Childhood Precursors of Affective vs. Social Deficits in Adolescents at Risk for Schizophrenia

by Robert H. Dworkin, Barbara A. Cornblatt, Ruth Friedmann, Lucy M. Kaplan, Julie A. Lewis, Anthony Rinaldi, Carol Shilliday, and L. Erlenmeyer-Kimling

Abstract

Childhood attentional and neuromotor precursors of social competence and affective deficits in adolescents at risk for schizophrenia, adolescents at risk for affective disorder, and matched comparison adolescents were examined. The subjects were offspring of parents with schizophrenia or affective disorder and of normal parents matched on age, sex, and socioeconomic status from the New York High-Risk Project (Sample B). On the basis of interviews conducted when the subjects were children and adolescents, social competence was rated from child reports and parent reports, affective deficits were assessed by affective flattening ratings, and smiling was assessed by counting broad smiles. Adolescents at risk for schizophrenia had significantly greater social and affective deficits than adolescents at risk for affective disorder and comparison adolescents. In subjects at risk for schizophrenia, childhood neuromotor dysfunction predicted adolescent affective flattening, and childhood attentional dysfunction predicted adolescent social deficits. The results suggest that affective and social deficits in schizophrenia have different childhood precursors.

In an influential paper, Strauss and his colleagues (1974) proposed that three types of schizophrenia symptoms—positive and negative symptoms and disorders of social relationships—are independent and reflect separate underlying processes in the pathogenesis of the disorder. The results of research in which genetic influences (Dworkin et al. 1988), sex differences (Dworkin 1990; Mueser et al. 1990), structural equation modeling (Lenzenweger et al. 1991), and developmental course (Knight and Roff 1983, 1985; Dworkin et al. 1990, 1991; Fenton and McGlashan 1991) have been examined are consistent with this suggestion. However, the etiological component of Strauss et al.'s (1974) hypothesis has been addressed only partially because these studies have not adequately identified the processes underlying these three types of symptoms.

Because the risk of schizophrenia is increased in the offspring of schizophrenia parents, studies of these offspring—who are typically termed "high risk"—provide a means of exploring the longitudinal development of schizophrenia symptoms and signs (Erlenmeyer-Kimling 1968; Garmezy 1974; Garmezy and Streitman 1974; Erlenmeyer-Kimling and Comblatt 1987a; Asarnow 1988). In two independent samples of subjects in the New York High-Risk Project (NYHRP), we examined the offspring of parents with schizophrenia and affective disorder and presented evidence of dissimilar patterns of group differences in positive formal thought disorder, affective flattening, poverty of speech, and social competence during childhood, early adolescence, and adolescence (Dworkin et al. 1990, 1991). Although these data are consistent with the suggestion that positive and negative symptoms and social competence reflect
separate underlying processes, they do not identify the nature of the underlying processes. In this article, we examine the processes underlying two of these symptoms by investigating childhood precursors of affective deficits and social deficits in adolescents at risk for schizophrenia. We have focused on these two symptoms for three reasons. First, affective deficits are considered a central part of the negative symptom syndrome (Crow 1985; Pogue-Geile and Zubin 1988; Walker and Lewine 1988; McGlashan and Fenton 1992). A second reason is that, although we previously found evidence that adolescents at risk for schizophrenia had significantly greater positive formal thought disorder than adolescents at risk for affective disorder and the adolescent offspring of normal parents, we concluded that positive thought disorder in high-risk children and adolescents is a subtle phenomenon that should be investigated with more sensitive measures than the clinical ratings we used (Dworkin et al. 1991). The final reason we have focused on affective and social deficits is that it has recently been suggested that these two types of symptoms have not been clearly distinguished in research on schizophrenia; apparent differences between affective and social deficits virtually disappear when current approaches to their definition and measurement are considered (Dworkin 1992). Because it may be difficult to distinguish these two types of symptoms completely at a descriptive-phenotypic level, it would be very important to determine whether different processes play a role in their development.

But what underlying processes? Although there are many hypotheses, the sources of social and affective deficits in schizophrenia are not well understood. Many candidates can be found among the biological and behavioral variables that differentiate patients with schizophrenia from normals and patients with other disorders. Such differences may, of course, reflect effects of schizophrenia rather than causes. For this reason, variables that also distinguish first-degree relatives, including high-risk offspring of patients with schizophrenia, from comparison subjects are more likely to reflect mechanisms involved in the development of the disorder. In studies using diverse methods and conducted by different investigators, attentional dysfunction and neuromotor dysfunction have been found to characterize not only schizophrenia patients but also their high-risk offspring and other first-degree relatives (Rutschmann et al. 1977; Nuechterlein and Dawson 1984; Cornblatt and Erlenmeyer-Kimling 1985; Kinney et al. 1986; Woods et al. 1986; Erlenmeyer-Kimling and Cornblatt 1987a; Heinrichs and Buchanan 1988; Erlenmeyer-Kimling et al. 1989).

It has been suggested that social deficits in schizophrenia may be a result of attention and information-processing dysfunction (Liberman et al. 1986; Bellack et al. 1989; Cornblatt et al. 1990, 1992). In a cross-sectional study of children at risk for schizophrenia (Nuechterlein et al. 1990), significant relationships between these two domains of variables were not found. However, in longitudinal analyses examining the two independent samples of the NYHRP, Cornblatt et al. (1990, 1992) reported that childhood attentional dysfunction predicted adolescent and adult social isolation in subjects at risk for schizophrenia. Affective deficits, on the other hand, have been reported to be associated with neuromotor dysfunction (Manschreck et al. 1982, 1985) and neuropsychological deficits in schizophrenia (Mayer et al. 1985), and it has been suggested that schizophrenia patients with flat affect may have compromised right hemisphere function (Borod et al. 1989). In addition, the negative symptom syndrome—of which affective deficits are a central component—has been found to be associated with neurological soft signs, neuromotor dysfunction (Merriam et al. 1990; McGlashan and Fenton 1992), and structural brain abnormalities (Walker and Lewine 1988) in patients with schizophrenia. Although it has also been suggested that affective deficits in schizophrenia may be a consequence of cognitive deficits, the results of a recent study were not consistent with this hypothesis (Berenbaum and Olmanns 1992).

In the present analyses, we examined the relationship between attentional and neuromotor dysfunction in childhood and social and affective deficits in adolescence in subjects at risk for schizophrenia. Specifically, we hypothesized that childhood attentional dysfunction predicts adolescent social deficits (Cornblatt et al. 1990, 1992) and that childhood neuromotor dysfunction predicts adolescent affective deficits.

**Methods**

**Subjects.** The NYHRP, a longitudinal investigation of the offspring of parents with schizophrenia and affective disorder, was begun in 1971 by Erlenmeyer-Kimling. Two independent samples are included in the project; the present analysis...
was based on the second of these (Sample B), ascertained in 1977–79. The study is described in detail elsewhere (Erlenmeyer-Kimling et al. 1984; Erlenmeyer-Kimling and Cornblatt 1987b).

From consecutive admissions to New York State psychiatric facilities in the New York metropolitan area, patients with intact marriages and 7- to 12-year-old children with no evident psychiatric disorder or mental retardation were diagnosed according to the Research Diagnostic Criteria (Spitzer et al. 1978) by two senior psychiatrists using information from hospital records. Only patients for whom there was agreement for a diagnosis of schizophrenia or affective disorder were accepted into the study. Subsequently, these patients were administered the Schedule for Affective Disorders and Schizophrenia-Lifetime Version (Endicott and Spitzer 1978) to confirm the diagnoses. On the basis of comparisons of the major variables examined in the study, the offspring of parents with diagnoses of schizophrenia and schizoaffective, mainly schizophrenic, disorder were combined, as were the offspring of parents with diagnoses of affective disorder and schizoaffective, mainly affective, disorder.

A population sampling firm identified a large number of families matched in socioeconomic status to the patient families, from which a group of normal comparison children matched to the high-risk children on age and sex was selected. Children in this group were 7 to 12 years of age, from intact homes, and without histories of psychiatric problems or mental retardation. Families in which either parent had had psychiatric hospitalization or treatment or a history of psychiatric problems were excluded from this normal comparison group.

**Procedure.** The three groups of offspring were assessed at 2- to 3-year intervals with a variety of biobehavioral and clinical measures (Erlenmeyer-Kimling et al. 1984; Erlenmeyer-Kimling and Cornblatt 1987b). At the first testing round, measures of attentional performance were administered by trained laboratory technicians, and a comprehensive structured neurological examination was conducted by a neurologist. At the first and third testing rounds, 30-minute semistructured videotaped interviews were administered by child psychiatrists blind to the subjects’ group membership; the interviews were subsequently rated on the Mental Health Assessment Form (Kestenbaum and Bird 1978). In addition, at both of these testing rounds, structured interviews consisting of a large number of questions designed to assess different aspects of psychopathology and adaptive behavior in the offspring were administered to the parents of all subjects.

The videotapes of the offspring interviews from the third testing round, at which the mean age of the subjects was 15.4 years, were rated for reported social competence and affective flattening both independently and in separate randomized orders by each of two raters randomly chosen from a group of three advanced graduate students in clinical psychology. These videotapes were also rated for smiling both independently and in random order by another advanced graduate student in clinical psychology who had appreciable experience in childhood psychopathology. Two-thirds of these videotapes were also rated for smiling both independently and in random order by another advanced graduate student in clinical psychology who had appreciable experience in childhood psychopathology. The remainder were rated by the graduate student who had rated smiling in the third-round interviews several months after those ratings were completed. All seven raters were blind to the subjects’ group membership.

**Measures.** The development of a measure of global attentional deviance in Sample A of the NYHRP has been described in detail (Cornblatt and Erlenmeyer-Kimling 1985; Cornblatt et al. 1992), and a similar measure has been developed for the sample examined in the present analyses (Sample B). This measure has been described by Cornblatt et al. (1989, 1990), who reported that the children at risk for schizophrenia had significantly greater attentional dysfunction than the children at risk for affective disorder and the normal comparison children.

The measure of neuromotor dysfunction used in the present analyses was based on the structured neurological examination conducted in childhood. This examination consisted of 77 items that assessed a large number of neurological signs. Neuromotor
dysfunction is the neurological finding that has been reported most consistently in schizophrenia patients and their high-risk offspring and other first-degree relatives (e.g., Orvaschel et al. 1979; Rieder and Nichols 1979; Erlenmeyer-Kimling et al. 1982; Kinney et al. 1986; Woods et al. 1986; Erlenmeyer-Kimling et al. 1989). For the present analyses, the 29 items from the neurological examination that assessed neuromotor dysfunction were selected. Several of these neuromotor dysfunction items had limited or even no variability among the sample of offspring. Nevertheless, all of these items were used to construct a scale of neuromotor dysfunction, both because this scale had excellent internal consistency (coefficient alpha = 0.88; Cronbach 1951) and because doing so makes it possible to examine the same neuromotor dysfunction scale at later ages when neuromotor deficits may develop that were not manifested in childhood. This measure is described in detail in Rinaldi et al. (1991), in which it is reported that the children at risk for schizophrenia had significantly greater neuromotor dysfunction than the children at risk for affective disorder and the normal comparison children in Sample B of the NYHRP.

Reported social competence was assessed by using the mean of the scores on items of the Premorbid Adjustment Scale (PAS; Cannon-Spoor et al. 1982). In previous analyses (Dworkin et al. 1990, 1991), we modified the first item of the scale—“sociability and withdrawal”—to reflect sociability and withdrawal observed in the interview, thereby more clearly distinguishing this rating from the second item of the scale, “peer relationships.” Because such ratings of observed sociability may reflect an affective deficit (Dworkin 1992), this item has not been included in the calculation of the social competence scores used in the present analyses, and we have used only PAS items that assess reported, rather than observed, social competence: peer relationships, adaptation to school, degree of interest in life (i.e., hobbies and interests), and social-sexual adjustment (in offspring >12 years). The intraclass correlation interrater reliabilities (Shrout and Fleiss 1979) of the means of the two people who rated reported social competence were 0.75 in childhood and 0.81 in adolescence.

The parent interviews administered at the third testing round included a larger number of items than the first-round parent interviews. Both sets of items were examined, and all items that assessed the aspects of social competence reflected in the PAS ratings—peer relationships, school adjustment, and degree of interest in life—were identified. There were 12 items in the first-round parent interviews and 23 items in the third-round parent interviews that assessed these three aspects of social competence. These items were used to calculate measures of childhood and of adolescence parent-reported offspring social competence. Each measure consisted of the overall mean score of mean ratings on three groups of items reflecting these aspects of social competence. These items were used in the present analyses, and we have used only PAS items that assess reported, rather than observed, social competence: peer relationships, adaptation to school, degree of interest in life (i.e., hobbies and interests), and social-sexual adjustment (in offspring >12 years). The intraclass correlation interrater reliabilities (Shrout and Fleiss 1979) of the means of the two people who rated reported social competence were 0.75 in childhood and 0.81 in adolescence.

As described in Dworkin et al. (1991), on the basis of research on affective deficits in schizophrenia (Knight and Roff 1985), affective flattening was assessed using the mean of four items (unchanging facial expression, poor eye contact, affective nonresponsivity, and lack of vocal inflection) selected from the affective flattening subscale of the Scale for the Assessment of Negative Symptoms (SANS; Andreasen 1982; Andreasen et al. 1990). Although it could be argued that the SANS anhedonia-asociality subscale is also a measure of affective deficits, ratings on this subscale may reflect a combination of affective deficits and social deficits (Dworkin et al. 1987). Therefore, to keep our ratings of social competence and affective deficits as distinct as possible, items from this SANS subscale were not examined. The intraclass correlation interrater reliabilities (Shrout and Fleiss 1979) of the means of the two people who made these rat-
ings of affective flattening were 0.71 in childhood and 0.73 in adolescence.

As discussed above, in current research on schizophrenia, the same behaviors have been considered both affective and social deficits (Dworkin 1992). One behavior that has been considered both is smiling. Broad smiles were defined (Friedmann, in preparation) as smiles involving muscles around the mouth and other facial muscles, usually exposing the teeth (small smiles that include just the mouth muscles were not included). For each of two 3-minute intervals of the videotaped offspring interviews—minutes 5 through 7 and 15 through 17—broad smiles were counted and have been analyzed as smiles per minute in the following analyses. These intervals were chosen in order to sample smiling from two different portions of the interview that were neither close to the beginning of the interview when discomfort would be greatest nor close to the end of the interview when interviewer and subject would be disengaging. In preliminary analyses using a set of 18 videotapes drawn from a different sample of early adolescents, the intraclass correlation interrater reliability (Shrout and Fleiss 1979) for counts of broad smiles made by the two graduate student raters was 0.91. Because of this excellent reliability, only one of these two raters made smile counts for each interview.

Results

Table 1 presents the mean ages, family socioeconomic status, Wechsler IQ scale (Matarazzo 1972) scores, and sex ratios for each of the three groups of offspring in childhood and adolescence. There were no significant group differences in age, sex, or family socioeconomic status at either age. However, in both childhood and adolescence, the three groups of offspring differed significantly in IQ. On the basis of Tukey-Kramer tests (Hochberg and Tamhane 1987), in childhood IQ was significantly lower in both high-risk groups than in the normal comparison group, and subjects at risk for schizophrenia had significantly lower IQ than subjects at risk for affective disorder. In adolescence, IQ was again significantly lower in both high-risk groups than in the normal comparison group, but the two high-risk groups did not differ significantly.

Tables 2 and 3 present the results of one-way analyses of variance (ANOVAs) and Tukey-Kramer tests comparing the three subject groups in childhood and adolescence. As can be seen from table 2, during childhood there were no significant differences among the groups in the social

### Table 1. Sample characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Offspring of parents with schizophrenia</th>
<th>Offspring of parents with affective disorder</th>
<th>Normal comparison children</th>
<th>p¹</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Childhood</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean age in years (SD)</td>
<td>8.7 (1.9)</td>
<td>9.3 (1.8)</td>
<td>9.0 (1.8)</td>
<td>0.36</td>
</tr>
<tr>
<td>Mean socioeconomic status² (SD)</td>
<td>93.8 (19.7)</td>
<td>89.1 (20.9)</td>
<td>88.7 (11.2)</td>
<td>0.26</td>
</tr>
<tr>
<td>Mean Wechsler IQ score³ (SD)</td>
<td>95.4x (13.7)</td>
<td>102.9y (14.8)</td>
<td>111.3x (11.6)</td>
<td>0.001</td>
</tr>
<tr>
<td>Number of males: females</td>
<td>24:22</td>
<td>18:21</td>
<td>37:28</td>
<td>0.57</td>
</tr>
<tr>
<td><strong>Adolescence</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean age in years (SD)</td>
<td>14.9 (2.1)</td>
<td>15.9 (2.1)</td>
<td>15.3 (2.2)</td>
<td>0.13</td>
</tr>
<tr>
<td>Mean socioeconomic status² (SD)</td>
<td>95.3 (20.2)</td>
<td>90.0 (21.8)</td>
<td>89.5 (11.6)</td>
<td>0.28</td>
</tr>
<tr>
<td>Mean Wechsler IQ score³ (SD)</td>
<td>92.4x (15.1)</td>
<td>97.3x (17.3)</td>
<td>110.9x, y (13.5)</td>
<td>0.001</td>
</tr>
<tr>
<td>Number of males: females</td>
<td>20:12</td>
<td>16:19</td>
<td>35:23</td>
<td>0.29</td>
</tr>
</tbody>
</table>

¹ Significance levels based on one-way analyses of variance for age, socioeconomic status, and IQ and on χ² tests for sex.
² Socioeconomic status based on the Hollingshead Index of Social Position (Hollingshead and Redlich 1959) score of the father of the study offspring in each family; higher scores reflect lower socioeconomic status.
³ Means sharing the same subscript (x or y) differed significantly at p < 0.05 in Tukey-Kramer tests (Hochberg and Tamhane 1987).
Table 2. Measures of social and affective deficits: Scores of children at risk for schizophrenia and affective disorder and of normal children

<table>
<thead>
<tr>
<th>Measure</th>
<th>Offspring of parents with schizophrenia</th>
<th>Offspring of parents with affective disorder</th>
<th>Normal comparison children</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social competence</td>
<td>0.17 (46) 0.83</td>
<td>0.00 (39) 0.78</td>
<td>-0.11 (65) 0.79</td>
</tr>
<tr>
<td>Affective flattening</td>
<td>0.42 (44) 0.48</td>
<td>0.38 (38) 0.45</td>
<td>0.46 (62) 0.59</td>
</tr>
<tr>
<td>Smiles/minute</td>
<td>0.58 (44) 0.64</td>
<td>0.63 (39) 0.86</td>
<td>0.72 (63) 0.72</td>
</tr>
</tbody>
</table>

Note.—SD = standard deviation.

1 Significance levels based on one-way analyses of variance.
2 Higher scores reflect greater pathology.
3 Data in this row drawn from Dworkin et al. (1991).

Table 3. Measures of social and affective deficits: Scores of adolescents at risk for schizophrenia and affective disorder and of normal adolescents

<table>
<thead>
<tr>
<th>Measure</th>
<th>Offspring of parents with schizophrenia</th>
<th>Offspring of parents with affective disorder</th>
<th>Normal comparison adolescents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Social competence</td>
<td>0.49xy (32) 0.70</td>
<td>-0.06x (35) 0.94</td>
<td>-0.18y (58) 0.74</td>
</tr>
<tr>
<td>Affective flattening</td>
<td>1.10xy (28) 0.88</td>
<td>0.62x (33) 0.65</td>
<td>-0.08y (45) 0.59</td>
</tr>
<tr>
<td>Smiles/minute</td>
<td>0.49xy (29) 0.62</td>
<td>0.93x (32) 0.74</td>
<td>0.97y (43) 0.71</td>
</tr>
</tbody>
</table>

Note.—SD = standard deviation.

1 Means sharing the same subscript differ significantly at p < 0.05 in Tukey-Kramer tests (Hochberg and Tamhane 1987)
2 Significance levels based on one-way analyses of variance
3 Higher scores reflect greater pathology.
4 Data in this row drawn from Dworkin et al. (1991).

competence, affective flattening, and smiling measures. However, as can be seen from table 3, adolescents at risk for schizophrenia had significantly poorer social competence, greater affective flattening, and reduced smiling compared with adolescents at risk for affective disorder and normal comparison adolescents.

Analyses of covariance (ANCOVAs) were conducted to determine whether the three groups of offspring differed in social competence, affective flattening, and smiling in adolescence after adjusting for the corresponding symptom in childhood. Significant group differences were found for social competence ($F = 7.30, p < 0.001$) and affective flattening ($F = 3.79, p < 0.05$); for smiling, the group differences did not achieve conventional levels of statistical significance ($F = 2.62, p = 0.08$). The analyses indicate that the three groups of offspring differed significantly in the extent to which social competence and affective flattening changed from childhood to adolescence, and that there was a trend for the groups to differ in the extent to which smiling changed from childhood to adolescence. In additional ANCOVAs examining each of the pairs of groups, worsening of social competence and increase in affective flattening from childhood to adolescence in the offspring at risk for schizophrenia differed significantly from the childhood-to-adolescence change in these measures in the offspring at risk for affective disorder and in the normal comparison offspring. The latter two groups did not differ significantly from each other.
The Pearson correlations among the measures of social and affective deficits in childhood and adolescence and the measures of childhood attentional and neuromotor dysfunction are presented in Table 4 for the offspring at risk for schizophrenia. As can be seen from the table, the measures of childhood attentional and neuromotor dysfunction were not significantly correlated with either social competence or affective flattening at the same testing round in childhood. Greater attentional and neuromotor dysfunction, however, significantly predicted poorer social competence and greater affective flattening 6 years later in adolescence. For smiling, the results were different; both greater childhood attentional and neuromotor dysfunction were significantly associated with reduced smiling in childhood, but only neuromotor dysfunction significantly predicted reduced smiling in adolescence.

On the basis of these correlations, multiple regression analyses were conducted to compare the childhood precursors of adolescent social competence, adolescent affective flattening, and adolescent smiling. In each analysis, three childhood measures were examined in the prediction of the adolescent measure: attentional dysfunction, neuromotor dysfunction, and the corresponding childhood symptom measure (social competence, affective flattening, or smiling).

The results of the three multiple regression analyses are presented in Table 5. In each of these analyses, the childhood symptom measure was entered first and the contribution of attentional and neuromotor dysfunction to predicting residualized change in each of the three symptom measures controlling for initial status was then examined. Although other approaches to the analysis of change have been proposed, this method is appropriate when initial status and the predictors of change are uncorrelated (Werts and Linn 1970), as was true for social competence and affective flattening in the present data. As can be seen from the table, adolescent social competence was significantly predicted by childhood social competence and, after controlling for this relationship, by childhood attentional dysfunction (but not by childhood neuromotor dysfunction). Adolescent affective flattening was significantly predicted by childhood affective flattening and, after controlling for this relationship, by childhood attentional dysfunction (but not by childhood neuromotor dysfunction). Adolescent smiling, however, was not significantly predicted by childhood smiling, and neither attentional nor neuromotor dysfunction significantly contributed to its prediction after controlling for the nonsignificant effect of childhood smiling.

Table 4. Pearson correlations among measures in children and adolescents at risk for schizophrenia

<table>
<thead>
<tr>
<th></th>
<th>Childhood</th>
<th>Adolescence</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AD</td>
<td>ND</td>
</tr>
<tr>
<td>Childhood</td>
<td>AD</td>
<td>—</td>
</tr>
<tr>
<td></td>
<td>ND</td>
<td>36</td>
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<tr>
<td></td>
<td>SC</td>
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<tr>
<td></td>
<td>AF</td>
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<td></td>
<td>S/M</td>
<td>35</td>
</tr>
<tr>
<td>Adolescence</td>
<td>SC</td>
<td>26</td>
</tr>
<tr>
<td></td>
<td>AF</td>
<td>24</td>
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<td></td>
<td>S/M</td>
<td>24</td>
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</tbody>
</table>

Note.—AD = attentional dysfunction; ND = neuromotor dysfunction; SC = social competence; AF = affective flattening; S/M = smiles per minute.

\(^1\) Figures below the diagonal are the numbers of subjects on which the corresponding correlations are based.
\(^2\) Higher scores reflect greater pathology for all of the measures except for smiles/minute.
\(^3\) \(p < 0.05\), two-tailed.
\(^4\) \(p < 0.01\), two-tailed.
\(^5\) \(p < 0.001\), two-tailed.
Table 5. Multiple regression analyses

<table>
<thead>
<tr>
<th>Measure</th>
<th>b</th>
<th>β</th>
<th>t</th>
<th>p</th>
<th>R</th>
<th>F</th>
<th>(df)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adolescent social competence</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>1. Childhood social competence</td>
<td>0.50</td>
<td>0.48</td>
<td>2.64</td>
<td>0.01</td>
<td>0.48</td>
<td>6.99</td>
<td>(1,23)</td>
<td>0.01</td>
</tr>
<tr>
<td>2. Childhood social competence</td>
<td>0.37</td>
<td>0.35</td>
<td>2.07</td>
<td>0.05</td>
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<tr>
<td>Attentional dysfunction</td>
<td>0.11</td>
<td>0.45</td>
<td>2.69</td>
<td>0.01</td>
<td>0.65</td>
<td>8.05</td>
<td>(2,22)</td>
<td>0.002</td>
</tr>
<tr>
<td>Adolescent affective flattening</td>
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<tr>
<td>1. Childhood affective flattening</td>
<td>1.40</td>
<td>0.58</td>
<td>3.29</td>
<td>0.004</td>
<td>0.58</td>
<td>10.81</td>
<td>(1,21)</td>
<td>0.004</td>
</tr>
<tr>
<td>2. Childhood affective flattening</td>
<td>1.04</td>
<td>0.43</td>
<td>2.51</td>
<td>0.02</td>
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<tr>
<td>Neuromotor dysfunction</td>
<td>1.71</td>
<td>0.41</td>
<td>2.35</td>
<td>0.03</td>
<td>0.69</td>
<td>9.31</td>
<td>(2,20)</td>
<td>0.001</td>
</tr>
<tr>
<td>Adolescent smiling</td>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Childhood smiling</td>
<td>0.48</td>
<td>0.36</td>
<td>1.76</td>
<td>0.09</td>
<td>0.36</td>
<td>3.08</td>
<td>(1,21)</td>
<td>0.09</td>
</tr>
</tbody>
</table>

Two aspects of these multiple regression analyses require additional consideration. As the tables show, the sample sizes on which the analyses were based were somewhat different for each of the different measures and in childhood versus adolescence. This was a result of videotape deterioration as well as some subject attrition at individual testing rounds. These missing data account for some discrepancies when the results of different analyses are compared. For example, on the basis of the correlations presented in table 4, childhood smiling would be expected to make a significant contribution in the multiple regression analysis of the precursors of adolescent smiling. However, this did not occur because this multiple regression analysis was conducted only on those subjects for whom there were no missing data for childhood smiling, adolescent smiling, attentional dysfunction, and neuromotor dysfunction, a somewhat smaller sample than that on which the correlation between the first two of these measures was based. Similarly, a nonsignificant correlation between childhood and adolescent affective flattening is presented in table 4, but in the multiple regression analysis of the precursors of adolescent affective flattening, which is again based on a smaller sample than the correlation, childhood affective flattening makes a significant contribution.

The second aspect of the regression analyses that must be considered is that in predicting adolescent affective and social deficits, the corresponding childhood clinical symptom measure was used to control for initial status. However, as can be seen from the correlations presented in table 4, childhood smiling, not childhood social competence, is the childhood clinical symptom measure that is most highly correlated with adolescent social competence. Likewise, childhood social competence, not childhood affective flattening, is the childhood clinical symptom measure that is most highly correlated with adolescent affective flattening. Such findings suggest that it is possible that the measures of social and affective deficits may not be measuring the same underlying variables in childhood and in adolescence. It could therefore be argued, for example, that childhood social competence may be a better control for initial status than childhood affective flattening in predicting adolescent affective flattening.

In order to address the sample size differences in the correlational and multiple regression analyses and the possibility that in predicting the adolescent measures the corresponding childhood measure may not be the best control for initial status, a second set of multiple regression analyses was conducted. These analyses differed from the analyses presented in table 5 in two ways. All three childhood clinical symptom measures—social flattening, social competence, and smiling—were entered as a set in the first step of the analyses. This step controlled for initial status in examining the contribution of attentional and neuromotor dysfunction in predicting each of the adolescent measures of social and affective deficits. In addition, these regression analyses were conducted by examining cases with complete data on the pairs of variables examined in the analysis regardless
of whether the cases had missing data on any other variables ("pairwise" deletion of missing data; Norusis 1985). The principal results of this second set of regression analyses were identical to those presented in table 5. After controlling for all three childhood symptom measures, adolescent social competence was significantly predicted by childhood attentional dysfunction (but not by childhood neuromotor dysfunction), adolescent affective flattening was significantly predicted by childhood neuromotor dysfunction (but not by childhood attentional dysfunction), and neither attentional nor neuromotor dysfunction significantly contributed to the prediction of adolescent smiling.

Discussion

In the present analyses, social competence, affective flattening, and smiling were assessed at two different ages in the offspring of parents with schizophrenia and affective disorder. Although none of these variables significantly differentiated the groups during childhood, in adolescence the subjects at risk for schizophrenia had significantly poorer social competence, greater affective flattening, and reduced smiling compared with the subjects at risk for affective disorder and the normal comparison subjects. To determine whether different processes underlie these three symptoms, correlational and multiple regression analyses were conducted in which the childhood precursors of social competence, affective flattening, and smiling in adolescents at risk for schizophrenia were compared. The predictors of childhood-to-adolescence change in social competence and in affective flattening were found to differ: controlling for initial status, childhood attentional dysfunction (but not childhood neuromotor dysfunction) significantly predicted adolescent social competence, and childhood neuromotor dysfunction (but not childhood attentional dysfunction) significantly predicted adolescent affective flattening. Although the small sample size on which these multivariate analyses are based requires that the results be interpreted with caution, the data provide support for the hypothesis proposed by Strauss et al. (1974)—that these different types of schizophrenia symptoms have separate underlying processes.

We examined smiling because it is an interpersonal behavior that has been considered both an aspect of social competence and a component of affective flattening in recent research on schizophrenia (Dworkin 1992). We hoped that our analyses would reveal whether, with respect to its childhood precursors, smiling was more similar to reported social competence or to observed affective flattening (which in fact includes "unchanging facial expression" as one of its components). Our results, however, did not resolve this question. The pattern of results for our measure of smiling in childhood and in adolescence was different from the results for the measures of affective flattening and social competence. In childhood, smiling was significantly correlated with both attentional and neuromotor dysfunction (whereas the measures of affective flattening and social competence were not), but neither attentional nor neuromotor dysfunction predicted adolescent smiling after controlling for initial status. (This may be a result of the significant correlations between childhood smiling and neuromotor and attentional dysfunction. In the presence of such correlations, the contribution of attentional and neuromotor dysfunction to predicting adolescent smiling in the multiple regression analysis will be underestimated [Werts and Linn 1970].) Although there were no significant group differences in our measure of childhood smiling, based on its appreciable correlation with childhood neuromotor dysfunction it is possible to speculate that smiling in children at risk for schizophrenia may be a subtle indicator of an affective deficit and its underlying process. The significant correlation between childhood neuromotor dysfunction and adolescent smiling is consistent with this suggestion.

An important aspect of the results is that attentional and neuromotor dysfunction in childhood predicted social competence and affective flattening in adolescence but were not associated with these measures concurrently. As we discussed above, it is possible that the childhood and adolescent measures are not iso-morphic, but it is also possible that there is a temporal lag in the relationship between attentional dysfunction and social competence and between neuromotor dysfunction and affective flattening. These longitudinal relationships could be a result of various processes, for example, neurodevelopmental mechanisms (e.g., Breslin and Weinberger 1990), gene regulation (e.g., Pogue-Geile 1991), or cumulative interference with social development (e.g., Comblatt et al. 1992). Our data provide support for such longitudinal approaches to the development of schizophrenia, but they do not reveal
the nature of the longitudinal processes involved.

Although it could be argued that the group differences in IQ in childhood and adolescence may have played a role in our results, existing methods for controlling for differences such as these are problematic (Meehl 1971; Chapman and Chapman 1973). It is important to emphasize, however, that there were no group differences in social competence, affective flattening, and smiling during childhood, when all three groups differed from each other in IQ, and that during adolescence there were significant differences between the two high-risk groups for all three of these measures, but not for IQ.

At the time of the videotaped psychiatric interviews, the offspring at risk for schizophrenia and affective disorder were not appreciably into the age period of risk for these disorders. It will be important, in following these offspring into adulthood, not only to continue examining the longitudinal development of social and affective deficits, but also to investigate specific psychiatric diagnoses. Structured diagnostic interviews are being conducted in ongoing testing rounds at which these offspring are older (Squires-Wheeler et al. 1989), which will make it possible to assess the extent to which social and affective deficits—alone and in combination—predict psychiatric diagnosis. Although it is likely that the group differences in the present analyses remain characteristic of those subjects who actually develop schizophrenia.

The results of our analyses are consistent with a multidimensional approach to the symptoms of schizophrenia and their underlying processes (Strauss et al. 1974, 1978; Crow 1980, 1985; Dworkin et al. 1988, 1991; Carpenter and Buchanan 1989; Carpenter 1992). But the extent to which these processes are relevant to a variety of mental disorders or are specific to schizophrenia has not been adequately addressed. Social and affective deficits do not appear to be specific to individuals with schizophrenia (Carpenter et al. 1985; Sommers 1985) and characterize many of the mental disorders described in DSM-III-R (American Psychiatric Association 1987) (Thoits 1985). In the present investigation, however, social and affective deficits were specific to adolescents at risk for schizophrenia. Our data therefore suggest that these symptoms (and their underlying processes) have greater specificity in individuals vulnerable to schizophrenia than in patients who have already developed the disorder.

The aim of the present analyses was to determine whether social and affective deficits in schizophrenia have different childhood precursors and in so doing to begin to identify the processes underlying these fundamental symptoms of schizophrenia (Strauss et al. 1974). On the basis of our results and previous research, we can provide a very preliminary, but we hope heuristic, set of hypotheses regarding these underlying processes. Genetic influences appear to play an important role in the pathogenesis of negative symptoms in schizophrenia (Dworkin and Lenzenweger 1984; Gottesman et al. 1987; McGuffin et al. 1987; Cannon et al. 1990), including anhedonia (Berenbaum et al. 1990; Clementz et al. 1991; Grove et al. 1991). Such influences also appear to play a role in social deficits in schizophrenia (Kendler et al. 1982; Dworkin et al. 1987, 1988; Grove et al. 1991), and these genetic influences on affective and social deficits may be at least partially independent (Dworkin et al. 1988). In the present data, attentional and neuro-motor dysfunction in children at risk for schizophrenia were significantly correlated, but the magnitude of the correlation ($r = 0.37$) suggests that these two well-researched indicators of the predisposition to develop schizophrenia are also partially independent.

Based on these findings, it can be proposed that a genetically influenced process leads to the development of attentional dysfunction (Cornblatt et al. 1988) in children who are at risk for schizophrenia and that this attentional dysfunction is a precursor of later social deficits in these individuals (Cornblatt et al. 1990, 1992; Erlenmeyer-Kimling et al. 1993). It can also be proposed that a second, partially independent, genetically influenced process
leads to early structural abnormalities of the brain (Crow 1980, 1985) and neuromotor dysfunction and subtle affective deficits (e.g., reduced smiling) in children who are at risk for schizophrenia and that this constellation of neurodevelopmental and early affective deficits is the precursor of later affective deficits in these individuals. Of course, these suggestions provide a very partial outline of the processes underlying social and affective deficits in schizophrenia, and they must be supplemented with more specific hypotheses about the interrelationships between these deficits and neuromotor and attentional dysfunction.

References


Orvaschel, H.; Mednick, S.; Schulzinger, F.; and Rock, D. The children of psychiatrically disturbed parents: Differences as a function of the sex of the dis-


Strauss, J.S. Schiz-affective disorders: “Just another illness” or key to understanding the psychoses? Psychiatry Clinica, 16:286-296, 1983.


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