Validating Schizotypal Personality Disorders: Problems With the Schizophrenia Connection

by Allen Frances

Abstract

The author questions the assumption that family relationship should be given primacy as the validating criterion in establishing diagnostic items for the DSM-III definition of schizotypal personality disorder (SPD). The presenting characteristics of clinically defined patients may be quite different from those that best describe the nonpatient relatives of schizophrenic patients encountered in genetic or high-risk studies. Since the DSM-III nomenclature is meant primarily for use in clinical settings, it must be based on symptom characteristics found in patients, not in nonpatient relatives. If the DSM-III SPD definitions are not optimal for genetic and high-risk studies, special criteria sets will be necessary for such research.

This is a rich and thought-provoking issue that raises a number of interesting and controversial questions. Kendler, Torgersen, and Siever have written brilliant literature reviews summarizing the descriptive, genetic, and biological data bases for schizotypal personality disorder (SPD). These authors seem to share a basic assumption which perhaps accounts for the convergence in their conclusions. This assumption is that familial relationship should be given primacy as a validating criterion for the SPD diagnosis.

For better, and in some ways for worse, DSM-III has not accepted the notion that familial relationship is the primary validating criterion for this syndrome. Although the SPD criteria items included in DSM-III were indeed derived from a systematic chart review of borderline schizophrenics in the Danish studies, Kendler notes that the abstracted records were not restricted to those derived from biological relatives.

Initial validation of the identified SPD items was made on a survey sample gathered from some 800 psychiatrists with no proviso that the patients selected have family histories of schizophrenia. Finally, the DSM-III text has only this unassertive description of the SPD link to schizophrenia:

There is some evidence that chronic schizophrenia is more common among family members of individuals with Schizotypal Personality Disorder than among the general population.

I will not summarize in any detail the many interesting points made by the first three articles because they speak with great clarity and have already been summarized in masterful fashion in the introduction. Simply stated, Kendler, Torgersen, and Siever all believe that the DSM-III SPD criteria set should be changed to describe more faithfully those characteristics that occur most frequently among the biological relatives of schizophrenic patients. As these authors read the descriptive, genetic, and biological literature, the so-called negative symptom SPD picture (e.g., social isolation, coldness, inadequate rapport, and poor functioning) is a more characteristic presentation in biological relatives than the positive symptom SPD picture (e.g., illusions, ideas of reference, and magical thinking). The authors converge on an SPD definition that would be narrower, more homogeneous, more useful in schizophrenia studies, and less frequently encountered in clinical populations.

A related thrust of these articles is to parallel in the Axis II definition of

Reprint requests should be sent to Dr. A. Frances, Dept. of Psychiatry, N.Y. Hospital/Cornell Medical Center, 525 E. 68th St., New York, NY 10021.
SPD some of the innovations made in the Axis I definition of schizophrenia. The negative/positive symptom distinction that has been fruitful in considering Axis I schizophrenic disorders may be usefully applied to SPD. The authors all seem to suggest that the essence of "schizotypality" is embodied in negative symptoms and that positive symptoms are more nonspecific. Moreover, Siever interprets his biological and clinical data to suggest that many of those patients who meet criteria for both SPD and borderline personality disorder are really more borderline than schizotypal. This is entirely analogous to evidence that schizoaffective patients are more affective than schizophrenic, which led to DSM-III's more narrowly inclusive definition of schizophrenia and more widely inclusive definition of affective disorder.

The suggestions offered for a new SPD definition would affect two other personality disorder criteria sets, with implications that are not discussed explicitly. The narrowing of the SPD definition would result in a broadening of the borderline personality disorder category, rendering it even more heterogeneous and nonspecific than the one we already have. Meanwhile, the schizoid personality disorder might disappear altogether, since its current definition seems to be very close to the new definition offered for SPD.

I came away from my reading of the three articles convinced that a more narrow and familial-based SPD definition would have great utility for schizophrenia research, but unconvinced that such changes would provide a useful clinical definition and also unconvinced that DSM-III should give familial relationships primacy as a validating criterion. The little available evidence suggests that subjects who meet the most characteristic familial descriptions do not often present for treatment. It is of interest in this regard that the current DSM-III schizoid personality disorder definition, which resembles the section's suggested new SPD revision, is rarely diagnosed in clinical samples. We will soon know more about the characteristics of clinical SPD samples since several groups (including our own) are in the process of gathering and analyzing data. Our own preliminary data suggest that positive symptoms are more common and discriminating in clinical SPD samples than has been the case in familial samples.

If clinical and familial samples do indeed differ, I would argue that the DSM-III definition should represent the symptomatic picture seen in clinical samples, even though the symptomatic presentation of familial samples has received more validation. The data reviewed by Kendler, Torgersen, and Siever were gathered almost exclusively on nonclinical samples of family members or college students identified through high-risk strategies. These subjects do not come within the purview of the psychiatrists who use DSM-III. The validation offered by available familial and biological studies is very useful in schizophrenia research, but is less meaningful to DSM-III since it does not pertain to the individuals who actually present for clinical evaluation and treatment.

Clinical utility is a more important value in establishing an official nomenclature than is the important validation offered by familial relationship. Moreover, until we know more about SPD diagnosed in clinical settings, it makes strategic sense to keep the category broadly inclusive, heterogeneous, and subject to later dissection as we have more precise information on where to slice. Familial data provide only one tool in making such decisions and should not be given inordinate weight merely because these are by far the best data we now have available.

The distinction between positive and negative symptoms is a dichotomy that lends itself to excessively clear statement. There is a very great overlap between positive and negative symptoms in schizophrenic populations, and there is likely to be an even greater overlap between them at the softer SPD spectrum level. Many patients present with the total DSM-III prototype combining positive and negative symptoms. Similarly, attempts to differentiate sharply between the schizophrenic and the affective disorders are easier on paper than in clinical practice, where a number of perplexing border cases always succeed in presenting themselves. This problem is likely to be more pronounced at the spectrum level, where one would expect even more overlap among SPD, other personality disorders, and various Axis I conditions. The attempts to define narrowly a homogeneous SPD group with clear boundaries distinguishable from its neighbors seem doomed to create a clear, consistent, and nonexistent clinical category. At this stage, at least for DSM-III purposes, it is preferable to allow for heterogeneity and then study it systematically than prematurely to select out an infrequent but more homogeneous group. Of course, schizophrenia researchers are encouraged to apply a narrower and more homogeneous criteria set in their genetic and biological studies.

Let us go on to another issue. There is an unfortunate inconsistency within DSM-III in the handling of the presumed schizophrenic and affective spectrum disorders. The
the cyclothymic and dysthymic syndromes are included within the Axis I rubric. The advantage of placing spectrum disorders within the Axis I rubric is the emphasis it places on descriptive, familial, biological, and treatment relationships and on possible predispositions to decompensation. The disadvantage is that such placement may seem to prejudge the spectrum question and imply a closer relationship than in fact exists. There is also no reason to object to the presence of spectrum disorders (even well-established ones) on Axis II. Many, if not all, personality disorders undoubtedly have a constitutional contribution.

We may close these remarks with some thoughts on the treatment of SPD patients that were stimulated by Stone's eloquent article. It should occasion no surprise that systematic treatment outcome data are sparse given the brief tenure of the SPD syndrome within an official nomenclature. Nonetheless, the general clinical opinion is that these are extremely tough and treatment-resistant patients. There is some early evidence that SPD symptoms do respond to low-dose neuroleptic medication but that SPD patients are especially sensitive to side effects and unlikely to comply with treatment. Antidepressants or anxiolytics may be indicated if specific target symptoms are present, but these medications are not without risks and their effectiveness has not been studied systematically.

How about psychotherapy? The treatment of patients meeting criteria for SPD often requires an extraordinarily gifted clinician (like Stone) who is creative and comfortable in melding together psychodynamic, cognitive, behavioral, and interpersonal techniques. SPD patients tend to be concrete, literal, unpsychologically minded, and likely to experience uncovering interpretations as criticism. The therapist's psychodynamic understanding is always helpful, and sometimes crucial, in treating SPD patients, but interpretations must be used sparingly and only when the therapeutic alliance is sufficiently developed. These are patients who require all the teaching, advice, and social skills training they can get and tolerate. Stone is not a bit bashful in teaching patients how to be tactful, choose a suit, or fend off intrusive parents. Often, such lessons must be repeated over and over, and tied to each new situation since SPD patients have difficulty abstracting the point and generalizing it to similar new situations. Stone becomes the patient's "reality organ," using cognitive techniques to correct distortions and misinterpretations. Finally, Stone is alive to the importance of the interpersonal relationship in his work with SPD patients. His vignettes reveal that he is direct, empathic, patient, helpful, and doesn't mind being a "hired friend" for someone who desperately needs one.

SPD patients have difficulty enjoying relationships. Their learning how to do so occurs best in the context of an extended, enjoyable therapeutic relationship. In my experience, the most common mistake made by therapists in their work with SPD patients is to underestimate the depth of attachment that develops because it is disguised by the patient's overt undemonstrativeness. The psychotherapeutic work with SPD patients is often tedious and only sometimes successful, but when it does work well, the results are unusually gratifying.

In summary, then, I think that Kendler, Torgersen, and Siever have overstated the usefulness of a "schizophrenia connection" in defining the SPD criteria set for DSM-III. Their suggestions for changes in the DSM-III SPD criteria provide a new definition of the syndrome that would be more useful for researchers studying schizophrenia spectrum disorders in biological relatives and high-risk groups, but less useful for clinicians and researchers treating and studying SPD patients encountered in clinical settings. Although I have some points of divergence with the conclusions reached in these articles, I am grateful to all of the authors. This is an extremely well-conceived and well-written series on a topic of emerging interest and importance.

References


The Author

Allen Frances, M.D., is Professor of Psychiatry, N.Y. Hospital/Cornell Medical Center, New York, NY.