

Disinhibited Personality and Sensitivity to Alcohol Reinforcement: Independent Correlates of Drinking Behavior in Sons of Alcoholics

Patricia J. Conrod, Jordan B. Petersen, and Robert O. Pihl

Thirty nonalcoholic young (18 to 30 years) males with extensive multigenerational family histories of male alcoholism and 29 age-matched, family history-negative controls completed a variety of trait personality questionnaires, participated in a competitive stress task (while sober and alcohol-intoxicated), and were assessed for self-report and laboratory drinking behavior. Low academic achievement, disinhibited personality (as measured by the P Scale of the Eysenck Personality Questionnaire), and sensitivity to alcohol reinforcement were significant and powerful independent predictors of self-report (approximate $R^2 = 0.40$, $p < 0.0001$) and laboratory (approximate $R^2 = 0.20$, $p < 0.0001$) drinking behavior. There seemed to be some specificity with respect to the facets of drinking behavior accounted for by each independent variable: low academic achievement and sensitivity to alcohol reinforcement were more related to quantity of alcohol consumption and frequency of excessive consumption, whereas psychoticism was more related to self-reported negative consequences with alcohol. A cluster analysis on three identified correlates of drinking behavior indicated that the two experimental groups could be more accurately subdivided into three homogeneous types. Multigenerational family history males were disproportionately represented in two of these groups: one characterized by enhanced sensitivity to alcohol reinforcement and the other characterized by high psychoticism scores and alcohol-related problems.

Key Words: Risk for Alcoholism, Alcohol Reinforcement, Impulsivity, Heart Rate Reactivity, Drinking.

ALCOHOLISM IS an end-state that may be reached from a variety of starting points. Cloninger¹ has suggested that two distinct syndromes constitute the disorder. A number of recent reports have lent credence to his supposition.²⁻⁴ The type I syndrome apparently comprises anxious, passive-dependent (primarily female) alcoholics; and type II syndrome is typified, in contrast, by heritable factors, comorbidity with externalizing disorders [childhood conduct disorder, adult antisocial personality (ASP) disorder], early age of onset, and male gender. Hill⁵ proposed a third type of alcoholism—a potential subdivision of Clon-

inger's type II¹—characterized by familial transmission, and severe early onset, with absence of familial sociopathy and intoxicated antisocial symptomatology. Finn et al.⁶ recently reported that heterogeneity within alcoholic families reliably classifies offspring into subtypes that are then distinguished from each other based on the presence or absence of antisocial traits. Prospective studies investigating precursors to problem drinking in young adulthood have also documented the separate causal influence of ASP, and of non-ASP-related familial factors.^{7,8}

Children of alcoholics are at enhanced risk for alcoholism.⁹ Like alcoholics, they seem to comprise a heterogeneous group. Finn et al.⁶ recently showed that alcoholic families clustered according to the co-occurrence of alcoholism with ASP and other substance abuse, alcoholism and depression, or alcoholism without co-morbid psychopathology. Sher^{10,11} has suggested that three independent pathways may lead to familial alcoholism: deviance proneness, negative-affectivity proneness, and sensitivity to alcohol reinforcement. The risk factors that constitute these pathways may exist independently, or in combination. Even higher risk subgroups, within the population of children of alcoholics, may still be subdivisible. Sons of male alcoholics (SOMAs), for example—at higher risk for type II^{9,12} and other forms of alcoholism (particularly when characterized by extensive alcoholic pedigrees¹³)—seem characterized by the presence of a number of potentially unrelated but frequently co-occurring risk “factors” or “markers.”^{14,15} However, few studies have actually tested whether such proposed risk factors actually play a role, or mediate, drinking behavior of individuals at risk for alcoholism. Verification of the mediating/moderating status of a variable involves determining the amount to which the relationship between family history and alcohol consumption behavior is accounted for or changed when the variable is considered.¹⁶ Several studies have demonstrated an association between high-risk characteristics and actual patterns of alcohol consumption.^{2,5,7,8,14} However, few have actually tested their role in the mediation of family risk on drinking behavior. Two widely studied risk characteristics constitute the focus of the present investigation. The first might be defined, broadly as, “comparative lack of behavioral constraint” or “disinhibited personality”; the second, as comprising “enhanced sensitivity to the [rising blood alcohol

From the Department of Psychology (P.J.C., R.O.P.), McGill University, Montreal, Quebec, Canada; and Department of Psychology (J.B.P.), Harvard University, Cambridge, Massachusetts.

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Reprint requests: Patricia J. Conrod, B.A., Department of Psychology, McGill University, 1205 Dr. Penfield Avenue, W8/1, Montreal, Quebec, Canada H3A 1B1.

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concentration (BAC) limb] pharmacologically reinforcing effects of alcohol." The first goal of the study is to determine the mediational/moderational status of such characteristics with respect to their influence on drinking behavior of young men at elevated risk for alcoholism.

With regard to "lack of behavioral constraint" (i.e., tendency for undercontrolled, disinhibited, and impulsive behavior): a number of theoretical and empirical reports have suggested that children of alcoholics in general, and SOMAs in particular, may be characterized by a specific cognitive profile that results in an increased prevalence of impulsivity, hyperactivity, childhood conduct disorder, and associated school failure.^{8,10,11,14,15} However, it has been noted that such characteristics may only partially explain the link between family history of alcoholism and drinking behavior,^{5-8,13} in that they apparently determine the age of onset⁵ and severity (e.g., fighting when intoxicated, benders, and morning drinking) of alcohol-related problems in offspring of alcoholics.^{2,5,6} Such studies also concluded, however, that additional familial factors that seem unrelated to such a personality/cognitive profile must also mediate the drinking behavior of children of alcoholics. To our knowledge, only one study has directly investigated the mediational role of such a personality profile on drinking behavior in children of alcoholics,¹⁷ and it was found that lack of "behavioral constraint" actually moderated, rather than mediated, familial risk for alcoholism. In combination with family history of alcoholism, therefore, lack of behavioral constraint corresponded to frequency/quantity of alcohol consumption and severity of drinking-related problems; however, the relationship between alcoholic family history and alcohol overuse and related problems could not be accounted for by such lack. Therefore, the evidence appears in support of a partial relationship between disinhibited personality and risk for familial alcoholism. The focus on other risk factors unrelated to such a personality profile, thus, becomes important.

With regard to alcohol reinforcement: SOMAs with multigenerational family histories (MFHs) of alcoholism have been shown to demonstrate exaggerated sober autonomic "stress responses," in that they demonstrate elevated heart rate, digital blood volume amplitude, and muscle tension reactivity to novelty, threat, and aversive stimulation.¹⁸⁻²¹ Ethanol intoxication dampens the exaggerated sober autonomic "stress responses" in MFH SOMAs, and such dampening seems dose-dependent,²² somewhat population-specific,^{20,21} and related to self-report alcohol consumption behavior.²³ It seems that this particular group of high-risk individuals demonstrates a sensitivity to the negatively reinforcing (anxiolytic, at least in theory²⁴) "stress-response dampening" (SRD) effects of ethanol and that such sensitivity may, to some extent, explain elevated alcohol consumption patterns characteristic of such individuals.²⁴ Ethanol also produces a pronounced increase in resting baseline heart rate during the rising limb of the blood alcohol curve, among MFH SOMAs.²⁵ This particular re-

sponse to alcohol intoxication typifies treatment-seeking alcoholics,²⁶ and corresponds with elevated postethanol plasma β -endorphin levels²⁶ characteristic of MFH SOMAs.^{27,28} Pihl et al.²⁴ have suggested that such heart rate increase provides an index of incentive reward, specifically marking activation in the dopaminergically mediated "behavioral activation system"²⁹⁻³² critically involved in producing positive affective response to certain drugs of abuse.³³ MFH SOMAs thus seem to manifest sensitivity to at least two alcohol effects: the negatively reinforcing "stress-dampening" effects of alcohol and the positively reinforcing "psychostimulant" effects of alcohol. To date, sensitivity to alcohol reinforcement has not been found to correspond with any relevant personality characteristics implicated in the predisposition to alcoholism (e.g., sensation-seeking or disinhibited personality). It is possible that this particular risk characteristic might account for the portion of the relationship between familial history of alcoholism and drinking behavior that cannot be accounted for by disinhibited personality and other personality dimensions. However, hard evidence for this claim is lacking. The present investigation explores this possibility.

Finally, the demonstration that disinhibited personality and sensitivity to alcohol reinforcement differentially mediate the influence of familial history of alcoholism on drinking behavior would suggest that MFH SOMAs might comprise at least two homogeneous groups characterized based on the presence of these two vulnerability characteristics, or a particular combination of them. Finn et al.'s⁶ recent report on the heterogeneity in families of sons of alcoholics is in partial support of this hypothesis. Furthermore, the literature on the association between disinhibited personality, sensitivity to alcohol reinforcement, and various drinking-related variables suggests that risk factors associated with each subtype of MFH SOMA should be differentially associated with patterns of alcohol use and misuse.^{5-8,13} Thus, two additional goals of the present investigation were to determine whether MFH SOMAs cluster into different groups according to the degree to which they manifest each proposed risk factor for alcoholism and whether such groups would subsequently differ in patterns of alcohol use and misuse. It is hypothesized that, among those differentially susceptible to reinforcement, levels of consumption should be particularly increased (as such consumption would be directly rewarding). Among the "behaviorally unconstrained," by contrast, problems in regulating the consequences of drinking should be more evident.

We determined to investigate these hypotheses by selecting a group of relatively heavy-drinking, nonalcoholic male social drinkers who, nevertheless, may demonstrate a limited number of problem drinking symptoms. Half of the subjects were additionally characterized by the presence of MFHs of male alcoholism. All participants were administered an extensive battery of personality tests, designed primarily to measure disinhibited personality and sensation-seeking; subjected to an elaborated alcohol-challenge

procedure designed to assess sensitivity to the differentially reinforcing effects of ethanol; and interviewed and assessed in the laboratory for alcohol-use and related problems.

We were particularly interested in measuring trait psychoticism/sensation-seeking, with regard to personality, and used the Psychoticism Subscale of Eysenck's Personality Questionnaire (EPQ)^{34,35} and Zuckerman's Sensation Seeking Scale (SSS) (form V)³⁶ to that end. Eysenck's Psychoticism Scale is an extensively studied personality trait and has been recently identified as the strongest and most reliable marker available for the assessment of impulsivity, sensation-seeking, and disinhibition of aggressive tendencies.³⁷ Psychoticism, and its associated behavioral profile, is associated with heightened mesolimbic dopaminergic activity, reduced executive function,^{32,38} and abnormalities in serotonergic and norepinephrine pathways,^{38,39} and may emerge as a consequence of variance in the function of several relatively independent brain systems.³⁷⁻³⁹ Sensation-seeking has been linked, similarly, to increased risk-taking, novelty-seeking, and alcohol/drug use in a variety of contexts.⁴⁰

We also used a novel, more ecologically valid, alcohol-challenge test, incorporating a number of different stressors, including three different types of video games and the performance-dependent receipt of incentive reward (money) and punishment (mild electric shocks). We were hoping, in this manner, to elicit more varied and potent stress and SRD measures. In addition to self-report methods of assessment of drinking behavior, we had subjects participate in a sham taste-rating task designed to assess laboratory alcohol consumption. Volume of alcohol consumed on this task discriminates alcoholics⁴¹ and relatively heavy-drinking nonalcoholics⁴² from light social drinkers and total volume consumed correlates significantly with self-report estimates of quantity per drinking occasion.⁴³ Furthermore, individuals characterized by heightened sober cardiovascular reactivity to signaled shock consume more alcohol while completing this task than their comparatively nonreactive peers.⁴⁴ Finally, self-report mood was assessed to determine whether sensitivity to alcohol reinforcement is also reflected at a subjective level by self-report changes in positive and negative mood states.

We first investigated the mediator versus moderator function of three proposed vulnerability characteristics (i.e., disinhibited personality, sensitivity to negative reinforcement from alcohol, and sensitivity to positive reinforcement from alcohol) relative to each other and relative to other personality and psychophysiological characteristics that might be less strongly associated with risk for alcoholism. We hypothesized that sensitivity to ethanol reinforcement would be powerfully associated with level of alcohol use: that disinhibited personality would be associated most particularly with problems with drinking (and less specifically to amount of ethanol consumed); and that positive family history of alcoholism would be associated in some individuals with reinforcement sensitivity, and in others

with lack of behavioral constraint. We could not predict a priori, the nature of the relationship between disinhibited personality and alcohol reinforcement sensitivity. However, there is sufficient evidence to suggest that such a personality profile will be highly related to poor academic achievement and that both disinhibited personality and poor academic achievement should prove to be key mediational variables specific to one vulnerability pathway to early-onset alcohol problems in individuals with family histories of alcoholism.^{2,5,6,14,15}

METHODS

Subjects

Fifty-nine nonalcoholic, Caucasian males between the ages of 18 and 30 took part in the present study. Thirty of these had extensive family histories of male alcohol dependence and/or abuse; the remaining 29 had no alcoholism in the current or previous two generations.

Tests and Materials

Drinking Behavior. We obtained three self-report (number of drinks per month, number of times per year above legal intoxication, and number of problem drinking symptoms) and one laboratory measure (sham taste test) of alcohol consumption. The self-report quantity/frequency index of drinks/month was derived from a questionnaire. Frequency of drinking was measured as number of occasions alcohol was consumed per week, upon average, within the last 12 months. Those who drank alcohol less than once weekly were asked to estimate monthly or yearly frequency. Quantity was measured as estimated average number of drinks (one beer = one mixed drink = one glass of wine) consumed per drinking occasion. Number of drinks per month was calculated as four times the average weekly quantity/frequency measure if the subject indicated drinking on a per weekly basis. Frequency of intoxication was measured based on derived estimates of number of times per year blood alcohol level was ≥ 0.08 , using information regarding drinking duration, quantity/frequency consumed, and subject weight, according to the procedure detailed in Conrod et al.,⁴⁵ and based on a BAC estimation chart.⁴⁶ Brief Michigan Alcoholism Screening Test (MAST)⁴⁷ scores provided an index of behavioral/social problems resulting from alcohol consumption.

The laboratory measure of alcohol consumption was obtained using a sham taste test procedure.^{41,48} Subjects were presented with five numbered nonalcoholic and alcoholic drinks: 400 ml each of water, orange juice, vodka and orange juice, rum and coke, and rye and ginger-ale prepared in a 500 ml capped glass bottle, accompanied by a standard 8 oz drinking glass. Subjects were asked to pour and consume each drink, as desired, and to rate its taste according to a list of 15 adjectives comprising the Taste-Rating Scale. The alcoholic drinks consisted of 80 ml of 40% alcohol and 320 ml of mixer. Volume of 40% alcohol consumed was summed across beverages; the total was divided by each subject's body weight.

Personality Questionnaires. All subjects were required to complete the EPQ^{34,35} and Zuckerman's SSS (form V).³⁶

Mood Inventory. Indices of state mood and change in state mood were derived from the Profile of Mood States (POMS)-Bipolar,⁴⁹ which has been well-validated on various normal and psychiatric populations. This inventory assesses change along six dimensions: composed-anxious, elated-depressed, energetic-tired, agreeable-hostile, clearheaded-confused, and confident-unsure.

Heart Rate. Heart rate was recorded using a Grass model 7D polygraph. Two model 7P4 EKG tachograph preamplifiers recorded heart rate from Medi-Trace pallet electrodes placed on both sides of the chest.

Stress Challenge. Three video games (Centipede, Qix, and Defender) were played on an Atari video games system. Electric shocks (punishment condition) were administered using a Farral Instrument Mark I at an

intensity of 1.85 mA for 0.5 sec using a concentric electrode attached to the inside of the elbow of the subject's nondominant arm. Monetary (incentive) reward was presented in the form of \$2 bills. A tone played on a Yamaha stereo cassette deck model TC-800GL channeled through Realistic Nova 10 stereo headphones was presented to the subjects 10 sec before the potential delivery of the shock or money.

Procedure

Subject Screening and Selection. Subjects responded by telephone to advertisements featured in freely circulated arts and entertainment and community newspapers, and were briefly screened for familial risk and for alcoholism status. Subjects were informed, nonspecifically, that individuals with "various characteristics" were desired; to assess these characteristics, a series of questions regarding familial alcoholism and personal drinking behavior (brief MAST)⁴⁷ were presented. If respondents met initial inclusion criteria, they were invited into the laboratory to participate in day 1 of the study. Subsequent arrivals participated in an hour-long semistructured psychiatric interview, incorporating diagnostic criteria relevant to alcohol use derived from the Family History-Research Diagnostic Criteria (FH-RDC),⁵⁰ for family members, and from the DSM-III-R⁵¹ and brief MAST⁴⁷ for personal status. An additional, shorter semistructured interview was also conducted to gather information regarding cigarette and "recreational" or prescription drug use and abuse. All subjects were also screened for personal and family history of psychotic and bipolar disorder, using DSM-III-R⁵¹ criteria, and were administered a physical examination by a registered nurse.

Thirty subjects had MFHs of male alcoholism: an alcohol-dependent biological father and paternal grandfather, and one additional first- or second-degree alcohol-abusing or dependent biological male relative. The remaining 29 had no familial history (FH-) whatsoever of alcohol (or other substance) abuse or dependence among their siblings or other first- or second-degree relatives in the previous two generations of biological relatives.

All subjects scored 10 or less on the brief MAST (past 6 months) and were neither dependent upon nor abused alcohol according to DSM-III-R criteria. Lifetime brief MAST scores were also obtained for each participant. Subjects were excluded from participation if they had mothers who had been or who were presently characterized by alcohol abuse or dependence (according to DSM-III-R or RDC; to minimize potential fetal alcohol effect confounds); if they were undergoing treatment for any active physical or psychiatric medical condition or if they reported psychotic or bipolar disorder in any first- or second-degree blood relative.

Subjects were assigned randomly to the reward or punishment conditions (detailed herein); all were asked to abstain from consuming alcohol for 24 hr before participation and instructed to eat a light breakfast the morning of the testing session.

Laboratory Procedure. All qualified subjects were asked to return to the laboratory on a second day between 9 and 10 o'clock in the morning. Upon arrival, they were given a brief outline of the procedure of the study and were presented with a consent form to sign. All subjects were aware that they could withdraw from participation at any time in the experiment. A short, semistructured interview was conducted to collect demographic and personal drinking and drug consumption information; this was followed by administration of the EPQ and SSS. Subjects were then seated in a reclining chair, asked to complete the first POMS, and attached to the cardiovascular recording device. Subjects were asked to sit quietly and relax for 10 min; during this period, a 5-min resting (sober) baseline heart rate measure was obtained.

Each subject then played each of three different video games for six 2-min trials. The order in which they were played was randomly determined; subjects were allowed 30 sec of practice time per game before the recorded trials. Each subject was assigned a specific set of performance criteria: if he obtained a set of number of points, he could avoid a shock (in the punishment condition) or receive money (in the reward condition). If subjects in the punishment condition did not meet criteria, they heard a tone that signaled the possibility of shock. Ten seconds after the tone,

they were either administered a shock or not. Receipt of shock was randomly predetermined; however, no subject was shocked more than three times over the six trials within a given game period. If subjects in the reward condition met criteria, by contrast, they heard a tone signaling the possibility of receiving a \$2 bill. Receipt of this reward was also predetermined, and no subject received more than three \$2 bills over the six trials within a given game period.

One experimenter remained in the room with the subject to record his video game scores and to assign him the appropriate criteria. This experimenter remained blind to the subjects' (familial) group membership and to the order in which shocks would be administered to the subject. The other experimenter remained in an adjacent room, following the procedure through a one-way mirror, administering tones and shocks, and signaling for administration of money, when appropriate.

Immediately after the completion of the first video game, each subject participated in the sham alcohol taste test,^{41,48} described previously. After this task was completed, an experimenter cleared away the remaining beverages, took them to an adjacent room, and calculated the amount of alcohol consumed, determining the amount that had to be additionally administered to ensure that each subject consumed 1.0 ml 95% USP alcohol/kg of body weight. Subjects were left to complete a second POMS, and then to relax and sit quietly for 10 min.

Subjects then engaged in a second video game (playing a different game), following the same procedure used during their first play. The results from the second POMS recording and video game play will not be reported herein, because subjects differed with respect to the amount of alcohol consumed, during the sham taste test, before the task (as described). Immediately afterward, subjects consumed the remaining alcohol (mixed 5:1 with orange juice) necessary to bring their total dose to 1.0 ml 95% USP alcohol/kg body weight (as calculated by the experimenter). The mean dose administered in the second alcohol consumption session was 0.73 ml/kg of 95% USP alcohol. Subjects then relaxed for 10 min (to allow time for alcohol absorption) and were then administered a third POMS. Another 5-min resting measure of (intoxicated) baseline heart rate followed (30 to 35 min post-onset of drinking). Subjects then engaged in video game play, a third and final time (35 to 55 min post-onset of drinking). Data described in the results below were derived from this (equalized-dose) session. The alcohol dosing and timing for postalcohol consumption physiological recordings were selected to ensure that individuals were tested while their BACs were approaching (ascending to) a legal level of intoxication (BAC > 0.08%), according to previous reports by Stewart et al.²² and Conrod et al.,²⁵ indicating that MFH versus FH-group differences in response to alcohol intoxication are dose- and limb of the BAC-dependent.

Subjects were then disconnected from the cardiovascular recording devices, fed, and debriefed. All subjects were paid \$5.00/hr of laboratory time and were allowed to leave once their BAC's reached 0.04 or less.

Autonomic Data Derivation. Polygraph data were scored manually by two people. Cardiovascular response measures were derived from game 1 (sober condition) and game 3 (equalized dose alcohol condition) only. The following measures were derived:

1. Resting baselines: (a) *sober resting baseline heart rate*: average heart rate during the most artifact free 2 min of the sober resting baseline heart-rate period; and (b) *alcohol-intoxicated resting baseline heart rate*: average heart rate during the most artifact free 2 min of the alcohol-intoxicated (equalized 1.0 ml dose) resting baseline heart rate period.
2. Resting baseline change: *alcohol-induced resting baseline heart rate change* (alcohol-intoxicated baseline heart rate minus sober resting baseline heart rate). This measure was derived to reflect sensitivity to the incentive rewarding properties of alcohol intoxication, as suggested by Pihl and Peterson¹⁵ and according to Fowles³⁰ three-arousal model.
3. Sober reactivity scores: (a) *sober heart rate change to video game* (sober game heart rate minus sober resting baseline heart rate); and (b) *sober heart rate change to reinforcement* (money or shock) (sober reinforcement heart rate minus sober resting baseline heart rate). Sober game heart rate was calculated by averaging heart rate during the last minute

of each 2-min game play period across the six sober game play periods. Sober reinforcement heart rate was calculated by averaging heart rate during the 7 sec after receipt of (sober period) reinforcement. Means for heart rate reactivity to receipt of shock and receipt of money did not significantly differ and therefore were considered together as a mean response to reinforcement. Such reactivity scores were proposed to reflect sensitivity to stress-induced arousal and were chosen to represent both active and passive coping responses to stressors that may be more ecologically valid than stress paradigms used with MFH subjects in the past (e.g., unavoidable shock paradigm).

4. Alcohol-intoxicated reactivity scores: (a) *alcohol-intoxicated heart rate change to video game* (alcohol-intoxicated game heart-rate minus alcohol-intoxicated resting baseline heart rate); and (b) *alcohol-intoxicated heart rate change to reinforcement* (money or shock) (alcohol-intoxicated reinforcement heart rate minus alcohol-intoxicated resting baseline heart rate). Alcohol-intoxicated game heart rate was calculated by averaging heart rate during the last minute of each 2-min game play period across the six alcohol-intoxicated game play periods. Alcohol-intoxicated reinforcement heart rate was calculated by averaging heart rate during the 7 sec after receipt of (alcohol intoxication period) reinforcement.
5. Reactivity change ("dampening") scores: (a) *video-game response dampening* (sober heart rate change to video game minus alcohol-intoxicated heart rate change to video game); and (b) *reinforcement response dampening* (sober heart rate change to reinforcement minus alcohol-intoxicated heart rate change to reinforcement). These dampening measures were derived to reflect sensitivity to negative reinforcement from alcohol ("SRD").

We used arithmetic change scores, in keeping with our previously published work, and because sober baseline heart rate measures did not differentiate between the groups.

POMS Change Score Derivation. Arithmetic change scores (change from baseline) were derived for each of the six POMS dimension, for each subject immediately after the recording period for alcohol-intoxicated resting baseline heart rate.

RESULTS

Comparison of MFH and FH- Men

MFH men were contrasted with their FH- counterparts with regard to age, years of education, personality (SSS total scores; EPQ subscales), alcoholic drinks/month, frequency of intoxication/year, brief MAST scores, cigarettes/day, quantity of illicit drug consumption/month, sober heart rate reactivity to video game, sober heart rate reactivity to reinforcement, dampening of heart rate reactivity to video game, dampening of heart rate reactivity to reinforcement, baseline heart rate change to alcohol, and change in mood after alcohol. Analysis of the data distribution for each variable indicated that drinks/month, frequency of intoxication/year, MAST scores, cigarettes/day, and quantity of illicit drug consumption/month were not normally distributed. In consequence, the drinking measures were square root-transformed and the smoking, drug consumption, and problem drinking symptoms measures were converted to categorical variables. Smokers were most efficiently subsumed into three categories: nonsmokers, <15 cigarettes/day, and >20 cigarettes (one pack)/day. Subjects either consumed drugs or did not; likewise, they either had drinking problems, or did not. One-way analyses of variances (ANOVAs) indicated that the risk groups differed only

with respect to years of education completed (MFH < FH-) drinks/month (MFH > FH-) and frequency of intoxication/year (trend MFH > FH-). χ^2 analyses indicated that a higher proportion of MFH males had problem drinking symptoms, smoked cigarettes, and used illicit drugs. Table 1 presents means and standard deviations for the various measures.

One-way ANOVAs were also performed on sober and alcohol-intoxicated and heart rate measures; means and standard deviations appear in Table 1. As indicated, the family history groups did not significantly differ from each other on any of the sober or alcohol-intoxicated heart rate measures. Multivariate ANOVA (MANOVA) was used to assess group differences in alcohol-induced changes in mood overall (vector of group means for all dimensions of mood) and changes on individual dimensions of mood. Evaluation of Box's test statistic indicated that the assumption of homogeneity of variance-covariance matrixes was satisfied. The multivariate effect was not significant [Hotelling's $T = 0.84$, $F(6,45) = 1.42$], indicating that the two groups did not differ in the degree to which alcohol affected their mood overall. However, subsequent one-way ANOVAs revealed that the MFH group self-reported less confusion (on the clearheaded-confused dimension) after alcohol consumption, compared with the FH- group, $F(1,51) = 5.15$, $p < 0.05$.

Analysis of the Relationship between Familial Risk and Self-Report Alcohol Use/Misuse

The simple correlations between measures of demographic status, personality, psychophysiological response, and alcohol use/misuse are presented in Table 2. Education, alcohol-induced resting baseline heart rate increase, and psychoticism seem most strongly associated with drinking behavior (self-report and laboratory). Baseline heart rate increase was mildly correlated with sober reactivity and moderately correlated with alcohol-induced dampening of such reactivity. Sober reactivity (game and reinforcement) was highly correlated with alcohol-induced dampening, indicating that alcohol reduced such reactivity to a level approaching 0, regardless of its initial magnitude (as we have demonstrated previously). Dampening, per se, correlated with a single drinking variable (frequency of intoxication per year).

Hierarchical multiple regression analyses were used to specifically examine the nature of the relationship between familial risk and alcohol use/misuse. Variables for entry into this analyses were selected using the "mediator/moderator" technique.¹⁶ "Mediating" variables account in whole or in part for the observed relationship between two other variables; "moderators," by contrast, modify the effect that one variable has on another. A third variable may be regarded as a potential mediator of the relationship between two others (one "independent" and one "dependent"), if three conditions are met: (1) there must be a

Table 1. Means (Standard Deviations/*n*) for MFH and FH— Groups: Demographic, Personality, Drug Consumption, and Response to Alcohol Intoxication Measures

	MFH	FH—	<i>F</i> or χ^2
% MFH	100 (30)	0 (0)	
Age	23.27 (3.24)	22.59 (2.32)	0.86
Years of education	13.83 (2.95)	15.76 (3.90)	8.62*
Personality variables			
SSS	25.40 (5.57)	23.86 (5.66)	1.11
Psychoticism	5.84 (3.01)	5.17 (2.19)	0.95
Neuroticism	9.88 (5.39)	10.28 (5.08)	0.08
Extraversion	15.67 (2.94)	14.93 (3.73)	0.72
Drinking variables			
Frequency of intoxication per year†	60.21 (57.08)	35.23 (48.24)	3.67, $p < 0.08$
Drinks/month†	47.42 (47.75)	20.81 (17.61)	6.30*, $p < 0.08$
Dose of alcohol consumed in laboratory	0.61 (0.43)	0.50 (0.36)	0.65
% Presence of problem drinking symptoms (<i>n</i>)	40.0 (12)	10.3 (7)	6.84*, ‡ $p < 0.08$
% Nonsmokers (<i>n</i>)	36.7 (11)	55.2 (16)	1.10‡
% Moderate smokers	16.7 (5)	31.0 (9)	1.30‡
% Heavy smokers (<i>n</i>)	46.7 (14)	13.8 (9)	5.20*, ‡
% Illicit drug use (<i>n</i>)	53.3 (16)	27.6 (8)	4.05*, ‡
Sober heart rate response			
Reactivity to reinforcement	4.56 (1.48)	3.37 (1.38)	0.34
Reactivity to game	10.75 (6.93)	13.08 (8.61)	1.31
Alcohol-induced heart rate response			
Baseline change	13.72 (11.65)	11.00 (7.40)	1.13
Dampening to reinforcement	2.90 (8.24)	2.33 (2.34)	0.10
Dampening to game	4.42 (8.56)	3.52 (7.67)	1.31
Alcohol-induced change in mood			
Composed-anxious	1.44 (6.04)	0.85 (8.46)	0.09
Elated-depressed	0.30 (4.47)	0.85 (5.17)	0.17
Energetic-tired	-0.74 (6.23)	-1.78 (7.39)	0.30
Confident-unsure	0.30 (6.02)	0.31 (6.39)	0.00
Agreeable-hostile	0.44 (3.85)	0.76 (3.83)	0.09
Clearheaded-confused	-2.56 (4.77)	-6.00 (6.21)	5.15*

p values are as follows: * $p < 0.05$; ** $p < 0.01$.

† Raw data (analyses performed on square root-transformed equivalents).

‡ χ^2 analysis performed on categorical data; values in parentheses are standard deviation from the mean or for χ^2 analyses (*n* of group members).

Table 2. Correlations between Demographic, Personality, Drinking, and Response to Alcohol Measures

	2	3	4	5	6	7	8	9	10	11	12	13	14
1. HRR to alcohol	0.30*	0.38**	0.38**	0.48***	0.08	-0.11	0.30*	-0.02	0.32*	0.40**	0.33**	-0.18	-0.15
2. HR reactivity to game play		0.72**	0.19	0.16	0.03	-0.08	0.13	0.00	0.24	0.32*	-0.10	-0.13	0.05
3. HR dampening to game play			0.17	0.26*	0.02	-0.16	-0.06	-0.12	0.23	0.24	-0.03	-0.08	-0.05
4. HR reactivity to reinforcement				0.82***	0.34**	-0.04	0.16	0.00	0.17	0.24	0.08	-0.27*	-0.15
5. HR dampening to reinforcement					0.32*	-0.11	0.23	-0.12	0.18	0.31*	0.19	-0.12	-0.18
6. Psychoticism						0.11	-0.04	0.35**	0.53***	0.44***	0.21*	-0.12	-0.31*
7. Extraversion							-0.27	0.14	0.14	0.07	0.10	-0.00	0.03
8. Neuroticism								-0.06	-0.08	0.11	-0.12	0.07	0.05
9. Sensation Seeking									0.20	0.13	0.07	0.12	0.01
10. Drinks/month										0.81***	0.29*	-0.14	-0.48***
11. Frequency of intoxication/year											0.31*	-0.22	-0.44***
12. Dose of alcohol consumed in laboratory												0.30*	-0.32*
13. Age													0.23
14. Education													

p values are as follows: * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

HRR, heart rate response; HR, heart rate.

significant relationship between the two measures in question; (2) the potential mediator must be correlated with the independent variable; and (3) the potential mediator must be correlated with the dependent variable.¹⁶ According to the information presented in Table 1, condition 1 was met for the variables drinks/month, frequency of intoxication/

year, and presence/absence of problem drinking symptoms (considered in relationship to family history), but not for laboratory alcohol consumption. Our analyses focused, in consequence, on identification of mediators and moderators of the family history/self-report alcohol use relationship.

Table 3. Assessment of Potential Mediators and Moderators of Alcoholic Family History on Drinking Behavior: Hierarchical Multiple Regression Predicting Drinking Measures from Family History, Education, Psychoticism, Indices of Response to Alcohol Intoxication, and Relevant Interactions

	Dependent measures					
	Quantity of alcohol/month		Frequency of intoxication/year		Problem drinking symptoms	
	β	R^2 -partial	β	R^2 -partial	β	R^2
FH	1.80	0.10*	1.86	0.06	1.75	0.10**
Mediating variable						
Education						
FH	0.93	0.03	0.77	0.01	1.48	0.06*
Education	-0.45	0.17***	-0.57	0.15**	-0.15	0.025
R^2 total		0.25***		0.20**		0.14
Psychoticism						
FH	0.88	0.04	0.72	0.01	1.60	0.06*
Education	-0.31	0.10*	-0.42	0.09*	-0.07	0.01
Psychoticism	0.46	0.22***	0.48	0.13**	0.51	0.11**
R^2 total		0.41***		0.30***		0.25***
Predicting variables: HR response to alcohol						
Step 1: Main effects						
FH	0.76	0.03	0.48	0.01	1.50	0.06
Education	-0.29	0.10*	-0.38	0.08*	-0.05	0.00
Psychoticism	0.45	0.23***	0.47	0.14**	0.53	0.11**
HRR to alcohol	0.07	0.09*	0.13	0.15**	0.04	0.02
R^2 total		0.47***		0.40***		0.27**
Step 2: Interactions						
Psychoticism \times HRR	0.02	0.05	0.00	0.00	-0.02	0.02
R^2 total		0.49***		0.40***		0.28***

p values are as follows: * $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$.

FH, family history; HR, heart rate; HRR, heart rate response.

Education was considered first, as a potential mediator, as it was clearly related to family history and alcohol consumption. Separate hierarchical regression analyses were conducted for each drinking outcome variable, as recommended by Baron and Kenny.¹⁶ The dependent measures (drinks/month, frequency of intoxication/year, and presence/absence of problem drinking symptoms) were first regressed upon the dummy-coded family history variable. Analyses, detailed in Table 3, indicated that family history accounted for 10% of the variance in drinks/month ($p < 0.02$), 6% of the variance in frequency of intoxication/year ($p < 0.06$), and 10% of the variance in presence/absence of problem drinking symptoms ($p < 0.01$). Education—the presumed mediator—was then entered into the regression analyses. Although total variance in drinks/month accounted for increased to 25% ($p < 0.001$), the effect of family history was reduced to nonsignificance ($\beta = 0.93$, $R^2 = 0.03$, $p < 0.05$). Similarly, education mediated the relationship between family history and frequency of intoxication/year, partially accounting for 15% of the variance ($\beta = -0.57$, $R^2 = 0.15$, $p < 0.003$), thus reducing the effect of family history to nonsignificance ($\beta = 0.77$; $R^2 = 0.01$; $p > 0.05$). Hierarchical logistic regression techniques were used for presence/absence of problem drinking symptoms, which was categorical in nature. Education was *not* a mediator of the family history/problem drinking relationship (family history/problem drinking, excluding education: $\beta = 1.75$; $R^2 = 0.10$; $\chi^2 = 6.02$; $p < 0.01$; family history/problem drink-

ing, including education: $\beta = 1.48$; $R^2 = 0.06$; $\chi^2 = 3.89$; $p < 0.05$).

We then examined the potentially mediating effect of psychoticism—highly correlated with education and self-report drinking—on the relationship between education and drinking behavior, in three additional regression analyses. Psychoticism indeed seemed to reduce the amount of variance in the family history/self-report drinking behavior relationship accounted for by education: 23% reduced to 10% and 15% to 9% for drinks/month and frequency of intoxication/year, respectively. Psychoticism was directly implicated in presence/absence of problem drinking, because it accounted for a significant portion of variance in problem drinking status over and above family history. In interaction with family history, however, no additional variance could be accounted for. These results are also portrayed in Table 3.

Resting baseline heart rate response to alcohol—highly correlated with self-report drinking measures—was then considered, according to Baron and Kenny's¹⁶ guidelines, as a potential moderator of the relationship between family history, education, and psychoticism and self-report drinking behavior. This meant that its main effects were examined, in addition to those of family history, education, and psychoticism; and that its interactions with the latter three variables were then assessed. The main effects of resting baseline heart rate response to alcohol were significant and substantial, for drinks/month and frequency of intoxication/

Table 4. Verification of Reliability of Regression Model Derived Based on Analysis on Self-Report Drinking Measures: Assessment of Correspondence Between Voluntary Alcohol Consumption in the Laboratory and Family History, Education, Psychoticism, Indices of Response to Alcohol Intoxication and Relevant Interactions

	Quantity of alcohol consumed in laboratory	
	β	R^2 -partial
Main effects		
FH	-0.03	0.00
Education	-0.04	0.05*
Psychoticism	0.02	0.03
HR response to alcohol	0.01	0.10**
R^2 total		0.21**
Interaction		
Psychoticism \times HRR	0.00	0.01
R^2 total		0.21**

p values are as follows: * $p < 0.05$; ** $p < 0.01$.

HR, heart rate; HRR, heart rate response.

year accounted for; interestingly, the original contributions of family history, education, and psychoticism did not change. The only interaction that seemed significant, by contrast, was that obtained with psychoticism/resting baseline heart rate response to alcohol and drinks/month (improvement in fit $R^2 = 0.02$). A different pattern of results emerged for presence/absence of problem drinking symptoms. Neither the main effects of resting baseline heart rate response to alcohol nor any of its two-way interactions improved "prediction" of problem drinking category, above that provided by family history and psychoticism. No other variables were assessed for mediator/moderator status, because they did not seem to correlate strongly or consistently enough with the drinking behavior measures.

Correlates of Alcohol Consumption in the Laboratory

The relatively large number of variables considered in the previous (exploratory) analyses increased the probability of capitalizing on chance and heightening the proportion of the variance accounted for by our models. To help control for this possibility, we assessed the reliability of the model by applying to the prediction of lab alcohol consumption the subset of variables that our previous regressions identified as of potential utility. Main effects for family history, education, psychoticism and heart rate response to alcohol were first tested; results appear in Table 4. Only the education and heart rate response to alcohol variables significantly contributed to the prediction of laboratory alcohol consumption ($R^2 = 0.05$; $p < 0.05$, $R^2 = 0.10$; $p < 0.01$, respectively). As a second step, the interaction variable (heart rate response to alcohol \times psychoticism) was included in the model and did not account for any additional variance in drinking behavior.

Cluster Analysis of Correlates of Drinking Behavior

We performed an exploratory cluster analysis [using the K-means algorithm (tentative relocation method) de-

scribed by Hartigan⁵²] to gain another perspective on the nature of the interrelationships between family history, demographic status, personality, psychophysiological response, and alcohol use/misuse. Cluster analysis allows the objective, statistical sorting of heterogeneous subjects into relatively homogenous groups; the K-means approach adopted is far less sensitive to deviant subjects and outliers than other methods.⁵² Initial analyses restricted to the four correlates of drinking behavior (i.e., family history, education, psychoticism, and heart rate response to alcohol intoxication) indicated the suitability of a three- or four-cluster model; the three-cluster alternative was deemed preferable, because cluster four of the four-cluster solution consisted of only three subjects.

Neither drinking nor drug-related behavioral indices (consumption rates or problems) were included in the cluster analysis; nor were data regarding mood. We chose to exclude these variables, to allow for the possibility of post-hoc testing of the utility of our derived clusters. We hypothesized that the different clusters, derived from analysis of various vulnerability factors, would differ in terms of these excluded variables. Table 5 contains the results of the statistical analyses for drug and alcohol-related behavior (one-way ANOVA or χ^2 , where appropriate), as well as relevant cluster means, standard deviations, and percentages (where applicable).

We also performed a MANOVA to assess cluster-group differences in alcohol-induced changes in global mood, and one-way ANOVAs for the various mood subscales. Evaluation of Box's test statistic indicated that the assumption of homogeneity of variance-covariance matrixes was satisfied. The multivariate effect was significant [Hotelling's $T = 0.53$, $F(12,88) = 2.73$, $p < 0.01$], indicating that the three groups differed in the degree to which alcohol affected their global mood. Subsequent one-way ANOVAs revealed that the three cluster groups differed significantly on three dimensions of mood. In addition to self-reporting better mood overall after alcohol consumption, cluster 2 self-reported feeling more elated, more confident, and more clear-headed, compared with the other two cluster groups. Table 5 also presents mean change scores, standard deviations, and F -ratios for the comparison of the cluster groups on the six dimensions of mood measured by the POMS. Table 6 presents a summary of the distinguishing features of the three clusters.

DISCUSSION

Sher and his colleagues^{10,11,14,17,53} have suggested that deviance-proneness/school failure, motivation to self-medicate negative mood states, and sensitivity to the reinforcing effects of alcohol predispose to alcoholism. Hill⁵ concluded, similarly, that genetic predisposition to early-onset alcoholism is mediated via two separate pathways: through antisocial traits and through strong genetic loading for alcoholism. She suggested the existence of a "type III"

Table 5. Means (Standard Deviations) for Three Clusters of Young Male Drinkers: Demographic, Personality, Drug Consumption, and Response to Alcohol Intoxication Measures

	Drinker cluster			F or χ^2
	1	2	3	
Variables used to derive clusters				
% MFH	23.00 (8)	75.00 (6)	100.0 (16)	28.31** \ddagger
Years of education	16.86 (1.88)	12.38 (2.83)	12.75 (1.84)	23.23** 1 > 2,3
Psychoticism	4.60 (2.32)	6.21 (1.68)	7.17 (2.89)	6.55** 3 > 1
Resting HR change	9.79 (6.04)	30.89 (10.32)	8.81 (5.26)	37.09** 2 > 1,3
Variables used to verify clusters				
Substance use variables				
Frequency of intoxication†	29.54 (45.18)	72.13 (46.59)	75.88 (60.68)	7.75** 2,3 > 1
Drinks/month†	17.34 (17.15)	59.00 (44.17)	59.25 (49.44)	13.16** 2,3 > 1
Ethanol dose consumed in laboratory	0.50 (0.33)	1.02 (0.35)	0.60 (0.43)	6.73** 2 > 1,3
Presence of problem drinking symptoms	8.6 (3)	37.5 (3)	56.3 (9)	13.88** \ddagger
% Nonsmokers (n)	60.0 (21)	25.0 (2)	25.0 (4)	3.80* \ddagger
Moderate smokers	25.7 (9)	37.5 (3)	12.5 (3)	1.50†
% Heavy smokers (n)	14.3 (5)	37.5 (3)	62.5 (10)	8.50** \ddagger
% Illicit drug use (n)	25.7 (9)	50.0 (4)	68.8 (11)	8.80** \ddagger
Alcohol-induced change in mood				
Composed-anxious	-0.26 (7.73)	4.88 (6.98)	2.14 (5.71)	1.82
Elated-depressed	-0.16 (4.15)	5.50 (4.90)	-0.64 (4.60)	6.04** 2 > 1,3
Energetic-tired	-2.71 (7.20)	2.50 (5.85)	0.00 (5.62)	2.11
Confident-unsure	-1.03 (6.14)	4.38 (5.61)	0.93 (5.69)	2.73* 2 > 1,3
Agreeable-hostile	0.23 (3.68)	1.38 (4.31)	0.93 (3.97)	0.35
Clearheaded-confused	-6.61 (5.33)	-1.62 (4.72)	-0.50 (4.64)	8.25** 2 > 1,3
Sober HR reactivity and dampening				
Reactivity to reinforcement	3.50 (7.00)	6.44 (5.41)	3.98 (9.97)	0.49
Reactivity to game	12.83 (8.89)	11.94 (5.94)	9.84 (5.87)	0.80
Dampening to reinforcement	1.87 (7.66)	6.65 (6.31)	2.41 (8.91)	1.26
Dampening to game	5.58 (8.61)	7.03 (7.16)	3.33 (7.36)	0.66
Personality variables				
SSS	24.33 (5.31)	23.75 (7.40)	26.00 (5.45)	0.66
Neuroticism	9.82 (5.50)	10.68 (6.01)	10.31 (4.35)	0.10
Extraversion	15.11 (3.53)	14.52 (3.89)	16.13 (2.60)	0.75

HR, heart rate.

p values are as follows: * $p < 0.05$; ** $p < 0.01$.

† Raw data (analyses performed on square root-transformed equivalents).

 \ddagger χ^2 analysis performed on categorical data; values in parentheses are standard deviation from the mean or for χ^2 analyses (n of group members).

alcoholism, characterized by "cardiovascular responsiveness" (opposed to the anxious type I and the antisocial type II). Harden and Pihl⁵⁴ have recently demonstrated that primary school-aged sons of MFH alcoholics differed from sons of FH—nonalcoholics in two respects: (1) in terms of performance on a cognitive test battery assessing specific cognitive functions; and (2) in terms of cardiovascular reactivity to a nonaversive stressor. Decrements in cognitive function were associated with the presence of conduct problems; however, cardiovascular reactivity and cognitive performance/conduct disorder were not associated.

The present study demonstrated, similarly, that drinking among relatively heavy social drinking young nonalcoholic males—some of whom are at heightened familial risk for the development of alcoholism—is partially mediated by disinhibited personality (affecting educational attainment, associated conceptually with antisocial/disagreeable/unconscientious/conduct-disordered/hyperactive behavior) and by differential sensitivity to the putatively positive reinforcing effects of alcohol (as assessed by alcohol-induced car-

diac acceleration). The former "risk marker" seems particularly associated with the presence/absence of problem drinking symptoms, in concert with familial risk; the latter "risk marker" seems more powerfully associated with self-report and laboratory measures of alcohol consumption.

Psychoticism/Education

Family history of alcoholism increases the likelihood that a young male will drink and drink excessively. However, level of academic achievement seems to mediate this relation. Disinhibited personality, in turn, accounted for half of the relationship between education and drinking, and was particularly related to the presence of problem-drinking symptoms. Cluster analyses clarified these findings. Clusters 2 and 3, who drank heavily, could be distinguished from cluster 1 primarily in terms of education, but could not be distinguished from one another in terms of quantity/frequency of drinking and frequency of drinking to intoxication. However, individuals in cluster 3 reported more dis-

Table 6. Descriptors of Three Clusters of Young Male Drinkers

Cluster		
1 (n = 35)	2 (n = 8)	3 (n = 16)
77% FH-	75% MFH	100% MFH
High academic achievement	Low academic achievement	Low academic achievement
	Sensitivity to alcohol reinforcement	Disinhibited personality traits
Mood dampening when drunk	Enhanced mood when drunk	No change in mood when drunk
Drunk 2-3 times/month	Drunk 1-2 times/week	Drunk 1-2 times/week
Mild dose of alcohol consumed in laboratory	Intoxicating dose of alcohol consumed in laboratory	Mild dose of alcohol consumed in laboratory
91% reporting no problem drinking symptoms	30% reporting problem drinking symptoms	60% reporting problem drinking symptoms
60% nonsmokers	75% smokers (38% heavy)	75% smokers (63% heavy)
26% illicit drug users	50% illicit drug users	69% illicit drug users

turbances to social, occupational, family, or personal functioning due to alcohol consumption, and were more likely to engage in illicit drug use relative to the other groups. These latter characteristics of such heavy drinkers seem related to their relative lack of behavioral constraint or disinhibited personality profile.

The relationship between poor academic achievement, disinhibited personality, and drinking-related problems may reflect the influence of a mildly abnormal cognitive profile across several domains of general function, including academic achievement, personality, and use of alcohol and other drugs. We did not assess this possibility directly; however, disinhibited personality has been generally associated with poor planning abilities and deficits in information processing.^{38,54-56} Individuals with a personality style reflecting disinhibition and lack of cognitive structure also demonstrate impulsive and under-controlled behavior associated with alcohol consumption.⁵⁷ Furthermore, specific cognitive deficits that have been associated with school failure,⁵⁴ aggression/behavioral undercontrol,⁵⁸ and sensitivity to alcohol's effects on provoked aggression⁵⁸ have been implicated in the familial predisposition to alcoholism.^{59,60} The early-onset alcohol problems characteristic of cluster 3 seem a likely consequence of the manner in which these "disinhibited" individuals behave—or misbehave—while sober, and while drinking, rather than a consequence of their increased susceptibility to ethanol reinforcement.

Sensitivity to Ethanol Reinforcement

Sensitivity to alcohol reinforcement does not seem to mediate the relationship between family risk status and drinking, but is a robust correlate of quantity of alcohol consumption (particularly with regard to frequency of drinking to intoxication). Individuals in cluster 2 (75% MFH; 13.3% of the total sample), characterized by poor academic achievement and heavy drinking in the absence of trait psychoticism, demonstrated marked physiological and

subjective sensitivity to alcohol. We suggest that this small portion of the sample is susceptible to alcoholism primarily as a consequence of this increased physiological and subjective sensitivity, and that this sensitivity renders alcohol intoxication more positively reinforcing. In keeping with this interpretation, the cluster analysis indicated that individuals who were cardiovascularly reactive to alcohol consumed, on average, a moderately intoxicating dose of alcohol when given the opportunity to do so in the lab (the mean dose consumed by cluster 2 was equivalent to 0.45 ml/kg of 95% USP ethanol). We know from previous and concurrent work that such reactivity is associated with enhanced mood postethanol consumption (Peterson JB, Conrad PJ, Pihl RO: Heart rate increase during the ascending limb of the blood alcohol curve: An index of incentive reward? Evidence from two studies, unpublished manuscript). (as well as with other features logically associated with increased activity in the behavioral activation system (BAS)).²⁹⁻³³ The combination of these results adds further credence to the notion that some individuals are predisposed to drink excessively due to a vulnerability to the (positively) reinforcing properties of alcohol.

Comparison of MFH/FH—Subjects

The characteristics identified as predictors of alcohol-related behavior did not distinguish between the groups of young men when they were separated solely based on family history of alcoholism. Cluster analyses revealed that the MFH males were *homogeneous* in their drinking practices, but were a *heterogeneous* group, with respect to the mechanisms influencing their tendency to use and misuse alcohol. This finding is in line with Sher's^{14,15} position, and the results reported by Hill,⁵ indicating that at least two vulnerability mechanisms may be at play in the genetic predisposition to alcoholism. In the present analysis, not all individuals with a family history of alcoholism manifested the proposed vulnerability characteristics. 53.3% of the 30

MFH SOMAs were characterized by impulsive personality; 20.0% were characterized by obvious sensitivity to alcohol reinforcement; 26.6%, by contrast, were not characterized by either of the two vulnerability profiles assessed in this study. Furthermore, a small number of FH- men demonstrated a sensitivity to alcohol reinforcement and related tendency to drink heavily. The pattern of findings seem reasonable, given that only a minority of children of alcoholics become alcohol-dependent.^{62,63}

Nonetheless, this lack of group differences, with regard to sober heart rate reactivity, alcohol-induced dampening, and alcohol-induced baseline heart rate increase stands in contrast to our earlier findings.¹⁸⁻²³ The present research design differed from previous studies in several respects. First, subjects were recruited through advertisements in arts and entertainment newspapers and were only excluded if they self-reported a certain number problem drinking symptoms (brief MAST ≥ 10). This recruitment procedure resulted in much heavier drinking MFH and FH- samples, compared with those of previous studies. These results highlight the importance of recruiting from outside a university population to investigate the predisposing factors of alcoholism. Furthermore, alcohol was administered in two separate drinking sessions, the first of which involved self-administered dosing and the second involved administration of a dose that was dependent on the first. Although MFH/FH- group differences were not yielded for dose of alcohol consumed in the first drinking session, volume of alcohol consumed in this first drinking session was highly related to cardiac response to alcohol intoxication resulting from the second dose and might explain why only a percentage of MFH individuals demonstrated sensitivity to alcohol reinforcement. An important finding was that, even though men of cluster 2 were administered lower doses of alcohol in the second drinking session, they remained highly cardiovascularly reactive to alcohol intoxication. These findings demonstrate the robustness of sensitivity to alcohol effects in this group of individuals.

A discussion of the relative lack of findings with respect to a relationship between SRD and family history or drinking behavior is warranted, because it is also at odds with findings from previous studies.¹³⁻²³ SRD to reinforcement did correlate mildly to moderately with the self-report drinking variables. However, it correlated less strongly, compared with the resting heart rate response and did not correlate with the laboratory measure of drinking behavior. Although not reported herein, there was a strong correlation between SRD immediately after the consumption of the self-administered dose of alcohol and the actual dose consumed. Therefore, the double dosing procedure is potentially responsible for the failure to find a relationship between SRD after the second drinking session and amount of alcohol consumed in the first drinking session.

It is important to note, nonetheless, that despite their similar personality and psychophysiological profiles, and the equivalent recruitment procedures for each group, the

MFH and FH- males contrasted on several important features. Having completed fewer years of education and self-reporting more numerous problem drinking symptoms, heavier smoking, drinking and drug use patterns, and more frequent alcohol intoxication, MFH males demonstrated greater risk factors for alcoholism than their age-matched counterparts. These group differences have been reported previously^{14,21} and provide an opportunity to investigate the mechanisms influencing elevated alcohol consumption patterns and the potential for the development of alcoholism.

The results of the present study, in line with results from previous investigations,^{5-8,53,54} advance the unfolding of the familial predisposition to alcoholism into discrete and identifiable vulnerability mechanisms. In the present investigation, disinhibited personality and cardiac response to alcohol intoxication accounted for independent, yet sizable, portions of variance in drinking behavior (assessed in a number of ways). Whereas sensitivity to alcohol intoxication might indicate vulnerability within an incentive reward system,⁵⁵ the contribution of disinhibited personality to elevated drinking patterns may be through its influence on lifestyle and the associated availability of alcohol and illicit drugs.¹⁴ Perhaps individuals high in disinhibition or those who are "behaviorally unconstrained" have difficulty regulating the consequences of their drinking or general substance use, even given average levels of sensitivity to ethanol (or drug) reinforcement. Elevated sensitivity to the putatively incentive-rewarding properties of alcohol,^{15,55} by contrast, proved to be highly predictive of elevated alcohol consumption in the laboratory, and may be the mechanism responsible for dosage of alcohol consumed during a single drinking occasion in the absence of such a disinhibited personality profile.

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