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Children of Alcoholics: An Update

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Abstract

One in four children in the US live in a family in which they exposed to alcoholism. Children of alcoholics (COAs) are two to ten times more likely to develop alcoholism than non-COAs. This paper reviews studies that have attempted to identify risk factors that mediate the increased vulnerability, and protective factors that moderate the risk. Factors discussed include parental antisocial personality disorder, externalizing behavior, internalizing symptoms, differential response to the effects of alcohol, and positive and negative alcohol-related expectancies. The heterogeneous nature of COAs is emphasized, and some of the challenges related to treatment and prevention are discussed.

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Introduction

There is a great deal of interest in the effects of parental alcoholism on children, and it appears that this problem is growing. A recent study found that one in four children in the United States are exposed to family alcohol abuse or dependence [1]. Families with an alcoholic member tend to be more disorganized, unstable, and can lead to abuse or neglect of the children. Children growing up in these families are vulnerable to economic hardship, medical problems, psychopathology, and have an increased risk of developing substance abuse problems themselves. The increased risk is mediated in part via genetic vulnerability, increased risk for externalizing behavior, and internalizing symptoms, lower IQ and school performance.

Children of alcoholics (COAs) are two to ten times more likely to develop alcoholism than non-COAs. The prevalence of alcoholism among adults ages 18 years and older is approximately seven percent [2]. Still, most COAs do not develop alcoholism or other substance abuse disorders, and most of them do not develop any kind of psychiatric disorder at all. This finding has led to an intense interest in identifying risk factors or pathways (Table 1) that lead to either psychopathology or health, so that effective prevention and treatment interventions can be developed.

Phenotypic Markers

Efforts have been made to better understand phenotypic markers of high risk for alcoholism among subjects who have not yet developed any alcohol-related problems. Better characterization of risk factors or markers for high risk will allow preventive interventions to be directed at those children who are at greatest risk. Abnormalities of event-related potentials are

one of the most extensively studied markers of high risk for alcoholism. Anomalies of event-related potentials have been found to be associated with both the acute and chronic effects of alcohol intake. Their association with pre-morbid vulnerability to alcoholism was investigated when it was found that some anomalies, specifically abnormalities of the P300 component, were found not to recover at all, even after long periods of abstinence [3*]. The P300 wave is a positive deflection which is elicited in particular paradigms in which the subject is required to attend to specific stimuli. Further studies established that these abnormalities were present in sons of alcoholic males, who had never been exposed to alcohol, and this finding led to the hypothesis that these abnormalities were markers of genetic risk for alcoholism [4].

More recent studies have examined this phenomenon more closely. Visual and auditory event-related potentials were studied in the context of three distinct groups: children of alcoholic fathers with and without other alcoholic relatives, and a control group of children of nonalcoholics [5]. Only the children with multiple relatives affected by alcoholism displayed significant abnormalities. This result suggests that the abnormalities are associated with genetic factors, and are not the result of some unknown environmental factor associated with having an alcoholic father. It also suggests that it is important to distinguish so called high-density family history children from those who have only a single affected parent when studying event-related potential anomalies. Failure to do so may lead to a failure to find significant differences between COA and non-COA groups.

The actual connection between event-related anomalies and increased risk of alcoholism remains unclear. Other studies have attempted to clarify the relationship between phenotypic

characteristics of children of alcoholics and increased risk for the development of alcoholism. Differential response to the effect of alcohol is a variable that appears to be directly linked to alcohol-related problems. A large, ongoing, prospective study [6**] looked at the level of response to alcohol in drinking, but not alcohol dependent 20-year old sons of alcoholics and controls. At 15 year follow-up it was found that a low level of response to alcohol predicted the subsequent development of alcoholism.

Other studies support this finding. An earlier study [7] found that sons of alcoholics produce less cortisol, less adrenocorticotrophic hormone (ACTH), and display less body sway after two or three drinks. It appears that sons of alcoholics innately possess greater tolerance to the effects of alcohol than do sons of nonalcoholics, and are therefore at greater risk for developing alcohol-related problems. It has been suggested [8] that people tend to regulate their drinking by monitoring the effects the alcohol has on them. The experience of being impaired or intoxicated leads them to stop, or at least slow down, their alcohol consumption. Greater tolerance to the intoxicating effects of alcohol interferes with this regulatory mechanism, and leads to increased consumption.

Increasing the risk even further, sons of alcoholics appear to be more sensitive to the rewarding [9, 10] and anxiolytic [11] effects of alcohol. This type of response is manifest during the drinking stage in which blood alcohol levels are rising. Subjects with multigenerational family histories of alcoholism were compared to age-matched family history negative controls with high and low sensitivity to anxiety. Subjects were exposed to aversive stimuli, and physiological response was measured. Both family history positive and family history negative subjects with

high anxiety experienced dampened heart rate reactivity in response to alcohol. The family history negative subjects with low anxiety did not experience this kind of reaction. Anxiety disorders are a known risk factor for alcoholism [12], because people suffering from anxiety may use alcohol to “self medicate” overly stimulated states. The fact that subjects in this study with family histories of alcoholism experienced reactions to alcohol similar to the subjects with anxiety suggests that the “self medication” hypothesis might also explain a portion of their increased risk as well.

Hangover severity is another variable that distinguishes sons of alcoholics from sons of nonalcoholics. Span and Earleywine [13] confirmed earlier studies [14, 15] demonstrating that sons of alcoholics report greater hangover symptoms after ingestion of alcohol. Hangover symptoms are conceptualized as a kind of withdrawal syndrome. Some drinkers will attempt to ameliorate the symptoms of a hangover, by further ingestion of alcohol. This strategy is colloquially termed “hair of the dog.” Because the hangover symptoms return as blood alcohol levels fall, this strategy can lead to a pattern of increasing alcohol use. The authors speculate that greater withdrawal and hangover symptoms experienced by sons of alcoholics, make them more likely to treat the hangover with alcohol injection, and consequently at greater risk for the development of problem drinking.

Intranuova and Powers have published data suggesting that COAs may have an increased risk of alcoholism because beer tastes better to them [16]. 6-n-propylthiouracil (PROP) is a chemical present in some brands of beer that gives it a bitter flavor. People are able to taste PROP to varying degrees. COAs are significantly more likely to be nontasters of PROP than children of

nonalcoholics [17]. When tasters, nontasters, and supertasters of PROP were asked to rate a beer high in PROP (Pilsner Urquell), perceived bitterness was directly related to ability to taste PROP, and inversely related to the degree to which the subjects liked the taste of the beer. Because the ability to taste PROP results in less enjoyment of certain beers, those who are able to detect it might drink less. As mentioned above, COAs are more likely to be non-tasters of PROP. Therefore, the PROP present in certain beers would not lead to an unpleasant bitter taste, and the non-tasting sons of alcoholics might be more likely to consume it.

The study enrolled subjects with varying abilities to detect the taste of PROP, but none of them had alcohol abuse or dependence. Among this normal population, there was no relationship between PROP tasting ability and amount of alcohol consumed. Further studies are needed to clarify the significance of diminished PROP tasting ability in COAs.

Psychosocial Issues

A number of studies have examined the psychosocial consequences experienced by children of alcoholics, and have attempted to determine the factors that mediate increased risk of substance abuse disorders among COAs. Comorbidity of antisocial personality disorder in an alcoholic father appears to be one variable that mediates the development of behavioral and other problems among COAs. Compared to children from nonalcoholic homes, COAs have been found to have more behavioral problems. Higher levels of behavioral problems, as measured by the Achenbach Child Behavioral Checklist-Parent Version are found in children whose fathers have comorbid antisocial personality disorder [18*]. The same study found lower intellectual functioning among

COAs, although no additional effect among children who had antisocial fathers. The authors note that although IQ scores were lower among COAs, they were still within the normal range.

The actual relationship between internalizing symptoms, externalizing behavior and the development of substance abuse disorders among COAs is the focus of a study by Hussong, et al [19**]. Externalizing behaviors may increase the likelihood of the subsequent development of substance abuse due to contact with antisocial peers, however it is unclear whether externalizing behavior is a cause of alcohol involvement, or if both problems are manifestations of a single underlying trait [20]. Similarly, internalizing symptoms may lead to substance use through an attempt to self-medicate anxiety or depression, or the two may also reflect an underlying vulnerability present throughout development [21].

Hussong, et al found greater degrees of externalizing behavior among children of alcoholic and antisocial parents, and also found that these kinds of behavioral problems were a unique mediator between parent psychopathology and heavy alcohol use. However, parental alcoholism remained a significant risk factor for heavy alcohol use in adolescent offspring even after controlling for externalizing behavior, indicating that it is not the only factor accounting for the increased risk. The study did not find a relationship between greater internalizing symptoms and heavy alcohol use, thus failing to support the self-medication model. Internalizing symptoms were related to higher initial alcohol consumption, indicating that subjects who drank large amounts of alcohol were more likely to suffer from anxiety or depression. However, internalizing symptoms were not associated with prospective changes in heavy drinking, indicating that drinking behavior was not directly influenced by the symptoms. Therefore it

appears that symptoms of anxiety and depression are a result, rather than a precursor, of excessive alcohol involvement [22]. Although the association between alcohol and depression is well established, the relationship appears to be a complex one, and the actual mechanism by which alcohol may cause depression remains unknown [23, 24].

With substantial evidence supporting the negative effects of an antisocial father on the development of his children, it might be expected that the absence of an alcoholic father would be protective [25]. Carbonneau, et al examined the effect of paternal absence on the development of sons of alcoholics [26*]. Divorce is common among alcoholics [27], and antisocial fathers are at greater risk for incarceration, so a significant portion of COAs are raised in single parent families. Although the study did confirm the relationship between paternal alcoholism and developmental problems in boys, specifically oppositional and hyperactive behavior, it did not find any effect of paternal absence. This finding led the authors to suggest that the problem behaviors begin early, and persist over time, despite changes in family structure.

Alcohol-related expectancies is another way in which risk for alcoholism can be transmitted from parents to offspring. Children who believe that use of alcohol will lead to positive benefits are at greater risk for developing alcohol-related problems than those who have negative expectancies [28]. Examples of positive expectancies include the belief that alcohol will make them experience positive mood states, enhance sexual functioning, and increase assertiveness [29].

A study involving a group of normal second graders who were asked about the expected effects of beer versus iced tea in adults found that the children had greater negative expectancies toward beer, and more positive expectancies for iced tea [30]. Their expectancies also varied by the gender of the drinker. They expected fewer desirable consequences for female drinkers of beer than for male drinkers. The authors concluded that children develop expectancies regarding the effects of alcohol long before they actually begin to consume it.

There is debate over whether having an alcoholic parent is more likely to lead to positive or negative expectancies. One hypothesis proposes that children of alcoholics should have more negative expectancies as a result of witnessing the negative effects of alcohol on their parents and family. The alternative hypothesis is that children of alcoholics should have positive expectancies due to either social learning, or as a result of a more positive response to alcohol [31*]. Wiers et al examined these contrasting hypotheses in a group of COAs aged 7 to 18 years, and a group of age-matched controls. They found that elementary school-aged COAs had greater negative expectancies than controls, consistent with the first hypothesis. However, older COAs had more positive expectancies, suggesting that once alcohol use is initiated, the more favorable response experienced by COAs outweighs the earlier effects of aversive learning.

Because of the relationship between alcohol-related expectancies and drinking patterns, directly challenging positive expectancies would be expected to result in lower levels of consumption. This hypothesis was tested with interventions that either challenged the expectancy of increased arousal or increased sociability [32]. Both interventions were found to reduce both positive expectancies and actual alcohol consumption during the intervention, and six weeks later.

Conclusion

Identifying COAs remains a significant challenge. Because of the societal stigma associated with alcoholism, COAs are often reluctant to identify themselves out of a sense of shame. Offering universal prevention programs in schools to all children is one way to reach COAs without distinguishing them from their peers in a way that would make them uncomfortable.

Unfortunately, universal prevention programs, in which the intervention is offered to all members of a group regardless of their individual risk status, can be prohibitively expensive. A less expensive strategy is to identify patients in treatment for alcohol problems who have children, and then offering a selective intervention to those children.

Information arising from these and other studies point to multiple pathways of risk for children of alcoholics. Risk of developing alcoholism or other drug addiction is increased by differential responses to the effects of alcohol, and the numerous environmental stresses they can be exposed to as a result of having an alcoholic parent. Nevertheless, COAs represent a heterogeneous group, most of whom develop into normal adults without substance abuse problems. More work is needed to achieve a better understanding of the specific factors that mediate and moderate risk in this population, so that effective prevention and treatment strategies can be delivered to this very large group of at-risk children.

References

1. Grant BF: **Estimates of US Children Exposed to Alcohol Abuse and Dependence in the Family.** *Am J Public Health* 2000, **90**:112-115.

2. Grant BF, *et al.*: **Prevalence of DSM-IV Alcohol Abuse and Dependence: United States, 1992.** *Alcohol Health & Research World* 1994, **18**(3):243--245.

*3. Rodriguez Holguin S, Porjesz B, Chorlian DB, Polich J, Begleiter H: **Visual P3a in male subjects at high risk for alcoholism.** *Biol Psychiatry* 1999, **46**(2):281--91.

This study compared event-related potentials in sons and daughters of alcoholics with and without other first degree relatives affected by alcoholism. Only boys from families with more than one alcoholic relatives had event-related potentials that were significantly different from controls.

4. Begleiter H, Porjesz B, Bihari B, Kissin B: **Event-related brain potentials in boys at risk for alcoholism.** *Science* 1984, **225**(4669):1493--6.

5. Rodriguez Holguin S, Corral M, Cadaveira F: **Visual and auditory event-related potentials in young children of alcoholics from high- and low-density families.** *Alcohol Clin Exp Res* 1998, **22**(1):87--96.

6. Schuckit MA: **Biological, psychological and environmental predictors of the alcoholism risk: a longitudinal study. *J Stud Alcohol* 1998, **59**(5):485--94.

This paper presents data from a large, prospective study at 15 year follow up. It reports on the relationship between level of response to alcohol and risk for the development of alcoholism.

Lower response to the intoxicating effects of alcohol were correlated with greater risk for alcoholism.

7. Schuckit MA, Tsuang JW, Anthenelli RM, Tipp JE, Nurnberger JI, Jr.: **Alcohol challenges in young men from alcoholic pedigrees and control families: a report from the COGA project.** *J Stud Alcohol* 1996, **57**(4):368--77.
8. Finn PR, Justus A: **Physiological responses in sons of alcoholics.** *Alcohol Health and Research World* 1997, **21**(3):227--231.
9. Cohen HL, Porjesz B, Begleiter H: **The effects of ethanol on EEG activity in males at risk for alcoholism.** *Electroencephalogr Clin Neurophysiol* 1993, **86**(6):368--76.
10. Gianoulakis C, Krishnan B, Thavundayil J: **Enhanced sensitivity of pituitary beta-endorphin to ethanol in subjects at high risk of alcoholism [published erratum appears in Arch Gen Psychiatry 1996 Jun;53(6):555].** *Arch Gen Psychiatry* 1996, **53**(3):250--7.
11. Conrod PJ, Pihl RO, Vassileva J: **Differential sensitivity to alcohol reinforcement in groups of men at risk for distinct alcoholism subtypes.** *Alcohol Clin Exp Res* 1998, **22**(3):585--97.
12. Barlow DH: **Anxiety disorders, comorbid substance abuse, and benzodiazepine discontinuation: implications for treatment.** *NIDA Res Monogr* 1997, **172**:33--51.
13. Span SA, Earleywine M: **Familial risk for alcoholism and hangover symptoms.** *Addict Behav* 1999, **24**(1):121--5.
14. McCaul ME, Turkkan JS, Svikis DS, Bigelow GE: **Alcohol and secobarbital effects as a function of familial alcoholism: extended intoxication and increased withdrawal effects.** *Alcohol Clin Exp Res* 1991, **15**(1):94--101.
15. Newlin DB, Pretorius MB: **Sons of alcoholics report greater hangover symptoms than sons of nonalcoholics: a pilot study.** *Alcohol Clin Exp Res* 1990, **14**(5):713--6.

16. Intrantuovo LR, Powers AS: **The perceived bitterness of beer and 6-n-propylthiouracil (PROP) taste sensitivity.** *Ann N Y Acad Sci* 1998, **855**:813--5.

17. Pelchat ML, Danowski S: **A possible genetic association between PROP-tasting and alcoholism.** *Physiol Behav* 1992, **51**(6):1261--6.

*18. Puttler LI, Zucker RA, Fitzgerald HE, Bingham CR: **Behavioral outcomes among children of alcoholics during the early and middle childhood years: familial subtype variations.** *Alcohol Clin Exp Res* 1998, **22**(9):1962--72.

This study compared children from families with antisocial alcoholism, nonantisocial alcoholism fathers, and nonalcoholic controls. Antisocial personality disorder in addition to alcoholism led to greater psychosocial pathology in both boys and girls.

19. Hussong AM, Curran PJ, Chassin L: **Pathways of risk for accelerated heavy alcohol use among adolescent children of alcoholic parents. *J Abnorm Child Psychol* 1998, **26**(6):453--66.

Internalizing symptoms and externalizing behavior have been proposed as mediators between parental psychopathology and adolescent alcohol abuse. This paper tests the two hypotheses, finding support for externalizing behavior as a mediator, but not internalizing symptoms.

20. Loeber R, **Natural histories of conduct problems, delinquency, and associated substance use: Evidence for developmental progressions,** in *Advances in Clinical Psychology*, B.B.

Lahey and A.E. Kazdin, Editors. 1988, Plenum Press: New York; 73--125.

21. Sher KJ, *Children of Alcoholics: A critical appraisal of theory and research.* 1991, Chicago: University of Chicago Press.

22. Aneshensel CS, Huba GJ: **Depression, alcohol use, and smoking over one year: a four-wave longitudinal causal model.** *J Abnorm Psychol* 1983, **92**(2):134--50.

23. Patten SB, Charney DA: **Alcohol consumption and major depression in the Canadian population.** *Can J Psychiatry* 1998, **43**(5):502--6.

24. Smith DM, Atkinson RM: **Mood Disorders Secondary to Drugs and Pharmacologic Agents.** *Semin Clin Neuropsychiatry* 1997, **2**(4):285--295.

25. Biller HB, **Father absence, divorce and personality development,** in *The Role of the Father in Child Development*, M.E. Lamb, Editor. 1981, John Wiley & Sons, Inc.: New York; 319--358.

*26. Carbonneau R, *et al.*: **Paternal alcoholism, paternal absence and the development of problem behaviors in boys from age six to twelve years.** *J Stud Alcohol* 1998, **59**(4):387--98.

This study examined the effects of alcoholic fathers on their sons. Consistent with earlier reports, sons of male alcoholics were found to be more oppositional, more hyperactive, and more prone to aggression than controls. Interestingly, no effect on these variables was found for paternal absence.

27. Jacob T, Seilhamer RA, **The impact on spouses and how they cope,** in *Alcohol and the Family*, J. Orford and J. Harwin, Editors. 1982, St. Martin's Press, Inc.: New York; 114--126.

28. Gaffney LR, Thorpe K, Young R, Collett R, Occhipinti S: **Social skills, expectancies, and drinking in adolescents.** *Addict Behav* 1998, **23**(5):587--99.

29. Wall AM, Hinson RE, McKee SA: **Alcohol outcome expectancies, attitudes toward drinking and the theory of planned behavior.** *J Stud Alcohol* 1998, **59**(4):409--19.

30. Query LR, Rosenberg H, Tisak MS: **The assessment of young children's expectancies of alcohol versus a control substance.** *Addiction* 1998, **93**(10):1521--9.

*31. Wiers RW, Gunning WB, Sergeant JA: **Do young children of alcoholics hold more positive or negative alcohol-related expectancies than controls?** *Alcohol Clin Exp Res* 1998, **22**(8):1855--63.

Positive and negative expectancies regarding the effects of alcohol can influence the risk of developing alcoholism. This paper describes the different ways that having an alcoholic parent can influence alcohol-related expectancies.

32. Darkes J, Goldman MS: **Expectancy challenge and drinking reduction: process and structure in the alcohol expectancy network.** *Exp Clin Psychopharmacol* 1998, **6**(1):64--76.

Risk Factors for the Development of Alcoholism in COAs

Risk factor	Reference
Lower level of response to intoxicating effects of alcohol	[6, 7, 8]
Higher level of response to rewarding and anxiolytic effects of alcohol	[9, 10, 11, 31]
Greater hangover/withdrawal symptoms after drinking alcohol	[13, 14, 15]
Inability to taste PROP	[16, 17]
Fathers with co-morbid antisocial PD	[18]
Externalizing behavior	[19]
Positive alcohol-related expectancies	[28, 29, 31, 32]

Table 1: COAs are two to ten times more likely to develop alcoholism than non-COAs. The increased risk appears to be mediated via multiple pathways.

