THE EFFECTS OF CHILDHOOD MALTREATMENT ON CRIMINAL AND SUBSTANCE ABUSE OUTCOMES IN URBAN YOUTH DIAGNOSED WITH ADHD

by

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Abstract

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Results from longitudinal studies of individuals diagnosed with attention-deficit/hyperactive disorder (ADHD) in childhood have clearly shown that these children are at heightened risk for poor outcomes as they enter into adolescence and early adulthood. Among poor outcomes criminality (Barkley, Fischer, Edelbrock, & Smallish, 1990; Hechtman & Weiss, 1986; Mannuzza, Klein, Konig, & Giampino, 1989; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993) and substance use disorders (SUDs; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998; Wilens, Biederman, & Mick, 1998; King, Iacono, & Mcgue, 2004) are particularly prevalent and cause significant hardship for the individual, their family, and society at large.

While early conduct disorder (CD) has been shown to account for a substantial portion of the risk associated with later criminality and substance use in youth with ADHD (Armstrong & Costello, 2002; Brook, Whiteman, Cohen, Shapiro, & Balka, 1995; Disney, Elkins, Mcgue, & Iacono, 1999; Barkley, Fischer, Smallish, & Fletcher, 2004), the extent to which other factors, such as a history of childhood maltreatment, contribute to poor outcomes remain relatively unexplored. This is surprising given the fact that 1) there is a clear literature showing that childhood maltreatment confers considerable risk for later poor outcome in general population

studies (Cicchetti & Manly, 2001; Widom, 1989a; Smith & Thornberry, 1995; Zingraff, Leiter, Myers, & Johnsen, 1993; Ireland, Smith, & Thornberry, 2002; Smith, Ireland, & Thornberry, 2005), and 2) children diagnosed with ADHD are at increased risk for maltreatment due to externalizing behaviors and dysfunctional parental relations (Briscoe-Smith & Hinshaw, 2006; Ford, Racusin, Daviss, Ellis,Thomas, Rogers, et al., 1999). To our knowledge, no study has examined the contributory role of childhood maltreatment on later poor outcome among children with ADHD. The identification of maltreatment as risk factor is important as it could 1) significantly change the way clinicians assess and treat children with ADHD, and 2) be instrumental in the development of more targeted treatment alternatives and interventions for this at risk population.

The following series of studies investigated the role of childhood maltreatment in the development of later criminality and SUDs in adolescents and young adults in a referred sample of urban, ethnically and socio-economically diverse children recruited in childhood between the ages of seven and 11 years and diagnosed with ADHD (N = 169). This group was re-assessed in adolescence (n = 98), almost ten years later, and compared to a well-matched, never-ADHD comparison group (n = 85). Official criminal records for the sample were obtained approximately three years after commencement of the adolescent follow-up.

The results of these studies clearly establish a history of childhood maltreatment as a potent risk factor for later antisocial and substance use outcome in ADHD youth. In addition, it appears from our results that at least some portion of the poor outcome that has been attributed to CD in ADHD studies may in fact be due to childhood maltreatment. These findings have important implications with regard to antisocial and substance use outcomes and emphasize the utility of assessing childhood maltreatment in ADHD populations.

In loving memory of my father

Thomas J. Fresiello Jr.

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GENERAL INTRODUCTION

ADHD and Childhood Maltreatment

Childhood maltreatment and ADHD are two major health problems that affect the lives of millions of children and their families each year (American Psychiatric Association, 1994; U.S.Department of Health and Human Services, 2006) with rates of ADHD in school-aged children ranging from 5-10% (Scahill & Schwab-Stone, 2000), and rates of child maltreatment in the general population ranging between 25-40% (Moran, Vuchinich, & Hall, 2004; Scher, Forde, McQuaid, & Stein, 2004). Further, several studies have reported elevated rates of maltreatment in ADHD samples (Glod & Teicher, 1996; Putnam, 1993; Beecker-Blease & Freyd, 2009). For example, in studies of individuals with a history of maltreatment, rates of ADHD have been found to range from 23-46% (Famularo, Fenton, Kinscherff, & Augustyn, 1996; Glod & Teicher, 1996; McLeer, Callaghan, Henry, & Wallen, 1994; De Bellis, Hall, Boring, Frustaci, & Moritz, 2001), which is quite high when compared to the aforementioned rates of ADHD in the general population. In addition, children diagnosed with ADHD are known to be at increased risk for maltreatment due to externalizing behaviors and dysfunctional parental relations (Appleyard, Egeland, Van Dulmen, & Sroufe, 2005; Briscoe-Smith & Hinshaw, 2006; Ford et al., 1999). Lastly, because of familial risk for ADHD, parents of children with ADHD are more likely to have the disorder, and parents with ADHD may have problems with impulsivity that could lead to a greater abuse potential.

There is considerable overlap in behavioral (De Bellis et al., 1999a; Hildyard & Wolfe, 2002; De Bellis, 2001; Gutman & Nemeroff, 2003) and cognitive symptoms (Beers & De Bellis, 2002; De Bellis et al., 1999a) between children with ADHD and children with a history of

maltreatment, and studies have revealed that they share some neuroanatomical substrates as well as disruptions to similar neurotransmitter systems. Of particular importance in disentangling the complexities of the relationship between ADHD and childhood maltreatment is the implication of misdiagnosis or missed diagnosis which may result in a specific treatment approach that could significantly affect outcome.

Additionally, both ADHD and childhood maltreatment predispose children to elevated rates of criminality and SUDs as they enter into adolescence and young adulthood. Considering the risk and prevalence of childhood maltreatment in ADHD populations, research contrasting subgroups of ADHD with and without childhood maltreatment histories is essential as this may change our approach to ADHD assessment and treatment. To further understand this relationship, following is a review the general characteristics and etiology of both ADHD and childhood maltreatment.

Attention-deficit/hyperactivity Disorder: General Characteristics and Etiologic Factors

Attention-deficit/hyperactivity disorder is a developmental disorder characterized by difficulties in attention, impulsivity and hyperactivity. Children with ADHD often experience significant functional difficulties such as low self esteem (Ek, Westerlund, Holmberg, & Fernell, 2008), cognitive deficits (Hinshaw, 2002), poor school performance (Hinshaw, 2002; Mannuzza, Klein, Bessler, Malloy, & Hynes, 1997), and trouble with peer and parental relationships (Melnick & Hinshaw, 1996; Hoza et al., 2005). Risk factors for the disorder include: male sex, genetics (Faraone et al., 2005; Khan & Faraone, 2006), maternal smoking and alcohol use during pregnancy [Linnet et al., 2003; Mick, Biederman, Faraone, Sayer, & Kleinman, 2002; Nomura,

Marks, and Halperin (in press)], low SES (Rieppi et al., 2002), and low birth weight (Mick, Biederman, Prince, Fischer, & Faraone, 2002).

Research from family, twin, and adoption studies strongly supports a genetic hypothesis for ADHD (Kendler et al., 2000; Faraone & Doyle, 2001; Faraone, 2004). Family studies have shown that ADHD is highly familial (Faraone & Doyle, 2001), twin studies have consistently shown high levels of concordance in monozygotics versus dizygotic twins (Goodman & Stevenson, 1989; Sherman, Iacono, & McGue, 1997), and adoption studies have reported higher rates of ADHD in biological relatives than adoptive relatives (Alberts-Corush, Firestone, & Goodman, 1986). One review of twin studies concluded the percentage of the variance accounted for by genes in ADHD is approximately 76% (Faraone et al., 2005).

Comorbidity with other psychiatric disorders is also common in children with ADHD, with approximately 50 – 60 % meeting criteria for oppositional defiant disorder (ODD), 30-50% meeting criteria for conduct disorder (CD), 25% meeting criteria for anxiety disorders, and 15-25% meeting criteria for comorbid mood disorders (Biederman, Newcorn, & Sprich, 1991; Jensen, Martin, & Cantwell, 1997).

Neurobiology of ADHD. Several models of ADHD implicate dysfunction in the prefrontal cortex (PFC) and its connections with the striatal complex (Barkley, 1997; Sonuga-Barke, 2002), while other models have implicated abnormalities in brainstem mechanisms regulating arousal, activation and alerting (Sergeant, 2005; Halperin & Schulz, 2006). Although progress has been made elucidating the neural substrates of ADHD, with most groups focusing on executive or inhibitory control deficits mediated by DA-sensitive fronto-striatal circuits (Bush, Valera, & Seidman, 2005; Castellanos, 2001; Seidman, Valera, & Makris, 2005; Durston, 2003), there is now mounting evidence implicating dysfunction in regions outside the

frontostriatal circuitry including cerebellar (Seidman et al., 2005; Castellanos et al., 2002; Berquin et al., 1998; Anderson, Polcari, Lowen, Renshaw, & Teicher, 2002), temporal (Castellanos et al., 2002), and parietal (Shaw et al., 2007) structures.

Disruptions in hypothalamic-pituitary-adrenal (HPA) axis functioning have also been implicated in the pathophysiology of ADHD (Hastings, Fortier, Utendale, Simard, & Robaey, 2009; King, Barkley, & Barrett, 1998). Under conditions of stress, cortisol binds to glucocorticoid receptors (GR) in the PFC and helps facilitate adaptive arousal and orienting. As such, HPA axis dysfunction could affect normal orienting and arousal, contributing to attentional and behavior difficulties seen in ADHD. Research in this area has been equivocal with some groups finding decreased cortisol levels and blunted HPA axis responsiveness to stress in youth with ADHD (Randazzo, Dockray, & Susman, 2008; Kaneko, Hoshino, Hashimoto, Okano, & Kumashiro, 1993) and others finding the opposite (White & Mulligan, 2005; Sondeijker et al., 2007). Kaneko (1993) examined HPA axis function in 30 children with ADHD by measuring diurnal variations in salivary cortisol as well as cortisol levels in response to challenge (Kaneko et al., 1993). They reported that 43% of the children with ADHD had normal diurnal variations in cortisol levels, and 46% showed suppression in response to challenge. A subsequent analysis which split the group into severely hyperactive and mildly hyperactive showed that abnormal diurnal levels and blunted response to challenge were more frequent in the severely hyperactive group. These finding suggest that there may be differences in HPA axis function among the subtypes of ADHD, or possibly between those with certain comorbid conditions. Hastings (2009) addressed these issues by examining HPA axis functioning across subtype (hyperactive/impulsive or inattentive) and comorbid conditions (anxiety disorders and ODD/CD) in a sample of youth with ADHD (Hastings et al., 2009). Those with comorbid anxiety disorders

showed an exaggerated response to challenge, whereas those with ODD/CD showed a blunted response. No differences were found between subtypes of ADHD.

Neuroanatomical findings. A review of structural neuroimaging studies of children with ADHD by Seidman (2005) reported the majority of groups showing a 3-5% decrease in total cerebral volume, particularly in the right hemisphere (Seidman et al., 2005). Abnormalities in the corpus callosum have been reported by several groups, although some report findings in the posterior region (splenium) (Hill et al., 2003; Hynd et al., 1991), and others in anterior regions (rostrum) and middle portion (rostral body) (Giedd et al., 1994). Structural abnormalities and volume reduction have consistently been found in the cerebellum of individuals with ADHD (Castellanos et al., 1996; Hill et al., 2003; Mostofsky, Reiss, Lockhart, & Denckla, 1998; Berquin et al., 1998). This is not surprising considering that striatal circuitry is modulated by the cerebellum. In fact, in a meta-analysis of 21 structural studies of ADHD youth, Valera (2007) found that the largest areas of difference in ADHD versus controls were seen in cerebellum (posterior inferior vermis) followed by the splenium, total cerebral volume, and the right caudate (Valera, Faraone, Murray, & Seidman, 2007). Two studies in the meta-analysis measured prefrontal regions (Mostofsky, Cooper, Kates, Denckla, & Kaufmann, 2002; Kates et al., 2002), and both showed a large significant difference in volume between ADHD and controls. Further, Casey (1997) found smaller right prefrontal regions in boys with ADHD which was correlated with performance on a response inhibition task. In a study of adults with ADHD, Seidman and colleagues (2006) reported significant reduction in the volume of the anterior cingulate gyrus and the dorsolateral PFC in adults with ADHD compared to controls (Seidman et al., 2006).

Castellanos and colleagues (2002) assessed volumetric changes longitudinally between 152 youth with ADHD (medicated and medication naïve) and 139 controls (Castellanos et al.,

2002). Rather than showing a selective fronto-striatal effect, by adolescence this group found decreased volume in all four lobes, with differences only reaching statistical significance in the cerebellum (even after controlling for comorbidity and medication exposure). Additionally, cerebellar volume was significantly and negatively correlated with ratings of attentional problems. Overall, findings from neuroanatomical studies in children and adolescents with ADHD have been inconsistent, perhaps due to the heterogeneity of the disorder or the frequency of comorbid pathology. Functional imaging studies generally support findings from the anatomical studies identifying dysfunction in neural circuitry in the frontal lobes (Bush et al., 1999), striatum (Rubia et al., 1999) and cerebellum (Valera, Faraone, Biederman, Poldrack, & Seidman, 2005).

Childhood Maltreatment: General Characteristics and Etiologic factors

Child maltreatment refers to all types of physical or emotional abuse or neglect, or sexual abuse of an individual under the age of eighteen that results in actual or potential harm to that individual's survival, health, development of dignity. There are several factors that contribute to childhood maltreatment. To begin with, parents are the perpetrators in 80% of the cases of childhood maltreatment (U.S.Department of Health and Human Services, 2006). Many of these parents are survivors of child abuse themselves, suffer from mental health problems, and have failed to develop appropriate coping mechanisms and parenting skills (Widom, 1989b). Conditions in the home environment such as poverty, isolation, lack of support network, or marital discord can also increase the likelihood of abuse (Mersky, Berger, Reynolds, & Gromoske, 2009). Some characteristics of the child can also contribute to his/her own

vulnerability. For example, children that are mentally retarded, or have a behavior disorder, such as ADHD, are at heightened risk for maltreatment (Appleyard et al., 2005; Ford et al., 1999).

A positive history of maltreatment has been associated with high rates of psychopathology with studies reporting elevated rates of mood and anxiety disorders (Nemeroff, 2004; Heim et al., 2008) and disruptive behavior disorders including ADHD (Gutman & Nemeroff, 2003; De Bellis et al., 2001). In addition, maltreated children exhibit functional deficits such as social problems, aggressive behavior, and difficulties in school (De Bellis, 2001; Chapman et al., 2004; Hillis, Anda, Felitti, Nordenberg, & Marchbanks, 2000). Poor outcomes in adolescence and adulthood are also associated with a childhood history of maltreatment. For example, in the Isle of Wight study (Collishaw et al., 2007) researchers found that adolescents with a history of maltreatment were 15 times more likely to be depressed and eight times more likely to have an anxiety disorder than their non-maltreated peers. Followed into adulthood, these individuals were eight times as likely to have a SUD. In a large population sample, Kendler et al. (2000) found an increased risk of depression, anxiety, alcohol dependence and divorce in adults who reported childhood abuse, even after controlling for parental psychopathology (Kendler et al., 2000). Although not all children that are victims of childhood maltreatment go on to develop PTSD, a substantial portion do. For example, in a study by Widom (1999) of individuals with substantiated child abuse and neglect histories, 37.5% went on to develop PTSD. Much of the research on the effects of childhood maltreatment has focused on groups of abused children who have gone on to develop PTSD. As the majority of abused children do not go on to develop the disorder, the results of these studies must be interpreted with caution as maltreated youth who meet diagnosis for PTSD might represent a more severe form of childhood maltreatment or have more genetic vulnerability to overall poor outcome.

Neurobiology of early trauma. The developing central nervous system is remarkably plastic. Genes dictate its basic framework and foundation, but the final form is shaped by personal experience. There are windows of vulnerability and resilience in early brain development that mediate the effects of early adversity (Bremner, 2003; Teicher, Andersen, Polcari, Anderson, & Navalta, 2002), and several brain areas in humans are particularly vulnerable to early stress due to protracted postnatal development [e.g. hippocampus, cerebellar vermis (Gould & Tanapat, 1999), limbic structures (Sowell, Trauner, Gamst, & Jernigan, 2002), and PFC (Casey, Giedd, & Thomas, 2000; Sowell, Thompson, & Toga, 2004; Casey, Tottenham, Liston, & Durston, 2005)]. The HPA axis plays a central role in the regulation of the stress response, and preclinical studies have shown that stress in early childhood leads to long-term changes in the HPA axis, as well as long-term changes in the noradrenergic system.

Preclinical investigations of the effects of early trauma. The mother–infant bond is the strongest, most enduring social attachment formed throughout life in most mammals (Bowlby, 1982). It is not surprising that disruptions to this bond produce profound behavioral and physiologic responses that reflect its potential as a psychological stressor. Several groups have demonstrated the serious and long lasting changes that result from such disruptions. Classic early work by Harlow (1965; 1966) comparing monkeys raised by their biological mothers to monkeys reared by surrogate mothers showed that the surrogate raised monkeys became socially deviant and highly aggressive as adults (Harlow & Harlow, 1965; Harlow & Harlow, 1966).

Numerous studies in non-human primates have demonstrated the effects of maternal separation (MS) on brain anatomy and function. Sánchez et al. (1998) compared infant male monkeys raised individually in a nursery from two to 12 months to age-matched controls raised in a semi-naturalistic social environment (Sanchez, Hearn, Do, Rilling, & Herndon, 1998) and

found that, although overall brain volumes did not differ between groups, the corpus callosum (specifically the mid sagittal region) was significantly decreased in the nursery group. When cognitive functioning was examined, the degree of cognitive impairment was correlated with differences in corpus callosum size. Rilling et al. (2001) examined the neural response to MS in Rhesus monkeys (Rilling et al., 2001) and found that MS was associated with increased right dorsolateral PFC and right ventral temporal/occipital lobe activation, decreased left dorsolateral PFC activation, and that activation was correlated with plasma cortisol levels. Further, offspring exposed to maternal aggression showed increased activity in the anterior cerebellar vermis and the ventral striatum.

Interesting results have also been demonstrated in non-primate animal models. Maternal separation has been used successfully to model many of the physiological and behavioral effects of early-life trauma with studies showing that variations in maternal care in the first week of life effect cognitive, emotional, and neuroendocrine responses to stress in adulthood (Caldji, Diorio, & Meaney, 2000; Heim & Nemeroff, 2001; Ladd, Owens, & Nemeroff, 1996; Liu et al., 1997; Meaney et al., 1991). In a series of seminal studies, Meaney and his group at McGill University reported on the various lasting changes in the molecular organization of the stress response system in MS rats (Caldji et al., 1998; Caldji et al., 2000; Caldji, Diorio, & Meaney, 2003; Liu, Caldji, Sharma, Plotsky, & Meaney, 2000). Corticosterone and adreno-corticotropin-hormone (ACHT) measures were compared between MS rats and non-separated rats. Baseline ACTH and corticosterone plasma levels did not differ between the groups, but the MS group showed exaggerated ACTH and corticosterone levels in response to stress. Maternal separation also resulted in attenuated development of central benzodiazepine and high-affinity gamma-aminobutyric acid (GABA)–A receptors in the amygdala and locus coeruleus (LC).

In another set of studies, Meaney's group compared dyads separated by rates of licking, grooming, and arch back nursing (LG-ABN) (Caldji et al., 1998; Fish et al., 2004). Offspring of high LG-ABN mothers showed increased hippocampal glucocorticoid receptor (GR) messenger RNA expression and enhanced glucocorticoid feedback leading to decreased hypothalamic CRH messenger RNA expression and modest HPA axis response to stress (reduced plasma ACTH and corticosteroids). Offspring of low LG-ABN mothers showed decreased GR, increased hypothalamic CRH messenger RNA expression, and diminished alpha-2 noradrenergic receptor density in the LC leading to an exaggerated stress response. In addition, an increased fearfulness in response to threat was found in the offspring of low LG-ABN mothers which was associated with decreased neurogenesis and synaptic density in the hippocampus (Caldji, Francis, Sharma, Plotsky, & Meaney, 2000). In a subsequent study, this group showed that treatment with selective serotonin reuptake inhibitors (SSRIs) greatly attenuated the negative sequelae associated with early trauma in MS rats (Huot, Thrivikraman, Meaney, & Plotsky, 2001) providing a sound pre-clinical model for treatment of individuals negatively affected by childhood maltreatment.

Early stress also appears to affect other aspects of the central nervous system. In the mesolimbic DA system, MS results in increased DA release as well as a decrease in the number of D2-DA receptors in the ventral tegmental area (VTA) and lowered DA transporter levels in the nucleus accumbens (Meaney, Brake, & Gratton, 2002). Behavioral measures of both reward and reinforcement involving DA are also affected (Matthews, Robbins, Everitt, & Caine, 1999). Further, MS offspring exhibit increased intake of sucrose solution and self-administration of cocaine when compared to controls (Michaels & Holtzman, 2006; Matthews et al., 1999).

As a whole, results of preclinical studies have shown the profound and long lasting effects of early trauma on neuroendocrine systems, neurodevelopment, and behavior. These studies have been important in furthering our understanding of the impact of childhood maltreatment on human development, and have revealed several brain regions that appear most vulnerable to early life stress.

Clinical studies of the effects of childhood maltreatment. In a study examining the impact of childhood maltreatment on behavior and biological stress systems, De Bellis et al. (1999a) measured urinary free cortisol, norepinephrine (NE), and DA levels among 18 pre-pubertal children with maltreatment-related PTSD, 10 non-maltreated children with generalized anxiety disorder (GAD), and 24 healthy controls (De Bellis et al., 1999a). Subjects with maltreatment-related PTSD excreted significantly higher concentrations of urinary NE and DA than the other groups. Further, children with maltreatment-related PTSD had significantly greater cortisol concentrations than controls. In addition, on clinical ratings, subjects with maltreatment-related PTSD showed significantly lower ratings on a scale of global functioning, higher ratings of depression on a depression checklist, more suicidal ideation and attempts, more problems with externalizing symptoms on the CBCL (specifically; social problems, aggressive and delinquent behaviors), increased levels of psychiatric comorbidity and poorer psychosocial outcomes than the other two groups.

Bremner et al. (2003) assessed cortisol responsivity to a stressful cognitive challenge in individuals with PTSD stemming from childhood maltreatment (Bremner et al., 2003) via salivary cortisol levels measured at baseline and after challenge in 23 patients with maltreatment-related PTSD and 18 controls. Maltreated individuals showed an increased cortisol response in anticipation of a cognitive challenge relative to controls and were unable to reduce responses to

cues that were not a true danger. Similarly, Heim et al. (2000) found heightened ACTH responsivity to psychosocial stress in a study of adult women with a history of childhood abuse (Heim et al., 2000).

Based on prior results showing that maltreated children with a diagnosis of PTSD had alterations in biological stress systems (De Bellis et al., 1999a; Bremner et al., 1997), De Bellis et al. (1999b) postulated that increased levels of catecholaminergic neurotransmitters and steroid hormones caused by maltreatment experiences in childhood could alter brain development (De Bellis et al., 1999b). To explore this hypothesis they conducted a study comparing 44 children/adolescents with maltreatment-related PTSD and 61 matched controls. Subjects underwent comprehensive psychiatric and neuropsychological assessments and a magnetic resonance imaging (MRI) scan. Results of imaging showed that maltreated subjects had smaller overall cerebral volumes, smaller volumes in PFC, right and left amygdala, left and right temporal lobes, and corpus callosum, and larger left and right lateral ventricles compared to controls (after controlling for SES). A trend for an increase in left hippocampal grey matter in subjects over controls was noted. When ODD, ADHD, and mood disorders were controlled for, no significant differences were seen in cerebral volume, lateral ventricles, corpus callosum, or the hippocampus.

Teicher et al. (2004) replicated some of these finding, reporting reduced corpus callosum in a study of maltreated youth – some who went on to develop PTSD, and some who did not - indicating that this finding is not limited to maltreated individuals who develop PTSD (Teicher et al., 2004). In a subsequent study, De Bellis (2006) investigated the relationship between structural volumes of the cerebellar hemispheres, vermis, brainstem, and clinical measures in children and adolescents with maltreatment-related PTSD and controls (De Bellis &

Kuchibhatla, 2006), and showed smaller left, right, and total cerebellum in the maltreatment-related PTSD group compared to controls after controlling for cerebral volume, SES, and IQ.

Employing steady-state fMRI (T2 relaxometry), Anderson et al. (2002) found a correlation between the activity in the cerebellar vermis and the degree of emotional lability in individuals with a history of sexual abuse (Anderson, Teicher, Polcari, & Renshaw, 2002). The amount of blood flow in the vermis was markedly decreased in the individuals with a history of abuse, indicating functional impairment in cerebellar vermis activity.

Based on results showing decreased volume in genu of the corpus callosum of maltreated youth diagnosed with PTSD (De Bellis et al., 1999b) coupled with clinical symptoms of intrusive thoughts and poor concentration, De Bellis, Keshavan, Spencer, & Hall (2000) hypothesized that PTSD symptomatology in abused children may result from damage to the ACC. Magnetic resonance spectroscopy (MRS) was used to measure metabolism in the ACC of 11 children/adolescents with maltreatment-related PTSD and 11 matched controls (De Bellis, Keshavan, Spencer, & Hall, 2000). Metabolism in the ACC was significantly lower in subjects with maltreatment-related PTSD when compared to controls suggesting that anterior cingulate neuronal metabolism may be altered in maltreatment-related PTSD. Further, a PET study by Bremner et al. (1999) showed altered function in the ACC in adult women with a history of abuse (Bremner, 1999).

Studies of adults with PTSD have also reported cognitive problems, particularly in the areas of concentration, learning, and memory (McNally, 1997; Yehuda et al., 1995). In a pilot study, Beers and De Bellis (2002) explored the effects of early abuse on neuropsychological functioning in 14 children and adolescents with maltreatment-related PTSD and 15 socio-demographically matched non-maltreated controls (Beers & De Bellis, 2002). Subjects with

maltreatment-related PTSD performed significantly worse than controls on the Stroop color/word trial, made significantly more omission errors on a measure of sustained visual attention (Digit Vigilance Test), completed fewer categories on the Wisconsin Card Sorting Test, and made fewer responses in the animal naming trial of the Controlled Oral Word Association Test than controls. These findings are consistent with a prior imaging study showing abnormalities in the PFC in individuals with PTSD (De Bellis et al., 2000; Bremner, 1999).

Overlap Between ADHD and Childhood Maltreatment

It is not uncommon for children exposed to maltreatment to exhibit what appear to be symptoms of ADHD or other disruptive behavior disorders. Externalizing behaviors such as acting out, aggression (De Bellis et al., 1999a), impulsiveness (Hildyard & Wolfe, 2002; Beecker-Blease & Freyd, 2009), problems in school, and disruptive relationships (De Bellis, 2001; Gutman & Nemeroff, 2003) are common, while internalizing behaviors such as forgetfulness, distractibility, inattention (Beecker-Blease & Freyd, 2009), and disorganization are also often seen (Beers & De Bellis, 2002; De Bellis et al., 1999a). Glod and Teicher (1996) measured activity level in 19 children with a history of abuse and 15 controls (Glod & Teicher, 1996), and found that abused children were 10% more active than controls. Further, physically abused children have been found to be more impulsive and irritable when compared to non-abused children (Kaplan, Pelcovitz, & Labruna, 1999; Sappington, 2000). Armengol and Cavanaugh-Sawan (2003) investigated differences in attention regulation among children with histories of ADHD, PTSD, and a group of matched controls via a continuous performance task (CPT) and the Stroop Test (Armengol & Cavanaugh-Sawan, 2003). On the CPT, the ADHD and PTSD groups did not differ from each other in errors of commission, but both groups performed

significantly worse than controls indicating that both ADHD and PTSD groups have deficits in inhibitory control. There were no significant differences between groups on the Stroop Test. Overall, these studies highlight that fact that many symptoms commonly reported as hallmark features of ADHD are also present in many children who have been maltreated.

Several groups have begun reporting on the association between ADHD and childhood maltreatment. For example, using data from a large population survey of adopted children, Simmel, Brooks, Barth & Hinshaw (2001) examined risk factors for ADHD and ODD (Simmel, Brooks, Barth, & Hinshaw, 2001) and found that when childhood maltreatment preceded adoption it doubled the odds of an ADHD diagnosis. Putnam (1993) found an association between exposure to childhood abuse and high rates of ADHD symptoms in girls with dissociative episodes (Putnam, 1993). In a study examining the relationships among substantiated cases of early maltreatment, PTSD, ADHD, and depression in prepubertal children, Glod & Teicher (1996) reported that 38% of children with a history of abuse and PTSD met criteria for ADHD (Glod & Teicher, 1996). Hinshaw (2002) examined background characteristics in a large sample of preadolescent girls with and without ADHD (Hinshaw, 2002), and found that abused girls were significantly more likely than non-abused girls to be diagnosed with ADHD.

Further, Ford and colleagues (1999) examined the association of trauma history to ADHD and ODD in 165 children diagnosed with ADHD, ODD, ADHD+ODD, or adjustment disorder (who served as controls) (Ford et al., 1999). In a univariate analysis, ADHD and ODD either alone or together carried an elevated likelihood of being exposed to victimization trauma (assault, mugging, community violence, family violence, or sexual molestation) when compared to controls. No relationship was found for non-victimization trauma (accident, disaster or

illness). Further, family psychopathology (positively) and parent education (negatively) were independently correlated to victimization trauma. Individuals with ADHD+ODD had the highest rate of victimization. When family psychopathology was controlled for, ADHD alone no longer predicted trauma history. Subsequent analysis of the same data set (Ford et al., 2000) examining subcomponents of the victimization trauma variable (specifically childhood maltreatment in the form of physical or sexual abuse) showed that the likelihood of experiencing physical or sexual maltreatment was greatest in the ADHD+ODD group (73% / 31% respectively), followed in order by ODD alone (41% /18%), ADHD alone (26% / 11%), and controls (10% / 0%).

To date, very little work has been done focusing specifically on differences between youth diagnosed with ADHD with and without a history of maltreatment. One such study by Briscoe-Smith and Hinshaw (2006) detailed the ways in which girls with ADHD plus histories of abuse differ systematically from non-abused girls with ADHD (Briscoe-Smith & Hinshaw, 2006). Their findings indicated that girls with ADHD and abuse histories had higher rates of externalizing behaviors and peer rejection when compared to ADHD girls without abuse histories. No differences in internalizing problems or cognitive deficits were evident.

Overlap of criminal and substance abuse outcomes. Several major prospective studies have linked childhood maltreatment to later antisocial behaviors reporting elevated rates of juvenile and adult arrests and violent crime in those with a maltreatment history (Appleyard et al., 2005; Maxfield & Widom, 1996; Smith & Thornberry, 1995; Widom, 1989c; Zingraff et al., 1993). For example, Smith and colleagues (2005) found that children with substantiated child abuse histories were almost five times as likely to be arrested as juveniles, and 11 times more likely to be involved in violent crime when compared to non-abused controls (Smith et al., 2005). Longitudinal studies of ADHD youth have also shown that childhood ADHD is a

significant risk factor for antisocial behavior in adolescence and adulthood (Barkley et al., 1990; Hechtman & Weiss, 1986; Mannuzza et al., 1989; Mannuzza et al., 1993) although the literature remains unclear as to why youth with ADHD are at such heightened risk for this adverse outcome.

In addition, individuals with a history of childhood maltreatment are at heightened risk for developing SUDs in adolescence and adulthood (Liebschutz et al., 2002a; Moran et al., 2004; Wall & Kohl, 2007). Rates of past maltreatment in samples of individuals with drug and alcohol abuse disorders are reported as high as 77-84% - three times that reported in the general population (Cohen & Densen-Gerber, 1982; Triffleman, Marmar, Delucchi, & Ronfeldt, 1995). Prevalence rates of adolescent and adult SUDs among individuals diagnosed with ADHD are also high, yet research into potential risk factors for later substance abuse in children with ADHD has yielded mixed results with sometimes small effect sizes.

Although results from longitudinal studies of ADHD youth clearly indicate these children are at risk for later criminality and SUDs, investigations into potential risk factors have been equivocal with no major unifying risk factor emerging. It seems likely that several factors play a contributory role in outcome and, while studies clearly indicate a role for CD in the emergence of later criminality and SUDs, the extent to which other factors play a role has received considerably less study. Considering the aforementioned elevated rates of SUDs and criminality in youth with a history of maltreatment, coupled with the frequent overlap of childhood maltreatment and ADHD, it seems logical that a history of childhood maltreatment should be examined as a possible risk factor for adverse outcomes in youth with ADHD.

The following series of studies will characterize the criminal and substance use outcomes of a large sample of ethnically diverse, lower SES urban youth diagnosed with ADHD in

childhood and a community matched control group, examine the relationship between a history of childhood maltreatment and these two adverse outcomes, and explore the relative impact of maltreatment history in relation to other well established risk factors. Study I will characterize the criminal and substance use outcomes of the sample and investigate the relationship between ADHD, a history of childhood maltreatment and these two adverse outcomes. In this study we hypothesize that 1) individuals diagnosed with ADHD in childhood will have elevated rates of criminality and substance use when compared to typically developing controls, 2) the presence of a history of maltreatment will be related to criminal outcome and substance use in both groups, and 3) the magnitude of the effect of childhood maltreatment on these outcomes will be highest among those with a childhood diagnosis of ADHD.

Further, as CD has been identified as a potent risk factor for both criminal and substance abuse outcomes in numerous studies of ADHD youth, maltreatment and its relative importance as risk factor for these poor outcomes will be examined related this established risk factor (CD). Study II will focus specifically on the contributions of childhood maltreatment as a risk factor for later criminal outcomes; measuring its salience in relation to CD. Study III will focus on the contributions of childhood maltreatment on later SUDs outcomes in adolescence, again contrasting against the contributions of CD. In the latter two studies, we hypothesize that a history of childhood maltreatment will emerge as an independent risk factor for later criminality and SUDs over and above the contributions of CD and other identified risk factors.

Study I: Characterizing Antisocial Outcomes in Urban ADHD Youth

Attention-deficit/hyperactivity disorder (ADHD) is among the most frequently diagnosed childhood psychiatric disorders, with prevalence rates ranging from 5-10% (Scahill & Schwab-Stone, 2000). Longitudinal studies have consistently shown children with ADHD to be at heightened risk for adverse outcomes characterized by adolescent psychopathology (Barkley et al., 2004; Gittelman, Mannuzza, Shenker, & Bonagura, 1985; Hechtman, Weiss, Perlman, & Amsel, 1984; Satterfield & Schell, 1997), persistence of ADHD symptoms (Barkley et al., 1990; Hechtman & Weiss, 1986; Mannuzza et al., 1993; Mannuzza et al., 1989), substance use disorders (SUDs; Barkley et al., 2004; Biederman et al., 2006; Mannuzza & Klein, 2000), and antisocial behavior (Mannuzza et al., 1989; Barkley et al., 1990; Fischer, Barkley, Smallish, & Fletcher, 2002; Weiss, Hechtman, Milroy, & Perlman, 1985).

Numerous prospective studies of children with ADHD have identified elevated rates of later criminality in probands when compared to non-ADHD peers. For example, in a study of clinically referred boys diagnosed with ADHD, Satterfield and colleagues (1997) showed significantly higher rates of arrests and incarcerations in adolescence and early adulthood for boys with ADHD when compared to controls (Satterfield & Schell, 1997; Satterfield, Hoppe, & Schell, 1982). In a follow-up study of clinic-referred children, Hechtman et al. (1984) documented that young adults diagnosed as hyperactive in childhood reported greater police involvement when compared to matched controls (Hechtman, Weiss, & Perlman, 1984). Using official arrest records, Mannuzza et al. (1989) showed that adolescents (Mannuzza et al., 1989) and adults (Mannuzza, Klein, & Moulton, III, 2008) diagnosed with ADHD in childhood were arrested, convicted, and incarcerated significantly more often than controls. Further, they

reported evidence that criminal outcomes were at least in part mediated by the development of SUDs. Based on official State arrest records, Barkley et al. (2004) found that clinic-referred children with ADHD had significantly more arrests and committed more felonies in early adulthood when compared to controls (Barkley et al., 2004). Interestingly, when separating criminal activities into predatory-overt and drug-related antisocial conduct, the ADHD group only differed significantly from the control group on drug-related activities, with the ADHD group having higher rates. Lastly, Biederman et al. (2006) reported elevated rates of antisocial disorders in adolescents/young adults diagnosed with ADHD in childhood when compared to non-ADHD controls (Biederman et al., 2006; Biederman et al., 2008). Results from community studies are similar. Farrington et al. (1990) found that youngsters with hyperactivity-impulsivity-attention deficit (HIA) had significantly more adolescent and adult criminality than children without HIA (Farrington, 1990). In a birth cohort study, Moffitt and Silva (1988) reported that in early adolescence, 58% of children with ADHD vs. 13% of those without ADHD were delinquents (Moffitt & Silva, 1988).

The aforementioned studies have been extremely influential in shaping our current thinking with regard to ADHD and antisocial outcomes; however, each has limitations. For example, relatively few studies examined children rigorously diagnosed at baseline using current classification systems and diagnostic instruments. Several of these samples were first recruited prior to 1980 when operationally-defined criteria for the diagnosis of ADHD had not yet been firmly established (Fischer, Barkley, Edelbrock, & Smallish, 1990; Hechtman, Weiss, Perlman, Hopkins, & Wener, 1979; Mannuzza et al., 1998; Satterfield et al., 1982). In these early studies, childhood diagnoses were made primarily via parent and teacher rating scales. As such, these studies may have been overly inclusive in their subject selection relative to more contemporary

diagnostic criteria. In addition, only three of the studies used official criminal records (Barkley et al., 2004; Mannuzza et al., 1989; Satterfield et al., 1982). Further, the majority of subjects in these studies were homogeneous in terms of ethnicity (mostly Caucasian) and socioeconomic profiles (mostly middle class) (Mannuzza et al., 1991; Satterfield et al., 1982; Barkley et al., 1990; Biederman et al., 2006), which limits generalizability. Considering the fact that minorities are much more likely to be arrested and convicted, and subsequently represent the bulk of prison populations (Washburn et al., 2008), it follows that more diverse samples should be the target of research in order to develop appropriate intervention strategies.

Finally, and central to the present study, none of the above mentioned studies of ADHD have investigated childhood maltreatment as a risk factor for later antisocial outcomes despite the fact that several major population based prospective studies carried out in the United States have reported robust links between a history of childhood maltreatment and the likelihood of engaging in antisocial behavior in adolescence and adulthood (Widom, 1989b; Cicchetti & Manly, 2001; Lansford et al., 2002; Widom, 1989a; Smith & Thornberry, 1995; Zingraff et al., 1993; Ireland et al., 2002; Smith et al., 2005). Therefore, although a relationship between ADHD and antisocial outcomes has been documented consistently in the literature, its exact nature and mediating factors, particularly in an ethnically diverse, low SES urban sample, remains unclear.

The present study is unique in that it consists of a referred sample of children recruited and assessed in childhood between the ages of 7 - 11 years as part of a study of ADHD youth with and without aggression. As the sample was recruited in the 1990's, standardized rating instruments, structured interviews, and DSM criteria were utilized. This sample is urban, ethnically and socio-economically diverse. The sample was re-assessed after approximately 10

years and compared to a well-matched, never-ADHD comparison group. Substance use and a history of childhood maltreatment were assessed for both probands and controls during the adolescent follow-up. Criminal data for the sample was obtained from the New York State Division of Criminal Justice Services approximately three years after the commencement of the adolescent follow-up.

This study is to our knowledge the first to examine childhood maltreatment as a risk factor for later criminal and substance use outcomes in a sample of ADHD youth. We characterize the antisocial outcomes among four groups, ADHD and controls with and without a history of childhood maltreatment and examine the effects of childhood maltreatment as a predictor of later antisocial behavior among probands and controls. We hypothesized that 1) individuals diagnosed with ADHD in childhood would have elevated rates of criminality and substance use when compared to typically developing controls, 2) the presence of a history of maltreatment would be related to criminal outcome and substance use in both groups, and 3) the effect of childhood maltreatment on criminal outcomes and substance use would be most robust among those with a childhood diagnosis of ADHD.

Method

Participants

The original cohort consisted of 169 children (88% male) recruited for two separate NIMH-funded studies (Schulz et al., 2001; Halperin et al., 1994) of children with ADHD conducted between 1990-1997. All participants were 7-11 years-old; mean (SD) age at initial evaluation was 8.9 (1.3) years. The sample was 21.2% Caucasian, 26.5% African American, 40.4% Hispanic and 11.9% of mixed ancestry. The group was primarily of lower to low-middle

SES (mean (SD) = 31.6 (14.51); range = 11 – 66) (Nakao & Treas, 1994), with a large portion at the poverty level. All participants were English speaking. Individuals were referred for behavioral difficulties by schools and mental health providers. The sample was rated as having significant behavior problems by both parents and teachers, and all participants were diagnosed with ADHD according to Diagnostic and Statistical Manual for Mental Disorders (3rd ed., rev.:DSM-III-R or 4th ed.:DSM-IV) criteria for ADHD using the Diagnostic Interview Schedule for Children (DISC; Shaffer et al., 1996). A diagnosis of schizophrenia, pervasive developmental disorder, Tourette's syndrome, or Full Scale IQ below 70 was exclusionary. Eighty-two (48.5%) met criteria for ODD, 53 (31.4%) met criteria for CD, 55 (32.5%) met criteria for at least one anxiety disorder, and 22 (13.0%) had a mood disorder.

We examined possible differences between the samples recruited for the two child studies – specifically, whether there might be distinctions between those recruited using DSM-III-R and DSM-IV definitions of ADHD, and those tested using the Wechsler Intelligence Scale for Children-Revised (WISC-R; Wechsler D., 1974), and the Wechsler Intelligence Scale for Children, Third edition (WISC-III; Wechsler D., 1991). We reviewed the ADHD symptom data from all structured interviews. All subjects would have met criteria for one of the DSM-IV ADHD subtypes, and most had a sufficient number of symptoms to suggest they would have met criteria for the combined subtype. The cohorts differed significantly on FSIQ (94.9 vs. 89.8; $t(159) = 2.19$, $p = .03$), most likely because the WISC-R generates higher IQ scores than the WISC-III. The samples also differed in rates of CD (21.9% vs. 38.5%; $\chi^2 = 5.32$, $p = .02$), although the overall rate of disruptive disorders (i.e., ODD+CD) was comparable (79.9% vs. 82.3%). The groups did not differ significantly on other variables (see Table 1).

Table 1

Characteristics of the Childhood Sample: Comparison of Samples from Child Study I and Child Study II

	Total N = 169		Child Study I n = 73		Child Study II n = 96	
	Mean	SD	Mean	SD	Mean	SD
Age	8.9	1.3	9.0	1.3	8.9	1.3
FSIQ*	92.0	14.7	94.9	11.1	89.8	14.4
SES	31.7	13.9	32.4	14.7	30.1	13.6
Parent Ratings – CBCL T-scores						
Externalizing	68.7	10.8	67.3	10.4	67.3	10.4
Internalizing	64.6	11.1	62.7	11.6	65.8	10.8
Teacher Ratings – IOWA Conners Scores						
Inattention/Overactivity	11.0	3.1	11.3	2.8	10.9	3.4
Oppositional/Defiant	8.2	4.6	7.8	4.7	8.5	4.6
Comorbid Diagnosis	**%**	**#**	**%**	**#**	**%**	**#**
ODD	48.5	82	54.8	40	43.8	54
CD*	31.4	53	21.9	16	38.5	37
Anxiety disorder	32.5	55	32.9	24	32.3	31
Mood disorder	13.0	22	15.1	11	11.5	11

*$p < .05$

The original group was not recruited for a longitudinal study and was comprised of a highly diverse and mobile inner-city population who were difficult to locate. As such, we did not anticipate re-evaluation of the complete sample. Of the 169 childhood subjects located, 18 refused participation, five were incarcerated, and one individual was deceased. Of the remaining 145 subjects, 98 (67.6%) completed the follow-up evaluation. The group that was lost to follow-up did not differ significantly from those followed in age at child evaluation, parent or teacher ratings of behavior, or in their rates of ODD, CD, mood or anxiety disorders as assessed during childhood (all $p > .05$). The two groups did differ significantly on childhood FSIQ, with those followed having a significantly higher scores than those lost to follow-up (94.0 vs. 89.3; $t(159) = 2.0$, $p = .05$). Overall, those included in the follow-up appear to be representative of the original childhood sample (see Table 2).

Table 2

Comparison of Baseline Status between Subjects Completed and not Completed

	Completed n = 98		Not Completed n = 71	
	Mean	SD	Mean	SD
Age at initial assessment (in years)	9.1	1.3	8.9	1.3
FSIQ*	94.0	14.3	89.3	14.9
SES	29.6	13.3	31.6	14.5
Parent Ratings – CBCL T-scores	Mean	SD	Mean	SD
Externalizing	69.7	11.2	67.2	10.1
Internalizing	65.1	12.0	64.0	9.8
Teacher Ratings – IOWA Conners Scores				
Inattention/ Overactivity	11.2	3.2	10.7	3.0
Oppositional/Defiant	8.0	4.7	8.2	4.5
Comorbid Diagnosis	%	#	%	#
ODD	48.0	47	47.9	34
CD	32.7	32	29.6	21
Anxiety disorder	31.6	31	33.8	24
Mood disorder	10.2	10	16.9	12

Note. n's may differ due to missing variables.

*t (159) = -2.0, p = .05

Follow-Up Sample

The follow-up sample included 98 probands and 85 adolescent controls recruited from the same communities as the probands and similar in age, sex, race/ethnicity, SES and FSIQ. Controls were initially screened for a history of ADHD using the ADHD module from the NIMH-DISC (Shaffer et al., 1996); those previously treated for ADHD or with a history of two or more symptoms during any 6-month period were excluded. Controls were not excluded for the presence of psychiatric disorders other than those described above for the initial sample of ADHD children. There were no significant differences between the ADHD and control groups in age (18.4 vs. 18.5), sex (88.8% vs. 87.1% male), SES (43.6 vs. 40.9), or FSIQ (93.0 vs. 96.8) (all *p*-values >.05).

Of the 183 probands and controls who participated in the follow-up study, 161 (88.0%) were male; all but three were between the ages of 16-21 years of age[1] [mean (SD): 18.4(1.7)]. As in the original childhood study, the sample (both probands and controls) was ethnically diverse – with 26.8% African American, 35.5% Latino, 25.1% Caucasian, and 12.6% of mixed ethnicity. The sample was varied in terms of SES, but was mainly lower to lower middle class, with a mean score of 42.3 (SD = 17.2, range = 20 - 96).

Footnote

[1]Three subjects fell outside this range; 15 years nine months, 25 years five months, and 22 years one month.

This study was approved by the Institutional Review Boards of the Mount Sinai School of Medicine and Queens College, City University of New York. Participants over the age of 18 signed their own statement of informed consent for participation in the study. When participants were under the age of 18, parents signed written statements of informed consent for their own and their child's participation. Assent was obtained from all participants younger than 18. Participants were compensated for their time and travel expenses.

Measures

Childhood evaluation. Children were assessed cognitively using the WISC-R or the WISC-III (Wechsler D., 1974; Wechsler D., 1991). The childhood diagnosis of ADHD was based on information obtained from parents and teachers. Only children with teacher ratings on the IOWA Conners Inattention/Overactivity scale indicative of clinically-significant difficulties with attention and hyperactivity at school (Pelham, Milich, Murphy, & Murphy, 1989) were entered into the study. Parents were interviewed using either the DISC version 2.1 (Shaffer, Fisher, Piacentini, Schwab-Stone, & Wicks, 1989), which uses DSM-III-R criteria (American Psychiatric Association, 1987) or the DISC version 2.3 (Shaffer et al., 1996), which reflects DSM-IV (American Psychiatric Association, 1994) criteria, depending on their date of entry. Parents also provided ratings of psychopathology on the Child Behavior Checklist (CBCL; Achenbach, 1991). Those classified using DSM-III-R and DSM-IV criteria did not differ on any parent or teacher ratings, suggesting that the subsamples were roughly equivalent.

Follow-up evaluation

Assessment of childhood maltreatment. Childhood Maltreatment was assessed during the follow-up evaluation using the short form of the Childhood Trauma Questionnaire (CTQ-SF; Bernstein et al., 1994; Bernstein et al., 2003a). The CTQ-SF is a 28-item self-report measure that screens adults and adolescents for histories of childhood abuse and neglect. The CTQ-SF is a brief, reliable and valid means of retrospectively assessing childhood maltreatment with test-retest reliability coefficients over four months ranging from .79 to .86, and internal consistency coefficients ranging from .66 to .92 across samples (Bernstein, Ahluvalia, Pogge, & Handelsman, 1997; Bernstein et al., 2003). Subjects rate statements about childhood trauma according to frequency on a 5-point Likert scale as 'never true', 'rarely true', 'sometimes true', 'often true', and 'very often true'. Minimization and denial of abuse and neglect are rated on a three-item scale which is incorporated into the questionnaire to detect false-negative trauma reports. The CTQ-SF assesses five types of maltreatment; Emotional Abuse, Physical Abuse, Sexual Abuse, Emotional Neglect, and Physical Neglect, with each type of maltreatment represented by five items. The CTQ-SF provides cutoff scores from none to low, low to moderate, moderate to severe, and severe to extreme exposure for each of the five types of maltreatment. Good specificity and sensitivity of cutoff scores to classify maltreated subjects have been reported (Bernstein et al., 2003; Bernstein, Stein, & Handelsman, 1998). For the purposes of this study, two maltreatment variables were created 1) a dichotomous variable of maltreated/not-maltreated where individuals were categorized as maltreated if they met criteria for one or more sub-types of maltreatment using the cut scores provide in the CTQ manual and 2) a dimensional variable where cutoff scores were enumerated (0=none to low, 1=low to moderate, 2=moderate to severe, and 3=severe to extreme) and summed across all five subtypes

subtype to form a composite severity score for overall maltreatment (range 0-15).

Assessment of socioeconomic status. Socioeconomic status was assessed using a measure of socioeconomic prestige developed at the National Opinion Research Center (Nakao & Treas, 1994). This measure approaches the issue of measuring socioeconomic status by ranking the relative prestige of an individual's occupation. Although this approach has been used in sociological and economics research, it has not been widely used in health research. Occupational prestige scaling is a process whereby occupations are ranked on a scale from 1 to 100 for its perceived prestige. The rankings are derived from surveys that ask respondents to attach a ranking to the occupation. Thousands of occupations are classified and the rankings are updated periodically. Information used to determine the socio-economic prestige score was obtained from the parents during both the baseline and follow-up assessments.

Assessment of adolescent substance use. Adolescent substance use was assessed using several measures. Initially, the Rutgers Alcohol and Drug Use Questionnaire (Labouvie, Bates, & Pandina, 1997) was used to systematically evaluate the subject's overall drug and alcohol use. The RADQ assesses current and past use of cigarettes, alcohol, marijuana, cocaine, and other prescription and non-prescription drugs. Respondents were asked to report the frequency and amount of drug and alcohol use in the past three years. Secondly, the substance abuse supplemental module of the Kiddie-SADS-Present Lifetime Version (K-SADS-PL; Kaufman, Ryan, Rao, Brent, & J.Brimaher, 1996) was used to interview adolescents and parents separately about subject's substance use. The K-SADS is a semi-structured diagnostic interview designed to ascertain current and lifetime psychopathology including SUDs. It contains algorithms to generate categorical diagnoses of substance use based on the criteria that have been established in the DSM-IV. Interviewing was conducted by trained clinicians, and interviews were carried

out separately with adolescents and their parents as informants. Responses were combined across raters by item; if either informant or the clinician indicated that the item caused significant distress or impairment, the symptom was judged to be present. Finally, a urine toxicology screen was collected from each subject on the day of evaluation, analyzed for the presence of marijuana, cocaine, amphetamines and opiates, and used to corroborate subject report, although this could not be used to determine the proband's diagnostic status. In addition, to facilitate honest responding and to maintain strict measures of confidentiality, we obtained a certificate of confidentiality from the National Institute of Health. Information gathered from all sources was used in conjunction to inform the clinician on the individual subject's drug and alcohol habits and guided the clinician to probing for the specific criteria needed to make a diagnosis of drug of alcohol abuse and/or dependence in accordance with the criteria set forth in the DSM-IV. Additionally, before a final SUD diagnosis was rendered, two independent teams of evaluators reviewed all pertinent clinical information provided during the course of the follow-up evaluation. Evaluator ratings were completed independently and final diagnosis was dependent upon evaluator agreement. A diagnosis of substance abuse or dependence for alcohol and drugs in accordance with DSM-IV criteria was formulated using parent and adolescent responses from the K-SADS along with information obtained from the RADQ. Measures of abuse and dependence were collapsed to create a dichotomous variable of substance abuse/dependence versus no substance abuse/dependence.

Criminal database. Detailed juvenile and adult criminal records for the entire sample were obtained from the New York State Division of Criminal Justice Services, Albany, which houses the official data for all offenses committed in New York State. A detailed description of our prospective follow-up study (including methods, sample characteristics, goals, and

significance) was submitted to the Division of Criminal Justice Services for review. On approval, a nondisclosure agreement was signed by the principal investigator (J.M.H). This agreement stipulated that all arrest history data would not be disclosed in a manner that could identify an individual, would be used only for research purposes, and would be treated as strictly confidential (e.g., the data would be coded, secured in locked cabinets, not copied or circulated). This large criminal records data set includes numerous variables such as; age of first arrest, number of arrests, type of offense (drug related or not), conviction records, as well as details of recidivism.

Statistical Procedures

Chi-square analyses were used to determine group (ADHD/control) differences in history of childhood maltreatment, SUDs, arrest, conviction, incarceration, and drug related crime type (dichotomous measures). Student's t-tests were used to determine group differences for age at time of criminal database (CDB) collection, childhood maltreatment severity, age of first arrest, and rates of recidivism (continuous variables). To further characterize the sample, chi-square analyses were used to determine differences among ADHD and controls groups separated by childhood maltreatment history on rates of arrest and substance use.

To investigate the predictive value of childhood maltreatment on criminal and SUD outcomes, separate logistic regressions for probands and controls were performed to determine the effect of maltreatment on arrest, drug related arrest, and SUD diagnosis (dichotomous outcomes), and linear regressions were performed to determine the effect of maltreatment on age of first arrest and rates of recidivism (continuous variables). Socioeconomic status and age at the time of CDB were entered as control variables in all regression analyses. To investigate whether individuals with both a history of maltreatment and SUD diagnoses are at increased risk for drug

related crime, this interaction term was entered into regression analyses examining drug related arrest outcome.

Separate logistic regression analyses using the whole group were used to explore the possibility that maltreatment increases the risk for arrest or SUD outcome primarily in those with a childhood diagnosis of ADHD. Age and SES were entered as control variables; group status (ADHD/control) and maltreatment (continuous variable) were entered as predictor variables; and a group by maltreatment variable was entered as an interaction term.

Results

At the adolescent follow-up, 46 probands (47.4%) and 25 controls (29.4%) met criteria for a SUD. At the time of the criminal data base collection, 41 probands (41.8%) and 30 controls (35.3%) had been arrested. Rates of arrest, conviction, incarceration, and recidivism did not differ significantly between probands and controls (all *p*-values >.05). Probands were significantly younger than controls at the time of their first arrest [16.9 (1.7) vs. 18.2 (2.3); t (63) = -2.6, $p = .011$] and had significantly higher rates of SUD diagnoses at the adolescent follow-up (47.4% vs. 29.4%; $\chi^2 = 6.2$, $p = .01$). Surprisingly, although the two groups differed significantly, with probands having almost twice the rate of SUD diagnosis when compared to controls, rates of drug related arrests did not differ between the two groups. Further, probands and controls did not differ on rates or severity of childhood maltreatment or SES. Group differences for select variables are listed in Table 3.

Table 3

Group Differences on Select Variables

	Probands n = 98	Controls n = 85	χ^2/t	*p*
Arrested[b**]	41.8 % (41)	35.3 % (30)	.82	.37
Convicted[b**]	24.4 % (24)	20 % (17)	.98	.32
Incarcerated[b**]	13.3 % (13)	8.2 % (7)	1.45	.23
Age of First Arrest[b*]	**16.9 (1.7)**	**18.2 (2.3)**	**-2.62**	**.01**
Rates of Recidivism[b*]	1.7 (3.3)	1.2 (2.5)	1.00	.32
Drug Related Arrest[b**]	19.4 % (19)	22.4 % (19)	.55	.46
SUD[a†]**	**47.4 % (46)**	**29.4 % (25)**	**6.18**	**.01**
SES[a*]	43.2 (17.5)	40.7 (16.8)	.97	.33
Childhood Maltreatment[a-] dimensional††*	3.5 (2.7)	2.9 (2.3)	1.14	.26
Childhood Maltreatment[a-] dichotomous††**	72.6 % (61)	62.2 % (51)	2.05	.15

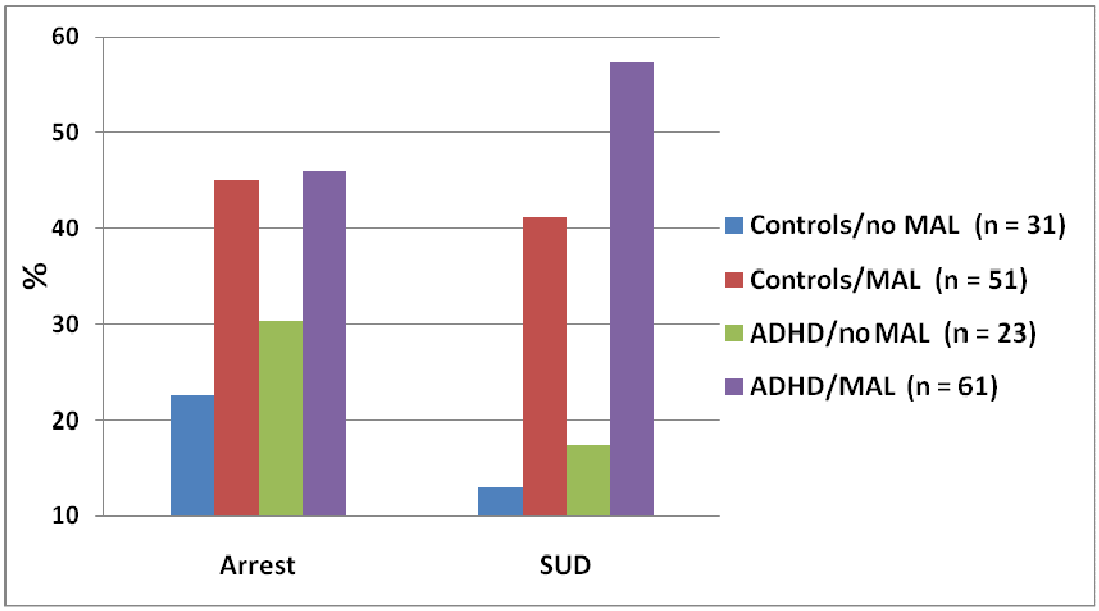
Note. [*]Mean (SD), [**] % (n), [a]assessed at follow-up, [b]assessed at time of criminal data collection. Significant variables are in bold. [†] SUD diagnosis was not obtained for one proband, [††] childhood maltreatment data was not completed for three controls and 14 probands.

When divided into four groups (probands and controls with and without childhood maltreatment history) probands and controls with maltreatment had significantly higher rates of arrest when compared to controls without maltreatment ($\chi^2 = 4.21$, $p = .04$ and $\chi^2 = 4.74$, $p = .03$ respectively). In terms of SUD outcome, probands and controls with maltreatment had significantly

higher rates of SUDs when compared to controls without maltreatment (χ^2 = 7.27, p = .007 and χ^2 = 17.23, p <.001 respectively). In addition, both probands and controls with maltreatment had significantly higher rates of SUD diagnoses when compared to the probands without maltreatment (χ^2= 4.01, p = .05 and χ^2= 11.19, p = .001 respectively). Although the difference did not meet statistical significance, there was a trend for higher rates of SUD diagnoses among the probands with maltreatment when compared to the controls with maltreatment (χ^2= 3.25, p = .07), which might suggest a possible synergistic effect of ADHD and maltreatment on substance use outcomes. Figure 1 shows the outcome rates of the four groups.

Figure 1

Rates of Arrest and SUD Diagnosis among the Four Groups formed by ADHD and Childhood Maltreatment Status.



Note. Significant contrasts for arrest 1 < 2 & 4. Significant contrasts for SUD 1 < 2 & 4, 2 > 3, 3 & 2 < 4

Does childhood maltreatment predict antisocial outcomes?

Results from regression analyses revealed that severity of maltreatment was a significant predictor of young adult arrest (Wald = 6.13, p = .01) and rates of recidivism (t = 3.23, p = .002) for probands, after controlling for SES and age at CDB. In contrast, severity of maltreatment was not a significant predictor of young adult arrest or rates of recidivism among controls. Severity of maltreatment did not predict age of first arrest or drug related crime for either probands or controls. In terms of substance use outcomes, severity of maltreatment was a significant predictor of SUDs for both probands (Wald = 13.79, p = <.001) and controls (Wald = 4.76, p = .03). Results of the regression analyses are presented in Tables 4 and 5. Further, the interaction term between maltreatment and SUD was not significantly associated with drug related arrest for either probands or controls. Substance use disorder diagnosis was a significant predictor of drug related arrest among controls (Wald = 3.97, p = .05), but not probands. The overall group regression analyses examining a group by maltreatment interaction did not reach significance for either arrest or SUD outcome.

Table 4

Logistic Regression Analyses Examining Severity of Childhood Maltreatment as a Predictor of Arrest, Drug Related Arrest, and SUD Diagnosis.

	β (SE)	*Wald*	*OR (95%CI)*	*P*
Probands				
Arrest	**.26 (.10)**	**6.13**	**1.29 (1.06 – 1.58)**	**.01**
Drug Related Arrest	.09 (.16)	.308	1.09 (0.79 -1.49)	.58
SUD	**.48 (.13)**	**13.79**	**1.62 (1.26 - 2.09)**	**<.001**
Controls				
Arrest	.13 (.12)	1.16	1.14 (0.90 - 1.44)	.28
Drug Related Arrest	.22 (.18)	1.51	1.25 (0.87 – 1.77)	.22
SUD	**.25 (.12)**	**4.76**	**1.29 (1.03 – 1.61)**	**.03**

Note. Significant variables are in bold.

Table 5

Linear Regression Analyses Examining Severity of Childhood Maltreatment as a Predictor of Age of First Arrest and Rates of Recidivism

	β (SE)	*t*	*P*
Probands			
Age of First Arrest	-.15 (.11)	-.76	.45
Rate of Recidivism	**.32 (.12)**	**3.23**	**.002**
Controls			
Age of First Arrest	.05 (.17)	.25	.80
Rate of Recidivism	.09 (.12)	.87	.42

Note. Significant variables are in bold.

Discussion

To our knowledge, this is the first prospective study to investigate the effect of childhood maltreatment on antisocial outcomes in an ethnically-diverse, lower SES sample of urban youth diagnosed with ADHD in childhood. Our results establish, for the first time, a robust link between maltreatment and later SUDs and criminality in ADHD youth. Among probands, maltreatment emerged as a significant predictor of arrest, recidivism, and SUDs. Among controls, maltreatment emerged as a significant predictor of SUDs, but was not associated with any variables of criminality. Overall, our findings regarding outcomes of ADHD youth are consistent with the extant child abuse literature demonstrating that children with histories of maltreatment have elevated rates of later criminality (Widom, 1989a; Smith & Thornberry, 1995;

Zingraff et al., 1993; Ireland et al., 2002; Smith et al., 2005) and SUDs (Liebschutz et al., 2002; Moran et al., 2004; Ondersma, 2007; Wall & Kohl, 2007). These results clearly indicate the need to consider a history of childhood maltreatment as an important component in the assessment and treatment of ADHD youth, as the presence of this additional adversity represents an added risk for both criminal and substance abuse outcomes in this already "at risk" group.

In contrast to most major prospective studies of children with ADHD (Mannuzza et al., 1989; Barkley et al., 1990; Fischer et al., 2002; Weiss et al., 1985) we did not identify significant discrepancies between probands and controls in rates of arrest, conviction, incarceration, or recidivism. Arrest rates were quite high for controls in this sample (35.3%); most probably a reflection of the sociodemographic composition of the sample and a possible reason for not identifying significant differences between ADHD and control subjects on most criminal outcome variables. This suggests that a diagnosis of ADHD may have less of an impact when studying criminal outcomes in lower SES, minority urban youth. The same cannot be said for substance use outcomes. Despite possible 'mitigating' sociodemographic variables, we identified significant discrepancies between ADHD and control groups in rates of SUDs, similar to extant studies of ADHD youth. Similar to Mannuzza et al. (1989), we found that subjects with an arrest record did have significantly higher rates of SUDs compared to subjects without an arrest record (66.2% versus 21.4%; $\chi^2 = 36.2$, $p = <.001$) suggesting that criminality may be at least in part mediated by the development of SUDs (Barkley, 2002), although the temporal sequence of events (e.g., if substance use preceded arrest) was not available in our data. Interestingly, SUD was a significant predictor of drug related crime among controls but not among probands, which is in contrast to findings of Barkley et al. (2004).

There are several limitations to be considered when interpreting these findings. First,

there is the relatively high attrition rate from the original sample. The difficulty to find many individuals from this highly mobile, largely lower SES group is not surprising – especially given the passage of nearly 10 years. While available data suggest that the follow-up sub-sample is representative of the original group, it is possible that those lost to follow-up had different and perhaps worse outcomes. Secondly, identification of childhood maltreatment was based solely on retrospective reports from each participant. Although considerable data support the reliability and validity of the CTQ (Bernstein et al., 1997; Bernstein et al., 2003; Fink, Bernstein, Handelsman, Foote, & Lovejoy, 1995; Scher, Stein, Asmundson, McCreary, & Forde, 2001), self-report measures are susceptible to a variety of biases including social desirability, mood at time of report, and memory limitations. Nevertheless, had maltreatment been assessed during childhood, there would be increased likelihood for false negatives due to parental under-reporting as well as the possibility of the childhood maltreatment occurring subsequent to our childhood evaluation. Furthermore, as is true of other longitudinal research in the area of ADHD, this sample is largely male, and it is therefore difficult to generalize the findings to females. Finally, the present study did not control for possible contributory effects of comorbid childhood psychopathologies (e.g. CD) or familial factors (e.g. parental psychopathology). The contributions of childhood psychiatric comorbidities, mainly CD, has been established as a potent risk factor for both criminal and SUDs outcomes in several studies of ADHD youth (Armstrong & Costello, 2002; Brook et al., 1995; Disney et al., 1999; Barkley et al., 2004), and should be included in future investigations into the salience of maltreatment as an independent risk factor for poor outcome in ADHD youth. In conclusion, this study has established a history of maltreatment as a potent predictor of SUD and criminal activity among ADHD youth. Existing longitudinal studies of ADHD may have limited our ability to fully understand key

issues regarding antisocial outcomes in non-Caucasian, urban children from lower and lower-middle socioeconomic strata. We have found that discrepancies in antisocial outcomes between probands and controls found in prior studies of ADHD youth are not necessarily applicable to samples of lower SES, ethnically diverse urban youth. Considering the fact that minorities are much more likely to be arrested and convicted and subsequently represent the bulk of prison populations (Washburn et al., 2008), more diverse samples should be the target of future ADHD research in order to develop a clearer understanding the mechanisms that underlie antisocial behavior in this group. From the results obtained in this study, it appears that SUD may be less dependent on sociodemographic variables as our data replicates those of prior studies of ADHD youth.

Study II: Childhood Maltreatment and Conduct Disorder: Independent Predictors of Criminality in Young Adults Diagnosed with ADHD in Childhood

Longitudinal studies of youth with attention deficit/hyperactivity disorder (ADHD) have consistently reported higher rates of antisocial behavior in adolescence and early adulthood when compared to typically-developing controls (Barkley et al., 1990; Mannuzza et al., 1989; Satterfield & Schell, 1997; Hechtman et al., 1984). The relationship between childhood ADHD and later criminality is clearly illustrated by arrest rates in these youth which range between 39–57% (Mannuzza et al., 1989; Babinski, Hartsough, & Lambert, 1999; Satterfield & Schell, 1997; Barkley et al., 2004). Further, among prison populations, rates of ADHD have been reported to range from 35 - 54% (Ulzen & Hamilton, 1998; Robertson, Dill, Husain, & Undesser, 2004; Andrade, Silva, & Assumpcao, 2004; Abram, Teplin, McClelland, & Dulcan, 2003), and studies have suggested that up to 70% of juvenile offenders meet criteria for the diagnosis (Shelton & Pearson, 2005; Teplin, Abram, McClelland, Washburn, & Pikus, 2005; Ulzen & Hamilton, 1998; Vreugdenhil, Doreleijers, Vermeiren, Wouters, & van den, 2004). When compared to rates of ADHD in the general population which range from 5-10% (Scahill & Schwab-Stone, 2000), the numbers clearly indicate a relationship between ADHD and later antisocial and/or criminal behavior.

ADHD and Criminal Outcomes

ADHD is a heterogeneous disorder which often presents with significant comorbidity making it difficult to discern if risk for negative outcome is attributable to ADHD alone or other conditions that commonly present with the disorder. While ADHD is believed to predispose

individuals to later antisocial behavior and criminality, data suggest that other factors, such as early aggression (Fergusson, Lynskey, & Horwood, 1996; MacDonald & Achenbach, 1999), hyperactive-impulsive symptoms (Lahey, Loeber, Burke, & Applegate, 2005; Satterfield & Schell, 1997), and/or early conduct disorder (CD) (Babinski et al., 1999; Hildyard & Wolfe, 2002) play a role. For example, prospective studies of community samples (Farrington, 1989; Farrington & Loeber, 2000; Stattin & Magnusson, 1989) have found that childhood conduct problems predict adult antisocial behavior, but that childhood ADHD alone does not. Similarly, in a comprehensive review of childhood ADHD studies examining antisocial activity, Lilienfeld and Waldman (1990) reported that ADHD, in the absence of aggression or conduct problems, was not related to later antisocial behavior (Lilienfeld & Waldman, 1990). Conversely, others have suggested that ADHD in childhood predicts later antisocial behavior (Lambert & Hartsough, 1998; Mannuzza et al., 1989) above and beyond that accounted for by early aggression or other factors. To date, much of the ADHD literature has focused on the contributions of childhood CD as a risk factor for later antisocial behavior and criminality. While it is clear that early CD accounts for a substantial portion of the risk associated with later antisocial behavior and criminality in youth with ADHD, with most researchers identifying links between early conduct problems and this poor outcome (Barkley et al., 1990; Disney et al., 1999), the extent to which other factors contribute to later criminality in this group remains unclear.

Childhood Maltreatment and Criminal Outcomes

Several major population based prospective studies carried out in the United States have reported robust links between maltreatment in childhood and subsequent delinquent behavior

(Maxfield & Widom, 1996; Widom, 1989a; Smith & Thornberry, 1995; Zingraff et al., 1993; Ireland et al., 2002; Smith et al., 2005) showing that childhood maltreatment significantly increases the risk of engaging in antisocial behavior in adolescence and adulthood (Widom, 1989b; Widom, 1989a; Cicchetti & Manly, 2001; Lansford et al., 2002). For example, in the Rochester Youth Development Study (Smith & Thornberry, 1995), adolescents with a history of abuse or neglect were significantly more likely to have an arrest record when compared to non-abused controls. Similarly, a longitudinal study of Midwestern youth found that childhood maltreatment increased the overall risk of juvenile arrest by 55% and arrests for violent crime by 96% (Maxfield & Widom, 1996).

Although childhood maltreatment has been identified as a potent predictor of later criminality in several population based studies, this risk factor has not been systematically examined in individuals with ADHD. Several major longitudinal studies of ADHD have investigated antisocial outcomes and the contributions of concomitant conduct problems; however, none has examined the contributory role of childhood maltreatment. Given that consistent links have been established between 1) childhood maltreatment and subsequent criminality (Widom, 1989a; Smith & Thornberry, 1995; Zingraff et al., 1993; Ireland et al., 2002; Smith et al., 2005); 2) ADHD and later antisocial behavior (Barkley et al., 1990; Klein & Mannuzza, 1991; Mannuzza et al., 1989; Satterfield & Schell, 1997; Hechtman et al., 1984); and 3) ADHD and the likelihood of being maltreated (Briscoe-Smith & Hinshaw, 2006; Cicchetti & Manly, 2001; Ford et al., 2000), elucidation of the influence of childhood maltreatment on criminal outcomes in ADHD is warranted and may provide important insights into the etiology of criminality in this highly heterogeneous group of children.

The present study seeks to establish the relationship between childhood maltreatment and

young adult criminality in a large, ethnically diverse sample of urban youth diagnosed with ADHD in childhood. Further, as CD has been identified as a potent risk factor to criminal outcomes in ADHD groups, we investigated whether childhood maltreatment predicts young adult criminality above and beyond contributions of CD and other known risk factors such as parental antisocial behavior (Gittelman et al., 1985; Barkley et al., 1990; Hechtman & Weiss, 1986). We hypothesized that a history of childhood maltreatment would increase the likelihood of later criminality, corroborated by official arrest records, and that childhood maltreatment would emerge as a contributor to this risk over and above the contributions of childhood CD and parental antisocial behavior.

Method

Participants

The study sample consisted of 98 adolescents (11 females) who represent a sub-sample derived at follow-up from a longitudinal study of adolescents diagnosed with ADHD at baseline during childhood (N = 169). The baseline childhood sample was referred by schools, physicians, or mental health providers to a study focusing on the biology of ADHD and other disruptive behavior disorders. The childhood sample as a whole was rated as having significant behavior problems by both parents and teachers, and all participants were diagnosed with ADHD. Children were between the ages of 7-11 years, with a mean (SD) age of 8.99 (1.30) years at the time of initial evaluation. The present young adult sample comprised those who participated in a 10 year follow-up assessment and were assessed for a history of childhood maltreatment. Criminal data for this sample was obtained approximately three years after the adolescent follow-up when participants ranged in age between 18 and 26 years, with a mean (SD) age of

21.6 (2.2) years. The follow-up sample was ethnically diverse, comprised of 32.2% Latino, 26.7% Caucasian, 23.3% African American, and 17.8% of mixed or other ethnicity. The participants were generally of lower to lower-middle socioeconomic status (Mean = 36.5; SD = 17.8) on a measure of socioeconomic prestige (Nakao & Treas, 1994), although a wide range of socio-economic status was represented (Range: 11-85). The sample was almost exclusively urban, and all participants were English speaking. Individuals with a diagnosis of schizophrenia, pervasive developmental disorder, Tourette's syndrome, or a Full Scale IQ below 70 were excluded from entry into the initial childhood study.

The original group was not recruited for a longitudinal study and was comprised of a highly diverse and mobile inner-city population who were difficult to locate. As such, we did not anticipate re-evaluation of the complete sample. Of the 169 childhood subjects located, 18 refused participation, five were incarcerated, and one individual was deceased. Of the remaining 145 subjects, 98 (67.6%) completed the follow-up evaluation. The group that was lost to follow-up did not differ significantly from those followed in age at child evaluation, parent or teacher ratings of behavior, or in their rates of ODD, CD, mood or anxiety disorders as assessed during childhood (all $p > .05$). The two groups did differ significantly on childhood FSIQ, with those followed having a significantly higher scores than those lost to follow-up (94.0 vs. 89.3; $t(159) = 2.0$, $p = .05$). Overall, those included in the follow-up were considered to be representative of the original childhood sample.

This study was approved by the Institutional Review Boards of the Mount Sinai School of Medicine and Queens College, City University of New York. Participants over the age of 18 signed their own statement of informed consent for participation in the study. When participants were under the age of 18, parents signed written statements of informed consent for their own

and their child's participation. Assent was obtained from all participants younger than 18. Participants were compensated for their time and travel expenses.

Measures

Childhood evaluation. Children were assessed cognitively using the WISC-R or the WISC-III (Wechsler D., 1974; Wechsler D., 1991). The childhood diagnosis of ADHD was based on information obtained from parents and teachers. Only children with teacher ratings on the IOWA Conners Inattention/Overactivity scale indicative of clinically-significant difficulties with attention and hyperactivity at school (Pelham et al., 1989) were entered into the study. Parents were interviewed using either the DISC version 2.1 (Shaffer et al., 1989), which uses DSM-III-R criteria (American Psychiatric Association, 1987) or the DISC version 2.3 (Shaffer et al., 1996), which reflects DSM-IV (American Psychiatric Association, 1994) criteria, depending on their date of entry. Parents also provided ratings of psychopathology on the Child Behavior Checklist (CBCL)(Achenbach, 1991). Those classified using DSM-III-R and DSM-IV criteria did not differ on any parent or teacher ratings, suggesting that the subsamples were roughly equivalent.

Parent history assessment at baseline. As part of the childhood evaluation, family history of each participant was assessed for symptomatology of antisocial behavior using a semi-structured interview (Halperin, Newcorn, Mckay, Siever, & Sharma, 2003). The interview was administered to either the child's mother, both parents, or another relative with whom the child lived. Initially, each respondent completed a genogram diagramming the child's first- and second-degree relatives. The genogram was then used as a guide while the interviewer systematically asked about the past and present antisocial behavior patterns of each family

member. When a positive report of antisocial behavior was elicited, additional probes were used to determine whether this represented a persistent pattern of behavior or caused functional impairment for the individual. Antisocial behavior in a family member was operationally defined as a persistent pattern of antisocial behavior that was marked by excessive violence, use of weapons, or persistent problems with the law. Table 6 shows childhood characteristics of the sample.

Table 6

Characteristics of the Childhood Sample for Study II

	Total (*N = 98*)
CBCL Externalizing	69.7 (11.2)*
CBCL Internalizing	65.1 (12.0)*
ODD	48.0%
CD	32.7 %
Anxiety Disorder	31.6 %
Mood Disorder	10.2 %
Parental ASB	43.0 %
Childhood Maltreatment**	72.6 %

* Mean *(SD),***assessed at follow-up.

Follow-up evaluation

Assessment of childhood maltreatment. Childhood Maltreatment was assessed using the short form of the Childhood Trauma Questionnaire (CTQ-SF) (Bernstein et al., 1994; Bernstein et al., 2003). The CTQ-SF is a 28-item self-report measure that screens adults and adolescents for histories of childhood abuse and neglect. The CTQ-SF is a brief, reliable and valid means of retrospectively assessing childhood maltreatment with test-retest reliability coefficients over four months ranging from .79 to .86, and internal consistency coefficients ranging from .66 to .92 across samples (Bernstein et al., 1997; Bernstein et al., 2003). Subjects rate statements about childhood trauma according to frequency on a 5-point Likert scale as 'never true', 'rarely true', 'sometimes true', 'often true', and 'very often true'. Minimization and denial of abuse and neglect are rated on a three-item scale which is incorporated into the questionnaire to detect false-negative trauma reports. The CTQ-SF assesses five types of maltreatment; Emotional Abuse, Physical Abuse, Sexual Abuse, Emotional Neglect, and Physical Neglect, with each type of maltreatment represented by five items. The Emotional Abuse items assess the extent to which the child was verbally demeaned or degraded (e.g., "People in my family said hurtful or insulting things to me"). The Physical Abuse items determine the degree of physical assault(s) the child has endured (e.g., "I was punished with a belt, a board, a cord, or some other hard object"). The Emotional Neglect items estimate the level at which the child's emotional needs were met (e.g., "My family was a source of strength and support" [reverse scored]). The Physical Neglect items assess if the child's physical needs were met (e.g., 'I didn't have enough to eat"). Finally, the Sexual Abuse items assess if the child was coerced into sexual scenarios (e.g., "Someone tried to touch me in a sexual way, or tried to make me touch them"). The CTQ-SF provides cutoff scores from none to low, low to moderate, moderate to severe, and severe to

extreme exposure for each of the five types of maltreatment. Good specificity and sensitivity of cutoff scores to classify maltreated subjects have been reported (Bernstein et al., 2003a; Bernstein et al., 1998). The CTQ produces both dimensional and categorical levels for each form of maltreatment to which cutoff scores are used to classify individuals as abused or not abused. For the purposes of this study, two maltreatment variables were created 1) a dichotomous variable of maltreated/not-maltreated where individuals were categorized as maltreated if they met criteria for one or more sub-types of maltreatment using the cut scores provide in the CTQ manual and 2) a dimensional variable where cutoff scores were enumerated (0=none to low, 1=low to moderate, 2=moderate to severe, and 3=severe to extreme) and summed across all five subtypes subtype to form a composite severity score for overall maltreatment (range 0 -15).

Assessment of socioeconomic status. Socioeconomic status was assessed using a measure of socioeconomic prestige developed at the National Opinion Research Center (Nakao & Treas, 1994). This measure approaches the issue of measuring socioeconomic status by ranking the relative prestige of an individual's occupation. Although this approach has been used in sociological and economics research, it has not been widely used in health research. Occupational prestige scaling is a process whereby occupations are ranked on a scale from 1 to 100 for its perceived prestige. The rankings are derived from surveys that ask respondents to attach a ranking to the occupation. Thousands of occupations are classified and the rankings are updated periodically. Information used to determine the socio-economic prestige score was obtained from the parents during the baseline assessment.

Criminal data. Detailed juvenile and adult criminal records for the sample were obtained from the New York State Division of Criminal Justice Services, Albany, New York, which

houses the official data for all offenses committed in New York State. A detailed description of our prospective follow-up study (including methods, sample characteristics, goals, and significance) was submitted to the Division of Criminal Justice Services for review. On approval, a nondisclosure agreement was signed by the principal investigator (J.M.H). This agreement stipulated that all arrest history data would not be disclosed in a manner that could identify an individual, would be used only for research purposes, and would be treated as strictly confidential (e.g., the data would be coded, secured in locked cabinets, not copied or circulated). This large criminal records data set includes numerous variables such as; age of first arrest, number of arrests, type of offense (drug related or not), as well as details of recidivism. In this study, arrest record, age of first arrest, and rates of recidivism will be used as indicators of young adult criminality.

Statistical Procedures

To examine group differences on several possible risk factors we separated the sample into two groups by arrest record (arrested/never arrested). Chi-square analyses were used to determine group differences on dichotomous variables (e.g. ODD, CD, anxiety disorder, mood disorder, parental antisocial behavior, and childhood maltreatment) and Student's t-test were used to determine differences between groups on continuous variables (e.g. age at time of criminal data base (CDB) collection, CBCL internalizing and externalizing scores, and childhood maltreatment severity).

To determine if childhood maltreatment contributed risk to the development of criminality in young adulthood over and above other known risk factors, binary logistic forward (Wald) regressions were used to determine the relative risk of childhood maltreatment, childhood

CD, and parental antisocial behavior on young adult arrest outcome. Childhood SES and age at the time of CDB were entered into the first step of the regression analysis as control variables. Dichotomous variables for childhood CD, and parental antisocial behavior, and a continuous variable for childhood maltreatment were entered on the second step and served as independent predictor variables. To explore the possibility that maltreatment increased risk for young adult arrest primarily in those with either parental antisocial behavior or childhood CD, these interaction terms were entered on the third step. All variables were centered before creating the multiplicative interaction terms and running the regression analysis. A dichotomously coded arrest variable (ever/never arrested) served as the dependent measure for the logistic regression. Linear regression analyses were used to determine if childhood maltreatment, childhood CD, and parental antisocial behavior added unique risk to 1) age of first arrest and 2) rates of recidivism. As with the logistic regression analysis, childhood SES and age at the time of CDB were entered into the first step of the analyses as control variables. Dichotomous variables for childhood CD, and parental antisocial behavior, and a continuous variable for childhood maltreatment were entered as predictor variables. Continuous variables for age of first arrest and rates of recidivism were used as dependent measures in the linear regression analyses.

Results

Among the 98 participants, eight individuals were not evaluated for a history of childhood maltreatment, parental antisocial behavior history was not collected for two subjects, and six individuals were detected as underreporting on the minimization and denial scales of the CTQ and excluded from further analyses. Of the remaining 82 participants (9 females), 33 (40.2%) had been arrested at the time of the CDB. Fifty-nine participants (72%) met criteria for

at least one type of maltreatment. Severity of childhood maltreatment was significantly higher in young adults who had been arrested than those without an arrest history [4.6 (3.0) vs. 2.6 (2.1); t (80) = -3.0, p =.004].

At baseline, 27 (32.9%) of the children were diagnosed with CD. Rates of childhood CD were significantly higher in young adults who had been arrested than those without an arrest history (51.5% vs. 20.4%; $\chi^2 = 8.6$, $p = .003$). The group that had been arrested did not differ significantly from the group without an arrest history in terms of socioeconomic background or parental history of ASB. Group comparisons are summarized in Table 7.

Table 7

Group Differences on Various Risk Factors

	Never arrested *(n=49)*	Arrested *(n=33)*	χ^2/t	*P*
Age[c] ***at time of CDB***	**20.8 (2.1)***	**22.5 (1.9)***	**-3.7**	**<.001**
SES[a]	38.3 (17.0)*	34.2 (19.3)*	1.0	.32
CBCL Externalizing[a]	69.1 (11.9)*	70.3 (11.1)*	-.47	.64
CBCL Internalizing[a]	66.0 (11.8)*	64.2 (12.6)*	.61	.54
ODD[a]	53.1 %	39.4 %	1.5	.22
CD[a]	**20.4 %**	**51.5 %**	**8.6**	**.003**
Anxiety disorder[a]	34.7 %	24.2 %	1.0	.31
Mood disorder[a]	6.1 %	15.2 %	1.8	.18
Parental ASB[a]	34.7 %	45.5 %	.96	.26
Childhood Maltreatment[b dimensional]	**2.6 (2.1)***	**4.6 (3.0)***	**-3.0**	**.004**
Childhood Maltreatment[b-dichotomous]	67.3 %	78.8 %	1.3	.26

Note. * Mean *(SD),* [a]assessed at baseline, [b]assessed at follow-up, [c] at time of criminal data collection. Significant variables are in bold.

As expected, parental antisocial behavior, low SES, childhood CD, and maltreatment were not unrelated. Although correlations were modest, parental antisocial behavior was significantly associated with childhood CD and lower SES, but not maltreatment. In addition,

the presence of childhood CD was associated with increased likelihood for maltreatment. Low SES was not significantly associated with childhood maltreatment (see Table 8). Despite statistically significant intercorrelations between predictor variables, use of the statistically conservative stepwise method in the regression analyses adjusts for intercorrelations within the model.

Table 8

Inter-correlations of Predictor Variables

	SES	Maltreatment	CD
Parental ASB	**†-.30 (<.01)****	-.10 (.37)	**.29 (<.01)****
SES		.07 (.51)	**-.25 (.02)***
Maltreatment			**.23 (.04)***

Note. †r-value(sig). Significant correlations are in bold.

Do Maltreatment, Conduct Disorder and Parental ASB Independently Predict Young Adult Criminal Outcome?

Results from the logistic regression analysis revealed that only one of the three risk factors was significantly associated with young adult arrest after controlling for other variables in the equation. Childhood maltreatment emerged as an independent risk for young adult arrest (Wald = 6.80, p = .01) over and above the contributions of other factors (see Table 9). Childhood CD and parental ASB were not independently associated with arrest outcome as

defined in the model. The interactions between childhood maltreatment and childhood CD and childhood maltreatment and parental ASB did not contribute significantly to young adult arrest. In terms of the relative influence of the risk factors on young adult arrest, a history of childhood maltreatment emerged as the strongest association over childhood CD and parental ASB. Specifically, a one-unit increase in the severity of childhood maltreatment increased the likelihood of arrest by 30 percent.

Table 9

Logistic Regression Analyses Examining Childhood Maltreatment, CD, and Parental Antisocial Behavior as Predictors of Young Adult Arrest

Variable	*β (SE)*	*Wald*	*OR (95%CI)*	*P*
Childhood SES (Control Variable)	**-.04 (.02)**	**4.71**	**.96 (.93-1.0)**	**.03**
Age at CDB (Control Variable)	**.51 (.15)**	**11.36**	**1.6 (1.24-2.23)**	**.001**
Childhood Maltreatment	**.26 (.10)**	**6.80**	**1.3 (1.07-1.58)**	**.01**
CD	.90 (.58)	2.43	2.5 (.79-7.69)	.12
Parental ASB	.45 (.61)	.56	1.6 (.48-5.17)	.46
Maltreatment x CD	2.0 (1.40)	2.07	7.5 (.48-116.34)	.15
Maltreatment x Parental ASB	.83 (.90)	.85	2.3 (.39-13.23)	.36

Note: significant variables are in boldface type.

Results from linear regression analyses revealed that the severity of childhood maltreatment ($\beta = .23$, $p = .03$) and childhood CD ($\beta = .28$, $p = .02$) were significantly related to recidivism, but not to the age of first arrest (see Tables 10 and 11).

Table 10

Linear Regression Analyses Examining Childhood Maltreatment, CD, and Parental Antisocial Behavior as Predictors of Age of First Arrest

Variable	b	*SE* b	*B*
Childhood SES (Control Variable)	.00	.03	-.01
Age at CDB (Control Variable)	.23	.25	.25
Childhood Maltreatment	-.01	.15	-.01
CD	.09	.86	.03
Parental ASB	-.38	.91	-.11

Note. All variables ns.

Table 11

Linear Regression Analyses Examining Childhood Maltreatment, CD, and Parental Antisocial Behavior as Predictors of Rates of Recidivism

Variable	*b*	*SE b*	*B*
Childhood SES (Control Variable)	-.03	.02	-.18
Age at CDB (Control Variable)	**.27**	**.13**	**.21***
Childhood Maltreatment	**.23**	**.11**	**.23***
CD	**1.65**	**.66**	**.28***
Parental ASB	-.06	.61	-.01

Note. Adjusted R^2 for the model = .22. * $p < .05$

Discussion

The major aim of this study was to investigate the association between childhood maltreatment and the development of criminal behavior among young adults diagnosed with ADHD in childhood. We sought to elucidate whether maltreatment predicted later criminality above and beyond other commonly associated risk factors such as childhood CD and parental ASB (Smith & Farrington, 2004; Thornberry, Freeman-Gallant, & Lovegrove, 2009). As hypothesized, our results identified a robust relationship between childhood maltreatment and later criminality in this group. Our data indicate that childhood maltreatment independently contributed to young adult arrest over and above the variance accounted for by the other variables in the model. Strikingly, after controlling for childhood SES and age at time of criminal data collection, childhood maltreatment was a stronger predictor than childhood CD and parental ASB, two traditionally potent predictors of young adult criminality. Further, both childhood maltreatment and childhood CD were related to rates of recidivism, indicating that perhaps CD is more related to the frequency of criminal behavior, rather than the initiation of criminal activity. Overall, our findings are consistent with the extant child abuse literature demonstrating that children with histories of maltreatment have elevated rates of criminality in adolescence and young adulthood (Ireland et al., 2002; Smith & Thornberry, 1995; Smith et al., 2005; Widom, 1989c; Widom & Maxfield, 1996; Zingraff et al., 1993).

Consistent links have been shown between parent's ASB and ASB in their offspring (Smith & Farrington, 2004; Thornberry, Freeman-Gallant, Lizotte, Krohn, & Smith, 2003; Thornberry et al., 2009), and it is likely that this familial transmission is accounted for by a combination of genetic and environmental factors. As parents with ASB have been reported to

be more likely to maltreat their children (Verona & Sachs-Ericsson, 2005), this may be a particularly important environmental factor, although the association was not identified in this sample. As previously noted, children with behavior problems, such as CD, are at increased risk for developing ASB in adolescence. Albeit plausible to suppose that those youth at increased risk of ASB due to early CD would be more susceptible to the influence of childhood maltreatment, we did not find an interaction effect between childhood CD and childhood maltreatment or parental ASB and childhood maltreatment in our sample. Not surprisingly, we did find that parental ASB was inter-correlated with childhood CD and childhood CD was inter-correlated with childhood maltreatment, but after accounting for all of these factors, only maltreatment was directly linked to young adult arrest.

The results of this study should be interpreted in the context of several limitations. First, identification of childhood maltreatment was based solely on retrospective reports from each participant. Although considerable data support the reliability and validity of the CTQ (Bernstein et al., 1997; Bernstein et al., 2003; Fink et al., 1995; Scher et al., 2001), self-report measures are susceptible to a variety of biases including social desirability, mood at time of report, and memory limitations. Nevertheless, had maltreatment been assessed during childhood, there would be increased likelihood for false negatives due to parental under-reporting as well as the possibility of the childhood maltreatment occurring subsequent to our childhood evaluation, which occurred nearly 10 years ago for most of the participants. In addition, the classification of parental ASB as determined during the baseline evaluation was developed to cast a broad net to encompass a wide range of behaviors, but specific/detailed information about the frequency and severity of antisocial acts was not obtained. Nevertheless, significant associations clearly emerged and increasing the adequacy of these measures would likely make the findings more

robust. Secondly, despite all efforts, we were unable to follow all 169 individuals from the initial childhood study. Although available data suggest that our subsample was representative of the original group, the current sample size limits power, which may have affected our ability to detect other main effects in our primary analysis independent of maltreatment; in particular CD. Finally, as in many studies of ADHD, the low rate of female participants in our sample limits the generalizability of these results as it relates to girls diagnosed with ADHD in childhood.

This study was designed to elucidate pathways leading to young adult criminality in children with ADHD using arrest record, age of first arrest, and rates of recidivism as indicators of this poor outcome. While data have consistently indicated that comorbid CD accounts for a substantial proportion of the variance associated with later criminality in this population, findings have been mixed as to whether ADHD alone poses increase risk for later criminal activity. Our findings indicate that a third, independent factor, childhood maltreatment, must be considered. This finding is not surprising given the fact that maltreatment has been linked to young adult antisocial behavior in other populations. However, it has been completely overlooked in longitudinal research in ADHD examining antisocial outcomes, despite the fact that these children are at increased risk for maltreatment.

Implications for Research, Policy, and Practice

Identifying childhood and family risk factors is essential in the prevention of juvenile antisocial behavior. Indeed, determining which specific risks, or combination of risks, impact on later delinquent outcomes allows practitioners to identify most efficiently which children are most at-risk and thus to target prevention and treatment more effectively. The findings from this

study have important implications with regard to antisocial outcomes and emphasize the utility of assessing childhood maltreatment in ADHD populations. Maltreatment has been largely ignored in the investigation of criminality in this population, and as previously noted, children with behavior disorders, such as ADHD, are at elevated risk for maltreatment and later criminality. As such, clinicians providing services to individuals with ADHD should be aware of the implications of co-occurring maltreatment and the risks associated therein. Accordingly, the assessment and diagnostic process of ADHD referrals should include screening for possible childhood maltreatment, as this would help to identify ADHD youth with enhanced vulnerability for later antisocial behavior.

Study III: Childhood Maltreatment and Conduct Disorder: Independent Predictors of Adolescent Substance Use Disorders in Youth with ADHD

Attention-deficit/hyperactivity disorder (ADHD) is one of the most frequently diagnosed childhood psychiatric disorders with prevalence rates in school-aged children ranging from 5-10% (Scahill & Schwab-Stone, 2000). Although once thought of as limited to childhood, ADHD is now recognized as a heterogeneous disorder (Biederman et al., 1991) that persists through adolescence and into adulthood (Barkley et al., 1990; Biederman et al., 1996; Mannuzza et al., 1998a; Weiss et al., 1985) for many individuals. Longitudinal studies have consistently shown childhood ADHD to be a significant contributing factor to poor outcome in adolescence and adulthood (Barkley et al., 1990; Fischer et al., 1990; Hechtman & Weiss, 1986; Mannuzza et al., 1989; Mannuzza et al., 1993). Negative outcomes often associated with childhood ADHD include heightened risk for adolescent psychopathology (Gittelman et al., 1985; Satterfield & Schell, 1997; Barkley et al., 2004; Hechtman et al., 1984), antisocial behavior, problems with employment, driving, and sexual relationships (Barkley, Guevremont, Anastopoulos, Dupaul, & Shelton, 1993; Barkley, Fischer, Smallish, & Fletcher, 2006) as well as substance use disorders (SUDs) (Biederman et al., 2006; Barkley et al., 2004; Mannuzza & Klein, 2000). Factors that contribute to negative outcomes in this population include high rates of psychiatric comorbidity such as oppositional defiant disorder (ODD), conduct disorder (CD), and a variety of mood and anxiety disorders (Biederman et al., 1991; Fischer et al., 2002; Jensen et al., 1997). In addition, there are many psychosocial and environmental factors such as family substance abuse and low socioeconomic status (SES) which potentially contribute to poor outcome in this population.

Among the negative outcomes often associated with ADHD, SUDs are particularly problematic (Mannuzza et al., 1993; Mannuzza et al., 1998; Wilens et al., 1998; King et al., 2004). Prevalence rates of adolescent and adult SUDs among individuals diagnosed with ADHD range between 30-43% (Fischer et al., 2002), a more than two-fold increase over the 8-15% reported in the general population (Olfson et al., 2000; Thomas et al., 2005). Investigations into potential risk factors for later substance abuse in children with ADHD have yielded mixed results. Some researchers have highlighted the role of early conduct problems and externalizing symptoms (Armstrong & Costello, 2002; Brook et al., 1995; Disney et al., 1999). Others have reported that severity of childhood ADHD symptoms predicts substance abuse over and above that accounted for by childhood antisocial symptoms (Molina & Pelham, 2003). Still others have emphasized the role of the persistence of ADHD, CD and antisocial symptoms into adolescence (Gittelman et al., 1985; Mannuzza et al., 1991; Mannuzza & Klein, 2000). It seems likely that several factors play a contributory role in SUD outcomes, and, while these studies clearly indicate a role for ADHD and comorbid externalizing disorders in the emergence of adolescent SUDs, the extent to which other factors play a role has received considerably less study.

Individuals with histories of childhood maltreatment are also at heightened risk for developing SUDs in adolescence and adulthood (Moran et al., 2004; Wall & Kohl, 2007a). Rates of maltreatment in samples of individuals with drug and alcohol abuse disorders are reported as high as 77-84% (Cohen & Densen-Gerber, 1982; Triffleman et al., 1995) while rates of childhood maltreatment in the general population range between 25-40% (Moran et al., 2004; Scher et al., 2004). Although children diagnosed with ADHD are known to be at increased risk for maltreatment due to externalizing behaviors and dysfunctional peer and parental relations (Briscoe-Smith & Hinshaw, 2006; Ford et al., 1999), the relationship between maltreatment and

SUDs is relatively unstudied in this population. Given that consistent links have been established between childhood maltreatment and SUDs as well as between externalizing disorders and SUDs (Appleyard et al., 2005; Biederman, Wilens, Mick, Faraone, & Spencer, 1998; Mannuzza et al., 1998), it seems logical to examine how these factors influence youth with ADHD who are known to be at heightened risk for both of these conditions.

To our knowledge, no study has examined the contributory role of childhood maltreatment in the development of later SUDs among individuals diagnosed with ADHD in childhood. Because of prior research indicating that children with ADHD are at heightened risk for maltreatment (Appleyard et al., 2005; Ford et al., 1999), as well as later SUDs (Biederman et al., 1998; Mannuzza et al., 1998), this is an important area of study. The major aim of the present study was to investigate whether maltreatment proffers added risk over other known contributory factors such as early CD (Gittelman et al., 1985; Barkley et al., 1990; Hechtman & Weiss, 1986), parental alcohol and drug use (Biederman, Faraone, Monuteaux, & Feighner, 2000; Prescott & Kendler, 1999; Clark, Cornelius, Kirisci, & Tarter, 2005; Chassin, Curran, Hussong, & Colder, 1996; Johnson, Leonard, & Jacob, 1989), and lower SES (Duncan, Duncan, Hops, & Alpert, 1997). We hypothesized that a history of childhood maltreatment would increase the likelihood of a SUD in adolescence, and that this risk would be above and beyond that accounted for by childhood CD, parental substance use and lower SES.

Method

Participants

The study sample consisted of 86 adolescents (75 males) who represent a sub-sample derived at follow-up from a longitudinal study of adolescents diagnosed with ADHD at baseline

during childhood (N = 169). The baseline childhood sample were all clinically referred for behavioral difficulties by schools, physicians, or mental health providers as part of a study focusing on the biology of ADHD and other disruptive behavior disorders. The childhood sample as a whole was rated as having significant behavior problems by both parents and teachers, and all participants were diagnosed with ADHD. Children were between the ages of 7-11 years, with a mean *(SD)* age of 8.99 (1.30) years at the time of initial evaluation. The present adolescent sample (*n* = 86) were those who participated in the follow-up assessment, approximately 10 years later. The follow-up participants ranged in age between 16-21 years, with a mean *(SD)* age of 18.21 (1.33) years. The follow-up sample was ethnically diverse, comprised of 22.1% African American, 26.7% Caucasian, 32.6% Latino, and 18.6% of mixed or other ethnicity. The participants were generally of lower to lower-middle SES status (Mean socio-economic prestige = 44.03; *SD* = 17.40) on a measure of socioeconomic prestige (Nakao & Treas, 1994), although a wide distribution of socio-economic prestige was represented (Range: 20-96). The sample was almost exclusively urban, and all participants were English speaking. Individuals with a diagnosis of schizophrenia, pervasive developmental disorder, Tourette's syndrome, or a Full Scale IQ below 70 were excluded from entry into the initial childhood study.

The original group was not recruited for a longitudinal study and was comprised of a highly diverse and mobile inner-city population who were difficult to locate. As such, we did not anticipate re-evaluation of the complete sample. Of the 169 childhood subjects, 112 families (66.3%) were located. Among those located, 86 (76.8%) completed the follow-up re-evaluation, 18 refused participation, seven were incarcerated, and one individual was deceased. The group that was lost to follow-up (*n* = 83) did not differ significantly in age at child evaluation, parent or teacher ratings of behavior, or in their rates of ODD, CD, mood or anxiety disorders as assessed

during childhood (all $p > .05$) from those followed ($n = 86$). Overall, those included in the follow-up appear to be representative of the original childhood sample.

This study was approved by the Institutional Review Boards of the Mount Sinai School of Medicine and Queens College, City University of New York. Participants over the age of 18 signed their own statement of informed consent for participation in the study. When participants were under the age of 18, parents signed written statements of informed consent for their own and their child's participation. Assent was obtained from all participants younger than 18. Participants were compensated for their time and travel expenses.

Baseline evaluation. The childhood diagnosis of ADHD, CD, ODD, anxiety and mood disorders was based on information obtained from parents and teachers. Parents were interviewed regarding their child's diagnosis using either the Diagnostic Interview Schedule for Children (DISC) version 2.1 (Shaffer et al., 1989), which incorporates diagnostic criteria from Diagnostic and Statistical Manual - Third Edition –Revised (DSM-III-R; (American Psychiatric Association, 1987), or the DISC version 2.3 (Shaffer et al., 1996), which reflects criteria from DSM-IV (American Psychiatric Association, 1994), depending on their date of entry into the study. Parents and teachers also rated the severity of various disruptive behaviors using the Child Behavior Checklist (CBCL; Achenbach, 1991) and the IOWA Conners Teacher Questionnaire (IOWA; Loney & Milich, 1982), respectively. Although diagnoses for those recruited before 1994 were made on the basis of DSM-III-R criteria, virtually all participants would likely have met DSM-IV criteria for ADHD, Combined Type. To examine differences due to differing criteria for ADHD between the DSM-III-R and DSM-IV, we reviewed all ADHD symptom data from the two groups. The groups did not differ with regard to parent or teacher ratings, or in rates of comorbidity (all $p > .05$). Table 12 shows childhood characteristics

of the sample.

Table 12

Characteristics of the Childhood Sample for Study III

	Total (N = 86)
CBCL Externalizing	68.8 *(11.4)**
CBCL Internalizing	64.7 *(11.9)**
ODD	48.8%
CD	26.7%
Anxiety Disorder	29.1%
Mood Disorder	9.3%
Parental Substance Abuse	51.3%
Childhood Maltreatment**	71.3%

* Mean *(SD),***assessed at follow-up

The family history of each proband was assessed for symptomatology of substance abuse using a semi-structured interview (Halperin, Schulz, Mckay, Sharma, & Newcorn, 2003) administered to either the child's mother alone, both parents, or another relative with whom the child lived. Initially, each respondent completed a genogram diagramming the child's first- and second-degree relatives. The genogram was then used as a guide while the interviewer systematically asked about the past and present substance use patterns of each family member. When a positive report of substance use was elicited, additional probes were used to determine

whether the use was abusive, represented a persistent pattern of behavior, or caused functional impairment for the individual. Since the family history interview examined both past and present disturbances, the reported rates of symptomatology in relatives reflect lifetime prevalence. Problematic substance use was operationally defined as use to a level where problems were experienced by the individual either at home or at work. This study focused exclusively on parental history of substance use.

Follow-up evaluation. Childhood Maltreatment was assessed using the Childhood Trauma Questionnaire (CTQ). The CTQ is a brief, reliable and valid means of retrospectively assessing childhood maltreatment with test-retest reliability coefficients over four months ranging from .79 to .86, and internal consistency reliability coefficients ranging from .66 to .92 across samples (Bernstein et al., 1997; Bernstein et al., 2003). The CTQ (Bernstein et al., 1994; Bernstein et al., 2003) is a 28-item self-report measure that screens adults and adolescents for histories of childhood abuse and neglect. Subjects rate statements about childhood trauma according to frequency on a 5-point Likert scale as 'never true', 'rarely true', 'sometimes true', 'often true', and 'very often true'. Minimization and denial of abuse and neglect are rated on a three-item scale which is incorporated in the questionnaire to detect false-negative trauma reports. Item scores are transferred to clinical scales for five types of maltreatment: Emotional Abuse, Physical Abuse, Sexual Abuse, Emotional Neglect, and Physical Neglect, with each type of maltreatment represented by five items. The Emotional Abuse items assess the extent to which the child was verbally demeaned or degraded (e.g., "People in my family said hurtful or insulting things to me"). The Physical Abuse items determine the degree of physical assault(s) the child has endured (e.g., "I was punished with a belt, a board, a cord, or some other hard object"). The Emotional Neglect items estimate the level at which the child's emotional needs

were met (e.g., “My family was a source of strength and support” [reverse scored]). The Physical Neglect items assess if the child’s physical needs were met (e.g., ‘I didn’t have enough to eat”). Finally, the Sexual Abuse items assess if the child was coerced into sexual scenarios (e.g., “Someone tried to touch me in a sexual way, or tried to make me touch them”). The CTQ produces both dimensional and categorical levels for each form of maltreatment to which cutoff scores are used to classify individuals as abused or not abused. For the purposes of this study, individuals were categorized as maltreated if they met criteria for one or more sub-types of maltreatment using the cut scores provide in the CTQ manual. A dichotomous variable of maltreated/not-maltreated was used in all analyses.

Socioeconomic status was assessed using a measure of socioeconomic prestige developed at the National Opinion Research Center (Nakao & Treas, 1994). This measure approaches the issue of measuring SES by ranking the relative prestige of the individual’s occupation. Although this approach has been used in sociological and economics research, it has not been widely used in health research. Occupational prestige scaling is a process whereby occupations are ranked on a scale from 1 to 100 for its perceived prestige. The rankings are derived from surveys that ask respondents to attach a ranking to the occupation. Thousands of occupations are classified and the rankings are updated periodically. Information about parental occupation was obtained from parents during the follow-up assessment.

Adolescent substance use was assessed using several measures. Initially, the Rutgers Alcohol and Drug Use Questionnaire (Labouvie et al., 1997) was used to systematically evaluate the subject’s overall drug and alcohol use. The RADQ assesses current and past use of cigarettes, alcohol, marijuana, cocaine, and other prescription and non-prescription drugs. Respondents were asked to report the frequency and amount of drug and alcohol use in the past

three years. Secondly, the substance abuse supplemental module of the Kiddie-SADS-Present Lifetime Version (K-SADS-PL; Kaufman et al., 1996) was used to interview adolescents and parents separately about subject's substance use. The K-SADS is a semi-structured diagnostic interview designed to ascertain current and lifetime psychopathology including SUDs. It contains algorithms to generate categorical diagnoses of substance use based on the criteria that have been established in the DSM-IV. Interviewing was conducted by trained clinicians, and interviews were carried out separately with adolescents and their parents as informants. Responses were combined across raters by item; if either informant or the clinician indicated that the item caused significant distress or impairment, the symptom was judged to be present. Finally, a urine toxicology screen was collected from each subject on the day of evaluation, analyzed for the presence of marijuana, cocaine, amphetamines and opiates, and used to corroborate subject report, although this could not be used to determine the proband's diagnostic status. In addition, to facilitate honest responding and to maintain strict measures of confidentiality, we obtained a certificate of confidentiality from the National Institute of Health. Information gathered from all sources was used in conjunction to inform the clinician on the individual subject's drug and alcohol habits and guided the clinician to probing for the specific criteria needed to make a diagnosis of drug of alcohol abuse and/or dependence in accordance with the criteria set forth in the DSM-IV. Additionally, before a final SUD diagnosis was rendered, two independent teams of evaluators reviewed all pertinent clinical information provided during the course of the follow-up evaluation. Evaluator ratings were completed independently and final diagnosis was dependent upon evaluator agreement. A diagnosis of substance abuse or dependence for alcohol and drugs in accordance with DSM-IV criteria was formulated using parent and adolescent responses from the K-SADS along with information

obtained from the RADQ. Measures of abuse and dependence were collapsed to create a dichotomous variable of *substance abuse/dependence* versus *no substance abuse/dependence.*

Statistical Procedures

To examine group differences on several possible risk factors, we separated the sample into two groups; adolescents with a SUD, and adolescents without a SUD. Chi-square analyses were used to determine group differences on dichotomous measures and Student's t-test were used to determine differences between groups on continuous variables. To determine if childhood maltreatment contributed risk to the development of SUDs in adolescence over and above other known risk factors, binary logistic forward (Wald) regressions were used to determine the relative risk of childhood maltreatment, childhood CD, and parental substance abuse on SUD outcomes. Socioeconomic status was entered into the first step of the regression analysis as a control variable. Dichotomous variables for childhood maltreatment, childhood CD, and parental substance abuse were entered on the second step, and served as independent predictor variables. To explore the possibility that maltreatment increased risk for adolescent SUD primarily in those with either parental substance abuse or childhood CD, these interaction terms were entered on the third step.

Results

Of the 86 participants, six individuals were detected as underreporting by the minimization and denial scales of the CTQ and excluded from further analyses. Of the remaining 80 participants (70 males), 34 (42.5%) were diagnosed with a SUD at follow-up. Fifty-seven participants (71.3%) met criteria for at least one type of maltreatment. Adolescents

diagnosed with SUDs were significantly more likely than those without a diagnosis to report histories of childhood maltreatment (91.2% vs. 56.5%; $\chi^2 = 11.46$, $p = .001$). Of those adolescents who reported a history of childhood maltreatment, 31 (55.4%) met criteria for a SUD while only three (13.0%) of those with no history of childhood maltreatment met SUD criteria.

Forty-one (51.3%) adolescents had at least one parent who was identified as having a history of problematic substance use. Adolescents diagnosed with SUDs had significantly higher rates of parental substance use problems as compared to those without a SUD diagnosis (67.6% vs. 39.1%; $\chi^2 = 6.36$, $p = .01$). Among those adolescents who had a parent with problematic substance use, 23 (56.1%) met criteria for a SUD while 11 (28.2%) of those who did not have a parental history of problematic substance use met SUD criteria.

At baseline, 23 (28.8%) of the children were diagnosed with CD. Adolescents diagnosed with SUDs had significantly higher rates of childhood CD as compared to those without a SUD diagnosis (47.1% vs. 15.2%; $\chi^2 = 9.68$, $p = .002$). Among those adolescents with a childhood diagnosis of CD, 16 (69.6%) met criteria for a SUD while 18 (31.6%) of those who did not have a childhood diagnosis of CD met SUD criteria. The SUD group came from significantly lower socioeconomic backgrounds when compared to the non SUD groups [39.2 (13.9) vs. 47.9 (19.7); t (78) = 2.21, $p = .03$]. Group comparisons are summarized in Table 13.

As expected, parental substance abuse, low SES, childhood CD, and maltreatment were not unrelated. Although correlations were modest, problematic parental substance use was significantly associated with childhood CD ($r = .23$, $p < .05$) and lower SES ($r = -.28$, $p < .05$), but not maltreatment ($r = .02$, $p > .10$). In addition, the presence of childhood CD was associated with increased likelihood for maltreatment ($r = .23$, $p < .05$); SES was not associated with maltreatment or childhood CD (both $p > .10$). Despite statistically significant intercorrelations

between predictor variables, none of the relationships account for more than five percent of the variance and therefore were not considered a threat the accuracy of the regression estimates (Cohen & Cohen, 1983).

Table 13

Group Differences on Various Risk Factors

	No SUD *(n=46)*	SUD *(n=34)*	χ^2/t	*p*
Age[b]	18.0 (1.36)*	18.4 (1.29)*	1.29	.72
SES[b]	**47.9 (19.7)***	**39.2 (13.9)***	**-2.21**	**.03**
CBCL Externalizing[a]	67.7 (11.0)*	67.2 (9.6)*	-0.24	.82
CBCL Internalizing[a]	62.8 (11.0)*	62.1 (11.8)*	-0.20	.84
ODD[a]	54.3 %	41.2 %	1.36	.24
CD[a]	**15.2 %**	**47.1 %**	**9.68**	**.002**
Anxiety disorder[a]	34.8 %	23.5 %	1.18	.28
Mood disorder[a]	6.5 %	11.8 %	0.67	.41
Parental Substance Abuse[a]	**39.1%**	**67.6%**	**6.36**	**.01**
Childhood Maltreatment[b]	**56.5%**	**91.2%**	**11.46**	**.001**

* Mean *(SD)*, [a]assessed at baseline, [b]assessed at follow-up

Do Maltreatment, Conduct Disorder and Parental Substance Abuse Independently Predict Adolescent Substance Use Disorders?

Results from the logistic regression analysis revealed that two of the three risk factors were significantly associated with the emergence of adolescent SUDs after controlling for other variables in the equation. Childhood maltreatment (Wald = 7.58 , p = .006), and childhood CD (Wald = 4.88, p = .03) were independently related to adolescent SUDs after controlling for SES. Despite a notable trend (p < .07), parental substance use was not independently associated with adolescent SUDs. The interaction between childhood maltreatment and childhood CD was not significant, although the interaction between childhood maltreatment and parental substance use showed a trend (p = .06) such that among those with parental substance use, maltreatment had a greater impact upon the probability of SUDs in the offspring (see Table 14). In terms of the relative influence of the risk factors on adolescent SUDs, a history of childhood maltreatment emerged with the strongest association over childhood CD and parental substance use.

Table 14

Logistic Regression Examining Childhood Maltreatment, CD, and Parental Substance Use as Predictors of Adolescent SUDs

Variable	*β (SE)*	*Wald*	*OR (95%CI)*	*p*
SES (Control Variable)	.03 (.02)	3.02	1.03 (0.99-1.07)	.08
Childhood Maltreatment	**1.99 (.72)**	**7.58**	**7.30 (1.77-31.14)**	**.006**
CD	**1.26 (.567)**	**4.88**	**3.52 (1.15-10.73)**	**.03**
Parental Substance Use	1.19 (.645)	3.38	3.28 (0.93-11.60)	.07
Maltreatment x Parental Substance Use	1.13 (.595)	3.58	3.08 (0.96-9.88)	.06
Maltreatment x CD	-2.37 (1.74)	1.85	.09 (.003-2.84)	.17

Note. Significant variables are in bold.

Discussion

The major aim of this study was to investigate the association between childhood maltreatment and the development of SUDs among adolescents diagnosed with ADHD in childhood. We sought to elucidate whether maltreatment predicted SUDs above and beyond other commonly associated risk factors such as childhood CD and parental substance use. As hypothesized, our results identified a robust relationship between childhood maltreatment and later SUDs in this group. Our data indicate that childhood maltreatment independently contributed to SUD outcome over and above the variance accounted for by the other variables in the model. Strikingly, childhood maltreatment was a better predictor than childhood CD and parental substance use, two traditionally potent predictors of adolescent substance use

(Armstrong & Costello, 2002; Brook et al., 1995; Disney, Elkins, Mcgue, & Iacono, 1999; Prescott & Kendler, 1999).

Overall, our findings are consistent with the extant child abuse literature demonstrating that children with histories of maltreatment have elevated rates of SUDs in adolescence (Liebschutz et al., 2002; Moran et al., 2004; Ondersma, 2007; Wall & Kohl, 2007). Notably, within our sample of adolescents with a history of childhood ADHD, those who were maltreated had elevated rates of SUDs (55.4%), while those with ADHD and no maltreatment history had rates consistent with those reported in the general population (13.0%). This suggests that childhood maltreatment may play an important role in SUD outcome, and that ADHD itself, in the absence of maltreatment and/or comorbid CD, may not increase risk for later SUD.

Although consistent links have been shown between parent's substance abuse and substance abuse problems in their offspring (Biederman, Faraone, Monuteaux, & Feighner, 2000; Johnson, Leonard, & Jacob, 1989; Chassin, Curran, Hussong, & Colder, 1996; Clark, Cornelius, Kirisci, & Tarter, 2005; Prescott & Kendler, 1999), it is likely that this familial transmission is accounted for by a combination of genetic and environmental factors. Parents with SUD have been reported to be more likely to maltreat their children (Ammerman, Kolko, Kirisci, Blackson, & Dawes, 1999). Thus it would seem reasonable to hypothesize that those youth at increased risk due to parental substance use would be differentially more vulnerable to the negative impact of maltreatment. In support of this hypothesis we identified a statistical trend for the interaction effect between parental substance use and childhood maltreatment.

As previously noted, children with behavior problems, such as CD, are at increased risk for developing SUDs in adolescence (Mannuzza, Klein, Abikoff, & Moulton, 2004). Interestingly, we found that childhood maltreatment was inter-correlated with childhood CD.

Albeit logical to suppose that those youth at increased risk of SUDs due to early CD would be more susceptible to the influence of childhood maltreatment, we did not find an interaction effect between childhood CD and childhood maltreatment in this sample.

The results of this study should be interpreted in the context of several limitations. First, identification of childhood maltreatment was based solely on retrospective reports from each participant. Although considerable data support the reliability and validity of the CTQ (Bernstein et al., 1997; Bernstein et al., 2003; Fink et al., 1995; Scher et al., 2001), self-report measures are susceptible to a variety of biases including social desirability, mood at time of report, and memory limitations (Babor, Brown, & and DelBoca, 1990). Nevertheless, had maltreatment been assessed during childhood, there would be increased likelihood for false negatives due to parental under-reporting as well as the possibility of the childhood maltreatment occurring subsequent to our childhood evaluation, which occurred nearly 10 years ago for most of the adolescents. In addition, the classification of problematic parental substance use as determined during the baseline evaluation was developed to cast a broad net to encompass a wide range of impairing substance use behaviors, but the parents were not formally diagnosed with a SUD. Nevertheless, increased error in our assessment of maltreatment and/or problematic parent substance use would decrease the likelihood of finding associations with adolescent SUDs. Yet, significant associations clearly emerged. Increasing the adequacy of these measures would likely make the findings more robust. Furthermore, there was a ten year time span between baseline and follow-up assessments and it is possible that some parents would have developed substance use disorders during this time. Unfortunately we did not employ a measure at follow-up to supplement the baseline data. Finally, as in many studies of ADHD, the low rate

of female participants in our sample limits the generalizability of these results as it relates to girls diagnosed with ADHD in childhood.

This study was designed to elucidate pathways leading to later SUDs in children with ADHD. While data have consistently indicated that comorbid CD and parental substance abuse account for a substantial proportion of the variance associated with later SUDs in this population, findings have been mixed as to whether ADHD alone poses increase risk for later SUD. Our findings indicate that a third, independent factor, childhood maltreatment, must be considered. This finding is not surprising given the fact that maltreatment has been linked to adolescent SUDs in other populations. However, it has been completely overlooked in longitudinal research of ADHD youth, despite the fact that these children are at increased risk for maltreatment (Briscoe-Smith & Hinshaw, 2006). Our data suggest that children with ADHD who did not have childhood CD and were not maltreated are at no greater risk for later SUDs than children from the general population. Thus, ADHD alone does not appear to be an independent risk factor for later SUD.

Implications for Research, Policy, and Practice

These findings have important implications with regard to substance abuse outcomes and emphasize the utility of assessing childhood maltreatment in ADHD populations. Maltreatment has been largely ignored in the investigation of SUDs in this population, and as previously noted, children with behavior disorders, such as ADHD, are at elevated risk for maltreatment (Appleyard, Egeland, Van Dulmen, & Sroufe, 2005) and later SUDs (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1993; Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998; Biederman et al., 1998). As such, clinicians providing services to individuals with ADHD should be aware of the implications of co-occurring maltreatment and the risks associated therein. Accordingly, the

assessment and diagnostic process of ADHD referrals should include screening for possible childhood maltreatment, as this would help to identify ADHD youth with enhanced vulnerability for later SUDs.

GENERAL DISCUSSION

This series of studies represents the first to investigate a history of childhood maltreatment as a risk factor for criminal and SUD outcomes in ADHD youth. We explored this in an ethnically-diverse, lower SES sample of urban youth who were diagnosed with ADHD in childhood, and a group of well matched community controls. The links between a history of childhood maltreatment and later antisocial behavior have been well established in population based studies. Surprisingly, this risk factor has remained uninvestigated in ADHD youth, despite the fact that these children are at heightened risk for maltreatment (Briscoe-Smith & Hinshaw, 2006). Overall, we confirmed our hypothesis that a history of childhood maltreatment is a significant predictor of later criminality and SUDs among ADHD youth, even above and beyond well-established risk factors, mainly CD.

Overview of Study Findings

Study I reported on the criminal and substance use outcomes of a large cohort of ethnically diverse, lower socio-economic inner city youth with and without ADHD and established childhood maltreatment as a potent risk factor for both criminality and SUD outcomes in ADHD youth. In this study, youth with ADHD were found to be younger at the time of their first arrest and had higher rates of SUDs when compared to community controls. One surprising finding was that, unlike most prior longitudinal studies of ADHD, we did not identify differences between ADHD youth and controls on rates of arrest, conviction, incarceration or recidivism. Severity of maltreatment emerged as a significant predictor of adolescent SUD diagnosis among both probands and controls, and of arrest and rates of

recidivism among probands. However, we did not identify a relationship between maltreatment and measures of criminality among the controls in our sample, which was somewhat unexpected, considering results from population-based studies. Further, we found that a SUD diagnosis was related to arrest, while SUDs were only a predictor of drug related crime among controls. Although there was a trend for those with both maltreatment and ADHD to have higher rates of SUD outcomes, there was no evidence of synergistic effects of maltreatment and ADHD for either outcome. Notably, within this study, there were no significant differences in rates of arrest between probands and controls without a maltreatment history, which might suggest that ADHD in the absence of a maltreatment history does not increase risk for arrest.

Study II was designed to elucidate pathways leading to young adult criminality in children with ADHD using arrest record, age of first arrest, and rates of recidivism as indicators of criminality and establish the relative importance of maltreatment as a risk factor. Specifically, we sought to determine whether maltreatment predicted later criminality above and beyond other commonly associated risk factors such as childhood CD and parental ASB (Smith & Farrington, 2004; Thornberry et al., 2009). As hypothesized, we found that childhood maltreatment was a robust predictor of later criminality and, remarkably, after controlling for age and SES, childhood maltreatment was a stronger predictor of young adult criminality than childhood CD and parental ASB, two traditionally potent predictors of this outcome. Further, we identified links between rates of recidivism and both childhood maltreatment and CD suggesting that CD may be more associated to the frequency of criminal behavior. It was somewhat surprising that we did not identify CD as a significant independent risk factor for other criminality outcome measures (e.g. arrest and age of first arrest). Although data indicate that the subsample utilized in this study was representative of the original group, power was limited by the current sample

size, which may have affected our ability to detect CD as an independent risk factor for other outcomes related to criminality.

Study III focused on the association between childhood maltreatment and the development of SUDs among adolescents diagnosed with ADHD, and tested the contribution of maltreatment relative to established risk factors, mainly CD and parental substance use. We identified a robust relationship between childhood maltreatment and later SUDs and showed that maltreatment independently contributed to SUD outcome over and above the variance accounted for by childhood CD and parental substance use, two traditionally potent predictors of adolescent substance use (Armstrong & Costello, 2002; Brook et al., 1995; Disney et al., 1999; Prescott & Kendler, 1999). Notably in this study, rates of SUDs among ADHD youth without a maltreatment history were comparable to those in the general population. This was echoed from Study I where rates of SUDs among ADHD youth without a maltreatment history were not significantly higher than rates in controls without a maltreatment history. Together these findings suggests that the variance in SUD outcomes among ADHD youth may be more accounted for by conditions that commonly co-occur with the disorder (e.g., CD, maltreatment history, parental psychopathology, etc.), rather than ADHD in and of itself.

Our findings, at least as it pertains to ADHD youth, are in agreement with extant child abuse literature indicating elevated rates of SUDs (Liebschutz et al., 2002; Moran et al., 2004; Ondersma, 2007; Wall & Kohl, 2007) and criminality in abused children. Interestingly, we found that childhood maltreatment was inter-correlated with childhood CD. Albeit logical to suppose that those youth at increased risk of criminality and SUDs due to early CD would be more susceptible to the influence of childhood maltreatment, we did not find significant interactions between childhood CD and childhood maltreatment for either outcome. Further,

although plausible to suppose that youth at risk for poor outcome due to a familial history of antisocial behavior and substance use would be more susceptible to the influence of childhood maltreatment, we did not find an interaction effect between childhood maltreatment and familial history for antisocial or substance use outcomes in our sample.

Together these studies clearly establish a history of childhood maltreatment as a potent and important risk factor for later poor outcome in ADHD youth. In addition, it appears from our results that at least some portion of the poor outcome that has been attributed to CD in ADHD studies may in fact be due to childhood maltreatment.

Directions for Future Research

Results of studies in ADHD and maltreated youth demonstrate remarkable similarities in risk factors, symptom, anatomical abnormalities, and neurobiological dysregulation. The clinical presentation of ADHD and maltreatment can be quite similar and it is plausible to suppose that maltreated individuals with symptoms of inattention, impulsivity, and mood dysregulation may be wrongly diagnosed with ADHD because of "ADHD like" symptoms related to an underlying trauma. Alternatively, they may indeed have ADHD, and a comorbid trauma history is overlooked. The literature for both ADHD and maltreatment clearly indicates that both scenarios portend poor outcome. Where the two scenarios differ is in treatment approach and perhaps treatment outcome.

Evidence from pre-clinical studies has shown that early adversity affects several aspects of the mesolimbic DA system, mainly; increased DA release, fewer D2-DA receptors in the ventral tegmental area, fewer DA transporters in the nucleus accumbens (Meaney et al., 2002), and changes in behavioral measures of both reward and reinforcement (Matthews et al., 1999).

Further, rats exposed to early adversity exhibit increased intake of sucrose solution and increased self-administration of cocaine (Michaels & Holtzman, 2006; Matthews et al., 1999). The DA hypothesis of ADHD suggests abnormalities in the synaptic mechanisms of DA transmission; a deficit in DA has been suggested (Levy, 1991), as well as abnormalities in DA receptors and DA transporters (e.g. (Swanson et al., 1998). It is plausible to suggest that some of the DA system abnormalities associated with ADHD may be associated with a trauma history, and that this may be a pathway for vulnerability to later substance abuse in this group.

Preclinical and clinical studies have shown that stress in early childhood leads to long-term changes in the HPA axis. Disruptions in HPA axis functioning have also been implicated in the pathophysiology of ADHD (Hastings et al., 2009; King et al., 1998). Further, HPA axis dysfunction has been identified in numerous studies of alcohol and drug dependent individuals (Lovallo, Dickensheets, Myers, Thomas, & Nixon, 2000), and some have suggested that cortisol hyporeactivity in children may be a reflection of biological mechanisms underlying a risk for later SUDs (Croissant & Olbrich, 2004; Moss, Vanyukov, & Martin, 1995). We suggest that some of the abnormalities identified in HPA axis functioning in ADHD youth may be attributed to a maltreatment history and that these abnormalities could play a role in the vulnerability for, or development and maintenance of substance abuse and dependence among ADHD youth.

As with neurochemistry, it is plausible that some of the structural abnormalities identified in children with ADHD could be attributed to maltreatment. The most consistently documented area of structural abnormality in ADHD youth is the cerebellum. Abnormalities in the cerebellar vermis have been associated with various psychiatric disorders, suggesting that this region plays a critical role in mental health. The cerebellar vermis has a high density of glucocorticoid receptors - making this area particularly vulnerable to damage from prolonged exposure to

glucocorticoids. The cerebellar vermis modulates the brain-stem nuclei that control NE and DA production and release. Dysregulation of NE and DA can produce symptoms of depression, hyperactivity, and impair attention. Given the high rates of maltreatment in ADHD youth, it is plausible to suppose that some of the cerebellar abnormalities identified in ADHD youth may also be related to the effects of early adversity.

Could Conjunctive Treatment Significantly Affect Outcome?

Empirically based research seems to point in that direction. Preclinical studies of early trauma in rats have shown that treatment with SSRIs greatly attenuates the negative sequelae associated with early adversity (Huot et al., 2001). Although symptom reductions are seen in individuals with ADHD who receive stimulant treatment (Barbaresi et al., 2006; Conners, 2002), psychostimulant treatment without recognition of an underlying trauma, could exacerbate symptoms of anxiety resulting from the trauma. Clinical trials have shown that depression, anxiety and PTSD stemming from childhood maltreatment are responsive to SSRIs (Emslie et al., 1997; Emslie, 2009), cognitive behavior therapy (Emslie, 2009; Reinecke, Ryan, & DuBois, 1998), and psychotherapy (Birmaher et al., 2007). Our approach to clinical assessment of ADHD youth should include a screening for maltreatment history. After a positive history of maltreatment has been identified and a diagnosis of ADHD has been established, empirically based treatments should be recommended on a "case by case" basis, addressing symptoms stemming from <u>both</u> conditions. Conjunctive evidence-based treatments which include both pharmaco- and psycho-therapy could prove most beneficial for those individuals with ADHD and a history of early trauma.

Summary of Limitations

The results of the preceding studies should be interpreted in the context of several limitations that have been outlined in the conclusions of each study. First, identification of childhood maltreatment was based solely on retrospective reports. In addition, the classifications of parental ASB and parental substance abuse were based on a broad interview conducted during the baseline evaluation, which screened for multiple problematic behaviors. Therefore, detailed information about the frequency and severity of antisocial acts and substance use was not obtained. Despite all efforts, we were unable to follow all 169 individuals from the initial childhood study. Although available data suggest that our subsample was representative of the original group, the current sample size limits power, which may have affected our ability to detect effects in our primary analysis independent of maltreatment; in particular CD. Finally, as with most studies of ADHD youth, due to the low number of female participants in our sample, these results as it relates to girls diagnosed with ADHD in childhood are of limited generalizability.

Implications for Future Policy and Practice

These results are unique in that they represent outcomes of an understudied and under-serviced portion of the population that, in general, have less adequate access to health care services. Identifying individual and environmental risk factors is essential in the prevention of juvenile antisocial and substance abuse behavior. Indeed, determining which specific risks, or combination of risks, impact on later delinquent and substance use outcomes allows practitioners to identify those children most at-risk and target prevention and treatment more effectively. The findings from this present study have important implications with regard to antisocial and

substance use outcomes and emphasize the utility of assessing childhood maltreatment in ADHD populations. We identified elevated rates of poor outcome in ADHD youth with a history of childhood maltreatment, suggesting that children with both adversities should be the target of more intense services.

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