The Factorial Structure of the Schedule for the Deficit Syndrome in Schizophrenia

David Kimhy1–3, Scott Yale2,3, Raymond R. Goetz2,3, Lynn Marcinko McFarr4, and Dolores Malaspina2,3

2Department of Psychiatry, Box 2, Columbia University, 1051 Riverside Drive, New York, NY 10032; 3Department of Medical Genetics, New York State Psychiatric Institute; 4Department of Psychiatry, Harbor University of California, Los Angeles, Medical Center

Deficit schizophrenia (DS) is considered a distinct subtype within the diagnosis of schizophrenia. While the common assumption is that DS represents a single, cohesive domain of psychopathology, the factorial structure of DS has not been investigated. We assessed 52 individuals with DSM-IV diagnoses of schizophrenia with DS. A principal component analysis (PCA) was conducted on the symptoms of the Schedule for the Deficit Syndrome. The PCA resulted in 2 distinct factors explaining 73.8% of the variance. Factor 1 (avolition) is made up of symptoms of curbing of interests, diminished sense of purpose, and diminished social drive. Factor 2 (emotional expression) is made up of symptoms of restricted affect, diminished emotional range, and poverty of speech. The results indicate that DS is best characterized by these 2 factors. The great majority of participants (86%) displayed DS symptoms from both factors. On average, participants had 4.19 (S.D. = 1.39) symptoms that were primary, enduring, and at least moderate in severity. The mean severity of symptoms was 2.25 (S.D. = 1.06). We discuss possible links between the obtained factors and putative neurobiological mechanisms, as well as directions for future research.

Key words: schizophrenia/deficit syndrome/ factor analysis/negative symptoms/avolition/emotion

Introduction

Schizophrenia has long been characterized by significant variability in symptoms, course of illness, and clinical profiles. In an attempt to reduce the heterogeneity of this complex disorder, researchers have tried to identify homogeneous subtypes that will facilitate the identification of links between symptoms and putative neurobiological mechanisms. Among these, deficit schizophrenia (DS)1–2 has been found to be a distinct subtype within the diagnosis of schizophrenia, with DS patients differing from nondeficit schizophrenia with regard to risk factors, symptom profiles, neuropsychological functioning, family history, course of illness, treatment response, and structural and functional neurobiology.3–13 DS has been shown to have solid long-term diagnostic stability,14–15 with a prevalence of 15% among first-episode patients and up to 32% among chronic patients.5, 16 Although some of the data are consistent with the view of DS as representing the severe end of a single continuum of schizophrenia, a large body of literature on risk factors and biological features is inconsistent with this view, supporting the hypothesis of a separate disease.5

While research findings support the concept of DS as a distinct subtype of schizophrenia, it has not been determined that the symptoms of DS represent a single, cohesive domain of psychopathology, as the factorial structure of DS has not been investigated. In fact, there is some evidence pointing to the possibility that the symptoms making up DS may be independent of each other. Kelley et al. found 2 factors (diminished motivation and affective flattening) in a study of negative symptoms among 93 neuroleptic-free males with schizophrenia.17 Sayers et al. used exploratory and confirmatory factor analyses to identify the factor structure of negative symptoms as assessed by the Scale for the Assessment of Negative Symptoms within a large sample of schizophrenia patients (N = 457).18 They found 3 factors: diminished expression, inattention/alogia, and social amotivation. However, there was a high interfactor correlation (r = .83) between the diminished expression and inattention/alogia factors.

Thus, the aim of this study is to investigate the factor structure of DS. Based on a review of the 6 symptoms of the Schedule for the Deficit Syndrome2 and previous factor-analytic studies of negative symptoms,17–18 we tested the hypothesis that DS may have 2 distinct factors: a factor relating to volition and a second factor relating to affect. Our belief is that clear delineation of specific areas of psychopathology will facilitate the discovery of links...
between symptoms and putative neurobiological mechanisms, possibly indicating that these phenomena are separate targets for treatment studies.

Methods

Participants

Fifty-two individuals with DSM-IV diagnoses of schizophrenia with DS were recruited from an inpatient research unit at the New York State Psychiatric Institute. The mean age of the sample was 32.86 years (S.D. = 9.95), with a range from 18 to 55 years. Eighty-six percent (N = 45) were men. Thirty-eight percent (N = 20) were Caucasian, 38% (N = 20) were African American, 14% (N = 7) were Hispanic, and 10% (N = 5) were Asian. The mean duration of illness was 12.60 years (S.D. = 9.37). The mean ages of onset of first emotional problems and first psychotic symptoms were 19.54 (S.D. = 6.66) and 20.34 years (S.D. = 5.04), respectively. Participants had on average 11.92 years (S.D. = 3.30) of education. Participants received either typical or atypical antipsychotics, depending on the protocol being conducted when the patient was studied.

Inclusion and Exclusion Criteria

Included were participants who met the following criteria: (1) aged 18 or older, (2) a DSM-IV diagnosis of schizophrenia disorder, and (3) identified as having DS. Excluded were individuals who (1) were not fluent in English, (2) were unable to give informed consent, and (3) had schizoaffective disorder or any nonaffective psychosis other than schizophrenia. The study was approved by the Institutional Review Board, and all participants provided written informed consent.

Diagnosis

All participants were interviewed by master’s-level or above clinicians. Diagnosis was assessed using the Diagnostic Interview for Genetic Studies,19 a structured diagnostic interview and review of medical records that is used to gather demographic, diagnostic, and course of illness information for the major affective, psychotic, and substance use DSM-IV Axis I disorders. Diagnoses were achieved by consensus among clinical and research staff based on the interviews, past medical records, and symptom ratings.

Deficit Schizophrenia Criteria

DS was identified by the Schedule for the Deficit Syndrome (SDS)2—a semistructured interview. DS, as defined by the SDS, is identified by the presence of 2 or more negative symptoms with at least moderate severity that have been determined to be both primary (i.e., not caused by neuroleptic akinesia, depression, anxiety, paranoia, or other psychotic symptoms) and enduring (present during the preceding 12 months, including during periods of clinical stability). Severity of symptoms is rated on a 5-point scale ranging from symptom not present (0), through moderate (2), to very severe (4). The primacy and stability of symptoms are rated categorically (present/absent).

Two raters (S.Y. and L.M.M.) were trained by the primary developer of the SDS (Brian Kirkpatrick, M.D., Maryland Psychiatric Research Center). The training consisted of reading the SDS manual, attending a lecture, viewing videotaped SDS interviews of 3 subjects, and co-rating (with B.K.) 7 patients during live interviews. The 3 raters agreed 100% on the deficit and nondeficit status of these 10 patients, with 5 being classified as having DS.

Results

Item Distributions

The SDS data from 52 participants were analyzed using SPSS version 12.0.1. On average, participants had 4.19 (S.D. = 1.39, range 2–6) symptoms that were primary, enduring, and at least moderate in severity. The mean severity of all symptoms was 2.25 (S.D. = 1.06), indicating a predominance of moderately severe symptoms. This mean is above the SDS severity criteria of 2 (moderate) for symptoms to be included for consideration for DS. A total of 5 out of the 6 SDS symptoms had a mean severity rate of 2 or higher. The only symptom with a mean smaller than 2 was poverty of speech, which was also the least prevalent symptom (41% of participants). Diminished emotional range was the most prevalent symptom that met DS criteria (80% of participants). The distributions of the SDS symptoms based on severity are presented in table 1.

Principal Components and Factor Analysis

Principal component analysis (PCA) using varimax rotation with Kaiser normalization was performed on the total score for each of the 6 SDS symptoms. The PCA resulted in 2 distinct and interpretable factors explaining 73.8% of the variance. The Kaiser-Meyer-Olken sampling adequacy measure was 0.70, indicating that the data were adequate for factor analysis. Our choice of using the varimax procedure was influenced by the aim of this study—to identify distinct categories of symptoms that will elucidate the clinical picture of DS. Thus, a statistical technique that accentuates differences between factors appeared more suitable. Table 2 shows the principal component structure after the components were rotated using varimax rotation. The number of factors extracted was based on the eigenvalue $\geq 1$ criterion. Symptoms were assigned to factors based on their highest level of loading. The results of the factor analysis suggest that DS has 2 distinct and interpretable factors. Factor 1
(avolition) comprises symptoms of curbing of interests, diminished sense of purpose, and diminished social drive. Factor 2 (emotional expression) comprises symptoms of restricted affect, diminished emotional range, and poverty of speech. None of the symptoms loaded heavily ($0.40$) on more than 1 factor. Coefficient alphas were computed to obtain internal consistency estimates of reliability for the 2 factors. The alphas for both factors were 0.78.

Fourteen participants (26%) had all 6 SDS symptoms meeting DS criteria as primary, enduring, and at least moderate in severity. Four participants (8%) had only avolition symptoms that met DS criteria. Three participants (6%) had only emotional expression symptoms that met DS criteria. Table 3 shows the distribution of SDS symptoms (primary, enduring, and at least moderate in severity) based on factor.

### Discussion

**Factor Structure and Symptoms**

This factor-analytic study supports our hypothesis that DS is made up of 2 distinct factors—avolition and emotional expression. To our knowledge, this is the first investigation of the factor structure of DS. As our goal was to assess the factor structure of DS, the PCA included only participants who met overall criteria for DS. While participants had on average more than 4 deficit symptoms that were primary, enduring, and at least moderate, their symptoms that did not meet these criteria were also included in the PCA. However, the inclusion of such symptoms did not appear to drastically influence the factor structure. In fact, Kelley et al. also found 2 similar factors (diminished motivation and affective flattening) in a study of negative symptoms among 93 neuroleptic-free males with schizophrenia.17 Further support for the validity of the symptoms can be found in the Cronbach’s alphas of each factor. Alphas greater than 0.70 are considered acceptable.20 The obtained alphas of 0.78 for each

### Table 1. Characteristics and Distribution of Schedule for the Deficit Syndrome Symptoms Based on Symptom Severity

| Symptom                        | Severity | % Met Deficit Schizophrenia Criteria | % With Primary Symptoms | % With Enduring Symptoms | % With Symptom Severity | Very Severe | Severe | Moderate | Mild |Absent |
|--------------------------------|----------|-------------------------------------|-------------------------|--------------------------|--------------------------|-------------------------|---------|---------|---------|------|-------|
| Curbing of interest            | 2.19     | 67                                  | 71                      | 6                        | 4                        | 12                      |         |         |         |      |       |
| Diminished sense of purpose    | 2.42     | 76                                  | 80                      | 18                       | 25                       | 4                      |         |         |         |      |       |
| Diminished social drive        | 2.75     | 78                                  | 82                      | 23                       | 25                       | 10                      |         |         |         |      |       |
| Restricted affect              | 2.27     | 73                                  | 74                      | 8                        | 39                       | 18                      |         |         |         |      |       |
| Diminished emotional range     | 2.23     | 80                                  | 84                      | 8                        | 47                       | 8                      |         |         |         |      |       |
| Poverty of speech              | 1.63     | 41                                  | 46                      | 3                        | 18                       | 35                      |         |         |         |      |       |

**Table 2. Factor Loadings for Schedule for the Deficit Syndrome Symptoms**

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Avolition</th>
<th>Emotional Expression</th>
</tr>
</thead>
<tbody>
<tr>
<td>Variance extracted (%)</td>
<td>37.1</td>
<td>36.7</td>
</tr>
<tr>
<td>Eigenvalue</td>
<td>2.23</td>
<td>2.20</td>
</tr>
<tr>
<td>Coefficient alpha (α)</td>
<td>0.782</td>
<td>0.776</td>
</tr>
<tr>
<td>Curbing of interests</td>
<td>.916</td>
<td></td>
</tr>
<tr>
<td>Diminished sense of purpose</td>
<td>.916</td>
<td></td>
</tr>
<tr>
<td>Diminished social drive</td>
<td>.607</td>
<td>.388</td>
</tr>
<tr>
<td>Restricted affect</td>
<td></td>
<td>.917</td>
</tr>
<tr>
<td>Diminished emotional range</td>
<td></td>
<td>.884</td>
</tr>
<tr>
<td>Poverty of speech</td>
<td>.383</td>
<td>.641</td>
</tr>
</tbody>
</table>

**Table 3. Distribution of Schedule for the Deficit Syndrome Symptoms That Met Deficit Schizophrenia Criteria Based on Factor**

<table>
<thead>
<tr>
<th>Number of Participants</th>
<th>Number of Participants Displaying Avolition Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>0  1  2  3  Total</td>
</tr>
<tr>
<td>1</td>
<td>0  1  2  3  6</td>
</tr>
<tr>
<td>2</td>
<td>3  4  5  8  20</td>
</tr>
<tr>
<td>3</td>
<td>3  0  2  14  19</td>
</tr>
<tr>
<td>Total</td>
<td>6  5  12  29  52</td>
</tr>
</tbody>
</table>

**Note:** N = 52. Extraction: Principal component analysis with varimax rotation. Loadings $<.35$ are not printed for clarity. Bold indicates primary symptom loadings for each factor.
factor suggest that the internal consistency of the factors is good.

The symptom distributions in our sample indicate that the majority of participants received ratings of at least moderate severity on most symptoms, suggesting significant and enduring deficits in numerous areas of functioning. The great majority of participants (86%) displayed symptoms from both factors that are primary, enduring, and at least moderate in severity, including 14 participants (26%) who displayed all 6 SDS symptoms meeting DS criteria. More than half (56%) of the participants displayed all 3 avolition symptoms meeting DS criteria, while about a third (36%) displayed all 3 emotional expression symptoms meeting criteria.

**Putative Neurobiological Underpinnings**

Our results invite speculations about the neurobiological underpinnings associated with each SDS factor. Results from neurocognitive, postmortem, and functional and structural imaging studies suggest dysfunction in the dorsolateral prefrontal cortex (DLPFC) and the inferior parietal cortex (IPC) as the neural basis of DS.

The DLPFC has been implicated as a candidate region associated with volition and motivation. Damage to the DLPFC in humans leads to lack of spontaneous activity and repetitive, stereotypic use of inappropriate behavioral responses. Patients with lesions in the DLPFC have been reported to show difficulties in initiating action sequences. Thus, it would be reasonable to assume a link between the symptoms of the avolition factor and neural circuitry in this region.

The neurobiological underpinnings of the emotional expression factor appear to be more complex. Restricted affect refers to diminished vocal, facial, and gestural expression of emotions. Diminished emotional range refers to limited subjective emotional experience. Poverty of expression refers to the limited production of both words and information by the subject. DS patients have been found to have more restricted affect than nondeficit (ND) patients when presented with emotional stimuli in the form of affectively valanced film clips. Similarly, Bryson et al. used videotaped monologues to assess recognition of affect and found that DS patients performed more poorly than ND patients. However, DS patients did not report experiencing less emotional response to films compared to ND patients. Horan and Blanchard were not able to replicate these findings, possibly due to different methodology (black-and-white photographs of faces). Thus, the affective disturbance in DS appears to be primarily expressive and receptive, rather than experiential. Difficulties in the outward facial expression of emotions have been linked with dysfunction in the IPC. Thus, it would be reasonable to assume a link between restricted affect in DS and neural circuitry in this region. On the other hand, the ability to perceive affect in others has been associated with activation of the prefrontal cortex. In particular, the DLPFC appears to play a central role in the modulation of attention, including to emotionally laden information. Thus, a link between the emotional expression factor and neural circuitry in this region is plausible.

**Limitations**

One limitation of the present study is the relatively modest sample size (N = 52). As a rule, a reliable PCA requires a minimum participant/variable ratio of 10:1. In this study the ratio was 8.7:1. Thus, given the sample size and possibility that the resulting factor structure may not be reliable, these results need to be replicated with a larger sample.

**Summary and Future Directions**

DS is considered a distinct subtype within the diagnosis of schizophrenia. The major finding of the present study is that the factor structure of DS consists of 2 distinct factors—avolition and emotional expression. Future studies should aim to replicate this factor structure. Additionally, future studies should aim to confirm the putative link between these factors and neurobiological underpinning in the prefrontal cortex and IPC. It will be important to use in vivo imaging techniques to support or disprove this putative link.

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