Looking at the Schizophrenia Spectrum Through the Prism of Self-disorders: An Empirical Study

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Nonpsychotic anomalies of subjective experience were emphasized in both classic literature and phenomenological psychiatry as essential clinical features of schizophrenia. However, only in recent years, their topicality with respect to the construct validity of the concept of the schizophrenia spectrum has been explicitly acknowledged, mainly as a consequence of the increasing focus on early detection and prevention of psychosis. The current study tested the hypothesis of a specific aggregation of self-disorders (SDs, various anomalies of self-awareness) in schizophrenia-spectrum conditions, comparing different diagnostic groups; 305 subjects, previously assessed in the Copenhagen Schizophrenia Linkage Study, were grouped into 4 experimental samples, according to their Diagnostic and Statistical Manual of Mental Disorders (Third Edition Revised) main diagnosis: schizophrenia, (n = 29), schizotypal personality disorder (n = 61), other mental illness not belonging to the schizophrenia spectrum (n = 112), and no mental illness (n = 103). The effect of diagnostic grouping on the level of SDs was explored via general linear model and logistic regression. The diagnosis of schizophrenia and schizotypy predicted higher levels of SDs, and SDs scores were significantly different between spectrum and nonspectrum samples; the likelihood of experiencing SDs increased as well with the diagnostic severity. The findings support the assumption that SDs are a discriminant psychopathological feature of the schizophrenia spectrum and suggest their incorporation to strengthen its construct validity, with potential benefit for both early detection and pathogenetic research.

Key words: schizophrenia-spectrum/psychosis/anomalous self-experience/vulnerability phenotype/diagnosis

Introduction

The notion of self is absent in the current descriptions and diagnostic criteria for schizophrenia (International Classification of Diseases, Tenth Revision [ICD-10], Diagnostic and Statistical Manual of Mental Disorders [Fourth Edition, Text Revision]). In fact, the notions of self, self-awareness (self-consciousness), and sense of identity have largely disappeared from the mainstream psycho-pathological vocabulary.

This descriptive lacuna is rather mysterious because profound and various transformations of the sense of self have been noted for long in schizophrenia and its spectrum conditions, ie, schizoidia, replaced by schizotypy (for a detailed review, see Parnas and Handest1 and Parnas et al5). The anomalies of the sense of self were considered as fundamental or core subjective clinical manifestations of schizophrenia since its earliest descriptions (see Sass and Parnas3 and Parnas and Handest1). Indeed, the Kraepelinian metaphor of “orchestra without a conductor,” which pointed to the “loss of inner unity” of consciousness, was echoed in the writings of nearly all prominent classic schizophrenia researchers.4–6

Thus, Bleuler4 observed that “the most manifold alterations” occurred to the patient’s ego, including splitting of the self and loss of feeling of agency or of the mastery to direct thoughts, pointing to a “basic disorder” of personality. Most notably, however, Berze5 (an Austrian psychiatrist) proposed that a subtle alteration of self-consciousness (“primary insufficiency”), ie, a peculiar and pervasive experience of diminished transparency and affectability of awareness, was the primary disorder of schizophrenia, a claim that Berze backed up with quite impressive empirical data. Fine-grained explorations of the pathology of the self were also performed in phenomenological psychiatry.6–10 However, these contributions remained virtually unknown on the international stage and were never referred to in the scientific debate, despite an arrival of uniquely original Anglophone studies in the 1960s and 1970s.11–14

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The marginal status of subjective experience as a topic of psychopathology was due to a confluence of factors: scarcity of translations into English; a mainstream, one-sided focus on operationally “objective” behavioral terms (with a corresponding neglect of the subjective); and a wide-ranging, yet inexpressive or tacit, heuristic impoverishment of psychopathology and its concepts to the level of a subsidiary, descriptive glossary modeled after diagnostic criteria (see Parnas et al15 and Andreasen16 for a comprehensive overview).

The recent revival of psychopathological interest in the subjective aspects of schizophrenia coincided with an increasing interest in early detection and treatment of the illness.17 These efforts exposed an insufficiency of a pure behavioristic framework and stimulated an increasing international recognition of the work by Huber, Gross, and Klosterkötter and their successors in Germany, who have been pursuing systematic studies of subjective experience in schizophrenia for several decades.18–23 This research established that anomalous subjective experiences (ASEs)—which are aberrations of experience of nonpsychotic intensity and quality (ie, nondelusional, nonhallucinatory) in the domain of affect, perception, cognition, acting, and body—are important and early phenotypes of schizophrenia.24–31 ASEs are detailed in an interview schedule, the Bonn Scale for the Assessment of Basic Symptoms—BSABS32 (translated and published in Danish by our group in 1995), and a more recent, abridged English language version, the Schizophrenia Proneness Instrument Adult version.33

Recent years’ growing multicenter empirical evidence seems to confirm that ASEs occur in various schizophrenia-related conditions (ie, full-blown schizophrenia, prodromal conditions, schizotypy, and among the subjects at high genetic risk).26–29,34–42

Our own approach—in the continuation of American-Danish high-risk and adoption studies, and, more recent, genetic studies43–48—has been concerned with expressive and experiential features characteristic of schizophrenia and schizotypy (“fundamental” in Bleuler’s sense or, in a more contemporary language, core features). We focused—continuously remaining anchored in clinical work—on what might considered as an expanded subset of ASEs (basic symptoms), namely, that of self and its pathology, such as abnormal self-awareness and self-experience (SDs = self-disorders). Naturally, the concept of self (more generic than that of ASEs that are basically considered as contingent independent symptoms) requires a psychopathological-phenomenological and philosophical grounding beyond a lay understanding1,3 (for detailed epistemological considerations, see Parnas et al15). However, in the empirical context, we use the concept of SDs, not as an inference or some formal construct but as expressing real complaints of real patients: eg, fear of dissolving or losing first-person perspective, lack of basic sense of identity, feeling of one’s privacy of consciousness becoming compromised and somehow accessible to others, etc. As it is the case with ASEs, we deal here with lived experiences (“Erlebnisse”) and not with reality judgments (these phenomena are not in themselves delusional). In a study succeeding the pilot data, we showed that SDs discriminated between ICD-10 schizophrenia in remission (elevated levels) compared with psychotic bipolar illness in remission.39 A prospective longitudinal study of 155 first-admission cases demonstrated that SDs aggregated selectively among the ICD-10 nonaffective psychotic patients (mainly schizophrenias) and in the patients with schizotypal disorders but not in the diagnostic categories outside the spectrum.41,42

However, with respect to the validation of SDs as a core feature of schizophrenia-spectrum disorders, there are still several critical drawbacks. First, the results of early detection studies may be difficult to interpret. For pragmatic reasons, early detection intervention programs are guided by a prenosological risk stratification, in which the increased risk is compiled as a new, paradiagnostic category. Obviously, the stratification of “at-risk” populations is actuarially assisted, and the resulting psychopathological profiles are therefore influenced in by geographically and culturally determined local referral habits and processes (service availability, socioeconomic factors, structure of the health system, who does makes a referral, on which behavioral criteria, etc). Consequently, the reported transition rates to schizophrenia may vary quite dramatically across time and space.49,50

On the other hand, the studies dealing with explicitly classified clinical conditions (and thus framed within a nosological schema) suffer from a narrowing of the sampling focus on the full-blown schizophrenic conditions and a preeminent emphasis on affective disorders as a preferable control condition. Moreover, the data are available on patients only, ie, there is no information on the (untreated) cases of schizophrenia and schizotypal disorders with no contact to a health system. Furthermore, the shortage of data concerning SDs in schizotypal disorder (schizotypal personality disorder [SPD]) samples prevents any empirically supported inference with respect to their relevance for the schizophrenia spectrum.

The current study was undertaken in order to validate the status of SDs as basic subjective phenotypes of the schizophrenia-spectrum disorders (schizophrenia and schizotypy) through an interdiagnostic design. More specifically, our hypothesis was that the distribution of SDs will mirror the pattern detected in our sample of 155 first-admission cases41. Schizophrenia and SPD would demonstrate comparable and elevated levels of SDs, significantly higher than those of other (nonspectrum) diagnostic groups or of individuals with no psychiatric diagnosis.

SDs’ lifetime prevalence was evaluated across 4 diagnostic groups, ie, schizophrenia, SPD, other mental illness (OMI), and a control sample without recognizable clinical psychopathology.
Methods

Participants

The current study explores psychopathological data collected in the Copenhagen Schizophrenia Linkage Study (CSLS, n = 618; see Matthysse et al48 and Vaever et al51). The study was guided by a hypothesis that extended pedigree information—comprising phenotypes such as schizotypal disorder and markers such as Thought Disorder Index32,53 and eye tracking dysfunction54,55 might substantially contribute to mapping the alleles implicated in schizophrenia. The study targeted 6 families, each including at least 2 affected members with Diagnostic and Statistical Manual of Mental Disorders (Third Edition Revised) (DSM-III-R) diagnosis of schizophrenia. The study began in 1989 as an outgrowth of the Copenhagen High-Risk Study.47 Each family was horizontally and vertically extended into rather large pedigrees (ie, families with many siblings, aunts, cousins, etc [see genograms in Vaever et al51]).

All CSLS subjects, who were personally interviewed (n = 347) and on whom complete psychopathological data on SDs were available, were included in the current protocol (n = 305). The sample was divided into 4 diagnostic groups in order to compare the SDs scores between Axis I (psychotic) schizophrenia-spectrum conditions (sample 1, schizophrenia), Axis II (nonpsychotic) schizophrenia-spectrum conditions (sample 2, SPD), OMI, not belonging to the schizophrenia spectrum (sample 3), and controls with no mental illness (NMI, sample 4). We thus repeated a diagnostic classification performed in an earlier study on the distribution of SDs among 155 first-admission cases,41 a study that inspired the current investigation. The assignment to sample 1 or sample 2 within the schizophrenia spectrum was based on the primary lifetime diagnosis representing the most severe diagnosis according to DSM-III-R criteria and the relevant Axis I/Axis II distinction. Hence, subjects with SPD and concomitant Axis I nonaffective psychotic conditions were included in group 1.

Diagnostic Groups

Individuals with either an Axis I nonaffective psychotic diagnosis or an Axis II SPD/schizoid personality disorder diagnosis were considered to be members of the spectrum. Thus, the “schizophrenia” group in the current study includes individuals with the following DSM-III-R diagnoses: (a) schizophrenia (n = 19) and (b) delusional disorder, brief reactive psychosis, and psychotic disorder not elsewhere classified but only with a simultaneous Axis II diagnosis of SPD (n = 10). The “SPD” group includes 61 individuals with no Axis I diagnosis and SPD as principal Axis II diagnosis. The “OMI” group includes any individual with an Axis I or Axis II diagnosis that was not in the schizophrenia spectrum (n = 112). The participants allocated in this group exhibited a wide variety of diagnoses, such as affective disorders, alcohol abuse, and eating disorders. Finally, the “NMI” group includes family members with no diagnoses on either Axis I or Axis II (n = 103). The groups are shown in table 1.

Measures

Psychopathological Assessment. Detailed diagnostic and psychopathological assessment was performed during the CSLS.51 Two senior clinicians blind to any diagnostic information, clues to the kinship status, and the surname of the subjects administered the Copenhagen Interview of Functional Illness56 to all the enrolled participants. The Copenhagen Interview of Functional Illness is an extensive semistructured diagnostic interview composed of a compilation of existing psychiatric questionnaires (Danish versions), as well as additional items that were either theoretically motivated or influenced by our experience in the original high-risk study. The interview contained the psychosis section from the Present State Examination, 10th edition57; an abbreviated Personality Disorder Examination (PDE)58; the Thought, Language, and Communication Scale59; Scales for the Assessment of Positive and Negative Symptoms60,61; Schedule for Affective Disorder and Schizophrenia—Life Time Version

Table 1. Characteristics of the Study Sample

<table>
<thead>
<tr>
<th>Statistic</th>
<th>N</th>
<th>Participants</th>
<th>Schizophrenia</th>
<th>Schizotypal Personality Disorder</th>
<th>Other Mental Illness</th>
<th>No Mental Illness</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender</td>
<td></td>
<td>Male/female ratio</td>
<td>141/164</td>
<td>5/24</td>
<td>29/32</td>
<td>50/62</td>
</tr>
<tr>
<td>% Male</td>
<td></td>
<td>46.2</td>
<td>17.2</td>
<td>47.5</td>
<td>44.6</td>
<td>55.3</td>
</tr>
<tr>
<td>Age, y</td>
<td></td>
<td>Mean</td>
<td>41.1</td>
<td>43.6</td>
<td>36.4</td>
<td>39.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD</td>
<td>16.6</td>
<td>18.9</td>
<td>14.7</td>
<td>15.0</td>
</tr>
<tr>
<td>Duration of illness, y</td>
<td></td>
<td>Mean</td>
<td>15.5</td>
<td>21.5</td>
<td>18.3</td>
<td>14.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>SD</td>
<td>13.4</td>
<td>14.7</td>
<td>14.3</td>
<td>14.3</td>
</tr>
</tbody>
</table>

$\chi^2 = 13.40$ (P = .004) F (Welch) = 4.34 (P = .006) F (Welch) = 1.44 (P = .247)
(items concerning all non-psychotic disorders); a list of basic symptoms corresponding to sections C and D from the BSABS; and single interview items used in the Copenhagen adoption and high-risk studies

Diagnosis. DSM-III-R “lifetime” psychiatric diagnoses of each participant were determined by the consensus among 4 research psychiatrists/psychologists using all available data sources. Interrater diagnostic reliability was assessed and prevented from drift via group interview sessions (interviews conducted by J.P.). The mean interrater reliability ranged between 0.900 and 0.956, similar to that reported in Parnas et al.

SDs Score. An SDs Scale was generated a priori to pick up the anomalies of subjective experience, that relate both empirically and theoretically, to a disturbed sense of core self in its manifold expressions (disorders of self-awareness or self-experience). The specific scale items (especially those from PDE and BSABS) were thus selected on the basis of their phenomenological affinity with the characteristics, not yet psychotic, morbid self-experiences that we have been investigating, clinically, for many years and, empirically, not yet psychotic, morbid self-experiences (disorders of self-awareness or self-experience). The specific scale items (especially those from PDE and BSABS) were thus selected on the basis of their phenomenological affinity with the characteristics, not yet psychotic, morbid self-experiences that we have been investigating, clinically, for many years and, empirically, in recent studies. In phenomenological terms, these experiences point to a diminished sense of being a self-coinciding pole of identity, spontaneity, and agency and encompass the decrease in first personal aspects of mental contents (which become more anonymous), the decrease of fluidity and transparency of the stream of consciousness, and a difficulty in world immersion and in the natural grasping of previously familiar meanings. Typical subjective reports are “I never feel fully awake, it is as if my head is constantly filled with a fog,” “It feels as if my point of view on the world sometimes shifts few centimetres backwards,” “I have to constantly monitor my thoughts,” “Thinking is simply going on in my head, with me as a spectator,” and “During a conversation, I lose the sense of whose thoughts initiate in whom.” Detailed clinical descriptions with verbatim quotations are provided elsewhere (see Parnas and Handest and Parnas et al).

The items were originally coded as 0 (not present), 1 (doubtfully present), or 2 (definitely present). However, because the majority of the participants only received scores 0 or 2, the score of 1 was recoded into 0 (not present) and 2 redefined as 1 (present), a recoding performed for all items.

Detailed composition of the SDs Scale is shown in Appendix 1. Its \( \alpha \) coefficient of internal coherence was at a very good level and reaffirmed a required dimensionality. Removing any single item from the scale would not substantially increase the \( \alpha \) coefficient. The SDs score for each subject was calculated as a sum of ratings of the individual scale items (which had values 0 or 1).

Data Analyses

We explored the sociodemographic features of the samples using \( \chi^2 \) test for categorical variables and Welch weighted analysis of variance for continuous variables.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>B</th>
<th>95% CI</th>
<th>SE</th>
<th>t</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenia</td>
<td>5.60</td>
<td>4.06 to 7.15</td>
<td>0.79</td>
<td>7.13</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>SPD</td>
<td>4.20</td>
<td>3.01 to 5.38</td>
<td>0.60</td>
<td>6.97</td>
<td>&lt;.001</td>
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<tr>
<td>OMI</td>
<td>1.20</td>
<td>0.21 to 2.20</td>
<td>0.51</td>
<td>2.38</td>
<td>.018</td>
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Note: CI, confidence interval; SPD, schizotypal personality disorder; OMI, other mental illness; NMI, no mental illness.

Bonferroni bounds–adjusted paired multiple comparisons: schizophrenia, SPD > OMI, NMI.

*Reference category.

bMale as reference category for sex.

To estimate the effect of diagnostic grouping on SDs score, we used the analysis of covariance (general linear model [GLM] procedure in SPSS). In a second step—with the logistic regression—we analyzed the predictive power of the adjusted model with respect to the presence/absence of SDs as outcome variable.

Results

The demographic features of the 4 experimental samples are presented in table 1. The groups differ with respect to sex distribution and mean age, whereas there is no significant difference of the duration of the illness among the clinical subsamples (ie, schizophrenia, SPD, OMI).

The GLM with SDs score as outcome measure, clinical grouping as fixed factor, and gender and age as covariates is presented in table 2. The GLM reveals a significantly different effect of the 4 clinical groups on the SDs score. Among the demographic covariates, age has a significant negative association with SDs.

Post hoc analysis (Bonferroni bounds–adjusted paired multiple comparisons between all the 4 levels of the factor diagnostic grouping) confirmed significant differences between spectrum vs nonspectrum samples: schizophrenia = SPD > OMI = NMI.

The logistic regression model, predicting the presence or absence of SDs as outcome variable, showed good fit (goodness-of-fit test, \( \chi^2 = 80.4, df = 5, P < .0001 \)) and a Nagelkerke approximation of \( R^2 = 0.31 \). Table 3 shows that the more the diagnosis is severe with respect to the schizophrenia spectrum, the higher is the odds ratio for SDs. Specifically, given NMI as reference group, the chances of experiencing at least one symptom of the SDs Scale are almost 3 times higher in OMI, 11 times higher in SPD, and 21 times higher in schizophrenia.

Table 2. General Linear Model: Effects of Diagnosis on Self-disorders Score

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Diagnosis

<table>
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<th>Diagnosis</th>
<th>Odds Ratio</th>
<th>95% Confidence Interval</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schizophrenia</td>
<td>20.97</td>
<td>6.82 to 64.46</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Schizotypal personality disorder</td>
<td>11.12</td>
<td>5.14 to 24.06</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Other mental illness</td>
<td>2.60</td>
<td>1.38 to 4.87</td>
<td>.003</td>
</tr>
<tr>
<td>No mental illness*</td>
<td>1</td>
<td>1.00 to 1.00</td>
<td></td>
</tr>
</tbody>
</table>

Sociodemographics

| Age, y         | 0.98    | 0.96 to 1.00 | .004 |
| Sex (female)   | 1.10    | 0.65 to 1.87 | .722 |

*aReference category.

*bMale as reference category for sex.

The effect of age remains statistically significant—as in the GLM—but with a relatively small effect on the odds ratio per each unit increase.

Discussion

Our findings confirm that subjects with a schizophrenia-spectrum diagnosis exhibit higher degrees of qualitatively similar ASE (SDs) when compared with clinical nonspectrum samples and with healthy controls. Thus, the SDs seem to express a potentially important trait phenotype for the clinical characterization of the schizophrenia spectrum.

Furthermore, our findings suggest that schizophrenia and SPD share comparable levels of nonpsychotic experiential anomalies and thus confirm a similar, previously reported pattern observed among 155 first-admission patients with spectrum and nonspectrum diagnoses. This similarity provides an additional validity to the schizotypal disorders as belonging to the schizophrenic spectrum and strengthens the conceptual salience of nonpsychotic anomalies of self-experience for the construct validity of the schizophrenia-spectrum notion. Indeed, the anomalies of self-experience, once explicitly considered of intrinsic relevance for the diagnosis and a comprehensive grasp of schizophrenia gestalt, sank into oblivion with the construction of contemporary diagnostic criteria, yielding to overriding concerns about reliability and a generally behavioristic bent of psychopathology.

Moreover, recent empirical work has rediscovered these phenomena, emphasizing anomalies of subjective experience as a fundamental, generative feature of symptomatology of schizophrenia-spectrum conditions. This aspect is of substantial importance for the issues of early identification of subjects at risk of transition to psychosis. Indeed, nonpsychotic disorders of subjectivity are increasingly acknowledged as potentially useful clinical target features for the purpose of early detection, particularly as a way to supplement the ultrahigh risk identification approach (based on the notion of attenuated or intermittent psychotic symptoms).

In this respect, it seems to us that SDs are mainly etiologically informative as early markers of clinical vulnerability but whose specificity is constituted or articulated through their position in a certain characteristic psychopathological gestalt. Therefore—to be of any clinical and heuristic use—SDs should not be considered (and assessed) as a contingent aggregate of otherwise mutually independent, atomized, and decomposable anomalous features/experiences but precisely as a coherent psychopathological structure indicative of a profound distortion of subjectivity as a whole. Also—given the peculiar nature of our experimental sample (ie, individuals at familial risk for schizophrenia-spectrum disorders)—the sort of vulnerability subtended by SDs might be more pertinently conceived as a broad vulnerability to the development of schizophrenia-spectrum conditions (ie, a “spectrum proneness” encompassing both psychotic and nonpsychotic conditions such as SPD), rather than as a more specific proneness toward the development of psychosis (defined in terms of full-blown psychotic symptoms) as such; therefore, the primary relevance of SDs would be in terms of etiological research in the genetic architecture of schizophrenia, and, only secondarily, it could serve as a further marker of lifetime risk of psychosis.

Some limitations of the current study need to be taken in account in interpreting the results. First, we use data collected in the CSLS. The latter included participants belonging to extended family pedigrees of probands with a diagnosis of DSM-III-R schizophrenia. The experimental sample is therefore by definition representative of a genetically high-risk population for the development of schizophrenia-spectrum disorders. However, this feature should reduce, rather than amplify, the quantitative differences in SDs across the diagnostic categories. Second, given the circumscribed hypothesis testing of the current study, which is not addressing/endorsing any specific genetic model of schizophrenia, we abstained from using the position of subjects in the genograms. Indeed, a simple stratification (first degree, second degree, etc) would not work due to the complexity of the pedigrees. We also refrained from applying an algorithm for a total computation of each family member’s true genetic vulnerability by taking into account his unique position in the genogram and hence all his affected relatives, close and distant (see Lawrie et al).

Third, the assessment of SDs was performed by aggregating available psychopathological items (see Appendix I), which does not exhaust the clinical richness, manifold and varying articulation of the disorders of self-awareness, as recently described in the Examination of Anomalous Self-experience scale. Finally, our assessments address only the lifetime prevalence of SDs, with no information concerning their degree of intrusiveness, pervasiveness, frequency, and temporal coaggregation, information that could be relevant for further distinctions into differential
patterns, at least within the schizophrenia-spectrum conditions. In a broader research perspective, it is to be pointed out that the study is based on a population sample, which is mainly nonclinical and only diagnosed in the contexts of a genetic research protocol. Thus, the diagnoses are independent of any contact with treatment facilities (and, eg, chronicity), and the clinical expressivity is not confounded by psychopharmacologic and other treatments.

In this respect, the results (1) confirm and further generalize the schizophrenia spectrum discriminating diagnostic power of SDs, previously reported within clinical samples\(^{36,41,42}\) and (2) prompt a promising phenotypical model suitable for further testing in the genetic research on the architecture of the vulnerability to schizophrenia.

**Conclusion**

In summary, the data point to an overall specific aggregation of SDs in schizophrenia-spectrum conditions and corroborate the internal coherence of the schizophrenia spectrum across *DSM-III-R* (and following versions) Axis I and Axis II. Indeed, elevated levels of nonpsychotic anomalies of subjective experience characterize both schizophrenia and SPD groups and distinguish them from nonspectrum clinical (ie, OMI) and nonclinical (ie, NMI) conditions.

Besides the implication for the validity of the schizophrenia-spectrum construct, the results further confirm the clinical importance of experiential anomalies for refining the identification of spectrum phenotypes both in early detection and in genetic research. Finally, the study provides the rational background for further investigation of SDs as a dimensional trait, potentially informative of the transgenerational latent vulnerability to the schizophrenia-spectrum disorders.

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