Identity-Based Negative Priming: Individual Differences in Typical and Atypical Development

A dissertation

submitted in partial fulfilment

of the requirements for the degree

of

Doctor of Philosophy in Psychology

in the University of Canterbury

by

Verena E. Pritchard

2007

ACKNOWLEDGEMENTS

First and foremost I wish to acknowledge my supervisor, Dr Ewald Neumann, Senior Lecturer, Department of Psychology, University of Canterbury to whom I am indebted for an invaluable introduction to the negative priming paradigm. For more than three years his support for my research has never wavered. At all times he has willingly shared his expertise in the field of selective attention and has offered guidance and suggestions at all stages of my work. His critical evaluation of my theories has refined and stimulated my thinking, and he has never stinted in giving of his time whether to review drafts of chapters as they appeared or discuss test procedures and findings. For this, I express my deep gratitude.

Next, I acknowledge those school children that participated in my tests for negative priming. Much of this dissertation has been built on their co-operation and that of their parents. To the Principal (Ms Sue Ashworth), whose assistance and co-operation has been indispensable, and teachers of Paparoa Street Primary School who welcomed me and facilitated my contact with their pupils, I offer my sincere thanks.

I also wish to express my gratitude to Dr. Julia Rucklidge, Department of Psychology, University of Canterbury for allowing me access to her clinical samples during the course of this research. I am also grateful to the University of Canterbury for the award of a Doctoral Scholarship, and to the Department of Psychology for its contribution towards research expenses.

To the many who have supported, encouraged and assisted me in various ways and at various times while I have been preoccupied in this study, I also offer my thanks. But especially I wish to thank my parents for their never-failing concern, support and interest throughout the long process of producing this dissertation.

Verena Pritchard
Christchurch
April, 2007

TABLE OF CONTENTS

Section 2

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

One means by which inhibitory control in selective attention may be studied is with the negative priming (NP) procedure. It is widely assumed that children are characterised by reduced capacity for inhibition (Diamond, 2002) and that inhibitory dysfunction is a key characteristic of children and adolescents with ADHD (Barkley, 1997). This should translate into reduced NP effects for these populations.

In this dissertation, four studies using the NP procedure find no evidence for reduced inhibitory function in typical children or in adolescents with ADHD. Study 1 examined the magnitude of NP in children compared with adults. An important line of support for the idea that children suffer an inhibitory decrement has been based an empirical report suggesting that conceptual (identity or semantic) NP effects, assumed to reflect the by-product of distractor inhibition, while consistently found in adults are lacking in children (Tipper, Bourque, Anderson, & Brehaut, 1989). In Study 1, the opposite result was found. Study 2 compared NP effects between 7-year-old children and adults while replicating the respective methodologies of the only two studies to explore conceptual NP effects in developmental populations to date (Pritchard & Neumann, 2004, vs. Tipper et al., 1989) to determine the nature of the divergent results between these studies. In Study 2, it was found that distractor inhibition effects are comparable between children and adults when a NP task contains trials in which the distractor stimulus is consistently incongruent with the target stimulus, but that children may be more susceptible than adults to divide attention between target and distractor when a NP task contains a number of trials in which target selection difficulty is reduced. These are critical new findings, highlighting that reduced NP may often relate to methodological artifacts, and when considered in the light of current theories of NP, are also problematic for anti-inhibitory

accounts of NP. Having distinguished more definitively the role of inhibition in developmental NP effects, Studies 3 and 4 explored whether the inhibitory process underpinning NP was implicated in young persons with ADHD. To date, evidence for NP in ADHD populations is equivocal. Study 3 found no evidence for a reduced NP effect in ADHD devoid of a corresponding diagnosis. Study 4 found that conduct and oppositional defiant disorders had the potential to confound the evaluation of NP in ADHD.

Taken together, results in Studies 1 - 4 parallel very recent results in the literature on NP in older adults and adult psychopathology where presumed reductions of NP in these populations may also be accounted for by methodological artifacts (Buchner & Mayr, in press). It is concluded that NP may reflect a primitive and robust form of inhibitory processing, one that develops early and one that is often the last to deteriorate.

CHAPTER 1

Preview

Inhibitory control in selective attention is a common focus in studies of cognitive development and developmental psychopathology. One means by which inhibitory control processes are studied in attention is with the negative priming (NP) procedure (Tipper, 1985). To date, the status of NP in developmental populations is clouded by empirical uncertainty. Data on cognitively defined inhibitory control processes in attention are sorely needed in developmental and clinical research (Huang-Pollock & Nigg, 2003; Nigg, 2000). The aim of this dissertation is to address issues raised by studies investigating inhibitory-based NP effects in typical and atypical development. By identifying more precisely the psychological determinants of NP phenomena, this dissertation furthers our understanding as to the nature of the inhibitory component of the selective attention process in typically and atypically developing populations.

1.1 A brief overview of the NP phenomenon and consideration of contemporary theory

Identity-based NP is the demonstration of slowed response to a target stimulus on a probe trial when that stimulus or close categorical relation was ignored as a distractor on a preceding prime trial (i.e., the ignored repetition [IR] condition). The effect is gauged by comparing response times in the IR condition with those in a control condition where probe target and preceding prime distractor are unrelated across trials. Since the NP effect was first reported (e.g., Dalrymple-Alford & Budyar, 1966; Tipper, 1985) three principle explanations have been proposed to account for it, all based on a representation of the stimulus event itself: distractor inhibition theory (Houghton & Tipper, 1994; Tipper, 1985; Tipper, 2001); episodic memory retrieval theory (Neill & Valdes, 1992); and temporal discrimination theory (Milliken, Joordens, Merikle, & Seiffert, 1998).

The distractor inhibition theory proposed by Tipper and colleagues contends that NP may reflect a critical inhibitory control component of selective attention. This theoretical framework incorporates the notion that the internal categorical representations for target and distractor stimuli are activated in parallel at initial exposure prior to selection (Neumann & DeSchepper, 1991; Tipper, 1985). Thus the presence of a distractor stimulus in the prime trial will produce interference in the form of attentional and response competition (Houghton & Mari-Beffa, 2005). For successful selective attention and goal-directed response to be achieved, an excitatory process acts to enhance target information while a co-existing inhibitory process operates to suppress non-target information. This process helps to coordinate integration between parallel perceptual processes and goal-directed response schema. However, as reflected by the NP effect, this process incurs a small after-effect. That is, if a stimulus was successfully inhibited as a distractor in the IR prime trial there will be a temporary reduction in the activation state for the internal mental representation associated with this stimulus. By this account, delayed response to a probe target in the IR condition is the consequence of residual carry-over inhibition.

A key aspect of this theory is that the inhibition of interfering or non-target information is “reactive” or activation sensitive. That is, the more activated or interfering the non-target information, the more strongly it is suppressed by way of an inhibitory feedback system (Houghton & Tipper, 1994; Houghton, Tipper, Weaver, & Shore, 1996). Therefore, the distractor inhibitory process may only be implemented, and the NP effect occur, if the activated representations of non-target items are associated with current behavioural goals and/or are likely to disrupt correct responding. The inhibitory process underlying the NP effect may take the form of active neural activity operating at various loci in the stream of information processing. In recent behavioural and brain-imaging studies, NP is widely interpreted as indicating that irrelevant information activated with concurrent target information is subject to an involuntary form of neural inhibitory activity to aid target selection and response (Grison, Tipper, & Hewitt, 2005; Vuilleumier, Schwartz, Duhoux, Dolan, & Driver, 2005).

The episodic memory retrieval theory proposed by Neill and Valdes (1992) denies any role for an inhibitory selective attention process and contends instead that the probe target stimulus operates as a memory retrieval cue. By this account, NP reflects the consequence relating to the retrieval of a memory trace containing specific prior response information incompatible with current correct response. Thus the delayed response in the IR condition (in which a prime distractor becomes a probe target) is attributed to the eliciting of an episodic representation which contains prime response information (a “do not respond” tag) that conflicts with and impairs the opposing response required by the probe (i.e., “respond”). The time taken to resolve this conflict between incompatible response tags produces the NP effect. Advocates of this theory argue that the retrieval of prime response information is contingent on a match between processing information present at the time of the probe and that present at the time of prime (e.g., Neill, 1997). Without this match, probe trial information is less likely to cue the retrieval of prime trial information and the NP effect less likely to eventuate. In short, the successful retrieval of the stimulus associated with a “do-not-respond” prime trace is a necessary pre-condition for NP effects to manifest.

The temporal discrimination theory of NP proposed by Milliken et al. (1998) holds that NP relates to the cognitive differentiation between events that occur at different points in time. This theory contends that NP is produced at the instance of response formation during the IR probe trial. Thus during initial exposure the current perceptual contents (i.e., in the probe trial) are classified as either “old” or “new”. According to this theory, if a probe target is classified as “old”, response is facilitated as there is an immediate integration and retrieval of an episodic record associated with that stimulus. If a probe target is coded as “new” response is less rapid, as performance is generated only on the basis of perceptual analysis. Within this framework, delayed response in the IR probe trial is the consequence of a partial overlap between “old” and “new” categorizations. That is, a probe target in an IR trial is ambiguous by classification because its prior appearance as a distractor in the IR prime trial renders it a faintly familiar stimulus, which prevents a classification of “new”, and yet as a stimulus recently unattended it is not sufficiently familiar to be classified as “old”. The resolution of this ambiguity is believed to cause the NP effect.

Of these three accounts of NP, only the distractor inhibition and the episodic retrieval theories of NP are considered as having survived empirical testing (see Mayr & Buchner, in press). Arguably, the inhibition-based account remains the most influential account of NP. For the sake of clarity, and because some of the severe challenges to anti-inhibitory versions of the episodic retrieval theory (see Pritchard & Neumann, 2004, for further review on this topic), the interpretation of NP in this dissertation is in terms of an inhibition account. However, the dissertation has some recourse to the episodic retrieval theory as the study of developmental differences in NP provides a unique opportunity to pit the predictions of inhibitory against memory-based accounts of NP.[1] It is worth noting that findings in this dissertation create a further set of challenges to the episodic memory retrieval theory and augment the growing body of research that questions the exclusion of inhibition in NP accounts (e.g., Grison et al. 2005; Tipper, 2001).

1.2 Overview of this dissertation

This dissertation comprises four self-contained and stand-alone studies, each with its own literature review, experiments, findings and conclusions. These studies are presented in two sections. In the first section, two studies (Chapters 2 and 3) contribute to the evolving literature on NP effects in typically developing children. The first study (Chapter 2) explores the developmental trajectory of NP across five distinct developmental age groups spanning 5- to 25-years in age. The second study (Chapter 3) attempts to reconcile divergent findings regarding the manifestation of NP in children as reported in respective studies by Pritchard and Neumann (2004) and Tipper, Bourque, Anderson, and Brehaut (1989). In the second section, two studies (Chapters 4 and 5) contribute to the clinical literature on ADHD. Although inhibitory dysfunction is a central focus in ADHD research, not only have there been few studies of NP in ADHD, but also the results of these studies have been equivocal. In an effort to clarify discrepancies in the literature, the third study (Chapter 4) compares NP effects between adolescents with and without ADHD. The fourth study (Chapter 5) takes this

[1] NP, inhibitory and episodic memory based theories are defined and discussed in more detail in each of the subsequent chapters of this dissertation. For more detailed discussion on recent theoretical views of NP phenomena the reader is referred to the review papers by Tipper (2001) and Mayr and Buchner (in press). More contemporary theory on NP favours integration of inhibition and memory retrieval accounts (see Grison et al., 2005). Because the temporal discrimination model of NP lacks convincing empirical support and faces mounting empirical counter evidence (see Frings & Wuhr, 2007), this theory will not be referenced further in this dissertation.

further through its consideration of the impact of comorbidity and subtype in ADHD on NP. A brief synopsis of Sections 1 and 2 follows.

Section 1

1.3 Two studies of NP in typical development: Chapters 2 and 3

Towards resolving discrepant findings in the developmental NP literature

Research Aim: To investigate developmental differences in NP effects and to evaluate the significance of this research for contemporary NP theory and for the clinical use of NP effects as an inhibitory index.

Until recently, it was widely assumed that NP effects, while consistently found in young adults, are not found in children. This assumption was formed on the basis of one study (consisting of three experiments) comparing NP effects between 7- to 8-year-old children and young adults (Tipper et al., 1989). NP effects in children have been readdressed only recently (Pritchard & Neumann, 2004). These authors obtained intact and similar NP effects between children aged 5- to 12-years. To date, while NP has come under close and extensive scrutiny in studies of adult cognition, research on developmental NP effects remains strikingly sparse. So far, no attempts have been made to determine whether NP is developmentally mediated or whether the inhibitory process underlying NP operates in children in a manner comparable to that in young adults. In addition, there has been no formal empirical test of the hypothesis Pritchard and Neumann (2004) put forward to account for developmental differences in NP. Chapters 2 and 3 of this dissertation address these issues.

Contemporary debate surrounding the differing theoretical accounts of NP has the potential to complicate interpretation of NP effects in studies of cognition in typical and atypical developmental populations. Therefore, a second goal in Section 1 has been to evaluate the consequences the outcomes of these two studies may have on inhibitory and episodic memory-based approaches to NP. This evaluation is important for two reasons. First, because most, if not all, theoretical conclusions negating the role of inhibition in NP have been formulated on the basis of research involving adults, and second, distinguishing more definitively the process underpinning children's NP effects may allow for a more accurate interpretation of NP effects obtained for the young clinical samples in Section 2 of this dissertation.[2]

Section 2

1.4 Two studies of NP in atypical development: Chapters 4 and 5

Is there evidence for NP in adolescents with a formal diagnosis of ADHD?

Research Aim: To clarify discrepant findings concerning NP in children and adolescents with ADHD and to consider these outcomes for popular process models of ADHD.

[2] Outside the cognitive literature, and beyond purely academic issues, NP research may have important practical consequences. Although not a specific focus of this dissertation, a secondary goal was to evaluate potential outcomes of developmental differences in NP on inhibitory and memory-based approaches to NP for the broader purposes of clinical research. This was done to distinguish more definitively the role of inhibition in developmental NP phenomena. It is hoped that the theoretical evaluation of the results obtained in studies 1 and 2 a) help to establish NP as a demonstrably valid index for inhibitory function in child clinical samples, and b) allow for a more accurate interpretation of NP effects obtained for children with ADHD in experiments reported outside of this dissertation (e.g., Pritchard, Healey, & Neumann, 2006) and of NP effects obtained for adolescents with ADHD in studies 3 and 4 reported within this dissertation. In combination, studies 1-4 converge to make a unique and clear contribution to the clinical literature on ADHD.

Outside of the cognitive literature, NP is often used to study inhibitory function in adult psychopathology. The study of NP in child clinical samples is rare. Although the constructs of ‘disinhibition’ or defective inhibition are central to contemporary process models of ADHD (e.g., Barkley, 1997; Quay, 1997), clinical research on ADHD has invested surprisingly little effort into tracing the implications for basic cognitive inhibitory control processes. ADHD is currently one of the most commonly diagnosed child clinical syndromes, and is estimated to affect 3 - 7 % of children, with 50 - 80 % of these cases persisting into adolescence (Barkley, 1998). Of the four existing studies that used NP procedures to evaluate inhibitory function in children and adolescents with ADHD, two report diminished NP effects relative to controls (Marriott, 1998, unpublished doctoral dissertation; Ozonoff, Strayer, McMahon, & Filloux, 1998) while two report intact NP effects (Gaultney, Kipp, Weinstein, & McNeill, 1999; Pritchard, Healey, & Neumann, 2006).

However, these studies varied widely in methodology and sampling techniques. Further complicating assessment is the ubiquitous tendency of ADHD to coexist with other more common psychiatric disorders, and the changing phenotypic descriptions and diagnostic criteria for the disorder. Thus, it is scarcely surprising that the pattern of results varied across the studies of NP in ADHD samples. Because of the variety of discrepancies and confounds in this limited NP literature, further investigations seem warranted before the status of NP in ADHD can be stated with confidence. A goal of clinical research is to understand which basic psychological functions, such as inhibitory processes, may develop atypically in particular disorders (Nigg, 2000; Wakefield, 1992). As yet, no prior research on NP in ADHD has attempted to evaluate the extent to which NP may vary as a function of comorbidity and subtype. Therefore it seems essential to clarify the impact of these factors on the neuropsychological effects specific to ADHD. Chapters 4 and 5 address these issues.

1.5 Methodological Note

In this dissertation, the experimental methodology used is similar to that used in the original study investigating NP in children (Tipper et al., 1989) and re-employed in the only follow-up study to date (Pritchard & Neumann, 2004). This deliberate strategy ensured that the experiments in this dissertation would tap NP effects similar in nature to those in the above studies. Holding such procedural variables constant broadens possible future applications of the findings of this dissertation. This may be important for a research area that has attracted little attention to date and may further our understanding of developmental differences (or similarities) in NP effects.

SECTION 1

TWO STUDIES OF

IDENTITY-BASED NEGATIVE PRIMING IN TYPICAL DEVELOPMENT

CHAPTER 2

Study 1

Pitfalls of developmentally inappropriate negative priming tasks[3]

2.1 Abstract

Despite being ignored, unattended visual distractors often produce traceable priming effects, which can be used to investigate inhibitory functioning involved in selection. Negative priming (NP) effects are indexed behaviourally as the increased reaction-time (or reduced accuracy) that occurs in response to a previously ignored stimulus. NP tasks typically demonstrate robust NP effects in young adults but not in children. We report an exception to this pattern. Using two different NP tasks, we compared the performance of children (5- to 12-years), adolescents (13- to 17-years), and adults (19- to 25-years). One task obtained significant NP for all age groups. Surprisingly, the other produced significantly larger NP effects in children than in adolescents, while no NP effects were found for adults. These results suggest particular task situations may be more conducive to producing NP effects in some developmental populations than others. They also challenge the major opponent to the inhibition-based account of NP; the memory-based episodic retrieval theory.

---

[3] Paper submitted to and under revision for *Developmental Psychology*: Pritchard, V. E., & Neumann, E. (under revision). Pitfalls of developmentally inappropriate negative priming tasks. *Developmental Psychology*.

2.2 Introduction

Much of the empirical evidence on selective attention failures in children in the developmental literature is consistent with the widely accepted theory of a developmental deficit in the ability to inhibit task-irrelevant information (e.g., Dempster, 1993; Dempster & Corkill, 1999; Harnishfeger & Bjorklund, 1994; Kail, 2002; Wilson & Kipp, 1998). Two similar conceptions exist in the cognitive literature. One conception rests on the idea that developmental changes in the cognitive system permit age-related increases in information processing ability that align with more effective performance in tasks where response hinges on the resolution of conflict between competing stimulus items (e.g., Hitch & Towse, 1995; Salthouse & Babcock, 1991; Swanson, 1999; Verhaeghen & Salthouse, 1997). The other conception is based on empirical evidence, which suggests that negative priming (NP) effects, while consistently found in adults, are lacking in children (Tipper, Bourque, Anderson, & Brehaut, 1989). The main goal of this article is to provide further empirical assessment of the above three suppositions in light of recent findings in the developmental NP literature that suggest that even young children have an intact ability to inhibit irrelevant stimulus dimensions.

The Present Experiments

In a recent article (Pritchard & Neumann, 2004), we reported intact conceptual (identity and semantic) NP effects for children across a series of NP experiments using a range of stimulus types. Although a long and widely held view contends that children have a diminished inhibitory control mechanism for dealing with distractors in NP tasks (Tipper, et al., 1989), we instead showed that such selective inhibitory capacities are intact, even in children as young as five years old (Pritchard & Neumann, 2004; see also Bub, Masson, &

Lalonde, 2006). These findings suggested that NP effects in children may be comparable to those found for adults when a developmentally suited task design is implemented. In the current study we had three specific aims. First, we revisited the procedure used in Pritchard and Neumann (2004) in an attempt to establish whether the conceptual NP effects found for children do in fact map directly on to those found for young adults. Our second aim was to provide some insight into the developmental trajectory of the NP phenomenon. Thus, we compared NP effects across five distinct developmental populations; 5- to 6-year-olds vs. 8- to 9-year-olds vs. 11- to 12-year-olds vs. 13- to 17-year-olds vs. 19- to 25-year-olds. Finally, while there is general consensus in the NP literature that the processing of ignored information appears to reflect an important component of visual selection, one lasting controversy concerns whether the cognitive process underlying the NP effect is primarily inhibition- or memory-based.

Because we also wanted to determine the stability of any potential differences or similarities between the NP effects observed for children vs. adolescents vs. adults we assessed the effect over two tasks differing in levels of distractor pre-potency. To anticipate the outcomes, a unique dissociation was revealed. In Experiment 1 NP effects in children were found to be equivalent to those in adolescents and adults, while in Experiment 2 significant NP effects were found for children, but *not* for adults. The trends in the second experiment, in fact, pointed towards a systematic decline in the amount of the NP effect produced in early childhood through adolescence to young adulthood. Among other things, this absence of NP effects in adults, but not in children, performing on an identical NP task poses a challenge for advocates of the memory-based episodic retrieval account of NP, which negates any role for an inhibitory process (e.g., Neill, 1997; Neill & Mathis, 1998). These findings also highlight the importance of designing developmentally appropriate NP tasks. Before considering these results in more detail, we provide an overview of the NP effect and pertinent theoretical issues relating to this phenomenon.

A Cost in Selective Attention: Negative Priming Effects

Because of the inherent complexity in typical visual environments, research on selective attention has aimed to better understand the mechanisms underlying the selection of and access to goal-relevant information from amongst competing but goal-irrelevant alternatives. Negative priming effects suggest that the processing of irrelevant information plays an integral part in visual selection (see Fox, 1995, for a review). Typically indexed over a series of sequential trials containing simultaneous target and distractor displays, NP refers to a response cost incurred when the distractor stimulus on the prime trial becomes the target stimulus on the probe trial (i.e., the ignored repetition [IR] condition) relative to trials where prime and probe stimuli are unrelated (i.e., the control condition). With NP procedures providing an indirect means to assess and determine the degree of distractor processing and the nature of conflict resolution during visual selection, NP is an invaluable developmental and clinical measure. However, any inferences from this measure will necessarily depend on the framework used to interpret the NP effect.

Positive and Negative Priming Effects: Memory- versus Inhibition-Based Accounts

While it is largely agreed that the NP effect is the cognitive consequence of ignoring irrelevant information there is less consensus on the precise mechanisms that underlie this effect. Broadly, theories of priming and NP can be separated into memory retrieval and activation-suppression based accounts. Memory-based accounts propose that NP is reflective of mechanisms underlying the automatic retrieval of encoding and processing episodes in memory rather than of suppression processes.

Both models posit a positive relationship between prime salience and the degree of priming benefit on the basis of repetition of a stimulus property, either through enhanced memory cueing (memory-based theory) or through residual increments in activation levels associated with the mental representation of the repeated item (activation-suppression theory). The direction and amount of priming are determined by the difference between either the degrees of response compatibility between features of prime and probe displays or the level of activation tied to such displays. Advocates of the memory retrieval explanation for NP (Neill, 1997: Neill & Mathis, 1998; Neill & Valdes, 1992) propose the NP effect is retrospective. That is, the presentation of the probe target cues the retrieval of past instances from memory containing prior response information associated with that stimulus. By this account, NP results from the implicit retrieval of a memory trace containing response information incompatible with a current correct response requirement. In short, this model explicitly rejects the idea that NP reflects any inhibitory selection mechanisms, and therefore it has the potential to nullify NP as a valid index of inhibitory efficiency in selective attention. Instead, it emphasizes the role of the probe target as a memory-retrieval cue and proposes that performance is mediated by the implicit retrieval of episodic memories containing incompatible information.

Alternatively, proponents for the distractor-inhibition account of NP suggest the effect is prospective. This theory holds that target selection is achieved via a competition-sensitive inhibitory mechanism that functions to reduce concurrent interference from distractor stimuli (Houghton & Tipper, 1994; Neumann & DeSchepper, 1992; Strayer & Grison, 1999).

Residual inhibition associated with the internal representations of distractor stimuli or with response mechanisms linked to these stimuli, increases response latencies when these items next appear as target stimuli. In short, this model incorporates the idea that selection is post categorical and entails both excitatory and inhibitory processes with an excitatory mechanism functioning to maintain or enhance initial activated representations of target stimuli, while a co-existing inhibitory mechanism acts to suppress competing distractor stimuli (Neumann & DeSchepper, 1991; Tipper, 1985).

Developmental Disparities with Conceptual Negative Priming Effects

While widely studied and consistently reported for young adults over a broad range of stimulus types, conceptual NP effects in children have received little attention. Although empirical research is beginning to establish the existence of reliable NP effects associated with location in infants and children (e.g., Amso & Johnston, 2005; Simone & McCormick, 1999), the position of conceptual NP in children is more tenuous. This issue rests largely on direct discrepancies between results from the only two studies to date to investigate conceptual NP in young children (Pritchard & Neumann, 2004, vs. Tipper et al., 1989). Pritchard and Neumann (2004) suggested that experimental manipulations affecting the strength or maintenance of selectional concentration might provide a plausible resolution for divergent findings relating to the presence and absence of conceptual NP effects in children in the respective studies by Pritchard and Neumann and Tipper et al.

In task situations where distracting stimuli are a salient variable, performance decrements are typically heightened in children relative to adults (Lane & Pearson, 1982). In an attempt to account for these increased decrements, Tipper et al. (1989) used a NP priming variant of the Stroop (1935) task in their initial experiment. Whereas standard Stroop trials consist of compound incongruent color-word stimuli and require participants to identify the print color of the color-word while ignoring the identity of the word itself (e.g. the word "blue" printed in yellow ink), the NP version of the Stroop task contains a series of IR trials where the print color corresponds to the identity of the color-word on the preceding trial. Findings from this experiment and two further experiments using Stroop and pictorial NP tasks supported their hypothesis that children (7- to 8-years) would demonstrate less NP than university-aged adults (19- to 21-years), and appeared to confirm the bulk of earlier developmental studies that uphold the childhood disinhibition hypothesis. More recently however, Pritchard and Neumann (2004), using a variant of the Stroop NP task employed by Tipper et al. (1989), and another NP task with incongruent flanker stimuli found significant conceptual NP effects in both experiments for children as young as five.

Advocates of inhibition-based accounts of NP have argued that the appearance of NP effects or selective inhibition in young adults can depend on the engagement and maintenance of a strategic processing set termed *selection state* (Tipper & Cranston, 1985). With *selection state* engaged to cope with selection requirements across the prime and probe displays of IR trials in NP tasks, these authors proposed that if anticipated selection difficulty between target and distractor stimuli is not upheld across IR trials, inhibition associated with distractor representations or response output may dissipate. According to Tipper and Cranston, such a scenario may result in the elimination of NP effects (see also May, Kane, & Hasher, 1995; Moore, 1994; and Schooler, Neumann, Caplan, & Roberts, 1997, for additional accounts of the conditions under which NP may be eliminated).

Pritchard and Neumann (2004) applied an extension of these arguments to account for the developmental differences in NP effects between children and adults noted by Tipper et al.

(1989), and offered a potential resolution for the discrepancies between the respective experiments of Tipper et al. and Pritchard and Neumann. Specifically, we suggested that children might be more susceptible than adults to reductions in *selection state* when processing difficulty is not maintained across IR trials, or the *wider* experimental context of NP tasks.

To account for the disparate findings of conceptual NP in children between the studies by Pritchard and Neumann (2004) and Tipper et al. (1989), we pointed toward seemingly minor, but potentially pertinent, differences in methodology between the respective studies. It was argued that the lack of NP effects for children in Tipper et al.'s study might relate to two additional priming conditions included in the experimental context of that study. While Pritchard and Neumann used only IR and control trials, Tipper and colleagues used neutral and repeated distractor (RD) trials in addition to IR and control trials. Although target and response selection are required in neutral and RD trials, processing difficulty is minimal in such conditions, because the distractor is either a non-interfering meaningless stimulus (neutral condition) or is repeatedly re-presented across trials (RD condition). As a consequence of lessened expectation or anticipation of processing difficulty across these trials, the intensity of *selection state* within the wider experimental context may be reduced. Pritchard and Neumann thus concluded that cognitive inhibitory processes in children are more likely to be engaged in contexts where processing difficulty is high during selection *and* where expectations of processing demand are thoroughly maintained across prime and probe trials in an experiment-wide manner.

Proponents of the episodic retrieval memory-based theory of NP also envisage links between the degree of processing difficulty and the magnitude of NP. For instance, increased

NP is predicted when target identification is difficult on probe trials as the retrieval of information from the prior prime trial may be helpful in initiating a response. The reverse scenario would hold for any reduction in selection difficulty (e.g., see May et al., 1995, for a review). However, while both memory-based episodic retrieval and inhibitory accounts of NP predict similar links between degrees of processing difficulty and NP effects, the combined results of Tipper et al. (1989) and Pritchard and Neumann (2004), provide evidence for a pattern of priming effects that place a strain on the memory-based theory, but might be predicted by inhibition-based frameworks.

To clarify, according to both episodic and inhibition- based accounts of NP, NP should occur when processing difficulty within the IR condition is maximized. However, for episodic memory such issues only relate to manipulations concerning processing difficulties within the IR condition and *not* to further manipulations within the broader experimental context that affect the engagement of *selection state*. Both Tipper et al. and Pritchard and Neumann held processing difficulty in the IR condition constant, yet the magnitude of NP obtained for children differed between the respective studies. Thus episodic retrieval theory loses some credibility as a viable account for NP in the studies by Tipper et al. and Pritchard and Neumann. For episodic retrieval theory to account for the absence of NP for children in Tipper et al.'s study and intact NP for children in Pritchard and Neumann's study, one would have to assume that the episodic memory system was somehow advanced in development for the relatively younger children in the latter study. Alternatively, it seems to us that degree of inhibitory engagement driven by experiment-wide influences on *selection states* were responsible for these disparate results.

Both of the experiments in the present study provide an opportunity to further test whether the exclusion of neutral and RD conditions from the context of an NP task creates the necessary conditions for observing NP effects in children. These experiments compare the conceptual NP effects Pritchard and Neumann (2004) found for children aged five to 12 years with Stroop and flanker NP tasks with those for adolescents (13- to 17-years) and university-age young adults (19- to 25-years) performing in the same tasks. Experimental procedures followed those outlined in Pritchard and Neumann (2004), which required participants to respond to the identity of a target object while ignoring a simultaneously presented distractor object across control and IR displays. In the experiments by Pritchard and Neumann, the prepotent response tendencies for distractor stimuli were greater than (i.e. Stroop NP task) or equal to (i.e., flanker NP task) those for concurrent targets across all prime and probe trials. And this processing demand was maintained within the entire experimental context through the omission of neutral and RD trials, using only control and IR trials, reducing spatial separation between target and distractor, and downplaying the saliency of IR trials.

Since the majority of theoretical, behavioural, and electrophysiological research suggests that the development of selective attention extends over the first two decades of life with children acquiring greater inhibitory control as they move into adolescence, it could be assumed that if anything, levels of NP would be heightened for adults relative to children for both Experiments 1 and 2. Moreover, one might expect to see a systematic increase in the degree of NP produced as a function of increasing age and peaking in young adulthood. The inclusion of adolescent participants in the current experiments gave the additional opportunity to track any potential variations in the developmental course of NP. Determining the degree of suppression used by children vs. adolescents vs. young adults in coping with the competing demands of a NP task may highlight distinct developmental differences in the inhibitory process.

2.3 Experiment 1

Research reporting increased interference effects for children relative to adults has formed much of the impetus behind the widely cited childhood disinhibition hypothesis (see Lechunga, Moreno, Pelegrina, Gomez-Ariza, & Bajo, 2006, for a review). Harnishfeger and Bjorklund (1994), however, caution against the tendency found in the majority of this literature to equate susceptibility to interference with the construct of cognitive inhibition. These authors suggest that whereas interference may suggest susceptibility to distractor intrusion under conditions of multiple distracting stimuli, cognitive inhibition refers to the active removal of task-irrelevant information from working memory during task performance. We add two further observations to those made by Harnishfeger and Bjorklund in order to clarify our concept of cognitive inhibition.

First, the concept of NP inhibition differs from that generally termed inhibition or cognitive inhibition in the developmental literatures. Indeed, it now appears that in terms of neural or cognitive processes there is no single source of inhibition, but rather a constellation of sources of inhibitory processing (Harnishfeger, 1995; Kok, 1999; Neumann, McCloskey, & Felio, 1999; Nigg, 2000). A potential explanation for the age-related differences between the findings regarding the status of inhibition in children reported within the wider developmental literature and the NP literature may be that the term “inhibition” within the developmental literature tends to be used much more broadly. As such, it may often refer to phenomena that might involve mechanisms quite different from those described in the NP selective attention literature (which specifically deals with clashing targets and concurrent distractors activated in initial perception). It is likely that a particular selective inhibitory mechanism is directly responsible for mediating the type of conceptual NP effects we report. Its function is to suppress the mental representation of potentially distracting information, and as such seems dedicated to inhibiting the severest competitor or competitors to a concurrent target, thereby producing a cost when such an item is re-presented as a target. From the outset, we emphasize that our definition of cognitive inhibition adheres firmly to the constructs of active suppression outlined above.

Second, NP tasks can oftentimes provide a more sensitive behavioral index of the degree of distractor processing and inhibition, than tasks assessing concurrent interference effects (Driver & Tipper, 1989; Mari-Beffa, Estevez, & Danziger, 2000; Neumann & Gaukrodger, 2005). If NP effects between children and adults are directly equivalent across NP tasks then there seems no further reason to believe that heightened interference effects imply a *generalized* inhibitory weakness in the attentional system of children. In contrast, the majority of recent studies using attention and electrophysiological measures to assess various selective processes across children and adults in visual, memory, and auditory modalities imply that processes involved in the active inhibition of memory nodes is not developed until at least after puberty and become more efficient over the first two decades of life (Hanauer & Brooks, 2003; Harnishfeger & Pope, 1996; Pearson & Lane, 1991; see also Sanders, Stevens, Coch, & Neville, 2006, for review).

Experiment 1 was designed to test our claim that children may show evidence for NP effects comparable to adults and explore the developmental trajectory of the conceptual NP effect. This experiment provides a direct empirical comparison of NP effects between children, adolescents, and adults engaged in the same child-accessible variant of the Stroop NP task used in the study by Pritchard and Neumann (2004).

2.4 Method

Participants. A total of 150 children[4], 54 adolescents, and 40 university-age adults participated in this experiment. They were spilt into five different groups according to age (i.e., fifty 5- to 6-year-olds, fifty 8- to 9-year-olds, fifty 11- to 12-year-olds, fifty-four 13- to 17-year-olds, and forty 19- to 25-year-olds). The average age for the first group (5- to 6-year-olds) was 6 years and 3 months (range 5 years 2 months to 7 years 1 month). The average age for the second group (8- to 9-year-olds) was 8 years 8 months (range 8 years 0 months to 10 years 0 months). The average age for the third group (11- to 12-year-olds) was 11 years 9 months (range 10 years 10 months to 13 years 0 months). The average age for the fourth group (13- to 17-year-olds) was 15 years 5 months (range 13 years 1 month to 17 years 6 months). The average age for the fifth group (19- to 25-year-olds) was 22 years and 7 months (range 19 years 3 months to 24 years 11 months).

All participants were recruited on a volunteer basis through advertising at local schools and community resources. Written consent was obtained from parents for children and adolescents under consenting age (i.e., below 18 years), and from participants above consenting age (i.e., 18 years and up). All participants had normal color vision and normal or corrected-to-normal visual acuity. The testing procedures were carried out at either the schools involved or at the laboratories at the Department of Psychology at the University of Canterbury.

---

[4] It is important to acknowledge that the sample of 150 children (5- to 12-years) in Study 1 and the NP data reported for this age group are taken from the study by Pritchard& Neumann (2004).

Design. A mixed design was used. The between-subjects variable was age group (5- to 6-year-olds vs. 8- to 9-year-olds vs. 11- to 12-year-olds vs. 13- to 17-year-olds vs. 19- to 25-year-olds). The within-subject variable was priming condition (control vs. IR). Trials consisted of 50% control (where neither the print color nor distractor color word in a Stroop NP stimulus were related to the subsequent Stroop NP stimulus), and 50% IR (where the distractor word in the previous Stroop NP stimulus named the subsequent target print color).

Stimuli and apparatus. The stimuli were presented on 26 x 18 cm cards and consisted of the words GREEN, PINK, BROWN, BLACK, GRAY, YELLOW, WHITE, RED, BLUE, ORANGE, and PURPLE. On each control and IR card all color words were arranged as a single vertical column against a light gray background with the print of each word presented in one of the 11 corresponding colors, with the constraint that the print color and color word were incongruent (see Appendix A). Each Stroop item measured 1.0 cm in height with each display spaced at 1.0 cm intervals down the list. The first two items on each IR card were unrelated in order to reduce the potential saliency of this condition. The 12 cards used in the experiment consisted of six control cards and six IR cards. Four additional control cards were used for practice trials. Presentation orders in the experiment proper were counterbalanced so that half of the participants began with an IR card and the remaining half with a control card. Subsequent cards were presented in regular alternation of the two conditions. A stopwatch was used to record the response latencies to complete color naming for each card. Error scores were tabulated by the experimenters.

Procedure. All participants completed a preliminary color identification task to ensure familiarity with the 11 colors used in the experiment. No participants reported any difficulty with this task. Before the experimental cards were administered, each participant encountered four control practice cards. They were told to name as quickly and accurately as possible the print color of each color word from the top to the bottom of the column on each card. They were also asked not to cease color naming if an error was made, but rather continue to complete the card. Participants were then given the 12 experimental cards (six per priming condition presented in alteration). Each card was covered with a blank sheet that was removed by the experimenter on the word "Go" (see Appendix C for administration details for the Stroop NP task). The stopwatch was started with the removal of the blank sheet and stopped in synchrony with the naming of the last color print on a card. Error scores for each card were tabulated. Errors were defined as either omissions or verbalizations of an absent or incorrect color.

2.5 Results

Reaction time. A mean reaction time (RT) per Stroop item for each participant was calculated for the six cards representing each of the control and IR conditions, respectively. Table 1 presents mean RTs per item and percentage of errors for each of the five age groups for the two priming conditions. Mean RTs were entered into a two-way mixed-design analysis of variance (ANOVA). Priming condition (control vs. IR) was treated as the within-subject factor and age group (5- to 6-year-olds vs. 8- to 9-year-olds vs. 11- to 12-year-olds vs. 13- to 17-year-olds vs. 19- to 25-year-olds) as the between-subjects factor. The between-subjects factor of age group was significant, F (4, 239) = 127.97, p < .01. To determine whether there were differences in the overall RTs between the different age groups, Newman-Keuls post hoc analyses were conducted. Overall RT latencies decreased as a function of age. The results indicated that the 5- to 6-year-olds responded significantly more slowly than the older age groups and that 8- to 9-year-olds also took longer to respond than the older age groups.

Importantly, the within-subject factor of priming condition (control vs. IR) was significant, with naming latencies longer on IR trials than on control trials indicating a NP effect, F (1, 239) = 44.16, $p < .01$. More critically, the interaction between priming condition and age group was not significant, F (4, 239) = 2.01, $p > .09$. The NP effect thus appears similar across the five age groups and was unrelated to differences in overall RT latencies. The mean NP effect per item was 144ms for 5- to 6-year-olds, 84ms for 8- to 9-year-olds, 70ms for 11- to 12-year-olds, 57ms for 13- to 17-year-olds, and 47ms for 19- to 25-year-olds.

Table 1

Mean Reaction Time (in Milliseconds) per Item and Percentage of Errors for each Age Group as a Function of Priming Condition in Experiment 1.

	Priming condition			
	Control (SD)	ER%	IR (SD)	ER%
Age group				
5- to 6-year-olds	2,125 (577)	4.2	2,269 (616)	5.2
8- to 9-year-olds	1,444 (333)	2.3	1,528 (369)	2.8
11- to 12-year-olds	1,119 (254)	2.9	1,189 (279)	3.8
13- to 17-year-olds	905 (210)	2.3	962 (211)	2.0
19-to 25-year-olds	765 (141)	4.1	812 (176)	4.3

Note. IR = ignored repetition

Error Scores. Similar analyses were performed for the error scores. The between-subjects factor of age group (5- to 6-year-olds vs. 8- to 9-year-olds vs. 11- to 12-year-olds vs. 13- to 17-year-olds vs. 19- to 25-year-olds) was significant, F (4, 239) = 5.52, $p < .01$. The within-subject factor of priming condition (control vs. IR) was also significant, F (1,239) =

6.17, $p < .01$ with all particpants making more errors on IR trials than on control trials. Newman-Keuls post hoc analyses indicated that 5- to-6-year-olds made more errors on IR trials and control trials than 8- to 25-year-olds. Finally, there was no significant interaction between priming condition and age group, F (4,239) = 1.44, $p > .22$. Thus, the error data do not appear to compromise the interpretation of the RT results because there was no indication of a speed accuracy trade-off.

2.6 Discussion

The main objective of Experiment 1 was to provide a direct comparison of NP effects between children and adults performing on an identical NP task. This was done primarily to resolve empirical uncertainty about the strength of the NP phenomenon in children. Two further objectives were to explore the developmental trajectory of the NP effect and determine the relation of distractor interference effects to inhibitory function over this course. These were largely undertaken to assess a central idea in cognitive psychology that increased efficiency in inhibitory control parallels increasing age. The primary finding in Experiment 1 suggests no dissociation between the NP effects observed for children and adults. Negative priming effects in five- to 6-year-olds were equivalent to those in adolescents (13- to 17-years) and young adults (19- to-25-years). There was no indication of an increase in cognitive inhibition as a function of increasing age, and thus no indication that children’s longer overall RTs related to inhibitory difficulties as indexed by NP. While there was a systematic decrease in overall RTs as a function of an increase in age, the NP effect was significant and similar across the five age groups.

The implications are that, at least within a Stroop-based NP paradigm, NP effects in children do not differ from those in adolescents and young adults. There appeared to be no obvious developmental trajectory for the NP effect. The results clearly contradict what was widely assumed in the NP literature on the basis of the Tipper et al. (1989) study. Our findings imply there is no clear developmental deficit in cognitive inhibition. Contrary to popular belief, at least one form of cognitive inhibitory control appears to be developed to adult-like capacity early in development.

More broadly, the results from Experiment 1 begin to resolve what seems to be a developmental paradox. From the outset, the conjecture of "childhood disinhibition" and its coexistence with a generalized selective attention deficit posed difficulties. As a developmental phase critical for key knowledge acquisition, childhood marks a period where the majority of constructive learning behaviours take place. Because a functional visual perception system appears in place from infancy on (Bertenthal, 1996; Kellman, 1993, Kellman & Short, 1987; Kellman, Spelke, & Short, 1986), it seems an intact ability to select and direct visual attention to meaningful stimuli should operate equally early in development. As Amso and Johnson (2005) point out, selection would be random without inhibition.

Anomalies between reports of selective attention failure in childhood and what is widely known about the gains made in structured learning behaviors from infancy onward implies that skilled behaviors should best be understood in terms of the processes that enable them. In line with these ideas, studies using retrieval-induced forgetting (RIF) procedures (Anderson, Bjork, & Bjork, 1994), believed to tap the same form of cognitive inhibition reflected in NP (e.g., Anderson & Spellman, 1995; Lechunga et al., 2006; Neumann & DeSchepper, 1992), are beginning to report intact inhibitory effects for children as young as 7, 8, and 9 years old (e.g., Ford, Keating, & Patel, 2004; Lechunga et al., 2006; Zellner & Bauml, 2005). Likewise, research using event-related potential (ERP) waveforms to assess and compare neural processing during auditory selective attention tasks for children and adults observe similar inhibitory effects between these age groups. For example, Sanders et al. (2006) found patterns of ERP waveforms that suggest that the type of excitatory and inhibitory processes implicated by activation-suppression models of NP (e.g., Neumann & DeSchepper, 1991; Tipper, 1985) are remarkably adult-like in children as young as 3 years old. Because inhibitory mechanisms are assumed to play an instrumental role in orchestrating performance in various domains, such as perception, selective attention, working memory, memory retrieval, and motor processes (Anderson & Spellman, 1995; Kok, 1999; Neumann & DeSchepper, 1992), knowing the precise forms of inhibition that operate in these multiple systems, along with their developmental pathways should help further empirical and theoretical knowledge in developmental, cognitive, and neuropsychology domains.

A further notable result in Experiment 1 was that NP effects appear to follow no particular developmental trajectory. While increased overall RTs for the younger three age groups imply that a developmental trend may exist for the other possible factors that may account for these within trial Stroop interference effects such as reading fluency, visual scanning, or time to initiate motor sequences (Everett et al., 1999), there was no discernable change in the strength of the cognitive inhibition process involved in NP across the five distinct developing age groups. Developmental research on selective attention appears to have underestimated some of the inherent skills in younger children. Understanding the developmental path of cognitive inhibition seems paramount to the study of cognitive development, given the recent resurgence of interest in inhibitory processes, their role in information processing, and their related neural systems. Elucidating the dynamics between what may constitute distractor interference (i.e., overall RTs in selective attention trials) and cognitive inhibition in early development remains a further challenge.

2.7 Experiment 2

Experiment 2 was designed to replicate and extend the results of Experiment 1 with a different stimulus type. If similar NP effects appear in children, adolescents, and adults, then such effects should generalize across a range of different stimuli. Participants in Experiment 2 were engaged in a flanker NP task where the relationship between a prior distractor and a current target was based on identity rather than semantics. More specifically, the stimuli consisted of a central target color blob flanked on both sides by non-target incongruently colored blobs. Using conflicting target and distractor blobs, rather than incongruent color-word stimuli, avoids the prepotent-alternative response dynamic inherent in the Stroop paradigm. While processing difficulty is held constant in the flanker NP task, with reduced spatial separation maintained between target and distractor stimuli, the response tendencies associated with such neutral stimuli are likely to be more equipotent.

Because this task has only previously been used to produce significant NP effects for children (see Pritchard & Neumann, 2004), it was not known whether NP effects with the same flanker stimuli would occur in adults. However, given the propensity for young adults to show robust and consistent NP effects across a range of stimuli and task requirements, the expectation was that this age-group should show significant NP effects on the flanker NP task. Furthermore, given that young adults typically produce robust NP effects (see Fox, 1995, for a review), while evidence for intact NP in children is more tenuous (Pritchard & Neumann, 2004, vs. Tipper et al., 1989), one might expect NP effects in young adults to be greater in magnitude than those in children, or that there may be a systematic increase in the NP effect as a function of increasing age.

2.7 Method

Participants. The same 150 children and 54 adolescents who participated in Experiment 1 were included in Experiment 2. A different group of fifty young adults, 19 to 25 years old, participated in Experiment 2 (mean age 19 years, 7 months).

Design. A mixed design was used. The between-subjects variable was age group (5- to 6-year-olds vs. 8- to 9-year-olds vs. 11- to 12-year-olds vs. 13- to 17-year-olds vs. 19- to 25-year-olds) and the within-subject variable was priming condition (control vs. IR). Trials consisted of 50% control (where there was no relationship between the colors of distractor blobs in the previous display and the color of the subsequent target blob color), and 50% IR (where the color of the distractor blobs in the previous display matches the subsequent target color blob).

Stimuli and apparatus. The stimuli consisted of 11 unique sets of three-different shaped color blobs presented in a column on twelve 32 x 22 cm manila cards. The sequential arrangement of rows differed for each card. In addition, each row was randomly staggered to either the left or right in an attempt to reduce the saliency of the IR condition. Visual distances between individual blob rows were the same for both control and IR cards. The outer blobs in each row functioned as distractors, and the centre blob functioned as the target. The 11 colors used in Experiment 1 were used again as colors for blobs in Experiment 2. The color for the target blob always differed from the color shared by the flanking distractor blobs (see Appendix B). Six control cards and 6 IR cards were used in the experiment. Four additional control cards were used for practice trials. Presentation orders were handled as in

Experiment 1. A stopwatch was used to record the time taken to complete color naming for each card. Error scores were tabulated by the experimenters.

Procedure. After the initial color identification task, participants were given verbal instructions for the color blob task. They were told to name as quickly and accurately as possible the color of each central blob while ignoring the outer blobs, from the top to the bottom of the column on a given card. Again, it was emphasized that they should not cease color naming if an error was made but rather continue to complete color naming for the card (see Appendix D for administration details for the colour-blob NP task). After completing the four practice control cards, participants were given the 12 experimental cards (6 per priming condition presented in alteration). Timing procedure was handled as in Experiment 1. Error scores for each card were recorded.

2.8 Results

Reaction time. A mean reaction time (RT) per flanker item for each participant was calculated for the 6 cards representing the control condition and the 6 cards representing the IR condition. Mean RTs per item and percentages of errors for the control and IR priming conditions are shown for the five age groups in Table 2. Mean RTs were submitted to a two-way mixed-design ANOVA. Priming condition (control vs. IR) was treated as the within-subject factor and age group (5- to 6-year olds vs. 8- to 9-year-olds vs. 11- to 12-year-olds vs. 13- to 17-year-olds vs. 19- to 25-year olds) as the between-subjects factor. The between-subjects factor of age group was significant, F (4, 249) = 119.71, $p < .01$. Overall RT latencies decreased as a function of age. Newman-Keuls post hoc analyses indicated that the 5- to 6-year-olds responded significantly more slowly than 8- to 25-year-olds and the 8- to 12-year-olds responded significantly more slowly than the 13- to 25-year-olds, all p's < .01. The within-subject factor of priming condition (control vs. IR) was highly significant indicating a NP effect with naming latencies longer on IR trials than on control trials, F (4, 249) = 49.85, p < .01.

Table 2

Mean Reaction Time (in Milliseconds) per Item and Percentage of Errors for Each Age Group as a Function of Priming Condition in Experiment 2.

	Priming condition			
	Control (SD)	ER%	IR (SD)	ER%
Age group				
5- to 6-year-olds	1,412 (425)	1.6	1,488 (403)	4.2
8- to 9-year-olds	963 (225)	1.1	1,007 (216)	2.3
11- to 12-year-olds	765 (167)	1.2	810 (165)	2.9
13- to 17-year-olds	619 (145)	1.1	640 (142)	0.6
19-to 25-year-olds	561 (110)	1.2	575 (97)	1.0

Note. IR = ignored repetition

Finally, the interaction between priming condition and age group was significant indicating between group differences in the NP effect, F (4, 239) = 3.70, p < .01. Unexpectedly, there was a systematic and significant decrease in the NP effect as a function of an increase in age. Post hoc analyses were carried out to establish where this difference lay. Results revealed that the NP effect for 5- to 6-year-olds was significantly larger than the NP effect for 13- to 17-year-olds and 19- to 25-year-olds and that the size of the NP effect for 8- to 12-year-olds was significantly greater than the NP effect for 19- to 25-year-olds, all p's < .01. Further analyses revealed the NP effect was only marginally significant for 13- to 17-

year-olds ($p < .09$), and did not even approach statistical significance for 19- to 25-year-olds ($p > .28$). See Figure 2.1 for a graphical depiction. The mean magnitude of the NP effect per item was 76ms for 5- to 6-year-olds, 44ms for 8- to 12-year-olds, 45ms for 11- to 12-year-olds, 21ms for 13- to 17-year-olds, and a non-significant 14ms for 19- to 25-year-olds.

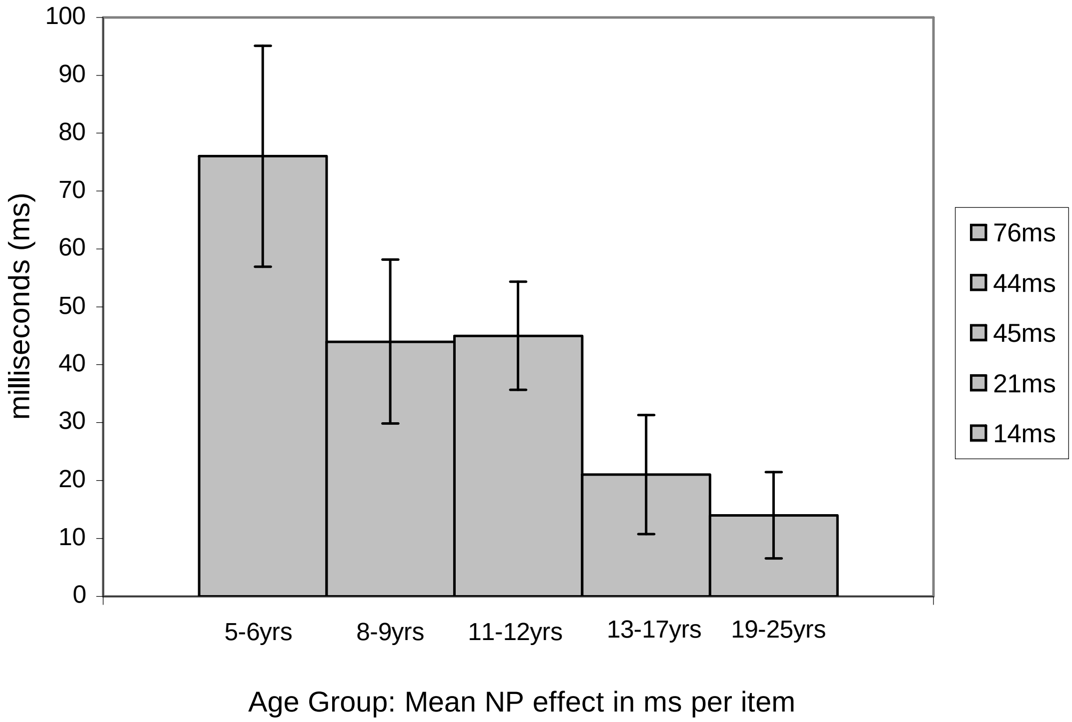


Figure 2.1. Mean NP effect in milliseconds per item with the color-blob NP task as a function of age group in Experiment 2. Bars depict standard errors.

Error scores. Error scores were submitted to similar analyses. The between-subjects factor of age group (5- to 6-year-olds vs. 8- to 9-year-olds vs. 11- to 12-year-olds vs. 13- to 17-year-olds vs. 19- to 25-year-olds) was significant, $F(4,249) = 7.87$, $p < .01$. Newman-Keuls ost hoc analyses revealed there was a decrease in error scores with age; however, the rate of decrease was different for each priming condition. In the control condition, there was a decrease in error scores for 5-to 12-year-olds that was significantly different from error scores displayed by 19- to 25-year-olds, (p's < .02). In the IR condition, there was a decrease in error scores for 5- to-6-year-olds and 12- to 19-year-olds but an increase for error scores for 8- to 9-year-olds (all p's < .05). No significant effects were found for 19- to 25-year-olds. The within-subject factor of priming condition (control vs. IR) was also significant, F (1,249) = 5.65, p < .02, with all participants making more errors on IR trials than control trials.

Finally, there was a significant interaction between priming condition X age group, F (4,249) = 3.92, p < .05. Post hoc analyses indicated that 5- to –12-year-olds made more errors on IR trials than 13- to-25-year-olds and 13- to 17-year-olds made significantly less errors on IR trials than 19- to 25-year-olds (all *p*'s < .05). Further analyses based on error performance indicated that increased error scores on IR trials relative to control trials for five- to 12-year-olds did not appear to compromise the interpretation of the RT results via inflationary effects on IR RTs (see Pritchard & Neumann, 2004). Error scores as a function of priming condition did not approach significance for 13- to 17-year-olds or 19- to 25-year-olds, all p's > .05.

2.9 Discussion

The main objective of Experiment 2 was to replicate and extend the results of Experiment 1 to a different stimulus type. Experiment 1 found no dissociation between the NP effects observed for children, adolescents, and adults. Negative priming effects in 5- to 6-year-olds were equivalent to those in adolescents (13- to 17-year-olds) and university-age young adults (19- to-25-year-olds). Negative priming results in Experiment 2 revealed a pattern of NP effects that was different from the pattern observed in Experiment 1. In fact, Experiment 2 yielded a number of unexpected and surprising findings. Despite producing highly significant NP effects even for children aged 5 and 6 years, Experiment 2 did not produce the same effect for young adults. Instead, Experiment 2 was characterized by a systematic decrease in NP as a function of increasing age and punctuated by complete disappearance of NP in young adults.

This is the very age group that in the past has been taken as the sine qua non for producing robust NP effects.

Even though Experiment 2 contained 10 additional adult participants, thus increasing statistical power for this age group, there was no evidence for a significant NP effect in 19- to 25-year-olds. A notable outcome given that an equivalent age group, but smaller in number, had produced highly significant NP effects with a different NP task in Experiment 1.

While Experiment 2 produced a very different pattern of NP than Experiment 1, the pattern for overall RTs was analogous to the pattern observed in the different groups in Experiment 1. That is, there was a systematic decrease in overall RTs as a function of an increase in age. Given, that within-trial RTs on incongruent Stroop and flanker trials are often calculated as and taken to indicate interference effects in the developmental literature (e.g., Balaban, Snidman, & Kagan, 1997; Comalli, Wapner, & Werner, 1962), data patterns relating to overall RTs in Experiments 1 and 2 may help to illuminate the developmental interplay between the processes underlying interference and cognitive inhibition.

Most importantly, however, the findings in Experiment 2 highlight the potential pitfalls of using developmentally inappropriate NP tasks, because instead of adults producing NP and children failing to do so, as most might expect, just the opposite pattern was revealed. Theoretical implications of this unique finding, along with the specific challenges it poses for memory-based accounts of NP, are treated in the General Discussion section.

2.9.1 General Discussion

The specific purpose of this study was to first provide direct empirical evidence to further our claim that significant conceptual NP effects found for children in a previous study may be similar to those found in adults (Pritchard & Neumann, 2004). A further aim was to distinguish definitively the role of inhibition in NP phenomena. It was hoped that this would help establish NP as a more demonstrably valid index for inhibitory function in developing and clinical populations. Two secondary goals were to explore the developmental trajectory of the NP effect over five distinct developmental samples, and to evaluate the potential outcomes of this course on inhibitory and memory-based approaches to NP.

To date, the series of NP experiments reported by Tipper et al. (1989) remains the only published research to provide a direct comparison of conceptual NP effects between children and adults. Their failure to find evidence for the NP effect in children relative to adults formed the basis for three almost universally accepted assumptions in NP research. The first of these being that children show no evidence for a NP effect; the second that the absence of NP in children and the presence of NP in young adults suggest a developmental deficit in cognitive inhibitory processes; and the third that while young adults consistently produce robust NP effects across a range of NP tasks and stimuli types children will not. The present set of results clearly challenges all these assumptions. Experiment 1 showed significant and similar NP effects in five differing developmental samples ranging from 5- to 25-years of age. More noteworthy was the critical new finding in Experiment 2 of the absence of NP effects in young adults coupled with the presence of significant NP effects in children as young as five engaged in the same NP task. These findings also present a challenge for the memory-based episodic retrieval theory; the major opponent of the inhibition-based account of NP. For episodic retrieval to account for the results in Experiment 2, for example, one would need to claim a more developed episodic memory system in young children than in adults.

What are the Pitfalls of Developmentally Inappropriate Negative Priming Tasks?

Currently, developmental studies on the various forms of inhibitory processes specified in cognitive and NP research domains suggest inhibitory function becomes increasingly effective over the first two decades of life with no notable deficits during early to middle adulthood. Evidence from neuropsychological studies suggests this is largely associated with the protracted development of the prefrontal cortex (Huttenlocher, 1990). The prefrontal cortex, seated anterior to the primary motor and premotor cortex, is assumed to play a central role in higher-level cognition and the mediation of various types of inhibitory function. Research using functional magnetic resonance imaging (fMRI) suggests the frontal lobes play an integral part during episodic memory retrieval (e.g., Hayes, Ryan, Schnyer, & Nadel, 2004). Episodic memory is regarded as highly age sensitive, undergoing sizeable expansion in functional capacity throughout childhood, peaking between the ages of 20 to 30, then remaining relatively constant until age 60 when it begins a gradual decline. If episodic-retrieval theory is to account for the presence of NP effects found for children but not young adults in Experiment 2, one would have to assume that our sample of 5 and 6 year olds showed unusually advanced development in the episodic memory system.

Moreover, to account for trends pointing towards a systematic decline in NP as a function of an increase in age in Experiment 2, one would further have to assume that during adolescence, episodic memory for visual material and the means for access to the tagging such stimuli have undergone may be subject to a decline as development progresses, with the effectiveness of this memory system declining even further in young adults. Taken together, the outcomes of Experiments 1 and 2 thus seem to pose significant challenges for both the idea of an underdeveloped inhibitory mechanism in children and for episodic retrieval accounts of NP. And because similar NP effects were found for five and 25-year-olds (at least in Experiment 1), it seems more likely that NP reflects the operation of one of the fundamental elements of a selective attention system. From a developmental perspective, the existence of attentional inhibitory processes crucial for learning, rather than processes underlying the automatic retrieval of episodic memories, seems the more plausible explanation for NP. In our view, the patterns of NP and interference observed in the present study may highlight distinct developmental differences in the cognitive system underlying the onset of an effective selection system. In the remaining sections, we attempt to pin down potential reasons why the pattern of NP and within-trial interference effects (as indexed by overall RTs) observed in Experiments 1 and 2 may have occurred, and consider more generally how these results might bear further on episodic retrieval accounts of NP.

Is There a Role for Interference in the Development of Cognitive Efficiency?

Based on the findings of longer overall RTs in children relative to adults across the two experiments reported here and those reported in the wider cognitive developmental literatures, it seems clear that some aspects of cognition do not mature until adulthood. One caveat, however, concerns the correlational nature of the trends found for overall RTs and for the NP effects for 5- to 25-year-olds engaged in the same NP tasks. For example, trends showing a systematic decrease in overall RTs with increasing age, maintained their respective patterns in both Experiments 1 and 2. However, there were clear differences in NP for adults and adolescents between Experiments 1 and 2 but no differences in NP for 5- to 6-year-old children in either experiment.

While it is important to acknowledge that not all of the age differences noted for overall RT scores may relate to distractor interference effects (see Everett et al., 1999), the present findings may also indicate that heightened interference effects (i.e., longer overall RTs) in 5- to 6-year-olds did not unduly affect the ability of this age group to select among response-competitive stimulus items. But then why might very young children encounter more interference and yet produce robust NP effects on a NP task that did not appear to produce interference effects or significant NP in young adults? More speculatively, it may be that in early developmental periods when erroneous selection is more likely, interference operates as a catalytic process to stimulate and ensure the proper engagement of the *selection state* and concomitant inhibitory process. An accurate selection process is likely to be key during a developmental phase where fundamental knowledge acquisition takes place. Interference may become relatively less critical for inhibitory engagement as development progresses and attentional concentration becomes more reliant.

Some support for this contention comes from a recent empirical study by Bub, Masson, and Lalonde (2006) on the dynamics between attentional set, and interference and suppression effects. To index the degree of word interference and suppression during color naming in a Stroop-like task, Bub et al. used incongruent color naming trials that required children to switch between naming the print color of an everday word (i.e., the word “face” printed in red) on one trial to reading a similar word that appeared in standard black type in the trial immediately subsequent. Word suppression effects were gauged by comparing children’s word reading times on incongruent trials relative to reading time for words appearing after neutral color-naming trials (i.e., “xxxx” printed in red) and interference effects were gauged by comparing children’s color naming times on colorword trials relative to neutral trials. These authors aimed to assess the idea that heightened Stroop interference noted in younger children in comparison to older children might relate to reasons other than inhibitory failure.

A series of delta analyses performed by Bub et al. (2006) revealed two seemingly contradictory results. While younger children showed a larger interference effect than older children, they also showed a greater degree of word suppression. To account for these findings, Bub et al. suggested younger children's heightened interference and larger suppression effects might relate to a greater difficulty in maintaining attentional priority on the color naming task set. This logic was based, in part, on earlier work by Ridderinkhof, van der Molen, Band, and Bashore (1997) that found increased flanker interference in children aged five-years relative to children aged 12-years related to an inconsistency in the ability to maintain task appropriate stimulus-response mappings. Applied to their own study, Bub et al. (2006) proposed that because younger children may be less effective in maintaining the color-naming task set than older children when task conditions require them to switch between color-naming and word reading, they may encounter stronger competition from a color word Of more relevance to the current discussion, was the interpretation given by these authors to account for the heightened suppression effects that appeared to coexist with heightened Stroop interference in younger rather than older children. Bub and colleagues suggested that for younger children to overcome such competition, in order to focus on the attentional set, they resort to a stronger magnitude of suppression.

The foregoing suggests that to us that interference in children may act as a magnifier of incongruent stimuli combinations that "warn" and focuses the response system for upcoming selection. This interference effect may become less important as development progresses and the cognitive system increases in efficiency. This seems in line with the decreased interference effects noted in developmental research for later childhood and adolescence in comparison to the earlier developmental periods. However, what may be important to realise is that increased interference effects observed during early childhood need not be reflective of an impaired ability to select among directly competing stimuli. Rather, heightened interference effects noted for younger age groups may be more reflective of a process that stimulates the attentional system into the necessary *selection state* crucial for effective inhibitory function to occur early in development. In this sense, interference may operate as the “training wheels” that allow for effective information processing in individuals whose attentional set is not yet adult-like in competency.

As no significant NP effects were found for adults with the flanker task in Experiment 2, we presume this may relate to task design and a developmental superiority in attention set. More speculatively, that *selection state* was not engaged to a degree necessary to observe NP in 19- to 25-year-olds. That is, there may have been a perceived decrease in processing difficulty with incongruent color flankers relative to that perceived for the Stroop stimuli in Experiment 1 where distractor competitiveness was more likely to be pre-potent rather than equi-potent. However, a lack of observable positive priming effects for young adults in Experiment 2, suggests that inhibition was engaged sufficiently to outweigh any possible facilitatory effects of persisting distractor activation. Thus, the *selection state*, although greatly minimised was not abandoned in this age group. More simply, it may be that because young children have fairly recently learned their colors, competing colors from interleaved blobs may still be relatively conflicting for them. Adults on the other hand, may not experience the same degree of conflict from colors in the immediate vicinity, and thus would not require large amounts of inhibition to resolve such minimal conflict. To summarize, it seems that at least for children, the threshold for perceived selection difficulty may be lower so as to activate the inhibitory process necessary at this stage of development to ensure that appropriate selection processes are in place (i.e., *selection state*). This appears to operate most effectively in task situations that are designed to maintain expectations of processing demand throughout the entire experimental context. These variables may become less critical for the engagement of *selection state* in later development.

Empirical Pitfalls for Memory-Based Episodic Retrieval Accounts of Negative Priming

Evaluating whether the inhibitory-based or the memory-based account of NP is more effective at explaining the NP effects obtained for the experiments reported here presents challenges. The results from the present experiments, combined with those of Pritchard and Neumann (2004), and Tipper et al. (1989) suggest that a single-underlying factor account of NP, such as the automatic retrieval of information would not be in line with the entire pattern of conceptual NP effects reported. That is, the relation between developmental differences in NP, processing demand, and maintenance of processing demand is not always clear. For example, child-based NP research suggests children show NP when processing difficulty is heightened but only in experimental contexts where processing demand is held constant (e.g., Stroop NP task in Pritchard & Neumann, 2004, vs. Stroop NP task in Tipper et al. 1989). The current experiments suggest adults show NP when processing difficulty is high and expectation of processing demand is held constant in the experimental context (e.g., Stroop NP task in Experiment 1), but *not* when processing demand is lowered (e.g., flanker NP task in Experiment 2). The same experiments found that when processing demand is held constant in the experimental context, children will show NP when processing difficulty was high (e.g., Stroop NP task), and that distractor color-blob stimuli were sufficiently interfering for children to be subject to inhibition during target selection (e.g., flanker NP task).

Episodic retrieval theory can accommodate this type of flexibility even less than inhibition-based accounts. It is difficult to see how an episodic retrieval explanation would account for these findings without some modification to include the alternating influences of the processing difficulty experienced by the participant and the maintenance of processing demand on the formation of episodic memories. Both children and adults performing in these Stroop NP tasks were exposed to highly similar if not identical stimuli across IR prime and probe trials offering the same retrieval cues. Unless the appearance of NP in children relative to adults was mediated by variables affecting processing demand across the *wider* experimental context in addition to variables directly affecting processing difficulty across prime and probe trials, there seems no clear reason why NP should have substantially differed between the 7 year-old children and adults in Tipper et al.'s study, yet be equivalent for 5 year-old children and adults in the present study. Findings such as these join a growing body of research that questions the exclusion of inhibition in NP accounts (e.g., Tipper, 2001).

Towards Designing Developmentally-Appropriate Negative Priming Tasks

To conclude, current theorising in developmental research seems to favor the differentiation of multiple types of inhibition (e.g., Harnishfeger, 1995; Kok, 1999; Nigg, 2000), and as the reports for intact NP in children (e.g., Pritchard & Neumann, 2004) illustrate, this is not without good reason. Despite popular consensus on the idea of a widespread inhibitory deficit in childhood, developmental deficits do not seem to apply to all forms of inhibitory capacity. Our results show that children even as young as five and six years of age can be as effective as university-age students in their ability to suppress distractor stimuli in a NP task. Findings from the current study, however, also caution that the appearance and disappearance of NP in either of these age groups may hinge on critical variables in task design conducive or unconducive to the developmental phase of the wider attentional system. More simply, developmental differences in distractor inhibition may be stimulus specific in nature. For example, a recent study of Stroop interference effects in children by Peru, Faccioli, and Tassinari (2006) found that 8-year-old children were highly susceptible to color interference when a word reading response was required, producing a significant Reverse Stroop effect. This effect was not found for older children. Applied to the results obtained in the current study with the flanker NP task, it may be that young children produced NP effects while adults did not, simply because distractor colors incongruent with target colors were more interfering for this age group than for adults. Such outcomes highlight the potential pitfalls of using developmentally inappropriate measures for developmental and cognitive research. The present study also indicates that apparent developmental differences on one measure of selective attention do not necessarily equate to deficits and cautions against relying on extrapolating a broad purported deficit from the evidence of a single study.

CHAPTER 3

Study 2

Adding nonconflict trials in Stroop negative priming tasks eliminates distractor suppression effects in children, but not adults[5]

3.1 Abstract

Incongruent Stroop trials require participants to ignore a color-word while naming its conflicting print color. To date, in negative priming (NP) versions of the Stroop task, intact NP effects have only emerged in young children when prepotent response tendencies for all distractor stimuli are greater than or equal to those for concurrent targets. Experiment 1 replicated those conditions by using consistently incongruent Stroop trials to assess NP effects between 7-year-old children and adults. Experiment 2 compared the same NP effects between children and adults when neutral and repeated distractor Stroop conditions were also encountered in a task modelled upon the work of Tipper et al. (1989), who failed to observe NP in children. Whereas Experiment 1 replicated Pritchard and Neumann's (2004) report of intact NP in children, Experiment 2 replicated Tipper et al.'s (1989) report of intact NP in adults but not children. We concluded that distractor inhibition processes are fully functional in children, but that including neutral and repeated distractor conditions in this Stroop NP task causes the obliteration of NP effects in young children.

---

[5] Paper submitted to *Developmental Psychology*. Pritchard, V. E., & Neumann, E. (submitted MS). Adding nonconflict trials in Stroop negative priming tasks eliminates distractor suppression effects in children, but not adults.

3.2 Introduction

Without a basic ability to prevent a response to the most dominant stimulus input, behavior would be chaotic and unrelated to current goals. How the human information processing system acts to overcome attentional competition generated by concurrent stimulus inputs has become a topic of enduring interest in cognitive psychology. A prominent view holds that the selection of targeted information relies on an activation-reducing inhibitory process to suppress concurrently competing distractor information (Driver & Tipper, 1989; Houghton & Tipper, 1994; Neumann & DeSchepper, 1991; see also Anderson & Spellman, 1995, and Neumann & DeSchepper, 1992, for similar ideas on inhibition as a selection mechanism in memory). Using a negative priming (NP) index, the experiments in this study were intended to determine whether empirical discrepancies regarding the operation of this inhibitory process in children are the result of a methodological artifact.

NP is defined as the slowed response to a target stimulus on a probe trial when that stimulus or close categorical relation was ignored as a distractor on a preceding prime trial (i.e., the ignored repetition condition). The effect is indexed by comparing response times on ignored repetition (IR) trials with those on control trials in which there is no relation between current target and preceding distractor. The inhibition-based account of NP (e.g., Tipper, 1985) incorporates dual process models of attention that contend that the internal representations for target and concurrent distractor are activated in parallel at initial exposure. To facilitate a task-relevant response, an excitatory process acts to enhance target information while an inhibitory process acts to suppress distracting information (Houghton & Tipper, 1994; Neumann & DeSchepper, 1991, 1992). Behavioral and brain-imaging studies interpret NP as indicating that information in competition with current target information is subject to an involuntary form of neural inhibitory activity during target selection (Grison, Tipper, & Hewitt, 2005; Vuilleumier, Schwartz, Duhoux, Dolan, & Driver, 2005).

A number of studies show that the onset of this suppression process is often activation-sensitive, with inhibitory activity maximal when the internal representation of the distractor stimulus is highly activated and in a response competitive state (Strayer & Grison, 1999; Grison & Strayer, 2001). Residual inhibitory activity tied to the internal representation of a recently ignored stimulus is believed to produce the NP effect. This NP effect is often thought to be transient, occurring only on an immediate IR probe trial. However, increasing evidence for long-term NP effects (Grison et al., 2005; Treisman & DeSchepper, 1996; Neumann & Russell, 2000; see also Pritchard, 2002, unpublished masters thesis, for similar evidence in 11- to 12-year old children) suggests the pattern of neural activity associated with an ignored stimulus is encoded and may be reinstated after longer temporal delays between the critical prime and probe displays (Grison et al., 2005). Although not the focus of the present study, it is important to note that non-inhibitory explanations for NP effects have also been proposed (e.g., Neill & Valdes, 1992; Neill, Valdes, Terry, & Gorfein, 1992). We defer discussion of these models until the General Discussion section, but note for now that most, if not all, theoretical conclusions negating the role of inhibition in NP phenomena have been formed on the basis of research involving adults.

Surprisingly, there are few studies of conceptual (identity or semantic) NP effects in children. Existing work shows conceptual NP in young children manifests in an inconsistent manner. For instance, while Tipper, Bourque, Anderson, & Brehaut (1989) first reported that NP is not observed in 8-year-old children, the only follow-up study to date reports intact and similar NP in varying age groups of children between 5- to 12-years old (Pritchard &

Neumann, 2004). Pritchard and Neumann suggested that NP in children might only emerge when prepotent response tendencies for all distractor stimuli involved in the task are greater than or equal to those for concurrent targets. The present experiments were designed to test Pritchard and Neumann's hypothesis that, relative to adults, the distractor inhibition process underpinning NP may not operate in children when the probability of encountering high degrees of distractor competition is reduced by 50% (as was the case in the Tipper et al. study, relative to the Pritchard and Neumann study). The following section presents a brief overview of the developmental differences that appear to exist in NP effects with the Stroop color-word task.

The Stroop task has become a mainstay of research on selective attention (MacLeod, 1991). The so-called Stroop effect typifies a class of interference whereby the introduction of stimuli incongruent with target stimuli slows reaction time (RT). The standard finding is that participants take longer name the print colors of incongruent word stimuli (e.g., the word “BLUE” printed in red) than to name the print colors of neutral stimuli (e.g., the letters “xxxx” printed in red) or congruent stimuli (e.g., the word “BLUE” printed in blue). Stroop (1935) surmised that as a consequence of learning to read, the associations between color-words and their meanings become more direct than those for perceptual color. The result is asymmetrical influence, with word reading exerting greater interference on color naming than vice versa.

NP was first documented in the context of a Stroop task. In their seminal study on the effect of Stroop stimuli sequencing on interference, Dalrymple-Alford and Budyar (1966) noted that relative to color-naming RTs on standard incongruent trials, RTs were further slowed on trials in which the color to be named matched the identity of the preceding color-word. To account for this NP effect, Dalrymple-Alford and Budyar ascribed a critical role to distractor suppression in the Stroop interference resolution process. Contemporary models of the Stroop effect also contend that the inhibitory process involved in the NP effect may play a critical role in ultimately resolving interference occurring during Stroop performance (see Schooler, Neumann, Caplan, & Roberts, 1997a, 1997b).[6]

It has long been known that Stroop interference is heightened in children relative to adults (Comalli, Wapner, & Werner, 1962; Friedman, 1971). In their initial experiment exploring the relationship between Stroop interference and inhibitory ability, Tipper et al. (1989, Experiment 1) compared Stroop-based NP effects between young adults and children. Results supported their prediction that children would show less Stroop NP than adults to the extent that while intact Stroop NP emerged in adults, there was a complete absence of Stroop NP in children. A further experiment by these authors (Tipper et al., Experiment 3) using pictorial stimuli yielded similar results, although some evidence for a slight (albeit nonsignificant) NP effect in children was obtained. These findings formed the basis for two almost universally accepted assumptions in the NP literature: first, that children show no evidence for NP, and second, that there are developmental differences in the inhibitory process underpinning NP. There is a further assumption that NP in children might be harder to detect because of the larger distractor intrusion effects in this age group (Nigg, 2000).

[6] These ideas are strengthened by recent work showing that interference between the color and colorword dimensions of the Stroop stimulus can occur during a change in response mode at the categorical level. For instance, Durgin (2000, 2003) obtained a reverse Stroop effect (i.e., color naming exerts greater interference on word reading) when participants were required to point to color patches while reading incongruent colorwords. These studies are consistent with translational models of Stroop interference (e.g., Sugg & McDonald, 1994) and demonstrate the theoretical importance of competition occurring among internal representations. Durgin’s findings are problematic for models of Stroop interference that ascribe the basis for Stroop asymmetry via the strength of stimulus-response associations or the automaticity of word reading (e.g., Cohen, Dunbar, & McClelland, 1990).

Conceptual NP effects in children have been readdressed only recently. Pritchard and Neumann (2004) found children aged 5- to 12-years produced intact NP in a Stroop NP task and in a newly devised flanker-like NP task that required participants to name the color of a central target blob while ignoring the incongruent color of two outer flanking distractor blobs. Additionally, in a recent follow-up NP experiment using their earlier Stroop NP task, Pritchard and Neumann (2007a, Experiment 1) found the Stroop NP effect indexed for 5- to 6-year-old children was highly compatible with, if not identical to, Stroop NP effects obtained for 19- to 25-year-old adults. These findings clearly challenge the notion that inhibitory processes involved with information selection are less effective in children than adults (see also Lechunga, Moreno, Pelegrina, Gomez-Ariza, & Bajo, 2005; Ford, Keating, & Patel, 2004, for findings of similar adult-like inhibitory effects for children in the retrieval-induced forgetting paradigm).

On the other hand, Tipper et al.'s (1989) failure to detect Stroop NP in children and equivocal evidence for pictorial NP effects in children has implied that the "negative priming effect develops inconsistently in early childhood up to first grade" (Nigg, 2000, p. 227). New evidence, however, suggests this view may have come through default. In their recent NP study Pritchard and Neumann (2007a, Experiment 2) compared NP effects between children and adults with the color-blob NP task and found a unique and unexpected dissociation. While the color blob task produced intact NP in 5-year-old children, there was a failure to detect NP in a large sample (n=50) of 19- to 25-year-old adults with this same task. This finding was particularly surprising given that young adults are known for producing robust NP effects across a diverse range of task and stimulus types (see Fox, 1995, and May, Kane, & Hasher, 1995). To account for this anomaly, Pritchard and Neumann (2007a) reasoned that the manifestation of NP could depend on the relative degree of target-distractor conflict with children finding adjacent incongruent color blobs more conflicting than adults. These findings caution against relying on extrapolating a broad purported deficit from the evidence of a single study and highlight the possibility that NP effects in either children or adults may be influenced by the specific stimuli used in the task and other experiment-wide contextual factors.

3.3 The Present Study

Pritchard and Neumann (2007a) suggested that findings of inconsistent Stroop NP effects between children and adults (cf, Pritchard & Neumann, 2004, 2007a, and Tipper et al., 1989) may be due to methodological differences concerning the maintenance of word interference during color naming. For example, Pritchard and Neumann noted that Stroop NP effects between children and adults were comparable in their study in which the distractor dimension of the Stroop stimulus was consistently response-competitive in all trials, and non-comparable in the study by Tipper et al. in which the distractor dimension was response competitive in only half of the trials.

On the basis of this data pattern, Pritchard and Neumann (2007a) argued that the inhibitory process underpinning NP is essentially intact in children but may be modulated as a consequence of encountering some trials in which target selection is relatively easy. More specifically, they reiterated their earlier theory (see Pritchard & Neumann, 2004) on the determining role of *selection state* in the manifestation of NP. Tipper and Cranston (1985) argued that selective inhibition is part of an attentional processing set termed *selection state* that is engaged to cope with selection requirements across prime and probe trials. With the *selection state* induced and maintained by the degree of difficulty involving target selection, Tipper and Cranston proposed that if target selection difficulty or anticipation of such is not upheld across IR trials, the active suppression of distractor stimuli (and hence, the resultant NP effect) would dissipate in strength. Consistent with this view, a number of studies have demonstrated that continuous anticipation of target selection difficulty across trials appears critical to maintaining the distractor inhibition process and eliciting NP effects in adults (Khurana, 2000; Moore, 1994; Schooler, et al., 1997a).

Pritchard and Neumann (2004) hypothesized that *selection state* and resultant NP effects in children might be particularly prone to elimination if target selection difficulty is minimized or not consistently upheld throughout the duration of the task at hand. Under such task conditions, children feel less incentive to ignore distractors across conditions, and thus attention may become less tightly selective. In support of this idea these authors pointed towards potentially pertinent differences in methodologies between the respective studies comparing Stroop NP effects between children and adults. Pritchard and Neumann's (2004, 2007a) studies contained only control and IR trials in which the distractor (i.e., word) dimension of the Stroop stimulus was consistently response competitive. Tipper et al.'s study, designed to test for concurrent interference and facilitation effects in addition to NP effects, had contained 50% of trials in which the distractor dimension was less response competitive (i.e., neutral trials in which the distractor consists of meaningless orthographical information, and repeated distractor [RD] trials where the same distractor color-word is repeatedly re-presented across trials). Because NP did not emerge in children under these conditions, Pritchard and Neumann argued that Tipper et al.'s use of neutral and RD trials may have reduced selective processing demands to a point that was detrimental to obtaining NP effects in children. From this they proposed that the operative *selection state* might have an important bearing on the level (or even the presence) of inhibition elicited in NP studies with children.

More specifically, Pritchard and Neumann's (2004, 2007a) *selection state* hypothesis predicts that there should be an interaction between the proportional number of trials in which target selection is difficult and age on the magnitude of NP. It follows that in an NP task containing only trials in which target selection is consistently difficult, children should show intact NP comparable in magnitude to NP in adults. Whereas, if children are less able than adults to re-engage or maintain selection state in an 'on-line' manner after experiencing a salient ease in target selection difficulty, then only adults should show intact NP in an NP task where there is a 50:50 ratio of difficult to easy target selection trials. [7]

Given the ambiguity of the empirical situation concerning the manifestation of Stroop NP effects in children, and the scarcity of research to date, the main purpose of the present study was to contribute more evidence to help clarify this issue. This was achieved by replicating the respective methodologies of the studies reporting Stroop NP effects in both children and adults (i.e., Pritchard & Neumann, 2004, 2007a) and the one reporting NP effects in adults, but not in children (i.e., Tipper et al., 1989). We thus compared Stroop NP in children and adults when neutral and RD trials were excluded from the NP task context (Experiment 1) or included (Experiment 2). If Pritchard and Neumann's *selection state* hypothesis is correct then Stroop NP should be intact and comparable between children and adults in Experiment 1, whereas in Experiment 2 only the adults should show intact Stroop NP.

---

[7] It is notable that in Tipper et al.'s (1989) Experiment 3 using pictorial stimuli, where the ratio of difficult selection to easy selection trials was 67:33, they obtained evidence possibly suggestive (albeit nonsignificant) of NP in children.

3.4 Experimental Overview

In the current study, Experiments 1 and 2 both used a blocked list-wise trial presentation format and a manual timing procedure similar to those employed in the seminal discovery of the Stroop NP phenomenon (Darymple-Alford & Budyar, 1966). These procedures were also used in Tipper et al.'s (1989) original comparison of Stroop NP between children and adults, and in the more recent studies by Pritchard and Neumann (2004, 2007a).

3.5 Experiment 1

Experiment 1 was designed to replicate the experimental conditions in which Pritchard and Neumann (2004, 2007a) found comparable Stroop NP between children and adults. Therefore, participants in Experiment 1 encountered only control and IR trials in which the distractor dimension of each Stroop stimulus was consistently response-competitive and incongruent with the target dimension. As in the earlier experiments by Pritchard and Neumann, control and IR trials appeared on separate cards that were presented in regular alternation. In case of an age-specific effect, participants in Experiment 1 were matched by age to those in the original Stroop NP experiment by Tipper et al. (1989).

3.6 Method

Participants. There were two participant age groups in Experiment 1. One group consisted of 35 children (18 female) with a mean age of 7 years 6 months, and the other of 30 adults (21 female) with a mean age of 18 years 5 months. Following ethics approval, children were selected from a local primary school that agreed to take part in the study. Adults were recruited from the university campus and a local tertiary business school through advertisements and word of mouth. Participation was voluntary with all participants giving written consent for their participation. Written consent was also obtained from the parents of children participating in the study. All participants had normal colour vision and normal or corrected-to-normal visual acuity.

Design. A 2 x 2 mixed design was used. The between-subjects variable was age group (children vs. adults) and the within-subject variable was priming condition (Stroop control vs. Stroop IR). Experimental trials (see Table 1, p.57) consisted of 50% control trials (in which neither the print color nor color-word of a Stroop stimulus related to the subsequent Stroop stimulus) and 50 % Stroop IR trials (in which the color-word in the preceding Stroop stimulus always identified the subsequent print color). The NP effect was computed as the difference between Stroop control and IR cards.

Stimuli. The stimuli were presented on 26 x 18cm cards and consisted of 11 different color-words. The 12 cards used in the experiment consisted of 6 control cards and 6 IR cards. On each card all 11 Stroop stimuli were arranged as a vertical list and printed against a light gray background. The print of each word was set in one of the 11 colors with the constraint that the color-word and print color were incongruent. Four additional control cards were used as practice trials. On IR cards, to minimise the potential saliency of the IR condition, the first two Stroop items were control trials. Presentation orders were counterbalanced. A stopwatch was used to record color-naming RTs.

Procedure. A double-blind procedure was followed and all participants were tested individually. Prior to the experiment, each participant completed a preliminary color identification task to ensure familiarity with the 11 colors used in the experiment. Participants were then given verbal instructions to name as quickly and as accurately as possible the ink color of each color-word from the top to the bottom of the list on each card.

They were asked not to stop color naming if an error was made but to continue until the color naming for the card was completed. Each participant completed four control practice cards before encountering the 12 experimental cards. For each card the experimenter said, “Ready” as a warning and on the word “Go” removed a blank sheet covering the test card. The stopwatch was started with the removal of the blank sheet and stopped in synchrony with the naming of the last color on a card. Errors, classified as either omissions or verbalizations of an absent or incorrect color, were recorded for each card.

Table 1

Example of Control and IR Stroop Trials in Experiment 1

Priming conditions	
Control	IR
PINK-r	ORANGE-br
BLUE-g	BLACK-y
RED-blk	PURPLE-blk
WHITE-y	GRAY-pur
ORANGE-pi	GREEN-g
BROWN-pur	PINK-gr
BLACK-w	RED-pi
PURPLE-bl	BROWN-r
GREEN-or	WHITE-br
GRAY-br	YELLOW-w
YELLOW-gr	BLUE-y

Note. Lower case letters depict the target print colors of Stroop stimuli (r = red. g = gray, pi = pink, gr = green, br =brown, blk = black, or = orange, w = white, pur = purple, bl = blue, y = yellow)

3.7 Results

Table 3 (p.66) shows the mean RT per Stroop item and percentage of error for Stroop control and IR conditions for children and adults in Experiment 1. Mean RTs and error rates were submitted to a 2 (age group: children vs. adults) x 2 (priming condition: control vs. IR) repeated measures analysis of variance (ANOVA).

RT analyses. There was a significant main effect of age group, F (1, 63) = 129.30, p < .01, with overall RTs significantly slower for children than for adults. The main effect for priming condition was highly significant and indicated a Stroop NP effect with color naming RTs longer for Stroop IR trials than control trials, F (1,63) = 29.51, p < .01. Importantly, there was no significant interaction between priming condition and age group, F (1,63) = 2.71, p > .10, suggesting Stroop NP effects were similar between children and adults. To confirm our findings, a mean NP cost score (IR minus control) was computed for both age groups. These NP scores were then compared via a t-test for independent samples with a significance level set at .01. Results showed the Stroop NP effect was invariant between children and adults, t (63) = -1.65, p >.10. The mean magnitude of the NP effect per item was 99ms for children and 55ms for adults (see Figure 3.1). The proportional magnitude of the NP effect in adults (7%) did not differ significantly from that found in children (6%) (Mann-Whitney U, p >.48). Furthermore, the effect size (Cohen's d) of the mean NP effect per item was d= 0.46 for children and d= 0.43 for adults.

Error analyses. Similar analyses were conducted for error scores. The main effect of age group was significant, F (1,63) = 12.87, p <. 01, indicating children made more errors than adults. There were no other effects, p's >.26.

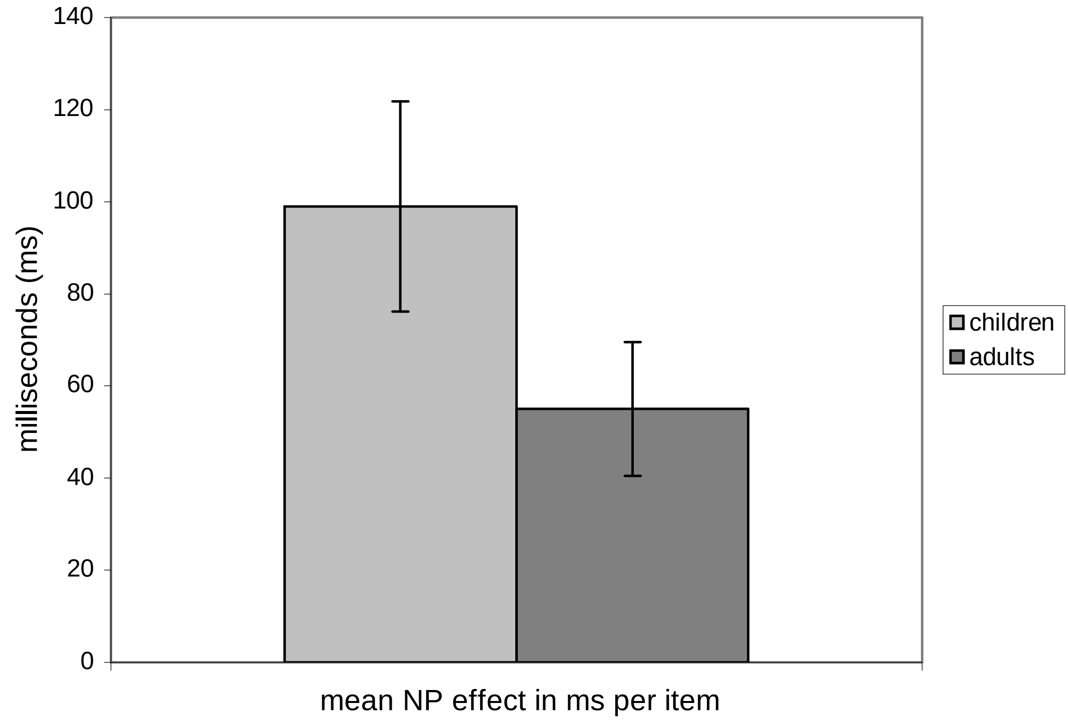


Figure 3.1. Mean Stroop NP effect in milliseconds per item for children and adults in Experiment 1 (context exclusive of neutral and RD trials). Bars depict standard errors.

3.8 Discussion

Experiment 1 clearly replicated the results obtained by Pritchard and Neumann (2004, 2007a). Stroop NP was intact and comparable between young children and adults when only control and IR trials were encountered, and when the distractor dimension of each Stroop stimulus was consistently response competitive and incongruent with the target dimension. Despite the long-held and widely accepted idea that NP is absent in young children, this age group produced an adult-like Stroop NP effect.

3.9 Experiment 2

Experiment 2 was designed to replicate the experimental methodology used by Tipper et al. (1989) that produced intact Stroop NP in adults, but not children. Thus, participants encountered neutral and RD conditions (in which the distractor either is [neutral condition], or ultimately becomes [RD condition], less response competitive than the target), in addition to the same control and IR conditions that were used in Experiment 1. As in the experiment by Tipper et al., neutral and RD Stroop trials were presented on separate cards and intermixed among control and IR cards. The ratio of neutral and RD trials to control and IR trials was 50:50. All other variables were kept identical with those in Experiment 1 to ensure that any potential modulation of NP in Experiment 2 was related only to the random presentation of easy and difficult target selection trials.

Following Pritchard and Neumann's (2004, 2007a) *selection state* hypothesis, it also seemed pertinent to take into account the potential effect of pre-set anticipation of target selection difficulty on NP results. If NP is potentially based upon an attentional processing strategy then any induced strategy set as a consequence of encountering only difficult selection trials in Experiment 1 might work to eliminate potential modulating effects of neutral and RD trials on NP in Experiment 2. To avoid this, Experiment 2 used different participants.

3.9.1 Method

Participants. Experiment 2 included 65 new participants recruited from the same sources as participants in Experiment 1. Participants were grouped by age to include 35 children (20 female) with a mean age of 7 years 9 months and 30 adults (12 female) with a mean age of 19 years 2 months.

Design. A 2 x 4 mixed design was used. The between-subjects variable was age group (children vs. adults) and the within-subject variable was priming condition (neutral vs. RD vs. control vs. IR). Experiment 2 contained 25% neutral trials, 25% RD trials, 25% control trials, and 25% IR trials.

Stimuli and procedure. Experiment 2 used the 6 control and 6 IR cards that were used in Experiment 1, and differed only by the addition of 6 neutral cards and 6 RD cards (see Table 2). Neutral and RD cards were similar in design to those used by Tipper et al. (1989). On each neutral card, the stimuli consisted of rows of x's ranging from three to 6 x's per stimulus item, with the print of each appearing in one of 11 colors used in Experiment 1. On each RD card, the same color-word was repeatedly re-presented across trials while its print color differed between trials (the color-words BLUE, GREEN, and RED were each used on two of the cards for this condition).

Administration and counterbalancing procedures were identical to those in Experiment 1. The same four additional control cards were used as practice trials and 50% of the participants began with a control card and 50% with an IR card. Subsequent cards were presented in regular alternation of the four conditions.

Table 2

Example of Neutral and RD Stroop Trials appearing among Control and IR Stroop Trials in Experiment 2

Priming conditions	
Neutral	Repeated Distractor
XXX-r	GREEN-pur
XXXXXX-gr	GREEN-y
XXXX-blk	GREEN-br
XXXXX-y	GREEN-w
XXXXXX-bl	GREEN-gr
XXX-pi	GREEN-blk
XXXX -w	GREEN-or
XXXXX-g	GREEN-pi
XXX-or	GREEN-br
XXXXXX-br	GREEN-g
XXXX-pur	GREEN-bl

Note. Lower case letters depict the target print colors of the Stroop items (r = red. g = grey, pink = pi, gr = green, br =brown, blk = black, or = orange, w = white, pur = purple, bl = blue, y = yellow)

3.9.2 Results

Participants' mean RTs per Stroop item and corresponding error rates were entered into a 2 (age group: children vs. adults) x 4 (priming condition: neutral vs. RD vs. control vs. IR) ANOVA. Mean RTs and error rates are shown in Table 3 (p.66).

RT analyses. The between-subjects factor of age group was highly significant, F (1, 63) = 100.24, $p < .01$, with the mean RT faster for adults than children. There was also a significant main effect of priming condition (neutral vs. RD vs. control vs. IR), F (3, 189) =

27.58, $p < .01$, indicating mean RTs for the four priming conditions increased in the following order (neutral < RD < control < IR). More importantly, there was a significant interaction between age group and priming condition, F (3, 189) = 27. 58, $p < .01$. To explore this interaction, planned contrasts between the control condition and each of the other three priming conditions (neutral vs. RD vs. IR) were conducted. These results are presented below.

Planned comparisons

Neutral and RD effects in children and adults. There was a significant main effect for the neutral condition, F (1, 63) = 95.60, p <. 01, and for the RD condition F (1, 63) = 98.42, $p < .01$, indicating that naming latencies were significantly faster on neutral and RD trials than control trials. Mean benefit scores for the neutral (control minus neutral RT) and RD (control minus RD RT) conditions were computed for each age group. Age group differences between these neutral (p <. 02), and RD (p <. 01) scores indicated that the RT benefits for these conditions were significantly greater for children than adults.

NP effects in children and adults. There was a significant main effect for the IR condition F (1,63) = 9.86, $p < .01$, indicating longer naming latencies on IR trials than control trials. However, there was also a significant interaction between priming condition and age group, F (1,63) = 4.94, $p < .03$. A mean NP cost score (IR minus control) was computed for both age groups. Single-sample t-tests following up group differences on these scores found that the NP score was significantly smaller than zero (indicating intact NP) for adults (t (29) = 7.34, p <. 01) but not for children (t (34) = 0.53, $p > .60$). The mean magnitude of the NP effect per item was 15ms for children and 85ms for adults (see Figure 3.2). Furthermore, the proportional size of the NP effect in adults (9%) was significantly greater than that in children (1%) (Mann-Whitney U, p <. 04). The effect size (Cohen's d) of the mean NP effect per item was d= 0.06 for children and d= 0.69 for adults. Bonferroni post hoc analyses confirmed that neutral, RD, and NP effects differed significantly between children and adults (all p's <. 01).

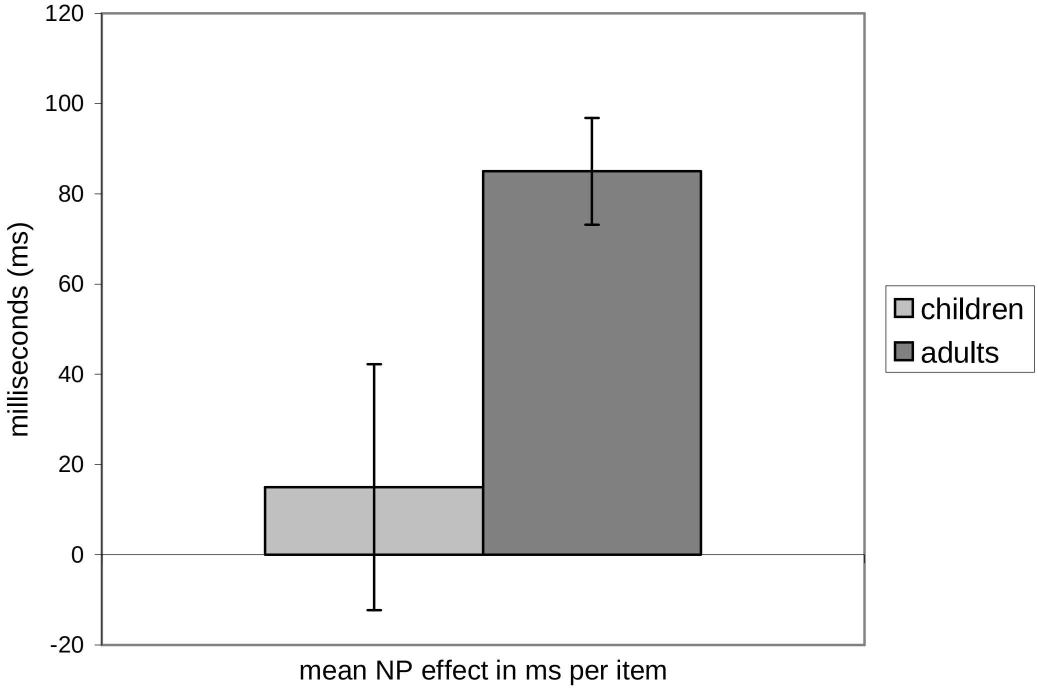


Figure 3.2. Mean Stroop NP effect in milliseconds per item for children and adults in Experiment 2 (context inclusive of neutral and RD trials). Bars depict standard errors.

To determine whether NP effects for the two age groups differed by experiment, separate ANOVAs for children and adults including only control and IR priming conditions were conducted. For children, a significant interaction between priming condition and experiment (F (1,68) = 5.62, p <. 02) was observed thus confirming there was an elimination of NP in children in Experiment 2, relative to Experiment 1. No such interaction was obtained for adults, F (1,58) = 2.93, p > .09, and in fact any trend toward marginal significance would indicate larger NP in Experiment 2, relative to Experiment 1, for the adults.

Error Analyses

The between-subjects factor of age group was not significant, F (1, 64) = .03, p > .95. There was a significant main effect of priming condition (neutral vs. RD vs. control vs. IR), F (3, 189) = 23.18, p < .01, with the mean error rate for the four priming conditions increasing in the following order (neutral < RD < control < IR). No other error effects approached significance.

Table 3

Mean Reaction Time (in Milliseconds) per Stroop Item and Percentage of Errors as a Function of Priming Condition for Children and Adults in Experiments 1 and 2

	Children				Adults			
	Experiment 1		Experiment 2		Experiment 1		Experiment 2	
Priming condition	M	SD (er%)	M	SD (er%)	M	SD (er%)	M	SD (er%)
Neutral			1,034	196 (1.5)			633	133 (1.5)
RD			1,288	185 (2)			754	145 (2)
Control	1,482	344 (4.0)	1,530	370 (2.5)	754	130 (2)	835	169 (3)
IR	1,581	338 (4.5)	1,545	309 (4)	807	175 (2)	920	200 (4.5)

3.9.3 Discussion

Experiment 2 replicated the developmental differences in Stroop NP reported in the original experiments by Tipper et al. (1989). Stroop NP emerged intact in adults but not in children when neutral and RD conditions were intermixed with control and IR conditions. The results of Experiment 2 are in striking contrast to those obtained in Experiment 1. Whereas Experiment 1 produced intact and comparable magnitudes of Stroop NP between children and adults, Experiment 2 failed to produce any evidence for Stroop NP in children, despite the fact that the identical control and IR cards were used in both experiments. Furthermore, analyses comparing mean RTs for control and IR priming conditions by experiment showed that the elimination of NP for children in Experiment 2 was coupled with control RTs that were slower relative to those for children in Experiment 1. Because the only manipulated variable distinguishing Experiment 2 from Experiment 1 was the addition of neutral and RD cards, it is reasonable to conclude that encounters with neutral and RD Stroop trials modulated NP effects and increased distractor intrusion for children in Experiment 2. In contrast, encounters with neutral and RD Stroop trials did not appear to modulate NP effects for adults in Experiment 2.

Another notable result in Experiment 2 was that color-naming RTs for children and adults were reliably faster on neutral and RD trials than control trials. This implies that target selection difficulty was significantly eased during performance on these trials. Moreover, the response benefits gained on neutral and RD trials were larger for children (369ms per item) than for adults (151ms per item). Therefore, it was evident that children experienced a significant ease in target selection difficulty on these trials. Tipper et al. (1989) reported similar findings concerning children’s RTs on neutral and RD trials. Thus, in Experiment 2, the outcome of absent Stroop NP in children may be attributable to reductions in target selection difficulty experienced within some of the conditions in the experiment-wide context of conditions testing for NP.

3.9.4 General Discussion

The principal goal of this study was to test the speculation that the inclusion of neutral and RD trials among control and IR trials might reduce NP in young children but not adults. This was done primarily to resolve the contradictory findings by Pritchard and Neumann (2004) of NP in children and Tipper et al.'s (1989) finding of a lack of NP in children. The results were clear-cut. Experiment 1 confirmed Pritchard and Neumann's findings by showing intact and comparable Stroop NP effects in children and adults when only control and IR conditions are encountered in the task. In stark contrast, Experiment 2 confirmed Tipper et al.'s (1989) findings by showing Stroop NP only in adults, but not children, when neutral and RD conditions are encountered in the task, in addition to control and IR conditions. Therefore, the discrepant results obtained in the two studies appear to be accounted for by this seemingly minor methodological difference. Importantly, these findings help re-affirm that, contrary to popular belief, the inhibitory process underpinning NP is essentially intact in young children. They also begin to establish the boundary conditions under which conceptual NP is apt to emerge in young children.

Are children more susceptible than adults to reductions in selection state?

Results from the current study seem in line with the contention that young children may be more susceptible than adults to modulations in their *selection state* (Pritchard & Neumann, 2004, 2007a). In Experiment 1, children appeared capable of inhibiting distractor stimuli in an adult-like manner when distractors were consistently response competitive. In

Experiment 2, however, children seemed less able than adults to selectively inhibit distractor stimuli after experiencing a significant ease in target selection difficulty when neutral and RD trials comprise half of the conditions in the experiment.

A recent independent study by Frings, Feix, Röthig, Brüser, and Junge, (in press), however, challenges Pritchard and Neumann's (2004) *selection state* hypothesis. Frings et al. used the color-blob NP task on a large sample of 152 children aged 6- to 11-years to test Pritchard and Neumann's idea of modulation of NP by RD trials. Half of the children in the study by Frings et al. encountered 1/3 RD, 1/3 control, and 1/3 IR trials, and the other half encountered only control and IR trials. Frings et al. found that RD trials did not modulate NP in children. Instead, these authors showed that NP was intact and comparable between those children who had encountered RD trials and those that had not. Frings et al. claimed that the inclusion of RD trials could not explain the different results of Pritchard and Neumann (2004) and Tipper et al. (1989), and were thus problematic for Pritchard and Neumann's *selection state* hypothesis. Their findings and the conclusions derived from them are perplexing given that the results reported in the current study appear to strongly support the hypothesis.

On closer examination it appears that Frings et al. (in press) mistook the conditions under which NP effects may fail to manifest in children. In the experiment by Frings et al. the ratio of difficult selection conditions to easy selection conditions was 67:33, whereas in Tipper et al.'s (1989) original experiment NP failed to emerge in children when the ratio was 50:50. Furthermore, Frings et al.'s experiment included only RD trials, whereas Tipper et al.'s experiment included RD *and* neutral trials. It is important to note that Pritchard and Neumann (2004) specifically contended that the inclusion of both neutral *and* RD trials may be necessary to ease target selection difficulty to a degree detrimental to obtaining NP effects in children. The inclusion of a neutral condition may be especially important in light of indications that the neutral condition yields greater ease of selection than the RD condition. Similar to the findings reported by Tipper et al. (1989), the present Experiment 2 shows that children's RTs are significantly faster on neutral than RD trials, confirming that target selection is comparatively easier. Consequently, the study by Frings et al. does not necessarily discredit the *selection state* hypothesis, nor does it discredit the claim that the inclusion of neutral *and* RD trials in a NP task may reconcile disparate findings between the studies of Tipper et al. (1989) and Pritchard and Neumann (2004, 2007a).

Increasing the number of difficult selection trials while decreasing the number of easy selection trials thus appears to boost NP levels in children. Further evidence for this comes from Tipper et al.'s (1989) Experiment 3 in which the RD condition was removed from the experimental context and a trend towards NP in children was found. Pritchard and Neumann contended that this relative increase in children's NP levels might relate to an increase in the degree of *selection state* intensity caused by the removal of the RD condition from the experimental context and that the retention of the neutral condition ultimately prevented NP effects from reaching significant levels in children. Had the proportion of easy nonconflict trials in Tipper et al.'s Experiment 3 been 1/3 RD trials rather than 1/3 neutral trials, then these authors may have obtained more straightforward evidence for intact NP in children.

When task situations encourage reductions in attentional concentration, selective attention in children may be subject to diffusion. Applied to control and IR priming conditions in the current study, a division of attention between target and distractor information should be evidenced by increased sensitivity toward distractor information on control trials and decreased NP effects on IR trials if there is a lessening of active inhibition toward distractors. This, in turn, could give rise to them being processed inadvertently or even occasionally in a more conscious sense (see Houghton & Tipper, 1994). A comparison of the RT data patterns for children in Experiments 1 and 2 are remarkably consistent with such predictions. Not only were the mean control RTs numerically larger for children in Experiment 2 (1581ms) than Experiment 1 (1482ms), but this was also coupled with the opposite trend for the IR RTs, which led to the significant interaction between experiment and priming condition. No such interaction was observed for adults. This implies children's slower RTs on control trials, or increased tendency to process distractor information, was due to the decrease in selective inhibition of the conflicting distractors in Experiment 2, which also eliminated NP.

The possibility that children have a tendency to process the distractor information in a more conscious sense than adults in Experiment 2 may also help explain the large RT benefits observed for this age group on RD trials via the process of habituation. The habituation hypothesis (Sokolov, 1963) holds that the repetitive presentation of identical stimuli results in the lessening of the orienting response (an automatic response typically elicited by the presentation of novel stimuli). Lorch, Anderson, and Wells (1984) argue that the repeated re-presentation of a stimulus in an experimental situation enables an individual to form a short-term neuronal representation of this stimulus. An integration or match between stimulus and internal representation serves to reduce the orienting response.

The suggestive evidence that children appear to be induced to process color-words to a greater degree than adults in Experiment 2, implies that the internal representations children form for color-word stimuli on RD trials are established more readily than in adults. This should mean a faster reduction of the orienting response for children on RD trials and leads to the prediction that there should be a greater response benefit for children than adults on these trials, as indeed there was. Similar RT benefits for children relating to distractor interference have been observed with congruent Stroop stimuli. Wright and Wanley (2003) found that children gained a larger RT benefit than adults on Stroop trials where the ink color to be named matched the identity of the concurrent colorword. These authors argued that because children are more prone to attend to the word dimension of the Stroop stimulus than adults, the semantic activation for color information served to facilitate children's response to the word-congruent ink color more than adults.

Implications for non-inhibitory accounts of NP

Although the inhibition-based account of NP remains the most influential explanation of the effect (Tipper, 2001; Grison et al., 2005), it should be noted that an alternative account explicitly rejects any role for inhibition in NP. The episodic retrieval theory proposed by Neill & Valdes (1992) emphasizes the role of the probe target stimulus as a memory retrieval cue. By this account, slowed response to a target stimulus in the IR condition is attributed to the elicitation of an episodic representation or instance that contains incompatible prime response information (a "do not respond" tag) that conflicts with and slows the opposing response required in the probe (i.e., respond). Resolving the conflict between these incompatible tags during the processing of the probe target produces the NP effect.

As noted in the introduction, such theories have been generated on the basis of research involving adults. It is difficult to see how the present data involving children could be understood in terms of episodic retrieval of "no response" tags. Children in Experiments 1 and 2 were exposed to identical IR prime and probe displays and presumably the probe target offered the exact same "do not respond" tag in both experiments. Yet while NP was obtained for children in Experiment 1, no NP effect was obtained for children in Experiment 2. Without some modification to include the influence of the broader experimental context on the formation of episodic memories, the episodic retrieval theory of NP cannot accommodate our findings (see also Pritchard & Neumann, 2007a).

Summary and Conclusion

The main implications from the present set of experiments are that children may only produce NP in task situations that place a consistent demand on *selection state.* Children may be less able than adults to attend selectively in task situations where distractor stimuli are less likely to disrupt correct responding over a number of trials. More generally, however, the distractor inhibitory process underpinning NP is fully intact in young children. Equivocal evidence for NP effects in children (Tipper et al., 1989) most likely relate to methodological factors that served to decrease the degree of *selection state* intensity in children, rather than signalling incomplete development in the inhibitory component of selective attention. It thus appears that the selective inhibition process in children, as in adults, is reactive to response competition, but for the children the encountered competition must be even more consistently prevalent.

SECTION 2

TWO STUDIES OF

IDENTITY-BASED NEGATIVE PRIMING IN ATYPICAL DEVELOPMENT

CHAPTER 4

Study 3

Interference and Negative Priming Effects in Adolescents with

Attention Deficit Hyperactivity Disorder[8]

4.1 Abstract

Three visual selective attention tasks were used to measure potential differences in susceptibility to interference and inhibitory cognitive control processes in 16 adolescents diagnosed with attention deficit hyperactivity disorder (ADHD) and 45 similar-aged Controls. Susceptibility to interference was assessed using the Stroop Color and Word Naming test. Efficiency of distractor inhibition was assessed in two conceptual negative priming tasks. The majority of studies in this area indicate that people with ADHD demonstrate higher levels of interference and lower negative priming effects in comparison with age-matched peers. However, we found that although the ADHD group was consistently slower to name target stimuli than the Control group, there were no differences in interference or negative priming between the two groups.

[8] Published paper: Pritchard, V. E., Neumann, E., & Rucklidge, J. J. (2007). Interference and negative priming effects in adolescents with attention deficit hyperactivity disorder. *American Journal of Psychology*, *120*, 91-122.

4.2 Introduction

The natural world contains many visual objects that compete for attention. Because some objects are likely to be more relevant than others at a given moment, the ability to selectively attend to relevant information while ignoring irrelevant information is fundamental to human information processing. Determining the underlying mechanisms involved in selective processing has evolved into an important research area. A prominent view is that the successful selection of targeted stimuli relies on an activation-reducing inhibitory mechanism to suppress concurrently competing distractor stimuli (e.g., Driver & Tipper, 1989; Neumann & DeSchepper, 1991). The present study is devoted to assessing whether this selective inhibitory mechanism is adversely affected in adolescents with a recognized attentional deficit.

Distractor Processing: Stroop Interference and Negative Priming Effects

Despite being ignored, unattended visual distractor stimuli often produce traceable priming effects, which can be used to investigate inhibitory functioning. More specifically, these “negative priming” (Tipper, 1985) effects are indexed behaviorally as an increase in the reaction time, (RT) or decrease in the accuracy of processing a stimulus that was previously ignored. This impaired response to recently ignored stimuli was first documented in Dalrymple-Alford and Budayr's (1966) seminal study on the effect of Stroop stimuli sequencing on interference. Stroop (1935) tasks require participants to identify the hue of the ink in which a color-word is written while ignoring the word itself. A response in the Stroop task is particularly difficult when the hue is incongruent with the color word (e.g., the word "blue" written in green ink). Stroop interference or intrusion from inadvertent word processing is gauged by comparing response times and accuracy scores for the incongruent color-word stimuli with neutral stimuli (e.g., the letters "xxxx" written in green ink). In their extension to the basic Stroop interference effect, Dalrymple-Alford and Budayr discovered that, relative to typical incongruent Stroop trials, response latencies were even greater when the target hue of the current color-word corresponded to the distractor color-word on the preceding trial. To account for this negative priming (NP) effect, Dalrymple-Alford and Budayr ascribed a crucial role to distractor suppression in the Stroop interference resolution process. Some contemporary models of the Stroop interference effect continue to advocate the important role of such a selective inhibitory mechanism in resolving Stroop interference (Schooler, Neumann, Caplan, & Roberts, 1997a, 1997b). And the idea that active inhibition is one of the fundamental components of selective attention has been reinforced by more recent studies using NP paradigms.

A wide variety of stimulus types and procedures have been used to demonstrate NP effects similar to those observed by Dalrymple-Alford and Budayr (1966). For example, Tipper (1985; Tipper & Cranston, 1985) obtained NP using sequential trials of concurrently presented target and distractor letters in one task and trials containing superimposed drawings of common objects in another task. Broadly, the NP effect is the response cost incurred when the distractor on a preceding trial becomes the target on a subsequent trial (ignored repetition [IR] condition), relative to control trials (unrelated [UR] condition) in which the current target is unrelated to the prior distractor (Neumann & DeSchepper, 1991; see Fox, 1995, for a review).

Although NP tasks have been used extensively to elucidate the nature of inhibitory mechanisms underlying visual selective attention in typical and atypical adults, they have been underused in the assessment of these abilities in typical and atypical adolescent populations. A primary aim of the present research was to redress the neglect of this tool for investigating potential inhibitory capacities in typical and atypical adolescent samples. Sixteen adolescents with a formal diagnosis of Attention Deficit Hyperactivity Disorder (ADHD) and 45 age-matched Controls were tested in each of three experiments. The Stroop Color and Word Naming Test developed by Golden (1978) was used in Experiment 1. A Stroop NP task modelled on the early work of Dalrymple-Alford and Budayr (1966) was used in Experiment 2, and a flanker NP task adopted from Pritchard and Neumann (2004) was used in Experiment 3. The sections that follow present a brief review of existing clinical and NP research concerned with attentional processing and inhibitory function in people diagnosed with ADHD.

ADHD, inhibitory function, and Stroop interference effects: The disinhibition hypothesis

ADHD is characterized by inattention, impulsivity, and hyperactivity (American Psychiatric Association, 2000). This disorder affects 3 - 6 % of children from varied cultures and geographic regions with some symptoms, particularly those relating to inattention, tending to persist into and throughout adulthood (Barkley, Fischer, Edelbrock, & Smallish, 1990; Barkley, Murphey, & Kwasnik, 1998; Hart, Leahy, Loeber, Applegate, & Frick, 1995). Deficits in inhibitory control and behavioral inhibition are seen as the most distinctive characteristics of ADHD. Defective behavioral inhibition results in a failure to resist an inappropriate behavioral response, and poor inhibitory control results in the inability to interrupt an initiated response, withhold a planned response, or protect an ongoing activity from interference (Barkley, 1998; Quay, 1988; Rubia, Oosterlann, Sergeant, Brandeis, & Leeuween, 1998; Schacher & Logan, 1990; Tannock, 1998).

Much of the contemporary theoretical and empirical literature on ADHD suggests that the disorder relates to deficiencies in the frontal lobe inhibitory systems. These deficiencies are attributed to a dysfunction in dopaminergic transmission in the prefrontal cortex and in striatal (basal ganglia) structures (Hynd, et al., 1993). The prefrontal cortex is assumed to modulate executive functions involved in complex goal-directed behavior and play a paramount role in the mediation of various types of inhibitory function. Pharmacologic treatment with psychostimulant medications designed to stimulate the release or block the reuptake of dopamine, a neurotransmitter instrumental in the brain's braking or inhibiting system, has been shown to alleviate some of the symptoms of ADHD.

Recent theoretical models of ADHD have tended to focus on deficits in various executive inhibition processes. Barkley's (1997) disinhibition hypothesis of ADHD, for instance, attributes some of the central causes of deficits in executive function in children with ADHD to a pervasive dysfunction in three key constituents of response inhibition; the ability to stop an ongoing response, inhibit a prepotent response, and control interference. He argued that these impairments cause deficits across a range of executive functions. Research assessing the performance of children with ADHD on tasks involving executive inhibitory functions have found that in comparison to typical children, children with ADHD perform poorly, exhibiting a greater tendency to commit perseverative errors, longer naming latencies, and difficulty in ignoring and inhibiting responses to irrelevant stimuli (Barkley, Grodzinsky, & DuPaul, 1992; Brodeur & Pond, 2001; Douglas, 1988; Pennington & Ozonoff, 1996; Ross, Hommer, Breiger, Varley, & Radant, 1994). These findings suggest that the dysfunction of the putative frontal lobe inhibitory systems associated with ADHD leads to ineffective selection, which may result in heightened interference or distractor intrusion. Barkley (1994) assigned a central role to inhibitory control in executive function arguing that impairments in inhibitory function in children with ADHD may cause deficits typically shown by these children across a range of executive tasks designed to assess prefrontal function (Barkley, et al., 1992; Pennington & Ozonoff, 1996; Ross, et al., 1994).

One test frequently used to assess executive interference control in ADHD samples is the Stroop Color and Word task. Stroop tasks typify a class of interference whereby the introduction of task-irrelevant stimulus characteristics increases response time. Given that poor interference control has been highlighted as one of the core deficits in ADHD (Barkley, 1997), it is perhaps surprising that recent research with the Stroop task has failed to detect significant differences in interference levels (the Stroop effect) between ADHD and control samples. Indeed, evidence of increased Stroop interference in people with ADHD is equivocal. Although Barkley (1997), Harnishfeger and Bjorklund (1994), and Pennington and Ozonoff (1996) presented comprehensive reviews suggesting children with ADHD demonstrate greater Stroop interference than typical children, more recent studies have found equivalent Stroop effects across ADHD and control samples, (see Nigg, 2001, for a review), results that clearly conflict with the inhibition deficit hypothesis of ADHD. Two potential explanations behind these empirical discrepancies, and those most relevant to any assessment of Barkley's disinhibition model, concern the method of calculating the Stroop interference score and the issue of comorbidity in ADHD.

First, in regard to the former of these two factors, earlier studies reporting greater Stroop interference in the incongruent color-word condition for ADHD participants than for controls have often failed to control for rapid naming deficiencies that have since been reported for these people (cf. Rucklidge & Tannock, 2002; Tannock, Martinussen, & Frijters, 2000). The standard Stroop Color and Word task (Golden 1978) assesses response latencies across three conditions: time taken to name color hues of neutral stimuli (color condition), color words (word condition), and color hues of incongruent color-words (color-word condition). One approach to calculating interference in ADHD research has been to compare naming latencies on all three variables between ADHD and control groups, with any difference between the two groups on the color-word score taken to indicate variance in interference control. Because participants with ADHD typically demonstrate slower naming on all three variables, comparisons between ADHD and control groups on the color-word condition tend to suggest greater interference in the former group than in the latter. When Stroop interference scores are evaluated in between group studies, unless naming speed is controlled for, it is unclear whether heightened interference scores observed in any particular group reflect a problem with interference control or merely impairments in rapid naming. Ambiguities regarding Stroop interference effects in the ADHD literature prompted a meta-analysis of studies conducted after 1990 (van Mourick, Oosterlaan, & Sergeant, 2005). The authors concluded that the method of calculating the interference score was crucial to the interpretation of results.

Methods used to calculate the interference score should control for overall speed of naming. There were two acceptable measures, the classical method (Hammes, 1971) and the Golden Measure (Golden, 1978). The former controls for overall color naming speed by subtracting the score derived from the color-word (CW) condition from the score on the color (C) condition. Because those with ADHD are slower on both conditions than typical participants, van Mourick et al. (2005) found that the Stroop effect did not distinguish ADHD groups from other clinical or typical groups across studies when the C-CW interference measure was used. The Golden Measure, which also controls for word reading in addition to color naming speed, was found to be more likely to differentiate between ADHD and typical groups than the C-CW measure. In this method the interference score is calculated by subtracting a CW score that is predicted for each participant (i.e., using either a regression or theoretical formula; see Golden, 1978) from his or her uncorrected raw CW score. The higher the resultant score, the less susceptible the participant to interference. Their final conclusion was that overall the Stroop Color and Word task did not provide strong evidence for a specific interference control deficit in populations with ADHD. One goal central to the current article was to confirm previous findings of equivalent levels of Stroop interference between ADHD and typical groups when naming speed is taken into account using an independent adolescent sample. If confirmed, this finding would indicate that caution is needed in evaluating results with ADHD samples from Stroop-like paradigms, and that contrary to popular belief, deficits in the three key constituents of response inhibition may not affect levels of interference in the Stroop task.

NP Effects with ADHD Populations: Discrepant Findings

Another primary objective was to use an alternative method to further assess interference control in response inhibition in ADHD adolescents. To this end, we used NP manipulations to determine whether cognitive or central inhibitory processes levelled at interference control are impaired, as implied by the disinhibition model of ADHD. Studies of people with ADHD have focused almost exclusively on deficits relating to executive inhibition. Little mention is ever made of more automatic inhibitory processes in this literature. In our view, NP reflects such an automatic inhibitory process in the sense that it can be an emergent byproduct of focusing on and responding to a target stimulus in the presence of a conflicting distractor stimulus. Importantly, the degree of competition induced by irrelevant distractors must exceed a critical threshold and be sustained throughout a given task for cognitive inhibitory control to be applied consistently and strongly enough to produce reliable NP effects (Neumann & DeSchepper, 1991, 1992; Pritchard & Neumann, 2004).

Results from the few studies that have investigated NP effects in children and adolescents with ADHD are contradictory. Given that much of the contemporary research on ADHD points to a widespread deficit in inhibitory function, researchers using the NP paradigm to investigate central or cognitive inhibition in this population have predicted, not unreasonably, that a deficit in this inhibitory process may also underlie the symptoms of ADHD. Therefore, in comparison to typical people, those with ADHD should demonstrate a lower NP effect. Three of the five studies investigating NP effects in people with ADHD between 8 and 20 years of age support this hypothesis (cf. Marriott, 1998; Ossman, & Mulligan, 2003; Ozonoff, Strayer, McMahon, & Filloux, 1998). However, studies by Gaultney, Kipp, Weinstein, and McNeill (1999) and Pritchard, Healey, and Neumann (2006) found significant and equivalent NP effects for people with ADHD and their controls in this age range.

Using an NP variant of the Stroop task, Gaultney et al. (1999) assessed NP effects for both people with ADHD and typical people. Because Gaultney and colleagues were also interested in assessing the effect of stimulant medication on NP effects, those with ADHD were required to complete two administrations of the Stroop NP task: one session while medicated and one session while unmedicated. These sessions were counterbalanced. Like Marriott (1998) and Ozonoff et al. (1998), Gaultney et al. predicted that people with ADHD would demonstrate a diminished NP effect. These authors also predicted that NP effects for participants with ADHD would increase when they were medicated. Neither hypothesis was supported. NP effects were invariant across the two sessions for participants with ADHD. More critically, although Gaultney et al. found that participants with ADHD took longer to respond on both UR and IR priming conditions than controls, the significant NP did not differ significantly between the two groups. In contrast, studies using letters (Marriott, 1998; Ozonoff et al., 1998) or monosyllabic nouns (Ossman & Mulligan, 2003) as target and distractor stimuli to compare NP effects between people with and without ADHD found NP effects to reach statistical significance only for those without ADHD. The effects of stimulant medication on NP levels for participants with ADHD did not appear to moderate NP performance for the latter two studies. Ozonoff et al. reported no difference in NP effects between medicated and nonmedicated participants with ADHD, whereas participants in Ossman and Mulligan's ADHD group were unmedicated on the day of testing. Marriott did not provide details concerning medication.

However, Pritchard et al. (2006) reported results that are consistent with those of Gaultney et al. (1999). They used an NP variant of the Stroop task to compare potential inhibitory processing differences between children with ADHD and typical children between 10 and 12 years of age. Their experiment followed a double-blind procedure and all participants in the ADHD group were unmedicated on the day of testing. Statistically significant NP effects were obtained for both children with ADHD and controls, with no difference between the two groups. These authors concluded that cognitive inhibitory capacity is intact in children with ADHD and that the "inhibitory" deficit in ADHD does not appear to extend to the mechanisms of cognitive inhibition underlying NP.

However, like Gaultney et al. (1999), Pritchard et al. (2006) found that in comparison to typical children, children with ADHD demonstrated significantly longer overall response times. Gaultney et al. suggest that this impairment may relate to a fatigue effect for participants with ADHD; that is, attending to selective stimuli may be more effortful for children with ADHD than for those without. However, Rucklidge and Tannock (2002) suggested that it is more likely that the greater overall response times noted in adolescents with ADHD relates to an overall slowness in information processing and name retrieval rather than interference effects associated with distractor intrusion.

In the present study, we used a Stroop interference task and two conceptual (i.e., semantic or identity) NP tasks to assess susceptibility to interference and question the hypothesized dysfunctions in the cognitive inhibitory component of selective attention in participants with ADHD, compared to a similar-aged control group. Each experiment involved the same two groups: one of typical adolescents and the other of adolescents with a formal diagnosis of ADHD.

ADHD tends to present with other comorbid psychiatric conditions such as anxiety disorder, conduct disorder, mood disorder, and oppositional defiant disorder (Angold, Costello, & Erkanli, 1999). Because there is some evidence that comorbidity in ADHD can influence interference and inhibitory control in Stroop (Lufi, Cohen, & Parish-Plass, 1990) and go/no-go paradigms (see Nigg, 2001, for a review), our ADHD sample contained participants *without* a comorbid diagnosis. We were specifically interested in determining whether a “pure” ADHD group would produce Stroop interference or NP effects comparable to those of a control group. The presence of comorbidity in an ADHD sample in comparison to a control sample may mask potential differences in executive or cognitive inhibitory processes tied to interference control that are specific to ADHD. Given the ambiguity of the empirical situation concerned with these two distinct types of inhibition in ADHD research, our primary goal was to assess the hypothesized deficit in inhibition proposed to underlie this disorder while avoiding potential confounds relating to comorbid diagnoses in ADHD.

Experiment 1 assessed Stroop interference effects for adolescents with ADHD while controlling for naming speed. Experiment 2 was designed as an extension of Pritchard et al.'s earlier Stroop NP ADHD study which involved younger children. The third experiment was carried out in order to determine whether NP effects in adolescents with ADHD are stimulus specific. In other words, do NP effects in these adolescents go beyond the types of Stroop NP stimuli that were used in the studies by Gaultney et al. (1999) and Pritchard et al. (2006)? Given the ambiguity caused by empirical discrepancies concerning NP effects in ADHD samples, the specific purpose of the latter two experiments was to provide additional findings that might help to clarify and resolve the current inconsistencies.

4.3 General Procedure

The interviews and tasks were carried out in the laboratories at the Department of Psychology at the University of Canterbury. The local institutional review board approved the study and written informed consent and assent (for children under the age of 16) were obtained from adolescents and their parents, respectively. Registered clinical psychologists conducted all psychiatric interviews with those referred to the clinical group. Graduate students in clinical psychology conducted all interviews with the Control group under the supervision of a clinical psychologist. Ten percent of the interviews were videotaped and reviewed by the same doctorate level clinical psychologist. Interrater reliability for agreement on presence or absence of diagnosis was 100%. All participants were reimbursed $ 20 (N.Z) for the costs of parking and lunch.

Participants who were receiving psychostimulant (dextroamphetamine or methylphenidate) medication (9 participants, or 56.3% of the ADHD group) discontinued this treatment 24 hours before the day of testing because of the known effects of methylphenidate on cognitive functioning (see Berman, Douglas, & Barr, 1999). Asking children not to take their medication on the morning of the testing is a procedure commonly used in ADHD research to determine specific deficits involved in the disorder, deficits that may be masked by medications (Rucklidge & Tannock, 2002; Clark, Prior, & Kinsella, 2000). On the day of testing, it was confirmed with parents that the children had not been given their ADHD medication that morning. Because methylphenidate has an approximate half-life of 4.5 hours (Shader, et al., 1999), a 24-hour elimination period should have ensured that the majority of the active ingredient had been eliminated before testing. Three (18.8%) of the ADHD group were taking a medication other than a stimulant (e.g., fluoxetine, paroxetine). One of the controls (2.2%) was taking paroxetine. These other medications were not discontinued.

4.4 Experiment 1

4.5 Method

Participants. A total of 61 adolescents (aged 13 to 17 years) were included in this experiment: 16 ADHD (7 male, 9 female), and 45 Controls (19 male, 26 female). Forty-one (91.1%) of the Control group and 13 (81.3%) of the ADHD group were New Zealanders of European ancestry. Two participants from the ADHD group were Maori. The remaining participants were “Other” European. Thirty-six (80%) of the Control group and 11 (68.75%) of the ADHD group were from intact families. The ADHD group was recruited from people who were previously assessed at a specialized mental health center that serves those with moderate to severe psychiatric disorders. Adolescents in the Control group were recruited through advertising at local schools and community resources. Sample characteristics are provided in Table 1 (see pg. 92).

Given our small sample size there was some potential for limitations relating to adequate statistical power to detect between group differences. We address these issues in detail, presenting a series of supplementary power and effect size (ES) computations performed for all experiments in the *Results* section of Experiment 3.

Diagnostic Protocol for ADHD and other psychiatric disorders. Systematic information about current and lifetime disorders was obtained from both the participant and the parent separately using the Schedule for Affective Disorders and Schizophrenia for School–Age Children-Present and Lifetime Version (K-SADS-PL), an interview that generates both DSM-IIIR and DSM-IV diagnoses. This semistructured interview has been used extensively to make diagnostic decisions based on DSM criteria and has been validated with children and adolescents 6 to 17 years of age (Kaufman, et al., 1997). The long versions of the Conners' Rating Scales-Revised (Conners, 1997) were used to assess ADHD and for inclusion and exclusion criteria. This instrument provides separate rating forms for parents, teachers, and adolescents.

Inclusion criteria for ADHD. To assess for presence of ADHD, the following diagnostic algorithm was used: 1) the participant met DSM-IV criteria for ADHD according to the clinician summary based on the K-SADS parent and adolescent interview; participants attending school regularly (n=10) met the clinical cutoffs for the externalizing symptoms of ADHD on the Teacher Report Form (TRF; Achenbach, 1991) or the Conners' teacher questionnaires in order to ensure pervasiveness of symptoms across settings; and the participant showed evidence of ADHD symptoms before the age of 7, established either through a past diagnosis of ADHD or in new cases, according to parental report, medical records and past school report cards. Impairment was confirmed using the K-SADS. The presence or absence of DSM-IV internalizing disorders was based on a clinician summary derived from the information gathered from the parent and adolescent K-SADS interview. Note that information from the adolescent K-SADS did not supersede the parental report for the presence or absence of externalizing symptoms.

According to this diagnostic protocol, 3 (18.8%) of the ADHD group were identified as Combined Type, 2 (12.5%) as Predominantly Hyperactive/Impulsive Type and 11 (68.8%) as Predominantly Inattentive Type.

Inclusion criteria for the control group. Only adolescents who did not meet ADHD criteria according to the K-SADS-PL and had T scores below 65 on the ADHD subscales on either the parent or teacher forms of the Conners' questionnaire were included in this group. Eight controls were excluded because of high scores on the parent or teacher Conners' questionnaire.

Exclusion criteria for all groups. Participants were excluded from analyses if they had: 1) an estimated IQ below 75, using the Block Design and Vocabulary subtests of the Weschler Adult Intelligence Scale-III (WAIS-III; Wechsler, 1997) or the Wechsler Intelligence Scale for Children-111 (WISC-III; Wechsler, 1991), a combination of subtests commonly used to estimate full scale IQ. Those with uncorrected problems in vision or hearing, serious medical problems (e.g., epilepsy or cerebral palsy) or serious psychopathology such as psychosis that would preclude a current differential diagnosis of ADHD were excluded from both groups.

Participants were also excluded if they had a comorbid diagnosis (e.g., Oppositional Defiant Disorder, Conduct Disorder, Major Depressive Disorder). All adolescents participating in the study were native English speakers. These criteria resulted in the exclusion of 6 participants because of low IQ (2 Control, 4 ADHD) and 32 (4 control, 28 ADHD) because of comorbid diagnoses. In addition, two were excluded because they were color blind and another two for missing data on the NP tasks.

Measures of demographic variables. The New Zealand Socioeconomic Index of Occupational Status (NZSEI; Davis, McLeod, Ransom, & Ongley, 1997) was used as a measure of socioeconomic status. This index assigns occupations a score from 20 (low socio-economic status) to 90 (high socio-economic status). Highest education level achieved by each parent (from 1, *no high school*, to 6, *university degree*) was also used as a measure of economic status.

Stimuli and apparatus. The Stroop Color and Word Naming test (Golden, 1978) was used. This test yields four dependent measures: Number of color words (red, blue, or green) named in 45 s, number of colors (red, blue, or green) named in 45 s, number of color names that are printed in a discordant color word named within 45 s (e.g., when the word *red* is written in green ink, the participant must respond with "green"), and an interference estimate that measures the ability to suppress a habitual response in favor of an unusual one, taking into account overall speed of naming. The stimuli were presented on 26 x 18 cm cards. There was one card per condition. On each card all Stroop items were arranged as five vertical columns. Letters measured 1.0 cm in height with each Stroop item placed at 1.0 cm intervals down each column. The method used to calculate interference scores was the Golden Measure (Golden, 1978). This method controls for both color naming speed and word reading and is more likely to distinguish ADHD samples from typical samples than the classical C-CW method (Appendix E provides details of the Golden [1978] procedure for calculating interference scores for children of varying ages). A stopwatch was used to indicate temporal duration.

Procedure. Participants were tested individually in a quiet room in one session of approximately 15 min. The experiment was administered according to the specifications of the Stroop Color and Word Naming test (Golden, 1978). Participants were first presented with the color word condition, followed by the color naming condition, followed by the condition containing color names printed in a discordant color word. For each condition, participants were told to begin naming on the command “Begin” and to continue naming until the command “Stop” was given. They were told to name each item from the top to the bottom of each column on each card as quickly as possible. It was also specified that any errors made during naming were to be self-corrected. Participants who failed to self-correct were alerted to this by the word “No.” The stopwatch was started in synchrony with the word “Begin” and stopped after 45-s duration. The number of words or colors named within the 45-s period was recorded for each participant.

4.6 Results

Sample characteristics. There were no group differences in sex distribution (_2 (1, *N* = 61) = .447, *ns*). According to Table 1, there were group differences in IQ and father's level of education: the controls had higher IQs than the ADHD group, and the fathers of the control group were more educated than the fathers of the ADHD group. As expected, there were group differences on all Conners’ scales.

Table 1

Sample Characteristics: Means and Standard Deviations

Variable	Controls (n=45)		ADHD (n=16)		F (1, 59)
	Mean	SD	Mean	SD	
Estimated IQ	108.38	13.19	98.25	11.90	7.306**
Age	15.01	1.35	15.53	1.12	1.883
Mother's education	4.45	2.001	5.10	1.52	.911
Father's education	4.68	1.93	4.5	1.96	.072
NZSEI	58.93	16.17	68.60	15.31	2.966
CPRS-R (T scores)					
DSM In	47.36	6.59	75.06	12.60	125.28***
DSM H/I	48.76	6.24	76.44	12.12	136.26***
CSRS-R (T scores)					
DSM In	44.16	9.15	59.00	13.16	24.44***
DSM H/I	42.16	8.88	53.69	12.46	15.975***
CTRS-R (T scores)†					
DSM In	45.59	13.43	62.00	13.90	9.169**
DSM H/I	45.24	12.45	63.70	17.13	10.484**

Note: NZSEI = New Zealand Socioeconomic Index of Occupational Status, CPRS = Conners Parent Rating Scale CSRS = Conners-Wells' Self-report Scale, CTRS = Conners Teacher Rating Scale, *p < .05, **p < .01, ***p < .001, † based on the teacher reports that were returned (i.e., for 19 controls and 15 ADHD participants).

Naming speed. Table 2 displays the mean color naming scores and standard deviations for the four Stroop measures in Experiment 1. Mean color naming scores for the Word, Color, and Color-Word measures were analyzed using a mixed analysis of variance (ANOVA). The three Stroop naming measures (word, color, and color-word) were treated as the within-subjects variable and group (control vs. ADHD) as the between-subjects variable. Comparison of the interference score between groups was assessed via a t-test for independent samples with a significance level set at .01. Results with naming measures revealed a significant main effect of group. The ADHD group demonstrated a significantly longer overall naming latency than the control group, F (1, 59) = 10.39, p < .01. However, there was no hint of an interaction between color naming scores and Group, F < 1, suggesting that the two groups did not differ in interference effects. To confirm our findings, a comparison of the interference score between groups was assessed via a *t* test for independent samples, with a significance level set at .01. Results suggested that Stroop interference was invariant between groups, t (59) = -.23, p > .82. The mean Stroop interference estimate was 7.96 for the Control group and 8.50 for the ADHD group.

Table 2

Mean Number of Target Items Named within 45 Seconds and Estimated Interference Scores for the Control and ADHD Groups in Experiment 1

	Group			
	Control		ADHD	
Variable	Mean	SD	Mean	SD
Stroop Test (raw scores)				
Word	92.56	11.55	79.88	12.27
Color	71.96	12.82	61.50	10.17
Color-Word	47.22	12.17	40.43	7.37
Interference	7.96	7.26	8.50	10.23

4.7 Discussion

Results from Experiment 1 suggest that attentional processing in ADHD populations is not unduly directed toward irrelevant information. Although participants with ADHD demonstrated greater overall naming latencies than control participants, there was no significant difference between the two groups on the interference estimate. An equivalent amount of Stroop interference between the ADHD and Control groups implies that adolescents with ADHD do not appear to exhibit a deficit in the ability to control prepotent response tendencies. The analysis of the data in the Stroop Color and Word test suggests that not all aspects of executive inhibition are affected in ADHD. Compared with controls, the ADHD group demonstrated an overall impairment in naming speed, but once this slowed naming was taken into consideration, there was no additional Stroop interference impairment. The processes ultimately enabling Stroop interference resolution thus appear to be intact in the

ADHD adolescents, regardless of comorbidity, and functioning in a manner that is the same as in the Control adolescents. We defer a more detailed discussion of this analysis to the *General Discussion*.

4.7 Experiment 2

4.8 Method

Participants. The participants were the same as those in Experiment 1.

Design. A mixed design was used. The between-subjects variable was group (ADHD vs. control) and the within-subjects variable was priming condition (UR vs. IR). Trials consisted of 50% UR control trials (in which neither the hue nor distractor color word in a Stroop NP stimulus was related to the subsequent Stroop NP stimulus), and 50% IR trials (in which the distractor word in the previous Stroop NP stimulus named the subsequent target hue).

Stimuli and apparatus. The stimuli were presented on 26 x 18 cm cards and consisted of the words WHITE, RED, BLUE, ORANGE, GREEN, PINK, BROWN, BLACK, GRAY, YELLOW, and PURPLE. On each UR and IR card all color words were arranged as a single vertical column against a light gray background with each word printed in one of the 11 corresponding colors, with the constraint that the ink color and word were incongruent (see Appendix A). Each Stroop item measured 1.0 cm in height with each display spaced at 1.0 cm intervals down the list. The first two items on each IR card were unrelated in order to reduce the potential saliency of this condition. The 12 cards used in the experiment consisted of 6 UR cards and 6 IR cards. Four additional UR cards were used for practice trials. Presentation orders in the experiment were counterbalanced so that half of the participants began with an IR card and the remaining half with a UR card. Subsequent cards were presented in regular alternation of the two conditions. A stopwatch was used to record the time taken to complete color naming for each card.

Procedure. Experiment 2 followed a double-blind procedure. All participants completed a preliminary color identification task to ensure familiarity with the 11 color hues used in the experiment. No participants reported any difficulty with this task. Before the experimental cards were administered, each participant encountered four UR practice cards. They were told to name as quickly and accurately as possible the ink color of each color word from the top to the bottom of the column on each card. They were also asked not to cease color naming if an error was made, but rather continue to complete the card. Participants were then given the 12 experimental cards (6 per priming condition presented in alternation). Each card was covered with a blank sheet that was removed by the experimenter on the word "Go". The stopwatch was started with the removal of the blank sheet and stopped at the naming of the last color hue on a card (see Appendix C for administration details for the Stroop NP task). Error scores for each card were tabulated. Errors were defined as either omissions or verbalizations of an incorrect or absent color.

4.9 Results

Reaction time. A mean RT per Stroop item for each participant was calculated for the six cards representing the UR condition and the six cards representing the IR condition. Table 3 presents mean RTs and percentage of errors for each group and the priming conditions in Experiment 2. Mean RTs were submitted to a mixed ANOVA treating priming condition (UR vs. IR) as the within-subjects variable and group (control vs. ADHD) as the between-subjects variable. A significant main effect of group indicated that the ADHD group responded significantly more slowly than the Control group, F (1, 59) = 9.52, $p < .01$. In addition, there was a significant NP effect, F (1, 59) = 7.00, $p < .01$. RTs were longer on IR trials than on UR trials. More importantly, the interaction between priming condition and group did not approach statistical significance, $F < 1$. The mean NP effect per item was 55 ms for the Control group and 39 ms for the ADHD group. Thus, although participants in the ADHD group were slower to name colors in both the UR and IR conditions, their NP effect was similar in magnitude to that of the Control group.

Table 3

Mean Reaction Time (in Milliseconds) per Item and Percentage of Errors for Control and ADHD Groups as a Function of Priming Condition in Experiment 2

	Group			
	Control		ADHD	
Priming condition	UR	IR	UR	IR
Reaction time				
M	900	955	1127	1166
SD	215	216	323	330
ER (%)	2.0	1.8	1.8	1.4

Errors. Similar analyses were conducted with error scores. No significant effects were found, all p's > .05. Thus, the error data do not compromise the interpretation of the RT results.

4.9.1 Discussion

The main objective of Experiment 2 was to resolve ambiguous findings concerning conceptual NP in children and adolescents with ADHD obtained from previous studies. Our Stroop NP task has provided a clear demonstration of intact NP effects in both typical and atypical adolescent samples. Furthermore, levels of NP appear to be similar between the two samples, suggesting that central or cognitive inhibitory capacity may be distinct from other inhibitory processes implicated in the reduced cognitive performance evidenced in ADHD symptoms. These findings lend credibility to previous findings of significant NP effects in people with ADHD obtained by Gaultney et al. (1999) and Pritchard et al. (2006). On the other hand, they contradict reported failures to obtain NP in people with ADHD (i.e., Marriott, 1998; Ossman & Mulligan, 2003; Ozonoff et al., 1998). Overall, the results from Experiment 2 provide evidence for an intact inhibitory mechanism of visual selective attention in participants with ADHD. The next experiment was designed to further examine these issues in the context of a flanker NP task involving identity, rather than semantic, relationships between a prior distractor and current target.

4.9.2 Experiment 3

Experiment 2 demonstrated that NP effects in participants with ADHD are replicable. However, it is noteworthy, that NP effects for children and adolescents with a formal diagnosis of ADHD thus far have been found only in the context of Stroop NP tasks. In contrast, the studies by Marriott (1998) and Ozonoff et al. (1998) both used a letter identification NP task. Ossman and Mulligan's (2003) NP task used monosyllabic, five-letter nouns as target and distractor stimuli. These three studies failed to find significant NP effects with people with ADHD. Therefore, it is not clear whether NP effects in these people can generalize to a wider range of stimuli and task demands. In order to gain greater confidence in our results concerning ADHD and NP, Experiment 3 aimed to investigate NP effects in adolescents with ADHD using different stimuli in a different task. In the present flanker NP task, the stimuli consisted of a central target color blob flanked on both sides by nontarget incongruently colored blobs. Although this task has previously produced significant NP effects in typical children aged 5 to 12 years (Pritchard & Neumann, 2004), it is not known whether these effects would also be found in individuals diagnosed with ADHD.

Using conflicting target and distractor color blobs, rather than incongruent color word stimuli, avoids the prepotent-alternative response dynamic inherent in the Stroop paradigm. Previous findings of NP effects in people with ADHD engaged in a Stroop NP task, but not in a letter identification task, suggests that inhibitory mechanisms in these people may be triggered only in situations where distractor interference is from a prepotent distractor. Finding significant NP effects in typical participants across a range of stimuli other than Stroop items implies that prepotency does not have to be the driving force of suppression. Experiment 3 attempted to determine whether an active inhibitory process can be demonstrated in people with ADHD in situations where representation of relevant and irrelevant stimuli are likely to initially receive equivalent amounts of processing, but without the distractor necessarily incurring a prepotent response.

4.9.3 Method

Participants. The participants were the same as those in the first two experiments.

Design. A mixed design was used. The between-subjects variable was group (ADHD vs. control) and the within-subjects variable was priming condition (UR vs. IR). Trials consisted of 50% UR (in which there was no relationship between the colors of distractor blobs in the previous display and the color of the subsequent target blob color), and 50% IR (in which the color of the distractor blobs in the previous display matched the subsequent target color blob).

Stimuli and apparatus. The stimuli consisted of 11 unique sets of three-different shaped color blobs presented in a column on twelve 32 x 22 cm manila cards. The sequential arrangement of rows differed for each card. In addition, each row was randomly staggered to either the left or right in an attempt to reduce the saliency of the IR condition. Visual distances between individual blob rows were the same for both UR and IR cards. The outer blobs in each row functioned as distractors, and the center blob functioned as the target. The 11 colors used in Experiment 2 were used for blobs in Experiment 3. The color for the target blob always differed from the color shared by the flanking distractor blobs (see Appendix B). Six UR cards and 6 IR cards were used in the experiment. Four additional UR cards were constructed and used for practice trials for the reasons given in Experiment 2. Similarly, presentation orders were handled as in Experiment 2. A stopwatch was used to record the time taken to complete color naming for each card.

Procedure. After the initial color identification task, participants were given verbal instructions for the color blob task. They were told to name as quickly and accurately as possible the color of each central blob while ignoring the outer blobs, from the top to the bottom of the column on a given card. Again, it was emphasized that they should not cease color naming if an error was made but rather complete color naming for the card (see Appendix D for administration details for the colour-blob NP task). After completing the 4 practice UR cards, participants were given the 12 experimental cards (6 per priming condition presented in alternation). The timing procedure was handled as in Experiment 2. Error scores for each card were recorded.

4.9.4 Results

Reaction time. Mean RTs per item and the percentages of errors for the UR and IR priming conditions are shown for the ADHD and control groups in Table 4. Mean RTs were entered into a mixed ANOVA. The between-subjects factor was group (control vs. ADHD) and the within-subjects variable was priming condition (UR vs. IR). There was a significant main effect of group, with the ADHD group taking significantly longer to respond than the Control group, F (1, 59) = 7.76, $p < .01$. More critically, there was a significant NP effect, F (1, 59) = 6.27, $p < .02$, with no significant interaction between priming condition and group, $F < 1$. The mean NP effect per item was 27 ms for the Control group and 45 ms for the ADHD group.

Errors. Error scores were submitted to similar analyses, but yielded no significant effects (all p's > .05).

Table 4

Mean Reaction Time (in Milliseconds) per Item and Percentage of Errors for Control and ADHD Groups as a Function of Priming Condition in Experiment 3

	Group			
	Control		ADHD	
Priming condition	UR	IR	UR	IR
Reaction time				
M	607	634	725	770
SD	144	130	221	230
ER (%)	.60	.84	.81	.63

4.9.5 Supplementary Analyses

Statistical Power Analyses. The high tendency of ADHD to present with comorbid diagnoses and the inherent difficulties in acquiring ADHD samples resulted in a smaller sample size for the ADHD group than for the control group. The consequence of this may be that our experiments lacked sufficient statistical power to detect between-group differences on both Stroop interference and NP scores. Issues relating to low statistical power and uneven sample size are a common problem in clinical research and plague the majority of studies assessing interference control and executive and cognitive inhibition in ADHD (cf. Barkley, 1997; Murphy, Barkley, & Bush, 2001; van Mourick et al., 2005). To further evaluate this issue, a compromise power analysis[9] (Buchner, Erdfelder, & Faul, 1997; Erdfelder, Faul, & Buchner, 1996) was performed for each experiment using the mean Stroop interference estimates (Experiment 1) for each group, and the mean NP effect (Experiments 2 and 3) for each group. The beta/alpha ratio q was set to 1 ($\beta/\alpha = 1$) in order to specify the relative seriousness of both errors and the total sample size was $N = 61$ for all the following analyses.

For Experiment 1, effects of size (cf. Cohen, 1977) $d = .54$ could be detected with the probability ($\beta/\alpha = 1$) = .82 for the group related difference in the Stroop interference estimate. For Experiment 2, effects of size $d = 0.18$ could be detected with the probability of ($\beta/\alpha = 1$) of .62 for the group related difference in the NP effects. For Experiment 3, effects of size $d = 0.20$ could be detected with the probability ($\beta/\alpha = 1$) of .63 for the group-related differences in the NP effect.

Effect Size Analyses. To counteract potential limitations arising from low statistical power, we conducted further analyses independent of sample size to determine the ES for both the Stroop and NP effect for each group using Cohen's d. For Experiment 1, the mean color naming scores for the Color and Color-Word measures were used to assess the ES of the interference score for each group. The ES for the Stroop effect was similar for the two groups, $d = 1.9$ for the control group and $d = 1.6$ for the ADHD group. For Experiments 2 and 3, mean

[9] The power calculations were conducted using the G*Power program (Buchner, Erdfelder, & Faul, 1997; Erdfelder, Faul, & Buchner, 1996). The compromise analysis differs from the post hoc analysis in that it enables an equal beta/alpha ratio to specify the relative seriousness of both errors in our experiments. Its use is specifically designed for situations in which work with clinical samples produces an N too small to satisfy conventional levels of alpha and beta given the effect size.

RT data from the two priming conditions (UR versus IR) were combined to determine the ES of the NP effect for each of the two groups. The ES for the NP effect was virtually equivalent for the two groups, d = .23 for the control group and d = .24 for the ADHD group. Although the ESs for both the Stroop effect and the NP effect were similar for the two groups, the numerical difference on the ES scores for these effects between the groups suggests a trend toward lower Stroop interference and greater NP for the ADHD group than for the control group, despite the much smaller sample size of the ADHD group in comparison to the control group.

4.9.6 Discussion

The main objective of Experiment 3 was to test for the potential generalizability of the NP phenomenon in ADHD populations. The results revealed a pattern of performance that was analogous to the pattern observed in Experiment 2 with very different stimuli. The significant and equivalent magnitudes of NP obtained across the control and ADHD "pure" groups imply that NP effects are similar between those with and without ADHD. Moreover, the NP effect for participants with ADHD emerges, as it does for similarly aged controls, in tasks where response tendencies associated with distractor and target are likely to be equipotent. Distractor prepotency is not a critical prerequisite of distractor suppression for adolescents with ADHD. To our knowledge, this is the first demonstration of significant NP effects in ADHD populations for stimuli other than NP Stroop items. Thus, it seems clear that the central or cognitive inhibitory mechanism underpinning NP effects in adolescents with ADHD is neither task nor stimuli specific.

4.9.7 General Discussion

The specific purpose of this study was to explore potential differences in Stroop interference and NP effects between adolescents with ADHD and controls. We were particularly interested in assessing the general applicability of the inhibitory deficit model of ADHD on executive and cognitive forms of inhibition with a view toward clarifying previous discrepancies in ADHD Stroop and NP literatures. To examine potential inhibitory deficits specific to ADHD, all participants with a corresponding psychiatric diagnosis other than ADHD were excluded from our experiments. As an additional precaution, stimulant medication was withdrawn from use 24 hours before testing. Experiment 1 showed that, when overall naming speed is controlled, Stroop interference effects are similar between ADHD and similarly aged Controls. Experiment 2 demonstrated that semantic NP effects are a replicable phenomenon in participants with ADHD. Furthermore, the NP effect for the ADHD group in this experiment did not differ significantly from the effect obtained for the Control group. Experiment 3 corroborated these findings and showed that NP for participants with ADHD is a generalizable effect, not limited to Stroop NP tasks. In the remaining sections we consider how these results bear on the disinhibition hypothesis of ADHD and attempt to identify potential reasons for the discrepancies in the ADHD NP literature.

Implications for the inhibition deficit hypothesis of ADHD

The general consensus in the clinical and developmental literature on ADHD has been that the inattention, impulsivity, and hyperactivity characteristics of ADHD closely relate to a deficit in resistance to interference and capacity for inhibition. Our findings of equivalent measures of Stroop interference between adolescents with and without ADHD and equivalent and intact levels of cognitive or central inhibition in these individuals challenge the above view on a number of levels.

Stroop interference is widely held as a measure of inhibitory response or control in the study of individual differences in attentional processing across various typical and atypical populations (Dempster, 1991; Mirsky, Anthony, Duncan, Ahearn, & Kellam, 1991; Seidman, Biederman, Monuteaux, Weber, & Faraone, 2000). Heightened levels of interference are taken to indicate deficits in executive inhibition or the ability to control or suppress the irrelevant reading process. A number of studies report increased Stroop interference in individuals with prefrontal pathology (e.g., see Stuss & Benson, 1984). Similar findings are commonly reported in the ADHD literature. In comparison to typical individuals, those with ADHD have been reported to demonstrate greater susceptibility to intrusion from the color word (Seidman, Biederman, Faraone, Weber, & Ouellette, 1997), data that fit nicely with the classification of ADHD as a predominantly prefrontal disorder. However, results from the current study and those obtained by Rucklidge and Tannock (2002) and van Mourick et al. (2005) regarding the issue of Stroop interference in people with ADHD are now calling into question the clarity of the relationship between poor executive interference control or response inhibition and ADHD. These results show that although the ADHD group demonstrated a significantly longer overall RT than the control group, the interference estimates did not differ between the groups when overall speed of naming is taken into account. Rucklidge and Tannock suggested that longer RTs in color naming (in the absence of a conflicting word) for people with ADHD may be linked to a color naming deficit and slower processing rather than greater interference specific to the incongruent Stroop condition.

Rucklidge and Tannock (2002) cite a number of studies linking color naming deficits with ADHD (cf. Brock & Knapp, 1996; Carte, Nigg, & Hinshaw, 1996; Nigg, Hinshaw, Carte, & Treuting, 1998; Semrud-Clikeman, Guy, Griffin, & Hynd, 2000; Tannock, Martinussen, & Fritjers, 2000). Reports such as these, in combination with the finding of equivalent amounts of Stroop interference between ADHD and controls in the current study and in that of Rucklidge and Tannock, suggest that caution should be exercised when attributing longer color naming RTs in people with ADHD to greater susceptibility to distractor intrusion and weakened inhibitory control. It appears that the ability to control interference is not inexorably affected in ADHD, at least not within the Stroop paradigm.

Our findings of intact cognitive inhibition in an ADHD sample place similar constraints on the notion of the disinhibition hypothesis of ADHD. As outlined earlier, inhibition is not a unitary construct. In terms of neural or cognitive processes it seems likely that multiple sources of inhibitory processing exist (Nigg, 2000). Although it is undeniable that people with ADHD show impairment across a range of tasks tapping a form of inhibitory control or response inhibition and executive motor inhibition, other inhibitory processes such as those underpinning NP effects appear to be spared in this disorder.

Evidence from neuroimaging and neuropsychological studies has suggested that the frontal deficit in ADHD is associated primarily with the prefrontal cortex, an area implicated in the mediation of various types of inhibition. Deficits in this region appear to account for poor performance on two tasks typically used to assess inhibitory function in people with ADHD: the stop signal task and the go/no-go task. In the stop signal task participants need to inhibit or interrupt a planned but not yet initiated response to a presented target stimulus when a stop signal, either auditory or visual, is presented directly after the onset of the target stimulus. In comparison to controls, those with ADHD exhibit longer RTs and a tendency to respond more often than controls on stop signal trials suggesting that the type of response inhibition tapped by the stop signal task may be implicated in the inhibitory psychopathology associated with this disorder. Similar findings in ADHD have also been found with the go/no-go task, a task that taps a type of inhibition similar to the stop signal task. The go/no-go task requires the execution or inhibition of a response to a stimulus in a series of sequential trials depending on whether the stimulus has been previously specified as a "go” stimulus or a "no-go” stimulus. Findings of impaired performance in the stop signal and go / no-go task are consistently reported in the ADHD literature (Aman, Roberts, & Pennington, 1998; Rubia et al., 1998; Schachar & Logan, 1990; Schachar, Tannock, Marriott, & Logan, 1995; Tannock, Schachar, Carr, Chajczyk, & Logan, 1989; Vaidya et al., 1998).

The types of control or response inhibition that are linked with the stop signal task and the go / no-go task are unlikely to relate to the central or cognitive inhibition that may underlie NP effects. Empirical evidence from two levels suggests that the stop signal task, go/no-go task, and NP tasks may index distinct types of inhibition. First, several studies investigating the effects of the psychostimulant medication methylphenidate on the performance of people with ADHD in the stop signal and go/no-go tasks have found this drug to modulate response inhibition in both tasks. In comparison to controls, those with ADHD show improvement in stopping performance when taking the dopamine agonist methylphenidate (Tannock et al., 1989; Tannock, Schachar, & Logan, 1995; Vaidya et al., 1998). No such effects have been found with NP effects (cf. Gaultney et al., 1999; Ozonoff et al., 1998). Stimulant medication does not appear to modulate NP effects, suggesting that this type of inhibition may not be implicated in prefrontal disorders.

Second, NP effects do not appear to correlate with other types of inhibition implicated in ADHD. For example, a study by Kramer, Humphrey, Larish, Logan, and Strayer (1994) found no correlation between NP effects and performance on the go / no-go task. Similar findings have been reported by Nigg, Butler, Huang-Pollock, and Henderson (2002). These authors compared the performance of adults with persistent childhood onset ADHD without psychiatric comorbidity on NP and antisaccade tasks. There is empirical evidence that symptoms associated with ADHD can persist into adulthood for a significant percentage of children diagnosed with this disorder (Mannuzza, Klein, Bessler, Malloy, & LaPadula, 1998; Hart et al., 1995), with ADHD now recognized as a valid adult diagnosis. Several studies have begun to report neuropsychological deficits in adult ADHD samples similar to those found in child ADHD samples (Corbett & Stanczak, 1999; Downey, Stelson, Pomerleau, & Giordani, 1997). Investigating the disinhibition hypothesis of ADHD with adult participants, Nigg et al. (2002) found that although adults with ADHD demonstrated a weakened ability to inhibit reflexive or anticipated oculomotor response on the antisaccade task, their scores on a Stroop NP task did not differ from those of typical controls. Data from this study suggested to Nigg et al. that deficits relating to inhibitory control in ADHD are associated primarily with problems in an executive inhibitory control system dependent on the prefrontal cortex. Similar to the conclusions drawn in this current study, further conclusions made by these authors were that this effect was limited to motor inhibition and appears to be independent from the type of cognitive suppression indexed by NP, and there is no evidence that a deficit in the type of inhibition underlying NP is associated with ADHD.

One potential explanation for the differences between the findings regarding inhibition in ADHD populations in the clinical or developmental literature and the NP literature is that the term *inhibition* tends to be used much more broadly in the former literature and often refers to phenomena that might involve mechanisms quite different from the inhibition described in the NP literature (which deals specifically with clashing targets and concurrent distractors in selective attention tasks). It is likely that a selective inhibitory mechanism is directly responsible for mediating conceptual NP effects. Its function is to suppress the mental representation of potentially distracting information, and it seems to be dedicated to inhibiting the severest competitor or competitors to a concurrent target, thereby producing a cost when such an item is presented again as a target (Neumann, Cherau, Hood, & Steinnagel, 1993).

The findings reported in this study, and those reported by Gaultney et al. (1999), Nigg et al. (2002), and Pritchard et al. (2006), demonstrate the importance of first isolating a specific form of inhibition when evaluating the disinhibition theory of ADHD, because there appear to be a variety of kinds of inhibition and not all are adversely affected in ADHD. As demonstrated here, the inhibitory deficit does not appear to extend to central or cognitive inhibition indexed by NP. The implication from this finding is that NP may reflect a specific type of cognitive inhibition that may operate independently of other processes deemed to play a pivotal role in prefrontal function.

Although it is largely agreed that the NP effect is the cognitive consequence of ignoring irrelevant information, there is less consensus on the precise mechanisms that underlie this effect. The two major theoretical accounts of NP effects are the memory retrieval and inhibition-based explanations. Proponents of the memory retrieval theory of the NP effect (Neill, 1997; Neill & Mathis, 1998; Neill & Valdes, 1992) suggest that NP arises from the retrieval of a memory trace containing response information that conflicts with current correct target selection. The distractor inhibition-based account of NP, on the other hand, holds that target selection is achieved via a competition-sensitive inhibitory mechanism functioning to reduce concurrent interference from distractor stimuli (Houghton & Tipper, 1994; Neumann & DeSchepper, 1992; Strayer & Grison, 1999). Determining the best theoretical model of NP has attracted much theoretical and empirical examination (i.e., the memory retrieval vs. inhibition debate). For the sake of brevity and because of the mounting evidence against the episodic retrieval account, in favor of an inhibitory account (e.g., Buchner & Mayr, 2004; Buchner & Steffens, 2001; Conway, 1999; Feuntes, Humphreys, Agis, Carmona, & Catena, 1998; Hughes & Jones, 2003; Khurana, 2000; Kramer & Strayer, 2001; Lavie & Fox, 2000; Neumann, McCloskey, & Felio, 1999; Strayer & Grison, 1999; Wong, 2000), our interpretation of NP effects is in terms of an inhibition account. For more detailed recent discussions of this issue, however, see Pritchard and Neumann (2004) and Tipper (2001).

An Intact Cognitive Inhibitory Mechanism in ADHD

NP tasks offer a unique opportunity to investigate dedicated inhibitory mechanisms involved in the selection of relevant over irrelevant material (Neumann et al., 1999). Findings of intact NP effects in children with ADHD (Gaultney et al., 1999; Pritchard et al., 2006), in the current adolescent ADHD sample, and in adults with ADHD (Nigg et al., 2002) converge to suggest that the inhibitory processes underlying NP develops early and is unaffected by ADHD symptoms, at least into early adulthood. These patterns of findings mirror those found with typical people in these age groups. Given the discrepancies that exist in the ADHD NP literature regarding the prevalence of this effect, however, it is suggested that NP effects in ADHD populations may be sensitive to variations in task design. Here it is of note that the monosyllabic noun NP task used by Ossman and Mulligan (2003) to investigate NP effects in adults with ADHD has also failed to produce significant NP effects in older adults (e.g., Kane, May, Hasher, Rahhal, & Stolzfus, 1997), despite the fact that it has been established (after a second meta analysis of this literature; see Gamboz, Russo, & Fox, 2002) that there are no differences in NP effects between younger and older adults. Although the lack of NP reported in some studies suggests a deficiency in an inhibitory mechanism in people with ADHD, the typically small size of the NP effect coupled with a propensity for greater variability in people with ADHD might help to explain the lack of NP effects in some of these studies. A similar situation may also hold for the letter identification NP tasks used by Marriott (1998) and Ozonoff et al. (1998) because such tasks are known for producing small NP effects, even in young typical adult samples. For a cogent discussion of how these issues apply more generally to atypical populations engaged in conceptual NP tasks, see Buchner and Mayr (2004). Clearly, these are important avenues for consideration in future research investigating potential individual differences in NP.

Finally, one other variable that should be considered in evaluating disparate findings in the ADHD NP literature relates to the issue of comorbidity in this disorder given that research by Lufi et al. (1990) and Nigg (2001) suggests that corresponding psychiatric diagnoses in ADHD may moderate certain levels of interference and inhibitory control. Of the previous studies investigating NP in ADHD only two have controlled for comorbidity (Nigg et al., 2002; Ossman & Mulligan, 2003). However, whereas Nigg et al. found NP effects to be independent of coinciding diagnoses in adults with ADHD, Ossman and Mulligan found no evidence for NP in adolescent ADHD participants without comorbidity. This area deserves further scrutiny because more subtle variables concerning developmental differences and NP task situations have been found to moderate NP effects (cf. Pritchard & Neumann, 2006;

Mason, Humphreys, & Kent, 2004). In a forthcoming article we assess the potential impact of comorbidity in ADHD on measures of cognitive inhibition across two NP tasks (Pritchard, Neumann, & Rucklidge, 2007b). Comorbidity may prove critical in future studies regarding cognitive inhibition in developmental ADHD populations and the status of the disinhibition model in the disorder. For now, however, what seems certain is that there is little evidence that the cognitive inhibition mechanism responsible for NP is defective in ADHD, at least in adolescents with a delimited diagnosis of ADHD (without comorbidities).

Because inhibitory mechanisms are assumed to play a crucial role in orchestrating performance in various domains, such as perception, selective attention, working memory, memory retrieval, and motor processes (Anderson & Spellman, 1995; Kok, 1999; Neumann & DeSchepper, 1992), knowing the precise forms of inhibition that operate in these multiple systems should enable the development of theoretical and clinical knowledge of the specific cognitive deficits confronted by people with ADHD. Based on the present findings, it seems reasonable to conclude that the disinhibition theory of ADHD should not encompass the particular form of cognitive inhibition that underlies Stroop interference resolution and NP effects.

CHAPTER 5

Study 4

Selective attention and inhibitory deficits in ADHD: Does subtype or comorbidity modulate negative priming effects?[10]

5.1 Abstract

Selective attention has durable consequences for behavioral and neural activation. Negative priming (NP) effects are assumed to reflect a critical inhibitory component of selective attention. The performance of adolescents with Attention-Deficit/Hyperactivity Disorder (ADHD) was assessed across two conceptually based NP tasks within a selective attention procedure. Subtype (combined vs. inattentive) and comorbidity (ADHD non-comorbid vs. ADHD comorbid) were considered key issues. Results found NP effects to differ as a function of comorbidity but not subtype. Findings are discussed in light of functional neuroimaging evidence for neuronal enhancement for unattended stimuli relative to attended stimuli that strongly complements an inhibitory-based explanation for NP. Implications for the 'AD' in ADHD and contemporary process models of the disorder are considered.

[10] Paper submitted to and under revision for *Brain and Cognition:* Pritchard, V. E., Neumann, E., Rucklidge, J. J. (under revision). Selective attention and inhibitory deficits in ADHD: Does subtype or comorbidity modulate negative priming effects? *Brain and Cognition*

5.2 Introduction

A topic of continuing interest in cognitive neuroscience concerns how the human information processing system overcomes attentional competition generated by concurrent stimulus inputs. Attention is modulated by both goal-directed (top-down) and stimulus-driven (bottom-up) factors. In selective attention, the control or regulation of behavior is restricted to some subset of information relevant to a current goal. According to biased competition theory (Desimone & Duncan, 1995), top-down effects enhance processing for stimulus representations most relevant to current behavior while reducing or gating this process for unwanted competing stimuli representations. An alternative view suggests unwanted representations are not simply screened out, but implicitly registered and automatically subjected to active inhibition (Neumann & DeSchepper, 1992; Tipper, 2001).

These issues are critical to the valid development of current process models of Attention-Deficit/ Hyperactivity Disorder (ADHD) connecting frontal lobe control systems (Barkley, 1997) and subsidiary attentional signalling systems in the anterior regions of the cortex (Nigg & Casey, 2005; see also Casey & Durston, 2006) to difficulties with interference control. Inhibitory mechanisms are assumed to play an integral role in orchestrating performance in various domains, such as perception, selective attention, motor processes, working memory, and memory retrieval. Elucidating the precise forms of inhibition that operate in these multiple systems should further the development of theoretical and clinical knowledge of the specific attentional and cognitive deficits confronted by those with ADHD.

Disinhibition models of ADHD

Much of the literature on disinhibition in ADHD has focused on deficits in response inhibition and interference control as operationalized by the Stroop task (see Nigg, 2001, for a review). Stroop tasks typify a class of interference whereby the introduction of task-irrelevant stimulus dimensions slows response time (RT). For instance, in Stroop interference tasks color naming times for color-hues are impaired by the presence of a task irrelevant incongruent color-word (e.g., the word "red" printed in blue) relative to color naming times for neutral stimuli (e.g., the letters "iii" printed in blue). The Stroop effect is widely used as an index for inhibitory response or interference control in the study of psychopathological populations. Increased Stroop effects are taken to indicate reduced capacity for inhibition or reduced ability to control and suppress the prepotent word reading process. However, debate continues as to whether Stroop interference activates an inhibitory process to resolve conflict between competing stimulus dimensions or some other process (cf. Cohen, Dunbar, Barch, & Braver, 1997; Schooler, Neumann, Caplan, & Roberts, 1997a, 1997b). Supplementing interference measures with negative priming procedures is more likely to provide an accurate assessment of the nature of the conflict resolution recently transpired.

Negative priming and active inhibition

Evidence from behavioral priming studies suggests unattended stimuli are implicitly registered and subjected to further processing. Despite being ignored, unattended stimuli often produce a traceable "negative priming" effect (NP; Tipper, 1985). Typically indexed over a series of sequential trials containing simultaneous target and distractor displays, NP refers to a response cost incurred when the distractor stimulus on the prime trial becomes the target stimulus on the probe trial (i.e., the ignored repetition [IR] condition) relative to trials where prime and probe stimuli are unrelated (i.e., the unrelated [UR] condition). NP was first documented in Dalrymple-Alford and Budyar's (1966) seminal study on the effect of Stroop stimuli sequencing on interference. This study found that naming the color hue of an incongruent color-word stimulus on a Stroop task was impaired if the current color had been employed as the distractor (i.e., the word stimulus) in the preceding trial relative to trials where current target and distractor stimuli were unrelated. Widely documented over a broad range of tasks (for reviews, see Fox, 1995; May, Kane, & Hasher, 1995) and operating at the level of semantic, perceptual, and auditory stimulus representations (Allport, Tipper, & Chmiel, 1985; Buchner & Mayr, 2004; Driver & Baylis, 1993; Tipper & Driver, 1988, Tipper, 1985) NP appears to reflect a general component of the selection process in situations with intensively clashing targets and concurrent distractors.

Inhibition-based accounts of NP phenomena hold that the selection of target stimuli is achieved via an active inhibitory process that operates to reduce concurrent interference from distractor stimuli (Houghton & Tipper, 1994; Neumann & DeSchepper, 1992; Strayer & Grison, 1999). These accounts incorporate activation-suppression models of attention in which initial analysis of both unattended and attended items takes place in parallel prior to selection (e.g., Neill & Westberry, 1987; Neumann & DeSchepper, 1991; Tipper 1985). For a response to be directed towards the target, an excitatory mechanism operates to maintain the internal representation of the target, while an inhibitory mechanism operates to suppress the competing distractor representation. Applied to priming procedures, activation-suppression models predict a priming benefit or positive priming for recently attended stimuli and NP for recently unattended stimuli. Theoretical accounts of NP however remain notoriously controversial as potentially opposing influences generated from the trace of an unwanted distractor object can also work to impair later response times (Neill & Valdes, 1992).

Neuronal enhancement as evidence for a functional inhibitory action on unattended stimuli

The notion that the active inhibition of unattended non-target stimuli forms a critical component of selective attention and interference resolution has become increasingly influential in the past two decades (see Tipper, 2001, for a review). In the realm of cognitive neuroscience, priming paradigms continue to offer insight into the mechanisms that may underlie selective processing. A possible inhibitory locus for perceptual NP revealed during a recent fMRI priming study strongly complements an inhibitory-based explanation for NP (Vuilleumier, Schwartz, Duhoux, Dolan, & Driver, 2005).

Vuilleumier et al. used a delayed repetition priming procedure during event-related fMRI to examine later neural traces for visual objects either attended or ignored during initial perceptual exposure in a selective attention task. At initial exposure, target and distractor objects in isoluminant colors were presented on screen as an overlapping visual display. Targets were selected by prespecified color via a manual key press. At later trial re-exposure, visual objects previously attended and unattended were presented in isolation for manual response. Vuilleumier et al. found fMRI response increases (neuronal enhancement) for recently unattended objects on re-exposure. These effects were in clear contrast to fMRI response decreases (neuronal suppression) found with behavioral positive priming effects for recently attended visual objects on re-exposure trials. Vuilleumier et al., (2005) concluded that the neuronal enhancement they observed for recently unattended objects on re-exposure would likely relate to prior inhibitory processes that ultimately produce behavioral NP under typical IR conditions.

Identifying the precise psychological determinants, neural mechanisms, and cortical profile of NP phenomena seems critical to our understanding of the nature of the selective attention process, both in typical as well as in pathological cognition. NP has proved an important paradigm in Alzheimer and schizophrenia research where cognitive difficulties in attention correspond with reduced NP effects (Beech, Powell, McWilliam, & Claridge, 1989; Laplante, Everett, & Thomas, 1992; MacQueen, Galway, Goldberg, & Tipper, 2003; Sullivan, Faust, & Balota, 1995).

Possible inhibitory loci for NP phenomena revealed through fMRI place the NP procedure at the forefront as a leading index for cognitive inhibitory processes involved in interference resolution and attentional control. Non- or anti-inhibitory accounts of NP, and models of selective attention (Desimone & Duncan 1995), and Stroop interference resolution (Cohen, et al., 1997; Cohen, Dunbar, & McClelland, 1990) that do not contain this type of inhibition-based processing in their frameworks, may be missing one of the key information processing mechanisms in the human repertoire. In terms of neural or cognitive processes it seems likely that multiple sources of inhibitory processing exist at various loci in the stream of information processing operations (see also Pritchard, Healey, & Neumann, in press).

NP effects in ADHD: A function of subtype and comorbidity?

Data on cognitively defined attentional processes with an established neural basis are sorely needed in ADHD research (Huang-Pollock & Nigg, 2003; Huang-Pollock, Nigg, & Halperin, 2006). This is particularly evident when regarding subtypes of the disorder, where distinct neuropsychological profiles associated with attention and inhibition, are proposed for each subtype (e.g., see Barkley, 1997). Neuropsychological and clinical research into ADHD has invested surprisingly little effort into tracing the implications for basic cognitive or neural networks that may subserve attentional control or more automatic forms of inhibition. These are key gaps in the field when the "AD" in ADHD is assumed as the near sine qua non of the disorder and inhibitory dysfunction is assumed to be primary to symptom presentation. In our view, NP reflects a relatively automatic cognitive inhibitory process in the sense that it can be an emergent by-product of focusing on and responding to a target stimulus in the presence of a conflicting distractor stimulus.

Out of the six existing studies that have used NP procedures to evaluate disinhibition theories of ADHD samples, two report diminished NP effects in ADHD relative to controls (e.g., Marriott, 1998; Ossman & Mulligan, 2003). On the other hand, increasing evidence for intact NP in children and adolescents with ADHD equivalent to those in age-matched controls (e.g., Gaultney, Kipp, Weinstein, & McNeill, 1999; Nigg, Butler, Huang-Pollock, & Henderson, 2002; Pritchard, Healey, & Neumann, 2006; Pritchard, Neumann, & Rucklidge, 2007a) suggests the disinhibition model of ADHD should not encompass the inhibitory process that may underlie Stroop interference resolution and NP effects. However, complicating accurate inference is the hetereogeneity of phenotypic descriptions of ADHD and the ubiquitous tendency for ADHD to present with comorbid diagnoses.

The specificity of attentional and inhibitory deficits to ADHD and ADHD subtypes are key questions (Barkley, 1997; Huang-Pollock et al., 2006; Milich, Balentine, & Lynam, 2001; Nigg, 2001; Sergeant, Oosterlaan, & van der Meere, 1999) and remain issues of extensive concern in the ADHD literature. The primary objective of the present paper was to assess the potential of subtype and comorbidity in ADHD to modulate NP effects before making a more definitive conclusion about the status of the NP effect in ADHD.

DSM-IV (American Psychiatric Association, 1994) distinguishes between three behavioral subtypes: predominantly hyperactive/impulsive (ADHD-H), predominantly inattentive (ADHD-I), and combined (ADHD-C). These subtypes, particularly the ADHD-I vs. the ADHD-C subtypes, have distinct neuropsychological correlates (Milich, et al., 2001). Theories of disinhibition in ADHD are largely confined to and reflected in symptoms characteristic of ADHD-C (a subtype characterized by executive dysfunction) rather than to the ADHD-I spectrum of symptoms. However, direct studies of cognitive inhibition pitting ADHD-I against ADHD-C are lacking, especially in regard to specific measures of cognitive inhibition (Nigg, 2001).

One important aim in the present study is to directly compare cognitive inhibitory and attentional function between ADHD-C and ADHD-I subtypes. Prior studies reporting intact NP effects in ADHD have evaluated the effect for ‘general ADHD’ without regard for subtype. Failure to dissociate between inattentive and combined subtypes in the NP paradigm may result in premature and erroneous exclusion of the form of cognitive inhibition tapped by NP procedures from disinhibition models of ADHD. If inhibitory difficulties are confined to the ADHD-C subtype as predicted by Barkley (1997), we would expect to see reduced NP effects for ADHD-C relative to ADHD-I. Alternatively, when the selective attention angle of the NP effect is emphasized a diminished or non-significant NP effect might be predicted for ADHD-I rather than ADHD-C. Studies suggest inattentive subtypes have a deficit specifically in focused or selective attention (see, Goodyear & Hynd, 1992; Lahey & Carlson, 1992). Evidence for qualitative distinctions between the neuropsychological profiles for inattentive and combined subtypes continues to emerge. For now, the putative neuropsychological distinction between subtypes in cognitively defined terms, particularly in attention, remains relatively undefined.

Another primary objective was to assess the degree to which NP might vary as a function of comorbidity in ADHD. ADHD has a high tendency to coexist with other psychiatric disorders such as Conduct Disorder, Oppositional Defiant Disorder, anxiety, and mood disorders (Angold, Costello, & Erkanli, 1999). Because such disorders may be associated with differential cognition (Angold et al., 1999; Lufi, Cohen, & Parish-Plass, 1990; MacLeod & Prior, 1996; Ozonoff, Strayer, McMahon, & Filloux, 1998; Seidman, Bierderman et al., 1997) it seems essential to clarify their impact on neuropsychological effects specific to ADHD either by covariance or exclusion. The issue of comorbidity in ADHD is pertinent to any evaluation of the disinhibition model, particularly with regards to types of inhibition (e.g., see Nigg, 2001; Nigg, et al., 2002) and interference resolution in Stroop-like paradigms.

Interference control in the Stroop task may be more impaired in ‘ADHD comorbid’ than in ‘ADHD non-comorbid’ or more interestingly, the effect may be reversed. Studies by MacLeod and Prior (1996), and Lufi et al. (1990) both found increased susceptibility to Stroop interference with groups of adolescent ADHD participants relative to control groups. These effects persisted even when naming speed deficits specific to ADHD had been controlled for via a standardized interference score. To address the impact of comorbidity on outcome, MacLeod and Prior conducted a series of separate analyses comparing interference scores between ‘ADHD non-comorbid’, and ‘ADHD comorbid’. No significant differences were found. On the other hand, Lufi et al. (1990) conducting similar analyses found a significant difference between the two groups for the Stroop effect, with 'ADHD non-comorbid' exhibiting heightened interference relative to 'ADHD comorbid'.

In the present study, two analyses were conducted to evaluate the impact of comorbidity (ADHD non-comorbid vs. ADHD comorbid) and subtype (ADHD-C vs. ADHD-I) on NP effects. In the first analysis we used a cohort control strategy in which the group with ADHD was subdivided into those with and without comorbidity. We were specifically interested in determining whether ADHD participants *with* a comorbid diagnosis would incur heightened or diminished NP effects in comparison to ADHD participants *without* a comorbid diagnosis. Although Pritchard et al. (2007a) found intact and comparable NP effects between ADHD participants without a comorbid diagnosis and age-matched controls[11], no prior research on NP in the ADHD literature has attempted to evaluate the direct impact of subtype or comorbidity on NP assessments. The extents to which comorbid conditions might be masking potential differences that are specific to ADHD are unclear. The second analysis used a similar strategy in which the group with ADHD was subdivided into ADHD-C and ADHD-I subtypes. Given that predictions for inhibitory problems in ADHD lie with the ADHD-C subtype, one could expect to see a reduced NP effect for ADHD-C relative to ADHD-I subtypes. However, the selective attention deficits purported to be specific to ADHD-I might see reduced NP for the ADHD-I subtype as well thus yielding a non effect for both subtypes.

---

[11] Study 4 is an extension of the study by Pritchard et al. (2007a). In that study it was demonstrated that Stroop and flanker NP effects are directly comparable between controls and ADHD participants without a comorbid diagnosis (the ADHD noncomorbid group that is in fact included in Study 4). Pritchard et al. (2007a) found no evidence for any deviation between the magnitude of NP produced by the ADHD and control group.

5.3 Method

General Procedure. The interviews and tasks were carried out in the laboratories at the Department of Psychology at the University of Canterbury. The local institutional review board approved the study and written informed consent and assent (for children under the age of 16) were obtained from parents and adolescents respectively. Registered clinical psychologists conducted all psychiatric interviews. Ten percent of the interviews were videotaped and reviewed by the same doctorate level clinical psychologist. Inter-rater reliability for agreement on presence/absence of diagnosis was 100%. All participants were reimbursed 20 dollars (N.Z) for costs of parking and lunch.

Those participants who were receiving psychostimulant (dextroamphetamine or methylphenidate) medication (n=22 (59%)) discontinued this treatment 24 hours before the day of testing because of the known effects of methylphenidate on cognitive functioning (e.g., Berman, Douglas, & Barr, 1999). This process of asking children not to take their medication on the morning of the testing is a commonly used procedure in ADHD research in order to determine specific deficits involved in the disorder, deficits that may be masked by medications (e.g., Rucklidge & Tannock, 2002; Clark, Prior, & Kinsella, 2000). On the day of testing, it was confirmed with parents that their child had not been given their ADHD medication that morning. As methylphenidate has an approximate half-life of 4.5 hours (Shader, Harmatz, Oesterheld, Parmelee, Sallee, & Greenblatt, 1999), a 24-hour elimination period should have ensured that the majority of the active ingredient had been eliminated prior to testing. Eight (21.6%) participants were taking a medication other than a stimulant (e.g., fluoxetine, clonidine, lithium, paroxetine). These other medications were not discontinued.

Participants. A total of 44 adolescents (aged 13- to 17-years) with ADHD were included in this experiment: (23 male, 21 female). Thirty-five (79.5%) of the ADHD group comprised New Zealanders of European ancestry. Two participants from the ADHD group were Maori (Indigenous people of New Zealand). The remaining participants were “Other” European. Twenty-eight participants (63.6%) were from intact families. The ADHD group was recruited from individuals who were previously assessed at a specialized mental health center that services the moderate to severe spectrum of psychiatric disorders. Sample characteristics are provided in Table 1 (see pg. 128).

Diagnostic protocol for ADHD and other psychiatric disorders. Systematic information about current and lifetime disorders was obtained from both the participant and the parent separately using the Schedule for Affective Disorders and Schizophrenia for School–Age Children-Present and Lifetime Version (K-SADS-PL), an interview which generates both DSM-IIIR and DSM-IV diagnoses. This semi-structured interview has been used extensively to make diagnostic decisions based on DSM criteria and has been validated with children and adolescents 6 to 17 years of age (Kaufman, Birmaker, Brent, Rao, Flynn, Moreci, Williamson, & Ryan, 1997). The long versions of the Conners’ Rating Scales-Revised (Conners, 1997) were used to assess ADHD were used for inclusion/exclusion criteria. This instrument provides separate rating forms for parents, teachers, and adolescents.

Inclusion criteria for ADHD. To assess for presence of ADHD, the following diagnostic algorithm was used: 1) the subject met DSM-IV criteria for ADHD according to the clinician summary based on the K-SADS parent and adolescent interview, 2) for those participants attending school regularly (n=30), met the clinical cutoffs for the externalizing symptoms of ADHD on either one or both of the TRF or the Conners’ teacher questionnaires in order to ensure pervasiveness of symptoms across settings, and 3) showed evidence of ADHD symptoms prior to the age of seven established either through a past diagnosis of ADHD or in new cases, according to parental report, medical records and past school report cards. Impairment was confirmed using the K-SADS-PL. The presence or absence of DSM-IV internalizing disorders was based on a clinician summary derived from the information gathered from both the parent and adolescent K-SADS-PL interview. Note that the information from the adolescent K-SADS-PL did not supersede parental report for the presence/absence of externalizing symptoms.

According to this diagnostic protocol, 18 (40.9%) of the ADHD group were identified as Combined Type, 3 (6.8%) as Predominantly Hyperactive/Impulsive Type and 23 (52.3%) as Predominantly Inattentive Type. Twenty-eight (63.6%) of the ADHD group had at least one other comorbid diagnosis, including Mood Disorder, (7 (15.9%)), Anxiety Disorder (5 (11.4%)), and Oppositional Defiant Disorder, (21 (47.7%)) of which 12 (27.3%) also met criteria for Conduct Disorder.

Exclusion criteria for the ADHD group. Participants were excluded from analyses if they had: 1) an estimated IQ below 75, using the Block Design and Vocabulary subtests of the Wechsler Adult Intelligence Scale-III (WAIS-III; Wechsler, 1997) or the Wechsler Intelligence Scale for Children-III (WISC-III; Wechsler, 1991), a combination of subtests commonly used to estimate full scale IQ. Any particiapnts with uncorrected problems in vision or hearing, serious medical problems (e.g., epilepsy or cerebral palsy), or serious psychopathology, such as psychosis, that would preclude a current differential diagnosis of ADHD were excluded from both groups. All adolescents participating in the study were native English speakers. These exclusion criteria resulted in four participants excluded due to low IQ. In addition, two were excluded due to being color blind and one for missing data on the NP tasks.

Measures of demographic variables. The New Zealand Socioeconomic Index of Occupational Status (NZSEI; Davis, McLeod, Ransom, & Ongley, 1997) was used as a measure of socio-economic status. This index assigns NZ occupations with a socio-economic score (SES) from 10 (low SES) to 90 (high SES). Highest education level achieved by each parent (from 1 'no high school' to 6 'university degree') was also used as a measure of economic status. Demographic data for all participants is presented in Table 1 (see pg. 128).

Experimental measures. Two conceptual NP tasks using distinct stimuli types were chosen to evaluate the NP effect as a function of subtype and comorbidity in ADHD. Both tasks have previously shown intact NP effects in ADHD samples (e.g., see Pritchard, et al., 2006; Pritchard et al., 2007a. These tasks yield two dependent measures: the time taken to name colors in an unrelated (UR) priming condition and the time taken to name colors in an ignored repetition (IR) priming condition

Stroop NP task. The task was administered via twelve 26 x 18 cm cards, with each containing the color words RED. ORANGE, BLUE, PINK, PURPLE, BROWN, YELLOW, GREEN, BLACK, WHITE, and GRAY. The order in which these words appeared was counterbalanced across all cards. On each card, all color words were arranged as a single vertical column against a light gray background with the print of each word presented in one of the 11 corresponding colors, with the constraint that the print color and word were incongruent (see Appendix A). Each Stroop item measured 1.0 cm in height with each item spaced at 1.0 cm intervals down the column on each card. Experimental trials consisted of

50% unrelated (UR) trials (where neither the print color nor distractor color word in a Stroop NP stimulus were related to the subsequent Stroop NP stimulus), and 50% IR trials (where the distractor word in the previous Stroop NP stimulus named the target print color of the subsequent word).

Flanker NP task. The task was administered via twelve 32 x 22 cm cards, with each containing a column of 11 unique sets of three-different shaped color blobs. These stimuli sets consisted of a central target color blob flanked on each side by non-target incongruently colored blobs. The color for the target blob always differed from the color shared by the flanking distractor blobs (see Appendix B). The 11 colors for blobs were as used above in the Stroop NP task. The sequential arrangement of stimuli sets differed for each card, and each set was randomly staggered to either the left or the right in an attempt to reduce the saliency of the IR condition. Trials consisted of 50% UR (in which there was no relationship between the colors of outer distractor blobs in the previous display and the color of the subsequent inner target blob) and 50% IR (in which the color of the distractor blobs in the previous display matches the subsequent target color blob). Visual distances between individual blob rows were the same for both UR and IR cards.

Table 1

Sample Characteristics: Means and Standard Deviations

Variable	ADHD (n=44)	
	Mean	SD
Estimated IQ	95.91	11.91
Age	15.18	1.25
Mother's education	4.03	1.79
Father's education	3.63	2.04
NZSEI	54.77	17.88
CPRS-R (T scores)		
DSM In	75.60	11.42
DSM H/I	80.76	11.51
CSRS-R (T scores)		
DSM In	61.95	13.38
DSM H/I	56.98	13.95
CTRS-R (T scores)		
DSM In	61.80	20.35
DSM H/I	61.93	21.15

Note: NZSEI = New Zealand Socioeconomic Index of Occupational Status, CPRS = Conners' Parent Rating Scale, CSRS = Conners-Wells' Self-report Scale, CTRS = Conners' Teacher Rating Scale.

Testing procedure. The experiment was conducted in one session. Administration of the two NP tasks followed an identical double-blind procedure. Prior to the experiment, all participants completed a preliminary color vision and identification task to ensure accuracy and familiarity with the entire set of 11 colors used within the experiment. No participants reported any difficulties with this task. Before the commencement of test cards for both NP tasks, each participant encountered four UR cards as practice trials. Depending on the task, they were given verbal instructions to name as quickly and as accurately as possible from the top to the bottom of the column on each card either the print color of each color word (Stroop NP task) or the color of each central blob (flanker NP task). They were also asked not to cease color naming if an error was made but rather continue to complete the card. After the initial practice phase for each NP task, participants were the given the 12 experimental cards (six per priming condition presented in alternation). Presentation orders were further counterbalanced so that half of the participants began with an IR card and the remaining half with a UR card. Each card was covered with a blank sheet removed by the experimenter on the word “Go” A stopwatch was used to record the time taken to complete color naming for each card. This was started with the removal of the blank cover sheet and stopped in synchrony with the naming of the last color on a card (see Appendices C and D for administration details for the Stroop and flanker NP tasks). Error scores were tabulated for each card. Errors were defined as either omissions or verbalizations of an incorrect or absent color.

5.4 Results

1. Comorbidity Analyses. To determine whether comorbidity had the potential to modulate NP in the Stroop NP task or eliminate the appearance of the effect in the flanker

NP task a comparison of the NP scores between 'ADHD non-comorbid' (n=16) and 'ADHD comorbid' (*n*=28) groups was analysed in a 2 x 2 ANOVA. The between-subjects factor was comorbidity (ADHD non-comorbid vs. ADHD comorbid) and the within-subject factor was priming condition (UR vs. IR). Mean RTs, SDs, and ER% as a function of comorbidity and priming condition are shown for Stroop and flanker NP tasks in Table 2. There was no significant main effect of group on overall RTs for either NP task, p's $> .70$. The within-subject factor of priming condition (UR vs. IR) for the Stroop NP task was highly significant, indicating an intact NP effect with naming latencies longer on IR trials than on UR trials, $F(1,42) = 10.64$, $p < .01$. The interaction between priming condition X comorbidity for the Stroop NP task did not approach statistical significance, $F < 1$.

However, the interaction between priming condition X comorbidity for the flanker NP task was significant, $F(1,42) = 5.20$, $p < .02$. Follow-up single sample *t*-tests found significant NP for the 'ADHD non-comorbid' group (45ms), $p < .02$. No significant NP effects were obtained for the 'ADHD comorbid' group (-3ms), $p > .92$, despite the larger sample size. Similar analyses were conducted for error scores across both NP tasks, but failed to yield any observable effects, p's $> .10$. These results suggest that inhibitory dysfunction may be more prominent in individuals with ADHD who present with comorbid diagnosis.

Table 2

Mean Reaction Time (in Milliseconds) per Item and Percentage of Errors for ADHD Non-Comorbid and ADHD Comorbid as a Function of Priming Condition

	Group			
	Non-comorbid		Comorbid	
Priming condition	UR	IR	UR	IR
Stroop NP task				
Reaction time				
M	1127	1166	1137	1247
SD	323	330	320	338
ER (%)	1.8	1.4	2.8	2.0
Flanker NP task				
Reaction time				
M	725	770	776	773
SD	221	230	272	228
ER (%)	.81	.63	.90	.90

2. Subtype Analyses. To investigate predictions that diminished NP effects may be found for combined rather than inattentive subtypes we re-combined ‘ADHD non-comorbid’ and ‘ADHD comorbid’ participants to form an ADHD group of 44. We then subdivided this group into ADHD-C and ADHD-I subtypes according to diagnostic protocol. The ADHD-C group contained 18 participants and the ADHD-I group contained 23. The 3 participants diagnosed with ADHD-H were not included in these analyses. NP scores for ADHD-C and ADHD-I were assessed via a 2 x 2 ANOVA treating subtype (ADHD-C vs. ADHD-I) as the between subjects factor and priming condition (UR vs. IR) as the within-subject factor. Mean

RTs, SDs, and ER% as a function of priming condition and subtype for both NP tasks are presented in Table 3.

Table 3

Mean Reaction Time (in Milliseconds) per Item and Percentage of Errors for ADHD-C and ADHD-I as a Function of Priming Condition

	Group			
	ADHD-C		ADHD-I	
Priming condition	UR	IR	UR	IR
Stroop NP task				
Reaction time				
M	1092	1155	1189	1295
SD	319	337	335	340
ER (%)	1.7	2.3	1.7	2.2
Flanker NP task				
Reaction time				
M	786	795	755	780
SD	241	285	236	222
ER (%)	.72	.89	.96	.73

The main effect of group on overall RTs did not approach statistical significance for either NP task, *p's* > .25. A significant NP effect was obtained for the Stroop NP task, F (1,39) = 9.70, p < .01. More critically, the interaction between priming condition X subtype did not approach statistical significance, F (1,39) = 1.38, p > .25. To confirm our findings, a comparison of the Stroop NP score between subtypes was assessed via a t test for independent samples with a significance level set at .01. Results obtained suggested the Stroop NP score was invariant between ADHD-C and ADHD-I, t (39) = 0.77, p > .45.

Because of the potential confounds associated with comorbidity on the flanker NP task, the NP effect did not approach significance, $F(1,39) = .67$, $p > .42$. However, similar to results obtained for the Stroop NP task, no significant interaction between priming condition X subtype was observed for the flanker NP task, $F < 1$. Error analyses yielded no significant effects, all F's < 1. Differing neuropsychological profiles for ADHD subtypes did not appear to be a moderating factor in NP performance.

5.5 General Discussion

The present experiments were designed to test purported difficulties with inhibition and attention specific to ADHD subtype, and to ADHD more generally. A further aim was to evaluate the direct impact of comorbidity on previous assessments of NP in ADHD. It was hoped that these assessments would allow for a more definitive statement regarding the status of the NP effect in ADHD. The current experiments were executed in light of the functional significance of NP in the evaluation of selective attention and inhibitory processes in pathological assessments. The integration of priming procedures and fMRI techniques have begun to provide compelling and more direct support for the theoretical notion of active inhibition as a critical processing component underpinning the NP effect (Vuilleumier et al., 2005). Linking specific neural sites and activity to inhibitory processes acting on unattended stimuli during IR prime trials that may correlate with behavioral NP effects on IR probe trials promotes a more detailed understanding of the mechanisms underpinning selective attention capacities. And stimulus specific neuronal traces generated at perceptual encoding for unattended objects that differ qualitatively as well as quantitatively from those for attended objects are uniquely informative with regard to the neural underpinnings of identity NP effects.

In the current report, we evaluated inhibition and selective attention in ADHD using NP variants of Stroop and flanker interference tasks across two different analyses. In the first analysis, we examined the potential for comorbidity in ADHD to modulate the NP effect. Comorbidity was found to have a direct influence on NP scores. Intact and equivalent NP effects were reported for both 'ADHD non-cormorbid' and 'ADHD comorbid' on the Stroop NP task. Interestingly, while NP effects were intact and highly significant for 'ADHD non-comorbid' on the flanker NP task, these effects failed to reach statistical significance for 'ADHD comorbid' on the same task.

The absence of a significant NP effect for ADHD with comorbidity on the flanker NP task suggests comorbidity may be accountable for some prior anomalies in the NP ADHD literature. However, findings of intact NP for ADHD with comorbidity on the Stroop NP task suggest some discrepancies in this literature may also relate to stimulus type and NP task design. Here, it is noteworthy that consistent and reliable NP effects for children and adolescents with ADHD regardless of comorbidity have thus far only been found in the context of Stroop NP tasks. Studies that reported diminished or absent NP effects in children and adolescents with ADHD used NP measures that comprised monosyllabic, five-letter nouns as target and distractor stimuli (Marriott, 1998; Ossman & Mulligan, 2003). Such tasks, not unlike the current flanker NP task, avoid the prepotent-alternative response dynamic inherent in the Stroop paradigm. Although one can only speculate, it may be that people with generalized ADHD require a more 'effortful' interference task to trigger active inhibitory mechanisms underpinning successful selective attention. This is an area that deserves further scrutiny. Similar variables concerning NP task situations are found with NP studies addressing the effect in early development (Pritchard & Neumann, 2004). For now, it appears that while comorbidity may have the potential to distort functional cognitive inhibition in ADHD, it seems increasingly clear that active inhibitory processes underlying Stroop interference resolution and response inhibition appear to be intact in ADHD.

In the second analysis, to evaluate inhibitory deficits associated with Stroop performance deemed central to the ADHD-C subtype but not to the ADHD-I subtype, we directly pitted NP scores found for the ADHD-C subtype against those found for the ADHD-I subtype. In keeping with the disinhibition model, the expectations were for a diminished NP effect in ADHD-C relative to ADHD-I. Contrary to predictions, the expected differences were not found on the Stroop NP task. Instead, the NP effect was intact and equivalent for ADHD-C and ADHD-I subtypes. Assessing predictions for inhibitory dysfunction in ADHD that are deemed relevant to only a particular subtype of the disorder helps to ensure a relatively accurate assessment of the dishibition model of ADHD. Because subtype did not appear to modulate NP, the results of the second analysis provide additional support for prior findings of intact NP in ADHD. NP effects in ADHD samples emerge intact irrespective of subtype.

Neuropsychological profiles of ADHD

The majority of ADHD literature implies the disorder has a pathological basis in top-down cortical control systems such as the prefrontal cortex (PFC) and frontostriatal networks (see Halperin & Schulz, 2006, for a review). Dedicated to the service of higher order cognitive control and executive action, the PFC is believed to bias subsidiary processing implemented by posterior cortical and subcortical regions in accordance with current goals.

One of the mechanisms by which the PFC is believed to exert its coordinating effects is via the suppression or gating of neural signalling irrelevant to current behavior or goal. Thus, noted deficits for ADHD participants on measures tapping executive actions such as planning (Barkley, 1997; Pennington, Groisser, & Welsh, 1993), and set shifting (Hall, Halperin, Schwartz, & Newcorn, 1997) that may require the suppression of extraneous information or response appear consistent with frontal defects. The Stroop task, often viewed as a valid measure of frontally mediated response inhibition (Stuss & Benson, 1984), provides much of the impetus for the idea of inhibitory problems in ADHD.

Several reviews in the ADHD literature concluded that heightened Stroop interference effects distinguish children with ADHD from controls (Barkley, 1997; Harnishfeger & Bjorklund, 1994; Pennington & Ozonoff, 1996). However, these reviews contained several studies that failed to control for rapid color naming deficits that have since been reported for ADHD samples (Semrud-Clikeman, Guy, Griffin, & Hynd, 2000; Tannock, Martinussen, & Frijters, 2000). Thus, calculation methods comparing naming scores for incongruent color-word trials between ADHD and controls were reflective more of heightened difficulties in rapid naming speed rather than of interference.

A recent meta-analysis on the Stroop effect in ADHD by van Mourick, Oosterlaan, and Sergeant (2005) found interference scores corrected for reading and naming speed (i.e., Golden, 1978) failed to differentiate those with ADHD from controls. Recent studies following the recommended calculation procedures have found Stroop interference estimates to be equivalent between ADHD and controls (Pritchard, et al., 2007a; Rucklidge & Tannock, 2002). It thus seems increasingly unlikely that the ability to control interference is compromised in ADHD, at least not within the Stroop paradigm. These findings are further bolstered by the intact Stroop NP effects reported here, and by previous reports of intact and equivalent Stroop NP effects between children and adolescents with ADHD and controls (Gaultney, et al., 1999; Nigg, et al., 2002; Pritchard et al., 2006; Pritchard et al., 2007a).

Moreover, the relation between frontal lobe lesions and Stroop interference is now rather less clear. A study of focal lesion patients by Stuss, Floden, Alexander, Levine, and Katz (2001) found that while frontal damage impaired color naming on incongruent Stroop trials, an interference deficit was not implicated. That is not to say the suppressional projections of the PFC do not mediate other forms of response behaviors (e.g., see Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004). For instance, impaired or impulsive reactions are consistently found for ADHD participants on other measures of inhibitory response such as go/no-go and stop signal tasks that require the suppression of a prepotent response (Aman, Roberts, & Pennington, 1998; Rucklidge & Tannock, 2002; Schachar & Logan, 1990; Vaidya, Austin, et al., 1998). These may well be in line with anomalies in the PFC. Fundamental forms of active cognitive inhibitory processes associated with interference resolution in attention such as those assessed in the current study may operate independently of suppressional control systems mediated by the PFC.

As emphasised in the introduction, inhibition is not a unitary construct. In terms of neural processes and cortical systems it seems likely that multiple sources of inhibitory processing exist. Attentional selection appears to operate at multiple loci. Vuilleumier et al. (2005), for instance, found the neural sites involved in positive behavioral priming effects for objects previously attended, and neuronal enhancement for recently unattended objects were functionally differentiated in the cortex. Activity associated with target objects and “selection for action” was diffused throughout the cortex; found in the right posterior fusiform and lateral occipital regions of the striate cortex, left inferior frontal cortex, and the premotor cortex. The active inhibitory control action tied to irrelevant distractor objects was sited in the bilateral lingual gyri in the posterior visual cortex.

It is worth noting that functional imaging data for stimulus driven or bottom-up attentional control may be of relevance to the development of emerging process models of ADHD. Further reviews on neuropsychological function in ADHD imply *nonfronta*l problems in visual perception and visuomotor integration seem as equally likely to distinguish participants with ADHD from controls as *frontal* problems (e.g., Frazier, Demaree, & Youngstrom, 2004). These reports coincide with a recent shift in focus to implications for more subsidiary processing systems based in the posterior parietal regions of the cortex. In line with these ideas, Nigg and Casey (2005) and Casey and Durston, (2006) suggest breakdowns in these systems may constrain attentional signalling and thus impede top-down cortical gating of context-irrelevant stimuli. There were suggestions that these dysfunctions may account for the observable difficulties for those with ADHD to regulate behavior to context (Casey & Durston, 2006). As delineated in the current study, it seems more likely that attentional control reflects an active inhibitory process. Direct evidence for such a process in ADHD via NP procedures suggests forms of attentional control are not compromised in ADHD.

A search for the attention deficit in ADHD

Increasing evidence for intact NP effects also bear on the issue of the ‘AD’ in ADHD. The precise nature of the attention deficit remains poorly understood and the cognitive implications have yet to be fully elucidated. Attention deficit hyperactivity disorder has been widely conceptualized as a developmental disorder of attention (Barkley, 1998), with particular implications for selective attention (Douglas, 1999) and sustained attention

(Sergeant et al., 1999). However, relatively few studies report the consequences for cognitively defined attentional control processes in ADHD and controversy remains as to whether these processes are in fact dysfunctional (Barkley, 1997; Huang-Pollock & Nigg, 2003; Huang-Pollock et al., 2006). To a large extent these controversies hinge on the fact that DSM-IV (APA, 1994) does not provide a formal definition of attention in cognitive terms. Thus the precise nature of the attention deficit remains poorly understood and undefined.

Advances in cognitive neuroscience have lately seen a resurgence of interest in the relationship between inattention and ADHD (Neufeld, 2002). Prior search tactics for the 'AD' in ADHD have often been hindered by the use of continuous performance measures that lack the specificity to systematically separate selective processes from sustained attention (see, Halperin, McKay, Matier, & Sharma, 1994, for a comprehensive review). Further concerns have been voiced over the potential for attentional measures with questionable construct validity to result in erroneous diagnosis (e.g., Halperin, 1996). Some of the most detailed investigations of selective attention come from studies using the negative priming paradigm. Backed by extensive cognitive theory, NP procedures offer a potentially innovative approach to the study of attention in ADHD. While questions have been raised concerning the ecological validity of these laboratory-type attention tasks and paradigms (Barkley, 1991; 1996, Barkley, Grodzinsky, & DuPaul, 1992), progressions in cognitive neuroscience over the past decade suggest it may be timely to reconsider the application of a more precise approach to attentional assessment in ADHD.

Whether or not laboratory measures of attention augment the generalizability of results to more natural settings, they can begin to clarify the degree to which attentional processes and their neural substrates are compromised in ADHD. As exemplified by the present report, there is certainly no reason to exclude them from assessment procedures. Mounting evidence for intact NP effects in ADHD are pertinent insofar as they show a lack of evidence for the 'AD' in ADHD.

CHAPTER 6

Review

As stated in the Preview, the aims of this dissertation were two-fold. A first aim was to investigate the possibility of age-related differences in NP and to evaluate the outcome of this research on inhibitory and memory-based approaches to NP. It was hoped that this would distinguish more definitively the role of inhibition in the NP phenomenon for the purposes of clinical use. These issues were addressed in studies 1 and 2 (Chapters 2 & 3). Study 1 compared NP performance on two different tasks for five distinct typically developing age groups spanning 5- to 25-years in age. Study 2 tested the idea that unlike adults, children may only produce NP in experimental contexts where distractor stimuli are highly likely to interfere with response to concurrent target stimuli in all trials.

A second aim was to assess the status of NP in children and adolescents with ADHD after issues relating to comorbidity and subtype had been taken into account in an effort to clarify discrepancies relating to the manifestation of NP in young ADHD samples. This may be critical for the valid development of current process models of ADHD that argue deficient inhibitory control is a key characteristic of children and adolescents with ADHD (e.g., Barkley, 1997; Quay, 1997). This issue was addressed in studies 3 and 4 (Chapters 4 & 5). Study 3 compared interference and NP effects between young individuals with non-comorbid ADHD and age-matched peers. To assess the stability of NP in the ADHD sample relative to the control sample, Study 3 examined NP performance using two distinct stimulus types. Study 4 assessed the impact of comorbidity in ADHD on NP and compared NP effects between two ADHD subtypes argued to differ by inhibitory ability (Barkley, 1997; Lahey & Carlson, 1992).

Summary of Findings

This dissertation makes a number of contributions to the NP and clinical literatures. First, no prior research has explored the developmental trajectory of the NP effect and shown that with certain stimulus types NP may in fact be more prevalent in children than adults. Second, this dissertation demonstrates clearly that NP in children is a replicable effect and comparable to NP in adults. Third, this is one of the first attempts to test and flesh out an account for the disparate reports of NP in children with the Stroop task, showing that Stroop NP effects in children modulate as a result of encountering neutral and RD trials in which target selection difficulty is eased. Fourth, by way of a systematic investigation of NP effects in distinct typically developing age groups, this dissertation has identified NP effects unique to development that provide a new basis for favouring an inhibitory approach over a memory approach to NP. Fifth, this dissertation has provided substantial new evidence to suggest that the inhibitory process involved in NP is intact in ADHD. Finally, investigations concerning the impact of comorbidity and ADHD subtype on NP are new. These investigations have shown that comorbidity may account for discrepancies in the ADHD NP literature, and also indicate that reduced NP in ADHD comorbid with CD and ODD may differentiate these disorders from ADHD. The implications of this dissertation are now presented for further discussion.

6.2 No evidence for reduced NP in children

Study 1 emphasized that inhibitory control in children is a central focus of developmental research. One of the most established regularities in the field of cognitive development is that children are slower than adults to select targeted stimuli from among competing alternatives. This apparent failure of selective attention in early development has given rise to the accepted notion that children are less able than adults to ignore or suppress distracting information (Dempster, 1995; Diamond, 2002; Kail, 2002; Pearson & Lane, 1991). Indeed, as outlined in Study 1, a variety of evidence favours the view that young children are less effective inhibitors than adults in task situations where distracting stimuli are a salient variable. And certainly, such data implies that some aspects of inhibitory control may well follow a protracted period of development. However, how valid is the conclusion of poor inhibitory ability in childhood?

One important line of evidence for the hypothesis of inhibitory failure in childhood has been empirical evidence suggesting that NP effects, while consistently produced by adults are lacking in children (Tipper et al., 1989). In the first study of this dissertation, the opposite result was reported. In fact, there were two critical new findings. Not only was NP found to be directly comparable between 5- to 6-year-old children and 19- to 25-year-old adults with Stroop stimuli, but also, when coloured blob flanker-like stimuli were used, it was found that the youngest children produced the strongest NP effect while no NP effect was found at all for young adults. These findings in Study 1 are incompatible with the general assumption that inhibitory control at a global level is less effective in children than adults. Together, these findings suggest that an absence of NP in one age group should not necessarily be taken as evidence for diminished ability in selective inhibition.

With regard to the second finding, it seems important to realise here that NP may often be stimulus dependent and largely determined by how response competitive a distracting stimulus appears to the individual. These are dynamic variables. That is, an older age group might not experience the same degree of response conflict between target and concurrent distractor that are of a specific stimulus type as might a younger age group, and vice versa. For instance, the comparable NP effects between children and adults in Study 1 indicate these two age groups experienced a similar degree of conflict between the target and distractor dimensions of Stroop stimuli. Consequently, similar degrees of inhibition were implemented in children and adults to resolve distractor intrusion, and so Stroop NP effects were comparable between the two age groups. The absence of NP in adults and intact NP in children on colour blob trials in Study 1 may indicate that children find task irrelevant colours incongruent with task relevant colours to be more distracting than adults. Thus, with coloured blobs as distractor stimuli, it is not unreasonable that a greater degree of inhibition was implemented in children than adults, and hence NP was only observed for children.

Such conclusions seem substantially more plausible than the alternative suggestion that absent NP in young adults engaged in the colour-blob NP task was evidence for a deficient inhibitory control process in this age group. To summarise, it seems more likely that age-related differences in NP are stimulus related, and not related to inhibitory ability. That is, what may appear as a developmental difference in inhibitory ability may merely reflect that the experience of response conflict in a NP task is not always the same for children as it is for young adults.

6.3 Towards understanding developmental differences in NP effects

Study 2 outlined that developmental differences in NP appear to occur when Stroop stimuli are used. The results of the NP study by Tipper et al. (1989) showed slight trends (albeit non-significant) toward NP in children when pictorial stimuli were used, but no evidence of NP in children when Stroop stimuli were used. In the contemporary developmental literature, this data pattern gave rise to the accepted view that the inhibitory process underlying NP may develop inconsistently during childhood (Nigg, 2001). This view, however, is not compatible with Pritchard and Neumann's (2004) report of intact Stroop NP in children. These empirical discrepancies between the studies of Stroop NP by Pritchard and Neumann and Tipper et al. led to more recent suggestions (see Lechunga et al., 2006; Muller, Dick, Gela, Overton, & Zelazo, 2006) that while there might be little or no developmental differences in NP when pictorial or coloured flanker stimuli are used, developmental differences appear to exist in NP effects when Stroop stimuli are used.

In the second study of this dissertation, it was revealed that divergent findings regarding the status of Stroop NP in children may be accounted for by the methodological differences between the respective studies of Tipper et al. (1989) and Pritchard and Neumann (2004). Pritchard and Neumann had conjectured that neutral and RD trials included in Tipper et al.'s NP study might have been detrimental to obtaining NP effects in children. As demonstrated in Study 2 this appeared to be the case. When the respective methodology of studies by Pritchard and Neumann (2004) and Tipper et al. (1989) were implemented and used to compare NP between children and adults, the exact same results reported by those authors were achieved. That is, Pritchard and Neumann's finding of intact NP in children was a replicable effect[12] when target selection was consistently difficult across all trials, and in the design after Tipper et al., neutral and RD trials eliminated NP in children, but not adults. From a methodological perspective, this data pattern provides a clear account for the divergent findings in the developmental NP literature.

6.4 Reviewing *selection state* as an account for developmental differences in NP: The critical role of distractor activation in NP manifestation

Pritchard and Neumann's (2004) *selection state* hypothesis may offer an effective explanation for the NP data in Study 2. These authors argued that children may be less able than adults to maintain *selection state* and concomitant inhibitory process in an effective experiment-wide or 'on-line' manner when neutral and RD trials in which target selection is easy are encountered in a NP task. More specifically, Pritchard and Neumann posited that when target selection difficulty is not consistently maintained or anticipated in a NP task, children may be less inclined than adults to actively ignore distractors and hence *selection state* may not properly engage. If true, Pritchard and Neumann's (2004, 2007a) *selection state* hypothesis would account for the observable lack of Stroop NP in children who encountered neutral and RD trials in Study 2. Under such experimental contexts, children may be susceptible to diffuse or divide their attention between target and distractors across all conditions.

Not only do the results in Study 2 begin to resolve discrepant findings in the developmental NP literature, but they also begin to establish the boundary conditions under which NP may manifest in children. When a large proportion of nonconflict or easy target selelection trials are encountered in a NP task, children seem unable to maintain the

[12] Note Study 1 was not a demonstration of replicable NP effects in children. The NP effects Pritchard and Neumann (2004) obtained for children aged 5- to 12-years were entered as data in the analyses undertaken in Study 1.

appropriate attentional set to automatically inhibit distractor stimuli. Instead, chidren's ability to inhibit distractor stimuli in a NP task appears to be maximally operable when only high conflict or difficult target selection trials are encountered. The exact proportion of nonconflict trials that is required in a NP task before the modulation of NP in children may be observed remains to be determined. For now, and given that Frings et al. (in press) found intact NP in children with a ratio of difficult to easy selection trials at 2:1, it seems that the lack of NP in children participating in the Stroop NP experiments by Tipper et al. (1989) and in Study 2 (experiment 2) may relate to the fact that the ratio of difficult to easy selection trials in those experiments was 50:50.

By this account, Tipper et al.'s (1989) findings of a lack of Stroop NP in children and an observable trend toward pictorial NP in children may not be stimulus specific effects (cf. Nigg, 2001; Lechuga et al., 2006), but rather the result of differences between the ratio of difficult to easy selection trials in those tasks (i.e., 50:50 in the Stroop NP task vs. 67:33 in the pictorial NP task). Furthermore, had the nonconflict trials in Tipper et al.'s pictorial NP task been RD trials instead of neutral trials then these authors may have obtained intact NP effects for children. As may be inferred by the RT data in Study 2 (experiment 2), children find target selection to be more difficult in RD trials than neutral trials. Thus an encounter with RD trials rather than neutral trials in a NP task may not be sufficient to disengage *selection state* in children. NP experiments using RD trials as nonconflict trials may require a higher proportion of such trials relative to highconflict trials before a lack of NP is observed for children. Such issues are worthy of further exploration.

Most importantly, Study 2 illustrates that distractor inhibition in children may only be triggered under experimental conditions where the prepotent response tendencies for *all* distractor stimuli are greater than or equal to those for concurrent targets. If such conditions are not maintained, children may process distractor stimuli in a more conscious or even aware sense. If there is a decreased ability to selectively inhibit distractor stimuli, then of course, NP cannot be expected to occur.

6.5 Converging evidence for intact cognitive inhibition in children from the retrieval induced forgetting paradigm

The findings presented in Section 1 are incompatible with the conventionally accepted notion that children have poor inhibitory ability or are less effective than adults in inhibiting distracting stimulus items in selective attention tasks. As tested with the NP procedure, children appear able to suppress information that is irrelevant to current behavioural goal to an adult-like degree. This result would of course be more convincing if found to hold in other suppression paradigms. Such evidence has now indeed been obtained.

Until recently the NP procedure was one of the sole empirical tools to demonstrate intact suppression effects in children (Pritchard & Neumann, 2004). Lately, however, there has been burgeoning research on children’s ability to inhibit “internal distraction” in the retrieval induced forgetting (RIF; Anderson, Bjork, & Bjork, 1994) paradigm. According to Anderson and Spellman (1995), the RIF procedure may tap an inhibitory process similar to the NP procedure. These authors demonstrated that the successful retrieval of a targeted word item in long-term memory produces traceable memory impairment for words that are categorically related to a retrieval target. Anderson and Spellman argued this RIF effect may reflect the by-product of an active inhibitory process that aids the retrieval of task-relevant word items from memory by suppressing activated and competing alternatives in the sematic network. Similar to NP manifestations, RIF may only occur if the word items related to the retrieval target are sufficiently activated to interfere during its retrieval from memory (see Racsmány & Conway, 2006).

An emerging and important finding in the RIF paradigm is that children produce intact RIF effects comparable with those produced by adults (Ford et al., 2004; Lechunga et al., 2006; Zellner & Bauml, 2005). To account for mounting evidence from NP and RIF paradigms that counters the accepted view of an inhibitory weakness in childhood, Lechunga et al. distinguished between intentional and automatic forms of inhibition, and proposed different developmental trajectories for each (see also Nigg, 2001). It was contended by Lechunga et al. that NP and RIF procedures might both source a type of inhibition that functions to automatically suppress interference during selection with no conscious awareness on the part of the individual. These authors suggested that the neural system for 'unintentional inhibition' might develop at an earlier age than those systems involved with more 'effortful' or conscious forms of inhibition.

Such distinctions may help clarify which inhibitory systems develop when, and may also explain why children's performance weaknesses on other inhibition-based tasks, such as go/no-go, stop signal and Simon tasks (Davidson, Amso, Anderson, & Diamond, 2006; Johnston et al., 2005; Zelazo, Craik, & Booth, 2004), do not translate into inhibitory deficits on NP tasks. From a functional perspective, it makes sense that neuronal systems associated with basic selection processes may develop earlier in life. As pointed out by Pritchard and Neumann (2004), an ability to attend selectively is imperative to everyday cognitive function. The ability of children to cope with attentionally taxing situations would be severely compromised without the ability to prioritise the relevant from the irrelevant and to dismiss the latter in an effective manner.

6.6 Theoretical and clinical implications: NP as an index of inhibitory function in young clinical samples

As emphasized throughout this dissertation, the theoretical accounts of NP remain notoriously controversial. Arguably there are now only two theoretical accounts of NP that have survived empirical testing to date; the inhibition-based account vs. the episodic memory based account (see Mayr & Buchner, in press, for a recent review). The inhibitory-based account (Houghton & Tipper, 1994; Tipper, 1985, 2001) holds that NP reflects the consequence of distractor suppression. The anti-inhibitory episodic memory-based account holds that NP reflects behavioural consequences relating to the retrieval of a memory trace containing specific prior response information incompatible with current correct response (Neill & Valdes, 1992). Because such debate has implications for the use of NP effects as a clinical measure of cognitive inhibitory control, and for the purpose of the clinical research on NP in Section 2, a further goal in the first section of this dissertation was to establish whether potential developmental differences in NP may distinguish more definitively the role of inhibition in NP.

To date, given the paucity of NP research on children, there has been no evaluation of inhibitory and memory-based accounts that are based on developmental outcomes. Current theoretical debate rides on the basis of research involving only adults. What may be inferred from the developmental research on NP in this dissertation? Regarding contemporary debate between memory and inhibitory-based accounts of NP, there are two critical results from the two studies in Section 1. In Study 1, coloured blob distractor stimuli produced NP in children but not adults. In Study 2, the experimental context appeared to determine the manifestation of NP in children on IR trials. Both results present a challenge for anti-inhibitory memory-based accounts of NP that hold prior processing instances preserve specific response and stimuli information. To account for the disappearance of NP in adults in Study 1, one would have to claim that children have superior episodic memory compared to adults. From a developmental perspective this seems untenable. Study 2 implies that the appearance of NP in children is mediated by variables conducive to focused attention or processing preference rather than by retrieval mechanisms. Inhibition-based accounts can explain these outcomes with broader flexibility than memory-based accounts of NP. It is difficult to see how an episodic retrieval theory could account for findings in Studies 1 and 2 without some modification to include the involvement of inhibition in the formation of processing instances (see Grison et al., 2005, for a related view). On the basis of the findings in Section 1, it is contended that inhibition should not be precluded as an account for NP.

6.7 Evidence for intact form of inhibitory control in ADHD

Empirical support for the inhibition-based account of NP in Section 1 may be of practical use for future study of inhibitory control in developmental psychopathology, and is directly applicable to Studies 3 and 4 in the second section of this dissertation. In the NP literature on children with ADHD, a potential problem lies in interpreting whether NP effects in these samples would be evidence for efficiency in the automatic retrieval of prior response encoding or in inhibitory control. On the outcome of results in Studies 1 and 2, it seems likely the intact NP effects for children with ADHD reported by Gaultney et al. (1999) and Pritchard et al. (2006) and for those in Study 3 are evidence for an intact form of inhibition in ADHD.

Contemporary research literature holds those with ADHD suffer a widespread deficit in control (Barkley, 1997; Quay, 1997). This is questionable on the basis of the findings obtained in Study 3. There is now critical new evidence to suggest that at least one form of inhibitory control is spared in ADHD. Too often, studies of inhibitory control in ADHD have used measures sourcing meta-cognitive or executive processes. What may appear as an inhibitory problem, may relate more to difficulties in response execution or planning strategies (Ozonoff et al., 1998; Pennington & Ozonoff, 1996). To start the search for fundamental differences in inhibitory function between children with ADHD and controls, it seems essential that investigation in this area should begin with the basic building blocks of cognitive control. As Nigg (2001) points out, and as results in Study 3 imply, inhibition is not a singular concept, and not all forms are implicated in ADHD.

Two unanswered questions in the literature on NP in ADHD relate to specificity and subtype. Here, results in Study 4 are pertinent. They show that prior reports of reduced NP in ADHD may relate instead to ODD and CD and that the inhibitory profile was similar for two ADHD subtypes alleged to differ in selective attention ability (Lahey & Carlson, 1992). These results add to a growing body of literature failing to find evidence for deficits in selective attention at what may be a very fundamental cognitive level.

6.8 Conclusions

Is there now any evidence for reduced NP in typical development?

Contrary to the accepted view, findings in Section 1 indicate there is no evidence for reduced NP in children relative to adults. Tipper et al.’s (1989) report of developmental differences in NP may only reflect that children do not always experience the same degree of distractor interference as young adults in certain task situations. Certainly, results in Study 1 indicate young adults do not always experience the same degree of distractor interference as young children with certain stimulus types. Thus, the absence of NP in one age group but not another under the same task conditions may be a reflection of experimental design and the choice of stimulus type, rather than any difference in inhibitory capacities. NP effects in older adults may also be sensitive to variations in task design. It is noteworthy that the empirical situation regarding NP at the other end of the developmental spectrum has also changed completely. Early reports of inhibitory impairment in older adults are being supplanted by studies revealing intact NP effects in the elderly.

Similar to studies of NP in children, it was initially reported that, in comparison with younger adults, older adults do not show evidence for NP (Hasher, Stoltzfus, Zacks, & Rypma, 1991; McDowd & Oseas-Kreger, 1991; Tipper, 1991). This implied that older adults have a compromised inhibitory mechanism. If true, such inhibitory deficits would help to explain age-related impairments in terms of delayed response and increased susceptibility to interference commonly noted across a variety of interference-sensitive tasks (e.g., Cohen, Dunbar, Barch, & Braver, 1997; Comalli et al., 1962; Davis et al., 1990; Farkas & Hayer, 1980). However, later investigations comparing NP effects in younger and older adults have produced results incompatible with earlier studies (Sullivan & Faust, 1993; Sullivan, Faust, & Balota, 1995; Kramer, Humphrey, Laris, Logan, & Strayer, 1994). This prompted a meta-analysis of studies conducted through 1996 (Verhaegan & DeMeersman, 1998). On the basis of this it was concluded that older adults do show significant NP effects, but not quite as strongly as younger adults, and deemed possible that adults' ability to inhibit may be compromised with advancing age. Since then, however, every study pursuing this issue has reported older adults show NP effects equivalent in magnitude to those shown by younger adults (Gamboz, Russo, & Fox, 2000; Langley, Overmier, Knopman, & Prod'Homme, 1998; Little & Hartley, 2000; Grant & Dagenbach, 2000; Kieley & Hartley, 1997; Kramer & Strayer, 2001; Pesta & Sanders, 2000; Schooler et al., 1997c). Only now, after a second meta-analysis (Gamboz, Russo, & Fox, 2002) are we assured that older and younger adults produce NP effects of comparable magnitude.

Is there now any evidence for reduced NP in atypical development?

Results in Section 2 indicate that the associated inhibitory process is intact in ADHD. Reduced NP in ADHD may only reflect heterogeneity of the sample. Again, it is noteworthy that early reports of inhibitory impairment through Alzheimer's disease and schizophrenia are being supplanted by studies revealing intact and comparable NP effects in individuals so affected (Moritz, Jacobsen, Mersmann, Krauz, & Andresen, 2000; Moritz et al., 2001; Langley, Overmier, Knopman, & Prod'Homme, 1998; Zabal & Buchner, 2006; see also Moulin et al., 2002, for evidence of intact inhibition in Alzheimer patients with the RIF procedure). The specific reasons for discrepancies between the earlier and more recent studies in these domains remain undetermined. However, on the basis of results in this dissertation, it is suggested that variance in data patterns may often relate to experimental design. For instance, in a study of schizophrenia, Mortiz et al. (2001) demonstrated that NP was intact in patients when stimuli were easy to identify, but reduced when stimuli were presented very quickly (100ms) and pattern masked.

6.9 NP effects: Reflections of a rudimentary processing mechanism?

The NP paradigm offers a unique opportunity to investigate dedicated inhibitory mechanisms involved in the selection of relevant over irrelevant information. As indicated in this dissertation, it is becoming increasingly evident that NP is a more omnipresent effect than first recognized (see Mayr & Buchner, in press), and that its absence in one population or another may merely be the result of a methodological artifact. Outcomes reported in Studies 1 - 4 strongly support the view that the inhibitory process underpinning conceptual

NP may be a basic or rudimentary processing resource (Neumann & DeSchepper, 1991, 1992), and they contribute to a growing body of research showing that NP does not diminish with age nor reduce in certain psychopathologies.

This dissertation found that there was no evidence for any significant variability between NP effects in children and adults when task design was considered, and neither was NP found to vary between adolescents with and without ADHD. To conclude, it is contended that NP effects reflect the workings of an elementary and perhaps fundamental information processing mechanism, one that is first to emerge and last to deteriorate, and one that survives even some psychopathologies.

REFERENCES

Achenbach, T. M. (1991*). Manual for the child behavior check list/4-18 and 1991 profile.* Burlington: University of Vermont.

Allport, D. A., Tipper, S. P., & Chmiel, N. R. (1985). Perceptual integration and postcategorical filtering. In M. I. Posner & O. S. M. Marin (Eds.), *Attention and performance XI* (pp. 107-132). Hillsdale, NJ: Erlbaum.

Aman, C. J., Roberts, R. J. Jr., Pennington, B. F. (1998). A neuropsychological examination of the underlying deficit in attention deficit hyperactivity disorder. Frontal lobe versus right parietal lobe theories. *Developmental Psychology, 34*, 956-969.

American Psychiatric Association. (1994). *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington DC: American Psychiatric Association.

Amso, D., & Johnson, S. P., (2005). Selection and inhibition in infancy: evidence from the spatial negative priming paradigm. *Cognition, 95*, B27-B36.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders: Text revision* (4th ed.). Washington, DC: Author

Anderson, M. C., Bjork, R. A., & Bjork, E. L. (1994). Remembering can cause forgetting: Retrieval dynamics in long-term memory. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *20,* 1063-1087.

Anderson, M. C. & Spellman, B.A. (1995). On the status of inhibitory mechanisms in Cognition; Memory retrieval as a model case. *Psychological Review, 102,* 68-100. *Academy of Child and Adolescent Psychiatry, 29,* 546-557.

Angold, A. E., Costello, J., & Erkanli, A. (1999). Comorbidity. *Journal of Child Psychology and Psychiatry, 40,* 57-87.

Balaban, M. T., Snidman, N., & Kagan, J. (1997). Attention, emotion, and reactivity in early childhood. In P. J. Lang & R. F. Simons (Eds.), *Attention and orienting: Sensory and motivational processes* (pp. 369-391). Hillsdale, NJ: Erlbaum.

Barkley, R. A. (1991). The ecological validity of laboratory and analogue assessments of ADHD symptoms. *Journal of Abnormal Child Psychology, 19*, 149-178.

Barkley, R. A. (1994). Impaired delayed responding: A unified theory of attention deficit hyperactivity disorder. In D. K. Routh (Ed.), *Disruptive behavior disorder in childhood: Essays honouring Herbert C. Quay* (pp. 11-57). New York: Plenum.

Barkley, R. A. (1996). Critical issues in research on attention. In G. R. Lyon & N. A. Krasnegor (Eds.), *Attention, Memory, and Executive Function* (pp. 119-136). Baltimore: Paul H. Brookes

Barkley, R.A. (1997). Behavioral inhibition, sustained attention, and executive functions: Constructing a unifying theory of ADHD, *Psychological Bulletin, 121*, 65-94.

Barkley, R. A. (1998). *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment*. (2nd Ed.). New York: Guilford Press.

Barkley, R. A., Fischer, M., Edelbrock, C. S., & Smallish, L. (1990). The adolescent outcome of children diagnosed by research criteria: I. An 8-year-follow-up study. *Journal of the American Academy of Child & Adolescent Psychiatry, 29*, 546-557.

Barkley, R. A., Grodzinsky, G., & DuPaul, G. J. X. (1992). Frontal lobe functions in attention deficit disorder with and without hyperactivity: A review and research report. *Journal of Abnormal Child Psychology, 20*, 162-188.

Barkley, R.A., Murphey, K., & Kwasnik, D. (1998). Psychological adjustment and adaptive impairments in young adults with ADHD. *Journal of Attention Disorders, 1*, 41-54.

Beech, A. R., Powell, T., McWilliam, J. & Claridge, G. S. (1989). Evidence of reduced 'cognitive inhibition' in schizophrenia. *British Journal of Clinical Psychology, 28*, 109-116.

Berman, T., Douglas, V. I., & Barr, R. G. (1999). Effects of methylphenidate on complex Cognitive processing in Attention-Deficit/Hyperactivity Disorder. *Journal of Abnormal Child Psychology, 108*, 90-105.

Bertenthal, B. I. (1996). Origins and early development of perception, action, and representation. *Annual Review of Psychology, 47*, 431-459.

Brock, S. E., & Knapp, P. K. (1996). Reading comprehension abilities of children with attention-deficit/hyperactivity disorder. *Journal of Attention Disorders, 1,* 173-185.

Brodeur, D. A., & Pond, M. (2001). The development of selective attention in children with attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology, 29,* 229-239.

Bub, D. N., Masson, M. E. J., & Lalonde, C. E. (2006). Cognitive control in children: Stroop interference and suppression of word reading. *Psychological Science, 17,* 351-357.

Buchner, A., Erdfelder, E., & Faul, F. (1997). *G*Power: A priori, post hoc, and compromise power analyses for the Macintosh* (Version 2.1.2) [Computer program]. Trier, Germany: University of Trier. http://www.psycho.uni.duesseldorf.de/aap/projects/gpower /

Buchner, A., & Mayr, S. (2004). Auditory negative priming in younger and older adults. *Quarterly Journal of Experimental Psychology, 57A*, 769-787.

Buchner, A., & Steffens, M. C. (2001). Auditory negative priming in speeded reactions and temporal order judgements. *Quarterly Journal of Experimental Psychology, 54A,* 1125-1142.

Carte, E. T., & Nigg, J. T., & Hinshaw, S. P. (1996). Neuropsychological functioning, motor speed, and language processing in boys with and without ADHD. *Journal of Abnormal Child Psychology, 24,* 481-498.

Carter, C. S., Mintun, M., & Cohen, J. D. (1995). Interference and facilitation effects during selective attention: A H2150PET study of Stroop task performance. *Neuroimage,* 2, 264-272.

Casey, B. J., & Durston, S. (2006). From behavior to cognition to the brain and back: What have we learned from functional imaging studies of attention deficit hyperactivity disorder?. *American Journal of Psychiatry, 163,* 957-960.

Clark, C., Prior, M., & Kinsella, J. (2000). Do executive function deficits differentiate between adolescents with ADHD and oppositional defiant/conduct disorder? A neuropsychological study using the Six Elements Test and Hayling Sentence Completion Test. *Journal of Abnormal Child Psychology, 28,* 403-414.

Cohen, J. (1977). *Statistical power analysis for the behavioral sciences* (Rev. ed.). Hillsdale, NJ: Erlbaum.

Cohen, J. D., Dunbar, K. O., & McClelland, J. L. (1990). On the control of automatic processes: A parallel distributed processing account of the Stroop effect. *Psychological Review, 97,* 332-361.

Cohen, J. D., Dunbar, K.O., Barch, D. M., & Braver, T. S. (1997). Issues concerning relative speed of processing hypotheses, schizophrenic performance deficits, and prefrontal function: Comment on Schooler et al. (1997). *Journal of Experimental Psychology:*

General, 126, 19-36.

Comalli, P. E., Jr. Wapner, S., & Werner, H. (1962). Interference effects of Stroop colour-word test in childhood, adulthood, and aging. *Journal of Genetic Psychology, 100*, 47-53.

Conners, C. K. (1997*). Conners' Rating Scales-Revised: Technical manual*. New York: Multi-Health Systems inc.

Conway, A. R. A. (1999). The time-course of negative priming: Little evidence for episodic trace retrieval. *Memory & Cognition, 27*, 575-583.

Corbett, B., & Stanczak, D. E. (1999). Neuropyschological performance of adults evidencing attention-deficit hyperactivity disorder. *Archives of Clinical Neuropsycho;ogy, 14,* 373-387.

Dalrymple-Alford, E. C., & Budayr, B. (1966). Examination of some aspects of the Stroop color-word test. *Perceptual and Motor Skills, 23*, 1211-1214.

Davidson, M. C., Amso, D., Anderson, L. C., & Diamond, A. (2006). Development of cognitive control and executive function from 4 to 13 years: Evidence from manipulations of memory, inhibition, and task switching. *Neuropsychologia, 44*, 2037-2078.

Davis, H. P., Cohen, A., Gondy, M., Colombo, P., Van Dusselorp, G., Simolke, N., & Romano, J. (1990). Lexical priming effects as a function of age. *Behavioral Neuroscience, 104*, 286-297.

Davis, P., McLeod, K., Ransom, M., & Ongley, P. (1997). The New Zealand socio-economic index of occupational status. Wellington: Statistics, New Zealand.

Dempster, F. N. (1991). Inhibitory processes: A neglected dimension of intelligence. *Intelligence, 15*, 157-173.

Dempster, F. N. (1993). Resistance to interference: Developmental changes in a basic processing mechanism. In M. L. Howe & R. Pasnak (Eds.), *Emerging themes in cognitive development, Vol. 1. Foundations* (pp. 3-27). New York: Springer-Verlag.

Dempster, F. N. (1995). Interference and inhibition in cognition. An historical perspective. In F. N. Dempster & C. J. Brainerd (Eds.), *Interference and inhibition in cognition* (pp. 3-26). San Diego, CA: Academic Press.

Dempster, F. N., & Corkill, A. (1999). Individual differences in susceptibility to interference and general cognitive ability. *Acta Psychologica, 101*, 395-416.

Desimone, R., & Duncan, J. (1995). Neural mechanisms of selective visual attention. *Annual Review of Neuroscience, 18*, 193-222.

Diamond, A. (2002). Normal development of prefrontal cortex from birth to young adulthood. Cognitive functions, anatomy, and biochemistry. In D. T. Stuss & R. T. Knight (Eds.), *Principles of frontal lobe function* (pp. 466-503. London: Oxford University Press.

Douglas, V. I. (1988). Cognitive deficits in children with attention deficit disorder with hyperactivity. *Journal of Child Psychology and Child Psychiatry* (monographic supplement).Oxford: Pergamon.

Douglas, V. I. (1999). Cognitive control processes in ADHD. In H. C. Quay & A. E. Hogan (Eds.), *Handbook of disruptive behavior disorders* (pp. 105-138). New York: Kluwer Academic/Plenum Publishers.

Downey, K., Stelson, F., Pomerleau, O., & Giordiani, B. (1997). Adult attention deficit hyperactivity disorder: Psychological test profiles in a clinical population. *Journal of Nervous and Mental Disease, 185*, 32-38.

Driver, J., & Baylis, G. C. (1993). Cross-modal negative priming and interference in selective attention. *Bulletin of the Psychonomic Society, 31*, 45-48.

Driver, J., & Tipper, S.P. (1989). On the nonselectivity of ‘selective’ seeing: Contrasts between interference and priming in selective attention. *Journal of Experimental Psychology: Human Perception and Performance, 15*, 304-314.

Durgin, F. H. (2000). The reverse Stroop effect. *Psychonomic Bulletin & Review, 7*, 121-125.

Durgin, F. H. (2003). Translation and competition among internal representations in a reverse Stroop effect. *Perception & Psychophysics, 65*, 367-378.

Erdfelder, E., Faul, F., & Buchner, A. (1996). GPOWER: A general power analysis program. *Behavior Research, Instruments, & Computers, 28*, 1-11.

Everett, J., McCorquodale, B., Smith, J., Culverwell, F., Wilks, A., Evans, D., Kay, M., & Baker, D. (1999). Associations between reading ability and visual processes. In J. Everatt (Ed.), *Reading and dyslexia: Visual and attentional processes* (pp.1-39). London, Routledge.

Farkas, M. S., & Hayer, W. J., (1980). Processing consequences of perceptual grouping in selective attention. *Journal of Gerontology, 35*, 207-216.

Ford, R. M., Keating, S., & Patel, R. (2004). Retrieval-induced forgetting: A developmental study. *British Journal of Developmental Psychology*, 2, 585-603.

Fox, E. (1995). Negative priming from ignored distractors in visual selection: A review. *Psychonomic Bulletin and Review, 2,* 145-173.

Frazier, T. W., Demaree, H. A., & Youngstrom, E. A. (2004). Meta-analysis of intellectual and neuropsychological test performance in attention-deficit/hyperactivity disorder. *Neuropsychology, 18*, 543-555.

Friedman, R. (1971). The relationship between intelligence and performance on the Stroop Color-Word test in second- and fifth-grade children. *Journal of Genetic Psychology, 118*, 142-148.

Frings, C., Feix, S., Röthig, U., Brüser, C., & Junge, M. (in press). Children do show negative priming: Further evidence for early development of an intact selective control mechanism. *Developmental Psychology*.

Frings, C., & Wuhr, P. (in press). On distractor repetition benefits in the negative priming paradigm. *Visual Cognition*.

Fuentes, L. J., Humphreys, G. W., Agis, I. F., Carmona, E., & Catena, A. (1998). Object-based perceptual grouping affects negative priming. *Journal of Experimental Psychology: Human Perception and Performance, 24*, 664-672.

Gamboz, N., Russo, R., & Fox, E. (2000). Target selection difficulty, negative priming, and aging. *Psychology and Aging, 15*, 542-550.

Gamboz, N., Russo, R., & Fox, E. (2002). Age differences and the identity negative priming effect: An updated meta-analysis. *Psychology and Aging, 17,* 525-530.

Gaultney, J. F., Kipp, K., Weinstein, J., & McNeill, J. (1999). Inhibition and mental effort in Attention Deficit Hyperactivity Disorder. *Journal of Developmental and Physical Disabilities, 11*, 105-114.

Goodyear, P., & Hynd, G. (1992). Attention deficit disorder with (ADD/H) and without (ADD/WO) hyperactivity: Behavioral and neuropsychological differentiation. *Journal of Clinical Child Psychology, 21,* 273-304.

Golden, C. J. (1978). *Stroop color and word test: A manual for clinical and experimental uses*. Wood Dale, Illinois: Stoelting Company.

Grant, J. D., & Dagenbach, D. (2000). Further considerations regarding inhibitory processes, working memory, and cognitive aging. *American Journal of Psychology, 113*, 69-94.

Grison, S., & Strayer, D. L. (2001). Negative priming and perceptual fluency: More than what meets the eye. *Perception and Psychophysics, 63,* 1063-1071.

Grison, S., Tipper, S., & Hewitt, O. (2005). Long-term negative priming: Support for retrieval of prior attentional processes. *The Quarterly Journal of Experimental Psychology, 58A*, 1199-1224.

Hall, S., Halperin, J. M., Schwartz, S., & Newcorn, J. (1997). Behavioral and executive functions in children with attention-deficit hyperactivity disorder and reading disability. *Journal of Attention Disorders, 1*, 235-247.

Halperin. J. M. (1996). Conceptualizing, describing, and measuring components of attention: A summary. In G. R. Lyon & N. A. Krasnegor (Eds.), *Attention, Memory, and Executive Function* (pp. 119-136). Baltimore: Paul H. Brookes.

Halperin, J. M., McKay, K. E., Matier, K., & Sharma, V. (1994). Attention, response inhibition, and activity level in children: Developmental neuropsychological perspectives. In M. G. Tramontana & S. R. Hooper (Eds.), *Advances in child neuropsychology* (Vol 2, pp. 1-54). New York: Springer-Verlag.

Halperin, J. M. & Schulz, K. P. (2006). Revisiting the role of the prefrontal cortex in the pathophysiology of attention-deficit/hyperactivity disorder. *Psychological Bulletin, 132,* 560-581.

Hammes, J. G. W. (1971). *De Stroop Kkeur-Woord Test* [The Stroop color word test]. Lisse, the Netherlands: Swets and Zeitlinger.

Hanauer, J., & Brooks, P. (2003). Developmental change in the cross-modal Stroop effect.

Perception and Psychophysics, 65, 359-366.

Harnishfeger, K. K. (1995). The development of cognitive inhibition. Theories, definitions and research evidence. In F. N. Dempster & C. J. Brainerd (Eds.), *Interference and inhibition in cognition*. San Diego: Academic Press.

Harnishfeger, K. K., & Bjorklund, D. F. (1994). A developmental perspective on individual differences in inhibition. *Learning and Individual Differences, 6,* 331-355.

Harnishfeger, K. K., & Pope, R. S. (1996). Intending to forget: The development of cognitive inhibition in directed forgetting. *Journal of Experimental Child Psychology, 62*, 292-315.

Hart, E., Leahy, B. B., Loeber, R., Applegate, B., & Frick, P. J. (1995). Developmental change in attention-deficit hyperactivity disorder in boys: A four year longitudinal study. *Journal of Abnormal Child Psychology, 23,* 729-749.

Hasher, L., Stolzfus, E. R., Zacks, R. T., & Rypma, B. (1991). Age and inhibition. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *17*, 163-169.

Hayes, S. M., Ryan, L., Schnyer, D. M., & Nadel, L. (2004). An fMRI study of episodic memory: Retrieval of Object, Spatial, and Temporal Information. *Behavioral Neuroscience, 118,* 885-896.

Hitch, G. J., & Towse, J. N. (1995). Working memory: what develops? In F. E. Weinhart & W. Schneider (Eds.), *Memory performance and competencies. Issues in growth and development* (pp. 3-21). Mahwah, NJ: Lawrence Erlbaum Associates.

Houghton, G., & Mari-Beffa, P. (2005). Dissociating object- and response-based components of negative priming through effects of practice. *Perception & Psychophysic, 67,* 1423-1436.

Houghton, G., & Tipper, S. P. (1994). A model of inhibitory mechanisms in selective attention. In D. Dagenbach & T. Carr (Eds.), *Inhibitory mechanisms in attention, memory and language* (pp.53-112). San Diego, CA: Academic Press.

Houghton, G., Tipper, S. P., Weaver, B., & Shore, D. I. (1996). Inhibition and interference in selective attention: Some tests of a neural network model. *Visual Cognition, 3,* 119-164.

Huang-Pollock, C. L., & Nigg, J. T. (2003). Searching for the attention deficit in attention deficit hyperactivity disorder: The case of visuospatial orienting. *Clinical Psychology Review, 23,* 801-830.

Huang-Pollock, C. L., Nigg, J. T., & Halperin, J. M. (2006). Single dissociation findings of ADHD deficits in vigilance but not anterior posterior attention deficits. *Neuropsychology, 20,* 420-429.

Hughes, R., & Jones, D. M. (2003). A negative order-repetition priming effect: Inhibition of order in unattended order sequences? *Journal of Experimental Psychology: Human Perception and Performance, 29*, 199-218.

Huttenlocher, P. R. (1990). Morphometric study of human cerebral cortex development. *Neuropsychologia, 28,* 517-527.

Hynd, G. W., Hern, K. L., Novey, E. S., Eliopulos, R. T., Marshall, R., Gonzalez, J. J., & Voeller, K. K. (1993). Attention deficit-hyperactivity disorder and asymmetry of the caudate nucleus. *Journal of Child Neurology, 8,* 339-347.

Johnstone, S.J., Dimoska, A., Smith, J. L., Barry, R. J., Pleffer, C.B., Chiswick. D., Clarke, A. R. (2007). The development of stop-signal and go/no-go response inhibition in children aged 7-12 years: Performance and event-related potential indices. *International Journal of Psychophysiology*, 63, 25-38.

Kail, R. (2002). Developmental change in proactive interference. *Child Development, 73*, 1703-1714.

Kaufman, J., Birmaker, B., Brent, D., Rao, U., Flynn, C., Moreci, P., Williamson, D., & Ryan, N. (1997). Schedule for affective disorders and schizophrenia for school-age children-present and lifetime version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child and Adolescent Psychiatry, 36*, 980-988.

Kellman, P. J. (1993). Kinematic foundations of infant visual perception. In C. Granrud (Ed.), *Visual perception and cognition in infancy: Carnegie Mellon Symposia on Cognition*. Hillsdale, NJ: Erlbaum.

Kellman, P. J., & Short, K. R. (1987). Development of three-dimensional form perception. *Journal of Experimental Psychology: Human Perception and Performance, 13*(4), 545-557.

Kellman, P. J., Spelke, E. S., & Short, K. R. (1986). Infant perception of object unity from translatory motion in depth and vertical translation. *Child Development, 57*, 72-86.

Khurana, B. (2000). Not to be and then to be: Visual representation for ignored unfamiliar faces. *Journal of Experimental Psychology: Human Perception and Performance, 26*, 246-263.

Kieley, J. M., & Hartley, A. A. (1997). Age-related equivalence of identity suppression in the Stroop color-word task. *Psychology and Aging, 12*, 22-29.

Kok, A. (1999). Varieties of inhibition: Manifestations in cognition, event-related potentials *Psychology: Human Perception and Performance, 26*, 1038-1052.

Kramer, A. F., Humphrey, D. G., Larish, J. F., Logan, G. D., & Strayer, D. L. (1994). Aging and inhibition: Beyond a unitary view of inhibitory processing in attention. *Psychology and Aging, 9,* 491-512.

Kramer, A. F., & Strayer, D. L. (2001). Influence of stimulus repetition on negative priming. *Psychology and Aging, 16,* 580-587.

Lane, D. M., & Pearson, D. A. (1982). The development of selective attention. *Merrill-Palmer Quarterly, 82,* 317-337.

Langley, L. K., Overmier, J. B., Knopman, D. S., & Prod'Homme, M. M. (1998). Inhibition and habituation: Preserved mechanisms of attentional selectionin aging and Alzheimer's disease. *Neuropsychology, 12,* 353-366.

Lahey, B. B., & Carlson, C. L. (1992). Validity of the diagnostic category of attention deficit disorder without hyperactivity: A review of the literature. In S. E. Shaywitz & B. Shaywitz (Eds.), *Attention deficit disorder comes of age: Toward the twenty-first century* (pp. 119-144). Austin, TX: Pro-Ed.

Langley, L. K., Overmier, J., B., Knopman, D. S., & Prod'Homme, M. M. (1998). Inhibition and habituation: Preserved mechanisms of attentional selection in aging and Alzheimer's disease. *Neuropsychology, 12*, 353-366.

Laplante, L., Everett, H., & Thomas, J. (1992). Inhibition through negative priming with Stroop stimuli in schizophrenia. *British Journal of Clinical Psychology, 31,* 307-326.

Lavie, N., & Fox, E. (2000). The role of perceptual load in negative priming. *Journal of Experimental Psychology: Human Perception and Performance, 26,* 1038-1052.

Lechunga, M. T., Moreno, V., Pelegrina, S., Gomez-Ariza, C. J., & Bajo, M. T. (2006). Age differences in memory control: Evidence from updating and retrieval-practice tasks. *Acta Psychologica, 123,* 279-298.

Little, D. H., & Hartley, A. A. (2000). Further evidence that negative priming in the Stroop task is equivalent in older and younger adults. *Psychology and Aging, 15,* 9-17.

Lorch, E. P., Anderson, D. R., & Wells, A. D. (1984). Effects of irrelevant information on speeded classification tasks: Interference is reduced by habituation. *Journal of Experimental Psychology: Human Perception and Performance, 10,* 850-864.

Lufi, D., Cohen, A., & Parish-Plass, J. (1990). Identifying attention deficit hyperactive disorder with the WISC-R and the Stroop color and word test. *Psychology in the schools,* 27, 28-34.

MacLeod, C. M. (1991). Half a century of research on the Stroop effect: An integrative review. *Psychological Bulletin, 109,* 163-203.

MacLeod, D., & Prior, M. (1996). Attention deficits in adolescents with ADHD and other clinical groups. *Child Neuropsychology, 2,* 1-10.

MacQueen, G. M., Galway, T., Goldberg, J. O., & Tipper, S. P. (2003). Impaired distractor inhibition in patients with schizophrenia on a negative priming task. *Psychological Medicine, 33,* 121-129.

Mannuzza, S., Klein, R. G., Bessler, A., Malloy, P., & LaPadula, M. (1998). Adult psychiatric status of hyperactive boys grown up. *American Journal of Psychiatry, 155*, 493-498.

Marriott, M. (1998). Selective attention, negative priming, and hyperactivity: Investigating the 'AD' in ADHD. Unpublished doctoral dissertation, McMaster University, Canada.

Malley, G. B., & Strayer, D. L. (1995). Effect of stimulus repetition on positive and negative identity priming. *Perception and Psychophysics, 57*, 657-667.

Mari-Beffa, P., Estevez, A.F., & Danziger, S. (2000). Stroop interference and negative priming: Problems with inferences from null results. *Psychonomic Bulletin and Review, 7,* 499-503.

Mason, D. J., Humphreys, G. W., & Kent, L. (2004). Visual search, singleton capture, and the control of attentional set in ADHD. *Cognitive Neuropsychology, 21*, 661-687.

May, C. P., Kane, M. T., & Hasher, L. (1995). Determinants of negative priming. Psychological Bulletin, 118, 35-54.

Mayr, S., & Buchner, A. (in press). Negative priming as a memory phenomenon: A review of 20 years of negative priming research. *Zeitschrift fur Psychology – Journal of Psychology*.

McDowd, J. M., & Oseas-Kreger, D. M. (1991). Aging, inhibitory processes, and negative priming. *Journal of Gerontology: Psychological Sciences, 46B,* 340-345.

Milich, R., Balentine, A., & Lynam, D. (2001). ADHD combined type and ADHD predominantly inattentive type are distinct and unrelated disorders. *Clinical Psychology, Science and Practice, 8*, 463-488.

Milliken, B., Joordens, S., Merikle, P. M., & Seiffert, A. E. (1998). Selective attention: A reevaluation of the implications of negative priming. *Psychological Review, 105*, 203-229.

Mirsky, A. F., Anthony, B. J., Duncan, C. C., Ahern, M. B., & Kellam, S. G. (1991). Analysis of the elements of attention: A neuropsychological approach. *Neuropsychology Review, 2*, 109-145.

Moore, C. M. (1994). Negative priming depends on probe-trial conflict: Where has all the inhibition gone? *Perception & Psychophysics, 56*, 133-147.

Moritz, S., Jacobsen, D., Mersmann, K., Krauz, M., & Andresen, B. (2000). Negative priming in schizophrenia: No evidence for reduced cognitive inhibition. *Journal of Nervous and Mental Disease, 188*, 624-627.

Moritz, S., Ruff, C., Wilke, U., Andresen, B. , Krauz, M., & Naber, D. (2001). Negative *priming in schizophrenia: Effects of masking and prime presentation time. Schizophrenia Research, 48*, 291-299.

Moulin, C. J., Perfect, T. J., Conway, M. A., North, A. S., Jones R. W., James, N. (2002). Retrieval-induced forgetting in Alzheimer's disease. *Neuropsychologia*, *40*, 862–7.

Muller, U., Dick, A. S., Gela., K., Overton, W. F., & Zelazo, P. D. (2006). The role of negative priming in preschoolers' flexible rule use on the dimensional change card sort task, *Child Development, 77*, 395-412.

Murphey, K., Russell, A., Barkley, R. A., & Bush, T. (2001). Executive functioning and olfactory identification in young adults with attention-deficit hyperactivity disorder, *Neuropsychology*, *15*, 211-220.

Neill, W. T. (1997). Episodic retrieval in negative priming and repetition priming. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 6*, 1291-1305.

Neill, W. T., & Mathis, K. M. (1998). Transfer-inappropriate processing: negative priming and related phenomena. In D. L. Medlin (Ed.), *The psychology of learning and motivation: Advances in research and theory (Vol.38, pp. 1-44).* San Diego, CA: Academic Press.

Neill, W. T., & Valdes, L. A. (1992). Persistence of negative priming: Steady state or decay.

Journal of Experimental Psychology: Learning, Memory, and Cognition, 18, 565-576.

Neill, W. T., Valdes, L. A., Terry, K. M., & Gorfein, D. S. (1992). Persistence of negative priming: II. Evidence for episodic trace retrieval. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 18,* 993-1000.

Neill, W. T., & Westberry, R. L. (1987). Selective attention and the suppression of cognitive noise. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 13,* 327-334.

Neufeld, R. W. (2002). Introduction to the special section on cognitive science and psychological assessment. *Psychological Assessment, 14,* 235-238.

Neumann, E., Cherau, J. F., Hood, K. L., & Steinnagel, S. L. (1993). Does inhibition spread in a manner analogous to spreading activation? *Memory, 1,* 81-105.

Neumann, E., & DeSchepper, B. G. (1991). Costs and benefits of target activation and distractor inhibition in selective attention. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 17,* 1136-1145.

Neumann, E., & DeSchepper, B. G. (1992). An inhibition based fan effect: Evidence for an active suppression mechanism in selective attention. *Canadian Journal of Psychology, 46,* 1-40.

Neumann, E. & Gaukrodger, S. J. (2005). Invariant effects of working memory load in the face of competition. *Proceedings of the 27th Annual Conference of the Cognitive Science Society,* pp. 1624-1629.

Neumann, E. McCloskey, M. S., & Felio, A. C. (1999). Cross-language positive priming disappears, negative priming does not: Evidence for two sources of selective inhibition. *Memory & Cognition, 27*, 1051-1063.

Neumann, E. & Russell, P. N. (2000, November). *Persistence of token resistence: Once unattended word produce long-term negative priming*. Poster session presented at the annual meeting of the Psychonomic Spciety, New Orleans, LA, USA.

Neumann, E., McCloskey, M. S., & Felio, A. C. (1999). Cross-language positive priming disappears, negative priming does not: Evidence for two sources of selective inhibition. *Memory & Cognition, 27*, 1051-1063.

Nigg, J. T. (2001). Is ADHD an inhibitory disorder? *Psychological Bulletin, 127,* 571-598.

Nigg, J. T. (2000). On inhibition/disinhibition in developmental psychopathology: Views from cognitive and personality psychology and a working inhibition taxonomy. *Psychological Bulletin, 126*, 220-246.

Nigg, J. T., Butler, K. M., Huang-Pollock, C. L., & Henderson, J. M. (2002). Inhibitory processes in adults with persistent childhood onset ADHD. *Journal of Consulting and Clinical Psychology, 70*, 153-157.

Nigg, J. T., & Casey, B. J. (2005). An integrative theory of attention-deficit/hyperactivity disorder based on the cognitive and affective neurosciences. *Developmental Psychopathology*, 785-806.

Nigg, J. T., Hinshaw, S. P., Carte, E. T., & Treuting, J. J. (1998). Neuropsychological correlates of childhood attention-deficit/hyperactivity disorder: Explainable by comorbid disruptive behavior or reading problems? *Journal of Abnormal Psychology, 107*, 468-480.

Ossman, J. M., & Mulligan, N. W. (2003). Inhibition and attention deficit hyperactivity disorder in adults. *American Journal of Psychology, 116*, 35-50.

Ozonoff, S., Strayer, D. L., McMahon, W. M., & Filloux, F. (1998). Inhibitory deficits in Tourette syndrome: A function of comorbidity and symptom severity. *Journal of Child Psychiatry, 39*, 1109-1118.

Pearson, D. A., & Lane. D. M. (1991). Auditory attention switching: A developmental study. *Journal of Experimental Child Psychology, 51*, 320-334.

Pennington, B. F., Grossier, D., & Welsh, M. C. (1993). Contrasting deficits in attention deficit disorder versus reading disability. *Developmental Psychology, 29*, 511-523.

Pennington, B. F., & Ozonoff, S. (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry, 37*, 51-87.

Peru, A., Faccioli, C., & Tassinari, G. (2006). Stroop effects from 3 to 10 years: The critical role of reading acquisition. *Archives Italiennes de Biologie, 144*, 45-62.

Pesta, B. J., & Sanders, R. E. (2000). Aging and negative priming: Is ignored information inhibited or remembered? *Experimental Aging Research, 26*, 37-56.

Pritchard, V. E. (2002). Evidence for negative priming in children. Unpublished masters thesis. University of Canterbury, New Zealand.

Pritchard, V. E., & Neumann, E. (2004). Negative priming effects in children engaged in non-spatial tasks: Evidence for early development of an intact inhibitory mechanism. *Developmental Psychology, 40*, 191-203.

Pritchard, V. E., & Neumann, E. (2007a). Pitfalls of developmentally inappropriate negative priming tasks. Manuscript under revision (*Developmental Psychology*).

Pritchard, V. E., & Neumann, E. (2007b). Adding nonconflict trials in Stroop negative priming tasks eliminates distractor suppression effects in children, but not adults.

Manuscript under review (*Developmental Psychology*).

Pritchard, V. E., Healey, D., & Neumann, E. (2006). Assessing selective attention abilities in ADHD, highly creative, and normal young children via Stroop interference and negative priming effects. In C. M. Fletcher-Flinn & G. M. Habermann (Eds.), *Cognition and language: Perspectives from New Zealand* (pp.207-224). Australia, Brisbane:Australian Academic Press.

Pritchard, V. E., Neumann, E., & Rucklidge, J. J. (2007a). Interference and negative priming effects in adolescents with attention deficit hyperactivity disorder. *American Journal of Psychology*, 120, 91-122.

Pritchard, V.E., Neumann, E., & Rucklidge, J. J. (2007b). Selective attention and inhibitory deficits in ADHD: Does subtype or comorbidity modulate negative priming effects? Manuscript under revision (*Brain and Cognition*).

Quay, H. C. (1998). Attention deficit disorder and the behavioral inhibition systems: The relevance of the neuropsychological theory of Jeffrey A. Gray. In L. M. Bloomingdale & J. A. Sergeant (Eds.), *Attention deficit disorder: Criteria, cognition, and intervention* (pp. 117-125). New York: Pergamon.

Racsmány, M., & Conway, M.A. (2006). Episodic inhibition. *Journal of Experimental Psychology: Learning, Memory & Cognition*, 32, 44-57.

Ridderinkhof, K. R., van der Molen, M. W., Band, G. P. H., & Bashore, T. R. (1997). Sources of interference from irrelevant information: A developmental study. *Journal of Experimental Child Psychology, 65,* 315-341.

Ridderinkhof, K. R., van den Wildenberg, W. P. M., Segalowitz, S. J., & Carter, C. S.

(2004). Neurocognitive mechanisms of cognitive control: The role of prefrontal cortex in action selection, response inhibition, performance monitoring, and reward-based learning. *Brain and Cognition, 56*, 129-140.

Ross, R. G., Hommer, D., Breiger, D., Varley, C., & Radant, A. (1994). Eye movement task related to frontal lobe functioning in children with attention deficit disorder. *Journal of the American Academy of Child & Adolescent Psychiatry, 33*, 869-974.

Rubia, K., Oosterlaan, J., Sergeant, J. A., Brandeis, D., & Leeuwen, T. (1998). Inhibitory function in hyperactive boys. *Behavioral Brain Research, 94,* 25-32.

Rucklidge, J. J., & Tannock, R. (2002). Neuropsychological profiles of adolescents with ADHD: Effects of reading difficulties and gender. *Journal of Child Psychology and Psychiatry, 43*, 988-1003.

Salthouse, T. A., & Babcock, R. L. (1991). Decomposing adult age differences in working memory. *Developmental Psychology, 27*, 763-776.

Sanders, L. D., Stevens, C., Coch, D., & Neville, H. J. (2006). Selective auditory attention in 3- to 5-year-old children: An event-related potential study. *Neuropsychologia, 44,* 2126-2138.

Schachar, R., & Logan, G. D. (1990). Impulsivity and inhibitory control in normal development and childhood psychopathology. *Developmental Psychology, 26*, 710-20.

Schacher, R., Tannock, R., Marriott, M., & Logan, G. (1995). Deficient inhibitory control in attention deficit hyperactivity disorder. *Journal of Abnormal Child Psychology, 23,* 411-437.

Schooler, C., Neumann, E., Caplan, L. J., & Roberts, B. R. (1997a). A time course analysis of Stroop interference and facilitation: Comparing normal individuals and individuals with schizophrenia. *Journal of Experimental Psychology General*, *126*, 19-36.

Schooler, C., Neumann, E., Caplan, L. J., & Roberts, B. R. (1997b). Stroop theory, memory, and prefrontal cortical functioning: Reply to Cohen et al. (1997). *Journal of Experimental Psychology: General*, *126*, 42- 44.

Schooler, C., Neumann, E., Caplan, L. J., & Roberts, B. R., (1997c). Continued Inhibitory Capacity throughout adulthood: Conceptual negative priming in younger and older adults. *Psychology and Aging, 4,* 667-674.

Seidman, L. J., Biederman, J., Faraone, S. V., Weber, W., & Ouellette, J. (1997). Toward defining a neuropsychology of Attention-Deficit-Hyperactivity Disorder: Performance of children and adolescents from a large clinically referred sample. *Journal of Consulting and Clinical Psychology*, *65*, 150-160.

Seidman, L. J., Biederman, J., Monuteaux, M. C., Weber, W., & Faraone, S. V. (2000). Neuropsychological functioning in nonreferred siblings of children with attention deficit/hyperactivity disorder. *Journal of Abnormal Psychology, 109,* 252-265.

Semrud-Clikeman, M., Guy, K., Griffin, J. D., & Hynd, G. W. (2000). Rapid naming deficits in children and adolescents with reading disabilities and attention deficit hyperactivity disorder. *Brain and Language, 74*, 70-83.

Sergeant, J. A., Oosterlaan, J., & van der Meere, J. (1999). Information processing and energetic factors in attention-deficit/hyperactivity disorder. In H. C. Quay & A. E. Hogan (Eds.), *Handbook of Disruptive Behavior Disorders*. New York: Kluwer/Plenum, 75-104.

Shader, R. I., Harmatz, J. S., Oesterheld, J. R., Parmelee, D. X., Sallee, F. R., & Greenblatt, D. J. (1999). Population pharmacokinetics of methylphenidate in children with attention-deficit/hyperactivity disorder. *Journal of Clinical Pharmacology, 39*, 775-785.

Simone, P. M., & McCormick, E. B. (1999). Effect of defining feature on negative priming across the life span. *Visual Cognition, 6*, 587-606.

Sokolov, E. N. (1963). *Perception and the conditioned reflex*. New York: Macmillan.

Strayer, C. D., & Grison, S. (1999). Negative priming is contingent on stimulus repetition. *Journal of Experimental Psychology: Human Perception and Performance, 25,* 24-38.

Stroop, J. R. (1935). Studies of interference in serial verbal reaction. *Journal of Experimental Psychology, 18*, 643-661.

Stuss, D. T., & Bensen, D. F. (1984). Neuropsychological studies of the frontal lobes. *Psychological Bulletin, 95,* 3-28.

Sugg, M. J., & McDonald,J. E. (1994). Time course of inhibition in color-response and word-response versions of the Stroop task. Hournal of Experimental Psychology:Human *Perception and Performance, 20*, 647-675.

Sullivan, M. P., & Faust, M. E. (1993). Evidence of identity inhibition during selective attention in older adults. *Psychology and Aging, 8,* 589-598.

Sullivan, M. P., Faust, M. E., & Balota, D. A. (1995). Identity negative priming in older adults and individuals with dementia of the Alzheimer type. *Neuropsychology, 9,* 537-555.

Swanson, H. L. (1999). What develops in working memory? A life span perspective.

Developmental Psychology, 35, 986-1000.

Tannock, R. (1998). Attention deficit hyperactivity disorder: Advances in cognitive, neurobiological and genetic research. *Journal of Child Psychology and Psychiatry, 39,* 65-99.

Tannock, R., Martinussen, R., & Fritjers, J. (2000). Naming speed performance and stimulant effects indicate effortful semantic processing deficits in attention-deficit/hyperactivity disorder. *Journal of Abnormal Child Psychology, 28*, 237-252.

Tannock, R., Schacher, 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.

Tipper, S. P. (1985). The negative priming effect: Inhibitory effects of ignored primes. *Quarterly Journal of Experimental Psychology, 37A*, 571-590.

Tipper, S. P. (1991). Less attentional selectivity as a result of declining inhibition in older adults. *Bulletin of the Psychonomic Society, 29,* 45-47.

Tipper, S. P. (2001). Does negative priming reflect inhibitory mechanism? A review and integration of conflicting views. *Quarterly Journal of Experimental Psychology, 54,* 321-343.

Tipper, S. P., Bourque, T. A., Anderson, & S. H., Brehaut, J.C. (1989). Mechanisms of attention: A developmental study. *Journal of Experimental Child Psychology, 48*, 353-378.

Tipper. S. P., & Cranston, M. (1985). Selective attention and priming: Inhibitory and facilitatory effects of ignored primes. *Quarterly Journal of Experimental Psychology, 37A*, 591-611.

Tipper, S. P., & Driver, J. (1988). Negative priming between pictures and words in a selective attention task: Evidence for semantic processing of ignored stimuli. *Memory & Cognition, 16*, 64-70.

Treisman, A., & DeSchepper, B. G., (1996). Object tokens, attention, and visual memory. In I. Inui & J. McClelland (Eds.), *Attention and performance XVI: Information integration in perception and communication* (pp. 15-46). Cambridge, MA: MIT Press.

Tzelgov, J. (1997). Specifying the relations between automaticity and conscious: A theoretical note. *Conciousness and Cognition, 6,* 441-451.

Vaidya, C. J., Austin, G., Kirkorian, G., Ridlehuber, H. W., Desmond, J. E., Glover, G. H., & Gabrieli, J. D. E. (1998). Selective effects of methylphenidate in attention deficit hyperactivity disorder: A functional magnetic resonance study. *Neurobiology, 95*, 14494-14499.

van Mourick, R., Oosterlann, J., & Sergeant, J. A. (2005). The Stroop revisited: A meta-analysis of interference control in ADHD. *Journal of Child Psychology and Psychiatry*. *46,* 150-165.

Verhaeghen, P., & DeMeersman, L. (1998). Aging and the negative priming effect. A meta-analysis. *Psychology and Aging, 13,* 435-444.

Verhaeghen, P., & Salthouse, T. A. (1997). Meta-analyses of age-cognition relations in adulthood; Estimates of linear and nonlinear age effects and structural models. *Psychological Bulletin, 122,* 231-249.

Vuilleumier, P., Schwartz, S., Duhoux, S., Dolan, R. J. & Driver, J. (2005). Selective attention modulates neural substrates of repetition priming and “implicit” visual\memory: Suppressions and enhancements revealed by fMRI. *Journal of Cognitive Neuroscience, 17*, 1245-1260.

Wakefield, J. C. (1992). Disorder as harmful dysfunction: A conceptual critique of the DSM-III-R definition of mental disorder. *Psychological Review, 99*, 232-247.

Wechsler, D. (1991). *Manual for the WISC-III.* New York: Psychological Corporation.

Wechsler, D. (1997). *Manual for the WAIS-III.* New York: Psychological Corporation.

Wilson, S. P., & Kipp, K. (1998). The development of efficient inhibition: Evidence from directed-forgetting tasks. *Developmental Review*, 18, 86-123.

Wong, K. F. E. (2000). Dissociative prime-probe contextual similarity effects on negative priming and repetition priming: A challenge to episodic retrieval as a unified account of negative priming. *Journal of Experimental Psychology: Learning, Memory, and Cognition*, *26*, 1411-1422.

Wright, B. C., & Wanley, A. (2003). Adults’ versus children’s performance on the Stroop task: Interference and facilitation, *British Journal of Psychology, 94,* 475-485.

Zabal, A. & Buchner, A. (2006). Normal auditory negative priming in schizophrenic patients. *Quarterly Journal of Experimental Psychology*, *59*, 1224-1236.

Zelazo, P. D., Craik, F. I. M., & Booth, L. (2004). Executive functions across the life span. *Acta Psychologica, 115,* 167-183.

Zelazo, P. D., & Muller, U. (2002). Executive functioning in typical and atypical development. In U. Goswami (ED.) *Handbook of Childhood Cognitive Development* (pp 445-469). Oxford, England: Blackwell.

Zellner, M., & Bauml, K. H. (2005). Intact retrieval inhibition in children's episodic recall. *Memory & Cognition, 33*, 396-404.

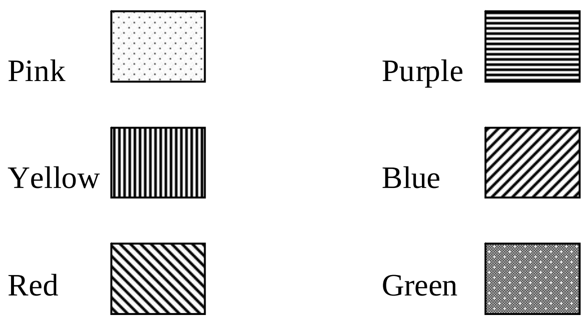
Appendices

Appendix A

Example of Stroop NP task control (or unrelated [UR]) and ignored repetition conditions used in Studies 1-4.

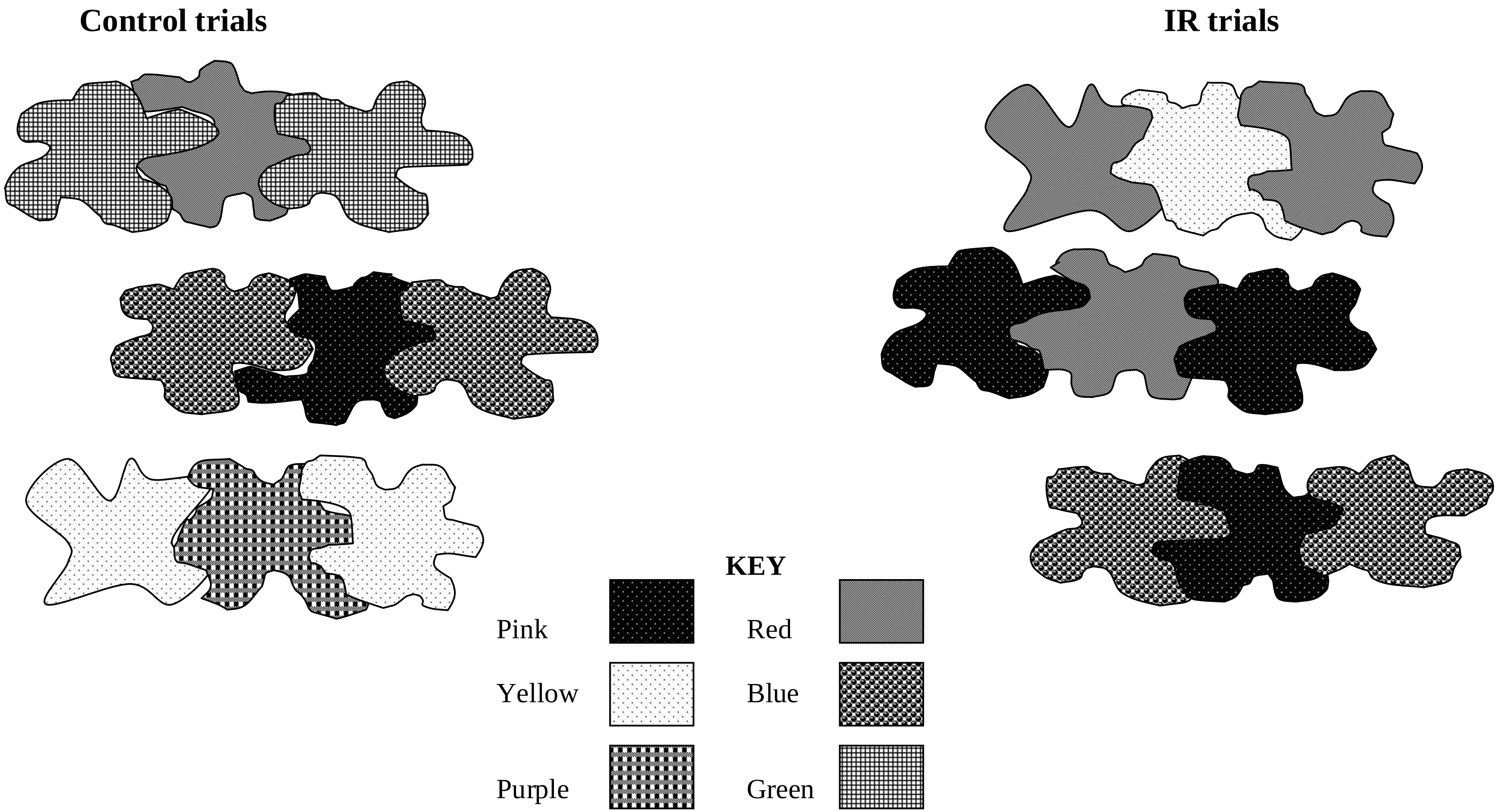
Control trials **IR trials**

Key



Appendix B

Example of color blob/flanker NP task control (or unrelated [UR]) and ignored repetition (IR) conditions used in Studies 1, 3, and 4.



Appendix C

Procedural and administrational instructions for the Stroop negative priming task

This card has several words written in different coloured inks (the participant is shown the first practice card). Point to the first colourword at the top of a list on a card and say “I want you to name the ink colour of each word that you see starting at the top of this card and working down to the bottom of this card. You will be doing this for each card I show you. Try and name the ink colour of each word as fast as you can without making any mistakes. If you do make a mistake don’t stop to fix it up but keep going until you finish the card. This is a test to see how fast you can see and name colours on a card. Before we start the real test you can practice with three cards. (start with the first “shown” practice card but cover it with the cover sheet and time it as follows). When I say “Go!” I will lift this (a blank card covering the experimental card) and you start straight away to name the ink colour of each word from the top to the bottom of the card that you see underneath. Do this as fast as you can, but try not to make any mistakes (participant completes three practice cards; record the times starting the stop watch in synchrony with lifting off the blank cover sheet and stopping as soon as the participant starts to verbalize the last colour regardless of whether they name it correctly or not)

Tell the particpant that the next card will be the first of the test cards (whereas, it is in fact the last practice card). After this has been completed, administer the 12 experimental cards in the order specified on the script sheet.

Appendix D

Procedural and administrational instructions for the colour blob/flanker negative priming task

This card has several groups of different coloured blobs on it (the participant is shown the first practice card). Point to the top blob set on a card and say “I want you to name the colour of the blob in the middle of each group starting at the top of this card and working down to the bottom of this card. You will be doing this for each card I show you. Try and name the colour of each middle blob as fast as you can without making any mistakes. If you do make a mistake don’t stop to fix it up but keep going until you finish the card. This is a test to see how fast you can see and name colours on a card. Before we start the real test you can practice with three cards. (start with the first “shown” practice card but cover it with the cover sheet and time it as follows). When I say “Go!” I will lift this (a blank card covering the experimental card) and you start straight away to name the colour of each middle blob from the top to the bottom of the card that you see underneath. Do this as fast as you can, but try not to make any mistakes (participant completes three practice cards; record the times starting the stop watch in synchrony with lifting off the blank cover sheet and stopping as soon as the participant starts to verbalize the last colour regardless of whether they name it correctly or not). Tell the particpant that the next card will be the first of the test cards (whereas, it is in fact the last practice card). After this has been completed, administer the 12 experimental cards in the order specified on the script sheet.

Appendix E

CALCULATING INTERFERENCE SCORES

(Golden, 1978, pg. 30)

To determine pure interference scores, a predicted CW score must be subtracted from the raw CW scores. The higher the resultant score, the less susceptible the client to interference.

The following formula is used for calculating the predicted CW scores:

$$\frac{C \times W}{C + W}$$

All predicted CW's may be determined directly from this formula.

Derivation of the Formula

The formula was devised by assuming that the easiest way to complete the CW page is to first read the word, then name the color. Thus, the time to complete a single item on the CW page is the time to read one word plus the time to name one color.

The time to name one word is 45 seconds divided by W, the number of words completed on Page 1. Similarly, the time to name one color is 45/C. Thus the time for one CW item is:

$$\frac{45}{W} + \frac{45}{C}$$

This simplifies to: $\frac{45\,(W + C)}{W \times C}$

The number of items completed in 45 seconds becomes:

$$\frac{45}{\frac{45\,(W + C)}{W \times C}}$$

<u>This simplifies to:</u>

$$\frac{1}{\frac{W + C}{W \times C}}$$

<u>This becomes:</u>

$$\frac{W \times C}{W + C}$$

Interference scores should be calculated using <u>age corrected scores</u>. (Golden, pg. 31)

AGE CORRECTIONS FOR CHILDREN

(Golden, 1978, Table II-B, pg.32)

Experimental Data

CARD

Age Group	Word	Color	Color-Word
7	52	40	26
8	46	36	24
9	41	29	20
10	34	24	16
11	26	16	11
12	15	10	7
13	10	7	5
14	5	0	2
15	3	0	0
16	0	0	0

Table II-B (Golden, 1978) lists the sums to be added to the scores of children.

The below provides an example of an age correction for a child aged 13 years that is based on Golden's (1978) criteria.

Age corrected W = Raw W + 10
Age corrected C = Raw C + 7
Age corrected CW = Raw CW + 5

Appendix F

Parent's information and consent form

Consent to Participate in a Research Study/ Selective Attention Study

Dear Parent,

Your child is invited to take part in a research study that investigates selective attention in typically developing children. This is a section of a wide study in which the selective attention skills of typically developing children and adults are compared with the attention skills of individuals with ADHD. Potential outcomes here will a) help to establish the theoretical validity of some diagnostic measures of attention and b) begin to help isolate deficits in attention specific to ADHD, which together with recent advance in neuro-imaging technique, may identify their associated neural substrates. It is important that you read and understand several general principles that apply to all children who take part in this study: (a) taking part in the study is entirely voluntary; (b) personal benefit may not result from taking part in this study, but knowledge may be gained that will benefit others; (c) if your child wishes to withdraw from participation or you wish to withdraw your child's participation, your child or you may do so at any time. The nature of the study, the risks, inconveniences, and other pertinent information about the study are discussed below.

Purpose
The purpose of this study is to investigate if and how children differ from adults in their ability to attend to and perceive information. Data gained from this study will be compared to data from adult participants who take part in an identical task. Your child will be asked to do a task that involves seeing verbal material and making timed responses to this material. Children will be asked to do the following:

1) Detect and respond to words that are presented relatively quickly

2) Name the ink colours of various visually presented words

Inconvenience, Risk, and Confidentiality
Your child will be required to participate in one 15-minute session. This will be held in a classroom at your child's school during school hours. It is not expected that your child find participation in this study unpleasant, and there are no foreseeable risks involved with participation. I would like you to know that your child's data will be held in the strictest confidence. No names or individual identification will be used in publications that may arise as a result of this research. Only the principal investigators will have access to the names of the participants and their data.

This study is being carried out as research for the degree of PhD by Verena Pritchard (MSc [hons]), under the primary supervision of Dr. Ewald. Neumann, who can be contacted at the Psychology Department, University of Canterbury (phone 364-2987 ext. 7955 or 6964). He will be pleased to discuss any concerns you may have about your child's participation in this study. The primary experimenter will be Verena Pritchard who may be contacted at ext. 3408.

Please note, that while parental consent is required (see over) it is also important that your child agrees to their involvement in this study and gives their written assent. An information sheet is also provided over for your child. Please ensure that your child understands what is involved in this study before she/he signs.

This study has been reviewed and approved by the University of Canterbury Human Ethics Committee.

Consent to Participate in a Research Study/ Selective Attention Study

I have read and understood the description of the above named project. On this basis, I agree to allow my child to participate as a subject in the project, and I consent to the publication of the results of the project with the understanding that anonymity will be preserved. I understand also that at any time I may withdraw my child from the project.

I agree to allow my child, ____ to participate in the study described above. I have informed my child about what is involved in taking part in this study and was present when he/she gave their written assent.

Signed: ____

Date: ___________________

Please Print:
Child's full name: ____

Child's birth date: _____________________

Appendix G

Child’s information and consent form

Selective Attention Study

Why are we doing this study? This study looks at how fast and how well children are able to respond to colours or words that are shown very quickly. We are interested in finding out how and if the way that children do this changes as children get older. We also want to learn if children are different from adults in the way they see and respond to colours and words. These things are important for us to know so that in the future we can help those (adults and children) that have problems with concentration. You have been chosen to take part in this study because you are going to act as a comparison to adults who will be taking part in the same study as you.

What will happen during the study? You will be seeing a whole lot of simple words and colours. You will be asked to respond to these as fast and as carefully as you can. You will be timed to see how fast you can do this. The study will not take long to do, about 15 minutes. The study will take place at your school in a quiet classroom.

Are there good things and bad things about the study? There are no bad things from being in this study. You can have a look at how fast you were able to respond to words and colours. You will get some stickers and a certificate to thank you for taking part in the study. You will be helping us to better understand how children and adults are able to concentrate.

Who will know about what I did in the study? No one, only you, is going to know what you did or how you did in the study. We keep this information safe.

Can I decide if I want to be in the study? If you do not want to be part of this study this is OK. No one will be disappointed or upset. If you say yes now, but change your mind, you can say no later and that will be OK. There will be a chance for you to ask any questions before taking part in the study at your school.

Please write out your name and sign in the spaces below and bring this form along with the form attached that your parents or legal guardian must sign, back to school.

Your name:____, and

Signature: ____

Date: ____________________

Child's consent form

Selective attention: A developmental study

I have read and understood the description of the above-named project. On this basis I agree to participate as a subject in the project, and I consent to publication of the results of the project with the understanding that anonymity will be preserved. I understand also that I may at any time withdraw from the project including withdrawal of any information I have provided.

NAME (please print): ____

Signature: ____

Date: _________________________

Date of birth:___________________