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Neuropsychological Characteristics in Children of Alcoholics: Familial Density

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ABSTRACT. *Objective:* The purpose of high-risk studies is to find characteristics that allow the identification of subjects with a higher vulnerability to alcoholism. The aim of this research was to verify if the familial density criterion is useful for subtyping children of alcoholics with different neuropsychological characteristics. *Method:* A battery of neuropsychological tests was administered to 102 boys and girls of 7-15 years of age; 66 were children of alcoholics with a high (n = 32) and low (n = 34) familial density of alcoholism, and 36 were children of nonalcoholic fathers with a negative family history of the disorder. The battery included tests to assess attention, visuospatial abilities and frontal functions. *Results:* MANCOVAs showed that high-density children scored lower than children of nonalcoholic fathers in attentional and visuospatial tasks. There were no differences between low-density and negative family history children in these cognitive domains. *Conclusions:* These results suggest that children of alcoholics are not a ho-mogeneous group. Children with multigenerational alcoholism, but not children with an alcoholic father, showed reduced performance in specific cognitive areas.

Since family, twin and adoption studies provided evidence of a genetic contribution in the etiology of alcoholism (see Hesselbrock, 1995, for a review), a number of researchers have accordingly explored different characteristics in children of alcoholics, with the aim of identifying risk markers that may be related to a vulnerability to develop the disorder.

Studies of the neurotoxic effects of ethanol have shown that alcoholics manifest several neuropsychological deficits, some of which remain after a year of abstinence (Yohman et al., 1985). Early studies of cognitive functioning in delinquent males with family history of alcoholism (Tarter et al., 1984) or in children with family history of psychopathology (alcoholism included) (Gabrielli and Mednick, 1983) also showed that they had characteristics similar to those observed in alcoholics. This evidence allowed for the hypothesis that, rather than a consequence of chronic alcohol consumption, some neuropsychological deficits may be premorbid (Parsons, 1987) and, therefore, potential risk markers.

High-risk studies addressing this hypothesis have consistently observed lower performance in children with a family history of alcoholism than in children without alcoholic relatives in attentional (Ozkaragoz et al., 1997; Tarter et al., 1989b; Whipple et al., 1988), visuospatial (Berman and Noble, 1995; Garland et al., 1993; Ozkaragoz and Noble, 1995; Schandler et al., 1988, 1993; Sher et al., 1991; Whipple et al., 1988) and frontal tasks (Drejer et al., 1985; Harden and Pihl, 1995; Peterson et al., 1992; Tarter et al., 1989a), although their scores always fell within the range of normality. These differences have been interpreted as either an indication of an anterior cerebral dysfunction (Peterson and Pihl, 1990; Tarter et al., 1990), attention and information-coding deficiencies (Schandler et al., 1993) or an alteration on the

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dopaminergic system (Berman and Noble, 1995). Other studies, however, did not observe significant performance differences related to a family history of alcoholism (Alterman et al., 1986, 1989; Gillen and Hesselbrock, 1992; Hesselbrock et al., 1988; Schuckit et al., 1987; Workman-Daniels and Hesselbrock, 1987).

These contradictory results may be due, in part, to the risk criteria used for sample selection. The conventional classification considers two groups of subjects, children of alcoholics and children without a family history of alcoholism. Alterman (1988) showed the limitations of this classification scheme for studying familial alcoholism. Furthermore, he concluded that other classifications based on the linearity and number of generations or relatives affected in the family were more sensitive for identifying characteristics related to familial alcoholism.

Only a few high-risk studies have used these or similar criteria (e.g., family history of those alcoholic subtypes in which genetic factors seem to be more relevant) to select children at greater risk for alcoholism (Garland et al., 1993; Harden and Pihl, 1995; Ozkaragoz et al., 1997; Tarter et al., 1989a). These studies have generally obtained more consistent results than those using conventional criteria. However, they maintain the limitations related to dichotic classifications. It is not possible to determine whether differences are due to environmental factors (e.g., being brought up in a family affected by alcoholism) or to genetic factors, as one group shares both of the influences and the other group neither of them. With this taken into consideration, the first aim of this study was to investigate whether there are differences in neuropsychological performance between children with different familial density of alcoholism. The second aim was to investigate whether the differences are related to the genetic background of the disorder or to the presence of an alcoholic parent.

Method

Subjects

The subjects were 102 boys and girls, 7-15 years of age, divided into three groups according to familial density of alcoholism (the number of first- and second-degree alcoholic relatives) (Table 1). The high-density (HD) group comprised 32 children with alcoholic fathers, with at least two other relatives who were alcoholics. The low-density (LO) group comprised 34 children with alcoholic fathers, without other affected relatives. The negative family history (FH-) group comprised 36 children without first- and second-degree alcoholic relatives.

The lower limit for the age range of the subjects was selected because some of the neuropsychological tests used are standardized for children older than six. The upper limit was selected in order to guarantee that subjects were not regular users of alcohol. As the age range corresponds to the compulsory schooling age in Spain, children were of a similar educational level. In order to assure the homogeneity of the groups and to control variables that might exercise a relevant influence on the neuropsychological performance of the children, we determined the following exclusion criteria: (1) use/abuse of alcohol or illicit drugs; (2) current illness or use of medication; (3) history of head injury, loss of consciousness or neurological disorders; (4) hearing, visual or motor deficiency; (5) leaming disability or mental retardation; (6) history of psychiatric disorders; (7) alcohol consumption by the mother dur- ing pregnancy (four or more drinks per week) or maternal his- tory of alcoholism; (8) familial history of psychiatric disorders in first- and second-degree relatives (except alco- holism in the case of subjects in the HD and LD groups).

Alcoholic families were contacted through alcoholism treatment centers. Nonalcoholic families were contacted through schools and their selection took place once the children of alcoholics had been chosen. The reason for this was to make the groups homogenous for the most relevant

socio- demographic variables (age, sex, grade, family income, parent occupations and level of education attained). Information necessary for the selection was gathered from individual interviews with the father, mother and children. We used a battery of semistructured interviews, based on DSM-11-R criteria (American Psychiatric Association, 1987), that includes sections for the assessment of each member of the family and a module for assessing the psychiatric family history. The diagnosis of alcoholism was made by staff of each treatment center according to DSM-11-R criteria and corroborated during the interview. As can be seen in Table 1, there were no differences among HD and LD alcoholic fathers regarding the age of drinking onset, abuse and treatment.

Families who failed to meet the basic inclusion criteria were excluded. Parents of selected children signed a consent form befare the assessment and received 5,000 pesetas (approximately \$35) for their participation.

Measures

The neuropsychological battery was composed of eight tests to assess those cognitive functions that were of interest. These tasks were selected for their sensitivity in detecting alterations in these areas. To facilitate the comparisons with the literature, tasks that had been used previously in other studies with young children of alcoholics were chosen when possible. The neuropsychological functions that interested us most were attention, visuospatial abilities and frontal functions. The following tasks were selected to measure abilities in these cognitive domains:

Attention. WISC-R Digit Span and Digit Symbol subtests (Wechsler, 1993) were selected as measures of short-term storage capacity and complex attention, respectively (Lezak, 1995). Measures were scored according to the standard system. The Toulouse-Piéron Test (Toulouse-Piéron, 1986), a cancellation task that requires sustained attention, was the third attention task selected. It consists of 40-character rows in which the target character is randomly interspersed 10 times in each row. Characters are squares with a dash on one side. The subject is instructed to cross out all target squares (those with a horizontal-left or bottom-diagonal-right dash). The correct responses (minus errors and omissions) overa 10-minute period are noted.

Visuospatial ability. The battery included three visuospatial tasks with different levels of difficulty: Embedded Figures Test (EFT) and Children's Embedded Figures Test (CEFf) (Witkin et al., 1987), Complex Figure Test (copy) (Rey, 1987) and Block Design subtest (WISC-R). These tasks were selected as measures of visuospatial organization because they imply the spatial component in perception and/or motor execution. They have been found to be sufficiently sensitive for detecting differences in both alcoholics and their children.

Frontal function. WISC-R Mazes subtest and computerized version of the Wisconsin Card Sorting Test (WCST) (Neurosoft, 1990) were selected as measures of planning and self-regulation components of the frontal function. The standard scoring system of Mazes provided information about the planning ability of subjects, and the two seores from WCST (perseverative errors and failure to maintain the set) provided information about cognitive flexibility and capacity to shift.

Tests were administered in the same order for all subjects in a single session and took approximately 1.5 hours to complete. There were no differences in the distribution of the subjects by hand dominance, as determined by the Edinburgh Inventory (Oldfield, 1971).

Results

Separate 3 x 2 MANCOVAs, familial density by gender with age as a covariate, were conducted for each group of measurements (attention, visuospatial and frontal). The dependent variables were Digit Span, Digit Symbol and the Toulouse-Piéron Test seores in the MANCOVA for the attentional tasks; Block Design and the Complex Figure Test (copy) seores in the MANCOVA of visuospatial tasks; and Mazes and the WCST (percentage of perseverative errors and failure to maintain the set) in the MANCOVA of frontal tasks. Embedded Figures Test performance was analyzed by a univariate ANCOVA, as adult and child seores systems are different (time of figure identification and number of correct responses, respectively). Where a MANCOVA F was significant ($p \le .05$), univariate ANCOVAs with post hoc contrasts were computed.

Age and familial density by gender effects

The covariate was significant in all three MANCOVAs, at tentional (Wilks' lambda= 0.328, F = 61.37, 3/90 df, $p \le .001$), visuospatial (Wilks' lambda= 0.527, F = 40.724, 2/91 df, $p \le .001$) and frontal (Wilks' lambda= 0.779, F = 8.307, 3/88 df, $p \le .001$).

Analysis showed no significant familial density by gender interaction effect on the attentional (Wilks' lambda = 0.960, F = 0.604, 6/180 df, p = .727), visuospatial (Wilks' lambda= 0.967, F = 0.765, 4/182 df, p = .549) or frontal (Wilks' lambda= 0.881, F = 1.901, 6/176 df, p = .083) measures.

Familial density

Table 2 shows the children's seores and significance for all tests. The 3 x 2 MANCOVA conducted for the attentional tasks showed a significant overall effect for familial density (Wilks' lambda= 0.860, F = 2.339, 6/180 df, $p \le .034$) due to performance in Digit Span (F = 6.537, 2/92 df, p := ;; .02). Post hoc contrasts showed that HD children had a lower performance than FH- children ($p \le .0008$). There were no significant differences among the performances of FH- and LD groups.

Table 2. Neuropsychological test scores (mean ± SD) for children by familial density of alcoholism.

Variable	FH-	LD	HD	p	Post hoc contrasts	
					FH-/LD	FH-/HD
Attention						
Digit Span	13.08 ± 3.77	12.62 ± 3.65	10.81 ± 2.46	.002	NS	0.0008
Digit Symbol	48.75 ± 14.83	46.29 ± 15.32	47.61 ± 14.35	.390		
Toulouse-Piéron (raw score)	120.41 ± 56.90	109.85 ± 55.83	104.47 ± 43.95	.221		
Visuospatial abilities						
EFT (mean sg./figure)	55.91 ± 27.08	60.54 ± 37.49	64.36 ± 28.13	.641		
CEFT (no. correct responses)	19.35 ± 5.46	20.00 ± 2.66	20.43 ± 3.94	.404		
Complex Figure Test (copy)	26.89 ± 5.94	27.89 ± 5.29	26.88 ± 4.56	.531		
Block Design	37.03 ± 11.03	33.15 ± 13.39	32.06 ± 10.23	.026	NS	0.007
Frontal functions						
Mazes	22.83 ± 3.87	23.12 ± 4.20	23.03 ± 3.85	.728		
WCST						
Perseverative errors (%)	22.92 ± 14.21	24.00 ± 11.93	24.00 ± 17.67	.942		
No. failures to maintain the set	1.50 ± 1.34	1.59 ± 1.21	1.91 ± 1.63	.565		

A main effect for familial density was also found in the visuospatial tasks (Wilks' lambda= 0.893, F = 2.623, 4/182 df, $p \le .036$). Univariate ANCOVAs showed that dif- ferences were due to performance in Block Design (F = 3.799, 2/92 df, $p \le .026$) where HD children scored lower than FH-children ($p \le .007$). There were no signifi- cant differences among FH- and LD children. Univariate ANCOVAs conducted on the CEFf (F = 0.92, 2/45 df, $p \le .404$) and EFf (F = 0.45, 2/41 df, $p \le .641$) did not show any significant differences between the groups.

The third MANCOVA, conducted in the frontal tasks, did not show any overall effect on the group's performance (Wilks' lambda = 0.974, F = 0.377, 6/176 df, $p \le .892$).

Discussion

The results here are in accordance with conclusions from other studies that suggest that children of alcoholics manifest a lower performance in specific cognitive areas than do children without a family history of alcoholism. We observed lower seores in attentional and visuospatial tasks, functions in which the literature consistently shows differences: Studies using the Digit Span (Ozkaragoz and Noble, 1995; Ozkaragoz et al., 1997; Whipple et al., 1988) and the Block Design (Whipple et al., 1988) subtests have shown differences between young children with and without family history of alcoholism (a study by Ozkaragoz et al. [1995] using the Block Design showed no such differences). Furthermore, the comparison of groups with different environmental and genetic vulnerability to alcoholism allows us to answer the second question, regarding the origin of the differences. These results show that only one group with a family history of alcoholism manifest differences in performance compared to children of nonalcoholic fathers. The differences are effectively due to children of high-density families, and the analyses did not show significant differences in any case between the performance of LO and FH- children. These results indicate that there are subgroups of children of alcoholics with different neuropsychological characteristics, which have been considered as a homogenous group in many previous studies.

Recently, this possibility was also defended by Noble et al. (Ozkaragoz and Noble, 1995; Ozkaragoz et al., 1997). They observed different neuropsychological characteristics between two groups of children of alcoholics based on the type of parental alcoholism. The authors observed that children of active alcoholic fathers (abstinence < 2 years) (Ozkaragoz and Noble, 1995) manifest lower performance on visuospatial and attentional tasks than do children of recovery alcoholic fathers. According to this criterion, 97.2% of the alcoholics in our sample may be considered active. The main difference between LO and HD alcoholics is the familial density of alcoholism. As may be seen in Table 1, there was a similarity between groups for other clinical variables. These results lead us to the conclusion that differences among HD and FH-children may be due to factors related to the familial density of alcoholism, more than to the environ- ment they grow up in. In view of our results, it does not seem that being raised in an alcoholic family significantly affects neuropsychologial test performance. These results could explain why studies using conventional criteria (e.g., only one alcoholic relative, usually the father) obtain homogenous results: They use broad criteria, and therefore their samples are composed of children with different antecedents of alco- holism. However, studies using familial density criteria (Berman and Noble, 1995; Harden and Pihl, 1995; Ozkaragoz and Noble, 1995; Whipple et al., 1988) have observed differences in cognitive performance between children of alcoholic fathers and children of nonalcoholic fathers in every case.

Unlike other authors (Drejer et al., 1985; Harden and Pihl, 1995; Peterson et al., 1992; Tarter et al., 1989a), we did not observe differences between the groups based on a familial history of alcoholism, as far as performance in frontal tasks is concerned. This discrepancy is not due to the sensitivity of the material employed in this study, given that the same tests have shown significant differences according to the family history of alcoholism in research carried out by other authors. A possible explanation for this discrepancy

could lie in the sample characteristics. In our study, a history of psychiatric disorders, including conduct disorder, was considered as criterion for exclusion. However, excluding the research of Tarter et al. (1989a), a review of studies mentioned above shows that the samples were composed of children with conduct problems, or this variable did not control adequately. Furthermore, Harden and Pihl (1995) emphasized that there is a positive correlation between performance in the frontal tasks and the score on a conduct problems scale. It is therefore possible that frontal difficulties may be related more to the presence of a conduct disorder than to a family history of alcoholism.

In summary, the assessment of the cognitive abilities of children of alcoholics allows us to give support to the premise that there are subgroups with different cognitive characteristics in relation to familial density of alcoholism. It seems that only children with several antecedents of alcoholism, and not all children living in a family affected by the disorder, manifest lower performance than children of non-alcoholic fathers. Furthermore, given that performance differences are systematically observed in the same functions, even when the tests are different, it is possible that these characteristics may be used as potential risk markers for alcoholism. Naturally, before we arrive at any conclusions, it will be necessary to conduct follow-up studies to discover the predictive value of these neuropsychological characteristics in relation to future alcohol use/abuse.

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References

AMERICAN PSYCHIATRIC ASSOCIATION Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R), Washington, D.C., 1987.

ALTERMAN, A.I. Pattems of familial alcoholism, alcoholism severity, and psychopathology. J. Nerv. Ment. Dis.**176:** 167-175, 1988.

ALTERMAN, Al., BRIDGES, K.R. AND TARTER, RE. The influence of both drinking and familial risk statuses on cognitive functioning of social drinkers. Alcsm Clin. Exp. Res. **10:** 448-451, 1986.

ALTERMAN, A.I., SEARLES, J.S. AND HALL, J.G. Failure to find differences in drinking behavior as a function of familial risk for alcoholism: A replication. J. Abnorm. Psychol. **98**: 50-53, 1989.

BERMAN, S.M. AND NOBLE, E.P. Reduced visuospatial performance in children with the D2 dopamine receptor Al allele. Behav. Genet. **25**: 45-58, 1995.

DREJER, K., THEILGAARO, A., TEASOALE, T.W., SCHULSINGER, F. AND GOODWIN, D.W. A prospective study of young men at high risk far alcoholism: Neuropsychological assessment. Alcsm Clin. Exp. Res. **9:** 498-502, 1985.

GABRIELLI, W.F., JR. AND MEDNICK, S.A. Intellectual performance in children of alcoholics. J. Nerv. Ment. Dis. **171**: 444-447, 1983.

GARLAND, M.A., PARSONS, O.A. AND NIXON, S.J. Visual-spatial learning in nonalcoholic young adults with and those without a family history of alcoholism. J. Stud. Alcohol **54:** 219-224, 1993.

GILLEN, R. AND HESSELBROCK, V. Cognitive functioning, ASP, and family history of alcoholism in young men at risk far alcoholism. Alcsm Clin. Exp. Res. **16**: 206-214, 1992.

HARDEN, P.W. AND PIHL, R.O. Cognitive function, cardiovascular reactivity, and behavior in boys at high risk far alcoholism. J. Abnorm. Psy- chol. **104:** 94-103, 1995.

HESSELBROCK, V.M. The genetic epidemiology of alcoholism: In BEGLEITER, H. AND KISSIN, B. (Eds.) The Genetics of Alcoholism, New York: Oxford Univ. Press, Inc., 1995, pp. 17-39.

HESSELBROCK, V., O'CONNOR, S., TASMAN, A. AND WEIDENMAN, M. Cognitive and evoked potential indications of risk far alcoholism in young roen. In: KURIYAMA, K., TAKADA, A. AND ISHII, H. (Eds.) Biomedical and Social Aspects of Alcohol and Alcoholism, New York: Elsevier Science Publishing Co., Inc., 1988, pp. 583-585.

LEZAK, M.D. Neuropsychological Assessment, 3rd Edition, New York: Oxford Univ. Press, Inc., 1995, pp. 352-384.

NEUROSOFf, INC. Cardsort, Version 1.8, El Paso: Neurosoft, Inc., 1990.

OLDFIELD, R.C. The assessment and analysis of handedness: The Edinburgh Inventory. Neuropsychologia **9:** 97-113, 1971.

OZKARAGOZ, T.Z. AND NOBLE, E.P. Neuropsychological differences between sons of active alcoholic and non-alcoholic fathers. Alcohol Alcsm **30**: 115-123, 1995.

OZKARAGOZ, T., SATZ, P. AND NOBLE, E. Neuropsychological functioning in sons of active alcoholic, recovering alcoholic, and social drinking fathers. Alcohol **14:** 31-37, 1997.

PARSONS, O.A. Neuropsychological consequences of alcohol abuse: Many questions-some answers. In: PARSONS, O.A., BUTTERS, N. AND NATHAN, P.E. (Eds.) Neuropsychology of Alcoholism: Implications fOr Diagnosis and Treatment, New York: Guilfard Press, 1987, pp. 153-175.

PETERSON, J.B.,FINN, P.R. AND PIHL, R.O. Cognitive dysfunction and the inherited predisposition to alcoholism. J. Stud. Alcohol 53: 154-160, 1992.

PETERSON, J.B. AND PIHL, R.O. Information processing, neuropsychological function, and the inherited predisposition to alcoholism. Neuropsychol. Rev.1: 343-369, 1990.

REY, A. Test de Copia de una Figura Compleja, Madrid: TEA (original work published, 1959), 1987.

SCHANDLER, S.L., BRANNOCK, J.C., COHEN, M.J., ANTICK, J. AND CAINE, K. Visuospatial learning in elementary school children with and without a family history of alcoholism. J. Stud. Alcohol **49**: 538-545, 1988.

SCHANDLER, S.L., BRANNOCK, J.C., COHEN, M.J. AND MENDEZ, J. Spatial learning deficits in adolescent children of alcoholics. Exp. Clin. Psychopharmacol. **1:** 207-214, 1993.

SCHUCKIT, M.A., BUTTERS, N., LYN, L. AND IRWIN, M. Neuropsychologic deficits and the risk far alcoholism. Neuropsychopharmacology **1:** 45-53, 1987.

SHER, K.J., WALITZER, K.S., WOOD, P.K. AND BRENT, E.E. Characteristics of children of alcoholics: Putative risk factors, substance use and abuse, and psychopathology. J. Abnorm. Psychol. **100**: 427-448, 1991.

TARTER, R.E., HEGEDUS, A.M., GOLDSTEIN, G., SHELLY, C. AND ALTERMAN, A.I. Adolescent sons of alcoholics: Neuropsychological and personality characteristics. Alcsm Clin. Exp. Res. **8:** 216-222, 1984.

TARTER, R.E., JACOB, T. AND BREMER, D.A. Cognitive status of sons of alcoholic men. Alcsm Clin. Exp. Res. **13**: 232-235, 1989a.

TARTER, R.E., JACOB, T. AND BREMER, D.L. Specific cognitive impairment in sons of early onset alcoholics. Alcsm Clin. Exp. Res. **13:** 786- 789, 1989b.

TARTER, R.E., LAIRD, S.B. AND MOSS, H.B. Neuropsychological and neurophysiological characteristics of children of alcoholics. In: WINDLE, M. AND SEARLES, J.S. (Eds.) Children of Alcoholics: Critical Perspectives, New York: Guilford Press, 1990, pp. 73-98.

TOULOUSE, E. AND PIÉRON, H. Toulouse-Piéron: Prueba perceptiva y de atención, Madrid: TEA (original work published, 1911), 1986.

WECHSLER, D. Escala de Inteligencia de Wechsler para Niños: Revisada, Madrid: TEA (original work published, 1974), 1993.

WHIPPLE, S.C., PARKER, E.S. AND NOBLE, E.P. An atypical neurocognitive profile in alcoholic fathers and their sons. J. Stud. Alcohol. **49:** 240-244, 1988.

WITKIN, H.A., OLTMAN, P.K., RASKIN, E. AND KARP, S.A. Test de Figuras Enmascaradas, Madrid: TEA (original work published, 1971), 1987.

WORKMAN-DANIELS, K.L. AND HESSELBROCK, V.M. Childhood problem behavior and neuropsychological functioning in persons at risk far alcoholism. J. Stud. Alcohol **48:** 187-193, 1987.

YOHMAN, J.R., PARSONS, O.A. AND LEBER, W.R. Lack of recovery in male alcoholics' neuropsychological performance one year after treatment. Alcsm Clin. Exp. Res. **9:** 114-117, 1985.