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Relationship of Age of First Drink to Child Behavioral Problems and Family Psychopathology

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Background: Studies have implicated a wide variety of variables as being associated with an early age of first drink (AFD). AFD in turn has been associated with a variety of negative outcomes in adolescence and early adulthood. This study is designed to quantify the contributions of these antecedent variables to prediction of AFD; in particular it will carefully examine the involvement of variables in four areas (child characteristics, family demographics, family psychopathology, and child behavior problems).

Methods: Using data from a multicenter study on alcoholism, we first investigated the differences between two groups of children (ages 7 to 17 years), one from families heavily loaded for alcohol dependence and the other from population controls. Second, a multidomain, multistep regression model using child characteristics, family demographics, family psychopathology, and child behavior problems was performed to determine significant contributors to predicted AFD.

Results: Five variables initially contributed to the prediction of AFD. These included gender, age at interview, the number of adult sibs with alcohol dependence, being held back a year in school, and conduct scale score. However, the number of conduct symptoms appeared to contain the contributions of gender and being held back a grade in school, and these two variables were subsequent removed from the model. The remaining three variables explained 45% of the model variance; age at interview accounted for 38.3%, conduct scale score accounted for 6.2%, and the number of alcohol-dependent adult sibs accounted for 0.5%. No family history measures of alcohol dependence or antisocial personality disorder were contributory to the prediction model for AFD.

Conclusions: Both the "number of conduct symptoms" and the "number of adult sibs with alcohol dependence" are inversely associated with predicted AFD. The latter variable appears marginally predictive of AFD and suggests a condition in which the child's household, regardless of strength of family history of AD (or antisocial personality disorder), appears conducive to early drinking. Thus, child and environmental factors are stronger predictors of age of first drink than family history.

Key Words: Age of first drink, Child behavioral symptoms, Family psychopathology.

INTRODUCTION

ALCOHOL USE IN late childhood/early adolescence is a common event. Johnston et al. (2003) reported that more than 50% of surveyed 8th graders (typical age of 14 years) have already used alcohol and that approximately half of these have already had at least one episode of being "drunk." Studies have demonstrated early age of first drink (AFD) may be associated with increased rates of childhood psychiatric disorders, lowered success in school and extracurricular activities, increased criminal behavior, and lowered overall life satisfaction and productivity (DeWit et al., 2000; Guo et al., 2000; Kuperman et al., 2001a; Legrand et al., 1999; McGue et al., 2001b; McGue et al., 2001a; Prescott and Kendler, 1999; York, 1999). This trend continues into adulthood, with reported increases in alcohol-related diagnoses (DeWit et al., 2000; Grant and Dawson, 1997; York, 1999) and non–alcohol-related problems of increased rates of psychiatric diagnoses, poorer physical health, less stability of employment and committed relationships, and increased criminal behavior (Sussman et al., 2000; York, 1999).

Despite these increased risks, the majority of children who drink alcohol by 8th grade do not go on to have significant problems. A likely cause for this variability may be the number or types of risk factors a child has that are associated with early AFD. Hypothesized risk factors for early AFD include being male (Dawson and Grant, 1998; Disney et al., 1999), having parents with alcohol dependence or antisocial personality disorder (ASPD) (As-

From The Collaborative Study on the Genetics of Alcoholism (COGA) (Principal Investigator: H. Begleiter; Co-Principal Investigators: L. Bierut, H. Edenberg, V. Hesselbrock, Bernice Porjesz), which includes nine different centers where data collection, analysis, and storage took place. The nine sites and Principal Investigators and Co-Investigators are University of Connecticut (V. Hesselbrock); Indiana University (H. Edenberg, J. Nurnberger Jr., P.M. Conneally, T. Foroud); University of Iowa (S. Kuperman, R. Crowe); SUNY HSCB (B. Porjesz, H. Begleiter); Washington University in St. Louis (L. Bierut, J. Rice, A. Goate); University of California at San Diego (M. Schuckit); Howard University (R. Taylor); Rutgers University (J. Tischfield); and Southwest Foundation (L. Almasy). Lisa Neuhold serves as the NIAAA Staff Collaborator. This national collaborative study is supported by the NIH Grant U10AA08403 from the National Institute on Alcohol Abuse and Alcoholism (NIAAA).

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sanangkornchai et al., 2002; Kuperman et al., 1999; Legrand et al., 1999), having a childhood diagnosis of a disruptive disorder (Assanangkornchai et al., 2002; Disney et al., 1999; Kuperman et al., 2001a; Kuperman et al., 2001b), positive peer attitudes toward substance use (Botvin et al., 1998; Hawkins et al., 1997; Mcculler et al., 2001), and other home environmental factors such as poor supervision and inconsistent and/or harsh discipline (Griffin et al., 2000; Kuperman et al., 2001b). However, complicating the relationship of early AFD to later negative outcomes is the fact that many of these risk factors have been hypothesized as being related themselves to these same negative outcomes (Garmezy and Masten 1994; Patterson et al., 1989; Patterson and Stouhamer–Loeber, 1984).

The goal of this study was to begin to untangle the complicated relationships between these risk factors and early AFD. Specifically, we examined whether AFD can be predicted better by the total number of proposed risk factors or by the existence of a specific type of risk factor(s). As part of these analyses, particular attention was paid to the contributions of a family history of alcohol dependence or ASPD (in parents, adult siblings, or adult second-degree relatives) to the predication of age of first drink.

The ability to test these relationships is brought about through the ongoing Collaborative Study on the Genetics of Alcoholism (COGA). This study provides an opportunity to examine these factors closely, since the data collected provides detailed information on all family members including children (ages 7 to 17 years). The data include the presence of the child's psychiatric symptoms, the age of onset of the child's use of alcohol and other substances, psychiatric diagnoses of the child's parents and other family members, and the child's home environment. Using these data, the current study will explore how well risk factors in the categories of child characteristics, family demographics, family psychopathology, and child behavioral problems predict age of first drink. Future studies will then be able to build on these results to determine whether these hypothesized risk factors are better predictors of negative outcomes than early AFD by itself.

MATERIALS AND METHODS

The subjects in this study were all participants in COGA. COGA is a multicenter, longitudinal project composed of six subject collection centers located in California, Connecticut, Indiana, Iowa, Missouri, and New York. The goal of COGA is to study various behavioral, biochemical, genetic, neuropsychological, and neurophysiologic phenomena related to alcoholism. Institutional review boards at all sites reviewed and approved the study design and procedures. Parents and their children provided written informed consent and assent, respectively, for participation in this study.

Subjects

Two types of families made up the COGA database; those defined as COGA families and control families. The COGA families were chosen by first inviting an adult who was receiving treatment for alcoholism to enter the study. A trained research assistant using the Semi-Structured Assessment for the Genetics of Alcoholism (SSAGA) (Bucholz et al., 1994; Hesselbrock et al. 1999) interviewed this person. Individuals fulfilling criteria for both a DSM-III-R diagnosis of alcohol dependence (AD) as well as a Feighner diagnosis of definite alcoholism (Feighner et al., 1972) were then diagnosed as a COGA alcoholic and were asked permission to interview other adult first-degree family members. If at least two more of these relatives were COGA alcoholics, then all available relatives (including children and extended family members) were also interviewed. All adult family members provided not only personal information but also family history psychiatric data about other adults in the extended family. Control families were recruited from dental and family practice clinics, businesses, churches, and driver's license renewal centers and received the same assessment battery. Control families were not selected with respect to the presence or absence of any psychiatric disorder; alcohol dependence was present in approximately 30% of these families. The recruitment procedure has been more fully detailed by Begleiter et al. (1995).

In the first phase of the COGA project, trained research assistants interviewed a total of 1333 children, age 7 to 17 years, using language appropriate to age versions of the Child Semi-Structured Assessment for the Genetics of Alcoholism (C-SSAGA) (Kuperman et al., 1999). In addition, a guardian (usually the mother) was given a parent version of the C-SSAGA (C-SSAGA-P) to obtain corroborative data. All versions of the C-SSAGA used in this study allowed a diagnosis to be made for most DSM-III-R childhood disorders.

The subgroup of children included in this study had to have (a) a reported AFD and (b) a completed parental C-SSAGA-P. AFD was determined through the use of the C-SSAGA question "How old were you when you had your very first whole drink?" This definition was used because it indicated a substantial amount of alcohol ingestion; it was more than that typically used in religious ceremonies and more than just a sip that parents might offer to a child at a family event. Of the original 1333 children, 440 had a reported AFD along with a completed C-SSAGA-P, one a had a reported AFD on the C-SSAGA but had no completed C-SSAGA-P, and 892 denied any exposure to alcohol (of the latter, 60.0% were under the age of 12 years). The 440 children in the final sample consisted of 339 (77.0%) offspring from COGA families and 101 (23.0%) from control families.

The characteristics of these two groups were compared across of a number of measures gleaned from the literature hypothesized as being important risk factors for age of first drink. The first set of measures included the following variables: gender, age of the child at interview, parental ages at interview, family composition (categorized as the child living with either the biological mom, the biological dad, both biological parents, or no biological parents), yearly income (defined as whether the family in which the child resided was above or below the median yearly income of $30,000 for the 440 children), and the diagnosis of either AD or ASPD in the child's parents, adult siblings, and/or second-degree family members. The second set of comparison measures consisted of the variables used to define various lifetime behavioral problems, including problems at school (broadly defined as being held back a grade regardless of cause), the presence of symptoms suggestive of psychiatric illness, problematic alcohol use, and the use of tobacco and marijuana.

Since we desired to explore the range of behavioral problems, symptom count scales were used instead of the absence or presence of a full DSM-III-R diagnosis. Combining both child and parent C-SSAGA data, scales were created such that information from either interview resulted in a positive symptom endorsement (Bird et al., 1992). In general, the behavioral scale scores consisted of the sum of the endorsed symptoms present in the part "A" sections of the DSM-III-R for a given child psychiatric diagnosis. The attention deficit hyperactivity scale consisted of the first eight symptoms of attention deficit hyperactivity disorder (ADHD), since these were asked of all children and parents (children without a positive response to at least one of these symptoms were "skipped out" of the rest of the ADHD section). The oppositional defiant scale consisted of the first five symptoms in part "A" for the diagnosis of oppositional defiant disorder (ODD); children skipped to the next section if there were no positive responses to any of these symptoms. The conduct

scale comprised all 13 symptoms in the part "A" section of conduct disorder (there were no skip outs in this section). The internalizing scale was developed as a composite measure of symptoms from three different DSM-III-R disorders: the first two symptoms in the part "A" section of the DSM-III-R for major depressive disorder, the first four symptoms in the part "A" section for separation anxiety disorder, and the first symptom in the part "A" section for overanxious disorder. These symptoms were included because all children and parents were asked about these symptoms; children who did not have a positive response to items early on in one of these sections (or parents who were reporting on their child) moved on to the next section of the C-SSAGA. The Cronbach α score was 0.65 for this composite scale and indicated sufficient item consistency. The alcohol problematic use scale was derived from summing the number of positive endorsements of the first nine symptoms in the part "A" section of the DSM-III-R for psychoactive substance dependence (all subjects and their parents provided a response to these items). Dichotomous scales were created for marijuana use, tobacco use, and other street drugs (defined as cocaine, speed, opiates, hallucinogens, downers, and/or inhalants) by asking the child (or the child's parent) whether the child has ever tried these substances. An endorsement of use resulted in a positive scale score for a given substance category.

Whenever possible, a parental diagnosis of AD or ASPD was directly made from the parent's own SSAGA interview. If the parent did not have an SSAGA interview, family history data were used to impute a psychiatric diagnosis by requiring a minimum of three separate positive implications for a COGA and a minimum of two separate positive implications for a control family member (an imputed psychiatric diagnosis in control families was adjusted to require one less implication since fewer adult control family members were interviewed). Rice et al. (1995) have demonstrated this method as producing valid results in the COGA database. To keep the diagnostic methodology similar between the COGA and control groups, psychiatric diagnoses of adult siblings and any second-degree relative were imputed because it was unlikely that more than just a few control family member in these categories completed a SSAGA interview.

Diagnoses of AD or ASPD were examined in three different ways across COGA and control families. The first was to examine the percentages of children that had a specified relative class with either of these diagnoses. The second method, applied only to adult siblings, compared the average number of adult siblings with either of theses diagnoses. The final method, applied only to second-degree relatives, attempted to compensate for the finding that the number of relatives in the family history database was greater for COGA than for control children and compared percentages of second-degree relatives with these diagnoses (e.g., the number of adult relatives per family class with a given diagnosis was divided by the total number of unique adult relatives in that class contained in the family history database).

Statistical Analyses

The relationships between family type (COGA and control) and variables of interest were examined through the use of the χ^2 test of independence, Fisher exact test of independence, and two independent-samples t tests. These variables included gender; family composition; child's age, age of first drink, parents' ages; family income; and the presence of a parent(s), adult sibling(s), and second-degree relative(s) with AD or ASPD. The level was set at 0.05 to determine whether a variable was significant or not.

Finally, a series of sequential multiple regression analyses were performed, using four domains of variables, each containing multiple steps, to determine which variables contributed the most to predicted AFD. At each step in the model, homoscedasticity (an assumption for the regression tests) was verified. In Domain 1, only the child characteristics of gender and age at time of interview were considered. In Domain 2, two demographic characteristics and eight family psychopathology variables were evaluated: 1) Yearly income below $30,000, 2) Child's living arrangement with respect to his/her biological parents 3) Parental AD diagnosis (diagnosis of each parent used separately); 5) Number of adult siblings with AD or ASPD diagnoses (diagnosis entered separately); and 6) Percentage of second-degree relatives with AD or ASPD diagnosis (diagnosis entered separately. In Domain 3, seven child problematic behavior variables were considered. These were divided into four categories: 1) Held back a grade in school; 2) Externalizing behavioral scale scores entered individually for attention deficit hyperactivity, oppositional defiant, and conduct; 3) Internalizing scale score; and 4) Tobacco or marijuana use, entered individually.

RESULTS

Demographic findings of the COGA and control children are shown in Table 1. Control children were significantly older than COGA children, though mean age of reported first drink was similar. Both COGA mothers and fathers were on average approximately 5 years younger than control mothers and fathers. In general, COGA children appeared to have more nonspecific difficulties/stressors. The rate of COGA children being held back a year in school was almost 2.5 times higher than their control counterparts. COGA children were almost 3 times less likely to live in homes with both biological parents present. The child's family income also appeared to be affected by family type; COGA children were 2.5 times more likely to reside in homes with an annual income less than $30,000.

Table 2 shows the mean and standard deviation of the scores derived for the behavioral scales. As a group, these were all significantly elevated for COGA children except for problematic alcohol use. The rate of marijuana use was significantly higher in COGA children but did not differ between the two groups in use of tobacco or other street drugs.

As shown in the first part of Table 3, the percentage of COGA children who had either an alcoholic mother or father were much higher than the percentage of control children. The COGA rate was increased approximately 9-fold for mothers and approximately 2-fold for fathers compared with controls. COGA children had higher rates of AD in second-degree family members as well; increased rates were significant for maternal grandfathers (1.8-fold), paternal aunts/uncles (3.5-fold), and maternal aunts/uncles (2.3-fold).

The same set of analyses was performed for an ASPD diagnosis. As shown in the second part of Table 3, the percentages of COGA children with an ASPD mother or father were much higher than the percentages of controls. Among COGA children, 7.1% of them had mothers with a diagnosis of ASPD versus none of the control mothers. A diagnosis of ASPD in fathers was 4 times more common among COGA children than among controls. The percent of COGA children with a known maternal or paternal aunt or uncle with ASPD was approximately 10 times greater than that of controls.

The average numbers of adult siblings with AD was 0.20 $\pm$ 0.59 for COGA versus 0.08 $\pm$ 0.27 for control children; this difference was significant ($t = 3.0$, $p = 0.0032$, $df = 365$). The average numbers of adult siblings with ASPD was

Table 1. Child and family characteristics

Variable	COGA	Control	Statistic (*p* value), *df*
	N (%)	*N* (%)	
Child family type	339 (77.0)	101 (23.0)	
Child gender			0.1* (0.7223), 1
Male	161 (47.5)	50 (49.5)	
Female	178 (52.5)	51 (50.5)	
Child held back a grade	110 (32.4)	13 (12.9)	14.8* (<0.0001), 1
Family composition			115.4* (<0.0001), 3
Both biological parents	105 (31.0)	93 (92.1)	
Only biological mom	183 (54.0)	6 (5.9)	
Only biological dad	22 (6.5)	2 (2.0)	
Neither biological parent	29 (8.5)	0 (0.0)	
Family income <$30,000/yr	195 (57.5)	23 (22.8)	37.6* (<0.0001), 1
Age	Mean (SD)	Mean (SD)	
Child	*N* = 339	*N* = 101	
Interview (C-SSAGA)	15.0 (1.9)	15.8 (1.5)	4.2^ (<0.0001), 209
First drink	12.7 (2.3)	13.1 (1.9)	1.1* (0.2598), 438
Mother	*N* = 299	*N* = 101	
Interview (SSAGA)	39.3 (5.8)	44.3 (5.1)	7.6* (<0.0001), 398
Father	*N* = 213	*N* = 95	
Interview (SSAGA)	42.1 (6.8)	47.0 (4.9)	7.2^ (<0.0001), 245

* χ^2 test for independence.
* *t* test (equal variances).
^ *t* test (unequal variances).

0.05 ± 0.24 for a COGA compared with 0.03 ± 0.17 for a control child, a nonsignificant difference. Among COGA families, 51.6 ± 28.3% of known second-degree relatives had AD compared with 34.5 ± 37.8% among control families ($t = 3.9$, $p = 0.0002$, $df = 109$). Likewise, among COGA families, 10.2 ± 17.3% of known second-degree adult relatives had ASPD compared with 0.7 ± 4.5% among control families; this difference was significant ($t = 8.8$, $p < 0.0001$, $df = 404$).

Figure 1 diagrams the four domains and the variable(s) contained within each for the series of regressions used to predict the age of first drink. Variables that contributed significantly to the model for prediction of AFD were carried forward to the next step. Any variable that subsequently lost significance was dropped. At each domain (and each step) there was not significant evidence to reject the common variance assumption.

Variables examined in the first domain consisted of child characteristics and included gender and age at interview. Using this model, both age at interview and gender contributed significantly to the model; predicted AFD decreased by approximately 0.80 years for each year of age at interview ($p < 0.0001$) and by 0.6 years for male gender ($p < 0.0008$).

The second domain examined family demographics and psychopathology. Neither yearly income information, nor family composition data, improved the model's ability to predict AFD, and both variables were subsequently removed from the model.

The second step of Domain 2 added the variables maternal and paternal AD to the model developed at the end of Domain 1; again, neither variable significantly contributed to the model and both were dropped. Step 3 added the variables of maternal and paternal ASPD to the variables of age at interview and gender. These parental ASPD variables did not result in improvement of the model, and they were both removed. Step 4 added the variables "number of adult siblings with AD" and the "number of adult

Table 2. Child behavioral and substance use scale scores by family type

	COGA	Control	Statistic (*p* value), *df*
Behavior scale score:	Mean (SD)	Mean (SD)	
Attention deficit hyperactivity (Max = 8)	2.6 (2.4)	1.9 (2.3)	2.6* (0.0092), 438
Oppositional defiant (Max = 5)	1.3 (1.6)	0.8 (1.3)	3.6^ (0.0004), 201
Conduct behavior (Max = 13)	3.1 (2.5)	2.3 (2.3)	2.8* (0.0050), 438
Total externalizing (Max = 26)	8.9 (6.0)	6.4 (6.0)	3.7* (0.0002), 438
Internalizing (Max = 7)	2.2 (1.5)	1.7 (1.5)	2.7* (0.0067), 438
Problematic alcohol use (Max = 9)	1.4 (2.2)	1.1 (1.7)	1.2^ (0.2509), 207
Positive substance use	*N* (%)	*N* (%)	
Marijuana	167 (49.3)	34 (33.7)	7.6* ~ (0.0057), 1
Tobacco	121 (35.7)	34 (33.7)	0.1* ~ (0.7078), 1
Street drug (any cocaine, speed, opiates, hallucinogens, downers, and/or inhalants)	59 (17.4)	15 (14.9)	0.4* ~ (0.5472), 1

* χ^2 test for independence.
* *t* test (equal variances).
^ *t* test (unequal variances).

Table 3. Percentage of children with a biological adult family member diagnosed as alcohol dependent or antisocial personality disorder in 339 COGA and 101 control children

Diagnosis of	COGA family	Control family	χ^2 (p value)*
Alcohol dependence	*N* (%)	*N* (%)	
First-degree adult relatives			
Mother	151 (44.5)	5 (5.0)	53.3 (<0.0001)
Father	204 (60.2)	30 (29.5)	29.0 (<0.0001)
Adult sibling (at least 1)[Character F048 did not convert]	50 (14.8)	8 (7.9)	3.2 (0.0750)
Second-degree adult relatives[Character F048 did not convert]			
Paternal grandfather	94 (27.7)	19 (18.8)	3.2 (0.0728)
Maternal grandfather	129 (38.1)	21 (20.8)	10.3 (0.0013)
Paternal grandmother	28 (8.3)	4 (4.0)	2.1 (0.1442)
Maternal grandmother	37 (10.9)	6 (5.9)	2.2 (0.1395)
At least 1 paternal avuncular	128 (37.8)	11 (10.9)	26.0 (<0.0001)
At least 1 maternal avuncular	154 (45.4)	20 (19.8)	21.4 (0.0001)
Antisocial personality disorder	*N* (%)	*N* (%)	
First-degree adult relatives			
Mother	24 (7.1)	0 (0.0)	7.6 (0.0060)
Father	81 (23.9)	6 (6.0)	15.8 (<0.0001)
Adult sibling (at least 1)[Character F048 did not convert]	17 (5.0)	3 (3.0)	1.8 (0.3866)
Second-degree adult relatives[Character F048 did not convert]			
Paternal grandfather	6 (1.8)	0 (0.0)	108 (0.1782)
Maternal grandfather	6 (1.8)	0 (0.0)	108 (0.1782)
Paternal grandmother	0 (0.0)	0 (0.0)	Not applicable
Maternal grandmother	3 (0.9)	0 (0.0)	0.9 (0.3428)
At least 1 paternal avuncular	54 (15.9)	0 (0.0)	18.3 (<0.0001)
At least 1 maternal avuncular	74 (21.8)	2 (2.0)	21.5 (<0.0001)

ˆ 1 *df*.
* Family history imputed diagnosis.

siblings with ASPD" to the previous model. Of these two variables, "number of adult siblings with AD" significantly improved the model and was retained. The final step involved adding the variables "percentage of known second-degree relatives" with AD or ASPD to the resulting model from Step 4. Neither variable was significant, and both were dropped. The model at the end of Domain 2 resulted in a decrease of approximately 0.81 years for each year of age at interview ($p < 0.0001$), a decrease of 0.60 years for male gender ($p < 0.0008$), and a decrease of 0.37 years for each adult sibling with AD ($p < 0.0286$).

Domain 3 of this regression series consisted of four steps that examined variables that related to child behavioral problems. Step 1 added the single variable "held back a grade in school" to the model at the end of Domain 3; this variable was significant and was retained. Step two sequentially added the scale scores for attention deficit hyperactivity, oppositional defiant, and conduct to the four variables that remained significant. In this step, the conduct scale score significantly improved the model for predicted AFD, though the variables "gender" and "held back a grade in school" lost significance and were removed. Step

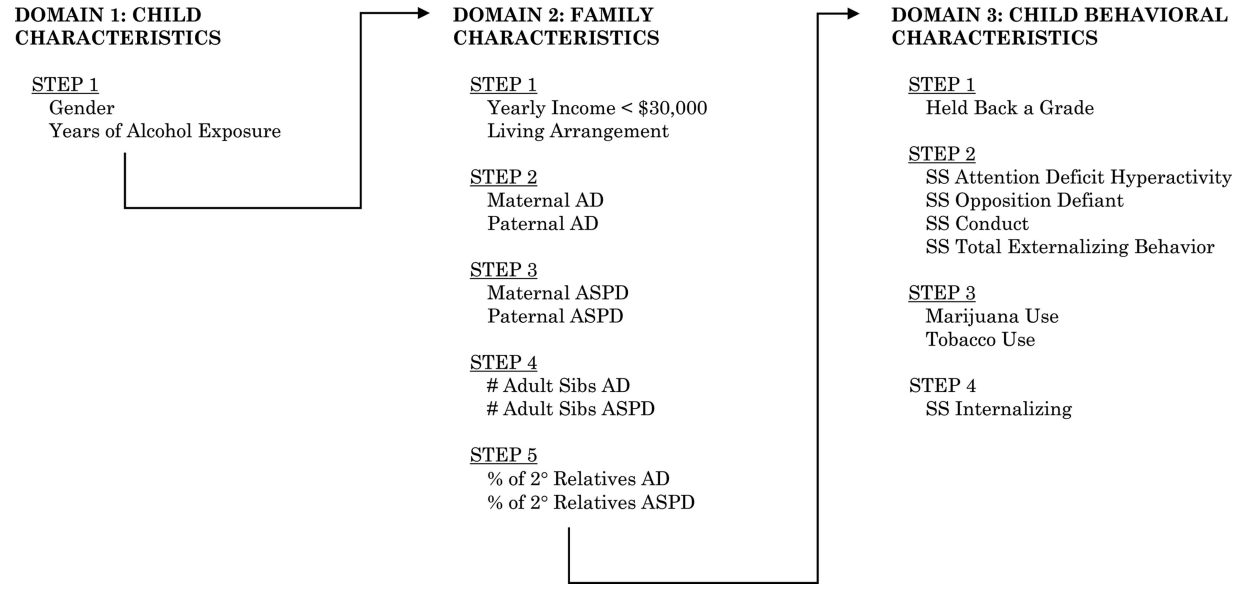

Fig. 1. Three domains and the variables contained within each for the series of regressions used to predict the age of first drink.

three added the variable internalizing scale score; again this variable was not contributory and was dropped from the analysis. The fourth step added the dichotomous variables of "used marijuana" and "used tobacco," though neither variable was significant and both were dropped from further consideration. (The variable "used other street drugs" was not used because of its relative rarity.) The final model consisted of the equation:

Predicted AFD in years = 0.15 + 0.82 * (age in years at interview) − 0.33 * (the number of alcoholic adult siblings) − 0.24 * (Conduct scale score).

This equation indicates that predicted AFD decreases: with decreasing age at interview ($p < 0.0001$); with each alcohol-dependent sibling ($p = 0.0413$); and with an increasing conduct scale score ($p < 0.0001$). This model explains 45% of the variance of predicted AFD; interview age accounted for 38.3% of the variance, conduct scale score accounted for 6.2% of the variance, and the number of alcoholic siblings accounted for 0.5% of the variance.

Because the addition of the conduct scale score to the model resulted in the variables of gender and "being held back a year in school" no longer remained significant, the relationships between conduct scale score and the latter two variables were further explored. The average conduct scale score for males was 3.85 ± 2.73 versus a scale score of 2.07 ± 1.88 for females, a significant difference ($p < 0.0001$, $df = 369$). Similarly, the average conduct scale score for individuals "held back a grade in school" was 3.83 ± 4.33 versus a scale score of 2.28 ± 2.13 for those not held back, again a significant difference ($p = 0.0001$, $df = 179$). This suggested that the conduct scale score contained the contributions of "gender" and "being held back a year in school" and resulted in the removal of these two variables.

DISCUSSION

This study explored the relationships between age of first drink and a number of variables described in the literature as being significantly associated with an early age of first drink. Two groups of children were compared in this study, the first were the offspring of families with a high loading for alcoholism, whereas the second consisted of offspring from population-based control families. Similar to the report by Johnston et al. (2003), the average age of first drink for these study subjects began during late childhood/early adolescence.

As the literature and our own ascertainment procedures would suggest, the two groups of children did vary across several different measures. The high-risk "COGA" children appeared to have significantly more stressors than controls. They were held back a grade more often at school, resided more often in homes missing one or both biological parents, resided in homes with lower yearly incomes, and secondary to the COGA selection process had higher rates of first- and second-degree relatives with AD (and ASPD). COGA children also scored higher on measures related to both externalizing and internalizing behavioral difficulties and were more likely to have tried marijuana. Neither COGA nor control children had frequent problematic drinking symptoms, though COGA children scores were marginally higher on this scale. Similarly, the rates of tobacco use and other street drug use were modestly higher in COGA than in control children

The important contribution of this study was the combining of associated variables in a multidomain, multistep regression to determine which variables contributed the most to prediction of AFD. In the first domain, gender and age at interview, were both important contributors to predicted AFD. In the next domain, neither yearly income nor family composition significantly contributed to the ability to predict AFD and were not retained in the model. Also within this domain, only the family psychopathology variables of the "number of adult siblings with a diagnosis of AD" contributed significantly to the prediction of AFD. The final domain added child behavioral variables; the solitary significant variable that persisted in this domain was the conduct scale score; this variable also accounted for the previous contribution of gender (Domain 1), which was subsequently removed. The remaining variables in the final model consisted of "interview age," "the number of adult siblings with AD," and "conduct scale score," and these variables accounted for more than 45% of the variance in the model's prediction of AFD.

There are three main findings from this study. First, interview age is the most significant contributor to the model and accounted for 38.3% of the model variance. This is not a surprising finding because the children in the study were constrained by two requirements: (1) their age at interview was between 7 and 17 years, and (2) they had a reported age of first drink in their C-SSAGA interview. Because of these requirements, age of first drink would never be greater than interview age and would most likely be within a few years of the interview age, since few children reported an AFD before the age of 12 years.

The second finding is that only two risk variables ("conduct scale score" and "number of adult siblings with AD") of the total of 18 variables hypothesized as contributing to the prediction of AFD were significant. As part of this finding is the unequivocal demonstration that having a "loaded" family history for alcoholism, including having a parent(s) with AD or having a high percentage of second-degree relatives with AD, does not influence age of first drink. There is a suggestion that children who have a greater number of adult siblings with AD are more likely to have a younger predicted AFD. However, this variable is only marginally significant and accounts for only 0.5% of the variance of the model. This supports the belief that genetic loading for alcohol dependence does not per se contribute to early AFD (since other "genetic" measures of family alcohol dependence do not contribute) but that having many siblings with alcohol dependence may somehow represent an environment conducive to early drinking.

The third finding is that there exists a significant inverse relationship between the conduct scale score and predicted AFD; the latter decreases as the number of conduct symptoms increases. Again, family history does not seem to suggest a direct genetic link for externalizing behavior and predicted age of first drink. None of the family history measures of the density of ASPD (an adult form of "severe" conduct symptoms) in parents, adult siblings, or adult second-degree relatives significantly contribute to the prediction of AFD. This appears to be in line with recent studies that suggest age of first drink may be the result of a number of environmental factors, including peer influences, accessibility of substances, and sibling interactions and is less heritable than problematic alcohol use (Prescot and Kendler, 1999; Rhee et al., 2003).

There are a number of strengths to this study. Data were collected through the use of trained interviewers in a methodical fashion with both COGA and control family members providing parallel data on both immediate and extended family members. On average, the interviews were performed less than 3 years after the child's actual reported age of first drink, minimizing the retrospective nature of the data. We purposely chose to use symptom counts instead of psychiatric diagnoses to cast the widest possible net to determine relationships between various behavioral problem areas and predicted AFD. This is also the only study to our knowledge that directly looks at the effect of parental and familial ASPD on the prediction of AFD.

This study has some limitations. First, study subjects came from two different family types, families with multiple alcohol-dependent adults and families selected as "population-based" controls. The ability to generalize the findings to other populations may be impaired because of this, though the results of the multidomain, multilevel regression indicated that there was no major contribution of the density of familial alcoholism on predicted AFD. The second limitation is that 60% of the children who did not report an actual AFD were under the age of 12 years and thus were likely to have limited access to alcohol. Perhaps as these children age and begin to drink, they may contribute additional data that will affect our model's ability to predict AFD. Third, multiple comparisons were made secondary to the multidomain, multilevel regression process with the potential of increasing type 1 error. However, only three of the 19 variables entered affected predicted age of first drink; two of these variables, age in years at interview and conduct scale score, were both highly significant ($p < 0.0001$), whereas the remaining one, the number of alcoholic adult siblings, was significant just under our cutoff level ($p = 0.0413$).

There are several questions this study does not answer. Although we are able to demonstrate that the variables selected contribute to prediction of age of first drink, the ages of the children limit the ability to determine whether these variables account for later development of alcohol, psychiatric, legal, job, or other life problems. The reported age of first drink in these children occurred on average approximately 2.5 years before their interview age. This length of exposure to alcohol was not associated with reported problematic alcohol use symptoms in 247 of the children (56.1%) in this study. At first glance, this suggests that in at least the short term, there may not be any relationship between age of first drink and early onset of alcohol-related problems. A different perspective would suggest that 193 of the children (43.9%) reported having at least one alcohol-related problem, and perhaps it is this subgroup that is at risk for the development of the negative outcomes associated with the selected variables. Since the COGA study is designed to be longitudinal in nature, many of the children have been or will be reinterviewed as they age. This will allow additional children an opportunity to report "age of first drink" and allow determination of whether the findings presented here still hold, and, more importantly, may allow us to tease out the contributions of each of the proposed variables to the risk of a negative outcome in young adulthood.

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