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dissertation entitled

Behavioral Dysregulation in Sons of Male Alcoholics: Parental and Sociodemographic Predictors

presented by

Hazen Paul Ham

has been accepted towards fulfillment of the requirements for

Doctoral degree in Psychology

Hiram E. Fitzgerald
Major professor

Date 8/11/94

BEHAVIORAL DYSREGULATION IN SONS OF MALE ALCOHOLICS: PARENTAL AND SOCIODEMOGRAPHIC PREDICTORS

by

Hazen Paul Ham

A DISSERTATION

Submitted to
Michigan State University
in partial fulfillment of the requirements
for the degree of

DOCTOR OF PHILOSOPHY

Department of Psychology

1994

ABSTRACT

BEHAVIORAL DYSREGULATION IN SONS OF MALE ALCOHOLICS: PARENTAL AND SOCIODEMOGRAPHIC PREDICTORS
by

Hazen Paul Ham

The present study was an extension of earlier work with hyperactive sons of alcoholics (Ham, 1992; Ham, Fitzgerald, & Zucker, 1994) that investigated the incidence and relationship of hyperactivity, attentional deficits, distractibility, and impulsivity (HADI) with conduct problems. Present study findings reiterate previous research findings that sons of male alcoholics (SOMAs) are significantly more behaviorally dysregulated (i.e., reveal higher incidence of HADI and conduct problems) than sons of male non-alcoholics (SOMnAs). Furthermore, when level of risk is more fully examined based on level of paternal antisociality as well as alcoholism, sons of antisocial alcoholics (AALs) are the most behaviorally dysregulated group of at-risk individuals compared to sons of non-antisocial alcoholics (nAALs) and sons of non-substance abusing, non-antisocial controls. This finding suggests that the antisocial alcoholic milieu that these at-risk children are being reared in is propelling them along a seriously dangerous developmental pathway; one that appears to lead to behavioral problems, and possibly antisocial personality disorder (APS) and/or alcoholism later on. Structural equation modelling (SEM) results were consistent with previous hierarchical regression findings from the same dataset (Ham, Fitzgerald, & Zucker, 1994; Jansen, Fitzgerald, Ham, & Zucker, under review; Zucker, Ellis, & Fitzgerald, 1993); indicating that antisociality and problems due to alcohol abuse/dependence, as well as sociodemographic indicators, are significantly involved in offspring regulatory functioning.

ACKNOWLEDGEMENTS

Thanks first and foremost to my mentor, Hiram E. Fitzgerald, for consistently, although not always gently, reminding me that I am a developmentalist and not a behavioral geneticist. Thanks Hi, for your scholarly advice, and for your influence in grooming me to be a research scientist. I look forward to future projects with you.

Thanks to the rest of my committee members, Drs. Robert A. Zucker, Ellen Strommen, and Jackie Lerner, and lastly to John Schulenberg. Bob, thanks for giving me the opportunity to be involved in the MSU-UMLS, and thanks for taking part in the beginnings of my career as research scientist. Your professional research ethics have encouraged me to strive for excellence - I will do so in the years to come. Ellen, thanks for your assistance with my writing, but most importantly for your thoughtful and provocative review of my work throughout the years. Jack, you have been most supportive throughout this endeavor through your scholarly suggestions but more so by your kind, familiar, and sensitive attitude toward certain issues that tend to get overlooked within the arena of academia. And John, thank you for all of your help - methodologically, "politically", and most especially personally. Your support during the past few years has really kept me going sometimes. Because of your friendship and scholarly advice I have a more hopeful outlook regarding science and academia in particular, thanks a million!

I want to honor my father and mother. First, for raising me "in the way I should go", knowing that I won't depart from it, but also for giving of themselves to me - completely, unselfishly, and lovingly. Mom, from the bottom of my heart, thank you for introducing me to books. The many hours of reading silently encouraged me to investigate "how things work", consequently, I do just that for a living - I love you so much. Pop, your *consistent* support of my dreams and aspirations has been like a shepherd to me, guiding me when I felt lost, and speaking to me when you weren't around. I'm half the man you are, but I continue to strive to become twice the man you are - you're my hero, I love you.

Last and most importantly, thanks to my Heavenly Father, without whose grace and mercy I would probably be driving a truck somewhere in Northeastern Oklahoma (delivering cigarettes for a living), and not headed for a bright future as a scholar and a scientist - thank you Father.

TABLE OF CONTENTS

List of Tables

List of Figures

CHAPTER I

Introduction

The present study is an extension of an earlier study (Ham, Fitzgerald & Zucker, 1994) that investigated Hyperactivity, Attentional deficits, Distractibility, and Impulsivity (HADI) and related conduct problems in a group of preschool-age sons of male alcoholics (SOMAs) and a group of demographically comparable sons of male non-alcoholics (SOMnAs). The previous study found the incidence of HADI and conduct disordered behavior to be significantly greater in SOMAs. Results also indicated that maternal antisociality was the most significant predictor of problem behavior in both SOMAs and SOMnAs. Problems related to alcoholism was a significant predictor of impulsivity in SOMAs; this is of significant importance in that impulsivity is implicated in adulthood antisocial personality disorders (Martin et al., in press).

The present study was an attempt to investigate behavioral regulation at a more sophisticated theoretical level utilizing more sophisticated analytic techniques (i.e., LISREL). Structural equation modeling techniques were used to investigate the multiple and interrelated causal relationships of several parental and contextual variables with offspring regulatory behavior; regulatory behavior being composed of HADI symptomatology, several components of neuropsychological functioning, and social functioning (i.e., antisocial behaviors such as those exhibited in Oppositional Defiant Disorder or Conduct Disorder). This allowed for the simultaneous examination of those variables suspected to be involved in increasing the risk load for SOMAs; some of these factors were not accounted for in the previous study. The present study also synthesized

several theoretically based hypotheses concerning behavioral dysregulation in SOMAs as they relate to neurobehavioral functioning. To the extent that the data yielded evidence for this type of behavioral dysregulation in SOMAs, it is suggested to implicate a pattern of dysregulated behavior symptomatic of Central Nervous System (CNS) involvement, most specifically in the anterior cortical areas (Tarter, Alterman, & Edwards, 1985).

The specific model tested is a variation of the neuropsychological model proposed by Tarter, Laird, and Moss (1990) and the biobehavioral dysregulation hypothesis discussed by Begleiter and others (Holden, 1991). For many years Tarter and his colleagues have suggested CNS involvement is etiologically relevant to hyperactivity in association with alcoholism. They have been testing their theory on a group of alcoholics and their offspring that is similar to the present sample, albeit boys in their study are much older. Begleiter has proposed the possibility of an inherited mechanism that gives an individual a propensity towards "generalized" dysregulated or unmonitored behavior. He is quick to point out that this proposed mechanism is not solely dependant on one's genetic make-up but rather, its expression depends heavily on the individual's developmental rearing environment. Tarter's model, Begleiter's theory, and the presently proposed model will be discussed in greater detail in the following sections.

Special Issues to Consider

Before laying the ground work of the present investigation, and before presenting the basic model that was tested, there are several issues to consider regarding the type of behavior dysregulation discussed here. First of all, there is the issue of defining hyperactivity. There is a great deal of debate concerning nomenclature and definitional attributes of the hyperactive syndrome that should be considered in this type of study (see

Hinshaw, 1987; Lilienfeld & Waldmen, 1990). Related to this issue is the never-ending contention among social and behavioral scientists about the relationship between hyperactive symptomatology (i.e., Attention-deficit Hyperactivity Disorder; ADHD) and conduct problems (i.e., ODD and CD). Some contend that the two behavioral regimes are part and parcel of the same syndrome, while others insist that they are separate syndromes appearing with high rates of comorbidity. Both of these issues will be addressed in detail in the upcoming literature review.

Second, studies investigating the etiology of hyperactivity and its impact on future outcome consistently report several highly associated biologically, psychologically, and socially relevant outcomes (i.e., biopsychosocial outcomes) (e.g., August, Stewart, & Holmes, 1983; Cantwell, 1972; Gittelman, Mannuzz, Shenker, & Bonagura, 1985; Goodman & Stevenson, 1989b; Hechtman, Gabrielle, Perlman, & Amsel, 1984). Such outcomes include cognitive deficits (e.g., low IQ, poor academic performance) (Frick et al., 1991), aggressive conduct problems (Hinshaw, 1992), language deficiencies (Hinshaw, 1987), poor motor skills (Ayres, 1972; Lyon & Gadisseux, 1991; Wolff, Gunnoe, & Cohen, 1985), as well as other less often reported problems such as anxiety, depression, somatic complaints, and inadequate social skills (McGee, Williams, & Silva, 1984). Such outcomes are thought to be intrinsically involved with, and possibly a result of, hyperactivity. Outcomes in adulthood include substance abuse, criminality, various forms of physical abuse (spousal, child), social and work-related difficulties, poor academic progress, and economic insufficiency (e.g., Bohman, Cloninger, Sigvardsson, & von Knorring, 1987; Cloninger, Bohman, Sigvardsson, & von Knorring, 1985; Cloninger, Sigvardsson, & Bohman, 1988) . Each of these potential correlates need to be examined

closely in connection to hyperactivity in that they may be antecedent to or consequential to it.

Finally, several methodological flaws that are inherent in many studies of hyperactive children will be addressed. One such flaw is that such studies tend to use clinically drawn samples of hyperactive children (e.g., Campbell, Breaux, Ewing, & Szumowski, 1986; Farrington, Loeber, & van Kammen, 1990) and thus the findings are not easily extrapolated to community children who may or may not be symptomatic for the disorder. Related to this, many studies investigating hyperactivity and/or symptoms pertaining to this diagnostic category of problem behavior do not use normal comparison groups (i.e., individuals asymptomatic for hyperactivity and other related pathology) to contrast their findings with (e.g., Gittelman et al., 1985; Moffit, & Silva, 1988). In fact, the majority of studies use pathological samples for controls (e.g., children diagnosed with anxiety disorders, depression, etc). By using asymptomatic controls this allows for better comparison and thus findings are more suitable for generalizing to similar groups of individuals. Furthermore, most studies of alcohol abuse/dependence investigating the prevalence of childhood hyperactivity in chronic alcoholism are based on retrospective reports and not prospective reports of early onset alcoholism (e.g., Alterman, Tarter, Baughman, Bober, & Fabian, 1985; Cloninger et al., 1988; Farrington et al., 1990; Rutter et al., 1990; West & Prinz, 1987).

Why Study Sons of Male Alcoholics?

One final issue should be addressed: Why study SOMAs? Behavioral scientists investigate SOMAs for several reasons. First, children of alcoholics (COAs) in general, and in particular SOMAs, are at heightened risk (25% to 35%) for becoming alcoholics or

alcohol abusers (Cloninger, Sigvardsson & Bohman, 1988). Thus, SOMAs are ideal subjects for "elucidating vulnerability characteristics" that may serve as etiologic indicators of alcoholism (Tarter, Kabene, Escallier, Laird, & Jacob, 1990). Second, it is widely established that COAs are more likely than non-COAs to come from disruptive homes marked by physical abuse, parental psychopathology, sociodemographic deprivation, and high levels of antisociality (Fitzgerald & Zucker, 1994). The combined effects of such family characteristics may account for a significant proportion of the variance responsible for the poor biopsychosocial outcomes seen in SOMAs.

All of the above mentioned methodological issues were dealt with to a certain extent in the present study. See the Methods section for more detail.

The Theoretical Model

Introduction

Before reviewing the various areas of the literature pertaining to the above issues, the basic model to be tested is briefly described so as to give the reader a mental portrait of the study to keep in mind throughout the literature review.

As previously mentioned, this study utilized structural equation modelling techniques to investigate the multiple, causal relationships that are hypothesized to exist within the dysregulated (i.e., chaotic) environment seen in the alcoholic family. The specific techniques used are described in detail elsewhere (Bolon, 1989; Byrne, 1989; Jorskog & Sorbom, 1989; Loehlin, 1987). Figure 1 shows the basic theoretical "behavioral regulatory" model; it is set up so that causal (parental) constructs precede outcome (child) constructs from left to right respectively. Figure 2 is an alternate model, but it expressly reveals those components proposed to cause dysregulated behavior in sons

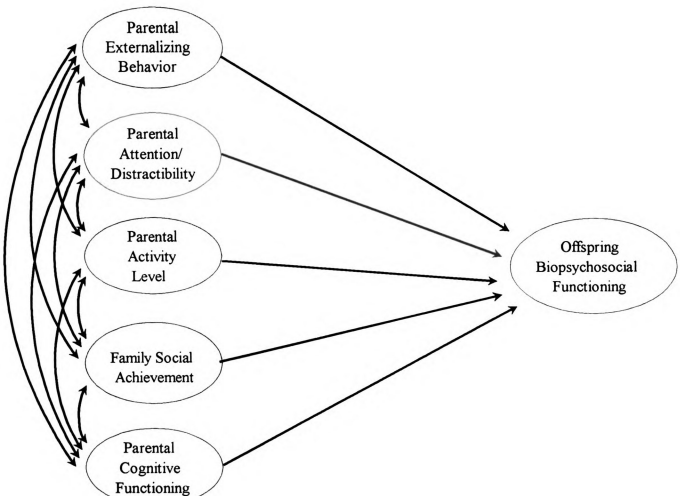

Figure 1. Variables Contributing to Behavioral Regulation: Structural Model 1

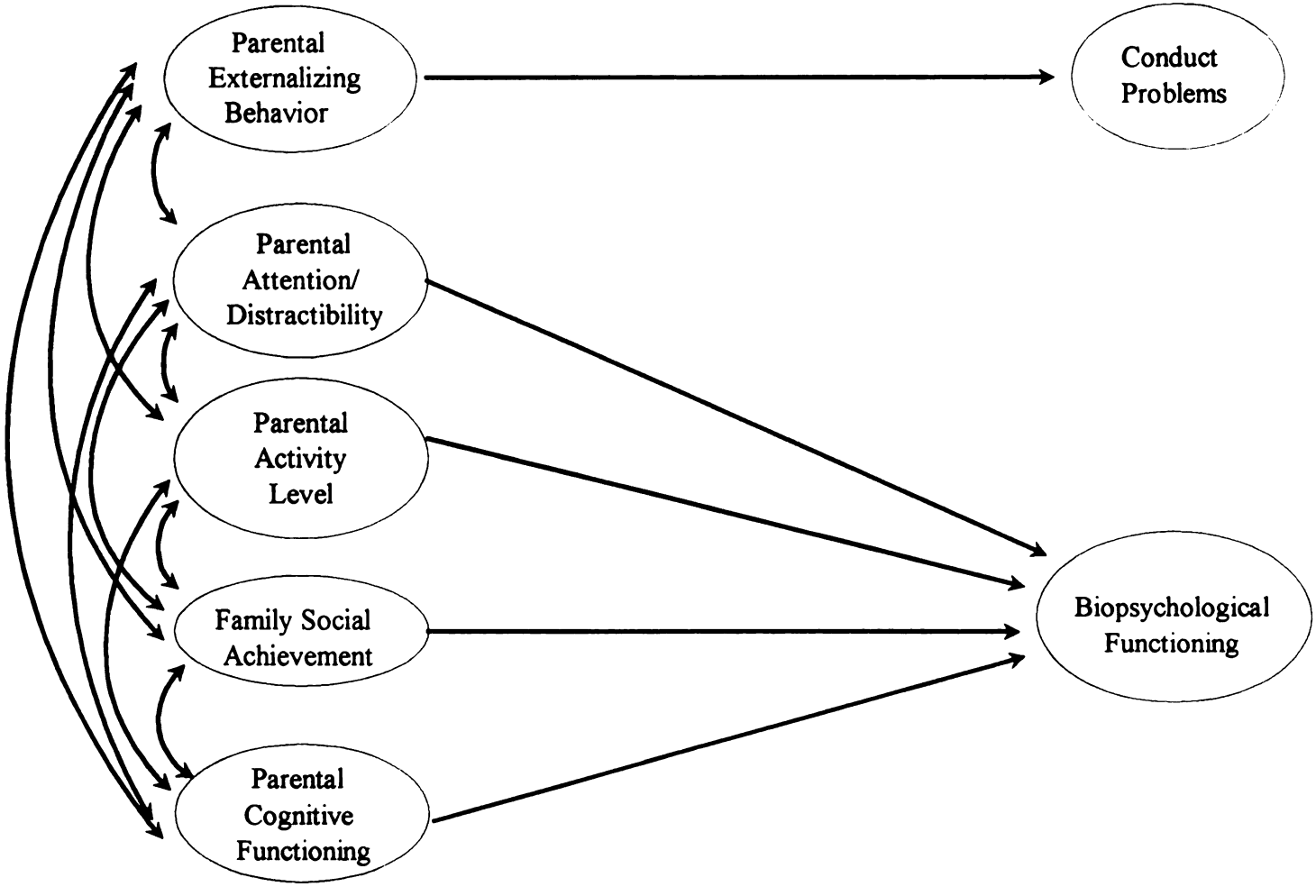

Figure 2. Variables Contributing to Behavioral Regulation: Structural Model 2

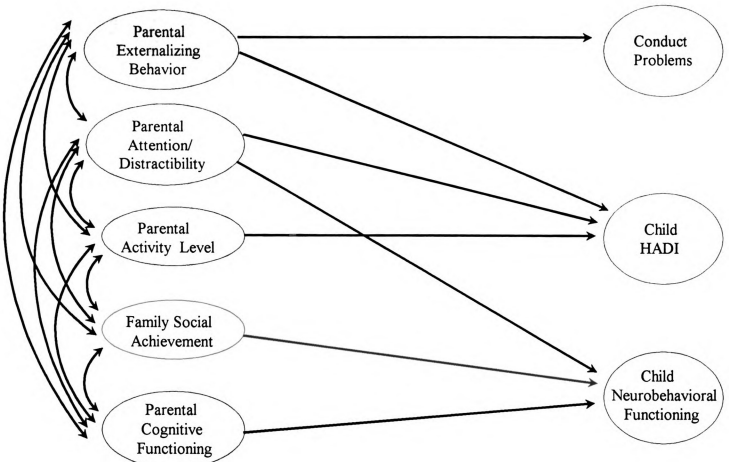

Figure 3. Variables Contributing to Behavioral Regulation: Structural Model 3

of male alcoholics. The hypothesized causal agents for these child outcomes are indicated by one headed arrows leading to them from the parental, contextual variables; curved arrows on both sides of the model indicate relationships (i.e., correlation) among variables. Figure 3 shows the final alternate model. In this model the underlying assumption is that there are aggressive and non-aggressive components of (dys)regulatory behavior. These models are discussed more fully in several upcoming sections.

A Brief Analysis of the Theoretical Model

The relevant literatures suggest that parental alcoholism *in conjunction* with high levels of antisociality has a direct and severe impact on behavioral regulation (HADI), cognitive functioning, and aggressive, non-compliant conduct problems). Figure 1 portrays the impact that the antisocial, alcoholic environment has on overall behavioral regulation. Also, retrospective and concurrent reports of hyperactivity in parents of hyperactive children are highly correlated with their child's present level of hyperactivity (August & Stewart, 1983), thus revealing an heritable component to hyperactivity (this has also been incorporated into the model; it is termed "Parental Hk"). Furthermore, hyperactivity is considered a reflection of cerebral integrity (Tarter et al., 1985), and has been shown to have an heritable component in connection with risk for alcoholism, thus the arrow from parental alcoholism to Child HADI. However, the rearing environment (i.e., the child's experiential world; Gottlieb, 1991; Turkheimer & Gottsman, 1991) also plays an integral role in the expression of hyperactive behaviors in conjunction with other aspects of behavioral functioning. For instance, a suboptimal rearing environment may facilitate or exacerbate a child's propensity toward dysregulated behavior and poor socioemotional/cognitive development (Fitzgerald, Zucker, Davies, & Klinger, 1993); its

deficient nature serves to lower cognitive stimulation, which has subsequently been shown to have adverse neuroanatomical consequences (i.e., decreased neuronal, dendritic growth, etc.), which ultimately leads to adverse effects on cognitive development and functioning. Figure 3 portrays the relationship between child hyperactivity and neurobehavioral functioning and the impact the rearing environment (parental hyperactivity, sociocultural influences, parental cognitive functioning) has on them separately from conduct problems. In Figure 2 this relationship is portrayed in a collective fashion as HADI, neurobehavioral functioning, and conduct problems have been collapsed into a global measure of biopsychological functioning. Figure 4 shows how each of the offspring behavioral variables were theoretically constructed.

Summary

The general model stipulates that parental alcoholism, antisociality, cognitive functioning, hyperactive symptomatology, and socioeconomic achievement collectively impinge upon child behavioral regulation. The model specifies that offspring HADI and neuropsychological status comprise an element of biobehavioral regulation (i.e., HADI being a reflection of the behavioral component and neuropsychological status being a reflection of biological functioning), and that conduct problems complete the picture of dysregulatory functioning; conduct problems pertaining to one's social interaction (i.e., conduct problems are more societally recognizable compared to hyperactivity). These aspects of the model should be kept in mind during the next few sections. This brief review of the model should help guide the reader through the following discussion as I synthesize the various literatures pertaining to the functional components of biobehavioral dysregulation in SOMAs. The model is discussed in greater detail, both technically and

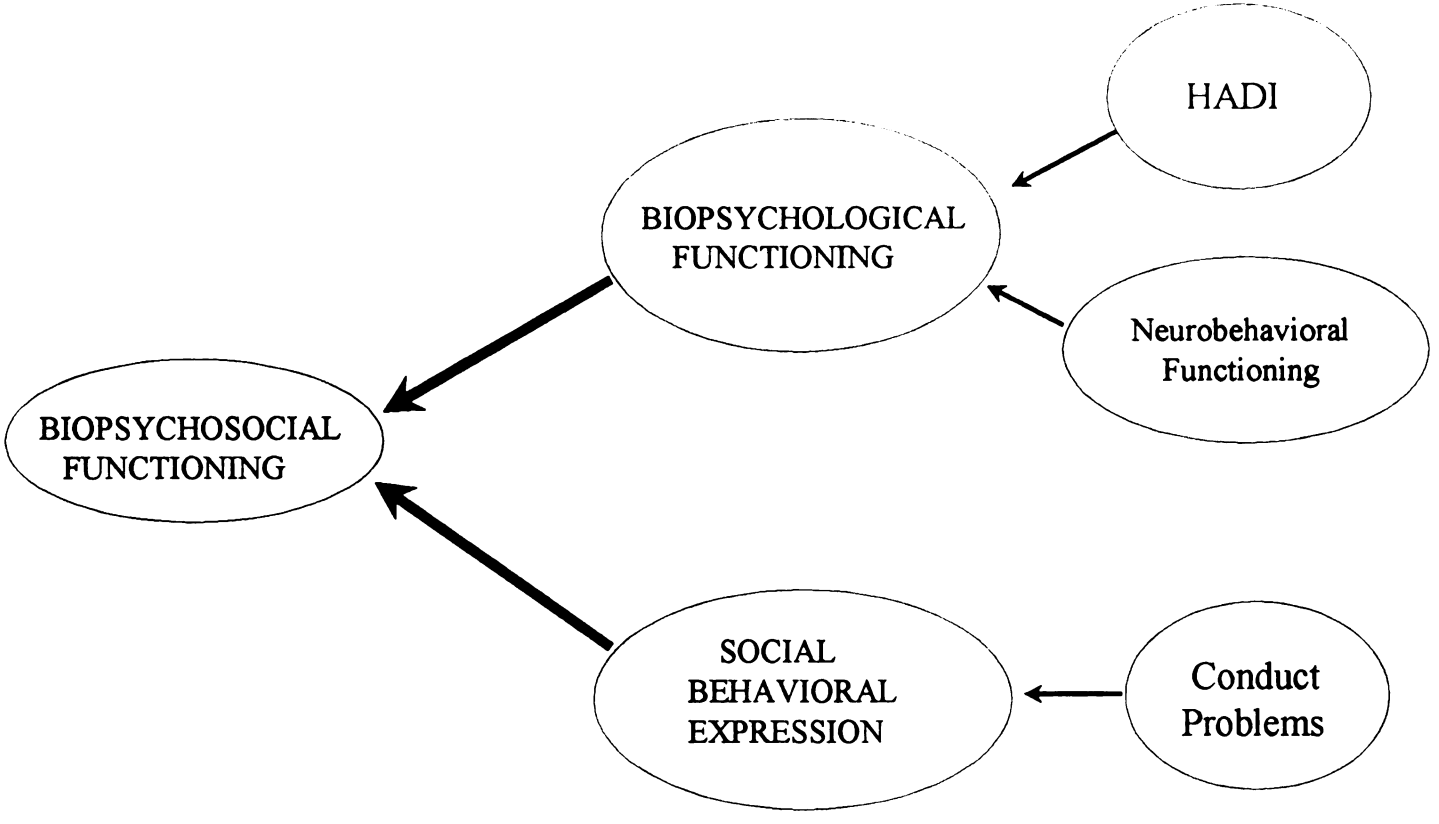

Figure 4. The Higher Order Structural Model of Behavioral Regulation

substantively, in the Methods section.

The present study was a prospective, cross-sectional, high risk study investigating the expression of HADI, conduct problems, and neurobehavioral functioning in two groups of demographically comparable SOMAs and SOMnAs during the critical developmental period of late infancy and early childhood. The focus of the study was on the above mentioned child related outcomes, as well as several parental and contextual influences on HADI and its related behaviors. This study also systematically addressed several of the above mentioned methodological issues. Subjects included a group of preschool age SOMAs and their parents, and a group of demographically comparable preschool age SOMnAs and their parents. The alcoholic and comparison groups used were community rather than clinically drawn samples (see Methods). Unique also to this study is the fact that alcoholics are much younger than other samples used in other alcoholic studies (i.e., they are not yet in formal treatment). Since the alcoholics and their sons in the study are relatively young this enables us to observe the developmental course of HADI and related pathology, and investigate their interaction with the alcoholic environment.

Plan of the Literature Review

The next few sections will review the basic findings from the pertinent literatures that deal with the above mentioned issues. First, I will describe HADI within the clinical context of ADHD, and I will review some of the diagnostic problems that plague this area of research. Second, findings from research on ADHD related behavioral and cognitive outcomes will also be reviewed. Third, the issues of cognitive functioning and behavioral expression and how they relate to neurobehavioral functioning in hyperactive children will

be synthesized together to paint an overall picture of "biopsychosocial" dysregulation. Finally, hyperactive symptomatology will be examined within the alcoholic family environment. In this last section the child behavior and alcoholism literatures will be merged into the specific neurobehaviorally based theoretical model to be tested.

Hyperactivity, Attention Deficits, Distractibility, and Impulsivity (HADI)

The Hyperactive Syndrome: What is it?

Hyperactivity is one of the most common behavioral problems of children referred to mental health professionals. It has been linked with many poor behavioral outcomes and is considered a precursor of such outcomes, thus it can be a formidable risk factor in certain individuals. Estimates of prevalence in school aged children vary greatly, the range being anywhere from 1% to 20% (Barkley, 1981; Safer & Allen, 1976), however, the agreed upon prevalence rate by most researchers and clinicians is around 3% in the general population (American Psychiatric Association, 1987; Hinshaw, 1992). Boys are generally at greater risk than girls for the development and maintenance of hyperactivity. Ratio rates range from 3:1 to 6:1, indicating that three-to six-times as many boys as girls display this kind of dysregulated behavior. Hyperactivity has also been documented at much higher levels in high risk populations (e.g., alcoholics) (Cantwell, 1972; Morrison & Stewart, 1983; Tarter et al., 1985).

One reason for variation in prevalence rate of the disorder is the inconsistency in criteria used for diagnosis, as well as the wide variety of labels given to the disorder. Historically, hyperactivity has been referred to as "Minimal Brain Dysfunction", "Hyperactive Child Syndrome", "Attention Deficit Disorder", "Attention-deficit Disorder with or without Hyperactivity", "Hyperkinesis", "Pervasive and Situational Hyperactivity",

"Hyperkinetic Reaction of Childhood", among others. Windle and Searles (1990) point out that the nomenclature for hyperactivity has undergone, and continues to undergo, many changes, while differentiations of the disorder continue to be classified and there is a continuous breakdown of more reliably measured subtypes. The primary features of the disorder, however, have been consistent throughout these periods of revision and are, for the most part, reflected in the research. For instance, as will be pointed out, most clinicians and researchers believe that hyperactivity includes attentional deficits, distractibility, impulsivity, and excessive motor activity exclusive of aggressive conduct problems.

Recent Attempts at Delineating Hyperactivity

In the 1987 revision of the Diagnostic and Statistical Manual (DSM-III-R; APA, 1987) hyperactivity is defined as Attention-deficit Hyperactivity Disorder (ADHD). The Manual describes the general features of the disorder as: "developmentally inappropriate degrees of inattention, impulsiveness, and hyperactivity", with excessive gross motor activity being the most prominent characteristic of the disorder in preschoolers (APA, 1987 p. 50-52). The Manual further defines excessive motor behavior as fidgeting, constant manipulating of objects, difficulty remaining seated, excessive jumping about, inability to await turn, difficulty playing quietly etc. Other identifying behavioral characteristics of ADHD are high levels of distractibility, low frustration tolerance, poor emotional control and lability, hyperexcitability, aggressiveness, antisocial behavior, and poor academic progress; a majority of hyperactive children repeat at least one grade (Baxley & LeBlanc, 1976; Horn & Ialongo, 1988).

The DSM-IV, the latest revision (APA, 1994), proposes minor revisions

concerning the expression of the primary symptomatology of the disorder. The disorder has been renamed from Attention-deficit Hyperactivity Disorder (ADHD) to Attention-deficit/Hyperactivity Disorder (AD/HD). No dramatic difference can be seen in the name or the acronym of the disorder, but there is a noticeable difference in categorization and diagnosis. The manual delineates the disorder into its two basic features, 1) attention deficit and 2) hyperactivity and impulsive behavior. A child can make a diagnosis if he or she meets the criteria for attention deficits (AD) **and/or** hyperactivity and impulsivity (HD)[1]. Thus, even though the defining characteristics of the disorder have once again undergone a change, DSM-IV will apparently retain the original characteristic behaviors of the hyperactive disorder, namely, 1) attention deficit (containing a distractibility component), 2) hyperactivity (developmentally inappropriate motoric behavior), and 3) impulsivity (behavior that connotes rash, situationally inappropriate decision making).

For the remainder of this paper behaviors pertaining to ADHD will be referred to generically either HADI or "hyperactivity". In this study no disorder is being dealt with, nor are any diagnoses being made. Rather, an amalgamation of behaviors similar to those that comprise the hyperactive disorder (i.e., ADHD) is being investigated. These behaviors consist of hyperactivity, attentional deficits, distractibility, and impulsivity, thus HADI. The term hyperactivity used throughout this paper does not refer to simple physical overactivity, but also includes attentional difficulties, distractibility, and impulsive behaviors as well. On the other hand, simple physical hyperactivity will be referred to as physical overactivity.

ADHD and Conduct Problems

At this point in the discussion, I want to briefly review a controversial and continuing debate regarding the diagnostic considerations of the hyperactive syndrome. The issue is how to distinguish between ADHD and ADHD comorbid with aggressive, conduct problem behaviors. Conduct Disorder (CD) is the most prominent behavioral disorder seen in the ADHD child (see Hinshaw, 1987, and Lilienfeld & Waldman, 1990). Many suggest that ADHD is precursorily related to CD but that the two are essentially distinctive disorders (Hinshaw, 1992). However, separation of the two is extremely difficult if not impossible. Others argue that the two disorders are mutually inclusive and thus inseparable. Many researchers agree with the notion that ADHD and CD may in fact be separate disorders, but that they coexist in some cases; despite the overlap in definitional ideology, the consensus is that these two types of externalizing behaviors, co-existent or not, "show at least partial independence and some degree of divergent validity" (Hinshaw, 1992, pp. 128).

Another behavior disorder often seen in the same milieu as ADHD is Oppositional Defiant Disorder (ODD; APA, 1987, 1994). The characteristic behaviors of ODD not only overlap with ADHD but some are embedded within the CD milieu (Loeber, Lahey & Thomas, 1991). Oddly enough, however, ODD is rarely mentioned in conjunction with ADHD when in fact it makes good diagnostic and definitional sense to rule out ODD before considering CD (especially in very young children, and more especially in conjunction with hyperactive children). Figure 5 portrays the prevalence and overlap of the three disorders, according to DSM guidelines (APA, 1987, 1994) whose behavioral characteristics were investigated in the present study.

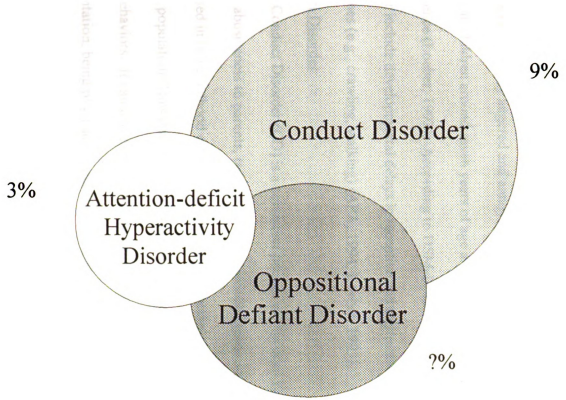

Figure 5. The Prevalence and Theorized Overlap of Disruptive Behavior Disorders

Note: DSM-III-R prevalence rates are in percentages beside each disorder

Oppositional Defiant Disorder

Oppositional Defiant Disorder (ODD) is a behavioral pattern that many times co-exists with ADHD and therefore its diagnosis is often times overlooked (Hinshaw, 1992). ODD reveals itself as very negativistic, hostile, and defiant behavior, however, it is exclusive of the physical violence seen in CD (i.e., without the encroachment of other's basic rights). Oppositional children have frequent temper outbursts, swear at their parents or teachers, are easily angered and annoyed, are argumentative, etc. The disorder usually surfaces in children around seven years of age and many times evolves into CD in adolescence (Loeber, 1993). According to DSM-IV criteria, associated features of the disorder include developmental delays in receptive and expressive language and locomotor milestones (e.g., crawling, walking) (APA, 1994; Loeber, 1993).

Conduct Disorder

Conduct Disorder (CD) is a consistent pattern of violatory behaviors revealing a general abusiveness to parents, teachers, peers, and often times strangers. It is usually diagnosed in late childhood or early adolescence and occurs in about 9% of boys in the general population (Hinshaw, 1992). CD can also be classified according to covert and overt behaviors. It can express itself in overt behaviors such as stealing with confrontation, being physically cruel to animals and/or people, and using weapons in a fight; or it can reveal itself in covert behaviors such as engaging in fire setting, breaking into someone else's car or house, deliberately and covertly destroying another's property, etc. Fewer behaviors are required to make the diagnosis of CD than ODD because of its more violent nature. The DSM-IV (APA, 1994) also makes the distinction of early versus late appearing CD (i.e., late being after the age of 16). Those individuals expressing the

disorder earlier in life (i.e., childhood) show much poorer outcome than those who exhibit the disorder later on in adolescence.

Empirical Attempts at Delineating the Comorbidity

The controversial issue of overlap of hyperactivity and conduct problems is still an ongoing one in child research. Attempts continue to be made at deciphering the differences and similarities of the disorders. The following section briefly describes results from some of the recent attempts at empirically describing the relationship of the disorders.

Some study results suggest that ADHD symptomatology does overlap with ODD and/or CD symptomatology, and that the three disorders may be part of one general behavioral dysregulatory disorder (e.g., Ham et al., 1994; Phil & Peterson, 1991). Other results suggest that the three maintain unique identities and should remain in distinct categories (see Lilienfeld & Waldmen, 1990). Most empirical studies, however, report moderate to high levels of overlap of hyperactivity and conduct problems, the range being anywhere from 30% to 90% (Hinshaw, 1987). Others concurrently report high degrees of correlation between them (0.6 to 0.7) using parent and teacher ratings (Farrington, Loeber, & van Kammen, 1990).

However, as mentioned previously, others have pointed out (Ham et al., 1994), that much of the apparent overlap of ADHD and CD may be due to poor theoretical planning. For instance, some factor analytic studies include items common to both hyperactive and conduct problem factors (Hinshaw, 1987; Lilienfeld & Waldman, 1990). For example, the Conners Conduct Disorder factor (Conners, 1990) includes items such as "restless or overactive", "fails to finish things s/he starts-short attention span", "excitable,

impulsive" - items that are specifically indicative of ADHD behaviors (APA, 1987, 1994) and not CD behaviors. The converse is often seen in the assembly of hyperactive factors - aggressive, antisocial behaviors are many times also included. However, in some cases factor analytic results may not be solely artifactual, as suggested here, but may actually reflect constructs that correlate highly because they appear together in children at similar rates and thus may be revealing one disorder, not several overlapping disorders.

Some findings strongly suggest that the two disorders are separate and distinct behavioral disorders simply because they have separate and distinct etiologies and outcomes (August et al., 1983; Cantwell, 1972; Taylor et al., 1986). For instance, Farrington et al. (1990) found several distinctions between ADHD and CD that may aid us in the diagnostic controversy surrounding their comorbidities. Using 411 boys (ages 8-14 years at the first testing session, and then 16-21 years at follow up) from the Cambridge Study in Delinquent Development, the authors investigated the etiologies and outcomes of ADHD and CD. Four groups were initially formed: one group with ADHD only; one group with CD only; one group comorbid with ADHD and CD; and the fourth group presented neither disorder. The primary findings were as follows: 1) there was considerable overlap of ADHD and CD (60%); 2) both ADHD and CD predicted juvenile offense, however, ADHD was only predictive of juvenile trouble when comorbid with CD and further, only when in the presence of antisocial "criminal" parents; 3) ADHD was related to antisocial parents, low cognitive level, and large family size, while CD was related mostly to poor parenting; and 4) with regard to antisocial outcomes, ADHD predicted juvenile offenses, while CD predicted adult convictions and recidivism.

When these data are carefully examined they reveal that ADHD and CD, while

often comorbid, maintain separate etiologies, predictive characteristics, and behavioral outcomes. The different correlates of ADHD and CD found in the Farrington et al. study can serve as guidelines for distinguishing the distinctive relationships of ADHD and CD, to the end of aiding in prevention, intervention, and treatment. It remains evident that CD and/or aggressive behaviors are most indicative of a developmental pathway of deviancy and antisociality (Loeber, 1990), while the behavioral and psychological outcomes of hyperactive children generally appear more hopeful. However, Farrington et al. also report that the hyperactive children in their study exhibited more difficulties related to higher brain functioning abilities (attentional, organizational, memory, and language) than the conduct disordered children.

In a pre-investigatory look at the data set to be used in the present study, an attempt was made to uncover the "separateness" or "sameness" of ADHD, ODD, and CD type behaviors with confirmatory factor analysis (CFA) via linear structural equation modelling techniques (LISREL). The Child Behavior Checklist (Achenbach, 1978) and the Conners Parent Questionnaires (Conners, 1990) were used in order to provide convergent validity to the model. Latent variables were constructed for the three disorders via DSM-III-R and DSM-IV defining characteristics. Subjects were children in the present study (n = 241), and ratings were used from both parents. Results from the CFA strongly suggest that the three behavioral categories maintain separate and distinct constructs. Goodness of fit (GFI) indices revealed satisfactory fits (average GFI = .90) (Jorskog & Sorbom, 1989) for the model regardless of instrument used; all of the other indices of fit concurred, indicating that the model fits well and the three behavioral categories have unique qualities. However, factor correlations of the three disorders were

between .78 and .92 suggesting that ADHD, ODD, and CD kinds of behaviors do share qualities in common. When the three categories of behavior problems were tested separately (i.e., each construct was run separately to test for reliability) fit indices were very high (range .94 - .98), suggesting excellent reliability of the latent constructs. Further psychometric testing of these constructs was conducted, the results are described in the Method section.

The final word about the extent to which these behavioral problems are distinctive or similar, however, does not depend solely on the data but rather, depends primarily on the individual and collective etiologies, precursors and correlates, different behavioral outcomes, and differences in treatment response [2] (Farrington et al., 1990).

Keeping these things in mind, it is appropriate to note that many researchers (see Hinshaw, 1987 and Lilienfeld & Waldman, 1990) suggest that although ADHD and CD may be separate disorders, ADHD may be precursorily related to conduct problems (both ODD and CD), which in turn are precursorily related to antisocial personality disorder (APS) (Loeber et al., 1991). If this assumption is correct it would simply constitute a continuum of behavior dysregulation, one that surfaces in infancy and early childhood as ADHD and/or ODD and continues to gain severity of expression during late childhood and adolescence. During childhood or adolescence the disorder may reveal itself as CD, and in adulthood it may express itself as APS and/or alcoholism.

However, this theorized spectrum of dysregulatory behavior is not expressly seen in all children. Most hyperactive children eventually stop displaying hyperactive, impulsive, and inattentive behaviors and progress on a normal developmental pathway. On the other hand, there is a specific subtype of hyperactive children with certain, as of yet

unsatisfactorily disclosed, antecedent factors that propel them along this unfortunate, undesirable developmental pathway of adolescent and adulthood antisociality.

To illustrate this point the reader is referred to the August et al. study (1983) previously mentioned. The authors conducted a 4 year follow-up of two groups of hyperactive youths, one with associated conduct problems, the other without. The first group consisted of what the authors referred to as "pure" hyperactive youths (n = 22; mean age at intake 10.7 yrs), meaning they exhibited no aggressive conduct problems but did exhibit the traditionally accepted behaviors indicative of ADHD. The second group consisted of "hyperactive - undersocialized aggressive" youths (n = 30; mean age 9.6 yrs); these boys were comorbid for aggression and hyperactivity by current diagnostic categorizations; aggression in this study contained behaviors involved in such behavioral disorders as ODD or CD. On average both groups of boys were clinically hyperactive (i.e., physical overactivity) at follow-up, suggesting that physical hyperactivity is a relatively stable characteristic in hyperactive boys regardless of associated symptomatology. However, the percentage of boys with physical hyperactivity in the "pure" hyperactive group was less than that in the aggressive hyperactive group. It seems that much of the childhood physical activity dissipated in "pure" hyperactives but remained relatively constant in aggressive hyperactives. Boys originally diagnosed as "pure" hyperactive exhibited primarily inattentive and impulsive behaviors at follow-up. The group of "aggressive" hyperactive boys (i.e., hyperactive symptoms co-morbid with conduct problems) continued to exhibit hyperactivity, inattention, and impulsivity to a significant extent, but were also significantly more physically aggressive, non-compliant, antisocial, and prone to alcohol use by the age of 14 years than were those in the "pure"

hyperactive group. In fact 30% of the aggressive hyperactives were abusing alcohol and drugs at follow-up compared to 0% of the "pure" hyperactives. Finally, an important aspect of these findings suggests that "pure" hyperactivity, is not prognostically related to conduct problems because none of the "pure" hyperactive boys were rated as clinically conduct disordered at either intake or follow-up.

Following from this study, August et al. have formulated a hypothesis concerning the overlap of hyperactive symptoms and aggressive conduct problems regarding child and adolescent outcome that is reminiscent of Earls et al's (1988) hypothesis concerning the relationship of alcoholism and antisociality and negative adult outcome. Earls et al. contend that it is neither alcoholism nor antisocial behavior alone that leads to extreme adverse outcome in the adult but rather, it is the coupling of the two disorders that leads to poor behavioral and social outcome. In other words, there is an aggregating effect produced by combining antisociality and alcoholism that exacerbates an individual's dysfunctional behavior. Similarly, August et al. suggest that it is not hyperactivity or conduct problems per se that predisposes one to various psychopathologies and adverse outcomes but rather, it is the *combination* of the two, particularly aggressive, antisocial behaviors, that most significantly impacts behavioral consequence. Loeber (1990) suggests that hyperactivity appearing together with conduct problems sets a child on a more hazardous developmental pathway resulting in more societally offensive behavior, and thus much poorer outcome in general compared to the child who is only hyperactive.

Summary

ADHD, ODD, and CD are three behavioral disorders whose primary symptomatology appears to be linked together in several ways: First, they are often found

with similar overlap and they are often found in the same children. Second, regarding ADHD, some definitions insist that the disorder contains an aggressive, antisocial, non-compliant behavioral component (Pihl & Peterson, 1991). Other definitions suggest that the disorders are mutually inclusive making separation of the two impossible. However, several behavioral correlates and outcomes for the disorders provide us with differentiating aspects of the two disorders which ultimately suggests that they have independent qualities. For example, childhood aggression and CD have been strongly linked with adolescent and adulthood antisociality (August et al., 1983), whereas childhood ADHD is more predictive of academic underachievment (Farrington et al., 1990; Frick et al., 1991), poor cognitive functioning (Goodman & Stevenson, 1989b; Schachar et al., 1981; Tarter et al., 1985), and soft neurological signs (Hynde, Voeller, Hern, & Marshall, 1991; Wolff, Gunnoe, & Cohen, 1985).

In keeping with other research (e.g., August et al., 1983), the current study is attempting to give precision to the definition of ADHD kinds of behavior by asserting that its component behavioral entities (hyperactivity, attentional deficits, distraction, and impulsivity - HADI) constitute a "pure" form of hyperactivity. Important to remember, this definition suggests that HADI behaviors are specifically exclusive of violatory, antisocial, and aggressive behaviors. Notwithstanding that the latter conduct disordered behaviors co-exist with hyperactive symptoms in some cases, they are here treated as separate dysfunctional behaviors. This is done because, as previously mentioned, many researchers note that children exhibiting hyperactivity solely have more favorable social and behavioral outcomes compared with children who exhibit hyperactivity with aggressive, conduct disturbances; the latter conduct problems are associated with

delinquent and antisocial behavioral outcomes, when expressed in hyperactive individuals (Campbell 1987; Earls et al., 1988).

Temperament and Hyperactivity

Introduction

The specific features of hyperactivity exemplify several aspects of behavior that are referred to by some as temperament (Thomas & Chess, 1984). Temperament refers to specific features of behavior that distinguish individuals from one another based upon their unique quality and intensity. Allport (1961, cited in Buss & Plomin, 1984) refers to temperament as "characteristic phenomena of an individual's nature...dependant on constitutional makeup, and therefore largely inherited in origin" (p. 34). Temperament has also been defined as constituting several separate, yet interactive, domains of behavior. These domains are consistently mentioned in conjunction with one another - activity level, attention span, ease of distraction, ability to adapt to new or novel situations, level of inhibition to people and circumstances, diurnal rhythmicity of a variety of behaviors such as elimination and eating, and intensity of stimulus response. Depending upon whose definition of temperament one uses the number of traits will vary, but nearly all of these attributes will be present in its defining characteristics.

Thomas and Chess (1984) characterize temperament as: 1) the physical speed with which one executes an act; 2) the manner in which one approaches a task, whether it be in a new social context or a new physical environment and; 3) the ease with which one is distracted from the present task. Thus, temperament applies to a broad spectrum of the child's everyday activities including responsibilities in the home and at school, obeying directives from parents and teachers, and following acceptable social norms and mores.

According to past and present research of hyperactive children, there are certain deviations within the aforementioned behavioral situations (i.e., everyday activities, responsibilities at home and school etc.) which, if expressed at certain ages and for certain periods of time, are indicative of behavior found in a child with ADHD. In the hyperactive child, these deviations of behavior may be a facet of what Thomas and Chess (1977) refer to as "difficult" temperament.

Temperament and the Developmental Rearing Environment

It has been suggested that certain children with specific difficult temperament traits are more predisposed to the pathology of their parents when these traits are exposed to recurring environmental situations representative of the parents pathology (e.g., Tarter et al., 1985, 1990); such situations might include an emotionally or physically chaotic home environment, parental substance abuse, or high levels of parental antisociality (Fitzgerald et al., 1990; Thomas & Chess, 1984). Additionally, when several difficult temperament traits are present in the child's behavioral repertoire (i.e., several difficult traits compounded), it may indicate that the child is on a hazardous pathway of behavioral problems which may ultimately lead to substance abuse and related psychopathology (Windle, 1991).

There is empirical evidence that a difficult temperament style in childhood is associated with substance abuse in adulthood (Lerner & Vicary, 1984). A good example can be seen in a recent study by Windle (1991). Windle examined 189 female and 122 male white adolescent high school students (average age of the students was 15.7 years). The study goals were to look at the association of number of difficult temperament traits (e.g., distractibility, hyperactivity, irritability etc.) and amount of substances used (e.g.,

alcohol, cigarettes, marijuana) in the last thirty days. Results indicate that as the number of difficult temperament traits increased so did the frequency of substances abused. For example, 97% of the students with 5 or more difficult temperament traits used alcohol, compared to only 58% of those with no difficult temperament traits. The same trend could be seen for tobacco use - 69% with 5 or more difficult traits, compared to 18% with 0 traits. For marijuana it was 24% with 5 or more traits and only 7% with 0 traits. One of the most dramatic increases was seen in those using hard drugs - 35% with 5 or more difficult temperament traits versus 5% with no difficult traits.

Ellis (1993) found coincident findings in a study of pre-school age sons of antisocial male alcoholics. She found a high and positive relationship between risk for difficult temperament and externalizing behavioral problems in boys in her study. Her suggestion is that although the risk for difficult temperament seems to be more evident in the children growing up in an antisocial alcoholic family, the difficult temperament "style" is probably indicative of negative behavioral outcomes in all children. She further suggests that a child at risk for alcoholism who displays a difficult temperament profile is already at heightened risk for poor behavioral outcome due to the genotypic underpinnings concealed within the dense family history of alcoholism.

These data suggest that a difficult temperament style serves as a significant risk factor for substance abuse and poor socioemotional/behavioral outcome. The Windle study also found a high degree of association between difficult temperament characteristics and externalizing behaviors (e.g., hyperactivity and conduct disordered behavior), further suggesting that difficult temperament involves the expression of maladaptive behavior. The Ellis study provides evidence that difficult temperament is

already apparent in the early pre-school years and may be indicative of risk for poor biobehavioral outcomes.

Jansen, Fitzgerald, Ham, and Zucker (in press) recently investigated the temperament profiles of clinically problem behaviored children being reared in high risk alcoholic, low socioeconomic environments. Actually, the majority of boys in this study are being reared by antisocial, alcoholic parents, whose mean family income was near or below poverty levels. Boys were classified as clinically problem behaviored if they surpassed cutoff scores for Total Problem Behaviors on the Child Behavior Checklist (Achenbach & Edelbrock, 1983). The primary findings of the study were coincident with those in the Windle (1991) and Ellis (1993) studies. Specifically, boys exhibiting clinical levels of problem behaviors coincidently displayed a difficult temperament profile whereas non-clinically problem behaviored boys did not. The authors also report that the number of difficult temperament traits was highly correlated with the level of problem behaviors. Parents of the problem behaviored boys revealed significantly higher levels of alcohol problems and antisocial behavior, and they also reported lower levels of family income, socioeconomic status, and education. These results support the notion that a difficult temperament profile thrives within the confines of an impoverished, antisocial alcoholic family environment.

Tarter et al. (1990) suggest that "certain childhood temperament characteristics may be (specifically) associated with the risk for alcoholism." Through the use of neuropsychological tests Tarter and his associates have been able to distinguish SOMAs from SOMnAs. They have found that sons of alcoholics perform more poorly on cognitive tests (e.g., attention capacity, abstract problem solving, and perceptual motor

tasks) as well as showing higher levels of behavioral tempo (i.e., hyperactivity)(Tarter, Jacob & Bremer, 1989). Tarter suggests that these results are linked to difficult temperament as well as dysfunction of the anterior cerebral cortex. Such speculation is a favorable proponent for a genetic etiology of alcoholism in conjunction with hyperactive behavior. However, even though the observance of these behavioral manifestations does support a genetic predisposition to certain temperament characteristics, such manifestations neither "confirm nor disconfirm a genetic hypothesis" (Tarter et al., 1989). Notwithstanding, these observations do lead us to believe that a difficult temperament profile may predispose a child to certain deficiencies or pathologies. And furthermore, when the child is exposed to parental pathology and a chaotic rearing environment, this further increases the child's risk for adverse outcome. This line of thinking is expressly used in the present study. Several negative aspects of temperament and cognitive functioning will be examined in SOMAs. If more difficult temperament traits (e.g., low attention rates, high activity level) and lower cognitive functioning are found it will be assumed to indicate mild cerebral involvement.

Predictors and Correlates of Hyperactivity

Investigators consistently report several biopsychosocial variables that are antecedent to, and concurrently associated with, hyperactivity (Fitzgerald & Zucker, 1994; West & Prinz, 1987). Among these are: 1) high levels of parental reported childhood hyperactivity, 2) parental psychopathic involvement, 3) low levels of cognitive functioning and consequent academic underachievment in both parents and offspring, and 4) several social, contextual variables indicative of an impoverished rearing environment. All of these have been incorporated into the present model in an attempt to explain

neurobehavioral dysregulation (Figure 1). The relevant literature pertaining to these variables is reviewed below.

Childhood Hyperactivity of Parents of Hyperactive Children

Hyperactive children are often times reared by parents who report having been hyperactive as children. One of the first studies to describe this phenomenon was conducted by Dennis Cantwell in 1972. Through extensive interviews, he assessed the prevalence rate of hyperactive behavior in the parents of a group of hyperactive boys (n = 50) and a demographically matched comparison group of boys (n = 50). Boys in the comparison group presented no clinical symptoms of hyperactivity. Results revealed significantly more self-reported childhood hyperactivity in male adult relatives of the hyperactive boys compared to the controls - fathers (16% vs 2%), uncles (10% vs 0%) first cousins (12% vs 2%), and more hyperactivity overall for male and female relatives combined (63% vs 0.6%).

In another classic study, Morrison and Stewart (1973) investigated the psychiatric status of family members of a group of adopted hyperactive children and the family members of a demographically similar group of non-adopted hyperactive children. An asymptomatic, demographically comparable group of children and their family members was used for comparison. The majority of individuals in each group were males, 89%, 97% and 95% respectively. Subjects consisted of 35 adopted-at-birth hyperactive children (mean age 11.2 years), 59 biologic (i.e., non adopted) hyperactive children (mean age 10.9 years), 41 non-hyperactive children from the comparison group (mean age 10.0 years), and their respective family members. Hyperactivity was defined in a similar fashion as current DSM diagnostic criteria for ADHD. Results indicated that significantly more male

relatives (fathers and uncles) in the biologic group reported childhood hyperactivity compared to male family members in the adopting or control groups (12.8% vs. 3.4% vs. 0.8% respectively). These results are consistent with others (Goodwin et al., 1975; Morrison & Stewart, 1970; Martin et al., 1992) who have found similar percentages of hyperactivity in male biologic relatives of hyperactive children.

In a recently reported study of child psychopathology conducted in Great Britain, Schachar and Wachsmuth (1990) report similar findings for childhood hyperactivity in family members of hyperactive boys. Subjects were 83 males aged 7-11 years referred to a children's hospital to be assessed for behavioral and emotional problems; a group of 20 same age and sex normals recruited from classrooms served as controls. Subjects were arranged into several groups based on DSM-III diagnostic assessments of Attention Deficit Disorder with Hyperactivity (ADDH) and CD as follows: 20 children were diagnosed as ADDH, 15 as CD, 28 as ADDH + CD, and 20 as emotionally disordered (e.g., anxiety disorder). Sixty-four percent of the biologic parents of both ADDH and CD groups reported being hyperactive as children, and 58% of the ADDH + CD parents, compared to 33% of the parents of emotionally disordered boys and 20% of the controls. These findings were consistent regardless of the type of hyperactivity diagnosed (i.e., on average 63% of the parents reported being hyperactive as children regardless of whether their son was diagnosed as *Situational* or *Pervasive* hyperactive). Such results reveal the strong association between childhood hyperactivity of parents of hyperactives and the present dysfunctional status of their hyperactive children.

Results from these studies strongly implicate a genetic component for the expression of hyperactivity (see Cantwell, 1975 for discussion). However, a more

plausible notion has been presented elsewhere for such "hyperactive" outcomes. For instance, Goldsmith (1989) illustrates the dependant relationship of genetic contributions from parents with the rearing environment that they (the parents) create. Hyperactive children being reared by parents who report past and/or present hyperactive symptoms may be displaying the *passive gene-environment interaction* that Goldsmith refers to. If the genetically prone hyperactive child is being reared by hyperactive parents, the parents may be supplying an environment that encourages hyperactive behavior (e.g., high levels of exploration, active play, etc.). According to this theory, the interaction of the child's genetic propensity for hyperactivity with the stimulating environment is passive regarding the parental input, however, the hyperactive behavior is free to express itself (i.e., it is unregulated). This theory fits comfortably with experiential-gene interaction theory (Gottlieb, 1991). (See Ham [1993a] for a more in-depth discussion of these ideas.)

Psychopathology in Parents of Hyperactive Children

As has just been noted, the majority of parents of hyperactive children report having been "hyperactive" themselves as children, however, some of these same parents consistently report being psychiatrically ill as adults, specifically revealing high prevalence rates of alcoholism, antisociality, and anxiety disorder (Cloninger et al., 1985; Cotton, 1970; Loeber, 1990). In two of the earlier studies reviewed (Cantwell, 1972; Morrison and Stewart, 1973) significantly greater amounts of alcoholism and antisocial behavior were found in the male relatives of hyperactive boys. In the Cantwell (1972) study for instance, structured interviews revealed twice the incidence of alcoholism in fathers of hyperactive boys compared to controls. When pedigrees were conducted 20% of the male relatives of hyperactive boys were diagnosed alcoholic vs. 5% of the male control relatives

(female alcoholism was very low in the hyperactive group [2%], and nonexistent in the controls). There was also significantly higher rates of antisociality in male relatives of hyperactive boys than male relatives of the controls, specifically, four times as much in fathers and 12 times as much in other male relatives. Furthermore, 12% of the hyperactive boy's mothers were diagnosed with hysteria and 8% of the remaining female relatives, versus none of the female relatives in the control group. This study remains a milestone regarding parental psychopathology in conjunction with childhood hyperactivity, and furthermore, it has been replicated many times (see Hinshaw, 1992).

Morrison and Stewart (1973) reported results similar to those found in the Cantwell study. Particularly, 20% of the biologic fathers of hyperactive boys were diagnosed with primary alcoholism compared to 3% of the adopting fathers and 10% of the controls. Also, more biologic male relatives of hyperactive boys were alcoholic compared to male relatives of adopted hyperactives and controls (12.7% vs. 7.4% vs. 6.5% respectively; $p<.05$ for bio vs. adopt and bio vs. controls). These significant findings for the relationship of alcoholism and hyperactivity as it relates to biologic status consequently buttress the argument for an heritable relationship of the two disorders.

Goodwin et al. (1975) reported significantly higher percentages of childhood hyperactivity and antisocial behavior in a group of adopted alcoholic men ($n = 14$) compared to a group of adopted non-alcoholic men ($n = 119$). Mean age of the two groups was thirty years. Fifty percent of the alcoholics reported having been hyperactive, aggressive, and impulsive as youths compared to 15% hyperactive, 18% aggressive, and 18% impulsive for controls; 21% of the alcoholics reported antisocial behavior in adolescence compared to 2% of the controls. Again, these findings link childhood

hyperactivity and adult psychopathology together strongly implicating hyperactivity as a developmental risk for poor adult behavioral outcome.

Finally, as a result of parental alcoholism and/or antisociality, hyperactive children are many times reared in chaotic home environments with high levels of spousal and child abuse (Reider et al., 1988, 1989; Spellman et al., 1992; Tarter, Hegedus, Goldstein, Shelly & Alterman, 1984; West & Prinz, 1987). Consequently, the high levels of child abuse may be one of the predisposing factors that encourage hyperactive children to exhibit the same type of aggressive, retaliatory behavior. Additionally, physical abuse is thought to account for much of the head trauma reported by hyperactive children which, incidentally, may actually account for much of the ADHD symptomatology in a majority of head injured (whether known or not) hyperactive children. This line of thinking is explored in more detail in the section on neurological implications of hyperactivity.

Cognitive Functioning and Academic Outcomes in Hyperactives

In conjunction with these findings, children exhibiting hyperactivity and hyperactivity related behaviors are often academically underachieved (Frick et al., 1991); overlap rates for academic underachievment and attention deficits in general may exceed 50% (McGee & Share, 1988, cited in Hinshaw, 1992), learning disordered (Lerner & Lerner, 1991), frequently assessed with below average IQs (Barkley, 1981; Goodman & Stevenson, 1989b), exhibit language deficits (Goodman, 1991), and some have even reported substantial portions of hyperactive children to be mentally retarded (i.e., IQ < 70) (Schachar, Rutter & Smith, 1981).

Weiss, Hechtman and Perlman have reported on cognitive outcomes in a group of 75 hyperactive children at a 10 year follow-up (Weiss, Hechtman, Perlman, Hopkins &

Wener, 1979; Hechtman, Weiss, Perlman & Amsel, 1984) and at a 15 year follow-up (Weiss, Hechtman, Milroy & Perlman, 1985). Subjects were initially seen when 6-12 years of age (90% male) on a referral basis at a children's hospital for sustained, long standing hyperactivity; a group of matched controls was used for comparison (n = 43). Follow-up at 10 years revealed that children formerly diagnosed as hyperactive had lower IQ's than the control match group, had completed significantly fewer years of education, had overall lower academic marks in high school, were referred to the courts more often, and a significant percentage were using illegal substances (e.g., marijuana, hash). The authors found that IQ was one of the most important variables in predicting educational achievement and substance abuse in hyperactive adolescents. They also found that SES predicted adolescent and adulthood antisociality (e.g., low SES was highly associated with police involvement). The authors point out that hyperactive individuals were more impulsive than matched controls based on rapid response rates on cognitive tasks and also by virtue of the fact that they had been involved in significantly more automobile accidents. This finding stands out in that some researchers believe impulsivity to be extremely integral in the poor outcome of problem behaviored children regardless of whether the problem is hyperactivity or more severe conduct problems (Loeber, 1990).

In the 15 year follow-up report on the same group of individuals (Weiss et al., 1985) consistent results were found. The majority of individuals diagnosed as hyperactive in childhood continued to exhibit lower WAIS IQ scores, had less education, reported persistent concentration difficulties, and complained of restlessness. Significantly more of the subjects were diagnosed with DSM-III Antisocial Personality Disorder (23% of hyperactive group vs 2% of the controls), and 50% of the hyperactive group reported

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alcohol abuse in the previous 3 year period compared to 25% of the controls.

Gittelman et al. (1985) report similar findings from a 10 year follow-up study of 100 male adolescents with ADDH and 100 controls. Subjects revealed no differences on sociodemographic characteristics or parental variables (e.g., age, education, years married etc.). However, statistically significant differences were found for Wechsler IQ scores; former hyperactive individuals scored lower on verbal and full scale scores. It is interesting to note that a third of the subjects maintained the disorder from childhood into adolescence, and further maintenance of the disorder proved to be a substantial risk for alcoholism and antisociality (substance abuse and criminality were significantly elevated in those maintaining the disorder). However, the authors report that substance abuse ensued only after the onset of CD in the "overwhelming majority of the cases," thus linking more serious outcome with comorbidity rather than "pure" hyperactivity.

The August and Stewart study (1982) was mentioned earlier. In this study the authors looked at the IQ's of two groups of boys, one presenting with what they referred to as "pure" hyperactivity and the other comorbid for hyperactivity and aggression, they referred to this group as "aggressive-undersocialized" hyperactive. Their overall finding regarding IQ was that hyperactive boys who were not aggressive or antisocial had lower IQ's than hyperactive boys comorbid with aggressive conduct problems. Consequently, the group of "pure" hyperactive youths also revealed greater amounts of academic failure than the aggressive hyperactive group. These findings are similar to the Weiss et al. follow-up results, and results from both studies implicate hyperactivity, possibly exclusive of aggressive behaviors, as risk for poor cognitive and academic outcomes.

These follow-up studies yield similar results to those found elsewhere (e.g.,

Mendelson, Johnson & Stewart, 1971), suggesting that hyperactive children are not only at greater risk for poor cognitive outcomes but are concomitantly at risk for various other negative psychosocial and behavioral outcomes. Specifically, the above studies found that in conjunction with poor cognitive and academic outcomes, substance abuse and antisociality were among the most serious outcomes, as well as lower social/emotional functioning.

Frick et al. (1991) looked specifically at academic underachievment in a group of 177 clinic-referred hyperactive children aged 7 to 12 years, some comorbid for CD and some not, in an attempt to elucidate the relationship between externalizing behavior problems and cognitive functioning. In this study academic underachievment referred to a significant discrepancy between the child's expected level of achievement based on full WISC-R assessments and their actual level of academic achievement based on scores of a standardized achievement test (Basic Achievement Skills Individual Screener; BASIS). Results indicate that hyperactivity is more influential than CD in the case of academic underachievment, however, these results are true only when evidence of physical hyperactivity is apparent (i.e., boys with attentional deficits only and not physical hyperactivity performed similar to clinic-controls). Following from these results, it seems apparent that measures of cognitive functioning, IQ in particular, appear to be reliable predictors of academic achievement in general, and it would seem that hyperactivity is especially important in mediating its expression (Hechtman, Weiss, Perlman & Amsel, 1984).

It should be noted, however, that in some cases children who display externalizing behaviors reminiscent of ADHD and CD appear to be at even greater risk for continued

academic underachievement and cognitive difficulties than children displaying only one or the other (Hinshaw, 1992; Loeber, 1993). This is an important prognostic consideration when investigating cognitive outcome in problem behaviored children. Also, it is very important to note that in several of these studies (e.g., Weiss et al., 1985; Gittelman et al., 1985) hyperactive symptomatology was reported to be more chronic in those who were also displaying significant conduct problems, and therefore may reflect a general inherent propensity for behavioral dysregulation. Finally, regardless of which label is used, it seems that inattention and impulsivity are mediating factors for poor cognitive and academic outcome (Hinshaw, 1992).

One study found a high prevalence rate of mental retardation in a group of hyperactive children (Schachar et al., 1981). Ten-year-old hyperactive urban twins in the Isle of Wight study revealed high incidence of extreme cognitive dysfunction. Nearly a fourth (23%) of the severely hyperactive children were mentally retarded according to IQ assessments, compared to the more mild hyperactives (1.5% home - 3.0% school), revealing a high risk ratio for severe cognitive dysfunction in some hyperactive children (reported in Goodman & Stevenson, 1989b).

Several final comments are worth noting regarding the cognitive abilities of hyperactive children. First of all, most studies investigating cognitive outcome in hyperactive children find age related trends (e.g., Frick et al., 1991; Hechtman et al., 1984; Weiss et al., 1985). For instance, lowered cognitive abilities are found often times in younger, hyperactive children (toddlers and young children), whereas, lower IQ scores are found in association with aggressive conduct problems in older children and adolescents (see Hinshaw, 1992). Second, factor analytic studies consistently find higher correlations

of IQ with hyperactivity than with aggressive/conduct behaviors. For example, Schachar, Rutter and Smith (1981) examined the partial correlations of Verbal and Performance IQ and reading level with three factors derived from the Rutter teacher scale - Aggressive-Antisocial, Hyperactive, and Anxious. Correlations for the aggressive and internalizing factors were essentially zero, whereas, hyperactive behaviors were significantly and negatively correlated with Verbal IQ, Performance IQ, and reading level. Hinshaw (1992) reports on findings from other factor analytic studies that have found virtually the same results (e.g., McGee et al., 1984; Anderson, Williams, McGee & Silva, 1989), all of which find attentional deficits and physical hyperactivity to be associated with subaverage IQ and reading delay much more often than aggressive conduct problems.

Language Difficulties and Hyperactive Children

Some hyperactive children exhibit deficits/delays in receptive and/or expressive language abilities, suggesting that "early linguistic deficits become commensurate with underachievment when academic curricula are introduced" (Hinshaw, 1992, pp. 130). In a comprehensive review on externalizing behavior and academic underachievment, Hinshaw (1992) summarized the general literature. Results from most studies revealed a high association between expressive language deficits and externalizing behaviors such as hyperactivity and aggression. He points out that language delays predate academic failure, and he cites research findings indicating that such deficits are causally related to externalizing behavior. Attention deficits are the most highly associated behavioral problem with language delay (aside from autism and severe pervasive developmental disorders). Verbal output, Hinshaw explains, plays a key role in the self-regulation of one's behavior. Consequently, the child who has difficulty expressing himself verbally is at

considerable risk for frustration and negative parent-child and peer-child interaction; such risk may provoke much of the acting out behavior seen in conduct problemed children.

Hinshaw further points out that language difficulties are highly associated with lowered IQ and perceptual - motor deficits, all of which point to neurologic involvement (i.e., neurodevelopmental delay). The neurodevelopmental delay hypothesis suggests that delays in neurodevelopment may predispose a child not only to language and motor difficulties, but also to externalizing behaviors such as hyperactivity and/or conduct problems; it is the perceptual and motor function that reflects neurodevelopmental delay in association with language deficits (Beitchman, 1985, cited in Hinshaw, 1992), and it is possibly the combination of these that predispose a child to acting out behaviors such as hyperactivity. Also, the neurologic involvement hypothesized here may have some responsibility for the associated attentional difficulties and impulsivity seen in language delayed hyperactive children. Similar lines of thinking are explored in the following section, and such outcomes are discussed in light of the present study findings.

Social/Contextual Factors

Most of the studies reviewed so far also found several contextual attributes that may be implicated in the etiology, expression, and developmental progression of hyperactivity. Socioeconomic status (SES) is often times involved in the expression of hyperactivity and hyperactive related difficulties (e.g., academic underachievment) (Schachar et al., 1981; Taylor et al., 1986b; Weiss et al., 1984); levels of SES are consistently lower for families where hyperactive children are being reared. Level of family income and parental education of hyperactive offspring is generally lower than that seen in either clinic and normal comparison samples. These findings indicate the integral

role that the rearing environment plays in the etiology and expression of behavioral regulation. Therefore, low levels of SES, income, and education should be considered indicative of high risk for poor behavioral outcome.

Goodman and Stevenson (1989a) looked at the prevalence rate of hyperactive symptoms in 570 thirteen-year-old twins being reared in London. Results revealed that 20% of the children met criteria for Situational Hyperactivity (hyperactivity seen in either the home or school setting - a milder form of hyperactivity) and 5% for Pervasive Hyperactivity (hyperactivity exhibited in all settings - a more severe form of hyperactivity). Pervasive Hyperactivity was significantly associated with low levels of SES. Specifically, 6% of the children from low SES families were pervasively hyperactive compared to 2% from high SES families. These results reveal a three-fold increase for risk of Pervasive Hyperactivity possibly due to influences of socioeconomic status. Further, Goodman and Stevenson compared these findings with the Isle of Wight study (Rutter et al., 1970, cited in Goodman and Stevenson, 1989b). They point out that the urban children in the Isle of Wight study who were being reared in lower sociodemographic conditions were at an even greater risk for problem behaviors than the poor rural children in their study.

In the Ham et al study (1994), higher levels of hyperactivity in 3- to 6-year-old SOMAs was found in the context of significantly lower levels of SES, family income, and parental education. Furthermore, regression analysis revealed that these social, contextual variables were also predictive of hyperactivity in SOMAs. Similarly, Ellis (1993) found lower sociodemographic functioning in homes of children who were more impulsive and conduct disordered. Such results lend credence to the fact that contextual traits are consistently and strongly implicated in poor behavioral expression such as hyperactivity

even in the presence of parental psychopathic involvement. Many other studies have found similar findings (e.g., Goodman & Stevenson, 1989a; Hechtman et al., 1984; Schachar, Rutter & Smith, 1981; Taylor et al., 1986a; Weiss et al., 1979, 1984). Overall, the data suggest that low levels of SES, parental education, etc. are involved in the expression of hyperactive behavioral problems.

Neurobiological Theories of Hyperactivity and Other Dysregulatory Behavior

This section reviews the basic neurology/neuropsychology literature pertaining to behavioral disorders. The particular focus is on hyperactivity and its relationship to neurologically mediated behavioral dysregulation in children of alcoholics. It is argued that such relationship may be the function of an inherited, but environmentally mediated, dysregulatory mechanism in certain individuals. Findings pertaining to neurologic status of brain damaged patients and how they are similar to hyperactive children regarding behavioral/cognitive functioning are also reviewed. Sensitive periods of CNS development and how they may be implicated in attention and motor function are reviewed, as well as an older theory of neurodevelopmental delay as it relates to hyperactivity. Last, a brief examination of the impact of environmental influences on neurologic development and consequent hyperactive related outcome is considered.

Mechanisms: Biobehavioral Dysregulation

Hyperactive symptomatology has been suggested by some to reflect an inherited, mechanistic phenomenon (see Tarter et al., 1990), however, one whose biological basis is dependent upon the developmental rearing environment for gaining its expression (Fitzgerald & Zucker, 1994). Within this conceptual framework Henri Begleiter (personal communication, May, 1992) has suggested that the behavioral patterns indicative of

hyperactivity may not be resultant of a genetic link with alcoholism or any other parental psychopathology, but may be indicative of an inherited, "pathologically non-specific" behavioral dysregulatory mechanism. There is a strong neural component to Begleiter's theory concerning this behavioral dysregulatory mechanism, thus the term "bio"behavioral is used to describe it. He specifically suggests a systems-like approach to the interaction between set genetic components and the developmental rearing environment. The present study hypothesis further suggests that hyperactive behaviors may not necessarily be related to alcoholism in a genetic sense but rather, may be more developmentally dependent upon consequential environmental events.

This notion of a neurologically mediated behavioral dysregulation was a vital part of the underlying premise of the present study. The general hypothesis was that SOMAs inherit an alcohol related neurobehavioral dysregulatory mechanism, and that this mechanism gains expression in his behavioral patterns depending upon his experiences with the rearing environment (see Gottlieb, 1991). In the present sample the rearing environment is extremely conducive to dysregulatory behaviors and therefore lends itself to a developmental systems perspective for explaining this mechanistic phenomenon. The present study could only assess the neurological implications of such a model in a cursory manner using neuropsychological assessment tools in conjunction with latent variable modelling techniques.

Sensitive Periods in Neurodevelopment

Goodman (1991) suggests the idea of "sensitive" or "critical" periods as a plausible explanation regarding the neurological underpinnings of hyperactive symptomatology. He believes that hyperactivity may result from an interruption in the growth process during

"sensitive" ontogenetic periods of development in areas of the brain thought to subserve functions of behavior regulation. Sensitive periods for brain development are stages in development of the CNS during which a particular segment is most vulnerable to environmental or experiential influence. Human beings have sensitive periods for all major organ structures, and also for higher, executive functional components of the brain. For example, sensitive periods are also apparent in the development of language acquisition. Any kind of alteration or interference during the normal sequence of development in areas of the brain subserving language has long been known to delay or alter normal language development (e.g., Lenneberg, 1967; Luria, 1966). There is also evidence for sensitive periods for the human visual system. In this case, if deprivation of one visual field occurs for a substantial period of time during early development it can leave one with an under-developed, dysfunctional visual system. Likewise, there is similar evidence for sensitive period(s) in the development of auditory discrimination (Birch & Stager, 1988; Werker & Tees, 1984, cited in Goodman, 1991) with similar consequences to the system if an interruption occurs during these period(s).

It follows then, that there may also be "sensitive" or "critical" periods during development for higher cognitive and behavioral functioning, especially regulation of attentional and motoric behaviors. Goodman stipulates several areas of the anterior cerebral cortex subserving attentional and motoric regulatory functions that may very well encounter similar critical periods during pre- and early post-natal development. Furthermore, an interruption in development of CNS functional systems (due to environmental deprivation or adverse circumstances, for instance) early in life, may irrevocably alter attention and/or regulatory capabilities. Hodges & Tizard (1966, cited in

Goodman, 1991) reported that orphans adopted before the age of 5 years experienced on average a 20 point increase in IQ compared to a very minimal increase in orphans adopted after 5 years of age. Goodman suggests that these findings indicate sensitive period(s) for cognitive development, and if children are exposed to adverse living conditions (e.g., institutional living) during these periods of time, cognitive capacities may remain under-developed. Furthermore, all of the children in the Hodges and Tizard study who experienced institutional care for at least two years experienced more inattentiveness and restlessness at follow-up several years later than the matched control group, again suggesting the possibility of a sensitive period for attention and motor regulation.

Children expressing the primary symptomatology of ADHD are also often characterized as clumsy, lazy, and poorly coordinated (Taylor et al., 1986b; Horn & Ialongo, 1988). Motor difficulties many times exhibit themselves as poor fine and gross motor control. Some have even suggested that the high incidence of accidents encountered by hyperactive children can be attributed to this associated clumsiness. These motor behavior anomalies are often believed to be related to CNS delay and/or dysfunction. In keeping with Goodman's theory of critical periods, there is reason to suspect that motor deficits reflect cerebellar involvement. Sensitive periods of cerebellar growth occur during fetal and first year postnatal development. It has been noted that several childhood disorders appearing as a result of atrophy, destruction, hypoplasia, and/or fusion of molecular layers in the cortex and vermis regions of the cerebellum are consistent with childhood hyperactivity (Lyon & Gadisseux, 1991; Rutter et al., 1990; Taylor et al., 1986b). Also, developmental cerebellar damage has been linked to disorders such as autism and mental retardation of varying types and furthermore, hyperactivity has

been noted to appear in many cases conjointly with these disorders; such disorders reveal no apparent structural abnormality of the telencephalon. It is therefore possible that the poor motor coordination so often seen in hyperactive children may be associated with cerebellar involvement, however, of a milder extent than that seen in the more serious cases of autism and mental retardation. Such an hypothesis extends the neurological implications of hyperactivity from neo-cortical areas to more primitive structures of the brain.

Evidence backing the plausibility of this theory can be seen in results of histological investigation of cellular migration in the cerebellum (Lyon & Gadisseux, 1991). It appears that there are no new neurons generated in the telencephalic cortex past gestational week 20 (for the most part). However, external granular cells in the cerebellum continue to multiply and migrate throughout the fetal period and up to 10 to 12 months postnatally. Therefore, one may assume that detrimental influences have a larger window of opportunity during early cerebellar development than during development of cortical areas involved in higher regulatory functioning (e.g., anterior cortex). This theory may even be tied into some of the findings related to cognitive dysfunction resulting from disorders of neuronal migratory processes and inappropriate (i.e., early or late) neural cell death (Lyon & Gadisseux, 1991) and/or poor neuronal pruning during fetal development (Goodman, 1991).

Evidence also exists that implicates higher order neural involvement in motor dysfunction of hyperactive children. Denckla and Roeltgen (1992) make reference to several studies in which choreiform movements were seen in individuals exhibiting primary inattention (e.g., Wolf & Hurwitz, 1973, cited in Denckla & Roeltgen, 1992). In another

study (Denckla & Rudel, 1978) motoric slowness and excessive-for-age overflow movements distinguished hyperactive from non-hyperactive individuals. Wolff et al. (1985) found that mirror movements were the single best predictor of hyperactivity in boys with learning disabilities, and that they were also the best predictor of aggression in psychiatrically disturbed boys; thus a connection between motoric dysfunction, conduct problems, and cognitive functioning can be seen. The authors further suggest that the inability to suppress unintended overflow motor behaviors may reflect an underlying neural substrate, one that is involved in the regulation of verbal and behavioral responses of selective attention. A plausible substrate for this type of dysfunction is most likely located in the left hemisphere, particularly in peri-sylvian areas (the supramarginal and angular gyri, the superior and middle gyri of the temporal lobe, and primary and supplemental motor areas of the anterior cortex, Faglioni & Basso, 1985).

Even though these hypotheses are speculative in nature they do present us with some fresh insights into possible etiologies of childhood behavioral disorders such as ADHD. They also suggest an exciting avenue of inquiry concerning the association of ADHD behaviors and their development in conjunction with environmental influences. For instance, if "sensitive" or "critical" periods for motor and attentional regulation do in fact exist, then this opens up new areas for exploration into prevention and treatment of a subset of hyperactive children who are disordered primarily because of neural (i.e., biologic) involvement.

Although all of this is very interesting and very pertinent to hyperactivity, how does it relate to hyperactivity in conjunction with the risk for alcoholism? In the present study I have treated motoric skill, attentional capabilities, hyperactivity, and impulsivity as

a global measure of cerebral integrity in SOMAs. It was hypothesized that deficient motor skills, attentional ability, hyperactivity, and impulsivity in children vulnerable to alcoholism and alcoholism related pathology may suggest cerebral and cerebellar involvement, and furthermore, the hyperactive dysfunction in SOMAs may be due to an interruption or alteration in normal pre- and/or post-natal development. Additionally, in such cases where CNS involvement is suspected in SOMAs, we cannot rule out hereditary influences (i.e., deficient genes transmitted from the parents) or pathophysiological circumstances that arise during pregnancy and/or peri-natally. No matter the case, future biologically based studies need to be conducted to determine the validity of such hypotheses. The present study took some theoretical liberty in assuming the possibility of neural involvement in hyperactive SOMAs. However, the indices used to measure neuropsychological involvement speak adequately to this possibility.

Anterior Cerebral Involvement

Anterior regions of the brain, particularly the prefrontal cortex (e.g., Supplementary Motor Areas), are known to subserve the executive functions of decision making, formulation of goals, attentional capabilities, and behavioral self-regulation (Lezak, 1983). Luria (1966) described this area of the brain as an "executive regulatory center" for behaviors intrinsic to ADHD (i.e., attention, motoric output, etc.). Thus, hyperactivity may have its origins in a dysfunctional portion of the anterior cortex in some individuals; such behaviors would reflect an inability or failure to self-monitor goal-directed behavior (Barkley, 1981). Alcoholics and their children have been reported to display inabilities in sustaining attention, excessive hyperactivity, poor perceptual-motor abilities, and impulsivity, all of which are reminiscent of individuals who have experienced

mild anterior cerebral trauma and/or dysfunction (see Tarter et al., 1985, 1990). Incidentally, parallel findings from animal studies also suggest the prefrontal cortex as a primary neurosubstrate for mediating the effects of ethanol on similar behavioral characteristics such as attention and hyperkinesis (e.g., Davenport, Hale & Whiteside, 1984). In addition to this, and in conjunction with present study hypotheses, Luria (1966) described the functions that the prefrontal cortex area has in language mediation, noting that interruptions of connectivity or damage to specific prefrontal areas has noticeable affects on verbal regulatory behavior. Pre-alcoholics and children at risk for alcoholism have been shown to exhibit disrupted language mechanisms (Tarter et al., 1985), specifically impairments in language capacity (Hegedus, Alterman & Tarter, 1984).

Several studies have identified neuropsychological abnormalities in both alcoholics and their offspring in connection with the hyperactive syndrome. Such deficits include inability to persist at visual tasks (Alterman et al., 1984), inability to delay gratification (Ellis, 1993), difficulties with abstract problem solving (Schaeffer, Parsons & Yohman, 1984), and attention and memory deficits (Tarter et al., 1985), all of which implicate the anterior cerebral cortex. For example, Schaeffer et al. (1984) looked at neuropsychological differences in 130 alcoholics, some with and some without family histories of alcoholism. The major finding of the study was that alcoholic men with a family history of alcoholism had significantly more difficulties with abstract problem solving and perceptual-motor tasks than alcoholics without a family history of alcoholism. The authors offer an interesting conclusion. They suggest that it may be the familial component of alcoholism (i.e., a genetic element) that enhances one's chances for neurological complications not simply alcohol consumption, and furthermore, that it may

be the alcoholic with a family history of alcoholism who has premorbid neurodeficits involving cognitive and motoric functions. These findings argue for a neurobehavioral explanation for the vulnerability to alcoholism and its relationship with hyperactivity (Hegedus et al., 1984), and one that specifically implicates frontal brain areas.

As previously stated, hyperactivity often occurs at substantially higher rates in children reared in high risk alcoholic environments. Keeping in mind that physical hyperactivity is usually the most apparent and first-to-be-diagnosed externalizing behavioral component of hyperactivity, it may specifically reveal neurologic involvement in some individuals with an inherent propensity for dysregulated motor behavior. Tarter and his colleagues (1985, 1989, 1990) suggest that high levels of physical overactivity reflect such CNS involvement. Particularly, this type of dysfunctional motoric behavior would implicate deficits in the pre-frontal cortex, as this portion of the brain mediates self-regulation of motor behavior. Results of a recent study by Tarter et al., (1989) strongly suggest that physical overactivity reflects a genetic predisposition towards alcohol abuse, and therefore one that is particularly relevant to the etiology of alcoholism. The authors are quick to point out, however, that these findings do not necessarily confirm or disconfirm a genetic predisposition for alcoholism, but they are suggestive of anterior cerebral involvement of self-regulatory function in children at risk for alcoholism, and therefore biologically relevant.

Additionally, August and Stewart (1982) suggest that since physical overactivity is commonly seen in isolation among brain damaged individuals, that when appearing as the primary symptomatology, it may be particularly indicative of brain involvement. Further, they discuss findings showing that when hyperactivity is found in the presence of conduct

problems, it is not correlated with any neurologic etiological outcome (i.e., it is not linked with measures of brain dysfunction). This suggests that a subgroup of hyperactive individuals may exist who are not aggressive by nature but rather, their pathology is neurobiologically mediated and expresses itself in societally non-offensive outcomes.

In summary, hyperactivity and attentional difficulties may have neurological implications, higher levels of which may be indicative of possible neurologically mediated behavioral dysregulation. Furthermore, these behaviors many times appear in conjunction with cognitive deficits and many times they appear exclusive of aggressive behaviors, and the literature strongly implicates a milieu of alcoholism.

Neurodevelopmental Delay

Bakwin and Bakwin (1966) discussed several types of hyperactive children that, from an etiological standpoint, may further aid in delineating group(s) of hyperactive children. One type of hyperactivity involves individuals born with neurologic lesions: 1) those with minor cerebral damage, thus the historical "minimal brain damage" syndrome, or 2) those who express hyperactive behaviors due to infantile autism or reactive behavior disorders. The authors also describe another group of "neurodevelopmentally delayed" hyperactive children; it is this group that is of interest in the present discussion. Outcome for these children is similar to those discussed by Goodman (1990) in which there was an interruption of neurodevelopment during "sensitive" ontogenetic periods, and for individuals with anterior cerebral involvement (Tarter et al., 1990). Such an hyperactive child is described as neurodevelopmentally delayed with tendencies toward hyperactivity (general fine and gross motoric overactivity) and associated clumsiness or awkward motoric behavior. The authors suggest that this type of developmental hyperactivity is not

associated with any known anatomical brain abnormality but rather, is thought to be due to a neurodevelopmental "delay or disturbance in the maturation of those areas of the brain which have to do with ... motor coordination" (Bakwin & Bakwin, 1966, pp. 166). However, more recent evidence from studies of clumsy children suggests that vestibular dysfunction may be involved where gross motor difficulties are apparent, particularly balance, postural acts, left-right confusion, and gravitational insecurity. Additionally, fine motor disturbances (i.e., those involving small or manipulative muscles) in clumsy children may be resultant of somatosensory (dorsal column medial lemnicscal) tract involvement (see Ayres, 1972).

Bakwin and Bakwin further divide developmental hyperactivity into two subtypes with respect to developmental course and outcome; one in which there is amelioration of the primary features of the disorder due to a "catch-up" phenomenon in neurodevelopment; the other in which there is maintenance of the disorder due to a failure in later neurodevelopment. Bakwin and Bakwin remind us that it is essential to ascertain whether the child has sustained peri- or post-natal head or cervical trauma in order to rule out neurologic implications due to lesions of primary or associative areas of the brain, which consequently would indicate a disorder of the first type (this has been a notable concern of researchers in this area, as high rates of unconsciousness have been reported due to head trauma in hyperactive children and children of alcoholics [e.g., Schaffer et al., 1984; Tarter et al., 1984]). When/if spontaneous disappearance of the primary symptoms of hyperactivity occurs in late childhood and/or adolescence this implies the "catch-up" phenomenon in neurodevelopment indicative of the first subtype of neurodevelopmental delayed hyperactivity. This reasoning is consistent with findings from several follow-up

studies reporting improvement and/or disappearance of the primary symptoms of hyperactivity in late childhood and early adolescence (e.g., August et al., 1983; Campbell, 1987; Weiss et al., 1979, 1985).

However, when there is no evidence of brain injury, and when "catch-up" does not occur, this would suggest the presence of a persistent neurodevelopmental abnormality rather than simply a delay in neuromaturation. It is possible that this subtype of neurodevelopmentally delayed hyperactivity (i.e., the persistence of the disorder) is a form of hyperactivity that appears to be more chronic in nature, and one that undergoes an evolutionary process leading to associated adolescent (e.g., ODD and/or CD) and adult related disorders (e.g., Alcoholism and APS). Further, children who express the primary symptoms of hyperactivity co-morbid with aggressive behavior and noncompliance may be those with this type of persistent neurodevelopmentally delayed hyperactivity.

Environmental Influences on Neurodevelopment and Hyperactivity

Concerning contextual or environmental factors and their impact upon neurodevelopment, it has been hypothesized that when children who are disposed to behavioral problems are reared in adverse environments (e.g., high levels of alcohol abuse, physically abusive behavior, low SES etc.), the rearing environment may serve to substantially exacerbate their already-present dysregulatory behavior and set them on a pathway of future pathology such as alcohol abuse and consequent negative alcohol related outcomes such as antisocial behavior and depression (Cloninger et al., 1985; Ham et al., 1994; Loeber, 1990; Tarter et al., 1984). Hyperactive symptomatology is also reported to be highly related to adverse family situations - marital discord, parental depression, and punitive, critical parenting strategies (Goodman & Stevenson, 1989b).

Some have suggested using caution when interpreting results where children are being reared in adverse, high risk environments, because parents are suspect at exaggerating their child's pathology. However, the majority of studies presently reviewed suggest that this is not a correct assumption in that high correlations between parental ratings of hyperactivity and teacher ratings and objective measures of hyperactivity are found (e.g., Goodman & Stevenson, 1989b; Rutter et al., 1990).

Summary

High levels of hyperactivity, attention span difficulties, distractibility, and impulsivity (HADI) according to the theories and empirical evidence presented here may have several neurologically based etiologies in connection with risk for alcoholism. They may be manifestations of: 1) inherited biobehavioral dysregulation, 2) an inherited pathologic condition, 3) an interruption in development of areas in the CNS that subserve these functions, 4) neurodevelopmental delay, or 5) a lack of normal neuronal development due to an inadequate, suboptimal rearing environment. Loeber (1990) suggests that hyperactive behaviors in certain individuals may have their origins in neurologic development, and it is these individuals that are at greatest risk for future development of ODD, CD, APS, and alcoholism.

These hypothesized roles of hyperactivity, its expression, outcomes, and relationship to adult outcomes are based on substantial findings in neurology, neuropsychology, and psychology, but they are by no means conclusive regarding hyperactivity and its etiology, progression, predictors, and outcome in conjunction with risk for alcoholism. They are still speculative in nature, but they do offer a base from which behavioral scientists can begin to look at this phenomenon from different

perspectives than in the past.

Alcoholism and its Relationship with Hyperactivity

As previously touched upon, many studies reveal a high degree of relationship between familial alcoholism and hyperactivity, postulating it to be one of the primary antecedents to alcoholism, especially in children at risk (Cloninger et al., 1989; Goodwin et al., 1975; Knop et al., 1985; Morrison & Stewart, 1970; Wood, Wender & Reimherr, 1983; Workman-Daniels & Hesslebrock, 1987). Based on the theoretical and empirical ideas presented so far it is necessary to reiterate that hyperactivity (and possibly related conduct problems) may be precursorily related to alcoholism **only** in children who are inherently predisposed to the biobehavioral dysregulation discussed here. Moreover, it is hypothesized that the alcoholic rearing environment facilitates or encourages the expression of this already-present propensity to dysregulation and consequently to the later development of a substance abusing personality, resulting in substantially more serious outcomes than the inherited propensity to behavioral dysregulation alone would produce.

However, as previously mentioned, the methodologies utilized in other alcohol/hyperactivity studies leave several things to be desired. First, prevalence rates of hyperactivity are mostly retrospective; there have been few prospective studies focusing on the early manifestations and developmental course of hyperactivity and conduct problems as they relate to the development of alcoholism and alcohol-related psychopathology (Campbell, Breaux, Ewing, & Szumowski, 1986). There have also been few studies investigating the occurrence of hyperactivity and related conduct problems in COAs during the preschool years (i.e., infancy and early childhood); most studies

reviewed thus far, and those which will be reviewed in this section, deal mainly with late childhood and adolescence. Therefore, one of the primary purposes of the present study included looking at hyperactivity (as indexed by high levels of hyperactivity, attentional deficits, distractibility, and impulsivity) and related conduct problems in SOMAs and SOMnAs during late infancy and early childhood (2 to 7 years old). If hyperactivity is a predisposing factor to alcoholism, its symptoms should then, by definition (APA, 1987; 1994), begin to surface in late infancy and be fully apparent by the age of seven. It follows then that this is the time period in which children should be monitored for such behavioral expressions, and also the time to begin investigation of the etiologic and precursive relationships of the problem behavior. Thus, one of the unique qualities of the present study is the age of the children under investigation and its prospective character.

Recent research on children with alcoholic fathers presents us with some convincing evidence that the hyperactive syndrome may be associated with the later development of alcoholism. The behavioral characteristics of hyperactivity are more evident in children at high risk for alcoholism compared to children not currently at such risk - they are also more evident in alcoholics themselves. For example, both alcoholics and their sons are consistently reported as overly active and exhibiting rapid tempo (Fitzgerald, et al., 1990; Tarter et al., 1985). Individuals who are particularly vulnerable to alcoholism have difficulties sustaining attention (Alterman et al., 1984) and show less freedom from distractibility (Tarter et al., 1985; Taylor et al., 1986b). Therefore, hyperactive symptomatology may exhibit itself in children with a predisposition towards alcoholism, and further, it may be one of the markers or precursors present in these children who will develop alcohol related difficulties later in life.

In the following sections the extant literature will be briefly reviewed linking the expression of hyperactive behaviors with alcoholism. In the first section, findings on the genetics of alcoholism in direct association with hyperactivity will be examined. Second, research evidence from adoption and twin studies regarding the relationship of hyperactivity and alcoholism will be reviewed. In the subsequent section, family studies and self-reports of hyperactivity in alcoholics and the various pathologies found in these families will be explored. Finally, child cognitive functioning, as it relates to hyperactivity and parental alcoholism, will be examined.

Genetic Theories of Alcoholism and Hyperactivity

There is some controversy as to whether hyperactivity (as a possible precursor to alcoholism) and alcoholism are etiologically due to genetic components, environmental influences or a combination of the two. Many studies support the notion of a specific heritability component of hyperactivity (e.g., Goodman, 1989), while others contend for an heritability component only in conjunction with the expression of alcoholism (Cantwell, 1972; McMahon, 1981; Morrison and Stewart, 1973) and antisociality (Robins, 1986 cited in Loeber, 1990). However, both the child behavioral disorders and adult alcoholism literatures (as well as behavioral genetics, e.g., Plomin & Daniels, 1987; Tarter, Alterman & Edwards, 1985) lead us to believe that there is a combination of both in the etiology of alcoholism (Cloninger, Bohman, Sigvardsson & von Knorring, 1985; Goodwin, 1979).

To broach this controversial subject, let me first briefly review a theoretical, yet somewhat common sense, approach to the nature-nurture controversy in this area. Goodman and Stevenson (1989b) remind us of the known fact that children and their parents have their genes in common. It is possible, they presume, that the gene(s) that a

child inherits which predispose him/her to hyperactivity and other developmentally related disorders, may be the same gene(s) s/he inherited from his/her parents that predisposed the parents to their own pathology (e.g., alcoholism, antisociality). They offer support for this theory with evidence from the extant literature (e.g., Moffitt & Silva, 1988; West & Prinz, 1987) that strongly suggests hyperactivity is developmentally linked with adulthood psychopathologies such as alcoholism and antisocial personality disorder (Gittelman et al., 1985). This theoretical assumption fits comfortably with Begleiter's behavioral dysregulation hypothesis and Tarter et al's. (1985) temperament theory. Both suggest that a child may inherit the genetic component which predisposes him/her to general behavioral dysregulation, and if the child is reared in a high risk environment conducive to a certain pathology, then s/he is more apt to express that specific pathologic state in some way or another. According to recent behavior genetic theory, this would increase the overall heritability of the dysregulatory mechanism described here (see Goldsmith, 1989). Furthermore, it is worth mentioning that the majority of hyperactivity is expressed in boys, gender ratios being six to nine times in favor of males to females, (APA, 1987), thus further suggesting a possible sex-linked genetic phenomenon; it also suggests that simply being male is in itself a risk factor for hyperactivity and other related developmental disorders (Geschwind & Galaburda, 1985; Rutter et al., 1990).

Willerman (1973) found high heritability rates for hyperactivity in a study of 93 sets of same-sexed twins. (Heritability refers to the proportion of the phenotypic [observable] variance accounted for by, or in association with, genetic differences among individuals [Goldsmith, 1989]). The heritability estimates in the Willerman study were calculated by dividing the additive genetic component of activity ($r_{MZ} - r_{DZ}$) by the

phenotypic portion of activity level ($1 - r_{DZ}$), where r_{MZ} refers to the interclass correlation for the monozygotic twins and r_{DZ} refers to the interclass correlation for the dizygous twins. Heritability rates were 0.82 for males and 0.58 for females (heritability for males and females combined = 0.77). When he looked at the top 20% of hyperactive children (8 MZ and 16 DZ twin pairs) the heritability estimate was still 0.71, strongly suggesting "a substantial genetic component to activity level". Although such evidence strongly implicates a genetic or "hard wired" component in the expression of hyperactivity, this by no means precludes a substantial interplay with the various milieu an individual is exposed to (i.e., neurophysiological, behavioral, and external environmental experience [Gottlieb, 1991]).

However, in order to claim that the etiology of hyperactivity is a genetic one, or to make the more complex claim that hyperactive related behavior is a genetically relevant precursor to alcoholism, biological markers must be identified in the relationship of the two. Research over the last decade has begun to uncover some of the biological characteristics relating to just such a relationship. One characteristic that is relevant to hyperactivity and is known to be genetically transmitted and distinctive to alcoholics is certain EEG patterns and Evoked Potential (EP) aberrations (Gabrielli, Mednick, Volavka, Pollock, Schulsinger & Itil, 1982; Volavka, Pollock, Gabrielli & Mednick, 1985). Alcoholics and their offspring have a general tendency to show excessive resting Beta activity unlike non-alcoholics (Volavka et al., 1985). The link between hyperactivity and the subsequent development of alcoholism has been noted and consequently, studies utilizing these techniques have observed similarities in brain wave activity between hyperactive youths and their alcoholic parents.

Recent progress has been made to further support a genetic claim utilizing these electrophysiological techniques. Specific areas of the brain that are affected through chronic alcohol abuse (e.g., frontal lobe) have been isolated and are consistent with those areas affected in hyperactive children (see Galanter, 1985). For instance, several studies evaluating brain EPs in adolescent males with alcoholic fathers (Begleiter, Porjesz, Bihari & Kissin, 1984) and hyperactive children (Zambelli, Stamm, Matinsky & Loisell, 1977) revealed marked differences in P300 and N100 wave components. Specifically, there is an attenuation of the P300 amplitudes to task relevant targets and a reduced N100 amplitude to all stimuli in hyperactive youths. The P300 is a positive wave form that particularly reflects a neurophysiological substrate of attention; this pattern occurs approximately 300 milliseconds after stimulus presentation (Tarter, Laird & Moss, 1990). It was noted in the Begleiter et al. study that Evoked Response Potentials (ERPs) in boys with alcoholic fathers were similar to those found in their fathers even though they had not started drinking. Note, however, that similar findings have been found for other substance abusers (e.g., cocaine; Holden, 1991) and thus, this phenomenon may not be specific to alcoholism per se but rather, may be related to a general substance abusing personality. These findings do "unequivocally reflect CNS functioning that is unrelated to alcohol's acute or chronic effects" (Tarter et al., 1990), and therefore probably reflect an heritable CNS defect - one that is related to a general propensity for dysregulated behavior.

Gabrielli et al. (1982) found faster general EEG patterns in children of alcoholics than children of non-alcoholics. The authors hypothesize that the faster EEG is an heritable neurological component that might be one of the notable biologic antecedents inherent to alcoholics and consequently their offspring. They argue that since fast EEGs

are associated with tension and anxiety while slower EEGs are associated with relaxation, this might be one of the biobehavioral mechanisms high risk individuals inherit. As one possible explanation the authors suggest that the alcoholic resorts to drinking in order to reduce the increased neuroelectroactivity associated with the fast EEG. In turn, the ethanol causes a slowing down of brain activity thus enabling the alcoholic to escape the "uncomfortable state associated with fast brain activity" (Gabrielli et al., 1982). Merging this theory with behavioral dysregulation theory in pre-consuming hyperactive children might render the notion that hyperactive, inattentive children, who are struggling with the associated tension and anxiety they have inherited from their fathers, might possibly be attempting to compensate for or relieve the tension by physical acting out behaviors (i.e., high motoric output - both tempo and duration) - in so doing, they are releasing their "pent-up" energies. (Interestingly, along these same lines Tarter, Laird and Moss (1990) have pointed out that amphetamines are the second most preferred drug by alcoholics. The implication being that the therapeutic effects of these drugs are found to be common among some alcoholic and hyperactive individuals).

Adoption and Twin Studies on Alcoholism and Hyperactivity

Adoption studies have been extremely useful in revealing associations of adult alcoholism and child psychopathology, as well as other factors that may predispose an at-risk child to alcoholism. It has been shown that adopted sons of alcoholics are four times more likely to become alcoholic than adopted sons of non-alcoholics (Cadoret & Gath, 1978; Goodwin, Schulsinger, Moller, Hermansen, Winokur, & Guze, 1974). In a landmark study, Morrison and Stewart (1973) evaluated the psychiatric status of a group of adopting parents (n = 35) and a group of biological parents (n = 59), all of whom had a

"hyperactive" child. A control group (n = 41) was used for comparisons - none of the children in this group were "hyperactive". Hyperactivity was diagnosed consistent with current diagnostic criteria. The majority of subjects in this study were males - 97% in the biological group, 89% in the adopted group and 95% of the controls. Results revealed that biological fathers had significantly higher rates of alcoholism (20.1%) than did adopting fathers (2.9%), and were more likely to report having been significantly more hyperactive as children than adopting fathers (biologic = 15.3%, adopting = 8.6%). Such findings favor the idea of an heritable component of hyperactivity in conjunction with alcoholism but, as the authors point out, with a co-dependency of alcoholism being a significant interactive factor in its expression (i.e., one aspect of environmental interplay).

In one of the most classic adoption studies, Cloninger and his associates (1985) identified and investigated two types of alcoholics - Type I and Type II alcoholics, both of which are pertinent to the present discussion. Type I alcoholics are alcoholics who meet several criteria: 1) their biological parents reveal mild alcohol abuse; 2) they exhibit low levels of antisocial behavior; and 3) they report a later onset of abuse and/or dependence. Children born to Type I alcoholics therefore are thought to have a genetic background for alcoholism, but not to as great a degree as the Type II alcoholic (see below). When the Type I child is raised in an environment characterized by lower sociodemographic factors, the suboptimal rearing environment serves to further increase their risk for alcohol abuse and/or dependence and consequently poorer outcome results.

Type II alcoholism, on the other hand, is expressed in those individuals whose biological fathers, 1) reveal more extensive levels of alcohol abuse (requiring more medical treatment), 2) have earlier onset of the abuse, and 3) express relatively high levels

of criminality or antisociality (requiring longer and more frequent incarcerations). Cloninger et al. calculated the heritability rate of Type II alcoholism to be about 90% in their male subjects. Type II sons revealed more severe levels of alcohol abuse regardless of the environment they were raised in thus buttressing the argument that this type of alcoholism contains a strong heritability component. It would appear then that Type II alcoholics have a greater inherent (i.e., genetic) propensity for alcohol abuse/dependence and consequently, the environment they are in has less of an impact on their outcome.

Zucker (1987) has suggested several alcohol typologies that may also aid us in elucidating vulnerability to alcoholism, one of which pertains specifically to alcoholics in the present study. The four alcoholisms he discusses are: 1) antisocial alcoholism, 2) developmentally limited alcoholism, 3) developmentally cumulative alcoholism, and 4) negative affective alcoholism. The antisocial alcoholic is of primary importance to the present study. In other analyses Zucker et al. (1993a, 1993b, 1994) have found a group of antisocial alcoholics to be significantly different on psychological (e.g., depression, alcohol problems) and sociodemographic (e.g., low socioeconomic achievement) (Fitzgerald & Zucker, 1994) and family history variables when compared to a group of non-antisocial alcoholics. The antisocial alcoholic is primarily characterized by a history of childhood aggressiveness and an early onset of alcohol problems, but also has a substantially stronger family pedigree for alcoholism involving a multigenerational history. Zucker also suggests that the antisocial alcoholic has been socialized to his/her antisocial behavior.

Similar ideas were investigated in the Goodwin et al. study (1975) briefly mentioned earlier. In this study the authors interviewed 133 male adoptees in a demographically similar Danish population sample. Fourteen of the men in the sample

were classified as chronic alcoholics, the remaining 119 men served as controls. Ten of the 14 alcoholics had biologic parents that were also alcoholic, again depicting the familial relevancy of alcoholism. Also, half of the alcoholic group reported being hyperactive as youths compared to only 15% of the controls - a statistically significant finding. Similar results were noted for truant and antisocial behavior (21% vs 2%), aggressive, impulsive, hot tempered behavior (50% vs 18%), and disobedience to parents and teachers (29% vs 4%). Again these data point to a link between poor behavioral outcome for hyperactive sons of male alcoholics. The results of this study indicate a strong hereditary component of hyperactive behavior in conjunction with Type II familial alcoholism. Similar alcoholics and their sons are the focus of the present study.

These findings when examined in conjunction with one another, suggest that alcoholism and hyperactivity have definite genetic components, and that these traits are further exacerbated by parental alcoholism and an inadequate or dysfunctional social/emotional rearing environment. Goodman (1989) has aptly pointed out that adoption studies suggest that "cross generational continuities reflect genetic rather than cultural transmission" (pp. 189) of hyperactivity and its associated behavioral regime. Other relevant findings relating to the heritability of hyperactivity in conjunction with alcoholism will be reviewed in more detail in subsequent sections.

Family Studies and Self Reports of Alcoholics

However interesting and provocative electrophysiological, twin, and adoption study findings may be, possibly the most convincing evidence for an heritable basis for hyperactivity in association with alcoholism is the history of alcoholics themselves. Many investigations reveal that a large majority of parents who report having been hyperactive

and attentionally deficient as children are currently psychiatrically ill with specifically high prevalence rates of alcoholism, antisociality, and other affective disorders, further suggesting a familial relationship to hyperactive behavior in conjunction with poor adult outcome (Cloninger et al., 1985). Self-reports from alcoholic samples reveal high incidence of childhood hyperactivity in adult alcoholics, while at the same time their offspring are also showing evidence of similar behavior (Fitzgerald et al., 1990; Goodwin, Schulsinger, Hermanse, Guze & Winokur, 1975; Morrison & Stewart, 1970; West & Prinz, 1987).

Morrison and Stewart were among the first to make the association between alcoholism and hyperactivity in this manner. In an early study Morrison and Stewart (1970) interviewed the parents of 59 "hyperactive" children and 41 "non-hyperactive" children. They found twice the incidence of alcoholism in the parents of the hyperactive children (20%) compared to the non-hyperactives (10%). Twenty percent of the parents of hyperactive children reported having been hyperactive as youths, and half of these were alcoholics at the time of the interview. The Goodwin et al. study (1975) reported similar findings in the group of Danish men. Fifty percent of the alcoholic men in their sample reported having been hyperactive and impulsive as youths. In a 10 to 15 year follow-up study of sons of alcoholics, Knop (1985) found significant differences between the high and low alcohol risk groups for impulsivity, restlessness, and verbal deficiency. The sons of alcoholic fathers from this cohort exhibited higher rates of hyperactive behaviors, specifically impulsive and restless behavior, than did the sons of non-alcoholic fathers. According to Knop, this finding may be of predictive importance in the etiology of alcoholism. Findings from the Gittelman et al. study (1985) reviewed earlier also revealed

that boys diagnosed with ADDH in childhood, who maintained the disorder into adolescence, reported significant amounts of delinquency, antisociality, and substance abuse.

In some of his earlier work done with hyperactive children, Cantwell (1972) studied the fathers of 50 hyperactive boys between the ages of 5 and 9 years, comparing them to fathers of 50 normal boys of the same age. He found twice the incidence of alcoholism in the fathers of hyperactive boys (30%) compared to the fathers in the control group (14%). He also found a greater degree of antisocial behavior in fathers of hyperactive boys. In that antisocial behavior was statistically higher in the alcoholic group, this lends support to the notion that hyperactive behaviors may carry over into adulthood, revealing themselves as aggression and antisociality (Blouin, Bornstein, & Trites, 1978; Loeber, Lahey & Thomas, 1991; Weiss & Hechtman, 1986).

One study that looked at the frequency of psychiatric disorders in sons of alcoholics found prevalence rates of ADHD, CD, and ODD to be higher in children who had either one or both parents who were alcoholic compared to those who had no parental alcoholism in their family (Earls, Reich, Jung & Cloninger, 1988). In this study the authors looked at psychopathology in children as it exists in the antisocial, alcoholic environment (i.e., Type II alcoholism). They found no significant differences in psychopathology when comparing children of alcoholic parents and children of antisocial parents. However, childhood psychopathology was 2 to 3 times greater in families where there was a parent(s) who revealed both alcoholism and antisocial personality compared to families where there was neither parental alcoholism or antisocial personality. These findings suggest that antisocial personality many times coexists with alcoholism, and

furthermore, the authors insist that it is the combination of alcoholism and antisocial personality that predispose the offspring of alcoholics to psychopathology and possibly to alcoholism. Remember, one of the primary hypotheses of this study is that the comorbidity of hyperactivity and conduct problems is primarily responsible for poorest outcome in SOMAs. Similar findings as those in the Earls et al. study were found by Gittelman et al. (1985). In this study the authors reported that boys comorbid with hyperactivity and antisocial behavior who maintained the problem behaviors into adolescence, progressed into more severe forms of psychopathology at follow-up (e.g., alcohol and drug abuse).

Zucker, Ellis, & Fitzgerald (1993) found that sons of male alcoholics were significantly more problem behaviored (externalizing and internalizing behaviors) than sons of male non-alcoholics. When they administered behavioral tasks of impulsivity (delay of gratification task) to boys in their study, they found that SOMAs were significantly more impulsive than SOMnAs, thus revealing an increased inability for alcoholic offspring to monitor or regulate their behavioral expression. When the authors divided the alcoholic group based on paternal level of antisociality they found that SOMAs with highly antisocial fathers displayed significantly more problem behaviors (including hyperactivity) than SOMAs of non-antisocial fathers and controls. These findings led the authors to agree with Earls et al. that the cumulative effects of alcoholism and antisociality further increase risk load for COAs.

In the previously reviewed Jansen et al. (under review) study, the authors investigated the temperament attributes of SOMAs based on incidence of clinical problem behaviors. They found that the incidence of difficult temperament traits consistent with

ADHD (high levels of physical activity and distractibility) were significantly more apparent in boys who were clinically problem behaviored compared to non-clinically problem behaviored boys. Also, parental level of alcohol related problems and antisociality was significantly higher in those boys with temperament characteristics consistent with ADHD symptomatology. Such findings are compatible with the extant literature which strongly suggests that children being reared in an alcoholic environment are often times hyperactive, distractible, and impulsive (Cloninger et al., 1988; Zucker, 1987).

It must be reiterated here that the current developmental perspective of delinquent, aggressive personality characteristics suggests that hyperactivity is antecedent to them (Farrington, Loeber & van Kammen, 1990). Problematic outcomes seem to follow a progression from minimal to maximal degrees of presentation, at the same time poor outcome runs parallel to the expression of hyperactivity and conduct problems and their degree of comorbidity. In other words, children with only hyperactive symptoms seem to have more favorable outcomes than children with only conduct problems, and children with only conduct problems have more favorable outcomes than children comorbid for both disorders. As the degree of severity of poor behavioral outcome increases one can see greater degrees of overlap (i.e., comorbidity) for the disorders and consequently more adverse developmental outcome.

Contradictory Findings

It is the perception of behaviors by alcoholic parents that has been linked conceptually to the etiology of alcoholism. For instance, activity levels for children at low risk for alcoholism seem to be related to demographic variables (Fitzgerald et al., 1993; Ham et al., 1993), in particular SES, family income, and family occupational status,

whereas activity levels for children at high risk for alcoholism are many times related to paternal alcoholism (Rutter et al., 1990; Tarter et al., 1984) and antisocial behavior (Ham et al., 1994; Noll, Zucker, Fitzgerald, & Curtis, 1992). However, there are a few studies that report contradictory findings. For example, Tarter et al. (1985) found only a weak link between alcoholism and hyperactive behaviors in two groups of adolescent SOMAs when they controlled for conduct problems, i.e. both groups were classified as conduct disordered. One group was at high risk for alcoholism (having a father who was alcoholic) and the other group was at low risk (the father being non-alcoholic). No significant differences were found in hyperactive symptomatology between high and low risk groups. In that the two groups did not differ on hyperactive behaviors, the authors concluded that although hyperactivity has been associated with higher risk for alcoholism, it does not directly influence one to becoming alcoholic more readily than other pathologies particularly when in the presence of conduct problems.

In the Tarter et al. study, however, hyperactive sons of alcoholics did perform more poorly on several neuropsychological tests measuring attention, reading comprehension, memory, and perceptual-motor coordination and speed than hyperactive boys of non-alcoholic fathers. These findings suggest that neuropsychological deficits noted in chronic alcoholics may actually precede alcohol onset[3]. They also seem to be stable characteristics of hyperactive SOMAs even in the presence of aggressive conduct problem behavior. Furthermore, I believe that high levels of alcohol use serve to substantially exacerbate the neurologically related deficits in the alcoholic. As SOMAs develop and begin to use alcohol, this will also exacerbate their neurologically mediated propensity for dysregulated behavior.

Schuckit, Sweeney and Huey (1987) obtained similar contradictory findings as the 1985 Tarter et al. study. They compared a group of young adult sons of alcoholics to a group of same aged sons of non-alcoholics of like sociodemographic status, examining their levels of childhood and adult symptoms of hyperactive behaviors. Inasmuch as no significant differences in levels of hyperactivity in childhood or adulthood were found, Schuckit et al. concluded that hyperactivity does not play a causal role in the etiology of alcoholism.

In summary, although several researchers have found contradictory findings, more than not, the findings are due to inadequate sample size. In the two studies mentioned above this was the case. Still, many investigators consistently report an association between alcoholism and hyperactivity. Behavioral attributes such as high activity levels, impulsiveness, and poor concentration appear frequently in high risk offspring of alcoholics (Cloninger et al., 1985; Goodwin et al., 1975; West & Prinz, 1987; Workman-Daniels & Hesslebrock, 1987). Although these findings are offered as support for the heritability of hyperactivity in conjunction with parental alcoholism, it was beyond the scope of the present study to address in any direct manner a genetic component for the etiology of hyperactivity and/or alcoholism. However, the study *did* investigate the genetic attributes of hyperactivity and related behavior in the alcoholic environment using theoretically driven data models. The focus of the current study was the psychopathology in children of antisocial, alcoholic parents, in particular hyperactive, hyperactive related conduct problems, cognitive development, and several possible contextual etiologic factors. The behavioral abnormalities under investigation (e.g., hyperactivity, attentional deficits, aggression etc.) may reflect a behavioral dysregulatory mechanism in SOMAs that

may precede the onset of alcoholism as well as play a role in the later expression of other psychopathological behavior (e.g., antisocial behavior, drug abuse). The dysregulatory mechanism discussed here may in fact be an heritable one, however, further and extensive study will need be done to ascertain the veracity of such an hypothesis.

Cognitive Functioning and Hyperactivity in Children of Alcoholics

Neuropsychological and intelligence studies in alcoholism research have found that a significant number of children at risk for alcoholism and alcohol related disorders are cognitively impaired. Moreover, "hyperactive" COAs are considered to be at an even further increased level of risk for poor cognitive outcome (e.g., Hesselbrock, Stabeneau & Hesselbrock, 1985). Although overall IQ scores are generally within normal ranges for hyperactive children (Baxley and LeBlanc, 1976), deficits have been found in abstract relations tasks, verbal and non-verbal memory tasks, and perceptual-spatial motor skills for COAs (Tarter, Jacob & Bremer, 1989; Workman-Daniels & Hesselbrock, 1987).

In the West and Prinz (1987) review of the alcoholism literature they report on several studies that consistently found lower levels of cognitive functioning in COAs. Their overall findings can be summarized as follows: Children of alcoholics reveal 1) "moderate adverse effects" regarding school performance; 2) high occurrence rates of hyperactivity, delinquency and truant behavior; and 3) these dysfunctional behaviors are seen in association with poor school performance outcomes. Six of the nine IQ studies reviewed found significant IQ differences between children of alcoholic parents and children of non-alcoholic parents. One study found only significant IQ differences for boys. Five of the six studies investigating academic performance reported significantly lower academic performance in COAs. Additionally, one study revealed that children of

antisocial, alcoholic parents had the lowest grade point and highest percentage of grade repeats (Schukit & Chiles, 1978, cited in West & Prinz, 1987). A majority of COAs fail to finish high school even though many times teachers and counselors feel they are capable of better schoolwork. Perhaps poor schoolwork is resultant of the child's inability to sustain attention, remain free from distraction, etc. - this would certainly foster poor academic outcome.

In the Zucker et al. (1994) study previously mentioned, the authors also investigated cognitive functioning in SOMAs and SOMnAs using Stanford-Binet IQ tests. Initial findings revealed significant disparity of IQ scores between SOMAs and SOMnAs. Specifically, SOMAs exhibited average scores ($\underline{x}$ = 101), but SOMnAs scored on average 7 points higher ($\underline{x}$ = 108). When they divided the group of SOMAs by level of paternal antisociality (i.e., one group of SOMAs having fathers with clinical levels of antisocial behavior the other had fathers with sub-clinical levels) they found that the SOMAs with antisocial fathers accounted for the lower IQ scores ($\underline{x}$ = 100) compared to the other two groups ($\underline{x}$ = 107 for non-antisocial SOMAs and $\underline{x}$ = 108 for controls). These findings continue to suggest that the antisocial environment is possibly more responsible for detrimental outcome in SOMAs rather than the alcoholic element, or else the comorbid status is the culprit.

Tarter and his colleagues have conducted numerous studies investigating overall cognitive functioning and neuropsychological outcome in children of alcoholics, particularly SOMAs (Tarter et al., 1984; Hegedus, Alterman & Tarter, 1984; Tarter et al., 1985). In a recent study Tarter, Jacob and Bremer (1989) looked at the offspring of early onset alcoholics (n = 16) and late onset alcoholics (n = 17). This study also included a

normal control group whose parents were referred to as social drinkers (n = 30), and another control group with depressed fathers (n = 29). Children in all of the groups were biological sons of the above groups of men, and all of the boys were between the ages of 8 and 17. A variety of standard neuropsychological tests were administered, including a Wechsler intelligence test. Study results indicate that sons of early onset alcoholics reveal specific cognitive deficits compared to normal boys. Sons of early onset alcoholics exhibited lower verbal IQ ability and had greater problems with inattention. Although no significant differences were found for other neuropsychological tests in this study, the authors point out that fathers were community recruited alcoholics compared to clinic drawn alcoholics (as is the case in many similar studies) and thus, the non-significant findings may be due to the "non-chronic" nature of the alcoholics. Also, none of the fathers in this study met research diagnostic criteria for antisocial personality disorder (APS). This being the case, the authors further postulate that the lack of significant neuropsychological differences found may be a result of the lack of comorbidity seen in the sample. The Zucker et al. (1993) results attest to this assumption also.

In another study from the same longitudinal data set, Tarter et al (1993) looked at differences between two groups of boys - one group (n = 37) had fathers who were alcoholic and the other group (n = 48) consisted of sons of non-alcoholic fathers. All boys were between the ages of 12 and 17. In this study, neuropsychological tests of verbal intellectual capacity, basic educational achievement skills, memory, and attention capacity were administered to the boys. Results indicate that SOMAs performed significantly lower on verbal intellectual levels, had significantly lower scores for educational achievement, were less attentive, and showed significant memory differences compared to

normal boys.

These are but a few of the studies that reveal SOMAs to be at significantly greater risk for poor cognitive, academic, and neuropsychological outcome. However, not dealt with in detail in the above review (except for the Zucker et al. [1993] study) is the fact that boys growing up with an alcoholic, antisocial father seem to fare even worse on such measures than sons of only alcoholic men. It seems that the cumulative effects of the antisocial-alcoholic environment fosters a disproportionate propensity for poor cognitive outcome. This is also in accord with Earls et al's. (1988) hypothesis that suggests it is the comorbidity of the situation that is causally related to the most severe offspring outcome rather than one or the other of the pathological states. This was of primary consideration in the present study.

The model tested in the present study assumed not only a direct genetic transference of intelligence from parent to child (as measured by IQ assessments), but also a mediational component of intelligence arising from influences of the impoverished sociocultural rearing environment. Psychometric intelligence in humans has yielded several key findings which allow us to test this theory (Turkheimer & Gottesman, 1991), and to consequently make several assumptions: First, moderate linear relationships have been found to exist between parental genotype and offspring intelligence; second, a "severely" deprived rearing environment has a "powerful" impact on child cognitive capacity; and third, very small environmental (i.e., experiential) effects are seen in the ranges of environments in intact families. Therefore, in the present study the model was tested with a fair amount of confidence in finding that parental and offspring intellectual abilities are directly involved with one another in the expression of dysregulated behavior.

And furthermore, it was hypothesized that the greater the impoverishment of the rearing environment, the greater will be the level of poor intellectual outcome.

Conclusions

Based on the previous review, the association between risk for alcoholism/abuse and alcohol related psychopathology, problem behavior attributes characteristic of ADHD, and neuropsychological and cognitive disorders can readily be seen. Moreover, it seems evident that a male child of an alcoholic father (i.e., SOMA) is likely to exhibit some or all of these attributes, and that these attributes might possibly be the predisposing factors leading to the subsequent development of alcoholism and alcohol related pathology.

The behavioral dysregulatory theory tested here is reminiscent of Tarter and his associates' (Tarter, Laird & Moss, 1990) neuropsychological model and Begleiter's dysregulation theory (Holden, 1991). These theories have attempted to theoretically and empirically address some of the issues presented here relating to behavioral dysfunction in COAs. Both theories are attempts at elucidating some of the vulnerability components of alcoholism. Similar to the Tarter et al model, the model investigated the offspring of early onset, antisocial alcoholics who present severe manifestations of alcohol symptomatology. Thus, the primary focus of the model was really an attempt at explaining some of the behavioral underpinnings of Cloninger's (1987) Type II alcoholics and their offspring as well as Zucker's (1987) antisocial alcoholic.

As previously mentioned, few studies have looked at the developmental occurrence of hyperactivity in a high risk alcoholic population (except in retrospective fashion). And certainly very few of these studies have looked at SOMAs from community samples, nor have many investigated the occurrence of such characteristics as early as the

preschool years. Also, most studies have not investigated the offspring of *young* alcoholics (i.e., alcoholics of early onset). It was interesting to note the incidence of hyperactivity and conduct problems in older male infants and toddlers of young, community recruited, antisocial, early onset alcoholic fathers rearing sons in impoverished conditions, compared to a demographically similar group of community recruited, non-antisocial, non-alcoholic parents. But, it will be even more interesting to follow the developmental progress of these boys into late childhood and adolescence as our overall study progresses, tracking their behavioral and cognitive outcomes as development unfolds.

To summarize the uniqueness of the present study: First, the primary interest was the etiologic issues of pre-alcoholics from the earliest point in time possible with respect to the expression of hyperactivity and hyperactivity related conduct problems. Thus, vulnerability to behavioral dysregulation in pre-school aged SOMAs was of primary importance in the present investigation. Second, this study was comparing results of SOMAs with a demographically comparable group of boys whose parents are asymptomatic for alcoholism and alcohol related psychopathology. Third, both groups of alcoholics and non-alcoholics under investigation were community drawn, rather than clinic drawn, and both were relatively young at the time of data collection. The present study was thus attempting to test a biobehavioral dysregulation model in a unique and carefully recruited sample of boys at heightened risk for alcoholism.

The basis for determining whether a child was hyperactive (HADI) or severely conduct problemed was based on DSM criteria (DSM-III-R, 1987; DSM-IV, 1993). Although a draft of DSM-IV was available at the time of this writing (Edith Gomberg,

personal communication, September, 1993) no significant changes have been made concerning ADHD, with the exceptions noted above. However, a complete preliminary draft of DSM-IV section on ODD and CD (Rolf Loeber, personal communication, June, 1993) was used to assess conduct problems reminiscent of ODD and CD, as some changes have been made since the DSM-III-R version. The relationship of hyperactive and non-compliant, antisocial behaviors was observed with respect to levels of risk for parental alcoholism and antisocial behavior and the ability of the parents externalizing psychopathology to predict their own child's externalizing psychopathology.

CHAPTER II

Study Goals and Specific Hypotheses

This study was designed to examine several biopsychosocial attributes of SOMAs and their parents in order to evaluate their etiologic influence on the expression of hyperactivity and conduct problems. It wad also an attempt to determine whether one or all of the problem behavior patterns under study are dominant behavioral characteristics of boys at risk for the later development of alcoholism and other alcohol related pathology compared to demographically matched controls. The controversial issue of overlap of hyperactivity and conduct problems was also of concern in the present study. With greater precision an attempt was made to define the behavioral categories and to observe their overlap of expression.

Three groups of boys were looked at in the present study (see Subjects section for complete information on sample compilation). The three groups consist of two alcoholic groups (based on paternal diagnosis of alcoholism and antisociality, i.e., Zucker's antisocial alcoholic) and a comparison group. The first alcoholic group was composed of SOMAs and their parents, fathers (and a good majority of mothers) of whom are highly involved in antisocial activities. The second alcoholic group consisted of SOMAs and their parents, fathers of whom are significantly less involved in antisocial behavior. The comparison subjects were boys and their biologic parents, parents of whom are asymptomatic for substance abusing problems and clinical levels of antisociality. The three groups of boys and their parents were from similar census tracts in the tri-county area surrounding Michigan State University, parents and sons are similar in age across

groups.

Several general findings were expected. First of all, it was expected that boys being reared in alcoholic families would exhibit higher levels of hyperactive behaviors, while simultaneously exhibiting lower levels of overall cognitive functioning. This assumption of the model suggested that behavioral and cognitive problems in these boys is most likely due to a neurologically mediated dysfunctional condition (neuropsychological), one that is familial/genetic in nature but one that is moderated by the suboptimal rearing conditions that have been found in the present sample. Second, boys being reared in the **antisocial**, alcoholic families were expected to exhibit similar aggressive, antisocial behaviors as their parents in conjunction with high incidence of hyperactive behaviors and cognitive dysfunctioning. This suspected high degree of behavioral overlap in this group of SOMAs suggests that their behavioral dysregulation is due to an inherited condition that gives one a general propensity for behavioral dysregulation (i.e., an inability to monitor physical, social, emotional etc. behaviors) that is fueled by the extremely chaotic, abusive environment these kids are being reared in.

Although the defining characteristics of the groups of boys, as well as the strategies for analysis, are primarily based on the father's pathology, I also investigated maternal characteristics within and across groups.

The specific hypotheses that tested using ANOVA (MANOVA and Oneway ANOVA) were as follows:

Hypothesis I

Boys being reared in the two groups of alcoholic families (**antisocial** and **non-antisocial, alcoholic**) families will exhibit: a) higher levels of hyperactivity (HADI)

(Conners); b) lower IQ scores (Stanford-Binet); and c) lower (i.e., delayed?) levels of motoric, language, and overall cognitive development (RYDS), compared to boys in the **control** group.

Hypothesis II

Boys in the **antisocial, alcoholic** group will exhibit: a) higher levels of hyperactive (HADI) (Conners); b) lower IQ scores (Stanford-Binet); and c) lower (i.e., delayed?) levels of motoric, language, and overall cognitive development (RYDS), compared to boys in the **non-antisocial** alcoholic group and the **control** group.

Hypothesis III

Boys being reared in **antisocial, alcoholic** families will exhibit higher levels of conduct problems (Conners) than the other two groups of boys, and SOMAs in the **non-antisocial** group will likewise exhibit higher levels of the same behavior compared to control boys. In other words, there will be a progression in symptomatology as level of risk increases.

Hypothesis IV

Higher incidence of hyperactive behaviors (DOTS-spouse) will be seen in alcoholic parents compared to non-alcoholic parents. Also, parents in the **antisocial, alcoholic** group will display higher levels of HADI symptomatology than the other two groups. In other words, levels of hyperactive symptomatology in parents will increase in accordance with level of externalizing psychopathology.

The final two hypotheses will be tested via correlational analysis:

Hypothesis V

Lower levels of SES, parental education, and family income will be highly

associated with hyperactivity and conduct problem behaviors.

Hypothesis VI

Parental externalizing psychopathology, parental intelligence, parental hyperactive symptoms, and contextual variables will be found to be collectively involved in a causal relationship with biobehavioral dysfunction (HADI, poor neurocognitive functioning, and conduct problems) in SOMAs.

Hypothesis VI was specifically tested using structural equation modelling techniques. There are several very good theoretical and empirical reasons for using structural equation modelling techniques. First, they allow us to combine familiar analytic techniques that investigate singular and multivariate phenomenon (e.g., ANOVA, Regression Analysis, Confirmatory Factor Analysis, etc.). They also "allow us to deal with *simultaneous* examination of the internal validity of psychological constructs and the external validity of their system of inputs and outputs" (McArdel, Hamagami & Hulick, 1992, pp. 1), and give us the opportunity to deal with investigations involving multiple variables, some of which are unobserved (Loehlin, 1987). Other specific benefits of using structural equation modeling with latent constructs are: 1) they offer the opportunity to investigate the validity of a formal theory in a "precise form" and 2) with them one can test hypotheses about **unobserved** variables and how they interact with observed measurements of such phenomenon (McArdel et al., 1992). (This was germane to the present study in that certain assumptions were made concerning certain neurobehavioral functions using instruments with fairly limited actual neuropsychological qualities. Parental questionnaire data was also being used that assesses behaviors which "reflect" neurological functioning but that do not necessarily observe the neurological

underpinnings of those behaviors.)

Buss and Plomin (1984) also point out some of the unique benefits of structural equation modelling. They point out that such techniques allow one to: 1) examine data simultaneously, while 2) making explicit assumptions about the data, as well as 3) testing several theoretical models at one time, and 4) they provide goodness of fit indices which permit ease of interpretation. McArdel and colleagues sum up this type of analysis as follows: "The current ... techniques allow the simultaneous estimation of the coefficients of internal and external validity and provide a formal statistical basis for testing the goodness of fit between (hypothetical) model and data" (pp. 3). Thus, using this technique one can test theoretical ideas with available data and ascertain whether or not the two are similar or dissimilar to any significant degree, and thus conclude a theoretical/empirical model to be construct valid or not.

CHAPTER III

Method

Design and Procedures

The present study is a subsidiary study of the Michigan State University-University of Michigan Longitudinal Study (MSU-UMLS). The specific aim of the MSU-UMLS is to "...trace the development of children who come from homes with alcoholic, drunk-driver fathers, and who therefore are statistically at high risk for problems involving aggression, negative mood, failures in persistence, difficulties in academic performance, and problems interacting with other family members" (Zucker, Noll, & Fitzgerald, 1986). These children are also at increased risk for the later development of alcoholism since approximately 25% to 35% of sons of male alcoholics (SOMAs) will themselves become alcoholics, and a portion of the rest will have difficulties with drinking behavior (Cloninger, 1988). The male child between 2 and 7 years of age from these alcoholic families make up the high risk group in the study; they were contrasted with a same age group of males considered to be at low risk for alcoholism and alcohol related problem behavior. When possible, control subjects are drawn from the same census tracts as the high risk group.

The predictive framework of the MSU-UMLS is designed to be consistent from childhood to adulthood. The dysfunctional characteristics of the children under study are presumed to be the "etiologic variables for later alcoholic outcome" (Zucker & Fitzgerald, 1991). Specific characteristics as set forth by the principal investigators are: 1) aggressive and hyperactive behaviors, 2) negative mood, 3) genetic loading for alcoholism, 4)

problematic social relationships (between parent and child, and child and sibling for the children; between parents, and between parent-child for the parents) which, as development progresses, enhance the characteristics of (1) and (2) above, and 5) a more elaborated, and earlier developed cognitive structure about alcohol and attitudes towards alcohol.

The specific criterion sought for in the present study was hyperactivity (and its overlap with conduct problems) as perceived by fathers and mothers at differing levels of risk for alcoholism and alcohol related pathology. The predictors of hyperactivity were 1) problems related to drinking, 2) parental antisociality, 3) parental intelligence, 4) parental hyperactivity symptomatology, and 5) specific demographic characteristics, namely, level of parental education, SES, and family income. The purpose of the investigation was fourfold in nature: 1) to isolate a constellation of abnormal behaviors that, in keeping with current research findings and diagnostic criteria, constitute ADHD, ODD, and CD as they specifically relate to risk for alcoholism and alcohol related psychopathology, 2) an attempt to further delineate the hyperactive syndrome as it may or may not exist in an aggressive, non-compliant child, 3) to observe conduct problems as they interact with the expression of hyperactivity in a group of boys at high risk for alcoholism and related psychopathologies, and 4) to observe several parental variables that are suspected to be casual agents in the expression of biobehavioral dysregulation in SOMAs; high levels of hyperactive symptomatology and low neurocognitive functioning being the indicators of biobehavioral dysregulation.

Utilizing several parental report instruments that target the specific behaviors indicative of ADHD, ODD, and CD, it was felt that a more valid and timely statement

could be made as to the behavioral status of the children under study and further, a more narrowly defined status of the currently sought after problem behaviors. However, it is impossible to validly establish the predisposing factors of behavioral and/or biological problems in a completely retrospective fashion. The most efficient means of attaining this type of information from any given population is to look at high and low risk groups prospectively, monitoring their biobehavioral states. Therefore, behaviors **indicative** of ADHD, namely impulsivity, attention span difficulties, distractibility, and locomotor activity as an index of "hyperactivity" (collectively referred to as HADI), and those found in ODD and CD (collectively referred to as conduct problems) were looked at in a group of children at high risk for various alcohol related adulthood externalizing psychopathologies (e.g., alcoholism and antisociality). These were compared to a group demographically similar children who are not presently thought to be at high risk for alcohol related pathology. Data utilized in the present study were archival data that have been collected prospectively over the past ten years.

The study was a three (Risk: highest risk [sons of antisocial, alcoholic fathers], high risk [sons of non-antisocial, alcoholic fathers], low risk [sons of non-antisocial, non-alcoholic fathers]) by two (Parent: father, mother) between subjects design (see Subjects section for more detail). Boys at highest risk for the later development of alcoholism and poor behavioral outcome are so defined by alcoholic fathers who exhibit high levels of antisociality; the majority of these boys have a normal alcoholic consuming mother (i.e., mother is not alcohol abusive or dependant) but most mothers are fairly high on measures of antisocial behavior similar to their mates. Boys at high risk for alcoholism have an alcoholic father, but the father has levels of antisocial behavior that are considered "non-

clinical". The low risk group are so defined by fathers who do not abuse alcohol (or other substances) and who are not antisocial.

Subjects

Subjects are a group of community recruited 2 to 7 year old boys (N = 301) and their parents from a four county area surrounding Michigan State University in East Lansing, MI. In the overall project (MSU-UMLS), subjects are divided into two groups of risk based upon paternal diagnosis of alcohol related problems. One group of boys is considered to be at high risk for alcoholism as a result of having a father (and in some cases a mother) who is alcoholic; this group is referred to as the **High Risk Group** (n = 210). The other group is considered to be at low risk for alcoholism, having neither parent who exhibits signs of alcoholism; this group is referred to as the **Control Group** (n = 91). Boys in this study come from an intact family at the time of recruitment. Boys in the high risk group are from similar, if not the same, census tracts as those in the control group, but control parents, unlike risk parents, are asymptomatic for alcohol or other substance abuse problems.

During the course of recent other analyses on the MSU-UMLS (Ellis, 1993; Zucker et al., 1993), it has proven profitable to evaluate family, parental, and offspring functioning based on level of antisociality in conjunction with alcoholism, particularly the father's levels of antisocial behavior. Therefore, the High Risk Group was also subdivided into two groups based upon paternal scores of antisocial behavior. In one group fathers are alcoholic and very antisocial (n = 77); this group is referred to as the **Antisocial, Alcoholic Group (AAL)**. In the other high risk group fathers are alcoholic but not high on antisocial behaviors (n = 133); this group will be referred to as the **non-Antisocial,**

Alcoholic Group (nAAL). Groups are based on a cutoff score from the Antisocial Behavior Checklist (Zucker et al., 1993); a score of 24 or higher is indicative of a level of antisociality necessary to make a DSM-III-R diagnosis of Antisocial Personality disorder (APS) with a sensitivity of .85 and specificity of .83 (Zucker et al., 1994).

Alcoholic fathers have been recruited via the district courts in the Mid-Michigan area. Using a population net involving four adjacent counties with six district courts, all convicted drunk drivers with a blood alcohol concentration (BAC) of 0.15 percent or higher (or 0.12 percent or higher if this was a second or more documented drinking related driving problem) who have a biological son between the ages of 2.0 and 7.0 years currently living with them and who are from intact families at the time of first contact, were recruited into a study of "child development and family health." Probation officers from the district courts request permission to release names and phone numbers to the project. When contacted by project staff, respondents are told that the study has no connection to the courts and that all information collected is confidential. Of the total number of men contacted by probation officers (n = 263), 78% (n = 198) agreed to have their name and phone number released to the project; of these, 86% (n = 150) agreed to participate. The remaining alcoholic families (n = 60) were recruited via a different method (see below).

After a high risk family is recruited into the study, a matched community control family whose parents are neither alcoholic nor drug dependent is located using door-to-door canvassing interviews. Canvassers begin a door-to-door search one block away from the alcoholic family, staying within the same census tract, and screening for an age appropriate (+/-6 months match) male child in a non-alcoholic home. To date, 18,989

families have been contacted. Of the 509 families with an age-appropriate male child, 475 of these were contacted and 475 agreed to participate. Thirty-four later refused participation; 215 families were ineligible due to ethnicity, SES, or parentage (i.e., were non-biological parents); 60 were contacted and recruited as control families but later were found to make an alcoholic diagnosis therefore they were assigned to the alcoholic group; and 91 were successfully recruited as control subjects for the entire longitudinal study.

Later data collected as part of the longitudinal protocol insures that each district court father meets Feighner diagnostic criteria (Feighner, Robins, Guze, Woodruff, Winokur, & Munoz, 1972) for probable or definite alcoholism, and that both parents in the control family do not make this diagnosis or one of drug dependence. Maternal alcoholism among the high risk families is neither a criterion for inclusion nor exclusion from the study. However, in accord with study screening criteria, no child manifested characteristics required for a diagnosis of fetal alcohol syndrome, i.e., prenatal and/or postnatal growth retardation, apparent central nervous system involvement, and/or characteristic facial dysmorphology (Sokol & Clarren, 1989).

Community canvassing to obtain control families was used to control for effects of age and sex of target child, community influences, and as an approximate control for SES. This procedure allows findings from the families with an alcoholic father to be contrasted to an ecologically comparable but non-alcohol/drug abusing population. All families in the study are paid for their participation.

In summary, this sample is very unique in several ways with respect to similar studies. First of all, this is a community derived sample rather than a clinically derived one. Therefore, results of this study are more readily generalizable to similar

demographically composed populations. Second, this study is using a demographically comparable control group that is asymptomatic for the study criterion variables (i.e., alcoholism). Third, the age of our target children (3 years to 6 years $\pm$ 6 months) is considerably younger than similar studies. (The boys in this study represent late infancy through early childhood - years of great vulnerability to environmental influences.) Fourth, the entire intact nuclear family is studied in great depth. Over eighty instruments are used to assess an array of biopsychosocial issues. Finally, this study is, for the most part, prospective in nature. We have been collecting longitudinal data every three years for an extended period of time; data is currently being collected for Wave 2 and Wave 3 for most families. In the present study, however, only data from the first wave of data collection were used.

All families participating in the project complete numerous questionnaires, interviews, and direct observation sessions. Data collection takes place across 9 sessions, requiring approximately 15 hours for each parent and 7 hours for each target child. All data are gathered at the participant's homes with the exception of a video taped session conducted at university facilities (this video session is not part of the present analyses).

Instruments

Parental Instruments

Several aspects of the parent's past and present behavior and life circumstances were measured and compared across groups to see which parental pathology exists in the hyperactive milieu and which are the best predictors for such problem behavior. Specific parental characteristics to be assessed are: 1) problems related to alcohol abuse/dependence, 2) antisociality, 3) adult hyperactive behavior, 4) cognitive functioning,

and 5) contextual/environmental factors. These characteristics are measured respectively by the Lifetime Alcohol Problems Score (LAPS; Zucker, 1991), the Antisocial Behavior checklist (ASB; Zucker, et al., 1993), the Dimensions of Temperament Survey for spouse (DOTS-Spouse; Lerner, Palermo, Spiro & Nesselroade, 1982), the WAIS-R (Wechsler, 1981), and a demographic questionnaire.

Demographic Questionnaire. This questionnaire is administered during the first visit to the families; it inquires about self-reported background information (occupation, education, income, years married, number of children in the house, age, etc.) and family of origin (SES, education, etc.). This instrument provides the data from which demographic items and information about family income etc. are coded. The SES of each parent is established using the occupation based Revised Duncan Socioeconomic Index (TSEI2; Steven's & Featherman, 1981).

Lifetime Alcohol Problems Score. The LAPS (Zucker, 1991) is the primary drinking variable used in the current study. The score is designed to assess differences in the extent of drinking problems over the life course, and is derived from information gained from the administration of the Drinking and Drug History interview (Zucker, Fitzgerald, & Noll, 1990), the Diagnostic Interview Schedule (Robins, Helzer, Croughan, & Ratcliff, 1980), and the short form of the Michigan Alcoholism Screening Test (SMAST) (Selzer, 1971, 1975). The LAPS provides a composite score derived from three component subscores: (a) the primacy component, involving the squared inverse of the age at which the respondent reported first drinking enough to get drunk; (b) the variety component, involving the number of areas in ones lifetime in which drinking problems are reported, and (c) the life percent component, involving a measure of interval

between most recent and earliest drinking problems, corrected for current age. Scores are standardized separately for males and females within our project sample. This measure is unrelated to current drinking consumption in problem drinking samples and has been shown to be a valid indicator of differences in long term severity of drinking difficulty in a wide variety of areas (Zucker, 1991). The LAPS yields a continuous score with a base of 10. Individuals scoring above 10 are those individuals who have experienced significant amounts of problems in direct association with their drinking patterns.

Antisocial Behavior checklist (ASB). The ASB (Zucker, Noll, Ham, Sullivan, & Fitzgerald, 1993) is a 46-item revision of an earlier antisocial behavior inventory utilized in the Rutger's Community Study (Zucker & Barron, 1973) that has been modified so that items are also salient for adult antisocial activity. A series of reliability and validity studies with populations ranging from college students to prison inmates has shown excellent test retest reliability (.94 over four weeks) and internal reliability (coefficient α, range .67 to .93) (Ham et al., 1994). Concurrent validity was found using Pearson's correlations for alcohol related problems ($r(315) = .54$, $p < .00$) and a measure of hostility ($r(409) = .43$, $p < .00$). The ASB also differentiates between groups of people with varying degrees of antisocial behavior such as prison inmates versus minor offenders in district courts versus college students, and between alcoholic and non-alcoholic adult males. Self reported lifetime antisocial behavior is rated on a 4-point scale of never = 0, rarely = 1, sometimes = 2, or often = 3. The total score is derived by summing across all items; a total score of 24 or greater is indicative of Antisocial Personality disorder (APS) based on DSM-III-R diagnostic criteria.

Dimensions of Temperament for Spouse (DOTS-Spouse). The DOTS utilized in

this study is the 34-item scale constructed by Lerner, Palermo, Spiro, & Nesselroade (1982) in order to provide a continuous measure of the primary components of temperament from late infancy to adulthood. This instrument was used in the present analyses to look at parental characteristics of temperament. Each parent assesses perceptions of their current spouses temperamental characteristics. This measure yields five domains or dimensions of temperament: 1) Activity Level, 2) Attention Span/Distractibility, 3) Adaptability/Approach-Withdrawal, 4) Rhythmicity, and 5) Reactivity. The DOTS specifically gives a good measure of activity level for both awake and sleep states as well as providing a measure of attention span and distractibility i.e., those temperament characteristics relating to hyperactivity of interest in the present study. Reliability coefficients (Cronbach's alpha) were obtained on all scales using samples of infants, preschoolers, school-aged children and young adults with only the subscale for reactivity being consistently below .60 (Lerner et al., 1982); it has also demonstrated acceptable test-retest reliability. The parent's perception of their spouses behavior is rated on a 4-point scale (0-3) with 0 indicating the behavior is "not at all" apparent, to 3 indicating a behavior to be "very much" apparent. Total scores for 1) activity level and 2) attention span and distractibility problems were calculated by summing across the two factors assessing such behavioral qualities. A score for adult hyperactive behavior (high activity levels and attention span/distractibility levels) was calculated by summing across *both* factors for a composite score.

Wechsler Adult Intelligence Scale (Revised). The Information and Digit Symbol subtests from the WAIS-R (Wechsler, 1981) will be used to obtain estimated Verbal, Performance and Full Scale IQ's. The Information subtest assesses mental alertness,

verbal skill, and general knowledge. It is a reliable measure of such and correlates strongly (r = .83) with Full Scale IQ. The Digit Symbol subtest assesses motor persistence, attention, visual-motor coordination, and response speed. This subtest has adequate reliability and is also highly correlated (r = .61) with Full Scale IQ. Each WAIS-R subtest score is multiplied by a constant to yield a prorated estimate of IQ (see Satler, 1988).

Child Instruments

Conners Parent Questionnaire The Conners Parent Questionnaire (Conners, 1990) was administered to both parents, and is the primary measure of hyperactive and conduct problem behaviors. This instrument is a very slightly modified version of Conners' 48-item version (Goyette, Conners, and Ulrich, 1978), but is nearly identical to it with only minor wording changes.

I extracted items from the Conners that are specific to behaviors indicative of ADHD, ODD, and CD symptomatology according to DSM-III-R and DSM-IV standards. I have constructed "pure" measures of hyperactivity (i.e., attention span difficulties, hyperactivity, and impulsivity) and conduct problems. The following table briefly outlines current behaviors needed to assess ADHD and compares them with the items that were selected from the Conners for use in the study to assess HADI. In order to isolate the individual factors for the HADI factor a confirmatory factor analysis was performed based on a previous (Ham, 1992) and present content (i.e., content validity) analyses. The various factors were then subjected to confirmatory factor analysis using LISREL. Goodness of fit indices indicate an adequate fit for the HADI model (GFI = .98; AGFI = .96); revealing that physical hyperactivity, attentional deficits and distractibility, and

Table 1. Behaviors Indicative of Attention-deficit Hyperactivity Disorder

DSM-III-R & DSM-IV*	Conners Parent Questionnaire
1) often fidgets with hands or feet or squirms in seat 2) difficulty remaining seated 3) easily distracted 4) difficulty waiting turn 5) often blurts out answers before question 6) difficulty following through on task 7) difficulty sustaining attention 8) shifts from uncompleted task to another 9) difficulty playing quietly 10) talks excessively 11) interrupts or intrudes 12) does not seem to listen 13) looses things 14) engages in dangerous activities * to qualify for diagnosis child must exhibit at least eight of the above	1) restless or overactive 2) fidgets and restless 3) always climbing 4) acts as if driven by a motor 5) inattentive and easily distracted 6) fails to finish things, short attention span 7) excitable and impulsive 8) demands must be met, easily frustrated 9) gets over excited easily

impulsivity are unique entities. Factor intercorrelations also reveal that these three factors are highly related to one another (average correlation among factors was .75, range .63 to .83). Cronbach Alphas for the three factors indicate substantial reliability (.77, .73, and .73 respectively). The overall measure of HADI was then computed by summing all items across the three factors. The reliability coefficient for the HADI factor was .86, also revealing excellent reliability of this overall factor.

The **Hyperactive** factor is a 4-item factor that specifically gives a measure of gross motor behaviors. Items for this factor are: 1) restless or overactive; 2) constantly fidgeting and restless in the "squirmy sense"; 3) always climbing; and 4) acts as if driven by a motor). The **Attention span/distractibility** factor is a 2-item factor that evaluates

behaviors indicative of inattentiveness and distractibility. The specific items in this factor are: 1) inattentive and easily distracted, and 2) fails to finish things he/she started, short attention span). The **Impulsivity** factor is a 3-item factor that assesses behaviors connotative of an impetuous, reckless nature. Items for this factor are: 1) excitable and impulsive, 2) demands must be met easily, is easily frustrated, and 3) gets over excited easily. As mentioned above, all items for these three factors were then summed together in order to gain a measure of behavior indicative of HADI (i.e., "pure" hyperactivity, exclusive of conduct disordered behavior) (see Table 1).

In order to establish concurrent validity for these HADI factors I compared them to measures of similar behavior from the Dimensions of Temperament Survey for children (DOTS; Lerner, Palermo, & Nessleroade, 1988) and the Child Behavior Checklist (McConaughy & Achenbach, 1983). The DOTS for children gives an overall measure of temperament based on Thomas and Chess's original temperament findings (Thomas & Chess, 1984). Based on previous factor analytic studies of the DOTS for children (Ham, 1992), the original attention span/distractibility factor reported by Lerner et al. remained the same, however, a pure measure of physical activity emerged from their "Reactivity" factor. A high score on the activity factor then would be indicative of "physical hyperactivity", a high score on the Attention Span/Distractibility factor would likewise indicate low levels of attention and high levels of distractibility. Cronbach Alphas for *th*ese two DOTS factors revealed adequate reliability within the current sample (.75 and .65 respectively) (see Ham, 1992 for a full description of factor analytic procedures with the DOTS for children).

The DOTS for children measure of attention span/distractibility significantly

correlated with the attention span/distractibility factor from the Conners (r = .49, p<.00). The activity factor from the DOTS and the hyperactivity factor from the Conners likewise correlated highly and significantly (r = .52, p<.00). These results indicate adequate concurrent validity for the Conners Attention/Distraction and Hyperactivity factors. The Impulsivity factor was correlated with an item from the Child Behavior Checklist, "impulsive or acts without thinking". Factor-item correlation for this was significant (r = .38, p<.00), also revealing this factor as a reliable measure of impulsive behavior.

Finally, a 14-item factor was extracted from the Conners that assesses conduct problems inclusive of behaviors indicative of Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD). In order to isolate oppositional behaviors (i.e., ODD similar behaviors) from more violatory conduct problems (i.e., CD similar behaviors) I broke the Conduct Problems factor into two groups of questions based on DSM-III-R and DSM-IV criteria. Items that comprise the Oppositional factor are 1) disturbs other children, 2) has temper outbursts, 3) denies having done wrong, 4) disobeys parents, 5) mean towards siblings, 6) blames others for his mistakes, 7) carries a chip on his shoulder, and 8) sassy to grown-ups. Table 2 summarizes current diagnostic criteria for ODD for comparison of my Oppositional factor. Items making up the Conduct factor are 1) bullying, 2) steals things, 3) throws and breaks things, 4) fights constantly, 5) picks on other children, and 6) tells stories which did not happen. Table 3 is a listing of DSM behaviors necessary for a child to be diagnosed as CD for comparison of the Conduct factor. Confirmatory factor analysis revealed that the two sets of items are uniquely different and yet highly correlated constructs (as one would expect). Goodness of fit indices indicated that the two unique factors can exist separately (GFI = .95; AGFI = .93). A second LISREL run was

Table 2. Behaviors Indicative of Oppositional Defiant Disorder

DSM-III-R & DSM-IV*	Conners Parent Questionnaire
1) loses temper 2) argues with adults 3) actively defies or refuses adult requests 4) deliberately does things that annoy 5) blames others for mistakes 6) touchy or easily annoyed 7) angry or resentful 8) spiteful or vindictive 9) swears or uses obscene language * to qualify for diagnosis a child must exhibit at least five of the above	1) disturbs other children 2) has temper outbursts 3) denies having done wrong 4) disobeys parents 5) mean towards siblings 6) blames others for his mistakes 7) carries a chip on his shoulder 8) sassy to grown-ups

Table 3. Behaviors Indicative of Conduct Disorder

DSM-III-R & DSM-IV*	Conners Parent Questionnaire
1) stolen without confrontation 2) run away from home 3) lies 4) engages in fire-setting 5) often truant 6) broken into a home or building 7) destroys property 8) physically cruel to animals 9) forced someone into sex 10) used weapons in fights 11) initiates fights 12) stolen with confrontation 13) physically cruel to people * to qualify for diagnosis a child must exhibit at least five of the above	1) bullying 2) steals things 3) throws and breaks things 4) fights constantly 5) picks on other children 6) tells stories which did not happen

conducted to test the reliability of the overall Conduct Problems factor (i.e., with both factors added together), and to see if they comprised a higher order factor as suspected. This was also done because they were to be used both separately and combined in the various model testing procedures in the study. Goodness-of-fit indices revealed that they in fact did constitute a reliable and higher order factor (GFI = .95; AGFI = .93).

As with the HADI sub-factors, I wanted to see if this factor and its two sub-factors revealed concurrent validity with other measures of similar behavior. The Aggression factor from the Child Behavior Checklist was selected for this purpose. Correlations of the overall Conduct Problems factor and the two sub-factors Oppositional and Conduct were significant and high with the Aggression factor ($\underline{r}$ = .63, $\underline{p}$<.00; $\underline{r}$ = .62, $\underline{p}$<.00; $\underline{r}$ = .54, $\underline{p}$<.00 respectively). This suggests that the Conduct Problems factor and its two sub-factors in this sample are valid and reliable measures of conduct disordered behavior in 3-6 year old boys.

The child's behavior is rated on a 4-point scale (0-3); 0 indicating "not at all" for a particular behavior thru 3 indicating that the behavior occurs "very much". Items are summed to obtain factor scores. Items selected from the Conners for inclusion in this study were done with the intent of adhering as closely as possible to current diagnostic criteria (i.e., DSM-III-R & DSM-IV) for isolating children who exhibit ADHD and ODD/CD behaviors. Cutoffs for each factor was set at the 90th percentile in order to classify boys into groups of problem behavior. This resulted in the following cutoffs: HADI = 15; Oppositional = 9; Severe Conduct = 6.

Delay of Gratification Task (Delay). This task was developed by Funder, Block & Block (1983) to evaluate the child's ability to delay gratification and obtain an objective

measure of his/her impulsivity, attentional abilities, concentration ability, and cooperativeness. Subsequent to the child's intellectual assessment session, the child is thanked for their participation and told they can have a present. As the present is being shown, the examiner apologizes to the child and says there is one more task they must first complete. The present is set to the side but within reach of the child; the child is then shown a complex block design task (Design #11-WISC-R) and is told they must first complete this task before getting their present. Total time of the task is 5 1/2 minutes, four minutes of task time on the block design with assistance, and 90 seconds of post-task time (i.e., delay) in which the examiner ignores the child (i.e., puts away his/her papers, writes notes, etc.). The task is terminated at any stage if the child spontaneously takes the package and opens it or takes the package and leaves the room.

Scores on this brief laboratory task, using a large sample of four-year-old children (n = 116), were compared to ratings of the child's personality by examiners and teachers using the California Q-sort. Personality data (Q-sorts) were available from the children when they were 3,4,7, and 11 years of age. Findings demonstrated that boys at age four who delayed gratification on this task were described as being more attentive, able to concentrate, reasonable, and cooperative. These results were interpreted as demonstrating the relationship between task performance at age four and measures of personality related to ego control or under-control that remained very stable during the lengthy time period of this project (Funder et al., 1983). Analysis of data from the current project involving two observers independently scoring the child's behaviors following the detailed scoring procedures developed by the Funder et al. demonstrated excellent inter-rater reliability (n = 12; $\underline{r}$ = .97). The child's performance on this task is revealed by his ability to delay

gratification (i.e., attempt to complete the block design task without taking the present); thus a high score (in time) indicates low impulsivity and a low score indicates high impulsivity.

Stanford-Binet (Form L-M) (SB). The Stanford-Binet was normed on a racially representative sample of 2,100 subjects in 1972 under the supervision of R.L. Thorndike. Subjects were chosen according to community size, geographic region, and socioeconomic status. The SB gives a measure of individual general cognitive functioning yielding a score indicative of the child's current level of mental functioning in relation to his/her chronological age. The SB is specifically appropriate for the present study for several reasons: 1) it has been shown to provide a reliable measure of intelligence, 2) it particularly provides a positive response format for culturally disadvantaged children from lower sociodemographic groups, and 3) younger children have been found to find the test interesting due to the variety of activities presented during the assessment (Munday & Rosenberg, 1979).

Revised Yale Developmental Schedules (RYDS or Yale). The RYDS (Provence & Naylor, 1983) is used to assess cognitive, motoric, adaptive, language, and social functioning. The measure includes portions of the Stanford-Binet, Bayley, and Gesell Scales in order to maximize opportunity to gain a comprehensive and relatively easy evaluation of the child's current level of functioning. The Yale specifically yields age appropriate scores for fine and gross motor functioning, receptive and expressive language, adaptive functioning (i.e., nonverbal problem solving, short-term memory, and the child's knowledge of space, size and numbers), personal/social competence, and an overall developmental quotient. A baseline of functioning is established on all five

domains and testing continues until the child is unable to succeed, thus a ceiling level is met.

Missing Data

There was less than 6% missing data for parental IQ scores, and less than 5% for all other variables used. Mean substitutions by group and sex were used for all missing data.

CHAPTER IV

Results

Group Differences: Main Effects

Multivariate analysis of variance (MANOVA) revealed several interaction, sex (parent), and group (risk group) differences for parental and child variables; these are discussed in their entirety in the text, and statistical results for all MANOVAs are presented in the text. Specific group main effects (univariate results) for all study variables are presented in Tables 4 and 5 along with results from oneway ANOVAs.

Interaction Effects

When the two measures of parental externalizing behavior were examined, MANOVA revealed significant group (risk) by sex (parent) **interaction** effects [$F(4,1202) = 44.2, p < .00$], specifically for antisociality [$F(2,602) = 75.1$, $p < .00$] and lifetime alcohol problems [$F(2,602) = 21.2$, $p < .00$]. These interaction effects are not surprising given that group status is based on paternal levels of alcohol problems. The interaction effects for antisociality and drinking problems can also be explained by the fact that maternal antisociality scores are significantly correlated with paternal scores ($r = .30$), and also because problem drinking scores are significantly correlated with antisocial scores ($r = .50$). No significant interaction effects were noted for the sociodemographic variables (SES, family income, family of origin SES, and parental education level) [$F(6,1200) = 1.9$, $p < .09$], nor were there any interaction effects for parental ratings of problem behaviors (HADI and conduct problems) [$F(10,1194) = .65$, $p < 78$]. Neurobehavioral variables were observer rated so MANOVA was not appropriate to use as interaction effects are

irrelevant.

Sex Effects

MANOVA revealed only one **Sex** (parental ratings) difference for parental externalizing behaviors [$F(2,601) = 131.9$, $p < .00$] that being for parental levels of antisociality [$F(1,602) = 192.3$, $p < .00$]. In this case men rated themselves higher than women (Table 4). Although not statistically significant, women consistently tended to report lower levels of problems related to drinking [$F(1,602) = 3.3$, $p < .07$]. For sociodemographic variables MANOVA revealed an apparent sex difference [$F(3,600) = 5.4$, $p < .00$]. Univariate output specifically identified higher levels of education reported for wives than husbands [$F(1,602) = 3.7$, $p < .06$], and women also scored higher on IQ tests than men [$F(1,602) = 3.6$, $p < .06$], but not for family of origin SES [$F(1,602) = .76$, $p < .38$] (see Table 4). No significant sex differences were found for the parental ratings of child problem behaviors [$F(5,597) = 1.26$, $p < .28$].

Group Effects

MANOVA also revealed several **group** (risk) effects [$F(4,1204) = 125.2$, $p < .00$] for parental externalizing behavior. Both antisociality [$F(2,602) = 221.6$, $p < .00$] and drinking problems [$F(2,602) = 112.1$, $p < .00$] showed a main effect for risk status. This was also the case with sociodemographic variables [$F(6,1200) = 10.1$, $p < .00$], specifically for education level [$F(2,602) = 28.8$, $p < .00$], family of origin SES [$F(2,602) = 5.4$, $p < .00$], present level of SES [$F(2,602) = 12.3$, $p < .00$], IQ [$F(2,602) = 16.3$, $p < .00$], and family level of income [$F(2,602)=35.2$, $p < .00$]. No group main effects were found for the parental temperament characteristics (levels of attention, activity, and persistence) [$F(6,1200) = .22$, $p < .97$] (see Table 4 for means and standard deviations).

Table 4. Means and Standard Deviations and Group Main Effects (Univariate and Oneway ANOVA results) for Parental and Family Variables

	Antisocial Alcoholic Families (AALs) (n = 72) m(sd)	non-Antisocial Alcoholic Families (nAALs) (n = 141) m(sd)	Control Families (n = 91) m(sd)	F(p)
Antisocial Behavior				221.6(.00)
- Maternal[a]	15.6(7.5)	11.0(7.5)	7.8(4.9)	
- Paternal[a]	35.6(12.4)	13.8(5.3)	10.7(6.4)	
Alcohol Problems				112.1(.00)
- Maternal[a]	10.7(2.2)	10.0(1.9)	9.0(1.3)	
- Paternal[a]	11.4(1.9)	10.1(1.6)	7.4(1.6)	
Intelligence Quotient				16.3(.00)
- Maternal[b]	95.8(13.5)	99.3(15.6)	100.4(13.4)	
- Paternal[a]	89.4(13.3)	95.7(16.2)	102.4(15.2)	
Age (years)				2.6(.08)
- Maternal	31.2(3.9)	31.0(4.2)	29.4(4.9)	
- Paternal	32.7(4.5)	32.8(5.1)	32.7(4.5)	
SES (Duncan)[c]	219(100)	279(158)	300(167)	12.3(.00)
Family Income Level[c]	16000	21500	26700	35.2(.00)
Family of Origin SES				5.4(.00)
- Maternal	346(181)	363(187)	384(200)	
- Paternal[c]	299(141)	361(185)	394(176)	
Education (yrs)				28.8(.00)
- Maternal[c]	12.5(1.6)	13.4(2.2)	13.6(1.7)	
- Paternal[a]	12.3(1.9)	13.5(2.3)	14.6(2.1)	
Activity Level				.2(.8)
- Maternal	5.1(.9)	4.9(1.0)	5.1(1.1)	
- Paternal	4.9(1.2)	4.9(1.2)	5.0(1.0)	
Attention/Distraction				.2(.9)
- Maternal	15.6(1.7)	15.7(1.6)	15.9(1.5)	
- Paternal	15.5(1.4)	15.9(1.6)	15.8(1.4)	

[a]=AALs different from nAALs different from Controls (Tukey HSD, $p < .05$)

[b]=AALs different from Controls (Tukey HSD, $p < .05$)

[c]=AALs and nAALs different from Controls (Tukey HSD, $p < .05$)

Group main effects were also found for child problem behavior variables (MANOVA) [F(10,1194) = 5.1, p <.00]. Specifically, main effects were noted for HADI [F(2,601) = 16.2, p <.00] and Conduct Problems [F(2,601) = 11.4, p <.00], the primary child outcome variables. Significant group main effects were also found for the neurobehavioral measures [F(10,1196) = 4.6, p < .00], specifically for the measure of impulsivity (Delay of Gratification) [F(2,602) = 9.6, p < .00] and IQ [F(2,602) = 12.6, p < .00], but not for the measures of gross motor development [F(2,602) = .12, p < .88], fine motor development [F(2,602) = .46, p < .63], and language acquisition [F(2,602) = 1.3, p < .28] (see Table 5 for means and standard deviations).

Group Differences: Specific

One way ANOVA was used to explore specific group hypotheses (see Table 4 and 5). Several differences were expected to emerge between the three groups under investigation (see Hypotheses). Results are reported separately for men and women where appropriate (i.e., parental ratings of child behavioral problems), and for men and women combined when appropriate (i.e., demographic variables and clinician observer ratings). Boys being reared in the **highest** risk group are hereafter referred to as sons of **Antisocial Alcoholics** or **AALs**; boys in the **high** risk group are hereafter referred to as sons of **non-Antisocial Alcoholics** or **nAALs** (Zucker et al., 1993).

Hypothesis I

Boys being reared in the two groups of alcoholic families (**antisocial** and **non-antisocial**) will exhibit: a) higher levels of hyperactivity and conduct problem behaviors (Conners); b) lower IQ scores (Stanford-Binet); and c) lower levels of gross and fine motor and language competency (RYDS), compared to boys in the **control** group.

Hypothesis I specifically suggested that boys in the two high risk groups would exhibit significantly higher levels of neurobehavioral dysregulation than control boys; this was aimed at elucidating the effects of paternal alcoholism on offspring regulatory functioning. From the perspective of mother's data, however, this supposition was not supported for the primary variable of interest, HADI. For instance, oneway ANOVAs for mothers revealed only higher perceived levels of HADI in sons of AALs compared to Controls [$\underline{F}(2,301) = 5.9$, $\underline{p} < .00$]. In fact, Controls and nAAL boys were not significantly different on maternal HADI ratings (i.e., these two groups looked more similar than not with regards to hyperactivity). For fathers, however, Hypothesis I was completely substantiated with respect to HADI; fathers rated boys in both high risk groups significantly higher than Controls [$\underline{F}(2,301) = 10.0$, $\underline{p} < .00$]. For conduct problems both parents perceived greater amounts of disordered behavior in both high risk groups compared to boys in the control group [mothers $\underline{F}(2,301) = 9.1$, $\underline{p} < .00$; fathers $\underline{F}(2,301) = 8.1$, $\underline{p} < .00$].

Hypothesis I also suggested that levels of impulsivity (Delay of Gratification), intelligence (IQ), and developmental functioning would follow the same pattern as HADI and conduct problems (i.e., Controls would score higher or be more highly regulated than the two groups of SOMAs). However, this was only partially substantiated for this sample. For example, scores for the Delay of Gratification were significantly higher for controls than for sons of AALs but not sons of nAALs [$\underline{F}(2,301) = 4.8$, $\underline{p} < .00$]. Similarly IQ followed the same pattern. Controls were significantly higher than sons of AALs but not nAALs [$\underline{F}(2,301) = 6.3$, $\underline{p} < .00$]. This pattern maintained itself throughout the analyses, i.e., nAALs and Control boys were more similar to one another than were

Table 5. Means and Standard Deviations and Specific Group Main Effects (Univariate and Oneway ANOVA results) for Child Variables

	Antisocial Alcoholic Families (AALs) (n = 72) m(sd)	non-Antisocial Alcoholic Families (nAALs) (n = 141) m(sd)	Control Families (n = 91) m(sd)	F(p)
Age	4.4(1.1)	4.3(1.0)	4.2(1.0)	1.1(.4)
Intelligence Quotient	99.9(15.6)	103.4(13.4)	107.7(13.6)	12.6(.00)
HADI				12.2(.00)
- Maternal[b]	9.3(5.9)	7.4(5.1)	6.6(4.1)	
- Paternal[b]	9.5(5.1)	8.0(5.0)	6.2(3.7)	
Conduct Problems				11.4(.00)
- Maternal[b]	10.9(6.6)	8.3(5.9)	7.6(4.6)	
- Paternal[b]	9.9(5.7)	7.7(5.1)	6.9(4.7)	
Delay Gratification (sec)[a]	250(115)	277(95)	296(74)	9.6(.00)
Gross Motor (months)	52.3(14.3)	52.8(12.9)	52.6(12.3)	.12(.9)
Fine Motor (months)	53.4(12.1)	54.4(11.7)	54.2(10.7)	.46(.6)
Language (months)	53.9(10.6)	55.1(10.7)	53.3(9.9)	1.3(.3)

[a]=AALs different from Controls (Tukey HSD, $p < .01$)

[b]=AALs different from nAALs and Controls (Tukey HSD, $p < .01$)

sons of AALs. As stated in the MANOVA results, no group main effects were noted for scores on developmental functioning for gross and fine motor and language proficiencies (see Table 5).

Hypothesis II

Hypothesis II stipulated that boys in the **antisocial, alcoholic** group will exhibit: a) higher levels of hyperactive related behaviors (Conners); b) lower IQ scores (Stanford-Binet); and c) lower (i.e., delayed?) levels of motoric, language, and overall cognitive development (RYDS), compared to boys in the **non-antisocial** alcoholic group and the **control** group.

Hypothesis II suggested that sons of antisocial alcoholics would be significantly less regulated than boys in both of the other two groups thus revealing the impact that paternal antisociality, in conjunction with alcoholism, has on behavioral regulatory functioning. As stated above, results of oneway ANOVAs for mothers substantiated this finding for HADI [$F(2,301) = 5.9$, $p < .00$] but not for fathers (see above). Also as stated above, oneway ANOVAs for both fathers and mothers substantiated this claim for conduct problems, thus suggesting that paternal antisociality and male offspring antisociality follow some sort of familial pattern. Hypothesis II was not substantiated with regards to the remaining offspring variables (i.e., delay of gratification, IQ, motor and language functioning). As the above section reported Delay of Gratification and IQ were different only between controls and sons of AALs; sons of nAALs were not different than either of the other two groups; measures of motor and language proficiency showed no group differences at all (see Table 5).

Hypothesis III

Boys being reared in **antisocial, alcoholic** families will exhibit higher levels of conduct problems (Conners) than the other two groups of boys. And likewise, SOMAs in the **non-antisocial** group will exhibit higher levels of the same behavior compared to control boys. In other words, there will be a progression in symptomatology of severe conduct problems as level of risk increases.

Hypothesis III suggested a progression of offspring antisociality which followed that of the fathers of boys in each group. As the above results suggest, this assumption was substantiated, although not completely. The progression of higher antisocial behaviors was seen for offspring measures of conduct problem symptomatology, however, results were not statistically significant in each case. Boys with the most antisocial parents (sons of antisocial alcoholic fathers) revealed the highest levels of conduct problem behavior compared to the other two groups. Even though sons of AALs had parents who were significantly more antisocial than control parents they were not statistically different than control boys with regards to conduct problems. They were, however, rater higher on conduct problem behaviors thus revealing the same pattern of antisocial behaviors in the offspring of antisocial parents (see Table 5).

Hypothesis IV

Hypothesis IV predicted a higher incidence of hyperactive behaviors (DOTS-spouse) for alcoholic parents compared to non-alcoholic parents. Also, AALs and their wives were expected to display higher levels of hyperactive symptomatology than the other two groups. In other words, levels of hyperactivity in parents would be seen as a function of level of psychopathology.

This hypothesis was completely unsubstantiated. Hyperactive symptomatology was identical for all groups according to the temperamental characteristics assessed for these behavioral attributes (see Table 4).

Parental Differences in Psychosocial Functioning

Results of oneway ANOVAs for parental differences on the various descriptor variables used in this study are presented below. The data report levels of antisociality to be significantly higher in wives of antisocial alcoholics (AAL) than wives of non-antisocial alcoholics (nAAL) and controls [$F(2,301) = 25.7$, $p < .00$]. Similarly, levels of antisociality were higher in wives of nAALs than controls. Lifetime problems due to drinking were significantly higher in wives of AALs than the other two groups, and significantly higher for wives of nAALs than controls [$F(2,301) = 16.4$, $p < .00$]. IQ scores for wives of AALs were also significantly lower only than controls, and maternal reported level of education was significantly lower for both wives of AALs and nAALs than wives of controls. There were, however, no significant difference between the two high risk groups (i.e., AALs and nAALs) for IQ scores [$F(2,301) = 3.0$, $p < .05$].

As one would expect, results for men were very similar as those for women. Antisociality [$F(2,301) = 241.6$, $p < .00$] and drinking problems [$F(2,301) = 127.9$, $p < .00$] following the same pattern (i.e., the progression of higher pathology for AALs than nAALs and controls, and higher pathology for nAALs than controls). Paternal IQ likewise was significantly different for all three groups, controls having the highest IQ and AALs having the lowest [$F(2,301) = 12.8$, $p < .00$]. For education level the results followed the same pattern as those for IQ; men in the highest risk group (AALs) had significantly lower levels of education than the other two groups, and likewise men in the

high risk group (nAALs) had significantly lower levels of education than controls [$F(2,301) = 24.7$, $p < .0$]. One disparate finding was noted between fathers and mothers; paternal reported family of origin SES was significantly higher for controls and nAALs than for AALs [$F(2,301) = 6.2$, $p < .00$], whereas no differences were noted for maternal ratings of family of origin SES (see Table 4).

For the remaining demographic indicators that reflect a combined rating for husband and wife in each family, oneway ANOVAs revealed that group main effects for SES and income are due to the antisocial alcoholic environment. AALs and their wives reported lower levels of SES [$F(2,301) = 6.1$, $p < .00$] and lower amounts of family income [$F(2,301) = 17.6$, $p < .00$] than the other two groups (see Table 4).

Incidence and Overlap of Hyperactivity and Related Problem Behaviors

One of the primary purposes of this study was to explore the incidence of hyperactivity (HADI) and related conduct problems (Oppositional and Conduct) in the two main groups of boys under study, namely, SOMAs and Controls. Results for the incidence and overlap of expression for these problem behaviors were consistent with other findings (Hinshaw, 1992; Lilienfeld & Waldmen, 1990). Based on maternal ratings, 12% of the entire sample met cutoffs for HADI, 8% for Conduct, and 11% for Oppositional (Figure 6). The rate for HADI is quite a lot higher than the national prevalence rate of ADHD (3%) as cited in the DSM-III-R (APA, 1987) and higher than rates of 3-6% as suggested by researchers (Lilienfeld & Waldmen, 1992); rates for Oppositional behavior and Conduct behavior are within normal national ranges. A good portion of children in this sample are being reared in alcoholic homes, homes where prevalence rates of problem behaviors are expected to be much higher, therefore the 6

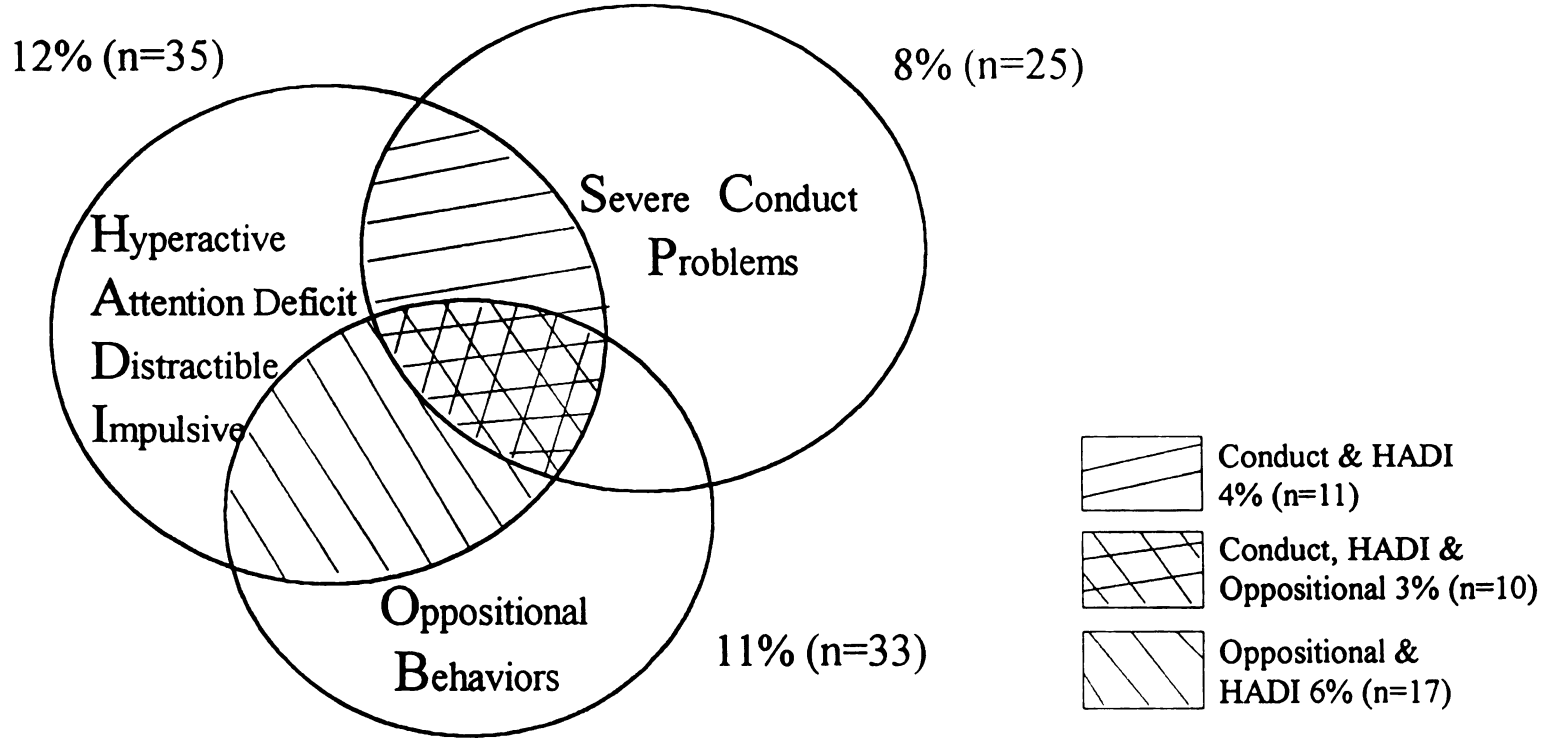


Figure 6. Prevalence and Overlap of Disruptive Behaviors for Entire Sample (Maternal Ratings)

Note: Prevalence rates and number of boys in the present study making cutoff are in percentages beside each content domain (N=304)

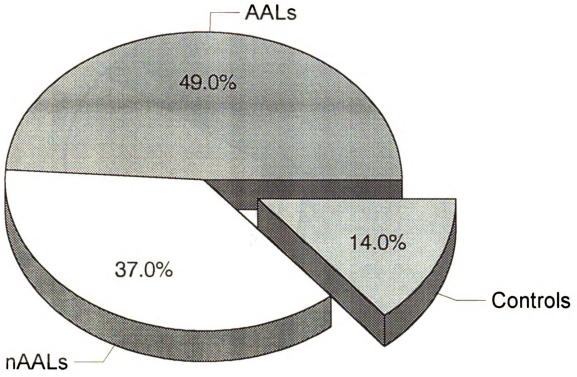


Figure 7. Prevalence of HADI by Group

Note1: This chart represents 12% of the entire sample (N=304)
Note2: AALs=Sons of Antisocial Alcoholics nAALs=Sons of non-Antisocial Alcoholics Controls=Sons of Comparisons

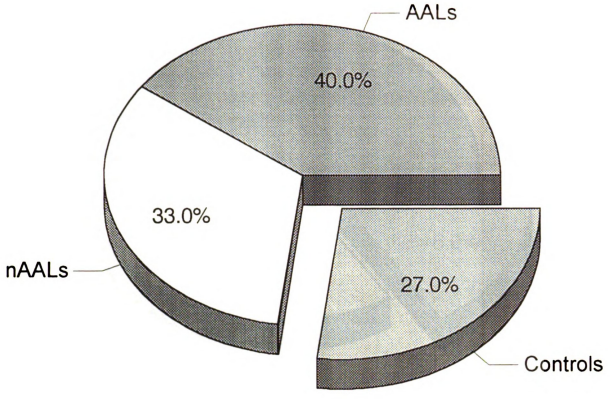


Figure 8. Prevalence of Oppositional Behavior by Group

Note1: This chart represents 11% of the entire sample (N=304)
Note2: AAL=Sons of Antisocial Alcoholics nAALs=Sons of non-Antisocial Alcoholics Controls=Sons of Comparisons

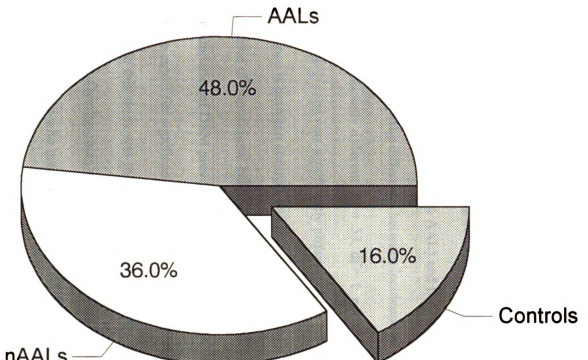


Figure 9. Prevalence of Severe Conduct Problems by Group

Note1: This chart represents 8% of the entire sample (N=304)
Note2: AALs=Sons of Antisocial Alcoholics nAALs=Sons of non-Antisocial Alcoholics Controls=Sons of Comparisons

increase in HADI may be explainable. More specifically, 5% of the boys being reared in control families met cutoff criteria for HADI, while 9% or the boys in non-antisocial alcoholic families were HADI and 24% of the sons of antisocial alcoholics were high on HADI. Thus we see a typical prevalence rate among controls and an expected increase among children of alcoholics. Another way of looking at prevalence rates in the present sample is as follows: Of the boys meeting cutoffs for HADI 86% of them are being reared in alcoholic homes (37% nAAL and 49% AAL) and the remaining are from "normal" or control homes (Figure 7) (the difference between incidence of HADI for nAALs and AALs was not significantly different: $\underline{X}^2 = .53$, df = 1, $\underline{p} = .5$, however, the difference between the three groups was significantly different: $\underline{X}^2 = 6.34$, df = 2, $\underline{p} < .05$ indicating that the difference is between controls and alcoholic offspring) .

Incidence of oppositional behavior and conduct problems for the entire sample was within the boundaries of DSM prevalence rates of ODD and CD. Specifically, 11% of the sample met the cutoff for significant oppositional behavior and 8% for conduct problems. Important also to this study was the overlap of hyperactivity with oppositional and conduct problems. Overlap rates were consistent with the findings of others (Hinshaw, 1992). Of those children who met cutoffs for HADI, 32% also met cutoffs for conduct problems and 50% for oppositional behavior (Figure 6). Also, there was 42% to 56% overlap of oppositional and conduct behaviors. Thus, there is a great deal of overlap within this sample, however, the majority of overlap is seen among SOMAs (Figures 8-9).

Odds Ratios for HADI

The incidence of HADI was of primary importance in the present study, therefore, odds ratios (OR) for HADI were calculated in order to understand the risk of alcoholism

and antisociality for this type of dysregulatory behavior. Table 6 reports the various OR for HADI in the boys in the present study. As one can see, a child of an alcoholic is at significantly greater risk for behavioral problems (2.6 times), but it is the combination of

Table 6. Odds Ratios for HADI

Groups	Odds Ratio	Chi-square(p)
Controls (5%) vs. Alcoholics (15%)	2.6	64.4(.00)
Controls (5%) vs. nAALs (9%)	1.7	10.5(.00)
Controls (5%) vs. AALs (24%)	4.3	11.9(.00)
nAALs (9%) vs. AALs (24%)	2.6	30.3(.00)

Note: Chi-squares all have 1 d.f.

alcoholism and antisociality, as hypothesized, that drastically increases the risk for such dysregulation (4.3 times). However, similar to Ellis's (1993) findings, it seems that antisociality is the most significant etiologic factor for HADI, and not alcoholism per se. Consider, for example, the data presented in Tables 2 and 3. The entire group of SOMAs are nearly 3 times more likely to exhibit HADI than control children and when one considers the two groups of SOMAs, the results are even greater; the sons of antisocial alcoholics are nearly 3 times more likely than sons of non-antisocial alcoholics to be behaviorally dysregulated. Also, sons of non-antisocial alcoholics are less than 2 times more likely to be dysregulated than controls. However, when one compares controls with sons of antisocial alcoholics, this is when the odds ratio is at its highest level.

Differences Among Hyperactives and non-Hyperactives

There were several assumptions tested in the present study concerning the apparent disparity in offspring and parental functioning for hyperactive and non-hyperactive children. Many studies, both within the alcoholism domain and those without, have concluded that hyperactive children are often reared by past or present hyperactive parents (e.g., Cantwell, 1972, Cloninger et al., 1988; West & Prinz, 1987) and that their level of functioning, both cognitive and behavioral, is greatly influenced by the hyperactive environment (see Lilienfeld & Waldmen, 1990). Some attribute the differences to genetics, whereby hyperactives inherit a substandard ability to monitor their behavior, and consequently are found lacking in behavioral and social regulation. Also, many report cognitive deficiency in hyperactive children.

In order to test these assumptions the entire sample of boys was segregated into two groups based on meeting a cutoff for HADI. One group (n = 35) is composed of hyperactive boys, the others (n = 269) served as controls. Group status was based on maternal ratings (see Fitzgerald, Zucker, Maguin, & Reider, 1994 for rational); a score of 15 or greater on the HADI factor placed a boy in the hyperactive group. ANOVA was employed to test for the various disparities assumed to be due to hyperactivity.

Cognitive Differences in Boys

One of the assumptions made for the present study was that boys meeting the criteria for HADI would be significantly more cognitively impaired than non-HADI boys. Cognitive adequacy was assessed using an IQ test and a language proficiency measure (see Methods). ANOVA was used to compare IQ scores and language proficiency among the two groups of boys to test this supposition. Results indicate that the assumption was

Table 7. Parent/Child Variables: Means and Standard Deviations for Hyperactives vs non-Hyperactives

		Hyperactive (n = 35) m(sd)	non-Hyperactive (n = 269) m(sd)	F(p)
Parent ASB	F	16.7(10.2)	10.4(6.6)	25.9(.00)
	M	27.2(18.3)	16.9(11.2)	22.3(.00)
Parent LAPS	F	10.5(2.5)	9.8(1.8)	4.5(.04)
	M	10.6(2.4)	9.5(2.2)	7.7(.01)
Parent Education	F	12.3(1.6)	13.4(1.9)	8.5(.00)
	M	12.1(2.2)	13.8(2.2)	17.7(.00)
Parent IQ	F	94.4(15.8)	99.8(14.1)	4.4(.04)
	M	86.1(13.7)	98.3(15.6)	19.4(.00)
Family Income Level		14,800	22,000	25.0(.00)
Family of Origin SES		289(117)	376(195)	6.6(.01)
Family SES		208(122)	280(154)	7.1(.00)
Child IQ		94.9(13.5)	105.1(14.1)	16.4(.00)
Gross Motor (months)		52.5(15.3)	52.7(12.8)	.01(.9)
Fine Motor (months)		52.3(12.6)	54.4(11.4)	1.0(.3)
Language (months)		50.3(13.3)	55.9(12.0)	6.6(.01)

Note: F = female or maternal scores, M = male or paternal score.

in fact a correct one. HADI boys had significantly lower intelligence quotients and they were significantly more impaired on the language proficiency examination (Table 7).

Parental and Sociodemographic Differences

Another assumption made was that hyperactive children would be being reared in homes where higher levels of parental externalizing psychopathology and environmental

deprivation would be present. Again, ANOVA revealed that these initial assumptions were in fact correct. Table 7 reports means and standard deviations and all ANOVA results for these variables. Specifically, parents of hyperactive boys are more antisocial, exhibit significantly more alcoholism, and they have significantly lower IQs than parents of non-hyperactives; their children are being reared in economically deprived environments, namely lower levels of income, lower socioeconomic status (this includes grandparent level of socioeconomic status as well, i.e., family of origin SES), and significantly less education.

Correlational Analysis

Bivariate Analyses

Hypothesis V

Hypothesis V predicted that lower levels of SES, parental education, and family income would be highly associated with HADI and oppositional and severe conduct behaviors. Hypothesis V was tested using simple bivariate correlational analysis. The hypothesis suggested that behavioral regulation, although elsewhere noted to be highly associated with parental pathology, is here related to the socioeconomic environment. It was specifically expected that high correlations between family of origin SES, present levels of SES, family income, and parental education with offspring HADI and conduct problem behaviors would be found. Table 8 represents the results of these correlational analyses. As the data in Table 8 suggest, offspring behavior regulation is not related to demographic indicators in this sample, although Family Income is moderately-lowly correlated with HADI; Hypothesis V was thus not substantiated. These results had significant impact on the hypothesis testing done with LISREL (see below).

Table 8. Bivariate Correlations for Demographic Variables and Dysregulatory Behaviors

	Family of Origin SES	Family Income	Education	SES
HADI	.09	.24	.16	.10
Conduct	.00	.12	.09	.09

Note: All correlations are **statistically** significant ($p < .05$), but not necessarily qualitatively strong/high.

Structural Equation Models: LISREL

Hypothesis VI

Parental externalizing psychopathology, parental intelligence, parental hyperactive symptoms, and contextual variables will be found to be collectively involved in a causal relationship with biobehavioral dysfunction (HADI, poor neurocognitive functioning, and conduct problems) in SOMAs. In order to estimate the multiple and interrelated dependent relationships of the various parental and child constructs as hypothesized in Hypothesis VI, structural equation modeling procedures were performed using LISREL (Joreskog & Sorbom, 1989).

Below are the previously shown path diagrams. In them the reader can see the theorized relationships between all exogenous (parental/contextual variables) and endogenous (child variables) constructs (Figures 1, 2, and 3). Straight arrows indicate "causal" relationships from exogenous to endogenous constructs (and from endogenous to endogenous constructs where appropriate); curvelinear lines indicate simple correlations (e.g., colinearity) between constructs.

The general model tested is presented in Figure 1. This diagram reveals the

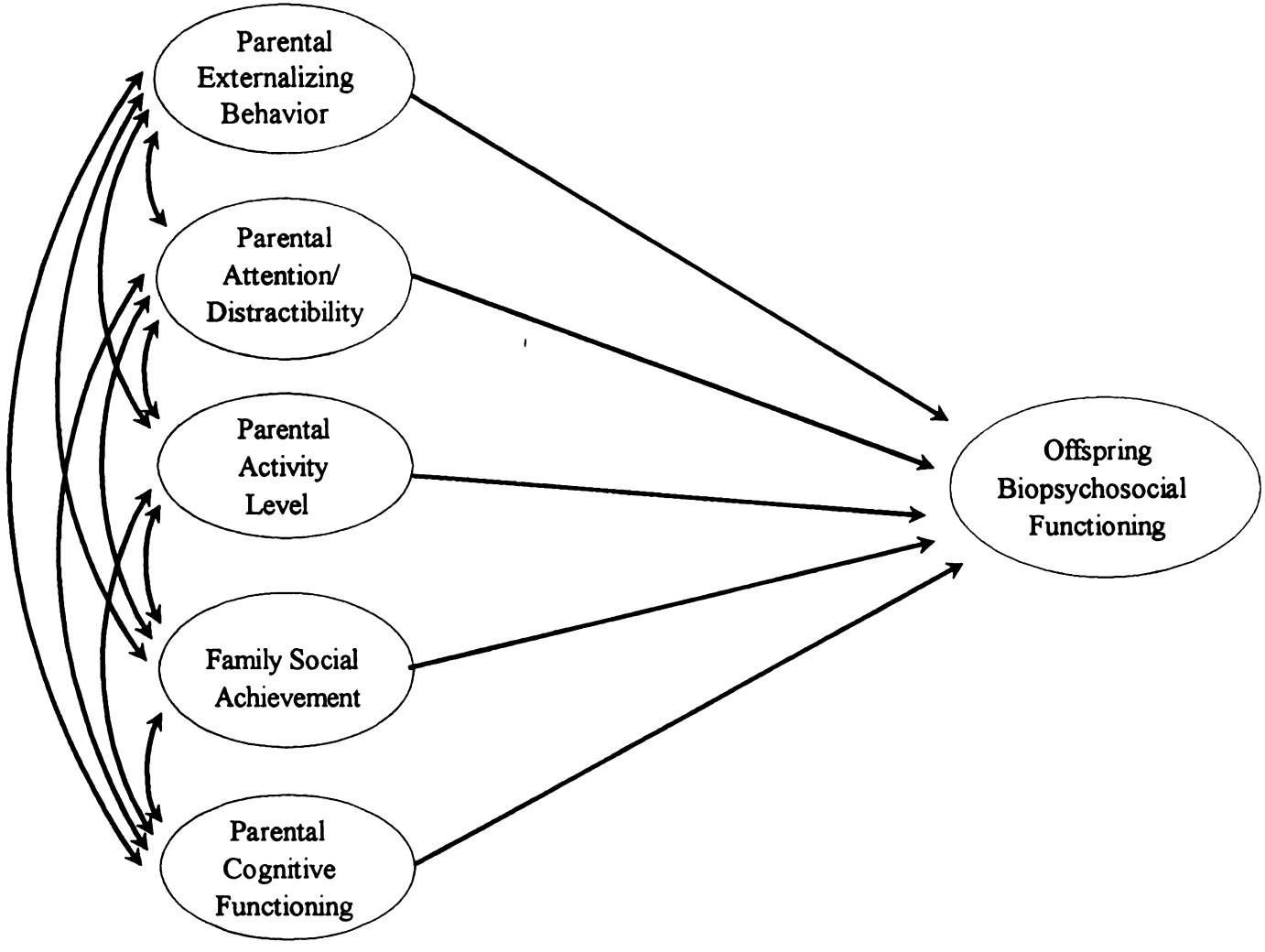


Figure 1. Variables Contributing to Behavioral Regulation: Structural Model 1

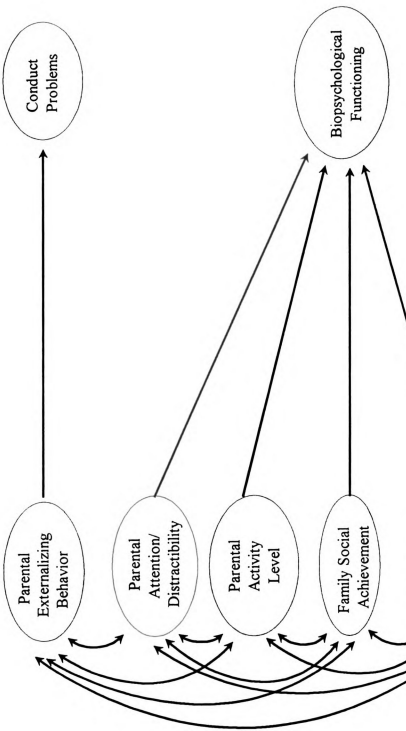


Figure 2. Variables Contributing to Behavioral Regulation: Structural Model 2

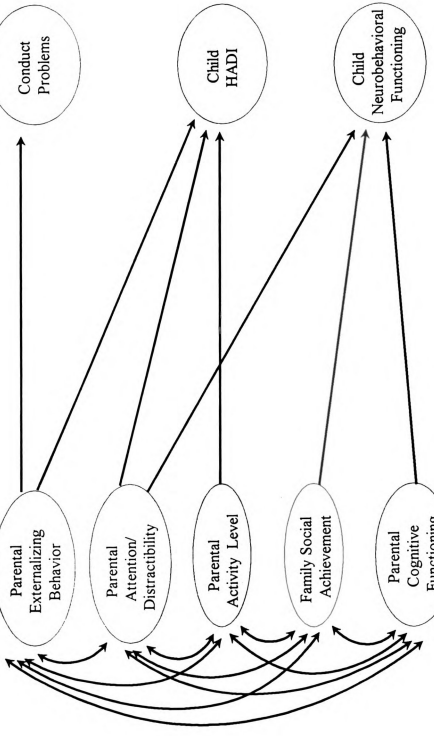


Figure 3. Variables Contributing to Behavioral Regulation: Structural Model 3

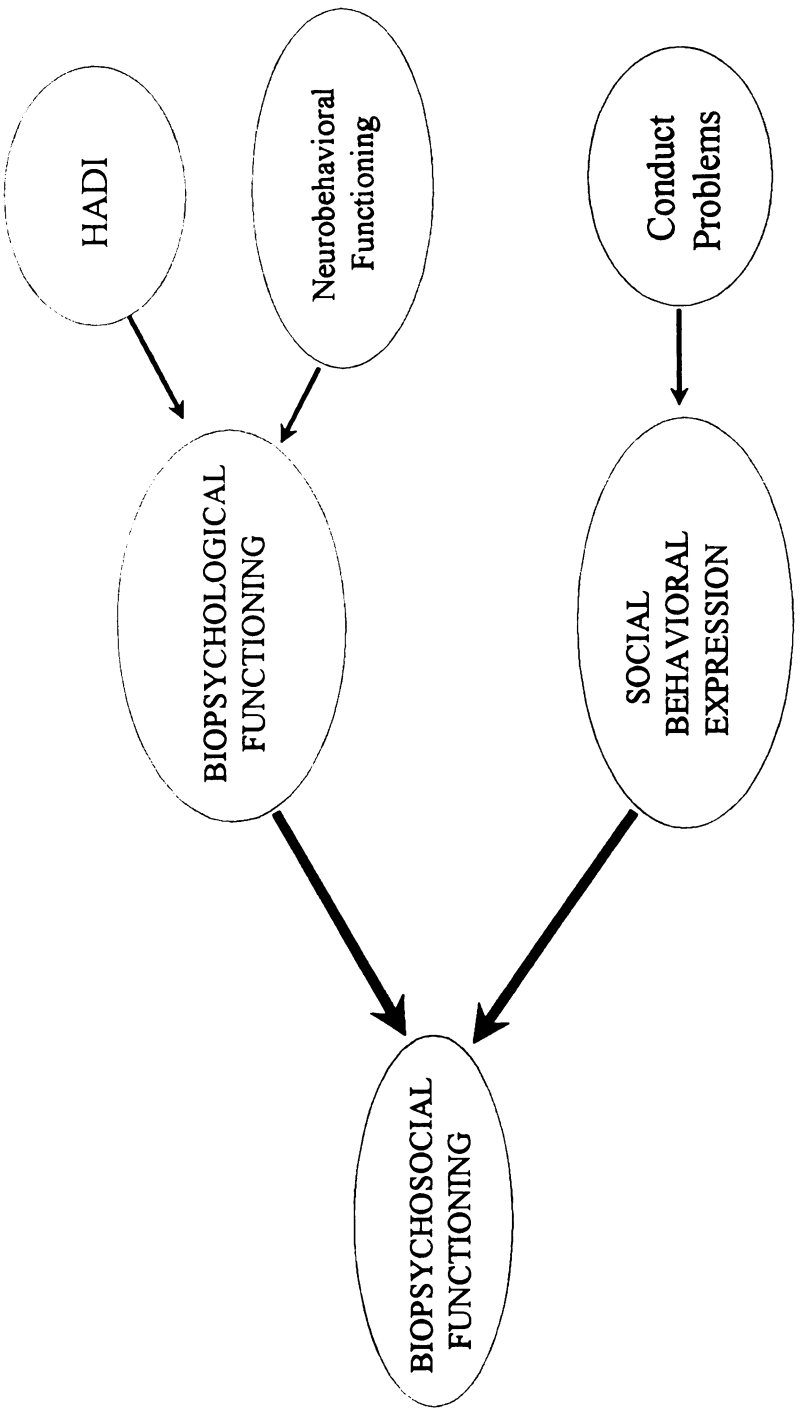


Figure 4. The Higher Order Structural Model of Behavioral Regulation

underlying premise of the study showing that certain parental variables predict **overall** behavioral regulation. Here it is showing the explicit variables that were empirically tested. Figure 2, on the other hand reveals the two primary outcome variables (Conduct Problems and Biopsychological Functioning) that compose the overall Biopsychosocial Functioning variable seen in the preceding figure. In Figure 3 one can see the final model to be tested; here the three primary variables that compose behavioral regulatory functioning have been separated out; they are, behaviors that measure social functioning (i.e., Conduct Problems), behaviors that assess attention, distractibility, and physical activity levels (i.e., HADI), and behaviors that imply neurobehavioral functioning (i.e., motor and language proficiency, ability to regulate impulse, and cognitive functioning via IQ testing).

The global child **measurement** model can be seen in Figure 4. This figure shows the relationships between the primary outcome variables. As stated above, it is believed that HADI and neurobehavioral functioning comprise a different element of regulatory behavior than aggressive, non-compliant behavior (such conduct problem behavior is described in the model as "Social Behavioral Expression" because these behaviors are reflected in one's social interactions and functioning). Both of these overall expressions of regulatory behavior (biopsychological functioning and social behavioral expression) were then collapsed into the overall measure of "Biopsychosocial Functioning". This variable then, reflects self-regulatory behaviors noted in measures of HADI, neurobehavioral functioning (as reflected in IQ tests, motor tasks, observer ratings of impulsivity, etc.), and conduct problems.

The model tested posits regulatory function to exist on a continuum from highly

regular to highly dysregular. In other words, children who are high regulators of behavior will score low on measures of HADI and conduct problems, and higher on global measures of neurobehavioral functioning; and children who have difficulties regulating their behavior will score high on the HADI and conduct problem measures and low on the neurobehavioral measures. Therefore, the present theory suggests that highly regulated parents will have children who are likewise highly regulated, and of course vice-versa.

The general structural model in Figure 1 indicates the theorized causal relationships of parental functioning and sociodemographic (i.e., environmental) influences on offspring biopsychosocial behavioral functioning *within the alcoholic family environment*. Here the model specifies several parental and contextual variables thought to impinge upon child behavioral regulatory outcome in sons of male alcoholics (SOMAs). The model indicates that parental alcoholism, parental antisociality, several depreciable environmental (i.e., sociodemographic) influences, parental hyperactive symptoms, and parental cognitive functioning have direct and causal influences of the same types of behavior on their offspring. There are, however, exceptions to the model. For instance, antisociality may only have an effect on the child's outcome as an indirect causal agent, possibly mediated by alcohol abuse/dependence. This is suggested because alcohol has been biologically related to neurologic insult in both alcoholics and their offspring, and although the model did not test a biologic relationship directly, it does suggest that parental alcoholism possibly contains an heritable component that is directly linked to offspring neurologic outcome (maybe even more so than antisociality).

Figure 2 is the alternate model. This model suggests that aggressive, non-compliant conduct problems (Conduct Problems) may have separate and different causal

relationships with the various parental constructs than Biopsychological Functioning (i.e., HADI behaviors and neurobehavioral functioning combined). This model is an attempt to illustrate differences in several causal relationships believed to exist between aggressive, non-compliant behaviors with HADI and other cognitively embedded ones.

Figure 3 is the final alternate model. Here the model attempts to describe the relationships believed to exist between parental characteristics and offspring regulatory functioning in the most explicit manner. In this model the theory suggests that parental externalizing psychopathology (ASB and drinking problems) is causally related to behaviors indicative of conduct problems and HADI, and that parental temperament characteristics (attention and activity level) are directly involved in similar HADI offspring behavioral functioning. Finally, the model suggests several relationships that may exist between family socioeconomic achievement and parental intelligence with offspring neurobehavioral functioning.

The two sides of the complete measurement model are depicted in Figures 10 and 11. Variables in circles represent latent constructs (not directly measured), variables in the rectangular boxes represent aspects of behavior (or whatever) that are directly measurable; these measured aspects of behavior are referred to as manifest variables or indicator variables. Latent variables are basically theorized constructs similar to factors in a factor analysis, which one feels are indicative of a certain underlying construct or attribute. For example, as Figure 10 illustrates, HADI is specifically composed of behaviors consistent with distractibility, attention deficit, impulsivity, and hyperactivity. If one were to conduct a factor analysis on items that tapped the above four areas one would expect them to load on one factor **if** they truly measure ADHD (exactly this was done with LISREL).

Similarly, the child's expression of neurobehavioral functioning is comprised of several components that are typically agreed upon as isolating neurocognitive capabilities. Specifically, these included a child's intelligence (expressed as verbal and performance abilities; both of which are used to derive an IQ score), direct measurements of fine and gross motor development, and receptive and expressive language adequacies; all of which are considered to contribute to the underlying construct of cognitive capacity. An observed measure was included that assesses impulsivity; this measure was labeled the "Delay of Gratification". This component of functioning (impulsivity) is believed to assess a neuropsychological component of behavioral regulation (Tarter et al., 1985). All of these elements compose the latent variable labelled "Neurobehavioral Functioning".

Following this, both of those latent constructs (HADI and Neurobehavioral Functioning) were collapsed into a variable that reflects what is believed to be the biopsychological element of regulatory behavior, and which excludes aggressive or antisocial behavior. The conduct problems construct is composed of behaviors that reflect how a child acts and reacts towards others and society; these then are collectively referred to as "Social Behavioral Expression". This construct is composed of two factors; one reflects behaviors indicative of oppositional and the other, of more severe conduct problems. Figure 11 reveals the logic behind the parental or causal constructs to be used. The same logic used for the child side of the measurement model was used in constructing this side of the measurement model.

Arrows in the measurement model are different from those in the structural model. Thin straight arrows from latent variables to measured or manifest variables represent those measured variables that comprise or makeup the latent variable. The thick short

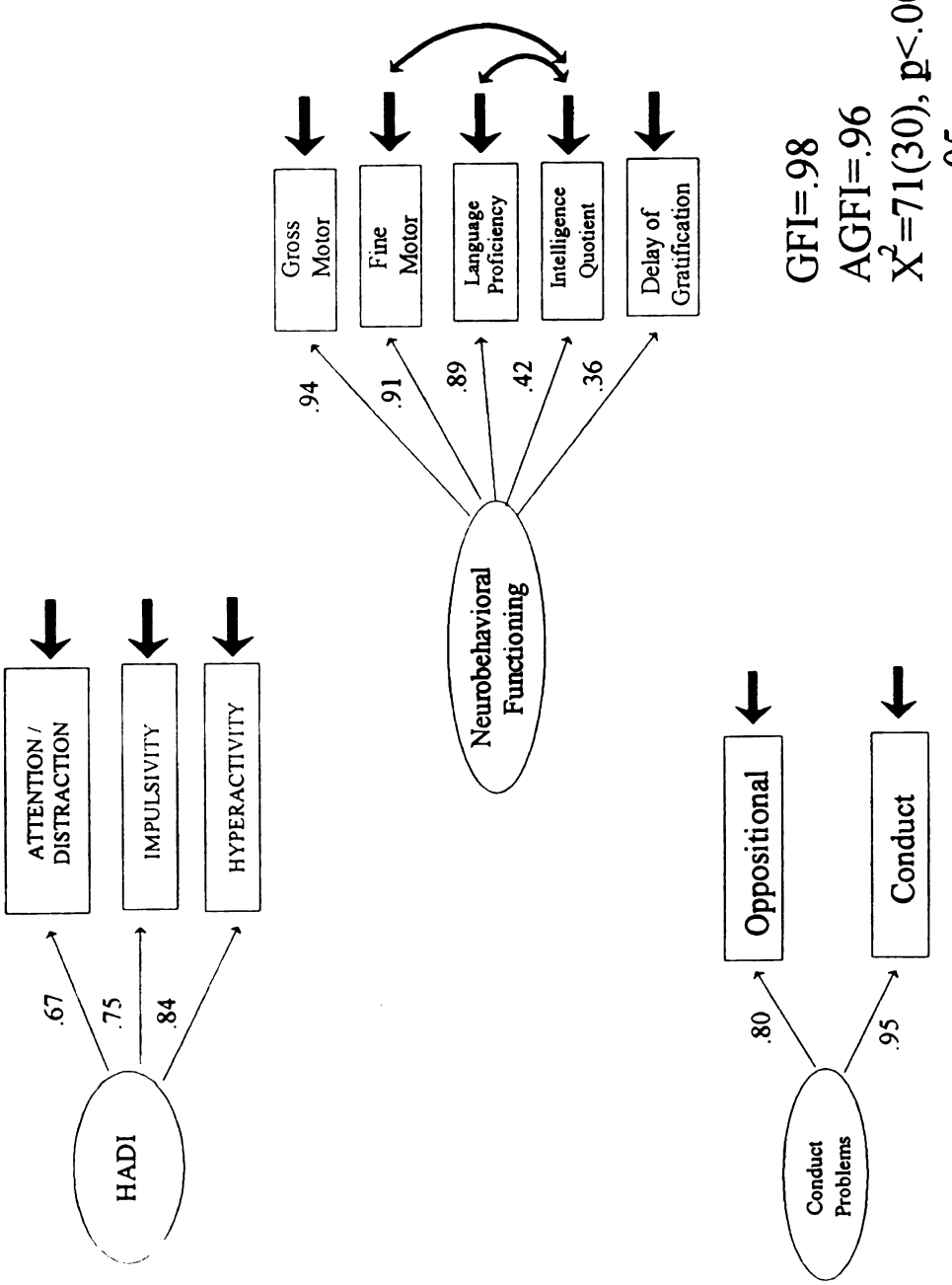


Figure 10. Behavioral Regulation: The Measurement Model for ETA (exogenous) variables.

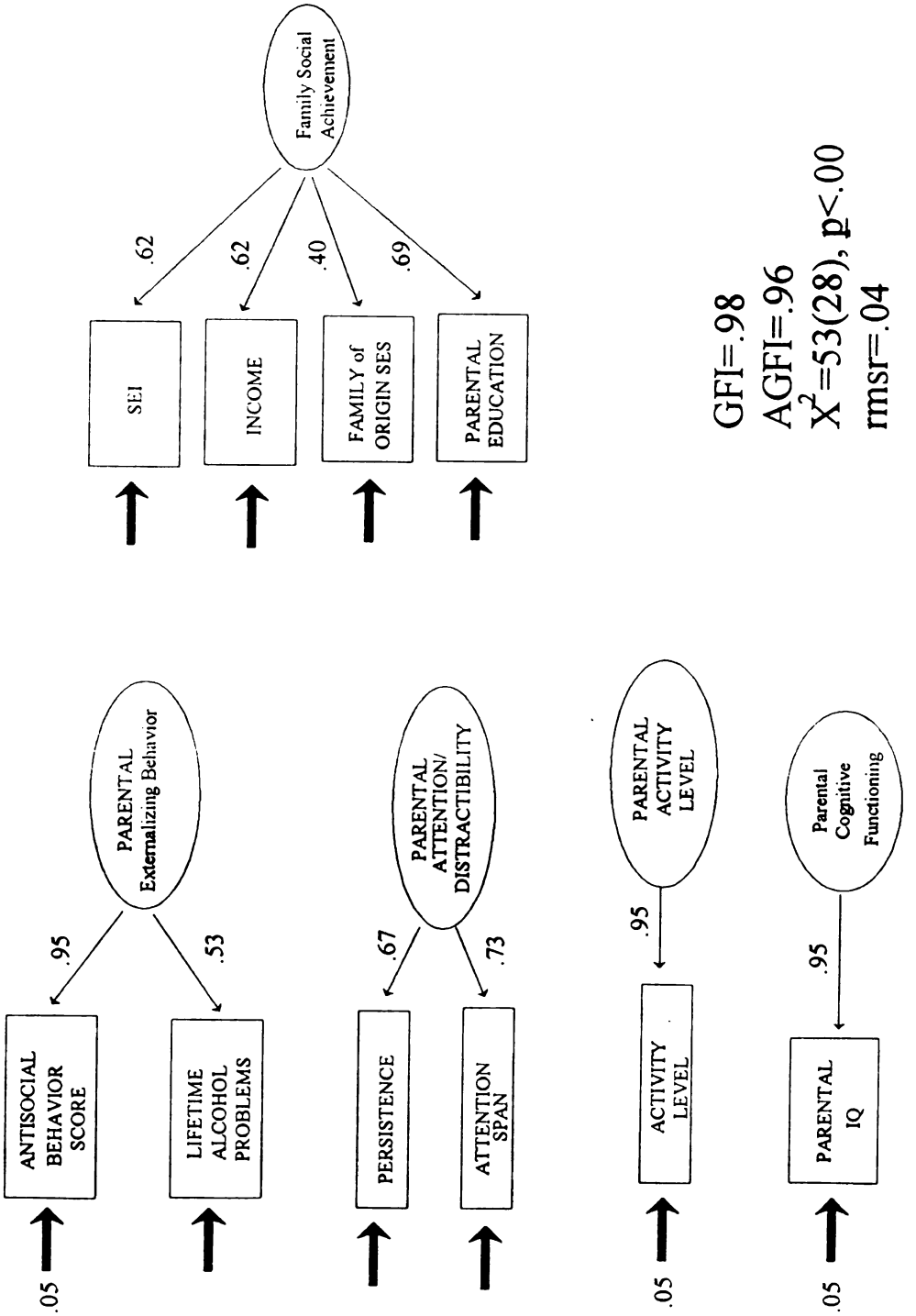


Figure 11. Behavioral Regulation: The Measurement Model for KSI (endogenous) variables.

arrows pointing to the rectangular boxes indicate that several items or assessments comprise that measured aspect of the model, e.g., the thick short arrow pointing to "language" indicates that multiple measurements go into this variable, which in this study included several questions and exercises from the Stanford-Binet and the RYDS that specifically assess receptive and expressive language abilities. The thick short arrows that point to circles (i.e., latent variables endogenous to the model) indicate residual variance (referred to as "residuals") not accounted for by the exogenous variables that the model assumes "cause" the expression of the endogenous latent variable.

Results of Linear Structural Relationships (LISREL) Testing

The Measurement Models

The first step in testing structural equation models is verifying the adequacy of the measurement model. Indicator elements that are assumed to contribute to latent variables must be tested for validity and reliability. This is essentially accomplished by performing the equivalent of a confirmatory factor analysis via a statistical package such as LISREL (Jorskog & Sorbom, 1989).

As stated in the Methods section, there are two measurement models that contribute to the overall structural models that were tested - the parental side (KSI) and the offspring side (ETA). The KSI portion of the model (see Figure 11) consists of five parental measures - externalizing psychopathology, parental temperament characteristics consistent with ADHD symptomatology in children, (specifically one that assesses attention span and distractibility, and the other that measures physical activity level), socioeconomic achievement, and intelligence.

The externalizing psychopathology variable is composed of two scores: one for

lifetime antisocial behavior and the second for lifetime alcohol problems. The attention span/distractibility variable consists of two scores also, one that assesses attention span (composed of 5 items), and another that assesses persistence or distractibility level (composed of 5 items) as perceived by one's spouse. The activity variable (composed of 3 items) measures ones level of physical activity as perceived by one's spouse. Socioeconomic achievement consists of four single score elements - SES, income, family of origin SES, and education level. And the final factor is a single indicator factor comprised of an intelligence quotient.

A confirmatory factor analysis was performed on the above mentioned five factors. Results for this model are presented in Figure 11. Initial fit indices indicate a very good fit to this measurement model (GFI = .98; AGFI = .96). Note that the error term (TD) for antisocial behavior score has been set to zero. This was done because initial runs estimated an error variance that was negative but not significantly different from zero; thus, this term was fixed to zero. For the two single indicator variables (Activity Level and Cognitive Functioning) the error terms were fixed to .05 as convention would suggest (see Bolon, 1989; Loehlin, 1987). Coefficients beside each indicator element are taken from the standardized solution in the LISREL output; thus they are comparable to one another within constructs only.

The ETA (offspring) side of the model (see Figure 10) was an attempt to confirm that several scores for Conduct Problems, HADI behaviors, and neurobehavioral functioning contributed significantly to their respective factors. The first factor (Conduct Problems) consisted of two scores, one for oppositional behavior composed of 8 items, and the other for severe conduct problems, which consisted of 6 items. The second factor

(HADI) consisted of three scores: attention (composed of 2 items), impulsivity (containing 3 items), and hyperactivity (made up of 3 items). The third factor which assessed neurobehavioral functioning was comprised of 5 single scores; a score for gross motor development, a score for fine motor development, a score for language development, an observed rating of impulsivity (Delay of Gratification), and an intelligence quotient.

Initial estimates of the confirmatory process indicated that this model was a fairly robust measurement model (GFI = .98; AGFI = .96). In order to achieve the most parsimonious model several of the TD's in the neurobehavioral variable were permitted to correlate. One should enter such analyses with the assumption that, if error terms *within* a factor indicate that they should be allowed to correlate, then they should be permitted to do so; however, it is usually not wise to allow error terms *between* latent variables to correlate simply because between factor items should, by theory, be uncorrelated (see Loehlin, 1989).

Upon retrospection, and after having conducted all analyses, there were several elements of this side of the model that should have been examined in closer detail. Specifically, there were two elements that did not seem to properly fit on the factor that they were forced to load on; these were the score for the Delay of Gratification and the Stanford-Binet IQ score, both of which are present in the Neurobehavioral Functioning variable. However, both items did load significantly on the factor (based on T-values) so it was decided to let them maintain their position and see how they fared in the structural models. As stated above, in retrospect it may have been wise to remove them from the analyses. The main reason that they were not removed was because they did load significantly, however low, and because they were a vital part of the present theory of

neurobehavioral functioning.

Upon further examination of the structural models, several suggestions can be offered as to why these two elements did not work well with this factor, and thus the consequential "stress" seen in the following models. First of all, these two measures may not be very eloquent assessment devices for the attribute trying to be assessed. Second, they may have contained excessive measurement error. Third, the other three elements on this factor were from the same instrument, and thus they are more closely related (average r for the three items was .80) to one another than these two. Fourth, it is entirely possible that the present theory needs revision, or possibly just better methods of assessing those qualities need to be found and used. Whatever may be the case, these two elements did not fit this factor as well as one would have liked. Nonetheless, they were allowed to remain in the analyses throughout the initial model fitting procedures. After the initial testing procedures post-hoc analyses were performed and here these two "non-fitting" elements were removed. Results of this action are discussed below.

Models Tested on Entire Sample (N = 608)

From the outset it was decided to test the three models on the entire sample. This way each of the models could be examined for fit for the sampled population. Afterwards the specific hypothesis testing could be conducted for each specific group. Therefore, results of the investigation of the various models are presented in a specific order. First, results are reported for the three models using the entire sample. Following this, the three models were tested on each of the three groups separately. The overall finding for the three models tested was that Model 1 best fit the data (see below), therefore, sex differences were investigated only at this level in order to keep to a minimum the number

of models tested (i.e., to maintain economy in the study and to minimize possible Type I errors).

The model presented in Figure 1 was tested with all subjects in the study in order to verify the present working theory concerning overall behavioral regulation. All parameter estimates and fit indices indicate that Model 1 fits very well for the entire sample (see Figure 12); that is, parental externalizing psychopathology (ASB and LAPS), parental physical activity level, levels of distractibility and attention, socioeconomic success, and intelligence, together, impact or "cause" overall offspring behavioral regulatory functioning. In this model, however, the data suggest that it is parental externalizing psychopathology (in this case antisocial behavioral and drinking problems) and socioeconomic achievement that are the statistically significant causal agents (see Figure 12). Parental temperament characteristics (attention, persistence, and activity) and intelligence do not significantly contribute to this model of offspring behavioral regulation.

Model 2 was next tested on the entire sample. Figure 13 reflects the theory and empirical evidence backing this model. Here conduct problems (behavioral dysregulation of an aggressive, societally offensive nature) have been separated from attention and neurobehavioral functioning. As with Model 1 (figure 12), all parameter estimates and fit indices suggest a good model. However, the output from the standardized solution suggests that the Neurobehavioral element is not working well with this model. Specifically, the Neurobehavioral component loads very weakly on this factor compared to the HADI indicators (.1 for Neurobehavioral compared to .8 for HADI); both conduct problems and oppositional behavior load similarly on their own factor (.8 and .9 respectively). The overall model fits very well, but this element appears as a very weak

indicator of functioning. In fact, the HADI indicator had to be fixed in order for the model to run, otherwise the Neurobehavioral element severely "disturbed" the model. This will be discussed in greater detail below.

Only the parental externalizing psychopathology and parental hyperactivity paths were significant in this model. Thus, the model suggests that parental externalizing psychopathology is significantly impacting offspring conduct problems, and that parental physical activity is the most significant casually related construct to offspring biopsychobehavioral functioning (see Figure 13).

Next, Model 3 was tested on the entire sample. This too was adequately estimated and all fit indices were sufficient (see Figure 14). Again, as in Model 2 (figure 13), the Neurobehavioral element caused undo stress to the model, and the standardized solution suggests that it should be removed from the model. To achieve some level of parsimony due to the stress of this element, several of the Neurobehavioral error terms (TE) were permitted to correlate with one another; these were IQ with language adequacy, and IQ with gross motor development.

As Figure 14 shows, this model fit very adequately with only the minimal "fine tuning" reported. However, the predictors varied somewhat surprisingly different than the preceding model would have suggested that they would. Specifically, parental externalizing psychopathology is still significantly causally related to conduct problems and HADI, but socioeconomic success was not significantly involved in the model. Parental IQ, however, emerged as a significant predictor of the Neurobehavioral construct of behavioral functioning. This would imply a causal relationship between parental cognitive functioning (IQ) with offspring cognitive functioning.

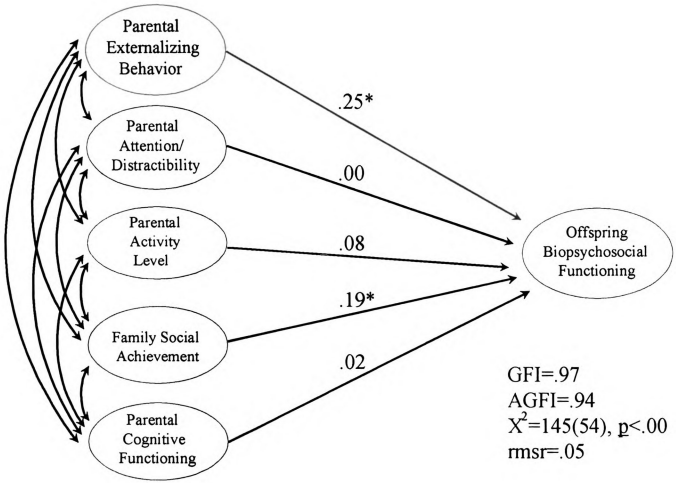


Figure 12. Behavioral Regulation for Entire Sample (N=608): Structural Model 1

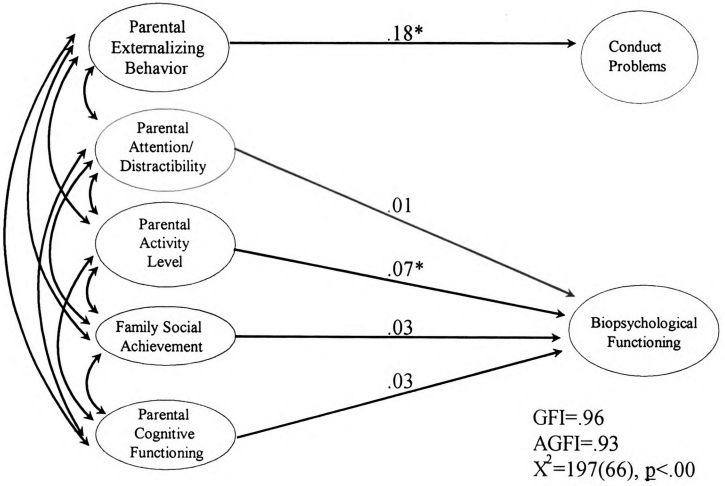


Figure 13. Behavioral Regulation for Entire Sample (N=608): Structural Model 2

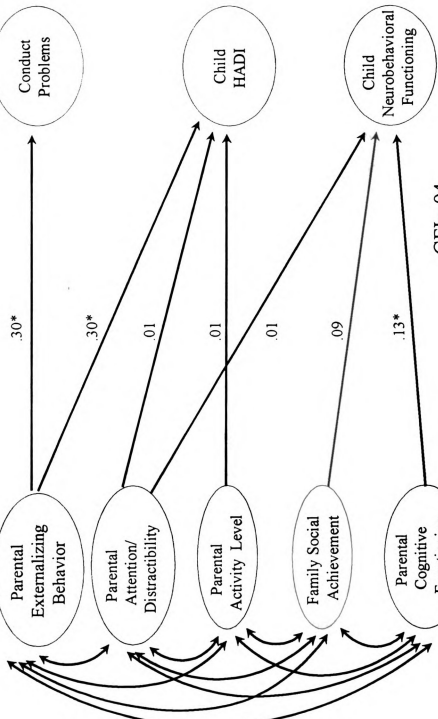


Figure 14. Behavioral Regulation for Entire Sample (N=608): Structural Model 3

From these findings, one could suggest several "overall" or "general" aspects of the three models before discussing group differences. First and foremost, it seems that the Neurobehavioral component in these models is causing more "stress" than is empirically desirable. It is possible that the Neurobehavioral component as defined in this theory was not adequately measured in this sample. Or perhaps the measures used were not sufficient to "tap into" this "Neurobehavioral" component of regulatory behavior. Or it may even be that this component of the model just does not fit well with the theory and thus the theory may need revision. In this study one can not adequately address this situation, but it does encourage one to find better Neuropsychological measures to assess the various components of Neurobehavioral functioning.

The second overall or general finding was that parental antisociality and drinking problems are heavily involved in behavioral regulation, and it seems from Model 2 and 3 that they are mostly involved with similar childhood conduct problems. One of the main hypotheses of this study was that parental externalizing psychopathology would be significantly involved with similar offspring behavioral regulation; these models begin to verify this hypothesis.

Third, as we have found in other investigations on this project, sociodemographic indicators are involved in a child's behavioral expression, usually lower behavioral regulation being highly associated with lower sociodemographic conditions. From previous similar analyses using regression techniques (Ellis, 1993; Ham et al., 1994), these sociodemographic indicators are fairly robust predictors of offspring behavioral functioning even in the presence of parental externalizing psychopathic indicators.

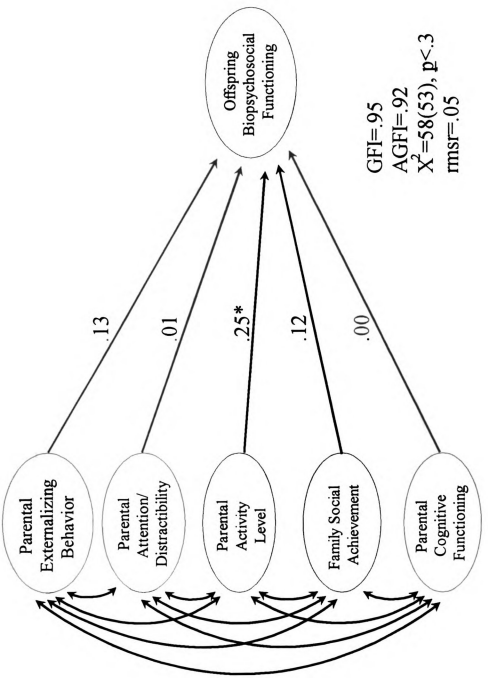


Figure 15. Behavioral Regulation for Sons of Controls (N=182): Structural Model 1

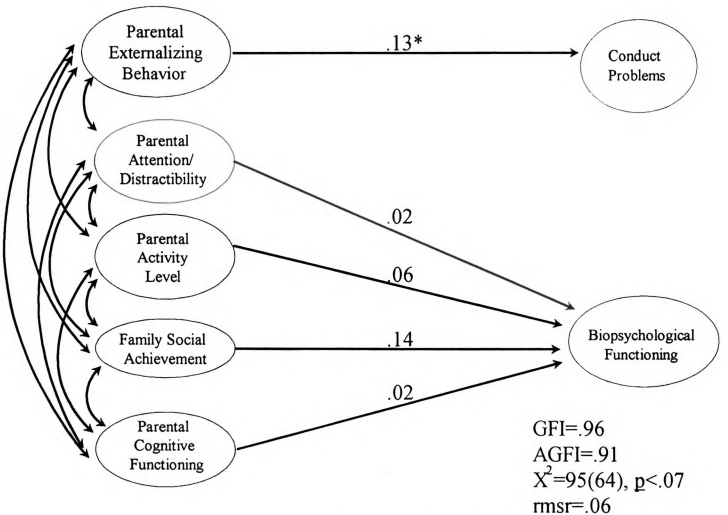


Figure 16. Behavioral Regulation for Sons of Controls (N=182): Structural Model 2

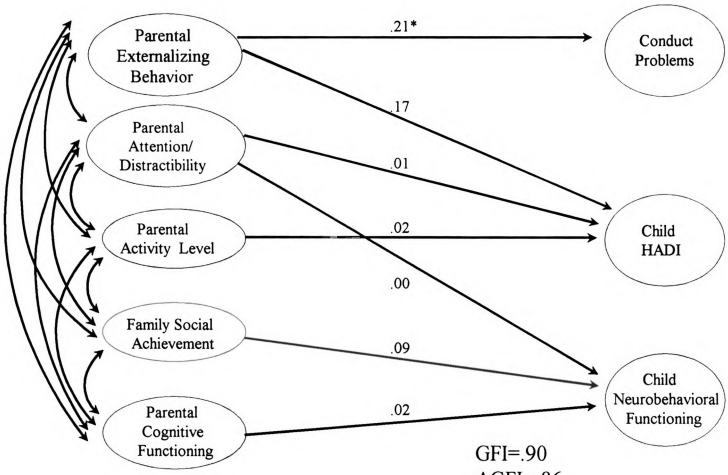


Figure 17. Behavioral Regulation for Sons of Controls (N=182): Structural Model 3

Controls (n = 182)

Figures 15-17 report the specific results and fit indices for each of the three models as they were tested on the group of control subjects. Findings are similar as those for the entire sample in some cases, thus suggesting that the models represent adequate measures of general behavioral regulatory functioning in "normal" families.

Results for Model 1 (see Figure 15) suggest that this model fits very well; it differed, however, from the overall model run in three ways. First, the only significant causal pathway was Parental Activity level. Second, it was necessary to allow two residual error terms (TD) to correlate in the socioeconomic achievement variable (income and SES); this procedure greatly improved the fit of the model. Finally, the smaller N for this group the chi-square test was non-significant (with the entire sample chi-square is too sensitive a test to be an adequate fit index), which further suggests a good-fitting model.

Model 2 (Figure 16) may give some insight as to why the pathway predictors changed as they did for controls in Model 1 in comparison to the overall sample. As Figure 16 shows, Parental Externalizing Psychopathology is a significant casual indicator of offspring conduct regulation, and Socioeconomic Achievement is also strong, (however, it was just shy of significance). Similar to Model 1, the error terms for SES and income were permitted to correlate in order to produce the best fit. However, the modification indices (MI) also suggested one other change that proved interesting. MIs for the Gamma coefficients suggested allowing Parental Activity level to predict Conduct Problems; this improved the fit in several ways, but interestingly enough it also removed the effects of Parental Externalizing Psychopathology, and even more interestingly it caused the pathway from Activity level to Biopsychological functioning to become very

strong and highly significant. The changes in path coefficients are in parentheses in Figure 16.

With Model 3 revealed somewhat consisted findings as those found in Models 1 and 2. However, here the model does not fit so well, and part of the problem could be attributed to the Neurobehavioral component of the measurement model; also attributed to it is the fact that the overall structural model does not seem to be very adequate (Figure 17). Again, the two error terms for SES and income were highly correlated. Parental Externalizing Psychopathology was a significant casual indicator of offspring Conduct Problems, and nearly so for HADI, but none of the other pathways even approached significance.

Non-Antisocial Alcoholics (n = 282)

Results for the non-Antisocial group looked similar to those for controls. First Model 1 was run for this group. A graphical and statistical representation of the outcome can be seen in Figure 18. This model was actually quite unremarkable. Although fit indices reveal a good fit to the model, none of the causal pathways predicted were significant. (However, it should be noted that Parental Externalizing Psychopathology has a very large path coefficient). What this amounts to is this: The basic measurement model maintains its robust quality for this group, but the hypothesized causal routes of offspring behavioral regulation are not supported.

Figure 19 shows the outcome for Model 2 for this group. Again, the data suggest a satisfactory fit, however, this model seems to get at the individual components of behavioral regulation better than the overall model seen in the preceding figure. In this run we still see that Parental Externalzing Psychopathology is the most significant causal

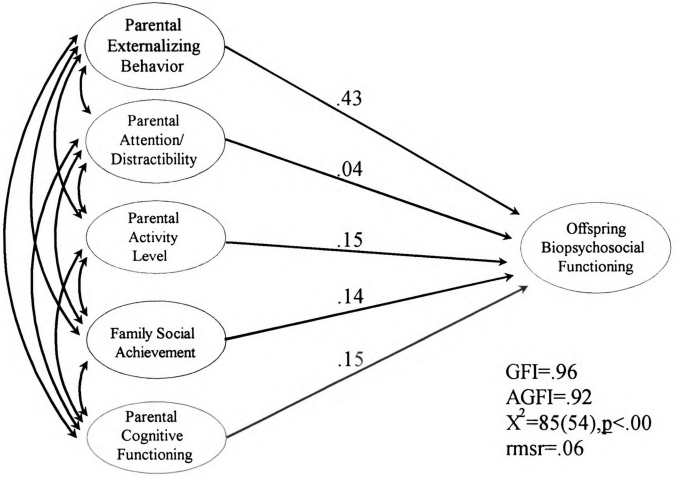


Figure 18. Behavioral Regulation for Sons of Non-Antisocial Alcoholics (N=282): Structural Model 1

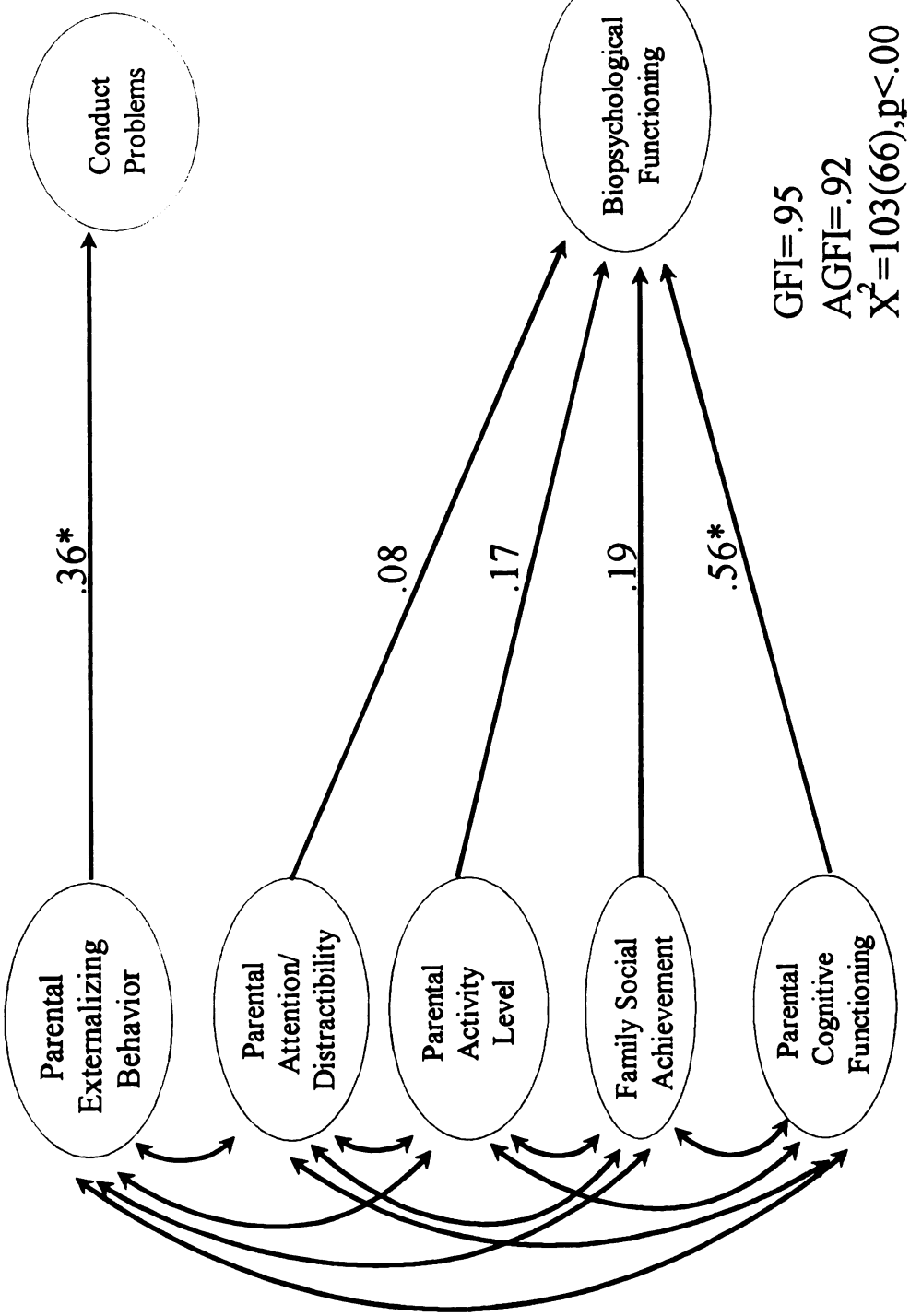


Figure 19. Behavioral Regulation for Sons of Non-Antisocial Alcoholics (N=282): Structural Model 2

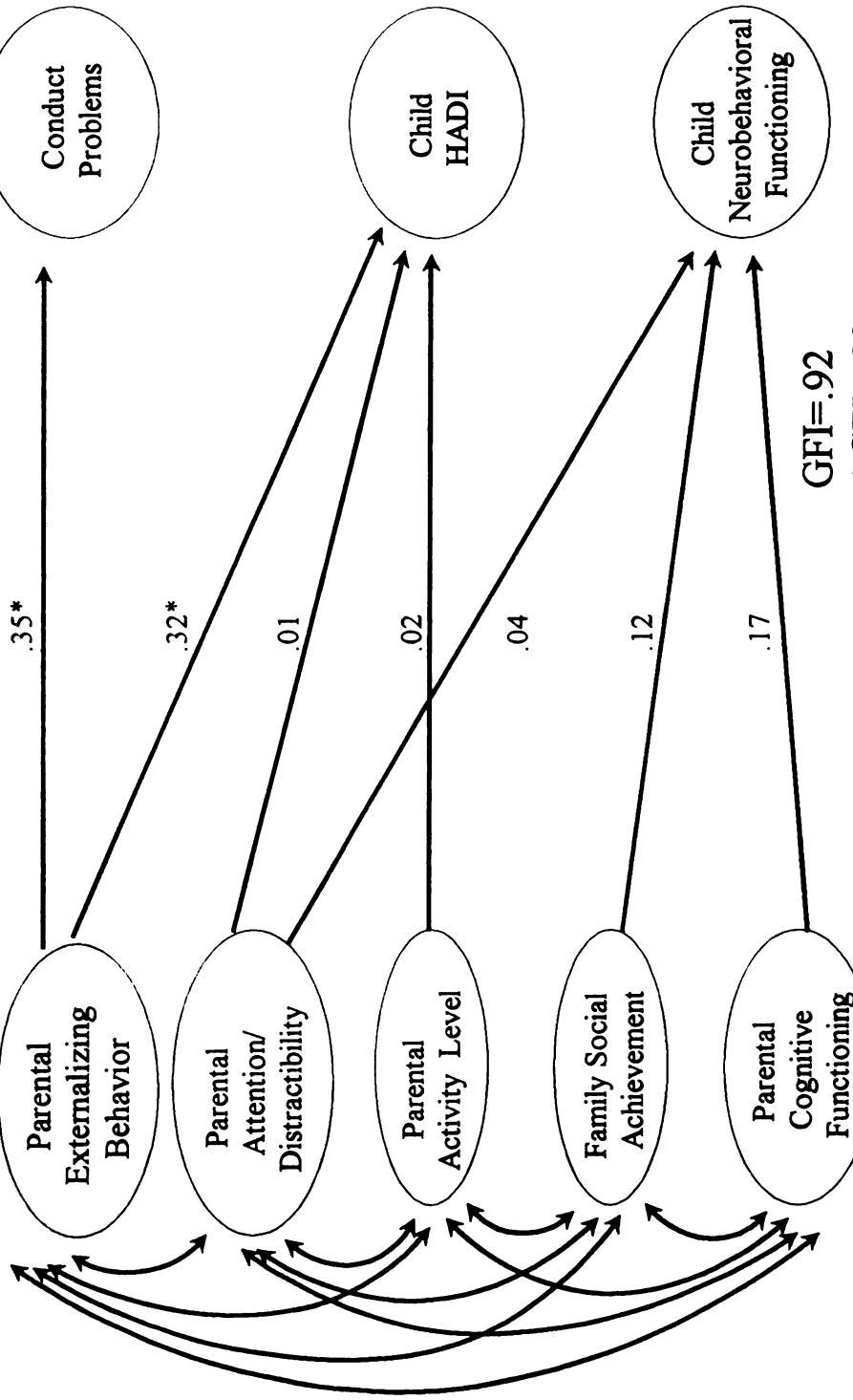


Figure 20. Behavioral Regulation for Sons of Non-Antisocial Alcoholics (N=282): Structural Model 3

variable involved in aggressive conduct regulatory behavior (similar to controls); however, what is remarkable here is that Parental IQ is a strong predictor of attention and Neurobehavioral regulatory functioning rather than Socioeconomic Achievement.

When Model 3 was tested on this group nearly the same results were apparent (Figure 20). Fit indices for the model reveal a somewhat less substantial model, however significant, and Parental Externalizing Psychopathology is not only a significant predictor of offspring conduct regulatory behaviors but also attentional-deficit-activity regulation (seen in controls also). One thing that was remarkable here is that when the individual components of the model were broken up (i.e., Conduct Problems, HADI, Neurobehavioral), offspring externalizing behaviors (conduct problems) were predicted from parental externalizing behaviors (antisociality and drinking problems) and nothing else in the model seems important. This suggests refinements of the present theory about the relationship that parental levels of functioning have with offspring functioning within the non-antisocial group.

Antisocial Alcoholics (n = 144)

Figures 21-23 report all significant findings and fit indices for models tested on this group. Of all models tested on the three groups, Model 1 for AALs was most sound from both measurement and structural perspectives. However, the predicted pathways failed to be seen for this group of individuals (with one exception). Although overall fit indices were acceptable they were marginal compared to the same model for the other two groups. Figure 21 reveals that several of the paths were quite large by comparison to the other groups, however, only Family Social Achievement was a significant causal predictor of general offspring behavioral regulatory functioning, and Parental Externalzing

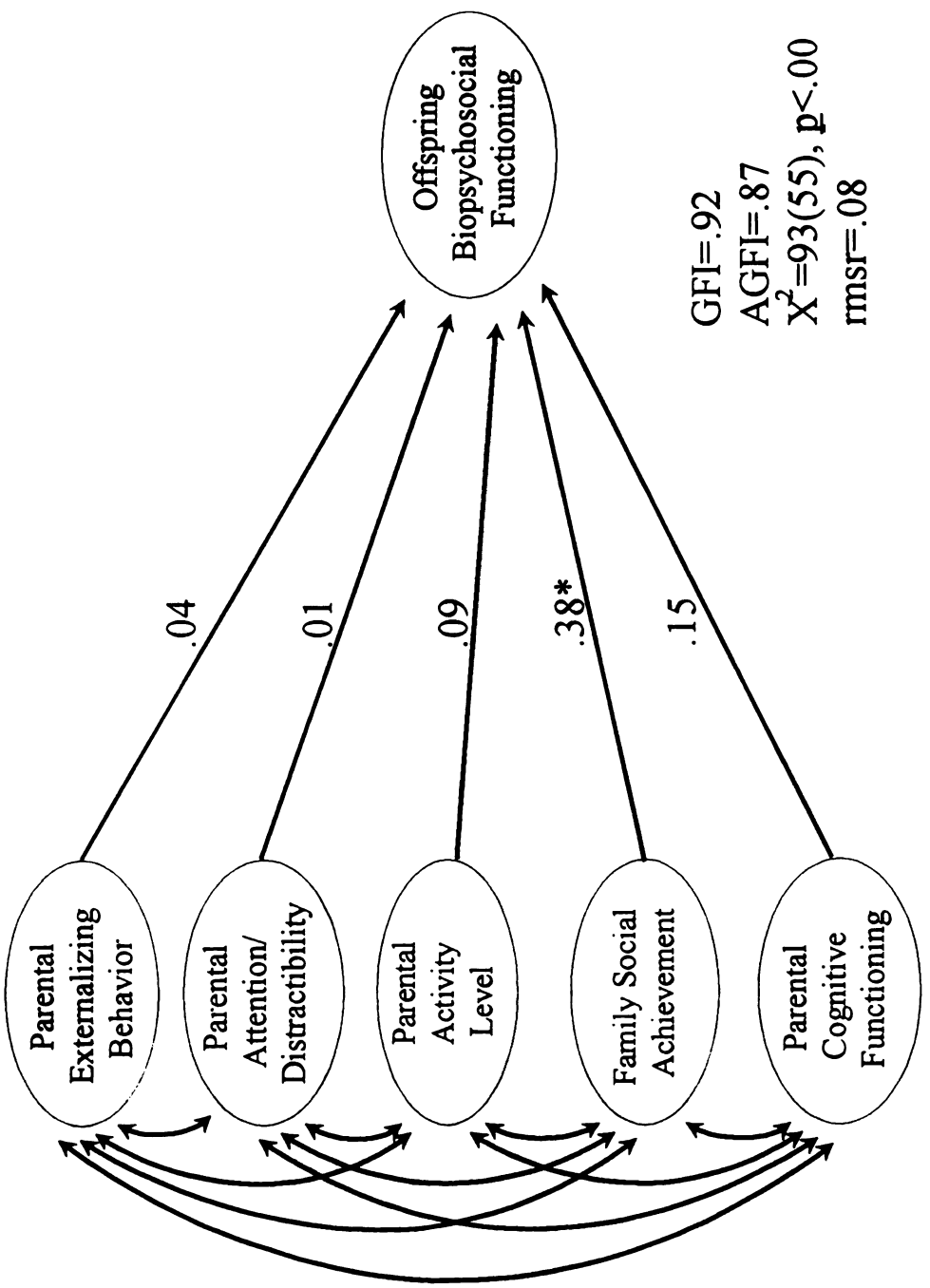


Figure 21. Behavioral Regulation for Sons of Antisocial Alcoholics (N=144): Structural Model 1

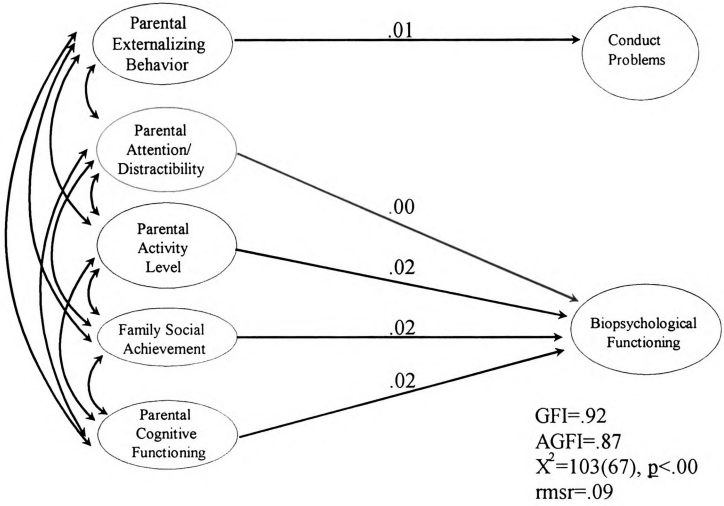


Figure 22. Behavioral Regulation for Sons of Antisocial Alcoholics (N=144): Structural Model 2

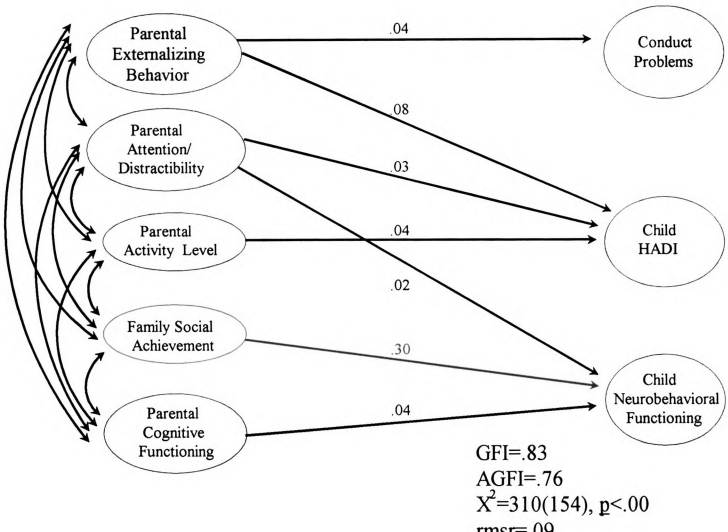


Figure 23. Behavioral Regulation for Sons of Antisocial Alcoholics (N=144): Structural Model 3

Psychopathology was not even close to being a significant contributor; this was an unexpected finding. It was expected, based on previous analyses with these groups (Ham et al., 1994), that parental externalizing psychopathology would be a significant causal factor to offspring behavioral regulation.

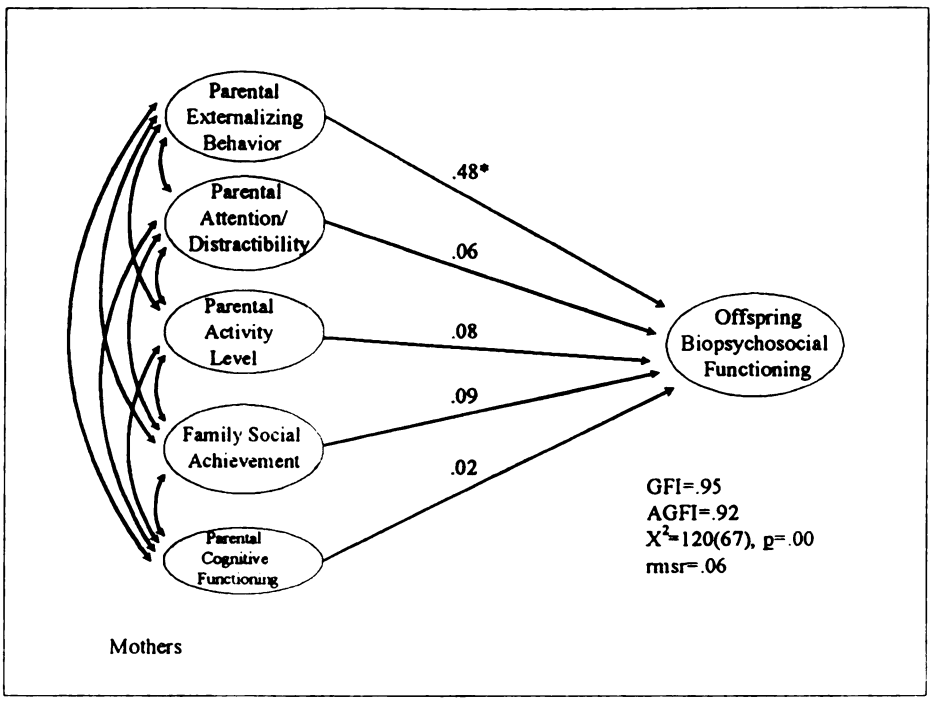
When behavioral regulation was broken down into the two order model (see Figure 22) all effects for sociodemographic success disappeared, and none of the other parental variables were contributing to offspring regulation. As one can see in the figure, none of the path coefficients were sizable, nor were any of them significant. Again, in light of the fact that the goodness-of-fit indices indicate a good model, what these data suggest is that the measurement model is quite robust, but that the hypothesized pathways (i.e., the structural portion of the model) are not accurate for this group of children.

Following this the three tiered model was conducted for this group. Figure 23 reports the basic results. This model was not a good fitting model, however, the basic measurement and structural components were fairly sound. Modification suggestions were minimal and ineffective, so it was concluded that this model really doesn't fit for this group of at-risk children.

Sex Differences in Model 1

After conducting the initial analyses on the entire sample and then the individual groups the best fitting models were tested separately for mothers and fathers. Again, this model was tested for the entire sample of men and women separately and then similarly for the three groups.

Results for model runs by sex for the entire sample are reported in Figure 24. Overall findings would indicate that **paternal** antisociality and drinking problems, along



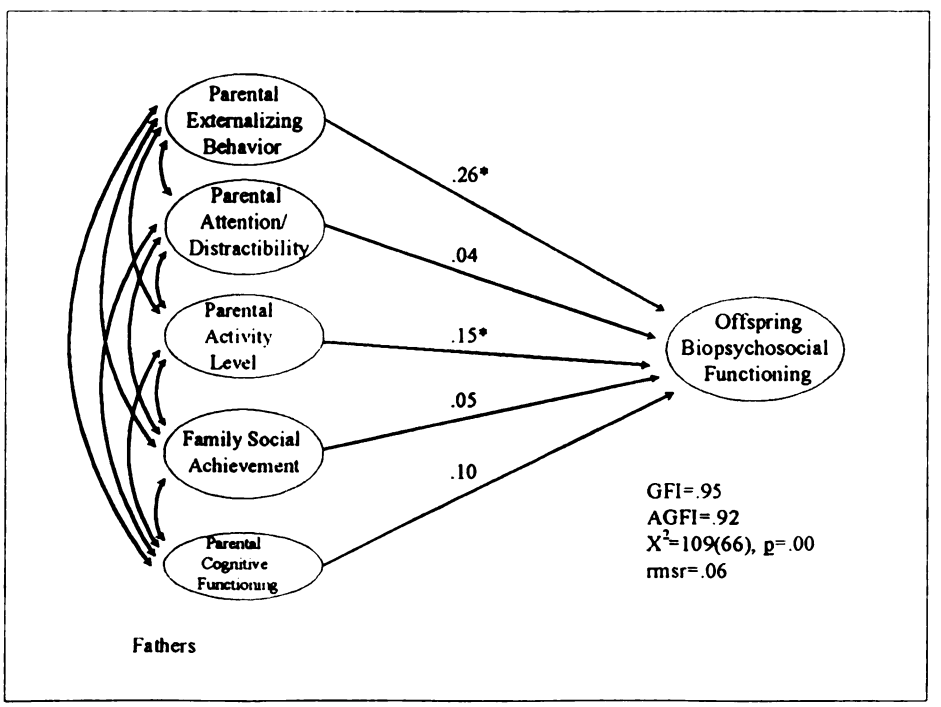
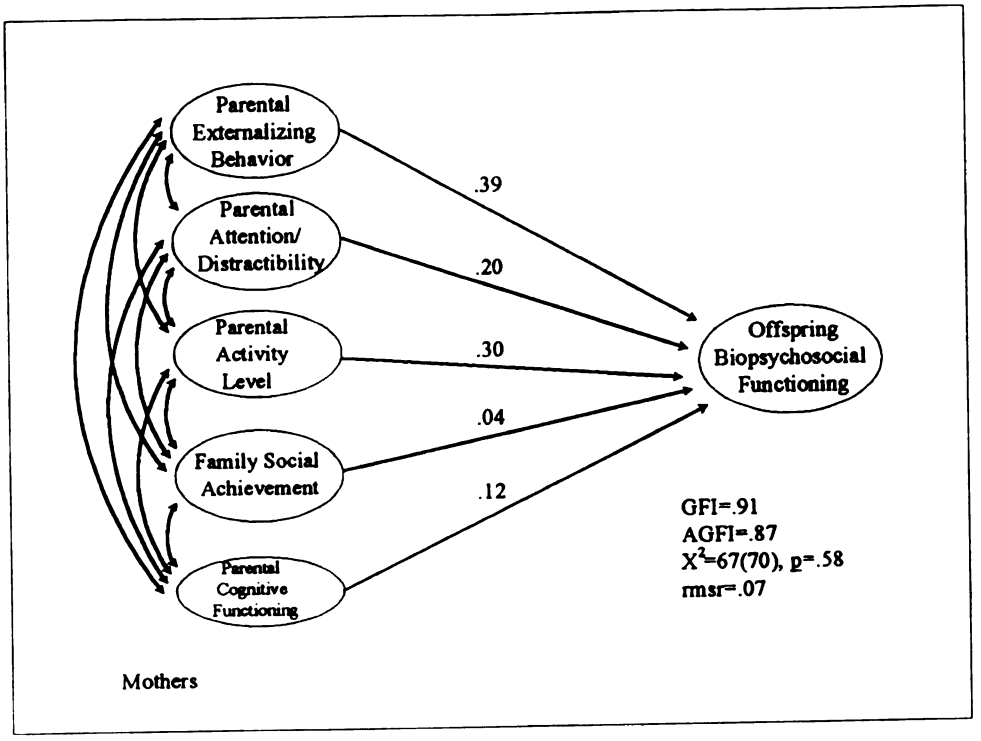


Figure 24. Behavioral Regulation for Sons of Parents in Entire Sample (N=304): Structural Model 1

with level of physical activity, are the most significant predictors of offspring behavioral regulation for the entire sample. For mothers the findings were consistent with those in the previous study (Ham et al., 1994). **Maternal** indices of externalizing psychopathology are most significantly associated with offspring behavioral functioning. Although both of these models fit fairly well, there was a lot of "noise" in their outcomes. In other words, several of the elements in the models were not very stable, and in order to maintain some stability to the model error terms for these elements had to be permitted to correlate with one another. Particularly, elements such as education and income in the Social Achievement variable were most "noisy".

Controls

Next, Model 1 was re-run for parents separately in the control group; final outcome statistics can be seen in Figure 25. For mothers, the goodness-of-fit indices indicated a well fitting model, but there were no significant causal pathways. However, Externalizing Psychopathology and Activity level were nearly significant (t = 1.8; t = 1.77 respectively), and this will be important, as will be shown, in the post-hoc analyses when dealing with the "troubling" Neurobehavioral elements. For fathers in this group the model fit was nearly identical to that for mothers and, similar to mothers, the causal pathway for Activity Level was nearly a significant one (t = 1.87). So in this group of children, it seems apparent that parental levels of physical activity have some bearing on their own levels of overall regulation. Remember, this variable was the only significant predictor (however small - see Figure 15) of offspring functioning for the combined parental run.



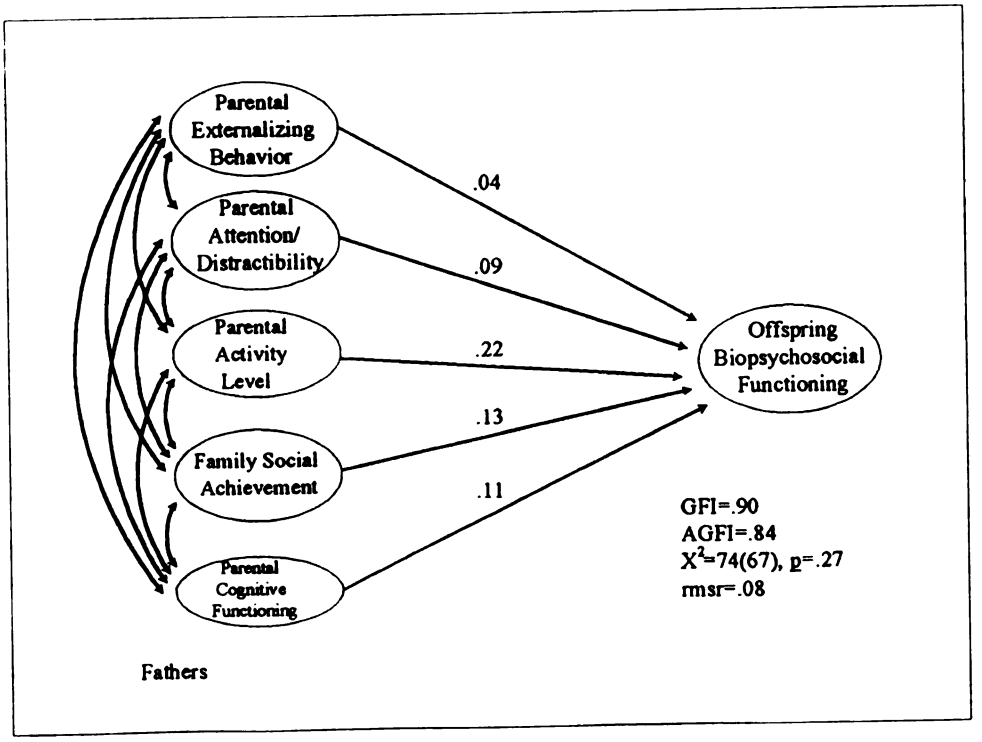
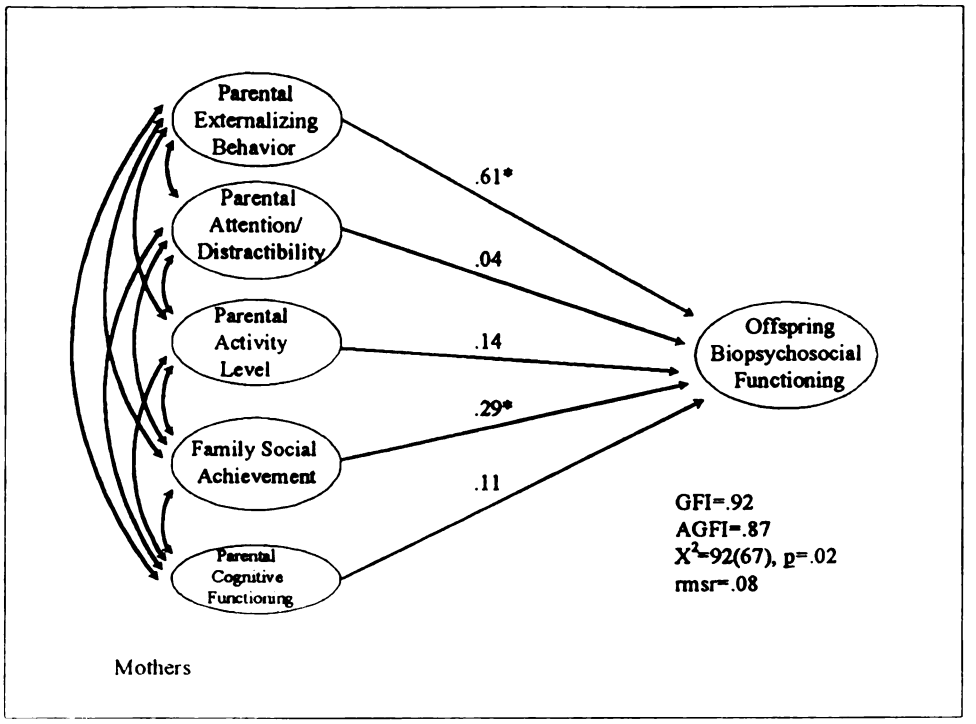


Figure 25. Behavioral Regulation for Sons of Control Parents (N=91): Structural Model 1



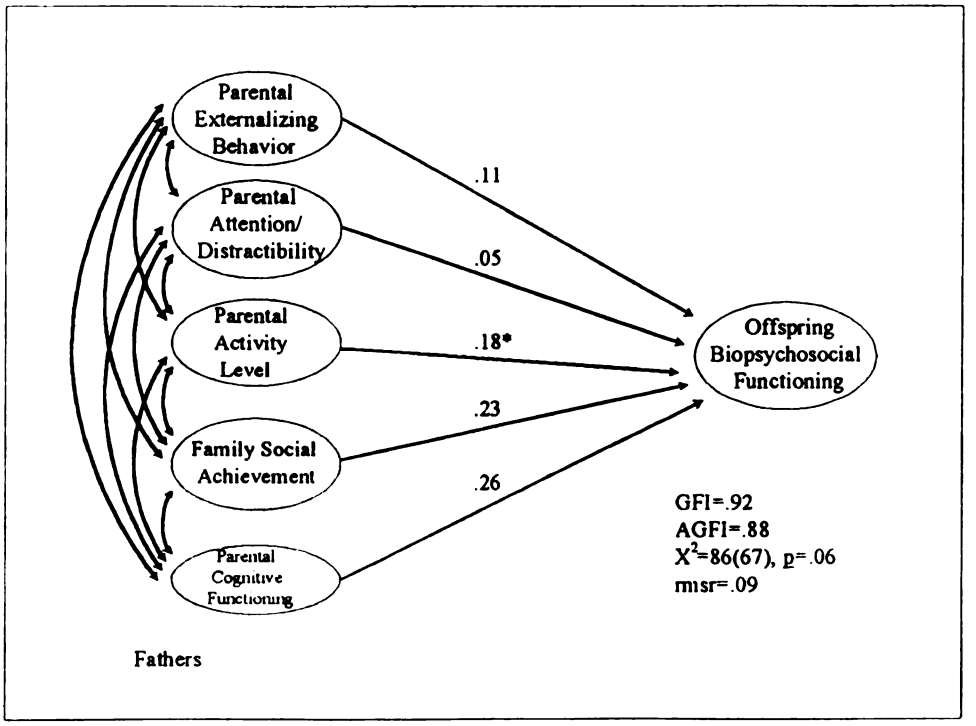


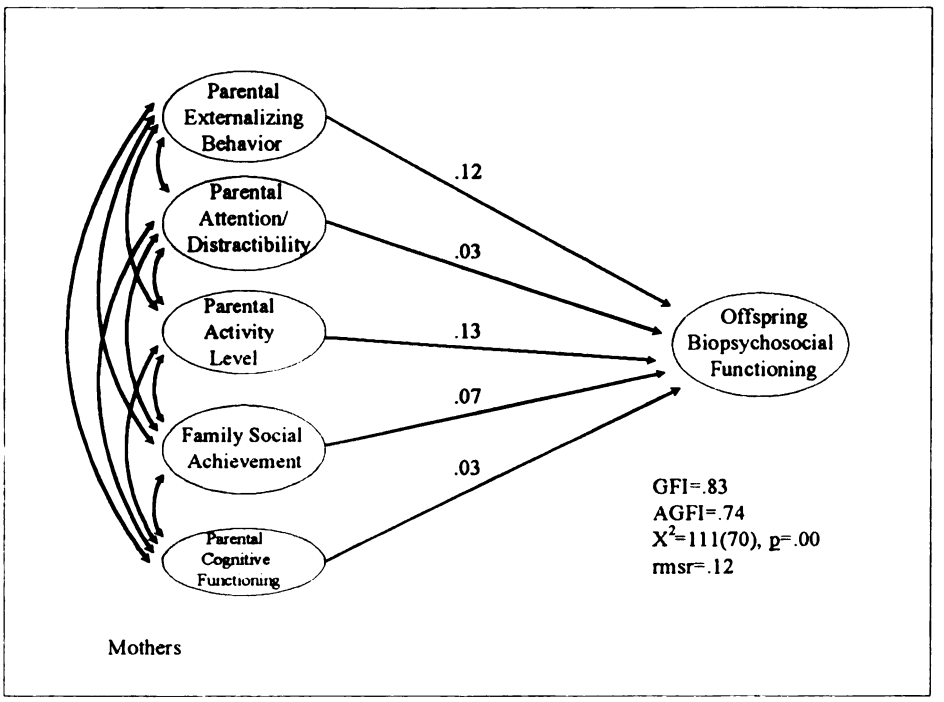
Figure 26. Behavioral Regulation for Sons of non-Antisocial Alcoholic Parents (N=141): Structural Model 1

non-Antisocial Alcoholics and Their Wives

The model fittings for this group of parents, as well as the causal pathways leading to offspring behavior regulation, were similar to those for control parents, but causal pathways were much more pronounced for this group (see Figure 26 for results). When the model was tested for mothers in this group both Externalizing Psychopathology and Social Achievement were significant predictors of offspring regulation, and Activity level was nearly a significant one as well. For fathers in this group the findings were likewise similar to fathers in the control group, and again, the casual pathways were more significant. Paternal Activity Level was the significant predictor of offspring behavioral regulation; none of the other pathways even approached significance for this group of men.

Antisocial Alcoholics and Their Wives

The overall findings for this group when parents were combined (see Figure 21) was that Family Social Achievement was a significant predictor of offspring behavioral functioning. The goodness-of-fit indices for this model were adequate, but not real robust. However, when the model was run on parents separately findings were fairly unremarkable and none of the pathways leading to offspring outcomes were significant. Figure 27 shows findings for mothers and fathers in this group. As can be seen, fit indices are very poor for mothers and thus, causal pathways are irrelevant. For fathers, the GFI was barely adequate but the Chi-square was significant, so one could conclude that, although this is only a fair fitting model it does meet the necessary requirements as a decent model. However, although the overall model for fathers was significant none of the causal pathways were significant.



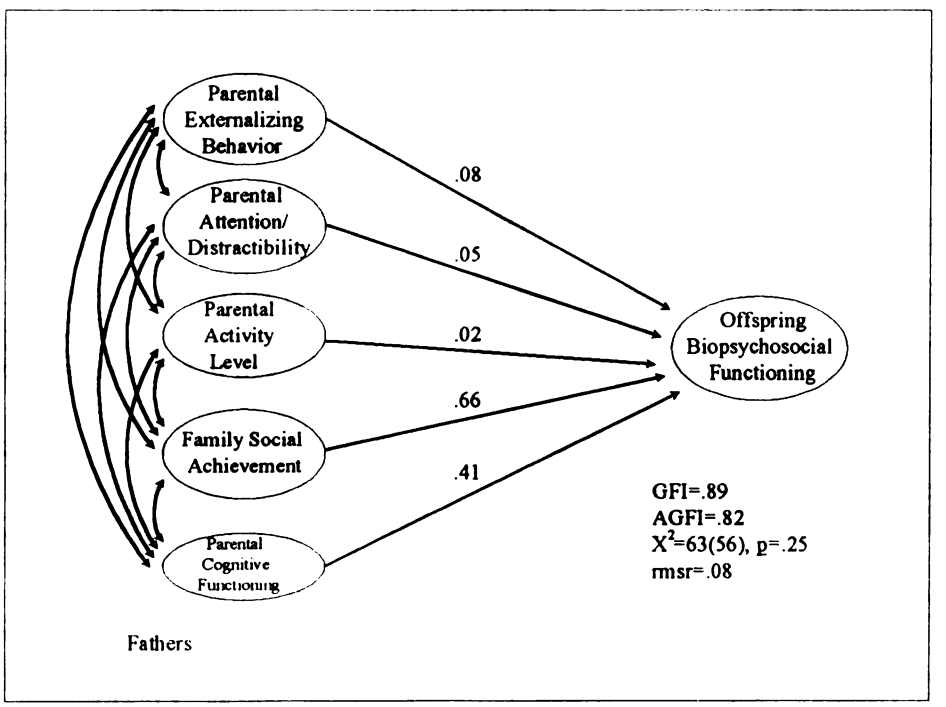


Figure 27. Behavioral Regulation for Sons of Antisocial Alcoholic Parents (N=72): Structural Model 1

Conclusions and Suggestions

Based on post-hoc analyses (see Appendix A) it may be concluded that neither the Neurobehavioral variable nor its constituent parts were really troubling the model nearly as much as originally suspected. However, when Model 1 was run for parents separately to uncover sex differences, it became very apparent that this element, although not nearly as distressing to the models as first suspected, does not belong on any of the factors. Specifically, in all of the runs for Model 1 for parents separately, the Neurobehavioral element had factor loadings that hovered around zero. Several changes could be proposed to the model that could be tested in subsequent studies.

First of all, antisociality and drinking problems could be separated and treated as single indicators. These variables were combined together into an overall externalizing psychopathology variable within the antisocial, alcoholic milieu. While this makes good theoretical and empirical ($\underline{r}$ = .50) sense, it would be interesting to examine the individual effects of antisociality and alcoholism on offspring behavioral regulation. This approach is suggested after seeing the consistent pattern that antisociality played on offspring behavioral functioning in other similar types of analyses (Zucker et al., 1993; Ham et al., 1994). Secondly, in all model runs the parental attention and distractibility variable didn't seem to be contributing to any of the predictability of offspring regulatory behavior. And in like manner, the variable that measured parental activity level seemed to **want** to be more involved in the models. For instance, in many of the model runs, modification indices suggested that this variable (Parental Activity level) be allowed to predict offspring conduct problem behaviors. As noted above, this was allowed in several of the models, and pathways significantly changed. However, a new model could be constructed based

on these outcomes with the Attention variable omitted and the two Externalizing Psychopathology variables separated. Another suggestion would be to remove the Neurobehavioral element all together and concentrate on hyperactive and conduct dysregulatory behaviors. This suggestion is made based solely on the assumption that the instruments available were not adequate or sufficient to assess the neurobehavioral component of functioning that was of interest, not that necessarily that the original theory was unsound. The theory is most likely adequate to explain this phenomenon, but the ability of the instruments used to ascertain a good measure of neurobehavioral functioning was lacking. In the "perfect" study one should use valid and reliable neuropsychological tests to assess this component of regulatory functioning in at-risk children of alcoholics.

CHAPTER V

Discussion

Focus of the Study

The focus of the present study was twofold: 1) to investigate the incidence of biobehavioral dysregulation in two groups of SOMAs (i.e., sons of antisocial and non-antisocial alcoholics) comparing them to boys who are not currently at such risk and 2) to explore several hypothesized causal variables assumed to be involved in the precarious developmental pathway that SOMAs are traversing. In conjunction with these study objectives, the study also explored the various detrimental outcomes that SOMAs are currently experiencing in light of those that they may consequently experience (i.e., those their parents are currently experiencing); outcomes such as severe substance abuse, increased levels of psychopathology, poor academic outcomes, lower socioemotional functioning, and substandard living conditions due to all of the above.

There are several uniqueness of the present study which permit generalizability of findings to a large portion of the general population. First of all, the target children in this study were much younger than those used in similar studies. In fact, this was one of the first scientific investigations of the incidence and etiologic causes of hyperactivity and conduct problems in conjunction with parental alcoholism in *pre-school* age boys. [The age range of the boys in the present study was late infancy (2 1/2 years) to early childhood (6 1/2 years)]. Findings reported here support some of the *hypothesized* early beginnings of behavioral dysregulatory functioning of children of alcoholics (Schuckit et al., 1987; Tarter et al., 1990). Another unique and beneficial quality of the present sample is that the alcoholic families used were community drawn rather than clinically drawn. Thus, the

developmental underpinnings of adult alcoholism in conjunction with childhood biobehavioral regulatory development could be examined. In that younger alcoholics rather than chronic alcoholics were used, this also offers a firsthand glimpse at the effects of early alcoholism on family and offspring functioning. Finally, this study used an asymptomatic group of families as controls; this strategy permits generalization of study findings to a large segment of individuals in our society.

These recruitment strategies also permit us to draw some interesting suppositions about possible genetic implications of alcoholism and alcohol related psychopathology with regards to offspring behavioral regulatory functioning in two sub-groups of alcoholics and their children. The effect of alcoholism on offspring outcome was investigated from an early developmental perspective, thus permitting an early look at the unfolding of biobehavioral dysregulation in a group of severely at-risk individuals drawn from average, white, lower class communities.

General Summary of Study Findings

The current study findings suggest several conclusions. First, the data suggest that the high level of externalizing psychopathology seen in the present group of alcoholics and their wives, as well as the sociodemographically deprived rearing environment that they are currently experiencing, are two substantial etiologic factors involved in the hazardous outcomes available to their at-risk offspring. Poor biobehavioral outcomes seen in sons of AALs in the present study were: 1) significant amounts of attentional difficulties, 2) high levels of physical activity, 3) high levels of distractibility and impulsivity, 4) higher incidence of conduct problems similar to ODD and CD, and 5) lowered cognitive functioning as reflected by IQ assessments and language developmental status compared

to sons of nAALs and controls. While sons of nAALs did exhibit similar behavioral and cognitive deficits, deficits could only be explained in comparison with sons of AALs rather than sons of controls, as originally expected. In fact, more than not, sons of nAALs and sons of controls were similar in behavioral regulation and cognitive functioning. This over-arching finding strongly agrees with Zucker et als. (1993, 1994) notion that the AALs' offspring is at significantly greater risk for, not only alcoholism later in life, but also for problem behaviors in childhood and adolescence due to the comorbid status of the parents. What this implies is that children being reared in an alcoholic family are not so much at risk for poor outcome as are children being reared in an alcoholic family whose parent(s) exhibit antisociality to significant degrees. AALs in this study presented antisociality (on average) at high enough levels to meet a DSM-III-R diagnosis of APS.

The second major finding of the present study was that hyperactive boys are significantly more impaired on cognitive functioning and developmental measures compared to non-hyperactive boys. Also, the large majority of hyperactives in the present sample were being reared by alcoholic fathers (85%) and the large majority of those were found in the AAL families (49%). Although sampling procedures bias these results, they do substantiate other findings in the literature (see Hinshaw, 1992 for review) suggesting that hyperactive children many times grow up with problem behaviored parents (e.g., alcoholic, antisocial).

Structural equation modelling results suggest that parental antisociality and alcoholism are primarily responsible for conduct problems similar to ODD and CD, while sociodemographic variables seem to be causally related to cognitive functioning and HADI symptomatology. None of the neurobehavioral measures figured prominently into the

models of biobehavioral regulation in any of the three groups of children and their parents. One reason for this lack of substantiation of my neurobehavioral theory may be a psychometric one (i.e., measures used to assess neurobehavioral functioning were not adequate in addressing neurobehavioral characteristics and therefore standardized neuropsychological tests/battery should be used), and another may be a neurological one. For instance, the neurobehavioral subtilities sought after in the present investigation may not be readily accessible in children during the preschool years. One reason for the inaccessibility of neurobehavioral functioning in preschoolers is the fact that neural development is still occurring and fluid, and therefore tapping into neuropsychological or neurobehavioral abnormalities at this early age may be difficult. For instance, Luria (1966) points out that the frontal lobes are still in the process of maturing until adolescence, and therefore, they may not be observable before that time. Tarter, Laird, and Moss (1990) point out that due to the still maturing frontal aspects of the neocortex during childhood, frontal-lobe related dysfunctioning in COAs may not be readily discernible until after the pre-adolescent period. However, the cognitive deficits present in preschool age boys in the present study bear out the frontal hypothesis, and therefore may be interpretable as neurobehavioral risk for alcoholism.

The combined efforts of behavioral scientists, and findings from the present study, strongly suggest that poor psychopathic status (in the present case alcoholism and antisociality), low socioeconomic status, and poor cognitive functioning of alcoholic men and their wives are responsible for creating and maintaining a precarious developmental pathway for a group of at-risk offspring; a pathway ultimately leading to similar and possibly even more severe behavioral problems, substance abuse problems, and poor social

consequences, etc. One of the objectives of this study was to replicate the general findings in the child problem behavior and alcoholism literatures that attest to the onset of poor developmental outcomes for COAs.

Children of alcoholic parents, especially behavior disordered alcoholic parents, are at heightened risk for substance abuse and severe behavioral problems (Cloninger, 1987; Cotton, 1970). The increased incidence of alcoholism and behavioral problems in COAs has led to genetic implications for the etiology of alcoholism (Schuckit et al., 1987). However, most researchers agree that a strictly genetic explanation for alcoholism or behavioral problems associated with alcoholism is highly unlikely. Rather, the general consensus among alcohol researchers is an interactive explanation for the cause of alcoholism and alcohol related pathology - an interaction between an individual's genetic composition and the developmental environment. Lerner (1991) refers to this interaction as the "fusion" of an individual's genotype and the environment surrounding him or her. Moreover, individuals have unique combinations of genetic makeup and environmental circumstances (Gottlieb, 1991), and therefore, individual variation of outcomes will be unique, and not necessarily or specifically a direct reflection of either one's genetic makeup or one's developmental environment.

In the present study, offspring biobehavioral regulatory outcomes were found to be significantly poorer in the two groups of boys being reared by alcoholic fathers (e.g., sons of **antisocial** alcoholics and sons of **non-antisocial** alcoholics) compared to children of asymptomatic fathers (controls). These findings substantiate claims that SOMAs are significantly more attentionally deficient, more hyperactive, more impulsive, have lower cognitive functioning, and exhibit more conduct disordered behaviors than "normals"

(Tarter et al., 1993; West & Prinz, 1987; Zucker et al., 1993). They also support a systems theory notion that the combined influence of genetic load for problem behaviors and the hazardous rearing environment they are connected to are collectively responsible for placing them on a detrimental developmental pathway (Fitzgerald et al., 1992; Zucker & Fitzgerald, in press).

Diagnostic Difficulties of Hyperactivity and Conduct Problems: Present Study Findings

Before discussing the possible explanations for present findings, I would like to address several more minor issues that were dealt with in the present study. First, one of the longstanding concerns of behavioral scientists regarding hyperactivity is that of nomenclature. Hyperactivity is one of those behavioral problem categories whose definition continues to undergo change, and not all of the parties involved are content with these changes. Originally hyperactivity was thought to be a vaguely definable brain disorder (e.g., Minimal Brain Disorder). Due to the observed effects of psychopharmacological agents so prevalently used in the 1960's and 1970's this line of thinking seemed appropriate. Social scientists have, for the most part, separated themselves from the medical philosophy concerning hyperactivity, and subsequently have embraced a more behaviorally oriented approach. To date, the definable characteristics of the hyperactive disorder have evolved into the present notion of AD/HD (APA, 1994), a disorder composed of hyperactivity, impulsivity, and attentional deficits, all of which are *exclusive* of aggressive/violatory behaviors.

Second, along with the difficulty of nomenclature for hyperactivity there is also disagreement about its etiology. Some have suggested that hyperactivity is a pre- or post-natally induced neurologically based disorder (Bakwin & Bakwin, 1966), others suggest

that it has neuropsychological implications (Tarter et al., 1985), some strongly argue that its etiology lies within the confines of societal influences (Schachar et al., 1989), and then there are those who insist that it exists within the context of more severe conduct problems such as ODD and CD (Hinshaw, 1992). The present study *tackled* both the issue of nomenclature and that of etiology. Consequently, several of the above mentioned ideas have been retained, while several have been discarded.

Regarding the defining characteristics of hyperactivity, most are of the opinion that hyperactivity exists in a pure form, one that is specifically marked by three diagnostic characteristics: 1) attentional deficits and distractibility, 2) physical hyperactivity, and 3) impulsivity, and furthermore, one that is distinguishable from conduct problems of an aggressive nature (APA, 1994). Notwithstanding that hyperactivity may exist in the same context with more severe conduct problems such as ODD and/or CD, it is, for the most part, still believed to be a problem behavior category in its own right. Data from the present study tend to support this notion. For instance, from a purely psychometric standpoint (i.e., confirmatory factor analysis) the data revealed that hyperactivity, attentional deficits and distractibility, and impulsivity are separate, yet highly related, constructs from conduct disordered behaviors. Although hyperactivity and conduct problems do correlate highly with one another, they did maintain individuality in the analysis. Furthermore, parental externalizing psychopathic and sociodemographic characteristics of the boys in the present study confirm Farrington et al's. (1990) notion that hyperactivity and conduct disorders maintain separate etiological influences. For example, the basic results of structural equation modelling revealed that parental externalizing psychopathology is more heavily involved with offspring conduct problems

than is sociodemographic indicators. Family socioeconomic achievement, on the other hand, was primarily responsible for HADI. Contradictory to this, however, Ham et al. (1994) found no differences in predictors of HADI symptoms and conduct problems in SOMAs. General findings for the Ham et al. study were that parental antisocial behavior was primarily involved in predicting both HADI and conduct problems regardless of the presence of demographic indicators. The differences between the two studies, however, may be found in the analytic approach. The present study used structural equation modelling (SEM), a technique which gains information concerning causal indicators in a *simultaneous* manner; the Ham et al. study, although using the same dataset, employed regression techniques; such techniques are far less able to elucidate the combinatorial effects of various suspected causal indicators, they are also not nearly as affected by shared variances across the multiple predictors as is SEM.

In another study conducted using the same sample, Jansen et al. (under review) found that maternal antisociality and family income level were the most powerful predictors of Total Problem Behaviors as reported by maternal ratings on the Child Behavior Checklist (CBCL; McConaughy & Achenbach, 1988) in boys who met clinical cutoffs for internal and externalizing problem behavior. The authors also report that paternal antisociality was a significant predictor of child internal and externalizing problem behaviors. Jansen et al. point out that these findings are surprising in that most of the analyses using this dataset find that maternal antisociality is more consistent at predicting offspring behavioral dysregulation. However, boys in this study were grouped according to presentation of problem behaviors while other studies using the same sample establish their experimental groups according to paternal diagnosis of alcoholism/abuse and

antisocial behavior (e.g., Ellis, 1993; Fitzgerald et al., 1990, 1993; Ham, 1992; Ham et al., 1993, 1994)

Parental Hyperactivity and Related Psychopathology: Offspring Outcomes

In the present study there were several assumptions made about parents of hyperactive children and the various outcomes they experience. One such assumption is that hyperactive children are biological offspring of parents who were/are hyperactive; the underlying notion was that hyperactivity has a genetic component that is passed from parent to child (Eaves et al., 1993). Another common assumption in the hyperactivity literature is that parents of hyperactive children share several consistent psychopathological states in common over the lifespan. There are several consistent findings in the problem behavior literature that document this notion. For instance, Cantwell (1972) reported that parents of hyperactives are more prone to antisociality, alcoholism and other substance abuse, depression, and generalized psychopathology. Others have noted similar findings (Goodwin et al., 1975; Morrison & Stewart, 1973). Many other studies have reported that parents of hyperactives are cognitively impaired (Hinshaw, 1992), and similarly, their children exhibit low levels of cognitive functioning (Frick et al., 1991; Weiss, et al., 1979, 1985).

Studies reporting high levels of past and present hyperactive symptomatology in parents of hyperactive children has led some researchers to insist on a behavior genetic claim for the etiology of hyperactivity (Goldsmith, 1989; Goodman & Stevenson, 1989); the idea being that parents of hyperactive children not only genetically transmit a propensity for hyperactivity, but that they stage the appropriate environment for hyperactive behavior (i.e., an environment which is *conducive* to hyperactive or acting out

behavior). Most often it is assumed that hyperactivity is passed from fathers to sons (Goldsmith, 1989) and that the once hyperactive fathers foster a hyperactive milieu for their sons to grow up in. Fitzgerald et al. (1993), buttress this claim by asserting that it is the "genetic variation within the family system" which serves to "trigger" the individual's behavioral propensity to a certain behavioral expression, whether pathologic or not. Fitzgerald and Zucker (1994) go one step further in explaining the relationship between the genotype and its environment with regard to the expression and progression of problem behaviors. They conceptualize problem behaviors (especially those found in the alcoholic family environment) within a life-span framework, suggesting that their origins are simply manifestations of social influences that begin to operate possibly even at conception, but that they are, however, impacted by an individual's biobehavioral characteristics as well.

In the present study spousal perceptions of hyperactive symptomatology (e.g., inattention, distractibility, and physical overactivity) could not directly substantiate nor discredit either a behavior genetic or developmental systems theory claim. In fact, parents of hyperactive and non-hyperactive children were indistinguishable from one another based on hyperactive symptomatology. I offer several suggestions for this null finding. One suggestion is that the measure of hyperactive symptoms used was not adequate to address this issue. However, a better and more viable assumption might be that hyperactivity is strictly a childhood disorder, and that adults who were once hyperactive are currently expressing their dysregulatory behavior in other, more "adult like" ways. For instance, Loeber (1990) suggests that childhood hyperactivity is precursorily related to antisociality and substance abuse, and that it may actually evolve into these pathological

states during adolescence and adulthood in certain individuals. If this idea is correct then we may be seeing hyperactives who are grown up. Their propensity towards dysregulation is obviously seen in the "acting out" behaviors associated with their alcoholism and antisociality but it is not expressly seen as traditional hyperactivity.

In accordance with this, if the behavior genetic assumption concerning hyperactivity is correct, then hyperactive boys are being reared by present or, more often than not, former hyperactives. This notion would accurately describe results from the present study, especially if we consider them in light of the notion of a developmentally based dysregulatory behavior continuum (see below). For instance, parents of the hyperactive boys in this study, although not apparently hyperactive themselves, were significantly more antisocial and prone to alcoholism than the parents of non-hyperactive boys. Therefore, according to Loeber's theory of an antisocial continuum, and the currently proposed theory of a dysregulatory continuum (see below), these "former" hyperactive individuals are now channeling their dysregulated behavior through externalizing behaviors such as alcoholism, antisociality, etc. Gittelman et al. (1985) found evidence to substantiate this claim. Specifically, they found that the most common adulthood outcome seen in their group of hyperactive boys was the profound incidence of antisocial behavior and substance abuse. Similarly, Weiss et al. (1979) have also reported excessive amounts of antisociality and criminality in adults who were hyperactive as children.

Somewhat juxtaposed to this line of thinking, others have suggested that there is also a subgroup of hyperactive children may actually "outgrow" their hyperactive symptoms and progress on a more positive developmental pathway (August & Stewart,

1983). However, even if hyperactive children do outgrow their hyperactivity (many do and many do not) many still experience detrimental outcomes such as cognitive deficiencies and socioeconomic underachievment (Frick et al., 1991; Gittelman et al., 1985). For instance, several studies have shown a significant association of hyperactive symptomatology and cognitive deficiency (see Hinshaw, 1992). Not only are hyperactive children many times cognitively impaired, but their parents also are many times cognitively impaired. In the present study hyperactive children were significantly more cognitively impaired than non-hyperactives, and likewise, parents of hyperactive boys were more cognitively impaired than parents of non-hyperactive boys. For example, parents of hyperactive children revealed lower cognitive abilities (IQ scores), and lower levels of education than parents of non-hyperactives. Again, this evidence supports a systems theory approach - the combinatorial effects of genetic load and the impoverished rearing environment are all conducive to "triggering" an individuals propensity towards dysregulatory behavior in conjunction with lower levels of cognitive functioning.

One developmental note is worth making here. As noted previously, some studies find age related trends regarding cognitive deficiency with hyperactivity and conduct problems (see Hinshaw, 1992 for review). First, aggressive conduct problems are, for the most part, un-correlated with cognitive deficiency when hyperactive symptoms are *absent*. For instance, high levels of hyperactivity (and not conduct problems) are highly associated with cognitive impairment (IQ) in toddlers and children, but low IQ is primarily related to aggressive conduct problems during adolescence. However, when one examines the correlations between hyperactive symptomatology, conduct problems, and IQ regardless of age, it is very apparent that hyperactivity is more strongly and more consistently related

to cognitive impairment than aggressive conduct problems. In the present study cognitive ability was correlated 3 times higher with hyperactive symptoms than with conduct problems. We may, however, begin to see an increase in the strength of association between cognitive impairments and conduct problems when these boys move into the adolescent period.

Along these same lines, a high association has been noted between externalizing problem behaviors and language proficiency (see Hinshaw, 1992 for review). Language ability is a strong predictor of academic outcome and future socioeconomic achievement and is highly associated with intellectual ability (IQ) and perceptual motor development. Therefore, if delayed language ability is etiologically relevant to hyperactivity, it should be apparent in early childhood in hyperactive children. Findings of cognitive deficiency and language impairments have been suggested to implicate neurologic involvement (i.e., neurodevelopmental delay) in hyperactive children (Martin et al., in press; Tarter et al., 1990). In that similar cognitive and lingual impairments were noted in the parents of hyperactives, and according to the neurobehavioral hypothesis discussed here, what we may be seeing are the effects of a neurologically mediated, behavioral dysregulatory mechanism, one whose individual expression is heavily tied to the behaviorally unmonitored rearing environment. Results from the present study revealed lower language development in hyperactive, cognitively impaired children.

Another suspected etiologically relevant factor of hyperactivity is a sub-optimal rearing environment. Severe sociodemographic deprivation has been noted to exist at high rates within the hyperactive milieu, and this has led behavioral scientists to conclude that the rearing environment plays an integral role in the etiology and progression of

dysregulatory behavior. Rutter and his colleagues (Schachar et al., 1981; Taylor et al., 1986b), for instance, have noted severely low levels of SES, income, and parental education in families of hyperactive children, concluding that a deprived environment places a child at an increased risk for other behavioral problems. Ham et al. (1994) found sociodemographic indicators to be significant predictors of hyperactivity regardless of parental level of externalizing psychopathology. The authors concluded that the rearing environment itself has a severe impact on child self-regulation regardless of parental externalizing psychopathologic involvement, but that there is also a combined or cumulative effect when both are present. These findings regarding the rearing environment don't necessarily discount a genetic claim to hyperactivity, rather they buttress a behavioral genetic theory of hyperactivity. According to Goldsmith (1989), there is a passive gene-environment interaction that accounts for the high heritability of hyperactivity. When children with a high genetic load for hyperactivity are placed in an environment that is conducive to their propensity for hyperactivity (e.g., hyperactive parents and siblings, unmonitored or unrestrained behavior is permitted), then heritability will be relatively higher, compared to if they are placed in an environment that attempts to restrict or regulate the expression of their genetic endowment.

The Antisocial Continuum: A Component of the Behavioral Regulatory Continuum

The present study found significant overlap or of HADI and conduct problems. Theorizing about the overlapping qualities of these disorders has led some to conclude that they are parts of one biobehavioral dysregulatory disorder (Ham et al., 1993). From a more focused perspective, Loeber, Lahey, and Thomas (1991) have speculated about the relatedness of only the severe conduct problem behavior disorders (i.e., ODD and CD).

They have suggested that the overlapping qualities of ODD and CD compose a *continuum* of antisocial behavior.

Such notions are certainly viable ones in light of the vast amount of research and clinical observations made concerning the relationship of hyperactivity and conduct disorders (Lilienfeld & Waldman, 1991). It seems reasonable to suggest then that hyperactivity and conduct problems may in fact be part of one behavioral disorder, and likewise, it is quite feasible that these disorders may also be segments of an antisocial behavior continuum. This has led me to suggest that these disorders may also be only one segment or portion of an even larger continuum, a continuum that constitutes *overall behavioral regulation* (see Figure 28); these dysregulated behaviors compose a portion of the poorer (lo regulation) side of the continuum. Furthermore, it is also likely the case that hyperactivity is not only one of the principal expressions of the dysregulatory portion of this continuum, but that it is the earliest expression of dysregulatory behavior. According to this view, the emergence of hyperactivity gives us our first glimpse of the dysregulatory phenomenon. Such a concept is consistent with infant research findings on temperament (Stavish, 1994) (see below).

In light of this suggested biobehavioral regulatory continuum, there are several empirical qualifications for discussing it within a developmental conceptual framework. The argument exists along these lines: First of all, we know that hyperactivity is precursorily related to antisocial behavior (Hinshaw, 1992), and that it is an integral participant in the development of antisocial disorders such as ODD, CD, and possibly even APS (APA, 1994). Thus we see the developmental relatedness of these dysregulatory behaviors. In fact, Loeber (1990) describes hyperactivity as the "catalyst" necessary in

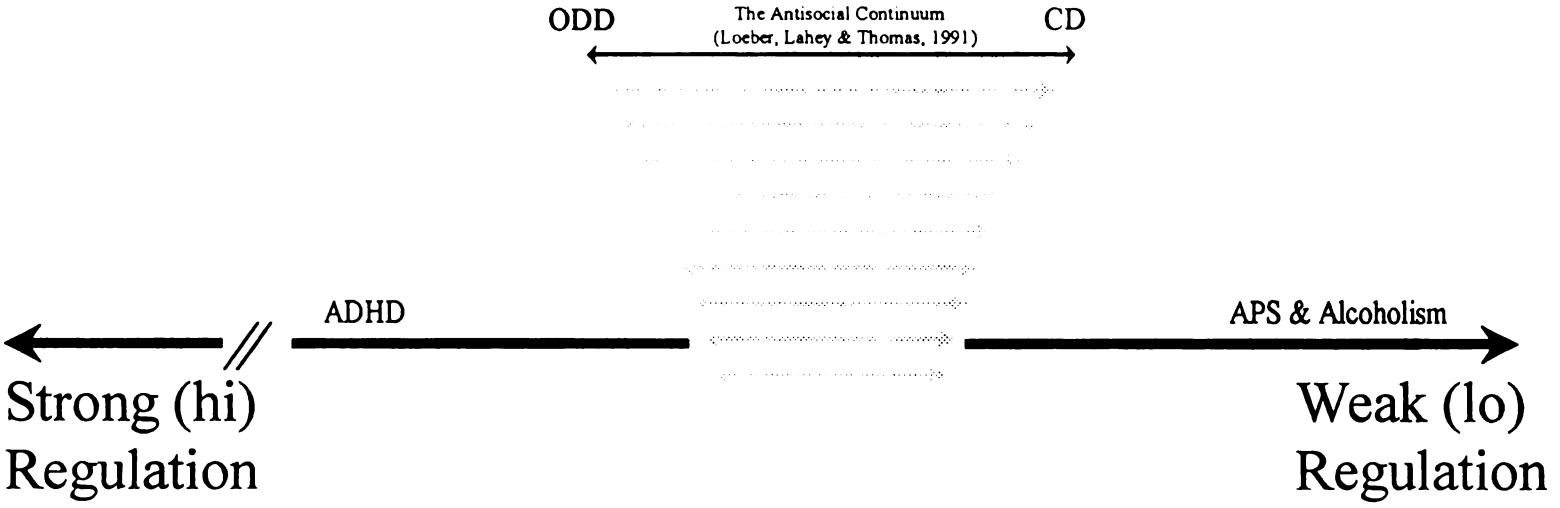


Figure 28. The Dysfunctional Portion of the Behavioral Regulatory Continuum: The Antisocial Continuum as an Embedded Feature

maintaining serious conduct problems like ODD and CD. Second, in that hyperactivity is developmentally precedent to ODD and CD, and in that it is less distressing for society (i.e., less societally offensive and violatory) than the other two disorders, it would logically be located at the higher functioning end of the dysregulatory portion of the continuum (this position on the continuum would signify its developmental precedence to the other two disorders). Thus, the dysregulatory portion of the behavioral regulatory continuum would have hyperactivity at the more functional pole of the dysregulatory portion of the continuum, Loeber et al's. antisocial continuum (composed of ODD and CD) in the middle, and adult outcomes such as APS and alcoholism at its furthest developmental and most severe behavioral qualitative point. All of these disordered behavioral expressions, therefore, would compose the developmentally maintained *dysregulatory* segment of the Behavioral Regulatory Continuum (Figure 29).

Current research findings on hyperactive and conduct disordered children reveals that certain individuals are on a more hazardous developmental pathway than others, and in some cases this pathway leads to severe adulthood outcomes (e.g., APS and alcoholism). In order to more fully describe this idea of a regulatory continuum, the "low" end, or the most dysregulated portion, exemplifies the present (hyperactivity and conduct problems) and future (APS and Alcoholism) hazardous developmental pathway that a group of at-risk children are believed to be on. It is only this subgroup of children who will follow this developmental progression, and it seems that those comorbid with symptoms of hyperactivity and conduct disordered behavior are most likely the ones to do so.

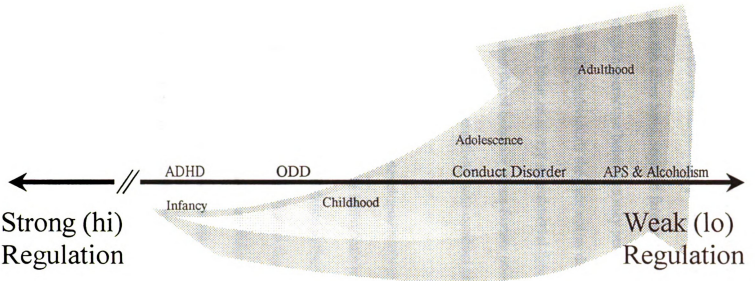


Figure 29. The Dysfunctional End of the Behavioral Regulatory Continuum: A Developmental Perspective

It is believed that some individuals have inherited some sort of *mechanism* or *propensity* (see below) which predisposes them towards dysregulated or unmonitored behavior. Furthermore, it is also hypothesized that it is these individuals who are most likely comorbid with hyperactivity and conduct problems in childhood. Children who are hyperactive only, on the other hand, seem to fare better developmentally (August & Stewart, 1983). Although hyperactive behaviors may persist into adolescence and adulthood in some hyperactive individuals they are not as debilitating as they are in the comorbid state, nor do they incur as severe a detrimental future outcome. The suggestion here is not that conduct disordered behavior alone fosters poor behavioral outcome but rather, it is the co-occurrence of hyperactivity with conduct problems that promotes the most detrimental outcome.

Therefore, a likely scenario for this developmentally mediated dysregulatory phenomenon might look something like this: In individuals who have a propensity for dysregulation, one might see it surface during infancy in the form of hyperactivity or "difficult temperament" (Thomas & Chess, 1984; Tarter et al., 1985); as development unfolds the innate propensity would (if the rearing environment is conducive to such) continue to gain severity of expression during adolescence revealing itself primarily as ODD and/or CD, but possibly maintaining the hyperactive symptomatology as well (August & Stewart, 1983); ultimately, adulthood behaviors indicative of Antisocial Personality disorder (APS) and possibly substance abuse (Loeber, 1990) would emerge.

In summary, it is very important to remember that although this notion of a regulatory continuum finds its expression developmentally, and thus suggests continuity, it is most likely the case that the propensity for dysregulated behavior is inherently present in

an individual at birth. And this being the case, dysregulatory behaviors can only express themselves in accordance with developmental capacity. In other words, as certain developmental proficiencies are attained the more dramatic behaviors (e.g., CD and APS) are proficiently available and begin to surface (see Figure 29). For instance, an infant or toddler is well able, and indeed usually diagnosable, to exhibit behavior connotative of ADHD but not CD. Why? One line of thinking suggests that an infant or toddler is developmentally unable or developmentally unprepared to exhibit behaviors necessary for conduct disordered behavior - behaviors such as deliberate fire setting, stealing, etc... If the propensity for dysregulation is in fact in place at birth, then the individual has the necessary "soft-wiring" (and I use the term reluctantly for fear of suggesting a mechanistic phenomenon) for the more severe problem behaviors, but not necessarily the physical and/or psychological developmental capabilities (see Greenough, 1991 for a discussion along these lines).

Temperament Characteristics Relative to Behavioral Regulation Theory

The above logic is consistent with temperament theories suggested by Thomas and Chess (1984), Buss and Plomin (1984), and Rothbart and Posner (1985). Similarly, others (see Stavish, 1994) have found convincing evidence which suggests that temperamental traits consistent with the concept of *self-regulation* are inherited. If children are born with similar temperamental characteristics as their parents, as suggested to be the case in an alcoholic family (Tarter et al., 1990), then this notion of an inherited dysregulatory propensity may be on the right track. However, one's inherited propensity for dysregulated behavior may or may not have its roots in any specific pathology as suggested by Tarter et al. but rather, it may be an unselective incident, one that finds its

expression in the unfolding developmental environment. Therefore, whatever the specific environment a child finds himself in, whether it be an alcoholic one, an abusive one, or whatever, he would exhibit behavior consistent with that specific environment. Couple that with his inherent inclination for dysregulation and you have a very behaviorally volatile situation. This is also reflective of the gene-environmental interaction that Goldsmith (1989) talks about regarding difficult temperament; the propensity is present at birth, the environment, however, serves as the ultimate guiding factor in determining an individual's final behavioral outcome.

The characteristics of hyperactivity have been referred to as integral parts of one's temperamental makeup (Thomas & Chess, 1984). For instance, attention, which also contains the notion of distractibility, reflects one's ability to monitor events in the outside world, all the while deciphering relevant and non-relevant information. Physical activity level, likewise, reflects an individual's ability to monitor his/her motoric output; the highly regulated person may be very physically active, but his/her motoric levels are situationally maintained. Similarly, one's ability to delay impulse is another qualitative characteristic of temperament. All of these characteristics, while reflecting temperamental qualities, may be indicative of pathology if they are expressed in an abnormally high manner. A person displaying this unmonitored temperament profile would be located on the "low" or "dysregulated" portion of the Behavioral Regulatory Continuum discussed above, while the person who is able to sustain attention, monitor physical activities so that they are situationally specific, and able to control impulse, would be located towards the "hi" or "regulated" portion of the continuum (see Figure 28).

Relevant to the present study, researchers have noted a link between high levels of

behavioral disorders (Rutter et al., 1988; West & Prinz, 1987; Zucker et al., 1994) or "difficult" temperament styles and substance abuse (Lerner & Vicary, 1984; Windle, 1991). Children with a difficult temperament profile typically come from very chaotic homes, homes where parents themselves are temperamentally difficult and behaviorally dysregulated (Fitzgerald et al., 1990). For example, the Jansen et al. (under review) study found that children being reared by substance abusing parents exhibiting high levels of dysregulated behavior are themselves temperamentally difficult, exhibiting similar dysregulated behavioral patterns as their parents (e.g., aggression). In the present study, the high correlation between parental and offspring regulatory behaviors in alcoholic families is very suggestive of the proposed inherited dysregulatory mechanism, at least within the alcoholic milieu. And the fact that there was a coincident cumulative nature seen for difficult temperament characteristics in the alcoholic environment strongly implicates alcoholism as a relevant etiologic factor in biobehavioral dysregulation.

Findings from the Ellis (1993) study shed further light on this suggestion. In her study, Ellis found a very high and positive relationship between a child's risk for difficult temperament and externalizing behavioral problems. She suggests that, although the risk for difficult temperament is more evident in the children growing up in an antisocial alcoholic family, the difficult temperament "style" is probably indicative of negative behavioral outcomes in all children. She goes on to further suggest that the difficult temperament child is at heightened risk for poor behavioral outcome due to genotypic underpinnings concealed within families where there is a dense family history of alcoholism.

Tarter et al. (1985, 1990) also theorize along these same lines. They suggest that

because difficult temperament traits are so highly correlated with the alcoholic environment they may be risk factors for alcoholism. Using a neuropsychological paradigm Tarter and his colleagues have found that children at risk for alcoholism are more attentionally challenged, easily distracted, and manifest very rapid behavioral tempo. The authors suggest that such behaviors reflect, not only the difficult temperament profile, but also a dysfunctional behavioral executive center (i.e., anterior frontal lobes of the brain, see Luria, 1966). If in fact this difficult temperament style is reflective of frontal lobe dysfunction, it would strongly plead for a genetic component to behavioral regulation in conjunction with the risk for alcoholism (see below).

Findings from the present study, as well as those from other studies using the same dataset (e.g., Ellis, 1993; Fitzgerald et al., 1990; 1993; Ham et al., 1993, 1994; Jansen et al., under review), have found that children of alcoholics are perceived by their parents and clinical observers as attentionally deficient, easily distracted, physically overactive, impulsive, and developmentally delayed. Although children from this dataset may be reflecting some sort of brain dysfunction in these deficits, we are unable to ascertain whether or not such deficits are in fact neurologically relevant. We can only speculate that such behavioral and cognitive deficits may reflect neurological involvement due to their very nature, and only in connection with our knowledge of brain dysfunction in brain damaged and neurodevelopmentally delayed children (Geschwind & Galaburda, 1985; Goodman, 1991).

Possible Explanations of Alcoholic Offspring Behavior Dysregulation

There were several assumptions made concerning the relationship between parental levels of externalizing psychopathology (i.e., in the present study alcoholism and

antisociality) and offspring behavioral dysregulation. Most alcohol studies investigating offspring behavioral characteristics report significant levels of hyperactivity and conduct problems in sons of male alcoholics (SOMAs; Tarter et al., 1990). SOMAs are at significantly greater risk for developing alcoholism (Cloninger et al., 1988), and therefore they are effective targets for elucidating the etiologic determinants of alcoholism (Tarter et al., 1990). The high levels of biobehavioral dysregulation seen in SOMAs in the current study strongly suggest that these children are on a precarious developmental pathway leading to similar adult outcomes as their parents (e.g., alcoholism, antisociality). In accordance with this notion of biobehavioral dysregulation as described by Tarter et al. (1990) and Begleiter (Holden, 1991), it seems that biobehavioral dysregulation may be a heritable condition for SOMAs, or at least for a group of SOMAs whose parents are antisocial.

Biobehavioral Dysregulation

High levels of excessive physical activity, difficulties sustaining attention, ease of distractibility, and impulsive behavior in children in the present study are here suggested that they may have their origins in a CNS dysfunctional condition, and may be manifestations of what some refer to as an inherited "biobehavioral dysregulatory mechanism" (Ham et al., 1994; Holden, 1991). Regarding the link between hyperactive symptomatology and alcoholism, Henri Begleiter (personal communication, May, 1992) suggests that behavior patterns typical of ADHD may not be grounded solely in the genetics of alcoholism per se, or any other parental externalizing psychopathology for that matter, but may be emergent from an inherited, "pathologically non-specific" behavioral dysregulatory mechanism. Begleiter's theory suggests that the underlying, neurally based

behavioral dysregulatory mechanism is a result of interplay between set genetic components and the environment. He suggests it is a "...set of biologic factors which are heavily influenced by environmental events and can lead to very different adverse outcomes" (Holden, 1991) depending on the rearing environment. To buttress this theory he refers to several electrophysiological experiments conducted by himself and others which reveal similar types of dysregulatory behavior in SOMAs and their fathers. For example, similar brain wave anomalies found in alcoholic men have been noted in their pre-consuming sons (Begleiter et al., 1984; Gabrielli et al., 1982). However, Begleiter is quick to point out that these anomalous brain wave patterns may not be specific to, or indicative of, the later development of alcohol abuse per se, as similar findings are consistent for cocaine abusers. The present hypothesis, therefore, suggests that hyperactive behaviors may not be precursorily related to alcoholism, or any specific pathology for that matter but rather, are suggestive of a more general behavioral dysregulation whose expression, I believe, is dependent upon developmentally consequential environmental events. The importance of environmental interplay with this hypothesized mechanism is buttressed in the present study by the significant high levels of economic deprivation and abusive behavior apparent in families and parents of SOMAs.

Tarter et al. (1990) make the observation that 50% of the variance accounting for severity of alcoholic expression can be predicted by childhood hyperactivity and social maladjustment. Moreover, they point out that the presence of hyperactive symptoms is significantly associated with neuropsychological performance, and that the severity of hyperactivity runs parallel to neuropsychological functioning. Therefore, the authors strongly suggests that the "disturbances in behavioral regulation may have a direct

neurological basis" (pp. 84).

As discussed above, this theory of neurologically mediated behavioral dysregulation was of primary importance to the underlying premise of the present study. The general hypothesis was that SOMAs may inherit a pathologically non-specific (albeit possibly alcohol related) neurobehavioral dysregulatory mechanism, and that this mechanism gains expression in the child's behavioral patterns depending upon his experiences in the rearing environment (see Gottlieb, 1991). For instance, if SOMAs are being reared by parents who themselves display hyperactive behaviors, this may facilitate similar behaviors in them, both as a result of their inherited disposition to dysregulated behavior and as a result of the influence of the rearing environment itself (e.g., imitating the hyperactive environment).

However, with the use of SEM techniques I was unable to effectively demonstrate this hypothesized neurobehavioral dysregulatory phenomenon. None of the parental predictors were causally related to my measures of neurobehavioral functioning. In fact, the variable that contained the neurobehavioral component did not seem to "fit" in the models altogether. I do not believe that the theory is unsubstantiated by this, however. What I think is happening has to do with the maturity level of the developing brain and the ability to detect the subtle neurobehavioral deficits that exist within SOMAs. More specifically, during the preschool years the brain is still attaining its final level of maturity; myelinization is still occurring at rapid rates (Dekaban, 1970; Huttenlocher, 1990, cited in Ham, 1993b), and thus inter- and intra-hemispheric connectivity is still in progress (Barr & Kiernan, 1988). This being the case, we would not be able to detect neurobehavioral differences in children at this age, therefore we must wait for neural development to slow

down (e.g., during adolescence) before we would be able to detect such anomalies. Although this suggestion is speculative in nature and is certainly un-testable with the present data set, it does reflect the current zeitgeist concerning neuronal growth in the maturing brain during late infancy and early childhood, and may, therefore, be a viable reason for these null findings.

Dysfunctional Areas of the Anterior Cerebral Cortex: A Possible Explanation.

Another explanation that ties into the biobehavioral mechanism hypothesis is the possibility of a brain dysfunctional condition. Tarter and his group at the University of Pittsburgh have been suggesting a dysfunctional mechanism related to the anterior cortical area of the brain as a possible explanation for the high relationship seen between risk for alcoholism and the incidence of hyperactivity. Anterior regions of the brain, particularly the prefrontal cortex (e.g., Supplementary Motor Areas), are known to subserve the executive functions of decision making, formulation of goals, attentional capabilities, and self-regulatory functioning (Lezak, 1983). This area of the brain has been described as the "executive regulatory center" for behaviors characteristic of ADHD (i.e., attention, motoric regulation, etc.) (Luria, 1966). Alcoholics and their children in the present study displayed inabilities in sustaining attention, excessive hyperactivity, poor intellectual skills, and impulsivity, all of which are reminiscent of individuals who have experienced mild anterior cerebral trauma and/or dysfunction (see Tarter et al., 1985, 1990).

Pre-alcoholics and children at risk for alcoholism have been shown to exhibit disrupted language mechanisms (Tarter et al., 1985), specifically impairments in language capacity (Hegedus, Alterman & Tarter, 1984). As discussed above, children of alcoholics in the present study were significantly developmentally delayed on linguistic capabilities

compared to normal children. Also, when the target boys were re-grouped based on meeting hyperactive cutoffs, hyperactive boys were significantly more impaired on language functioning compared to non-hyperactives. Luria (1966) also notes that an interruption of, or damage to language sites in the frontal cortex has serious repercussions on language regulation. This is a possible explanation for the deficient language development in SOMAs in the present study.

Neuropsychological abnormalities found in alcoholics and their hyperactive offspring may further substantiate the anterior cortical hypothesis. For instance, deficits have been found for visual task persistence (Alterman et al., 1984), delay gratification (Ellis, 1993), abstract problem solving (Schaeffer, Parsons & Yohman, 1984), and attention and memory (Tarter et al., 1984), all of which implicate the anterior cerebral cortex. Schaeffer et al. (1984) suggest that it is the familial component of alcoholism that enhances one's chances for neurological complications not simply alcohol consumption, and furthermore, it may be the alcoholic with a family history of alcoholism who has *premorbid* neurodeficits involving cognitive and motoric functions. These findings argue for a neurobehavioral explanation for the vulnerability to alcoholism and its relationship with ADHD (Hegedus et al., 1984), and one that specifically implicates frontal brain areas.

Tarter et al. (1985, 1989, 1990) suggest that high levels of physical overactivity may also reflect CNS involvement. Physical hyperactivity is usually the most apparent behavioral component of hyperactivity, and therefore, it may specifically reveal neurologic involvement in some individuals with an inherent propensity for dysregulated motor behavior. Unregulated motoric behavior would implicate deficits in the pre-frontal cortex in that this portion of the brain mediates self-regulation of motor behavior. August and

Stewart (1982) suggest that since physical overactivity is commonly seen in isolation among brain damaged individuals, this facet of hyperactivity, when appearing as the primary symptomatology, may be particularly indicative of brain involvement. Results of a recent study by Tarter et al., (1989) strongly suggest that physical overactivity reflects a genetic predisposition towards alcohol abuse, and therefore one that is particularly relevant to the etiology of alcoholism. This finding can not confirm or disconfirm a genetic predisposition for alcoholism, but it is suggestive of anterior cerebral involvement of self-regulatory function in children at risk for alcoholism, and therefore it is certainly biologically relevant. As previously stated, physical overactivity occurred at substantially higher rates in the children being reared in high risk alcoholic families.

Antisociality, Alcoholism, and Economic Deprivation: How they Relate to Biobehavioral Dysregulation

Results of the present study revealed a much higher incidence of biobehavioral (i.e., neurobehavioral) dysregulation in SOMAs than SOMnAs. (Neurobehavioral regulation was defined as attentional ability, behavioral tempo, ability to delay impulse, and cognitive and lingual functioning). As expected, SOMAs were responsible for the highest (i.e., abnormal) incidence of biobehavioral dysregulation. However, one of the more interesting findings was uncovered when the offspring of alcoholics were examined in light of paternal levels of antisocial behavior. Separation of antisocial alcoholic offspring from non-antisocial alcoholic offspring shed some interesting light on offspring regulatory functioning. Consistent with Zucker et al's. (1993) findings, it is apparent from present study findings that alcoholism *is* responsible for an increase in offspring dysregulatory behavior, however, alcoholism *comorbid* with parental antisociality is

involved in the most dramatic cases. This finding has led researchers on the MSU-UMLS to make several hypotheses concerning the comorbidity of alcoholism and antisociality with regards to offspring outcome; these hypotheses agree with those suggested by others.

For example, Earls et al. (1988) were the first to suggest that serious adult outcomes are more prominent in the antisocial alcoholic than the non-antisocial alcoholic. Results from the present study confirm such a notion. For instance, antisocial alcoholics were more cognitively impaired, they had lower levels of education, and they were more sociodemographically challenged than non-antisocial alcoholics. More importantly, however, is the finding that the offspring of antisocial alcoholics are also more dysfunctional than the offspring of alcoholics, and therefore they are at more serious risk. Children of antisocial alcoholics in this study were more likely to be severely behaviorally dysregulated (over 5 times) than sons of non-antisocial alcoholics and normal control children. Not only was there a dramatic increase in hyperactivity and conduct problems in children of antisocial alcoholics, but these children also had the highest incidence of overlap of hyperactivity and conduct problems. Parallel to Earls et al's. notion of the cumulative effects of adult alcoholism and antisociality on poor adult outcomes, behavioral effects of antisocial alcoholic offspring are likewise revealing significantly more severe childhood outcomes during the pre-school years than offspring of alcoholics only.

Therefore, it is not a leap of faith to say that the cumulative outcomes of childhood behavior disorders (i.e., comorbidity) are the result of the cumulative effects of parental externalizing psychopathology. Similar to Zucker et al's. (1993) findings, it appears that antisociality, in conjunction with alcoholism, is the driving force behind behavioral dysregulation in SOMAs. Sons of **antisocial** alcoholics exhibited significantly more

dysregulated behavior than sons of non-antisocial alcoholics and controls; they showed greater incidence of hyperactivity, conduct problems, and neurobehavioral impairments than the other two groups. In fact, for the most part, sons of non-antisocial alcoholics and controls in this study looked very similar.

These findings suggest two possible reasons for the increase in dysregulatory behaviors among offspring of antisocial alcoholics. First of all, it is possible that antisociality is more etiologically relevant (i.e., genetically) to behavioral disorders such as ADHD, ODD, and CD than alcoholism. As stated above, ADHD, ODD, and CD may be biobehavioral precursors to antisociality (Loeber, 1990). If this is so, then children of antisocial alcoholics may inherit a propensity towards, not only alcoholism, but antisocial behavior as well. This line of thinking has been suggested elsewhere. For instance, the DSM-III-R (APA, 1987) reports that antisociality is five times more common in first-degree male relatives and there is usually comorbidity with a substance abuse disorder. Nigg and Goldsmith (1994) report on the vastness of literature which supports a heritability theory for antisociality, noting that none of the conclusions from these studies have been overturned.

Cloninger and his associates (Bohman et al., 1987; Cloninger et al., 1985) have also reported high heritability rates for antisociality. In the present case, if antisociality is so highly heritable a trait, and if ADHD, ODD, and CD are precursors to antisociality, then what we may be seeing is the emergence of the *antisocial individual* as he progresses along the dysregulated portion of the behavioral regulatory continuum (see Figure 29). Furthermore, children who are comorbid with ADHD and conduct problems may be 1) genetically tied into this pathway more strongly than those who are not comorbid and 2)

the rearing environment may play a vital role in behavioral regulation and expression. If it is not the case that antisociality is more etiologically relevant to behavioral dysregulation than alcoholism, it may be that the coupling of antisociality with alcoholism, as suggested by Earls et al. (1988), is the etiologically relevant situation that is. The comorbidity of alcoholism and antisociality may not only predict severe adulthood outcomes, but may also set their offspring on a hazardous developmental pathway leading to similar outcomes.

Regardless of which situation is the cause of poor offspring outcome, it is very apparent that both alcoholism and antisociality are somehow involved in encouraging behavioral dysregulation in SOMAs. However, the sociodemographically deprived rearing environment in which the antisocial alcoholics in this study are rearing their children in may also be very responsible for some of these detrimental outcomes. Not only was parental and child psychopathology greatest among the antisocial alcoholic families, but sociodemographic indicators were also lowest in these families. Therefore, one must bear in mind that the possible heritability of biobehavioral dysregulation and the deprived rearing environment both are playing a role in determining offspring outcome. Table 9 summarizes the logic behind the possible outcomes of the gene-environment interaction that I have been discussing.

Structural equation modelling was fairly unsuccessful in demonstrating the effectiveness of parental externalizing psychopathology and socioeconomic achievement in the models tested. However, parental externalizing psychopathology was more *consistently* predictive of behavioral regulation than socioeconomic achievement. A tentative conclusion, therefore, would be that parental externalizing psychopathology is

Table 9. Possible Genetic/Environmental Combinations and Outcomes

Biological Load	Environmental Load	Outcome
hi heritability	negative environment	high risk
hi heritability	positive environment	buffered high risk
low heritability	negative environment	buffered high risk
low heritability	positive environment	low risk

more influential on dysregulatory behaviors than the sociodemographic situation of the alcoholic family. However, a behavioral genetic hypothesis would suggest that it is *both* the genetic propensity for dysregulatory behavior and the dysregulated rearing environment that are dually responsible for poor childhood outcomes. In other words, levels of psychopathology may be so high and may create so much chaos and inconsistency in the home environment, that the genetic propensity for dysregulation and the extremely dysregulated environment are significantly contributing to the severe outcomes that these boys are currently experiencing, as well as those that they will most likely experience in adolescence and adulthood.

In summary, behavioral dysregulatory behaviors such as hyperactivity may have neurological implications. Short attention span, high levels of distractibility and impulsivity, and physical overactivity may be indicative of a neurologically mediated behavioral disorder. Furthermore, these behaviors many times appear in conjunction with conduct disordered behaviors consistent with ODD and CD. They also are seen in conjunction with cognitive deficits and language impairment. The literatures on both alcoholism and alcohol related behavioral problems strongly implicates a milieu of alcoholism and sociodemographic impoverishment as etiologically relevant to behavioral

dysregulation.

CHAPTER VI

Limitations of the Study

There are several limitations to the current study that must be addressed, several of the most outstanding pertain specifically to the kinds of data used. For instance, data used to categorize children into the various behavioral problem categories were obtained solely using parentally-perceived questionnaire data. When using such data alone, one runs the risk of introducing and capitalizing on method variance which may account for the effects found. For instance, it may well be that more troubled parents (AALs) perceive their sons also as being more troubled, thus the higher ratings of disordered behavior. It may be more desirable to have corroborative observed data measures such as clinician ratings of hyperactivity and conduct problem behaviors.

Not only were parental reports used for behavioral classification, but maternal questionnaires were solely used in order to group boys into hyperactive and non-hyperactive groups. However, this is a common practice in the child literature (see Campbell et al., 1986). In fact, most researchers agree that maternal reports of child behavior are more reliable than paternal ratings (see Achenbach, 1978; Cowan & Cowan, 1988).

Another limitation related to this first issue pertains to the neurobehavioral measures used. None of the measures used (with the exception of the Stanford-Binet) are clinically referred neuropsychological constructs. Standardized neuropsychological tests/battery would be more desirable for use in order to ensure that the theorized neuro-functions believed to be assessed in the current study were actually being tapped.

Also, related to the above issue is the fact that only self- and parent-reported data

points figured significantly into the LISREL models. This may be an example of capitalizing on method variance, however, there is also a developmentally based neurological explanation. For instance, it is widely accepted (see Geschwind & Galaburda, 1985) that brain development is extremely plastic and fluid until late childhood and early adolescence, therefore, it may be that subtle neuropsychological deficits are not as accessible during the preschool years. Neuroimaging study findings have documented differences in cerebral metabolic activity in hyperactive children compared to normals (e.g., Hynde et al., 1990; Lou, Henriksen, & Bruhn, 1984), however, findings are small and not outstanding compared to adult findings of similar phenomenon (e.g., Zametkin et al., 1990).

Second, there is a major concern in the present study concerning the validity of constructs used to assess hyperactivity and conduct problem behaviors. Although content (based on DSM-III-R and DSM-IV criteria) and concurrent validity was ensured (i.e., correlations with other similar measured), it would be most beneficial to establish external validity (i.e., parallelism, discriminant) to each of the measures of child problem behaviors.

Third, in that we are primarily interested in the **development** of alcoholism and alcohol related difficulties arising from an alcoholic heritage, a longitudinal design would be most conducive to elucidating etiologically relevant family characteristics of hyperactive children that ultimately lead to such outcomes. The cross-sectional design of the present study is a good starting point for tapping into such etiologies, but examining the variables over time is the most effective means of establishing causality.

Finally, control subjects used for comparison were originally believed to exist within similar census tracts as alcoholics and their families, thus comparability was readily

permitted concerning sociodemographic disparity. However, Pallas (1992) reports that the prevalence of alcoholic families was inherently tied to population density, in other words, she found the highest correlation for alcoholism with that of urbanicity. She also found high correlations among alcoholic families and percent living below poverty levels. This amounts to the fact that alcoholism is inversely related to family income and socioeconomic achievement. Her findings also indicate a positive relationship of rates of divorce, unemployment, and recipients of public assistance, among other variables, with level of alcoholism. These results are interpreted as indicating that controls and alcoholics are located in very different census tracts and thus comparability is not as easily explained as originally suggested.

CHAPTER VII

Summary

Present study findings are congruent with much of the behavioral research in the hyperactivity and alcoholism literatures. Children of alcoholics, and particularly preschool aged SOMAs, are at an increased risk for biobehavioral dysregulation. Study conclusions are interpreted in light of behavioral genetic, developmental systems theories, and neuropsychological research. Results suggest that the biobehavioral dysregulation experienced by SOMAs in the present sample may be a result of a passive gene-environment interaction. Not only does the genotype of SOMAs reflect the propensity for dysregulatory behavior, but the interaction of their genotype with the dysregulated rearing environment (which is conducive to a child expressing his inherent propensity towards dysregulatory functioning) is responsible for severely dysregulated offspring functioning. Study findings also reveal that it is not simply the alcoholic parent nor the alcoholic environment that is solely responsible for the most severe child outcomes, but rather, it is the comorbidity of alcoholism and *antisociality* that produces the most detrimental outcomes in alcoholic offspring. It is possible that this comorbid pathological situation may drive the gene-environment interaction responsible for biobehavioral dysregulation in the subgroup of SOMAs with antisocial parents.

APPENDIX

APPENDIX

Post-hoc LISREL Analyses

Parental Models

Due to concerns about the stress seen in the models due to the Neurobehavioral elements, post-hoc analyses were run to address this situation. Results are briefly presented and discussed.

Two levels of post-hoc analyses were run with regards to this element of the models. First, by removing the two troubling elements of the Neurobehavioral variable (Delay and IQ) it could be seen if the other measures (gross and fine motor, and language functioning) were adequate measures for this variable. As stated above, these three elements correlated a little too highly with one another than would be desirable. However, these three elements were left in the variable and then the models were re-run on each group.

The long and short of these analyses was that very little change took place in the final outcomes of the models using this strategy. For instance, when the post-hoc model was tested on the control group, goodness-of-fit indices all improved, but only marginally, and all causal pathways remained essentially unchanged. Results were similar for the group of non-antisocial alcoholics, although in some of the runs the fits actually decreased with removal of the two elements. Likewise, for the group of antisocial alcoholics fits increased slightly, but all causal pathways remained virtually unchanged.

In that none of the models significantly changed when the two troubling elements in the Neurobehavioral variable were removed it was decided to probe a little further, and

to take the post-hoc analyses one level deeper. Models 1 and 2 were re-run for all of the groups, but this time the Neurobehavioral variable was omitted. This essentially resulted in a model of Externalizing behavioral outcomes. What this amounted to was essentially an analogous study to the previous one (Ham et al., 1994), except here structural equation modelling techniques were used instead of simple regressions, and the sample size was doubled.

When the two models were run again without the Neurobehavioral variable, results for control families remained unchanged. Fits were identical and paths remained unchanged. For the non-antisocial alcoholic group several changes were noted when the models were re-run without this variable. When Model 1 was run the goodness-of-fit indices remained essentially the same, however, the pathways from Externalizing Psychopathology and Activity Level became significant predictors of offspring behavioral regulation (see results for sex differences for this group). No changes were noticed for the second model on this group. Next, the refined model on the antisocial alcoholics was run. Results were similar to controls, basically no changes in fits or pathways. However, on Model 2, the pathway from Socioeconomic Achievement was a significant predictor of HADI (Gamma coefficient = .20, t = 1.93).

Models by Sex

Controls

After concluding the post-hoc's for the models with parental ratings combined, the maternal model was refined and re-run for Model 1. As stated above, the removal of the Neurobehavioral element didn't change the final outcomes significantly. When the Delay and IQ elements were removed from the Neurobehavioral variable the goodness-of-fit

indices were all adequate but low; the Externalizing Psychopathology and Activity Level pathways that were nearly significant in the full model for these women now became significant. By removing these two troubling elements this decreased the "noise" sufficiently to bring out the relationship of these two variables. When the Neurobehavioral variable was completely removed the fits increased significantly and the pathways for Externalizing Psychopathology and Activity Level became even more robust. So for this group of individuals the Neurobehavioral element was causing significant distress to the model, its refinement and ultimate removal were significant assets to the model.

Similar results were found for fathers in this group. In the initial, full model fits were pretty good and the Activity Level variable was nearly significant, however, the model was refined GFI's increased quite a lot and this causal pathway became significant. Also, the "noise" in this model was quieted down significantly. Results remained identical for both post-hoc tests (i.e., removal of the two elements first, then the complete removal of the Neurobehavioral variable).

non-Antisocial Alcoholics and Their Wives

Effects of post-hoc analyses for both mothers and fathers in this group were similar to one another and fairly unremarkable. Goodness-of-fit indices and significant causal paths remained unchanged from the initial model runs regardless of removal of the two items or the entire variable. For mothers the Externalizing Psychopathology and Family Social Achievement variables were consistently significant throughout the testing procedures. Likewise for fathers the Activity Level variable remained the consistent predictor of offspring behavioral regulation.

Antisocial Alcoholics and Their Wives

Model 1 was poorly fitting for fathers and didn't fit at all for mothers during the initial testing phase. Post-hoc analyses neither enhanced the models' adequacy nor degraded it.

ENDNOTES

ENDNOTES

[1]It has been argued that the use of labels, particularly in the case of simple physical hyperactivity, many times may be artifactually applied to children. The use of the hyperactive label in this case is applied to individuals "whose behavior is annoying to adults because it does not comply with the demands" of those being affected by the child's level of physical overactivity, namely, parents and teachers (Schachar, Rutter, & Smith, 1981). In light of this, it must be remembered that the majority of childhood hyperactivity ratings are obtained from parents (mostly mothers) and teachers. This being the case, there may be biasing circumstances for both parties that may reveal either intolerance to, or in some cases tolerance to, the behavior. Parents and/or teachers may even show a lack of interest (e.g., as may be the situation with depressed mothers) in the child's excessive physical activity and thus under-reporting of the problem behavior may result.

To illustrate, consider an example pertinent to subjects in the present study. SOMAs are reportedly reared in homes marked by high levels of chaotic and fast paced behavior due to high parental pathologic involvement (Fitzgerald et al., 1989; Tarter, Alterman & Edwards, 1985). Thus, the child who displays high levels of hyperactivity in this situation may go unnoticed by his/her parents simply because they (the parents) are used to this kind of behavior, not only from their children but most likely from themselves also. In other words, this situation might reveal a *tolerance* to the child's behavior. Another way of describing this would be in temperament language - "goodness of fit" (Thomas & Chess, 1984). This then would lead to an inappropriate parental non-diagnosis/classification of ADHD. However, another situation might be one in which parents have a low threshold for high activity levels (an intolerance to the behavior **and/or**

a low level of arousal). In this case they may very well label their otherwise normally active child as hyperactive due to their *intolerant* nature. However, in such a case the label would not be warranted.

[2]A parenthetical illustration of such can be found in studies pertaining to drug response in hyperactive and conduct problemed individuals. Taylor et al. (1986) report that children diagnosed with ADHD respond better to drug therapy than children diagnosed with CD. These results, and related findings in the literature, point to the possibility that ADHD is grounded in the genotype, i.e., contains a neurophysiological element (see Heilman, Voellar, & Nadeau, 1991), more so than CD. Therefore, ADHD may be a distinct disorder from CD with different etiologies and consequently different treatment strategies, as well as different psychological and behavioral outcomes.

[3]It should be noted that the neurologic deficits suggested by Tarter et al. may also be the result of higher incident of early head trauma for high and low risk groups (69% vs. 12%, $p < .001$ respectively), and/or higher reports of physical abuse in the high risk group compared to the low risk group (31% vs. 4%, $p < .02$ respectively). West & Prinz (1987) reviewed the alcoholism literature concerning physical abuse of children in alcoholic families, and they report the incidence of child abuse to be alarmingly higher in alcoholic homes. Rates of child abuse in alcoholic families was 14.6% in a national study of 32,000 alcohol abusing families. In a sample of 1,013 Finish adolescents, of whom 29% had alcoholic parents, 33% of COAs were reported to have been physically abused. Other studies reviewed by West and Prinz report that up to 75% of alcoholics in treatment have a high potential for abusing their children.

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