Examining the Underlying Structure of Schizophrenic Phenomenology: Evidence for a Three-Process Model

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Abstract

The present report examined the latent structure of schizophrenic phenomenology. Schizophrenic patient case histories \((n = 192)\) were rated for positive symptoms, negative symptoms, and premorbid social adjustment and the observed covariation among these clinical features was evaluated using a model-based confirmatory factor analytic approach. Our results indicated that schizophrenic phenomenology was best characterized by three distinct underlying structures. These data provide empirical support for Strauss et al.'s (1974) three-process model, which suggests that positive symptoms, negative symptoms, and disordered premorbid personal-social relationships are three distinct classes of phenomenology possibly reflective of three relatively independent pathological processes in schizophrenia. The data are also consistent with Crow's (1980, 1985, 1987) model of schizophrenic symptomatology, differentiating social impairment from both positive and negative symptoms. The heuristic implications of these data for the development of schizophrenia are discussed and the utility of a replication of the present study is noted.

Strauss et al. (1974) hypothesized that three independent pathological processes might underlie the observed occurrence of three general classes of schizophrenic signs and symptoms, namely, positive symptoms (hallucinations, delusions, formal thought disorder, and bizarre behavior), negative symptoms (blunted affect, alogia, and apathy), and disordered personal-social relationships. Although the precise etiologies of the three putative disease processes remained unspecified, Strauss et al. (1974) argued persuasively that the patterning of phenotypic manifestations in schizophrenia might reflect the actual pathological processes. Thus, Strauss et al. proposed a model of three functional processes in schizophrenia that consisted of two distinct components: (1) At the descriptive level, three separate symptom dimensions (or clusters) were hypothesized to characterize the phenotypic manifestations of the illness. (2) At the level of pathogenesis, the three symptom dimensions were hypothesized to be reflective of three relatively distinct etiologies or pathophysiologies or both.

Two of the three symptom classes emphasized by Strauss et al. (1974), positive and negative symptoms, have been the focus of recent theory development and research (Crow 1980; Andreasen and Olsen 1982; Crow 1985). Currently available empirical data support the validity of positive and negative symptoms as two relatively independent symptom dimensions (Lenzenweger et al. 1989) probably reflective of two pathological processes (Walker and Lewine 1988). Deficits in premorbid social functioning, however, are known to be associated with negative symptoms but not with positive symptoms (Walker and Lewine 1988); yet it is unclear whether such deficits should be distinguished from negative phenomenology as suggested by Strauss et al. (1974). An empirical examination of the structures underlying schizophrenic phenomenology might help to further establish disordered

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social functioning as a third independent dimension underlying observed schizophrenic symptoms, one which is relatively distinct from both positive and negative symptoms. Such data would provide additional support for Strauss et al.'s third pathological process.

An important conceptual issue arises in considering the nature of disordered personal-social relationships in relation to positive and negative symptoms in schizophrenia. What is the nature of the relationship between a psychotic illness and social functioning across the lifespan? Strauss et al. (1974) hypothesized that the personal relationships dimension has its own "longitudinal history and consistency as a psychological process" (p. 68) independent of schizophrenic symptoms. However, it is unclear to what extent this process may be influenced by the emergence of schizophrenia as a disease. Social and personal relationship deficits occurring after the onset of schizophrenia could reflect (1) the continued expression of Strauss et al.'s third pathological process, essentially the post-psychotic reemergence of poor premorbid personality traits (Zubin 1985; Pogue-Geile and Zubin 1988); (2) a genuine negative schizophrenic symptom developing as a feature of the core disease (Andreasen and Olsen 1982); or (3) a "secondary" negative symptom resulting from the impact of psychosis or antipsychotic medication or both (Crow 1985, 1987; Zubin 1985; Carpenter et al. 1988). It is also plausible that impaired post-onset social relations may not be related to the schizophrenia disease process per se but, rather, reflects a strategy used by patients to withdraw from social contact for fear of undesired societal reactions to the diagnosis of schizophrenia (Strauss et al. 1974; Link et al. 1989). Clearly, premorbid deficits in social functioning must be distinguished from social functioning impairments noted in patients after the onset of schizophrenia, and this distinction should be accounted for in any evaluation of Strauss et al.'s three-process model.

We undertook the present study to address two specific hypotheses, both derived from the "descriptive" component of the Strauss et al. three-process model. First, we sought to determine if three relatively distinct dimensions underlie schizophrenic phenomenology as Strauss et al. conjectured. In particular, we sought to evaluate how well Strauss et al.'s third process, reflected by disordered personal relationships, could account for an identifiable domain of schizophrenic phenomenology independent of positive and negative symptoms. Second, we sought to specify the nature of the associations between personal-social adjustment and positive and negative schizophrenic symptoms in both the premorbid and the morbid (i.e., post-onset) periods.

Methods

Subjects. As detailed in previous publications (Dworkin and Lenzenweger 1984; Lenzenweger et al. 1989), the case histories of all monozygotic schizophrenic probands and their co-twins from the five major European twin studies of schizophrenia that have been published in English were assembled. These 302 case histories of 151 monozygotic twin pairs were carefully edited to remove all identifying information that might enable a rater to match a proband with his or her co-twin. Zygosity and family history data were also deleted from the case histories before they were rated, and the case histories were then randomly ordered. Because of the intermixing of the histories of schizophrenic probands and their co-twins, the degree and type of psychopathology described in the case histories ranged from psychosis to absence of significant psychopathology. The histories typically covered psychosocial and psychiatric developments across the lifespan for each twin.

Measures. A manual was developed to assess positive and negative schizophrenic symptoms in these case histories. This manual consists of detailed descriptions of five categories of negative symptoms (affective flattening, alogia, avolition-apathy, asociality-withdrawal, and attentional impairment) and five categories of positive symptoms (hallucinations, delusions, positive formal thought disorder, catatonic motor phenomena, and bizarre behavior). The negative symptom portion of the manual is a modification of Andreasen's (1981) interview-based Scale for the Assessment of Negative Symptoms that we adapted for use with case histories. Most of these modifications were minor, with one exception: we shifted anhedonia to the affective flattening symptom section and made asociality-withdrawal a separate symptom section (Andreasen's manual includes the category anhedonia-asociality). Doing so increased reliability by reducing some ambiguity that the raters experienced in pilot trials. The positive symptom portion of the manual consists of phenomenologic descriptions drawn from traditional sources in descriptive psychopathology for the positive symptoms listed above (Hamilton 1976; Wing et al. 1974; American Psychiatric Association 1980). The validity of symptom ratings based on this manual is supported by our pre-
vious research, which found the predicted relationships between these ratings and attentional measures (Cornblatt et al. 1985) and genetic influences for schizophrenia (Dworkin and Lenzenweger 1984; Dworkin et al. 1988).

Two experienced clinicians—a clinical psychologist and a psychiatrist—were trained to rate the 302 case histories, which found the presence or absence of each of the five negative and the five positive symptoms. The two raters were not informed of any theoretical organization concerning positive and negative symptoms that might influence their assessments, and they were instructed to rate as present only those symptoms that were clearly described in the case history material. The data used to make the positive and negative symptom ratings were confined exclusively to the morbid period of the lifespan (i.e., the post-onset period). The raters were instructed to avoid any form of hypothetical inference in their phenomenological assessments if the presence of a symptom was equivocal. The following analyses make use of the average of their ratings for each symptom (for each of the positive and negative symptoms each patient received a symptom rating that was the average of the two raters' assessments). Average ratings were used because of their greater reliability. Acceptable levels of interrater reliability for each of the 10 symptoms were demonstrated with intraclass correlation coefficients (ICC) (Shrout and Fleiss 1979) for the average symptom ratings ranging from 0.68 to 0.91 (the mean of the reliabilities across the 10 symptoms was 0.79). Following Shrout and Fleiss (1979), the formula ICC[3,k] was used to calculate the ICCs we report; ICC[3,k] is equivalent to Cronbach's alpha for average ratings.

Each case was rated by two new raters, a clinical psychologist and an advanced doctoral student in clinical psychology, using both the Phillips Abbreviated Scale of Premorbid Sexual Adjustment (Harris 1975) and the Zigler-Phillips Social Competence Scale (Zigler and Phillips 1960). The case history data used for these ratings were confined exclusively to the premorbid period (Lenzenweger and Dworkin 1987), and interrater reliabilities for the average ratings were 0.95 (Phillips) and 0.82 (Zigler-Phillips). The premorbid period was defined as that phase of the lifespan before the first psychiatric hospitalization for an individual or the first appearance of clear-cut psychotic symptoms. In the vast majority of cases, the premorbid period and onset of schizophrenia were unambiguously noted in the case history material. Each case was also rated for paranoid phenomenology (an additional positive symptom) by a clinical psychologist and an advanced doctoral clinical student using the Venables-O'Connor Scale for Rating Paranoid Schizophrenia (Venables and O'Connor 1959). The reliability of the average Venables-O'Connor Scale ratings was 0.87.

Statistical Analysis. Of the 302 individuals represented in the case history material, 220 (151 schizophrenic probands and 69 schizophrenic co-twins) had received a diagnosis of schizophrenia based on traditionally conservative European diagnostic criteria for the illness, and most had received a hospital diagnosis of schizophrenia. Complete positive and negative symptom, paranoid phenomenology, and premorbid social adjustment ratings based on the mean of the two raters' ratings were available for 192 of the 220 schizophrenic individuals; these average ratings were used in the following analyses.

To evaluate four nested competing measurement models of the latent structure underlying positive symptoms, negative symptoms, and premorbid social relations, the confirmatory factor analysis (CFA) routine of the statistical system and computer program LISREL VI (Linear and Structural Relations VI) (Joreskog and Sorbom 1984) was used. CFA provides the most direct and informative statistical approach to evaluating competing measurement models. In CFA an investigator constructs a measurement model (i.e., a factor structure) that is derived from assumptions of the theory of interest and specifies an implied model of how covariances between a group of variables should have been caused by latent variables (i.e., underlying factors). The LISREL CFA program estimates a solution covariance matrix based on the measurement model and then, using maximum likelihood-based procedures, compares the estimated covariance matrix with the actual input covariance matrix. A measurement model producing a solution closely matching the input covariance matrix is a good fit to the data. The quality of the fit between estimated and solution matrices can be evaluated statistically; the appropriate test is the chi-square test (Long 1983; Joreskog and Sorbom 1984).

The goal of a model is to explain as much of the covariance present in the obtained data as possible within the specifications of the model. In this instance, the null hypothesis is that all of the population covariance has been extracted from the correlation matrix by the prespecified measurement model. If the chi square is statistically significant (e.g., $p < 0.05$), then the residual matrix still
has significant covariance in it and one may conclude that the model being tested does not fit the data well (Gorsuch 1983, p. 129). If the chi square is not statistically significant, then the null hypothesis is accepted and one may conclude that the prespecified model fits the observed data well, leaving little covariance in the residual matrix. When large samples are being studied, a model may provide a good fit to observed data but will generate a statistically significant chi-square value (Bentler and Bonett 1980; Marsh et al. 1988). In such instances, chi-square contrasts and incremental fit indexes are typically used to assess the relative fits of competing models (Bentler and Bonett 1980; Marsh et al. 1988). Extensive reviews and introductions to the mathematical approach to parameter estimation involved in CFA and the CFA approach to statistical comparisons of competing substantive models are readily available elsewhere (see Long 1983; Joreskog and Sorbom 1984; Hayduk 1987; Bollen 1989).

Using the data contained in table 1, we estimated four models. We began by estimating a null model (Bentler and Bonett 1980), which evaluates the fit of a model that assumes no structure underlying schizophrenic phenomenology. In this instance the null model posits that each of the 13 phenomenologic features represented an independent dimension (i.e., a "13-factor" model), thereby implying that no underlying structure related the symptoms and signs. Though clinically implausible, estimation of the null model establishes a useful baseline against which one can compare alternative models that do make assumptions concerning latent structure.

Model 1 was a basic single-factor model predicting one common process (or factor) underlying schizophrenic symptoms and premorbid social adjustment. This model is consistent with Andreasen's model of schizophrenic phenomenology (Andreasen and Olsen 1982), which predicts a bipolar (or inverse) relationship between positive symptoms and both negative symptoms and premorbid adjustment. It is also consistent with a general "vulnerability" model (Zubin 1985), which predicts direct relationships among positive symptoms, negative symptoms, and premorbid adjustment. Model 2 was a two-factor model predicting two underlying pathological processes, one consisting of positive phenomenology and the other consisting of both negative symptoms and premorbid deficits. This model is consistent with research finding that deficits in premorbid adjustment are related to negative symptoms, whereas positive symptoms are unrelated to both negative symptoms and poor premorbid adjustment (Walker and Lewine 1988). Model 3 was the three-factor model proposed by Strauss et al. (1974), predicting three independent pathological processes underlying positive symptoms, negative symptoms, and premorbid adjustment. Model 3 is also consistent with Crow's (1985, 1987) recent theoretical approach suggesting that some deficits in social relations are best distinguished from positive and negative symptoms.

Because symptom ratings in psychopathology research can be skewed in ways that may violate the statistical assumption of multivariate normality underlying LISREL computations, we conducted a completely parallel set of CFAs for all models described above using Bentler's (1985) structural equations program, EQS. EQS allows an investigator to conduct CFAs using data that may deviate from multivariate normality by employing elliptical distribution theory-based computational procedures, thereby verifying the robustness of the LISREL results.

Finally, in addition to model 3, we estimated two supplementary three-factor models to clarify the association between post-onset asociality-withdrawal and premorbid social impairment. Model 3 held that post-onset asociality-withdrawal represents a genuine negative symptom and should be associated with other negative phenomena (e.g., Andreasen and Olsen 1982). The first supplementary model placed asociality-withdrawal with the positive symptom factor, testing the possibility that this symptom may be "secondary" and closely related to positive phenomena (e.g., Crow 1985, 1987; Zubin 1985; Carpenter et al. 1988). Placement of this symptom with the premorbid social impairment factor in the second supplementary model reflected the hypothesis that post-onset asociality-withdrawal might be a post-onset extension or reemergence of premorbid adjustment difficulties (Strauss et al. 1974; Zubin 1985).

Results

Results of the LISREL analyses evaluating goodness of fit for each model are contained in the upper left side of table 2. The first model

The present sample consisted of twins diagnosed with schizophrenia and, not infrequently, both a proband and his or her affected co-twin were included. It could be argued on an a priori basis that inclusion of twin pairs concordant for schizophrenia might have weight our sample toward the more severely affected end of the schizophrenia liability continuum. Such an imbalance might be hy-
estimated, the null model, revealed a relatively large and highly significant chi square and relatively low LISREL goodness-of-fit index (a "perfect" fit between model and data would generate a fit index of 1.000). These data indicate that a model assuming no latent structure underlying schizophrenic symptomatology fits the data poorly and, in fact, they suggest the presence of latent structure is likely. Model 1, the unidimensional model, produced a smaller chi square relative to the null model and an improved goodness-of-fit index. Inspection of the factor loadings did not reveal the bipolar pattern for positive and negative symptoms predicted by Andreasen's model (Andreasen and Olsen 1982); rather, all factor loadings were positive in direction as predicted by a vulnerability model (e.g., Zubin 1985). Model 2, the two-factor model, revealed an even smaller chi square relative to both the null model and unidimensional models and yielded an improved goodness-of-fit index. Finally, model 3, the Strauss et al. (1974) model, produced the smallest chi-square value and the highest goodness-of-fit index, indicating it would be expected using a hypothetically better model, nonnormed incremental fit indexes were calculated (Tucker and Lewis 1973; Bentler and Bonett 1980; Marsh et al. 1988). The results of the LISREL-based model comparisons as well as the incremental fit indexes are contained in the upper right side of table 2. All three models that made assumptions about the underlying structure of schizophrenic phenomenology provided significantly better fits to the observed data than the null model (p < 0.001 for all comparisons).

As can be seen from table 2, model 2 provided a significantly better fit to the data than did model 1. The model contrast of central concern to the present investigation concerned models 3 and 2. Model 3 clearly provided a significantly better fit to the data than did model 2. The incremental fit indexes in the right part of table 2 are consistent with the sequential chi-square contrasts; it is most important to note that the improvement in fit provided by model 3 over model 2 is substantial (Bentler and Bonett 1980). Moreover, the cumulative increment in fit provided by model 3 over the null model is appreciable, although additional improvement in fit appears possible.

Results of the parallel EQS CFAs are contained in the lower half of table 2. The overall pattern of results based on elliptical distribution theory-based CFAs is highly similar to, and in reasonable accord with,
Table 1. Correlations among positive, negative, and paranoid symptoms and premorbid adjustment in 192 schizophrenic patients

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Note.—1 = hallucinations; 2 = delusions; 3 = positive formal thought disorder; 4 = catatonic motor behavior; 5 = bizarre behavior; 6 = flattened affect; 7 = alogia; 8 = avolition; 9 = asociality; 10 = attentional impairment; 11 = Zigler-Phillips Social Competence Scale; 12 = Phillips Abbreviated Scale of Premorbid Sexual Adjustment; and 13 = Venables-O'Connor Scale for Rating Paranoid Schizophrenia. Correlations reported are Pearson product moment correlation coefficients. Decimal points are omitted.

Discussion

Our results reveal that positive symptoms, negative symptoms, and premorbid social impairment represent three relatively independent phenomenologic domains in schizophrenia, thus providing empirical evidence to support Strauss et al.'s (1974) theoretical conjectures concerning the underlying organization of schizophrenic phenomenology hypothesized to reflect three discrete disease processes. Our results are also consistent with and support Crow's (1980, 1985, 1987) independent dual-process model, which identifies some forms of pathological social relations as distinct from the two independent dimensions of pathology reflected by positive and negative symptoms. Also, consistent with a recent summary of univariate results documenting an association between negative symptoms and premorbid impairments (Walker and Lewine 1988), our data reveal an association between the latent variables underlying these two domains. Our confirmatory analytic approach, however, demonstrates that negative symptoms and premorbid impairment in social relations are best viewed as relatively independent processes. Finally, our results clearly indicate that single-process models of schizophrenic symptoms, such as the Andreasen bipolar unidimensional model (Andreasen and Olsen 1982)
Table 2. Evaluation of symptom models using confirmatory factor analysis

<table>
<thead>
<tr>
<th>Model</th>
<th>Model test</th>
<th>Model comparison</th>
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<td>M₃</td>
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Note.—M₀ = null model; M₁ = unidimensional model; M₂ = two-dimension model; and M₃ = three-dimension or Strauss et al. three-process model. All chi-square values are statistically significant (p < 0.001).

¹GFI indicates the LISREL goodness-of-fit index (GFI values range from 0.000 to 1.000; a GFI = 1.000 would be a perfect fit between a measurement model and observed data). EQS does not produce a goodness-of-fit index.

²p_{ki} = the Tucker-Lewis incremental fit index, a measure of the degree of improvement in fit (specifically, the proportionate reduction in the fitting function) obtained when moving from a null model (or less restrictive model) to a maintained (or more restrictive) model.

³Cum = cumulative increment in fit (Tucker-Lewis formula) to the observed data by the maintained model relative to the fit provided by the null model. Using the null model as a baseline comparison model, the Tucker-Lewis incremental fit index has a minimum of 0.000 and will tend toward 1.000 for a perfect fit. See Bollen (1989) for greater detail.

and the general vulnerability model (Zubin 1985), fit actual observed symptoms rather poorly.

Our data indicate that some degree of improvement in fit over and above the Strauss et al. three-process model is possible; however, to our knowledge, no other model of schizophrenic phenomenology based exclusively on positive symptoms, negative symptoms, and premorbid social adjustment exists currently.

The two supplementary three-process models we estimated did not fit the data better than the Strauss et al. model. We also estimated several quasi-plausible four-factor models, none of which significantly improved on the fit observed for model 3. Thus, simply adding factors to a measurement model does not necessarily improve the fit of the model to observed data.

The present investigation also sought to clarify the relations between premorbid and post-onset social impairment. Our supplementary analyses suggested that post-onset asociality-withdrawal is more closely associated with other negative symptoms than with either pathological premorbid social relations or positive symptoms. Unfortunately, our data do not allow us to determine the precise etiology of post-onset asociality and social withdrawal. It is conceivable that such behavior may represent an aspect of the negative symptom domain, but it could also represent a reaction to learning that one has a psychiatric illness and the subsequent purposeful avoidance of social interaction (Link et al. 1989). Furthermore, in a previous analysis of these symptom data, one of us (R.H.D.) reported gender differences both in premorbid adjustment and in the negative symptom asociality-withdrawal (Dworkin 1990). Differences in mean levels of social functioning across gender are intriguing (see Zigler and Glick 1986) in light of the fact that the symptom covariance matrices for our male and female subjects do not differ significantly; the Strauss et al. (1974) three-process model provides the best fit in both the male and female samples taken separately. Although our data support the existence of premorbid social functioning as a third class of schizophrenic phenomenology, they clearly raise questions about...
Strauss et al.'s proposal that social functioning is a longitudinally consistent psychological process. Prospective developmental research is needed to illuminate the nature and causes of social impairments across the life-span of schizophrenic patients.

Three methodological aspects of our study should be kept in mind when our results are being evaluated. First, the symptom ratings were made from case history data. Although case histories can often be tapped profitably to extract meaningful data in psychopathology research (e.g., Spitzer et al. 1979; Strauss and Harder 1981; Loranger et al. 1982; Lenzenweger and Loranger 1989), those data obtainable from case histories and hospital charts can be subject to noteworthy limitations (see Andreasen et al. 1977). For example, the case histories we examined were not originally prepared with this research in mind, and it is possible that some histories might have omitted symptom descriptions. However, the case histories generally contained relatively rich phenomenological accounts of symptoms and premorbid social functioning. This was probably so because the histories were recorded within a European psychiatric context that emphasized careful attention to phenomenology, particularly deficit symptoms, in the diagnosis of schizophrenia (Essen-Moller 1941; Langfeldt 1953). Second, as is the case with essentially all assessment procedures in clinical psychopathology research (e.g., clinical and structured interviews, rating scales), the possibility that some degree of method-related covariation might exist among the symptom ratings used in our study could be considered.

A third feature of these data is that the patients were originally diagnosed as schizophrenic using pre-

DSM-III criteria. Although the patients were diagnosed within a European context that traditionally employed a "narrow" concept of schizophrenia, it is possible that diagnostic criteria for the illness varied across the original study research settings. We recommend, therefore, that future investigations of the models we evaluated use symptom ratings derived from structured psychiatric interviews with patients who have been diagnosed for schizophrenia using modern operational criteria. Future research, however, must attend to the possibility that the DSM-III and DSM-III-R (American Psychiatric Association 1987) definitions of schizophrenia largely exclude negative symptoms from the diagnostic criteria for the illness; this feature of the newer operational definitions may, in fact, substantially reduce observable variation in negative symptoms among study populations (see Dworkin and Lenzenweger 1984, p. 1545).

The role of neuroleptic medication in relation to the underlying structure of schizophrenic phenomenology could also be considered in statistical analyses, provided detailed and reliable knowledge of dosage regimens is available. Overall, despite the possible limitations of our data base, our results are highly consistent with an accumulating literature, which suggests that positive symptoms, negative symptoms, and social functioning reflect three relatively separate underlying processes in both the manifestation and the pathogenesis of schizophrenia (Pogue-Geile and Zubin 1988; Walker and Lewine 1988; Lenzenweger et al. 1989; Bellack et al. 1990). We offer our findings for their heuristic value and emphasize that replication of our findings is warranted and encouraged.

By translating rich, clinically based theoretical positions into testable statistical models, we were able to objectively evaluate competing views concerning the underlying organization of schizophrenic symptoms. Although our data do not address the specific etiologies of each of Strauss et al.'s three putative pathological processes, they do provide empirical support for the hypothesized relations among symptoms predicted by the three-process model. Moreover, they are consistent with Crow's (1980, 1985, 1987) dual-process model of schizophrenic phenomenology, and they support the utility of a multidimensional approach in understanding schizophrenic symptoms (Strauss et al. 1974; Dworkin et al. 1988).

Finally, while our data may be of use in theoretical discussions of schizophrenic symptoms, we suggest that they have an important clinical implication as well. As clinicians, through careful differential attention to the three relatively independent dimensions of schizophrenic phenomenology described by Strauss et al. and supported by our data, we might further refine our understanding of our schizophrenic patients and thereby better tailor our therapeutic interventions to their needs.

References


Andreasen, N.C. Scale for the Assessment of Negative Symptoms (SANS). Iowa City, IA: University of Iowa, 1981.


Langfeldt, G. Some points regarding the symptomatology and diagnosis of schizophrenia. Acta Psychiatrica et Neurologica Scandinavica, 28(Suppl. 80):7–26, 1953.


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