PART I
Introduction

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Conceptual and theoretical issues in schizotypal personality research

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Many researchers argue that research into schizotypal personality is important not only in furthering our understanding of this personality disorder, but also in providing key insights into our understanding of schizophrenia. For example, several studies have indicated that schizotypal personality disorder (SPD) is a disturbance that may be genetically related to schizophrenia, insofar as it is prevalent among first-degree biological relatives of schizophrenics. It is also argued that SPD research is of key importance in overcoming methodological weaknesses of schizophrenia research. Psychotic symptoms may mask and blur the more subtle cognitive impairments underlying the schizophrenia spectrum, and they also create difficulty in ensuring that schizophrenics fully understand and comply with the experimental protocol. In addition, effects of neuroleptics and lengthy hospitalization create further confounds in interpreting research on schizophrenic subjects. Thus, research in SPD has become increasingly important as a means of studying biological and cognitive markers of the schizophrenia spectrum without the confound of severe clinical symptoms.

Research since the 1980s has provided grounds for this optimistic view. Schizotypals have been shown to display a number of the biological/neuropsychological markers for schizophrenia, such as eye-tracking impairment, abnormalities on evoked potentials, and attentional deficits. In schizophrenics, these deficits have been linked to damage at brain sites such as the frontal lobes, temporal lobes, and subcortical areas. Examination of these markers is one of the central paradigms in schizophrenia research, and extension of these methods to those afflicted with SPD represents a significant test of the biological and neuropsychological processes thought to underlie schizophrenia. Such research in SPD may also identify those intact cognitive abilities that serve as "protective factors," protecting some schizotypals from developing schizophrenia.

Nevertheless, research on schizotypal personality (relative to other conditions) is still in its infancy, and there are important conceptual and theoretical issues that need to be addressed head-on in the next decade of research in order that genuine advances be made in this area. The aim of this chapter is to outline some of these issues in the hope that this may be of help in directing future research along productive lines. Some of these issues will be addressed in chapters that follow; other issues have yet to be addressed. These issues are grouped under the five headings that follow, namely, diagnostic issues, phenomenological and assessment issues, methodological issues, mechanisms and etiology, and clinical issues.

Diagnostic issues

Axis I or Axis II?

SPD is currently viewed as an Axis II disorder alongside other personality disorders, but the issue of moving it into Axis I alongside schizophrenia was raised during discussions on changes to be made for DSM-IV. Such a move has not occurred but it raises the question of whether SPD is best construed as a deviation representing an exaggeration of an ostensibly normal personality process which places subjects at risk for schizophrenia, or a major disorder that is much more integrally related to schizophrenia itself. The question that needs to be considered in the next decade is what type of research can help address this issue for the future DSM-V. This issue also relates to the issue of dimensional versus categorical conceptualizations of schizotypy, in that retention as an Axis II disorder debatably argues more in favor of a dimensional conceptualization of schizotypy.

Changes in diagnostic content

Should there be changes in the diagnostic content of SPD? Virtually no change was made in the revision for DSM-IV, but there are a number of important issues that require further resolution over the next few years. For example, should anhedonia, which has frequently been viewed as central to schizophrenia, be included as a key feature of SPD? Conversely, social anxiety is a broad-based symptom that does not so cleanly "map on" to other schizophrenia prodromal symptoms as do other features, such as unusual perceptual experiences or paranoid ideation. Recent findings by Chapman and Chapman (1992, and Chapter 5, this volume) on the extent to which different schizotypal personality features predict breakdown for later psychosis represent the type of future research that can help address such questions.

A related issue concerns which of the nine features of schizotypal personality disorder share a common genetic loading with schizophrenia. Answers to such questions can help refine the way in which SPD is conceptualized in DSM-IV, and further promote research on its etiology.

Boundary between SPD and schizophrenia

Where is the boundary between SPD and schizophrenia, and how may this boundary be best defined or conceptualized? The problem of deciding where the defining line between disorder and normality lies has created considerable debate (e.g., Braff et al., 1992). Central to this issue lies the question of whether there is *any* boundary between the two conditions. The possibility must also be faced that our approach to how we answer this question may in part be biased by pragmatic issues and our research experiences. For example, researchers who have regular access to large populations of undergraduates may be biased more to an individual difference / dimensional approach (no discrete boundary), whereas hospital-based researchers who have ready access to clinical schizotypals may be biased more to a categorical (relatively clear boundary) approach.

Late-onset SPD

Are we correct in stipulating that SPD must be lifelong for a diagnosis to be given? In our clinical work we have come across cases where SPD has become manifest relatively later in life (e.g., mid-thirties). Given that we have the concept of late-onset schizophrenia, is it conceivable that late-onset SPD is also a meaningful concept? And if so, can it shed light on the etiology of late-onset schizophrenia, and does it present differently from SPD with onset in early adulthood?

Phenomenological and assessment issues

Categories versus dimensions

Perhaps the most important conceptual issue concerns whether schizotypal personality is best construed as a category or a dimension. This will be reflected in some of the chapters that follow. A number of possibilities come to mind. First, can this issue be addressed using statistical techniques such as MAXCOV-HITMAX (see Lenzenweger & Korfine, Chapter 7, this volume)? Alternatively, can the issue of categories versus dimensions best be addressed by pursuing both approaches (i.e., the clinical approach and the individual dif-

ference approach) and assessing which research approach produces the strongest set of findings?

How many dimensions/factors?

An important issue that needs to be addressed concerns the number of factors or dimensions that underlie schizotypal personality, and the implications for our conceptualization of both SPD and schizophrenia. If schizophrenia and SPD are closely linked phenomenologically, factors of SPD should reflect factors of schizophrenia. To date there has been relatively little factorial validity research on SPD, though this is crucial to helping us confirm or reject syndrome models of SPD.

"Schizophrenias" and "schizotypes"

Given that we often refer to the "schizophrenias," should we also talk about the "schizotypes"? We do this in part by referring to positive and negative schizotypal processes, but to what extent is this dichotomy overly simplistic? Is it more accurate to talk about cognitive–perceptual and interpersonal deficit subtypes of SPD? Whereas the paranoid–nonparanoid division is frequently made in schizophrenia research, this has not to date been applied to SPD research.

Family history of SPD

The question of whether SPD with a family history of schizophrenia differs from SPD without such a history is a potentially very important one, but to date there has been very little research on this issue (although see Condray & Steinhauer, 1992). Important questions that need to be addressed in this area are: (1) Are there major phenomenological differences in terms of presenting symptoms between these two groups? (2) Are there fundamentally different mechanisms underlying these two SPD "types"? (3) Do these differences reflect those found in schizophrenics with and without a family history of schizophrenia?

Psychosis proneness versus schizotypal personality

In what ways does the concept of self-report psychosis-proneness map onto concepts of self-report and clinical schizotypal personality? There is a tendency in the literature to assume that research on psychosis proneness is of

automatic relevance to SPD research, but to what extent are these only partially overlapping processes? For example, some psychosis proneness scales such as Psychoticism, Physical Anhedonia, Social Anhedonia, and Schizoidia do not directly reflect schizotypal features, at least as defined by DSM-III-R and DSM-IV. Whereas on the one hand findings from research on such scales may be of less relevance to SPD as defined in DSM-III-R, on the other hand they may yield valuable information on ways in which such psychiatric definitions of SPD may require extension and revision.

Mechanisms and etiology

Same or different causal mechanisms?

There is an assumption that mechanisms underlying individual differences in schizotypal personality are the same ones underlying schizophrenic symptomatology and that therefore such research yields important insights into schizophrenia. Is this assumption justified? Although schizotypal features appear to reflect milder versions of schizophrenic symptoms, this does not guarantee that the causes of schizophrenia are an exaggeration of the dysfunctional mechanisms that cause SPD. Just as there can be many different causes of what ostensibly presents as the same symptom (e.g., cough, which may be caused by smoking, food lodging in throat, anxiety and nervous habits, air pollution, infection of mucous membranes of nose/throat, inflammation of bronchial tubes via a viral infection), so too may very different mechanisms underlie, for example, thought disorder in schizophrenia and its analogue in SPD, odd speech. Is it even possible that further understanding of the mechanisms underlying SPD actually misinforms us about processes involved in schizophrenia?

Protective factors

We currently know very little about what factors may protect against the development of schizophrenia. Can an elaboration of the differences between SPD and schizophrenia give us important leads into understanding what these factors may be? Presumably, schizotypals who do not go on to develop schizophrenia may possess some characteristic that shields them from a full breakdown (although it is also possible that they lack an important risk factor, such as birth complications). Conversely, do differences between schizophrenics and SPDs merely indicate that SPD is not really a good model for understanding schizophrenia?

SPD as the basic disorder

The extensive amount of research conducted on schizophrenia leads one to assume that schizophrenia is obviously the fundamental disorder that needs to be explained, but is this assumption really justified? May not SPD instead represent the fundamental disorder, with schizophrenia arising as a complication of this more fundamental process through the presence of some additional risk factor? If the latter is true, what factors, when present in schizotypals, lead to eventual breakdown for schizophrenia?

Single or multiple vulnerability factors?

Although there is growing evidence for multiple vulnerability factors for SPD, several authors conceptualize SPD as the product of one fundamental vulnerability factor (e.g., Grove et al., 1991). Although there may be a number of identifiable etiological mechanisms underlying SPD (e.g., eye tracking abnormalities, sustained attention deficits, negative priming), these deficits may be interrelated rather than independent, thus reflecting the operation of a relatively unitary dysfunction. Research needs to go beyond the establishment of individual correlates and move toward a more integrative framework for understanding SPD.

New processes or neurodegenerative processes?

We need to know more about the way in which SPDs eventually break down for schizophrenia. Are there discrete new processes that come to act as triggers that transform a schizotypal into a schizophrenic (e.g., psychosocial stress or a new physiological process) and that are additional to some other more static predisposition? Alternatively, does schizophrenia largely develop with the passage of time, allowing an existing neurodegenerative process to develop fully? The latter scenario might suggest that SPD is merely the clinical manifestation of some midpoint in this slowly evolving process and that the rate and extent to which the neurodegenerative process develops determine whether a schizotypal eventually is transformed into a schizophrenic.

Is schizophrenia a good model for understanding SPD?

There are probably many causes of schizophrenia, just as there are many causes of mental retardation. If we use mental retardation as an analogy, we might consider that having a borderline IQ of 75 is to mental retardation (IQ below 70) as SPD is to schizophrenia. Just as there are many relatively dis-

crete causes of mental retardation, there may be many more (normal as well as pathological) processes that play a role in determining borderline IQ. By analogy, there may be many more processes that play a role in determining SPD which we never assess because our strategy in SPD research is invariably to take a correlate of schizophrenia and see if it is found in SPD. If such a correlate successfully extrapolates from schizophrenia to SPD, we take this to support the notion that it is a true vulnerability factor for schizophrenia. But if not, we often argue for methodological/conceptual problems (e.g., small *N* or inappropriateness of SPD as a model for schizophrenia) as explanations for null findings. This approach, however, may lead to a misunderstanding of SPD, because SPD may in reality be a broader clinical concept than schizophrenia, involving many more processes and mechanisms. If understanding schizophrenia requires a full understanding of SPD, and if researchers never step outside schizophrenia research to select alternative potentially relevant processes to help explain SPD, will we inevitably fail to understand schizophrenia fully? One question therefore concerns whether there are any other alternative models to schizophrenia that we can utilize to help study SPD. In this context, research on more "normal" personality processes related to SPD traits may provide one important lead.

Mechanisms and etiology

Clinical versus individual difference approach

Perhaps the clearest differentiator among researchers of schizotypal personality concerns whether a clinical or an individual difference approach is taken. This bifurcation is paralleled by a preference for the use of either (1) psychiatric diagnostic instruments to assess clinical manifestations of SPD or (2) more psychologically based self-report questionnaires to assess degrees of schizotypy. An obvious question concerns which approach is more likely in the long term to result in greater advances in our understanding of SPD. Alternatively, will the greatest advances be made by a combination of these two approaches? Currently there are very few empirical data that can help address this issue. This is important, because it is likely to be the first and most fundamental question to which any new researcher in this area requires an answer.

Cause–effect relationships

Teasing out causality is one of the major challenges for the next generation of SPD research. The conceptual issue concerns the fact that although it is not

explicitly stated, we tend to make assumptions of causality in interpreting findings from SPD research; for example, if cognitive deficits are found in schizotypals, it is implicitly assumed that such deficits are in some way causally related to SPD. Yet are schizotypal symptoms a result of the proposed pathophysiological process, or is the pathological process caused by the symptoms? For example, does poor or inappropriate resource allocation (e.g., as indexed by the P300 event-related potential or the skin conductance orienting response) result in unusual perceptual experiences, or do perceptual aberrations interfere with resource allocation and produce the observed attentional deficits? Alternatively, is a reiterative process involved whereby both causal processes make up a deleterious feedback loop, exacerbation of which may result in perceptual aberrations being upgraded to hallucinations? One way of teasing out such cause–effect relationships may be by looking at a subgroup that possesses the deficit in question and then showing that members of the subgroup can be differentiated in meaningful ways from those without the deficit on an unrelated variable/process (e.g., family history of schizophrenia, being winter-born, possession of minor physical anomalies) that cannot easily be viewed as an artifact of the symptomatology in question (e.g., unusual perceptual experiences). Such a pattern of results would lend support to the notion that the deficit is a meaningful one and is not purely an artifact of the symptoms it correlates with. A second alternative is to make use of the longitudinal prospective design in the hope of demonstrating that the deficit in question precedes the onset of the symptoms.

Comorbidity

The issue of comorbidity has not seriously impinged on published research on SPD to date. The reason is probably that the state of such research is largely still at the initial level of establishing correlates of SPD. Nevertheless, future research – that is, both clinical research and individual difference research – needs to deal seriously with the issue of the potential confound of comorbidity. Affective disturbance is common among clinical schizotypals, and we have recently found in our own research on community schizotypals that alcohol abuse, drug use, and antisocial behavior are common in SPDs within the community. Consequently, we need to establish that correlates of SPD that we believe are of etiological significance are independent of coexisting psychiatric conditions. On the other hand, there is a danger that researchers will correct for comorbid conditions, such as depression, that may turn out to be an essential, core feature of SPD. The pressing conceptual question therefore concerns how future research should best ride between these competing concerns.

The importance of normal personality processes

Do we have to turn much more to understanding normal processes in order to understand pathological processes? For example, in order to understand more fully what it means for SPDs to show abnormalities in brain structure and function, we need to map in greater detail just exactly what constitutes "normal" brain processes in order to both define and understand deviations from normality. Just as we use SPD as a tool to understand schizophrenia, do we also have to understand normal personality processes in order fully to understand SPD symptomatology, and in turn schizophrenia? This process is not the same as researching individual differences in schizotypy in the normal population; rather, it refers to understanding normals who have no discernible schizotypal tendencies. For example, what are the normal processes that shape appropriate levels of social anxiety and suspiciousness?

Hospital versus community approaches

Clinical studies of SPD recruit subjects from a hospital sample; or alternatively (and less frequently), they are recruited from the community and have not previously been hospitalized. It is possible that these two sampling approaches may yield fundamentally different patterns of findings, as such populations differ in important ways. Both approaches have advantages and disadvantages. The advantage of hospital samples is that they represent relatively severe clinical cases; a potential disadvantage is that the reason for hospitalization means that subjects are likely to have a coexisting major disorder that complicates the clinical picture. The advantage of community schizotypals is that they have not been hospitalized and may be less likely to have a serious coexisting condition (though this advantage is probably more relative than absolute); the disadvantage may be that social dysfunction may be less serious, and hence it may be more difficult to establish the correlates of this relatively weaker clinical manifestation of SPD.

Expected effect sizes

Current research is largely concerned with the statistical significance of a finding, however, given the difficulty in obtaining large samples of schizotypals, the size of the effect obtained may be a more meaningful guide to assessing the "significance" of findings. This in turn raises the question, What effect sizes should one expect? One might expect stronger effect sizes in SPDs than in schizophrenics because there are no or fewer drug/institutional contami-