The Israeli High-Risk Study: Reply to Kaffman

by Allan F. Mirsky

Abstract

Kaffman's critique (1986) of the Israeli High-Risk Study centers on a number of issues: research design, information reported, appropriateness of sample, and Kaffman's own direct experience with psychiatric populations in kibbutzim and elsewhere in Israel. I contend that Kaffman has not fully understood the goals, methods, design, or results of the investigation. The information reported was as complete as possible if it is borne in mind that many of the issues and problems he has raised were not considered in research done on psychiatric disorders 25 years ago. Nevertheless, I contend that the Israeli study meets currently accepted standards of psychiatric research, that the results are valid, and that they raise questions about possible pathogenic influences on vulnerable persons raised on kibbutzim.

Dr. Kaffman (1986) has offered a series of criticisms of the Israeli High-Risk Study, most of which stem from his 25 years of experience as Medical Director and Chief Psychiatrist of the Kibbutz Child and Family Clinic, "which provides psychological and psychiatric services for the majority of kibbutzim in Israel" (p. 152). Criticisms of our study from such a source would appear to carry special importance and to merit a reply. I appreciate that our findings may be difficult for him to accept in view of his experience and professional position. Nevertheless, I believe the results are valid, however unexpected they may be.

Dr. Kaffman's criticisms can be classified into four categories: (1) the design of the study was flawed; (2) insufficient information was either gathered or reported; (3) the sample was inadequate, biased, or both; (4) our findings must be in error because they are contrary to his own experience. I shall attempt to deal with the majority of these criticisms.

I wish to state at the outset that I am grateful for Dr. Kaffman's comments. Although I argue against the saliency of most of them, I appreciate the time he spent preparing his analysis of our study. I must add that, in the interests of expediency, I did not communicate with the other major participants in this work. These comments, for better or for worse, are mine.

It is my view that Dr. Kaffman is essentially misinformed with respect to the first and third categories of criticism, and this may be due in part to a misunderstanding of the principles of research design employed in clinical research and, specifically, of the design of this study. With respect to the second class of criticisms, although we have attempted to report the data as fully as possible, there are some gaps. We are attempting to provide additional information on the subjects as time permits (e.g., Marcus et al., in press; Mirsky et al., in press). Concerning the fourth criticism, I must say that assertions are only assertions unless they can be supported by facts, preferably in the form of published data.

Let us deal with some of the specific questions Dr. Kaffman has raised.

1. Study Design. The quadrion analysis, whereby subjects were matched in squads of four (an at-risk and control subject within each rearing condition), was implemented...
after considerable study of the problem by Rosenthal and colleagues (1971). This design provided a powerful tool with which to evaluate (in experimental design terms) main effects as well as interaction effects in the study. The children were, in fact, matched on as many variables as were considered relevant and possible to achieve at the time; the original criteria were violated (e.g., with respect to age range) only when it proved infeasible to find suitable subjects in the age group intended. Dr. Kaffman argues that the study is biased because about one-fourth of the original sample of control subjects was replaced after some type of psychopathology was discovered in their parents. He obviously misunderstands the nature of the study and of control subjects. Replacement of control parents with psychopathology by those without psychopathology would yield more sharply defined differences between the at-risk and control groups and an increased likelihood of an unambiguous outcome.

The objection was raised that in the index cases, there would more likely be recurrent family difficulties and traumatic experiences because of frequent hospitalizations of one of the parents. It is obvious that this would occur in families of schizophrenics, and it is for this reason that the kibbutz setting, with its potential to moderate these traumatic effects, was selected.

It is alleged that there was an exaggerated tendency to diagnose schizophrenia in the early 1960s. It is certainly unfair to criticize a study designed in 1964 on the basis of standards used in 1986. Nevertheless, European-trained psychiatrists (this encompasses virtually all our psychiatrists) have tended to use the diagnostic label of schizophrenia more sparingly than American-trained psychiatrists, so I am not impressed by this argument. The researchers who reviewed the diagnoses, including Drs. David Rosenthal, Joseph Marcus, and Shmuel Nagler, were extremely sophisticated; collectively, they represented the state of the art in psychiatric diagnosis in the 1960s, if not the 1980s. While it is true that severity of illness data were not provided in the Schizophrenia Bulletin, some data that bear on that question do exist and were presented in conjunction with the outcome data analysis in the paper by Mirsky et al. (1985). This variable (i.e., severity) is not often reported in this research, since it can be defined in many ways and is therefore extremely difficult to quantify.

Dr. Kaffman has pointed to the outcome of 10 children with affective disorders as proof that the original diagnoses of the parents were incorrect. This is a weak argument. As already noted, strenuous efforts were made to exclude persons who did not fit the diagnosis of schizophrenia as generally agreed upon in the early 1960s; symptoms that were included have been listed and described by Nagler (1985). As pointed out by Mirsky et al. (1985), we were not able to show that parents with affective-type symptoms (which are, after all, common in schizophrenia) had children with affective diagnoses. The symptoms we found that differentiated the parents of subjects with affective disorders from those of subjects with schizophrenia spectrum disorders included inappropriate affect, catatonia, and total number of symptoms. In each case, the schizophrenia spectrum parents had more of the symptoms in question.

The criticism, further, that the diagnostic interviews were not conducted by the researchers themselves misses the point about the double-blind condition in behavioral research. Since the researchers were excluded from this contact, they could be relatively unbiased in their evaluations and testing when they saw the children. It should be recalled that in the landmark studies of adopted-away Danish offspring of schizophrenic parents (e.g., Rosenthal et al. 1971), only Joseph Welner actually interviewed the patients; the other investigators (Kety, Rosenthal, Wender, Schulsinger, and others), as in the present instance, reviewed the records and scored the interview data.

2. Insufficient Information. I do not agree that we have provided insufficient information about the subjects and related issues in this investigation. In our Schizophrenia Bulletin report, we provided more than 130 pages of detailed study findings. This does not include prior publications by Marcus et al. (1981) on neurological (soft sign) factors in these subjects. Additional details concerning the early history of these subjects are forthcoming in subsequent issues of the Schizophrenia Bulletin, as well as in other publications. In passing, I can add that we did look into the location of the kibbutzim vis-a-vis terrorist attacks. We found no relation between proximity of the kibbutz to hostile borders and/or a history of terrorism, and the diagnoses of the children living therein. We did provide considerable detail about the nature of family factors in the risk families with respect to the parents' attitudes toward their offspring; this is summarized in some detail in the chapter by Shotten (1985). It would, of course, have been most illuminating to have had family interaction-derived measures of
Affective Style (Goldstein 1985), Expressed Emotion (Brown et al. 1962, 1972), or Communication Deviance (Wynne et al. 1976); but those measures either did not exist or were not in wide use in 1964.

It is true that we did not provide sufficient information about the pathology of the spouses of the index parents, since we know that assortative mating does occur in patients with schizophrenic disorders. The possible influence of psychopathology in the spouses of index parents was not fully appreciated in genetic studies in the 1960s, nor is it always considered or measured even now. We intend to publish what information we have in the near future. Some preliminary analyses indicate that there is no evidence of a relation between the pathology of the spouses of the index parents and disorders in the children.

3. Inadequate or Biased Sample. Dr. Kaffman has proposed that, because of the early findings of cognitive and “soft” neurological sign differences between the index and control cases, we were really following a group of children with the diagnosis of ADD (attention deficit disorder) who happened also to be at risk for schizophrenia. Dr. Kaffman does not acknowledge the extensive data on neurological factors in children at genetic risk for schizophrenia (e.g., Fish 1977; Marcus et al. 1981, 1985). Nor does he make it clear why more of these ADD risk cases were found in the kibbutz setting. I firmly believe that children with ADD become adults with residual ADD and not schizophrenia or other related spectrum diagnoses. The two disorders are distinct entities with distinct clinical courses and outcomes.

Dr. Kaffman has also raised the question of whether one interview by a psychologist who was “brought in especially from the United States” (p. 155) was sufficient to characterize the subjects. The majority of current research in schizophrenia is also based on one patient contact. In the present case, however, this was a detailed interview involving administration of the Schedule of Affective Disorders and Schizophrenia—Lifetime Version, the Social Adjustment Scale, and various subtests of the Wechsler Adult Intelligence Scale. Although Dr. Latz was brought in from the United States, he consulted extensively with Dr. Nagier, who usually made the initial contact with the subject. Dr. Latz is an experienced, senior clinical psychologist who is also an Israeli. He was blind to the identity of the subjects during conduct of the study. Furthermore, the actual diagnoses were made after thorough discussion and consultation on each case with Dr. Silberman, an experienced psychiatrist.

4. Findings Counter to Dr. Kaffman’s Experience. It seems to me that the underlying difficulty Dr. Kaffman has with the study is that the results came out the wrong way; that is, the high-risk children raised on kibbutzim appear now to be faring worse than those who grew up in their nuclear families. He asserts that there is less psychopathology in kibbutzim than in towns in Israel. Further, in some subjects he is following (although whether or not this is a study is not clear), he has obtained a result contrary to our own. That is, the vast majority of a group of 40 offspring of schizophrenic parents who were brought to kibbutzim to be raised showed, on average, “good psychosocial functioning” and no signs of any “significant illness.” This would be a unique outcome in the literature on the offspring of schizophrenics. One must ask the questions of his study that he has asked of ours: How, and by whom, were the parents diagnosed? What instruments or interview techniques were used to evaluate the parents and the children? Who performed the research, and were the examiners aware of the genetic history of the children? Were there control subjects? Until and unless such data are provided and can be evaluated, Dr. Kaffman’s assertion must be viewed as unsupported.

A surprising, if not incomprehensible, finding for Dr. Kaffman is that somewhere between ages 17 and 25 (not 20, as he asserts), some factors may come into play that have a detrimental effect on kibbutz children. Although we do not yet have the data, we suspect that the military experience could provide such an influence. Many discussions with knowledgeable Israelis have emphasized the fact that kibbutz children are under special pressure to achieve when serving in the Israeli Defense Forces. Certainly, kibbutzniks contribute vastly more in numbers to the officer corps than would be expected on the basis of the modest 3 to 4 percent of the Israeli population they constitute. The stress of having to cope and even to excel under the dangerous conditions of military life could interact with genetic vulnerability and lead to the excess of psychopathology we found. This may not be a completely satisfactory explanation for the excess of affective disorders in the kibbutz-index group, since the majority of such cases (seven of nine in the kibbutz index group) were female and women are known to be assigned to less personally dangerous roles in the Israeli Defense Forces. Nevertheless, the effects of the stress of military life on vulnerable women
must be considered. The additional stress of having to decide whether or not to remain on the kibbutz after military service may also provide special difficulties for vulnerable children, both male and female.

We are attempting to learn more about this period of the lives of our subjects; we do know that more persons manifest schizophrenia for the first time between the ages of 17 and 25 than during any other time of life (Gottesman and Shields 1982).

To the extent that this period represents some stage of biopsychological vulnerability, it is conceivable that there are stresses in kibbutz life that are especially salient at this time. In his critique of our study, Breznitz (1985) has provided some useful ideas about what some of those pressures might be— including the special role of the peer group in kibbutzim.

In conclusion, I am concerned when a senior clinician reports that the outcome of a research study and his experience based on day-to-day clinical practice are discrepant. I do not believe, however, that Dr. Kaffman has established that a discrepancy actually exists or that his comments have shed much light on the supposed discrepancy. His arguments, in my view, have not ruled out the possibility that there are features of kibbutz life that may have pathogenic effects in vulnerable children (e.g., Mirsky and Duncan-Johnson 1984) and that may provide useful clues to the etiology of schizophrenia.

References


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