

ATTENTION AND IMPULSIVITY CHARACTERISTICS OF THE BIOLOGICAL AND ADOPTIVE PARENTS OF HYPERACTIVE AND NORMAL CONTROL CHILDREN

Jody Alberts-Corush, Ph.D., Philip Firestone, Ph.D., John T. Goodman, Ph.D.

On tests comparing 176 biological and adoptive parents of hyperactive and normal control children, biological parents of hyperactives evidenced more attentional difficulties, slower mean reaction times, and fewer correct recognitions than did the other parents. They showed no significant differences in impulsivity. A familial association between childhood hyperactivity and attentional deficits in the biological parents was suggested, as was the persistence of attentional difficulties as compared to impulse control problems.

Hyperactivity is a leading cause of child clinical referrals, accounting for approximately one-third of all diagnosed psychiatric disorders in school age children.³⁵ Studies of the incidence of hyperactivity in the general population is variably estimated to range from 3% to 10%.²¹ Although physical overactivity has historically been identified as the major component of hyperactivity, the recent diagnostic category revisions in the *DSM-III* indicate a shift in viewpoint from overactivity to a disorder of attention as the most central factor in the disorder.

As Whalen⁴³ aptly pointed out, it is no longer necessary to prove that hyperactivity exists. Nevertheless, despite the general acceptance of the reality of

hyperactivity as a clinical phenomenon, relatively little is known of its etiological roots although recent theories have focused on biologically-based factors. Of particular interest to the present investigation has been the work on family morbidity and risk. Several papers have reported a higher incidence of psychopathology in the parents of hyperactive children when compared with control groups.^{3, 27} When the parents of hyperactive children were compared to the parents of psychiatric outpatient children, a higher incidence of antisocial personality and hysteria was found in the former than in the latter group. Both groups appeared to have a higher than normal incidence of alcoholism and the parents of the psychiatric

A revised version of a paper submitted to the Journal in January 1986. Authors are at: Department of Psychology, Ottawa Board of Education (Alberts-Corush), Child Study Center, School of Psychology, University of Ottawa (Firestone), and Department of Psychology, Children's Hospital of Eastern Ontario (Goodman).

population evidenced a higher incidence of schizophrenia and schizophreniform psychoses than the parents of hyperactives.²⁶

In support of a genetic hypothesis to explain the higher incidence of psychopathology in the parents of hyperactives as compared to controls, several adoption studies have found support for a greater frequency of disorders in the biological parents of hyperactive children when compared to the adoptive parents of hyperactives.^{2, 28, 37} Finally, further but limited support for a genetic etiology for hyperactivity has been presented by Lopez,²² whose study of ten sets of twins found a 100% concordance rate for hyperactivity in the monozygotic pairs and only a 17% concordance in the dizygotic pairs.

In addition to the family risk studies, research concern as to the course and long-term prognosis of hyperactivity into adolescence and adulthood has served to broaden the area of study of this complex disorder. The findings of an increasing number of prospective longitudinal studies since 1970 indicate that while the problems of physical overactivity and distractibility evidence some improvement, the attentional deficits and impulse control problems endure into adolescence and adulthood.^{6, 16, 19, 41} Impulsive and immature-dependent personality disorders have been reported to be more frequently diagnosed in hyperactives than in controls.¹⁷ Independent investigators examining other areas of psychopathology have retrospectively linked adult impulse and personality disorders with childhood hyperactivity.^{15, 33} The recent studies suggest that there is a chronic, developmental component to hyperactivity. Moreover, the findings raise important questions regarding the role that familial and envi-

ronmental factors may play in the psychosocial outcome in hyperactive teenagers and adults.

Reviews by Dubey¹⁰ and McMahon²³ have pointed out several flaws in the family risk research, including poor diagnostic procedures for the populations being studied, lack of homogeneity of the groups, poor sampling procedures, lack of "blindness" as to diagnostic groups being studied by the investigators, and the lack of well-known standardized assessment tools in the measurement of the psychological characteristics in question. Despite these criticisms, the large number of studies involved strongly suggests a familial association for hyperactivity in which genetic factors are of major importance.

In the present study more empirical measures of psychological functioning were utilized in an attempt to answer one major question: do the parents of hyperactive children show the same cognitive deficits as the hyperactive children? In order to answer this, the biological and adopted parents of hyperactives and normal controls were set the experimental tasks on which hyperactives have demonstrated poor performance—namely, those related to attention and impulse control.

METHOD

Subjects

Forty-three hyperactive children and their families participated in the study. The children were selected from the files of the department of psychology of the Children's Hospital of Eastern Ontario (CHEO) in Ottawa. Twenty-five were the biological offspring of the parents under investigation and 18 were adopted before six months of age. The children were required to meet the following criteria for the study: 1) having been referred by a pediatrician who suspected hyperactivity; 2) having been

diagnosed as hyperactive by a registered psychologist (PF) in accordance with *DSM-III* criteria for Attention Deficit Disorder with Hyperactivity; 3) having received a Conners's Teacher Hyperactivity Index (HI) score of 1.5 or higher on the teachers' rating scale.⁷

A total of 25 biological and 20 adopted normal-control children and their parents participated in the study. The 25 biological and two of the adopted normal controls were selected from the medical records of CHEO. After the identification and acceptance of participation by the parents of the hyperactive index case, the name of a prospective normal control child was drawn from the next medical record to occur chronologically after that of the hyperactive child. Another 18 adopted normal controls were solicited through an advertisement in the local newspapers. These children met the following criteria: 1) no history of learning or behavior problems; 2) no history of psychotropic medication; 3) no hospitalization for more than one four-day period during the previous 36 months; 4) Conners's Teachers' Hyperactivity Index less than 1.0; 5) Peabody Picture Vocabulary Test (PPVT) IQ at least 85. The subjects in the study were the 176 (88 sets) biological and adoptive parents of these children. The parenting couples had maintained intact relationships from the time of birth or adoption at six months of age or earlier. Adoptions by biological relatives were excluded.

Rating Scales

Shipley Institute of Living Scale. This scale was originally devised as a measure of organic pathology and cognitive deterioration³⁸ but is presently used as a brief screening measure of the subject's current level of intellectual functioning. Several investigators have found correlations between the Shipley Scale and

the full-scale WAIS score to range from .78 to .90.^{30, 39}

Porteus Maze Test. The test was originally developed as a supplement to the Stanford-Binet Intelligence Scale in identifying mental retardation.^{31, 32} More recently, it has been reported to be a valid and reliable measure of planning ability, judgment, impulsiveness, attention, and ability to delay gratification.^{34, 40} In a discriminant analysis of 27 measures frequently used to distinguish between hyperactive and normal control children, Homatidis and Konstantareas¹⁸ found that the Porteus Maze Test was one of only three measures needed to discriminate accurately between their sample of hyperactive and normal control boys. An automated version of the Porteus Maze Test was used in the investigation. The instrument automatically recorded three scores: the number and duration of contacts with the sides of the maze pathway, and the total time to the completion of the maze.

Span of Apprehension. The Span of Apprehension is a task which measures the amount of information that can be simultaneously processed during a brief visual presentation. Based on early signal detection procedures, Estes¹¹ developed a measure that taps the variability of attention span. The task was designed to minimize the effects of memory or motivational influences. The subject is required to make a forced-choice letter recognition response to one of two signal letters (*i.e.*, the letter T or F) upon a tachistoscopic exposure of short duration.^{1, 8} The Span of Apprehension Task has frequently been used in research with schizophrenic children and adults.¹ Neale and his co-workers²⁹ asserted that the reduction in correct detections on the Span task represents a true deficit in attention. Recently, Denton⁸ found differences between hyperactive and normal control

boys on the Span test. In the present study, the target stimuli were embedded in arrays containing either 0, 4, or 8 additional irrelevant letters (i.e., matrices of 1, 5, or 9 letters).

Reaction Time Apparatus. The delayed reaction time task (DRT) has often been used as a measure of attentional processes. The task has been found to discriminate between hyperactive and normal children^{4, 13, 14} as well as to be drug-sensitive^{5, 12} (see Firestone¹³ for a full description). This task results in two dependent measures. The first is of mean reaction time (RT) measured in milliseconds. The second is of unnecessary responses to the various stimuli; these are recorded and designated Extraneous Responses (RTEXT).

Procedures

Following the telephone contact and the family's acceptance of involvement in the study, a Conners's Teachers' Rating Scale was sent to the index child's teacher for completion and return. After the rating scale was returned and an appropriate hyperactivity criterion was achieved, the parents were contacted to arrange an appointment at their family's convenience.

Each hyperactive and normal control index child was tested alone in a small room free of extraneous visual and au-

ditory stimuli. The PPVT was administered in one period of approximately 15 minutes. The parents were tested separately with a battery of five tests. These were presented in random order for each partner, all five in a single session of approximately two hours. All tests were administered in accordance with standard instructions.

RESULTS

Analyses of variance (ANOVAs) significant at .05 or less were followed by tests of simple effects. The analyses revealed significant differences between the groups on the PPVT and the HI, $F(1,84) = 5.21, p < .03$, and $F(1,84) = 757.27, p < .001$ respectively (TABLE 1). The results indicated that the normal control children had higher IQs than the hyperactives and, not surprisingly, the hyperactive children were rated as more hyperactive. There was no difference between the groups on the age factor.

Demographic Variables

The ANOVAs on the demographic variables (TABLE 2) revealed that the adoptive parents as a group were older than the biological parents, $F(1, 168) = 26.92, p < .001$, and that mothers were younger than fathers, $F(1,168)=4.27, p < .04$.

Table 1
MEAN SCORES AND STANDARD DEVIATIONS OF CHILDREN FOR
AGE, IQ, AND CONNERS'S TEACHER HYPERACTIVITY INDEX (CTHI)

VARIABLE	BIOLOGICAL HYPERACTIVE CHILDREN	ADOPTED HYPERACTIVE CHILDREN	BIOLOGICAL NORMAL CONTROL CHILDREN	ADOPTED NORMAL CONTROL CHILDREN
Age	9.55 (1.32)*	9.57 (1.31)	9.53 (1.33)	9.44 (1.43)
PPVT-IQ	111.92 (15.73)	116.00 (11.75)	119.60 (14.49)	121.20 (10.65)
CTHI	1.84 (.30)	1.76 (.36)	.23 (.21)	.25 (.19)

* Standard Deviations are given in parentheses.

Table 2
MOTHERS' AND FATHERS' GROUP MEANS AND STANDARD
DEVIATIONS FOR AGE, IQ, AND LEVEL OF EDUCATION

VARIABLE	BH	AH	BC	AC
Age (yrs)				
Mothers	33.64 (4.63)*	39.89 (6.41)	36.04 (4.70)	38.95 (5.60)
Fathers	36.04 (5.69)	41.83 (6.73)	37.32 (5.22)	40.30 (6.34)
IQ				
Mothers	106.40 (8.89)	113.61 (6.48)	114.72 (6.28)	114.90 (7.51)
Fathers	108.92 (8.87)	115.28 (10.60)	116.32 (7.87)	118.95 (7.18)
Education (yrs)				
Mothers	11.88 (2.26)	13.67 (1.65)	14.23 (2.38)	14.55 (1.91)
Fathers	12.24 (3.56)	15.17 (2.98)	14.97 (2.73)	15.70 (2.99)
Family Income (thousands)	27.85 (12.15)	31.06 (11.34)	30.88 (10.61)	32.75 (12.69)

BH: Biological parents of hyperactive children ($N=25$)

AH: Adoptive parents of hyperactive children ($N=18$)

BC: Biological parents of normal control children ($N=25$)

AC: Adoptive parents of normal control children ($N=20$)

* Standard Deviations are given in parentheses.

The analyses of the IQs revealed a significant Group \times Relationship interaction, $F(1,68)=4.81, p<.03$. Results of the simple main effects analyses indicated that the biological parents of hyperactive children (BH) had lower IQs than the adoptive parents of hyperactive children (AH) and the biological parents of normal control children (BC). No IQ differences were found between the adoptive parents of normal control children (AC) and the AH group or between the AC and BC groups. In addition, there was a significant main effect for Sex, indicating that fathers obtained higher IQ scores than mothers, $F(1,168)=4.01, p<.05$.

The results of the ANOVA for years of education indicated a significant interaction for Group \times Relation, $F(1,168)=4.50, p<.04$, as well as significant main effects for all three main factors. The results of the tests of simple main effects indicated that the BH group completed fewer years of education than the AH and the BC groups. No

difference in years of education was found between the AC and the AH or between the AC and the BC. Furthermore, fathers completed more school than mothers, $F(1,168)=5.58, p<.02$. Correlational analyses were undertaken and several correlations, all low, were found to exist between the demographic and the attention variables, ranging from $r=.17$ to $r=.28$, irrespective of sign. The amount of variance shared in common between any combination of demographic and dependent measure was also low, ranging from $r^2=.03$ to $r^2=.06$. Keppel²⁰ has proposed that "the main criterion for a control variable is a high correlation with the independent variable." Similarly, Winer⁴² has cautioned about the employment of covariates in factorial designs. Because the correlations between the dependent variables and the demographic variables were so low and the shared variance so small, it was decided that analysis of variance would be the preferred procedure.

Dependent Measures

A MANOVA was conducted on the five attention variables resulting in a significant Group effect $F(5,164)=8.03$, $p<.001$; Relation effect $F(5,164)=3.56$, $p<.004$; Sex effect $F(5,164)=2.90$, $p<.02$; and Group \times Relation interaction $F(5,164)=3.77$, $p<.003$. Therefore, $2 \times 2 \times 2$ ANOVAS were undertaken for each attention variable. TABLE 3 contains the scores on the DRT and Span Apprehension tasks.

ANOVAS on the RT achieved significance for Group $F(1,168)=28.61$, $p<.001$; for Relation $F(1,168)=8.24$, $p<.01$; and for Sex $F(1,168)=11.88$, $p<.001$; and on the Group \times Relation interaction $F(1,168)=9.88$, $p<.002$. The tests of simple main effects revealed the BH had slower mean reaction time than

the AH and the BC. No differences in reaction time were found between the AC and the BC or between the AC and the AH groups (FIGURE 1). In addition, mothers had slower mean reaction times than fathers. The analyses of the RTEXT and Span Size 1 yielded no statistically significant effects.

The ANOVA on Span Size 5 (FIGURE 2) indicated statistically significant differences for Group $F(1,168)=4.21$, $p<.04$ and on the Group \times Relation interaction $F(1,168)=6.44$, $p<.01$. Tests for simple main effects revealed that the BH had fewer correct recognitions than the BC and the AH. The AC were not different from the BC and the AH.

The Span Size 9 ANOVA (FIGURE 3) produced a significant Group effect $F(1,168)=13.32$, $p<.001$; a significant

Table 3
MOTHERS AND FATHERS GROUP MEANS AND STANDARD DEVIATIONS FOR THE FIVE ATTENTION VARIABLES

VARIABLE	BH	AH	BC	AC
RT				
Mothers	367.50 (59.04)*	306.92 (40.56)	306.08 (54.72)	295.19 (44.47)
Fathers	328.57 (65.07)	298.14 (38.26)	270.62 (33.18)	284.21 (38.71)
RTEXT				
Mothers	2.92 (2.78)	2.22 (1.83)	2.04 (2.13)	2.20 (2.29)
Fathers	2.56 (2.57)	2.50 (2.36)	2.24 (2.07)	2.35 (1.93)
Span Size 1				
Mothers	29.96 (.20)	29.89 (.32)	30.00 (.00)	29.90 (.31)
Fathers	29.96 (.20)	30.00 (.00)	29.96 (.20)	29.95 (.22)
Span Size 5				
Mothers	29.28 (.94)	29.83 (.38)	29.76 (.60)	29.70 (.80)
Fathers	29.28 (1.28)	29.78 (.55)	29.84 (.63)	29.70 (.80)
Span Size 9				
Mothers	27.36 (1.58)	28.61 (.98)	28.76 (.93)	28.30 (1.17)
Fathers	26.96 (1.97)	28.28 (1.57)	28.32 (1.46)	28.60 (1.35)

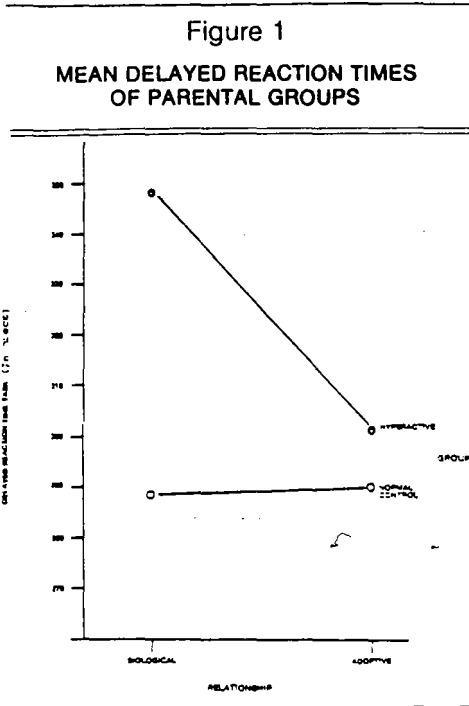
BH: Biological parents of hyperactive children ($N=25$)

AH: Adoptive parents of hyperactive children ($N=18$)

BC: Biological parents of normal control children ($N=25$)

AC: Adoptive parents of normal control children ($N=20$)

* Standard Deviations are given in parentheses.



Relation effect $F(1,168)=7.02, p<.01$; and a significant Group \times Relation interaction $F(1,168)=9.96, p<.002$. Results of the tests of simple main effects indicated that the BH had fewer correct recognitions on Span Size 9 than the BC and the AH. No differences in correct recognitions on Span Size 9 were found between the AH and the AC or between the AC and the BC.

A MANOVA conducted on the impulsivity variables of the Porteus Maze Test proved to be nonsignificant and consequently no further analyses were conducted.

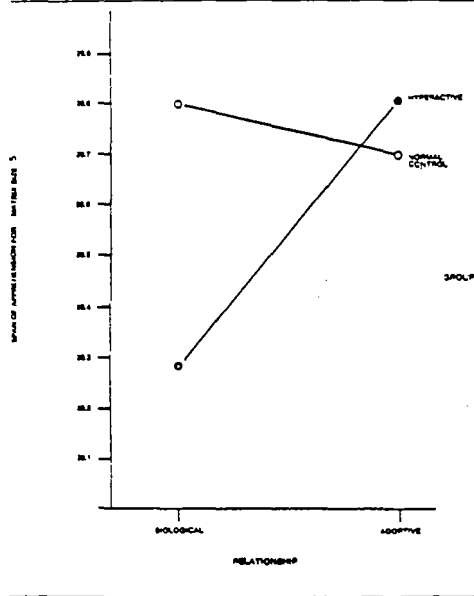
DISCUSSION

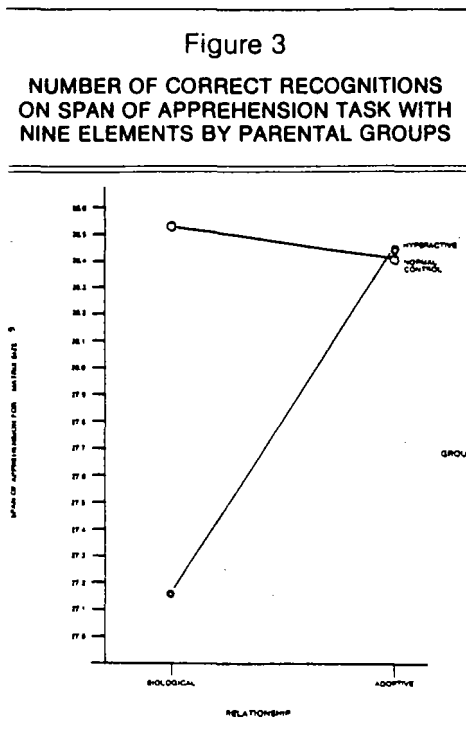
The results of the present study provide strong evidence for a familial association between childhood hyperactivity and attentional deficits in the biological parents of hyperactive children. As anticipated, the biological parents of hyperactive children exhibited more attentional difficulties than

the other groups of parents as reflected by slower mean reaction times on the DRT task and fewer correct recognitions with increasing matrix size on the Span of Apprehension Task. In addition, it was confirmed that the adoptive parents of normal controls did not differ from the adoptive parents of hyperactives or the biological parents of controls on these attention measures. The hypothesis of greater impulsivity, as measured by the Maze test, in the biological parents of hyperactive children was not confirmed.

The demographic variables also yielded interesting findings: The biological parents of children were found to be significantly younger than the adoptive parents. Thus may be explained by the fact that adoptive parents often have spent time trying to conceive their own child and then have to undergo a lengthy screening process by adoption

Figure 2
NUMBER OF CORRECT RECOGNITIONS
ON SPAN OF APPREHENSION TASK WITH
FIVE ELEMENTS BY PARENTAL GROUPS





agencies. Mothers in general were also found to be younger than fathers and this has been reported in family studies by other workers.^{24, 28}

The biological parents of hyperactive children obtained significantly lower scores on intellectual functioning and completed fewer years of education than the other parental groups. Level of education in this study was found to correlate highly and positively with the Paulson-Lin IQ estimates of the Shipley Institute of Living Scale ($r = .62$, $r^2 = .38$). The high degree of interdependence suggests that these two demographic variables are probably measuring aspects of the same factor or factors. The total sample of parents was found to obtain an above average, relatively narrow range of scores of intellectual functioning. While the scores of the biological parents of hyperactive children fell at the upper end of the average range of intelligence, the parents in the

three comparison groups obtained scores in the high average range of intelligence. Although no significant difference was found among the groups with respect to total family income, the biological parents appeared to have a somewhat lower family income than the other parent groups. It is noteworthy, however, that the mean (\$27,850) of the biological parents of hyperactive children is comparable to the combined family income (\$28,290) for the Ottawa-Carleton metropolitan area reported by Statistics Canada (Bulletin Number 13207, 1980). The IQ and educational differences reported in this study may be explained in terms of the different sample solicitation procedures employed, as well by type of parental relationship and differences in utilizing medical facilities. Miller and Keirn²⁴ reported similar findings in educational levels of parents of mentally retarded and emotionally disturbed children compared with parents of non-clinic control children.

Comparing the accepting and the invited samples who met the previously defined inclusion criteria, 25 of the 32 (approximately 78%) invited biological parents of hyperactive children (drawn exclusively from an assessment and treatment program of the CHEO department of psychology) agreed to participate. In contrast, the biological parents of normal controls were selected solely from the medical records department of CHEO; their acceptance rate was 25 out of 65 (approximately 39%) of those invited. A high proportion of adoptive families participated in the study: 18 of the 20 (90%) adoptive families of hyperactives and 20 of the 33 (61%) adoptive families of normal controls agreed to participate. The adoptive parents of hyperactive children were selected predominantly from the assessment files of the department of psy-

chology (*i.e.*, 17 adoptive families of hyperactives, with one adoptive family selected from response to media advertisement). The adoptive parents of control children were mainly selected from response to media advertisement (*i.e.*, 18 adoptive control families, with two adoptive controls selected from the medical records department. It may be that of families with non-clinic children contacted to participate in a psychological study, greater curiosity and voluntary involvement was shown by the control group population who had completed more formal education. In the adoptive population, parents who have made a personal commitment and emotional investment in adoption and parents who already have close involvement in a treatment program for their child may be more motivated to participate voluntarily in a study. Adoption agencies may also tend to place children with couples who are older as well as with those who have more education. The sex differences with respect to education and IQ may be linked with a systematic bias due to assortative mating whereby husbands are not only older but more educated. Another explanation of the mothers' lower IQ scores may be their lesser education and test-taking experience.

In general, there is a concern about the effect of pre-test family differences with respect to IQ and educational level in the groups. ANOVAs were undertaken in view of the factorial design of the study. As a point of interest, however, analyses of covariance were performed on the dependent measures, incorporating years of education and IQ as covariates. The separate ANACOVAs yielded similar results, at the .05 level of confidence based on a two-tailed test, insofar as the biological parents of hyperactives exhibited slower mean reaction times on the DRT task and

made fewer correct recognitions on matrices of five and nine letters on the Span task. However, it is presently unclear as to whether the IQ and educational differences may be the cause or the outcome of the attentional finding. Other studies have reported that hyperactive children obtain lower IQ scores than control children.³⁶ Minde and co-workers³⁴ reported that although hyperactive adolescents did not differ from controls with respect to IQ scores derived from individually administered tests, the hyperactives had lower scores on a group IQ test, suggesting no differences in intellectual functioning *per se*. Furthermore, this suggests that hyperactive subjects may do better in one-to-one, interactive situations where attentional processes may be better monitored. This, then, is a possible explanation of the poorer performances of the biological parents of hyperactive children in the present study on an independent, form-administered intellectual test with little active monitoring of attentional processes. Follow-up studies have suggested that the persistence of attentional and stimulus-processing deficits may, in part, explain the poorer academic classroom functioning of hyperactive adolescents.

The performance of the parents of hyperactive children on the measures of attention duplicate in essence the findings of numerous studies with hyperactive children. On three of four measures of attention, the biological parents of the hyperactives performed significantly more poorly than the other parents. They evidenced 15% slower mean reaction times on the DRT task and achieved fewer correct recognitions on the Span tasks for matrix sizes of five and nine letters, although not for that of one letter. (Interestingly, the mothers in the present study were found to be consistently slower than the fathers, suggest-

ing sex differences in reaction time.) Their extraneous responses on the DRT task and on the Porteus maze test, which are often seen as measures of impulsivity, were not significantly different from other parents' responses, although the results were in the expected direction. This may be reflective of the poor power of these tests or may indicate that impulsivity in the parents is not problematic. It is also possible that impulse control may be moderated by a variety of learning and maturation factors. Certainly, *DSM-III* finds attention to be central to the problem of hyperactive children and perhaps it is this deficit that persists into adulthood.

The data presented here provide support for an association between childhood hyperactivity and attentional deficits in the biological parents of hyperactives. The argument for a familial association is further strengthened by the fact that the attention findings were consistently demonstrated across two separate types of attention measures, both protracted vigilance and visual search strategies. Although highly suggestive, our data nevertheless raise questions about the specificity of the relationship between hyperactivity and attentional deficits in the parents and about the methodological aspects of the research design. In general, the specificity of the attentional relationship will require a more detailed study of these parental characteristics in terms of cross-group comparisons involving non-hyperactive psychiatric index groups as well as parents of normal controls. Cross-fostering studies involving the biological and adoptive parents of the same hyperactive child would assuredly provide a more definitive analysis of the gene-environment interaction.

REFERENCES

1. ASARNOW, R.F. AND MACCRIMMON, D.J. 1981. Span of apprehension deficits during the postpsychotic stages of schizophrenia: a replication and extension. *Arch. Gen. Psychiat.* 38:1006-1011.
2. CANTWELL, D.P. 1975. Genetic studies of hyperactive children: psychiatric illness in biologic and adopting parents. *In Genetic Research in Psychiatry*, R. Fieve, D. Rosenthal and H. Brill, eds. Johns Hopkins University Press, Baltimore.
3. CANTWELL, D.P. 1972. Psychiatric illness in the families of hyperactive children. *Arch. Gen. Psychiat.* 27:414-417.
4. COHEN, N.J. AND DOUGLAS, V.I. 1972. Characteristics of the orienting response in hyperactive and normal control children. *Psychophysiology* 9:238-245.
5. COHEN, N.J., DOUGLAS, V.J. AND MORGENSTERN, G. 1971. The effect of methylphenidate on attentive behavior and autonomic activity in hyperactive children. *Psychopharmacologia* 22:282-294.
6. COHEN, N.J., WEISS, G. AND MINDE, K. 1972. Cognitive styles in adolescents previously diagnosed as hyperactive. *J. Child Psychol. Psychiat.* 13:203-209.
7. CONNERS, C.K. 1970. Symptom patterns in hyperkinetic, neurotic and normal children. *Child Develpm.* 41:667-682.
8. DENTON, C.L. AND MCINTYRE, C.W. 1978. Span of apprehension in hyperactive boys. *J. Abnorm. Child Psychol.* 6:19-24.
9. DOUGLAS, V.I. 1974. Differences between normal and hyperkinetic children. *In Clinical Use of Stimulant Drugs in Children*, C.K. Conners, ed. Excerpta Medica, The Hague.
10. DUBEY, D.R. 1976. Organic factors in hyperkinesis: a critical review. *Amer. J. Orthopsychiat.* 46:353-366.
11. ESTES, W.K. 1965. A technique for assessing variability of perceptual span. *Proc. Nat. Acad. Sci.* 54:403-407.
12. FIRESTONE, P. ET AL. 1978. The effects of caffeine and methylphenidate on hyperactive children. *J. Amer. Acad. Child Psychiat.* 7:445-456.
13. FIRESTONE, P. AND DOUGLAS, V.I. 1975. The effects of reward and punishment on reaction times and autonomic activity in hyperactive and normal children. *J. Abnorm. Child Psychol.* 3:201-215.
14. FIRESTONE, P. AND MARTIN, J.E. 1979. An analysis of the hyperactive syndrome: a comparison of hyperactive, behavior problem, asthmatic, and normal children. *J. Abnorm. Child Psychol.* 7:261-273.
15. HARTCOLLIS, P. 1968. The syndrome of minimal brain dysfunction in young adult patients. *Bull. Menn. Clin.* 32:102-114.
16. HECHTMAN, L. AND WEISS, G. 1983. Long-term outcome of hyperactive children. *Amer. J. Orthopsychiat.* 53:532-541.
17. HECHTMAN, L. ET AL. 1979. Hyperactive children in young adulthood: a controlled, pros-

- pective ten-year follow-up. *Inter. J. Ment. Hlth* 8:52-66.
18. HOMATIDIS, S. AND KONSTANTAREAS, M.M. 1981. Assessment of hyperactivity: isolating measure of high discriminant ability. *J. Consult. Clin. Psychol.* 49:533-541.
 19. HOY, E. ET AL. 1978. The hyperactive child at adolescence: cognitive, emotional, and social functioning. *J. Abnorm. Child Psychol.* 6:311-324.
 20. KEPPEL, G. 1973. *Design and Analysis: A Researcher's Handbook*. Prentice-Hall, Englewood Cliffs, N. J.
 21. LANGHORNE, J. ET AL. 1976. Childhood hyperkinesis: a return to the source. *J. Abnorm. Psychiat.* 85:201-209.
 22. LOPEZ, R.E. 1965. Hyperactivity in twins. *Canad. Psychiat. Assoc. J.* 10:421-426.
 23. MCMAHON, R.C. 1980. Genetic etiology in the hyperactive child syndrome: a critical review. *Amer. J. Orthopsychiat.* 50:145-150.
 24. MILLER, W.H. AND KEIRN, W.C. 1978. Personality measurement in parents of retarded and emotionally disturbed children: a replication. *J. Clin. Psychol.* 34:686-690.
 25. MINDE, K., WEISS, G. AND MENDELSON, N. 1972. A five-year follow-up study of 91 hyperactive school children. *J. Amer. Acad. Child Psychiat.* 11:595-610.
 26. MORRISON, J.R. 1980. Adult psychiatric disorders in parents of hyperactive children. *Amer. J. Psychiat.* 137:825-827.
 27. MORRISON, J.R. AND STEWART, M.A. 1971. A family study of the hyperactive child syndrome. *Biol. Psychiat.* 3:189-195.
 28. MORRISON, J.R. AND STEWART, M.A. 1973. The psychiatric status of the legal families of adopted hyperactive children. *Arch. Gen. Psychiat.* 28:888-891.
 29. NEALE, J.M. ET AL. 1969. Span of apprehension in acute schizophrenia. *J. Abnorm. Psychol.* 74:593-596.
 30. PAULSON, M.J. AND LIN, T. 1970. Predicting WAIS IQ from Ship-Hartford scores. *J. Clin. Psychol.* 26:453-461.
 31. PORTEUS, S.D. 1933. *The Maze Test and Mental Differences*. Smith Publishers, Vineland, N.J.
 32. PORTEUS, S.D. 1939. The validity of the Porteus Maze Test. *J. Ed. Psychol.* 30:172-178.
 33. QUITKIN, F. AND KLEIN, D.F. 1969. Two behavioral syndromes in young adults related to possible minimal brain dysfunction. *J. Psychiat. Res.* 7:131-142.
 34. RIDDLE, M. AND ROBERTS, A.H. 1974. *The Porteus Mazes: a critical evaluation*. Report No. PR-74-3, University of Minnesota, Department of Psychiatry, Research Laboratories Minneapolis.
 35. ROSS, A.O. 1974. *Psychological Disorders of Children*. McGraw-Hill, New York.
 36. ROSS, D.M. AND ROSS, S.A. 1976. *Hyperactivity: Research, Theory, and Action*. John Wiley, New York.
 37. SAFER, D.J. 1973. A familial factor in minimal brain dysfunction. *Behav. Genet.* 3:175-186.
 38. SHIPLEY, W.C. 1940. A self-administering scale for measuring intellectual impairment and deterioration. *J. Psychol.* 9:371-377.
 39. SINES, L.K. AND SIMMONS, H. 1959. The Shipley-Hartford Scale and the Doppelt Short Form as estimators of WAIS IQ in a state hospital population. *J. Clin. Psychol.* 14:452-453.
 40. SROUFE, L.A. 1975. Drug treatment of children with behavior problems. *In Review of Child Development Research*, Vol. 4, F.D. Horowitz, ed. University of Chicago Press, Chicago.
 41. WEISS, G. ET AL. 1979. Hyperactives as young adults. *Arch. Gen. Psychiat.* 36:675-681.
 42. WINER, B.V. 1971. *Statistical Principles in Experimental Design*. McGraw-Hill, New York.
 43. WHALEN, C.K. AND HENKER, B. 1980. *Hyperactive Children: The Social Ecology of Identification and Treatment*. Academic Press, New York.