Behavioral Inhibition in Children From Families at High Risk for Developing Alcoholism

SHIRLEY Y. HILL, PH.D., LISA LOWERS, M.A., JEANNETTE LOCKE, M.B.A., NANCY SNIDMAN, PH.D., AND JEROME KAGAN, PH.D.

ABSTRACT

Objective: To determine whether children at risk for the development of adult alcohol dependence would show greater "behavioral inhibition" to the unfamiliar, an early childhood temperament characteristic. **Method:** One hundred peer play evaluations were conducted blindly with preschool children from families selected to be at high or low risk for developing alcohol dependence. Each child was paired with different children (same-sex pairs) in independent sessions to determine the stability of the behavioral response. **Results:** High-risk children spent significantly more time staring at the other child during the peer play session while refraining from engaging in play, and significantly less time speaking to the other child. Significantly more time was spent proximal to the parent, but only on the first peer play session. These behaviors have been shown to be indicators of behavioral inhibition. **Conclusions:** These findings suggest that the presence of behavioral inhibition to the unfamiliar in childhood may be a risk factor for later development of alcohol dependence. While there is abundant evidence that childhood externalizing behaviors are risk factors for later development of substance dependence, the present results suggest that internalizing behaviors may be a pathway as well. *J. Am. Acad. Child Adolesc. Psychiatry,* 1999, 38(4):410–420. **Key Words:** alcoholism risk, behavioral inhibition, peer play, temperament.

Twin, adoption, and family studies provide considerable evidence that alcoholism is heritable (see Hill, 1994, for review). The risk of developing alcohol dependence by adulthood is higher for both female and male offspring of alcoholic parents (4- to 6-fold higher) than it is for offspring of nonalcoholics (Bohman et al., 1981; Cloninger et al., 1981; Goodwin et al., 1973). Several research groups have proposed that there are temperament predispositions to substance use (Hill et al., 1986, 1990; Tarter et al., 1990; Watson and Clark, 1993). Among the temperament dimensions most often discussed as being related to substance use in adolescence, behavioral undercontrol and negative affectivity appear most prominent (see Stice et al., 1998, for review).

The relationship between behavioral undercontrol in adolescence and later development of alcoholism was emphasized in 3 early longitudinal studies (Jones, 1968, 1971; McCord and McCord, 1962; Robins, 1966). A more recent longitudinal study (Mannuzza et al., 1993) found that the presence of a number of externalizing disorders in childhood, including conduct disorder and attention-deficit hyperactivity disorder, is predictive of young adult alcohol problems. Similarly, Caspi et al. (1996) found that boys who were classified as undercontrolled at age 3 were 2.7 times more likely to have a diagnosis of alcohol dependence at age 21.

However, less is known about internalizing disorders as a pathway to adolescent and/or adult substance abuse. Emotional distress including depression and anxiety has been related to adolescent substance use (Newcomb and Bentler, 1989). Also, internalizing symptoms have been reported to be in excess of population rates among high-risk offspring of female alcoholics (Hill and Muka, 1996) and among community-based children of alcoholics (Colder and Chassin, 1993). With possible links between internalizing symptoms and alcohol use in adolescence,

Accepted September 23, 1998.

Dr. Hill, Ms. Lowers, and Ms. Locke are with the Department of Psychiatry, University of Pittsburgh Medical Center, Western Psychiatric Institute and Clinic, Pittsburgh. Drs. Snidman and Kagan are with the Department of Psychology and Social Relations, Harvard University, Cambridge, MA.

This study was supported by grants from National Institute on Alcohol Abuse and Alcoholism (AA05909 and AA08082). The support of numerous individuals involved in the research program, especially Shawn Gronlund, Kathleen Walsh, Ember Blauser, and Tim Pitts, is gratefully acknowledged.

Reprint requests to Dr. Hill, Western Psychiatric Institute and Clinic, 3811 O'Hara Street, Pittsburgh, PA 15213.

the question arises as to how early in development temperament differences can be observed in high-risk children (children who collectively have a higher statistical risk than controls due to greater genetic loading).

Temperament refers to the constitutionally based, inherited predisposition to respond to situations and persons in particular ways. This individual difference in reactivity and self-regulation has captured the interest of a number of investigators over the past 20 years (Buss and Plomin, 1975; Goldsmith and Gottesman, 1981; Thomas et al., 1970). Temperament characteristics, thought to provide the foundation for adult personality, are present early in life, stable over time, and strongly influenced by biological factors (Thomas and Chess, 1977). One temperament dimension that has been extensively studied in both children and adults is the initial reaction to unfamiliar people or situations. In adults, this temperament style includes variation in the tendency to withdraw from the novel or variation in the disposition toward either introversion or extraversion. Children described as shy or inhibited typically withdraw when confronted by novelty, stopping their ongoing behavior, ceasing vocalization, and clinging to a familiar figure.

Thomas and Chess (1977), in their landmark study, found that infants with a tendency to withdraw from new situations and people were at risk for developing avoidant or overanxious disorders in childhood. Thus, withdrawal from novelty appears to be an enduring temperamental dimension from infancy through childhood (Chess and Thomas, 1977, 1984; Kagan, 1994).

The Harvard Child/Infant Study, one of the largest ongoing research efforts devoted to the study of behavioral inhibition in infants and preschool children, has tracked the continuity of this temperament across development (Kagan et al., 1984; Reznick et al., 1986). From this effort it has been determined that approximately 10% to 15% of U.S. white children are born with a temperamental predisposition to be irritable, shy, or introverted. This determination was based on scored behaviors from laboratory observations, enabling the investigators to determine a consistent pattern of approach or withdrawal using familiar or unfamiliar persons or toys as stimuli. Among the signs of inhibition observed were a long latency to interact, a tendency to remain proximal to their parent when facing new situations, and a tendency to stare at other preschool children without speaking rather than entering into play with the other children. (Note that these children spoke freely with their parent, without hesitation and with clarity.)

While the links between early childhood behavior and adult adjustment have not been firmly established with respect to alcohol dependence, we do know that children of alcoholics are at higher risk for a number of childhood psychopathologies (Hill and Hruska, 1992; Hill and Muka, 1996; Reich et al., 1993). Also, we know that internalizing symptoms appear to predispose adolescents to substance dependence (Clarkin et al., 1984; Kellam et al., 1983). To our knowledge, however, preschool children of alcoholics have not previously been studied in a peer play situation designed specifically to elicit behavioral inhibition. Such a study would provide empirical evidence about possible continuities between behavioral characteristics in early childhood and elevated risk for alcoholism in adulthood. On the basis of the increased risk of internalizing disorders seen in children of alcoholics compared with controls, we predicted that children of alcoholics might show greater behavioral inhibition than children of controls.

METHOD

Subjects

Two groups of children between the ages of 4 and 6 years were studied: a high-risk group consisting of 18 children (11 males and 7 females) and a low-risk group of 18 children (13 males and 5 females). All resided in the Pittsburgh metropolitan area. Within the constraints of availability for testing, an attempt was made to match eligible children from the experimental and control groups for age, socioeconomic status, and gender (Table 1). All participating parents signed informed consent forms.

A unique feature of this study was that unlike previous studies of 2-child play behavior, which based conclusions on a single pairing of children (Kagan et al., 1984; Reznick et al., 1986; Rosenbaum et al., 1988), each child was paired with up to 3 children, 1 per session. These sessions were separated by no less than a week and no more than 2 months. This design enabled testing for the stability of the observed behavior across sessions (e.g., a temperamental trait rather than simply a response to a particular child or type of child). As a result of this repeated-measures design, 100 evaluation sessions were conducted (36 children participated in the first session, 34 completed 2 sessions, and 30 completed 3 sessions). Because of the highly selected nature of the families available for study, multiple members of some families were included.

The high-risk children were the offspring of parents who came from multigenerational, high-density alcoholism pedigrees. These target pedigrees had been developed as part of an ongoing family study. Pedigrees were included in the larger study only if there were 2 alcoholic brothers available for in-person psychiatric interviews and laboratory testing. Thus, one parent came from the "target" pedigree and met the following inclusion criteria: (1) member of a pedigree with a high density of alcoholism which included 2 alcohol-dependent probands, at least 1 of whom had been in treatment at the time of selection for study; (2) absence of recurrent depression, schizophre-

TABLE 1
Demographic Characteristics of High- and Low-Risk Children Evaluated Over 100 Sessions

	High-Risk (n = 18)	Low-Risk (n = 18)
Age (months)	65.67 ± 9.7	58.28 ± 8.6
Gender		
Male	11	13
Female	7	5
No. of 1st- and 2nd-degree relatives alcoholic	4.78 ± 1.8	0.11 ± 0.3
Mother's age (years)	33.78 ± 4.3	35.06 ± 2.7
Father's age (years)	35.17 ± 5.3	36.50 ± 2.9
No. of children in family[a]	2.89 ± 0.8	2.50 ± 1.2
No. (%) of children in professional, semiprofessional, and skilled occupation homes[b]	16 (88.9)	18 (100)

Note: Values represent mean ± SD or no. (%).

[a] Includes 9 half- or adopted siblings.

[b] A 2 × 2 χ^2 analysis compared the number of children whose parents were determined to be in these 3 categories with those falling in the semiskilled and unskilled categories (χ^2 = 2.12, p not significant). Where both parents were employed, an average score was obtained. Calculations, using a combination of education and occupational scores, were based on Hollingshead's Four Factor Index (1975).

nia, and primary drug dependence in the proband pair or first-degree relatives. The psychiatric background of the marrying-in parent was free to vary. Although the larger study contains nonwhite families, only white families were included in the subsample to increase the homogeneity of the sample. Kagan (1994) noted that behavioral inhibition does appear to vary with race, with those of Asian background having higher rates of behavioral inhibition.

Control children were selected on the basis of one parent's being a member of a pedigree with a low density of alcoholism that had been screened for both alcoholism and absence of other Axis I psychopathology (the same psychopathology excluded from the alcoholic pedigrees). In most cases, the other parent also had minimal family history of alcoholism (Table 2).

The parents were recruited by the project coordinator, who was not involved in the laboratory-based assessment or scoring of the behavioral inhibition measures. Other research staff conducted the testing and coding and were blind to the risk status of the child's family. The 30-minute peer play session involved 2 same-sex children, randomized for affected status of their parents. The procedures were designed to obtain an index of each child's behavior when confronted with having to play with an unfamiliar child. Prior to their arrival, parents had been instructed to meet the research staff at a different location so that the children would not meet each other before testing. The children were introduced immediately before they entered the testing room, at which time on-line coding began.

Diagnostic Assessments

At least one parent of each child (N = 37) had participated in the larger family study. They had been evaluated in person by a trained, master's-level clinician, using portions of the National Institute of Mental Health's Diagnostic Interview Schedule. All structured interviews were followed by a second clinician-administered interview (M.D. psychiatrist or Ph.D. psychologist), and a consensus diagnosis was reached as previously described (Hill, 1992). Some parents, usually those marrying into the pedigree (N = 27), were administered an abbreviated lifetime screening interview, based on portions of the Diagnostic Interview Schedule, to obtain Axis I diagnostic information (alcoholism, major depressive disorder, anxiety disorders, schizophrenia, and drug dependence) at the time they accompanied their child to the laboratory.

Therefore, in-person diagnostic information was available for a total of 64 parents. Eight parents were diagnosed by family history only. In addition, on the day of the child's assessment, a spouse family history which sought current information on the parent not participating in the play session and his/her first-degree relatives was completed. This enabled us to determine whether the parent not accompanying the child to the play session (usually the father) was in remission or currently meeting criteria for alcohol dependence (Table 2).

TABLE 2
Parental Diagnoses

	High-Risk				Low-Risk			
	Lifetime[a]		Current[b]		Lifetime		Current	
	Mother (n = 18)	Father (n = 18)	Mother (n = 18)	Father (n = 16)	Mother (n = 18)	Father (n = 18)	Mother (n = 18)	Father (n = 18)
Alcohol dependence	7 (38.9)	11 (61.1)	1 (5.6)	11 (68.8)	0	2 (11.1)[c]	0	1 (5.6)[c]
Anxiety disorders	0	0	0	0	0	3 (16.7)	0	3 (16.7)
Major depressive disorder	0	2 (11.1)	0	0	0	0	0	0
Drug abuse/dependence	1 (5.6)	4 (22.2)	0	0	1 (5.6)	2 (11.1)	0	1 (5.6)

Note: Values represent no. (%).

[a] Lifetime diagnosis (Research Diagnostic Criteria and *DSM-III* criteria).

[b] Current diagnosis (Research Diagnostic Criteria and *DSM-III* criteria) of the parent at the time the child was assessed. There was no information available for 2 high-risk fathers who did not live in the child's home.

[c] All control families and marrying-in spouses were free of alcoholism, with 2 exceptions. In the case of the alcoholic father who married into the family, 4 of 12 first- and second-degree relatives were alcoholic. In the other case (from a target family), the alcoholic father was the only relative affected (1 of 12 first- and second-degree relatives). Thus, the familial loading for alcoholism remained quite small. The 2 alcohol-dependent control fathers also met criteria for drug dependence.

Assessing children from the ongoing family study provided 2 advantages. First, the psychiatric status of first- and second-degree relatives of these children was known. Alcoholism was diagnosed in 74% of the male probands' male siblings and 41% of their sisters, resulting in approximately 4 first- and second-degree relatives of the child being alcohol-dependent. Second, the comorbidity in parents was lower than that seen in epidemiological studies of alcoholics (Helzer and Pryzbeck, 1988; Kessler et al., 1994). This factor is especially important in identifying possible temperament characteristics in offspring of alcoholics that might be specifically prodromal of alcoholism. For example, other work (Rosenbaum et al., 1988) suggests that children of parents with major depressive disorder show elevated rates of behavioral inhibition.

Procedures

The parent accompanying the child to the session remained with his/her child throughout the testing, sitting quietly in the playroom. The play session was explained to the parent, who was asked to attend to the child but to not participate in the session. The parent was asked to not give the child any instructions unless he/she believed the child was imminently at risk of harming himself/herself or the other child. Compliance with this request along with any nonverbal behaviors of the parent were scored off-line by use of a videotape of the sessions. (Any noncompliance would be the occasion for exclusion of the child's data.)

Observations were made during the 30-minute play session through a 1-way mirrored window supplemented by cameras which provided additional views of the playroom through monitors in the observation room where coders were located. Two primary raters, one per child, listened with headphones to verbalizations of the children recorded by microphones placed at strategic points in the playroom ceiling. In addition, during the on-line live coding, each primary rater had another staff member observing the same child to serve as a backup so that no behaviors were missed. Primary coders operated a software-based data collection system of microswitches (one for each behavior) to precisely quantify the time spent in each behavior. A videotape of the session enabled coders to perform off-line scoring for verification. Any discrepancies were noted and resolved through the consensual agreement of 3 coders.

Before initiating coding of data, the laboratory coders first received extensive training in using explicit inclusion/exclusion criteria to categorize the behaviors to be scored. At the completion of training, all coders met an interrater reliability criterion of $r = 0.90$ with other coders. The sessions were scored for (1) latency of the first occasion to speak; (2) amount of time spent proximal to the parent (within the parent's reach); (3) the amount of time staring at the other child, neither speaking nor playing with the child at the time the staring occurred; (4) total time speaking (not including any interchange with parent); and (5) latency to touch the toys.

Data Analysis

A repeated-measures analysis of variance (ANOVA) was performed to determine the effects of risk group, repeated sessions, and their interaction on 5 selected dependent measures (latency to touch toys, latency to speak, total time staring at the other child, total time speaking, and total time proximal to the parent). The goal of this analysis was to determine whether the indicators of behavioral inhibition were stable over time (independent of session effects resulting from pairing the child with 3 different children). This analysis included all subjects tested including families with multiple sibs.

Because of concerns related to the nonindependence of data collected for sibs, a second analysis was performed using only one child per family. Because of the smaller number of children available for this analysis, nonparametric procedures were used. These analyses were conducted for averaged scores across multiple sessions as no significant session or session by group effects had been found in the ANOVA.

RESULTS

Demographic Characteristics

Table 1 shows the demographic characteristics of the children and the degree of familial loading for alcohol dependence. All peer play sessions were conducted with same-sex children who were between the ages of 4 years, 4 months and 6 years, 11 months. Children from the high- and low-risk groups did not differ with respect to the age of the mother or father or number of children in the family. The socioeconomic status of the high-risk children was somewhat lower than that of the low-risk group, though not significantly.

Parental Diagnosis

The high-risk children (having an average of 4.8 first- and second- degree alcoholic relatives) frequently came from homes in which a parent was alcoholic (Table 2). The parents' diagnoses were made using *DSM-III* criteria for Axis I psychopathology. (*DSM-III* was the diagnostic system in use when many of the families first entered into the larger family study.) In addition, a Research Diagnostic Criteria (RDC) diagnosis of alcoholism (3 out of 4 symptom categories must be positive) was also made. Thus individuals who met our criteria for alcoholism met criteria for both *DSM* and RDC.

Detailed assessments of the mother's drinking and/or drug use during pregnancy were evaluated because of the high rate of lifetime diagnoses of alcoholism. It was determined that 21% of the mothers did drink during pregnancy, though most decreased their intake once they determined they were pregnant.

Analysis of Continuous Data

Data for 30 children who completed all 3 sessions were analyzed with a repeated-measures ANOVA, using the appropriate Greenhouse-Geisser corrected degrees of freedom. (Two high-risk children completed 2 assessments, and 3 high-risk children and 1 low-risk child completed only 1 assessment.) Measures revealing unequal variances by risk group were corrected by using a log transform of 1 + the raw scores (several values were 0). Significant high- and low-risk differences were found

for total time staring at the other child ($F = 5.72$, $df = 1/28$, $p = .024$). In addition, the high-risk children exhibited less total time speaking ($F = 4.07$, $df = 1/28$, $p = .053$). It should be noted that the Harvard studies have shown that a reduction in total frequency of spontaneous comments and a long latency to begin to speak to the examiner differentiated the inhibited from the uninhibited children (Kagan et al., 1988).

No significant differences by session or group by session were detected. Absence of session effects indicated that the behavioral measures studied show stability over time and in encounters with various children. A repeated-measures ANOVA for the proximal to parent variable was not significant ($F = 3.27$, $df = 1/28$, $p = .082$). However, this was largely the result of habituation across sessions. A significant difference between risk groups was seen for the first session ($t = 2.19$, $df = 33$, $p = .036$).

Although data were first analyzed using raw scores from each session (ANOVA), the average time spent in each of the behaviors of interest was calculated and analyzed to illustrate the magnitude of the effect by risk group. As may be noted, the differences were rather dramatic in some cases. The high-risk children spent a significantly longer time staring at the other child (46.4 ± 17.7 seconds versus 7.3 ± 1.5 seconds; $t = 2.26$, $df = 33$, $p = .03$). Also, high-risk children spent a significantly shorter time speaking to the other child (106.5 ± 17.8 seconds versus 172.2 ± 20.5 seconds; $t = 2.42$, $df = 34$, $p = .02$).

Analysis Using One Child per Family

To ensure that the results were not found because multiple children from some families had been included, a Mann-Whitney U test was performed for the average time the child engaged in each of the behaviors found to be significant in the ANOVA. The U test for average amount of time speaking was significant ($U = 26.0$, $p = .030$), whereas the average amount of time staring was only marginally significant ($U = 27.0$, $p = .058$).

Nonparametric Analysis of Extreme Scores

As an additional test of the robustness of our findings, we sought to classify the children into 2 groups based on scores found to be most extreme from data collected by Kagan and colleagues (Kagan, personal communication, 1996). Thus, cutoffs of >1 minute for staring and for being proximal to the parent, along with a cutoff of <1 minute of speaking during the 30-minute session, were chosen. Chi-square analyses of data collected from the 100 evaluations revealed that significantly more high-risk children met the >1 minute criteria for staring ($\chi^2 = 3.91$, $df = 1$, $p < .05$). Also, significantly more high-risk children spent >1 minute proximal to the parent ($\chi^2 = 4.09$, $df = 1$, $p < .04$).

DISCUSSION

The presence of internalizing disorders in childhood and adolescence has been discussed with respect to elevated risk for alcohol dependence (Hill and Muka, 1996; Kellam, 1990; Kellam et al., 1983; McCord, 1988; Parker and Asher, 1987), though much less frequently than externalizing disorders. Kellam (1990) noted that shyness in combination with aggressive behavior among first-grade boys is associated with a greater likelihood of developing both delinquency and substance use than is aggressive behavior alone. A similar finding was reported by McCord (1988), who found that boys rated as both aggressive and shy were most likely to become alcoholics or criminals.

The present data demonstrate that preschool white children, with familial loading for alcoholism far in excess of that found in the general population, are more likely to be behaviorally inhibited to the unfamiliar than are children of parents without familial loading. Dawson and Grant (1998) found that only 9.4% of a representative sample of 42,862 U.S. adults had 25% or more of their relatives affected with alcoholism. This is in contrast to the high-risk sample used in the present study, in which all of the parents had 25% or more of their relatives affected with alcoholism.

Increased behavioral inhibition among the high-risk children was seen for the 3 major variables (staring, spending time proximal to the parent, and total time speaking to the other child). We did not find statistically significant differences by risk group for latency to touch toys or latency to speak. These results indicate that the amount of time spent staring and speaking during a peer play session is relatively independent of with whom the child is paired. However, response to the play setting appears to habituate somewhat inasmuch as the proximity to the parent measure was found to be significant only at the time of the first session.

There are a number of possible explanations for why increased behavioral inhibition (increased staring, increased time proximal to the parent, and decreased speaking to the other child) was observed in these high-risk children. Behavioral inhibition appears to represent

the behavioral manifestation of a lower threshold for limbic system arousal and increased central noradrenergic activity, both of which have been implicated in fear, anxiety, and inhibition (Charney and Redmond, 1983). Kagan et al. (1987) found that inhibited children had higher heart rates, greater pupil dilation, higher cortisol levels, and increased urinary catecholamine levels, all of which would appear to be consistent with the notion of greater limbic arousal.

The high-risk children may exhibit a higher frequency of inhibition because of biological susceptibility for increased limbic arousal, perceived as an undesirable anxious condition which makes the use of antianxiety agents, such as alcohol, particularly attractive. Increased limbic arousal may represent a common vulnerability factor for a number of adult psychopathological conditions including panic disorder, agoraphobia, major depressive disorder, as well as alcohol and/or drug dependence. Offspring of parents with panic disorder, agoraphobia, and major depressive disorders, who have a higher risk for developing anxiety disorders as a result of their familial loading, are more likely to exhibit a behaviorally inhibited temperament (Rosenbaum et al., 1988). These investigators, like the present study, used the peer play observation method developed at the Harvard Child/Infant Laboratory. However, the present study was designed to reduce comorbidity in the parents and extended kin of studied children, a condition necessary to describe adequately the relationship between temperamental antecedents in childhood and alcoholism in adulthood.

Although we do not expect that all children in the high-risk group will develop alcohol dependence, we expect that, as a group, a higher proportion of high-risk children (those with multiple alcoholic family members) will develop alcohol dependence (Bohman et al., 1981; Cloninger et al., 1981; Goodwin et al., 1973). Consistent with the hypothesis of increased limbic arousal among these high-risk children, we have found increased baseline heart rate in both adult alcoholics and their nonalcoholic siblings when they are confronted with the minimal challenge of a cognitive task in a laboratory setting (Hill et al., 1992).

Although the biological explanation appears particularly attractive, we must entertain the possibility that the environmental conditions presented to children from alcoholic homes could facilitate the presence of surveillant behavior, fear, and uncertainty. In this study it was not possible to separate the effects of genetic loading for alcoholism from the effects of living with an alcoholic parent. With respect to environmental exposure, the majority of the fathers who were alcoholic by lifetime diagnosis were drinking at the time of assessment. In contrast, only 1 of the 7 mothers who were alcoholic by lifetime diagnosis was currently drinking. In either case, only 7 high-risk children lived in homes in which neither parent met criteria for alcohol dependence at the time of assessment. Currently, it is unknown what effect living with an actively drinking parent might have on preschool child's tendency to be fearful of new situations or unfamiliar persons.

The research design chosen emphasizes families in which greater severity is exhibited (multiple affected cases within the extended pedigree). A research design that enriches the sample for alcohol dependence in relatives increases the possibility of finding a relationship between a biological predisposition (e.g., behavioral inhibited temperament) and alcohol dependence should such a relationship exist. Therefore, studies of children from alcoholic families with minimal comorbidity and greater severity would appear to be an important step in determining whether temperament contributes to the etiology of alcohol dependence (Hill and Neiswanger, 1997). Thus, we believe that the temperament effects seen in these high-risk children are largely the result of the alcohol dependence diathesis of the pedigrees from which the parents were drawn and/or the effects of living with alcoholic parents.

While the present results are intriguing, further work is needed with expanded samples of offspring of alcohol-dependent parents. Ideally, one would prefer to study children of affected parents who have been abstinent during the duration of the child's life. Obviously, this is a difficult sample to acquire. Nevertheless, the present results suggest that alcohol dependence can be included among other major psychiatric disorders (major depressive disorder, agoraphobia, anxiety disorders) as one that may have underpinnings from the temperament domain which are most easily seen in childhood through the study of offspring of clinically diagnosed parents (Rosenbaum et al., 1988).

Clinical Implications

The results of this study indicate that children from alcoholic families may be at greater risk for displaying a behaviorally inhibited temperament. While the childhood risks associated with behavioral undercontrol (acci-

dents, school problems) are well known and more prominent among children from alcoholic families, the risks associated with extreme behavioral inhibition are less well studied. In contrast, behaviorally inhibited children are often viewed by parents and teachers as "good" children because they are not as disruptive as children with externalizing behavior problems. However, there may be adverse effects for the child. The shy child may find making friends much more difficult. The child may experience greater distress from living with an alcoholic parent without the buffering effects of childhood friends. This early childhood pattern could lead to further alienation and lack of social skills in adulthood. The shy, young child also may disengage more readily from classroom participation, ultimately finding school less rewarding. Recognition by teachers that behaviorally inhibited children are at risk for a variety of problems may provide opportunities for intervention. For example, school-based programs that teach social skills to children identified as behaviorally inhibited could be effective in reducing psychiatric and substance abuse problems, when applied both to community samples as well as to high-risk children from alcoholic homes.

REFERENCES

Bohman M, Sigvardsson S, Cloninger CR (1981), Maternal inheritance of alcohol abuse: cross fostering analysis of adopted women. *Arch Gen Psychiatry* 38:965–969

Buss AH, Plomin R (1975), *A Temperament Theory of Personality Development*. New York: Wiley

Caspi A, Moffitt TE, Newman DL, Silva PA (1996), Behavioral observations at age 3 years predict adult psychiatric disorders. *Arch Gen Psychiatry* 53:1033–1039

Charney DS, Redmond DE (1983), Neurobiological mechanisms in human anxiety: evidence supporting central noradrenergic hyperactivity. *Neuropharmacology* 22:1531–1536

Chess S, Thomas A (1977), Temperamental individuality from childhood to adolescence. *J Am Acad Child Psychiatry* 16:218–226

Chess S, Thomas A (1984), *Origins and Evolution of Behavior Disorders*. New York: Brunner/Mazel

Clarkin JF, Friedman RC, Hurt SW, Corn R, Aronoff M (1984), Affective and character pathology of suicidal adolescent and young adult inpatients. *J Clin Psychiatry* 45:19–22

Cloninger CR, Bohman M, Sigvardsson S (1981), Inheritance of alcohol abuse: cross-fostering analysis of adopted men. *Arch Gen Psychiatry* 38:861–868

Colder CR, Chassin L (1993), The stress and negative affect model of adolescent alcohol use and the moderating effects of behavioral undercontrol. *J Stud Alcohol* 54:326–333

Dawson DA, Grant BF (1998), Family history of alcoholism and gender: their combined effects on *DSM-IV* alcohol dependence and major depression. *J Stud Alcohol* 59:97–106

Goldsmith HH, Gottesman II (1981), Origins of variations in behavioral style: a longitudinal study of temperament in young twins. *Child Dev* 52:91–103

Goodwin DW, Schulsinger F, Hermansen L, Guze SB, Winokur G (1973), Alcohol problems in adoptees raised apart from alcoholic biological parents. *Arch Gen Psychiatry* 28:238–243

Helzer JE, Pryzbeck TR (1988), The co-occurrence of alcoholism with other psychiatric disorders in the general population and its impact on treatment. *J Stud Alcohol* 49:219–224

Hill SY (1992), Absence of paternal sociopathy in the etiology of severe alcoholism: is there a type III alcoholism? *J Stud Alcohol* 53:161–169

Hill SY (1994), Etiology. In: *Annual Review of Addictions Research and Treatment*, Langenbucher J, McCrady B, Frankenstein W, Nathan P, eds. Tarrytown, NY: Elsevier Science (Pergamon Press), pp 127–148

Hill SY, Hruska DR (1992), Childhood psychopathology in families with multigenerational alcoholism. *J Am Acad Child Adolesc Psychiatry* 31:1024–1031

Hill SY, Muka DH (1996), Childhood psychopathology in children from families of alcoholic female probands. *J Am Acad Child Adolesc Psychiatry* 35:725–733

Hill SY, Neiswanger K (1997), The value of narrow psychiatric phenotypes and "super" normal controls. In: *Handbook of Psychiatric Genetics*, Blum K, Noble E, eds. Boca Raton, FL: CRC Press, pp 37–46

Hill SY, Steinhauer SR, Zubin J (1986), Biological markers for alcoholism: a vulnerability model conceptualization. In: *Alcohol and Addictive Behavior*, Rivers PC, ed. Lincoln: University of Nebraska Press, pp 207–256

Hill SY, Steinhauer SR, Zubin J (1992), Cardiac responsivity in individuals at high risk for alcoholism. *J Stud Alcohol* 53:378–388

Hill SY, Zubin J, Steinhauer SR (1990), Personality resemblance in relatives of male alcoholics: a comparison with families of male control cases. *Biol Psychiatry* 27:1305–1322

Hollingshead AB (1975), *Four Factor Index of Social Status*. New Haven, CT: Yale University Department of Sociology

Jones MC (1968), Personality correlates and antecedents of drinking patterns in adult males. *J Consult Clin Psychol* 32:2–12

Jones MC (1971), Personality antecedents and correlates of drinking patterns in women. *J Consult Clin Psychol* 36:61–69

Kagan J (1994), *Galen's Prophecy: Temperament in Human Nature*. New York: Basic Books

Kagan J, Reznick JS, Clarke C, Snidman N, Garcia-Coll C (1984), Behavioral inhibition to the unfamiliar. *Child Dev* 55:2212–2225

Kagan J, Reznick JS, Snidman N (1987), The physiology and psychology of behavioral inhibition in young children. *Child Dev* 58:1459–1473

Kagan J, Reznick S, Snidman N, Gibbons J, Johnson MO (1988), Childhood derivatives of inhibition and lack of inhibition to the unfamiliar. *Child Dev* 59:1580–1589

Kellam SG (1990), Developmental epidemiology framework for family research on depression and aggression. In: *Depression and Aggression in Family Interaction*, Patterson GR, ed. Hillsdale, NJ: Erlbaum, pp 1–48

Kellam SG, Brown CH, Rubin BR, Ensminger ME (1983), Paths leading to teenage psychiatric symptoms and substance use: developmental epidemiology studies in Woodlawn. In: *Childhood Psychopathology and Development*, Guze SB, Earls FJ, Barrett JE, eds. New York: Raven, pp 17–51

Kessler RC, McGonagle KA, Zhao S et al. (1994), Lifetime and 12-month prevalence of *DSM-III-R* psychiatric disorders in the United States. *Arch Gen Psychiatry* 51:8–19

Mannuzza S, Klein RG, Bessler A, Malloy P, LaPadula M (1993), Adult outcome of hyperactive boys: educational achievement, occupational rank, and psychiatric status. *Arch Gen Psychiatry* 50:565–576

McCord J (1988), Identifying developmental paradigms leading to alcoholism. *J Stud Alcohol* 49:357–362

McCord W, McCord J (1962), A longitudinal study of the personality of alcoholics. In: *Society, Culture, and Drinking Patterns*, Pittman DJ, Snyder CR, eds. New York: Wiley, pp 413–430

Newcomb MD, Bentler PM (1989), Substance use and abuse among children and teenagers. *Am Psychol* 44:242–248

Parker JG, Asher SR (1987), Peer relations and later personal adjustment: are low-accepted children at risk? *Psychol Bull* 102:357–389

Reich W, Earls F, Frankel O, Shayka JJ (1993), Psychopathology in children of alcoholics. *J Am Acad Child Adolesc Psychiatry* 32:995–1002

Reznick JS, Kagan J, Snidman N (1986), Inhibited and uninhibited behavior: a follow-up study. *Child Dev* 51:660–680

Robins L (1966), *Deviant Children Grown Up*. Baltimore: Williams & Wilkins

Rosenbaum JF, Biederman J, Gersten M et al. (1988), Behavioral inhibition in children of parents with panic disorder and agoraphobia. *Arch Gen Psychiatry* 45:463–470

Stice E, Myers MG, Brown SA (1998), A longitudinal grouping analysis of adolescent substance use escalation and de-escalation. *Psychol Addict Behav* 12:14–27

Tarter RE, Kabene M, Escallier EA, Laird SB, Jacob T (1990), Temperament deviation and risk for alcoholism. *Alcohol Clin Exp Res* 14:380–382

Thomas A, Chess S (1977), *Temperament and Development*. New York: Brunner/Mazel

Thomas A, Chess S, Birch HG (1970), The origins of personality. *Sci Am* 223:102–109

Watson D, Clark LA (1993), Behavioral disinhibition versus constraint: a dispositional perspective. In: *Handbook of Mental Control*, Wegner DM, Pennebacker JW, eds. New York: Prentice Hall, pp 506–527

COMMENTARY:

Behavioral Inhibition and Developmental Risk

NAIMAH Z. WEINBERG, M.D.

Why do children from the same environment and gene pool experience such different developmental outcomes? Why do children with the same diagnosis follow such varied courses? One key to help us understand these different trajectories is the study of temperament, a true biopsychosocial phenomenon.

The article by Hill et al. (1999) brings together this important subject, temperament, and a significant population, children from alcoholic families. Hill and her associates found significantly elevated rates of "behavioral inhibition to the unfamiliar" in children from families with a strong alcohol pedigree. Given that children of alcoholics (COAs) face a variety of developmental challenges, this timely and provocative report raises several important questions:

What is the developmental significance of behavioral inhibition in children? Some studies have found behavioral inhibition to increase the risk for later anxiety disorders (Rosenbaum et al., 1993), particularly in children with a family history of anxiety disorders, or later depression (Caspi et al., 1996). Of note, however, only about 30% of the inhibited children in each sample developed the outcome of interest; Kagan and Zentner (1996) have deemed the association between preschool psychological profiles and later psychopathology "modest." In terms of other aspects of functioning, individuals rated as inhibited at age 3 were found to be weak on "social potency" at age 18 (Caspi and Silva, 1995), but these same subjects were also described as conscientious, with normative adjustments in romantic relationships and work situations, at age 21 (Newman et al., 1997). Therefore, while behavioral inhibition in childhood merits attention and concern, it cannot be assumed to predict an adverse outcome in the majority of cases, and it particularly does not represent an equation with internalizing disorders.

For the sake of this discussion, "behavioral inhibition to the unfamiliar" is assumed to represent a temperament characteristic, that is, a constitutionally based tendency to respond to the environment with a pattern of behavior that is generally consistent across situations and stable over time. However, the laboratory paradigm used by many studies (including that by Hill et al.) does not assess cross-situational or long-term persistence of the pattern; there is evidence that the subset of children at true developmental risk may be those whose behavioral inhibition is consistent across a variety of situations (Rubin et al., 1997) and stable over time (Rosenbaum et al., 1993).

Is this finding specific to alcoholic families, or is behavioral inhibition a nonspecific characteristic of high-risk offspring? Behavioral inhibition has been found to be elevated in offspring of parents with panic disorder and agoraphobia, and there may be a weak tendency for it to occur at greater rates in offspring of parents with depression (Rosenbaum et al., 1988). However, family history of alcohol use disorders was not reported. Therefore, the specific relationship between parental psychopathology and offspring inhibition needs further study.

Will the findings of Hill and colleagues generalize to children from other alcoholic families? In other words, are offspring from other types of alcoholic pedigrees also at increased risk for behavioral inhibition? There are several

Accepted September 23, 1998.

Dr. Weinberg is a medical officer in the Division of Epidemiology and Prevention Research, National Institute on Drug Abuse, NIH, Rockville, MD.

Reprint requests to Dr. Weinberg, NIDA, 6001 Executive Blvd., Room 5N-5160, MSC #9589, Bethesda, MD 20892-9589.