Introduction: The Israeli High-Risk Study

by Shmuel Nagler and Allan F. Mirsky

This issue of the Schizophrenia Bulletin is dedicated to David Rosenthal, Ph.D., Chief of the Laboratory of Psychology and Psychopathology, Intramural Research Program of the National Institute of Mental Health from 1966 to 1977. Dr. Rosenthal conceived this study; and his wisdom, energy, and gentle organizational skills made it possible. Commentaries on this study will be published in a subsequent issue of the Bulletin.

Abstract

The literature on theories of the etiology of schizophrenia (the "nature-nurture dilemma") is reviewed, and the rationale is explicated for a study comparing the development and outcome of kibbutz- and town-reared children of schizophrenic parents. Various possible outcomes of the kibbutz-rearing experience in vulnerable children are discussed. The research team and the time table are described.

David Rosenthal (1963, 1970) classified theories regarding the etiology of schizophrenia into three main categories:

• Monogenetic-biochemical theories.
• Life experience theories.
• Diathesis-stress theories.

Proponents of monogenetic-biochemical theories conceive of schizophrenia, in the tradition of Kraepelin (1971), as a clinical entity transmitted by a single gene. The main thrust of their effort is in biochemical research (e.g., Heath 1959, 1960; Kety 1960) aimed at discovering and characterizing aberrations in cerebral metabolism.

In this view, environmental variables are not afforded the same weight as biological ones: Differences in the course and severity of the disease are due to individual variations in "resistance," which may also be transmitted by genes.

Life experience theories (Lidz et al. 1957; Wynne et al. 1958; Lidz, Fleck, and Cornelison 1965; Wynne 1967, 1968, 1972; Lidz 1972) are strongly influenced by psychoanalytic thinking. Bowlby (1960, 1963, 1969), Spitz (1945, 1946, 1951, 1965), Mahler (1952), and Mahler et al. (1954) all have contributed to the concept of the so-called "schizophrenogenic mother," which was later replaced by the concept of the "schizophrenogenic family" (Bateson et al. 1956; Bateson 1961).

In contrast to Freud himself, who never denied a constitutional-hereditary predisposition to mental illness,1 many of his followers (particularly after World War II in the United States) considered faulty parent-child relations to be the only antecedents of schizophrenia. These include inadequate mothering in early childhood and disordered family interactions characterized by confusing communication patterns and opposite messages, forcing the listener into a "double-bind situation." Unlike the geneticists, the advocates of life experience theories have concentrated their research efforts on the nuclear family rather than on genealogical study.

Those researchers who have favored a diathesis-stress theory have generally shared the view that under

1 Freud used the concept of a complementary relationship (Erganzungsreihe) between endogenous and environmental factors.

Reprint requests should be sent to Dr. A.F. Mirsky, Chief, Laboratory of Psychology and Psychopathology, NIMH, Bldg. 10, Rm. 4C-110, Bethesda, MD 20205.
certain conditions of stress, a genetically determined vulnerability facilitates the expression of the illness. Their work has explored the nature of this predisposition and the stress conditions that interact to bring about the schizophrenic phenotype.

The research strategies employed by the diathesis-stress theorists were categorized by Rosenthal (1970) into four classes: (1) consanguinity studies, which examine the distribution of schizophrenic disorders among blood relatives of schizophrenics as compared to the population at large; (2) twin studies, which compare the incidence of concordance of the disorder in monozygotic vs. dizygotic twin pairs, reared together or apart; (3) retrospective studies, which examine the so-called "premorbid" behavior and development of schizophrenic patients; and (4) prospective studies, which follow children at risk for schizophrenia by virtue of their having a schizophrenic parent. This latest development in research methodology in the field inspired our own research design.

Although not included specifically in this list, adoption and cross-fostering studies are also employed by diathesis-stress theorists. Examples are investigations reported by Heston (1966) and in a series of publications by Rosenthal et al. (1968, 1971).

Consanguinity studies with relatives of schizophrenic patients show a decrease in incidence of the disease with increasingly distant blood kinships (Kallmann 1946, 1950; Gregory 1960; Shields and Slater 1961). Even in second degree and further removed relatives of schizophrenic patients, however, the incidence of this disease is higher than in the general population. These findings suggest a genetic basis of schizophrenic disorders, but can by no means be taken as final proof of a genetic explanation. The degree of consanguinity is confounded with living under similar schizophrenogenic family—or broader—social influences (Lidz and Lidz 1949; Hollingshead and Redlich 1958; Lidz et al. 1959; Kohn 1968).

The maximum degree of genetic influence should be expected in the cotwin of a schizophrenic index twin in monzygotic (MZ) twin pairs (Gottesman and Shields 1966). In spite of the fact that both MZ twins share all genes in common, there is a wide range in reported concordance rates for schizophrenia (Rosenthal 1970), from one extreme of 69-86 percent (Kallmann 1946) to the other extreme of 0-6 percent (Tienari 1968).\(^2\)

The concordance rate in same-sexed dizygotic twins drops sharply to 17.6 percent, approaching the percentage for ordinary sibs (Gottesman and Shields 1982). On the other hand, Slater (cited in Rosenthal 1970) reports a larger difference in concordance rate between these two kinds of relationships—14 percent for twins vs. 5.4 percent for ordinary sibs. If Slater's figures are correct, this might be related to the fact that parents treat twin pairs more alike than they treat ordinary siblings. Moreover, the blurred identity that characterizes twins might be among the nongenetic factors that contribute to the result Slater obtained.

Retrospective studies have sought to discover environmental factors that precipitate the development of schizophrenia. Such studies have sought also to describe the "preschizophrenic" attributes in personality and behavior that appear before the onset of the disease.

These studies have also identified the following as precursors of schizophrenia: gradual decline of IQ; deterioration in scholastic achievement; impairment of speech (taciturnity, soft voice); social isolation (shyness); depressive trends; lack of motivation; and—in contrast—antisocial acting out (mainly found with boys) (Watt et al. 1970; Watt 1972).

Retrospective procedures, which are basically a refinement of the clinicians' practice in taking a case history, have a basic weakness—the fallibility of human memory, particularly when anamnestic material of high ego involvement is derived from interviews with patients or relatives. But even more objective information such as clinical reports and test results from child guidance clinics or schools is of limited value; usually the material has been collected for clinical purposes and may not be useful for research (Garmezy 1974).

The avoidance of these methodological shortcomings has been the main motive for the preference of prospective strategies in research investigating the etiology of schizophrenia.\(^3\) Researchers who favor the
prospective strategy take advantage of the possibility of deciding from the very beginning which behavioral manifestations should be investigated and which assessment procedures should be applied, thus assuming a systematic and uniform way of data collection.

In prospective studies applied to schizophrenia research, the following four steps are generally taken:

• The next step is an investigation into the different courses of development from the starting point of referral to the child guidance clinic to the end point of different diagnostic outcomes. The aim is to detect personality traits and behavioral manifestations that differentiate children who developed schizophrenia from the others. In other words, the aim is to look for precursors of schizophrenia detectable in childhood."  

Robins (1966) found that among male child guidance patients who developed into schizophrenic adults there were high incidences of severe forms of antisocial acting out (such as aggressive acts, particularly against the family: delinquency; and truancy) as the reason for referral. The author emphasizes, however, that these children, unlike those who were later diagnosed as adult sociopaths, did not join juvenile gangs. They failed to "belong" but primarily because of their own hostility. According to Ricks and Berry (1970), extremely socially isolated children, as well as those with severe conduct disorders, are likely to develop schizophrenia (particularly in its chronic form) when the social maladaptations are combined with symptoms indicative of neurological defects. The latter include poor motor coordination, hyperactivity, and impaired attentional span.

This symptom combination in childhood seems to be of more predictive value for later process schizophrenia than any other one. Regarding environmental factors precipitating a schizophrenic outcome, prospective studies lend support to the findings revealed by retrospective methods, particularly regarding the pathogenic influence of mother-child and marital relations.

About half of the children who became chronic schizophrenics had mothers diagnosed as "clearly psychotic" or schizoid. In 40 percent the sick parent-child relationship was characterized as "symbiotic," and in 27 percent the husband-wife relations were seen as "emotional divorce." The two pertinent questions are still open:

• Is a genetically determined biological deficit essential in the etiology of schizophrenia, at least in its chronic course?
• To what extent is a noxious home environment created by a schizophrenic parent responsible for the development of the disease?

In search of answers to these questions, Mednick and Schulsinger (1968, 1970) made a methodologically decisive step forward by introducing the high-risk methods in schizophrenia etiology research. Based on a clearly stated theory (Mednick 1958), these investigators worked with an index and carefully matched comparison group. They used methods to reveal their theoretically derived biological dysfunction in the development of schizophrenia.

The index group consisted of 207 children at high risk for schizophrenia. This criterion is defined genetically, i.e., being the offspring of a mother diagnosed as a severe
case of process schizophrenia. These high-risk subjects were selected in pairs matched for sex, age, social class, and upbringing by institutional vs. family setting. For each subject who would eventually break down, it was anticipated that there would be many comparably high-risk subjects who would remain free from psychopathology. To each high-risk pair, a single low-risk subject (child of a mother without schizophrenic symptomatology) was matched on the above-mentioned variables. A 20-year followup was planned, thus covering the risk period for onset of the disease. A well-organized alarm network was set up to ensure cooperation from the family physician and mental hospitals that would convey information immediately about any contacts with the subjects under study. Evaluated were (1) psychosocial adaptation (by means of a psychiatric interview, a shortened Minnesota Multiphasic Personality Inventory (MMPI) pointing inter alia to premorbid characteristics of schizophrenics, an adjective checklist, and school reports); (2) developmental and rearing conditions (obtained by interviewing parents and using midwives' reports); and (3) intelligence and thought processes (by Wechsler Intelligence Scale for Children and Word Association Test (WAT)).

The findings can be summarized as follows: Index subjects showed more disordered associations, lower achievement in arithmetic, less capacity for continuous concentration, and poorer peer relations. All these differences reached statistical significance. Rated for overall adjustment on a rating scale from 1 to 5 (from poor to good) 24 percent of the high-risk subjects vs. 1 percent of the controls were rated 1 or 2. Special weight was given to psychophysiological measurements of autonomic responsivity because of their relation to Mednick's learning theory of schizophrenic behavior as learned avoidance-behavior, which is reinforced by reducing anxiety. It was therefore expected that compared to the controls, high-risk children would show increased autonomic arousal, slower recovery rate, and proneness to stimulus generalization as the psychophysiological indicators of a high level of anxiety. The predicted slow recovery time was not verified. The index subjects returned to their physiological baseline after a quick and vigorous response, and they did so more quickly than the controls. But the predicted differences in irritability of the autonomic nervous system proved highly significant; index subjects showed shorter latency, less discrimination (overgeneralization), and greater amplitudes of galvanic skin response to stress stimuli. It can be assumed that the unstable conditions of life with a schizophrenic mother must be an unbearable burden for the hyperresponsive autonomous nervous system of the high-risk child and detrimental to his psychosocial development.

Within 5 years from the start of the project, 20 high-risk subjects broke down, presenting symptoms of severe schizophrenic pathology. These 20 subjects (the S or sick group) were compared with other high-risk subjects who showed either no change or some improvement in their mental health (the W or well group); they were also compared with the original low-risk control group by means of the scores obtained in the initial personality assessment. The S group was found to have the largest galvanic skin response (GSR) amplitude to stress stimuli, progressively decreasing response latencies (indicative of negative habituation), a high-risk generalization score, and again the fastest recovery rate. On all these test variables, the differences between the S and W groups were significant. With respect to the recovery rate, the difference between the means of the W group and the low-risk group was minimal. Mednick and Schulsinger (1968) argue that because of this fast rate of recovery the preschizophrenic is more easily, quickly, and thoroughly reinforced for avoidance than is the normal person. Autonomic hypersensitivity, combined with abnormally fast recovery from autonomic imbalance, should therefore be considered the genetically transmitted biological basis of vulnerability for schizophrenia.

On a behavioral level, the following variables significantly differentiated the S and W groups: disturbed chain associations to a single stimulus word (WAT), and higher and more persistent excitability (and consequently more disciplinary problems in the classroom as reported by teachers). These appear to be identifiable premorbid characteristics of schizophrenia.

An environmental factor that differentiated significantly between

7 In such a high-risk population, the incidence rate for schizophrenia is about 10 times higher than in the population at large.

8 Mednick (1968) argues that because of this fast rate of recovery the preschizophrenic is easier, quicker, and thoroughly reinforced for avoidance than is the normal person. Autonomic hypersensitivity, combined with abnormally fast recovery from autonomic imbalance, should therefore be considered the genetically transmitted biological basis of vulnerability for schizophrenia.

9 Meehl (1962) holds a different position: A neural integration-defect termed "schizotaxia" is a necessary but not sufficient condition for development of schizophrenia.
the S and W groups was the child's age at mother's hospitalization and the length of time the child spent with the mother before her psychiatric hospitalization. The length of time spent with a schizophrenic mother seems to influence the symptomatology and course of the disease. Subjects in the S group who had been separated from an extremely disturbed mother at kindergarten age were more likely to develop symptoms of reactive schizophrenia. Subjects separated from relatively less disturbed mothers only after reaching school age developed schizophrenic symptoms closer to the other end of the reactive-process dimension.

So the nature-nurture dilemma returned to the foreground. In the late 1970s, it was successfully attacked by David Rosenthal and a team of American and Danish researchers applying a combination of high-risk and adoption strategies. As previously noted, using a high-risk sample improves the chances of coming across preschizophrenics conspicuously representing the schizophrenic genotype. The selection of adoptees as subjects makes the separation of genetic and rearing variables possible; otherwise, these are always interwoven when subjects are reared at home. Another distinctive feature of the research designs proposed by Rosenthal and his associates is the use of the concept of "schizophrenia spectrum disorder" as a diagnostic category for assessing and selecting subjects. The spectrum includes, in addition to process schizophrenia, reactive schizophrenia, simple schizophrenia, and paranoia, diagnoses such as borderline schizophrenia, pseudo-neurotic borderline, borderline paranoia, schizoid prepsychotic, and schizoid character. The use of schizophrenia spectrum diagnoses is advantageous not only from the practical aspect of facilitating sampling, and thus perhaps increasing sample size, but on theoretical grounds. In view of the growing evidence for a genetic basis in etiology, the different clinical pictures of schizophrenia might be considered only different manifestations of some grade of a genetic relatedness.

The studies by Rosenthal and his coworkers of adoptees in Denmark are well known. Only in Denmark was it possible to locate high-risk subjects and their biological and adoptive parents among 5,500 cases of nonfamilial adoption. This allowed the selection of the different index, control, and comparison groups that were used for three different complementary research designs. The following are examples of the differing designs:

- The adoptees-family method. This was designed to compare the distribution of schizophrenia spectrum disorders among the biological and adoptive relatives (parents, sibs, half-sibs) of both the index group (consisting of schizophrenic adoptees) and the control group (consisting of adoptees without any known psychopathology) (Kety et al. 1968, 1971).

- The adoptees-study method. This was designed to compare the mental health status of high-risk adoptees as index subjects with low-risk adoptees as controls (Lowing, Mirsky, and Pereira 1983).

- The adoptive-parent method. This was designed to compare psychopathology in three parent groups: (1) biological parents of home-reared schizophrenics; (2) adoptive parents of schizophrenic adoptees; and (3) adoptive parents of adoptees without known symptoms of psychopathology (Wender, Rosenthal, and Kety 1968).

The adoptees-family study provided further evidence for the operation of genetic factors in the etiology of schizophrenia: 13 (9.8 percent) of the 150 biological relatives of index subjects, as compared with only 3 (1.9 percent) of the 156 biological relatives of the controls, showed schizophrenia spectrum disorders. In contrast to this highly significant difference, no significant difference was found between adoptive relatives of index subjects and those of controls.

The decisive influence of the genetic load is also demonstrated in the third of the Danish studies referred to above, namely, the adoptive parent study (Wender, Rosenthal, and Kety 1968). The biological parents of schizophrenic offspring reared at home and the adoptive parents of schizophrenics were rated on the basis on semistructured clinical interviews. A 7-point rating scale was used, with 1 as normal and 7 as schizophrenic psychosis. Significantly higher scores were obtained by the biological parents than by the adoptive parents of schizophrenics (p < .005). This finding was expected; however, the adoptive parents were also significantly more disturbed than adoptive parents of offspring without psychiatric disorders (p < 0.025).

This finding might be of some interest in connection with the second of the Danish studies which, as noted, was designed to understand the relative contribution of genes vs. adoptive-home environment in the development of schizophrenic pathology (Rosenthal et al. 1968, 1971). This work will be discussed in more detail because the index subjects in this study are also referred to under the heading of "children at risk for schizophrenia."

The index group consisted of 76 offspring of schizophrenic parents.
(about two thirds were mothers and one third were fathers) selected from a pool of 11,000 parents who had given away their child for adoption at an early age (mean age of the children was 5.9 months). The criterion for being included in the index parents’ group was a schizophrenia spectrum diagnosis (with the exception of seven cases diagnosed as manic-depressive psychosis). Parental diagnosis was made independently by four (later three) clinicians on the basis of case reports or of elaborated summaries of these reports translated from Danish into English. Among the various spectrum diagnoses, there were 30 consensus diagnoses of process schizophrenia: in an additional 14 cases, this diagnosis was made by at least one of the judges. The control group was composed of 67 subjects matched to the index subjects for sex, age (mean age about 32 years), age of transfer at adoption, and socioeconomic status of the adoptive parents. Subjects were invited for a 2-day examination including a very detailed psychiatric interview, a shortened MMPI, a self-rating scale, the Singer and Wynne methods for measuring thought disorder, attention, and transactional patterns (Singer and Wynne 1966), and electrophysiological procedures for measuring autonomic reactivity. All assessment procedures were performed without knowledge of the subject’s group membership.

The results, based primarily on the clinical evaluation, indicated that there was a significantly higher ($p < .05$) incidence of schizophrenia spectrum diagnoses in the index group and that the cases were more serious: among 24 cases with schizophrenia spectrum diagnoses, there were 3 clear-cut schizophrenics, 13 borderline, 1 manic-depressive or “schizophreniform psychosis” (by way of an offspring of a manic-depressive biological parent), and 4 with schizoid personality traits.\footnote{It is noteworthy that not one index subject was diagnosed as “reactive schizophrenia” in contrast to four such diagnoses among the index parents.} In contrast, in the 12 spectrum diagnoses in the control group, there were no cases of schizophrenia, seven diagnoses of borderline, and five more questionable diagnoses, like “schizophrenic diathesis” (with some doubt), “pronounced prepsychotic features,” “suspicion of organic brain syndrome,” “schizoid, beginning schizophrenia,” and “moderately schizoid.”\footnote{Compared to the index adoptees, the incidence rate of schizophrenia spectrum pathology in the controls is relatively low. In an absolute sense it seems rather high; however, it is possible that parents who give their children up for adoption have more schizoid character traits than are found in the general population. Those parents could be carriers of genes which produce schizophrenia spectrum symptoms in their children without being themselves in need of psychiatric intervention.}

In summarizing the findings, it could reasonably be concluded that the predominance of heredity is emphasized again. However, adherents of a diathesis-stress theory will note that adoptive parents of schizophrenic adoptees scored significantly higher on a behavioral pathology rating scale than adoptive parents of control adoptees. Consequently, environmental factors might also have played a role in the development of schizophrenia in these adoptees.

None of the research strategies, including the adoption strategy, have succeeded in separating fully genetic from environmental factors. Therefore, it seems promising to examine patterns of child rearing which diverge in essential features from the socializing process in the usual family setting. In this way we might learn how such variations influence the contribution of hereditary factors to the development of schizophrenia.

In order to make such a study feasible, the necessary variations in child-rearing practices and settings have to be found within the Western cultural tradition so that standard research principles and procedures can be applied.

Besides the residential setting (Heston 1966; Heston and Denney 1968),\footnote{These researchers examined children separated from their hospitalized schizophrenic mothers at birth and reared in institutions or by relatives. They found that 16.6 percent of these children developed schizophrenia.} a useful research setting exists within the Israeli kibbutz (Nagler 1970; Kaffman 1972; Gerson 1978). There is a vast literature on the kibbutz, and the ideology and structure of its educational system. In brief, the kibbutz in its classical form\footnote{The kibbutz, like other social structures in our dynamic culture, is undergoing many changes. These influence the educational system: however, the kibbutz subjects who participated in our project were all brought up in the more traditional patterns.} is a small community of about 100-2,000 full members who, in accordance with their Zionist-socialistic ideology, have decided to live in a society that is based on collective ownership of land and means of production. The kibbutz members oppose the principle of profit from surplus value created by paid labor and by socioeconomic stratification. Each kibbutz member is obliged to work for the economic and cultural development of the collective and, when necessary, to consider needs of the collective above.
personal aspirations. In exchange, the collective is obligated to satisfy the needs of its members and their families for food, housing, clothing, social and cultural life, and, above all, an adequate education for their children. The educational system reflects the social structure and the belief in collective education as the best preparation for collective living.

Each married couple has its own flat, but children from the age of about 6 weeks to graduation from high school live in separate children's houses under the care of a "metapelet." The children's house is located in the parents' kibbutz and easily accessible to them. The children come to their parents' homes daily for 2 or 3 hours in the late afternoon and also spend the Sabbath and holidays with their parents. Adolescents sometimes live in kibbutz boarding schools outside, but close to, their kibbutz and visit their parents once or twice a week. However, the children spend the great majority of their time in the children's house with their peer group under the care of the metapelet. Besides the parents, the peer group and the metapelet are very influential socializing agents. Life conditions of kibbutz high-risk children are therefore quite different from those of high-risk children raised in an adoptive home or in an institutional setting or by a schizophrenic parent in the nuclear family. How will these educational arrangements, unique in Western culture, affect the development of children at risk? We have avoided stating any explicitly formulated hypotheses, thus acknowledging the exploratory character of the project. There are various possible outcomes that might be predicted.

The first possible outcome views the kibbutz as a protective environment for the child at risk. Thus, children brought up by a schizophrenic parent in the nuclear family are frequently exposed to irrational, erratic behavior; hurt by outbursts of aggression; or forced to cope with emotional remoteness. By contrast, the kibbutz child will be relatively protected against such confusing anxiety-arousing experiences in interaction with the sick parent. Even at times of parental hospitalization, the child will continue the daily routine in the children's house, involved in the learning and play activities of peers, and looked after by concerned and caring educators. In this respect, children at risk for schizophrenia might find favorable conditions for further personality development in the kibbutz. Accordingly, the likelihood of developing a schizophrenic disorder might be less in kibbutz-reared than in town-reared children.

The second possible outcome is based on the theory that stresses the pathogenicity of early mother-child relations. Advocates of this view would argue that it is the quality of this relation which contributes to the development of schizophrenia; consequently, a quantitative reduction of mother-child contact will not neutralize its pathogenic effect. This would suggest no difference in the development of schizophrenia in the kibbutz as opposed to the more typical environment.

The third possible outcome takes cognizance of the conditions inherent in the kibbutz structure which might have a detrimental effect on the development of children of schizophrenic parents. In such a small, closely knit community it might be more difficult than in a city or town to keep knowledge of mental illness in the family; parents' bizarre behavior in public might induce feelings of embarrassment and shame in children. Group life itself from earliest childhood on, with its clash of interests and friction, might be an unbearable burden to the kibbutz child. Unlike the child in town, the kibbutz child has to stay with his classmates day and night without any possibility of escape. If these influences proved to be salient, one might predict greater likelihood of the development of schizophrenic disorders in the kibbutz than in cities and towns.

On the basis of these considerations, it is reasonable to conclude that the kibbutz children's house may have both a corrective and detrimental influence on the developmental potential of the child at high risk for schizophrenia. Therefore, a firm prediction of the outcome of the study could not be made. Instead, the present project is an attempt to add some knowledge to the nature-nurture dilemma in the etiology of schizophrenia.

The Research Team

The project was initiated and basically conceived by David Rosenthal as a further methodological step in his research on the etiology of schizophrenia. This work started with the Danish adoption studies discussed above. In his function as project officer, Rosenthal visited Israel twice to discuss research problems and to gain deeper insight into the project's progress through personal contact with the local team. The members of the Israeli team were: Moshe Ayalon, clinical psychologist; Loni Bonwitt, project secretary; Zeev Glueck, clinical psychologist; Judith Heller Shotten,
psychiatric social worker; Shlomo Kugelmass, clinical and experimental psychologist; Michaella Lifshitz, clinical psychologist; Eitan Lewow, neurologist; Joseph Marcus, psychiatrist; Hanna Merom, clinical psychologist; Shmuel Nagler, clinical psychologist; Joseph Shmueli, experimental psychologist; Shaul Sohlberg, clinical psychologist; and Shoshana Yaniv, educational psychologist.

The different functions of the team members are described in more detail in the articles that follow. The division of responsibility reflected in the different assignments of the team members should not obscure the fact that much of the planning of research design, methodology, and procedures was achieved by group discussion and group participation. In numerous meetings methods of sampling, measurement, statistical analysis, and their rationale were discussed by team members who took full advantage of a group approach in this scientific endeavor. Therefore, although each article was written by one or two team members, it embodies, in some way, the thinking of the entire team.

In carrying out a longitudinal research project that extended over a decade, one had to expect complications of many kinds. There were issues of changes in research staff, loss of subjects, and advances in current methodology. However, these problems were not particularly difficult for the team. We had to overcome the more serious obstacles caused by three wars: the 6-day war, the 3-year war of attrition, and the Yom Kippur war. All of these had their accompanying tensions and anxieties. But the heaviest blow was the untimely death of two beloved and highly appreciated team members. Moshe Ayalon fell ill suddenly while deeply involved in interviewing teachers. All medical efforts to save him were in vain. Moshe Ayalon was one of the most influential and creative teachers in the kibbutz movement. He had returned to postgraduate studies in clinical psychology, relatively late in life. By his talent, scientific conscientiousness, and devotion, he contributed a great deal to the team's work spirit and progress.

Some years later, in 1978 we attended Michaella Lifshitz's funeral. She had been suffering from a malignant disease for about 2 years. Michaella bore her tragic fate with full awareness in a heroic fashion. She worked during and between her numerous hospitalizations, sometimes in a feverish hurry to accomplish her tasks in the short time she had remaining. Only a week before her death she promised to schedule a meeting date to discuss some of the problems in her material. This was the first time that she could not keep a promise. Michaella was highly esteemed as a teacher and as a researcher. Her many publications show her deep respect for methodological rigor in empirical research and also display her ingenuity. On April 15, 1982, Zeev Glueck died. He was a clinical psychologist with outstanding skills in the area of adolescent problems. These skills, in combination with his warm and perceptive personality, enabled him to motivate even the most aloof subjects to participate in the followup interviews. May this publication be accepted as a commemoration to our three colleagues who were denied the opportunity to witness the completion of our project.

**Time Table**

The project was carried out in the following phases:

<table>
<thead>
<tr>
<th>Year</th>
<th>Phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>1966-1968</td>
<td>Planning, preparation, and pretesting</td>
</tr>
<tr>
<td>1968-1971</td>
<td>First examination period</td>
</tr>
<tr>
<td>1970-1973</td>
<td>Data analysis; maintenance of contact with subjects, their parents, and educators</td>
</tr>
<tr>
<td>1973-1977</td>
<td>Second examination period</td>
</tr>
<tr>
<td>1981</td>
<td>Third examination and followup period</td>
</tr>
<tr>
<td>1976-1984</td>
<td>Data analysis and preparation for publication</td>
</tr>
</tbody>
</table>

**References**


Garmezy, N. *Children at risk: The search for the antecedents of schizophrenia*. 1984. Although this third examination was not conceived as part of the original design, it was conducted at the suggestion of David Rosenthal and with the active participation of Shmuel Nagler, Arje Latz, and Edward Silberman.


Acknowledgment

The research project described in this report was undertaken by David Rosenthal with the assistance of a grant from the William T. Grant Foundation. The wise statistical and scientific advice of William Lawlor, S.J., in the early phases of this project is also gratefully acknowledged.

The Authors

Shmuel Nagler, Ph.D., now retired, was formerly Clinical Professor, Kounin Lonenfeld Chair of Special Education, University of Haifa, and Director of the Child Guidance Clinic of the Kibbutz Movement, Oranim
An Invitation to Readers

Providing a forum for a lively exchange of ideas ranks high among the Schizophrenia Bulletin’s objectives. In the section At Issue, readers are asked to comment on specific controversial subjects that merit wide discussion. But remarks need not be confined to the issues we have identified. At Issue is open to any schizophrenia-related topic that needs airing. It is a place for readers to discuss articles that appear in the Bulletin or elsewhere in the professional literature, to report informally on experiences in the clinic, laboratory, or community, and to share ideas—including those that might seem to be radical notions. We welcome all comments.—The Editors.

Send your remarks to:

At Issue
Center for Studies of Schizophrenia
National Institute of Mental Health
Alcohol, Drug Abuse, and Mental Health Administration
5600 Fishers Lane, Rm. 10C-16
Rockville, MD 20857