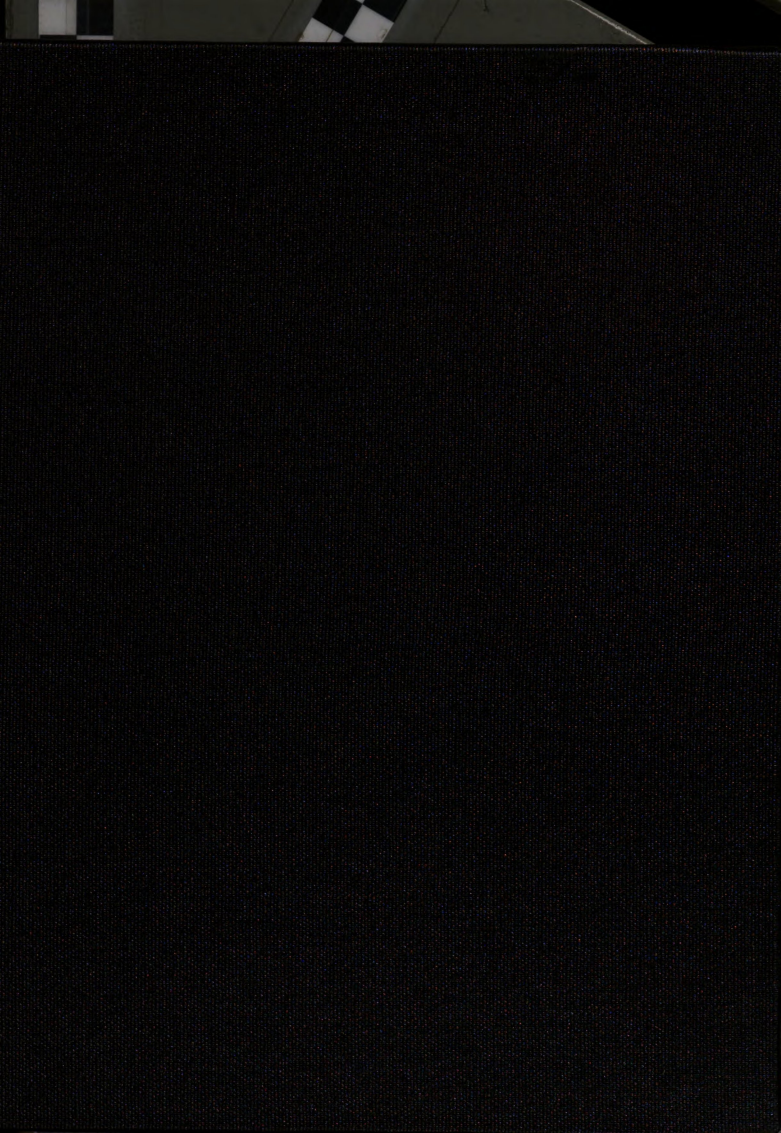

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THE RELATIONSHIP BETWEEN ALEXITHYMIC TRAITS IN PRIMARY CAREGIVERS AND A DIAGNOSIS OF ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

By

James Denison Jones

A DISSERTATION

Submitted to

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Abstract

THE RELATIONSHIP BETWEEN ALEXITHYMIC TRAITS IN PRIMARY CAREGIVERS AND A DIAGNOSIS OF ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

By

James Denison Jones

The relationship between alexithymic traits in primary caregivers and a diagnosis of Attention-Deficit/Hyperactivity Disorder (ADHD) in their children was evaluated in this study. Seventy-five children and adolescents were assessed at a small outpatient clinic. The children in the study completed a standard behavioral and neuropsychological assessment battery and their primary caregivers were administered standard behavior rating scales and the 20-Item Toronto Alexithymia Scale (TAS-20). Results revealed that primary caregivers of children with ADHD do not exhibit higher levels alexithymia than do primary caregivers of children who do not carry this diagnosis. In addition, the TAS-20 was not significantly correlated with two neuropsychological measures of inattention and inhibition. Overall, there was no support for the prediction of a positive relationship between children diagnosed with ADHD and alexithymic traits in their primary caregivers.

This dissertation is dedicated to my wife, Debra Jones, my family, and my friends.

Without their patience and support, this project would have been impossible.

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I want to acknowledge those colleagues and mentors who made this project possible. I would like to recognize Dr. Bertram Karon for his kind, insightful, and indispensable guidance. I would also like to mention my profound gratitude to Dr. Greg Lamberty, a mentor and friend who made this project possible. I would also like to recognize Dr. Jean Baker, Dr. Robert Caldwell, and Dr. Albert Rabin for their constructive and insightful contributions to this final product. All of the help is deeply appreciated.

TABLE OF CONTENTS

LIST OF TABLES

INTRODUCTION

Attention-Deficit/Hyperactivity Disorder (ADHD) is one of the most common and disruptive behavior disorders of childhood and adolescence. Symptoms of ADHD represent developmentally inappropriate levels of inattention, impulsivity, and hyperactivity (American Psychiatric Association, 1994). Because ADHD is one of the most commonly diagnosed and persistent of the childhood disorders, it carries a substantial risk for escalating problems (Klein & Mannuzza, 1991). Consequently, more research investigating potential risk factors for the disorder is needed.

As summarized in Robison, Sclar, Skaer, and Galin (1999), cross-national epidemiologic studies utilizing standardized diagnostic criteria suggest that 3% to 6% of the school-age population may suffer with ADHD. Similarly, in a Quebec study of prevalence rates of mental disorders, Breton et al. (1999) found rates of ADHD ranged from 3.8% to 9.8% for elementary school children; these rates decreased in high school. Zito et al. (1999) found that physician visits for children aged 5-14 years old with ADHD rose by 90% between 1989 and 1996 (as cited in Bonn, 1999). Estimates have had a wide range of variability that may be explained by differences in informants (parent or teacher), culture, and the degree of impairment needed for diagnosis (Elia, Ambrosini, & Rapoport, 1999). However, studies confirm an increasing trend in the diagnosis of ADHD (e.g., exceeding two million children; Erdman, 1998) and an increasing trend in the prescribing of stimulant pharmacotherapy for its treatment (Robison, Sclar, Skaer, & Galin, 1999). Though

there is general agreement regarding the prevalence and course of ADHD, the etiology of this behavioral disorder remains in question. Consequently, treatment recommendations are based upon arguable theoretical assumptions regarding the origins of the behaviors and neuropsychological impairments found in children diagnosed with ADHD.

Children with ADHD exhibit a wide range of problematic academic and interpersonal deficits, including poorer social skills than in children without this problem (Merrel & Wolfe, 1998). These children were found "especially lacking in social cooperation skills: the ability to follow rules, structure, and important social expectations of both children and adults" (p. 101). It has also been reported that noncompliance, interpersonal friction with adults, peer rejection, aggression, and school problems are associated impairments related to ADHD (Melnick & Hinshaw, 2000). Consistent with a plethora of research articles regarding neurological deficits in ADHD, Seidman et al. (1995) found that ADHD children performed more poorly on tasks of attention, executive function, and learning and memory, than did normal controls. Additionally, "executive function" measures of planning or controlling motor output were more impaired in ADHD than in comparison boys (Nigg, Hinshaw, Carte, & Treuting, 1998). Barkley (1998) offers a comprehensive model of ADHD suggesting that ADHD is a developmental disorder of behavioral inhibition, inattention, and self-regulation. The "developmental delay in inhibition gives rise to deficits in the executive functions that subserve self-regulation. It is these secondary deficits that result in inattentive, distractible, impersistent, and poorly regulated behavior" (p. ix).

The most widely researched etiological models concerning the development of ADHD suggest that this disorder is one that develops because of genetic or biologic factors or that it is "largely a result of neurological dysfunction" (Frick & Lahey, 1991, p. 169). Information distributed to parents of children with ADHD makes these claims clearly. For example, Harvey Parker, Ph.D., co-founder of the largest national ADHD organization (Children and Adults with Attention Deficit Disorders; C.H.A.D.D.) reported that ADHD is a "neurobiological disorder...due to a chemical imbalance" (Merrow, 1995).

Given the pervasive belief that ADHD is a genetic or biological disorder, parenting problems, family factors, and traumatic life events in the lives of children diagnosed with ADHD may actually be under-reported or under-estimated (e.g., Overmeyer, Taylor, Blanz, & Schmidt, 1999). Parenting characteristics, particularly the role of the primary caregiver's emotional availability and ability to be attuned and empathic, may be one risk factor for the etiology or exacerbation of the developmental struggles faced by children with ADHD. A disturbing trend was noted by Gibbs (1998), in that the percentage of children with an ADHD diagnosis walking out of a doctor's office with a prescription for stimulants dramatically increased from 55% in 1989 to 75% in 1996. The number receiving psychotherapy fell from 40% in 1989 to 25% in 1996. This trend suggests that treatment options are being limited by theoretical assumptions regarding the etiology and symptoms of ADHD. In this proposal, I will review evidence supporting the hypothesis that a parental personality trait, alexithymia (described below) might be one risk factor in the child's development of ADHD. This contextual factor seems to more accurately represent

the current state of the developmental literature regarding the influence of early parenting for later problematic behaviors.

The charge that many children are misdiagnosed, might be correct. The DSM-IV requires that ADHD "must be distinguished from difficulty in goal-directed behavior in children from inadequate, disorganized, or chaotic environments..." (American Psychiatric Association, 1994, p. 83). It is felt by some that abnormal psychosocial factors are under-detected in the lives of children with ADHD if "clinicians...presume a biological aetiology" (Overmeyer, Taylor, Blanz, & Schmidt, 1999, p. 262). They found that clinicians who knew the diagnosis of the child for whom they were doing an assessment found different rates of psychosocial adversity in the histories of children with conduct disorder and children with hyperkinetic problems. However, when clinicians blind to the diagnosis did the same interviews, they found no difference in rates of psychosocial adversity between the two types of disorders; this finding was contrary to the existing literature. Similarly, in a study of parental attribution styles for child behavior, parents of ADHD children were less likely to see themselves as the cause of child behavior and were more likely to mention medication (Johnston, Reynolds, Freeman, & Geller, 1988). These authors also reported that the attributions they found were consistent with current theoretical views of ADHD as a primarily biological condition amenable to pharmacological treatment and may reflect information parents have received from health care professionals, parent support groups, and other sources. Finally, criticisms have been leveled that "the diagnosis meets the needs of the parents more than it does that of the child" (Smelter et al., 1996, p. 430). Psychiatrist Peter Breggin, referring to materials

given to parents of children with ADHD which blame the child for causing parental frustration and upset, reports that "there could be no better example of pedist [i.e., prejudice against children] child-blaming and mistaken parental exoneration" (Breggin & Breggin, 1994, p. 89).

Crittenden (1992) stressed the importance of considering the behaviors of these children in the context of their function, rather than to simply look at isolated overt behaviors. Additionally, DeKlyen, (1996) found a link between childhood disruptive behavior disorder, the quality of the mother-child interactions, and the mother's recollections of her attitudes toward her parents. Similarly, Harnish, Dodge, and Valente (1995) reported that "children who experienced negative-quality interactions with their mothers were more likely than children who experienced positive-quality interactions to have increased levels of externalizing behavior problems" (p. 749). Another study, looking at contextual factors, found that the strongest factors in accounting for ADHD-type behaviors (and change in such behaviors) were measures of relationship status at birth, social support for the parent, and the direct measures of parental overstimulation (Carlson, Jacobvitz, & Sroufe, 1995).

Heritability and the influence of parenting on development

Attention-Deficit/Hyperactivity Disorder has been reported to be a genetic disorder (Barkley, 1998). Despite some assertions that there is little evidence of parents' influence on behavior and personality in adolescence and adulthood (e.g., Harris, 1998; Rowe, 1994), recent evidence suggests that this may not be the final word (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000). A recent

meta-analysis concluded that heredity rarely accounts for as much as 50% of the variation in heritable traits among individuals in a particular population, perhaps even less when personality characteristics are the focus (McCartney, Harris, & Bernieri, 1990). Lastly, even highly heritable traits can be highly malleable (Collins, Maccoby, Steinberg, Hetherington, & Bornstein, 2000). For example, the Finnish adoption study (Tienari et al, 1994) suggests how a genetic predisposition can either manifest itself or not, depending on whether certain environmental triggering events are present.

Even though "parental behavior is influenced by child behavior, parents' actions contribute distinctively to the child's later development" (Collins et al., 2000, p. 222). The style and quality of parenting, to some extent, moderates associations between early temperamental characteristics of difficultness, impulsivity, and unmanageability and later externalizing disorders (Bates, Pettit, Dodge, & Ridge, 1998; Rothbart & Bates, 1998). On the adaptive side, Collins et al., (2000) concluded

> findings suggest that well-functioning parents can buffer children at genetic risk and circumvent the processes that might ordinarily lead from genotype to phenotype. The more general point is that genetic vulnerabilities (or strengths) may not be manifested except in the presence of a pertinent environmental trigger such as parenting (p. 223).

The human infant is born with relatively few of its neural pathways committed (Hoffer, 1984). Differences in the social environment of an individual during development can result in changes in the biology of the brain and body just as much as in behavior (Lewontin, Rose, & Kamin, 1984, p. 142). "During its long infancy,

connections between nerve cells are formed on the basis not merely of specific epigenetic programming but in light of experience" (p. 145). Endocrine, immune, cardiovascular, metabolic, and neurochemical variables are all hidden within the mother-infant interaction (Hofer, 1984). More specifically, Schore (1994) theorizes that the degree to which the mother stimulates and modulates the infant's affect-arousal states, and maintains them within a moderate range, may influence the balance between sympathetic and parasympathetic components of the autonomic nervous system" (as cited in Fukinishi, Sei, Morita, & Rahe, 1999). Empirical evidence suggests that brain development in young organisms is dependent, in part, on experience in humans (e.g., Schore, 1994) and non-human animals such as rats (e.g., Caldji et al., 1998; Liu et al., 1997) and Rhesus monkeys (Suomi, 1997; as cited in Collins et al., 2000).

Parental behavior has been shown to be highly stable across time (Holden & Miller, 1999), however, changes in parenting practices are found to be associated significantly with changes in teacher-reported school adjustment and with changes in both child-reported and parent-reported maladjustment (Forgatch & DeGarmo, 1999). Parents also "mediate the association between broader social, cultural, economic, and historical contexts and children's behavior and personality" (Collins et al., 2000, p. 228).

> Infants of mothers with psychosocial problems are found to be less likely to develop secure attachments [as defined by attachment theory] in the first place, suggesting both direct and indirect pathways for the influence of maternal problems on later child adaptation (Lyons-Ruth, Alpern, & Repacholi, 1993, p. 582).

The development of affects and affect-regulating capacities is facilitated early in life by the experience of affect sharing and "mirroring" of affective expressions with the primary caregiver (Krystal, 1988; McDougall, 1988, Taylor, Bagby, & Parker, 1997). The presence and activities of the infant stimulate a set of maternal behaviors needed by the infant...and these reciprocal maternal behaviors serve to facilitate the infant's adaptation and development (Stern, 1985). Therefore, evidence suggests that both genes and parenting affect brain processes and neuroendocrine systems (Collins et al., 2000). One current model of these processes will be explored in detail in this study, though it may not be the only pathway.

A contextual model of ADHD as an alternative to biological determinism

In this proposal, I will offer a alternate consideration to the largely biologically deterministic views within most ADHD researchers' reports. In general, adherents to biological etiological theories of emotional and behavioral disorders

> answer that human lives and actions are inevitable consequences of the biochemical properties of the cells that make up the individual; and these characteristics are in turn uniquely determined by the constituents of the genes possessed by each individual (Lewontin, Rose, & Kamin, 1984, p. 6).

If the scientific and lay communities assume a genetic or purely neurodevelopmental etiology of ADHD, then it becomes easy to dismiss important contextual/environmental variables which may have direct impact on the development of the symptoms associated with ADHD and treatment. In fact, it is commonplace to read that the differences that are found in parenting styles and practices are the result, rather than a possible cause, of the disordered behavior of these children (e.g., Barkley, 1998). He writes,

> ADHD is associated with an impairment in self-regulation and this impairment must radiate into the social ecology of these children, affecting others and the manner in which they may reciprocate (p. 143). In view of the twin studies...that show minimal or nonsignificant contributions of the common or shared environment to the expression of symptoms of ADHD, theories based entirely on social explanations of the origins of ADHD are difficult to take seriously any longer (p. 175). Thus, common parenting factors are not viewed as a major contributor to the occurrence of ADHD symptoms or the disorder in children (p. 176). The overly critical, commanding, and negative behavior of mothers of hyperactive children is most likely a reaction to the difficult, disruptive, and noncompliant behavior of these children rather than being a cause of it (p. 176)

Hechtman (1996), in her review of families of children with ADHD, also reported that negative parenting styles of mothers of hyperactive children were probably a consequence of the child's behavior. Barkley (1998) did report, however, that cases of ADHD can also occur without a genetic predisposition, "provided the child is

exposed to significant disruption or neurological injury to this final common neurological pathway (the prefrontal cortical-striatal network;" p. 177). He felt, however, that this would seem to account for a small minority of ADHD children. Biederman et al (1993) reported that indices of psychosocial adversity were predictive of ADHD outcome (as cited in Seidman et al., 1995). Perhaps the role of parental alexithymia and its influence on attachment behaviors is one example of "significant disruption" to which Barkley concedes. While there is little doubt that there is complex interaction between parenting behaviors and children's behavioral and emotional problems, the tone of many articles and books regarding ADHD strikes this reader as blaming the child. Fortunately, the scientific community is not of one mind in this regard.

Erdman (1998), in her paper conceptualizing ADHD as a contextual response to parental attachment, reported that even diagnoses of ADHD that are clinically valid send a message to the parent and to the child that the problem lies within the child, when in reality it is a family issue (p. 182). "The repercussions of viewing ADHD as an individual problem to be treated solely with medication, behavioral management techniques, or both, has serious implications" (p. 182). Specifically, "children who are already frustrated and anxious over the lack of a parental bond will feel more victimized if they are focused on as the problem" (p. 182). Similarly, on a more philosophical level, Lewontin, Rose, and Kamin (1984) wrote:

> The disordered brain is seen as the cause of an unacceptable interaction of individuals and social organizations. The political consequence is that, since the social institution is never questioned, no alteration in it is

> therefore contemplated; individuals are to be altered to fit the institutions or else sequestered to suffer in isolation the consequences of their defective biology (p. 21)

If we translate the notion of "social organization" into the more immediate family and school organizations, the implications are clear. The consistent predictive power of caregiving and contextual factors to distractibility and later hyperactivity, however, suggests that there may be significant psychogenic contributions to ADHD for many children (Carlson, Jacobvitz, & Sroufe, 1995, p. 52). In their longitudinal analysis of families, they reported that relationship stability and external emotional support of the primary caregiver emerged as particularly influential factors in the development of ADHD symptoms. (p. 52). Such contextual considerations may be crucial to the prevention or amelioration of symptoms for some children and appear to be important to an understanding of the course and treatment of the disorder regardless of etiology (Carlson, Jacobvitz, & Sroufe, 1995, 54). Speltz (1990) explained conduct problems—which he defined as chronic noncompliance, aggressiveness, and frequent discontrol—as an outcome of the absence of immediate, consistent, and developmentally appropriate parental responses to children's behaviors.

Studies confirm that parents of ADHD children are more likely to have ADHD, or some residual characteristics (e.g., Alberts-Corush, Firestone, & Goodman, 1986; Barkley, 1998). Additionally, parents of ADHD children are more likely to experience a wide variety of other psychiatric disorders, including conduct problems and antisocial behavior, alcoholism, and affective disorders (Cantwell, 1972; Faraone & Biederman, 1997; Morrison & Stewart, 1973; Singer et al., 1981, as

cited in Barkley, 1998, p. 146). Parents who exhibit affectionless control are more likely to have children with anxiety disorders, obsessive-compulsive disorder, and attention deficit disorder (Hafner & Spence, 1988; Parker, 1984).

Barkley's neuropsychological model of ADHD

Barkley's (1998) conceptual model of ADHD is a developmental neuropsychological model of human self-regulation (p. 229). A comprehensive review of this model and supporting literature is prohibitive in length; for a review look to Barkley's (1998) text. A deficit in inhibition has become a theoretical assumption, replacing the older importance placed on hyperactivity. Wender's theory (1971) of "minimal brain dysfunction," a precursor to the more modern ADHD diagnosis, gave a prominent role to the construct of poor inhibition. He believes it to explain the activation difficulties and the attentional problems that stem from them, as well as the excessive emotionality, low frustration tolerance, and hot temperedness of ADHD children (as cited in Barkley, 1998, p. 12). Irritability, hostility, excitability, and a general emotional hyperresponsiveness toward others have been frequently described in the literature (Barkley, 1990). Later he summarized findings that suggest that emotional self-control may be problematic for children with ADHD (Barkely, 1998). Barkley's (1998) model:

> specifies that behavioral inhibition, representing the first and foundation component of the model, is critical to the proficient performance of the four executive functions: non-verbal working memory, internalization of speech (verbal working memory), the self-regulation of affect/motivation/arousal, and reconstitution (p. 229).

Deficits in any particular executive function will produce a relatively distinct impairment in self-regulation, different from that impairment in self-control produced by deficits in other functions (p. 234). On measures of sustained attention (specifically of the goal-directed type) the prefrontal cortex and the executive functions that the cortex and its networks permit, are essential (p. 246). Behavioral inhibition and at least three of the executive functions appear to be mediated by separate but surely interacting regions of the prefrontal lobes. Behavioral inhibition and its component processes seem to be localized in the orbital-frontal regions and its interconnections to the striatum.

> Behavioral inhibition delays the decision to respond to an event. The self-directed actions occurring during the delay in the response constitute the executive functions....Over development, they may become progressively more private or covert in form. The development of internalized, self-directed speech seems to exemplify this process. Although eventually "internalized," these self-directed actions remain essentially self-directed forms of behavior despite the fact they have become disengaged form their more obvious and public motor manifestations (muscoloskeletal movements). Therefore, the term "executive function" refers here to those self-directed actions of the individual that are being used to self-regulate (p. 233).

In summary, ADHD is not just a deficit in behavioral inhibition, but also a deficit in executive functioning and self-regulation as a consequence of that inhibitory impairment. "The deficit results in a renegade motor control system that is not under

the same degree of control by internally represented information, time, and the future" (249).

Alternatives to pharmacological treatment for ADHD

The most consistently recommended and implemented treatment for children with ADHD is stimulant medications, such as methylphenidate. When considering Barkley's theoretical model of ADHD, he felt that only a treatment that improves the underlying neuropsychological deficit in behavioral inhibition is likely to result in an "improvement or normalization of the executive function dependent on such inhibition" (Barkley, 1998, p. 252). "To date," he wrote, "the only treatment that exists that has any hope of achieving this end is stimulant medication or other psychopharmacological agents that improve or normalize the neural substrates in the prefrontal regions that likely underlie this disorder" (p. 252). Given this belief, pharmacological treatments for ADHD are far more widely employed, are less expensive, and have much more short-term empirical support than psychosocial treatments (Pelham, Wheeler, & Chronis, 1998). However, Barkley also reminds us that behavioral interventions and parent training programs are also useful for children with ADHD and their families (Barkley, 1998).

However, McGuiness (1989) reviewed follow-up studies showing that children taking psychostimulant medication for long periods of time feel worthless and have low self esteem. Another major limitation of stimulant therapy is that long-term studies (up to five years) fail to provide any evidence that the drugs improve ADHD children's long-term prognosis (Charles & Schain, 1981; Weiss & Hechtman, 1993).

Widener (1998) found improvement in a patient's ADHD symptoms when his underlying feelings of depression, sadness, and loneliness have been addressed in psychodynamic psychotherapy. She feels that the child's hyperactivity may actually be a way the child signals the presence of very painful affects (p. 274). She also reported that some of the changes in the child being treated were dependent on the mother's therapeutic work which resulted in her recognition of unresolved issues with her own mother and her feelings about her self-as-mother. Orford (1998) offers one therapeutic approach, by stating

> what is effective with these children is help in organizing the terrifying chaos of their inner worlds; it is a terrifying chaos that has been with them since babyhood, which was not regulated at the time within the maternal environment and which has led to subsequent habitual and primitive responses of a hyperactive, hypervigilant kind (p. 264).

In my own clinical experience working with patients diagnosed with ADHD, I have also found a dramatic and immediate attenuation of ADHD behavioral symptoms when unacknowledged, uncomfortable feelings have been interpreted and brought into the clinical exchange. When patients are able to find words for and express their underlying feelings, the behavioral symptoms, likely serving a defensive/protective function, are no longer needed to cope with them (i.e., keeping them at bay).

Alexithymia

The construct of alexithymia originally came from within the field of psychosomatic medicine, but was later described also by authors from within

psychoanalysis. According to his review (Taylor, 2000), this personality construct has gradually captured the attention of "many psychiatrists and psychologists throughout the world" (p. 134). He conducted a database review and found that 120 articles were published on alexithymia by the mid-1980's, but since, well over 700 journal articles have been published on alexithymia. Alexithymia "reflects deficits in the cognitive processing and regulation of emotion" (p. 135). Sifneos (1973) coined the term alexithymia (from the Greek: a = lack, lexis = word, thymos = emotion) to denote a cluster of cognitive and affective characteristics. Taylor et al. (1988) described alexithymia, in accordance with formulations by Nemiah and Sifneos (1970), as a "multidimensional construct defined by the following cognitive-affective characteristics: a) a difficulty in identifying and describing feelings, b) a difficulty in distinguishing between feelings and bodily sensations, c) a paucity of fantasies, and d) a preoccupation with external events" (p. 500). Empirical evidence shows that alexithymia is associated with difficulties in discriminating among different emotional states (Bagby et al, 1993), and with a limited ability to think about and use emotions to cope with stressful situations (Parker, Taylor, & Bagby, 1998; Schaffer, 1993). Other features of alexithymia include: a tendency toward social conformity, a tendency toward action to express emotion or to avoid conflicts, an infrequent recollection of dreams, a somewhat stiff wooden posture, and a paucity of facial emotional expressions (Krystal, 1979; Nemiah, Freyberger, & Sifneos, 1976; Ruesch, 1948; Sifneos, Apfel-Savitz, &Frankel, 1977).

This construct is best considered as a personality dimension, rather than as a dichotomous category (Salminen, Saarijarvi, & Aarela, 1995). There is "consensus in

the literature on the definition of the alexithymia construct, and there is now a substantial body of empirical data supporting the validity of the construct" (Taylor, 1997, p. 267).

Alexithymia as distinct from other forms of psychopathology

Individuals with alexithymic features have historically been labeled as "normopaths" (McDougall, 1984) or "normotics" (Bollas, 1987), and are similar to descriptions of people who have "inner blindness" (Horney, 1952). Despite its origins from within psychosomatic medicine, alexithymia has not been found to be a personality dimension that is specific to psychosomatic patients (Ahrens & Deffner, 1986; Salminen, Saarijarvi, & Aarela, 1995). Alexithymic features have been found to be common in patients suffering from: anorexia nervosa, bulimia, obesity, substance abuse, depression, panic disorder, post-traumatic stress disorder, perverse sexual behavior, and hypochondriasis and somatization (Taylor, Bagby, & Parker, 1997; Salminen, Saarijarvi, & Aarela, 1995). Importantly, however, alexithymia has been found to be distinct from each of these disorders (Wise, Mann, & Randell, 1995; Parker, Bagby, Taylor, 1991).

These deficits in representing and regulating emotions cognitively are thought to render alexithymic individuals more susceptible to a variety of medical and psychiatric illnesses (Taylor, Bagby, & Parker, 1997). Studies comparing alexithymia (as measured by the Toronto Alexithymia Scale; TAS) and depression (as measured by the Beck Depression Inventory; BDI) have yielded consistent evidence that alexithymia is a separate and distinct construct (Parker, Bagby, & Taylor, 1991). "While there is some shared variance between measures of depression, anxiety, and

alexithymia, the data from prospective studies with various medical and psychiatric populations indicate that alexithymia is not simply a secondary response to anxious or depressed states" (Parker & Taylor, 1997, p. 73). Treatment studies of persons with substance or alcohol abuse problems show that after brief treatment periods, levels of depression and psychological distress dropped significantly, while mean alexithymia scores were not significantly lowered (Haviland, Shaw, Cummings, & MacMurray, 1988; Keller, Carroll, Nich, & Rounsaville, 1995; Pinard, Negrete, Annable, & Audet, 1996). "These each lend support for the view that alexithymia is not merely a state phenomenon secondary to depression or withdrawal" (Taylor, 1997, p. 188).

Further considerations of the alexithymia construct

Contemporary psychoanalytic theoreticians are of the opinion that serious or long-term interferences in early life, such as the unavailability of an empathic and responsive parent, may lead to stagnation in emotional development, which reveals itself in later life as alexithymia (Taylor, Bagby, & Parker, 1997). While some researchers believe alexithymia has a neurobiological basis (Dewaraja & Sasaki, 1990; Zeitlin, Lane, O'Leary, & Schrift, 1989), others feel that alexithymia may be caused by dysfunctional family environments, especially if the dysfunction was experienced during critical developmental periods in the first few years of life (Crittenden, 1994). For example, in a study of Japanese college students (Fukunishi, Sei, Morita, & Rahe, 1999), it was found that poor parental bonding is related to the perceived difficulty in articulating feelings. They also found that alexithymia and poor maternal care is related to high sympathetic activity during the resting period. Additionally, Krystal (1988) has also written extensively about the alexithymic

characteristics of people who have survived infantile or adult trauma, such as being interned at a Nazi concentration camp.

McDougall (1984) in her psychoanalytic paper on the "dis-affected" patient discovered that patients who were unaware of their affective reactions and internal feeling states, would immediately disperse into action any emotional arousal.

> In other words, these patients, instead of capturing and reflecting upon the emotional crises that arose in their daily lives or in the analytic relationship, would tend to act out their affective experiences, discharging them through inappropriate action rather than 'feeling' them and talking about them in the sessions (p. 388).

In her work with patients with "dis-affected" presentations, she also reported that she was able to reconstruct a paradoxical mother-child relationship in which the mother seems to have been out of touch with the infant's emotional needs, yet at the same time has controlled her baby's thoughts, feelings, and spontaneous gestures in a sort of archaic "double-bind" situation (p. 391).

According to Taylor, Bagby, and Parker (1997), alexithymia is not a culture-bound construct, as evidenced by both clinical and non-clinical populations in diverse cultures, and by successful cross-validation studies of a self-report measure of the construct in many different countries, including Italy, India, Japan, and Korea. Additionally, there is strong support for alexithymia being a stable personality feature, rather than just a consequence of psychological distress (p. 37). Alexithymia was also found to be unrelated to age, gender, educational level, socioeconomic

status, vocabulary skills, and general intellectual ability (Parker, Taylor, & Bagby, 1989).

People with alexithymic features have been found to have a host of psychological adjustment problems associated with an impaired ability to know and label their feelings. Individuals with alexithymic traits are in a "state of internal alarm" (due to higher vegetative arousal), exhibiting high anxiety and high level sympathetic reactivity, which is expressed somatically (Infrasca, 1997, p. 279). Also, alexithymia is associated with primitive (Kooiman, Spinhoven, Trijsburg, & Rooijmans, 1998) or immature (Parker, Taylor, & Bagby, 1998) defensive styles. Alexithymic individuals are prone to use action-oriented behaviors such as binging on food or abusing alcohol, seemingly in an attempt to regulate distressing emotional states (Lane & Schwartz, 1987; Taylor, Bagy, & Parker, 1997). Schaffer (1993) showed that highly alexithymic individuals tend to employ oral and somatic styles of affect regulation, such as bingeing on food or developing somatic symptoms. Alexithymic individuals are prone to high levels of negative emotion and somatic distress (Taylor, Bagby, & Parker, 1997; Bagby, Taylor, & Parker, 1990). They have also been shown to have an impaired ability to recognize both verbal and nonverbal emotional stimuli (Lane et al., 1994). For alexithymic people, there is not only a difficulty in expressing emotions verbally but a deficit in their cognitive processing (Salminen, Saarijarvi, & Aarela, 1995). This causes emotions to remain undifferentiated and poorly regulated (Taylor, Bagby, & Parker, 1991).

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Parental alexithymic features, empathy, attachment, and affect regulation

Because of the dual process of acquisition of affect tolerance (by learning and by identification), children whose parents have difficulty in handling affect also have difficulty in developing advantageous ways of dealing with their emotions (Krystal, 1988). These difficulties are not likely to be under conscious control, as it has been reported that parents vary in their own responsiveness and sensitivity to their babies' communications depending on their own early experiences (Orford, 1998). Securely attached individuals showed lower levels of alexithymia, and employed interpersonal behavior and fantasies of talking to a caring person to help regulate affect (Schaffer, 1993). According to Taylor, Bagby, and Parker (1997),

> ...the development of affect and affect regulating capacities is facilitated early in life by the experience of affect sharing and "mirroring" of affective expressions with the primary caregiver, and subsequently by engaging in pleasurable playful interactions and being taught to name and talk about feelings (p. 41).

Accurate empathic attunement has been found to be integral to the attachment dynamic (McCluskey, Hooper, & Miller, 1999).

Empathy involves the ability to understand both self and others. Tangey (1991) reports that other-oriented empathy requires the ability to take another's perspective, reading the other's internal emotional experiences, and being capable of experiencing a range of emotional states. When the primary caregiver is emotionally unavailable or when the child is subjected repeatedly to inconsistent responses because of parental "misattunement," the child is likely to manifest abnormalities in

affect development and affect regulation, as well as an insecure attachment style (Emde, 1988a,b; Osofsky, 1992; Slade & Aber, 1992; Stern, 1985). Maternal sensitive responsiveness is defined by the attachment figures' success in fitting their own response patterns to those of their offspring in ways that are mutually satisfying (Crittenden, 1995). "Anything else must, by definition, be considered insensitive" (p. 389). Research studies on attachment styles in infancy and childhood have confirmed that the sensitivity and responsiveness of the primary caregiver to the child's emotional states is a major determinant of the way the child learns to regulate distressing affects and to relate to other people (Bretherten, 1985; Goldberg, MacKay-Soronka, & Rochester, 1994).

Normal affect development does not occur when the parents are unable to read the emotional cues of the infant, and fail to function as external regulators of the infant's emotional states (Taylor, Bagby, & Parker, 1997). Taylor (1992) links failures in physiological self-regulation to psychological deficits, particularly in recognition of moods, that stem from early attachment problems. Insensitive mothers impede an infant's ability to modulate affective expression and arousal (Susman-Stillman, Kalkoske, Egeland, & Waldman (1996). Successful affect development and regulation of the experience of secure attachment provides "predictable positive outcomes to affective communications, and thereby facilitates a satisfactory integration of affective information with cognitive information" (Crittenden, 1994, p. 25). Primary caregivers who have alexithymic features, cannot imagine themselves in another person's situation and are consequently unempathic and ineffective in modulating the emotional states of others (Goleman, 1995; Krystal, 1979; Lane &

Schwartz, 1987). In a study where participants were asked to rate the emotional content of ambiguous stimuli, it was reported that a general ability to perceive consensual emotional content in visual stimuli was most strongly associated with the ability to respond empathically (Mayer, DiPaolo, & Salovey, 1990).

One contextual variable: Attachment and ADHD

According to Bowlby (1951), "the quality of the parental care which a child receives in his earliest years is of vital importance for his future mental health" (p. 11). As reported in Bowlby (1988), prospective studies revealed that those children who showed an anxious avoidant pattern (of attachment) are likely to be described later as emotionally seeking of attention. Those who showed an anxious resistant pattern are also likely to be described as unduly seeking of attention and either as tense, impulsive, and easily frustrated or else as passive and helpless (p. 169). Results indicate that stability in family of origin, emotional stability at three weeks, and maternal empathy and confidence at 18 months were significantly related to infant-mother attachment security. (Wieczorek-Deering, Greene, Nugent, & Graham, 1991).

The attachment relationship is widely believed to exert significant influence on a child's later development (Susman-Stillman, Kalkoske, Egeland, & Waldman, 1996, p. 35). As is consistent with previous findings, they found a relationship between distractibility at 3 ½ years and ADHD symptoms at 6-8 years. However, although attachment theory has been linked to conduct disorders and other behavioral problems, it has not been linked to ADHD behaviors (Erdman, 1998, p. 184).

Critical periods, brain development, and affect regulation

An infant's genetic endowment (genotype) is transformed and modified, through the brain's plasticity, by environmental events. Evidence from the different domains of developmental neuroscience, neurobiology, behavioral neurology, and developmental psychology suggests that the experiences within the early mother-infant dyad are responsible for functional and structural brain maturation, particularly in the frontal lobe structures that mediate the executive functions outlined in Barkley's (1998) model of ADHD. For example, there has been found a striking similarity between ADHD symptoms and stress-induced prefrontal cortical deficits (Arnsten, 1999). Below is a review of one current model of a mechanism by which the infant gradually develops the ability to regulate its affect. This model might explain a mechanism involved in the development of ADHD.

During early infancy, the infant is ill-equipped to effectively regulate its affect, leaving it prone to being overwhelmed, and "because it lacks the means for modulation of behavior, which is made possible by the development of cortical control...[t]he role of higher structures is played by the mother; she is the child's auxiliary cortex" (Diamond, Balvin, & Diamond, 1963, p. 305, as cited in Schore, 1994, p. 30). It is during infancy and early childhood that the brain is more malleable to experience than the mature brain (Perry, Pollard, Blakely, Baker, & Vigilante, 1995).

> In the developing brain, undifferentiated neural systems are critically dependent upon a set of environmental and micro-environmental cues (e.g., neurotransmitters, cellular adhesion molecules, neurohormones,

amino acids, ions) to appropriately organize from their undifferentiated form (Perry et al., 1995, p. 275)

For instance, Weber and Sackheim (1978) reviewed developmental neurobiological studies and suggest that critical periods of development associated with significant changes in the environmental repertoire are correlated with shifts in nervous system growth and reorganization (as cited in Schore, 1994, p. 29). Mutually interactive experiences in the first 2 years are occurring while various sensory systems are maturing, and, these experiences are internalized as "permanent idiosyncratic modifications of the nervous system" (Freedman, 1981, p. 841).

Critical early affective transactions with the social environment are mentally stored (internalized) in the form of representations of the self interacting with significant objects (Schore, 1994, p. 25); these representations are the same as Bowlby's (1988) "internal working models." In a still-face paradigm, it was reported that very young infants (by four months) begin to internalize aspects of the maternal parenting environment in a manner that contributes to their response to stress and interpersonal regulation strategies (Kogan & Carter, 1996). From an attachment theory perspective, sensitive mothers are responsive to their children's needs, which helps infants modulate affective expression and arousal (Susman-Stillman, Kalkoske, Egeland, & Waldman, 1996). Attachment theorists have argued that attachment systems permit flexible responses to environmental circumstances, influence the regulation of emotion, and function through mental representations that children hold of themselves and their relationships with others (Cassidy, 1994).

Neurobiological studies reveal that the dramatic onset of function in the first 18 months of life reflects the immense synaptogenesis that occurs during this period of infancy. Indeed the specific period from 7 to 15 months (roughly Bowlby's period for the establishment of attachment patterns) has been shown to be critical for the myelination and therefore the maturation of particular rapidly developing limbic and cortical association areas (Kinney, Brody, Kloman, & Gilles, 1988).). At 18 to 20 months, representational processes mature rapidly and infants become substantially more symbolic and verbal than they have been previously (Zeanah, Boris, Bakshi, & Lieberman, 2000, p. 97). The onset of mature function in these corticolimbic structures is instrumental to the emergence of infant affective, affect regulatory, and cognitive processes (Schore, 1994, p. 30). According to Gilbert (1989), there is now convincing evidence indicating that this maturation is experience-dependent and directly influenced by the caregiver-infant relationship (as cited in Schore, 1994, p. 30).

Perry at al. (1995) reported that "abnormal micro-environmental cues and atypical patterns of neural activity during critical and sensitive periods...can result in malorganization and compromised function in brain-mediated functions such as humor, empathy, attachment, and affect regulation (p. 276).

Regarding the development of the ability to regulate one's affect, Wilson et al., (1990) asserted that secure attachment facilitates the transfer of regulatory capacities from caregiver to infant. Thompson (1990) underscores the cardinal principle that emotion is initially regulated by others, but as development proceeds it becomes increasingly self-regulated as a result of neurophyisiological maturation (as

cited in Schore, 1994, p. 31). In summary, these observations suggest that "the mother's external regulation of the infant's developing yet still immature emotional systems during particular critical periods may represent the essential factor that influences the experience-dependent growth of brain areas prospectively involved in self-regulation" Schore, 1994, p. 32). "The dyad's response to stressful transaction, such as occur in socialization experiences in the second year, are particularly instrumental to the final structural maturation of an adaptive cortical system that can self-regulate emotional states" (Schore, p. 1994, p. 33). "Early object relational experiences thus directly influence the emergence of a frontolimbic system in the right hemisphere than can adaptively autoregulate both positive and negative affect in response to changes in the socioemotional environment" (Schore, 1994, p. 33). These functional advances reflect the structural maturation of the right frontolimbic areas and the emergence of more complex and efficient delay and inhibitory operations that underlie regulatory capacities (p. 489). "Dyadic failures of affect regulation result in the developmental psychopathology that underlies various forms of later forming psychiatric disorders" (Schore, 1994, p. 33).

It is suggested that "a mother's lack of affective involvement thus produces a growth-inhibiting environment which severely retards the experience-dependent growth of right hemispheric temporal-frontal and frontal-subcortical connections that are necessary for the integrated functioning of the right cortex" (Schore, 1994, p. 485). Taylor (1987) describes:

> It is likely that the right hemisphere contributes to the development of reciprocal interactions within the mother-infant regulatory system,.

and when these are deficient the child may fail to develop a capacity for being "in tune" with himself (p. 191).

He reports that deficiencies in the early mother-infant relationship result in an alexithymic deficit associated with a limitation of symbolic function and an impaired capacity for self regulating emotional states and physiological functioning when under stress. Schore (1994) concludes that maternal stress regulating verbalizations are also internalized into the multimodal interactive representations that encode the self-caring functions of evocative memory. The child who is deprived of such affective communicational experiences in this critical period (second year) is in danger of developing the regulatory disturbance of alexithymia (p. 489).

The orbitofrontal region is critically involved in attachment processes (Steklis & Kling, 1985). Schore (1994) suggests that attachment experiences specifically and directly influence the early maturation of the orbitofrontal cortex, a corticolimbic structure that is critically involved in attachment processes (p. 255). Schore reports that maternal stress regulating object relations transactions "act as a selection pressure to critically shape...the maturing orbitofrontal cortex" (p. 256).

Studies of adult animals (Kolb, 1974; Ruesch & Shenkin, 1943) have shown that lesions in the orbital frontal cortex have resulted in hyperactivity (as cited in Schore, 1994, p. 94). Similarly, Arntsen (1999) reported that both animals and humans with lesions in the prefrontal cortex exhibit poor attention regulation, disorganized and impulsive behavior, and hyperactivity.

Conclusion

The child's nascent capacity to regulate its affect is dependent on an attuned and empathic caregiver. Among other factors, affect regulation is a key component in both cognitive and behavioral self-control, both of which are problematic for children with ADHD. Problems associated with the development of self-regulatory capacities are hypothesized to underlie some of the symptoms posed by current models of ADHD. Given the empirical and theoretical models outline above, "good enough" empathic attunement and mirroring of a child's emotional states seems essential for the successful development of the executive functions that underlie ADHD symptoms. Accordingly, a parental characteristic, such as alexithymia, might negatively affect the child's developing regulatory capacities and place them at risk for developing ADHD.

Hypotheses

In review of the current developmental literature and theoretical models of Attention Deficit-Hyperactivity Disorder (ADHD), alexithymia in primary caregivers is hypothesized to be a risk factor for the development of ADHD in their children. The following specific hypotheses were investigated in this study.

1) Primary caregivers of children diagnosed with ADHD will evidence greater levels of the alexithymia personality features as compared to caregivers of clinic-referred children who do not have this diagnosis (e.g., mood disorders and conduct problems).

2) Alexithymic features in primary caregivers will be more strongly correlated with subscales in the parent-rated behavioral measure consistent with symptoms associated with ADHD, irrespective of diagnosis. Specifically, alexithymic features will be more strongly correlated with the "Attention Problems" subscale than with the "Anxious/Depressed" subscale.
3) Alexithymic features in primary caregivers will not be found to correlate highly with age, socioeconomic status, or education levels of the primary caregivers.
4) Alexithymic features in primary caregivers will significantly correlate with the two neuropsychogical measures of sustained attention and inhibition assessed for the participants with ADHD.

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Methods

Participants

Participants for this study were drawn from a private psychological clinic in a small midwestern city containing a major university. The children and adolescents (age range of 6 to 18 years old, N = 75) were referred to this clinic for outpatient psychotherapy and/or neuropsychological testing due to emotional, behavioral, and/or academic difficulties. There were 19 females (12 in the ADHD group and 7 in the Non-ADHD group) and 56 males (26 in the ADHD group and 30 in the Non-ADHD group) included in this study. The sample was drawn from consecutive referrals to the clinic, rather than from a randomized sample.

There were two groups used in this study. The first was composed of children and adolescents who have a diagnosis of Attention-Deficit/Hyperactivity Disorder (ADHD). This diagnosis was made by examining parent and teacher behavioral rating scales, clinical interview, and neuropsychological testing; this is a "multi-method" approach to assessment and diagnosis recommended by Barkley (1998) and others. The second (comparison) group was composed of clinic-referred children who were being assessed, but who do not carry ADHD as a primary diagnosis. Parents or guardians of the referred patients were given a consent form prior to becoming participants in this study. The clinic-referred children and adolescents also carry a clinician-based primary DSM IV Axis I diagnosis that was determined independent of this research project.

The mean age within the ADHD group was 9.18 (SD = 3.51), while the mean age within the Non-ADHD group was 11.05 (SD = 3.55). This is a significant

difference between the two groups (p = .025; results shown in Table 1). The Non-ADHD group was composed of children with the following diagnostic classifications: mood/depressive

disorders (n = 6), conduct/oppositional disorders (n = 5), adjustment disorders (n = 8), anxiety disorders (n = 5), learning disabilities (n = 6), and neurological problems (e.g., Asperger's syndrome, n = 7).

Instruments

Child Behavior Checklist 4-18 (CBCL 4-18; Achenbach, 1991). This measure provides an objective assessment of the identified child's social and emotional functioning. It is designed to record, in a standardized format, children's competencies and problems as reported by their parents. This measure is included in the battery because a parent's views of their children's behavior is often crucial in determining what will be done about the behavior (Achenbach, 1991). The primary caregivers of clinic-referred children completed the CBCL 4-18. The measure can be filled out by most parents who have at least fifth grade reading skills; the measure takes 10-15 minutes to complete. The CBCL 4-18 was normed on healthy children (i.e., they had not recently received professional help for behavioral/emotional problems) 4 to 18 years of age; they were chosen to be representative of the 48 contiguous states with respect to SES, ethnicity, region, and urban-suburban-rural residence. This measure has solid psychometric properties in that both its reliability and validity have been extensively supported.

The CBCL 4-18 has been found to have very high test-retest reliability (intraclass correlation coefficient (ICC) = .952) for the 118 specific problem items used in this

Table 1

Demographic variables by diagnostic groups analysis of variance (n=75)

	ADHD		Non-ADHD				
Variable	Means (SD)	n	Means (SD)	n	t-value	df	p
Child's Age	9.18 (3.51)	38	11.05 (3.55)	37	-2.29*	73	.025*
Grade Level	3.65 (3.64)	37	5.49 (3.70)	37	-2.15*	72	.035*
Mother's Age	36.18 (7.48)	38	36.83 (7.46)	36	-0.37	72	.71

$p < .05$

study. Additionally, the test-retest reliability of scale scores (test-retest rs) was .89 for the problem scales.

Content validity of the CBCL 4-18 is supported by the ability of nearly all CBCL 4-18 items to discriminate significantly between demographically matched referred and nonreferred children (Achenbach, 1991). Construct validity has also been supported by numerous correlates of CBCL 4-18 scales, including significant associations with

analogous scales on the Conners (1973) and Quay-Peterson (1983) scales (as cited in Achenbach, 1991, p. 109).

Only two of the CBCL Problem Subscales (Anxious/Depressed and Attention Problems) were utilized for this study. The scale has several more Problem scales, including: Withdrawn, Somatic Complaints, Social Problems, Thought Problems, Delinquent Behavior, Aggressive Behavior.

Conners Continuous Performance Test (Connors, 1995).. The standard Conners' Continuous Performance Test (CPT; 1995) is a test used to assess lapses in attention or vigilance and impulsivity (Spreen & Stauss, 1998, p. 236). This test is a computer-based assessment instrument that requires the test-taker to press the appropriate key for any letter except the letter X. There are six blocks, each with three 20-trial sub-blocks (letters presented, whether targets or not).

There are two primary types of errors included in the CPT scoring system. The first are errors of omission, which reflect deficits in sustained attention or vigilance (Halperin et al., 1991). The second are errors of commission, which appear to reflect a deficit of different underlying processes, including impulsivity and

inattention/memory (Halperin et al., 1991). Continuous performance tests have been found to be reliable tests for discriminating groups of ADHD from normal children (Corkum & Siegal, 1993). However, no single neuropsychological measure has been found to adequate to arrive at a valid diagnosis of ADHD.

Demographic/Social/Medical Questionnaire. The parents of the clinic-referred children were asked to fill out a detailed questionnaire regarding both the referred child and themselves. The questionnaire elicits detailed demographic information (e.g., age, sex, socioeconomic status, etc.) regarding both the referred patient and his/her parents, pregnancy and birth history of the referred child, social behavior questions, and medical/educational/psychological history questions.

Stroop Word-Color Test (Stroop, 1935). The Stroop Word-Color Test is a timed test measuring the ability to suppress or inhibit automatic responses. The test requires the test-taker to read the names of colors although the names are printed in a different colored ink from the color specified in the name; for example, the word "blue" is printed in green ink.

The Stoop test has generally been found to discriminate between ADHD children and control groups of children (Barkley, 1998). However, it has also been reported that this test has a fairly high false-negative rate of 53% (Barkley & Grodinsky, 1994). Consequently, this measure is not used exclusively to assess or diagnose ADHD, but is added to the test battery as one functional measure to tap deficits associated with ADHD.

The Twenty-Item Toronto Alexithymia Scale (TAS-20; Bagby, Taylor, & Parker, 1994a,b). This instrument is a 20-item self-report measure of alexithymia.

Items are rated on a five-point scale, ranging from "strongly disagree (1) to "strongly agree (5). This measure contains three subscales measuring: (factor 1) difficulty identifying feelings and distinguishing them from the bodily sensations of emotions; (factor 2) difficulty describing feelings to others, and (factor 3) an externally oriented cognitive style of thinking (Parker, et al., 1993; p. 223). These subscales are consistent with the theoretical underpinnings of the alexithymia construct.

The Toronto Alexithymia Scale (TAS), the measure upon which the TAS-20 improved and replaced, measured a personality dimension that is distinguishable from neurotic psychopathology (Taylor et al., 1988). The modifications made to the original scale did not change the basic utility of the original TAS, which was able to discriminate between clinically designated alexithymic and nonalexithymic patients. The authors reported internal consistency reliability coefficients for the subscales of .78, .75, and .66, respectively, good internal consistency (Crohnbach's alpha = 0.81), along with a three-week test-retest reliability of .77 for the total scale (Bagby, Taylor, & Parker, 1994). In this sample, the Crohnbach's alpha level for the TAS-20 Total Score was 0.79.

As with the original TAS (Taylor et al., 1988), the TAS-20 was also found to have construct validity with clinical interview ratings of alexithymia (Parker, et al., 1993) and with measures of psychological mindedness and need for cognition on the NEO Personality Inventory (Costa & McCrae, 1985). In their investigation of the TAS-20 and the NEO-PI, Bagby et al., (1994), discriminant validity of the TAS-20 was supported by the finding of non-significant correlations with the personality dimensions of Agreeableness and Conscientiousness. Convergent validity was

supported in the same study by the finding of negative correlations between the TAS-20 and the subscales assessing openness to feelings and openness to fantasy. According to Bagby and Taylor (1997), provides evidence that the TAS-20 is assessing deficiencies in emotional awareness and imaginal activity – salient features of the alexithymia construct" (p. 62).

Additional convergent validity of the TAS-20 has also been empirically established. The TAS-20 was assessed by examining the relationship of the scale with the Need for Cognition Scale (NCS; Cacioppo, Petty, & Kao, 1984) and the Psychological Mindedness Scale (PMS; Conte et al., 1990, 1995) in a sample of undergraduates (Bagby et al., 1994). This study found that the three factors of the TAS-20 all correlated strongly and negatively with both the NCS and the PMS.

Procedure

In this study, the primary caregivers of clinic-referred children filled out the standard behavioral measures, history questionnaires, and the alexithymia scale described above. Each was enlisted to participate in the study and had an informed consent form explained to them by their clinician. They understood that participation was voluntary and the data gathered will remain anonymous to the researcher. Participant confidentiality was maintained. The two neuropsychological measures (CPT and Stroop) were included in the standard clinical assessment battery used in the assessment of children and adolescents.

RESULTS

The statistics package STATISTICA was used for data analyses in this study. An alpha level of .05 was used for all statistical tests. In order to maximize sample size, each analysis was performed on the maximum number of cases (casewise deletion) that contained relevant data for each hypothesis. In some case, the sample sizes for individual analyses are smaller than the total sample due to casewise deletion.

Alexithymia and ADHD Diagnosis Hypothesis

The hypothesis stated that primary caregivers of children diagnosed with ADHD would evidence greater levels of the alexithymia, as measured by the 20-Item Toronto Alexithymia Scale (TAS-20), as compared to primary caregivers of clinic-referred children who do not have this primary diagnosis. This hypothesis was not supported (see Table 2). One-way ANOVA results revealed a significant main effect, contrary to that hypothesized, between TAS-20 scores and diagnostic groups, $F(1,73) = 7.07$, $p = 0.01$. In order to control for the possible effect of gender, an additional analysis was performed without including the girls in the sample. One-way ANOVA results still revealed a significant main effect between TAS-20 scores and diagnostic groups, $F(1,55) = 5.38$, $p = 0.024$, as shown in Table 3.

Alexithymia and CBCL subscale (Anxious/Depressed & Attention Problem) Hypothesis

The hypothesis stated that alexithymic features in primary caregivers would be more strongly correlated with subscales in the parent-rated behavioral measure (CBCL) consistent with symptoms associated with ADHD, irrespective of diagnosis;

Table 2

Diagnostic group by TAS-20 scores one-way ANOVA (N=75)

Variable	ADHD (n=38) Means (SD)	Non-ADHD (n=37) Means (SD)	df	F	p
TAS-20 Total	39.45 (9.68)	46.92 (14.28)	(1,73)	7.07**	.010**
TAS-1	11.68 (4.93)	14.65 (6.99)	(1,73)	4.52*	.037*
TAS-2	10.97 (4.32)	13.08 (5.02)	(1,73)	3.80	.055
TAS-3	16.79 (4.29)	19.19 (3.84)	(1,73)	6.50*	.013*

TAS-Total = TAS-20 Raw Total; TAS-1 = Difficulty identifying feelings; TAS-2 = Difficulty describing feelings; TAS-3 = Externally oriented thinking.

- * $p < .05$, ** $p < .001$

Table 3

Diagnostic group by TAS-20 scores one-way ANOVA (boys only, n=56)

Variable	ADHD (n=26) Means (SD)	Non-ADHD (n=30) Means (SD)	df	F	p
TAS-20 Total	38.46 (9.90)	46.40. (14.82)	(1,54)	5.38*	.024*
TAS-1	11.23 (4.71)	14.20 (7.21)	(1,54)	3.22	.078
TAS-2	10.92 (4.48)	12.93 (5.33)	(1,54)	2.29	.136
TAS-3	16.31 (4.38)	19.27 (3.86)	(1,54)	7.23*	.010*

TAS-Total = Raw Total; TAS 1 = Difficulty identifying feelings; TAS 2 = Difficulty describing feelings; TAS 3 = Externally oriented thinking

$p < .05$

this hypothesis was not supported. The correlation between TAS-20 Total Scores and the CBCL "Anxious/Depressed" and "Attention Problems" subscales was .09 and .07, respectively (see Table 4). The CBCL subscales were also not found to be significantly different from each other, given that they had positive correlations and neither was significantly different from zero.

Alexithymia and Demographics Hypothesis

The hypothesis stated that alexithymic features in primary caregivers would not significantly correlate with age (r = -.04) and socioeconomic status levels (r = -.11) of the primary caregivers; this hypothesis was supported (see Table 5).

Alexithymia and Neuropsychological Measures Hypothesis

The hypothesis stated that alexithymic features in primary caregivers would positively correlate significantly with the two neuropsychological measures of sustained attention and inhibition; this hypothesis was not supported. The correlation between the TAS-20 Total score and the Conners' Continuous Performance Test (CPT) Omission and Commission scores was .03 and -.14, respectively. The correlation between the TAS-20 Total score and the Stroop Color/Word subtest was -.08, as shown in Tables 6 and 7, respectively. Simultaneous regression analyses were performed with the CPT Omission and Commission and Stroop Color/Word scores to predict TAS-20 Total score. Results indicate that these variables do not predict a significant amount of the variability in the dependent measure (TAS-20 Total score).

Table 4

Intercorrelations Among TAS-20 Total and Subscale Scores and CBCL Scores (N=75)

Variable	1	2	3	4	5	6
1. TAS-Total	--	.89*	.88*	.72*	.09	.07
2. TAS 1		--	.72*	.41*	.11	.08
3. TAS 2			--	.47*	.01	.10
4. TAS 3				--	.08	-0.01
5. CBCL 3					--	.51*
6. CBCL 6						--

- TAS-Total = TAS-20 Raw Total; TAS-1 = Difficulty identifying feelings; TAS-2 = Difficulty describing feelings; TAS-3 = Externally oriented thinking; CBCL 3 = Anxious/Depressed; CBCL 6 = Attention problems

* $p < .05$

Table 5

Intercorrelations Among TAS-20 Scores and demographic variables (n=71)

Variable	1	2	3	4	5	6
1. Mother's Age	--	.26*	-.04	-.04	-.13	.08
2. Income		--	-.11	-.11	-.11	-.03
3. TAS-20 Total			--	.86*	.87*	.69*
4. TAS-1				--	.66*	.33*
5. TAS-2					--	.46*
6. TAS-3						--

* $p < .05$

Table 6

Intercorrlations Among TAS-20 Scores and CPT Scores (n=74)

Variable	1	2	3	4	5	6	7
1. TAS-Total	--	.87*	.88*	.72*	-.18	.03	-.14
2. TAS 1		--	.69*	.39*	-.15	-.03	-.09
3. TAS 2			--	.47*	-.17	.06	-.07
4. TAS 3				--	-.15	.07	-.20
5. CPT Index					--	.38**	.06
6. CPT Omision						--	-.47***
7. CPT Commision							--

- TAS-Total = Raw Total; TAS 1 = Difficulty identifying feelings; TAS 2 = Difficulty describing feelings; TAS 3 = Externally oriented thinking; CPT Index = Continuous Performance Test (CPT) Index Scores, CPT Omission = CPT Omission errors; CPT Commission = CPT Commission errors
- * $p < .05$, ** $p < .01$, *** $p < .001$

Table 7

Intercorrelations Among TAS-20 Scores and Stroop Color-Word Scores (n=58)

Variable	1	2	3	4	5
1. TAS-Total	--	.89*	.89*	.77*	-.08
2. TAS 1		--	.71*	.48*	-.09
3. TAS 2			--	.54*	-.08
4. TAS 3				--	-.01
5. Stroop					--

* $p < .05$

Additional analysis

The relationship between the ADHD group and the diagnostic subgroups (those which were included in the Non-ADHD group) and the TAS-20 was examined by independent samples t-tests (see Table 8). Group differences were observed between the ADHD and Adjustment Disorder groups (t (44) = -2.84, p. = .007) and the ADHD and Anxiety Disorders groups(t (41) = -3.42, p. = .001).

Given the significant difference in age between the two primary diagnostic groups, an analysis of covariance of the TAS-20 was conducted. Results indicate that age was not a significant variable ($F(1,72) = .025$, $p = .876$).

Alexithymia and sample characteristics

The developers of the TAS-20 report preliminary cut-off scores for research purposes (Bagby & Taylor, 1997). A person with a TAS-20 score ≤ 51 is considered non-alexithymic, while a person with a TAS-20 score ≥ 61 is considered alexithymic. Descriptive statistics for the TAS-20 scores and diagnostic groups can be seen in Table 9.

In this entire sample, nearly 76% of the caregivers scored in the non-alexithymic range, nearly 12% scored in the mid-range between alexithymic and non-alexithymic, and nearly 12% scored in the alexithymic range. These results show that most caregivers in this clinical sample did not have alexithymia, as measured by the TAS-20. I expected higher levels of alexithymia in this clinical sample, given that the deficits associated with alexithymia should interfere with a caregiver's ability to be emotionally available to their child. While these deficits may negatively impact a

child, clearly others factors are no doubt responsible for the difficulties found in these clinic-referred children.

Table 8

Summary of independent sample t-tests for diagnostic groups and TAS-20 Total

Group	n	Group	n	t-value	df	p-value
ADHD 39.45 (9.68)	38	Mood/Dep 38.5 (11.64)	6	.217	42	.829
ADHD	38	Conduct/Opp 35.2 (9.20)	5	.926	41	.359
ADHD	38	Adj Disorder 51.25 (14.86)	8	-2.84	44	.007**b
ADHD	38	Anxiety 56.20 (14.86)	5	-3.42	41	.001**b
ADHD	38	L.D. 47.83 (13.73)	6	-1.86	42	.069
ADHD	38	Neuro 49.86 (14.46)	7	-2.42	43	.021

- $p < .05$, ** $p < .01$

b = Bonferoni correction for multiple comparisons = .008

Table 9

Classification of TAS-20 scores by diagnostic group (N=75)

Group	ADHD (n=38)	Non-ADHD (n=37)
1. Non-Alexithymic	33	23
2. Mid-range Alexithymic	4	6
3. Alexithymic	1	8

As defined for research purposes by Bagby and Taylor (1997) 1 = TAS-20 scores $\leq$ 51; 2 = TAS-20 scores between 52-60; 3 = TAS-20 scores $\geq$ 61

In the ADHD sample, nearly 88% of the caregivers scored in the non-alexithymic range, 10% scored in the mid-range between alexithymic and non-alexithymic, and nearly 3% scored in the alexithymic range. Whereas, in the Non-ADHD sample, nearly

63% of the caregivers scored in the non-alexithymic range, 13% scored in the mid-range between the two, and 21% scored in the alexithymic range (see Table 10). The means of the ADHD and Non-ADHD TAS-20 Total scores fell within the normative range (Taylor, Bagby, & Parker, 1992).

Only two of the diagnostic subgroups (Anxiety Disorders and Adjustment Disorders) showed caregiver's TAS-20 total mean scores above the non-alexithymic range (see Table 10). Though the sample size for each was small (n=5), a brief discussion is in order. Children with adjustment disorders are struggling in reaction to some identifiable stressor. They no doubt struggle with both depressive, anxiety, and/or conduct problems, but none in such quantity so that full criteria are met for other disorders. Consequently, the stressors with which they are struggling outstrip their capacities to cope effectively. Children with anxiety disorders are also clearly struggling with symptoms that suggest fears, worries, and other internal states that are outstripping their capacities to cope effectively. Although speculative, it may be that children of caregivers who are unable to adequately know their feelings and regulate their affect are more vulnerable to being overwhelmed with fears, worries, and general uneasiness.

Table 10

Descriptive statistics by diagnostic group and TAS-20 total scores (N=75)

Diagnosis	n	M(SD)	95% CI	Min	Max
ADHD	38	39.31 (9.68)	36—42	24	62
Non-ADHD Group					
Mood/Depressive Disorders	6	38.50 (11.64)	26—51	25	59
Conduct/Oppositional Disorders	5	35.20 (9.20)	24—47	26	48
Adjustment Disorders	8	51.25 (14.86)	39—64	32	75
Anxiety Disorders	5	56.20 (14.86)	38—75	36	76
Learning Disabilities	6	47.83 (13.73)	33—62	31	63
Neurological Problems	7	49.86 (14.46)	36-63	33	72

DISCUSSION

The results of the present study failed to support the hypothesized relationship between caregiver alexithymia and a diagnosis of Attention-Deficit/Hyperactivity Disorder (ADHD) in their children. Also, these same features were not significantly correlated with caregivers' behavioral ratings of ADHD symptoms or neuropsychological measures associated with sustained attention and inhibition. Each result will be discussed in turn, followed by a general discussion and recommendations.

The primary hypothesis of this study was that primary caregivers of children with ADHD would evidence greater levels of alexithymia (as measured by the 20-Item Toronto Alexithymia Scale; TAS-20) than would primary caregivers of clinic-referred children who do not have this diagnosis. This hypothesis was not supported and, in fact, the opposite finding was revealed. In other words, primary caregivers of the Non-ADHD group showed significantly greater levels of alexithymia than did those of the ADHD group. Possible reasons for these findings remain unclear, though the most conservative speculation is simply that alexithymia in caregivers is not a risk factor for the development of ADHD in children. However, in this sample, none of the primary caregivers (in either primary group) had alexithymia scores in the alexithymic range. Therefore, despite the hypothesized relationship, this sample does not allow a definitive statement regarding the relationship between alexithymia and ADHD because the primary caregivers were not, on average, alexithymic.

The caregivers' empathic failures that were hypothesized to lead to ADHD might still be an important risk factor, but these empathic failures seem to be

unrelated to the presence of alexithymia in the caregivers. In other words, though alexithymia is one problematic parental variable that leads to trouble with empathy, it is not a necessary characteristic. It may be speculated, however, that an alexithymic parent might be able to be empathic with an infant whose affects are not yet differentiated, though the data in this study would not validate or invalidate this speculation.

In this sample, the data suggest that caregivers of children with ADHD do not report having the deficits associated with alexithymia. Though greater levels of psychopathology generally and psychiatric disorders specifically have been found in parents of children with ADHD (Breen & Barkley, 1988), alexithymia does not appear to be a risk factor, according to these findings. Consequently, other constitutional and environmental risk factors must be present in order for children to develop ADHD.

Alexithymic features in primary caregivers were not significantly correlated with either the "Anxious/Depressed" or "Attention Problems" subscales of the Child Behavior Checklist (CBCL). The hypothesis that alexithymic traits would be more strongly correlated with parent ratings of attention problems versus ratings of anxiety or depression ratings was not supported by the data. This finding is not surprising, given that the primary hypothesis was also not supported, as described above.

Neuropsychological measures of sustained attention and inhibition (scores from the Conners Continuous Performance Test and the Stroop) were not significantly correlated alexithymia scores. Despite the theoretical model presented above regarding the likely relationship between alexithymia and these laboratory-

based measures of these abilities, this hypothesis was not supported. Again, these data suggest that the presence of alexithymia in primary caregivers does not contribute to the development of neuropsychological difficulties as measured in this sample.

Lastly, there was support for the secondary hypothesis that alexithymic features in primary caregivers would not significantly correlate with age and socioeconomic status of the primary caregivers. This finding is consistent with existing literature regarding the more general relationship between the alexithymia construct and demographic variables.

Limitations and Recommendations

The theoretical model guiding this study would have been more directly assessed if it included a direct measure of parent-child attachment. Attachment was not directly measured in this study due to the complexity and training required of interviewers and time constraints. One focus of further research on ADHD should be its relationship with attachment style and its implications for ADHD pathology.

A second limitation of this study was the heterogeneity of diagnoses within the Non-ADHD group of children (see Table 9). While this type of heterogeneity is common in clinical samples, future research would benefit from larger and more specific comparison groups. Furthermore, future research would also benefit by looking at a non-clinical sample as a comparison group. This would allow an ADHD group to be compared to a group of children whose symptoms do not overlap between diagnostic categories.

Lastly, future studies would also greatly benefit from an age-scaled alexithymia measure, one that could be used with younger populations. At present, no such scale has yet been validated (G. J. Taylor, personal communication, sometime Spring 2000). Recent models suggest that affect dysregulation is a fundamental mechanism of all psychiatric disorders (Taylor, Bagby, & Parker, 1997).

More generally, the results of this study parallel a difficult problem plaguing ADHD research. Over the years, complicated models have been developed to explain the symptoms and underlying pathologies associated with ADHD, including Barkley's (1998) model outlined above. Aside from strictly behavioral ratings, we have not been able to put forth a specific test or battery of tests that accurately and reliably quantify ADHD. Despite the theoretical link between the behavioral symptoms of ADHD and neuropsychological measures of sustained attention and inhibition, none has been recommended to accurately diagnose children as having ADHD (Barkley & Edwards, 1998). Barkley and Edwards (1998) write, "[I]t is hard to establish whether an arrow hits the mark when the mark's location is itself uncertain" (p. 296).

The theoretical model relating ADHD with attachment problems is still one that deserves further investigation. In their chapter on treating ADHD as "Attachment Deficit Hyperactivity Disorder," Ladnier and Massanari (2000) report that the two main areas of deficits identified in ADHD children (self-regulation and relating skills) were consistent with the classic symptoms of an attachment-disordered child" (p. 29, italics added). This position is similar to that outlined above. Empirical investigation of this relationship and the further development of theoretical models is,

no doubt, an area that will be important to investigate ADHD and more effectively treat these children.

APPENDIX A

APPENDIX A

TAS-20

Using the scale provided as a guide, indicate how much you agree or disagree with each of the following statements by circling the corresponding number. Please remember that you are rating your own feelings and styles, not your child's. Give only one answer for each statement.

Circle 1 if you STRONGLY DISAGREE
Circle 2 if you MODERATELY DISAGREE
Circle 3 if you NEITHER DISAGREE NOR AGREE
Circle 4 if you MODERATELY AGREE
Circle 5 if you STRONGLY AGREE

	Strongly Disagree	Moderately Disagree	Neither Disagree Nor Agree	Moderately Agree	Strongly Agree
1. I am often confused about what emotion I am feeling.	1	2	3	4	5
2. It is difficult for me to find the right words for my feelings.	1	2	3	4	5
3. I have physical sensations that even doctors don't understand.	1	2	3	4	5
4. I am able to describe my feelings easily.	1	2	3	4	5
5. I prefer to analyze problems rather than just describe them.	1	2	3	4	5
6. When I am upset, I don't know if I am sad, frightened, or angry.	1	2	3	4	5
7. I am often puzzled by sensations in my body.	1	2	3	4	5
8. I prefer to just let things happen rather than to understand why they turned out that way.	1	2	3	4	5
9. I have feelings that I can't quite identify.	1	2	3	4	5
10. Being in touch with emotions is essential.	1	2	3	4	5
11. I find it hard to describe how I feel about people.	1	2	3	4	5
12. People tell me to describe my feelings more.	1	2	3	4	5
13. I don't know what's going on inside me.	1	2	3	4	5

14. I often don't know why I am angry.	1	2	3	4	5
15. I prefer talking to people about their daily activities rather than their feelings.	1	2	3	4	5
16. I prefer to watch "light" entertainment shows rather than psychological dramas.	1	2	3	4	5
17. It is difficult for me to reveal my innermost feelings, even to close friends.	1	2	3	4	5
18. I can feel close to someone, even in moments of silence.	1	2	3	4	5
19. I find examination of my feelings useful in solving personal problems.	1	2	3	4	5
20. Looking for hidden meanings in movies or plays distracts from their enjoyment.	1	2	3	4	5

Child and Adolescent History Questionnaire

Child's age _____ Present school grade level _____

Age of Patient's mother _____ Occupation _____

Age of Patient's father _____ Occupation _____

Parents are: (circle one) SINGLE MARRIED SEPARATED DIVORCED DECEASED

Is there a step-parent in the patient's home (circle one) YES NO

Patient is (circle one) NATURAL ADOPTED FOSTER

Does the patient have siblings? YES NO

If yes, please list gender and ages of siblings _____

_____

Do both parents work? YES NO father: FULL PART mother: FULL PART

Approximate family income:

0-14,999	_____
15,000-34,999	_____
35,000-54,999	_____
55,000-74,999	_____
75,000 or higher	_____

Are there any significant family or marital conflicts? YES NO _____

_____

Is there a family history of alcohol or drug abuse?(explain)_____

PREGNANCY AND BIRTH HISTORY

Age of mother at delivery _____ Maternal health problems during pregnancy? YES NO

Delivery was: VAGINAL CESAREAN

Baby was: FULL TERM PREMATURE (_____ weeks gestation)

Birth weight: _____pounds _____ounces

Was labor prolonged? YES NO

Did the patient go home from the hospital with you? YES NO

If no, how many days until the patient came home _____

Any medical problems after discharge (e.g., jaundice, fever, feeding problems, etc.) YES NO

If yes, explain:_____

SOCIAL BEHAVIOR

Does your child get along well with other children? _____adult? _____

Have friends? _____Keep friends? _____

Have problems with peer pressure (e.g., alcohol or drug use)?_____

_____

MEDICAL HISTORY

List any serious illnesses/injuries/hospitalizations/surgeries:

Date Incident (please explain)

_____ _____

_____ _____

_____ _____

_____ _____

Describe any head injuries (e.g., date, type, loss of consciousness, resulting change in behavior?)

_____

_____

Current medications and reasons (please include dosage):_____

_____

Is there a family history of learning difficulties in any family member? YES NO

Does anyone in the family have a problem similar to your child's reason for referral? YES NO

(describe)_____

EDUCATIONAL HISTORY

Any grades that were skipped or repeated?_____

Teachers report problems in (circle):

Reading	attention/concentration	spelling
Behavior	arithmetic	social adjustment
Writing		

Was/is your child unusually hyperactive? YES NO Inattentive? YES NO

Do teachers report problems that you do not notice? YES NO

My child's intelligence level is likely (circle one)

Below average average high average superior

PRIOR PSYCHOLOGICAL HISTORY

Have you previously had direct contact with any social agency or mental health professional?

(explain incl. dates)_____

CONSENT AGREEMENT

We are conducting a study to examine parent's feelings and styles of coping in order to better understand the difficult process involved in caring for children and adolescents who are having academic, emotional, and/or behavioral problems. We are asking for your participation.

Your participation requires filling out one or two additional questionnaires that take approximately 15 minutes. Your participation is entirely voluntary. Your decision to participate will not change any aspect of your child's assessment or treatment and you can withdraw permission at any time without penalty. Aside from a few extra minutes of your time, there is no risk of injury or costs associated with participating. In addition to the questionnaires you complete, we may be analyzing assessment data from your child's assessment battery. Your child will not be given any tests or questionnaires that are not part of the standard clinical battery.

Your privacy will be protected to the maximum allowable by law. All data will be kept strictly confidential. Participant identification numbers will be assigned to each test form. The only form that contains both the identification number and your name will be this consent form; it will be kept separate from all other data.

Your participation is greatly appreciated. You indicate your voluntary agreement to participate by completing the returning the enclosed questionnaires. If you have any questions regarding this study, you can speak to Greg Lamberty, Ph.D., at the Alpine Clinic (765/446-9394) or James Jones, M.A., (248/650-9141). David E. Wright, Ph.D. (517/355-2180) can be contacted for questions about your role and rights as a subject of research.

_____ _____

Parent/Guardian Signature date Witness date

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