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# Brief Reports

## A Controlled Family Study of Adopted Patients with Temper Outbursts

There is considerable evidence that the tendency to be hot tempered is familial (Lefkowitz et al., 1977; Mattes and Fink, 1987; Stewart and deBlois, 1983) and may be transmitted as a personality trait rather than as part of an associated diagnosis (Mattes and Fink, 1987). While conducting a family study of patients with temper outbursts, seven adopted patients were identified; this provided an opportunity to evaluate whether the familial transmission of temper outbursts is genetic or environmental.

*Methods*

Patients met the first two DSM-III criteria for intermittent explosive disorder (IED), specifically: a) several discrete episodes of loss of control of aggressive impulses resulting in serious assault or destruction of property, and b) behavior that is grossly out of proportion to any precipitating psychosocial stressor. Unlike the criteria for IED, patients could have generalized aggressiveness as well as a number of other diagnoses; however, patients with schizophrenia, bipolar disorder, or other psychotic disorders were excluded.

Family interviews were conducted blind to adoptive status. A version of the Family History Research Diagnostic Criteria (Endicott et al., 1975) was used, modified to obtain relative diagnoses of IED and attention deficit disorder (ADD) and to rate temper outbursts. Generally, one first-degree relative was interviewed, on the phone, with each interview lasting approximately 30 minutes. Information was obtained, if possible, on all first-degree relatives.

*Results*

Clinical characteristics and associated diagnoses of the patients have been described previously (Mattes and Fink, 1987). The mean ages for the two groups of patients were 24.4 ± 10.0 years for the 34 nonadopted temper outburst patients and 20.3 ± 6.68 years for the seven adopted patients. The percentage of male patients in each group was 79.4% and 100%, respectively. The number of first-degree relatives on whom we obtained information was 138 and 22, respectively, for the two groups. Relatives under the age of 6 (below the age of incidence for any of the diagnoses we were considering) were not included.

Adopted patients were significantly ($p < .025$, one-tailed) less likely than nonadopted patients to have a family history of temper outbursts, as indicated in Table 1. In addition, as indicated in Table 1, the adoptive relatives of the seven adopted patients with temper outbursts were significantly ($p < .025$, one-tailed) less likely than the biological relatives of the nonadopted temper outburst patients to have histories of temper problems. In fact, none of the adopted patients had relatives with temper problems, whereas 29 of 138 relatives of patients with temper outbursts had temper problems. None of the relatives of the adopted patients had histories of ADD or IED either, while of the relatives of nonadopted patients 3.6% had ADD and 6.5% had IED. These differences, however, were not statistically significant because of the relatively low frequency even in the biological relatives. The only diagnosis identified among the 22 relatives of the adopted patients was a case of depression.

TABLE 1
*Adopted v. Nonadopted Patients: Family Histories*

| Group | Family As a Whole[a] | | Total Number of Relatives[b] | |
|---|---|---|---|---|
| | History of temper problem | No history of temper problem | With temper problems | Without temper problems |
| Adopted patients with temper outbursts ($N = 7$) | 0 | 7 | 0 | 22 |
| Nonadopted patients with temper outbursts ($N = 34$) | 17 | 17 | 29 | 109 |

[a] $\chi^2 = 4.10$; $p < .025$, one-tailed.
[b] $\chi^2 = 4.32$; $p < .025$, one-tailed.

*Discussion*

Our results suggest that the familial transmission of temper problems is due to genetic, not environmental factors, though the sample size of only seven adopted patients limits the firmness of this conclusion. In addition, these results may be an artifact of reduced psychopathology in adoptive parents, due to the selection process involved in adopting a child. It would be preferable to also evaluate the biological parents of the adopted patients, but this was not within our means. In some respects, however, the evidence that aggressiveness is transmitted genetically in humans is not surprising; different strains of animals differ in aggressiveness, clearly due to genetic factors (Eleftheriou et al., 1974). Environmental factors must also play a role in the development of aggressiveness (Lefkowitz et al., 1977; Stewart and deBlois, 1983). It may be heuristically useful to consider the capacity for aggressiveness to be inherited with variable penetrance, with penetrance dependent partly on environmental factors. It may also be, as theorized by Lefkowitz et al. (1977), that aggressiveness itself is not inherited, but rather is a phenotypic expression resulting from a complex interaction between the environment and an underlying genetic predisposition to learn certain response modes to stress.

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## Premorbid Adult Social Competence, Socioeconomic Status, and Psychopathology

Premorbid social competence as measured by the Zigler-Phillips Social Competence Scale (ZPSCS) has figured prominently as a developmental construct in psychopathology research (Zigler and Glick, 1986). However, the usefulness of the construct has been challenged because of an apparent overlap with socioeconomic status (SES; Nuttal and Solomon, 1965; Raskin and Golob, 1966; Turner and Zabo, 1968). Is it the case that ZPSCS-based estimates of premorbid adult social competence are merely a direct reflection of the demographic factors that determine SES? Zigler (Zigler and Glick, 1986) argue that the premorbid competence as measured by the ZPSCS includes a factor of social class; however, they suggest that the construct is conceptually broader than SES (p. 62).

Premorbid adjustment in patients has been found to be inversely related to parental SES (Chapman and Baxter, 1963). However, Allon (1971) found that patient premorbid adjustment was directly related to patient SES. No association between patient premorbid social competence and parental SES was found in studies by Raskin and Golob (1966) and McCreary (1974). The present study was undertaken to examine both ZPSCS scores and SES in relationship to psychiatric diagnosis. We hypothesized that patient premorbid adult social competence (ZPSCS) would be directly associated with patient SES. Consistent with Zigler's position (*cf.* Zigler and Glick, 1986), we also hypothesized premorbid adult social competence would differ across diagnostic groups after removing the influence of SES.

### *Methods*

The data were drawn from the medical records of 130 patients admitted to a state psychiatric hospital in the southern United States between 1982 and 1984. Patients with a history of organic brain disease or who were admitted for drug or alcohol rehabilitation were excluded from the study. The patient sample was 45% female. Racial makeup of the sample was 55% white and 45% nonwhite. The patients averaged 27.7 ± 8.8 years of age at first lifetime hospitalization. All patients were diagnosed according to the DSM-III (American Psychiatric Association, 1980). Patient diagnoses were as follows: schizophrenia ($N = 79$), schizoaffective disorder ($N = 13$), bipolar disorder (manic, $N = 18$), and mixed nonpsychotic ($N = 20$).

The data used to rate *premorbid* adult social competence using the ZPSCS and *premorbid* SES were gathered as of the first lifetime psychiatric hospitalization for all patients (Lenzenweger and Dworkin, 1987). ZPSCS scores are based on six dimensions—age, marital status, intelligence, education, occupation, and employment history—each of which is rated for one of three levels (Zigler and Glick, 1986). (Intelligence was not rated because IQ scores were unavailable.) Premorbid patient SES was calculated using the Hollingshead Two-Factor Index of Social Position (Hollingshead, 1965). Use of patient SES ratings allowed for direct examination of the ZPSCS-SES relationship; therefore, we coded both indexes based on data confined to the premorbid period for each patient. Moreover, reliable parental SES estimates were unobtainable.

### *Results*

As hypothesized, overall premorbid adult social competence and patient premorbid SES ratings were significantly associated, *i.e.*, better premorbid adult social competence was associated with higher SES ($r = -.39$, $p < .001$). The overall patient sample had a mean Zigler-Phillips score of .46 ± .33. The mean premorbid adult social competence scores (ZPSCS) for the four diagnostic groups were as follows: schizophrenics, .41 ± .30; schizoaffective, .62 ± .45; bipolar, .65 ± .28; and mixed psychiatric disorders = .42 ± .39. The mean SES score for the entire sample was 65.0 ± .80; 87.7% of the patients in the sample came from Hollingshead's class V.

Differences in ZPSCS scores across the four diagnostic groups were examined using analysis of variance and results revealed a significant main effect for diagnosis ($F[3,126] = 3.62$, $p < .015$). Post hoc $t$-tests revealed two significant differences in premorbid competence across diagnostic groups. Schizophrenic patients displayed lower ZPSCS scores than the bipolar patients ($t = 2.787$, $p < .01$) and the bipolar patients displayed lower ZPSCS scores than the nonpsychotic psychiatric patients ($t = -2.148$, $p < .05$). We conducted an analysis of covariance using psychiatric diagnosis as our independent variable, ZPSCS scores as the dependent variable, and SES as the covariate. When the effect of SES was removed, premorbid adult social competence ratings remained significantly different across diagnostic groups ($F[3,125] = 3.746$, $p < .05$). Post hoc $t$-tests on the covariance adjusted means continued to reveal a significant difference in premorbid social competence scores between the schizophrenic and bipolar groups ($t = 3.083$, $p < .01$) as well as the bipolar and the nonpsychotic psychiatric groups ($t = -2.422$, $p < .05$).

### *Discussion*

This study sought to investigate the relationship of premorbid social competence and patient premorbid SES as well as to examine the differences in premorbid social competence across diagnostic groups with the effect of premorbid SES removed. Results suggest that significant differences in premorbid adult social competence exist among diagnostic groups, and our findings are consistent with those reported by Zigler (Zigler and Glick, 1986). Furthermore, we found that significant differences in ZPSCS-assessed premorbid adult social competence exist among diagnostic groups after the variance attributable to premorbid SES was statistically