Neurocognitive Performance and Demographic Variables in Children at risk of Attention Deficit/Hyperactivity Disorder

Ariane Kalff
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ADHD in early childhood: An introduction
Attention Deficit/Hyperactivity Disorder (ADHD) is currently characterized by a persistent pattern of inattention, hyperactivity, and impulsiveness that is inappropriate for the developmental level of the child (American Psychiatric Association, 1994). It is the most commonly diagnosed psychiatric disorder of childhood and affects approximately 3–5% of the school-age population (Barkley, 1998; Tannock, 1998). These estimations vary considerably (between 1% and 24%), depending on the exact definition of ADHD and whether the population studied is a sample of the general population or a clinic-referred sample (Swanson et al., 1998). Overall, the prevalence has tended to increase over the last 20 years. The sex ratio also varies with male-to-female ratios ranging from 4:1 to 9:1 (American Psychiatric Association, 1994).

The disorder usually arises early in development and is clinically persistent (American Psychiatric Association, 1994). Follow-up studies have demonstrated that 30-80% of affected children continue to display symptoms of ADHD through adolescence into adulthood (Barkley, 1998; Biederman et al., 1998). The core symptoms cause significant impairments in family and peer relationships as well as an inability to succeed in school or work. Moreover, children with ADHD have an increased risk of additional psychopathology such as antisocial conduct, substance abuse, or even antisocial personality (Biederman, Wilens et al., 1995; Weiss & Hechtman, 1993). All together, ADHD has a substantial impact on society, and thus early diagnosis and early intervention programs are important. The enormous amount of research on ADHD that has been published in the past 20 years has mainly involved school-aged children, whereas only very few studies have focused on the early characteristics of ADHD (Mariani & Barkley, 1997). Moreover, the scant research on young children with or at risk of ADHD has focused primarily on behavioral and social precursors (Blackman, 1999). Because ADHD is a multidimensional disorder, studies of ADHD should incorporate a comprehensive assessment protocol involving various domains of functioning (Barkley, 1998; Hechtman, 2000; Tannock, 1998). In particular, the cognitive profile has hardly been examined in an early phase of ADHD. This could be extremely important to reveal whether certain deficiencies contribute to the behavioral pattern of ADHD. The present dissertation is devoted to the early diagnosis of ADHD from a cognitive point of view. In addition, this thesis deals with the relation between demographic factors and behavior problems being precursors of ADHD.

### Diagnostic criteria of ADHD

The conceptualization and diagnostic criteria for ADHD often have changed over the years, from ‘a defect of moral control’ (Still, 1902), ‘Minimal Brain Damage’ (Straus & Lehtinen, 1947), to ‘Hyperkinetic Reaction of Childhood Disorder’ (DSM-II; American Psychiatric Association, 1968), and finally, ‘Attention Deficit/Hyperactivity Disorder’ in the most recent edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994).

According to the DSM-IV, the primary symptoms in children with ADHD are inattention, hyperactivity, and impulsivity. Inattention refers to, for example, a failure to give close attention to details or making careless mistakes in schoolwork, difficulty in sustaining attention in tasks or play, or appearing as if the mind is elsewhere or not listening when spoken to directly. Hyperactivity results in behaviors, such as, fidgeting with hands or feet, squirming in one’s seat or not remaining seated
when expected to do so, appearing to be ‘on the go’ or often acting as is ‘driven by a motor’. Impulsivity manifests itself as impatience: blurring out answers before questions have been completed, difficulty awaiting one’s turn, or interrupting or intruding on others. At least some of the symptoms that cause impairment must be present before 7 years of age and in at least two different settings. Also, the disorder must cause impairment in social or academic functioning (American Psychiatric Association, 1994).

In addition to the DSM-IV criteria, the tenth edition of the International Classification of Diseases (ICD-10; World Health Organisation, 1992) uses the term ‘Hyperkinetic Syndrome’ to refer to the same cluster of behaviors. The ICD-10 uses similar but not identical diagnostic criteria. Children defined according to ICD criteria seem to be a more severely impaired subgroup of ADHD (Schachar, 1991). The abbreviated term ADHD as well as the diagnostic criteria of DSM-IV will be used throughout this thesis.

**Early diagnosis of ADHD**

In most cases, the disorder is first diagnosed during elementary school years, when school adjustment is compromised (American Psychiatric Association, 1994, p.82). However, the disorder, or at least symptoms of the disorder, arise early in development, with a mean age of onset of symptoms between 3 and 4 years (Barkley, Fischer, Edelbrock, & Smallish, 1990) and, according to the DSM-IV criteria, an upper limit for age-onset of 7 years. Although the diagnostic criteria and requirements for a diagnosis seem clear, it remains difficult to clinically establish a diagnosis of ADHD in young children. The behavior of young children is often much more variable than that of older children. They are often active, constantly on the go and into everything, and darting back and forth. Their characteristic behavior may include features that are similar to the symptoms of ADHD (American Psychiatric Association, 1994, p.81). It is difficult to state whether this is aberrant behavior and deviates from the normal variation in behavioral development (Campbell, 1995). Or, stated differently, it is difficult to judge whether certain behavior is appropriate for a certain developmental period. Furthermore, most young children have not been exposed to situations in which sustained or close attention is demanded, conditions which tend to elicit, for example, symptoms of inattention. Thus, these are not always observed in young children (American Psychiatric Association, 1994, p.81). Moreover, many overactive toddlers will not be diagnosed with ADHD at later age. Longitudinal studies have shown that only about 50% of children with severe behavioral problems at an age of 3-4 are diagnosed with ADHD in later childhood (Campbell & Ewing, 1990; Palfrey, Levine, Walker, & Sullivan, 1985). The severity and the duration of the symptoms (e.g. longer than 6 months) determine in which children the symptoms will have a chronic course in later childhood. Other investigators have demonstrated that not only the difficult behavior of the child but also the interaction with a stressed environment are determinants of the later emergence of ADHD (Sanson, Smart, Prior, & Oberklaid, 1993). Despite this knowledge, it remains difficult to predict in which children behavioral problems will persist and meet the diagnostic criteria for ADHD at a later date. For this reason, more longitudinal research in young children focusing on other domains of functioning, such as the cognitive domain, is needed.
Cognitive research in young ADHD children

The few studies that did focus on the cognitive functioning of young children with ADHD mainly used intelligence tests and pre-academic skills (DuPaul, McGoey, Eckert, & vanBrakle, 2001; McGee, Partridge, Williams, & Silva, 1991). They demonstrated an association between ADHD and reduced intellectual abilities, poorer pre-reading abilities and simple math concepts. Marakovitz and Campbell (1998) were the first to include some neurocognitive measures in their longitudinal study of hard-to-manage preschool boys. Their results showed that these measures hardly differed between the children later diagnosed with ADHD and control children. Two other studies investigated the neurocognitive performance of young children with ADHD in more detail (Byrne, DeWolfe, & Bawden, 1998; Mariani & Barkley, 1997). Contrary to the Marakovitz study, these studies demonstrated deficits in vigilance, fine motor control, and working memory in 4- and 5-year-old children with ADHD.

Cognitive research in young children at risk of ADHD is relevant for several reasons. First, cognitive tests may contribute to the accuracy of the early identification of children at risk of ADHD and provide information about what difficulties exist. The literature on school-age ADHD children reveals a variety of cognitive problems (see next paragraph), which may already be present at a young age. Second, this research yields information about the cognitive mechanisms that underlie the symptoms of ADHD. For example, impulsivity seems to consist of a cognitive and a behavioral component (Barkley, 1991; DuPaul, Anastopoulos, Shelton, Guevremont et al., 1992). DuPaul and colleagues have demonstrated that cognitive test scores often result in classifications that disagree with a diagnosis of ADHD formed on the basis of scores for behavior rating scales. The authors point out the necessity of objective cognitive assessment measures that are more ecologically and clinically valid. Hardly any cognitive assessment has been examined in young children. Third, in line with the first two reasons, cognitive research in young children can be useful to test the validity of a recent theoretical view of ADHD (Barkley, 1997, see later) in an early stage of the development.

Cognitive research in school-age ADHD children

Most cognitive research on ADHD has involved school-age children. Several studies have demonstrated that a number of cognitive problems are associated with ADHD. For example, there is strong evidence that children with ADHD have deficits in executive functions, especially an inhibitory control deficit (Barkley, 1997; Oosterlaan, Logan, & Sergeant, 1998; Pennington & Ozonoff, 1996; Schachar, Mota, Logan, Tannock, & Klim, 2000). A deficient inhibitory control can be described as a failure to withhold or suppress inappropriate responding. This would lead to an impaired ability to use control strategies in order to optimize behavior. Some investigators believe that the deficient inhibitory control can be explained by non-optimal motivation (Slusarek, Velling, Bunk, & Eggers, 2001) or by an aversion of delay (Sonuga-Barke, Williams, Hall, & Saxton, 1996). Other executive processes, such as organizing and planning behavior, goal-directed behavior, working memory, verbal fluency, or the ability to shift response sets during tasks, are also impaired in ADHD children (Grodzinsky & Barkley, 1999; Oosterlaan & Sergeant, 1998).
Studies of *attention and information processing* have yielded inconsistent results. De Sonneville and coworkers (De Sonneville, Njokiktjen, & Bos, 1994) found ADHD children to have attention deficits in several stages of the information processing chain, including encoding, memory search and decision, as well as focused and sustained attention deficits. In contrast, Van der Meere and coworkers (1989; 1991) found no differences in these stages of information processing and in focused and sustained attention between ADHD and control children. Yet, Van der Meere et al. did find that ADHD children showed impairments of the output side of the information processing chain, that is, in the motor decision process. This process links perception and action, and provides the ability to preset motor processes prior to the presentation of information (Sanders, 1983). This motor decision process was slower in children with ADHD, which has been interpreted as reflecting a poor control adjustment (Van der Meere et al., 1991). In a recent study this motor control was further examined (Börger & Van der Meere, 2000). They demonstrated that poor motor activation in ADHD children was associated with poor motor preparation (as measured by heart rate deceleration) and initiation (as reflected in a delayed cardiac shift) when stimuli were presented slowly. They concluded that ADHD is associated with poor effort allocation.

In addition, there is discussion about *automatic versus controlled information processing* in ADHD children (Ackerman, Anhalt, Dykman, & Holcomb, 1986; Ackerman, Anhalt, Holcomb, & Dykman, 1986). Automatic processes are executed rapidly and require little attention capacity whereas controlled processes demand more effort and attention capacity in order to be successful. These controlled processes are often sensitive to motivation and arousal. While no evidence was found for deficits in acquired automatic processing in ADHD children (Van der Meere & Sergeant, 1988), some studies have demonstrated deficits in controlled or effortful processing in ADHD children, revealed by comparing their performance on verbal and spatial memory tasks with that of controls (Borcherding et al., 1988; Ott & Lyman, 1993). However, Hazell et al. (1999) could not replicate this finding in visual information processing tasks.

**Theoretical model of ADHD**

All research findings on cognitive and behavioral deficits have been interpreted within the context of one theoretical model (Barkley, 1997). According to this model, behavioral disinhibition, as central impairment in ADHD, is linked to four executive neuropsychological functions. These functions, namely, working memory, self-regulation of affect-motivation-arousal, internalization of speech, and reconstitution, regulate the motor control of behavior. Working memory represents the capacity to hold information in mind in order to anticipate and prepare behavior in the future. Self-regulation of affect-motivation-arousal refers to the regulation of emotional and motivational states and may begin as early as 5-10 months. Internalization of speech is quiet or inhibited speech turned on the self that contributes to the self-guidance and rule governance of behavior. This stage follows the stage of public speech seen in preschool children and begins between 6-9 years of age. Reconstitution refers to the capacity to analyze internally represented information from the past and recombine it with new behavioral sequences for novel situations. Children with ADHD have deficiencies in each of these functions secondary to the behavioral disinhibition and these deficiencies lead to problems with the
motor control of behavior. Although the model is promising and the theory is widely held, more research is required to evaluate its validity.

Other risk factors in the development of ADHD
Beside the behavioral and cognitive risk factors that have been described above, a number of other factors have been related to ADHD. For example, pregnancy or perinatal complications have often been studied in relation to ADHD but the results are not univocal. A consistent finding is the relationship between maternal smoking and alcohol abuse during pregnancy and ADHD (Milberger, Biederman, Faraone, Wilens, & Chu, 1997). Furthermore, one of the most important risk factors for ADHD is a family history of ADHD. There is substantial evidence for the heritability of ADHD, implying a strong genetic component (Tannock, 1998). Yet, not all the variance in ADHD is due to genetic differences, which leaves an important role for environmental factors. Certain family-related issues and environmental factors have also been associated with ADHD (Hechtman, 1996). Severe marital discord, single parenthood, low socio-economic status (as evidenced by low-level parental occupation and low family income), large family size, paternal criminality, poor maternal health, and younger motherhood are related to ADHD (Biederman et al., 1995; Hartsough & Lambert, 1985). However, in a later study Faraone and Biederman (1998) pointed out that these measures of psychosocial adversity may reflect effects of the same genes that cause ADHD rather than independent causes of the disorder.

Methodological issues in studying ADHD
Several methodological issues are important when studying ADHD and the cognitive functioning of these children. First, there is an extremely high rate of comorbidity in ADHD (Biederman, Newcorn, & Sprich, 1991; Kadesjo & Gillberg, 2001). Kadesjo and Gillberg (2001) showed that 87% of ADHD children have one or more comorbid diagnoses. The overlap with oppositional defiant disorder (ODD) and conduct disorder (CD), the so-called Disruptive Behavior Disorders, is the greatest (35% to 60%). The small body of literature on the cognitive deficits of ADHD with and without ODD/CD comorbidity is inconsistent. Some studies have found that response inhibition deficits are similar in ADHD and comorbid ADHD+ODD/CD (Oosterlaan et al., 1998; Schachar & Tannock, 1995), while others have found inhibitory control to be significantly impaired in ADHD compared with comorbid ADHD+ODD/CD (Schachar et al., 2000).

Internalizing forms of psychopathology, such as mood and anxiety disorders (ANX), also co-occur with ADHD in approximately 30% of the cases. Manassis, Tannock, and Barbosa (2000) have demonstrated that comorbid ADHD+ANX are cognitively distinct from pure ADHD and pure anxiety disorders. Lastly, children with ADHD also have a significant risk of learning disorders (LD) and developmental coordination disorders (DCD; 40% for LD; 47% for DCD). Children with comorbid ADHD+LD show all the deficiencies found in children with pure ADHD or pure LD (Korkman & Pesonen, 1994). In contrast, Pennington, Groisser, and Welsh (1993) found that children with comorbid ADHD and a reading disorder (RD) had cognitive deficits similar to those seen in children with RD but dissimilar to those of ADHD children. With respect to DCD, Kadesjo and Gillberg
(1998) demonstrated that children with ADHD and DCD, a group called DAMP because of their Deficits in Attention, Motor control, and Perception, are more likely to be dysfunctional in the classroom than children with ADHD or DCD.

Second, there is a large fluctuation in the severity of symptoms across different situations (Barkley, 1998, p.73; Schachar, Rutter, & Smith, 1981). Some children show the symptoms of ADHD in all situations (pervasive hyperactivity) whereas others exhibit ADHD symptoms only at home or at school (situational hyperactivity) (American Psychiatric Association, 1987). Pervasive hyperactivity is associated with greater cognitive deficits than is situational hyperactivity (Rutter, 1989; Schachar, Tannock, Marriott, & Logan, 1995).

In line with this issue is the debate whether ADHD represents a pathological category or a dimension of behavior (Barkley, 1997). Perhaps children with pervasive hyperactivity are at the extreme end of a dimension of behavior, with normal children at the other end and children with situational hyperactivity somewhere in between. Yet, the DSM-IV does not permit such a dimension, but instead requires that the symptoms be demonstrated in at least two different settings before ADHD can be diagnosed. Nevertheless, genetic studies support the notion that ADHD represents a dimensional trait (Levy, Hay, McStephen, Wood, & Waldman, 1997) but until now there is no evidence whether or not qualitative differences exist between individuals. Thus, it is interesting to investigate whether the cognitive functions of certain groups of children with ADHD or ADHD-like problems fall along a continuum of normal functioning.

**Study of Attention Disorders in Maastricht (SAM)**

As stated earlier, it is difficult to predict in which children behavioral problems will persist and will meet diagnostic criteria for ADHD later. This requires longitudinal research with epidemiological and at-risk samples using a multimethod approach because ADHD is a multidimensional syndrome (Barkley, 1998, p71). Such studies in particular are uncommon (Lavigne et al., 1998).

We have started a large research program entitled ‘Study of Attention Disorders in Maastricht’ (SAM). This multidisciplinary research program is carried out jointly by the University of Maastricht (Department of Psychiatry and Neuropsychology), the University Hospital of Maastricht (Department of Neurology, Department of Psychiatry, and Department of Pediatrics), the Youth Health Care of the Municipal Health Center of Maastricht, and the Child Revalidation Center Franciscusoord.

SAM is a community study and has a prospective longitudinal design performed in four phases over a time span of 4 years. The sampling design of SAM and the instruments used are outlined in Table 1. The main goal of SAM is to examine the developmental profile and risk factors of ADHD in a stage in which most children are not yet clinically diagnosed. The study uses standardized as well as experimental questionnaires and tests.
Table 1. *Sampling design of and instruments used in SAM*

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<th>Phase</th>
<th>Months</th>
<th>Description</th>
<th>Instruments</th>
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| 1     | 1-9    | 1317 responders  
|       |        | 973 non-responders  
|       |        | CBCL  
|       |        | PAQ  
|       |        | Demographic information  
|       |        | Behavior observation list |
|       |        | 452 selected children |
| 2     | 4-13   | 443 first assessment of children  
|       |        | Neuropsychological assessment  
|       |        | Motor assessment  
|       |        | Developmental questionnaire  
|       |        | Behavior observation list |
| 3     | 15-25  | 403 follow-up assessment  
|       |        | ADIKA  
|       |        | AFS  
|       |        | TRF |
| 4     | 39-49  | 284 second follow-up assessment  
|       |        | Neuropsychological assessment  
|       |        | ADIKA |

Note. CBCL= Child Behavior Checklist; PAQ= Precursors of ADHD Questionnaire; ADIKA= Amsterdam Diagnostic Interview for Children and Adolescents; TRF= Teacher Report Form; AFS= Assessment of Family Stress

Children who attended second grade of normal kindergarten schools were recruited during a periodic systemic health examination performed by the Youth Health Care in the Dutch region of South Limburg. The *first phase* started in October 1996. The parents of the children who agreed to participate in the study completed the Child Behavior Checklist (CBCL; Verhulst, Koot, & van der Ende, 1996) as well as an experimental questionnaire for the early detection of children at risk of ADHD, the Precursors of ADHD Questionnaire (PAQ; Hendriksen & Steyaert, 1999). In addition, demographic information was collected anonymously from the medical records of the Youth Health Care and the school doctor filled in a short experimental behavior observation list. On the basis of the CBCL scores, children were selected for further assessment in *phase two*. This consisted of a neuropsychological examination and an assessment of the motor performance of the child. In both test situations, the examiners completed the behavior observation list. The teachers also filled in this observation list. In addition, an extensive developmental questionnaire including items related to pregnancy, neonatal period, developmental milestones, physical and psychiatric illnesses in the
family, and sociodemographic items was filled in partly by the parents and partly by the school doctor. In the *third phase*, 1.5 years later, detailed psychiatric information about the children was collected through a structured interview with the parents using the Amsterdam Diagnostic Interview for Children and Adolescents (ADIKA; Kortenbout van der Sluijs, Levita, Manen, & Defares, 1993). The parents completed an additional questionnaire containing items concerning the upbringing of their child and their need for help, the Assessment of Family Stress (AFS; Wels & Robbroeckx, 1996). The teachers of the children were asked to fill in the Teacher Report Form (TRF; Verhulst, van der Ende, & Koot, 1997). *Phase 4* took place 3 years after the start of the study and consisted of a follow-up neuropsychological assessment of the children as well as a follow-up ADIKA with the parents of the children. In order to enlarge the study population, a second cohort of children was included in October 1998. Basically, the same procedure was followed as for the first cohort.

**Outline and aims of the studies**

The studies in this thesis deal with the results of the first three phases of SAM and relate to the neurocognitive profiles of young children at risk of ADHD as well as demographic and behavioral variables. In the first three chapters (2, 3, and 4), the neurocognitive data of the children are presented.

Because ADHD is associated with various cognitive deficits at school age, a broad range of neurocognitive functions were assessed in young children in order to examine whether these deficits already manifest at young age (Chapter 2). The tasks were chosen to tap several aspects of executive functioning, such as shifting response sets, working memory, retrieval of semantic memory, and planning versus other cognitive functions such as perceptual and language functions. Successively, two different group designs were used. The performance on these tasks of children who were diagnosed with ADHD at follow-up was first compared with that of children with ‘borderline ADHD’ (children exhibit all ADHD symptoms but these do not lead to disruptions in at least two situations), and children without ADHD to examine whether ADHD represents a dimensional trait. Second, the task performance of the pure ADHD children was compared with that of children who were later diagnosed with pure ODD/CD, children with comorbid ADHD+ODD/CD, and control children. The small body of literature on executive deficits in these diagnostic groups is still inconsistent.

**Chapter 3** presents more detailed information on different aspects of attention and information processing assessed using a computerized test battery. The literature on this topic is still inconclusive for school-aged ADHD children and there is none for preschool and kindergarten children. This study evaluated whether attention dysfunctions are present in young children at risk of ADHD and whether they are specific for ADHD. The task performance of the children later diagnosed with ADHD was compared with that of children later diagnosed with other psychiatric disorders. All different diagnoses were included in this pathological control group because of the high rate of comorbidity in the ADHD group. The study also addressed the question whether ADHD represents a dimensional trait by comparing the results of ADHD, ‘borderline ADHD’, and healthy control children.

The study reported in **Chapter 4** examined the performance of children at risk of ADHD on two computerized complex motor tasks that differ in their cognitive processing requirements: one
requires relatively little attention capacity and thus, requires a low-level of controlled processing while the other requires a high-level of controlled processing involving continuous attention capacity and visual feedback. In addition, a simple test of movement speed was used to compare with the complex motor control tasks in order to determine whether specific deficits in the motor control underlie ADHD. The same diagnostic groups were used as in the study described in chapter 3, in order to specify the results and to examine the continuum hypothesis.

The demographic information of the study population and the behavioral data of the 5- to 6-year-old children gathered in the first phase of the study were critically appraised in Chapters 5 and 6. It is well known that behavioral problems at young age increase the risk of later psychopathology, especially ADHD. However, the etiology of early behavioral problems and the identification of the children in which these problems will persist are not yet clear. Therefore, several risk factors, including family and environmental risk factors, were studied in relation to behavioral problems to clarify their concerted action in the early development of children. In particular, Chapter 5 investigated (i) whether problem behavior in 5- to 6-year-old children is related to parental education and occupation, and (ii) if so, to what extent do correlated factors, such as family situation, family size, living area, country of birth, and maternal age at delivery, contribute to this association. A greater knowledge of the interaction of these factors may lead to more directed interventions.

Chapter 6 examined whether neighborhood-level socioeconomic variables have an independent effect on reported child behavioral problems over and above the effect of individual-level measures of socioeconomic status. If living in a deprived neighborhood has an independent effect on problem behavior not caused by, for example, low-level parental occupation or marital discord, this may have implications for intervention programs that should also focus on neighborhood characteristics. In this study, multi-level techniques were used to take into account the hierarchical nature of the data of individuals being clustered into neighborhoods.

Finally, in Chapter 7 the findings of the experimental studies in this thesis are drawn together. All neurocognitive measures were included in a principal component analysis to examine a model of interrelations between the measures and to determine whether certain constructs should be given extra attention in clinical practice when young children at risk of ADHD are subjected to neurocognitive examination. Concluding remarks as well as implications for further research are presented.
References


Neurocognitive performance in 5- and 6-year-old children with attention deficit/hyperactivity disorder: Results from a prospective population study

Abstract
The aim of this prospective study was to examine the neurocognitive performance of 5/6-year-old children at risk of ADHD after adjustment for behavioral measures. Out of a general population of 1317 children, 366 children were selected on the basis of their scores on the Child Behavior Checklist (CBCL). Eighteen months later, the parents were interviewed using a standardized child psychiatric interview. Successively, two different group designs were used. First, children later diagnosed with ADHD (N=33) were compared with 'borderline ADHD' children (N=75) and 'no ADHD' children (N=258) to examine whether ADHD represents a dimensional trait. Second, children with pure ADHD (N=9) were compared with children with comorbid ADHD and Oppositional Defiant Disorder/Conduct Disorder (ADHD+ODD/CD; N=24), children with pure ODD/CD (N=59), and control children (N=274) to specify the results. Children with ADHD were significantly impaired on measures of visuomotor ability and working memory compared to 'no ADHD' children after adjustment for CBCL results. The performance of 'borderline ADHD' children was in between that of ADHD and 'no ADHD' children. Furthermore, the comorbid ADHD+ODD/CD children performed significantly worse on these tasks compared with the pure ODD/CD and control children while they did not differ from the pure ADHD children. Our results imply that neurocognitive measures can contribute to the early identification of ADHD with and without comorbid ODD/CD.
Attention Deficit/Hyperactivity Disorder (ADHD) is characterized by the core behavioral symptoms of inattention, hyperactivity, and impulsivity (American Psychiatric Association, 1994). It is the most common and the most extensively studied child psychiatric syndrome (Tannock, 1998). Although ADHD symptoms start before the age of 7 (American Psychiatric Association, 1994), the vast majority of research is based on school-aged children between 7 and 12 years of age (Biederman et al., 1998). A major reason for the paucity of research on younger children with symptoms of ADHD is the difficulty in determining whether their behavior deviates from the normal variation in behavioral development (Campbell, 1995). Furthermore, children younger than 7 may not show all the symptoms of ADHD (Weiss & Hechtman, 1993) which, according to Rappaport and coworkers, might cause a delay in the formal diagnosis (Rappaport, Ornoy & Tenenbaum, 1998). Yet, it has been demonstrated that behavior problems at a young age can persist and even meet criteria for ADHD at a later age (Campbell & Ewing, 1990; McGee, Partridge, Williams & Silva, 1991; Pierce, Ewing & Campbell, 1999). It is presently unclear in which children these problems persist, and whether these children also have other characteristics, such as neurocognitive dysfunctions, which predict a diagnosis of ADHD at a later age. There is a need for prospective studies in order to evaluate these questions.

The small body of research available on cognitive performance in children younger than 7 years with symptoms of ADHD has shown that ADHD is associated with reduced intellectual abilities (DuPaul, McGoey, Eckert, & VanBrakle, 2001; McGee, et al., 1985). Only two studies have carried out a more detailed investigation of the neurocognitive performance of young children (Byrne, DeWolfe & Bawden, 1998; Mariani & Barkley, 1997). These studies demonstrated deficits in vigilance, motor control, and working memory in 4- and 5-year-old children with ADHD. However, both cross-sectional studies used small samples and involved a highly selected sample of children who had been referred to a clinic. More information concerning neurocognitive performance is available on children older than 7 years. For example, there is strong evidence that children with ADHD have deficits in attention and executive functions, especially involving inhibition control (Barkley, 1997; Oosterlaan, Logan & Sergeant, 1998; Pennington & Ozonoff, 1996; Quay, 1997; Schacher, Mota, Logan, Tannock & Klim, 2000). Furthermore, deficient motivation (Sonuga-Barke, Williams, Hall & Saxton, 1996) and difficulties with serial information processing (Van der Meere, Van Baal & Sergeant, 1989) have been described. It is of interest to note that this pattern of deficits is similar to that found in adults with frontal lobe damage (Grodzinsky & Barkley, 1999; Seidman, Biederman, Faroane, Weber & Ouellette, 1997). Indeed, recent theories point to a primary involvement of prefrontal mechanisms in the development of ADHD, especially those mechanisms involving executive functioning, such as inhibition and planning (eg. Barkley, 1998, p 87, p 125).

Thus, there are strong indications that particular neurocognitive deficiencies are already present in young children, but it is not yet clear whether children characterized by impaired performance have a greater risk of being diagnosed with ADHD at a later age. The aim of the present prospective study was therefore to investigate the performance on a neurocognitive test battery at 5-6 years of age of
children who were diagnosed with ADHD at a later age and to investigate the neurocognitive performance after taking into account measures of behavior. The study was performed according to a controlled design and involved a group of children who were drawn from a large community sample. Neurocognitive tasks were chosen in order to tap several aspects of executive functioning (such as shifting response sets, working memory, retrieval from semantic memory), and visuomotor planning versus other cognitive functions (such as perceptual functions and language) as control measures. In studying a population at risk of ADHD, it is of major importance to take into account the heterogeneity in the pervasiveness of ADHD symptomatology (Schacher, Tannock, Marriot & Logan, 1995) as well as the high rate of comorbidity in ADHD (Biederman, Newcorn & Sprich, 1991).

Therefore, the objective of the present study was twofold.

The first objective of our study was to examine whether neurocognitive performance at 5-6 years of age distinguishes children who were later diagnosed with ADHD or 'borderline ADHD' from 'no ADHD' children. The 'borderline ADHD' group consisted of children who exhibited ADHD symptoms in fewer than two situations. Our interest in this borderline group is among others based upon research by Schacher and colleagues (1995), who showed that neurocognitive deficits were greater in school-age children with pervasive ADHD who met all the criteria for ADHD than in school-age children with situational ADHD who met the criteria for ADHD in only one situation. Whether these deficits are already evident at young age is not clear. In line with this issue is the debate whether ADHD represents a dimensional trait (Barkley, 1998, p.73) expressed in qualitative differences between individuals with ADHD on one extreme, followed by ‘borderline ADHD’ and the ‘no ADHD’ children on the other end.

The second objective was to evaluate the possible influence of comorbid psychopathology. The high rate of comorbidity in ADHD and other externalizing disorders is of interest when studying the neurocognitive performance of young children in order to delineate the etiology of ADHD (Schacher & Tannock, 1995; Schacher et al. 2000). Therefore, we compared the neurocognitive performance at 5-6 years of age of children who were later diagnosed with pure ADHD, pure Oppositional Defiant Disorder and/or Conduct Disorder (ODD/CD), or comorbid ADHD+ODD/CD with that of control children.

METHOD

This study is based on data collected as part of a large research program entitled SAM (‘Study of Attention disorders in Maastricht’), involving a longitudinal design with 5- to 6-year-old children in the south of the Netherlands (see Kalff, Kroes, Vles, Bosma et al., 2001, Kalff, Kroes, Vles, Hendriksen et al., 2001; Kroes et al., 2001). The study was approved by the local Medical Ethics Committee and informed consent was obtained from the participating children's parents. The study was performed in three phases, as follows:
Subjects and Procedure

Phase 1: The first stage of the study involved all children in the second grade of a normal kindergarten class who visited the Youth Health Care (YHC) for a periodic health examination. In the Netherlands, this school year precedes the first class of elementary school in which the children learn to read and write. Parents were asked to give their permission for participation of their children in the study. The initial sample comprised 2,290 children. The parents of 1,317 children (57.5%; 699 boys and 618 girls) agreed to participation. Mean age of the children was 5.87 years (SD=0.41). Subsequently, parents were asked to complete the Dutch version of the Child Behavior Checklist (CBCL; Achenbach, 1991; Verhulst, Koot & van der Ende, 1996). Responders and non-responders were compared by randomly sampling 200 children of each group for child, family, and environmental factors. Information concerning the non-responders was obtained anonymously from the medical records of the YHC, which is allowed by law for epidemiological research purposes. Child factors consisted of sex and age of the child, as well as pregnancy-related problems, psychosocial and physical findings reported by the school doctor of the YHC. Family factors were parental education and occupation, family status and size, and country of the birth of the parents. Environmental factors were the living area and the neighborhood. No significant differences were found between the groups with regard to sex, age, and demographic factors. The only significant difference found was a higher substance abuse during pregnancy in the non-responders group (Kalff, Kroes, Vles, Bosma et al., 2001).

On the basis of their scores on the CBCL, 452 children were selected and the parents of 443 of these children (98%) gave permission for further participation of their children. Three groups of children were formed. Group E (‘CBCL Externalizing group’; N=167) consisted of children with scores above the clinical cut-off on the CBCL 'Externalizing scale' (above the 90th percentile) and/or scores above the borderline cut-off on the subscale 'Attention Problem' (above the 95th percentile). This group contained children with a putative risk for the diagnosis of ADHD (Chen, Faraone, Biederman & Tsuang, 1994). Group I (‘CBCL Internalizing group’; N=56) consisted of children with scores above the clinical cut-off on the CBCL 'Internalizing scale' (above the 90th percentile) and who were not members of group E. Thus, children with high scores on internalizing and externalizing scales were assigned to group E. Group I was included to contain children with a risk of other pathology. Group C (CBCL Control group; N=220) consisted of children without any behavioral problems as assessed by the CBCL; their 'Total problem' scores were below the 90th percentile. The children in group C were matched for age (± 2 months), sex, and school with the children in the other two groups.

Phase 2: The second stage started four months after phase 1. The three groups together consisted of 443 children (252 boys and 191 girls). They completed a battery of neurocognitive tasks in a separate room at their school. Their mean age at the time of assessment was 6.19 years (sd 0.45, range 5.38-7.78). Forty-three children could not be assessed for logistic reasons, for example, because there was not enough school time or space available to assess the children. The tests took about 1.5 hours to complete and were administered by one of ten well-trained psychologists who were blind to the group assignments.
Phase 3: In the third stage of the study, 18 months after the first stage, the parents of the selected children were interviewed using the Dutch version of the Diagnostic Interview for Children and Adolescents (DICA; Herjanic & Reich, 1982; ADIKA; Kortenbout van der Sluijs, Levita, Manen & Defares, 1993) by one of three interviewers. These interviewers were trained and supervised by a senior child psychiatrist and were blind to group membership. Nine percent of the parents of the children (N=40) refused to participate in the third stage or had moved prior to this stage, leaving 403 parents (91%) who finally participated. In this stage, children were classified as ADHD if they had at least 6 of the 10 Inattentive symptoms or 11 Hyperactive/Impulsive symptoms of the ADIKA questionnaire, which is highly comparable with the DSM-IV criteria, or both. These symptoms had to be present in at least two situations. Also, they had to have persisted for at least 6 months and to have started before the age of 7. Children who had ADHD symptoms in fewer than two situations were assigned to the 'borderline ADHD' group. All other children not meeting criteria for ADHD or 'borderline ADHD' were classified as a 'no ADHD' group (regardless of any other diagnosis assigned). For our second research question concerning comorbidity, children were classified as ODD or CD according to the DSM-III-R criteria (American Psychiatric Association, 1987) (age of onset, severity, and type of symptom) and had at least 5 of the 11 symptoms of ODD or at least 3 of the 13 symptoms of CD of the ADIKA questionnaire. The diagnoses were combined into one variable (ODD/CD) for the analyses. The other children not meeting criteria for ADHD or ODD/CD were considered as the control group. The performance of the children in the four groups (pure ADHD, ADHD+ODD/CD, pure ODD/CD, control) were compared.

For the present study, neurocognitive and ADIKA data were complete for 366 children (208 boys, 158 girls). Their mean age was 6.19 years (SD 0.45). In the third stage, 33 children were classified as ADHD, 75 children as 'borderline ADHD', and 258 children as 'no ADHD'. The ADHD group consisted of 19 children with the combined ADHD type, seven children with the predominantly inattentive type, and seven children with the predominantly hyperactive-impulsive type (DSM IV; American Psychiatric Association, 1994). The 'borderline ADHD' group consisted of 20 children with the combined ADHD type, nine children with the predominantly inattentive type, and 46 children with the predominantly hyperactive-impulsive type. Thirty percent of these children only had problems at home, 24% only had problems at school, and 46% did not have problems in any situation. For the second analyses, 9 of the 33 ADHD children were classified as pure ADHD, 24 as comorbid ADHD+ODD/CD, 59 as pure ODD/CD, and 274 children were regarded as controls. There was no significant overlap between the children with different subtypes of ADHD and the children with comorbidity. None of the children included at the beginning of the study used medication, except for two children during the third phase of the study (one child used Ritalin and the other child used Dipiperon). At the end of the third phase, the test results of the children with a psychiatric diagnosis according to the ADIKA were given to the school doctor of the YHC, who then decided whether the children needed a treatment program.
Measures

The Child Behavior Checklist (CBCL; Achenbach, 1991; Verhulst et al., 1996) is a parent-reported measure of child behavior problems. This questionnaire yields T-scores for a Total problem scale and for the two broadband scales (Externalizing and Internalizing behavior). It also yields T-scores for several subscales, one of which (Attention subscale), was used in the present study.

The Level of Occupational Achievement (LOA) of the parents was scored on a 7-point scale (Van den Brand et al., 1990), ranging from unskilled to professional. For children living with both parents, the highest level was used; for the remaining children, the level of the parent with whom the child lived was used.

The Vocabulary subtest from the Revised Amsterdam Child Intelligence Test (RAKIT; Bleichrodt, Drenth, Zaal & Resing, 1987) was used to estimate intellectual functioning. The RAKIT is a Dutch intelligence test for children aged 4 to 11 years. The Vocabulary subtest measures the verbal ability of the child and is similar to the well-known Peabody Picture Vocabulary Test, which is considered to give a valid approximation of IQ (Marakovitz & Campbell, 1998). The child must choose one out of four pictures that matches the meaning of the word read aloud. The raw score (range 1-60) is the number of correct answers. This score is transformed into an IQ score (mean=100, sd=15).

The Embedded Figures subtest of the RAKIT (Bleichrodt et al., 1987) measures the perceptual ability of the child. The child is instructed to look carefully at six complex drawings with many abstract lines and to choose the one in which the complete concrete figure is shown that matches exactly an example figure. Again, the number of correct answers becomes the raw score (range 1-45) and is transformed into a scaled score (mean=15, sd=5, range 1-30) according to Dutch normative data.

The Gestalt Closure subtest of the Kaufman Assessment Battery for Children aged 2.5 to 12.5 years (K-ABC; Kaufman & Kaufman, 1983) was used to evaluate perceptual closure capacity. The child must recognize a picture in an incomplete drawing. The number of correct answers is the raw score (range 1-25). This score is transformed into a scaled score (mean=10, sd=3, range 1-19) according to German normative data because Dutch data are not available.

The Number Recall subtest and the Word Order subtest of the K-ABC (Kaufman & Kaufman, 1983) were used to assess short-term auditory working memory. In the first test, the child must recall digits in a sequence given verbally by the psychologist. The number of digits increases every two trials (range of raw scores 1-19). The Word Order subtest is a more complex auditory-nonverbal task because of an extra mental task. The child must point out the correct pictures that have been named in the correct order instead of just repeating digits (range of raw scores 1-20). This subtest can also be seen as a measure of executive function.

The Progressive Figures Test (Reitan & Wolfson, 1985) is a test for children aged 4 and 5 years and was originally used in the Halstead Reitan battery. It measures the ability to shift constantly between small and large figures by connecting a line between them. This test is used to assess the executive functions of mental control and shift response sets. The time to finish the line is transformed into a T-score (mean=50, sd=10, range 20-70) according to Dutch normative data.
The Verbal Fluency subtest of the RAKIT (Bleichrodt et al., 1987) was used in order to assess retrieval from semantic memory and verbal organization. The child must generate as many words as possible from five semantical categories, each within one minute (e.g. ‘What can you drink?’). The raw score is the number of words correctly generated in response to the five questions and is transformed into a scaled score (mean=15, sd=5, range 1-30).

The Developmental Test of Visual-Motor Integration (VMI Beery; Beery, 1997) can be used for children aged 2 to 16 years and measures the integration of visual perceptual and motor (finger and hand movement) abilities. The child must copy geometric forms that become progressively more difficult and which are presented simultaneously with three forms on one page. The number of correctly drawn forms is the raw score (range 1-24) and is transformed into a scaled score (mean=10, sd=3, range 1-19).

The Diagnostic Interview for Children and Adolescents (DICA; Herjanic & Reich, 1982; ADIKA; Kortenbout van der Sluijs et al., 1993) is a semi-structured interview and yields scores on several child psychiatric disorders, according to DSM-III-R guidelines (American Psychiatric Association, 1987). The DICA has been described recently as a useful measure for both research and clinical settings to provide reliable psychiatric information about children (Reich, 2000). The ADIKA was adapted, using the criteria of DSM IV, to make it suitable for ADHD (Westereich, 1998).

Statistical analysis
Differences in sex and CBCL group assignment between ADHD, 'borderline ADHD' and 'no ADHD' groups were tested using Chi-square tests. Differences in age, parental occupation, estimated IQ, and CBCL T-scores between the three groups were tested using one-way analysis of variance with posthoc Tukey tests for Honestly Significant Differences (HSD). CBCL T-scores are reported here solely to permit others to compare these results with past studies. CBCL group assignment was used in the further analyses (see below).

To test for internal relationship between neurocognitive measures associations were calculated for each group separately by using Pearson correlation coefficients. To determine the overall effect of neurocognitive performance at 5 and 6 years of age on the subsequent diagnosis of ADHD, a multivariate analysis of variance (MANOVA) in a between-group design (ADHD, 'borderline ADHD', 'no ADHD') was conducted. Multivariate analysis of covariance was conducted to assess the effect of sex, age, parental occupation, and estimated IQ on the relationship between ADHD and neurocognitive performance. Subsequently, the CBCL group assignment was added as a covariate to assess whether this relationship remained significant. When the overall omnibus F test was significant, one-way analyses of covariance (ANCOVA) were conducted with sex, age, parental occupation, estimated IQ, and CBCL group assignment as covariates. The Levene test was used to evaluate the homogeneity of the variance in neurocognitive performance because of the large differences in the number of children in the three groups (De Heus, Van der Leeden & Gazedam, 1995). To analyze group differences posthoc Tukey-HSD tests were conducted. Similarly, differences in neurocognitive performance of children with ADHD with and without comorbid ODD/CD, children with pure ODD/CD, and control children were analyzed to test the specificity of the results. Two-
tailed probabilities of 5% or less were considered significant. All statistical analyses were performed by using the SPSS statistical package for Macintosh (version 6.1).

RESULTS

Group characteristics for the classifications assigned in the third stage are shown in Table 1. There were significant differences in sex, parental occupation, and estimated IQ between the three groups. The CBCL groups differed significantly between the three diagnostic groups in the third stage ($\chi^2 = 52.08$, p<0.00) in that almost all incident cases of ADHD at follow-up came from the Externalizing group (91%), compared to 24% in the 'no ADHD' group. All CBCL T-scores were significantly higher in the ADHD children than in the children of the other two groups. The 'borderline ADHD' children had significantly higher scores than the 'no ADHD' children for all CBCL scales, except the scale for Internalizing behavior.

Table 1. Description of diagnostic groups and group comparisons on behavioral measures

<table>
<thead>
<tr>
<th></th>
<th>(1) ADHD (N=33)</th>
<th>(2)'borderline ADHD' (N=75)</th>
<th>(3)'no ADHD' (N=258)</th>
<th>F</th>
<th>Tukey$^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (m / f)</td>
<td>24 / 9</td>
<td>50 / 25</td>
<td>134 / 124</td>
<td>8.87*</td>
<td>-</td>
</tr>
<tr>
<td>Age (in years)</td>
<td>6.18 (0.32)</td>
<td>6.25 (0.55)</td>
<td>6.17 (0.43)</td>
<td>0.76</td>
<td>-</td>
</tr>
<tr>
<td>LOA</td>
<td>3.53 (2.02)</td>
<td>3.81 (1.82)</td>
<td>4.31 (1.84)</td>
<td>3.91*</td>
<td>1,2&lt;3</td>
</tr>
<tr>
<td>Estimated IQ</td>
<td>90.33 (19.13)</td>
<td>88.04 (21.56)</td>
<td>96.30 (23.07)</td>
<td>4.43*</td>
<td>2&lt;3</td>
</tr>
<tr>
<td>CBCL group (E/I/C)</td>
<td>30 / 1 / 2</td>
<td>47 / 7 / 21</td>
<td>61 / 40 / 157</td>
<td>52.08**</td>
<td></td>
</tr>
</tbody>
</table>

**CBCL T-scores**

<p>| | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total problem</td>
<td>68.55 (6.57)</td>
<td>60.88 (10.05)</td>
<td>52.73 (11.81)</td>
<td>39.49**</td>
<td>1&gt;2&gt;3</td>
</tr>
<tr>
<td>Externalizing scale</td>
<td>69.15 (7.00)</td>
<td>61.92 (11.49)</td>
<td>52.69 (11.39)</td>
<td>45.34**</td>
<td>1&gt;2&gt;3</td>
</tr>
<tr>
<td>Internalizing scale</td>
<td>62.09 (10.53)</td>
<td>55.99 (10.43)</td>
<td>53.28 (11.13)</td>
<td>10.21**</td>
<td>1&gt;2,3</td>
</tr>
<tr>
<td>Attention subscale</td>
<td>65.39 (8.72)</td>
<td>59.52 (7.63)</td>
<td>54.31 (6.43)</td>
<td>47.35**</td>
<td>1&gt;2&gt;3</td>
</tr>
</tbody>
</table>

Note. LOA=level of occupational achievement of parents scored on 7-point-scale; CBCL=Child Behavior Checklist; E=Externalizing group; I=Internalizing group; C=Control group; $^a$ Post hoc Tukey's HSD, p<.05; $^b$ Chi-square test; $^c$ Chi-square test with Group E and I together because of too small minimum expected frequency; * p<.05, **p<.01

The investigation of internal relationship between neurocognitive measures yielded only one highly significant correlation in all groups between the Word Order and Number Recall subtest scores (R=0.67, p=.00; R=0.66, p=.00, and R=0.69, p=.00 for ADHD, 'borderline ADHD', and 'no ADHD'
group, respectively). All other task scores were moderately correlated and the correlations ranged from 0.01 to 0.40 in all groups.

The MANOVA of neurocognitive performance at age 5-6 revealed a significant main effect of group assignment at later age (F(14,708)=2.41, p=.003, Pillais' test). After adjustment for sex, age, parental occupation, and estimated IQ, the main effect remained significant (F(14,666)=1.71, p=.050, Pillais' test). The addition of the CBCL group assignment as a covariate did not reduce the effect any further (F(14,664)=1.71, p=.050, Pillais' test).

### Table 2. Means (standard deviations) and F-statistics of neurocognitive performance at 5-6 years of age for the ADHD, 'borderline ADHD', and 'no ADHD' children

<table>
<thead>
<tr>
<th></th>
<th>(1) ADHD (N=33)</th>
<th>(2) 'borderline ADHD' (N=75)</th>
<th>(3) 'no ADHD' (N=258)</th>
<th>F</th>
<th>Tukeya</th>
</tr>
</thead>
<tbody>
<tr>
<td>Embedded Figures</td>
<td>13.70 (4.57)</td>
<td>13.75 (5.30)</td>
<td>16.00 (5.18)</td>
<td>3.25*</td>
<td>1.2&lt;3</td>
</tr>
<tr>
<td>Gestalt Closure</td>
<td>10.09 (3.37)</td>
<td>9.89 (2.84)</td>
<td>10.07 (3.02)</td>
<td>0.21</td>
<td>-</td>
</tr>
<tr>
<td>Number Recall</td>
<td>8.42 (3.15)</td>
<td>9.29 (2.65)</td>
<td>9.81 (2.56)</td>
<td>1.48</td>
<td>-</td>
</tr>
<tr>
<td>Word Order</td>
<td>7.58 (2.00)</td>
<td>8.59 (2.72)</td>
<td>9.18 (2.62)</td>
<td>3.83*</td>
<td>1&lt;3</td>
</tr>
<tr>
<td>Progressive Figures</td>
<td>49.56 (14.45)</td>
<td>53.49 (12.32)</td>
<td>56.37 (9.59)</td>
<td>3.90*</td>
<td>n.t.</td>
</tr>
<tr>
<td>Verbal Fluency</td>
<td>14.12 (4.85)</td>
<td>13.80 (4.66)</td>
<td>14.05 (5.09)</td>
<td>0.57</td>
<td>-</td>
</tr>
<tr>
<td>VMI Beery</td>
<td>9.45 (2.03)</td>
<td>10.49 (2.21)</td>
<td>11.01 (2.47)</td>
<td>5.10**</td>
<td>1&lt;3</td>
</tr>
</tbody>
</table>

Note. VMI=Visual Motor Integration.; n.t.=no longer tested because of too large differences in variance; a Post hoc Tukey's HSD, p<.05; *p<.05; **p<.01

The ANCOVAs revealed significant group differences between the performance on four neurocognitive tests, even after controlling for CBCL measures (see Table 2). Differences in group variances of scores for the Progressive Figures test appeared to be too large according to the Levene test (F(2,361)=7.91, p<.001). Therefore, scores for this test were excluded from further analyses. Post-hoc comparisons indicated that the ADHD children performed significantly worse than the 'no ADHD' children on the Embedded Figures subtest, the Word Order subtest, and the VMI Beery. The children in the 'borderline ADHD' group performed significantly worse than the 'no ADHD' children on the Embedded Figures subtest. Although no significant differences were found between the ADHD and the 'borderline ADHD' children, the performance of the 'borderline ADHD' children was generally in between that of the ADHD children and 'no ADHD' children.
Table 3. Means (standard deviations) and F-statistics of neurocognitive performance at 5-6 years of age for the pure ADHD, ADHD+ODD/CD, pure ODD/CD, and control children

<table>
<thead>
<tr>
<th></th>
<th>(1) ADHD (N=9)</th>
<th>(2) ADHD+ODD/CD (N=24)</th>
<th>(3) ODD/CD (N=59)</th>
<th>(4) controls (N=274)</th>
<th>F</th>
<th>Tukey*a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Embedded Figures</td>
<td>13.44 (3.71)</td>
<td>13.79 (4.92)</td>
<td>14.24 (5.49)</td>
<td>15.76 (5.21)</td>
<td>1.00</td>
<td>-</td>
</tr>
<tr>
<td>Gestalt Closure</td>
<td>10.89 (2.52)</td>
<td>9.88 (3.77)</td>
<td>9.98 (2.84)</td>
<td>10.05 (2.90)</td>
<td>0.77</td>
<td>-</td>
</tr>
<tr>
<td>Number Recall</td>
<td>7.89 (2.37)</td>
<td>7.71 (3.16)</td>
<td>9.31 (2.15)</td>
<td>8.80 (2.50)</td>
<td>3.77**</td>
<td>2&lt;3</td>
</tr>
<tr>
<td>Word Order</td>
<td>6.00 (1.94)</td>
<td>6.96 (2.29)</td>
<td>8.51 (3.37)</td>
<td>8.35 (3.11)</td>
<td>3.36*</td>
<td>1,2&lt;3,4</td>
</tr>
<tr>
<td>Progressive Figures</td>
<td>51.89 (15.73)</td>
<td>48.65 (14.18)</td>
<td>53.64 (12.10)</td>
<td>56.16 (9.86)</td>
<td>2.85*</td>
<td>n.t.</td>
</tr>
<tr>
<td>Verbal Fluency</td>
<td>13.44 (3.78)</td>
<td>14.38 (5.25)</td>
<td>14.44 (4.85)</td>
<td>13.90 (5.02)</td>
<td>0.73</td>
<td>-</td>
</tr>
<tr>
<td>VMI Beery</td>
<td>10.10 (1.90)</td>
<td>9.21 (2.06)</td>
<td>10.71 (2.35)</td>
<td>10.93 (2.43)</td>
<td>4.32*</td>
<td>2&lt;3,4</td>
</tr>
</tbody>
</table>

Note. VMI=Visual Motor Integration.; n.t.=no longer tested because of too large differences in variance; * Post hoc Tukey's HSD, p<.05; **p<.01

Table 3 shows the neurocognitive performance of ADHD children with and without comorbid ODD/CD, pure ODD/CD children, and control children. The groups differed significantly in performance on the four tests after controlling for CBCL measures (see Table 3) and, again, differences in group variances of scores for the Progressive Figures test were too large (F (3,360)=4.07, p=.007). Posthoc comparisons indicated that the comorbid ADHD+ODD/CD children performed significantly worse than the pure ODD/CD children on the Number Recall subtest, the Word Order subtest, and the VMI Beery. The comorbid ADHD+ODD/CD children performed significantly worse than the control children on the Word Order subtest and the VMI Beery. The children in the pure ADHD group had a significantly worse performance on the Word Order subtest than the pure ODD/CD children and the control children. No significant differences were found between the pure ADHD children and comorbid ADHD+ODD/CD children or between the pure ODD/CD children and the control children.

DISCUSSION

The first objective of the present study was to examine whether neurocognitive performance of children aged 5-6 years distinguishes children who were diagnosed with ADHD or 'borderline ADHD' at a later age from 'no ADHD' children. The prospective longitudinal study involved a group of children drawn from the general population who were or were not at risk of ADHD. The most salient finding was that children diagnosed with ADHD 18 months later and 'no ADHD' children differed in performance on a number of neurocognitive tasks (VMI Beery, Word Order, and
Embedded Figures), even when the CBCL behavior scales were taken into account. It is known that high externalizing scores on the CBCL form a risk for child psychiatric disorders (Lavigne et al., 1996). Also, follow-up studies of hard-to-manage boys at risk of ADHD have shown that observational measures of activity and some earlier measures of impulsivity are predictive of a later ADHD (Marakovitz & Campbell, 1998). Yet, the latter authors state that behavioral measures seem to be more sensitive to group differences than cognitive measures because only a few group differences were evident on cognitive measures. Thus, these findings do not seem sufficient for the diagnosis of ADHD and additional indicators are needed. Our results show that neurocognitive measures are such an indicator and could be of value in strengthening the early identification of ADHD.

An interesting additional finding of our study was that the neurocognitive performance of the 'borderline ADHD' children was generally in between that of the ADHD children and the 'no ADHD' children, which is in line with the view that ADHD is a continuum or a dimension, and not an entity with strict boundaries (Barkley, 1998, p.73; Mercugliano, 1999; Levy et al., 1997). The finding that the 'borderline ADHD' children performed better than the ADHD children on a few tasks is interesting because both groups were equally symptomatic. Apparently, the poor neurocognitive performance was not due to the symptoms of ADHD only. This is in line with Schacher et al. (1995), who reported that it is possible to exhibit the symptoms of ADHD without there being a deficit in inhibitory control. Yet, we found no significant differences in performance of neurocognitive tasks between children with ADHD and children with 'borderline ADHD'. Thus, further research with other factors is needed to evaluate whether children with ADHD can be distinguished from children with all the symptoms of ADHD but who do not fulfill all criteria for the diagnosis.

The nature of the three neurocognitive tasks showing significant differences in the performance of children with ADHD and 'no ADHD' children is consistent with past research on school-age children with ADHD. First, the VMI Beery test is often considered as a task for assessing motor maturation and it measures the integration of visual perceptual and motor (finger and hand) abilities (Beery, 1997). Interestingly, performance on the perceptual Gestalt Closure task did not differentiate between the three groups, indicating that perceptual abilities are intact in these young children. This is in agreement with the findings of Van der Meere and coworkers that the input side of the information-processing chain in ADHD is intact (Van der Meere et al., 1989). The deficient performance on the VMI Beery can therefore be taken to indicate that the underlying problem in young children at risk of being diagnosed with ADHD at a later age may involve the cross-modal transfer of information, particularly at the level of motor control output or psycho-motor planning. Second, the Word Order subtest involves not only working memory but also the translation of information recalled from memory into a motor plan in order to obtain the correct answer. Our results suggest there is not a pure working memory deficit in young children, as the results on the basals Number Recall subtest showed, but rather a deficit in an extra control process. This is in line with studies reporting deficits in executive functioning in school-age ADHD children (Oosterlaan et al., 1998; Pennington & Ozonoff, 1996). Our results show that these deficits may very well already be present in young children aged 5-6 years. Third, the impaired performance on the Embedded Figures
subtest is in accordance with other studies reporting lower measures of performance intelligence in children with ADHD (Rispens et al., 1997).

The diminished performance with respect to motor planning and executive functioning found in ADHD children suggests that prefrontal regions are possibly involved in the development of ADHD. Recently, Grodzinsky & Barkley (1999) concluded that differences between children with ADHD and normal controls may only emerge when the relevant substrates in the frontal region have reached maturity. Our results suggest that the maturation of prefrontal structures in children at risk of a diagnosis of ADHD at a later age lags behind that of children of the same age. In this respect, certain frontal lobe functions are thought to reach maturity at preschool age (Dowsett & Livesey, 2000). Further research into this possibility should be undertaken because subtle differences in brain maturation among children could indeed explain the fact that children with ADHD are dysmature in their behavioral and cognitive functioning.

The second objective of this study was to examine whether the deficits in children with ADHD are also influenced by comorbid psychopathology. To this end, we compared the neurocognitive performance between ADHD children with and without comorbid ODD/CD, children with pure ODD/CD, and control children. Interestingly, the children in the comorbid ADHD+ODD/CD group were found to have a significantly impaired performance on tasks measuring working memory and visuomotor ability compared to the children with pure ODD/CD and control children, while their performance did not differ from that of the children in the ADHD group with ODD/CD comorbidity. The children in the pure ADHD group differed from the children with pure ODD/CD and control children in their performance of a task measuring working memory. This finding must be regarded as ‘tentative’ in view of the small number of children in this ADHD group. Thus, our results seem to be specific for ADHD children with comorbid disruptive behaviors, because neurocognitive deficits were not evident in children with pure ODD/CD. This is in agreement with the results of Schacher & Tannock (1995). Moreover, our results showed that the neurocognitive deficits associated with ADHD, but not those associated with ODD/CD, seem to be present already at age 5-6.

There are several potential drawbacks to our study. Because we selected children on the basis of their CBCL scores, our results do not reflect the ‘true’ incidence of ADHD in the population. Our selected group consisted of children with clinically relevant externalizing and internalizing scores, and for each of these children one control child was drawn from the original group. Thus, we had relatively few control children compared to the original sample whereas all of the problem children were included. Moreover, because we matched the control children, this group contained, for example, more boys and older children than the control group of the original sample. To adjust for the selection procedure in our two-stage sampling design, a posthoc test in STATA (Statacorp, 1999) was performed with weighted prevalence rates that would be found in a random sample of a population of children visiting the YHC. These analyses yielded essentially the same results (data not shown). Thus, we can conclude that our results can be generalized to the original population.

A second potential drawback of our study is that the diagnosis ADHD was based on the ADIKA scores, which make use of information provided by the parents. The ADIKA may generate a subtle difference in diagnosis compared to the situation in which a child psychiatrist observes the
child in the clinic. Moreover, no reliability studies have been performed with the Dutch version of the DICA and we did not use multiple informants, which makes it difficult to confirm the pervasiveness of symptoms. Structured interviews with the teachers were not feasible because this was too time-consuming for such a large population study. Third, the relative low response rate (57.5%) could have biased the findings and should be mentioned as a limitation. However, bias-testing between responders and non-responders did not suggest systematic differences. Lastly, we did not include instruments to screen for learning disability. Thus, we cannot rule out the possibility that some of our results might be explained by learning disabilities. In conclusion, this longitudinal study underlines the importance of the manifestation of early neurocognitive deficits for a later diagnosis of ADHD even after adjustment for behavioral measures. Our results suggest that neurocognitive testing of visuomotor ability as well as several aspects of executive functioning can contribute in clinical practice to the early identification of children at risk of ADHD later, especially those with comorbid ODD/CD. On the other hand, it is not possible to distinguish between ADHD and 'borderline ADHD' on the basis of their neurocognitive profile at 5-6 years of age. More research is needed to elaborate this contribution of neuropsychology to predict a clinically significant syndrome that is primarily described in terms of behavioral characteristics. Along with neuroimaging studies (eg. Barkley, 1998, p165-168), prospective studies can lead to better hypotheses on the brain and behavioral dysfunctions of ADHD children. A better understanding of the origin of this child developmental disorder will improve the accuracy of early identification because we will then be better able to determine whether the behavior of a young child is age-appropriate or not. This will lead to better and earlier intervention programs.
References


StataCorp (1999). *Stata statistical software: release 6*. College Station, TX: Stata Corporation.


Westereich. (1998) *Amsterdams Diagnostisch Interview voor Kinderen en Adolescenten, ADHD-deel op basis van DSM IVcriteria* [Amsterdam Diagnostic Interview for Children and Adolescents, ADHD-part on the basis of DSM-IV criteria]. Internal publication.
3

Speed, Speed variability, and Accuracy of Information Processing in 5/6-year-old Children at risk of ADHD

Abstract

Objective: To examine whether 5/6-year-old children at risk of ADHD are characterized by specific attention deficits.

Method: By screening 1317 children in Southern Limburg (the Netherlands) with the Child Behavior Checklist, 363 were selected and underwent a computerized neurocognitive examination. Eighteen months later, standardized psychiatric information was obtained. Thirty-three ADHD children were compared with 75 'borderline ADHD' children, 122 pathological controls, and 133 healthy controls.

Results: ADHD and 'borderline ADHD' children were slower and more variable in their processing speed on all tasks than did children with no or other pathology. These differences were most pronounced for the divided and focused attention tasks. Support for a sustained attention deficit was less convincing. With regard to accuracy, only the proportion of misses on a Go-NoGo task was higher in the ADHD group than in the other groups. Evidence was found for a continuum, with ADHD at one extreme, followed by 'borderline ADHD', and healthy controls at the other.

Conclusions: Already at a young age, children at risk of ADHD show impairments of central processing, especially when cognitive demands are high, which suggests the presence of specific attention deficits. A lack of consistent effort is discussed as an underlying problem in ADHD.

INTRODUCTION

It is generally accepted that school-age children with Attention Deficit/Hyperactivity Disorder (ADHD) have characteristic deficits in attention and executive functioning (e.g., Barkley, 1997; Brodeur and Pond, 2001; Schachar et al., 2000; Swaab-Barneveld et al., 2000). However, most studies of ADHD have included children aged 7 and older, even though symptoms of the disorder arise before this age (American Psychiatric Association, 1994). A main reason for the lack of research with younger children is the difficulty in establishing the diagnosis, because the behavior of children of this age is variable and may temporarily include features of ADHD (Campbell and Ewing, 1990). Moreover, young children probably have not been exposed to situations in which attentional demands are high. For these reasons, symptoms of inattention are not always seen (American Psychiatric Association, 1994, p. 81).

The studies that did investigate young children with or at risk of ADHD mainly focused on behavioral, social, and preacademic functioning (DuPaul, et al., 2001; McGee et al., 1991; Mesman and Koot, 2001; Pierce et al., 1999). These studies showed that severe and persistent behavioral problems in young children are a potential risk of ADHD. Lower socioeconomic status, less optimal environment, reduced intellectual abilities, and preacademic skill deficits were also associated with ADHD. However, few studies used neurocognitive tests to examine young children (Mariani and Barkley, 1997), even though these tests may contribute to the early identification of ADHD. Marakovitz and Campbell (1998) included some laboratory measures of inattention and impulsivity in their study of hard-to-manage boys but found that these measures hardly differed between children later diagnosed with ADHD and control children. In contrast, two other detailed neurocognitive studies demonstrated deficits in vigilance, motor control, and working memory in young hyperactive children (Byrne et al., 1998; Mariani and Barkley, 1997). Both studies, however, used small and clinic-referred samples and did not use a range of attention tasks to detect specific attention deficits (in for example, sustained attention -the ability to continuously maintain a stable performance over time-, divided attention -the ability to divide attention across the necessary cognitive operations-, and focused attention -the ability to attend to relevant information while ignoring irrelevant information-). Therefore, study of the attention and information processing ability of young children at risk of ADHD may reveal which deficits contribute to the behavioral pattern of ADHD.

The main goal of this study is to examine whether 5/6-year-old children from a community sample who are at risk of ADHD are characterized by specific dysfunctions on computerized attention tasks (Amsterdam Neuropsychological Tasks (ANT), De Sonneville, 1999; De Sonneville et al., 1999). The ANT is a sensitive instrument for detecting various attention deficits in school-age child psychiatric populations (De Sonneville et al., 1994, Swaab-Barneveld et al., 2000). We examined whether the attention deficits are specific for ADHD, expecting that children later diagnosed with ADHD would perform significantly worse on attention tasks, especially on tasks of sustained attention, than children with psychopathological conditions other than ADHD (referred to as pathological controls). We also examined whether ADHD represents a dimensional trait expressed
in qualitative differences between individuals, with ADHD children at one extreme and healthy controls at the other (Barkley, 1998, p.73). For this purpose, we included a 'borderline ADHD' group (children exhibiting ADHD symptoms in fewer than two situations) and hypothesized that they would perform better than the ADHD children but worse than the healthy controls.

METHOD

This report is part of the 'Study of Attention Disorders in Maastricht' (SAM), a prospective cohort study of precursors of ADHD in the south of the province of Limburg, the Netherlands (Kalff et al., 2001, Kroes et al., 2001). The study was approved by the local ethics committee and informed consent was obtained from the participating children's parents. The study was performed in three phases (see below).

Subjects and Procedure

Phase 1 (selection): All parents of children in the second grade of normal kindergarten schools who visited the Youth Health Care (YHC) for a periodic health examination were approached to participate in the study. Of the 2290 eligible children, 1317 children (57.5%; 699 boys and 618 girls, mean age 5.87 (SD 0.41)) were granted permission to participate. Subsequently, parents were asked to complete the Child Behavior Checklist (CBCL; Verhulst et al., 1996). Responders and non-responders were compared for child characteristics, family factors, and environmental factors by randomly sampling 200 subjects in each group (for a more detailed description, see Kroes et al., 2001). Information was obtained anonymously from the medical records of the YHC. No significant differences were found between the groups.

On the basis of their CBCL scores, three groups of children (in total N=452) were selected for the second phase. The first group consisted of children with scores above the 90th percentile on the Externalizing scale and/or scores above the 95th percentile on the Attention Problem subscale (group 1; N=173). This group contained children with a putative risk for a later diagnosis of ADHD. The second group consisted of children with scores above the 90th percentile on the Internalizing scale and who were not members of group 1 (group 2; N=59). This group was included to contain children with a risk of other pathology. The third group, matched for age (± 2 months), sex, and school, consisted of children with Total problem scores below the 90th percentile (group 3; N=220).

Phase 2 (assessment): Four months after selection, 400 of the 452 selected children underwent a neurocognitive examination especially aimed at various aspects of attention. Nine children no longer participated and 43 children could not be assessed for logistic reasons, for example, because there was not enough school time or space available to assess the children. The assessment was carried out by one of ten well-trained psychologists who were blind to the group assignments and took place in a room at the children's school.

Phase 3 (follow-up diagnosis): Eighteen months after selection, 403 parents of the 452 selected children were interviewed using the Amsterdam Diagnostic Interview for Children and Adolescents.
(ADIKA; Kortenbout, van der Sluijs et al., 1993), a Dutch version of the Diagnostic Interview for Children and Adolescents (DICA; Herjanic and Reich, 1982) based on DSM-III-R criteria (American Psychiatric Association, 1987). DSM-IV criteria were used to diagnose ADHD (Westereich, internal publication, 1998). Eleven percent of the children's parents (N=49) refused further participation or had moved. The three interviewers were trained and supervised by a senior child psychiatrist and were blind to group membership. Four groups of children were classified: (1) an ADHD group; (2) a 'borderline ADHD' group which had the ADHD symptoms but whose symptoms did not lead to disruption in at least two situations; (3) a pathological control group which met the DSM-III-R criteria for oppositional defiant disorder, conduct disorder, anxiety disorders, mood disorders, obsessive compulsive disorder, Asperger's disorder, or disorders of elimination but not ADHD; (4) a healthy control group which had no psychiatric disorder. Comorbidity with other psychiatric disorders was allowed in the ADHD and 'borderline ADHD' groups because this appears the rule rather than the exception in ADHD in the general population (Kadesjo and Gillberg, 2001). Because of the diversity of the comorbidity, the various diagnoses were included in the pathological control group.

Complete neurocognitive and ADIKA data were available for 363 children (80% of the original selected sample). There were no significant differences in sex or CBCL selection group between the children included or excluded from the analyses. The groups differed significantly with regard to their age (younger in the included group; $F_{1,450}=27.23$, $p<.001$) and parental occupation (higher in the included group; $F_{1,450}=5.11$, $p=.024$).

Demographic characteristics for the four diagnostic groups are shown in Table 1. There were significant differences in sex, estimated IQ, and parental occupation between the groups. The CBCL groups were disproportionately distributed with regard to the ADIKA classification.

Table 1. Descriptives of the diagnostic groups

<table>
<thead>
<tr>
<th></th>
<th>(1) ADHD</th>
<th>(2) 'Borderline ADHD'</th>
<th>(3) Pathological controls</th>
<th>(4) Healthy controls</th>
<th>F</th>
<th>p a</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>33</td>
<td>75</td>
<td>122</td>
<td>133</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex (m/f)</td>
<td>24/9</td>
<td>50/25</td>
<td>68/54</td>
<td>63/70</td>
<td>11.23</td>
<td>.01 b</td>
</tr>
<tr>
<td>Age (in years)</td>
<td>6.17 (0.33)</td>
<td>6.24 (0.55)</td>
<td>6.14 (0.44)</td>
<td>6.20 (0.44)</td>
<td>0.85</td>
<td>.47</td>
</tr>
<tr>
<td>Estimated IQ c</td>
<td>90.33 (19.13)</td>
<td>88.04 (21.56)</td>
<td>97.90 (22.21)</td>
<td>94.71 (24.04)</td>
<td>3.31</td>
<td>.02 2&lt;3</td>
</tr>
<tr>
<td>LOA</td>
<td>3.41 (2.11)</td>
<td>3.69 (1.94)</td>
<td>3.83 (2.04)</td>
<td>4.55 (1.77)</td>
<td>5.33</td>
<td>.00 1,2,3&lt;4</td>
</tr>
<tr>
<td>CBCL (1/2/3)</td>
<td>30/1/2</td>
<td>47/7/21</td>
<td>44/26/52</td>
<td>16/12/105</td>
<td>116.09</td>
<td>.00 b</td>
</tr>
</tbody>
</table>

Note: LOA=level of occupational achievement of parents; CBCL=Child Behavior Checklist groups: 1=externalizing group, 2=internalizing group, 3=normal group; a Post hoc Tukey's HSD, $p<.05$; b Chi-square test; c estimated intelligence measure (mean=100, sd=15)
Dependent Measures

Five subtasks of the computerized test battery, *Amsterdam Neuropsychological Tasks* (ANT; De Sonneville, 1999), were used as dependent measures. All tasks were reaction time tasks. A Baseline Speed task, which requires no cognitive information processing, was followed by a Go-NoGo task to measure impulsivity. The other tasks measure different aspects of attention (sustained, divided, focused) which all reflect a higher level of controlled information processing or central processing (Schneider and Shriffrin, 1977). The children were required to respond as rapidly and accurately as possible to visual stimuli presented on a laptop screen. Trials with response times falling outside a predefined valid response window were automatically replaced by similar trials. For all tasks this window was 200-8000 ms post-stimulus onset, except for the Go-NoGo task (200-2300ms).

**Baseline Speed task.** A cross is displayed on the computer screen as fixation point, which, after a random post-response interval (PRI) of 500-2500 ms, changes into a white square. When this change occurs, the child is required to press the mouse key, after which the cross returns again. The task consists of two parts with 32 trials for each hand.

**Go-NoGo task.** In this task Go-signals, to which the child has to press a key, are randomly mixed with NoGo-signals, to which the child has to withhold a response (24 signals of each type, see Figure 1, upper left). The signals are presented for 800 ms (but disappear if a response is given within this period) with an event-rate of 3000 ms.

**Sustained Attention task.** This is a variant of a continuous performance task. During this task a house is continuously depicted on the screen. Each trial consists of the presentation of one animal randomly in one of the windows (see Figure 1, the shaded animals indicate the other possible locations of the stimulus, PRI = 250 ms). In total, 20 series of 12 trials are presented. The child is instructed to press the 'yes' key with the preferred hand when they detect a bee (target signal) and the 'no' key with the non-preferred hand when the signal does not contain the bee (non-target signal). During each series of 12 trials, 6 targets and 6 non-targets are randomly presented. Visual feedback on error responses is given by a red square that appears at the center of the house.

**Divided Attention task.** During this task the same house (see Figure 1) is continuously present on the screen. Each trial consists of the simultaneous presentation of four animals (instead of one animal) in the windows and in the door. The child is instructed to press the 'yes' key when the signal contains an animal from a memory set (target signal), and to press the 'no' key when this is not the case (non-target signal). This task consists of two parts: in part I the memory set is a mouse, and in part II it is a dog and a butterfly. Per task 20 targets and 20 non-targets are presented in random order with a PRI of 1000 ms.

**Focused Attention task.** During this task a fruit basket is continuously present on the screen (see Figure 1, upper right). Each trial consists of the simultaneous presentation of four pieces of fruit in the basket. Two pieces of fruit are aligned in a vertical fashion (top and bottom) and two pieces in a horizontal fashion (left and right). The child is instructed to attend the vertical axis and to ignore the pieces of fruit on the horizontal axis. The child has to press the 'yes' key when there are cherries (target fruit) on the vertical axis (target signal). If cherries are on the horizontal axis (irrelevant target
signal) or there are no cherries (non-target signal) the child has to press the 'no' key. These three signal types are presented in a random order (28 target signals, 14 irrelevant target signals, and 14 non-target signals, PRI = 1200 ms).

Figure 1. Go-NoGo task (two upper left pictures), Focused Attention task (upper right), Sustained Attention and Divided Attention tasks (lower left). The lower right diagram depicts the timing between signals. In the Go-NoGo task an event rate is used and signal duration is 800 ms. The other tasks use a postresponse interval (PRI); stimuli remain on the screen until a response is given. The tasks use a 500 ms warning signal (+), except the sustained attention task.

Other measures

The Vocabulary subtest from the Revised Amsterdam Child Intelligence Test (RAKIT) (Bleichrodt et al., 1987) was used to estimate intellectual functioning. The RAKIT is a Dutch intelligence test for children aged 4 to 11 years. The Vocabulary subtest measures the verbal ability of the child and is comparable with the well-known Peabody Picture Vocabulary Test, which is considered to give a valid approximation of IQ (Marakovitz and Campbell, 1998). The child must choose out of four pictures the one that matches the meaning of the word read aloud. The raw score (range 1-60) is the number of correct answers. This score is transformed into an IQ score (mean=100, sd=15).
The Level of Occupational Achievement (LOA) of the parents was scored on a 7-point scale based on the mental complexity of the work, as rated by job experts (DGA, 1989). The scale ranges from unskilled to academic labor. For children living with both parents, the highest level was used; for the remaining children, the level of the parent with whom the child lived was used.

**Scores**
The following main parameters were calculated for all tasks:

*Speed.* Reaction times were determined for correct responses (in ms) as an index for speed of information processing.

*Variability in speed.* Within-subjects standard deviations of reaction times were calculated as a measure of speed variability.

*Accuracy of processing.* The percentage of false alarms and misses were summed to obtain a percentage of errors as an index of accuracy. For the Go-NoGo task, the two error measures were analyzed separately because we were specifically interested in a measure of impulsivity (false alarms) and a measure of inattention (misses).

For the Sustained Attention task, additional variables were calculated to measure the continuous maintenance of alertness over time as well as the ability to adjust behavior following feedback on errors:

*Time-on-task.* Changes with time-on-task were calculated per block of 5 series (4 blocks in total) with respect to mean reaction time (tempo) and percentage error (accuracy).

*Fluctuation in speed across total task time.* The within-subjects standard deviation of completion time per series across 20 series was computed.

*Behavioral change after feedback.* Errors usually make subjects adjust their behavior by taking more time to process the next signal, which is reflected in a response delay when compared with speed of responses after correct (regular) responses. This ‘Shift’, computed as % of the regular speed \[100\times \frac{RT_{AFTER\ ERROR} - RT_{REGULAR}}{RT_{REGULAR}}\], is considered a measure of impulsivity.

**Statistical analyses**
Overall task performance at 5/6 years of age in the diagnostic groups was analyzed in a GLM repeated measures design with group as between-subjects factor and task (type) as within-subjects factor with separate runs for the three main scores, using the Geiser Greenhouse corrected probabilities to deal with unequal sample sizes. The Group x Task interaction reflects the extent to which differences between groups are task dependent, i.e. associated with specific attention deficits. Polynomial contrasts were used to examine the linear effect across the group classifications, i.e. whether the groups can be arranged on a continuum with ADHD at one extreme and healthy control children at the other. In order to test whether differences in speed, speed variability, and accuracy were specific for ADHD, GLM multivariate analyses were performed *per task*, in which all other groups were compared with the ADHD group, using simple contrasts. To investigate the effect of time-on-task in the Sustained Attention task, a GLM repeated measures analysis was performed with time-on-task as within-subjects factor (blocks 1,2,3,4) with separate runs for reaction time and error.
rate. ANOVAs were used to analyze group differences in fluctuations in speed and behavioral change after feedback ('Shift').

All analyses were done with and without IQ and Baseline Speed as covariates. Sex and LOA were not entered as covariates because univariate F-tests indicated that there were no significant differences between high and low LOA and no significant differences between boys and girls in the healthy control group on all measures except for the percentage of false alarms in the Go-NoGo task and the percentage of errors in the Divided Attention task (boys made more errors than girls). Therefore, sex was entered as covariate in the 'accuracy' GLM repeated measures analysis. All data were analyzed using SPSS 10.0.

RESULTS

Before all analyses, the tasks were examined separately for extremes in mean reaction times (>3 times the interquartile range from the median) and for error rates > 50%. For the repeated measures analyses including all tasks simultaneously, a child was excluded when its results on at least one task met the above-mentioned criteria. In total, 47 children were excluded. For the specificity analyses per task, the number of children excluded differed for each task: 1 child for the Baseline Speed task, 7 children for the Go-NoGo task, 6 children for the Sustained Attention task, 13 children for the Divided Attention task, and 32 children for the Focused Attention task. In the last task, a psychologist who tested 16 children did not instruct them properly. Figure 2 shows the three main parameters (speed, speed variability, and accuracy) for the diagnostic groups as a function of task type.

![Figure 2](image-url)

Figure 2: Speed, speed fluctuation, and accuracy of processing as a function of group classification and task type. BS=Baseline Speed, GNG=Go-NoGo, FA=Focused Attention, DA=Divided attention, SA=Sustained Attention, GNG-mi/fa=misses/false alarms GNG
**Speed**

The repeated measures analysis revealed a significant main effect of group on reaction times ($F_{3,291}=5.18, p=.002$) and a significant Group x Task interaction ($F_{12,1164}=3.55, p<.001$). Thus, the groups differed in speed of processing and these differences depended on the task, with the greatest differences being found for the Divided Attention task and, in particular, the Focused Attention task, and the smallest differences being found for the Baseline Speed task and the Go-NoGo task (see Figure 2, left panel). The polynomial contrast was significant ($p=.001$), showing a linear effect for reaction time, with controls being fastest and ADHD children being slowest. The simple contrasts in the multivariate analysis per task revealed that the healthy controls were faster than the ADHD children on all tasks (see RTs in Table 2), but only marginally faster on the Sustained Attention task and the Go-NoGo task. The pathological controls were faster than the ADHD children on the Baseline Speed task and the Focused Attention task, and the 'borderline ADHD' children were marginally slower than the ADHD children on the Divided Attention task only.

*Table 2. P-levels <.10 for simple contrasts with ADHD as the reference group, per task, per type of score*

<table>
<thead>
<tr>
<th>Task</th>
<th>Baseline</th>
<th>Go-NoGo</th>
<th>Sustained</th>
<th>Divided</th>
<th>Focused</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measure</td>
<td>RT</td>
<td>SD</td>
<td>RT</td>
<td>SD</td>
<td>RT</td>
</tr>
<tr>
<td>'Borderline' vs ADHD</td>
<td>.008</td>
<td>.092</td>
<td>.015</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pathol.contr. vs ADHD</td>
<td>.006</td>
<td>.090</td>
<td>.001</td>
<td>.010</td>
<td>.098</td>
</tr>
<tr>
<td>Healthy contr. vs ADHD</td>
<td>.001</td>
<td>.081</td>
<td>.026</td>
<td>.073</td>
<td>.095</td>
</tr>
</tbody>
</table>

Note: RT=reaction time; SD= Within-subjects standard deviation; PM=percentage of misses; PE=percentage of errors

**Variability in speed**

The repeated measures analysis revealed a significant main effect of group on speed variability ($F_{3,291}=6.78, p<.001$) and a significant Group x Task interaction ($F_{12,1164}=3.10, p<.001$). Thus, the groups differed in speed variability and these differences were task dependent, with the largest differences being found for the Divided Attention task and the Focused Attention task (see Figure 2, middle panel). The polynomial contrast was significant ($p<.001$), showing a linear effect for speed variability, with controls showing the least variability and ADHD the most. The simple contrasts in the multivariate analysis per task revealed that healthy controls and pathological controls showed less variability in speed than the ADHD children on all tasks, except for the Go-NoGo task (see SDs in Table 2). The 'borderline ADHD' children showed less variability in speed than the ADHD children on the Divided Attention task only.
Accuracy of processing
The repeated measures analysis revealed neither a significant main effect of group on accuracy of processing nor an interaction of Group x Task (see Figure 2, right panel). No linear effect was found for accuracy. The simple contrasts revealed that healthy controls and pathological controls made fewer misses on the Go-NoGo task than the ADHD children (see PM and PE in Table 2).

Sustained Attention measures
Figure 3 shows the additional Sustained Attention task parameters for the diagnostic groups. With respect to time-on-task, significant effects were found for tempo (F_{3,338}=4.80, p=.003) and accuracy (F_{3,338}=22.36, p<.001), indicating a decrease in vigilance over time (two left panels in Figure 3). However, no significant interaction effect was found between group and time-on-task, indicating that the effects did not differ between the groups. Figure 3 (inner right panel) shows that groups differed in the fluctuations in tempo across total task time, with the greatest fluctuations being seen in the ADHD children (F_{3,334}=4.91, p=.002). Figure 3 (outer right panel: ‘Shift’) shows that the groups had significantly different response delay times, with the 'borderline ADHD' children having a smaller response delay after feedback than the healthy controls (F_{3,338}=4.14, p=.007).

Figure 3 Tempo and accuracy during sustained attention with time-on-task (block 1–block 4), overall fluctuation and effect of feedback on response latency. 1=first 25% of trials, 2=second 25% of trials, 3=third 25% of trials, 4=last 25% of task trials
Analyses with IQ and Baseline Speed as covariates yielded similar results as the analyses without the covariates with regard to the main effects, linear effects, and simple contrasts in all analyses, and therefore data are not shown.

DISCUSSION

Five- to six-year-old children diagnosed with ADHD or 'borderline ADHD' at 18 months’ follow-up were found to be generally slower and, especially, more variable in their speed of information processing than children who were later diagnosed with no or other psychopathology. Interestingly, these differences between the groups were greatest on the Divided Attention task and, in particular, on the Focused Attention task, which suggests that central processing with more complex cognitive demands, may distinguish between the groups. Thus, when task demands are high, for instance when children have to divide their attention over multiple stimuli or have to ignore irrelevant information while attending to relevant information, young children at risk of ADHD have disproportionately greater difficulty in performing well than do control children. Thus, specific attention deficits, in addition to a slower baseline speed, occur early in the development of ADHD.

Regarding the Sustained Attention task, ADHD and 'borderline ADHD' children showed greater fluctuations in tempo than did the pathological and healthy controls, which indicates that such children have difficulty maintaining a stable level of performance. This deficit was present from the beginning: tempo and accuracy did not decline disproportionately during this task in the ADHD children. As only one of the three markers of a sustained attention deficit distinguished between the groups, it is questionable whether this deficit is the main problem in ADHD. Slusarek et al. (2001) found evidence that motivational problems could explain the attention problems of these children. We found that speed variability yielded the most significant differences between ADHD and no or other pathology, which suggests that the underlying problem may be a 'lack of consistent effort', as proposed by Oosterlaan and Sergeant (1996) and later confirmed by Kuntsi et al. (2001) with school-age ADHD children. Our results extend these findings to children aged 5-6 years, suggesting that a non-optimal performance or a lack of consistent effort may already be present early in the development of ADHD.

An inability to adjust behavior to error feedback is specific for school-age children with ADHD (De Sonneville et al., 1994; Swaab-Barneveld et al., 2000). To what extent this is also true for children at risk of ADHD remains to be seen, because we found only 'borderline ADHD' children to be significantly less responsive to feedback (35% delay) than the healthy control children (52% delay). The ADHD children showed a mean delay of 40% but it is possible that a small sample size has precluded reaching a significant result.

Remarkably, the ADHD or 'borderline ADHD' children could not be distinguished from the pathological and healthy controls by their accuracy on almost all tasks. Most studies with school-aged ADHD children have demonstrated an overall inaccuracy in responding (e.g. Kuntsi et al., 2001, Swaab-Barneveld et al., 2000). Our results only showed a difference in the percentage of misses on
the Go-NoGo task between the groups, with the ADHD group making more errors than the two control groups. This is indicative of a specific deficit of inattention (Corkum and Siegel, 1993). In contrast, the ADHD group did not make more false alarms on the Go-NoGo task, a measure of impulsivity, than the two control groups. Other authors have also failed to find significant differences in impulsivity (Marakovitz and Campbell, 1998; Mariani and Barkley, 1997), which suggests that the behavioral component of impulsivity, rather than the cognitive component of impulsivity, is involved in ADHD. However, in contrast with earlier findings is the high proportion of misses made by the ADHD group in this study, showing that difficulties with inattention may emerge later (Hart et al. 1995). Our results clearly demonstrate that inattention is associated with ADHD from an early age and therefore may be an important predictor of the disorder.

Our findings support the hypothesis that ADHD represents a dimensional trait. There was a linear change in speed and variability in speed between the groups, with ADHD at one extreme, followed by 'borderline ADHD', pathological controls, and then healthy controls. This suggests that the behavioral symptoms of ADHD alone do not necessarily lead to poor performance on computerized attention tasks. This is consistent with the findings of Schachar et al. (1995), who reported that the symptoms of ADHD can be exhibited without there being an inhibitory control deficit.

Limitations
Because of our study design, with a selection procedure based on CBCL scores, our results do not reflect the 'true' incidence of ADHD in the population. All children with problem behavior were selected whereas only some of the control children relative to the original population were selected. Weighted analyses can adjust for this, although there is some debate concerning this methodology (Korn and Graubard, 1999, p.159-182). Second, the relatively low-response rate (57.%) may have biased the findings. However, no systematic differences were found between responders and non-responders. Third, the psychiatric diagnoses in our study were generated by means of the ADIK. Although the reliability and validity of the original interview are good (Reich, 2000), no reliability studies have been done with the Dutch version of this diagnostic interview. Moreover, we did not use multiple informants – we were not able to obtain teacher information and the information provided by young children is considered unreliable. However, according to Barkley (1998, p.69) "parent reports may suffice for diagnostic purposes because this will lead to the same diagnosis based on teacher reports 90% of the time".

Clinical implications
Our results show that the diagnosis of ADHD requires a multidimensional approach in which neurocognitive assessment can contribute to the early identification of ADHD. Exposing young children to test situations in which demands are made on various aspects of attention makes it possible to observe specific attention deficits in these children. When task demands are high, which is often the case in daily life, children at risk of ADHD have difficulty performing well. In addition, the overall slowness and variability in speed of processing may result in their missing relevant
information, for example, at kindergarten and could lead to subsequent learning problems. More knowledge of the cognitive developmental profiles of children at risk of ADHD may lead to directed intervention programs, which may prevent further difficulties.
References


Low- and High-level Controlled Processing in Executive Motor Control Tasks in 5/6-year-old Children at risk of ADHD

Abstract
This study examined the performance of 5/6-year-old children who were later diagnosed with Attention Deficit/Hyperactivity Disorder (ADHD) on computerized motor control tasks involving low- and high-level controlled processing. In addition, motor control was compared with movement speed. By screening a general population sample of 1317 children, four groups of children were selected. Thirty children later diagnosed with ADHD were compared with 74 children later diagnosed with 'borderline ADHD' (children exhibiting all ADHD symptoms but without disruptions on at least two situations), 113 children later diagnosed with psychopathology other than ADHD, and 126 healthy controls. The ADHD children were in general less accurate and more variable in their movements than the children with other psychopathology and healthy controls. Under conditions of high-level controlled processing, the ADHD children were disproportionately more inaccurate and had a more unstable performance with their preferred hand than the other children. In addition, linear effects were found, with the ADHD children having the worst performance, followed by the 'borderline ADHD' children, and then both groups of control children. No significant group differences were found in movement speed. The main findings are interpreted as evidence for a specific deficit in high-level controlled processing in young children at risk of ADHD. Furthermore, the results support the notion that ADHD represents a dimensional trait rather than a pathological category. In addition, problems in movement control (the need to allocate attentional capacity) rather than problems in movement speed distinguish children at risk of ADHD from other children. The findings are interpreted as evidence that higher-order cognitive processes, such as planning and self-regulation, are already affected early in the development of ADHD.

Attention Deficit/Hyperactivity Disorder (ADHD) is the most commonly diagnosed psychiatric disorder of childhood, affecting approximately 3% to 5% of the school-age population and carries a potentially poor prognosis (American Psychiatric Association, 1994; Barkley, 1998; Stubbe, 2000). It is characterized by developmentally inappropriate and persistent symptoms of inattention, hyperactivity, and impulsivity. Several deficits that may contribute to the behavioral pattern of ADHD have been suggested, such as an attention deficit (Douglas, 1972), a motor control deficit (Van der Meere, Van Baal, & Sergeant, 1989), a working memory impairment, delay aversion, or a deficit in executive functions, in particular the response inhibition and self-regulation that control subordinate cognitive processes (Barkley, 1997; Kuntsi, Oosterlaan, & Stevenson, 2001).

It is difficult to draw straightforward conclusions about which deficit is primary because investigators have often used a wide range of tests that make demands on various aspects of perceptual, motor, and cognitive performance. For example, deficits in motor control in ADHD have been reported especially when more complex motor sequences have to be performed, which suggests that higher-order cognitive processes, such as planning and behavioral programming, organization in memory, and self-regulation, are involved (Barkley, 1998, p. 119). Indeed, Leung and Connolly (1998) found that neither motor organization nor motor execution was impaired in ADHD in a study in which they tried to unravel the nature of the motor deficits by examining the organization and execution of motor sequences in a simple sequential tapping task. They suggested that more complex motor tasks, which make demands on higher-order cognitive processing, should be used when trying to identify impairments in ADHD.

Higher-order cognitive processes, which are also referred to as effortful or controlled processes (Hasher & Zacks, 1979), require continuous attention capacity and are often opposed to automatic processes, which can be innate or acquired with practice and are not affected by attention capacity (Schneider & Shiffrin, 1977). While no evidence was found for deficits in acquired automatic processing (Van der Meere & Sergeant, 1988), most studies have demonstrated deficits in controlled processing in school-aged children with ADHD (Borcherding et al., 1988; De Sonneville, Njioikiktjen, & Bos, 1994; Nigg, Hinshaw, Carte, & Treuting, 1998). However, a recent study did not find support for a deficit in controlled processing in ADHD children (Hazell et al., 1999).

Most studies have involved only school-aged children and there is little information on the characteristics of ADHD in younger children (Mariani & Barkley, 1997). It is not clear whether deficits are already evident in children younger than 7 years. Moreover, it is difficult to establish a diagnosis of ADHD in young children because many symptoms of ADHD are transient (Marakovitz & Campbell, 1998). Thus, knowledge of the cognitive and motor characteristics of young children at risk of ADHD gained from longitudinal studies may contribute to more accurate and stable diagnoses at young ages.

The main goal of the present study was to compare the performance of 5/6-year-old children at risk of ADHD on two computerized complex motor control tasks that differ in the level of controlled processing required. These tests have proven suitable for use with young children (De Sonneville,
Visser, & Licht, 1999). The first task involves the tracking of a circular motor pattern, which requires relatively little attention capacity as the child is required to draw a well-practiced figure which can be planned in advance. Thus, this 'Tracking task' requires a low-level of controlled processing. The second task involves the close pursuit of a target that moves continuously and in random directions, necessitating continuous attention capacity and visual feedback. This 'Pursuit task' requires a high-level of controlled processing (Huijbregts et al., in press). Additionally, a simple test of movement speed was used to compare with the complex motor control tasks in order to determine whether particularly specific deficits in motor control underlie ADHD.

The performance of 5/6-year-old children diagnosed with ADHD at follow-up was compared not only with that of healthy control children, but also with that of children with psychopathology other than ADHD (referred to as pathological control children). This was done in order to evaluate the specificity of the results. In addition, a group of children with so-called 'borderline ADHD' (children exhibited ADHD symptoms but these symptoms did not lead to disruptions in at least two situations) was included to examine whether ADHD can be conceptualized as a dimensional trait expressed as qualitative differences between individuals.

METHODS

This report is part of a large research program entitled 'Study of Attention Disorders in Maastricht' (SAM), involving a prospective cohort study of 5- and 6-year-old children in the south of the province of Limburg, the Netherlands (Kalff et al., 2001; Kroes et al., 2001). The study was approved by the local ethics committee. The study was performed in three phases (see below).

Subjects and Procedure

Phase 1 (selection): The initial sample comprised 2290 children in the second grade of a normal kindergarten who visited the Youth Health Care (YHC) for a periodic health examination, of which 1317 (57.5%; 699 boys and 618 girls, mean age 5.87 (SD 0.41)) were granted permission by their parents to participate. Subsequently, parents were asked to complete the Dutch version of the Child Behavior Checklist (CBCL; Verhulst, Koot, & van der Ende, 1996). Responders and non-responders were compared for child characteristics, family factors, and environmental factors by randomly sampling 200 subjects in each group (for a full description, see Kroes et al., 2001). The information was obtained anonymously from the medical records of the YHC. No significant differences were found between the groups.

On the basis of the CBCL scores, three groups of children (in total N=452) were selected for the second phase. The first group consisted of children with scores above the 90th percentile on the Externalizing scale and/or scores above the 95th percentile on the subscale Attention Problem (Group I; N=173). This group contained children with a putative risk for a later diagnosis of ADHD (Chen, Faraone, Biederman, & Tsuang, 1994). The second group consisted of children with scores above the 90th percentile on the Internalizing scale and who were not members of group I (Group II; N=59).
Group II was included to investigate children at risk of other psychopathology. The third group, matched for age (± 2 months), sex, and school, consisted of children with Total problem scores below the 90th percentile (Group III; N=220).

Phase 2 (assessment): Four months after selection, 400 of the 452 selected children were administered the neurocognitive tasks. Nine children no longer participated in the study and 43 children could not be assessed for logistic reasons, for example, because there was not enough school time or space available to assess the children. The assessment was carried out by one of ten well-trained psychologists who were blind to the group assignments and took place in a separate room at the children's school.

Phase 3 (follow-up diagnosis): One and a half years after selection, 403 parents of the original 452 selected children were interviewed using the Dutch version of the Diagnostic Interview for Children and Adolescents (DICA; Herjani & Reich, 1982; ADIKA; Kortenbout van der Sluijs, Levita, Manen, & Defares, 1993). Eleven percent of the parents (N=49) refused further participation or had moved. The interviews were conducted by three interviewers who were trained and supervised by a senior child psychiatrist and who were blind to group membership. The ADIKA was adapted, using the criteria of DSM IV, to make it suitable for ADHD classification (Van Grimbergen, Célestin-Westreich, & Ponjaert-Kristofferson, 1999).

For the present study, complete neurocognitive and ADIKA data were available for 343 children (76% of the original selected sample). There were no significant differences between the children who were included or excluded in the analyses in terms of sex, CBCL selection groups, and parental occupation. The groups differed significantly only with regard to their age, the participating group being younger (F_{1,450}=12.37, p<.000).

Four groups of children were identified: (1) children who met the diagnostic criteria for ADHD at follow-up, hereafter called ADHD (N=30); (2) children who met the diagnostic criteria for ADHD at later age but whose symptoms did not lead to disruption in at least two situations, hereafter called 'borderline ADHD' (N=74); (3) children who met the DSM-III-R-criteria (American Psychiatric Association, 1987) for oppositional defiant disorder, conduct disorder, anxiety disorders, mood disorders, obsessive compulsive disorder, Asperger's disorder, or disorders of elimination at later age but without ADHD, hereafter called pathological controls (N=113); and (4) children who had no psychiatric disorder, hereafter called healthy controls (N=126). Comorbidity with other psychiatric disorders in the ADHD and 'borderline ADHD' groups was allowed because this appears to be the rule rather than the exception in children with ADHD (Kadesjö & Gillberg, 2001). Because of the diversity of the comorbid disorders, the different diagnoses were included in the pathological control group.

Group characteristics are shown in Table 1. There were significant differences in sex, estimated IQ, and parental occupation. The CBCL groups were disproportionately distributed with regard to the ADIKA classification. Hand preference was equally distributed between the groups.
Table 1. *Descriptive data of the diagnostic groups*

<table>
<thead>
<tr>
<th></th>
<th>(1) ADHD (N=30)</th>
<th>(2) 'borderline ADHD' (N=74)</th>
<th>(3) pathological controls (N=113)</th>
<th>(4) healthy controls (N=126)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sex (m/f)</strong></td>
<td>23 / 7</td>
<td>49 / 25</td>
<td>64 / 49</td>
<td>58 / 68</td>
</tr>
<tr>
<td><strong>Age (in years)</strong></td>
<td>6.17 (0.34)</td>
<td>6.25 (0.55)</td>
<td>6.15 (0.44)</td>
<td>6.22 (0.44)</td>
</tr>
<tr>
<td><strong>Estimated IQ</strong></td>
<td>90.37 (19.53)</td>
<td>88.49 (21.36)</td>
<td>98.62 (22.16)</td>
<td>94.21 (24.46)</td>
</tr>
<tr>
<td><strong>LOA</strong></td>
<td>3.31 (2.09)</td>
<td>3.69 (1.94)</td>
<td>3.82 (2.04)</td>
<td>4.50 (1.79)</td>
</tr>
<tr>
<td><strong>CBCL (1/2/3)</strong></td>
<td>28 / 1 / 1</td>
<td>47 / 7 / 20</td>
<td>42 / 24 / 47</td>
<td>16 / 12 / 98</td>
</tr>
<tr>
<td><strong>hand pref. (r/l)</strong></td>
<td>28 / 2</td>
<td>65 / 9</td>
<td>103 / 10</td>
<td>110 / 16</td>
</tr>
</tbody>
</table>

**F** 13.43  **p**<sub>a</sub> .00<sub>b</sub>

**Age (in years)** 0.87 .46

**Estimated IQ** 3.28 .02<sub>c</sub><sup>≤</sup><sub>3</sub>

**LOA** 4.83 .00<sub>1,2,3</sub><sup>≤</sup><sub>4</sub>

**CBCL (1/2/3)** 110.07 .00<sub>b</sub>

**hand pref. (r/l)** 1.59 .66<sub>b</sub>

Note. CBCL=Child Behavior Checklist groups: 1=externalizing group, 2=internalizing group, 3=matched control group; LOA=Level of Occupational Achievement of parents scored on 7-point-scale; hand pref.=hand preference. <sup>a</sup>Post hoc Tukey's HSD, p<.05; <sup>b</sup>Chi-square test; <sup>c</sup>=estimated intelligence screening measure (mean=100, sd=15)

**Intelligence screening measure and parental occupation**

Intellectual functioning was estimated by using the *Vocabulary subtest* from the *Revised Amsterdam Child Intelligence Test* (RAKIT; Bleichrodt, Drenth, Zaal, & Resing, 1987). This subtest measures the verbal ability of the child and is similar to the well-known *Peabody Picture Vocabulary Test*, which is considered to give a valid approximation of IQ (Marakovitz & Campbell, 1998).

The *Level of Occupational Achievement* (LOA) of the parents was scored on a 7-point scale based on the mental complexity of the work, as rated by job experts (DGA, 1989). The scale ranges from unskilled to academic labor. For children living with both parents, the highest level was used; for the remaining children, the level of the parent with whom the child lived was used.

**Dependent measures**

The *Tracking task* of the computerized test battery *Amsterdam Neuropsychological Tasks* (ANT; De Sonneville, 1999) requires the child to trace the mouse cursor in between a fixed outer and inner circle presented on the computer display (see Figure 1): once with the right hand (the cursor being moved in clockwise direction) and once with the left hand (the cursor being moved in counterclockwise direction). The program divides the trajectory into 60 radially equal segments and computes the mean distance between the cursor trajectory and the (invisible) midline per segment, resulting in 60 deviation scores. The total mean distance and the within-subjects variability of the mean distance for each hand were used as dependent measures for the accuracy of movement and stability of movement, respectively. In addition, the total time to complete the task with each hand was recorded in seconds and used as dependent measure as an index for movement speed.

The *Pursuit task* of the ANT requires the child to follow a target (an asterisk) that randomly moves across the computer screen as closely as possible with the mouse cursor (see Figure 1). The
task has to be executed with each hand lasting 60 seconds. The program computes the mean distance between the mouse cursor and the moving target per second, resulting in 60 deviation scores. The total mean distance and the within-subjects variability of the mean distance for each hand are used as dependent measures for the accuracy of movement and stability of movement, respectively.

The Purdue Pegboard (Tiffin, 1948) measures simple movement speed. The child is required to place pegs in holes as fast as possible using the dominant hand, then the non-dominant hand, followed by both hands together. Each trial lasts 30 seconds. The number of pegs placed correctly with each hand and both hands are used as dependent variables as an index for movement speed.

**Statistical analyses**

Task performance in the four diagnostic groups was analyzed using a GLM repeated measures design with group (4 levels) as between-subjects factor, and task (2 levels) as within-subjects factor with separate runs for each hand and each dependent measure. Task performance was analyzed separately for the preferred and the non-preferred hands because of the young age of the children. Hand preference is not established until approximately age six (Bryden, Pryde, & Roy, 2000), and an earlier study of normal young children showed differences in performance between the two hands (De Sonneville et al., 1999). Separate runs for mean distance and within-subject variability were
performed to avoid high correlations between the dependent variables. The Geiser Greenhouse corrected probabilities was used to deal with unequal sample sizes. Planned comparisons with the simple contrast were used to compare each of the groups with the ADHD group. The Group x Task interaction reflects the extent to which differences between groups are task dependent. Second repeated measures analyses were used with polynomial contrasts to examine the linear effect across the group classifications. That is, to examine whether the groups can be arranged on a continuum. The movement speed measures (total time on the Tracking task and the number of placed pegs in the Purdue Pegboard) were analyzed in univariate analyses of variance.

All analyses were conducted with and without IQ and parental occupation as covariates. Although sex is also suspected to correlate with ADHD, sex was not entered as a covariate because univariate F-tests indicated that there were no significant differences between boys and girls in the healthy control group on all measures except for the two movement speed measures: girls being slower than boys on the Tracking task and boys placing fewer pegs than girls in the Purdue Pegboard. In these 'speed' analyses, sex was entered as covariate. All data were analyzed using SPSS 10.0.

RESULTS

Five children were excluded from the analyses because their data for mean distance and within-subjects variability of the Tracking and Pursuit tasks were outliers on multivariate analysis (greater than 3 SD above or below mean). These children were equally distributed across the diagnostic groups. As all results with and without covariates were essentially the same, only the data without IQ and parental occupation as covariates are presented.

Figure 2 shows the mean distance and within-subjects variability for the diagnostic groups in the Tracking and Pursuit tasks for each hand separately. The repeated measures analyses revealed significant main effects of group on the mean distance (left panels) for the preferred hand \(F_{3,325}=8.37, p<.001\) as well as for the non-preferred hand \(F_{3,316}=4.00, p=.008\). Thus, the groups differed in terms of the accuracy of movements made with both hands. With the preferred hand, the responses of the ADHD children were less accurate than those of all other groups (simple contrasts: \(p=.004\), \(p<.001\), and \(p<.001\) compared to the 'borderline ADHD', pathological controls, and healthy controls, respectively). With the non-preferred hand, the ADHD children were only less accurate than the healthy controls \(p=.008\).

A significant Group x Task interaction was found for the preferred hand \(F_{3,325}=4.86, p=.003\) but not for the non-preferred hand \(F_{3,316}=0.60, p=.616\). Thus, the group differences depended on the task performed but only when the preferred hand was used, with the ADHD children being more inaccurate on the Pursuit task than on the Tracking task compared with the children from the other groups. The second repeated measure analysis to examine whether there was a linear effect between the groups yielded a significant polynomial contrast for the preferred and non-preferred hands \(p<.001\) and \(p=.003\), respectively), indicating a linear effect in accuracy, with ADHD children being the least accurate and healthy controls being the most accurate.
Similar results were found for the within-subjects variability (see Figure 2, right panels). The main group effects were significant for both the preferred hand ($F_{3,334}=8.76$, $p<.001$) and the non-preferred hand ($F_{3,334}=5.24$, $p=.002$). Thus, the groups differed in the stability of movement with both hands. With the preferred hand, the ADHD children had a more variable response than the other children ($p=.009$, $p<.001$, and $p<.001$ for the 'borderline ADHD', pathological controls, and healthy controls comparisons, respectively). However, with the non-preferred hand, the ADHD children only had a more variable response than the healthy controls ($p=.019$).

The two-way interaction Group x Task was significant for the preferred hand ($F_{3,334}=4.06$, $p=.007$) but not for the non-preferred hand ($F_{3,334}=0.12$, $p=.951$). Again, this shows that group differences depended on the task performed only when the preferred hand was used, with differences between ADHD and the other groups being much greater on the Pursuit task than on the Tracking task. The polynomial contrast in the second repeated measure analysis was significant for the preferred and non-preferred hands ($p<.001$ and $p=.006$, respectively), indicating a linear effect on the stability of movement, with ADHD children showing the most variability and healthy controls showing the least variability.

Figure 3 shows the results for the movement speed measures: total time on the Tracking task and the number of placed pegs on the Purdue Pegboard. No significant differences in movement speed were found between the groups for the preferred hand, the non-preferred hand, or both hands on the Purdue Pegboard test.
DISCUSSION

The present study was designed to examine the performance of young children at risk of ADHD on two complex motor control tasks that differ in their level of cognitive processing: the Tracking task requires low levels of controlled processing and the Pursuit task demands high levels of controlled processing. The ADHD children were compared with pathological control children, healthy control children, and 'borderline ADHD' children. Results showed that 5/6-year-old children who were later diagnosed with ADHD were less accurate and more variable in their movements with each hand on both tasks than the children later diagnosed with other or no pathology. Thus, in general, inaccuracy and instability of the movement pattern are found in ADHD but not in other psychopathologies, which demonstrates the specificity of the results.

Interestingly, ADHD children were disproportionately more inaccurate and had more variable movement patterns on the Pursuit task, which involves high-level controlled processing, than on the Tracking task, which involves low-level controlled processing, when using their preferred hand. Thus, young children at risk of ADHD have a more pronounced deficit in movement patterns when task demands are high, demonstrating a specific deficit in higher-order controlled processes in these children. The current findings corroborate those of most studies of school-aged ADHD children (Borcherding et al., 1988; De Sonneville et al., 1994; Nigg et al., 1998), but are new in that they concern younger children at risk of ADHD and yield information on cognitive processing in an early stage of ADHD. The results indicate that higher-order cognitive processes, such as planning, self-control, and self-regulation, are already affected early in the development of ADHD.

The failure to find disproportionately poorer results for the non-preferred hand may be because the children in our study were relatively young. Before the age of 6, hand preference is not yet
established in most children (Bryden et al., 2000) and the non-preferred hand may be unpracticed. A recent study showed that children aged 6 to 10 years overuse their preferred hand (Pryde, Bryden, & Roy, 2000). Consequently, every task with the non-preferred hand will require continuous attention, which may attenuate or even counteract the assumed contrast in low- versus high-level controlled processing of the two motor control tasks. In other words, both tasks probably involve highly controlled processing when performed with the non-preferred hand in all groups of children aged 5 to 6 years.

The results of this study support the view that ADHD is a dimensional trait rather than a pathological category (Barkley, 1998, p.73). Our results showed a linear effect in the accuracy and stability of movements across the groups, with ADHD at one extreme, followed by 'borderline ADHD', pathological controls, and finally healthy controls at the other. This suggests that defects in the control of complex perceptual-motor functions and in the planning of movements that are characteristics of ADHD occur over a continuum across the normal population. Categorical diagnoses are increasingly felt to be at variance with reality. The notion of a continuum has already been formulated with respect to other neurodevelopmental disorders (Bax, 1999), such as dyslexia (Shaywitz, Escobar, Shaywitz, Fletcher, & Makuch, 1992). Moreover, the intermediate position of the 'borderline ADHD' children is interesting in this respect, because the ADHD and 'borderline ADHD' children were equally symptomatic but differed in that the ADHD symptoms in the latter group did not lead to disruptions in at least two situations, a requirement for the diagnosis of ADHD according to DSM-IV (American Psychiatric Association, 1994). Apparently, the symptoms themselves do cause a poor performance. This is comparable to the results of Schachar, Tannock, Marriott, and Logan (1995), who reported that it is possible for children to exhibit symptoms of ADHD without having an inhibitory deficit. However, our results should be interpreted with care because the present study was not designed to determine whether ADHD symptoms cause poor performance or vice versa, that poor performance leads to ADHD symptoms.

The groups of children were not different in terms of movement speed, whereas they were in terms of motor control. These results are consistent with the literature on school-age children with ADHD (Leung & Connolly, 1998; Piek, Pitcher, & Hay, 1999; Steger et al., 2001). Our results demonstrate that these findings can be extended to even younger children aged 5/6 years. Apparently, it is not the quantitative aspect of a movement (namely, movement speed) but the qualitative aspect (namely, how controlled the movement is or how attentional capacity can be appropriately allocated) that distinguishes children at risk of ADHD from other children. This was also suggested by Kroes et al. (subject to revision), who found that qualitative aspects of all motor domains were predictive of ADHD. ADHD children appear to have problems with controlling the pattern of motor movements, which is in line with the self-regulation theory of Barkley (1997). According to this theory, a deficit in self-control or behavioral inhibition is the underlying deficit of ADHD.

Several limitations of the present study need to be considered. First, the relatively low response rate (57.5% of the initial sample) and the exclusion of selected children in the analyses (24% of the selected sample) may restrict generalization of the findings to larger populations. Yet, there were no significant differences between a subgroup of responders and non-responders. Comparison of the
excluded and included children showed only a difference in age, with excluded children being older. Second, the psychiatric diagnoses were based on the criteria on the ADIKA. Although the reliability and validity of the original interview are reported to be good (Reich, 2000), no reliability studies have been performed for the Dutch version of this diagnostic interview. Moreover, multiple informants were not used because the ADIKA only makes use of information provided by the parents. This may have led to over-inclusiveness. On the other hand, there is evidence of a 90% overlap between parent reports and teacher reports (Barkley, 1998, p. 69).

In conclusion, the present study demonstrated a disproportionately worse performance in terms of accuracy and stability of movements when task demands are high in 5/6-year-old children at risk of ADHD than in children suffering from other forms of pathology and healthy controls. This can be explained by a specific deficit in high-level controlled processing, in addition to a general poor performance on motor control tasks, which involve low-level controlled processing. The lack of differences in movement speed between the groups of children supports the view that qualitative aspects of movement are affected in ADHD. Thus, deficits in self-control and self-regulation already seem to be present early in the development of ADHD, and these deficits cause problems in several domains. This underscores the notion that a comprehensive assessment is needed to diagnose ADHD in young children.


Factors Affecting the Relation Between Parental Education as well as Occupation and Problem Behavior in Dutch 5- to 6-year-old Children

Abstract

Background: This study investigated whether problem behavior in 5- to 6-year-old children is related to parental education and occupation. It also analysed the contribution of correlating factors to explain this association.

Method: The Child Behavior Checklist was administered to a large community sample of 1317 children who were in the 1st year of normal primary school in the Netherlands. Outcome measures were total problem score, and externalising and internalising scale scores.

Results: Results of the logistic regression analyses indicated that higher rates of reported behavior problems were significantly associated with low parental education and occupation. These associations were mediated by low maternal age at delivery and single-parent families. The number of children in a family and physical illness of the parents did not contribute to these associations.

Conclusions: Parental education and occupation have a large impact on the mental health of young children. Psychosocial and biological factors are possible explanations for this phenomenon.

INTRODUCTION

Behavior problems at a young age are potential indicators of later psychopathology (Campbell, 1995). Several studies report that about 50% of 'hard-to-manage' pre-school children meet the criteria of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1994) for a disruptive disorder when they are older, compared to 16% of normal young children (Campbell and Ewing, 1990; McGee et al., 1991; Pierce et al., 1999). As it is still unclear in which children these problems persist, it is essential to examine the influence of risk factors at a young age to clarify their concerted action in the aetiology of psychopathology.

Numerous risk factors have been implicated in the early development of problem behaviors (Spieker et al., 1999). A consistent finding is the relation between low socio-economic status and higher rates of reported child behavior problems (see for review, Campbell, 1995). Frequently used markers of socio-economic status are parental education and occupation and family income (e.g. Belek, 2000; Mathiesen et al., 1999). Whereas family income has often been related to problem behavior in children (Bor et al., 1997; Shaw et al., 1998), the direct association between parental education and occupation and behavior problems is not so evident (Sonuga-Barke et al., 1996; Thomas et al., 1991). However, we believe that parental education and occupation are of potential relevance because they are the most important causal factors for a high income. The present study was therefore set up to investigate whether parental education and occupation are directly associated with child behavior problems. The effects of educational and occupational level were separately examined to determine whether they are related to problem behavior in a similar way.

Furthermore, it is unclear whether correlates such as family status, family size, maternal age, or living areas, contribute to this potential association between parental education and occupation and child behavior problems. Some recent studies have shown that these factors are associated with higher rates of reported behavior problems in children. For example, this has been demonstrated for single-parent families (Lavigne et al., 1998; Shaw et al., 1999), smaller family sizes (Lavigne et al., 1996; Taylor et al., 1986) as well as larger family sizes (Ghodsian et al., 1980), and decreasing maternal age at delivery (Orlebeke et al., 1998). Likewise, Rutter (1981) has shown that behavior problems in children are associated with personal overcrowding, unattractive layout, bad upkeep of housing estates, and poor mutual support, as found in urban areas. It is of importance to investigate these factors in a large-scale study to find whether they may confound, modify, or mediate the association between education and occupation and behavioral problems.

This report is part of a large longitudinal population study of the precursors of Attention Deficit/Hyperactivity Disorder (ADHD) which is being carried out in the city of Maastricht, the Netherlands (Kalff et al., 2001; Kroes et al., 2001). This paper addresses the following questions: (1) is there a direct association between behavior problems in a cohort of Dutch 5- to 6-year-old children and parental education and occupation, and (2) to what extent do family situation, family size, living area, country of birth, maternal age at delivery, and illness of parents contribute to this association?
METHODS

The ‘Study of Attention disorders in Maastricht’ (SAM) involves a prospective cohort study of the precursors of ADHD using a two-stage design. The methods described in this study are only related to this report, which used variables of the first stage of the SAM study. In this stage, the Child Behavior Checklist (CBCL; Achenbach, 1991; Dutch version: Verhulst et al., 1996) is used to investigate reported behavioral problems of a community sample of children.

Subjects and procedure
Children who attended the first year of primary school (±5-6 years of age) were recruited during a periodic systemic health examination performed by the Youth Health Care (YHC) in the Dutch region South Limburg from October 1996 through June 1997. In the Netherlands, this year precedes the year in which children learn to read and write. The sample is population based because school attendance is compulsory in the Netherlands for all children aged 5 and older. Moreover, almost all 4-year-olds already attend primary schools. The YHC organisation examines all children in the region and the response rate of the invited parents is 98%.

The initial group consisted of 2290 eligible children. Out of this group, the parents of 1317 (57.5%) children agreed to participate in the study and completed the CBCL. In 89.0% of the cases the respondent was the mother, in 8.7% the father, in 1.2% another caretaker, and in 1.1% the respondent was unknown. Written informed consent was obtained from the parents or guardians. Additional demographic information was collected from the medical records of the YHC. The study was conducted in accordance with the guidelines of the local ethics committee. No parents were excluded from the study and sufficient care was taken to assist those foreign-born parents who had less knowledge of the Dutch language. To compare the responders (N=1317) with the non-responders (N=973), a sub-sample of both groups were compared by randomly sampling 200 subjects from both groups, for sex, age, demographic, and child factors. The information was obtained anonymously from the medical records of the Youth Health Care.

The group of responders consisted of 699 boys and 618 girls. The children were aged from 4.44 to 7.33 years (mean 5.87 years, SD 0.41 years). All children were in the first year of primary school: 122 children (9.3%) were old-for-grade (based on their birth dates using a cut-off of 1 October 1990, which is used by the Dutch school system as an actual cut-off date for school entry) and 22 children (1.7%) were young-for-grade (born after 1 October 1991).

Measures
Demographic information
Parental education was scored on an 8-point scale (De Bie, 1987), ranging from primary education to higher vocational training and university, divided into three levels: low (1 and 2), middle (3, 4, and 5), and high (6, 7, and 8). This scoring system takes into account the qualitative aspects of education and thus is better than counting the years of scholastic education.
Level of occupational activity was obtained from a full description of parents’ occupational activities and was coded according to the system used by Statistics Netherlands (CBS, 1985). This code was then transformed to a 7-point-scale, based on the mental complexity of the work, as rated by job experts (DGA, 1989). This 7-point score ranges from low skilled to scientific labour and was finally divided into three levels for the present study: low (1 and 2), middle (3, 4 and 5), and high (6 and 7). For children living with both parents, the highest level was used; for the remaining children, the level of the parent with whom the child lived was used.

Other variables were transformed into dichotomous variables as follows: family status (child living with two parents versus one parent); living area (families living in an urban area (city of Maastricht: 121,000 inhabitants) versus non-urban areas (surrounding towns and municipalities with population densities of 12,000 to 20,000)); country of birth of parents (parents born in the Netherlands versus one or both parents born in a foreign country); illness of mother and father (chronic physical illness of the both parents versus healthy parents). Continuous intermediate factors were maternal age at delivery (mean age 29.75 years, SD 4.47 years) and the number of children in a family (mean number of children 2.22, SD 0.87). Concerning illness, 88 of the mothers (6.7%) and 77 of the fathers (5.8%) were chronically physical ill.

**Child Behavior Checklist (CBCL).** The CBCL (Achenbach and Edelbrock, 1983), translated into Dutch (Verhulst et al., 1985), is a widely used questionnaire with 120 items providing parent-reported data on problem behavior in children. A total problem behavior score is computed by summing the scores obtained for each item. We dichotomised the total problem score and considered children with a total problem score T > 63 (90th percentile) as 'clinical' cases. Achenbach (1991) has recommended this cut-off point as being clinically meaningful. In addition to the total problem score, the revised CBCL (Achenbach, 1991; Verhulst et al., 1996) produces nine clinical scales and generates two broad-band syndromes: externalising and internalising behavior problem syndromes. Externalising behavior comprises aggressive and delinquent behavior, and internalising behavior comprises withdrawn, anxious, and depressed behavior, and somatic complaints. Scores for externalising and internalising behavior are computed by summing the scores for the corresponding clinical scales.

Because there is considerable support in the literature for using these two broad-band syndromes (Egeland et al., 1996), we analysed the scores for externalising and internalising behavior in addition to the total problem behavior score. We also included 'comorbid' problems comprising both externalising and internalising behavior. The broad-band scores and the ‘comorbid’ outcome were dichotomised as described by Lavigne et al. (1996): child behavior is considered as 'pure' externalising if the externalising score exceeds the clinical range (90th percentile), while the internalising score is below the clinical range. Child behavior is considered as 'pure' internalising if the internalising score exceeds the 90th percentile while the externalising score is below the clinical range. 'Comorbid' problem cases were those children whose scores for both syndromes exceeded the 90th percentile.
Statistical analyses

All analyses were performed by using SPSS 6.1 for Macintosh. The prevalence of the total behavior problems, 'pure' externalising problems, 'pure' internalising problems, and 'comorbid' problems was calculated. Associations between the different behavior types and parental education and occupation and mediating factors were calculated with Chi-square analysis. U-shaped associations were tested with the quadratic terms. Logistic regression analysis was performed to examine the relationships between parental education, parental occupation, and child behavior problems after controlling for sex and age (in months). Odds ratios (OR) with 95% confidence intervals (CI) were calculated.

The relationships between correlates and behavior problems were analysed separately by logistic regression analyses adjusted for sex and age. Correlates that appeared to be significantly related to behavior problems were subsequently introduced in the models including parental education and occupation with child behavior problems as dependent variable. The contribution of each correlate to the association between parental education as well as occupation and child behavior problems was examined by introducing the correlates separately and simultaneously into these models. These logistic regression models were compared to the model without correlates and the percentage reduction in odds ratios was computed as a measure of size of the contribution of the correlates by means of the following formula:

\[
\frac{\text{OR}_{\text{PEO-mf}} - \text{OR}_{\text{PEO+mf}}}{\text{OR}_{\text{PEO-mf}} - 1} \times 100
\]

where PEO = parental education/occupation and -/+ mf = without/with mediating factors.

RESULTS

The initial sample consisted of 2290 children. The response rate was 57.5%, which resulted in a responding sample of 1317 children and 973 non-responders. A comparison of child, family, and environmental characteristics between a random subgroup of both responders and non-responders (N=2 x 200) yielded no significant differences in sex, age, and demographic factors. Also, child characteristics such as behavior problems reported by the school doctor were equal in both samples. The distribution of the demographic characteristics of the responders' group and a random subgroup of the non-responders sample is presented in Table 1.

Included sample

Out of the 1317 responders, 1140 children (86.6%) were included for further analyses in Tables 2 and 3, representing 49.8 % of the initial sample; 177 children were excluded because of missing data for one or more demographic variables (see Table 1). Comparable data were necessary in order to analyse whether parental education and behavior problems were associated and if this association was mediated by several factors.
Table 1. *Demographic characteristics of the responders' sample and a random subgroup of the non-responders sample*

<table>
<thead>
<tr>
<th>Variable</th>
<th>Responders (N=1317)</th>
<th>Non-responders (N=200)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n (%)</td>
<td>n (%)</td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>males</td>
<td>699 (53.1)</td>
<td>100 (50.0)</td>
</tr>
<tr>
<td>females</td>
<td>618 (46.9)</td>
<td>100 (50.0)</td>
</tr>
<tr>
<td><strong>Parental education</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>high</td>
<td>476 (36.1)</td>
<td>n.a.</td>
</tr>
<tr>
<td>middle</td>
<td>532 (40.4)</td>
<td>n.a.</td>
</tr>
<tr>
<td>low</td>
<td>185 (14.1)</td>
<td>n.a.</td>
</tr>
<tr>
<td>missing</td>
<td>124 (9.4)</td>
<td>n.a.</td>
</tr>
<tr>
<td><strong>Parental occupation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>high</td>
<td>442 (33.6)</td>
<td>52 (26.0)</td>
</tr>
<tr>
<td>middle</td>
<td>381 (28.9)</td>
<td>53 (26.5)</td>
</tr>
<tr>
<td>low</td>
<td>377 (28.6)</td>
<td>59 (29.5)</td>
</tr>
<tr>
<td>missing</td>
<td>117 (8.9)</td>
<td>36 (18.0)</td>
</tr>
<tr>
<td><strong>Family status</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>two-parent families</td>
<td>1153 (87.6)</td>
<td>167 (83.5)</td>
</tr>
<tr>
<td>single-parent families</td>
<td>141 (10.7)</td>
<td>32 (16.0)</td>
</tr>
<tr>
<td>missing</td>
<td>23 (1.7)</td>
<td>1 (0.5)</td>
</tr>
<tr>
<td><strong>Living area</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>urban area</td>
<td>775 (58.8)</td>
<td>124 (62.0)</td>
</tr>
<tr>
<td>rural areas</td>
<td>542 (41.2)</td>
<td>76 (38.0)</td>
</tr>
<tr>
<td><strong>Country of birth</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Netherlands</td>
<td>1147 (87.1)</td>
<td>168 (84.0)</td>
</tr>
<tr>
<td>foreign-born parents</td>
<td>141 (10.7)</td>
<td>32 (16.0)</td>
</tr>
<tr>
<td>missing</td>
<td>29 (2.2)</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

Note. na = not available

There were no significant differences in mean scores for the total problem scale, externalising scale, or internalising scale of the CBCL between the children who were excluded from further analysis (N=177) and those who were included in the analysis (N=1140). However, there were more clinical cases (above the 90th percentile) with the total problem behavior and 'comorbid' behavior among the excluded children than among the included children ($\chi^2=19.92$, $p=.000$ and $\chi^2=4.32$, $p=.038$, respectively) (see Table 2). There were no differences in the prevalence of clinical cases of externalising and internalising behavior between the included and excluded children.
Table 2. Number and percentages of children in clinical ranges (>90th percentile) on the total problem; 'pure' externalising; 'pure' internalising; and 'comorbid' scores of the Child Behavior Checklist (CBCL)

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Total problem behaviour (%)</th>
<th>Externalising behaviour (%)</th>
<th>Internalising behaviour (%)</th>
<th>'Comorbid' behaviour (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td>1140</td>
<td>10.4</td>
<td>7.2</td>
<td>5.1</td>
<td>3.5</td>
</tr>
<tr>
<td>excluded children</td>
<td>177</td>
<td>22.0***</td>
<td>10.7</td>
<td>5.6</td>
<td>6.8*</td>
</tr>
<tr>
<td>Parental education</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>high</td>
<td>458</td>
<td>6.6</td>
<td>4.8</td>
<td>3.9</td>
<td>3.1</td>
</tr>
<tr>
<td>middle</td>
<td>510</td>
<td>9.2</td>
<td>7.3</td>
<td>5.5</td>
<td>2.9</td>
</tr>
<tr>
<td>low</td>
<td>172</td>
<td>23.8***</td>
<td>13.4**</td>
<td>7.0</td>
<td>6.4</td>
</tr>
<tr>
<td>Parental occupation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>high</td>
<td>426</td>
<td>6.3</td>
<td>5.2</td>
<td>4.5</td>
<td>1.6</td>
</tr>
<tr>
<td>middle</td>
<td>366</td>
<td>6.0</td>
<td>6.3</td>
<td>4.9</td>
<td>2.2</td>
</tr>
<tr>
<td>low</td>
<td>356</td>
<td>18.3***</td>
<td>9.8</td>
<td>6.2</td>
<td>7.0***</td>
</tr>
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<td>Confounders</td>
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<td></td>
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<td>Sex</td>
<td></td>
<td></td>
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<td></td>
<td></td>
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<tr>
<td>males</td>
<td>601</td>
<td>11.0</td>
<td>7.2</td>
<td>5.3</td>
<td>3.8</td>
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<tr>
<td>females</td>
<td>9.6</td>
<td>7.2</td>
<td>4.8</td>
<td>3.2</td>
<td></td>
</tr>
<tr>
<td>Age child (in years)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ 5.5</td>
<td>230</td>
<td>9.6</td>
<td>7.8</td>
<td>5.2</td>
<td>3.0</td>
</tr>
<tr>
<td>&gt; 5.5 - ≤ 6.0</td>
<td>489</td>
<td>8.0</td>
<td>6.3</td>
<td>5.5</td>
<td>2.9</td>
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<tr>
<td>&gt; 6.0</td>
<td>421</td>
<td>13.5*</td>
<td>7.8</td>
<td>4.5</td>
<td>4.5</td>
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<tr>
<td>two-parent</td>
<td>1043</td>
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<td>6.7</td>
<td>4.9</td>
<td>2.9</td>
</tr>
<tr>
<td>single-parent</td>
<td>97</td>
<td>24.7***</td>
<td>12.4*</td>
<td>7.2</td>
<td>10.3***</td>
</tr>
<tr>
<td>Number of children</td>
<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>161</td>
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<td>8.1</td>
<td>4.3</td>
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<td>656</td>
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<td>6.6</td>
<td>5.9</td>
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<tr>
<td>3</td>
<td>252</td>
<td>10.3</td>
<td>8.7</td>
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<tr>
<td>≥ 4</td>
<td>71</td>
<td>9.9</td>
<td>5.6</td>
<td>7.0</td>
<td>4.2</td>
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<tr>
<td>Living area</td>
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<td></td>
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<tr>
<td>urban</td>
<td>467</td>
<td>12.3</td>
<td>8.3</td>
<td>4.6</td>
<td>4.5</td>
</tr>
<tr>
<td>nonurban</td>
<td>673</td>
<td>7.5**</td>
<td>5.6</td>
<td>5.8</td>
<td>2.1*</td>
</tr>
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<tr>
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<td>6.5</td>
<td>5.4</td>
<td>3.2</td>
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<tr>
<td>foreign-born</td>
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<td>17.0*</td>
<td>13.4**</td>
<td>2.7</td>
<td>6.3</td>
</tr>
<tr>
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<td>≤ 26</td>
<td>246</td>
<td>17.5</td>
<td>10.6</td>
<td>7.3</td>
<td>4.5</td>
</tr>
<tr>
<td>&gt; 26 - ≤ 33</td>
<td>671</td>
<td>8.9</td>
<td>6.1</td>
<td>5.2</td>
<td>3.6</td>
</tr>
<tr>
<td>&gt; 33</td>
<td>223</td>
<td>6.7***</td>
<td>6.7</td>
<td>2.2*</td>
<td>2.2</td>
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<td>10.4</td>
<td>7.3</td>
<td>5.2</td>
<td>3.6</td>
</tr>
<tr>
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<td>71</td>
<td>9.9</td>
<td>5.6</td>
<td>2.8</td>
<td>2.8</td>
</tr>
</tbody>
</table>

Note. Chi-square tests: *p<.05; **p<.01; ***p<.001; a reference group in logistic regression analyses; b Comparing parental occupation and problem behaviour and its correlates without having missing data lead to a group of 1148 children; c variables categorised in table for convenience of comparison and in Chi-square analyses; for maternal age this is done as in Orlebeke et al. (1998); in the other analyses these variables are included as continuous variables.
Parental education was significantly related to the total problem behavior and to externalising behavior (Table 2). Disturbed behavior was more prevalent in children of parents with the lowest level of education than in children of parents with the highest level of education (total problem behavior: \( \chi^2=41.55, p=.000 \); externalising behavior: \( \chi^2=13.76, p=.001 \)). The same was true for parental occupation (total problem behavior: \( \chi^2=40.04, p=.000 \); externalising behavior: \( \chi^2=6.90, p=.032 \)) and parental occupation was also related to 'comorbid' behavior (\( \chi^2=19.38, p=.000 \)). Thus, all behavior problems, except internalising behavior problems, were more prevalent in children whose parents had the lowest level of occupation than in children whose parents had the highest level of occupation.

No statistically significant differences were found between boys and girls on all problem scales. Age was associated with the total behavior problem scale (\( \chi^2=7.74, p=.021 \)). The data suggested a U-shaped association, but the quadratic term of age (in months) was not statistically significant (\( p=.148 \)). No significant differences were found for the age groups on the other problem scales.

Four correlates were significantly related to the total problem scale (see Table 2). A higher prevalence of child behavior problems was significantly related to living in single-parent families (\( \chi^2=23.66, p=.000 \)), living in an urban area (\( \chi^2=6.95, p=.008 \)), having foreign-born parents (\( \chi^2=5.85, p=.016 \)), and having a younger mother at delivery (\( \chi^2=18.06, p=.000 \)). After adjustment for sex and age in separate logistic regression analyses, significant odds ratios were obtained on the total behavior problems for single-parent families (OR=3.27; 95% CI=1.96 to 5.44), urban area (OR=1.72; 95% CI=1.14 to 4.49), foreign-born parents (OR=1.99; 95% CI=1.16 to 3.42), and younger maternal age (continuous scale in years; OR=0.91; 95% CI=0.87 to 0.95). The number of children in a family and physical illness of the parents were not significantly related to behavior problems. Similar results were found for the other behavioral outcomes.

The results for the separate logistic regression models are shown in Table 3 for parental education and Table 4 for parental occupation. The correlation between parental education and parental occupation was moderately strong (\( r=.6511, p=.000 \)). Parental education was significantly associated with total behavior problems. Children of parents with lower educational levels had a 4.17-fold higher risk of having reported behavior problems than did children of parents with higher educational levels. Two correlates strongly contributed to the association between parental education and reported child behavior problems: maternal age and family situation. The odds ratios for low parental education and total problem behavior decreased from 4.17 to 3.24 (29% reduction) for maternal age and from 4.17 to 3.60 (18% reduction) for family situation. Living area and country of birth of the parents hardly contributed to this association (9% and 3% reduction, respectively). When all correlates were considered simultaneously, the odds ratio for total behavior problems decreased to 2.76 (44% reduction), which was still significant.

With regard to the other problem behavior scales, the pattern of results was similar. Children of parents with low educational levels were 2.96 times more likely to have externalising behavior problems than were children of parents with higher educational levels. Inclusion of all correlates reduced the association between low parental education and high rates of externalising behavior problems from 2.96 to 2.83 (7% reduction), which was still significant. For the internalising behavior
problems, the odds ratio decreased from 1.93 to 1.43 (54% reduction) and for the 'comorbid' behavior problems the decrease was 1.97 to 1.04 (96% reduction). Except for the externalising behavior, maternal age explained most of the associations between parental education and various types of problem behavior.

Table 3. **Odds ratios (OR) and 95% confidence intervals (95% CI), adjusted for sex and age, for clinical scores on CBCL by parental education (PE) separately and simultaneously adjusted for correlates (N=1140)**

<table>
<thead>
<tr>
<th></th>
<th>Total problem behavior</th>
<th>Externalising behavior</th>
<th>Internalising behavior</th>
<th>'Comorbid' behavior</th>
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<tbody>
<tr>
<td></td>
<td>OR (95% CI)</td>
<td>OR (95% CI)</td>
<td>OR (95% CI)</td>
<td>OR (95% CI)</td>
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<tr>
<td><strong>Main model</strong></td>
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<tr>
<td>Parental Education (PE)</td>
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</tr>
<tr>
<td>High</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Middle</td>
<td>1.43 (0.88 to 2.31)</td>
<td>1.54 (0.89 to 2.65)</td>
<td>1.44 (0.78 to 2.63)</td>
<td>0.95 (0.45 to 1.99)</td>
</tr>
<tr>
<td>Low</td>
<td>4.17 (2.48 to 6.99)***</td>
<td>2.96 (1.59 to 5.52)***</td>
<td>1.93 (0.90 to 4.12)</td>
<td>1.97 (0.86 to 4.50)</td>
</tr>
<tr>
<td><strong>Correlates</strong></td>
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<tr>
<td>Family situation</td>
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</tr>
<tr>
<td>PE high</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Middle</td>
<td>1.35 (0.83 to 2.18)</td>
<td>1.50 (0.87 to 2.59)</td>
<td>1.41 (0.77 to 2.60)</td>
<td>0.86 (0.41 to 1.82)</td>
</tr>
<tr>
<td>Low</td>
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<td>2.76 (1.47 to 5.20)***</td>
<td>1.84 (0.85 to 3.99)</td>
<td>1.54 (0.65 to 3.63)</td>
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</tr>
<tr>
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<td>1.00</td>
<td>1.00</td>
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</tr>
<tr>
<td>Middle</td>
<td>1.43 (0.89 to 2.31)</td>
<td>1.54 (0.89 to 2.65)</td>
<td>1.44 (0.79 to 2.65)</td>
<td>0.95 (0.45 to 1.99)</td>
</tr>
<tr>
<td>Low</td>
<td>3.90 (2.32 to 6.58)***</td>
<td>2.79 (1.49 to 5.23)***</td>
<td>2.04 (0.95 to 4.40)</td>
<td>1.77 (0.77 to 4.08)</td>
</tr>
<tr>
<td>Country of birth</td>
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</tr>
<tr>
<td>PE high</td>
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<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Middle</td>
<td>1.40 (0.87 to 2.26)</td>
<td>1.50 (0.87 to 2.58)</td>
<td>1.47 (0.80 to 2.69)</td>
<td>0.92 (0.44 to 1.94)</td>
</tr>
<tr>
<td>Low</td>
<td>4.09 (2.43 to 6.88)***</td>
<td>2.88 (1.54 to 5.38)***</td>
<td>1.96 (0.92 to 4.21)</td>
<td>1.91 (0.83 to 4.37)</td>
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<tr>
<td>Age mother</td>
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</tr>
<tr>
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<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Middle</td>
<td>1.24 (0.76 to 2.04)</td>
<td>1.61 (0.92 to 2.81)</td>
<td>1.22 (0.65 to 2.28)</td>
<td>0.78 (0.36 to 1.67)</td>
</tr>
<tr>
<td>Low</td>
<td>3.24 (1.86 to 5.66)***</td>
<td>3.21 (1.64 to 6.25)***</td>
<td>1.40 (0.61 to 3.17)</td>
<td>1.37 (0.55 to 3.36)</td>
</tr>
<tr>
<td><strong>All variables (simultaneously)</strong></td>
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</tr>
<tr>
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<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Middle</td>
<td>1.16 (0.71 to 1.92)</td>
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<td>1.22 (0.65 to 2.29)</td>
<td>0.70 (0.32 to 1.53)</td>
</tr>
<tr>
<td>Low</td>
<td>2.76 (1.56 to 4.89)***</td>
<td>2.83 (1.43 to 5.57)***</td>
<td>1.43 (0.62 to 3.29)</td>
<td>1.04 (0.41 to 2.64)</td>
</tr>
</tbody>
</table>

p<.05; ** p< .01; *** p<.001
Parental occupation and child behavior problems were also significantly related (Table 4). Children of parents with a low-level occupation had a 3.05-fold higher risk of having reported behavior problems than did children of parents with a high-level occupation. Again, low maternal age explained most (40%) of the association. If all correlates were entered simultaneously, the odds ratio for total problem behavior decreased from 3.05 to 1.94 (54% reduction). The results for the other problem scales were essentially the same as those for the regression models with parental education.

Table 4  Odds ratios (OR) and 95% confidence interval (95% CI), adjusted for sex and age, for clinical scores on CBCL by parental occupation (PO) separately and simultaneously adjusted for correlates (N=1148)

<table>
<thead>
<tr>
<th></th>
<th>Total problem behavior</th>
<th>Externalising behavior</th>
<th>Internalising behavior</th>
<th>'Comorbid' behavior</th>
</tr>
</thead>
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<td><strong>Main model</strong> Parental Occupation (PO)</td>
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<td>1.00</td>
</tr>
<tr>
<td>middle</td>
<td>0.90 (0.50 to 1.61)</td>
<td>1.21 (0.66 to 2.21)</td>
<td>1.12 (0.58 to 2.16)</td>
<td>1.26 (0.45 to 3.53)</td>
</tr>
<tr>
<td>low</td>
<td>3.05 (1.89 to 4.93)***</td>
<td>1.94 (1.11 to 3.39)*</td>
<td>1.43 (0.76 to 2.69)</td>
<td>4.12 (1.75 to 9.71)**</td>
</tr>
<tr>
<td>Correlates Family situation</td>
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</tr>
<tr>
<td>PO high</td>
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<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>middle</td>
<td>0.91 (0.51 to 1.63)</td>
<td>1.21 (0.66 to 2.22)</td>
<td>1.11 (0.58 to 2.17)</td>
<td>1.30 (0.46 to 3.63)</td>
</tr>
<tr>
<td>low</td>
<td>2.94 (1.82 to 4.76)***</td>
<td>1.89 (1.09 to 3.31)*</td>
<td>1.39 (0.73 to 2.63)</td>
<td>3.83 (1.62 to 9.09)**</td>
</tr>
<tr>
<td>Living area</td>
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</tr>
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<td>PO high</td>
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</tr>
<tr>
<td>middle</td>
<td>0.90 (0.50 to 1.62)</td>
<td>1.21 (0.66 to 2.22)</td>
<td>1.11 (0.57 to 2.16)</td>
<td>1.28 (0.46 to 3.58)</td>
</tr>
<tr>
<td>low</td>
<td>2.94 (1.81 to 4.76)***</td>
<td>1.87 (1.07 to 3.28)*</td>
<td>1.51 (0.79 to 2.87)</td>
<td>3.81 (1.61 to 9.02)**</td>
</tr>
<tr>
<td>Country of birth</td>
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</tr>
<tr>
<td>PO high</td>
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<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>middle</td>
<td>0.88 (0.49 to 1.57)</td>
<td>1.18 (0.64 to 2.16)</td>
<td>1.13 (0.52 to 2.48)</td>
<td>1.23 (0.44 to 3.43)</td>
</tr>
<tr>
<td>low</td>
<td>2.82 (1.74 to 4.58)***</td>
<td>1.76 (1.00 to 3.10)*</td>
<td>1.52 (0.80 to 2.88)</td>
<td>3.74 (1.57 to 8.88)**</td>
</tr>
<tr>
<td>Age mother</td>
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</tr>
<tr>
<td>PO high</td>
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<td>1.00</td>
<td>1.00</td>
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</tr>
<tr>
<td>Middle</td>
<td>0.79 (0.49 to 1.42)</td>
<td>1.22 (0.66 to 2.24)</td>
<td>0.96 (0.49 to 1.89)</td>
<td>1.12 (0.39 to 3.15)</td>
</tr>
<tr>
<td>Low</td>
<td>2.24 (1.39 to 3.61)**</td>
<td>1.97 (1.08 to 3.62)*</td>
<td>0.95 (0.47 to 1.92)</td>
<td>3.03 (1.20 to 7.65)*</td>
</tr>
<tr>
<td>All variables (simultaneously)</td>
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</tr>
<tr>
<td>PO high</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
<td>1.00</td>
</tr>
<tr>
<td>Middle</td>
<td>0.78 (0.43 to 1.42)</td>
<td>1.19 (0.65 to 2.20)</td>
<td>0.98 (0.50 to 1.92)</td>
<td>1.13 (0.40 to 3.21)</td>
</tr>
<tr>
<td>Low</td>
<td>1.94 (1.14 to 3.12)*</td>
<td>1.71 (0.92 to 3.18)</td>
<td>1.05 (0.52 to 2.13)</td>
<td>2.45 (0.95 to 6.32)</td>
</tr>
</tbody>
</table>

*p<.05; **p<.01; ***p<.001
DISCUSSION

This study shows that reported problem behavior in children attending normal primary schools is significantly related to a low level of parental education and occupation, and that low maternal age at delivery and single-parent family contributed significantly to these associations. These findings were fairly consistent for all different types of problem behavior investigated (externalising, internalising, and 'comorbid' behavior). This adds weight to the notion that socio-economic differences in behavior problems in childhood may be causal to lower parental education and occupation and can provide clues about possible interventions with respect to the behavioral problems of these children. Although these variables are related to one another, as early parenthood may disrupt education and prejudice subsequent occupational opportunities, the finding that the association between parental education and occupation and child behavior problems is driven by maternal age is an interesting finding and deserves several explanations.

The strong association between socio-economic status and child problem behavior has often been explained in terms of economic hardship (McLoyd, 1990). Parents with a low-level occupation, and thus a low income, have fewer resources (e.g. money, housing condition, spare time) resulting in "less capacity for supportive, consistent, and involved parenting" than do parents with a high-level occupation (McLoyd, 1998). Because of the availability of material and immaterial resources, parents with a high level of occupation have the opportunity to choose from different ways of parenting. This freedom of choice among "better off" parents may underlie the lower prevalence of behavior problems among their children.

However, it may not all be explained by economic hardship. The fact that parental education is separately related to problem behavior can be explained from a psychological point of view, especially as this association is highly explained by maternal age. Education can be seen as a proxy of mental ability and intellectual functioning, and thus has to do with 'knowledge and experience'. Parents with a higher level of education will have more knowledge, resulting in a different approach towards their children, which may prevent problem behavior in their children. The finding that maternal age was the most important correlate supports this explanation because younger mothers tend to be less well educated.

Another line of approach is the different rearing styles that are related to various behavioral patterns (Castro et al., 1999). A relation between the role of parenting and problem behavior in children has also been identified (Campbell, 1995; Webster-Stratton and Hammond, 1999). Thus, rearing styles may be a determinant of the association between parental education and problem behavior in children. This is also consistent with the importance of maternal age because rearing behavior or rearing styles change as parents gain experience, as they grow older, and this could affect the child's behavior. Of course, the parents' nature cannot be ruled out in this discussion.

Finally, studies by Zumoff et al. (1995) and Orlebeke et al. (1998) are relevant to the question whether biological factors are responsible for the effects found. The latter authors stressed the importance of the continuous relation between child behavior problems and maternal age at delivery in their study rather than the dichotomous relation of teenage mothers with problem children versus
non-teenage mothers with healthy children. Our study also demonstrated increasing child behavior problems with continuously decreasing maternal age at delivery. On the basis of this relation, Orlebeke et al. put forward a biological hypothesis in which the testosterone level played a key role. Levels of testosterone are higher in younger women than in older women, and high testosterone levels in a pregnant mother might influence the child's brain development, leading to more 'aggressive' attitudes. However, our results showed that maternal age had the largest protective effect on internalising problem behavior and had no effect on externalising problem behavior, which is contrary to the testosterone hypothesis. The several explanations thus lead to the belief that more research should be performed to get a clear picture of the relative contribution of correlates related to economic hardship, experience and 'learning' versus biological factors.

Although the study involved a substantial number of subjects, selection bias cannot be excluded. This is because the subjects were recruited as part of a larger study of the precursors of Attention Deficit/Hyperactivity Disorder. Parents of children who may be at risk for developing this disorder may have been more willing to participate than do parents of children that are not at risk. This selection bias might explain the somewhat higher prevalence of externalising behavior problems in our study compared to other studies (Lavigne et al., 1998). Also, the response rate (57.5%) and differences between children included and excluded from further analysis might limit the extent to which the results can be generalised to the general population. However, our study is population-based because school attendance is compulsory in the Netherlands for children aged 5 and older and because the health check has a coverage of 98% in this region. With respect to the differences between the included and excluded children, we believe that the results can be considered as a conservative estimate of the actual situation because there were more clinical cases in the excluded group. Thus, if these excluded children had been included in the analyses, the situation might have been even worse.

Second, our study did not include all possible correlates that could influence the relationship between parental education and occupation and child behavior problems. We obtained information from the medical records of the children, and these did not provide substantial information on, for example, family income, the psychiatric state of the parents, and teacher information.

In conclusion, our findings indicate that parental education and occupation are closely and consistently associated with various types of disturbed behavior in 5- to 6-year-old children. There are several correlates of this association, the most important one being maternal age. This may be a confounder rather than an intermediate factor in the above-mentioned association. In the absence of longitudinal information, the causal direction remains unknown though. Still, maternal age contributed strongly to the associations and may thus be important when interventions aimed at reducing socio-economic differences in problem behavior are planned. These findings are of relevance in the continuing search for factors that determine the development of problem behavior and psychopathology, notably antisocial behavior in children. They attract attention to the possibly relevant influence of education and experience and suggest that more studies should be devoted to this topic which is of great importance to society. Controlled studies of psycho-education for young
mothers with a lower education could be planned with the aim of reducing the risk for development of problem behavior.
References


Neighborhood-level and Individual-level SES Effects on Child Problem Behavior: A Multilevel Analysis

Abstract

Objective: This study examined whether neighborhood-level socio-economic variables have an independent effect on reported child behavior problems over and above the effect of individual-level measures of socio-economic status.

Design and setting: Multilevel analysis of cross-sectional survey data relating individual-level child behavioral problems and parental measures of socio-economic status with neighborhood-level measures of socio-economic deprivation in the city of Maastricht, the Netherlands.

Participants: Children born in the years 1990-1991 attending the second grade of normal kindergarten schools in the city of Maastricht. Out of 1417 eligible 5-7 year olds, the parents of 734 children (51.8%) agreed to participate.

Main results: Child behavior problems were more frequent in families of low parental occupation and education (F=14.51, df 3, 721, p<0.001; F=12.20, df 3, 721, p<0.001, respectively) and in families living in deprived neighborhoods (F=13.26, df 2, 722, p<0.001). Multilevel random effects regression analysis showed that the effect of neighborhood-level deprivation remained after adjustment for individual-level socio-economic status (B over three levels of deprivation: 1.36; 95% CI=0.28-2.45).

Conclusions: Living in a more deprived neighborhood is associated with higher levels of child problem behavior, irrespective of individual-level socio-economic status. The additional effect of the neighborhood may be attributable to contextual variables such as the level of social cohesion among residents.

INTRODUCTION

As it is known that behavior problems in children increase the risk for later psychopathology (Campbell, 1995), unravelling the aetiology of early problem behavior may provide possibilities for prevention of adult mental disorder. Many studies have shown that individual-level variables, such as exposure to marital discord or coming from a low-income family, are associated with behavior problems in children (Lavigne et al., 1996; Shaw et al., 1998; Verhulst, Akkermans & Althaus, 1998). In addition, behavior problems occur more frequently in children living in deprived urban areas than in children living in rural communities (Offord et al., 1987; Rutter, 1981). However, whether neighborhood-level socio-economic variables have an independent effect on child behavior problems over and above the effect of individual-level variables has scarcely been studied. Duncan and colleagues (1994) have demonstrated that neighborhood economic conditions and poverty status are powerful correlates of the behavior of children even after accounting for family structure and maternal education. However, most studies on the effects of neighborhood on child behavior have been hampered by the absence of data combining information at the individual, family, and neighborhood levels in the appropriate statistical model (Brooks-Gunn et al., 1993).

Most studies on neighborhood differences on child behavior problems have not taken into account the hierarchical fashion in which such data are organised. Data that are grouped according to neighborhood are, in statistical terms, part of a multilevel structure, with level-one units (individuals) being clustered into level-two units (neighborhoods). Individuals from the same neighborhood are more similar to each other than individuals from different areas, implying that the variation of reported child behavior problems is smaller than if it were completely random. A conventional regression technique cannot take into account the variance components of two different levels, thus underestimating the standard errors of regression coefficients (Hox, 1995). Therefore, multilevel techniques should be used with a two-level hierarchical structure (individual and neighborhood level).

Recently, several studies on adult mental health have used such multilevel techniques. There is now increasing interest in the question to what extent adult mental health outcomes are influenced by neighborhood-level socio-economic variables, over and above the effect of their individual-level counterparts (Driessen, Gunther & Van Os, 1998; Rijeneveld & Schene, 1998; Shouls, Congdon & Curtis, 1996; Van Os et al., 2000). In line with these studies, the goal of the present study is to examine the independent impact of ecological, neighborhood-level variables on the behavior problems in children with multilevel analyses, to give further insight into the pathways of risk. For example, the socio-economic status of deprived neighborhoods, which is associated with a low-level of neighborhood social capital (Kawachi, Kennedy & Glass, 1999) and poor neighborhood social cohesion (Sampson, Raudenbush & Earls, 1997), could contribute to the development of behavior problems in addition to the role of the low socio-economic status of the family. This may have implications for prevention programs aiming to reduce the prevalence of behavioral problems in children.
This report is part of a larger study, the ‘Study of Attention disorders in Maastricht’ (SAM), and involves a prospective cohort study of the precursors of Attention Deficit/Hyperactivity Disorder (ADHD) (Kalff et al., 2001). We used multilevel techniques (Goldstein, 1987) to examine the distribution of parent-reported behavior problems in a large urban community sample of 5-to-7-year-old children living in neighborhoods of varying degrees of deprivation. We hypothesised that behavior problems in children would be affected by contextual neighborhood-level effects over and above the effect of individual-level socio-economic variables.

METHODS

Subjects and procedure
Two data sets were combined, namely, individual characteristics (children from the birth cohort 1990-1991 and their families) from the SAM study (Kalff et al., 2001) and neighborhood characteristics from the Maastricht Mental Health Case Register data base (Driessen et al., 1998). Maastricht is a relatively small city (population 121,000) located in the extreme south of the Netherlands, and is the capital of the province of Limburg. The population is of relative ethnic homogeneity, there being relatively few non-Dutch inhabitants, in comparison with the ethnically more heterogeneous populations in the cities in the Northwest of the country. Only children living within the city limits were included in the present study.

Children were recruited during the basic periodic systemic health examination performed by the Youth Health Care (YHC) in the province of Limburg. All children were in the second grade of normal kindergarten schools and their parents were asked to participate in accordance with the guidelines of the local ethics committee. Demographic information was obtained from the medical records of the YHC. Out of 2290 children in second kindergarten grade, the parents of 1317 children (57.5%) agreed to participate in the SAM study; 973 parents (42.5%) refused participation. For a sample of the non-responders (N=200) demographic information was obtained anonymously from the medical records of the YHC to compare their characteristics with those of the responders. There were no large or significant differences in sex, age, parental education and occupation, marital state, country of birth or living areas. No data on behavior problems were available for the non-responders.

As we included only children living in the city of Maastricht in the present study, our final sample included 734 children, representing 51.8% of the total number of eligible children in the SAM study who were living in the city of Maastricht (n=1417). The sample consisted of 388 boys and 346 girls; the mean age was 66.9 months (sd 4.5, range 57.7 - 83.0). Children were also excluded if they lived in very small neighborhoods, or neighborhoods consisting mainly of industrial estates (N=7); or if one or more of the individual socio-economic variables were missing (N=41).
Dependent variables: Child behavior problems
The parent-reported behavior problems of the children were assessed with the Child Behavior Checklist (CBCL; Achenbach, 1991; Dutch version, Verhulst, Koot & Van der Ende, 1996). This questionnaire consists of 120 items on behavior and emotional problems on a 3-point Likert scale. The questionnaire yields T-scores with a mean of 50 and a standard deviation of 10. A total problem score is computed by summing all the scores. A total problem score of T > 63 is usually considered as clinically deviant behavior (Verhulst et al., 1996). In this study, the continuous total problem score was used rather than the frequently used dichotomization because the latter results in loss of information (Van Os et al., 2000).

In most cases, the mothers completed the CBCL (88.1% mothers; 9.4% fathers; 1.6% both parents; 0.8% unknown). After adjustment for missing data, the CBCL total scores were found to be significantly higher when reported by the mothers than when reported by the fathers (F(2, 725)=3.13; p=.04).

Independent variables: individual level
The educational level and occupational status of the parents were used as indicators of individual-level socio-economic status. The highest level of the mother or father was used. Level of parental education was measured on an 8-point scale ranging from primary education to higher vocational training and university (De Bie, 1987). It was divided a priori into three levels: low (1 and 2), middle (3, 4 and 5), and high (6, 7 and 8). Parental occupation was scored on a 7-point-scale ranging from low skilled to scientific labour (Van den Brand et al., 1990), also divided a priori into three levels: low (1 and 2), middle (3, 4 and 5), and high (6 and 7). Other independent variables were parental marital status (married, divorced, widowed, or lone parents who were neither married, divorced nor widowed) and country of birth of the parents (the Netherlands or another country). Of the 11 lone parents, four were living as a two-parent family and were therefore added to the category ‘married’. The remaining were living as a single-parent family and were added to the ‘divorced’-category. If either one or both of the parents were born in another country, this was registered as ‘foreign-born’.

Independent variables: neighborhood level
In a previous study, the 36 different neighborhoods in Maastricht had been classified using six neighborhood-level socio-economic indicators obtained from the local authority: neighbour-hood-level of unemployment, dependence on social welfare, single-parent families, non-voters, foreign born, and migrations (moving to and from Maastricht) (Driessen et al., 1998). The first principal component of these variables was divided by its tertiles and served as a three-level indicator of neighbour-hood socio-economic deprivation (least, intermediate and most deprived neighborhoods) (Driessen et al., 1998).
**Statistical analyses**

All analyses were performed using STATA, version 6 (StataCorp, 1999). First, the distribution of parent-reported child behavior problems was computed for the individual-level socio-economic variables and for the three neighborhood-levels of deprivation. One-way analysis of variance was performed to examine the effect of these socio-economic status variables on total problem behavior, adjusted for sex, age, and reporter of the CBCL. Tukey multiple comparison procedure was used to analyse group differences. Then, the effect of neighborhood-level of deprivation on total problem behavior was examined. In order to assess whether neighborhood characteristics had an independent effect on total problem behavior over and above the effect of individual socio-economic factors, the measures of individual-level socio-economic variables were added to the multilevel model in addition to neighborhood-level of deprivation. The regression coefficients reflect the adjusted correlations between child behavior problems and neighborhood-level of deprivation. Differences between the neighborhoods on individual-level socio-economic and demographic variables were analysed using the chi-square test. The total problem score of the CBCL was analysed as a continuous dependent variable.

**Key points**

- Behavior problems in children were more frequent in families living in deprived neighborhoods irrespective of individual-level socio-economic status.
- Neighborhood effects may be mediated by contextual influences such as poor social cohesion.
- Intervention programmes for high-risk children should not only focus on individual-level characteristics but also on neighborhood-level characteristics.

**RESULTS**

**Demographic characteristics**

Of the total sample, 235 children (32.0%) lived in least deprived neighborhoods, 231 children (31.5%) in intermediate neighborhoods, and 268 children (36.5%) in most deprived neighborhoods (Table 1). There were no differences between the neighborhoods in sex. Most parents were married (87.7%). There were significant differences between the neighborhoods in marital state ($\chi^2=10.01$, $p=0.04$). Of the divorced parents, 50.6% lived in the more deprived neighborhoods. Most parents were born in the Netherlands (87.5%). The country of birth of the parents differed significantly between the three neighborhoods ($\chi^2=15.26$, $p=0.00$), foreign-born parents living in the more deprived neighborhoods than parents born in the Netherlands.
Table 1. *Demographic characteristics of the sample by three neighborhood-levels (number and percentage (in parenthesis))*

<table>
<thead>
<tr>
<th></th>
<th>Number</th>
<th>Least deprived</th>
<th>Intermediate</th>
<th>Most deprived</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total</strong></td>
<td>734</td>
<td>(100.0)</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Sex</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>boys</td>
<td>388</td>
<td>(52.9)</td>
<td>125 (32.2)</td>
<td>119 (30.7)</td>
</tr>
<tr>
<td>girls</td>
<td>346</td>
<td>(47.1)</td>
<td>110 (31.8)</td>
<td>112 (32.4)</td>
</tr>
<tr>
<td><strong>Marital state</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>married</td>
<td>644</td>
<td>(87.7)</td>
<td>216 (33.5)</td>
<td>205 (31.8)</td>
</tr>
<tr>
<td>divorced</td>
<td>85</td>
<td>(11.6)</td>
<td>17 (20.0)</td>
<td>25 (29.4)</td>
</tr>
<tr>
<td>widowed</td>
<td>5</td>
<td>(0.7)</td>
<td>2 (40.0)</td>
<td>1 (20.0)</td>
</tr>
<tr>
<td><strong>Country of birth of parents</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Netherlands</td>
<td>642</td>
<td>(87.5)</td>
<td>219 (34.1)</td>
<td>204 (31.8)</td>
</tr>
<tr>
<td>foreign-born</td>
<td>92</td>
<td>(12.5)</td>
<td>16 (17.4)</td>
<td>27 (29.3)</td>
</tr>
<tr>
<td><strong>Parental education</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>high</td>
<td>318</td>
<td>(43.3)</td>
<td>136 (42.8)</td>
<td>114 (35.8)</td>
</tr>
<tr>
<td>middle</td>
<td>224</td>
<td>(30.5)</td>
<td>72 (32.1)</td>
<td>66 (29.5)</td>
</tr>
<tr>
<td>low</td>
<td>126</td>
<td>(17.2)</td>
<td>14 (11.1)</td>
<td>42 (33.3)</td>
</tr>
<tr>
<td>unknown</td>
<td>66</td>
<td>(9.0)</td>
<td>13 (19.7)</td>
<td>9 (13.6)</td>
</tr>
<tr>
<td><strong>Parental occupation</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>high</td>
<td>227</td>
<td>(30.9)</td>
<td>112 (49.3)</td>
<td>66 (29.1)</td>
</tr>
<tr>
<td>middle</td>
<td>198</td>
<td>(27.0)</td>
<td>70 (35.4)</td>
<td>78 (39.4)</td>
</tr>
<tr>
<td>low</td>
<td>261</td>
<td>(35.6)</td>
<td>48 (18.4)</td>
<td>77 (29.5)</td>
</tr>
<tr>
<td>unknown</td>
<td>48</td>
<td>(6.5)</td>
<td>5 (10.4)</td>
<td>10 (20.8)</td>
</tr>
</tbody>
</table>

Of the parents with a high level of education, 42.8% lived in the least deprived neighborhoods. In contrast, 55.6% of the parents with a low level of education lived in the most deprived neighborhoods. With regard to occupational level, 49.3% of the parents with the highest occupational level lived in the least deprived neighborhoods, and 52.1% of the parents with lowest occupational level lived in the most deprived neighborhoods. The level of parental education and occupation differed significantly between the neighborhoods ($\chi^2 = 89.94$, $p=0.00$, $\chi^2 = 102.28$, $p=0.00$, respectively).

**Child behavior problems and univariate analyses**

Individual-level and neighborhood-level socio-economic variables were significantly associated with the total problem score of the CBCL after adjustment for sex, age, and reporter of the CBCL (Table 2). Living in a divorced family was significantly associated with parent-reported problem behavior in
children. There were no differences in reported problem behavior between parents born in the Netherlands and parents born elsewhere. Children with parents with a lower level of education and lower level of occupation scored significantly higher on the total problem score than children with parents with a higher level of education and occupation. Also, children living in the most and intermediate deprived neighborhoods had significantly more reported behavior problems than children living in the least deprived neighborhoods.

Table 2. Univariate analyses with individual-level and neighborhood-level socio-economic variables for the total problem scores on the CBCL adjusted for sex, age, and reporter of the CBCL

<table>
<thead>
<tr>
<th>Variable</th>
<th>CBCL total T-score</th>
<th>Post hoc pairwise comparisons</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
</tr>
<tr>
<td><strong>Marital state</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>married (M)</td>
<td>48.77</td>
<td>11.65</td>
</tr>
<tr>
<td>divorced (D)</td>
<td>55.65</td>
<td>13.46</td>
</tr>
<tr>
<td>widowed (W)</td>
<td>42.00</td>
<td>4.84</td>
</tr>
<tr>
<td><strong>Country of birth of parents</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Netherlands</td>
<td>49.25</td>
<td>11.82</td>
</tr>
<tr>
<td>foreign-born</td>
<td>51.47</td>
<td>13.48</td>
</tr>
<tr>
<td><strong>Parental education</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>high (H)</td>
<td>46.79</td>
<td>11.60</td>
</tr>
<tr>
<td>middle (M)</td>
<td>50.54</td>
<td>11.04</td>
</tr>
<tr>
<td>low (L)</td>
<td>53.92</td>
<td>11.90</td>
</tr>
<tr>
<td>unknown (U)</td>
<td>50.88</td>
<td>14.56</td>
</tr>
<tr>
<td><strong>Parental occupation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>high (H)</td>
<td>46.02</td>
<td>11.28</td>
</tr>
<tr>
<td>middle (M)</td>
<td>48.73</td>
<td>11.47</td>
</tr>
<tr>
<td>low (L)</td>
<td>52.27</td>
<td>11.87</td>
</tr>
<tr>
<td>unknown (U)</td>
<td>54.44</td>
<td>13.95</td>
</tr>
<tr>
<td><strong>Level of deprivation</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>least (L)</td>
<td>46.38</td>
<td>12.06</td>
</tr>
<tr>
<td>intermediate (I)</td>
<td>50.30</td>
<td>11.87</td>
</tr>
<tr>
<td>most (M)</td>
<td>51.61</td>
<td>11.68</td>
</tr>
</tbody>
</table>

Note. CBCL=Child Behavior Checklist, ***p<.001; 1 t-test for independent samples
Multilevel analyses

The aggregated measure of neighborhood deprivation could be a reflection of some individual-level variables, such as sex, age, or parental occupation. To examine whether the neighborhood characteristics had an independent effect on children’s behavior problems over and above the individual SES factors, multilevel random effects regression analyses were conducted (Table 3). The coefficients in Table 3 reflect the adjusted effects of these ecological variables of interest.

The results showed that problem behavior in children was associated with neighborhood socio-economic variables (summary linear trend p<.001; Table 3). Children living in the intermediate and most deprived neighborhoods had significantly more behavior problems than children living in the least deprived neighborhoods. After adjustment for sex, age, and reporter of the problem behavior, a significant neighborhood effect remained (summary linear trend p<.001). This effect was attenuated when all individual-level socio-economic variables (sex, age, reporter of the CBCL, marital status, country of birth, parental education and occupation) were added to the multilevel model but remained statistically significant (summary linear trend p=.014).

Table 3. Multilevel random regression analyses with individual (level 1) and neighborhood (level 2) socio-economic status for the total problem scores on the CBCL

<table>
<thead>
<tr>
<th>Neighborhood socio-economic status</th>
<th>Without adjustment</th>
<th>Adjusted for sex/age/reporter</th>
<th>Adjusted for sex/age/reporter CBCL/ marital state/country of birth of parents/parental education and occupation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B (95% CI)</td>
<td>B (95% CI)</td>
<td>B (95% CI)</td>
</tr>
</tbody>
</table>
| least deprived                   | 0*
| intermediate                      | 3.92 (1.77 - 6.07)**
| most deprived                    | 5.23 (3.16 - 7.31)**
| Summary                           | 2.59 (1.55 - 3.63)** |
| linear trend                      | 2.67 (1.63 - 3.71)** |
|                                   | 1.36 (0.28 - 2.45)** |

Note. CBCL=Child Behavior Checklist; B=regression coefficient; 95%CI=95% confidence interval. *baseline; **p < .001; * p < .05

DISCUSSION

The most important finding of the present study is that the neighborhood-level socio-economic variables have an independent effect on reported child behavior problems, over and above the effect of individual-level socio-economic variables. The prevalence of child behavior problems was higher...
in families living in deprived neighborhoods, but this finding could only be partly explained by the individual-level socio-economic measures which are known to be highly correlated with level of neighborhood deprivation. Thus, children of parents with a low level of education and occupation or children of single-parent families have a higher risk of having behavior problems when they live in a deprived neighborhood than when they live in more affluent neighborhoods.

Our results are in line with those of some other multilevel studies of individual and neighborhood differences in adult mental health. Shouls et al. (1996), for example, demonstrated that contextual factors associated with deprived neighborhoods have an effect on reported illness over and above the effect of individual-level socio-economic status. Also, Van Os et al. (2000) showed that neighborhood variables, even after adjustment for their individual-level counterparts, contributed to the onset of psychotic symptoms. In contrast, Reijneveld and Schene (1998) did not find an additional contextual effect on mental disorders over and above the effect of individual-level factors. In this last study, mental disorders were assessed by means of a questionnaire and subsequently dichotomised according to a widely used, but arbitrary, cut-off point. Their results thus possibly remained inconclusive because of loss of information associated with dichotomization of the dependent variable (Van Os et al., 2000).

This study suggests that child behavior problems cannot be explained entirely by individual factors such as low parental education or living in a single-parent family, and that neighborhood factors are also important. This leads to speculation about the nature of neighborhood-level risk factors. Rutter (1981) has demonstrated that factors such as the design of housing estates, personal overcrowding, and migrations (moving to and from a city) which give rise to fewer neighborhood support networks, are associated with a child's behavioral outcome. Also, aspects of the shared family environment influence comorbid behavior problems in children (Gjone & Stevenson, 1997). However, characteristics of the individuals living in the neighborhoods can still bring about these risks. The findings of ecological studies that controlled for individual-level variables suggest that the effect of neighborhood deprivation may be explained as an effect of what is commonly referred to as social capital and social cohesion (Kawachi & Kennedy, 1997; Kawachi, et al., 1999; Sampson et al., 1997). For example, Kawachi et al. (1999) demonstrated that self-rated poor health was associated with living areas with low levels of social capital, characterised by lack of social trust between citizens and few norms of reciprocity, even after adjustment for the individual-level factors. In addition, Sampson et al. (1997) showed that social cohesion among neighbours and collective efforts to maintain control are important factors in explaining variations in crime rates, irrespective of the socio-economic status of the individuals. The studies suggest that the shared social environment, which is not attributable to individual-level factors, may be an important contributor to risk for later social and psychopathological outcomes. Although the term social capital is often used in this context, its precise meaning is not clear, and there are many neighborhood factors that can affect child behavior. For example, differences in child rearing practices, provision of pre-school educational facilities, access to family advice and support, perception of risk of danger leading to children being protected differentially, could all exist at neighborhood level and affect child behavior. Future studies can additionally include these variables to assess their effects. However, the fact that in this study
effects were found for a measure with high loadings on, for example, single-parent families and proportion of foreign-born does suggest that level of social isolation and social cohesion may be involved.

Three points of consideration emerge from the study. First, not all individuals of the cohort participated, as is the case with most epidemiological studies. The response rate was low and this could have biased the findings. However, in order for such bias to explain the findings, one would have to assume that parents of children with few behavior problems were differentially inclined towards non-response in deprived areas, but more likely to respond in non-deprived areas. While not impossible, this scenario is unlikely, and bias-testing between responders and non-responders did not suggest systematic differences. Second, the data were limited to one city, a relatively small city in the Netherlands with a fairly ethnic homogeneous Dutch population. For this reason, our findings may not necessarily be valid for big cities with ethnically more mixed populations. Third, we assessed the effect of neighborhood deprivation at the individual level for three of the variables, namely, the proportion of single-parent families, the level of parental occupation, and the number of foreign-born parents. The other neighborhood variables, such as non-voters or migrations (moving to and from Maastricht), were not available at the individual level. Therefore, it is possible that, for example, individual-level voting behavior caused the additional neighborhood effect. However, as single-parent families and foreign-born loaded highest on the neighborhood deprivation score derived from the principal component analysis (Driessen et al., 1998), we are confident that adjustment for these individual-level variables would have caused the largest possible reduction in effect size.

In conclusion, irrespective of the socio-economic status of the individual, parents report more behavior problems in their children when they live in socially deprived neighborhoods than when they live in more affluent neighborhoods. Many studies have shown that children with behavior problems at a young age are at risk for developing psychopathology later (Campbell, 1995). Verhulst, Van der Ende and Rietbergen (1997) have stressed the need for improved prevention and intervention measures in order to help children cope with the demands imposed by changing society. The fact that deprived neighborhoods may pose an additional risk would imply that prevention programmes for high-risk children should also focus on neighborhood characteristics, in addition to the interventions already provided for high-risk individuals. Further research is needed to determine the pathway of risk associated with the neighborhood environment.
References


Concluding Remarks
The studies described in this thesis investigated the neurocognitive profile of young children at risk of Attention Deficit/Hyperactivity Disorder (ADHD), in order to contribute to the accuracy of the early identification of children at risk of ADHD. In addition, the relation between several demographic factors and behavior problems that are precursors of ADHD was assessed. The studies were part of the *Study of Attention Disorders in Maastricht* (SAM), a large longitudinal research program on ADHD and the role of neurocognitive functions, motor functions, psychosocial factors, and comorbidity in its development. SAM consists of several related projects of which the present thesis is one.

Briefly, the design of SAM is as follows: out of the general population, children were selected on the basis of their scores on the Child Behavior Checklist (CBCL; Verhulst, Koot, & van der Ende, 1996). These selected children underwent a neuropsychological assessment at kindergarten age (5/6 years) and psychiatric information for these children was collected 1.5 years later. The results of four groups of children were compared: children later diagnosed with ADHD, children later diagnosed with so-called 'borderline ADHD' (children displaying all ADHD symptoms but not impaired in two situations as required by DSM-IV (American Psychiatric Association, 1994)), children later diagnosed with other forms of psychopathology (hereafter called pathological controls), and healthy controls. In this chapter, the findings of the neurocognitive studies are drawn together and an attempt is made to evaluate the empirical findings in terms of possible mechanisms and possible clinical applications. In the last part of this chapter, a discussion of demographic factors is presented in relation to behavior problems as determinants of ADHD.

**Neurocognitive profile in young children at risk of ADHD**

Figure 1 depicts the performance scores for 35 measures used in the three experimental studies (chapters 2, 3, 4) converted to z-scores for the diagnostic groups, using the performance of the healthy control children as reference. For the sake of clarity, not all 35 measures are labeled but rather certain cognitive domains are used although it remains difficult to make theoretically acceptable distinctions between different cognitive functions (Lezak, 1995).

Performance decrements in the ADHD children compared with the pathological and healthy control children were most pronounced for neurocognitive tasks measuring visuomotor integration, shifting, and working memory. Similar findings were obtained for variables measuring speed and speed fluctuations in more complex attention and information processing tasks. Group differences were not found for accuracy of information processing. In addition, the ADHD children performed significantly worse on a specific measure of inattention and on motor control tasks with high controlled processing demands that measure the quality of movement. No significant differences were found for simple motor speed measures reflecting the quantity of movement.
Overall, children at risk of ADHD appeared to have normal results on relatively simple tasks that involved only one modality. However, their performance on tasks that involved higher-order cognitive processes and which depended on the control and integration of information was disproportionately worse than that of pathological controls and healthy controls. These findings support the theoretical model formulated by Barkley on the basis of research with older children with ADHD. The findings suggest that ADHD is not a disorder of attention per se, but rather involves a loss of self-control that impairs brain functions crucial for the maintenance and integration of information (Barkley, 1998).

Although our results are consistent with the literature on school-age children with ADHD (De Sonneville, Njokiktjen, & Bos, 1994; Grodzinsky & Barkley, 1999; Kuntsi, Oosterlaan, & Stevenson, 2001; Nigg, Hinshaw, Carte, & Treuting, 1998; Pennington & Ozonoff, 1996; Seidman, Biederman, Faraone, Weber, & Ouellette, 1997), we now demonstrated that the pattern of impairment can be extended to children aged 5-6 years, which is important because at this age children start formal schooling. The neurocognitive impairments may lead to difficulties at school, which in turn may cause learning disabilities or social problems. Thus, our findings of specific cognitive impairments in young children at risk of ADHD suggest the need for neurocognitive assessment at a young age in order to identify children at risk of ADHD in an early stage.

**Specificity of the findings**

An interesting and rather consistent finding was that the abovementioned pattern of impairments in ADHD children could be distinguished from that of pathological control children without ADHD (see Figure 1). Although both groups had a high rate of comorbid diagnoses, which seems to be the rule rather than the exception in ADHD (Kadesjo & Gillberg, 2001), the main
difference between the two groups was the presence of ADHD. Thus, the pattern of impairment seems to be accounted for by ADHD rather than by other psychopathology.

However, it remains interesting to compare the findings of diagnostic groups that are less diverse. Comparison of children with ADHD with children with the related disruptive disorders of oppositional defiant disorders (ODD) and conduct disorder (CD), which have the largest overlap (35-60%) with ADHD, may shed light on whether these disorders are characterized by their own neurocognitive deficits. Data on the neurocognitive measures showed that the performance of visuomotor integration and working memory tasks of children with comorbid ADHD+ODD/CD resembled that of children with 'pure' ADHD, but was significantly poorer than that of children with 'pure' ODD/CD and control children (chapter 2). This suggests that deficits in visuomotor integration and working memory at kindergarten age are associated with ADHD rather than with disruptive behaviors. In addition, we re-analyzed data obtained for attention and information processing tasks (chapters 3 and 4) with the preceding diagnostic groups and found rather specific deficits in motor control and attention on tasks demanding high levels of controlled processing in the 'pure' ADHD group compared with the 'pure' ODD/CD group, the comorbid ADHD+ODD/CD group, and the control group. The performance of the children with comorbid ADHD+ODD/CD was now significantly better than that of children with 'pure' ADHD and was comparable to that of children with 'pure' ODD/CD and control children. Although the findings are rather preliminary and must be interpreted with caution because of the small number of 'pure' ADHD children available in this study (N=9), there is considerable support for the notion that tasks with high processing demands are rather specific for detecting differences between 'pure' ADHD and comorbid ADHD+ODD/CD in children of kindergarten age.

**ADHD: a continuum or categorical?**
The inclusion of a 'borderline ADHD' group made it possible to examine whether ADHD represents a pathological category or a dimension of behavior. According to the DSM system, ADHD is a categorical disorder: an individual either has ADHD or not. However, genetic studies (Levy, Hay, McStephen, Wood, & Waldman, 1997) and various other studies in the field of developmental psychopathology have provided evidence for a dimensional view of ADHD, but there is still no proof of qualitative differences between individuals with ADHD at one end of an extreme and normal controls at the other (Barkley, 1998, p73).

Our results generally confirmed the notion that ADHD is an extreme of a continuum rather than a pathological category (chapters 2, 3, 4). There was a linear effect in most tasks, with ADHD children at one end, healthy controls at the other end and 'borderline ADHD' children in an intermediate position (see Figure 1). Categorical diagnoses are increasingly felt to be at variance with reality. The notion of a continuum has already been formulated with respect to other neurodevelopmental disorders (Bax, 1999), such as dyslexia (Shaywitz, Escobar, Shaywitz, Fletcher, & Makuch, 1992). Furthermore, the intermediate position of the 'borderline ADHD' is interesting, because ADHD and 'borderline ADHD' children were equally symptomatic but differed in that the ADHD symptoms in the last group did not lead to disruption in at least two situations. Apparently,
the behavioral symptoms of ADHD do not necessarily lead to poor performance on neurocognitive tasks. However, it may be that the better neurocognitive abilities of these 'borderline ADHD' children “prevent” their symptoms from leading to disruptions in different situations. Some studies have indeed found better cognitive abilities to protect against the development of ADHD (Barkley, 1998, p.188). Nevertheless, our data do not enable conclusions to be drawn about the direction of these presumed relationships because no psychiatric information of the children was available in the first phase of our study. Thus, it cannot be stated whether better cognitive abilities cause less impairment (for example, at school) or whether less impairment in different settings (as in the 'borderline ADHD' group) results in better cognitive abilities.

The dimensional approach to ADHD has implications for clinical practice. Levy et al. (1997) mentioned that ADHD can be explained in terms of a deviance from an acceptable norm. This, in turn, prompted discussion about when intervention is required. According to Faraone and Biederman (2001), only those individuals at the extreme of the normal variation, which is associated with disability, should be medically treated. However, other forms of intervention, such as psycho-education for parents, may be of value to those in the middle or low end of the continuum. This is because children at this point of the continuum, such as the 'borderline' children, might be vulnerable to develop psychopathology later.

Reduction of variables to interpretable constructs

The various neurocognitive variables that were included in the three experimental studies (chapters 2, 3, and 4) may overlap and perhaps measure the same underlying construct. We therefore decided to perform a post hoc analysis in which a model of inter-relations between the neurocognitive measures was examined. This may provide a more robust estimate of statistically distinct interpretable constructs that could be relevant to clinical practice. All measures were entered in a principal component analysis (PCA) with varimax rotation. Subsequently, we examined whether these constructs were associated with children at risk of ADHD.

The PCA revealed a 10-factor solution with an eigenvalue >1. To facilitate interpretation of the results, we looked at 4, 5, and 6-factor solutions, which accounted for 45.0%, 49.8%, and 54.3% of the variance. After examining the content of the different factors and inspecting the scree-plot with eigenvalues as a function of component values (De Heus, Van der Leeden, & Gazendam, 1995), we used a 5-factor solution to summarize the data. The first factor (eigenvalue 7.7; amount of variance explained 22.0%) appeared to be a factor with high loadings on the reaction times and standard deviations of the reaction time measures of the attention and information processing tasks (loadings ranging from .50 to .81). This factor was termed Speed and Speed Fluctuation of Information Processing. The second factor (eigenvalue 3.5; amount of variance explained 10.1%) was composed of the measures from the pursuit and tracking tasks (loadings ranging from .54 to .76) and was termed Motor Control. The third factor (eigenvalue 2.3; amount of variance explained 6.6%) had mainly high loadings on the error rate measures of the attention tasks, as well as on the behavioral control measure of the sustained attention task to adapt behavior after error feedback (loadings ranging from .52 to .71) and was termed Accuracy and Behavioral Control. The fourth factor (eigenvalue 2.2;
amount of variance explained 6.3%) had high loadings on a number of paper-and-pencil tasks (loadings ranging from .56 to .77) involving complex perceptual ability, shifting, integration of visuomotor abilities, and verbal ability. Most tasks required information to be remembered while the subject acted on it. This factor was termed Working Memory and Executive Functions. The fifth factor (eigenvalue 1.7; amount of variance explained 4.8%) had high loadings on the Purdue pegboard measures (loadings ranging from .67 to .75) and was termed Simple Motor Speed.

The five factors were then used in a MANOVA for comparisons between ADHD, 'borderline ADHD', pathological controls, and healthy controls. The main effect was significant ($F_{(15,846)}=3.30$, $p<.001$). Univariate analyses revealed significant differences on three of the five factors: speed and speed fluctuation of information processing ($F_{(3,284)}=4.47$, $p<.004$), accuracy and behavioral control ($F_{(3,284)}=5.63$, $p<.001$), working memory ($F_{(3,284)}=4.46$, $p<.004$). Comparison of the ADHD, ODD/CD, ADHD+ODD/CD, and healthy control children revealed a significant main effect was also found ($F_{(15,846)}=2.51$, $p<.001$). Significant univariate differences were found for the following three factors: speed and speed fluctuation of information processing ($F_{(3,284)}=3.80$, $p<.011$), motor control ($F_{(3,284)}=4.21$, $p<.006$), working memory ($F_{(3,284)}=3.19$, $p<.024$). The results of the post hoc Tukey tests for both group classifications are summarized in Table 1.

<table>
<thead>
<tr>
<th>ADHD total group</th>
<th>↓ speed and fluctuation</th>
<th>healthy controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>'borderline ADHD'</td>
<td>↓ speed and fluctuation</td>
<td>healthy controls</td>
</tr>
<tr>
<td></td>
<td>↓ working memory and EF</td>
<td>pathological and healthy controls</td>
</tr>
<tr>
<td>pure ADHD</td>
<td>↓ speed and fluctuation</td>
<td>ADHD, pathological and healthy controls</td>
</tr>
<tr>
<td></td>
<td>↓ accuracy and beh. control</td>
<td>controls</td>
</tr>
<tr>
<td></td>
<td>↓ motor control</td>
<td>ODD, ADHD+ODD/CD, controls</td>
</tr>
<tr>
<td>ADHD+ODD/CD</td>
<td>↓ working memory and EF</td>
<td>controls</td>
</tr>
</tbody>
</table>

Note. ↓=worse performance; EF=executive function; beh. control=behavioral control

Thus, speed and fluctuation in the speed of information processing, working memory, and executive functions are important functions when examining young children at risk of ADHD. The factor motor control seems to be even more specific for ADHD because this was the only factor that detected differences between 'pure' ADHD children and the other groups of children. These results support the main findings, which are summarized in Figure 1. Children at risk of ADHD had a poorer performance on the working memory and visuomotor integration tasks than were the control children (second domain in Figure 1) and were slower and were more variable in their processing speed on complex attention and information processing tasks (third domain in Figure 1). Regarding motor control, we found that children at risk of ADHD performed worse on these tasks only when controlled processing was involved (part of fifth domain in Figure 1).
Two points should be borne in mind. First, these results need to be replicated with larger groups, because the number of cases in the pure ADHD group was rather small (N=9) to draw firm conclusions. Second, these results were based on group comparisons, in other words, the differences found may not be apparent in the cognitive profile of an individual.

**Demographic factors and behavior problems**

Substantial support was found for the notion that behavior problems in 5/6-year-old children are associated with a low level of parental education and occupation (chapters 5 and 6). This is a rather consistent finding in the literature, despite different approaches to sample selection and problem definition being used, and parallels the findings for school-age children (Campbell, 1995). By analyzing the contribution of several family correlates to this association between parental education or occupation and child behavior problems, we found that low maternal age was the most important mediator in this association, followed by single parent families (chapter 5).

With more sophisticated multilevel analyses, the independent influence of environment on child behavior problems over and above the influence of individual level measures of socioeconomic status could be analyzed properly. We found that living in a deprived neighborhood was associated with higher levels of child behavior problems, irrespective of the low-level of parental education or occupation or single parent families (chapter 6). This environmental mechanism clearly has a role in the risk of child behavior problems that is not explained by individual mechanisms.

There is a wide range of potential risk factors for the development of behavior problems and psychopathology, ranging from genetic and environmental influences (such as sociocultural factors, parenting, and caregiving factors) to child factors (such as school and peer experience factors). They may all operate in conjunction with each other, which makes it difficult to predict in which children behavior problems will develop and persist. The last decade has seen considerable research performed on the role of genetic factors and this research has revealed that behavior problems are subject to substantial genetic influence (Plomin, 1995). However, genetic differences account for about half of the variance in behavior problems, leaving the other half to be explained by environmental factors (Plomin & Daniels, 1987). Indeed, our results demonstrate that shared environmental factors, not attributable to individual level socioeconomic factors, contribute to child behavior problems (Kalff et al., 2001). In addition, Wichers et al. (2001) have found associations in a large twin study between nonshared environmental factors, influencing only one twin of a pair, and child behavior problems. Also, Gjone and Stevenson (1997) have demonstrated that mainly shared environmental components account for the co-occurrence of externalizing and internalizing behavior problems in twins, though genetic factors were also important, which provides support for there being gene-environment interactions.

Yet, there remains much debate in the literature about the causation of effects. Are the behavior problems or later ADHD a result of the interaction of multiple risk factors or do externalizing behavior problems cause, for example, stress in the family, leading to conflicting parental relations? These questions cannot be answered on the basis of cross-sectional data. Therefore, longitudinal studies are needed, such as those done by Mesman and Koot (2001). These
authors have demonstrated that early adverse family circumstances and parenting characteristics do not contribute to the prediction of later psychopathology after child characteristics (i.e. externalizing and internalizing problems and physical health problems) were taken into account. Moreover, as several individual risk factors as well as the number of risks have been related to behavior problems (Deater-Deckard, Dodge, Bates, & Pettit, 1998) and these, in turn, are precursors of ADHD, a multidimensional approach is necessary when studying the early identification of children at risk of ADHD.

**Future research**
The SAM program is such a multidisciplinary longitudinal research project, which has as aim the improvement of our understanding of the early development of ADHD in order to develop early intervention programs. The studies reported in this thesis comprise only part of the SAM project. In the near future, information for the entire SAM project must be integrated using, for example, structural equation modeling in order to gain a complete overview of the multiple risk factors that are involved in the early development of ADHD. This information could be useful for school doctors at Youth Health Care Centers who are responsible for performing periodic health examinations in children of kindergarten age. They could use the integrated information to improve the accuracy of the identification of children at risk of ADHD. The Health Council of the Netherlands (Gezondheidsraad, 2000) also recommended new instruments should be developed to facilitate the early identification of ADHD, which could lead to more focused early interventions and this, in turn, may prevent further difficulties. Furthermore, the follow-up of the results of SAM may demonstrate the diagnostic continuity of ADHD. Until now, the persistence of ADHD in community samples of young children has not been studied.

Like all studies, SAM has its limitations, which could be improved in future research. First, the study design does not really permit to determine precursors of ADHD since no psychiatric information was available at the beginning of our study. Thus, the design was restricted to determine whether neurocognitive variables measured in phase 2 were associated with a diagnosis in phase 3 and could not predict the development of ADHD. Second, the ADHD diagnosis was solely based on parental reports (ADIKA), whereas it is recommended that multiple informants should be used to increase the accuracy of the diagnosis (Applegate et al., 1997).

**Conclusion**
ADHD is the most prevalent and intensively studied syndrome in the field of developmental psychopathology and carries a poor prognosis. Yet little attention has been paid to its manifestation in early childhood, even though the mean age of onset is between 3 and 4 years. The present thesis focused on the neurocognitive characteristics and demographic variables of young children at risk of ADHD. The results demonstrate that the use of neurocognitive tests can contribute to the accuracy of early identification of children at risk of ADHD and provide information on what problems exist. Neurocognitive deficiencies, in particular deficiencies of higher-order cognitive processes requiring planning, working memory, and motor control output and of controlled information processing
demanding continuous attentional capacity, distinguish between children later diagnosed with ADHD and children with other pathology. These functions in particular are needed to cope with the high levels of information density in daily life and are needed increasingly often in school and society as the child grows older. Furthermore, the results demonstrate that several family and environmental factors are associated with behavior problems at a young age that are precursors of ADHD. Taken together, the results form a relevant starting point for the difficult job of improving the accuracy of the early identification of children at risk of ADHD who are in the phase before they enter formal primary schooling.
References


Summary

Attention Deficit/Hyperactivity Disorder (ADHD) in early childhood has not received much attention, even though the mean age of onset of its behavioral symptoms of inattention, hyperactivity, and impulsivity is between 3 and 4 years, with an upper limit for age-onset of 7 years according to the DSM-IV criteria. This thesis is devoted to the early identification of children at risk of ADHD and is part of a large research program, the so-called Study of Attention disorders in Maastricht (SAM). The first three experimental studies in this thesis deal with the neurocognitive characteristics of young children at risk of ADHD. The few studies of young children with ADHD or at risk of ADHD that have been published focused on behavioral and psychosocial characteristics of the disorder. Hardly any of the published studies focused on the neurocognitive functioning of these young children. The last two experimental studies described in this thesis deal with the relation between demographic factors and behavior problems that are precursors of ADHD.

The following issues were addressed (i) whether the neurocognitive performance of 5/6-year-old children can distinguish between children who are later diagnosed with ADHD or 'borderline ADHD' (children exhibiting all ADHD symptoms but without disruptions in at least two situations) and children not diagnosed with either disorder ('no ADHD'), and whether the performance is specific for ADHD and does not occur in other comorbid disruptive behavior disorders; (ii) whether specific attention deficits; and (iii) specific deficits in controlled processing are uniquely present in children at risk of ADHD compared with children with 'borderline ADHD', children with other psychopathology ('pathological controls'), and healthy controls; (iv) whether child behavior problems are related to parental education/occupation and to what extent correlating factors contribute to this association; and (v) whether neighborhood level socioeconomic variables have an independent effect on child behavior problems over and above the effect of individual level socioeconomic variables.

Chapter 1 gives a detailed overview of these topics and explains the rationale of SAM. The reasons why it is difficult to establish the diagnosis ADHD at early age are discussed. Young children are often active, constantly on the go and into everything, darting back and forth. Their characteristic behavior may include features that are similar to the symptoms of ADHD. It is difficult to state whether this is aberrant behavior or whether it is part of normal behavioral development. In addition, it remains difficult to predict in which children behavior problems will persist and meet diagnostic criteria for ADHD at a later date. Research into the neurocognitive functioning of young children could therefore contribute to the early diagnosis of ADHD and provide information about what difficulties exist. Only a few studies have focused on this topic in young children, in contrast to the large number of studies on the cognitive functioning of school-age children with ADHD. These studies demonstrated cognitive deficits in executive functions, such as response inhibition and working memory, but also deficits in attention and controlled information processing. Whether these deficits are evident at kindergarten age is unclear. Next, a recent theoretical model of ADHD and a concise overview of risk factors that have been related to ADHD are described. A discussion of
certain issues pertinent to the selection of children when studying ADHD, such as the high rate of comorbidity in ADHD and the heterogeneity of the symptoms is provided.

The second part of chapter 1 outlines the content of SAM and the derived studies described in this thesis. SAM is a prospective longitudinal study performed in four phases over 4 years, the first three phases of which are described in this thesis. The main goal of SAM is to examine the developmental profile and risk factors of ADHD in a stage in which most children are not yet clinically diagnosed.

Chapter 2 describes the results of a study on the neurocognitive performance of 5/6-year-old children at risk of ADHD on tasks tapping several aspects of executive functioning, such as shifting response sets, working memory, and visuomotor planning, versus perceptual and language functions, after adjustment for behavioral measures. Two different group designs were used in succession. First, children later diagnosed with ADHD (N=33) were compared with children with 'borderline ADHD' (N=75) and 'no ADHD' children (N=258). To evaluate the effects of comorbid disruptive behavior disorders (Oppositional Defiant Disorder and Conduct Disorder (ODD/CD), the sample was then re-stratified into a four-group design: children later diagnosed with pure ADHD (N=9) were compared with children later diagnosed with ADHD+ODD/CD (N=24), or pure ODD/CD (N=59), and control children without psychopathology (N=274). After the behavioral measures were taken into account, the performance of the children at risk of ADHD was still significantly impaired on measures of working memory and visuomotor integration compared with that of the 'no ADHD' children, while no differences were found on measures of perceptual ability and verbal fluency. The performance of the 'borderline ADHD' children fell in between that of the ADHD and 'no ADHD' children but did not differ significantly from either group. The same pattern of impairment on the working memory and visuomotor planning tasks was seen in the children with comorbid ADHD+ODD/CD but not in the children with pure ODD/CD or the control children, while their performance did not differ significantly from that of the children with pure ADHD. It was concluded that neurocognitive measures could contribute to the early identification of children at risk of ADHD. The deficits found were specific for children at risk of ADHD with comorbid disruptive behavior disorders. Strong support was found for the dimensional view of ADHD with ADHD at one extreme, followed by 'borderline ADHD', and children with 'no ADHD' at the other.

Chapter 3 examines the question whether the attention and information processing profile of young children at risk of ADHD provides clues about which aspects of attention are deficient and which deficits may contribute to the behavioral pattern of ADHD. Baseline speed, impulsivity, and different aspects of attention (sustained, divided, focused) were assessed, using a computerized test battery, in 33 children later diagnosed with ADHD, 75 children later diagnosed with 'borderline ADHD', 122 children later diagnosed with psychopathology other than ADHD, and 133 healthy controls. The most salient finding was that the children at risk of ADHD and 'borderline ADHD' had specific deficits in the performance of attention tasks demanding more controlled processing, in addition to being generally slower on a simple task than the children with other psychopathology or healthy controls.
This suggests that central processing demands may distinguish between the groups. Furthermore, the children at risk of ADHD showed a great fluctuation in task performance, which demonstrates that these children have difficulties sustaining a stable performance. In addition, a specific deficit of inattention was found in children at risk of ADHD while no differences were found on the cognitive measure of impulsivity. As the greatest differences were found on measures of performance fluctuation, it was concluded that a lack of consistent effort might be the underlying problem in ADHD. Furthermore, the results provide strong support for the dimensional view of ADHD with ADHD at one extreme, followed by 'borderline ADHD' and controls at the other.

*Chapter 4* presents the results of a study on the performance of children at risk of ADHD on two computerized complex motor tasks that differ in their level of controlled processing required. In addition, a simple motor task for movement speed was compared with the motor control tasks. The same diagnostic groups were used as in chapter 3. On both complex tasks, children at risk of ADHD were less accurate and more variable in their performance with each hand than were the pathological and healthy control children. The performance of the 'borderline ADHD' children was generally in between that of the ADHD children and the control children. Interestingly, children at risk of ADHD were disproportionately more inaccurate and had a more unstable task performance when they performed tasks requiring high-level processing with their preferred hand. This was not the case when they used their non-preferred hand. This is probably because the non-referred hand is unpracticed in these young children, and thus continuous attention capacity is needed to perform both high-level and low-level controlled processing tasks. No differences between the groups were found on movement speed. The findings were interpreted as evidence for a specific deficit in high-level controlled processing in young children at risk of ADHD. Support was found for the notion that ADHD represents a dimensional trait rather than a pathological category. Results also indicated that it is not quantitative aspects of movement (speed) but rather qualitative aspects (movement control) that discriminate children at risk of ADHD from children with other pathology. These results provide support for the self-regulation theory of Barkley, in which a deficit in self-control is described as the underlying deficit of ADHD.

*Chapter 5* describes the results of a study dealing with the relationship between behavior problems in 5/6-year-old children and parental education/occupation, and investigates the contribution of correlating factors to explain these associations. Different types of behavior (total problem behavior, externalizing behavior, internalizing behavior, and comorbid behavior) derived from a questionnaire were analyzed in a group of 1140 children. Reported problem behaviors in children were significantly associated with low-level parental education/occupation. 'Low maternal age at delivery' explained 29% of the association between problem behavior and parental education, 40% of the association between problem behavior and parental occupation. 'Single-parent families', and to a lesser extent 'living in an urban area' and 'foreign-born parents', also contributed significantly to these associations, whereas the 'number of children in the family' and 'physical illness of the parents' did not contribute to these associations. Several interpretations, varying from psychosocial to more biological
explanations, are discussed with regard to the finding that maternal age was the most important correlate. For example, older mothers with a higher educational/occupational level may have more knowledge and mental ability, resulting in a different approach towards their children such that ADHD becomes manifest less often. Or rearing styles are different as parents are older, because they have gained experience, which also may result in a different approach. Also, the testosterone level, which is higher at young age, may play a key role. High testosterone levels in young pregnant women might influence the child's brain development, leading to more aggressive attitudes.

Chapter 6 deals with the question whether neighborhood level socioeconomic variables have an independent effect on reported problem behavior in children over and above the effect of individual level measures of socioeconomic status, such as those demonstrated in chapter 5. Not only does the socioeconomic status of the individual, but also the socioeconomic status of the neighborhood has an effect on health and mental diseases. Because of the hierarchy of individuals being clustered within neighborhoods, multilevel techniques were used to analyze these data properly. The study sample included 734 children living in the city of Maastricht in the Netherlands. Problem behavior was assessed using a questionnaire on behavior and emotional problems. Results indicated that child behavior problems are associated with living in deprived neighborhoods, irrespective of the individual level measures of socioeconomic status. It is argued that shared social environment that is not attributable to individual level factors, such as social cohesion among neighbors based on trust between citizens or differences in provision of educational facilities, may be an important contributor to risk of later psychopathology.

Chapter 7 combines the findings of the first three experimental studies of this thesis and reviews the results with regard to their specificity and whether ADHD is a continuum or a category. The impaired performance on the working memory and visuomotor integration tasks was related to ADHD but not to ODD/CD. In addition, when higher-order cognitive processes were involved, disproportionate slowing and variability was uniquely seen in the children at risk of ADHD when compared to pathological and healthy control children. Substantial evidence was found for the dimensional approach to ADHD: the ADHD children had the worst performance on most tasks, followed first by the 'borderline ADHD' children and then the control children. It was argued that better neurocognitive abilities might be protective against the manifestation of ADHD.

Chapter 7 also presents the results of a post hoc principal component analysis in which all the neurocognitive measures of the experimental studies (chapter 2, 3, and 4) were included in order to reduce them to interpretable constructs that could be relevant for clinical use. A 5-factor solution was found; these factors were termed (1) 'Speed and speed fluctuation of information processing', (2) 'Motor control', (3) 'Accuracy and behavioral control', (4) 'Working memory and executive functions', and (5) 'Simple motor speed'. Group differences were found for factors 1, 3, and 4, with ADHD and 'borderline ADHD' children having a worse performance than the control children. When comparing pure ADHD with comorbid ADHD+ODD/CD, pure ODD/CD, and control children, group differences were found for factors 1, 2, and 4. The pure ADHD had a worse performance on ‘motor
control’ than the other groups and their performance was poorer on ‘speed and speed fluctuation of information processing’ than that of the control children. The comorbid ADHD+ODD/CD children had a worse performance on ‘working memory and executive functioning’ than the control children. The extent to which these results are in line with the above-mentioned main findings is discussed. It is argued that information processing as well as working memory and executive functions are important functions to examine in the neuropsychological assessment of young children at risk of ADHD.

Last, the associations between individual-level as well as neighborhood-level socioeconomic variables and problem behavior in children are discussed, followed by a concise discussion of risk factors of problem behavior and psychopathology. Because the causation of effects remains unknown, longitudinal studies are needed in the future to elucidate the issue of causality. Other recommendations for future research within the context of the SAM project are given.
Samenvatting

Tot op heden is slechts weinig onderzoek gedaan naar de vroege fasen van Attention Deficit/Hyperactivity Disorder (ADHD), ondanks het feit dat de gemiddelde leeftijd van het ontstaan van de eerste symptomen tussen 3 en 4 jaar ligt. Volgens de DSM-IV classificatie mogen de symptomen zich uiterlijk op 7-jarige leeftijd manifesteren. De eerste drie onderzoeken die in dit proefschrift worden beschreven, handelen over de neurocognitieve functies en vaardigheden bij jonge kinderen met een verhoogd risico op ADHD. De enkele studies die tot nu toe zijn uitgevoerd naar vroege fasen van ADHD hebben zich voornamelijk gericht op de gedragskenmerken en psychosociale eigenschappen en niet op het neurocognitief functioneren. In de overige twee studies in dit proefschrift wordt de samenhang van enkele demografische factoren met gedragsproblemen als voorloper van ADHD onderzocht om de etiologie te verhelderen. De experimentele studies zijn een onderdeel van een grootschalig onderzoeksprogramma, de zogeheten Study of Attention disorders in Maastricht (SAM).

De volgende vraagstellingen komen aan de orde: (i) is het mogelijk om op grond van de neurocognitieve prestaties van 5/6-jarige kinderen een onderscheid te maken tussen de volgende drie groepen: kinderen die op latere leeftijd gediagnostiseerd worden met ADHD, kinderen die later gediagnostiseerd worden met 'borderline ADHD' (kinderen die weliswaar alle ADHD symptomen hebben maar dit slechts in een enkele context vertonen, bijvoorbeeld alleen thuis, of alleen op school) en kinderen zonder ADHD, en zijn deze prestaties specifiek voor ADHD in vergelijking met comorbide gedragsstoornissen, (ii) zijn er specifieke aandachtsstoornissen in kinderen met een verhoogd risico op ADHD vergeleken met 'borderline ADHD' kinderen, kinderen met andere vormen van pathologie zonder ADHD en gezonde controle kinderen, (iii) zijn er specifieke stoornissen in gecontroleerde informatieverwerking in dezelfde groepen genoemd bij tweede vraagstelling, (iv) zijn gedragsproblemen bij kinderen gerelateerd aan het opleidings-/beroepsniveau van ouders en welke samenhangende factoren dragen bij aan deze associatie, (v) heeft de woonomgeving cq. het 'sociaal milieu' een onafhankelijk effect op gedragsproblemen bij kinderen dat niet verklaard wordt door de individuele sociaal-economische variabelen zoals opleidings-/beroepsniveau van ouders.

In hoofdstuk 1 wordt een beknopt overzicht gegeven van de onderwerpen die in dit proefschrift aan de orde komen. Tevens worden de bewegredenen van de Study of Attention disorders in Maastricht (SAM) uiteengezet. Er wordt ingegaan op de vraag waarom het moeilijk is om op jonge leeftijd een diagnose ADHD te stellen. Kleuters vertonen vaak druk en uitbundig gedrag hetgeen kenmerken zijn van ADHD. Het is niet gemakkelijk om te bepalen of dit gedrag tot het normale ontwikkelingspatroon behoort of dat het pathologisch is. Bovendien is het tot op heden niet duidelijk bij welke kinderen deze gedragsproblemen persisteren en derhalve op latere leeftijd voldoen aan de criteria van ADHD. Onderzoek naar het neurocognitief functioneren bij jonge kinderen zou een waardevolle bijdrage kunnen leveren aan vroegdiagnostiek van ADHD en daardoor duidelijkheid kunnen verschaffen in de aard van de problematiek. Er zijn slechts weinig studies over cognitieve
vaardigheden bij jonge kinderen. Daarentegen is bij schoolgaande ADHD kinderen veel onderzoek gedaan. Deze studies tonen verschillende cognitieve stoornissen aan; o.a. in de zogenaamde 'executieve functies', die zijn gekenmerkt door deel-functies zoals het inhibitievermogen en het werkgeheugen, maar ook in de aandacht en gecontroleerde informatieverwerking. Hierna volgt een beschrijving van een recent theoretisch model van ADHD en een korte inleiding op de risicofactoren voor ADHD. Vervolgens worden enkele onderwerpen besproken die van belang zijn bij het wetenschappelijk onderzoek naar ADHD, zoals het veel voorkomen van comorbiditeit en de diversiteit van de symptomen in verschillende situaties.

In het tweede deel van hoofdstuk 1 wordt de methodologie van SAM besproken gevolgd door een beschrijving van de verschillende studies in dit proefschrift. SAM is een prospectief longitudinaal onderzoek dat vier jaar in beslag heeft genomen waarvan de resultaten uit de eerste drie, van in totaal vier, fasen in dit proefschrift behandeld worden. Het voornaamste doel van SAM is de bestudering van het ontwikkelingsprofiel en de risicofactoren van ADHD in een stadium waarin bij de meeste kinderen nog geen klinische diagnose is vastgesteld.

**Hoofdstuk 2** beschrijft de resultaten van een studie naar de neurocognitieve prestaties bij 5/6-jarige kinderen met een verhoogd risico op ADHD. Door middel van executieve taken werden ondermeer cognitieve flexibiliteit, werkgeheugen en visuo-motorische planning gemeten. Tevens werden perceptuele en verbale taken afgenomen. Er werd gecontroleerd op verschillen in gedrag. In eerste instantie werden drie groepen vergeleken: kinderen met een risico op ADHD (N=33), kinderen met een risico op 'borderline ADHD' (N=75) en kinderen zonder ADHD (N=258). Daarna werden vier nieuwe groepen gevormd om de invloed van comorbide gedragsstoornissen (Oppositional Defiant Disorder en Conduct Disorder (ODD/CD) te onderzoeken: kinderen met risico op pure ADHD (N=9), kinderen met risico op ADHD+ODD/CD (N=24), kinderen met risico op pure ODD/CD (N=59), en controle kinderen zonder deze vormen van pathologie (N=274). Na controle voor gedragsverschillen waren de prestaties van de ADHD kinderen significant slechter op taken die werkgeheugen en visuo-motorische integratie meten vergeleken met kinderen zonder ADHD. Er waren geen groepsverschillen in de perceptuele en verbale taken. De prestaties van de 'borderline ADHD' kinderen lagen tussen die van kinderen met en zonder ADHD maar deze verschillen waren niet significant. Vergelijkbare zwakke prestaties op taken van werkgeheugen en visueel-motorische integratie werden gevonden bij de kinderen met ADHD+ODD/CD vergeleken met kinderen met pure ODD/CD en controle kinderen. Hun prestaties verschillen niet significant van de kinderen met pure ADHD. Er werd geconcludeerd dat neurocognitief onderzoek een belangrijke bijdrage kan leveren aan de vroegdiagnostiek van kinderen met een risico op ADHD. Tevens werd aangetoond dat het cognitief profiel specifiek was voor kinderen met ADHD+ODD/CD. Er werd bewijs gevonden voor de stelling dat ADHD verloopt langs een glijdende schaal met ADHD als ene uiterste, gevolgd door kinderen met 'borderline ADHD' en kinderen zonder ADHD als andere uiterste.

**Hoofdstuk 3** beschrijft een onderzoek naar het aandachts- en informatieverwerkingsprofiel in de vroege ontwikkeling van kinderen met een risico op ADHD. Diverse computertaken waaronder een
een eenvoudige snelheidstaak, een impulsiviteitstaak en volgehouden, verdeelde en gerichte aandachtstaken werden afgenomen. De prestaties van vier groepen kinderen werden vergeleken: 33 kinderen die op latere leeftijd ADHD bleken te hebben, 75 kinderen met 'borderline ADHD', 122 kinderen met andere vormen van psychopathologie dan ADHD en 133 gezonde controle kinderen. De belangrijkste bevinding was dat de ADHD en 'borderline ADHD' kinderen specifieke aandachtstoornissen hadden indien gecontroleerde informatieverwerking werd vereist. Deze stoornis was disproportioneel ten opzichte van de algemene traagheid op de eenvoudige snelheidstaak. Daarnaast werd in alle taken bij de ADHD kinderen een grotere variabiliteit gevonden in de snelheid van reageren. Dit betekent dat zij moeite hebben met het behouden van een gelijkmatig prestatie niveau. Bovendien werd een specifieke stoornis gevonden bij de ADHD kinderen in de mate van oplettendheid terwijl er geen verschil werd gevonden in impulsiviteit. Aangezien de grootste verschillen werden gevonden in variabiliteit, werd geconcludeerd dat een tekort aan continue inspanning het probleem is dat ten grondslag ligt aan ADHD. Tevens ondersteunen de resultaten de opvatting dat ADHD verloopt langs een glijdende schaal met ADHD aan de ene kant en gezonde kinderen aan de andere kant.

_Hoofdstuk 4_ richt zich op verschillen in gecontroleerde informatieverwerking in kinderen met een risico op ADHD. Twee computer-gestuurde complexe motorische taken die in verschillende mate een beroep doen op gecontroleerde informatieverwerking werden afgenomen bij dezelfde diagnostische groepen als in hoofdstuk 3. Tevens werd een eenvoudige taak voor motorische snelheid afgenomen. ADHD kinderen waren minder nauwkeurig en vertoonden een grotere variabiliteit in uitvoering van de beide complexe taken vergeleken met pathologische en gezonde controle kinderen. Dit gold zowel voor de prestatie met de voorkeurshand als voor de niet-voorkeurshand. De prestaties van de 'borderline ADHD' kinderen lag tussen die van de ADHD en gezonde controle kinderen in. Tevens werd bij de taak die een groot beroep doet op gecontroleerde informatieverwerking, een disproportioneel verschil gevonden bij de ADHD kinderen in nauwkeurigheid en variabiliteit van de bewegingen met de voorkeurshand. Er werden geen disproportionele resultaten gevonden met de niet-voorkeurshand. Dit kan mogelijk worden verklaard doordat jonge kinderen deze hand nog weinig gebruiken. Daardoor vereisen beide taken continue aandacht en is dus in hoge mate gecontroleerde informatieverwerking nodig. Er waren geen verschillen tussen de groepen in bewegingssnelheid. De resultaten vormen bewijs voor het bestaan van een specifieke stoornis in informatieverwerking onder gecontroleerde condities in jonge kinderen met een risico op ADHD. Tevens ondersteunen de gegevens de opvatting dat ADHD een continuum is. Daarnaast is aangetoond dat niet zozeer snelheid van bewegen (kwantitatieve maat) maar eerder het controleren van de bewegingen (kwalitatieve maat) discrimineert tussen kinderen met een risico op ADHD en kinderen met andere pathologie. De bevindingen sluiten aan bij de zelf-regulatie theorie van Barkley die stelt dat een stoornis in de zelf-controle ten grondslag ligt aan ADHD.

_Hoofdstuk 5_ presenteert een studie die de relatie tussen gedragsproblemen in 5/6-jarige kinderen en opleidings- en beroepsniveau van ouders onderzocht, alsmede samenhangende factoren die deze
relatie zou kunnen verklaren. Aan de hand van een vragenlijst die bij 1140 kinderen werd afgenomen werd vier soorten probleemgedrag (totaal probleemgedrag, externaliserend, internaliserend en comorbide gedrag) bepaald. Gedragsproblemen bij kinderen waren significant geassocieerd met lage opleidings- en beroepsniveaus van hun ouders. Deze associaties werden respectievelijk voor 29% en 40% verklaard door de factor 'leeftijd van moeder bij de geboorte': jonge moeders hadden significant vaker een kind met ADHD. Bovengenoemde associaties werden ook verklaard door 'één-ouder gezinnen' en in iets mindere mate ook door 'wonen in een stad' en 'ouders die in het buitenland geboren zijn'. Het 'aantal kinderen in een gezin' en de 'fysieke gesteldheid van ouders' droegen niet bij aan de associaties. Verschillende verklaringen, variërend van psychosociaal tot biologisch, werden gegeven voor het feit dat de leeftijd van de moeder de belangrijkste mediërende factor was. Oudere moeders met een hoger opleidings- en/of beroepsniveau beschikken over meer kennis en intellectuele vermogens hetgeen kan resulteren in een andere aanpak van het kind waardoor ADHD minder tot uiting komt. Een andere mogelijkheid is dat door toegenomen ervaring de opvoedingsstijl anders is in oudere moeders hetgeen eveneens zorgt voor een andere aanpak. Tot slot, werd aangestipt dat het testosterongehalte een rol kan spelen aangezien dit hoger is op jongere leeftijd. Als een jonge vrouw zwanger is, zou een hoog testosterongehalte mogelijk de ontwikkeling van de hersenen van het ongeboren kind kunnen beïnvloeden hetgeen een belangrijke factor is die leidt tot meer agressief gedrag.

Hoofdstuk 6 beschrijft een onderzoek naar het effect van ‘woonomgeving’ op gedragsproblemen bij kinderen dat niet verklaard wordt door de individuele sociaal-economische status, zoals aangetoond in hoofdstuk 5. Uit onderzoek is gebleken dat niet alleen de sociaal-economische status van het individu, maar ook de sociaal-economische woonomgeving een effect kan hebben op gezondheid en ziekte. Vanwege de hierarchische structuur van de gegevens waarbij individuen geclusterd zijn in buurten werd gebruik gemaakt van multilevel technieken om de data op de juiste wijze te analyseren. Bij een groep van 734 kinderen uit Maastricht werd probleemgedrag bepaald aan de hand van een vragenlijst. De resultaten laten zien dat gedragsproblemen bij kinderen geassocieerd waren met wonen in sociaal zwakke buurten, onafhankelijk van de sociaal-economische status van de ouders. Er wordt gesuggereerd dat eenzelfde sociale omgeving, zoals sociale samenhang tussen buren gebaseerd op onderling vertrouwen of verschillen in onderwijs faciliteiten, een belangrijke bijdrage kan leveren aan het risico op het ontwikkelen van psychopathologie.

Hoofdstuk 7 voegt de resultaten van de experimentele studies van dit proefschrift samen. De zwakke prestaties bij werkefficiëntie en visuo-motorische integratie taken bleken geassocieerd met ADHD maar niet met ODD/CD. Wanneer er sprake was van hogere orde cognitieve processen bleken de kinderen met een risico op ADHD disproportioneel trager te zijn. Bovendien vertoonden zij een disproportioneel grotere variabiliteit in snelheid van reageren ten opzichte van pathologische en gezonde controle kinderen. Sterke aanwijzingen werden gevonden dat ADHD moet worden opgevat als een continuum: ADHD kinderen presteerden het zwakst op vrijwel alle neurocognitieve taken,
gevolgd door de 'borderline ADHD' en tenslotte de gezonde controle kinderen. Betoogd wordt dat door goede neurocognitieve vaardigheden ADHD minder tot uiting komt.

Vervolgens beschrijft hoofdstuk 7 de resultaten van een principale componenten analyse waarin het aantal onderzochte neurocognitieve variabelen worden gereduceerd tot interpreteerbare factoren. Daarna werd gekeken of deze factoren geassocieerd konden worden met ADHD om tevens de empirische bevindingen te vertalen voor de klinische praktijk. Er werden 5 factoren onderscheiden, te weten (1) 'Snelheid en variabiliteit in snelheid van informatieverwerking', (2) 'Controle van de motoriek', (3) 'Nauwkeurigheid en controle van het gedrag', (4) 'Werkgeheugen en executieve functioneren', en (5) 'Simpele motorische snelheid'. Er werden groepsverschillen gevonden bij factoren 1, 3 en 4, waarbij ADHD en/of 'borderline ADHD' kinderen slechter presteerden dan de controle kinderen. Na vergelijking van pure ADHD kinderen met kinderen met ADHD+ODD/CD, pure ODD/CD, en controle kinderen werden eveneens groepsverschillen gevonden bij factoren 1, 2 en 4. Kinderen met pure ADHD presteerden slechter bij 'controle van de motoriek' dan de andere groepen en presteerden eveneens slechter bij 'snelheid en variabiliteit in snelheid van informatieverwerking' dan de controle kinderen. Kinderen met ADHD+ODD/CD presteerden slechter dan de controle kinderen bij 'werkgeheugen en executief functioneren'. Er werd ingegaan in welke mate deze resultaten overeen komen met de hierboven beschreven algemene bevindingen. Verder werd aangegeven dat informatieverwerking alsmede werkgeheugen en executieve functies belangrijke functies zijn in een neuropsychologisch onderzoek bij jonge kinderen met een verhoogd risico op ADHD.

Tenslotte worden de associaties tussen zowel individuele als buurt sociaal-economische variabelen en gedragsproblemen bij kinderen besproken. Hierop volgt een discussie over risicofactoren en hun mogelijk belang. Gesteld wordt dat op dit moment nog onduidelijk is of de risicofactoren het probleemgedrag en psychopathologie veroorzaken of andersom. Om deze causaliteitsvraag te kunnen beantwoorden zijn longitudinale studies nodig. De SAM studie is een dergelijk longitudinaal onderzoek waarvan dit proefschrift de eerste fasen beschrijft. Vervolgonderzoek van de kinderen die in dit onderzoek zijn bestudeerd zal in de nabije toekomst mogelijk een betere uitspraak over de causaliteit kunnen opleveren. Verdere suggesties worden gedaan voor toekomstig onderzoek zowel binnen als buiten het SAM-project.