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Twenty-Five-Year Followup of the Israeli High-Risk Study: Current and Lifetime Psychopathology

by Loring J. Ingraham, Sol Kugelmass, Etha Frenkel, Michael Nathan, and Allan F. Mirsky

Abstract

Current and lifetime psychopathology was assessed in 50 Israeli children of parents with schizophrenia who were either of kibbutz families and raised collectively with the help of child care workers, or of urban families and raised by their parents. Index subjects were compared with 50 matched control children of healthy parents by means of the Schedule for Affective Disorders and Schizophrenia–Israel. Subjects were evaluated in adulthood at a mean age of 31 years; schizophrenia was found exclusively among children of ill parents, and no effect of town or kibbutz rearing on risk for schizophrenia was observed. Major affective illness was more common among kibbutz index subjects. Affective symptomatology observed in some index parents was evenly distributed among town and kibbutz parents and was not related to the diagnosis of affective disorders in at-risk children. Current adult functioning was similar between town- and kibbutz-raised subjects (and in general reflected good adjustment); an excess of personality disorders was found among index subjects. The present findings support the concept that both familial and environmental factors operate in the expression of psychopathology.

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The investigation of schizophrenia is not amenable to many of the tools available to investigators of animal models of illness. Thus, while we aim to understand the interplay of genetic and environmental contributions in the genesis of schizophrenia, we do not manipulate such variables but rather focus our studies on naturally occurring differences and attempt to determine the links among disease expression, kinship, and environmental variation.

Of particular interest and difficulty is the investigation of childhood antecedents of adult illness; the attendant hope is that prevention can be made possible through early identification and intervention. While acknowledging that such investigation is fraught with peril, Hanson et al. (1990) point out that in the original descriptions of schizophrenia, both Kraepelin and E. Bleuler suggest that "more than half" (Bleuler 1911/1950, p. 251) or a "considerable number" (Kraepelin 1919/1971, p. 236) of adults with schizophrenia showed personality anomalies in childhood.

In an attempt to identify and characterize the earliest signs of illness, longitudinal designs spanning the years from childhood into the adult period of disease onset are necessary. For an illness as relatively rare as schizophrenia, concentrating on individuals at increased risk, such as the offspring of ill parents, can increase the number of informative subjects in a sample. In the present study, investigation of two radically differing rearing environments provides a nonintrusive method for comparing the effect of quite different childhood environments on adult expression of psychopathology in children of a parent with schizophrenia.

Reprint requests should be sent to Dr. L.J. Ingraham, NIH/NIMH, Bldg. 10, Rm. 4C-110, 9000 Rockville Pike, Bethesda, MD 20892.

A previous issue of *Schizophrenia Bulletin* (Vol. 11, No. 1, 1985) presented an overview and initial results from the National Institute of Mental Health (NIMH) joint study of schizophrenia by the United States and Israel, known as the Israeli High-Risk Study. In brief, this study compared 50 high-risk children of parents with schizophrenia (25 children raised collectively with the help of child care workers on a kibbutz and 25 by their biological parents in towns) with 50 matched control children (likewise raised in a kibbutz or in town) of healthy parents.

The initial assessment of adult outcome (Mirsky et al. 1985) reported diagnoses from the Schedule for Affective Disorders and Schizophrenia–Lifetime Version (SADS–L; Spitzer and Endicott 1977) interviews conducted with 90 of the original 100 at-risk children and controls when the subjects had reached an average age of 25, approximately 15 years after their first assessment as children. As expected, schizophrenia was found exclusively among high-risk children. Schizophrenia was present in both town- and kibbutz-reared children, providing no support for the concept that differential risk of adult expression of schizophrenia depends on differences between the observed rearing environments.

An unexpected finding of the SADS–L interviews reported in 1985 was an increased risk for affective disorder among kibbutz index (KI) subjects, such that more than 20 percent (5 of 23) of the interviewed subjects reported the symptoms of a major affective disorder versus fewer than 5 percent (1 of 23) of town index (TI) subjects and none of the 44 control subjects. Since a hierarchical diagnostic system that assigned only one diagnosis to each subject had been used in the previous analysis, the excess of affective disorder was not due to the depression observed in some cases of chronic schizophrenia. The variability in the risk for affective disorder observed between kibbutz-raised and town-raised index subjects led to the conclusion that environmental effects associated with kibbutz life might affect the expression of severe psychiatric illness.

To confirm and extend the initial findings reported in 1985, we have recently returned to our subjects for a second diagnostic interview. As our sample ages and passes through the period of risk for schizophrenia, we are able to become more certain that most at-risk individuals who will develop schizophrenia have done so. Another reason for reinterviewing subjects was to be able to report both current and lifetime diagnoses. Beyond the issue of vulnerability to illness, we felt it would be important to represent the current level of functioning of our subjects and to examine whether individuals affected with psychopathology in the past had a differential likelihood of recovery of premorbid function depending on risk and habitat status.

To address the influence of parents' affective symptoms on the development of psychopathology in their grownup children, we rated the index parents for the presence or absence of affective symptomatology in addition to the reassessment of overall parental diagnosis mentioned above, and we compared parents and their adult offspring for the presence of affective symptoms. As a further test of the influence of parental illness on the presence and form of psychiatric illness observed in the children, we also rated the severity and chronicity of parental illness and asked whether parental severity or chronicity was predictive of psychiatric disorder in grownup children.

Methods

Instrument. For this phase of assessment, we were able to take advantage of the recently developed Schedule for Affective Disorders and Schizophrenia–Israel (SADS–I; Levav et al. 1993). The SADS–I is based on the SADS–L used in English-speaking countries, modified for accurate epidemiological assessment of psychopathology in Israel. We added some culture-specific questions to our interview to reflect contemporary Israeli life. For example, the nearly universal experience of military service in Israel (and the problems that can arise during this crucial transition during adolescence) is reflected in questions addressing problems experienced during military service. To assess less severe psychopathology and assess schizophrenia spectrum diagnoses more carefully than is usual in the SADS–L, we asked questions relevant to adjustment and personality disorders. For a more complete description of the SADS–I and its use in Israel, see Levav et al. (1993).

Procedure. Subjects were initially contacted by mail with thanks for their previous participation, a description of the current phase of the study, and an invitation to continue to participate. The letter was followed up with a telephone call to answer any questions about the study. Subjects who were willing to participate were visited in their homes for an interview by a

psychiatric social worker or psychologist blind to the index or control status of the subject. Since the interview was conducted in subjects' homes, indication of rearing environment was often clear at the outset and was inevitably disclosed in the early part of the interview. The interviewer, trained in the SADS–I, described the assessment protocol, answered any further questions about the study, and completed a record of informed consent. Subjects were then queried about life history and interviewed with the SADS–I and in addition completed several questionnaires described in Frenkel et al. (1995), Kugelmass et al. (1995), Mirsky et al. (1995) in this issue of the *Bulletin*.

The interview was tape-recorded and independently reviewed by a judge (E.F. or M.N.) who was blind to the identity of the subject and thus blind to the index or control status and the town or kibbutz background of the subject. The interviewer and judges made both lifetime and current *DSM–III–R* (American Psychiatric Association 1987) consensus diagnoses. The interviewers and judges had previously been trained to criterion on the SADS–I; if the independent diagnoses reached by the interviewer and the judge on the basis of the interview were in disagreement, a second judge (S.K.) reviewed the interview, and the judges arrived at a final consensus diagnosis. The Research Diagnostic Criteria (Spitzer et al. 1978) diagnoses of the SADS–I were translated into *DSM–III–R* diagnoses after the blinded judges had further reviewed the interviews. Only after a final consensus diagnosis was determined was the blind nature of the data broken; subjects' records were then assigned to their respective groups.

In one case, a subject had died shortly before his interview was to have been scheduled. This subject's parents were interviewed for recent information to add to the 1981 diagnostic interview to determine the most recent as well as the correct lifetime diagnosis. In two additional cases where subjects were unavailable for interviews, the interviewers were able to obtain additional diagnosis-related information from the subjects' family members.

Review of Past Records. The written records describing the symptoms, course of illness, and response to treatment in the ill parents made at the time of selection for the study (Nagler 1985) were independently reexamined by two of the authors (L.I. and S.K.) in the light of *DSM–III–R* diagnostic conventions. In cases where raters made a diagnosis other than schizophrenia for an ill parent, a consensus diagnosis of that parent was made on the basis of both raters' reviews of the records.

At the time of selection for the study, the investigators had access to more information about parental illness than the notes that we reviewed, and they also had the benefit of conversations with patients' physicians to help clarify their diagnosis. We were thus reluctant to change a parental diagnosis from schizophrenia where there was insufficient information in the records to make a definitive *DSM–III–R* diagnosis of schizophrenia. However, in those cases where there was evidence consistent with an alternative diagnosis, a new consensus parental diagnosis was assigned.

In addition to overall diagnoses being reviewed, the records were independently scored by two raters (L.I. and S.K.) for presence of affective symptoms, severity of psychotic symptoms, and chronicity of illness. Ratings were made on simple three-point Likert scales with categories "low," "moderate," and "high."

Data Analysis. After *DSM–III–R* diagnoses were determined, lifetime and current diagnoses were grouped to reflect diagnostic categories of interest. If a subject had more than one diagnosis, the most severe diagnosis was used for purposes of classification. Diagnoses were hierarchically rated from schizophrenia to affective disorder to other disorders. Chronic schizophrenia and schizotypal personality disorder (but not schizoid personality disorder) were grouped adjacently to reflect schizophrenia spectrum diagnoses; this grouping was based on evidence from adoption studies that these diagnoses are more common in the biological relatives of individuals with chronic schizophrenia (Kety et al. 1975; Kendler et al. 1981; Ingraham and Kety 1988). Major affective disorders reflected either bipolar disorder or major depression. Minor affective disorders comprised dysthymic disorder, cyclothymia, and adjustment disorder with depressed mood. Other diagnostic groupings were anxiety disorders (generalized anxiety disorder, phobic disorder, or panic disorder), personality disorders (schizoid personality, obsessive-compulsive personality, or personality disorder not otherwise specified), and other diagnoses (adjustment disorder with mixed disturbance of emotions and conduct).

Results

Children at Risk Grown Up. We were able to contact and complete an interview with 84 of the original subjects and gain new information on 3 more. At the time of the interview, subjects were a mean of 30.9 years old, with a range of 26–34. Missing subjects for the SADS–I interviews were distributed among the four groups in a nonsystematic fashion, making it unlikely that we interviewed a biased sample of the original cohort. In combination with the 1981 diagnostic interviews, we now have adult evaluations of 98 of the 100 original subjects; one of the remaining two control subjects died prior to the 1981 diagnostic interviews, and we have never been successful in locating the other.

Table 1 presents the lifetime and current *DSM–III–R* diagnoses for those subjects with a diagnosis. The lifetime diagnosis reflects the most severe illness experienced by subjects up to the present time, while the current diagnosis reflects the *DSM–III–R* diagnosis (if any) at the time of the SADS–I interview. For 14 subjects who did not complete the SADS–I interview during the most recent phase of assessment, lifetime diagnosis (or the determination of no mental illness) was based on previous interviews. In one case of a subject who had died since 1981, the diagnosis was based on previous interviews and interviews of family members.

Table 2 presents the lifetime *DSM–III–R* diagnoses by group. As expected, schizophrenia was found only among index subjects, and index subjects are more likely to suffer from schizophrenia than control subjects (Fisher's exact one-tail $p = 0.059$). The observed 8-percent incidence of schizophrenia in the 30-year-old children of parents with schizophrenia in our study is comparable with the average lifetime risk of 13 percent for developing schizophrenia in the children of individuals with schizophrenia reported by Gottesman (1991, p. 96).

In addition to schizophrenic illness, index subjects were more likely to suffer from diagnoses other than schizophrenia spectrum disorders (Fisher's exact one-tail $p = 0.006$). Although breaking these other diagnoses into subcategories results in small groups that provide weak tests for comparing among various groups, major affective disorders (Fisher's exact one-tail $p = 0.17$) and personality disorders (Fisher's exact one-tail $p = 0.053$) are numerically more common among the index subjects.

As observed in the 1981 diagnoses (Mirsky et al. 1985), the greatest prevalence of major affective disorders was in the KI group, where a lifetime diagnosis of major affective disorder was present in one-fourth of the subjects, significantly greater than among TI subjects (Fisher's exact one-tail $p = 0.049$). Table 2 (which assigns the single most severe diagnosis to each individual) does not show that the two KI subjects with schizophrenia also met the criteria for experiencing major depressive episodes during their lifetime, thereby further increasing the incidence of major affective disorder in the KI cell. Although there are more female kibbutz subjects (16 of 25) than female town subjects (11 of 25) in both the index and the control groups, the pattern of increased major affective disorder in KI subjects remains when analyzed by gender, but in the case of these smaller samples the increase does not attain statistical significance.

Table 3 presents the current *DSM–III–R* diagnoses of the 84 recently interviewed subjects by group. The most striking finding is the relative absence of severe psychopathology evident at present in most of the subjects. Aside from the three remaining index subjects with chronic schizophrenia (one subject with a lifetime diagnosis of chronic schizophrenia was free of symptoms at the time of the most recent interview), there is currently no major affective illness, and only three subjects have a minor affective disorder. Nevertheless, there is an overall excess of non-schizophrenia spectrum illness among index subjects (Fisher's exact one-tail $p = 0.035$) concentrated among the personality disorders (Fisher's exact one-tail $p = 0.048$).

Parents of Children at Risk. The original investigators' records of parental illness were reviewed for 45 cases for which information was available from the original records. In four cases the raters' consensus was that a revised diagnosis was appropriate from the perspective of *DSM–III–R*. In 22 cases, there was insufficient information to make a clear diagnosis on the basis of available records; in these cases we are unwilling to suggest alternative diagnoses, because the original investigators had access to additional information.

The four revised parental diagnoses included two TI parents (subjects 26 and 94) and one KI parent (subject 1) who were retrospectively rediagnosed on the basis of earlier records as having had bipolar disorder; one TI parent (subject 98) was retrospectively re-

Table 1. Lifetime and current *DSM–III–R* diagnoses of adult Israeli high-risk subjects

Group	Lifetime	Current
Index, kibbutz	Schizophrenia, undifferentiated, + major depression, recurrent	Schizophrenia, undifferentiated
	Schizophrenia, undifferentiated, + major depression, single episode	No mental illness
	Bipolar disorder, manic	No mental illness
	Major depression, recurrent	(Missing)
	Major depression, recurrent	Dysthymia
	Major depression, recurrent	Personality disorder NOS
	Major depression, recurrent	No mental illness
	Major depression, single episode	No mental illness
	Adjustment disorder with depressed mood	Adjustment disorder with depressed mood
	Adjustment disorder with depressed mood	No mental illness
	Simple phobia	Simple phobia
	Panic disorder without agoraphobia	No mental illness
	Generalized anxiety disorder	No mental illness
	Schizoid personality disorder	(Missing)
	Personality disorder NOS	Personality disorder NOS
	Obsessive-compulsive personality disorder	Obsessive-compulsive personality disorder
	Adjustment disorder with mixed disturbance of emotions and conduct	(Missing)
Index, town	Schizophrenia, paranoid	Schizophrenia, paranoid
	Schizophrenia, paranoid	Schizophrenia, paranoid
	Bipolar disorder, manic	No mental illness
	Dysthymia	Dysthymia
	Depressive disorder NOS	Personality disorder NOS
	Adjustment disorder with depressed mood	No mental illness
	Adjustment disorder with depressed mood	No mental illness
	Adjustment disorder with depressed mood	No mental illness
	Panic disorder without agoraphobia	Panic disorder without agoraphobia
	Schizoid personality disorder	No mental illness
	Personality disorder NOS	Personality disorder NOS
	Personality disorder NOS	Personality disorder NOS
	Personality disorder NOS	Personality disorder NOS
	Personality disorder NOS	Personality disorder NOS
	Adjustment disorder with mixed disturbance of emotions and conduct	No mental illness
Control, kibbutz	Major depression, single episode	No mental illness
	Major depression, single episode	No mental illness
	Cyclothymia	Social phobia
	Adjustment disorder with depressed mood	No mental illness
	Adjustment disorder with depressed mood	Social phobia
	Depressive disorder NOS	No mental illness
	Schizoid personality disorder	Schizoid personality disorder
Control, town	Major depression, recurrent	No mental illness
	Adjustment disorder with depressed mood	No mental illness
	Adjustment disorder with depressed mood	No mental illness
	Adjustment disorder with depressed mood	No mental illness
	Adjustment disorder with depressed mood	No mental illness
	Generalized anxiety disorder	Generalized anxiety disorder
	Personality disorder NOS	Personality disorder NOS

Note.—NOS = not otherwise specified. *DSM–III–R = Diagnostic and Statistical Manual of Mental Disorders–Revised* (American Psychiatric Association 1987)

Table 2. Lifetime *DSM–III–R* diagnoses of adult Israeli high-risk subjects by group, 1991[1]

Group	Schizophrenia spectrum[2]		Affective disorders plus all other disorders[3]							
	Chronic schizophrenia	SPD	Major[4]	Minor	Anxiety disorders	Personality disorders[5]	Other diagnoses	No diagnosis	Missing	Total
Kibbutz index	2	0	6	2	3	3	1	8	0	25
Town index	2	0	1	5	1	5	1	10	0	25
Kibbutz control	0	0	2	4	0	1	0	17	1	25
Town control	0	0	1	4	1	1	0	17	1	25
Total	4	0	10	15	5	10	2	52	2	100

Note.—Diagnosis reflects most severe illness ever present. SPD = schizotypal personality disorder Probabilities given below are Fisher's exact, one-tailed, index versus control. *DSM–III–R = Diagnostic and Statistical Manual of Mental Disorders–Revised* (American Psychiatric Association 1987)

[1]This table includes 1981 diagnoses where 1991 diagnoses are unavailable.
[2]$p = 0.059$.
[3]$p = 0.006$.
[4]$p = 0.17$.
[5]$p = 0.053$.

Table 3. Current *DSM–III–R* diagnoses of adult Israeli high-risk subjects by group

Group	Schizophrenia spectrum[1]		Affective disorders plus all other disorders[2]							
	Chronic schizophrenia	SPD	Major	Minor	Anxiety disorders	Personality disorders[3]	Other diagnoses	No diagnosis	Missing	Total
Kibbutz index	1[4]	0	0	2	1	3	0	14	4	25
Town index	2[4]	0	0	1	1	5	0	14	2	25
Kibbutz control	0	0	0	0	2	1	0	18	4	25
Town control	0	0	0	0	1	1	0	20	3	25
Total	3	0	0	3	5	10	0	66	13	100

Note.—Diagnosis reflects most severe present symptoms. SPD = schizotypal personality disorder. Probabilities given below are Fisher's exact, one-tailed, index versus control. *DSM–III–R = Diagnostic and Statistical Manual of Mental Disorders–Revised* (American Psychiatric Association 1987).

[1]$p = 0.125$
[2]$p = 0.035$.
[3]$p = 0.048$.
[4]Subjects not available for interview; diagnosis based on previous interview and interview of family.

diagnosed as having had cyclothymic disorder. We were unable to confirm these potentially revised diagnoses by interview. Of the three parents we rediagnosed as suffering from bipolar illness, one refused to participate, and in the other two cases the children refused permission to contact their parents. The parent rediagnosed as having cyclothymic disorder had died.

Although the original investigators' written records did not always provide all the information necessary for making diagnoses, they did enable us with some consistency to rate the presence or absence of reported affective symptoms and the severity and chronicity of parental illness. The three-point scales (low, medium, high) were reduced to two-point scales, since raters were consistent in rating the presence or absence of affective symptoms, severe illness, and chronicity but were poor at discriminating levels of each. Kappas for each scale were acceptable: for moderate or high presence (n = 27) versus low presence (n = 18) of affective symptoms, k = 0.69; for high presence of severe symptoms (n = 28) versus medium or low presence (n = 17), k = 0.47; and for high chronicity (n = 24) versus medium or low chronicity (n = 21), k = 0.65.

Ratings of parental affectivity did not differ between town and kibbutz (table 4; Fisher's exact one-tail p = 0.52). A one-tail was used on the basis of the assumption that greater numbers of affective disorders in the high-risk kibbutz children might be associated with increased affective disorder in their parents.

The presence of parental affectivity was not associated with an excess of affective disorders in the children (table 5; Fisher's exact one-tail p = 0.42). A one-tail test was used on the basis of the assumption that greater numbers of affective disorders in the high-risk children might be associated with increased affective disorder in their parents.

Similarly, parental severity and chronicity were not strongly associated with affective disorder in the children (Fisher's exact two-tail p = 0.16 and p = 1.0, respectively),

Table 4. Presence of affective symptoms in parents of high-risk town and kibbutz children

	Town	Kibbutz
Few affective symptoms	10	8
Affective symptoms present	14	13

Note.—Fisher's exact one-tailed p = 0.52.

Table 5. Presence of affective symptoms in parents of high-risk children with major and minor affective disorders

	No affective diagnosis in child	Major or minor affective illness
Few affective symptoms in parent	14	4
Affective symptoms present in parent	19	8

Note.—Fisher's exact one-tailed p = 0.42.

nor were parental differences in severity or chronicity associated with any other diagnosis among the high-risk children.

There was a tendency for kibbutz parents to be less likely than town parents to have a chronic course of illness with multiple hospitalizations (table 6; Fisher's exact two-tail p = 0.08).

Discussion

In brief, our study of 50 high-risk children and 48 controls found schizophrenia exclusively among the children of ill parents and no effect of town or kibbutz rearing was observed on the expression of schizophrenic illness. Past episodes of major affective illness were more common among the KI subjects, and more personality disorders were observed among both kibbutz- and town-raised index subjects. At present, adult functioning is similar between town- and kibbutz-raised children (and in general reflects good adjustment).

Finding schizophrenic illness exclusively among the children of parents with schizophrenia is consistent with familial transmission of liability to schizophrenia (Kety et al. 1994). That the risk for schizophrenic illness observed here

Table 6. Course of illness in parents of high-risk town and kibbutz children

	Town	Kibbutz
Low or medium chronicity	8	13
Highly chronic course of illness	16	8

Note.—Fisher's exact one-tailed p = 0.08.

is similar in both family- (town-) and kibbutz-raised children suggests that putative environmental effects that contribute to the expression of schizophrenia may be present in both rearing conditions. The finding of similar risk between town- and kibbutz-raised individuals is comparable to the report of Rahav et al. (1981) of similar rates of psychiatric hospitalization for kibbutz and nonkibbutz individuals.

The present assessment with the SADS–I yielded more lifetime affective disorder diagnoses than the previous SADS–L assessment (Mirsky et al. 1985). Like the Mirsky et al. report, the present assessment revealed an excess of major affective psychopathology in the KI group. Even when analyzed by gender (since the kibbutz groups had more females than the town groups), major affective disorder remains concentrated in the KI group. One possible explanation is that individuals experiencing psychiatric distress in the kibbutz environment are more likely to acknowledge their distress and seek help. Another possibility is that the stresses of adolescence and individuation on the kibbutz are more difficult than in town, similarly resulting in an unequal distribution of major and minor affective disorder between kibbutz and town. Consistent with such hypotheses of similar risk between groups but with more severe expression in kibbutz-raised children, the overall incidence of combined major and minor affective disorder seen here is nearly identical between town and kibbutz, but kibbutz-raised children are weighted toward major affective disorder and town-raised children toward minor affective disorder.

A final possibility that may interact with the above-mentioned potential sources of increased affective morbidity in the KI group is that KI parents and their spouses may have themselves experienced a higher rate of affective comorbidity than TI parents. However, we found that the presence of affective symptoms in parents' previous records did not predict the presence of an affective disorder in the children. This finding is consistent with the previous analysis of depressive trends in parents by Mirsky et al. (1985, table 2, p. 153). Thus, while symptoms of affective illness are identified in some of our parents, affective symptoms are not exclusionary for the diagnosis of schizophrenia, and these symptoms do not predict liability for affective illness in their children.

In summary, our finding of an increased risk of major affective illness specifically among KI subjects suggests that environmental contributions to the expression of major affective illness in the children of ill parents are more common in the kibbutz environment, regardless of whether those contributions are related to the development of such psychopathology or to the expression of it.

Another focus of the present inquiry has been the current functioning of adult subjects in addition to their lifetime diagnoses; in general, all the subjects (with the exception of those at-risk index subjects who have developed chronic schizophrenia) are functioning well. In particular, the dramatically different rearing conditions in the kibbutz have generally produced adults free of ongoing psychiatric problems; despite increased likelihood of a history of major affective illness, KI subjects are currently not experiencing such illness, just as kibbutz control subjects are experiencing no major mental illness at all.

Subjects' current functioning, while relatively free of major mental illness, reflects an increased risk of a personality disorder among index subjects overall. Personality disorder was not typified by schizoid personality; there were only three diagnoses of schizoid personality—two among index subjects and one among controls. The personality disorders reported here, which were distributed between both TI and KI subjects, were generally mixed personality disorders that did not fit easily within a specific *DSM–III–R* diagnosis. The presence of an excess of a mixed personality disorder (not schizotypal personality disorder [SPD] or schizoid personality disorder) among subjects at high risk for schizophrenia raises the question of whether the current definition of the schizophrenia-related personality disorder, SPD, is the best descriptor of the personality disorder found in the families of individuals with schizophrenia; this concern has been raised by others (Squires-Wheeler et al. 1988, 1989).

Overall, the present diagnoses of subjects who are in their late twenties and early thirties and well within the age range of risk for schizophrenia show clear evidence of familial risk for schizophrenia. Although our sample is too small for a definitive test, this risk appears to be unaffected by the differences between town and kibbutz rearing. Likewise, the observation of increased risk for personality disorders in the current functioning of both town- and kibbutz-raised high-risk children minimizes the likelihood that differences between these environments are associated with such ill-

ness; rather, these results suggest that nonpsychotic dysfunction among the offspring of ill parents may not be well characterized by the SPD diagnosis. No evidence of ill (or beneficial) effects of town or kibbutz rearing on other adult psychopathology is seen, except for an increased likelihood of past episodes of major depression among KI subjects. This observed increase in lifetime affective symptomatology may reflect differences in liability to psychopathology effected by differing rearing environments, or it may reflect a tendency among kibbutz members to discuss symptoms more openly and to seek treatment more readily. In either case it supports the idea of environmental influences having an effect on psychopathology observed in adults. While kibbutz subjects spent considerably less time with their parents than town-raised children, they did have regular daily contact with their parents and are thus not directly comparable with children who lack regular parental contact.

In conclusion, the present findings of schizophrenic illness observed exclusively among the children of ill parents, an excess of personality disorders among these high-risk children, and a variable risk for major affective episodes depending on rearing environment support the concept that both familial and environmental factors operate in the expression of psychopathology.

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The Authors

Loring J. Ingraham, Ph.D., is Research Psychologist; and Allan F. Mirsky, Ph.D., is Chief, Laboratory of Psychology and Psychopathology, National Institute of Mental Health, National Institutes of Health, Bethesda, MD. Sol Kugelmass, Ph.D., is Professor of Psychology; and Etha Frenkel, M.A., is Doctoral Candidate, Department of Psychology, Hebrew University, Jerusalem, Israel. Michael Nathan, Ph.D., is Psychologist, Institute for Kibbutz Education, Haifa University, Israel.