study, and we included on the Social Amotivation factor two items assessing social relationships that were not included in the study by Keefe et al. (“recreational interests and activities” and “sexual interest and activities”). The divergence between the findings in the current study and those of Keefe et al. (1992) is in the remaining factor. In the current study this factor represented the patient’s inattention and alogia symptoms, whereas in the Keefe et al. (1992) study this remaining factor included “inappropriate affect” and “poverty of content of speech,” both of which were excluded in our 18-item solution. As noted above, the wisdom of including inappropriate affect as a negative symptom has been questioned in several studies.

The best-fitting model resulted from dropping the “poverty of content of speech” item. The difficulties with this item may have resulted more from methodological problems than from theoretical problems with the item; “poverty of content” is frequently considered central to the construct of negative symptoms. The item has low reliability (Mueser et al., 1994), and rating “poverty of content” requires adequate levels of speech production (i.e., low levels of “poverty of speech”), making it dependent on the value of the “poverty of speech” item. It is not currently possible to know on which factor the “poverty of content” item belongs without the modification of the definitions of these items or the development of a different measurement strategy. For example, a new approach to measuring these symptoms might involve asking patients to recount some personal information until a criterion amount of speech is produced that allows the interviewer to adequately judge the degree of content contained in this standardized sample of speech.

There is evidence from several studies that suggests that inattention indeed may not be a negative symptom (Peralta, Cuesta, & de Leon, 1994). Inattention is most often included in a Disorganization factor (Liddle, 1987) with other symptoms such as inappropriate affect, bizarre behavior, and positive formal thought disorder. It is unclear in our sample why the Inattention-Alogia factor correlates so highly with Diminished Expression in that inattention could reflect either disorganization or lack of social connection. Clearly, the cause for the inattentiveness might be important in understanding whether it should be considered a negative symptom, and this should be the focus of continued research.

It should be noted that because items on the SANS were rationally arranged on separate factors, this grouping may be significant for the current findings. As the interviewer conducts the assessment session, he or she is guided by the SANS to think about negative symptoms according to this structure. This is most notable in the inclusion of globally rated items for each factor (e.g., Item 7 requires a “global rating of the overall severity of affective flattening or blunting”). To minimize the effects of the global items, we excluded them from the analyses; however, the extent of the effect of the global items on the interviewer’s other ratings is not estimable in the current data set. One

strategy might be to examine the structure of the SANS as in the current study but to use a version of the SANS with the global items omitted. Furthermore, random ordering of the items across interviews may ensure that measurement confounds are minimized.

Although we consider the factor structure in the current study to be the most parsimonious available, we also note that this structure was based not on a priori predictions, but instead on an EFA of an earlier assessment with the same sample. Sources of error inherent in the Time 1 sample would be substantially the same for the Time 2 sample, possibly leading to overconfidence in the CFA solution at Time 2. It is important that these findings be replicated on an independent sample before more definitive conclusions are drawn.

It is also important to consider that the EFA and CFA were conducted at different phases of the illness; the Time 1 assessment occurred 2–4 weeks following the index episode, whereas the Time 2 assessment occurred after a period of time in which many of the patients were stabilized (3–6 months). The crux of this concern is the possibility that the structure of negative symptoms as measured by the SANS is different at different phases of the illness. There is some evidence that some negative symptoms may decline more than others during the patient's stabilization on FPZ with medication (see Mueser et al., 1994). Thus, although these treatment-specific changes do not indicate that the structure of the SANS changes over time, it raises this related question. The CFA results, however, mitigate this concern. The structure that seemed to fit the data at Time 2 was predominantly the same as the EFA factors at Time 1 (minus the item omitted for the Time 2 CFA). Furthermore, the sample we used to conduct the current EFA and CFA included patients with schizophrenia, schizophreniform, and schizoaffective disorders, whereas the sample used for the original EFA conducted by Mueser et al. (1994) included only patients with schizophrenia. It is heartening that greater diagnostic heterogeneity had little or no effect on the structure of the SANS.

In the current study we made no attempt to distinguish so-called "primary" negative symptoms from those that were "secondary" to other symptoms such as depression. Carpenter et al. (1988) made this distinction in defining the deficit syndrome, which is based on the presence of stable negative symptoms across time. The SANS does not call for making these distinctions in assigning severity of the individual negative symptoms; however, there are advantages associated with not making this distinction. First, a lower level of inference is required to make judgments about the severity of symptoms than to determine whether the symptom is secondary to another class of symptoms. Decreasing the degree of inference needed for clinical ratings has the potential for maximizing reliability, especially because the rating instrument in question is used in a variety of settings where the standards for drawing such inferences may vary widely. Indeed, the only study that has examined the reliability of judgments distinguishing primary versus secondary negative symptoms reported only a fair degree of agreement, with Kappas ranging between 0.48 and 0.68 (Flaum & Andreasen, 1995). Second, if a symptom is secondary to another symptom, then this must be demonstrated empirically before an assumption can be made that this is in fact the case. More specifically, depression (as a disorder) may involve increased

anhedonia, but anhedonia can also be elevated as a result of schizophrenia. One must demonstrate that if a full affective syndrome of depression exists, it accounts for the severity of the other negative symptoms both cross-sectionally and longitudinally. Thus, the benefit of using the SANS is that because symptom severity is not discounted owing to some other hypothesized confounding factor, these issues can be examined empirically.

Questions concerning the structure and measurement of negative symptoms are far from settled. Many methodological and substantive questions remain about the class of negative symptoms and their measurement. However, it appears that the SANS can play a useful role in facilitating a detailed examination of which symptoms to include in the construct of negative symptoms, the differential relationships among negative symptoms, and their relation to the etiology, psychopathology, and treatment of schizophrenia.

References

- American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., rev.). Washington, DC: Author.
- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Andreasen, N. C. (1982). Negative symptoms in schizophrenia: Definition and reliability. *Archives of General Psychiatry*, 39, 784–788.
- Andreasen, N. C. (1984). *Modified Scale for the Assessment of Negative Symptoms*. Bethesda, MD: U.S. Department of Health and Human Services.
- Andreasen, N. C. (1985). Positive vs. negative schizophrenia: A critical evaluation. *Schizophrenia Bulletin*, 11, 380–389.
- Andreasen, N. C., Flaum, M., Swayze, V. W., Tyrrell, G., & Arndt, S. (1990). Positive and negative symptoms in schizophrenia. *Archives of General Psychiatry*, 47, 615–621.
- Andreasen, N. C., & Olsen, S. (1982). Negative v. positive schizophrenia: Definition and validation. *Archives of General Psychiatry*, 39, 789–794.
- Andreasen, N. C., Roy, M.-A., & Flaum, M. (1995). Positive and negative symptoms. In S. R. Hirsch & D. R. Weinberger (Eds.), *Schizophrenia* (pp. 28–45). London: Blackwell Scientific.
- Bentler, P. M. (1990). Comparative fit indices in structural models. *Psychological Bulletin*, 107, 238–246.
- Bentler, P. M., & Bonett, G. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin*, 88, 588–606.
- Bentler, P. M., & Chou, C. P. (1988). Practical issues in structural modeling. In J. S. Long (Ed.), *Common problems/proper solutions: Avoiding error in quantitative research* (pp. 161–192). Newbury Park, CA: Sage.
- Berenbaum, H., & Oltmanns, T. F. (1992). Emotional experience and expression in schizophrenia and depression. *Journal of Abnormal Psychology*, 101, 37–44.
- Blanchard, J. J., Bellack, A. S., & Mueser, K. T. (1994). Affective and social-behavioral correlates of physical and social anhedonia in schizophrenia. *Journal of Abnormal Psychology*, 103, 719–728.
- Boomsma, A. (1982). The robustness of LISREL against small sample sizes in factor analysis models. In K. G. Jöreskog & H. Wold (Eds.), *Systems under indirect observation: Causality, structure, prediction, Part I*. Amsterdam: North-Holland.
- Carpenter, W. T., Heinrichs, D. W., & Wagman, A. M. I. (1988). Deficit and nondeficit forms of schizophrenia: The concept. *American Journal of Psychiatry*, 145, 578–583.

- Crow, T. J. (1980). Molecular pathology of schizophrenia: More than one disease process? *British Medical Journal*, 280, 66-68.
- Crow, T. J. (1985). The two-syndrome concept: Origins and current status. *Schizophrenia Bulletin*, 11, 471-486.
- Czobor, P., Bitter, I., & Volavka, J. (1991). Relationship between the Brief Psychiatric Rating Scale and the Scale for the Assessment of Negative Symptoms: A study of their correlation and redundancy. *Psychiatry Research*, 36, 129-139.
- Dworkin, R. H. (1992). Affective deficits and social deficits in schizophrenia: What's what? *Schizophrenia Bulletin*, 18, 59-64.
- Dworkin, R. H., Cornblatt, B. A., Friedmann, R., Kaplansky, L. M., Lewis, J. A., Rinaldi, A., Shilliday, C., & Erlenmeyer-Kimling, L. (1993). Childhood precursors of affective vs. social deficits in adolescents at risk for schizophrenia. *Schizophrenia Bulletin*, 19, 563-577.
- Fenton, W. S., & McGlashan, T. H. (1991). Natural history of schizophrenia subtypes. II. Positive and negative symptoms and long-term course. *Archives of General Psychiatry*, 48, 978-986.
- Flaum, M., & Andreasen, N. (1995). The reliability of distinguishing primary versus secondary negative symptoms. *Comprehensive Psychiatry*, 36, 421-427.
- Gerbing, D. W., & Anderson, J. C. (1993). Monte Carlo evaluations of the goodness of fit indices for structural equation models. In K. A. Bollen & J. S. Long (Eds.), *Testing structural equation models* (pp. 40-65). Newbury Park, CA: Sage.
- Harrow, M., Westermeyer, J. F., Silverstein, M., Strauss, B. S., & Cohler, B. J. (1986). Predictors of outcome schizophrenia: The process-reactive dimension. *Schizophrenia Bulletin*, 12, 195-206.
- Jonsson, H., & Nyman, A. K. (1991). Predicting long-term outcome in schizophrenia. *Acta Psychiatrica Scandinavica*, 83, 342-346.
- Jöreskog, K. G., & Sörbom, D. (1988). *LISREL-7*. Mooresville, IN: Scientific Software.
- Kane, J. M., & Mayerhoff, D. (1989). Do negative symptoms respond to pharmacological treatment. Symposium: Negative Symptoms in Schizophrenia (1987, London, England). *British Journal of Psychiatry*, 155, 115-118.
- Keefe, R. E., Harvey, P. D., Mohs, R. C., Lenzenweger, M. F., Davidson, M., Apter, S., Schmeidler, J., & Davis, K. L. (1992). Empirical assessment of the factorial structure of clinical symptoms in schizophrenia: Negative symptoms. *Psychiatry Research*, 44, 153-165.
- Keith, S. J., Bellack, A., Frances, A., Mance, R., & Matthews, S. (1989). The influence of diagnosis and family treatment on acute treatment response and short term outcome in schizophrenia. *Psychopharmacology Bulletin*, 25, 336-339.
- Kring, A. M., Kerr, S., Smith, D. A., & Neale, J. M. (1993). Flat affect in schizophrenia does not reflect diminished subjective experience of emotion. *Journal of Abnormal Psychology*, 102, 507-517.
- Lenzenweger, M. F., Dworkin, R. H., & Wethington, E. (1991). Examining the underlying structure of schizophrenic phenomenology: Evidence for a three-process model. *Schizophrenia Bulletin*, 17, 516-524.
- Liddle, P. F. (1987). The symptoms of chronic schizophrenia: A re-examination of the positive-negative dichotomy. *British Journal of Psychiatry*, 151, 145-151.
- MacCallum, R. (1986). Specification searches in covariance structure modeling. *Psychological Bulletin*, 100, 107-120.
- McDonald, R. (1985). *Factor analysis and related methods*. Hillsdale, NJ: Erlbaum.
- McGlashan, T. H., & Fenton, W. S. (1992). The positive-negative distinction in schizophrenia: Review of natural history validators. *Archives of General Psychiatry*, 49, 63-72.
- Miller, D. D., Arndt, S., & Andreasen, N. C. (1993). Alogia, attentional impairment, and inappropriate affect: The status in the dimensions of schizophrenia. *Comprehensive Psychiatry*, 34, 221-226.
- Moscarelli, M., Maffei, C., Cesana, B. M., Boato, P., Farma, T., Grilli, A., Lingardi, V., & Cazzullo, C. L. (1987). An international perspective on assessment of negative and positive symptoms in schizophrenia. *American Journal of Psychiatry*, 144, 1595-1598.
- Mueser, K. T., Bellack, A. S., Morrison, R. L., & Wixted, J. T. (1990). Social competence in schizophrenia: Premorbid adjustment, social skill, and domains of functioning. *Journal of Psychiatric Research*, 24, 51-63.
- Mueser, K. T., Douglas, M. S., Bellack, A. S., & Morrison, R. L. (1991). Assessment of enduring deficit and negative symptom subtypes in schizophrenia. *Schizophrenia Bulletin*, 17, 565-582.
- Mueser, K. T., Sayers, S., Schooler, N. R., Mance, R. M., & Haas, G. L. (1994). A multi-site investigation of the reliability of the scale for the assessment of negative symptoms. *American Journal of Psychiatry*, 151, 1453-1462.
- Nicholson, I. R., & Neufeld, R. W. (1993). Classification of the schizophrenia according to symptomatology: A two-factor model. *Journal of Abnormal Psychology*, 102, 259-270.
- Overall, J. E., & Gorham, D. R. (1962). The Brief Psychiatric Rating Scale. *Psychological Reports*, 10, 799-812.
- Overall, J. E., & Gorham, D. R. (1988). The Brief Psychiatric Rating Scale (BPRS): Recent developments in ascertainment and scaling. *Psychopharmacology Bulletin*, 24, 97-99.
- Peralta, V., & Cuesta, M. J. (1995). Negative symptoms in schizophrenia: A confirmatory factor analysis of competing models. *American Journal of Psychiatry*, 152, 1450-1457.
- Peralta, V., Cuesta, M. J., & de Leon, J. (1991). Premorbid personality and positive and negative symptoms in schizophrenia. *Acta Psychiatrica Scandinavica*, 84, 336-339.
- Peralta, V., Cuesta, M. J., & de Leon, J. (1994). An empirical analysis of latent structures underlying schizophrenic symptoms: A four-syndrome model. *Biological Psychiatry*, 36, 726-736.
- Pogue-Geile, M. F. (1989). The prognostic significance of negative symptoms in schizophrenia. *British Journal of Psychiatry*, 155, 123-127.
- Pogue-Geile, M. F., & Zubin, J. (1988). Negative symptomatology and schizophrenia: A conceptual and empirical review. *International Journal of Mental Health*, 16, 3-45.
- Rajkumar, S., & Thara, R. (1989). Factors affecting relapse in schizophrenia. *Schizophrenia Research*, 2, 403-409.
- SAS Institute. (1990). The LIFETEST procedure. In *SAS/STAT user's guide: Version 6* (4th ed., Vol. 2, pp. 1027-1069). Cary, NC: SAS Institute.
- Schooler, N. R., Hogarty, G., & Weissman, M. (1978). Social Adjustment Scale II (SAS-II). In W. A. Hargreaves, C. C. Atkinson, & J. E. Sorenson (Eds.), *Resource materials for community mental health program evaluations* (DHEW Publication No. ADM (79-328). Rockville, MD: National Institute of Mental Health.
- Schooler, N. R., Keith, S. J., Severe, J. B., & Matthews, S. (1989). Acute treatment response and short term outcome in schizophrenia. *Psychopharmacology Bulletin*, 25, 331-335.
- Schooler, N. R., Keith, S. J., Severe, J. B., Matthews, S., Bellack, A. S., Glick, I. D., Hargreaves, W. A., Kane, J. M., Ninan, P. T., Frances, A., Jacobs, M., Lieberman, J. A., Mance, R., Simpson, G. M., & Woerner, M. G. (1996). *Relapse and rehospitalization during maintenance treatment of schizophrenia: The effects of dose reduction and family treatment*. Manuscript submitted for publication.
- Schulderberg, D., Quinlan, D. M., Morgenstern, H., & Glazer, W. (1990). Positive and negative symptoms in chronic psychiatric outpatients: Reliability, stability, and factor structure. *Psychological Assessment*, 2, 262-268.
- Spitzer, R. L., Williams, J. B. W., Gibbon, M., & First, M. B. (1988). *Structured Clinical Interview for DSM-III-R (SCID)*. New York: New York State Psychiatric Institute, Biometrics Department.
- Strauss, J. S., & Carpenter, W. T. (1977). Prediction of outcome in

- schizophrenia III. Five-year outcome and its predictors. *Archives of General Psychiatry*, 34, 159-163.
- Strauss, J. S., Carpenter, W. T., Jr., & Bartko, J. J. (1974). Diagnosis and understanding of schizophrenia, Part II: Speculations on the process that underlie schizophrenic symptoms and signs. *Schizophrenia Bulletin*, 11, 61-76.
- Tucker, L. R., & Lewis, C. (1973). A reliability coefficient for maximum likelihood factor analysis. *Psychometrika*, 38, 1-10.
- Walker, E., Harvey, P. D., & Perlman, D. (1988). The positive and negative symptoms distinction in psychosis. A replication and extension of previous findings. *Journal of Nervous and Mental Disease*, 176, 359-363.
- Wheaton, B. (1988). Assessment of fit in overidentified models with latent variables. In J. S. Long (Ed.), *Common problems/proper solutions: Avoiding error in quantitative research* (pp. 193-225). Newbury Park, CA: Sage.
- Woerner, M. G., Mannuzza, S., & Kane, J. M. (1988). Anchoring the BPRS: An aid to improved reliability. *Psychopharmacology Bulletin*, 24, 112-117.

Received May 8, 1995

Revision received March 11, 1996

Accepted April 3, 1996 ■

Call for Nominations

The Publications and Communications Board has opened nominations for the editorship of *Developmental Psychology* for the years 1999-2004. Carolyn Zahn-Waxler, PhD, is the incumbent editor.

Candidates should be members of APA and should be available to start receiving manuscripts in early 1998 to prepare for issues published in 1999. Please note that the P&C Board encourages participation by members of underrepresented groups in the publication process and would particularly welcome such nominees. Self nominations are also encouraged.

To nominate candidates, prepare a statement of one page or less in support of each candidate and send to

Janet Shibley Hyde, PhD, Search Committee Chair
c/o Lee Cron, P&C Board Search Liaison
American Psychological Association
750 First Street, NE, Room 2004
Washington, DC 20002-4242

Members of the search committee are Bennett Bertenthal, PhD; Susan Crockenberg, PhD; Margaret Spencer, PhD; and Esther Thelen, PhD.

First review of nominations will begin December 9, 1996.