STIMULANT TREATMENT AND THE MIXED ADHD/CD SUBTYPE: AN INVESTIGATION OF ACUTE EFFECTS IN A SAMPLE OF ADOLESCENT OFFENDERS

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Stimulant treatment and the mixed ADHD/CD subtype:

an investigation of acute effects in a sample of adolescent offenders.

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ABSTRACT

Previous research investigating parameters of Attention Deficit Hyperactivity Disorder (ADHD), including response to stimulant drug treatment, has typically failed to control for associated conduct symptomatology, thereby preventing delineation of potentially important subgroup responses. In view of both the markedly high comorbidity of Conduct Disorder (CD) with ADHD and empirical robust indications of a chronic poor prognosis for this subgroup, a need for the evaluation of specific interventions for the mixed ADHD/CD subtype has been voiced. To date, the short-term effects of psychoactive drug treatment have not been researched in mixed ADHD/CD adolescent groups. The present study investigated the acute effects of a 10-day, triple-blind, within-subject trial of placebo and two doses of methylphenidate (MPH) on ADHD symptomatology in a subgroup of male, adolescent, inpatient offenders who met the combined DSM-III diagnostic criteria for ADDH and CD. Thirty-three youths, aged 12 to 17 years, were followed across four randomly assigned and counterbalanced experimental phases of baseline, placebo, and low dose (0.3-mg/kg) and moderate dose (0.5-mg/kg) of MPH. Computerized laboratory measures of sustained attention (vigilance), distractibility, inhibition of impulsive responding (delayed response learning), and responsivity to reward-passive avoidance learning were administered across the four experimental phases. Multivariate and univariate analyses revealed significant medication effects relative to baseline, but not to placebo, levels of performance on 3 of the 7 dependent measures. Contrary to expectation, an overall drug effect was not obtained. A principal component analysis of the drug effect change score data did not indicate the presence of a homogeneous, favourable responder subgroup in terms of subjects' global responses to the active drug. Evaluation of subjects' mean baseline performances did not reveal pre-treatment levels of impairment in attentional capacity, impulsive

responding, and passive avoidance learning on the vigilance, distractibility, and reward dominance-passive avoidance measures. Attentional dysfunction was observed for the sample in association with the introduction of reward and response-cost. Results are discussed in terms of the importance of interindividual variability in severity of attention deficit and interindividual and intraindividual variability of drug treatment response. The implications of the findings for psychopharmacological research with the mixed ADHD/CD subtype and for the identification of favourable responder subgroups are addressed.

DEDICATION

To Maurice and Monica,

whose youthful aspirations were punctuated by a time
when the doors to academic and graduate pursuits
were closed to the jewry,
when women had no real expectations
that the doors might even be ajar.

So go ahead, yes, you can call me "Dactah".
There is triumph
in the success of succeeding generations.
But the true triumph is multi-generational.
It is you who taught me
the value
and the beauty
of the written word.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

PART A

INTRODUCTION

Chapter 1
ADHD: Changing Conceptualizations, Changing Terminology

The past two decades of research and clinical practice in child and adolescent psychopathology have witnessed a continually changing nosology that has been applied to children who show clusters of symptoms of attentional and impulse control difficulties, excessive motor activity, disruptive behaviour, and psychosocial impairment. The shifts in diagnostic labels from Hyperkinetic Reaction of Childhood (DSM-II, American Psychiatric Association, 1968) to Attention Deficit Disorder with or without Hyperactivity (DSM-III, American Psychiatric Association, 1980), to the more recent Attention Deficit Hyperactivity Disorder (DSM-III-R, American Psychiatric Association, 1987), have reflected an attempt to capture the core or essential deficits of the disorder, unlike various historical concepts, such as "organically driven children" (Kahn & Cohen, 1934), "minimal brain damage" (Strauss & Lehtinen, 1947), or "minimal brain dysfunction" (MBD; Clements, 1966; Clements & Peters, 1962) that emphasized organic etiology with no sound empirical basis (Rutter, 1982). Moreover, historical referents such as MBD, hyperactivity, and hyperactive child syndrome (Stewart, Pitts, Craig, & Dieruf, 1966) did not include recognition and specification of the full behavioural syndrome. Consensus was lacking, not only in the variety of terms used, but also in the definition of the disorder and its diagnostic criteria. Primary or core symptoms believed to consititute the disorder have ranged from a low of one - overactivity (Werry & Sprague, 1970) - to an astounding high of 99 (Clements, 1966).

The confusion in terminology and absence of a consensual definition have significantly hampered empirical progress and the comparability of results across research endeavours (Cantwell, 1986a; Douglas, 1983; Ross & Ross, 1982). For example, given that the symptom of hyperactivity was ubiquitously accepted as the sine qua non of the disorder, a large body of research has accrued which purports to have examined parameters of ADHD, but rather is likely based on a population of children who showed high levels of

motor activity rather than the full behavioural syndrome that is implied by the current designation ADHD.[1] Thus, the majority of studies have included a heterogeneous group of children with often unspecified degrees of learning disabilities, overactivity, aggression, brain damage, and family pathology.

The historic reliance on hyperactivity as the defining symptom of the disorder is additionally problematic when the distinctions between sign, trait, and syndrome are considered. As a sign, hyperactivity, or excessive motor activity, may occur in a broad range of psychopathology, such as epilepsy, mental retardation, mania, and schizophrenia. As a trait, an excessively high activity level may be a variant of activity level in normal children. Similarly, attention deficits are also nonspecific traits or signs, and possibly occur in all the disorders mentioned above. In other words, as Carey (1988) has succinctly stated, "Every child who has a rash does not have the Rash Syndrome" (p. 349). The problem of distinguishing between sign, trait, and syndrome is not solved by changes in terminology, but rather by a specific intensional definition of the disorder based on clear clinical inclusionary and exclusionary criteria.

Terminology has had its progressions. The advent of the term Attention Deficit Disorder (ADD; DSM-III, American Psychiatric Association, 1980) reflected an empirical approach to nosology and definition. Terminology and defining criteria for the disorder were based on a decade of intensive research (Douglas, 1972; Douglas & Peters, 1979; Dykman, Ackerman, Clements, & Peters, 1971; Dykman, Peters, & Ackerman, 1973) indicating that these children's inappropriate activity and disruptive behaviours were accompanied by more subtle, but equally important, deficits in cognitive functioning.

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[1] The various diagnostic labels are used interchangeably in this paper. The DSM-III-R descriptor ADHD is the most frequently applied as it is the current label for the disorder. Research on MBD is included when seeming to deal with the same disorder as ADHD. Distinctions among the varying diagnostic labels are drawn when relevant to assessment and comparability of empirical findings.

Three constellations of defining symptoms were specified: inattentiveness, impulsivity, and overactivity, as well as diagnostic inclusionary and exclusionary criteria. The DSM-III descriptor ADD firmly established impaired attention as the central diagnostic concept that may or may not be accompanied by hyperactivity. The DSM-III nomenclature included an additional subtype, termed ADD Residual State, which was intended to classify individuals (adolescents and adults) who met the criteria for ADD in childhood and currently experience symptomatic transformations of core deficits and associated social/occupational impairment.

The current, newly revised classification (DSM-III-R, American Psychiatric Association, 1987) has returned to a single undifferentiated list of symptoms, as is reflected in the shift in terminology to Attention Deficit Hyperactivity Disorder (ADHD), indicating acceptance of the notion that motor hyperactivity and attentional difficulties co-exist as core symptoms of the disorder. The subtypes of associated hyperactivity and Residual State are no longer included in the classification. The list of defining criteria specifies 14 symptoms (two fewer than in DSM-III), and a child must exhibit at least eight, any eight, of these indicators to meet the diagnostic requirements.

Like its predecessor, the DSM-III-R requires that the onset of the disorder occur prior to the age of 7 and that symptoms must be present for a minimum duration of 6 months. These temporal restrictions are intended to minimize false positive diagnoses of children whose symptomatology is a reaction to recent stressful life events. Other exclusionary criteria include conditions such as mental retardation, psychoses, gross neurological disease, pervasive developmental disorder, and severe sensory defects. Although children with any of the latter medical or psychiatric conditions may exhibit clusters of ADHD behaviour, the ADHD symptoms are considered to be secondary to these conditions in that they differ markedly in etiology, associated symptomatology, and outcome.

The more recent nomenclatures, therefore, laid the groundwork for clear and consensual guidelines for syndrome identification. Over the last decade, clinicians and researchers have reached increasing consensus regarding primary and secondary symptomatology (Cantwell, 1979; Douglas & Peters, 1979; Minde, 1977; Rapoport & Zametkin, 1980; Satterfield, Cantwell, & Satterfield, 1979; Whalen & Henker, 1980). Researchers attempting to delineate the nature of the disorder cite overactivity, inattention, and impulsivity as core symptoms that are apparent early in life to an extent that the child is consistently unable to comply with situational demands in an age-appropriate manner (Cantwell, 1975; Ross & Ross, 1976; Routh, 1978; Safer & Allen, 1976). Among the secondary or complicating symptoms noted are learning difficulties, often despite normal intelligence, conduct problems, aggression, low self-esteem, and poor social competence. The less salient aspects of the disorder are referred to as associated symptoms in that they are attributed to the ADHD child's flawed interactions with his social environment. However, at present, there is no empirical basis supporting this causal assumption (Milich & Loney, 1979; Ross & Ross, 1982).

Although there is widespread agreement regarding the triad of primary symptoms, unresolved issues in the definition and diagnosis of the disorder remain. These include: (1) how to best operationalize the symptoms (Pelham & Murphy, 1986); (2) the relative weighting of symptoms in diagnosis (Loney, 1982); (3) whether additional symptoms should be included in the intensional definition (Barkley, 1982); and (4) the actual nature of the putative cognitive deficits (Douglas, 1983; Pelham & Murphy, 1986).

Likely a byproduct of, as well as an influence on, changes in terminology, scientific interest in the study of children and adolescents with ADHD has dramatically increased over the past 20 years. It has been estimated that 31 articles were published in the scientific literature between the years 1957 and 1960, as compared to 700 articles published

between the years 1977 and 1980 (as cited in Weiss & Trokenberg Hechtman, 1986). The ADHD syndrome is currently recognized for its status as the most extensively researched and widely discussed of the childhood behaviour disorders (Cantwell, 1986a; Ross & Ross, 1982; Weiss et al., 1986). Various explanations have been proposed to account for this (e.g., Cantwell, 1986a; Weiss et al., 1986). These include: (1) the high prevalence of the behaviour disorder and its comorbidity with other psychiatric disorders; (2) the distressing nature of primary and secondary symptoms to the ADHD individual and his environment; (3) the core features of the syndrome yield well to experimental investigation and intervention; (4) the finding that ADHD is not limited to childhood but, in fact, often persists into adolescence and adulthood with varying manifestations; and (5) the discovery of the efficacy of stimulant medication on the behavioural and cognitive aspects of the syndrome.

It is these areas -- morbidity and comorbidity, outcome in adolescence and early adulthood, empirical investigation of core features, and assessment of pharmacological intervention -- that provide the historical and conceptual background for the current research. Since it is the author's opinion that ADHD cannot be adequately discussed independent of issues surrounding terminology and taxonomic validity, the first major theme of this review will be to address difficulties in establishing the morbidity of the disorder and the related problem of heterogeneity within the ADHD population. Accordingly, the very issues of nosology and classification of a homogeneous patient population in epidemiological research on ADHD speak to the diagnostic boundaries of the category and its comorbidity with other psychiatric disorders. In particular, the overlap of ADHD with Conduct Disorder (CD) is of special relevance to the present study. The identification and validation of more homogeneous subgroups of ADHD based on associated conduct symptomatology is presently in vogue. The rationale for such a subclassification, based on empirical findings from studies of syndrome comorbidity, factor analyses of syndromal independence, external validity of homogenous subgrouping, and outcome

research on the course of the ADHD syndrome, is discussed. A subsequent section addresses empirical findings underlying current conceptualizations of the core features of ADHD, and is meant to provide a framework for the use of particular assessment measures in the present investigation of a selected treatment option for the mixed ADHD/CD subtype. A final area of review embarks in a decidedly more pragmatic direction. Here, the current status of stimulant treatment for the population of ADHD children and adolescents is explored. The primary emphasis is on empirical findings, clinical facts, and practical considerations relevant to the use of pharmacological treatment with adolescents who have ADHD concurrent with CD. To begin then, a review addressing the epidemiology of the syndrome is in order.

ADHD: Prevalence in Childhood and Adolescence

Differences in terminology have been reflected in wide fluctuations in estimations of the prevalence of the disorder. Prevalence rates reported in the American and Canadian child psychiatry literature have varied from a low of 1% to 14.3% (Szatmari, Offord, & Boyle, 1989a; Trites, 1979), to a high of 20% (Huessy & Gendron, 1973; Trites, 1979). The most frequently reported estimates fall in the range of 5% to 10% (Wender, 1971); however, the consensual opinion in the child psychopathology literature emphasizes an overall rate of 3% to 5% in the population of school-age children as a reasonable and conservative estimate of the prevalence of ADHD (Lambert, Sandoval, & Sassone, 1978; Trites, 1979; Varley, 1984). The reported sex ratios of males to females have ranged from 5:1 in community samples to 9:1 in clinic samples (Bosco & Robin, 1980; DSM-III-R, American Psychiatric Association, 1987; Sandoval, Lambert, & Sassone, 1980; Szatmari et al., 1989a). Reviews of the literature have consistently indicated that ADHD is the most common behaviour disorder in the preadolescent group (Ross & Ross, 1982; Wender, 1975), accounting for 30 percent to 50 percent of child behaviour problem referrals (Miller, Palkes, & Stewart, 1973; Safer & Allen, 1976; Stewart, et al., 1966).

The prevalence rate of ADHD in adolescence was investigated in only one community study (Szatmari et al., 1989a). In a survey (the Ontario Child Health Study) of a sample of several hundred Ontario children aged 4 to 16 years old, in which measurement of ADHD was based on DSM-III criteria and multiple data sources, overall prevalence rates of 9% for males and 3.3% for females were obtained, which generally fall in the middle range of other studies. The reported prevalence rate for 12 to 16 year olds was 7.3% for males and 3.4% for females. Previous clinic reports (Aman, 1984) have suggested a significant decrease in the prevalence of ADHD in adolescence, a finding which is not supported by the Ontario community study. However, no information was obtained in the Ontario study regarding age of onset or duration, a methodological limitation which may have contributed to inflated prevalence rates. Moreover, additional data on the prevalence of pervasive, as contrasted with situational, ADHD in the Ontario sample indicated that the pervasive type is actually quite rare in childhood and possibly non-existent in adolescence -- only 15.5% of the 4 to 11 year-old ADHD males were rated as pervasive and virtually no cases of pervasive ADHD in girls or in the adolescent group were observed. These findings would suggest that the severity of ADHD symptomatology may diminish with age, and/or that the clinical picture is modified cross-situationally as the child matures and enters the adolescent age range.

Sources of Variation in Prevalence Rates

Recently, Szatmari, Offord, and Boyle (1989a) reviewed the findings of 11 epidemiological studies examining prevalence rates of childhood "hyperactivity" and "ADD" in community samples across cultures. They identified four important sources of variation which likely contribute to observed fluctuations in reported prevalence rates.

The first source of variation addressed the aforementioned historical problem of lack of both symptom specificity and a clear definition of the disorder. Although the majority of the studies defined the sample group in terms of the three defining features of ADHD (inattention, impulsivity, and overactivity) (Glow, 1980; Holborow, Berry, & Elkins, 1984; Lambert et al., 1978; McGee, Williams, Bradshaw, Chapel, Robins, & Silva, 1985; Rutter, Tizard, & Whitmore, 1970; Schachar, Rutter, & Smith, 1981; Shekim, Kashani, Beck, Cantwell, Martin, Rosenberg, & Costello, 1985), not all studies used the same symptoms to define the disorder. Thus, several investigators (Miller et al., 1973; Nichols & Chen, 1977; Trites, Dugas, Lynch, & Ferguson, 1979; Werner, Bierman, French, Simonian, Connor, Smith, & Campbell, 1968) included symptoms such as low frustration tolerance, irritability, temper tantrums, and poor peer relationships as inclusionary criteria.

Secondly, studies have varied with respect to the diagnostic procedures for identifying ADHD children. Methods have included clinical interviews, rating scales, questionnaires, and direct observation. Sources of diagnostic information have also differed in important ways, as have cutoff criteria for arriving at diagnoses, particularly in cases where rating scales and questionnaires were employed. Thus, differences in sample characteristics have likely contributed to variation in prevalence data.

An additional source of variation in estimates of the incidence of ADHD is the rater -- the professional group or discipline conducting the assessment and formulating the diagnosis (Prinz & Loney, 1974; Taylor & Sandberg, 1984). It has been suggested that teachers overestimate ADHD relative to clinic staff (Prinz et al., 1974), although other researchers have disputed this conclusion (Rubin & Balow, 1971). In particular, the influence on diagnosis of cross-cultural variation in the rater has been widely addressed in the epidemiological and clinical literature on ADHD. Taylor and Sandberg (1984) called attention to a 20-fold variation in the diagnostic rate of ADHD between the U.S. and the U.K. ADHD is reportedly diagnosed in 40% of children seen by U.S. psychiatrists

(Greenberg & Lipman, 1971; Gross & Wilson, 1974) as compared to 1.6% of children of normal intelligence seen by psychiatrists in the Isle of Wight studies (Rutter et al., 1970). This marked disparity in obtained prevalence rates between nations is largely attributable to differences in diagnostic practice (Prendergast, Taylor, Rapoport, Bartko, Donnelly, Zametkin, Ahearn, Dunn, & Wieselberg; 1988; Varley, 1984) rather than to cross-cultural differences in rates of psychopathology.

Research examining cross-national diagnoses of ADHD reveal that both the diagnostic scheme, or nomenclature, and clinician training contribute to differences in reported prevalence rates (Prendergast et al., 1988). For example, both the DSM-III-R and the International Classification of Diseases (ICD-9; World Health Organization, 1978) recognize ADHD as a disorder, but vary in their intensional definitions and terminology. The British reserve the referent "hyperactivity", or ADHD, for severe cross-situationally deviant children with marked cognitive deficits (Sandberg, Rutter & Taylor, 1978), whereas, as is evident in the DSM-III-R criteria, the U.S. practice does not require that the symptom behaviours be pervasive. Moreover, most children diagnosed with ADHD in the U.S. and Canada would be diagnosed with a conduct disorder in Britain (Ferguson & Rapoport, 1983; Rutter, 1983; Rutter & Garmezy, 1983). The British-employed ICD-9 scheme expects a single diagnosis while the DSM-III and DSM-lll-R explicitly recognize a frequent need for multiple diagnoses. Hence, children presenting with conduct symptomatology in Great Britain are diagnosed as having only a conduct disorder, whether or not ADHD symptoms are also present.

Other research indicates that when the diagnostic criteria, the sampling method, and the method of assessment used are similar across studies, prevalence rates of ADHD in Australia, Germany, New Zealand, U.S.A., and Britain show remarkable agreement (Holborow & Berry, 1986), suggesting that, in fact, variation in prevalence is an artifact of the diagnostic decision process.

A further source of variation which contributes to discrepant findings from epidemiological studies on ADHD is the heterogeneous composition of the ADHD population. It was earlier assumed that ADHD was a behavioural syndrome with a common basic symptom pattern, etiology, and response to treatment (e.g., Stewart et al., 1966). The consistent conclusion drawn from more recent empirical investigations, however, is that children given the label of ADHD are heterogenous along a number of dimensions (Loneys, 1980; Ullman, Barkley, & Brown, 1978; Whalen & Henker, 1980).

Children with ADHD vary with regard to onset, course of the disorder, constellation of associated symptoms, biological findings, and response to pharmacological, behavioural, and cognitive-behavioural interventions (Amado & Lustman, 1982; Cantwell, 1985). The nature of ADHD symptomatology has been found to be erratic and variable as a function of time and situational factors (Barkley & Ullman, 1975; Routh & Schroeder, 1976; Schleifer, Weiss, Cohen, Elman, Cvejic, & Kruger, 1975; Whalen & Henker, 1980), and the failure to distinguish between situational and cross-situational ADHD has been found to contribute to discrepant prevalance rates in cross-national epidemiological studies (Lambert et al., 1978). ADHD symptomatology has further been observed in other clinical groups of children and adolescents (Firestone & Martin, 1979; Sandberg et al., 1978), as well as in non-referred school-age samples (Prinz, Connor, & Wilson, 1981). Although reviews of drug studies (e.g., Barkley, 1977) indicate that up to 75% of ADHD children can be classified as positive short-term responders to stimulant medication, which would suggest the presence of a relatively homogeneous group, recent research (Douglas, Barr, Amin, O'Neill, & Britton, 1988; Rapport, DuPaul, Stoner, & Jones, 1986; Ullman et al., 1978) has shown that, even among those ADHD children and adolescents who show a clear positive response to stimulant therapy, there is marked heterogeneity in terms of their major symptom behaviours on and off drugs.

An additional facet of the heterogeneity problem in epidemiological research on ADHD, which is of special relevance to the present study, is the issue of the comorbidity of ADHD with other psychiatric disorders, in particular, with CD. Affective disorders have frequently been reported in ADHD children and adolescents (Carlson & Cantwell, 1980; Munir, Biederman, & Knee, 1987), as have communication disorders and learning disabilities (August & Garfinkel, 1990; Livingston, Dykman, & Ackerman, 1990; Munir et al., 1987; Silver, 1981). Perhaps one of the most controversial issues in the child and adolescent psychiatry literature, however, is the overlap of ADHD with CD and delinquency (August, Stewart, & Holmes, 1983; Barkley, 1982; Cantwell, 1986b; Rutter, 1983; Sandberg et al., 1978; Shaffer & Greenhill, 1979; Stewart, Cummings, Singer, & DeBlois, 1981). Findings in this area have sparked considerable debate over the taxonomic validity of ADHD as distinct from CD, paralleling the diagnostic debate over appropriate terminology.

Chapter 2
ADHD And Its Relationship To Conduct Disorder

ADHD and CD: "Pure" Versus "Mixed" Groups

Conduct Disorder, along with ADHD, is one of the most common behaviour disorders in childhood (Pelham & Murphy, 1986) and has been considered by some to comprise the largest group of adolescent psychiatric disorders (e.g., Kashani, Daniel, Sulzberger, Rosenberg, & Reid, 1987). General population surveys of school-age children (Graham, 1977; Quay, 1979; Rutter & Giller, 1983; Shapiro & Garfinkel, 1986) and studies of clinic-referred samples (Rutter, Shaffer, & Shepherd, 1975) have consistently found disturbances of conduct to be among the most frequent causes of concern regarding children's behaviour.

As a syndrome, or constellation of symptoms distinct from isolated antisocial acts, CD is marked by repetitive patterns (at least 6 months' duration) of antisocial, rule-breaking, and disruptive behaviours. The prevalence of CD varies with the stringency of the diagnostic criteria employed in epidemiological research; estimates range from 3% to 8% of child populations, with U.S. estimates falling in lower ranges than U.K. ones (LaGreca & Quay, 1984; Schwarz, 1985). Again, as with ADHD, terminological and diagnostic differences are at issue here, as seen in the above-mentioned DSM-III-R concept of ADHD and the ICD-9 concept of CD (Taylor & Sandberg, 1984).

Emphasizing the topographical similarity of ADHD as diagnosed in the U.S. and CD as diagnosed in the U.K., various investigators, particularly British researchers such as Shaffer and Greenhill (1979) and Sandberg, Rutter, and Taylor (1978), have contended that ADHD and CD are variants of the same condition. In fact, behavioural symptoms that define ADHD (inattention, impulsivity, and hyperactivity) and conduct problems

(aggression and noncompliance) are difficult to distinguish in preschool (Campbell & Cluss, 1982; Campbell, Szumowski, Ewing, Gluck, & Breaux, 1982) and school-age samples (Prinz et al., 1981), largely because the prominence of particular behaviours may vary as a function of situational and task demands. Moreover, symptoms of ADHD, such as excesses of motor activity, impulsivity, and inattentiveness, are commonly seen in children and adolescents with CD (Kazdin, 1987; Sandberg et al., 1978; Stewart, DeBlois, & Cummings, 1980; Stewart et al., 1981), as are conduct problems and aggressiveness typically observed in children and adolescents with ADHD.

ADHD and CD: Studies of Syndrome Comorbidity

This finding of a large overlap between the symptoms of ADHD and CD has been observed cross-culturally by virtually all studies of the general population (Prinz et al., 1981; Sandberg, Wieselberg, & Shaffer, 1980; Shapiro & Garfinkel, 1986; Szatmari et al., 1989a; Szatmari, Boyle, & Offord, 1989b) and clinic samples (Biederman, Munir, & Knee, 1987; McGee, Williams, & Silva, 1984; Munir et al., 1987; Sandberg et al., 1978; Stewart et al., 1981). For example, a syndrome comorbidity study conducted by Stewart and coworkers in Iowa (Stewart et al., 1981) on an inpatient service found that 67% of children (aged 5 to 14) with CD also met the diagnostic research criteria for ADHD, while 61% of children diagnosed with ADHD had associated CD. Other psychiatric comorbidity research examining the problem of heterogeneity within child and adolescent ADHD samples has shown CD and ADHD overlap rates of 64% (Munir et al., 1987), 40% to 50% in mixed child and youth samples (Hamden-Allen, Stewart, & Beeghly, 1989), and 47% in youth-only samples (Szatmari et al., 1989a, 1989b).

In conclusion, national and cross-national epidemiological research on the morbidity of ADHD and its comorbidity with CD, as well as studies of cross-cultural variations in diagnostic practice, raise the issue of whether ADHD and CD represent a continuum of

impulsivity and attentional problems rather than separate diagnostic entities. Consideration of this issue raises a number of questions. The first addresses whether primary ADHD symptoms cluster together and are differentiated from primary conduct problems of aggression and antisocial behaviour. This question has generally been studied by means of factor analytic research, which takes a dimensional approach to behavioural classification, and typically relies on quantitative scores of ratings of behavioural features as its data base. A second question speaks to the discriminant validity of homogeneous subclassifications based on mixed ADHD/CD, only ADHD, and only CD symptomatology.

ADHD and CD: Factor Analytic Reports of Syndromal Independence

The majority of factor analytic studies have involved the use of parent and teacher rating instruments, with extreme scores on scales reflecting ADHD and CD symptomatology, that are used to define the presence of the disorders (Achenbach, 1978; Goyette, Conners, & Ulrich, 1978; Herbert, 1974; Thorley, 1983; Werry, Sprague, & Cohen, 1975). Data analyses have typically yielded separate factors on which symptoms of ADHD and CD load (Achenbach & Edelbrock, 1978; Quay, 1979), however, considerable shared variance between the ADHD and CD factors has also frequently been observed (Quay, 1979).

In an extensive review by Hinshaw (1987) of 60 empirical factor analytic studies published since 1970, that excluded reports of exclusively learning disabled samples, 41 of the 60 studies reported orthogonal CD and ADHD factors. Athough the precise composition of the factors varied across studies, variations in the composition of the samples, in the rater, and in the type of extraction method used (principal factor analysis versus principal component analysis) were found to be not statistically relevant to the presence or absence of findings of factor orthogonality. Failures to obtain separate ADHD and CD factors were most often attributed, by Hinshaw, to restricted item-pool composition

and problems with broad-band versus narrow-band factor analyses. However, despite the emergence of separate factor domains, robust findings of moderate to high levels of association between the pertinent dimensions were obtained, particularly when the Conners Parent and Teacher Rating Scales (CPRS and CTRS; Conners, 1969, 1970, 1973) were used as rating instruments.

In view of substantial findings of shared variance between the ADHD and CD factors, Hinshaw concluded that it then becomes pertinent to examine the question of possible differential patterns of association with criterion measures of interest. Indeed, the groundbreaking approach of Loney and her associates (Paternite, Loney, & Langhorne, 1976; Loney, Langhorne, & Paternite, 1978) to scale construction and validation demonstrated that careful item selection lowers correlations between empirical factors and, when aggressive and ADHD symptomatology are analyzed concurrently, many key etiologic, prognostic, treatment-response, and concurrent mediating and descriptive variables are differentially related to the two symptom dimensions (Loney, Kramer, & Milich, 1981; Milich, Loney, & Landau, 1982; Paternite & Loney, 1980). For example, factor analysis of the primary and secondary symptomatology at referral of 135 ADHD boys yielded two orthogonal symptom dimensions: Aggression and Hyperactivity (Loney et al., 1978; Paternite et al., 1976). The Hyperactivity factor correlated significantly with impulsivity, poor social competence, visual motor difficulties, and favourable response to stimulant drug treatment. Conversely, the Aggression factor scores were observed to be systematically related to age, socioeconomic status (SES), parenting styles, and delinquency in adolescence. A later series of investigations (Loney et al., 1981; Milich et al., 1982; Paternite & Loney, 1980) strongly indicated that separate consideration of the two domains of primary and secondary symptomatology is critical to prognosis. (See subsequent section titled ADHD and Adolescent Antisocial Outcome: A Developmental Risk Factor for CD?).

The series of studies by Loney and associates, therefore, provide good evidence of the independent relations of ADHD and aggression dimensions with a variety of criterion measures. Overall, the extant factor analytic research suggests that conduct problems and ADHD are best conceptualized as related dimensions of behaviour and that concurrent information regarding both dimensions of behaviour is critical to an adequate understanding of the disorders and their correlates.

An important limitation of the factor analytic research is that dimensions of behaviour, as opposed to clear categorical diagnoses, are investigated for their clinical and empirical relevance. The dimensional approach to classification assumes that key features are continuously and normally distributed in a population, whereas the categorical approach assumes that key features or symptoms constitute discrete types or classes (Hinshaw, 1987; Werry, Reeves, & Elkind, 1987). Many of the conclusions derived from factor analytic research on the association between ADHD and CD involved the extrapolation of high scores on a dimension of behaviour to membership in a discrete diagnostic category, a leap of faith which is problematic, at best. This practice would be analogous to the medical practice of diagnosing hypertension based on a high score on the blood pressure dimension, irrespective of whether the hypertension also clustered with renal symptoms, high blood catecholamine levels, or signs of raised intracranial pressure, all of which would indicate three quite different disease states (Werry et al., 1987).

ADHD and CD: External Validity of Homogeneous Subgrouping

Accordingly, the separation and validation of categorically diagnosed subgroups within the ADHD category is required to address the more clinically and empirically relevant question of whether there are meaningful differences between samples who show mixed ADHD/CD symptomatology and those who show only ADHD, or only CD, symptomatology (Achenbach & Edelbrock, 1978; Rutter, 1983). One beneficial offshoot of

the heterogeneity problem and the issue of the overlap with CD has been an intensification of empirical and clinical interest in the identification and study of ADHD subgroups. This is based on the assumption that the observed heterogeneity in the ADHD population is an artifact, or product, of the presence of several relatively homogeneous subgroups of children and adolescents. Examples of subgroups that have been considered in the literature include the differentiation between pervasive cross-situational ADHD and situational ADHD (Campbell, Endman, & Benfield, 1977; Sandberg et al., 1978; Schachar et al., 1981), as well as between "pure" ADHD and ADHD with co-occurring CD (Stewart et al., 1981). Subtyping strategies have largely focused on behaviour (e.g., Sandberg et al., 1978; Schachar et al., 1981) and/or the presence of associated symptomatology as the bases of identifying specific subgroups (e.g., Stewart et al., 1981). One such approach has been the identification of "pure" versus "mixed" ADHD/CD groups, a subclassification which has received support from research on external validation and long-term outcome.

Empirical research examining the correlates of "mixed" as compared to "pure" ADHD and CD groups was largely pioneered by the research groups of Stewart (August & Stewart, 1982; August & Stewart, 1983; August et al., 1983; Stewart et al., 1981) and Loney (Langhorne & Loney, 1979; Loney & Milich, 1982; Milich et al., 1982). Early work by Stewart and his coworkers (Stewart et al., 1980, 1981), investigating the empirical validity of homogeneous subtype classification based on ADHD and CD symptomatology, suggested that child aggression and CD, not ADHD, were associated with antisocial disturbance and alcoholism in parents -- a finding that is in contrast to other research which failed to include an ADHD/CD distinction (Cantwell, 1972; Morrison & Stewart, 1971, 1973). The validity and generalizability of these data are limited, however, in that diagnostic classification was exclusively based on parental interview data.

Later work by Stewart and his associates (August & Stewart, 1982, 1983) that utilized multiple data sources to identify a group of 126 ADHD boys with pervasive ADHD,

and then, within the pervasive group, compared those with and without associated CD, provided stronger evidence for subgroup independence. The pure ADHD group exhibited greater problems in academic achievement and lower Verbal IQ scores when compared to the mixed ADHD/CD group. However, prenatal/perinatal, developmental, and neurological measures failed to distinguish the two groups. Interestingly, the finding of an association between academic underachievement and pure ADHD, as contrasted with mixed ADHD and CD, is supported by recent data (Frick, Kamphaus, Lahey, Loeber, Christ, Hart, & Tannenbaum, 1991) suggesting that the relation between CD and school learning difficulties observed in other studies (Broder, Dunivant, Smith, & Sutton, 1981; Morgan, 1979; Pasternack & Lyon, 1982; Rutter & Yule, 1970) is likely due to the comorbidity of CD with ADHD.

Further analysis of August and Stewart's sample from their 1982 study, using presence or absence of antisocial spectrum symptomatology in the biological parents of the probands as a basis for sample division, suggested the presence of two distinct clinical subtypes of ADHD (August & Stewart, 1983). Pure ADHD was positively associated with intellectual and academic deficits, a likelihood of siblings having attentional and learning disabilities, but not conduct problems, and an absence of antisocial spectrum disturbance in parents. Conversely, the mixed ADHD/CD group tended to have siblings with conduct but not attentional problems, was more likely to come from broken homes, and was positively associated with antisocial psychopathology and alcoholism in parents. Recent research examining the parental psychiatric comorbidity of pure and mixed ADHD/CD samples of clinic-referred children provide further support for a link between CD, but not ADHD, and parental psychopathology (Biederman et al., 1987; Lahey, Piacentini, McBurnett, Stone, Hartdagen, & Hynd, 1988; Reeves, Werry, Elkind, & Zametkin, 1987). Further, the co-occurrence of CD with ADHD is strongly associated with a greater severity of aggression and antisocial activity in both children and their fathers (Lahey et al., 1988; Walker, Lahey, Hynd, & Frame, 1987).

Loney and colleagues have supplemented their previously discussed factorial work with subgrouping strategies. Although the generalizability and validity of this research is limited in that diagnostic classification was based on questionnaire cutoff scores (Langhorne & Loney, 1979; Loney & Milich, 1982), findings generally supported distinctions between mixed and pure ADHD subgroups in terms of visual motoric skills, classroom and observational measures, and peer-derived variables. Data indicated that mixed, as compared to pure, ADHD/CD groups were at a particularly high risk for poor social outcome and were more often rejected, and designated severe behaviour problem children, by peers. Other research on external validation of childhood psychopathology (Reeves et al., 1987) supports the finding that the concurrence of ADHD and CD in clinic-referred samples, as compared to anxiety disorders and a single diagnosis of ADHD, is associated with greater social dysfunction.

The data, therefore, indicate that the presence of concurrent CD in ADHD samples is associated with greater behavioural and psychosocial maladjustment. In fact, a correlation between mixed ADHD/CD symptomatology and increased incidence of antisocial behaviour in childhood and adolescence has been a robust finding in the literature, even when controls with primary features of delinquency and unsociability were included in designs. For example, Offord, Sullivan, Allen, and Abrams (1979) observed that retrospectively diagnosed ADHD delinquents had an earlier age of onset and demonstrated significantly more aggression and antisocial symptoms than non-ADHD delinquents. A study conducted by the present author (Reznick & Freeman, 1985) found striking differences in antisocial conduct between mixed ADHD/delinquent and non-ADHD delinquent groups; ADHD offenders committed 3 times the number of serious and non-serious offences, served 8 times the amount of institution time, and 700 times the amount of remand time of the non-ADHD offenders. The mixed ADHD/delinquent subjects also displayed significantly more psychiatric disturbance than the delinquent controls on the

Child Behaviour Checklist (CBCL; Achenbach & Edelbrock, 1983), including a greater incidence of reported suicidal ideation, aggression, peer difficulties, and general behaviour problems. Similarly, studies by Schachar, Rutter, and Smith (1981) and Magnusson, Slottin, and Duner (1983) indicated that the antisocial conduct observed in mixed ADHD/CD, as compared to pure CD, groups of children was more likely to persist or worsen. More recent data (Loeber & Schmaling, 1985; Moffit, 1990; Walker et al., 1987) are consistent with the finding that the co-occurrence of CD and ADHD is associated with a more serious form of CD.

Recently, Szatamari, Boyle, and Offord (1989b) reanalyzed their cross-sectional data on pure versus mixed ADHD/CD probands from the Ontario Health Study (Szatamari et al., 1989a). They observed that the diagnostic overlap of ADHD and CD was far higher than would be expected by chance -- ADHD males were 14 times more likely to have associated CD than were non-ADHD males. Results further indicated the presence of a differential pattern of correlates for the pure as compared to the mixed groups. CD children and youths were significantly older and had experienced greater psychosocial disadvantage and fewer developmental delays than the pure ADHD group. The co-occurrence of ADHD and CD, however, was associated with the psychosocial and developmental correlates found for both the pure CD and pure ADHD groups. Thus, similar to the ADHD group, the mixed ADHD/CD probands had a greater number of developmental delays than did the CD group, but, like the CD children, they had a history of greater psychosocial adversity than did the ADHD children. These findings suggest the possibility that ADHD, in interaction with psychosocial stress, is linked with the development of associated CD. As the authors speculated, children and adolescents who present with both ADHD and CD may "represent a true hybrid disorder rather than one diagnosis or the other" (Szatamari et al., 1989, p. 865).

At this juncture, several conclusions are warranted. The studies of syndrome comorbidity, factorial independence, and the discriminant validity of ADHD subtypes based on the presence or absence of co-existing CD provide unequivocal evidence of heterogeneity within the ADHD syndrome and, further, indicate that a subgroup of ADHD children with antisocial comorbidity can be identified. Although it is possible to identify pure ADHD and pure CD groups, more often there is a substantial degree of overlap with 30% to 90% of children exhibiting both disorders concurrently (Hinshaw, 1987; Lahey et al., 1988). Despite this overlap, there are robust indications that the specific comorbidity of CD with ADHD offers discriminant validity in a number of areas, including peer relationship difficulties, severity of psychosocial impairment and antisocial conduct, (fewer) academic and neurodevelopmental difficulties, and association with antisocial spectrum disturbance in biological parents.

The relationship between ADHD and CD is therefore far more complex than earlier supposed and likely does not reflect a single underlying disorder. Several different relationships between the two conditions are possible. One explanation is that children who are primarily CD exhibit restlessness, inattentiveness, and impulsiveness as nonspecific secondary symptoms rather than as a consequence of any underlying pathology (Schachar, 1989). This is suggested because data indicate CD is primarily linked with psychosocial adversity (Rutter & Giller, 1983; Werry et al., 1987; Szatamari et al., 1989b). An alternative explanation which has received moderate support (Cantwell, 1975, 1978; Satterfield, 1978; Satterfield & Cantwell, 1978) is that ADHD, in its more severe form, may act as a predisposing factor which places a child at risk for the development of associated CD. This latter approach to understanding the diagnostic overlap of ADHD with CD is based on outcome data that suggest a developmental association between the two disorders.

ADHD and Antisocial Outcome in Adolescence: A Developmental Risk Factor for CD?

Concomitant with changes in terminology over the past two decades, there has been a major shift in understanding the prognosis of the ADHD syndrome. Indeed, the emergence of ADHD as a possible risk factor in the development of antisocial behaviour in adolescence constitutes a fascinating chapter in the history of research and clinical lore on childhood hyperactivity. Prior to 1970, clinical reports tended to emphasize that it was a time-limited condition that diminished with age or disappeared completely with the advent of adolescence (Bakwin & Bakwin, 1966; Eisenberg, 1966; Laufer & Denhoff, 1957; Lytton & Knobel, 1958). This view characterized the "developmental delay" (e.g., as discussed in Loney et al., 1981), or "maturational lag", theory of adolescent outcome which postulated a benign prognosis. However, some 30 retrospective and prospective studies directly addressing the sequelae of ADHD that have been published over the past two decades have unequivocally refuted the view that ADHD is a time-limited condition with a uniform course. Although the symptom of hyperactivity per se may diminish with age (Ackerman, Dykman, & Peters, 1977; August et al., 1983; Loney, 1980; Weiss, 1975), disorders of attention, concentration, impulsivity, and irritability continue into adolescence and adult life in a substantial proportion of ADHD children (Borland & Heckman, 1976; Mendelson, Johnson, & Stewart, 1971; Minde, Weiss, & Mendelson, 1972; Weiss, Hechtman, Perlman, Hopkins, & Wener, 1979). Serious emotional and educational sequelae may persist as well (Dykman, Peters, & Ackerman, 1973; Hechtman, Weiss, & Perlman, 1981; Menkes, Rowe, & Menkes, 1967; Milman, 1979; Minde, Lewin, Weiss, Lavigeur, Douglas, & Sykes, 1971; Morrison, 1980).

Outcome studies further suggest that ADHD may continue into adolescence with symptom transformations. This is consistent with a second postulated outcome of ADHD, the "continual display theory" (e.g., as discussed in Cantwell, 1985a, 1986a), which

suggests that core ADHD symptoms persist in modified form. This view is reflected in the DSM-III diagnostic subtype of ADD Residual State. Within symptom modalities, the form of the behaviour changes through the developmental stages, paralleling changes in maturation and functioning (Ross & Ross, 1982). For example, manifestations of deficient impulse control may take the form of impaired sphincter control (enuresis, encopresis) during infancy (Wender & Eisenberg, 1974), accident-proneness during the preschool years (Stewart, Thach, & Freidin, 1970), low frustration tolerance, inability to delay gratification (Ross & Ross, 1982) and negative interactions with peers (Klein & Young, 1979; Riddle & Rapoport, 1976) during middle childhood, and truancy and antisocial behaviour during adolescence (Loeber, 1990). The behaviours perceived as most serious and problematic also change with development and maturation (Ross & Ross, 1982). Sleeping problems and crying are most salient in infancy, hyperactivity is the most conspicuous problem in middle childhood, and rebelliousness and antisocial behaviour constitute the predominant problems in adolescence. In fact, delinquent behaviour is the major reason for referral during the adolescent years (Wender & Eisenberg, 1974) and outcome research on ADHD probands followed into adolescence and early adulthood report a marked increase in antisocial symptoms and delinquent acts at follow-up (see Table 1).

Due to changing behavioural manifestations of the syndrome, core deficits that persist into adolescence and early adulthood may attract a new set of diagnostic labels. This latter course is postulated by the "developmental decay theory" of outcome (e.g., as outlined in Cantwell, 1985a, 1986a) which suggests that ADHD in childhood predisposes the development of particular psychopathology in adolescence. Of relevance to the present study are the diagnoses of CD and antisocial personality disorder (APD), which are frequent findings at follow-up in outcome studies of ADHD children (see Table 1).

Current knowledge of the changing behavioural manifestations of the ADHD syndrome (the continual display theory), and the possibility of a developmental association

between ADHD in childhood and CD in adolescence (the developmental decay theory), draws from three types of outcome research: retrospective, cross-sectional and prospective longitudinal designs.

Outcome studies of ADHD children are summarized in Table 1 and are classified as retrospective (post-facto), follow-back, longitudinal prospective, or cross-sectional follow-up methodology. Retrospective follow-back involves assessment of diagnosis based on the recollections of the patient and/or family; follow-back assessment involves rating subjects' psychopathology on the basis of prior information such as data contained in medical records; the longitudinal prospective approach requires a set of measures completed at both initial referral and at follow-up, whereas the cross-sectional (catch-up) method involves only measures taken at follow-up.

Refer to Table 1 on the following pages.

Table 1

ADHD: Outcome of Retrospective and Prospective Studies

Study	Methodology	Controls	N ADHD	Age at Follow -up	OUTCOME	
					ADHD Symptoms	Antisocial Behaviour
Hartocollis (1968)	Case study - retrospective & current diagnosis	NoC	15	15 - 25	Persisted	2/3 of formerly MBD subjects showed delinquent behaviour
Quitkin & Klein (1969)	Case study - follow back	Psychiatric C	18	18 - 25	Impulsive & Destructive	Not reported
Maletzky (1974)	Retro	NoC	28	13 - 18	Not reported	Teenage delinquency
Virkkunen & Nuutila (1976)	Follow-back	Reading Retardation C	17	15 - 20	Not reported	ADHD symptoms associated with criminal behaviour
Menkes, Rowe, & Menkes (1967)	Follow-back	NoC	14	22 - 40	Improved but still persisted	25% to 30% showed antisocial behaviour
Blouin, Bornstein, & Trites (1978)	Retro	Psychiatric C	42	14	Persisted	No difference between ADHD and controls
White, Barratt, & Adams (1979)	Retro	Normal & Psychiatric C	12	13 - 16	Persisted	Not reported

Table 1 - continued

ADHD: Outcome of Retrospective and Prospective Studies

Study	Methodology	Controls	N ADHD	Age at Follow -up	OUTCOME	
					ADHD Symptoms	Antisocial Behaviour
Morrison (1979)	Retro	Psychiatric C	48	28 - 30	Not reported	Greater incidence of sociopathy than psychiatric controls
Morrison (1980)	Retro	Psychiatric C	48	28 - 30	Not reported	Antisocial behaviour seen in 3 x ADHD subjects than in controls
Offord, Sullivan, Allen, & Abrams (1979)	Retro	Non-ADHD delinquents	31	11 - 14	Not reported	More antisocial symptoms, earlier onset, and poorer prognosis in ADHD group
Shelley & Riester (1972)	Retro	NoC	16	18 - 23	Decrease in core symptoms	Not reported
Laufer (1971)	Cross-sec	NoC	66	15 - 26	Improved but still persisted	25% to 30% showed antisocial behaviour
Mendelson, Johnson, & Stewart (1971)	Cross-sec	NoC	83	12 - 16	Improved	59% contact with police; 18% seen in court - in ADHD group

Table 1 - continued

ADHD: Outcome of Retrospective and Prospective Studies

Study	Methodology	Controls	N ADHD	Age at Follow -up	OUTCOME	
					ADHD Symptoms	Antisocial Behaviour
Minde, Lewin, Weiss, Lavigueur, Douglas, & Sykes (1971)	Prospective	Super NC	37	12	Improved	ADHD group worse than controls
Minde, Weiss, & Mendelson (1972)	Prospective	Normal C	91	13	Improved	ADHD group worse than controls
Weiss, Minde Werry, Douglas, & Nemeth (1971)	Longitudinal	Normal C	64	10 - 18	Improved	25% history of antisocial behaviour & 15% referred to courts - in ADHD group
Hechtman, Weiss, Finklestein, Werner, & Benn (1976)	Longitudinal	Normal C	35	17 - 24	Improved but still persisted	37% ADHD subjects had police records compared to 20% of controls; ADHD subjects committed significantly more thefts

Table 1 - continued

ADHD: Outcome of Retrospective and Prospective Studies

Study	Methodology	Controls	N ADHD	Age at Follow -up	OUTCOME	
					ADHD Symptoms	Antisocial Behaviour
Weiss, Hechtman, Perlman, Hopkins, & Wener (1979)	Longitudinal	Super NC	75	17 - 24	Improved but still persisted	Trend for ADHD have more court referrals in past 5 years, but no difference in past year
Hechtman, Weiss, & Perlman (1981)	Cross-sec	Normal C	53	18 - 24	Not reported	Trend for ADHD subjects have more court referrals in past 5 years, but no difference in past year
Hechtman, Weiss, & Perlman (1984)	Cross-sec	Super NC	91	17 - 24	Not reported	Aggression a problem in 55% of ADHD subjects as compared to 31% of controls; no significant differences in number of thefts
Weiss, Hechtman, Milroy, & Perlman (1985)	Longitudinal	Super NC	63	21 - 33	50% sample continuing ADHD	23% ADHD sample diagnosed with APD

Table 1 - continued

ADHD: Outcome of Retrospective and Prospective Studies

Study	Methodology	Controls	N ADHD	Age at Follow -up	OUTCOME	
					ADHD Symptoms	Antisocial Behaviour
Hechtman, & Weiss (1986)	Cross-sec	Super NC	61	25	Not reported	All ADHD subjects who had antisocial behaviour at 15 years follow-up had early and persistent histories of antisocial behaviour; significantly more ADHD subjects received additional diagnosis of APD; subgroup of ADHD subjects with severe negative outcome seen
Cohen, Weiss, & Minde (1972)	Longitudinal	Super NC	20	15	Much improved	Not reported
Milman (1979)	Longitudinal	NoC	73	15 - 23	Persisted	23% of ADHD sample received diagnosis of APD

Table 1 - continued

ADHD: Outcome of Retrospective and Prospective Studies

Study	Methodology	Controls	N ADHD	Age at Follow -up	OUTCOME	
					ADHD Symptoms	Antisocial Behaviour
Loney, Whaley-Klahn, Kosier & Conboy (1981)	Longitudinal	Brothers	22	21	Not reported	40% of ADHD sample met criteria for APD
Huessy, Metoyer, & Townsend (1974)	Cross-sec	Statistics based on general population	74	15	Improved but still persisted	25% to 30% ADHD subjects had antisocial behaviour; ADHD group was 20 times more likely to be institutionalized in facility for CD youths
Stewart, Mendelson, & Johnson (1973)	Prospective	NoC	81	14	Improved but still	25% ADHD sample had antisocial behaviour

Table 1 - continued

ADHD: Outcome of Retrospective and Prospective Studies

Study	Methodology	Controls	N ADHD	Age at Follow -up	OUTCOME	
					ADHD Symptoms	Antisocial Behaviour
Riddle & Rappoport (1976)	Prospective	Normal C	72	10	Persisted	Not reported
Zambelli, Stam, Maintinsky, & Loiselle (1977)	Prospective	Super NC	9	14	Improved but still persisted	25% to 30% had antisocial behaviour
Ackerman, Dykman, & Peters (1977)	Longitudinal	Normal C & Learning Disabled C	23	14	Persisted	50% ADHD group had conflict with the law; no other groups showed such problems
Hoy, Weiss, Minde, & Cohen (1978)	Prospective	Normal C	15	15	Improved but still persisted	High incidence in ADHD group
Satterfield, Hoppe & Schell (1982)	Longitudinal	Normal C	102	14 - 21	Group on stimulant medication for longer period was more improved	ADHD subjects had more multiple arrests than controls

Table 1 - continued

ADHD: Outcome of Retrospective and Prospective Studies

Study	Methodology	Controls	N ADHD	Age at Follow -up	OUTCOME	
					ADHD Symptoms	Antisocial Behaviour
Reznick & Freeman (1985)	Cross-sec & follow-back	Non-ADHD delinquents	24	12 - 18	Persisted	Among the ADHD group, rate of offenses was 3 times higher than controls
Garfinkel & Klee (1985)	Cross-sec	Normal C	54	17	Persisted & asymptomatic groups; 37% index group diagnosed with RADD	RADD group elevated on delinquency scale
Gittelman, Mannuzza, Shenker, & Bonagura, (1985)	Longitudinal	Normal C	100	18	ADHD persisted in 31% of probands vs. 3% of controls; RADD seen in only 5% of probands	20% increased prevalence of diagnosed CD in ADHD probands vs. controls; those with continuing ADHD were significantly more likely to have CD relative to their asymptomatic counterparts

Table 1 - continued

ADHD: Outcome of Retrospective and Prospective Studies

Study	Methodology	Controls	N ADHD	Age at Follow -up	OUTCOME	
					ADHD Symptoms	Antisocial Behaviour
Mannuzza, Gittelman -Klein, Bonagura, Horowitz -Konig, & Shenker (1988)	Longitudinal	Normal C	52	16 - 23	Group that had <u>not</u> received ADHD diagnosis at follow-up showed more symptoms of inattention & hyper-activity but were generally indistin-guishable from controls	Not reported
Mannuzza, Gittelman -Klein, Horowitz -Konig, & Giampino (1989)	Cross-sec	Normal C	103	16 - 23	Not reported	Significantly more probands than controls had been arrested and incarcerated

Table 1 - continued

ADHD: Outcome of Retrospective and Prospective Studies

Study	Methodology	Controls	N ADHD	Age at Follow -up	OUTCOME	
					ADHD Symptoms	Antisocial Behaviour
Gillberg & Gillberg (1988)	Longitudinal	Normal C	18	NA	Subgroup showed continuing ADHD, while a second subgroup showed situ-ational ADHD	25% of index group had CD at follow-up, however, this was not significantly different from controls
Lambert, Hartsough, Sassone, & Sandoval (1987)	Longitudinal	Normal C	59	14	20% of index group were asymp-tomatic; 37% diagnosed with RADD; 43% showed persistent ADHD	significantly more ADHD subjects vs. controls had antisocial behaviour
Lambert (1988)	Longitudinal	Normal C	166	17 - 18	Not reported	significantly greater incidence of CD in ADHD subjects relative to controls

Table 1 - continued

ADHD: Outcome of Retrospective and Prospective Studies

Study	Methodology	Controls	N ADHD	Age at Follow -up	OUTCOME	
					ADHD Symptoms	Antisocial Behaviour
Fischer, Barkley, Edelbrock, & Smallish (1990)	Longitudinal	Super NC	123	12 - 20	Persisted	Not reported

Note. Retro = retrospective
NoC = no controls
RADD = Residual ADD
APD = Antisocial Personality Disorder
Normal C = normal controls
Psychiatric C = psychiatric controls
Super NC = supernormal controls,
(i.e., screened for the exclusion of any behavioural or cognitive disturbance)
NA = not available

A review of the extant outcome data indicate that the course of the ADHD syndrome is variable. Although a substantial number of ADHD children are asymptomatic by adolescence (Garfinkel & Klee, 1985; Gittelman et al., 1985; Lambert et al., 1987; Weiss, 1985), core ADHD symptoms continue in anywhere from 30% to 80% of samples. For example, Gittelman and colleagues (Gittelman et al., 1985) compared 100 male adolescents aged 16 to 23, who had received a diagnosis of ADHD in childhood, to 100 normal controls, and found that the DSM-III symptoms of ADDH were present in 31% of the index sample and in 3% of the controls.

Similarly, in a large-scale epidemiological study conducted by Lambert (Lambert et al., 1987) of nonclinic-referred ADHD boys and asymptomatic matched controls who were followed into adolescence, 20% of the ADHD group were identified at follow-up as asymptomatic by family, teacher, and physician. An additional 37% of the ADHD cohort met criteria for Residual State (RADD) at follow-up, while 43% continued to evidence the full syndrome and were receiving relevant pharmacological treatment. Additional analyses by the Lambert group, examining early and concurrent correlates of the three differential courses of the disorder, indicated that the RADD group generally showed deficits only in the area of cognitive functioning, whereas both behavioural (including aggression) and cognitive impairment characterized the group with continuing ADHD. These findings gain importance in view of other data (Garfinkel & Klee, 1985; Gittelman et al., 1985) indicating that it is the subgroup of children who continue to manifest core ADHD symptomatology in adolescence that are more likely to have concurrent CD.

With remarkable consistency, the outcome data further indicate the presence of conduct problems and delinquency in a substantial number of ADHD probands assessed in adolescence and early adulthood (e.g. Laufer, 1971; Lambert, 1988; Offord et al., 1979; Mendelson et al., 1971; Morrison, 1979; Reznick & Freeman, 1985, Weiss et al., 1971). The

weight of this evidence prompted Cantwell, in his comprehensive reviews of the outcome literature (1975, 1978, 1981), to conclude that anywhere from 10% to 50% of ADHD adolescents exhibit serious persistent delinquent behaviour, with CD and APD frequently accompanying diagnoses of ADHD in adolescence and early adulthood.

One study that shows a clear and strong association between childhood ADHD and concurrent CD in adolescence is that of the Satterfield group (Satterfield et al., 1982) which controlled for differences in SES, used official arrest records (as opposed to self-report) as an index of antisocial outcome, and had a low attrition rate. Striking differences between the offender rates of the 110 ADHD adolescents and the group of 88 normal controls at follow-up assessment, at 14 to 21 years of age, were observed. The ADHD cohort, examined at 8 to 10 year follow-up, had significantly greater rates of single and multiple serious offences and 19 times the correctional institutionalization rate for delinquency than controls. Moreover, the frequency of offences was found to be evenly distributed across low, middle and upper-middle social strata, suggesting that the observed differences in adjudicated antisocial behaviour were not confounded by differences in SES as is common in studies of criminal behaviour. The Satterfield data further underscored the importance of examining the influence of attrition rate in determining outcome; they observed a two-fold increase at follow-up in the arrest rate for serious offences for the nonparticipating as compared to the participating subjects. This finding, that the group lost to follow-up constitutes a more negative outcome group, is supported by other research (Cox, Rutter, Yule, & Quinlan, 1977; Loney et al., 1983). Therefore, it is likely that subject sample bias plays a role in accounting for the comparably lower rates of antisocial outcome occasionally reported by other authors (e.g., the Hechtman, Weiss, & Perlman (1981) study had an attrition rate of 28% and reported a relatively low rate of delinquent conduct in ADHD probands compared to matched normal controls).

The finding of concurrent CD in a subset of ADHD adolescents is further supported by the work of Gittelman and colleagues (Gittelman et al., 1985; Mannuzza et al., 1988, 1989). At 10-year follow-up, the DSM-III diagnoses of continuing ADDH and co-existing CD were present in 31% and 27%, respectively, of the original ADDH cohort of 100 males, as compared to 3% and 8% of the group of 100 male nonpsychiatric controls (Gittelman et al., 1985). Subsequent research (Mannuzza et al., 1989), using official arrest records as a complementary measure of antisocial outcome in late adolescence and early adulthood, reported substantial comorbidity between ADDH and CD at follow-up. Half of the probands with continuing ADDH (versus 17% without ADDH) had concurrent CD. The persistence of ADDH symptoms was found to be a major concomitant of later CD, and the presence of concurrent CD at follow-up almost completely accounted for the increased risk in adjudicated criminal activities, whether or not the conduct problems were also accompanied by a substance use disorder. Moreover, the possibility of a childhood CD in a portion (36%) of the initial ADDH cohort did not account for the strong relationship between arrest history and CD at follow-up -- no significant differences in arrest rate were observed between the group of ADDH probands with early as compared to late onset of associated CD.

In conclusion, the current literature addressing the outcome of ADHD in adolescence identifies a number of developmental pathways that are consistent with the two of the three models of outcome outlined earlier, the continual display and the developmental decay models (Cantwell, 1985a), but provide weaker evidence for the developmental delay view. Generally, a subsample of ADHD children are found to be asymptomatic in adolescence. However, this subsample continues to evidence poorer classroom behaviour and academic performance relative to normal controls (Gittelman et al., 1985), with a further subgroup meeting diagnostic criteria for other psychiatric illness, including schizophreniform disorder, substance abuse, or major affective disorder (Garfinkel & Klee, 1985). The overall course of the disorder includes a subgroup that remains chronically impaired in academic

achievement, inattention, and behavioural disinhibition (the RADD group), while a final subgroup shows continuing ADHD with additional psychopathology, typically that of CD and/or substance use disorder.

The core ADHD symptoms that persist into adolescence are associated with impairment in psychosocial functioning and may be "masked" by secondary problems of antisocial behaviour, truancy, school underachievement, and poor social competence. Moreover, the presence of only one cardinal symptom in adolescence does not appear to differentiate ADHD probands from same-age normal controls, suggesting that it is the syndromal quality of the disorder, rather than any one symptom in isolation, that leads to psychosocial impairment (Gittelman Klein et al., 1985). Of particular interest is the indication that there exists a significant overlap between the three major outcomes of ADHD, in that associated CD at follow-up is seen only in the subgroup who continue to display primary ADHD symptomatology. Thus, the picture appears to be one of continuing ADHD which is associated with an outcome of mixed ADHD/CD in adolescence. This suggests that it is the persistence of ADHD, rather than the mere presence of ADHD in childhood, that is a risk factor in the development of associated CD.

A critical perspective on the matter of ADHD as it relates to antisocial outcome in adolescence requires, however, a consideration of the various methodological shortcomings of the outcome literature, with particular attention to the issue of subject selection. The available outcome research have been aptly criticized (Brown & Borden, 1986; Cantwell, 1985a; Thorley, 1984) for investigators' reliance on normal and "supernormal" (Thorley, 1984) control groups, variability in subject selection criteria, lack of control for medication status, and significant attrition of original patient samples. The problem of variation in subject selection has been most pervasive, however, and is pivotal to the issue of persistent ADHD as it relates to CD in adolescence. Follow-up investigations, particularly those conducted prior to 1980, have largely failed to obtain information on concurrent degree of

aggression and/or conduct disturbance in original samples; it is, therefore, unclear whether antisocial disturbance at follow-up was an outgrowth of ADHD or an artifact resulting from an early, undiagnosed CD. Loney and Milich's (1982) widely quoted criticism (e.g., Hinshaw, 1987) continues to reflect the thinking in this area:

> We do not have a literature about childhood hyperactivity as such; instead, we have a literature about childhood externalizing behaviour problems (hyperactivity and aggression) that we call a literature about childhood hyperactivity. (Loney & Milich, 1982, p. 143)

It is clear that outcome and validation research on the ADHD syndrome require distinct subgroups at the outset. The crucial data concerning the associative links between ADHD and CD have yet to be collected and it is unclear whether the severity of ADHD per se is linked to later CD, or if, instead, the association is between childhood forms and adolescent manifestations of co-existing CD. More recent investigations that have examined the predictive utility of mixed ADHD/CD versus pure ADHD subgrouping have provided conflicting results. For example, August, Stewart, and Holmes (1983) evaluated adolescent outcome separately for groups of pure ADHD and mixed ADHD/undersocialized aggressive males, and found that only probands (37%) within the mixed subgroup met diagnostic criteria for concurrent CD at follow-up. Similarly, the series of investigations by the Loney group, examining the predictive utility of primary ADHD and secondary aggressive symptomatology, found that early aggression and parenting style, not childhood hyperactivity, were associated with aggression and delinquent outcome in adolescence (Loney et al., 1981; Paternite & Loney, 1980). However, many of the ADHD cases in the Loney sample were likely contaminated with CD (Quay, 1985) and the design lacked a necessary non-ADHD comparison group (Thorley, 1984). Certainly, other studies (Gittelman Klein, 1985; Mannuzza et al., 1989; Reznick & Freeman, 1985; Schachar et al., 1981) have identified an association between antisocial outcome in ADHD samples and core ADHD symptomatology, particularly impulsivity, rather than with conduct symptoms per se.

Whether or not early conduct symptomatology proves to be a better predictor of adolescent antisocial outcome, the picture at present does not preclude the possibility of an additional subgroup of children, who show pervasive and persistent ADHD, developing CD in later life, even in the absence of significant conduct symptomatology in childhood. The current data suggest that the catalytic link in this latter group may be parenting style (Loney et al., 1983) and/or psychosocial adversity (Szatamari et al., 1989). Moreover, as Loeber (1990) has pointed out in his discussion of the genesis of conduct disorders, many risk factors, by themselves, may increase the likelihood of later antisocial conduct. However, it is common, and more likely, that the more potent effects occur when these risk factors present in conjunction with other particular risk factors. (The reader is referred to Rutter (1978) for examples of studies showing such cumulative effects). Investigators have largely emphasized the singular role of hyperactivity, the ADHD syndrome (Cantwell, 1975, 1978), or associated CD and aggression (Loney et al., 1982) in effecting antisocial outcome, yet it may well be the interaction between certain risk factors, over and above the contribution of individual risk factors, that acts to substantially magnify risk for antisocial behaviour. The current data point to the conclusion that the various risk variables associated with poor outcome are the persistence of ADHD, associated CD and/or aggression, parenting style, and social disadvantage.

As a result of outcome data indicating substantial comorbidity of ADHD with CD, and the associated problem of contaminated ADHD samples, researchers investigating ADHD have highlighted the need to "take particular care to select subjects in order to eliminate, or control, the interrelationships of Conduct Disorder, Socialized Aggression, [and] Attention Deficit Disorder..." (Quay, 1985, p. 39). An additional impetus for systematic evaluation of concurrent CD in ADHD samples is the least favourable long-term outcome of this mixed group relative to that of either of the diagnoses alone (Offord et al., 1979; McGee et al., 1984, 1985; Moffit, 1990; Reznick & Freeman, 1985). A dual diagnosis

of ADHD and CD reflects the severity and negative characteristics of each disorder. This dual developmental "stacking" of problem behaviours (Loeber, 1990) may act to further complicate adjustment in adolescence, thereby magnifying the degree of psychosocial impairment and antisocial conduct. Given the comparatively poor prognosis for this subgroup, questions about treatment options have come to the fore.

A clear understanding of the nature of the core deficits of ADHD, and the manner in which they have yielded to empirical investigation, is essential for the planning and assessment of treatment programmes. Indeed, treatment planning and selection of criteria for improvement are profoundly influenced by current views regarding assessment and measurement of the cognitive and behavioural deficits that constitute ADHD (Douglas, 1980a). Thus, a review of theoretical and empirical inquiries into the nature of the core features of the disorder is warranted at this juncture, and is meant to provide a framework for the use of particular treatment outcome measures in the current study and subsequent discussion of a selected treatment consideration for the mixed ADHD/CD subtype.

Chapter 3
ADHD And The Empirical Study Of Its Core Features

In contrast to diagnostic, clinical, and outcome models, empirical research on ADHD has also been conducted from a heuristic perspective which has "viewed ADHD as the interaction term in a child-by-task or child-by-challenge matrix" (Henker & Whalen, 1989, p. 217). The past two decades have seen a proliferation of studies that have examined the performance deficits of ADHD children from the child-by-task perspective, and have suggested that the particular deficits involve defective attentional, inhibitory, arousal, and reinforcement mechanisms (Conners, 1975; Douglas, 1972, 1980a, 1980b, 1983; Douglas & Peters, 1979; Rosenthal & Allen, 1978; Swanson & Kinsbourne, 1979). Research has repeatedly pointed to a strong relationship among the deficits. There has been considerable confusion in the literature with respect to how the terms are used, and authors have found it difficult, even conceptually, to separate them (Douglas, 1983, 1985, 1989).

Probably the best articulated and substantiated theory of ADHD is that of Douglas (1972, 1980a, 1980b, 1983, 1984, 1985, 1989), whose model was so sweeping in its effects that it served as a major impetus for the change in terminology from Hyperkinetic Reaction of Childhood to ADD in the revisions of DSM-II and DSM-III. A recent updating of Douglas' model (Douglas, 1983, 1985, 1989) has brought together a number of the observed performance deficits of ADHD children by proposing a generalized defect in self-regulatory or executive control. Douglas continues to maintain that attention, inhibition, arousal, and reinforcement abnormalities represent the major aspects of defective functioning in ADHD children. However, she now conceptualizes all four as interdependent manifestations of impaired self-regulation that are secondary to a broader, underlying self-regulatory defect.

Douglas has extensively reviewed (1980a, 1980b, 1983) investigations of a wide variety of cognitive and behavioural tasks on which ADHD children perform poorly relative

to normal (i.e., nonreferred) and clinic-referred children. Differences between samples of index and control subjects have been obtained on relatively simple tasks that measure attention or vigilance, on complex cognitive tasks requiring perceptual and logical search strategies, on measures that involve motor control, and on tasks that demand organized, reflective effort, and effective problem-solving (Aman, 1978; Barkley, 1977; Douglas, 1972; Douglas & Peters, 1979; Messer, 1976; Ross & Ross, 1976; Whalen & Henker, 1976). The following will entail a brief overview of research on ADHD from the child-by-task perspective, with particular attention devoted to tasks that are of special relevance to the present study, namely, those in the areas of attention, impulsivity, and reinforcement learning. I begin with an emphasis on "attentional" measures.

Measures of Attentional Deficits

Although the DSM-III and DSM-III-R descriptions of the disorder represented a significant improvement over that of their predecessor, DSM-II, the definition of attentional deficit continues to be ambiguous. Attention, in the recent nomenclatures, is treated largely as a single trait or unitary characteristic that is evidenced by a wide array of behaviours (i.e., "careless errors", "not staying with tasks", "omissions and inappropriate insertions in work"). This considerable breadth of description has paralleled definitions of inattention in mainstream scientific psychology and psychiatry, hence, a multiplicity of labels and tasks have been used with disparate meanings. In fact, the reader will find that serious methodological and conceptual problems in the area of impulsivity plague the empirical literature as well.

The task of investigating attentional difficulties in the ADHD population is complicated by the fact that attention is not a unitary dimension, nor is inattention a static deficit. Attention is a diffuse and wide ranging psychological (and physiological) construct involving various facets which include, at the very least, search, set, selective attention,

concentration, activation, and vigilance (Prior & Sanson, 1986). An attention deficit may be exhibited in a variety of cognitive areas, including the inability or failure to sustain attention over an extended period of time (vigilance), to focus on relevant external stimuli while ignoring distractions (selectivity), and to engage in an organized search for critical cues in the environment (selective attention) (Douglas, 1980a). Children and adolescents with ADHD may demonstrate deficits in various combinations of the basic functions that comprise attention (Shaywitz & Shaywitz, 1984). Additionally, the quality of attention may fluctuate in relation to both dimensions of the task at hand and variables present in the environment.

Much of the early work examining attention deficits in ADHD children involved the use of vigilance and reaction-time tasks. Although a variety of vigilance tasks have been used, the basic paradigm has been one of selective attention for an infrequently occurring stimulus under monotonous conditions. Here, a series of relatively simple stimuli (either auditory or visual) are presented repeatedly over an extended period of time to a subject who is required to respond to certain target stimuli and to refrain from responding to nontarget stimuli. In view of the extended and constant nature of the demands made on subjects, these tasks were first described (Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956), and continue to be referred to, as "continuous performance tests" (CPTs). The number of correct responses on the CPT has been used as an index of attentiveness, whereas omission errors (failure to respond to target stimuli) are considered to reflect inattentiveness, and commission errors (responses to nontarget stimuli) to reflect impulsivity (Sostek, Buchsbaum, & Rapoport, 1980).

The vigilance performance of ADHD children has generally been found to be inferior to that of normal and clinic-referred children (Anderson, Halcomb, & Doyle, 1973; Chee, Logan, Schachar, Lindsay, & Wachsmuth, 1989; Doyle, Anderson, & Halcomb, 1976; Dykman et al., 1971; Kaspar, Millichap, Backus, Child, & Schulman, 1971; Sykes, Douglas,

& Morgenstern, 1973; Sykes, Douglas, Weiss, & Minde, 1971). Findings have included fewer correct detections (i.e., omission errors) and more incorrect responses (i.e., commission errors) in index children, suggesting difficulties with the maintenance of attention over time and with inhibitory control (Douglas, 1983; Douglas & Peters, 1979). Similar deficits on CPT tasks are also evident among ADHD adolescents (Cohen et al., 1972; Garfinkel, Brown, Klee, Braden, Beauchesne, & Shapiro, 1986; Klorman, Salzman, & Borgstedt, 1987; Loiselle, Stamm, Maitinsky, & Whipple, 1980; Sykes et al, 1973). Follow-up studies have reported differences between hyperactive adolescents and matched controls in errors of commission (Fischer et al., 1990; Hoy et al., 1978) and omission (Fischer et al., 1990). This similarity of performance on CPT tasks among pre- and postpubertal children supports follow-up reports of a continuity of attentional and inhibition deficits into the adolescent age range.

Although studies indicate that CPT performance is related to behaviours and cognitive deficits that are associated with ADHD, poor CPT performance is not unique to ADHD (Klee & Garfinkel, 1983; Quay, 1986; Schachar, Logan, Wachsmuth, & Chajczyk, 1988), nor does it define the ADHD population (Trommer, Hoeppner, Lorber, & Armstrong, 1988). For example, CPT scores have been found to significantly correlate with other psychometric measures of inattention, with behavioural ratings of inattention and impulsivity (Klee & Garfinkel, 1983), and with measures of abstract reasoning and problem-solving (Trommer et al., 1988). However, impaired CPT performance does not characterize all ADHD children. Recent work by Trommer and her associates (Trommer, Lorber, & Armstrong, 1987; Trommer et al., 1988), examining the diagnostic validity of CPT performance in children who met the DSM-III diagnostic criteria for ADDH, indicated that performance on the CPT may yield both false negative and false positive results. Therefore, accurate performance on the CPT does not rule out ADHD, nor is poor performance specific to ADHD.

Of relevance to the current study, and its focus on the mixed ADHD/CD subtype, are findings of extreme responding on the CPT in conduct disordered samples relative to normal and behaviour disordered controls (Klee & Garfinkel, 1983; Orris, 1969; Quay, 1986; Raine & Jones, 1987; Schachar et al., 1988). This evidence suggests that subgroups of both the ADHD and CD categories may show overlapping deficits in sustained attention as measured by the CPT. However, it is possible that the attentional deficits are a correlate of only one of these disorders, more likely of ADHD. The apparent association of inattention with CD may be due to a failure to disentangle co-occurring ADHD in studies of conduct disordered samples. At present, investigation of the severity of attention deficit, as measured by the CPT, has not been researched in ADHD children who show concurrent CD.

Simple reaction-time tasks, including reaction-time tasks involving delay schedules (DRT tasks), have also been used to measure attention in ADHD children. Here, stimuli are delivered at either random or constant intervals, and the subject is required to respond as quickly as possible. The DRT task includes the addition of a warning signal, followed by a waiting period, and a signal to indicate the termination of the waiting period. Similar to the CPT, reaction-time tasks are experimenter-paced, or instrument-paced, and involve prolonged monitoring and the withholding of inappropriate responses on the part of the subject over a period of several to 30 minutes. Measures have focused on the speed of response to simple task stimuli, based on the assumption that speed or reaction to a signal stimulus is related to the alertness component of attention (Samuels & Edwall, 1981).

Reaction-time task studies have generally indicated that ADHD subjects, relative to normal and clinical controls, are generally slower in the mean latency of response, show greater intraindividual variability of reaction time, and make a greater number of inappropriate responses, such as responding to the warning signal and pushing more than once in response to the warning signal (Cohen & Douglas, 1972; Douglas & Parry, 1983;

Firestone & Douglas, 1975; Grunewald-Zuberbier, Grunewald, & Rasche, 1975; Porges, Walter, Korb, & Sprague, 1975; Spring, Greenberg, Scott, & Hopwood, 1973). These findings suggest that a significant proportion of ADHD children fail to respond effectively and consistently to target stimuli, and fail to withhold inappropriate responses during a repetitive and prolonged task (Barkley, 1977; Douglas, 1972; Douglas & Peters, 1979; Ross & Ross, 1976).

Investigators have also examined the effects of task-irrelevant stimuli (distractors) on tasks of selective attention and incidental learning. The predominant hypothesis under study has been that ADHD children, relative to controls, are impaired in the ability to filter out extraneous stimuli. Other assumptions, such as an impaired ability to discriminate between central and irrelevant stimuli, to perform concurrent analyses on stimuli (Ross, 1976), or to inhibit strong response tendencies (Douglas, 1983), have also been proposed.

Laboratory findings in this area have been equivocal. In an earlier review of the literature, Douglas and Peters (1979) concluded that there is little empirical evidence to support the distractibility hypothesis. They reviewed 11 studies employing a wide range of tasks and task-irrelevant stimuli that were either extrinsic (Sykes et al., 1971) or intrinsic (Davidson & Prior, 1978; Douglas & Peters, 1979) to the task, and found that, when interference with task performance was observed, it was no greater in ADHD subjects than in normal controls. They further argued that the few existing incidental learning studies did not provide evidence that ADHD children are more likely to process and recall task irrelevant stimuli, although there have been some recent exceptions (e.g., Copeland & Wisniewski, 1981). Similarly, Prior and Sanson (1986), in their critique of the literature on ADHD, asserted that "distractibility studies have generally failed to demonstrate that either extra-task or intra-task distractors have a differential effect on hyperactives and controls" (p. 309).

However, other authors (e.g., Rosenthal & Allen, 1978) have criticized research on distractibility for a general reliance on the use of distractors that are external to the central task, arguing that distractibility is more correctly defined in terms of the child's ability to respond to relevant stimuli, while inhibiting responding to irrelevant stimuli that are intrinsic to the task. Certainly, impaired performance in ADHD subjects relative to controls has been reported when irrelevant dimensions were presented within the stimulus array (Fischer et al., 1990; Rosenthal & Allen, 1980). Similarly, Taylor (1988) has disputed the inclusion of studies using dichotic listening tasks (e.g. Davidson & Prior, 1978; Loiselle et al., 1980) in previous reviews of research on selective attention in ADHD samples (Douglas, 1983; Prior & Sanson, 1986), contending that

> the attentional requirements of dichotic listening differ from both sustained and selective attention in that in dichotic listening tasks the stimuli are presented in brief, discrete trials and there are no irrelevant (distracting) stimuli presented to the channel being shadowed. Consequently, measures of dichotic listening do not constitute adequate indices of sustained or selective attention and so fail to adequately address the issues at hand. (Taylor, 1988, p. 218)

Moreover, more recent data indicate a number of conditions under which high distractibility is observed in ADHD child samples (Denton & McIntyre, 1978; Radosh & Gittelman, 1981; Rosenthal & Allen, 1980). Prior and Sanson (1986) have argued that the critical variable is increasing complexity, however, selective attention deficits may well be exacerbated by an increase in distractor salience (Radosh & Gittelman, 1981; Rosenthal & Allen, 1980). For example, Rosenthal and Allen (1980) confirmed earlier observations (Sykes et al., 1971) that relatively weak distractors do not impair the performance of ADHD children when compared to controls. However, when the effects of salient and meaningful distracting stimuli were considered, more serious impairment was observed in the ADHD group relative to controls.

In a more recent review of the available literature on selective attention in child ADHD samples, Douglas (1983) concluded that distractors do, indeed, produce differential

effects on ADHD children when particular stimulus conditions are taken into consideration. These include the

> degree of boredom, distaste, or difficulty associated with a particular task; the salience or novelty of potential distractors; [and] the disinclination of hyperactive children to process beyond the more obvious or salient aspects of a task... (Douglas, 1983, p. 296)

Again, as with impairment in attentional capacity as measured by the CPT, distractibility to stimuli that are embedded within the stimulus array has yet to be investigated in mixed ADHD/CD groups.

The finding that eliciting a selective attention deficit in ADHD children is contingent on the presence of specific task parameters converges with the broader issue of the situation-specificity of performance deficits in ADHD samples. As mentioned earlier, inattention is not a static deficit and may fluctuate in response to variation in task dimensions and situational variables. However, it is significant that the quality of attention in ADHD children has been found to fluctuate in relation to experimental variables that have less, or no, effect on the attention of normal children. These experimental manipulations have generally involved the schedule of reinforcement (Douglas & Parry, 1983; Firestone & Douglas, 1975; Parry & Douglas, 1983), the arousal level of the child (Sykes et al., 1971), and the speed and pacing exhibited by the child in completing the tasks (Milich et al., 1982; Prior, Sanson, Freethy, & Geffen, 1985; Sykes, Douglas, & Morgenstern, 1972).

This area of findings has fueled a controversy (e.g., Prior & Sanson, 1986; Taylor, 1988) over whether the performance deficits observed in ADHD samples may be due to an application deficit (Prior & Sanson, 1986), resulting from low motivation to comply with environmental demands. However, careful analysis of the features of experimental manipulations that have demonstrated effectiveness in promoting good attentional behaviour reveals that such manipulations likely serve to combat, or compensate for, the

effects of the child's attentional, inhibitory, and arousal problems (Douglas, 1983; Douglas & Peters, 1979). For example, performance in ADHD children has been shown to improve when the experimenter provides continuous reinforcement for each response (Douglas & Parry, 1983; Parry & Douglas, 1983), suggesting that performance is enhanced when an external aid directs attention to the occurrence of correct responses, thereby facilitating the deployment of attentional strategies. Similarly, improved performance has been observed in a Choice Reaction-Time task when the child's attention is captured before each trial (Sykes et al., 1971), indicating that performance is best when cues serving to direct attention to the task are externally generated. Researchers have begun to emphasize the "production deficiency" (Douglas, 1983) or "production deficit" (Kinsbourne, 1989) component of ADHD, using a term borrowed from developmental psychology (Flavell, 1970) to describe situations when a child can be induced to use a mediator that was not produced spontaneously. Hence, as Kinsbourne (1989) has aptly stated, ADHD is "ill named, as all ADD individuals are capable of focused and sustained attention under certain, though sometimes very restricted, circumstances" (p. 114). It is clear that the boundaries of effective attention in ADHD are limited to tasks that are novel, self-determined, interesting, and mildy stressful. Many of these same factors also seem to determine whether or not "impulsivity" is found.

Measures of Impulsivity

The operational criteria for impulsivity in the most recent diagnostic classification systems (DSM-III and DSM-III-R) reflect a variety of cognitive and behavioural difficulties, including overlap with other core symptoms of the syndrome, such as excessive, undirected activity and poor attentional abilities (e.g., "often acts before thinking", "shifts excessively from one activity to another", "has difficulty organizing work, and frequently calls out in class"). The dimension of impulsivity is clearly not a unitary one, as reflected in the various manifestations of impulsive behaviour that have been emphasized in theoretical

accounts of ADHD children. These have included unresponsiveness to environmental constraints (Conners, 1969; Renshaw, 1974), inability to delay gratification (Denhoff, 1973; Sandoval, Lambert, & Yandell, 1976; Whalen & Henker, 1976), poor resistance to temptation (Douglas, 1972), failure to evaluate all aspects of a situation (Campbell, Schleifer, Weiss, & Perlman, 1971), and risk taking or accident-proneness (Mannheimer & Mellinger, 1967; Stewart et al., 1970).

Similarly, as with its attentional counterpart, empirical study of the core feature of impulsivity has been hampered by the use of a multiplicity of labels designating different aspects of impulsive behaviour. As Douglas (1983, 1989) and Ross (1976) have pointed out, terms such as "impulsivity", "inhibitory control", and "disinhibition" have been used with confounding meanings. In her more recent writing, Douglas (1989) has addressed this confusion in descriptive labels and has attempted to clarify her position in keeping with her model of self-regulation. She writes:

> Although I still use the term "impulsivity" occasionally, I agree that it has created unnecessary confusion....I have preferred the terms "inhibitory control" or "withholding of inappropriate responses. I intend these concepts to encompass the notion of a failure to withhold responding until sufficient information has been gathered....I see processes involving the withholding of inappropriate responses as representing inhibitory, as opposed to facilitatory, aspects of self-regulation. (p. 238)

As with attentional deficits, impulsive tendencies in child ADHD samples have been observed on a range of both simple and complex cognitive tasks, including the inability or failure to withhold responding until target stimuli appear, the tendency to respond repeatedly to a single target stimulus, the inclination to act before a conceptual problem is clearly understood, and the proclivity to respond before consideration of all possible response alternatives.

The most extensively studied of the various aspects of impulsivity in the ADHD population has been the dimension of cognitive tempo. Rapid responding with little critical evaluation of alternatives has been considered to be an essential component of the impulsive

cognitive style (Campbell, Douglas, & Morgenstern, 1971; Kagan, Rosman, Day, Albert, Phillips, 1964). Kagan and his associates have introduced (Kagan et al., 1964) and studied (Kagan, 1965a, 1965b, 1966; Kagan, Moss, & Sigel, 1963; Kagan, Pearson, & Welch, 1966) a construct of cognitive style labelled "reflection-impulsivity", which is measured by Kagan's Matching Familiar Figures Test (MFFT) (Kagan et al., 1964). The MFFT is a perceptual search task that involves scanning of a visual display while seeking to find critical attributes of a stimulus. The central requirement is concentration in finding the exact match to a target picture.

Studies using the MFFT have uniformly reported more impulsive responding (shorter response latencies and increased errors) in several age groups of ADHD children relative to normal peers, including preschool, elementary-school, and high-school samples (Aman, 1978; Campbell et al., 1971; Cohen et al., 1972; Douglas & Peters, 1979; Juliano, 1974; Messer, 1976; Sandoval, 1977; Schleifer et al., 1975). These findings are consistent with the previously discussed high rate of false positive responding (commission errors) noted on vigilance tasks (Sykes et al., 1971, 1973) and delayed reaction-time tasks (Cohen, 1970; Cohen, Douglas, & Morganstern, 1972; Firestone & Douglas, 1975; Parry & Douglas, 1983) in ADHD samples.

Latency of response and error scores on the MFFT have likely been the most commonly used indices of impulsivity in ADHD children. However, largely because of the close association drawn by Kagan and his associates between reflection-impulsivity and conceptual tempo, many investigators have equated response times with impulsivity. This tendency to equate impulsivity with rapid responding has been criticized by Douglas (Douglas, 1983, 1989) for a failure to consider additional factors, not directly related to impulsivity, that may influence latency of responding on the MFFT, such as processing efficiency and off-task behaviours (Tant & Douglas, 1982; Douglas, Barr, O'Neill, & Britton, 1986). This criticism, in part, provided the impetus for Douglas' more recent

emphasis (1989) on the failure to "withhold responding until sufficient information has been gathered" as the defining feature of impulsivity. Douglas' conceptualization of impulsivity is further supported by signal detection analyses of performance on vigilance tasks. Results have indicated that ADHD children have a lower response criterion (i.e., require less certainty that a stimulus is a target stimulus before responding to it as if it is a target) (O'Dougherty, Neuchterlein, & Drew, 1984). Moreover, the simple use of speed of response as an index of inhibitory control may also be misleading in that studies using reaction time tasks have observed longer response latencies in ADHD samples relative to controls. These findings would suggest that, similar to attentional difficulties, the situational determinants of impulsivity may be the amount of interest in, or degree of difficulty of, task demands (McMahon, 1984).

The impulse control dimension in ADHD has also been addressed from the perspective of the outcome of an action or decision, rather than from the latency and accuracy of response. One outcome, labelled the failure to delay gratification (Mischel, 1958), is defined as the choice of an immediately available, smaller reward over a delayed, but larger reward. What little experimental documentation exists on this feature of impulsivity in child and adolescent ADHD samples is equivocal. Mann (1973) reported a strong relationship between impulsivity, as measured by MFFT scores, and a failure to delay gratification. Similarly, Campbell (1985) observed that parent-referred ADHD youngsters were more impulsive than controls on a delay task. Ward (1973) and Reznick and Freeman (1985), by contrast, failed to observe a preference for immediate, as opposed to delayed, gratification in preschool and youth samples.

One paradigm that shows considerable promise as a useful and direct measure of the ability to inhibit response tendencies and tolerate delay, as discussed, for example, by Douglas (1983, 1985), is the differential reinforcement for low-rate responding (DRL) task. The procedure does not involve responding to correct versus incorrect target stimuli, but

entails a repeated withholding of responses over a series of time intervals and provides for reinforcement of a response correctly emitted after a set time interval (i.e., 6 seconds) has elapsed. Responses that occur before the set time interval has elapsed are not reinforced and, moreover, they serve to reset the timer governing reinforcement.

Gordon and his associates (Gordon, 1979, 1986; McClure & Gordon, 1984) have conducted a series of studies comparing the DRL performance of children classified as ADHD on the basis of the Teacher Rating Scale (Conners, 1969) with matched samples of clinic-referred controls. Gordon has consistently observed highly significant group differences in performance, with children designated as ADHD showing a relative inability or failure to perform efficiently on the task. The most sensitive measure in differentiating the two groups has been the efficiency ratio (ER), (derived by dividing the total number of correct responses by the total number of responses emitted), which has indicated that ADHD children, relative to controls, tend to emit a greater number of responses overall and fail to refrain from emitting a high number of nonrewarded responses. The validity of Gordon's data remains unclear, however, in that diagnostic classification was based exclusively on teacher ratings of behaviour.

In his earlier study, Gordon (1979) also examined the nature of self-generated mediational strategies employed by each subject in his adaptation to the DRL schedule. Behaviours were rated as either covert/cognitive (e.g., counting silently) on the basis of posttest inquiry, or overt/behavioural on the basis of observer ratings. Analyses of subjects' collateral behaviours indicated that the use of more cognitively oriented, as opposed to behavioural, mediational strategies was associated with significantly more efficient adaptation to the DRL schedule. Hence, although the delay task appears to tap most broadly into the area of impulse inhibition, additional processes, such as time estimation ability, motivation, and the capacity to develop an efficient strategy, likely play a role in determining performance.

Interestingly, poor DRL performance has been observed in ADHD groups independent of the nature of reinforcement used in studies. In Gordon's (1979) original study, subjects received candy rewards that were dispensed at the end of the session, whereas in a subsequent study (McClure & Gordon, 1984), there were no tangible reinforcers.

DRL performance has also been found to differentiate between ADHD and emotionally disordered groups independent of age and IQ. This has important methodological and conceptual implications for research on ADHD when considered in light of problems found with other measures of impulsivity, such as performance on the MFFT, which has shown an association with intellectual functioning and developmental status (Carins & Cammock, 1978), as well as with anxiety states (Messer, 1970) and depression (Schwartz, Friedman, Lindsay, & Narrol, 1982). Of particular interest to the present research on the mixed ADHD/CD subtype are findings that impaired performance on both the MFFT (Shaffer, McNamara, & Pincus, 1974) and the DRL task (Shapiro, Quay, Hogan, & Schwartz, 1988) is seen in conduct disordered groups. This is not surprising, in view of the topographical similarity of ADHD and CD with respect to noncompliant, disruptive, and impulsive behaviours. In fact, it has been suggested that poor impulse control (Freeman, 1978; Freeman & Kinsbourne, 1984; Freeman & Reznick, 1984; Loeber, 1990; Reznick & Freeman, 1985), or disinhibition (Gorenstein & Newman, 1980), may be the common denominator between ADHD and antisocial outcome in later life.

For example, Gorenstein and Newman (1980) have contended that ADHD, together with CD, psychopathy, hysteria and alcoholism, belongs to a class of disorders that they labelled "disinhibitory psychopathology". As evidence for their argument, they reviewed a number of experimental paradigms in which similar abnormalities have been found in antisocial disorders and septal-lesioned animals, based on their assumption that the

syndrome produced by lesions in the septal-hippocampal-frontal (SHF) system of animals serves as a "functional research model" of human disinhibitory psychopathology. With respect to the area of performance on DRL tasks, Gorenstein and Newman reported a number of studies where deficits in DRL performance were observed in septal-lesioned animals, and where the SHF animals' deficits were overcome by providing an external stimulus to indicate the interval during which the response was to be withheld. They suggested that this reflects a "loss of a normal ability to mediate temporal intervals" (p. 310), paralleling, they argue, the clinical and experimental findings indicating that ADHD and antisocial adolescents have difficulty withstanding delay and "are less disposed than normals to forego immediate gratification as a means of obtaining a larger reward later on" (p. 310). Douglas (1983) has also noted that the external stimuli used in research on DRL performance in SHF animals may act to decrease the need for inner controls, similar to the findings with ADHD children where the presence of external controls increased task-relevant behaviours. She suggested that it is possible the SHF animals are so highly motivated to obtain the reinforcers that little effort is directed toward mediation or control. A particular attraction to immediate, salient reinforcers and a concomitant failure to show punishment avoidance have been observed in both ADHD and antisocial groups, which leads us into the area of reinforcement learning in ADHD children.

Reinforcement Learning: Reward Dominance and Passive Avoidance

Although neither of the recent nomenclatures addressed the role of reinforcement abnormalities in ADHD, Douglas' (1983, 1985, 1989) proposed model of ADHD extends the triad of core features to include a fourth deficit involving reinforcement mechanisms. Specifically, she argues that the behaviour of ADHD children reflects a particular inclination to seek immediate, salient reinforcement and a failure to consider long-term consequences of their acts. In her theoretical conceptualization, which has had a profound

impact on the field, no one of the four core problems is considered to be more basic or central than any other (Douglas, 1983, 1985).

Evidence for Douglas' hypothesis stems from a series of studies by her research group at McGill University in which ADHD subjects demonstrated distinctive responses to reinforcement contingencies. Research on the performance of hyperactive children on a concept learning task (Freibergs & Douglas, 1969) indicated that their performance differed from normal controls under conditions of partial, but not continuous, reinforcement schedules. In a subsequent study, designed to control for the amount of feedback provided across reinforcement conditions, Parry and Douglas (1983) obtained the same ADHD-control differences in the standard partial reinforcement condition. Drawing on Amsel's (1962) "partial frustration" theory, Douglas (Douglas, 1985; Freibergs & Douglas, 1969) theorized that the occurrence of non-reward during partial reinforcement schedules evokes frustration, causing subjects to abandon hypotheses that fail to result in reinforcement.

Evidence that inconsistent reward, even when intangible in the form of praise or verbal feedback, can impair the performance of ADHD youngsters has also been offered from research using a delayed reaction time task (DRTT) in which performance under three reinforcement conditions, continuous reward, partial reward, and noncontingent partial reward, was contrasted (Douglas & Parry, 1983). No differences in mean reaction time between ADHD children and control conditions were observed on this task under partial and continuous reinforcement schedules. However, the noncontingent partial reward condition had a uniquely negative impact on the response-time performance of the ADHD group. This stood in stark contrast to the performance of the normal sample which actually benefited from the motivating effects of the positive feedback, even though it was delivered randomly. Further, the hyperactive children's reaction times improved when the noncontingent reward was withdrawn during extinction trials. Douglas and Parry (1983) speculated that noncontingent positive feedback may increase arousal or distraction in

ADHD children, without guiding their attention to the specific features of the responses that are required. Overall, this data suggested that ADHD children may be strongly influenced by the withdrawal or withholding of rewards, whether this occurs during extinction trials or on partial reinforcement schedules.

An additional study conducted by Douglas and her colleagues (Firestone & Douglas, 1975) investigated the differential effects of positive feedback, negative feedback, and positive plus negative feedback (based on a continuous reinforcement schedule) on reaction time on a DRTT. The mean response time data revealed that all three reinforcement conditions were effective in increasing response times in both hyperactives and controls. However, the number of interstimulus responses, or impulsive behavioural errors, exhibited by the ADHD sample increased dramatically in only the reward, or positive feedback, condition. In contrast, the normal control children evidenced a nonsignificant increase in impulsive responses in this condition. Douglas (Douglas, 1983, 1985; Firestone & Douglas, 1975) has interpreted these findings as highlighting the particular salience that reward has for ADHD children; although rewards appear to motivate these children to respond faster, they also increase the likelihood of associated difficulty in inhibitory control. Moreover, other studies (Cohen, 1972; Douglas & Parry, 1983) indicate that hyperactive children are less likely than normal controls to maintain reward-induced improvements when the rewards are removed. These data suggest that the reinforcement abnormalities observed in child ADHD groups are manifested in an unusual sensitivity "both to the presence of rewards and to the loss of anticipated rewards" (Douglas, 1983, p. 302).

Although these studies are presented by Douglas (1983, 1985) to support her theory of abnormal reinforcement mechanisms in ADHD children, the evidence is not entirely conclusive. Research that has systematically investigated the reinforcement hypothesis is limited and inconsistent. Differential patterns of response to partial reinforcement (Cunningham & Knights, 1978; Pelham, Milich, & Walker, 1986) and to punishment, in

the form of response-cost or loss of monetary reward (Solanto, 1990), have not been confirmed on different tasks in subsequent research, although the samples used most often comprised mixed ADHD/CD groups of children (e.g., Pelham et al., 1986; Solanto, 1990). However, careful examination of data for individual subjects was undertaken in one study (Solanto, 1990) and revealed the presence of a small subgroup who showed a pronounced increase in impulsive errors in the response-cost condition on a DRL task. This suggests the possibility that only a subgroup of the ADHD population shows a differential pattern of response to specific reinforcement contingencies. Interestingly, research examining avoidance learning and risk taking behaviour in ADHD children provides tentative support for this conclusion (Freeman, 1978).

Freeman (1978) used the Lykken maze (1957) paradigm, originally designed for the study of antisocial populations, as a measure of avoidance learning in groups of ADHD children and normal controls. A deficit in passive avoidance learning has played a prominent role in theories of CD and psychopathy (e.g., Blackburn, 1983; Gorenstein & Newman, 1980). Various derivatives of the Lykken maze test have documented a passive avoidance deficit in conduct disordered and psychopathic samples (Davies & Maliphant, 1974; Lykken, 1957; Schachter & Latane, 1964; Schmauk, 1970), although diminished responsiveness to punishment appears to be influenced by the probability and type of reinforcement. In view of the prominence of impulse control problems in ADHD, CD, and psychopathy, the performance of hyperactive children on the Lykken maze test was of particular interest.

In Freeman's modified version of the Lykken maze, subjects pushed buttons corresponding to coloured lights in an attempt to progress from top to bottom of the maze. The subjects' task was to use feedback to discover the correct sequence of lights and progress through the maze (the "manifest" task). In addition to the provision of positive feedback for correct choices, the task involved the administration of negative feedback for

incorrect choices in the form of noxious, or unpleasant, noise. Hence, the subjects' task also included learning to avoid the particular lights that were associated with punishment (the "latent" task). Avoidance learning was measured by the ability to learn the correct response in order to avoid the aversive event.

In a series of studies using this general design, Freeman obtained a differential pattern of performance for favourable stimulant drug responders (n=33/n=31) as compared to adverse responders (n=20) and quasi-normal controls (n=11), who ranged in age from 6 years to 16 years. Favourable drug responders ("true hyperactives" by the experimenter's definition) on placebo evidenced significantly more passive avoidance errors than the quasi-normal sample, whereas adverse responders on placebo did not differ from the quasi-normal group of children. Freeman interpreted these findings as corroborating defective avoidance learning in ADHD children. Douglas, however, in her reviews of the literature on reinforcement mechanisms in ADHD (Douglas, 1983, 1984), has proposed that particularly strong approach tendencies and attraction to salient, rewarding stimuli can account for these findings. She suggested that the "interesting and exciting game of finding their way through the maze" may have absorbed the attention and motivation of the "true hyperactives" to the extent "that they were either less aware of the specific cues provided by the unpleasant noise or were less influenced by them" (Douglas, 1983, p. 320).

Similarly, Douglas (1983, 1984, 1985) has offered an alternative explanation, in terms of strong approach tendencies, for Freeman's (Freeman, 1978; Freeman & Reznick, 1984) results from a risk-taking task with dichotomous groups of favourable responders, adverse responders, and normal peers. Risk taking behaviour was examined under two conditions: threat of loss of monetary reward (response-cost) and threat of electric shock. Findings indicated a significant effect for type of risk. In the monetary punishment condition, all subjects took about the same number of risks, irrespective of diagnosis or drug-response status. By contrast, the favourable responders on placebo did not

significantly reduce their risk taking behaviour in the threat of electric shock condition, whereas the adverse responders and normal controls substantially reduced the number of risks they took.

Although this data can be understood in terms of deficient avoidance learning in the presence of imminent electric shock, Douglas (1983) has emphasized the ADHD children's differential pattern of response to the variation in punishing reinforcers. The pattern of results in the Freeman study is in close agreement with findings from a similar investigation of impulse control in antisocial adults (Schmauk, 1970) in which subjects demonstrated an avoidance deficit on the Lykken maze when errors were punished by electric shock, but were able to perform as well as nonpsychopaths when response-cost (loss of money) was used to punish errors. This suggests a particular sensitivity to punishment when it involves response-cost, but not when it involves noxious stimuli. Gorenstein and Newman (1980) addressed the findings from the Schmauk study, along with the data of a number of other experiments presented in their review, and disagreed with interpretations that focus exclusively on the apparent avoidance deficit of antisocial groups while ignoring the role of reward dominance and the tendency to seek immediate gratification. They emphasized the "irresistable and exaggerated hold that ... immediate reward has [on the disinhibited individual's attention]" (p. 313). Extending this reasoning to ADHD samples, Douglas (1983, 1985) has argued that the results of the Freeman study on risk taking behaviour are consistent with other research indicating that ADHD is characterized by a heightened sensitivity to reward and to the anticipated loss of expected rewards. Thus, deficient inhibition per se may not be the problem, but may interact with a hyperresponsivity to reward.

At this juncture, recent parallel developments in the literature on CD and psychopathy that mirror the conclusions of Douglas (1983, 1985) and Gorenstein and Newman (1980) merit consideration. Quay (1988; Shapiro et al., 1988) and Newman

(Kosson, Smith, & Newman, 1990; Newman, 1987; Newman & Kosson, 1986; Newman, Patterson, & Kosson, 1987; Newman, Widom, & Nathan, 1985) have independently developed and investigated similar learning theories of antisocial behaviour in understanding conduct disordered adolescents and adult psychopaths. They have suggested that antisocial individuals tend to focus on the prospect of reward, at the expense of attending to cues of punishment, when the competing contingencies of reward and punishment are present.

Drawing on Gray's (1976, 1981) two-factor learning theory, Quay (1988) has argued that hyperresponsivity to reward, or reward dominance, is an important element underlying antisocial behaviour and may be linked to an imbalance between a Behavioural Activation, or Reward, System (BAS) and a Behavioural Inhibition System (BIS). Gray conceptualized the BAS as activating behaviour in response to cues of reward and nonpunishment, and the BIS as inhibiting behaviour in response to cues of punishment or frustrating nonreward. Gray attributed deficits in passive avoidance learning to a less sensitive BIS coupled with a BAS that was hyperresponsive to signals of reward.

Quay's theory of reward dominance has received support from studies of conduct disordered adolescents (Shapiro et al., 1988) and adult psychopathic inmates (Newman et al., 1987). Reward dominance was operationalized as the number of cards played in a computerized gambling task that pitted the probability of reward stimuli (cards) against punishing stimuli (losing cards). When compared to non-CD and non-psychopathic controls, both conduct disordered adolescents and psychopathic offenders chose to see significantly more cards, suggesting a relative increased responsivity to reward as opposed to punishment involving response-cost.

Similarly, Newman and his coworkers have conceptualized deficient passive avoidance learning as a trade-off between approach tendencies and response inhibition, with

an increased focus on reward leading to decreased attention to cues of punishment. Newman and Kosson (1986) speculated that earlier findings of adequate passive avoidance learning in psychopaths under punishment conditions involving response-cost (Schmauk, 1970) might not hold up under conditions of competing reward and response-cost contingencies. Following this logic, Newman, Widom, and Nathan (1985) assessed passive avoidance learning in psychopathic delinquents and controls under two conditions of reward and punishment versus reward only. They used a paradigm that provided monetary rewards for responding to positive stimuli and monetary punishments (response-cost) for responding to negative stimuli. As predicted, psychopathic delinquent subjects evidenced significantly more passive avoidance (commission) errors than controls in the task involving reward and punishment contingencies, but did not differ from controls in the task involving reward only. Subsequent studies (Kosson et al., 1990; Newman & Kosson, 1986), using adult psychopathic offender and nonpsychopathic offender controls, provided further evidence that the absence of performance differences between psychopathic and nonpsychopathic groups is specific to experimental conditions involving only one motivationally significant goal: in these cases, punishment (response-cost).

Interestingly, although the differences were not significant, each of these studies (Kosson et al., 1990; Newman et al., 1985, 1986) observed a tendency toward fewer omission errors (i.e., failure to respond to positive stimuli associated with reward) in psychopathic subjects under conditions of competing reward and punishment, but not under conditions of reward only or punishment only. This suggests that psychopathic adolescents and adults were more cautious and effective in making correct detections when presented with conditions involving cues for both reward and punishment. These findings are in keeping with Quay's (1988; Shapiro et al., 1988) theory of reward dominance, which predicts a greater responsivity to stimuli associated with reward and decreased avoidance to stimuli associated with punishment when competing reward and punishment incentives are present.

More recent research (Scerbo, Raine, O'Brein, Chan, Rhee & Smiley, 1990) provides direct support for Quay's hypothesis of hyperresponsivity to rewards in psychopathic adolescent offenders. Scerbo and her coworkers assessed the performance of psychopathic and nonpsychopathic delinquents on Newman and Kosson's (1986) computerized discrimination task under a condition involving reward and punishment incentives. Findings revealed that psychopathic adolescents demonstrated significantly fewer omission errors relative to nonpsychopathic delinquent controls, indicating a relative increased attention to, or responsivity to, reward stimuli. The passive avoidance error measure, however, failed to distinguish the two groups. Scerbo interpreted these results as suggesting that psychopathic subjects are capable of enhanced attention when sufficiently motivated or aroused.

In view of evidence of the high comorbidity of CD with ADHD, and that psychopathy arises out of CD (Quay, 1986; Robins, 1979), the literature on reward dominance and passive avoidance learning in conduct disordered and psychopathic groups has special relevance for the study of ADHD, particularly in the case of the mixed ADHD/CD subtype. For example, as previously discussed, studies of conduct disordered and psychopathic groups did not obtain performance differences in passive avoidance errors and in omission errors when experimental conditions included only one motivational goal (i.e., reward only or punishment only). This pattern of findings may explain failures to obtain such differences in ADHD child samples when contingencies involved reward only, or response-cost only (Solanto, 1990). At present, the robustness and precise nature of the reinforcement deficit in the ADHD population is unclear. The available data suggest that ADHD children, or else a subgroup of the ADHD population, show a relative increased sensitivity both to the presence of rewards and to the loss of anticipated rewards. Avoidance learning has been observed in ADHD samples only under punishment conditions involving aversive stimuli, or threat of presentation of noxious stimuli, but not under

conditions involving response-cost. Reward dominance and passive avoidance learning in ADHD groups have yet to be examined under conditions of competing reward and response-cost contingencies. The findings from the literature on CD and psychopathy are provocative and have potential importance for understanding the mixed ADHD/CD subtype, particularly with respect to the planning of treatment programmes.

Chapter 4
Stimulant Therapy: A Selected Treatment Option For The Mixed ADHD/CD Subtype

Although the past two decades have witnessed shifts in the terminology applied to the clinical condition, in the assumed primacy of one cardinal feature as compared to another (i.e., hyperactivity versus inattention), and in conceptualizations of clinical course, the primacy of the role of one specific mode of intervention in treating ADHD has remained constant, namely, the use of psychostimulant medication. Uniformly intense research attention has focused on pharmacological intervention for ADHD children with one of the stimulant drugs (i.e., methylphenidate hydrochloride (MPH, Ritalin), amphetamine (Benzedrine), dextroamphetamine sulphate (d'amphetamine, Dexedrine), or magnesium pemoline (Cyclert)) for more than 25 years. Despite the availability of various forms of behavioural, cognitive-behavioural, and combination behavioural-pharmacological therapies, psychostimulant medication has been the most prevalent, and often preferred (Gadow, 1981), method of treatment for ADHD, largely owing to its pronounced effects on a wide range of symptomatology and its relative cost-effectiveness (Bosco & Robin, 1980; Safer & Krager, 1984; Sandoval et al., 1980). In fact, stimulant drug treatment for ADHD has enjoyed the most careful and extensive empirical documentation of all the pharmacological treatments of childhood psychiatric disorders (Cantwell, 1979; Cantwell & Carlson, 1978).

Historically, a stimulant medication (amphetamine) was the first effective agent employed in ameliorating the disruptive and impulsive behaviours of ADHD children (Bradley, 1937). Since that observation some 50 years ago, no other medication has generally replaced stimulant drug treatment for the disorder, although other classes of drugs, such as antidepressants and neuroleptics, have been tried with ADHD (Conners & Werry, 1979). It has been estimated that 80% to 90% of all ADHD children have been treated with one of the stimulant drugs at some time (Bosco & Robin, 1980) and that

between 1% and 2% of all elementary school children in North America receive stimulant medication for treatment of learning or behavioural problems (Sprague & Gadow, 1976; Gadow, 1981). In most cases, the specific drug administered is either MPH or dextroamphetamine; these two drugs, which are very similar in their behavioural effects, have the best documented, most positive, and least toxic influence on the largest number of hyperactive children.

Consequently, the past two decades have witnessed a considerable accumulation of knowledge of the effects of stimulant medication for the treatment of ADHD. Research unequivocally demonstrates that stimulants given to children with ADHD are effective in reducing core symptomatology in the short-term, although the long-term efficacy of stimulant treatment has yet to be determined, largely due to methodological shortcomings inherent in outcome studies (Brown & Borden, 1986). To date, there have been over 100 controlled studies of acute stimulant effects demonstrating significant improvement in 60% to 90% of affected children (e.g., Campbell et al., 1971; Cantwell & Carlson, 1978; Rapport et al., 1986). The short-term positive effects of stimulants, and their superiority over placebo, have been documented in a wide range of areas, including behavioural, social, motor activity, perceptual performance, response inhibition, attention regulation, classroom disruption, and cognitive performance (see reviews by Barkley (1977) and Whalen & Henker (1976)). While it is not yet clear whether stimulant treatment in ADHD children improves performance on general aptitude and cognitive measures, such as concept learning and academic achievement, there is a solid body of evidence reporting positive effects when specific laboratory measures are used to assess acute treatment effects (Barkley, 1977; Kavale, 1982; Ottenbacher & Cooper, 1983).

The observed short-term effects of stimulant medication upon the cognitive and behavioural functioning of ADHD children include the following: (1) performance on fine motor tasks is improved and there is a reduction in task-irrelevant behaviour (Abikoff &

Gittelman, 1985; Conners & Werry, 1979; Cunningham, Siegel, & Offord, 1985; Gittelman Klein, Klein, Feingold, 1983; Pelham, McBurnett, Harper, Milich, Murphy, Clinton, & Thiele, 1990; Solanto, 1986); (2) there is a reduction in errors of omission on laboratory measures that require sustained performance (Campbell et al., 1971; Michael, Klorman, Salzman, Borgstedt, & Dainer, 1981; Rapport et al., 1986; Rapport, Jones, DuPaul, Kelly, Gardner, Tucker, & Shea, 1987); (3) there is an increase in the accuracy of performance in laboratory measures that require vigilance, as well as immediate and delayed perceptual judgements (Anderson, Halcomb, Gordon, & Ozolins, 1974; Campbell et al., 1971; Conners, 1972; Conners & Rothschild, 1968; Michael et al., 1981; Werry & Aman, 1975; Rapport et al., 1986); (4) there is a reduction in the number of impulsive errors on the MFFT and the CPT (Brown & Sleator, 1979; Campbell et al., 1971; Milich, Licht, Murphy, & Pelham, 1989; Rapport, DuPaul, Stoner, Birmingham, & Masse, 1985; Rapport et al., 1986; Rapport, Stoner, DuPaul, Kelly, Tucker, & Schoeler, 1988); (5) stimulant medication leads to more deliberate and regulated responding in reaction-time tasks where the desired response is inhibition of impulsive responding (Cambpell et al., 1971; Tannock, Schachar, Carr, Chajczyk, & Logan, 1989); (6) stimulants positively affect performance and reduce variability of performance on reaction-time tasks (Barkley, 1976; Cohen et al., 1972; Sprague, Barnes, & Werry, 1970; Stroufe, 1975); and finally, (7) there is some data to suggest that stimulants improve avoidance learning on the Lykken maze and reduce risk taking behaviour when punishment consists of noxious stimuli (Freeman, 1978).

Observational studies indicate that stimulant treatment in ADHD children reduces the disruptive, oppositional, and aggressive behaviours associated with the disorder (Arnold, Huestis, Smeltzer, Scheib, Wemmer, & Colner, 1976; Barkley, 1977; Gittelman Klein 1975; Gittelman Klein & Klein, 1976; Hinshaw, Henker, Whalen, Erhardt, & Dunnington, 1989; Sprague, Barnes, & Werry, 1970; Ullmann & Sleator, 1985). Data further indicate that the beneficial effects of stimulant medication on activity level, sustained attention, and impulse control translate into significant improvements in social interactions with parents (Barkley & Cunningham, 1980; Barkley, Karlsson, Strzelecki, & Murphy, 1984; Barkley, Karlsson,

Pollard, & Murphy, 1985;) and peers (Cunningham et al., 1985; Cunningham, Siegel, & Offord, 1991; Hinshaw, Buhrmester, & Heller, 1989; Whalen, Henker, Buhrmester, Hinshaw, Huber, & Laski, 1989).

In conclusion, evidence of stimulant-induced improvement in ADHD children has been documented on a wide array of measures that share common sources of variance involving self-regulatory processes or executive functions. These include the effective and consistent deployment of attentional capacity, cognitive and behavioural inhibitory control, improved capacity to plan and consider response alternatives, and improved ability to shift mental set productively (Douglas, 1983, 1989; Douglas et al., 1988). Executive, or self-regulatory, control is involved in tasks that require the choice, construction, execution, and maintenance of optimal strategies for performance, as well as the inhibition of strategies that are either inappropriate to task demands or become counterproductive with the occurrence of errors (Logan, 1985). Another approach to understanding the processes that measures used in pharmacological treatment studies may have in common is to statistically examine shared variance and the strength of correlations. For example, Kupietz and Richardson (1978) examined the relationship between laboratory and naturalistic measures of attention and observed significant correlations between errors on the CPT and off-task behaviour in the classroom setting. Although the correlations were moderate, these results provided tentative support for the external validity of vigilance tasks as analog measures of academically relevant attentiveness and are consistent with other research findings in this area (Rapport et al., 1986). Therefore, stimulants may improve focal attention over time and under conditions in which systematic nonresponse to extraneous stimuli is required.

The picture that is emerging from the literature is that stimulants activate self-regulatory processes in such a way that information processing skills are mobilized and task performance is facilitated (Douglas, 1972, 1983; Douglas et al., 1988; Humphries, Swanson, Kinsbourne, & Yiu, 1979). Investigators have largely emphasized the critical role of

psychoactive medication in improving the deployment of attentional capacity and inhibition of impulsive responding. Although it is generally agreed upon that these cardinal deficits are ameliorated by stimulant therapy, it is not yet clear whether the processes involved are affected independently. Moreover, it may well be that the drug-induced improvement of core ADHD symptomatology is related to the observed reduction in noncompliant and disruptive behaviours associated with the disorder.

It was earlier assumed that a positive response to stimulants was both necessary and sufficient to make a diagnosis of ADHD; it is now clear that response to a stimulant drug is not an adequate criterion (Douglas, 1984; Rutter, 1983; Taylor, 1983). This assumption was largely based on the erroneous conclusion that stimulants had a uniquely calming, or "paradoxical", effect in ADHD children. The growing consensus is that stimulant-induced task and behavioural improvements are neither paradoxical nor specific to individuals with biological dysfunction. Studies of mixed diagnostic groups have not shown differential efficacy by diagnosis (Bradley, 1937; Arnold, Christopher, Huestis, & Smeltzer, 1978) and, moreover, normal children and adults respond to stimulant medication in ways that are similar to ADHD children (Rapoport, Buchsbaum, Weingartner, Zahn, Ludlow, & Mikkelsen, 1980; Rapoport, Buchsbaum, Zhan, Weingartner, Ludlow, & Mikkelsen, 1978), particularly when tasks requiring relatively low-level processing are employed (i.e., CPT and reaction-time tasks) (Swanson & Kinsbourne, 1979).

Further, although substantial research has demonstrated unequivocally that the majority of ADHD children respond positively to psychostimulant treatment, not all do. The present discussion has previously addressed a range of dimensions along which ADHD children are heterogeneous, namely, in symptom severity, presence of associated symptomatology, and prognosis. ADHD children also show considerable interindividual and intraindividual variability in response to stimulant treatment (Ross & Ross, 1982). Only 50% to 70% of ADHD children show a beneficial response to psychostimulant

medication, with another group showing either no effect or an adverse response that requires medication withdrawal (Barkley, 1977; Cantwell & Carlson, 1978; Conners & Werry, 1979; Swanson & Kinsbourne, 1979). Of the group who respond favourably to stimulant medication, one-third to one-half appear to generally show immediate and marked improvement, with the remainder showing only moderate improvement (Fish, 1971; Ross & Ross, 1982). Heterogeneity in stimulant drug response is found not only across children, but also across performance and task domains for the individual child (Douglas et al., 1988; Rapport et al., 1986). Hence, stimulant-related improvements in one behavioural domain are not necessarily obtained in other domains for a particular child. There is also evidence suggesting that different dosage levels affect changes in different behavioural domains (Sprague & Sleator, 1977). This wide heterogeneity in stimulant treatment responsiveness across child, task, time, situation, and dosage has hampered attempts to specify predictors of positive drug response (Douglas et al., 1986; Pelham, Bender, Caddell, Booth, & Moorer, 1985; Sebrechts, Shaywitz, Shaywitz, Jatlow, Anderson, & Cohen, 1986). Therefore, the available evidence do not support a core syndrome of ADHD that is uniformly responsive to stimulant drugs, nor are stimulant drug effects specific to the ADHD syndrome (Taylor, 1983).

Another erroneous assumption that has historically pervaded the literature on ADHD involves the use of psychoactive medication with adolescents. In contrast to the well-documented short-term efficacy of psychopharmacotherapy in ADHD children, pharmacological intervention as a potentially adjunctive measure in the treatment of ADHD adolescents has been seriously neglected. This has had much to do with clinical lore that stimulants were to be used only with ADHD preadolescent children experiencing attentional and concentration problems and that ADHD symptomatology was outgrown in adolescence (Bakwin & Bakwin, 1966; Eisenberg, 1966). It was further believed that the "paradoxical" organizing effects obtained in ADHD prepubertal children were unique to this age group and would be replaced by a "normal" excitatory response after puberty

(Gross & Wilson, 1974). However, as outcome and psychopharmacological data became available, these myths have fallen by the wayside and have reawakened interest in the question of treatment considerations for adolescents with the disorder (e.g., the 1984 National Institute of Mental Health (NIMH) workshop co-chaired by Weiss and Hechtman addressed "Pharmacotherapy for ADD-H Adolescents"). The observation that ADHD adolescents often manifested attentional and impulse control deficits that were similar to those observed in their younger counterparts suggested that stimulant medication would yield beneficial results in the adolescent population. Consequently, there is a growing consensus in the literature that ADHD adolescents may benefit markedly from stimulant pharmacotherapy (Brown, Borden, & Clingerman, 1985; Cantwell, 1979, 1986; Clampit & Pirkle, 1983; Conners, 1985; Sorosky, 1979; Weiss & Trokenberg Hechtman, 1986). Ross and Ross (1982), for example, in their comprehensive book *Hyperactivity: Current Issues, Research and Theory*, concluded that "stimulant drug treatment for older hyperactives is a research area destined for marked expansion in the early 1980's" (p. 186).

Stimulant Treatment With the ADHD Adolescent Population

To date, there is a dearth of research examining the efficacy of stimulant medication on ADHD adolescents. This is problematic because data indicate that the rate of pharmacological treatment with stimulants among adolescents between 12 and 15 years of age has dramatically increased between the years 1975 and 1983 (Safer & Krager, 1985) and anywhere from 30% to 50% of patients, who received stimulants in childhood, currently continue their treatment during the adolescent years (Safer & Kruger, 1985). The most systematic and controlled investigations of pharmacological intervention with ADHD adolescents are studies of the acute effectiveness of psychoactive medication. However, such studies are remarkably few in number. Similarly, there are relatively few empirical data on the efficacy of psychopharmacotherapy as an adjunct to other forms of treatment with ADHD adolescents. The purpose of the current study, therefore, was to examine the

potential efficacy of stimulant therapy for the ADHD adolescent age group and to explore the acute effects in a more homogeneously defined sample of ADHD adolescents who show co-existing CD. Preliminary to a discussion of the present investigation, however, is a review of the literature on reports of stimulant drug efficacy in ADHD adolescents. Studies are summarized in Table 2 and are categorized on the bases of methodology and sample diagnoses.

Refer to Table 2 on the following pages.

The first investigators to report the efficacy of MPH in an uncontrolled study of 10 youths, aged 13 to 18 years, who showed symptoms of hyperkinesis and associated learning disabilities, were MacKay, Beck, and Taylor (1973). All youths were judged to be improved clinically, general improvement in school performance was observed, and 9 of the 10 patients improved in their visual perceptual performance as measured by the Raven Progressive Matrices test. A later study by Lerer and Lerer (1977) confirmed these findings.

Safer and Allen (1975) studied response to an open trial of stimulants in three groups: hyperactive adolescents who received stimulant medication as children (n=14), hyperactive adolescents who continued use of stimulant medication from childhood through to adolescence (n=13), and hyperactive adolescents who began stimulant therapy in adolescence (n=14). These youths were judged to be hyperactive on the basis of developmental signs of hyperactivity, classroom inattentiveness, learning or perceptual delay, and conduct problems. Hence, the sample used in this uncontrolled study likely represented a group of ADHD adolescents who showed some degree of associated aggression, oppositional disorder, or CD. The outcome measures at 1-year and 2-year follow-ups included teacher ratings, with improvement of 50% over baseline considered to be a satisfactory treatment outcome. The authors observed that the therapeutic response of

Table 2

Studies of the Effects of MPH in the ADHD Adolescent Population

Study	Methodology	Diagnosis	Dosage	N	Findings
Mackay, Beck, Taylor (1973)	Open trial No Control	Hyperkinesis, Associated LD, "Neurophysiologic immaturity"	10-mg 2 x day - 20-mg 3 x day	10	General clinical improvement; Enhanced school performance; Improved visual perceptual performance
Safer & Allen (1975)	Open trial No control	Developmental hyperactivity, Inattentiveness, Conduct problems	10-mg - 40-mg day	41	Improvement on teacher ratings
Lerer & Lerer (1977)	Open trial No control	Hyperactivity, Inattention	20-mg 2 x day - 30-mg 2 x day	27	Improvement on teacher ratings; Improved visual motor performance
Coons, Klorman, & Borgstedt (1987); Klorman, Coons, & Borgstedt (1987)	Double-blind control	ADDH in childhood	25-mg day vs.	19	Improvement on measures of attention and memory
		RADD in adolescence	40-mg day		Improvement in parent ratings of inattentiveness and noncompliance; Marginal improvement on teacher ratings

Table 2 - continued

Studies of the Effects of MPH in the ADHD Adolescent Population

Study	Methodology	Diagnosis	Dosage	N	Findings
Varley (1983)	Double-blind control	ADD in childhood, RADD in adolescence, CD excluded	0.15-mg/kg vs. 0.3-mg/kg	22	Improvement on parent and teacher ratings
Brown & Sexson (1983)	Double-blind control	ADDH in childhood, RADD in adolescence, 45% of sample had mixed ADDH/CD	0.15-mg/kg vs. 0.3-mg/kg vs. 0.5-mg/kg	11	Improved performance on measures of attention and impulsivity; General improvement on 75% of 36 behavioural, academic, and laboratory measures used

Note. LD = learning disability
RADD = Residual ADD

stimulants in improving ADHD symptomatology did not significantly change from age 6 to 16 years. Overall, 67% to 100% of youths across the three groups were deemed to have shown improvement at the end of 1 and 2 years. The mean doses of MPH and dextroamphetamine found to be effective in improving class performance were not significantly different for older adolescents than for children. These findings suggested that ADHD adolescents respond as well as their younger counterparts to psychoactive medication, and supported earlier clinical reports of Oettinger (1973) and Gross and Wilson (1974).

Despite the relatively successful open trials and clinical reports of the use of stimulants with an ADHD or mixed ADHD adolescent population, at present, only three controlled studies have been reported with ADHD youths (Brown & Sexson, 1988; Coons, Klorman, & Borgstedt, 1987; Klorman, Coons, & Borgstedt, 1987; Varley, 1983). As outlined in Table 2, in all cases, subjects were diagnosed with ADHD in childhood based on either DSM-III criteria (Brown & Sexson, 1988; Varley, 1983) or retrospective questionnaire ratings (Coons et al., 1987; Klorman et al., 1987) and met current criteria for ADD, Residual type (RADD). Importantly, the presence of associated CD was systematically evaluated in two of the studies; subjects with concurrent CD were excluded in Varley's (1983) study, whereas 45% of Brown and Sexson's (1988) sample had co-existing CD. Over 60% of the adolescent subjects had been identified as previous responders to stimulant medication in two of the studies (Coons et al., 1987; Varley, 1983).

Coons and her coworkers (Coons et al., 1987; Klorman et al., 1987) assessed the acute effects of MPH on 19 RADD adolescents (aged 12 to 19) in a 6-week double-blind crossover design of two doses of MPH (25-mg/day as compared to 40-mg/day) and placebo. Under MPH, subjects detected significantly more targets, responded faster to target stimuli, and showed augmented sensitivity of detection, as indexed by d', on the CPT. MPH treatment also enhanced the accuracy and precision of information processing as measured

by a memory search task. Additionally, the performance of a pilot subgroup that had been treated with stimulant medication in childhood (n=6) was compared to an equal number of controls, who did not have a history of stimulant therapy, and were matched for age, I.Q., and diagnostic data. No differences in performance between the two groups were observed, suggesting that exposure to stimulant medication in childhood is unrelated to drug response in adolescence. In a companion paper (Klorman et al., 1987), the stimulant drug effects on parent and teacher rated inattentiveness and compliance were reported. Here, abbreviated Conners ratings completed by parents indicated significant reductions in inattentiveness and disobedience, whereas the magnitude of the change as rated by teachers in the active phases of the trial was markedly less and became salient only with the higher dose.

The observation of MPH-related improvement on parent ratings of behaviour is further supported by the work of Varley (1983). Twenty-two adolescents (17 male and 5 female), aged 13 to 18 years, were assessed in a 3-week double-blind active drug (0.15-mg/kg versus 0.3-mg/kg) and placebo study. Results indicated that both the low and moderate dose levels of MPH were significantly more effective than placebo in improving general behaviour, as measured by parent and teacher ratings. Sixteen of the 22 subjects (73%), all identified as previous stimulant drug responders, continued to show improvement with MPH treatment in adolescence. There was no correlation of drug treatment response with the sex of the subject.

More recently, Brown and Sexson (1988) examined the effects of three doses of MPH (0.15-mg/kg, 0.3-mg/kg, and 0.5-mg/kg) and placebo on 11 black males, aged 12 to 14 years, in an 8-week double-blind crossover design. They observed significant improvement on 28 of the 36 (75%) dependent measures, which included behavioural (Conners parent and teacher ratings scales) and laboratory (CPT and MFFT) measures of attention and impulsivity, as well as academic and physiological indices. In general, the higher doses resulted in the most beneficial response to the measures. Although the authors concluded

that their findings corroborate the efficacy of MPH with black adolescents who have ADHD or mixed ADHD/CD, these conclusions are tempered by problems in their methodological design and in the statistical comparisons made; namely, 36 dependent measures were derived with a small subject sample and control for family-wise type I error was not included in statistical analyses.

What emerges from the available literature on stimulant pharmacotherapy with ADHD adolescents is a suggested picture of beneficial effects in reducing core symptomatology in the short-term. Contrary to the notions that psychostimulants have a paradoxical effect on prepubertal children and that the actions of stimulants are reversed at puberty, the data point to many similarities between the acute drug effects in ADHD adolescents and their younger counterparts. These include improvements on behavioural ratings by parents and teachers, enhanced cognitive functioning, and better inhibitory control. Moreover, the drug dosages used in the reviewed studies were comparable to effective, absolute dosages in prepubescent children (Varley, 1983). Dosages in pharmacotherapy studies with adolescents have ranged broadly from the equivalent of 10-mg of MPH a day to a maximum of 60-mg a day, the latter being higher than a 1.0-mg/kg dosage for a 100 pound adolescent. Although, some researchers have suggested that the cognitive effects of psychostimulants for school-age children are maximized at a dose of 0.3-mg/kg (Brown & Sleator, 1979; Sprague & Sleator, 1977), gains on cognitive and academic measures have been reported with the prepubertal population on dosages up to 0.8-mg/kg (Pelham, Bender, Caddell, Booth, & Moorer, 1985; Rapport, DuPaul, Stoner, & Jones, 1985) and 1.0-mg/kg (Gittelman Klein, Klein, & Feingold, 1983).

Importantly, no addiction or increased drug abuse has been reported thus far in adolescents exposed to psychoactive medication. Historical allegations of a predisposing role of stimulant drugs to the development of substance abuse (Clampit & Pirkle, 1983) have not received empirical support (Ackerman, Dykman, & Peters, 1977; Hechtman &

Weiss, 1986; Henker et al., 1981; Klorman, Coons, & Borgstedt, 1987). In fact, the presumptive evidence is that a favourable response to stimulant treatment is associated with a lower probability of drug abuse in children followed into adolescence (Kramer & Loney, 1981). Nevetheless, given the abuse potential and street value of stimulant drugs, safeguards in prescription and administration must be established with adolescents. Clearly, stimulants would be contraindicated in cases where there is pre-existing substance abuse and/or when siblings, or parents, have substance abuse disorders.

Additional support for the use of pharmacotherapy during adolescence is suggested by comprehensive reviews of stimulant drug effects (Solanto, 1984; Whalen & Henker, 1984) which have concluded that the areas of clearest improvement are the disruptive and antisocial behaviours associated with the disorder. More recent data also indicate quite promising effects of stimulant drugs on the secondary features of negative social interaction and aggression (Amery, Minichiello, & Brown, 1984; Cunningham et al., 1985, 1991; Hinshaw et al., 1989; Winsberg, Press, Bialer, & Kupietz, 1974; Whalen et al., 1989). These findings gain importance in view of the high prevalence of persistent delinquent behaviour in 10% to 50% of ADHD children followed into adolescence and the high comorbidity of CD with ADHD. They further raise the possibility that stimulant treatment may exert a particularly beneficial influence on the subgroup of adolescents who show mixed ADHD/CD.

Due to the considerable overlap between ADHD, aggression, and CD, and a historic failure to control for presence or absence of associated CD in ADHD samples, past psychopharmacological studies of samples of children labelled ADHD, or CD, have been quite heterogeneous, resulting in obscuration of potentially important subgroup responses. Few investigations have assessed stimulant effects on children specifically diagnosed as CD, largely because clinical lore has suggested that stimulants were to be used with ADHD rather than with CD. Studies of the effects of amphetamines in delinquent samples have

largely been uncontrolled investigations of acute drug effects; positive outcomes have, nevertheless, been uniformly reported (Eisenberg, Lachman, Molling, Lockner, Mizelle, & Conners, 1963; Korey, 1944; Maletzky, 1974).

In view of the empirical association between ADHD and CD, and the failure of research designs to distinguish ADHD subgroups on the basis of comorbidity, leading authors in the field of pediatric psychopharmacology have articulated a need for stimulant treatment studies with child and adolescent groups that show both ADHD and conduct symptomatology (Cantwell, 1985b, 1986b; Weiss & Hechtman, 1986). Cantwell (1986b) offered the following:

> What is needed are comparative studies of children with "pure ADDH", "pure conduct disorder", and with both ADDH and conduct disorder. These groups should be compared on baseline and over time on a wide variety of parameters, including measures of attention, activity, impulsivity, laboratory learning, academic performance, other cognitive areas, association with other problems, ... and short- and long-term response to stimulant drug treatment. (pp. 409-410)

At present, the acute effects of psychoactive drug treatment have not been investigated in mixed ADDH/CD adolescent groups. Recent controlled studies of the short-term effects of stimulant therapy on homogenous groups of ADHD prepubertal children may shed light on the potential responses of this subgroup. For example, Taylor and his coworkers (Taylor, 1983; Taylor, Everitt, Thorley, Schachar, Rutter, & Wieselberg, 1986; Taylor, Schachar, Thorley, Wieselberg, Everitt, & Rutter, 1987) observed that the major effects of stimulants (MPH) in ADDH, ADDH/CD, and CD-only groups of boys aged 6 to 10 years were on the symptoms of restlessness, impulsivity, and inattention based on behavioural ratings by parents and teachers. This "antihyperkinetic" effect of stimulant therapy was observed in each of the three groups of boys, including the CD-only group. The stimulant-related effects on the defiant and unruly behaviour of all three groups were less marked and fell short of statistical significance. Similarly, the obtained predictors of positive response to stimulant medication were restless-inattentive behaviours, rather than

features of impulsivity, defiance, or non-compliance (Taylor et al. 1987). However, neither the DSM-III diagnosis of ADDH or the ICD-9 diagnosis of "hyperkinetic syndrome" successfully predicted all children who showed a favourable drug-response. Taylor (Taylor et al., 1987) concluded that some children who show mixed ADDH/CD are likely to benefit from stimulant treatment, but "conduct disordered children with none of the features of hyperactivity are unlikely to respond well" (p. 140). Other research has consistently indicated that the benefits of acute stimulant treatment (MPH) on both laboratory and observational measures of attention, impulsivity, aggression, and general behaviour are comparable for ADHD children with and without high aggression scores (Barkley, McMurray, Edelbrock, & Robbins, 1989; Cunningham, Siegel, & Offord, 1991; Hinshaw, Henker, Whalen, Erhardt, & Dunnington, 1989; Klorman, Brumaghim, Salzman, Strauss, Borgstedt, McBride, & Loeb, 1988; Milich, Licht, Murphy, & Pelham, 1989). When differential patterns of acute response to stimulant drugs were obtained on particular measures (e.g., Barkley et al., 1989; Cunningham et al., 1991), the effect was attributed to initial base rate differences in performance.

In conclusion, controlled investigations of stimulant drug treatment in child groups who show ADHD and concurrent aggression suggest that there is not an overall differential response to stimulant medication in the presence of ADHD and co-existing CD. Previous research examining the acute effects of psychostimulant medication in the ADHD adolescent age group provides promising results of favourable responsiveness comparable to prepubertal peers. Thus, if a group of conduct disordered adolescents who have a childhood history of ADHD and currently meet diagnostic criteria for ADHD can be isolated and treated with stimulant medication, a proportion may be responsive. Additional acute studies are mandated to guide pharmacotherapy efforts with this population.

Chapter 5
The Current Study

As changing conceptualizations and concomitant shifts in terminology have confronted us with the limitations of previous knowledge, so too have new empirically precipitated developments within the area of ADHD prompted revision or expansion of research and clinical practice. One such area of change has been the identification and study of homogenous subgroups based on associated conduct symptomatology. There is now almost universal recognition that concurrent aggression and conduct disturbance must be addressed in research on ADHD in order to provide for more valid and homogeneous subject populations. Interestingly, a call for research designs to distinguish delinquent subgroups on the basis of ADHD comorbidity has also been voiced in the literature on CD (Moffit, 1990). These are timely and welcome developments, in that previous research that failed to consider the influence of associated psychopathology likely obscured important information with regard to key etiologic, prognostic, treatment-response, and concurrent mediating variables. This increased interest, in recent years, in the delineation of ADHD subtypes based on antisocial comorbidity may represent the most influential factor in furthering progress in research on ADHD, research which has implications that bridge the realms of methodology, theory, and intervention. Greater homogeneity of research samples will not only facilitate replication, a clearer understanding of the disorder and its comorbidity with CD, but valuable insight into the possible divergent etiologies of the subtypes and their responses to specific treatment regimes may be advanced as well.

A second area of change in research and clinical practice has been the investigation and use of psychopharmacotherapy with the ADHD adolescent population. Traditionally, pharmacotherapy with stimulant medication was considered to be most effective and exclusively applicable during middle childhood; the temporal window is currently being extended to include the adolescent age range, particularly in view of the serious negative

long-term outcome in a large proportion of children with the disorder. Clinical investigators are now documenting positive stimulant drug responsiveness with this population.

In view of both the high comorbidity of CD with ADHD and the robust indications of a chronic poor prognosis for this subgroup, a mandate for the evaluation of interventions for the mixed ADHD/CD subtype has been strongly voiced. Thus, the current research took as its point of departure the need for exploration of treatment options for ADHD adolescents who have co-existing CD and the currently reported successes with the use of stimulant treatment for the ADHD adolescent population. The present study further satisfied a number of research criteria which have been articulated in the literatures on ADHD, delinquency, and assessment of treatment efficacy. For example, well-controlled studies which examine the effectiveness of specific interventions for specific populations with specific symptoms in particular situations have been advocated (Mash & Dalby, 1979) as a guide to future intervention studies with the ADHD population of children and adolescents. Similarly, a need for more objective and precise measures of cognitive processing and behavioural change in research on ADHD has been emphasized (Hinshaw, 1987; Kinsbourne, 1989). The methodological shortcomings of the follow-up literature on ADHD have also highlighted the importance of selecting assessment instruments that are similar to those employed with their younger counterparts. Follow-up observations have supported continuing difficulties in attentional capacity, cognitive style, impulse inhibition, and reinforcement learning among ADHD adolescent probands. It is clear that adolescents should be evaluated in the same problem areas, using objective assessment instruments that tap similar processes and that have normative data in the adolescent age range (Brown & Borden, 1986; Cantwell, 1986a).

Accordingly, the current research investigated the acute effects of MPH on ADHD symptomatology in a subgroup of male, adolescent, inpatient offenders who met the combined diagnostic criteria for ADHD and CD. Various theoretical and methodological

limitations of previous research investigating ADHD were addressed in the study. The present design included: (1) careful selection of a homogeneous sample based on a dual diagnosis of ADHD and CD, with assessment of associated antisocial behaviour based on self-report, parental report, as well as official corrections history records; (2) selection of a specific treatment regime (MPH) with a history of reported success in the ADHD population and some indication of success with the mixed ADHD/CD subtype; (3) use of blind and pharmacologically inert treatment controls; and (4) use of objective laboratory measures of core ADHD symptomatology that have empirically researched reliability and validity with ADHD or delinquent populations. Laboratory measures of sustained attention, distractibility, inhibition of impulsive responding, responsivity to reward, and passive avoidance learning were selected for their similarity to measures employed in research with child ADHD samples and delinquent youth samples, with the intent that the use of similar measures would allow for continuity and comparison of results across ADHD age cohorts. Although these phenomena have received extensive investigation in the area of ADHD and/or CD, this study is one of the first to evaluate impairment in attentional capacity, impulse inhibition, reward dominance, and passive avoidance learning in ADHD adolescents with associated CD. The present research, therefore, represents an exploratory investigation of behavioural and cognitive deficits in the adolescent ADHD/CD subtype and was guided by the expectation that the sample would show deficits on tasks.

The low and/or moderate doses of MPH were expected to exert significant improvements, relative to placebo, on subjects' performances on the selected laboratory measures, although drug efficacy was expected to vary from measure to measure and to depend on the degree of impairment in baseline performance. This is based on data indicating that the amount of the drug tends to interact with the target behaviour (Douglas et al., 1986; Sprague & Sleator, 1975) and that stimulant drug effects are dependent on base-state, or base-rate of responding (Gualtieri, Hicks, Mayo, & Schroeder, 1984; Kinsbourne, 1985; Rapport, DuPaul, & Smith, 1985; Weber, 1985). There is also ample

data to suggest that the placebo treatment would be therapeutically efficacious on various measures with the present sample (Barkley, 1981; Gualtieri et al., 1984; Ottenbacher & Cooper, 1973; Ross & Ross, 1982; Varley, 1983; Werry, 1977; Werry et al., 1987). A significant proportion of ADHD children and adolescents evidence a beneficial response to placebo and ameliorative effects have been observed with placebo treatment in inpatient settings where expectations of improvement tend to be enhanced by the therapeutic milieu (Shapiro & Morris, 1978).

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PART B

METHOD

Chapter 1
Subjects

Subjects were 33 male adolescents between the ages of 12 and 17 (mean age=14.5, *SD*=1.5) who were remanded into custody at a closed, inpatient forensic assessment unit (Inpatient Assessment Unit (I.A.U.) of Juvenile Services to the Courts) for a psychological and psychiatric assessment due to criminal charges against them. Subjects selected for inclusion in the study met the combined criteria for ADDH and CD as specified by DSM-III and as diagnosed by a psychiatrist following a structured interview[2]. Subjects included in the study had formerly received a diagnosis of childhood hyperactivity by a physician (34% of the sample had a psychiatric history of childhood ADDH), and/or had met the operational criteria for childhood ADDH based on parental report. Additionally, subjects met the following criteria: (1) absence of significant neurological or psychiatric impairment outside the ADDH and CD spectrums; (2) no medical or clinical contraindications to MPH therapy such as tics, cardiovascular disease, or substance use disorder; (3) not currently receiving medication other than MPH and no abuse of substances for a 2-week time period before entering the protocol; and (4) a minimum Full IQ score of 75 on the Wechsler Intelligence Scale for Children - Revised (WISC-R, Wechsler, 1974) or the Wechsler Adult Intelligence Scale - Revised (WAIS-R, Wechsler, 1981) (mean FSIQ=98, *SD*=13.5).

One hundred and sixteen males (57%) met the diagnostic criteria for inclusion in the study out of a possible 204 males who were screened over a 20 month time period, from September 1, 1987 to June 12, 1989. The 204 males screened constituted 51% of a total of 404 admissions to the I.A.U. during the above time period. Of the possible candidates, 23 youths (20%) refused to enter the study (often reporting experience of previous side-effects

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[2] The diagnostic label ADDH will be used when specifically referring to the present sample. This is for the purpose of accuracy as diagnostic criteria were based on the DSM-III classification which was currently in use at the time of the study.

with MPH), 9 youths (8%) had clinical contraindications to Ritalin treatment such as cardiac complications and substance use disorder, and 51 adolescents (44%) were excluded for reasons such as a transfer to another facility due to overcrowding at I.A.U., their remand was of insufficient duration to permit completion of the protocol, and/or the nature of the assessment precluded their participation in the study (i.e., a possible transfer to adult court or consideration of fitness to stand trial).

Although 33 subjects entered the study, only 26 provided complete data. Two subjects' participation was terminated due to adverse side effects observed during the moderate dose phase, 3 subjects were unable to complete certain measures during one phase due to computer malfunctioning, 1 subject missed the placebo phase, and another subject missed the moderate dose phase due to a transfer to another forensic facility.

Subjects were predominantly Caucasian (n=29, 88%), but also included youths of Native Indian (n=3, 9%) and East Indian (n=1, 3%) descent. The majority of subjects' histories were characterized by disrupted and chaotic family backgrounds (65%). Only 12 adolescents (35%) were living in intact families at the time of the study; 13 (38%) were living with a single parent, 4 (12%) were placed in foster homes, and 5 (15%) were currently residing in group home facilities.

Self-report of recreational substance use was obtained prior to participation in the study. Five subjects (15%) reported regular use of alcohol and 8 subjects (24%) reported use of illicit drugs, such as marijuana, hashish, cocaine, heroin, L.S.D., and inhalation of glue or gasoline, on at least one occasion. Only 2 subjects (6%) reported use of any one of the above substances on more than one occasion during the 2-week time period prior to admission to I.A.U. Subjects' self-reports in this area were rarely substantiated by other data sources, such as a social worker, probation officer, or mental health worker. Therefore,

it is likely that the above statistics underrepresent the actual rate and extent of substance abuse in the present sample.

Subjects' official corrections histories were recorded using computerized offense records covering childhood through to the age of the subject as recorded by the Corrections Branch, Ministry of the Solicitor General, Province of British Columbia. Frequency and type of offense were measured, with type of offense classified into two broad-band categories of serious and non-serious offenses based on the criteria used by Satterfield, Hoppe and Schell (1982), and the listings of offenses as either summary or indictable in the Canadian *Criminal Code* (Greenspan, 1982). Serious offenses included robbery, breaking and entering, theft over $200, escaping lawful custody, drug trafficking, public fraud, extortion, arson, possession of a weapon, assault, and assault with a weapon and/or intention to harm. Non-serious offenses included alcohol intoxication, negligent and/or impaired driving, taking an auto without consent, breach of probation, possession of marijuana, vandalism, possession of stolen property, and theft under $200. Subjects' total numbers of serious and non-serious offenses ranged from 1 to 20 (M=6.16, SD=5.83).

Two narrow-band categories of offense were also included: offenses against persons and offenses against property. The category of offenses against persons encompassed a variety of aggressive acts including assault, assault with a weapon and/or intention to harm, and possession of a weapon. Offenses against property included such acts as theft, vandalism, possession of stolen property, arson, and breaking and entering. Table 3 presents the means, standard deviations, range, and the number of subjects engaging in each type of offense calculated for 25 of 33 subjects.

Table 3

Corrections History Expressed in Means, Standard Deviations, and Range of Offenses

Category of Offense	*n* (%)	*M*	(*SD*)	Range
Non-Serious Offenses	20 (80%)	3.36	(3.53)	1 - 11
Serious Offenses	20 (80%)	2.84	(3.06)	1 - 9
Offenses Against Persons	12 (48%)	1.04	(1.42)	1 - 5
Offenses Against Property	21 (84%)	3.40	(3.71)	1 - 14

Note. Corrections history records were available for only 25 of 33 subjects.

Diagnosis and Reliability

Diagnoses of ADDH and CD were based on behavioural criteria identical to those specified in DSM-III. An Interview Schedule (IS) was developed to include questions that covered the DSM-III symptom criteria for these disorders (see Appendix A). The IS followed both a retrospective assessment and a current assessment format for the diagnoses of ADDH in childhood and in adolescence. For the retrospective assessment of childhood ADDH, the subject was instructed to answer the IS questions in terms of his behaviour when he was between the ages of 4 to 10 years. During pilot work, however, it became readily apparent that the youths were poor historians and had considerable difficulty answering the retrospective questions. This is consistent with other findings (Gittelman & Mannuzza, 1985) indicating that ADHD adolescents' self-reports provide poor diagnostic information. The retrospective assessment portion of the patient IS (but not the parent version of the IS) was therefore discarded and was not used during the study proper. The current assessment format of the IS, for the diagnoses of both ADDH and CD in adolescence, included instructions for the subject to answer the questions in terms of his behaviour now or within the past 6 months. A requirement was provided in the IS for symptom items to be rated on a 4-point severity scale as well as for an overall two-category

rating of severity (mild/moderate or severe) of ADDH and of CD. Items were rated as deviant only if the subject provided evidence of severity and persistence of the problem, as manifested in his behaviour and verbal report.

A female psychiatrist employed by Juvenile Services to the Courts conducted the structured diagnostic interview with the subject. Three reliability raters, one female and two male research assistants, were trained to a criterion of 80% agreement for ratings on the IS prior to beginning data collection. Reliability was assessed for 158 (78%) of 204 interviewed males admitted to I.A.U. between September 1, 1987 and June 12, 1989. The kappa coefficients[3] for the reliability ratings of the three raters with the psychiatrist are summarized in Table 4. As can be seen, the levels of interrater agreement for the diagnosis of ADDH averaged 97% (kappa=.95) and for the diagnosis of CD averaged 100% (kappa=1). The levels of observed agreement for severity ratings of ADDH and of CD were 77% (kappa=.55), and 86% (kappa=.71), respectively.

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[3] Kappa is a "chance-corrected" measure of agreement ranging in value from 1 (complete agreement) to 0 (no agreement beyond chance) to a lower limit between 0 and -1 (less than chance agreement) (Hanley, 1987).

Table 4

Diagnostic Interrater Reliability

Diagnosis:	Rater 1	Rater 2	Rater 3
ADDH			
Kappa	.97	.96	.91
% Agreement	98	98	95
ADDH - Severity			
Kappa	.91	.26	.47
% Agreement	95	68	67
CD			
Kappa	1.00	1.00	1.00
% Agreement	100	100	100
CD - Severity			
Kappa	.65	.69	.78
% Agreement	91	86	81
Overall Diagnostic Agreement			
Kappa	.88	.90	.91
% Agreement	93	95	95

Note. Psychiatrist was constant.

A structured interview, based on a revised version of the IS, was administered by telephone to the parent(s) or guardian(s) of prospective subjects by a psychiatric social worker or one of the research assistants. The only changes in the parent version of the IS were that ADDH symptomatology was assessed retrospectively (when the patient was between the ages of 4 to 10 years), as well as currently, and symptom items were covered by questions which posed three alternatives to the parent (see Appendix B). Each parent or guardian was asked to compare his or her child to the average child his age and, for each question, to answer whether the behaviour was not true, was sometimes or somewhat true, or was very often or often true of the child. If the deviant alternative was selected, the interviewer then inquired as to how much of a problem the behaviour had posed for the family and the child. Again, symptom items were rated on a 4-point severity scale.

Given the predominance of a disrupted and chaotic family background in the youths in this sample, interview information with a parent or guardian was difficult to obtain and is available for only 18 subjects (54%). However, all successfully completed interviews corroborated an early onset of ADDH in the probands. When historical and current data from a parent or guardian were unavailable, medical, probation, and/or social worker reports were examined. This data revealed that the remaining subjects all exhibited core symptoms characteristic of ADDH in early or late childhood.

Furthermore, with respect to the diagnosis of ADDH, both the subject and the parent versions of the IS included questions which addressed the medical and psychiatric history of the subject (see Appendices A and B). In addition to currently meeting the DSM-III criteria for ADDH and CD, subjects included in the present study had to have a previously diagnosed childhood history of ADDH, (which may or may not have entailed treatment with MPH), and/or have met the operational criteria for childhood ADDH based on parental retrospective report or follow-back data. The present design, therefore, employed a combination of current, retrospective, and follow-back assessment methods for the diagnosis of ADDH in a sample of young offenders.

Chapter 2
Experimental Design

A triple-blind (subject, experimenter, and nursing staff), placebo-control, within-subject (crossover) experimental design was employed in which subjects received a 10-day trial of placebo and two doses of MPH in a randomly assigned, counterbalanced sequence. The median dose of the active drug was 0.3-mg/kg daily in the low dose condition and 0.5-mg/kg in the moderate dose condition. The study was broken into four 2-day phases (baseline, low dose, moderate dose, and placebo), with random orders and 1-day washouts between treatment phases. The treatment-order combinations were defined to include an equal number of all possible drug-dosage-placebo orders and subjects were randomly assigned to one of the 6 treatment orders. The experimental design is shown below.

PHASE 1:	BASELINE	
Days 1-2:	Subject adapts to the I.A.U.	
	Test battery was administered in the morning of day 2.	
PHASE 2:	TREATMENT (low/moderate dose/placebo)	PLACEBO
Days 3-4:	Drug side-effects were monitored by nursing staff.	
	Test battery was administered in the morning of day 4.	
Day 5:	WASHOUT	
PHASE 3:	TREATMENT (low/moderate dose/placebo)	LOW DOSE
Days 6-7:	Drug side-effects were monitored by nursing staff.	
	Test battery was administered in the morning of day 7.	
Day 8:	WASHOUT	

PHASE 4:	TREATMENT (low/moderate dose/placebo)	MOD DOSE
Days 9-10:	Drug side-effects were monitored by nursing staff.	
	Test battery was administered in the morning of day 10.	

Drug Treatment and Procedure

Prior to obtaining consent for participation in the study, the risks and benefits of stimulant medication were explained to the youth and his parent or guardian. An information letter explaining the rationale and procedures of the project were provided (see Appendix C). Informed and written consent were then obtained from both the adolescent and his parent or legal guardian (see Appendix D).

Methylphenidate hydrochloride (Ritalin) was prepared in indistinguishable gelatin capsules of 0.3-mg and 0.5-mg doses for body weights ranging from 50 kilograms to 80 kilograms. On days when subjects were assigned to active medication, they received the quantity of medication closest to a calculated dose of 0.3-mg/kg in the low dose condition and 0.5-mg/kg in the moderate dose condition. Placebo was prepared in capsules identical in size, shape, and colour to those containing MPH.

Capsules were sealed by a pharmacist in individually daily-dated envelopes to ensure accurate dose administration. Medication was administered by nursing staff, who were blind to the contents of the capsules, 30 minutes before the morning and noon meals, at 0800 and 1130 hours. During each experimental phase, the test battery was administered in the morning, 60 minutes following oral ingestion. The selection of a 60-minute interval was based on the assumption of plasma levels of MPH peaking around this point and remaining relatively stable for the ensuing 1 to 3 hours (Gualtieri, Wargin, Kanoy, Patrick, Shen, Youngblood, Mueller & Breese, 1982).

Treatment emergent (side) effects were monitored by nursing staff who completed the Conner's Side-Effects Questionnaire (SEQ) (Barkley, 1981) daily across the 3 treatment phases (see Appendix E). The SEQ includes 16 items of frequently observed short-term side-effects of stimulant drugs which are rated on a 9-point continuum from absent to serious. Retrospective ratings were completed at 1500 hours each day for the period of observations from 0830 to 1500 hours so as to correspond with the medication procedure described above.

A composite measure of side effects was derived for each treatment phase by summing the number of symptoms rated as present for 16 items relevant to MPH (sleep problems, nightmares, appetite loss, stomachaches, irritability, headaches, drowsiness, anxiety, euphoria, dizziness, biting nails, daydreams, social disinterest, unhappiness, crying, and reduced talk) (Ross & Ross, 1982), and averaged over the number of days per phase. This data, along with the number of subjects evidencing side effects during each treatment phase, are presented in Table 5. The most frequently reported side effects included irritability and decreased appetite, and the least frequently reported was nightmares.

Table 5

Side Effects Questionnaire

	Treatment Phase		
	Placebo	0.3-mg/kg	0.5-mg/kg
Average # of side effects ($n = 16$)	3.5	3.9	4.6
Number of subjects showing side effects ($n = 33$)	32	33	33

Subjects were withdrawn from the study under the following conditions: (1) if serious side-effects developed as monitored daily by nursing staff; (2) if the patient was prescribed any other drug(s) that was psychotropic; (3) if the subject, or parent, or guardian, withdrew his or her consent for participation in the study. Two subjects experienced side-effects during the moderate dose phase that warranted exclusion from the study. One subject experienced symptoms of decreased appetite, stomachaches, and drowsiness, whereas the second subject reported signs of euphoria, overactiveness, and feeling energized. Interestingly, it was later learned that the latter subject had abused Ritalin in the past.

Chapter 3
Test Battery

The experimental tasks consisted of three Gordon Diagnostic System (GDS), Inc. tasks which included a Vigilance Task (VT) based upon the CPT, a Distractibility Task (DT), and a Delay or Differential Reinforcement of Low Rate Responding (DRL) Task (Gordon, 1986). Reward dominance and passive avoidance learning were measured using a modified version of a computerized task developed by Newman and Kosson (1986).

Gordon Diagnostic System (GDS) Tasks

A microprocessor-based portable unit called the GDS, which allows for the administration of multiple game-like tasks, was used in the present study. The GDS was developed to permit standardized assessment of attentional and impulse control deficits. The GDS has been shown to accurately discriminate between groups classified as ADDH and as non-ADDH in outpatient clinic samples (Gordon, 1979), in a day treatment centre for severely emotionally disturbed children (McClure & Gordon, 1984), and in a school-referred population (Gordon & McClure, 1984). Research suggests that the performance differences observed between ADDH and non-ADDH groups on the GDS persist, regardless of age or IQ, and are stable over the duration of the tasks (McClure & Gordon, 1984). Data also indicate that the GDS tasks are sensitive to the effects of stimulant medication (Shue & Douglas, 1983) and do not show practice effects for repeated administrations (Gordon, 1986). Normative data on GDS performance have been established for a sample of 1,300 non-ADDH boys and girls aged 3 to 16. Test-retest reliability coefficients have been found to be satisfactory after both short and long intervals (i.e., a 30 to 45 day interval yielded correlations between .60 and .77 and a one year interval yielded correlations ranging from .52 to .94) (Gordon, 1986; Gordon & Mettelman, 1988).

Apparatus

The GDS unit is a one-button (coloured blue) solid-state console manufactured by Clinical Diagnostics, Inc. With this system, the subject played games with lights and numbers that showed up on the front electronic display of the console. Stimuli consisted of numbers presented in variable interval and block lengths according to standard parameters. The lengths of blocks (1-999 seconds) and presentation intervals (i.e., the time between a number appearing on the display and the presentation of the next digit), (1-99 seconds), varied depending on the task administered. Stimuli were presented at fixed intervals of 2 tenths of a second (i.e., 200 milliseconds).

An internal microprocessor generated the tasks and recorded quantitative features of the subject's performance for the duration of the task, as well as for the individual time blocks. The GDS response data selected for use in the present study included summary scores and error analyses.

Sustained Attention

The GDS Vigilance Task (VT) was used to assess the short-term effects of MPH on subjects' abilities to sustain attention and effort to a task over time. The VT measured performance over a 9-minute time block in the absence of feedback and required the subject to respond following the presentation of designated target stimuli presented at .2 second intervals on the electronic front display. The subject was required to press the button after a specific number, or pair of numbers, appeared on the screen. For example, the 1-9 mode of the VT required the subject to press the button every time the number 1 was immediately followed by the number 9. The "1" served as the alerting stimulus, which prepared the subject to respond, and the "9" acted as a target stimulus to which the subject was to respond only after having been alerted. The 3-5 mode of the VT, where a

target stimulus "5" immediately followed an alerting stimulus "3", was also used in the study. The sequence of digits presented for each task mode was fixed. There were 15 "hot" stimuli (i.e., 1-9 or 3-5) presentations in each of the three, 3-minute time blocks.

The VT was described to subjects as a computer game in which the object was to press the blue button when certain numbers appeared in a particular order. The following instructions were based on the standard GDS instructions as outlined in the *GDS User's Manual* (Gordon, 1986) and were presented to subjects prior to the administration of the task on each of the four experimental sessions.

> In this computer game, you will see numbers flash quickly on the display, and I want you to press the blue button on the machine every time you see the number 9 come right after the number 1 (i.e., for the administration of the 1-9 mode). If the number 9 comes right after any other number, do not press the button. The only time that you should press the button is if you see a 9 that comes right after a 1. Now the red light on the machine won't go on at all, but at the end of the game, I will tell you how many points you've won. You will know when the game is over when this green light comes on. Do you understand?

The instructions were identical for the 3-5 mode of the VT except that subjects were told that the target number was a "5" when it appeared immediately after the number "3".

The two 1-9 and 3-5 modes of the VT were used in the current study and were administered in alternate orders. Therefore, no subject received the 1-9 mode or the 3-5 mode for any two consecutive task administrations. Subjects were assigned randomly to the different task-order combinations. Dependent variables for the VT were the number of omission errors (i.e., the number of misses or times the subject did not press the button upon the appearance of a correct number pair) and the number of commission errors (i.e., the number of extraneous button presses or false alarms).

Distractibility

The ability to focus on and correctly respond to critical target stimuli in the presence of extraneous, or potentially distracting, stimuli was measured with the GDS Distractibility Task (DT). The design of the DT introduced distractors that were embedded within the stimulus array of the task so as to address methodological concerns raised in the literature (McMahon, 1984; Rosenthal & Allen, 1978). The term 'distractors' refers in fast process research to non-target stimuli that require processing time in the limited attention capacity system (Shiffrin & Schneider, 1977). The DT was essentially identical to the VT except that random digits, or visual distractors, flashed at random intervals on the outer two positions of the electronic front display. The subject was still required to press the button when a designated target stimulus followed a designated alerting stimulus. The difference was, however, that distractor digits flashed on either side of the center of the relevant digit.

As with the VT, the two 1-9 and 3-5 modes were administered in alternate orders across the four experimental conditions. Similarly, the DT was described to subjects as a computer game in which the object of the game was to press the button when designated target numbers appeared in a particular order on the display. The dependent variables under study were the number of omission errors (misses) and commission errors (false alarms).

Inhibition of Impulsive Responding

The GDS Delay Task (DRL - Delayed Response Learning) was used as a measure of subjects' abilities to suppress or delay impulsive behavioural responses. The task requires minimal use of sustained attention skills; rather, it primarily places demands on subjects' abilities to delay or to refrain from emitting non-reinforced responses. The DRL task is

based on a delayed response (6 second) learning operant schedule and requires the subject to inhibit responding in order to gain a reward, or to elicit positive feedback.

Numerical stimuli were presented for .2 second intervals and subjects were required to determine the minimum time to refrain from responding in order to win a point. Specifically, subjects were instructed to press the button, wait, and then press it again in order to win a point; however, if they pressed it prematurely (sooner than 6 seconds), no reward would be obtained. Feedback was provided via a flashing light on the front of the console and a reward counter which incremented when the subject refrained from responding for the 6 second interval. If the subject responded before the set interval elapsed, the timer reset and no points were recorded on the reward counter. DRL task performance was recorded for four successive 2-minute time blocks. Subjects were not shown when one time block ended and another began.

As the duration of the interval was not mentioned in the instructions to subjects, they were required to develop a method of determining the minimum time needed to refrain from responding in order to win a point. The DRL task required that subjects efficiently utilize the feedback provided by the GDS to guide their responses; they had to develop a strategy for estimating the interresponse interval, as well as refraining from responding until the set interval had elapsed.

The following instructions, as outlined in the *GDS User's Manual*, were presented to subjects on each of the four task administrations.

> In this computer game, you will get a chance to win alot of points, not just 1 or 2 points, but a whole bunch. Every time that you see this red light go on, you'll earn a point and this counter will keep track of how many points that you've won. At the end of the game, we will see how many points you've earned. Now, to make the light go on, all that you have to do is press the blue button and wait a little while, then press it again. If you press the button too soon, though, you will have to wait a while before you can press it to get another point. But if you press the button, wait awhile, then press it again, you'll earn a point every time.

The measure of performance for the DRL task was the percentage of correct responses (the number of correct responses divided by the total number of responses), an efficiency ratio. This represents the percentage of times the subject pressed the button after having successfully waited the 6 second time interval, and is an indicator of impulsivity, or inhibitory control.

Reward Dominance and Passive Avoidance Learning (RDPA) Task

Apparatus

Reward dominance and passive avoidance (RDPA) learning under a continuous reinforcement schedule were measured using a computerized task developed and previously used by Newman and Kosson (1986) in delinquent (Newman et al., 1985) and psychopathic (Newman & Kosson, 1986) samples. The experimental task was conducted using an Apple II Plus computer, a 13-inch monitor, and a hand-held response button. The response switch consisted of a conical plastic box (65 mm by 30 mm) with a single push button on the top of the surface of the box. Auditory feedback was provided to the subject via a small enclosed speaker connected to the computer. The software written by Newman and Kosson (1986) was modified to permit multiple task administrations.

Paradigm

Reward dominance and passive avoidance learning were assessed using a paradigm that provided monetary rewards for responses to positive, or correct, stimuli (S+'s) and monetary punishments (i.e., loss of reward, or response-cost) for responses to negative, or incorrect, stimuli (S-'s). Two versions of a go/no-go discrimination learning task, in which subjects were required to respond to S+'s and and to withhold responses to S-'s, were

administered across the four experimental phases. Subjects received auditory, visual, and tangible reinforcements under both task conditions.

In the reward + punishment (R + P) condition, subjects were provided with the competing goals of avoiding monetary punishment while earning monetary rewards. Subjects earned rewards for responding to S+'s and received punishments for responding to S-'s. Subjects were given 10 chips (each chip worth five cents) prior to starting the task. Responses to correct stimuli (S+'s) were reinforced by the presentation of a moderately pitched tone (625 Hz as estimated by a Tectronix 475a oscilloscope), the visual presentation of the word "CORRECT" on a 13-inch Apple ll Plus computer monitor screen, and the experimenter adding a chip to the subject's pile of earnings. Under the R + P condition, no punishment or computer feedback was provided for failure to respond to S+'s. When subjects incorrectly responded to S-'s, a lower-pitched, non-aversive tone (148 Hz) sounded, the word "WRONG" appeared on the monitor screen, and the experimenter removed a chip from the pile of earnings.

In the punishment only (P-only) condition, subjects were provided with only punishment incentives. Feedback was provided for failure to respond to S+'s and for incorrectly responding to S-'s; the low-pitched tone sounded, "WRONG" appeared on the monitor screen, and the experimenter withdrew a chip. Subjects began with 40 chips in condition P-only and could not earn additional money.

Stimuli consisted of 8 different, 2-digit numbers repeated 10 times in different, randomized orders for a total of 80 trials per condition. Numbers ranged from 01 to 99. Four of the 8 stimuli were S+'s (stimuli paired with reward) and 4 were S-'s (stimuli paired with punishment), and were evenly divided with regard to the attributes of above versus below 50 and even versus odd. Numbers were further selected on the condition that no attribute of a number (e.g., is a multiple of 7) be associated differentially with either reward

or punishment. Eight different sets of 8 stimulus numbers were used so that a stimulus set appeared only once in either a R + P or P-only condition across the four task administrations.

Each number stimulus was presented on the monitor as a green light on a dark background and measured 5.1 cm by 2.5 cm in size. Stimuli were presented for 3-second intervals or until subjects responded. The interstimulus interval was 1 second. This task took approximately 15 minutes to complete.

Procedure

Subjects were instructed to learn by trial and error when to respond (by pressing a hand-held response button) and when not to respond. The nature of the task and the two varying conditions were explained to subjects. Practice trials, consisting of 4 stimuli repeated 4 times in a randomized order for a total of 16 trials, were presented to subjects prior to starting the task for each task administration. Total winnings were provided to subjects at the end of the fourth experimental session.

Subjects received the following instructions prior to administration of the task.

> This is the task we call the Computer-Chip Game. In this experiment, we will be working with the computer and we will be using chips that will later be exchanged for money. Each chip is worth 5 cents. However, you will be playing this computer game for a total of 8 times during the next 10 days, therefore you will be able to accumulate a total of 15 dollars winnings depending upon your performance. So each chip lost or won, though only worth 5 cents, will count in the end.
>
> You will also be playing the Computer-Chip Game under two different sets of conditions. In one condition, you will win chips for correct responses or correct answers, and you will lose money or chips for wrong answers. In this condition, you will start off with 10 chips. In another condition, you will begin with 40 chips, and you will lose chips for incorrect responses. So you see, in one condition you can win or lose depending upon your performance, in the other condition, you only lose money if you make mistakes.

> In this game, the computer will be flashing a series of numbers on the screen. Each number will come on for only 3 seconds and then disappear. However, you will see the same numbers over and over again during the game. Each time that a number appears, you have to decide whether you are going to press the button. The object of the game is that you must figure out when to press the button and when not to press the button.

In the R + P condition, subjects were told the following:

> Sometimes when you press the button, I will give you a chip. Other times when you press the button, if you were wrong, I will take a chip away. You won't win or lose any money when you don't press the button. The only time you win or lose is when you press the button.

In the P-only condition, the instructions to subjects included the following:

> Sometimes when you press the button, if you were wrong, I will take a chip away. Other times when you don't press the button and you should have pressed the button, I will take a chip away. You won't lose any money as long as you are right about when to press the button and when not to press the button.

The nature of the task was fully explained to subjects. The experimenter completed a sample trial with the subject and demonstrated how to determine when to press and when not to press the response button. The above instructions, with modifications such as "You remember last time....." to accomodate subjects' repeated exposures to the task, were given prior to each of the four task administrations.

Order of condition (R + P and P-Only) was randomized between subjects and within subjects, and subjects were randomly assigned to the different condition-number set orders. Dependent measures included the number of omission errors (failure to respond to S+'s) and the number of commission, or passive avoidance, errors (failure to inhibit responses to S-'s).

Test Battery Procedure

The test battery was administered in the morning of the second day of each experimental phase from 0900 to 1030 hours. The testing sessions took place in a research room located in a separate area from the inpatient unit, on the upstairs floor of the I.A.U.

Subjects were tested individually. Over the course of the study, a male and a female research assistant individually administered the test battery. Each subject was tested by the same experimenter for each of the four testing sessions. Therefore, subjects were nested and not crossed with sex of experimenter.

During testing, the experimenter sat behind and off to the side of the subject to reduce the possibility of distraction, and conversation was discouraged. At the conclusion of each testing session, subjects were informed of their total earnings. Total earnings won over the four testing sessions were paid to subjects upon their release from I.A.U.

All possible test-order combinations for the test battery were calculated prior to data collection. Test-order was randomized between subjects, but not within subjects. For example, one subject may have received the tasks in the order of ABCDE across the four experimental phases, whereas another subject may have received the tasks in the order of BECAD across the four phases. Subjects were assigned randomly to the test-order combinations.

Upon completion of the study, the subject and his parent or guardian received feedback as to the youth's performance on the test measures and his drug treatment response. A recommendation for pharmacological treatment, contingent on the observed drug response during the study, was included in the psychiatrist's report to the courts.

PART C

RESULTS

Chapter 1
Treatment Effects

Data analysis was carried out using BMDP Statistical Software programmes (University of California Press, 1983). Multivariate analyses of variance (MANOVAs) were conducted to examine differences between treatment conditions and to investigate the possible influences of counterbalancing variables and selected variables. Subjects with missing data for a given measure were excluded from statistical analyses of that measure. Univariate analyses of variance (ANOVAs) were computed to determine which variables contributed to significant multivariate effects. Probability levels for univariate analyses of variance were adjusted by the Huynh-Feldt correction and a significance level of .05 was adopted. As a follow-up to significant ANOVAs, a step-wise Bonferroni procedure (Hays, 1988) was used to protect against family-wise Type I error for analyses of differences between means.

Counterbalancing Variables

A MANOVA using one between-group (drug order) and two within-group (dependent measure and experimental phase) factors was performed on the GDS task data. The multivariate and univariate tests of the main effects and the interaction effects of the Drug Order grouping variable did not reach significance. Similarly, a multivariate analysis of the Reward Dominance-Passive Avoidance (RDPA) data, with one between-group factor (drug order) and three within-group factors (dependent measure, experimental phase, and reward condition), did not yield significant main effects or interactions for the Drug Order factor. These findings indicate that the dependent measures were not modified by the administration sequence of the active and inactive drug treatments. A further MANOVA of the RDPA data, using task order as the between-group factor, revealed no significant main effects or interactions for this variable. Appendices F, G, and H present the sources

of variance and the *F*-values and *p*-values for the tests of the Drug Order and Task Order factors.

Sex of Experimenter

The MANOVAs calculated on the GDS and RDPA data, using Sex of Experimenter as the between-subjects variable, did not yield significant multivariate *F*-values for the main effects or interactions for this grouping variable. The *F*- and *p*-values, and the sources of variance, for the Experimenter variable can be found in Appendices I and J.

Treatment Effects

GDS Tasks: Vigilance (VT), Distractibility (DT), and Delay (DRL) Tasks

The one-between and two-within factor MANOVA calculated for the GDS task data, using Drug Order as the grouping variable, failed to yield a significant multivariate main effect for the treatment phases. However, subsequent ANOVAs with repeated measures, using a Bonferroni probability set at .01 for the five analyses, revealed a significant treatment phase effect for the Efficiency Ratio variable of the DRL task ($F(3,66) = 6.63$, $p < .001$). In addition, the univariate analysis of the treatment phase effect for the Omission Error measure of the DT approached significance ($F(3,66) = 3.08$, $p < .05$). The means of the remaining nonsignificant ANOVAs (VT - Omission Errors and Commission Errors, DT - Commission Errors) were comparatively low and yielded little variability across experimental phases. These results suggest the possibility of floor effects, as few errors were made by subjects on these particular dependent measures. (See the subsequent section on Baseline Performance and GDS norms). Further, the GDS measures for the VT and the DT showed considerable between-subject variability, thus reducing the variance attributable to MPH and reducing the measures' sensitivities to treatment-related changes.

Table 6 presents the univariate main effects of the experimental conditions as expressed in *F*- and *p*-values, and Appendix K presents the sources of variance for these effects. The means and standard deviations of the dependent measures for the GDS tasks are presented across experimental conditions in Table 7.

Table 6

Univariate Main Effects of Treatment Phases

Dependent Variable	df	*F*-value	*p*
Vigilance Task:			
Omission Errors	3,66	0.36	.783
Commission Errors	3,66	0.95	.411
Distractibility Task:			
Omission Errors	3,66	3.08	.033
Commission Errors	3,66	0.85	.471
Delay (DRL) Task:			
Efficiency Ratio	3,66	6.63	.001
Passive Avoidance Task:			
Omission Errors	3,72	6.00	.002
Commission Errors	3,72	8.00	.001

Analyses of differences between means were conducted for the GDS dependent variables that yielded significant or near-significant *F*-values, using a Bonferroni probability set at .008 based on a .05 criterion per family of pairwise comparisons. Table 8 presents the *p*-values for these contrasts. Findings revealed that the mean number of omission errors on the DT and the mean efficiency ratio scores on the DRL task were significantly enhanced in the two active drug phase conditions relative to the baseline condition. Pairwise comparisons of the placebo and baseline conditions were significant for only the

Table 7

Means and Standard Deviations of Dependent Measures by Experimental Conditions

Dependent Variable	Baseline		Placebo		0.3-mg/kg		0.5-mg/kg	
	M	*(SD)*	*M*	*(SD)*	*M*	*(SD)*	*M*	*(SD)*
Vigilance Task:								
Omission Errors	2.92	(3.42)	2.42	(2.89)	2.10	(3.54)	1.81	(1.79)
Commission Errors	3.00	(3.99)	2.53	(4.46)	2.14	(3.82)	2.74	(4.16)
Distractibility Task:								
Omission Errors	9.26	(9.46)	6.73	(8.45)	5.82	(9.55)	4.17	(5.20)
Commission Errors	3.23	(2.99)	3.89	(5.71)	2.53	(4.34)	2.82	(4.85)
Delay (DRL) Task:								
Efficiency Ratio	0.69	(0.24)	0.87	(0.13)	0.86	(0.14)	0.86	(0.16)
Passive Avoidance Task:								
Omission Errors	14.37	(8.98)	10.17	(8.60)	10.33	(8.70)	10.52	(9.06)
Commission Errors	7.18	(8.79)	3.83	(4.09)	3.60	(2.61)	3.03	(2.48)
Passive Avoidance R + P:								
Omission Errors	13.33	(8.56)	9.89	(9.38)	11.41	(9.47)	10.85	(8.93)
Commission Errors	6.41	(3.64)	4.70	(4.92)	3.56	(3.76)	3.14	(3.91)
Passive Avoidance P-only:								
Omission Errors	15.41	(9.42)	10.44	(10.18)	9.25	(9.70)	10.19	(9.33)
Commission Errors	7.96	(11.96)	2.96	(2.89)	3.59	(3.35)	2.92	(2.96)

Note. Efficiency Ratio - higher scores indicate improved performance
R + P = Condition reward & punishment
P-only = Condition punishment only

Table 8

P-Values for Pairwise Comparisons Between Experimental Conditions

Dependent Variable	Placebo vs. Baseline	0.3-mg/kg vs. Baseline	0.5-mg/kg vs. Baseline	Placebo vs. 0.3-mg/kg	Placebo vs. 0.5-mg/kg	0.3-mg/kg vs. 0.5-mg/kg
Distractibility Task:						
Omission Errors	.031	.007*	.002*	.480	.069	.272
Delay (DRL) Task:						
Efficiency Ratio	.001*	.0006*	.001*	.925	.926	.928
Passive Avoidance Task:						
Omission Errors	.007*	.004*	.004*	.839	.670	.662
Commission Errors	.008*	.008*	.003*	.766	.255	.409

*Contrasts significant at the Bonferroni $p < .008$

efficiency ratio scores on the DRL task. For both variables, no significant differences were observed between the active and the inactive drug conditions, as well as between the two active drug conditions. These findings indicate that MPH moderately improved subjects' abilities to make correct detections on the DT relative to baseline but not to placebo. Similarly, the MPH-related improvement in behavioural response inhibition on the DRL task involved change above baseline but not above placebo levels.

Reward Dominance and Passive Avoidance (RDPA) Task

For this task, the first block of 8 trials was excluded from analyses because subjects' performance could not reflect learning until each stimulus number had been viewed at least once.

Table 7 presents the means and standard deviations of the dependent measures for the RDPA task across experimental conditions, type of reward programme, and averaged over the two reward programmes. A MANOVA of this data, using Drug Order as the between-subjects factor, yielded a significant multivariate main effect for treatment phase ($F(6,19) = 3.65$, $p < .025$). The univariate tests were significant for both the Omission Error ($F(3,72) = 6.00$, $p < .005$) and Commission Error ($F(3,72) = 8.00$, $p < .001$) measures. The multivariate and univariate tests of the main effects for type of Reward Programme (R + P, P-only), and the programme x treatment phase interaction, did not reach significance. Table 6 presents the univariate main effects of the experimental conditions as expressed in *F*- and *p*-values, and Appendix K presents the sources of variance for these effects. The sources of variance, and the *F*- and *p*-values for the main effects and the interaction effects of the Reward Programme factor, can be found in Appendix L.

The p-values for pairwise comparisons for variables that yielded significance are shown in Table 8. Comparisons of the number of omission and commission errors across treatment phases indicated a significant drug response for both the low dose and the moderate dose drug conditions relative to the baseline phase. Hence, subjects' detection errors and passive avoidance errors were significantly reduced in the two active drug conditions relative to the baseline phase. However, a placebo response was also obtained, wherein subjects' numbers of omission and commission errors were also significantly reduced during the placebo trial when compared to baseline levels. As with the GDS task data, significant differences were not obtained between the two dosage levels, nor between the placebo trial and the two drug conditions.

Analysis of Overall Drug Effects

The presence of an overall drug effect, which refers to change beyond placebo levels, was analyzed using the average of the two drug conditions with the placebo condition serving as a control. Change scores (the difference between the placebo score and the average of the low dose and moderate dose drug condition scores) were derived for each dependent variable. Contrary to expectation, the separate MANOVAs calculated for the GDS and RDPA data, using Drug Order as the grouping factor, failed to yield significant multivariate or univariate main effects for the treatment phases. See Appendix M for the sources of variance and the F- and p-values for these effects.

Positive Responder/Non-Responder Status

A principal component analysis, with varimax rotation, of the drug effect change score data for both the GDS and RDPA tasks was performed as a means of identifying a homogeneous favourable drug responder subgroup. Four factors were obtained that showed a spread of eigenvalues and no dominant eigenvalue. The notion of a homogeneous drug

responder or non-responder subgroup implies the presence of a dominant eigenvalue and this was not observed. Details of the principal component analysis are presented in Appendix N.

Chapter 2
Subject Grouping Variables And Baseline Performances

Additional planned multivariate analyses were conducted on the battery of repeated tasks to provide information regarding the possible role that diagnostic severity and a formerly diagnosed, versus a retrospectively diagnosed, childhood history of ADDH may have played in subjects' responses to the treatment protocol. Separate MANOVAs were conducted for the GDS tasks and for the RDPA measure, using either severity of ADDH (moderate/severe), severity of CD (moderate/severe), or prior diagnosis of a childhood history of ADDH (yes/no) as the between-group variable. In view of the number of analyses conducted overall and the associated problem of an increase in familywise error, the following MANOVAs were conducted using a Bonferroni probability set at .008 for the multivariate F-values based on a .05 criterion for the family of 6 analyses.

Severity of ADDH

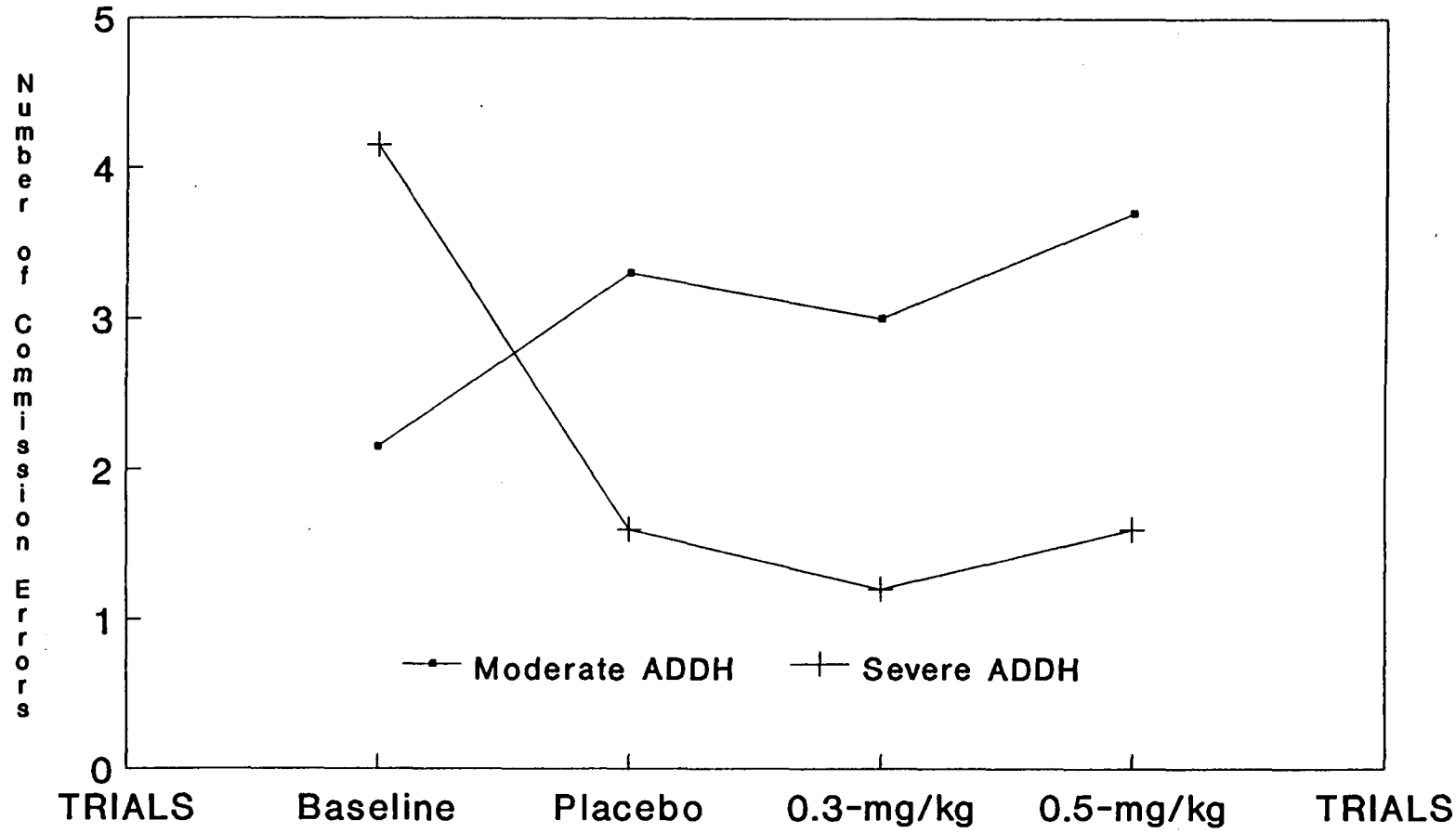
A one-between (severity of ADDH) and two-within (dependent measure and experimental phase) factor MANOVA calculated for the GDS data (on 25 of 33 subjects) did not yield significant multivariate or univariate main effects for the ADDH Severity factor. Similarly, the multivariate test of the experimental phase x ADDH Severity interaction did not approach significance. However, subsequent ANOVAs with repeated measures revealed interaction effects that approached significance for the Commission Error measure of the VT ($F(3,21) = 3.20$, $p < .05$), and for the Efficiency Ratio measure of the DRL task ($F(3,21) = 2.74$, $p < .05$). Contrasts between means for these interactions failed to yield significant p-values after the Bonferroni correction for family-wise error was applied. See Appendices O and P for the sources of variance and the F- and p-values for the tests of the ADDH Severity factor.

For descriptive purposes, the mean performances of the Moderate ADDH (n=13) and Severe ADDH (n=12) subgroups on the Commission Error measure of the VT and the Efficiency Ratio measure of the DRL task are presented in Figures 1 and 2. Compared to the performance of the Moderate ADDH subjects on the VT, subjects rated as Severely ADDH scored more commission errors in the baseline condition and showed greater improvement across the inactive and the active drug conditions. In contrast, Moderate ADDH subjects showed a tendency toward a deterioration in performance across the treatment phases. On the DRL task, the mean baseline performance of the Severe ADDH subjects was marginally superior to that of the Moderate ADDH subjects, with the greatest improvement in the Severe ADDH subgroup occurring in the low dose drug phase. Conversely, the Moderate ADDH subjects showed the greatest improvement in the placebo and moderate dose drug phases. Both groups showed a tendency toward improvement across the inactive and active drug treatment phases.

The MANOVA of the RDPA data (calculated on 24 of 33 subjects), with one-between (severity of ADDH) and three-within (dependent measure, experimental phase, reward programme) subject factors, revealed no significant multivariate F-values, and univariate F-values, for the tests of the main effects and the two-way and three-way interactions of the ADDH Severity factor. Appendices O and P show the sources of variance and the F- and p-values for these tests.

However, subsequent ANOVAs of the Reward Programme x the ADDH Severity factor revealed an interaction effect which approached significance for the Omission Error measure ($F(1,23) = 4.13$, $p < .06$). Analyses of differences between means for this interaction did not reach significance when the appropriate correction for family-wise error was applied. As shown in Figure 3, the Severe ADDH subgroup (n=11) scored more omission errors than the Moderate ADDH subgroup (n=13) under both reward conditions. However, the

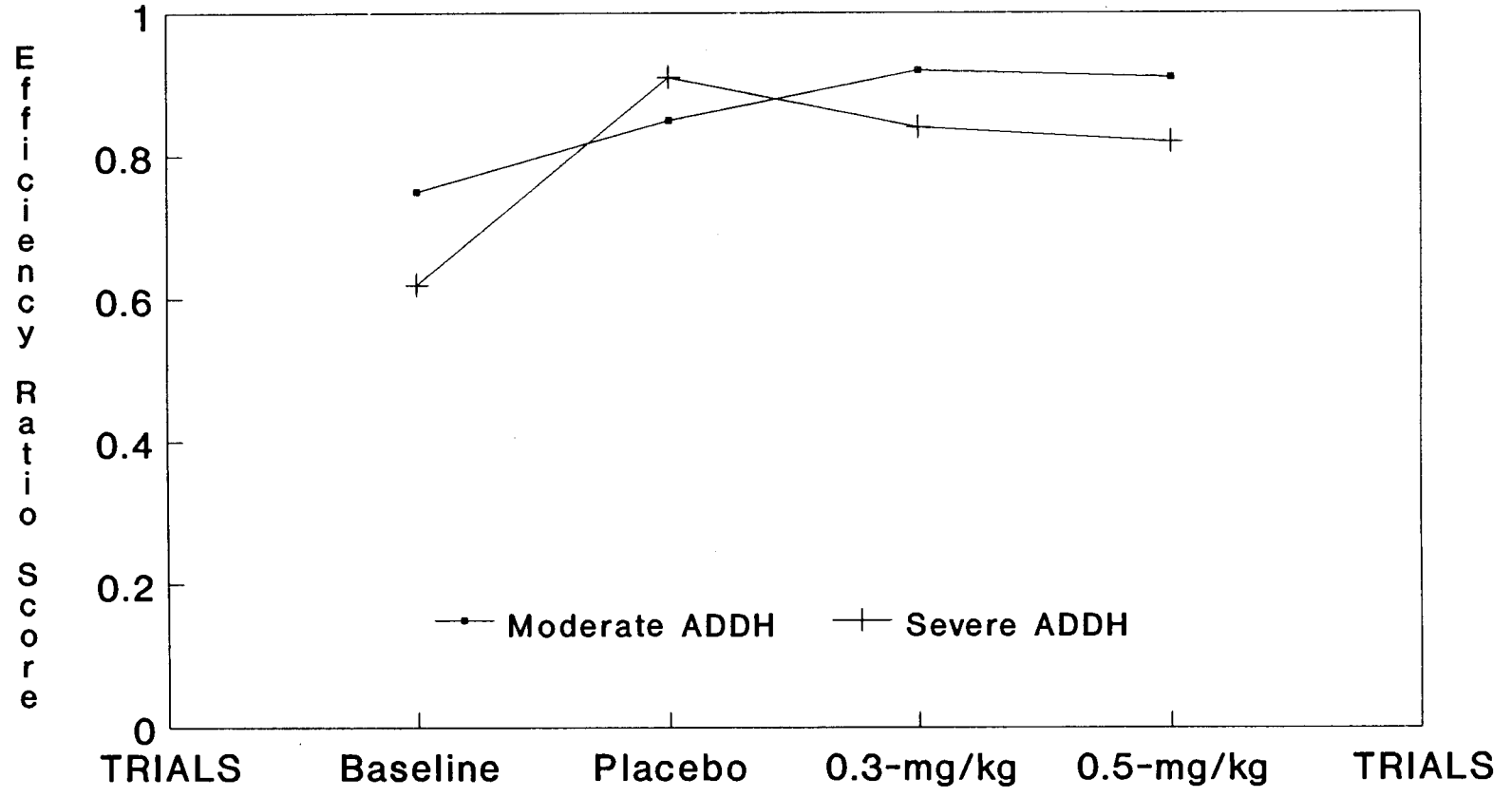
Figure 1
VT: Mean Number of Commission Errors as a Function of Severity of ADDH



Moderate ADDH *n* = 13
Severe ADDH *n* = 12

Figure 2

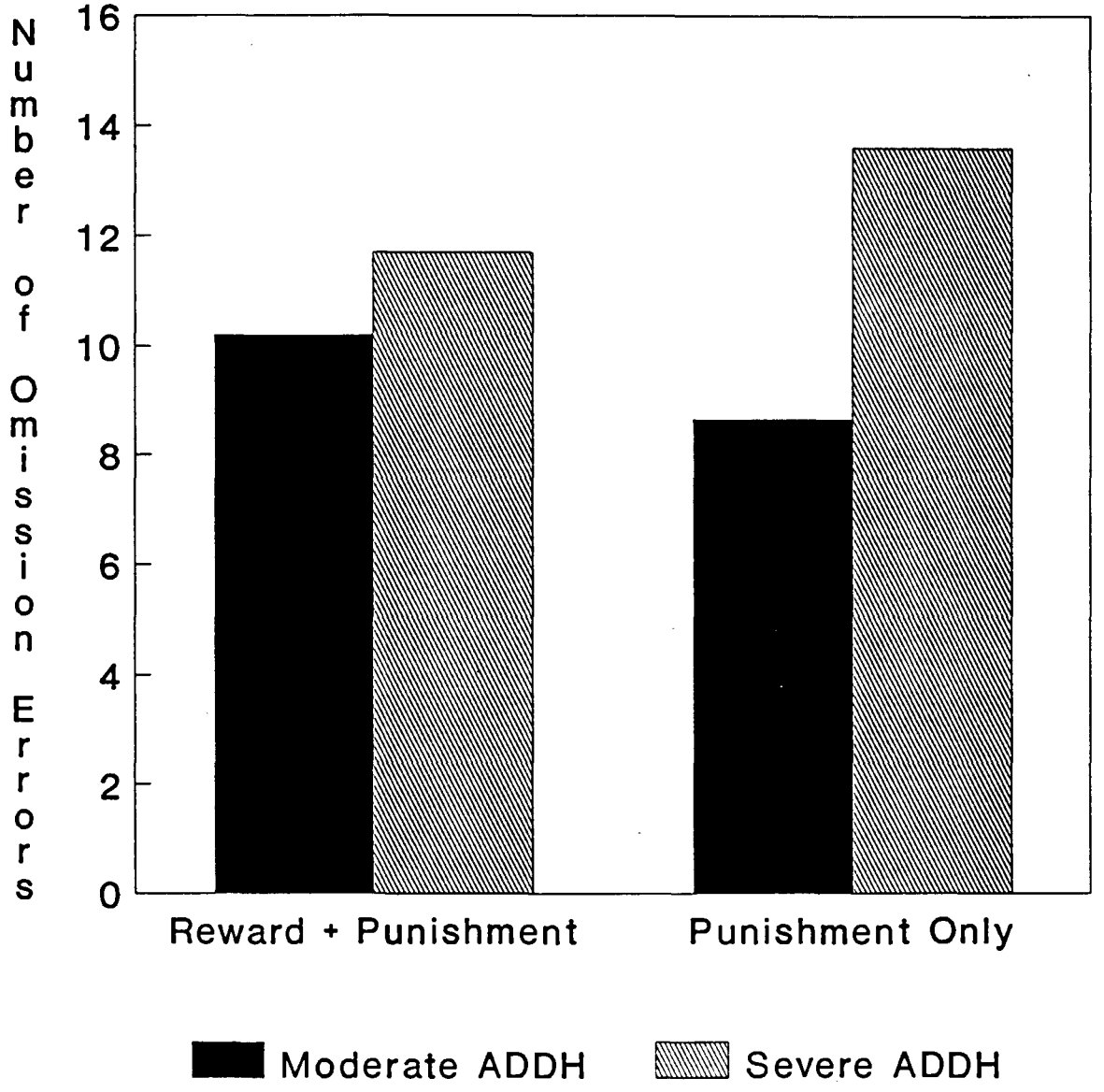
DRL Task: Mean Efficiency Ratio Scores as a Function of Severity of ADDH



Moderate ADDH *n* = 13
Severe ADDH *n* = 12

Figure 3

RDPA Task: Omission Errors as a Function of Severity of ADDH X Reward Programme



Moderate ADDH *n* = 13
Severe ADDH *n* = 11

mean omission error performance of the Severe ADDH subjects more closely approximated that of the Moderate ADDH subjects in the R + P condition, but not in the P-only condition. The Severe ADDH subgroup also showed a tendency to score more omission errors in the P-only condition when contrasted with their mean performance in the R + P condition. Taken together, these findings indicate that the Severe ADDH subgroup demonstrated a relative increase in attentiveness for correct responses (S+'s) under conditions of mixed R + P incentives, when compared to their performance under the P-only contingency. Conversely, the Moderate ADDH subjects evidenced marginally superior performance in detection (omission errors) in the P-only condition relative to the R + P condition.

Severity of CD

The MANOVAs (based on an n of 26) calculated for the GDS and RDPA data, using Severity of CD as the between-subject factor, did not yield significant multivariate or univariate main effects or interactions for this subject grouping variable. See Appendices Q and R for the tests of these effects.

Childhood History of ADDH

The MANOVA of the GDS data (calculated on 27 of 33 subjects), using presence of a previously diagnosed versus a retrospectively diagnosed childhood history of ADDH as the between-group factor, failed to yield significant multivariate and univariate F-values for the tests of this subject grouping variable. The sources of variance and the F- and p-values for these tests can be found in Appendices S and T.

Follow-up ANOVAs of the main effects for the childhood history subject grouping variable revealed an effect which approached significance for the Omission Error measure of the DT

($F(1,25) = 4.68$, $p < .05$). Figure 4 illustrates this main effect, where subjects found to have a formerly diagnosed childhood history of ADDH (n=8) scored twice as many omission errors on the DT when compared to subjects who were retrospectively diagnosed with a childhood history of the disorder (n=19).

Interestingly, the ADDH childhood history grouping variable served to significantly reduce the error variance for the multivariate and univariate tests of the main effect for treatment phase. A significant multivariate main effect ($F(15,11) = 3.25$, $p < .025$) and univariate main effects for the Omission Error measure of the DT ($F(3,75) = p < .02$) and the Efficiency Ratio measure of the DRL task ($F(3,75) = p < .001$) were now obtained[4].

The MANOVA of the RDPA data (calculated on 26 of 33 subjects) did not reveal significant multivariate or univariate tests for the main effects or interaction effects of the childhood history subject grouping variable. The data for these analyses are shown in Appendices S and T.

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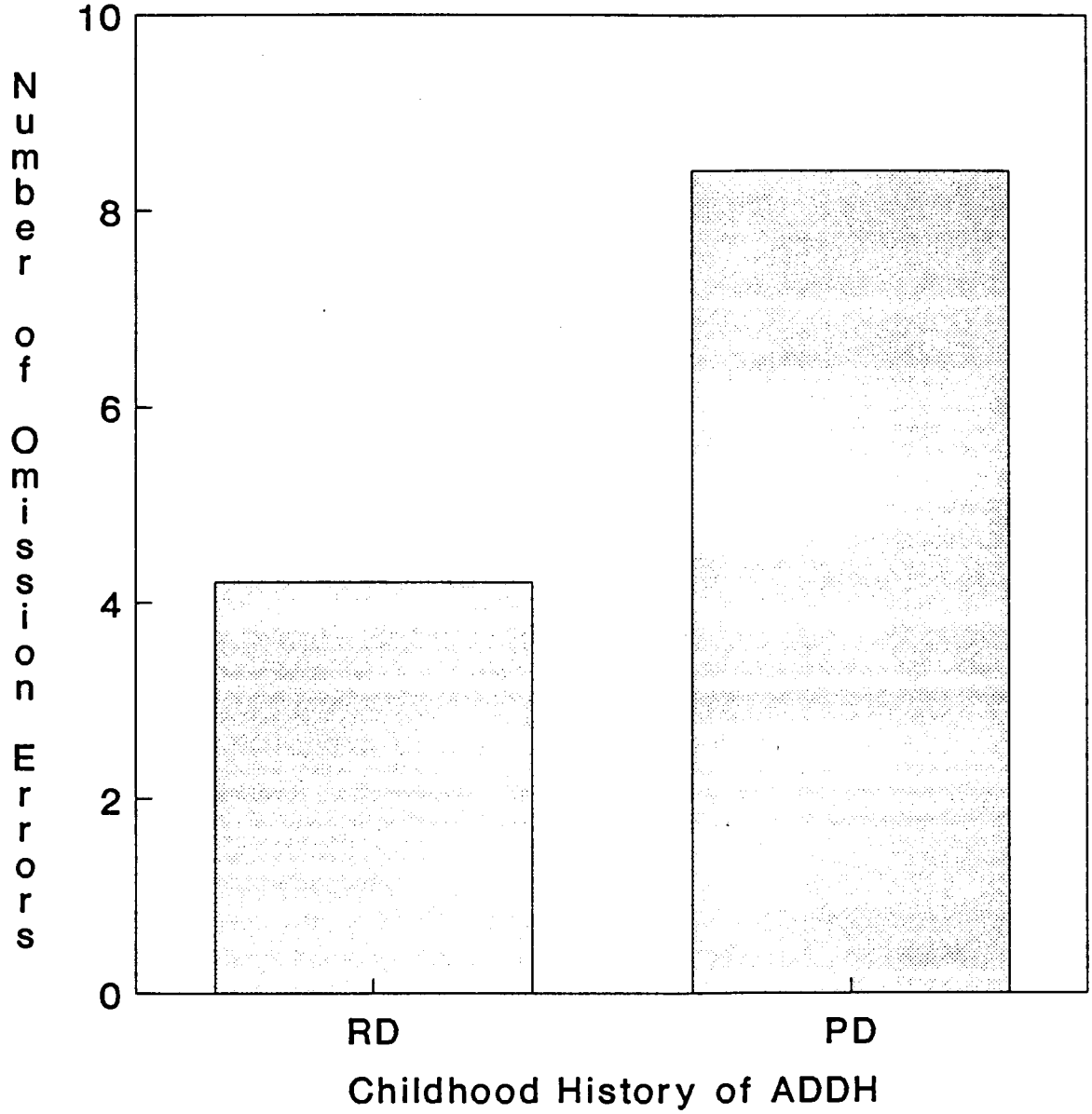
[4] This refers to significance at the .05 criterion, not at the Bonferroni probability level of .008 for the above family of 6 analyses.

Figure 4

DT: Mean Number of Omission Errors as a Function of Prior Diagnosis of ADDH



RD = Retrospectively Diagnosed *n* = 19
PD = Prior Diagnosis *n* = 8

Baseline Performances

GDS Tasks

Table 9 presents the mean baseline performances of subjects on the GDS tasks along with the norms for ADHD and non-ADHD 12 to 16 year-olds obtained by Gordon (1986). The mean performances of subjects in the present study on the VT and the DT fell within the normal and borderline ranges of Gordon's norms, whereas subjects' mean performance on the DRL task was closer to the performance of hyperactive adolescents (labelled abnormal in Table 9) in the Gordon (1986) sample.

Table 9

Baseline Performances Compared to Threshold Norms for GDS Tasks

GDS Tasks	Baseline *M*	(*SD*)	Abnormal	Borderline	Normal
Vigilance Task:[a]					
Omission Errors	2.92	(3.42)	> 5	3 - 5	< 3
Commission Errors	3.00	(3.99)	> 10	4 - 10	< 4
Distractibility Task:[b]					
Omission Errors	9.26	(9.46)	> 20	12 - 20	< 12
Commission Errors	3.23	(2.99)	> 8	4 - 8	< 4
Delay (DRL) Task:[c]					
Efficiency Ratio	0.69	(0.24)	0 - 65	.66 - .82	.83 - 1.00

Note. Scores classified as ABNORMAL were observed in less than 5% of the normal controls (i.e., the 5th percentile or less).

Scores classified as BORDERLINE were observed in 6% to 25% of the normal controls.

Scores classified as NORMAL were observed in over 25% of the normal controls.

a Norms: age = 12 to 16 years (N=218).
b Norms: age = 12 to 16 years (N=82).
c Norms: age = 12 to 16 years (N=240).

Reward Dominance and Passive Avoidance (RDPA) Task

The mean baseline performances of subjects on the two reward conditions (R + P and P-only) of the RDPA task are presented along with the mean performances of adolescent psychopathic offenders, adolescent delinquents, adult psychopaths, and adolescent and adult non-psychopathic controls (Kosson et al., 1990; Newman et al., 1985, 1986; Scerbo et al., 1990) in Table 10. The diagnostic criteria and relevant demographic characteristics of the samples are also presented in Table 10. Although the age and diagnostic criteria for subject selection in the reported samples differed from that of the present study, non-statistical comparisons revealed that delinquents, psychopathic inmates, and non-psychopathic inmate controls consistently made more commission (or passive avoidance) errors than omission errors across the two reward conditions, whereas the ADDH/CD adolescents in the current study consistently made more omission than commission errors across the reward conditions. A post hoc MANOVA of subjects' baseline performances on the RDPA task, using task order as the between-subjects factor and dependent measure and reward programme as the within-group factors, failed to reveal significant differences in performance under the two reward conditions ($F(2,31) = 0.73$, $p < 0.5$).

Table 10

Reward Dominance-Passive Avoidance Task Baseline Performance Compared to Samples in Other Studies[a]

		Reward & Punishment		Punishment Only	
		OE	CE	OE	CE
Present Study - ADDH/CD Baseline	*M*	13.33	6.41	15.41	7.96
	(SD)	(8.56)	(3.64)	(9.42)	(11.96)
White Adult Psychopaths[b]	*M*	6.73	13.80	9.60	14.33
	(SD)	(4.86)	(6.43)	(6.97)	(7.99)
White Adult Offender Controls	*M*	8.27	9.07	9.67	15.60
	(SD)	(6.62)	(8.16)	(6.65)	(7.38)
Black Adult Psychopaths[c]	*M*	5.93	16.13	12.07	17.13
	(SD)	(5.36)	(8.14)	(6.49)	(7.12)
Black Adult Offender Controls	*M*	8.00	11.87	12.00	15.29
	(SD)	(6.12)	(6.88)	(6.26)	(6.37)

Note. OE = Omission Errors
CE = Commission Errors

[a] Expressed in means and standard deviations. SD presented when available.

[b] Newman et al. (1986): n = 15 in each cell; mean age = 26; subjects male inmates; diagnosis based on Hare's Psychopathy Checklist & DSM-III criteria for APD; computerized task administration; monetary reinforcement; each subject tested in one condition only

[c] Kossan et al. (1990): n = 15 in each cell; diagnosis; subject characteristics and task procedure same as Newman et al. (1986).

Table 10 - continued

Reward Dominance-Passive Avoidance Task Baseline Performance Compared to Samples in Other Studies

		Reward & Punishment		Punishment Only	
		OE	CE	OE	CE
Adolescent Psychopathic Offenders[d]	*M*	9.95	11.75	-	-
	(SD)	(5.47)	(5.83)	-	-
Adolescent Nonpsychopathic Offenders	*M*	13.62	10.94	-	-
	(SD)	(7.26)	(6.79)	-	-
Adolescent Delinquent[e]	*M*	7.98	14.29	-	-
	(SD)	(4.50)	(5.84)	-	-
Extraverted College Students[f]	*M*	5.68	12.32	7.16	10.26
Introverted College Students	*M*	7.67	8.52	7.48	10.48

Note. OE = Omission Errors
CE = Commission Errors

[d] Scerbo et al. (1990): N = 40; mean age = 15.8 years; subjects male, inpatient juvenile offenders divided into psychopaths (n = 24) & nonpsychopaths (n = 16) based on DSM-III-R criteria for APD and questionnaire measures for CD; Newman et al. (1985) computerized R + P task; monetary reinforcement

[e] Newman et al. (1985): N = 11; mean age = 17; subjects male, Caucasian, secondary school students; diagnosis based on MMPI & Welsh Anxiety Scale; manual file card task administration; candy/cigarettes as reinforcements.

[f] Newman et al. (1985): *n* = 40 in both groups; subjects male; group categorization based on Eysenck Personality Questionnaire; manual file card task administration; monetary reinforcement; subjects tested in both conditions.

PART D

DISCUSSION

Chapter 1
Treatment Effects

This research represents an exploratory investigation of an acute pharmacological intervention that has received extensive study in the context of childhood ADHD, but not in the contexts of either adolescent ADHD or co-occurring CD. A large body of literature encompassing studies of syndrome comorbidity with ADHD, factorial analyses of syndromal independence, discriminant validity of homogeneous subgrouping based on associated aggression or CD, and outcome of ADHD children in adolescence, has unequivocally established the high frequency with which ADHD, in both childhood and adolescence, presents with concurrent CD. It is clear that progress is required in the management of the mixed ADHD/CD subgroup; empirical data indicate that the prognosis for the long-term psychosocial adjustment of this subtype, relative to that of child and adolescent groups who show only ADHD or only CD, is quite poor. However, previous research examining parameters of ADHD, particularly response to stimulant drug treatment, has largely failed to control for associated conduct symptomatology, thereby preventing delineation of potentially important subgroup responses. Furthermore, psychopharmacotherapy as a potentially adjunctive measure in the treatment of the ADHD adolescent age group has been seriously neglected, largely because historically pervasive myths erroneously contraindicated its application in the adolescent population. To date, only three controlled studies of the acute effects of psychoactive medication in ADHD youths have been reported. The present study was therefore guided by the need for the evaluation of a controlled trial of stimulant medication with the adolescent ADHD/CD subtype, and the expectation that this group would show a positive overall drug response comparable to that observed in both ADHD adolescents and children with ADHD and concurrent conduct symptomatology.

A logical point of departure for discussion of the results is an evaluation of the efficacy of the pharmacologic manipulation. Here, interpretation of the data rests on the

distinction between drug *response* and drug *effect* (Liberman, 1962; Ross & Buckalew, 1979, 1983, 1985). Drug response(s) refers to statistically significant change(s) above baseline levels of performance or symptomatology. By contrast, drug effect(s) refers to such change(s) above placebo levels. The use of the placebo phase as a control for the drug effect rests on the assumption that the administration of a pharmacologically inert substance acts as a control for the well-documented effects of non-specific factors in treatment efficacy, such as patient expectancies of change, mere participation in a treatment protocol, personal contact with a help-providing professional, and exposure to a logical rationale for troubling symptomatology (Kazdin, 1980; Ross & Buckalew, 1985). Therefore, in contrast to the drug effect, the drug response may include the confounding effects of non-specific placebo factors, in addition to the effects of repeated testing and spontaneous fluctuations in symptomatology. It is for these reasons that the drug effect is the criterion of efficacy in pharmacological treatment research. It should be noted that placebo-baseline differences do not constitute the placebo effects, but rather the placebo responses. Here, a baseline phase serves as a control for the placebo response(s), whereas the inclusion of a no-treatment control group that separates the effects of repeated testing and spontaneous changes in symptomatology from the effects of the placebo per se, is necessary for the determination of placebo effects. This latter distinction is based on the position of Ross and Buckalew (1983, 1985) that placebo effect refers to significant behavioural change that can be directly and unequivocally attributed to the "specific" effects of the placebo, analogous to the drug effect which "represents that portion of the response domain that can be directly attributed to the specific action of the drug" (Ross & Buckalew, 1985, p. 71).

The results of the present investigation revealed, at a group level, significant medication responses on 3 of the 7 dependent measures. Pairwise comparisons between the baseline and the two active drug conditions did not indicate a differential change in target behaviours as a function of change in dosage. On the delay (DRL) task, the administration

of MPH significantly enhanced subjects' abilities to delay or refrain from emitting non-reinforced responses, and may have concomitantly improved subjects' cognitive control and execution of strategies for estimating the interresponse interval. On the reward dominance-passive avoidance task, subjects showed improved capacity to detect and respond to stimuli associated with reward (S+), and to reduce inappropriate, impulsive responses to stimuli associated with response-cost (S-). A medication response for the omission error measure of the distractibility task also approached significance ($p < .05$), implying a tendency toward improvement with MPH, relative to baseline levels, in subjects' selective attention and processing of concurrent stimuli. Taken together, these findings demonstrate that both the low and moderate doses of MPH served to strengthen subjects' inhibitory control and the effective deployment of attention to task relative to pre-treatment levels.

Despite the pronounced drug responses of both doses of the active drug, drug effects were not obtained on any of the dependent measures for the battery of repeated tasks. Furthermore, an analysis of the average of subjects' performances on the two drug conditions, with placebo levels serving as the control, failed to reveal significant overall MPH treatment effects for the present sample. A subsequent component analysis of this data did not suggest the presence of a homogeneous positive responder subgroup in terms of subjects' global responses to the active drug. Hence, the expectation that the low and/or moderate doses of MPH would exert significant improvements, relative to placebo, on measures of ADHD symptomatology in the present sample was not sustained. Although few studies provide an evaluative context for the current study, these findings are discrepant with previous reports of positive stimulant drug effects in ADHD adolescents and ADHD children with co-existing CD. Possible explanations for the present outcome rest on divergent sources of error, such as pharmacological errors of inadequate dosage, sampling bias or diagnostic error, repeated testing, and floor effects. The presence of potent placebo responses and interindividual and intravariability of drug treatment response in the present sample may also account for the findings. The results will, therefore, be discussed

according to the following subject areas: medication, diagnosis, placebo responses, measurement of ADHD symptomatology, and positive drug responder versus non-responder status.

Medication

It is clear from the findings of significant drug responses that a beneficial response to the stimulant therapy occurred and was noticeable, but fell short of an optimal drug effect when compared to placebo levels of performance. The low and moderate doses that were used in the present study were standard doses used in psychopharmacology research with ADHD children and adolescents. Although administration of identical doses to all study subjects, based on standard doses used in the psychopharmacology of ADHD, increased standardization of the study, standardization of dosage did not allow for the possibility of between-subject variability in the dosage level required for optimal response. To determine optimal efficacy for individual cases, trials of progressively higher doses are necessary until an adverse response occurs (Goodman & Gillman, 1970; Kinsbourne & Swanson, 1980). Kinsbourne and Swanson (1980) have stated in this regard, "if this is not done, some children who are favourable responders may be untreated. In a long-term outcome study, this would result in the underestimation of the benefit of stimulant therapy" (p. 209). It is conceivable that the use of fixed mg/kg doses in the present study effectively undermedicated certain subjects, resulting in a deflated estimation of the potential acute efficacy of MPH for the current sample. Indeed, individual titration of dosage based on a flexible dosage regime, with optimal dosage assessed for each youth, might have yielded better results. This practice would have had the further advantage of being directly comparable to sound clinical practice (Taylor et al., 1987).

However, the more flexible approach to dosing posed a number of disadvantages for the present study. These included possibly compromising the triple-blind procedures,

potentially undermining the cooperation of subjects and inpatient staff by requiring a longer trial than seemed practical in a crossover design, introducing further subjectivity in decision-making regarding dosing, and increasing the difficulty in supervising the trial. Moreover, there was a reluctance to prescribe doses above 0.5-mg/kg per dose because serious side-effects at higher doses raise ethical concerns (Sprague & Sleator, 1977; Winsberg, Kupietz, Sverd, Hungund, & Young, 1982). There was also the concern that the physical concomitants of higher doses might interfere with subjects' performances and confound the more direct cognitive effects of the medication.

Nevertheless, the possibility of considerable interindividual variability in optimal dose level in the present sample may have contributed to the present failure to obtain an overall drug effect. Ideally, future research assessing the short-term effects of MPH on the mixed ADHD/CD adolescent subgroup would include designs that establish therapeutic dosage levels on an individual basis.

Diagnosis

Alternative explanations for this study's failure to obtain significant drug effects address the roles of diagnostic error and bias in subject selection due to the use of retrospective assessment. In the present study, the three diagnostic reliability raters and the psychiatrist conducting the clinical assessment interview were trained to a criterion of 80% agreement prior to beginning data collection. Also, detailed instructions were given to all raters to not discuss a case before each rater had independently filled out the diagnostic IS form. The interrater reliabilities that were obtained, expressed in kappa statistics, ranged from .97 and .91 for the diagnosis of ADDH, and from .65 to .78 for the diagnosis of CD, all of which are quite high. A high kappa is generally considered to be 0.7 or above (DSM-III-R; American Psychiatric Association, 1987) and is indicative of good interrater agreement. However, the IS interrater reliability data, on their own, say little of the

validity of the diagnoses made. None of the raters, nor the psychiatrist, were blind to the diagnoses required for the study, and were certainly motivated to obtain subjects so as to increase sample size. Similarly, the psychiatric social worker and the research assistant, who were conducting the current and retrospective assessments of ADDH with the parents or guardians of the subjects, were not blind to the purposes of the study. It is therefore possible that the participants in the diagnostic decision process were biased toward observing ADHD symptomatology in the larger sample of young offenders and may have, unwittingly, selected a number of "false positives". This is more likely to be the case for the assessment of ADDH than for CD, as the DSM-III diagnostic criteria for ADDH are far less precise and clear than are the criteria for CD.

A second interpretation of the diagnostic data, which does not preclude the possible contribution of bias in the diagnostic process, centers on the validity and the clinical implications of the retrospective assessment method. Nearly two-thirds (21 of 33) of the subjects in the current study were diagnosed with ADDH in childhood based on parental retrospective report. By contrast, only one-third (11 of 33) of subjects had a clear, prior diagnosis of the disorder in childhood. Of importance in this context is the clinical literature on the "referability" (Weisz & Weiss, 1991) of child psychopathology. Data indicate that the referability of the same behaviour problem may differ as a function of child characteristics, as well as of the responses of adults to the problem behaviours (Walker, Bettes, & Ceci, 1984; Weisz, Suwanlert, Chaiyasit, Weiss, Walter, & Anderson, 1988). Hence, teacher and parental judgements about how serious and how much in need of treatment a problem is are related to how troubling or bothersome the behaviour is to others. The more serious a behaviour problem, and the greater its perceived severity, the more likely that the child manifesting the behaviour problem will be referred for medical or psychiatric treatment. Following this logic, it is probable that the group of subjects that had received a diagnosis of ADHD in childhood, as contrasted with the group of subjects that was retrospectively diagnosed with a childhood history of ADDH, represented a more

severe and pervasive variant of the disorder. Support for this contention comes from analyses examining the contribution of a prior diagnosis of childhood ADHD in subjects' responses to the treatment protocol.

Here, subjects who had previously received a childhood diagnosis of the disorder (n=8 for those with complete data) were found to show twice the omission error rate on the distractibility task, across the four experimental phases, of subjects who were retrospectively diagnosed with ADDH in childhood (n=19 for those with complete data). This indicates that the previously diagnosed group evidenced greater lapses in attention and were more vulnerable than their retrospectively diagnosed counterparts to distractors embedded within the stimulus array. Stated differently, these findings suggest that subjects with a former diagnosis of childhood hyperactivity represented a subgroup with greater severity of attention deficit, at least in terms of distractibility. Although these findings fell short of statistical significance when the correction for family-wise error rate was applied, it is, nevertheless, meaningful that a probability level of .05 was obtained with the small sample sizes, particularly in the priorly diagnosed group (n=8). Furthermore, the childhood history grouping variable served to significantly reduce the error variance for the tests of the main effects of the treatment manipulations. This indicates that control for retrospective measurement of ADDH reduced treatment variability and possibly identified a subgroup with milder cognitive impairment.

The implications of these findings for empirical research on the mixed ADHD/CD adolescent subtype and, in particular, on the assessment of stimulant drug treatment efficacy for this subgroup, are twofold. From a diagnostic perspective, one can question the validity of retrospective measurement of ADHD based on both patient and parental report. Although it is unclear whether the use of a retrospective assessment method in the present study resulted in the unwanted inclusion of a number of "false positives", the data do suggest that this method of identifying ADHD in childhood yields a sample with a

considerably milder form of the disorder. Secondly, the results of the present study bring into question both the validity of research findings based on retrospective data alone, and the wisdom of continuing to include retrospective assessment techniques in studies of ADHD in adolescence and adulthood. The trade-off between diagnostic confidence and an increase in sample size may be a costly one; larger groups of subjects may nominally provide more statistical power, but at the price of increased error variance if diagnostic error occurs. More useful knowledge and improved comparability of findings can be obtained if researchers do not compromise on the quality of subject selection procedures and study clearly defined samples.

Placebo Responses

Placebo responses, using baseline levels as controls, were obtained in this research on 3 of the 7 dependent measures. Specifically, significant improvements in the inhibition of impulsive behavioural responses on the delay (DRL) task, and in the ability to reduce the number of misses of correct (S+) stimuli and the number of impulsive, false alarms to incorrect (S-) stimuli on the reward dominance-passive avoidance task, were observed in the placebo condition relative to baseline. These findings are noteworthy for their possible contribution in limiting the effectiveness of the active pharmacologic manipulation, as well as for their relevance to the treatment of adolescents who show ADHD concurrent with CD.

Given that the drug effect is contingent on the magnitude of the difference between performance under the inactive drug level versus the active drug level, the presence of a significant placebo response may limit the drug effect (Ross & Buckalew, 1985). It is possible that the optimal level of performance on the delay task and the reward dominance-passive avoidance task for the present sample, as a group, was seen during the placebo phase, thereby limiting any significant incremental differences due to the effects of the active drug. Although the absence of a no-treatment control group in the present study

precludes a precise determination of the magnitude of the placebo effect, it is reasonable to assume that the psychological concomitants of pill-taking, such as suggestion, hope, faith in the treatment, and expectation of change, contributed to the enhancement of target behaviours under the placebo condition. Further, it is important to consider the possible influence of the environmental context on subjects' performances. The present findings are consistent with data indicating that placebo effects may be magnified in inpatient settings where expectations of improvement are amplified by the therapeutic milieu (Shapiro & Morris, 1978). Additionally, subjects' knowledge that findings from the study would be included in the psychiatrist's report to the courts may have increased their motivation to perform on tasks. Interestingly, a companion stimulant treatment study conducted by Lysak (1989) that used 27 of the 33 subjects comprising the present sample, also obtained significant placebo responses on 5 of 11 dependent measures derived from a battery of neuropsychological tests. Lysak's data provides additional support for the presence of potent placebo responses in the current sample. Preliminary to a discussion of the possible value of placebo factors to pharmacological intervention with the adolescent ADHD/CD subtype, it is appropriate to review the example of previous pharmacological research with these groups that has guided, or misguided, an understanding of this phenomenon.

Much of the research assessing acute stimulant medication effects on ADHD children and adolescents have excluded baseline measures from their designs (e.g., Barkley, Fischer, Newby, & Breen, 1988; Brown & Sexson, 1988; Coons et al., 1987; Cunningham et al., 1991; Hinshaw et al., 1989; Klorman et al., 1987; Varley, 1983; Whalen, Henker, Swanson, Granger, Kliewer, & Spencer, 1987), or have used baseline scores as the covariate in statistical analyses with the intention of statistically controlling for premedication differences in the dependent measures (e.g., Rapport et al., 1986; Vyse & Rapport, 1989). This is problematic because it is essential that baseline levels, as well as placebo levels, of symptomatology are established as criteria for change (Werry, 1978). Although covarying out baseline levels of performance in statistical analyses allows for calculation of drug

treatment effects relative to placebo levels, this practice precludes an important determination of placebo responses per se, where baseline levels are used as the control. Furthermore, the more convenient methodological design of only comparing placebo levels to active drug levels of performance carries the risk of confounding placebo and baseline responses, such that the magnitude of the inert treatment response cannot be estimated. Indeed, performances on placebo are likely to be different from baseline levels because of the effects of patients' expectancies of beneficial change (Kazdin, 1980; Sprague, 1978). The present research findings tentatively suggest that the placebo response, and possibly the placebo effect, may be particularly potent in the adolescent ADHD population, and/or in the mixed ADHD/CD adolescent group. However, it is difficult to evaluate this finding in that the small body of research on pharmacological treatment of both ADHD adolescents and ADHD children with high aggression scores typically failed to include baseline levels as controls (Brown & Sexson, 1988; Coons et al., 1987; Cunningham et al., 1991; Hinshaw et al., 1989; Klorman et al., 1987, 1988; Milich et al., 1989; Varley, 1983). Moreover, a review of the literature failed to reveal studies that included no-treatment control groups to specifically evaluate the role of placebo effects in these ADHD subgroups.

By contrast, support for the ameliorative value of placebo responses and placebo effects in the ADHD child population is considerable. In a meta-analysis of 61 outcome studies reported by Ottenbacher and Cooper (1983), approximately 30% of stimulant-related improvement in attentional capacity and impulsivity was attributed to the components of the placebo effect. Similarly, other authors have reported placebo responses in as many as 26% (Sleator, von Neumann, & Sprague, 1974) and 40% of ADHD children (Varley, 1984; Werry, 1977). Perhaps this data should not be surprising given reports that medication is commonly perceived as a panacea among medicated hyperactive children (Rosen, O'Leary, & Conway, 1985; Whalen & Henker, 1976, 1980). For example, Whalen and Henker (1980) observed that ADHD children and adolescents receiving stimulant medication had expectations of improved abilities to "concentrate" and "calm down" that

were associated with ingestion of the drug, although older ADHD children were less likely than younger ones to view medication as a primary solution to their problem. Thus, patient perception of treatment may be an important influence that interacts with the "active" components of the intervention. More recent research (Milich et al., 1989), however, on ADHD children's attributions for task performance on medication versus placebo, is not consistent with the conclusion that pill-taking is related to external evaluations for performance. In fact, Milich and his co-workers (Milich et al., 1989) observed that medication, as an attributional factor compared to effort, ability, and task, was selected the least often as an explanation of CPT task performance on both medication and placebo. This data stands in contrast to findings that external pill-taking attributions for improved performance are components of the beneficial effects of MPH, have detrimental effects in terminating stimulant therapy with ADHD children, and can interfere with the planned withdrawal of medication (Rosen et al., 1985).

It is clear that nonspecific placebo factors have generally been treated as artifactual, transitory, and tangential to the ultimate goals of treatment in studies of the acute efficacy of stimulant medication in the ADHD population. This is most likely due to an assumption that placebos produce only palliative symptomatic treatment (i.e., psychological changes tangential to core ADHD deficits), whereas active pharmacological treatment is believed to effect true amelioration of the putative core deficits. However, the possibility of psychological change, and the confirming evidence of symptomatic change, with placebos that have been obtained in the present study, consistent with the literature on ADHD children, question the logic of this assumption. Such findings argue, instead, that placebo-related change is meaningful in its own right and should not be dismissed peremptorily on the presumption that its mechanisms are spurious or unworthy of systematic inquiry. Systematic investigation of placebo-related attenuation of ADHD symptomatology may shed light on the question of whether ADHD may be due to an application deficit, associated with low motivation to comply with environmental demands, as some have

argued (e.g., Prior & Sanson, 1986). The present findings of placebo-induced changes in symptomatology are also consistent with the theories of Douglas (1983) and Kinsbourne (1989) that ADHD is characterized by a "production deficiency", suggesting that ADHD children and adolescents are capable of showing focused and sustained attention, and good impulse inhibition, under certain circumstances. The question then becomes one of investigating which components of placebo treatment and environmental manipulations promote the effective deployment of attention and impulse control, and why. Is it the case that the mere act of pill-taking, independent of the active pharmacological properties of the substance ingested, serves to engage the individual, and thereby facilitate arousal and the effective deployment of attention to task? Similarly, does an inpatient setting, or the expectation of adjudication of alleged antisocial acts, increase arousal and/or motivation, thereby magnifying the placebo response or effect? The possible efficacy of placebo treatment in improving ADHD symptomatology converges with the broader issue of situation-specificity of performance deficits in ADHD samples. Further research in these areas would prove valuable from a therapeutic, as well as a theoretical, standpoint.

The importance of adequate baseline and placebo controls in clinical trials in psychopharmacology also extends to the question of the sensitivity of the assessment measures used (Klerman, 1986). In psychopharmacological research, the measures of pre- and post-treatment changes should be "calibrated" against placebo levels to establish instrument sensitivity, analogous to the field of chemistry where laboratory instruments are calibrated against blanks and standards to maintain quality control (Klerman, 1986). Hence, placebo levels of performance serve as a test of the sensitivity of the research measures used. The present failure to obtain overall medication effects for the sample, as a group, therefore, raises the question of the sensitivity of the measures that were employed in this research. This leads us into a discussion of the general class of issues falling under the category of instrumentation.

Measurement of ADHD Symptomatology: Battery of Tasks

The issues falling under the area of measurement that address the current failure to obtain significant medication effects include task sensitivity to drug treatment effects, floor effects, and task difficulty. Evaluation of possible threats to the internal validity of the study is not complete, however, without a preliminary consideration of the possible contribution of effects associated with repeated testing.

Practice Effects

As a means of controlling for potential practice effects due to repeated testing, such as learning and transfer, the administration sequence of the active and inactive drug treatments, as well as the test battery, were counterbalanced in the current research. Subjects were also randomly assigned to the different treatment- and test-order combinations. Counterbalancing permits a spread of possible practice effects equally over treatment conditions and an analysis of treatment effects "unadulterated or colored by the effects of practice" (Keppel, 1982, p. 373) when the counterbalancing variable is used as a grouping variable in MANOVA designs. Further, task stimuli were modified across the experimental conditions to control for the possible effects of repeated task administrations, with the exceptions of the vigilance and distractibility tasks where only two versions of target stimuli were available to the researcher. The two versions of these tasks were therefore administered in alternate orders across the four experimental phases.

Analyses of the counterbalanced treatment order variable did not reveal significant effects for either the GDS or the RDPA tasks. Moreover, the *F*-values for these analyses were small (i.e., less than or equal to 1.0). Taken together, these results indicate the absence of appreciable, or significant, practice effects across the three treatment phases and, more importantly, the absence of differential practice effects for treatment conditions.

Careful inspection of the sample means across the four experimental conditions, as illustrated in Table 7, reveals that subjects' performances on the set of attentional, impulse control, and passive avoidance learning measures tended to show a gradual, but moderate, improvement with repeated testing. This would suggest that if such effects were due, in part, to successive experience with the tasks, then the practice effects were generally positive, as contrasted with negative effects where deterioration is observed. Moreover, if these means do, in fact, reflect a linear trend toward enhanced performance across the treatment conditions, independent of treatment order effects, this indicates that the practice effects were comparable for each of the three treatment conditions.

However, a precise determination of practice effects in the present study would have required the inclusion of a no-treatment control group, matched on selected variables, that received the four administrations of the tasks according to the same time sequence as the treatment sample. Although the absence of this control group precludes the ability to draw unequivocal conclusions regarding the therapeutic benefit of the active and inactive treatment manipulations independent of the effects of repeated testing, the data do, nevertheless, indicate that practice effects did not play an appreciable role in limiting, or confounding, task sensitivity to medication effects.

Sensitivity to Drug Treatment Effects

In pharmacological investigations of the efficacy of stimulant drugs on the treatment of ADHD, the choice of target behaviours as the dependent variables and the method of their assessment are critical decisions. The selection of the current assessment battery and the dependent variables under study were guided by the assumption, emphasized in the literature on the psychopharmacology of ADHD, that direct objective measurement of specific behavioural responses is the most accurate means of assessing acute drug effects.

However, reviews of the pediatric psychopharmacology literature (Knights, 1974; Sulzbacher, 1976) have indicated that ratings of behaviour are generally more sensitive than an objective test, or test battery, to the beneficial effects of the drug.

Different modes of assessment of stimulant treatment effects have generally yielded different outcomes. Sulzbacher (1976), for example, found that the likelihood of a beneficial effect being reported in pediatric psychopharmacological studies varied according to the type of response measure used. Across drugs and studies, the probability of an ameliorative drug effect being reported was .88 when global clinical impressions were used to index the drug effect, .57 when rating scales were employed, .41 when behaviour was directly observed, and .17 when scores on psychometric tests were examined. Knights (1974) reported similar findings when summarizing the effects of psychomotor stimulants, per se, on the behaviour of ADHD children; teachers' and parents' ratings were generally more sensitive than psychometric tests to the presence of drug effects. These data suggest that the evaluation of treatment-related changes on finer and finer aspects of ADHD behaviour may not necessarily provide for greater accuracy and greater amounts of information on the effects of stimulant drug therapy. It is likely that rating scales and clinical impressions best capture drug-related improvements in the multidimensional nature of core ADHD symptoms and in their manifestations in cross-situational, natural settings over longer time intervals than is possible with the administration of laboratory measures. Certainly, the present investigation might have profited from the additional use of rating scales, and/or from staff observations of behaviour, as supplementary measures of drug-induced behavioural change.

The usefulness of the present laboratory measures for detecting stimulant drug effects also pertains to the question of their ecological validity and generalizability. Barkley (1991) recently reported findings on the ecological validity of the CPT (vigilance) and the delay (DRL) tasks. Based on reviews of stimulant drug studies using these measures of

attention and impulsivity, and correlations of test scores with parent and teacher ratings, Barkley observed that CPT omission and commission scores have demonstrated sensitivity to stimulant medication effects, but not always reliably so. In general, he rated the ecological validity for the CPT measures as moderate, although this is less so for adolescent samples than for younger ADHD children. Barkley's conclusion is also consistent with Lovejoy and Rasmussen's (1990) study of the convergent and discriminant validity of vigilance tasks. Given that Gordon's delay (DRL) task is a more recent measure (Gordon, 1986), few studies have used it as an index of drug treatment responsiveness. At present, its ecological validity remains to be established, although it did not prove sensitive to drug treatment effects in one recent study (Barkley et al., 1988). These findings suggest, and others have similarly concluded (Barkley, 1991; Rapport et al., 1987) that the recent calls in the literature to incorporate laboratory measures into the clinical evaluation of ADHD, and into the empirical evaluation of pharmacological intervention for the disorder, should be tempered by the limited to moderate ecological validity of these measures.

Apart from the ecological validity of a measure, however, any given test of drug responsiveness is sensitive only to the extent that it effectively captures pre-treatment levels of impairment in the target behaviour(s). This leads us into discussion of subjects' mean baseline performances on the battery of tasks and whether such deficits were, in fact, observed.

Baseline Performances: Floor Effects and the Role of Base-State

Perhaps the most cogent explanation for this study's failure to obtain significant drug effects on the battery of repeated measures lies in the evaluation of subjects' mean baseline performances. Contrary to expectation, subjects, as a group, did not consistently demonstrate baseline deficits on tasks. Examination of mean baseline performances according to Gordon's (1986) norms for ADHD and non-ADHD same-age peers did not

reveal baseline levels of impairment in attentional capacity (misses) and impulsive responding (false alarms) on the vigilance and distractibility measures. Subjects mean performances fell in the normal and borderline abnormal ranges on these GDS tasks. Examination of the means for the ANOVAs of these tasks, across the four experimental conditions, revealed that they were quite low and yielded little variability across the three treatment phases. By contrast, mean performance on the delay (DRL) task fell in the lower limit of the borderline abnormal range and a significant main effect for treatment phase was obtained for this measure. Mean baseline performance on the reward dominance-passive avoidance task reflected deficits in the number of misses for correct stimuli, but did not indicate a deficit in passive avoidance learning, under either of the two reward conditions, when compared to the mean performances of adolescent delinquents and adult psychopaths. Nevertheless, passive avoidance errors were displayed.

This pattern of findings indicates that the majority of the sample did not exhibit baseline deficits on 2 of the 3 GDS tasks. It is likely, then, that error rates were so low on the vigilance and distractibility measures that a floor effect operated and prevented the emergence of a pharmacologic effect on performance. Conversely, the delay (DRL) and reward dominance-passive avoidance tasks yielded relatively higher rates of performance errors during the pre-treatment phase and, as expected, errors on these tasks were significantly reduced by MPH relative to pre-treatment levels. Therefore, together with results of MPH-related enhancement of performance, the baseline performance data point to the conclusion that it is only when less-than-optimal performance is observed on moderately difficult or difficult tests (as reflected in error rates) that improvement from the organizing effects of the stimulant occurs. In fact, this is precisely what has been discussed in the literature on ADHD and stimulant drugs. Douglas has hypothesized, in keeping with her self-regulatory model of ADHD, that the efficacy of stimulants is contingent upon the extent to which an ADHD child is performing below his level of competence (Douglas et al., 1988). Stimulant-induced improvement, therefore, is limited to situations involving a

performance-competence discrepancy. However, Douglas does not elaborate on how the discrepancy between competence and performance is to be defined. It is thus not clear what the operational criteria for the performance-competence discrepancy might be, other than invoking tautological or circular reasoning. A more parsimonious explanation for this pattern of results has received increasing attention in the literature on the psychopharmacology of ADHD, namely, that base-state or base-rate of responding influences the magnitude and direction of the drug response (e.g., Hicks, Gualtieri, Mayo, Schroeder, & Lipton, 1985; Kinsbourne, 1985; Robbins & Sahakian, 1979).

There is, at present, a relatively large body of research reporting that stimulant medication effects are contingent on the rate of the target behaviour, or degree of symptomatology, shown by the individual in the undrugged state. Wilder (1957) first discussed this phenomenon and named it the *law of initial values.* The *law of initial values* states that the higher the pre-stimulus level of functioning, the smaller the response to a function-raising stimulus, and the larger the response to a function-lowering stimulus. At more extreme pre-treatment levels, there is a tendency for no response to stimulation or even for paradoxical responses that reverse the expected direction of response. Consistent with this formulation, the initial or baseline rate of target behaviour has been found to influence psychopharmacological response in ADHD children on a range of both simple and complex measures of behavioural and cognitive functioning. These include a paired associate learning task, the MFFT, the CPT, actometers, a simple DRL operant task, and Freeman's (1978) risk-taking and passive avoidance learning tasks (Gualtieri et al., 1984; Hicks et al., 1985; Kinsbourne, 1985; Rapport et al., 1985; Weber, 1985). Base-state dependent drug effects have also been observed on a diverse set of physiological measures (Hicks et al., 1985; Kinsbourne, 1985). The statistical relationship between base-rate of responding and medication efficacy also predicts the direction of the drug response (Kinsbourne, 1985, 1989). For example, if the ADHD child shows excessive activity in the drug-free state, the stimulant-related effects will be seen in a reduction of activity. If

inactive in the drug-free state, activity levels will increase in response to stimulant medication. In addition, the magnitude of base-state drug effects are dose-dependent (Robbins & Sahakian, 1979); pre-treatment levels of target behaviours are reduced, or improved, as a function of increasing dose. However, toxic levels of stimulant drugs (i.e., above 1.0-mg/kg) cause levels of behaviour that do not reflect base-state (Kinsbourne, 1985; Robbins & Sahakian, 1979). Therefore, MPH overdose may cause a treatment emergent effect of, say, twitching, at a great rate, regardless of how much the individual twitched in the drug-free state. Moreover, data indicate that the phenomenon of base-state dependent drug effects is not an artifact of statistical regression to the mean for repeated measurements (Hicks et al., 1985; Robbins & Sahakian, 1979), where both high and low scores obtained on the first, or initial, assessment of target behaviours will shift towards the mean on a subsequent assessment.

Although the statistical relationship between base-state and amount of drug-induced change can explain the present findings, this phenomenon has further relevance for an understanding of the psychopharmacology of ADHD and the identification of favourable drug responders. The *law of initial values* seems to reflect, or implicate, a homeostasis mechanism. Hicks and his colleagues (Hicks et al., 1985) have stated in this regard:

> We assume that moderate scores in the drug-free state reflect an adequately regulated neural mechanism controlling that system. Methylphenidate has a minimal effect in this case. If the regulatory mechanism is malfunctioning because the setpoint is too extreme or because of underdamping (i.e., variable, oscillatory states), methylphenidate appears able to engage homeostatic mechanisms which take over and operate to return functioning to a more normal level. (p. 138)

The implications of such a conceptualization are threefold. First, it suggests that the psychopharmacology of MPH involves a reversal or normalization of a homeostatic imbalance. Whether the mechanism of drug action involves a normalization of an unstable, or biased, control system or a shift in state remains unclear, at present (Kinsbourne, 1989). Secondly, the phenomenon also casts new light on the task specificity of stimulant effects. Of importance are findings that drug effects on different measures do not correlate beyond a

chance level (Hicks et al., 1985). Hence, drug effects are not uniform across measures, but depend on the degree of abnormality of symptomatology, or performance, in the drug-free state. This argues against unitary deficit/drug effect theories and has important practical implications for the identification of favourable responders. According to unitary deficit theories, such as those emphasizing either hypoarousal (Klove & Hole, 1979; Satterfield & Dawson, 1971; Zentall & Zentall, 1967) or hyperarousal (Laufer, Denhoff, & Solomons, 1957) as the pathophysiology underlying ADHD, stimulants act to normalize the dysfunctional state. A favourable drug response should therefore generalize across all areas of dysfunction involving a hypoaroused state, or a hyperaroused state, as the case may be. However, the data suggest that a single favourable drug response will not necessarily generalize to other target behaviours. Thirdly, then, these findings have clinical implications for how a favourable response to the drug is defined and investigated.

Favourable Responder/Non-Responder Status

Previous research has shown that generalizing results obtained from group-level statistical analyses to individual cases is frequently misleading because of the idiosyncratic and task-specific responses associated with MPH (Rapport et al., 1985, 1986, 1987, 1988). Consequently, a component analysis of the drug effect change score data was conducted to determine whether a subgroup responded positively to the drug-dosage manipulations across the battery of measures. Negative findings were obtained. However, the failure to identify a favourable drug responder subgroup may well be an artifact underlying the use of a component analysis, namely, of a homogeneous global response, as opposed to a task-specific response, to the drug. This is suggested as a large body of literature on the psychopharmacology of ADHD provides robust evidence that (1) stimulant drugs have a somewhat nonspecific action on a range of ADHD symptomatology (Kinsbourne, 1989; Taylor, 1983; Taylor et al., 1987); (2) a positive response to MPH can be task-specific (Douglas et al., 1986, 1988; Rapport et al., 1986; Robbins & Sahakian, 1979); and (3) there

is considerable interindividual and intraindividual variability in response patterns across laboratory tasks and global assessment measures of change (Barkley et al., 1989; Brown & Sexson, 1988; Cunningham et al., 1991; Douglas et al., 1988; Rapport et al., 1986, 1987). Hence, although a proportion of favourable responders do show consistent changes across various tasks and behavioural domains (Rapport et al., 1986), the positive drug-response observed in other favourable responders does not necessarily generalize across all targeted domains.

Extensive literature has developed concerning the identification and study of favourable responder and non-responder subgroups of ADHD children. There are many methodological problems associated with this area of investigation. Most critical, of course, are the definitions of improvement and of treatment refractoriness. As Klein, Gittelman, Quitkin, and Rifkin (1980) have discussed, how much consensus across tasks and raters should be required to confirm a positive treatment response? Assuming resolution of this issue, however, there would remain the central problem of arriving at consensual criteria of the magnitude of improvement that signifies responsiveness. Magnitudes of improvement believed to indicate positive responsiveness that have been selected in the literature have markedly varied from a low of 5% (Whalen et al., 1987), 10% (Whalen et al., 1989), and 25% (Rapport et al., 1985; Swanson, Kinsbourne, Roberts, & Zucker, 1978), to a high of 50% (Taylor et al., 1987). Moreover, the selection of baseline or placebo levels as the controls for drug-induced changes has varied across studies. A recent study by Taylor (Taylor et al, 1987) is worthy of note as he selected a ratio score of 50% improvement that allowed for consideration of placebo levels as well as baseline levels of change (i.e., placebo score minus drug score, divided by baseline score, with this figure multiplied by 100). Four indices of improvement, including clinical, teacher, and parent ratings of hyperactive behaviour, were selected as the measures of drug responsiveness. However, the meaning of a ratio statistic in this context is questionable, as the classification of the level of observed ADHD behaviours more likely requires ordinal or interval scale measurements. Douglas

(Douglas et al., 1988) recently identified unfavourable responders on the basis of consistent superior performance on placebo, using more than 35 dependent variables. She did not specify the required magnitude of improvement between placebo and drug levels. Given her lax criteria, it is not surprising that she was unable to classify any of the ADHD children in the sample as consistent non-responders. Although it was not clearly specified by Douglas, it is suggested, deductively, that a favourable responder would then be characterized by the absence of a consistent non-response to the drug.

Applying the criteria of Douglas (Douglas et al., 1988) and Taylor (Taylor et al., 1987) to the present sample, 27 subjects, out of a total of 30 with complete data, were identified as favourable responders according to Douglas' criteria, and 4 favourable responders were identified according to Taylor's ratio score, using 50% change on 4 out of 7 measures as the criteria. The application of Taylor's ratio score to the present measures was problematic, however, not only in assuming a ratio scale distribution, but also in the event that few errors were made in the baseline phase. For example, if only two errors were scored on a task under the baseline condition, one error was made on placebo, and zero errors were observed on both doses of the active drug, a ratio score of 50% improvement was obtained. Clearly, this does not constitute clinically meaningful change. Similarly, application of Douglas' criteria is likely to result in a number of "false positives". Douglas' criteria do not take into account factors that may confound performance on placebo, such as spontaneous fluctuations in symptomatology and the possibility of deterioration with repeated testing that is often seen on simple repetitive tasks with ADHD children. Moreover, Douglas's study did not include baseline levels as controls. Therefore, her formulation does not allow for the possibility of deterioration in performance across treatment phases, such that performance on placebo may be inferior to that observed on either dosage, but that performance on both the active and inactive treatments is actually worse than pre-treatment levels. A finding of 27 responders and 3 non-responders in the present sample, using Douglas' criteria, does not seem tenable in view of both the problems

with her formulation and the failure to obtain significant medication effects. Nevertheless, it is quite likely that a proportion of subjects in the current sample exhibited a favourable response to the drug on a number of measures. The between-subject variability in manifestation of positive drug-response that was observed with examination of individual subject profiles suggested that positive responsiveness depended not only on subjects' reactions to a dose, but also on the behaviour being assessed. Unfortunately, the absence of sound consensual criteria for the identification of a favourable drug responder subgroup precludes meaningful interpretation of this data. Suggestions for future research in this area are presented following a discussion of other results in this study.

In summary, the present study is one of the first to examine the acute efficacy of a controlled trial of stimulant medication for the adolescent ADHD/CD subgroup. Much of what has been written to date on the use of MPH with both the ADHD adolescent and the mixed ADHD/CD subtype has relied on case studies, poorly designed research (e.g., confounds of baseline with placebo levels), and speculation, rather than systematic, methodologically sound investigation. The present results are generally inconsistent with what is currently believed about medication effects in ADHD adolescents and ADHD co-existing with CD. The possibility that failure to individually titrate dosage may have underestimated the efficacy of MPH for the current sample was addressed. Further, assessment of drug-related improvement may have been compromised by the inclusion of a number of "false positives" in the sample, or a large number of subjects who had a considerably milder form of ADHD, due to the use of retrospective assessment in diagnosis of childhood history of ADHD. Similarly, it is possible that the presence of potent placebo responses that were obtained in this study limited any significant incremental differences due to the effects of the active drug. When the treatment outcome data were examined for individual subjects, considerable interindividual and intraindividual variability in response to the drug was observed. This is consistent with the literature on the effects of stimulant therapy with the ADHD population, including the subtype with co-occuring CD. Criteria

of positive responder status that have been used by other investigators was applied to individual subject profiles, but conceptual and methodological problems with these criteria precluded the ability to draw conclusions regarding responder status in this sample. What is likely the most cogent explanation for this study's failure to obtain medication effects on the battery of repeated measures addressed the relatively normal baseline performances that were obtained in the sample, as a group, on the vigilance, distractibility, and passive avoidance measures. In view of these findings, it can be argued that the current trial did not provide an adequate test of the efficacy of MPH in improving subjects' attentional capacity and impulsive behavioural responses that were assessed by these tasks. Subjects' performances in these areas left little room for improvement, precluding an adequate test of the efficacy of MPH in attenuating these aspects of ADHD symptomatology. The statistical role of pre-treatment levels of symptomatology, or base-state, in determining the efficacy of stimulant treatment was discussed. However, critical to an understanding of the mixed ADHD/CD adolescent subgroup is the question of whether the absence of baseline impairment in these areas is attributable to specific characteristics of the sample studied, in terms of the presence of associated CD, or to the inadequacy of the measures used to assess functioning in these areas. Consideration of recent research investigating attention deficit in the mixed ADHD/CD subtype and passive avoidance in CD sheds some explanatory light on this issue.

Chapter 2
Attention Deficit And Reinforcement Mechanisms In The Mixed ADHD/CD Subtype

Despite the exploratory nature of the current study's focus on the mixed ADHD/CD adolescent subtype, there was ample reason to assume that the sample would demonstrate deficits on tasks. If the tasks were sensitive to the behavioural and cognitive dimensions under study, one would conclude that the youths with a dual diagnosis of ADDH and CD in this study displayed relatively normal baseline vigilance, distractibility, and passive avoidance learning because they were not impaired in these areas. Or alternately, it is possible that these tasks were not sufficiently demanding for the adolescent subjects to yield impaired performances. It is pertinent that a number of task parameters have varied in research with vigilance tasks -- for example, length of the task, stimuli characteristics of complexity and presentation rate, and whether it was experimenter- or subject-paced. The specific effects of changes in parameters on attentional functioning in the ADHD population have been largely unexplored in any systematic way. Although inattention has received focus as the primary, or supraordinate, deficit in ADHD, the clinical and empirical emphasis on this core feature has not been paralleled by a consensual systematic approach to the investigation of attentional functioning. This is problematic in view of evidence that changes in stimuli complexity (Nuechterlein, Parasuraman, & Jiang, 1983), presentation rate (Parasuraman & Davies, 1977), and increasing age (Seidel & Joschko, 1990) can affect performance on vigilance tasks. Similarly, there may be certain components to the reward dominance-passive avoidance task, such as instructional set and a continuous reinforcement schedule, that reduced the likelihood of deficient passive avoidance learning. The question as to whether the absence of baseline impairment in these areas of cognitive processing and behaviour is due to specific characteristics of the sample studied, or to specific parameters of the tasks, is not easily answered. However, recent research on the severity of attention deficit in the mixed ADHD/CD subtype suggests that the present failure to obtain baseline

impairment in vigilance and distractibility was due to the characteristics of the present sample, rather than to limitations of the tasks themselves.

As discussed in the introduction to this study, impaired vigilance does not characterize all ADHD children (Trommer et al., 1987, 1988). Therefore, the use of vigilance tasks, such as the CPT, to classify ADHD children and adolescents may yield false negative results. Of importance in this context is recent data suggesting that about 50% of ADHD children, diagnosed on the basis of DSM-III-R criteria and teacher ratings of ADHD, show no evidence of objectively assessed attentional dysfunction on the CPT (Halperin, Newcorn, Sharma, Healey, Wolf, Pascualvaca, & Schwartz, 1990). This lack of sensitivity raises questions as to whether or not deficits in attention as measured by the CPT characterize a unitary group of children. Group data on impairment in attentional capacity and attentional strategies to task requirements can reflect either a shared trait, common to all members of the group, or a greater prevalence of the trait in a subgroup. Of particular importance to an interpretation of the current findings is increasing evidence that the absence of attentional dysfunction in ADHD children, as measured by objective laboratory tasks, is associated with the co-occurrence of conduct symptomatology (Aman & Turbott, 1986; Chee et al., 1989; Cunningham et al., 1991; Halperin, O'Brien, Newcorn, Healy, Pascualvaca, Wolf, & Young, 1990; Halperin et al., 1990; Schachar & Logan, 1990; Schachar et al., 1988; Werry, Elkind, & Reeves, 1987). These studies have most often used CPT-type paradigms to assess deficits in attentional capacity. In view of this recent data, the failure to observe significant performance deficits on measures of sustained attention and distractibility in the present sample of dually diagnosed adolescents is not surprising. In fact, when individual subject scores on these measures were examined in the baseline phase and compared to Gordon's (1986) norms for ADHD and non-ADHD same-age peers, it was observed that 22 subjects scored within the normal range on the two measures of the vigilance task, and that 21 subjects scored within the normal range for the two measures of the distractibility task. This reveals that virtually two thirds of the sample failed to display

baseline deficits on these tasks. Although the results only approached statistical significance when the correction for family-wise error rate was applied, relationships obtained in this study as a function of rated severity of ADDH are relevant at this juncture.

First, it is interesting to note that the dependent measures for the vigilance and distractibility tasks, and the omission error measure for the reward dominance-passive avoidance task, showed considerable between-subject variability, suggesting that attentional deficits were more pronounced in some subjects than in others. Second, when the data for the experimental conditions was grouped according to the severity of ADDH subject variable, interaction effects for the number of false alarms on the vigilance task, the efficiency ratio scores of the delay (DRL) task, and the number of misses on the reward dominance-passive avoidance task approached significance ($p < .05$). Perusal of the data revealed differential baseline performances and responsiveness to the two doses of MPH on the two GDS tasks, and differential performances across the two reward conditions of the passive avoidance task, as a function of rated severity of ADDH. On the vigilance task, conduct disordered adolescents with Severe ADDH scored twice the number of commission errors in the baseline condition, and displayed greater improvement across the inactive and active treatment conditions, when compared to the performances of conduct disordered adolescents rated as moderately ADDH. The mean baseline performance of the Moderate ADDH subgroup did not even approach impairment in the number of false alarms on this task, as illustrated by the low error rate and comparison to Gordon's norms for same-age peers. Conversely, on the delay (DRL) task, the pretreatment level of impairment in behavioural response inhibition in the Moderate ADDH subgroup was actually more severe than in their counterpart subgroup with Severe ADDH. Drug-induced improvement in the control of impulsive responding appeared to be comparable for the two groups on the delay task. Lastly, data for the reward dominance-passive avoidance task, as a function of subject grouping according to rated severity of associated ADDH, revealed that the Severe ADDH subgroup exhibited a greater number of misses of correct stimuli across the two

reward programmes, collapsed across experimental conditions, when compared to the performance of the Moderate ADDH subgroup.

Taken together, these data suggest a pattern of findings that characterizes the Severe ADDH/CD subgroup as more cognitively impaired and, possibly, more drug-responsive. This is suggested as a relative increase in impulsive behavioural responses, or false alarms for incorrect stimuli, was observed in this subgroup only to the extent that the task at hand tapped attentional capacity (vigilance task). Similarly, a relatively greater difficulty with detection of correct stimuli was observed in the Severe ADDH/CD subgroup on a task that involved discrimination learning, divided attention for correct versus incorrect stimuli, and the introduction of reward and response-cost contingencies (reward dominance-passive avoidance task). By contrast, a profile of relatively milder cognitive impairment, together with a relatively more pronounced deficit in impulse control, appears to characterize the Moderate ADDH/CD subgroup. Evidence of impairment in impulse control in this subgroup was observed only when the task at hand (delay task) required behavioural control to suppress or delay impulses in the absence of attentional demands (i.e., for rapidly occurring target stimuli). Additionally, these subjects appeared to have less difficulty than their Severe ADDH counterparts with attention to the passive avoidance task and the inhibition of responses to correct target stimuli. Notwithstanding the fact that the conclusions suggested by this data are limited by the absence of strong statistical significance, they are, nevertheless, important in view of their descriptive value. Moreover, levels of statistical significance for this group of analyses were stringent ($p < .008$) in order to control for an increase in family-wise error.

The present data on baseline performances and between-subject variability in attention deficit point to a number of conclusions. Studies conducted during, and subsequent to, completion of this investigation have provided a rather robust picture of the absence of attentional impairment in ADHD co-occurring with CD, relative to the degree of

severity observed for children who show only ADHD. The present failure to obtain baseline performance decrements on the vigilance and distractibility tasks for the sample as a group are, therefore, consistent with what has been reported in the literature on severity of attention deficit in the mixed ADHD/CD subtype. However, definitive statements about the severity of deficits in sustained attention and distractibility in the mixed ADHD/CD adolescent age group, based on the current paradigm, cannot be made without the inclusion of "pure" ADHD and "pure" CD comparison groups. Moreover, the previously discussed problems with the use of retrospective assessment for the diagnosis of ADHD in childhood complicates interpretation of the results. Is it the case that the present sample failed to demonstrate deficits in attentional capacity because the use of retrospective assessment in the subject selection process resulted in a biased selection of subjects with a milder form of ADHD? Or do the data reflect the recent findings in the literature that the co-occurrence of CD with ADHD identifies a subgroup with less severe attentional impairment? Further research in this area, using stringently defined diagnostic criteria, will be required to answer these questions. Notwithstanding this limitation in ability to draw firm conclusions, the presence of heterogeneity of severity of attention deficit, even within the more homogeneously defined subgroup of the mixed ADHD/CD adolescent subtype, is suggested by the current findings. If we assume this to be the case, it would have implications for the empirical study of ADHD concurrent with CD. It is possible that identification of subgroups based on antisocial comorbidity does not necessarily define a homogeneous subclassification of ADHD children and adolescents. Rather, severity of ADHD may need to be controlled for, even in the presence of co-existing CD.

When the presence of only *current* ADHD symptomatology is considered, the absence of objectively assessed severe attentional dysfunction in a proportion of the sample suggests that the attentional deficits seen in some adolescents who show a mixed clinical presentation of ADHD and CD may be non-specific secondary symptoms, or epiphenomena, of their CD. This view of the relationship between ADHD and CD has, in fact, been

proposed elsewhere (Schachar, 1989; Schachar & Logan, 1990). Extrapolating from this, it is possible that the mixed clinical presentation of ADHD and CD is largely attributable to the core feature of impulsivity, in terms of response inhibition and the abilty to tolerate delay, that is common to both disorders. This is suggested as the co-occurrence of CD and ADHD was observed in a number of subjects, based on clinical assessment of current symptomatology, even in the absence of an objectively assessed deficit in sustained attention and distractibility. Further, there was objective evidence that impulse control was problematic in both the moderate and severe ADDH groups. The present data, therefore, tempt conclusions that the common denominator between ADHD and CD in adolescence, at the behavioural level of analysis, may be the feature of impulsivity or disinhibition, as researchers have suggested (Freeman & Kinsbourne, 1984; Loeber, 1990; Reznick & Freeman, 1985). Reinforcement abnormalities have also been emphasized in both the ADHD and CD categories. Given the interplay between features of impulsivity and response to reinforcement, such as failure to consider all response alternatives, unresponsiveness to environmental constraints, and impulsive responding for that which is salient or rewarding, further examination of subjects' performances on the reward dominance-passive avoidance task becomes relevant to consideration of such an hypothesis.

As previously discussed, during the baseline phase, a performance decrement in the number of misses, but not in the number of passive avoidance errors, was observed relative to the mean performances of adolescent delinquents, adult psychopaths, and introverted and extroverted college students. Contrary to expectation, subjects, as a group, did not exhibit a passive avoidance learning deficit, nor was a differential pattern of performance observed under the two reward conditions. This pattern of findings is in contrast to studies that find deficits in passive avoidance learning in delinquent and psychopathic groups when competing reward and response-cost incentives are present, but not when contingencies involve only one motivationally significant goal (Newman et al., 1985, Newman & Kosson, 1986; Kosson et al., 1990). Most of the research on this concept has been undertaken with

incarcerated male offenders so that the applicability of the findings to male juvenile offenders who have associated ADHD remains uncertain, but is appropriate given the probable course for many young offenders. However, recent research (Scerbo et al., 1990) examining the effects of only the reward and response-cost (R + P) condition on juvenile offenders with CD and APD provides a better basis for comparison of results.

The sample used in Scerbo's study comprised a group of subjects that was not too dissimilar from that of the present study; subjects were male juvenile offenders residing in a correctional home and were grouped according to the presence and absence of CD and the DSM-III-R criteria for APD. Unfortunately, Scerbo's study did not evaluate the presence of associated ADHD. However, it seems fair to assume, in view of the substantial co-occurrence of the two disorders, that a portion of Scerbo's sample included offenders with ADHD. Interestingly, the findings of Scerbo's study failed to corroborate the earlier data of Newman and his colleagues with adolescent delinquents (Newman et al., 1985), but more closely parallel the present study's results. Scerbo did not observe a passive avoidance deficit in the adolescent CD/APD group relative to her offender controls. Both adolescent psychopathic and control groups, however, were more impaired by the contingent occurrence of reward and response-cost when contrasted with the passive avoidance performance of the present sample.

It is possible that a number of parameters of the RDPA task actually facilitated the occurrence of punishment avoidance in the present sample of offenders with a dual diagnosis of CD and ADHD. For example, the reward dominance-passive avoidance task involves processes of initiating, maintaining, modifying, and inhibiting actions with accordance to task demands. Task demands and the reinforcement contingencies involved are made explicit to the subject prior to administration of the task and are continuously reinforced. Clarification of conditions that appear to promote, versus interfere, with the learning of punishment cues in the ADHD group are important here. As discussed in the

introduction to this study, both contingent feedback and a continous reinforcement schedule have been identified as factors that enhance information-processing and increase task-relevant behaviour in ADHD children (Douglas, 1983, 1985; Douglas & Parry, 1983; Parry & Douglas, 1983). Rather, it is only on partial reinforcement and non-contingent feedback schedules that differences between ADHD and normal children have been observed, but not always reliably so (e.g., Douglas & Parry, 1983; Freibergs & Douglas, 1969). Moreover, in Freeman's (1978) study of passive avoidance learning with the Lykken maze, which is the only other investigation of this phenomenon in ADHD children to date, the task of avoiding the noxious noise was an implicit, or latent, one. Research also suggests that monetary reward and response-cost have particular salience for ADHD, as well as for CD and psychopathic groups (Freeman, 1978; Reznick & Freeman, 1985; Schmauk, 1970). These data suggest a number of interpretations for the absence of punishment avoidance in this sample. First, it is possible that explanation of the reinforcement contingency to subjects served to facilitate utilization of punishment cues. Second, it is likely that the continous contingent feedback had an organizing effect on subjects' information processing and ability to avoid punishment. It is probable that young offenders, in the presence of co-occurring ADHD, would show dramatic impulsive responding and poor punishment avoidance on the reward dominance-passive avoidance task under a partial reinforcement schedule. Varying the use of explicit versus implicit task instructions to subjects might also yield differential performance in passive avoidance. These variations of the task would be particularly interesting, as both implicit procedures and partial reinforcement contingencies provide better analogues of naturally occurring environmental conditions. Thirdly, it could be argued that the presence of salient punishment in the form of response-cost enhanced the adolescents' motivation and efforts to avoid loss of monetary reward.

Another interpretation of the current results, favoured by those who espouse a hypoarousal theory of ADHD (e.g., Satterfield & Dawson, 1971; Zentall & Zentall, 1967)

and APD (e.g., Chesno & Kilmann, 1975), centers on the stimulus value of the passive avoidance task itself. Subjects were observed to be particularly interested and motivated when participating in the passive avoidance "computer game" relative to their performance on the GDS tasks. This is consistent with adolescents' attraction to video and computer games, which have a number of features in common with the reward dominance-passive avoidance task. Assuming that arousal reflects an "intensity dimension" of behaviour (Duffy, 1962) and that underarousal reduces attentional capacity (Hasher & Zacks, 1979), one might speculate that, behaviourally, subjects were in a state of relative arousal and enhanced attention on this task. Psychophysiological and neurophysiological research on psychopathy and CD indicate that groups with these disorders will demonstrate passive avoidance when sufficiently aroused, with the use of either adrenaline, white noise, or presentation of events of interest (Chesno & Kilmann, 1975; Jutai, Hare & Donnolly, 1987; Raine & Venables, 1987, 1988; Schachter & Latane, 1964). Correspondingly, research on the differential effects of varying task parameters on ADHD children and adolescents indicates that the boundaries of successful performance include tasks and situations that are perceived as interesting and mildly stressful (Kinsbourne, 1989). The literature is therefore consistent with the notion that when sufficiently aroused or motivated, conduct disordered adolescents with ADHD are capable of effective utilization of punishing cues. The hypoarousal - increased arousal theory would also argue, however, that increased arousal would have a focusing effect on subjects' attention to task. Following this logic, we would expect comparably good performance in subjects' detection of correct stimuli on the passive avoidance task. However, this was not obtained, questioning the applicability of this interpretation of the data.

As noted previously, a substantial performance deficit in the ability to detect and respond to correct stimuli relative to data for delinquent adolescents and psychopathic adults was observed in this sample. When the present data was evaluated on the basis of subject grouping according to rated severity of ADDH, an interaction effect between rated

severity and reward condition was obtained that approached statistical significance ($p < .06$). This effect indicated that the Severe ADDH subgroup tended to demonstrate a greater number of misses of correct stimuli under both reward conditions when compared to their CD counterparts with Moderate ADDH. Also, the Severe ADDH subgroup exhibited a relative decrease in the number of misses of correct responses under the condition involving mixed reward and punishment incentives (R + P), relative to their performance under the punishment only (P-only) contingency. Conversely, the Moderate ADDH subgroup evidenced marginally superior performance in detection in the P-only condition when contrasted with their performance under the R + P condition.

These patterns of performance, as a function of rated severity of ADDH, are provocative, as they suggest the presence of reward dominance in the Severe ADDH subgroup. In the current paradigm, the theory of reward dominance (Quay, 1988) predicts a relative greater responsivity to reward stimuli when presented with conditions involving cues for both reward and punishment. Consistent with that prediction, the Severe ADDH subgroup showed a tendency toward superior focusing on stimuli associated with reward under conditions of mixed reward and response-cost incentives, but not under a condition involving only response-cost incentives. This performance profile may therefore be interpreted as a greater responsivity to reward. Furthermore, it suggests that when motivated by the prospect of reward, adolescents with CD and severe ADDH are capable of improved attentional performance. This hypothesis is particularly viable in light of empirical and clinical observations that the occurrence of attentional difficulties in ADHD children and adolescents is often idiosyncratic and appears to covary with the nature of environmental demands and task stimuli (e.g., Sykes et al., 1972; Milich et al., 1982; Prior et al., 1985). Interestingly, although the comparison is tempered by differences between within-subject and between-subject experimental designs, Scerbo and her coworkers (Scerbo et al., 1990) observed a similar profile in the adolescent CD/APD group relative to non-CD/APD controls. A response bias toward fewer misses, but not false alarms, was

observed in the CD/APD group compared to controls under the R + P condition, indicating greater reward dominance in the CD/APD group.

Having examined task parameters and characteristics of the sample that likely contributed to the absence of baseline impairment on selected measures, it is now possible to address the original question impelling this discussion. In the case of attentional capacity and distractibility, it was argued that the absence of an objectively assessed attentional deficit is, in fact, consistent with recent reports on the mixed ADHD/CD subtype. The present study further suggested the presence of interindividual variability in severity of attention deficit within the sample of adolescent offenders with ADHD and CD. Although it is not possible to evaluate the contribution of subject selection bias to this finding, the presence of heterogeneity in severity of attention deficit in the more homogeneously defined ADHD/CD subclassification would have particular relevance for study of the mechanisms underlying the association between ADHD and CD. Conversely, in the case of passive avoidance learning, it was suggested that the failure to observe a deficit in this area is likely attributable to specific parameters of the reward dominance-passive avoidance task. Several aspects of the current findings from this task suggest the need for further investigation. It is probable that impairment in passive avoidance learning would be observed in the mixed ADHD/CD subgroup as the probability of punishment, or response-cost, becomes increasingly uncertain. A more powerful test of the relationship between ADHD/CD and a deficit in passive avoidance learning would include the evaluation of remote implicit stimulus-response and partial reinforcement contingencies, both of which are better approximations of naturally occurring relationships. Future research with the mixed ADHD/CD subtype might examine the effects of increasing the magnitude of the possible loss or gain, and of varying the types and probabilities, of expected reinforcers. In addition, the finding of response bias toward misses, or inappropriate inhibitions, for correct stimuli and the variability in this bias according to severity of ADDH, requires replication. Research that incorporates methods to elucidate

the role of attentional mechanisms in responsivity to reward and response-cost would be valuable. This is recommended as the performance deficit in attention displayed by adolescents on this task indicates impairment in their information processing when in the presence of potential immediate reward and loss of reward. Investigations of this phenomenon may have important implications for an understanding of the apparent unresponsiveness to environmental contingencies, as well as the reputed tendency to overfocus on immediate goals, that have been emphasized in ADHD as well as in CD.

The foregoing leads into a discussion of what might be the most interesting result to emerge from this study. When focus is directed on the sample as a group, and the pattern of attentional deficits, and lack thereof, are considered at the pre-treatment level across all tasks, a picture emerges of attentional dysfunction that was largely dependent upon, or exacerbated by, the distracting influence of reward. Baseline impairment in the number of misses, or ability to detect and respond to correct stimuli, relative to data for ADHD, delinquent, or psychopathic groups, was not observed on simple tasks that did not involve reward contingencies, such as the vigilance and distractibility measures. However, when the prospect of obtaining and losing monetary reward was introduced, as with the reward dominance-passive avoidance task, so too did we see the emergence of attentional dysfunction in this sample. This suggests that reward, at least in the form of monetary reinforcement and response-cost, has particular salience for the ADHD/CD adolescent subtype. It may act to increase distraction and interfere with attention to the specific features of responses that are required. This response profile across tasks is consistent with views of both ADHD (Douglas, 1985; Freeman & Reznick, 1984) and CD (Quay, 1988) that an unusual sensitivity to both the presence of rewards and to the loss of anticipated rewards is characteristic of these disorders. More importantly, these data provide support for Douglas's hypothesized role of reinforcement mechanisms in ADHD and its interplay with attentional problems (Douglas, 1983). ADHD may be characterized by a cognitive style in which attention is easily dominated by what is of immediate, concrete interest. As

a result, integration of short-range goals with information about other relevant aspects of a situation, and/or the likely long-term consequences of actions, may be impeded. Although it is beyond the scope of this discussion, it is thought-provoking that the role of cognitive controls and reinforcement abnormalities in psychopathy have been presented in a strikingly similar way (e.g., Shapiro, 1965). In contrast to the core features of inattention and overactivity, much less is known about deficient impulse control and reinforcement mechanisms in ADHD. The present findings suggest that these latter "core" features of ADHD are fertile fields for further study.

Chapter 3
Stimulant Treatment And The ADHD/CD Subtype: Considerations For The Identification Of Favourable Responders

Just as confusion in terminology and absence of a consensual definition of ADHD have significantly hampered empirical progress in the study of ADHD, so too has the lack of consensual criteria for the identification of favourable stimulant drug responders impeded empirical progress in the psychopharmacology of the disorder. The question as to what constitutes favourable responsiveness and non-responsiveness to stimulant drugs in ADHD is a controversial one. Many researchers in the field of ADHD and pediatric psychopharmacology have espoused a categorical approach which classifies an ADHD individual as either treatment-sensitive or treatment-insensitive (e.g., Rapport et al., 1985; Sprague & Sleator, 1975; Swanson & Kinsbourne, 1978). Others have argued that, when ADHD is carefully diagnosed and a comprehensive battery of measures is employed, most, if not all, ADHD individuals will show a beneficial task-specific response to the drug (e.g., Douglas et al., 1988). The majority of researchers examining the efficacy of stimulant medication in ADHD children have assumed the categorical approach to identification of positive drug responders. Consequently, non-responders have been screened a priori and excluded from experimental designs (e.g., Rapport et al., 1985, 1986; Swanson et al., 1978; Thurston, Sobol, Swanson, & Kinsbourne, 1979). Swanson and Kinsbourne (1978) and Sprague and Sleator (1973, 1975, 1977) have recommended that objective measurement of cognitive response to a single cognitive test, the paired associate learning test (PAL), based on a 1-day or 2-day double-blind, drug-placebo testing sequence, be used as a diagnostic test of favourable and non-favourable drug response. However, there are serious conceptual and methodological problems with this procedure. To begin with, it is unlikely that the efficacy of a pharmacologic manipulation on ADHD behaviour is an all-or-none matter. The manipulation will not usually succeed or fail completely, but will probably affect subjects differently across targeted domains. Secondly, to only examine subjects who

responded favourably on the PAL test is to test an hypothesis that is different from an investigation of the effects of stimulant treatment on ADHD. Rather, dropping PAL-identified non-responders from subject samples tests the hypothesis that *PAL-identifed responders* will show changes on other dependent measures in response to administration of a stimulant drug. This leads us to the most important criticism of the practice recommended by Swanson and Kinsbourne (1978) and Sprague and Sleator (1973, 1975, 1977). The importance of not discarding subjects identified as non-responders on the basis of performance on one cognitive test is that the pharmacologic manipulation may have beneficial effects on other measures without showing any effect on the original criterion test. This criticism receives support from data indicating that both favourable and unfavourable responders identified by the PAL test have demonstrated a positive drug response on several other laboratory measures (Swanson & Kinsbourne, 1979). Thus, a single rating of "improved" or "unimproved" obscures the nature of the individual drug-response. Both interindividual and intraindividual variability in drug-response appear to be the rules rather than the exceptions. Behaviour changes observed in one subject may not correspond to those observed in another. For an individual, improvements in one behavioural domain are not necessarily seen in other targeted domains. Needless to say, the non-specificity of the stimulant response makes it extraordinarily difficult to develop criteria of an overall favourable drug response in ADHD. Given this situation, there is likely to be no single referent, but instead a pattern of replicated improvement across time and situations.

Although a revision of the a priori PAL screening procedure, involving extension to a 4-week assessment and use of subjective ratings of behaviour in the natural environment, was more recently introduced (Swanson, Sandman, Deutsch, & Baren, 1983), conceptual and methodological difficulties remain. Assessment of behavioural improvement over time, based on ratings of behaviour by the same observer, is subject to the artifact of statistical regression. If a particular behaviour in a group of ADHD individuals is measured at, say Time 1 and Time 2, one would expect to find some changes in those scores over time due to

the natural variability in behaviour and its measurement. Specifically, the deviant scores at both the low and high ends of the scale at Time 1 should shift towards the mean at Time 2. Thus, assuming that ADHD individuals would obtain scores at either the high end or the low end of the scale, depending upon the nature of the behaviour being assessed, there is a statistical likelihood of reduction in these scores when repeated measurements are taken. The statistical regression phenomenon has been observed in ratings of ADHD behaviour (Milich, Roberts, Loney, & Caputo, 1980; Zentall & Zentall, 1986) and has important methodological implications for the investigation of stimulant treatment efficacy.

In view of the problems involved with assessment of drug treatment response in ADHD, namely, of a nonspecific action on a range of ADHD symptomatology, cross-situational variability in ADHD behaviour, and statistical regression, simple within-subject design and between-subject design strategies include insufficient controls for the unequivocal determination of responder status. Rather, it is necessary to demonstrate stability, or *replication*, of both *deficits* and *improvements* on selected measures over time that are best captured by intrasubject-replication design, or A-B-A withdrawal design, strategies. The idiosyncratic task-specific drug response that is observed in ADHD samples of children and adolescents highlights the need for multiple task measures and test-retest assessment over selected time intervals in investigations of drug efficacy. In addition, the problem of the statistical regression phenomenon requires demonstration that behavioural change occurred with the implementation, as well as the reinstatement, of the stimulant treatment, so that a causal inference can be drawn between the active intervention and behavioural change. Similarly, the problem of differentiating the active components from placebo components of the pharmacological intervention require that pre-treatment, or baseline, levels of target behaviours are gathered over time and across situations, and that placebo levels are contrasted with both baseline and active treatment levels of change. Hence, replications of deficits and improvements due to the withdrawal and reinstatement of the active drug, where improvements are superior to placebo, are required for accurate

and unequivocal conclusions regarding favourable and non-favourable drug response. Furthermore, the effects of varying drug doses on behavioural change will have to be addressed if optimal response, and non-response, are to be established. If the foregoing is to be accepted as a methodological guideline for identification of responder status, the present author is unable to find any study, to date, that meets this standard.

A related problem in identifying drug responders addresses the clinical meaningfulness of the behaviour change that is observed in response to stimulant medication. For example, Swanson and Kinsbourne (1979) have argued that many simple behavioural tasks overestimate the beneficial response to stimulants and that performance on the PAL test provides a good predictor of change "in the clinical sense". Similarly, other investigators have stressed the discriminatory value of "high level" or "high load" information processing tasks in discriminating ADHD drug responders and non-responders (Douglas et al., 1986, 1988; Rapport et al, 1985; Swanson, 1985). The question of clinically meaningful change can only be assessed by careful, individual assessment of behavioural improvement based on ratings by multiple sources in diverse settings, so that replication of improvement in critical areas of functioning, such as in the home, classroom, and/or playground, can be argued. Needless to say, it is essential that a variety of measures are used, ideally from the social, clinical, familial, and academic domains. The failure to obtain significant medication effects in this study, concomitant with a failure to include rated observations of behaviour, underscores the need to include multilple methods of assessment that best capture the multidimensional nature of ADHD. Observations of behaviour rated by multiple sources, in addition to consideration of laboratory measures, is recommended, at a minimum. A battery of clinic-based measures that samples various behavioural domains, that includes task varying in high versus low processing demands, and that has documented sensitivity to stimulant dosage manipulations, would augment clinical decision-making. In essence, then, intrasubject replication designs that employ a multitrait-multimethod-multisource, or multi-situation, approach to assessment of both deficits and

treatment change are suggested. These designs are commonly known as multiple baseline, across setting, treatment withdrawal designs.

From a practical standpoint, practitioners may choose to focus on those measures that address the more serious presenting problems. This will likely depend on social factors and the nature of parental complaints. Thus, what one parent may emphasize as requiring intervention, another parent may be relatively unconcerned with. This will particularly be the case with the mixed ADHD/CD subtype, as conduct problems may be of paramount importance to parents and/or teachers. Consideration of the use of pharmacological intervention in clinical practice raises the issue that intervention programmes should be tailored to suit individual needs. Pediatric psychopharmacological research on ADHD has near uniformly slotted ADHD children into experimental designs that investigate drug-induced changes on pre-selected target behaviours, rather than meeting a priori-determined individual needs. It would therefore be important to conduct an investigation of deficits and problem areas, across time and situations, prior to selection of targeted domains. Target behaviours selected for intervention would then have personal relevance to the ADHD individual and his socio-familial world.

Other practical considerations involve the use of stimulant drugs with the adolescent ADHD population and, particularly, with adolescents who show ADHD co-existing with CD. Clinicians have remarked that ADHD adolescents, as well as their CD counterparts, are relatively unaware of their symptomatology and its impact on others (e.g. Gittelman & Mannuzza, 1985; Wender, Wood, & Reimherr, 1985). It follows, therefore, that ADHD adolescents, with or without associated CD, may be imperceptive to changes in symptoms produced by stimulant medication and more disposed to discontinue treatment than are their prepubertal counterparts who are under closer parental supervision. Thus, the issue of compliance with the adolescent ADHD population is of critical concern. The researcher or clinician may choose to consider the usefulness of specific interventions aimed at increasing

compliance (Wender et al., 1985). ADHD adolescents with concurrent CD may also present with multiple problems. In addition to ADHD and CD symptomatology, these may include learning disabilities, associated depression, substance abuse, peer difficulties, and significant family dysfunction. Clearly, the use of stimulant therapy would be contraindicated in cases where there is pre-existing substance abuse, and/or when parents or siblings have substance abuse disorders. The presence of significant problems in other areas would mandate the use of multiple treatment modalities. Treatment may need to address a variety of deviant behaviours and a single technique or procedure may be inherently limited in its effects. Whalen and Henker (1991) have recently provided an excellent treatise on this issue.

As a concluding remark, it is hoped that the foregoing suggestions can be profitably employed as building blocks by those who choose to embark upon the empirical 'grandeur' of comprehensive, multi-outcome, intrasubject replication design strategies. In this scheme of things, the present study provides but one more example of 'findings on a small scale'. Current findings did not support the superiority of stimulant medication over placebo as a treatment option for the mixed ADHD/CD adolescent subtype. However, the discussed lack of consensual criteria in identifying favourable responders, and the problems associated with the failure to observe baseline deficits on certain tasks, precluded the ability to draw firm conclusions. Permit me to say "I know that favourable responders are in there, somewhere". To paraphrase Swanson (1989), it is a statement of the complexity of the problems posed by the ADHD syndrome, and its overlap with CD, that a firm characterization of a favourable and a non-favourable drug response pattern has not yet been established. As the practice of maintaining adolescents on psychostimulants increases in popularity, and the financial costs of alternate interventions become progressively more prohibitive, meeting the need for careful and systematic assessment of drug effects in the ADHD population becomes even more obligatory. In view of the particularly guarded prognosis for the mixed ADHD/CD adolescent subgroup, additional studies investigating

the potential efficacy of pharmacotherapy for this subtype would be important in providing empirical bases for selection of treatment options and planning of multimodal intervention designs.

PART E

APPENDICES

Appendix A
INTERVIEW SCHEDULE

Ask the patient the following questions and please circle the answer.

1. Has a doctor ever told you or your parents that you were hyperactive (or overactive, or "hyper" or as having Attention Deficit Disorder)?

   Yes No Unsure

2. Have you ever been prescribed methylphenidate which is a drug also known as Ritalin?

   Yes No Unsure

(If child indicates "no" or "unsure", please inquire as to whether a physician has ever prescribed amphetamine or caffeine for hyperactive symptoms. If the patient remembers being prescribed a drug, but does not recall its brand name, inquire as to how many times he took the drug daily and what the drug or pill looked like).

If the answer is YES to any of the above, ask how long a period of time the patient was on the drug.

3. Have you ever been brought to a mental health professional for problems with overactivity, being "hyper", or for difficulty paying attention for extended periods of time?

   Yes No Unsure

Go on to next page.....

DSM-III CRITERIA FOR CURRENT ASSESSMENT OF ADD(H)

The child displays, for his mental and chronological age, signs of developmentally inappropriate inattention, impulsivity and hyperactivity. Because the symptoms are typically variable, they may not be observed by the clinician. Symptoms typically worsen in situations that require self-application, as in the classroom. Signs of the disorder may be absent when the child is in a new or a one-to-one situation.

When assessing ADD(H), always keep in mind the following:

THE NUMBER OF SYMPTOMS SPECIFIED IS FOR CHILDREN BETWEEN THE AGES OF 8 AND 10. IN YOUNGER CHILDREN, MORE SEVERE FORMS OF THE SYMPTOMS AND A GREATER NUMBER ARE USUALLY PRESENT. THE OPPOSITE IS TRUE OF OLDER CHILDREN. THE MANIFESTATIONS OF SYMPTOMATOLOGY MAY CHANGE WITH AGE, THEREFORE DEVELOPMENTALLY APPROPRIATE QUESTIONS MAY NEED TO BE ASKED.

SEVERITY OF THE BEHAVIOUR should be rated in terms of how much it poses a problem for the patient, his/her parents or family, and/or the degree of disruption in performance at school.

SEVERITY SCALE:

0 Was not a problem
1 Mild problem
2 Moderate problem
3 Severe problem

For each item, rate the severity in the corresponding column in terms of the patient's CURRENT symptomatology.

ATTENTION: At least 3 of the following

_____ 1. Fails to finish things he or she starts.

_____ 2. Often doesn't seem to listen.

_____ 3. Easily distracted.

_____ 4. Difficulty concentrating on school work or other tasks requiring sustained attention.

_____ 5. Has difficulty sticking to a play activity

IMPULSIVITY: At least 3 of the following

_____ 1. Acts before thinking.

_____ 2. Shifts excessively from one activity to another.

_____ 3. Difficulty organizing work (not due to cognitive impairment).

_____ 4. Needs a great deal of supervision

_____ 5. Frequently calls out in class.

_____ 6. Problems awaiting turn in games/group situations.

HYPERACTIVITY: At least 2 of the following

_____ 1. Runs about or climbs on things excessively.

_____ 2. Difficulty sitting still or fidgets excessively.

_____ 3. Difficulty staying seated.

_____ 4. Moves about excessively during sleep.

_____ 5. Is always "on the go" or acts as if "driven by a motor".

ONSET before the age of 7.

Yes No Unsure

DURATION of at least 6 months in childhood.

Yes No Unsure

NOT DUE to schizophrenia, affective disorder, or mental retardation.

Yes No Unsure

SUBJECT DIAGNOSIS:

Meets criteria for ADD without Hyperactivity - Present _____
Meets criteria for ADD with Hyperactivity - Present _____

ADD(H) Diagnosis:
Mild _____
Moderate_____
Severe _____

Please proceed to the DSM-III criteria for CONDUCT DISORDER

Go on to next page.....

DSM-III CRITERIA FOR ASSESSMENT OF CONDUCT DISORDER

SEVERITY SCALE:

0 Was not a problem
1 Mild problem
2 Moderate problem
3 Severe problem

AGGRESSIVE TYPE: At least 1 of the following

_____ 1. Physical violence against persons or property (not defend oneself) (e.g., assault, vandalism)

_____ 2. Thefts outside the home involving confrontation with the victim (e.g., extortion, purse-snatching)

NONAGGRESSIVE TYPE: At least 1 of the following

_____ 1. Chronic violations of a variety of important rules (that are reasonable and age-appropriate) at home or at school (e.g., persistent truancy, substance abuse)

_____ 2. Repeated running away from home overnight

_____ 3. Persistent serious lying in and out of the home

_____ 4. Stealing not involving confrontation with the victim

For each item mark a check in the corresponding column if the behaviour is present

UNDERSOCIALIZED TYPE: No more than 1 of the following

SOCIALIZED: At least 2 of the following

_____ 1. Has one or more peer-group friendships that have lasted over 6 months

_____ 2. Extends himself for others even when no immediate advantage is likely

_____ 3. Apparently feels guilt or remorse when appropriate

_____ 4. Avoids blaming or informing on companions

_____ 5. Shows concern for the welfare of friends or companions

DURATION of pattern of conduct of at least 6 months

Yes No Unsure

DOES NOT MEET the criteria for Antisocial Personality Disorder, if 18 years age or older.

Yes No Unsure

SUBJECT DIAGNOSIS

Patient meets the criteria for Conduct Disorder?

Yes No

Patient meets the criteria for which of the following subtypes?

Very Mild:	Socialized Nonaggressive	_____
Mild:	Undersocialized Nonaggressive	_____
Moderate:	Socialized Aggressive	_____
Severe:	Undersocialized Aggressive	_____

Appendix B

INTERVIEW SCHEDULE - PARENT VERSION

Ask the parent the following questions and please circle the answer.

1. Has a doctor ever told you that your child was or is hyperactive (or overactive, or "hyper" or as having Attention Deficit Disorder)?

   Yes No Unsure

2. Has he ever been prescribed methylphenidate which is a drug also known as Ritalin?

   Yes No Unsure

   (If parent indicates "no" or "unsure", please inquire as to whether a physician has ever prescribed amphetamine or caffeine for hyperactive symptoms. If the parent remembers being prescribed a drug, but does not recall its brand name, inquire as to how many times the child took the drug daily and what the drug or pill looked like.)

   If the answer is YES to any of the above, ask how long a period of time the patient was on the drug.

3. Did you ever bring your child to a mental health professional for problems with overactivity, being "Hyper", or for difficultly paying attention for extended periods of time?

   Yes No Unsure

Go on to next page.....

Interview Questions for the Assessment of ADDH

Please use the following questions when assessing the presence or absence of ADDH symptomology so the standardization of the interview is assured. Go through the list of criteria twice; first assessing RETROSPECTIVELY (when the patient was 4 to 10 years old) and then assessing in terms of CURRENT symptomatology (now or within the past 6 months).

SUGGESTED PROBES:

How often has your son had these difficulties?

Has this led to difficulties in school or with other people?

How much of a problem (has that been/has that created) for your son and your family?

CURRENT STATUS:

Does your son have this (these) problems presently and over the past 6 months?

Does he still tend to _____?

WHEN INTRODUCING THE FORMAT OF THE FOLLOWING QUESTIONS, PLEASE POSE THREE ALTERNATIVES TO THE PARENT/GUARDIAN. THE INTERVIEWEE SHOULD COMPARE HIS OR HER CHILD TO THE AVERAGE CHILD HIS AGE AND, FOR EACH QUESTION, TO ANSWER WHETHER THE BEHAVIOUR IS NOT TRUE, IS SOMETIMES OR SOMEWHAT TRUE, OR IS VERY OR OFTEN TRUE OF THE CHILD.

ATTENTIONAL DIFFICULTIES:

1. Has your child had trouble finishing things he started? (Has he tended to start several things at one time and had trouble finishing any?)

   Not True Sometimes True Very True

2. Did it seem as though his mind was frequently "somewhere else"? Did you or his teachers complain that he didn't listen to you?

   Not True Sometimes True Very True

3. What about at work or school? Was he easily distracted? (Was it difficult for him to keep his mind on things he had to do?)

   Not True Sometimes True Very True

4. Did he have trouble concentrating or paying attention to things? For example, as a child in school, did he get fidgety or want to stand-up and run around or leave during the class lesson? Did he have problems keeping his mind on a conversation or on reading material that was interesting to him? Did you, his teachers or friends often complain that he wasn't paying attention?

   Not True Sometimes True Very True

5. Did he have problems as a child sticking to a play activity or watching a long TV programme?

Not True Sometimes True Very True

IMPULSIVITY

1. Did he tend to act on things immediately (right away)?
   Did he tend to make decisions too quickly and too easily without thinking them through?
   Did he often get involved in things or make decisions that he later regretted?

Not True Sometimes True Very True

2. Did he have difficulty sticking to one activity?

Not True Sometimes True Very True

3. Did he have difficulty organizing or planning his work in school or other types of work such as family chores?

Not True Sometimes True Very True

4. Did he require a great deal of supervision?

Not True Sometimes True Very True

5. Did he often call out in class or speak when it wasn't his turn?
   Have you or his teachers often referred to him as being impatient?

Not True Sometimes True Very True

6. Did he have a hard time awaiting his turn in games or waiting in lines?

Not True Sometimes True Very True

7. Did he tend to get involved in activities without thinking about or recognizing the risk involved, such as the possibility of harming himself or getting punished?

Not True Sometimes True Very True

HYPERACTIVITY:

1. As a young child, did he have an extreme amount of energy so that he was running around a lot or often climbing up on things?

Not True Sometimes True Very True

2. Was he very restless, fidgety or unable to sit still?

Not True Sometimes True Very True

3. Did he have difficulty staying seated for a period of time, or sticking to quiet activities like reading, watching TV or listening in class?

Not True Sometimes True Very True

5. Has he been always "on the go" as though he was "driven by a motor"?

Not True Sometimes True Very True

SUBJECT DIAGNOSIS:

Meets criteria for ADDH - past _____

ADDH Diagnosis Current
Mild _____
Moderate _____
Severe _____

Go on to next page.....

Interview Questions for the Assessment of Conduct Disorder

AGGRESSIVE TYPE:

1. What is your son charged with?
   Has he ever committed various crimes - whether he was charged or not?
   How many times?

2. Has he ever harmed anyone physically or come close to harming anyone?
   How often does he get into fist fights?

3. Has he ever committed any crimes which might have involved contact with the victim (e.g., purse-snatching or breaking and entering when the occupant was home)?

NONAGGRESSIVE TYPE:

1. About how often does or did he skip school?
   Do you know what sort of drugs he has taken?
   About how often does or did he use _____ drugs?
   Does he have difficulty keeping the rules at home?

2. Does he run away from home overnight repeatedly? How often?

3. Do you find that he is often telling lies to cover up things he has done?

4. Do you know how often he steals - whether he has been charged with stealing or not?

SOCIALIZED/UNSOCIALIZED TYPE:

1. About how many friends does he have?
   How many of them has he known for a period of 6 months or more?

2. How does he feel about helping others when there is nothing in it for him?

3. Do you think he feels badly after committing a crime?
   Why do you think so?

4. Does he avoid blaming his friends or telling on companions?

5. Does he show concern for the welfare of his friends or companions?

SUBJECT DIAGNOSIS:

Yes No

CD Diagnosis:

Mild (socialized & Undersocialized Nonaggressive) _____
Moderate (Socialized Aggressive) _____
Severe (Undersocialized Aggressive) _____

Appendix C
Patient Information

Dr. Emlene Murphy of Juvenile Services to the Courts and Erica Reznick, a doctoral candidate in clinical psychology at Simon Fraser University, are conducting clinical drug trials to examine the effectiveness of a drug called Ritalin in treating adolescents with behaviour problems associated with a history of childhood attention deficit disorder with hyperactivity.

Children or adolescents with hyperactivity or attention deficit disorder usually have difficulty in the following areas: paying attention and concentrating, controlling impulsive behaviour - acting before they stop to think, and controlling their activity level. The Ritalin medication you will be receiving is prescribed by your doctor at the In-patient Assessment Unit and is helpful in treating persons who have difficulties in the above described areas. The medication is not addictive. The possible side-effects are stomachaches and fatigue, decreased appetite and sleep problems. If you do experience any of the above side-effects, we ask you to notify the nurse. Do not be alarmed, as most side-effects are eliminated when the dosage is decreased. If you have any further questions about the medication, please ask the nurse or doctor.

The study will also involve the administration of some psychological tests, none of which are harmful or invasive. You will be asked to complete a questionnaire and to participate individually in some computer games. Depending upon your performance on the computer tasks, you could win up to $10.00. The money you win will be paid to you upon your release from the In-patient Assessment Unit.

At the end of the study, the doctor and Ms. Reznick will discuss the results with you. The findings may be included in the doctor's recommendations to the court.

Please note that, should you consent to participate and later have serious complaints such that you would like to withdraw from the study, the above described procedures will be terminated at the time of your request.

Thank you for your time and cooperation.

To: Parents,

Your child, who is attending the Outpatient Unit of Juvenile Services to the Courts, is noted to have a history of hyperactivity in childhood. Children with hyperactivity (also known as attention deficit disorder) are considered to have the following problems:

1. Inattention: they often fail to finish things they start and are easily distracted.

2. Impulsivity: they often act before thinking and shift from one activity to another.

3. Hyperactivity: they run about excessively and have difficulty being still.

One medication which has been helpful in treating children and adolescents with this disorder is methylphenidate, commonly known as Ritalin. This drug has been quite effective in treating a large group of hyperactive children and has been sagely used for many years. The most common effects are helping these children and/or adolescents to better pay attention and to control impulsive behaviour. Possibly your child has been previously treated with this drug. We are presently conducting clinical drug trials with hyperactive adolescents at the Inpatient Assessment Unit and we would like to assess the possible beneficial effects of Ritalin on your child's' behaviour.

As with any drug, there is a possibility of side effects. The most common side effects with Ritalin are upset stomach and sleeplessness at night. The drug is not addictive. There are no known long-term side effects. (Please see enclosed information on the most frequently asked questions about Ritalin medication).

Your child would be administered the drug for a total of 4 to 6 days under the supervision of a physician. Nursing staff will be available 24 hours a day to monitor any possible ill-effects. If side effects are severe, your child will be taken off the drug. Should your child show a beneficial response to the drug, this may be included in the psychiatrist's recommendations to the court.

We therefore kindly request your consent to conduct clinical drug trials with your child. If you consent, we ask you to please complete the enclosed consent form. A written consent will also be obtained from your child. We would also appreciate your completing the enclosed questionnaire and mailing it back to us.

The information you can provide us with is extremely important. Once the clinical drug trials have been completed, a form will be mailed to you indicating whether or not your child responded to this medication.

Should you have any questions about this letter, please contact the social worker at Juvenile Services to the Courts. Our phone number is 660- 5788 and our hours are Monday to Friday, 8:30 AM to 4:30 PM.

Enclosed is an information package on hyperactivity and Ritalin drug therapy.

Sincerely yours,

E. Murphy, M.D. F.R.C.P. (C)
Psychiatrist
Juvenile Services to the Courts
Inpatient Assessment Unit
Forensic Psychiatric Services Commission

Go on to next page.....

Commonly Asked Questions About Ritalin Drug Treatment

Is Hyperactivity a real disorder or is it only in the eyes of the beholder?

> Years ago, people argued that hyperactivity itself is a myth. However, there is now objective documentation of the learning and performance difficulties hyperactive children and adolescents experience.

Doesn't hyperactivity disappear in adolescence?

> It was earlier believed that the hyperactive child's problems were age-limited or time-limited; however, this has not been supported by studies following hyperactive children into adolescence and adulthood. Although it appears that some children with hyperactivity may outgrow their problems, many of them continue to have social and academic difficulties later in life.
>
> The problems also tend to change in form or in mode of expression, for example, impulsive behaviour may be expressed during the preschool years as accident-proneness, during the grade school years as problems in tolerating frustration and getting along with classmates and friends, and during adolescence as trouble with the law.

Isn't drug treatment reaching epidemic proportions?

> Much media coverage and public outcry against drug treatment for hyperactivity during the early 1970s was based on the incorrect assumption that perhaps ten percent of United States school children were receiving drugs for behavioural control. Rather, recent studies show that only between one and two percent of school-aged children are receiving drug treatment for hyperactivity.

Do all hyperactive children show the same response to Ritalin?

> For many children considered to be hyperactive, medication appears to improve their behaviour. The major effects of the drug seem to be a better ability to focus and to sustain attention, and to control impulsive behaviour. The main benefit of Ritalin treatment is that it helps the child or adolescent to "Stop, look and listen".

However, not all hyperactive children show the same response to drug therapy. For one child, improvement might be seen in less squirming and less fidgety behaviour, for another child improvement might be seen in a better ability to concentrate, whereas for still another child, improvement might mean smoother social relations.

Will Ritalin medication improve the intelligence of a hyperactive child?

No. No drug can directly improve intelligence. However, studies show that Ritalin can improve the performance of hyperactive children on learning and school-related tasks. It is believed that this is a result of the drug's effect in improving the ability to concentrate and to pay attention.

Does Ritalin sedate the child?

No. Research suggests that the drug does not slow children down so much as enable the child to better regulate their behaviour, to concentrate, to pay attention and to think before they act.

What are the possible drug side effects?

Decreased appetite and sleep problems are the most frequently reported side effects. Other effects include headaches, stomachaches and fatigue. However, most side effects are eliminated when the dosage is decreased.

What about diet treatment for hyperactivity?

At present, a number of dietary treatment approaches have been recommended for children and adolescents with hyperactivity. These have included eliminating food additives from the diet and changing the vitamin or sugar content in the diet. However, there is very little, if any, scientific support for the use of dietary treatments as part of an intervention programme for hyperactive children and adolescents. Most of the proposed treatment programmes have received little scientific investigation and the little research that has been done has produced no clinically important or impressive results.

Appendix D

Juvenile Services to the Courts Inpatient Assessment Unit

Consent to Participate

in

Research Study: Hyperactivity - Attention Deficit Disorder

(youth)

I, _____, presently attending the Outpatient Assessment

Unit of Juvenile Services to the Court, declare as follows:

a) THAT I have read the attached information sheet which describes the research study on hyperactivity and the use of the drug Ritalin and I have had the contents explained to me by _____;
(name of Psychiatrist)

b) THAT I understand that the assessment indicates that I have a history of hyperactivity in my childhood;

c) THAT I understand the possible side effects that I may experience if I take the drug Ritalin;

d) THAT the drug will be administered for a period of six (6) days under the supervision of a doctor with nursing supervision on a 24-hour basis.

e) THAT I consent to participate in this research study and authorize the administration of the drug Ritalin.

Date:_____

_____ _____
(Signature of Youth) (Signature of Psychiatrist)

Juvenile Services to the Courts Inpatient Assessment Unit

Consent to Participate

in

Research Study: Hyperactivity - Attention Deficit Disorder

(parent/legal guardian)

I, _____, parent or legal guardian of

_____, who is presently attending the Outpatient

assessment Unit of Juvenile Services to the Courts, declare as follows:

a) THAT I have read the attached information sheet that describes and explains the research study on hyperactivity (also known as attention deficit disorder) and the use of the drug methylphenidate (Ritalin) in the treatment of this disorder;

b) THAT the assessment of the above named youth indicates a history of hyperactivity in childhood;

c) THAT I am aware of the possible side effects of the use of the drug which are explained in the attached information sheet;

d) THAT the drug will be administered for a period of six (6) days under the supervision of a qualified medical practitioner with nursing supervision on a 24-hour basis.

e) THAT the drug will only be administered on the written and informed consent of the youth;

f) THAT I will be advised whether or not the youth responded to this medication; and

e) THAT I, in my capacity of parent/legal guardian, do hereby consent to participation in this research study and authorize the administration of the drug methylphenidate (Ritalin) to the youth on the conditions set out in this consent form.

Date:_____

_____ (Signature of Parent/Guardian)

_____ (Signature of Psychiatrist)

Appendix E

Side Effects Questionnaire

Name of Patient_____ Subject # _____

Time Now:_____ Date:_____

Completed by:_____ Rater #:_____

Reliability being assessed? (Y / N) _____

Reliability Rater:_____ Rater #:_____

Rate each behaviour from "0" (absent) to "9" (serious). Circle only one number beside each item. "0" means that you have not seen this behaviour in the patient during the observation period (the past day) and "9" means that you have noticed it and believe it to be very serious or to occur frequently.

Behaviour	Absent									Serious
Insomnia	0	1	2	3	4	5	6	7	8	9
Nightmares	0	1	2	3	4	5	6	7	8	9
Stares alot/daydreams	0	1	2	3	4	5	6	7	8	9
Talks less with others	0	1	2	3	4	5	6	7	8	9
Uninterested in others	0	1	2	3	4	5	6	7	8	9
Decreased appetite	0	1	2	3	4	5	6	7	8	9
Irritable	0	1	2	3	4	5	6	7	8	9
Stomachaches	0	1	2	3	4	5	6	7	8	9
Headaches	0	1	2	3	4	5	6	7	8	9
Drowsiness	0	1	2	3	4	5	6	7	8	9
Sad/unhappy	0	1	2	3	4	5	6	7	8	9
Prone to crying	0	1	2	3	4	5	6	7	8	9
Anxious	0	1	2	3	4	5	6	7	8	9
Bites nails	0	1	2	3	4	5	6	7	8	9
Euphoric	0	1	2	3	4	5	6	7	8	9
Dizziness	0	1	2	3	4	5	6	7	8	9

Appendix F

ANOVA Source Table: Main Effects of Drug Order

Dependent Variable	Mean Square	Mean Square Error	df	*F*-value	*p*
Vigilance Task:					
Omission Errors	28.88	57.78	5,22	0.50	.773
Commission Errors	46.44	43.36	5,22	1.07	.403
Distractibility Task:					
Omission Errors	227.16	193.30	5,22	1.18	.352
Commission Errors	78.54	46.00	5,22	1.71	.174
Delay (DRL) Task:					
Efficiency Ratio	0.25	0.43	5,22	0.57	.723
Passive Avoidance Task:					
Omission Errors	309.49	522.34	5,24	0.59	.705
Commission Errors	66.51	72.65	5,24	0.92	.487

Appendix G

ANOVA Source Table: Drug Order x Experimental/Reward Condition Interactions

Dependent Variable	Mean Square	Mean Square Error	df	*F*-value	*p*
Vigilance Task:					
Omission Errors	11.99	17.70	15,66	0.68	.751
Commission Errors	12.69	6.89	15,66	1.84	.068
Distractibility Task:					
Omission Errors	25.58	29.78	15,66	0.86	.610
Commission Errors	14.41	11.60	15,66	1.24	.264
Delay (DRL) Task:					
Efficiency Ratio	0.23	0.30	15,66	0.75	.730
Passive Avoidance Task:					
Omission Errors	30.59	31.21	15,72	0.98	.480
Commission Errors	36.91	25.45	15,72	1.45	.180
x Reward Programme:					
Omission Errors	67.78	29.65	5,24	2.29	.088
Commission Errors	15.55	22.71	5,24	0.68	.639
x Experimental Condition x Reward Programme:					
Omission Errors	31.81	31.54	15,72	1.01	.456
Commission Errors	19.64	23.34	15,72	0.84	.598

Appendix H

ANOVA Source Table: Main Effects of Experimenter

Dependent Variable	Mean Square	Mean Square Error	df	*F*-value	*p*
Vigilance Task:					
Omission Errors	234.17	45.44	1,26	5.15	.042
Commission Errors	46.88	43.82	1,26	1.07	.310
Distractibility Task:					
Omission Errors	520.01	187.25	1,26	2.78	.107
Commission Errors	189.75	46.73	1,26	4.06	.064
Delay (DRL) Task:					
Efficiency Ratio	0.17	0.41	1,26	0.34	.566
Passive Avoidance Task:					
Omission Errors	2162.42	425.76	1,28	5.08	.057
Commission Errors	3.13	74.04	1,28	0.04	.838

Appendix I

ANOVA Source Table: Experimenter x Experimental/Reward Condition Interactions

Dependent Variable	Mean Square	Mean Square Error	df	*F*-value	*p*
Vigilance Task:					
Omission Errors	3.53	17.15	3,78	0.21	.836
Commission Errors	0.18	8.26	3,78	0.02	.987
Distractibility Task:					
Omission Errors	24.50	29.17	3,78	0.84	.476
Commission Errors	4.31	12.42	3,78	0.35	.791
Delay (DRL) Task:					
Efficiency Ratio	0.57	0.28	3,78	2.03	.116
Passive Avoidance Task:					
Omission Errors	88.87	29.05	3,84	3.06	.063
Commission Errors	11.69	27.99	3,84	0.42	.648
x Reward Programme:					
Omission Errors	40.41	36.08	1,28	1.12	.298
Commission Errors	1.39	22.19	1,28	0.06	.803
x Experimental Condition x Reward Programme:					
Omission Errors	14.94	32.18	3,84	0.46	.704
Commission Errors	7.05	23.26	3,84	0.30	.737

Appendix J

ANOVA Source Table: Main Effects of Treatment Phases

Dependent Variable	Mean Square	Mean Square Error
Vigilance Task:		
Omission Errors	4.64	17.70
Commission Errors	6.39	6.89
Distractibility Task:		
Omission Errors	91.75	29.78
Commission Errors	9.88	11.60
Delay (DRL) Task:		
Efficiency Ratio	0.20	0.30
Passive Avoidance Task:		
Omission Errors	187.31	31.21
Commission Errors	203.73	25.45

Appendix K

ANOVA Source Table: Main Effects and Interaction Effects of Task Order

Dependent Variable	Mean Square	Mean Square Error	df	*F*-value	*p*
Passive Avoidance Task:					
Omission Errors	3.01	502.88	1,28	0.01	.938
Commission Errors	176.46	67.85	1,28	2.60	.118
x Experimental Conditions:					
Omission Errors	9.89	31.87	3,84	0.31	.796
Commission Errors	57.68	26.35	3,84	2.19	.122
x Reward Programme:					
Omission Errors	109.48	33.61	1,28	3.26	.091
Commission Errors	38.78	20.87	1,28	1.86	.183
x Experimental Condition x Reward Programme:					
Omission Errors	12.56	32.27	3,84	0.39	.760
Commission Errors	39.58	22.09	3,84	1.79	.174

Appendix L

ANOVA Source Table: Main Effects and Interaction Effects of Reward Programme

Dependent Variable	Mean Square	Mean Square Error	df	*F*-value	*p*
Passive Avoidance Task: Reward Programme:					
Omission Errors	4.36	29.65	1,24	0.15	.704
Commission Errors	0.26	22.71	1,24	0.01	.916
x Experimental Conditions:					
Omission Errors	43.85	31.54	3,72	1.39	.252
Commission Errors	57.68	26.35	3,72	1.07	.355

Appendix M

ANOVA Source Table: Main Effects of Treatment Phases

Placebo vs. Average of Drug Conditions

Dependent Variable	Mean Square	Mean Square Error	df	*F*-value	*p*
Vigilance Task:					
Omission Errors	0.93	3.17	1,21	0.03	.866
Commission Errors	0.23	2.27	1,21	0.01	.922
Distractibility Task:					
Omission Errors	22.19	17.72	1,21	1.25	.275
Commission Errors	14.59	14.45	1,21	1.01	.326
Delay (DRL) Task:					
Efficiency Ratio	0.92	0.79	1,21	0.12	.736
Passive Avoidance Task:					
Omission Errors	5.18	10.53	1,20	0.49	.491
Commission Errors	7.86	9.51	1,20	0.83	.374

Appendix N

Rotated Factor Loadings for Principal Components of Drug Effect Change Score Data

Variable[a]	Factor 1	Factor 2	Factor 3	Factor 4
Vigilance Task:				
Omission Errors	.72	.10	.45	.16
Commission Errors	.27	.10	.12	.81
Distractibility Task:				
Omission Errors	.84	.02	-.05	-.06
Commission Errors	.74	-.32	-.34	-.03
Delay (DRL) Task:				
Efficiency Ratio	.01	.89	-.08	-.12
Passive Avoidance Task:				
P-only Programme:				
Omission Errors	-.05	-.04	-.75	-.01
Passive Avoidance Task:				
P-only Programme:				
Commission Errors	.06	-.84	-.02	-.13
Passive Avoidance Task:				
R + P Programme:				
Omission Errors	-.09	-.12	.78	-.01
Passive Avoidance Task:				
R + P Programme:				
Commission Errors	.47	.14	.16	-.68
Variance Explained[b]	2.08	1.67	1.56	1.19

Note. P-only = Punishment Only
R + P = Reward and Punishment

[a] Change score = difference between the placebo score and the average of the low dose and the moderate dose drug conditions.

[b] The variance explained by each factor is the eigenvalue for that factor.

Appendix O

ANOVA Source Table: Main Effects of ADDH Severity Subject Grouping Variable

Dependent Variable	Mean Square	Mean Square Error	df	*F*-value	*p*
Vigilance Task:					
Omission Errors	0.54	11.45	1,23	0.05	8.29
Commission Errors	23.78	49.19	1,23	0.48	4.93
Distractibility Task:					
Omission Errors	247.51	94.52	1,23	2.62	.119
Commission Errors	9.75	59.05	1,23	0.17	.688
Delay (DRL) Task:					
Efficiency Ratio	0.29	0.44	1,23	0.07	.798
Passive Avoidance Task:					
Omission Errors	440.41	485.00	1,23	0.91	.350
Commission Errors	9.82	69.51	1,23	0.14	.710

Appendix P

ANOVA Source Table: ADDH Severity x Experimental/Reward Condition Interactions

Dependent Variable	Mean Square	Mean Square Error	df	*F*-value	*p*
Vigilance Task:					
Omission Errors	0.57	4.86	3,69	0.12	.948
Commission Errors	24.94	7.79	3,69	3.20	.033
Distractibility Task:					
Omission Errors	26.23	20.97	3,69	1.25	.298
Commission Errors	17.45	12.80	3,69	1.36	.261
Delay (DRL) Task:					
Efficiency Ratio	0.72	0.26	3,69	2.74	0.49
Passive Avoidance Task:					
Omission Errors	47.84	26.49	3,69	1.81	.155
Commission Errors	25.86	29.39	3,69	0.88	.418
x Reward Programme:					
Omission Errors	114.87	27.82	1,23	4.13	.054
Commission Errors	0.33	22.75	1,23	0.01	.905
x Experimental Condition x Reward Programme:					
Omission Errors	6.47	31.84	3,69	0.20	.893
Commission Errors	25.90	24.03	3,69	1.08	.351

Appendix Q

ANOVA Source Table: Main Effects of CD Severity Subject Grouping Variable

Dependent Variable	Mean Square	Mean Square Error	df	*F*-value	*p*
Vigilance Task:					
Omission Errors	3.96	11.71	1,24	0.34	.567
Commission Errors	0.33	48.35	1,24	0.01	.935
Distractibility Task:					
Omission Errors	253.87	91.36	1,24	2.78	.108
Commission Errors	1.55	57.21	1,24	0.03	.870
Delay (DRL) Task:					
Efficiency Ratio	0.32	0.43	1,24	0.01	.931
Passive Avoidance Task:					
Omission Errors	355.14	478.81	1,24	0.74	.397
Commission Errors	1.76	67.27	1,24	0.03	.873

Appendix R

ANOVA Source Table: CD Severity x Experimental/Reward Condition Interactions

Dependent Variable	Mean Square	Mean Square Error	df	*F*-value	*p*
Vigilance Task:					
Omission Errors	6.42	4.60	3,72	1.40	.251
Commission Errors	4.03	8.47	3,72	0.48	.657
Distractibility Task:					
Omission Errors	6.68	22.29	3,72	0.30	.825
Commission Errors	4.27	13.36	3,72	0.32	.811
Delay (DRL) Task:					
Efficiency Ratio	0.81	0.29	3,72	0.27	.846
Passive Avoidance Task:					
Omission Errors	18.73	28.47	3,72	0.66	.578
Commission Errors	2.56	29.33	3,72	0.09	.911
x Reward Programme:					
Omission Errors	35.05	31.09	1,24	1.13	.298
Commission Errors	0.66	22.13	1,24	0.00	.987
x Experimental Condition x Reward Programme:					
Omission Errors	40.32	32.54	3,72	1.24	.302
Commission Errors	18.09	24.29	3,72	0.74	.475

Appendix S

ANOVA Source Table: Main Effects of Prior Diagnosis of Childhood ADDH Subject Grouping Variable

Dependent Variable	Mean Square	Mean Square Error	df	*F*-value	*p*
Vigilance Task:					
Omission Errors	15.59	11.94	1,25	1.31	.264
Commission Errors	0.19	46.70	1,25	0.00	.949
Distractibility Task:					
Omission Errors	396.41	84.72	1,25	4.68	.040
Commission Errors	2.61	55.66	1,25	0.05	.830
Delay (DRL) Task:					
Efficiency Ratio	0.12	0.41	1,25	0.28	.604
Passive Avoidance Task:					
Omission Errors	98.08	393.22	1,24	0.24	.629
Commission Errors	1.98	5.94	1,24	0.03	.865

Appendix T

ANOVA Source Table: Prior Diagnosis of Childhood ADDH x Experimental/Reward Condition Interactions

Dependent Variable	Mean Square	Mean Square Error	df	*F*-value	*p*
Vigilance Task:					
Omission Errors	2.56	5.29	3,75	0.48	.694
Commission Errors	15.23	7.70	3,75	1.98	.134
Distractibility Task:					
Omission Errors	28.07	22.94	3,75	1.22	.307
Commission Errors	2.84	12.91	3,75	0.22	.882
Delay (DRL) Task:					
Efficiency Ratio	0.84	0.29	3,75	0.29	.835
Passive Avoidance Task:					
Omission Errors	48.72	25.59	3,72	1.90	.137
Commission Errors	18.06	29.33	3,72	0.62	.541
x Reward Programme:					
Omission Errors	17.59	31.83	1,24	0.55	.465
Commission Errors	7.95	22.18	1,24	0.36	.555
x Experimental Condition x Reward Programme:					
Omission Error	4.10	35.94	3,72	0.11	.951
Commission Errors	5.59	24.89	3,72	0.22	.8061

PART F

REFERENCES

Abikoff, H., & Gittelman, R. (1985). Hyperactive children treated with stimulants. Is cognitive training a useful adjunct? *Archives of General Psychiatry, 42*, 953-961.

Achenbach, T.M. (1978). The Child Behavior Profile: I. Boys aged 6-11. *Journal of Consulting and Clinical Psychology, 46*, 478-488.

Achenbach, T.M., & Edelbrock, C.S. (1978). The classification of child psychopathology: A review and analysis of empirical efforts. *Psychological Bulletin, 85*, 1275-1301.

Achenbach, T.M., & Edelbrock, C.S. (1983). *Manual for the child behavior checklist and revised child behavior profile.* U.S.A.: Queen City Printers.

Ackerman, P.T., Dykman, R.A., & Peters, J.E. (1977). Teenage status of hyperactive and nonhyperactive learning disabled boys. *American Journal of Orthopsychiatry, 41*, 577-596.

Amado, H., & Lustman, P.J. (1982). Attention deficit disorder persisting in adulthood: A review. *Comprehensive Psychiatry, 33*, 300-314.

Aman, M.G. (1978). Drugs, learning, and the psychotherapies. In J.S. Werry (Ed.), *Pediatric psychopharmacology: The use of behavior-modifying drugs in children.* New York: Brunner/Mazel.

Aman, M.G. (1984). Hyperactivity: Nature of the syndrome and its natural history. *Journal of Autism and Developmental Disorders, 14*, 39-56.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.). Washington, D.C.: Author.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, D.C.: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed. - Revised). Washington D.C.: Author.

Amery, B., Minichiello, M.D., & Brown, G.L. (1984). Aggression in hyperactive boys: Response to d-amphetamine. *Journal of the American Academy of Child Psychiatry, 23*, 291-294.

Amsel, A. (1962). Frustrative nonreward in partial reinforcement and discrimination learning. Some recent history and a theoretical extension. *Psychological Review, 69*, 306-328.

Anderson, R., Halcomb, C., & Doyle, R. (1973). The measurement of attentional deficits. *Exceptional Children, 39*, 534-540.

Anderson, R., Halcomb, C., Gordon, W., & Ozolins, D. (1974). Measurement of attention distractibility in LD children. *Academic Therapeutic*, 9, 261-266.

Arnold, L.E., Christopher, J., Huestis, R., & Smeltzer, D.J. (1978). Methylphenidate vs. dextroamphetamine vs. caffeine in minimal brain dysfunction. *Archives of General Psychiatry, 35*, 463-473.

Arnold, L.E., Huestis, R., Smeltzer, D., Scheib, J., Wemmer, D., & Colner, G. (1976). Leroamphetamine versus dextroamphetamine in minimal brain dysfunction. *Archives of General Psychiatry , 33*, 292-301.

August, G.J., & Garfinkel, B.D. (1990). Comorbidity of ADHD and reading disability among clinic-referred children. *Journal of Abnormal Child Psychology, 18*, 29-45.

August, G.J., & Stewart, M.A. (1982). Is there a syndrome of pure hyperactivity? *British Journal of Psychiatry, 140*, 305-311.

August, G.J., & Stewart, M.A. (1983). Familial subtypes of childhood hyperactivity. *Journal of Nervous and Mental Disease, 71*, 362-368.

August, G.J., Stewart, M.A., & Holmes, C. (1983). A four-year follow-up of hyperactive boys with and without conduct disorder. *British Journal of Psychiatry, 143*, 192-198.

Bakwin, H., & Bakwin, R. (1966). *Clinical management of behavior disorders in children.* Philadelphia: W.B. Saunders Co.

Barkley, R.A. (1976). Predicting the response of hyperkinetic children to stimulant drugs: A review. *Journal of Abnormal Child Psychology, 4*, 327-348.

Barkley, R.A. (1977). A review of stimulant drug research with hyperactive children. *Journal of Child Psychology and Psychiatry, 18*, 137-165.

Barkley, R.A. (1981). *Hyperactive children: A handbook of diagnosis and treatment.* New York: Guilford.

Barkley, R.A. (1982). Guidelines for defining hyperactivity in children. In B.B. Lahey & A.E. Kazdin (Eds.), *Advances in clinical child psychology (Vol. 5).* New York: Plenum Press.

Barkley, R.A. (1991). The ecological validity of laboratory and analogue assessment methods of ADHD symptoms. *Journal of Abnormal Child Psychology, 19*, 149-178.

Barkley, R.A., & Cunningham, C.E. (1980). The parent-child interactions of hyperactive children and their modification by stimulant drugs. In R.M. Knights & D.J. Bakker (Eds.), *Treatment of hyperactive and learning disordered children* (pp. 219-236). Baltimore: University Park Press.

Barkley, R.A., Fischer, M., Newby, R.F., & Breen, M.J. (1988). Development of a multimethod clinical protocol for assessing stimulant drug response in children with attention deficit disorder. *Journal of Clinical Child Psychology, 17*, 14-24.

Barkley, R.A., Karlsson, J., Pollard, S., & Murphy, J.V. (1985). Developmental changes in the mother-child interactions of hyperactive boys: Effects of two dose levels of Ritalin. *Journal of Child Psychology and Psychiatry, 26*, 705-715.

Barkley, R.A., Karlsson, J., Strzelecki, E., & Murphy, J.V. (1984). Effects of age and Ritalin dosage on the mother-child interactions of hyperactive children. *Journal of Consulting and Clinical Psychology, 52*, 750-758.

Barkley, R.A., McMurray, M.B., Edelbrock, C.S., & Robbins, K. (1989). The response of aggressive and nonaggressive ADD children to two doses of methylphenidate. *Journal of the American Academy of Child and Adolescent Psychiatry, 28*, 873-881.

Barkley, R.A., & Ullman, D.C. (1975). A comparison of objective measures of activity and distractibility in hyperactive and nonhyperactive children. *Journal of Abnormal Child Psychology, 3*, 231-244.

Bierderman, J., Munir, K., & Knee, D. (1987). Conduct and oppositional disorder in clinically referred children with attention deficit disorder: A controlled family study. *Journal of the American Academy of Child and Adolescent Psychiatry, 26*, 724-727.

Blackburn, R. (1983). Psychopathy, delinquency, and crime. In A. Gale & J.A. Edwards (Eds.), *Physiological correlates of human behavior* (pp. 187-203). New York: Academic Press.

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

Borland, B., & Heckman, H. (1976). Hyperactive boys and their brothers: A 25-year follow-up study. *Archives of General Psychiatry, 33*, 669-675.

Bosco, J.J., & Robin, S.S. (1980). Prevalence of treatment. In C.K. Whalen & B. Henker (Eds.), *The social ecology of identification and treatment* (pp. 173-187). New York: Academic Press.

Bradley, C. (1937). The behavior of children receiving Benzedrine. *American Journal of Psychiatry, 94*, 577-585.

Broder, P.K., Dunivant, N., Smith, E.C., & Sutton, L.P. (1981). Further observations on the link between learning disabilities and juvenile delinquency. *Journal of Educational Psychology, 73,* 838-850.

Brown, R.T., & Borden, K.A. (1986). Hyperactivity at adolescence: Some misconceptions and new directions. *Journal of Clinical Child Psychology, 15*, 194-209.

Brown, R.T., Borden, K.A., & Clingerman, S.R. (1985). Pharmacotherapy in ADD adolescents with special attention to multimodality treatments. *Psychopharmacology Bulletin, 21* , 192-211.

Brown, R.T., & Sexson, S.B. (1988). A controlled trial of methylphenidate in Black adolescents. *Clinical Pediatrics, 27*, 74-81.

Brown, R.T., & Sleator, E.K. (1979). Methylphenidate in hyperkinetic children: Differences in dose effects on impulsive behavior. *Pediatrics, 64*, 408-411.

Campbell, S.B. (1985). Early identification and follow-up of parent-referred "hyperactive" toddlers. In L.M. Bloomingdale (Ed.), *Attention deficit disorder: Identification, course and rationale (Vol. 2)* (pp. 1-16). New York: Spectrum.

Campbell., S.B., & Cluss, P. (1982). Peer relationships of young children with behavior problems. In K.H. Rubin & H.S. Ross (Eds.), *Peer relationships and social skills in childhood.* New York: Springer-Verlag.

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

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

Campbell, S.B., Schleifer, M., Weiss, G., & Perlman, T. (1971). A two-year follow-up of hyperactive preschoolers. *American Journal of Orthopsychiatry, 47*, 149-162.

Campbell, S.B., Szumowski, E.K., Ewing, L.J., Gluck, D.S., & Breaux, A.M. (1982). A multidimensional assessment of parent-identified behavior problem toddlers. *Journal of Abnormal Child Psychology, 10*, 569-591.

Cantwell, D.P. (1972). Psychiatric illness in the families of hyperactive children. *Archives of General Psychiatry, 21*, 414-417.

Cantwell, D.P. (1975). *The hyperactive child.* New York: Spectrum.

Cantwell, D.P. (1978). Hyperactivity and antisocial behavior. *American Academy of Child Psychiatry, 17*, 252-262.

Cantwell, D.P. (1979). Use of stimulant medication with psychiatrically disordered adolescents. In S.C. Feinstein & P.L. Giovacchini (Eds.), *Adolescent psychiatry (Vol. 2).* Chicago: University of Chicago Press.

Cantwell, D.P. (1981). Hyperactivity and antisocial behavior revisited: A critical review of the literature. In D.O. Lewis (Ed.), *Vulnerabilities to delinquency* (pp. 21-38). New York: Spectrum.

Cantwell, D.P. (1985a). Hyperactive children have grown up. What have we learned about what happens to them? *Archives of General Psychiatry, 42*, 1026-1028.

Cantwell, D.P. (1985b). Pharmacotherapy of ADD in adolescents: What do we know, where should we go, how should we do it? *Psychopharmacology Bulletin, 21*, 251-257.

Cantwell, D.P. (1986a). Attention deficit disorder in adolescents. *Clinical Psychology Review* (Special Issue: Psychopathology in Adolescence), **6**, 237-247.

Cantwell, D.P. (1986b). Attention deficit and associated childhood disorders. In T. Millon, & G.L. Klerman (Eds.), *Contemporary directions in psychopathology* (pp. 403-427). New York: Guilford Press.

Cantwell, D.P., & Carlson, G.A. (1978). Stimulants. In J.S. Werry (Ed.), *Pediatric psychopharmacology: The use of behaviour modifying drugs in children.* New York: Brunner/Mazel.

Carey, W.B. (1988). Clinical commentary. A suggested solution to the confusion in attention deficit diagnoses. *Clinical Pediatrics, 27*, 348-349.

Carins, E., & Cammock, T. (1978). Development of a more reliable version of the Matching Familiar Figures Test. *Developmental Psychology, 14*, 555-560.

Carlson, G.Z., & Cantwell, D.P. (1980). Unmasking masked depression in children and adolescents. *American Journal of Psychiatry, 137*, 445-449.

Chee, P., Logan, G., Schachar, R., Lindsay, P., & Wachsmuth, R. (1989). Effects of event rate and display time on sustained attention in hyperactive, normal, and control children. *Journal of Abnormal Child Psychology, 17*, 371-391.

Chesno, F.A., & Kilmann, P.R. (1975). Effects of stimulation intensity on sociopathic avoidance learning. *Journal of Abnormal Psychology, 84*, 144-150.

Clampit, M.K., & Pirkle, J.B. (1983). Stimulant medication and the hyperactive adolescent: Myths and facts. *Adolescence, XVIII*, 811-822.

Clements, S.D. (1966). Task Force One: Minimal brain dysfunction in children. *National Institute of Neurological Diseases and Blindness, Monograph No. 3,* U.S. Department of Health, Education, and Welfare.

Clements, S.D., & Peters, J.E. (1962). Minimal brain dysfunctions in the school age child. *Archives of General Psychiatry, 6*, 185-197.

Cohen, L. (1972). Attention-getting and attention-holding processes of infant visual preferences. *Child Development, 43*, 869-879.

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

Cohen, N.J., Weiss, G., & Minde, K. (1972). Cognitive style in adolescents previously diagnosed hyperactive. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 13*, 203-205.

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

Conners, C.K. (1970). Symptom patterns in hyperkinetic, neurotic and normal children. *Child Development, 41*, 667-782.

Conners, C.K. (1973). Rating scales for use in drug studies with children. *Psychopharmacology Bulletin* (Special Issue: Pharmacotherapy with Children), 24-88.

Conners, C.K. (1975). Minimal brain dysfunction and psychopathology in children. In A. Davids (Ed.), *Child personality and psychopathology: Current topics (Vol. 2).* New York: Wiley.

Conners, C.K. (1985). Issues in the study of adolescent ADDH/hyperactivity. *Psychopharmacology Bulletin, 21*, 243-250.

Conners, C.K., & Rothschild, G.H. (1986). Drugs and learning in children. In J. Hellmuth (Ed.), *Learning disorders (Vol. 3).* Seattle: Special Child Publications.

Conners, C.K., & Werry, J.S. (1979). Pharmacotherapy. In H.C. Quay & J.S. Werry (Eds.) (2nd Ed.), *Psychopathological disorders of childhood.* New York: Wiley and Sons.

Coons, H.W., Klorman, R., & Borgstedt, A.D. (1987). Effects of methylphenidate on adolescents with a childhood history of attention deficit disorder: II. Information processing. *Journal of the American Academy of Child and Adolescent Psychiatry, 26*, 368-374.

Copeland, A.P., & Wisniewski, N.M. (1981). Learning disability and hyperactivity: Deficits in selective attention. *Journal of Experimental Child Psychology, 32*, 88-101.

Cox, A., Rutter, M., Yule, B., & Quinlan, B. (1977). Bias resulting from missing information: Some epidemiological findings. *British Journal of Preventive Social Medicine, 31*, 131-136.

Cunningham, C.E., Siegel, L.S., & Offord, D.R. (1985). A developmental dose-response analysis of the effects of methylphenidate on peer interactions of attention deficit disordered boys. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 26*, 955-971.

Cunningham, C.E., Siegel, L.S., & Offord, D.R. (1991). A dose-response analysis of the effects of methylphenidate on the peer interactions and simulated classroom performance of ADD children with and without conduct problems. *Journal of Child Psychology and Psychiatry, 32*, 439-452.

Cunningham, S.J., & Knights, R.M. (1978). The performance of hyperactive and normal boys under differing reward and punishment schedules. *Journal of Pediatric Psychology, 3*, 195-201.

Davidson, E.M., & Prior, M.R. (1978). Laterality and selective attention in hyperactive children. *Journal of Abnormal Child Psychology, 6*, 475-481.

Davies, J.G., & Maliphant, R. (1974). Refractory behavior in school and avoidance learning. *Journal of Child Psychology and Psychiatry, 15*, 23-32.

Denhoff, E. (1973). The natural life history of children with minimal brain dysfunction. *The Annals of the New York Academy of Sciences, 205*, 188-206.

Denton, C.L., & McIntyre, C.W. (1978). Span of apprehension in hyperactive boys. *Journal of Abnormal Child Psychology, 6*, 475-481.

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

Douglas, V.I. (1980a). Higher mental processes in hyperactive children: Implications for training. In R.M. Knights & D.J. Bakker (Eds.), *Rehabilitation, treatment, and management of learning disorders.* Baltimore: University Park Press.

Douglas, V.I. (1980b). Treatment and training approaches to hyperactivity: Establishing internal or external control. In C.K. Whalen, & B. Henker (Eds.), *Hyperactive children: The social ecology of identification and treatment.* New York: Academic Press.

Douglas, V.I. (1983). Attention and cognitive problems. In M. Rutter (Ed.), *Developmental neuropsychiatry* (pp. 280-329). New York: Guilford Press.

Douglas, V.I. (1984). The psychological processes implicated in ADD. In L.M. Bloomingdale (Ed.), *Attention deficit disorder: Diagnostic, cognitive, and therapeutic understanding (Vol. 1)* (pp. 147-162). New York: Spectrum.

Douglas, V.I. (1985). The response of ADD children to reinforcement: Theoretical and clinical implications. In L.M. Bloomingdale (Ed.), *Attention deficit disorder: Identification, course and rationale (Vol. 2)* (pp. 49-66). New York: Spectrum.

Douglas, V.I. (1989). Can Skinnerian theory explain attention deficit disorder? - A reply to Barkley. In L.M. Bloomingdale & J.M. Swanson (Eds.), *Attention deficit disorder: Current concepts and emerging trends in attentional and behavioral disorders of childhood (Vol. 4)* (pp. 235-254). New York: Pergamon Press.

Douglas, V.I., Barr, R.G., Amin, K., O'Neill, M.E., & Britton, B.G. (1988). Dosage effects and individual responsivity to methylphenidate in attention deficit disorder. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 29*, 453-475.

Douglas, V.I., Barr, R.G., O'Neill, M.E., & Britton, B.G. (1986). Short-term effects of methylphenidate on the cognitive, learning and academic performance of children with attention deficit disorder in the laboratory and the classroom. *Journal of Child Psychology and Psychiatry, 27*, 191-211.

Douglas, V.I., & Parry, P.A. (1983). Effects of reward on delayed reaction time task performance of hyperactive children. *Journal of Abnormal Child Psychology, 11*, 313-326.

Douglas, V.I., & Peters, K.G. (1979). Toward a clearer definition of the attentional deficit of hyperactive children. In G.A. Hale & M. Lewis (Eds.), *Attention and the development of cognitive skills.* New York: Plenum Press.

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

Duffy, E. (1962). *Activation and behavior.* New York: Wiley & Sons.

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

Dykman, R.A., Peters, J.E., & Ackerman, P.T. (1973). Experimental approaches to the study of minimal brain dysfunction. *Annals of the New York Academy of Sciences, 205*, 93-108.

Eisenberg, L. (1966). The management of the hyperkinetic child. *Developmental Medicine and Child Neurology, 8,* 593-632.

Eisenberg, L., Lachman, R., Molling, P.A., Lockner, A., Mizelle, J.D., & Conners, C.K. (1963). A psychopharmacologic experiment in a training school for delinquent boys: Methods, problems, findings. *American Journal of Orthopsychiatry, 33*, 431-447.

Ferguson, H.B., & Rapoport, J.L. (1983). Nosological issues and biological validation. In M. Rutter (Ed.), *Developmental neuropsychiatry* (pp. 369-384). New York: Guilford Press.

Firestone, P., & Douglas, V.I. (1975). The effects of reward and punishment on reaction times and autonomic activity in hyperactive and normal children. *Journal of Abnormal Child Psychology, 3,* 201-216.

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

Fischer, M., Barkley, R.A., Edelbrock, C.S., & Smallish, L. (1990). The adolescent outcome of hyperactive children diagnosed by research criteria. II: Academic, attentional, and neuropsychological status. *Journal of Consulting and Clinical Psychology, 58,* 580-588.

Fish, B. (1971). The "one child, one drug" myth of stimulants in hyperkinesis. *Archives of General Psychiatry, 25,* 193-203.

Flavell, J.H. (1970). Developmental studies of mediated memory. In W.H. Reese & L.P. Lipsitt (Eds.), *Advances in child development and behavior (Vol. 5).* New York: Academic Press.

Freeman, R.J. (1978). *The effects of methylphenidate on avoidance learning and risk-taking by hyperkinetic children.* Unpublished doctoral dissertation, University of Waterloo, Ontario.

Freeman, R.J., & Kinsbourne, M. (1984). *A stimulant-correctable avoidance learning deficit in children with attention deficit disorder with hyperactivity: Commonality with psychopathic behavior.* Unpublished manuscript.

Freeman, R.J., & Reznick, E.D. (1984). *Attention deficit disorder, delinquency and antisocial personality: A developmental disorder of impulse control?* Unpublished manuscript.

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

Frick, P.J., Kamphaus, R.W., Lahey, B.B., Loeber, R., Christ, M.A., Hart, E.L., Tannenbaum, L. (1991). Academic underachievement and the disruptive behavior disorders. *Journal of Consulting and Clinical Psychology, 59,* 289-294.

Gadow, K.D. (1981). Prevalence of drug treatment for hyperactivity and other childhood behavior disorders. In K.D. Gadow & J. Loney (Eds.), *Psychosocial aspects of drug treatment for hyperactivity* (pp. 13-76). Boulder: Westview Press.

Garfinkel, B.D., Brown, W.A., Klee, S.H., Braden, W., Beauchesne, H., & Shapiro, S.K. (1986). Neuroendocrine and cognitive responses to amphetamine in adolescents with a history of ADD. *This Journal, 25,* 503-508.

Garfinkel, B.D., & Klee, S.H. (1985). Behavioral and personality characteristics of adolescents with a history of childhood ADD. In L.M. Bloomingdale (Ed.), *Attention deficit disorder: Identification, course and rationale (Vol. 2)* (pp. 17-32). New York: Spectrum.

Gillberg, I.C., & Gillberg, C. (1988). Generalized hyperkinesis: Follow-up study from age 7 to 13 years. *Journal of the American Academy of Child and Adolescent Psychiatry, 27*, 55-59.

Gittelman R., & Mannuzza, S. (1985). Diagnosing ADD-H in adolescents. *Pyschopharmacology Bulletin, 21*, 237-242.

Gittelman R., Mannuzza, S., Shenker, R., & Bonagura, N. (1985). Hyperactive boys almost grown up. I: Psychiatric status. *Archives of General Psychiatry, 42*, 937-947.

Gittelman Klein, R. (1975). Stimulant drug treatment of hyperkinesis. In D. Klein & R. Gittelman-Klein (Eds.), *Progress in psychiatric drug treatment (Vol. 1)*. New York: Brunner/Mazel.

Gittelman Klein, R., & Klein, D. (1976). Methylphenidate effects in learning disabilities: Psychometric changes. *Archives of General Psychiatry, 33*, 655-664.

Gittelman Klein, R., Klein, D.F., & Feingold, I. (1983). Children with reading disorders. II: Effects of methylphenidate in combination with reading remediation. *Journal of Child Psychology and Psychiatry, 24*, 193-212.

Glow, R.A. (1981). Cross validity and normative data on the Conners' parent and teacher rating scales. In K.D. Gadow & J, Loney (Eds.), *The psychosocial aspects of drug treatment for hyperactivity* (pp. 107-150). Boulder: Westview Press.

Goodman, L.S., & Gillman, A. (1970). *The pharmacological basis of therapeutics.* New York: Macmillan.

Gordon, M. (1979). The assessment of impulsivity and mediating behaviors in hyperactive and non-hyperactive children. *Journal of Abnormal Psychology, 7*, 317-327.

Gordon, M. (1986). *GDS user's manual.* New York: Clinical Diagnostics Inc.

Gordon, M., & McClure, F. (1984). *Assessment of attention deficit disorders using the Gordon Diagnostic System.* Paper presented at the meeting of the American Psychological Association, August, Toronto, Canada.

Gordon, M., & Mettelman, B.B. (1988). The assessment of attention: I. Standardization and reliability of a behavior-based measure. *Journal of Clinical Psychology, 44*, 682-690.

Gorenstein, E.E., & Newman, J.P. (1980). Disinhibitory psychopathology: A new perspective and a model for research. *Psychological Review, 87*, 301-315.

Goyette, C.H., Conners, C.K., & Ulrich, R.F. (1978). Normative data on revised Conners' parent and teacher rating scale. *Journal of Abnormal Child Psychology, 6*, 221-236.

Graham, P.J. (1977). (Ed.), *Epidemiological approaches in child psychiatry.* London: Academic Press.

Gray, J. (1976). The neuropsychology of anxiety. In I.G. Sarason & C.D. Spielberger (Eds.), *Stress and anxiety (Vol. 3)* (pp. 3-26). Washington, D.C.: Hemisphere.

Gray, J. (1981). A critique of Eysenck's theory of personality. In H.J. Eysenck (Ed.), *A model of personality* (pp. 246-276). New York: Springer.

Greenberg, L.M., & Lipman, R.S. (1971). Pharmacotherapy of hyperactive children: Current practices. *Clinical Proceedings of Children's Hospital, 27*, 101-105.

Greenspan, E. (1982). *Martin's Annual Criminal Code.* Canada Lawbook Ltd.

Gross, M.D., & Wilson, N.C. (1974). *Minimal brain dysfunction.* New York: Brunner/Mazel.

Grunewald-Zuberbier, E., Grunewald, G., & Rasche, A. (1975). Hyperactive behavior and EEG arousal reactions in children. *Electroencephalography and Clinical Neurophysiology, 38*, 149-159.

Gualtieri, C.T., Hicks, R., Mayo, J., & Schroeder, S. (1984). The persistance of stimulant effects in chronically treated children: Further evidence of an inverse relationship between drug effects and placebo levels of response. *Psychopharmacology, 83*, 44-47.

Gualtieri, C.T., Wargin, W., Kanoy, R., Patrick, K., Shen, D., Youngblood, W., Mueller, R.A., & Breese, G.R. (1982). Clinical studies of methylphenidate serum levels in children and adults. *Journal of the American Academy of Child Psychiatry, 21*, 19-26.

Halperin, J.M., Newcorn, J.H., Sharma, V., Healey, J.M., Wolf, L.E., Pascualvaca, D.M., & Schwartz, S. (1990). Inattentive and noninattentive ADHD children: Do they constitute a unitary group? *Journal of Abnormal Child Psychology, 18*, 437-449.

Halperin, J.M., O'Brien, J.D., Newcorn, J.H., Healey, J.M., Pascualvaca, D.M., Wolf, L.E., & Young, J.G. (1990). Validation of hyperactive, aggressive, and mixed hyperactive/aggressive childhood disorders. *Journal of Child Psychology and Psychiatry, 31*, 455-459.

Hamden-Allen, G., Stewart, M.A., & Beeghly, J.H. (1989). Subgrouping conduct disorder by psychiatric family history. *Journal of Child Psychology and Psychiatry, 30*, 889-897.

Hanley, J.A. (1987). Standard error of the kappa statistic. *Psychological Bulletin, 102*, 315-321.

Hartocollis, P. (1968). The syndrome of minimal brain dysfunction in young adult patients. *Bulletin Menninger Clinical, 32*, 102-114.

Hasher, L., & Zacks, R.T. (1979). Automatic and effortful processes in memory. *Journal of Experimental Psychology, 108*, 356-388.

Hays, W.L. (1988). *Statistics* (4th ed.). New York: Holt, Rinehart, & Winston.

Hechtman, L., & Weiss, G. (1986). Controlled prospective fifteen year follow-up of hyperactives as adults: Non-medical drug and alcohol use and anti-social behaviour. *Canadian Journal of Psychiatry, 31*, 557-567.

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

Hechtman, L., Weiss, G., & Perlman, T. (1981). Hyperactives as young adults: Past and current antisocial behavior (stealing, drug abuse) and moral development. *Psychopharmacology Bulletin, 17,* 107-110.

Hechtman, L., Weiss, G., Perlman, T. (1984). Hyperactives as young adults: Past and current substance abuse and antisocial behavior. *American Journal of Orthopsychiatry, 54,* 415-425.

Henker, B., & Whalen, C.K. (1989). Hyperactivity and attention deficits. *American Psychologist, 44*, 216-223.

Herbert, G.W. (1974). Teachers' ratings of classroom behavior: Factorials structure. *British Journal of Educational Psychology, 44,* 233-240.

Hicks, R.E., Gualtieri, T., Mayo, J.P., Schroeder, S.R., & Lipton, M.A. (1985). Methylphenidate and homeostasis: Drug effects on the cognitive performance of hyperactive children. In L.M. Bloomingdale (Ed.), *Attention deficit disorder: Identification, course and rationale (Vol. 2)* (pp. 131-141). New York : Spectrum.

Hinshaw, S.P. (1987). On the distinction between attentional deficits/hyperactivity and conduct problems/aggression in child psychopathology. *Psychological Bulletin, 101*, 443-463.

Hinshaw, S.P., Buhrmester, D., & Heller, T. (1989). Anger control in response to verbal provocation: Effects of stimulant medication for boys with ADHD. *Journal of Abnormal Child Psychology, 17,* 393-407.

Hinshaw, S.P., Henker, B., Whalen, C., Erhardt, D., & Dunnington, R.E. (1989). Aggressive, prosocial and nonsocial behavior in hyperactive boys: Dose effects of methylphenidate in naturalistic settings. *Journal of Consulting and Clinical Psychology, 57*, 636-643.

Holborow, P.L., & Berry, P. (1986). A multinational, cross-cultural perspective on hyperactivity. *American Journal of Orthopsychiatry*, 56, 320-322.

Holborow, P.L., Berry, P., & Elkins, J. (1984). Prevalence of hyperkinesis: A comparison of three rating scales. *Journal of Learning Disabilities, 17*, 411-417.

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

Huessy, H., & Gendron, R. (1973). Five hundred children followed from Grade 2 through Grade 5 for the prevalence of behavior disorder. *Acta Paedopsychiatrica, 39*, 302-309.

Huessy, H., Metoyer, M., & Townsend, M. (1974). 8-10 year follow-up of 84 children treated for behavioral disorders in rural Vermont. *Acta Paedopsychiatrica, 10*, 230-235.

Humphries, T., Swanson, J.M., Kinsbourne, M., & Yiu, L. (1979). Stimulant effects on persistence of motor performance of hyperactive children. *Journal of Pediatric Psychology, 4,* 55-56.

Juliano, D.B. (1974). Conceptual tempo, activity, and concept learning in hyperactive and normal children. *Journal of Abnormal Psychology, 83,* 629-634.

Jutai, J., Hare, R.D., & Donnolly, J.F. (1987). Psychopathy and event-related brain potentials (ERPs) associated with attention to speech stimuli. *Personality and Individual Differences, 8,* 175-184.

Kagan, J. (1965a). Individual differences in the resolution of response uncertainty. *Journal of Personality and Social Psychology, 2,* 154-160.

Kagan, J. (1965b). Reflection-impulsivity and reading ability in primary grade children. *Child Development, 36,* 609-628.

Kagan, J. (1966). The generality and dynamics of conceptual tempo. *Journal of Abnormal Psychology, 71,* 17-24.

Kagan, J., Moss, M.A., & Sigel, I.E. (1963). Psychological significance of style of conceptualization. In J.C. Wright & J. Kagan (Eds.), Basic cognitive processes in children. *Monographs of the Society for Research in Child Development, 28,* (Serial No. 86).

Kagan, J., Pearson, L., & Welch, L. (1966). Conceptual impulsivity and inductive reasoning. *Child Development, 37,* 583-594.

Kagan, J., Rosman, B.L., Day, P., Albert, J., & Phillips, W. (1964). Information processing in the child: Significance of analytic and reflective attitudes. *Psychological Monographs, 78,* (Whole No. 578).

Kahn, E., & Cohen, L.H. (1934). Organic drivenness - A brain stem syndrome and an experience - With case reports. *New England Journal of Medicine, 210,* 748-756.

Kashani, J.H., Daniel. A.E., Sulzberger, L.A., Rosenberg, T.K., & Reid, J.C. (1987). Conduct disordered adolescents from a community sample. *Canadian Journal of Psychiatry, 32,* 756-760.

Kaspar, J.C., Millichap, J.G., Backus, R., Child, D., & Schulman, J.L. (1971). A study of the relationship between neurological evidence of brain damage in children and distractibility. *Journal of Consulting and Clinical Psychology, 36,* 329-337.

Kavale, K. (1982). The efficacy of stimulant drug treatment for hyperactivity: A meta-analysis. *Journal of Learning Disabilities, 15,* 280-289.

Kazdin, A.E. (1980). *Research design in clinical psychology.* New York: Harper & Row.

Kazdin, A.E. (1987). Conduct disorders in childhood and adolescence. Beverly Hills: Sage.

Keppel, G. (1982). *Design and analysis: A researcher's handbook.* New Jersey: Prentice Hall.

Kinsbourne, M. (1985). Base-state dependency of stimulant effects on the cognitive performance of hyperactive children. In L.M. Bloomingdale (Ed.), *Attention deficit disorder: Identification, course and rationale (Vol. 2)* (pp. 143-154). New York: Spectrum.

Kinsbourne, M. (1989). Comments by mentor. The laboratory stimulant medication assessment in the ligh of models of attention deficit disorder. In L.M. Bloomingdale (Ed.), *Attention deficit disorder: Current concepts and emerging trends in attentional and behavioral disorders of childhood (Vol. 4)* (pp. 113-124). New York: Pergamon Press.

Kinsbourne, M., & Swanson, J.M. (1980). Evaluation of symptomatic treatment of hyperactive behavior by stimulant drugs. In R.M. Knights & D.J. Bakker (Eds.), *Treatment of hyperactive and learning disordered children* (pp. 207-218). Baltimore: University Park Press.

Klee, S.H., & Garfinkel, B.D. (1983). The computerized continuous performance task: A new measure of inattention. *Journal of Abnormal Child Psychology, 11*, 487-496.

Klein, A., & Young, R. (1979). Hyperactive boys in their classroom: Assessment of teacher and peer perceptions, interactions, and classroom behaviours. *Journal of Abnormal Child Psychology, 7*, 425-442.

Klein, D.F., Gittelman, R., Quitkin, F., & Rifkin, A. (1980). *Diagnosis and drug treatment of psychiatric disorders: Adults and children* (2nd ed.). New York: Williams & Wilkins.

Klerman, G.L. (1986). Scientific and ethical considerations in the use of placebo controls in clinical trials in psychopharmacology. *Psychopharmacology Bulletin, 22*, 25-29.

Klorman, R., Brumaghim, J.T., Salzman, L.F., Strauss, J., Borgstedt, A.D., McBride, M.C., & Loeb, S. (1988). Effects of methylphenidate on attention-deficit hyperactivity disorder with and without aggressive/noncompliant features. *Journal of Abnormal Psychology, 97*, 413-422.

Klorman, R., Coons, H.W., & Borgstedt, A.D. (1987). Effects of methylphenidate on adolescents with a childhood history of attention deficit disorder: I. Clinical findings. *Journal of the American Academy of Child and Adolescent Psychiatry, 26*, 363-367.

Klorman, R., Salzman, L.F., Borgstedt, A.D. (1988). Brain event-related potentials as a tool for evaluating cognitive deficits in attention deficit disorder and outcome of stimulant therapy. In L.M. Bloomingdale (Ed.), *Attention deficit disorder (Vol. 3)*. Oxford: Pergamon Press.

Klove, H., & Hole, K. (1979). The hyperkinetic syndrome: Criteria for diagnosis. In R.L. Trites (Ed.), *Hyperactivity in children: Etiology, measurement, and treatment implications* (pp. 121-136). Baltimore: University Park Press.

Knights, R.M. (1974). Psychometric assessment of stimulant-induced behavior change. In C.K. Conners (Ed.), *Clinical use of stimulant drugs in children* (pp. 221-231). Amsterdam: Excerpta Medica.

Kosson, D.S., Smith, S.S., & Newman, J.P. (1990). Evaluating the construct validity of psychopathy in Black and White male inmates: Three preliminary studies. *Journal of Abnormal Psychology, 99,* 250-259.

Kramer, J., & Loney, J. (1981). Childhood hyperactivity and substance abuse: A review of the literature. In K. Gadow & I. Bialer (Eds.), *Advances in learning and behavior disabilities (Vol. 1)* (pp. 225-229). Greenwich, C.T.: JAI Press.

Kupietz, S.S., & Richardson, E. (1978). Children's vigilance performance and inattentiveness in the classroom. *Journal of Child Psychology and Psychiatry, 19,* 155-160.

LaGreca, A.M., & Quay, H.C. (1984). Behavioral disorders of children. In N.S. Endler & J.M. Hunt (Eds.), *Personality and the behavior disorders (Vol. 2)* (pp. 711-746). New York: Wiley.

Lahey, B.B, Piacentini, J.C., McBurnett, K., Stone, P., Hartdagen, S., & Hynd, G. (1988). Psychopathology in the parents of children with conduct disorder and hyperactivity. *Journal of the American Academy of Child and Adolescent Psychiatry, 27,* 163-170.

Lambert,'N.M. (1988). Adolescent outcomes for hyperactive children. Perspectives on general and specific patterns of childhood risk for adolescent, educational, social, and mental health problems. *American Psychologist, 43,* 786-799.

Lambert, N.M., Hartsough, C.S., Sassone, D., & Sandoval, J. (1987). Persistence of hyperactivity symptoms from childhood to adolescence and associated outcomes. *American Journal of Orthopsychiatry, 57,* 22-32.

Lambert, N.M., Sandoval, J., & Sassone, D. (1978). Prevalence of hyperactivity in elementary school children as a function of social system definers. *American Journal of Orthopsychiatry, 48,* 446-463.

Langhorne, J., & Loney, J. (1979). A four-fold model for sub-grouping the hyperkinetic/MBD syndrome. *Child Psychiatry and Human Development, 9,* 153-159.

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

Laufer, M.W., & Denhoff (1957). Hyperkinetic behavior syndrome in children. *Journal of Pediatrics, 50,* 463-474.

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

Lerer, R.J., & Lerer, M.P. (1977). Responses of adolescents with minimal brain dysfunction to methylphenidate. *Journal of Learning Disabilities, 10,* 35-40.

Liberman, R. (1962). An analysis of the placebo phenomenon. *Journal of Chronic Diseases, 15,* 761-783.

Livingstone, R.L., Dykman, R.A., & Ackerman, P.T. (1990). The frequency and significance of additional self-reported psychiatric diagnoses in children with attention deficit disorder. *Journal of Abnormal Child Psychology, 18,* 465-478.

Loeber, R. (1990). Development and risk factors of juvenile antisocial behavior and delinquency. *Clinical Psychology Review, 10*, 1-41.

Loeber, R., & Schmaling, K.B. (1985). The utility of differentiating between mixed and pure forms of antisocial child behaviors. *Journal of Abnormal Child Psychology, 13*, 315-336.

Logan, G.D. (1985). Executive control of thought and action. *Acta Psychological, 60*, 193-210.

Loiselle, D., Stamm, J.S., Maitinsky, S., & Whipple, S.C. (1980). Evoked potential and behavioral signs of attentive dysfunctions in hyperactive boys. *Psychophysiology, 17*, 193-201.

Loney, J. (1980). Hyperkinesis comes of age: What do we know and where should we go? *American Journal of Orthopsychiatry, 50*, 28-42.

Loney, J. (1982). *Research diagnostic criteria for attention deficit disorder.* Paper presented at the meeting of the American Psychopathological Association, February, New York.

Loney, J., Kramer, J., & Milich, R. (1981). The hyperkinetic child grows up: Predictors of symptoms, delinquency, and achievement at follow-up. In K.D. Gadow & J. Loney (Eds.), *Psychosocial aspects of drug treatment for hyperactivity.* Boulder: Westview Press.

Loney, J., Langhorne, J, & Paternite, C. (1978). An empirical basis for subgrouping the hyperkinetic/minimal brain dysfunction syndrome. *Journal of Abnormal Psychology, 87*, 431-441.

Loney, J., & Milich, R. (1982). Hyperactivity, inattention, and aggression in clinical practice. In M. Wolraich & D. Routh, (Eds.), *Advances in developmental and behavioral pediatrics (Vol. 3)* (pp. 113-147). Greenwich, C.T.: JAI Press.

Loney, J., Whaley Klahn, M.A., Kosier, T., & Conboy, J. (1981). *Hyperactive boys and their brothers at 21: Predictors of aggressive and antisocial outcome.* Presented at the meeting of the Life History Research, November, Monterey, California.

Lovejoy, M.C., & Rasmussen, N.H. (1990). The validity of the vigilance tasks in differential diagnosis of children referred for attention and learning problems. *Journal of Abnormal Child Psychology, 18*, 671-681.

Lykken, D.T. (1957). A study of anxiety in the sociopathic personality. *Journal of Abnormal and Social Psychology, 55*, 6-10.

Lysak, H. M. (1989). *The effects of Ritalin (methylphenidate) on self-regulatory processes of hyperactive young offenders.* Unpublished doctoral dissertation, Simon Fraser University, Burnaby, B.C.

Lytton, G., & Knobel, E. (1958). Diagnosis and treatment of behaviour disorders in children. *Diseases of the Nervous System, 20*, 5-11.

Magnusson, D., Slottin,H., & Duner, A. (1983). Aggression and criminality in a longitudinal perspective. In K.T. Van Dusen & S.A. Mednick (Eds.), *Antecedents of aggression and antisocial behavior* (pp. 1-54). Boston: Kluwer-Nijhoff.

Maletzky, B. (1974). Amphetamine and delinquency: Hyperkinesis persisting? *Diseases of the Nervous System, 35*, 543-547.

MacKay, M.C., Beck, L., & Taylor, R. (1973). Methylphenidate for adolescents with minimal brain dysfunction. *New York State Journal of Medicine, 73*, 550-554.

Mann, L. (1973). Differences between reflective and impulsive children in tempo and quality of decision-making. *Child Development, 44*, 274-279.

Mannheimer, D.I., & Mellinger, G.D. (1967). Personality characteristics of the child accident repeater. *Child Development, 38*, 491-513.

Mannuzza, S., Gittelman Klein, R., Horowitz Konig, P., & Giampino, T. (1989). Hyperactive boys almost grown up. *Archives of General Psychiatry, 46*, 1073-1079.

Mannuzza, S., Gittelman Klein, R., Bonagura, N., Horowitz Konig, P., & Shenker, R. (1988). Hyperactive boys almost grown up. II. Status of subjects without a mental disorder. *Archives of General Psychiatry, 45*, 13-18.

Mash, E.J., & Dalby, T. (1979). Behavioral interventions for hyperactivity. In R. Trites (Ed.), *Hyperactivity in children: Etiology, measurement, and treatment implications* (pp. 161-216). Baltimore: University Park Press.

McClure, F., & Gordon, M. (1984). Performance of disturbed hyperactive and nonhyperactive children on an objective measure of hyperactivity. *Journal of Abnormal Child Psychology, 12*, 561-572.

McGee, R., Williams, S., Bradshaw, J., Chapel, J.L., Robins. A., & Silva, P.A. (1985). The Rutter scale for completion by teachers: Factor structure and relationships with cognitive abilities and family adversity for a sample of New Zealand children. *Journal of Child Psychology and Psychiatry, 26*, 727-739.

McGee, R., Williams, S., & Silva, P.A. (1984). Behavioral and developmental characteristics of aggressive, hyperactive and aggressive-hyperactive boys. *Journal of the American Academy of Child Psychology and Psychiatry, 23*, 270-279.

McMahon, R.C. (1984). Hyperactivity as dysfunction of activity, arousal, or attention: A study of research relating to DSM-III's attention deficit disorder. *Journal of Clinical Psychology, 40*, 1300-1308.

Mendelson, W., Johnson, N., & Stewart, M.A. (1971). Hyperactive children as teenagers: A follow-up study. *Journal of Nervous and Mental Disease, 153*, 273-279.

Menkes, M.M., Rowe, J.S., & Menkes, J.H. (1967). A twenty-five year follow-up study on the hyperkinetic child with minimal brain dysfunction. *Pediatrics, 39*, 393-399.

Messer, S.B. (1970). The effect of anxiety over intellectual performance on reflection-impulsivity in children. *Child Development, 41*, 723-735.

Messer, S.B. (1976). Reflection-impulsivity: A review. *Psychological Bulletin, 83,* 1026-1052.

Michael, R.L., Klorman, R., Salzman, L.F., Borgstedt, A.D., & Dainer, K.B. (1981). Normalizing effects of methylphenidate on hyperactive children's vigilance and evoked potentials. *Psychophysiology, 18,* 665-671.

Milich, R., Licht, B.G. Murphy, D.A., & Pelham, W.E. (1989). Attention-deficit hyperactivity disordered boys' evaluations of and attributions for task performance on medication versus placebo. *Journal of Abnormal Psychology, 98,* 280-284.

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

Milich, R., Loney, J., & Landau, S. (1982). Independent dimensions of hyperactivity and aggression: A validation with playroom observation data. *Journal of Abnormal Psychology, 91,* 183-198.

Milich, R.G., Roberts, M.A., Loney, J., & Caputo, J. (1980). Differentiating practice effects and statistical regression on the Conners Hyperactivity Index. *Journal of Abnormal Child Psychology, 8,* 549-552.

Miller, R.G., Palkes, M.S., & Stewart, M.A. (1973). Hyperactive children in suburban elementary schools. *Child Psychiatry and Human Development, 4,* 121-127.

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

Minde, K.K. (1977). Hyperactivity: Where do we stand? In M. Blau, I. Rapin & M. Kinsbourne (Eds.), *Topics in child neurology.* New York: Spectrum.

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

Minde, K., Weiss, G., & Mendelson, B.A. (1972). A five-year follow-up study on the hyperkinetic child with minimal brain dysfunction. *Journal of the American Academy of Child Psychiatry, 11,* 595-616

Mischel, W. (1958). Preference for delayed reinforcement: An experimental study of a cultural observation. *Journal of Abnormal and Social Psychology, 66,* 57-61.

Moffit, T.E. (1990). Juvenile delinquency and attention deficit disorder: Boys' developmental trajectories from age 3 to age 15. *Child Development, 61,* 893-910.

Morgan, D.I. (1979). Prevalence and types of handicapping conditions found in juvenile correctional institutions: A national study. *Journal of Special Education, 13,* 283-294.

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

Morrison, J.R. (1980). Childhood hyperactivity in an adult psychiatric population. *Journal of Clinical Psychiatry, 41*, 40-43.

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

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

Munir, K., Biederman, J., & Knee, D. (1987). Psychiatric comorbidity in patients with attention deficit disorders: A controlled study. *Journal of the American Academy of Child and Adolescent Psychiatry, 26*, 844-848.

Neuchterlein, K.H., Parasuraman, R., & Jiang, Q. (1983). Visual sustained attention: Image degradation produces rapid sensitivity decrement over time. *Science, 220*, 327-329.

Newman, J.P. (1987). Reaction to punishment in extroverts and psychopaths: Implications for the impulsive behavior of disinhibited individuals. *Journal of Research in Personality, 21*, 464-480.

Newman, J.P., & Kossan, D.S. (1986). Passive avoidance learning in psychopathic and nonpsychopathic offenders. *Journal of Abnormal Psychology, 95*, 252-256.

Newman, J.P., Patterson, C.M., & Kosson, D.S. (1987). Response perseveration in psychopaths. *Journal of Abnormal Psychology, 96*, 145-148.

Newman, J.P., Widom, C.S., & Nathan, S. (1985). Passive avoidance in syndromes of disinhibition: Psychopathy and extraversion. *Journal of Personality and Social Psychology, 48*, 1316-1327.

Nichols, P.L., & Chen, T.C. (1980). *Minimal brain dysfunction: A prospective study.* Hillsdale, N.J.: Erlbaum.

O'Dougherty, M., Neuchterlein, K.H., & Drew, B. (1984). Hyperactive and hypoxic children: Signal detection, sustained attention and behavior. *Journal of Abnormal Psychology, 93*, 178-191.

Oettinger, L. (1973). General discussion. *Annals of the New York Academy of Science, 205*, 345-348.

Offord, D.R., Sullivan, K., Allen, N., & Abrams, N. (1979). Delinquency and hyperactivity. *Journal of Nervous and Mental Disease, 167*, 734-741.

Orris, J.B. (1969). Visual monitoring performance in three subgroups of male delinquents. *Journal of Abnormal Psychology, 74*, 227-229.

Ottenbacher, K.J., & Cooper, H.M. (1983). Drug treatment of hyperactivity in children. *Developmental Medicine and Child Neurology, 25*, 358-366.

Parasuraman, R., & Davies, D.R. (1977). A taxonomic analysis of vigilance performance. In R.R. Mackie (Ed.), *Vigilance: Theory, operational performance and physiological correlates* (pp. 559-574). New York: Plenum.

Parry, P.A., & Douglas, V.I. (1983). Effects of reinforcement on concept identification in hyperactive children. *Journal of Abnormal Child Psychology, 11*, 327-340.

Paternite, R., & Lyon, R. (1982). Clinical and empirical identification of learning disabled juvenile delinquents. *Journal of Correctional Education, 33*, 7-13.

Paternite, C,E., & Loney, J. (1980). Childhood hyperkinesis: Relationships between symptomatology and home environment. In C.K. Whalen & B. Henker (Eds.), *Hyperactive children: The social ecology of identification and treatment* (pp. 105-141). New York: Academic Press.

Paternite, C.E., Loney, J., & Langhorne, J. (1976). Relationships between symptomatology and SES-related factors in hyperkinetic/MBD boys. *American Journal of Orthopsychiatry, 46*, 293-318.

Pelham, W.E., Bender, M.E., Caddell, J., Booth, S., & Moorer, S.H. (1985). Methylphenidate and children with attention deficit disorder. Dose effects on classroom, academic and social behavior. *Archives of General Psychiatry, 42*, 948-952.

Pelham, W.E., McBurnett, K., Harper, G.W., Milich, R., Murphy, D.A., Clinton, J., & Thiele, C. (1990). Methylphenidate and baseball playing in ADHD chilren: Who's on first? *Journal of Consulting and Clinical Psychology, 58*, 130-133.

Pelham, W.E., Milich,R., & Walker, J.L. (1986). Effects of continuous and partial reinforcement and methylphenidate on learning in children with attention deficit disorder. *Journal of Abnormal Psychology, 95*, 319-325.

Pelham, W.E., & Murphy, H.A. (1986). Attention deficit and conduct disorders. In M. Hersen (Ed.), *Pharmacological and behavioral treatment: An integrative approach* (pp. 109-148). New York: Wiley.

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

Prendergast, M., Taylor, E., Rapoport, J.L., Bartko, J., Donnelly, M., Zametkin, A., Ahearn, M.B., Dunn, G., & Wieselberg, H.M. (1988). The diagnosis of childhood hyperactivity. A U.S.-U.K. cross-national study of DSM-III and ICD-9. *Journal of Child Psychology and Psychiatry, 29*, 289-300.

Prinz, R.J., Connor, P.A., & Wilson, C. (1981). Hyperactive and aggressive behaviors in childhood: Intertwined dimensions. *Journal of Abnormal Child Psychology, 9*, 191-202.

Prinz, R.J., & Loney, J. (1974). Teacher-rated hyperactive and elementary school girls. An exploratory developmental study. *Child Psychiatry and Human Development, 4*, 246-257.

Prior, M., & Sanson, A. (1986). Attention deficit disorder with hyperactivity. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 27*, 307-319.

Prior, M., Sanson, A, Freethy, C., & Geffen, G. (1985). Auditory attentional abilities in hyperactive children. *Journal of Child Psychology and Psychiatry, 26*, 289-304.

Quay, H.C. (1979). Classification. In H.C. Quay & J.S. Werry (Eds.), *Psychopathological disorders of childhood* (2nd ed.) (pp. 1-42). New York: Wiley.

Quay, H.C. (1985). Aggression, conduct disorder, and attention problems. In L.M. Bloomingdale (Ed.), *Attention deficit disorder: Identification, course and rationale (Vol. 2)* (pp. 33-48). New York: Spectrum.

Quay, H.C. (1986). Conduct disorders. In H.C. Quay & J.S. Werry (Eds.), *Psychopathological disorders of childhood* (pp. 35-72). New York: Wiley & Sons.

Quay, H.C. (1988). The behavioral reward and inhibition system in childhood behavior disorders. In L.M. Bloomingdale (Ed.), *Attention deficit disorder (Vol. 3)* (pp. 176-186). Oxford: Pergamon Press.

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

Radosh, A., & Gittelman, R. (1981). The effect of appealing distractors on the performance of hyperactive children. *Journal of Abnormal Child Psychology, 9*, 179-189.

Raine, A., & Jones, F. (1987). Attention, autonomic arousal and personality in behaviorally disordered children. *Journal of Abnormal Child Psychology, 15*, 583-600.

Raine, A., & Venables, P.H. (1987). Contingent negative variation. P3 evoked potentials and antisocial behavior. *Psychophysiology, 24*, 191-199.

Rapoport, J.L., Buchsbaum, M.S., Weingartner, H., Zahn, T.P., Ludlow, C., & Mikkelsen, E.J. (1980). Dextroamphetamine: Its cognitive and behavioral effects in normal and hyperactive boys and normal men. *Archives of General Psychiatry, 37*, 933-942.

Rapoport, J.L., Buchsbaum, M.S., Zahn, T.P., Weingartner, H., Ludlow, C., & Mikkelsen, E.J. (1978). Dextroamphetamine: Cognitive and behavioral effects in normal and prepubertal boys. *Science, 199*, 560-563.

Rapoport, J.L., & Zametkin, A. (1980). Attention deficit disorder. *Psychiatric Clinics of North America, 3*, 425-441.

Rapport, M.D., DuPaul, G.J., & Smith, N.F. (1985). Rate-dependency and hyperactivity: Methylphenidate effects on operant responding. *Pharmacology, Biochemistry and Behavior, 23*, 77-83.

Rapport, M.D., DuPaul, G.J., Stoner, G., Birmingham, B., & Masse, G. (1985). Attention deficit disorder with hyperactivity: Differential effects of methylphenidate on impulsivity. *Pediatrics, 76*, 938-943.

Rapport, M.D., DuPaul, G.J., Stoner, G., & Jones, J.T. (1986). Comparing classroom and clinic measures of attention deficit disorder: Differential, idiosyncratic and dose-response effects of methylphenidate. *Journal of Consulting and Clinical Psychology, 54*, 334-341.

Rapport, M.D., Jones, J.T., DuPaul, G.J., Kelly, K.L., Gardner, M.J., Tucker, S.B., & Shea, M.S. (1987). Attention deficit disorder and methylphenidate: Group and single-subject analyses of dose effects on attention in clinic and classroom settings. *Journal of Clinical Child Psychology, 16*, 329-338.

Rapport, M.D., Stoner, G., DuPaul, G.J., Birmingham, B.K., & Tucker, S.B. (1985). Methylphenidate in hyperactive children: Differential effects of dose on academic, learning, and social behavior. *Journal of Abnormal Child Psychology, 13*, 227-243.

Rapport, M.D., Stoner, G., DuPaul, G.J., Kelly, K.L., Tucker, S.B., & Schoeler, T. (1988). Attention deficit disorder and methylphenidate: A multilevel analysis of dose-response effects on children's impulsivity across settings. *Journal of the American Academy of Child and Adolescent Psychiatry, 27*, 60-69.

Reeves, J.C., Werry, J.S., Elkind, G.S., & Zametkin, A. (1987). Attention deficit, conduct, oppositional, and anxiety disorders in children: II. Clinical characteristics. *Journal of the American Academy of Child and Adolescent Psychiatry, 26*, 144-155.

Renshaw, D. (1974). *The hyperactive child.* Chicago: Nelson-Hall.

Reznick, E.D., & Freeman, R.J. (1985). *Attention deficit disorder and the young offender: Relationships between symptomology and corrections history.* Paper presented at the meeting of the Canadian Psychological Association, June, Halifax, Nova Scotia.

Riddle, K.D., & Rapoport, J.L. (1976). A 2-year follow-up of 72 hyperactive boys. *Journal of Nervous and Mental Disease, 162*, 126-134.

Robbins, T.W., & Sahakian, B.J. (1979). "Paradoxical" effects of psychomotor stimulant drugs in hyperactive children from the standpoint of behavioral pharmacology. *Neuropharmacology, 18*, 931-950.

Robins, L.N. (1979). Follow-up studies. In H.C. Quay & J.S. Werry (Eds.), *Psychopathological disorders of childhood* (2nd Ed.). New York: Wiley & Sons.

Rosen, L.A., O'Leary, S.G., & Conway, G. (1985). Case studies and clinical replication series. The withdrawal of stimulant medication for hyperactivity: Overcoming detrimental attributions. *Behavior Therapy, 16*, 538-544.

Rosenthal, R.H., & Allen, T.W. (1978). An examination of attention, arousal, and learning dysfunctions of hyperkinetic children. *Psychological Bulletin, 85*, 689-715.

Rosenthal, R.H., & Allen, T.W. (1980). Intratask distractibility in hyperkinetic and nonhyperkinetic children. *Journal of Abnormal Child Psychology, 8*, 175-187.

Ross, A.O. (1976). *Psychological aspects of learning disabilities and reading disorders.* New York: McGraw-Hill.

Ross, D.M., & Ross, S.A. (1976). *Hyperactivity: Research, theory, action.* New York: Wiley & Sons.

Ross, D.M., & Ross, S.A. (1982). *Hyperactivity. Current issues, research and theory.* New York: Wiley & Sons.

Ross, S., & Buckalew, L.W. (1979). On the agentry of placebos. *American Psychologist, 34*, 277-278.

Ross, S., & Buckalew, L.W. (1983). The placebo as an agent in behavioral manipulation: A review of problems, issues, and affected measures. *Clinical Psychology Review, 3*, 457-471.

Ross, S, & Buckalew, L.W. (1985). Placebo agentry: Assessment of drug and placebo effects. In L. White, B. Tursky, & G.E. Schwartz (Eds.), *Placebo: Theory, research and mechanisms* (pp. 67-82). New York: Guilford.

Rosvold, H.E., Mirsky, A.F., Sarason, I., Bransome, E.D., & Beck, L.H. (1956). A continuous-performance test of brain damage. *Journal of Consulting and Clinical Psychology, 20*, 343-352.

Routh, D.K. (1978). Hyperactivity. In P. Magrab (Ed.), *Psychological management of pediatric problems (Vol. 2).* Baltimore: University Park Press.

Routh, D.K., & Schroeder, C.S. (1976). Standardized playroom measures as indices of hyperactivity in children. *Developmental Psychology, 10*, 163-168.

Rubin, R., & Balow, B. (1971). Learning and behavior disorders: A longitudinal study. *Exceptional Child, 38*, 293-299.

Rutter, M. (1982). Syndromes attributed to "minimal brain dysfunction" in childhood. *American Journal of Psychiatry, 139*, 21-33.

Rutter, M. (1983). Behavioral studies: Questions and findings on the concept of a distinctive syndrome. In M. Rutter (Ed.), *Developmental neuropsychiatry* (pp. 259-279). New York: Guilford Press.

Rutter, M., & Garmezy, N. (1983). Developmental psychopathology. In P.H. Musson (Ed.), *Handbook of child psychology* (pp. 776-871). New York: Wiley.

Rutter, M., & Giller, H. (1983). *Juvenile delinquency. Trends and perspectives.* New York: Penguin Books.

Rutter, M., Shaffer, D., & Shepherd, M. (1975). *A multiaxial classification of child psychiatric disorders.* Geneva: World Health Organization.

Rutter, M., Tizard, J., & Whitmore, K. (1970). *Education, health and behaviour.* London: Longmans.

Rutter, M., & Yule, W. (1975). The concept of specific reading retardation. *Journal of Child Psychology and Psychiatry, 16*, 181-197.

Safer, D.J., & Allen, R.P. (1975). Stimulant drug treatment of hyperactive adolescents. *Diseases of the Nervous System, 36*, 454-457.

Safer, D.J., & Allen, R.P. (1976). *Hyperactive children: Diagnosis and management.* Baltimore: University Park Press.

Safer, D.J., & Krager, J.M. (1984). Trends in medication therapy for hyperactivity: National and international perspectives. *Advances in Learning and Behavioral Disabilities, 3*, 125-149.

Safer, D.J., & Krager, J.M. (1985). Prevalence of medication treatment for hyperactive adolescents. *Psychopharmacology Bulletin, 21*, 212-215.

Samuels, S.J., & Edwall, G. (1981). The role of attention in reading with implications for the learning disabled student. *Journal of Learning Disabilities, 14*, 353-361.

Sandberg, S.T., Rutter, M., & Taylor, E. (1978). Hyperkinetic disorder in psychiatric clinic attenders. *Developmental Medicine in Child Neurology, 20*, 279-299.

Sandberg, S.T., Wieselberg, M., & Shaffer, D. (1980). Hyperkinetic and conduct problem children in a primary school population: Some epidemiological findings. *Journal of Child Psychology and Psychiatry, 21*, 293-311.

Sandoval, J. (1977). The measurement of hyperactive syndrome in children. *Review of Educational Research, 47*, 293-318.

Sandoval, J., Lambert, N.M., & Sassone, D. (1980). The identification and labeling of hyperactivity in children: An interactive model. In C.K. Whalen & B. Henker (Eds.), *Hyperactive children: The social ecology of identification and treatment* (pp. 145-171). New York: Academic Press.

Sandoval, J., Lambert, N.M., & Yandell, W. (1976). Current medical practice and hyperactive children. *American Journal of Orthopsychiatry, 46*, 323-334.

Satterfield, J.H. (1978). The hyperactive child syndrome: A precursor of adult psychopathy? In R. Hare and P. Schalling (Eds.), *Psychopathic behavior: Approaches to research.* New York: Wiley & Sons.

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

Satterfield, J.H., Cantwell, D.P., & Satterfield, B.T. (1979). Multimodality treatment. *Archives of General Psychiatry, 36*, 965-974.

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

Satterfield, J.H., Hoppe, C.M., & Schell, A.M. (1982). A prospective study of delinquency in 110 adolescent boys with attention deficit disorder and 88 normal adolescent boys. *American Journal of Psychiatry, 139*, 795-798.

Schachar, R. (1989). *Guidelines for the assessment and management of childhood hyperactivity for mental health professionals.* Paper presented at the Attention Deficit-Hyperactivity Disorders in Children and Adolescents conference, November, Oakville, Ontario.

Schachar, R., & Logan, G. (1990). Are hyperactive children deficient in attentional capacity? *Journal of Abnormal Child Psychology, 18*, 493-513.

Schachar, R., Logan, G., Wachsmuth, R., & Chajczyk, D. (1988). Attaining and maintaining preparation: A comparison of attention in hyperactive, normal and disturbed control children. *Journal of Abnormal Child Psychology, 16*, 361-378.

Schachar, R., Rutter, M., & Smith, A. (1981). The characteristics of situationally and pervasively hyperactive children: Implications for syndrome definition. *Journal of Child Psychology and Psychiatry, 22*, 375-392.

Schacter, S., & Latane, B. (1964). Crime, cognition and the autonomic nervous system. In M. Jones (Ed.), *Nebraska Symposium on Motivation.* Lincoln: University of Nebraska Press.

Schleifer, M., Weiss, G., Cohen, N.J., Elman, M., Cvejic, H., & Kruger, E. (1975). Hyperactivity in preschoolers and the effect of methylphenidate. *American Journal of Orthopsychiatry, 45*, 35-50.

Schmauk, F. (1970). Punishment, arousal and avoidance learning in sociopaths. *Journal of Abnormal Psychology, 76*, 325-335.

Schwartz, M., Friedman, R., Lindsay, P., & Narrol, H. (1982). The relationship between conceptual tempo and depression in children. *Journal of Consulting and Clinical Psychology, 50*, 488-490.

Schwarz, J.C. (1985). Childhood psychopathology. In S.I. Pfeiffer (Ed.), *Clinical child psychology: An introduction to theory, research, and practice* (pp. 93-126). Orlando, F.L.: Grune & Stratton.

Sebrechts, M., Shaywitz, S., Shaywitz, B., Jatlow, P., Anderson, G., & Cohen, D. (1986). Components of attention, methylphenidate dosage, and blood levels in children with attention deficit disorder. *Pediatrics, 77*, 222-228.

Seidel, W.T., & Joschko, M. (1990). Evidence of difficulties in sustained attention in children with ADDH. *Journal of Abnormal Child Psychology, 18*, 217-229.

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

Shaffer, D., McNamara, N., & Pincus, T.H. (1974). Psychiatric outcome of localized head injury in children. In R. Porter & D.W. Simmons (Eds.), *Outcome of severe damage to the central nervous system.* Amsterdam: Excerpta Medica.

Shapiro, D. (1965). *Neurotic styles.* New York: Basic Books.

Shapiro, S.K., & Garfinkel, B.D. (1986). The occurrence of behavior disorders in children: The interdependence of attention deficit disorder and conduct disorder. *Journal of the American Academy of Child and Adolescent Psychiatry, 25*, 809-819.

Shapiro, S.K., & Morris, L.A. (1978). The placebo effect in medical and psychological therapies. In S.L. Garfield & A.E. Bergin (Eds.), *Handbook of psychotherapy and behavioral change: An empirical analysis* (2nd ed.) (pp. 369-400). New York: Wiley & Sons.

Shapiro, S.K., Quay, H.C., Hogan, A.E., & Schwartz, K.P. (1988). Response perseveration and delayed responding in undersocialized aggressive conduct disorder. *Journal of Abnormal Psychology, 97*, 371-373.

Shaywitz, S.E., & Shaywitz, B.E. (1984). Diagnosis and management of attention deficit disorder: A pediatric perspective. *Pediatric Clinics of North America, 31*, 429-457.

Shekim, W.O., Kashani, J., Beck, N., Cantwell, D., Martin. J., Rosenberg, J., & Costello, A. (1985). The prevalence of attention deficit disorders in a rural midwestern community sample of nine-year-old children. *Journal of the American Academy of Child and Adolescent Psychiatry, 24*, 765-770.

Shelley, E.M., & Riester, A. (1972). Syndrome of minimal brain damage in young adults. *Diseases of the Nervous System, 33*, 335-338.

Shiffrin, R.M., & Schneider, W. (1977). Controlled and automatic human information processing - II. Perceptual and learning, automatic attending, and a general theory. *Psychological Review, 84*, 127-190.

Shue, K., & Douglas, V.I. (1983). *The effect of Ritalin on hyperactive children's performance on a DRL measure of impulsivity.* Unpublished manuscript.

Silver, L.B. (1981). The relationship between learning disabilities, hyperactivity, distractibility, and behavioral problems. *This Journal, 20*, 385-390.

Sleator, E.K., von Neumann, A.W., & Sprague, R.L. (1974). Hyperactive children: A continuous long-term placebo-controlled follow-up. *Journal of the American Medical Association, 229*, 316-317.

Solanto, M.V. (1984). Neuropharmacological basis of stimulant drug action in attention deficit disorder with hyperactivity: A review and synthesis. *Psychological Bulletin, 95*, 387-409.

Solanto, M.V. (1986). Behavioral effects of low-dose methylphenidate in childhood attention deficit disorder: Implications for a mechanism of stimulant drug action. *Journal of the American Academy of Child Psychiatry, 25*, 96-101.

Solanto, M.V. (1990). The effects of reinforcement and response-cost on a delayed response task in children with attention-deficit hyperactivity disorder: A research note. *Journal of Child Psychology and Psychiatry, 31*, 803-808.

Sorosky, A.D. (1979). Psychopharmacology and the adolescent: An introduction. In S.C. Feinstein & P.L. Giovacchini (Eds.), *Adolescent psychiatry (Vol. 2).* Chicago: University of Chicago Press.

Sostek, A.J., Buchsbaum, M.S., & Rapoport, J.L. (1980). Effects of amphetamine on vigilance performance in normal and hyperactive children. *Journal of Abnormal Child Psychology, 8*, 491-500.

Sprague, R.L. (1978). Principles of clinical trials and social, ethical, and legal issues of drug use in children. In J.S. Werry (Ed.), *Pediatric psychopharmacology: The use of behavior modifying drugs in children* (pp. 109-135). New York: Brunner/Mazel.

Sprague, R.L., Barnes, K.R., & Werry, J.S. (1970). Methylphenidate and thioridazine: Learning, reaction-time, activity, and classroom behavior in disturbed children. *American Journal of Orthopsychiatry, 40*, 615-628.

Sprague, R.L., & Gadow, K.D. (1976). The role of the teacher in drug treatment. *School Review, 85*, 109-140.

Sprague, R.L., & Sleator, E.K. (1973). Effects of psychopharmacologic agents on learning disorders. *Pediatric Clinics of North America, 20*, 719-735.

Sprague, R.L., & Sleator, E.K. (1975). What is the proper dose of stimulant drugs in children? *International Journal of Mental Health, 4*, 75-104.

Sprague, R.L., & Sleator, E.K. (1977). Methylphenidate in hyperkinetic children: Differences in dose effects on learning and social behavior. *Science, 198*, 1274-1276.

Spring, C., Greenberg, L., Scott, J., & Hopwood, J. (1973). Reaction time and the effect of Ritalin on children with learning problems. *Perceptual and Motor Skills, 36*, 75-82.

Stewart, M.A., DeBlois, C.S., & Cummings, C. (1980). Psychiatric disorder in the parents of hyperactive boys with conduct disorder. *Journal of Child Psychology and Psychiatry, 21*, 283-292.

Stewart, M.A., Cummings, C., Singer, S., & DeBlois, C.S. (1981). The overlap between hyperactive and unsocialized aggressive children. *Journal of Child Psychology and Psychiatry, 22*, 35-45.

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

Stewart, M.A., Pitts, F., Craig, A., & Deiruf, W. (1966). The hyperactive child syndrome. *American Journal of Orthopsychiatry, 36*, 861-867.

Stewart, M.A., Thach, B., & Freidin, M. (1970). Accidental poisoning and the hyperactive child syndrome. *Diseases of the Nervous System, 31*, 403-407.

Strauss, A.A., & Lehtinen, V. (1947). *Psychopathology and education of the brain-injured child (Vol. 1).* New York: Grune & Stratton.

Stroufe, L. (1975). Drug treatment of children with behavior problems. In F. Harowitz (Ed)., *Review of child developmental research (Vol. 4)* (pp. 347-407). Chicago: University Press.

Sulzbacher, S.I. (1976). Psychotropic medication with children: An evaluation of procedural bias in results of reported studies. *Pediatrics, 51*, 513-517.

Swanson, J.M. (1985). Measures of cognitive functioning appropriate for use in pediatric psychopharmacology research studies. *Psychopharmacology Bulletin, 21*, 887-890.

Swanson, J.M. (1989). Paired-associate learning in the assessment of ADD-H children. In L.M. Bloomingdale & J.M. Swanson (Eds.), *Attention deficit disorder: Current concepts and emerging trends in attentional and behavioral disorders of childhood (Vol. 4)* (pp. 87-112). New York: Pergamon Press.

Swanson, J.M., & Kinsbourne, M. (1978). Should you use stimulants to treat the hyperactive child? *Modern Medicine, 46*, 71-80.

Swanson, J.M., & Kinsbourne, M. (1979). The cognitive effects of stimulant drugs on hyperactive children. In G.A. Hale & M. Lewis (Eds.), *Attention and cognitive development.* New York: Plenum Press.

Swanson, J.M., Kinsbourne, M., Roberts, W., & Zucker, K. (1978). Time-response analysis of the effect of stimulant medication on the learning ability of children referred for hyperactivity. *Pediatrics, 61*, 21-29.

Swanson, J.M., Sandman, C.A., Deutsch, C., & Baren, M. (1983). Methylphenidate (Ritalin) given with or before breakfast: Part I. Behavioral, cognitive and electrophysiological effects. *Pediatrics, 72*, 49-55.

Sykes, D.H., Douglas, V.I., & Morgenstern, G. (1972). The effect of methylphenidate (Ritalin) on sustained attention in hyperactive children. *Psychopharmacologia, 25*, 262-274.

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

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

Szatmari, P., Offord, D.R., & Boyle, M.H. (1989a). Ontario Child Health study: Prevalence of attention deficit disorder with hyperactivity. *Journal of Child Psychology and Psychiatry and Allied Disciplines, 30*, 219-230.

Szatmari, P., Boyle, M.H., & Offord, D.R. (1989b). ADDH and conduct disorder: Degree of diagnostic overlap and differences among correlates. *Journal of the American Academy of Child and Adolescent Psychiatry, 28*, 865-872.

Tannock, R., Schachar, R.J., Carr, R.P., Chajczyk, D., & Logan, G.D. (1989). Effects of methylphenidate on inhibitory control in hyperactive children. *Journal of Abnormal Child Psychology, 17*, 473-491.

Tant, J.L., & Douglas, V.I. (1982). Problem-solving in hyperactive, normal, and reading-disabled boys. *Journal of Abnormal Child Psychology, 10*, 285-306.

Taylor, E. (1983). Drug response and diagnostic validation. In M. Rutter (Ed.), *Developmental neuropsychiatry* (pp. 348-368). New York: Guilford Press.

Taylor, E., Everitt, B., Thorley, G., Schachar, R., Rutter, M., & Wieselberg, M. (1986). A cluster analytic approach to the identification of a behavioral syndrome. *British Journal of Psychiatry, 149*, 768-777.

Taylor, E., & Sandberg, S. (1984). Hyperactive behaviour in English school children: A questionnaire survey. *Journal of Abnormal Child Psychology, 12*, 143-156.

Taylor, E., Schachar, R., Thorley, G., Wieselberg, H.M., Everitt, B., & Rutter, M. (1987). Which boys respond to stimulant medication? A controlled trial of methylphenidate in boys with disruptive behavior. *Psychological Medicine, 17*, 121-143.

Taylor, S. (1988). Some comments on Prior and Sanson's "attention deficit disorder with hyperactivity: A critique". *Journal of Child Psychology and Psychiatry and Allied Disciplines, 29*, 217-221.

Thorley, G. (1983). Data on the Conners teacher rating scale from a British clinic population. *Journal of Behavioral Assessment, 5*, 1-10.

Thorley, G. (1984). Review of follow-up and follow-back studies of childhood hyperactivity. *Psychological Bulletin, 96*, 116-132.

Thurston, C.M., Sobol, .P., Swanson, J.M., & Kinsbourne, M. (1979). Effects of methylphenidate (Ritalin) on selective attention in hyperactive children. *Journal of Abnormal Psychology, 7*, 471-481.

Trites, R.L. (1979). Prevalence of hyperactivity in Ottawa, Canada. In R.L. Trites (Ed.), *Hyperactivity in children: Etiology, measurement, and treatment implications* (pp. 29-52). Baltimore: University Park Press.

Trites, R.L., Dugas, E., Lynch, G., & Ferguson, H.B. (1979). Prevalence of hyperactivity. *Journal of Pediatric Psychology, 4*, 179-188.

Trommer, B.L., Hoeppner, J.B., Lorber, R., & Armstrong, K. (1988). Pitfalls in the use of a continuous performance test as a diagnostic tool in attention deficit disorder. *Journal of Developmental and Behavioral Pediatrics, 9*, 339-345.

Trommer, B.L., Lorber, B., & Armstrong, K. (1987). Neuropsychological correlates of a continuous performance test in attention deficit disorder. *Pediatric Research, 21*, 185A.

Ullman, D.G., Barkley, R.A., & Brown, H.W. (1978). The behavioral symptoms of hyperkinetic children who successfully responded to stimulant drug treatment. *American Journal of Orthopsychiatry, 48*, 425-437.

Ullmann, R.K., & Sleator, E.K. (1985). Attention deficit disorder with or without hyperactivity: Which behaviors are helped by stimulants? *Clinical Pediatrics, 24*, 547-551.

University of California Press (1983). *BMDP Statistical Software*. UCLA: Author.

Varley, C.K. (1983). Effects of methylphenidate in adolescents with attention deficit disorder. *Journal of the American Academy of Child Psychiatry, 22*, 351-354.

Varley, C.K. (1984). Attention deficit disorder (the hyperactivity syndrome): A review of selected issues. *Developmental and Behavioral Pediatrics, 5*, 254-258.

Varley, C.K. (1985). A review of studies of drug treatment efficacy for attention deficit disorder with hyperactivity in adolescents. *Psychopharmacology Bulletin, 21*, 216-221.

Virkkunen, M., & Nuutila, A. (1976). Specific reading retardation in hyperactive child syndrome and juvenile delinquency. *Acta Psychiatrica Scandinavica, 54*, 25-28.

Vyse, S.A., & Rapport, M.D. (1989). The effects of methylphenidate on learning in children with ADDH: The stimulus equivalence paradigm. *Journal of Consulting and Clinical Psychology, 57*, 425-435.

Walker, E., Bettes, B., & Ceci, S. (1984). Teachers' assumptions regarding the severity, causes, and outcomes of behavioral problems in preschoolers. *Journal of Consulting and Clinical Psychology, 52*, 899-902.

Walker, J.L., Lahey, B.B., Hynd, G.W., & Frame, C.L. (1987). Comparison of specific patterns of antisocial behavior in children with conduct disorder with or without coexisting hyperactivity. *Journal of Consulting and Clinical Psychology, 55*, 910-913.

Weber, K. (1985). Methylphenidate: Rate-dependent drug effects in hyperactive boys. *Psychopharmacology, 85*, 231-235.

Wechsler, D. (1974). *Manual for the Wechsler Intelligence Scale for Children - Revised.* New York: Psychological Corporation.

Wechsler, D. (1981). *Manual for the Wechsler Adult Intelligence Scale - Revised.* New York: Psychological Corporation.

Weiss, G. (1975). 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,* 213-226.

Weiss, G. (1985). Pharmacotherapy for ADD-H adolescents workshop. Follow-up studies on outcome of hyperactive children. *Psychopharmacology Bulletin, 21,* 169-177.

Weiss, G., Hechtman, L.,Milroy, T., & Perlman, T. (1985). Psychiatric status of hyperactives as adults: A controlled prospective 15-year follow-up of 63 hyperactive children. *Journal of the American Academy of Child Psychiatry, 24,* 211-220.

Weiss, G., Hechtman, L., Perlman, T., Hopkins, J., & Wener, A. (1979). Hyperactives as young adults: A controlled prospective 10-year follow-up of 75 children. *Archives of General Psychiatry, 36,* 675-681.

Weiss, G., Minde, K., Werry, J., Douglas, V., & Nemeth, E. (1971). Studies on the hyperactive child: VIII. Five-year follow-up. *Archives of General Psychiatry, 24,* 409-414.

Weiss, G., & Trokenberg Hechtman, L. (1986). *Hyperactive children grown up.* New York: Guilford Press.

Weisz, J.R., Suwanlert, C., Chaiyasit, W., Weiss, B., Walter, B.R., & Anderson, W.W. (1988). Thai and American perspectives on over- and undercontolled child behavior problems: Exploring the threshold model among parents, teachers, and psychologists. *Journal of Consulting and Clinical Psychology, 56,* 601-609.

Weisz, J.R., & Weiss, B. (1991). Studying the "referability" of child clinical problems. *Journal of Consulting and Clinical Psychology, 59,* 266-273.

Wender, P.H. (1971). *Minimal brain dysfunction in children.* New York: Wiley-Interscience.

Wender, P.H., & Eisenberg, L. (1974). Minimal brain dysfunction in children. In S. Arieti (Ed.), *American Handbook of Psychiatry (Vol. 2)* (2nd ed.). New York: Basic Books.

Wender, P.H., Wood, D.R., & Reimherr, F.W. (1985). Pharmacological treatment of attention deficit disorder, residual type (ADD, RT, "minimal brain dysfunction", "hyperactivity") in adults. *Psychopharmacology Bulletin, 21,* 222-231.

Werner, E.E., Bierman, J.M., French, F.E., Simanian, K., Connor, A., Smith, R.S., & Campbell, M. (1968). Reproductive and environmental casualties: A report on the 10-year follow-up of the children of the Kauai pregnancy study. *Pediatrics, 42,* 112-127.

Werry, J.S. (1977). The use of psychotropic drugs in children. *American Academy of Child Psychiatry, 16,* 446-468.

Werry, J.S. (1978). Measures in pediatric psychopharmacology. In J.S. Werry (Ed.), *Pediatric psychopharmacology: The use of behavior modifying drugs in children* (pp. 29-78). New York: Brunner/Mazel.

Werry, J.S., & Aman, M.G. (1975). Methylphenidate and haloperidol in children: Effects on attention, memory, and activity. *Archives of General Psychiatry, 32*, 790-795.

Werry, J.S., Elkind, G.S., & Reeves, J.C. (1987). Attention deficit, conduct, oppositional and anxiety disorders in children: III. Laboratory differences. *Journal of Abnormal Child Psychology, 15*, 409-428.

Werry, J.S., Reeves, J.C., & Elkind, G.S. (1987). Attention deficit, conduct, oppositional, and anxiety disorders in children: I. A review of research on differentiating characteristics. *Journal of the American Academy of Child and Adolescent Psychiatry, 26*, 133-143.

Werry, J.S., & Sprague, R.L. (1970). Hyperactivity. In C.G. Costello (Ed.), *Symptoms of psychopathology.* New York: Wiley & Sons.

Werry, J.S., Sprague, R.L., & Cohen, M.N. (1975). Conners' teacher rating scale for use in drug studies with children - An empirical study. Journal of Abnormal Child Psychology, 3, 217-229.

Whalen, C., & Henker, B. (1976). Psychostimulants in children: A review and analysis. *Psychological Bulletin, 83*, 1113-1130.

Whalen, C.K., & Henker, B. (1980). The social ecology of psychostimulant treatment: A model for conceptual and empirical analysis. In C.K. Whalen & B. Henker (Eds.), *Hyperactive children: The social ecology of identification and treatment* (pp. 3-51). New York: Academic Press.

Whalen, C.K., & Henker, B. (1984). Hyperactivity and the attention deficit disorders: Expanding frontiers. *Pediatric Clinics of North America, 31*, 397-427.

Whalen, C.K., & Henker, B. (1991). Therapies for hyperactive children: Comparisons, combinations, and compromises. *Journal of Consulting and Clinical Psychology, 59*, 126-137.

Whalen, C.K., Henker, B., Buhrmester, D., Hinshaw, S.P., Huber, A., & Laski, K. (1989). Does stimulant medication improve the peer status of hyperactive children? *Journal of Consulting and Clinical Psychology, 57*, 545-549.

Whalen, C.K., Henker, B., Swanson, J.M., Granger, D., Kliewer, W., & Spencer, J. (1987). Natural social behaviors in hyperactive children: Dose effects of methylphenidate. *Journal of Consulting and Clinical Psychology, 55*, 187-193.

White, J., Barratt, E., & Adams, P. (1979). The hyperactive child in adolescence. *Journal of the American Academy of Child Psychiatry, 18,* 154-159.

Wilder, J. (1957). The Law of Initial Value in neurology and psychiatry. *Journal of Nervous and Mental Disease, 125*, 73-86.

Winsberg, B.G., Kupietz, S.S., Sverd, J., Hungund, B.L., & Young, N.L. (1982). Methylphenidate oral dose plasm concentrations and behavioral response in children. *Psychopharmacology, 76*, 329-332.

Winsberg, B.G., Press, M., Bialer, I., & Kupietz, S.S. (1974). Dextroamphetamine and methylphenidate in the treatment of hyperactive/aggressive children. *Pediatrics, 53*, 236-241.

World Health Organization. (1978). *Glossary of mental disorders and guide to their classification* - for use in conjunction with the *International classification of disease* (9th revision). Geneva: World Health Organization.

Zambelli, A.J., Stam, J.S., Maintinsky, S., & Loiselle, D.L. (1977). Auditory evoked potential and selective attention in formerly hyperactive boys. *American Journal of Psychiatry, 134*, 742-747.

Zentall, S.S., & Zentall, T.R. (1967). Amphetamine's paradoxical effects may be predictable. *Journal of Learning Disabilities, 9,* 67-68.

Zentall, S.S., & Zentall, T.R. (1986). Hyperactivity ratings: Statistical regression provides an insufficient explanation of practice effects. *Journal of Pediatric Psychology, 11*, 393-396.