

**Sensitivity to reward frequency and reward delay in children with
Attention-Deficit/Hyperactivity Disorder**

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Abstract

ADHD is a commonly diagnosed disorder of children, and is characterized by difficulties with inattention and/or hyperactivity/impulsivity. Some theories and models of ADHD have noted that children with ADHD respond differently from controls to rewards. The two studies in this thesis used response bias ($\log b$, a measure derived from signal detection theory) during a computerized detection task to examine the response to rewards in children with ADHD. The participants were children aged between 6 and 12 years of age. One hundred and fifty two children were recruited from Dunedin schools, and 136 children were referred by the Otago District Health Board for assessment of ADHD related difficulties. From these two groups of children 68 boys with combined type ADHD (ADHD-C) and 91 normally developing boys were identified. Both studies used a similar computer task to present stimuli and measure responses. A matrix of computerized characters was presented on a computer screen. Participants were asked to identify whether there were more red or more blue characters present. Correct responses were occasionally rewarded with an on-screen animated cartoon, verbal praise from the examiner, and tokens which were exchanged for a small gift at the end of the task. Study 1 examined sensitivity to reward frequency, and generated response bias by arranging more frequent rewards for correct responses to one stimulus than the other stimulus. Study 1 found that boys with ADHD-C developed bias towards more frequently rewarded stimuli more slowly than normally developing boys – this was particularly apparent towards the end of the task. The results were also consistent with previous literature identifying that children with ADHD show reduced bias towards frequent rewards following individual rewards to the infrequently rewarded stimulus. Study 2 examined sensitivity to reward delay, and was designed to generate response bias by arranging immediate rewards for correct responses to one stimulus and delayed rewards for correct identifications of the other

stimulus. In Study 2 there were no clear differences between groups. The interpretation of both experiments was confounded by confounding variables: task version (in Study 1); and task order (in Study 2). An unintended but interesting finding is that children with ADHD-C may be more influenced by their past history of reward than control children. Children with ADHD-C who completed Study 2 after Study 1 tended to continue to show bias towards the response that had been rewarded frequently in the previous task, despite the same response being associated with delayed rewards in the current task. Children with ADHD-C may be less able than normally developing children to reverse their preference in response to altered reward contingencies. The results of the current experiments are discussed in relation to the broader literature on ADHD and rewards/reinforcement and current theories of ADHD.

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Contents

ABSTRACT	II
ACKNOWLEDGEMENTS	IV
LIST OF FIGURES	VII
LIST OF TABLES	XI
CHAPTER 1: ATTENTION-DEFICIT/HYPERACTIVITY DISORDER. INTRODUCTION, EPIDEMIOLOGY, COMORBIDITY, AND AETIOLOGY	1
CHAPTER 2: BEHAVIOURAL ANALYSIS AND THEORIES OF ADHD.....	30
CHAPTER 3: MAIN METHOD	58
CHAPTER 4: SENSITIVITY TO REWARD FREQUENCY METHOD AND RESULTS...	73
CHAPTER 5: SENSITIVITY TO REWARD FREQUENCY DISCUSSION	91
CHAPTER 6: SENSITIVITY TO REWARD DELAY METHOD AND RESULTS	101
CHAPTER 7: SENSITIVITY TO REWARD DELAY DISCUSSION.....	120
CHAPTER 8: GENERAL DISCUSSION.....	129
REFERENCES	148
APPENDICES	188
Appendix A: Participant Information and Consent Forms	188
Appendix B: Statistical Tables from Study 1 – Sensitivity to Reward Frequency.....	208
Appendix C: Statistical Tables from Study 2 – Sensitivity to Reward Delay	221

List of Figures

Figure 1. A typical stimulus array containing more blue than red characters.	60
Figure 2. Possible correct and incorrect responses for each stimulus type.	67
Figure 3. Mean bias ($\log b$) for combined type ADHD (ADHD-C) and normally developing (ND) groups over blocks of 60 trials by Version. For Version 1, more frequent rewards were provided for correct 'more blue' (right button) responses; the reverse was true for Version 2. Because there was a main effect of Version, $\log b$ is plotted separately for each version.	81
Figure 4. Mean bias ($\log b$) for the ADHD-C and ND Groups calculated within moving windows of 20 trials over the whole task.	83
Figure 5. Bias ($\log b$) toward the more frequent reward calculated for trials following the 'Rich' reward and 'Lean' reward for combined type ADHD (ADHD-C) and ND groups.	84
Figure 6. Discriminability ($\log d$) for combined type ADHD (ADHD-C) and normally developing (ND) groups over the final two blocks of 60 trials. Because the ANOVAs did not identify effects of version or order, both task versions and task orders are combined.	86
Figure 7. Mean response times in milliseconds for combined type ADHD (ADHD-C) and normally developing (ND) groups over the final two blocks of 60 trials.	87
Figure 8. $\log b$ is plotted for the combined type ADHD (ADHD-C), predominantly inattentive ADHD (ADHD-PIA), and normally developing (ND) groups. Because earlier analyses identified effects of Order and Version, these are plotted separately.	89
Figure 9. $\log b$ towards frequent rewards is plotted for responses following frequent (rich) and infrequent (lean) rewards, for the combined type ADHD (ADHD-C), predominantly	

inattentive ADHD (ADHD-PIA), and normally developing (ND) groups. Because earlier analyses identified an effect of version, each version is plotted separately.	90
Figure 10. Mean Bias ($\log b$) for ADHD-C and normally developing (ND) groups over blocks of 60 trials. Because there was a main effect of Order, $\log b$ values are plotted separately for each Order. The left panel includes participants who completed the current task before the Study 1 task (Order 1). The right panel includes participants who completed the Study 1 task before the current task (Order 2).....	108
Figure 11. Mean response bias ($\log b$) for combined type ADHD (ADHD-C) and normally developing (ND) groups calculated within moving windows of 20 trials over the whole task. Participants that received Order 1 are plotted separately from those that received Order 2.....	110
Figure 12. Response bias ($\log b$) towards immediate reward calculated for trials following immediate reward and delayed reward for combined type ADHD (ADHD-C) and normally developing (ND) groups. Because ANOVAs revealed effects of Order and Version each Order-Version combination is plotted separately.	111
Figure 13. Discriminability ($\log d$) for combined type ADHD (ADHD-C) and normally developing (ND) groups over final block of 60 trials, by Order.	114
Figure 14. Mean response time (RT) for combined type ADHD (ADHD-C) and normally developing (ND) groups over final block of 60 trials. Because there were no main or interaction effects, both orders and versions have been combined.	116
Figure 15. Response bias ($\log b$) is plotted for the combined type ADHD (ADHD-C), predominantly inattentive ADHD (ADHD-PIA), and normally developing (ND) groups, over each block of 60 trials. Because earlier analysis identified an effect of order, each order is plotted separately.....	118

Figure 16. Log b towards immediate rewards is plotted for responses following immediate and delayed rewards for the combined type ADHD (ADHD-C), predominantly inattentive ADHD (ADHD-PIA), and normally developing (ND) groups. Because earlier analyses identified an effect of Order and Version, each Order-Version combination is plotted separately. 119

Figure 17. Hypothetical delay of reinforcement gradient for rewards of two amounts. The gradients are based on the hyperbolic formula (3) from Green et al., (1994). The value of the larger reward is marked as A on the y-axis. The value of the smaller reward is marked as B on the y-axis. 125

Figure 18. Left Panels: Preference reversal occurring in normally developing (ND) and combined type ADHD (ADHD-C) participants who completed Study 1 before Study 2 by task version. Preference reversal was calculated by adding the bias score for each participant within the final block of Study 1 to the bias scores within the first block of Study 2. Right panels: A comparison with participants who completed Study 2 before Study 1 by task version. Scores were calculated by adding the bias score for each participant within the final block of Study 1 to the bias scores within the first block of Study 2. F1 and F2 are version 1 and version 2 of Study 1. D1 and D2 are version 1 and version 2 of Study 2. 133

Figure 19. Left Panels: Preference reversal occurring in normally developing (ND) and combined type ADHD (ADHD-C) participants who completed Study 2 before Study 1 by task version. Preference reversal was calculated by adding the bias score for each participant within the final block of Study 2 to the bias scores within the first block of Study 1. Right panels: A comparison with participants who completed Study 1 before Study 2 by task version. Scores were calculated by adding the bias score for each

participant within the final block of Study 2 to the bias scores within the first block of
Study 1. F1 and F2 are version 1 and version 2 of Study 1. D1 and D2 are version 1 and
version 2 of Study 2..... 135

List of Tables

Table 1.	59
Clinical, Cognitive, and Demographic Characteristics.	
Table 2.	79
Age and Estimated IQ (ESTIQ) for combined type ADHD (ADHD-C), predominantly inattentive ADHD (ADHD-PIA), and normally developing (ND) Groups. Ranges are in Parentheses.	
Table 3.	106
Mean age in years and estimated IQ (ESTIQ) for combined type ADHD (ADHD-C), inattentive ADHD (ADHD-PIA), and normally developing (ND) groups. Ranges are in parentheses.	
Table 4.	208
Mean response bias ($\log b$), standard deviation, and number of participants for normally developing (ND), combined type ADHD (ADHD-C), and inattentive ADHD (ADHD- PIA) groups. Means are provided separately for each block of trials, task version, and order.	
Table 5.	209
Descriptive statistics for response bias ($\log b$), following frequent ('Rich') reward and infrequent ('Lean') reward, for normally developing (ND), combined type ADHD (ADHD-C), and inattentive ADHD (ADHD-PIA) groups. Statistics are provided separately for each reward type, version, and order.	
Table 6.	210
Mean discriminability ($\log d$), standard deviation, and number of participants for normally developing (ND), combined type ADHD (ADHD-C), and inattentive ADHD (ADHD-	

PIA) groups. Means are provided separately for each block of trials, task version, and order.

Table 7.....	211
--------------	-----

Mean response time, standard deviation, and number of participants for normally developing (ND), combined type ADHD (ADHD-C), and inattentive ADHD (ADHD-PIA) groups.

Means are provided separately for each block of trials, task version, and order.

Table 8.....	212
--------------	-----

Response bias ($\log b$): Mixed Design ANOVA Table. Between subjects factors are Group, Version, and Order. The within subjects factor is Block.

Table 9.....	213
--------------	-----

Response bias ($\log b$): Tests of within-subjects contrasts. Between-subjects factors are Group, Version, and Order. The within-subjects factor is Block.

Table 10.....	214
---------------	-----

Response bias ($\log b$): Univariate ANOVA Table. Between subjects factors are Group, Version, and Order. Separate tests are provided within each block of trials.

Table 11.....	215
---------------	-----

Response bias ($\log b$) following frequent ('Rich') and infrequent ('Lean') rewards: Mixed Design ANOVA Table. Between subjects factors are Group, Version, and Order. The within subjects factor is RewardType.

Table 12.....	216
---------------	-----

Discriminability ($\log d$): Multivariate ANOVA Table. Mixed Design. Between-subjects factors are Group, Version, and Order. The within-subjects factor is Block.

Table 13.....	217
---------------	-----

Discriminability ($\log d$): Univariate ANOVA Table. Between-subjects factors are Group, Version, and Order.

Table 14. 218

Response time (RT): Multivariate ANOVA Table. Mixed Design. Between-subjects factors are Group, Version, and Order. The within-subjects factor is Block.

Table 15. 219

T-tests (one sample) for difference between $\log b$ and zero across trial blocks for combined type ADHD (ADHD-C) and normally developing (ND) groups, by Version and Block.

Table 16. 219

Pearson product moment correlations between independent (age and estimated IQ score) and dependent ($\log b$, $\log d$, and response time) variables within the combined type ADHD (ADHD-C) and normally developing (ND) groups.

Table 17. 220

Pearson product moment correlations between signal detection variables ($\log b$, $\log d$, and response time) for combined type ADHD (ADHD-C) and normally developing (ND) groups. Correlations between variables within the same trial block are underlined and in bold text.

Table 18. 221

Mean response bias ($\log b$), standard deviation, and number of participants for normally developing (ND), combined type ADHD (ADHD-C), and inattentive ADHD (ADHD-PIA) groups. Means are provided separately for each block of trials, task version, and order.

Table 19. 222

Descriptive statistics for response bias ($\log b$), following immediate reward and delayed reward, for normally developing (ND), combined type ADHD (ADHD-C), and inattentive ADHD (ADHD-PIA) groups. Statistics are provided separately for each reward type, task version, and order.

Table 20. 223

Mean discriminability ($\log d$), standard deviation, and number of participants for normally developing (ND), combined type ADHD (ADHD-C), and inattentive ADHD (ADHD-PIA) groups. Means are provided separately for each block of trials, task version, and order.

Table 21. 224

Mean response time, standard deviation, and number of participants for normally developing (ND), combined type ADHD (ADHD-C), and inattentive ADHD (ADHD-PIA) groups. Means are provided separately for each block of trials, version, and order.

Table 22. 225

Response bias ($\log b$): Mixed Design ANOVA Table. Between subjects factors are Group, Version, and Order. The within subjects factor is Block.

Table 23. 226

Response bias ($\log b$): Univariate ANOVA Table. Between subjects factors are Group, Version, and Order. Separate tests are provided within each block of trials.

Table 24. 226

Response bias ($\log b$): Univariate ANOVA Table for Participants who completed the task in Order 2. Between subjects factors are Group and Version. Separate tests are provided within each block of trials.

Table 25. 227

Response bias ($\log b$) following immediate and delayed rewards: Mixed Design ANOVA

Table. Between subjects factors are Group, Version, and Order. The within subjects factor is RewardType.

Table 26. 228

Response bias ($\log b$) following immediate and delayed rewards: Mixed Design ANOVA

Table. Between subjects factors are Group and Version. The within subjects factor is RewardType.

Table 27. 228

Test of the effect of RewardType on response bias ($\log b$): RewardType is the within-subjects variable. The effect of RewardType is tested separately within each Group, Order, and Version.

Table 28. 230

T-tests (one sample) for difference between $\log b$ and zero for combined type ADHD (ADHD-C) and normally developing (ND) groups, by Block and Order.

Table 29. 230

T-tests (one sample) for difference between $\log b$ and zero for combined type ADHD (ADHD-C) and normally developing (ND) groups, by Version, Order, and Reward Type.

Log b is calculated over all trials.

Table 30. 231

Discriminability ($\log d$) within the last block of trials: Univariate ANOVA Table. Between-subjects factors are Group, Version, and Order.

Table 31. 231

Discriminability ($\log d$) within the last block of trials: Univariate ANOVA Table. Between-subjects factors are Group and Version.

Table 32.	232
Discriminability ($\log d$) within the last block of trials for normally developing (ND) and combined type ADHD (ADHD-C) groups: Univariate ANOVA Table. Between-subjects factors are Version, and Order.	
Table 33.	232
Response time (RT): Univariate ANOVA Table. Between-subjects factors are Group, Version, and Order.	
Table 34.	233
Pearson product moment correlations between independent (age and estimated IQ score) and dependent ($\log b$, $\log d$, and response time) variables within the combined type ADHD (ADHD-C) and normally developing (ND) groups.	
Table 35.	233
Pearson product moment correlations between signal detection variables ($\log b$, $\log d$, and response time) for combined type ADHD (ADHD-C) and normally developing (ND) groups. Correlations between variables within the same trial block are underlined and in bold text.	

Chapter 1: Attention-Deficit/Hyperactivity Disorder. Introduction, Epidemiology, Comorbidity, and Aetiology

Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is a disorder characterised by difficulties with inattention and/or hyperactivity and impulsivity (American Psychiatric Association, 2000). ADHD is one of the most commonly diagnosed disorders of childhood, affecting approximately 3-7 percent of school-aged children (American Psychiatric Association, 2000). Children with ADHD have serious difficulties with social and academic functioning (American Psychiatric Association, 2000), and many continue to experience ADHD symptoms and related difficulties throughout adolescence and adulthood (Barkley, Fischer, Smallish, & Fletcher, 2006; Mick, Faraone, Biederman, & Spencer, 2004). The current study examines the impact of reward frequency and reward delay on the response preference of children with ADHD. The current chapter reviews the literature on epidemiology, comorbidity, assessment, and aetiology of ADHD. The following chapter reviews some of the experimental approaches that have been used to study ADHD, and also introduces some of the prominent theories of ADHD.

Assessment and Diagnosis of ADHD

Currently, ADHD is diagnosed according to the criteria of the Diagnostic and Statistical Manual of Mental Disorders – Fourth Edition (DSM-IV: American Psychiatric Association, 2000). DSM-IV criteria for ADHD include nine symptoms of inattention, nine symptoms of hyperactivity, and three symptoms of impulsivity. These symptoms include both specific, observable behaviours (e.g., “often fidgets with hands or feet or squirms in seat”), and behavioural patterns or tendencies that require some interpretation on the part of the observer (e.g., “often avoids, dislikes, or is reluctant to engage in tasks that require sustained

mental effort (such as schoolwork or homework)"). The DSM-IV criteria for ADHD were developed based on field trials involving 440 individuals aged from 4 to 17 years (Faber et al., 2010; Lahey et al., 1994). DSM-IV criteria for ADHD require that an individual must have exhibited six of symptoms of inattention, and/or six symptoms of hyperactivity and impulsivity for 6 months or more (Criterion A). These symptoms are deemed present if they are maladaptive and inconsistent with developmental level. Impairment caused by inattention or hyperactivity/impulsivity must have been present before the age of 7 years (Criterion B), be present in two or more settings (Criterion C), and must be clinically significant (Criterion D). Criterion E states that the symptoms cannot be accounted for by other disorders. The DSM-IV currently defines three types of ADHD:¹

- 1) *ADHD, predominantly inattentive type* (ADHD-PIA), where at least six symptoms of inattention occur, but there are fewer than six symptoms of hyperactivity-impulsivity;
- 2) *ADHD, predominantly hyperactive-impulsive type* (ADHD-HI), where at least six symptoms of hyperactivity-impulsivity occur, but there are fewer than six symptoms of inattention; and
- 3) *ADHD, combined type* (ADHD-C), where the individual exhibits six or more symptoms of inattention and six or more symptoms of hyperactivity/impulsivity.

Although the DSM-IV symptoms of ADHD are relatively specific compared with earlier diagnostic criteria, there remains some room for interpretation. For example, it is unclear in the DSM-IV whether the symptoms should be summed over settings. Criterion A requires the presence of at least 6 symptoms of inattention and/or hyperactivity/impulsivity

¹ A fourth category of ADHD diagnosis in the DSM-IV is *ADHD not otherwise specified*, which is given to individuals who exhibit prominent symptoms of inattention or hyperactivity without meeting full criteria for ADHD.

while Criterion C requires that these symptoms be present in two or more settings. To illustrate this point, consider a child who exhibits 4 symptoms of inattention at home, and a further two symptoms at school. If symptoms are added across settings, then this child would meet diagnostic criteria, whereas if symptoms are not added across settings, the child would not meet the diagnostic threshold.

In order to make a diagnosis of ADHD there needs to be a thorough assessment. Assessors should collect information from multiple sources (e.g., the child, parents, and teachers) and utilise multiple methods of assessment, including clinical interviews, behaviour checklists, and behavioural observations (American Academy of Paediatrics Committee on Quality Improvement Subcommittee on Attention-Deficit/Hyperactivity Disorder, 2000, 2001). Medical assessment should be undertaken in order to determine whether there is an underlying physical cause for the ADHD symptoms (Taylor et al., 1998). Finally, it is also important to assess for the presence of comorbid conditions as there are a number of disorders that often occur in children with ADHD.

Epidemiology of ADHD

Prevalence rates for ADHD are often cited as 3 to 7 percent in school-aged children (American Psychiatric Association, 2000), although a recent large-scale study found that approximately 8.4 percent of children in the USA have ADHD (Pastor & Reuben, 2008). There is considerable variation, however, depending on the diagnostic criteria used, the method of assessment, and the sample. Lower prevalence rates are often found in studies that employ more stringent criteria, such as requiring evidence of impairment, or requiring that multiple informants report symptoms (Barkley, 2006a). There is also some variability across countries, with prevalence rates across nine countries varied from 0 to 29 percent (Barkley, 2006a). A large meta-analysis of studies containing over 170,000 individuals estimated the

global prevalence of ADHD at around 5.3 percent (Polanczyk, de Lima, Lessa Horta, Biederman, & Rohde, 2007). Prevalence estimates of ADHD in New Zealand are generally consistent with estimates obtained in other countries. A large prospective study on a birth cohort of 1000 individuals found prevalence rates of DSM-III-R ADHD of 2.8 and 4.8%, depending on method of analysis used (Fergusson, Horwood, & Lynskey, 1993).

ADHD is more frequently diagnosed in boys than in girls. The average male to female ratio in clinical samples is approximately 6:1, although considerable variation exists across studies, with estimates ranging from 2:1 to 10:1 (Barkley & Murphy, 2006). Gender differences have also been found in community based samples, although these tend to be smaller than those found in clinical samples, and range from 2.5:1 to 5.1:1 with an average ratio of 3.4:1 (Barkley & Murphy, 2006). Barkley and Murphy suggest that the greater difference in clinic-based samples occurs because males exhibit more frequent antisocial and aggressive behaviours in general, and are, therefore, more likely to be clinically referred.

In addition to having different overall prevalence rates, gender differences also appear in the relative rates of ADHD subtypes. Graetz, Sawyer, Hazell, Arney, and Baghurst (2001) examined the prevalence of DSM-IV ADHD in 4-17 year old children and adolescents and found that 7.5% of the sample met criteria for ADHD (1.9% ADHD-C, 3.7% ADHD-PIA, and 1.9% ADHD-HI). Males were over-represented in all subtypes, although this was most pronounced for ADHD-C (82 % of the ADHD-C group). This finding is consistent with earlier research using DSM-III (American Psychiatric Association, 1980) criteria, which found that boys had higher rates of Attention Deficit Disorder with Hyperactivity (ADD+H) than girls (9.4% vs. 2.8%), although their rates of Attention Deficit Disorder without Hyperactivity (ADD-H) was similar to that of boys (Szatmari, Offord, & Boyle, 1989).

Course of ADHD

Although the DSM-IV diagnostic criteria have been designed to apply to children, children with ADHD often continue to exhibit symptoms of ADHD in adolescence and adulthood. DuPaul et al. (2001) examined the prevalence of ADHD in 1209 university students from New Zealand, Italy, and the United States. Using DSM-IV symptom criteria, threshold levels of ADHD symptoms were found in 8.1% of New Zealand male students (2.7%, ADHD-PIA; 5.4%, ADHD-HI type; and 0% ADHD-C), and 1.7% of New Zealand female students (all ADHD-HI). The New Zealand estimates were similar to those obtained from Italy and the USA (e.g., total ADHD of 7.4 % for Italy, and 2.9% for USA). It should be noted, however that these are likely to be liberal estimates of the prevalence of ADHD because DuPaul et al. based their prevalence estimates on information from self-report only, and data regarding impairment and developmental course were not obtained. Barkley (2006a) reviewed a number of outcome studies of individuals with ADHD, and concluded that up to 80 percent of children diagnosed with ADD/ADHD continue to display high levels of ADHD symptoms in adolescence. Although the prevalence of ADHD in adolescence is lower than that found in children, this is likely a result of the diagnostic criteria being selected for use with children. In other words, some individuals may ‘outgrow’ the symptom criteria rather than the actual impairments that the symptom criteria reflect. In a recent large-scale community study in the USA, the prevalence of ADHD in adults was 4.4 percent (Kessler et al., 2006).

Associated Impairments and Comorbidity

In addition to the core symptoms, individuals with ADHD experience a number of other difficulties. There are differences between individuals with ADHD and controls on

measures of intellectual functioning, prevalence rates of other psychiatric conditions, and life outcomes.

In terms of comorbid conditions, there is considerable variation in the reported prevalence rates. Factors likely to influence prevalence include: the rigour of the procedures for screening for comorbid conditions, the number of comorbid conditions examined, the methods used to assess comorbid conditions, and the sample (i.e., clinically or community based). Szatmari et al.(1989) examined the prevalence of DSM-III ADD and other disorders in a large community sample of children and adolescents, and found that 44% of the individuals with ADD+H met criteria for at least one additional disorder, 32% met criteria for at least two additional disorders, and 11% had at least three additional disorders. Rates of comorbidity as high as 87 percent have been found in community based samples (Kadesjo & Gillberg, 2001). It is likely that rates of comorbidity are even higher in clinically-referred individuals (Barkley, 2006a). Overall, comorbidity in children with ADHD appears to be the norm, rather than the exception.

Disruptive behaviour disorders.

Oppositional defiant disorder (ODD) and conduct disorder (CD) are two related conditions that are commonly observed in individuals with ADHD. Both ODD and CD involve angry and aggressive behaviour, although in CD, the behaviour involves more severe violation of societal norms, for example, criminal and antisocial behaviour (American Psychiatric Association, 2000). ODD and CD are relatively rare in the general population, with prevalence estimates of ODD ranging between 2 and 16 percent, and rates of CD ranging from 0 to 10 percent (American Psychiatric Association, 2000). Rates of ODD and CD are much higher in children with ADHD than children without ADHD. ODD has been found in approximately 35-60 percent of children with ADHD (e.g., American Psychiatric Association,

2000; Elia et al., 2009), although lower rates can be found when screening procedures for comorbid disorders are less rigorous (e.g., Faber et al., 2010). Rates of CD in children with ADHD are typically somewhat lower than rates of ODD. Biederman, Newcorn, and Sprich (1991) reviewed a number of studies of ADHD comorbidity, and concluded that CD occurs in approximately 30-50% of both clinical and community samples of children with ADHD.

Anxiety and mood disorders.

Anxiety disorders (e.g., obsessive compulsive disorder) and mood disorders (e.g., bipolar disorder, major depressive disorder) are found relatively frequently in children with ADHD. Jensen, Shervette, Xenakis, and Richters (1993) found that 48 percent of their children with ADD+H had at least one additional diagnosis of depression or anxiety. Estimates of the prevalence rates of anxiety disorders in children with ADHD range from around 17 percent (Szatmari et al., 1989) to around 60 percent (e.g., Hammerness et al., 2010). The rates of comorbid mood disorders in children with ADHD are similar to the rates of anxiety disorders. Biederman et al. (1991) found mood disorders were found in approximately 15 to 75 percent of individuals with ADHD. In addition to high rates of comorbidity between ADHD and mood and anxiety disorders, it also appears that ADHD might predict the emergence of anxiety and depression at later stages (Peterson, Pine, Cohen, & Brook, 2001).

The diagnosis of bipolar disorder in individuals with ADHD is somewhat complicated. The DSM-IV Criterion E for ADHD precludes a diagnosis of ADHD if the symptoms occur exclusively in the course of other disorders (pervasive developmental disorders and schizophrenia) or are better accounted for by other disorders (e.g., mood disorders, anxiety disorders, dissociative disorders or personality disorders). The symptoms of bipolar disorder

overlap with those of ADHD (Barkley, 2006a)², leading to difficulty determining what diagnosis is appropriate for some children (see Biederman, 1998 for a discussion of this topic). There is, however, some evidence that bipolar disorder occurs independently of ADHD and can be separated diagnostically (Milberger, Biederman, Faraone, Murphy, & Tsuang, 1995). The rate of comorbid ADHD in children with bipolar disorder is very high (98 percent according to Wozniak et al., 1995). Estimates of the prevalence of bipolar disorder in children with ADHD are somewhat lower, ranging from approximately 12 percent (Biederman et al., 1996) to around 20 percent (Wozniak et al., 1995). These high rates of comorbidity occurred even with overlapping symptoms removed from the diagnostic algorithm (Wozniak et al., 1995).

Pervasive developmental disorders.

The diagnosis of pervasive developmental disorders (PDD) in individuals with ADHD is also complicated. The DSM-IV criteria preclude a diagnosis of ADHD if the symptoms of ADHD occur exclusively in the course of a pervasive developmental disorder (American Psychiatric Association, 2000). Although the diagnostic overlap between symptoms of PDDs (e.g., autistic disorder) and ADHD are less obvious than the Bipolar Disorder-ADHD overlap, there are similarities in the types of difficulties that children with ADHD and PDDs experience. For example, DSM-IV criteria for autistic disorder require the presence of impairments in social interaction and communication, as well as repetitive or stereotyped patterns of interests or activities. Although the criteria for ADHD are different from the

² The DSM-IV criterion for ADHD 2(f) “often talks excessively” is equivalent to the Manic Episode criterion B(3) “more talkative than usual or pressure to keep talking.” The ADHD criterion 1(h) “is often easily distracted by extraneous stimuli” is equivalent to the Manic Episode criterion B(5) “distractibility.” The ADHD criteria 2(a)-(e) all cover symptoms of hyperactivity which are likely related to the Manic Episode criterion B(6) “increase in goal-directed activity (either socially, at work or school, or sexually) or psychomotor agitation.”

criteria for autistic disorder, children with ADHD often have impairments in aspects of language use and comprehension (Mathers, 2005) and social interaction (Landau, Milich, & Widiger, 1991). Some of these impairments that are related to ADHD are similar to the core symptoms of autistic disorder. Additionally, particular symptoms of autistic disorder, such as “stereotyped and repetitive motor mannerisms” (Criterion 3c) are similar to the ADHD symptoms “often fidgets with hands or feet or squirms in seat” (2a) and “is often “on the go” or often acts as if “driven by a motor”” (2e). There is some evidence, however that pervasive developmental disorders occur comorbidly with ADHD and should be treated as separate diagnostic entities (Goldstein & Schwebach, 2004). Several studies have found that around 50 to 75 percent of children with pervasive developmental disorders also have high levels of ADHD symptoms (Rich, Loo, Yang, Dang, & Smalley, 2009). The proportion of children with ADHD who also meet criteria for a pervasive developmental disorder appears lower, however. In one of the few studies to specifically examine this overlap, (Frazier et al., 2001) found that 5 percent of children who met DSM-IV criteria for ADHD also met criteria for a PDD.

Intellectual functioning, mental retardation, and learning disorders.

Children with ADHD tend to obtain lower scores on standardised tests of intellectual functioning (Mayes & Calhoun, 2006; Wechsler, 2003b), although individuals with ADHD represent the entire range of IQ scores (Barkley, 2006a). In a recent meta-analysis, children with ADHD performed worse on standardised tests of intellectual functioning with a difference of approximately 9 Full Scale IQ (FSIQ) points (Frazier, Demaree, & Youngstrom, 2004). A similar, although less marked pattern has been found in adults with ADHD, with adults with ADHD obtaining 6 FSIQ points less than controls (Hervey, Epstein, & Curry, 2004). These figures may, however, underestimate the true difference in intellectual

functioning between children with ADHD because children with low IQs are often excluded from analysis (e.g., Mayes & Calhoun, 2006).

In addition to having lower average IQ scores, individuals with ADHD have higher rates of mental retardation (MR)³. Currently, the DSM-IV (American Psychiatric Association, 2000) defines MR as consisting of significantly sub average intellectual functioning with concurrent deficits in adaptive behaviour. The issue of comorbidity is often examined from the MR perspective; that is, what proportion of individuals with MR also has ADHD? (Di Nuovo & Buono, 2007). There is a growing literature, however, on the converse perspective. One study found that 11 percent of their ADHD sample had FSIQ scores below 70, while 17 percent of their ADHD sample had FSIQ scores in the borderline (i.e., FSIQ scores between 70 and 84) range (Ishii, Takahashi, Kawamura, & Ohta, 2003). Kube, Petersen, and Palmer (2002) assessed a large group of children for ADHD, behavioural, or learning problems and found that 16 percent of the children referred for evaluation of ADHD met criteria for MR. The rate of MR in individuals with ADHD appears markedly higher than the 1 percent found in the general population (American Psychiatric Association, 2000). It is possible, however, that children with ADHD and MR are more likely to be referred for assessment and therefore to be diagnosed with MR than children with ADHD alone.

Life Outcomes in Individuals with ADHD

The symptoms of ADHD have a significant impact on the functioning and quality-of-life of affected individuals. Children with ADHD experience impaired social functioning (Rich et al., 2009; Wehmeier, Schacht, & Barkley, 2010). In adolescence, individuals with ADHD have been found to have difficulties in a number of health and quality-of-life

³ The term 'Intellectual disability' is generally currently preferred to 'Mental Retardation.' The term "Mental Retardation' has been retained, however, as it is consistent with the DSM-IV.

compared with controls, such as interference with family and peer/school activities, as well as higher levels of pain and discomfort (Sawyer et al., 2002).

Children with ADHD continue to suffer various impairments into adolescence and adulthood in addition to the core symptoms of ADHD. Individuals who have high levels of ADHD symptoms often continue to have social and academic difficulties in adolescence, even if they do not meet full diagnostic criteria for ADHD (Fischer, Barkley, Edelbrock, & Smallish, 1990; Schaugency, McGee, Raja, Feehan, & Siva, 1994). Adolescents with ADHD also have elevated rates of alcohol and substance abuse (Alterman, Petrarulo, Tarter, & McGowan, 1982; Andersson, Magnusson, & Wennberg, 1997; Lambert, Hartsough, Sassone, & Sandoval, 1987), and criminal behaviour (Satterfield & Schell, 1997).

Adults previously diagnosed with ADHD continue to have elevated rates of alcohol and substance use, have more unexplained absences at work, and face increased health costs (Secnik, Swensen, & Lage, 2005) compared to controls. Adults with ADHD are also more likely than controls to have engaged in antisocial behaviour (Satterfield & Schell, 1997; Secnik et al., 2005) and to have been arrested (Biederman et al., 2006). ADHD is also more prevalent in incarcerated adults than would be expected based on community prevalence estimates (Curran & Fitzgerald, 1999), although the relationship between early hyperactivity and later criminality is less clear when the presence of early conduct problems is taken into account (Mannuzza, Klein, Konig, & Giampino, 1989).

Adults previously diagnosed with ADHD are more likely to face a number of economic costs than individuals without ADHD. Adults with ADHD are more likely than controls to be unemployed and have more frequent changes of employment (Biederman et al., 2006). The employment difficulties adults with ADHD have may be explained by the fact that adults previously diagnosed with ADHD have more unexplained absences from work (Secnik

et al., 2005), and are less likely to have completed high school or graduated from college (Biederman et al., 2006).

Research on the driving behaviour of adults with ADHD found that adults with ADHD made more errors on tests of driving rules and decision making than controls (Barkley, Murphy, DuPaul, & Bush, 2002). Drivers with ADHD also self-report high levels of lapses, errors, and rule violations when driving, compared with controls. These high rates of errors, lapses, and rule violations are a likely cause of the relatively high numbers of citations for driving offences, license suspensions, and increased accident rates that have been observed in adults with ADHD (Barkley et al., 2002; Fried et al., 2006).

Aetiology

A number of different aetiologies have been proposed in relation to ADHD. These factors include: Genetics; family environment; prenatal risk factors such as maternal illness and prenatal exposure to toxins; perinatal risk factors (e.g., obstetric complications); and neurobiological factors.⁴

Genetics.

ADHD appears to contain a large genetic component. Evidence regarding the genetic component of ADHD comes from two sources – family studies and genetic studies. Family studies include pedigree, sibling, twin, and adoption studies, and examine the prevalence of ADHD in the biological and adoptive relatives of individuals with ADHD. Genetic studies on the other hand analyse the DNA of individuals with ADHD.

Pedigree studies have consistently shown that relatives of individuals with ADD/ADHD are more likely than individuals without ADD/ADHD to meet diagnostic

⁴Male gender was also identified as a risk factor, while high school achievement was identified as a protective factor.

criteria for the disorder. Biological parents, compared with adoptive parents of children with ADHD are more likely (18% vs. 6%) to have ADHD themselves (Sprich, Biederman, Crawford, Mundy, & Faraone, 2000). Parents of hyperactive children have a higher incidence of hyperactivity than parents of control children (Cantwell, 1972, 1975; Morrison & Stewart, 1971, 1973), and first-degree relatives of children with ADD are more likely to meet diagnostic criteria for ADD than relatives of normal and psychiatric control children (Biederman, Faraone, Keenan, Knee, & Tsuang, 1990). Mick and Faraone (2008) estimated that parents of children with ADHD are two-to-eight times more likely to have ADHD. There is also some evidence that the prevalence of ADHD is higher in second-degree relatives of children with ADHD than relatives of control children (Faraone, Biederman, & Milberger, 1994).

Pedigree studies have a major problem; shared environment co-occurs with shared genetics (i.e., relatives often share genes *and* environments). To separate the influences of genes and environment, a number of studies have compared the concordance rates between full and half siblings, and between mono- and dizygotic twins. Safer (1973) examined the full and half siblings of a group of children with minimal brain dysfunction (a diagnostic precursor to ADD/ADHD). There was a higher concordance rate between full siblings than half siblings. Because these children were raised by their biological mothers in similar environments, the differences in concordance rates were thought to mainly result from genetic differences.

Monozygotic (MZ) twins share 100 percent of their genetic material, while dizygotic (DZ) twins share only 50 percent of their genetic material (Samudra & Cantwell, 1999). Differences in concordance rates between mono- and dizygotic twins are often interpreted as reflecting genetic differences, because twins are exposed to similar environments (although this assumption has been critiqued by Joseph (2000). Gillis, Gilger, Pennington, and DeFries

(1992) examined the concordance rates for ADHD between same-sex di- and monozygotic twin pairs, and found higher concordance between monozygotic twins.-

Adoption studies attempt to separate environmental and genetic influences on behaviour by comparing the concordance of biologically related family members with the concordance of adopted family members. The biological parents of hyperactive children are more likely to have been hyperactive as children than the adoptive parents of hyperactive children (Morrison & Stewart, 1973). Overall, estimates of the heritability of ADHD range from approximately .50 to .90, that is, .50 to .90 of the variability in ADHD symptoms can be attributed to genetic variation (Castellanos, 1999; Castellanos & Rapoport, 1992; Goodman & Stevenson, 1989; Waldman & Gizer, 2006; Waldman & Rhee, 2002). After reviewing 20 twin studies, (Faraone et al., 2005) estimated the heritability of ADHD as 76 percent.

Although family and adoption studies show that ADHD has a genetic component, they shed little light on the heritable mechanisms that cause individuals with ADHD to behave the way they do. Recently, a number of studies have examined specific genes in individuals with and without ADHD. The majority of these studies have focussed on genes related to the neurotransmitter dopamine. When compared to controls, individuals with ADHD have been found to have higher frequencies of particular polymorphisms of genes coding for the DRD2 (Comings et al., 1991) and DRD4 (Faraone et al., 1999; LaHoste et al., 1996; Rowe et al., 1998; Smalley et al., 1998) dopamine receptors. Differences between ADHD and controls have also been found in the frequencies of gene allele variants which code for dopamine transporters (Cook et al., 1995), which are involved in the reuptake of dopamine from the synapse. Recent reviews of the genetic literature noted that there are reliable and well replicated differences between ADHD and controls in the DRD4 and DRD5 (dopamine receptor) and DAT1 (dopamine transporter) gene allele variants (Brookes et al., 2006; Carrasco et al., 2006; Faraone & Mick, 2010; Mick & Faraone, 2008; Zhou et al., 2008).

There have also been between-group differences in gene allele variants coding for various proteins involved in broader neurotransmitter activity, for example, serotonin receptors, and SNAP-25, which regulates neurotransmitter release (Faraone & Mick, 2010). In addition to other genes involved in the activity of dopamine, norepinephrine, and serotonin, research has also examined genes in the cholinergic and glutamate systems, although findings to date have been less consistent than those noted above (Faraone & Mick, 2010). One of the difficulties interpreting the genetic research is that different chromosome regions are identified in different studies, leading to suggest that there are multiple genes involved in ADHD, with each individual gene having small or moderate effects (Zhou et al., 2008).

Family environment.

Family characteristics that are more prevalent in the families of children with ADHD than control children include marital discord, poor family relationships, low social class, large family size, paternal criminality, maternal mental disorder, and foster care placement (Biederman et al., 1995a; McGee, Williams, & Silva, 1985). Additionally, when compared to control families, families of children with ADHD have higher levels of parental conflict and less family cohesion (Biederman et al., 1995b). Biederman et al. also found that higher levels of adversity predicted lower levels of adaptive functioning, independent of an ADHD diagnosis.

Goodman and Stevenson (1989) proposed four hypotheses regarding the direction of causality between family adversity and ADHD:

- 1) Family adversity causes ADHD: Goodman and Stevenson (1989) found only a small relationship between family adversity and ADHD symptoms. Less than 10 percent of the variance in ADHD symptoms was attributed to the combined effect of all of the family factors considered. This compares with approximately 30-50

percent of the variance attributed to genes (Castellanos, 1999; Castellanos & Rapoport, 1992; Goodman & Stevenson, 1989; Waldman & Gizer, 2006; Waldman & Rhee, 2002).

- 2) Family adversity and ADHD are genetically linked: Hyperactive children are more likely than control children to develop problems such as alcohol and substance use (Andersson et al., 1997) and criminal behaviour (Satterfield & Schell, 1997). Given the likely genetic underpinnings of ADHD (e.g., Waldman & Gizer, 2006) it seems likely that children with ADHD would be more likely to have parents with high levels of adversity.
- 3) ADHD and family adversity share common environmental causes (e.g., toxins, low socioeconomic status): This is perhaps the most difficult of the four hypotheses to test because genes and environment are frequently linked. Although there is some evidence that children with ADHD are more likely to come from low SES families (Velez, Johnson, & Cohen, 1989), it is difficult to separate the potential influences of broad environmental factors from specific factors such as genetic endowment and family adversity.
- 4) ADHD causes family adversity: Goodman and Stevenson (1989) examined the relation between family adversity and hyperactivity in a subgroup of children selected on the basis of a clinical screening questionnaire, reasoning that both the hyperactive and non-hyperactive children would be likely to experience similar levels of family adversity (e.g., high parental criticism and low parental warmth). They found no relation between family adversity and ADHD symptoms. Although stimulant medication treatment of children with ADHD has been found to lead to increased maternal warmth and decreased maternal criticism (Schachar, Taylor, Wieselberg, Thorley, & Rutter, 1987), this is most likely due to changes in the

children's behaviour. If ADHD was a result of parenting behaviour, it would perhaps be unusual to find an association between stimulant treatment of children and a change in parenting behaviour.

To summarise, the direction of causality between ADHD and family environment is far from clear. Several authors (e.g., Goodman & Stevenson, 1989; Samudra & Cantwell, 1999) have favoured the fourth hypothesis (ADHD causes family adversity) but more evidence needs to be gathered regarding the relation between ADHD and environmental factors.

Prenatal and perinatal risk factors.

Birth and obstetric complications.

One area of studies that has a long history is the study of difficulties with pregnancy and birth in relation to ADHD and other behaviour problems. Children with broadly defined behaviour disorders have often had increased rates of complications during pregnancy and delivery, and are more likely to have been born preterm than matched control children (Pasamanick, Rogers, & Lilienfeld, 1956). These differences were most pronounced in a subset of children classified as "confused, disorganized, and hyperactive". Pasamanick et al. suggested that behaviour disorders (along with epilepsy and mental deficiency) were caused by a mild form of brain damage. Additionally, Connors (1975) noted that the mothers of children with minimal brain dysfunction had high rates of previous abortions and toxemia during pregnancy.

Mothers of children with ADHD have been found to be younger than mothers of control children, more likely to be first-time mothers, and more likely to have experienced poor health during pregnancy (Hartsough & Lambert, 1985), although poor health during pregnancy has not always been found (Minde, Webb, & Sykes, 1968). The deliveries of the

children with ADHD have been found to be longer than deliveries of control children (Hartsough & Lambert, 1985), and more likely to be complicated (Minde et al., 1968; Sprich-Buckminster, Biederman, Milberger, Faraone, & Lehman, 1993). Babies who develop ADHD are more likely to have experienced foetal distress during delivery, been delivered late (i.e., foetal post maturity), and more likely to suffer from congenital problems and health problems during infancy (Hartsough & Lambert, 1985). However, Sprich-Buckminster, Biederman, Millberger, Faraone, and Lehman (1993) found differences in birth complications between mothers of children with and without ADHD only when a family history of ADHD was absent. The authors concluded that a history of pre- and perinatal complications was a risk factor for 'non-familial ADHD,' but noted also that complications increased risk for comorbid disorders and general psychopathology; that is, the risks of complications were not specific to ADHD.

Low birth weight has been identified as a risk factor for a number of medical, developmental, and behaviour problems (McCormick, Brooks-Gunn, Workman-Daniels, Turner, & Peckham, 1992; Teplin, Burchinal, Johnson-Martin, Humphry, & Kraybill, 1991). Infants weighing less than 1000g at birth have shorter attention spans than normal weight controls (Teplin et al., 1991), and babies weighing less than 1500g at birth are more hyperactive than children of normal birth weight (Hack et al., 1992). Children weighing less than 2500g at birth have higher rates of ADHD, but not anxiety disorders or ODD (Breslau et al., 1996).

Prenatal toxin exposure.

A number of studies have examined the etiological role of prenatal environmental toxins in ADHD. Streissguth, Barr, Sampson, and Bookstein (1994) prospectively examined a cohort of children for 14 years, and found that maternal alcohol use during pregnancy

predicted poor performance on vigilance test performance that indicated difficulties with both sustained attention and response inhibition. These relative deficits in performance remained when other environmental factors (e.g., maternal tobacco and drug use, level of maternal education, and demographic variables) were controlled for, and were present at 4, 7, and 14 years of age. Boyd, Ernhart, Greene, Sokol, and Martier (1991) failed to replicate these findings, but this result may have arisen because they did not distinguish adequately between regular and binge drinking (Samudra & Cantwell, 1999). Pineda et al. (2007) found that children whose mothers consumed large amounts of alcohol during pregnancy⁵ were more likely to have children with ADHD than mothers who consumed no alcohol, whereas children whose mothers only consumed small amounts⁶ of alcohol during pregnancy were not significantly more likely to have offspring with ADHD.

Brown et al. (1991) compared the children of mothers who continued to drink alcohol during pregnancy with the children of mothers who did not drink, and mothers who ceased drinking during pregnancy. Children of mothers who continued drinking had the highest teacher-reported levels of externalising behaviours, lowest levels of academic achievement, the highest rates of internalising behaviours, and the lowest scores on a computerised measure of sustained attention, although these group differences were no longer significant when current maternal drinking was controlled for.

There is some evidence that prenatal heroin exposure may lead to higher levels of hyperactivity and inattention (Ornoy, Michailevskaya, Lukashov, Bar-Hamburger, & Harel, 1996). This may be due to the relation between maternal heroin use and low birth weight

⁵ A high level of alcohol exposure was defined as having been intoxicated at least once during the first two months of pregnancy.

⁶ A small level of alcohol exposure was defined as having consumed less than 10 drinks per week during pregnancy.

(Samudra & Cantwell, 1999). Ornoy, Segal, Bar-Hamburger, and Greenbaum (2001) found elevated rates of ADHD in the children of heroin-dependent parents. Rates of ADHD were highest in the children of heroin-dependent mothers who were raised by their biological mothers. Rates of ADHD were also elevated, although to a lesser degree, in the children of heroin-dependent fathers, and children of heroin-dependent mothers who were adopted shortly after birth. Ornoy et al. noted that compared to heroin-dependent fathers and the adoptive and control parents in the study, heroin-dependent mothers had higher rates of retrospectively reported ADHD symptoms. Ornoy et al. also noted that for the overall sample of children in their study, there was a positive correlation between the number of parent and child ADHD symptoms. Ornoy et al. concluded that prenatal exposure to heroin leads to high rates of hyperactivity and inattention in children, although they also acknowledged the role of genetics.

There has also been some research examining the relation between prenatal exposure to cocaine and ADHD in offspring. One group of cocaine-exposed infants was found to have higher levels of aggressive behaviour and lower scores on a measure of verbal reasoning (Azuma & Chasnoff, 1993; Griffith, Azuma, & Chasnoff, 1994). Richardson, Conroy, and Day (1996) found that children who were prenatally exposed to cocaine made more errors than non-exposed children on a vigilance task, indicating possible difficulties with inattention. The difference between groups remained when potential confounds (child age, child IQ, school grade, mother's race, mother's self-esteem, maternal alcohol use, and timing of cocaine exposure) were controlled for. Interestingly, there were no differences between groups on measures of disruptive behaviour.

One of the difficulties of research examining the effects of various illicit substances is that the use of additional substances (e.g., tobacco and alcohol) is often not controlled for

(e.g., Ornoy et al., 2001). In studies that controlled for alcohol and tobacco use, the findings tend to be less clear (e.g., Richardson et al., 1996).

The link between maternal smoking during pregnancy and ADHD has been fairly well established (see Linnet et al., 2003 for a review). In a group of children and adolescents, rates of ADHD were found to be significantly higher in individuals whose mothers smoked more than 10 cigarettes per day during pregnancy, compared with individuals whose mothers did not smoke while pregnant (Schmitz et al., 2006). This result remained significant when potential confounds (maternal ADHD, prenatal alcohol exposure, birth weight, and comorbid disorders) were controlled for. A similar pattern of results was found by (Linnet et al., 2005), who found a threefold increase in the rate of hyperkinetic disorder in the offspring of women who smoked during pregnancy, compared with women who did not smoke during pregnancy. Interestingly, a multivariate model of risk factors for ADHD found that maternal smoking was significantly predicted ADHD in boys, but not girls, leading the authors to suggest that maternal smoking is only a significant predictor of ADHD in boys.

Lead is a substance known to be toxic at high doses, and even sub-toxic levels of lead exposure have been linked to a range of behaviour problems. In one of the earlier studies of sub-toxic levels of lead exposure, Needleman et al. (1979) found that dentine lead levels were related to distractibility, difficulties with organisation, daydreaming, hyperactivity, impulsivity, and low overall functioning. Similar findings have been reported by several investigators (Baloh, Sturm, Green, & Gleser, 1975; Thomson et al., 1989), even when potential confounds (i.e., gender, age, and SES) were controlled for (Tuthill, 1996). Not all investigators have found a relation between lead levels and ADHD symptoms however (e.g., Landrigan et al., 1975). Castellanos and Rapoport (1992) suggested that some factors (e.g., low parental education levels, family adversity, and prenatal alcohol exposure) may

sometimes mask the effects of lead exposure, which may explain some of the negative findings.

Chen et al. (2007) found that blood-lead levels were negatively associated with IQ scores in a cohort of children examined at 2, 5, and 7 years of age. When the children were 5 years old there was no significant relation between lead levels and ADHD symptoms. When the children were 7 years old, there was a significant association between lead levels and externalising and school-related problems,⁷ although IQ partially mediated the relationship between lead levels and IQ behaviour problems.

Of the pregnancy and delivery complications studied in relation to ADHD, Faraone and Biederman (2000) noted that the factors that tend to predispose children to ADHD are those that often lead to hypoxia, and are often chronic (e.g., maternal smoking) rather than acute (e.g., delivery complications) in nature.

Gene-environment interactions.

Earlier research examining the linkage between individual gene allele variants (e.g., Rowe et al., 1998) and behaviour disorders has been supplanted by recent research examining specific mechanisms of heritability and gene-environment interactions. Goos, Ezzatian, and Schachar (2007) compared children whose fathers were diagnosed with ADHD (Paternal ADHD group) to children whose mothers were diagnosed with ADHD (Maternal ADHD Group). The Maternal ADHD group had higher rates of behaviour problems (symptoms of ADHD, ODD, and CD) but lower rates of depressive symptoms than the Paternal ADHD Group. Additionally, in the Maternal ADHD group, the levels of ADHD\ODD\CD problems in the girls were equal with or higher than the problems in the boys. This contrasted with the

⁷ Due to the differences in age, different measures were used for the cohort of children at age 5 and age 7. ADHD scores were not reported for the cohort at age 7.

Paternal ADHD group, where boys showed the highest levels of ADHD\ODD\CD problems. The authors concluded that the differences in depression between groups were suggestive of genomic imprinting: children may have inherited genetic vulnerabilities from one parent, but not the other. The finding that girls showed relatively high rates of ADHD\ODD\CD symptoms in the Maternal ADHD Group, but not the Paternal ADHD Group was interpreted as implying that girls were particularly vulnerable to maternal risk factors.

In order to account for some of the inconsistencies in genetic research, Laucht et al. (2007) examined the gene by environment interactions. Children with specific gene allele variants coding for the DAT1 dopamine transporters showed higher levels of ADHD symptoms only when they had high levels of family adversity. Children with the vulnerable gene allele variant and low levels of family adversity showed similar levels of ADHD symptoms to children with non-vulnerable gene allele variants. Additionally, children with non-vulnerable gene allele variants showed similar levels of ADHD symptoms regardless of their levels of family adversity.

In children raised in deprived environments, increased levels of ADHD symptoms have been found in some children, but not others (Sonuga-Barke & Rubia, 2008; Stevens et al., 2008). A later study found that the risk was greatest in these children when they had a particular polymorphism coding for the dopamine transporter gene (Stevens et al., 2009).

Sonuga-Barke et al. (2009) examined the impact of positive expressed emotion on conduct problems in children with ADHD. Children exposed to high levels of expressed emotion showed reduced levels of conduct problems only when high risk polymorphisms of the dopamine transporter and serotonin transporter genes were absent.

Although gene-environment research on ADHD is at an early stage, there have been some promising findings (Wermter et al., 2010). Future genetic research is likely to be more fruitful if relevant environmental factors are taken into account.

Brain structure and function

The structure and function of a number of brain regions have been studied in relation to ADHD. There are three main approaches to examining the differences between the brains of children with ADHD. The first two approaches compare the brain structure and function of children with and without ADHD. Differences in brain structure are often examined using radiographic techniques (e.g., MRI), whereas differences in the function of brains are examined using techniques designed to measure brain activity (e.g., fMRI, EEG). The third approach compares the behaviour of children with lesions to specific brain regions to the behaviour of children without lesions. In addition to children with ADHD having smaller brains than children without ADHD (Kieling, Goncalves, Tannock, & Castellanos, 2008), children with ADHD have specific regions of the brain that are different in both size and function.

Frontal lobes.

From the few morphometric studies of the frontal lobes of individuals with ADHD, it appears that individuals with ADHD have smaller frontal lobes than control children although there is some inconsistency regarding particular regions. Bilateral reductions in the inferior prefrontal cortex have been found in children and adolescents with ADHD relative to controls (Sowell et al., 2003). Hill et al. (2003) however, found no differences between children with ADHD and controls in inferior prefrontal cortex volumes, but found that children with ADHD had smaller superior right (but not left) prefrontal volumes relative to controls. Children with ADHD have reduced left white matter volume in the frontal lobes compared with controls (Kates et al., 2002; Mostofsky, Cooper, Kates, Denckla, & Kaufmann, 2002) and bilateral reductions in grey matter volume (Mostofsky et al., 2002).

Individuals with ADHD differ from controls in their levels of activity in several regions of the frontal lobes. Activity in the right frontal cortex is generally lower in children with ADHD relative to controls (Kim, Lee, Shin, Cho, & Lee, 2002; Yu-Feng et al., 2007). During auditory attention tasks, low activity has been found in the left anterior frontal lobe of adults with ADHD (Zametkin et al., 1993), whereas teenagers with ADHD have shown low activity in several frontal regions, including the superior prefrontal cortex and premotor cortex (Zametkin et al., 1990). During tests of inhibition, adolescents with ADHD show less activity than controls in the left rostral mesial frontal cortex and bilateral prefrontal regions (Smith, Taylor, Brammer, Toone, & Rubia, 2006), and right mesial prefrontal cortex and right inferior prefrontal cortex (Rubia et al., 1999; Rubia, Smith, Brammer, Toone, & Taylor, 2005). Low frontal lobe activity has not always been found in individuals with ADHD however. High levels of frontal lobe activity in individuals with ADHD relative to controls have also been found during tests of inhibition in both children (Vaidya et al., 1998) and adolescents (Schulz et al., 2004; Schulz et al., 2005). Additionally, Durston et al. (2003) found that during a test of inhibition children with ADHD had less activity than controls in the ventral prefrontal cortex, but increased levels of activity in other prefrontal regions. The inconsistency of frontal lobe activity studies during inhibition tasks may reflect differences in task across studies, with increased frontal lobe activity reflecting increased effort during more difficult tasks (Vaidya et al., 1998).

Basal ganglia.

In normal children several regions of the right striatum (caudate nucleus and globus pallidus) are slightly larger than the left. In children with ADHD, caudate nuclear asymmetry is often absent (Castellanos et al., 1994; Castellanos et al., 1996b), although this has not always been observed (Filipek et al., 1997; Hill et al., 2003; Mataro, Garcia-Sanchez, Junque,

Estevez-Gonzalez, & Pujol, 1997). A lack of normal asymmetry in the globus pallidus has also been found in children with ADHD (Castellanos, Giedd, Hamburger, & Marsh, 1996a). Monozygotic twins with ADHD have smaller caudate volumes than their non-affected twins (Castellanos et al., 2003). When overall differences in brain volume were controlled for, only caudate volumes remained significantly smaller in children and adolescents with ADHD relative controls (Waldman & Rhee, 2002).

During the course of normal development, the volume of the caudate nucleus in the basal ganglia reduces from childhood to adolescence; in children with ADHD, however, this does not occur (Casey, Castellanos, Giedd, & Marsh, 1997; Castellanos et al., 1994; Castellanos et al., 1996b; Waldman & Rhee, 2002). The absence of caudate nuclear shrinkage in children with ADHD is likely to be a result of a relative lack of synaptic pruning that occurs in children with ADHD, which indicates that the brains of children with ADHD may be less mature or efficient than control children (Castellanos et al., 1994).

Studies of the activity of basal ganglia structures have also found differences between individuals with and without ADHD, although again there are some conflicting findings. Children with ADHD appear to have less activity than control children in the right striatum (Lou, Henriksen, & Bruhn, 1990; Lou, Henriksen, Bruhn, Borner, & Nielsen, 1989; Vaidya et al., 1998) and left caudate nucleus (Durstun et al., 2003). Schulz et al. (2005) however, found that during a test of interference control, adolescents with ADHD showed *higher* activity than controls in the left basal ganglia; which was positively correlated with ADHD symptom severity. This discrepancy with previous studies may be due to the unique task used by Schulz et al.

Further evidence that the basal ganglia may be involved in ADHD comes from lesion studies, which have found that lesions to regions of the basal ganglia are associated with ADHD symptoms (Gerring et al., 2000; Herskovits et al., 1999).

Cerebellum.

Children with ADHD have smaller cerebellar lobules than controls (Berquin et al., 1998; Hill et al., 2003). The vermis, which divides the cerebellar hemispheres, is also smaller in children with ADHD compared to controls (Castellanos et al., 2001). Studies of cerebellar activity have generally found that individuals with ADHD have less activity than controls during tests of interference (Zang et al., 2005) and inhibition (Suskauer et al., 2008), and also when resting (Kim et al., 2002; Yu-Feng et al., 2007).

Other regions.

Several regions of the corpus callosum have been found to be smaller in children and adolescents with ADHD (Baumgardner et al., 1996; Giedd et al., 1994; Hill et al., 2003; Hynd et al., 1991; Lyoo et al., 1996).

In addition to the previously mentioned studies finding lower levels of activity in multiple brain regions, several studies have also found increased levels of activity in the brains of individuals with ADHD relative to controls. Durston et al. (2003) found that during a test of inhibition, children with ADHD had increased levels of activity in posterior regions of the parietal and occipital cortex. Compared to controls, individuals with ADHD have also been found to have increased activity of the caudal right anterior cingulate (Ernst et al., 2003), and in the left anterior cingulate gyrus (Schulz et al., 2004; Schulz et al., 2005). Suskauer et al. (2008) used a simplified test of inhibition to minimise cognitive demands, and found that children with ADHD had lower levels of activity than controls in areas typically involved in response selection and inhibition (the pre-supplementary motor area of the frontal cortex, right cerebellum, bilateral occipital lobes, and right temporal parietal junction). There were no significant differences between groups during normal responses (i.e., where participants were

not cued to withhold responses). Interestingly the differences in neural activity that occurred were present despite the control group being matched on error rates on the task.

Summary of brain structure and function.

Overall, the most robust findings in the morphometric studies are that the brains of individuals with ADHD have different regional volumes than controls in the frontal lobes, basal ganglia (particularly the right caudate nucleus), and cerebellum (Valera, Faraone, Murray, & Seidman, 2007). Parallel differences in brain activity have also been noted in several regions; individuals with ADHD exhibit different levels of activity in the frontal lobes (e.g., Yu-Feng et al., 2007), basal ganglia structures (e.g., Durston et al., 2003), and cerebellum (e.g., Zang et al., 2005).

Emerging evidence suggests that in addition to having *low* activity in the aforementioned regions, individuals with ADHD also have *high* levels of activity in some regions, notably in the cingulate cortex, and parietal and occipital lobes. In general, it appears that individuals with ADHD have low activity in the frontal-basal ganglia circuit, coupled with high activity in parietal and occipital regions. It has been suggested that children with ADHD have a dysfunctional frontal-striatal system (Heilman, Voeller, & Nadeau, 1991), and that the cerebellum serves to modulate the fronto-striatal circuit (Giedd, Blumenthal, Molloy, & Castellanos, 2001).

Although the main findings suggest that individuals with ADHD have less activity than controls in regions of the frontal lobes, basal ganglia, and cerebellum, recent evidence suggests that individuals with ADHD have a more diffuse pattern of activation: they show increased levels of activity in areas of the frontal, parietal, and occipital lobes (Durston et al., 2003). This may indicate that the brains of individuals with ADHD are relatively immature.

Replication of the emerging findings, coupled with future developments in imaging technology will likely elucidate this matter.

Overall Summary

In summary, ADHD is a disorder characterised by difficulties with inattention and hyperactivity/impulsivity. ADHD is commonly diagnosed in childhood, and many of the difficulties with ADHD are persistent into adolescence and adulthood. In addition to the core symptoms, children with ADHD are at risk for developing a number of other disorders, including disruptive behaviour disorders (ODD and CD), learning problems, and mood and anxiety disorders. Children with ADHD also tend to have relatively poor life outcomes compared to unaffected individuals.

Many potential aetiologies for ADHD have been examined. There is some evidence that the structure and function of the brains of children with ADHD are different from children without ADHD. Recent literature has examined genes that could be associated with ADHD, and found that in particular, genes related to the transport and reception neurotransmitter dopamine may be implicated. Most recently, studies of gene-environment interactions have examined the relationship between genetic and environmental factors, and may resolve some of the inconsistencies in earlier research.

The current experiments examine the sensitivity to rewards in children with ADHD. The following chapter reviews some of the experimental techniques that have been used to study ADHD. Additionally, some of the major theories of ADHD are discussed and their relationship to the experimental findings discussed.

Chapter 2: Behavioural Analysis and Theories of ADHD

Many different experimental procedures have been used to study ADHD, giving rise to a wide range of theories. This thesis examines the response of children with ADHD to rewards, in particular the effects of different frequencies of reward and also delayed versus immediate reward. In order to contextualise the current research, it is first necessary to discuss the broader experimental literature in relation to ADHD, and to the related theories. This chapter reviews the standard experimental procedures used and their results obtained with children that have ADHD. Broadly speaking, these experimental procedures can be grouped into tests of inhibition, tests of working memory, and tests of response to reinforcement. Following the review of experimental findings, the theories of ADHD that have emerged from these results are discussed.

Tests of Inhibition and Impulsivity in Children with ADHD

Continuous performance test.

The original Continuous Performance Test (CPT) was developed by Rosvold, Mirsky, Sarason, Bransome, and Beck (1956) to detect brain damage in children, adults with mental retardation, and adults without mental retardation. CPTs present visual stimuli to the participant, who is required to respond to a target stimulus, and not respond to non-target stimuli. Errors of omission occur when the participant fails to respond to the target stimulus, and are thought to reflect a deficit in arousal (Corkum & Siegel, 1993). Errors of commission occur when the participant responds to a non-target stimulus and are often interpreted as a measure of impulsive responding (Barkley, Edwards, Laneri, Fletcher, & Metevia, 2001). Many different versions of the CPT have been used. Some use auditory rather than visual stimuli, whereas others vary the complexity of the target stimulus (see Riccio, Reynolds, Lowe, & Moore, 2002 for a review).

Barkley, DuPaul, and McMurray (1990) used a commercially available CPT (GDS: Gordon, 1983) to compare the performance of children with attention deficit disorder with hyperactivity (ADD+H), attention deficit disorder without hyperactivity (ADD-H), children with learning disabilities, and control children. Digits were presented on a screen, and the children were required to respond when a '9' appeared after a '1'. Although no significant differences in the number of correct responses⁸ were found between groups, the groups differed in their error rates. Errors of commission (i.e., making responses when a '9' did not follow a '1') and omission (i.e., failing to respond when a '9' followed a '1') occurred more often in the ADD+H group than the learning disabled and control groups.

Although there have been conflicting findings (e.g., Schachar, Logan, Wachsmuth, & Chajczyk, 1988; Van der Meere & Sergeant, 1988; Werry, Elkind, & Reeves, 1987), the majority of research shows a similar pattern to Barkley et al. (1990); children with ADHD have increased errors of omission and commission when compared to controls (Corkum & Siegel, 1993; Losier, McGrath, & Klein, 1996). Although there have been fewer CPT studies with adults with ADHD than children, adults with ADHD also have higher rates of errors of omission and commission (Riccio & Reynolds, 2001).

Go/no-go.

The Go/No-Go task (Newman, Widom, & Nathan, 1985) is a variant of the CPT. Participants are presented with successive stimuli (e.g., numbers) and are required to respond to some stimuli, but not others. Errors of omission and commission are recorded. In parallel with the CPT literature, children with ADHD make more errors of commission (i.e.,

⁸ Although there were no statistically significant differences in the *number* of correct responses between groups, the ADD+H group made more overall responses and errors. The *proportion* of correct responses was lowest in the ADD+H group, although this was not reported.

impulsive errors) than control children. Children with ADHD do not appear to make more omission errors than controls however (Iaboni, Douglas, & Baker, 1995; Milich, Hartung, Martin, & Haigler, 1994).

Stop task.

The Stop Task is based on the work of Logan and others (e.g., Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984; Schachar & Logan, 1990; Tannock, Schachar, Carr, Chajczyk, & Logan, 1989), and is designed to measure the ability to inhibit motor responses. In the Stop Task the primary task is a simple discrimination where the participant must press a key corresponding to each of the two choice stimuli (e.g., 'X' or 'O'). In the secondary task the participant inhibits his choice response when a tone (stop signal) is sounded after the primary stimulus is presented. The delay between the primary stimuli and the stop signal is varied (delays of between 100 and 800 ms are typical) for each participant. Longer delays make it more difficult to inhibit the response (i.e., participants are more likely to respond to the primary stimulus). The basic measures obtained are the reaction time for correct choice responses and the probability of inhibiting responses when the stop signal is presented. From these measures, two further measures are derived. The inhibition slope is a measure of the rate at which the probability of inhibiting responses decreases with increasing stop signal delays (corrected for individual differences in reaction time). A steep slope indicates stronger inhibition than a shallow slope. The Stop Signal Reaction Time (SSRT) is an estimate of the time taken to inhibit a response (see Logan & Cowan, 1984 for an explanation of how the SSRT is derived). Shorter SSRTs are interpreted as indicating a relatively efficient inhibition process, whereas longer SSRTs indicate a less efficient inhibition process.

A number of studies have compared children with ADHD to normal controls (e.g., Daugherty, Quay, & Ramos, 1993; Oosterlaan & Sergeant, 1996; Schachar & Logan, 1990).

A meta-analysis performed by Oosterlaan, Logan, and Sergeant (1998) found that children with ADHD have slower reaction times than controls (both on responses to the primary task and the derived SSRT measure). Children with ADHD also have a shallower inhibition slope than controls.

Stroop word-color association test.

The Stroop Word-Color Association Test (Stroop Test: Stroop, 1935) has three stages: first, the child must name the colours of rectangles arranged in rows across a page; second, the child must read the names of colours printed in black ink (e.g., 'RED,' 'GREEN' etc.); and finally, the child must name the colours that different colour words are printed in. For example, if the word 'RED' is printed in green ink, the correct answer is 'green.' It is this final stage in which the child must resist interference (from the prepotent response of reading the word). Considerable evidence indicates that children with ADHD make more errors, and take more time to complete the third (i.e., interference) part of the Stroop Test (Barkley, Grodzinsky, & DuPaul, 1992; Grodzinsky & Diamond, 1992; Leung & Connolly, 1996; Seidman et al., 1995; Seidman, Biederman, Faraone, Weber, & Ouellette, 1997; Shallice et al., 2002). This indicates that children with ADHD have difficulty inhibiting a prepotent response when faced with an interfering stimulus.

Matching familiar figures task.

The matching familiar figures task (MFFT: Kagan, 1966) is designed to measure the dimensional construct of reflection-impulsivity. The task involves the presentation of a picture of a familiar object (e.g., a tree) accompanied by a selection of very similar pictures that differ only in minor points of detail. The participant must select the picture that is identical to the target. Reaction times and correct responses are recorded. Participants that make rapid responses and a high number of errors are classified as impulsive whereas those

that make a slower responses and a low number of errors are classified as reflective (Kagan, 1966)⁹.

Children with ADHD are more likely than control children to make fast and inaccurate (i.e., impulsive) responses on the MFFT (Inoue et al., 1998). A similar pattern of differences has been found between adults with ADHD and psychiatric clinic controls (Young & Gudjonsson, 2005). In normal children, MFFT impulsivity is associated with elevated scores of inattentive and hyperactive/impulsive ADHD symptoms (Avila, Cuenca, Felix, Parcet, & Miranda, 2004).

Working Memory in Children with ADHD

Children with ADHD tend to perform worse on tests of both nonverbal memory span (Martinussen & Tannock, 2006; McInnes, Humphries, Hogg-Johnson, & Tannock, 2003; Tripp, Ryan, & Peace, 2002; Westerberg, Hirvikoski, Forssberg, & Klingberg, 2004) and nonverbal working memory (Martinussen & Tannock, 2006; McInnes et al., 2003; Tripp et al., 2002).

In a typical nonverbal memory task, such as Spatial Span (Wechsler et al., 2004) the experimenter taps a sequence of blocks and the participant is asked to repeat the sequence. Difficulty can be increased by increasing the number of taps required. Completing the task in normal order is thought to reflect nonverbal memory span (i.e., capacity), whereas completing the task in reverse order is thought to reflect nonverbal working memory (McInnes et al., 2003).

A typical verbal working memory task is the Digit Span test of the Wechsler Intelligence Scale for Children, Fourth Edition (Wechsler, 2003b). In this test, the child is

⁹Response times are classified as rapid or slow based on a median split as are the number of errors (High vs. Low).

read a sequence of numbers, and then asked to repeat the sequence back to the experimenter. Like the nonverbal working memory tasks, the level of difficulty is increased by increasing the number of digits presented. Digits repeated in normal order reflect verbal memory span. Digits repeated in reverse order reflect verbal working memory.

Children with ADHD are less able than control children to correctly recall sequences of digits in both forward and reverse order (Kilic, Sener, Kockar, & Karakas, 2007; Stevens, Quittner, Zuckerman, & Moore, 2002), although ADHD-control differences are not always found with forward digits (e.g., Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005).

In a recent meta-analysis, Martinussen, Hayden, Hogg-Johnson, and Tannock (2005) found consistent differences between children with ADHD and controls on both verbal and nonverbal working memory tests. These differences were found on both memory span (i.e., standard order) and working memory (i.e., reversed) tests. Overall, between group differences were greater on tests of nonverbal working memory than for tests of verbal working memory.

In a recent study examining both verbal and nonverbal working memory, children with ADHD were found to have lower scores than controls on tests of nonverbal working memory, whereas only younger children had low verbal working memory scores (Sowerby, Seal, & Tripp, 2010).

Response to Reinforcement in Children with ADHD

This thesis examines the response to rewards in children with ADHD. Consequently, it is necessary to examine some of the many different procedures that have examined the behaviour of individuals with ADHD in the context of various reinforcement conditions. Reinforcement can be manipulated in many ways, although the most commonly used manipulations involve changing the frequency of reinforcement associated with responses, or

altering the immediacy of reinforcement (i.e., by varying the delay that occurs between the response and reinforcer). Impulsivity is often used in ADHD literature to refer to rapid, uninhibited behaviour. In the behavioural literature, however, impulsivity is defined specifically as a preference for smaller, immediate reinforcers over larger, delayed reinforcers. Conversely, self-control is the preference for larger, delayed reinforcers over smaller, immediate reinforcers (e.g., Logue & Chavarro, 1992).

Effect of reinforcer rate and extinction.

Although individual studies vary considerably, there are several ways of arranging reinforcement: continuous reinforcement, where every response is reinforced; partial reinforcement, where only some responses are reinforced; and extinction, where no responses are reinforced.

Douglas and Parry (1994) used a procedure designed to measure both attention and frustration. Children were asked to choose heads or tails at the start of the task. Each trial began with a light, after which children were asked to press a lever to 'flip a coin.' Children were told that they would receive a reinforcer for each head or tail, depending on their initial choice. There was, in fact, no coin in the machine, each child received reinforcers on 100%, 50%, or 30% of trials, for 50 trials, followed by 20 trials of extinction. The reaction time to commence each lever press was taken as a measure of attention, with shorter RTs indicating better attention. The time taken to complete the lever press was taken as a measure of frustration (i.e., shorter time is evidence of faster or harder lever pulling). The attention of the control group was best (i.e., shorter RTs) when partially reinforced, whereas the attention of the ADHD group did not differ under different reinforcement conditions. The authors suggested that the partial schedules had a motivating effect on the control children, but not the children with ADHD. Additionally, the ADHD group pulled the lever harder than the control

groups in the extinction phase following the continuous reinforcement condition. The authors interpreted this as evidence that children with ADHD show higher levels of frustration than controls when expected reinforcers are withheld.

Douglas and Parry (1983) examined the reaction times of hyperactive and control children under continuous, partial, and non-contingent (i.e., reinforcers were delivered irrespective of reaction times) reinforcement. Each trial of the task began with a warning tone, upon which participants placed a finger on a key. When a light was displayed, participants removed their finger from the key. Testing was conducted over three phases of 15 trials each: baseline phase, during which no reinforcement was delivered; reinforcement phase, during which partial, continuous, or non-contingent reinforcers were delivered; and an extinction phase. In the continuous and partial reinforcement conditions participants received reinforcers if their reaction times were shorter than their median reaction times during baseline. Overall, control children made quicker responses than the hyperactive children over all three phases, although this difference was most marked in the partial and continuous reinforcement conditions. Control children also reduced their reaction times in all reinforcement conditions relative to baseline. During the extinction phase, reaction times of the control group remained quick relative to the ADHD group and their own baseline response rates. The hyperactive group, however, showed improved performance relative to baseline only in the partial and continuous reinforcement conditions, and showed a performance decrement (i.e., longer RTs) under non-contingent reinforcement. Additionally, the performance of the hyperactive group returned to baseline following the extinction phase regardless of reinforcement condition. The authors suggested that hyperactive children are unusually sensitive to rewards. It is worth noting, however, that hyperactive children showed a similar pattern of improvement to controls under partial and continuous reinforcement. Between group differences were clearest

under non-contingent reinforcement, which improved the performance of the control group, but diminished performance in the hyperactive group.

In a related study, Parry and Douglas (1983) examined the performance of hyperactive and control children on a concept identification task. During the task, children were asked to select one of two pictures (e.g., a flower or another object). Responses to the correct item (e.g., the flower) were reinforced. The task was completed when the child had completed 10 consecutive trials correctly. Under continuous reinforcement, hyperactive and control children required similar numbers of trials to complete the tasks. Control children took a similar number of trials to complete the tasks under partial and continuous reinforcement. The hyperactive children, however, took more trials to complete the task under partial than continuous reinforcement; that is, the reduction in reinforcer frequency led to a decline in performance in hyperactive children, but not in control children.

Aase and Sagvolden (2006) examined the effect of rate of reinforcement on sustained attention. Participants clicked a target (one of two squares) on a computer screen using a mouse. Correct responses were reinforced with small trinkets or coins according to a multiple VI 2-s VI 20-s¹⁰ schedule. Young (6-9 year old) children with ADHD, but not older children (9-12 year old) had deficient sustained attention (task accuracy) than controls when rewards were infrequent. When rewards were more frequent, however, there were no significant differences between groups. The authors concluded that young children with ADHD are more sensitive to reward contingencies than controls.

Sagvolden, Aase, Zeiner, and Berger (1998) compared the performance of children with and without ADHD under conditions of reinforcement and extinction. Children were

¹⁰ A variable interval, or VI schedule is a schedule of reinforcement where reinforcers are available after a variable interval. For example, a VI 20s schedule arranges reinforcers to be available every 20 seconds on average.

presented with a model of a clown's face. Responses (pushing the clown's nose) were reinforced on a multiple schedule. The fixed interval component was signalled by lights in the clown's eyes, and reinforcers were available every 30 seconds (i.e., a FI 30-s schedule). After a period of 2.5 minutes, the lights were turned off, signalling a two minute extinction period. This pattern was repeated six times. Response rates of children with ADHD increased over time compared with controls under FI reinforcement. A similar pattern occurred during extinction, with the response rates decreasing in controls, and increasing in the ADHD group over successive extinction periods. Sagvolden et al. suggested that the extinction period primarily measures sustained attention, which was deficient in the ADHD group. This pattern appears to be form of perseveration or impulsivity, or the inability to inhibit a prepotent (previously reinforced) response (c.f. Barkley, 2006a). Alternatively, this could be a form of local behavioural contrast, where a signal of a change in reinforcement contingencies (light switched off in this case) leads to a temporary suppression of responding, which then recovers to previous levels (Alsop, 2007).

Effect of relative rate of reinforcement.

Kollins, Lane, and Shapiro (1997) examined the effect of relative reinforcement rate on the response allocation of children with ADHD. Children pressed a left or right button to shoot aliens on the left or right side of the display. Left and right aliens were destroyed according to a concurrent schedule.¹¹ The schedules ranged from equal (VI 3s VI 3s) reinforcer density for each response to a VI 24s VI 3s schedule. Although this study was limited by its small number of participants (6 children with ADHD and 6 controls), there was some evidence of a difference in response to reinforcement between children with and without

¹¹ In a concurrent schedule, different reinforcement schedules are associated with each of two simultaneously available but mutually exclusive responses.

ADHD. When the ratio of left to right responses was compared with the reinforcers obtained for left and right responses (i.e., a matching law analysis, see Herrnstein, 1961) the control group showed a greater slope than the ADHD group, indicating a greater sensitivity to differences in reinforcer frequency.

In an experiment on which some of this thesis is based, Tripp and Alsop (1999) used a signal detection procedure to examine the effect of differences in reinforcer frequency on the behaviour of children with ADHD. Children distinguished between faces with small and large mouths. Correct identifications of each mouth type were reinforced differently; identifications of one mouth type received reinforcement at three times the rate of reinforcement for correct identifications of the other mouth type. Differences in bias (i.e., the tendency to make one response over another) emerged between groups. Although there were no overall differences in bias between the ADHD and control group, the two groups differed in their responses following reinforcers on the lean (i.e., less frequently reinforced) response. Children with ADHD showed less bias towards the frequently rewarded response than controls following lean reinforcers. The authors concluded that children with ADHD are strongly influenced by individual instances of reward compared with controls, who are more influenced by their overall reinforcement history.

Effect of reinforcer delay.

The previously discussed research examined the presence and absence of reinforcement, and the relative rate of reinforcement. Research has also examined the effect that delaying reinforcement has on subsequent behaviour.

Schweitzer and Sulzer-Azaroff (1995) examined the tendency of boys with ADHD to make impulsive choices in a forced choice procedure. Each trial consisted of a response made to either of two levers. Responses on one lever received a reinforcer immediately while

responses on the other lever received a larger reinforcer after a delay of 16 seconds. Children with ADHD showed a preference for the small immediate reward over the larger delayed reward. Control children showed the opposite tendency, preferring the larger delayed reinforcer.

Sonuga-Barke, Taylor, Sembi, and Smith (1992b) examined the effect of delay on choice in hyperactive children. In Experiment 1, children chose between an immediate small reward and a larger delayed reward. For the *no post delay* condition of the experiment, the next trial occurred immediately after the delivery of the previous reinforcer. In the *post delay* condition, a delay was imposed after each reinforcer, such that the overall delay was balanced for each response alternative. To maximise rewards, the most effective choices were to choose the small reward in the *no post delay* condition, and to choose the larger reward in the *post delay* condition. There were no significant differences between hyperactive and control children; both groups of children preferred the response alternative associated with the higher density of reinforcement.

In Experiment 2, Sonuga-Barke et al. (1992b) used a similar procedure to that used in the *no post delay* condition of *Experiment 1*. In the *trials constraint* condition, children made 20 responses per session; the most effective strategy in this condition was to select the larger delayed reinforcer. In the *time constraint* condition, sessions lasted for 10 minutes; the most effective strategy in this condition was to select the smaller immediate reinforcer. In the *time constraint* condition, both the hyperactive and control groups showed a similar preference for the smaller reinforcer. In the *trials constraint* condition, however, the control group showed a greater preference than the hyperactive group for the larger reinforcer. Sonuga-Barke concluded that the hyperactive children were not impulsive per se (i.e., they did not show a maladaptive sensitivity to pre-reward delay), but rather, hyperactive children were delay averse, in that they acted to reduce the overall delay (i.e., length of the task).

Neef et al. (2005) examined the effects of reinforcer rate, reinforcer immediacy, and reinforcer 'quality' on the choice between effortful and less effortful responses. Children with and without ADHD chose a succession of easy or difficult mathematical problems. Variables manipulated were reinforcer rate (VI 30s vs. VI 90s), reinforcer quality (preferred vs. least preferred tangible reinforcers) reinforcer immediacy (reinforcer received at end of session or the following delay), and response effort (easy versus difficult problems). In the first part of the study, both groups showed a preference for the expected problem set; that is, they tended to choose the easier problems, and those that resulted in frequent, high quality, and immediate reinforcers. In the second part of the study, problem dimensions were compared. For example, in one condition, easy problems were associated with low quality reinforcers that were delivered frequently, whereas hard problems were associated with high quality reinforcers delivered at a low rate. This manipulation allowed a ranking of each of the dimensions for each participant (e.g., Rate>Immediacy>Effort>Quality). For children with ADHD, reward immediacy had the greatest impact on choice, followed by quality, rate and effort. For the control children, reward quality had the greatest effect on choice, followed by immediacy, rate, and effort. Neef et al. concluded that the relatively high salience of reward immediacy in the ADHD group is evidence of impulsivity.

In another study on which this thesis is based, Tripp and Alsop (2001) used a signal detection approach to compare the effects of immediate and delayed reward in children with and without ADHD. The task involved distinguishing between faces with small and large mouths. Participants received token reinforcers for correctly identifying (via pressing one of two response keys) each of the two mouth types. Correct identifications of each mouth type were reinforced differently; identifications of one mouth type received immediate reinforcement (with a post-reinforcer delay of 3500 ms), whereas correct identification of the other mouth type received reinforcement after a 3500 ms delay. The main variable of interest

in this study was response bias, or the tendency to make one response over another. Children with ADHD showed a greater bias toward immediate reinforcement than control children. When trials occurring immediately following instances of delayed and immediate reward were examined, group differences emerged. Children in the control group showed lower accuracy and greater response bias (towards immediate reward) on trials immediately following instances of delayed reward. Children with ADHD showed similar levels of response bias irrespective of whether they had just received an immediate or delayed reward. Tripp and Alsop suggested that children with ADHD are unusually sensitive to pre-reward delays. It also appeared that the preference for immediate reward was more robust in the ADHD group, as their response bias was not influenced by their immediate reinforcement history, whereas the control group showed a greater preference for immediate reward following instances of delayed reward.

Theories of ADHD

Still (1902) was one of the first to describe in the scientific literature the set of behaviours synonymous with what is now known as ADHD. Still described a group of children with what he termed a defect of moral control, which he distinguished from a “disorder of intellect.” In modern parlance, Still’s defect of moral control subsumes concepts of altruism, inhibition, and adherence to social norms. Barkley (2006b) noted that the children described by Still would likely meet criteria for ADHD today, that the gender ratio (3M:1F) was similar to that found in children with ADHD today, and that additional behaviours described by Still, such as aggression and antisocial activity, were consistent with current observations of high rates of ODD and CD in children with ADHD. Since Still’s defect of moral control, there have been many theories that have attempted to explain ADHD.

Douglas' theory of deficient reinforcement mechanisms.

In one of the first papers to integrate clinical observations of hyperactive children with experimental data, Douglas (1972) summarised her research group's studies comparing hyperactive children with non-hyperactive children. Douglas proposed that hyperactive children had a number of cognitive deficits that could not be explained solely by their high activity levels. Specifically, Douglas suggested that hyperactive children have an impaired ability to "stop, look, and listen."

Douglas (1972) noted that 12-year-old hyperactive children obtained lower grades than controls in most academic subjects, and more frequently failed to obtain passing grades. Hyperactive children also obtained lower scores than controls on group-administered tests of academic achievement, particularly in mathematics and reading rate, but not on tests of comprehension and vocabulary. Hyperactive children obtained lower scores than controls on intelligence tests. Teacher ratings of behaviour highlighted consistent differences between hyperactive and controls on reports of frustration tolerance, concentration, and ability to organise activities. During classroom behavioural observations of hyperactive and non-hyperactive 7-year- and 12-year-old children, Douglas (1972) found that hyperactive children spent more time than controls engaged in behaviour unrelated to the classroom activity. Younger hyperactive children spent more time moving around the classroom, vocalised more often, and were more disruptive than controls. Older hyperactive children were more likely to engage in off-task behaviour while seated (e.g., playing with a toy, or working on the wrong task).

On a large battery of tests including measures of intelligence and clinical measures often used to identify various clinical populations, Douglas (1972) reported that most measures did not differentiate between hyperactive and control children. Although Douglas found that the hyperactive children's performance on intelligence tests was lower than that of

controls, the main differences were on aggregate measures of intellectual functioning and there were no reliable differences on individual subtest scores. Hyperactive children, did, however, obtain lower scores than controls on tests of visual motor co-ordination, and tests requiring fine- and gross- motor skills.

Douglas (1983) developed her original explanation of hyperactivity to propose four mechanisms to account for the array of difficulties experienced by hyperactive children: 1) poor investment and maintenance of effort; 2) deficient modulation of arousal to meet situational demands; 3) a strong inclination to seek immediate reinforcement; and 4) difficulties with impulse control. Douglas (1988) attributed these difficulties to deficient self-regulation. Specifically, Douglas suggested that hyperactive children have difficulties organising of information, poor mobilisation of attention (i.e., to deploy and maintain attention over time), and inadequate inhibition of responses (to inappropriate stimuli or reinforcers).

Douglas and colleagues (e.g., Sykes, Douglas, & Morgenstern, 1972; Sykes, Douglas, Weiss, & Minde, 1971) found that during CPTs, hyperactive children made more errors of omission and commission. This finding is consistent with the large body of evidence gathered on CPT performance of children with ADD/ADHD (e.g., Losier et al., 1996). Douglas's theory also predicts later findings that children with ADHD show a greater preference than children without ADHD to seek immediate reinforcement (e.g., Tripp & Alsop, 2001). Although Douglas was one of the first theorists to consider the role of reinforcement mechanisms in ADHD, her focus was on the immediacy of reinforcement. Additionally, Douglas's theory does not predict other findings regarding, for example, differences between ADHD and control children in response to differences in reinforcer rate (e.g., Tripp & Alsop, 1999).

Quay's theory of behavioural inhibition.

Quay (1988a, 1988b, 1988c, 1997) placed ADD/ADHD within the context of Gray's (1987, 1994) neuropsychological model of anxiety. Gray (1987, 1994) proposed three independent systems in learning and emotion: the behavioural activation system (BAS), behavioural inhibition system (BIS), and the fight/flight system. According to Gray, signals of positive and negative reinforcement activate the BAS, which initiates and maintains approach behaviour. Signals of positive and negative punishment activate the BIS, which inhibits or diminishes approach behaviour. The fight/flight system is activated by unconditioned aversive stimuli. Gray suggested that anxiety disorders are caused by an overactive BIS.

According to Quay's (1988a, 1988b, 1988c, 1997) theory, children with ADHD have an underactive BIS. This means that they are less sensitive to signals of punishment. This accounts for the failure of children with ADHD to inhibit responses in tests of inhibition such as the CPT (e.g., Losier et al., 1996), Stop Task (e.g., Oosterlaan et al., 1998), and Go/No-Go task (e.g., Iaboni et al., 1995).

Although Quay's theory accounts for findings on tests of inhibition, the focus of the theory on inhibition and punishment does not address differences between ADHD and control children under various reinforcement conditions (e.g., Aase & Sagvolden, 2006). According to Quay's theory, ADHD is caused by deficits in the BIS. Reinforcement, however, is governed by the BAS – a mechanism not purportedly implicated in ADHD. Additionally, if anxiety disorders arise from an over active BIS and ADHD arises from an under active BIS, ADHD and anxiety disorders would not occur concurrently. In fact, children with ADHD have relatively high rates of anxiety disorders (Szatmari et al., 1989).

Barkley's model of ADHD.

Barkley (1997a, 1999, 2006a) proposed a theory of ADHD based on disinhibition. Barkley groups ADHD symptoms into two clusters: inattention and hyperactivity/impulsivity (or disinhibition). Barkley suggests that these two dimensions are distinct, and subsequently points out that his theory applies only to cases of ADHD that contain a substantial disinhibition component (i.e., DSM-IV predominantly hyperactive-impulsive and combined types, but not predominantly inattentive type). In Barkley's model, the core impairment in ADHD is a deficit of response inhibition. Barkley defines behavioural inhibition as containing three interrelated processes: 1) inhibiting a prepotent response; 2) stopping an on-going response; and 3) interference control (preventing interference with the period of delay and self-directed behaviour that occurs following 2).

Barkley's (1997a, 1999, 2006a) evidence that children with ADHD have difficulty inhibiting prepotent and on-going responses is drawn from the previously discussed literature on tests of inhibition (e.g., Iaboni et al., 1995; Losier et al., 1996; Oosterlaan et al., 1998), and the Wisconsin Card Sorting Test (WCST: Heaton, 1981).

Barkley (1997a) suggested that the impaired performance of children with ADHD relative to controls on studies of the Stroop Word-Color Association Test (discussed earlier this chapter) is evidence of impaired control of interference of the competing response (i.e., colour naming).

According to Barkley (1999), deficits in inhibition lead to deficits in four executive functions: nonverbal working memory; verbal working memory; self-regulation of affect, motivation, and arousal; and reconstitution.

Barkley (1999) cites several findings as evidence that children with ADHD have deficits in nonverbal working memory. Children with ADHD have difficulty imitating a range of different motor tasks after a delay (e.g., Breen, 1989; Mariani & Barkley, 1997). Children

with ADHD also perform worse than controls on tests of spatial working memory (Martinussen et al., 2005; McInnes et al., 2003; Westerberg et al., 2004). Additionally, Barkley (1999) noted that time perception, an ability reliant on working memory, is impaired in children with ADHD (Barkley, Koplowitz, Anderson, & McMurray, 1997).

Barkley (1999) equates verbal working memory with internalisation of speech (i.e., audible self-talk). When compared to controls, children with ADHD have deficits in verbal working memory (e.g., Kilic et al., 2007; Stevens et al., 2002), and tend to use internalised speech less often than controls (Berk & Potts, 1991). According to Barkley, the delay in internalisation of speech leads to a delay in the privatisation of speech (i.e., inaudible or covert self-talk), which in turn leads to deficient ability use rules to regulate behaviour. The deficits in private speech and rule governed behaviour also lead to delayed moral reasoning, and hence moral development (Barkley, 1997b).

According to Barkley (1999), when children with ADHD experience negative emotion (e.g., boredom, anger, frustration, sadness), they are less able than control children to ameliorate these negative emotional states using internal processes such as imagery and private speech. Children with ADHD have been found to be more sensitive than controls to delayed rewards, in that they show a smaller preference than controls for larger rewards when the reinforcers are delayed (Rapport, Tucker, DuPaul, Merlo, & Stoner, 1986). Additionally, children with ADHD show increased levels of 'frustration behaviour' (i.e., harder lever pulling) during periods of extinction (Douglas & Parry, 1994). Barkley believes that this inability to tolerate negative emotion may cause children with ADHD to have an impaired ability to persistently maintain goal directed behaviour in the absence of immediate external sources of reinforcement.

Barkley (1999) defines reconstitution as the ability to generate novel combinations of pre-existing behaviours. Evidence that reconstitution is impaired in children with ADHD

comes from experiments in which children are required to generate novel responses. Children with ADHD have impaired verbal fluency relative to controls (Hurks et al., 2004; Koziol & Stout, 1992; Sandler et al., 1993; Tripp et al., 2002). There is also accumulating evidence that children with ADHD have difficulty generating novel nonverbal motor responses such as generating unique designs (Tripp et al., 2002). Similar deficits have also been found in adults with ADHD (Tucha et al., 2005).

Barkley's theory is perhaps one of the most broadly grounded theories, in that it accounts for many of the experimental findings from tests of inhibition (Corkum & Siegel, 1993) and working memory (e.g., Martinussen et al., 2005). The theory also predicts that children with ADHD are more sensitive to delayed reinforcement (due to increased levels of frustration). Barkley's theory does not, however, account for differences between children with and without ADHD when reinforcers are delivered immediately, but at different rates (e.g., Tripp & Alsop, 1999).

Sergeant's cognitive-energetic model of ADHD.

Sergeant (2000; Sergeant, Oosterlaan, & van der Meere, 1999) has proposed a model of ADHD in which the executive functions serve to modulate three 'pools' of cognitive energy (Arousal, Activation, and Effort), which, in turn, affect information processing.

Arousal is defined as phasic responding that is related to stimulus onset (Sergeant et al., 1999), and can be manipulated by varying the novelty and intensity of the stimulus. Novel or relatively intense stimuli are thought to induce higher levels of arousal. Physiological measures such as electrodermal response and change in heart rate (Pribram & McGuinness, 1975), and measures of perceptual sensitivity, or the ability to detect a signal in the presence of noise (Sanders, 1983), have all been purported to gauge arousal.

Activation directly influences motor organisation, and can be seen as a form of ‘readiness to respond.’ Factors believed to impact activation include task variables (e.g., warning signals increase activation), alertness (affected by variables such as tiredness and time-of-day), and time on task (Sergeant et al., 1999).

According to Sergeant’s model, Effort is the energy (i.e., neural activity) required to meet the demand of a task. Effort can increase or decrease arousal and activation, and can be manipulated by increasing the difficulty or cognitive load of the task. Effort has been measured indirectly via physiological responses that covary with task demands, such as pupil dilation (Granholm, Asarnow, Sarkin, & Dykes, 1996).

Sergeant’s (2000; Sergeant et al., 1999) theory offers an alternative interpretation of the CPT literature. He suggests that CPT errors indicate readiness to respond rather than disinhibition (e.g., Barkley, 1997a). High rates of omission errors coupled with low rates of commission errors indicate a conservative strategy (i.e., lower activation), whereas high rates of commission errors coupled with low rates of omission indicate a less conservative strategy (i.e., higher activation). Sergeant (1999) suggests that CPT error patterns should be examined over the course of the task. Commission errors early in the task would indicate over arousal, whereas omission errors towards the end of the task would indicate under arousal. There is a difficulty with Sergeant’s emphasis on high commission errors coupled with low omission errors, however, as children with ADHD have increased rates of omission *and* commission errors relative to controls (e.g., Corkum & Siegel, 1993; Losier et al., 1996).

Sergeant’s theory focuses on the neuropsychological mechanisms purported to cause ADHD, and questions traditional assumptions about how various experimental findings should be interpreted (e.g., Sergeant et al., 1999). Generally, Sergeant’s theory has a similar focus to Barkley’s (2006a) theory in that it focuses on executive functioning and disinhibition/activation. Like Barkley’s theory, Sergeant’s theory does not account for

differences between children with and without ADHD under different rates of reinforcement (e.g., Tripp & Alsop, 1999).

Sonuga-Barke's dual pathway model of ADHD.

Sonuga-Barke (2002; 2003; Sonuga-Barke, Dalen, & Remington, 2003) proposed a dual pathway model of ADHD in which delay aversion and executive dysfunctions make separate contributions to ADHD symptoms. Delay aversion refers to the tendency of children with ADHD to act to reduce the delay that occurs before reinforcers are delivered (Sonuga-Barke, Taylor, & Heptinstall, 1992a; Sonuga-Barke et al., 1992b). Sonuga-Barke's (2003) view of executive functioning is consistent with that of Sergeant (2000): executive functions modulate effort, arousal, and activation, which, in turn, affect information processing, and ultimately, behavioural output.

Sonuga-Barke's theory is essentially a synthesis of theories focusing on reinforcement (e.g., Sagvolden et al., 1998) and those focusing on executive functioning (e.g., Barkley, 2006a). The strength of the theory lies in its predictions regarding the neurological mechanisms underlying behaviour. According to (Sonuga-Barke, 2002) the delay aversion component of ADHD is a result of dysfunctions in mesolimbic circuits of the brain, whereas the executive dysfunction component is a result of dysfunction in the mesocortical circuits.

Sagvolden's developmental reinforcement model of ADHD.

Sagvolden's approach (e.g., Sagvolden, Aase, Johansen, & Russell, 2005; Sagvolden et al., 1998) differs from previous theories in that it treats the symptoms of ADHD as expressions of deficits in reinforcement mechanisms, rather than underlying constructs such as executive functioning (c.f., Barkley's theory). In Sagvolden's model, ADHD results from initial deficits in three (mesocortical, mesolimbic, and nigrostriatal) dopaminergic pathways, which can be a result of genetic endowment, and/or exposure to toxins or drugs. Sagvolden

also describes environmental influences such as parenting and societal influences that can affect the outcome of individuals with ADHD.

According to Sagvolden's model, deficits in the mesocortical dopamine system lead to poor attention and deficient behavioural organisation, deficits in the nigrostriatal system lead to clumsiness and poor non-declarative habit learning, and deficits in the mesolimbic system cause shorter delay-of-reinforcement gradients and deficient extinction processes.

Sagvolden's theory also generates predictions about how parental and environmental reinforcement affect children with ADHD. An optimal environment provides frequent, predictable, and immediate reinforcement of behaviour (Sagvolden et al., 2005). In the short-term, children with ADHD exposed to optimal conditions will likely be creative and impulsive, and impatient and eager; in a suboptimal environment, children will likely be oppositional and impulsive, frustrated when waiting, and delay averse (Sagvolden et al., 2005). In the long-term, children with ADHD exposed to optimal conditions will tend towards 'Type A' personality, will be easy-going with positive self-esteem, and will direct activity towards work. When raised in a suboptimal environment, children with ADHD will show antisocial behaviour and personality disorder, suffer from negative self-esteem and depression, and will underachieve in work and education (Sagvolden et al., 2005).

Central to Sagvolden's theory is mesolimbic dopamine system dysfunction, which results in both a shorter delay of reinforcement gradient and deficient extinction processes. Reinforcers that occur immediately following a behaviour exert a greater effect on behaviour than those that occur following a delay. The effectiveness of a reinforcer, when plotted against the immediacy of reinforcers, produces a delay of reinforcement gradient (e.g., Winstanley, Eagle, & Robbins, 2006). According to Sagvolden et al. (2005), the slope of this function is steeper in individuals with ADHD than without ADHD. This means that the behaviour of individuals with ADHD is less effectively shaped or maintained when

reinforcement is delayed. Sagvolden et al. also suggest that in individuals with ADHD, extinction is less effective. Individuals with ADHD are, therefore, more likely to persist with a behaviour that is no longer reinforced. Sagvolden et al. (2005) suggested that the hyperactive and impulsive behaviour exhibited by individuals with ADHD is a result of these impaired reinforcement and extinction processes.

Sagvolden's theory is based on reinforcement mechanisms, and, as such, accounts for more experimental differences between ADHD and controls under different reinforcement conditions than other theories do. According to Sagvolden's theory, the disinhibition seen in the behaviour of children with ADHD (including experimental evidence of disinhibition) is a result of deficient reinforcement mechanisms.

Dopamine transfer deficit theory of ADHD.

Tripp and Wickens (2008; 2009) have proposed a theory in which children with ADHD have a normal response to the delivery of reward, but diminished response to the signals that precede a reward. Tripp and Wickens note that dopamine tends to be released in short bursts following instances of reward. When an individual has sufficient experience on a task to be able to predict reward, dopamine is released earlier in response to signals that reward is likely to be delivered. According to this theory, in children with ADHD the dopamine is released in response to actual rewards, but is not released in response to reward signals. According to this theory, in normal children, when reward is delayed or inconsistent, reinforcement at the cellular level still occurs in response to reward cues. In children with ADHD, however, the anticipatory dopamine response is diminished or absent, leading to reduced effectiveness of rewards.

Summary and Conclusion

The theories proposed by Barkley, Sergeant, and Sonuga-Barke, and Sagvolden differ from the theories discussed earlier in the chapter (Still, Gray/Quay, and Douglas) in the level of detail contained in the theories. Specifically, Barkley, Sergeant, and Sonuga-Barke propose models with discrete mechanisms that are purported to cause individuals with ADHD to behave the way they do. Barkley's, Sergeant's and Sagvolden's models are all both predominantly hierarchical-serial in that they propose that an ultimate process(es) (disinhibition, cognitive energy, and reinforcement/extinction processes respectively) is/are disrupted in ADHD, leading to impairments in the dependent mechanisms. Sonuga-Barke's model differs in that two separable mechanisms, delay aversion and executive dysfunction make independent contributions to the behavioural symptoms of ADHD.

The Current Studies

Theories of ADHD that focus on reinforcements are hindered by the variability of experimental techniques in the literature. There are two main difficulties with this situation. First, different experimental techniques relate to different aspects of reward behaviour. Although examining different aspects of reward-related behaviour is useful, it is difficult to interpret what the range of findings actually means. Second, because a wide range of techniques have been used, some individual techniques have not been sufficiently replicated. This is a problem because until the techniques have been replicated, it is not certain if any apparent effects are 'real' or a result of publication bias.

This thesis is based primarily on the work of Tripp and Alsop (1999; 2001), and relates to reward-based theories and models of ADHD, particularly to the delay aversion component of Sonuga-Barke's (2002; 2003; Sonuga-Barke et al., 2003) dual pathway model of ADHD, Sagvolden's developmental reinforcement model of ADHD (e.g., Sagvolden et

al., 2005; Sagvolden et al., 1998), and Tripp and Wickens' (2008; 2009) dopamine transfer deficit theory of ADHD.

The signal detection technique pioneered by Tripp and Alsop offers three clear advantages over other techniques used to examine the influence of rewards on behaviour in children with ADHD. First, two of the measures, response bias and discriminability, are independent. Response bias is a measure of the influence of rewards on response allocation or preference, whereas discriminability measures task performance. Having both measures means that the effect of reward can be examined in relation to task performance. Second, Tripp and Alsop's signal-detection methodology provides a measure of reward sensitivity (response bias) that is less influenced by cognitive processes (e.g., executive functioning) than other techniques, such as simple choice procedures (e.g., Schweitzer & Sulzer-Azaroff, 1995; Sonuga-Barke et al., 1992b) where children choose between small-immediate rewards and large-delayed rewards. This distinction is important given that some theories of ADHD posit that reward mechanisms and cognitive processes independently influence ADHD symptoms (e.g., Sonuga-Barke, Sergeant, Nigg, & Willcutt, 2008). Third, there is an extensive literature that has used signal detection procedures with animals, therefore Tripp and Alsop's techniques may prove useful in examining animal models of ADHD (e.g., Sagvolden, 2000).

Although Tripp and Alsop's original studies revealed some promising findings, they suffered from a number of limitations. An obvious limitation is the small number of children (15 ADHD and 15 controls) in the study. This limited the power of the study to identify small effects, and also raises questions about the extent to which findings from this small group can be applied to the wider group of children with ADHD. The main difficulties of the study, however, related to the diagnostic procedure. First, Tripp and Alsop's (1999) study used DSM-III-R criteria, which do not distinguish diagnostically between primarily inattentive children and primarily hyperactive-impulsive children. Second, no information was provided

about how or if comorbid conditions (other than neurological disorders or psychosis) were assessed for. This is a particular problem given the many conditions that are often comorbid with ADHD. Third, diagnostic procedures likely varied considerably between clinicians. Children were assessed by either a child psychiatrist, clinical psychologist, or a paediatrician. There was no standardised assessment process, so it is likely that individual clinicians conducted assessments according to their own training and experience, thereby leading to increased variability in how symptoms were interpreted.

Tripp and Alsop's (2001) study contained a number of improvements over their 1999 study. The diagnostic procedure was standardised, teachers were interviewed about the children's behaviour, and comorbid disorders were assessed for. There were some problems however. Again, the number of participants was still fairly small (36 ADHD and 36 controls), and there was a marked discrepancy in the socioeconomic status between the two groups.

The two studies in this thesis attempt to replicate and extend the findings of Tripp and Alsop (1999) in Study 1 and (Tripp & Alsop, 2001) in Study 2. One of the difficulties in interpreting the literature ADHD-reward literature is the vast number of techniques used (see Luman, Oosterlaan, & Sergeant, 2005 for a review). Attempting to replicate initial findings using studies with good design and adequate sample sizes is vital to separating 'real' findings from artefacts of the publication process (Ozonoff, 2011). Replication of Tripp and Alsop's studies is particularly important given the limitations of their original studies and the advantages of the signal detection methods over other ways of examining reward behaviour described earlier.

The studies contained in this thesis contain a number of improvements over Tripp and Alsop's preliminary studies. The current studies use a relatively large sample of children with ADHD who were diagnosed using multiple-method multiple-informant assessments using well validated assessment tools. The children in the current studies were diagnosed according

to current (DSM-IV) criteria, which identify separate subtypes of ADHD. Additionally, comorbid conditions were also assessed. The ADHD and control participants in the current studies were also similar in terms of their socioeconomic status.

In addition to the factors related to study design and assessment techniques, the current study also contained a number of modifications to the signal detection procedure. The original studies used a task which was somewhat visually unexciting by today's standards. The current studies contain updated stimuli, which should make the task more appealing.

The current studies also aimed to increase the effectiveness of rewards in two ways. First, the current studies used a large number of brief (about 3 s) animated cartoons which were displayed as part of the rewards. Tripp and Alsop's studies used an animated fireworks display with each reward, which did not change throughout the task. It was hoped that the increased novelty of the current rewards would increase the salience of the rewards. Finally, Tripp and Alsop (1999) arranged for the frequently rewarded response to receive three times as many rewards as the infrequently rewarded response (i.e., a 3:1 ratio). Study 1 of this thesis uses a 4:1 ratio of rewards; it was hoped that this would also increase the effect of the unequal reward distribution. The changes to the rewards in the current studies should increase the probability of finding significant between-group differences.

Chapter 3: Main Method

Participants

The participants were 201 boys and 87 girls aged between six and twelve years of age recruited through Otago schools and the Paediatric Outpatients Department and the Child and Family Mental Health Services of the Otago District Health Board (ODHB). One hundred and nine boys and twenty six girls were referred for assessment related because their ODHB clinician believed they may have symptoms of ADHD. The control group consisted of ninety one boys and sixty one girls recruited from Otago schools. Table 1 shows the clinical, cognitive, and demographic characteristics of the participants. The majority of participants completed both studies, although some participants were excluded from analysis for one or both of the studies for various reasons. Details about excluded participants are discussed in Chapters 4 and 6.

Signal detection equipment.

The signal detection tasks were run using two IBM compatible computers: a laptop computer attached to a separate 15 inch colour monitor; and a desktop computer with a 15 inch colour monitor.

Two response panels were connected to each computer using a custom interface. The response panels measured 30 cm in width, 8 cm in height, and 8 cm in depth. Two 4 cm diameter response buttons were located on the top of the response panel 4 cm from each end, 22 cm apart. The left and right buttons were coloured red and blue respectively. Stimuli consisted of a 10X10 array of alien face characters. The characters measured approximately 9 mm X 9 mm and were separated by their horizontal and vertical neighbours by 3 mm. The array as it appeared on the screen measured approximately 11.7 cm X 11.7 cm

Table 1.

Clinical, Cognitive, and Demographic Characteristics.

	ADHD-C ¹	ADHD-PIA ²	ADHD-PHI ³	Diagnostic Exclusions ⁴	Normally Developing
n per group	78	29	3	26	152
n Males (%)	68 (87)	21 (72)	3 (100)	18 (69)	91 (60)
Mean age in years (SD)	7.9 (1.9)	9.0 (1.6)	7.7 (1.9)	8.8 (2)	8.9 (1.7)
Ethnicity⁵					
n NZE (%)	61 (78)	24 (83)	3 (100)	20 (77)	91 (60)
n NZM (%)	2 (3)	1 (3)	0 (0)	1 (4)	0 (0)
n NZE and NZM (%)	5 (6)	3 (10)	0 (0)	3 (12)	16 (11)
n All Other (%)	10 (13)	1 (3)	0 (0)	2 (8)	17 (11)
Socioeconomic Status					
Mean NZDep2006 ⁶ (SD)	1021 (107)	1004 (99)	889 (13)	986 (71)	1002 (77)
Mean Income Category ⁷ (SD)	3.2 (2)	3.6 (1.9)	4.3 (0.6)	2.9 (2)	4.4 (1.8)
Mean BASC Ratings⁸					
PRS: Hyp. (SD)	73 (21)	67 (14)	74 (18)	67 (25)	45 (11)
TRS: Hyp. (SD)	60 (17)	54 (9)	64 (5)	54 (16)	43 (9)
PRS: Att. (SD)	66 (17)	71 (6)	64 (5)	64 (18)	47 (11)
TRS: Att. (SD)	61 (16)	63 (3)	55 (2)	56 (14)	43 (10)
Comorbid Disorders					
n ODD (%)	46 (59)	20 (69)	2 (67)	13 (50)	
n CD (%)	8 (10)	2 (7)	0 (0)	1 (4)	
n Learning Problems (%)	16 (21)	12 (41)	0 (0)	7 (27)	
n Missing LP Data (%)	12 (15)	1 (3)	0 (0)	2 (8)	
n prescribed medication (%)	8	3	0	0	
IQ Scores					
Mean FSIQ (SD)	86 (16)	85 (15)	102 (17)	89 (16)	
Mean ESTIQ (SD)	90 (15)	89 (16)	101 (9)	92 (17)	103 (14)
n FSIQ<70 (%)	11 (14)	5 (17)	0 (0)	2 (8)	
n FSIQ 70+ (%)	67 (86)	24 (83)	3 (100)	24 (92)	
n ESTIQ<70 (%)	6 (8)	2 (7)	0 (0)	2 (8)	1 (1)
n ESTIQ 70+ (%)	72 (92)	27 (93)	3 (100)	24 (92)	151 (99)

¹ ADHD Combined Type

² ADHD Predominantly Inattentive Type

³ ADHD Predominantly Hyperactive-Impulsive Type

⁴ Participants were included in this group if they did not meet DSM-IV criteria for ADHD, or on the basis of the DSM-IV ADHD exclusion criterion E.

⁵ NZE=New Zealand European, NZM=New Zealand Maori. The ethnic groups reported here are mutually exclusive. Participants endorsing more than two ethnic groups are included in 'All Other' even if NZE and NZM were endorsed. There were 28 Children in the control group for which ethnicity information was not completed.

⁶ NZDep2006 Index of Deprivation (Salmond, Crampton, & Atkinson, 2007) scores have a mean of 1000 and an SD of 100. Higher scores indicate higher levels of deprivation.

⁷ 1=\$10-20,000; 2=\$20-30,000; 3=\$30-40,000; 4=\$40-50,000; 5=\$50-60,000; 6=>\$60,000. These figures are gross per annum incomes.

⁸ Behaviour Assessment Scale for Children (Reynolds & Kamphaus, 1992). PRS=Parent Rating Scale. TRS=Teacher Rating Scale. Hyp.=Hyperactivity. Att.=Attention Problems.

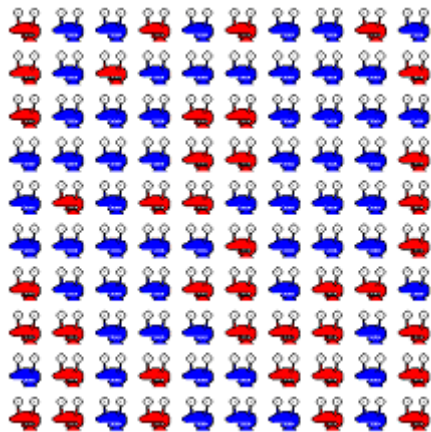


Figure 1. A typical stimulus array containing more blue than red characters.

square. The characters were red and blue, with the proportion of red and blue characters manipulated to alter the difficulty of the detection task. An animated juggler character was used to signal the start of each trial. The juggler character appeared in the centre of the screen and measured 10 mm high by 7 mm wide. A typical stimulus array is shown in Figure 1.

Coloured circular plastic tokens were used as reinforcers. At the end of the task the tokens could be exchanged for a prize. Prizes were of approximately \$5.00 value and included stationery, stickers, art supplies, and small age-appropriate toys.

Apparatus

Clinical and behavioural assessment measures.

Interview schedule.

The Schedule for Affective Disorders and Schizophrenia, Present and Lifetime Version (K-SADS: Kaufman et al., 1997; Kaufman & Schweder, 2004) is a semi structured diagnostic interview that is consistent with DSM-IV criteria. The K-SADS was administered to children and parents from the clinically referred group only.

The K-SADS contains five diagnostic supplements: *affective disorders*, *psychotic disorders*, *anxiety disorders*, *behavioral disorders*, and *substance use and other disorders*. Items from the screen interview and diagnostic supplements from the K-SADS are scored on 0-2 scale for items where a symptom is judged as being either present or absent (e.g., drug use): a rating of 1 means that the symptom is not present, while a rating of 2 indicates that the symptom is present. Items that require a clinical judgement regarding the significance of the symptom (e.g., low mood) are scored on a 0-3 scale: a rating of 1 means that the symptom is not present; a rating of 2 indicates that the symptom is present, but not to a clinically significant degree; and a rating of 3 means that the symptom is present to a clinically significant level. Each item (on both 0-2 and 0-3 scales) for which no information was available was scored 0. Symptoms are rated both at the present time, and their most severe past level. This allows the K-SADS to generate both present and lifetime diagnoses.

Kaufman et al. (1997) examined the psychometric properties of the K-SADS in a study of 66 children and adolescents that included both psychiatric and normal controls. Inter-rater reliability was examined in a sub-group of 15 randomly selected participants. Average agreement for the screen interview items was 99.7%. Test-retest reliability (Kappa statistic) over an interval of 18 days ranged from good to excellent for current diagnoses and fair to excellent for lifetime diagnoses according to Landis and Koch's (1977) criteria. For ADHD, the test-retest reliability was good and fair for current and lifetime diagnoses respectively. Concurrent validity was examined by comparing the scores on a number of rating scales of participants who screened positive with those who screened negative for K-SADS diagnoses. Children who screened positive for ADHD scored significantly higher on a commonly used measure of ADHD symptoms (Conners Parent Rating Scale: Conners & Barkley, 1985).

Behaviour rating scales.

The Behavior Assessment System for Children (BASC: Reynolds & Kamphaus, 1992) is a rating scale designed to measure both adaptive and problem behaviours in children and adolescents. The current study used the parent (PRS-C and PRS-A) and teacher (TRS-C and TRF-A) report forms. Each BASC item is a description of a behaviour that is rated on a 4-point Likert scale. The BASC contains 10 problem scales (Hyperactivity, Attention Problems, Aggression, Conduct Problems, Anxiety, Somatization, Depression, Withdrawal, Learning Problems, and Atypicality¹²). The 10 individual problem scales are grouped into three composite problem scales (Externalizing Problems, Internalizing Problems, School Problems) and a Behavioral Symptoms Index. The BASC contains 4 adaptive scales (Adaptability, Social Skills, Leadership, and Study Skills). Scores for each scale are summed and then compared to general, male, female, or clinical normative groups. Standard scores ($M=50$, $SD=10$) and percentile scores are provided.

Evidence of the reliability of the BASC TRS and PRS is contained in the manual (Reynolds & Kamphaus, 1992) and includes studies of internal consistency, test-retest reliability, and inter-rater reliability. Internal consistency (coefficient Alpha) for the TRS ranged from 0.62 to 0.95 for the problem and adaptive scales, and from 0.78 to 0.97 for the composite scales. Alpha values on the PRS ranged from 0.42 to 0.89 for the problem and adaptive scales, and from 0.85 to 0.94 for the composite scales. Alpha values tended to be somewhat higher in groups from which the clinical norms were derived.

Test-retest reliabilities over intervals of between 2 and 8 weeks ranged from 0.59 to 0.95 for the TRS problem scales and from 0.89 to 0.95 for the TRS composite scales. On the PRS, test retest reliabilities ranged from 0.41 to 0.92 for the problem scales and 0.71 to 0.94

¹² The Atypicality scale measures behaviours that are generally considered odd, unusual, or immature for a child's age, such as 'Chews clothing and blankets.'

on the composite scales. Inter-rater reliabilities were somewhat lower than other methods of computing reliability, and ranged from 0.44 to 0.93 for the TRS problem scales and 0.69 to 0.89 for the TRS composite scales. Inter-rater reliabilities on the PRS ranged from 0.44 to 0.93 for the problem scales and 0.46 to 0.73 for the composite scales. As noted by Reynolds and Kamphaus, inter-rater reliability can be low for a number of reasons including differences in how raters interpret items, differences in a child's behaviour over time, and the confounding of rater with environment (i.e., different raters observe the child in different settings).

Reynolds and Kamphaus (1992) reported two main sources of evidence of validity of the BASC TRS and PRS: the correlation of PRS and TRS scales with other instruments (i.e., concurrent validity), and profile analysis of clinical groups. The study of concurrent validity found that BASC PRS scores were highly correlated with the Child Behaviour Checklist (CBCL: Achenbach, 1991a) scales and the externalising scales of the Conners' Parent Rating Scales (CPRS: Conners, 1989); and moderately correlated with the Personality Inventory for Children-Revised (PIC-R: Lachar, Gdowski, & Snyder, 1982) and Behavior Rating Profile (BRP: Brown & Hammill, 1983) scores. The BASC TRS scales were highly correlated with their analogue scales on the Teacher's Report Form (TRF: Achenbach, 1991b), Revised Behavior Problem Checklist (RBPC: Quay, 1983), and Burks' Behavior Rating Scales (BBRS: Burks, 1977). The BASC TRS was moderately correlated with the Conners' Teacher Rating Scales (CTRS: Conners, 1989) and BRP were moderate. Correlations were generally higher between scales measuring externalising behaviours and school related problems.

The Disruptive Behaviour Disorders Rating Scale (DBD: Molina, Smith, & Pelham, 2001; Pelham, Evans, Gnagy, & Greenslade, 1992a; Pelham, Gnagy, Greenslade, & Milich, 1992b; Pelletier, Collett, Gimpel, & Crowley, 2006) was used to measure symptoms of ADHD, oppositional defiant disorder (ODD), and conduct disorder (CD) in the control and

clinic groups. The DBD is a 45 item symptom checklist that contains all of the DSM-IV symptoms of ADHD, ODD, and CD. Each item is rated as being present “Not at all,” “Just a little,” “Pretty much,” or “Very much.” Symptoms are treated as present if either of the latter two ratings is applied.

Evidence of the reliability of the DBD includes studies of inter-rater agreement and internal consistency. When diagnoses were assigned based on the DBD ratings of teachers there is fair agreement (Kappa) between raters for all three of the ADHD diagnoses and moderate agreement for the diagnoses of ODD and CD (Molina, Pelham, Blumenthal, & Galiszewski, 1998). Internal consistency estimates based on the DBD ratings of teachers were acceptable for the inattentive and hyperactive-impulsive symptoms, and good for symptoms of ODD and CD.

Evidence for the validity of the DBD comes from factor analytic studies and comparisons with other measures. Molina et al. (1998) found that the DBD had medium to large correlations with a number of other measures of classroom behaviour. Several factor-analytic studies have supported the structure of the DBD in which there are two ADHD factors (hyperactivity-impulsivity and Inattention) and two further “childhood aggressive disorders factors, which correspond to diagnoses of ODD and CD (Molina et al., 2001; Pelham et al., 1992a; Pelham et al., 1992b; Pillow, Pelham, Hoza, Molina, & Stultz, 1998).

Measures of cognitive and academic functioning.

The Australian language adaptation of the Wechsler Intelligence Scale for Children, Fourth Edition (WISC-IV: Wechsler, 2003a) and the Wechsler Primary and Preschool Intelligence Scale, Third Edition (WPPSI-III: Wechsler, 2002a) were used to measure cognitive functioning. Both of these tests comprise a number of individual subtests (15 in the WISC-IV and 14 in the WPPSI-III) that yield standard scores based on age norms. The

standard scores for each subtest are combined to yield various composite measures. Although both tests yield a Full Scale IQ (FSIQ) score, the underlying structures of the two tests are different. The WISC-IV uses a four factor model in which individual subtests measuring related skills are grouped to yield four index scores: Verbal Comprehension (VCI), Perceptual Reasoning (PRI), Working Memory (WMI) and Processing Speed (PSI) indices. In the WPPSI-III, groups of subtests are combined to yield verbal and performance IQ scores (VIQ and PIQ respectively) in addition to the FSIQ. Processing Speed (PSQ) and General Language (GLC) composites can also be derived. The WPPSI-III is designed for use with children aged between 2 years, 6 months and 7 years, 3 months. The WISC-IV is designed for use with children and adolescents aged between 6 and 17 years of age.

The WISC-IV and WPPSI-III both have good psychometric properties. For the WISC-IV, stability coefficients of index and FSIQ scores ranged from 0.84 to 0.96 (Wechsler, 2003b). When the WISC-IV scores of children with ADHD (all subtypes) were compared with control children, children with ADHD obtained significantly lower scores on a number of subtests and indices (Wechsler, 2003b). The PRI and contributing subtests showed minimal differences between ADHD and controls, whilst the PSI and WMI and contributing subtests showed the greatest differences.

The stability coefficients for the WPPSI-III range from 0.80 to 0.97 for the composite/IQ scores for children in the standardisation sample. When children with ADHD were compared with matched controls selected from the standardisation sample, subtest, IQ, and index/composite scores were similar in both groups. The only significant difference between groups was on the Comprehension subtest, where children with ADHD obtained lower scores than controls (Wechsler, 2002b).

The Wechsler Individual Achievement Test- Second Edition- Abbreviated (WIAT-II-A: The Psychological Corporation, 2001) was used to measure the academic achievement of

the clinical participants, and to assess for learning disabilities. The WIAT-II-A provides age normed scales scores for Word Reading, Spelling, and Numerical Operations. Scores from the individual subtests can be aggregated to provide an abbreviated composite score. The internal consistency reliability of the individual subtests ranged from 0.91 to 0.99 for children of the same age range as used in the current study. The WIAT-II-A subtests are reliable (0.91 to 0.99 over an average interval of 10 days) and are correlated with a number of different measures of academic achievement as well as age and estimated grade level (The Psychological Corporation, 2001). When actual WIAT-II-A scores were significantly lower than predicted based on the WISC-IV FSIQ scores, participants were identified as having a possible learning disability (Wechsler, 2003b).

Socioeconomic status.

The NZDep2006 Index of Deprivation (Salmond, Crampton, & Atkinson, 2007) is a measure of deprivation based on 2006 census data. Participants' addresses were compared with a national database to provide deprivation scores. NZDep2006 deprivation scores are standard scores with a mean of 100 and a standard deviation of 100, with higher scores indicating a higher level of deprivation.

Signal detection measures.

Three parameters were derived from the signal detection tasks: response time (RT), Response Bias ($\log b$), and Discriminability ($\log d$). In the signal detection tasks participants identified whether there were more red or blue characters in an array. Each trial presented an array containing more red than blue characters or a matrix containing more blue than red characters (see Figure 1). There were two response alternatives ('red' or 'blue'). Figure 2 shows the possible combinations of correct and incorrect responses for the two stimulus types. When a stimulus array containing more red than blue characters was presented, 'red'

	Correct	Incorrect
Red	Red _{Correct}	Blue _{Incorrect}
Blue	Blue _{Correct}	Red _{Incorrect}

Figure 2. Possible correct and incorrect responses for each stimulus type.

responses were counted in the Red_{Correct} cell, and ‘blue’ responses were counted in the Blue_{Incorrect} cell. Conversely, when a stimulus array containing more blue than red characters was presented, ‘red’ responses were counted in the Red_{Incorrect} cell, and ‘blue’ responses were counted in the Blue_{Correct} cell.

The response bias ($\log b$) and discriminability ($\log d$) measures are derived from the behavioural model of signal detection (e.g., Davison & Tustin, 1978; McCarthy & Davison, 1981). $\log b$ was calculated using the following formula as used by (Tripp & Alsop, 1999).¹³

$$\log b = \frac{1}{2} \log \left[\frac{Red_{Correct} \times Red_{Incorrect}}{Blue_{Correct} \times Blue_{Incorrect}} \right] \quad (1)$$

Discriminability is a measure of accuracy on the signal detection task and is calculated according to Tripp and Alsop’s formula below.

$$\log d = \frac{1}{2} \log \left[\frac{Red_{Correct} \times Blue_{Correct}}{Red_{Incorrect} \times Blue_{Incorrect}} \right] \quad (2)$$

Procedure

The studies described in this thesis were approved by the Lower South Regional Ethics Committee.

¹³ Because the number of responses in some cells was occasionally zero, 0.5 was added to each cell.

Recruitment of clinically referred participants.

The Paediatric Outpatients Department and Child and Family Service of the Otago District Health Board were provided with information about the current research. Clinicians from these services provided information packs containing a permission to contact form (see Appendix A) to the families of children who were likely to have ADHD. Parents who wished to participate in the study returned the completed permission to contact form to the researchers. Families were then contacted and any questions about the research or assessment process were addressed. Families wishing to participate were first given consent forms (see Appendix A), behaviour rating scales (BASC and DBD) and an appointment letter with directions to the appointment location along with a reply-paid envelope. Assessment sessions were then scheduled for the parent(s) and child.

Assessment of clinically referred participants.

The assessment of all clinically referred participants was consistent with recent best practice guidelines (e.g., AACAP Work Group on Quality Issues, 1991, 1997; AACAP Work Group on Quality Issues, 2007; American Academy of Paediatrics Committee on Quality Improvement Subcommittee on Attention-Deficit/Hyperactivity Disorder, 2000). All participants underwent multiple-method, multiple-informant assessments which were conducted by a supervised postgraduate student in clinical psychology or a doctoral level registered clinical psychologist. All assessments comprised a developmental history interview, and semi structured clinical interviews (K-SADS) conducted separately with the participant and the participant's parent. Behaviour rating forms (BASC and DBD) were completed by the parents and teachers. The majority of interviews were conducted with the participants' mothers only, and occasionally with the participants' mother and father together, or father only. Teachers were also interviewed by telephone in order to collect information

regarding the child's behaviour in the classroom setting, social functioning, and academic performance. Cognitive assessments (WISC-IV or WPPSI-III, and WIAT-II-A) were completed for the majority of participants.

Participants were diagnosed according to DSM-IV criteria using information from the K-SADS structured clinical interview which was interpreted together with the behaviour rating forms and remaining interview data.

The DSM-IV criteria for ADHD state that a child must exhibit six or more symptoms of inattention and/or hyperactivity-impulsivity for a period of six months or more (Criterion A). For Criterion A, the number of symptoms was taken as met if six or more symptoms were present at home *or* at school, and some symptoms were also present in the other setting. Symptoms were not summed across settings. Symptoms were taken to be present at home if the requisite number of symptoms were reported by K-SADS interview or endorsed on the DBD. Symptoms were taken as present at school if the requisite number of symptoms were reported during the teacher interview or endorsed on the DBD. Criterion B states that the symptoms must have been present since before the age of 7 years. Information about the onset and duration of inattentive and hyperactive/impulsive symptoms relevant to Criteria A and B was collected during the K-SADS interview.

The DSM-IV criteria also require that there must be some impairment in two or more settings (Criterion C), and that the impairment is clinically significant (Criterion D). The impairment required for Criterion C and D to be met was judged to be present if significant difficulties related to inattention or hyperactivity-impulsivity were reported by either the participant, his parent or teacher, or if symptoms were perceived to be causing difficulties during the clinical interview or cognitive assessment.

Criterion E requires that the symptoms do not occur exclusively during a pervasive developmental disorder or psychotic disorder and are not better explained by the presence of

other disorders (e.g., mood or anxiety disorders). These other disorders were screened for using the K-SADS interview and cognitive assessment, and participants who met DSM-IV criteria for these disorders were excluded from the ADHD groups.

Once the assessment and signal detection tasks were completed a summary report was written that included any relevant diagnoses. Copies of this report were sent to the clinician who had originally provided parents with the information packs for the current research. The clinician gave the parent(s) and child feedback about the research assessment and provided the family with copies of the reports. Parents were given a \$10 petrol voucher each time they attended. Once the child's teacher had completed the interview and returned the behaviour rating forms, the teacher was sent a \$15 book voucher.

All assessments were completed, and reports written by either a registered clinical psychologist, or by a postgraduate clinical psychology student supervised by a registered clinical psychologist.

Recruitment of control participants.

The control group participants were recruited through a number of Otago schools. The principals of these schools were approached and a meeting was arranged to discuss the nature and purpose of the research. Schools that wished to participate were given a number of information packs that contained information regarding the research and a permission to contact form (see Appendix A). These information packs were passed to individual teachers who were asked to give them to children to take home to their parents if the children had no apparent behaviour or emotional problems. Once the families had completed and returned the permission to contact forms, the school principals were contacted to schedule school visits to meet with the participants and complete the signal detection tasks. Parents were then sent a consent form (see Appendix A), behaviour rating forms (BASC and DBD), and a reply paid

envelope. Once parents had returned the forms, a copy of the consent form was provided to the child's teacher. The teacher was then given an information sheet (see Appendix A) and asked to complete a consent form and behaviour rating forms (BASC and DBD). Once teachers completed the forms, they were given a \$10 book voucher.

Assessment of control participants.

Participants assigned to the control group were assessed using an abbreviated version of the techniques used with the ADHD group. Participants' parents and teachers completed BASC and DBD rating scales. Participants completed a cognitive screening assessment which comprised the Vocabulary and Matrix Reasoning subtests from the WISC-IV. Scores from the WISC-IV subtests were used to estimate a WISC-IV FSIQ score using data provided by Sattler and Dumont (2004). Participants were included in subsequent analyses if they had an estimated FSIQ scores of 70 or higher, fewer than 4 inattentive or hyperactive/impulsive symptoms endorsed on the DBD, and BASC Hyperactivity and Hyperactivity T scores below 60.

Signal Detection Procedure.

Each child completed two signal detection tasks in which they identified whether a stimulus array contained more red or blue characters. In Study 1 correct responses of each type were associated with different probabilities of reward. In Study 2, the probability of reward was equal for correct responses of each type but responses of one type were rewarded immediately, whereas correct responses of the other type were rewarded after a brief delay. Because the procedure was different in each study, the procedures for each study are discussed separately in the following chapters. There were some participants whose signal detection data indicated that they had not completed the tasks in the expected manner. These exclusions are described in Chapters 4 and 6.

Counterbalancing.

Although pilot testing had not indicated that there were likely to be any stimulus/place or order effects, between subjects counterbalancing was arranged to control for these effects should they occur. Each participant completed two signal detection tasks (Study 1 and Study 2). Two versions of each signal detection task were used. In Version 1 of Study 1 (Study 1 V1), participants received more frequent rewards when they correctly pressed the ‘Blue’ (right) button than when they pressed the ‘Red’ (left) button. The reverse was true in Study 1 V2.

In Study 2 V1, participants received immediate rewards when they correctly pressed the ‘Blue’ (right) button and delayed rewards when they pressed the ‘Red’ (left) button. The reverse was true in Study 2 V2.

Each participant was quasi-randomly assigned to an order/version condition and completed the two studies in one of the following orders: Study 1 (V1) then Study 2 (V2); Study 1 (V2) then Study 2 (V1); Study 2 (V1) then Study 1 (V2); or Study 2 (V2) then Study 1 (V1). No participants completed the tasks in an order where each study would likely develop a preference for the same stimulus, such as Exp 1 (V1) then Exp 2 (V1).

The 11 participants who had been previously prescribed stimulant medication completed each signal detection task twice over two sessions. In one session, medication had been temporarily discontinued for 24 hours before the experimental session. In the other session both experimental tasks were completed when the child had been taking their medication as prescribed. Whichever order/version condition they were initially assigned to was repeated in the second session. The medication order was counterbalanced between participants so that similar number of participants completed the first session on and off medication.

Chapter 4: Sensitivity to Reward Frequency Method and Results

Method

Participants.

From the initial pool of participants described in Chapter 3, data from three groups of male participants were analysed: Forty one boys with combined type ADHD (ADHD-C), 13 boys with predominantly inattentive ADHD (ADHD-PIA), and 70 normally developing boys (ND). A number of participants (16 ADHD-C, 4 ADHD-PIA and 9 ND)¹⁴ were excluded due to unusual performance on the signal detection task (discussed below). Data from the female participants was not included in the analyses as boys and girls may have showed different patterns of performance but there were too few with ADHD to conduct sufficiently powerful analyses. Age and estimated IQ (ESTIQ) were not matched between groups. Because of the relatively small number of participants in the ADHD-PIA group, this was excluded from the main analyses, but is examined briefly towards the end of the chapter.

Apparatus.

As described in Chapter 3.

Procedure.

Participants sat at a desk approximately 40 cm in front of the screen with their heads approximately level with the top of the screen. The response panel was on the desk between the participant and the screen. The experimenter sat to the right of the child, facing the same screen as the child.

¹⁴ These are the participants excluded based on SDT performance only. Other participants have been already excluded for other reasons. These exclusions have been discussed in Chapter 3.

The task began with instructions and two practice trials. All text on screen was read aloud by the experimenter, and clarification was given where necessary. If the participants made incorrect responses on either of the practice trials, the experimenter corrected the participants verbally. The instructions began with the text *“Hi, This is a simple computer game. You will see some patterns of blue greeblies and red greeblies. You must decide if there are more blue ones or red ones, and press the blue or red button. Here’s an example”* appearing on the screen in coloured text approximately 5 mm high. Once the instructions had been read aloud the first practice trial began. The trial was signalled by a small juggler character appearing in the centre of the screen for 750 ms. After a delay of 250 ms, a stimulus array containing 60 blue and 40 red characters then appeared for 2000 ms. After the stimulus array disappeared, the text *“Press the more blue button”* appeared and was read aloud by the experimenter. When the participant pressed the blue button, the experimenter activated the next screen using the mouse. The text *“Great, now try another one!”* appeared and the experimenter activated the second trial.

The second practice trial was identical to the first, except the ratio of colours on the stimulus array were reversed (i.e., there were 60 red and 40 blue characters). After the stimulus presentation, the text *“Press the more red button”* appeared and was read aloud by the experimenter. Once the participant pressed the red button the experimenter activated the following screen which read: *“Sometimes, but not always, you can win a token for being right. You never get a token for being wrong. Win lots of tokens and you get a prize. When you get a token it looks like this -”*. An animated cartoon then appeared, a brief musical tone played, and the experimenter placed a token in the clear plastic container. The experimenter then activated the following screen, which read *“Are you ready to start? Remember that you don’t always get a token when you are right – Just sometimes.”* When the participant indicated that he was ready, the experimenter activated the 180 experimental trials.

Each trial began with the animated juggler character appearing in the centre of the screen for 750 ms, followed by a blank white screen for 250 ms. This was followed by a 2000 ms presentation of the stimulus array. After the stimulus presentation, the screen remained white with no characters present until a response was made. If the response was correct and scheduled to be rewarded, then the reward was delivered. After the reward there was a delay of 750 ms before the next trial. If the response was incorrect or not scheduled to be rewarded, the next trial began after a delay of 1750 ms.

Over the first 60 trials, a titration procedure adjusted the difficulty of the task by altering the proportion of blue to red characters in the stimulus array. This procedure attempted to ensure that each participant would achieve similar levels of accuracy on the task. The initial difficulty level of the task was set to 58:42 (i.e., 58 blue characters and 42 red characters or vice versa). After 10 trials, the proportion correct was calculated. If accuracy was greater than 90%, the level of difficulty increased by two (e.g., from 58:42 to 56:44); if the accuracy was between 80% and 90%, the difficulty increased by one (e.g., from 58:42 to 57:43). If accuracy was between 60% and 69%, the difficulty level decreased by 1 (e.g., from 58:42 to 59:41), and if the accuracy was less than 60% the difficulty level decreased by two (e.g., from 58:42 to 60:40). If the accuracy was between 70% and 80% there was no change made to the difficulty level of the task. After 20 and 30 trials, the same procedure checked the accuracy over the previous 20 trials and adjusted the task difficulty where necessary. At 40 trials the accuracy checking procedure was altered to be more 'delicate.' The difficulty was increased by one when accuracy over the previous 20 trials was greater than 79% and the difficulty decreased by one when the accuracy was less than 70%. When the accuracy was between 71% and 79% the difficulty was not adjusted. This procedure was repeated at 50 and 60 trials. The task difficulty then remained constant for the remaining 120 trials.

For 90 of the 180 trials the stimulus arrays contained more red than blue characters. The stimulus arrays in the remaining trials contained more blue characters than red characters. The order of the ‘more red’ and ‘more blue’ stimuli was quasi randomly arranged in blocks of eight trials with four trials of each type. It was not possible, therefore, for participants to encounter more than eight trials in a row of the same type (i.e., ‘more red’ or ‘more blue’).

Rewards.

Correct responses were occasionally rewarded according to a schedule in which correct identifications of one stimulus type were rewarded more frequently than correct identifications of the other stimulus type. The computer program arranged potential rewards to be delivered quasi-randomly, with close to four times as many rewards delivered for one type of correct response (e.g., ‘red’) than for the other correct response (e.g., ‘blue’). Both incorrect responses and correct responses that were not scheduled to be rewarded received no feedback.

Rewards contained multiple components. The message “*You win a token well done!*” appeared on the screen accompanied by a picture of a pile of coins and a randomly selected (without replacement) animated picture (graphics interchange format, GIF). A brief sound also played. Children were given verbal praise (e.g., “well done!”) by the experimenter, who also placed a token into a clear plastic cup to the right of the response panel.

The length of the reward sequence varied between 2500 ms and 3500 ms depending on the duration of the animated GIF for that instance of reward. Each animated GIF was displayed for long enough for one ‘cycle’ of the animation to complete (i.e., for each frame of the animation to be displayed). Where there was no clear cycle (i.e., it did not matter how long it was displayed for) the animation was played for 3000 ms.

Counterbalancing.

Two versions of the task were used: a version where correct ‘red’ responses were rewarded more frequently than correct ‘blue’ responses, and a version where correct ‘blue’ responses were rewarded more frequently than correct ‘red’ responses. Additionally, each participant completed two experimental tasks, the current study, and the task described in Chapter 6. So, for each of the two studies the order was noted, that is, whether it was the first or second study the participant had completed.

Each participant was quasi-randomly assigned to ‘version’ and ‘order,’ with near equal numbers of participants completing each version of the task, and near equal numbers of participants completing the task first and second. Due to subject attrition, however, the final number of participants in the four ‘counterbalanced conditions’ (Version 1-Order 1, Version 1-Order 2, Version 2-Order 1, and Version 2-Order 2) was not identical. The numbers in each condition were similar in the ND and ADHD-C groups.

Study 1: Results

Preliminary analyses.

For each participant, the session was analysed in three blocks of 60 trials. The measures derived were response time (RT), discriminability ($\log d$), and response bias ($\log b$). $\log d$ and $\log b$ were calculated in the manner of Tripp and Alsop (1999, left panel), and the equations for these measures were shown in Equations 1 and 2 in Chapter 3. The performance of each participant was scrutinised to determine if they did not follow instructions or attend to the task.

First, the RTs were examined to determine whether there were individual trials for each participant that had aberrant RTs, or whether there were some participants who had persistently aberrant RTs. For each participant, individual trials with RTs of less than 100 ms

and trials with RTs greater than 5000 ms were excluded from further analysis. Response times of less than 100 ms are likely to have been triggered by presentation of the stimulus before any meaningful processing of the stimulus could occur. RTs of greater than 5000 ms occur 3000 ms after the stimulus has disappeared, and would likely be determined partially by working memory processes in addition to the participant's ability to discriminate between stimuli. Some participants were completely excluded from the analysis because of persistently aberrant RTs. If greater than 10% of a participant's responses were 'too fast' (i.e., $RT < 100$ ms) or 'too slow' (i.e., $RT > 5000$ ms) the participant was excluded from further analysis.

The second exclusion criterion was discriminability ($\log d$). For each participant, $\log d$ was calculated within each block of 60 trials. Because a titration procedure occurred during the first 60 trials and task difficulty could alter during this time, the focus for exclusion was on the final two blocks of 60 trials. Participants who obtained $\log d$ scores of less than 0.10 in either of the final two blocks of 60 trials were excluded from further analysis. The rationale for this decision is that $\log d$ scores of 0.10 or below are close to chance (i.e., less than approximately 55% correct).

Dealing with potential confounds.

The three groups were not individually matched for Age or Estimated IQ Scores (ESTIQ). T-tests were used to examine group differences on these variables as indicated in Table 2. The ADHD-C group was younger, and had a lower mean ESTIQ score than the ND group. Because there were significant group differences in Age and ESTIQ, the next step determined if Age or ESTIQ were significantly correlated with any of the dependent variables. Pearson product moment correlations were calculated between Age and ESTIQ for both groups, and the three dependent variables over three blocks of trials (see Table 16, Appendix B). Age was negatively correlated with RT within each block for both groups,

Table 2.

Age and Estimated IQ (ESTIQ) for combined type ADHD (ADHD-C), predominantly inattentive ADHD (ADHD-PIA), and normally developing (ND) Groups. Ranges are in Parentheses.

	ADHD-C	ADHD-PIA	ND	Significant Differences
Age (Years)	8.0 (5.5 to 12.9) SD=1.8	9.4 (6.2 to 11.8) SD=1.6	9.0 (6.0 to 12.3) SD=1.7	ADHD-C<ADHD-PIA*, ND**
ESTIQ	95.9 (68 to 129) SD=14.7	90.5 (71 to 129) SD=15.6	105.2 (77 to 135) SD=13.3	ND>ADHD-C****, ADHD-PIA****
* p<.05	***p<.005			
p<.01	**p<.001			

although this was significant at the .05 level in the ADHD-C group only (in all three trial blocks). ESTIQ was positively correlated ($\alpha=0.05$) with RT in the third block of trials in the ADHD-C group only. Neither Age nor ESTIQ were significantly correlated with $\log b$ or $\log d$.

In addition to the variables discussed above, it is also possible that task order (i.e., whether the Study 1 task was the first or second task the participant completed) and task type (i.e., which response received more frequent rewards) were related to the dependent variables. Task order and task version are dichotomous variables, and have been included in the following analyses to determine if they influenced the dependent variables.

Main analyses.

Response bias ($\log b$).

$\log b$ was calculated for each participant within each block of 60 trials. Because of counterbalancing, approximately half of the participants received more frequent rewards for correct 'blue' responses than for correct 'red' responses (Version 1); this pattern was reversed for the remaining participants (Version 2). For the participants who completed Version 2, $\log b$ values were reversed in order to derive response bias towards the more frequently rewarded response alternative.

Figure 3 plots $\log b$ over each of the three blocks of 60 trials. One sample t-tests with an Alpha level of 0.05 (see Table 15, Appendix B) examined the $\log b$ values for each group over each block and task version to examine whether $\log b$ had developed, that is, whether $\log b$ was greater than zero. Both groups tended to develop bias over the course of the task. For Version 1 of the task $\log b$ was significantly greater than zero for both the ADHD-C and ND groups in each trial block with one exception: $\log b$ in the ND group in the first trial block approached, but did not reach significance ($t(34)=2.03$, $p<0.06$).

In Version 2 of the task the $\log b$ of both groups was close to zero in the first block of trials. $\log b$ in the ND group was significantly greater than zero over the second and third trial blocks. The ADHD-C group only developed bias significantly greater than zero in the third block of trials.

A 2X3X2X2 (Group X Block X Version X Order) ANOVA (see Table 8, Appendix B) compared the differences in $\log b$ between groups. There were no significant main effects of Group or Order. Significant main effects of Block ($F(2,102)=21.00$, $p<.001$) and Version ($F(1,103)=5.94$, $p<.05$) were found. The Block X Version interaction was significant ($F(1,102)=4.29$, $p<.05$). No other interactions were significant.

Because there was some evidence of between-group differences in the initial omnibus ANOVAs, linear contrast ANOVAs (see Table 9, Appendix B) were performed to examine between-group differences over repeated measurements (i.e., trial blocks) of $\log b$. Between-group differences in bias became more pronounced over successive task blocks, with the ND group developing higher $\log b$ scores than the ADHD Combined Type group. The BlockXGroup linear contrast was significant ($F_{\text{Linear}}(1,103)=4.94$, $p<.05$). The Block X Version linear contrast was also significant ($F_{\text{Linear}}(1,103)=7.38$, $p<.01$), with $\log b$ increasing more rapidly over successive trial blocks in Version 2 of the task compared with Version 1 of the task.

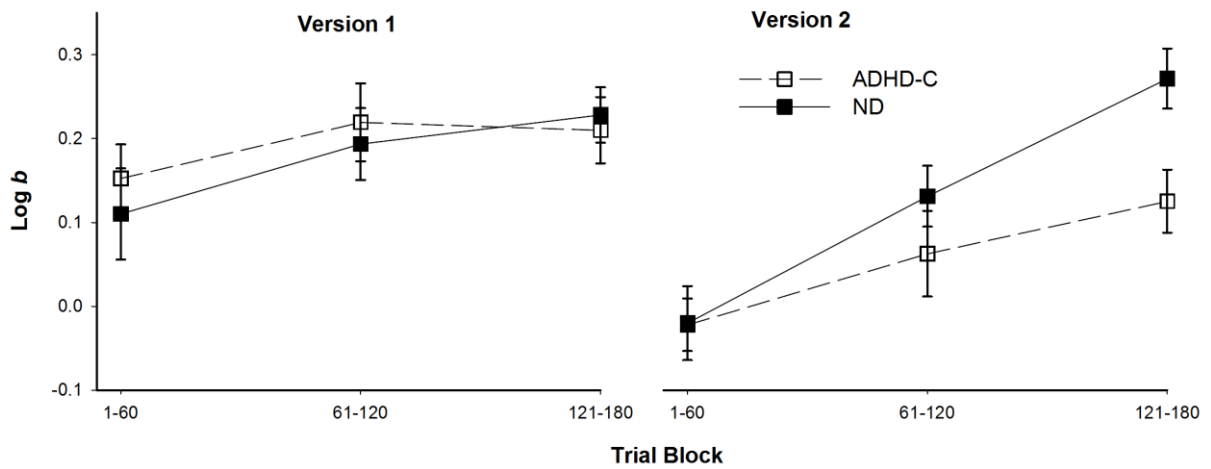


Figure 3. Mean bias ($\log b$) for combined type ADHD (ADHD-C) and normally developing (ND) groups over blocks of 60 trials by Version. For Version 1, more frequent rewards were provided for correct ‘more blue’ (right button) responses; the reverse was true for Version 2. Because there was a main effect of Version, $\log b$ is plotted separately for each version.

To examine the group differences in $\log b$ within each trial block, additional ANOVAs were performed (see Table 10, Appendix B). In the first block of trials, there was a significant main effect of version ($F(1,103)=8.41, p<.01$), but no other significant main or interaction effects. This effect is clear in Figure 3 which shows that $\log b$ is lowest in Version 2 of the task in the first block of trials.

In the second block of trials, there were significant main effects of version ($F(1,103)=5.32, p<.05$) and order ($F(1,103)=4.64, p<.05$),¹⁵ but no other significant main or interaction effects. The main effect of version is visible in Figure 3 which shows that $\log b$ is lowest in Version 2 of the task in the second block of trials.

¹⁵ The effect of order is not shown in Figure 3, which provides separate plots by Version. The table of means is included in Appendix B (Table 4) and shows that $\log b$ is generally higher for Order 1 participants.

In the third block of trials, there was a significant main effect of group ($F(1,103)=4.12$, $p<.05$) with the ND group showing greater bias towards frequent reward than the ADHD-C group (see Figure 3). There were no other significant main or interaction effects.

Response bias ($\log b$) over moving windows of 20 trials.

Because differences in $\log b$ between groups appeared to develop throughout the course of the task, an additional exploratory analysis was performed. $\log b$ was calculated over the first 20 trials for each participant. Then, $\log b$ was calculated from trials 2 to 21. This process was continued (with the final calculation being from trials 161 to 180) to provide a ‘moving window’ of $\log b$ over the course of the task. Figure 4 shows that $\log b$ in the ADHD-C group follows a similar pattern to the ND group in Version 1 of the task. In Version 2 of the task, however, the increase in bias over trials is clearly less consistent in the ADHD-C group than in the ND group.

Effect of recent reward history on response bias ($\log b$).

This analysis examined the effect of recent reward history on $\log b$. For each participant, trials that occurred following rewards for each of the two types of correct responses were analysed separately. This allowed trials to be divided into those occurring immediately following rewards for correct ‘red’ responses and those that occurred following rewards for correct ‘blue’ responses. Because this analysis only included trials that occurred following rewards, the effective number of trials for each participant was greatly reduced. In order to increase reliability, data from all blocks of trials was combined to calculate $\log b$.

When $\log b$ was calculated for responses that occurred when the most recent reward had been delivered for the *most* frequently rewarded response, this was designated as a ‘Rich’ reward. When $\log b$ was calculated for responses that occurred when the most recent reward

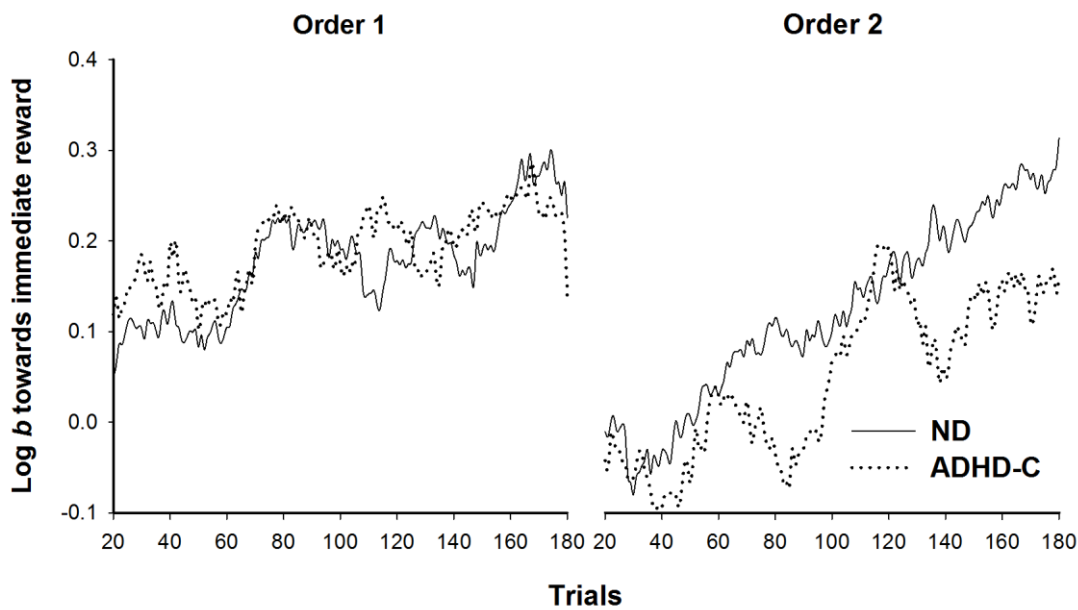


Figure 4. Mean bias ($\log b$) for the ADHD-C and ND Groups calculated within moving windows of 20 trials over the whole task.

had been delivered for the *least* frequently rewarded response, this was designated as ‘Lean’ reward. Due to counterbalancing, for approximately half of the participants, ‘blue’ responses were the more frequently rewarded response alternative, whereas for the remaining participants this pattern was reversed.

Figure 5 compares $\log b$ following ‘Rich’ rewards with $\log b$ following ‘Lean’ rewards for the ADHD-C and ND groups. The ADHD-C group showed a greater bias towards the more frequently rewarded response alternative on trials following ‘Rich’ rewards compared to trials following ‘Lean’ rewards. The ND group showed similar levels of bias towards the more frequently rewarded response regardless of which reward type was recently delivered. A 2X2X2X2 (Group X Reward Type X Version X Order) ANOVA (see Table 11,) examined these effects. There were no significant main or interaction effects.

Although there were no statistical differences between groups, Figure 5 shows that the overall value of $\log b$ appears relatively low in the ADHD-C group following lean rewards.

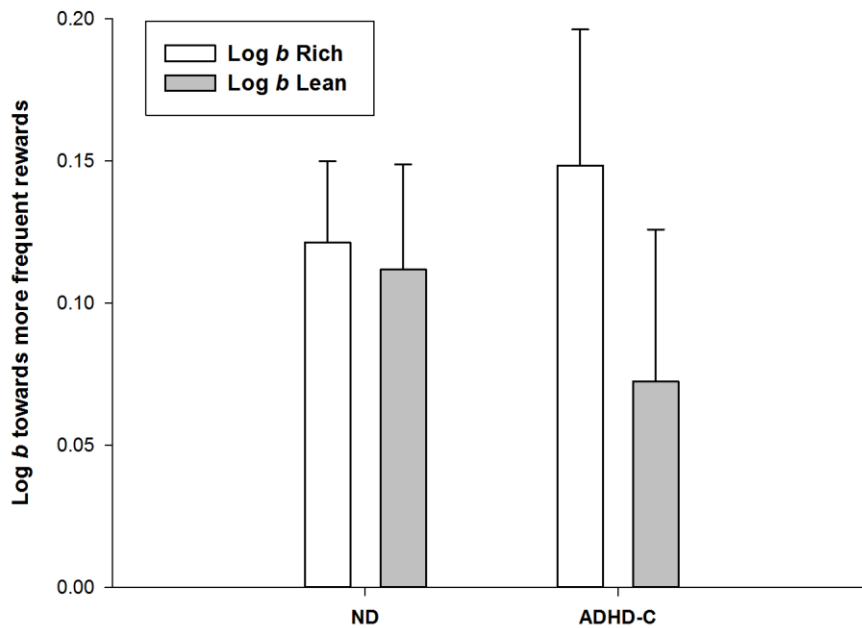


Figure 5. Bias ($\log b$) toward the more frequent reward calculated for trials following the 'Rich' reward and 'Lean' reward for combined type ADHD (ADHD-C) and ND groups.

When $\log b$ values were compared with zero by paired t-tests (see Table 16, in Appendix B) it was apparent that $\log b$ following both 'Rich' and lean rewards was greater than zero in the ND group. $\log b$ in the ADHD-C group was greater than zero in trials following 'Rich' rewards only.

Discriminability ($\log d$).

$\log d$ was calculated for each participant for each of the three blocks of 60 trials. During the first block of 60 trials, task difficulty was manipulated (i.e., a titration procedure) and only over the final two blocks of 60 trials did task difficulty remain constant for each participant. Consequently, $\log d$ over the final two blocks more accurately reflects task accuracy than $\log d$ calculated within the first block of the task.

Figure 6 plots $\log d$ over the final two blocks of the task. Clearly, both the ADHD-C and ND groups discriminated between stimuli. The range in mean $\log d$ scores (approximately .45 to .54) shown in Figure 6 corresponds to approximately 74% to 78% correct. In Trials 61-120, there were no differences between groups. This was not unexpected because the titration procedure was likely to have minimised between group differences in $\log d$. A 2X2X2X2 (Group X Block X Version X Order) ANOVA (see Table 12 in Appendix B) compared the differences in $\log d$ between groups. There were no significant main or interaction effects.

An additional (Group X Version X Order) ANOVA (see Table 13 in Appendix B) examined the differences between groups in each block of trials. In the second block of trials there were no significant main or interaction effects. In the third block of trials there was a significant main effect of Group, with the ADHD-C group showing lower discriminability than the ND group. There were no further significant main or interaction effects.

Response time (RT).

Median RTs were calculated for each participant within each block of 60 trials. Individual trials with RTs of 0 ms were excluded from the analysis, but trials with RTs between 1 and 100 ms were retained. Because this analysis examines response times, it would be counterproductive to exclude fast RTs, which may be more common in children with ADHD. Responses of 0 ms were excluded because they reflect responding that is unrelated to stimulus presentation. Responses of this type are not considered fast responses in the usual sense, but indicate that the participant was not engaging in the task as instructed. Once median RTs were calculated for each participant, mean RTs were calculated for each group over each successive block of trials. RTs in the first block of 60 trials have not been analysed because the first block of trials contained variation in task difficulty, which could reasonably be

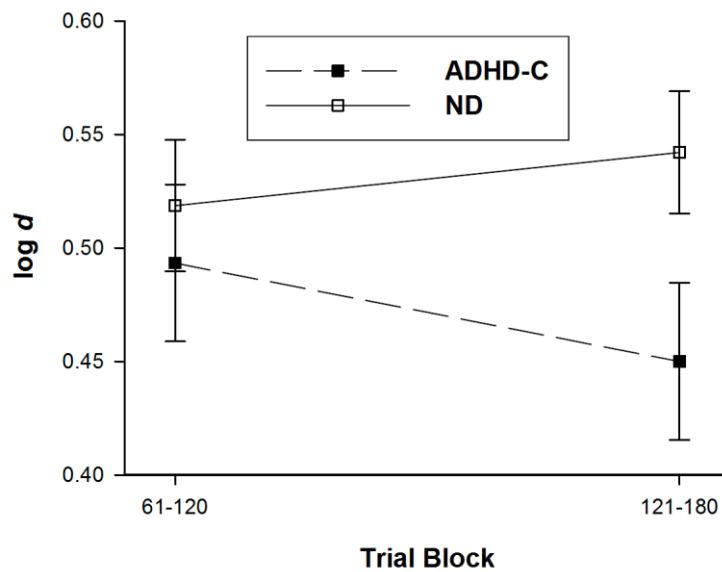


Figure 6. Discriminability ($\log d$) for combined type ADHD (ADHD-C) and normally developing (ND) groups over the final two blocks of 60 trials. Because the ANOVAs did not identify effects of version or order, both task versions and task orders are combined.

expected to influence RTs. Figure 7 shows that mean Response Times (RT) do not change from the second to the third block of trials in either group. A 2X2X2X2 (Group X Block X Version X Order) ANOVA (see Table 14 in Appendix B) examined the differences in RT between groups. No main or interaction effects were significant. Notably, the Group X Block interaction was not significant ($F(1,103)=2.50, p>.11$).

Speed-accuracy and discriminability vs. response bias.

The three signal-detection dependent variables (RT, $\log d$, and $\log b$) may be related. These relations are worth examining because a relation between the variables could conflate or obscure between group differences in another variable. For example, very high discriminability would likely lead to low bias scores, and very high bias scores would likely lead to low discriminability.

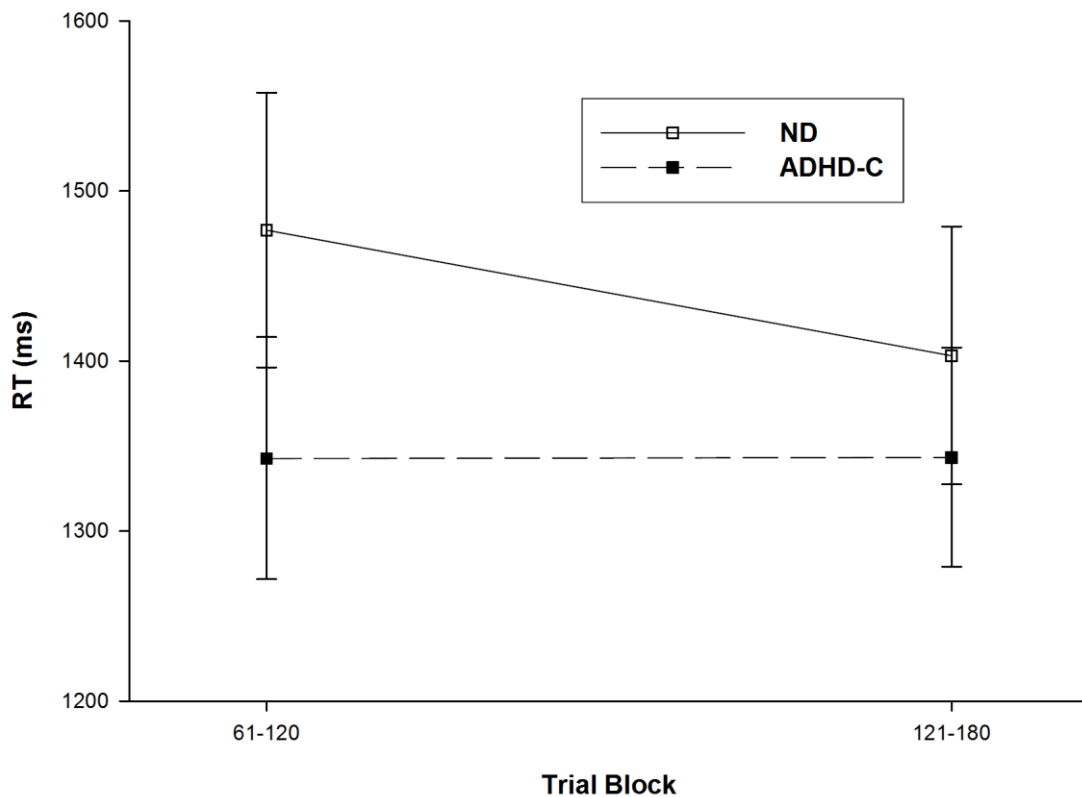


Figure 7. Mean response times in milliseconds for combined type ADHD (ADHD-C) and normally developing (ND) groups over the final two blocks of 60 trials.

The two main relationships of interest are between RT and $\log d$, and between $\log d$ and $\log b$. When responses are very rapid, participants have allowed less time to thoroughly process and respond to the stimuli. The relationship between RT and $\log d$ is known as *speed-accuracy trade-off*. If there is a significant positive correlation between RT and $\log d$ then it is likely that rapid responses have led to decreased accuracy of responding (i.e., $\log d$).

Correlations were performed within the ADHD-C and ND groups for each of the three blocks of trials (see Table 17.). In the first block of 60 trials there was a small positive correlation between $\log d$ and RT for the ND group ($r=.24$, $p<.05$) but not in the ADHD-C group. RT and

$\log d$ were not correlated significantly in the second or third blocks of trials. There was, therefore, no consistent evidence of speed accuracy trade-off.

There is some (although limited) evidence that response bias can be related to discriminability (Alsop & Davison, 1991; Alsop & Porritt, 2006). The relationship between $\log d$ and $\log b$ was examined within each group in all trial blocks. There were no significant correlations between $\log b$ and $\log d$ in either group within any of the three trial blocks (see Table 17). Overall, it appears that the between group differences in $\log b$ are unlikely to have been an artefact of any differences in discriminability.

Exploratory analysis of predominantly inattentive ADHD group.

The ADHD-PIA group contained 13 participants, which is significantly fewer than the ADHD-C (41) and ND (70) groups. Because effects of Order and Version were present, formal analyses would have required these variables to be considered in any ANOVA. This would have left approximately 3 participants in each cell, which would have provided insufficient power. Consequently, $\log b$ in the ADHD-PIA has been examined informally. Figure 8 shows that in Version 2 of the task (right hand panels), $\log b$ in the final block of trials is similar in the ADHD-PIA and ND groups, while $\log b$ in the ADHD-C group is somewhat lower than the ND group (cf. Figure 3).

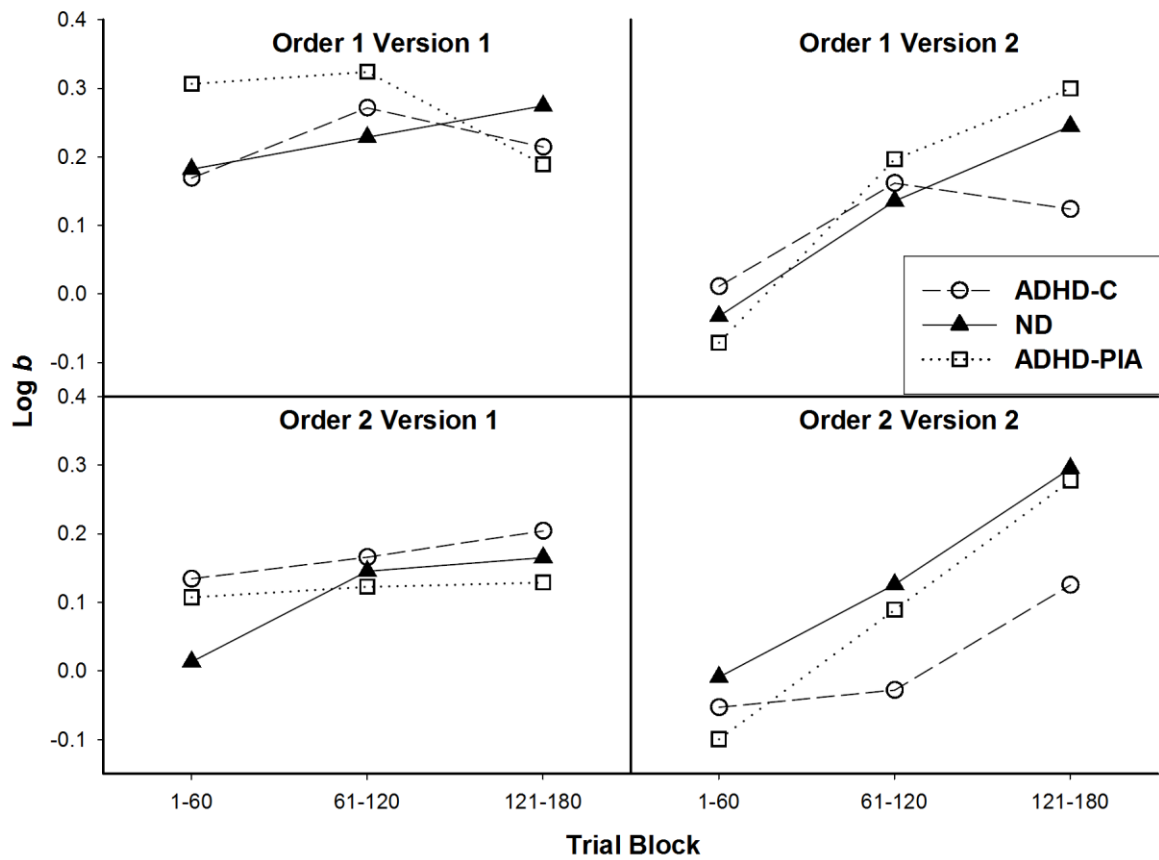


Figure 8. Log b is plotted for the combined type ADHD (ADHD-C), predominantly inattentive ADHD (ADHD-PIA), and normally developing (ND) groups. Because earlier analyses identified effects of Order and Version, these are plotted separately.

For the analysis comparing responses following frequent and infrequent rewards, Figure 9 shows that the response of the ADHD-PIA group was similar to the ADHD-C group for participants who completed Version 1 of the task (left panel) with log b following ‘lean’ rewards being lower than log b following ‘rich’ rewards. For participants who completed Version 2 of the task although the ADHD-PIA group is most similar to the ND group (right panel), with little to no difference between log b following ‘lean’ rewards and log b following ‘rich’ rewards.

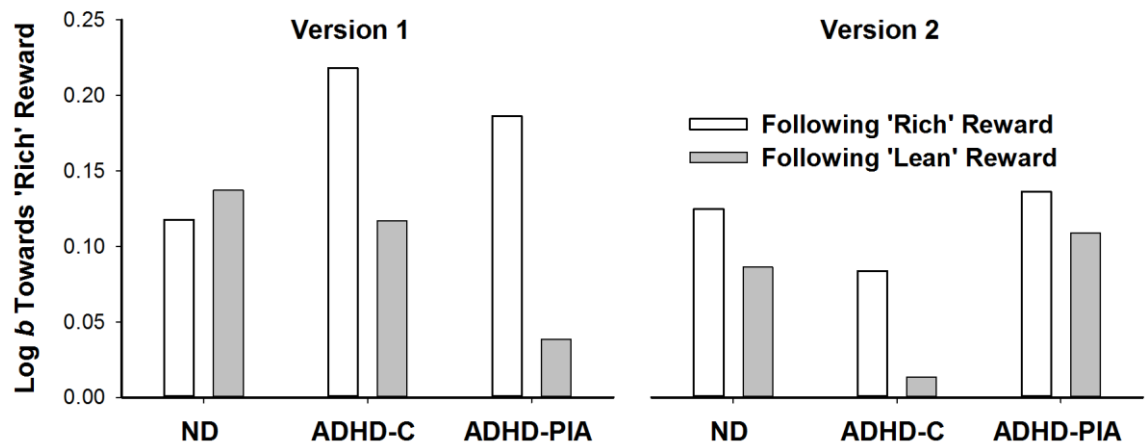


Figure 9. Log b towards frequent rewards is plotted for responses following frequent (rich) and infrequent (lean) rewards, for the combined type ADHD (ADHD-C), predominantly inattentive ADHD (ADHD-PIA), and normally developing (ND) groups. Because earlier analyses identified an effect of version, each version is plotted separately.

Chapter 5: Sensitivity to Reward Frequency Discussion

The present study examined the effects of unequal reward frequencies on the behaviour of children with and without ADHD in a signal-detection task. Rewards for correct responses of one alternative (e.g., 'red') were delivered more frequently than correct responses of the other alternative (e.g., 'blue'). Figures 3 and 4 show that children with combined type ADHD (ADHD-C) and normally developing children developed bias towards the more frequently reinforced response over the course of the task. Response bias in the normally developing group increased consistently over the course of the task. Children in the ADHD-C group, however, did show a slower increase in response bias; in the final block of trials, children with ADHD-C showed lower response bias towards frequent rewards than the normally developing children.

The local effects of reward were also examined by separating responses for each participant into those that occurred immediately following the more frequently reinforced ('rich') response and those occurring immediately following the less frequently reinforced ('lean') response. Although the differences between groups were not statistically significant, the ADHD-C group showed a trend towards lower response bias towards frequent rewards following instances of infrequent reward compared with bias following frequent rewards. The normally developing group showed similar levels of response bias regardless of which reward type had been most recently received.

Although response bias was the main variable of interest, the current study also examined discriminability and response times in the two groups. The current experiment found no significant between-group differences in response time (RT). Because of the titration procedure, the level of task difficulty varied between participants, but successfully ensured that all participants achieved similar levels of discriminability by the second block of 60

trials.¹⁶ From the second block of trials to the third block of trials, however, discriminability increased slightly in the normally developing group, but decreased slightly in the ADHD-C group. The normally developing group achieved greater discriminability than the ADHD-C group in the third block of trials. Given that performance on many tasks improves with practice, and that children with ADHD by definition have difficulty attending to tasks (e.g., Egeland, Johansen, & Ueland, 2009), this could reasonably be expected to impact their ability to learn the current experimental task. Given that the titration procedure led to similar discriminability scores in the intermediate stage of the task, subsequent differences between groups likely reflect differences in sustained attention rather than task performance as such..

To determine whether there were any variables that may have been confounded with the dependent variables, a number of additional analyses were performed. Response bias and discriminability were not correlated with one another. There was also no clear pattern that indicated that age or cognitive functioning had any notable relationship with the dependent variables. The counterbalancing procedure, however, complicated the interpretation of the data to some extent.

Two versions of the task were used. In Version 1, correct ‘blue’ responses were rewarded more often than were correct ‘red’ responses, whereas this pattern was reversed in Version 2. Overall, response bias was higher in Version 1 of the task than in Version 2, particularly during the first two blocks of trial. In the final block of trials, however, there were no differences in the response bias of participants who completed Version 1 of the task and those who completed Version 2 of the tasks. There are two possible explanations for the difference in response bias between task versions. First, the response bias differences between task versions may reflect differing perceptions of the two stimulus types (i.e., ‘more red’ and

¹⁶ Because task difficulty varied within the first block of 60 trials, and this would affect task accuracy, log d data from the first block of 60 trials were not examined.

‘more blue’ stimulus arrays). One coloured character may have appeared more salient than the other coloured character in the stimulus array, that is, one colour may have ‘popped out’ from the background more than the other colour. This would have led participants to develop a bias in favour of the more prominent stimulus. The second possibility is that the difference in response bias between task versions reflects a preference for a particular response (i.e., blue\right side button or red\left side button). If this were the case, $\log b$ would be highest in the task version where the preferred response was reinforced more frequently than the other response.

It is difficult to determine which of the above possibilities explain the current results. Stimulus effects on response bias in signal detection tasks do occur (e.g., Johnstone & Alsop, 1996), although there is limited literature on these effects. In Version 1 of the task, $\log b$ was calculated towards the ‘more blue’ response, whereas in Version 2 of the task, $\log b$ was calculated towards the ‘more red’ response. The ‘more blue’ response was to push the blue button on the right side of the response panel, whereas the ‘more red’ response was to push the red button on the left side of the response panel. It seems reasonable that participants may have showed an initial tendency to push the right button, which diminished over the course of the task as they were exposed to the reward contingencies.

Although there are some differences between the methodology and analysis in the present study and Tripp and Alsop (1999), their results can be compared. In terms of overall response bias, the results of the two studies are consistent; children with ADHD-C and normally developing children did not differ significantly in their overall response bias. However, the current study also examined response bias over successive blocks of trials throughout the entire task and found that bias in the ADHD-C group develops more slowly than the normally developing group.

When participants' responses following frequent (i.e., 'rich') rewards were compared with responses following less frequent (i.e., 'lean') rewards, children with ADHD-C and normally developing children showed slightly different patterns to one another, although these differences were numerical only. Children with ADHD-C showed lower response bias towards the more frequently rewarded response following 'lean' rewards, than they showed following 'rich' rewards. The normally developing group showed similar levels of bias irrespective of whether they had recently received a 'rich' or 'lean' reward. Although the overall difference between bias following 'rich' and 'lean' rewards were not significant in either group, the pattern was similar to Tripp and Alsop's study for both groups. It is also worth noting that the current study found that bias was significant and positive (i.e., non-zero) following 'rich' and 'lean' rewards in the normally developing group, but in the ADHD-C group bias was significant and positive only following 'rich' rewards. Although Tripp and Alsop did not explicitly state this, their graph (Figure 1) suggests that their results are consistent with the current study in this aspect.

When discriminability was examined, the current experiment found that the normally developing group had higher discriminability than the ADHD-C group in the third block of trials, but not in the second block of trials. The similarity in $\log d$ values in the intermediate stage of the task was expected, given that the task was designed to ensure that all participants achieved similar levels of task accuracy. The difference in the final block of trials is consistent with a slight deficit in sustained attention in the ADHD-C group. These results are similar to Tripp and Alsop's which found that overall discriminability in their control group was higher than the ADHD-C group, although not significantly so.

The current experiment showed no significant between-group differences in response time (RT). Tripp and Alsop also found that there were no significant differences in RT between groups. The current experiment also examined the relationship between

discriminability and RT, and found that there was minimal evidence of speed-accuracy trade-off. Neither group showed a tendency to make rapid but less accurate responses, so it is unlikely that the differences in response bias between groups in the final block of trials reflected differences in response time.

Although the current methodology is similar in many respects to Tripp and Alsop's (1999), there are some differences worth considering. Both studies compared the signal detection performance of similar-aged groups of boys with and without ADHD. Both arranged different frequencies of reward for two response alternatives to develop response bias. Despite these broad similarities, there are several differences, which may, in part, account for some of the differences in results between the studies. Some of the procedural differences may have influenced discriminability, others are more likely to have influenced response bias, and some procedural differences may have influenced both discriminability and response bias.

The two studies did not use exactly the same rewards, which may account for different findings in terms of response bias. In both studies, when a reward was delivered, participants were given a token, the experimenter praised the participant, and a message and visual display appeared on screen. At the end of the experiment the tokens could be exchanged for prizes. The two studies differed in the types of visual display when rewards were delivered; the current study paired a different animated cartoon with each reward delivered, whereas children in the Tripp and Alsop study were shown the same fireworks display with each reward. Overall, the rewards in the current experiment might be expected to be more reinforcing than those used by Tripp and Alsop. Furthermore, in the current study the more frequently rewarded response received four times as many rewards as the less frequently rewarded response, whereas Tripp and Alsop arranged a reward ratio of 3:1. The reinforcer ratio in the current study might be expected to produce higher overall bias than in Tripp and

Alsop's study, and perhaps to exaggerate any between-groups differences. In fact, the overall levels of response bias in the current study were similar, if not slightly *lower* than that reported by Tripp and Alsop. A likely explanation for the differences in rewards to produce higher response bias is the relatively low number of trials in the current study.

Participants in the current study completed 180 trials whereas Tripp and Alsop's (1999) participants completed 300 trials. The current results show that response bias developed over the course of the task. Although Tripp and Alsop (1999) did not examine this, there is no good reason to suppose that this did not also occur. Because the difference in bias between the ADHD and normally developing groups likely increases over time, the relatively short length of the current task (c.f., Tripp and Alsop) could have rendered any between-group differences in response bias more difficult to detect. Although differences were still noted in the final block of trials in the current experiment, the local effect of reinforcers (i.e., 'rich' vs. 'lean' response bias) was less clear than that reported by Tripp and Alsop. It seems reasonable to assume that the local effects of reinforcers might also develop over time. However, given that many trials are removed from the analysis when focussing only on trials that occur immediately following rewards, the number of trials in the current experiment was too low to effectively detect the development of the local effects of reinforcement over time.

When considering the two main differences in methodology between the current experiment and Tripp and Alsop (1999), it appears that the fewer number of trials in the current experiment may have led to a lower overall levels of response bias and reduced the between-group differences somewhat. This has occurred despite the rewards in the current experiment being potentially more salient, and the reward ratio being higher compared with Tripp and Alsop. It seems likely that if participants had completed more trials, difference in response bias following 'rich' versus 'lean' rewards would have been clearer.

Four main differences between the current experiment and Tripp and Alsop (1999) might be expected to influence discriminability. First, the titration procedure that was used in the current experiment (but not by Tripp and Alsop) was intended to produce similar levels of discriminability in all participants, regardless of ability level. This accounts for the results of the current experiment which found that differences in discriminability between groups only emerged towards the end of the task. Tripp and Alsop found higher discriminability in the control group than the ADHD group. Second, the current experiment was shorter than Tripp and Alsop's (180 trials vs. 300 trials). The current experiment found some evidence that discriminability changed throughout the course of the task, with the normally developing group becoming more accurate, and the ADHD-C group becoming less accurate over time. Shorter tasks could be expected to minimise differences in discriminability between ADHD and control groups. Third, the current study used an array of red and blue cartoon characters, and participants responded that there were 'more red' or 'more blue' characters present. Tripp and Alsop used a face stimulus where participants responded to indicate whether the face had a large or a small mouth. Fourth, stimuli were displayed for a longer duration in the current study than in the Tripp and Alsop study (2000ms vs. 100ms respectively). The discrimination task in the current study is somewhat more complex than that used by Tripp and Alsop (1999), although this is 'balanced' by the longer stimulus duration. Overall, it appears that participants in the Tripp and Alsop study may have found the task more difficult than participants in the current study, as their discriminability values were somewhat lower than in the current experiment.¹⁷

¹⁷ Tripp and Alsop (1999) reported log d values of 0.34 and 0.46 for the ADHD and control groups respectively. Log d values in the current study ranged from approximately .48 to .45 from the second to third blocks of trials for the ADHD group and from .52 to .54 for the control group.

Finally, there were differences in inclusion criteria between the current experiment and Tripp and Alsop that are worth a brief mention. The current study included more participants than Tripp and Alsop (41 ADHD-C and 70 controls vs. 15 ADHD and 15 controls respectively). It is also possible that the children with ADHD in the current study are more ‘typical’ than the children with ADHD included in the Tripp and Alsop study. Tripp and Alsop’s groups had similar age and estimated IQ; whereas the two groups in the current study were different (the ADHD-C group was younger and had lower estimated IQs than the normally developing group). Children in the ADHD-C group in the current study had lower Full Scale IQ scores ($M=96$, $SD=15$) than the ADHD group in the Tripp and Alsop study ($M=108$, $SD=18$). Because children with ADHD tend to have lower IQs than control children (Barkley, 2006a; Frazier et al., 2004; Hervey et al., 2004; Wechsler, 2003a), it is possible that the ADHD-C group in the current study are more ‘typical’ in terms of intellectual functioning than the ADHD group in the Tripp and Alsop study. It is also possible that the children in the current study were a more homogeneous group than the Tripp and Alsop participants. The current study used DSM-IV-TR (American Psychiatric Association, 2000) criteria, whereas Tripp and Alsop identified their ADHD group using DSM-III-R (American Psychiatric Association, 1987) criteria. The DSM-IV-TR criteria for ADHD (Combined Type) require that children exhibit significant six out of nine symptoms of inattention *and* six out of nine symptoms of hyperactivity\impulsivity. The DSM-III-R criteria require that a total of eight out of fourteen symptoms be present. These symptoms include both inattentive and hyperactive\impulsive symptoms. It is possible, therefore, that there was more variability in the symptom profiles of the ADHD group in Tripp and Alsop’s study compared with the current study.

The main analyses in the current study examined a group of boys with ADHD-C and a group of normally developing boys. A smaller group of boys with inattentive ADHD (ADHD-

PIA) was included as an exploratory analysis towards the end of the previous chapter. Although the number of participants in the ADHD-PIA group was too small to conduct sufficiently powerful statistical analyses, an informal analysis reveals some suggestive patterns. When the response bias towards the frequently rewarded stimulus/response was examined over trial blocks, the pattern in the ADHD-PIA group was more similar to the normally developing group than the ADHD-C group. Both the ADHD-PIA and normally developing groups continued to develop response bias throughout the task, whereas the response bias in the ADHD-C group tended to be lower towards the end of the task. This pattern was clearest in participants who completed Version 2 of the task where left/red responses received more frequent rewards than right/blue responses (Figure 8: right hand panels).

When the response bias following frequent ('rich') and infrequent ('lean') rewards was examined, the ADHD-PIA group appeared similar to the ND group for participants who completed Version 2 of the task, but was similar to the ADHD-C group for participants who completed Version 1 of the task. This lack of clarity of the results is likely a result of the small sample size of ADHD-PIA participants.

The current experiment examined the effects of different frequencies of reward on response bias in normally developing children and children with ADHD, and is most readily compared with Tripp and Alsop (1999). There is a wide range of literature, however, that examines the responses to rewards of children with ADHD using other methods that are not directly related to the current experiment. The results of the current study are generally consistent with previous studies showing that children with ADHD are less sensitive to changes in reward frequency in both concurrent schedule procedures (e.g., Kollins et al., 1997) and in simple choice procedures (e.g., Schweitzer & Sulzer-Azaroff, 1995). Some additional literature is also relevant to reward delay, which is examined in the following

experiment. Consequently, the discussion of the broader reward literature will be left until the general discussion.

Chapter 6: Sensitivity to Reward Delay Method and Results

Method

Participants.

From the initial pool of participants, data from three groups of male participants were analysed: Forty four boys with ADHD, combined type (ADHD-C), 15 ADHD, predominantly inattentive type (ADHD-PIA), and 80 normally developing boys (ND). Some participants (3 ADHD-C, 3 ADHD-PIA and 4 ND) were excluded due to concerns about their performance on the signal detection task (discussed below). Female participants were not included in the analyses because there were too few with ADHD to conduct sufficiently powerful analyses. Age and estimated IQ (ESTIQ) were not matched between groups. Because of the relatively small number of participants in this group, children with ADHD-PIA were excluded from the main analyses. This group is examined less formally towards the end of the current chapter.

Apparatus.

The apparatus was as described in Chapter 3.

Procedure.

Participants sat at a desk in front of the screen with their heads approximately level with the top of the screen and approximately 40 cm from the screen. The response panel was on the desk between the participant and the screen. The experimenter sat to the right of the child, facing the same screen as the child.

The task began with instructions and two practice trials. All text on screen was read aloud by the experimenter, and clarification was given where necessary. If the participants made incorrect responses on either of the practice trials, the experimenter corrected the participants verbally. The instructions began with the text *“Hi, This is a simple computer*

game. You will see some patterns of blue greeblies and red greeblies. You must decide if there are more blue ones or red ones, and press the blue or red button. Here's an example"

appearing on the screen in coloured text approximately 5 mm high. Once the instructions had been read aloud the first practice trial began. The trial was signalled by a small juggler character appearing in the centre of the screen for 750 ms. After a delay of 250 ms, a stimulus array containing 60 blue and 40 red characters then appeared for 2000 ms. After the stimulus array disappeared, the text "*Press the more blue button*" appeared and was read aloud by the experimenter. When the participant pressed the blue button, the experimenter activated the next screen using the mouse. The text "Great, now try another one!" appeared and the experimenter activated the second trial.

The second practice trial was identical to the first, except the ratio of colours on the stimulus array were reversed (i.e., there were 60 red and 40 blue characters). After the stimulus presentation, the text "*Press the more red button*" appeared and was read aloud by the experimenter. Once the participant pressed the red button the experimenter activated the following screen which read: "*Sometimes, but not always, you can win a token for being right. You never get a token for being wrong. Win lots of tokens and you get a prize. When you get a token it looks like this -*". An animated cartoon then appeared, a brief musical tone played, and the experimenter placed a token in the clear plastic container. The experimenter then activated the following screen, which read "*Are you ready to start? Remember that you don't always get a token when you are right – Just sometimes.*" When the participant indicated that he was ready, the experimenter activated the 120 experimental trials.

Each trial began with an animated juggler character appearing in the centre of the screen for 750 ms, followed by a blank white screen for 250 ms. This warning stimulus was followed by presentation of the stimulus array which remained for 2000 ms unless terminated by a response. After the stimulus presentation, the screen remained white with no characters

present until a response was made. If the response was correct and scheduled to be rewarded, then the reward was delivered.

Over the first 60 trials, a titration procedure adjusted the difficulty of the task by altering the proportion of blue to red characters in the stimulus array. This procedure attempted to ensure that each participant would achieve similar levels of accuracy (between approximately 70% and 80% correct) on the task. This titration procedure was identical to that used in Study 1 and has been explained in detail in Chapter 4.

The sequence of stimuli for the 120 trials was quasi-randomly arranged; each successive block of eight trials contained four trials with a ‘more blue’ array and four with a ‘more red’ array ordered randomly. Therefore, participants could encounter no more than eight ‘more red’ or ‘more blue’ trials in a row.

Delay.

This study examined the effect of reward delay/immediacy on response allocation. A crucial aspect of the experimental design was the location of delays within each trial. In this study each correct response was scheduled to receive an immediate reward, a delayed reward, or no reward. Incorrect responses and correct responses that were not scheduled to be rewarded both had a delay of 3500 ms inserted before the next trial. Immediate rewards had a post-reward delay of 3500 ms, whereas delayed rewards were delivered after a delay of 3500 ms (with no post-reward delay). This design ensured that the overall trial length was equal for rewarded responses (both immediate and delayed) and unrewarded responses.

Rewards.

Rewards were the same as described in Chapter 4. A text message appeared on the screen, along with an animated picture, and children were given verbal praise by the

experimenter, who also placed a token into a clear plastic cup. Equal numbers of rewards were delivered for correct responses to each stimulus type in a quasi-random order.

The length of each reward varied between 2500 ms and 3500 ms (not including the pre-or post-reward delay) depending on the duration of the animated GIF for that instance of reward. Each animated GIF was displayed for long enough for one ‘cycle’ of the animation to complete. Where there was no clear cycle (i.e., it did not matter how long it was displayed for) the animation was played for 3000 ms.

Counterbalancing.

Two versions of the task were used. In Version 1, correct ‘red’ responses were rewarded immediately and correct ‘blue’ responses were rewarded after a delay. In Version 2, the correct responses associated with immediate and delayed rewards were reversed.

Each participant completed both Study 1 and Study 2. The order was recorded for each participant, that is, whether the current study was completed first, or whether the participant had completed Study 1 first.

As each participant was recruited, they were quasi-randomly assigned to ‘task version’ and ‘task order,’ with near equal numbers of participants completing each version of the task, and near equal numbers of participants completing the task first and second. Due to subject attrition (e.g., diagnostic exclusions, task performance exclusions) the final number of participants in the four ‘counterbalanced conditions’ (Version 1-Order 1, Version 1-Order 2, Version 2-Order 1, and Version 2-Order 2) was not identical, although the numbers in each condition were similar in the ND and ADHD-C groups. Table 18 (in Appendix C) shows the final number of participants in each Order and Version condition.

Study 2: Results

Preliminary analyses.

For each participant, the session was analysed in two blocks of 60 trials. The measures used were response time (RT), discriminability ($\log d$), and response bias ($\log b$), calculated within each block of 60 trials. $\log d$ and $\log b$ were calculated as described in Chapter 3 (Equations 1 and 2 respectively).

The performance of each participant was scrutinised to determine if there were aspects of their performance that indicated that they may not have followed instructions or attended adequately to the task. For each participant, individual trials were excluded if they had RTs of less than 100 ms or greater than 5000 ms. Participants' data were completely excluded from the analysis if their greater than 10% of their RTs were fast (less than 100 ms) or slow (more than 5000 ms). The second exclusion criterion was discriminability ($\log d$). For each participant, $\log d$ was calculated within each block of 60 trials. Because the first 60 trials used the titration procedure, the focus for exclusion was on the final block of 60 trials. Participants who obtained $\log d$ scores of less than 0.10 in the final block of 60 trials were excluded from further analysis.

Dealing with potential confounds.

The three groups were not individually matched for Age or Estimated IQ Scores (ESTIQ) as matching on these variables would have significantly reduced the number of ADHD-C participants. T-tests were used to examine group differences on these variables as indicated in Table 3. The ADHD-C group was younger than the ND and ADHD-PIA groups. The ND group had higher ESTIQ scores than the two ADHD groups.

Table 3.

Mean age in years and estimated IQ (ESTIQ) for combined type ADHD (ADHD-C), inattentive ADHD (ADHD-PIA), and normally developing (ND) groups. Ranges are in parentheses.

	ADHD-C	ADHD-PIA	ND	Significant Differences
Age	8.2 (5.6 to 12.9) SD=2	9.4 (6.2 to 11.8) SD=1.5	9.0 (6 to 12.3) SD=1.7	ADHD-C<ND*,ADHD-PIA*
ESTIQ	94 (68 to 129) SD=15	91 (65 to 129) SD=17	105 (77 to 135) SD=13	ND>ADHD-C*****,ADHD-PIA**
= $p < .05$ *= $p < .005$ *****= $p < .0005$ **= $p < .01$ *****= $p < .001$				

Because there were significant group differences in Age and ESTIQ, the next step determined if Age or ESTIQ were significantly correlated with any of the dependent variables. Pearson product moment correlations were calculated between Age and ESTIQ for both of the groups included in the main analyses (i.e., ND and ADHD-C), and the three dependent variables over two blocks of trials (see Table 33, Appendix C). Older participants tended to have shorter RTs than younger participants, although this correlation was only significant for the ADHD-C group ($r = -0.39$, $p < .01$) in the second block of 60 trials. Neither Age nor ESTIQ were correlated significantly with any other outcome variables in either group in the trials blocks analysed.¹⁸

In addition to the variables discussed above, it is also possible that task order (i.e., whether the current experimental task was the first or second task completed) and version (i.e., which response received immediate rewards) were related to the dependent variables. Task order and version are dichotomous variables, and have been included as variables in the following analyses.

¹⁸ There was a significant correlation between $\log d$ and Age in the first block of trials for the ADHD-C group, as shown in Table 35, Appendix C. However, $\log d$ in the first block of trials has not been analysed for reasons discussed below.

Main analyses.

Response bias (log b).

Log b was calculated for each participant within each block of 60 trials. Because of the counterbalancing, approximately half of the participants received Version 1 (i.e., immediate rewards for correct ‘blue’ responses and delayed rewards for correct ‘red’ responses), and the remaining participants received Version 2 of the task. For the participants in Version 2, log b values were reversed in order to derive log b measures in terms of the immediately rewarded response alternative. Because the distribution of rewards across response alternatives remained constant throughout the task, it was unlikely that log b , primarily a function of reward contingencies, would be affected by the titration procedure.

Figure 10 plots log b over each of the two blocks of 60 trials. Where participants completed the current study before the Study 1 task (i.e., Order 1, left panel) both groups showed bias towards the immediate reward in both blocks of trials, although this appeared more pronounced in the ND group than in the ADHD-C group. Because participants were expected to develop bias towards the immediate reward, a series of t-tests were performed to determine whether the log b values were significantly different from zero (see Table 29, Appendix C).

When participants completed the current study after the Study 1 task (i.e., Order 2, right panel) both groups showed a negative bias towards the immediate reward. For ND participants who received Order 1, log b was significantly greater than zero in both blocks of trials. For participants in the ADHD-C group who received Order 1, log b values were close to zero in both blocks of trials.

For ND participants who received Order 2, log b values were close to zero in each block of trials. For participants in the ADHD-C group who received Order 2, log b values

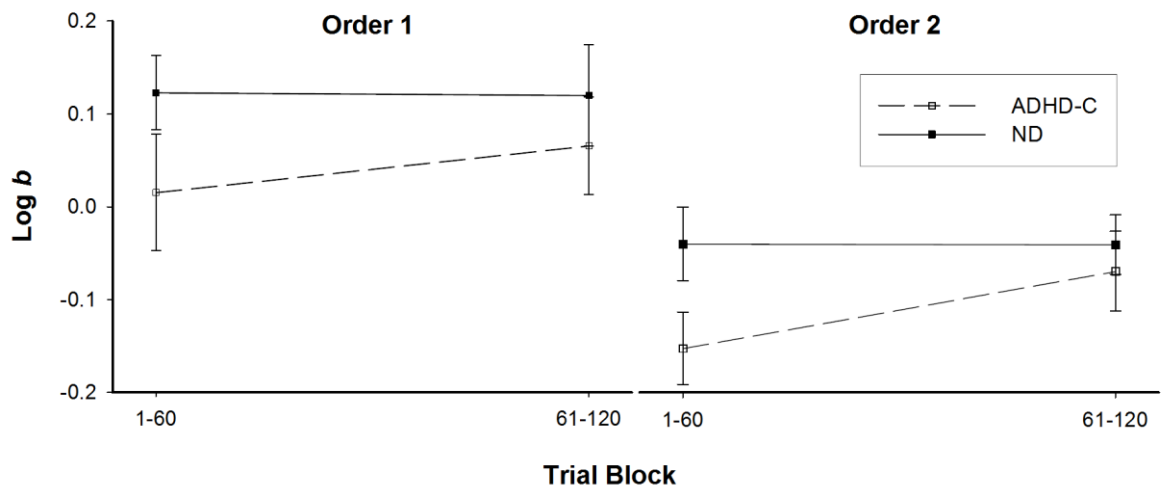


Figure 10. Mean Bias ($\log b$) for ADHD-C and normally developing (ND) groups over blocks of 60 trials. Because there was a main effect of Order, $\log b$ values are plotted separately for each Order. The left panel includes participants who completed the current task before the Study 1 task (Order 1). The right panel includes participants who completed the Study 1 task before the current task (Order 2).

were significantly *less* than zero in the first block of trials, and close to zero in the second block of trials. The finding that both groups showed negative bias towards the immediate reward (i.e., bias *towards* the delayed reward) when they completed the current experimental task in Order 1 is interesting. It was assumed that participants would develop bias towards the immediate reward, and this did not occur in the Order 2 participants. The likely reason for this involves the effect of the previously completed task, and is discussed in Chapter 7.

A 2 X 2 X 2 X 2 ANOVA compared differences in $\log b$ between groups (see Table 22, Appendix C). Group, Order, and Version were entered as between-subjects variables, and Block was entered as a within-subjects variable. There was a significant main effect of Order ($F(1,116)=13.69$, $p<.001$). No remaining main or interaction effects were significant.

Because there was a significant main effect of Order, additional 2 X 2 X 2 ANOVA were completed separately for Order 1 and Order 2 participants (see Table 23, Appendix C). Group and Version were entered as between-subjects variables, and Block was entered as a within-subjects variable. For Order 1 participants, there were no significant main or interaction effects.

For Order 2 participants, there was a significant Block X Version effect ($F(1,63)=9.40$, $p<.005$). There were no significant main effects. To determine what effects were significant within each block, for Order 2 participants only, 2 X 2 ANOVAs were completed separately within each trial block, with Group and Version entered as between subjects variables (see Table 24, Appendix C). In both blocks of trials, there were no significant main or interaction effects.

In summary, participants who completed Study 2 first (i.e., Order 1) generally showed higher bias towards immediate reward than participants who completed Study 2 second (i.e., Order 2). When $\log b$ was examined separately for Order 1 and Order 2 participants, there were no significant effects for the Order 1 participants although in the Order 2 participants, there was a trend towards the ADHD-C group obtaining lower $\log b$ scores in the first block of trials.

Response bias ($\log b$) over moving windows of 20 trials.

Because there was some, albeit limited, evidence that $\log b$ increased over the course of the task, an additional analysis was performed. $\log b$ was calculated over the first 20 trials, then over Trials 2 to 21, Trials 3 to 22 and so on, to provide a moving window of 20 trials. Figure 11 shows that $\log b$ in the ADHD-C group is lower than $\log b$ in the ND group early in the task, although there is little difference between groups towards the end of the task.

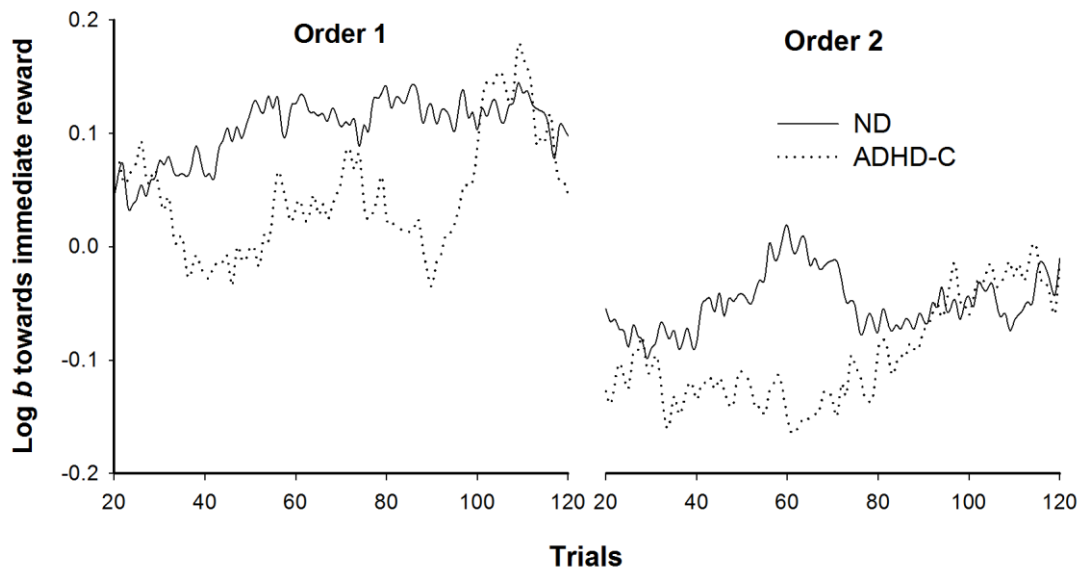


Figure 11. Mean response bias ($\log b$) for combined type ADHD (ADHD-C) and normally developing (ND) groups calculated within moving windows of 20 trials over the whole task. Participants that received Order 1 are plotted separately from those that received Order 2.

The previous analyses of response bias examined $\log b$ over each successive block of trials for the ADHD-C and ND groups. The next analysis examined the effect of recent reward history on $\log b$ over all 120 trials of the task. For each participant, trials were divided into those that occurred in the next trial after an immediate reward and those that occurred following delayed rewards. $\log b$ was then calculated separately for each of these situations.

Figure 12 compares $\log b$ towards immediate reward following immediate and delayed rewards for the ADHD-C and ND groups. Both groups show a greater bias towards the immediately rewarded response alternative on trials following delayed rewards compared to trials following immediate rewards. A $2 \times 2 \times 2 \times 2$ ANOVA examined these effects (see Table 25, Appendix C). Group, Order, and Version were entered as between-subjects variables, and Reward Type was entered as a within-subjects variable.

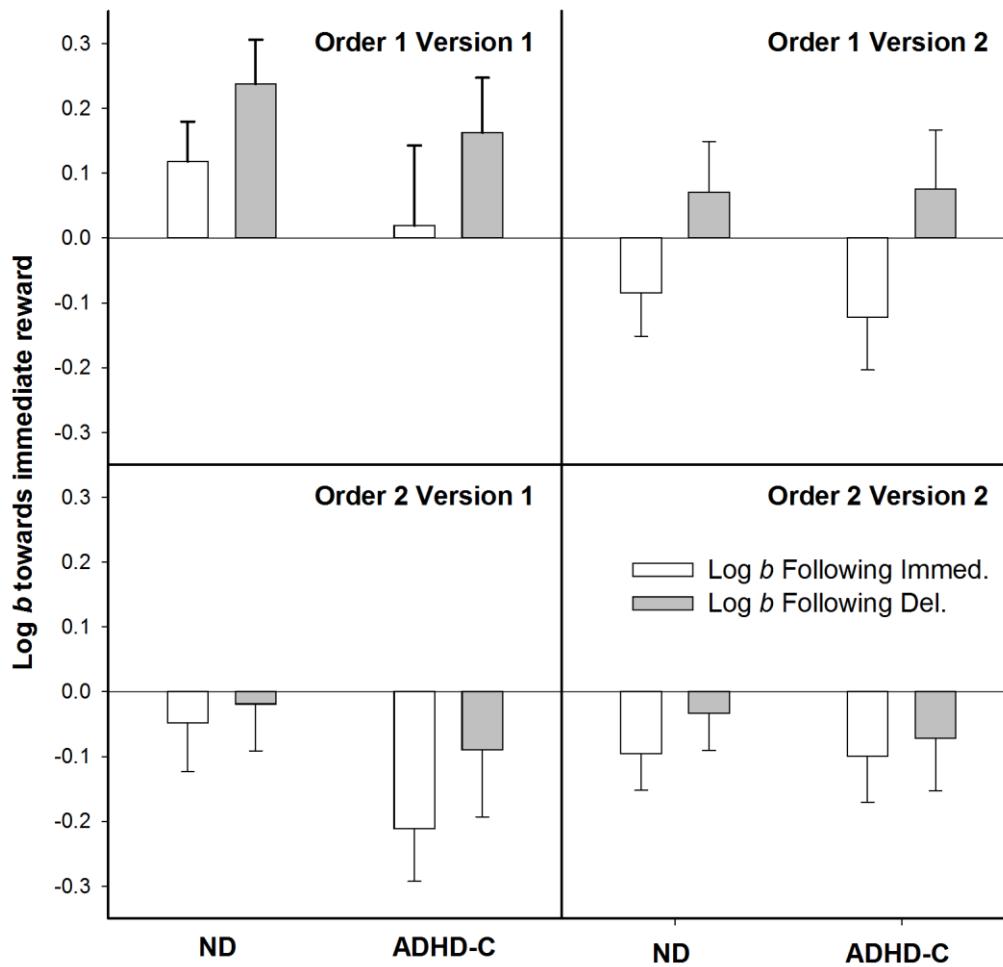


Figure 12. Response bias (log b) towards immediate reward calculated for trials following immediate reward and delayed reward for combined type ADHD (ADHD-C) and normally developing (ND) groups. Because ANOVAs revealed effects of Order and Version each Order-Version combination is plotted separately.

There were significant main effects of Order ($F(1,116)=10.11, p<.005$) and Reward Type ($F(1,116)=9.83, p<.005$), which can be seen in Figure 11. Of note, none of the main or interaction effects involving Group were significant. Additional ANOVAs were performed within each order (see Table 26, Appendix C). For participants who received Order 1, there was a significant main effect of Reward Type ($F(1,53)=9.42, p<.005$), with log b being higher

following delayed rewards. There was also a significant main effect of Version ($F(1,53)=5.20$, $p<.05$), and no interaction effects. For participants who received Order 2 there were no significant main or interaction effects.

For Order 1 participants, those who completed Version 1 generally obtained higher log b scores than participants who completed Version 2. Additional ANOVAs examined the effects of Group and Reward Type separately for each Version. For participants who completed Version 2, there was a significant main effect of Reward Type ($F(1,24)=5.82$, $p<.05$) with both groups obtaining higher log b scores following delayed reward than following immediate reward.¹⁹ There were no further significant main or interaction effects.

Figure 12 shows that many of the values of log b are close to zero. A number of one-sample t-tests (see Table 28, Appendix C) were performed to determine whether log b was significantly different from zero. The ND group who completed Version 1 of the task in Order 1 (upper left panel of Figure 12) showed significant (i.e., non-zero) bias towards immediate reward following instances of delayed reward ($t(20)=-3.48$, $p<.005$). The ADHD-C group who completed Version 1 of the task in Order 2 (lower left panel of Figure 12) showed significant bias towards *delayed* reward²⁰ following instances of immediate reward ($t(10)=-2.58$, $p<.05$).

In summary, some participants developed bias towards immediate reward, but this depended more on recent reward history and task variables (Version or Order), rather than diagnostic group. Participants who had not completed a previous signal detection task (i.e., Order 1) showed higher bias towards immediate reward following instances of delayed

¹⁹ Another ANOVA (see Table 27, Appendix C) examined the effect of Reward Type within each group for the Order 1 Version 2 participants. The results were not significant, which is unsurprising given the low numbers in each cell (11 ADHD-C and 15 ND).

²⁰ Negative bias towards immediate reward represents bias towards delayed reward.

reward. This pattern was similar for both the ADHD-C and ND groups.

Discriminability ($\log d$).

$\log d$ was calculated for each participant the final block of 60 trials. $\log d$ within the first block of trials was not examined because the first 60 trials contained a titration procedure where the task difficulty was altered for each participant. For each participant, fast (i.e., RTs of less than 100 ms) and slow (i.e., RTs of more than 5000 ms) trials were excluded from analysis. Task difficulty was manipulated during the first 60 trials (i.e., the titration procedure), so $\log d$ over the final block more accurately reflects performance than $\log d$ calculated within the first block of the task. Consequently, only $\log d$ in the final block of trials is reported below.

Figure 13 plots $\log d$ over the final block of the task. Performance in each of the groups is plotted separately for Order 1 and Order 2. Clearly, both the ADHD-C and ND groups discriminated between stimuli. The range in mean $\log d$ scores (approximately .50 to .65) shown in Figure 13 corresponds to approximately 76% to 82% correct. A 2 X 2 X 2 ANOVA was performed with Group, Version, and Order all included as between-subjects variables (see Table 30, Appendix C). There were no significant main effects, although the Group X Order interaction was significant ($F(1,116)=5.58, p<.05$). There were no other significant interaction effects.

The significant Group X Order interaction was examined in two ways. First, order effects were examined separately in both the ADHD-C and ND groups. A 2 X 2 ANOVA was performed with Version and Order included as between-subjects variables (see Table 31, Appendix C). Children in the ADHD-C group, showed higher discriminability ($\log d$) scores when the task was novel (i.e., they completed the current experimental task before the Study 1 task, than when the task was familiar (i.e., they had completed the Study 1 task first), although

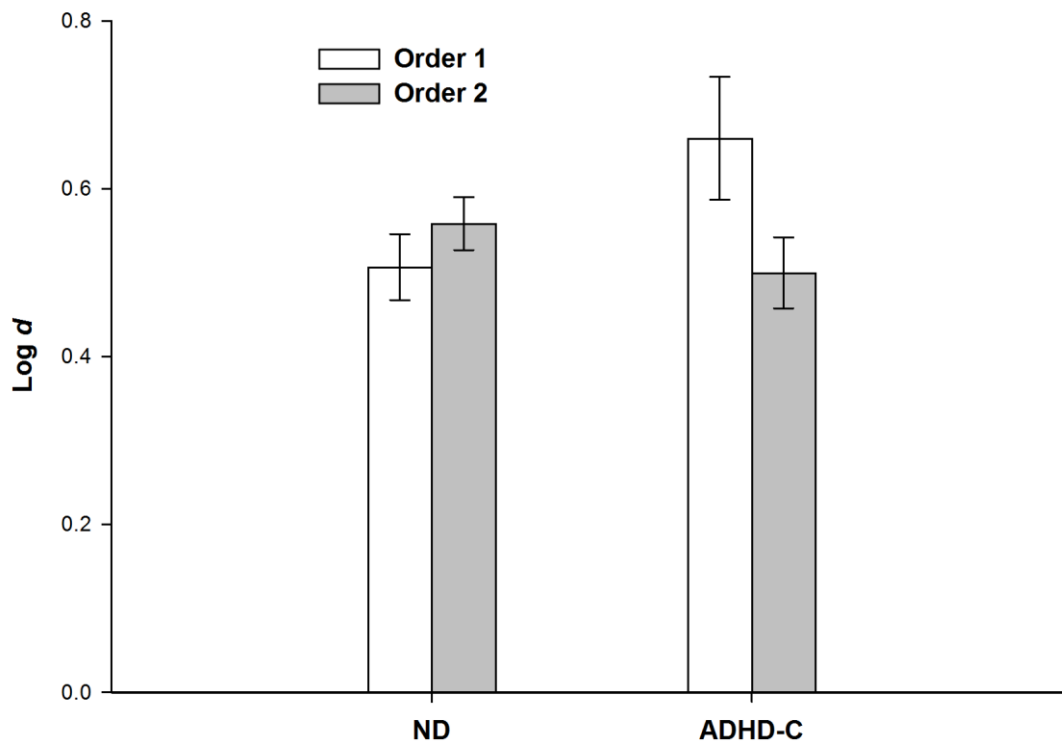


Figure 13. Discriminability ($\log d$) for combined type ADHD (ADHD-C) and normally developing (ND) groups over final block of 60 trials, by Order.

this difference was not significant. Children in the ND group showed a similar level of accuracy, regardless of the order in which they had completed the task.

The second way of describing the interaction effects is to examine the effect of group separately for each order (i.e., whether children completed the task first or second). A 2 X 2 ANOVA was performed with Group and Version included as between-subjects variables (see Table 32, Appendix C). For children who completed the current experimental task first the ADHD-C group was more accurate than the ND group, ($F(1,53)=4.26, p<.05$). For children who completed the current experimental task second (i.e., the signal-detection task was familiar to them) both the ND and ADHD-C groups showed similar levels of accuracy.

Response time (RT).

Median RTs were calculated for each participant within the final block of 60 trials. Individual trials with RTs of 0 ms were excluded from the analysis because these responses were clearly triggered by something other than stimulus onset (e.g., playing with the keys). Trials with RTs less than 100 ms but greater than 0 ms were included in these analyses (cf. analyses of $\log b$ and $\log d$) because to exclude them may obscure any differences in RTs between groups. Mean RTs were then calculated for each group. Because there was likely to have been some variability in RT as participants became familiar with the task (e.g., asking the examiner for clarification), and the task difficulty altered during the first 60 trials, RTs from the first 60 trials have not been analysed. Figure 14 shows that mean Response Times are similar in the two groups. A 2 X 2 X 2 ANOVA with Group, Version and Order entered as between-subjects variables (see Table 33, Appendix C) found no significant main or interaction effects between the two groups.

Speed-accuracy and discriminability vs. response bias.

To determine whether the three signal detection parameters ($\log d$, $\log b$, and RT) were related, these variables were checked to see if they were correlated with one another (see Table 35, Appendix C). Because the variables of Group, Order, Version, and Block may have exerted various main or interaction effects on both $\log b$ and $\log d$, data were grouped according to diagnostic group (ADHD-C or ND), Block (Trials 1-60 or Trials 61-120), Order (1st or 2nd Task) and Version (Version 1 or Version 2). Correlations were then calculated within each of these cells. An Alpha value of 0.0031 (two tailed) was used to correct for multiple comparisons (Curtin & Schulz, 1998). There was no evidence of *speed-accuracy trade-off*, that is, there were no significant correlations between RT and $\log d$ in either of the groups within either block of trials.

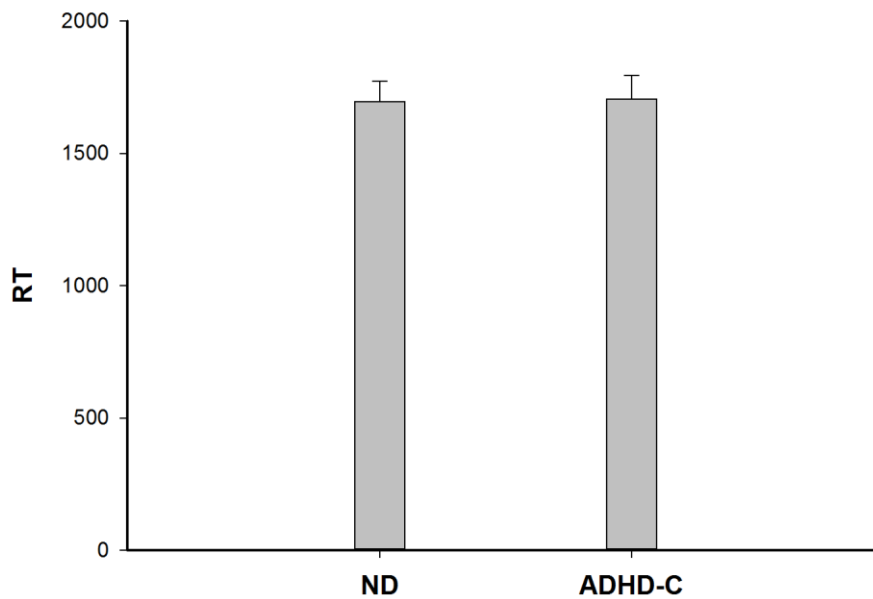


Figure 14. Mean response time (RT) for combined type ADHD (ADHD-C) and normally developing (ND) groups over final block of 60 trials. Because there were no main or interaction effects, both orders and versions have been combined.

Log b and log d were also checked to see if they were correlated with one another. In the ADHD-C group who completed Version 1 of the current study after Study 1 (i.e., Order 2), more accurate performance tended to occur in participants who were less strongly influenced by the differences in reward immediacy. This trend, however, was only significant in the second block of 60 trials ($r=-0.93$, $p<.0001$).

Children in the ND group who completed Version 1 of the task in Order 1 tended to have *more* accurate performance when they were more strongly influenced by the differences in reward immediacy, although this was not significant at the .0031 level.

To summarise the relationship between log b , log d , and RT, there was no evidence of speed-accuracy trade-off (i.e., relationship between RT and log d). The relationship between

$\log b$ and $\log d$ was somewhat difficult to interpret, given that the relationship was only significant in a small subgroup of children.

Exploratory analysis of predominantly inattentive ADHD group.

The ADHD-PIA group contained 15 participants, which is significantly fewer than the ADHD-C (44) and ND (80) groups. Because effects of Order and Version were present, formal analyses would have required these variables to be considered in any ANOVA. This would have left between 3 and 4 participants in each cell, which would have provided insufficient power. Consequently, $\log b$ in the ADHD-PIA has been examined informally. Figure 15 shows that for participants who completed the task in Order 1 (left hand panel), $\log b$ in the first block of trials is similar in the ADHD-PIA and ADHD-C groups, although the ADHD-PIA group shows more bias towards immediate reward in the second block of trials than the ADHD-C group. For participants who completed the task in Order 2, although $\log b$ in the ADHD-PIA group is higher than the ND and ADHD-C groups initially, all three groups showed a similar level of bias in the second block of trials, although this was still less than zero (i.e., a bias towards delayed reward). In general, these results are difficult to attach any weight to, given the inconsistent pattern, and the small number of participants in the ADHD-PIA group.

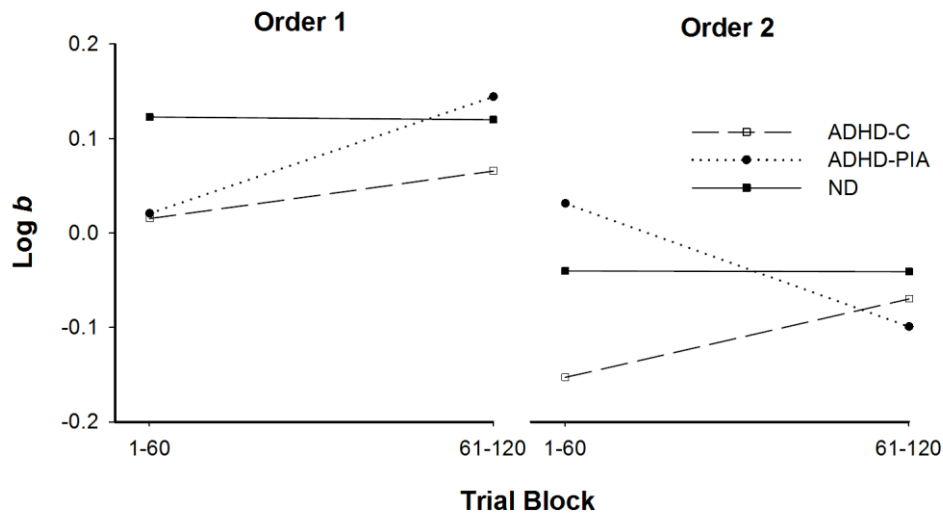


Figure 15. Response bias ($\log b$) is plotted for the combined type ADHD (ADHD-C), predominantly inattentive ADHD (ADHD-PIA), and normally developing (ND) groups, over each block of 60 trials. Because earlier analysis identified an effect of order, each order is plotted separately.

In the analysis of response bias following instances of immediate and delayed reward, Figure 16 shows that the ND and ADHD-C groups showed higher bias following delayed rewards. The ADHD-PIA group, however, showed higher bias towards immediate reward following instances of immediate reward for most Order-Version combinations except Order 1 - Version 1 (top left panel). The differences in all groups are relatively small however, and the most obvious finding is that Order 1 participants showed higher bias than Order 2 participants.

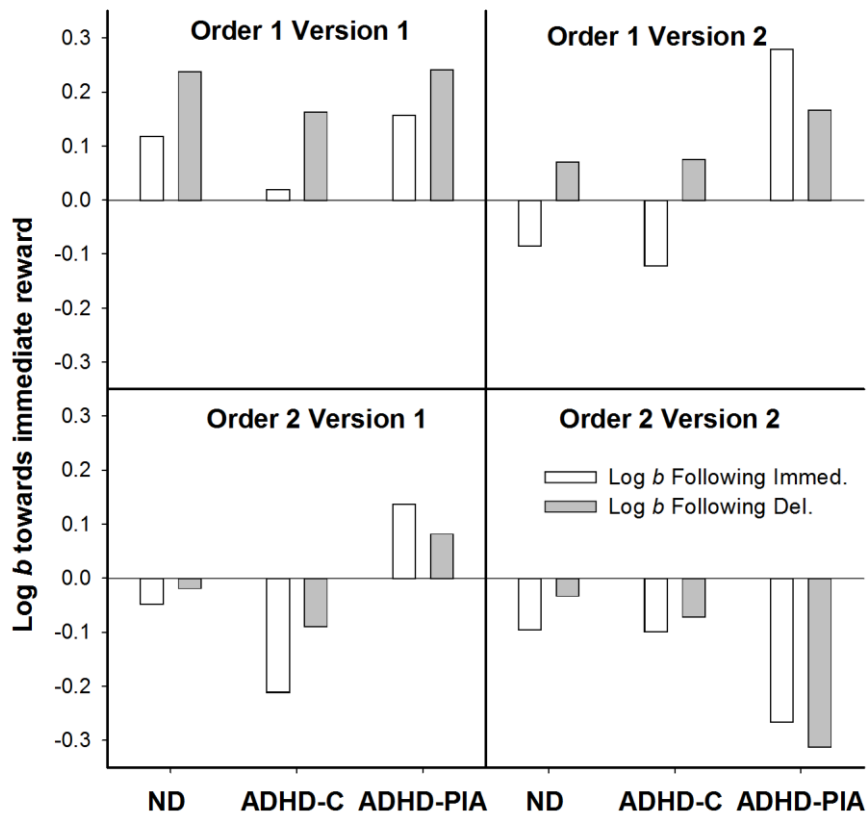


Figure 16. Log b towards immediate rewards is plotted for responses following immediate and delayed rewards for the combined type ADHD (ADHD-C), predominantly inattentive ADHD (ADHD-PIA), and normally developing (ND) groups. Because earlier analyses identified an effect of Order and Version, each Order-Version combination is plotted separately.

Chapter 7: Sensitivity to Reward Delay Discussion

The present study examined the effects of immediate and delayed rewards on the response bias of children with and without combined type ADHD²¹ in a signal detection task. Rewards were delivered either immediately, or after a 3.5 second delay for each of the two response alternatives. Based on previous research (Tripp & Alsop, 2001), it was expected that children would have developed response bias towards the immediately rewarded response. The experimental procedure did not, however, consistently generate the expected response bias towards the immediately rewarded response. There are some trends worth noting, however. There was a trend suggesting that the normally developing group showed a greater preference than the ADHD-C group for immediate reward early in the task, although the two groups were similar in the later part of the task.

Additionally, there was an effect of Order; participants who received Order 1 generally showed the expected positive response bias throughout the task. Conversely, participants who completed the current experiment after Experiment 1 (i.e., Order 2) generally showed *negative* response bias throughout the task, regardless of group membership. In other words, they showed a bias towards the *delayed* reward; this was an unexpected result and will be discussed later. Post-hoc analysis showed that the normally developing group showed bias significantly greater than zero in both trial blocks for Order 1, but not for Order 2. Response bias in the ADHD-C group was significantly *less* than zero in the first trial block for Order 2, but close to zero in the remaining conditions. This finding is surprising at first, but there is a likely explanation; the counterbalancing procedure appears to have complicated the results.

²¹ A small group of children with inattentive ADHD was also included. Informal analyses of these children were performed in the previous chapter, but will not be discussed in depth as the small number of participants precluded any firm conclusions from being drawn.

All participants completed both experiments and there were two versions of each task. Each participant completed a task where one stimulus was paired with immediate rewards in Study 2, and the same stimulus was paired with infrequent reward in Study 1. In other words, each participant completed two tasks with competing processes (e.g., develop a bias towards ‘red’ in one task and develop a bias towards ‘blue’ in the other). The difference in overall levels of response bias between the two Orders (see Figure 10) show a hysteresis effect, where bias in the first task reduced the bias that would usually develop in the second task. This hysteresis or interference effect is discussed further in the general discussion.

The previous analysis examined overall response bias within each trial block. In addition to this, the effect of recent reward history (i.e., whether the most recent reward was delayed or immediate) was examined. Responses were separated into those that followed immediate rewards and those that followed delayed rewards. The bias towards immediate reward was greater on trials following delayed rewards than following immediate rewards, although this finding was not significant.

The current experiment also examined discriminability (i.e., accuracy) and response times (RT) in the two groups. There were no significant differences between groups in RT. Both groups discriminated adequately between stimuli, with $\log d$ values indicating that most participants obtained approximately 75% to 80% correct. The experiment included a titration procedure to minimise inter-individual differences in discriminability. The titration procedure appears to have been effective as there were no significant between-group differences in discriminability.

Because there were differences between the two groups in age and estimated IQ scores, additional analyses were performed to determine whether age or estimated IQ were related to the dependent variables, and would, therefore, complicate the analyses. Correlations were performed between age and estimated IQ and the dependent variables. There was no

consistent evidence that age or cognitive functioning was significantly related to the dependent variables. Older children in the ADHD-C group tended to have shorter RTs in the second block of trials (i.e., there was a negative correlation between age and RT), but not in the first block of trials. Neither age nor estimated IQ was correlated with discriminability or response bias within either group during each of the trial blocks analysed.²²

The relationship between bias and discriminability was examined to determine whether any between-group differences in response bias could be attributed to differences in discriminability. There was no evidence that bias and discriminability were correlated in any of the trial blocks analysed.²³

Although there were some minor differences, the current experiment used methodology that is broadly largely similar to Tripp and Alsop (2001). Both studies compared the signal detection performance of similar-aged groups of boys with and without ADHD. Both arranged rewards for correct responses that occurred immediately, or after a delay. Both studies examined any resultant response bias towards the immediate reward. The similarities in design of the two studies allow their results to be compared. In terms of response bias, the results of the current study differed from Tripp and Alsop's results. First, the current study found that children with ADHD-C showed slightly less bias towards immediate reward than normally developing children (although this difference was not significant), whereas Tripp and Alsop found that children with ADHD showed greater bias than normally developing

²² There was a significant correlation between age and response bias in the first block of trials for the ADHD-C group, although this block of trials was excluded from further analysis because task difficulty was not consistent for each participant due to the titration procedure.

²³ There were significant correlations between response bias and discriminability in the first block of trials, although this pattern was different for each group (negative correlation in the ADHD-C group, positive correlation in the normally developing group). Again, this block of trials was excluded from further analysis due to the titration procedure.

children towards immediate reward. Second, when participants' responses following immediate rewards were compared with responses following delayed rewards, children with and without ADHD-C both showed a trend suggestive of a higher response bias (towards immediate reward) following instances of delayed reward. Tripp and Alsop found that children with ADHD showed similar levels of bias regardless of whether they had recently received immediate or delayed rewards, whereas control children showed higher bias towards immediate rewards when they had just received delayed rewards. Overall, the results of the current experiment did not replicate Tripp and Alsop's finding that children with ADHD differ from control children in terms of the local effects of reward. In terms of discriminability and RT, the results of the current experiment are similar to Tripp and Alsop's. There were no significant differences between groups in either study.

Despite the similarities between the present experiment and Tripp and Alsop's (2001), there are several methodological differences, which may, in part, account for some of the differences between their results. Because the two studies showed very similar results in terms of discriminability and RT, the following discussion will focus on those factors that are likely to have influenced response bias. First, in the current study, participants completed two experiments. The order effect found complicated the interpretation of results, and may have obscured differences between the ADHD-C and normally developing groups. This was not an issue for Tripp and Alsop, whose participants only completed one experimental task. Secondly, the two studies did not use exactly the same rewards, which may account for different findings in terms of response bias. In both studies, when a reward was delivered, participants were given a token, the experimenter praised the participant, a message was displayed on screen, and an animation was displayed on screen. At the end of the experiment the tokens could be exchanged for prizes. The two studies differed in the types of animation displayed on screen when rewards were delivered; the current study paired a novel animated

cartoon with each reward delivered, whereas children in the Tripp and Alsop study were shown the same animated fireworks display with each reward. Overall, the rewards in the current experiment might be expected to be more reinforcing than those used by Tripp and Alsop. The more salient rewards might have been expected to produce greater response bias towards the immediately rewarded response than Tripp and Alsop found. Clearly, this did not occur. The range of response bias in the current experiment was similar to that reported by Tripp and Alsop for participants who received Order 1, but markedly lower for participants who received Order 2. A possible explanation for the lack of expected between-group differences in response bias is the relationship between reward delay and reward value.

When a delay occurs between behaviour and the reward that follows the behaviour, the reinforcing effect of the reward is lessened. The greater the delay the less effective the reinforcer is. The relation between the delay before the reward and the effectiveness of the reward is the delay of reinforcement gradient (e.g., Ainslie & Herrnstein, 1981; Green, Fry, & Myerson, 1994; Green & Myerson, 2004). Although there is some debate about the exact shape of function that describes the delay of reinforcement gradient (reviewed by Green & Myerson, 2004), hyperbolic solutions are generally favoured over exponential as they predict the preference reversal that occurs when a smaller reward is preferred over the larger reward when the larger reward is sufficiently delayed. Equation 3 describes the function suggested by (Green et al., 1994).

$$V = \frac{A}{(1+kD)^s} \quad (3)$$

V is the subjective value of the reinforcer, A is the amount of the reinforcer, and D is the delay that occurs before the reinforcer is delivered. K and s are both parameters that affect the resultant function. Figure 17 shows the delay of reinforcement gradients for two rewards of different amounts. The subjective value of each reward declines with increasing delays. The two rewards depicted in Figure 17 have their highest value when they are delivered

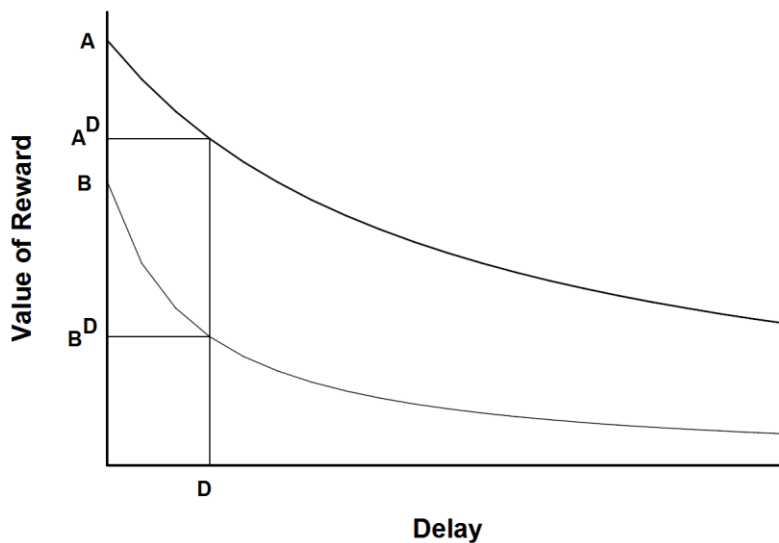


Figure 17. Hypothetical delay of reinforcement gradient for rewards of two amounts. The gradients are based on the hyperbolic formula (3) from Green et al., (1994). The value of the larger reward is marked as A on the y-axis. The value of the smaller reward is marked as B on the y-axis.

immediately (i.e., at zero delay). The value of the larger reward when delivered immediately is denoted A on the graph, and the value of the smaller reward is denoted B . When rewards are delayed by a given interval (D) their value declines. The reduced values for the two rewards after a delay of D are denoted A^D and B^D respectively. Because the current experiment uses rewards of equal amount, but unequal delay to produce a response preference the difference between the value of the immediate and delayed rewards (i.e., $A - A^D$ and $B - B^D$) are of primary interest. These differences reflect response preference and are analogous to response bias.

Given that the current experiment was somewhat more visually interesting than Tripp and Alsop's (2001) experiment, and the reward sequence was more varied, it is possible that the overall strength or 'amount' of each reward was greater in the current experiment. Figure

17 shows that $A - A^D$ is greater than $B - B^D$ which suggests that larger rewards might be less effective than smaller rewards at developing response bias towards immediate reward, at least for the delay D selected in Figure 17. Therefore, a possible reason for the lack of consistent bias towards the more immediately rewarded response in the current experiment is that the effective amount of reward on each alternative (e.g., A and A^D in Figure 17) was too similar in the current experiment. Another difference between the current experiment and Tripp and Alsop (2001) is the number of trials used. Participants in the current study completed 120 trials whereas Tripp and Alsop's participants completed 200 trials (although only data from the final 150 trials were analysed). Although there were no significant differences between groups in the current experiment, there was a trend towards $\log b$ values increasing from the first to the second block of trials (Figures 10 and 11) in the ADHD-C group. It also appears that response bias may have developed more rapidly in the normally developing group than in the ADHD-C group. Towards the end of the task, however, response bias for the two groups was similar. Because Tripp and Alsop did not examine their data in the same manner as the current experiment, and their participants completed a longer task, direct comparisons are not possible. It is possible, however to make an educated guess about why the response bias findings in the two studies are different. Early in the current task, the normally developing group rapidly developed response bias towards the immediate reward, whereas bias was slower to develop in the ADHD-C group, and the two groups showed similar levels of bias towards the end of the task. Tripp and Alsop found that the children in the ADHD group showed a higher response bias towards the immediate reward. This pattern was not present at any stage of the current experiment, so it is possible that children with ADHD developed an increased response bias relative to the normally developing group towards the end of Tripp and Alsop's task. Essentially, it is possible that children with ADHD show a 'delayed development' of response bias compared to controls – and there is some evidence of this in

Study 1 - but if allowed enough experience to develop the bias, children with ADHD may develop higher response bias than control children.

The extended training in Tripp and Alsop' (2001) study could also account for the contrast between their experiment and the current experiment in terms of the local effects of rewards. It seems reasonable to assume that the local effects of reinforcers might also develop over time. However, given that many trials are removed from the analysis when focussing only on trials that occur immediately following rewards, the number of trials in the current experiment was too low to examine the development of the local effects of reinforcement over time (i.e., to examine the localised effect of reward within each trial block).

When considering the differences in methodology between the current experiment and Tripp and Alsop (2001), it appears that the fewer number of trials in the current experiment may have obscured potential between-group differences that may have developed over the course of a longer task. It is also possible that the rewards used in the current experiment were more salient than those used by Tripp and Alsop. Reward delay and reward frequency are two aspects of reward that can affect response bias. It also possible, however, that reward saliency also affects response bias, although this has received little attention in the literature. It is likely that these three aspects of reward can sometimes act in concert, and sometimes in conflict. It is possible that in cases where rewards are particularly salient, the reward delay becomes less disruptive.

Although there were few statistically significant results in the current experiment, there were some interesting discrepancies between the results of the current experiment, and Tripp and Alsop's results. The current experiment found that children with ADHD may develop bias towards immediate reward more slowly than do control children, whereas Tripp and Alsop found that children with ADHD developed higher levels of bias over a long task than did control children. It seems likely that task length is a variable that may markedly alter

the pattern of results found. There were also differences in the localised effects of reward between the current study and Tripp and Alsop. In the current experiment, both groups of children showed a trend towards greater bias towards the immediate reward following individual instances of immediate reward compared with responses following delayed rewards. Tripp and Alsop, however, found that control children (but not children with ADHD) showed more bias towards immediate reward following individual instances of delayed reward. It is possible that this difference only developed towards the end of Tripp and Alsop's procedure, although this cannot readily be determined as Tripp and Alsop did not report their results over successive blocks of trials. Another aspect of the current study, which complicated the interpretation of results, is the hysteresis effect of task order. Participants developed markedly different levels of response bias depending on whether or not they had previously experienced a task designed to develop response bias. Because this hysteresis effect is a result of the combined results of both experiments in the current study, this will be discussed further in the final chapter.

Chapter 8: General Discussion

Two studies both used signal detection procedures to compare the response to reward of boys with- and without attention deficit-deficit/hyperactivity disorder. The purpose of these studies was to examine the nature of altered reward sensitivity in children with ADHD. Study 1 arranged different frequencies of reward for each response. Study 2 arranged equal numbers of reward for the two responses, but ensured that rewards for one response were immediate while the other responses were rewarded after a delay. In both experiments the reward contingencies were intended to develop a preference (i.e., response bias) towards one response. Based on previous literature (Tripp & Alsop, 1999; Tripp & Alsop, 2001), participants were expected to show a response bias towards the more frequently rewarded response (in Study 1) or the immediately rewarded response (in Study 2). In addition to response bias, measures of response time and discriminability were also obtained, although these will not be discussed in this chapter as there were minimal differences between groups.

In Study 1, boys with combined type ADHD (ADHD-C) showed reduced effects of unequal reward frequency relative to normally developing boys, although this pattern was only evident towards the end of the experimental task. This pattern suggests that reinforcement for children with ADHD-C may be less effective. Study 1 also included a small number of boys with inattentive ADHD (ADHD-PIA). In terms of response bias, the boys with ADHD-PIA tended to show a similar pattern to the normally developing boys. This implies that altered reward sensitivity to unequal reward frequency may only be relevant for theories regarding ADHD-C but not ADHD-PIA. The results of Study 1 also suggest that boys with ADHD-C are more influenced by their immediate reward history than normally developing boys. When responses that occurred after frequently and infrequently rewarded responses were examined, boys with ADHD-C showed reduced response bias following

‘lean’ rewards, whereas response bias in the normally developing boys did not alter based on the most recently delivered reward. Some of the boys with ADHD-PIA appeared similar to the normally developing boys, although these results were less consistent, probably due to the small number of participants in the ADHD-PIA group.

In Study 2, there were a number of unexpected effects. In general, the degree of response bias towards immediate reward was low and was close to zero in many cases. The ADHD-C group showed slightly less bias towards immediate reward (particularly in the early stages of the task) than the normally developing group. These results were not significant, and were opposite to what was expected based on previous research (Tripp & Alsop, 2001). When the local effects of reward were examined, the ADHD-C and normally developing groups showed a greater bias towards immediate reward on trials directly following instances of delayed reward than on trials directly following instances of immediate reward, although these results were not significant. In general, the results of Study 2 did not show any marked differences between groups.

There were some aspects of the current method that complicated the interpretation of results, but also highlighted areas that are worthy of further examination. Because each participant completed both tasks, task order was examined as a potential confound. Additionally, two versions of each task were used to control for stimulus and position effects; each participant completed one task where ‘more red’ responses were more frequently (or immediately) rewarded and one task where ‘more blue’ responses were more frequently (or immediately) rewarded. The results of the current experiments indicate that there were both order and task effects.

In Study 1, there was a significant Block by Version effect, with participants who completed Version 2 of the task (where ‘more red’ left-hand-side responses were rewarded more frequently than ‘more blue’ right-hand-side responses) showing a greater increase in

bias over the course of the task than participants who completed Version 1 of the task (where ‘more blue’ right-hand-side responses were rewarded most frequently). In Study 2 there was a significant main effect of Order, with participants completing Study 2 before Study 1 (i.e., Order 1) showing a higher bias towards the immediately rewarded stimulus than participants who completed the tasks in the reverse order (i.e., Order 2).

There are no obvious explanations for the task version effect being found in Study 1 but not in Study 2. It is worth noting, however, that the version by block interaction approached significance in Study 2. It is possible, therefore, that the task version effect was present in both experiments, but was not large enough to have been statistically significant in Study 2. It is also possible that the large order effect found in Study 2 may have obscured any possible effects of task version.

In both studies when more frequent (or immediate) reward was paired with the ‘more blue’ response, bias was relatively high and stable, but when more frequent (or immediate) reward was paired with ‘more red’ responses, bias was lower originally, but increased relatively rapidly over the course of the tasks. The ‘more blue’ response was made by pressing the right hand button of the response panel in both experiments. It is possible that this pattern of responding reflects an initial handedness preference which alters when the handedness bias is ‘overcome’ when the opposing responses are reinforced more frequently or immediately. When ‘more blue’ (right handed) responses are reinforced more frequently/immediately than ‘more red’ responses bias tends to be higher initially because of the innate handedness preference.

The order effect may have a relatively simple explanation. Because each participant completed two tasks and the stimuli paired with more immediate or frequent reinforcement were counterbalanced, each participant was effectively expected to show a preference reversal. Because the overall level of bias was lower in Study 2 than in Study 1, preference

reversal would likely occur more rapidly when Study 2 was completed first. For example, if a participant completed Study 1 task first, he might have had ‘more red’ responses paired with more frequent reinforcement, and then, in the second experiment, he would have had ‘more blue’ responses paired with more immediate reward. In this case, we would expect to see a positive bias towards ‘more red’ develop over the course of Study 1. In Study 2, the participant might initially show a negative bias towards the ‘more blue’ response²⁴ but then develop positive bias over the course of the task. Because bias was generally higher in Study 1 than Experiment 2, participants who completed Study 1 first effectively had a higher level of response bias to overcome to achieve preference reversal than participants who completed Study 2 first. This could explain why the order effect was apparent in Study 2 but was not apparent in Study 1.

Given that the interpretations of the current experiments were complicated by order effects, a post-hoc measure of preference reversal was calculated for each participant. The bias in the final block of the first study (Block 3 for Study 1, Block 2 for Study 2) that the participant completed was subtracted (with the sign reversed) from the bias in the first block of the second study the participant completed. For participants who completed Study 1 before Study 2, this is a measure of preference reversal that occurred from the end of Study 1 to the early stage of the Study 2. Figure 18 (left panels) suggests that when participants completed Study 1 first, the normally developing (ND) group showed a quicker preference reversal than the ADHD-C group, particularly those participants who completed Version 2 of Study 1 (lower left panel). The right panels of Figure 18 include participants who completed Study 2 before Study 1 and are included as a comparison. The measure is calculated the same way (i.e., $\log b$ in first block of Study 2 minus the negative of $\log b$ in the third block of Study 1).

²⁴ A positive bias towards one response is equivalent to negative bias towards opposite response.

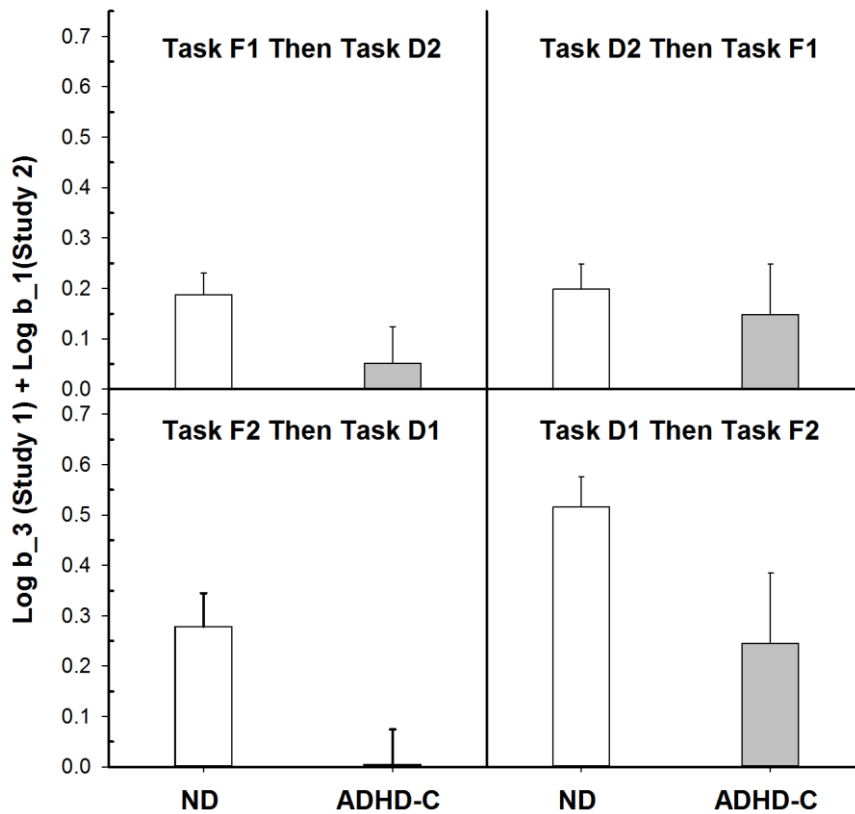


Figure 18. Left Panels: Preference reversal occurring in normally developing (ND) and combined type ADHD (ADHD-C) participants who completed Study 1 before Study 2 by task version. Preference reversal was calculated by adding the bias score for each participant within the final block of Study 1 to the bias scores within the first block of Study 2. Right panels: A comparison with participants who completed Study 2 before Study 1 by task version. Scores were calculated by adding the bias score for each participant within the final block of Study 1 to the bias scores within the first block of Study 2. F1 and F2 are version 1 and version 2 of Study 1. D1 and D2 are version 1 and version 2 of Study 2.

If the differences in preference reversal in the left hand panels were purely a function of between-group differences in response bias (in the third block of trials in Study 1 and the first block of trials in Study 2), then the left and right panels would show the same pattern. Because the difference between the ADHD-C and ND groups is larger in the left panels, this

difference likely reflects a difference in preference reversal rather than merely differences in response bias within blocks of trials.

The left panels of Figure 19 show a similar analysis examining preference reversal occurring from the final block (Block 2) of Study 2 to the first block of Study 1. Again, the right panels of Figure 19 compare ND and ADHD-C participants who completed Study 1 before Study 2. The left panels show no clear differences in preference reversals between groups. This lack of a between-groups difference in preference reversal probably reflects the relatively small response bias developed in the Study 2 task. If the participants completed Study 2 before Study 1, then they effectively had less response bias to ‘overcome’ during the second task than if they completed the tasks in the reverse order. The difference in preference reversal is consistent with literature showing that children with ADHD have difficulty adjusting to rule changes on the Wisconsin Card Sorting Task (e.g., Barkley, 1997a), although it should be noted that the Wisconsin Card Sorting Task differs from the current tasks in that it involves conscious cognitive processing of the sorting rules.

The possibility that ADHD-C and normally developing children have different responses to changes in reward contingency is interesting and represents another potential difference between children with ADHD and controls that was not initially expected. Future research could examine this aspect of response to rewards intentionally. For example, a task similar to Study 1 could be designed to reverse the reward contingencies part way through the task. This would allow a formal analysis of the preference reversal and would also allow an intra-individual comparison.

Effect of Reward Frequency on Preference

Differences between children with ADHD-C and normally developing children were most pronounced in Study 1, where children with ADHD-C showed a lower rate of increase

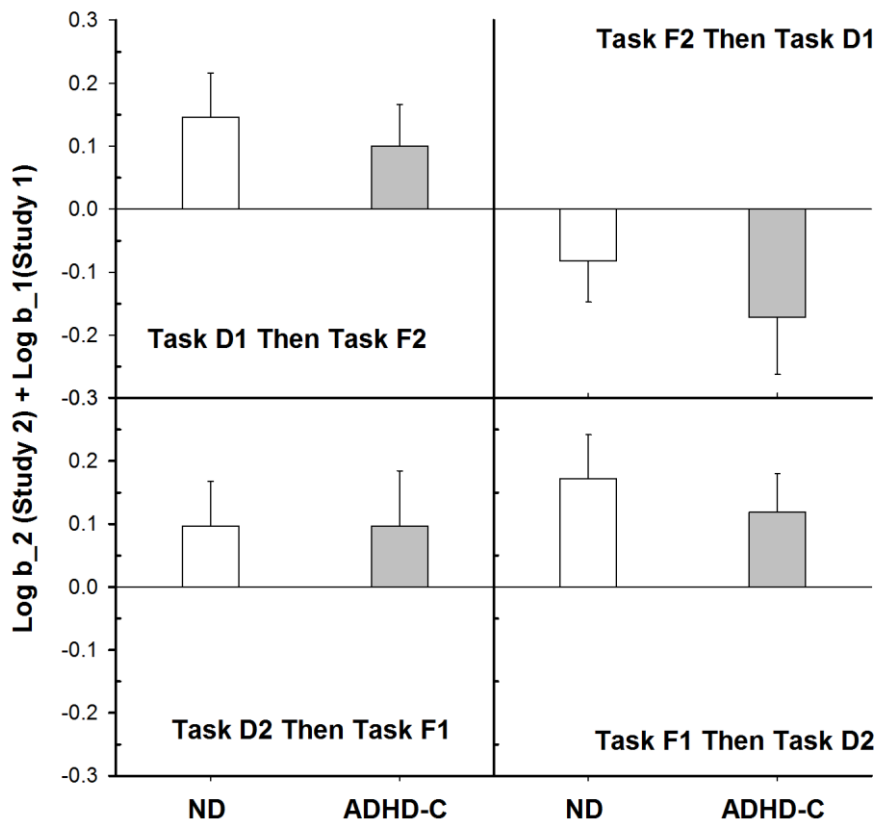


Figure 19. Left Panels: Preference reversal occurring in normally developing (ND) and combined type ADHD (ADHD-C) participants who completed Study 2 before Study 1 by task version.

Preference reversal was calculated by adding the bias score for each participant within the final block of Study 2 to the bias scores within the first block of Study 1. Right panels: A comparison with participants who completed Study 1 before Study 2 by task version. Scores were calculated by adding the bias score for each participant within the final block of Study 2 to the bias scores within the first block of Study 1. F1 and F2 are version 1 and version 2 of Study 1. D1 and D2 are version 1 and version 2 of Study 2

in response bias over trials compared to normally developing children. Although this result is most readily compared with Tripp and Alsop (1999), the results of Study 1 are also consistent with previous studies showing that children with ADHD are less sensitive to changes in

reward frequency in both concurrent schedule procedures (e.g., Kollins et al., 1997) and in simple choice procedures (e.g., Schweitzer & Sulzer-Azaroff, 1995).

Effect of Reward Delay on Preference

In Study 2 the results were somewhat unexpected in that participants did not consistently generate response bias towards the immediately rewarded response alternative. This finding contrasts with Tripp and Alsop (2001) and with other literature showing that children with ADHD show a greater preference for immediate reward than control children (Schweitzer & Sulzer-Azaroff, 1995), and that children with ADHD are more strongly influenced by reward immediacy than control children (Neef et al., 2005). Alternatively, the results of Study 2 could be interpreted as being consistent with (Sonuga-Barke et al., 1992b) who found that children with ADHD only showed a preference for smaller immediate rewards when this lead to a higher level of reinforcement overall, and that children with ADHD were generally delay averse, in that they tended to behave in a manner that was likely to reduce the overall length of the task. The results of Study 2 cannot, however, be confidently interpreted as being consistent with the delay aversion hypothesis favoured by Sonuga-Barke et al. (1992b). An aspect of the current results that is inconsistent with Sonuga-Barke et al. is that the ADHD-C group showed a bias towards *delayed* rewards. It should be noted, however that this ‘finding’ is not consistent with any other known theory regarding reinforcement and ADHD either. This apparent bias towards delayed rewards is likely a result of certain aspects of the experimental procedure, which are discussed later in this chapter.

Theories of ADHD

Some of the theories on ADHD discussed in Chapter 2 do not make clear predictions about how the performance of children with ADHD would differ from that of controls on signal detection tasks such as those used in the current experiments. For example, it somewhat

difficult to make predictions about performance in the current experiments from Sergeant's model (2000; Sergeant et al., 1999), which hypothesises that ADHD is a result of differences between ADHD and control children in their allocations of various modes of cognitive energy. The following theories can, however, be used to make predictions on the current experiments.

Although the methods used in the current experiments differ from those used as evidence for the following theories, the results are generally consistent with predictions drawn from Barkley's (1997a, 1999, 2006a) theory of ADHD. Barkley's theory of ADHD proposes that children with ADHD (ADHD-C and ADHD-PHI) have a core deficit in response inhibition. According to Barkley, response inhibition contains three interrelated processes: inhibiting a previously reinforced response, stopping an on-going response, and interference control. There is also evidence that children with ADHD satiate to reinforcement more rapidly than control children (Barkley, 1989; Iaboni, Douglas, & Ditto, 1997). Although Barkley's theory does not relate directly to the current experiments, it is possible to interpret aspects of current results in light of Barkley's theory, in particular, the ability to inhibit a previously reinforced response. For participants that completed Study 1 first (where, for example, blue responses were rewarded more frequently than red responses) the participants would be expected to inhibit to some degree the 'blue' response when they later completed Study 2. Figure 18 shows that for the participants who completed Study 1 first, the normally developing group achieved preference reversal more readily than the ADHD-C group.

The second aspect of Barkley's theory relevant to the current experiments is his suggestion that children with ADHD satiate to reward more rapidly than control children. Although the response bias in the ADHD-C group did not diminish over trial blocks in Study 1 (as would be predicted by Barkley's theory), bias in the ADHD-C group developed relatively slowly, whereas response bias in the normally developing group increased more

rapidly throughout the task. Children with ADHD may not satiate to rewards per se, but the accumulation of multiple rewards over time appears to have a lesser influence on children with ADHD-C than normally developing children.

Of the mechanisms Douglas (1983) posited as accounting for many of the difficulties experienced by hyperactive children, a strong inclination to seek immediate reinforcement relates best to the primary measures used in the current experiments. A strong inclination to seek immediate reinforcement would result in a higher response bias (towards the immediately rewarded response) in Study 2. The results of Study 2 were not consistent with this prediction for reasons discussed earlier. Specifically, the relative saliency or value of the rewards used (relative to the delay in place) and the relatively short number of trials may have obscured between group differences.

Sonuga-Barke (2003) and Sagvolden (2005; 1998) both proposed theories that are relevant to the current experiments. The results of the current experiments are not strongly consistent with either theory. Sonuga-Barke proposed a dual pathway model of ADHD in which delay aversion and executive dysfunctions make separate contributions to ADHD symptoms (2002; 2003; Sonuga-Barke et al., 2003). Although the proposed executive dysfunctions are less relevant to the current experiments, Sonuga-Barke's theory of delay aversion makes a prediction about response bias in the Study 2 that contrasts with predictions based on the work of Tripp and Alsop (2001). According to Sonuga-Barke (2003) children with ADHD are delay averse, and will behave in ways that reduce the overall delay of tasks. According to this theory, children with ADHD should not show a preference for immediate rewards, provided the overall delay and density of reinforcement is equal for the two response alternatives. Tripp and Alsop (2001) found that children with ADHD showed a greater preference for immediate rewards than did control children. Study 2 at first seems consistent with Sonuga-Barke's predictions, although there are reasons to interpret the current results

with caution. The current experiment was short compared to that used by Tripp and Alsop (2001). It seems likely that between-group differences may have eventually developed, had the task contained more trials.

Sagvolden's approach (2005; Sagvolden et al., 1998) views symptoms of ADHD as expressions of deficits in reinforcement mechanisms. In Sagvolden's model, ADHD results from initial deficits in three (mesocortical, mesolimbic, and nigrostriatal) dopaminergic pathways, which can be a result of genetic endowment, and/or exposure to toxins or drugs. Although there are several different facets of this model, the pathway that is more relevant for the current experiment is mesolimbic system, which causes shorter delay-of-reinforcement gradients and deficient extinction processes. In this model, the behaviour of children with ADHD is more persistent when reinforcement is removed (i.e., extinction) than the behaviour of control children. More importantly (for the current experiment) the delay-of-reinforcement gradients are shorter in children with ADHD than controls. Short (or steep) delay-of-reinforcement gradients mean that the relationship between reinforcer delay and reinforcer effectiveness declines steeply. Although there is debate about whether the delay-of-reinforcement gradients are exponential or hyperbolic in form there is evidence that the effectiveness of rewards decays with increasing pre-reward delays (Green & Myerson, 2004). According to Sagvolden's theory, delayed rewards should be more effective for control children than children with ADHD. In light of the current (delay) experiment, we should expect to see a greater bias towards immediate reward in the ADHD group than in the control group. In fact, this was not the case in the current experiment, although Tripp and Alsop's (2001) results were consistent with Sagvolden's position. Given that the current procedure used different (possibly more effective) rewards than Tripp and Alsop (2001) used, and that this difference could reasonably be expected to influence the delay-of-reinforcement gradient (Green & Myerson, 2010), it is possible that the delays used in the current experiment were

not long enough to reveal clear differences between groups. Additionally, as noted earlier, because the current experiment was short compared with Tripp and Alsop's, it is possible that the length of the task was too short for measurable statistically significant differences between groups to occur.

In Tripp and Wickens' (2008; 2009) dopamine transfer deficit theory of ADHD some of the symptoms of ADHD occur because children with ADHD have deficits in dopamine function. Specifically, children with ADHD experience dopamine release only when rewards are actually delivered, whereas control children also experience dopamine release when an imminent reward is signalled. This could explain the divergence between response bias in ADHD and normally developing children towards the end of Study 1 (see Figure 3) where the response bias of the normally developing group increased at a greater rate than the bias of the ADHD-C group. If the apparent effect of a reward is the sum of the direct effect of the reward and the effect of the reward signal, and the response to the reward signal is attenuated in children with ADHD, then the response bias of the ADHD-C group would likely have a lower 'ceiling' than the response bias in the normally developing group. It is also possible that the variable response bias in the ADHD-C group compared with the ND group shown in Figure 4, which examines response bias over floating windows of 20 trials, is consistent with Tripp and Wickens' theory. In this case, the apparent 'slumps' in response bias could be a result of periods where rewards are not delivered, or are delivered infrequently. Periods of non-reward in the ND group would be less likely to be associated with a reduction in bias.

Limitations and Strengths of Current Experiments

There are several strengths in the methodology of the current experiments. Clinically-referred participants were diagnosed using a multi-informant, multi-method assessment. The assessment procedure also screened for other disorders that could influence performance on

the dependent variables. The control participants were selected from schools that minimised any differences in socioeconomic factors between groups. Additionally, IQ was measured in both the control and ADHD participants in order to examine whether IQ differences confounded the results.

The signal detection procedures used in the current experiments offer the benefit of providing two independent measures of response distribution: discriminability and response bias (i.e., preference). Further, the titration procedure used in the current experiments manipulated task difficulty to ensure that all participants achieved a similar level of discriminability despite likely individual differences in skill level. The titration procedure allowed differences in task difficulty between groups, meaning that group differences in discriminability do not necessarily imply differences in task ability. Discriminability can, however, be interpreted as a measure of sustained attention over the course of the tasks.

The demographic characteristics of the three groups were similar. Although there were some differences between groups in age and IQ, these did not appear to influence markedly the dependent variables. Of particular importance, there were no significant correlations between IQ and Age and response bias. It is likely, therefore, that any differences in response bias between groups can be attributed primarily to true differences between groups (i.e., diagnostic status) rather than other differences between groups such as intelligence, age, or socioeconomic status.

One of the limitations of the current experiments was the relatively short number of trials used. Study 1 contained 180 trials, which was fewer than the 300 trials used by Tripp and Alsop (1999). Study 2 contained 120 trials. Again, this is fewer than the 200 trials used in the delay task of Tripp and Alsop (2001). In Study 1, bias did not remain static, but developed over the course of the task. This pattern was less clear in Study 2, where there were small

increases from the first trial block to the second trial block in the ADHD-C group, but relatively constant bias in the normally developing group.

If bias develops over trials, then between-group differences might be accentuated in long tasks. For example, in Study 1, the normally developing group showed significantly greater bias than the ADHD-C group in the third block of trials, but not earlier in the task. It is also possible that longer tasks could show opposite results to shorter tasks. For example, in Study 2, the bias in the ADHD-C group (which is lower than the bias in the normally developing group) increases slightly from the first to the second block of trials, whereas bias in the normally developing group remains stable over trial blocks. It is possible that if this pattern were to continue (i.e., if participants had completed more trials) the ADHD-C group might develop greater bias than the control group. It is also possible that had the numbers of trials used in the current experiments been higher between-group differences in the local effects of reward may have been more apparent (cf. Tripp & Alsop, 1999; Tripp & Alsop, 2001).

Summary

The most robust result from the current two experiments is that boys with ADHD-C developed bias towards frequently reinforced stimuli more slowly than normally developing boys. Although there were no significant differences in the overall level of bias between groups, the results of the frequency experiment were consistent with previous literature which found that children with ADHD show a lower bias towards frequently rewarded stimuli than control children (Tripp & Alsop, 1999). In terms of theories of ADHD, this finding is consistent with Barkley's (1989) position that children with ADHD habituate to rewards more rapidly than control children. Tripp and Wickens (2008; 2009) have proposed a potential mechanism for this effect. According to Tripp and Wickens' Dopamine Transfer Deficit

theory of ADHD, some of the symptoms of ADHD occur because children with ADHD have deficits in dopamine function. This theory could explain the divergence between response bias in ADHD and normally developing children towards the end of Study 1

There were several factors that complicated the interpretation of the results of the current studies, particularly Study 2. Effects of Version and Order may have partially obscured between-group differences. Additionally, it is possible that both experiments contained too few trials for clear between-group differences in response bias to emerge. The hysteresis effect that emerged, where response bias in the first experiment influenced response bias in the second experiment is worth a brief discussion. Although the current studies were not designed to detect this, it also appears that children with ADHD are less able to show a preference reversal in response to a change in reward contingencies (see Figure 18).

Future research examining the effect of reward frequency and delay can be informed by the results of the current experiment in several ways. Clearly, any future attempts to replicate the findings of Tripp and Alsop should address some of the limitations discussed earlier.

First, it seems likely that the relatively short number of trials used in the current experiments obscured between-group differences. The primary reason that the current tasks used fewer trials than Tripp and Alsop (1999; 2001) was that the addition of animated pictures to the reward sequence added time to each individual trial. In the future, if this type of experiment were to be repeated, it would be beneficial to increase the number of trials. This would likely increase the likelihood of revealing any differences between groups, particularly differences in the rates at which groups acquire response bias. Increasing the number of trials could also increase the likelihood of detecting differences between groups in the effect of recent reward history on response bias.

Secondly, it is also possible that because the between-group differences were relatively small, the Order and Version effects, in Study 2 and Study 1 respectively, became relatively prominent. The Version effect found in the Study 1 would be difficult to control for. As discussed in Chapter 6, stimulus and place preference effects do occur (Johnstone & Alsop, 1996). With the current methodology, it cannot be determined with certainty whether the task version effects are a result of stimulus or place preference. This could be addressed by reversing the key positions for half the participants. This would allow a comparison of right preference versus left preference, which is not possible in the current studies. This would, however, require higher number of participants. Alternatively, participants could be required to use only one hand to respond with (e.g., by using a mouse to click responses). This could reduce any possible place preference, and would not require additional participants to maintain statistical power.

The order effect found in the Study 2 is likely a result of interference. Participants who completed Study 2 after Study 1 had to overcome a previously acquired preference in order to develop a bias towards the immediately rewarded stimulus. Participants who completed Study 2 first did not have any pre-existing preference to overcome. This artefact of the experimental procedure can be viewed in two ways. This artefact can be viewed as a confounding variable to be controlled for. It could be possible to achieve this by increasing the time period of time between the first and second tasks. Alternatively, the Order effect can be viewed as an additional source of information. A similar task to the tasks used in the current experiments could reverse the contingencies during the task to compare the rate of preference reversal for ADHD and control groups. If, as suggested by Barkley (2006a) children with ADHD have difficulty inhibiting a prepotent (i.e., previously reinforced) response, then control children would be likely to reverse their preference more rapidly than children with ADHD.

Finally, there were some features of responses that were not captured in the current experiments. During the tasks, some children were observed to push the response keys very hard, and hold the keys down for longer periods of time than required for a response to be recorded. Also, some children pressed the same button repeatedly. Because only the first response following stimulus onset was recorded, other responses were missed. It could be useful to compare children with ADHD and controls on these dimensions of responding. Differences in how hard the keys are pressed and how long they are pressed for could reflect differences in motor coordination or emotional regulation.

Conclusion

Of the many different methods to examine response to reinforcement of children with ADHD, the signal detection methods developed by Tripp and Alsop (1999; 2001) used in the current experiments offer a number of strengths. Reward delay and frequency are not confounded (cf. Schweitzer & Sulzer-Azaroff, 1995) and the response to reward in both of the current experiments is independent of task performance. The current experiments contained refinements on the original methods used by Tripp and Alsop (1999; 2001) that were designed to minimise the between-group differences in accuracy (discriminability) and minimise differences in the number of rewards between groups. Additionally, the ratio of rewards associated with each response (in Study 1) was increased, and the experimental task contained a number of improvements (e.g., the addition of animated pictures) that were designed to make the scheduled reinforcers more rewarding. Despite these changes, the results were somewhat equivocal. In Study 1, boys with ADHD-C developed a preference towards frequent reward more slowly than normally developing boys. Additionally, there was a trend suggestive that boys with ADHD-C are more sensitive to their recent history of reward, whereas the preference of normally developing children is governed more by their overall

reward history. In Study 2, there were no clear differences between ADHD-C and normally developing groups in the preference for immediate reward.

Although the number of children with ADHD-PIA was smaller than the number of children in the normally developing or ADHD-C groups, the results of Study 1 were suggestive that children with ADHD-PIA may have more in common with control children than children with ADHD-C, at least in terms of their responses to reward. If this was to be found consistently, this could be evidence that ADHD-PIA and ADHD-C may involve different behavioural or neurological mechanisms. This could go some way to resolving the difficulty in identifying which children with ADHD have altered reward mechanisms (Luman, Tripp, & Scheres, 2010).

There have been many different approaches that have examined the effect of rewards in children with ADHD (reviewed by Luman et al., 2005; Luman et al., 2010). Although there remains considerable work to be done in this area, using response bias to measure the effect of reward has several advantages over other approaches as it is independent of task competence or accuracy, and response rate. Additionally, response bias does not appear to be influenced by IQ, which tends to covary with diagnostic status.

Unexpected effects of Version (i.e., whether ‘more red’ or ‘more blue’ responses were associated with immediate/frequent reward) and Order were found. Although there are no obvious theoretically relevant implications of the Version effect (for further discussion of this see Chapter 5), there are implications of the Order effect. As discussed earlier, there is some evidence that boys with ADHD-C were less able than normally developing boys to overcome their previously developed response/stimulus preference when the reward contingencies were reversed. This could be seen as a form of perseveration, which has been suggested as a characteristic of children with ADHD (e.g., Barkley, 2006a). It is unclear how preference reversal relates to most of the reinforcement based theories of ADHD, particularly the

theories relating to reward delay (e.g., Sonuga-Barke, 2003). If replicated, the current findings could be counter-evidence for the theory of Tripp and Wickens (2008; 2009), who suggested that children with ADHD are less likely than controls to receive dopaminergic activation (i.e., neural reinforcement) in response to reward signals. The preference reversal found in the current experiments suggests that the rewards have a more 'resilient' effect in boys with ADHD-C than normally developing boys. Future applications of the current experimental techniques could deliberately examine the manner in which preference reversal occurs in response to reversed reward contingencies.

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Appendices

Appendix A: Participant Information and Consent Forms

Appendix A1: Permission to contact form for control families.

SENSITIVITY TO REWARD

I,, hereby give my consent for the researchers

(Parent's full name)

from the "Reward Sensitivity" study to contact me by telephone.

I understand that the purpose of the telephone interview will be to check that my

child,, is eligible to take part in the study

(Child's full name)

and to answer any questions that I might have about the study.

I understand that allowing the researchers to contact me does not commit me to taking part in the study.

Signature:

Date:

Child's Date of Birth:

Phone Number:

Address:

.....

.....

Principal Investigator: Dr E. Gail Tripp
Senior Lecturer in Psychology

Co-investigators: Dr Brent Alsop
Mr Ben McEachen
Ms Dianne Morrison (03) 479 5114
Dr Paula Sowerby (03) 479 5692
Ms Holly Fentiman
Ms Katrina Sugrue
Ms Brigitte Gorman
Mr Simon Seal

Appendix A2: Permission to contact form for control families.**SENSITIVITY TO REWARD**

Referral for assessment: Paediatric Department

I,, hereby give my consent for the researchers

(Parent's full name)

from the "Reward Sensitivity" study to contact me by telephone.

I understand that the purpose of the telephone interview will be to check that my

child,, is eligible to take part in the study

(Child's full name)

and to answer any questions that I might have about the study.

I understand that allowing the researchers to contact me does not commit me to taking part in the study.

Signature:

Date:

Child's Date of Birth:

Phone Number:

Address:

.....

.....

Principal Investigator:

Dr E. Gail Tripp

Senior Lecturer in Psychology

Co-investigators:

Dr Brent Alsop

Mr Ben McEachen

Dianne Morrison (03) 479 5114

Dr Paula Sowerby (03) 479 5692

Holly Fentiman

Brigette Gorman

Simon Seal

Appendix A3: Teacher consent form for control children.

SENSITIVITY TO REWARD

I have read and I understand the Information Sheet for volunteers participating in the study designed to assess the sensitivity to reward of children with and without attention deficit hyperactivity disorder (ADHD). I understand the nature and purpose of this research and the time required in taking part. I have had the opportunity to discuss this study and to ask questions which have been answered to my satisfaction.

I understand that taking part in this study is voluntary and that I may withdraw from the study at any time without penalty of any kind.

I understand that my participation in this study is confidential and that no material that could identify me will be used in any reports on this study.

I have been given sufficient time to understand and decide whether to participate in the current study.

If you have any concerns or questions regarding this study please contact Dr Gail Tripp (479 7624) or Dianne Morrison at the ADHD Research Clinic, University of Otago.
Ph: 479 5114

I,, hereby consent to take part in this study.

(Teacher's full name)

Signed Date

Appendix A4: Teacher consent form for clinically referred children.

SENSITIVITY TO REWARD

I have read and I understand the Information Sheet for volunteers participating in the study designed to assess the sensitivity to reward of children with and without attention deficit hyperactivity disorder (ADHD). I understand the nature and purpose of this research and the time required in taking part. I have had the opportunity to discuss this study and to ask questions which have been answered to my satisfaction.

I understand that taking part in this study is voluntary and that I may withdraw from the study at any time without penalty of any kind.

I understand that my participation in this study is confidential and that no material that could identify me will be used in any public reports on this study.

I understand the researchers will prepare a summary report of the findings of each child's diagnostic assessment which will be made available to that child's parents and, with their authorisation, to health and/or educational professionals involved in the child's care.

I have been given sufficient time to understand and decide whether to participate in the current study.

If you have any concerns or questions regarding this study please contact Dr Gail Tripp (479 7624) or Dianne Morrison at the ADHD Research Clinic, University of Otago. Ph: 479 5114

I,, hereby consent to take part in this study.

(Teacher's full name)

Signed Date

Appendix A5: Parent consent form for control children.

SENSITIVITY TO REWARD

I have read and I understand the Information Sheet for volunteers taking part in the “Reward sensitivity” study. I understand the nature and purpose of this research and the time required in taking part. I have had the opportunity to discuss this study and to ask questions which have been answered to my satisfaction.

I understand that taking part in this study is voluntary and that I may withdraw from the study at any time without any penalty of any kind for my family or my child.

I understand that my taking part and my child’s taking part in this study is confidential and that no material that could identify my child, my family, or me will be used in any public reports on this study.

I have had sufficient time to consider whether to take part in the current study.

If you have any concerns or questions about the study please contact Dr Gail Tripp (479 7624) or Dianne Morrison at the ADHD Research Clinic, University of Otago. Ph: 479 5114.

I,hereby consent for my child,

(Parent’s/Guardian’s full name)

..... to take part in this study.

(Child’s full name)

Signature of Parent/Guardian Date

I give my permission for my child's teacher, at

(Teacher's name)

in room at to complete the described

(room number)

(School name)

questionnaires about my child's behaviour.

Signature Date

Appendix A6: Parent consent form for clinically referred children.

SENSITIVITY TO REWARD

I have read and I understand the Information Sheet for volunteers taking part in the “Reward sensitivity” study. I understand the nature and purpose of this research and the time required in taking part. I have had the opportunity to discuss this study and to ask questions which have been answered to my satisfaction.

I understand that taking part in this study is voluntary and that I may withdraw from the study at any time without any penalty of any kind for my family or my child.

I understand that my taking part and my child’s taking part in this study is confidential and that no material that could identify my child, my family, or me will be used in any public reports on this study.

I understand the researchers will prepare a summary report of the findings of my child’s diagnostic assessment which will be made available to me, and with my permission to the health professionals involved in my child’s care.

I have had sufficient time to consider whether to take part in the current study.

If you have any concerns or questions about the study please contact Dr Gail Tripp (479 7624) or Dianne Morrison at the ADHD Research Clinic, University of Otago. Ph: 479 5114.

I,hereby consent for my child,

(Parent’s/Guardian’s full name)

..... to take part in this study.

(Child's full name)

Signature of Parent/Guardian Date

I give my permission for my child's teacher, at

(Teacher's name)

.....to complete the described questionnaires and interview

(School name)

about my child's behaviour.

Signature Date

I give my permission for a copy of the diagnostic assessment report to be sent to my
child's health professional

(Health Professionals name)

Signature Date.....

Principal Investigator:

Dr E. Gail Tripp (Phone: (03) 479 7624)

Senior Lecturer in Psychology

Co-investigators:

Dr Brent Alsop

Ben McEachen

Dianne Morrison (03) 479 5114

Dr Paula Sowerby

Holly Fentiman

Sasha Gold

Brigette Gorman

Child's date of birth **Child's Age**

Address: **Phone number**

.....

.....



Appendix A7: Information sheet for control children.

SENSITIVITY TO REWARD

We would like to invite your family to take part in a study we are carrying out that compares the way children with and without attention deficit hyperactivity disorder (ADHD) respond to reward. In particular we are interested in how the frequency of reward and delays in reward affects children's performance on a computer task. This information will be helpful in the development of treatment and educational programmes for children with ADHD. Your family is being asked to take part because your child does not have ADHD.

About The Study

Families of children who have been diagnosed with ADHD, families of children referred for an assessment for ADHD, and a group of families whose children do not have ADHD (control group) are being asked to consider taking part in this study. Families who are interested in taking part are asked to complete and return the enclosed consent form within two weeks of receiving this letter. The study will take place at your child's school.

If your family agrees to participate in the study the following things will happen. We will:

- Ask you to complete three questionnaires. One questionnaire asks about background information such as your child's age and sex, the other two questionnaires ask about your child's behaviour. Altogether these questionnaires will take about 30 minutes to complete. These questionnaires will be posted to you together with a stamped envelope for their return.

- Obtain your permission to send your child's teacher two questionnaires about your child's behaviour to complete, and a brief scale to rate their academic progress.
- Approach your child at school and ask them to complete two computer tasks, some tasks designed to assess working memory ability, and an abbreviated measure of intellectual functioning. The latter takes about 25 minutes and involves defining the meanings of some words and selecting pictures that best match or complete a series of pictures. The two computer tasks take place in separate sessions in the morning and afternoon. When all tasks are included, the morning session will take approximately one hour, and the afternoon session will take approximately 35 minutes.

In the computer tasks your child will be shown a series of checkerboards of blue and red cartoon characters. He or she will be asked to choose between two response buttons to indicate whether there were more blue or more red cartoon characters. These tasks assess how the frequency of reward and delays in reward influence children's responses on the task. Each task takes 20 minutes to complete and will be carried out on the same day. After completing each computer task children can cash in the tokens they earned playing the game for art supplies up to the value of \$5.00.

In the working memory tasks your child will be asked to repeat back sequences of numbers and letters, copy spatial sequences (by tapping and pointing), and repeat back lists of words in a specified order. The working memory tasks are brief, and together will take about 25 minutes to complete.

Risk, Benefits, Safety

Taking part in this study will not directly benefit your child, however other children who have completed the tasks report enjoying them. The information collected from the computer tasks will help us to better understand how children with and without ADHD respond to differences in the frequency of reward and delays in reward. The information collected from the working memory tasks will help us to better understand the types of learning and memory difficulties that children with ADHD often experience. It is hoped that this information will assist in the development of more effective behavioural and educational programmes for children with ADHD.

All of the data collected during the study will remain confidential. All families who take part will be given a code number, and this number, not your names, will be used on all questionnaires. Any written reports on the findings of the study will describe the data from groups of children and their families. The information collected about your family will not be reported in such a way that would allow you or your child to be identified.

Your family's participation is voluntary (your choice). You do not have to take part in this study. If you do agree to take part you are free to withdraw from the study at any time, without having to give a reason and with no repercussions (negative outcomes) for you or your child.

A summary of the results of the study will be sent to all participating families and teachers.

If you have any questions or would like to know more about the study before deciding whether or not your family would like to participate please contact Dr Paula

Sowerby (03 479 5692) or Dianne Morrison (03 479 5114). Asking questions about the study does not commit you to taking part. If your family would like to take part in the study please complete the consent form and return it to the researchers in the stamped addressed envelope, within two weeks.

This research has been approved by the Lower South Regional Ethics Committee.

If you have any queries or concerns regarding your rights as a participant in this study you may wish to contact a Health and Disability Advocate, telephone (03) 479 0265 or 0800 377 766.

If there are specific Māori issues/concerns please contact Linda Grennell on 0800 377 766.

Yours sincerely,



Principal Investigator: Dr Gail Tripp

Co-investigators:	Dr Brent Alsop	Holly Fentiman
	Ben McEachen	Brigette Gorman
	Simon Seal	
	Dr Paula Sowerby (03 479 5692)	
	Dianne Morrison (03 479 5114)	

Please keep this Information Sheet for future reference.

Appendix A8: Information sheet for clinically referred children.

SENSITIVITY TO REWARD

We would like to invite your family to take part in a study we are carrying out that compares the way children with and without attention deficit hyperactivity disorder (ADHD) respond to reward. In particular we are interested in how the frequency of reward and delays in reward affects children's performance on a computer task. This information will be helpful in the development of treatment and educational programmes for children with ADHD. Your family is being asked to take part because your child has been referred to Paediatric outpatients with symptoms of inattention, overactivity, or impulsivity.

About The Study

Families of children who have been diagnosed with ADHD, families of children referred for an assessment for ADHD, and a group of families whose children do not have ADHD (control group) are being asked to consider taking part in this study. Families who are interested in taking part are asked to complete and return the permission to contact form in the enclosed envelope. After receiving the permission to contact form the researchers will telephone you to answer any questions you have about the study, to check that your child is eligible to participate, and arrange for you and your child to participate if you are still interested. The study will take place at the ADHD Research Clinic at the University of Otago.

If your family agrees to participate in the study the following things will happen. We will:

- Ask you and your child to complete participant consent forms.

- Ask you to complete two questionnaires about your child's behaviour. Together these questionnaires take about 25-30 minutes to complete.
- Obtain your permission to send your child's teacher two questionnaires about your child's behaviour and to interview them over the telephone about how your child is getting on at school.
- Make appointments for you and your child to visit the ADHD Research Clinic, Psychology Department, University of Otago. This will involve three visits, first, an appointment with you alone, and second, two appointments with your child.

In the first visit, we will interview you about your child's current and previous difficulties. At this time, you will be asked whether or not you are prepared for the interview to be audio taped for interviewer training purposes. This decision is entirely voluntary. If you agree, we will ask you to sign a consent form. This interview covers a wide range of questions relating to emotional and behavioural problems that can occur in childhood. Part of this standard interview will include questions about whether your child has ever experienced any traumatic or frightening life events. Your child will be asked a similar series of questions in their interview.

This visit will take approximately 3 hours.

Your child will then be asked to visit the ADHD Research Clinic on two occasions. At one of the visits we will ask your child to:

- Complete a cognitive assessment designed to assess their thinking, problem solving, and academic skills.

- Complete two computer tasks. In the computer tasks your child will be shown a series of checkerboards of blue and red cartoon characters. He or she will be asked to choose between two response buttons to indicate whether there were more blue or more red cartoon characters. These tasks assess how the frequency of reward and delays in reward influence children's responses on the task. After completing each computer task children can cash in the tokens they earned playing the game for art supplies up to the value of \$5.00.

This visit will take approximately 3 hours.

At another visit we will ask your child to:

Complete an interview about their feelings, behaviour and any past and current difficulties.

Complete five brief tasks designed to assess both verbal and spatial working memory. In these tasks your child will be asked to repeat back sequences of numbers and letters, copy spatial sequences (by tapping and pointing), and repeat back lists of words in a specified order. The working memory tasks require a total time duration of approximately 25 minutes.

This visit will take up to 2 hours.

To cover the cost of transport all families will receive a \$10.00 petrol voucher each time they visit the ADHD Research Clinic.

Risk, Benefits, Safety

Individual children will receive a comprehensive diagnostic assessment together with an assessment of their cognitive and academic functioning. The information collected from the computer tasks will help us to better understand how children with

ADHD respond to differences in the frequency of reward and delays in reward. The information collected from the working memory tasks will help us to better understand the types of learning and memory difficulties that children with ADHD often experience. It is hoped that this information will assist in the development of more effective behavioural and educational programmes for children with ADHD.

A report will be prepared summarizing the results of each child's assessment and this will be made available to you through the person your child has been seeing at Paediatric Outpatients. This may assist them in planning future treatment for your child.

All of the data collected during the study will remain confidential. All families who take part will be given a code number, and this number, not your names, will be used on all questionnaires. Apart from the assessment report that you receive about your child (and any reports you authorize to be supplied to other professionals involved with your family), any written reports on the findings of the study will describe the data from groups of children and their families. The information collected about your family will not be reported in such a way that would allow you or your child to be identified.

Your family's participation is voluntary (your choice). You do not have to take part in this study. If you do agree to take part you are free to withdraw from the study at any time, without having to give a reason and with no repercussions (negative outcomes) for you or your child.

When the study is completed, a summary of the overall results of the study will be sent to all participating families and teachers.

If you have any questions or would like to know more about the study before deciding whether or not your family would like to participate please contact Dr Paula Sowerby (03 479 5692) or Dianne Morrison (03 479 5114). Asking questions about the study does not commit you to taking part. If you think your family might like to take part in the study please complete and return the permission to contact form in the stamped addressed envelope.

This research has been approved by the Lower South Regional Ethics Committee.

If you have any queries or concerns regarding your rights as a participant in this study you may wish to contact a Health and Disability Advocate, telephone (03) 479 0265 or 0800 377 766.

If there are specific Māori issues/concerns please contact Linda Grennell on 0800 377 766.

Yours sincerely,



Principal Investigator: Dr Gail Tripp

Co-investigators: Dr Brent Alsop

Ben McEachen

Dr Paula Sowerby (03 479 5692)

Dianne Morrison (03 479 5114)

Holly Fentiman

Brigette Gorman

Simon Seal

Please keep this Information Sheet for future reference.

Table 5.

Descriptive statistics for response bias (log b), following frequent ('Rich') reward and infrequent ('Lean') reward, for normally developing (ND), combined type ADHD (ADHD-C), and inattentive ADHD (ADHD-PIA) groups. Statistics are provided separately for each reward type, version, and order.

			ND			ADHD-C			ADHD-PIA		
Reward Type	Version	Order	Mean	SD	N	Mean	SD	N	Mean	SD	N
Following Lean Reward	F1	1	0.159	0.320	20	0.204	0.386	11	0.007	0.503	2
		2	0.109	0.357	15	0.030	0.265	11	0.060	0.357	3
		Total	0.137	0.332	35	0.117	0.335	22	0.039	0.358	5
	F2	1	0.082	0.249	17	0.130	0.358	9	0.306	0.047	2
		2	0.090	0.327	18	-0.091	0.332	10	0.043	0.086	6
		Total	0.086	0.288	35	0.014	0.354	19	0.109	0.143	8
	Total	1	0.124	0.288	37	0.171	0.366	20	0.156	0.339	4
		2	0.099	0.336	33	-0.028	0.298	21	0.049	0.191	9
		Total	0.112	0.310	70	0.069	0.344	41	0.082	0.236	13
Following Rich Reward	F1	1	0.165	0.257	20	0.251	0.180	11	0.467	0.409	2
		2	0.055	0.277	15	0.185	0.347	11	-0.001	0.182	3
		Total	0.118	0.268	35	0.218	0.272	22	0.186	0.352	5
	F2	1	0.109	0.223	17	0.158	0.313	9	0.105	0.081	2
		2	0.139	0.204	18	0.017	0.357	10	0.147	0.243	6
		Total	0.125	0.211	35	0.084	0.336	19	0.136	0.209	8
	Total	1	0.139	0.240	37	0.209	0.246	20	0.286	0.319	4
		2	0.101	0.240	33	0.105	0.354	21	0.097	0.225	9
		Total	0.121	0.239	70	0.156	0.307	41	0.155	0.260	13

Table 6.

Mean discriminability (log d), standard deviation, and number of participants for normally developing (ND), combined type ADHD (ADHD-C), and inattentive ADHD (ADHD-PIA) groups.

Means are provided separately for each block of trials, task version, and order.

			ND			ADHD-C			ADHD-PIA			
Trial	Block	Version	Order	Mean	SD	N	Mean	SD	N	Mean	SD	N
1-60	F1		1	0.772	0.202	20	0.657	0.216	11	0.602	0.199	2
			2	0.713	0.216	15	0.629	0.148	11	0.518	0.121	3
		Total	0.747	0.207	35	0.643	0.181	22	0.552	0.139	5	
			1	0.684	0.167	17	0.481	0.189	9	0.511	0.021	2
			2	0.737	0.236	18	0.671	0.136	10	0.599	0.283	6
		Total	0.711	0.204	35	0.581	0.186	19	0.577	0.243	8	
	Total		1	0.732	0.189	37	0.578	0.218	20	0.557	0.127	4
			2	0.726	0.224	33	0.649	0.141	21	0.572	0.235	9
			Total	0.729	0.205	70	0.614	0.184	41	0.567	0.203	13
61-120	F1		1	0.525	0.257	20	0.536	0.227	11	0.851	0.013	2
			2	0.491	0.235	15	0.475	0.218	11	0.778	0.107	3
		Total	0.510	0.245	35	0.506	0.220	22	0.807	0.086	5	
			1	0.608	0.236	17	0.515	0.272	9	0.600	0.081	2
			2	0.451	0.232	18	0.447	0.189	10	0.405	0.167	6
		Total	0.527	0.244	35	0.479	0.228	19	0.453	0.170	8	
	Total		1	0.563	0.248	37	0.527	0.242	20	0.726	0.152	4
			2	0.469	0.231	33	0.462	0.201	21	0.529	0.235	9
			Total	0.519	0.243	70	0.493	0.221	41	0.590	0.227	13
121-180	F1		1	0.530	0.258	20	0.475	0.216	11	0.806	0.511	2
			2	0.565	0.207	15	0.539	0.303	11	0.547	0.071	3
		Total	0.545	0.234	35	0.507	0.259	22	0.651	0.296	5	
			1	0.565	0.221	17	0.409	0.192	9	0.668	0.012	2
			2	0.516	0.219	18	0.361	0.102	10	0.403	0.168	6
		Total	0.539	0.218	35	0.384	0.149	19	0.469	0.188	8	
	Total		1	0.546	0.239	37	0.446	0.203	20	0.737	0.305	4
			2	0.538	0.212	33	0.454	0.243	21	0.451	0.155	9
			Total	0.542	0.225	70	0.450	0.221	41	0.539	0.241	13

Table 9.

Response bias (log b): Tests of within-subjects contrasts. Between-subjects factors are Group, Version, and Order. The within-subjects factor is Block.

Source	Block	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Observed Power ^a
Block	Linear	42.41	1	103	0.000	0.292	1.000
	Quadratic	1.40	1	103	0.239	0.013	0.217
Block x Group	Linear	4.94	1	103	0.028	0.046	0.596
	Quadratic	0.07	1	103	0.798	0.001	0.057
Block x Version	Linear	7.38	1	103	0.008	0.067	0.768
	Quadratic	0.37	1	103	0.544	0.004	0.093
Block x Order	Linear	0.88	1	103	0.350	0.008	0.153
	Quadratic	2.45	1	103	0.121	0.023	0.341
Block x Group x Version	Linear	0.73	1	103	0.395	0.007	0.135
	Quadratic	0.00	1	103	0.990	0.000	0.050
Block x Group x Order	Linear	0.00	1	103	0.982	0.000	0.050
	Quadratic	2.85	1	103	0.094	0.027	0.387
Block x Version x Order	Linear	0.00	1	103	0.961	0.000	0.050
	Quadratic	1.43	1	103	0.234	0.014	0.220
Block x Group x Version x Order	Linear	0.15	1	103	0.696	0.001	0.067
	Quadratic	0.03	1	103	0.854	0.000	0.054

a. Computed using alpha = .05

Table 12.

Discriminability (log d): Multivariate ANOVA Table. Mixed Design. Between-subjects factors are Group, Version, and Order. The within-subjects factor is Block.

Effect	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Observed Power ^a
Group	2.291	1	103	0.133	0.022	0.323
Version	0.666	1	103	0.416	0.006	0.128
Order	0.966	1	103	0.328	0.009	0.164
Block ^b	0.278	1	103	0.599	0.003	0.278
Group x Version	0.989	1	103	0.322	0.010	0.166
Group x Order	0.083	1	103	0.774	0.001	0.059
Version x Order	1.005	1	103	0.318	0.010	0.168
Block x Group ^b	3.019	1	103	0.085	0.028	0.3019
Block x Version ^b	2.304	1	103	0.132	0.022	0.2304
Block x Order ^b	3.729	1	103	0.056	0.035	0.3729
Group x Version x Order	0.074	1	103	0.787	0.001	0.058
Block x Group x Version ^b	0.657	1	103	0.419	0.006	0.657
Block x Group x Order ^b	0.039	1	103	0.844	0.000	0.039
Block x Version x Order ^b	0.156	1	103	0.694	0.002	0.156
Block x Group x Version x Order ^b	0.77	1	103	0.382	0.007	0.770

a Computed using alpha = .05

b This ANOVA only compared log *d* over the second and third blocks of trials

Table 14.

Response time (RT): Multivariate ANOVA Table. Mixed Design. Between-subjects factors are Group, Version, and Order. The within-subjects factor is Block.

Effect	F	Hypothesis df	Error df	Sig.	Partial Eta Squared	Observed Power^a
Group	0.90	1	103	0.344	0.009	0.156
Version	0.42	1	103	0.517	0.004	0.099
Order	0.07	1	103	0.799	0.001	0.057
Block ^b	2.42	1	103	0.123	0.023	0.338
Group x Version	0.16	1	103	0.687	0.002	0.069
Group x Order	0.72	1	103	0.399	0.007	0.134
Version x Order	0.48	1	103	0.490	0.005	0.105
Block x Group ^b	2.50	1	103	0.117	0.024	0.347
Block x Version ^b	0.67	1	103	0.416	0.006	0.128
Block x Order ^b	0.04	1	103	0.848	0.000	0.054
Group x Version x Order	2.18	1	103	0.143	0.021	0.309
Block x Group x Version ^b	0.35	1	103	0.555	0.003	0.090
Block x Group x Order ^b	1.44	1	103	0.232	0.014	0.222
Block x Version x Order ^b	2.41	1	103	0.124	0.023	0.336
Block x Group x Version x Order ^b	2.15	1	103	0.145	0.020	0.307

a Computed using alpha = .05

b This ANOVA only compared RT over the second and third blocks of trials

Table 15.

T-tests (one sample) for difference between $\log b$ and zero across trial blocks for combined type ADHD (ADHD-C) and normally developing (ND) groups, by Version and Block.

Version	Group	Trial Block	t	df	p (2 tailed)
Version 1	ND	1-60	2.03	34	0.051
		61-120	4.51	34	<0.001
		121-180	6.91	34	<0.001
	ADHD-C	1-60	3.76	21	0.001
		61-120	4.72	21	<0.001
		121-180	5.31	21	<0.001
Version 2	ND	1-60	-0.45	34	0.654
		61-120	3.62	34	<0.001
		121-180	7.60	34	<0.001
	ADHD-C	1-60	-0.70	18	0.492
		61-120	1.23	18	0.235
		121-180	3.34	18	0.004

Table 16.

Pearson product moment correlations between independent (age and estimated IQ score) and dependent ($\log b$, $\log d$, and response time) variables within the combined type ADHD (ADHD-C) and normally developing (ND) groups.

Variable	Block	ND		ADHD-C	
		Age	ESTIQ	Age	ESTIQ
$\log b$	1-60	0.16	-0.04	-0.07	0.18
	61-120	0.18	-0.05	0.03	0.01
	121-180	0.09	0.00	0.20	0.06
$\log d$	1-60	0.12	-0.02	0.07	0.23
	61-120	-0.10	0.21	-0.09	-0.07
	121-180	-0.12	-0.01	-0.15	-0.21
RT	1-60	-0.21	-0.17	-0.32*	0.17
	61-120	-0.13	-0.14	-0.34*	0.27
	121-180	-0.17	-0.07	-0.37*	0.38
* = $p < .05$					

Table 17.

Pearson product moment correlations between signal detection variables (log b, log d, and response time) for combined type ADHD (ADHD-C) and normally developing (ND) groups. Correlations between variables within the same trial block are underlined and in bold text.

Group	Variable	Log <i>b</i>		Log <i>d</i>			RT			
		Block	61-120	121-180	1-60	61-120	121-180	1-60	61-120	121-180
ND	Log <i>b</i>	1-60	0.61*****	0.45****	0.03	-0.26*	-0.30*	0.22	0.19	0.14
		61-120	1	0.54*****	-0.11	-0.21	-0.26*	0.07	-0.02	-0.02
		121-180		1	-0.03	0.02	0.10	-0.01	-0.04	-0.04
	Log <i>d</i>	1-60			1	-0.29*	-0.28*	0.24*	0.38***	0.35***
		61-120				1	0.58*****	-0.11	-0.09	-0.07
		121-180					1	-0.05	-0.04	-0.02
	RT	1-60						1	0.87*****	0.79*****
		61-120							1	0.94*****
		121-180								1
ADHD-C	Log <i>b</i>	1-60	0.41**	0.51***	0.10	-0.07	0.36*	0.11	-0.06	0.13
		61-120	1	0.54*****	-0.05	-0.08	0.19	0.34*	0.08	0.15
		121-180		1	-0.01	-0.27	0.15	0.06	-0.20	-0.16
	Log <i>d</i>	1-60			1	-0.21	-0.15	0.11	0.36*	0.39*
		61-120				1	0.56*****	0.22	0.25	0.09
		121-180					1	0.24	0.08	0.04
	RT	1-60						1	0.71*****	0.67*****
		61-120							1	0.85*****
		121-180								1
*=p<.05		***=p<.005		*****=p<.0005						
=p<.01		**=p<.001		*****=p<.0001						

Appendix C: Statistical Tables from Study 2 – Sensitivity to Reward Delay

Table 18.

Mean response bias (log b), standard deviation, and number of participants for normally developing (ND), combined type ADHD (ADHD-C), and inattentive ADHD (ADHD-PIA) groups. Means are provided separately for each block of trials, task version, and order.

			ND			ADHD-C			ADHD-PIA		
Trial											
Block	Version	Order	Mean	SD	N	Mean	SD	N	Mean	SD	N
1-60	D1	1	0.188	0.231	21	0.055	0.396	10	0.111	0.167	4
		2	0.052	0.300	20	-0.132	0.198	11	0.085	0.298	4
		Total	0.121	0.272	41	-0.043	0.315	21	0.098	0.224	8
	D2	1	0.033	0.229	15	-0.020	0.144	11	-0.051	0.254	5
		2	-0.116	0.204	24	-0.171	0.184	12	-0.074	0.178	2
		Total	-0.059	0.224	39	-0.099	0.180	23	-0.057	0.220	7
	Total	1	0.123	0.240	36	0.016	0.287	21	0.021	0.223	9
		2	-0.040	0.263	44	-0.152	0.187	23	0.032	0.257	6
		Total	0.033	0.264	80	-0.072	0.252	44	0.025	0.228	15
61-120	D1	1	0.147	0.250	21	0.134	0.173	10	0.153	0.180	4
		2	-0.034	0.268	20	-0.132	0.247	11	0.152	0.160	4
		Total	0.059	0.271	41	-0.005	0.250	21	0.152	0.158	8
	D2	1	0.083	0.418	15	0.004	0.283	11	0.138	0.108	5
		2	-0.046	0.157	24	-0.012	0.147	12	-0.600	0.424	2
		Total	0.003	0.289	39	-0.004	0.217	23	-0.073	0.409	7
	Total	1	0.120	0.327	36	0.066	0.241	21	0.145	0.134	9
		2	-0.041	0.212	44	-0.069	0.206	23	-0.099	0.449	6
		Total	0.032	0.280	80	-0.005	0.231	44	0.047	0.312	15

Table 19.

Descriptive statistics for response bias (log b), following immediate reward and delayed reward, for normally developing (ND), combined type ADHD (ADHD-C), and inattentive ADHD (ADHD-PIA) groups. Statistics are provided separately for each reward type, task version, and order.

			ND			ADHD-C			ADHD-PIA		
Reward											
Type	Version	Order	Mean	SD	N	Mean	SD	N	Mean	SD	N
Following Delayed Reward	D1	1	0.238	0.313	21	0.163	0.268	10	0.241	0.092	4
		2	-0.019	0.323	20	-0.089	0.345	11	0.082	0.194	4
		Total	0.113	0.340	41	0.031	0.329	21	0.162	0.164	8
	D2	1	0.071	0.303	15	0.075	0.303	11	0.167	0.313	5
		2	-0.033	0.281	24	-0.072	0.281	12	-0.312	0.157	2
		Total	0.007	0.290	39	-0.001	0.295	23	0.030	0.352	7
	Total	1	0.168	0.315	36	0.117	0.283	21	0.200	0.232	9
		2	-0.027	0.297	44	-0.080	0.306	23	-0.049	0.262	6
		Total	0.061	0.319	80	0.014	0.309	44	0.100	0.267	15
Following Immediate Reward	D1	1	0.118	0.284	21	0.019	0.392	10	0.157	0.437	4
		2	-0.048	0.338	20	-0.211	0.271	11	0.137	0.255	4
		Total	0.037	0.318	41	-0.101	0.346	21	0.147	0.332	8
	D2	1	-0.085	0.259	15	-0.122	0.271	11	0.279	0.227	5
		2	-0.095	0.281	24	-0.099	0.246	12	-0.266	0.015	2
		Total	-0.091	0.269	39	-0.110	0.252	23	0.124	0.324	7
	Total	1	0.034	0.288	36	-0.055	0.333	21	0.225	0.319	9
		2	-0.074	0.305	44	-0.153	0.259	23	0.003	0.287	6
		Total	-0.025	0.301	80	-0.106	0.297	44	0.136	0.316	15

Table 20.

Mean discriminability (log d), standard deviation, and number of participants for normally developing (ND), combined type ADHD (ADHD-C), and inattentive ADHD (ADHD-PIA) groups.

Means are provided separately for each block of trials, task version, and order.

			ND			ADHD-C			ADHD-PIA		
Trial											
Block	Version	Order	Mean	SD	N	Mean	SD	N	Mean	SD	N
1-60	D1		1 0.802	0.231	21	0.808	0.300	10	0.721	0.375	4
			2 0.788	0.161	20	0.668	0.193	11	0.724	0.180	4
		Total	0.795	0.198	41	0.735	0.254	21	0.723	0.272	8
	D2		1 0.744	0.224	15	0.516	0.200	11	0.588	0.084	5
			2 0.816	0.180	24	0.772	0.203	12	0.707	0.016	2
		Total	0.788	0.199	39	0.650	0.237	23	0.622	0.090	7
	Total		1 0.778	0.227	36	0.655	0.288	21	0.647	0.247	9
			2 0.803	0.170	44	0.722	0.201	23	0.718	0.140	6
		Total	0.792	0.197	80	0.690	0.246	44	0.675	0.208	15
61-120	D1		1 0.542	0.231	21	0.621	0.389	10	0.421	0.253	4
			2 0.572	0.176	20	0.533	0.206	11	0.663	0.328	4
		Total	0.556	0.204	41	0.575	0.302	21	0.542	0.300	8
	D2		1 0.457	0.242	15	0.696	0.294	11	0.491	0.352	5
			2 0.547	0.237	24	0.469	0.204	12	0.671	0.284	2
		Total	0.513	0.240	39	0.578	0.271	23	0.543	0.322	7
	Total		1 0.506	0.236	36	0.660	0.336	21	0.460	0.296	9
			2 0.558	0.209	44	0.500	0.203	23	0.666	0.284	6
		Total	0.535	0.222	80	0.576	0.283	44	0.542	0.299	15

Table 21.

Mean response time, standard deviation, and number of participants for normally developing (ND), combined type ADHD (ADHD-C), and inattentive ADHD (ADHD-PIA) groups. Means are provided separately for each block of trials, version, and order.

			ND			ADHD-C			ADHD-PIA		
Trial											
Block	Version	Order	Mean	SD	N	Mean	SD	N	Mean	SD	N
1-60	D1	1	1719	818	21	1564	631	10	1381	433	4
		2	1483	735	20	1353	173	11	1270	209	4
		Total	1604	778	41	1453	454	21	1325	320	8
	D2	1	2074	740	15	1558	567	11	1511	434	5
		2	1391	570	24	1503	497	12	1079	110	2
		Total	1654	715	39	1530	520	23	1388	415	7
	Total	1	1867	796	36	1561	583	21	1453	411	9
		2	1433	643	44	1431	378	23	1206	196	6
		Total	1628	744	80	1493	485	44	1354	355	15
61-120	D1	1	1692	694	21	1777	591	10	1375	500	4
		2	1660	805	20	1578	443	11	1362	279	4
		Total	1676	741	41	1673	516	21	1368	375	8
	D2	1	1950	674	15	1735	759	11	1464	402	5
		2	1571	551	24	1739	580	12	1292	14	2
		Total	1717	621	39	1737	656	23	1415	339	7
	Total	1	1799	688	36	1755	668	21	1424	420	9
		2	1612	671	44	1662	514	23	1338	219	6
		Total	1696	681	80	1706	587	44	1390	346	15

Table 28.

T-tests (one sample) for difference between $\log b$ and zero for combined type ADHD (ADHD-C) and normally developing (ND) groups, by Block and Order.

Order	Group	Trial Block	t	df	p (2 tailed)
Order 1	ND	1-60	3.08	35	0.0040
		61-120	2.21	35	0.0337
	ADHD-C	1-60	0.25	20	0.8047
		61-120	1.26	20	0.2234
Order 2	ND	1-60	-1.00	43	0.3206
		61-120	-1.27	43	0.2100
	ADHD-C	1-60	-3.90	22	0.0008
		61-120	-1.61	22	0.1211

Table 29.

T-tests (one sample) for difference between $\log b$ and zero for combined type ADHD (ADHD-C) and normally developing (ND) groups, by Version, Order, and Reward Type. $\log b$ is calculated over all trials.

Order	Version	Group	Reward Type	t	df	p (2 tailed)
Order 1	Version 1	ND	Foll Immediate	1.91	20	0.071
			Foll Delayed	3.48	20	0.002
		ADHD-C	Foll Immediate	0.15	9	0.881
			Foll Delayed	1.92	9	0.087
	Version 2	ND	Foll Immediate	-1.27	14	0.226
			Foll Delayed	0.91	14	0.379
		ADHD-C	Foll Immediate	-1.49	10	0.166
			Foll Delayed	0.83	10	0.429
Order 2	Version 1	ND	Foll Immediate	-0.64	19	0.532
			Foll Delayed	-0.26	19	0.797
		ADHD-C	Foll Immediate	-2.58	10	0.028
			Foll Delayed	-0.85	10	0.413
	Version 2	ND	Foll Immediate	-1.66	23	0.111
			Foll Delayed	-0.58	23	0.569
		ADHD-C	Foll Immediate	-1.40	11	0.190
			Foll Delayed	-0.88	11	0.396

Table 34.

Pearson product moment correlations between independent (age and estimated IQ score) and dependent (log b, log d, and response time) variables within the combined type ADHD (ADHD-C) and normally developing (ND) groups.

Variable	Block	ND		ADHD-C	
		Age	ESTIQ	Age	ESTIQ
Log <i>b</i>	1-60	-0.10	0.03	0.08	0.04
	61-120	0.07	-0.03	-0.08	0.16
Log <i>d</i>	1-60	0.12	0.18	0.33*	0.08
	61-120	-0.06	-0.03	-0.15	-0.28
RT	1-60	-0.14	-0.15	-0.18	-0.02
	61-120	-0.18	-0.09	-0.39**	0.28

* = $p < .05$ ** = $p < .01$

Table 35.

Pearson product moment correlations between signal detection variables (log b, log d, and response time) for combined type ADHD (ADHD-C) and normally developing (ND) groups. Correlations between variables within the same trial block are underlined and in bold text.

Group	Variable	Block	Log <i>b</i>		Log <i>d</i>		RT	
			1-60	61-120	1-60	61-120	1-60	61-120
ND	Log <i>b</i>	1-60	1	0.56*****	<u>0.23*</u>	-0.17	<u>-0.04</u>	-0.08
		61-120		1	0.27*	<u>-0.17*</u>	0.10	<u>0</u>
	Log <i>d</i>	1-60			1	-0.25*	<u>0.05</u>	0.09
		61-120				1	-0.14	<u>-0.16</u>
	RT	1-60					1	0.91*****
		61-120						1
ADHD-C	Log <i>b</i>	1-60	1	0.55*****	<u>-0.31*</u>	-0.07	<u>0.25</u>	0.13
		61-120		1	-0.04	<u>-0.06</u>	0.17	<u>0.14</u>
	Log <i>d</i>	1-60			1	-0.21	<u>0.18</u>	0.11
		61-120				1	0.18	<u>0.10</u>
	RT	1-60					1	0.78*****
		61-120						1

* = $p < .05$ *** = $p < .005$ ***** = $p < .0005$
 ** = $p < .01$ **** = $p < .001$ ***** = $p < .0001$