

# The UCLA High-Risk Project

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## Abstract

The UCLA High Risk Project studied a cohort of 64 families over a 15-year followup period. At entry to the study, the families each contained one adolescent who was having behavioral difficulties for whom help was sought from a psychological clinic. Two criteria of the degree of risk for schizophrenia were devised initially, one based on the form of the adolescent's problem and the other on the parental attributes of communication deviance (CD), affective style (AS), and expressed emotion (EE). It was hypothesized that schizophrenia would be the likely outcome when certain patterns of adolescent disturbance and negative communicational and affective patterns were present in the family. The index offspring of 54 of the 64 families were successfully followed up over the 15-year period (mean age = 30 at last followup), and blind psychiatric diagnoses were done. Contrary to the initial hypothesis, the form of the adolescent problem had limited prognostic value; however, the combination of CD and AS correctly identified the overwhelming number of cases who developed schizophrenia and related disorders. CD did even better if the dependent variable was the risk for schizophrenia spectrum disorder in *any* offspring in the family.

Despite frequent reports in the literature of disordered relationship systems in families containing a schizophrenic relative, it has proved difficult to separate those relationships reactive to the psychotic relative from those antedating the onset of the disorder. This issue could be answered only within a high-risk design in which the hypothesized intrafamilial risk factors are meas-

ured prospectively and the offspring followed throughout the risk period for the disorder. The purpose of the present article is to report the current results of such a longitudinal-prospective study designed to establish whether certain characteristic patterns of family relationships were risk markers for the subsequent development of schizophrenia and related disorders such as schizotypal, paranoid, and borderline personality disorders.

The study began over 20 years ago with a cohort of 64 families, each of whom contained a mild to moderately disturbed teenager. Each family had applied for help for their teenager from a university-based psychology clinic. The cohort was believed to contain a number of individuals at risk for subsequent schizophrenia and schizophrenia-linked disorders because we hypothesized that disturbances in adolescence increased the likelihood of more severe psychopathology in adulthood, a hypothesis supported by earlier follow-back (Nameche et al. 1964) and followup studies (Robins 1966) on comparable populations of clinic patients.

## Design of the Study

All families studied were intact at the time of the initial assessment. The families were predominantly Caucasian, of middle- to upper middle-class status, and above average in intelligence. None of the adolescents were considered psychotic or borderline psychotic, at the time of admission. Within this heterogeneous sample, we subdivided the adolescent cases into four groups on the basis of the nature of their pre-

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senting problem. Further, hypotheses concerning relative risk for schizophrenia were articulated, based on our review of the aforementioned follow-back and followup studies, such that two of these groups (active family conflict and withdrawn) were hypothesized to be at higher than average risk, while the other two groups (aggressive-antisocial and passive-negative) were hypothesized to be at much lower risk. (See Goldstein et al. [1968] for the criteria for these groupings and the rationale for relative risk designations.)

All families agreed to participate in a six-session series of family assessment procedures designed to reveal characteristic patterns of family interaction. The family assessment consisted of two main elements—individual assessment of the parents and index case, and family assessment in which the family was observed discussing a series of conflictual family problems. Details of the assessment procedures are described in Goldstein et al. (1968).

Our working hypothesis was that early signs of maladjustment in an adolescent, coupled with the presence of disturbances in communicational and affective climate within the family, would increase the risk for schizophrenia or schizophrenia-spectrum disorders in the offspring.

### Dimensions of Family Behavior

There are obviously many aspects of family behavior that have been hypothesized as relevant to the development of schizophrenia. We have relied on measures that have been well operationalized and have been found empirically valid in systematic studies of families containing a schizophrenic offspring. These measures are: *communication deviance*

(CD), *expressed emotion* (EE), and *affective style* (AS). All of these measures are derived from parental behavior in one context or another.

**Communication Deviance.** CD is derived from the work of Wynne et al. (1977) and refers to an inability of a parent or parents to establish and maintain a shared focus of attention during transactions with another person. Typically, this measure is derived from transactions between a parent and a tester during the administration of a projective technique, usually the Rorschach or Thematic Apperception Test (TAT).

In our study, we used the individual TATs administered to each parent to rate CD. A factor-analytic solution and scoring rules developed by Jones (1977) were used to classify parental units into three levels of CD as follows: *High CD*—both parents show at least one CD factor score  $> T = 60$  or *one* parent shows an elevation  $> T = 60$  on one of two selected factor scores, misperceptions or major closure problems. These two patterns were found associated with schizophrenia in an offspring in a cross-sectional study done by Jones. *Intermediate CD*—only one parent shows an elevation  $> a T$  score of 60 on other than the critical factors cited above and the second parent shows no elevation  $> 60$  on any factor. *Low CD*—neither parent has an elevation  $> a T$  score of 60 on any CD factor. Congruent with the original model outlined by Wynne et al., we hypothesized that all cases of schizophrenia and schizophrenia-spectrum disorders at followup would be in high-CD family units.

**Affective Attitudes.** Affective attitudes have been suggested as important to the course of schizo-

phrenia once the disorder develops (Leff 1976). The relationship of negative attitudes toward an offspring to the onset of schizophrenia has not been established. However, the present study deals with the course of psychiatric disorders from adolescence to adulthood, and it was hypothesized that similar attitudes might be related to this life course as well. Specifically, we hypothesized that negative affective attitudes might serve as a potentiator of a psychopathological process and would increase the likelihood of schizophrenic development in a vulnerable offspring from a high-CD home environment.

Two measures of affective attitude were used in this study, a measure of what is termed expressed emotion (EE) and a measure of affective style (AS).

Expressed emotion is a construct derived from the previously cited British work and reflects *attitudes* of criticism and/or emotional overinvolvement expressed during a tape-recorded interview with an examiner. While the original assessment involved a special interview, the Camberwell Family Interview (Vaughn and Leff 1976), similar assessments were done on parents in our study from a parent interview administered at the time of the original family assessment. Parents are categorized as high or low in EE, based largely on the criticism criterion ( $> 6$  criticisms expressed = high EE), and then formed into parental groups as follows: *dual high EE*, both parents high; *mixed*, one parent high, the other low; and *dual low EE*, both parents are low EE.

The second measure of affective attitudes was termed *negative affective style* and derives from *directly* observed interactions during which family members discussed con-

flictual problems. These interactions were coded with affective style (AS) measures that resemble the EE dimension as they might be expressed transactionally. A full description of this system is contained in an article by Doane et al. (1981) which describes the rationale and details of the AS codes. Families are classified as negative, intermediate, or benign in affective style, based on profile criteria originally developed by Doane and her colleagues. Other studies by our group have revealed that high-EE attitudes and negative AS behaviors coexist in a number of persons (Valone et al. 1983; Miklowitz et al. 1984). However, the relationship is far from perfect, and so we have included both measures as predictors in this study.

### Followup Procedures

Five years after the initial contact, the now young adult index cases were sought and, where located and amenable, interviewed with a structured psychiatric interview and diagnosed by Research Diagnostic Criteria (RDC) (Spitzer et al. 1978) by a clinician without knowledge of any other data on the case. Independent parent interviews were also done to corroborate data from the offspring, and any relevant hospital records were sought. Ten years after this diagnostic assessment, the process was repeated again, although the data from the 15-year assessment were categorized according to *DSM-III* (American Psychiatric Association 1980). The earlier RDC diagnoses were converted to the closest *DSM-III* equivalent. (See table 1 for the number of cases seen at each point).

A substantial percentage of the cases had data that covered the full 15-year period. For cases that had not been available or had refused

**Table 1. Followup status of sample of 64 cases over 15 years**

Status of case	n
5- and 15-year data available	38
15-year only	8
5-year only	11 <sup>1</sup>
Unable to locate, both contacts	5
Refused both contacts	2
	<u>64</u>

<sup>1</sup>Four of these cases died between 5- and 15-year contact. Data on life course sought from parent where possible.

the 5-year followup, the 15-year contact was used to reconstruct the psychiatric status at the earlier period as well as for the intervening years. Thus, there was little problem including these cases in the analysis. However, there was a problem concerning the 11 cases with only a 5-year contact. As seen in table 1, only seven survived this period, and extensive data are available on the psychiatric status of the four deceased cases. Of the seven who survived, only three failed to reveal any diagnosable mental disorder at the 5-year contact. Because our concern was with the lifetime prevalence of the most severe mental disorder in this sample, cases were included in the analysis reported here if such a disorder was evident at the 5-year point. One possible bias in this procedure is an underestimation of the severity of disorder in these cases. However, in a number of instances, the disorder observable at 5 years was quite severe as the sample contained one case of probable schizophrenia, one schizoid personality, four cases of severe substance abuse with associated antisocial personality disorder, and one borderline personality disorder. It is unlikely

that more severe diagnoses would have been substituted on the basis of the later contact. Thus, those cases with diagnosable mental disorders who only had a 5-year assessment were included in the followup analysis.

If during the followup interview either the index case or the parents indicated that another sibling had manifested a pattern of severe psychopathology, that sibling was contacted, interviewed, and diagnosed with the same procedures used with the index case. Eight siblings from seven families were so assessed. Three siblings were diagnosed as schizophrenic, two from the same family; two were diagnosed schizotypal personality disorder; one as a major depressive disorder, possibly bipolar; and one received no psychiatric diagnosis. Since some of the family predictors discussed below were not linked to a particular child in the family, these sibling diagnoses could also be used as outcome criteria. In these analyses, the most severe outcome among the different offspring was used as an alternative outcome measure to test the predictive validity of selected family measures.

**Possible Biases.** It is always possible that the sample available for longitudinal analysis was not typical of the original cohort of 64. Fortunately, this did not prove to be the case. For CD, the percentages of the original 64 cases in the three CD categories were: 28 percent low, 37 percent intermediate, and 41 percent high; within the sample of 54 with diagnostic outcomes, the comparable percentages were: 23 percent low, 38 percent intermediate, and 40 percent high. Thus, the cases available for analysis were not atypical on CD. Similarly, with regard to AS, the percentages for the total sample

were: 47 percent benign, 16 percent intermediate, and 37 percent negative; the percentages for the subsample available for longitudinal analyses were 44 percent, 17 percent, and 38 percent, respectively. A similar pattern was observable for EE as well.

**Diagnostic Procedures at Followup Contacts.** At the time of the 15-year contact, a special interview was constructed that was coded to *DSM-III*. It was thus possible to use it to make most Axis I and II diagnoses. These interviews with the young adult were videotaped whenever possible and at least audiotaped where staff had to travel to a distant site to do the interview. The great majority of these interviews were carried out by our colleague Jeri Doane, who was unaware of the prior psychiatric status of each case. In addition, separate, two-part interviews were held with the parents; one part reviewed similar data as the 5-year parent interview (covering symptomatic and social-role functioning), and a second part used the Family History RDC to evaluate history of mental illness. This interview was administered to each parent separately when possible, and the family histories of the parent and the spouse were done with each member. Pedigrees were then drawn up by a judge without knowledge of any data on the index young adult or any prior data on family interaction.

The interviews at the 15-year followup were reviewed by at least two raters, the interviewer and a blind rater who had had no prior contact with the cases or any of the family data before viewing the videotape or listening to the audiotape. All diagnoses were placed in *DSM-III* format for Axes I, II, and III, and evidence from the interview was documented

in detail to justify any diagnosis. The blind rater also provided a written diagnostic report describing the person in narrative terms. The blind and nonblind raters' diagnostic impressions were then compared, and where any significant discrepancy existed, a second blind rater reviewed the tape and made independent diagnoses. The three sets of diagnoses were then reconciled at a case conference and a consensus diagnosis made. In fact, in only 4 of 46 cases assessed at the 15-year contact was a second blind rating required. In 42 cases, agreement was extremely close.

To follow procedures used in recent psychiatric epidemiology studies, diagnoses were classified as definite, probable, and possible, using criteria developed at Yale University (Leckman et al. 1982).

Because *DSM-III* can yield a plethora of diagnoses, it was necessary to establish some hierarchy for ordering diagnoses, so we followed the procedure used by Leckman et al. (1982) and established our hierarchy according to the purpose of the study to identify cases at risk for schizophrenia and schizophrenia-spectrum disorders. The hierarchy

established was as follows: (1) schizophrenia; (2) schizotypal personality; (3) paranoid personality; (4) schizoid personality; and (5) borderline personality disorder. Beyond this sequence of diagnoses, all others were given a primary diagnosis based on the syndrome with the greatest impact on social-role functioning.

At the time of the final diagnostic appraisal, covering the 15-year period, the most severe primary diagnosis over that period was used as the criterion disorder for predictive purposes. For example, if a person received a diagnosis of simple phobia at 5 years but schizotypal personality at 15 years, the latter was used as the main outcome criteria.

## Results

Table 2 presents the number and percentages of cases in each primary diagnosis category for the sample of 54 index cases originally selected as the disturbed adolescent in the family. To establish the predictive validity of the three family measures, it was necessary to group these diagnoses into clusters. As with the family predictors, a trichotomy was

**Table 2. Primary lifetime diagnosis for index case observed over followup period**

	<i>n</i>	%
No mental illness	16	30
Major depressive disorder <sup>1</sup>	6	11
Antisocial personality/substance abuse	11	20
Mixed personality disorders	6	11
Borderline personality disorder	6	19
Schizoid personality disorder	3	6
Paranoid personality disorder	1	2
Schizotypal	1	2
Schizophrenia	4	7
	54	100

<sup>1</sup>Includes one obsessive-compulsive disorder with marked depressive features and dysthymic disorder.

used. Key to the use of this trichotomy is the notion of the extended schizophrenia spectrum suggested originally in the Danish Adoption Studies (Kety et al. 1968). Recent analyses of the Danish Adoption cases by Kendler and Gruenberg (1984) using the more recent *DSM-III* criteria revealed an aggregation of cases with diagnoses of schizophrenia, schizotypal, and paranoid personality disorder in this same sample of biological relatives of schizophrenics. The status of the borderline and schizoid personality disorders was more questionable in that reanalysis. However, given the ambiguity in the literature concerning the association of borderline personality disorder and the extended schizophrenia spectrum, we used two spectrum categories as outcome criteria: a *broad* schizophrenia spectrum that included borderline and schizoid personality disorder along with those disorders identified by Kendler and Gruenberg as part of the spectrum, and a *narrow* one that excluded the borderline and schizoid categories. In addition, the trichotomy included two other categories; no mental illness (NMI) over the 15-year period, and "other" psychiatric disorder, which includes all cases of *DSM-III* diagnoses not

classified in the broad or narrow schizophrenia spectrum. The "other" group was particularly important theoretically, as it provided an opportunity to test whether factors such as high CD are indeed specific to schizophrenia or merely identify intrafamilial stressors that increase the liability among offspring for psychiatric disorders in general.

The three family predictors then were entered into a log linear analysis to evaluate: (1) how they related to the three outcome categories as independent factors, and (2) how well they related when the variance in the other two predictors was partialled out. The distributions of outcomes are presented first for each predictor taken by itself, as shown in table 3.

When the log linear procedure was used to test the contribution of each variable with overlapping variance with the others removed, probabilities for the partial association were .002 for CD, .001 for AS, but a clearly nonsignificant .789 for EE. Thus, when both affective measures are in the log linear analysis, EE no longer reaches a significant contribution to the prediction of outcome.

Next, a similar log linear analysis

was done testing the relative contribution of CD and AS only. Here each variable still made a significant contribution to the placement of subjects in the three outcome groups; CD had a partial  $\chi^2$  of 17.90,  $p < .001$ , and AS, 22.94,  $p < .0001$ .

The results of this type of analysis can be pursued in more detail to determine whether CD and AS have utility because they segregate the nonmental illness cases from all others, or whether they actually distinguish those families with spectrum disorders as distinctive from those containing offspring with some other form of psychiatric disorder. This is an issue of considerable theoretical significance as it relates to the *specificity* of these family factors to discrete forms of psychiatric disorders. When the matrix from the original log linear analyses was subdivided into submatrices and specific  $\chi^2$  tests run, it was revealed that the most reliable separation of groups was between the spectrum cases and all others ( $p < .003$ ); the segregation between the NMI and nonspectrum cases was only marginally significant ( $p < .054$ ). Thus, the combination of high CD and negative AS specifically identifies a subset of families with a higher probability of spectrum disorder.

**Table 3. Distribution of outcome**

	Communication deviance (CD)			Affective style (AS)			Expressed emotion (EE)		
	NMI	Other	Spectrum	NMI	Other	Spectrum	NMI	Other	Spectrum
Low <sup>1</sup>	8	3	1	11	11	1	5	12	1
Intermediate	3	11	5	1	6	2	6	5	6
High	3	7	10	4	4	12	1	4	7
	$(p < .002)$			$(p < .0008)$			$(p < .04)$		

Note.—NMI = no mental illness; Other = other psychiatric disorder than schizophrenia spectrum; Spectrum = extended schizophrenia spectrum, broad grouping.

<sup>1</sup>For AS, comparable categories are benign, intermediate, and negative; for EE, dual low, mixed, and dual high EE.

ders in particular, and not psychiatric cases in general.

It may be helpful for the reader to see the actual number of cases assigned to the three psychiatric outcome groups according to the three levels of CD and AS. These data are presented in table 4. They indicate quite clearly that the overwhelming number of schizophrenia-spectrum cases occur when high CD and a negative AS profile were present at the original family assessment. The other notable clustering of spectrum cases is present when a negative AS profile and an intermediate CD pattern coexisted at the original assessment. Note also the apparently protective role of low CD in parents: the overwhelming majority of cases from low-CD families had no further mental disorder after their original adolescent difficulties.

Up to this point, we have relied on the broad concept of the extended schizophrenia spectrum, which included borderline and schizoid personality disorders as well as paranoid, schizotypal, and schizophrenic disorders. Next, we

organized the third outcome group by these diagnoses found by Kendler and Gruenberg (1984) to be present in the biological relatives of adopted-away schizophrenics.

The restriction of the spectrum category to this narrower range of diagnoses posed one problem as it reduced the *n* in the third diagnostic category to 6, limiting the possibilities for group segregation. Despite this obvious limitation, we reran the log linear analyses with the new, narrower spectrum grouping. The results were much poorer than previously and the  $\chi^2$  just bordered on statistical significance ( $p < .06$ ). Thus, the combination of high CD and negative AS does not isolate a group of individuals with specific disorders in the narrowly defined schizophrenia spectrum.

**Inclusion of Data From Siblings.** If at any point during the 5- or 15-year followup the index case or respondent provided information suggestive of a severe psychiatric disorder in another offspring in the family, that

sibling was contacted, interviewed, and diagnosed with similar instruments and procedures to those used with the index cases. Siblings were interviewed if reports suggested any of the broad spectrum of disorders in our third diagnostic group, as these were the disorders of particular interest to our hypothesis. Eight siblings from seven families qualified for intensive assessment. Three siblings were diagnosed as schizophrenic (two from the same family), two were diagnosed schizotypal personality disorders, one was diagnosed borderline personality disorder, one major depressive disorder, possibly bipolar, and the last received no psychiatric diagnosis.

These data were included in a subsequent analysis that relied on another method for estimating risk for schizophrenia-spectrum disorders. The measure of CD is not linked to any one child in the family. Therefore, we can ask whether high CD families as a *unit* are at risk for these disorders. By substituting the most severe disorder in any one sibling as the outcome for a family unit, we can examine whether CD is associated with a high likelihood of schizophrenia-spectrum disorder. This substitution of outcomes was done so that the most severe outcome in the family was the outcome diagnosis for that family. Once again, only one outcome diagnosis was assigned per family unit, so that there were still 54 outcome diagnoses. The substitution process was as follows: schizophrenia would replace schizotypal or another spectrum diagnosis; schizotypal would replace any other personality disorder diagnosis, using our previously identified hierarchy of diagnoses. These new outcomes for each family unit were then related to the three levels of CD. (Note that a similar

**Table 4. Combination of communication deviance (CD) and affective style (AS) as predictors of psychiatric outcome status at 15 years**

CD	AS group			Outcome group
	Benign	Intermediate	Negative	
Low	4	1	3	NMI
	1	1	1	OPD
	0	0	1	Spectrum
Intermediate	2	0	1	NMI
	6	4	2	OPD
	0	1	3	Spectrum
High	3	0	0	NMI
	4	2	1	OPD
	1	1	8	Spectrum

*Note.*—NMI = no mental illness; OPD = other psychiatric disorder; Spectrum = extended schizophrenia-spectrum disorders, broad criterion.

process could not be done for AS and EE as these were directed at a specific offspring in the family, the index case.)

A stronger association emerged between CD level and the number of schizophrenia-spectrum disorders when the "worst" case was used as the dependent variable than when the index case was used. In fact, 14 of 19 high CD families had at least one broad-spectrum case in the family, whereas only 10 broad spectrum diagnoses were found in the sample of index offspring. Note also that for the narrow-spectrum diagnoses, as defined by Kendler and Gruenberg (1984), 7 of 19 (37 percent) high CD cases, where the worst case outcome was used, fit the narrow spectrum category, while in the sample of index cases only 4 of 19 (22 percent) manifested narrow-spectrum outcomes. Thus, it appears that the CD level of the parents serves as a familial risk marker for schizophrenia-spectrum disorder for *some* offspring in the family, but does not permit one to target the specific offspring.

**Form of the Adolescent Behavior Problem as a Predictor of Long-term Psychiatric Status.** While most of this report has dealt with the predictive value of parental attributes assessed at the time of the target adolescent's difficulties, we did, in fact, hypothesize that certain patterns of adolescent psychopathology were more likely than others to be precursors of subsequent schizophrenia-spectrum disorders in adulthood. Specifically, in our 1968 article cited previously, we hypothesized that two groups, the withdrawn (group 4), and what we termed the active family conflict (group 2) forms of adolescent behavior problems, were at higher risk for schizophrenia-spectrum disorders than members of the other two groups

termed aggressive-antisocial and passive-negative. Contrary to our hypothesis, no systematic relationship was found between the form of adolescent problem and subsequent psychiatric status, except that one group deemed passive-negative (group 3) was found to have a very low rate of schizophrenia-spectrum disorders.

**Family Mental Illness and CD as Predictors of Outcome**

As indicated previously, we have carried out interviews concerning the history of mental illness in the parents, siblings, and other second degree relatives. This phase of our research is not complete, but about half of the sample has been studied and analyzed. One way that we have looked at these data is to consider whether there is any evidence of a severe mental disorder in any first or second degree relative. Severe mental disorder was defined as one of the following by Family History RDC: psychosis, schizophrenia, or severe recurrent mood disorder, either unipolar or bipolar. Families were considered family-history positive if one of the above was present, and negative when none was present. This represents only one approach to classifying families by

family history, and we plan others that will take into account whether the relative was first or second degree, and also the number of generations in which disorders were present.

We examined the relationship between CD level and the presence of a positive or negative history for severe mental disorder. With 31 families studied to date, there is no association. Approximately half of the families with a positive history are high CD and the rest intermediate or low. So, they are not equivalent markers of risk. Next, we examined whether within the context of a stress-diathesis theory, a combination of high CD and a positive family history increased the risk for schizophrenia-spectrum disorder in any offspring in the family. Here we used the worst case diagnosis (described above) as the outcome variable. Of 12 high CD families studied to date, 5 had a negative family history and 7 a positive one. The rates for broad and narrow spectrum disorders as a function of CD and family history status are presented in table 5. We can see that the combination of a positive family history for severe psychopathology and high CD is a very high risk indicator as 86 percent of these families had at least one offspring who

**Table 5. Rate of broad- and narrow-spectrum disorder in high communication deviance families as a function of family history of mental illness**

	Broad spectrum outcomes		Narrow spectrum outcomes	
	n	%	n	%
Positive family history (n = 7)	6	86	5	71
Negative family history (n = 5)	1	20	1	20

later manifested a broad spectrum disorder and 71 percent a narrow spectrum one, while high CD families with a negative history have a 20 percent offspring rate for both broad and narrow spectrum disorders. We recognize that a positive family history does not necessarily imply a genetic, as contrasted to a psychosocial, mode of transmission. However one interprets these data, they do point to an important combination of predictors.

## Discussion

In an early publication from our research group (Doane et al. 1981), we concluded that parent CD was a significant marker of the potential for subsequent schizophrenia or schizophrenia-spectrum disorder in an offspring. The longer period of our followup has only strengthened this conclusion. In fact, when we expand our analysis beyond the original index cases who were the focus of intensive study to include their siblings, the high-CD parental pattern shows a strong association with the subsequent appearance of a schizophrenia-spectrum disorder in *some* offspring in the family. In retrospect, it was obviously a tactical error not to obtain attitudinal and interactive behavior samples with the other siblings at the time of our original contact, a point raised by other articles in this issue. For while we can use CD level as a generic marker for the family unit, the measures of affective attitude collected in this study were specifically linked to the original index cases. Thus, it is not possible to extend the analysis of the interactive effects of CD, EE, and AS to the other siblings in the family, some of whom are obviously at risk for schizophrenia-spectrum disorders.

Despite these limitations, the second conclusion from our earlier report covering the significant interaction between CD level and affective attitude in influencing the course of psychiatric disorder from adolescence to adulthood is also more strongly confirmed with data from the extended followup. The adolescent at risk for schizophrenia-spectrum disorders comes from a home environment characterized by high CD and a strongly negative affective climate. The behavioral indicator of affective attitude, AS type, proved to be a better addition to CD than the attitudinal measure of EE. Previously (Goldstein 1985), we drew a different conclusion as we found that all of the narrow spectrum outcomes, in addition to high CD, had *both* negative AS and high EE. However, in that report we treated AS as a dichotomy because we grouped the intermediate and negative AS cases together. In the present report we used the trichotomy originally suggested by Doane et al. (1981), and in this instance the overlap between the EE and the AS groupings is considerable. Thus, EE had less to add to prediction when we segregated the extreme, negative AS group from the intermediate one.

It is interesting to note that the only difference between the intermediate and negative AS profiles was the presence of a minimum of one supportive statement by a parent in the context of critical AS behaviors. While it is hard to envision that one such positive remark could make such a notable difference in a 10-minute, emotionally laden interaction, it may reflect tendencies toward compromise or a latent positive attitude toward the adolescent largely masked by the conflict of the moment, a quality that may

be totally lacking in the negative AS profile family.

## Recommendations

The results of this study indicate that measures such as CD, AS, and EE are effective in identifying family units whose offspring are at greater than normal risk for subsequent schizophrenia and related disorders. However, they appear to have predictive validity in the context of a family unit with at least one child who is having behavioral difficulties. Future studies need to establish that these variables are true risk markers in the absence of notable offspring psychopathology.

The answer to this question can be greatly facilitated if simple and economical measures are developed by future researchers so that they can be used in broad-scale community-wide screening of more representative populations. One example of movement in this direction is the work on the development of a brief method for rating EE by members of our research group (Magana et al. 1986). Ultimately, there is a need to incorporate measures of significant family variables into psychiatric epidemiological surveys and family history studies to ascertain their relationship to the traditional risk indicators used in these studies and whether they are informative concerning modes of familial transmission.

While family variables such as CD or AS have predictive validity, it is unclear how they relate to psychopathology in parents. Are they merely reflections of the psychiatric status of a family member, or do they provide additional information about the offspring's risk for psychiatric disorder beyond that provided by knowledge of the parent's psy-



chiatric status? The preliminary data presented in this article, from the analyses of the family psychiatric history, suggest that these sources of information are not redundant, a point also made by Wynne et al. (1987).

The family measures used in our study appear to identify a family unit at risk, but are not helpful in telling us who among the offspring is at risk. Obviously, the development of a genetic marker for schizophrenia, either from behavioral or biochemical studies, would greatly facilitate our understanding of who is at risk and in what environmental context the at-risk person manifests a schizophrenic disorder. Lacking such markers, future studies should avoid the error of this study (and others in this issue) of selecting and assessing only one offspring in the high- and low-risk family units. All offspring must be studied if one is to understand why only some offspring, in high-risk family units develop schizophrenia or schizophrenia-spectrum disorders.

Along these lines, it would be helpful if future high-risk studies could incorporate the individual vulnerability markers, uncovered in other high-risk studies, such as deficits in neuropsychological performance, information processing, and social relationships, in projects in which family risk markers are measured as well. The nature of the linkage between intrafamilial transactions and the development of cognitive and social competence is still not well worked out, and future high-risk studies could provide important information about the subtle interplay among them across the life span. Are attentional deficits expressions of the diathesis that interact with intrafamilial processes, or do they arise from deviant patterns of

communication and affect expression? These are important issues that only prospective longitudinal studies of high- and low-risk populations can resolve.

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