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An examination of the effects of stimulant medication on response inhibition: A comparison between children with and without attention deficit hyperactivity disorder

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Abstract

This study investigated whether methylphenidate is effective in improving response inhibition in children with Attention Deficit Hyperactivity Disorder (ADHD). Children with ADHD were compared with normally developing children on measures of response inhibition. Participants with ADHD were compared across two conditions – medicated and unmedicated. There was no significant difference between the inhibitory control of children with and without ADHD. Children with ADHD showed significant improvements in inhibitory control following methylphenidate. The findings of the present study contrast with previous studies which document reduced inhibitory control in ADHD, compared with normally developing children. Reports of methylphenidate improving functioning in children with ADHD are supported. Limitation and implications of the study are discussed.

Keywords: ADHD, Response Inhibition, Medication, Methylphenidate, Animal Stroop

1. Introduction

Attention Deficit Hyperactivity Disorder (ADHD) is a developmental disorder with the cardinal features of developmentally inappropriate levels of sustained attention, distractibility, hyperactivity, and impulse control (Barkley, 2000). It arises early in childhood and persists through adolescence and into adulthood in 30-70% of cases (Weiss & Murray, 2003). Current estimates indicate that 3-5% of the American population has ADHD (Goldman, Genel, Bezman, & Slanetz, 1998) with similar proportions within the UK population of primary-school aged children (Taylor, 1999). ADHD is known to have neuropsychological consequences that are evident from psychological tests and measures of school failure (Seidman, Biederman, Faraone, Weber, & Ouellett, 1997). As such, the disorder is associated with both concurrent and long-term impairments in academic and social functioning (Johnston, 1998). Comorbidity is common in ADHD and a significant proportion of those diagnosed go on to develop delinquent activities and antisocial personalities (Barkley, 2000).

Neuropsychological models now consider children with ADHD to have difficulties in terms of cognitive attention and executive functioning deficits (e.g. Lawrence, Houghton, Tannock, & Douglas 2002). More specifically, researchers have emphasized poor behavioural/response inhibition as the central impairment of the disorder (Barkley, 1990). Response inhibition is the ability to inhibit a prepotent response, interrupt an ongoing sequence and resist interference (Barkley, 1997) i.e. 'to stop (suddenly and completely) a planned or ongoing thought and action' (Williams, Ponsse, Schachar, Logan, & Tannock 1999, pg. 205).

Research has demonstrated that children with ADHD show marked impairments in inhibitory responses across a variety of tasks (e.g. Booth, Burman, Meyer, Lei, & Tronmer, 2005; Halperin, Newcorn, Sharma, Healey, & Wolf, 1990) and a review by Pennington and Ozonoff (1996) concluded that executive functioning deficits are consistent in ADHD. In addition, a high proportion of the measures that were found to be most sensitive to ADHD were tapping processes of inhibition, suggesting that measures of inhibition would appear to be particularly sensitive to ADHD. This suggestion is backed up by functional magnetic resonance imaging (fMRI) data (Booth et al., 2005; Rubia, Taylor, Smith, Oksannen, & Overmeyer, 2001). Thus, there appears to be a wealth of evidence offering support for a response inhibition deficit in children with ADHD

1.1 Treatment for children with ADHD

While the behavioural difficulties associated with ADHD are often managed with psychotherapeutic approaches, there is an absence of positive outcome studies (Brown & Levers, 1999). Instead, a large-scale multi-modal treatment study of children with ADHD demonstrated that the most effective treatment for ADHD was closely managed pharmacotherapy (MTA Cooperative Group, 1999). Despite several challenges to these findings (e.g. Owens & Hoza, 2003), well-titrated medical treatment for ADHD remains the treatment of choice (Hood, Baird, Rankin, & Isaacs, 2005).

Psychostimulants are the most commonly prescribed psychotropic agent for children and typically come in the form of short-acting immediate release stimulants, such as methylphenidate. Methylphenidate is believed to work by influencing the processes involved in the uptake and release of dopamine, which is associated with motivation and reward (Volkow, Wang, Fowler, Logan, Gerasimov, Maynard, et al., 2001). It has been suggested that people with ADHD may have low levels of dopamine within the brain and that methylphenidate allows interest in less motivating tasks to be maintained, and performance to be improved (Volkow et al., 2001). Indeed, there is evidence to suggest that stimulant medication significantly improves both the classroom and social behaviour of children with ADHD (Pelham, 1986; Miller, Lee, Raina, Klases, Zupancic, & Olsen, 1998). Contentions of improved functioning are also supported by the neuropsychological evidence. In comparison to unmedicated ADHD controls, children receiving stimulant treatment demonstrate enhanced performance on tests of executive functioning, (Kempton, Vance, Maruff, Luk, Costin, & Pantelis, 1999) and attentional processes (Hood et al., 2005)

With stimulant medication being recommended as the first-line treatment in ADHD (NICE, 2000), and the dominant neuropsychological theory citing response inhibition as the cardinal feature in ADHD (Barkley, 1997), it is perhaps unsurprising that research has begun to look at the relationship between these factors.

1.2 The effects of medication on response inhibition

Broyd, Johnstone, Barry, Clarke, McCarthy, & Selikowitz (2005) used the Go/NoGo task, alongside a number of electrophysiological measures, to examine the effects of methylphenidate on response inhibition in children with ADHD. The ADHD group were found to make more overall errors (omission and commission) in the pre-medication condition. After receiving medication, the ADHD group still made more omission errors than controls, but no longer differed with regard to the number of commission errors. The authors interpreted this as evidence that methylphenidate does ameliorate deficits in response inhibition and these behavioural findings were further substantiated by the electrophysiological findings of reduced skin conductance levels, overall supporting a hypoarousal model of ADHD (Broyd et al., 2005).

Scheres, Oosterlaan, Swanson, Morein-Zamir, & Schut, (2003) investigated the effect of methylphenidate and placebo on response inhibition in 23 boys with ADHD. Response inhibition was broken down into three components: inhibition of a prepotent response, inhibition of an ongoing response, and interference control. It was found that, compared to placebo, inhibitory control did improve with methylphenidate. This effect was, however, only significant for inhibition of a prepotent response and one of two measure of inhibition of an ongoing response. There was no significant effect of methylphenidate on interference control. This contrasts with the findings of an earlier study where, in comparison to pre-treatment baseline assessment, Stroop performance was reported to have improved following

one year of methylphenidate treatment (Everett, Thomas, Cote, Levesque, & Michaud, 1991).

Overall then, the few studies in this area generally suggest that pharmacological techniques improve both the behavioural and neuropsychological performance of children with ADHD. There is, however, the need for further research which examines the direct effects of medication on response inhibition, particularly as the latter is now widely viewed as the cardinal feature of ADHD (Barkley, 1997).

1.3 Research aims

The aim of this study is to examine whether methylphenidate is effective in improving response inhibition in children with ADHD, using a using the Animal Stroop Task (Wright, Waterman, Prescott, & Murdoch-Eaton, 2003). It was hypothesised that:

1. Children with ADHD will show more impaired response inhibition than control participants;
2. Following their prescribed methylphenidate dose, children with ADHD will show an improvement in response inhibition.

2. Method

2.1 Design

A between subjects design was used to investigate the performance of children with ADHD and a control group on a measure of response inhibition. Additional within subjects comparisons were made in the group of participants with ADHD to compare response inhibition in the ‘medicated’ vs. ‘unmedicated’ condition.

2.2 Power Calculation

A large effect size was posited from previous research (e.g. Pennington & Ozonoff, 1996; Scheres et al., 2004). Based on Cohen’s (1992) estimate of sample size (setting power at 0.8 and alpha at 0.05) one-tailed between subjects tests of difference would require that $N=20$, and one-tailed within subjects tests of difference would require that $N=12$.

2.3 Participants

Two groups participated in this study, an experimental group of individuals with ADHD and a control group of non-ADHD individuals, matched for age, gender and IQ. The age of participants with ADHD ranged from 6-14 (mean CA = 9.3, SD = 2.6). A total of 16 boys and 5 girls participated. Estimated Full-Scale IQ (FSIQ) scores ranged from 62 - 144 (mean FSIQ = 94.3, SD = 18.2).

Participants in the control group were 20 boys and 5 girls, aged between 6 – 13 years (mean CA = 9.2, SD = 2.0). Estimated Full-Scale IQ scores ranged from 71 - 132 (mean FSIQ = 102.5, SD = 17.0). Independent samples t-tests, found no significant differences between the two groups in relation to age ($t(45)=-.020$, $p=0.84$) or IQ ($t(45)=1.58$, $p=0.12$). A chi-square illustrated no significant difference between the distribution of males and females in the two groups ($X^2=0.10$, $p=0.78$).

2.4 Inclusion and exclusion criteria

Children had to be aged between 5-16 years to be included in the study. Children in the experimental condition had to have a primary diagnosis of ADHD and be receiving treatment with psychostimulant medication (those receiving non-methylphenidate based drugs, e.g. Atomoxetine were excluded). Children in the control group had no diagnosis of ADHD. Children in both groups were excluded if they had a diagnosis of any other significant mental health condition.

The children in the experimental group were current patients of a rural Child and Adolescent Mental Health Service (CAMHS) In Scotland. They had been given a diagnosis of ADHD prior to taking part in the study and all were currently being treated with methylphenidate based psychostimulant medication. Diagnostic assessment was carried out by a multi-disciplinary team in accordance with Scottish Intercollegiate Guidance Network recommendations for assessment for ADHD (SIGN, 2001). The methylphenidate dosage received by each participant varied dependent on their individual requirements. The length of time that participants had

been receiving psychostimulant medication varied from between 4 and 84 months (mean = 17.8, SD = 17.3).

Participants in the control group were recruited from a variety of local schools. None of this group had a diagnosis of ADHD or had had previous contact with the Child and Adolescent Mental Health Service.

2.5 Description and Application of Measures

2.5.1 Estimated Full Scale IQ

Full-scale IQ was estimated using a shortened version of the Wechsler Intelligence Scale for Children, UK (WISC-III UK, Wechsler, 1991), comprised of one verbal subtest (Vocabulary) and one performance subtest (Block Design). The reliability and validity of this short form is high ($r = 0.906$) (Sattler, 1992) and it is considered to be a good screening combination (Kilian & Hughes, 1978).

2.5.2 Response inhibition

Response inhibition was measured using the Animal Stroop Task (Wright et al., 2003). This is a relatively new, pictorial modification of the original Colour-Word Stroop Task (Stroop, 1935) and is based on four exemplar animal stimuli; a cow, a pig, a duck and a sheep. The task comprises three conditions. The first is an 'Incongruent Condition' where each of the animals' heads is substituted with a head from another of the three animal prototypes. Thus, within the incongruent condition,

there are 12 animal-stroop stimuli. The second condition is a 'Matching Condition' where each of the four animal prototypes is displayed as a whole animal - i.e. the animal's body is coupled with the appropriate, matching head. Lastly, the task includes a 'Control Condition' where the animal's head is replaced by a caricature of a face.

The Animal Stroop Task is based on the premise that facial information is preferentially processed (Johnson, 1993) and utilized preferentially in semantic categorization (Quinn & Eimas, 1996). Thus, in both the incongruent and control conditions, stroop-like interference can be induced by asking the child to name the animal's body and inhibit a preferred response to identify the head (Wright et al., 2003). The control task is intended to act as a semantic control in that it contains similar semantic content as a face, but produces less activation of animal representations. As such, it is believed to serve as the most appropriate comparison with the incongruent task (Wright et al., 2003).

The Animal Stroop comprises three blocks, with twenty four images in each. The first and third blocks consist of a mixture of incongruent and control images – twelve of each within a block. The second block contains 'matching' images only. In blocks one and three, children are required to name the animal's body whereas, in block two, they are asked to simply name the animal. The difference between reaction times in the incongruent and control conditions (i.e. 'Reaction Time Difference') is used as a measure of inhibitory control (Wright et al., 2003). Thus, blocks one and three are used to provide a measure of response inhibition. The images are presented on a computer screen and displayed in a random order.

The present study utilized the procedure outlined above. Before commencing the Animal Stroop Task, participants were shown flashcards of each of the four animals and asked to name them in order to ensure correct identification. Following this, participants were given verbal task instructions and completed a series of 'warm-up' trials. An example image was presented at the start of each block whereupon the task instructions were repeated. Participants were required to identify the example image correctly before proceeding with the test.

The accuracy of children's naming was recorded by the experimenter. Reaction times were recorded by 'voice key', elicited by the participants' vocal response, with the first author operating a manual timing procedure as back-up.

2.5.3 Strengths and Difficulties Questionnaire (SDQ)

The Strengths and Difficulties Questionnaire (SDQ) (Goodman, 1997) was used to assess levels of hyperactivity in control group participants. The reliability and validity of the SDQ are relatively good and, as such, it is considered as a useful, brief measure of the adjustment and psychopathology of children and adolescents (Goodman, 2001). Questionnaires were completed for each participant in the control group - either by their parent(s) or classteacher. All participants in the control group scored within the 'normal' range with regard to 'hyperactivity/inattention'.

2.6 Procedure

All participants were tested on a one-to-one basis by the first author. Each participant in the experimental group was assessed under a ‘medicated’ condition where they had taken their prescribed stimulant medication as usual and an ‘unmedicated’ condition where they were assessed following an unmedicated period of at least 12 hours. This time period was chosen in consultation with the Consultant Psychiatrist attached to the CAMHS and ADHD Service. This was on the basis that missing a single dosage of routinely prescribed medication was unlikely to have any significant adverse effects as, due to the relatively short half-life of stimulant medications, children prescribed these treatments often experience a dip in medication level during some points of the day (Pelham, Gnagy, Burrows-Maclean, Williams, Fabiano, & Morrisey, 2001). The 12 hour time period was also considered sufficient for slower release methylphenidate preparations, such as Concerta, which are designed to last 12 hours.

Equal numbers of participants were allocated to the ‘medicated’ or ‘unmedicated’ conditions initially and the condition-type was reversed for their second assessment session. The period of time between assessment sessions varied for each participant and ranged from between four and fourteen days (mean = 6.8, SD = 2.0). Participants in the control group were assessed on only one occasion.

3. Results

Data which were not normally distributed was transformed using a natural logarithm ($x+1$) transformation. The data were subsequently analysed using parametric tests. The significance level of test results, unless otherwise stated, was set at $p=0.05$ (one-tailed).

3.1 Exploratory Data Analysis

Two participants were removed from each of the ADHD group (medicated condition) and the Control group due to outlier scores on the inhibitory control measure of the Animal Stroop. There were no outliers in the ADHD-unmedicated condition.

3.2 Test order effects

An ANOVA was used to examine whether there was any effect of the order in which the experimental participants were tested (i.e. whether they were in the medicated followed by unmedicated condition or vice versa). The ANOVA showed there was no interaction between order and group ($F = 0.83$, $p = 0.37$, $df = 1$) on Reaction Time Difference scores. The 'order' variable was therefore removed from further analysis.

3.3 Hypothesis 1

Independent Samples t-tests showed no significant differences in Reaction Time Difference Scores between either the ADHD-medicated and control groups ($t(44) = -0.98, p = 0.17$) or between the ADHD-unmedicated and control groups ($t(40) = 1.56, p = 0.06$). Table 1 illustrates the mean reaction time difference scores.

INSERT TABLE 1 ABOUT HERE

3.4 Hypothesis 2

A Paired-Samples t-test showed a significant difference between the reaction time difference scores of participants with ADHD-medicated and participants with ADHD-unmedicated ($t(18) = 2.28, p = 0.02$). ADHD participants showed significantly higher reaction time difference scores in the unmedicated condition compared to the medicated condition (see table 1).

4. Discussion

The present study examined the impact of methylphenidate on response inhibition in children with ADHD, using the Animal Stroop Task (Wright et al., 2003). No significant differences in Reaction Time Difference Scores were found between either the ADHD-medicated and control group or between the ADHD-unmedicated and control group, although the latter was approaching significance. This indicates that children with ADHD (irrespective of whether they are being treated with methylphenidate medication) do not differ from children without ADHD with regard to their level of inhibitory control, as measured by the Animal Stroop Task.

This result is inconsistent with the now wide-spread theory of response inhibition as the central deficit of ADHD (Barkley, 1997) and with previous research which contends that, in comparison to controls, children with ADHD have significant impairments in inhibitory responses across go/no-go tasks (e.g. Booth et al., 2005), the continuous performance task (Halperin et al., 1990), the stop signal task (Schachar & Logan, 1990) and Stroop interference tasks (e.g. Houghton et al., 1999). Many of these tasks, however, comprise pure measures of motor inhibition and may fail to tap into inhibitory control more broadly e.g. across a cognitive and behavioural level (Wright et al., 2003). By contrast, the Stroop tasks rely on vocal responses which may make higher demands on inhibitory control in general. Some studies using the colour-word version of the Stroop task often fail to find impairments in response inhibition in children with ADHD (e.g. Kerns, McInerney, & Wilde, 2001; vanMourik, Oosterlaan, & Sergeant, 2005). Such results could be

interpreted as evidence that, once the motor component of response inhibition is removed, children with ADHD do not display any deficit in inhibitory control. Other researchers have, however, demonstrated such deficits by using the same Stroop task (e.g. Houghton, Douglas, West, Whiting, Wall, Langsford et al., 1999; Pennington & Ozonoff, 1996), although, given the high rates of co-morbidity between ADHD and reading disorder (August & Garfinkel, 1990), the fact that many of these studies fail to control for reading ability has been put forward as a possible explanation for these conflicting results (vanMourik et al., 2005). However, measures such as the Animal Stroop task, which use pictorial images rather than words, should be devoid of such shortcomings.

As such, the failure of the current study to find an inhibitory control deficit in children with ADHD may be more representative of Kerns et al.'s (2001) contentions that different tasks may tap different components of inhibition and that not all levels of inhibition are necessarily impaired in children with ADHD (Kerns et al., 2001). Thus it is possible that inhibitory control, as measured by the Animal Stroop, might not be affected by ADHD. It may then have been useful for this study to have employed more than one measure of inhibitory control in order to assess the potential for different components of inhibition more thoroughly.

Alternatively, these results may have relevance for the 'delay aversion' hypothesis put forward by Sonuga-Barke, Williams, Hall, & Saxton, (1996). This argues that the cognitive deficits thought to be shown by children with ADHD could actually be more motivational in nature, i.e. children with ADHD are averse to delay. Sonuga-Barke et al. (1996) highlighted that studies measuring inhibitory control are

often effected by trial constraints (i.e. as soon as one trial ended another began) and, therefore, are confounded with delay. When these experiments have been repeated in such a manner that early or impulsive responses have no influence on delay, the responses from children with ADHD are comparable with those of controls (Sonuga-Barke et al., 1996). The trials in the Animal Stroop Task are of equal length, irrespective of participant's speed of responding. Thus, the current lack of discrepancy between the inhibitory control of children with ADHD and children in the control group may be due to the fact that, as participants could not opt to minimise the delay by acting more impulsively, there was little motivational incentive for children with ADHD to respond more rapidly.

Nonetheless, these explanations fail to account for the initial findings of Wright et al. (2003) who, with reference to the behavioural data of a large sample of children aged between 3 and 16 years, suggested that the Animal Stroop appeared to identify those at risk of hyperactive symptomatology (Wright et al., 2003). It should be noted, however, that Wright et al's (2003) findings were based on data gathered from a *school-based* sample and only utilised behavioural data gathered from teachers rather than the cross-situational information required in considering a diagnosis of ADHD. Thus, the contrast in findings with those of the current study may suggest that using the Animal Stroop task as a measure of inhibitory control is not sufficient in differentiating between children with a *clinical diagnosis* of ADHD and normally developing children.

Despite this, children with ADHD showed significant improvements in inhibitory control after receiving their methylphenidate medication, supporting previous reports of methylphenidate ameliorating deficits in response inhibition (Broyd et al., 2005; Everett et al., 1991), classroom and social behaviour of children with ADHD (Miller et al., 1998; Pelham et al., 1998), performance on attentional measures (Hood et al., 2005) and various other executive functioning tasks (Kempton et al., 1999). Horrobin, McNair, Kirk, & Waldie (2007) suggest a mechanism for this. Their case study of an adult with ADHD suggested that dexamphetamine improved interference control on a Stroop task. This was indicated by the fact that, when on medication, the individual's event-related potentials (as measured by electroencephalography) for the incongruent condition closely resembled those of the control participant, with a concurrent improvement in reaction times and accuracy.

Scheres et al. (2003), however, found that while methylphenidate produced significant improvements in the inhibition of a prepotent response and partial improvements in the inhibition of an on-going response, there was no significant effect of methylphenidate on interference control (as measured by both the Stroop Colour-Word Test and the Eriksen Flanker Task).

The differing results may have been due to Scheres et al. (2003) not directly controlling for reading ability or to the differing levels of methylphenidate received by the children in each study. Scheres et al. (2003) examined the effects of methylphenidate dose specifically up to a 20mg dose and found no effects of medication dosage on any of the response inhibition measures. Many of the children with ADHD in the current study were, however, receiving doses of methylphenidate-

based medication that exceeded a 20mg dose. Indeed, other studies have reported an optimal response to medium or high doses of methylphenidate in cognitive tasks (e.g., Douglas, Barr, Amin, O'Neill, & Britton, 1988), yet Tannock, Schachar, and Logan, (1995) found that inhibitory performance declined in their high dose condition compared to the medium dose condition. In contrast, a study by Rapport and Kelly (1991) which examined the effects of low and high doses of methylphenidate on cognitive tasks reported there was no evidence to support an optimal lower dose. Due to the different methylphenidate-based prescriptions received by the children with ADHD in the current study (e.g. long- vs. short-acting preparations), along with the various timings at which these were taken, it was not possible to accurately control for the effects of methylphenidate dosage. Thus, it is not possible to ascertain whether the dosage level of methylphenidate had an effect on the inhibitory control of participants with ADHD.

It may also be of note that all participants within the experimental group of the current study had been receiving medication for a minimum period of 4 months. This is in contrast to some of the previous research in which participants are naïve to medication and subsequently experience a period of titration before the study commences. Although these titration periods are relatively extensive (e.g. four weeks in the case of Scheres et al., 2003), it is possible that the length of time participants have been receiving medication has an effect on any subsequently experienced improvements in response inhibition. Indeed, while Scheres et al. (2003) found no improvement in response inhibition following methylphenidate administration, others have reported significant improvements in the inhibitory control (as measured by the Stroop Colour-Word Test) of hyperactive children

following a year of treatment with methylphenidate (Everett et al., 1991). Again, due to the wide range in the length of time that participants had been receiving psychostimulant medication (i.e. between 4 and 84 months) and changes in medication dose and type that had occurred within this time, alongside the different doses and types of medication prescribed presently, it was not possible to accurately control for the length of time on medication in the current study.

4.1 Methodological Considerations

As was noted one limitation of the current study was the inability to control for type, dosage and length of time on medication. The study had a number of additional limitations. In terms of measures, the Animal Stroop had the advantages of being a reliable and valid measure of inhibitory control (Wright et al., 2003) and, being pictorial-based, it did not require a certain level of reading ability in order to yield reliable scores. Wright et al. (2003), however, warn that the Animal Stroop may be a less sensitive measure of impulse control in older children. It is possible that ceiling effects amongst the older participants in the present study may be masking a difference between the inhibitory control of participants with and without ADHD.

In addition, while the hyperactivity/inattention questions of the SDQ were used with the control group, this only provides a broad screening. While none of the children in this group were identified by teachers as having behaviours indicative of ADHD or indeed were known to CAMH services, failure to carry out diagnostic

assessments means the possibility that some of the children in the control group had ADHD can't be ruled out.

Another limitation in relation to participant diagnosis is that ADHD encompasses three subtypes – ‘inattentive’, ‘hyperactive-impulsive’, and ‘combined’ ADHD (Rappley, 2005). While all participants in the ADHD group had been given the diagnosis by a multi-disciplinary team, they had not been classified as a particular subtype and, as such, the current study did not take into account the potential influence of different subtypes on performance. As the ADHD group did not complete the SDQ, a broad subtype classification could not even be made on this basis. Nonetheless, classifying participants with ADHD into the different subtypes can be problematic and not always easy to define. For instance, children in either the inattentive or hyperactive-impulsive subtype may be just one symptom below the threshold for the combined subtype, thus specific subtypes may be contaminated by contrasting features of another subtype (vanMourik et al., 2005). Finally, there were limitations in terms of sample size. While the required number of participants were recruited to achieve statistical power overall, two participants were removed from each of the ADHD group (medicated condition) and the Control group due to outlier scores on the inhibitory control measure of the Animal Stroop. While these scores needed to be removed in order for the groups to be matched on the inhibitory control task, it meant that in analyses examining inhibitory control, the number of participants within the medicated condition of the experimental group was one short of that required to reach statistical power.

4.2 Clinical Implications

The current study highlights the need to monitor the effects of medication treatments on response inhibition. Whether medication is improving a child's ability to control their inhibition is an important area as such improvements may aid them in other areas of development, such as attentional ability, social skills etc. (Tannock, Schachar, Carr, Chajczyk, & Logan, 1989). Conversely, monitoring the symptoms of ADHD is also important when we consider the abundance of negative consequences that can develop from the disorder, such as conduct disorder (Taylor, Chadwick, Heptinstall, & Danckaerts, 1996). In clinical practice, the effects of medication on children with ADHD are monitored in relation to their effects on observed behaviour. These observations, however, can be fairly subjective and may not pick up improvements in response inhibition. Neuropsychological tests, such as the Animal Stroop (Wright et al., 2003), may provide another means of monitoring the potential effects of medication on response inhibition. However, the lack of significant difference between the inhibitory control of children with ADHD (irrespective of medication status) and children in the control group found in the present study suggests that further research is required into the Animal Stroop Task as a potential screening measure to identify children at risk of hyperactive symptomatology.

5. Conclusion

The present research used the Animal Stroop Task (Wright et al., 2003) to examine whether methylphenidate-based medication is effective in improving inhibitory control in children with ADHD. The study found that methylphenidate improved the inhibitory control of children with ADHD, however, no significant differences between the inhibitory control of children with ADHD (irrespective of medication status) and children in the control group were found. This result contrasts with a considerable amount of evidence documenting deficits in the inhibitory control of children with ADHD, however, may provide support for contentions that different tasks tap different components of inhibition and that not all levels of inhibition are necessarily impaired in children with ADHD (Kerns et al., 2001). Alternatively, it may offer support for the ‘delay aversion’ hypothesis in which cognitive deficits in children with ADHD are thought to be more motivational in nature (Sonuga-Barke et al., 1996).

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References

- August, G.J., & Garfinkel, B.D. (1990). Comorbidity of ADHD and reading disability among clinic-referred children. *Journal of Abnormal Child Psychology*, 18, 29 - 45.
- Barkley, R.A. (1990). *Attention deficit hyperactivity disorder: A handbook for diagnosis and treatment*. New York: Guilford Press.
- Barkley, R.A. (1997). Behavioural inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD. *Psychological Bulletin*, 12 (1), 65 – 94.
- Barkley, R.A. (2000). Genetics of Childhood Disorders: XVII. ADHD, Part 1: The Executive Functions and ADHD. *American Academy of Child and Adolescent Psychiatry*, 39 (8), 1064-1068.
- Booth, J.R., Burman, D.D., Meyer, J.R., Lei, Z., Tronmer, B.L., Davenport, N.D. et al. (2005). Larger deficits in brain networks for response inhibition than for visual selective attention in attention deficit hyperactivity disorder. *Journal of Child Psychology and Psychiatry*, 46 (1), 94 – 111.

Brown, R.T., & Levers, C.E. (1999). Psychotherapy and pharmacotherapy treatment outcome research in pediatric populations. *Journal of Clinical Psychology in Medical Settings*, 6, 63 – 88.

Broyd, S.J., Johnstone, S.J., Barry, R.J., Clarke, A.R., McCarthy, R., Selikowitz, M. et al. (2005). The effect of methylphenidate on response inhibition and the event-related potential of children with attention deficit/hyperactivity disorder. *International Journal of Psychophysiology*, 58 (1), 47 – 58.

Cohen, J. (1992). A power primer. *Psychological Bulletin*, 112(1), 155-159.

Douglas, V. I., Barr, R. G., Amin, K., O'Neill, M. E., & Britton, B. G. (1988). Dosage effects and individual responsivity to methylphenidate in attention deficit disorder. *Journal of Child Psychology and Psychiatry*, 29, 453-475.

Everett, J., Thomas, J., Cote, F., Levesque, J., & Michaud, D. (1991). Cognitive effects of psychostimulant medication in hyperactive children. *Child Psychiatry and Human Development*, 22, 79– 87.

Goldman, L.S., Genel, M., Bezman, R.J., & Slanetz, P.J. (1998). Diagnosis and treatment of attention-deficit/hyperactivity disorder in children and adolescents. *Journal of the American Medical Association*, 279, 1100 – 1107.

Goodman, R. (1997). The Strengths and Difficulties Questionnaire: A research note. *Journal of Child Psychology, Psychiatry, and Allied Disciplines*, 38 (5), 581-586.

Goodman, R. (2001). Psychometric properties of the strengths and difficulties questionnaire. *Journal of the American Academy of Child and Adolescent Psychiatry* 40 (11), 1337 – 1345.

Halperin, J.M., Newcorn, J.H., Sharma, V., Healey, J.M., Wolf, L.E., Pascualvaca, D.M. et al. (1990). Inattentive and non-inattentive ADHD children: Do they constitute a unitary group? *Journal of Abnormal Child Psychology*, 18, 437-449

Hood, J., Baird, G., Rankin, P.M., & Isaacs, E. (2005). Immediate effects of methylphenidate on cognitive attention skills of children with attention-deficit-hyperactivity disorder. *Developmental Medicine and Child Neurology*, 47 (6), 408 – 415.

- Horrobin, S. L. , McNair, N. A. , Kirk, I. J., & Waldie, K. E. (2007). Dexamphetamine normalises electrophysiological activity in Attention Deficit-Hyperactivity Disorder during the Stroop Task, *Neurocase*, 13(5), 301- 310
- Houghton, S., Douglas, G., West, J., Whiting, K., Wall. M., Langsford, S. et al. (1999). Differential patterns of executive function in children with attention-deficit hyperactivity disorder according to gender and subtype. *Journal of Child Neurology*, 14, 801–805.
- Johnson, M.H. (1993). Cortical maturation and the development of visual attention in early Infancy. In M.H. Johnson (Ed.), *Brain Development and Cognition: A Reader* (pp. 167–193). Oxford: Blackwell.
- Johnston C. (1998). The impact of attention-deficit/hyperactivity disorder on social and vocational functioning in adults. *Program and Abstracts of NIH Consensus Development Conference on Diagnosis and Treatment of ADHD; September 17-19, 1998; Los Angeles, California.*
- Kempton, S., Vance, A., Maruff, P., Luk, E., Costin, J., & Pantelis, C. (1999). Executive function and attention deficit hyperactivity disorder: stimulant medication and better executive function performance in children. *Psychological Medicine*, 29 (3), 527-538.

- Kerns, K.A., McInerney, R.J., & Wilde, N.J. (2001). Time reproduction, working memory and behavioural inhibition in children with ADHD. *Child Neuropsychology* 7(1), 21-31.
- Kilian, J.B. & Hughes L.C.A. (1978). A comparison of short forms of the Wechsler Intelligence Scale for Children—Revised in the screening of gifted referrals. *Gifted Child Q*, 22, 111–15.
- Lawrence, Y., Houghton, S., Tannock, R., & Douglas, G. (2002). ADHD outside the laboratory: boys' executive function performance on tasks in video game play and on a visit to the zoo. *Journal of Abnormal Psychology*, 30, 447 – 463.
- Miller, A., Lee, S., Raina, P., Klassen, A., Zupancic, J., & Olsen, L. (1998). A review of therapies for attention deficit/hyperactivity disorder. *Ottawa: Canadian Coordinating Office for Health Technology Assessment*.
- MTA Cooperative Group. (1999). Fourteen-month randomized clinical trial of treatment strategies for attention-deficit hyperactivity disorder. *Archives of General Psychiatry*, 56, 1073-1086.
- National Institute of Clinical Excellence (NICE) (2000). Guidance on the use of methylphenidate (Ritalin, Equasym) for Attention Deficit/Hyperactivity Disorder (AD/HD) in childhood. *Technology Appraisal Guidance No 13*.

- Owens, J.S. & Hoza, B. (2003). The role of inattention and hyperactivity/impulsivity in the positive illusory bias. *Journal of Consulting and Clinical Psychology, 71* (4), 680 – 691.
- Pelham, W.E. (1986). The effects of psychostimulant drugs on learning and academic achievement in children with attention-deficit disorders and learning disabilities. In J. K. Torgesen & B.Y.L. Wong (Eds.). *Psychological and Educational Perspectives on Learning Disabilities* (pp. 259 – 295). New York: Academic Press.
- Pelham, W. E., Gnagy, E. M., Burrows-Maclean, L., Williams, A. Fabiano, G.A., Morrisey, S.M. et al. (2001). Once-a-day concerta methylphenidate versus three-times-daily methylphenidate in laboratory and natural settings. *Pediatrics, 107*, e105-120.
- Pennington, B.F. & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry, 37* (1), 51 – 87.
- Quinn, P.C., & Eimas, P.D. (1996). Perceptual cues that permit categorical differentiation of animal species by infants. *Journal of Experimental Child Psychology, 63*, 189–211.
- Rappley, M.D. (2005). Attention deficit-hyperactivity disorder. *New England Journal of Medicine, 352* (2), 165 – 172.

Rappoport, M. D. & Kelly, K. L. (1991). Psychostimulant effects on learning and cognitive function: Findings and implications for children with Attention Deficit Hyperactivity Disorder. *Clinical Psychology Review, 11*, 61-92.

Rubia, K., Taylor, E., Smith, A.B., Oksannen, H., Overmeyer, S., & Newman, S. (2001). Neuropsychological analyses of impulsiveness in childhood hyperactivity. *The British Journal of Psychiatry, 179*, 138 – 143.

Sattler, J.M. (1992). *Assessment of Children (3rd Edition)*. San Diego, CA: Jerome M. Sattler.

Schachar, R. & Logan, G.D. (1990). Impulsivity and inhibitory control in normal development and childhood psychopathology. *Developmental Psychology, 26 (5)*, 710 – 720.

Scheres, A., Oosterlaan, J., Swanson, J., Morein-Zamir, S., Meiran, N., Schut, H. et al. (2003). The effect of methylphenidate on three forms of response inhibition in boys with AD/HD. *Journal of Abnormal Child Psychology, 31*, 105 – 120.

Scottish Intercollegiate Guidance Network (SIGN) (2001) *Attention Deficit and Hyperkinetic Disorders in Children and Young People*. Edinburgh:SIGN

- Seidman, L.J., Biederman, J., Faraone, S.V., Weber, W., & Ouellette, C. (1997). Toward defining a neuropsychology of attention deficit-hyperactivity disorder: Performance of children and adolescents from a large clinically referred sample. *Journal of Consulting & Clinical Psychology, 65* (1), 150 – 160.
- Sonuga-Barke, E.J.S., Williams, E., Hall, M., & Saxton, T. (1996). Hyperactivity and delay aversion III: The effect on cognitive style of imposing delay after errors. *Journal of Child Psychology and Psychiatry, 37*, 189 – 194.
- Stroop, J.R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18*, 643 – 662.
- Tannock, R., Schachar, R.J., Carr, R.P., Chajczyk, D., & Logan, G.D. (1989). Effects of methylphenidate on inhibitory control in hyperactive children. *Journal of Abnormal Child Psychology, 17*, 473 – 491.
- Tannock, R., Schachar, R., & Logan, G. (1995). Methylphenidate and cognitive flexibility: Dissociated dose effects in hyperactive children. *Journal of Abnormal Child Psychology, 23*, 235-266.

Taylor, E. (1999). Issues in the management of hyperkinetic children. *Progress in Neurology and Psychiatry, 3 (Supplement)*, 1 – 19.

Taylor, E., Chadwick, O., Heptinstall, E., & Danckaerts, M. (1996). Hyperactivity and conduct problems as risk factors for adolescent development. *Journal of the American Academy of Child and Adolescent Psychiatry, 35*, 1213 - 1226.

vanMourik, R., Oosterlaan, J., & Sergeant, J.A. (2005). The Stroop revisited: a meta-analysis of interference control in AD/HD. *Journal of Child Psychology and Psychiatry, 46(2)*, 150-165.

Volkow, N.D., Wang, G.J., Fowler, J.S., Logan, J., Gerasimov, M., Maynard, L. et al. (2001). Therapeutic doses of oral methylphenidate significantly increase extracellular dopamine in the human brain. *Journal of Neuroscience, 21*, 1-5.

Wechsler, D. (1991). *Wechsler Intelligence Scale for Children (3rd Edition)*. San Antonio, TX: The Psychological Corporation.

Weiss, M. & Murray, C. (2003). Assessment and management of attention deficit hyperactivity disorder in adults. *Canadian Medical Association Journal, 168 (6)*, 715 – 722.

Williams, B.R., Ponesse, J.S., Schachar, R.J., Logan, G.D., & Tannock, R. (1999). Development of inhibitory control. *Developmental Psychology, 35* (1), 205-213.

Wright, I., Waterman, M., Prescott, H., & Murdoch-Eaton, D. (2003). A new Stroop-like measure of inhibitory function development: typical developmental trends. *Journal of Child Psychology and Psychiatry, 44* (4), 561-575

Table 1: Means, Ranges and Standard Deviations for the experimental and control groups

	ADHD-Medicated (n=19)			ADHD- Unmedicated (n=21)			Control (n=23)		
	Mean	SD	Range	Mean	SD	Range	Mean	SD	Range
Reaction time difference	39.95	58.6	-66 to 141	103.86	103.02	-66 to 298	76.57	86.99	-80 to 262