Affective Deficits and Social Deficits in Schizophrenia: What’s What?

by Robert H. Dworkin

Abstract

Affective deficits and social deficits have played an important role in theory and research on schizophrenia, and it has generally been assumed that these are two different types of symptoms. The aim of these comments is to argue that the distinction between these symptoms is not straightforward and that the apparent differences between affective and social deficits virtually disappear when current approaches to their definition and measurement are considered. The implications of this point with regard to theory and research on the etiology and treatment of schizophrenia are discussed, and an approach that has the potential to reveal whether specific schizophrenic symptoms are more closely associated with affective deficits or with social deficits is described.

Deficits in affective response have long been considered a prominent feature of schizophrenia (Kraepelin 1919/1971; Bleuler 1911/1950; Rado 1953; Meehl 1962; Strauss et al. 1974) and play a major role in recent theory and research on negative symptoms (Crow 1980, 1985; Andreasen 1982; Knight and Roff 1985; Pogue-Geile and Zubin 1988; Walker and Lewine 1988). Deficits in social functioning also have a distinguished place in the history of schizophrenia (Bleuler 1911/1950; Kraepelin 1919/1971; Meehl 1962; Strauss et al. 1974) and are increasingly targeted in comprehensive approaches to treatment (e.g., Hogarty et al. 1986; Liberman et al. 1986; Bellack 1989). Not only has it generally been assumed that these are two different types of symptoms, but it has also been proposed that affective deficits (and other negative symptoms) and social deficits reflect separate realms of functioning in schizophrenia (Strauss et al. 1974; Crow 1985).

The aim of the present comments is to argue that the distinction between affective and social deficits is not as straightforward as it has seemed. Indeed, this distinction virtually disappears when current approaches to the definition and measurement of these deficits are considered. Perhaps the best way to demonstrate this point is to begin by examining the items of the affective flattening subscale of the Scale for the Assessment of Negative Symptoms (SANS; Andreasen 1982, 1984). The SANS, probably used in more studies than any other method of assessing negative symptoms in schizophrenia, provides subscales for assessing affective flattening, alogia, avolition, anhedonia, and attention. The affective flattening subscale consists of seven symptom ratings, which can be summed to provide an affective flattening score; these items are unchanging facial expression, decreased spontaneous movements, paucity of expressive gestures, poor eye contact, affective nonresponsivity, inappropriate affect, and lack of vocal inflections. It has been suggested that several of these ratings may not reflect the negative symptom syndrome (inappropriate affect [Walker and Lewine 1988]; decreased spontaneous movements and paucity of expressive gestures [Knight and Roff 1985]), but the validity of the subscale as a measure of affective flattening has not been questioned. It is important to recognize that the clinical ratings constituting the SANS...
affective flattening subscale are quite representative of other measures that have been used to assess affective flattening in schizophrenia (e.g., Abrams and Taylor 1978; Iager et al. 1985; Kay et al. 1987; Alpert et al. 1989).

The SANS ratings of affective flattening are based on the patient’s behavior during an interview—an interpersonal situation in which the patient is expected to interact with another person. When the SANS affective flattening subscale is considered in this context, it becomes clear that an alternative way of conceptualizing these ratings is that they are measures of social functioning or social skills. Eye contact, facial expressiveness, vocal inflection, and affective responsivity are all social behaviors that facilitate communication. It is certainly possible that a patient’s interpersonal behavior lacks these qualities because the patient suffers from an affective deficit. But it is just as plausible that a patient has, for example, poor eye contact or a lack of vocal inflections because the patient suffers from a social deficit and lacks the ability to engage in the socially appropriate behaviors that constitute competent interpersonal interaction.

An examination of research on social deficits in schizophrenia makes these two different possibilities very clear. In much of this research, the very same behaviors that have been used to measure affective flattening—eye contact, affective responsivity, facial expressiveness, and various voice characteristics—have been considered social skills (e.g., Lindsay 1982; Liberman et al. 1986; Morrison and Wixted 1989). Although it has been acknowledged that measures of affective flattening are also measures of social behavior (Wixted et al. 1988; Bellack et al. 1989b, 1990; Jackson et al. 1989a, 1989b), the fact that identical behaviors are used to measure two domains of symptoms that are thought to be different has important implications that have not been generally recognized.

One implication of this confounding of measures is obvious. It can be expected that affective flattening and social functioning will be correlated when measures of these two symptoms are based on identical or similar behaviors. For example, the correlations between affective flattening measured by the SANS and social skills measured during simulated social encounters (including eye contact, facial expressiveness, and tone of voice) were significant in several recent studies (Jackson et al. 1989b; Bellack et al. 1990; Mueser et al. 1990b). This finding is hardly noteworthy, however, because the same behaviors were used to assess these presumably different symptoms. Significant correlations between the negative symptom syndrome and various aspects of social competence—for example, educational and vocational impairments and deficits in social relationships—have also been reported (Pogue-Geile and Zubin 1988; Walker and Lewine 1988; Andreason et al. 1990) and possibly reflect, in part, such a confounding of measures. However, research on genetic influences (Dworkin et al. 1988), sex differences (Dworkin 1990; Mueser et al. 1990a), and premorbid development (Knufft and Roff 1983, 1985; Dworkin et al. 1990, 1991) suggests that negative symptoms and social functioning also reflect relatively independent processes in schizophrenia. If overlap in the measurement of these two domains of symptoms is taken into account in future research, support for this conclusion is likely to become more widespread.

The use of similar definitions of affective flattening and social skills also has implications for research on the etiology of schizophrenia. Although there are a great many hypotheses, the sources of social and affective deficits in schizophrenia are not well understood. For example, it has been suggested that social deficits in schizophrenia may be a result of faulty learning experiences (Morrison and Bellack 1987), genetic factors (Kendler et al. 1982; Dworkin et al. 1988), neurodevelopmental processes (Quitkin et al. 1976; Bellack et al. 1989a), and attention and information-processing dysfunction (Liberman et al. 1986; Bellack et al. 1989a; Cornblatt et al., in press). It is also possible, of course, that social deficits are a consequence of the disorder or its treatment (Brady 1984). There are various potential causes of affective deficits in schizophrenia, including the same factors that have been proposed for social deficits (e.g., Knight and Roff 1985; Berenbaum et al. 1990). One intriguing possibility is based on research demonstrating that neurologic and neuromotor deficits are characteristic of schizophrenic patients and their high-risk offspring and other first-degree relatives (e.g., Kinney et al. 1986; Woods et al. 1986; Heinrichs and Buchanan 1988; Erlenmeyer-Kimling et al. 1989). These results suggest that deficits in interpersonal behaviors such as facial expressiveness or eye contact (which have been conceptualized as reflecting both affective flattening and impaired social skills) may be manifestations of neurologic or neuromotor deficits, reflecting lesions in, for example, the right hemisphere (Borod et al. 1989) or dorsolateral prefrontal cortex (Weinberger 1987). Clearly, additional research on the sources of affective and social deficits is neces-
sary. But in addition to investigating each one of these symptoms individually, it will also be necessary to examine the sources of the association between them, sources that may include both genetic and environmental components. In research on the etiology of social and affective deficits and their covariation is to be worthwhile, it is essential to ensure that approaches to defining and measuring these symptoms keep them carefully distinguished.

The way in which affective and social deficits are conceptualized also has implications for the treatment of schizophrenia. It has been suggested that negative symptoms in schizophrenia are relatively stable and resistant to treatment (Crow 1985). If, for example, poor eye contact and lack of vocal inflections are thought to reflect the negative symptom of affective flattening, then it might be assumed that such symptoms are not amenable to change. However, there is evidence that negative symptoms such as affective flattening are not necessarily static phenomena and that they can be responsive to treatment (e.g., Goldberg 1985; Lindenmayer and Kay 1987). This possibility suggests that treatment approaches could be developed that might, for example, seek to increase the frequency of (positive) affective responses by reinforcing those that do occur or by identifying situations in which such responses are likely and increasing the frequency of the patient's exposure to them (cf. Meehl 1974-75, 1975, 1987). On the other hand, if poor eye contact and lack of vocal inflections are considered social skills, then it follows that social skills training programs may be effective in ameliorating such deficits (e.g., Liberman et al. 1986; Morrison and Wixted 1989). It is interesting that poverty of speech—another prominent negative symptom (Andreasen 1982, 1984; Crow 1985)—has also been considered a social deficit (Wixted et al. 1988). Although a discussion of poverty of speech is beyond the scope of these comments, it should be noted that the implications are very similar to those that have been discussed here. Attention to the possibility that poverty of speech reflects a social deficit rather than a formal thought disorder (i.e., a cognitive deficit) would be just as important in research on schizophrenia as would additional consideration of the relationship between affective and social deficits.

There is appreciable overlap in the definition and measurement of affective and social deficits in recent research on schizophrenia, and this overlap must be addressed in future research. This conclusion, however, does not answer the question posed in the title of these comments. That is, are poor eye contact, diminished facial expressiveness, and other such behaviors affective deficits, or are they social deficits? This question has important implications for research on etiology and treatment, but it cannot be answered satisfactorily with the data that are currently available on affective and social deficits in schizophrenia. One simple approach to this question would be to operationally define each behavior as one type of deficit or the other. This approach, however, would not address the distinction between affective and social deficits in a theoretically meaningful and heuristic fashion. A much better way of determining whether these behaviors reflect an affective deficit or a social deficit would be to begin with a theoretical model of these deficits that is as clearly articulated as possible and that can be readily operationalized. In such a model, it would be important to recognize that there may be significant areas of overlap between affective and social deficits. Emotional expressivity is often a response to various kinds of social stimuli and in turn often plays an important role in communication and interpersonal interaction. Indeed, facial expressions such as smiles may reflect a dimension of "social affect" that is neither simply affective responsiveness nor competent interpersonal behavior.

One valuable approach to examining these areas of overlap would be to systematically vary both the social versus nonsocial nature of the eliciting stimuli (cf. the distinction between physical and social anhedonia in Chapman et al. [1976]) and the social versus nonsocial context of the affective response (e.g., watching movies alone or with other people present). This approach may not be as straightforward as it sounds, however, because both videotape and audiotape recording, integral methodological features of such studies, imply the eventual presence of another person as viewer or listener.

A multitrait-multimethod matrix approach to research (Campbell and Fiske 1959) provides a valuable method for examining associations between measures of affective and social deficits. In this approach, two or more methods of measuring each of two or more traits are intercorrelated; the patterning of these correlations reflects the construct validity of measures of a trait and permits an assessment of convergent and discriminant validity as well as the influence of method factors. Multitrait-multimethod matrices of correlations using different methods of measuring both affective and social deficits have the potential to reveal whether, for example, poor eye contact is more
strongly associated with affective deficits or with social deficits in schizophrenia. In addition to the simple visual inspection of such a correlation matrix, confirmatory factor analysis can also be used to test the fit of alternative models of the factor structure of the data (e.g., Schmitt and Stults 1986; Marsh and Hocevar 1988). In designing such studies, it will be important to consider the possibility that affective and social deficits may themselves be multidimensional. Various authors have proposed that social functioning in schizophrenia has several different components (e.g., Zigler and Levine 1981; Tanaka and Bentler 1983; Morrison and Bellack 1987; Cornblatt et al., in press), a suggestion that may also be a valid characterization of affective deficits (e.g., Chapman et al. 1976; Berenbaum et al. 1987; Lindemayer and Kay 1987). It will also be important to incorporate a longitudinal component in such research, which will allow the relationships between affective and social deficits to be studied over the developmental course of schizophrenia, beginning with the premorbid period (e.g., Dworkin et al. 1990, 1991) and continuing with followup assessments through the acute illness and its outcome (e.g., Knight and Roff 1983, 1985).

In concluding, it must be emphasized that the SANS has been used to illustrate the preceding comments because it is representative of other measures of affective flattening and because it is a widely used measure of this negative symptom. These comments are therefore applicable to a variety of approaches to the assessment of affective flattening and are not meant to be a criticism of this one specific measure. Similarly, these comments should not be construed as a criticism of recent approaches to the assessment of social skills. Andreasen’s SANS and these research programs represent sophisticated, state-of-the-art approaches to the assessment of symptoms and social functioning. The intent of these comments has been to draw attention to a neglected issue in the conceptualization of affective and social deficits in research on schizophrenia.

References


Andreasen, N.C. The Scale for the Assessment of Negative Symptoms (SANS). Iowa City, IA: Department of Psychiatry, University of Iowa, 1984.


Cornblatt, B.A.; Lenzenweger, M.F.; Dworkin, R.H.; and Erlenmeyer-Kimling, L. Childhood attentional deficits predict social isolation in adults at risk for schizophrenia. *British Journal of Psychiatry*, in press.


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