CANADIAN THESES ON MICROFICHE

I.S.B.N.

THESES CANADIENNES SUR MICROFICHE

National Library of Canada
Collections Development Branch

Canadian Theses on
Microfiche Service

Ottawa, Canada
K1A 0N4

Bibliothèque nationale du Canada
Direction du développement des collections

Service des thèses canadiennes
sur microfiche

NOTICE

The quality of this microfiche is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us a poor photocopy.

Previously copyrighted materials (journal articles, published tests, etc.) are not filmed.

Reproduction in full or in part of this film is governed by the Canadian Copyright Act, R.S.C. 1970, c. C-30. Please read the authorization forms which accompany this thesis.

THIS DISSERTATION
HAS BEEN MICROFILMED
EXACTLY AS RECEIVED

AVIS

La qualité de cette microfiche dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de mauvaise qualité.

Les documents qui font déjà l'objet d'un droit d'auteur (articles de revue, examens publiés, etc.) ne sont pas microfilmés.

La reproduction, même partielle, de ce microfilm est soumise à la Loi canadienne sur le droit d'auteur, SRC 1970, c. C-30. Veuillez prendre connaissance des formules d'autorisation qui accompagnent cette thèse.

LA THÈSE A ÉTÉ
MICROFILMÉE TELLE QUE
NOUS L'AVONS REÇUE

NL-339 (r. 82/08)

Canada

Attention, Impulsivity and Personality Characteristics of the Biological and Adoptive Parents of Hyperactive and Normal Control Children

Jo Elaine Alberts-Corush

Thesis presented to the School of Graduate Studies of the University of Ottawa as partial fulfillment of the requirements for the degree of Doctor of Philosophy

Ottawa, Canada, 1983

ABSTRACT

The present study was conducted to investigate specific attention, impulsivity, and personality characteristics in the biological parents of hyperactive children. In particular, the study examined objective evidence for the hypotheses proposed by Cantwell (1977) and Morrison and Stewart (1973b) that hyperactivity is a familial, genetically linked disorder marked by the presence of personality disturbances in terms of alcoholism, hysteria, and sociopathy in the biological parents of hyperactive children. The study also sought to provide for cross-group comparisons between the parents of hyperactive and normal control children. In order to further facilitate examination of gene-environment influences, the study provided for comparisons between biological and adoptive parents as well as for comparisons between mothers and fathers. The subjects for the study were the 176 parents of 43 hyperactive and 45 normal control boys. Attention, impulsivity, and personality data were obtained for each parent via the administration of a series of behavioural and self-report measures. Results of the analyses indicated that the biological parents of hyperactive children experience more attentional difficulties than the adoptive parents of hyperactives and the biological parents of controls, suggesting a familial association between childhood hyperactivity and attentional deficits in the biological parents of hyperactives.

No evidence was found for the prediction that the biological parents of hyperactives would differ from the comparison groups of parents with respect to impulsivity. It was concluded that attentional difficulties, in contrast to impulse control problems, may represent a more unique and persistent deficit. The results of the personality analyses suggest a complex interaction of both genetic and environmental factors. Biological parents of hyperactive children evidence a higher overall level of psychopathology and are at greater risk for alcohol dependency than the comparison groups of parents. Contrary to the predictions based on the genetic model, the present findings indicate that environmental factors are associated with lower ego strength in the parents of hyperactives and compared to the parents of normal controls. The biological and adoptive mothers of hyperactives, in contrast to the mothers of controls, may be particularly susceptible to psychological stresses attendant to raising a child with long-standing behavioural problems. Issues related to the implications of the findings and future directions for research were discussed.

ACKNOWLEDGMENTS

This thesis was prepared under the supervision of Firestone, Ph.D., Professor of the School of Psychology, at the University of Ottawa. Dr. Firestone's interest, support, and advice were most appreciated in the early formulations of the research project as well as in the subsequent revision of the manuscript.

The writer is also grateful to Associate Professor J. Paul Dionne, Ph.D., and Professor James Carlson, Ph.D., of the Faculty of Education, for the valuable statistical consultations; to Mr. Ed Achorn for his help with the research apparatus; to Mrs. Ann Hurd for assisting in computer programming; to the Medical Records Department staff, C.H.E.O., who aided in the selection of index case files; to fellow student, Danielle Nahon, who assisted in the data collection; and to all of the families who volunteered and made this present study possible.

Finally, deep gratitude is expressed to my husband, Jerrold Corush, for his continued gentle support and his critical yet encouraging review of this project and for the sacrifices it entailed.

CURRICULUM STUDORIUM

Jo E. Alberts-Corush was born February 2, 1946, in Urbana, Illinois, U.S.A. She received her Bachelor of Science degree from the University of Illinois, Urbana, Illinois, in 1968. She received her Master of Science degree also from the University of Illinois in 1969.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

STATEMENT OF THE PROBLEM

Clinicians have experienced recurrent problems with the diagnosis and treatment of hyperactive children. This study seeks to investigate objective evidence for the presence of specific characteristics in the biological parents of hyperactive children. The existence of such parental characteristics would be of theoretical as well as applied significance to the diagnostic and treatment process. Theoretically, objective evidence for specific differences in the characteristics of the biological parents of hyperactive children would be of assistance in further delineating the association between genetic-environmental influences and the behavioural manifestation of hyperactivity (HA). In addition, the presence of specific parental differences might provide relevant information for clinicians insofar as different counselling or treatment approaches are required to meet the adjustment characteristics of each particular family.

Interest in the etiological relationship between the characteristics of parents and the hyperactive behaviour of their children has been heightened over the past decade by the lack of clear-cut evidence supporting other causative

hypotheses. Recent work has suggested that HA stems from a complex interaction of both genetic variation and environmental variation. Although the family, twin, and adoption evidence is limited, it has indicated that HA is a familial disorder transmitted from one generation to the next. More specifically, it has been argued that childhood HA is linked with adult psychiatric illness, notably, sociopathy, hysteria, and alcoholism (Cantwell, 1977; Morrison & Stewart, 1974).

The available evidence suggests that research designs for the investigation of gene-environment interaction may hold great promise for facilitation of the diagnostic and rehabilitative process for hyperactive children (DeFries & Plomin, 1978; Shields, 1977). However, the family study literature to date has been subject to serious methodological and design problems, thus confounding the interpretation of the findings. At present, there is a lack of objective data about the personality and other specific characteristics of the biological parents of hyperactive children.

The selection of a research design to investigate genetic and environmental influences is a primary consideration in current family research. It is important that the design provide as much control over extraneous sources of variance as is possible. The independent variables specified for the present investigation were chosen to employ controls for the type of parental relationship (biological or

adoptive), to compare parents of children in different index groups (hyperactive or normal control), and to control for the sex of the parent (male or female).

In addition to the design issue, the designation of dependent measures is a central concern. The dependent variables selected should include a variety of personality, behavioural, and demographic characteristics in order to increase the possibility of delineating both genetic and environmental influences. The dependent variables should also be consistent in nature with those previously employed in family investigations. At the same time, however, the variables should provide more objective data via standardized and mechanized means in order to effectively facilitate comparison with the clinically derived observations and structured interview data presented in prior studies.

Attentional dysfunction and impulse control problems have long been considered primary characteristics of HA. In fact, the category of Hyperkinetic Reaction as a Behaviour Disorder of Childhood and Adolescence in the DSM-II has been changed to Attention Deficit Disorder with Hyperactivity in the new Diagnostic and Statistical Manual of Mental Disorders (DSM-III) (1980) of the American Psychiatric Association. The three diagnostic criteria specific to Attention Deficit Disorder with Hyperactivity include the presence of developmentally inappropriate

inattention, impulsivity, and hyperactivity.

Insofar as the chasm between gene action and the behavioural classification of HA may be mediated by a complex and intricate interaction of organic, genetic, socio-familial, and psychogenic factors, it is important to consider alternative means of delineating the behavioural phenotypes juxtaposed to the genotype. Kidd and Matthysee (1978) have suggested that biochemical and physiological methods may provide a useful means of bridging the gap and resolving the heterogeneity problem. Attention and impulsivity measured at behavioural and neurophysiological levels may provide a useful means of defining the variation at a genetic locus and the concomitant phenotypic expression of HA.

HA merits consideration as one of the more important areas of child psychopathology in terms of its high prevalence in child guidance clinic populations and its likely precursor role in the development of psychiatric pathology in adulthood. In view of the aforementioned concerns and issues, it is apropos to design a study which will:

1) provide objective data concerning the specific attention, impulsivity, and personality characteristics of parents of hyperactive children. More objective self-report and behavioural measures would provide information needed to accept or refute the retrospective and often subjective findings reported in previous family studies of HA as well as to increase the possibility of delineating

specific familial characteristics associated with HA.

2) provide comparison between biological and adoptive parents of hyperactive children.

3) provide a cross-group comparison between parents of hyperactive and normal control children.

Hypotheses

The family study literature to date suggests that genetic and environmental factors uniquely interface with the hyperactive syndrome. It was hypothesized that the biological parents of hyperactive children would evidence more attentional deficits, impulsivity, and personality disturbances than the adoptive parents of hyperactive children, the biological parents of normal control children, and the adoptive parents of normal control children. More specifically, the hypotheses can now be stated in the null form.

Null Hypotheses

1. There is no Group X Relationship interaction effect in the various attention measures.

2. There is no Group X Relationship interaction effect in the various impulsivity measures.

3. There is no Group X Relationship interaction effect in the various personality measures.

Predictions

(Genetic Hypotheses)

1A. There will be a Group X Relationship interaction effect in the various attention measures. More specifically, the biological parents of hyperactive children will exhibit more

attentional difficulties than the adoptive parents of hyperactive children and the biological parents of normal control children, as reflected by fewer correct recognitions on the various matrix sizes of the Span of Apprehension Task and by slower mean reaction times, and by more extraneous responses on the Delayed Reaction Time Task. The adoptive parents of normal control children will not differ from the adoptive parents of hyperactive children and the biological parents of normal control children on the various attention variables.

2A. There will be a Group X Relationship interaction effect in the various impulsivity measures. More specifically, the biological parents of hyperactive children will exhibit more impulsivity than the adoptive parents of hyperactive children and the biological parents of normal control children, as indicated by a higher number and longer duration of contacts on the Porteus Maze Test as well as a shorter total time to completion on the test. The adoptive parents of normal control children will not differ from the adoptive parents of hyperactive children and the biological parents of normal control children on the various impulsivity variables.

3A. There will be a Group X Relationship interaction effect in the various personality measures. More specifically, the biological parents of hyperactive children will exhibit more personality disturbances than the adoptive parents of hyperactive children and the biological parents of normal control children, as indicated by higher elevations on four two-point scales of the Minnesota Multiphasic Personality Inventory, i.e. scales 1-3, 2-4, 2-7, and 4-9, as well as by lower Ego Strength scores, higher Repression-Sensitization scores, and higher Total Pathology Scores. The adoptive parents of normal control children will not differ from the adoptive parents of hyperactive children and the biological parents of normal control children on the various personality variables.

CHAPTER II

REVIEW OF LITERATURE

Hyperactivity (HA) is now thought to be a leading cause of child clinical referrals, accounting for approximately one-third of all diagnosed psychiatric disorders in school age children (Ross, 1974). Studies of the incidence of HA in the general population is variably estimated to range from 3 to 10% (Langhorne, Loney, Paternite & Bechtoldt, 1976).

Although physical overactivity has historically been identified as the major component of the hyperkinetic syndrome or HA, several recent studies indicate that two related difficulties of short attention span and impulsivity are fundamental characteristics of childhood HA (Denton & McIntyre, 1978; Douglas, 1974; Dykman, Ackerman & Oglesby, 1979; Sroufe, 1975). The recent diagnostic category revisions in the Diagnostic and Statistical Manual of Mental Disorders, DSM-III, (1980) indicate a shift in viewpoint from overactivity to a disorder of attention as the more central factor in the disorder. Sykes, Douglas and Morgenstern (1973), for instance, found that hyperactive children differed from normal control children on sustained attention tasks. The investigators concluded

that the hyperactive children exhibited a lack of inhibitory control as well as an inability to maintain attention on extended tasks. In a cross-group comparison study of hyperactive, behaviour problem, asthmatic, and normal control children, Firestone and Martin (1979) reported that an attentional deficit was the only diagnostic characteristic distinguishing the hyperactive children from the other disordered children and the normal controls.

Despite the apparent clarity of these clinical criteria, researchers have tended to regard HA as a poorly delineated, amorphous construct due to the diversity of clinical descriptions, lack of operational diagnostic systems, and problems of associating the disorder with specific etiological factors. For instance, Cruickshank (1971) found over 40 different labels listed in the literature to describe the childhood disorder of HA. Furthermore, Clements (1966) noted that more than 100 different behaviours had been variously described in the literature as clinical characteristics of the hyperactive disorder.

The problem of delineating the hyperactive disorder is further hindered by the lack of a well-defined, operational diagnostic system. Loney (1979) has noted that major work is now needed to develop such a system to measure the symptoms of the disorder via particular scales, tests, and procedures.

The inconclusive findings to date with respect to the etiological factors which underlie the disorder further complicate the problem of what constitutes HA. A number of theoretical models have been proposed to account for the disorder. Theories which focus on the neurological and biochemical level assume that factors such as brain activity, neurological damage, arousal mechanisms, and biochemical errors are the primary causes of HA (Clements & Peters, 1962; Wender, 1976). The genetic model assumes that the disorder is due to such factors as constitution, genes, and possibly inherited metabolic deficits (Cantwell, 1977; Morrison & Stewart, 1974; Stewart, 1970). Family interaction and environmental models presuppose that HA is the result of disturbances in the familial environment such as parent-child identification and faulty learning (Battle & Lacey, 1972; Warner, 1976). Although these models and others have stimulated considerable interest and research, no particular theory has been sufficiently supported to warrant the rejection of the others.

An examination of research work in HA has found a shift in attention over the past fifteen years from descriptive studies to studies related to diagnosis and etiology as well as to the treatment and the outcome of the disorder. During this time, there has also been a research shift from viewing hyperactive children as a homogeneous group to considering hyperactives as a heterogeneous group with

respect to etiology, symptom characteristics, response to treatment, and outcome. There is a current need in the study of HA and other childhood psychopathologies to develop multivariate techniques of analysis that can separate etiologies which present similar symptom pictures and to elaborate means of associating apparently different symptom features in some disorders to common causal factors (Chess, 1972).

The primary focus of hyperactive research has generally been the primary school age child. Recently, concern as to the course and long-term prognosis of the disorder into adolescence and adulthood and issues related to genetic and environmental factors associated with HA have served to broaden the area of study of this complex disorder.

Follow-up and Longitudinal Studies

The clinical literature reviewed in the 1950's and 1960's indicated that HA was viewed as a childhood disorder which tended to diminish with age. However, more recent evidence contradicts the previous, clinically-based contentions that HA diminishes or disappears by late childhood or early adolescence. The findings of follow-up and longitudinal studies since 1970 indicate that while the problems of physical over-activity and distractibility evidence some improvement, the attentional deficits and impulse control difficulties endure into adolescence and adulthood (Cohen,

Weiss, & Minde, 1972; Hoy, Weiss, Minde, & Cohen, 1978; Weiss, Hechtman, Perlman, Hopkins, & Wener, 1979).

In addition to the on-going attentional deficits, it is suggested that many of these individuals continue to experience associated problems, such as academic difficulties, social and interpersonal problems, lower self esteem, and tendencies toward more antisocial and delinquent behaviours. These recent follow-up studies indicate that there may be a chronic developmental component to HA.

Follow-up into Adolescence. A number of prospective follow-up studies concerning adolescents who were diagnosed hyperactive during childhood have been undertaken by the Montreal group (Cohen et al., 1972; Hoy et al., 1978; Minde, Lewin, Weiss, Lavigueur, Douglas, & Sykes, 1971; Minde, Weiss, & Mendelson, 1972; Weiss, Kruger, Danielson, & Elman, 1975; Weiss, Minde, Werry, Douglas, & Nemeth, 1971). Weiss and Minde and their associates reported on a series of five year follow-up investigations at the Montreal Children's Hospital involving youngsters who had presented with long-term, generalized HA exhibited at both home and school.

Minde et al. (1972) reported that their sample of hyperactive adolescents repeated more grades and did less well in all academic areas than a matched group of classroom controls. The investigators noted that approximately 57% of the hyperactive sample repeated at least one grade as compared to about 16% of the nonhyperactive control sample.

Similarly, Weiss et al. (1971) found that hyperactives at follow-up had poor academic functioning, with 80% of the adolescents repeating at least one school grade.

Although hyperactives did not differ from controls with respect to IQ scores derived from individually administered tests, the hyperactive adolescents had inferior scores on a group IQ test, indicating no differences in cognitive functioning per se (Minde et al., 1972). Furthermore, this suggests that hyperactive subjects do better in one-to-one situations where attentional processes may be better monitored. Hoy and her colleagues (1978) concluded that their sample of hyperactive children evidenced both sustained-attention and stimulus-processing deficits. The investigators noted that these processing, rather than cognitive, deficits may account for the general finding that hyperactives perform poorly on new acqusition learning tasks whereas they rarely differ from controls on tasks involving overlearned skills. Hoy et al. also speculate that these deficits may explain the lower sociability and self esteem ratings of hyperactives insofar as multidimensional information and stimulus processing is required in social interactions. Cohen et al. (1972) compared adolescent hyperactives and controls on measures of cognitive style and found that hyperactive adolescents tended to be more field dependent and showed more cognitive impulsivity on tasks involving a greater degree of response uncertainty. Thus, the persistence of these central attention

and stimulus-processing deficits may, in part, explain the poorer academic classroom functioning of hyperactive adolescents.

In addition to the on-going attentional and academic difficulties, the hyperactive at adolescence experiences problems in social and interpersonal relationships and emotional well being. Various follow-up studies (Mendelson, Johnson, & Stewart, 1971; Weiss et al., 1971) have found problems with respect to lower self esteem and sadness in from 30 to 62% of their samples of hyperactive children at adolescence. Associated problems of disobediency, peer problems, and family related difficulties have also been reported (Huessy, Metoyer, & Townsend, 1974; Weiss et al., 1971). At follow-up, from 20 to 26% of various samples of hyperactive adolescents have been found to have a history of antisocial behaviours (Ackerman, Dykman, & Peters, 1977; Huessy, et al., 1974; Mendelson, et al., 1971; Weiss, et al., 1971). Additionally, approximately 15% of the adolescent hyperactives in two separate studies had court referrals or had been institutionalized at state correctional facilities (Huessy, et al., 1974; Weiss, et al., 1971).

In relation to prognosis, investigators have noted that the poorly adjusted groups of adolescent hyperactives came from families evidencing higher incidences of psychopathology and poor mother-child relationships (Mendelson, et al., 1971; Weiss, et al., 1971). Recently, Loney, Kramer, and

Milich (1979) have proposed a tentative model for explaining these adolescent outcomes. While neither the primary attention deficits or the effects of treatment appear to predict outcome, secondary symptoms of childhood aggressive behaviour are related to adolescent antisocial behaviour. Thus, Loney and her co-workers suggest that childhood aggressivity is associated with adolescent self esteem deficits and antisocial-delinquent behaviour. The authors conclude that aggression and related 'secondary' symptoms merit further investigation with respect to adolescent and adult outcome.

Follow-up into Adulthood. Much needed prospective follow-up studies on the course of HA into adulthood have recently been undertaken by the Montreal group. The results suggest that the adult outcome for hyperactive subjects may be somewhat more optimistic as compared to the adolescent findings. Although the hyperactives tended to lag slightly behind in academic areas and completed fewer years of education, the hyperactive adults did not differ from controls in terms of job competency as rated by employers and job status (Weiss, Hechtman, Perlman, Hopkins, & Wener, 1979). These findings contradict Borland and Heckman's (1976) comparison study of retrospectively diagnosed hyperactive subjects and their brothers which found that the hyperactives had not met the job status or socioeconomic status of their brothers. These contradictory results may be the result of diagnostic differences and sampling procedures.

Studies involving cognitive style tests indicate continued difficulties in stimulus processing and reflection in hyperactive adults as compared to normal controls (Hopkins, Perlman, Hechtman, Weiss, 1979). Thus, hyperactive adults were more easily distracted by irrelevant stimuli, less accurate in selecting the relevant aspects of complex stimuli, and less able to inhibit incorrect responses than controls.

Compared to controls, hyperactive subjects also continued to have poorer socialization skills, a lower sense of well being, and to experience more anxiety, tension, and hostility (Hechtman, Weiss, Finklestein, Wener, & Benn, 1976; Hechtman, Weiss, Perlman, Hopkins, & Wener, 1979). Although adult hyperactives do not see themselves as more pathological than do normal control subjects (Weiss, Hechtman, & Perlman, 1978), 'impulsive' and 'immature-dependent' personality disorders were diagnosed more often in hyperactive subjects than in controls, approximately 40% versus 22%, respectively (Hechtman, et al., 1979).

Several studies have reported that a higher incidence of symptoms of childhood HA was found in a variety of adult psychiatric disorders. In a recent study, Gomez, Janowsky, Zetin, Huey, & Clopton (1981) found that psychotic and nonpsychotic psychiatric patients reported a higher frequency of target HA symptoms than a matched control sample. Moreover, personality disorder patients have reported more

HA symptoms in childhood than controls (Gomez, et al., 1981, Morrison, 1979; Weiss, et al., 1979). While Morrison (1979) reported no differences between psychiatric and control groups with respect to drug abuse, studies conducted by Eyre, Rounsaville, and Kleber (1982) and Schuckit, Petrich, Chiles (1978) involving alcohol and drug abusers found that from 17 to 22% of their treatment samples reported histories of HA in childhood.

Although the adolescent and adult follow-up studies of HA are subject to a number of methodological and design problems and inconsistencies, there is a growing body of consistent evidence across studies that hyperactive adolescents and adults are subject to a number of subsequent academic and psycho-social problems. There are indications of increased risk of psychopathology in hyperactive adults as well as higher incidences of psychopathology in the families of hyperactive subjects. These findings raise important questions regarding the role that familial and environmental factors may play in the psycho-social outcome in hyperactive teenagers and adults. Furthermore, how does childhood HA relate to the familial characteristics of hyperactive children?

Genetic and Family Studies

Research and clinical interest has heightened over the past fifteen years in examining the relationship of genetic and environmental influences in HA. Genetic research in

psychopathology has traditionally incorporated two primary strategies: chromosomal analyses and empirical risk analyses.

The first strategy of genetic research in psychopathology is molecular and chromosomal analyses. In the only known study to date in this area, Warren, Karduck, Bussaratid, Stewart, and Sly (1971) found no evidence of sex chromosome aneuploidy in a group of 96 hyperactive children. A complete kartotype analysis of 23 children from the original sample revealed no abnormalities in chromosome structure or number. There have been no reported attempts to replicate these findings with a more homogeneous subgrouping of children.. However, as psychiatric disorders and behavioural traits appear to reflect the involvement of several genes and rarely demonstrate clear-cut dominant or recessive effects, some other techniques are required to resolve the heterogeneity and gene-environment problems.

The second, more popular approach to current genetic research in psychopathology is based on empirical risk analyses. Three related sources of evidence involving family risk, twin, and adoption studies have been used to support the contention for the role of genetics in HA.

Family Risk Studies. Family morbidity studies have provided the first body of supportive evidence in demonstrating familial resemblance in HA. The evidence is obtained from data on the incidence of HA and related

psychopathology in the immediate family and the relatives of hyperactive children grouped according to the closeness of the blood relationship. Morrison and Stewart (1971) conducted a study on morbidity risk for HA among first- and second-degree relatives of hyperactives. The investigators compared the families of 59 hyperactive and 41 control children and found that there was a higher prevalence of sociopathy, alcoholism, and hysteria in the mothers and fathers of hyperactives than in the mothers and fathers of controls. In 21 of the 59 (36%) families of hyperactive children at least one parent was found to be alcoholic, sociopathic, or hysteric as compared to only 4 of 41 (10%) control families. Fathers were reported to be more likely affected than mothers. Approximately 20 percent of the parents of hyperactives were self-described as being hyperactive in childhood, whereas only five percent of the parents of controls were so described. The incidence of HA as well as adult disorders was higher in first-order relatives (parents, siblings) of hyperactive children than in second-order relatives (aunts, uncles, cousins), which in turn was higher than the incidence of HA in the normal control sample. Morrison and Stewart proposed that HA is a familially based syndrome passed from one generation to the next. The investigators additionally argued that their findings were consistent with either a genetic or an environmental mode of transmission from parent to child.

In a comparable study, Cantwell (1972) interviewed the parents of 50 hyperactive and 50 control children. The control children were matched to the hyperactive sample for age, sex, race, and social class. Ten percent of the parents of hyperactive children were retrospectively diagnosed as hyperactive in childhood as compared to two percent of the parents of controls. The family history data revealed that the only psychiatric disorders found more frequently among the first-degree relatives of hyperactives than among the first-degree relatives of controls were alcoholism, sociopathy, and hysteria. Based on structured interview data, Cantwell reported that 45 percent of the parents of hyperactive children had some psychiatric diagnosis, whereas only 18 percent of the parents of controls were so diagnosed. More specifically, 33 percent of the parents of hyperactives were diagnosed as being sociopathic, alcoholic, or hysteric as compared to 9 percent of the parents of normal controls. Interestingly, the fathers in both groups were found to be more psychiatrically ill than the mothers. Cantwell asserted that his study demonstrated a familial association in HA. Cantwell also felt that HA in childhood may be a precursor to alcoholism, sociopathy, and hysteria in adulthood.

The family risk results are further supported by the findings of Welner, Welner, Stewart, Palkes, and Wish (1977) that HA is more common among brothers of hyperactive children than among siblings of controls. The study involved the

comparison of 43 hyperactive proband boys with at least one sibling age six or older (total of 89 full siblings; 42 brothers and 47 sisters) and 38 nonhyperactive controls (total of 104 full siblings; 54 brothers and 50 sisters). The children attended public school and were matched for sex, age, and race. Welner and her co-workers reported that 26 percent of the brothers of hyperactive probands and nine percent of the brothers of controls were assessed on the basis of interview data as exhibiting the hyperactive disorder. The male hyperactive probands and their brothers exhibited more symptoms of depression-anxiety than the control males and their respective brothers. No significant differences were found between sisters of hyperactives and controls. Welner and her colleagues concluded that their findings supported the general literature impression that HA tends to run in families. The investigators also observed that HA appears to be related to the male side of the family.

Nichols (1979) conducted an analysis of risk indicators for two symptoms of minimal brain dysfunction, hyperkinetic-impulsive behaviour and learning difficulties. An affected sibling was found to be a better predictor than any of the socio-economic, maternal, pregnancy, birth complication, neonatal, or infancy variables. Hyperkinetic-impulsive behaviour was found more frequently among siblings of more affected probands. In addition, there was a greater incidence of hyperkinetic behaviour in the siblings of hyperkinetic-

impulsive girls, the less frequently affected sex. Nichols concluded that the findings of the siblings and second-degree relatives supported a genetic basis for the familial association. Nichols also argued that the findings were consistent with the polygenetic mode of transmission proposed by Morrison and Stewart (1973a, 1974).

Dubey (1976) noted that a deficiency in family risk research designs had been the absence of studies employing deviant control groups. In addition to demonstrating that specific characteristics occur more frequently in the families of hyperactive children than in the families of normal controls, it is important to demonstrate that these characteristics likewise occur more frequently in the families of hyperactives than in the families of nonhyperactive, disturbed children. The author argued that different groups of disturbed children and their families may evidence a number of similar characteristics. Therefore, Dubey noted the value of examining the specificity of the relationship between the proband and the familial characteristics.

In response to Dubey's (1976) recommendation regarding deviant control groups, Morrison (1980) replicated the two prior family risk studies (Cantwell, 1972; Morrison & Stewart, 1971) to provide comparisons of the family histories of hyperactive children with the family histories of nonhyperactive, psychiatrically ill index children. The 140 hyperactive and 91 psychiatrically ill children and adolescents

had been seen in a general private psychiatric practice and were matched for age and sex. The investigator found that there was a higher incidence of Briquet's syndrome (hysteria) and antisocial personality in the parents of hyperactives than in the parents of psychiatrically ill children. Sixteen (11%) of the hyperactive index cases had at least one parent with either hysteria or antisocial personality as compared to 2 (2%) of the families of disturbed children. More specifically, 6 (4%) of the mothers of hyperactives were diagnosed with hysteria, whereas only 1 (1%) of the mothers of psychiatric probands were so affected. The fathers of hyperactive and psychiatric probands were not found to have hysteria. Two (1%) mothers and nine (7%) fathers of hyperactives obtained a diagnosis of antisocial personality as compared to 1 (1%) mother and no fathers in the comparison group. Although alcoholism occurred slightly more often in the parents of hyperactive children than in the parents of psychiatrically disturbed children, the two proband groups were not different in terms of alcoholism. Nineteen (14%) of the mothers and 30 (22%) of the fathers of hyperactives were diagnosed as alcoholic, while eight (9%) of the mothers and 14 (16%) of the fathers of psychiatrically ill probands were so affected. There was a higher incidence of schizophrenia and schizophreniform psychosis in the parents of psychiatric probands than in the parents of hyperactive children.

Morrison (1980) noted that the findings based on the deviant control group reinforced the results of the previous family risk studies that there is a higher incidence of antisocial personality and hysteria in the parents of hyperactive children than in the families of nonhyperactive, psychiatric children. However, there was no support for a specificity of a relationship between alcoholism and childhood HA. While the rate of alcoholism in parents of hyperactives was similar to those reported in previous studies (Cantwell, 1972; Morrison & Stewart, 1971), the parents of hyperactive children did not differ significantly from the parents of disturbed children in terms of the incidence of alcoholism. Morrison concluded that while his findings did not substantiate a genetic etiology for HA, support was provided for the specificity of a relationship between childhood HA and the two personality disorders of sociopathy and hysteria.

Independent investigators examining other areas of psychopathology have suggested that genetic factors are also involved in adult impulse and personality disorders, such as alcoholism (Goodwin, 1979; Goodwin, Schulsinger, Hermansen Guze, & Winokur, 1975; Schuckit, Goodwin, & Winokur, 1972), sociopathy (Crowe, 1975; Hutchings & Mednick, 1977; Mednick, & Hutchings, 1978; Robins, 1966; Schulsinger, 1972), and hysteria (Brown, 1942; Guze, 1967; Guze, Woodruff, & Clayton, 1971; Oki, 1967). Several family studies have also

demonstrated links between the three adult personality disorders of alcoholism, sociopathy, and hysteria (Arkonac & Guze, 1963; Guze, Wolfgram, McKinney, & Cantwell, 1967; Woerner & Guze, 1968). Furthermore, investigators have retrospectively linked adult disorders of alcoholism and sociopathy with childhood impulsive-antisocial behaviour problems (Barcai & Rabkin, 1974; Cadoret, Cain, & Grove, 1980; Cadoret & Gath, 1978; Nylander, 1960; Quitkin & Klein, 1969; Robins, 1978) and with HA (Goodwin, et al., 1975; Hartocollis, 1968; Morrison & Minkoff, 1975; Tarter, McBride, Buonpane, & Schneider, 1977; West & Farrington, 1973; Wood, Reimherr, Wender, Bliss, & Johnson, 1976). The allied evidence lends additional support to a familial association between HA and alcoholism, sociopathy, and hysteria in adulthood.

The family incidence and risk studies are subject to three criticisms. First, while an association between the degree of blood relationship and the incidence of HA and related disorders has been demonstrated, a familial link does not imply genetic causation. Theorists of other persuasions dispute the genetic interpretation, proposing that the findings of the kinship studies are understandable in terms of socio-psychological factors (Rosenthal, 1971), such as modelling, child-parent identification, and stress factors related to dealing with a family member who is chronically hyperactive. Second, several reviewers (Dubey, 1976;

McMahon, 1980; Rapoport & Ferguson, 1981) have discussed the serious problem of diagnostic bias. The interviewers in the major family studies (Cantwell, 1972; Morrison, 1980; and Morrison & Stewart, 1971) were fully cognizant of the diagnostic status of the index child during the assessment and diagnosis of family members. Third, many of the results are based on retrospective and subjective interview data which is additionally prone to subject recall and bias as well as experimenter bias (McMahon, 1980).

Twin Studies. A second source of evidence for a genetic factor is provided by twin studies. Rates of concordance for HA are computed for the identical monozygotic (MZ) and the fraternal dizygotic (DZ) same-sex twin pairs. Genetic theorists have proposed that the higher concordance rates for MZ twins might be due to the sharing of identical gene properties. The concordance rate for DZ pairs would be predictably lower and akin to non-twin siblings who share approximately half their genes in common.

The results of the only known study of HA in twin pairs have been largely rejected due to serious methodological difficulties. Lopez (1965) conducted a concordance study of HA involving 10 sets of twins (four MZ and six DZ pairs) with an age range of five to 12 years. One member of each pair was previously diagnosed HA based on specified clinical characteristics and the evaluation of a psychiatrist with the consensual agreement of HA by both teacher and mother.

A 100 percent concordance rate for HA was reported for the four pairs of MZ twins as compared to a 17 percent concordance rate for the six pairs of DZ twins. Cantwell (1976) and Omenn (1973) have indicated that the results of the twin study have been confounded by the small sample size and sex differences within the pairs. The use of the physical similarities method of determining zygosity rather than the more rigorous serological tests of blood characteristics also raises questions about the screening procedures used in attempting to accurately identify the type of twin relationship, i.e. MZ or DZ.

In two related studies, Scarr (1966) and Willerman (1973) found a significantly higher correlation on activity level measures among normal control MZ twin pairs in contrast with normal DZ pairs. The investigators interpreted their results as supportive evidence for a heritability component to activity level. Willerman designated the twins (93 sets of same-sex twins) who scored in the upper 20 percent on the activity level measure as 'hyperactive'. However, the two activity level studies involved children who were ostensibly drawn from a normal population of twin pairs and who had not been clinically diagnosed as hyperactive.

Genetic involvement has been suggested via twin studies involving sociopaths and alcoholics. Mednick and Hutchings (1978) summarized concordance rates for criminality reported

in studies of MZ and DZ twins conducted over the past twenty years. The estimated pairwise concordance rates were found to vary from 36 to 60 percent for the MZ twins and from 12 to 30 percent for the DZ twins. The reviewers observed that there were serious problems in sampling due to an inflated proportion of MZ twin pairs as well as geographical bias which thereby confabulate the interpretation of the results.

Kaij (1960) conducted a twin study of alcoholism involving 174 pairs (48 MZ and 126 DZ pairs). Kaij reported concordance rates of 54 percent for MZ probands as compared to 31 percent for DZ proband sets. Jonsson and Nilsson (1968) and Partanen, Bruun, and Markkanen (1966) found that MZ twins were more concordant than DZ twins in terms of amount of alcohol consumed and frequency of drinking. The allied literature on sociopathy and alcoholism suggests a genetic link in terms of antisocial and drinking behaviour.

In general, the interpretation of the twin studies is complicated by the argument that MZ and DZ twins may have different environmental experiences. Cantwell (1976) presents the contention that the higher concordance rates for MZ twins might be due to environmentally created similarities. This position is based on the assumption that MZ twins are treated as being more alike than DZ twins, thus creating more environmental similarities and reducing intrapair differences for the MZ twins. Recent research, however, has failed to support the environmental treatment claims against the twin

method (Loehlin & Nichols, 1976). Environmental theorists might also argue that MZ twins form unique identification bonds which differ from the sibling indentification experienced by DZ twins or non-twin siblings. Finally, many clinicians and researchers argue that the findings of both the family risk and the twin studies can be regarded as supportive evidence for genetic and/or environmental modes of transmission.

Adoption Studies. The third source of supportive evidence is drawn from adoption studies. Adoption studies serve as an attempt to separate genetic and environmental effects by comparing phenotypic similarity for biological families (sharing both heredity and environment) with phenotypic similarity for nonbiological, adoptive families (sharing only environment). Rosenthal (1971) suggests that the use of the nonfamilial adoption method may provide the most effective means of separating genetic from child-rearing factors. In contrast to twin and family morbidity studies, DeFries and Plomin (1978) also propose that adoption studies provide a more persuasive argument for the role of genetic influence.

McMahon (1980) and Shaffer and Greenhill (1979) recommend that decisive evidence for genetic heritability would involve the investigation of the adoptive and the biological parents of the same group of hyperactive children. However, to date, no full adoption studies in HA have been conducted, primarily

due to the legal problems related to contacting the natural parents. Two partial adoption studies (Cantwell, 1975a; Morrison & Stewart, 1973b) have involved comparison of the biological parents of one group of hyperactive children with the nonbiological parents of another group of adopted hyperactive children. Both of the partial adoption studies reported a higher incidence of HA and adult psychiatric disorders (hysteria, alcoholism, and sociopathy) among the parents of hyperactives than among relatives who were not biologically related but with whom the children shared rearing experiences.

Morrison and Stewart (1973b) collected structured interview data on 35 nonbiological families of adopted hyperactive children. The adopted probands had had no contact with their biological parents since one month of age and had been permanently placed with their adoptive parents by two and a half years of age. The psychiatric diagnoses for the 35 adoptive families were compared with the diagnostic prevalence rates reported previously (Morrison & Stewart, 1971) for the 59 biological families of hyperactive children and the 41 biological families of normal controls. The investigators found significantly higher incidences of paternal alcoholism and sociopathy and maternal hysteria in the biological parents of hyperactives as compared to the adoptive parents of hyperactives and the biological parents of normal controls. In particular, they reported that the diagnoses of sociopathy and

hysteria were found exclusively in the biological parents of hyperactive children, thereby clearly differentiating the biological group from the adoptive and control groups. The biological first- and second-degree relatives of hyperactives were found to have higher prevalence rates for alcoholism and retrospectively diagnosed HA than did the adoptive parents of hyperactive children and the biological parents of controls. Morrison and Stewart concluded that their results demonstrated an association of childhood HA with the adult personality disorders of alcoholism, sociopathy, and hysteria in the biological parents of hyperactive children, while such associations were concomitantly not found in the adoptive or control groups. The authors further postulated a polygenic mode of transmission for HA.

Cantwell (1975a) utilized a similar partial adoption method involving 39 nonbiological families of adopted hyperactive children, 50 biological families of another group of hyperactive children, and 50 biological families of control children. Cantwell reported similar main findings including higher incidences of retrospectively diagnosed HA among male relatives, higher rates of paternal alcoholism and sociopathy, and higher rates of maternal hysteria in the biological families of hyperactive children than in the adoptive and normal control comparison groups. The prevalence rates for HA as well as alcoholism, sociopathy, and hysteria found in the adoptive families of hyperactive children were not

different from the rates found in the biological families of controls. Cantwell felt that the higher prevalence rates for the adult personality disorders and familial HA found in the biological families of hyperactives and the concomitant absence of higher rates in the first- and second-degree nonbiological relatives of adopted hyperactives provided a strong argument that HA involves a genetic rather than a purely environmental mode of transmission.

Safer (1973) conducted a sibling study involving the partial adoption method which compared 19 full and 22 half siblings of 17 index children with minimal brain dysfunction. The diagnosis was based on an assessment by a psychiatrist or psychologist, with difficulties noted in learning, attention, and behaviour typical of the clinical characterisitcs. The 17 foster children who served as the index cases were over five years of age and had been placed in foster care prior to eight years of age. In 15 of the 17 cases, the index child had also been described as hyperactive by two trained, independent examiners. Safer found that there was a higher incidence of minimal brain dysfunction in the full siblings of the index children than in their half sibs. Ten of the 19 full sibs and only 2 of the 22 half sibs were diagnosed as evidencing minimal brais dysfunction. In addition, approximately 47 percent of the full siblings as compared to 23 percent of the half sibs obtained a diagnosis of HA. Safer felt that his findings were consistent with a

genetic mode of transmission.

Independent, supportive evidence has also been obtained via the adoption method for the influence of genetic factors in sociopathy and alcoholism. Schulsinger (1972) and Hutchings and Mednick (1977) utilized a cross-fostering strategy for comparing the biological and nonbiological relatives of psychopathic and nonpsychopathic control adoptees and found some evidence for a familial factor in sociopathy. However, the investigators were unable to satisfactorily separate the effects of genetics and environment. Crowe (1975) conducted a partial adoption study involving 41 female inmates who had given their children up for adoption at birth. A control group matched for age, sex, race, and age at adoption was obtained from a state adoption registry. Eight (20%) of the probands and two (5%) of the control offspring had arrest records. Seven (17%) of the probands had been convicted of criminal acts as compared to one (2%) of the control offspring. Additionally, Crowe noted that there was a similarity in the types of crimes of the proband and their biological mother. Crowe felt that the findings suggested a form of specificity in the genetic effect of criminal behaviour.

Goodwin and his co-workers conducted a series of adoption studies examining the offspring of alcoholic parents (Goodwin, et al., 1973; Goodwin, Schulsinger, & Moller, 1974; Goodwin, Schulsinger, Knop, Mednick, & Guze, 1977).

The studies involved nonfamilial adoptions prior to six weeks of age. In summarizing the series of Danish adoption studies, Goodwin (1979) concluded that the offspring of alcoholics were vulnerable to alcoholism whether they were raised by their biological, alcoholic parents or by their nonalcoholic, adoptive parents. The sons of alcoholics were approximately four times more likely to become alcoholic than the sons of nonalcoholics irregardless of their rearing environments. Goodwin also noted that the vulnerability appeared to be limited to and specific for alcoholism insofar as parental alcoholism did not increase the offsprings risk for other psychopathology or for abuse of other substances. Cadoret and Gath (1978) examined environmental variables and family background data utilizing a multiple regression method. Alcoholism in the biological parents was found to be the only significant predictor of alcoholism in the adoptee. Cadoret, Cain, and Grove (1980) recently conducted a study involving adopted male offspring with first-degree alcoholic relatives, adoptees with second-degree (siblings, grandparents) alcoholic relatives, and a control group of adopted males whose biological relatives either had some other psychiatric diagnosis or no diagnosis. Cadoret and his co-workers found that alcoholism in biological first- and second-degree relatives significantly predicted alcoholism in the offspring. None of the environmental variables, such as socioeconomic status, psychiatric or

alcoholic problems in the adoptive family, or discontinuity of mothering, were significant in predicting adoptee alcoholism. The investigators concluded that the findings suggest a genetic, rather than an environmental, basis for alcoholism.

Goodwin and his colleagues (1975) compared alcoholic and nonalcoholic males from a sample of 133 Danish adoptees. The sample included 14 adoptees who were diagnosed alcoholic based on interview data. Retrospective data concerning childhood factors and current demographic and personality characteristics were collected for each adoptee. The investigators found that 10 (71%) of the 14 alcoholic adoptees had biological parents who were alcoholic as compared to no known cases of alcoholism among the biological parents of nonalcoholic adoptees. The alcoholic adoptees also experienced significantly more difficulties than the nonalcoholic adoptees in terms of lower academic performance, truancy, antisocial behaviour, disobedience, and insecurity. Seven (50%) of the 14 alcoholic adoptees described themselves as hyperactive-impulsive during childhood and early adolescence. The investigators felt that the results tended to support a genetic hypothesis for alcoholism and that childhood HA may predispose to alcoholism in adulthood.

Dubey (1975) and McMahon (1980) have discussed several methodological problems which confound the interpretation of the available adoption study results. First, the

identification and selection of hyperactive index cases was based solely on the presence of at least five of the criterion characteristics as outlined by Stewart, Pitts, Craig, and Dieruf (1966) as well as the presence of 'some other' indicative symptoms. Neither Cantwell (1975a) or Morrison and Stewart (1973b) provided data on the reliability of the diagnostic criteria procedures or whether the criteria were adjudged by direct observation of the child, by interview with the parents, by teacher-school reports, or by a combination of these procedures.

The lack of homogeneity of the samples due to either the unreliability of phenotypic diagnosis or the possibility of undetected sub-groups of the disorder has been cited as a central design and methodological issue (Block, 1969; Cantwell, 1975c; Kidd & Matthysee, 1978; Satterfield, Cantwell, Lesser, & Posodin, 1972; Schuckit, et al., 1972). The phenotypic diagnosis of HA for the index children in the adoption studies was apparently based on interview data rather than on standardized behaviour rating scales from teachers and parents or on other measures of attention or activity level. Thus, the selection procedures raise serious questions about the homogeneity of the sample.

Second, both reviewers (Dubey, 1975; McMahon, 1980) cite the problems of subject sampling procedures. The hyperactive proband and his biological relatives were obtained from a public outpatient clinic (Morrison & Stewart,

Safer, 1973) or from a dependents clinic on a military base (Cantwell, 1975a). On the other hand, the adopted hyperactive group were solicited completely (Cantwell, 1975a) or partially (Morrison & Stewart, 1973b) from the private practices of pediatricians or from Social Service records (Safer, 1973). There may be problems created by the incomparability of diagnoses provided by psychiatrists in clinical settings, pediatricians in private practice, and other diagnosticians. The generalizability of the findings are likewise hindered by the fact that the various proband and control groups were not matched on relevant demographic variables which would facilitate comparability of the group results. Morrison and Stewart proposed, for example, that the higher socioeconomic status in the adoptive group may be due to the higher proportion of private patients in that group.

Third, the initial screening procedure for prospective adoptive parents may in itself selectively rule out individuals evidencing the personality disorders under examination. The resultant group of adoptive parents with their higher level of socioeconomic status would likely produce spuriously low incidence rates of the disorders (Dubey, 1976).

Fourth, the interviewers in the three family adoption studies involving hyperactive subjects were not blind to

either the familial relationship (biological-adoptive; full sib-half sib) or to the diagnosis of the identified proband or control child. Although the investigators endeavored to utilize structured interview formats, the issue of examiner bias adversely limits the interpretation of the results.

Fifth, the results are likewise effected by the problem of subject recall and bias. The relatives of the hyperactive probands may have been uniquely sensitized to the interview data in such a way as to respond more unfavorably and selectively than did the controls. The use of more objective self-report and behavioural measures may be indicated (McMahon, 1980).

Mode of Genetic Transmission. Several theories have been posed regarding the genetic mechanisms of transmission in HA. The proposed models include chromosomal anomaly, monogenic dominant and recessive transmission, sex-linkage, polygenic inheritance, polyfactorial transmission and genetic heterogeneity.

The chromosome anomaly model suggests that specific sex chromosome aneuploidy or other chromosome abnormality results in HA. As previously noted, the only reported chromosomal study (Warren, et al., 1971) was inconclusive.

The monogenic theories suggest that only a single aberrant gene from the parent is sufficient to produce HA. If the mechanism is simple autosomal dominant, only a single pathological gene is required from either parent. The parent

contributing the dominant gene also exhibits the trait, which implies that HA would not skip generations. In addition, there would be no sex differences in the incidence of HA in the population, as it would be equally likely for the female or male offspring to inherit the dominant gene. However, the available family risk and incidence literature does not appear to meet the conditions for simple autosomal recessive: each parent would contribute a predisposing recessive gene. Each parent would carry the gene for HA, but the parent would not necessarily be affected. If HA were the result of a recessive gene, h, the contributing parent would be either normal (Hh) and a carrier of HA or the parent would also be hyperactive (hh). The autosomal recessive model suggests that 25 percent of the full sibs of hyperactive probands should exhibit HA. Safer's (1973) study involved a small sample of only 17 index cases of children with minimal brain dysfunction. No other studies involving siblings of diagnosed hyperactive children are available. In addition to the lack of sibling evidence, the recessive gene model is also questioned on the basis of the higher than expected percentage of parents exhibiting HA (Cantwell, 1975b).

The variously reported sex ratios, ranging from 3:1 (Safer & Allen, 1976) to 9:1 (Werry, 1968) of boys to girls with HA, would implicate a sex-linked mode of transmission. However, Morrison and Stewart (1973b) suggest

that the high degree (approximately 10 percent) of apparent transmission from father to son reported in the family risk studies would argue against a sex-linkage model.

The model of polygenic inheritance assumes the involvement of a large number of genes, referred to as polygenes, whose combined effect is additive. Therefore, the more predisposing hyperactive polygenes that a person inherits, the more likely he is to manifest the disorder. The polygenic theory is the most popular and widely accepted model of genetic transmission in the study of psychopathology at present (Rosenthal, 1976). Morrison and Stewart (1973a, 1974) have presented preliminary evidence for a model of polygenic inheritance. The investigators (1973a) found supportive evidence for two conditions of polygenic transmission: families with higher incidences of HA are at risk for related pathological conditions (i.e. alcoholism) and families with more hyperactive polygenes are more at risk for HA. In their second study (1974), the investigators utilized Slater's (1976) method for examining family history data in order to attempt to differentiate between polygenic inheritance and dominant gene transmission with reduced penetrance. Slater's strategy is based on the assumption that autosomal dominance with reduced penetrance is primarily a unilaterally inherited condition, where one would expect secondary cases on one side of the family. The polygenetic view would suggest bilateral inheritance with secondary cases occurring

on both maternal and paternal sides of the family. The investigators found that both sides of the family were so affected, concluding that on the basis of their small sample, polygenetic inheritance was the probable mode of transmission.

The model of polyfactorial transmission of HA proposed by Morrison and Stewart (1973a) and Wender (1971) is similar to Meehl's (1962) model of diathesis-stress interaction posited for schizophrenia. Polyfactorial transmission assumes that a variety of hereditary and environmental influences uniquely interface to produce HA. The underlying genetic mechanism is presumed to be polygenic. The model predicts that if the hyperactive genotype is subjected to certain environmental influences and stresses, such as birth complications, the result will be HA, a hyperactive phenotype. Polyfactorial transmission provides an attractive model for investigation. High risk studies, such as Mednick and Schulsinger's (1968) investigation of schizophrenia, might provide supportive evidence regarding constitutional and stress factors related to the development of childhood HA.

The genetic heterogeneity model has been posited by several investigators who propose that HA is a heterogeneous disorder containing several distinct and, as yet, unclearly delineated sub-groups (Langhorne & Loney, 1979; Lesser, 1970; Loney, Langhorne, & Paternite, 1978; Ney,

1974; Omenn, 1973). Genetic heterogeneity suggests that there are separate genes for each clinical sub-group. The model predicts that the different sub-types of HA may be the result of single genes which are not alleles (one of two or more alternative forms of a gene at the same loci giving rise to contrasting Mendelian characteristics) of one another. Studies identifying and examining hyperactive children who appear to form separate, distinct sub-groups would provide a means of assessing the model. McMahon (1980), for example, has suggested groups based on age of onset and the hyperactive child's response to medication. Loney and her colleagues (1979) have indicated that sub-grouping based on 'secondary' symptoms such as aggressivity may be beneficial in delineating differences within the HA disorder.

Summary

The literature reviewed in the preceding sections provided the framework for the theoretical and practical issues considered in planning the present study. The present review has suggested that there is a longitudinal, developmental component to HA which is evidenced by continued academic and psycho-social problems in adolescence and adulthood. The family risk, twin, and adoption studies over the past decade provide a consistency of findings supporting a familial association in HA. The tentative evidence suggests that childhood HA is linked with adult

psychiatric disorders, notably sociopathy, hysteria, and alcoholism, in the biological parents of hyperactive children. Although the bulk of the family literature supports the role of genetic factors in HA, the interpretation of the findings are guarded due to the various methodological problems previously addressed and the lack of more objective, standardized measures of familial characteristics.

In general, studies seeking to establish the relationship between childhood HA and parental and family pathology, need to consider a multiplicity of factors. The studies must provide for relevant controls of genetic and environmental factors. They must also provide objective, empirical measurement of the familial characteristics under investigation.

CHAPTER III

METHOD

This chapter is divided into six sections. The first section describes the identification and selection of the hyperactive and normal control index cases, i.e. the designated children who possessed the traits under investigation. The second section reports on the subject sample. Section three presents a description of the criterion measures for the index children. The assessment measures for the parents are presented in the fourth section. The procedures and ethical considerations are discussed in the fifth and sixth section respectively.

Index Cases

The index cases (i.e. the identified child or proband who possessed the traits under investigation and who served as the starting point of the family study) were 88 male children between 6 and 12 years of age. Of the 88 selected index cases, 69 of the probands were referred to the out-patient services at the Children's Hospital of Eastern Ontario (C.H.E.O.), and 19 were identified in response to media advertisements. The hyperactive children were selected at random from the assessment files of the Department of Psychology, C.H.E.O. After the families of

the hyperactive children accepted participation in the study, the hyperactive child's birthdate was used to locate prospective normal control children from the medical record files, C.H.E.O. The names of potential control families were drawn from the file chronologically following the selected hyperactive index case. The selection procedures for the hyperactive and normal control index children are discussed more fully later in this section.

All probands achieved Peabody Picture Vocabulary Test (PPVT) IQ equivalents of 85 or higher and regularly attended school. Each child came from an intact family with both parents living at home. In the adopted groups, the child was adopted prior to six months of age. Children evidencing psychosis, brain damage and epilepsy, as noted in the medical record histories or in the initial telephone contact with the family, were excluded from the study.

The average age for all probands was 9.52 (SD ± 1.33) with an age range of 6.83 to 11.67 years. The average PPVT IQs for all index cases was 117.05 (SD ± 13.84) with a range of 86 to 145 (Table 1). A subsequent analysis of variance on these two factors (Appendix A) indicated no age difference between the four index groups. However, there was a significant Group main effect for the PPVT IQs, $F(1,84) = 5.21$, $p < .03$, indicating that the normal control index cases produced higher IQ scores than the hyperactive

Table 1

Mean Scores and Standard Deviations of Children for Age, IQ and Conner's Teacher Hyperactivity Index (CTHI)

	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 Deviation

index cases. The average PPVT IQs for the normal control probands was 120.31 (SD ± 12.78) with an IQ range of 86 to 143; the average IQs for the hyperactive probands was 113.63 (SD ± 14.06) with a range of 93 to 145. The Relationship main effect and the Group X Relationship interaction proved to be non-significant for the PPVT IQs. There was no difference between the biological and adopted children in terms of PPVT IQ scores.

Hyperactive Index Children. Forty-three hyperactive probands (i.e. 25 biological hyperactive index cases and 18 adopted hyperactive index cases) and their families accepted participation in the study. In order to be invited to participate as a hyperactive proband, the child had to satisfy the following criteria:

1) the child was referred to the Department of Psychology, C.H.E.O. with a suspected diagnosis of HA made by the referring pediatrician.

2) the child was diagnosed as hyperactive by a registered psychologist, in accord with the diagnostic criteria for Attention Deficit Disorder with Hyperactivity as outlined in the DSM-III (1980).

3) the assessment diagnosis was based on the presence of three cardinal characteristics of physical overactivity, short attention span, and impulsivity. A history of disciplinary problems as per tantrums, aggressive, destructive, or oppositional behaviours was present. These behaviours were generalized and pervasive, occurring at both home and school. Additionally, the child exhibited these characteristics prior to 2½ years of age, i.e. onset in early childhood.

4) the child received a Conner's Teacher Hyperactivity Index (CTHI) score of 1.5 or higher on the teachers rating scale (Goyette, Conners & Ulrich, 1978).

A total of 57 hyperactive index cases (37 biological and 20 adopted probands) were initially drawn from the assessment files of the Department of Psychology, C.H.E.O. The 57 families of the identified probands who appeared to meet the inclusion criteria were contacted by mail to explain the nature of the study and to request their participation. A copy of the letter can be found in Appendix B. Upon receipt of the letter, the parents were contacted by phone within seven days time. Subsequent to the phone contact a total of 15 families were excluded from participation on the basis of personal preference or the previously noted proband inclusion criteria. Six families declined participation due to involvement in other on-going studies or treatment programs. One family had moved away and two families had experienced a divorce or a separation since their initial hospital assessment. The teachers of two index cases noted that the children exhibited no problems at school, and the teacher's scores on the CTHI was below 1.0 for each of the boys. The parents of one child stated that their son presented no problems at home, while another three families provided no reason for refusal.

One other adopted hyperactive index case was selected

(as meeting all of the inclusion criteria) in response to the media advertisements to be discussed in the following section.

The average CTHI for the biological hyperactive index cases was 1.84 (SD ± .30) with a range of 1.5 to 2.5. The average CTHI for the adopted hyperactive probands was 1.76 (SD ± .36), range being 1.5 to 2.9 (Table 1).

Normal Control Index Children. A total of 45 normal control probands (25 biological normal controls and 20 adopted normal controls) and their parents participated in the study. The normal control index cases were selected from the medical record files of the C.H.E.O. and from response to newspaper advertisements. The children selected via the medical record files had been seen in the out-patient clinics for minor problems or yearly medical examinations. Children with a history of learning or behavioural problems were not included. No child was on psychotropic medication or had been hospitalized for more than one-4 day period during any previous 36 month interval of time, i.e. children with chronic medical problems were also excluded. In order to be selected as a normal control index case, the child had to receive a CTHI of 1.0 or lower.

Following the identification and acceptance of participation by the parents of the hyperactive index cases, the name of a prospective normal control proband was drawn from the medical record files, C.H.E.O., located chronologically

next after the accepting hyperactive index case in the files. The prospective control proband had to satisfy the aforementioned criteria. The parents of these children were sent a letter (Appendix B) describing the nature of the study and noting that they would be contacted directly by telephone within seven days time. A total of 98 families of identified biological normal control index cases were contacted and 27 agreed to participate (25 biological and 2 adopted normal controls). At the time of phone contact, 36 families declined due to time constraints or personal reasons. Four families refused because of serious illness. In addition, 31 families were excluded because they did not meet the criteria. In 17 of these families, there were single parents. Seven of the families involved second marriages or reconstituted relationships. Four children had been diagnosed as having learning disabilities. Another three children were excluded due to either a diagnosis of emotional problems, epilepsy, or a head injury. Subsequent to the phone contact, three children were excluded based on their teacher's ratings on the CTHI of 1.0 or higher. Another teacher reported that a child had been receiving remedial assistance due to a reading disability. One child was not included because he achieved a PPVT IQ equivalent of less than 85.

In addition to the two adopted normal control index cases selected through the medical records files, C.H.E.O.,

18 adopted normal control probands were selected for participation based on their parents' response to media advertisements. A display advertisement was placed on five occasions in Ottawa's major newspaper (Appendix C). A total of 51 phone calls were received in response to the newspaper advertisement. Six of the families included single parents and two families involved second marriages. Two families declined due to illness of a parent, and 11 families refused to participate. Four children were excluded due to learning disabilities. One child was excluded due to a chronic medical problem. Another child was excluded because he was receiving treatment for an emotional problem. Two children were not included because the teacher's score on the CTHI exceeded 1.0. Three children were found to be adopted after six months of age. Another child was subsequently selected to participate as an adopted hyperactive index case based on previous diagnosis and treatment for HA at the C.H.E.O.

The average CTHI score for the biological normal control probands was .23 (SD ± .21) with a range of 0 to 7; the average score for the adopted control probands was .25 (SD ± .19) with a range of 0 to 6 (Table 1). The ANOVA for the CTHI (Appendix A) indicated that the interaction effect was non-significant. However, a significant Group main effect was revealed, $\underline{F}(1,84) = 757.27$, $\underline{p} < .000$, indicating that the hyperactive probands scored higher on

the CTHI than the normal control index cases. There was no difference between the biological and adopted children in terms of CTHI scores.

Subjects

The subjects in this study were the 176 (88 sets) biological and adoptive parents of the 88 Selected hyperactive and normal control index cases. The parenting couples had had intact relationships from the time of the probands birth or adoption. Adoptions by biological relatives were excluded.

The participating parents averaged 37.36 years of age (SD ± 6.00). The age range for the parents was 26 to 57. The mean IQ for all parents was 113.39 (SD ± 8.74), with an IQ range of 85 to 135. The parents averaged 13.93 years of education (SD ± 2.83). The range for years of education was 6 to 21. Average family income for the couples was $30,480. (SD ± $11,770), with an income range of $13,000. to $70,000. A further discussion of the parent's demographic data is included in the results section.

Measures for Index Children

The following two measures were collected on each hyperactive and normal control index child:

Conners' Teacher Rating Scale (CTRS). Conners (1969) introduced a rating scale for teachers of 39 behavioural items which has become a popular and widely used means of evaluating hyperactive children. The scale is scored via

a four point numerical weighting system (0-3), with the higher values indicating more severe symptomatology. Factor analyses of the scale yielded five factors: (1) Conduct Problems, (2) Inattentive-Passive, (3) Tension-Anxiety, (4) Hyperactivity, and (5) Sociability. A symptom score is obtained by totaling the sum of the weighted items within each factor.

Conners (1969) originally developed the rating scale for teachers for use in psychopharmacological studies with children. The scale has repeatedly been shown to be sensitive to drug effects (Firestone, Davey, Goodman and Peters, 1978; Sprague, Christensen and Werry, 1974; Sroufe, 1975). Moreover, it has been demonstrated that the scale is successful in discriminating between hyperactive and normal control children (Kupietz, Bialer and Winsberg, 1972; Sprague, et.al., 1974).

Test-retest reliabilities for the five factors ranged from .70 to .90 (Conners, 1973). Research by Goyette, Conners and Ulrich (1978) has confirmed the high test-retest factor reliabilities based on new normative data, and has further delineated a ten point Hyperkinesis Index (CTHI). The Hyperkinesis Index has been shown to correlate at the .94 level with the Hyperactivity Factor (Werry, Sprague & Cohen, 1975). Mean scores of 1.5 to 1.8 on the CTHI have frequently been used as minimal cut-off points for establishing an inclusion criteria in HA studies (Loney, 1978).

The Hyperactivity Index with a cut-off point of 1.5 (based on a 0-3 weighted scoring system) was utilized for this investigation.

Peabody Picture Vocabulary Test (PPVT). The PPVT is a measure of receptive language which provides a verbal IQ equivalent via measuring a subject's aural vocabulary (Dunn, 1965). The examiner verbally presents a vocabulary stimulus word for each plate in the spiral-bound (Series of Plates) book. The subject is asked to make a choice from the four alternative drawings on each plate. The response may be either motor (pointing to) or verbal (saying the number of the drawing which best describes the stimulus word). The examiner continues until the subject produces six errors in any eight successive plates. Three types of scores are derived from the subjects' raw score: a mental age equivalent, an intelligence quotient (based on a standard score), and a percentile equivalent.

The test norms are based on data from 4,012 subjects aged 2.5 to 18.0 years. Alternate form reliability coefficients ranged from .67 at 6.0 years of age to .84 at 17.0 to 18.0 years of age. Congruent validity correlations comparing Wechsler full scale IQ scores for children with PPVT scores are reported to range from .30 to .84, with a median of .61.

Assessment Measures for Parents

Shipley Institute of Living Scale. The Scale was originally devised as a measure of organic pathology and cognitive deterioration (Shipley, 1940). The Shipley Scale is presently used as a brief screening measure of the subject's current level of intellectual functioning. The measure is self-administered in 20 minutes. It is comprised of two 20-item vocabulary and abstract reasoning tests wherein the subject has to mark the correct answer or to complete a pattern of responses.

Several investigators (Paulson & Lin, 1970; Sines & Simmons, 1959; Watson & Klett, 1968) have found correlations between the Shipley Scale and the full-scale WAIS score to range from .78 to .90. The Scale is reported to provide a suitable measure for estimating verbal intelligence and for deriving a WAIS IQ equivalent when average to above average Shipley scores are obtained, but not for below average scores (Prado & Taub, 1966; Watson & Klett, 1968). However, Paulson and Lin (1970) reported an acceptable correlation of .54 for IQs below 92 as per their own norms and a Shipley total raw score below 36.5. The Paulson-Lin Tables of IQ estimates were used in the present investigation.

Minnesota Multiphasic Personality Inventory (MMPI). The MMPI is the most widely used clinical and research measure of personality and disturbance (Butcher & Tellegen, 1978; Dahlstrom, 1972). The inventory is a symptomatic

self-report which covers a broad spectrum of concerns, such as health, family, sexual, occupational and social attitudes. It is self-administered and consists of 566 short, affirmative statements which the subject responds to by answering true or false. The structured test yields three validity scales which measure test taking attitude as well as provide a reference system for interpreting the clinical scales. The ten clinical scales include: hypochondriasis (1), depression (2), hysteria (3), psychopathic deviate (4), masculinity-femininity (5), paranoia (6), psychasthenia (7), schizophrenia (8), hypomania (9), and social introversion (10).

The test-retest and split-half reliabilities for several of the scales have been reported to exhibit wide variation (Dahlstrom, Welsh & Dahlstrom, 1972b, 1975). McKinley and Hathaway (1943) reported that the MMPI exhibited predictive validity. The inventory was successful in predicting the final clinical diagnosis in slightly better than 60% of new psychiatric admissions. Despite the stability problems, Waskow (1975) proposes that the MMPI is the best measure of psychological disturbance to date.

Several investigators have examined the MMPI patterns of parents of children referred to guidance and assessment clinics (Johnson & Lobitz, 1974; Lauterbach, London & Bryon, 1961; Wolking, Quant & Lawton, 1966). Anderson (1969) reported that the parents of aggressive, acting out boys

obtained higher Sc and Pd scale scores than the parents of neurotics or normal controls. It is concluded that these higher elevations were indicative of the parents' difficulty with overt aggression and with inadequate impulse control.

Due to problems of item redundancy and the multifactorial nature of several of the scales, pattern or actuarial analysis has been recommended as an additional means of identifying traits and diagnoses associated with particular profile types (Marks, Seeman & Haller, 1974). Actuarial descriptions of various two-point profile types have been provided by several authors (Gilberstadt & Duker, 1965; Lachar, 1974; Marks, Seeman & Haller, 1974). Individuals with a 1-3/3-1 code type tend to see themselves as physically ill and to employ repression as a primary mechanism of defense. The 1-3 individual is frequently described as demanding, dependent, and self-centered. The 2-4/4-2 code suggests an overly-sensitive, demanding, and resentful person. The 2-4 code type is also seen to represent a characterological adjustment with chronic depression and low frustration tolerance. Code 2-7/7-2 individuals are typically described as anxious, tense, apprehensive, and depressed. In addition to perfectionistic and compulsive tendencies, the 2-7 individual frequently tends to have conflicts about self-assertion. The 4-9/9-4 profile type is likely to be impulsive, hostile, rebellious,

irritable, and restless. The 4-9 individual typically experiences conflicts about emotional dependency and intimacy. Moreover, these four two-point codes have been associated with the types of adult psychopathology (alcoholism, sociopathy, and hysteria) found in the families of hyperactive children by Cantwell (1975) and Morrison and Stewart (1973).

Although a number of different code types have been associated with alcoholism, elevated scores on 2-4 and 2-7 have frequently been found in more serious drinkers (Caster & Parsons, 1977; Whitelock, 1971). The pairing of 4-9 has commonly been employed for assessing the potential for impulsive action and sociopathy (Dahlstrom, 1972; Marks and Seeman, 1963). Lewandowski and Graham (1972) reported that a 1-3 profile was characteristic of hysterical, conversion type patients. These four two-point code patterns (1-3/3-1, 2-4/4-2, 2-7/7-2 and 4-9/9-4 were examined in the present investigation. The score for the two-point code was obtained by combining the K-corrected T scores for the individual scales. The justification for utilizing an additive approach in combining separate MMPI scales is supported by Goldberg's (1969) work in combining scales linearly to classify profiles as neurotic or psychotic. In addition to the two-point codes, the Repression-Sensitization scale (Byrne, Barry & Nelson, 1963), and the Ego Strength scale (Barron, 1953), and the Total

Pathology Score (Streiner, Goodman & McLean, 1977; Streiner, Woodward, Goodman & McLean, 1973) were also used.

Porteus Maze Test. The Maze test was originally developed as a supplement to the Stanford-Binet Intelligence Scale in identifying mental retardation (Porteus, 1933, 1939). More recently, the test has been reported to be a valid and reliable measure of planning ability, judgement, impulsiveness, attention and ability to delay gratification (Riddle & Roberts, 1974; Sroufe, 1975).

The Maze test involves a series of graded mazes which yield two scores, a quantitative score (Test Age) and a qualitative (Q) score. The Q score is based on the subject's neatness in executing the mazes. The number of errors committed, such as cutting across corners and contacts with the sides of the pathway, are recorded. In a review of the Porteus Maze Test, Riddle and Roberts (1977) reported that subjects who tend to delay gratification obtain lower Q scores than more impulsive subjects. It was also concluded that Q scores are valid predictors of impulse control problems in delinquency. A high weighted Q score has been found to positively predict the corresponding final clinical assessment of delinquency or non-delinquency in more than 70% of the cases tested. Parry (1973) found differences between hyperactives and normal controls, with the hyperactive children scoring significantly more qualitative

errors. Investigators have reported that training in self-directed verbal commands resulted in improved maze performance due to reduced impulsiveness in hyperactive boys (Meichenbaum, 1977; Palkes, Stewart & Kahana, 1968).

More recently, in a discriminant analysis of 27 measures frequently used to distinguish between hyperactive and normal control children, Homatidis and Konstantareas (1981) found that the Porteus Maze Test was one of only three measures needed to accurately discriminate between their samples of hyperactive and normal control boys. The investigators utilized the quantitative test age score, rather than the more sensitive Q score.

An automated version of the Porteus Maze Test was used in the present investigation. The subject was directed to guide a stylus through a metallic maze. The instrument automatically recorded three scores: the number and duration of contacts with the sides of the maze pathway as well as the total time to the completion of the maze was recorded. Knights and Moule (1968) have provided normative data with respect to the automated version of the Porteus Maze Test.

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, Estes (1965) 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 letters T or F) upon a tachistoscopic exposure of short duration. Each signal letter is displayed in 50% of the presentations and is embedded in an array of competing noise letters. The span of apprehension is defined by the number of letters scanned. Estes reported that the formula for deriving a span estimate

is A = (2Pc - 1), with A representing the span; D, the number of letters present in the specific display; and Pc, the percentage of correct recognitions.

The stimulus displays were constructed by typing arrays of letters onto white index cards. The card was then mounted on a cardboard insert to facilitate presentation via a portable tachistoscope. An imaginary matrix of 16 letters (4 letters wide and 4 letters high) at the centre of each card was used to allocate the display letters, each matrix subtended 2^{o} X 3^{o} of visual angle. Each display array contained one of two signal letters (T or F) with each target letter appearing once on three separate cards in each of 16 possible matrix positions, producing a total of 48 T and 48 F arrays. The first set of 32 arrays consisted of 16 T and 16 F cards containing only the signal letter (Matrix Size 1). A second set of 32 arrays with 16 T and 16 F cards contained the target letter plus four additional noise letters (Matrix Size 5). A third set of 32 arrays (16 T and 16 F cards) contained the signal letter plus 8 noise letters (Matrix Size 9). For the last two sets, the signal letter was located in one of the 16 possible matrix positions. Then, either three or eight noise letters were randomly selected and allocated to three or eight of the 15 remaining matrix positions. The stimulus array was presented on a portable tachistoscope (Lafayette Prototype).

The subject was given a standard set of instructions

and 24 practice trials. In the practice trials, the subject was presented with three randomly selected blocks of eight trials. Each block was drawn from a separate matrix size, with the restriction of an equal occurrence of the signal elements (4 Ts and 4 Fs). After a 2 minute rest period, the experimental blocks were administered. Each subject then viewed three sets (Matrix Sizes I, 5 and 9) of three 10 trial blocks, with each set separated by a 1 minute break. The order of presentation of the three blocks as well as the stimulus arrays within the blocks was randomized for each subject. The subject initiated each trial by pressing a start button. Following a delay interval of 750 msec, the stimulus display was presented for 90 msec. The subject was required to report the target letter (T or F) observed on the display.

The Span of Apprehension Task has been frequently used in research with schizophrenic children and adults (Asarnow, Steffy & Cleghorn, 1978; Asarnow & MacCrimmon, 1981; Neale, McIntyre, Fox & Cromwell, 1969). Neale and his co-workers (1969) asserted that the reduction in correct detections on the Span task represents a true deficit in attention.

Denton and McIntyre (1978) found differences between hyperactive and normal control boys on the Span test. As the matrix size and number of noise letters increased, the hyperactive boys evidenced fewer correct recognitions, suggestive of a central attentional deficit. The investiga-

tions of Asarnow and MacCrimmon (1981) have provided normative data in terms of means and standard deviations (Asarnow, personal communication).

Reaction Time Apparatus. The delayed reaction time task (DRT) has been used as a measure of attentional processes. The task has been found to discriminate between hyperactive and normal children (Cohen & Douglas, 1972; Firestone & Douglas, 1975; Firestone & Martin, 1978), as well as to be drug sensitive (Cohen, Douglas & Morgenstern, 1971; Firestone, et. al., 1978; Firestone, Poitras-Wright & Douglas, 1978).

The reaction time apparatus provided a preprogrammed series of auditory and visual stimuli. An auditory tone of 70-db intensity and 1 second duration was fed directly to the subject's earphone and served as the warning signal to activate the sequence. The reaction signal consisted of a 7.5-watt lightbulb which was enclosed in a metal console container along with the response button. The reaction signals were set at fixed intervals of 20 seconds, and each signal was followed by a 5-second wait period. Following the specified wait period, the auditory warning signal was presented on a variable schedule within the subsequent 10-second interval. Each reaction signal was preceeded by a fixed 2-second preparatory interval.

The subject was directed to depress the reaction time button throughout the DRT except during the reaction signal

presentations. At the onset of the visual reaction signal, the subject was instructed to release the response button and to redepress it as quickly as possible. An electronic timer was simultaneously started with the onset of the reaction signal and continued recording until the subject released the response button. The mean reaction time over 80 trials was then calculated. A counter automatically recorded all false starts elicited by the warning signals as well as inappropriate interstimulus responses. Thus, the second measure on this task consisted of the total number of extraneous responses.

Procedures

Following the telephone contact and the family's acceptance of involvement in the study, the parents were notified that they would receive an informed consent form (Appendix D) and a release of information (Appendix E). The parents were requested to sign the forms and to return them in the attached self-addressed, stamped envelope. Upon written permission from the parents, a Conners Teachers Rating Scale (CTRS) was sent to the index child's teacher for completion and return via a self-addressed envelope. After the CTRS was returned and an appropriate CTHI criterion score 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

auditory stimuli. The PPVT was administered in one period of approximately 15 minutes.

The parents were tested separately with a battery of six tests. The tests were presented in random order for each partner, with the complete battery being administered in one session of approximately three hours. All tests were administered as per standard instructions. A fifteen minute break was given between the third and fourth test in the series.

Ethical Considerations

The parents of the index children were informed as to the general objectives and the procedures of the study. A release of information form for the child's school and a consent form for the study were signed by the parents. The parents were also informed of their option to withdraw themselves or their children from the study at any time.

Data on the subjects in the study are kept in strict confidence and presented in group form only.

CHAPTER IV

RESULTS

This chapter is divided into two sections. In the first section, the results of the statistical analyses for the subject population obtained on the demographic variables will be presented. The intercorrelations among the demographic variables, as well as the interrelationships between the demographic and the dependent variables will also be given. The second section will provide a report of the interrelationships among the dependent measures. Subsequent to the correlational analyses, three separate MANOVAs were undertaken for the impulsivity, attention, and personality groupings of variables respectively. Where appropriate, the results derived from subsequent ANOVA analyses and tests of simple main effects will also be presented. The .05 level of confidence, based on a two-tailed test, was utilized as the measure of significance for the MANOVA and ANOVA analyses.

Demographic Variables

In separate ANOVAs on the dependent measures, it was found that there was no difference between the groups for the parents total family income (Appendix F). However, there were differences between the groups for three of the

demographic variables: the parents ages, IQs, and years of education. The group means and standard deviations for the demographic variables are presented in Table 2.

Age: Analysis of variance revealed no significant interaction effects for the Age variable (Appendix G). However, there was a significant Relationship main effect, $F(1,168) = 26.92$, $p < .000$, indicating that the adoptive parents of the hyperactive and normal control children were older than the biological parents of hyperactive and normal control children. In addition, a significant Sex main effect, $F(1,168) = 4.27$, $p < .04$, was obtained, reflecting that mothers were younger than fathers (see Table 3 regarding the mothers and fathers means and standard deviations for the demographic variables).

IQ: The ANOVA conducted on the parents IQs revealed a significant two-way interaction for Group X Relationship, $F(1,168) = 4.81$, $P < .03$ (Appendix H). As recommended by Keppel (1973) and Kirk (1968), in the event of significant interactions, analyses of simple main effects were performed. Results of the simple main effects analyses (see Appendix H) revealed that the biological parents of hyperactive children (B·HC) had lower IQs than the adoptive parents of hyperactive children (A·HC) and the biological parents of normal control children (B·NCC). No IQ differences were found between the adoptive parents of normal control children (A·NCC) and the A·HC or between the A·NCC and the B·NCC. In addition, there

Table 2

Parent Group Mean Scores and Standard Deviations for Age, IQ, Education, and Family Income

	B·HC	A·HC	B·NCC	A·NCC
Age	34.84 5.27*	40.86 6.55	36.68 4.96	39.63 5.94
IQ	107.66 8.88	114.44 8.70	115.52 7.10	116.93 7.54
Education	12.06 2.96	14.42 2.49	14.60 2.60	15.13 2.54
Family Income	27.85 12.15	31.06 11.34	30.88 10.61	32.75 12.69

B·HC: Biological parents of hyperactive children (n=50)
A·HC: Adoptive parents of hyperactive children (n=36)
B·NCC: Biological parents of normal control children (n=50)
A·NCC: Adoptive parents of normal control children (n=40)

*Standard Deviation

was a significant main effect for Sex, $F(1,168) = 4.01$, $p < .05$, indicating that the fathers obtained higher IQ scores than the mothers (Table 3).

Education: The results of the ANOVA for years of education indicated a significant two-way interaction for Group X Relationship, $F(1,168) = 4.50$, $p < .04$, as well as significant main effects for all three main factors (Appendix I). In view of the significant interaction effect, analysis of simple main effects was performed. The results of the tests of simple main effects (Appendix I) indicated that the B·HC completed fewer years of education than the A·HC and the B·NCC. No difference in years of education was found between the A·NCC and the A·HC or between the A·NCC and the B·NCC. The Sex main effect was also found to be significant, $F(1,168) = 5.58$, $p < .02$, reflecting that the fathers completed more years of education than the mothers (Table 3).

Income: As previously noted, the ANOVA conducted on the total family income proved to be non-significant (Appendix F). As all interaction effects and main effects were not significant, further analyses of the income variable are not reported.

The results of the correlational analysis performed on the demographic variables are presented in Table 4. The matrix reveals a number of low but significant associations

Table 3

Mothers and Fathers Group Means and Standard Deviations for Age, IQ, and Level of Education

	B·HC	A·HC	B·NCC	A·NCC
Age (years)				
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 (years)				
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

B·HC: Biological parents of hyperactive children (n=25)
A·HC: Adoptive parents of hyperactive children (n=18)
B·NCC: Biological parents of normal control children (n=25)
A·NCC: Adoptive parents of normal control children (n=20)

*Standard Deviation

Table 4

Pearson Product Moment Correlation Coefficients for the Demographic Variables

	Age	IQ	Education	Total Family Income
Age	1.00			
IQ	.19*	1.00		
Education	.12	.62**	1.00	
Family Income	.15	.28**	.39**	1.00

*r=.17, p<.01 (n=176)

**r=.22, p<.001 (n=176)

between the demographic variables. Level of education correlates highly and significantly with IQ (r = .62, r^2 = .38, p < .000).

Table 5 presents the intercorrelations between the demographic variables and the dependent variables. Several significant but low correlations exist, ranging from r = .17 to r = .28, irrespective of sign. The amount of variance shared in common between any combination of demographic and dependent measures is also low, ranging from r^2 = .03 to r^2 = .08. Keppel (1973, p. 477) has proposed that "the main criterion for a control variable is a high correlation with the dependent variable". Similarly, Winer (1971) 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

The results of the correlational analyses between the personality variables and the attention and impulsivity variables (Table 6) revealed only four significant yet low correlations, ranging from r = .19 to r = .28, irregardless of sign. The Delayed Reaction Time Task-Mean Time (DRTT) was negatively correlated with the Ego Strength (Es) Scale. The DRTT was positively correlated with the Total Pathology

Table 5

Pearson Product Moment Correlation Coefficients Among the Demographic Variables and Dependent Variables

	Age	IQ	Education	Family Income
Delayed Reaction Time Task (DRT) Mean Time (DRTT)	-.05	-.24**	-.28**	-.06
DRT Extraneous Responses (DRTEXT)	-.01	-.03	-.05	-.12
Span Size 1	.11	.06	.02	.03
Span Size 5	.00	.12	.22*	.13
Span Size 9	-.02	.25**	.19*	-.08
Maze-Time	.08	.05	-.05	.06
Maze-Contacts	-.08	-.17*	-.15	-.05
Maze-Duration of Contacts	.11	-.18*	-.09	-.04
Revised Repression-Sensitization (R-S)	-.19*	-.14	-.19*	.00
Ego Strength (Es) Scale	.19*	.15	.26**	.08
Total Pathology Score (TPS)	-.12	-.17*	-.21*	-.06
Code 1-3	-.08	-.07	-.12	-.01
Code 2-4	-.12	-.18*	-.22**	-.02
Code 2-7	-.15	-.18*	-.21*	-.02
Code 4-9	-.05	-.03	-.04	.03

*r=.17, p<.01 (n=176)

**r=.22, p<.001 (n=176)

Table 6

Pearson Product Moment Correlation Coefficients
Between Personality Variables and Attention-Impulsivity Variables

	Attention Measures					Impulsivity Measures		
	DRTT	DRTEXT	Span Size 1	Span Size 5	Span Size 9	Maze-Time	Maze-Contacts	Maze Duration of Contacts
Revised Repression-Sensitization Scale	.03	-.01	-.17*	.05	.08	.09	-.01	.03
Ego-Strength Scale	-.18*	.02	.03	.05	-.02	-.11	-.04	.00
Total Pathology Score	.20*	.03	.03	-.16	-.11	-.11	.10	.12
Code 1-3	.11	.00	.00	-.05	-.09	.00	.01	.10
Code 2-4	.20*	-.01	-.02	-.05	-.07	-.02	.05	.06
Code 2-7	.14	-.03	-.02	-.05	-.02	-.03	.01	.02
Code 4-9	.02	.00	.03	.04	-.08	.07	.06	.11

*r=.17, p<.01 (n=176)

**r=.22, p<.001 (n=176)

Score (TPS) and with the 2-4 two-point code. The revised Repression-Sensitization (RS) Scale was negatively correlated with Span Size 1 of the Span of Apprehension test.

The results of the correlational analysis between the attention and the impulsivity variables revealed that none of these variables were significantly associated (Table 7).

The intercorrelation matrix for the three impulsivity measures is presented in Table 8. The matrix reveals that Porteus Maze Time correlates significantly, but lowly, with both Maze Contacts ($r = .33$, $r^2 = .11$, $p < .001$), and Maze Duration of Contacts ($r = .36$, $r^2 = .13$, $p < .001$). Maze Contacts correlates highly and significantly with Maze Duration of Contacts ($r = .71$, $r^2 = .50$, $p < .001$).

An examination of the intercorrelation matrix for the four attention variables (Table 9) reveals several low but significant correlations, ranging from $r = .16$ to $r = .48$, irregardless of sign. A significant and moderate correlation exists between Span Size 5 and Span Size 9 ($r = .48$, $r^2 = .23$, $p < .001$).

The intercorrelation matrix for the seven personality variables is presented in Table 10. The present study found the following codes and research scales to be highly and positively correlated: Code 2-4 and TPS, ($r = .76$, $r^2 = .58$,

Table 7

Pearson Product Moment Correlation Coefficients Between The Attention and The Impulsivity Variables

	DRTT	DRTEXT	Span Size 1	Span Size 5	Span Size 9
Maze-Time	-.02	.05	-.01	.10	.01
Maze-Contacts	.04	-.04	-.04	-.13	-.05
Maze-Duration of Contacts	.10	.07	-.02	-.10	-.15

*r=.17, p<.01 (n=176)

**r=.22, p<.001 (n=176)

Table 8

Pearson Product Moment Intercorrelation Matrix
For The Three Impulsivity Variables

Variable	Maze Time	Maze Contacts	Maze Duration of Contacts
Maze-Time	1.00		
Maze-Contacts	-.33**	1.00	
Maze-Duration of Contacts	-.36**	.71**	1.00

*r=.17, p<.01 (n=176)

**r=.22, p<.001 (n=176)

Table 9

Peason Product Moment Intercorrelation Matrix
For The Four Attention Variables

	DRTT	DRTEXT	Span Size 1	Span Size 5	Span Size 9
DRTT	1.00				
DRTEXT	.16*	1.00			
Span Size 1	-.09	-.28**	1.00		
Span Size 5	-.21*	-.16*	.33**	1.00	
Span Size 9	-.25**	-.12	.22**	.48**	1.00

*r=.17, p<.01 (n=176)

**r=.22, p<.001 (n=176)

Table 10

Pearson Product Moment Intercorrelation Matrix
For the Seven Personality Variables

	Revised R-S Scale	Es Scale	Total Pathology Score	Code 1-3	Code 2-4	Code 2-7	Code 4-9
Revised Repression-Sensitization (R-S) Scale	1.00						
Ego Strength (Es) Scale	-.49**	1.00					
Total Pathology	.29**	-.49**	1.00				
Code 1-3	.17*	-.29**	-.55**	1.00			
Code 2-4	.29**	-.52**	.76**	.47**	1.00		
Code 2-7	.44**	-.57**	.75**	.48**	.84**	1.00	
Code 4-9	.13	-.28**	.43**	.37**	.56**	.31**	1.00

*r=.17, p<.01 (n=176)

**r=.22, p<.001 (n=176)

$p < .001$), Code 2-7 and TPS ($r = .75$, $r^2 = .56$, $p < .001$), and Code 2-4 and Code 2-7 ($r = .84$, $r^2 = .71$, $p < .001$). Several significant and moderate, positive correlations were revealed: Code 2-7 and Repression-Sensitisation (R-S) Scale ($r = .44$, $p < .001$), TPS and Code 1-3 ($r = .55$, $p < .001$), TPS and Code 4-9 ($r = .43$, $p < .001$), Code 4-7 and Code 2-4 ($r = .47$, $p < .001$), Code 1-3 and Code 2-7 ($r = .48$, $p < .001$), and Code 2-4 and Code 4-9 ($r = .56$, $p < .001$). Moderately significant negative correlations were obtained for: R-S Scale and Es Scale ($r = -.49$, $p < .001$), Es Scale and TPS ($r = -.49$, $p < .001$), Es Scale and Code 2-4 ($r = -.52$, $p < .001$), and Es Scale and Code 2-7 ($r = -.57$, $p < .001$).

In view of the absence of significant associations between the attention and impulsivity variables and the few, low correlations between the personality and attention/impulsivity variables (as well as the moderate to highly significant associations among the impulsivity, attention and personality variables), three separate MANOVA,s were conducted for the five attention, three impulsivity, and seven personality variables.

Attention Variables: The MANOVA analysis for the five attention variables within the Group X Relationship X Sex design, was conducted. All main effects and the Group X Relationship interaction were significant (Table 11).

Table 11

Results of MANOVA for the Five Attention Variables

	df	F	p<
Group	5,164	8.03	.000***
Relationship	5,164	3.56	.004**
Sex	5,164	2.90	.02*
Group X Relationship	5,164	3.77	.003**
Group X Sex	5,164	.26	.94
Relation X Sex	5,164	1.76	.12
Group X Relationship X Sex	5,164	.22	.95

*p<.05

**p<.01

***p<.001

Therefore, 2 X 2 X 2 ANOVAs were undertaken for each attention variable. Table 12 contains the means and standard deviations for the Delayed Reaction Time Task and Span of Apprehension attention measures.

DRTT: The findings of the ANOVA for the Delayed Reaction Time Task-Mean Time (Appendix J) revealed significant results for all main effects as well as a significant Group X Relationship interaction effect, $F(1,168) = 9.88$, $p < .002$. No other interaction effects were found to be significant. As a follow-up to the significant Group X Relationship interaction, analyses of simple main effects were undertaken.

The results of the tests of simple main effects (Appendix J) revealed that the B-HC had slower mean reaction times than the A-HC and the B-NCC. No differences in reaction time were found between the A-NCC and the A-HC or between the A-NCC and the B-NCC (see Figure 1).

In addition, there was a significant main effect for Sex, $F(1,168) = 1.88$, $p < .001$, indicating that mothers had slower mean reaction times than fathers. (See Table 13 regarding mothers and fathers group means and standard deviations on the attention variables.)

DRTEXT: The Delayed Reaction Time Task-Extraneous Responses were then analysed through ANOVA (Appendix K) and yielded no significant main or interaction effects.

Table 12

Parent Group Means and Standard Deviations for the Five Attention Variables

	B-HC	A-HC	B-NCC	A-NCC
DRTT	348.04	302.53	288.35	289.70
	64.56*	39.11	48.23	41.53
DRTEXT	2.74	2.36	2.14	2.28
	2.66	2.09	2.08	2.18
Span Size 1	29.96	29.94	29.98	29.93
	.20	.23	.14	.27
Span Size 5	29.28	29.81	29.80	29.70
	1.11	.47	.61	.79
Span Size 9	27.16	28.44	28.54	28.45
	1.78	1.30	1.23	1.26

B-HC: Biological parents of hyperactive children (n=50)
A-HC: Adoptive parents of normal control children (n=36)
B-NCC: Biological parents of normal control children (n=50)
A-NCC: Adoptive parents of normal control children (n=40)

*Standard Deviation

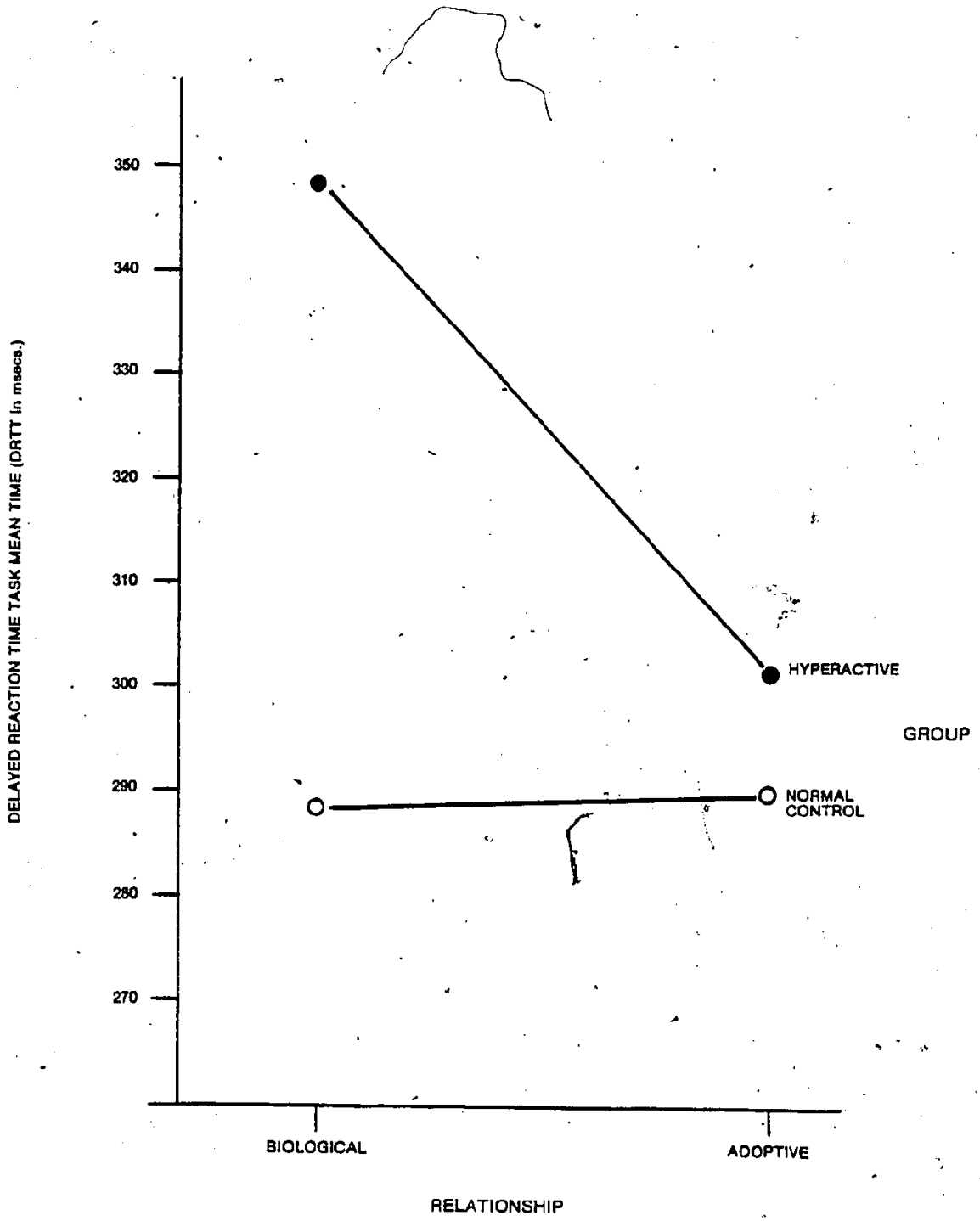

Fig. 1 — Delayed Reaction Time Task Mean Time (msecs.) to parental relationship (biological adoptive) and index group (hyperactive, normal control).

Table 13

Mothers and Fathers Group Means and Standard Deviations for the Five Attention Variables

		B·HC	A·HC	B·NCC	A·NCC
DRTT					
	Mothers	367.50	306.92	306.08	295.19
		59.04*	40.56	54.72	44.47
	Fathers	328.57	298.14	270.62	284.21
		65.07	38.26	33.18	38.71
DRTEXT					
	Mothers	2.92	2.22	2.04	2.20
		2.78	1.83	2.13	2.29
	Fathers	2.56	2.50	2.24	2.35
		2.57	2.36	2.07	1.93
SPAN SIZE 1					
	Mothers	29.96	29.89	30.00	29.90
		.20	.32	.00	.31
	Fathers	29.96	30.00	29.96	29.95
		.20	.00	.20	.22
SPAN SIZE 5					
	Mothers	29.28	29.83	29.76	29.70
		.94	.38	.60	.80
	Fathers	29.28	29.78	29.84	29.70
		1.28	.55	.63	.80
SPAN SIZE 9					
	Mothers	27.36	28.61	28.76	28.30
		1.58	.98	.93	1.17
	Fathers	26.96	28.28	28.32	28.60
		1.97	1.57	1.46	1.35

B·HC: Biological parents of hyperactive children (n=25)
A·HC: Adoptive parents of hyperactive children (n=18)
B·NCC: Biological parents of normal control children (n=25)
A·NCC: Adoptive parents of normal control children (n=20)

*Standard Deviation

Therefore, further analyses of that variable are not reported.

Span Size 1: The ANOVA results on Span Size 1 also proved to be non-significant (Appendix L). In view of the lack of significant main effects and interaction effects, subsequent analyses are not recorded on the variable.

Span Size 5: The 2 X 2 X 2 ANOVA findings for Span Size 5 (Appendix M) indicated a significant result for the Group main effect, $F(1,168) = 4.21$, $p < .04$, and a significant Group X Relationship interaction effect, $F(1,168) = 6.44$, $p < .01$. Therefore, tests for simple main effects were conducted. The results of the simple main effects analyses (Appendix M) indicated that the B·HC had fewer correct recognitions on Span Size 5 than the B·NCC and the A·HC. The A·NCC were not different from the B·NCC and the A·HC on correct recognitions on this variable (see Figure 2).

Span Size 9: The Span Size 9 ANOVA produced a significant Group result, $F(1,168) = 13.32$, $p < .000$, a significant Relationship main effect, $F(1,168) = 7.02$, $p < .01$, and a significant Group X Relationship interaction effect, $F(1,168) = 9.96$, $p < .002$ (Appendix N). No other effects were significant.

In keeping with the convention of attending to the highest order significant interaction within a design, tests of simple main effects were undertaken. The results of the

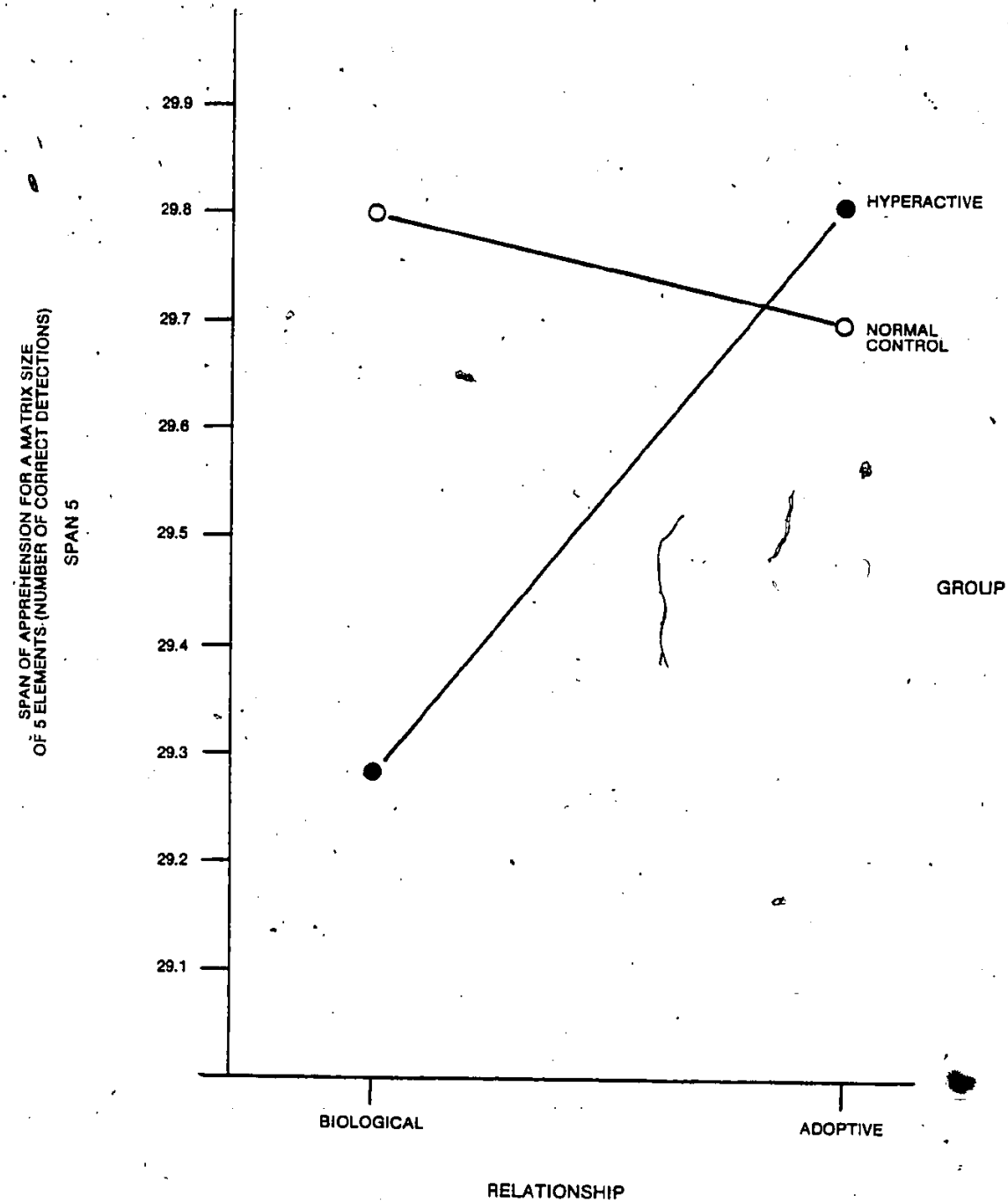

Fig. 2 — Number of correct recognitions of Span of Apprehension matrices with 5 elements (Span 5) to parental relationship (biological, adoptive) and index group (hyperactive, normal control).

analyses (Appendix N) revealed that the B·HC had fewer correct recognitions on Span Size 9 than the B·NCC and the A·HC. No differences in correct recognitions on Span Size 9 were found between the A·NCC and the A·HC or between the A·NCC and the B·NCC (see Figure 3).

Impulsivity Variables: A MANOVA conducted on the three impulsivity variables of the Porteus Maze Text (Appendix O) proved to be non-significant. As a matter of interest in view of the generally low correlations among the impulsivity measures, separate ANOVA's were performed for the three impulsivity variables of Maze Time, Maze Contacts, and Maze Duration of Contacts (Appendices P, Q, and R, respectively). As all main effects and interaction effects on the separate ANOVA's were again not significant, further analyses on the impulsivity variables are not reported. The group means and standard deviations for the impulsivity measures are presented in Table 14.

Personality Variables: Three research scales and four two-point codes of the MMPI were incorporated into one MANOVA and these results are presented in Table 15. The Group X Relationship interaction effect proved to be significant, $F(7,162) = 2.47$, $p < .02$. In addition, there was a significant Group main effect, $F(7,162) = 5.99$, $p < .000$, and a significant Sex main effect, $F(7,162) = 4.18$, $p < .000$. Therefore, 2 X 2 X 2 ANOVAs for each variable

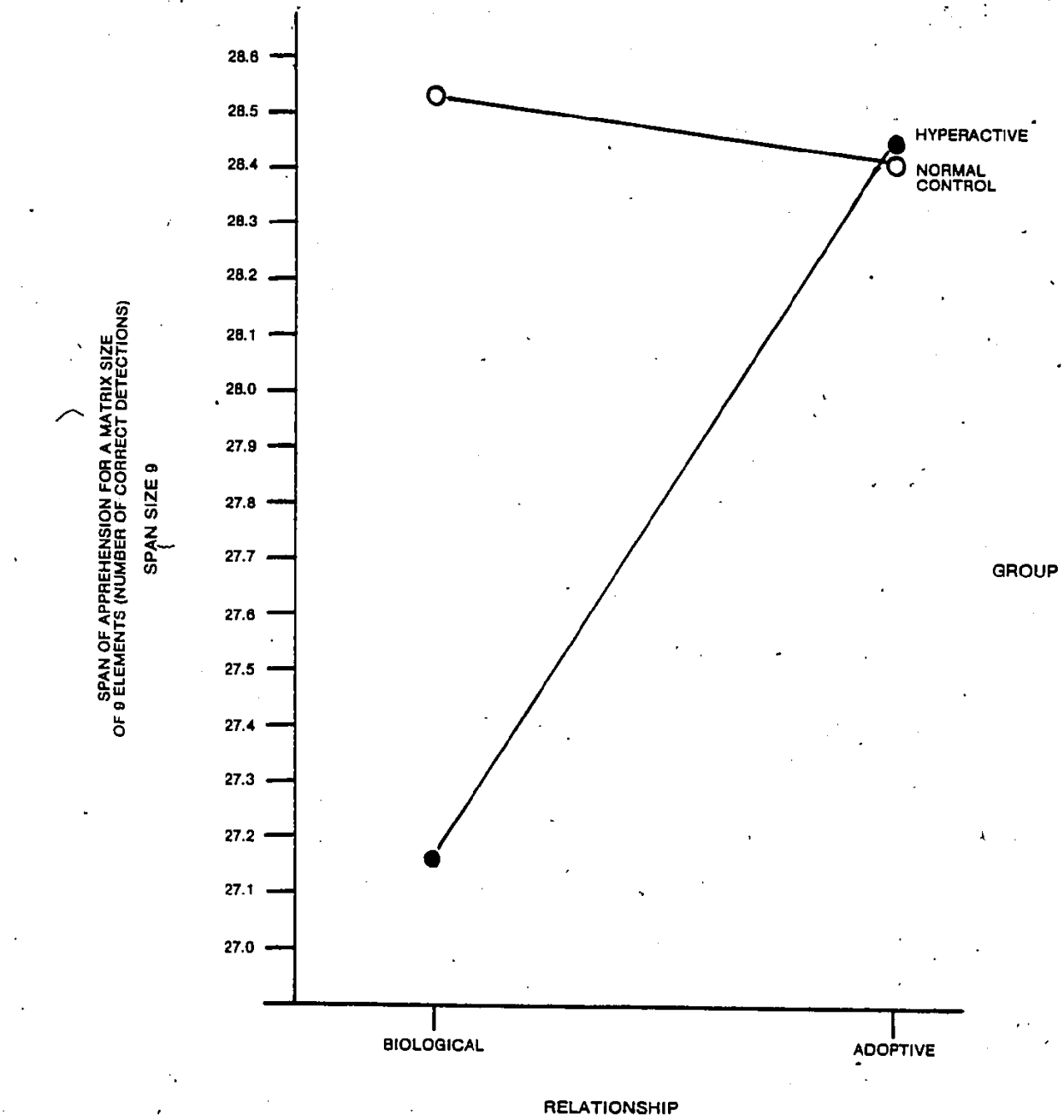

Fig. 3 — Number of correct recognitions of Span of Apprehension matrices with 9 elements (Span 9) to parental relationship (biological, adoptive) and index group (hyperactive, normal control).

Table 14

Parent Group Means and Standard Deviations for the Three Impulsivity Variables

	B·HC	A·HC	B·NCC	A·NCC
Maze - Time to Completion	21.99 7.98*	24.56 6.75	23.14 5.23	23.72 6.17
Maze - Number of Contacts	47.19 18.05	42.97 17.32	41.82 16.76	44.04 19.04
Maze - Duration of Contacts	4.38 1.80	4.34 1.76	4.06 1.45	4.60 1.80

B·HC: Biological parents of hyperactive children (n=50)
A·HC: Adoptive parents of hyperactive children (n=36)
B·NCC: Biological parents of hyperactive children (n=50)
A·NCC: Adoptive parents of hyperactive children (n=40)

*Standard Deviation

Table 15

Results of MANOVA for the Seven Personality Variables

	df	F	p<
Group	7,162	5.99	.000***
Relationship	7,162	1.68	.12
Sex	7,162	4.18	.000***
Group X Relationship	7,162	2.47	.02*
Group X Sex	7,162	1.01	.43
Relation X Sex	7,162	.53	.81
Group X Relationship X Sex	7,162	1.19	.31

*p<.05

**p<.01

***p<.001

were undertaken. The group means and standard deviations for the seven personality variables are presented in Table 16.

R-S Scale: The ANOVA for the R-S Scale indicated that the interaction effects were non-significant (Appendix S). However a significant main effect for Sex was revealed, $F(1,168) = 5.14$, $p < .03$, indicating that the fathers produced lower R-S Scale scores than the mothers. (See Table 17 regarding the mothers and fathers group means and standard deviations for the seven personality variables.)

Es Scale: Es Scale scores were analysed through ANOVA (Appendix T) and yielded a significant difference for the Group main effect, $F(1,168) = 7.71$, $p < .01$, indicating that the parents of normal control children obtained higher Es Scale scores than the parents of hyperactive children. There was also a significant main effect for Sex, $F(1,168) = 4.72$, $p < .03$, reflecting that fathers achieved higher Es Scale scores than mothers (see Table 17). No differences in interactions or other main effects were found, therefore further analyses are not reported.

TPS: The ANOVA results for the Total Pathology Score (Appendix U) revealed a significant Group X Relationship interaction effect, $F(1,168) = 9.57$, $p < .002$, as well as all three significant main effects. Tests for simple main

Table 16

Parents Group Means and Standard Deviations for the Seven Personality Variables

	B-HC	A-HC	B-NCC	A-NCC
R-S Scale	32.58	31.25	32.02	33.73
	18.51*	16.29	11.99	15.92
Es Scale	29.24	31.25	31.72	31.65
	4.36	3.78	3.45	3.56
TPS	11.66	9.33	8.54	8.90
	3.98	2.04	2.35	2.57
Code 1-3	112.70	107.14	104.90	107.45
	17.53	12.40	11.56	14.15
Code 2-4	121.80	108.56	103.06	105.43
	19.48	12.73	12.01	12.26
Code 2-7	118.26	106.50	106.14	104.63
	20.59	11.77	14.31	14.87
Code 4-9	111.72	108.72	101.08	104.98
	17.74	14.35	14.08	12.94

B-HC: Biological parents of hyperactive children (n=50)
A-HC: Adoptive parents of hyperactive children (n=36)
B-NCC: Biological parents of normal control children (n=50)
A-NCC: Adoptive parents of normal control children (n=40)

*Standard Deviation

Table 17

Mothers and Fathers Group Means and Standard Deviations for the Seven Personality Variables

	B-HC	A-HC	B-NCC	A-NCC
R-S Scale				
Mothers	37.60	32.94	33.80	35.50
	17.11*	15.41	11.46	14.93
Fathers	27.56	29.56	30.24	31.95
	18.82	17.39	12.47	17.05
Es Scale				
Mothers	28.36	30.28	30.92	31.90
	4.20	3.97	4.02	2.88
Fathers	30.12	32.22	32.52	31.40
	4.42	3.42	2.60	4.20
TPS				
Mothers	12.08	8.61	7.80	8.55
	4.24	1.88	1.83	1.70
Fathers	11.24	10.06	9.28	9.75
	3.73	1.98	2.61	3.02
Code 1-3				
Mothers	117.64	105.17	101.96	105.55
	19.56	12.49	12.81	16.12
Fathers	107.76	109.11	107.84	109.35
	13.92	12.34	9.53	11.98
Code 2-4				
Mothers	123.32	107.39	99.84	102.30
	20.88	12.29	9.52	8.05
Fathers	120.28	109.72	106.28	108.55
	18.26	13.40	13.51	14.94
Code 2-7				
Mothers	120.12	104.44	103.16	99.80
	21.01	10.17	12.43	9.42
Fathers	116.40	108.56	109.12	109.45
	20.43	13.15	15.65	17.78
Code 4-9				
Mothers	112.56	109.28	96.72	102.00
	20.45	13.66	10.50	14.37
Fathers	110.88	108.17	105.44	107.95
	14.94	15.38	15.96	10.89

*Standard Deviation

effects were conducted. The analysis (Appendix U), revealed that the B·HC had higher scores on the TPS than the A·HC and the B·NCC. No differences in TPS were found between the A·NCC and the B·NCC (see Figure 4).

In addition to the significant interaction effect, there was a significant main effect for Sex, $F(1,168) = 4.04$, $p < .05$, indicating that the fathers obtained higher Total Pathology Scores than the mothers (see Table 17 regarding means and standard deviations on TPS for mothers and fathers).

Code 1-3: The two-point Code 1-3 was then analysed via ANOVA (Appendix V) and indicated a significant main effect for Groups, $F(1,168) = 4.16$, $p < .04$, and a significant Group X Sex interaction effect, $F(1,168) = 4.45$, $p < .04$. The subsequent tests of simple main effects (Appendix V) revealed that the mothers of hyperactive children had higher scores on Code 1-3 than the mothers of normal control children. No differences on Code 1-3 were found between the mothers and fathers of hyperactive children. In addition, the fathers of normal control children were not different from the mothers of normal control children and the fathers of hyperactive children on Code 1-3 (see Figure 5).

Code 2-4: The 2 X 2 X 2 ANOVA for Code 2-4 (Appendix W) revealed a significant Group X Relationship

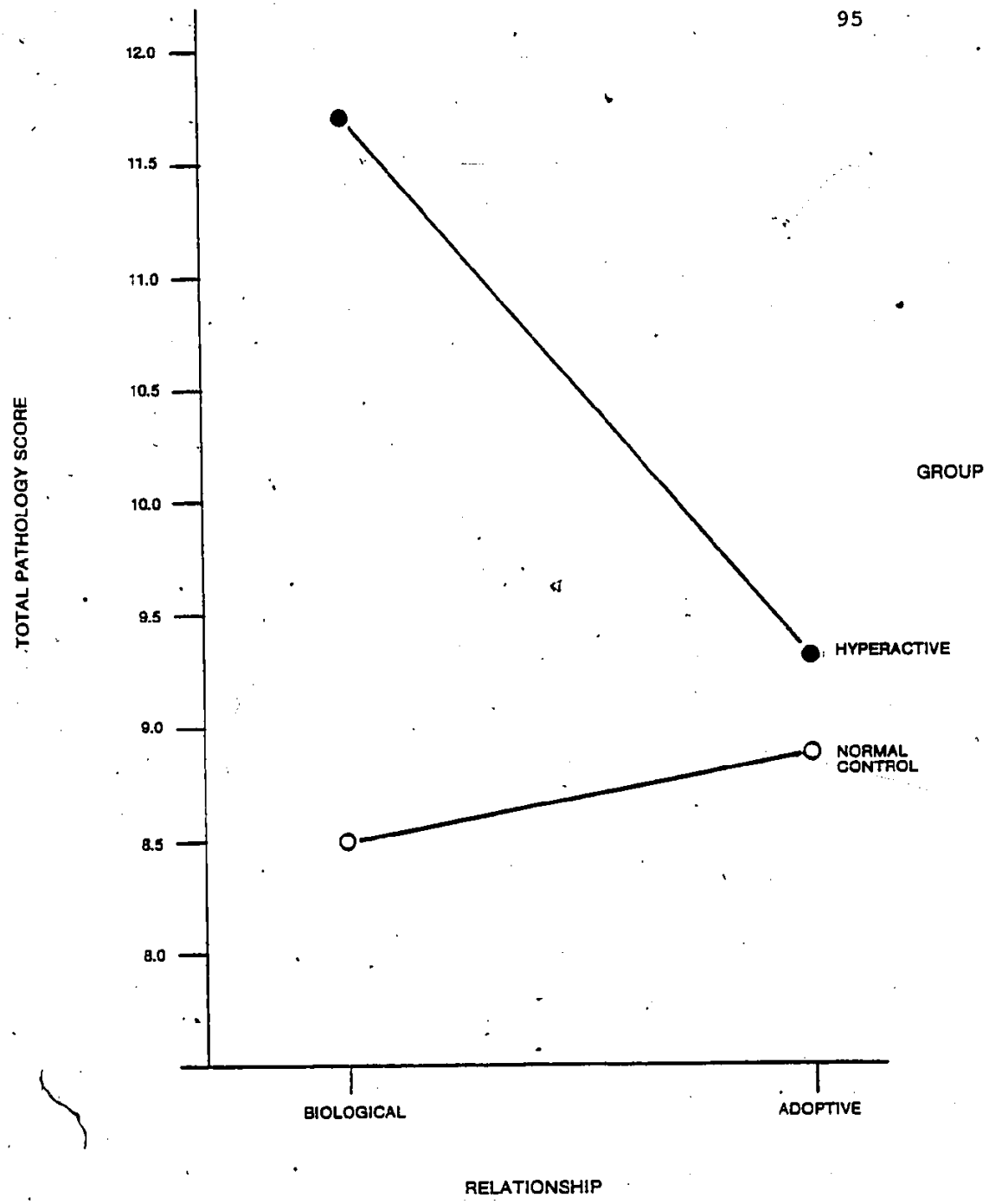

Fig. 4 — Total Pathology Score (TPS) to parental relationship (biological, adoptive) and index group (hyperactive, normal control).

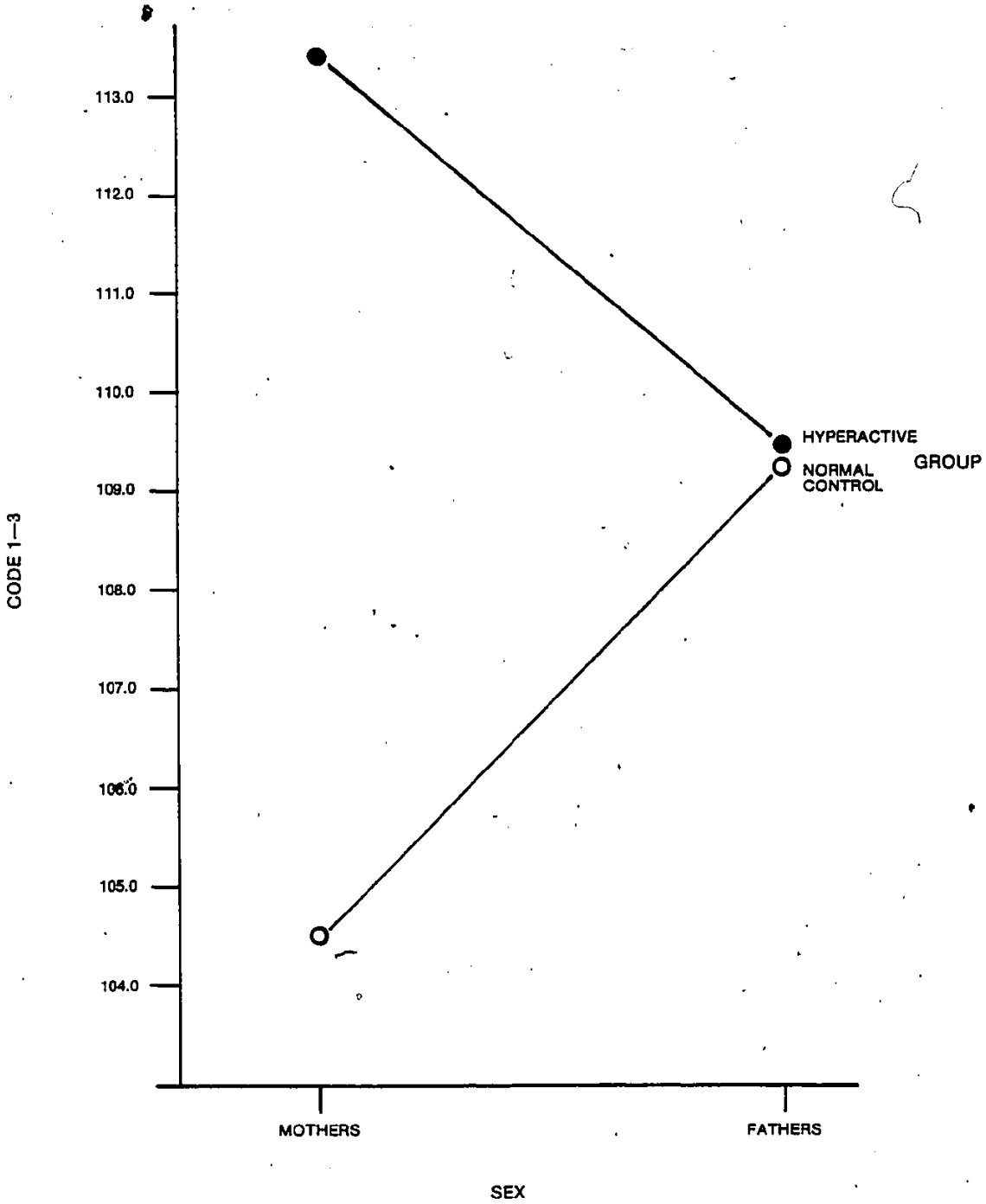

Fig. 5 — Code 1-3/3-1 MMPI Profile (Code 1-3 with combined T-scores) to parents sex (mothers, fathers) and index group (hyperactive, normal control).

interaction effect, $F(1,168) = 12.22$, $p < .001$, a significant main effect for Groups, $F(1,168) = 29.49$, $p < .000$, and a significant main effect for Relationship, $F(1,168) = 5.44$, $p < .02$. A follow-up analysis for simple main effects was then performed. The results of the tests of simple main effects (Appendix W) indicated that the B·HC obtained higher Code 2-4 scores than the B·NCC and the A·HC. The A·NCC were not different from the B·NCC and the A·HC on Code 2-4 (see Figure 6).

Code 2-7: ANOVA results for Code 2-7 (Appendix X) showed a significant Group X Relationship interaction effect, $F(1,168) = 4.46$, $p < .04$, as well as a significant main effect for Groups, $F(1,168) = 10.29$, $p < .002$, and a significant Relationship main effect, $F(1,168) = 7.16$, $p < .01$. In view of the significant interaction effect, tests for simple main effects were conducted. The analysis of simple main effects (Appendix X) revealed that the B·HC obtained higher scores on Code 2-7 than the A·HC and the B·NCC. No differences in Code 2-7 scores were found between the A·NCC and the A·HC or between the A·NCC and the B·NCC (see Figure 7).

Code 4-9: A summary of the ANOVA for Code 4-9 is presented in Appendix Y. The results of the ANOVA revealed a significant interaction effect for Group X Sex, $F(1,168) = 3.95$, $p < .05$, and a significant main effect for Groups, $F(1,168) = 11.57$, $p < .001$. Tests for simple main effects

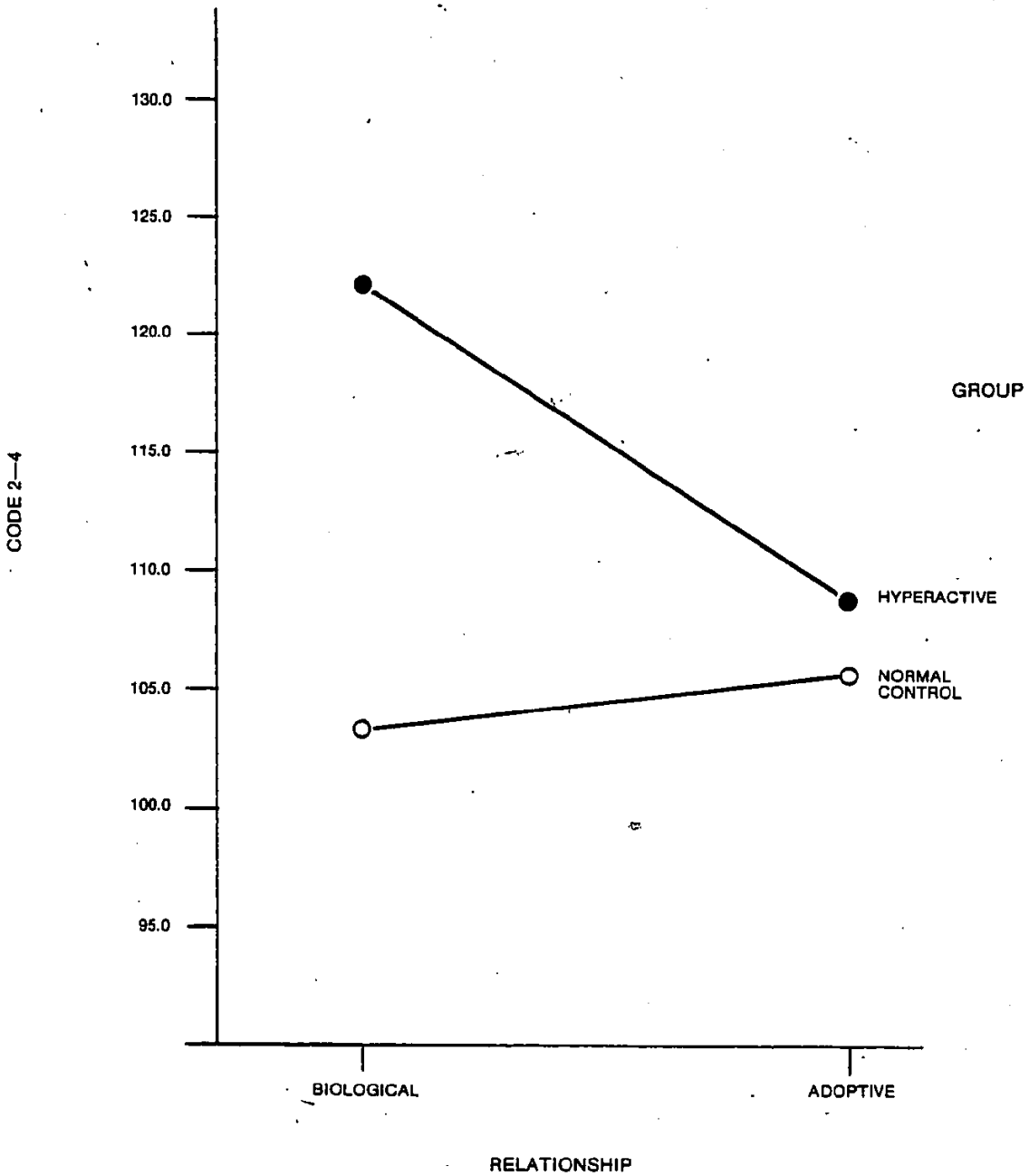

Fig. 6 — Code 2-4/4-2 MMPI Profile (Code 2-4 with combined T-scores) to parental relationship (biological, adoptive) and index group (hyperactive, normal control).

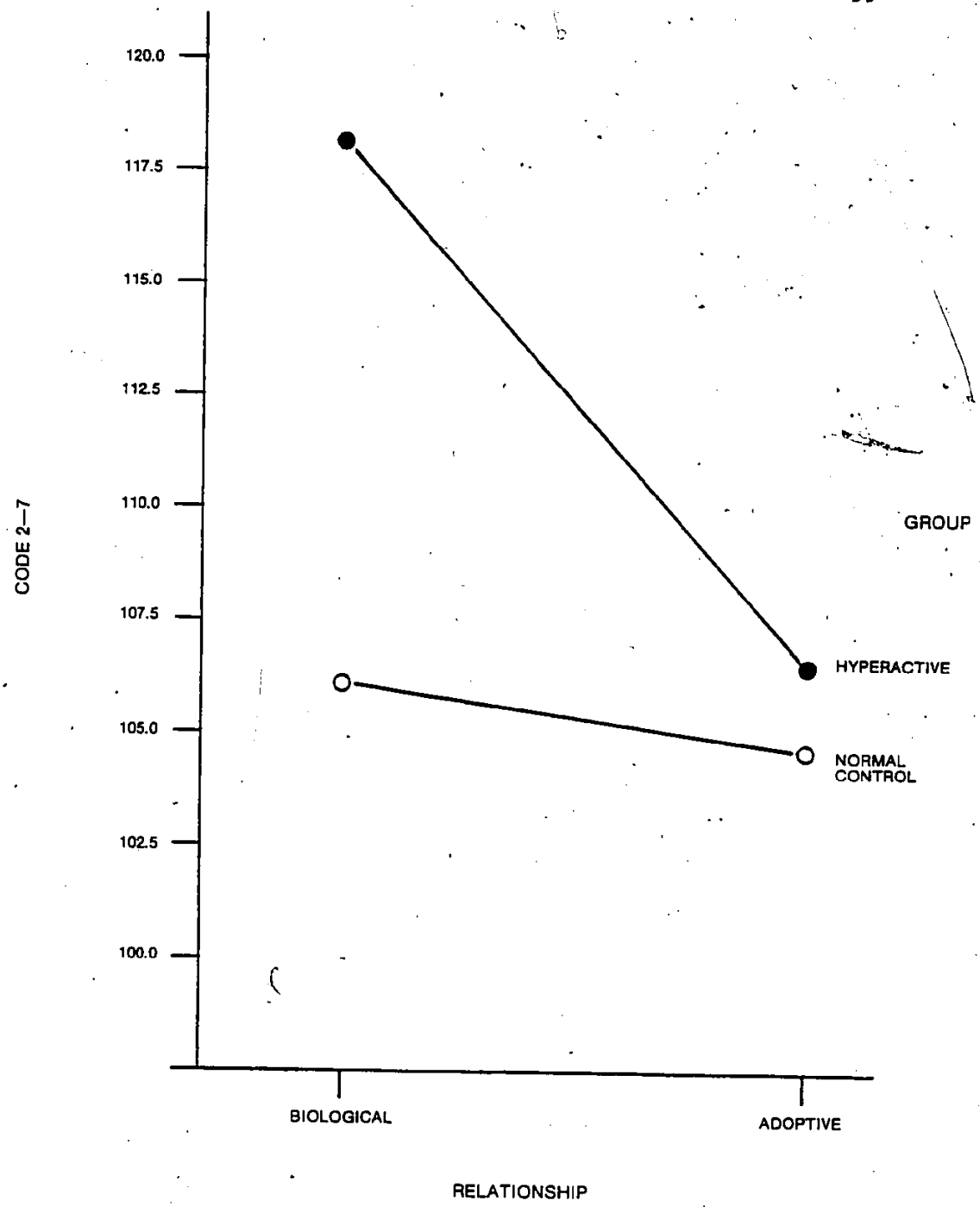

Fig. 7 — Code 2-7/7-2 MMPI Profiles (Code 2-7 with combined T-scores) to parental relationship (biological, adoptive) and index group (hyperactive, normal control).

were conducted in view of the significant interaction effect. The analysis of simple main effects (Appendix Y) revealed that the mothers of hyperactive children were found to have higher scores on Code 4-9 than the mothers of normal control children. No differences in Code 4-9 scores were found between the mothers and fathers of hyperactive children. The mothers of normal control children obtained lower scores on Code 4-9 than the fathers of normal control children. There was no difference on Code 4-9 between the fathers of hyperactive children and the fathers of normal control children (see Figure 8).

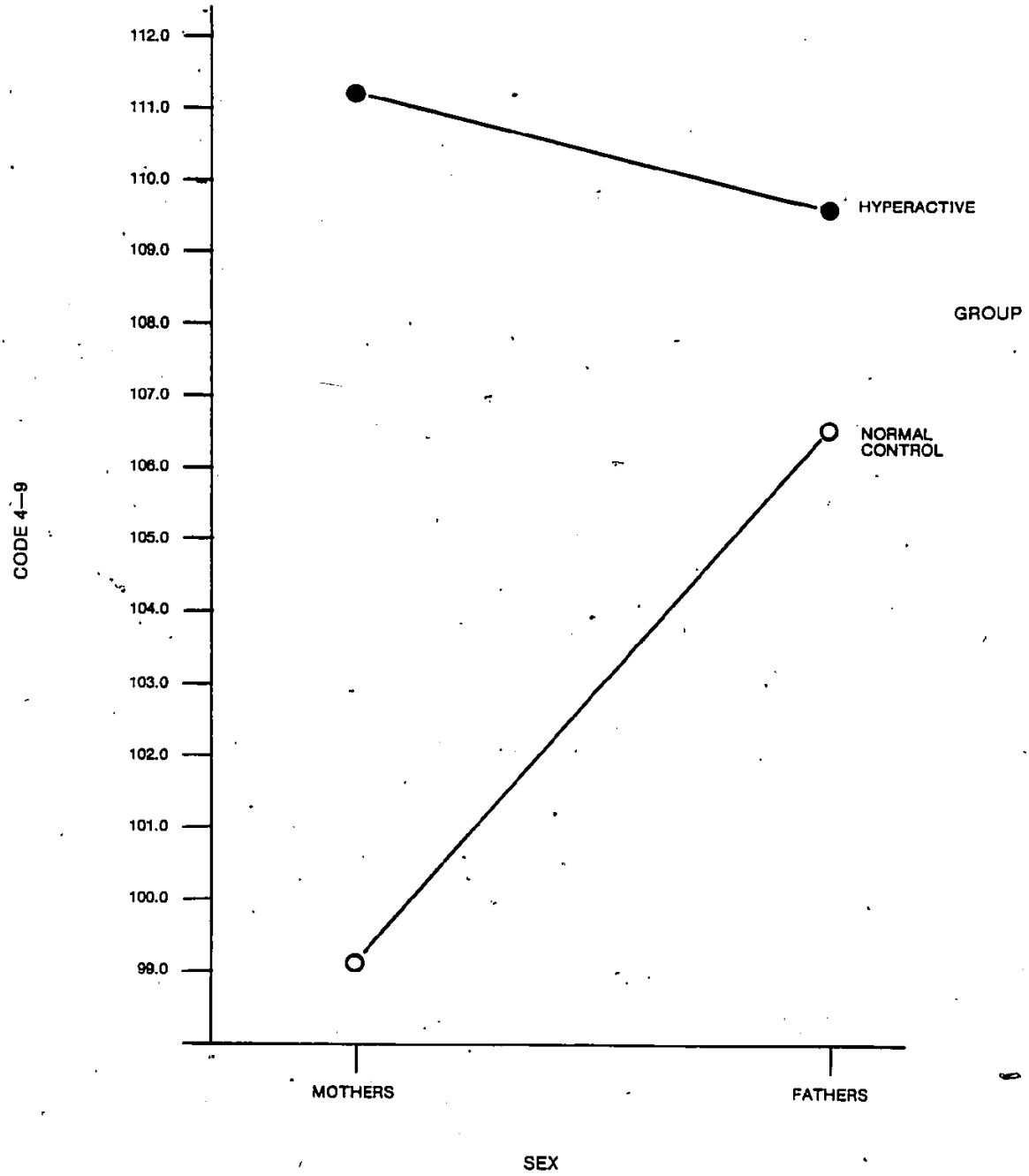

Fig. 8 — Code 4-9/9-4 MMPI profiles (Code 4-9 with combined T-scores) to parents sex (mother, father) and index group (hyperactive, normal control).

CHAPTER V

DISCUSSION

The present research was conducted to investigate specific attention, impulsivity, and personality differences in the biological parents of hyperactive children as compared to the adoptive parents of hyperactive children and the biological and adoptive parents of normal control children. In particular, the study examined objective evidence for the hypothesis proposed by Cantwell (1975a, 1977) and Morrison and Stewart (1973b) that HA is a familial, genetically-linked disorder marked by the presence of adult psychiatric illness, notably sociopathy, hysteria, and alcoholism, in the biological parents of hyperactive children. It was further hypothesized that attentional deficits and impulsivity problems in the biological parents of hyperactives may provide a means of defining the variation at a genetic locus and the simultaneous phenotypic expression of HA.

The present investigation also sought to provide for cross-group comparisons between the parents of hyperactive children and the parents of normal control children. In order to further facilitate examination of gene-environment influences, the design additionally provided for comparisons between the biological and adoptive parents of hyperactive

and normal control children. The Sex factor was originally included in the study as a means of controlling for sex differences between mothers and fathers.

The results of the study provide strong support for the genetic hypothesis proposed in this investigation for the presence of attentional deficits in the biological parents of hyperactive children. As anticipated, the biological parents of hyperactive children exhibited more attentional difficulties than the adoptive parents of hyperactives and the biological parents of controls, 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 and the biological parents of controls on these attention measures.

The hypothesis of greater impulsivity in the biological parents of hyperactive children was not confirmed in the present study.

In general, the results of the analyses of the personality variables indicate the involvement of both genetic and environmental factors. The genetic prediction of more personality disturbances in the biological parents of hyperactive children was partly confirmed with moderate support being found for the claims of Cantwell (1975a) and Morrison and Stewart (1973b) with respect to alcoholism (i.e. higher

elevations on Codes 2-4 and 2-7) in the biological parents of hyperactive children as compared to the comparison groups of parents. As predicted, the biological parents of hyperactive children also evidenced a higher overall level of psychopathology than did the comparison groups. The prediction of more hypervigilant, anxiety approach (sensitizor) responses to threatening stimuli in the biological parents of hyperactive children was not confirmed.

The predictions of lower ego strength and greater psychological impairment with respect to hysteria and sociopathy were not confirmed, thus not supporting the genetic hypothesis. The findings reported for the latter three variables were, in fact, supportive of an environmental explanation. The results suggest that the biological and adoptive parents of hyperactive children experience lower levels of ego resiliency than the biological and adoptive parents of controls. Additionally, the mothers of hyperactives were found to experience greater hysteria and sociopathy or impulse control problems than the mothers of control children. Furthermore, the finding that fathers of hyperactives did not differ significantly from fathers of controls with respect to hysteria and sociopathy, encourages various directional and bi-directional environmental speculations concerning maternal adjustment and HA.

These results will be discussed in the following four sections of the last chapter. The first section provides a

discussion of the demographic measures obtained in the present study and possible problems related to the generalizability of the findings. A discussion of the findings related to the hypotheses stated previously in Chapter I is presented in the second section. Further implications related to the attention and personality results will be discussed in the third section. The final section of the chapter contains some suggestions for future research.

Analyses of the Demographic Variables

The results of the separate analyses of variance for the demographic variables reported in this study revealed significant differences for three of the four population parameters. While there was no difference between the groups with respect to total family income, there were differences in terms of parents' ages, IQs, and total years of education.

Total Family Income. Although no significant difference was found between the groups, the biological parents of hyperactive children appeared to have a somewhat lower family income (mean $27,850) than the adoptive parents of hyperactives (mean $31,055), the biological parents of controls (mean $30,880), and the adoptive parents of controls (mean $32,750). In contrast to the present findings, Morrison and Stewart (1971) and Stewart and his co-workers (1966) found that hyperactive children tended to come from lower income families than nonhyperactive

control children. It is noteworthy that the means of the parent groups reported in this study are comparable to the combined family income for Ontario reported by Statistics Canada (Bulletin Number 13207, 1980) of $28,854 and for the Ottawa-Carleton metropolitan area of $28,290 (Bulletin Number 13207).

Age. The biological parents of hyperactive and normal control children (means 34.84 and 36.68 years, respectively) were found to be significantly younger than the adoptive parents of hyperactive and control children (means 40.86 and 39.63 years, respectively). Morrison and Stewart (1973b) reported similar findings, observing that their sample of biological parents were three to five years younger than the adoptive groups of parents. It is possible to speculate that the age findings in both cases may be explained in terms of the subject sampling procedures.

In the Morrison and Stewart study and the present investigation, the biological index cases were drawn from a public outpatient clinic, while approximately one-half of the adopted index cases were obtained via outpatient clinics and the remaining adoptive cases were solicited from either the private offices of pediatricians (Morrison & Stewart, 1973b) or from response to public media advertisements (present study). An alternative and more likely explanation for the age difference may be that the adoptive parents had waited a longer period of time to conceive their

own child and subsequently also had to undergo a lengthy screening process by the adoption agency.

The mothers in general were also found to be younger than the fathers. Similar sex differences in parental ages have been reported in family studies by Miller and Keirn (1978) and Morrison and Stewart (1973b).

IQ. The biological parents of hyperactive children were found to obtain significantly lower scores of intellectual functioning (mean 107.66) than the adoptive parents of hyperactives (mean 114.44) and the biological parents of controls (mean 115.52). The adoptive parents of controls (mean 116.93) did not differ from the adoptive parents of hyperactives and the biological parents of controls with respect to intellectual functioning. While the scores of the biological parents of hyperactive children fell within the average range of intelligence, the parents in the three comparison groups obtained scores in the high average range of intelligence. Similar trends have been reported in the literature with respect to hyperactive children obtaining lower IQ scores than control children (Ross & Ross, 1976).

The IQ finding reported in this study might be explained in terms of the different sample solicitation procedures discussed previously. The biological and adoptive parents of the control children were selected from the medical records department of C.H.E.O. (i.e. 25 biological and two adoptive control families or 60%) and from response to

media advertisement (i.e. 18 adoptive control families or 40%). The higher intellectual abilities of the two control comparison groups might be due to the fact that these groups were drawn from a non-clinical population wherein the more informed and intellectually curious individuals in the invited sample might be more willing to accept voluntary involvement in the study. However, the sampling explanation is not supported by the finding that while the adoptive parents of hyperactives achieved higher overall levels of intellectual functioning than the biological parents of hyperactives, both of these groups were selected predominately from the assessment files of the Department of Psychology, C.H.E.O. (i.e. 25 biological and 17 adoptive families of hyperactives or approximately 98%, with one adoptive family selected in response to media advertisements).

In addition, it was found that parents of adopted children and parents of children involved in a treatment program for HA were more likely to accept participation in this study. In a comparison of the accepting sample to the invited sample who met the previously defined inclusion criteria, it was noted that 25 of the 32 (approximately 78%) invited biological parents of hyperactive children agreed to participate as compared to 18 of the 20 (90%) adoptive families of hyperactives and 20 of the 33 (approximately 61%) adoptive families of normal controls. The biological families of normal controls were somewhat less likely to

accept participation with 25 of the 65 (approximately 39%) invited sample of families agreeing to involvement in the study. It is possible that parents who have made a personal committment and emotional investment in adopting a child and parents who have already chosen involvement in and adherence to a treatment program for their child are more likely to be motivated to voluntarily participate in a study than would the biological parents of non-clinic control children.

It was surprising that mothers obtained significantly lower scores of intellectual functioning than fathers. The reasons for the IQ differences between mothers and fathers are not clear. Fathers in general also completed more years of schooling than mothers. The additional years of education may provide more experience in test-taking which would result in fathers obtaining higher scores on the IQ measure.

Education. The biological parents of hyperactive children completed fewer years of education (mean 12.06) than the adoptive parents of hyperactives (mean 14.42) and the biological parents of controls (mean 14.60). The adoptive parents of control children (mean 15.13) were not found to differ from the adoptive parents of hyperactives and the biological parents of controls with respect to years of education.

Miller and Keirn (1978) reported similar findings with respect to the parents of mentally retarded and emotionally

disturbed children completing fewer years of education than the parents of non-clinic control children. Nahon (1982), on the other hand, found that the parents of hyperactive children completed fewer years of education than the parents of asthmatic as well as the parents of normal controls. Insofar as education is probably associated with family occupational level and IQ, Morrison and Stewart (1973b) observed that the adoptive parents of hyperactive children were significantly higher in family occupational status than the biological parents of hyperactives. The investigators also noted that while the biological parents of control children were higher in occupational status than the biological parents of hyperactives, this difference was not significant. In contrast to the present, Campbell and Redfering (1979) found no educational differences between parents of hyperactive and nonhyperactive children.

Another interesting finding was that fathers in general completed more years of education than mothers. A similar trend was reported by Miller and Keirn (1978) with respect to fathers of emotionally disturbed, mentally retarded and normal control children having more years of schooling than mothers.

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.

In general, there is a concern about the effect of the pre-test family differences with respect to IQ and education level in the groups. The finding that the biological parents of hyperactive children completed fewer years of education and obtained lower IQ scores than the three comparison groups of parents may reflect demographic differences generally observed in clinic populations of families of retarded, emotionally disturbed, and behaviour problem children. However, it is presently unclear as to whether the IQ and educational differences may be the cause or the outcome of the attentional and MMPI findings reported in this study. In view of the infrequent and often contradictory findings with respect to family background factors, future studies might examine and report on this important data.

Analyses of the Dependent Variables

The results of the correlational analyses indicated that there were no significant associations between the impulsivity and attention variables or between the impulsivity and personality variables. In addition, only a few, low correlations were found between the personality and attention variables.

In an extensive review of psychophysiological research involving hyperactive children, Hastings and Barkley (1978)

observed that a number of investigators (Langhorne, et al., 1976; Routh & Roberts, 1972; Ullman, et al., 1978) have found that various measures of inattentiveness, impulsivity, and physical overactivity do not appear to significantly intercorrelate or cluster. The lack of significant associations between measures of attention and impulse control may indicate that the measures are tapping separate and independent central processes and deficits related to HA. Hastings and Barkley also suggest that the weak intercorrelations between measures of the core symptoms of HA might be due to the behavioural heterogeneity of hyperactive children.

Hypothesis 1 - Attention Variables. The first set of predictions were based on the genetic model that the biological parents of hyperactive children will evidence more attentional difficulties than the adoptive parents of hyperactives and the biological parents of controls, as indicated by slower mean reaction times and more extraneous responses on the DRT task and by a reduction in correct recognitions with increasing matrix sizes of one, five, and nine letters on the Span of Apprehension task. It was further anticipated that the adoptive parents of controls will not differ from the adoptive parents of hyperactives and the biological parents of controls on the various attention variables.

The results provide strong support for the genetic hypothesis of attentional difficulties with respect to three of the five attention variables. The biological parents of

hyperactive children evidenced slower mean reaction times on the DRT task and achieved fewer correct recognitions on the Span task for matrix sizes of five and nine letters than the adoptive parents of hyperactives and the biological parents of normal controls. As anticipated, the adoptive parents of control children did not differ from the adoptive parents of hyperactives and the biological parents of controls in terms of mean reaction time and number of correct recognitions of matrix sizes of five and nine letters.

Results indicate that the mean reaction times of the biological parents of hyperactives were about 13% slower than the adoptive parents of hyperactives, and about 16% slower than the biological and adoptive parents of controls. Interestingly, the mothers in the present study were found to be consistently slower than the fathers, suggestive of sex differences in reaction time. The performance of the biological parents of hyperactives with respect to slower mean reaction times on the DRT task indicates difficulties in sustaining attention. These results are consistent with the findings of Cohen and her co-workers (1973), Firestone and Martin (1979), and Zahn and his co-workers (1975) who found poorer attention in hyperactive children as compared to normal control children.

However, the biological parents of hyperactives did not differ from the other groups of parents in terms of

extraneous responses on the DRT task, a variable which has been found in some previous research to distinguish hyperactive from normal control children (Firestone & Douglas, 1975; Firestone & Martin, 1979). It is not altogether clear why a higher number of extraneous responses was not found in the present investigation. Douglas and Peters (1979) noted that the increased incidence of extraneous responses on the DRT task by hyperactive children suggests that once hyperactives have been primed to respond, they may experience more difficulty than normal children in impulse control and motor inhibition of anticipatory and redundant responses. While the biological parents of hyperactives did not differ from the comparison groups of parents with respect to extraneous responses, the biological parents of hyperactives scored more extraneous responses than the control groups, indicating a response pattern in the anticipated direction. The lack of significant differences between the parent groups may be due to the fact that the measure was not sufficiently strong enough to lead to differences in an adult population.

The comparisons of the occurrence of correct recognitions on the Span of Apprehension task indicates that the biological parents of hyperactives did not differ from the other groups of parents when no distraction or noise letters were present (Span 1). The lack of differences may be accounted for by the fact that the biological parents of hyperactives were as able to maintain attention and select

the relevant target letter as the parents of the comparison groups when no noise letters were present because of the relative ease of the task. Thus, Span 1 may serve as a basal score for the Span task. However, as the number of noise letters increased with matrices of five or nine letters, the number of correct recognitions decreased for the biological parents of hyperactives as compared to the other groups of parents. In addition, there was an increased reduction in the number of correct recognitions as the matrix sizes increased from five to nine letters, suggesting that when more noise letters are present, the biological parents of hyperactive children evidence even greater difficulty in maintaining attention when visual search tactics are required.

In a recent investigation, McIntyre and his co-workers (1978) have rejected the distractibility explanation for the decrease in correct recognitions on the Span task in hyperactive boys as compared to control boys. The investigators are currently pursuing two alternative explanations that either the decaying afterimage on the Span task fades more rapidly for hyperactive boys than for control boys or the information pickup from the decaying afterimage may be much slower for hyperactive boys.

In general, these findings suggest that the performance of the biological parents of hyperactive children on the DRT task and the Span of Apprehension task is similar to that

reported in previous research involving hyperactive children, indicating that the biological parents of hyperactives may evidence comparable attentional difficulties. The deficits in attentional processes in the biological parents of hyperactives were noted on tasks involving both protracted vigilance and visual search strategies.

Several investigators have attempted to demonstrate that the core symptomatology of HA, in terms of attentional deficits, impulse control problems, and physical overactivity, is related to a central disorder of arousal mechanisms. Three sets of theories have been posited, as follows: (1) hyperactive behaviours result from physiological overarousal which arises from an inability to effectively filter incoming sensory stimuli and to employ inhibitory mechanisms (Friebergs & Douglas, 1969; Laufer, et al., 1957; Strauss & Lehtinen, 1947); (2) hyperactive responses typify stimulus-seeking behaviour due to physiological underarousal (Rosenthal, 1973; Satterfield & Dawson, 1971; Zentall, 1975); or (3) some hyperactive children are physiologically underaroused whereas others are overaroused (Wender, 1971; Zentall & Zentall, 1976). A fundamental concept underlying the various theories involves an assumed homeostatic mechanism which seeks to maintain a moderate state or optimal level of arousal. Hasting and Barkley's (1978) review of studies involving autonomic measures of arousal concludes that there is little support

for the overarousal or filter interpretation. The reviewers further note that while hyperactive children do not differ from normal children in their resting levels of autonomic functioning, hyeractive children are probably underreactive or underarousable in conditions involving environmental stimulation, indicating tentative support for the stimulus-seeking or underarousal interpretation.

Douglas and Peters (1979) proposed that studies of the effects of stimulant medication and reinforcement contingencies during vigilance or search task performance (i.e. attentional processes) are applicable and germane to arousal hypotheses in hyperactive children. Hastings and Barkley (1978) have also pointed out that studies of the effects of stimulant drugs indicate that the stimulants appear to 'energize or increase the arousal' of hyperactive children.

It is possible that the attentional difficulties noted in the biological parents of hyperactives in this study may be due to similar underarousability to environmental stimulation or to a deficiency in the ability to respond optimally to such stimulation. What is unclear at present is whether these attentional difficulties represent a central attentional deficit or are the result of other psychological factors effecting attention and concentration.

Hypothesis 2 - Impulsivity Variables. The second set of predictions stated that the biological parents of hyperactive children will evidence more impulsivity as per more

contacts and longer duration of contact between the stylus and the edge of the channel as well as a faster total time to completion on the Porteus Maze task than the adoptive parents of hyperactives and the biological parents of controls. It was also asserted that the adoptive parents of controls would not differ from the adoptive parents of hyperactives and the biological parents of controls on the various impulsivity variables. Based on the genetic model, it was anticipated that the performance of the biological parents of hyperactive children would resemble that of hyperactive children in general on the Maze task. The Porteus Maze task has demonstrated differences between hyperactive and control children (Firestone & Martin, 1979; Parry, 1973) and has been found to be sensitive to the effects of stimulant drugs (Conners, 1973; Knights & Hinton, 1969).

The present results failed to support the second set of predictions that the biological parents of hyperactive children would exhibit more impulse control problems. In fact, there were no differences on the three impulsivity variables between the four groups of parents.

Although the biological parents of hyperactive children did not differ significantly from the comparison groups of parents on the impulsivity variables, the biological parents of hyperactives produced responses in the expected direction in terms of both hastier execution and more contacts on the Maze task. These findings suggest that the Porteus Maze

task may not be a sufficiently sensitive measure for detecting impulse control deficits in an adult population of parents of hyperactive children.

An alternative explanation of the test results from the impulsivity measure is that the ability of the biological parents of hyperactive children to inhibit impulsive responding has improved via maturation and experience (Wright & Vliestra, 1975). Thus, the biological parents of hyperactive children now display a more reflective, motor inhibitory approach which more closely approximates the performance style of the comparison groups of parents. Conceivably, performance on more difficult motor tasks of impulse control would lead to a difference in the predicted direction, similar to the results on the Span of Apprehension task. Nonetheless, within the limits of this measure, no evidence exists for such a difference.

Hypothesis 3 - Personality Variables. The third hypothesis stated that the biological parents of hyperactive children would exhibit more personality disturbances than the adoptive parents of hyperactives and the biological parents of controls, as evidenced by higher profile elevations on four two-point codes of the MMPI, lower Ego Strength (Es) scores, higher Repression-Sensitization (R-S) scores, and higher Total Pathology scores (TPS). The predictions of higher elevations for the biological parents of hyperactives on the four two-point codes were stated to test

the genetic claims of Cantwell (1975) and Morrison and Stewart (1973) for the greater incidence of hysteria (Code 1-3), alcoholism (Codes 2-4 and 2-7), and sociopathy (Code 4-9). It was anticipated that the comparison groups of parents would not differ from each other on the various personality variables.

The results partially confirmed the predictions for the genetic hypothesis of more personality disturbances in the biological parents of hyperactive children with respect to significant differences in three of the seven personality variables. The biological parents of hyperactives obtained higher profile elevations on the alcoholism codes as well as higher Total Pathology scores than the adoptive parents of hyperactives and the biological parents of controls. As predicted, the comparison groups of parents did not differ from each other on these variables.

These results also indicate that the biological parents of hyperactive children experience a mild degree of depression in comparison the the other groups of parents. A primary emotion underlying scale 2 (Depression), which is integral to both of the significant two-point profile elevations, is a profound sense of loss of either a love object, or more generally a loss of health, self-esteem, or social position. The elevation of the Code 2-4 pair indicates that the basic sense of loss and life dissatisfaction may be dealt with extrapunitively, by tending to externalize and

blame others for sensed failures and losses. The significantly higher elevation of Code 2-4 also indicates that the biological parents of hypractives may become more easily frustrated by their failures to achieve, more resentful of demands placed on them by others, less able to effectively moderate their impulses, and more prone to alcohol and drug addiction in times of stress, as compared to the other groups of parents.

In contrast to the Code 2-4 type, the elevation of the Code 2-7 pair indicates a tendency to respond to stress and loss experiences intropunitively, via excessive worrying, perfectionism, and self-blame for all their life problems. The results obtained on the 2-7 code indicate that the biological parents of hyperactives are more apt to employ obsessional dynamics, intellectualization, and repression in order to defend against feelings of anxiety, guilt, and inadequacy. The biological parents of hyperactives are also more likely to express strong achievement needs and to be susceptible to the accumulation of life stresses, tending to become more tense, agitated, clinging, and depressed than the other groups of parents, as well as to have more problems with alcohol when experiencing additional stress.

The results with respect to the Total Pathology scale indicate that the biological parents of hyperactives exhibit a lower overall level of personality adjustment, compared to the comparison groups of parents. It was noted that there

was a Sex factor difference on the TPS, indicating that fathers in general exhibited higher levels of psychological distress than mothers.

The results of the 2-4 and 2-7 code pairs on the MMPI indicate partial confirmation of the genetic claims of Cantwell and of Morrison and Stewart with respect to a higher incidence of alcoholism in the biological parents of hyperactive children. Independent investigators have found that individuals with high point pairs of 2-4 (Clopton, 1978; Gynther, Altman, & Sletten, 1973) and 2-7 codes (Caster & Parsons, 1977) are frequently identified as alcoholics or problem drinkers. However, profile elevations of these two code pairs are only moderately predictive of alcoholism per se. Hodo and Fowler (1976), for example, found that only 21% of the 1,009 male alcoholics in their investigation obtained a 2-4 high-point pair.

In addition, there is a question of the specificity of the association between alcoholism and childhood HA. While the biological parents of hyperactives seem to be at higher risk for alcoholism as suggested by the higher profile elevations on the 2-4 and 2-7 codes, this association may not be specific to hyperactive index groups. Morrison (1980) reported that while the parents of hyperactive children exhibited a significantly higher incidence of alcoholism than the parents of normal controls, the parents of hyperactives did not differ from the parents of nonhyperactive psychiatric

children with respect to the incidence of alcoholism. Morrison further noted that the issue of specificity of an association between childhood HA and alcoholism reported in previous investigations is questionable. Thus, the findings of the present study regarding higher elevations on the alcoholism codes and possible associations of alcoholism risk in the biological parents of hyperactive index children are further moderated by the fact that a comparison group of parents of psychiatric or other clinic index groups was not included.

The results reported in this paper do not support the genetic predictions that the biological parents of hyperactive children will exhibit lower Es scores, higher R-S scores, and higher elevations on the hysteria and sociopathy codes than the other groups of parents. Indeed, the findings reported on these variables are supportive of an environmental explanation. On the Es scale, the biological and adoptive parents of hyperactive children obtained lower scores than the biological and adoptive parents of controls, indicating that parents of hyperactives experience somewhat less ego resiliency and feelings of adequacy and vitality. This finding may reflect the effect of on-going psychological strain experienced by both the biological and adoptive parents in their efforts to cope with their hyperactive children. Other interesting findings relate to the fact that mothers generally obtained lower Es scores and higher R-S

scores than did fathers. The higher R-S scores tentatively suggest that mothers may tend to be somewhat more prone to respond to threatening stimuli by maintaining a hypervigilant attitude and by engaging in anxiety approach (sensitizor) behaviours of intellectualization and obsessional worry. The anxiety approach behaviours and hypervigilance may, in turn, cause mothers to experience somewhat higher levels of anxiety and adversely effect their general capacity for personality integration or ego strength in comparison to fathers.

The results of this study do not support the genetic hypothesis proposed by Cantwell and by Morrison and Stewart with respect to a higher incidence of hysteria and sociopathy in the biological parents of hyperactive children. The evidence resported here is, in fact, quite contrary to what was anticipated vis-à-vis the genetic theory. The present findings indicate that environmental factors are associated with increased hysteria and sociopathy in the biological and adoptive mothers of hyperactive children.

The results obtained on the hysteria code indicate that the biological and adoptive mothers of hyperactives are more likely to employ the defense mechanisms of denial, repression, and externalization of blame in response to emotional or interpersonal problems in contrast to the biological and adoptive mothers of control children. In addition, the mothers of hyperactives are slightly more prone to using

somatic complaints to avoid dealing with or thinking about their psychological distress.

The examination of the individual scales, however, reveals that the scores obtained on scale 1 (Hypochondria) by the adoptive mothers of hyperactives (mean 51.00) fell in between the scores of the biological and adoptive mothers of controls (means 49.96 and 52.05, respectively), with all three groups of mothers obtaining lower scores than the biological mothers of hyperactives (mean 55.92). On scale 3 (Hysteria), the adoptive mothers of hyperactives obtained a mean score (54.18) which fell in between the scores of the biological mothers of hyperactives (mean 61.72) and the biological and adoptive mothers of controls (means 52.00 and 53.50, respectively). The fathers of control children did not differ from the fathers of hyperactives and the mothers of controls on the hysteria code. In addition, the mothers of hyperactives did not differ from the fathers of hyperactives on this variable.

Overall, the results of the analyses of the sociopathy code indicate that the biological and adoptive mothers of hyperactives are mildly more resentful about their life situation than the mothers of normal controls, and tend to have less regard for social standards and to experience more interpersonal difficulties. The mothers of hyperactive children are also somewhat more irritable, hostile, and impulsive, tending to act out more frequently due to their

lower frustration tolerance. The examination of the separate scales indicates that the biological and adoptive mothers of hyperactives obtained higher elevations on scale 4 (Psychopathic Deviate) with means of 60.00 and 55.61, respectively, than the mothers of control children with means of 48.32 and 52.05, respectively. However, on scale 9 (Hypomania), the biological mothers of hyperactives obtained a lower score (mean 52.56) than the adoptive mothers of hyperactives (mean 53.67), with the biological and adoptive mothers of controls obtaining the lowest scores on this scale (means 48.40 and 49.95, respectively). Furthermore, while the fathers of hyperactives did not differ from the fathers of normal controls and the mothers of hyperactives on the sociopathy code, the mothers of control children obtained significantly lower scores on the sociopathy code than the fathers of controls. This suggests that biological and adoptive mothers of control children are more prone to comform to social codes of conduct and to have fewer impulse control problems than the fathers of controls as well as the mothers of hyperactives.

In general, the results of the hysteria and sociopathy codes and the Es scale comparisons suggest that some form of environmental interaction is involved. Alternative explanations of these findings might involve directional and bi-directional speculations about these personality differences in parents of hyperactive children and familial

factors of living with a chronically problematical child. Environmental theorists might argue that the development of hyperactive behaviours in the child is related to the mother-child relationship and family environment. In particular, childhood HA may be viewed as a psycho-social outcome of mother-child modelling or the emotional-behavioural sequelae of living with mothers who evidence hysteria and sociopathy and parents who have lower ego strength and experience feelings of inadequacy.

An alternate environmental explanation would suggest that personality differences found in parents of hyperactive children represent a psychological reaction to the stress of parenting a difficult child. That is, the psychological stresses attendant to raising and living with a child who exhibits long-standing behaviour problems may adversely affect a parents' overall capacity for personality integration and ego resiliency. In addition, mothers of hyperactives may, by their unique social and familial position as primary care-giver to the hyperactive child, be particularly susceptible to these stresses. The maternal stress would, in turn, be manifested by more repressive defenses, more passive-dependent modes of interpersonal behaviour, and denial of emotional problems as well as by more impulse control difficulties and lower frustration tolerance for the mothers of hypractives as compared to the mothers of control children.

Implications

The most important finding of the present investigation is that the biological parents of hyperactive children evidenced significantly more attentional difficulties than the adoptive parents of hyperactives and the biological parents of controls. Strong support is found for the argument that there is a specific association between childhood HA and attentional deficits in the biological parents of hyperactives. Furthermore, the finding that the adoptive parents of control children did not differ on the various attention variables from the adoptive parents of hyperactives and the biological parents of controls lends additional support to a genetic bases for the familial association of attentional problems.

In addition, 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, including both protracted vigilance and visual search strategies. The performance of the biological parents of hyperactives in this study is similar to previously reported findings for hyperactive children, insofar as the biological parents evidenced slower mean reaction times on the prolonged vigilance task and fewer correct recognitions with increasing matrix size on the visual search task. The findings reported in the present study strengthen the view that HA is a familial condition

which is related to a deficit in attentional processes.

There is, however, an issue of the specificity of the relationship between HA and attentional deficits. Attentional difficulties have been reported in a variety of pathological conditions, including schizophrenia (Neale, et al., 1969; Venables, 1964), hysteria (Horvath, Friedman, & Meares, 1980), and sociopathy (Quay, 1977).

Recently, Firestone and Martin (1979) addressed the issue of specificity via a cross-group comparison study of the cardinal deficits of short attention span, low frustration tolerance, impulsivity, and physical overactivity associated with childhood HA. The investigators found that attention deficits were the only diagnostic characteristic which clearly distinguished hyperactive children from other pathological groups of asthmatic and behaviour problem children as well as from normal control children. Although the hyperactive children differed from the normal control group with respect to all four of the diagnostic criteria, Firestone and Martin found that hyperactives share problems of impulse control and low frustration tolerance in common with both asthmatic and behaviour problem children. Thus, it is suggested that impulsivity and frustration tolerance problems may not be uniquely associated with HA. Rather, it is possible that these two characteristics are manifested by a variety of childhood pathological groups.

No evidence was found in the present study to support a genetic prediction that the biological parents of hyperactive children differ from the comparison groups of parents with respect to impulse control. Several studies have reported that the Porteus Maze task has been found to be differentially sensitive to the effect of stimulant drugs (Knights, 1974; Sroufe, 1975) and to successfully differentiate hyperactives from normal control children. However, as noted previously, impulsivity may be viewed as a characteristic which is non-specifically manifested by a number of pathological groups of children. Additionally, it is possible to speculate that impulse control may be moderated by a variety of learning and maturation factors (Meichenbaum & Goodman, 1969; Palkes et al., 1968).

Attentional problems, in contrast to impulse control difficulties, may represent a more central, unique, and specific deficit in hyperactive children which, in turn, persists into adulthood. Wender and his co-workers (1981) noted that the persistence of this central deficit into adult life is formally identified as Attention Deficit Disorder (ADD), residual type. The speculation of chronic attentional problems related to HA raises several important questions: (1) How is the attentional deficit and its residual forms associated with psychopathology in children and in adults? (2) What are the difficulties encountered

when moving from an identified, specific deficit, such as attention, to a more global concept of personality?

In general, the findings of the personality variables reported in this study suggest a complex interaction of both genetic and environmental factors. The biological parents of hyperactive children significantly differ from the comparison groups of parents with respect to a greater tendency toward alcoholism and a lower overall level of psychological adjustment. More generally, the biological parents of hyperactive children experience a fundamental sense of sadness and loss, manifest lower frustration tolerance, and exhibit a more anxious, tense, perfectionistic coping style involving obsessive-compulsive defense mechanisms.

It is interesting to speculate about the relationship between the attentional and personality difficulties in the biological parents of hyperactive children and the psychopathology of hyperactive children. The attentional deficits observed in the present study in the biological parents and in the literature for hyperactive children would tend to implicate the stimulation seeking or underarousal interpretation of the disorder. The underarousal line of thought would suggest that these individuals tend to be underaroused by environmental stimuli and therby more prone to increased activity, less able to focus and sustain attention, and to inhibit inappropriate responses. In addition, Wender (1972)

has suggested a second related primary deficit involving a diminished capactiy for positive and negative affect, which involves a lowered responsiveness to consequences and a diminished ability to experience pleasure and pain.

Wender further speculates that these two central deficits are chronic in nature and due to a disorder of monoamine metabolism. In turn, he argues that the primary deficits give rise to a complex set of secondary behavioural, social, and emotional problems. The proposed observable, secondary characteristics include, for example, lowered self-esteem, impaired academic functioning, increased anxiety and guilt, refractoriness to socialization and delinquency, and impulsivity. Thus, the lower self-esteem, poorer school performance, and social difficulties observed in hyperactive children and adolescents may stem from the primary attentional and stimulus-processing difficulties which may, in turn, be viewed as forerunners of adult psychopathology.

The mild level of depression, anxiety, lower frustration tolerance, and tension as well as the higher level of psychopathology and tendency toward alcoholism found in the biological parents of hyperactive children in this study would tend to support this view. On-going problems of raising a problematic child who exhibits similar attentional and personality characteristics might tend to place additional stresses on the biological parent who is already

experiencing social and personality distress. Attentional deficits observed in the biological parents of hyperactives would thus be viewed as the phenotypic expression of the central disorder which persists, at least in some instances, on a genetic basis. The personality differences noted in the biological parents of hyperactives are then viewed as secondary behavioural and social characteristics which are attendant to a central attentional deficit. The lower overall level of intellectual functioning and fewer years of education found in the biological parents may represent similar problems in sustaining attention and sticking to the task.

In contrast to previously reported family studies of HA, no support was found in the present study for a higher incidence of hysteria and sociopathy in the biological parents of hyperactives. Alcoholism, on the other hand, was the only previously reported adult psychopathology to be found in the biological parents of hyperactive children. Of additional interest is the suggestion by Cadoret and his co-workers (1980) that some types of alcoholism may also be caused by a metabolic defect. Thus, HA may share similar etiological factors with alcoholism, although the specificity of this similarity is questionable.

In addition to the familial, and possibly genetic, factors, various environmental factors were also seen to be central and germane to the personality functioning of the

parents of hyperactive children. The biological and adoptive parents of hyperactives tend to experience less ego resiliency in dealing with life stresses as compared to the parents of normal control children. The mothers of hyperactives appear to be particularly effected by the stresses of coping with a problem child. Mothers of hyperactive children were more prone to hysteria and sociopathy than mothers of normal controls. In general, the mothers of hyperactives tend to utilize a greater degree of repression and denial and to convert their psychological problems into physical complaints than mothers of normal controls. The mothers of hyperactives also exhibit a greater tendency to externalize blame for their problems, to be more irritable and easily frustrated, and to be more likely to act out their distress behaviourally and to be less socially conforming than the mothers of control children. The fathers of hyperactive children were interestingly not found to differ from the fathers of control children on these personality variables. Thus, bi-directional speculations might view aspects of childhood HA as a complex interplay of two-way interactions between parent and child and particularly between mother and child.

These findings of differences in the attention and personality characteristics of parents of hyperactive children are of theoretical and applied importance. Theoretically, the results underscore the importance of further

investigations of the attentional and personality characteristics of the biological parents of hyperactive children based on the genetic model. In addition, the results also indicate the need for more work to investigate the personality functioning of the biological and adoptive parents, particularly the mothers, of hyperactives from the standpoint of family interaction and environmental models. The findings are of particular relevance with respect to clinical application. The early identification of attentional difficulties and problems in personality adjustment in parent and child would assist the mental health practitioner in family assessment as well as in establishing appropriate therapeutic and counselling plans to meet particular individual, couple, or family needs.

Future Directions

The present study sought to investigate specific attention, impulsivity, and personality characteristics of parents of hyperactive children as compared to parents of normal control children via the use of more objective behavioural and self-report measures. A logical outgrowth of the present study would be to investigate these parental characteristics in terms of cross-group comparisons involving parents of nonhyperactive psychiatric index cases as well as parents of normal controls. The design of this study did not allow for the assessment of the specificity of these characteristics in the biological

families of hyperactives as compared to the families of other child clinical populations.

In addition, research is merited to further examine the familial association in the primary attentional and the affective characteristics of the hyperactive child and the biological parents and siblings. Cross-fostering studies involving the biological and adoptive parents of the same hyperactive child would assuredly provide more definitive analysis of the influence of gene-environment interaction. However, ethical, legal, and pragmatic considerations may necessitate continued comparisons between the biological and adoptive parents of different groups of hyperactive children.

The results of the present study regarding attentional difficulties, and a posited underarousal problem, in the biological parents of hyperactives can be extended by studying the performance of the biological parents and siblings on a variety of psychophysiological measures. Hastings and Barkley (1978) have observed that studies utilizing cortical and neurophysiological measures have indicated that hyperactive children in some instances may be underaroused when compared to normoactive children. The results of the present study would predict similar autonomic and cortical changes suggestive of underarousal in the biological family members of hyperactive children.

Similarly, the present findings can be extended by further investigating the presence of possible secondary behavioural characteristics (as opposed for example to primary attentional deficits) such as academic difficulties, impulsivity, and anxiety. Although the Porteus Maze task has proven reasonably successful in detecting impulse control problems in hyperactive as compared to normal control children, this task may not be sufficienty sensitive to detect true differences in adult populations. In view of the directional trend in favour of the research hypothesis, further investigation of other impulsivity measures is warranted. Assessment of such secondary problems as academic difficulties and delinquency may require the investigation of school and legal records, with the family members' consent.

While it is concluded that the attentional data obtained in the present study provide strong support for a genetic theory, the present findings with respect to the personality data are far from clear-cut. Cantwell's and Morrison and Stewart's hypothesis concerning alcoholism in the biological parents of hyperactives received partial confirmation, despite concerns about the specificity of this association between HA and parental alcoholism risk. However, the genetic hypothesis with respect to hysteria and sociopathy was not supported. Indeed, it was demonstrated that environmental factors of raising a

a chronically problematic hyperactive child are associated with maternal hysteria and sociopathy.

More work is required to investigate socio-familial factors in the families of hyperactive children. In particular, studies investigating directional and bi-directional environmental hypothesis and involving personality measures to delineate behavioural and interactional patterns would extend the present findings.

REFERENCES

Abe, K. Parent-child transmission of some childhood behaviour characteristics. Acta Paedopsychiatrica (Basel), 1978, 44, 9-16.

Ackerman, P. T., Dykman, R. A., & Peters, J. E. Teenage status of hyperactive and nonhyperactive learning disabled boys. American Journal of Orthopsychiatry, 1977, 47, 577-596.

Anderson, L. M. Personality characteristics of parents of neurotic, aggressive, and normal preadolescent boys. Journal of Consulting and Clinical Psychology, 1969, 33, 575-581.

Anderson, R. P., Halcomb, C. G., & Doyle, R. B. The measurement of attentional deficits. Journal of Learning Disabilities, 1973, 6, 534-539.

Arkonac, O., & Guze, S. B. A family study of hysteria. New England Journal of Medicine, 1963, 268, 239-242.

Asarnow, R. F., & MacCrimmon, D. J. Residual performance deficit in clinically remitted schizophrenics: A marker of schizophrenia? Journal of Abnormal Psychology, 1978, 87, 597-608.

Asarnow, R. F., & MacCrimmon, D. J. Span of apprehension deficits during the postpsychotic stages of schizophrenia: A replication and extension. Archives of General Psychiatry, 1981, 38, 1006-1011.

Asarnow, R. F., Steffy, R., & Cleghorn, J. M. The McMaster-Waterloo studies of children at risk for severe psychopathology. In J. Shamsie (Ed.), New Directions in Children's Mental Health. New York: Spectrum Publications, 1978, 77-104.

Ault, R. L., Mitchell, C., & Hartmann, D. P. Some methodological problems in reflectivity-impulsivity research. Child Development, 1976, 47, 227-231.

Backman, J., & Firestone, P. A review of psychopharmacological and behavioural approaches to the treatment of hyperactive children. American Journal of Orthopsychiatry, 1979, 49, 500-504.

Barcai, A., & Rabkin, L.Y. A precursor of delinquency: The hyperkinetic disorder of childhood. Psychiatric Quarterly, 1974, 48, 387-399.

Barkley, R. A. The effects of methylphenidate on various types of activity level and attention in hyperkinetic children. Journal of Abnormal Child Psychology, 1977 5, 351-362.

Barkley, R. A. Hyperactive Children: A Handbook for Diagnosis and Treatment. New York: Guilford Press, 1981.

Barkley, R. A. Recent developments in research on hyperactive children. Journal of Pediatric Psychology, 1978, 3, 158-163.

Barkley, R. A., & Ullman, D. G. A comparison of objective measures of activity and distractibility in hyperactive and nonhyperactive children. Journal of Abnormal Child Psychology, 1975, 3 231-243.

Barron, F. An ego-strength scale which predicts response to psychotherapy. Journal of Consulting Psychology, 1953, 17, 327-333.

Battle, E. S., & Lacey, B. A context for hyperactivity in children over time. Child Development, 1972, 43, 757-773.

Beck, M. A. A follow-up study of adults who were clinically diagnosed as hyperkinetic in childhood. Unpublished doctoral dissertation, Wayne State University, 1977.

Bentzen, F. Sex ratios in learning and behavior disorders. American Journal of Orthopsychiatry, 1963, 33, 92-98.

Berlyne, D. E. Attention as a problem in behavior theory. In D. I. Mostofsky (Ed.), Attention: Contemporary Theory and Analysis. New York: Appleton-Century-Crofts, 1970, 25-49.

Block, G. H. Hyperactivity: A cultural perspective. Journal of Learning Disabilities, 1977, 10, 236-240.

Block, J. Parents of schizophrenic, neurotic, asthmatic, and congenitally ill children. Archives of General Psychiatry, 1969, 20, 659-674.

Blouin, A. G., Bornstein, R., & Trites, R. Teenage alcohol use among hyperactive children: A five year follow-up study. Journal of Pediatric Psychology, 1978, 3, 188-194.

Borland, B., & Heckman, H. Hyperactive boys and their brothers. Archives of General Psychiatry, 1976, 33, 669-675.

Bowdan, N. Hyperactivity or affective illness (letter to ed.). American Journal of Psychiatry, 1977, 134, 329.

Bower, K. B., & Mercer, C. D. Hyperactivity: Etiology and intervention techniques. Journal of School Health, 1975, 45, 195-202.

Bradley, P. E., Wakefield, J. A., Yom, B. L., Doughtie, E. B., Cox, J. A., & Kraft, I. A. Parental MMPIs and certain pathological behaviors in children. Journal of Clinical Psychology, 1974, 30, 379-382.

Bremer, D. A., & Stern, J. A. Attention and distractibitily during reading in hyperactive boys. Journal of Abnormal Child Psychology, 1976, 4, 381-387.

Brown, F. W. Heredity in the psychoneuroses. Proceedings of the Royal Society of Medicine, 1942, 35, 785-790.

Brumback, R. A., & Weinberg, W. A. Relationship of hyperactivity and depression in children. Perceptual and Motor Skills, 1977, 45, 247-251.

Butcher, J. N., & Tellegen, A. Common methodological problems in MMPI research. Journal of Consulting and Clinical Psychology, 1978, 46, 620-628.

Byrne, D., Barry, J., & Nelson, D. Relation of the revised Repression-Sensitization Scale to measures of self-description. Psychological Report, 1963, 13, 323-334.

Cadoret, R. J., Cain, C. A., & Grove, W. M. Development of alcoholism in adoptees raised apart from alcoholic biologic parents. Archives of General Psychiatry, 1980, 37, 561-563.

Cadoret, R. J., & Gath, A. Inheritance of alcoholism in adoptees. British Journal of Psychiatry, 1978, 132, 252-258.

Cameron, J. R. Parental treatment, children's temperament, and the risk of childhood behavioral problems: 1. Relationships between parental characteristics and changes in children's temperament over time. American Journal of Orthopsychiatry, 1977, 47, 568-576.

Campbell, E. S., & Redfering, D. L. Relationship among environmental and demographic variables and teacher-rated hyperactivity. Journal of Abnormal Child Psychology, 1979, 7, 77-81.

Campbell, S. B. Cognitive styles and behavior problems in clinic boys. A comparison of epileptic, hyperactive, learning-disabled and normal groups. Journal of Abnormal Child Psychology, 1974, 2, 307-312.

Campbell, S. B. Cognitive styles in reflective, impulsive and hyperactive boys and their mothers. Perceptual and Motor Skills, 1973, 36, 747-752.

Campbell, S. B. Hyperactivity: Course and treatment. In A. Davids (Ed.), Child Personality and Psychopathology: Current Topics. New York: Wiley, 1976, 3, 201-236.

Campbell, S. B., Douglas, V. I., & Morgenstern, G. Cognitive styles in hyperactive children and the effect of methylphenidate. Journal of Child Psychology and Psychiatry, 1971, 12, 55-67.

Campbell, S. B., Endman, M. W., & Bernfeld, G. A three-year follow-up of hyperactive preschoolers into elementary school. Journal of Child Psychology and Psychiatry, 1977, 18, 239-249.

Cantwell, D. P. Genetic factors in the hyperkinetic syndrome. Journal of the American Academy of Child Psychiatry, 1976, 15, 214-223.

Cantwell, D. P. Genetic studies of hyperactive children: Psychiatric illness in biologic and adopting parents. In R. Fieve, D. Rosenthal, & H. Brill (Eds.), Genetic Research in Psychiatry. Baltimore: Johns Hopkins University Press, 1975, 273-280. (a).

Cantwell, D. P. Genetics of hyperactivity. Journal of Child Psychology and Psychiatry & Applied Disciplines, 1975, 16, 261-264. (b).

Cantwell, D. P. The Hyperactive Child: Diagnosis, Management, Current Research. New York: Spectrum, 1975. (c).

Cantwell, D. P. Hyperactivity and Antisocial Behavior. Journal of the American Academy of Child Psychiatry, 1978, 17, 252-262.

Cantwell, D. P. The hyperkinetic syndrome. In M. Rutter, and L. Hershov (Eds.), Child Psychiatry: Modern Approaches. Oxford: Blackwell Scientific Publications, 1977, 524-555.

Cantwell, D. P. Parents of hyperactive children: Cause or effect. Medical Insight, 1973, 5, 24-28.

Cantwell, D. P. Psychiatric illness in the families of hyperactive children. Archives of General Psychiatry, 1972, 27, 414-417.

Carlson, R. W. Dimensionality of the Repression-Sensitization Scale. Journal of Clinical Psychology, 1979, 35, 78-84.

Caster, D. U., & Parsons, O. A. Relationship of depression, sociopathy, and locus of control to treatment outcome in alcoholics. Journal of Consulting and Clinical Psychology, 1977, 45, 751-756.

Chess, S. Childhood psychopathologies: The search for differentiation. Journal of Autism and Childhood Schizophrenia, 1972, 2, 111-113.

Chess, S., Thomas, A., Rutter, M., & Birch, H. Interaction of temperament and environment in the production of behavioral disturbances in children. American Journal of Psychiatry, 1963, 120, 142-148.

Clarkson, F. E., & Hayden, B. S. The relationship of hyperactivity in the normal class setting with family background factors and neurological status. In American Psychological Association, 80th, Hololulu, Proceedings, 1972, 7, 559-560.

Clements, S. D. Minimal brain dysfunction in children. NINDB Monograph, No. 3, Washington, D.C.: U.S. Department of Health, Education and Welfare, 1966.

Clements, S. D., & Peters, J. E. Minimal brain dysfunction in the schoool age child. Archives of General Psychiatry, 1962, 6, 185-197.

Clopton, J. R. Alcoholism and the MMPI: A review. Journal of Studies on Alcohol, 1978, 39, 1540-1558.

Cohen, N. J., & Douglas, V. I. Characteristics of the orienting response in hyperactive and normal control children. Psychophysiology, 1972, 9, 238-245.

Cohen, N. J., Douglas, V. I., & Morgenstern, G. The effect of methylphenidate on attentive behavior and autonomic activity in hyperactive children. Psychopharmacologia, 1971, 22, 282-294.

Cohen, N. J., Weiss, G., & Minde, K. Cognitive styles in adolescents previously diagnosed as hyperactive. Journal of Child Psychology and Child Psychiatry, 1972, 13, 203-209.

Cohler, B. J., Grunebaum, H. U., Weiss, J. L., Gamer, E., & Gallant, D. H. Disturbance of attention among schizophrenic, depressed and well mothers and their young children. Journal of Child Psychology and Psychiatry, 1977, 18, 115-135.

Conners, C. K. Rating scales for use in drug studies with children. Psychopharmacology Bulletin (Special Issue, Pharmacotherapy of Children, 1973, 24-29.

Conners, C. K. Symptom patterns in hyperkinetic, neurotic and normal children. Child Development, 1970, 41, 667-682.

Conners, C. K. A teacher rating scale for use in drug studies with children. American Journal of Psychiatry, 1969, 126, 884-888.

Crowe, R. R. An adoptive study of psychopathology. In R. Fieve, D. Rosenthal, & H. Brill (Eds.), Genetic Research in Psychiatry. Baltimore: John Hopkins University Press, 1975, 326-341.

Cruickshank, W. M. (Ed.) Psychology of Exceptional Children and Youth. Englewood Cliffs: Prentice-Hall, 1971.

Dahlstrom, W. G. Whither the MMPI? In J. N. Butcher (Ed.), Objective Personality Tests: Current Developments. New York: Academic Press, Inc., 1972.

Dahlstrom, W. G., & Dahlstrom, L. Basic Readings on the MMPI: A New Selection on Personality Measurement. Minneapolis: University of Minnesota Press, 1980.

Dahlstrom, W. G., Welsh, G. S., & Dahlstrom, L. E. An MMPI Handbook, Vol. I, Clinical Interpretation. Minneapolis: University of Minneapolis Press, 1972.

Dahlstrom, W. G., Welsh, G. S., & Dahlstrom, L. E. An MMPI Handbook, Vol. II, Research Developments and Applications. Minneapolis: University of Minnesota Press, 1975.

Dee, C., & Dee, H. L. MMPIs of parents of emotionally disturbed, motor dysfunctional, and normal children. Journal of Consulting and Clinical Psychology, 1972, 38, 464.

DeFries, J. C., & Plomin, R. Behavioral genetics. Annual Review of Psychology, 1978, 29, 473-515.

Denton, C. L., & McIntyre, C. W. Span of Apprehension in hyperactive boys. Journal of Abnormal Child Psychology, 1978, 6, 19-24.

Douglas, V. I. Differences between normal and hyperkinetic children. Paper presented at the Symposium on the Clinical Use of Stimulant Drugs in Children, Key Biscayne, Florida, 1972. (a).

Douglas, V. I. Perceptual and cognitive factors as determinants of learning disabilities: A review chapter with special emphasis on attentional factors. In R. M. Knights, & D. J. Becker (Eds.), The Neuropsychology of Learning Disorders: Theoretical Approaches. Baltimore: University Park Press, 1976, 413-421.

Douglas, V. I. Stop, look and listen: The problem of sustained attention and impulse control in hyperactive and normal control children. Canadian Journal of Behavioural Science, 1972, 4, 259-282. (b).

Douglas, V. I. Sustained attention and impulse control: Implication for the handicapped child. In J. A. Swets, & L. L. Eliot (Eds.), Psychology and the Handicapped Child. Washington, D.C.: U.S. Department of Education, 1974.

Douglas, V. I., Parry, P., Marton, P., & Garson, C. Assessment of a cognitive training program for hyperactive children. Journal of Abnormal Child Psychology, 1976, 4, 389-410.

Douglas, V. I., & Peters, K. G. Toward a clearer definition of the attentional deficit in hyperactive children. In G. A. Hale, & M. Lewis (Eds.), Attention and Cognitive Development. New York: Plenum Publishing, 1979, 173-247.

Douglas, V. I., Werry, J. S., & Weiss, G. Hyperactive behavior in children: Some findings regarding aetiology and treatment. Canadian Psychologist, 1965, 6, 219.

Doyle, R. B., Anderson, R. P., & Halcomb. C. G. Attention deficits and the effects of visual distraction. Journal of Learning Disabilities, 1976, 19, 48-54.

Dubey, D. R. Organic factors in hyperkinesis: A critical review. American Journal of Orthopsychiatry, 1976, 46, 353-366.

Duckworth, J. C. MMPI Interpretation Manual for Counselors and Clinicians. Muncie, Ind.: Accelerated Development Inc., 1979.

Dunn, L. M. Expanded Manual for the Peabody Picture Vocabulary Test. Circle Pines, Minn.: American Guidance Service, Inc., 1965.

Dworkin, R. H., Burke, B. W., Maher, B. A., & Gottesman, I. I. Genetic influences on the organization and development of personality. Developmental Psychology, 1977, 13, 164-165.

Dykman, R. A., Ackerman, P. T., Clements, S., & Peters, J. E. Specific learning disabilities: An attentional deficit disorder. In H. R. Myklebust (Ed.), Progress in Learning Disabilities, 2. New York: Grune & Stratton, 1971.

Dykman, R. A., Ackerman, P. T., & Oglesby, D. M. Selective and sustained attention in hyperactive, learning-disabled, and normal boys. Journal of Nervous & Mental Disease, 1979, 167, 288-297.

El-Guebaly, N., & Offord, D. R. The offspring of alcoholics: A critical review. American Journal of Psychiatry, 1977, 134, 357-365.

El-Guebaly, N., Offord, D. R., Sullivan, K. T., & Lynch, G. W. Psychosocial adjustment of the offspring of psychiatric inpatients. The effect of alcoholic, depressive and schizophrenic parentage. Canadian Psychiatric Association Journal, 1978, 23, 281-289.

Erickson, R. V., & Roberts, A. H. Some ego functions associated with delay of gratification in male delinquents. Journal of Consulting Psychology, 1971, 36, 378-382.

Estes, W. K. A technique for assessing variability of perceptual span. Proceedings of the National Academy of Sciences, 1965, 54, 403-407.

Eyre, S. L., Rounsaville, B. J., & Kleber, H. D. History of childhood hyperactivity in a clinic population of opiate addicts. Journal of Nervous and Mental Disease, 1982, 170, 522-529.

Eysenck, H. J., & Prell, D. B. The inheritance of neurotocism: An experimental study. Journal of Mental Science, 1951, 97, 441-465.

Firestone, P., Davey, J., Goodman, J. T., & Peters, S. The effects of caffeine and methylphenidate on hyperactive children. Journal of American Academy of Child Psychiatry, 1978, 7, 445-456.

Firestone, P., & Douglas, V. I. The effects of reward and punishment on reaction times and autonomic activity in hyperactive and normal children. Journal of Abnormal Child Psychology, 1975, 3, 201-215.

Firestone, P., & Martin, J. E. An analysis of the hyperactive syndrome: A comparison of hyperactive, behavior problem, asthmatic, and normal children. Journal of Abnormal Child Psychology, 1979, 7, 261-273.

Firestone, P., & Witt, J. Characteristics of families completing and prematurely discontinuing a behavioural parent training program. Journal of Pediatric Psychology, 1982, 7, 209-221.

Fisher, M. A. Dimensional interaction in hyperactive children: Classification of computer-displayed stimuli. Bulletin of the Psychonomic Society, 1977, 10, 443-446.

Fleischmann, David J. Etiological subgrouping of hyperkinetic boys. Unpublished doctoral dissertation, University of Iowa, 1978.

Franks, D. D., & Thacker, M. A. Assessing familial factors in alcoholism from MMPI profiles. American Journal of Psychiatry, 1979, 136, 1084-1085.

Friebergs, V., & Douglas, V. I. Concept learning in hyperactive and normal children. Journal of Abnormal Psychology, 1969, 74, 388-395.

Fries, M. E., & Wolff, P. J. Some hypotheses on the role of the congenital activity type in personality development. Psychoanalytic Study of Childhood, 1953, 8, 48-62.

Garfield, S., & Fey, W. A comparison of the Wechsler-Bellevue and Shipley-Hartford Scales as measures of mental impairment. Journal of Psychology, 1948, 12, 259-264.

Gomez, R. L., Janowsky, D., Zetin, M., Huey, L., & Clopton, P. L. Adult psychiatric diagnosis and symptoms compatible with the hyperactive child syndrome: A retrospective study. Journal of Clinical Psychiatry, 1981, 42, 389-394.

Goodman, J. T., Streiner, D. L., Bartolucci, G., & Woodward, C. A. The effects of psychiatric hospitalization. Paper presented at the Canadian Psychiatric Association Meeting, Halifax, Nova Scotia, 1978.

Goodwin, D. W. Alcoholism and heredity: A review and hypothesis. Archives of General Psychiatry, 1979, 36, 57-61.

Goodwin, D. W., Schulsinger, F., Hermansen, L., Guze, S. B., & Winokur, G. Alcohol problems in adoptees raised apart from alcoholic biological parents. Archives of General Psychiatry, 1973, 28, 238-243.

Goodwin, D. W., Schulsinger, F., Hermansen, L., Guze, S. B., & Winokur, G. Alcoholism and the hyperactive child syndrome. Journal of Nervous and Mental Disease, 1975, 160, 349-353.

Goodwin, D. W., Schulsinger, F., Knop, J., Mednick, S. & Guze, S. B. Psychopathology in adopted and nonadopted daughters of alcoholics. Archives of General Psychiatry, 1977, 34, 1005-1009.

Goodwin, D. W., Schulsinger, F., & Moller, N. Drinking problems in adopted and nonadopted sons of alcoholics. Archives of General Psychiatry, 1974, 31, 164-169.

Gordon, M. The assessment of impulsivity and mediating behaviors in hyperactive and nonhyperactive boys. Journal of Abnormal Child Psychology, 1979, 7, 317-326.

Gottesman, I. I. Heritability of personality: A demonstration. Psychology Monogram, 1963, 77 (9, Whole No. 572), 1-21.

Goyette, C. H., Conners, C. K., & Ulrich, R. F. Normative data on revised Conners parents and teachers rating scales. Journal of Abnormal Child Psychology, 1978, 6, 221-236.

Graham, J. R. The MMPI: A Practical Guide. New York: Oxford University Press, 1982.

Greene, R. L. The MMPI: An Interpretive Manual. New York: Grune & Stratton, 1980.

Guze, S. B. The diagnosis of hysteria: What are we trying to do? American Journal of Psychiatry, 1967, 124, 491-498.

Guze, S. B., Wolfgram, E. D., McKinney, J. K., & Cantwell, D. P. Psychiatric illness in the families of convicted criminals: A study of 519 first-degree relatives. Diseases of the Nervous System, 1967, 28, 651-659.

Guze, S. B., Woodruff, R. W., & Clayton, P. Hysteria and antisocial behavior: Further evidence of an association. American Journal of Psychiatry, 1971, 127, 957-960.

Gynther, M. D., Altman, H., & Sletten, I. Replicated correlates of MMPI two-point code types: The Missouri actuarial system. Journal of Clinical Psychology, 1973, 29, 263-289.

Hafner, A., Butcher, J., Nall, M. & Wentworth, O. Parent personality and childhood disorders: A review of MMPI findings. In J. N. Butcher (Ed.), MMPI: Research Developments and Clinical Applications. New York: McGraw- Hill, 1969.

Haier, R. J., Rieder, R. D., Khouri, P. J., & Buchsbaum, M. S. Extreme MMPI scores and the research diagnostic criteria. Archives of General Psychiatry, 1979, 36, 528-534.

Hanvik, L. J., & Byrum, M. MMPI profiles of parents of child psychiatric patients. Journal of Clinical Psychology, 1959, 15, 427-431.

Harris, R. J. A Primer of Multivariate Statistics. New York: Academic Press, 1975.

Hartocollis, P. The syndrome of minimal brain dysfunction in young adult patients, Bulletin of the Menninger Clinic, 1968, 32, 102-114.

Hastings, J. E., & Barkley, R. A. A review of psychophysiological research with hyperactive children. Journal of Abnormal Child Psychology, 1978, 6, 311-324.

Hechtman, L., Weiss, G., Finklestein, J. Werner, A., & Benn, R. Hyperactives as young adults: Preliminary report. Canadian Medical Association Journal, 1976, 115, 625-630.

Hechtman, L., Weiss, G., Perlman, T., Hopkins, J., & Wener, A. Hyperactive children in young adulthood: A controlled, prospective ten-year follow-up. International Journal of Mental Health, 1979, 8, 52-66.

Hodo, G. L., & Fowler, R. D. Frequency of MMPI two-point codes in a large alcoholic sample. Journal of Clinical Psychology, 1976, 32, 487-489.

Homatidis, S., & Konstantareas, M. M. Assessment of hyperactivity: Isolating measures of high discriminant ability. Journal of Consulting and Clinical Psychology, 1981, 49, 533-541.

Hopkins, J., Perlman, T., Hechtman, L., & Weiss, G. Cognitive style in adults originally diagnosed as hyperactives. Journal of Child Psychology and Psychiatry, 1979, 20, 209-215.

Horvath, T., Friedman, J., & Meares, R. Attention in hysteria: A study of Janet's hypothesis by means of habituation and arousal measures. American Journal of Psychiatry, 1980, 137, 217-220.

Hoy, E., Weiss, G., Minde, K., & Cohen, N. The hyperactive child at adolescence: Cognitive, emotional, and social functioning. Journal of Abnormal Child Psychology, 1978, 6, 311-324.

Huessy, H. R., Metoyer, M., & Townsend, M. Eight- to ten-year follow-up of 84 children treated for behavioral disturbance in rural Vermont. Acta Paedopsychiatra, 1974, 40, 230-235.

Hutchings, B., & Mednick, S. A. Criminality in adoptees and their adoptive and biological parents. In S. A. Mednick & K. O. Christiansen (Eds.), Biosocial Bases of Criminal Behavior. New York: Gardner Press, 1977.

Johnson, S. M., & Lobitz, G. K. Ther personal and marital adjustment of parents as related to observed child deviance and parenting behaviors. Journal of Abnormal Child Psychology, 1974, 2, 193-207.

Johnsson, E., & Nillson, T. Alkohol konsumption Ho s monozygota och dizuygota tvillingar. Nord Hgg Tidskr, 1968, 49, 21.

Kahneman, D. Attention and Effort. Englewood Cliffs, New Jersey: Prentice-Hall, 1973.

Kaij, L. Studies on the etiology and sequels of abuse of alcohol. Unpublished thesis, Sweden: University of Lund, 1960.

Kenny, T., Balwin, R., & Mackie, J. B. Incidence of minimal brain injury in adopted children. Child Welfare, 1967, 1, 24-29.

Kenny, T. J., Clemmens, R. L., Hudson, B. W., Lentz, G. A., Cicci, R., & Nair, P. Characteristics of children referred because of hyperactivity. Journal of Pediatrics, 1971, 79, 618-622.

Keogh, B. K., & Margolis, J. S. Learn to labor and wait: Attentional problems of children with learning disorders. Journal of Learning Disabilities, 1976, 9, 276-286.

Keppel, G. Design and Analysis: A Researcher's Handbook. Englewood Cliffs, New Jersey: Prentice-Hall, Inc., 1973.

Kidd, K. K., & Matthysee, S. Research designs for the study of gene-environment interactions in psychiatric disorders. Archives of General Psychiatry, 1978, 35, 925-232.

Kirk, R. E. Experimental Design: Procedures for the Behavioral Sciences. Belmont, California: Brooks-Cole, 1968.

Klein, D. F., & Gittelman-Klein, R. Problems in the diagnosis of minimal brain dysfunction and the hyperkinetic syndrome. International Journal of Mental Health, 1975, 4, 45-60.

Knights, R. M. Psychometric assessment of stimulant-induced behavior change. In C. K. Conners (Ed.), Clinical Use of Stimulant Drugs in Children. New York: American Elsevier, 1974, 221-231.

Knights, R., & Hinton, G. The effects of methylphenidate on the motor skills and behavior of children with learning problems. Journal of Nervous and Mental Disease, 1969, 148, 643-653.

Kupietz, S., Bialer, I., & Winsberg, B. A behavior rating scale for assessing improvement in behaviorally deviant children: A preliminary investigation. American Journal of Psychiatry, 1972, 128, 1432-1436.

Lachar, D., & Sharp, J. R. Use of Parents' MMPIs in the research and evaluation of children: A review of the literature and some new data. In James N. Butcher (Ed.), New Developments in the Use of the MMPI, Minneapolis: University of Minnesota Press, 1979.

Lambert, N. M., & Windmiller, M. An exploratory study of temperament traits in a population of children at risk. Journal of Special Education, 1977, 11, 37-47.

Langhorne, J. E., Jr., & Loney, J. A four-fold model for subgrouping the hyperkinetic/MBD syndrome. Child Psychiatry and Human Development, 1979, 9, 153-159.

Langhorne, J. E., Jr., Loney, J., Paternite, C. E., & Bechtoldt, H. P. Childhood hyperkinesis: A return to the source. Journal of Abnormal Psychiatry, 1976, 85, 201-209.

Laufer, M. W. Long-term management and some follow-up findings on the use of drugs with minimal cerebral syndromes. Journal of Learning Disabilities, 1971, 4, 55-58.

Laufer, M. W., Denhoff, E., & Soloman, G. Hyperkinetic impulse disorder in children's behavior problems. Psychosomatic Medicine, 1957, 19, 38-49.

Lauterbach, C., London, P., & Bryan, J. MMPI's of parents of child guidance clinics. Journal of Clinical Psychology, 1961, 17, 151-154.

Lesser, L. L. Hyperkinesis in children. Clinical Pediatrics, 1970, 9, 548-554.

Lewandowski, D., & Graham, J. R. Empirical correlates of frequently occurring two-point MMPI code-types: A replicated study. Journal of Consulting and Clinical Psychology, 1972, 39, 467-472.

Liverant, S. MMPI differences between parents of disturbed and nondisturbed children. Journal of Consulting Psychology, 1959, 23, 256-260.

Loehlin, J. C., & Nichols, R. C. Heredity, Environment, and Personality: A Study of 850 Sets of Twins. Austin: University of Texas Press, 1976.

Loney, J. Childhood hyperactivity. In R. Woddy (Ed.), Encyclopedia of Clinical Assessment, San Francisco: Josey-Bass, 1978.

Loney, J. Hyperkinesis comes of age: What do we know and where should we go? Paper presented at the Annual Meeting of the American Orthopsychiatric Association, Washington, D.C., April, 1979.

Loney, J. The Iowa theory of substance abuse among hyperactive adolescents. In D. J. Lettieri, M. Sayers, & H. W. Pearson (Eds.), In Theories on Drug Abuse. National Institute of Drug Abuse Research Monograph 30. Washington, D.C.: U.S. Department of Health and Human Services, 1980, 132-136.

Loney, J., Kramer, J. & Milich, L. The hyperkinetic child grows up: Predictors of symptoms, delinquency, and achievement at follow-up. Symposium presentation, American Association for the Advancement of Science, annual meeting, 1979, Houston.

Loney, J., Langhorne, J. E., & Paternite. An empirical basis for subgrouping the hyperkinetic/minimal brain dysfunction syndrome. Journal of American Psychology, 1978, 87, 431-441.

Lopez, R. E. Hyperactivity in twins. Canadian Psychiatric Association Journal, 1965, 10, 421-426.

Mann, H. B., & Greenspan, S. I. The identification and treatment of adult brain dysfunction. American Journal of Psychiatry, 1976, 133, 1013-1017.

Marks, P. A., & Seeman, W. The Actuarial Description of Abnormal Personality: An Atlas for Use with the MMPI. Baltimore: Williams & Wilkins, 1963.

Marks, P. A., Seeman, W., & Haller, D. L. The Actuarial Use of the MMPI with Adolescents and Adults. Baltimore: The Williams & Wilkins Co., 1974.

Marwit, S J., & Stenner, A. J. Hyperkinesis: Delineation of two patterns. Exceptional Children, 1972, 38, 401-406.

McAdoo, W., & Connally, F. MMPIs of parents of dysfunctional families. Journal of Consulting and Clinical Psychology, 1975, 43, 270.

McGlannan, F. Abstracts: Childhood hyperactivity tied to risk of later alcoholism; arousal, activation, and effort in the control of attention - Part II: Activation. Journal of Learning Disabilities, 1977, 10, 560-563.

McIntyre, C. W., Blackwell, S. L., & Denton, C. L. Effect of noise distractibility on the spans of apprehension of hyperactive boys. Journal of Abnormal Child Psychology, 1978, 6, 483-492.

McKinley, J. C., & Hathaway, S. R. The identification and measurement of the psychoneuroses in medical practice: The MMPI. Journal of the American Medical Association, 1943, 122, 161-167.

McMahon, R. C. Genetic etiology in the hyperactive child syndrome: A critical review. American Journal of Orthopsychiatry, 1980, 50, 145-150.

Mednick, S. A., & Hutchings, B. Genetic and Psychophysiological factors in a social behavior. Journal of the American Academy of Child Psychiatry, 1978, 48, 209-223.

Mednick, S. A., & Schulsinger, F. Some premorbid characteristics related to breakdown in children with schizophrenic mothers. In D. Rosenthal, & S. S. Kety (Eds.), The Transmission of Schizophrenia. London: Pergamon Press, 1968, 267-291.

Meehl, P. E. Schizotaxia, schizotypy, and schizophrenia. American Psychologist, 1962, 17, 827-828.

Meichenbaum, D. Cognitive Behavior Modification. New York: Plenum Press, 1977.

Mellsop, G. W. Psychiatric patients seen as children and adults: Childhood predictors of adult illness. Journal of Child Psychology and Psychiatry, 1972, 13, 91-101.

Mendleson, W., Johnson, J., & Stewart, M. A. Hyperactive children as teenagers: A follow-up study. Journal of Nervous and Mental Disease, 1971, 153, 273-279.

Menkes, M., Rowe, J., & Menkes, J. A twenty-five year follow-up study on the hyperkinetic child with minimal brain dysfunction. Pediatrics, 1967, 39, 392-399.

Merikangas, K. R. Assortative mating for psychiatric disorders and psychological traits. Archives of General Psychiatry, 1982, 39, 1173-1180.

Messer, S. B. Reflection-Impulsivity: A review. Psychological Bulletin, 1976, 83, 1026-1052.

Milich, R., & Loney, J. The role of hyperactive and aggressive symptomatology in predicting adolescent outcome among hyperactive children. Journal of Pediatric Psychology, 1979, 4, 93-112.

Miller, L. G. Genetic disease and social pathology. Ethics in Science and Medecine, 1977, 4, 29-50.

Miller, J. S. Hyperactive children: A ten-year study. Pediatrics, 1978, 61, 217-223.

Miller, W. H., & Keirn, W. C. Personality measurement in parents of retarded and emotionally disturbed children: A replication. Journal of Clinical Psychology, 1978, 34, 686-690.

Milman, D. Minimal brain dysfunction in childhood: Outcome in late adolescence and early adult years. Journal of Clinical Psychiatry, 1979, 40, 371-380.

Minde, K., Lewin, D., Weiss, G., Lavigueur, H., Douglas, V., & Sykes, E. The hyperactive child in elementary school: A five-year controlled follow-up. Exceptional Children, 1971, 38, 215-221.

Minde, K., Weiss, G., & Mendelson, N. A five-year follow-up study of 91 hyperactive school children. Journal of the American Academy of Child Psychiatry, 1972, 11, 595-610.

Morrison, J. R. Adult psychiatric disorders in parents of hyperactive children. American Journal of Psychiatry, 1980, 137, 825-827.

Morrison, J. R. Diagnosis of adult psychiatric patients with childhood hyperactivity. American Journal of Psychiatry, 1979, 136, 955-958.

Morrison, J. R. The family histories of manic depressive patients with and without alcoholism. Journal of Nervous and Mental Disease, 1975, 160, 227-229.

Morrison, J. R. Letter: Hereditary factors in hyperkinesis. American Journal of Psychiatry, 1974, 131, 472.

Morrison, J. R., & Minkoff, K. Explosive personality as a sequel to the hyperactive-child syndrome. Comprehensive Psychiatry, 1975, 16, 343-348.

Morrison, J. R., & Stewart, M. A. Bilateral inheritance as evidence for polygenicity in the hyperactive child syndrome. Journal of Nervous and Mental Disease, 1974, 158, 226-228.

Morrison, J. R., & Stewart, M. A. Evidence for polygenetic inheritance in the hyperactive child syndrome. American Journal of Psychiatry, 1973, 132, 791-792. (a).

Morrison, J. R., & Stewart, M. A. A family study of the hyperactive child syndrome. Biological Psychiatry, 1971, 3, 189-195.

Morrison, J. R., & Stewart, M. A. The psychiatric status of the legal families of adopted hyperactive children. Archives of General Psychiatry, 1973, 28, 888-891. (b).

Murley, H. D., Milam, D. R., & Gorman, W. F. The hyperactive child grows up. Arizona Medicine, 1977, 34, 767-768.

Nahon, D. Coping styles of parents of hyperactive children. Unpublished masters thesis, University of Ottawa, 1983.

Neale, J. M., McIntyre, C. W., Fox, R., & Cromwell, R. L. Span of apprehension in acute schizophrenics. Journal of Abnormal Psychology, 1969, 74, 593-596.

Ney, P. Four types of hyperkinesis. Canadian Psychiatric Association Journal, 1974, 19, 543-550.

Nichols, P. L. Minimal brain dysfunction: Genetic studies. Behavior Genetics, 1977, 7, 80-81.

Nichols, P. L. Minimal brain dysfunction: Family studies. Behavior Genetics, 1979, 9, 473-474.

Nylander, I. Children of alcoholic fathers. Acta Paediatrial Scandinavia, 1960, 49, 1-134.

Offord, D. R. The natural histories of schizophrenia, depression, disorder and psychopathy: Current status. Psychosomatics, 1971, 12, 179-185.

O'Keefe, E. J. Porteus Maze Q Score as a measure of impulsivity. Perceptual and Motor Skills, 1975, 41, 675-678.

Oki, T. The psychological study of early childhood neurosis. In H. Mitsuda (Ed.), Clinical Genetics in Psychiatry. Tokyo: Igaku Shoin, 1967.

Omenn, G. S. Genetic approaches to the syndrome of minimal brain dysfunction. Annals of the New York Academy of Science, 1973, 205, 212-222.

Orvaschel, H., Mednick, S., Schulsinger, F., & Rock, D. The children of psychiatrically disturbed parents. Archives of General Psychiatry, 1979, 36, 691-695.

Owen, F. W., Adams, P. A., Forrest, T. 'Learning disabilities in children: Sibling studies. Bulletin of the Orton Society, 1968, 18, 33-62.

Palkes, H., Stewart, W., & Kahana, B. Porteus maze performance of hyperactive boys after training in self-directed verbal commands. Child Development, 1968, 39, 817-826.

Parry, P. The effect of reward on the performance of hyperactive children. Unpublished doctoral dissertation, McGill University, 1973.

Partanen, J., Bruun, K., & Markkanen, T. Inheritance of drinking behaviour: A study on intelligence, personality, and use of alcohol of adult twins. Helsinki: Finnish Foundation for Alcohol Studies, 1966.

Paternite, C. E. Childhood hyperkinesis: A cross-sectional and longitudinal examination of the relationships between symptomatology and the home or family subenvironment. Unpublished doctoral dissertation, University of Iowa, 1978.

Paternite, C., Loney, J., & Langhorne, J. Relationship between symptomatology and SES-related factors in hyperkinetic/MBD boys. American Journal of Orthopsychiatry, 1976, 46, 291-301.

Paulson, M. J., Afifi, A. A., Thomason, M. C., & Chaleff, A. The MMPI: A descriptive measure of psychopathology in abusive parents. Journal of Clinical Psychology, 1974, 30, 387-390.

Paulson, M. J., & Lin, T. Predicting WAIS IQ from Ship-Hartford scores. Journal of Clinical Psychology, 1970, 26, 453-461.

Porges, S. W., Walter, G. F., Korb, R. J., & Sprague, R. I. The influence of methylphenidate on heart rate and behavioral measures of attention in hyperactive children. Child Development, 1975, 46, 727-733.

Porteus, S. D. New applications of the Porteus Maze test. Perceptual and Motor Skills, 1968, 26, 787-798.

Porteus, S. D. The Maze Test and Mental Differences. Vineland, New Jersey: Smith, 1933.

Porteus, S. D. The validity of the Porteus Maze test. Journal of Educational Psychology, 1939, 30, 172-178.

Prado, W., & Taub, D. Accurate prediction of individual intellectual functioning by the Shipley-Hartford. Journal of Clinical Psychology, 1966, 22, 294-296.

Pribram, K. H., & McGuiness, D. Arousal, activation, and effort in the control of attention. Psychological Review, 1975, 82, 116-149.

Quay, H. C. Psychopathic behavior: Reflections on its nature, origins, and treatment. In F. Weizman, & I. Uzgiris (Eds.), The Structuring of Experience. New York: Plenum Press, 1977, 371-383.

Quay, H. C., & Werry, J. S. Psychopathological Disorders of Childhood. New York: John Wiley & Sons, 1979.

Quitkin, F., & Klein, D. F. Two behavioral syndromes in young adults related to possible minimal brain dysfunction. Journal of Psychiatric Research, 1969, 7, 131-142.

Rapoport, J. L., & Ferguson, H. B. Biological validation of the hyperkinetic syndrome. Journal of Pediatric Psychology, 1982,

Riddle, M., & Roberts, A. H. Delinquency, delay of gratification, and the Porteus Maze tests. Psychological Bulletin, 1977, 84, 417-425.

Riddle, M., & Roberts, A. H. The Porteus Mazes: A critical evaluation. (Report No. PR-74-3). Minneapolis: University of Minnesota, Department of Psychiatry, Research Laboratories, 1974.

Robins, L. N. Deviant Children Grown Up. Baltimore: Williams & Wilkins, 1966.

Robins, L. N. Sturdy childhood predictors of adult antisocial behaviour: Replications from longitudinal studies. Psychological Medicine, 1978, 8, 611-622.

Roff, M. Childhood antecedents of adult neurosis, severe bad conduct, and psychological health, in D. F. Ricks, A. Thomas & M. Roff (Eds.), Life History Research in Psychopathology: III. Minneapolis: University of Minneapolis Press, 1974.

Rosenthal, D. Genetic Theory and Abnormal Behavior. New York: McGraw-Hill, Inc., 1970.

Rosenthal, D. Genetics of Psychopathology. New York: McGraw-Hill, Inc., 1971.

Rosenthal, J. H. Neurophysiology of minimal cerebral dysfunctions. Academic Therapy, 1973, 8, 291-294.

Ross, A. O. Psychological Disorders of Children. New York: McGraw-Hill, Inc., 1974.

Ross, D. M., & Ross, S. A. Hyperactivity: Research, Theory, and Action. New York: John Wiley & Sons, 1976.

Routh, D. K., & Roberts, R. Minimal brain dysfunction in children: Failure to find evidence for a behavioral syndrome. Psychological Reports, 1972, 31, 307-314.

Ruff, C. F., Ayers, J. L., & Templer, D. I. Hyperactivity of alcoholics and their children. Catalog of Selected Documents in Psychology, 1975, 5, 333.

Rutter, M. Other family influences. In M. Rutter, & L. Hershov (Eds), Child Psychiatry: Modern Approaches. Oxford: Blackwell Scientific Publications, 1977, 74-108.

Rutter, M. Relationship between the psychiatric disorders of childhood and adulthood. Acta. Psychiatrica Scandinavia, 1972, 48, 3-21.

Safer, D. J. A familial factor in minimal brain dysfunction. Behavior Genetics, 1973, 3, 175-186.

Safer, D. J., & Allen, R. P. Hyperactive Children: Diagnosis and Management. Baltimore: University Park Press, 1977.

Sargeant, J. A., Van Velthoven, R., & Virginia, A. Hyperactivity, impulsivity and reflectivity: An examination of their relationship and implications for clinical child psychology. Journal of Child Psychology and Psychiatry, 1979, 20, 47-60.

Satterfield, J. H., & Cantwell, D. P. Psychopharmacology in the prevention of antisocial and delinquent behavior. International Journal of Mental Health, 1975, 4, 227-237.

Satterfield, J. H., Cantwell, D. P., Lesser, L. I., & Posodin, R. L. Physiological studies of the hyperkinetic child: I. American Journal of Psychiatry, 1972, 128, 1418-1424.

Satterfield, J. H., & Dawson, M. E. Electrodermal correlates of hyperactivity in children. Psychophysiology, 1971, 8, 191-197.

Scarr, S. W. Genetic factors in activity motivation. Child Development, 1966, 37, 663-673.

Schuckit, M. A., Goodwin, D. W., & Winokur, G. A half-sibling study of alcoholism. American Journal of Psychiatry, 1972, 128, 1132-1136.

Schuckit, M. A., Petrich, J., & Chiles, J. Hyperactivity: Diagnostic confusion. Journal of Nervous and Mental Disease, 1978, 166, 79-87.

Schulsinger, F. Psychopathology. International Journal of Mental Health, 1972, 1, 190-206.

Sergeant, J. A., Van Velthoven, R., & Virginia, A. Hyperactivity, impulsivity and reflectivity: An examination of their relationship and implications for clinical child psychology. Journal of Child Psychology and Psychiatry, 1979, 20, 47-60.

Shaffer, D., & Greenhill, L. A critical note on the predictive validity of the hyperkinetic syndrome. Journal of Child Psychology and Psychiatry, 1979, 20, 61-72.

Shields, J. Polygenic influences. In M. Rutter, & L. Hershov (Eds.), Child Psychiatry: Modern Approaches. London: Blackwell Scientific Publications, 1977, 22-46.

Shields, J. Some recent developments in psychiatric genetics. Archiv. fur Psychiatrie und Nervenkrankheiten, 1975, 220, 347-360.

Shipley, W. C. A self-administering scale for measuring intellectual impairment and deterioration. Journal of Psychology, 1940, 9, 371-377. (a).

Shipley, W. C. Shipley Institute of Living Scale for Measuring Intellectual Impairment: Manual for Directions and Scoring Key. Hartford: Neuropsychiatric Institute for the Hartford Retreat, 1940. (b).

Siggers, D. C. Human behavioural genetics. Developmental Medicine and Child Neurology, 1977, 19, 818-820.

Silver, L. B. Familial patterns in children with neurologically-based learning disabilities. Journal of Learning Disabilities, 1971, 4, 349-358.

Silver, L. B. Frequency of adoption in children with the neurological learning disability syndrome. Journal of Learning Disabilities, 1970, 3, 306-310.

Sines, L. K., & Simmons, H. The Shipley-Hartford Scale and the Doppelt Short Form as estimators of WAIS IQ in a state hospital population. Journal of Clinical Psychology, 1959, 14, 452-453.

Slater, E. Expectation of abnormality on paternal and maternal sides: A computational model. Journal of Medical Genetics, 1966, 3, 159-161.

Sperling, G. The information available in brief visual presentations. Psychological Monographs, 1960, 74, (Whole No. 498).

Spezzano, C. J. The intellectual, neuropsychological, social, and emotional characteristics of children referred because of hyperactivity. Unpublished doctoral dissertion, City University of New York, 1976.

Sprague, R. L., Christensen, D. E., & Werry, J. S. Experimental psychology and stimulant drugs. In C. Conners (Ed.), Clinical Use of Stimulant Drugs in Children. New York: American Elsevier Publishing Co., Inc., 1974.

Sroufe, L. A. Drug treatment of children with behavior problems. In F. D. Horowitz (Ed.), Review of Child Development Research (Vol. 4). Chicago: University of Chicago Press, 1975.

Statistics Canada, Bulletin Number 13207, 1980.

Stewart, M. A. Hyperactive children. Scientific American, 1970, 222, 94-98.

Stewart, M. A. Is hyperactivity abnormal? And other unanswered questions. School Review, 1976, 85, 31-42.

Stewart, M. A., & Leone, L. A family study of unsocialized aggressive boys. Biological Psychiatry, 1978, 13, 107-117.

Stewart, M. A., Mendelson, W. B., & Johnson, N. E. Hyperactive children as adolescents: How they describe themselves. Child Psychiatry and Human Development, 1973, 4, 3-11. (a).

Stewart, M. A., & Morrison, J. R. Affective disorder among the relatives of hyperactive children. Journal of Child Psychology and Psychiatry, 1973, 14, 209-212. (b).

Stewart, M. A., & Olds, S. W. Raising a Hyperactive Child. New York: Harper & Row, 1973.

Stewart, M. A., Pitts, F. N., Craig, A. G., & Dieruf, A. The hyperactive child syndrome. American Journal of Orthopsychiatry, 1966, 36, 861-867.

Strauss, A., & Lehtinen, L. Psychopathology and Education in the Brain-injured Child. (Vol. 1). New York: Grune and Stratton, 1947.

Streiner, D. L., Goodman, J. T., & McLean, A. Correspondence between the MMPI and the Mini-Mult. Psychological Reports, 1977, 40, 551-554.

Streiner, D. L., & Miller, H. R. A table for prorating incomplete Form R MMPIs. Journal of Consulting and Clinical Psychology, 1979, 47, 474-477.

Streiner, D. L., Woodward, C. A., Goodman, J. T., & McLean, A. Comparisons of the MMPI and the Mini-Mult. Canadian Journal of Behavioral Science, 1973, 5, 76-82.

Sykes, D. H., Douglas, V. I., & Morgenstern, G. Sustained attention in hyperactive children. Journal of Child Psychology and Psychiatry, 1973, 14, 213-220.

Sykes, D. H., Douglas, V. I., Weiss, G., & Minde, K. K. Attention in hyperactive children and the effect of methylphenidate (Ritalin). Journal of Child Psychology and Psychiatry, 1971, 12, 129-139

Tarter, R. E., McBride, H., Buonpane, N., & Schneider, D. U. Differentiation of alcoholics: Childhood history of minimal brain dysfunction, family history, and drinking pattern. Archives of General Psychiatry, 1977, 34, 761-768.

Torgensen, A. M., & Kringlen, E. Genetic aspects of temperamental differences in infants. Journal of the American Academy of Child Psychiatry, 1978, 17, 433-444.

Tsuang, M. T. Genetic counselling for psychiatric patients and their families. American Journal of Psychiatry, 1978, 135, 1465-1475.

Ullman, D. G., Barkley, R. A., & Brown, J. W. Behavioral symptoms of hyperkinetic children who responded to methylphenidate treatment. American Journal of Orthopsychiatry, 1978, 48, 425-437.

Vandenberg, S. G. Assortative mating, or who marries whom? Behavioral Genetics, 1972, 2, 127-157.

Vandenberg, S. G. The hereditary abilities study: Hereditary components in a psychological test battery. American Journal of Human Genetics, 1962, 14, 220-237.

Vandenberg, S. G. Possible hereditary factors in minimal brain dysfunction. Annals of the New York Academy of Science, 1973, 205, 223-230.

Venables, P. H. Performance and level of activation in schizophrenics and normals. British Journal of Psychology, 1964, 55, 207-218.

Warner, B. J. Psychogenic hyperactivity: Situational and relationship factors in the hyperactive couple. Unpublished doctoral dissertation, California School of Professional Psychology, 1976.

Warren, R. J., Karduck, W. A., Bussaratid, S., Stewart, M., & Sly, W. The hyperactive child syndrome: Normal chromosome findings. Archives of General Psychiatry, 1971, 24, 161-162.

Waskow, I. E. Selection of a core battery. In I. E. Waskow, & M. B. Parloff (Eds.), Psychotherapy Change Measures. Report of the Clinical Resarch Brance Outcome Measures Project. Rockville, Mc.: National Institute of Mental Health, 1975.

Weiner, A. S., & Berzonsky, M. D. Development of selective attention in reflective and impulsive children. Child Development, 1975, 46, 545-549.

Weiss, G. The natural history of hyperactivity in childhood and treatment with stimulant medication at different ages: A summary of research findings. International Journal of Mental Health, 1975, 4, 213-226.

Weiss, G., Hecthman, L., & Perlman, T. Hyperactives as young adults: School, employees and self-rating scales obtained during ten year follow-up evaluation. American Journal of Orthopsychiatry, 1978, 48, 439-445.

Weiss, G., Hechtman, L., Perlman, T. Hopkins, J., & Wener, A. Hyperactives a young adults. Archives of General Psychiatry, 1979, 36, 675-681.

Weiss, G., Kruger, F., Danielson, U., & Elman, M. Effect of long-term treatment of hyperactive children with methylphenidate. Canadian Medical Association Journal, 1975, 112, 595-665.

Weiss, G., Minde, K. K., Werry, J. S., Douglas, V., & Nemeth, E. Studies on the hyperactive child: A five-year follow-up. Archives of General Psychiatry, 1971, 24, 409-414.

Weisskopf, B. Learning disorders in children: Behavioral and emotional aspects of child and family. Southern Medical Journal, 1969, 62, 811-815.

Welner, Z, Welner, A., Stewart, M., Palkes, H., & Wish, E: A controlled study of siblings of hyperactive children. Journal of Nervous and Mental Disease, 1977, 165, 110-117.

Wender, P. H. Minimal brain dysfunction: An overview. Paper presented at meeting of American College of Neuropharmacology, 1976.

Wender, P. H. The minimal brain dysfunction syndrome in children. Journal of Nervous and Mental Disease, 1972, 155, 55-71.

Wender, P. H. Minimal Brain Dysfunction in Children. New York: John Wiley, 1971.

Wender, P. H., Reimherr, F. W., & Wood, D. R. Attention deficit disorder (Minimal brain dysfunction) in adults. Archives of General Psychiatry, 1981, 38, 449-456.

Werry, J. S. Developmental hyperactivity. Pediatric Clinics of North America, 1968, 15, 581-599.

Werry, J. S., Sprague, R. L., & Cohen, M. N. Conner's Teacher Rating Scale for use in drug studies with children: An empirical study. Journal of Abnormal Child Psychology, 1975, 3, 217-229.

West, D. J., & Farrington, D. P. Who Becomes Delinquent? London: Heinemann, 1973.

Whalen, C. K., & Henker, B. Hyperactive Children: The Social Ecology of Identification and Treatment. New York:, Academic Press, 1980.

Willerman, L. Activity level and hyperactivity in twins. Child Development, 1973, 44, 288-293.

Winer, B. V. Statistical Principles in Experimental Design. New York: McGraw-Hill, 1971.

Woerner, P. I., & Guze, S. B. A family and marital study of Hysteria. British Journal of Psychiatry, 1968, 114, 161-168.

Wolking, W. D., Dunteman, G. H., & Bailey, J. P. Multivariate analyses of parents' MMPIs based on the psychiatric diagnoses of their children. Journal of Consulting Psychology, 1967, 31, 521-524.

Wolking, W. D., Quant, W., & Lawton, J. J. MMPI profiles of the parents of behaviorally disturbed children and parents from the general population. Journal of Clinical Psychology, 1966, 22, 39-48.

Wood, D. R., Reimherr, F. W., Wender, P. H., & Johnson, G. E. Diagnosis and treatment of minimal brain dysfunction in adults. Archives of General Psychiatry, 1976, 33, 1453-1460.

Wright, J. C., & Vlietstra, A. G. The development of selective attention: From perceptual exploration to logical search. In H. W. Reese (Ed.), Advances in child Development and Behavior (Vol 10). New York: Academic Press, 1975, pp. 196-235.

Wright, J. C., & Vlietstra, A. G. Reflection-impulsivity and information-processing from three to nine years of age. In M. J. Fine (Ed.), Principles and Techniques of Intervention with Hyperactive Children. Springfield, Illinois: Chas. C. Thomas, Publisher, 1977.

Zahn, T. P., Abate, F., Little, B. C., & Wender, P. H. Minimal brain dysfunction, stimulant drugs, and autonomic nervous system activity. Archives of General Psychiatry, 1975, 32, 381-387.

Zentall, S. Optimal stimulation as a theoretical basis of hyperactivity. American Journal of Orthopsychiatry, 1975, 45, 549-563.

Zentall, S., & Zentall, T. R. Activity and task performance of hyperactive children as a function of environmental stimulation. Journal of Counselling and Clinical Psychology, 1976, 44, 693-697.

Zentall, S., Zentall, T. R., & Booth, M. E. Within task stimulation: Effects on activity and spelling performance in hyperactive and normal children. Journal of Educational Research, 1978, 71, 223-230.

Appendix A

Summary of ANOVA for the Children's Age, IQ, and Conners' Teacher Hyperactivity Index Between Groups

	df	MS	F	p<
AGE				
Group	1	16.47	.06	.80
Relationship	1	4.39	.02	.90
Group X Relationship	1	9.95	.04	.85
Residual	84	261.89		
Total	87	253.22		
PPVT IQ EQUIVALENT				
Group	1	960.30	5.21	.03 *
Relationship	1	169.51	.92	.34
Group X Relationship	1	33.15	.18	.67
Residual	84	184.23		
Total	87	191.49		
CTHI				
Group	1	5565.75	757.27	.000***
Relationship	1	.20	.03	.87
Group X Relationship	1	.31	.04	.84
Residual	84	7.48		
Total	87	72.42		

*p<.05

**p<.01

***p<.001

Children's Hospital of Eastern Ontario
Hôpital pour enfants de l'est de l'Ontario

Dear Parents:

This letter is to inform you of a study that is being conducted by the Psychology Department of the Children's Hospital of Eastern Ontario with the assistance of the Psychology Department of the University of Ottawa. The investigation is concerned with examining the relationship between the personality and social characteristics of parents and the behavioural characteristics of their natural or adopted children. More specifically, we are investigating how the event of adoption or natural parentage influences the later adjustment of both parent and child.

Parents who agree to participate will be asked to complete questionnaires concerning their child's behaviour, their marriage, and their own personal attitudes and behaviours. We will also measure each parents responses to a variety of behavioural tasks. The identified son in each family will be asked to complete similar behavioural tasks. The child's teacher will also be requested to fill in a standardized rating form concerning his behaviour at school.

All information provided by parents and children will be kept in strict confidentiality. At no time will the parent's or child's name be identified on any presentation of the data. The objective of this study is to provide scientific data relating to the important question of the effects of adoption or non-adoption to the subsequent adjustment of both parent and child.

You will be contacted by telephone in a few days time in order that we might provide further details and answer any specific questions you or your family might have concerning the study. You will also be asked at that time to make the decision as to whether or not your family wishes to participate in the study. If you agree to participate, appointments will be arranged at your family's convenience.

Thank you for your consideration of this matter.

Sincerely,

Philip Firestone, Ph.D.
Department of Psychology

J. Alberts-Corush

Jody Alberts-Corush, M.S.

Appendix C

ADOPTION STUDY

The Faculty of Psychology at the University of Ottawa and the Psychology Department of the Children's Hospital of Eastern Ontario are currently conducting a study examining the relationship between the personality and social characteristics of parents and the behavioural characteristics of their natural or adopted child. Required are families with an adopted son between 5 and 12, who was adopted prior to six months of age. The study will involve one family session of approximately 2 ½ hours.

FOR MORE INFORMATION CALL

236-5608

between 9 a.m. & 5 p.m.

Appendix D

INFORMED CONSENT

This is to affirm that I was informed of and agreed to participate in an investigation conducted by Dr. Philip Firestone, Department of Psychology, Children's Hospital of Eastern Ontario. I will be asked to fill out several questionnaires and rating scales on my family, their attitudes and my child's behavior. My child's teacher will also be asked to describe various aspects of the child's behavior.

I have been told that my family is free to withdraw from the investigation at any time with absolutely no repercussions and that strict anonymity concerning all information is assured.

(Signed)_____

Relationship_____

Date_____

Witness_____

Children's Hospital of Eastern Ontario

Hôpital pour enfants de l'est de l'Ontario

Dear Parents:

This form authorized Dr. Philip Firestone, or others authorized by him, to contact your child's teacher, school or other concerned agencies. All information will be treated with the strictest confidence and will be used only for clinical teaching or research purposes. At no time will any information that might lead to recognition of the patients involved be made available to non-essential personnel.

Signature _____
Parents or Guardian

Child's name: _____

Address: _____

Telephone no: _____

Date: _____

Appendix F

Summary of Analysis of Variance for Total Family Income Between Groups

	df	MS	F	p<
MAIN EFFECTS				
Group	1	264.94	1.94	.17
Relationship	1	273.76	2.00	.16
TWO-WAY INTERACTION				
Group X Relationship	1	19.28	.14	.71
RESIDUAL	172	136.80		
TOTAL	175	137.73		

*p<.05

**p<.01

***p<.001

Appendix G

Summary of Analysis of Variance for Age Between Groups

	df	MS	F	p<
MAIN EFFECTS				
Group	1	11.60	.37	.55
Relationship	1	849.54	26.92	.000***
Sex	1	134.75	4.27	.04 *
TWO-WAY INTERACTIONS				
Group X Relationship	1	101.99	3.23	.07
Group X Sex	1	8.77	.28	.60
Relationship X Sex	1	.37	.01	.91
THREE-WAY INTERACTION				
Group X Relationship X Sex	1	.74	.02	.88
RESIDUAL	168	31.56		
TOTAL	175	36.66		

*p<.05
**p<.01
***p<.001

Appendix H

Summary of Analysis of Variance for IQ Between Groups

	df	MS	F	p<
MAIN EFFECTS				
Group	1	1349.17	20.80	.000***
Relationship	1	695.35	10.72	.001**
Sex	1	260.20	4.01	.05 *
TWO-WAY INTERACTIONS				
Group X Relationship	1	311.91	4.81	.03 *
Group X Sex	1	2.79	.04	.84
Relationship X Sex	1	7.75	.12	.73
THREE-WAY INTERACTION				
Group X Relationship X Sex	1	29.41	.45	.50
RESIDUAL	168	64.86		
TOTAL	175	77.75		

SIMPLE MAIN EFFECTS - GROUP X RELATIONSHIP

	MS	F
Group at Relationship 1	1544.50	23.81*
Group at Relationship 2	117.48	1.81
Relationship at Group 1	962.13	14.83*
Relationship at Group 2	44.19	.68

Group 1: Hyperactive children
Group 2: Normal control children

Relationship 1: Biological
Relationship 2: Adoptive

*p<.05
**p<.01
***p<.001

Appendix I

Summary of Analysis of Variance for Education Between Groups

	df	MS	F	p<
MAIN EFFECTS				
Group	1	124.34	17.74	.000***
Relationship	1	93.91	13.40	.000***
Sex	1	39.14	5.58	.02*
TWO-WAY INTERACTIONS				
Group X Relationship	1	31.58	4.50	.04*
Group X Sex	1	.40	.06	.81
Relationship X Sex	1	4.57	.65	.42
THREE-WAY INTERACTION				
Group X Relationship X Sex	1	2.43	.35	.56
RESIDUAL	168	7.01		
TOTAL	175	8.46		

SIMPLE MAIN EFFECTS - GROUP X RELATIONSHIP

	MS	F
Group at Relationship 1	146.42	20.89*
Group at Relationship 2	9.55	1.36
Relationship at Group 1	116.58	16.63*
Relationship at Group 2	9.39	1.34

Group 1: Hyperactive children
Group 2: Normal control children

Relationship 1: Biological
Relationship 2: Adoptive

*p<.05

**p<.01

***p<.001

Appendix J

Summary of the 2X2X2 ANOVA for the DRTT

	df	MS	F	p<
GROUP	1	68531.38	28.61	.000***
RELATIONSHIP	1	19727.68	8.24	.01**
SEX	1	28450.38	11.88	.001***
GROUP X RELATIONSHIP	1	23666.23	9.88	.002**
GROUP X SEX	1	11.77	.01	.94
RELATIONSHIP X SEX	1	8033.55	3.34	.07
GROUP X RELATIONSHIP X SEX	1	87.38	.04	.85
RESIDUAL	168	2395.30		
TOTAL	175	3159.37		

SIMPLE MAIN EFFECTS - GROUP X RELATIONSHIP

	MS	F
Group at Relationship 1	89072.28	37.19*
Group at Relationship 2	3118.91	1.30
Relationship at Group 1	43349.86	18.10*
Relationship at Group 2	40.50	.02

Group 1: Hyperactive children
Group 2: Normal control children

Relationship 1: Biological
Relationship 2: Adoptive

*p<.05
**p<.01
***p<.001

Appendix K

Summary of The 2X2X2 ANOVA for The DRTEXT

	df	MS	F	p<
GROUP	1	6.29	1.21	.27
RELATIONSHIP	1	.56	.11	.74
SEX	1	.09	.02	.90
GROUP X RELATIONSHIP	1	2.85	.55	.46
GROUP X SEX	1	.76	.15	.70
RELATIONSHIP X SEX	1	.87	.17	.68
GROUP X RELATIONSHIP X SEX	1	1.28	.24	.62
RESIDUAL	168	5.22		
TOTAL	175	5.08		

*p<.05

**p<.01

***p<.001

Appendix L

Summary of The 2X2X2 ANOVA for The Span Size 1

	df	MS	F	p<
GROUP	1	.00	.01	.93
RELATIONSHIP	1	.06	1.26	.26
SEX	1	.02	.52	.47
GROUP X RELATIONSHIP	1	.02	.38	.54
GROUP X SEX	1	.03	.60	.44
RELATIONSHIP X SEX	1	.11	2.46	.12
GROUP X RELATIONSHIP X SEX	1	.00	.03	.87
RESIDUAL	168	.04		
TOTAL	175	.04		

*p<.05

**p<.01

***p<.001

Appendix M

Summary of The 2X2X2 ANOVA for The Span Size 5

	df	MS	F	p<
GROUP	1	2.75	4.21	.04*
RELATIONSHIP	1	1.79	2.73	.10
SEX	1	.01	.01	.93
GROUP X RELATIONSHIP	1	4.22	6.44	.01**
GROUP X SEX	1	.05	.08	.77
RELATIONSHIP X SEX	1	.05	.08	.78
GROUP X RELATIONSHIP X SEX	1	.00	.00	.96
RESIDUAL	168	.66		
TOTAL	175	.68		

SIMPLE MAIN EFFECTS - GROUP X RELATIONSHIP

	MS	F
Group at Relationship 1	6.76	10.17*
Group at Relationship 2	.23	.35
Relationship at Group 1	5.88	8.84*
Relationship at Group 2	.22	.34

Group 1: Hyperactive children
Group 2: Normal control children

Relationship 1: Biological
Relationship 2: Adoptive

*p<.05
**p<.01
***p<.001

Appendix N

Summary of The 2X2X2 ANOVA for the Span Size 9

	df	MS	F	p<
GROUP	1	27.25	13.32	.000***
RELATIONSHIP	1	14.35	7.02	.01 **
SEX	1	2.51	1.23	.27
GROUP X RELATIONSHIP	1	20.36	9.96	.002**
GROUP X SEX	1	.69	.34	.56
RELATIONSHIP X SEX	1	1.84	.90	.34
GROUP X RELATIONSHIP X SEX	1	1.22	.60	.44
RESIDUAL	168	2.05		
TOTAL	175	2.36		

SIMPLE MAIN EFFECTS - GROUP X RELATIONSHIP

	MS	F
Group at Relationship 1	47.61	23.28*
Group at Relationship 2	.00	.00
Relationship at Group 1	34.29	16.77*
Relationship at Group 2	.18	.09

Group 1: Hyperactive children
Group 2: Normal control children

Relationship 1: Biological
Relationship 2: Adoptive

*p<.05
**p<.01
***p<.001

Appendix O

Results of MANOVA for The Three Impulsivity Variables

	df	F	p<
GROUP	3,166	.45	.72
RELATIONSHIP	3,166	2.22	.09
SEX	3,166	.61	.61
GROUP X RELATIONSHIP	3,166	.62	.60
GROUP X SEX	3,166	1.45	.23
RELATIONSHIP X SEX	3,166	.73	.53
GROUP X RELATIONSHIP X SEX	3,166	.49	.69

*p<.05

**p<.01

***p<.001

Appendix P

Summary of The 2X2X2 ANOVA for Maze Time

	df	MS	F	p<
GROUP	1	3.75	.08	.77
RELATIONSHIP	1	103.03	2.31	.13
SEX	1	.92	.02	.89
GROUP X RELATIONSHIP	1	42.13	.95	.33
GROUP X SEX	1	10.20	.23	.63
RELATIONSHIP X SEX	1	.21	.01	.95
GROUP X RELATIONSHIP X SEX	1	44.23	.99	.32
RESIDUAL	168	44.56		
TOTAL	175	43.96		

*p<.05

**p<.01

***p<.001

Appendix Q

Summary of The 2X2X2 ANOVA for Maze Contacts

	df	MS	F	p<
GROUP	1	295.79	.93	.35
RELATIONSHIP	1	35.36	.11	.74
SEX	1	539.35	1.70	.19
GROUP X RELATIONSHIP	1	446.75	1.41	.24
GROUP X SEX	1	74.85	.24	.63
RELATIONSHIP X SEX	1	373.49	1.18	.28
GROUP X RELATIONSHIP X SEX	1	20.54	.07	.80
RESIDUAL	168	317.64		
TOTAL	175	315.12		

*p<.05
**p<.01
***p<.001

Appendix R

Summary of The 2X2X2 ANOVA for Maze Duration of Contact

	df	MS	F	p<
GROUP	1	.23	.08	.78
RELATIONSHIP	1	2.79	.97	.33
SEX	1	1.54	.54	.47
GROUP X RELATIONSHIP	1	3.71	1.29	.26
GROUP X SEX	1	8.91	3.09	.08
RELATIONSHIP X SEX	1	.02	.01	.94
GROUP X RELATIONSHIP X SEX	1	.16	.05	.82
RESIDUAL	168	2.88		
TOTAL	175	2.87		

*p<.05
**p<.01
***p<.001

Appendix S

Summary of The 2X2X2 ANOVA for The R-S Scale

	df	MS	F	p<
GROUP	1	24.62	.10	.75
RELATIONSHIP	1	2.34	.01	.92
SEX	1	1265.82	5.14	.03*
GROUP X RELATIONSHIP	1	99.28	.40	.53
GROUP X SEX	1	143.72	.58	.45
RELATIONSHIP X SEX	1	112.63	.46	.50
GROUP X RELATIONSHIP X SEX	1	118.85	.48	.48
RESIDUAL	168	246.17		
TOTAL	175	246.46		

*p<.05

**p<.01

***p<.001

Appendix T

Summary of The 2X2X2 ANOVA for The Es Scale

	df	MS	F	p≤
GROUP	1	110.16	7.71	.01**
RELATIONSHIP	1	38.04	2.66	.11
SEX	1	67.51	4.72	.03*
GROUP X RELATIONSHIP	1	46.63	3.26	.07
GROUP X SEX	1	14.40	1.00	.32
RELATIONSHIP X SEX	1	10.62	.74	.39
GROUP X RELATIONSHIP X SEX	1	14.06	.98	.32
RESIDUAL	168	14.30		
TOTAL	175	15.47		

*p≤.05

**p<.01

***p<.001

Appendix U

Summary of The 2X2X2 ANOVA for The TPS

	df	MS	F	p<
GROUP	1	169.12	20.81	.000***
RELATIONSHIP	1	38.38	4.72	.03*
SEX	1	32.82	4.04	.05*
GROUP X RELATIONSHIP	1	77.80	9.57	.002**
GROUP X SEX	1	22.47	2.76	.10
RELATIONSHIP X SEX	1	16.09	1.98	.16
GROUP X RELATIONSHIP X SEX	1	11.49	1.41	.24
RESIDUAL	168	8.13		
TOTAL	175	9.94		

SIMPLE MAIN EFFECTS - GROUP X RELATIONSHIP

	MS	F
Group at Relationship 1	243.00	29.90*
Group at Relationship 2	3.50	.43
Relationship at Group 1	113.63	13.98*
Relationship at Group 2	2.88	.35

Group 1: Hyperactive children
Group 2: Normal control children

Relationship 1: Biological
Relationship 2: Adoptive

*p<.05
**p<.01
***p<.001

Appendix V

Summary of The 2x2x2 ANOVA for The Code 1-3

	df	MS	F	p<
GROUP	1	813.72	4.16	.04*
RELATIONSHIP	1	82.67	.42	.52
SEX	1	12.55	.06	.80
GROUP X RELATIONSHIP	1	709.12	3.63	.06
GROUP X SEX	1	870.86	4.45	.04*
RELATIONSHIP X SEX	1	342.45	1.75	.19
GROUP X RELATIONSHIP X SEX	1	681.62	3.49	.06
RESIDUAL	168	195.52		
TOTAL	175	208.03		

SIMPLE MAIN EFFECTS - GROUP X SEX

	MS	F
Group at Sex 1	1726.10	8.83*
Group at Sex 2	4.40	.02
Sex at Group 1	359.66	1.84
Sex at Group 2	574.26	2.93

Group 1: Hyperactive Children
Group 2: Normal Control Children

Sex 1: Females
Sex 2: Males

*p<.05

**p<.01

***p<.001

Appendix W

Summary of The 2X2X2 ANOVA for The Code 2-4

	df	MS	F	p<
GROUP	1	6339.16	29.49	.000***
RELATIONSHIP	1	1169.56	5.44	.02 *
SEX	1	360.82	1.68	.20
GROUP X RELATIONSHIP	1	2626.22	12.22	.001***
GROUP X SEX	1	550.96	2.56	.11
RELATIONSHIP X SEX	1	67.88	.32	.58
GROUP X RELATIONSHIP X SEX	1	83.41	.39	.53
RESIDUAL	168	214.97		
TOTAL	175	271.27		

SIMPLE MAIN EFFECTS - GROUP X RELATIONSHIP

	MS	F
Group at Relationship 1	8779.69	40.84*
Group at Relationship 2	186.81	.87
Relationship at Group 1	3669.02	17.07*
Relationship at Group 2	123.77	.58

Group 1: Hyperactive children
Group 2: Normal control children

Relationship 1: Biological
Relationship 2: Adoptive

*p<.05
**p<.01
***p<.001

Appendix X

Summary of The 2X2X2 ANOVA for The Code 2-7

	df	MS	F	p<
GROUP	1	2607.67	10.29	.002**
RELATIONSHIP	1	1814.30	7.16	.01 **
SEX	1	592.79	2.34	.13
GROUP X RELATIONSHIP	1	1131.31	4.46	.04 *
GROUP X SEX	1	684.76	2.70	.10
RELATIONSHIP X SEX	1	350.33	1.38	.24
GROUP X RELATIONSHIP X SEX	1	46.21	.18	.67
RESIDUAL	168	253.48		
TOTAL	175	285.46		

SIMPLE MAIN EFFECTS - GROUP X RELATIONSHIP

	MS	F
Group at Relationship 1	3672.36	14.49*
Group at Relationship 2	66.26	.26
Relationship at Group 1	2894.69	11.42*
Relationship at Group 2	50.67	.20

Group 1: Hyperactive children
Group 2: Normal control children

Relationship 1: Biological
Relationship 2: Adoptive

*p<.05
**p<.01
***p<.001

Appendix Y

Summary of The 2X2X2 ANOVA for The Code 4-9

	df	MS	F	p<
GROUP	1	2584.20	11.57	.001***
RELATIONSHIP	1	13.14	.06	.81
SEX	1	429.69	1.92	.17
GROUP X RELATIONSHIP	1	512.09	2.29	.13
GROUP X SEX	1	882.14	3.95	.05 *
RELATIONSHIP X SEX	1	14.28	.06	.80
GROUP X RELATIONSHIP X SEX	1	30.04	.13	.71
RESIDUAL	168	223.36		
TOTAL	175	239.87		

SIMPLE MAIN EFFECTS - GROUP X SEX

	MS	F
Group at Sex 1	3230.01	14.46*
Group at Sex 2	187.64	.84
Sex at Group 1	115.21	.52
Sex at Group 2	1262.26	5.65*

Group 1: Hyperactive children
Group 2: Normal control children

Sex 1: Females
Sex 2: Males

*p<.05
**p<.01
***p<.001