CORRELATES OF EXTERNALIZING SYMPTOMS IN CHILDREN FROM FAMILIES OF ALCOHOLICS AND CONTROLS

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Abstract — Aims: This paper describes a new stage in the ongoing evaluation of the original families of sons of alcoholics and controls where we now focus on the relationships among relevant domains of functioning in their young sons and daughters. Methods: The data were gathered from the 15-year follow-up of the families of the original probands (the fathers of these offspring) who had been selected from among students and non-academic staff at a university at approximately age 20. At the 15-year evaluation of these families, a structured interview and the Child Behavioral Checklist (CBCL) questionnaire were administered to a parent, usually the mother, of 145 offspring age seven through 17. The eight domains evaluated here included the extended family histories of alcohol use disorders, parental alcoholism, independent mood or anxiety disorders in the grandparents and parents, the history of potential brain insults early in life, the absence of a biological parent in the home, and scores for internalizing symptoms, with externalizing symptoms as the dependent variable. Results: Correlations among the domains were all in the predicted direction, a structural equation model revealed empirical results with an $R^2$ of 0.26, and there were high goodness of fit characteristics for hypothesized and empirical models. The results were similar for boys and girls and older versus younger offspring. Conclusions: An understanding of the relationships among characteristics in the offspring of the original probands offers the opportunity of establishing levels of functioning in relevant domains before the onset of alcohol-related problems or related disorders. The data presented here represent a baseline upon which future follow-ups will evaluate substance-related problems and disorders as this population matures.

INTRODUCTION

Alcohol use disorders (AUD) develop through a complex interplay between environmental and biologic/genetic factors, with the latter combining to explain an estimated 60% of the variance of risk (McGue, 1997; Heath et al., 1999; Prescott and Kendler, 1999; Hill et al., 2000; Schuckit, 2002). The genetic factors operate in the context of multiple pathways that contribute to various aspects of the choice to begin to drink, and the progression of problems during adolescence and onto adulthood, although no one model is likely to explain all of the risk (Sher, 1991; Chassin et al., 1999; Labouvie and Bates, 2002). Possible paths include a socialization model through which drinking in the home and among peers, along with expectations of the effects of alcohol and the intensity of response to this drug, might promote heavier drinking and associated problems (Hawkins et al., 1992; Jacob and Leonard, 1994; Chassin et al., 1996; Newcomb, 1997; Windle, 2000; Labouvie and Bates, 2002; Schuckit, 2002). There are also a number of psychological symptom-related pathways, including an affect-related model where an escalation in the use of alcohol and other drugs might develop through attempts to deal with the underlying disturbing symptoms (Pandina et al., 1992; Tarter and Vanukov, 1994; Finn et al., 1997; McGue et al., 1997; Carpenter and Hasin, 1998; Twitchell et al., 1998; Chassin et al., 1999; Jacob and Windle, 2000; Schuckit, 2002).

A third model focuses on deviance proneness, and posits that a family history (FH) of disinhibited behaviours (including substance use disorders) relates to cognitive difficulties in the offspring which might produce symptoms of sadness and of disinhibited, acting out, or externalizing behaviours (Jessor and Jessor, 1977; Hesselbrock et al., 1991; Sher, 1991, 1997; Pihl and Bruce, 1995; Zucker et al., 1996; Finn et al., 1997; Bauer and Hesselbrock, 1999; Giancola and Parker, 2001; Tarter, 2002). Eventually, these externalizing symptoms and associated phenomena may contribute to the selection of deviant or substance-using peers, and to the early onset of substance use and an enhanced risk for alcohol and drug problems. High levels of externalizing behaviours have been noted in adolescents who use substances, might characterize children of alcoholics, and have been reported to predict future substance-related problems (Sher, 1991; Cadoret et al., 1995; Crowley et al., 1998; Puttler et al., 1998; Jacob and Windle, 2000; Zucker et al., 2000; Tarter, 2002). While much of the relationship of externalizing symptoms to substance use might be explained by extreme conditions such as conduct disorder or antisocial personality disorder (ASPD), some aspects of disinhibited or impulsive behaviour may contribute to substance use even after the impact of these conditions is controlled (Brown et al., 1996; Biederman et al., 1998; Giancola and Parker, 2001). The ideal approach to evaluate models of risk would involve a longitudinal study where data were obtained regarding biological background and multiple domains of functioning (Casp et al., 1996; Newcomb, 1997; Chassin et al., 1999; Ferdinand et al., 2001). This technique may be especially useful if observations begin early in life and domains of interest are measured repeatedly, without over-reliance on long-term retrospective data. Prospective research offers the opportunity to evaluate intermediate phenotypes as they develop and subsequently contribute to the problematic use of substances such as alcohol. Just as there is no single, perfect model or set of domains for study in the development of AUD (Carpenter and Hasin, 1999; Labouvie and Bates, 2002; Tarter, 2002), there are also several different, but complementary, ways to select and evaluate populations appropriate for such longitudinal research.
Some investigations focus on children already demonstrating extreme forms of major intermediate phenotypes relevant to one or more of the models, including young subjects with relatively severe depressions or anxiety disorders, and those already demonstrating intense externalizing symptoms (Merikangas et al., 1996; Tarter, 2002). The latter has involved identification of young persons who already meet criteria for conduct disorder, or who are selected because of involvement with the juvenile justice system (Bauer and Hesselbrock, 1999; Tarter, 2002). Such subjects have many assets for enhancing understanding of the approximately 20% of alcohol-dependent individuals who have ASPD, and for analyzing the impact of understanding of the approximately 20% of alcohol-dependent AUD associated with additional substance dependencies and criminality (e.g. Types 2 and B alcoholism) (Cloninger, 1987; Hesselbrock et al., 1991; Babor et al., 1992; Slutske et al., 1998; Bauer and Hesselbrock, 1999; Schuckit, 2000).

However, the focus on extreme cases, which are likely to represent a minority of alcoholics, might produce a situation where the high proportion of the variance explained by the externalizing symptoms in these subjects could diminish the magnitude of effect of additional influences that contribute to the risk for AUD in the less extreme case.

In the mid 1970s, our research group adopted a strategy of identifying subjects at high risk for future AUD who were felt to represent the more usual, relatively functional alcoholic (Schuckit, 1998; Schuckit and Smith, 2000; Schuckit et al., 2002). We excluded individuals in whom extreme levels of disinhibition were likely to operate by selecting subjects who, by the average age of 20 years, had not developed ASPD, and limited the potential extreme effects of two major psychiatric conditions known to be associated with subsequent substance use disorders (SUD) by excluding subjects with manic depressive disease or schizophrenia in themselves or their parents (Schuckit, 2002). Our face-to-face follow-ups of over 97% of these 453 men, 10 and 15 years after their initial evaluation, revealed the expected high rate of AUD (Schuckit and Smith, 1996, 2000). Approximately 45% of the FH positive subjects developed alcohol abuse or dependence, compared with 20% of the FH negatives, figures that included 32 vs 14% with alcohol dependence.

While not studied until late adolescence or early adulthood, the data on these 453 men generated information about several additional domains of functioning applicable to the three models described above (Schuckit and Smith, 2000). Most directly germane to the current discussion is the fact that a measure of externalizing symptoms, behavioural undercontrol (Sher, 1991), was related to an alcoholic outcome, although did not link to the FH of alcoholism in these families. Behavioural undercontrol operated as a predictor of AUD in a path model, with the influence of this variable appearing to flow primarily through expectations of the effects of alcohol, ways of coping with stress, and association with heavy-drinking peers (Schuckit and Smith, 2000).

Recently, this prospective investigation has turned to an emphasis on gathering information on the offspring of our original subjects. For these children, data have been generated from the mother regarding pregnancy and birth complications (PBC), a phenomenon reported to relate to cognitive problems, externalizing behaviours, and internalizing symptoms in children (Pihl and Bruce, 1995; van Os et al., 1997; Tapert et al., 1999; Hill et al., 2000; Giancola and Parker, 2001; Tarter, 2002). The mother also supplied information on a host of additional activities and symptoms among these offspring beginning at age 4 years through the Child Behavioral Checklist (CBCL), along with a standardized evaluation of life functioning and psychiatric symptoms through the parent report version regarding children for the Semi Structured Assessment for the Genetics of Alcoholism (C-SSAGA-P) at age seven (Achenbach, 1991; Kuperman et al., 2001). This prospective process offers the opportunity to evaluate domains of life functioning repeatedly as these children mature, including externalizing and internalizing symptoms which are directly relevant to several of the models of substance-related problems. Data from these analyses can then be interpreted in the light of intensely studied families without the liabilities encountered in long-term retrospective analyses spanning a decade or more.

Information on 444 children had been gathered during the recently completed 15-year evaluation, with 503 offspring identified in the ongoing 20-year stage. As recently reported (Barnow et al., 2002; Preuss et al., 2002), 151 of these offspring were between ages 7 and 18, with complete data available on 146. These were analyzed to determine the relationship between the FH of AUD and internalizing as well as externalizing symptoms. Here, externalizing problems were not related to the presence or absence of a FH of AUD overall, but were higher in children with high family densities of alcoholism. Internalizing symptoms had a relatively weak relationship to a FH of AUD, but were robustly tied to the FH of non-substance-related (i.e. independent) mood and anxiety disorders in those families.

Thus, the studies to date on these offspring have evaluated how a FH of AUD relates to a single area of functioning. The current analyses explore the interrelationships among several characteristics of the children from these intensely studied families, and their link to an intermediate phenotype likely to be related to the future risk for AUD, externalizing symptoms. We have focused on aspects of the deviance-prone model, limiting the number of domains to those appropriate to 146 subjects by selecting those characteristics we projected might be most relevant to this blue-collar and white-collar set of families.

SUBJECTS AND METHODS

Data were generated with written informed consent from the 15-year follow-up of the families of the 453 sons of alcoholics and controls (Schuckit and Smith, 1996, 2000). The original subjects, the fathers of the offspring reported here, had been chosen from among respondents to a questionnaire mailed to random students and non-academic staff at a university, selecting 18- to 25-year-old drinking but not alcohol-dependent sons of alcoholics and FH negative controls matched on demographic background and drinking histories. At the time of enrolment, subjects were evaluated with an interview based on the Schedule of Affective Disorders and Schizophrenia and, subsequently, on the Structured Clinical Interview for DSM-III-R (Spitzer and Endicott, 1977; Spitzer et al., 1992). Data were gathered regarding demographic backgrounds, FH of SUD and major psychiatric conditions, as
well as alcohol and drug use patterns and problems. The men also participated in alcohol challenges where their levels of response to alcohol were determined after consuming the equivalent of three-to-five standard drinks (Schuckit and Smith, 2000).

The original subjects were followed-up with face-to-face structured interviews approximately 10 years after initial testing (T10), at which time all 453 had been located, and all but three (99.3%) agreed to an evaluation with themselves and an additional informant, usually the spouse (Schuckit, 1998). These interviews focused on changes in demography, FH, and SUD or psychiatric conditions for the subject during the interval since initial testing.

Five years later (i.e. 15 years after the initial evaluation or at T15), subjects were located once again, and complete information was obtained on the 440 men among the 449 who were still living (Schuckit and Smith, 2000). At T15, for all subjects who had married, the spouse was interviewed regarding her demography and personal as well as FH of SUD and psychiatric conditions using an interview similar to that originally given to the subjects and to the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA) instrument (Bucholz et al., 1994; Hesselbrock et al., 1999). Spouses were queried regarding pregnancies, and information gathered on gestation, parturition, and the neonatal period for each child. Mothers of the 151 offspring who were ages 7 through 17 years (out of the total of all ages of 444 children) were asked to fill out the CBCL (Achenbach, 1991). This instrument consists of 20 questions about the child’s activities, participation in organizations, completion of chores, interactions with siblings and peers, as well as school performance, along with 120 items on behavioural and emotional problems, with the latter scored on a three-point scale from not true to very true. The CBCL can be used to generate standardized scores, including those that summarize acting out or problematic behaviours (externalizing symptoms), as well as a score for depressive and anxiety-type symptoms (internalizing symptoms) (Puttler et al., 1998; Barnow et al., 2002; Preuss et al., 2002). Major scores on the CBCL have been reported to have a three-month retest reliability as high as 0.84, agreement among parents of 0.62, and an adequate external validity when compared to clinical evaluations (Bird et al., 1991; Puttler et al., 1998; Johnston et al., 2000).

Parents of children age seven and above were also given to a face-to-face SSAGA interview about the children (Kuperman et al., 2001; Barnow et al., 2002; Preuss et al., 2002), selecting that minimum age as most appropriate for both gathering information from the major interview and questionnaire used, and slightly before the youngest age at which children might be expected to be exposed to alcohol (Reich, 1988; Reich et al., 1993; Johnston et al., 2001). The basic instrument, with good to excellent validity when compared to another standardized interview (Hesselbrock et al., 1999), reviews 17 Axis I psychiatric conditions and the ASPD, and has been reported to have good to excellent across interviewer and retest reliabilities, with kappas in excess of 0.60 for most diagnoses, including alcohol dependence (Bucholz et al., 1994). The parent version reporting on children (C-SSAGA-P) was developed as an alternative form to facilitate evaluation of demography and psychiatric symptoms and syndromes in children through age 17 (Kuperman et al., 2001).

Data from these instruments and from interviews of the original subjects and spouses were used to generate a series of scores in domains. The ideal evaluation would require personal interviews and testing of all children, a step which was considered too intrusive for these parents to accept for young offspring. Thus, the construction of relevant domains was guided by a parent’s perception of the child’s functioning in areas that often parallel domains currently being studied in the original subjects. Additional areas relate to domains hypothesized in our model to be relevant for young children in families of alcoholics and as predictors of future alcohol involvement and associated problems.

For these analyses, the lifetime FH of DSM-III-R alcohol abuse or dependence in biological grandparents (GRPAR alc) was noted as the number of the four such individuals with an AUD, while a similar approach was used regarding a score of zero to two for the biological parents (PAR alc) (American Psychiatric Association, 1987). The lifetime FH of affective and anxiety disorders reflected a similar score for grandparents (GRPAR aff) and parents (PAR aff) for DSM-III-R independent (i.e. not including substance-induced) major mood syndromes (major depressive or bipolar disorders) or major anxiety disorders (panic, generalized anxiety, or phobic conditions; American Psychiatric Association, 1987; Schuckit et al., 1997). As explained in more detail elsewhere (American Psychiatric Association, 1994; Schuckit et al., 1997), an alcohol-induced disorder is diagnosed if the syndrome only occurred in the context of a relevant substance dependence, while an independent condition either predated the dependence or occurred during a several month or more subsequent period of abstinence. Evidence of PBC was determined from the z-score transformation of three items gathered from the interview of the mother including a gestational period less than nine months, having to be placed in an incubator after birth, and staying in the hospital longer than the mother at birth (α = 0.78). Symptoms of externalization (EXT) were measured by the sum of z-score transformed values from the CBCL T-score (i.e. sex- and age-corrected) externalizing, as well as a count of the number of externalizing symptoms endorsed from among the 42 possible appropriate problematic behaviours queried on the C-SSAGA-P (α = 0.82) (Barnow et al., 2002). The internalizing score (INT) (α = 0.73) was a combination of the CBCL internalizing T-score and a symptom count of the 39 questions relevant to anxiety and mood symptoms in the C-SSAGA-P (Preuss et al., 2002). The stability of the home environment (HOME) during the 5 years leading up to the interview was measured on a 3-point scale relating to the continuous presence in the home of the biological father and/or mother. For all domains, higher scores indicated a more problematic condition.

Based on the literature cited above, these domains were hypothesized to relate together in the manner described in the Introduction and presented as a modified deviance-prone model in Fig. 1. Briefly, both FH measures of AUD were predicted to relate to EXT, as well as to each other. AUD in the parents were predicted to relate to PBC, as well as to the measure of HOME, while both PBC and HOME were felt to be likely to relate to EXT. Because internalizing symptoms (INT) play a role in the hypothesized deviance prone model, the FH of affective and anxiety disorders were included, with projections that such disorders in parents and grandparents would
indicated by the ratio of the chi-squared ($\chi^2$) were calculated to estimate the overall model goodness of fit: of the error terms were set at one, and the significance level of all indicators were manifest, individual pathway coefficients Reflecting these numbers and the nature of the available data, is generally accepted as between 5:1 and 10:1 (Bentler and Chou, 1987). We have used the even more conservative approach of the root mean square error of approximation (RMSEA; Steiger, 1990; Hu and Bentler, 1998). For 1987) noted that as few as 100 participants can be acceptable, and the recommended ratio of sample size to free parameters (1:4.0: Arbuckle and Wothke, 1999). While the traditional power tables (e.g. Cohen, 1988) are not directly applicable to structural modelling, and while there are no hard and fast rules for the number of variables appropriate for an SEM, Schumacker and Lomax (1996) have presented some guidelines pointing to the relatively wide range of appropriate sample sizes for this approach. For example, Ding et al. (1995) and Boomsma (1987) noted that as few as 100 participants can be acceptable, and the recommended ratio of sample size to free parameters is generally accepted as between 5:1 and 10:1 (Bentler and Chou, 1987). They have used the even more conservative approach of Jöreskog (1993), which would justify up to nine domains including the dependent variable, in an analysis of 145 subjects. Reflecting these numbers and the nature of the available data, all indicators were manifest, individual pathway coefficients of the error terms were set at one, and the significance level of the individual pathways was set at less than 0.05. Five indices were calculated to estimate the overall model goodness of fit: the chi-squared ($\chi^2$) goodness of fit (GFI; Jöreskog and Sörbom, 1989), the Bentler–Bonnett non-normed fit index (NNFI; Bentler and Chou, 1995), the comparative fit index (CFI; Bentler, 1990), and the root mean square error of approximation (RMSEA; Steiger, 1990; Hu and Bentler, 1998). For $\chi^2$, fit is indicated by the ratio of $\chi^2$ to the degrees of freedom, with a good fit reflected by a rate of 2 or 3 to 1 (Carmines and McIver (1981). GFI values are always between 0 and 1, where a value of 0.8 indicates a perfect fit and a value above 0.9, a good fit (Schumacker and Lomax, 1996). NNFI is less distorted by small samples, and the typical range usually lies between 0 and 1, with values close to 1 indicating very good fit. CFI values range from 0 to 1, with values close to 1 indicating a very good fit. For RMSEA, values close to 0 denote better fit, with 0.05 or less indicating a close fit, while values of 0.08 or less indicating a reasonable error of approximation (Browne and Cudeck, 1993).

RESULTS

Relevant data were initially available on 151 subjects, five of whom were excluded because of key missing information. In one additional instance, scores on both of the relevant CBCL scales were greater than two standard deviations higher than the mean, and this subject was deleted from the current analysis. For the remaining 145 subjects, the offspring were within the ages of 7 and 17 years, had a mean of 10.3 ± 2.74 years, were fairly equally divided by sex (52.4% male), and had 5.1 ± 2.80 years of schooling at T15. Regarding race and ethnicity, for 90.3% both parents were Caucasian, and in 6.9% at least one parent was white Hispanic, 0.7% Asian, and 0.7% African-American, with 1.4% other. The mean income for the family at follow-up was $8124 ± $5715 per month, and years of education were 18.4 ± 2.83 for fathers and 15.4 ± 2.53 for mothers.

A major goal in the current paper with these young subjects is to describe how potential predictors of future substance use and problems related to the intermediate phenotype of externalizing symptoms as well as to each other at this early age. Regarding these domains, reflecting the selection criteria for the original families, 80.7% of the offspring had at least one parent or grandparent with alcohol misuse or dependence, including 51.0% with one such relative, 22.1% with two, 4.8% with three, and 2.8% with four. One in three offspring (33.1%) had at least one relative with an independent mood or anxiety disorder, including 25.5% who had one relevant parent or grandparent, 3.4% with two, and the remainder with three such relatives. For HOME, 11.0% of the offspring had at least one parent not living with them. The mean z-scores on the additional domains were 0.003 (± 0.83) for PBC, 0.009 (± 1.62) for EXT, 0.006 (± 1.55) for INT.

Table 1 presents the zero-order correlations among the seven variables. All but one domain significantly related to externalizing symptoms, with the exception being the parental FH of AUD, where the FH relationship was carried solely by AUD in the extended family. The two alcohol FH variables correlated positively with each other, and each related to the absence of one or more of the biological parents in the home. FH_{aff} measures were related, each correlated with all domains except for the two FH of AUD and HOME, and demonstrated their highest correlations with INT, with the latter variable correlating with PBC.

The test of the hypothesized model from Fig. 1 yielded excellent goodness of fit indices ($\chi^2 = 13.62; d.f. = 11; P = 0.26$, with a $\chi^2$ to d.f. ratio of 1.24; GFI = 0.98; NNFI = 0.95; CFI = 0.98; and RMSEA = 0.041). Dropping non-significant paths yielded the empirical model presented in Fig. 2, where

![Fig. 1. Theoretical model of relationships among the eight observed variables in a study on the correlates of externalizing symptoms in children from families of alcoholics and controls.](image-url)
only vectors with beta weights (or standardized regression coefficients) that were significant (\(P < 0.05\)) are retained. Again, the relationships among variables were all in the predicted direction and the final \(R^2\) (the proportion of the variance accounted for) was 0.26. Overall, the goodness of fit of this model was high (\(\chi^2 = 51.10; \text{d.f.} = 44; P = 0.22\); with a \(\chi^2\) to d.f. ratio of 1.16; GFI = 0.92; NNFI = 0.93; CFI = 0.95; and RMSEA = 0.034), suggesting that this model fits the data for boys and girls equally. Regarding the path from HOME to INT, the beta weight was 0.36 (\(P < 0.001\)) for boys while it was –0.05 (\(P < 0.05\)) for the girls. Second, regarding age, while this variable correlated with HOME (0.39; \(P < 0.001\)), INT (0.21; \(P < 0.02\)), and EXT (0.20; \(P < 0.02\)) on the zero-order level, when age was added to the model in Fig. 2, the relationships of age to INT and EXT were no longer significant, and the overall \(R^2\) remained at 0.26. Also, selecting the usual age break used for the CBCL, when all paths were constrained to be equivalent across two age groups (11 or younger versus 12 or older), this two-group model fits well (\(\chi^2 = 48.35; \text{d.f.} = 46; P = 0.34\), with a \(\chi^2\) to d.f. ratio of 1.07; GFI = 0.92; NNFI = 0.96; CFI = 0.97; and RMSEA = 0.23), suggesting this model fits the data from the younger and older subjects equally.

It is important to note that, in light of the young age of the sample, information regarding most domains was obtained only from the parents. An effort was made to estimate the probable accuracy of the parents’ reports through an evaluation of
data from subjects where information had already been gathered from the next stage, T20 evaluations. Here, 63 children age 13 and above have been subsequently interviewed to date with the adolescent version of the SSAGA (C-SSAGA-A), while the parents reported on similar items through the C-SSAGA-P. In that subset, the correlation between the number of conduct-related symptoms reported by parent and child was 0.90 \((P < 0.001)\), and, reflecting few subjects with more than one mood or anxiety problem noted, the correlation for the presence or absence of such difficulties was 0.81 \((P < 0.001)\). In each instance when a discrepancy was seen, the child was more likely to report an item than the parent.

**DISCUSSION**

This study evaluates aspects (i.e. domains) of a modified deviance-prone model of AUD, focusing on the intermediate phenotype of externalizing symptoms. The overall investigation from which these data came offers several unique attributes including well characterized families where the parents of these offspring have been evaluated over time, use of some similar domains in both generations, and a paradigm where, eventually, specific genes can be tested as they relate to additional domains of influence (Schuckit and Smith, 1996, 2000; Schuckit, 2002). The original probands, the fathers of these offspring, were chosen to facilitate the evaluation of several models of risk for AUD while excluding extreme influences, such as ASPD, bipolar disorder, or schizophrenia, which might overpower the potential impact of other domains. The current report focuses on cross-sectional relationships among selected domains at a relatively early age, laying a base for the future prediction of substance-related problems.

The hypothesized model worked well with excellent goodness of fit indicators for the hypothesized and empirical pathways, and the latter explaining 26% of the variance. The empirical model appeared to work similarly in both sexes and across ages, although with sex, the path from HOME to INT appeared to be important only for boys. The latter might reflect a special vulnerability for boys when a father is absent from the home, as fathers were more often the absent parent. The impacts of age and sex were also considered by using adjusted \(T\) scores from the CBCL for internalizing and externalizing symptoms. Future follow-ups of this population will determine whether the model is stable over time, and if the current domains predict future substance-related problems.

The dependent variable, externalizing symptoms, proved to be important to this model despite the exclusion of families where extreme externalizing conditions such as ASPD were prominent. These results support the usefulness of externalizing-type behaviours even in the more usual family from which alcoholics come, as only 20% or so of alcohol-dependent persons carry an ASPD label (Bauer and Hesselbrock, 1999; Schuckit, 2000). At the same time, the complex nature of EXT is supported by the links to FH of both AUD and mood/anxiety disorders, as well as to internalizing symptoms and to the number of biological parents in the home.

The report of the child’s mood and anxiety symptoms (INT) was included here because it is often incorporated in evaluations of deviance-prone models. Our prior analyses of this domain demonstrated the expected close relationship between INT and a FH of similar disorders, and thus, the FH domain was also incorporated. Both INT and the FH of affect and anxiety-related variables worked well within the model, with INT in a non-recursive relationship with EXT. This might reflect a tendency of relatively young children with acting out behaviours to develop sadness and anxiety symptoms as they face subsequent problems, and/or the probability that internalizing disturbances can at times present as behavioural difficulties. As reported in prior evaluations (Schuckit et al., 1997; Schuckit and Smith, 2000; Preuss et al., 2002), neither FH of alcoholism measure related significantly to INT on either a zero-order level or within the model.

The association between an AUD in a grandparent (but not in a parent) and the externalizing symptoms in these offspring is worthy of note. Regarding the parents, our original subjects were only included if they were relatively highly functional. Thus, the grandparents, having no such restrictions, might have a wider range of externalizing phenomena, and might be better predictors of a predisposition toward such behaviours than the parents themselves. Perhaps similar factors operate regarding the FH of mood and anxiety disorders. The current results are consistent with our prior work with the fathers of these children where another measure of externalizing-type behaviours, behavioural undercontrol, related to the future risk for AUD, but was not directly tied to the FH of alcoholism (Schuckit and Smith, 1996). Similarly, the current findings are consistent with our prior analysis where no significant tie was found for externalizing symptoms and the presence or absence of a FH of AUD in a subset of these children (Schuckit et al., 2000). The relationship of externalizing symptoms to an FH of alcoholism in parents may be more prominent in other samples, especially those where the original subjects include persons with ASPD.

A second unexpected finding was the limited role for a history of PBC. The one correlation to the FH of mood and anxiety disorders in parents might reflect difficulties during pregnancy or the early neonatal period when mothers were depressed, and such conditions are more prevalent among women than are AUD (Regier et al., 1988; Kessler et al., 1996). Perhaps PBC might operate more robustly in the model among less highly functional families or those where parents are not as highly educated as our current group. The results might also reflect the limited scope of the PBC measure used here, and direct evaluations of cognitive performance in the children will be incorporated in the future as offspring over age 12 are directly tested. The current findings, however, may demonstrate how the impact of any single domain in any model or research paradigm is likely to depend on the context (e.g. other domains) within which the variable is measured.

While there are a number of relatively unique aspects to this study, the results must be evaluated in light of the specific methods used. First, all evaluations were carried out cross-sectionally in a modest sized sample, with the latter dictating that a conservative eight domains could be used in the SEM. Second, although the young age of the subjects facilitates evaluation of phenomena at an appropriate age while avoiding the problems of long-term recall, at this stage it is inappropriate to directly measure our eventual dependent variable, alcohol use and related problems. Thus, the emphasis here is on what is felt to be an important intermediate phenotype related to the AUD risk. Third, our results evaluate part of a
single model, and future analyses will turn to additional models and a wider range of components of the deviance-prone hypothesis as the number of offspring in an appropriate age range grows. Fourth, reflecting the young age of this sample, all data were obtained from a parent. Future evaluations of offspring over age 12 will incorporate additional domains relevant to the deviance-prone, socialization, and affect-related models including the direct evaluation of cognitive functioning, the child’s perceptions of deviance and substance use in peers, alcohol expectancies, ways of coping with stress, along with an item felt to be important to a socialization model, the level of response to alcohol. Fifth, some of the domains (e.g. HOME) were evaluated through use of a single variable, and, because the modest size of the sample, when multiple items were used, the manifest score was incorporated in the evaluation of the model. Latent variables will be used in the next stage of the work when we estimate 350 subjects will be age 12 or older. Also, a shorthand was used to label each of the domains, with the result that the reader is encouraged to carefully consider the actual sources of information while interpreting the data. Finally, while the population used here is likely to represent a substantial proportion of people in the USA (i.e. more functional blue- and white-collar Caucasian and Hispanic families), the original decision to exclude individuals with several severe psychiatric conditions and to emphasize more highly functional families is also likely to impact on the results.

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