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Heterogeneity of risk aggregation for alcohol problems between early and middle childhood: Nesting structure variations

MARIA M. WONG,[a] ROBERT A. ZUCKER,[a] LEON I. PUTTLER,[a]
AND HIRAM E. FITZGERALD[b]
[a]*University of Michigan; and* [b]*Michigan State University*

Abstract
We examined how family and child risk factors jointly affected stability and change in externalizing behavior over time in a prospective study of eventual alcohol use disorder. Study participants were community-recruited alcoholic and control families, and their initially preschool-aged male and female children ($N = 335$). Family risk varied as a function of both parental alcoholism (ALC) and antisocial personality disorder (ASPD) and was evaluated for both parents. Child risk was characterized by a set of risky temperament attributes pertaining to high activity, high reactivity, and low attention span. Externalizing behavior was used as the proxy indicator for later alcohol problems. For children in the high family risk group (involving current ALC in both parents or current ALC + ASPD comorbidity or both), child risk when children were 3–5 years old (Wave 1) directly predicted externalizing behavior when children were 6–8 years old (Wave 2), even when Wave 1 child risk was controlled for. In addition, parents' negative interaction with children at Wave 1 mediated the effect of child risky temperament on Wave 2 externalizing behavior. No such pattern was observed in the low family risk group, where only autostability effects were predictive of outcomes at Wave 2. The importance of nesting structure as an ingredient in the epigenesis of risk was discussed. Its particular relevance in understanding the process of risk transmission among offspring from antisocial alcoholic families was emphasized.

Evidence for heterogeneity among alcohol use disorders in adulthood has been in existence for well more than a century (Babor & Lauerman, 1986), although modern interest in the issue is most immediately traced to Jellinek's (1960) typology. Although a number of characterization schemes have emerged, one cluster has repeatedly been described that involves more severe clinical presentation of the alcoholism, the comorbid presence of antisocial symptomatology (which in turn ensures substantial social complications along with the alcoholism), earlier onset of disorder, greater presence of other comorbid psychopathology, and the presence of a positive and denser family history of alcoholism (Babor & Dolinsky, 1988). This variant has alternatively been called Type B (Babor et al., 1992), Type II (Cloninger, Bohman, & Sigvardsson, 1981), and antisocial alcoholism (Hesselbrock et al., 1984; Zucker, 1987), and Babor (1996) has included it under a broader rubric involving "Dionysian" (as compared to "Appolonian") characteristics. Zucker, Ellis, Bingham, and Fitzgerald (1996) have described how the antisocial symptomatology functions as one of the core identifiers, but they explicitly recognize the multiple attributes that are part of the clinical and etiologic picture. In contrast

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Address correspondence and reprint requests to: Maria M. Wong or Robert A. Zucker, University of Michigan, Department of Psychiatry and Alcohol Research Center, 400 E. Eisenhower Parkway, Suite 2A, Ann Arbor, MI 48108-3318; E-mail: mmwong@umich.edu or zuckerra@umich.edu.

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to this cluster, alcoholism without the severity and comorbidities is likely to be of shorter course and of later (adult rather than adolescent) onset, and less often comes out of a family structure where alcoholism has been identified.

Despite the increasing attention to this phenotypic variant in the recent literature (Farren & Dinan, 1996; Holdcroft, Iacono, & McGue, 1998), relatively little is yet known about course variations leading to this outcome, as compared to other adult alcohol dependence outcomes. The primary explanatory structure for the etiology has been genetic (Cloninger, 1987; Hesselbrock, 1995), although from a developmental perspective the heterogeneity of the adult disorder is de facto evidence of risk variations, and probably also of course variations in earlier life (Zucker, Ellis, Bingham, & Fitzgerald, 1996; Zucker, in press).

In the past several years, this epigenetic evidence has started to emerge. Thus, in the Dunedin Study, Caspi, Moffitt, Newman, and Silva (1996) found that boys rated as undercontrolled at age 3 years were 2.7 times more likely to develop alcohol dependence in young adulthood, although this association was moderated by an interaction with sex. No differences existed between high and low undercontrolled girls. The Dunedin findings parallel three other studies from middle childhood and early adolescence, and together suggest substantial developmental consistency after the preschool years. Cloninger, Sigvardsson, and Bohman (1988) found that boys who were high in novelty seeking and low in harm avoidance (i.e., behaviorally undercontrolled) at age 11 years were more likely to show alcoholic outcomes at age 27 years. In another prospective study, Masse and Tremblay (1997) found teacher ratings at both ages 6 and 10 years were predictive of drunkenness onset during the 11- to 15-year age range. Both (low) fearfulness and hyperactivity were significant predictors, with the fearfulness indicator working better at age 6 years and the hyperactivity indicator working better at age 10 years. In a third longitudinal study of the long-term correlates of aggressive behavior among *S*s followed from age 8 years to age 30 years, aggression ratings of children by their peers at age 8 years correlated significantly with citation records for Driving While Intoxicated in adulthood (Eron, Huesmann, Dubow, Romanoff, & Yarmel, 1987).

Taken altogether, these studies indicate that externalizing characteristics almost from toddlerhood on are predictive of later, early emerging adult alcohol disorder. Although the studies did not characterize antisocial comorbid symptomatology as part of the adult alcoholism outcome, the nature of the antecedent predictors suggests that it would be substantial. This work also implies that the developmental process is a relatively autoregressive one, given the parallelism of externalizing characteristics across a variety of ages, all of which are predictive of an alcoholic or problem-drinking outcome in early adulthood. At the same time, the variable networks used in these studies have been notably sparse in their characterization of the family environmental structure that might be facilitative of the apparent autostability of process. In particular, given the well-known association between early child externalizing behavior and a family structure involving poorer monitoring, greater violence and abuse, and higher levels of parental antisocial alcoholism (Chilcoat, Breslau, & Anthony, 1996; Felliti et al., 1998; Loeber & Southamer–Loeber, 1986; Rickman & Davidson, 1994; Tolen, Gorman–Smith, Huesmann, & Zelli, 1997; Zucker, Ellis, Bingham, & Fitzgerald, 1996; Zucker, Ellis, Fitzgerald, Bingham, & Sanford, 1996), a reasonable hypothesis is that the apparent autoregressive process is actually mediated by contextual reinforcers that transmit behavioral risk as well as the genetic risk transmitted at conception. In addition, the relative absence of these contextual reinforcers in nonalcoholic families, and also in alcoholic ones where the alcoholism is of lesser severity or is lacking of the antisocial comorbidity (Ellis et al., 1999; Zucker, Ellis, Bingham, & Fitzgerald, 1996), would suggest that such mediational mechanisms may not be present within a less damaging context structure.

The present study examines these issues within the framework of an ongoing, prospective family study utilizing a high-risk design

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for an eventual adult alcoholic outcome. The study uses child temperament as a proxy marker for constitutional variation, and includes a broad range of contextual variables that were hypothesized to produce continuity of risk and the emergence of an eventual alcohol problem outcome on the one hand, and protection against risk aggregation on the other (Zucker, Fitzgerald, et al., in press; Fitzgerald et al., 1993). Using family risk as a marker for context structure, the first question we asked was whether child and family risk were independent of each other, or whether these influences were best characterized as potentiating of one another (i.e., was there a Nesting Structure × Child Diathesis interaction effect on risky outcome?). If an interaction existed, we wanted to describe the nature of this relationship. It is possible that risk factors aggregate and their effects on outcomes multiply, as predicted by a risk-cumulation model (Fitzgerald, Davies, Zucker, & Klinger, 1994; Zucker, Chermack, & Curran, in press). It is also possible that risk factors dilute the effects of one another (e.g., the absence of one risk factor cancels out the effects of another). For instance, a child who has a risky family background but no difficult temperament may fare as well as other children. The first question concerns whether there is an interaction effect between parent and child risk in producing a damaged outcome. The second question concerns the nature of such an interaction.

Because the ongoing longitudinal study has not yet reached the point where child outcomes would involve alcohol use and problems, we needed to use a proxy indicator for this end point. The one we selected was externalizing behavior, because this measure, involving components of aggression and delinquent activity, very effectively captures the antisocial deviancy construct that has been so strongly and consistently linked to adolescent problem alcohol use, as well as adult alcoholic outcomes (Kandel, 1978; Rydelius, 1981; Zucker & Gomberg, 1986). In defining child risk, we used those difficult temperament characteristics identified by our group (cf. Jansen, Fitzgerald, Ham, & Zucker, 1995; Zucker, Chermack, & Curran, in press) and by others (Sher, 1991; Tarter & Vanyukov, 1994; Wills, Windle, & Cleary, 1998) as potentially precursive to an alcoholic or substance-abusing outcome. These were (high) activity level, (high) reactivity (as a precursor to impulsivity), and (short) attention span (as an indicator of attentional undercontrol). In establishing family risk variation, given the prior literature on antisocial alcoholism as a major differentiator of course, history, and clinical outcome, we used a summary index that scaled the degree to which this was present among one or both of the parents.

Method

Participants

The present study is part of the ongoing Michigan State University–University of Michigan Longitudinal Study (Zucker & Fitzgerald, 1991; Zucker, Ellis, Bingham, & Fitzgerald, 1996). The sample involves 335 children, 257 with alcoholic parent(s) (227 boys and 31 girls), and 77 with nonalcoholic parents (64 boys and 13 girls) participating in Waves 1 and 2 (W1, W2) of the longitudinal study. The study has recruited a population-based sample of alcoholic men, their partners (whose substance abuse status was free to vary), their initially 3- to 5-year-old sons, and their 3- to 11-year-old daughters when present. Biological parents of the children were required to be coupled and living with the child at time of W1 recruitment. Study exclusionary criteria ruled out presence of fetal alcohol syndrome. A contrast–control group of families who resided in the same neighborhoods as the alcoholic families was also recruited. Parents in the contrast group had no lifetime history of substance abuse or dependence; offspring were age matched to the male child in the alcoholic family residing in the same neighborhood. (For a detailed description of the recruitment strategies and eligibility criteria, see Jansen et al., 1995; Zucker, Ellis, Fitzgerald, et al., 1996; and Zucker, Fitzgerald, et al., in press.)

Families were first assessed when the male target children were 3–5 years old and at 3–year intervals thereafter. Data analyzed in this

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paper came from the families who participated in Waves 1 and 2 of the longitudinal study, provided information on the measures reported here, and who had children that were 3–5 years old at W1 and 6–8 years old at W2.

All families were Caucasian. In the area where we sampled, less than 4% of the population meeting inclusion criteria were non-Caucasian. Given the study's sample size, if non-Caucasian ethnic or racial groups were included, the number available would not permit any effective analysis to be done on this variation. As there is an extensive literature showing the relationship between substance abuse and ethnic or racial status, including such variation in the study without being able to statistically model its effects would only contribute to error. We therefore opted to exclude this variation instead.

Procedures

Trained interviewers who were blind to the family diagnostic status collected the data. The contact time for each family varied, depending on data collection wave. Typically, each parent was involved for 9–10 hr and each child for 7 hr spread over seven sessions. A variety of age-appropriate tasks (e.g., questionnaires, semistructured interviews, and interactive tasks) were administered, and most of the contacts occurred in the families' homes. Special arrangement was made to collect data from families who had relocated. No families were lost due to relocation.

Measures

Parent alcoholism (*abuse or dependence*) (*ALC*). ALC was assessed by the Short Michigan Alcohol Screening Test (SMAST; Selzer, Vinokur, & van Rooijen, 1975), the Diagnostic Interview Schedule Version III (DIS; Robins, Helzer, Croughan, & Ratcliff, 1980), and the Drinking and Drug History Questionnaire (DDHQ; Zucker, Fitzgerald, & Noll, 1990). On the basis of information collected by all three instruments, a diagnosis of alcohol abuse or dependence (Lifetime as well as Past 3 Years) was made by a trained clinician using DSM-IV criteria. For the family risk index (see below), the diagnoses were scored as follows: 0, no lifetime diagnosis; 1, alcohol abuse or dependence diagnosis prior to the last 3 years; 2, alcohol abuse or dependence diagnosis in the last 3 years.

Parent antisocial personality disorder (*ASPD*). ASPD was assessed by the Diagnostic Interview Schedule and the Antisocial Behavior Inventory (ASB; Zucker, Noll, Ham, Fitzgerald, & Sullivan, 1994; Zucker, Ellis, Fitzgerald, et al., 1996). Information on the ASB was used to supplement the DIS data in establishing a diagnosis. The ASB is a 46-item questionnaire that assesses the frequency of aggressive and antisocial activities in both childhood (e.g., lying to parents, being suspended from school for fighting) and adulthood (e.g., being fired for absenteeism, defaulting a debt, resisting arrest). For the risk index, ASPD was scored as follows: 0, no diagnosis; 2, presence of diagnosis. (Only a lifetime indicator was used here, because this is a personality disorder [DSM-IV Axis II] diagnosis that requires a long course over childhood and adulthood in order to be established).

Family risk for later alcoholism. The family risk index we used was a composite to evaluate joint contribution of both alcoholism and comorbid antisociality. The index summed the individual ALC and ASPD scores of each parent, and then was summed across them both to provide a *Family Risk* score. Thus, it provided an indicator involving currency of the alcoholism, presence of the antisocial comorbidity, and degree of penetration in the family as a function of both father and mother characteristics. The risk score had a range of 0–8. A score of 0–3 was classified as "low family risk," and a score of 4–8 was "high family risk." This cutoff insured that families in the high group would either make an antisocial alcoholic diagnosis (64%)[1] or involve two parents with currently active alcoholism

1. Technically, low-risk-group families could also have either (a) one parent with an ASPD and the other parent with a lifetime but not current alcoholism diagnosis, or (b) one parent with ASPD and the other with neither ASPD nor an alcoholism diagnosis. These two alternatives are epidemiologically very rare (Zucker, 1996b) and in fact were present in only 4 (1.7%) of the low-risk-group families.

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(52%) or both (13%). Using the scheme, there were 232 and 103 children respectively in the low and high groups.

The 64% ALC +ASPD co-occurrence in the high family risk group is simply a reflection of the known very high comorbidity between ASPD and alcoholism (Zucker, Fitzgerald, et al., 1996). High assortative mating has been reported among antisocial alcoholics (Jacob & Bremer, 1986) and the rate in this sample was 18.8%. On assortment grounds, we anticipated that high but subclinical levels of antisocial symptomatology would also be present among those families where both parents were alcoholic but where neither made a formal ASPD diagnosis. We compared the ASB scores of this subset of the high group against those of the alcoholics (whether lifetime or current diagnosis) without ASPD in the low-risk group. As we expected, the former had significantly higher child ASB scores (fathers: low 7.76 (±4.40); high 10.62 (±4.93); $t(197) = -3.47$, $p = .001$; mothers: low 5.57 (±3.84); high 8.16 (±5.03); $t(199) = -3.49$, $p = .001$) as well as adult ASB scores (fathers: low 6.56 (±3.76); high 10.46 (±5.42); $t(44.22) = -4.15$, $p < .001$; mothers: low 3.89 (±2.66); high 6.10 (±4.49); $t(41.85) = -2.89$, $p < .01$) than did the latter.[1]

To verify alcohol problem severity differences among the noncomorbid alcoholics, we examined differences on the Lifetime Alcohol Problems Score (LAPS; Zucker, Davies, Kincaid, Fitzgerald, & Reider, 1997). This index incorporates information on the primacy (onset), variety, and degree of life invasiveness of drinking problems. This information was obtained from the DDHQ (Zucker et al., 1990), the SMAST (Selzer et al., 1975), and the DIS (Robins et al., 1980). When compared to alcoholics without ASPD in the low family risk group, alcoholics without ASPD in the high-risk group had significantly higher LAPS scores (fathers: low 10.11 (±1.80); high 11.15 (±2.11); $t(198) = -3.10$, $p < .01$; mothers: low 9.68 (±1.21); high 11.64 (±1.58); $t(197) = -8.45$, $p < .001$). These are very substantial differences, given that LAPS units are in standard deviations.

Parent negative affect expression. This measure is the sum of three variables in the Parent Daily Report (PDR): "Were you in a bad mood?" (BAD), "Were you feeling sad?" (SAD), and "Did you spank your child in the past 24 hours?" (SPA) (0 indicates No and 1 indicates Yes for each item). Confirmatory factor analyses indicated these three items have significant loadings on the same factor. The PDR is a revision of the same-named instrument designed by Chamberlain (1980). Items in the PDR assess multiple domains of child behavior and parent–child interaction (e.g., discipline, punishment, time spent with child, activities). The PDR was administered on alternate days to fathers and mothers via phone interview over a 6-consecutive-day interval spanning a weekend. Responses to the three items listed above were summed across both parents' three PDR interviews (range: 0–6).

Child risk (*risky temperament*). Child's temperament was measured by the Dimensions of Temperament Survey (DOTS; Lerner, Palermo, Spiro, & Nesselroade, 1982). The DOTS is a 34-item questionnaire designed to measure five dimensions of temperament: activity level, attention span and distractibility, approach–withdrawal, rhythmicity, and reactivity. Both parents completed the DOTS. As noted earlier, prior research has indicated that certain childhood temperament characteristics are predictive of adult alcohol problems. On the basis of these studies, we used three factors to define risky temperament: activity level (ACT; range: 3–6), reactivity (REA; range: 5–10), and attention span (ATT; range: 11–22). The scores for each dimension were obtained by averaging mothers' and fathers' ratings. Preliminary analyses indicated that these three characteristics had significant loadings on the same factor. The loadings for activity level and reactivity were positive, whereas the loading for attention span was negative. Thus, high child risk implies high activity level, high reactivity, and low attention span. In the within-wave regression analysis (see below section on Plan of analysis), child risk was the sum of REA, ACT, and ATT (reversed). In the cross-wave structural equation modeling analysis, REA, ACT, and ATT (reversed) were used as indicators of child risk.

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Child externalizing behavior. Child externalizing behavior was measured by the Child Behavior Checklist—Parent Version (CBCL; Achenbach, 1991). The CBCL provides scores on two broadband factors (i.e., internalizing as well as externalizing problems). For reasons already described, we make use of only the externalizing measure here. The CBCL manual defines externalizing behavior to include two scales: delinquent behavior and aggression. However, factor analyses in the preliminary analyses indicated that items for aggression could be further divided into two categories: physical (e.g., bullying, destroys other's things, physically attacks people) and verbal (e.g., argues, bragging, talks too much). Both parents completed the CBCL. Their scores were averaged to obtain a single rating of delinquency (DEQ), physical aggression (AGP), and verbal aggression (AGV) for each child. In the within-wave regression analysis, externalizing behavior was the sum of DEQ, AGP, and AGV. In the cross-wave structural equation modeling analysis, DEQ, AGP, and AGV were used as indicators of externalizing behavior.

Plan of analysis

Within-wave analysis (*Wave 1 data* [*children at ages 3–5 years*]). We hypothesized that both family risk and child risk would be significant predictors of externalizing behavior, but we were uncertain about the presence of an interaction effect between family and child risk. To address these questions, hierarchical multiple regression was used to examine W1 data. To reduce multicollinearity between the main effect and interaction terms, all variables in the analysis were standardized. The interaction term was then created by multiplying the standardized variables of family risk and child risk (Aiken & West, 1991; Friedrich, 1982). Using this method, the unstandardized parameters from the analysis are the appropriate standardized solution (see Aiken & West, pp. 43–44). Since previous research showed that boys have higher externalizing behavior scores than girls, gender was used as a covariate in the model. The predictor variables were entered in three steps. After entering the covariate in the first step, the main effects of family risk and child risk were entered in the second step. Finally, the interaction term, Family Risk × Child Risk, was entered in the model.

Across-wave analysis (*W1* [*ages 3–5 years*] *and W2* [*ages 6–8 years*]) *data*). We had two hypotheses. First, we expected that the relationship between child risk and externalizing behavior would be stronger at both waves in the high family risk group than in the low family risk groups. Second, we hypothesized that child risk would lead to an increase in externalizing behavior over time only in the high family risk group. To test these hypotheses, structural equation modeling (SEM) analyses were performed separately for (a) the high family risk group and (b) the combined low family risk groups. These two sets of results were then compared to one another. As noted in the introductory section, this anticipated variation in effects between the high- and low-risk groups was based upon our understanding of the adult subtyping literature. It also was reinforced by earlier analyses of Wave 1 data out of the longitudinal study (Ellis et al., 1999), which have indicated that, at least at the outset, risk aggregation is much denser in antisocial alcoholic families (i.e., the high-risk group).

SEM includes both a measurement model, which describes the relationships among the observed variables, and a structural model, which describes the relationships among the latent constructs. The inclusion of a measurement model makes it possible to isolate the unique variance of observed variables. Thus the structural coefficients are disattenuated from measurement error.

All SEM analyses were conducted using EQS. Maximum likelihood estimation procedures were used. Standardized parameters were presented to facilitate interpretation. Two fit indices—the comparative fit index (CFI; Bentler, 1990a) and the incremental fit index (IFI; Bollen, 1989)—were used to determine model fit. These indices represent the extent to which the model accounts for the observed variances and covariances. For each index, a value of .9 or above indicates good fit. The chi-square goodness of fit statistic was also used to evaluate the models (a signif-

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icant chi square suggests an inadequate fit). However, the sensitivity of the statistic to sample size is well known (e.g., Marsh, Balla, & McDonald, 1988; McDonald & Marsh, 1990; Tanaka, 1993) and thus should be interpreted with caution.

Analyzing the measurement model. This was a two-step process. First, a separate measurement model was estimated for each risk group. There were four latent factors, child risk and externalizing behavior, in both waves. In the measurement model, each observed variable was linked to a latent construct within each wave. Serial correlations among measurement errors were included, as repeated measurement of the same variables often led to correlated measurement errors. In addition, correlations among all the latent constructs were included. A confirmatory factor analysis (CFA) was used to determine the overall fit of the measurement model.

If the fit was satisfactory, the consistency of the measurement model across time was examined for each risk group. Such differences might represent developmental changes or instability in the model. Using the suggestions proposed by Bollen (1989), Farrell (1994), Bryne (1994), and Pentz and Chou (1994), a set of hypotheses representing increasing levels of measurement invariance were tested: (a) The factor loadings had the same pattern at each wave, (b) the factor loadings were identical across wave, (c) the factor loadings and the measurement error variance were identical across time, and (d) the factor loadings and the measurement error variances and covariances are identical across time. These constraints were imposed until the model received a significantly poorer fit than the previous one. At that point, the less constrained model was retained and no further constraints were imposed.

After examining the consistency of the measurement model across time, multiple-group analyses were conducted to test measurement invariance across groups. Similar to the analyses on measurement invariance across time, a hierarchy of hypotheses was used to test measurement invariance across groups (i.e., factor-loading patterns, factor loadings, measurement error variances and covariances).

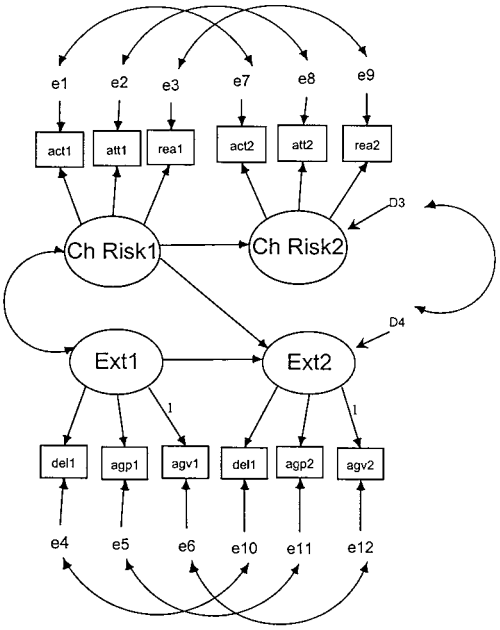


Figure 1. Heuristic model used to test the relationship between child risk and externalizing behavior in different family risk groups.

Analyzing the structural model. When the measurement model was established, we examined the structural models within each risk group. The structural model specified the hypothesized relationships between the latent variables. Figure 1 shows the heuristic model used in the analyses. The endogenous variables were variables in W2. They were supposed to be affected by variables measured at W1. Autoregression effects were also included. Each endogenous variable was affected by its value at the preceding wave. The correlations between disturbances of the endogenous variables were estimated. This is equivalent to assuming that the correlations between the endogenous variable within the same wave cannot be completely accounted for by the exogenous variables in the model. Finally, the correlations between the exogeneous variables were also examined.

After a structural model was established for each risk group, we further analyzed whether the models were consistent across groups. The following sequence of hypotheses were tested: (a) path coefficients had the same pattern for both risk groups; (b) path coefficients were identical for both risk groups; (c)

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path coefficients and variances for the disturbance terms were the same across groups; (d) path coefficients, variances, and covariances for the disturbance terms, as well as the correlation between the exogenous variables, were the same across groups.

Identifying factors that explain differences in the relationship between child risk and externalizing behavior. If the structural models established for the two risk groups were different, we planned to identify factors that would account for the differences.

Results

Characteristics of the observed variables

The means, standard deviations, and zero-order correlations for all the variables are summarized in Table 1. All variables except two (DEQ1 & DEQ2) were normally distributed. Because of a need to satisfy the normality assumption in the ML estimation procedure, a logarithmic transformation was applied to these variables before they were used in the SEM analyses. The distribution of the transformed variables was within acceptable ranges: LDEQ1 (skewness, 1.28; kurtosis, 3.72) and LDEQ2 (skewness, 1.12; kurtosis, 1.98).

At W1 children from the high family risk group had higher scores on reactivity, delinquent behavior, physical aggression, and verbal aggression than the low family risk group. At W2 children from the high-risk group had worse scores on all temperament indicators (i.e., higher reactivity, higher activity level, and shorter attention span) and externalizing behavior indicators (i.e., more delinquent behavior, more physical aggression, and more verbal aggression) than the low-risk group.

Regression analyses of Wave 1 data

Hierarchical multiple regression analysis was used to examine the effects of family risk and child risk on externalizing behavior at W1. Of particular interest was whether there was any interaction between the two risk variables. As expected, the covariate, gender, had a significant effect on externalizing behavior ($\beta = -.19$, $t = -3.38$, $p < .001$; $R^2 = .03$, $p < .001$). The negative beta shows that boys were higher on externalizing behavior than girls. Both the main effects of family risk and child risk were highly significant (family risk: $\beta = .26$, $t = 5.34$, $p < .001$; child risk: $\beta = .18$, $t = 7.28$, $p < .001$; $R^2 = .25$, $p < .001$, $\Delta R^2 = .22$, $p < .001$). The Family Risk × Child Risk interaction was small but also significant ($\beta = .05$, $t = 2.27$, $p < .05$; $R^2 = .26$, $p < .001$, $\Delta R^2 = .01$, $p < .05$).

The simple slopes associated with the Family Risk × Child Risk interaction were probed by methods described in Aiken and West (1991). The relationship between child risk and externalizing behavior was estimated across low (−1 *SD*), middle (mean), and high (+1 *SD*) critical values of family risk by three separate regression analyses. As shown in Figure 2, child risk was a significant predictor at all levels of family risk. There is also an increasingly steeper slope across levels of child risk as family risk increases. Although the interaction is relatively small, the figure also shows its still considerable practical importance: The difference in externalizing behavior between children of low versus high temperament risk is 1.6 times greater in the high family risk context than in the low family risk context.

These findings indicate (a) that the effect of child risk on externalizing behavior is different across different family risk groups, and (b) there is evidence of risk aggregation between family risk and child risk (i.e., family risk potentiates child risk). There is also no evidence of mismatch between these two independent variables.

SEM analyses of W1 and W2 data

The measurement models. Confirmatory factor analyses were performed separately for the two risk groups.[2] For the high-risk group, the

2. The small number of girls in this study made it impossible to conduct SEM analyses separately on boys and girls. We conducted separate analyses on the boys only and found that the results were similar to those presented here.

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Table 1. *Means, standard deviations, and correlation matrices by family risk group*

			Variable																	
	M	*SD*	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
M			8.19	15.72	4.66	1.86	1.09	1.12	1.10	4.86	7.11	8.10	16.11	4.46	1.98	0.89	0.26	1.09	3.83	6.73
SD			1.08	2.31	1.10	0.06	0.05	0.05	0.06	3.06	2.96	1.06	2.10	0.94	0.04	0.04	0.02	0.05	2.08	2.30
Variable																				
1. REA1	7.77**	1.31	—	−0.38	0.22	0.20	0.26	0.20	0.33	0.39	0.42	0.65	−0.28	0.09	−0.12	0.20	0.01	0.30	0.41	0.40
2. ATT1	16.12	2.49	−0.27	—	−0.10	−0.20	−0.01	0.08	−0.12	−0.22	−0.27	−0.18	0.54	−0.01	0.15	−0.08	0.06	−0.14	−0.14	−0.29
3. ACT1	4.42	1.17	0.18	0.02	—	−0.10	−0.11	−0.08	−0.01	0.22	0.30	0.28	−0.25	0.41	0.13	0.18	−0.27	0.14	−0.04	0.16
4. BAD1	1.69	0.05	0.07	−0.01	0.09	—	0.51	0.33	0.02	0.13	0.07	0.13	−0.05	0.18	0.21	0.24	0.12	0.14	0.30	0.43
5. SAD1	0.72*	0.04	0.13	−0.09	0.06	0.30	—	0.39	0.06	0.14	0.09	−0.03	−0.07	−0.02	0.07	0.20	0.06	−0.09	0.41	0.10
6. SPA1	0.89	0.04	0.11	−0.14	0.05	0.11	0.15	—	0.10	0.26	0.08	0.25	0.02	0.05	0.39	0.38	0.17	0.05	0.39	0.19
7. DEL1	1.06***	0.04	0.29	−0.12	0.05	0.03	0.09	0.05	—	0.56	0.52	0.49	−0.21	0.13	0.15	0.04	−0.02	0.38	0.31	0.37
8. AGP1	3.36***	2.39	0.30	−0.14	0.06	0.03	0.04	0.21	0.42	—	0.67	0.52	−0.15	0.06	0.10	0.42	0.16	0.37	0.50	0.57
9. AGV1	5.86***	2.42	0.46	−0.14	0.13	0.06	0.08	0.09	0.53	0.61	—	0.56	−0.19	0.09	−0.14	0.23	0.07	0.36	0.18	0.60
10. REA2	7.41***	1.10	0.57	−0.27	0.05	0.09	0.24	0.21	0.26	0.39	0.45	—	−0.25	0.27	0.03	0.28	0.09	0.24	0.30	0.52
11. ATT2	17.54***	2.31	−0.10	0.40	0.18	0.08	−0.19	−0.19	0.09	0.07	0.10	−0.29	—	−0.12	0.14	−0.02	−0.20	−0.25	−0.21	−0.39
12. ACT2	4.20*	0.94	0.07	0.04	0.50	−0.10	−0.04	−0.03	0.14	0.16	0.15	0.12	0.10	—	0.09	0.07	0.04	−0.01	−0.10	0.10
13. BAD2	1.62**	0.04	0.04	0.13	0.12	0.41	0.01	0.13	0.13	0.17	0.06	0.15	0.02	0.15	—	0.42	0.09	0.14	0.24	0.00
14. SAD2	0.59**	0.03	−0.03	−0.04	−0.01	0.26	0.22	0.01	0.31	0.24	0.19	0.08	−0.05	0.02	0.33	—	0.06	−0.00	0.29	0.25
15. SPA2	0.38*	0.02	0.03	−0.10	−0.07	−0.01	−0.05	−0.06	0.13	0.11	0.12	0.20	−0.08	−0.09	0.16	0.17	—	−0.00	0.21	0.21
16. DEL2	1.06***	0.04	0.19	−0.03	0.10	0.00	−0.04	0.16	0.46	0.30	0.27	0.27	−0.04	0.11	0.24	0.02	0.24	—	0.47	0.57
17. AGP2	2.53***	1.98	0.23	−0.09	0.07	0.12	0.11	0.28	0.28	0.54	0.30	0.42	−0.14	0.12	0.31	0.19	0.23	0.53	—	0.51
18. AGV2	5.32***	2.35	0.29	−0.10	0.04	0.01	0.02	0.17	0.43	0.42	0.57	0.50	−0.14	0.11	0.29	0.26	0.39	0.56	0.58	—

Note: High Family Risk group is shown above diagonal; Low Family Risk group is shown below diagonal. REA, reactivity; ATT, attention; ACT, activity; BAD, parent bad mood; SAD, parent sadness; SPA, parent spanking; DEL, delinquency ($\log_{10}$); AGP, aggression (physical); AGV, aggression (verbal); 1, Wave 1; 2, Wave 2. *r*s < −.20 or >.20 are significant at $p < .05$ for the high-risk group. *r*s < −.13 or >.13 are significant at $p < .05$ for the low-risk group. Significant group differences are indicated by asterisks *$p < .05$. **$p < .01$. ***$p < .001$.

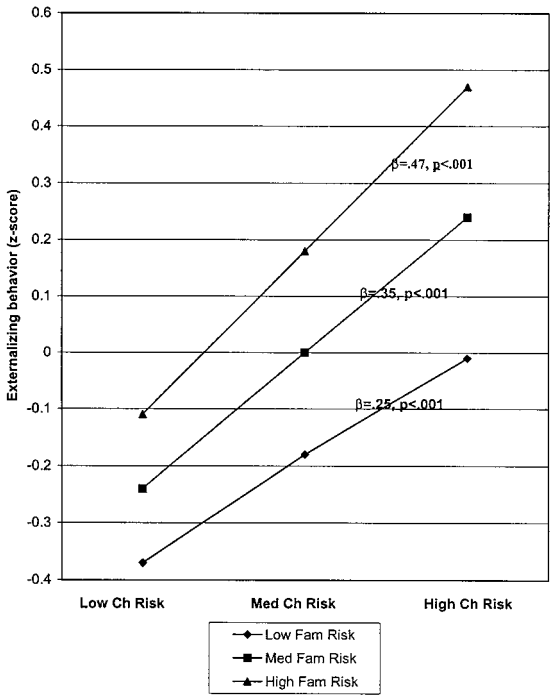


Figure 2. Simple slope analyses: the relationship of child risk and externalizing behavior across different levels of family risk among boys (Wave 1). The slopes of the regression lines for the girls are the same. The intercepts are smaller than those of the boys.

proposed measurement model had a very good fit, $\chi^2(36, N = 103) = 43.17$, *ns*, CFI = .99, IFI = .99. This model fit the data significantly better than the one without correlated measurement errors, $\chi^2(12, N = 103) = 136.74$, $p < .001$.

The next set of analysis tested measurement invariance across time. We compared a model which allowed the factor loadings to freely vary and a model which constrained the loadings to be equal. The two models did not significantly differ from one another, $\chi^2(4, N = 103) = 1.48$, *ns*. Thus, the constraints were imposed correctly. We then further constrained the error variances of the variables to be equal. However, this model had a significantly poorer fit than the less restricted ones, $\chi^2(6, N = 103) = 22.20$, $p < .01$. Therefore no further constraints were imposed. Only the factor loadings were constrained to be equal across time. The fit indices suggested that this model fit the data very well, $\chi^2(40, N = 103) = 44.65$, *ns*, CFI = .99, IFI = .99.

The low-risk group results were very similar to those of the high-risk group. The proposed measurement model fit the data well, $\chi^2(40, N = 232) = 64.47$, $p < .01$, CFI = .97, IFI = .98. Although the chi-square statistic was significant, the fit indices showed that the model had a very good fit relative to a null model. This model had a significantly better fit than the one without correlated measurement errors, $\chi^2(8, N = 232) = 224.60$, $p < .001$.

Further analysis testing measurement invariance across time showed that the loadings for child risk were equal across time whereas the loadings for externalizing behavior were not. The model that imposed constraints on the loadings for child risk only did not differ significantly from the model which allowed the loadings to vary freely, $\chi^2(2, N = 232) = 2.37$, *ns*. No further constraints were imposed across time. All subsequent analyses were conducted on a model in which the loadings for child risk were constrained to be equal, whereas the loadings for externalizing behavior were estimated independently across time. The fit indices for this model were $\chi^2(42, N = 232) = 66.84$, $p < .01$, CFI = .97, IFI = .97.

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Multiple-group analyses were then used to test for measurement invariance across groups. The next set of analyses involved constraining sets of parameters to be identical for high- and low-risk groups. Given the results presented above, we first compared the model where all the loadings were allowed to freely vary with the model in which the loadings for child risk were constrained to be equal across time for the two groups. The two models did not differ significantly from one another, $\chi^2(2, N = 335) = 0.18$, *ns*, suggesting that the factor loadings for child risk at both waves were identical across groups. No further constraints were imposed.

The structural models. Separate structural models were examined for the two risk groups. In the high-risk group, after controlling for autoregressive effects and the correlation between W1 child risk and W1 externalizing behavior, W1 child risk resulted in an increase in W2 externalizing behavior for the high risk group. The disturbance terms of the W2 variables were not correlated. The model fits the data very well, $\chi^2(41, N = 103) = 52.95$, *ns*, CFI = .98, IFI = .98.

In the low-risk group, only autoregressive effects and correlations between variables from the same waves were significant. Child risk in W1 did not lead to an increase in externalizing behavior in W2. The fit indices were as follows: $\chi^2(43, N = 232) = 68.13$, $p < .01$, CFI = .97, IFI = .97. Although the chi-square statistic was significant, the fit indexes indicate a good fit relative to a null model.

The next set of analyses tested the invariant structure of the model across groups. Given the results presented above, the loadings for child risk were constrained to be the same in W1 and W2 for both groups. We also tested the invariance of (a) the covariance between child risk and externalizing behavior in W1 and (b) the path coefficient from W1 child risk to W2 child risk. The loadings for child risk in both waves were identical for the two groups. The covariance between W1 child risk and W1 externalizing behavior was identical as well. However, the path coefficient from W1 child risk and W2 child risk was different for the two groups. The relationship between the two variables was stronger for those children in the low-risk group. The structural coefficients for the two groups are presented in Figure 3.

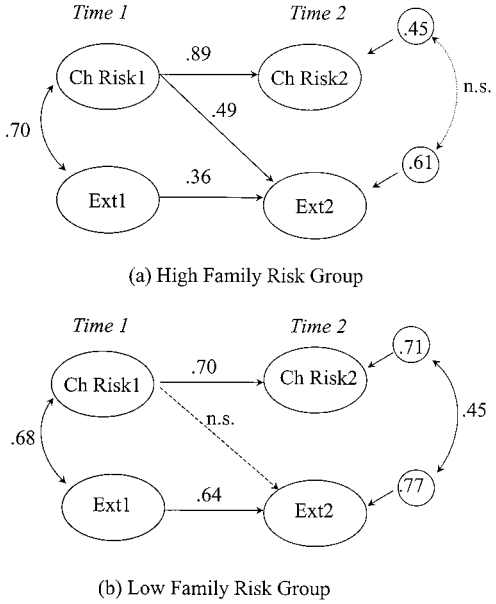


Figure 3. Relationship between child risk and externalizing behavior over time. Chrisk, child risk; Ext, externalizing behavior. Solid lines represent pathways with significant coefficients ($p < .05$). Dashed lines represent nonsignificant pathways.

The relationship among child risk, parent negative affect expression, and child externalizing behavior. What factors may explain the differences in the two risk groups? In the high-risk group, one possible mediator might be the way that parents respond to the child. We examined whether a negative affect expression (NAE) cluster (spanking, bad mood, and sadness) might explain the relationship between W1 child risk and W1 externalizing behavior. In one model, we hypothesized that W1 child risk caused W1 parent NAE, which caused an increase in W2 externalizing behavior. This model fit the data well, $\chi^2(96, N = 102) = 106.51$, *ns*, CFI = .99, IFI = .99 (see Figure 4). The results suggest that W1 child risk is positively related to W1 parent NAE, which influenced W2 externalizing behavior. The direct effect of W1 child risk on W2 externalizing behavior was nonsignificant.

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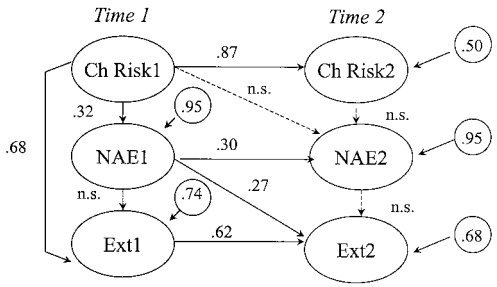


Figure 4. Structural model of externalizing behavior for high family risk groups. Chrisk, child risk; Nae, negative affect expression; Ext, externalizing behavior. All coefficients are significant, $p < .05$. Dashed lines represent nonsignificant pathways.

These results, when considered in conjunction with the previous model, suggest that parent negative affect expression mediates the effect of child risk on child externalizing behavior. It is important to point out that any causal relationship between variables of the same wave needs to be inferred on the basis of theory, because the observed relationship is contemporaneous. However, given that W1 child temperament (the operational definition of child risk), as measured by DOTS, is an indicator of a constitutional variable, this would appear to be a reasonable stipulation. On these grounds, we hypothesized that W1 child risk caused changes in W1 NAE, not the other way around.[3]

Discussion

The present study is one of a series that supports the position that heterogeneity of family structure among alcoholic families has important short- and longer term consequences in terms of (a) the contemporaneous relationship between child and family risk, (b) the cross-time relationship of family risk to child outcome, and (c) the mediational structure that supports those relationships. We address these issues and then discuss some of the broader implications of the work.

With regard to the contemporaneous association between child and family risk, results indicate that there are strong initial relationships between child temperament risk and externalizing behavior, our proxy indicator of later alcohol problems. These relationships exist in both higher risk and lower risk family structures. Moreover, the regression analyses showed that both family risk and child risk had an incremental effect on externalizing behavior, that the two sets of risk factors cumulated, and that they also had a small multiplicative effect on the externalizing behavior outcome. There was no evidence for mismatch of risk.

In addition, the across-time analyses indicate that the relationship between child temperament risk and externalizing behavior is clearest in the high family risk group. The across-wave SEM analyses across the two family risk groups showed that (a) both child risk and externalizing behavior are relatively stable variables over the 3-year interval between late preschool and the early school years but that (b) prior child risk appears to have a more detrimental impact on later behavior within the high family risk structure. That is, even after the autoregressive effects are taken into account, W1 child risk still predicted W2 externalizing behavior. This was not the case in the low family risk group.

Logical questions that follow are "How does the high family risk environment create this differential effect?" and "What factors between temperament and the externalizing outcome mediate the across time relationship?" Our work suggests that parent negative affect expression (bad mood, sadness, and spanking) at W1 mediated the effects of W1 child risk on W2 externalizing behavior, even when W1 externalizing behavior was controlled. In the high family risk environment, W1 child risk

3. We also tested a model in which W1 NAE causes chrisk; the fit statistics of this model were the same ($\chi^2[96, N = 102] = 106.51$, *ns*, CFI = .99, IFI = .99) as the model we selected. In short, there is no statistical reason to infer which direction of effect is correct here. However, we believe the model presented above makes better theoretical sense. To more conclusively infer that child risk causes parental negative affect expression, which in turn causes externalizing behavior, we would need three data waves. Only then can the putative cause and effect relationship among the three variables be resolved (i.e., that W1 child risk causes changes in W2 parent negative affect expression, which in turn causes changes in W3 externalizing behavior).

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was related to W1 parent negative affect expression, which in turn predicted W2 child externalizing behavior. In these families, children who were reactive, hyperactive, and had a short attention span were more likely to be spanked and treated negatively. The negative treatment in turn led to changes in externalizing behavior.

There may be other risk factors in this environment that also contributed to such changes. For instance, did parents treat their children with less warmth, patience, and understanding rather than neglect and harsh punishment? Did parents explain and encourage children to behave well rather than threatening them with punishment? Were parents overly controlling rather than supporting their children to develop in a direction they desire? In their adoption study of environmental and genetic influences upon the development of child antisocial behavior, Ge et al. (1996) have already observed some of these factors making an independent nongenetic contribution to child externalizing outcomes. Such protective factors clearly need to be systematically examined in future work.

How adequate is externalizing behavior as a proxy for later alcohol problem outcomes?

We have already briefly reviewed the strong relation reported in the earlier literature between externalizing behavior and alcohol problem outcomes. Given the strength of this literature, it is likely that the index we are using will prove to be highly adequate. Nonetheless, this relation is heavily based upon associations observed in adolescence and thereafter. In that regard, it is relevant to note that recent reports extend the strength of this connection to younger age groups. Thus, Johnson, Arria, Borges, Ialongo, and Anthony (1995) observed that high levels of conduct problem behaviors by the ages of 10–12 years were associated with earlier alcohol use without parental permission, and higher rates of growth of conduct problems were more common among early users than abstainers. Such findings continue to lend confidence to the use of the externalizing behavior index as a proxy marker for the more distal problem alcohol involvement outcome.

Beyond the early school years: Continuity and discontinuity of risk over later childhood

The present study brings us to the point where the children were in their early school years. Wave 3 data collection (when the children were age 9–11 years) has just been completed as this paper is being written, and these data will soon be available for analysis. This will allow us to examine whether preschool child risk continues to have a sustaining effect on externalizing behavior even well after school entry. If it does, we will then again need to examine the potential family factors that mediate this relationship. If not, we will need to examine what factors lead to dilution. Especially as the children get older, the theoretical base for the study posits that the peer influencing structure will play an additional role (Zucker, Fitzgerald, et al., in press). The degree to which these effects are separate, and additive to the familial effects, will then need to be evaluated.

Fluidity and stability of risk structure over time

A probabilistic risk perspective on epigenetic process would posit that autoregressive processes should be most evident when the early risk structure is heavily determined by biological variation. Under those circumstances, within a rearing structure that is an "average expectable environment," autostability of risk would most likely take place. The present findings, although providing imprecise measures of constitutional structure, in fact suggest the opposite (viz., that those children who have lower levels of risky temperament show greater autostability in externalizing behavior). Of interest is the finding that this stability is found in a group containing alcoholic and nonalcoholic families who both share a common set of characteristics. The parents' alcohol involvement, short and long term, and their antisocial involvement, both short and long term, are of substantially lesser magnitude than what exists in the high-risk group.

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In addition, the potentiating effects of the coaction of these factors is not present, and the relational potentiation of having both spouses with active alcohol dependence in the same household is also missing.

Conversely, our findings suggest that within the high-risk family households it is the immediate relational structure of the family, and in particular the affective interchanges between parents and child (e.g., sadness, spanking), that serve to enhance or dampen the externalizing behavior pattern that is proxy for heightened risk for alcohol and other drug involvement. We have elsewhere (Zucker, Fitzgerald, & Moses, 1995; Zucker Chermack, et al., in press; Zucker, Fitzgerald, et al., in press) characterized this environmental surround as a "nesting structure" primarily because the relationship between child risk and family characteristics is rarely of zero order, and in the high family risk group (what are called antisocial alcoholics in the adult literature) the relationship among a variety of risk factors, such as spousal violence, marital assortment, and co-occurring other psychopathology, is quite strong (Zucker, Ellis, Bingham, & Fitzgerald, 1996). In other words, the family structure has the characteristics of a nest, with interwoven strands which surround the child and serve also to sustain (contain) the cumulation of risk over time. Thus, in the present data at W1, children in high-risk families were more reactive and had more externalizing behavior than children from low-risk families (Table 1). Moreover, the high-risk parents more often were sad while interacting with their children. At W2 the differences were even stronger.

These findings are in strong accord with the recent work of O'Connor and his colleagues (O'Connor, Deater–Deckard, Fulker, Rutter, & Plomin, 1998), who in a slightly older age range (7–12 years) observed that the genetics of antisocial behavior are such that adopted away youngsters at elevated risk for antisocial behavior were more likely to evoke negative parenting from their nonantisocial adoptive parents across a 5-year interval. That is, putative genetically mediated child characteristics were systematically evocative of an environmental response. At the same time, an additional environmental negative control component was present that was not genetically evoked but simply a parental negative response to child out of control behavior. Although the present study does not involve a genetic design, the child risk temperament variables were chosen precisely because of their genetic underpinnings (Tarter & Vanyukov, 1994), and child risk load, as an index of their presence, is higher in the high family risk group. In addition, the evidence for a genetic base for alcoholic disorder exists primarily within the subpopulation identified by the antisocial subtype (McGue, 1994). On these grounds, focused studies of the manner in which genetic and environmental effects contribute to the emergence and maintenance of the full-blown child disorder are of direct relevance. This is particularly true of studies dealing with the development of conduct disorder and antisocial behavior. Within this framework, the present study's identification of a potential evocative effect (W1 child risk leads to higher W1 NAE) as well as an environmental effect (W1 NAE leads to higher W2 externalizing behavior) is an interesting parallel.

Alcoholic subtypes revisited

The present work has utilized a relatively fuzzy definition of the antisocial alcoholic subtype on several counts. For one, ASPD was present in approximately two thirds of the families, not in all. For another, the criterion for high-risk group membership among those without a comorbid ASPD diagnosis involved assortment for current alcoholism, not for severity. However, subsequent analyses showed that even among the high-risk families where no ASPD was present, parents had more antisocial symptoms than the parallel alcoholic but low-risk group. (In fact, the ASB score differences translate to the lifetime presence of three more antisocial symptoms, on average, among the high-risk parents, indicating that antisocial behavior is to a greater degree present here than among the low-risk alcoholics, but it does not meet diagnostic criterion level.) Similarly, the LAPS analyses indicate much more severe and more pervasive alco-

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holic symptomatology in the high-risk group. Thus the diagnostic data indicate that the high-risk group we have identified is at least within the family of the "classical antisocial alcoholic" subtype, even though one third of its members do not qualify for the formal definition.

From the perspective of the child's socialization structure, individual parent subtyping is only a marker for a substantially larger influence matrix. For offspring of parents with disorders that have an identifiable genetic diathesis, the parent's subtype is a crude index of the child's potentially heightened constitutional vulnerability. But, in addition, the adult diagnosis is of interest because the individual may be assortatively linked to others who differentially carry their own structure for transmission of riskiness or are at lower levels of risk. The interesting feature of the antisocial alcoholism subtype is its tendency to aggregate, both intraindividually by way of its high psychiatric comorbidity, and also interpersonally, in the choice of partners with similarly high levels of risk. Our data plausibly suggest that the high coaggregation is what creates the high-risk family mediational structure.

Finally, we note that this definition, involving high alcoholic severity, relatively high antisociality, the expression of higher levels of negative affective expression and a dense family relational structure of alcoholism, is quite close to the revisionist definition for antisocial alcoholism recently proposed by McGue and his associates (McGue et al., 1997). They suggest that there is a continuum of personality risk associated with a continuum of alcoholism severity, and that the so-called antisocial form of alcoholism is perhaps best characterized by low levels of control and high levels of expression of negative emotionality, as well as by high alchoholism severity. This picture maps well onto our high family risk group.

Implications for prevention work

Several prevention-related implications stem from this work. For one, the differences in process that are derivative from the high and low group models of risk flow over time suggest that the targets of intervention should be different in the high and low family risk groups, even for children with relatively similar levels of risky temperament and externalizing behavior. Findings for the low-risk group suggest that programming should be directed at changing the child's temperament style, to the extent that is possible, or, alternatively, at helping the child to learn how better to live with it. Given the relative lack of parent effects as mediators of this process, programming directed primarily at the child would likely have significant impact. In contrast, findings from the high family risk group suggest that, in addition to this individual level of intervention, targeted programming needs to modify family interactions so that the parent–child interchange is shifted (Boyd, 1999; Brody, Flor, Hollett–Wright, McCoy, & Donovan, 1999). This strategy has already been tested in a preliminary way by our own group, utilizing social learning theory and the hypothesis that reduction of conduct problems (externalizing behaviors) in young children will serve as a preventive intervention for the pathway to earlier and more problematic alcohol and other drug use (Nye, Zucker, & Fitzgerald, 1995). Given the mediational role of parent negative affective expression, it would appear that successful programming also needs to address parent disciplinary practices as well as the parent's own negative affective image. And finally, the assortative nature of relationships in high-risk families would suggest that the engagement process for treatment would be initially more difficult, and change would be slower in coming (Nye, Zucker, & Fitzgerald, 1999).

In a related vein, the slope differences depicted in Figure 2 for the different family risk groups again underscore the potential for heterogeneity of outcome (and of subsequent risk for alcohol and other drug involvement) for children with equivalent temperamental risk structures but who are growing up in higher versus lower risk family structures. This perspective on the epigenesis of risk involves a systemic view of risk aggregation and implies, as well, a systemic perspective on prevention and early intervention.

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