Perception of parent–child relationships in high-risk families, and adult schizophrenia outcome of offspring

Jason Schiffmana,*, Joseph LaBriea, John Cartera, Tyrone Cannonb, Fini Schulsingerc, Joseph Parnas, Sarnoff Mednick

aUniversity of Southern California, Social Science Research Institute, Los Angeles, CA 90089-0375, USA
bUniversity of California, Los Angeles, California, USA
cThe Institute of Preventive Medicine, Copenhagen Hospital Corporation, Denmark

Received 2 November 2000; received in revised form 30 August 2001; accepted 28 September 2001

Abstract

The current investigation examines perceived family relationships prior to the onset of psychopathology in a sample at high-risk for schizophrenia. Previous research suggests that environmental factors, such as family relationships, may contribute to later schizophrenia in high-risk individuals. This investigation extends work by Burman et al. [Burman B, Mednick SA, Machon RA, Parnas J, Schulsinger F. Children at high risk for schizophrenia: parent and offspring perceptions of family relationships. Journal of Abnormal Psychology 1987;96(4):364–6] by examining high-risk subjects from a longitudinal data set who had not yet decompensated to schizophrenia at the time of the Burman study. Findings suggest that having positive relationships with both the mother and father may be protective against schizophrenia among HR children.

Keywords: Schizophrenia; Premorbid; Family relationships; High-risk; Prospective

1. Introduction

Burman et al. (1987) found significantly less satisfactory family relationships among the offspring of schizophrenic mothers ("high-risk" or "HR") who later developed schizophrenia as compared to high-risk individuals who developed schizotypal personality disorder (SPD) or no mental illness (NMI). The authors suggest a buffering effect of positive family relationships for individuals at genetic risk for schizophrenia. The authors conducted this study when subjects averaged 25 years; they were not through the risk period for schizophrenic onset.

The Burman et al. (1987) study only included 16 schizophrenic subjects diagnosed in 1972. This sample size was insufficient for some analyses. The current investigation examines 1986–1989 lifetime diagnoses for all subjects in the same sample, providing larger groups for analysis (31 schizophrenics, 36 SPD, and 68 NMI). The larger n makes more reliable analyses possible.

1.1. Adoption and institutional rearing studies

Tienari (1991) reported that the offspring of schizophrenics given up for adoption later develop schizophrenia more often if placed in a disturbed adoptive family than if placed in a healthy, "protective" family environment. Subjects with non-schizophrenic biological parents ("low risk" or "LR") were not, however, more likely to develop psychopathology when placed with disturbed adoptive families. Other researchers also report increased risk of future psychopathology in high-risk children separated from their parents during childhood (Quinton et al., 1985), and, more specifically, increased risk of schizophrenic symptoms (Walker et al., 1981). This pattern holds only for high-risk, not low-risk, children. Cumulatively, these studies suggest that a HR child may be especially vulnerable to unsatisfactory family relationships.

1.2. Perception of family relationships

Burman et al. (1987) found HR families to report worse family relationships than low-risk families. Further, families with children who later became schizophrenic
perceived both mother and father relationships as worse than families with children who later developed schizotypal personality disorder or no mental illness. The SPD and NMI subjects did not differ significantly from each other.

A study by Nettelbladt et al. (1996) found that patients with schizoaffective disorders reported poor parental relations. Scott et al. (1993) found worse long-term outcome for schizophrenic patients with poor perceptions of family relationships, as compared to schizophrenics with more positive perceptions of family relationships. Walker et al. (1989) found that high-risk children exposed to parental maltreatment show greater behavioral dysfunction than high-risk children not exposed to parental maltreatment. Finally, evidence suggests that high levels of critical attitudes in the family predict onset of schizophrenia-spectrum disorders in adolescents (Valone et al., 1983).

1.3. Present investigation

The current study analyzes data from a 38-year prospective longitudinal study investigating the pre-morbid correlates of schizophrenia among Danish children. Measures from this investigation were assessed prior to age of risk. Though the Danish psychiatrist noted a variety of symptoms in the children in 1962, in no case did the number and/or quality of symptom rise to a level suggesting a psychotic diagnosis. Danish psychiatric diagnoses were, and still are, conservative. Our research group followed subjects from adolescence to adulthood. This method of data collection reduces the likelihood of the adult clinical picture influencing the perception of early childhood events, as is possible in retrospective studies.

In view of the literature, we hypothesize that, among high-risk individuals, high levels of perceived family discord in adolescence will relate to increased rates of schizophrenic adult outcome. We predict that the rate of schizophrenic outcome will be greater in high-risk offspring with more poorly perceived family relations as compared to those with less poorly perceived family relations.

2. Method

2.1. Sample

To identify factors involved in the etiology of schizophrenia, Mednick and Schulsinger, in 1962, began a longitudinal study of 207 offspring of mothers with schizophrenia and a control group of 104 children of parents with no psychiatric illness (Mednick & Schulsinger, 1965). Low-risk participants were matched with high-risk participants for age, sex, social class, institutional rearing during childhood, and urban-rural residence. At the inception of the study, the mean age of the subjects was 15.1 years, with a range from 9 to 20. Two-hundred and seven HR (89 female, 118 male), and 104 LR (44 female, 60 male), participated.

2.2. Diagnostic assessment

Psychiatric functioning for these subjects was evaluated in 1972–1974 and again in 1986–1989. The diagnostic procedure in 1972–1974 included two structured psychiatric interviews from which the interviewer arrived at a diagnosis: the Present State Examination (PSE; Wing et al., 1974) and the Current and Past Psychopathology Scales (Endicott & Spitzer, 1972).

The 1986–1989 diagnostic procedure included: the Schedule for Affective Disorders and Schizophrenia-Lifetime Version (Spitzer & Endicott, 1977); the PSE; the PSE Syndrome Checklist for current and lifetime psychopathology; the PSE lifetime ratings of psychiatric symptoms; the Personality Disorder Examination (Loranger et al., 1985); and the Scales for the Assessment of Positive and Negative Symptoms (Andreasen, 1983, 1984). In addition, the entire sample was screened for psychiatric hospitalizations through the National Psychiatric Register (Institute of Psychiatric Demography, Aarhus, Denmark). Six psychologists and psychiatrists1 conferred to assign a lifetime DSM-III-R diagnosis to each subject based on all the data derived from the assessment processes. The assessment of the lifetime diagnoses in the HR group yielded 31 schizophrenia diagnoses, and 36 schizotypal diagnoses (Parnas et al., 1993). We also identified 68 subjects free of mental illness.

2.3. Procedure

In 1962 two highly experienced, leading Danish social workers (Lise Maaloe and Grethe Skat-Andersen) conducted an extensive psychosocial interview with the person with whom the child spent most of his or her life. A leading Danish psychiatrist (Fini Schulsinger) also interviewed each child regarding his or her clinical status and family relationships. Data for this study are based on coding completed by the interviewers at the time of the interview. Questions were standardized so that ratings could be entered in a computer. We constructed two scales by combining dichotomous items from the interviews that related to offspring’s family relationships in 1962. The Perception of Relationship with Mother (Mother Scale) and the Perception of Relationship with Father (Father Scale) included all relevant items from the interviews. Burman and colleagues originally developed these scales (Burman et al., 1987).

1 Psychologists and psychiatrists included Sarnoff Mednick, Fini Schulsinger, Trone Cannon, Josef Parnas, Hanne Schulsinger, and Bjorn Jacobson.
The Mother and Father Scales contain items that relate to the children’s attitudes toward their mothers and fathers, the nature of the relationship as perceived by the parents, and frequency of contact (Table 1). Each item on the scales was scored either 0 or 1. The range of scores for the Mother Scale was from 0 to 5, and for the Father Scale, from 0 to 4. The Father Scale contained one less item. High scorers perceived the least satisfactory relationships. Both scales yield modest internal reliability (Mother Scale, \( \alpha = 0.70 \); Father Scale, \( \alpha = 0.78 \)).

It should be emphasized that families answered the questions in the scales prior to overt signs of mental illness in the child. The coding of the items was completed during the 1962 assessment. Further, questions were selected from the interviews blinded to how the questions relate to outcome. All apparently relevant interview questions were included.

### 3. Results

We first performed an analysis comparing the HR group to the LR group on the Mother and Father Scales. Not surprisingly, HR subjects had significantly worse relationships with their schizophrenic mothers than LR subjects with their normal mothers (HR vs. LR, \( t = 10.81, P < 0.001 \)). This finding is consistent with the finding of Burman et al. (1987). HR subjects show a trend towards having significantly worse relationships with their fathers than LR subjects (HR vs. LR, \( t = 1.86, P < 0.07 \)).

We performed the remaining analyses within the HR group only. Burman et al. (1987) analyzed the relationships between the mother and the offspring, and the father and the offspring, separately. With this method of analysis, if a HR child had a positive relationship with the father, we would hypothesize a relatively good outcome for the child. On the other hand, if a HR child had a poor relationship with the mother, we might expect a relatively poor outcome for that child. Prediction is unclear for children with both good and bad relationships with parents. In view of this argument, we determined to identify cases with both positive mother and positive father relationships, comparing these cases to cases with both negative mother and negative father relationships. Such comparisons permit less equivocal interpretations.

The data are non-normally distributed with heavy tails (kurtosis of mother scale = 0.797 S.E. = 0.287; skewness of mother scale = 1.280, S.E. = 0.144; kurtosis of father scale = −1.018, S.E. = 0.280, skewness of father scale = 0.599, S.E. = 0.140). Because of the skewed nature of the data, as well as the interest in examining the mother and father relationships together to gain a complete family picture, we divided HR subjects into groups based on their Mother and Father Scale scores. Analyses using categorization of subjects, as opposed to continuous measures, may be less influenced by non-normal distributions.

We split Mother and Father Scale scores for the entire HR sample at the median. Subjects scoring equal to, or above, the median on the Mother Scale (median = 2.5), and scoring above the median of the Father Scale (median = 0) were categorized as having “poor” parental relationships, while those scoring below the median on the Mother Scale, and at the median on the Father Scale were categorized as having “good” parental relationships (Table 2). (It should be noted that subjects only needed one endorsement on the Father Scale to be considered in the “poor” father group.) The terms “poor” and “good” are used relatively, as all mothers in the HR group suffered from schizophrenia.

#### 3.1. Good vs. poor parental relations: child outcome

We performed these analyses using the 1986–1989 lifetime diagnoses. As seen in Table 2, among the HR children, poor relationships with both parents are associated with elevated rates of schizophrenia in offspring. Of HR individuals with poor parental relations, 40.7%

### Table 1

<table>
<thead>
<tr>
<th>Family relationship scalesa</th>
<th>Perception of Relationship with Mother Scale</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1. Attitude toward mother is not positive (P)</td>
</tr>
<tr>
<td></td>
<td>2. Child and mother do not get along well (S)</td>
</tr>
<tr>
<td></td>
<td>3. Mother often scolds child (S)</td>
</tr>
<tr>
<td></td>
<td>4. Child does not tell mother his/her troubles (S)</td>
</tr>
<tr>
<td></td>
<td>5. Frequency of contact with mother is not regular (P)</td>
</tr>
<tr>
<td></td>
<td>Perception of Relationship with Father Scale</td>
</tr>
<tr>
<td></td>
<td>1. Attitude toward father is not positive (P)</td>
</tr>
<tr>
<td></td>
<td>2. Effect of contact with father is not positive (P)</td>
</tr>
<tr>
<td></td>
<td>3. Frequency of contact with father is not regular (P)</td>
</tr>
<tr>
<td></td>
<td>4. Father is described as weak, ineffective, reserved (P)</td>
</tr>
</tbody>
</table>

a P, based onitem from psychiatric interview with subject; S, based on item from social work interview with parent.

### Table 2

<table>
<thead>
<tr>
<th>Quality of parental relationships and offspring lifetime diagnosesa</th>
<th>Schizophrenia</th>
<th>SPDb</th>
<th>NMIc</th>
<th>nsd</th>
</tr>
</thead>
<tbody>
<tr>
<td>Good</td>
<td>4 (9.8%)</td>
<td>10 (24.4%)</td>
<td>27 (65.9%)</td>
<td>41 (100%)</td>
</tr>
<tr>
<td>Mixed</td>
<td>9 (21.4%)</td>
<td>9 (21.4%)</td>
<td>24 (57.1%)</td>
<td>42 (100%)</td>
</tr>
<tr>
<td>Poor</td>
<td>11 (40.7%)</td>
<td>6 (22.2%)</td>
<td>10 (37.0%)</td>
<td>27 (100%)</td>
</tr>
</tbody>
</table>

a Percentages are calculated across rows
b SPD, schizotypal personality disorder.
c NMI, no mental illness.
d n’s reflect subjects who have both Mother and Father Scale scores. Cases where one Father or Mother Scale score was missing were considered missing data.
became schizophrenic; among HR offspring with good parental relations, only 9.8% became schizophrenic (these percentages are high because the N for the NMI Group is limited).

The HR offspring who received an adult diagnosis of schizophrenia experienced significantly poorer relations with both of their parents in comparison with (1) HR SPDs \( \chi^2 (1) = 4.01, P < 0.05 \); and (b) HR offspring who had an outcome of No Mental Illness \( \chi^2 (1) = 9.51, P < 0.01 \). HR offspring in the SPD Group did not significantly differ from HR offspring in the NMI Group on parental relations \( \chi^2 (1) = 0.58, \text{NS} \). As seen in Table 2, outcomes for the “mixed group” (good relationship with one parent, poor with the other) in most cases fall in between outcomes for the good–good and poor–poor groups.

Note that in the total HR sample of 207, 16.1% had an outcome of schizophrenia. Among those HR subjects with good relations with both parents (\( n = 57 \)), only 7.0% (\( n = 4 \)) suffered an adult schizophrenic outcome. Among those HR subjects with poor relations with both parents (\( n = 47 \)), 23.4% (\( n = 11 \)) suffered an adult schizophrenia outcome. HR subjects with poor parental relationships develop schizophrenia more often than HR subjects with good parental relationships \( \chi^2 (1) = 5.60, P < 0.05 \). Tienari (1991) reports similar findings; HR children reared in a harmonious family have a relatively low risk of becoming schizophrenic.

4. Discussion

Table 2 and the subsequent analyses suggest an association (within the HR group) between positive relationships with both parents and NMI and SPD outcomes, as well as an association between negative relationships with both parents and schizophrenia. Only 7.0% of the total sample of HR subjects with good parental relationships developed schizophrenia, whereas 23.4% of HR subjects with poor parental relationships developed schizophrenia. Getting along well with both parents may protect HR individuals from schizophrenia, whereas, poor parental relations may increase risk for future development of schizophrenia.

The difference in family relations between the schizophrenia and SPD individuals in the HR sample further suggests the importance of family relationships in the development of schizophrenia. In light of a diathesis-stress model of schizophrenia, SPD individuals may have a genetic vulnerability to schizophrenia, yet escape environmental stressors contributing to schizophrenic decompensation. We found significantly more positive family relations in the SPD subjects than schizophrenia subjects. Perhaps positive family relationships buffer already at-risk individuals from stressors leading to schizophrenia. Also, the actual stress of a negative family relationship may contribute to schizophrenia in at-risk individuals. Conceivably, avoiding this stress protects at-risk individuals from full schizophrenic development.

4.1. Possible confounding factors

We interpret our findings as suggesting that, among HR individuals, poor parent–child relationships increase risk of offspring schizophrenia. We will call this the “Interactive Hypothesis.” Another interpretation, however, might hold that HR offspring who are genetically predisposed to develop schizophrenia are likely to evidence psychopathological behavior in childhood and adolescence which, in turn, increases risk for poor parent–child relations. This interpretation would view these poor parent–child relations as side effects, not influencing risk of offspring schizophrenia. We will call this the “Genetic Hypothesis.” We test the viability of these hypotheses in the sections to follow.

4.1.1. Genetic influences

A Genetic Hypothesis interpretation of these results suggests that the children with greater genetic liability for schizophrenia have poor relationships with their parents. These children may inherit characteristics that adversely affect family relations. Further, the genetic contribution may also increase the risk for schizophrenia in the offspring, independent of family relations. This interpretation predicts that high genetic liability leads to both schizophrenic outcome and worse family relations. In this alternative model, family relationships would not be causally related to later diagnostic outcome of offspring, but would be associated with having parents with high genetic loading.

A sub-sample of the HR group from this study had both parents diagnosed with a schizophrenia spectrum disorder. These Super High-Risk (SHR) subjects are at higher genetic risk for schizophrenia than individuals with only one parent with a schizophrenia spectrum disorder (Gottesman and Shields, 1982). Comparing SHR (\( n = 30 \)) and HR (\( n = 177 \)) subjects, we tested the hypothesis that increased genetic liability leads to worse family relationships. Within the combined schizophrenia group, there was, however, no significant difference between SHR and HR on offspring-mother relationships \( \chi^2 (1) = 0.69, \text{NS} \), nor offspring-father relationships \( \chi^2 (1) = 1.41, \text{NS} \). SHR individuals with schizophrenia are not significantly more likely to have a poor relationship with their parents than HR individuals with schizophrenia. These results do not support the Genetic Hypothesis.

Additionally, we already alluded to the study by Tienari (1991) suggesting that poor family relationships contribute to risk of schizophrenia in the offspring of schizophrenics. Tienari (1991) found that HR offspring are more likely to develop psychopathology when
adopted into a disturbed family than if placed in a healthy family. The Tienari findings lend support to the Interactive Hypothesis.

4.1.2. Infant attributes

Bell (1968) raises concern regarding the issue of direction of effects in studies of children’s socialization. Unfavorable infant attributes might cause poor family relationships, as the stress of having a poorly behaved child may disrupt parent–child relations (Bell, 1968). Also, unfavorable attributes in infancy may signal later decompensation into schizophrenia. In line with the Genetic Hypothesis, from this perspective, poor parental relations do not influence later schizophrenia, rather, early signs of schizophrenia influence poor parental relations. “Infant attributes” of the HR sample (collected retrospectively during a 1962 interview) were investigated to determine the nature of the relationship between early attributes and perceived parental relationship scale score.

Infant (one year of age) characteristics of attention span, passivity, stubbornness, and temperament were assessed retrospectively at the time of the 1962 assessment. We included all relevant interview items. Each variable was dichotomous, thus allowing for the summation of items to form an “Attribute” Scale Score. We split HR subjects at the median of the Attribute Scale Score, creating two groups, Positive Infant Attributes and Negative Infant Attributes. We compared the Positive and Negative Infant Attribute Groups’ parental relationship grouping. A chi-square analysis failed to detect a significant association between infant characteristics and parental relationships [X^2 (1) = 2.01, NS; Table 3]. Infant attributes do not significantly relate to later adequacy of parent–child relationships. These results do not support the hypothesis that poor early attributes of pre-schizophrenia children negatively affect perception of parental relationships.

4.1.3. Adolescent psychiatric symptoms

Similar to the analyses above investigating infant attributes, adolescent psychiatric status may contribute to poor family relationships. The stress of a disturbed adolescent may disrupt parent–child relations. Previous research suggests that HR children exhibit more signs of psychiatric disturbance than non-HR children. HR adolescents with psychiatric symptoms may signal later decompensation into schizophrenia, and also disrupt parent–child relationships. Again, from the Genetic Hypothesis perspective, poor parental relations do not influence later schizophrenia, rather, early signs of schizophrenia influence poor parental relations.

We derived an index of adolescent psychopathology based on the psychiatrist’s (Fini Schulsinger’s) rating of clinical symptoms observed in the psychiatric interview. The psychiatrist conducted the interviews in 1962–1963 when the social worker also assessed family relations. We factor analyzed all items relating to the subject’s mental status, emotional state, or psychological health after removing items with a large number of missing values or insufficient variance. The first factor combined items suggestive of schizophrenia-like pathology with indices of global adjustment. Items loading greater than 0.40 were weighted equally and summed to form the Psychiatric Symptom Scale (PSS; Table 4). The scale has an internal reliability of 0.82.

We compared HR and LR subjects’ scores on the PSS. As found by previous investigators, HR subjects evidence significantly elevated PSS scores compared to LR subjects (HR vs. LR, \( t = 7.50, P < 0.001 \)). We also tested whether high PSS scores in HR subjects would predict later schizophrenic outcome. We did not find a significant relationship (HR; schizophrenia vs. all other outcomes, \( t = 0.805, \) NS). It seems that many adolescents who evidence psychiatric symptoms do not later develop schizophrenia.

We tested the Genetic and Interactive Hypotheses using the PSS. We asked whether family relations would help determine whether those with high PSS scores would develop schizophrenia. In this analysis we included subjects with schizophrenia and NMI subjects with PSS scores and who were in either Good or Poor Family Relations groups. Fifty HR subjects (15 schizophrenia subjects, 35 NMI) had both complete family

---

**Table 3**

| Quality of parental relationships and infant attributes | Good infant attributes | Poor infant attributes | n's

| Good parental relations | 34 (59.6%) | 23 (40.4%) | 57 (100%)
| Poor parental relations | 20 (45.5%) | 24 (54.5%) | 44 (100%)

*a Percentages are calculated across rows.

*b n's reflect subjects who have both Mother and Father Scale scores and Infant Attribute scores.

**Table 4**

<p>| Adolescent psychiatric interview items |</p>
<table>
<thead>
<tr>
<th>Scale and source</th>
<th>Item</th>
</tr>
</thead>
<tbody>
<tr>
<td>Psychiatric Health</td>
<td>Subject’s opinion of own ability</td>
</tr>
<tr>
<td></td>
<td>Normal modulation of affect</td>
</tr>
<tr>
<td>Psychiatric Interview</td>
<td>Normal train of associations</td>
</tr>
<tr>
<td></td>
<td>Coherent thoughts</td>
</tr>
<tr>
<td></td>
<td>Normal facial expressions</td>
</tr>
<tr>
<td></td>
<td>Subject shows signs of mental illness [–]*</td>
</tr>
<tr>
<td></td>
<td>Intelligence</td>
</tr>
<tr>
<td></td>
<td>Normal speech process</td>
</tr>
<tr>
<td></td>
<td>Normal mood</td>
</tr>
<tr>
<td></td>
<td>Degree of tenseness [–]*</td>
</tr>
<tr>
<td></td>
<td>Favorable prognosis</td>
</tr>
</tbody>
</table>

*a [–], item is reverse-scored.*
relations scores and PSS scores. (Two NMI subjects had missing psychiatric health data.)

We split HR subjects at the median of the PSS score, creating two groups, Good Psychiatric Symptoms and Poor Psychiatric Symptoms. We considered subjects scoring at or below the median in the Good Psychiatric Symptom group (median = 8.333, lower scores indicate less symptoms). We hypothesize that among HR subjects with Good and Poor Psychiatric Symptoms, high levels of perceived family discord in adolescence will relate to increased rates of adult schizophrenic outcome.

Twenty-four HR schizophrenia and HR NMI subjects fell in the Poor Psychiatric Symptom group. Twelve Poor Psychiatric Symptom adolescents had good parental relationships, of those 12, only two developed schizophrenia and 10 had an NMI outcome. Twelve Poor Psychiatric Symptom adolescents had poor parental relationships, of those 12, eight developed schizophrenia and four had an NMI outcome [$\chi^2 (1) = 6.17$, $P < 0.05$]. Those subjects with high psychiatric symptoms and poor family relations developed schizophrenia significantly more than those subjects with high psychiatric symptoms and good family relations. Among adolescents with higher psychiatric symptomatology, quality of family relations seems to influence later psychiatric outcome.

We observe a similar, yet non-significant, pattern among HR subjects with low adolescent psychiatric symptoms. We categorized 26 HR schizophrenia or NMI subjects in the Good Psychiatric Symptom group. Eighteen Good Psychiatric Symptom adolescents had good parental relationships, of the 18, only two developed schizophrenia and 16 had an NMI outcome. Eight Good Psychiatric Symptom adolescents had poor parental relationships, of these, three developed schizophrenia and five had an NMI outcome [$\chi^2 (1) = 2.48$, $P = 0.12$].

To increase the sample size among subjects in the Good Psychiatric Symptom group, we re-analyzed the data combining the “mixed” parental relations group (one good and one bad parental relationship) with the subjects with poor relations with both parents. Comparing the combined “poor and mixed” group to subjects in the “good” group reveals a significant difference. Again, 18 Good Psychiatric Symptom adolescents had good parental relationships; two developed schizophrenia and 16 had an NMI outcome. Twenty Good Psychiatric Symptom adolescents had poor or mixed parental relationships, eight developed schizophrenia and 12 had an NMI outcome [$\chi^2 (1) = 4.08$, $P < 0.05$].

We have noted an association between perception of parental relationships and adult schizophrenia in high-risk offspring. We interpret this association as suggesting that (among these high-risk offspring) good family relationships protect against later adult schizophrenia. As described above, the Genetic Hypothesis might suggest that those HR offspring destined to develop schizophrenia evidenced disturbed behavior in infancy, childhood and adolescence and this disturbed early behavior both sours child–parent relations and accounts for the later psychiatric illness. We attempted to test this second interpretation, but the data analyses failed to support it.

Fini Schulsinger, an experienced, senior Danish psychiatrist, carried out a diagnostic clinical interview with all these individuals in 1962–1963. As mentioned above, he did not find a significant level of psychopathology in these high-risk and low-risk (then) children. It is possible that his threshold for detection of psychopathology was high since he typically worked with adult patients. Conceivably, therefore, psychopathology which existed in these children was not reported. It is difficult to see why that failure to report child/adolescent psychopathology was concentrated in the families with poor perceived parent–child relations. This possibility suggests caution in the attribution of causality to the associations of early family relationships and later development of schizophrenia noted in this study.

Although clearly not definitive, the collective findings from the adolescent psychiatric health data do not support the Genetic Hypothesis that disturbed adolescents cause poor family relationships. More consistent with the Interactive Hypothesis, quality of family relations appears to “interact” with a genetic predisposition towards schizophrenia to predict adult psychiatric outcome regardless of adolescent psychiatric functioning.

5. Conclusions

Considering the entire HR group ($n = 207$), 23.4% of HR subjects in this sample with poor relationships with both parents develop schizophrenia; only 7.0% of high-risk subjects with good parental relationships develop schizophrenia. Although the causal role of family relationships in the development of schizophrenia remains in question, among HR children, positive relationships with both the mother and father seem to protect against schizophrenia.

Acknowledgements

This research was supported by a Research Scientist Award to S.A. Mednick from the National Institute of Mental Health. Grant #: 5 K05 MH 00619

References

Andreasen NC. The scale for the assessment of negative symptoms (SANS). Iowa City: University of Iowa, 1983.

Andreasen NC. The scale for the assessment of positive symptoms (SAPS). Iowa City: University of Iowa, 1984.


